Hyperventilation before Resistance Exercise: Cerebral Hemodynamics and Orthostasis

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ABSTRACT

ROMERO, S. A. and W. H. COOKE. Hyperventilation before Resistance Exercise: Cerebral Hemodynamics and Orthostasis. Med. Sci. Sports Exerc., Vol. 39, No. 8, pp. 1302–1307, 2007. Hyperventilation performed by athletes during preparation for resistance exercise might contribute to reports of postexercise orthostatic instability. Purpose: To test the hypothesis that post–resistance exercise orthostatic instability is associated with exaggerated reductions of cerebral blood-flow velocity after hyperventilation. Methods: We recorded the ECG, end-tidal CO₂, beat-by-beat finger arterial pressure, and cerebral blood-flow velocity in 10 healthy subjects. Subjects performed 10 repetitions of recumbent leg press using resistance equivalent to 80% of their six-repetition maximum during three separate trials (randomized): 1) no prior hyperventilation (NOHV); 2) after hyperventilation to an end-tidal CO₂ of 3% (HV3%); and 3) after hyperventilation to an end-tidal CO₂ of 2% (HV2%). After exercise, subjects stood upright for 10 s and rated symptoms of lightheadedness on a scale of 1 (none) to 5 (faint). Results: Mean cerebral blood-flow velocity (CBFV\textsubscript{MEAN}) increased by 12% during exercise after NOHV and decreased by 14 and 25% during exercise after HV3% and HV2% (all \(P < 0.0001\)). During standing, mean arterial pressure (MAP) decreased by 96 mm Hg and CBFV\textsubscript{MEAN} decreased by 41 cm/s (pooled across conditions; all \(P < 0.0001\)). Absolute reductions of CBFV\textsubscript{MEAN} during standing were greater after HV2% compared with both NOHV and HV3% (\(P = 0.003\)). Ratings of perceived lightheadedness during standing increased with prior hyperventilation (\(P = 0.02\)) and correlated to the magnitude of reductions in MAP (\(r = 0.51; \ P = 0.003\)) and CBFV\textsubscript{MEAN} (\(r = 0.37; \ P = 0.04\)). Conclusions: Hyperventilation before lower-body resistance exercise exacerbates CBFV\textsubscript{MEAN} reductions during standing. Increased symptoms of orthostatic instability are associated with the magnitude of reductions in both MAP and CBFV\textsubscript{MEAN}. Key Words: STRENGTH TRAINING, CEREBRAL BLOOD-FLOW VELOCITY, ORTHOSTATIC TOLERANCE, CEREBRAL AUTOREGULATION

Under quiet, resting conditions, cerebral blood flow is maintained constant through autoregulation and is not dependent on modulation by systemic arterial pressure (13). During mild cycle ergometry exercise, blood pressure increases but remains within the cerebral autoregulatory range, resulting in unchanged cerebral blood flow (6). However, during more intense cycle ergometry exercise, arterial pressure and pulse pressure increase substantially, along with cerebral blood-flow velocity (11). The effects of exercise intensity on cerebral blood-flow velocity during resistance exercise are less clear. When arterial pressures increase moderately during the double leg press, mean cerebral blood-flow velocity (CBFV\textsubscript{MEAN}) is unchanged (4). However, arterial pressures on the order of 450/380 mm Hg have been reported during high-intensity weight lifting (10), and a direct linear association between the amount of weight lifted and the reduction of cerebral blood-flow velocity has been demonstrated (3). Changes in cerebral blood-flow velocity during resistance exercise could be associated with a number of factors, including body position, the potential for Valsalva straining, or changes in arterial CO₂ content (4, 8). Cerebral blood-flow velocity is unchanged during isometric handgrip exercise in subjects who maintain eucapnic ventilation, but it decreases in subjects who hyperventilate during exercise (8). Reductions of cerebral blood-flow velocity during resistance exercise may put weight lifters at risk for postexercise lightheadedness and orthostatic instability. Because hypocapnia constricts cerebral vessels (13), symptoms of dizziness or outright “weight lifters’ blackout” could be linked to the degree of hyperventilation performed before resistance exercise (1).

Although anecdotal evidence suggests that immediate postexercise orthostatic instability experienced by some athletes may be related to a preexercise routine that includes voluntary hyperventilation (1, 4), this notion has not been tested experimentally. Therefore, the purpose of this study was to test the hypothesis that hypocapnia induced with hyperventilation before heavy lower-body resistive exercise exacerbates reductions of CBFV\textsubscript{MEAN} and increases acute symptoms of orthostatic instability.
METHODS

Subjects. Before the recruitment of subjects, a power analysis was run to estimate the minimum number of subjects required to test our hypothesis. Recent unpublished data have shown that subjects who experience orthostatic instability when they stand up after a period of sustained squatting on their heels (squat-stand test) decrease their CBFVMEAN about 10 cm s⁻¹ more than subjects who do not experience orthostatic instability (V.A. Convertino, personal communication, 2006). According to this observation, we chose to estimate an appropriate sample size for the current study on the basis of the magnitude of expected change in CBFVMEAN during standing after leg-press exercise as our continuous variable. For CBFVMEAN, we used a Student’s r-based algorithm to estimate that a sample size of N = 9 would allow a true least difference of interest of 10 cm s⁻¹ with an expected standard deviation of 5 cm s⁻¹ to be detected by a two-tailed test P < 0.05 with 80% power (15).

Five males and five females (age 26 ± 4.4 yr; height 170 ± 6.7 cm; weight 72 ± 8.9 kg; means ± SD) volunteered to participate in the study. Subjects were all experienced weight trainers and were currently participating in an active weight-training program. Because of the potential influences on autonomic blood pressure regulation, subjects abstained from exercise, caffeine, and alcohol at least 12 h before experimentation. This research study was approved by the institutional review board of the University of Texas at San Antonio, and written informed consent was obtained from all subjects before experimentation.

Study protocol. Before testing, subjects first performed sets of six repetitions with progressively increasing resistance on a recumbent leg-press machine (Cybex, Medway, MA) to establish their six-repetition maximum (6RM). Eighty-five percent of a subject’s 6RM was taken as the subject’s estimated 10-repetition maximum (10RM).

Subjects were instrumented with an ECG to record cardiac electric potentials (Harvard Apparatus, Holliston, MA), a finger plethysmography device to record beat-by-beat finger arterial pressure (Finometer, Finapres Medical Systems, Arnhem, the Netherlands), a facemask connected to a sampling line to assess end-tidal CO₂, Systems, Arnhem, the Netherlands), a facemask connected to a sampling line to assess end-tidal CO₂ (MulTi Dop T, DWL Electronics, Sipplingen, Germany).

Subjects rested quietly on the recumbent leg-press device for 1 min to establish preexercise (baseline) values; then, they performed three sets of exercise at their calculated 10RM on three different occasions (in randomized order): 1) no prior hyperventilation (NOHV); 2) after voluntary hyperventilation to an end-tidal CO₂ of 3% (HV3%); and 3) after voluntary hyperventilation to an end-tidal CO₂ of 2% (HV2%). Subjects were positioned so that they could not see the output from the CO₂ meter, and they were prompted to begin exercise by one of the investigators when end-tidal CO₂ reached the required level. Subjects were instructed to avoid Valsalva straining during exercise, and lack of Valsalva straining was confirmed with breath-by-breath monitoring of CO₂. After each set of exercise, subjects stood immediately upright for approximately 10 s and rated their symptoms of lightheadedness (if any) using the rating scale of Convertino et al. (2). The nomogram used by subjects to rate symptoms was scaled as follows: 1) none, 2) mild, 3) moderate, 4) severe, and 5) faint. After rating any symptoms, subjects returned to the recumbent position in the leg-press device and rested for 1 min before performing another set.

Data acquisition and analysis. Data were sampled at 500 Hz and recorded directly to computer with commercial hardware and software (WINDAQ, Dataq Instruments, Akron, OH). Data were then imported into a commercial analysis program (WinCPRS, Absolute Aliens, Turku, Finland). R waves generated from the ECG signal were detected automatically, and then the accuracy of the computer detection algorithm was checked manually for errors and edited as necessary. Diastolic (DAP) and systolic (SAP) pressures were subsequently marked automatically from the Finometer and Doppler tracings and edited as necessary. CBFVMEAN was calculated as a true average of each integrated waveform. We calculated a pulsatility index as an estimate of cerebral vascular resistance by dividing mean arterial pressure by CBFVMEAN. Increases and decreases in the pulsatility index are associated with cerebral-vessel constriction and dilation (5,18).

Statistical analysis. Data were analyzed with commercial statistical software (SAS Institute, Cary, NC). We performed a repeated-measures analysis of variance to explore the magnitude of differences among the means of our dependent variables of interest. Differences between trials were probed further with Duncan’s post hoc mean separation procedure. Exact P values were calculated to reflect the probability that the observed response represented chance effects, given random sampling variability (9). Data are presented as means ± SE unless specified otherwise.

RESULTS

Hyperventilation. End-tidal CO₂ during baseline before exercise was 4.5 ± 0.1%. Verbal encouragement for subjects to breathe more deeply and quickly was a successful strategy to ensure close matching of end-tidal CO₂ to experimental targets. End-tidal CO₂ decreased to 2.9 ± 0.2% for the HV3% trial and decreased to 1.8 ± 0.3% for the HV2% trial.

Responses during exercise. HR increased compared with baseline for all conditions, and HR was higher for HV2% compared with NOHV but not compared with HV3%. Arterial pressures increased compared with baseline.
for all conditions. SAP was higher for NOHV but was not different compared with HV3% and HV2%. DAP and MAP were higher during exercise for NOHV compared with both HV3% and HV2%. CBFV_MEAN increased compared with baseline during exercise with NOHV, and it decreased compared with both baseline and NOHV during HV3% and HV2%. The pulsatility index was unaffected compared with baseline for NOHV, but it increased during both HV3% and HV2% compared with baseline and NOHV. End-tidal CO₂ was reduced during exercise for both HV3% and HV2% compared with baseline and NOHV (Table 1). CBFV_MEAN and MAP responses to exercise are presented graphically in Figure 1.

**Responses during standing.** The effects of prior hyperventilation on CBFV_MEAN and MAP during exercise and standing are shown for one subject in Figure 2. Oscillations of both pressure and flow velocity are apparent during each repetition, and the reductions in pressure and flow velocity are pronounced during standing.

HR during standing was 120 ± 2.3 (NOHV), 122 ± 2.2 (HV3%), and 122 ± 2.7 (HV2%) \((P = 0.47)\). During recovery after exercise and standing, HR was 115 ± 3.6 (NOHV), 119 ± 2.6 (HV3%), and 120 ± 3.4 (HV2%) \((P = 0.12)\). The absolute CBFV_MEAN minimal values recorded during standing are shown with MAP in Figure 3.

**Symptoms of orthostatic instability.** Feelings of lightheadedness, tunnel vision, or other feelings of orthostatic instability were greater \((P = 0.02)\) for HV2% \((2.1 ± 0.31)\) than for HV3% \((1.7 ± 0.26)\) and NOHV \((1.5 ± 0.16)\). Correlation analysis across all three exercise conditions \((N = 30\) observations\) revealed associations among symptoms of orthostatic instability and both the magnitude of reduction during standing of MAP \((r = 0.51; P = 0.003)\) and CBFV_MEAN \((r = 0.37; P = 0.039)\). According to the nomogram developed by Convertino et al. (2), symptoms across all conditions were mild. No subjects reported serious presyncopal symptoms above a 3.0 (moderate).

**DISCUSSION**

We studied the influence of hypocapnia induced with hyperventilation before lower-body resistance exercise on cerebral blood-flow velocity and symptoms of orthostatic instability. The primary new findings from this study are that 1) CBFV_MEAN increases during leg-press exercise without prior hyperventilation but decreases with prior hyperventilation, 2) CBFV_MEAN and arterial pressures are reduced more when subjects stand after leg-press exercise with prior hyperventilation than without, and 3) symptoms of orthostatic instability are associated with the magnitude of reductions in both MAP and CBFV_MEAN during standing after exercise. We conclude that voluntary hyperventilation before lower-body resistance exercise could contribute to orthostatic instability or even frank syncope by decreasing cerebral blood-flow velocity both during exercise and on standing after exercise.

**Responses during exercise.** Arterial pressures on the order of 450/380 mm Hg have been reported during

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**TABLE 1. Responses to leg-press resistive exercise.**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>NOHV</th>
<th>HV3%</th>
<th>HV2%</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>83 ± 3.7</td>
<td>115 ± 3.6*</td>
<td>119 ± 2.7</td>
<td>121 ± 3.4†</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>131 ± 2.4</td>
<td>180 ± 9.0*</td>
<td>177 ± 8.2*</td>
<td>166 ± 4.8*</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>72 ± 4.7</td>
<td>100 ± 3.3‡</td>
<td>96 ± 3.3*</td>
<td>91 ± 4.7†</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>95 ± 2.6</td>
<td>133 ± 5.5‡</td>
<td>129 ± 3.5*</td>
<td>121 ± 3.4†</td>
</tr>
<tr>
<td>PI</td>
<td>1.5 ± 0.1</td>
<td>1.8 ± 0.2</td>
<td>2.4 ± 0.2**</td>
<td>2.5 ± 0.1**</td>
</tr>
<tr>
<td>End-tidal CO₂ (%)</td>
<td>4.5 ± 0.1</td>
<td>4.4 ± 0.2</td>
<td>3.7 ± 0.1**</td>
<td>3.4 ± 0.1**</td>
</tr>
</tbody>
</table>

Values are means ± SE. NOHV, no prior hyperventilation; HV3%, end-tidal CO₂ of 3% after hyperventilation; HV2%, end-tidal CO₂ of 2% after hyperventilation; HR, heart rate; SAP, systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure; PI, pulsatility index. *P < 0.05 compared with baseline; †P < 0.05 compared with both baseline and NOHV; ‡P < 0.05 compared with baseline, NOHV, and HV3%; ‡P < 0.05 compared with baseline, HV3%, and HV2%.
leg-press exercise (10). Such dramatic increases have been attributed in part to weight lifters performing Valsalva maneuvers (3). In the present study, we instructed our subjects to avoid Valsalva straining, and we confirmed that they continued to breathe during lifting by monitoring breath-by-breath end-tidal CO$_2$. Arterial pressures increased to 180/100 mm Hg with no hyperventilation, and to 177/95 mm Hg (HV3%) and 166/91 mm Hg (HV2%) with hyperventilation. Although not as dramatic as pressures reached during maximal lifting (10), increases of arterial pressure during leg-press exercise in the current study were higher than the 140/50 mm Hg thought to define normal cerebral autoregulatory ranges (13).

Both arterial pressure and CBFV MEAN oscillated with each repetition, as shown in Figure 2. Similar results have been reported by Pott et al. (14) during rowing and by Edwards et al. (4) during double-leg press. Cerebral vessels autoregulate effectively with a response time of about 3–5 s (16), which is probably slower than our subjects performed their repetitions. Figure 2 is representative of all subjects with respect to the speed at which they completed their sets. Although we did not document repetition time, it seems from the subject depicted in Figure 2 that each repetition averaged about 2 s. In the study by Edwards et al. (4); in the current study, it increased by about 40%, from 95 to 133 mm Hg. This observation may help to explain disparate results; an MAP of 133 mm Hg (and SAP and DAP of 180 and 100 mm Hg) moves the cardiovascular system beyond the cerebral autoregulatory range.

Hypocapnia induced by hyperventilation to either 3 or 2% end-tidal CO$_2$ caused CBFV MEAN to decrease compared with both baseline and exercise with no prior hyperventilation (Fig. 1). Reduced CBFV MEAN occurred in conjunction with increases in cerebrovascular resistance (Table 1), as estimated from the pulsatility index (18). Arterial pressures were higher than at baseline during hypocapnic exercise, but CBFV MEAN was lower than at baseline. This observation supports the concept that reductions in velocity are likely attributable to vasoconstriction and are not simply reductions in cerebral perfusion pressure. The vasoactive effects of arterial CO$_2$ are well established, with reductions causing cerebral-vessel constriction and reduced flow velocity (7). Cerebral blood-flow velocity decreases by about 3.5% for every change in end-tidal CO$_2$ of 1 mm Hg (7). Although we recorded CO$_2$ as a percentage, conversion to millimeters of mercury reveals that CO$_2$ decreased from about 34 mm Hg (NOHV) to about 26 mm Hg (HV2%), which should have resulted in a difference between the two conditions of about 30%. CBFV MEAN decreased from 73 to 52 cm s$^{-1}$ (about 39%; data shown graphically in Figure 1), consistent with predictions. Although end-tidal CO$_2$ is an imperfect predictor of arterial
CO₂ (17), it is reasonable to propose that reductions of CBFV为主要原因 during lower-body resistance exercise after hyperventilation are associated with reductions of arterial CO₂. In conjunction with reduced CBFV的主要原因, both SAP and DAP were lower during exercise for HV3% and HV2% compared with NOHV. Reduction of CO₂ with hypocapnia causes vasoconstriction in cerebral arteries and vasodilation in systemic arteries (12). The vasodilatory effects of reduced CO₂ in systemic arteries likely underlie our observation of reduced DAP and MAP during exercise after hyperventilation compared with NOHV.

Responses during standing. Edwards et al. (4) have observed reductions of cerebral blood-flow velocity at the conclusion of 10 repetitions of leg-press exercise. None of the subjects in their study experienced dizziness or other symptoms related to presyncope, but they also remained seated during recovery from exercise. Nevertheless, Edwards et al. (4) propose that the postexercise orthostatic instability experienced by many weight trainers and competitive lifters (1) is probably related to reductions in cerebral blood flow, increased cerebral vascular resistance, reductions in MAP, or some combination thereof. We undertook the current study largely on the basis of the data of Edwards et al. (4), and we found that when subjects stood after exercise with prior hyperventilation, their CBFV为主要原因 fell to levels below those seen during standing after exercise without prior hyperventilation (Fig. 3). On the basis of the representative response to our experimental protocol, as shown in Figure 2, it is apparent that standing after heavy resistance exercise challenges both arterial pressure and cerebrovascular regulatory mechanisms extensively. Such dramatic reductions of pressure and flow velocity likely do explain postexercise orthostatic instability, and the addition of hyperventilation before exercise exacerbates the postexercise reduction of CBFV为主要原因. We also would argue that hypocapnia exacerbates the postexercise reductions of arterial pressure, because differences between HV2% and NOHV deviated from chance effects with a P value of 0.052 (Fig. 3).

We were unable to run a blinded experimental design, because subjects were required to hyperventilate voluntarily. Although we did not reveal to our subjects that we expected them to experience a greater degree of dizziness during standing after hyperventilation, we cannot discount the possibility that this outcome was anticipated. Therefore, we cannot say with certainty that subjects’ knowledge that they were hyperventilating before exercise did not influence their subjective ratings of dizziness. With this limitation, subjects reported feeling more mild lightheadedness after HV2% compared with NOHV and HV3%. This observation is consistent with the CBFV的主要原因 and arterial pressure reductions during standing after hyperventilation. Symptoms correlated with both the magnitude of reductions in CBFV的主要原因 and MAP.

**SUMMARY**

At least one study has documented the voluntary hyperventilation performed by weight lifters as part of a routine to “psych up” for a maximal weight-lifting effort (1). Some athletes who incorporate preexercise hyperventilation experience orthostatic instability or even frank syncope at the conclusion of exercise. We have shown that hypocapnia reduces CBFV的主要原因 during lower-body resistance exercise and exacerbates the fall in postexercise blood pressure and CBFV的主要原因 on standing. The reduced CBFV的主要原因 during exercise seems to be independent of arterial pressure changes, suggesting a direct effect of hypocapnia. Combined, our results suggest that the hypocapnia-induced falls in arterial pressure and CBFV的主要原因 may lead to symptoms of postexercise syncope on standing after lower-body resistance exercise.

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