EFFECTS ON MAN OF PROLONGED EXPOSURE TO OXYGEN AT A TOTAL PRESSURE OF 190 MM. HG

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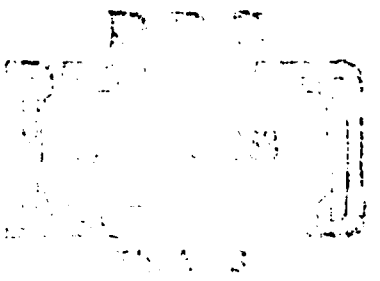

Task No. 793002

FOREWORD

This report was prepared by the following personnel in the Environmental Systems Branch:

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ABSTRACT

Four 17-day experiments were conducted on healthy pilots in the two-man space cabin simulator. Total pressure averaged 192 mm. Hg; partial pressure of oxygen, 174 mm. Hg. This atmosphere was reasonably well tolerated by all test subjects. The symptoms consisted of irritation of the upper respiratory tract, aural atelectasis, and eye irritation. Postexperimentally, 2 of the 8 subjects demonstrated reduced arterial oxygen saturation immediately. Pictures, however, gave no x-ray evidence of pulmonary atelectasis. The incidence of baropathies following decompression from 750 mm. Hg to 190 mm. Hg exceeded 50% (5 of 8 subjects), even following 2 to 3 hours of preoxygenation.

The utilization of an atmosphere consisting of oxygen, carbon dioxide, and water vapor at a total pressure of 190 mm. Hg appears to be physiologically feasible, at least for 17 days. Care must be taken to overcome the problem of bends during the initial decompression and to insure that engineering gains are sufficiently great to offset the symptoms noted in these studies.

This technical documentary report has been reviewed and is approved.

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1. INTRODUCTION

The development of rocket flights and the prospect of prolonged manned extraterrestrial journeys have stimulated much interest in the choice of the spacecraft atmosphere. Several gaseous environments have been considered which range from sea-level conditions to 100% oxygen at an ambient cabin pressure of 190 mm. Hg. The use of low ambient pressure and 100% oxygen at that pressure appears attractive since such an atmosphere makes possible economy of weight, engineering simplification, monitoring ease, and reduction of atmospheric gas-leak rates. Very little physiologic information is available, however, which can be related to prolonged exposure to an atmosphere containing only oxygen at reduced barometric pressure.

The administration of oxygen at high partial pressures is known to produce the syndrome of "oxygen toxicity" (1, 3). The experimental work of Becker-Freyseng and Clamann (2) and Ohlsson (13) amply documents the clinical syndrome of oxygen toxicity and the pathologic changes which occur on exposure to oxygen pressures of more than 600 mm. Hg. Therefore, the possible use of oxygen atmospheres in the absence of inert gases and at ambient partial pressures, greater than that encountered at sea level (P_{O_2} = 159 mm. Hg), led us to undertake the experimental studies described in this report.

2. METHODS

Four 17-day experiments were conducted on healthy male pilots in the two-man space cabin simulator at the USAF School of Aerospace Medicine. Essentially, the simulator is an altitude chamber which contains facilities for the maintenance of two men under carefully controlled conditions for prolonged periods. The general outline of the experiments has been described previously (11, 16). Total pressure was sensed by an absolute pressure transducer; P_{O_2} by a Beckman oxygen analyzer, model F-3; and P_{CO_2} by a Beckman carbon dioxide analyzer, model 15-A. The P_{H_2O} was calculated from relative humidity data obtained with an El-Tronic hygrometer, model 101C, and from temperature data obtained with a Minneapolis-Honeywell resistance bulb, model 6630. These results were cross-checked with a modified Assman-type wet-bulb–dry-bulb thermometer. Nitrogen was calculated by difference, or determined with a gas chromatograph by using a molecular sieve column, with periodic checks against a Waters nitrogen analyzer, model A-6. Volunteer subjects were examined extensively by the Aeromedical Evaluation Service of the USAF School of Aerospace Medicine, and baseline studies were conducted at ground level (average barometric pressures 750 mm. Hg, P_{O_2} 156 mm. Hg). Subjects giving a history of dysbarism were excluded. The subjects were then exposed to the experimental environment (table I). After 17 days' exposure to the experimental environment, physical examinations were repeated and postexperimental studies were obtained at ground level. All subjects completed the experiments without interruption. In 3 subjects, however, dysbarisms occurred despite oxygen prebreathing (100% O_2 at 750 mm. Hg pressure from 2 to 3 hours) before ascent to altitude. In these cases, the experimental plan was altered during the first 24 hours so that both subjects were

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TABLE I

Experimental conditions

Duration	17 days
Number of subjects	8
Equivalent altitude	33,500 feet
Ambient pressure	192 ± 15 mm. Hg
P_{O_2}	174 ± 15 mm. Hg
P_{CO_2}	3.2 ± 3 mm. Hg
Temperature	20.7 ± 1° C.
Relative humidity	58 ± 20%

exposed to higher oxygen tensions for periods up to 14 hours (total pressure 250 mm. Hg, P_{O_2} 237 in one case, and total pressure 380 mm. Hg, P_{O_2} 365 in the others).

Several measures of pulmonary function were studied serially by using the methods previously reported (11). Primary emphasis was placed on vital capacity and timed expiratory volume studies which were carried out as described by Comroe et al. (4). All volume measurements were converted to body temperature and ambient pressure saturated with water vapor (BTPS). The following formula was used:

$$V_{BTPS} = V_{OBS} \times \frac{(273 + 37)\ (P_B - P_{H_2O})}{(273 + t)\ (P_B - 47)}$$

where V_{OBS} is the volume measured; t is the ambient temperature of the gas in °C.; P_{H_2O}, the water vapor pressure in mm. Hg at temperature t °C.; P_B, the ambient pressure, and 47, the vapor pressure of water at body temperature. The ground-level altitude of the laboratory was 577 feet and average station pressure was 750 mm. Hg.

Medical appraisal was carried out twice daily at 7:00 a.m. and 7.00 p.m. by systematic review. Symptoms obtained were noted but not discussed, and leading questions were avoided wherever possible. Daily blood pressure, pulse, rectal temperature, and electrocardiogram were recorded, and performance on a psychomotor apparatus was monitored continuously from each subject for 10 hours each day. The work-rest schedule and schedule of test periods were described previously (9). The psychomotor apparatus was designed to test functions of the peripheral and central nervous system and motor responses at various levels of behavior (6). The performance results have been reported in detail by Hartman et al. (7).

During these studies, pre-experimental and postexperimental brachial or femoral artery punctures were performed on subjects breathing 100% oxygen by face mask for 10 minutes, and oxygen content and capacity were determined by the method of Van Slyke (15). Postero-anterior and lateral chest x-ray films were obtained during the baseline studies and the postexperimental studies immediately following the arterial-puncture studies.

3. RESULTS

During the course of the experiment, all subjects reported symptoms which previous workers have attributed to oxygen toxicity. These symptoms were of varying intensity and are tabulated in table II. At 33,500 feet, subject 2 had unequivocal burning substernal pain; this pain occurred at the ninth day and persisted for a period of about 24 hours. As a trial, oxygen partial pressure was increased for several hours (increased total pressure) and the subject reported amelioration of the symptoms which did not then recur on returning to 190 mm. Hg total pressure (P_{O_2}, 175, equivalent altitude, 33,500 feet). The experiment was completed uneventfully. Nasal congestion and eye irritation were almost universal complaints, the coryza and irritation being especially severe during the early days of exposure. Nasal dryness, congestion, and rhinorrhea all occurred, sometimes in the same subject. A mild cough was noted in half of the subjects. All subjects noted that they were forced to clear their ears by Valsalva maneuver or by swallowing, especially after arising from sleep; in 5 subjects there was actual ear discomfort at some time during the experiment with difficulty in clearing the blocked ears. Transient paresthesia (cold sensation, flushing, tingling of extremities) was noted in 50% of the subjects. Two subjects had postural dizziness which was transient and did not interfere with the experiment. Aching in the teeth was noted in 1 subject who had had an episode of barodontalgia in

TABLE II

Manifestations of oxygen toxicity

Symptom	Occurrence (number of subjects)
Substernal pain	1
Lower respiratory tract:	
Reduced vital capacity*	8
Upper respiratory tract:	
Cough	4
Nasal congestion	6
Sore throat	1
Ear discomfort (aural atelectasis)	5
Fatigue	—
Eye irritation	7
Other	
Paresthesia	4
Dizziness	2
Aching teeth*	1
Joint or muscle pain†	1

*Probably related to reduced barometric pressure.

†Four other cases were noted and were probably related to reduced barometric pressure.

the same tooth several years previously. This pain was probably not due to oxygen, per se. Joint pain, as reported by Comroe et al. (3), occurred frequently in our experiments; however, in only one instance might the pain have been due to oxygen toxicity. In this case, mild, aching pain in the left thigh occurred on the sixth day and diminished without therapy by the eighth day. In the remaining 4 cases, bends of grade 2 severity (moderately severe pain, but tolerable) were encountered at the beginning of the experiment as a result of the change in total barometric pressure. These bends were relieved by appropriate temporary adjustment of cabin pressure.

Auscultation of the lungs was performed by medical observers at 750 mm. Hg ambient pressure immediately following the conclusion of the experiment. In 6 of the 8 subjects, definite crepitant rales were heard on inspiration at both lung bases posteriorly; these cleared after repeated deep inspiration. As noted previously (11, 14), vital capacity was uniformly reduced in subjects exposed to altitude. This reduction in vital capacity cannot be separated from any possible reduction due to the atmospheric composition (table III). Vital capacity was reduced in all subjects, the average experimental value being 7.6% less than the pre-experimental baseline control values. This reduction occurred in the first measurements made after altitude was reached. Values returned to the pre-experiment level promptly on return to ground-level pressures. Chest x-ray films taken at the close of the experimental period revealed no abnormalities, but the subjects had been breathing a normal sea-level atmosphere for 10 to 40 minutes prior to these studies.

Postexperimental arterial oxygen saturations determined while the subjects were breathing 100% oxygen at 750 mm. Hg pressure were normal in 5 subjects (table IV). In 2 subjects, there was evidence of arterial

TABLE III

Forced vital capacity

Subject	Pre-experiment (liters)	Experiment (liters)	Postexperiment (liters)	Percent change (pre-experiment vs. experiment)
1	5.55 ± 0.25*	4.79 ± 0.21	5.29 ± 0.10	−13.7
2	5.47 ± 0.07	5.36 ± 0.18	5.54 ± 0.09	− 2.0
3	5.86 ± 0.18	5.52 ± 0.23	5.75 ± 0.13	− 5.8
4	5.82 ± 0.12	5.39 ± 0.18	5.79 ± 0.13	− 7.4
5	4.60 ± 0.08	4.43 ± 0.20	4.60 ± 0.22	− 3.7
6	6.32 ± 0.14	5.54 ± 0.27	6.18 ± 0.17	−12.3
9	4.64 ± 0.10	4.05 ± 0.20	4.59 ± 0.11	−12.7
10	5.73 ± 0.11	5.53 ± 0.17	6.02 ± 0.16	− 3.5
Av.	5.50	5.08	5.47	− 7.6

*Standard deviation of the observations.

TABLE IV

Arterial oxygen saturation data

Subject	Pre-experiment (%)	Postexperiment (%)
1	*	94.9
2	91.8	88.2
3	96.0	100.0
4	97.0	99.0
5	97.3	100.7
6	99.9	90.0
9	97.3	100.0
10	98.4	101.0

*No pre-experimental data.

unsaturation. Veno-arterial shunts were estimated[1] to be 30% of pulmonary blood flow in the second subject, and 21% in the sixth subject. In a third subject, arterial unsaturation of borderline degree was present but, in the absence of baseline data, no conclusions can be drawn as to the degree of arterial unsaturation resulting from exposure to the experimental conditions.

Serial electrocardiograms consisting of standard leads, aVF, aVL, and a chest lead (usually V_2) selected to show QRS transition

[1] Percent shunt $= \frac{\dot{Q}_S}{\dot{Q}} = \frac{\text{arterial oxygen content - oxygen capacity}}{\text{venous oxygen content - oxygen capacity}} \times 100.$

(Arterial-venous oxygen difference was assumed to be 5 vol. %.)

in the frontal plane were normal at all times during the experiments. Asymptomatic sinus bradycardia and sinus arrhythmia of marked degree were noted on several resting electrocardiograms. This is reflected by the decline in pulse seen in several subjects (table V), but it should be noted that systolic and diastolic blood pressures were well maintained and that pulse pressure was essentially unaltered.

4. DISCUSSION

The experimental atmosphere employed in this study represents one approach to a solution of the multiple problems of the design of spacecraft atmospheres. Engineering requirements (gas-leak rates, weight, ease of monitoring and control, etc.) are of sufficient interest to require the examination of the physiologic response of man to prolonged exposure to atmospheres chiefly composed of oxygen at reduced barometric pressure.

At a total pressure of 190 mm. Hg and in an oxygen atmosphere, ambient oxygen partial pressure is increased approximately 20 mm. Hg above that prevailing at sea level when partial pressures due to ambient water vapor and carbon dioxide have been subtracted. This 20 mm. Hg increase in P_{O_2} is diminished by increased P_{H_2O} and P_{CO_2} in the respiratory tract, until, at the alveolar level, only very

TABLE V

Blood pressure and pulse rates (pre-experiment and experiment)

Subject	Pre-experiment		Experiment		Comments
	Blood pressure	Pulse	Blood pressure	Pulse	
1	No data		92/63	59	BP decreased transiently to 70/50 without symptom on the 3d and 16th days while subject was asleep.
2	No data		109/68	61	
3	117/83	67	114/79	65	
4	104/66	71	118/76	60	Subject had two episodes of transient, narrowed vision, cold sweat, tachycardia, and BP 90/55 on the 6th and 15th days—probably from hyperventilation.
5	110/62	68	108/62	49	
6	106/58	58	104/62	50	
9	110/65	62	107/57	56	
10	111/62	60	126/65	55	

slightly increased Po_2 prevails. Nevertheless, a variety of symptoms occurred (table II) which were very similar to those described for oxygen toxicity (3). These symptoms were generally mild but some were persistent and reproducible, and we cannot exclude the possibility that the symptoms noted in this study were due to the oxygen-rich, nitrogen-poor atmosphere. It is also possible that they were related to increased insensible water loss from the mucous membranes due to the reduced pressure involved. This is based on the observations of Hale et al. (5) who performed their experiments over a pressure range from 760 to 253 mm. Hg and a water vapor pressure from 6 to 26 mm. Hg. They reported that total insensible water loss was inversely related to both barometric pressure and water vapor pressure—i.e., the lower the pressure or the lower the humidity, the greater the loss of insensible water. The possibility also exists that those irritative effects were induced by the dehydrating effect of dry oxygen used during prebreathing and at the beginning of the experiment. Since normal relative humidity was quickly established and maintained continuously at 35 to 65%, we do not feel that dehydration was a significant factor. The substernal pain which occurred in 1 subject on the ninth day was of the type noted by Comroe et al. (3) but was mitigated by increasing the oxygen partial pressure. Thus, it seems likely that the pain was related to factors other than increased ambient Po_2.

Although reduced vital capacity may be caused by oxygen toxicity at high partial pressures of oxygen (2, 3), it is more likely that the reduction in these experiments was due to the poorly understood effect of reduced pressure on vital capacity (11, 14). There remains, however, the possibility that the near absence of nitrogen can produce pulmonary atelectasis by a combination of physical and chemical effects (in much the same manner as aural atelectasis is produced under these conditions). Such atelectasis would be expected to manifest itself by reduced vital capacity, x-ray evidence of linear coalescent densities, and reduced arterial oxygen saturation due to veno-arterial shunting through unaerated pulmonary tissue. The first two manifestations have been noted at oxygen partial pressures of 760 mm. Hg (2) and 418 mm. Hg (10) after 2 and 7 days' exposure, respectively, but none of these findings were expected in the present experiment. Thus, the finding of arterial oxygen unsaturation in 2 (possibly 3) subjects (table IV) was surprising. This finding suggests that occasional subjects will respond to oxygen-rich atmospheres in a one-gravity environment in a manner similar to that reported more frequently in oxygen-rich, multiple-gravity environments (8, 12). As the causes of this condition are not well understood, they are being studied further.

A potential hazard of exposure to reduced barometric pressure was the production of decompression symptoms. Subjects were screened for history of previous altitude reaction (bends, chokes, etc.). Denitrogenation was carried out by having the subjects wear well-fitting aviators' masks and prebreathe 100% O_2 at 750 mm. pressure for a minimum of 2 hours. Nevertheless, there was a very high incidence of bends (more than 50%) in our subjects. This suggested that a reappraisal of the criteria for denitrogenation and crew selection be made when prolonged exposure to cabin altitudes similar to the pressure utilized here is anticipated.

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