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ORIGINAL PAPER

Possible influences of exercise-intensity-dependent increases in non-cortical hemodynamic variables on NIRS-based neuroimaging analysis during cognitive tasks: Technical note

Kyeongho Byun[1], Kazuki Hyodo[1], Kazuya Suwabe[1], Sylwester Kujach[2], Morimasa Kato[3] and Hideaki Soya[1]*

[1]Laboratory of Exercise Biochemistry and Neuroendocrinology, Faculty of Health and Sport Sciences, University of Tsukuba, Ibaraki, Japan
[2]Department of Physiology, Gdansk University of Physical Education and Sport, Gdansk, Poland
[3]Department of Health and Nutrition, Yonezawa Nutrition University of Yamagata Prefecture, Yamagata, Japan

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Kyeongho Byun, Kazuki Hyodo, Kazuya Suwabe, Sylwester Kujach, Morimasa Kato and Hideaki Soya. Possible influences of exercise-intensity-dependent increases in non-cortical hemodynamic variables on NIRS-based neuroimaging analysis during cognitive tasks: Technical note. *JENB*., Vol. 18, No. 4, pp.327-332, 2014 **[Purpose]** Functional near-infrared spectroscopy (fNIRS) provides functional imaging of cortical activations by measuring regional oxy- and deoxy-hemoglobin (Hb) changes in the forehead during a cognitive task. There are, however, potential problems regarding NIRS signal contamination by non-cortical hemodynamic (NCH) variables such as skin blood flow, middle cerebral artery blood flow, and heart rate (HR), which are further complicated during acute exercise. It is thus necessary to determine the appropriate post-exercise timing that allows for valid NIRS assessment during a task without any increase in NCH variables. Here, we monitored post-exercise changes in NCH parameters with different intensities of exercise. **[Methods]** Fourteen healthy young participants cycled 30, 50 and 70% of their peak oxygen uptake (Vo_{2peak}) for 10 min per intensity, each on different days. Changes in skin blood flow velocity (SBFv), middle cerebral artery mean blood velocity (MCA V_{mean}) and HR were monitored before, during, and after the exercise. **[Results]** Post-exercise levels of both SBFv and HR in contrast to MCA V_{mean} remained high compared to basal levels and the times taken to return to baseline levels for both parameters were delayed (2-8 min after exercise), depending upon exercise intensity. **[Conclusion]** These results indicate that the delayed clearance of NCH variables of up to 8 min into the post-exercise phase may contaminate NIRS measurements, and could be a limitation of NIRS-based neuroimaging studies. **[Key words]** NIRS, non-cortical hemodynamic changes, exercise intensity, post-exercise phase.

INTRODUCTION

Recently, it has been possible to non-invasively assess cortical brain activity through the use of near-infrared light in human subjects [1-3]. Numerous studies using this novel optical neuroimaging technique have demonstrated that several types of brain activity, including motor activity [4,5], visual activation [6,7], and performance of cognitive tasks [1,7,8], can be assessed based on the fact that increased cortical brain activity is associated with increased oxygenated hemoglobin and decreased deoxygeneted hemoglobin that occurs within several seconds after the onset of increased brain activity [9].

Due to several methodological benefits including easy installation in a gym and an acceptable signal-to-noise ratio over other neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), near-infrared spectroscopy (NIRS) methods have been broadly used to investigate the relation between exercise-induced cognitive performance and cortical neural

* Corresponding author: Hideaki Soya, Tel. 81-29-853-2620, Email: hsoya@taiiku.tsukuba.ac.jp

activations [10-12]. Recent NIRS studies have attempted to investigate changes in brain activations during cognitive tasks when subjects do exercise, and have reported increased cortical oxygenation as the neural basis for exercise-induced cognitive improvement [13,14]. However, the question of whether it is possible that these NIRS-derived measurements might, in fact, not reflect brain- activity-elicited increases in hemoglobin oxygenation because an acute bout of exercise also increases non-cortical hemodynamic (NCH) variables that may influence NIRS measurements as NIRS-specific noise has been raised [15-19]. In regard to these NIRS-specific contaminations, previous research has established a specific experimental condition allowing the investigation of exercise-induced changes in neural activation without NCH-contamination after 10 min of mild or moderate intensity exercise [10,12], but it is still difficult to generalize from these examples because NCH values vary among individuals and are dependent upon exercise intensity.

For NIRS measurements, near-infrared light passes through the skin, skull, and cerebral cortex to measure changes in regional cerebral oxygenation, and thus it is easy for the measurements to be contaminated by NCH changes induced by skin blood flow and cerebral blood flow. For instance, several studies have revealed that exercise-induced thermoregulatory cutaneous vasodilation temporarily increases oxy-Hb in skin blood flow and possibly influences NIRS-derived oxy-Hb signals on the forehead [15,20]. In addition, cerebral auto-regulation maintains a relatively stable cerebral blood flow over a range of perfusion pressures from 60 to 150 mmHg in a resting state. However, in response to acute exercise, MCA V_{mean}, which represents changes in regional blood flow to the cerebral cortex, becomes elevated [21-24]. Thus, it is possible to postulate that exercise-increased global blood volume to the cerebral cortex might affect NIRS measurement. Moreover, it is well-known that systemic cardiovascular fluctuations such as heart rate affect cerebrovascular fluctuations [19]. To overcome these methodological issues, there has been growing interest in experimental conditions with the potential to clarify the expected influences of NCH variables on exercise-induced neural activation changes.

Based on the theoretical concept of acute-exercise-cognition interaction, it is generally hypothesized that exercise intensity might have differential effects on cognitive processing. This concept has brought our research interest towards investigating the underlying neural substrates of the differential effects of various intensities of exercises on brain function using the NIRS method. To explore the underlying neural mechanisms of the effects of each intensity of exercise on cognitive function using NIRS methods, how each intensity of exercise influences NCH variables during and after exercise, which could interfere with NIRS measurements, must be examined.

In this study, we examined changes in NCH variables including SBFv, MCA V_{mean} and HR in response to 10 min of exercise at 30, 50, or 70% of peak oxygen uptake ($\dot{V}O_{2peak}$). We hypothesized that exercise intensity affects the time it takes for possible NIRS-derived contaminators to recover to baseline levels in order to investigate the effects of exercise on its neural substrates.

METHODS

Subjects

The experiments were performed on fourteen healthy subjects (5 males) with the following morphometric characteristics (mean ± SD), age 20.3 ± 1.7 years, height 163.5 ± 7.4 cm, weight 53.9 ± 7.4 kg, and $\dot{V}O_{2peak}$ 39.3 ± 7.9 $mL \cdot min^{-1} \cdot kg^{-1}$. Participants provided written informed consent before the experiment. All protocols were approved by the Institutional Review Board at the University of Tsukuba and were conducted in accordance with the Declaration of Helsinki principles. All subjects refrained from caffeine, alcohol, and exercise for 24 h before the experiment.

Maximal oxygen consumption

Before the main experiments, subjects performed an incremental recumbent cycling test with the pedaling frequency set at 60 rpm starting at 30 W for 3 min and increasing by 20 W (15 W for female subjects) every 1 min until volitional exhaustion. Expired gasses were continuously collected and analyzed at 15 s intervals (Aeromonitor AE300, Minato Medical Science, Japan) for the direct determination of individual $\dot{V}O_{2peak}$. Rating of perceived exertion (RPE) was assessed at the end of every minute (Borg-scale) and heart rate (HR) was recorded at 1 s intervals (Polar RS400, Polar Electro, Finland).

The exercise was considered to be maximal when three of the following criteria were obtained: no change in oxygen consumption while increasing workload, respiratory exchange ratio > 1.1, HR within 10% of predicted maximal value, and maximal rating of perceived exertion or the inability of the subjects to maintain the pedaling frequency despite maximum effort and verbal encouragement.

Exercise trials

Within 2 weeks of completing of the incremental exercise test, each participant arrived at the laboratory having abstained from exercise and alcohol for 24 h, and having not consumed a heavy meal or items containing caffeine for 4 h. The participants rested in a sitting position for 15 min at an ambient temperature of 27 ± 0.3℃ and relative humidity of 45.5 ± 2.4%. During this time, the instrumentation was attached to the participant.

After measuring baseline values for 3 min, participants cycled at an intensity of 30, 50, or 70% Vo_{2peak} for 10 min and rested their forearms and hands on their thighs to keep their upper body and hands relaxed for 20 min. To determine exertion (or workload) needed to achieve 30, 50, and 70% of Vo_{2peak}, Vo_2 was plotted against the output power of the strength ergometer to Vo_{2peak} [25]. The order of the exercise intensity was randomized. The response at each level of exercise intensity was tested on a different day with at least one day separating each exercise session. Each test was performed at the same time of day to avoid potential effects of circadian variations.

Measurements of changes in HR, SBFv and MCA V_{mean} responses

HR was continuously monitored, as aforementioned. SBFv was monitored with a laser-Doppler probe (FLO-C1; Omegawave, Japan) attached at the Fpz point of the international 10-20 System. MCA V_{mean} was measured in the right temporal window using a 2 MHz pulsed Doppler ultrasound system (WAKI 1-TC; Atys Medical, France) [26]. The Doppler probe was fixed with an adjustable headband device and adhesive ultrasonic gel to maintain optimal insonation position and angle throughout the protocol. All data were acquired continuously using an analog-to-digital converter (Powerlab 16/30 ML305; ADInstruments, USA) interfaced with a computer, and subsequently analyzed using commercially available software (LabChart 7.0, ADInstruments).

Statistical analysis

The data are presented as mean ± SEM. A two-way analysis of variance was performed using Bonferroni correction when F-values were significant, to clarify the interaction between time and exercise intensity during the exercise. Each parameter measured at the resting period was set at 100%. Then, the relative percentage changes were averaged every minute, and all parameters were frequently plotted. The effect of each exercise, compared to the average value during the resting period (of 3 min before exercise), was evaluated using a t-test with Dunnett correction. Statistical significance was set at $p < 0.05$.

RESULTS

The ANOVA for HR exhibited significant interaction between time and exercise intensity ($F(1, 24) = 39.67$, $p < 0.001$). HR at the end of exercise increased significantly with increasing exercise intensity (Low$^{*,\#}$: 108.0 ± 2.6 bpm at 30% Vo_{2peak}; Mod$^{\$,\#}$: 131.4 ± 2.3 bpm at 50% Vo_{2peak}; High$^{*,\$}$: 160.4 ± 3.6 bpm at 70% Vo_{2peak}; * significantly differ from Mod, $p < 0.001$, Bonferroni correction; $^{\#}$ significantly differ from High, $p < 0.001$, Bonferroni correction; $^{\$}$ significantly

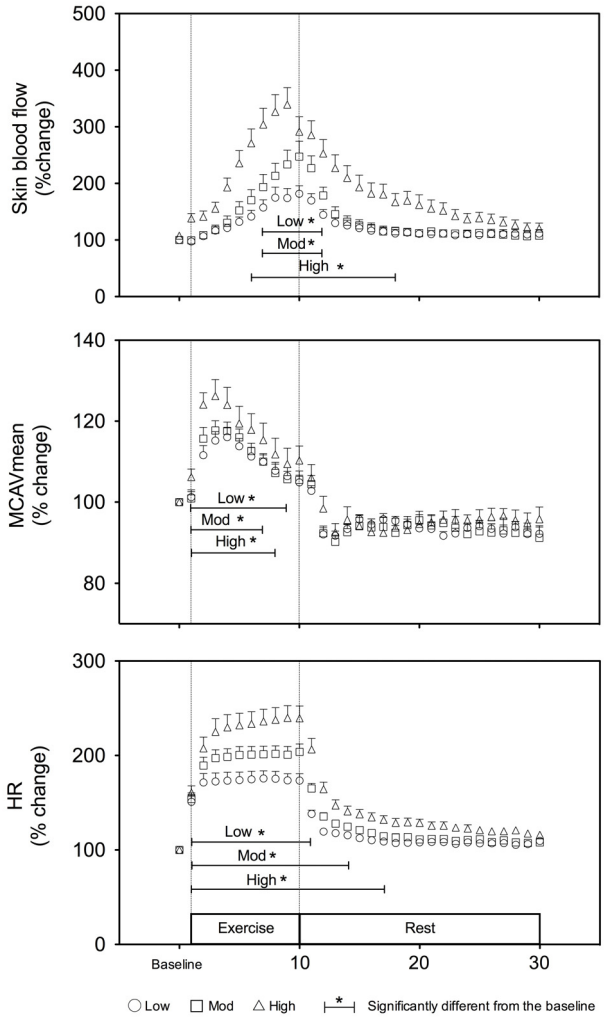

Fig. 1. Time course of relative changes in levels of non-cortical hemodynamic physiological parameters. (A) SBFv (B) MCA V_{mean}, and (C) HR at 30% Vo_{2peak} (Low ○), 50% Vo_{2peak} (Mod □), and 70% Vo_{2peak} (High △). Mean values ± SEM are presented. *$p < 0.05$: significantly different from baseline levels.

differ from Low, $p < 0.001$, Bonferroni correction). None of the baseline values of the measured variables during the 3 min before exercise differed significantly among exercise sessions. Fig. 1 shows the percent changes over time for each physiological parameter. Significant increases of SBFv in response to each intensity (30, 50, and 70%) of exercise were observed from 6, 6, and 5 min after beginning the exercise to 2, 2, and 8 min into each recovery period, respectively. Although MCA V_{mean} significantly increased 1 min after the onset of each intensity of exercise, that after the end of exercise immediately decreased to the basal level regardless of exercise intensity. HR also increased significantly at the beginning of each exercise period, and returned to an insignificant level 1, 4, and 7 min, respectively, into each recovery period.

DISCUSSION

Here we delineated the post-exercise dynamic changes in SBFv, MCA V_{mean} and HR in response to 10 min of exercise at 30, 50 or 70% of individual Vo_{2peak} and found that both the SBFv and HR levels decreased simultaneously and that times to return to respective basal levels increased in an exercise-intensity-dependent manner. These SBFv and HR levels, which were higher than the basal levels with each intensity of exercise, may prevent the assessment of cortical activations during a cognitive task as measured using NIRS neuroimaging methods.

After the onset of exercise, various cardiovascular responses occur in accordance with elevated metabolic demands in skeletal muscle and the brain [27,28]. Typically, HR, which is an indicator of changes in cardiac output, increases in an exercise-intensity-dependent manner [29,30], increasing the blood supply to relevant organs and, to some extent, to cutaneous capillaries for thermoregulation [31,32]. As expected, our results revealed that both SBFv and HR parameters similarly increased in response to exercise, and that the time required for each value to return to its respective baseline levels increased, depending upon exercise intensity. It is known that NIRS-derived measurements can be overestimated due to higher skin blood flow [15,33] and can be corrupted with strong noise artifacts such as the cardiac cycle and blood pressure [19,34,35]. Thus, unless SBFv and HR return to their respective basal levels, NIRS-determined cerebral oxy-Hb during or immediately after dynamic exercise may not necessarily correspond to regional neural activity in the cortex.

In contrast to other organs, it was believed that total blood flow to brain tissue remains relatively unaffected by a variety of conditions. However, a significant increase in MCA V_{mean} has been reported in recent studies with various modes of exercise such as dynamic handgrip, cycling, and running [21, 23,24,36,37]. These exercise-induced changes are consistent with our findings and have been taken to reflect an increase in global cerebral blood flow to the cortical regions during exercise. Thus, increases in MCA V_{mean} during exercise potentially impacts NIRS measurements. However, regardless of exercise intensity, MCA V_{mean} after the end of exercise immediately decreases to the resting level. This is probably due to the vasoconstrictive effects of reduced $PaCO_2$ as a result of increased ventilation after exercise [38]. Therefore, it is postulated that NIRS-derived brain activity during a cognitive task would be not affected by changes in cerebral blood volumes after exercise.

Similar to previous findings in regard to NIRS-specific contamination by NCH variables [10-12], our results also indicate that even mild exercise influences post-exercise heart rate and skin blood flow in the forehead, and that this influence extends depending upon exercise intensity. On the basis of the present findings, it is postulated that NIRS-specific concerns are greatest for experimental protocols involving high intensity or prolonged exercise that have the potential to cause substantial thermal stress and energetic demands, leading to further increases in skin blood flow and HR values [27,31,32]. Therefore, researchers who attempt to investigate exercise-induced neural activation changes with NIRS-based methods should carefully consider the contribution of post-exercise increases in skin blood flow and heart rate, depending upon exercise intensity.

In the present study, however, we did not monitor changes in NIRS-derived oxygenation during a cognitive task. Thus, we were actually unable to determine the extent to which the discrepancies between exercise-induced NCH changes and brain-activity-elicited cerebral oxygenation were due to exercise intensity. Moreover, even if many previous studies have suggested that an acute bout of exercise could have facilitative effects on cognitive processing [39-41], its effects could decline over time after the termination of exercise because the exercise-induced modulation of the brain activity of a neural network involved in certain cognitive processes may be transient [42]. Together with the present results, these declining effects of exercise on cognitive function should be considered when assessing the effects of acute exercise on task-dependent cortical activation with the use of NIRS.

Collectively, our results suggest that the increased post-exercise levels of both SBFv and HR remain high compared to basal levels, depending upon exercise intensity, and that the times required for both parameters to return to baseline

values are delayed with increased exercise intensity. Thus, skin blood flow and heart rate levels that remain high after the termination of exercise may contaminate NIRS analysis when assessing the effect of each intensity of exercise on functional neural activations in the early phase of post-exercise recovery, which is a potential limitation of NIRS-based neuroimaging studies.

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CONFLICT OF INTEREST

The authors have no financial, consultational, institutional, or other relationships that might lead to bias or a conflict of interest.

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