Cerebral hemodynamics and resistance exercise

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ABSTRACT

EDWARDS, M. R., D. H. MARTIN, and R. L. HUGHSON. Cerebral hemodynamics and resistance exercise. Med. Sci. Sports Exerc., Vol. 34, No. 7, pp. 1207–1211, 2002. Purpose: Repetitive resistance exercise with large muscle mass causes rapid fluctuations in mean arterial blood pressure (MAP). We sought to determine the effect of these fluctuations on the cerebrovasculature response determined by mean flow velocity (Vmean) of the middle cerebral artery. Methods: Nine subjects performed 10-repetition maximum leg press exercise. MAP was estimated by finger photoplethysmography, Vmean by Doppler ultrasound, and end-tidal CO₂ (PETO2) by mass spectrometry. Results: Vmean fluctuated with MAP with each repetition however averaged over the 10 repetitions, Vmean was unchanged from resting baseline values (66.9 ± 10.8 vs 67.7 ± 12.3 cm·s⁻¹, baseline vs exercise, P > 0.05) despite an increased MAP (89.5 ± 8.4 vs 105.0 ± 4.9 Torr, P < 0.05). PETO2 also remained unchanged from rest to exercise (37.7 ± 10.6 vs 36.6 ± 2.7 Torr, P > 0.05). Vmean decreased below resting levels for the first 5 s of recovery (59.8 ± 9.1 cm·s⁻¹, P < 0.05) as MAP returned rapidly to slightly below baseline (83.3 ± 6.1, P > 0.05). MAP/Vmean, an index of cerebrovascular resistance, was elevated during exercise and returned to baseline after exercise. An increase in Vmean at 30 s post-exercise (78.4 ± 10.6 cm·s⁻¹, P < 0.05) corresponded with elevated PETO2 (43.0 ± 4.8 Torr, P < 0.05). Conclusion: The results suggest that fluctuations in MAP with individual muscle contractions during resistance exercise appear to be too rapid to be countered by cerebrovascular autoregulation. However, the progressive increase in MAP over a number of contractions was effectively countered to maintain Vmean near baseline values before a decrease in Vmean immediately after exercise. Key Words: DOPPLER ULTRASOUND, ARTERIAL BLOOD PRESSURE, LEG PRESS.

Cerebral blood flow remains relatively constant across a range of mean arterial pressure (MAP) from 50 to 140 mm Hg by the process called autoregulation (14,18). In healthy individuals, the autoregulatory response to step changes in MAP returns cerebral blood flow within 3 to 5 s (28). Heavy resistance exercise might challenge the ability of the autoregulatory system due to both the rapid changes in MAP and the fact that MAP might exceed the autoregulatory zone (15). Injuries such as stroke, subarachnoid hemorrhage, and retinal hemorrhage may be associated with resistance exercise.

Recent measurements of mean flow velocity (Vmean) of the middle cerebral artery (MCA) with Doppler ultrasound in a group of elite power athletes revealed a significant reduction in Vmean from the resting levels during a single maximal lift (7). Loss of consciousness, commonly referred to by weight lifters as “blackouts,” has been reported during heavy resistance exercise (6), suggesting that cerebral blood flow and autoregulation may be critically impaired under these circumstances. Anecdotally, some recreational athletes complain of presyncopal symptoms when performing resistance exercise. The mechanism for the decreased Vmean or insufficient autoregulation, which may lead to syncope, remains elusive; however, it is likely due to an interaction among MAP, intracranial pressure, and cerebrovascular resistance. To date, there is no information on cerebral blood flow during repetitive resistance exercise that might be performed by recreational athletes.

The objective of the present study was to characterize the Vmean response to the double leg press, a common resistance exercise, in a group of recreationally active subjects. Double leg press was selected as it involves a large muscle mass and generates high physiological changes in MAP (15,16). We hypothesized that the rapid fluctuations in MAP with each leg press would be transmitted into oscillations in Vmean while any systematic increase in MAP would be met by an increase in cerebrovascular resistance to keep Vmean close to baseline. To obtain an indicator of cerebrovascular resistance, we determined MAP/Vmean at rest and during resistance exercise to explore autoregulation and factors including arterial CO₂ that influence the control of cerebral circulation.

METHODS

Subjects

Nine healthy subjects (six females) were tested after being fully informed of the experimental details. Their mean age was 21.0 ± 3.6 yr (mean ± standard deviation), height 167.5 ± 8.0 cm and weight 67.3 ± 11.0 kg. All subjects had at least 3 months of resistance exercise experience and were
presently involved in regular training at least 3 times/wk. The Office of Research Ethics at the University of Waterloo approved all procedures, and written consent was obtained.

Procedure

Subjects were instrumented and seated in a 45° inclined bilateral leg press apparatus with the back supported and the feet resting on foot plates. The load selected for testing was designed to allow the subjects to complete 10 and only 10 repetitions of the leg press exercise. This load is equivalent to ~75% of each subject’s theoretical maximum voluntary contraction (MVC) (23). To determine the test load, subjects self-selected a weight based on their own experience. Weights were added or removed so that the 10-repetition criterion was achieved within 2 to 3 trials. After a 5-min rest period, 5 min of continuous baseline data were collected, followed by the 10-repetition leg press exercise. All subjects were instructed to breathe normally and avoid holding their breath. Vmean, MAP, end-tidal CO2 (PETCO2) and heart rate (HR), were continuously recorded for resting baseline collection, exercise, and recovery.

Experimental measures

MAP was determined from the finger placed at the level of the heart using noninvasive arterial photoplethysmography (Finapres Ohmeda, Englewood, CO) and corrected using standard cuff measurements. Vmean of the middle cerebral artery was estimated from transcranial Doppler ultrasonography (Transpect TCD Medasonics, Freemont, CA) as previously described (2). Briefly, a 2 MHz probe was used to isolate the right middle cerebral artery through the temporal bone window and held in place with the use of a headband. Breath-by-breath PETCO2 was collected continuously using an ultrasonic flow meter (Kou Consulting Inc., Redmond, WA) and mass spectrometry (MGA-1100, Perkin-Elmer Medical Gas Analyzer, Pomona, CA). HR was determined by a 3-lead electrocardiograph.

Data Analysis

Data were recorded on digital format tape (TEAC Instruments, Montebello, CA), then transferred for analysis by a computer based system to yield a data set sampled at 100 Hz. Vmean was determined from the outer envelope of the fast Fourier transformed Doppler signal. The ratio, MAP/Vmean, was calculated as an index of averaged cerebrovascular resistance.

Statistics

Differences among mean values during rest, exercise, and three recovery periods (first 5 s, 5 to 15 s, and 15 to 30 s of recovery) were assessed with a one-way repeated measures ANOVA for the dependent variables Vmean, MAP, MAP/Vmean, HR, and PETCO2. If significance was obtained (P < 0.05), a Bonferoni post hoc was used to assess the differences from baseline.

RESULTS

A representative data set from one subject is illustrated in Figure 1. The MAP response to resistance exercise followed a distinct sinusoidal oscillatory pattern with each repetition that increased with successive repetitions. Vmean followed a similar sinusoidal oscillatory pattern to MAP with each repetition, but unlike MAP it did not increase with successive repetitions. The first solid vertical line provides a reference for the time relationship between MAP, Vmean and MAP/Vmean. The second solid, vertical line marks the time period in which Vmean reached its lowest point at 5 s of recovery. Horizontal lines through each plot represent averaged baseline level. MAP and Vmean oscillated with each lift however, only MAP progressively increased with successive repetitions. MAP/Vmean appeared to respond to the progressive increase in MAP. The increase in Vmean during recovery appeared to be in response to the elevated PETCO2.

FIGURE 1—Arterial blood pressure (MAP), mean flow velocity (Vmean) of the middle cerebral artery, MAP/Vmean as an index of cerebrovascular resistance, end-tidal CO2 (PETCO2) and heart rate (HR) are shown for baseline, exercise, and recovery for one representative subject. The first dotted line indicates the onset of exercise and the second dotted line indicates the termination of the exercise or onset of recovery. The first solid, vertical line provides a reference for the time relationship between MAP, Vmean and MAP/Vmean. The second solid, vertical line marks the time period in which Vmean reached its lowest point at 5 s of recovery. Horizontal lines through each plot represent averaged baseline level. MAP and Vmean oscillated with each lift however, only MAP progressively increased with successive repetitions. MAP/Vmean appeared to respond to the progressive increase in MAP. The increase in Vmean during recovery appeared to be in response to the elevated PETCO2.
Significantly different from resting baseline values, \( P < 0.05 \) during exercise as well as in the first 5 s, 5 to 15 s, and 15 to 30 s of recovery (Table 1). Averaged data are shown in Table 1. Compared to resting values, averaged Vmean was unchanged during exercise (\( P > 0.05 \)), depressed during the first 5 s of recovery (\( P < 0.05 \)), and increased within 15 to 30 s of recovery (\( P < 0.05 \)). The MAP increased from resting levels during exercise (\( P < 0.05 \)) but returned to baseline values during recovery (\( P > 0.05 \)), although there was a trend for MAP to be decreased at the first 5 s of recovery. The data for a single subject (Fig. 1) shows with the second solid vertical line that the decrease in Vmean corresponded to the rapid drop in MAP. MAP/Vmean was elevated during exercise (\( P < 0.05 \)) and declined toward baseline within the first 5 s of recovery (\( P < 0.05 \)). HR was increased above baseline during exercise as well as in the first 5 s, 5 to 15 s, and 15 to 30 s of recovery (\( P < 0.05 \)).

### DISCUSSION

The major new observations from this study were that the average value of Vmean remained constant during leg press exercise, yet Vmean oscillated directly with the changes in MAP during the exercise. Further, we observed that Vmean decreased significantly below baseline in the first 5 s of recovery. P\( _{ET}CO_2 \) was increased at 5 to 15 s, and 15 to 30 s of recovery (\( P < 0.05 \)). The arterial partial pressure of CO\(_2\) has an important role in the regulation of cerebral vascular resistance (13,26,27). It initiated almost immediately with a change in MAP, but the full response requires 3 to 5 s in healthy individuals and up to 10 to 15 s with impaired autoregulation (28). Thus, the cerebral circulation can effectively minimize changes in Vmean in response to slowly developing changes in MAP. Interpretation of the cerebrovascular resistance response has to be made with caution because resistance is calculated from perfusion pressure gradient divided by blood flow. Consistent with all other studies of cerebrovascular control in humans, we were unable to measure either of these variables directly. At least for the baseline period and the recovery period it would be anticipated that venous or intracranial pressure would be relatively constant so that changes in MAP would reliably reflect changes in perfusion pressure gradient, and MAP/Vmean would provide an index of cerebrovascular resistance. Thus, our observation of somewhat elevated MAP/Vmean immediately after exercise suggests that cerebrovascular resistance probably increased during the exercise to counter the progressive rise in MAP. On the other hand, it is not appropriate to interpret MAP/Vmean directly as an index of cerebrovascular resistance during the leg press exercise, as the venous and intracranial pressures were almost certainly increased at some phase of the activity. Although our subjects did not perform a Valsalva maneuver against a closed glottis, as evidenced by the expired air profile, they probably had some increase in intrathoracic pressure that could alter central venous pressure (19,20) and thus affect the pressure gradient across the cerebrovascular system (4,22).

During the 10-repetition leg press exercise there was no change in average Vmean compared with baseline. This differed from an observation of reduced Vmean in elite power athletes during a single maximal lift (7). There are several differences between experiments that might account for this. A simple explanation might be the timing of measurements. While average Vmean was not affected (Fig. 1, Tab. 1), there were large fluctuations about the mean value during the leg press exercise of the current study as well as during a rowing cycle (19). An apparent under perfusion is immediately followed by an over perfusion: the physiological significance of this finding to cerebral function/metabolism is unknown. During a single maximal lift MAP is markedly elevated, probably in part from the contribution of a Valsalva maneuver. When performed in the absence of weight lifting exercise the Valsalva causes a reduction in Vmean (20).

The arterial partial pressure of CO\(_2\) has an important role in the regulation of cerebrovascular resistance (13,26,27). It
has been suggested that the increase in Vmean during dynamic exercise might (10) or might not (12) be related to arterial PCO₂. We observed that P ejected CO₂ was constant during resistance exercise but that it increased to its highest point at 30 s into recovery. This was followed after a short delay by the peak response of Vmean during the recovery period. It seems reasonable to suggest that the observed amplitude and time delay between P ejected CO₂ and Vmean indicated that during recovery, P ejected CO₂ resulted in cerebral vasodilatation. The cerebrovascular response to P ejected CO₂ from baseline to 30 s recovery (% change Vmean/change in pressure and modest changes in PET CO₂ (25), and similar to other Doppler studies reported in the literature under resting conditions (5, 17). In addition, Poulin et al. (21) recently found the vascular response of Vmean to step changes in P ejected CO₂ was initiated after a 6-s delay, much more rapid than previously thought.

None of our subjects experienced dizziness or syncope during the testing protocol. Syncopal symptoms are probably related to a reduction in cerebral blood flow that could be due to some combination of increased cerebrovascular resistance or decreased MAP. It has been suggested that hyperventilation before or during heavy resistance exercise might be linked to the syncope experienced by some athletes (6). Based on P ejected CO₂ values, none of our subjects hyperventilated before exercise. Given the decline in Vmean immediately after exercise in individuals with normal P ejected CO₂, it would seem prudent to recommend that individuals taking part in resistance exercise avoid voluntary hyperventilation before or during exercise as this would further elevate cerebrovascular resistance, potentially causing a more marked decline in Vmean.

**Limitations**

Doppler ultrasound of the middle cerebral artery measures velocity and not flow, and therefore relies on the assumption that the diameter of the artery does not change. This assumption has been considered extensively (1, 29), including in studies with magnetic resonance imaging (MRI) of the cerebral artery during lower body negative pressure and modest changes in P ejected CO₂ (25), and comparisons with ¹³³Xe clearance during dynamic cycle exercise (12). However, flow indices calculated by spectral weighted sums of the transcranial Doppler signal have questioned this assumption. Giller et al. (9) evaluated the cerebral blood flow response to rhythmic handgrip exercise and found that their calculated cerebral blood flow index was heterogeneous between subjects and concluded that ordinary cerebrovascular velocities obtained during exercise may be invalid. To date this issue has not been resolved. In the study of Giller, it appeared from the typical subject presented that during the first 100 s of exercise, as MAP increased Vmean and their indicator of MCA diameter remained constant. It was only in the final 200 s of exercise that the indicator of diameter changed. Thus, it is not apparent how their data might affect our interpretation as the current exercise lasted approximately 40 s (Fig. 1). If there were any reduction in MCA diameter at the end of exercise, then our observed decrease in Vmean would underestimate the decline in cerebral blood flow.

As considered above, calculation of MAP/Vmean to provide an index of cerebrovascular resistance to study auto-regulation is appropriate during steady state conditions, but should be viewed with caution during exercise or Valsalva maneuver. With repetitive resistance exercise or dynamic exercise, MAP, cerebrovascular resistance, intracranial pressure, and cerebral venous outflow might be expected to change. Poit et al. (19) found that Vmean increased by 10 cm/s⁻¹, MAP increased by 11 Torr, and central venous pressure by 8 Torr during rowing exercise. Similar to the present study, intracranial pressure could not be assessed, yet the authors concluded that Vmean was dominated by MAP. Furthermore, under a resistance exercise protocol such as ours, numerous other factors such as brain metabolism, blood volume, and thermoregulation may have also influenced cerebral blood flow (11). In spite of these limitations, the main measured variable, Vmean, did fluctuate markedly during exercise and showed a postexercise pattern that was consistent with an elevated cerebrovascular resistance within the first 5 s after exercise, and a reduced MAP/Vmean subsequently as P ejected CO₂ increased.

**CONCLUSION**

The results of the present study indicate that Vmean was dependent on the fluctuations in MAP that occur during individual repetitions of resistance exercise but independent of the progressive rise in MAP with successive repetitions. These findings suggest that each muscle contraction during resistance exercise may be too fast to engage an effective autoregulatory response, but that systematic changes in MAP are countered to maintain mean cerebral blood flow close to the baseline value.

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