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ORIGINAL ARTICLE

Does cerebral oxygenation affect cognitive function during exercise?

Soichi Ando · Masahiro Kokubu · Yosuke Yamada · Misaka Kimura

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Abstract This study tested whether cerebral oxygenation affects cognitive function during exercise. We measured reaction times (RT) of 12 participants while they performed a modified version of the Eriksen flanker task, at rest and while cycling. In the exercise condition, participants performed the cognitive task at rest and while cycling at three workloads [40, 60, and 80% of peak oxygen uptake ($\dot{V}O_2$)]. In the control condition, the workload was fixed at 20 W. RT was divided into premotor and motor components based on surface electromyographic recordings. The premotor component of RT (premotor time) was used to evaluate the effects of acute exercise on cognitive function. Cerebral oxygenation was monitored during the cognitive task over the right frontal cortex using near-infrared spectroscopy. In the exercise condition, we found that premotor time significantly decreased during exercise at 60% peak $\dot{V}O_2$ relative to rest. However, this improvement was not observed during exercise at 80% peak $\dot{V}O_2$. In the control condition, premotor time did not change during exercise. Cerebral oxygenation during exercise at 60% peak $\dot{V}O_2$ was not significantly different from that at rest, while cerebral oxygenation substantially decreased during exercise at 80% peak $\dot{V}O_2$. The present results suggest that an improvement in cognitive function occurs during moderate exercise, independent of cerebral oxygenation.

Keywords Premotor time · Reaction time · Near-infrared spectroscopy · Hyperventilation

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S. Ando · M. Kimura
School of Nursing, Kyoto Prefectural University of Medicine, Kyoto, Japan

S. Ando
Osaka University of Health and Sport Sciences, Kumatori, Japan

M. Kokubu · Y. Yamada
Graduate School of Human and Environmental Studies, Kyoto University, Kyoto, Japan

Present Address:
S. Ando (✉)
Faculty of Sports and Health Science, Fukuoka University, 8-19-1 Nanakuma, Jonan-ku, Fukuoka 814-0180, Japan
e-mail: sando@fukuoka-u.ac.jp; soichi.ando@gmail.com

Introduction

Cognitive function is fundamentally important to most human activities. The importance of cognitive function in sports has been emphasized (e.g. Williams and Ericsson 2005). Many sports require high-level cognitive functioning under conditions of physiological stress. For example, soccer players must make quick and accurate decisions to pass a ball to better placed teammates on the field. Since cognitive function is likely to be associated with athletes' performance, it is crucial for cognitive function to be maintained during strenuous exercise for maximal sports performance.

Many studies have examined the effects of acute exercise on cognitive function (see reviews by Brisswalter et al. 2002; Etnier et al. 1997; McMorris and Graydon 2000; Tomporowski 2003). However, the specific ways in which cognitive function is affected during strenuous exercise remains unclear. In two recent studies, McMorris et al. (2008, 2009) reported that cognitive function was impaired during strenuous exercise at 80% maximum aerobic power, and discussed an exercise–cognition interaction from a

hormonal perspective. Exercise has been found to induce several physiological changes in the central nervous system, including circulatory, metabolic, and neurohormonal effects (Dalsgaard 2006; Nybo and Secher 2004; Meeusen et al. 2006). These findings imply that multiple factors may affect cognitive function during exercise (Brisswalter et al. 2002; Tomporowski 2003). Thus, to understand the way in which strenuous exercise affects cognitive function it is necessary to investigate the range of potential mechanisms individually, and to accumulate empirical evidence bearing on each.

Brain function and tissue integrity are dependent on continuous oxygen supply because aerobic metabolism is the major energy source to the brain (Zauner et al. 1997). Earlier studies have shown that cognitive performance may be impaired under resting conditions in a hypoxic environment (e.g. Fowler et al. 1987; Kida and Imai 1993; Noble et al. 1993). These findings suggest that decreases in cerebral oxygenation may compromise cognitive function. Cerebral oxygenation during exercise is dependent on the cardiovascular and pulmonary systems (Koike et al. 2004). When the brain is activated during exercise, an increase in cerebral oxygen supply is required to match the enhanced level of neuronal metabolism (Secher et al. 2008). However, hyperventilation induced by strenuous exercise may lead to decrease in cerebral oxygenation (Ando et al. 2010; Bhambhani et al. 2007; Secher et al. 2008). Thus, in the present study, we expected that if cerebral oxygenation is associated with cognitive function during exercise, a decrease in cerebral oxygenation may impair cognitive function during strenuous exercise.

The current study sought to test whether cerebral oxygenation affects cognitive function during exercise. We hypothesized that a decrease in cerebral oxygenation during strenuous exercise would have detrimental effects on cognitive function. This study is relevant to sports in which cognitive function plays an important role. Our findings also have implications for cognitive performance during exercise, in general.

Methods

Participants

Twelve male participants (mean ± SD, age = 25.3 ± 3.1 years; height = 1.75 ± 0.06 m; body mass = 69.8 ± 10.1 kg) gave written informed consent to participate in this study. None of the participants had a history of cardiovascular, cerebrovascular, or respiratory diseases. Participants were asked to refrain from engaging in strenuous exercise for 48 h prior to each experiment. All experimental procedures were approved by the local ethics committee and were in accordance with the Declaration of Helsinki.

Cognitive task

The participants performed a modified version of the Eriksen flanker task (Colcombe et al. 2004; Pontifex and Hillman 2007). A computer and a reaction time (RT) measurement apparatus (Qtec Co., Ltd., Osaka, Japan) were used to control visual stimulus presentation and record the RT of each trial. At the beginning of each trial, a white fixation cross (99 cd/m^2) was presented on a black background (0.02 cd/m^2) for 3 s, which was followed by a 1 s pre-cue that consisted of five horizontal bars (- - - - -). The pre-cue informed the participants that an array of five arrows was about to appear. The array of five arrows then appeared on the screen for 1 s. The participants were asked to respond to the orientation of a central arrow embedded in the array of five arrows. The central arrow pointed either to the left or the right. In congruent trials, the flanking arrows pointed in the same direction as the central arrow (<<<<< or >>>>>). In incongruent trials, the flanking arrows pointed in the opposite direction as the central arrow (<<><< or >><>>). One block consisted of 20 congruent and 20 incongruent trials. The order of congruent and incongruent trials was randomized. The left and right central arrows were presented with equal probability. The total time for one block was 3 min and 20 s. Participants were asked to release a response button as quickly and accurately as possible with their left thumb when the central arrow pointed to the left and with their right thumb when the central arrow pointed to the right. Response buttons were attached at the handlebars of the cycle ergometer.

During the cognitive task, each participant faced a computer screen with their head on a chin rest so the eyes were directly in front of, and level with, the position of the visual stimulus on the screen. The chin rest was located at the middle of the handlebars. The distance from the chin rest to the screen was 58 cm. Participants stabilized their heads on the chin rest while the cognitive task was performed. We used an electro-oculogram (EOG) to monitor overt eye movements and eye blinks during the cognitive task.

Experimental procedure

The experiment was performed on three non-consecutive days. On the first day, participants performed a maximal exercise test on a cycle ergometer (Cordless bike V60, Senoh, Tokyo, Japan) to determine peak oxygen uptake ($\dot{V}O_2$). Following a warm-up exercise at 80 W for 4 min, a maximal exercise test began at a freely chosen cadence

with a 10 W increment every minute in a step-wise manner. The maximal exercise test was stopped when the participants reached the limit of tolerance. The peak $\dot{V}O_2$ was defined as the highest $\dot{V}O_2$ attained in this period. The mean peak $\dot{V}O_2$ was 45.7 ± 7.6 ml/kg/min. Participants performed practice blocks a few days before the second day of experiments. They completed three to five practice blocks of 40 trials each, in which they sat on the cycle ergometer. These practice blocks minimized the possibility that learning effects could interfere with the effects of acute physiological changes.

On the second and third days, participants performed the cognitive tasks in the exercise and control conditions. The experimental protocol is summarized in Fig. 1. The order of the exercise and control conditions was counterbalanced across the participants. At the beginning of the experiment, the cognitive task was performed while participants rested on the cycle ergometer. Then, in the exercise condition, participants cycled at three different workloads (40, 60, and 80% peak $\dot{V}O_2$) in an incremental manner. The pedaling rate was freely chosen by each participant. The duration of each workload was 6 min 30 s. During exercise at each workload, the cognitive task was performed from 3 min after the start of every increase in workload. In the control condition, the workload was fixed at 20 W, with a duration of 19 min 30 s. The control condition was included to determine the effects of time course on cognitive performance and the potential learning effects arising from repetition of the tasks (Ando et al. 2005). Cognitive tasks in the control condition were temporally matched with the tasks in the exercise condition. In the present study, we defined the first cognitive task in the control condition as block one (B1), the second cognitive task as block two (B2), and the third cognitive task as block three (B3).

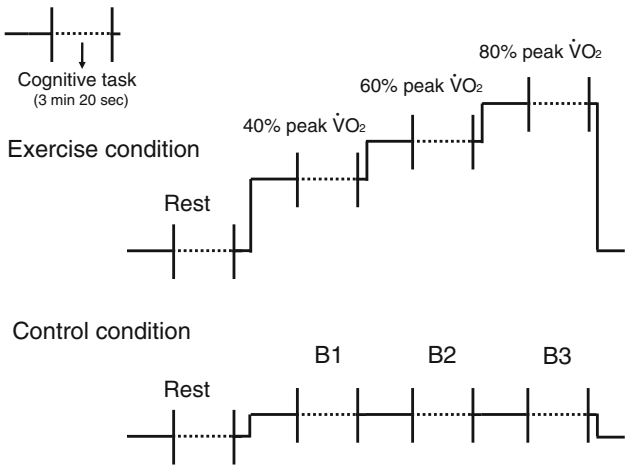

Fig. 1 Experimental protocol. *Dashed lines* indicate the duration of the cognitive task. Block 1 (*B1*), block 2 (*B2*), and block 3 (*B3*) represent the first, second, and third cognitive tasks during exercise in the control condition, respectively

EMG measurement and analysis

A surface EMG was recorded over the extensor pollicis longus muscles of the left and right forearms with a low cut filter of 5 Hz. This procedure allowed us to determine the onset of EMG activity in response to the central arrow without interference from muscle contraction during grasping of the handlebars. We divided RT into premotor and motor components (premotor time and motor time) based on the onset of EMG activity (Botwinick and Thompson 1966). We determined the EMG onset using a computer program combined with visual inspection. Details of the program for determining the EMG onset were previously described (Ando et al. 2009). In brief, the EMG signal was rectified from 500 ms before to 1,000 ms after the onset of the array of five arrows. The background EMG was averaged over 500 ms before stimulus onset. The mean background EMG was subtracted from the rectified EMG. After this, the rectified EMG was summed up over 1,000 ms after the onset, and the value set as 100%. We tentatively defined EMG onset as the point at which the cumulative sum of the rectified EMG reached a preset threshold of 5%. We changed this threshold with a step of 0.1% on the computer display until we confirmed by visual inspection that the EMG onset was appropriate.

In the present study, premotor time was the time from the onset of the array of five arrows to the onset of EMG activity. Motor time was the time from the onset of the EMG activity to the button release. Premotor time represents the amount of time needed by the central nervous system to process a visual stimulus, develop motor output, and send a motor command to the periphery (Laroche et al. 2007). Because premotor time can serve as a valid indicator for assessing effects of exercise on central processes (Ando et al. 2008, 2009), we used premotor time as a measure of effects of exercise on cognitive function.

Near-infrared spectroscopy (NIRS) measurement

Cerebral oxygenation was monitored with a NIRS system (BOM-L1 TRW, Omegawave, Tokyo, Japan). The principles of NIRS measurement were previously described (e.g. Subudhi et al. 2007; Villringer and Chance 1997). NIRS continuously measured concentration changes in oxyhemoglobin (oxy-Hb) and deoxyhemoglobin (deoxy-Hb). Total hemoglobin (total-Hb) was calculated by the sum of oxy-Hb and deoxy-Hb. Hemoglobin concentrations were corrected using an age-dependent differential path-length factor (Duncan et al. 1996). Cerebral oxygenation was

expressed as oxy-Hb/total-Hb × 100 (expressed as a percentage).

A probe holder was attached at the right side of the forehead (over the right frontal cortex), and a black cloth was wrapped around the probe holder to shield it from the light. The probe holder contained one light source probe and two detectors placed at 2 cm (detector 1) and a 4 cm (detector 2) from the source. The source generated three wavelengths of near-infrared light (780, 810, and 830 nm). Hemoglobin concentrations were calculated using NIR light received by each detector. The hemoglobin concentrations received by detector 1 were then subtracted from those received by detector 2, which allowed us to exclude effects of near-surface blood flow (e.g. skin blood flow) on hemoglobin concentrations in the cortical tissue. In both the exercise and control conditions, before the cognitive task at rest we measured hemoglobin concentrations and cerebral oxygenation for 30 s as a baseline, while participants were at rest on the ergometer. We used landmarks on the forehead (nasion, eyebrow, and hairline) to ensure that the probe holder was set at the same position on both experimental days.

Ventilatory and other measurements

Ventilatory parameters were measured using an Aeromonitor AE-300S (Minato Medical Science, Osaka, Japan). Breath-by-breath measurements of ventilation ($\dot{V}_E$) and $\dot{V}O_2$ were averaged every 15 s. We also measured tidal volume, end-tidal partial pressure of O_2 ($P_{ET}O_2$), and end-tidal partial pressure of CO_2 ($P_{ET}CO_2$). Although the difference between $P_{ET}CO_2$ and arterial partial pressure of CO_2 ($PaCO_2$) is negligible at rest, $P_{ET}CO_2$ overestimates $PaCO_2$ during exercise (Jones et al. 1979). Thus, we adjusted $P_{ET}CO_2$ values during exercise to provide a better reflection of $PaCO_2$ using the equation proposed by Jones et al. (1979). Because the duration of each RT measurement was 3 min and 20 s, we averaged ventilatory parameters over a 3 min 30 s period that included the duration of the cognitive task.

An electrocardiogram (ECG) was continuously recorded throughout the experiment to assess heart rate (HR). The analog outputs of the ECG, EMG, and EOG were connected to an amplifier (MEG-6100, Nihon Kohden, Tokyo, Japan) and digitized at a sampling rate of 1 kHz using a Powerlab analog-to-digital converter (ML785 Powerlab/8sp, A/D instruments Japan, Tokyo, Japan). We recorded ratings of perceived exertion (RPE; 6-20 Borg scale; Borg 1975) immediately after each cognitive task. Body temperature was recorded from the tympanic membrane. The ambient temperature was between 21 and 23°C, and the relative humidity was less than 40% in both the exercise and control conditions.

Statistical analysis

We defined omissions of a response and incorrect responses (e.g., response with the left thumb to the right central arrow) as error trials. The error rate was analyzed after an arcsine transformation to avoid violating the assumption of non-normality of distributions required by analysis of variance (ANOVA). We also excluded trials in which overt eye movements or eye blinks were detected. After these trials were excluded, premotor time was averaged for congruent and incongruent trails, respectively. Ventilatory parameters, HR, hemoglobin concentrations, and cerebral oxygenation during the RT measurement period were averaged for further analysis. Baseline values of hemoglobin concentrations and cerebral oxygenation were included in the ANOVA.

For premotor time, error rate, and motor time, we performed three-way repeated measures ANOVAs with task (congruent and incongruent), condition (exercise and control), and workload (exercise: rest, 40, 60 and 80%; control: rest, B1, B2 and B3) as factors. We performed two-way repeated measures ANOVAs on hemoglobin concentrations and cerebral oxygenation, with condition (exercise and control) and workload (exercise: baseline, rest, 40, 60 and 80%; control: baseline, rest, B1, B2 and B3) as factors. For the other variables, we performed two-way repeated measures ANOVAs with condition and workload as factors. The degree of freedom was corrected using Huyhn Feldt Epsilon when the assumption of sphericity was violated. We used Tukey's multiple comparisons test for post hoc analysis. Polynomial trend analysis was used to characterize trends in premotor time. In addition, we performed paired t tests with Bonferroni correction to compare the differences between the exercise and control conditions. All data are expressed as the mean ± SD. The level of significance was set at $P < 0.05$.

Results

Premotor time and motor time

Figure 2 illustrates the premotor time in the exercise (A) and control (B) conditions. A three-way ANOVA revealed a significant main effect of task ($F[1, 11] = 56.06$, $P < 0.001$, $\eta^2 = 0.84$). We observed no significant interactions involving task. These results indicate that the premotor time was longer in the incongruent trials than that in the congruent trials irrespective of the degree of exercise intensity. We found a significant interaction between condition and workload ($F[3, 33] = 2.92$, $P = 0.048$, $\eta^2 = 0.21$). Thus, we performed the ANOVA for the exercise and control conditions, respectively. In the exercise

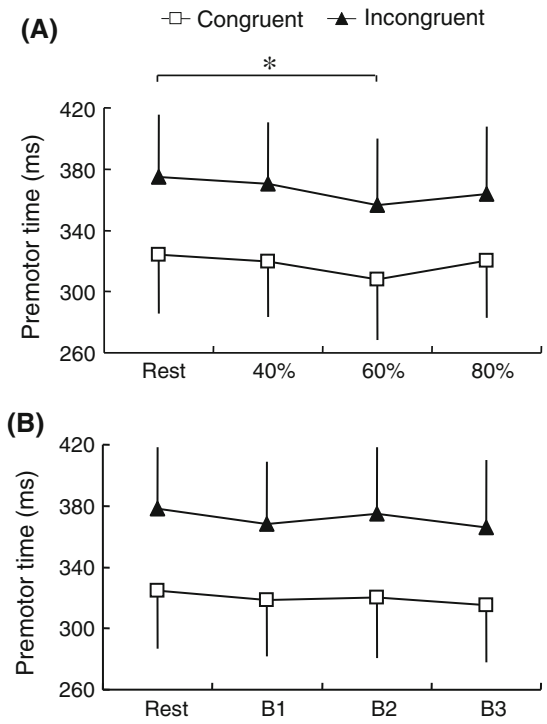

Fig. 2 The premotor time in the exercise (**a**) and control (**b**) conditions. *Filled triangles* represent incongruent trials. *Open squares* represent congruent trials. Data are expressed as mean ± SD. Significantly different between rest and 60%, $*P < 0.05$

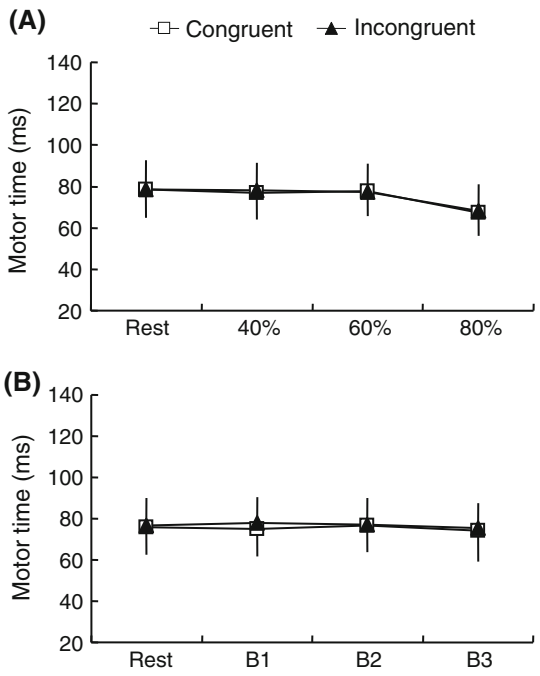

Fig. 3 The motor time in the exercise (**a**) and control (**b**) conditions. *Filled triangles* represent incongruent trials. *Open squares* represent congruent trials. Data are expressed as mean ± SD

condition, there was a significant main effect of workload ($F[3, 33] = 3.17$, $P = 0.037$, $\eta^2 = 0.22$). The premotor time during exercise at 60% peak $\dot{V}O_2$ (congruent: 307.8 ± 38.0 ms, incongruent: 356.7 ± 47.9 ms) was significantly shorter relative to that at rest (congruent: 324.0 ± 34.6 ms, incongruent: 375.0 ± 45.8 ms; $P = 0.028$). However, we did not observe any improvement in the premotor time during exercise at 80% peak $\dot{V}O_2$ ($P = 0.58$, vs. rest). Furthermore, trend analysis showed that the premotor time tended to change in a quadratic manner during exercise ($F[1, 11] = 3.78$, $P = 0.078$, $\eta^2 = 0.26$). In the control condition, there was no significant main effect of workload ($F[3, 33] = 1.13$, $P = 0.35$, $\eta^2 = 0.09$), which means that the premotor time did not change during exercise at 20 W in the control condition.

Figure 3 illustrates the motor time in the exercise (A) and control (B) conditions. In the exercise condition, the motor time tended to decrease during exercise at 80% peak $\dot{V}O_2$. However, a main effect of workload ($F[2.13, 23.46] = 3.29$, $P = 0.052$, $\eta^2 = 0.23$) and an interaction between condition and workload ($F[3, 33] = 2.54$, $P = 0.074$, $\eta^2 = 0.19$) failed to reach statistical significance. There was no significant main effect of task ($F[1, 11] = 1.82$, $P = 0.20$, $\eta^2 = 0.14$), indicating that the motor time was not different between congruent and incongruent trials.

Error rate

Table 1 shows error rate in the exercise and control conditions. A three-way ANOVA revealed a significant main effect of workload ($F[3, 33] = 2.94$, $P = 0.047$, $\eta^2 = 0.21$). We also observed a significant interaction between task and condition ($F[1, 11] = 7.48$, $P = 0.019$, $\eta^2 = 0.41$). Thus, we performed the ANOVA for the exercise and control conditions, respectively. We found significant main effects of task in the exercise ($F[1, 11] = 87.07$, $P < 0.001$, $\eta^2 = 0.89$) and control ($F[1, 11] = 15.74$, $P = 0.002$, $\eta^2 = 0.59$) conditions. These results indicate that the error rate in the incongruent trials was higher than in the congruent trials although the degree of difference was somewhat different between the exercise and control conditions.

NIRS measurement

Table 2 shows oxy-Hb, deoxy-Hb, and total-Hb in the exercise and control conditions. Analysis of oxy-Hb revealed a significant interaction between condition and workload ($F[4, 44] = 14.60$, $P < 0.001$, $\eta^2 = 0.57$). We observed a significant main effect of workload for the exercise condition ($F[4, 44] = 16.62$, $P < 0.001$, $\eta^2 = 0.60$). In the exercise condition, oxy-Hb increased during the cognitive task at rest from the baseline value ($P = 0.008$). By contrast, oxy-Hb during exercise at 80% peak $\dot{V}O_2$ was smaller than at baseline and B3 in

Table 1 Error rate (%) in the exercise and control conditions

Condition	Task	Rest	Exercise		
			40% peak $\dot{V}O_2$	60% peak $\dot{V}O_2$	80% peak $\dot{V}O_2$
Exercise	Congruent	0.4 ± 1.4	0.8 ± 1.9	0.4 ± 1.4	1.7 ± 3.1
	Incongruent	7.9 ± 6.9	7.1 ± 3.8	6.7 ± 7.7	10.0 ± 6.1
Condition	Task	Rest	B1	B2	B3
Control	Congruent	0.4 ± 1.4	0.4 ± 1.4	0.4 ± 1.4	1.7 ± 2.4
	Incongruent	7.1 ± 6.6	7.5 ± 6.3	3.3 ± 4.2	4.2 ± 4.5

Values are expressed as mean ± SD

Table 2 Oxy-Hb, deoxy-Hb, and total-Hb in the exercise and control conditions

Condition	Variables (μM)	Baseline	Rest	Exercise		
				40% peak $\dot{V}O_2$	60% peak $\dot{V}O_2$	80% peak $\dot{V}O_2$
Exercise	Oxy-Hb	14.14 ± 1.35	14.58 ± 1.22[a]	14.32 ± 1.29	14.37 ± 1.14	13.62 ± 1.26[a,f]
	Deoxy-Hb	10.74 ± 1.16	10.51 ± 1.02	10.45 ± 1.08	10.33 ± 1.25	10.94 ± 1.33[c,d,e]
	Total-Hb	24.87 ± 2.22	25.10 ± 1.97	24.78 ± 2.07	24.69 ± 2.00	24.56 ± 2.07
Condition	Variables (μM)	Baseline	Rest	B1	B2	B3
Control	Oxy-Hb	14.18 ± 1.98	14.36 ± 1.92	14.46 ± 2.01	14.47 ± 2.00	14.54 ± 2.04
	Deoxy-Hb	10.41 ± 1.59	10.33 ± 1.65	10.19 ± 1.52	9.95 ± 1.36[a]	9.88 ± 1.31[b]
	Total-Hb	24.60 ± 3.22	24.69 ± 3.28	24.65 ± 3.30	24.42 ± 3.14	24.42 ± 3.17

Values are mean ± SD

[a] $P < 0.01$, [b] $P < 0.001$ versus values at the baseline; [c] $P < 0.05$ versus value at 40%; [d] $P < 0.01$ versus value at 60%; [e] $P < 0.05$, [f] $P < 0.01$ versus values in the control condition

the control condition ($P = 0.002$ and $P = 0.001$, respectively).

Our analysis of deoxy-Hb revealed a significant interaction between condition and workload ($F[2.66, 29.24] = 6.30$, $P = 0.003$, $\eta^2 = 0.36$). We observed significant main effects of workload for the exercise ($F[2.99, 32.87] = 4.58$, $P = 0.009$, $\eta^2 = 0.29$) and control ($F[1.99, 21.83] = 8.26$, $P = 0.002$, $\eta^2 = 0.43$) conditions. In the exercise condition, deoxy-Hb during exercise at 80% peak $\dot{V}O_2$ was significantly greater than during exercise at 40% ($P = 0.034$) and 60% peak $\dot{V}O_2$ ($P = 0.004$). In the control condition, deoxy-Hb at B2 and B3 was significantly smaller than the baseline value ($P = 0.002$ and $P < 0.001$, respectively). Deoxy-Hb during exercise at 80% peak $\dot{V}O_2$ was significantly greater than that at B3 in the control condition ($P = 0.02$). Total-Hb did not change in the exercise and control conditions.

The results of our cerebral oxygenation analysis are shown in Fig. 4. The analysis revealed a significant interaction effect between condition and workload in terms of cerebral oxygenation ($F[2.60, 28.64] = 16.54$, $P < 0.001$, $\eta^2 = 0.60$). We observed significant main effects of workload in the exercise ($F[3.15, 34.59] = 13.28$, $P < 0.001$, $\eta^2 = 0.55$) and control ($F[2.10, 23.07] = 12.76$, $P < 0.001$, $\eta^2 = 0.54$) conditions. In the exercise condition, cerebral oxygenation increased at rest from the baseline value ($P = 0.047$). Cerebral oxygenation during exercise at 60% peak $\dot{V}O_2$ was greater than the baseline value ($P = 0.024$). By contrast, cerebral oxygenation during exercise at 80% peak $\dot{V}O_2$ was lower than at baseline ($P = 0.033$), at rest, and during exercise at 40 and 60% peak $\dot{V}O_2$ ($P < 0.001$, respectively). In the control condition, cerebral oxygenation at B1 was greater than the baseline value ($P = 0.014$). Cerebral oxygenation at B2 was significantly greater than at baseline ($P < 0.001$) and at rest ($P = 0.011$). Cerebral oxygenation at B3 was significantly greater than at baseline ($P < 0.001$), at rest ($P = 0.001$), and at B1 ($P = 0.045$). Cerebral oxygenation during exercise at 80% peak $\dot{V}O_2$ was significantly smaller than at B3 in the control condition ($P < 0.001$).

Ventilatory and other parameters

Table 3 summarizes the results of ventilatory parameters, HR, and RPE. In the exercise condition, $\dot{V}_E$ and $\dot{V}O_2$ progressively increased as workload increased ($P < 0.001$,

respectively). In the control condition, $\dot{V}_E$ and $\dot{V}O_2$ slightly increased during exercise relative to rest ($P < 0.001$, respectively), and remained unchanged throughout exercise. $\dot{V}_E$ and $\dot{V}O_2$ were consistently greater during exercise in the exercise condition than in the control condition ($P < 0.001$, respectively).

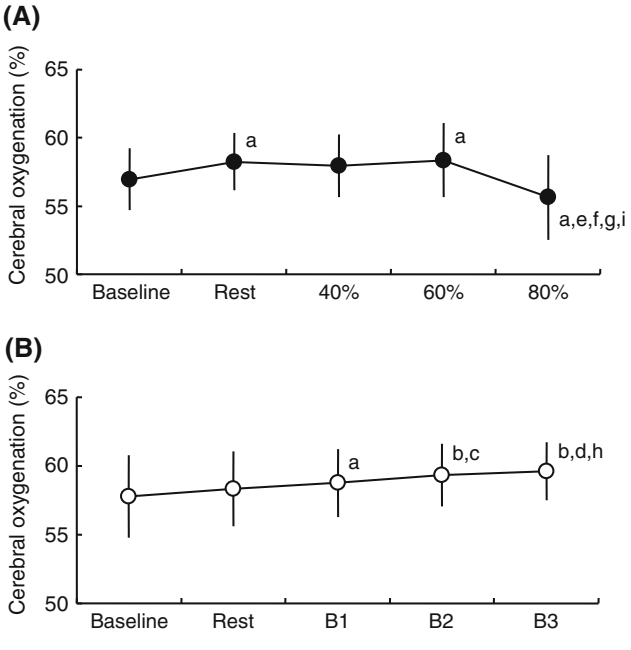

Fig. 4 Cerebral oxygenation in the exercise (**a**) and control (**b**) conditions. Data are expressed as mean ± SD. [a]$P < 0.05$, [b]$P < 0.001$ versus values at the baseline; [c]$P < 0.05$, [d]$P < 0.01$ versus [e]$P < 0.001$ versus values at rest; [f]$P < 0.001$ versus value at 40%; [g]$P < 0.001$ versus value at 60%; [h]$P < 0.05$ versus value at B1; [i]$P < 0.001$ versus value in the control condition

In the exercise condition, $P_{ET}O_2$ significantly decreased during exercise at 40% peak $\dot{V}O_2$ relative to that at rest ($P = 0.031$), while $P_{ET}O_2$ during exercise at 80% peak $\dot{V}O_2$ was significantly greater than at rest and during exercise at 40 and 60% peak $\dot{V}O_2$ ($P < 0.001$, respectively). In the control condition, $P_{ET}O_2$ slightly decreased at B1 in the control condition ($P = 0.015$). $P_{ET}O_2$ during exercise at 80% peak $\dot{V}O_2$ was greater than that in the control condition ($P < 0.001$).

In the exercise condition, $P_{ET}CO_2$ during exercise at 40% peak $\dot{V}O_2$ was greater than at rest ($P = 0.001$). However, $P_{ET}CO_2$ sharply decreased during exercise at 80% peak $\dot{V}O_2$. $P_{ET}CO_2$ during exercise at 80% peak $\dot{V}O_2$ was significantly smaller than at rest ($P = 0.034$) and during exercise at 40 and 60% peak $\dot{V}O_2$ ($P < 0.001$, respectively). In the control condition, $P_{ET}CO_2$ was consistently greater during exercise than that at rest ($P < 0.001$, respectively). $P_{ET}CO_2$ during exercise at 40% peak $\dot{V}O_2$ was greater than at B1 in the control condition ($P = 0.004$), while $P_{ET}CO_2$ during exercise at 80% peak $\dot{V}O_2$ was smaller than at B3 in the control condition ($P = 0.001$).

In the exercise condition, HR and RPE progressively increased as workload increased ($P < 0.001$, respectively). In the control condition, HR and RPE were significantly

Table 3 Ventilatory parameters, HR, and RPE in the exercise and control conditions

Condition	Variables	Rest	Exercise		
			40% peak $\dot{V}O_2$	60% peak $\dot{V}O_2$	80% peak $\dot{V}O_2$
Exercise	Workload (W)		87.1 ± 19.7	145.8 ± 26.6	202.9 ± 34.4
	VE (l/min)	11.7 ± 1.4	37.3 ± 6.0[c,g]	59.6 ± 11.2[c,d,g]	96.7 ± 18.9[c,d,e,g]
	$\dot{V}O_2$ (ml/kg/min)	4.6 ± 0.6	19.5 ± 4.2[c,g]	28.8 ± 5.8[c,d,g]	38.8 ± 6.9[c,d,e,g]
	$PETO_2$ (Torr)	104.4 ± 5.1	100.5 ± 3.2[a]	103.8 ± 2.3	110.8 ± 3.1[c,d,e,g]
	$P_{ET}CO_2$ (Torr)	37.4 ± 3.4	41.7 ± 2.6[b,f]	40.1 ± 2.3	34.5 ± 3.1[a,d,e,f]
	HR (bpm)	74.5 ± 10.1	112.6 ± 14.7[c,g]	145.1 ± 13.4[c,d,g]	178.0 ± 10.9[c,d,e,g]
	RPE	6.8 ± 1.4	10.0 ± 1.7[c,g]	13.4 ± 1.8[c,d,g]	16.8 ± 1.6[c,d,e,g]
Condition	Variables	Rest	B1	B2	B3
Control	VE (l/min)	11.4 ± 1.3	19.9 ± 1.3[c]	20.2 ± 1.8[c]	20.2 ± 2.1[c]
	$\dot{V}O_2$ (ml/kg/min)	4.6 ± 0.6	9.7 ± 1.5[c]	9.7 ± 1.4[c]	9.7 ± 1.7[c]
	$PETO_2$ (Torr)	103.7 ± 3.1	101.2 ± 3.0[a]	102.3 ± 2.6	102.1 ± 3.0
	$P_{ET}CO_2$ (Torr)	37.5 ± 3.3	39.9 ± 2.5[c]	40.0 ± 2.5[c]	39.8 ± 2.8[c]
	HR (bpm)	74.7 ± 8.6	86.0 ± 10.2[c]	87.9 ± 11.0[c]	87.9 ± 11.2[c]
	RPE	6.5 ± 0.9	7.7 ± 1.4[c]	8.3 ± 1.6[c]	8.5 ± 1.8[c]

Values are mean ± SD

[a] $P < 0.05$, [b] $P < 0.01$, [c] $P < 0.001$ versus values at rest; [d] $P < 0.001$ versus values at 40%; [e] $P < 0.001$ versus values at 60%; [f] $P < 0.01$, [g] $P < 0.001$ versus values in the control condition

greater during exercise than that at rest ($P < 0.001$, respectively). We observed significant differences in HR and RPE during exercise between the exercise and control conditions ($P < 0.001$, respectively). Body temperature did not differ at rest between the exercise (36.7 ± 0.4°C) and control (36.6 ± 0.4°C) conditions. In the exercise condition, body temperature significantly increased after exercise (37.6 ± 0.6°C, $P < 0.001$) relative to rest. In the control condition, body temperature after exercise (36.6 ± 0.5°C) was not different from that at rest, indicating that the exercise workload in the control condition was insufficient to induce an increase in body temperature.

Discussion

In the present study, cerebral oxygenation was measured during cognitive task performance at rest and during exercise. The results revealed that premotor time in the cognitive task decreased during exercise at 60% peak $\dot{V}O_2$ relative to rest. Cerebral oxygenation during exercise at 60% peak $\dot{V}O_2$ was no different to that at rest. These results indicate that the improvement in cognitive function during moderate exercise is independent of cerebral oxygenation. We found no improvement or deterioration in cognitive function during exercise at 80% peak $\dot{V}O_2$. It should be noted, however, that the results of cerebral oxygenation only relate to the right frontal cortex in the present study.

Premotor time is the time from the onset of a visual stimulus to muscle activation, and it reflects the central processing involved in RT (Botwinick and Thompson 1966). Hence, premotor time can be used as a valid indicator for assessing the effects of acute exercise on cognitive function. In the exercise condition, premotor time did not change during exercise at 40% peak $\dot{V}O_2$. This result indicates that light exercise was not sufficient to influence cognitive function. By contrast, exercise of a moderate intensity has been previously found to improve cognitive function (e.g. Chmura et al. 1998; Davranche et al. 2009; Joyce et al. 2009; Pesce et al. 2007). In the present study, premotor time significantly decreased during exercise at 60% peak $\dot{V}O_2$, in accord with previous reports. However, despite an improvement in cognitive function during exercise at 60% peak $\dot{V}O_2$, cerebral oxygenation during exercise at 60% peak $\dot{V}O_2$ was no different from that at rest. This result indicates that the improvement in cognitive function during moderate exercise was not directly associated with changes in cerebral oxygenation.

It has been suggested that an increase in arousal level induced by exercise leads to an improvement in cognitive function (Brisswalter et al. 2002; Tomporowski 2003). Indeed, the result that the premotor time tended to change in a quadratic manner during exercise seems to be in line with the inverted-U relationship between arousal level and cognitive performance (see details in Brisswalter et al. 2002; Tomporowski 2003). The present results suggest that increased arousal is not directly related to increased cerebral oxygenation. Exercise increases brain neurotransmitters such as catecholamine, serotonin, or dopamine (Meeusen et al. 2006). Increase in brain neurotransmitters may be one of the candidates that affect arousal level (Brisswalter et al. 2002; Chmura et al. 1994, 1998). However, it remains to be elucidated how moderate exercise affects cognitive function. Further studies are required to shed light on the physiological mechanisms underlying the improvement in cognitive function during exercise.

Error rate was higher in the incongruent trials than in the congruent trials, probably due to the nature of the task. In addition, we found a significant main effect of workload for error rate. This could be ascribed to relatively higher error rate during exercise at 80% peak $\dot{V}O_2$. Nevertheless, error rate was not affected by exercise at 60% peak $\dot{V}O_2$ in the present study. Hence, we can assume that the improvement in cognitive function during moderate exercise did not appear to be related to a speed-accuracy trade-off (Pachella 1974).

Cerebral oxygenation substantially decreased during exercise at 80% peak $\dot{V}O_2$. Since cerebral oxygenation reflects the balance between oxygen availability and utilization (Boushel et al. 2001), the decrease in cerebral oxygenation suggests that oxygen availability may be insufficient to meet metabolic demand. In the present study, beneficial effects of exercise on cognitive function were reduced during strenuous exercise. However, the premotor time at 80% peak $\dot{V}O_2$ was no different from that at rest. Further studies are required to assess whether decrease in cerebral oxygenation has detrimental effects on cognitive function in a variety of tasks. In the present study, decrease in cerebral oxygenation during exercise at 80% peak $\dot{V}O_2$ was accompanied by a decrease in adjusted $P_{ET}CO_2$. Pronounced hyperventilation induced by strenuous exercise lowers $PaCO_2$ (Nybo and Secher 2004). The decrease in adjusted $P_{ET}CO_2$ indicates that hyperventilation occurred during exercise at 80% peak $\dot{V}O_2$. Hyperventilation leads to constriction of the arterioles in the brain (Nybo and Rasmussen 2007; Querido and Sheel 2007) and may decrease cerebral oxygenation (Ando et al. 2010; Bhambhani et al. 2007; Secher et al. 2008). Hence, together with arterial de-saturation during strenuous exercise (Nielsen et al. 1999), hyperventilation is likely to contribute to the decrease in cerebral oxygenation we observed during strenuous exercise.

McMorris et al. (2009) examined the effects of acute incremental exercise on cognitive function. They reported

that cognitive function was impaired during strenuous exercise corresponding to 80% maximal aerobic power, in contrary to the present results. However, the type of visual stimulus used in the present study substantially differed from that in McMorris et al.'s (2009) experiment. In the current study, participants responded to the orientation of a central arrow embedded in an array of five arrows, whereas the task used by McMorris et al. (2009) required participants to respond to the color of the central target circle while ignoring the color of flanking circles. The response to the orientation of the arrow is analogous to a conventional two choice task and is less complex than responding to the color of a circle (Davranche et al. 2009). According to the transient hypofrontality hypothesis (Dietrich and Sparling 2004; Dietrich 2003), a greater impairment in cognitive function during endurance exercise would be expected in complex tasks that are known to heavily recruit prefrontal circuits. Thus, the simplicity of the task in the present study may account for the relatively small detrimental cognitive effects we observed during strenuous exercise. The discrepancy between previous studies and our present findings may thus be ascribed to differences in the type of visual stimulus used in the respective tasks.

In the exercise condition, the motor time tended to decrease during strenuous exercise. Previous studies have shown that motor time decreased during exercise at 50% maximal aerobic power (Davranche et al. 2005, 2006). They ascribed the decrease in motor time during exercise to better synchronization of motor unit discharge and at a lesser extent to efficient sensory processing (Davranche et al. 2009). These factors would account for the decrease in the motor time during exercise in the present study. Given that decrease in the motor time was exclusively observed during strenuous exercise, decrease in the motor time might be associated with increased catecholamine level during strenuous exercise (Mazzeo and Marshall 1989) and/or increased conduction velocity of muscle fiber induced by increase in body temperature (e.g., Stewart et al. 2003) .

In the control condition, premotor time did not change during exercise relative to rest after long habituation time had been undertaken. This indicates that premotor time was not affected by the time course, or potential learning effects. This finding supports the notion that the improvement in cognitive function in the exercise condition is related to physiological changes induced by moderate exercise. In the control condition, cerebral oxygenation exhibited a small but significant increase during exercise. This increase in cerebral oxygenation is likely to be due to factors affecting vascular tone. In particular, because adjusted $P_{ET}CO_2$ increased during exercise in the control condition, it is plausible that an increase in $PaCO_2$ induced vasodilatation and consequently increased cerebral oxygenation. In the control condition, an increase in cerebral oxygenation did not affect cognitive function. This result indicates that an increase in cerebral oxygenation does not directly improve cognitive function, supporting the notion that the improvement in cognitive function in the present study was independent of changes in cerebral oxygenation.

Several limitations in the present study should be acknowledged. First, we measured cerebral oxygenation over the right frontal cortex during exercise. Although the frontal cortex is activated in the flanker task (e.g. Colcombe et al. 2004), activity in the anterior cingulate cortex also plays a key role in response inhibition (Colcombe et al. 2004; Fan et al. 2005), which is a crucial component in the flanker task. Therefore, our interpretation is based on the assumption that the NIRS signal measured over the frontal cortex reflects cerebral oxygenation in discrete brain areas involved in performing the task. Second, although previous studies have reported good reproducibility of NIRS measurements during exercise (Koike et al. 2004; Subudhi et al. 2007), even a slight variation in probe placement can affect the measured hemoglobin concentrations (Subudhi et al. 2009). Because we measured cerebral oxygenation in two conditions on two separate days, we cannot rule out the possibility that a slight displacement of the probe position could have affected the relative changes in cerebral oxygenation we observed during exercise. Despite these limitations, our results confirm that NIRS is a useful tool to assess cerebral oxygenation in a non-invasive manner during exercise.

In summary, this study tested whether cerebral oxygenation affects cognitive function during exercise. We observed a significant improvement in cognitive function during moderate exercise. Cerebral oxygenation during moderate exercise was not different from that at rest. No improvement in cognitive function was observed during strenuous exercise, and cerebral oxygenation substantially decreased. The present results suggest that an improvement in cognitive function during moderate exercise is independent of cerebral oxygenation.

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