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16. Abstract

Tolerances of human subjects for orthostasis and physical work were determined at a simulated altitude of 3,048 m. Orthostasis was induced with a lower body negative pressure (LBNP) device and physical work was done on a pedal ergometer. Altitude was simulated in a hypobaric chamber. Tests were carried out under two experimental conditions: (i) after subjects drank an alcoholic beverage, or (ii) after subjects drank a placebo beverage (no alcohol). The alcoholic beverage produced blood alcohol concentrations (BAC's) of about 90 mg/100 ml of blood (90 mg percent). At altitude, arterial oxyhemoglobin saturation (HbO_2) remained adequately compensated but was lower after alcohol than after placebo intake. Arithmetic and eye/hand coordination performances were both significantly decreased after alcohol. Ergometry, after alcohol, was well tolerated despite some decreased cardiorespiratory efficiency. The LBNP applied around peak BAC at altitude was tolerated without subjectively adverse symptoms despite significant decreases in several cardiovascular parameters. Cardiovascular adequacy along with maintained plasma volume around peak BAC appeared to be temporarily protective against orthostatic incapacitation during LBNP. Reversal of this temporary orthostatic protection during BAC recession is possible.

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ALCOHOL-INDUCED PHYSIOLOGICAL DISPLACEMENTS AND THEIR EFFECTS ON FLIGHT-RELATED FUNCTIONS

INTRODUCTION

Billings et al. (1) amply demonstrated that alcohol degrades a pilot's ability to safely operate an aircraft. They studied the effects of various blood alcohol concentrations (BAC's) on the capability of instrument-rated pilots to execute instrument landing approaches in light aircraft. They found that significant increases in the number and potential seriousness of procedural errors occurred at BAC's as low as 40 mg percent and that these errors increased as the BAC was increased up to 120 mg percent. BAC's lower than 40 mg percent were not evaluated (1). Previous Civil Aeromedical Institute (CAMI) studies have assessed some of the effects of alcohol on altitude tolerance (10,11) and on psychomotor performance (4).

Ryan et al. (17) recently reported that the incidence of fatal accidents associated with alcohol decreased gradually from 43 percent in 1963 to approximately 16 percent for the 1969-1976 period. Unpublished data from the Aviation Toxicology Laboratory of CAMI revealed that an average of 8.5 percent of fatal aviation accidents were associated with alcohol for the 1968-1979 period. The difference between this 8.5 percent incidence value and the 16 percent incidence value reported by Ryan et al. is due mainly to the fact that the minimum BAC associated with a fatal accident was defined as 40 mg percent in the CAMI study and 15 mg percent in the study by Ryan et al. CAMI toxicological data further show that the fatal accident rate associated with BAC's in excess of 40 mg percent in 1980 was 6.5 percent; in Fiscal Year 1981 (October 1, 1980, to September 30, 1981) an all-time low incidence of 5.1 percent was reached.

Despite a Federal Aviation Regulation (Part 91.11) (8) which prohibits the operation of civil aircraft while the pilot is under the influence of alcohol, or within 8 h after the consumption of any alcohol beverage, a recent survey (5) reported that about 30 percent of general aviation (GA) pilots considered flying after moderate social drinking to be safe behavior.

Many of the physiological effects of alcohol are well known. The thermogenic effect of moderate alcohol intake produces increased heat loss by means of peripheral cutaneous vasodilation (21). The resulting decrease in central blood volume is compensated by vasoconstriction of central arteries and by an increased heart rate (HR) (21). The diuretic effect of alcohol occurs only while the BAC is rising and is ascribed to an inhibitory effect of alcohol on secretion of antidiuretic hormone (21). Increased respiration and perspiration resulting from thermogenesis also increase loss of body water (21).

The purpose of this study was to determine the presence and degree of alcohol-related physiological decrements that could compromise safety of flight. Specifically, our study was directed at assessing the effect of alcohol on an individual's ability to: (i) tolerate altitude as measured by maintenance of adequate oxyhemoglobin saturation (HbO_2) and psychomotor performance; (ii) maintain adequate

cardiovascular function and useful consciousness during applied lower body negative pressure (LBNP); and (iii) perform moderate physical work efficiently as measured by quantitative shifts in cardiorespiratory functions during pedal ergometry.

METHODS

Because LBNP testing had not been conducted at altitude in humans with substantial BAC's, a group of six subjects was initially tested (Phase I) at ground level (GL) (388 m mean sea level, MSL). There were no untoward occurrences at GL in Phase I; therefore, a second group of 10 subjects (Phase II) was tested at an altitude of 3,048 m MSL.

Subjects. The participants were paid, healthy male volunteers (25-40 years old) who admitted previous experience in moderate consumption of liquor. Each subject had to pass a third-class medical examination equivalent to that required of a private pilot. The qualified subjects signed a standard consent form after a thorough briefing. Each subject experienced a complete practice session of the research protocol. For Phase I, this included a 1-h training session in our hypobaric chamber at GL. The subject was trained to properly blow an expired air sample into the apparatus ("Intoxilyzer") which measured the BAC. He took two timed arithmetic tests (psychomotor performance), he was subjected to LBNP at -40 torr differential pressure for 2 min as a test of orthostatic tolerance, and he carried out pedal ergometry at a load of 50 watts (W) for 4 min. The subject was disqualified from further participation if consciousness was not maintained during LBNP, if his HR exceeded 150 beats per min (bpm) during pedal ergometry, or if his monitored single-lead electrocardiogram (ECG) manifested evidence of ischemia or arrhythmia at any time during this session.

Each Phase II subject experienced a 1-h training session in our hypobaric chamber at 3,048 m MSL during which he practiced single-breath "Intoxilyzer" measurements of BAC, took two timed arithmetic tests, and underwent 2 min of LBNP. After chamber pressure was returned to GL, the subject practiced 4 min of pedal ergometry at 50 W. Besides the disqualification criteria of Phase I, the subject was disqualified if his HbO_2 fell below 80 percent during altitude exposure (16). The subjects' age, height, and weight data are summarized in Table I.

Protocols and Variables. Each subject participated in one experiment per week for 2 consecutive weeks. The protocols for Phases I and II are outlined in Table II. Each subject arrived at 1115 when his temperature and health status were checked. At 1130 he was fed a ham and swiss cheese sandwich and a cup of chicken soup in order to avoid alcohol intake on an empty stomach and to avoid any possible effect of fasting through the midday meal period. After 30 min of resting digestion, the subject took two timed arithmetic tests. Each test consisted of 20 pages of simple addition and subtraction problems (20 per page) to be answered true or false. The subject was instructed to make a checkmark in the correct answer block provided for each problem. The score equaled the number of incorrect answers per min of total test time. As a separate test of eye/hand coordination, the subject was asked to make the checkmark within the appropriate answer block without the checkmark touching any of the block s

TABLE I. Age, Height, and Weight Data

		Age (yr)	Height (cm)	Weight (kg	No. of Subjects
PI	$\bar{X}$	29.3	178.6	92.0	6
	SE	2.0	2.2	7.4	
PII	$\bar{X}$	28.1	178.5	74.0	10
	SE	0.8	2.9	3.1	

$\bar{X}$ = mean SE = Standard error of the mean

PI = Phase I experiments at GL only

PII = Phase II experiments at 3,048 m MSL altitude

TABLE II. Experimental Protocols

Phase I Experiments		Phase II Experiments	
Time	Activity	Time	Activity
1115	Subject Appears	1115	Subject Appears
	Health Questionnaire		Health Questionnaire
1130-1145	Light Lunch	1130-1145	Light Lunch
1145-1215	Seated Rest	1145-1215	Seated Rest
1220-1240	1st Arithmetic Test	1220-1240	1st Arithmetic Test
1250-1310	2nd Arithmetic Test	1250-1310	2nd Arithmetic Test
1315-1345	Sensor Placements	1315-1345	Sensor Placements
1345-1358	Physiological Measurements	1345-1358	Physiological Measurements
1358-1400	1st BAC Measurement	1358-1400	1st BAC Measurement
1400-1430	Alcohol or Placebo Intake	1400-1430	Alcohol or Placebo Intake
1438-1440	2nd BAC Measurement	1430-1440	Altitude Ascent
1445-1500	3rd Arithmetic Test	1443-1445	2nd BAC Measurement
1500-1508	Physiological Measurements	1445-1500	3rd Arithmetic Test
1508-1510	3rd BAC Measurement	1500-1502	3rd BAC Measurement
1515-1530	4th Arithmetic Test	1502-1505	Physiological Measurements
1530-1535	Adjust LBNP Waist Seal	1505-1520	4th Arithmetic Test
1535-1537	4th BAC Measurement	1520-1523	Adjust LBNP Waist Seal
1537-1547	LBNP Procedure	1523-1525	4th BAC Measurement
1555-1557	5th BAC Measurement	1525-1535	LBNP Procedure
1600-1608	Pedal Ergometry	1535-1537	5th BAC Measurement
1608-1610	6th BAC Measurement	1537-1547	Altitude Descent
1610-1625	Sensor Removal	1547-1555	Pedal Ergometry
		1558-1600	6th BAC Measurement
		1600-1615	Sensor Removal

boundary lines. Each block measured 9 mm by 20 mm. The score for this test equaled the number of boundary violations per min of total test time. This test evolved from Phase I observations that the intoxicated subjects experienced substantial difficulty with writing during the arithmetic tests. This test was subsequently added to the Phase II experiments. Simple arithmetic has previously been shown to be adversely affected by peak BAC's as low as 35 mg percent (7). Our experience has shown that the asymptotic portion of the learning curve for this combined psychomotor test is attained by the fourth test. Any residual learning differences were accounted for in the randomization of experimental order. The effects of alcohol versus placebo on the arithmetic and eye/hand coordination scores were based only on the two tests taken after intake of the alcohol or the placebo.

After completing the second arithmetic test, the subject donned a surgical scrub suit and was taken to the hypobaric chamber where he was instrumented for subsequent testing. He was then seated in our LBNP box and loosely sealed in it from the waist down. The LBNP box and its built-in pedal ergometer have been described elsewhere (14). After 10 min of resting physiological measurements, the subject provided the first pair of BAC measurements in order to confirm a starting BAC of zero.

Alcohol intake started at 1400. The alcohol load was based on 2 ml of 100 proof bonded bourbon per kg of body weight in a "standard" man 70 kg in weight, 170.2 cm in height and 1.81 m^2 of body surface area (BSA). The total load for such a man would be 140 ml (about 4.66 oz) of 100 proof bourbon. The load for each subject was proportional to his BSA as calculated from his measured height and weight (6). One part bourbon per two parts of Coca Cola were mixed, divided equally into three drinks and drunk at an even rate over a period of 30 min. In the placebo experiments, an equal volume of tap water replaced the bourbon in the mixed drinks. All drinks were served ice cold.

Ascent to chamber altitude of 3,048 m MSL started immediately after completion of alcohol or placebo intake. At altitude, after rinsing orally with water, the subject provided the second pair of BAC measurements. The third arithmetic test, the third pair of BAC measurements, the fourth arithmetic test, adjustment of the LBNP waist seal and the fourth pair of BAC measurements ensued up to 1525. At this time, the subject had been at altitude for 45 min; 55 min had elapsed since the end of alcohol or placebo intake.

Next, the LBNP procedure was performed. This consisted of 8 min of control physiological measurements after which the subject underwent an LBNP of -40 torr for 2 min. A fifth pair of BAC measurements ensued. The chamber was then returned to GL in 10 min. The subject next underwent pedal ergometry of 30 W for 2 min and 50 W for 6 min. After the subject rested for 5 min, the sixth pair of BAC measurements was obtained, all sensors were removed, and the subject changed to his street clothes. In the alcohol experiment, the subject's BAC was monitored until it approximated 40 mg percent, at which time a researcher personally transported the subject to his home. The consent form included the subject's agreement to abstain from operating any vehicle or hazardous machinery until the next day.

The subject returned one week later for the second experiment. To compensate for any potential effects of experimental order, half of the subjects drank alcohol in the first experimental session and the remaining half drank alcohol in the second experimental session. The data were pooled and, using Student's paired t test (19), were statistically compared on the basis of alcohol versus placebo conditions. Statistical significance was based on a probability value of $p \leq 0.05$ (19).

Specifically measured variables were: HR using a single-lead ECG; blood pressure (BP) using auscultative manometry; HbO_2 using an ear oximeter (18); and temporal artery blood flow velocity (TAFV) using a directional Doppler device (13). Besides HR and BP, pulmonary ventilation ($\dot{V}_E$), respiratory frequency (f), tidal volume (V_T), and oxygen uptake ($\dot{V}O_2$) were measured during pedal ergometry. Gas volumes were expressed as volume per kg of body weight. The CM_5 lead (2) was used to monitor ECG function. This signal was fed to an oscilloscope for visual ECG monitoring, a cardiotachometer for continuous indication of HR and a standard ECG recorder for periodic sampling. Also monitored were the digital meter of the HbO_2 for any indication of hypoxemia and the pulsatile meter signal of the TAFV for any flow-reversal indication of approaching syncope (13). At altitude, criteria for immediate termination of any experiment consisted of strong subjective symptoms of impending syncope (lightheadedness, nausea, and grayout, tunneling, or blackout of vision) accompanied by hypotension and bradycardia (12), ECG evidence of ischemia or arrhythmia, TAFV approximating zero, and falling values of HbO_2 below 80 percent.

During each altitude exposure one researcher remained in the hypobaric chamber with the subject. Emergency medical equipment, 100 percent oxygen and a staff physician were always available on a standby basis. Because each subject breathed through a valve mouthpiece during pedal ergometry three simple hand signals were taught to each one to communicate that "everything is OK," "subjective distress is present," and "stop the test."

Temperature and relative humidity in the hypobaric chamber were controlled within the ranges of 21.4°-24.4°C and 19.0-26.0 percent, respectively.

RESULTS

Figure 1. presents the mean BAC values measured during Phases I and II experiments. In both phases the mean BAC peaked slightly above 90 mg percent and did so within 30-60 min after completion of alcohol intake. The mean BAC peak occurred earlier in the Phase II altitude experiments than in the Phase I GL experiments. These data are consistent with those of two previous CAMI studies (10,11) which used the same protocol for alcohol intake. The BAC which existed at the time of each testing procedure was obtained by interpolation from each subject's plotted curve of BAC versus time.

The data from all testing procedures are summarized in Tables III-VII. Starred mean values in these tables indicate statistically significant differences between alcohol and placebo conditions.

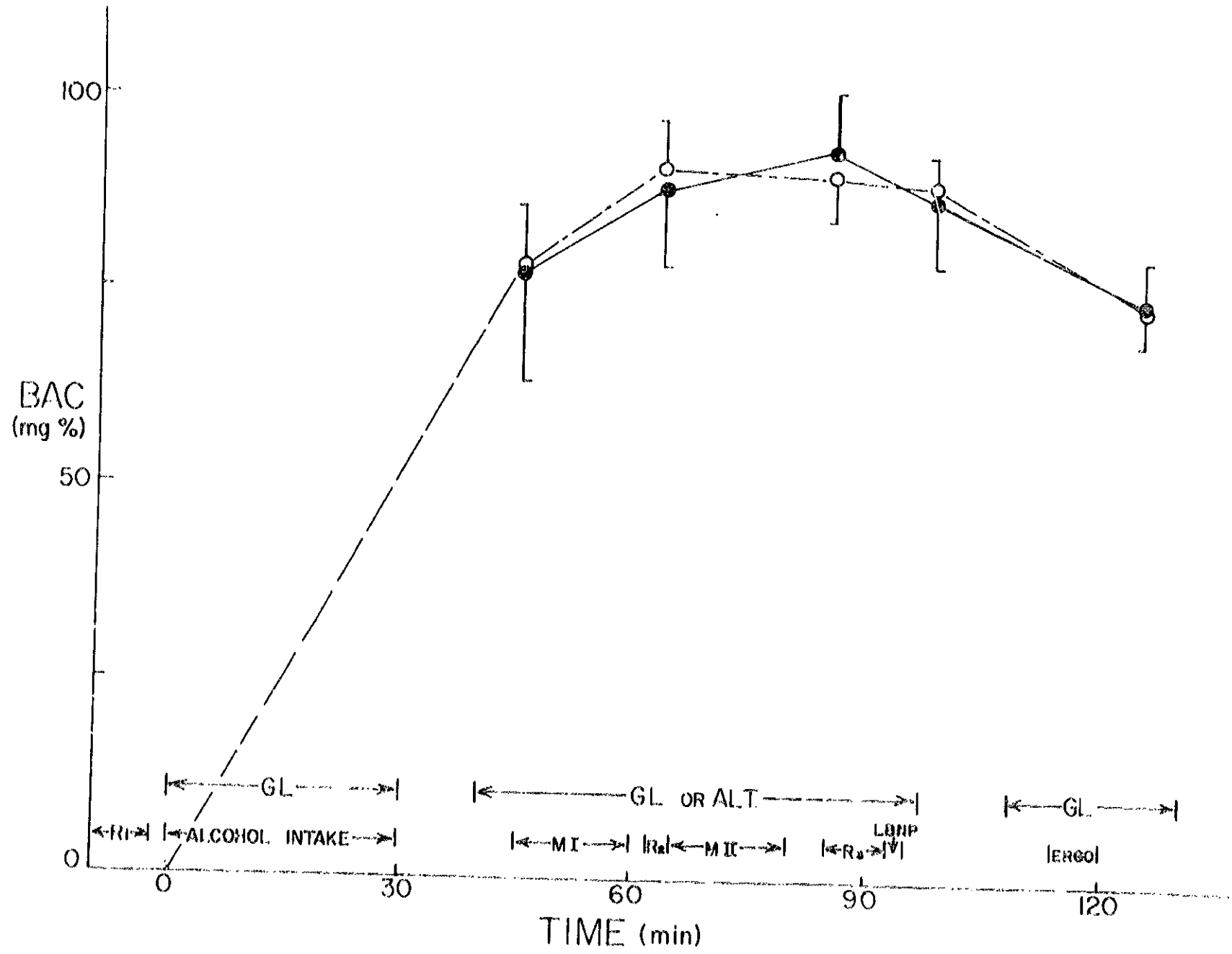
100
50
0
BAC
(mg %)
GL
ALCOHOL INTAKE
Ri
GL OR ALT
MI
Rd
MII
Ra
LBNP
V
GL
ERGO
0
30
60
90
120
TIME (min)

Figure 1. Blood Alcohol Concentration Versus Time.

BAC = Blood alcohol concentration in mg percent
GL = Ground level altitude of 388 m MSL
Alt. = Hypobaric chamber altitude of 3,048 m MSL
● = Mean BAC values for Phase I GL experiments
O = Mean BAC values for Phase II altitude experiments
R_1, R_2, R_3 = Periods of resting physiological measurements
ERGO = Period of pedal ergometry at 50 W
MI, MII = Two arithmetic and eye/hand coordination tests
LBNP = Lower body negative pressure of -40 torr differential pressure for 2 min

In Phase I GL experiments alcohol (as compared to placebo) was associated with statistically significant decrements in arithmetic performance (Table VII) and increments in HR during the concluding ergometry test (Table VI).

In Phase II altitude experiments alcohol (as compared to placebo) was associated with statistically significant decreases in diastolic BP (DBP, Table III), mean arterial pressure (AP, Table IV), and HbO_2 (Table V); HR increased (Table V). During LBNP at altitude alcohol (as compared to placebo) was associated with statistically significant decreases in systolic BP (SBP, Table III), pulse pressure (PP, Table IV), and TAFV (Table V); HbO_2 increased (Table V). In arithmetic performance and eye/hand coordination at altitude, alcohol (as compared to placebo) was associated with statistically significant decrements (Table VII). During postaltitude ergometry alcohol (as compared to placebo) was associated with a statistically significant increase in HR (Table VI).

DISCUSSION

The statistically significant displacements associated with alcohol in Phase II experiments were of greater magnitude than were the corresponding statistically significant displacements that occurred in Phase II placebo experiments. Alcohol caused significant decrements in both physiological and psychomotor functions without causing the subject to lose useful consciousness.

Altitude Tolerance. The supply of cerebral oxygen needed to maintain useful consciousness depends on both the amount of oxygen in the arterial blood and the rate of blood flow through the brain. The thermogenic effect of moderate alcohol intake and its resultant compensating heat loss via cutaneous vasodilation are well known (21). The resulting decrease in central blood volume is compensated by vasoconstriction of central arteries and by an increase in HR (21). When alcohol intake is combined with altitude exposure (3,048 m) some of the vasoconstrictor compensation is lost (DBP and AP data in Tables III and IV) but cerebral circulatory adequacy is maintained by increased HR (Table V).

The mean decrease in HbO_2 from GL to a 3,048 m altitude was significantly greater in the alcohol than in the corresponding placebo experiments (Table V). This finding is consistent with that of a previous study (9). The lowest mean HbO_2 value (Table V) at altitude in the alcohol condition was 86.3 percent. Although lower than the corresponding placebo value at altitude, the mean value of HbO_2 approaches the lower edge of the adequately compensated HbO_2 range ($\geq$ 85 percent) for a 3,048 m altitude (16). Because a decreased but minimally adequate mean HbO_2 existed at altitude in our study, the statistically significant decrements in both arithmetic and eye/hand coordination performances (Table VII) associated with the alcohol condition were probably related to a histotoxic rather than hypoxic effect of alcohol (16). This corroborates the statistically significant decrements in arithmetic performance which occurred in Phase I GL experiments in which cerebral blood flow (TAFV data) and HbO_2 (normoxia) were both fully adequate. These findings are consistent with those of another study (4) conducted under similar altitude and peak BAC conditions. Adjunct data analysis revealed that individual decrements in psychomotor performance were greater when the BAC was rising than when it was subsequently falling. This general pattern is also consistent with previous observations (10,11). Therefore, although alcohol quantitatively shifted some functions which are involved in altitude tolerance, a substantial compromise of altitude tolerance was not demonstrated.

TABLE III. Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP)

		SBP (mmHg)				SBP (% of Control)	
		R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI Alcohol	$\bar{X}$	123.4	123.0	121.9	115.7	98.6	94.8
	SE	2.9	4.1	4.7	5.3	2.1	1.5
PI Placebo	$\bar{X}$	121.3	125.3	120.8	115.0	99.8	95.3
	SE	4.1	4.1	2.8	2.4	1.7	1.2
PII Alcohol	$\bar{X}$	111.4	112.4	112.8	99.8	101.2	88.1 *
	SE	3.8	4.2	4.4	6.0	1.4	3.0
PII Placebo	$\bar{X}$	111.8	115.7	116.9	112.9	104.6	96.2
	SE	3.3	3.7	3.8	5.2	1.7	1.6

		DBP (mmHg)				DBP (% of Control)	
		R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI Alcohol	$\bar{X}$	61.8	63.9	61.9	65.2	101.1	105.8
	SE	4.2	3.5	3.5	2.8	5.0	2.4
PI Placebo	$\bar{X}$	61.5	61.3	64.3	66.0	104.8	102.0
	SE	3.8	4.2	4.0	5.7	3.6	4.3
PII Alcohol	$\bar{X}$	62.0	63.8	60.6	59.4	97.0 *	99.6
	SE	2.2	3.4	2.9	2.5	3.8	3.2
PII Placebo	$\bar{X}$	59.3	63.9	65.8	63.6	111.0	97.0
	SE	2.2	3.2	2.6	2.4	2.4	2.1

$\bar{X}$ = mean SE = Standard error of the mean PI = Phase I experiments at GL only
R_1, R_2, R_3 = Periods of resting physiological measurements PII = Phase II experiments at 3,048 m MSL altitude
LBNP = Lower body negative pressure of -40 torr for 2 min
* = Statistical significance at $p \leq 0.05$

TABLE IV. Pulse Pressure (PP) and Mean Arterial Pressure (AP)

		PP (mmHg)				PP (% of Control)	
		R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI Alcohol	$\bar{X}$	61.6	64.1	60.0	50.5	97.2	84.5
	SE	7.0	7.4	7.5	6.5	4.5	4.0
PI Placebo	$\bar{X}$	59.8	64.0	56.5	49.0	95.8	86.3
	SE	7.7	8.1	6.3	6.9	2.7	4.6
PII Alcohol	$\bar{X}$	49.4	48.6	52.8	40.4	106.7	76.9 *
	SE	3.0	3.1	3.6	4.3	3.1	6.9
PII Placebo	$\bar{X}$	52.5	51.8	51.2	49.3	97.0	94.9
	SE	2.7	3.9	3.7	5.0	4.0	3.5

		AP (mmHg)				AP (% of Control)	
		R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI Alcohol	$\bar{X}$	82.3	85.3	81.9	82.0	99.7	100.1
	SE	1.9	1.3	1.7	2.3	3.1	1.1
PI Placebo	$\bar{X}$	81.5	82.6	83.1	87.0	102.1	104.1
	SE	1.5	1.6	2.0	7.4	2.5	6.6
PII Alcohol	$\bar{X}$	78.5	80.0	77.7	72.9	98.9 *	93.9
	SE	2.5	3.4	3.1	3.4	2.5	2.4
PII Placebo	$\bar{X}$	76.8	82.3	82.8	79.9	107.9	96.6
	SE	2.3	3.4	2.5	2.7	1.7	1.6

X = mean SE = Standard error of the mean
AP = Mean arterial pressure, calculated as the value of DBP + 1/3 PP
PI = Phase I experiments at GL only PII = Phase II experiments at 3,048 m MSL altitude
R_1, R_2, R_3 = Periods of resting physiological measurements
LBNP = Lower body negative pressure of -40 torr for 2 min
* = Statistical significance at $p \leq 0.05$

TABLE V. Oxyhemoglobin Saturation (HbO_2), Heart Rate (HR), and Temporal Artery Blood Flow Velocity (TAFV)

			HbO_2 (%)				HbO_2 (% of Control)	
			R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PII	Alcohol	$\bar{X}$	95.2	86.3*	87.7*	89.0	92.1 *	101.5 *
		SE	0.3	0.8	0.5	0.7	0.6	0.2
PII	Placebo	$\bar{X}$	94.9	89.1	89.8	90.1	94.6	100.4
		SE	0.6	0.5	0.6	0.5	0.6	0.4
			HR (bpm)				HR (% of Control)	
			R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI	Alcohol	$\bar{X}$	64.8	65.6	67.2	75.2	105.0	111.1
		SE	5.8	4.2	5.6	7.9	7.4	3.0
PI	Placebo	$\bar{X}$	59.3	55.8	58.5	63.8	98.9	109.6
		SE	4.2	4.3	4.3	4.4	2.7	4.7
PII	Alcohol	$\bar{X}$	63.4	71.4	75.1	85.2	119.3 *	113.1
		SE	2.9	3.1	3.0	4.5	4.0	2.3
PII	Placebo	$\bar{X}$	63.2	65.8	67.2	73.2	106.5	109.0
		SE	2.9	2.7	3.0	3.8	1.8	2.9
			TAFV (cm/s)				TAFV (% of Control)	
			R_1	R_2	R_3	LBNP	R_3/R_1 X100	LBNP/R_3 X100
PI	Alcohol	$\bar{X}$	3.5	3.9	3.5	2.5	97.5	69.4 *
		SE	0.7	0.8	0.7	0.6	9.8	6.0
PI	Placebo	$\bar{X}$	4.8	4.6	4.7	4.2	100.7	90.6
		SE	1.0	1.1	1.1	1.0	15.0	0.8
PII	Alcohol	$\bar{X}$	4.1	3.6	3.9	2.7	95.0	67.8 *
		SE	0.5	0.7	0.7	0.6	11.8	5.5
PII	Placebo	$\bar{X}$	4.1	3.9	3.8	3.3	88.7	83.6
		SE	0.7	0.8	0.8	0.7	7.0	5.5

$\bar{X}$ = mean SE = Standard error of the mean
HR = Heart rate in beats per min (bpm)
PI = Phase I experiments at GL only
PII = Phase II experiments at 3,048 m MSL altitude
R_1, R_2, R_3 = Periods of resting physiological measurements
LBNP = Lower body negative pressure of -40 torr for 2 min
* = Statistical significance at $p \leq 0.05$

TABLE VI. Postaltitude Ergometry (50 W Load)

		SBP (mmHg)	DBP (mmHg)	PP (mmHg)	AP (mmHg)	HR (bpm)
PI Alcohol	$\bar{X}$	156.8	64.2	92.6	95.0	104.2 *
	SE	5.0	3.2	6.9	2.2	3.1
PI Placebo	$\bar{X}$	147.4	61.4	85.8	90.0	93.9
	SE	6.9	4.6	8.9	3.4	2.5
PII Alcohol	$\bar{X}$	146.4	64.4	82.0	91.7	105.6 *
	SE	7.8	3.4	6.1	4.4	2.4
PII Placebo	$\bar{X}$	139.4	64.7	74.7	89.6	97.3
	SE	6.2	3.0	4.5	3.8	1.5

		$\dot{V}O_2$/kg (ml/min/kg)	TAFV (cm/s)	$\dot{V}_E$/kg (ml/min/kg)	f (rpm)	V_T/kg (ml/kg)
P1 Alcohol	$\bar{X}$	10.2	3.8	266.0	23.8	11.2
	SE	0.7	0.7	33.9	1.9	1.1
P1 Placebo	$\bar{X}$	10.0	4.5	250.2	21.4	12.0
	SE	0.8	0.9	25.6	1.4	1.5
PII Alcohol	$\bar{X}$	12.0	3.7	294.3	20.4	15.4
	SE	0.3	0.5	13.1	1.5	1.8
PII Placebo	$\bar{X}$	11.6	4.3	290.8	18.4	16.5
	SE	0.3	0.7	13.1	1.1	1.4

$\bar{X}$ = mean SE = Standard error of the mean SBP = Systolic blood pressure
DBP = Diastolic blood pressure PP = Pulse pressure AP = Mean arterial pressure
HR = Heart rate in beats per min (bpm) TAFV = Temporal artery blood flow velocity
$\dot{V}O_2$/kg = Oxygen uptake per kg of body weight PI = Phase I experiments at GL only
$\dot{V}E$/kg = Pulmonary ventilation per kg of body weight PII = Phase II experiments at
f = Respiratory frequency in respirations per min (rpm) 3,048 m MSL altitude
LBNP = Lower body negative pressure of -40 torr for 2 min
* = Statistical significance at $p \leq 0.05$ V_T/kg = Tidal volume per kg of body weight

TABLE VII. Arithmetic and Eye/Hand Coordination Testing

		Arithmetic Score I			Arithmetic Score II		
		Alc.	Plac.	Alc./Plac. X100	Alc.	Plac.	Alc./Plac. X100
PI	$\bar{X}$	1.30	0.57	215.0 *	1.31	0.51	254.3 *
	SE	0.47	0.18	35.2	0.41	0.10	56.5
PII	$\bar{X}$	1.20	0.62	233.2 *	0.95	0.66	201.5
	SE	0.53	0.24	45.9	0.48	0.32	71.4

		Coordination Score I			Coordination Score II		
		Alc.	Plac.	Alc./Plac. X100	Alc.	Plac.	Alc./Plac. X100
PII	$\bar{X}$	0.98	0.22	730.5 *	1.25	0.18	858.8 *
	SE	0.24	0.06	184.7	0.34	0.05	191.2

$\bar{X}$ = mean SE = Standard error of the mean
Alc. = Alcohol condition
Plac. = Placebo condition
PI = Phase I experiments at GL only
PII = Phase II experiments at 3,048 m altitude

Arithmetic Score = Number of incorrect answers per minute of time for test completion
Coordination Socre = Number of block-boundary violations per minute of time for test completion

Mean BAC in PI during arithmetic test I = 83.3 mg percent
Mean BAC in PI during arithmetic test II = 90.9 mg percent
Mean BAC in PII during arithmetic test I and Coordination Score I = 85.0 mg percent
Mean BAC in PII during arithmetic test II and Coordination Test II = 90.6 mg percent

* = Statistical significance at $p \leq 0.05$

Physical Work Tolerance. The interpolated mean BAC during pedal ergometry was about 80 mg percent. As shown in Table VI, all the variables in the alcohol condition of Phases I and II experiments changed in the direction of lessened ergometric efficiency as compared to corresponding placebo values; however, only the increase in HR was statistically significant. Our findings are consistent with previous findings (3) in which moderate and maximum pedal ergometry loads were used; m an peak BAC's of 125 and 156 mg percent were attained, ergometric inefficiency was significantly present at moderate workloads, and at maximum workloads the $\dot{V}O_2$ was only slightly depressed but $\dot{V}_E$ and respiratory quotient were significantly depressed. The alcohol-related inefficiency at moderate workloads was probably related to the circulatory adjustments to alcohol thermogenesis (21). Therefore, BAC's of about 80 mg percent do not significantly alter the body's overall tolerance to a moderate physical workload.

Orthostatic Tolerance. Mean BAC during the LBNP test was about 90 mg percent. As shown in Tables III-V, 2 min of LBNP (Phase I placebo experiments at GL) produced statistically significant displacements of several physiological variables. Alcohol was associated with greater displacements than those of the corresponding placebo experiments at GL or at altitude. Although greater displacements by alcohol were in the direction of decreasing orthostatic tolerance, only those of SBP, PP, and HbO_2 in the altitude experiments and TAFV in both GL and altitude experiments were statistically significant. In the GL and altitude placebo experiments, half of the subjects reported mild to moderate transient lightheadedness and visual blurring during the LBNP. Therefore, it was somewhat surprising that, in the alcohol experiments in which physiological decrements exceeded those of the placebo experiments, all of the subjects reported complete absence of any adverse symptoms during LBNP. Despite quantitative physiological decrements imminent danger of unconsciousness was not demonstrated under these experimental conditions.

Alcohol has been shown to cause diuresis beyond that induced by a comparable amount of water intake (21). The diuresis generally occurs only while the BAC is rising and is ascribed to an inhibitory effect of alcohol on secretion of antidiuretic hormone (21). As BAC falls from its peak value diuresis transitions into oliguria but water loss continues via increased respiration and perspiration (21). Thirst is not usually felt during acute alcohol intoxication in spite of increasing serum osmolality but occurs only after alcohol has disappeared from the body (21). While BAC is rising, movement of intracellular water into the circulatory compartment offsets the simultaneous diuretic loss from the circulation (21). Therefore, during the rise in BAC there is a net temporary protection against a plasma volume decrease (21). In our study, the imbibed volume of alcohol plus Coca Cola (about 350-550 ml) temporarily expanded the plasma volume. In general, postexperimental micturition was much greater in the alcohol than in the placebo experiments. Also, both the BP and HR showed slight elevations during the rising phase of the BAC. These elevations are consistent with the findings of others (10,11). Because decreases in plasma volume, BP and HR are known to decrease orthostatic tolerance (15,20), it is possible that, in our study, a net positive effect of internal liquid exchanges on plasma volume along with the slightly elevated BP and HR around peak BAC were temporarily protective against orthostatic syncope during the applied LBNP.

Both HR and BP, which rise along with the BAC, have been observed to decrease as the BAC decreases (10,11). As BAC recedes towards zero, respiration and perspiration water losses continue and plasma volume decreases by loss of water to both

the intracellular compartment and to the urine (21). The net effect of these water losses usually remains uncompensated during BAC recession since thirst does not ordinarily return until the alcohol has disappeared from the body (21). These observations suggest the possibility that the accommodation to applied orthostatic stress during peak BAC may diminish during BAC recession. To our knowledge orthostatic tolerance has not been tested during BAC recession.

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