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 cerebrovascular 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 (P_{ET} CO₂) 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). P_{ET} CO₂ also remained unchanged from rest to exercise (37.7 ± 2.8 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 P_{ET} CO₂ (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 (P_{ET}CO₂) 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 non-invasive 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 P_{ET}CO₂ 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 P_{ET}CO₂. If significance was obtained (P < 0.05), a Bonferroni 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 in Figure 1 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 MAP 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 P_{ET}CO₂.
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 after exercise. These observations during and immediately after exercise were consistent with our hypotheses that rapid oscillations in MAP transmit directly to Vmean but that systematic increases in MAP are countered by an efficient autoregulatory response to maintain Vmean. The reason that Vmean declined significantly below baseline in the first 5 s after exercise were consistent with the 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 CO2 has an important role in the regulation of cerebrovascular resistance (13,26,27). It initiates 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.
vascular response of $V_{mean}$ to step changes in $P_{ET\ CO_2}$ was (5,17). In addition, Poulin et al. (21) recently found the studies reported in the literature under resting conditions comparisons with $^{133}$Xe clearance during dynamic cycle exercise pressure and modest changes in $P_{ET\ CO_2}$ (25), and comparisons with MRI of the cerebral artery during lower body negative pressure and the assumption that the diameter of the artery does not change. The cerebrovascular response to $P_{ET\ CO_2}$ from baseline to 30 s recovery (% change $V_{mean}$/change in $P_{ET\ CO_2}$) was 3.2% per Torr $P_{ET\ CO_2}$, 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 $V_{mean}$ to step changes in $P_{ET\ CO_2}$ was initiated after a 6-s delay, much more rapid that 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 $P_{ET\ CO_2}$ values, none of our subjects hyperventilated before exercise. Given the decline in $V_{mean}$ immediately after exercise in individuals with normal $P_{ET\ CO_2}$, 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 $V_{mean}$.

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 (1,3,29), including in studies with magnetic resonance imaging (MRI) of the cerebral artery during lower body negative pressure and modest changes in $P_{ET\ CO_2}$ (25), and comparisons with $^{133}$Xe clearance during dynamic cycle exercise (12). However, flow indices calculated by spectral weighed 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 $V_{mean}$ 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 $V_{mean}$ would underestimate the decline in cerebral blood flow.

As considered above, calculation of MAP/$V_{mean}$ to provide an index of cerebrovascular resistance to study autoregulation 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. Pott et al. (19) found that $V_{mean}$ increased by 10 cm$^2$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 $V_{mean}$ 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, $V_{mean}$, 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/$V_{mean}$ subsequently as $P_{ET\ CO_2}$ increased.

CONCLUSION

The results of the present study indicate that $V_{mean}$ 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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