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The Effect of Acute Hypoxia on Visual Evoked Potentials and Color Vision

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Abstract

Exposure to reduced levels of breathable oxygen results in hypoxic hypoxia. In tactical aviation, hypoxia is recognized as a serious potential risk. One of the most well-documented and robust effects of hypoxia is on the visual system, which has profound implications for human performance during aviation. Specifically, hypoxia is known to impair color perception, perception of light intensity, and result in symptoms that include tunnel vision, graying, and blurry vision. However, the mechanism by which vision is impaired during hypoxia is poorly characterized. The current study sought to specifically examine S-cone sensitivity along with visual evoked potentials (VEPs) during a moderate and severe normobaric hypoxia exposure. Participants completed a modified blue cone contrast test (CCT) as well as a pattern reversal paradigm while electroencephalography (EEG) was recorded to measure visual evoked potentials. Participants completed these tasks during three separate, counterbalanced visits that only differed in the oxygen concentration they were exposed to: normoxia (21% oxygen), moderate hypoxia (11.7% oxygen), or severe hypoxia (9.7% oxygen). Results showed null effects on both CCT performance and VEP amplitude by condition. Hypoxia symptom reporting did demonstrate high intra-individual variability in subjective symptom reporting across the two altitudes used here. Future work should continue to investigate the mechanism(s) of visual impairment during acute hypoxia while taking into consideration lighting conditions and stimuli that are most relevant to the operational environment.

The Effect of Acute Hypoxia on Visual Evoked Potentials and Color Vision

Exposure to reduced levels of breathable oxygen results in hypoxic hypoxia. In tactical aviation, hypoxia is recognized as a serious potential risk. Current military aircraft have warning systems only for the oxygen concentration provided upstream in the life support system and the gas reaching the pilot is not tested. This threat to aviators has performance implications because hypoxic exposure is known to impair a number of sensory, cognitive, and motor processes (Fowler et al., 1987; Malle et al., 2013; Blacker & McHail, 2021). Indeed, prior work has shown that acute hypoxia impairs the ability to maintain a constant airspeed, altitude, and directional heading (Steinman et al., 2017; Temme et al., 2010).

One of the most well-documented and robust effects of hypoxia is on the visual system (Hovis et al., 2013), which has profound implications for human performance during aviation. Specifically, hypoxia is known to impair color perception (e.g., Vingrys & Garner, 1987; Barbur & Connolly, 2011), perception of light intensity (Fowler et al., 1993), and result in symptoms that include tunnel vision, graying, and blurry vision (e.g., Sausen et al., 2001). However, the mechanism by which vision is impaired during hypoxia is poorly characterized. The visual system is sensitive to its oxygen supply at multiple levels including the retina, photoreceptors, and cortical and sub-cortical pathways. Estimates of visual system projections throughout the cortex are thought to be in the hundreds, with interactions beyond visual cortex into areas such as frontal, temporal, parietal lobes, and the midbrain (Mather, 2016). Therefore, it is unknown where and when the visual system is being disrupted during low oxygen exposure and/or how changes in oxygen supply at one level (e.g., the retina) changes functioning at downstream levels (e.g., primary visual cortex).

Testing the effects of hypoxia on the visual system using behavioral paradigms that require a response by the participant complicate matters because it is also well-established that reaction time (RT) is slowed during acute hypoxia (Fowler et al., 1993; Fowler et al., 1992; Fowler et al., 1987; Phillips et al., 2015; Blacker & McHail, 2021). One approach to eliminate the issue of an overt response is to use passively elicited event-related potentials (ERPs) measured with electroencephalography (EEG). Previous work has shown that an ERP known as the visual mismatch negativity (MMN) is disrupted during acute hypoxia (Blacker et al., 2021). In this study, the visual MMN was elicited by using a visual oddball paradigm where a reversing checkerboard was presented bilaterally to the peripheral visual fields. The majority of checkerboard stimuli were a standard color (e.g., green) while occasional “oddball” checkerboards (e.g., red) were presented. The visual MMN is a difference wave contrasting the neural response to the oddball stimuli compared to the standard stimuli and results in a negative going waveform that peaks at approximately 150 ms (for a review see, Pazo-Alvarez et al., 2003). Blacker and colleagues (2021) found a significant reduction in the amplitude of the visual MMN during an acute hypoxia exposure, equivalent to 17,500 ft, compared to a normoxia control. The visual MMN is thought to represent preconscious attentional orienting following a pattern violation.

There are earlier ERPs in the visual information processing stream that might also be disrupted during low-oxygen exposure. At least one prior study examined the effects of acute hypoxia on visual evoked potentials (VEPs) using a pattern reversal paradigm. Blacker and McHail (2022) compared passively elicited visual P100 components during normoxia and hypoxia. After an adjustment for low frequency noise that influenced the amplitude of the preceding N75 and subsequent N135 components, the effect of hypoxia was not statistically significant. Therefore, the full scope of visual impairment during acute hypoxia remains unclear and the mechanisms of that impairment are still unknown. The current study sought to specifically examine S-cone sensitivity along with VEPs during a moderate and severe normobaric hypoxia exposure.

Method

Participants

A total of 31 healthy adults (age: *M*=31.19 *SD*=4.98; 20 males) participated for monetary compensation. All participants were recruited through flyers and online announcements. The study protocol was approved by the Naval Medical Research Unit – Dayton's (NAMRU-D) Institutional Review Board in compliance with all applicable federal regulations governing the protection of human participants. All participants self-reported normal or corrected-to-normal vision (including color vision), no history of psychological, neurological, or medical diagnosis, no use of tobacco in the past 6 months, and no excessive alcohol use. The number of participants who reported previously experiencing hypoxia was *n*=19 (i.e., through previous NAMRU-D hypoxia studies and/or flight crew or other training).

Stimuli and Tasks

Modified Cone Contrast Test

A 60 Hz NEC PW-232w color-critical monitor with a resolution of 1920 x 1080 was used to display the experimental stimuli. A modified Cone Contrast Test (CCT) modeled after that used by Gaska et al. (2016) was used to test each participant's S-cone color vision. The CCT used a Landolt C task in which the participant was instructed to respond with the direction of the opening in the letter C (i.e., left, right, top, bottom). Each stimulus subtended a visual angle of 1.2°. During the initial visit, before any altitude exposure, participants completed a baseline threshold test consisting of 80 trials. For the baseline, stimuli were displayed on the screen for 100 ms and participants had 2500 ms to respond. The interstimulus interval was 500 ms and the size of the gap in the Landolt C stimuli was 1/5th the diameter of the C. The baseline began at a contrast level of 8% and then used an adaptive staircase procedure as follows. Three correct sequential trials resulted in a decrease in contrast of three steps, whereby each step decreased by 0.25% contrast. An incorrect trial resulted in an increase in contrast by one step. We then defined each participant's threshold as the lowest contrast level at which they maintained an accuracy of 62.5% or better.

During the experimental exposure portions, participants completed four blocks of the CCT task. Each block contained 12 trials at their threshold, 12 trials one step above their threshold, and 12 trials one step below their threshold. The order of these trial types was randomly presented within each block. Each trial began with a 400 ms fixation cross, followed by the Landolt C stimulus appearing for 100 ms. The participant had 1600 ms maximum to respond. Accuracy and response time (RT) were the dependent variables of interest.

Checkerboard Reversal Paradigm

Participants were presented with four blocks of flickering checkerboard stimuli (Halliday et al., 1973). Presented on a gray background, the stimuli consisted of a black and white checkerboard pattern, presented centrally, where the black and white squares switched at a rate of 1 Hz. The total checkerboard subtended a visual angle of 11°. Participants saw 16 reversals, followed by a 12 sec fixation cross (i.e., rest). Five iterations of 16 sec on and 12 sec rest were presented per block. This resulted in a total of 320 reversals per experimental visit. Participants were instructed to fixate centrally during these blocks.

Hypoxia Symptom Questionnaire (HSQ)

The 15-item Hypoxia Symptom Questionnaire (HSQ; Sausen et al., 2001) was used to assess participants’ hypoxia symptoms after each exposure. The HSQ was developed directly from the didactic portion of the U.S. Navy’s hypoxia familiarization training program and contains items related to commonly experienced hypoxia symptoms (tingling, hot flashes, cold flashes, dizziness, tunnel vision, loss of coordination, loss of consciousness, light dimming, euphoria, headache, fatigue, breathlessness, blurred vision, nausea, and apprehension). Participants rated each item on a 4-point scale (0 = not observed, 1 = mild, 2 = moderate, 3 = severe).

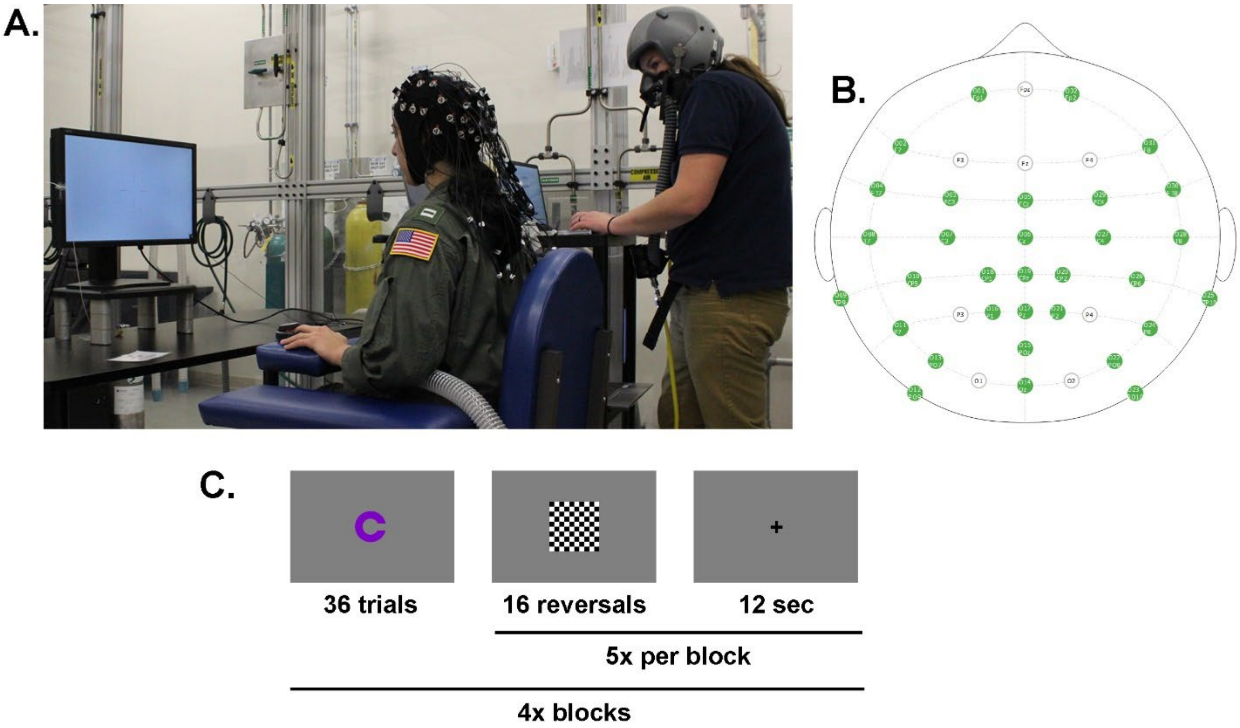

Figure 1. A) Experimental setup in the Reduced Oxygen Breathing Environment (ROBE). B) Custom 32-channel EEG montage. C) Task parameters. Participants completed 36 CCT trials followed by 5 iterations of 16 checkerboard reversals and 12 sec of rest. Each of these blocks was completed 4 times. *Photo courtesy of NAMRU-D.*

Procedures

Participants completed three sessions on separate days that only differed by the oxygen content they were exposed to: normoxia (21% oxygen), moderate hypoxia (11.7% oxygen; equivalent to 15k ft), and severe hypoxia (9.7% oxygen; equivalent to 20k ft). The order of conditions was counterbalanced and participants were blinded until their participation was complete.

During the first session and following informed consent, participants completed an initial screening questionnaire, a demographics questionnaire, a blood draw to check hemoglobin and hematocrit levels for safety, and the CCT baseline to determine their S-cone threshold. Participants were then outfitted with an elastic cap that contained the EEG electrodes. Exposure and testing all took place inside NAMRU-D's Reduced Oxygen Breathing Environment (ROBE;

Hypoxico, Inc) under photopic visual conditions. The ROBE is a normobaric hypoxia chamber (Figure 1A). The ROBE works by drawing in ambient room air and separating the oxygen molecules from the nitrogen molecules via a zeolite molecular sieve bed. The trapped, unwanted oxygen is exhausted while the hypoxic air, with a specific oxygen content, is pumped into the enclosure.

In the ROBE, participants were seated 90 cm from the NEC monitor. Each exposure and testing period lasted 14 minutes. Participants completed four interleaved blocks of the CCT and the checkerboard stimuli (Figure 1C). After the exposure, participants completed the HSQ and were asked to guess which condition they had experienced (i.e., normoxia, moderate hypoxia, severe hypoxia). Following the final visit, participants were debriefed.

Data Acquisition and Analysis

Event-Related Potentials

EEG data were recorded continuously from 32 electrodes covering the whole scalp using a custom montage (Figure 1B; ActiCHamp, Brain Products) referenced to the right mastoid (TP9) in DC mode, at a sampling rate of 1000Hz. Electrode impedance for all channels were kept below 25 kΩ.

EEG data were processed using the Fieldtrip software package (Oostenveld et al., 2011). Data were segmented into epochs covering the time from 100ms before to 900ms after the onset of each checkerboard reversal. After trial epochs were created, data were low-pass filtered at 20Hz, high-pass filtered at 1Hz, and re-referenced to FCz. Independent components analysis (ICA) was performed on epoched data and the eye blink component and lateral eye movement component (if present) were removed for every participant. After ICA, EEG waveforms from frontal electrodes (i.e., Fp1, Fp2) were visually inspected to identify voltage fluctuations typical of gross motor movements (amplitude > ±50μV). Trials containing these types of artifacts were rejected entirely.

After artifact rejection, average waveforms were calculated for electrode Oz. For each dataset, the P100 was defined as the most positive going waveform at electrode Oz between 50 and 110 ms. To compromise between peak- and mean-based measures, as described in Kappenman and Luck (2016), we reported the mean amplitude in a 20 ms window centered around the peak, such that the window varied for each dataset. We also examined the N75 and N135 components. The N75 was defined as the most negative going waveform preceding the P100 between 60 and 90 ms. The N135 was defined as the most negative going waveform following the P100 between 110 and 190 ms. Finally, we calculated N75-P100 peak-to-peak amplitude, as well as P100-N135 peak-to-peak amplitude.

Physiological Measures

During all study visits, both peripheral oxygen saturation (SpO_2) and heart rate (HR) were monitored and recorded at a sampling rate of 1 Hz. Both measures were acquired via a Nonin finger-mounted pulse oximeter (Nonin Medical Inc.) and recorded by an iPad via Bluetooth connection. A safety cut-off criterion of 55% SpO_2 was used.

Results

Our analyses focused on three main questions of interest: 1) did CCT performance change with condition, 2) did varying hypoxia conditions effect the amplitude of VEPs, and 3) were participants' hypoxia symptoms consistent across the two varying altitude exposures? In addition, the Greenhouse-Geisser correction was applied where Mauchly's test showed that the sphericity assumption was violated.

CCT

The dependent variables of interest for the CCT were accuracy and RT. RT was calculated only for correct trials. Two participants had missing data due to technical errors and are not included in any of the below analyses. To examine changes in performance across the different conditions, a 3 (condition: normoxia, moderate hypoxia, severe hypoxia) × 3 (threshold:

at, above, below) repeated-measures ANOVA was tested. Participants with accuracy ±2 SD from the group mean were excluded (*n*=6).

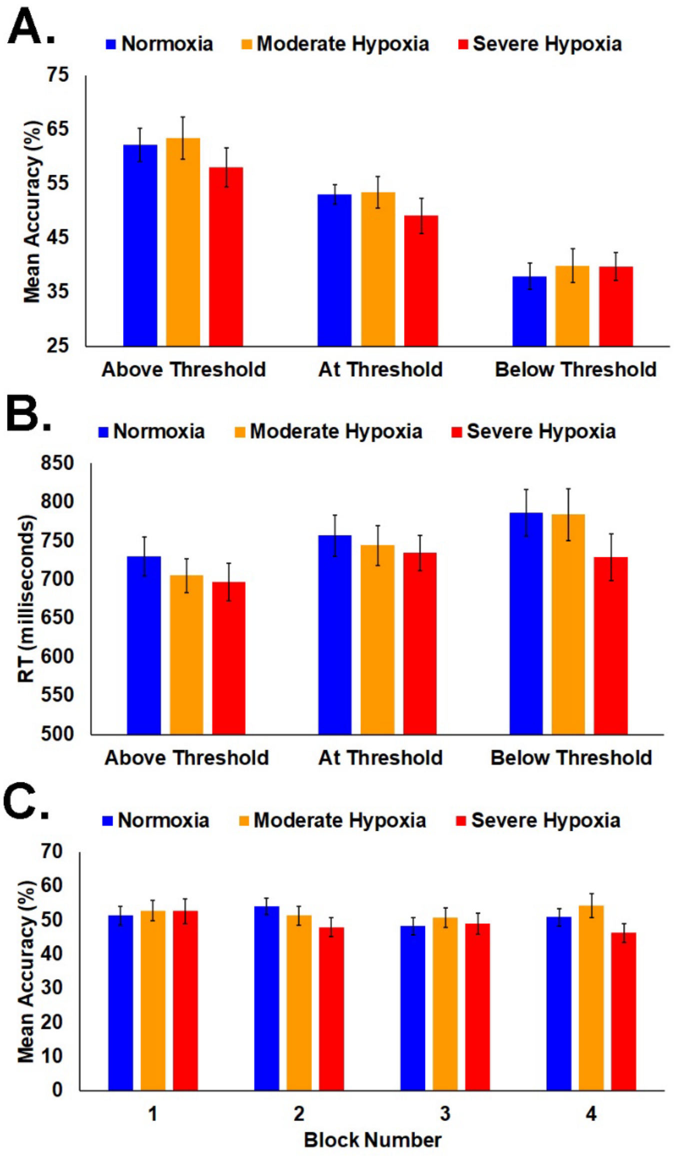

Figure 2. A) CCT accuracy results by threshold presentation and condition. B) CCT RT results for correct trials by threshold presentation and condition. C) CCT accuracy results by block and condition averaged across threshold presentations. Error bars represent standard error of the mean (SEM).

For accuracy, the main effect of threshold was significant, *F*(1.693, 34.624) = 38.534, *p* <.001, η_p^2 = .637, with decreased accuracy for below threshold compared to at or above threshold and decreased accuracy for at threshold compared to above threshold, all *p*s $< .01$. The main effect of condition did not reach significance, $F(2, 21) = 1.235$, $p = .310$. Finally, the condition × threshold interaction was also not significant, $F(4, 19) = 1.587$, $p = .185$. For RT, the main effect of threshold was significant, $F(2, 21) = 11.445$, $p < .001$, $\eta_p^2 = .342$, with faster RTs for above threshold compared to at or below threshold, all *p*s $< .01$. The main effect of condition was also significant, $F(2, 21) = 4.834$, $p = .013$, $\eta_p^2 = .180$, with faster RTs during severe hypoxia compared to normoxia, $p < .01$. Finally, the condition × threshold interaction was not significant, $F(3.056, 67.228) = 2.223$, $p = .092$. Figure 2A-B illustrates these accuracy and RT results.

For accuracy only, we also tested a 3 (condition: normoxia, moderate hypoxia, severe hypoxia) × 4 (block) repeated-measures ANOVA to examine changes in performance over blocks by condition. To do this, we averaged across the three different threshold types. Neither the main effect of condition, $F(2, 21) = 1.235$, $p = .301$, nor the main effect of block, $F(3, 20) = 1.138$, $p = .340$, was significant. However, the condition × block interaction approached significance, $F(6, 17) = 1.996$, $p = .071$. Results can be seen in Figure 2C.

Visual Evoked Potential Results

One participant was excluded from the below analyses due to having N75 and N135 peak amplitudes that were >2SD from the group mean. A one-way ANOVA with condition as a factor (normoxia, moderate hypoxia, severe hypoxia) was tested on P100 mean amplitude, as well as N75-P100 and P100-N135 peak-to-peak amplitudes. For P100 amplitude, the main effect of condition did not reach significance, $F(2, 28) = 1.107$, $p = .338$. For N75-P100 amplitude, the main effect of condition was also not significant, $F(2, 28) = 1.423$, $p = .249$. Finally, for P100-N135 amplitude the main effect of condition was not significant, $F(1.625, 47.139) = 0.234$, $p = .746$. Figure 3 shows grand average waveforms for all 3 conditions.

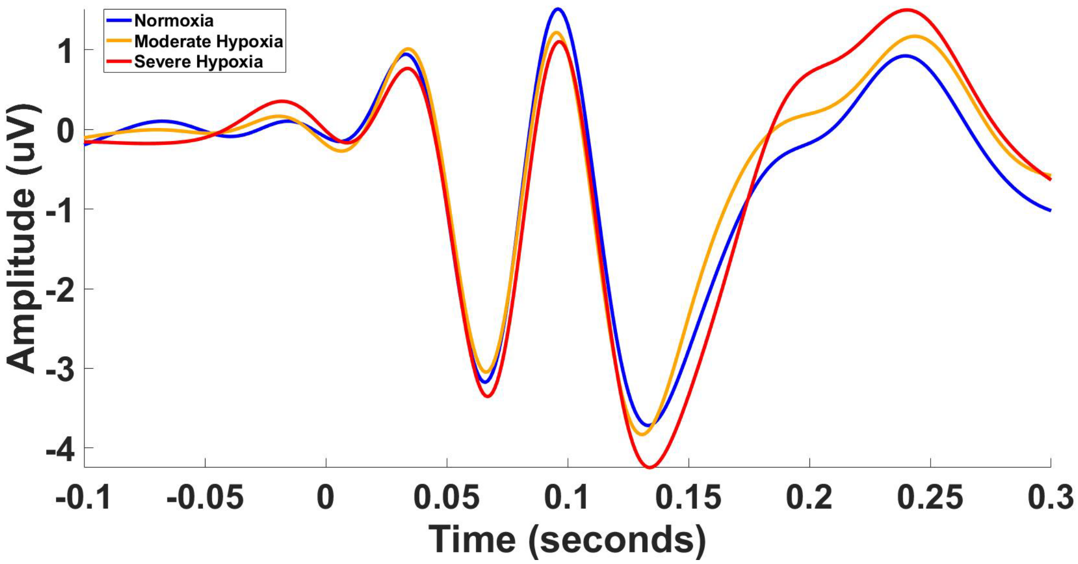

Figure 3. Grand averaged waveforms for each condition showing no significant main effect of condition for any of the VEP measures.

We also examined potential changes in VEP amplitude by block and condition. To do so, 4 (block) × 3 (condition: normoxia, moderate hypoxia, severe hypoxia) repeated-measures ANOVAs for each amplitude measure of interest (i.e., P100, N75-P100, and P100-N135) were tested. First, to ensure we had adequate reliability separating our data into blocks, we assessed Cronbach's alpha for all 12 (i.e., four blocks, three conditions) amplitude measures for the P100, with a resulting $\alpha = .977$ indicating excellent reliability.

For P100 amplitude, the main effect of condition was not significant, $F(2, 28) = 2.169$, $p = .123$. The main effect of block was significant, $F(2.131, 61.807) = 16.578$, $p < .001$, $\eta_p^2 = .364$, with increasing amplitude at later blocks (Figure 4). The condition × block interaction also reached significance, $F(6, 24) = 2.577$, $p = .020$, $\eta_p^2 = .082$. Planned follow-up one-way ANOVAs were performed with condition as a factor for each block separately. Blocks one, three, and four all had non-significant main effects of condition, all Fs ≤ 1.870, all ps $\geq .163$. Block two did result in a significant main effect of condition, $F(2, 28) = 4.002$, $p = .024$, $\eta_p^2 = .121$, whereby normoxia had a higher amplitude than both hypoxia conditions, ps $< .05$.

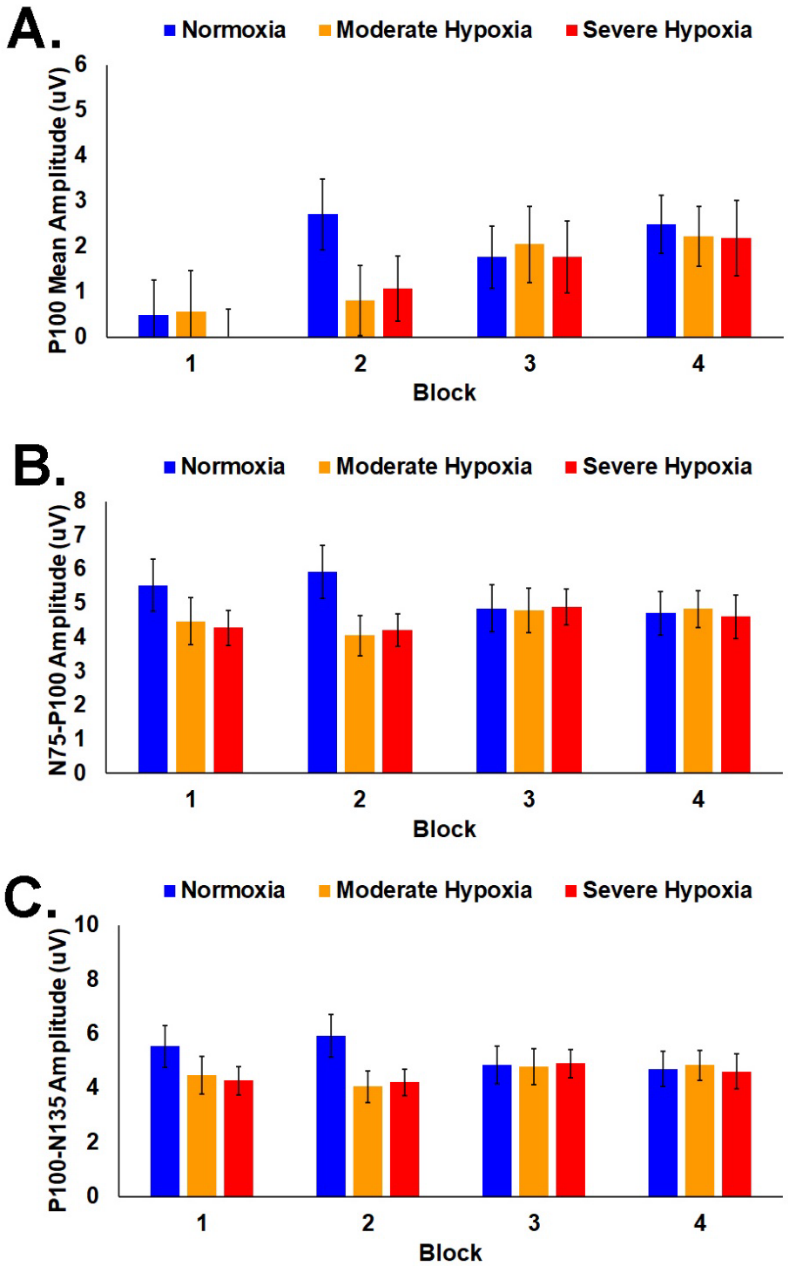

Figure 4. VEP amplitude measures by condition and block. Error bars represent SEM.

For the N75-P100 amplitude, the main effect of condition was significant, *F*(2, 28) = 3.661, *p* = .032, η_p^2 = .112, whereby pairwise comparisons showed higher amplitude for normoxia compared to both hypoxia conditions, *p*s < .05. The main effect of block was not significant, *F*(2.028, 58.821) = 0.079, *p* = .971. However, the block × condition interaction was significant, $F(4.179, 121.203) = 3.190$, $p = .014$, $\eta_p^2 = .099$. Planned follow-up one-way ANOVAs were performed with condition as a factor for each block separately. Block one showed a significant main effect of condition, $F(2, 28) = 3.515$, $p = .036$, $\eta_p^2 = .108$, whereby pairwise comparisons showed higher amplitude for normoxia compared to both hypoxia conditions, ps < .05. Block two showed a marginally significant main effect of condition, $F(2, 28) = 3.119$, $p = .052$, $\eta_p^2 = .097$. For blocks three and four, the main effect of condition was not significant, Fs ≤ 0.102, ps ≥ .904.

For the P100-N135 amplitude, the main effect of condition was not significant, $F(2, 28) = 0.836$, $p = .439$, but the main effect of block was significant, $F(1.847, 53.550) = 21.684$, $p < .001$, $\eta_p^2 = .428$. Pairwise comparisons demonstrated block one was lower amplitude than all other blocks and that block two was lower amplitude than block four, all ps < .05. Finally, the condition × block interaction was also significant, $F(4.196, 121.671) = 4.718$, $p = .001$, $\eta_p^2 = .140$. Planned follow-up one-way ANOVAs were performed with condition as a factor for each block separately. For block one, $F(2, 28) = 4.839$, $p = .070$, and block two, $F(2, 28) = 3.136$, $p = .051$, the main effect of condition approached significance. For blocks three and four, the main effect was not significant, all Fs ≤ 1.352, all ps ≥ .266.

Physiological Measures

Physiological data from three exposures were missing due to technical issues with the acquisition device. Therefore, the below analyses include n=28. We averaged SpO_2 and HR variables into 1 min bins. For both SpO_2 and HR separately, 3 (condition: normoxia, moderate hypoxia, severe hypoxia) × 14 (time bin) repeated-measured ANOVAs were tested to look for changes over time by condition. For SpO_2, both the main effect of condition, $F(2, 26) = 470.169$, $p < .001$, $\eta_p^2 = .946$, and the main effect of time, $F(2.136, 57.664) = 110.240$, $p < .001$, $\eta_p^2 = .803$, were significant with lower SpO_2 at later time bins and with decreased oxygen concentrations. Additionally, the condition × time interaction was significant, $F(4.410, 119.077) = 45.755$, $p < .001$, $\eta_p^2 = .629$. As seen in Figure 5A, SpO_2 decreased more quickly during severe hypoxia compared to moderate hypoxia, as expected. Similarly for HR, both the main effect of condition, *F*(2, 26) = 61.685, $p < .001$, $\eta_p^2 = .696$, and the main effect of time, *F*(2.590, 69.918) = 9.235, $p < .001$, $\eta_p^2 = .255$, were significant with higher HR at later time bins and with decreased oxygen concentrations. Additionally, the condition × time interaction was significant, *F*(4.556, 123.015) = 8.841, $p < .001$, $\eta_p^2 = .247$. As seen in Figure 5B, HR increased more quickly during severe hypoxia compared to moderate hypoxia, as expected.

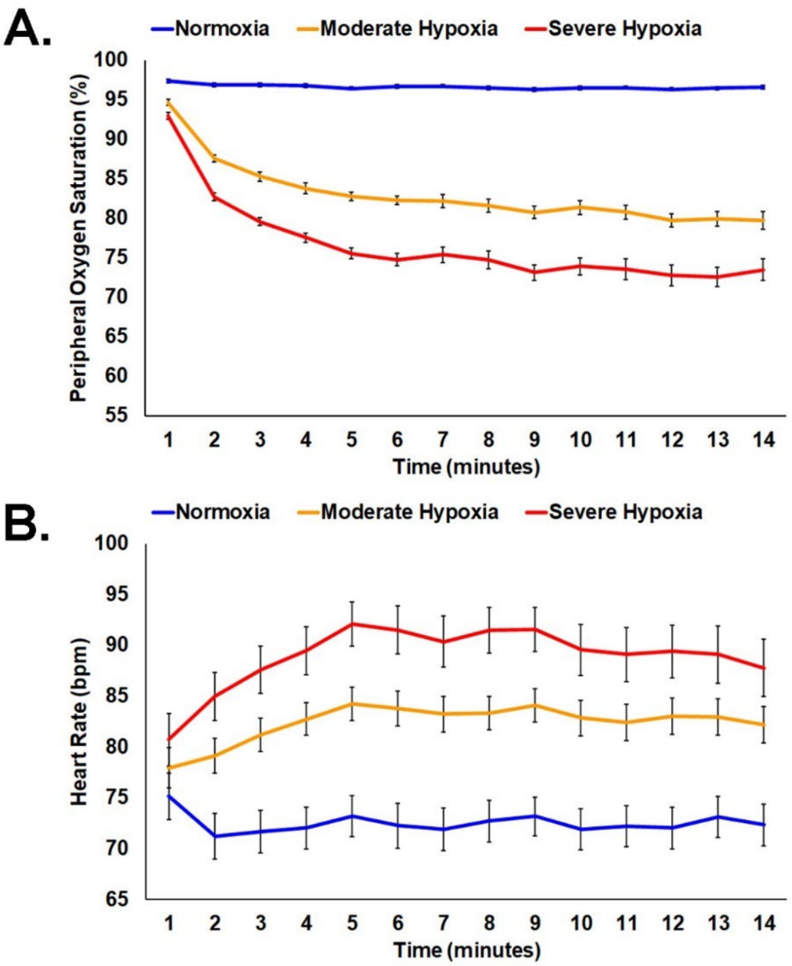

Figure 5. A) SpO2 and B) heart rate by condition across the 14 min exposure. Error bars represent SEM.

Hypoxia Symptoms

For each condition, a total HSQ score was calculated as the sum of all symptom ratings. A repeated-measures ANOVA with condition as a factor (normoxia, moderate hypoxia, severe hypoxia) revealed a significant main effect of condition, $F(2, 29) = 40.106$, $p < .001$, $\eta_p^2 = .572$. Planned post-hoc comparisons demonstrated that participants reported higher HSQ scores for severe hypoxia compared to moderate hypoxia and normoxia, and had higher scores for moderate hypoxia compared to normoxia, all *p*s < .005. Figure 6A illustrates these results.

To focus on symptoms that were only attributable to hypoxia and not to extraneous variables such as the stimuli presented or time on task, we subtracted symptom ratings from the normoxia visit for the two hypoxia visits separately (Blacker & McHail, 2022). In other words, if a participant reported mild fatigue (score of 1) during the normoxia visit and mild fatigue (score of 1) during the moderate hypoxia visit, the resulting score was a zero (i.e., symptom not observed) for moderate hypoxia because that fatigue cannot be attributed to the effects of low oxygen exposure. Alternatively, if a participant reported mild blurred vision (score of 1) for normoxia and moderate blurred vision (score of 2) for moderate hypoxia, then their hypoxia symptom rating for that condition would be a 1, because they experienced greater blurred vision with the addition of low oxygen exposure. To then explore how consistent participants were in their symptoms across moderate and severe hypoxia conditions, we calculated the number of symptoms that each participant reported in both hypoxia visits (i.e., consistent symptoms) and we calculated the number of symptoms that each participant reported in only one of the hypoxia visits (i.e., inconsistent symptoms). Frequency of consistent and inconsistent symptoms can be seen in Figure 6B. If we define the presence of a “hypoxia signature” as individuals who had a higher number of consistent symptoms compared to inconsistent symptoms, then in our sample of 31 a total of only 4 individuals showed a “signature”. This data suggests that the presence of a hypoxia signature may not be as common as traditionally thought, especially across differing altitude exposures.

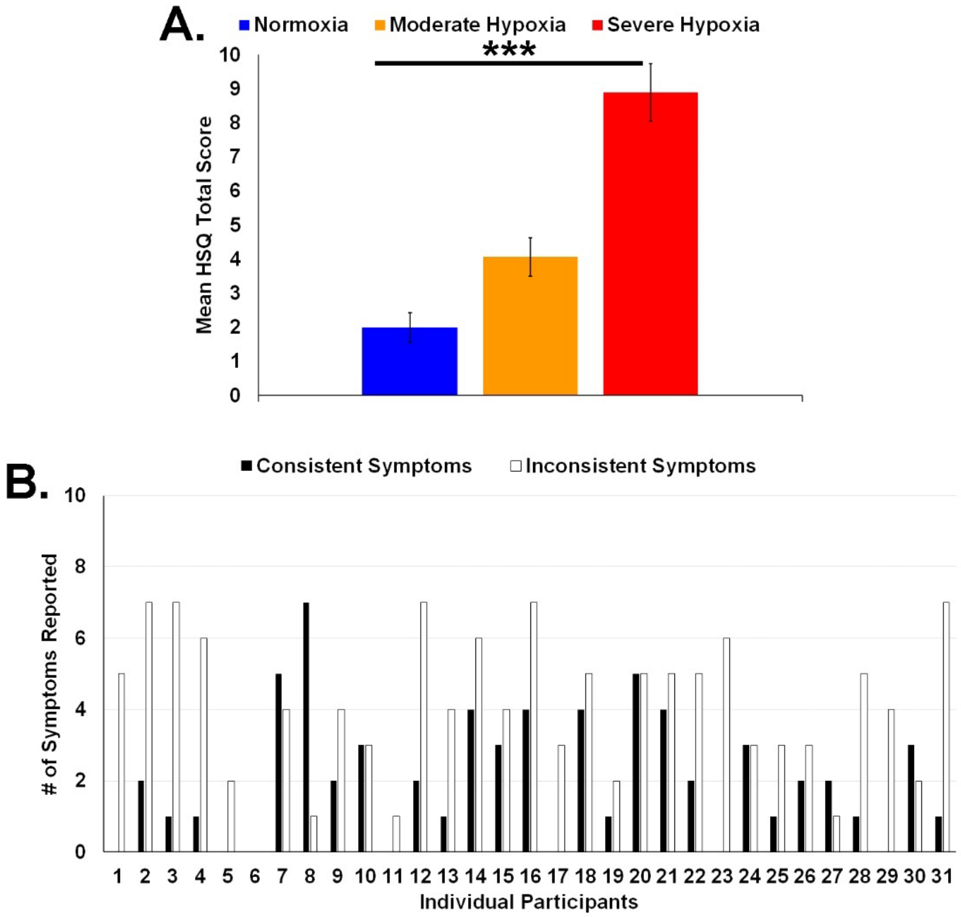

Figure 6. A) Mean total HSQ scores by condition. ***$p < .001$. Error bars represent SEM. B) Frequency of consistent versus inconsistent symptoms reported across the two hypoxia conditions for each individual participant. Only four participants reported more consistent than inconsistent symptoms (i.e., black bar higher than white bar).

Discussion

In the current study, we aimed to examine the extent to which the visual system is impaired during acute hypoxia, as well as investigate a potential neural mechanism of this impairment. Using a modified CCT paradigm that targeted only S-cones, we did not find any effect of hypoxia on accuracy or RT. Additionally, we evaluated VEPs using a pattern reversal paradigm and found no significant effect of hypoxia on VEP amplitude. Both our physiological and symptom data demonstrated that our experimental manipulations worked well, as a typical hypoxia ventilatory response was present in the physiological measures and participants reported higher HSQ scores with increasing altitude. Finally, when comparing symptom reporting between the moderate and severe hypoxia conditions, very few individuals reported consistent symptoms suggesting that high intra-individual differences exist.

Our null results here for the S-cone CCT task are inconsistent with prior work showing that there are color vision deficits during hypoxia, with emphasis on the effects on the S-cone pathway (for a review see, Barbur & Connolly, 2011). However, closer inspection shows some critical differences between our study and those prior. Multiple previous studies have shown that yellow-blue (YB) chromatic sensitivity is impaired under hypoxic conditions, but that light level also plays an important factor in this effect. For example, using the Color Assessment and Diagnosis (CAD) test, Connolly et al. (2008) found that YB sensitivity was significantly worse when light levels were lower. Additional work using other assessment measures found similar interactions between hypoxia and light level with respect to YB thresholds (Richalet et al., 1989; Kobrick, 1970). In our current study, we were unable to administer the test in a dark environment due to the nature of where the ROBE is located. Therefore, this null result may be due our photopic environment. Future work should investigate this distinction.

We also did not find any significant changes in VEP amplitude under hypoxic compared to normoxic conditions. At least one prior study has shown mixed evidence of whether a pattern reversal induced VEP is sensitive to hypoxia (Blacker & McHail, 2022). The current study serves to replicate that prior work and demonstrably suggests that there is no effect. The alternating checkerboard stimulus used to elicit the VEPs here activates the luminance pathway in the brain. The luminance pathway is a very robust set of connections due to its evolutionary importance. It seems likely that this pathway is too robust to be affected by an acute hypoxia exposure of the magnitude used here. A more appropriate and sensitive stimulus to specifically target the S-cone pathway would be an isoluminant chromatic sinusoidal grating specific to S-cones (e.g., Schneck et al., 1997). This type of stimulus has been used to demonstrate differences in VEP latency based on varying blood glucose levels (Schneck et al., 1997).

In conclusion, while hypoxia effects on the visual system are well-documented in the literature, we failed to find any in the current study. Based on a close comparison of our experimental stimuli and conditions and previous work, these null results are attributed to sub-optimal testing conditions (i.e., lighting) and stimuli used for VEPs. Given the importance of optimal visual performance during tactical aviation and the ever-present risk of hypoxia, future work should work to identify hazards that may cause mishaps and develop strategies to mitigate performance impairment if hypoxia should occur in flight.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Copyright Statement

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