Observations in the SAM Two-Man Space Cabin Simulator

II. Biomedical Aspects

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EXPERIMENTATION in the space cabin simulator at the School of Aviation Medicine has been designed, in part, to study the physiologic and psychologic responses of the human organism to a "space-equivalent" environment. This environment represents the predicted space cabin environment in most respects, the most important absent environmental parameter being zero-gravity. The subjects are maintained under ideal experimental conditions: continuous observation, continuous environmental control, uniform activity and control of food and water intake and excretory output. Under these conditions we have been able to study the physiologic effects of prolonged inactivity, isolation, exposure to altitude and exposure to high oxygen percentage in the absence of inert gas.

This report will deal first with observations on the physiological responses and physical performance of the space cabin subjects. Secondly, the problem areas of water balance, pulmonary function, oxygen toxicity and dysbarism will be discussed in relation to recent results obtained in the space cabin simulator.

METHODS

The experimental altitude chamber, its life support equipment and the experimental design have been described.[15] The subjects were volunteer pilot officers with a scientific background. Testing of the subjects in the pre-flight period consisted of psychological and physical examination, measurement of work capacity and orthostatic tolerance, pulmonary function studies and dental, audiologic, roentgenologic and ophthalmologic examinations. Complete urinalysis and hematologic studies were made and serum electrolytes, glucose, BUN, lipids, protein electrophoresis, protein bound iodine, pH, osmolarity and carbon dioxide were measured. A cardiologic and bacteriologic survey was performed; metabolic rate and alveolar gas determinations were made using a Custom Engineering and Development Company Servo-Spirometer and the Scholander micromanometric gas apparatus. Body water determinations were made by the antipyrine method[12] or with deuterium oxide,[16] extracellular space by the inulin calibrated infusion method[10] and blood and plasma volumes by the use of radio-iodinated serum albumin.[6] Many of these examinations were carried out in the facilities of the Consultation Service of the School of Aviation Medicine.

Following a period of indoctrination, the subjects entered the chamber for 30 days at a simulated altitude of 18,000 feet (oxygen enriched to 40 per cent, pO_2 150 mm. Hg) or for 17 days at 33,500 feet (100 per cent oxygen, pO_2 176 mm. Hg). Most of the pre-flight clinical tests were continued through the experiment and body weight, temperature, pulse, respiration, blood pressure and ECG were monitored. At the conclusion of the flight and at intervals

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NOTE: This manuscript reflects the views of the authors and is not to be construed as official Air Force policy.

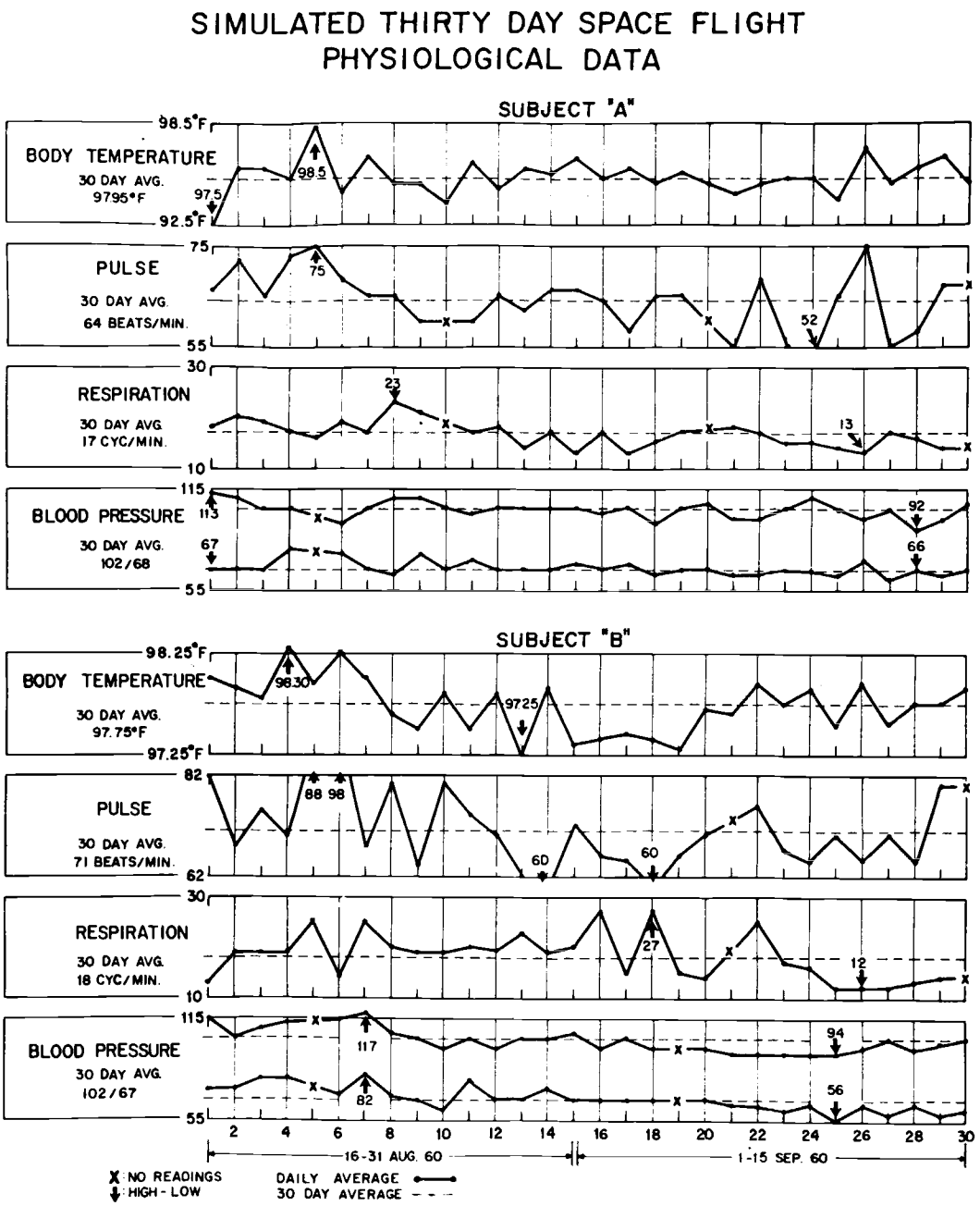

Fig. 1. In-flight physiological parameters.

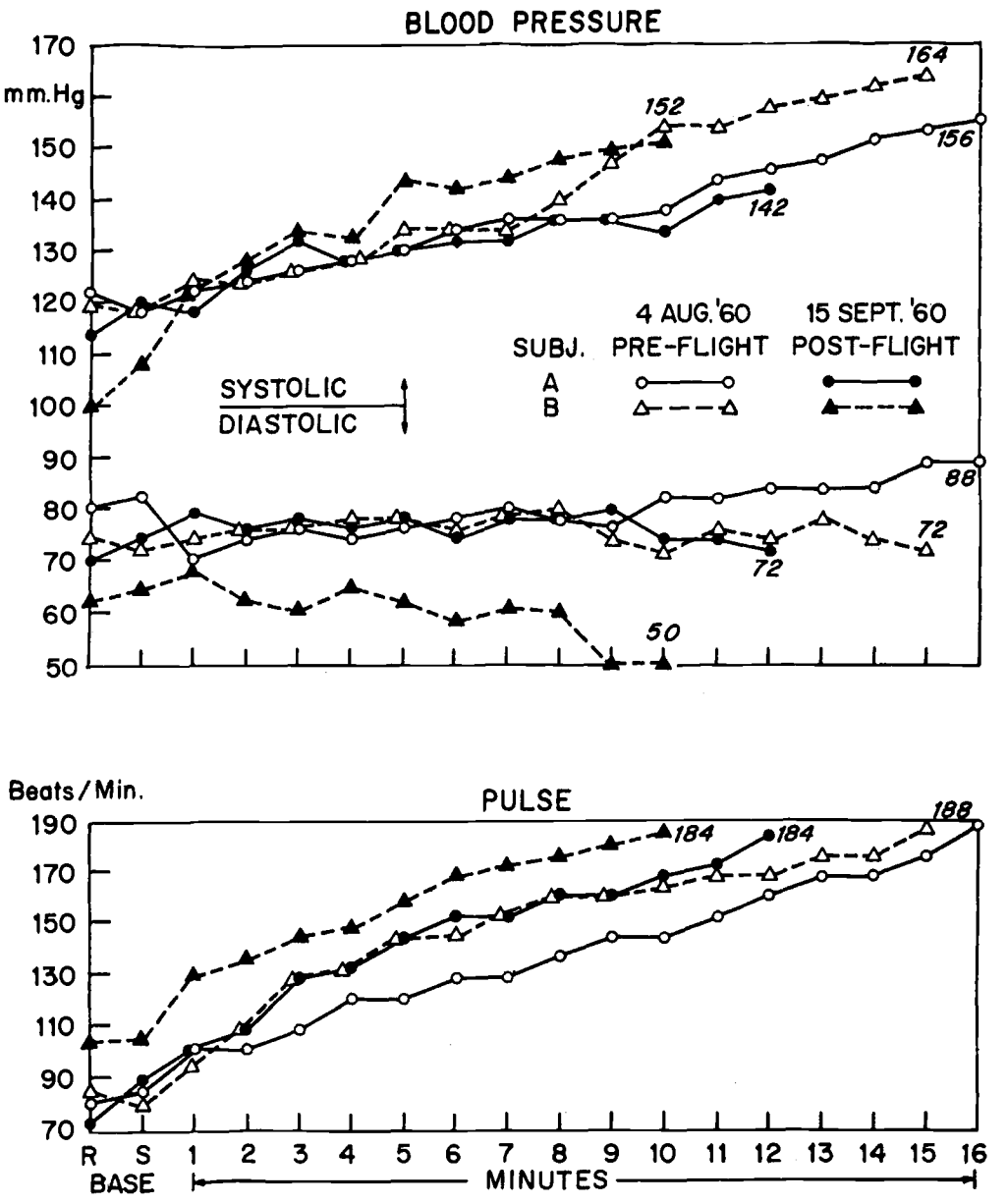

Fig. 2. Work capacity data preflight and postflight.

thereafter, the pre-flight testing was repeated as indicated.

PHYSIOLOGICAL RESULTS

Hematologic, bacteriologic, and clinical chemical studies and urinalysis, audiological, dental and ophthalmological examinations showed no significant changes during and after the experiment.

Figure 1 presents representative physiological data from one of the experiments (a 30-day simulated space flight) which demonstrates the relative stability of these parameters. Variations in temperature, pulse and respiration were small

at both 18,000 and 33,500 feet and were consistently at or near normal values. There was a significant decrease in resting diastolic blood pressure ($p < 0.05$) in both men at 18,000 feet and in one man at rest in a subsequent experiment at 33,500 feet altitude. Both systolic and diastolic pressures were maintained during sedentary activity and no hypotensive episodes were seen.

TABLE I. SUMMARY OF ORTHOSTATIC TOLERANCE DATA

Baseline	Subject A				Subject B			
	Pre-flight		Post-flight		Pre-flight		Post-flight	
	BP	P	BP	P	BP	P	BP	P
5 Min. 90° Tilt	120/76	62	122/80	75	114/72	58	104/72	84
1 Min.	120/78	79	118/84	88	112/78	69	108/98	88
2 Min.	118/80	80	124/84	95	110/76	63	112/84	93
3 Min.	124/82	83	122/80	88	116/80	75	112/99	96
4 Min.	118/80	64	120/84	94	118/76	73	99/78	97
5 Min.	122/80	85	126/86	101	118/78	77	—	94
6 Min.	122/82	74	112/80	99	116/80	70	104/82	82
7 Min.	120/84	93	122/82	95	118/80	75	104/76	103

Physical performance at the conclusion of the simulated flight was estimated by use of the treadmill, the tilt-table and extensive electrocardiographic and vectorcardiographic analyses conducted by the Department of Internal Medicine in the SAM Consultation Service. Work capacity was measured by the angle-increment treadmill method of Balke.[2] Representative responses are shown in Figure 2 in which pre-experiment and post-experiment data for the 30-day flight are shown. Though total treadmill time was reduced after the flight (from 16 to 12 minutes and 15 to 10 minutes), the decrease was not as marked as that seen in the seven day water immersion hypodynamic experiment of Graveline and Balke[7] in which total time was reduced from 15 to 8 minutes. On the other hand, the decrement was larger than that produced by four weeks of bed rest alone.[2] Only a minimal increase in pulse pressure and fall in diastolic blood pressure was noted. Also, pulse rate was higher for a given work load after the flight than before. These latter changes agree with the findings of Balke[2] and are typical of the effects of four weeks bed rest described by him.

Orthostatic tolerance, as determined on the tilt-table, was not greatly affected by either the 30-day, 18,000 feet or the 17-day, 33,500 feet experiments. Representative data for the tilt-table responses are shown in Table I; the minor changes seen here are not comparable to the severe pulse pressure decrease seen in the water immersion hypodynamic study.[7]

Changes in myocardial irritability and activity occurred and persisted into the post-flight period after both experiments. These changes consisted of a lengthening of atrio-ventricular conduction time (PR interval), atrial and ventricular premature contractions, variable atrial pacemaker or T-wave lability and were produced by tilt-table, Valsalva or Master's two-step maneuvers. By the time of the two-month follow-up examination, all changes had reverted to normal.

It is apparent that the effect of a prolonged simulated space cabin flight on work capacity, orthostatic tolerance and myocardial activity is mild but definite. The changes observed were more marked than those induced by comparable periods of forced inactivity and were approximately the same at both 18,000 and 33,500 feet simulated altitude.

OBSERVATIONS ON WATER BALANCE

At the conclusion of the first 30-day simulator experiment, it became evident that an unexpected weight loss together with decreases in total body water, total blood volume and plasma volume had occurred (Fig. 3). Loss of total body water was larger than the corresponding loss of body weight. Such a situation might be accounted for by a shift in body composition with

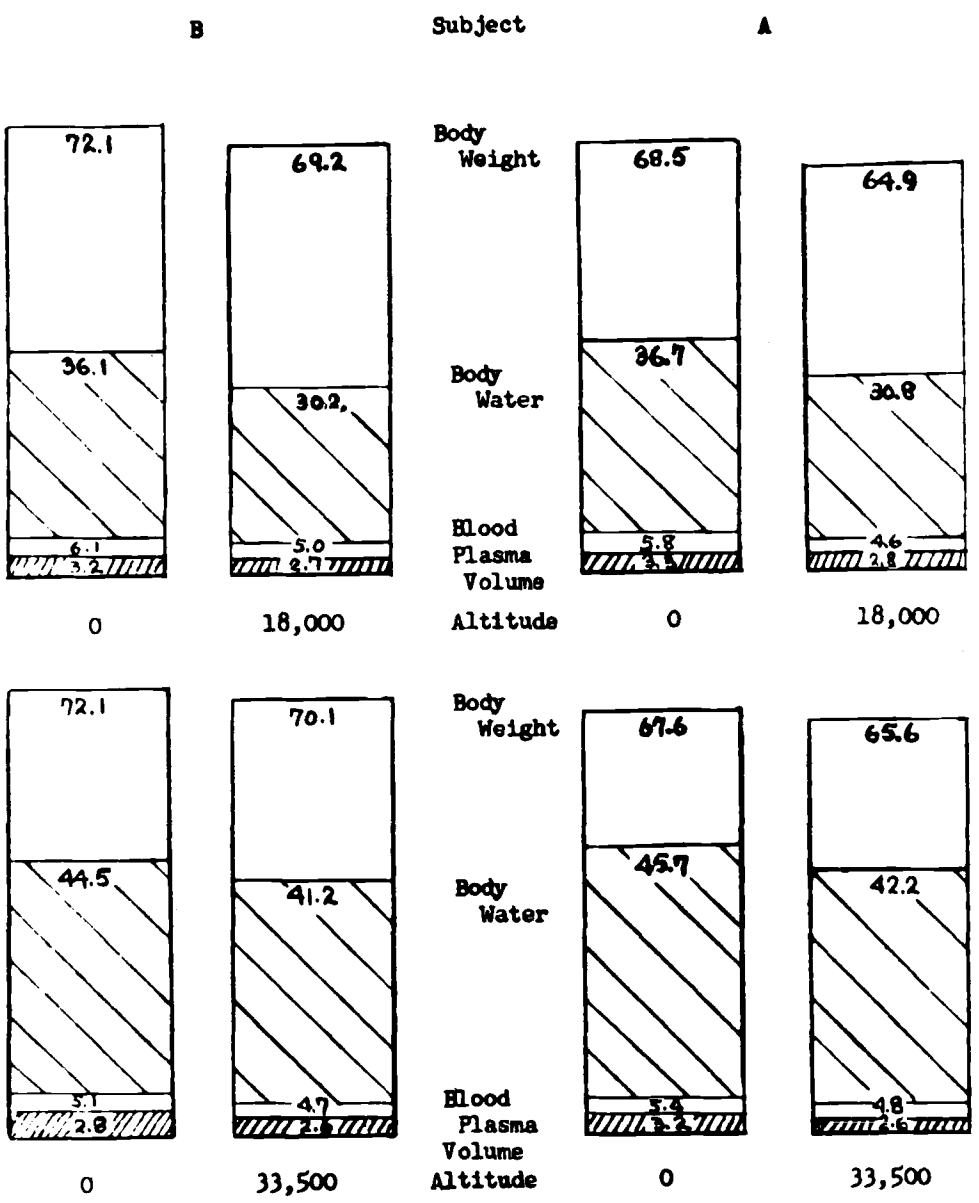

Fig. 3. Body fluid compartments after altitude exposure.

a greater proportion of fat at the conclusion of the experiment but no definite conclusions can be drawn.

Changes similar to those observed in the 30-day flight were seen in the more recent 17-day simulated flight at higher altitude (Fig. 3). The characteristics of the shift in body water stores appear to be such that a gradual weight loss occurs at about the same rate regardless of altitude (Fig. 4). On return to sea level, after 17 days of exposure, a rapid recovery ensues as contrasted with a slow, long delayed recovery after 30 days at altitude. This course suggests, among other explanations, that primarily dehy-

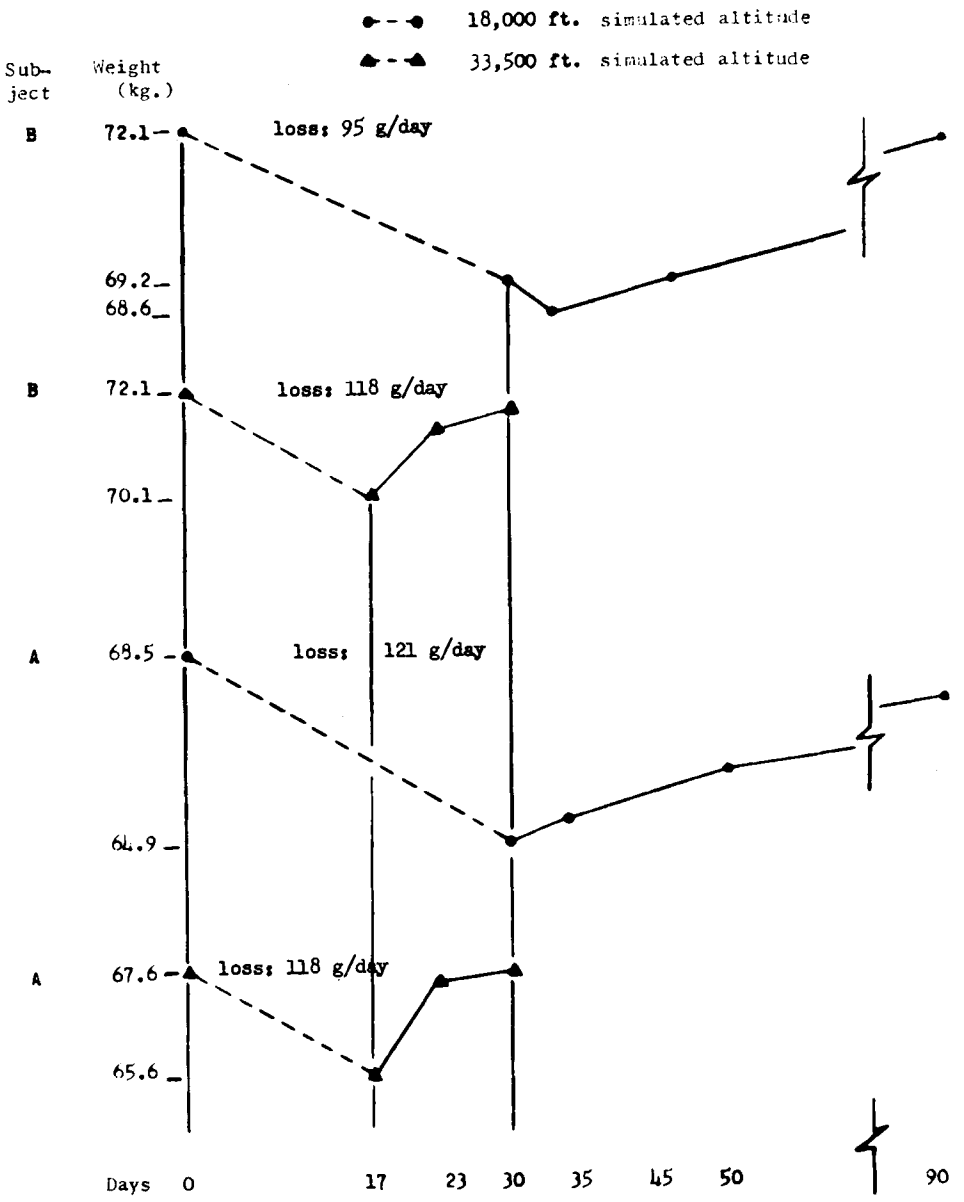

Fig. 4. Body weight during space cabin experimentation.

dration with reduction of extracellular water has occurred after 17 days and that after 30 days a change in body cellular composition has also occurred.

The genesis of this change is not clear. Simple dehydration is not clearly involved since water supplies were more than adequate, cabin relative humidity was 50-70 per cent and temperature was consistently near 23°C (74°F). Several workers[1,11,12] have suggested that altitude exposure produces a diuresis though later experimental work has not clearly established this fact or distinguished the effect of altitude from that of hypoxia.[8,14] Our observations also

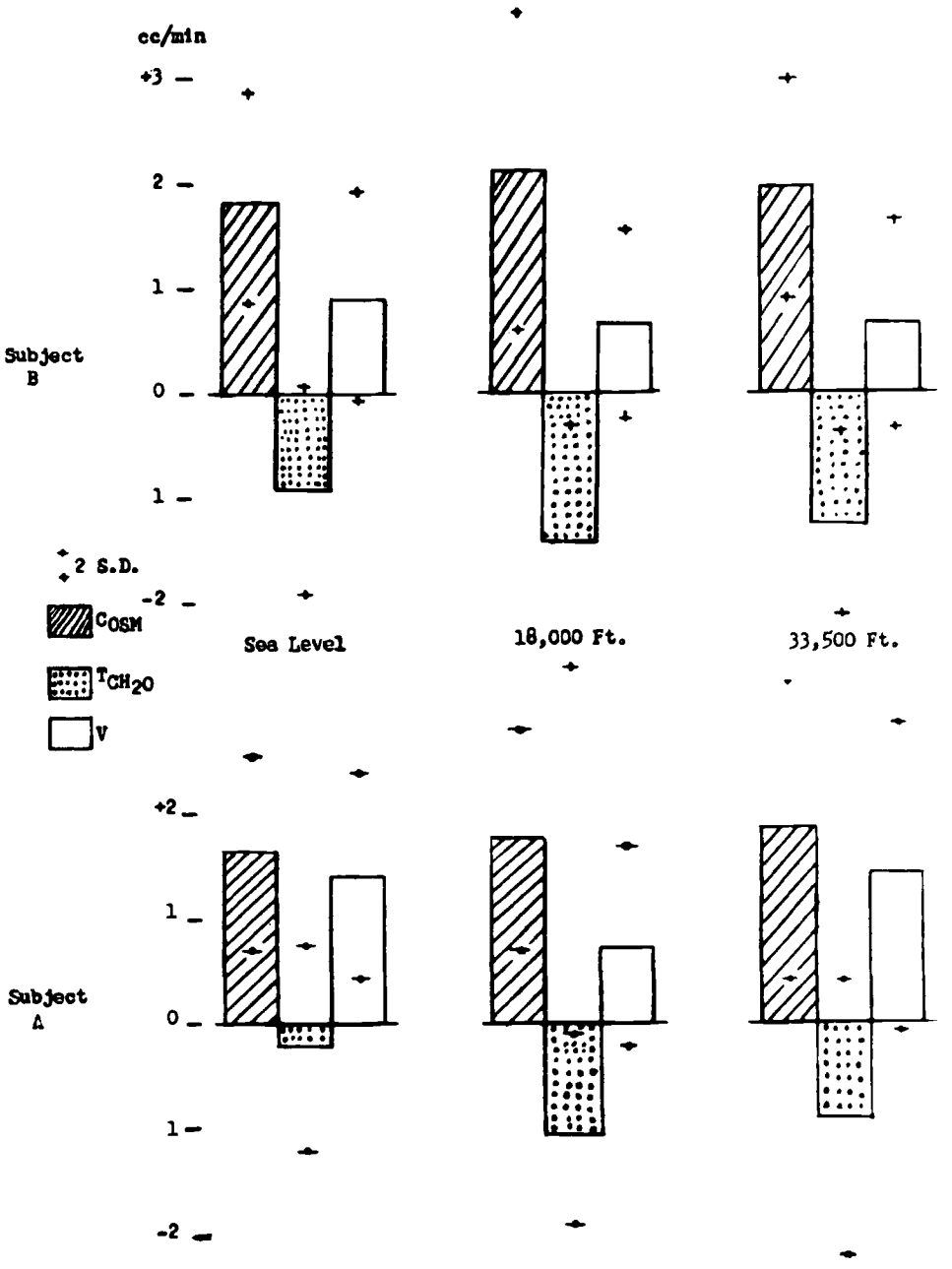

Fig. 5. Renal function at altitude.

rule out such an altitude induced diuresis; data are presented in Figure 5 in which no significant increase in urine flow (V), osmolar clearance (C_{osm}) or free water clearance ($T^{C}_{H_2O}$) are seen after altitude exposure. Finally, no hemoconcentration occurred during the experiments as noted by the lack of increase in hematocrit or plasma osmolarity (Table II). These changes tend to suggest that dehydration in the usual sense is not the cause of the observed water loss. An at-

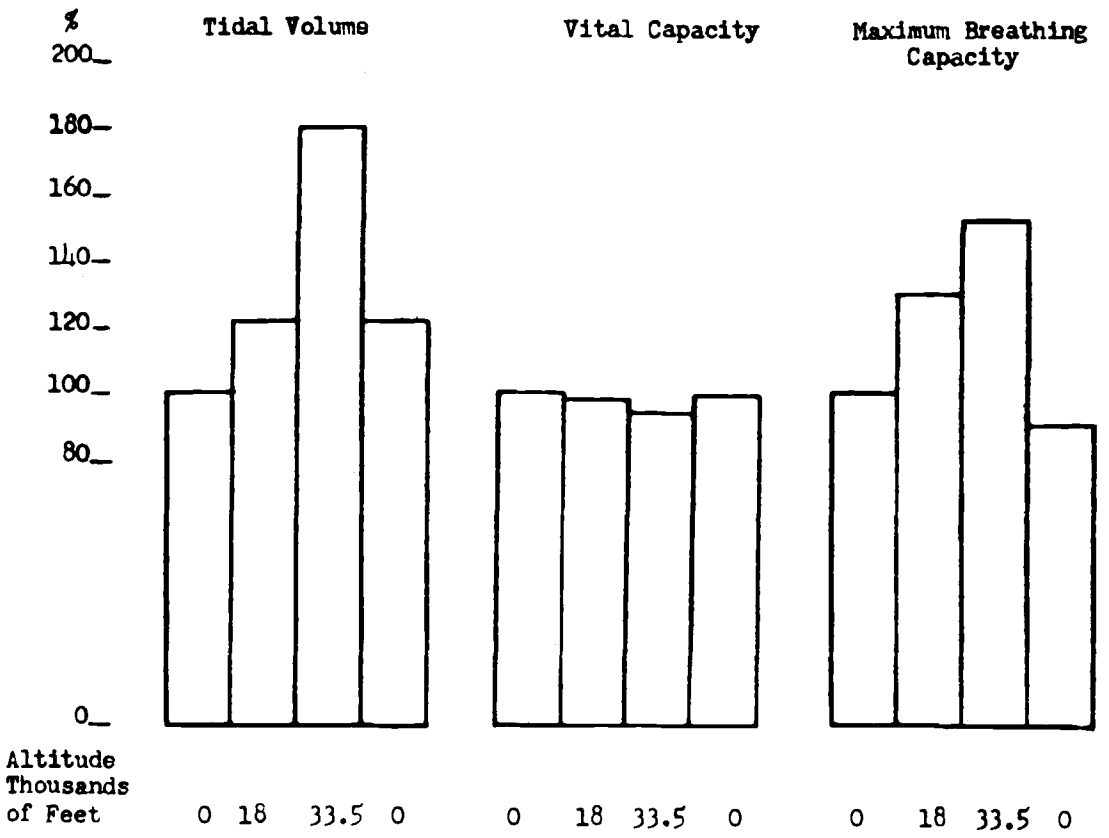

Fig. 6. Pulmonary function at altitude.

TABLE II. HEMATOCRIT AND PLASMA OSMOLAR CONCENTRATION

Subject	Duration of Flight	Test	Pre-flight	Flight	Post-flight
B	17 days	Hematocrit, % Plasma Osmolarity, mOsm/L	47 280	45 283	44 282
B	30 days	Hematocrit Plasma Osmolarity	48 287	44 281	49.5 292
A	17 days	Hematocrit Plasma Osmolarity	44 282	42 292	38 288
A	30 days	Hematocrit Plasma Osmolarity	44 290	40 289	40 294

tempt is now being made to obtain the necessary data to elucidate this problem by further simulator experimentation.

PULMONARY FUNCTION

We have been able through the use of a servo-spirometer within the space-cabin simulator to follow vital capacity (total and timed), tidal volume and maximum breathing capacity. Results of these studies at sea level, 18,000 feet and 33,500 feet are shown in Figure 6.

A slight reduction in total vital capacity at altitude was noted throughout the experiments. Several investigators have noted the reduction in vital capacity which occurs at simulated altitude and have distinguished this effect from that

due to hypoxia. Becker-Freyseng and Clamann[4] conducted a study at 30,000 foot altitude with 100 per cent oxygen for 3 days in which a sharp reduction of vital capacity occurred immediately observed increases are due to the reduced air density and frictional resistance at altitude. When an analysis of timed vital capacity is made the effect of reduced density and friction is

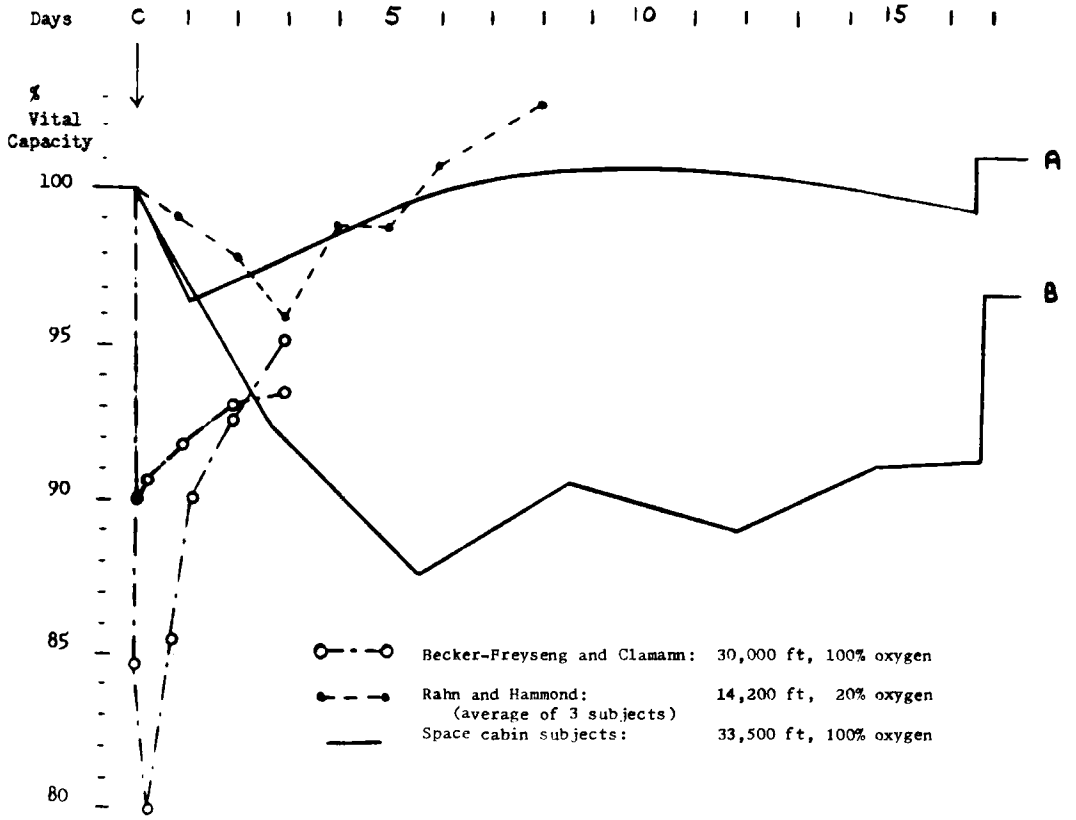

Fig. 7 Reduction in vital capacity after exposure to altitude.

followed by a slow recovery (Fig. 7). A similar study at Mt. Evans on three acclimatized men is reported by Rahn and Hammond[9]; an initial decrease in vital capacity is noted with recovery over an eight-day period. For comparison our results for 17 days at 33,500 feet in a 100 per cent oxygen atmosphere are presented and the pattern is again apparent.

An increase in tidal volume and maximum breathing capacity (MBC) was seen at altitude (Fig. 6); the magnitude of the increase appears to be directly related to the simulated altitude. Since tidal volume and MBC measurements are highly flow dependent it is probable that the again evident (Fig. 8). Here the volume of air moved in the first second of effort is greatest at 33,500 feet, intermediate at 18,000 feet and least at sea level.

OXYGEN TOXICITY

A primary purpose of the 17-day experiment was the testing of man's ability to tolerate a pure oxygen atmosphere at reduced pressure. Comroe *et al*,[5] Bean[3] and Becker-Freyseng and Clamann[4] have documented the deleterious effects of oxygen at partial pressures in excess of 400 mm. Hg. Symptoms of oxygen poisoning (Table III) occur under these conditions within

48 hours and become severe, usually progressing to death from atelectasis, pulmonary irritation and effusion and direct central nervous system toxicity unless the subject is removed from the oxygen-rich environment.

TABLE III. SYMPTOMS OF OXYGEN TOXICITY
(After Comroe et al[5])

Symptom	Incidence Per Cent
Substernal pain	82
Lower respiratory tract	
Reduced vital capacity	79
Upper respiratory tract	
Cough	54
Nasal congestion	43
Sore throat	32
Ear discomfort (Aural Atelectasis)	25
Unusual fatigue	25
Eye irritation	23
Others	
Pains in muscles or joints, Paresthesiae, Dizziness, Aching teeth	<10

day, it is possible that the drying effect on the mucous membranes was initiated in this way. Cabin relative humidity reached normal levels in about 24 hours, nevertheless symptoms continued for about 72 hours longer. Minimal paresthetic sensations were noted in the calves and arms during the first five days; no neurological signs were elicited. Ear discomfort (aural atelectasis) occurred early and persisted throughout the flight. Valsalva maneuvers were required to clear the ears about every two hours due to absorption of oxygen from the middle ear.

The reduction in vital capacity has been described above; cough and labored breathing were not noted but the effect of reduced air density on voice pitch and ease of respiratory motion was pronounced. The problem of atelectasis due to absorption of oxygen from alveoli behind obstructed bronchioles was investigated by x-ray immediately post-flight and

TABLE IV. TIMED VITAL CAPACITY AT 33,500 FEET SIMULATED ALTITUDE

	Subject B	Number of Tests	Subject A	Number of Tests
Total volume	(Liters)		(Liters)	
First test	4.79±.17[1]	15	5.29±.24	14
Second test	4.82±.27	15	5.37±.27	14
One second volume	(Per cent)		(Per cent)	
First test	82.9±6.5	12	86.5±5.6	12
Second test	84.2±2.9	12	88.3±3.6	12

[1]mean ± 1 standard deviation

Preliminary animal studies in this laboratory indicated that a 100 per cent oxygen atmosphere at reduced pressure (pO_2 176 mm. Hg) for three weeks was not deleterious. The 17-day exposure of two men to an oxygen atmosphere (pO_2 176 mm. Hg, equivalent altitude 33,500 feet) was then carried out. Preliminary denitrogenation was carried out to reduce or eliminate symptoms due to dysbarism. Complaints began to be voiced by the subjects almost as soon as altitude was reached. The first complaints were of dryness of the upper respiratory tract, nasal congestion and eye irritation; these symptoms appeared during the first day, were mild to moderate and improved markedly after the fourth day. Since dry gas was being introduced during the first by analysis of the timed vital capacity data. No x-ray evidence for atelectasis was seen. Two vital capacity measurements separated by a short time interval were made at each daily testing period during the flight. If significant atelectasis were present, the second measurement should have been greater both in total volume and first second volume due to opening of collapsed alveoli during the first vital capacity measurement. As can be seen in Table IV, no significant change was encountered suggesting that atelectasis, if present, was minimal.

On the ninth day of exposure, one subject noted the onset of mild, burning, retrosternal pain which increased on inspiration. This pain continued with slight increase in severity for

about 24 hours. Altitude was decreased to approximately 27,500 feet by *increasing* oxygen pressure (pO_2 244 mm. Hg) whereupon the subject's symptoms subsided completely within twelve hours and did not recur on regaining 33,500 foot altitude (pO_2 176 mm. Hg). He completed the flight asymptomatic. In oxygen poisoning such a course is unusual in the experience of Clamann[4] and Comroe[5] since substernal pain due to this cause, once established, almost invariably progresses until the oxygen tension is reduced.

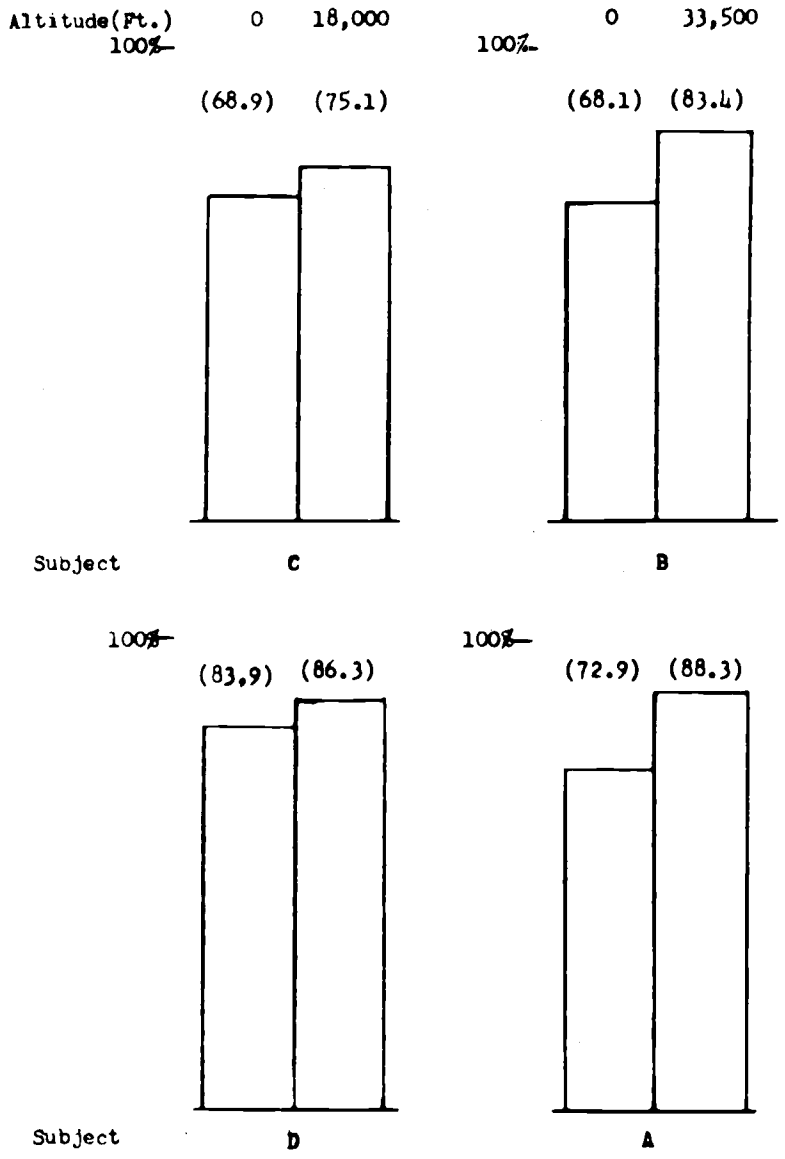

Fig. 8. Effect of air density on timed vital capacity.

SUMMARY

Certain physiologic aspects of the sustained high altitude exposure of men in the SAM two-man space cabin simulator have been discussed. Special emphasis has been placed on physical performance and work capacity, on problems of water balance and body composition, on changes in pulmonary function and on the potential hazards of oxygen toxicity in experiments on man in a "space-equivalent" environment. This experiment has served to clarify the problem areas for further work now in progress at the School of Aviation Medicine.

ACKNOWLEDGMENT

Total body water determinations were made by the antipyrine method by Dr. Robert Jenson, SAM Consultation Services and by the deuterium oxide method by Dr. James E. Roberts and Mr. William Lackay, SAM Department of Medical Sciences. Dr. Ralph J. Gorten, SAM Consultation Services, made the blood and plasma volume determinations. The authors are indebted to them for their cooperation. We are also grateful for the help of the many medical officers who have aided in the conduct of these experiments.

REFERENCES

1. ARMSTRONG, H. G.: Principles and Practice of Aviation Medicine. 2nd Ed. Baltimore: Williams and Wilkins, 1943.
2. BALKE, B.: Correlation of static and physical endurance. I. A test of physical performance based on the cardiovascular and respiratory response to gradually increased work. USAF SAM Project No. 21-32-004, Report No. 1, April 1952.
3. BEAN, J. W.: Effects of oxygen at increased pressure. *Physiol Rev.*, 25:1, 1945.
4. BECKER-FREYSENG, H. and CLAMANN, H. G.: Die Wirkung langdauernder Sauerstoffatmung in verschiedenen Hohen auf den Menschen. *Luftfahrtmedizin*, 7:272, 1943.
5. COMROE, J. H., DRIPPS, R. D., DUMKE, P. R. and DEMING, M.: Oxygen toxicity. *J.A.M.A.*, 128:710, 1945.
6. GORTEN, R. J. and STAUFFER, J. C.: A study of the techniques and sources of error in the clinical application of the external counting method of estimating cardiac output. *Amer. J. Med. Sci.*, 238:274, 1959.
7. GRAVELINE, D. E., BALKE, B., MCKENZIE, R. E. and HARTMAN, B.: Psychobiologic effects of water-immersion-induced hypodynamics. *Aerospace Med.*, 32:387, 1961.
8. LEWIS, R. A., THORN, G. W., KOEPF, G. F. and DORRANCE, S. S.: The role of the adrenal cortex in acute anoxia. *J. Clin. Invest.*, 21:33, 1942.
9. RAHN, H., and HAMMOND, D.: The vital capacity at reduced barometric pressure. AF Technical Report No. 6528, Aug. 1951.
10. SCHWARTZ, I. L., SCHACHTER, D. and FREINKEL, N.: The measurement of extracellular fluid in man by means of a constant infusion technique. *J. Clin. Invest.*, 28:1117, 1949.
11. SILVETTE, H.: Some effects of low barometric pressures on kinds of kidney function in the white rat. *Amer. J. Physiol.*, 140:375, 1943.
12. SOBERMAN, R., BRODIE, B. B., LEVY, B. B., AXELROD, J., HOLLANDER, V. and STEELE, J. M.: The use of antipyrine in the measurement of total body water in man. *J. Biol. Chem.*, 179:31, 1949.
13. SWANN, H. G., COLLINS, W. D., CLINE, J. K. and DERNEHL, C. U.: Negative water balances during exposure to low barometric pressures. *Science*, 96:588, 1942.
14. VAN LIERE, E. J., PARKER, H. S., CRISLER, G. R. and HALL, J. E.: Effects of anoxemia on secretion of urine in the dog. *Proc. Soc. Exp. Biol. Med.*, 33:479, 1935.
15. WELCH, B. E., MORGAN, T. E., JR. and ULVEDAL, F.: Observations in the SAM two-man space cabin simulator: I. Logistic aspects. *Aerospace Med.*, 32:583, 1961.
16. WENTZEL, A. D., IOCANO, J. M., ALLEN, T. H. and ROBERTS, J. E.: Determination of heavy water (HDO) in biological fluids by direct introduction of water into a mass spectrometer. *Phys. Med. Biol.*, 3:1, 1958.

Diagnostic Procedure for Amyloidosis

REX B. CONN, JR., M.D., and R. DOROTHY SUNDBERG, M.D., *Am. J. Path.*, 38:61-71, 1961

Amyloid disease can be diagnosed from sections of bone marrow obtained by sternal aspiration. Amyloid was found at autopsy in the marrow of 8 of 9 patients with amyloidosis. In 7, vessels were affected to a limited extent; marrow was replaced extensively in 1. In both primary and secondary amyloidosis, slight to moderate marrow plasmacytosis occurred and Russell's bodies were apparent in plasma cells in a traceable sequence of formation. The amyloid deposit first appeared in the small vessels of the medullary cavity and then spread into adjacent hematopoietic tissue, finally replacing much of the marrow.—From *Modern Medicine*, June, 1961.