Hemodynamic Response to Upright Resistance Exercise: Effect of Load and Repetition

BLAKE G. PERRY1, ZACHARY J. SCHLADER1, MATTHEW J. BARNES1, DARRYL J. COCHRANE1, SAMUEL J. E. LUCAS2,3, and TOBY MÜNDEL1

1School of Sport and Exercise, Massey University, Palmerston North, NEW ZEALAND; 2Department of Physiology, University of Otago, Dunedin, NEW ZEALAND; and 3School of Sport, Exercise and Rehabilitation, University of Birmingham, Birmingham, England, UNITED KINGDOM

ABSTRACT

PERRY, B. G., Z. J. SCHLADER, M. J. BARNES, D. J. COCHRANE, S. J. E. LUCAS, AND T. MÜNDEL. Hemodynamic Response to Upright Resistance Exercise: Effect of Load and Repetition. Med. Sci. Sports Exerc., Vol. 46, No. 3, pp. 479–487, 2014. Introduction/ Purpose: Upright resistance exercise causes large transient fluctuations in blood pressure during and immediately after the performance. We examined the effect of resistance load and the number of repetitions on the middle cerebral artery blood flow velocity (MCAv) response during and after upright squatting exercise. Methods: Healthy males (n = 12; mean ± SD: 26 ± 5 yr) completed 30%, 60%, and 90% of their six-repetition maximum load, completing two and six repetitions of these loads during two visits (order randomized). Beat-to-beat MCAv, blood pressure, and continuous end-tidal PCO2 during exercise, at nadir, and during recovery are reported as the change from preexercise standing baseline. Results: During exercise, MCAvmean increased 31% ± 16% (P < 0.001) across all loads (P = 0.74) and repetitions (P = 0.89), whereas mean arterial pressure (MAP) increased (all P < 0.05) as load and repetitions increased (e.g., 122 ± 9 (two repetitions) vs 135 ± 11 mm Hg (six repetitions) and 128 ± 13 vs 143 ± 14 mm Hg, at 30% and 60%, respectively). Within the six-repetition sets, peak MCAvmean remained unchanged across the set (P = 0.61), whereas MAP increased (P = 0.003). The 90% load produced the lowest MCAvmean nadir (pooled means, −34 ± 7 and −43 ± 5 mm Hg, for two and six repetitions, respectively; P < 0.001) after exercise. Postexercise MCAvmean reductions occurred via a selective, load-dependent (P < 0.001) decrease in diastolic MCAv. MCAvmean remained below baseline for the longest period after the 90% six-repetition set (10 s postexercise, P < 0.01) and took the longest to recover (14.8 ± 6.9 s, P = 0.002). Conclusion: These data indicate that higher relative loads produce a greater postexercise hypotension and result in a proportionate reduction in MCAvmean. Key Words: RESISTANCE EXERCISE, LOAD, REPETITION, CEREBRAL HYPOPERFUSION, VALSALVA MANEUVER

Resistance training is a common mode of exercise in both recreational and professional settings because of its positive effects on muscular strength, cardiovascular function, metabolism, and psychological well-being (7). Furthermore, many clinical populations, including those with cardiovascular disease, are now participating in resistance-type training (21). However, resistance exercise can produce extremely high blood pressures, with systolic and diastolic values as high as 480 and 350 mm Hg, respectively, being reported (15). Paradoxically, hypotension and cerebral hypoperfusion has also been observed after heavy, upright resistance exercise (4,17,27). However, to date, no research has examined the initial postexercise blood pressure response to upright resistance exercise and how this subsequently affects middle cerebral artery blood flow velocity (MCAv).

To date, studies investigating the hemodynamic response during resistance exercise have used a leg-press–type movement (17,27), static/isometric-type exercise (16,20), or both (14). Squatting exercise, with additional load, likely poses a further challenge via its orthostatic component, as, for example, body weight squats have been found to significantly challenge dynamic cerebrovascular control (3). In fact, standing alone causes a 15% reduction in MCAv (23). Thus,
the upright posture may put individuals at greater risk of cerebral hypoperfusion after load-bearing squatting-type movements because of large reductions in mean arterial pressure (MAP). However, the effect of load lifted and/or the number of repetitions on the postexercise hypotension and the subsequent influence of this reduction in cerebral perfusion pressure on cerebral blood flow velocity have not been examined to date.

Therefore, the purpose of this study was to investigate the influence of the load and number of repetitions on the cardio- and cerebrovascular responses during repeated squatting exercise, specifically, the influence of the load lifted on the postexercise arterial blood pressure response after squatting exercise and how this subsequently affects cerebral blood flow (indexed via velocity measures). The primary working hypothesis for this study was that greater loads will induce a greater postexercise hypotension that would result in a greater pressure passive reduction in MCAv.

METHODS

Twelve healthy, resistance-trained males were recruited for the study (mean ± SD: age, 26 ± 5 yr; body mass, 94 ± 13 kg; height, 184 ± 8 cm; resistance training age, 4.3 ± 3.0 yr). Each participant was fully informed of all potential risks and experimental procedures, after which, informed written consent was obtained. All experimental procedures and protocols were approved by the Massey University Human Ethics Committee and performed in accordance with the Declaration of Helsinki. All participants were free from cardiovascular (including orthostatic hypotension and recurrent syncope) and cerebrovascular disease and were not taking medication. Participants arrived at the laboratory for the familiarization, and both experimental trials abstained from strenuous exercise, alcohol, caffeine, and nicotine for at least 24 h.

Study design. Participants visited the laboratory on three occasions: one occasion for familiarization and two for experimental trials at the same time of day. During the familiarization session, the participants were familiarized with all experimental procedures, including ideal squatting technique, and their six-repetition maximum (6RM) was determined. The squatting exercise was conducted on a fixed barbell path (Smith) machine (FitnessWorks, New Zealand) with the participants wearing an adjustable front squat harness (Getstrength.com, New Zealand), which was fitted over the shoulders and rested on the chest and abdomen. This harness included two outward projecting metal pins that were located just inferior to each clavicle and were used to support the Smith machine barbell (Fig. 1). Collectively, the harness and Smith machine allowed for the squats to be executed without the participants holding the barbell (left hand free, refer to Fig. 1), allowing for the measurement of finger blood pressure (see Measurements). All participants were required to squat to a depth that was equivalent to the point at which the femur was parallel to the floor, which was confirmed via quantification of barbell displacement. Participants were instructed to breathe normally through a custom-made mouthpiece apparatus, which allowed for the measurement of end-tidal expiratory and facilitated a Valsalva maneuver (VM), if required. Participants were instructed to only perform this modified VM if they saw fit and to maintain normal ventilation otherwise. When a VM was required, participants exhaled forcefully through the mouth, which temporarily closed a valve in the mouthpiece apparatus to allow for the VM to be completed during the squat. Mouth pressure served as a surrogate for intrathoracic pressure (15, 18) and accurately reflects changes in esophageal pressure (surrogate for intrathoracic pressure) in a variety of postures (6). All participants were instructed to avoid hyperventilation immediately preceding each set.

Experimental protocol. The experimental design and protocol is shown in Figure 2. The two experimental trials were randomized with one trial consisting of two repetitions and the other of six repetitions of 30%, 60%, and 90% of the 6RM load. The order in which each load was lifted within each trial was also randomized. First, participants stood for 2 min during which baseline measures were obtained. This was followed by body weight squats being performed, the number of which was equal to the number of squats performed during the following work set. This allowed for the randomization of all loads and excluded the possibility of participants having to complete the 90% set without a warm-up. A stable baseline period was established and recorded before each work set. During each work set, verbal confirmation of adequate squat depth was given. Participants were instructed to maintain a 2-s down, 2-s up pace during all sets. After completing all sets, participants were instructed to stand for 2 min as still as possible and to avoid muscle tensing, because this has been previously shown to restore circulatory stability (13, 35).

Measurements. Blood flow velocity in the right middle cerebral artery (MCAv) was measured using a 2-MHz pulsed Doppler ultrasound system (DWL; Compumedics Ltd., Germany) using search techniques described elsewhere (1). The Doppler probe was secured with a plastic headband (DWL) once an optimum signal-to-noise ratio had been achieved. This maintained a constant insonation angle throughout the protocol. The day-to-day intraobserver coefficient of variation for MCAv measures was 4% (n = 8, determined across 4 d). Participants breathed through the aforementioned modified mouthpiece, which allowed both the measurement of mouth pressure (substitute for intrathoracic pressure) and the partial pressure of end-tidal CO₂ (PETCO₂, gas analyzer model ML206; ADInstruments, Colorado Springs, CO). Blood pressure was measured noninvasively using finger photoplethysmography (Finapres Medical Systems; Biomedical Instruments, Amsterdam, the Netherlands), and heart rate was measured via three-lead electrocardiogram (ADInstruments). Finometer blood pressure values were checked against a manual sphygmomanometer initially and regularly during rest periods.
Squatting depth, via barbell displacement, was recorded using a potentiometer (Model 533; Vishay, Malvern, PA) mounted to the upper cog of the Smith machine. All data were acquired continuously via an analogue-to-digital converter (PowerLab ML870, ADInstruments) at 1 kHz. Data were displayed in real time and recorded for off-line analysis using commercially available Lab Chart software (v7.3.3, ADInstruments).

Cardiac output $\dot{Q}$ was calculated from the blood pressure waveform using the Modelflow method (36), which incorporates age, sex, height, and weight (BeatScope 1.02 software, Biomedical Instruments). Mean MCAv (MCAvmean) was calculated as the integral for each cardiac cycle divided by the corresponding pulse interval. An index of total peripheral resistance (TPRi) was estimated using the equation $\frac{MAP}{\dot{Q}}$ and cerebrovascular conductance (CVC) via the equation $\frac{MCAv_{\text{mean}}}{MAP}$. Relative to baseline measures, the percentage decrease in MCAvmean was divided by the percentage reduction in MAP at MCAvmean nadir to assess differences in the MAP contribution to the MCAvmean reduction.

**Data analysis.** Baseline data were acquired in the second minute of each passive stand between sets and presented as the mean across that minute. All variables upon the attainment of the highest MCAvmean (peak) during each repetition within a set were recorded, and the average peak values were used in the analysis for exercise. After each set, i.e., after racking the barbell (time = 0, as in Ref. (30)), time to nadir, recovery, and peak values for MCAvmean and MAP were used in the postexercise analysis. Nadir was defined as the lowest measured value immediately after the completion of the set, recovery as the point when the variable returned to baseline after the nadir, and peak as the maximum point after the racking of the barbell and before the subsequent seated rest. In addition, the area under the curve (AUC) for data after exercise (from time = 0 to 15 s post exercise) was calculated as previously described (25).

**Statistical analysis.** All dependent variables were analyzed using a two-way (repetition × load, 2 × 3) repeated measures ANOVA. Data within the six-repetition trials were analyzed using a load by time (2 × 6) repeated-measures ANOVA. Data for the 30-s period immediately postexercise within each repetition trial was compared with the preexercise
baseline using an ANOVA for repeated measures of time and load. Data were assessed for approximation to a normal distribution and sphericity with no corrections required. Main effects were isolated using post hoc pairwise comparisons (Bonferroni corrected, where necessary). All data were analyzed using SPSS statistical software (v18; SPSS, Chicago, IL) with a priori statistical significance set at \( P < 0.05 \). All data are presented as the mean \( \pm \) SD absolute and/or relative change from the baseline preceding the respective work set, unless denoted otherwise.

**RESULTS**

**Hemodynamic responses during upright resistance exercise.** Average peak MCA\(_{\text{mean}}\) values were elevated 31\% \pm 16\% from baseline \( (P < 0.001) \), and this was consistent between loads \( (P = 0.74) \) and repetitions \( (P = 0.89) \). For the six-repetition sets, peak MCA\(_{\text{mean}}\) remained unchanged across all repetitions \( (P = 0.61) \), despite MAP increasing as the set progressed in both the 30\% and 60\% sets \( (P = 0.003) \). Heart rate was higher at greater loads \( (P = 0.001) \) and repetitions \( (P < 0.001) \): specifically, 98 \pm 4 versus 108 \pm 4 beats per minute for two and six repetitions at 30\%, respectively; 106 \pm 4 versus 117 \pm 4 beats per minute for two and six repetitions at 60\%, respectively; and 112 \pm 5 versus 129 \pm 3 beats per minute for two and six repetitions at 90\%, respectively. Because of loss of the blood pressure trace, only 30\% and 60\% loads were compared \( (n = 6) \), because participants could not refrain from placing pressure on the finger used for the measurement of blood pressure while lifting the 90\% load, resulting in erroneous data at this workload in nine participants. When peak values for a repetition were not suitable, the data were excluded from the exercise (only) values. When applicable, the physiological (Finometer automatic calibration) procedure was then performed to allow the use of MAP data (and therefore \( Q \) and TPR\(_{\text{i}}\)) after exercise. Exercise-induced increases in MAP were greater during the 60\% compared with the 30\% set \( (P = 0.015) \) and during six repetitions \( (P = 0.002 \text{ vs two repetitions}) \): specifically, 122 \pm 9 versus 135 \pm 11 mm Hg for two and six repetitions at 30\%, respectively; and 128 \pm 13 versus 143 \pm 14 mm Hg for two and six repetitions at 60\%, respectively (Fig. 3). In three participants, a satisfactory blood pressure trace for both 90\% sets was acquired for all repetitions with an average peak \( (\pm \text{SD}) \) of 150 \pm 2 and 176 \pm 6 mm Hg for two and six repetitions, respectively. Systolic blood pressure was dependent on the number of repetitions \( (P = 0.001) \) but not intensity \( (P = 0.49) \) with pressures during the 60\% set \( (169 \pm 19 \text{ and } 194 \pm 30 \text{ mm Hg for two and six repetitions, respectively}) \) and during the 30\% load \( (168 \pm 15 \text{ and } 189 \pm 18 \text{ mm Hg for two and six repetitions, respectively}) \). Diastolic blood pressures were dependent upon the number of repetitions \( (P = 0.012) \) and also on load \( (P = 0.033) \), with pressures during the 60\% work load

![Figure 3](http://www.acsm-msse.org)
TABLE 1. Changes from baseline at MCAv nadir for 30%, 60%, and 90% 6RM loads.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Repetitions</th>
<th>30</th>
<th>30</th>
<th>60</th>
<th>60</th>
<th>90</th>
<th>90</th>
<th>Load</th>
<th>Reps</th>
<th>R x L</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCAv mean (cm s⁻¹)</td>
<td>57 ± 10</td>
<td>2</td>
<td>-8 ± 7</td>
<td>-11 ± 8</td>
<td>-18 ± 6</td>
<td>-8 ± 7</td>
<td>2</td>
<td>-11 ± 8</td>
<td>-18 ± 6</td>
<td>0.001</td>
<td>0.016</td>
</tr>
<tr>
<td>Systolic MCAv (cm s⁻¹)</td>
<td>94 ± 16</td>
<td>6</td>
<td>-13 ± 6</td>
<td>-19 ± 8</td>
<td>-21 ± 6</td>
<td>-32 ± 6</td>
<td>10</td>
<td>-19 ± 8</td>
<td>-21 ± 11</td>
<td>0.84</td>
<td>0.10</td>
</tr>
<tr>
<td>Diastolic MCAv (cm s⁻¹)</td>
<td>42 ± 8</td>
<td>2</td>
<td>16 ± 8</td>
<td>19 ± 12</td>
<td>21 ± 11</td>
<td>14 ± 12</td>
<td>9</td>
<td>19 ± 12</td>
<td>21 ± 11</td>
<td>0.003</td>
<td>0.041</td>
</tr>
<tr>
<td>CVC (mm Hg cm⁻¹ s⁻¹)</td>
<td>0.61 ± 0.11</td>
<td>6</td>
<td>-19 ± 6</td>
<td>-20 ± 12</td>
<td>-25 ± 8</td>
<td>-19 ± 12</td>
<td>6</td>
<td>-20 ± 12</td>
<td>-25 ± 8</td>
<td>0.035</td>
<td>0.05</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>96 ± 11</td>
<td>2</td>
<td>-18 ± 13</td>
<td>-23 ± 27</td>
<td>-29 ± 15</td>
<td>-23 ± 17</td>
<td>6</td>
<td>-23 ± 27</td>
<td>-29 ± 16</td>
<td>0.007</td>
<td>0.55</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>137 ± 15</td>
<td>6</td>
<td>-13 ± 24</td>
<td>-11 ± 27</td>
<td>-53 ± 14</td>
<td>-11 ± 27</td>
<td>12</td>
<td>-11 ± 27</td>
<td>-53 ± 14</td>
<td>0.001</td>
<td>0.30</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>75 ± 11</td>
<td>2</td>
<td>-16 ± 5</td>
<td>-19 ± 11</td>
<td>-29 ± 15</td>
<td>-16 ± 5</td>
<td>11</td>
<td>-19 ± 11</td>
<td>-29 ± 15</td>
<td>0.003</td>
<td>-0.001</td>
</tr>
<tr>
<td>HR (beats per minute)</td>
<td>81 ± 14</td>
<td>2</td>
<td>22 ± 5</td>
<td>28 ± 8</td>
<td>31 ± 12</td>
<td>22 ± 5</td>
<td>2</td>
<td>28 ± 8</td>
<td>31 ± 12</td>
<td>0.30</td>
<td>0.37</td>
</tr>
<tr>
<td>PETCO₂ (mm Hg)</td>
<td>32 ± 3</td>
<td>6</td>
<td>-4 ± 6</td>
<td>-3 ± 7</td>
<td>-4 ± 5</td>
<td>-4 ± 6</td>
<td>3</td>
<td>-3 ± 7</td>
<td>-4 ± 5</td>
<td>0.003</td>
<td>-0.001</td>
</tr>
</tbody>
</table>

Values are absolute mean difference from baseline ± SD and percentage change from baseline values (%) denoted in parentheses. For all blood pressure data n = 8.

*Statistically different from 30%, P < 0.05.
**Statistically different from 60%, P < 0.05.
***Statistically different from six repetitions, P < 0.05.
****Trend for a difference between 90% and 60%, P = 0.076.
*****Trend for a difference between 30% and 60%, P = 0.069.

MCAv, middle cerebral artery velocity; BP, blood pressure; HR, heart rate; PETCO₂, partial pressure of end tidal carbon dioxide; Reps, repetitions; R x L, repetitions–load interaction.
Cerebrovascular and cardiorespiratory variables at MCA\textsubscript{mean} nadir. Cerebrovascular and cardiorespiratory data at MCA\textsubscript{mean} nadir during the passive stand are reported in Table 1. Briefly, the 90% load produced the largest reduction in MCA\textsubscript{mean} (for \(P \) values, see Table 1). These differences in MCA\textsubscript{mean} were not mediated by differences in \(P\text{\textsubscript{T}}\text{CO}_2\) but by a greater reduction in diastolic flow velocity at nadir. The 60% load tended to reduce MCA\textsubscript{mean} to a greater degree than the 30% load (\(P = 0.069\)). The magnitude of the MCA\textsubscript{mean} decrease was only significantly different between the number of repetitions at the 30% and 60% relative loads. At MCA\textsubscript{mean} nadir, the reductions in MAP were also load dependent, as were the reductions in both systolic and diastolic blood pressure (for \(P \) values, see Table 1). The ratio of percentage change in MCA\textsubscript{v} versus percentage change in MAP was not significantly different between loads (\(P = 0.11\)) or repetitions (\(P = 0.48\); grouped means: 1.1 \(\pm\) 1.2, 2.6 \(\pm\) 3.4, and 1.1 \(\pm\) 0.6 for 30%, 60%, and 90% load, respectively). Time to MCA\textsubscript{mean} recovery data were pooled into 1-s bins (Fig. 3), an interaction effect was present (\(P < 0.001\)). Post hoc analyses revealed that the period for which MCA\textsubscript{mean} was below baseline after the six-repetition set was 2 s (4–6 s postexercise), 6 s (3–9 s), and 10 s (4–14 s) for 30%, 60%, and 90%, respectively (all \(P < 0.01\)). MCA\textsubscript{mean} nadir was not reliably (\(P > 0.05\)) below baseline for any load after the two-repetition sets using this analysis approach. In support of this, the AUC analysis for MCA\textsubscript{mean} indicated a significant effect of load for six repetitions (\(P = 0.05\), –26 \(\pm\) 97, –96 \(\pm\) 97, –118 \(\pm\) 52 uA for 30%, 60%, and 90% loads, respectively), but not for the two-repetition sets (\(P = 0.50\)). Time to MCA\textsubscript{mean} peak was significantly slower at greater loads (\(P < 0.001\)) and higher repetitions (\(P < 0.001\)).

Time to MAP recovery was delayed by both the load (\(P < 0.001\)) and number of repetitions (\(P = 0.01\)), with the slowest being the 90% two- and six-repetition sets (46.5 \(\pm\) 20.7 and 49.9 \(\pm\) 12.9 s, respectively; load–repetition interaction effect: \(P < 0.01\)). Post hoc analyses revealed that the period below baseline after six-repetition sets was 16 s (6–22 s postexercise), 22 s (8–30 s), and 24 s (6–30 s) for 30%, 60%, and 90%, respectively (all \(P < 0.01\)), whereas the hypotensive response after two-repetition sets was shorter (Fig. 3).

Cerebrovascular and cardiorespiratory variables at MAP nadir. Cerebrovascular and cardiorespiratory data at MAP nadir during the passive stand immediately after the exercise are reported in Table 2 (also see Fig. 3). Briefly, the 90% load caused the greatest reduction in MAP after exercise. MCA\textsubscript{mean} was significantly greater at MAP nadir than MCA\textsubscript{mean} nadir for all loads and repetitions (all \(P < 0.05\)). Overall, MAP nadir occurred later than MCA\textsubscript{mean} nadir for all loads and repetitions (all \(P \leq 0.05\)). Specifically, although time to MCA nadir was similar across the different loads and repetitions (reported previously), MAP nadir was influenced by load and repetitions with a higher percentage of 6RM increasing the time to nadir (\(P = 0.05\), Fig. 3).

Recovery after exercise. MCA\textsubscript{mean} recovery was load dependent (\(P = 0.002\)), with the greatest time to recovery taken after the 90% two- and six-repetition sets (17.5 \(\pm\) 8.7 and 14.8 \(\pm\) 6.9 s, respectively), whereas the number of repetitions did not alter this time course of response (\(P = 0.572\)). When MCA\textsubscript{mean} recovery data were pooled into 1-s bins, an interaction effect was present (\(P < 0.001\)). Post hoc analyses revealed that the period for which MCA\textsubscript{mean} was below baseline after the six-repetition set was 2 s (4–6 s postexercise), 6 s (3–9 s), and 10 s (4–14 s) for 30%, 60%, and 90%, respectively (all \(P < 0.01\)), whereas MCA\textsubscript{mean} was not reliably (\(P > 0.05\)) below baseline for any load after the two-repetition sets using this analysis approach. In support of this, the AUC analysis for MCA\textsubscript{mean} indicated a significant effect of load for six repetitions (\(P = 0.05\), –26 \(\pm\) 97, –96 \(\pm\) 97, –118 \(\pm\) 52 uA for 30%, 60%, and 90% loads, respectively), but not for the two-repetition sets (\(P = 0.50\)). Time to MCA\textsubscript{mean} peak was significantly slower at greater loads (\(P < 0.001\)) and higher repetitions (\(P < 0.001\)).

Values are absolute mean difference from baseline \(\pm\) SD, and percentage change from baseline values \(\pm\) SD are denoted in parentheses.

**Statistically different from 30%, \(P < 0.05\).
** Statistically different from 60%, \(P < 0.05\).
** Statistically different from six repetitions, \(P \leq 0.05\).

Reps: repetitions; \(R \times L\); repetitions–load interaction.

TABLE 2. Hemodynamic changes from baseline at MAP nadir for 30%, 60% and 90% 6RM loads.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Repetitions</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>(\Delta) From Baseline</th>
<th>Load</th>
<th>Reps</th>
<th>(R \times L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA\textsubscript{mean}, cm\textsuperscript{3} s\textsuperscript{-1}</td>
<td>57 ± 10</td>
<td>2</td>
<td>–5 ± 8 (–8 ± 10)</td>
<td>–5 ± 6 (–8 ± 10)</td>
<td>–5 ± 6 (–8 ± 10)</td>
<td>0.047</td>
<td>0.46</td>
<td>0.071</td>
<td></td>
</tr>
<tr>
<td></td>
<td>94 ± 16</td>
<td>6</td>
<td>–9 ± 9 (–16 ± 16)</td>
<td>–10 ± 9 (–16 ± 14)</td>
<td>–16 ± 8 (–15 ± 6)*</td>
<td>0.038</td>
<td>0.97</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td></td>
<td>42 ± 8</td>
<td>2</td>
<td>17 ± 8 (20 ± 9)</td>
<td>25 ± 8 (28 ± 9)</td>
<td>20 ± 11 (21 ± 13)</td>
<td>0.59</td>
<td>0.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6 ± 8</td>
<td>6</td>
<td>24 ± 13 (26 ± 14)</td>
<td>28 ± 11 (30 ± 10)</td>
<td>30 ± 9 (33 ± 13)</td>
<td>0.05</td>
<td>0.001 &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic MCA\textsubscript{v}, cm\textsuperscript{3} s\textsuperscript{-1}</td>
<td>96 ± 11</td>
<td>2</td>
<td>–18 ± 11 (–36 ± 15)</td>
<td>–12 ± 6 (–28 ± 12)</td>
<td>–20 ± 9 (–45 ± 15)</td>
<td>0.059</td>
<td>0.027 0.029 0.063</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6 ± 12</td>
<td>6</td>
<td>–16 ± 9 (–38 ± 23)</td>
<td>–20 ± 20 (–42 ± 43)</td>
<td>19 ± 7 (–44 ± 10)</td>
<td>0.005</td>
<td>0.001 &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>18 ± 11</td>
<td>2</td>
<td>–21 ± 12 (–22 ± 11)*</td>
<td>–34 ± 7 (–34 ± 7)*</td>
<td>–34 ± 7 (–34 ± 7)*</td>
<td>0.005</td>
<td>0.001 &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>21 ± 9</td>
<td>6</td>
<td>–29 ± 8 (–28 ± 9)*</td>
<td>–43 ± 5 (–43 ± 5)*</td>
<td>–43 ± 5 (–43 ± 5)*</td>
<td>0.005</td>
<td>0.001 &lt;0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVC, cm\textsuperscript{-1}</td>
<td>0.61 ± 0.11</td>
<td>2</td>
<td>0.080 ± 0.072 (13 ± 10)</td>
<td>0.10 ± 0.091 (16 ± 12)*</td>
<td>0.069 ± 0.12 (11 ± 10)*</td>
<td>0.027</td>
<td>0.029 0.029 0.063</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.026 ± 0.13 (6 ± 20)</td>
<td>6</td>
<td>0.069 ± 0.11 (12 ± 16)*</td>
<td>0.22 ± 0.08 (44 ± 18)*</td>
<td>0.22 ± 0.08 (44 ± 18)*</td>
<td>0.027</td>
<td>0.029 0.029 0.063</td>
<td></td>
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DISCUSSION

The main findings from this study were as follows: 1) The MCAvmean response during upright squatting resistance exercise was similar regardless of load and number of repetitions, whereas 2) blood pressure increased progressively with increased exercise load and repetition. 3) After upright squatting exercise, the reduction in MCAvmean was mediated largely by reductions in diastolic MCAv and was dependent on the load lifted and number of repetitions. Because MCAvmean was unchanged between exercise loads, despite the prevailing increasing MAP, regulatory mechanisms (e.g., myogenic and/or mechanical) appear to adequately account for these increases in perfusion pressure. However, immediately after repeated upright squatting exercise, the cerebral vasculature alone was unable to actively counteract the rapid reduction in MAP such that proportionate reductions in MCAv ensued.

MAP response during upright resistance exercise. At rest, the MCAvmean response to MAP oscillations is dominated by the relationship between the change in MAP and the change in time (32), and it appears to extend to exercise. During resistance exercise (5,27) and rowing (22), MAP perturbations are too rapid to be counteracted by dynamic cerebrovascular control, because the brain’s vasculature is more successful at dampening low-frequency MAP oscillations (37). Our observations of increased MCAvmean from baseline during the exercise is consistent with these previous reports (27). Interestingly, however, there was no difference in the magnitude of the increase between loads or repetitions despite the load-dependent nature of the MAP response. Furthermore, within the six-repetition sets, peak MCAvmean remained unchanged across the set, again, despite the increasing MAP as the set progressed.

The restraint of MCAv during resistance exercise. Animal models propose a graded sympathetic response to acute hypertension that may restrain cerebral blood flow (2). If this protective mechanism extends to humans, one would expect a constriction of vessels downstream of the MCA and a reduction (or a restricted increase) in cerebral perfusion at very high perfusion pressures. If applicable to humans, this may explain the restrained MCAvmean during higher blood pressures at the greater loads. Also, if extremely high blood pressures do indeed produce a sympathetic mediated change in vessel tone, the influence of this activation on the large conduit vessels is also unclear. Nevertheless, if MCA diameter were to decrease via a sympathetic activation, an increase in flow velocity would be expected; however, MCAv was similar across all the exercise conditions. Thus, we believe that this potential issue does not influence our findings.

The restraint of MCAv during exercise may also be attributable to recruitment of the VM during the highest load, because increases in thoracic pressure during a VM are translated to the cerebrospinal fluid such that an increase in intracranial pressure ensues (10). A rise in intracranial pressure can minimize the change in transmural pressure, and thus, any increase in perfusion pressure will be reduced, reflected in only a modest increase in MCAvmean (31). Moreover, the VM produces large increases in central venous pressure (23) that may reduce the pressure difference across the cerebral circulation. The internal jugular vein is responsible for venous drainage while supine; however, upon standing, the internal jugular vein collapses and blood is drained from the brain predominantly via the vertebral venous plexus (9). The collapsed internal jugular vein may increase cerebral outflow resistance (i.e., a Starling resistor) that may be opened by an increase in CVP like that seen during a VM; however, this may require a prolonged increase in pressure to distend (24). Another possible explanation is that the large transient increases in MAP exceed the maximal rate of cerebral vessel dilation, and no further increase via changes in MAP were translated into further increases in MCAvmean. The additional mechanical effect of the VM and possibility of sympathetic activation may also contribute to dynamic cerebrovascular control during such exercise, particularly at higher relative loads.

The reduction in MCAvmean is due to a selective decrease in diastolic flow velocity. Dynamic cerebral autoregulation is reported to be maintained during both static (20) and dynamic exercise (19). Furthermore, although dynamic cerebral autoregulation has been reported to be more effective at safeguarding against hypertension than hypotension during both rest (33) and exercise (19), the blood pressure fluctuations and subsequent MCAv response after the type of activity examined in the present study has not been previously described. Data from the current study indicates that this selective reduction in diastolic flow velocity after upright resistance exercise is the main contributor to the reduction in MCAv and is strikingly similar to that seen at syncope (28). Furthermore, syncope has been reported after maximal Olympic style lifts (4). In addition, MCAvmean recovery was load dependent and remained below baseline for the longest period (~15 s) at the highest relative load (90%). This was supported by all of our data analyses approaches, i.e., time-to-recovery, time below baseline, and AUC. Thus, higher loads caused a greater and more sustained drop in MCAvmean.

The role of arterial CO2 during and after heavy resistance exercise. PETCO2 was reduced after all trials but not significantly between trials (i.e., no effect of load or repetitions, Table 1). However, during exercise and possibly for some time afterward (due to circulation time), PETCO2 may not accurately reflect arterial PCO2 (PArCO2) because of both the recruitment of the VM and the effect.
of thoracic loading-altered ventilation and/or pulmonary perfusion patterns. For these reasons, the accuracy of \( P_{ET}CO_2 \) as an index of \( P_{a}CO_2 \) during upright resistance exercise with concomitant thoracic loading may be limited. Although the MAP and \( P_{ET}CO_2 \) response was not altered by the number of repetitions, more repetitions at the 30% and 60% loads appeared to reduce MCAvmean after exercise (Table 1). The load-dependent reductions in MCAv reflect greater reductions in MAP because there was no significant difference in the ratio of the relative changes in MCAv and MAP at MCAvmean nadir. However, because there was no difference in the reduction in MAP between repetitions after 30% and 60% loads (Table 1), the discrepancy in MCAv is likely due to \( P_{a}CO_2 \) differences that were not reflected by the \( P_{ET}CO_2 \) values. This may reflect the greater metabolic stress during six repetitions, which is supported by the higher heart rates during and after these sets.

**Technological considerations.** Transcranial Doppler ultrasound provides a measure of flow velocity rather than flow. Under various stimuli (34), including simulated orthostasis (29), MCAv accurately reflects the magnitude of changes in flow as the diameter of the MCA remains unchanged. During the modest changes in MAP, the MCA diameter changes <4% (8). Resistance exercise, however, produces much higher MAP, and the subsequent effect on conduit vessel diameter remains unknown. Despite this uncertainty, transcranial Doppler measurements of MCAv have been used extensively in the research of the cerebral-vascular response to resistance exercise (5,17,20,24,27), squatting (3), and the VM (23,31) because of its superior temporal resolution. Nevertheless, caution should be taken with any interpretation of transcranial Doppler data under conditions where the diameter of the measured vessel may change. If MCA diameter did increase, absolute blood flow may have increased without an evident increase in MCAvmean. Further research is warranted to investigate the effects of large MAP perturbations on conduit vessel diameter (including the MCA).

The indirect measurement of arterial blood pressure via Finometer has been shown to accurately reflect intraarterial recordings during initial orthostatic hypotension and at syncope (30). Likewise, several studies have used the Finometer to measure blood pressure during heavy resistance exercise (5,17,27), and squatting (3) and has been validated against intraarterial recordings during orthostasis (12). However, its reliability during upright resistance exercise is not known. Given that we used a protocol to minimize the use of the hand during our exercise maneuvers and confirmed the Finometer recordings with manual blood pressure measures, we feel our Finometer data accurately depicted changes in arterial pressure. Furthermore, our Finometer-derived calculation of \( Q \) has been validated against the thermodilution technique during open heart surgery (26) and accurately tracks changes in \( Q \) (11) during orthostasis. However, the validity and reliability of the calculated \( Q \) during large increases in MAP is unknown. Accordingly, \( Q \) and TPRi data have only been presented during the stand and only as the relative percentage change from baseline.

**CONCLUSION**

Immediately after upright resistance squatting exercise, changes in MAP and MCAvmean are dependent on the load lifted and the number of repetitions. MCAvmean was elevated similarly across all exercise loads regardless of the differential increase in MAP during exercise. The large oscillations in MAP induced by squatting exercise and the large decrease immediately after exercise reflect the “high pass filter” characteristics of the cerebral circulation. This was most apparent at higher loads, which subsequently produced the greatest reductions in both MAP and MCAv. The decrease in MCAvmean during the postexercise hypotension was mediated via a selective reduction in diastolic flow velocity and was consistent with our hypothesis. Finally, dynamic cerebralvasculature control processes rectified MCAv within the same time frame regardless of the magnitude of the drop in MCAv and the magnitude and time course of the reduction in MAP.

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The current address of Zachary J. Schlader is the Institute for Exercise and Environmental Medicine, Texas Health Presbyterian Hospital of Dallas, Dallas, TX.

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**REFERENCES**
