Hyperoxia and Hypoxic Hypoxia Effects on Simple and Choice Reaction Times

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INTRODUCTION: Effects of exposure to hyperoxia ($PiO_2 > 105$ mmHg), normoxia (PiO_2 95–105 mmHg) and hypoxia ($PiO_2 < 95$ mmHg) on simple and choice reaction performance tasks were evaluated.

METHODS: Ten subjects performed simple and choice reaction time tests (SRT and CRT, respectively) at ground level for 40 min (20 min normoxic, 20 min hyperoxic, randomly assigned), 3048 m (10,000 ft) for 75 min (15 min hyperoxic, 60 min hypoxic), 4572 m (15,000 ft) for 60 min (15 min hyperoxic, 45 min hypoxic), and 6096 m (20,000 ft) for 35 min (15 min hyperoxic, 20 min hypoxic). SRT and CRT tests were also conducted at ground level 1 h after normoxic rest (recovery) to assess any recovery time effect on these psychomotor tasks.

RESULTS: Total response time (TRT) significantly increased by 15 ms to 25 ms at all three altitudes for both the SRT and CRT tasks. At and below 4572 m, the performance changes were gradual over the duration of the exposures, whereas at 6096 m these changes were immediate. After 1 h, no performance decrement was measured. There was no statistical evidence that ground-level performance on these tasks was improved in hyperoxic vs. normoxic conditions.

DISCUSSION: Results suggest mild decrements in reaction time due to hypoxia may occur as low as 3048 m (10,000 ft) while hyperoxia showed no positive effect on accuracy or reaction time at ground level or higher when performing simple and choice psychomotor reaction tasks.

KEYWORDS: hypoxia, hyperoxia, cognition, total response time, recovery.

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With the rise of aviation in the first half of the 20th century, along with the increasing interest in travel to mountainous terrain afforded by this new transport technology, questions concerning the effects of acute ascent (i.e., ascent without time for physiological acclimation) to altitude on cognition became increasing relevant. One of the earliest assessments of cognitive performance at altitude was published by McFarland in 1937, who presented evidence for cognitive degradation, including degraded psychomotor reaction time, in subjects following acute ascent by aircraft from Lima (sea level) to Morococha, Peru (4538 m;14,900 ft).[14] Subsequent studies and reviews further defined the effects of altitude on cognitive impairment, including degraded performance,[8,11,22] while also identifying the altitude conditions under which cognitive impairment begins. For example, Li et al.[13] reported degraded reaction time above 3600 m (11,811 ft), but not below 2800 m (9186 ft) while Ledwith[12] reported changes in response time at altitudes as low as 1524 m (5000 ft) when the reaction response was paired with a mathematical task. Some studies have even addressed cognitive performance effects of hypoxia at 3048 m (10,000 ft) with exercise[2] and at 3810 m (12,500 ft), without and with moderate exercise.[1,13] Pilmanis et al.[19] reported on a low-grade hypoxia study with 93 subjects that indicated a very minor decrease in continuous performance at 1524 m, 2438 m, and 3657 m (5000, 8000 and 12,000 ft) out of the 7 cognitive performance tasks measured by the Automated Neuropsychological Assessment Metrics (ANAM) battery, although the authors questioned the operational relevance of the measured decrements. More recently, subjects operating a multitask workstation at higher chamber

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altitudes of 5486 m and 7620 m (18,000 ft and 25,000 ft) exhibited sharper deficits, particularly in mathematical and auditory tasks, the latter of which reportedly remained slightly impaired in a short postexposure period.[3]

Since mental workload can be very high in aircrew,[21,25] understanding the effects of brain oxygen (O_2) saturation level on cognition is important to maximize aircrew performance. Hypoxia has been associated with declines in various aspects of cognitive performance.[11,18,24] The converse relation, however, whereby hyperoxia (i.e., breathing O_2 at concentrations greater than the sea level alveolar oxygen pressure equivalent of about 102 mmHg[4]) yields enhanced performance, has not been established as clearly. While some investigations have suggested positive effects of hyperoxia on cognition,[5,15,20] other studies have not found such a link.[1,7,9] One example of this ambiguity is reported by Hemelryck et al.,[10] who compared hyperoxia and normoxia conditions and obtained mixed results, with statistical differences reported in mathematical and trail making tasks but no difference in a reaction time task. Since hyperoxic breathing mixtures are common in aviation as a prophylactic measure against hypoxia and decompression sickness, demonstrating that hyperoxia delivers beneficial effects on mental and/or motor performance would provide an additional impetus for advocating high breathing O_2 concentrations above those necessary for peak performance in the demanding flight environment.

Conversely, hypoxic hypoxia studies have historically focused on providing O_2 levels sufficient to maintain consciousness but not necessarily to provide for optimal mental performance. Training on, and research into, hypoxic hypoxia with respect to brain function is most often focused on time of useful consciousness (TUC), that is, the duration from exposure to hypoxia until the point of cognitive impairment sufficient to cause an inability to take corrective action.[6] Less well understood is the progression of cognitive impairment prior to TUC, i.e., whether hypoxia induces a steady decline over time or remains fairly stable until a critical point of impairment leads to a rapid decline. Understanding the degree and rate of cognitive decay prior to reaching TUC will provide a clearer perspective of the O_2 requirements needed to optimize aircrew performance.

While early studies relied primarily on hypobaric hypoxia to address acute hypoxia effects on cognitive performance, the use of normobaric hypoxia is becoming more common, although information on the congruency of normobaric and hypobaric hypoxia symptoms and onset characteristics are still being established. Phillips et al.[18] helped close this knowledge gap at higher altitudes in their investigation into hypoxia at 5486 m (18,000 ft) using a reduced O_2 breathing device (ROBD). Performance was measured over the duration of the hypoxic exposure using five cognitive test batteries: the Freiburg Visual Acuity and Contrast Test (FrACT), the Number Stroop Task, Simple Reaction Time, Choice Reaction Time, and the NASA Task Load Index (NASA-TLX). Their study reported overall performance during the hypoxic and recovery phases rather than performance change during each phase. A result of particular note from their study is the reported persistence of elevated response times (a performance decline) at 60 and 120 min postexposure, although values had returned to baseline levels after 24 h. Further investigation of this observation is therefore pertinent to the understanding of any persistent effects of hypoxia on cognitive performance in aviators.

This present study was implemented to address two specific issues. The first is whether exposure to ground level hyperoxia improves the ability to perform simple psychomotor tasks as compared to ground level normoxia (specifically, 100% O_2 at ground level vs. 21% O_2 at ground level). The second issue is to validate the rate of decline in performance of these simple tasks at altitudes of 3048, 4572, and 6096 m (10,000, 15,000, and 20,000 ft), and, as a corollary, to determine whether performance returns to baseline by 1 h after exposure.

METHODS

Subjects

Ten nonsmoking, active duty military male personnel completed this protocol, which included ground-level training and testing. The subjects (average age 31.4 $\pm$ 6.8 yr) gave informed consent to participate in the study in accordance with the U.S. Air Force 711th Human Performance Wing Institutional Review Board guidelines. All subjects met medical requirements for a U.S. Air Force Class III flight physical, were screened for conditions that might abnormally impair their tolerance to altitude, and received a medical screening before every altitude exposure. One female subject initiated the study but withdrew due to pregnancy.

Equipment

A hypobaric chamber located at the KBRwyle facilities at Brooks City-Base in San Antonio, TX, was used for all tests. Subjects wore the following standard and modified Air Force aircrew flight equipment (AFE) for altitude chamber exposures: HGU-55/P flight helmet modified with Nonin 8000R Reflector transducer positioned in the left ear cup over the left superficial temporal artery, CRU-60/P O_2 hose connector, and MBU-20/P O_2 mask fitted with taps for measurement of pressure and gas content via mass spectrometer.

Oxygen and air were provided to subjects via a CRU-73 O_2 regulator set to normal pressure, 100% O_2, and the ON position. This setting provided subjects with undiluted breathing gas supplied from pressurized bottles of either aviator's breathing oxygen (ABO, $\geq$ 99.5% O_2) or air (21% O_2). For altitude testing conditions, subjects breathed ABO for 15 min, which produced hyperoxic conditions. After this, the breathing gas was switched to air (21% O_2) to enable the collection of performance data while normoxic (ground level or GL) or hypoxic (altitude) conditions obtained. This was accomplished through a gas manifold system which allowed subjects to be blinded to the gas switch.

The psychomotor tests comprised a simple reaction time (SRT) and a choice reaction time (CRT) task.[18] These relatively

low-level tasks were selected to correspond to aircrew tasks such as deactivating a warning light or reacting to more than one input option on a multifunction display. Subjects' responses were recorded and timed using a 10-key number board. For SRT testing, the subject was required to hold down the number 5 ("home") key on the key pad until an up arrow appeared on the subject monitor, at which point the subject was to release the 5 key and press the 8 key (directly above the 5 key). For CRT the display arrow had four possible orientations: up, down, left, or right. The subject was instructed to press and hold the 5 key until an arrow appeared, at which time the subject was to release the 5 key and press the corresponding direction key on the key pad. For both tests, the arrows were presented at random intervals from 2 to 10 s. Each test set consisted of five to seven SRTs followed by, or preceded by, five to seven CRTs (order was randomly determined by the software). Subjects performed an SRT/CRT set at the start of each minute. The time from a directional arrow being presented on the screen to releasing of the 5 key is defined as reaction time (RT). The time from the 5 key being released to when a response key is pressed is defined as movement time (MT). The total response time (TRT) is defined as the sum of the RT and MT. In addition to the TRT measures, accuracy of the response (right/wrong) was also recorded for analysis. Subjects were able to complete an SRT/CRT set in 45 to 48 s, giving them an approximately 15-s rest between test sets. After nine test sets during baseline and hypoxia testing, subjects were given a 1-min break from performing the tests to help reduce attention fatigue.

Procedure

Prior to testing, subjects completed two half-hour computer task training sessions on separate days. Subjects completed four chamber test sessions; the initial session was always at ground level as a control. The subsequent three flights were at altitude and randomly assigned. Each chamber test run lasted 35 to 75 min in duration, depending on altitude, over four tests. At 1 h postflight, subjects performed the tests for 15 min at ground level to assess if any residual effects were detectable.

For the initial test condition (Normoxia and Hyperoxia) subjects were randomly selected to breathe either 21% O_2 (normoxic) or ABO (hyperoxic) while performing the psychomotor assessment task at ground level for 20 min. Following this initial phase, the breathing gas was switched (from 21 to ABO or vice versa) and subjects again performed the psychomotor assessment task at ground level for 20 min. Subjects were blinded to the breathing gas change.

For test Conditions 2 through 4 [3048 m (10,000 ft), 4572 m (15,000 ft), 6096 m (20,000 ft); Hypoxia] subjects breathed ABO (hyperoxic condition) while performing the psychomotor assessment task for the first 15 min of exposure as a daily control condition (baseline phase). Following this phase, the breathing gas was switched to air (21% O_2, hypoxic condition) and subjects performed the psychomotor assessment task for 60, 45, or 20 min [for altitudes of 3048 m (10,000 ft), 4572 m (15,000 ft), 6096 m (20,000 ft), respectively] until completion of time or until either one of two termination criteria were met: onset of subject's hypoxia symptoms (based on their altitude chamber training) or if end tidal Po_2 fell below 30 mmHg as measured by a gas chromatograph mass spectrometer. Subjects were blinded to the breathing gas change. Upon completion of time or early termination the psychomotor testing was stopped and subjects switched to ABO for duration of the altitude chamber descent to ground level, whereupon subjects were returned to breathing ambient air by allowing them to drop their mask.

For test condition 4 (6096 m) only, subjects breathed ABO for 30 min prior to ascent as a precaution against decompression sickness. This resulted in having subjects exposed to a total time of 45 min under hyperoxic conditions prior to the hypoxic condition.

At 1 h after completion of each test condition, subjects performed 15 min of psychomotor testing at ground level breathing ambient air (21% O_2) inside the altitude chamber to assess whether any effects of hypoxia on cognition persisted under postexposure normoxic conditions. During this phase of testing, subjects did not wear AFE.

Statistical Analysis

For statistical analysis, the raw data were compressed over time into quantities amenable for testing as follows. To estimate accuracy, the percent of correct responses was calculated (for each psychomotor test, separately) for each subject in each phase of each experimental condition. For each experimental measure (RT, MT, and TRT), the median of all responses within a given phase was calculated (for each psychomotor test, separately) for each subject in each phase of each experimental condition. These compressed data were used in all of the statistical testing reported in this paper.

To test whether hyperoxia increased psychomotor function, each outcome measure from test condition 1 was analyzed using two Student's paired *t*-tests: one comparing the hyperoxia phase with the normoxic phase, and one comparing the hyperoxic phase with the postexposure normoxic phase. To test for hypoxic effects, each outcome measure from test conditions 2 through 4 was subjected to a repeated measures analysis of variance (ANOVA) with two independent factors (phase and altitude). The ANOVA was followed up with post hoc paired *t*-tests for each altitude, separately, to determine whether there was a performance decrease when going from the hyperoxic (baseline) phase to the hypoxic (altitude) phase, and to compare the post exposure normoxic mean with the hyperoxic mean to determine whether any detrimental effects were present after 1 h of recovery. Since there was no historical precedent for performance to be better during the hypoxic phase[14,17,22] nor evidence the normoxic recovery postexposure phases would be better compared to the initial hyperoxic phase,[7,15,20] it was determined that one-tailed *t*-tests were appropriate for the comparisons made in this report. The use of one-tailed tests increases the power of detecting differences, and since, ultimately, the question of pilot safety when exposed to altitude is a major issue, it was deemed important to make the tests as sensitive as possible. For the same reason, no adjustments for

multiple testing were made to the *t*-tests. For all tests, $P = 0.05$ (one-tailed) was chosen as the critical level for determining statistical significance. It was calculated that the use of 10 subjects would provide an 89% chance (power) of detecting changes of 1 SD of the difference in magnitude when testing at the one-tailed 0.05 alpha level.

RESULTS

For Test Condition 1 (Normoxia; Hyperoxia), for both the CRT and SRT tasks, no significant performance degradation was seen in accuracy, RT, MT, or TRT for the hyperoxic vs. normoxic baseline or the hyperoxic vs. normoxic recovery postexposure comparisons. Results are summarized in **Table I**. In short, no evidence was found that simple psychomotor function was better when breathing ABO at ground level pressure than it was when breathing 21% O_2 at ground level pressure.

For test Conditions 2 through 4 [Hypoxia; 3048 m (10,000 ft), 4572 m (15,000 ft), 6096 m (20,000 ft)], descriptive statistics for Accuracy, RT, MT, and TRT for both CRT and SRT, along with post hoc *t*-test results are shown in **Table II**. Note that to maintain table uniformity and provide as much information as possible, *t*-test results are shown even for the outcome measures where the primary ANOVA did not yield significant results. As will be seen, the ANOVA results and *t*-test results were generally in good agreement.

For Total Response Time the ANOVA detected a significant phase main effect for both CRT and SRT [$MSE = 544.1$, $F(2,18) = 5.33$, $P = 0.015$; and $MSE = 643.9$, $F(2,18) = 12.09$, $P < 0.001$]. No significant phase by altitude interaction was found. Table II shows that, for each of the three altitude test conditions, TRT increased significantly (i.e., degraded) for both psychomotor tests during the hypoxic phase compared to the corresponding hyperoxic baseline phase. Visual inspection of the minute-by-minute data showed that, for the 3048 and 4572 m (10,000 and 15,000 ft) test conditions, TRTs at the beginning of the hypoxic phase were comparable to baseline TRTs, but increased in magnitude as hypoxia exposure time increased, with the rate of increase being greater in the 4572 m (15,000 ft) test condition than in the 3048 m (10,000 ft) test condition (**Fig. 1** summarizes 1-min data for CRT; for brevity, SRT data are not shown). For the 6096 m (20,000 ft) test condition, TRT tended to be higher than hyperoxic baseline values throughout the hypoxic phase, but did not show a clear increasing trend over time (Fig. 1). It is reasonable to assume that degradation should increase as exposure time increases at 6096 m (20,000 ft), but since the length of exposure for this condition was intentionally short to meet safety concerns (or was shorter yet due to early recognition of symptoms), confirmation of such a trend was not possible in this study.

There was no statistical evidence that TRT during the normoxic postexposure phase was significantly different than during the hyperoxic baseline phase for either psychomotor test in any of the three altitude conditions, suggesting that TRT, measured 1 h after completion of the runs, had returned to preexposure baseline levels.

The ANOVA indicated a significant main effect of phase on RT for both CRT and SRT tasks [$MSE = 398.1$, $F(2,18) = 4.01$, $P = 0.036$; and $MSE = 430.3$, $F(2,18) = 9.71$, $P = 0.001$, respectively]. No significant phase by altitude interaction was detected. As shown in Table II, the CRT task showed significant increases in RT in the hypoxic vs. the hyperoxic phase for the 3048 m (10,000 ft) and 4572 m (15,000 ft) conditions, but not for the 6096 m (20,000 ft) condition (although that difference approached significance [$P = 0.056$]). For the SRT task, significant RT increases in the hypoxic phase relative to the hyperoxic phase were seen at 4572 m (15,000 ft) and at 6096 m (20,000 ft) but not at 3048 m (10,000 ft), although this difference, too, approached significance ($P = 0.054$). These results for RT

Table I. Accuracy, Reaction Time, Movement Time, and Total Response Time for the CRT and SRT Cognitive Tasks during Hyperoxic, Normoxic, and Normoxic Post-Exposure Conditions at Ground Level (0 m).*

	CONDITION PHASE						PAIRED *t*-TEST RESULTS†							
	HYPEROXIC		NORMOXIC		NORMOXIC POST-EXPOSURE		NORMOXIC – HYPEROXIC				NORMOXIC POST-EXPOSURE – HYPEROXIC			
							DIFFERENCE				DIFFERENCE			
COGNITIVE COMPONENT	MEAN	SD	MEAN	SD	MEAN	SD	MEAN	SD	*t*-value (9df)	*P*-VALUE	MEAN	SD	*t*-value (9df)	*P*-VALUE
Choice Reaction Time Task														
Accuracy (%)	97.6	2.1	97.1	2.30	96.2	5.0	-0.4	2.0	0.69	0.255	-1.3	3.9	1.07	0.156
RT (ms)	455.2	31.3	449.0	32.7	449.5	22.6	-6.2	15.1	1.28	0.115	-5.7	28.0	0.65	0.268
MT (ms)	117.8	19.8	117.6	19.6	118.4	27.3	-0.2	10.0	0.06	0.475	0.6	11.9	0.15	0.441
TRT (ms)	580.6	23.2	572.5	31.3	574.6	35.0	-8.2	14.2	1.82	0.051‡	-6.1	32.6	0.59	0.285
Simple Reaction Time Task														
Accuracy	99.7	0.5	99.5	0.5	99.6	0.6	-0.2	0.5	1.33	0.108	0.0	1.0	0.14	0.496
RT (ms)	416.6	41.5	409.6	35.7	402.8	33.2	-7.0	19.5	1.14	0.141	-13.9	22.7	1.93	0.043‡
MT (ms)	106.7	17.5	107.2	17.4	108.4	27.7	0.5	8.9	0.16	0.438	1.7	19.2	0.28	0.394
TRT (ms)	524.6	39.4	519.6	41.0	507.3	45.5	-5.1	19.8	0.81	0.220	-17.4	28.8	1.90	0.045‡

* Table entries are means (averaged over subjects) and associated standard deviations of the compressed data for each phase of each condition.
† Student's one-tailed paired *t*-tests are testing for performance degradation when going from the hyperoxic to normoxic and from the hyperoxic to normoxic postexposure phases.
‡ Even though the *P*-value is 0.05 or less, the difference is in the wrong direction with respect to the one-tailed hypothesis that performance would degrade at the normoxic and normoxic postexposure phases.

Table II. Accuracy, Reaction Time, Movement Time, and Total Response Time for the CRT and SRT Cognitive Tasks during Hyperoxic, Hypoxic, and Normoxic Post-Exposure Conditions at Altitude.*

		CONDITION PHASE						PAIRED *t*-TEST RESULTS†							
		HYPEROXIC		HYPOXIC		NORMOXIC POST-EXPOSURE		HYPOXIC – HYPEROXIC				NORMOXIC POST-EXPOSURE – HYPEROXIC			
								DIFFERENCE				DIFFERENCE			
COGNITIVE COMPONENT	ALTITUDE (m)	MEAN	SD	MEAN	SD	MEAN	SD	MEAN	SD	*t*-value (9df)	*P*-VALUE	MEAN	SD	*t*-value (9df)	*P*-VALUE
Choice Reaction Time Task															
Accuracy (%)	3048	98.6	1.0	98.0	0.8	97.1	2.8	-0.6	1.3	1.42	0.095	-1.5	2.9	1.61	0.071
	4572	98.2	1.9	97.5	1.7	97.9	2.0	-0.7	2.0	1.13	0.143	-0.3	3.0	0.30	0.388
	6096	98.1	1.6	96.9	3.1	98.0	1.9	-1.2	2.7	1.43	0.092	-0.1	1.5	0.18	0.430
RT (ms)	3048	454.3	34.6	464.8	40.7	465.6	37.8	10.5	15.7	2.13	**0.031**	11.3	23.0	1.55	0.078
	4572	451.5	38.1	466.4	37.7	460.0	38.7	14.9	21.9	2.16	**0.030**	8.5	24.9	1.09	0.154
	6096	453.4	39.6	470.1	51.8	444.4	30.5	16.7	29.8	1.77	0.056	-9.1	19.6	1.46	0.089
MT (ms)	3048	119.2	22.7	125.2	21.7	118.0	24.1	6.1	14.2	1.34	0.106	-1.2	13.9	0.26	0.400
	4572	114.5	19.8	121.6	26.2	120.0	23.0	7.1	11.4	1.96	**0.040**	5.5	13.4	1.29	0.165
	6096	119.8	14.2	125.0	18.5	122.0	21.8	5.2	8.8	1.87	**0.047**	2.2	14.8	0.47	0.375
TRT (ms)	3048	579.5	22.4	594.8	28.6	591.5	33.0	15.4	20.5	2.37	**0.021**	12.0	25.2	1.51	0.083
	4572	576.1	30.0	591.7	33.9	586.3	31.0	15.7	22.7	2.18	**0.029**	10.2	24.7	1.30	0.113
	6096	577.1	31.7	603.9	31.8	573.5	27.3	26.8	25.2	3.36	**0.004**	-3.7	28.7	0.41	0.348
Simple Reaction Time Task															
Accuracy (%)	3048	99.9	0.4	98.8	1.6	99.5	0.8	-1.1	1.6	2.17	**0.029**	-0.4	1.0	1.20	0.130
	4572	99.9	0.4	99.4	0.9	99.8	0.4	-0.5	1.0	1.52	0.081	-0.1	0.6	0.33	0.375
	6096	99.7	0.7	97.9	3.2	99.8	0.5	-1.8	3.3	1.69	0.063	0.1	0.9	0.32	0.380
RT (ms)	3048	412.7	33.9	426.4	38.6	411.5	40.6	13.8	24.5	1.78	0.054	-1.2	22.1	0.17	0.436
	4572	405.9	43.2	426.8	41.2	403.5	41.5	20.9	26.2	2.52	**0.016**	-2.4	25.8	0.29	0.390
	6096	419.1	42.8	436.5	43.5	407.0	36.3	17.4	13.0	4.22	**0.001**	-12.1	24.9	1.53	0.080
MT (ms)	3048	109.3	15.5	108.6	19.0	102.0	16.7	-0.7	9.9	0.23	0.411	-7.4	8.0	2.93	0.008‡
	4572	103.8	15.2	106.2	16.7	100.0	11.6	2.4	6.4	1.15	0.139	-3.8	10.8	1.11	0.147
	6096	107.6	11.7	109.3	14.2	102.8	13.5	1.6	7.7	0.67	0.260	-4.8	11.2	1.36	0.103
TRT (ms)	3048	523.5	35.7	538.8	35.5	516.7	41.5	15.3	20.8	2.32	**0.023**	-6.8	22.5	0.95	0.183
	4572	512.3	42.7	535.9	49.1	503.1	44.4	23.6	30.9	2.41	**0.020**	-9.2	28.5	1.03	0.166
	6096	524.2	42.7	548.0	41.9	507.8	41.2	23.8	13.4	5.61	<**0.001**	-16.4	31.8	1.63	0.069

* Table entries are means (averaged over subjects) and associated standard deviations of the compressed data for each phase of each condition.
† Student's one-tailed paired *t*-tests are testing for performance degradation when going from the hyperoxic to hypoxic and from the hyperoxic to normoxic postexposure phases.
‡ Even though the *P*-value is less than 0.05, the difference is in the wrong direction with respect to the one-tailed hypothesis that performance would degrade at the normoxic postexposure phase.

mirror those seen for TRT, suggesting that a large part of the negative impact of hypoxia seen on TRT is due to the impact on RT (i.e., the ability to see, recognize, and begin to react to a stimulus).

No statistical evidence of RT degradation was found when going from the hyperoxic baseline phase to the normoxic recovery postexposure phase for either psychomotor test during any of the three altitude test conditions, indicating that RT, measured 1 h after completion of the runs, had returned to pre-exposure baseline levels.

No significant ANOVA results were detected for MT. As shown in Table II, *t*-tests used to assess MT increases from the hyperoxic to hypoxic phases identified significant differences only for the CRT task during the 4572 and 6096 m (15,000 and 20,000 ft) conditions, although the increases were relatively small (7 and 5 ms, respectively). The CRT mean difference in MT at 3048 m (10,000 ft) was of the same magnitude, but was not significant due to larger variability. These results suggest that hypoxia, at the levels used in this study, had minimal effects on movement time. Finally, there were no statistical indications that MT during the normoxic recovery postexposure phase was elevated relative to the hyperoxic baseline phase for either task under any of the three altitude test conditions.

Although subjective assessment of the means in Table II suggests that accuracy was slightly reduced during the hypoxic phase compared to the baseline hyperoxic phase in both psychomotor tasks and all three altitude conditions, the ANOVA indicated a significant main effect of phase on accuracy in the SRT task only [$MSE = 1.817$, $F(2,18) = 6.09$, $P = 0.010$]. No significant phase by altitude interaction was found for accuracy in either CRT or SRT. The only significant difference identified via *t*-test between accuracy means in the hyperoxic baseline and hypoxic phases was for the SRT task in the 3048 m (10,000 ft) test condition (1.1% change, $P = 0.029$). The accuracy

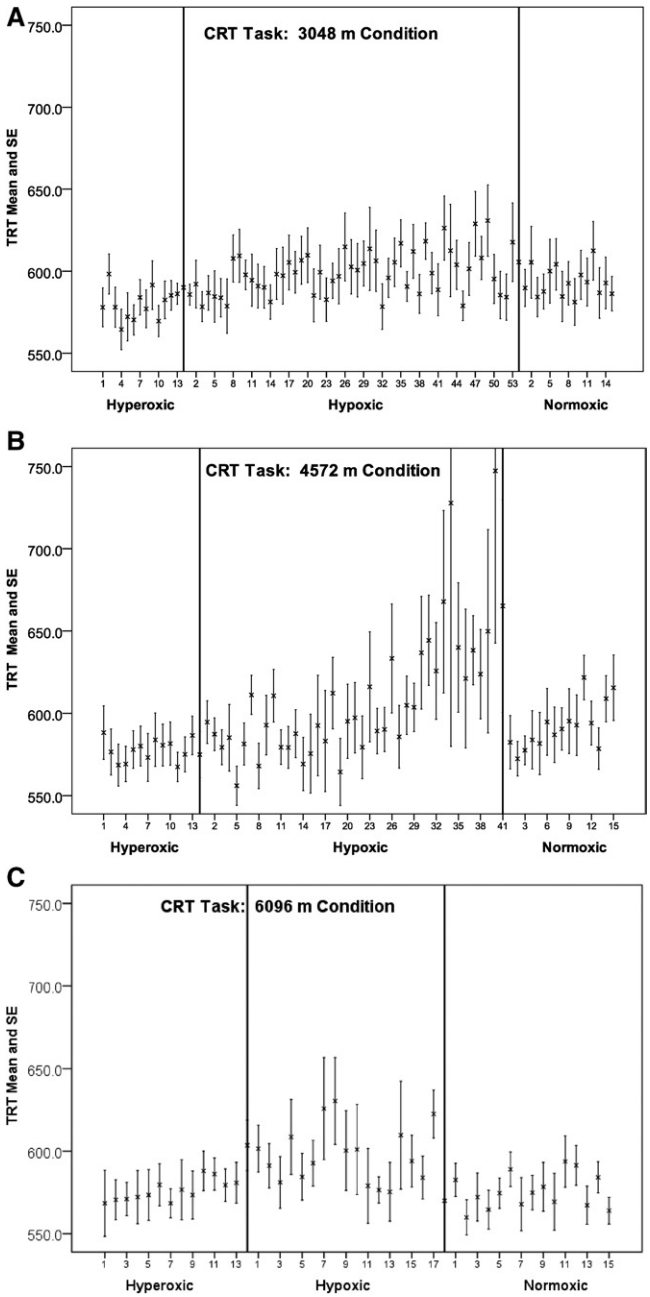

Fig. 1. CRT task: minute-by-minute descriptive statistics (mean ± SE) for TRT at: A) 3048 m; B) 4572 m; and C) 6096 m.

differences in the 6096 m (20,000 ft) test condition were just as great (1.2% and 1.8% for CRT and SRT respectively), but due to greater variability in the data, these differences did not reach significance at the one-tailed 0.05 level. Finally, there was no statistical evidence that accuracy was lower during the normoxic recovery phase than during the hyperoxic baseline phase in any of the altitude conditions for either psychomotor test. Because the accuracy data are not normally distributed, a separate set of nonparametric analyses (Wilcoxon's signed rank tests) was performed to test for hypoxic and recovery effects. The results of these tests were in agreement with the *t*-test results and are not presented here. In summary, although one significant result was found among the accuracy comparisons, it appears that when subjects are required to execute a quick detection response or decide among a limited number of simple responses, hypoxia at the levels used in this study has a lesser negative impact on accuracy than on speed.

Percent change in TRT for hypoxic and normoxic recovery postexposure phases compared to the hyperoxic phase are shown in **Fig. 2**. The percent decrement (increase) in TRT between the statistically significant hypoxia phases ranged from just under 3% for the CRT test at 3048 m (10,000 ft) and 4572 m (15,000 ft) and the SRT test at 3048 m (10,000 ft) to just under 5% for the CRT test at 6096 m (20,000 ft) and for the SRT tests at 4572 and 6096 m (15,000 and 20,000 ft). Although the differences between total RTs in hyperoxic vs. normoxic recovery test phases were not found to be significant in SRT or CRT testing, the normoxic recovery postexposure mean RT for the CRT test at 3048 and 4572 m (10,000 and 15,000 ft) indicated a 2% and 1.7%, respectively, slower TRT. Normoxic recovery postexposure TRTs for the 6096 m (20,000 ft) CRT test and all SRT test altitudes showed nonsignificant percent increases in TRTs.

DISCUSSION

This study found no statistical or subjective evidence, for either the CRT or SRT task, that accuracy or speed of performance was improved under hyperoxia compared to normoxia. This is consistent with earlier findings by Andersson et al.,[1] Dimpel et al.,[7] Goodwin et al.,[9] and also Hemelryck et al.,[10] who observed no significant difference in reaction time at ground level breathing 100% O_2 vs. air, but did report differences in mathematical processing and trail-making tasks. The possibility remains, then, that hyperoxia could induce changes in tasks requiring greater cognitive processing while not affecting performance in simple detection and choice tasks as used in this study that may warrant further investigation.

The second issue is whether performance on these simple psychomotor tasks is negatively impacted in various low to moderate altitude environments, viz. altitudes of 3048, 4572, and 6096 m (10,000, 15,000, and 20,000 ft) in this study. With respect to accuracy (i.e., percent correct responses), only one statistically significant result was found in SRT accuracy which significantly declined during the hypoxic phase of the 3048-m (10,000-ft) exposure by an average of 1.1%. Decrements of equal magnitude were seen for CRT and SRT during the 6096-m (20,000-ft) test exposure but, due to larger variability, were not statistically significant. Given the dearth of significant results, and the magnitude of the observed differences, it is concluded that hypoxia did not have a notable effect on the ability to execute these simple detection and decision responses accurately. In contrast, TRT for both the CRT and SRT tasks did significantly degrade under all three hypoxic conditions. This degradation was not found to carry into the postexposure recovery period.

While the results for RT and MT are of scientific interest, TRT holds the most interest from an operational standpoint. In this study, TRT was significantly higher during the hypoxic

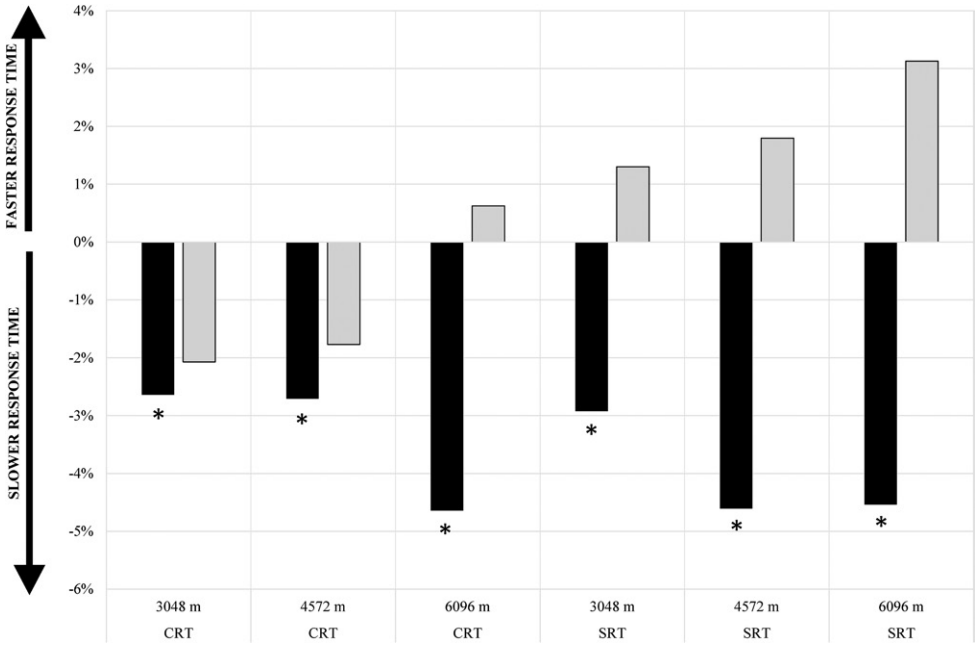

Fig. 2. Percent difference in CRT and SRT test mean TRTs for hypoxic (solid) and normoxic postexposure (gray) conditions compared to hyperoxic (baseline) mean responses at 3048, 4572, and 6096 m. Asterisk (*) indicates significant difference ($P \leq 0.05$).

phase than during the hyperoxic baseline phase for both the CRT and SRT tasks under all three altitude conditions. An inspection of the minute-by-minute data showed that, for the two lower altitudes, TRT at the beginning of the hypoxic phase of testing was about equal to or slightly higher than average TRT during the corresponding hyperoxic baseline phase, but significantly increased over the duration of the hypoxic phase. The rate of increase appeared to be greater at 4572 m (15,000 ft) than at 3048 m (10,000 ft), which is in accordance with previous observations.[16,19,22] For the 6096 m (20,000 ft) altitude, the negative effect of hypoxia was more immediate and of greater magnitude than at the lower altitudes, but the degree of degradation did not increase over the duration of the exposure. However, it is reasonable to suspect that such a trend would have been found if the time of exposure had been of the same length as for the lower altitudes.

The results for RT resembled those seen for TRT, whereas fewer effects (and smaller in magnitude) were seen for MT. This is commensurate with other studies involving tasks of similar psychomotor demand.[12] Thus, it is concluded that the degradation seen for TRT is due primarily to the effect of hypoxia on the time it takes to detect the stimulus, decide (in CRT) among a limited menu of responses, and begin to execute the response. This suggests that the most detrimental effects of hypoxia are to be found within the perceptual and executive components of the central nervous system, occurring prior to the stimulation of motor pathways, a finding that substantiates reported differences in nervous tissue hypoxia tolerances.[23]

Statistical significance notwithstanding, a relevant concern is the relative impact that these decrements may have on overall flight performance. As the results of Fig. 2 demonstrate, the actual TRT while hypoxic was less than 5% slower than that seen during hyperoxia. Below 4572 m (15,000 ft), a 3% decrement in RT on flight performance is likely to have little impact during normal flight operations, but could become significant in an emergency requiring motor movement in order to avoid a midair collision or decision and action to initiate ejection from the aircraft. For example, a 15-ms reaction time delay (which corresponds to the approximate delay seen between the hypoxic and hyperoxic phases in SRT and CRT tests at and below 4572 m) for an aircraft traveling at 740.8 km/h (400 knots, 460.3 mph) equates to an additional 3 m (9.8 ft) traveled.

With respect to earlier reports of cognitive deficits from hypoxia that persist following return to normoxic conditions, this study found no significant difference between the 60-min normoxic recovery postexposure and hyperoxic baseline conditions for either the SRT or CRT psychomotor tests at any of the altitudes evaluated. Indeed, in some cases it appeared that performance might have improved slightly during the normoxic recovery compared to baseline, potentially due to the subject having had a one hour rest prior to retesting. This finding may be compared to the response-time findings reported by Phillips et al.[18] and findings from a synthetic workstation study[3] in which slight auditory deficits were observed to linger during hypoxia recovery. It is important to point out, however, that since the intention of this research effort was to evaluate the null hypothesis that performance was not degraded 60 min after exposure compared to baseline, and since the sample size was relatively small (10 subjects), one cannot conclude confidently that no differences existed. Rather, it can only be stated that, within the framework and limitations of this study, there was no statistical evidence that psychomotor performance was degraded 1 h after completion of the exposures. Furthermore, the disparity between the nonsymptomatic recovery results reported here and the posthypoxia decrements reported in other studies[3,18] might have stemmed from differences in test design and from the comparatively lesser severity of the hypoxia induced in the current study. In the Phillips et al. study, hypoxia was induced using ROBD, subjects breathed O_2 at a partial pressure equivalent to 5486 m (18,000 ft), and subjects' participation was terminated upon reaching time at altitude or reaching 50% blood O_2 saturation levels. In contrast, subjects in the current study were simply instructed to terminate once they were able to recognize at least two of their subjective hypoxia symptoms, as per their altitude chamber training. Consideration of the current findings in conjunction with earlier studies leaves open the complex

question of whether cognitive impairment persists during recovery: whereas certain studies have reported persistent deficits in response time, early perceptual storage or auditory processing, lingering impairment was not observed using the SRT and CRT psychomotor tasks here.

In this context, it is important to remain aware of the potential difference between hypobaric (altitude- or chamber-induced) hypoxia and normobaric (ROBD-induced) hypoxia. In particular, hypobaric hypoxia may induce hyperventilation and thereby foster greater carbon dioxide exhalation and subsequent hypocapnia, which could alter or modulate the emergence of hypoxia-like symptoms.

While the SRT and CRT tasks were selected in this study to represent psychomotor aspects of certain aircrew tasks, no test or battery can represent the totality or even the majority of cognitive performance. Piloting, navigation, communication and problem-solving depend upon a complex arsenal of sensory, perceptual, executive and motor components, all of which may be affected differently by hypoxia, hypobaria, and hypocapnia (and assessed via different cognitive instruments). For this reason, it is hazardous to cite findings obtained using one or two psychomotor tasks as representative of "cognition."

The results of this study validate the capacity to use reaction time tasks to detect quantifiable decrements in psychomotor performance resulting from hypoxia at altitudes as low as 3048 m (10,000 ft). In addition, the study indicates that hyperoxia has no positive effect on accuracy or reaction time when performing tasks equivalent to the simple tests employed in this study.

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