U. S. Naval School of Aviation Medicine

U. S. NAVAL AIR STATION

PENSACOLA, FLORIDA

RESEARCH REPORT

THE EFFECTS OF DECOMPRESSION ON SUBJECTS REPEATEDLY EXPOSED TO 43,000 FEET WHILE USING STANDARD PRESSURE BREATHING EQUIPMENT: INVOLUNTARY HYPERVENTILATION DURING PRESSURE BREATHING AT 43,000 FEET

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SUMMARY

Thirty-six healthy young male Navy seamen were exposed to a simulated altitude of 43,000 feet for one-half hour a day for 20 consecutive work days. Continual anoxia photometer readings were taken on each subject to determine arterial blood oxygen saturations. X-rays, electrocardiograms and speech intelligibility tests were taken for each subject. This paper is a report of the incidence of involuntary hyperventilation in these subjects. Other physiological phenomena observed will be reported in other papers.

A total of 43 cases of involuntary hyperventilation were observed during 695 man-exposures (240.7 man-hours) at 43,000 feet.

The similarity of early subjective symptoms of hypoxia and hyperventilation is noted.

INVOLUNTARY HYPERVENTILATION DURING PRESSURE BREATHING AT 43,000 FEET

Several types of hyperventilation have been described in the literature. Some of these are:

1. Voluntary Hyperventilation occurs when the subject voluntarily overventilates. Symptoms which result depend on the composition of the gases breathed. Forced breathing of air results in tingling in the hands and sometimes in the feet[1], dizziness, faintness, perhaps coolness and tremors in various parts of the body and slight tightening of the muscles. Tetany varies from a slight stiffness of the muscles to well marked contractures.[2] CO_2 tensions have been measured as low as 5.3 mm. Hg in this type of hyperventilation.[3]

2. Passive Hyperventilation occurs when the subject allows his lungs to be ventilated by respirator or other means[5] to the point where CO_2 is blown off, alkalosis occurs, and symptoms similar to those found in voluntary hyperventilation appear.

3. Involuntary Hyperventilation at sea level.

a. Physiologic. It is probable that "real anxiety"[4] situations resulting in fight or flight reactions give rise to involuntary hyperventilation even in normal persons[2] at sea level pressures. There is considerable variation in alveolar gas composition in normal persons at rest.[6]

b. Pathologic. "Neurotic anxiety"[4] states can lead to involuntary hyperventilation with symptoms of light-headedness, dizziness, tingling of hands and feet, palpitation or heart consciousness and muscle cramps.[7] This is the type of hyperventilation most frequently seen by the clinician.[8]

4. Involuntary Hyperventilation at altitude. Hyperventilation is of practical significance at high altitude, where the physiologic effects of

hypoxia provoke overbreathing through reflex mechanisms.[8] Symptoms of hypocapnia are produced in healthy individuals if the alveolar CO_2 drops below 25 mm.[18,19] If hypoxia cannot be overcome by hyperpnea so that O_2 and CO_2 tensions both are low, the effect is worse than either condition alone.[20] Terror, extreme anger, severe pain, or other intense emotion introduced in addition to the hypoxic drive[2,9,12,13] can cause severe involuntary hyperventilation in normal persons at altitude. The effects due to hypoxia and psychogenic factors seem to be additive. If the two stresses cause naive subjects to hyperventilate involuntarily to the point that symptoms are produced, it is possible that greater anxiety is caused by the awareness of these conditions to the extent that additional hyperventilation results. This is a vicious cycle[14] that can progress to the point of unconsciousness.[10] However, there is considerable variation between persons in reaction to the effects of hyperpnea.[2]

The following conditions were present during this experiment: Subjects breathed oxygen under 10 inches of water pressure at 43,000 feet, which should give them the same blood oxygen saturation as breathing oxygen under ambient pressure at 40,000 feet.[10] or breathing ambient air from 10,000 feet[10] to 18,000 feet[2] (depending on CO_2 tensions in the alveolar air). Alveolar CO_2 tensions theoretically would be 40 mm. Hg at 40,000 feet on oxygen. Hyperpnea due to the hypoxic drive would reduce it to between 36 and 32 mm. Hg.[10,11] Thus a tendency toward hyperventilation is present at all times at the test altitude.

This paper is a report of the incidence of involuntary hyperventilation in normal young men exposed to a simulated altitude of 43,000 feet for one-half hour a day for 20 consecutive work days. Complete information on phenomena observed other than hyperventilation will be published in other papers.

PROCEDURE

Thirty-six young Navy seamen participated in the experiment. All were free from defects insofar as could be determined by physical and psychological examinations. They were given high altitude indoctrination similar to that given Naval Aviators. Subjects were put in the low pressure chamber in groups of six or less and allowed to preoxygenate for one-half hour before being taken to a simulated altitude of 43,000 feet.

Anoxia photometer, electrocardiograph, and communications equipment were attached to each subject. X-rays could be taken of the men at any altitude. Standard word lists to determine intelligibility were read by each man at altitude on each exposure. Some subjects were instructed to shout for periods of from 1 to 4 minutes per exposure to determine the effect of shouting on blood oxygen saturation and on the hyperventilation syndrome. A complete description of test procedures is given in another paper.[15]

Four criteria were used to designate involuntary hyperventilation:

1. Arterial blood oxygen saturation at least 85%. (An average of 86½% would be expected at 43,000 feet under 10 inches of water pressure.[10])

2. Arterial blood oxygen saturation increasing.

3. (Unrequested subjective statement of) symptoms of dizziness, tingling of extremities, carpopedal spasm, or muscle cramp.[7]

4. Observation of hyperpnea.

If hyperpnea was observed, the subject was allowed to continue until he complained of symptoms. If all four criteria were met, but symptoms were not severe, he was designated as having had "mild" hyperventilation, or Grade 1; if symptoms were more severe, including tetany, spasm, or cramps, he was designated as having had moderate hyperventilation, 2; if collapse was judged to be imminent and descent was necessary, he was designated severe, 3; and if unconsciousness occurred, 4 (Table 1). Grade 4 was avoided if possible, but symptoms were not recognized quickly enough on two occasions and the man proceeded to unconsciousness. Others probably would have proceeded to unconsciousness except for prompt action of the observers. The only corrective action taken was to tell the subject to hold his breath. This action was taken as soon as possible after the subject complained of symptoms; therefore, most subjects were not allowed to proceed past Grade 1 (Table 2). Some subjects took personal corrective action without outside help.

A total of 43 cases of hyperventilation were observed (Table 1) during 695 man-exposures (240.7 man hours) at 43,000 feet. Of 36 men exposed, 18 hyperventilated one or more times. (Table 1) Incidence according to severity reached is shown in table 2.

Table 2

Severity	1	2	3	4
Cases	28	5	8	2

A total of 676.3 man-minutes were spent in speech and shouting tests. In only one instance was hyperventilation noted during a shouting test; none was noted during the speech tests.

DISCUSSION

Inasmuch as all subjects participating in this study were interviewed and examined to be sure none were gross pathological involuntary hyperventilators, incidents of hyperventilation should have been due to the effects of altitude or breathing oxygen against pressure. In addition to psychogenic and hypoxic factors, Rice[16] suggests that an overdistended stomach

pressing on the diaphragm can produce pain, tachycardia, premature ventricular systoles and hyperventilation. Our subjects had distended abdomens (148 incidents of pain due to expansion of gas in the abdomen out of 695 exposures). However, statistical analysis of the gas pains in relation to hyperventilation in our subjects showed no significant correlation. Our data on gas pain are not differentiated as to whether pain occurred in the stomach or in the intestines. It is entirely possible that gas distention could be great enough to cause pain in the intestines without affecting the diaphragm.

HYPOXIA AND HYPERVENTILATION

Unfortunately, some of the arly subjective symptoms of both hypoxia and hyperventilation are similar enough to be confusing to the untrained person. Dizziness, faulty vision, depressed cerebration and sensory dullness are often symptoms of either. Hyperpnea is caused by hypoxia and can result in hyperventilation. Whether or not hyperpnea is cause or effect can be confusing to the naive subject especially when symptoms from the condition causing hyperpnea and symptoms resulting from the effects of hyperpnea are similar. These confusing symptoms coupled with the mental dullness associated with both hypoxia and hyperventilation might lead to a dangerous condition in an aircrewman who has nothing but subjective symptoms on which to base corrective action.

Cyanosis is a symptom of hypoxia which is occasionally experienced in hyperventilation.[2] More advanced symptoms peculiar to hyperventilation are numbness and tingling of the extremities, muscle spasm, and tetany.

It has been the experience of the author that dizziness and faulty vision are quite often the first and most consistent symptoms of both hypoxia and hyperventilation.

It seems possible that hypoxia and hyperventilation would have similar symptoms of central nervous system origin since there is indication that comparatively mild hyperventilation results in decreased blood flow to the cerebrum[17] of 33 to 35% of the control value.[5] The Bohr effect from lowered CO_2 possibly would result in a net loss of oxygen to the tissues.[14] Fenn[19] modifies this statement to the extent that loss of oxygen to the tissues results from hyperventilation only when arterial blood oxygen saturation is 93% or over. Under certain conditions it might be that central nervous system hypoxia does result from hyperventilation. No definitive reference has been found on this point, however.

In some subjects it was noticed that there is a "point of no return" in the hyperventilation syndrome beyond which there is no tendency for the subject to cease hyperventilation short of unconsciousness. This is probably due to the vicious cycle of fear = hyperventilation symptoms = more fear = more hyperventilation. However, it is conceivable that a contributing factor could be the tissue hypoxia mentioned in the preceding paragraph.

It is possible that the incidence of involuntary hyperventilation has been overlooked by some of the persons who fly at high altitudes because of the similarity of subjective symptoms of hyperventilation and hypoxia. Unfortunately the symptoms of dizziness, blurred vision, and/or lightheadedness do not necessarily mean that the aircrewman is suffering from hypoxia. This point might be brought to the attention of persons who fly at high altitudes so that hyperventilation erroneously self-diagnosed as hypoxia will not result in increased ventilation.

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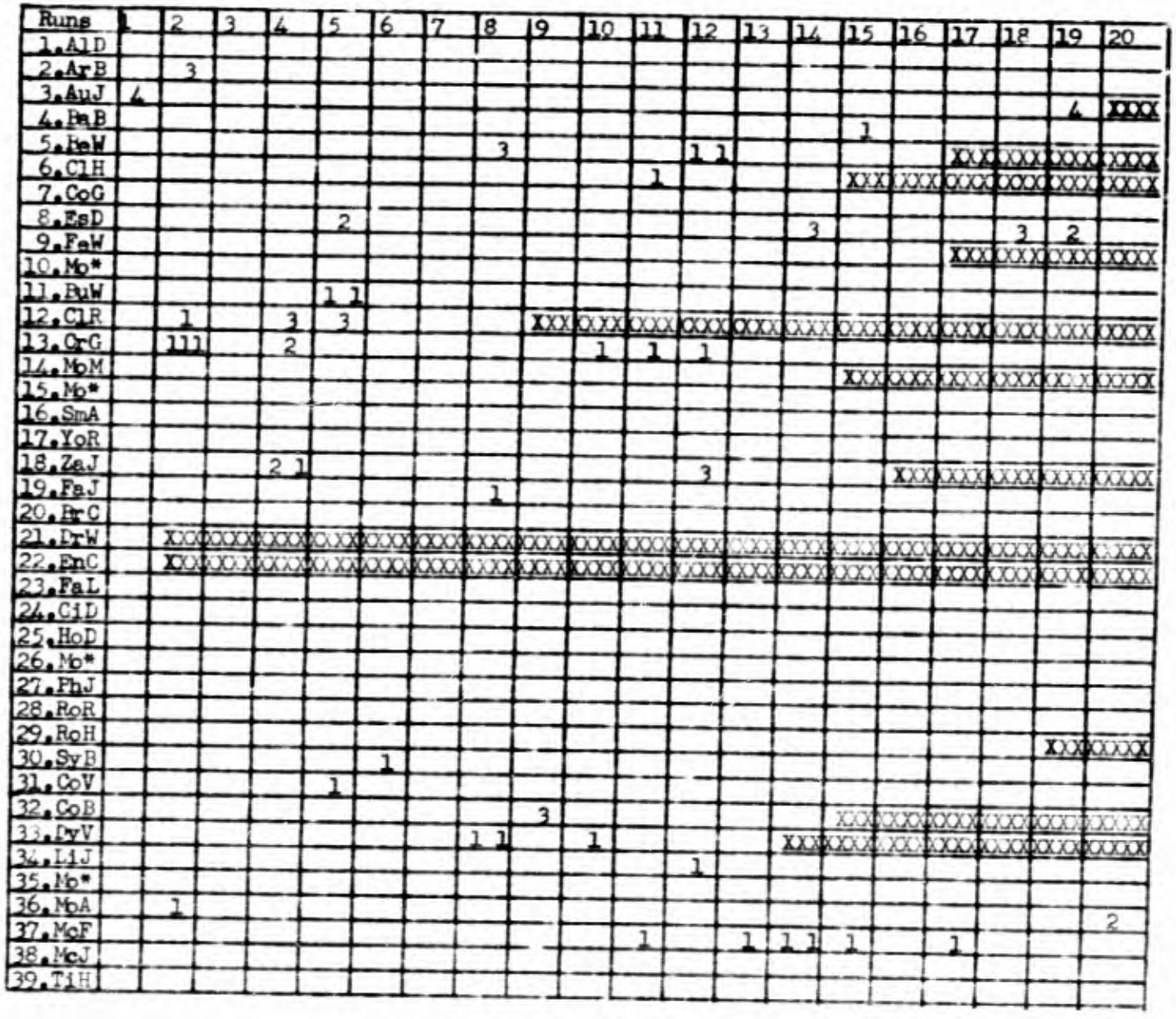

TABLE 1

Incidence and Severity of Involuntary Hyperventilation Observed in Indicated Human Subjects Exposed to a Simulated Altitude of 43,000 Ft.

LEGEND

MILD	1
MODERATE	2
SEVERE	3
UNCONSCIOUS	4

* Mo was exposed a total of 82 times without hyperventilation.