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The Influence of Exercise on Cognitive Performance in Normobaric Hypoxia

Yongsuk Seo,[1] Keith Burns,[1] Curtis Fennell,[1] Jung-Hyun Kim,[2] John Gunstad,[1] Ellen Glickman,[1] and John McDaniel[1,3]

Abstract

Seo, Yongsuk, Keith Burns, Curtis Fennell, Jung-Hyun Kim, John Gunstad, Ellen Glickman, and John McDaniel. The influence of exercise on cognitive performance in normobaric hypoxia. *High Alt Med Biol* 16:298–305, 2015.—Although previous reports indicate that exercise improves cognitive function in normoxia, the influence of exercise on cognitive function in hypoxia is unknown. The purpose of this study was to determine if the impaired cognitive function in hypoxia can be restored by low to moderate intensity exercise. Sixteen young healthy men completed the ANAM versions of the Go/No-Go task (GNT) and Running Memory Continuous Performance Task (RMCPT) in normoxia to serve as baseline (B-Norm) (21% O_2). Following 60 minutes of exposure to normobaric hypoxia (B-Hypo) (12.5% O_2), these tests were repeated at rest and during cycling exercise at 40% and 60% of adjusted Vo_{2max}. At B-Hypo, the % correct ($p \leq 0.001$) and throughput score ($p \leq 0.001$) in RMCPT were significantly impaired compared to B-Norm. During exercise at 40% ($p = 0.023$) and 60% ($p = 0.006$) of adjusted Vo_{2max}, the throughput score in RMCPT improved compared to B-Hypo, and there was no significant difference in throughput score between the two exercise intensities. Mean reaction time also improved at both exercise intensities compared to B-Hypo ($p \leq 0.028$). Both peripheral oxygen saturation (Spo_2) and regional cerebral oxygen saturation (rSo_2) significantly decreased during B-Hypo ($p \leq 0.001$) and further decreased at 40% ($p \leq 0.05$) and 60% ($p \leq 0.039$) exercise. There was no significant difference in Spo_2 or rSo_2 between two exercise intensities. These data indicate that low to moderate exercise (i.e., 40%–60% adjusted Vo_{2max}) may attenuate the risk of impaired cognitive function that occurs in hypoxic conditions.

Key Words: exercise; hypoxia; Go/No-Go task; Running Memory Continuous Performance Task

Introduction

Cognitive function while at altitude plays an important role in optimizing performance and safety during work and recreational activities. Due to the reduced partial pressure of oxygen, high altitude environments often induce arterial hypoxemia (Auerbach and Geehr, 1989), which leads to poor cognitive function (Crow and Kelman, 1973). Specifically, impairment of psychomotor performance, mental skills, reaction time, vigilance, memory, and logical reasoning has been reported at altitudes of 3000 meters and above (Denison et al., 1966; Crow and Kelman, 1971; Bahrke and Shukitthale, 1993). Cudaback (1984) reported a reduced ability to perform complex cognitive tasks in hypoxia before a compromised ability to perform simple tasks. This cognitive dysfunction can easily lead to dangerous outcomes, especially considering that individuals may be unaware of their compromised cognitive state. Therefore, developing a countermeasure to restore and/or improve cognitive function at altitude would likely reduce an individual's vulnerability to accidents.

Interestingly, previous studies have indicated that cognitive function is mediated by brain cortical activity (Crabbe and Dishman, 2004), which can be positively influenced by exercise. The possible explanations for this interaction between brain cortical activity and cognitive function may be increased metabolism, neural activity, and cardiovascular regulation during exercise (Koriath et al., 1987; Youngstedt et al., 1993; Nielsen et al., 2001).

[1]Department of Exercise Physiology, Kent State University, Kent, Ohio.
[2]National Personal Protective Technology Laboratory, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Pittsburgh, Pennsylvania.
[3]Louis Stokes Cleveland Veterans Affairs Medical Center, Cleveland, Ohio.

Specifically, previous studies indicated that alpha activity in brain is highly related with working memory (Başar et al., 1989; Klimesch, 1996) and the ratio of alpha activity to beta activity in the frontal lobe was significantly associated with an increase in core body temperature by exercise (Nielsen et al., 2001). Alpha activity also increases with cortical inhibition and cardiovascular control during exercise (Koriath et al., 1987). These improvements in cognitive function depend on the intensity (Hall et al., 2010) and mode (Schneider et al., 2009) of exercise.

For example, the choice reaction time, a test used to assess psychomotor speed (Overton et al., 2011), improved during exercise at up to 75% $V_{O_{2max}}$, with further increases in exercise intensity resulting in impaired reaction time (Chmura et al., 1994). These results were further supported by Brisswalter et al., (1997) who reported that moderate intensity exercise below lactate threshold can improve choice reaction time, whereas high intensity exercise leads to impaired cognitive function (Brisswalter et al., 2002). Chmura et al., (1998) also reported that during a sub-lactate threshold exercise protocol, choice reaction time improved through the first 40 min of exercise, after which choice reaction time remained constant for the remainder of the 60 min exercise protocol. Thus, it is possible that exercise, at low to moderate intensities, can also improve cognitive function in hypoxic conditions.

A better understanding of the interaction between cognitive performance and exercise in a hypoxic condition could prove beneficial to promote the health and performance of those who work and play in such conditions. Although improvements of cognitive performance have been observed during submaximal exercise (low to moderate intensities) in normoxia by numerous investigators (Baddeley, 2003; Sibley et al., 2006; Pontifex et al., 2009; Chang et al., 2012; Roig et al., 2013), to our knowledge few studies investigated the effect of acute exercise on cognitive function in hypoxia (Ando et al., 2013; Komiyama et al., 2015).

As such, the purpose of this investigation is to focus solely on hypoxic condition to determine if the impaired cognitive function in hypoxia can be restored by low to moderate intensity exercise by comparing physiological responses and indices of cognitive function during rest, and at 40% and 60% adjusted $V_{O_{2max}}$ in hypoxic conditions. Based on aforementioned literature, it was hypothesized that cognitive function would be impaired during rest in hypoxia, and low-moderate intensity exercise (40% and 60% $V_{O_{2max}}$) would restore cognitive function.

Methods

The Institutional Review Board at Kent State University approved this study and all subjects signed a consent form prior to participation. Sixteen young healthy men (24 ± 4 years of age; height = 176.5 ± 6.5 cm and weight = 78.3 ± 9.4 kg) volunteered for the current investigation. The subjects were excluded if they reported presence or history of pulmonary disease, cardiovascular disease, postural orthostatic tachycardia syndrome, skeletal muscle injury in the lower limbs, and metabolic disorders or were exposed to normobaric hypoxia or an altitude above 2500 m within 2 months prior to participation in the study. Women were not included in this initial study due to established gender differences in working memory under psychological stress (Schoofs et al., 2013).

Experimental procedures

Each participant reported to the laboratory on two separate occasions (familiarization trial and experimental trial). During the familiarization trial, participants underwent prescreening and were introduced to the simulated altitude chamber. Subjects were also familiarized with the protocol and instrumentation including performing the cognitive function tests a minimum of three times. Participants then performed two exercise protocols on a cycle ergometer (Lode Excalibur Sport, Lode, Groningen, Netherlands) to determine the submaximal exercise intensities that would be used during the subsequent experimental trial.

The first protocol required them to pedal through three 4 min stages at 50, 100, and 150 watts to develop their V_{O_2}–workrate relationship. Upon completion of the first protocol, participants rested for at least 20 minutes. The second protocol was a $V_{O_{2max}}$ test that required participants to pedal on the cycle ergometer through increasing stages of intensity, starting at 20 watts and increasing by 25 watts every minute (Amann et al., 2004) until volitional fatigue. During both protocols V_{O_2} and HR were measured with a metabolic cart (Parvo, Sandy, Utah) and a Polar heart rate monitor (Polar RS800 CX, Polar Electro Oy, Kempele, Finland), respectively. The combination of these two protocols allowed for the determination of $V_{O_{2max}}$ as well as the power output required to elicit 40% and 60% $V_{O_{2max}}$, which was ultimately reduced by 27% for the experimental trial to adjust for the $V_{O_{2max}}$ decrements with altitude (Young et al., 1985; Fulco et al., 1998). The adjusted 40% and 60% exercise intensities were selected as they span the range of exercise intensities previously reported to improve cognitive function at sea level (Reilly and Smith 1986; Arcelin et al., 1998; Tomporowski 2003).

On the day of the experimental trial (Fig. 1), participants reported to the Exercise Physiology Laboratory at Kent State University following a 3 hour self-reported fast intended to stabilize substrate utilization (Ruby and Robergs, 1994) and

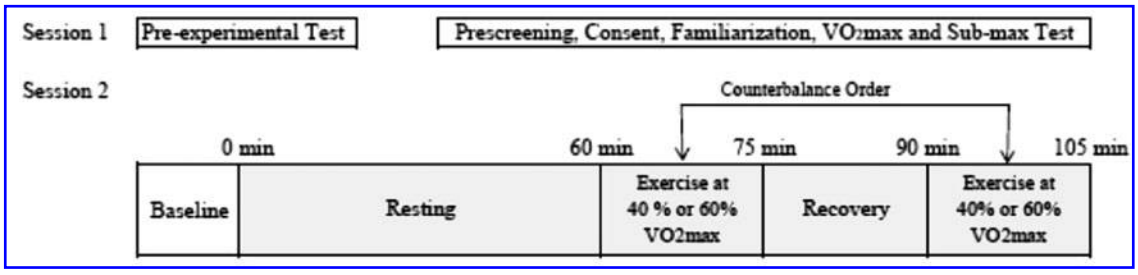

FIG. 1. Experimental design and procedure. Pre-experimental session (*upper*) and experimental session (*lower*). *Shaded gray area* occurred in hypoxic condition.

reduce the risk of subjects becoming nauseous during exercise in the hypoxic chamber. Participants were initially equipped with a HR monitor, mouthpiece for the metabolic cart, Near-Infrared Spectroscopy (NIRS) sensors over the frontal lobe (Somanetics, Troy, MI) for regional cerebral oxygen saturation (rSo_2) monitoring and digit pulse-oximeter (Oxi-Go, Roslyn, NY) for peripheral oxygen saturation (Spo_2) measurement. Subjects sat on a chair quietly during 5 min baseline recordings of resting metabolic rate, blood pressure, heart rate, Spo_2, and rSo_2. GNT and RMCPT were conducted via a computerized test battery.

Following baseline measurements (B-Norm), participants entered the hypoxic chamber (Colorado Altitude Training, Louisville, CO) where the oxygen concentration was reduced to 12.5% with similar increases in % N_2 but no changes in the % CO_2. The 12.5% O_2 is equivalent to the oxygen level present at an altitude of 4300 meters (14,110 feet). The room temperature and relative humidity in the hypoxic chamber were consistently 22°–24°C and 30%–40% throughout testing. After resting on a chair for 60 min in the hypoxic chamber cerebral oxygenation, Vo_2, HR, and Spo_2 were recorded, and GNT and RMCPT were administered to obtain hypoxic baseline measurements (B-Hypo).

Submaximal bouts of exercise

Following the completion of hypoxic baseline measurements, the participants performed two 15 min bouts of cycle ergometry at 40% and 60% of adjusted Vo_{2max} with 15 min recovery period between bouts. The pedaling rate (revolution per min) was freely chosen but workload was maintained automatically and exercise intensities were counterbalanced. All aforementioned cognitive tests were performed in order during the final 5 min of each of the two 15 minute exercise stages (or from 70–75 and 100–105 min into the start of the protocol) (Fig. 1). The 15 min recovery period was chosen based on previous studies indicating the effects of exercise on cognitive function dissipates within a few minutes following cessation of exercise (Audiffren et al., 2008; 2009; Labelle et al., 2013). Upon completion of the hypoxia trial, participants stepped out of the hypoxic chamber and rested until their Spo_2 returned to baseline levels.

Cognitive task measurement

Cognitive function was assessed through administration of specific subsets of the Automated Neuropsychological Assessment Metrics-4th Edition (ANAM4), a computerized cognitive performance test battery consisting of variety of cognitive domains. The ANAM4 has been administered to military and sports-related concussion, exposure to radiation, high altitude, undersea, and toxic conditions (Bleiberg et al., 2004; Eonta et al., 2011). Furthermore, the ANAM4 was developed to minimize the learning effect and provides randomized stimuli across testing sessions and almost limitless number of alternative forms and facilitate repeated-measures testing (Roebuck-Spencer et al., 2007; Eonta et al., 2011).

The specific subtests utilized in this study include the Go/No-Go Test (GNT) and Running Memory Continuous Performance Task (RMCPT), since we wanted to focus on inhibitory control and working memory. The working memory has been considered as fundamental cognitive function that retention of limited amount of information for short period time without presence of information in the environment (Smith et al., 1998). Furthermore, working memory is an emergent properties for language comprehensive, learning, planning, reasoning, and general intelligence (Baddeley, 1992). In addition, the Go/No-Go and RMCPT were also selected due to ease of performance while cycling on an ergometer. (Note these cognitive tests took approximately 118 ± 2 and 122 ± 3 sec, thus the total time was around 5 min for the cognitive tests.)

The GNT was designed to assess response inhibition where participants were instructed to click the left mouse button as quickly as possible in response to an ''X'' stimulus on the monitor that was presented at random intervals. If the monitor displayed ''O'' stimulus, they were instructed not to click the mouse button. The RMCPT performance measured short-term memory whereby participants were instructed to discriminate between two response alternatives (left or right button) if the displayed number matched or did not match the preceding number, respectively. The % correct and throughput scores were recorded. The throughput score was calculated as: Throughput score = number of correct response per minute.

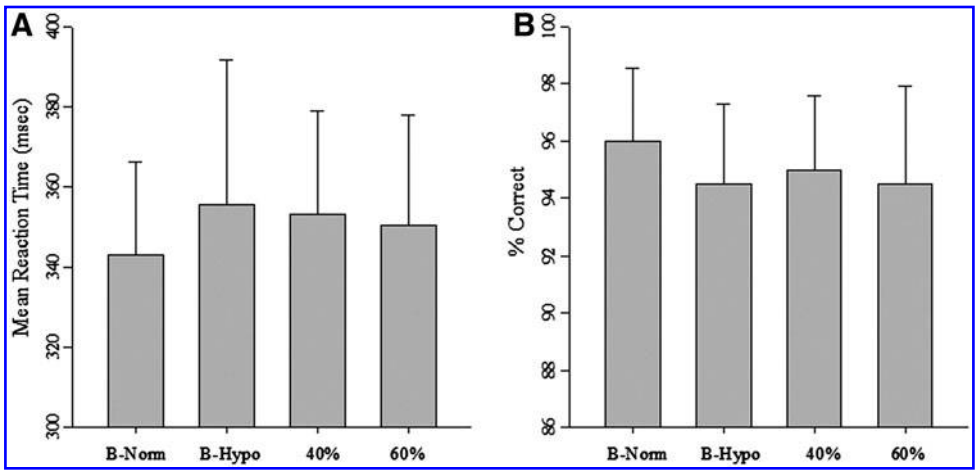

FIG. 2. Mean reaction time **(A)** and % correct in Go/No-Go **(B)** at baseline, following 60 minutes of rest in hypoxia and during exercise at 40% and 60% Vo_{2max} in hypoxia. There were no statistical differences across the four conditions.

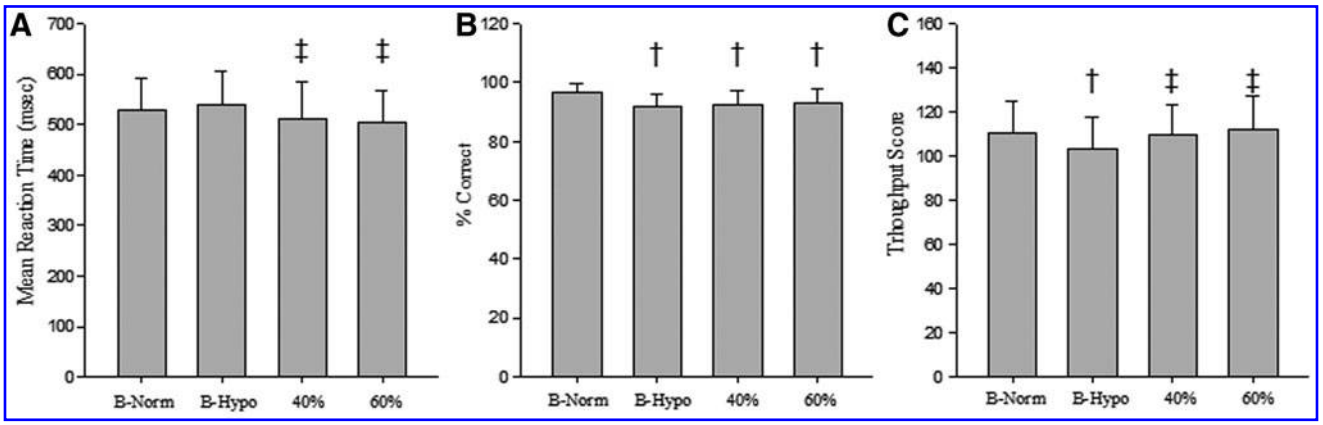

FIG. 3. Mean reaction time **(A)**, % correct **(B)**, and throughput score **(C)** in running memory continuous performance task at baseline, following 60 minutes of rest in hypoxia and during exercise at 40% and 60% Vo_{2max} in hypoxia. $^{\dagger}p < 0.05$, versus values at B-Norm; $^{\ddagger}p < 0.05$, versus values at B-Hypo.

Data analysis

Using SPSS 17.0, one-way repeated measures ANOVA was utilized for mean reaction time and correct response of GNT and mean reaction time, correct response and throughput score of RMCPT, Vo_2, BP, HR, Spo_2, and rSo_2 across conditions (B-Norm, B-Hypo, 40% and 60%). When the ANOVA indicated a significant main effect, post-hoc pair-wise comparisons with LSD was utilized to determine where those differences exist. The level of statistical significance was set at alpha ≤ 0.05 and all data are presented as mean $\pm$ SD.

Results

The average Vo_{2max} was 46.9 ± 7.7 and adjusted Vo_{2max} was 34.3 ± 5.6 ml/kg/min. The corresponding workload of 40% and 60% was 52 ± 16 and 101 ± 23 Watts, respectively.

Cognitive and physiological measurements

The reaction time and % correct in Go/No-Go demonstrated no significant main effect for condition ($F = 1.8$, $p = 0.168$), ($F = 2.2$, $p = 0.098$), respectively (Fig. 2A and 2B). The reaction time, % correct, and Throughput score (number of correct response per minute) in Running Memory Continuous Performance Tasks demonstrated a significant main effect for condition ($F = 3.4$, $p = 0.025$), ($F = 7.6$, $p \leq 0.001$), and ($F = 5.0$, $p = 0.005$), respectively (Fig. 3A–C). The dependent variables mean arterial pressure, heart rate, and oxygen consumption showed a main effects for condition ($F = 16.2$, $p \leq 0.001$), ($F = 185.8$, $p \leq 0.001$), and ($F = 206.1$, $p \leq 0.001$), respectively (Fig 4A–C). Regional cerebral oxygen saturation and peripheral oxygen saturation demonstrated a main effect for condition ($F = 196.9$, $p \leq 0.001$), and ($F = 225.2$, $p \leq 0.001$), respectively (Fig. 5A and 5B).

Cognitive tasks and physiological measurement at rest in hypoxia

The reaction time in Running Memory Continuous Performance Tasks was not impaired at rest in hypoxia compared to rest in normoxia ($p = 0.180$). However, there was an impairment of % correct ($p \leq 0.001$) and throughput score ($p \leq 0.001$) in Running Memory Continuous Performance Tasks at rest in hypoxia compared to rest in normoxia (Fig. 3A–C).

The mean arterial pressure, heart rate, and oxygen consumption in hypoxia did not differ compared to rest in normoxia ($p > 0.05$) (Fig. 4A–C). However, average of both left and right regional cerebral oxygen saturation ($p \leq 0.001$) and

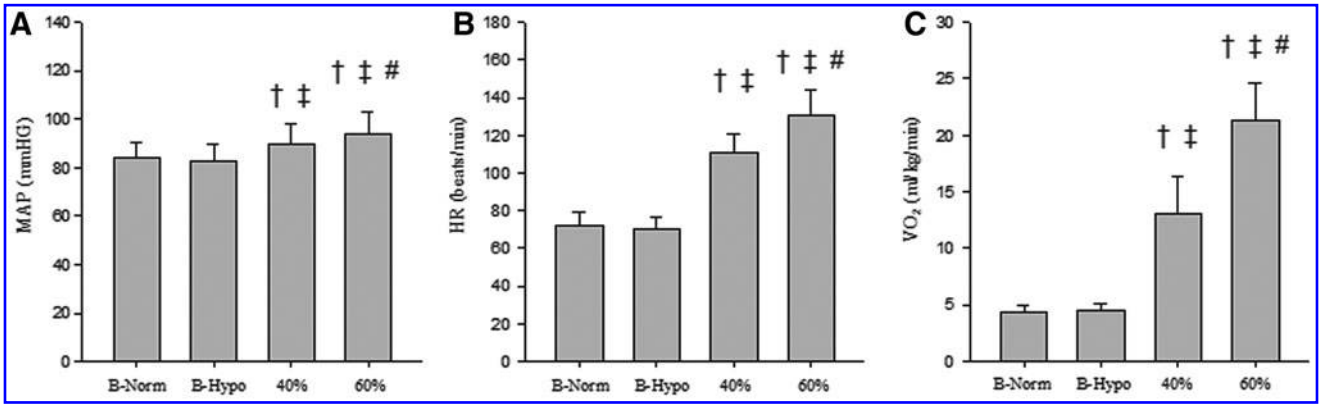

FIG. 4. Mean arterial pressure **(A)**, heart rate **(B)**, and oxygen consumption **(C)** at baseline in normoxia, following 60 minutes of rest in hypoxia and during exercise at 40% and 60% Vo_{2max} in hypoxia. Everything to the *right of the vertical dashed line* occurred in hypoxic conditions. $^{\dagger}p < 0.05$, versus values at B-Norm; $^{\ddagger}p < 0.05$, versus values at B-Hypo; $^{\#}p < 0.05$, versus values at 40%.

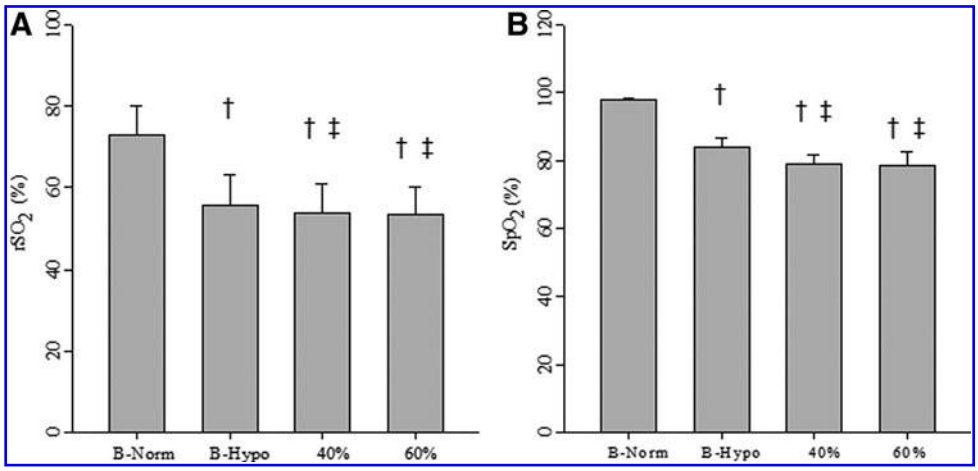

FIG. 5. Regional cerebral oxygen saturation **(A)** and peripheral oxygen saturation **(B)** at baseline in normoxia, following 60 minutes of rest in hypoxia and at 40% and 60% Vo_{2max} in hypoxia. Everything to the *right of the vertical dashed line* occurred in hypoxic conditions. $^{\dagger}p < 0.05$, versus values at B-Norm; $^{\ddagger}p < 0.05$, versus values at B-Hypo.

peripheral oxygen saturation ($p \leq 0.001$) was significantly reduced at rest in hypoxia compared to rest in normoxia (Fig. 5A and 5B).

Cognitive tasks and physiological measurement during exercises in hypoxia

During 40% and 60% of exercise, reaction time in Running Memory Continuous Performance Tasks significantly improved compared to rest in hypoxia ($p = 0.028$ and $p = 0.009$, respectively) and did not differ compared to rest in normoxia ($p = 0.256$ and 0.08, respectively) (Fig. 3A). The % correct in Running Memory Continuous Performance Tasks during 40% and 60% of exercise intensity remained significantly lower from rest in normoxia ($p = 0.001$, $p = 0.001$, respectively) but did not differ from rest in hypoxia ($p > 0.262$) (Fig. 3B). With regards to throughput score, 40% and 60% of exercise intensity improved performance compared to rest in hypoxia ($p = 0.023$ and 0.006, respectively) and were not significantly different compared to rest in normoxia ($p = 0.676$ and 0.522, respectively). There was no significant difference in throughput score between exercise intensities ($p = 0.329$) (Fig. 3C).

As expected, significantly higher mean arterial pressure, heart rate, and oxygen consumption were observed during both 40% and 60% of exercise compared to rest in normoxia ($p < 0.05$) and rest in hypoxia ($p < 0.05$). Furthermore, all cardiorespiratory parameters were significantly different between 40% and 60% bouts of exercise ($p < 0.05$). After 60 min resting in hypoxia, 40% and 60% of adjusted exercise further decreased cerebral oxygenation compared to rest in hypoxia ($p = 0.05$ and 0.04, respectively). There was no effect of exercise intensity in cerebral oxygenation ($p = 0.752$) (Fig. 5A). Following rest in hypoxia, peripheral oxygen saturation was further reduced significantly during 40% and 60% of exercise ($p \leq 0.001$, for each comparison) compared to rest in hypoxia. Peripheral oxygen saturation was also not different between the two exercise intensities ($p = 0.824$) (Fig. 5B).

Discussion

We hypothesized that cognitive performance (i.e., GNT and RMCPT) would be impaired after 60 min of resting in hypoxia, but exercise at 40% and 60% of adjusted Vo_{2max} would restore and/or improve the performance to near normoxic values. The main findings of this study were: (1) 60 min of hypoxia exposure in a resting condition resulted in impairment of some of our indices of cognitive function. Specifically, % correct and throughput score in RMCPT were significantly impaired compared with rest in normoxia; (2) low and moderate exercise performed in hypoxia restored throughput score in RMCPT, but not %correct; (3) although mean reaction time was not impaired at rest in hypoxia, exercise at both 40 and 60% improved reaction time; (4) at least in the current study, reaction time and % correct in GNT were not sensitive enough to assess cognitive impairment in hypoxia; (5) there was no significant difference between the two exercise conditions on improving cognition.

Cognitive and physiological measurements at rest in hypoxia

Previous investigators have reported that cognitive function was impaired after 60 min of hypoxia exposure and complex cognitive tasks were impaired before simple tasks (Cudaback 1984; Adam et al., 2008; Hewett et al., 2009; Kryskow et al., 2013). The data support these previous investigations in that some of our indices of cognitive function were impaired during at the B-Hypo compared to B-Norm. Specifically, we did not observe a significantly impaired mean reaction time and % correct in GNT, which is based primarily on motor reaction time (Etnier and Chang 2009; Lambourne and Tomporowski, 2010; Komiyama et al., 2015). However, our data indicated a significantly impaired % correct and throughput score of RMCPT at the B-Hypo compared to B-Norm. The % correct and throughput score of RMCPT are influenced by reaction time as well as attention, concentration, and working memory.

It appears that a speed-accuracy trade off occurs with fewer correct responses with as unchanged or slightly increased mean reaction times during both GNT and RMCPT. Inhibitory control and working memory represent two facets of executive function that individually depend on distinctive brain regions. The inhibitory control tasks has been shown to involve both the right inferior frontal cortex (rIFC) (Aron

et al., 2014) and the anterior cingulate cortex (Botvinick et al., 2004) while working memory involves the dorsal lateral prefrontal cortex (dlPFC) and parietal regions (Pontifex et al., 2009).

The current study suggested that brain regions in working memory process might be more sensitive to hypoxia and moderate intensity exercise than inhibitory control. The results from RMCPT are in agreement previous studies, indicating that participants made more errors and increased mean reaction time in hypoxic conditions (Van Diest et al., 2000). Results from Asmaro et al., (2013) also supports these findings in that reaction time and correct responses were decreased on the Word-Color Stroop task, which includes attention and concentration rather than simply reaction time.

In the current study, rSo_2 significantly decreased during rest in hypoxia which is in agreement with previous reports (Ando et al., 2013; Komiyama et al., 2015). Cerebral oxygenation was further decreased during exercise in hypoxia which confirms the previous finding of exercise-induced reduction in rSo_2 in mild hypoxia (15% and 18% O_2). However, we did not observe a significant further reduction in rSo_2 ($0.4 \pm 4.7\%$) as exercise progressed from 40% to 60% of Vo_{2max}. This result is somewhat in contrast to the previous study by Subudhi et al., (2007) who reported progressive reduction in rSo_2 during incremental maximal exercise, and Imray et al., (2005) who reported a progressive but very small reduction in cerebral oxygenation during progressive submaximal exercise at 30, 50, and 70% of Vo_{2max} (61.4 ± 2.2, 60.6 ± 2.0, and $58.9 \pm 2.1\%$, respectively) in a similar hypoxic condition (4750 m).

Similar to rSo_2, Spo_2 also decreased with exposure to hypoxia in the resting condition. Kryskow et al., (2013) observed that after 8 and 30 hours of altitude exposure, Spo_2 decreased compared normoxic conditions (78 ± 8 and 79 ± 6, respectively). Furthermore, Gomes et al., (2013) reported that after 5–10 min of hypoxia exposure (12%–15% Fraction of inspired O_2) Spo_2 was reduced at rest in healthy subjects. In this investigation, other physiological measurements (HR, MAP, and Vo_2) were not significantly different compared to baseline.

Cognitive and physiological measurements during exercise in hypoxia

The mean reaction time and % correct of GNT were not improved during either 40% or 60% exercise intensities compared to B-Hypo. Thus it appears that mean reaction time and % correct from GNT were not sensitive enough to show changes with hypoxia. Furthermore, exercise in hypoxia also did not alter the values of these specific cognitive function tests. However, the two exercise conditions improved mean reaction time compared to rest in hypoxia and restored throughput score of RMCPT back to levels equal to those in the resting normoxia condition. Furthermore, there was no significant difference in cognitive function between the two exercise intensities. The results from the RMCPT are in agreement with previous studies, indicating that dynamic exercise at low to moderate intensities of short duration improve cognitive function in normoxia (Brisswalter et al., 1995; 1997; Arcelin et al., 1998; Ando et al., 2013; Martins et al., 2013). Although exercise did not improve or restore % correct, it also did not further impair this aspect of cognitive function.

All physiological measurements were statistically different between two exercise intensities including HR, Vo_2, and MAP, with the highest values being obtained during the 60% condition. These results are to be expected during the higher exercise intensity as a greater metabolic demand, induces a greater physiological response. Interestingly, during both 40% and 60% Vo_{2max} exercise, both cerebral oxygenation and Spo_2 were reduced compared to the B-Hypo (Fig. 5). This decrease in cerebral oxygenation and peripheral oxygen saturation with exercise and partial improvement, or at least maintenance of cognitive performance indicates that oxygen saturation and cognitive function becomes dissociated under hypoxic conditions.

There are several possible explanations for the improvement in cognitive function despite a reduction in rSo_2 and Spo_2. First, exercise stimulates the central nervous system (CNS), which ultimately improves psychomotor performance (Chmura et al., 1994). Several investigations suggested that improvements of cognitive performance result from adjustment of brain neurotransmitter such as dopamine, noradrenaline, serotonin, and adrenocorticotropic (ACTH) with onset of exercise (Tomporowski, 2003; Davranche and Audiffren, 2004; McMorris, 2009). Another possible explanation is that exercise induces arousal which improves cognitive function (Lambourne and Tomporowski, 2010). The exercises also increase the production of cortisol which inhibits the synthesis of corticotrophin and ACTH. As exercise intensity and duration increase, arousal levels also increase until cognitive performance is limited by strenuous exercise (Lambourne and Tomporowski, 2010). More research is needed to elucidate which specific mechanism(s) may be offsetting the decrease in cerebral oxygenation.

The current study is limited by a few factors. A limitation of this study may be the small sample size, although previous study reported improvement in reaction time in Go/No-Go task with sixteen and twelve participants (Ando et al., 2013; Komiyama et al., 2015). Indeed, retrospective power analysis with the current 16 subjects, determined based on the analysis of the reaction time (96.1 ± 2.5 vs. 94.5 ± 2.8), the percent correct (96.5 ± 3.0 vs. 91.8 ± 4.5), and throughput score (110 ± 14.3 vs. 103.6 ± 14.8) in RMCPT, showed the study power of 0.73, 0.98, and 0.88, respectively. Second, this study recruited only young Caucasian men. Hence, a more diverse range (e.g., gender, age, and ethnicity) need to be evaluated to generalize the results. Third, it might be beneficial to assess unaltered Vo_{2max} values or absolute power values and exercise intensity in the hypoxic condition. Lastly, additional measurement of physiological parameters (e.g., middle cerebral artery, sympathetic nerve activity) and pharmacological/nutritional strategies could be used for future experiments to assess the cause-and-effect mechanism and determine if cognitive performance correlated to physiological parameters.

Conclusion

The present findings suggest that, despite further reductions in cerebral oxygenation, low to moderate intensity exercise (40%–60%) in hypoxia does not further impair inhibitory response and working memory within ANAM4 and may likely benefit specific aspects of cognition. Additional studies are required to determine the breadth in which exercise can restore the cognitive dysfunction often observed in hypoxic conditions.

Author Disclosure Statement

The findings and conclusions of this article are those of the authors and do not necessarily represent the views of NPPTL/NIOSH/CDC. Mention of commercial products does not constitute endorsement by NPPTL/NIOSH/CDC.

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Address correspondence to:
Dr. Yongsuk Seo
Department of Exercise Physiology
Kent State University
Gym Annex 167
Kent, OH 44242

E-mail: yseo@kent.edu

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Abbreviations Used

B-Hypo = baseline hypoxia
B-Norm = baseline normoxia
GNT = Go/No-Go Task
HR = heart rate
MAP = mean arterial pressure
Vo_2 = oxygen consumption
Spo_2 = peripheral oxygen saturation
rSo_2 = regional cerebral oxygen saturation
RMCPT = Running Memory Continuous Performance Task