

Lingering Altitude Effects During Piloting and Navigation in a Synthetic Cockpit

Jeremy Beer; Bria Morse; Todd Dart; Samantha Adler; Paul Sherman

- INTRODUCTION:** A study was performed to evaluate a cockpit flight simulation suite for measuring moderate altitude effects in a limited subject group. Objectives were to determine whether the apparatus can detect subtle deterioration, record physiological processes throughout hypobaric exposure, and assess recovery.
- METHODS:** Eight subjects trained to perform precision instrument control (PICT) flight and unusual attitude recovery (UAR) and completed chamber flights dedicated to the PICT and UAR, respectively. Each flight comprised five epochs, including ground level pressure (GLP), ascent through altitude plateaus at 10,000, 14,000, and 17,500 ft (3050, 4270, and 5338 m), then postexposure recovery. PICT performance was assessed using control error (FSE) and time-out-of-bounds (TOOB) when pilots exited the flight corridor. UARs were assessed using response times needed to initiate correction and to achieve wings-level attitude. Physiological indices included S_{pO_2} , heart rate (HR), end tidal O_2 and CO_2 pressures, and respiration metrics.
- RESULTS:** Seven subjects completed both flights. PICT performance deteriorated at altitude: FSE increased 33% at 17,513 ft and 21% in Recovery vs. GLP. Mean TOOB increased from 11 s at GLP to 60 s in Recovery. UAR effects were less clear, with some evidence of accelerated responses during and after ascent.
- CONCLUSIONS:** The test paradigm was shown to be effective; piloting impairment was detected during and after exposure. Physiological channels recorded a combination of hypoxia, elevated ventilation, and hypocapnia during ascent, followed by respiratory slowing in recovery. Findings indicate precision piloting and respiration are subject to changes during moderate altitude exposure and may remain altered after S_{pO_2} recovers, and changes may be linked to hypocapnia.
- KEYWORDS:** hypoxia, physiological episodes, precision piloting, respiration rate, hyperventilation.

Beer J, Morse B, Dart T, Adler S, Sherman P. *Lingering altitude effects during piloting and navigation in a synthetic cockpit*. *Aerosp Med Hum Perform*. 2023; 94(3):135–141.

Moderate hypoxia presents a particular challenge in aviation safety since its effects are not necessarily conspicuous or related to self-observed symptoms:¹² if subtle hypoxia—perhaps below the threshold where the pilot recognizes it—increases control error only slightly, impairment could be dangerous precisely because of this subtlety, as in Type I spatial disorientation. This challenge is amplified by the variety of cognitive components in aviation. Whereas hypobaric and normobaric (ground-level) hypoxia reliably induce physiological effects, including increased heart rate (HR), decreased oxygen saturation (S_{pO_2}), and changes to cerebral oxygen delivery,^{1,15,20} the range of reported cognitive manifestations has been varied. Declines in executive and auditory processing, speeded arithmetic, and vigilance have been reported,^{3,4} whereas certain other components, including simulated

aviation control metrics, can remain relatively unscathed during mild or moderate exposure (up to 13,999 ft/4267 m).⁵ In the realm of normobaric exposure, breathing mixtures at more severe hypoxia levels ranging from 18,000–25,000 ft (5486–7620 m) equivalent altitude increased control error in simulated flight,^{13,16} and in a multiple-exposure paradigm,

From KBR, Brooks Aerospace Environment Protection Laboratory, San Antonio, TX, USA; and USAFSAM/FE and the Department of Radiology, 59th Medical Wing, JBSA Lackland, San Antonio, TX, USA.

This manuscript was received for review in July 2022. It was accepted for publication in December 2022.

Address correspondence to: Jeremy Beer, 2485 Gillingham Dr., Bldg. 170, Brooks, TX 78235, USA; Jeremy.Beer@us.kbr.com.

Reprint and copyright © by the Aerospace Medical Association, Alexandria, VA.

DOI: <https://doi.org/10.3357/AMHP.6149.2023>

initial exposure to 25,000 ft (7620 m) equivalent hypoxia reportedly caused piloting impairment during subsequent mild exposure.¹⁴

Additional concern arises from reports that pilots are vulnerable to a “hypoxia hangover” comprising lingering impairment during posthypoxia recovery.¹⁸ In this hypothesis, returning the pilot to pre-exposure oxygen levels—either O₂ breathing concentrations or peripheral S_pO₂—might not restore proficiency immediately. To date, studies have employed diverse tasks (simulated sorties,¹⁴ synthetic workstation,³ psychomotor vigilance,⁴ and simple and choice reaction tasks⁶) to examine the persistence of impairment following hypoxia, with some identifying continuing impairment and others reporting none or only equivocal hangover effects.^{6,17} This divergence indicates a need for further investigation to characterize the prevalence, duration, and nature of postexposure impairment.

Because of the potential danger of creeping control raggedness, the need to detect subtle impairment using flight-relevant tasks, and the additional hazard if posthypoxia effects emerge, we saw a need to develop an affordable platform to assess piloting during and after exposure to moderate altitude conditions. This exploratory study was performed to verify the effectiveness of a synthetic environment and test concept incorporating a model cockpit, a visual simulator, physiological monitoring, and hypobaric exposure. The test paradigm was designed to employ control and navigation tasks relevant to aviation during progressive ascent through moderate hypobaric plateaus. Since aviation takes place in a pressure-varying environment and the physiological and phenomenal effects of normobaric vs. hypobaric exposure are not necessarily identical,^{1,19} the cockpit was situated in a hypobaric chamber. A physiological monitoring suite including oximetry, respiration, and gas analysis was employed, and testing included a recovery period against which baseline performance was compared.

The study's primary objective was to determine whether this apparatus could identify moderate altitude effects in a small group of volunteers who were not career aviators but would train to proficiency on tasks with manifest face validity. These tasks were employed to test whether exposure would render the pilots susceptible to ragged control or impaired directional corrections as physiological metrics were recorded. The first, precision instrument control (PICT) task, resembled tasks in earlier studies^{13,14,16} wherein subjects maintained altitude, airspeed, and heading while countering simulated disturbances. A second task presented unusual attitude recoveries (UAR): pilots must level the aircraft after being placed in a scenario depicting unpredictable bank and pitch states. Subjects completed two identical hypobaric ascents (hereinafter called “flights”): one for PICT and one for UAR. Both flights progressed through five 10-min epochs: baseline ground-level pressure (GLP) equivalent to 659 ft (201 m) above sea level, then equivalent pressure altitudes of 10,000 ft (3050 m), 14,000 ft (4270 m), and 17,500 ft (5338 m), and finally a Recovery epoch following return to GLP.

Two hypotheses were tested to validate the sensitivity of the apparatus and paradigm for assessing piloting at altitude,

and to determine effect sizes to inform future studies examining moderate exposure. The first null hypothesis was that altitude exposure would not affect performance, measured using PICT control error and UAR response times and with emphasis on the comparison between baseline GLP and 17,500 ft (5338 m). The second null hypothesis was that no difference would emerge between performance recorded during Recovery vs. GLP.

METHODS

Subjects

The study protocol was approved by the AFRL 711th HPW Institutional Review Board. A total of eight nonsmoking, U.S. Air Force active-duty volunteers ages 33–40 (seven men, one woman) enrolled with written informed consent. Subjects were screened to a 20/20 distance vision criterion, with two participating wearing vision correction. All were encouraged to forego participation if they were not well rested or had consumed alcohol or excessive caffeine in the day before testing. Subjects completed three training sessions. After introducing the subject to flight controls and PICT in session 1, sessions 2 and 3 presented instruction on both PICT and UAR. During UAR training, subjects were encouraged to correct bank before pitch and apply throttle to facilitate recovery. Subjects then completed two flights at least 44 h apart: one each for PICT and UAR. Flight presentation order was counterbalanced across subjects to the extent possible; three subjects completed the PICT flight first and four completed UAR first. One subject, scheduled to complete PICT first, experienced physiological difficulties, including discomfort from a poor mask fit in both flights, and was removed.

Flights

In the PICT flight, subjects performed the task for 10 min at GLP and then the chamber executed controlled decompressions, ascending to three successive 10-min altitude plateaus at 10,000, 14,000, and 17,500 ft (3050, 4270, and 5338 m). The chamber then returned to GLP and a 10-min Recovery epoch ensued. Chamber pressure ascents and descents were executed at 5000 ft (1524 m) · min⁻¹ unless the subject experienced ear or sinus pain upon descent, in which case descent rate was reduced. Subjects breathed air (21% O₂) throughout all five epochs.

The same five-epoch profile was executed in the other flight; in this case, each epoch contained one block of eight UAR trials. After completing each block, the subject was instructed to return to the straight-and-level indicated state of 15,000-ft (4572-m) altitude, 350-kn airspeed, and 090° heading for the remainder of the epoch.

Equipment and Tasks

A cockpit mockup (Sage Cheshire, Inc., Lancaster, CA, USA) was situated in Hypobaric Chamber E of the Brooks Aerospace Environment Protection Laboratory. The cockpit, which duplicates the interior dimensions of a U-2, incorporates a Hyundai

P224W LED monitor (1680 × 1050 pixels; 52 × 41°) for the pilot-ing display and Thrustmaster® Warthog flight controls.

During flights, subjects wore an HGU-55/P helmet and breathed through an MBU-20/P mask and CRU-60 connector. A CRU-73 regulator was used to deliver filtered air at demand (not safety) pressure continuously. A Fleisch pneumotachograph, standardized daily against a calibrated flowmeter, was interposed between regulator and mask. Mask gases and pressure were sampled using tap lines connecting the mask to an Extrel MAX300-LG mass spectrophotometer (Process Insights, Houston, TX, USA) and Validyne (Northridge, CA, USA) pressure transducer. Spectrophotometer and pressure transducer were calibrated daily against high-purity sample gases and independently calibrated instruments, respectively.

Respiration and gas data channels (mask inflow, pressure, %O₂, %CO₂, chamber altitude) were recorded at 100 Hz via LabVIEW script. S_pO₂ and HR were recorded at 1 Hz using a Nonin (Plymouth, MN, USA) Wrist-Ox® 3150 oximeter with 8000R sensor on the left temple. Peripheral S_pO₂ and HR were also monitored for safety via displays outside the chamber, using a Masimo (Irvine, CA, USA) SET® Rainbow oximeter on the left hand or an Athena GTX® (Johnston, IA, USA) HSPro on the left arm. Data recording devices were synchronized before each flight.

The PICT was presented using a laptop running an F/A-18F aircraft model in X-Plane V11 (Laminar Research, Columbia, SC, USA). Presentation was controlled using a LabVIEW script to set conditions and record data. The visual simulation depicted overcast instrument conditions and a head-up display (HUD) incorporating a horizon, climb-dive ladder, altitude (feet), airspeed (knots), and heading indicators. Below this, a synthetic panel displayed head-down instruments and engine settings. In the PICT, the subject was instructed to maintain a straight flight path at altitude 15,000 ft (4572 m), 350 kn indicated airspeed, and heading 090°. Performance was assessed in part by recording how continuously subjects could maintain corridor values between 14,800–15,200 ft (4511–4633 m), 340–360 kn, and 088–092°, respectively. Task difficulty was added by incorporating a time-varying disturbance in the aerodynamic model: a prevailing 10-kn wind was added to the surrounding airmass in a vector whose direction changed continuously at 4°/s. As a result, maintaining target airspeed required continual throttle modulation, with attendant effects (via nonconstant lift and drag) on altitude and heading. Aircraft states and flight control settings were recorded in LabVIEW at a sampling rate of approximately 39 Hz. Tracking performance was calculated across the last 7 min of each epoch to allow subjects time at the start to establish straight-and-level flight. Altitude, airspeed, and heading tracking were assessed using root-mean-square (RMS) error:

$$RMS = \sqrt{\frac{\sum_1^n (x_i - \bar{x})^2}{n}}$$

where $(x_i - \bar{x})$ is the deviation from each parameter's target value and n is the number of samples in the epoch. Each

component RMS error value was normalized by dividing by the target value: altitude 15,000, airspeed 350, heading 090. The mean of the three normalized RMS values was then calculated to represent overall piloting error within each epoch. This overall performance metric is named flight-sim error (FSE).¹⁴ PICT performance was also assessed using a time-out-of-bounds (TOOB) metric comprising the cumulative time during which the subject permitted any of the three flight parameters to deviate from the corridor boundaries. TOOB was recorded across the last 7 min of each epoch.

The UAR was also presented via LabVIEW script using XPlane. This task assessed subjects' ability to perceive and correct an unusual attitude state as might occur during situational distraction. UAR presented eight far-from-level attitude states in random order, comprising all possible combinations of upward and downward 30° pitch with left and right bank states of 45° and 135°. In each trial, the script reset XPlane to display a dark screen and then restored the display with the aircraft placed in one of the above states. The subject was instructed to use the flight controls and HUD indicators to correct bank and then pitch to regain straight-and-level flight. UAR performance was assessed using three metrics: total response time (RTT) from trial start until the subject stabilized attitude for 2 s continuously, keeping pitch within ±10° of the horizon and bank within ±5° from level; initial response time (RTI) recorded from trial start until the subject's first joystick deflection beyond 10% in the bank dimension; and correct response time (RTC) from trial start until the subject's first joystick deflection beyond 10% in the correct direction (e.g., leftward when the trial presents a right-banked attitude).

Statistical Analysis

FSE and TOOB were analyzed using one-way repeated measures ANOVA (SPSS Version 19, IBM, Armonk, NY, USA) including one independent variable called Epoch with five periods: GLP, 10,000 ft (3050 m), 14,000 ft (4270 m), 17,500 ft (5338 m), and Recovery. Datasets were screened for normality using Kolmogorov-Smirnov tests. Where Mauchly's test identified departures from sphericity, Greenhouse-Geisser (G-G) correction was applied to degrees of freedom. Least significant difference contrasts (equivalent to two-tailed *t*-tests; $\alpha = 0.05$) were used to detect differences between 5338 m vs. GLP and Recovery vs. GLP epochs. To increase sensitivity with this small sample, contrasts did not employ multiple-comparison correction, which slightly increased the chance of Type I error. UAR RTT, RTI, and RTC ANOVAs employed additional parameters, Bank and Pitch, to identify effects of aircraft state on attitude recovery, yielding a three-way, $5 \times 2 \times 2$ repeated measures design.

Mean values for S_pO₂ and HR were calculated across each epoch and averaged across flights. Respiration metrics were calculated in postprocessing using temporal analysis of flow and spectrophotometric data. Time boundaries for each breath were assigned at the minima of recorded flow into the mask. Each breath was assigned a time stamp at the time of maximum inhalation flow. Respiration rate (*f*) was calculated in breaths/min using time elapsed between successive breaths.

Table 1. PICT and UAR Piloting Metrics.

	GLP	3050 m	4270 m	5338 m	RECOVERY	F(WITHIN DF, ERROR DF)	SIG.	GLP vs. 5338 m	GLP vs. RECOVERY
PICT Metrics									
Flight simulator error (FSE, normalized metric)	0.024 (0.002)	0.025 (0.003)	0.031 (0.004)	0.032 (0.004)	0.029 (0.003)	$F(4, 24) = 4.0$	0.013	x	x
Time out of bounds (TOOB; seconds)	10.81 (6.6)	23.12 (12.1)	53.89 (21.9)	73.44 (32.4)	60.06 (13.9)	$F(4, 24) = 4.6$	0.007	d = 0.97 [0.057]	d = 1.13 x
UAR Metrics									
Total Response Time (RTT; seconds) by epoch:	10.17 (0.553)	9.94 (0.554)	10.07 (0.650)	9.62 (0.457)	9.86 (0.534)	$F(4, 24) = 2.0$	NS	x	d = 1.63
RTT in seconds, by bank									
L/R 45°	L/R 135°	L/R 135°	F(WITHIN DF, ERROR DF)	SIG.					
PITCH UP	PITCH DOWN	PITCH DOWN	$F(1, 6) = 4.5$	[0.078]					
RTT in seconds, by pitch									
GLP	3050 m	4270 m	5338 m	RECOVERY					
10.73 (0.398)	9.13 (0.702)	0.901 (0.036)	0.940 (0.057)	0.898 (0.034)	$F(4, 24) = 5.8$	0.002			x
1.035 (0.046)	0.969 (0.045)								d = -1.28
Initial response time (RTI; seconds) by epoch:									
L/R 45°	L/R 135°	F(WITHIN DF, ERROR DF)	SIG.						
0.956 (0.056)	0.940 (0.031)	$F(1, 6) = 5.8$	NS						
RTI in seconds, by bank:									
PITCH UP	PITCH DOWN								
0.910 (0.031)	0.986 (0.048)	$F(1, 6) = 10.2$	0.019						
Initial correct RT (RTC in seconds) by epoch:									
GLP	3050 m	4270 m	5338 m	RECOVERY					
1.053 (0.050)	1.000 (0.036)	0.938 (0.043)	0.979 (0.054)	1.039 (0.118)	$F(1.3, 7.6 (G-G)) = 313.8$	NS			
RTC in seconds, by pitch:									
L/R 45°	L/R 135°	F(WITHIN DF, ERROR DF)	SIG.						
1.029 (0.068)	0.975 (0.034)	$F(1, 6) = 0.503$	NS						
RTC in seconds, by bank:									
PITCH UP	PITCH DOWN	F(WITHIN DF, ERROR DF)	SIG.						
0.925 (0.034)	1.079 (0.057)	$F(1, 6) = 7.3$	0.035						
CONTRAST OF INTEREST									
COHEN'S d	REQUIRED N								
GLP vs. 4270 m	13								
GLP vs. 4270 m	11								
GLP vs. 5338 m	12								
GLP vs. 5338 m	5								
GLP vs. Recovery	-1.28								

Marginal means are listed followed by standard errors of the mean in parentheses "0". Right two columns list two-tailed contrast; "x" indicates t significance at $\alpha = 0.05$. Cohen's d indicates effect size. Brackets "[]" indicate trends approaching significance ($0.05 < \alpha < 0.1$). Bottom rows indicate comparisons of potential interest in future studies including milder, 4270-m exposure, with projected sample sizes.

PICT: precision instrument control; UAR: unusual attitude recovery; DF: degrees of freedom; GLP: ground level pressure; SIG: significance; NS: not significant; L/R: left/right.

Inhalation tidal volume (V_T) was recorded in L/breath by integrating mask flow across breath duration. Ventilation (\dot{V}_E) in $L \cdot \text{min}^{-1}$ comprised the product of f and V_T . A peak-finding algorithm was applied to the spectrophotometric mask data to identify end-tidal minimum O_2 and maximum CO_2 concentrations within each breath. These were entered in a conversion equation, which accounts for water vapor pressure (not registered by the spectrometer) using an assumed value of 47 mmHg to calculate end-tidal partial pressures (P_{ET,O_2} and P_{ET,CO_2}). (Note that assuming this fixed vapor pressure introduces some imprecision to P_{ET,O_2} and P_{ET,CO_2} calculations.) Mean f , V_T , \dot{V}_E , P_{ET,O_2} , and P_{ET,CO_2} values were calculated across breaths within each epoch and averaged across flights.

RESULTS

Pilot performance metrics are shown in **Table I**. Among PICT metrics, Epoch influenced FSE [$F(4, 24) = 4.0$, $P < 0.02$], with contrasts identifying greater normalized piloting error during 17,500 ft (5338 m) and Recovery than GLP. TOOB varied with Epoch [$F(4, 24) = 4.6$, $P < 0.01$], with subjects exceeding corridor boundaries longer during 5338 m and Recovery than GLP, though the former difference missed significance ($P = 0.057$). **Fig. 1** illustrates FSE and TOOB means by Epoch.

Among UAR metrics, RTT varied with Pitch [$F(1, 6) = 17.0$, $P < 0.01$], but not Bank [$F(1, 6) = 4.5$, $P = 0.078$] or Epoch [$F(4, 24) = 2.0$, $P = 0.121$], though a contrast identified shorter RTT at 17,500 ft (5338 m) vs. GLP; a Bank*Pitch interaction was identified [$F(1, 6) = 13.8$, $P < 0.02$] whereby the gain from starting pitch-down was greater with shallower bank. The nondirectional RTI metric varied with Epoch [$F(4, 24) = 5.8$, $P < 0.003$] and Pitch [$F(1, 6) = 10.2$, $P < 0.02$], with faster initial responses recorded during Recovery vs. GLP, and no interaction. With correct response direction considered in the

RTC metric, only the Pitch effect remained [$F(1, 6) = 7.3$, $P < 0.05$]; subjects responded sooner in pitch-up attitudes, with no interactions or Epoch effect identified. We note that while analyses of variance are typically robust against nonnormality, the Kolmogorov-Smirnov test detected some departure from normality in the UAR metrics.

Physiology metrics also exhibited nonnormality, but nevertheless showed striking variation among altitude conditions (**Table II**). S_pO_2 varied with Epoch, with lower values at 17,500 ft (5338 m) than GLP. HR varied with Epoch, with higher values at 17,500 ft (5338 m) than GLP and lower values during Recovery. Mean respiration rate remained largely uniform from GLP throughout exposure, but decreased during Recovery (see **Fig. 2**, which also illustrates S_pO_2). V_T and \dot{V}_E varied across Epoch, with subjects inspiring more L/breath and more L/min at 17,500 ft (5338 m) vs. GLP. P_{ET,O_2} decreased at altitude vs. GLP. P_{ET,CO_2} also decreased at altitude, with lower means at 17,500 ft (5338 m) and in Recovery.

DISCUSSION

The primary study objective was accomplished: the apparatus was shown to be effective for measuring moderate hypoxia effects. While the study's limited scope marks findings as preliminary, the paradigm proved sufficiently sensitive to detect performance changes, associated mainly with piloting precision, in a hypoxia regime where measured effects are typically subtle,^{5,16} while continuously monitoring a meaningful group of physiological metrics. In PICT, the normalized FSE and temporal TOOB metrics both indicated effects of progressive hypoxia, whereby piloting error increased with altitude and remained elevated relative to baseline levels. Observed findings extended from psychophysical to physiological domains and included both direct and indirect hypoxia markers. Hypobaric elicited a classic altitude response in which subtle piloting impairment was accompanied by indicators of hypoxia (decreased S_pO_2 and P_{ET,O_2} , elevated HR), hyperventilation (elevated V_T and \dot{V}_E), and hypocapnia (decreased P_{ET,CO_2}).

UAR findings were more complex; while the task offers a promising instrument, no coherent configuration of effects emerged, and our observations suggest that subjects required more training and were distinguished by individual performance differences. In RTT, attaining stability from pitch-up probably took longer because controls responded sluggishly on a slowing aircraft model. Conversely, faster initial RTI and RTC responses in pitch-up trials could be explained perceptually: since pitch-up HUD symbology is solid, it might be processed faster than the dashed pitch-down indicators. The effect of Epoch on UAR was ambiguous; responses were not slowed during exposure and contrasts indicated accelerated responses at altitude in RTT and during recovery in the nondirectional RTI response. It is possible that subjects continued to learn throughout UAR testing, accelerating responses in later epochs. Alternatively, these contrasts resemble an earlier observation of decreased RT in executive tasks during

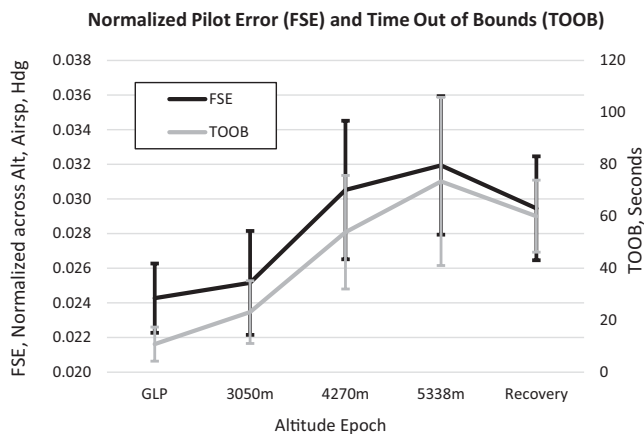


Fig. 1. Mean precision piloting error metrics recorded across five altitude epochs. FSE represents RMS error across altitude, airspeed, and heading parameters (metric is normalized and dimensionless). TOOB represents total duration of all data intervals in which a subject permitted any of the parameters to exit the corridor boundaries. Bars represent the standard error of the mean in each epoch.

Table II. Physiological Metrics: Means by Epoch.

METRICS	GLP	3050 m	4270 m	5338 m	RECOVERY
O ₂ saturation (S _{pO₂} ; %)	97.9 (0.5)	92.1 (0.7)	85.7 (1.2)	79.5 (1.6)	97.7 (0.4)
Heart rate (HR; bpm)	79.1 (5.0)	83.9 (5.1)	88.1 (5.2)	93.0 (5.3)	76.2 (4.9)
Respiration rate (f; breaths/min)	14.9 (0.6)	14.8 (0.6)	15.1 (0.5)	14.9 (0.7)	12.2 (0.9)
Tidal volume (V _T ; L/breath)	0.699 (0.027)	0.742 (0.029)	0.806 (0.037)	0.929 (0.059)	0.859 (0.091)
Ventilation (V _E ; L/min)	10.4 (0.3)	11.0 (0.3)	12.1 (0.5)	13.7 (0.6)	10.3 (1.1)
End-tidal O ₂ (PP P _{ET} O ₂ ; mm)	108.0 (5.0)	65.8 (4.3)	53.5 (3.9)	45.0 (3.5)	104.2 (6.5)
End-Tidal CO ₂ (PP P _{ET} CO ₂ ; mm)	32.5 (4.4)	30.4 (4.0)	28.3 (3.7)	25.9 (3.4)	29.0 (4.1)

Marginal means are listed followed by standard errors of the mean in parentheses.

hypoxia,² which could be explained as a speed-accuracy trade-off. This could be tested in future studies incorporating more training and larger sample sizes to determine whether subjects recovering from altitude execute corrections sooner and with less inhibition.

The persistent elevation in PICT error metrics bears comparison to various findings of post-hypoxic impairment, which have been referred to as “hypoxia hangover.” Lingering impairment has been reported in paradigms where S_{pO₂} recovered rapidly, suggesting that S_{pO₂} might not be the only predictor of postexposure performance and that other, slower-recovering factors such as cortical perfusion, inflammation, or axonal potentiation be considered.^{4,14} Here, metrics of precision piloting error remained elevated after exposure even as S_{pO₂} recovered swiftly. Seeking to explain this, we observe that V_E increased with altitude as P_{ET}CO₂ decreased, embodying a combination of hyperventilation (a hypoxia sequela) with hypocapnia (depressed CO₂), which can induce respiratory alkalosis,¹¹ cognitive deficits,^{7,8} and attendant chemoreceptor responses that could require 45–100 min for recovery.^{9,10}

To account for effects during recovery, the most parsimonious explanation is that hypobarica, combined with progressive hypoxia-induced hyperventilation of considerable duration—at least 33 min above 10,000 ft (3050 m)—resulted in respiratory alkalosis that recovered more slowly than S_{pO₂}. Consistent with

this, persistently ragged piloting could be an indicator of incomplete neurocognitive recovery while compensatory respiratory inhibition engaged to counter the effects of hypocapnia. Future studies should investigate with greater temporal resolution indicators of delayed psychomotor recovery, including elevated FSE, and physiological indicators, including P_{ET}CO₂ and respiration rate.

The primary limitation of this exploratory study is its modest statistical power. In recognition of this, these findings justify a robust subsequent effort to refine this paradigm for measuring piloting impairment and recovery. Future studies can incorporate larger samples (Table I recommends sample sizes to guide specific comparisons) and counterbalanced presentation. Future UAR applications should also include more training.

Existing findings regarding hypoxia and hypocapnia etiology and recovery indicate that moderate altitude exposure induces changes in executive processing, early perception, and auditory performance which may persist after blood O₂ saturation has recovered but CO₂ concentration may not have. This study demonstrates a hypobaric test paradigm to characterize altitude-related changes in a broad range of additional constructs, including precision piloting and respiration.

ACKNOWLEDGMENTS

This study was completed with the sponsorship of the USAF School of Aerospace Medicine. It was approved by the Air Force Research Laboratory 711th HPW Institutional Review Board in compliance with applicable regulations governing the protection of human subjects.

The authors are grateful for the support of USAFSAM and 711th HPW and for the valuable technical contributions of Nathan Dillon and David Bowden (interface programming and data acquisition), William Ercole and Rick Evans (developing PICT and UAR task parameters), Andrew Mojica and Joseph Fischer (data evaluation), and Lynn Menchaca (data acquisition).

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the authors' employers, the U.S. Air Force, the Department of Defense, or the U.S. Government.

Financial Disclosure Statement: The authors have no competing interests to declare.

Authors and Affiliations: Jeremy Beer, Ph.D., M.Phil., Bria Morse, M.S., B.S., Todd Dart, Ph.D., M.S., and Samantha Adler, Ph.D., B.S., KBR, Brooks Aerospace Environment Protection Laboratory, San Antonio, TX, USA, and Paul Sherman, M.D., B.S., USAF School of Aerospace Medicine/FE and Department of Radiology, 59th Medical Wing, JBSA Lackland, San Antonio, TX, USA.

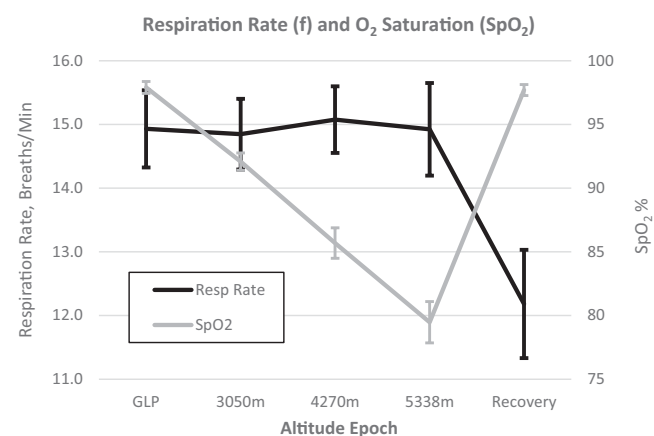


Fig. 2. Mean respiration rate (f) and percentage of oxygenated hemoglobin (S_{pO₂}) recorded across five altitude epochs. Bars represent the standard error of the mean in each epoch.

REFERENCES

1. Aebi MR, Bourdillon N, Noser P, Millet GP, Bron D. Cognitive impairment during combined normobaric vs. hypobaric and normoxic vs. hypoxic acute exposure. *Aerosp Med Hum Perform.* 2020; 91(11):845–851.
2. Asmaro D, Mayall J, Ferguson S. Cognition at altitude: impairment in executive and memory processes under hypoxic conditions. *Aviat Space Environ Med.* 2013; 84(11):1159–1165.
3. Beer JMA, Shender BS, Chauvin D, Dart TS, Fischer J. Cognitive deterioration in moderate and severe hypobaric hypoxia conditions. *Aerosp Med Hum Perform.* 2017; 88(7):617–626.
4. Blacker KJ, McHail DG. Time course of recovery from acute hypoxia as measured by vigilance and event-related potentials. *Physiol Behav.* 2021; 239:113508.
5. Bouak F, Vartanian O, Hofer K, Cheung B. Acute mild hypoxic hypoxia effects on cognitive and simulated aircraft pilot performance. *Aerosp Med Hum Perform.* 2018; 89(6):526–535.
6. Dart T, Gallo M, Beer J, Fischer J, Morgan T, Pilmanis A. Hyperoxia and hypoxic hypoxia effects on simple and choice reaction times. *Aerosp Med Hum Perform.* 2017; 88(12):1073–1080.
7. Friend AT, Balanos GM, Lucas SJE. Isolating the independent effects of hypoxia and hyperventilation-induced hypocapnia on cerebral haemodynamics and cognitive function. *Exp Physiol.* 2019; 104(10):1482–1493.
8. Gradwell DP. Hypoxia and hyperventilation. In: Gradwell DP, Rainford DL, editors. *Ernsting's aviation and space medicine*, 5th ed. Boca Raton (FL): CRC Press; 2016:49–64.
9. Higashi H, Kano T, Shimoji K, Moriora T, Sances AN, Jr. Effects of acute hypocapnia and hypercapnia on neuromuscular transmission and on monosynaptic spinal reflex in wakeful man. *Br J Anaesth.* 1972; 44(11):1128–1132.
10. Krapf R, Caduff P, Wagdi P, Stäubli M, Hulter HN. Plasma potassium response to acute respiratory alkalosis. *Kidney Int.* 1995; 47(1):217–224.
11. Leacy JK, Day TA, O'Halloran KD. Is alkalosis the dominant factor in hypoxia-induced cognitive dysfunction. *Exp Physiol.* 2019; 104(10):1443–1444.
12. Pilmanis AA, Balldin UI, Fischer JR. Cognition effects of low-grade hypoxia. *Aerosp Med Hum Perform.* 2016; 87(7):596–603.
13. Rice GM, Snider D, Drollinger S, Grell C, Bogni F, et al. Dry-EEG manifestations of acute and insidious hypoxia during simulated flight. *Aerosp Med Hum Perform.* 2019; 90(2):92–100.
14. Robinson FE, Horning D, Phillips JB. Preliminary study of the effects of sequential hypoxic exposures in a simulated flight task. *Aerosp Med Hum Perform.* 2018; 89(12):1050–1059.
15. Shaw DM, Cabre G, Gant N. Hypoxic hypoxia and brain function in military aviation: basic physiology and applied perspectives. *Front Physiol.* 2021; 12:665821.
16. Temme LA, Still DL, Acromite MT. Hypoxia and flight performance of military instructor pilots in a flight simulator. *Aviat Space Environ Med.* 2010; 81(7):654–659.
17. Uchida K, Baker SE, Wiggins CC, Senefeld JW, Shepherd JRA, et al. A novel method to measure transient impairments in cognitive function during acute bouts of hypoxia. *Aerosp Med Hum Perform.* 2020; 91(11):839–844.
18. Varis N, Parkkola KI, Leino TK. Hypoxia hangover and flight performance after normobaric hypoxia exposure in a hawk simulator. *Aerosp Med Hum Perform.* 2019; 90(8):720–724.
19. Viscor G, Torrella JR, Corral L, Ricart A, Javierre C, et al. Physiological and biological responses to short-term intermittent hypobaric hypoxia exposure: from sports and mountain medicine to new biomedical applications. *Front Physiol.* 2018; 9:814.
20. Williams TB, Corbett J, McMorris T, Young JS, Dicks M, et al. Cognitive performance is associated with cerebral oxygenation and peripheral oxygen saturation, but not plasma catecholamines, during graded normobaric hypoxia. *Exp Physiol.* 2019; 104(9):1384–1397.