

## EFFECTS ON TASK PERFORMANCE AND PSYCHOPHYSIOLOGICAL MEASURES OF PERFORMANCE DURING NORMOBARIC HYPOXIA EXPOSURE

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Cognitive impairment during flight is a factor in which human-autonomous systems integration presents opportunities for improved safety. A research team at NASA Langley conducted an experiment to study the impact of mild normobaric hypoxia induction on aircraft pilot performance and psychophysiological state. A within-subjects design involved non-hypoxic and hypoxic exposures while performing three 10-minute tasks. Results indicated the effect of 15,000 feet simulated altitude did not induce significant performance decrement but did produce increase in perceived workload. Analyses of psychophysiological responses evince the potential of biomarkers for hypoxia onset. This study represents on-going work at NASA intending to add to the current knowledge of psychophysiological-based input to automation to increase aviation safety.

The Next Generation Air Transportation System (NextGen; FAA, 2011) is designed to incorporate airspace information, e.g., traffic, weather, and operator state to improve flight safety. Within the NASA Aeronautics Research Mission Directorate (ARMD), the Safe Autonomous Systems Operations (SASO) project is supporting and directing research into increasingly autonomous systems-supported operations. This future flight deck system is being developed to be aware of the vehicle, operator, and airspace system state and respond appropriately (Stephens et al., 2011). These future systems will sense internal and external hazards, evaluate them, and facilitate timely and suitable responses for mitigation.

A controlled method for inducing poor operator functional state (OFS) will further development of behavioral, psychophysiological, and performance indices to augment automation capabilities and potential enable the creation of technologies necessary for reduced crew operations. Hypoxic hypoxia is a reduction of oxygen in the arterial blood with a resulting decrease in oxygen for diffusion into the tissues (Gradwell, 2006). Hypobaric hypoxia, a mechanism for hypoxic hypoxia, is caused by a reduction of oxygen partial pressure in inspired

air at altitude, and has been an aeromedical and human performance concern since the dawn of aviation (Dille, 2002). Brief hypoxia in humans results in temporary cognitive impairment including lapses of attention or loss of situation awareness related to human error. The intentional use of hypoxia in human test subjects for the concomitant cognitive impairment has potential for understanding limitations of human operators and performance augmentation from autonomous systems.

A research team at NASA LaRC applied normobaric hypoxia induction in human subjects to study the impact on aircraft pilot performance. Voluntary subjects in the study experienced simulated altitudes of Sea Level (21% O<sub>2</sub>) and 15,000 feet (11.2% O<sub>2</sub>) induced by an Environics, Inc. Reduced Oxygen Breathing Device (ROBD-2). During non-hypoxic and hypoxic exposures each test subject performed a battery of written, computer-based, and flight simulation tasks. Task performance measures, NASA Task Load Index subjective self-report of workload, and physiological responses including: Pulse oxygen saturation (SPO<sub>2</sub>), electrocardiogram (ECG), respiratory effort, and electroencephalogram (EEG) were recorded. The performance, subjective, and physiological data were examined to understand cognitive impairment due to mild hypoxia exposure. The purpose of this study is to add to the current knowledge of psychophysiological-based input to automation to increase aviation safety.

### **Technical Goals**

- Stage and deploy hypoxia induction equipment, psychological testing batteries, and physiological recording equipment.
- Assess efficacy of cognitive impairment induced by mild hypoxia exposure.
- Determine safe and effective method for performing hypoxia induction in future studies to support OFS assessment.

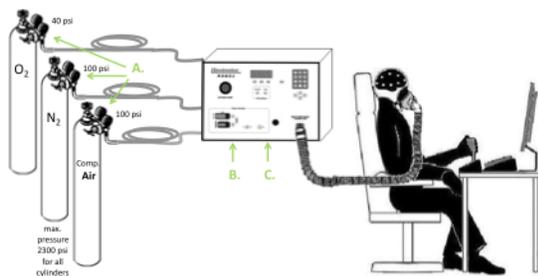
### **Method**

#### **Experiment Subjects**

The experiment sample (N = 57) included 8 women and 49 men. All subjects were screened for disorders and excluded from participating if they reported neurological, cardiovascular, prescription medication affecting central nervous system and autonomic nervous system activity, and smoking. Furthermore, a medical examination conducted at the NASA LaRC Clinic consisting of 12-lead electrocardiogram, complete blood count with differential, and pulmonary function test with all results being within normal limits was required in order to participate.

#### **Experiment Apparatus**

An Environics, Inc. ROBD-2 (see Figure 1) portable computerized gas-blending instrument used to induce hypoxia, without changes in atmospheric pressure, through an aviator's oxygen mask worn by the human research subject. The ROBD-2 is capable of producing Sea Level (21% O<sub>2</sub>) to 34,000 feet (4% O<sub>2</sub>).



**Safety Mechanisms for Protection against Over Pressurization:**  
 A. Regulators have built-in pressure relief valve between first and second stage to prevent catastrophic failure.  
 B. ROBD2 has built-in overpressure detect mechanism to limits pressure in mask to 0.75 PSIG.  
 C. ROBD2 has built-in mechanical pressure relief valve prevents pressure in mask from exceeding 1 PSIG.



Figure 1. Schematic depiction of ROBD-2 configuration      Figure 2. Test subject in flight sim

## Physiological Recording

EEG was recorded from 16 electrode sites ( $AF_1$ ,  $AF_2$ ,  $F_1$ ,  $F_2$ ,  $F_Z$ ,  $C_1$ ,  $C_2$ ,  $C_Z$ ,  $P_1$ ,  $P_2$ ,  $PO_Z$ ,  $T_7$ ,  $T_8$ ,  $O_1$ ,  $O_2$ ,  $O_Z$ ; International 10-20 system; Jasper, 1958) through a gTEC gUSBamp amplifier (see Figure 2).  $SPO_2$ , ECG, respiration effort, and galvanic skin response (GSR) were also recorded through a gTEC gUSBamp amplifier. All signals were digitized at 256 Hz.  $SPO_2$  and pulse rate were redundantly monitored and recorded from the ROBD-2.

## Experiment Tasks

Each test subject performed a battery of written, computer-based, and flight simulation tasks. A Cognitive Function Test (Westermann, 2004) battery of written tasks was completed by half of the subjects including: simple computational problems – addition, subtraction, or multiplication; serial 7 subtraction; eye-hand coordination drawing; semantic memory and visual-motor coordination; working memory digit and address recall; and trail-making A and B. The other half of the subjects completed the CogScreen Hypoxia Edition (Kay, 1995). The Multi-Attribute Task Battery-II was performed involving tasks analogous to activities performed by aircraft crew members in flight (see Figure 3). Also, a flight simulation task was performed using X-Plane 10 simulation (KC-10 aircraft model) connected to force feedback sidestick control interceptors.

## Experimental Procedure

The experimental session lasted approximately 4 hours. Subjects completed informed consent documentation. Subjects were briefed on the operation of the ROBD-2 and connected to physiological recording equipment. Subjects completed training sessions for each experiment task. Subjects sat quietly breathing room air while wearing mask to establish physiological baseline. Subjects performed each task three times under the following conditions: 1) Breathing room air while wearing mask 2) Breathing sea level gas mixture through mask 3) Breathing 15,000 feet gas mixture through mask. Subjects recovered from hypoxia exposure by breathing 100%  $O_2$  for 2 minutes following 15,000 feet exposure. Subjects completed self-reported workload measure (NASA-Task Load Index, NASA-TLX) after each trial. After completing all trials, subjects were debriefed regarding the study purpose.

## Dependent Measures

The experimental design was within-subjects, so all subjects experienced hypoxia while performing all of the experiment tasks. Self-reported hypoxia symptoms are recorded and will be examined to better understand the individual variability of hypoxia exposure. Self-reported workload (NASA-TLX) was examined to assess effect of hypoxia on subjective experience of each task. Performance on each of the tasks was assessed for errors of commission and omission. Raw physiological data were reduced to variables indicative of OFS to determine the effect of hypoxia on indices of OFS.

The alternative hypothesis is: human subjects experience cognitive impairments to a greater extent ( $p < 0.05$ ) during ROBD-2 equivalent altitude = 15,000 feet (11.2% Oxygen) than during ROBD-2 equivalent altitude = sea level (21% Oxygen). ANOVA were used to test for main and interaction effects of subjective, behavioral, psychophysiological, and performance indices during sea level condition compared to hypoxia condition. Additional analyses of physiological and cortical responses were conducted.

## Results

Statistical analyses of subjective workload ratings revealed significant difference between the sea level and 15,000' normobaric hypoxia conditions only during the flight simulation task:

- NASA-TLX Overall Score  $t(52) = 1.8136, p = .0036$
- NASA-TLX Mental Effort  $t(52) = 1.1726, p = .0488$
- NASA-TLX Performance  $t(52) = 2.668, p = .0412$
- NASA-TLX Frustration  $t(52) = 2.189, p = .0154$

ANOVA statistical analyses of the EEG and task performance revealed no significant difference between the sea level and 15,000' normobaric hypoxia conditions. More advanced analysis techniques were employed to examine complex coupling of multiple body systems. Specifically, ECG multivariate respiration entropy was calculated by temporally syncing the respiration tidal volume signal with the ECG signal. The ECG signal was then used as the reference where a QRS detector was applied to determine the indice locations of the R wave (Pan & Tompkins, 1985). Utilizing the indices of the array from the ECG signal, we extract the tidal volume of the respiration at that specific time instance of when the R-Wave occurred. We then obtained the R-R interval from the ECG signal and the 'downsampled' tidal volume of the respiration signal to apply the multivariate entropy calculation to the two sequences (Von Tscharner, 2000). The specifications of the multivariate entropy are as follows: a time delay of  $\tau = (1,1)$ , embedded dimension vector  $M = (2,2)$ , and tolerance value (threshold) of  $r = 0.2$  of the standard deviation.

From these analysis, the ECG-Respiration Multivariate Coupling indicated the following:

- Overall: Mean = 1.37, Std Dev = 0.30 (N=363)
- Non-Hypoxic: Mean = 1.46, Std Dev = 0.29 (N=178)
- Hypoxic: Mean = 1.29, Std Dev = 0.28 (N=185)

The EEG data was analyzed using inspiration around the core idea proposed by Vinzenz von Tscherner (2000) and is similar to bandpass filtering and the concept of equalizers. The connection with wavelet theory is that the filter is constructed by rescaling a single basis function,  $\hat{\phi} = e^{-\alpha(f-f_c)^2 + \alpha(f-f_c)^4}$ , using a special array of scales in the frequency domain with no imaginary components. (need to define the variables, alpha, f, fc, psi) These wavelets were then projected in the time domain using the Fast Fourier Transform. This allows us to obtain a complex wavelet design (real and imaginary components) in the time domain. Thus, when convolved with the EEG signal we produce a filtered signal intensity as a function of time. The filter bank design and optimization for this methodology is discussed in Napoli et al (2017). The signal is then smoothed using a gaussian filter, providing 12 wavelet filters each with their own specified frequency bands tailored for EEG analysis. These frequency bands represent the typically delta, beta, theta, alpha and gamma bands. Each subject's EEG band intensities are z-scored prior to conducting statistical tests.

The analysis demonstrated a significant decrease in the normalized power of wavelet  $W_6$  (mid-level) beta band: 15.19-18.37 Hz) across all but three sites ( $O_1$ ,  $F_1$ , and  $C_2$ ) during hypoxic trials.

## Discussion

Analyses involving coupling across physiological systems and wavelet transforms of cortical activity revealed patterns that can discern between the simulated altitude conditions. Specifically, multivariate entropy of ECG/Respiration components were found to be significant predictors ( $p < 0.02$ ) of hypoxia. Furthermore, in EEG, there was a significant decrease in mid-level beta (15.19-18.37Hz) during the hypoxic condition in thirteen of sixteen sites across the scalp. Task performance was not appreciably impacted by the effect of 15,000 feet simulated altitude. Analyses of psychophysiological responses evince the potential of biomarkers for mild hypoxia onset.

The potential for identifying shifts in underlying cortical and physiological systems could serve as a means to identify the onset of deteriorated cognitive state. Enabling such assessment in future flight decks could permit increasingly autonomous systems-supported operations. Augmenting human operator through assessment of cognitive impairment has the potential to further improve operator performance and mitigate human error in safety critical contexts. This study represents ongoing work at NASA intending to add to the current knowledge of psychophysiological-based input to automation to increase aviation safety.

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