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Increase in Whole-Body Peripheral Vascular Resistance During Three Hours of Air or Oxygen Prebreathing

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INCREASE IN WHOLE-BODY PERIPHERAL VASCULAR RESISTANCE DURING THREE HOURS OF AIR OR OXYGEN PREBREATHING

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ABSTRACT:

Male and female subjects prebreathed air or 100% oxygen through a mask for 3.0 hours while comfortably reclined. Blood pressures, heart rate, and cardiac output were collected before and after the prebreathe. Peripheral vascular resistance (PVR) was calculated from these parameters and increased by 29% during oxygen prebreathing and 15% during air prebreathing. The oxygen contributed substantially to the increase in PVR. Diastolic blood pressure (DBP) increased by 18% during the oxygen prebreathe while systolic blood pressure (SBP) showed no change under either procedure. The increase in PVR during air prebreathing was attributed to procedural stress common to air and oxygen prebreathing.

INTRODUCTION:

Prebreathing 100% oxygen while comfortably seated at sea level for several hours has been the traditional approach for aviators to remove nitrogen prior to high altitude exposure. The removal of nitrogen decreases the probability of subsequent decompression sickness. However, oxygen at one atmospheric pressure has a vasoconstrictive affect on the vasculature (refs. 1,2,3). Sitting quietly for hours could also reduce heart rate, blood pressure, and cardiac output which may decrease a nitrogen washout. Studies on nitrogen washout indicate that cardiac output is directly related to the efficiency of the washout (refs. 4,5,6).

Increasing the resistance to blood flow through a tissue may retard the removal of nitrogen from that region. This study attempts to quantify the change in whole body (PVR) in males and females prebreathing oxygen at sea level for 3.0 hours. It attempts to distinguish what fraction of the PVR change is attributed to 100% oxygen prebreathing and what fraction is attributed to other procedural factors.

METHODS

Three males and two females prebreathed dry aviators oxygen at 21° C for 3.0 hours for a total of 9 tests. The same 5 individuals also prebreathed dry compressed air on one occasion for the same duration. Table I shows the physical characteristics and total number of oxygen exposures for each subject.

The oxygen or air was delivered through a pressure demand full face diving mask[1]. Subjects were reclined with legs parallel to the floor for 3.0 hours in a comfortable recliner during the air or oxygen prebreathe. They were allowed to stand and stretch after the first and second hour and were required to stand during the transition from the sitting to the supine position after the third hour.

Peripheral vascular resistance was calculated prior to and following the 3.0 hour tests. A simple relationship exists to discribe PVR (ref. 7).

$$\text{EQ (1)} \qquad PVR = \frac{\text{mean arterial pressure (mmHg)}}{\text{cardiac output (liters/min)}}$$

Mean arterial pressure (MAP) being calculated as (ref. 8):

$$\text{EQ (2)} \qquad MAP = \frac{SBP + 2(DBP)}{3}$$

Blood pressures were obtained by standard

TABLE I

SUBJECT PHYSICAL CHARACTERISTICS

SUBJECT #	NUMBER OF TESTS	AGE	WEIGHT (KG.)	HEIGHT (CM.)	BODY FAT (%)	SEX
1	2	28	88.6	185.4	18	M
2	3	35	68.0	180.3	12	M
3	1	24	68.0	170.1	13	M
4	1	28	58.9	167.6	24	F
5	2	34	58.0	165.1	22	F

sphygmomanometry with the cuff over the brachial artery. Cardiac output was calculated from data supplied during noninvasive 2D and M-Mode echocardiography[2] (refs. 9,10) by a trained nurse.

After stabilizing the subjects in a supine position for 5.0 minutes the cardiac output was determined. Three quality estimates were collected in 5.0 minutes and an average of the 3 was our baseline value. Still in the supine position, 3 measures of SBP, DBP, and heart rate were taken and averaged. This same procedure was repeated after 3.0 hours of oxygen or air prebreathing. Blood pressure and heart rate were collected at 30, 60, 120, 150, and 180 minutes during the tests. The first oxygen prebreath test was done without the PVR estimate for each subject. This was done to lessen any "anxiety" effect caused by naive subjects prior to a PVR estimate.

RESULTS:

The collected data was analysed by two methods. A nondirectional paired t-test was applied to all pre- and postoxygen or air prebreathe data. Probability values less than 5.0% were considered significant. This analysis determines significant changes within each procedure. Since changes occurred under oxygen and air prebreathing an analysis of the absolute difference between measured parameters using air or oxygen was also performed. This was possible by applying the same t-test and analyzing the mean differences between oxygen and air data. This would determine significant changes between both procedures.

During 9 oxygen prebreathes for 3.0 hours in males and females PVR increased on an average by 29%. This was a statistically significant increase ($p<0.01$) from the baseline value (15.2 $\pm$ 3.4 vrs. 21.3 $\pm$ 5.6 mmHg/lit/min). During 5 air prebreathe tests a significant increase ($p<0.05$) of 15% in PVR was also evident (15.5 $\pm$ 3.6 vrs. 18.3 $\pm$ 3.5

mmHg/lit/min).

During quiet prebreathing using air, changes occurred in DBP, heart rate, and cardiac output that were in the same direction as changes noted under oxygen prebreathing. However, the magnitude of the changes using oxygen were always greater and statistically significant while the changes under air prebreathing were not statistically significant except for the PVR which is a derived parameter based on data that did show trends but never achieved statistical significance.

Figure 1 shows the percent change from baseline of the measured and derived data. The shaded areas indicate that portion of the change caused by oxygen that was above the contribution of sitting quietly in a reclined position with the breathing system.

Table II shows the average data for each subject during the oxygen prebreathing. Applying a paired t-test to the pre- and postsupine data reveals that DBP, heart rate, and cardiac output were statistically different at least at the $p<0.05$ level. Since MAP and PVR were derived from the above, data they also showed significant differences.

Heart rate decreased by 15%, cardiac output decreased by 18%, and DBP increased by 18% over the course of the oxygen prebreathe. Mean arterial pressure increased by 12% which reflected the increased DBP.

Table III shows the data for each subject during the air prebreathe. Heart rate decreased by 12%, cardiac output decreased by 14%, DBP increased by 7%, and MAP increased by 4% over the 3.0 hours. These were not statistically significant changes from pretest data.

To establish which parameters were substantially affected by oxygen prebreathing a paired comparison of the differences in the air and oxygen data was performed. This analysis

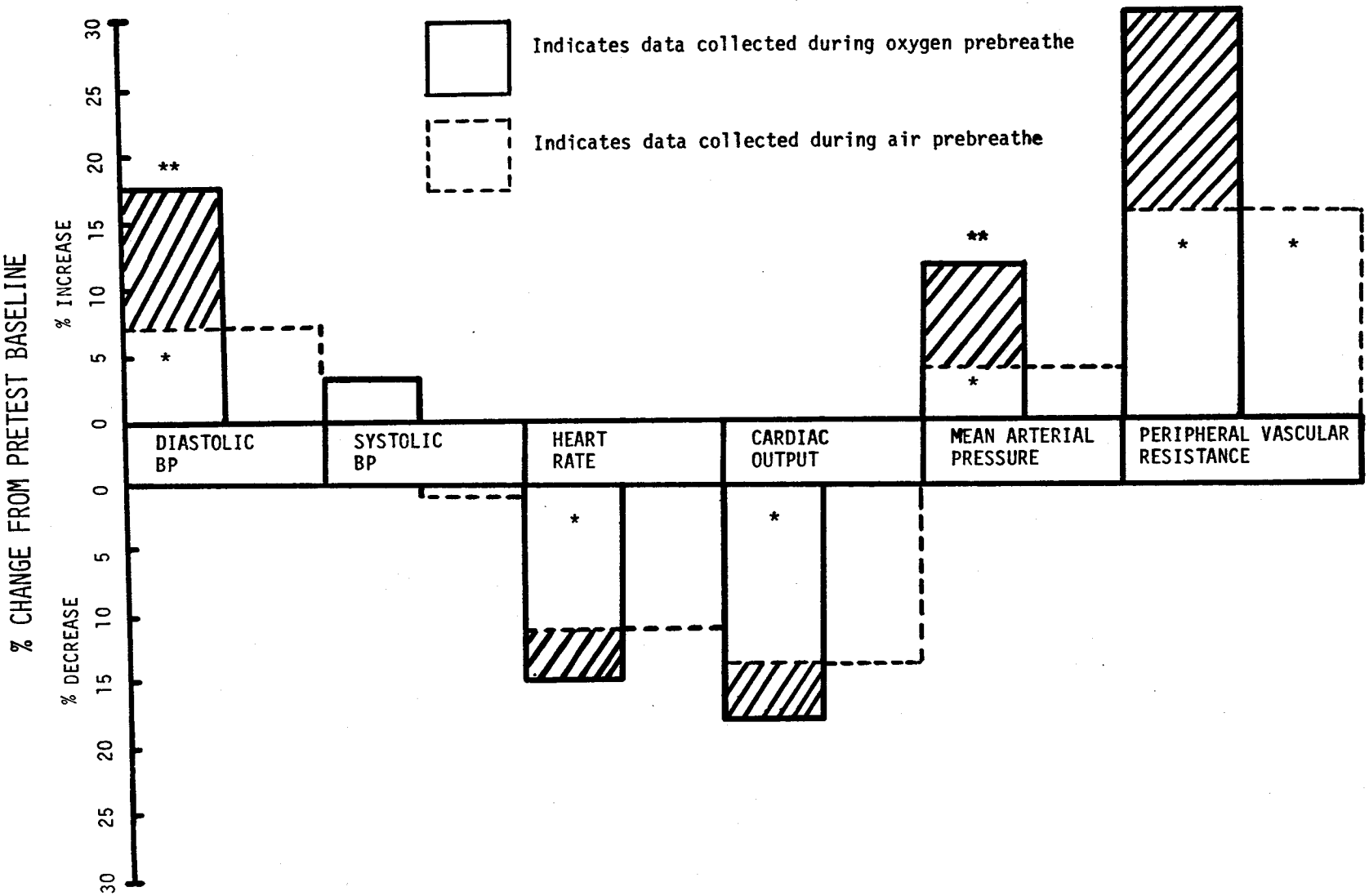

Figure 1. Changes induced in various parameters by oxygen and other procedural factors. Shaded areas indicate changes above those observed foe the same procedure using air. * indicates significant differences ($p<0.05$) from pre and post data in air or oxygen. ** indicates the oxygen induced change that was significantly different ($p<0.05$) from the changes induced by procedural factors.

TABLE II

STATISTICAL ANALYSIS OF PRE AND POST PREBREATHE PROCEDURES(OXYGEN)

SUBJECT #	NUMBER OF TESTS (OXYGEN)	SYSTOLIC BP MMHG	DIASTOLIC BP MMHG	HEART RATE	MEAN ARTERIAL PRESSURE MMHG	CARDIAC OUTPUT LIT/MIN	PERIPHERAL VASCULAR RESISTANCE MMHG/LIT/MIN
PRE 1	2	123	69	71	87	7.81	11.11
POST 1		128	79	63	95	6.56	14.65
PRE 2	3	141	84	74	103	5.53	18.97
POST 2		152	111	52	125	4.61	27.19
PRE 3	1	115	60	55	79	6.26	12.56
POST 3		115	84	51	95	5.83	16.20
PRE 4	1	115	67	78	83	5.53	15.04
POST 4		116	75	68	89	3.52	25.17
PRE 5	2	120	80	71	93	5.43	18.29
POST 5		123	88	63	100	4.45	23.50
MEAN ($\bar{X}$) PRE N=5		123 ± 11	72 ± 10	70 ± 9	89 ± 9	6.11 ± 1.00	15.19 ± 3.4
MEAN ($\bar{X}$) POST		127 ± 15	87 ± 14	59 ± 7	101 ± 14	4.99 ± 1.20	21.34 ± 5.6

TABLE III

Statistical Analysis of Pre and Post Prebreathe Procedures(AIR)

SUBJECT #		NUMBER OF TESTS (AIR)	SYSTOLIC BP MMHG	DIASTOLIC BP MMHG	HEART RATE	MEAN ARTERIAL PRESSURE MMHG	CARDIAC OUTPUT LIT/MIN	PERIPHERAL VASCULAR RESISTANCE MMHG/LIT/MIN
1	PRE	1	126	69	73	88	7.38	11.92
	POST		130	76	66	94	6.70	14.02
2	PRE	1	133	77	76	96	6.80	11.32
	POST		136	93	60	107	5.07	15.38
3	PRE	1	116	58	49	77	5.50	17.45
	POST		112	61	53	78	5.05	21.18
4	PRE	1	111	68	81	82	4.17	19.66
	POST		108	74	72	85	4.47	19.01
5	PRE	1	126	84	82	96	5.63	17.12
	POST		118	79	65	92	4.18	22.00
MEAN ($\bar{X}$) PRE		N=5	122 ± 9	71 ± 10	72 ± 13	88 ± 8	5.89 ± 1.24	15.49 ± 3.7
MEAN ($\bar{X}$) POST			121 ± 12	77 ± 11	63 ± 7	91 ± 11	5.09 ± 0.97	18.31 ± 3.5

showed that the increase displayed in figure 1 for DBP above the level for the air prebreathe (shaded area) was statistically significant. This was also true for MAP, but not for cardiac output, heart rate, and PVR. There was no statistically significant change in heart rate, cardiac output, or PVR on oxygen when compared to the changes observed in the air prebreathe, however the trends are evident with the magnitude of the differences always being greater for oxygen prebreathing. A statistically significant difference may have been obtained for PVR with a larger subject population.

CONCLUSION:

It was possible to measure a true whole body increase in PVR during oxygen prebreathing with the techniques described. An effective increase in PVR of 29% occurred on oxygen with 15% of the increase being attributed to other procedural factors.

Diastolic blood pressure and therefore MAP consistantly increased while breathing air or oxygen over the course of 3.0 hours. They increased to a greater degree on oxygen which indicates general vascular constriction. Systolic blood pressure remained constant under all test conditions while heart rate and cardiac output decreased in both procedures. Only during the oxygen prebreathe were these decreases statistically significant.

Past data (ref. 3) indicates that specific organs, especially brain structures and viscera, increase vascular resistance in response to 100% oxygen. This technique does not allow a detailed analysis of specific organ systems. Instead, a whole- body response to oxygen was determined. This has operational implications since the technique reproduces the general procedures used by aviators to remove tissue nitrogen prior to altitude exposure.

Oxygen can be considered as a drug that has dose-response characteristics. Individuals could respond to oxygen differently. This may explain some of the variability in individual susceptibility to decompression sickness.

Sitting quietly for 3.0 hours breathing air did increase PVR to a degree (15%). This proved to be a counterproductive situation in this procedure. It is desirable to keep PVR as low as possible so the total magnitude of the oxygen contribution and the contribution due to specific prebreathe procedures will not be excessive. The amount of nitrogen removed from the body may be improved if PVR can be reduced.

The results from this study show that oxygen as well as general sedentary conditions increased PVR over extended periods. It is suggested that aviators perform moderate physical activity during the course of lengthy oxygen prebreaths. This will increase cardiac output which may offset the increase in PVR since carbon dioxide in active tissue will regulate vascular beds. It is not certain that vascular structures near the knees, ankles, and elbows respond to 100% oxygen as do more sensitive neural structures. Moderate exercise may not have a beneficial effect in these "bends" susceptible areas but any increase in cardiac output has the potential of improving a nitrogen washout.

The results presented in this study were obtained from a pre- and postmeasurement period. The measurements were separated by 3.0 hours. The oxygen and procedural effects were cummulative in nature and a detailed study of the changes during the 3.0 hours would be beneficial.

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FOOTNOTES

1. Intertechnique Type 409716 Oxygen Breathing Mask. 78 Plaisir, France.

2. ATL/ADR Model 4000S/LC Ultrasound. Tempe, Arizona.

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16. Abstract

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