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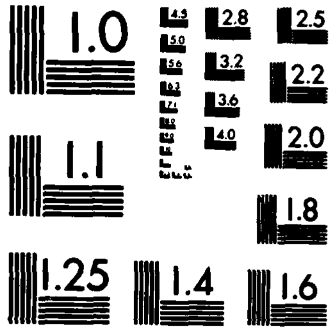

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NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

SUBMARINE BASE, GROTON, CONN.

MEMO REPORT 84-5

POSITION PAPER:

THE FEASIBILITY OF LOWERING OXYGEN CONCENTRATIONS
ABOARD SUBMARINES
IN ORDER TO IMPROVE FIRE SAFETY

by

Douglas R. Knight, CAPT, MC, USN

Released by:

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Commanding Officer
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Nitrogen serves as a retardant of fires by diluting the concentration of atmospheric oxygen. Submarine crews could gain long-term protection from fire damage by diluting their oxygen supply if that would not reduce the partial pressure of atmospheric oxygen (P_{O_2}) to the point of causing hypoxia. Since residents of Denver perform complex tasks at a P_{O_2} of 130 torr, submarine crews should be able to dilute their oxygen concentration until P_{O_2} falls to 130 torr. Consequently, oxygen con-

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centrations of 13-19% could be used to retard fires if the ship's barometric pressure were maintained at appropriate values within the range of 700-1000 torr. The risks of nitrogen narcosis and decompression sickness should not exist when barometric pressure ≤ 1000 torr. It is not known whether crew performance would be degraded by hypoxia since few experiments, if any, have evaluated the effects of long-term exposures to 130 torr oxygen, 1% carbon dioxide, and 10 ppm carbon monoxide. Nor is it known if the factors of an 18-hour work day, lack of sunlight, and the stressful life-style of submarine duty would interact with the oxygen-deficient atmosphere to degrade watchstanding. Crew health and performance in diminished concentrations of oxygen should be evaluated in the laboratory and at sea before the current standards of atmosphere control are redesigned to lower the risk of fire damage aboard submarines.

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SUMMARY PAGE

PROBLEM. Intentional dilution of the shipboard oxygen concentration can improve fire safety by retarding combustion. But, crewmembers may experience unknown problems when exposed to 19% oxygen in the contaminated environment of a nuclear submarine. Matters could be worse during transient reductions of oxygen concentration to 17%.

FINDINGS. Crew health and performance depend on the partial pressure of oxygen (P_{O2}) rather than oxygen concentration. A review of the literature indicated that chronic exposure to 19% oxygen is not harmful when the P_{O2} is 130-160 torr. The performance of complex tasks was observed to be quite adequate when men worked at altitudes above Denver, Colorado (where P_{O2} is 130 torr), and symptoms of acute hypoxia did not appear until P_{O2} dropped below 117 torr. Nor did carbon dioxide interfere with the rate of oxygen uptake by humans exposed to reduced P_{O2}'s. Laboratory studies did indicate that the performance of complex tasks and the visual adaptation to dark environments were slightly impaired at ambient P_{O2}'s of 130-140 torr. Furthermore, it was difficult to predict the effect of 10 ppm carbon monoxide on human performance in P_{O2} 130 torr. Therefore, laboratory and field investigations should attempt to show that submarine crew performance will not be degraded at P_{O2} 130 torr.

APPLICATION. Ships' engineers can diminish the risk of fire damage aboard submarines by reducing oxygen concentration below 19%.

ADMINISTRATIVE INFORMATION

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ABSTRACT

Nitrogen serves as a retard nt of fires by diluting the concentration of atmospheric oxygen. Submarine crews could gain long-term protection from fire damage by diluting their oxygen supply if that would not reduce the partial pressure of atmospheric oxygen (P_{O2}) to the point of causing hypoxia. Since residents of Denver perform complex tasks at a P_{O2} of 130 torr, submarine crews should be able to dilute their oxygen concentration until P_{O2} falls to 130 torr. Consequently, oxygen concentrations of 13-19% could be used to retard fires if the ship's barometric pressure were maintained at appropriate values within the range of 700-1000 torr. The risks of nitrogen narcosis and decompression sickness should not exist when barometric pressure $\leq$ 1000 torr. It is not known whether crew performance would be degraded by hypoxia since few experiments, if any, have evaluated the effects of long-term exposures to 130 torr oxygen, 1% carbon dioxide, and 10 ppm carbon monoxide. Nor is it known if the factors of an 18-hour work day, lack of sunlight, and the stressful life-style of submarine duty would interact with the oxygen-deficient atmosphere to degrade watchstanding. Crew health and performance in diminished concentrations of oxygen should be evaluated in the laboratory and at sea before the current standards of atmosphere control are redesigned to lower the risk of fire damage aboard submarines.

ACKNOWLEDGEMENT. The author appreciates the assistance of Mrs. Elaine Gaucher in obtaining the references used to conduct this review.

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Tables and figures

Terminology

Acclimatization. Short term adjustment to strange environments (43).

Acclimatization, complete. The total of the long term adjustments by organisms to maintain body weight, growth, fertility, and well being (43).

Accomodation. Short term adjustment to strange environments (43).

Acute Mountain Sickness. The reversible, ill effects of acute hypoxia.

Adaptation. Short term adjustment to strange environments (43).

Aerohypoxia. [synonyms: anoxic anoxia, hypoxic hypoxia (52)] The reduced partial pressure of atmospheric oxygen (21).

ATA. An abbreviation for "atmospheres of absolute pressure". One ATA = 760 torr.

Chronic Aerohypoxia. The sustained lack of oxygen to which an individual can accomodate by a complex process termed altitude acclimatization (27).

Critical Flicker Fusion Frequency. The lowest frequency, in cycles per second, at which a flickering light appears to be steady (82).

Critical Venous Tension. The minimum venous tension of oxygen which assures an adequate tissue oxygen tension for oxidative metabolism of cells (modified from ref. 41).

Gas Stores. Reservoirs of gases in the body (39).

HbCO. Carboxyhemoglobin.

HbO_2. Oxyhemoglobin.

Hypercapnia. The condition of an elevated P_{CO2} in the atmosphere or tissues.

Hypoxemia The condition of an abnormally low P_{O2} in blood (124).

Hypoxia.	[synonym: anoxia (52)] The condition of reduced oxygen tension in the body (43). A subnormal quantity of oxygen in the body (5).
Hypoxia, complete.	The sustained lack of oxygen to which an individual can accomodate by a complex process termed "altitude acclimatization" (27).
Hypoxidosis.	The impairment of aerobic metabolism (21).
Normoxia.	The ambient partial pressure of oxygen is 159 torr.
Oxygen pressure.	The partial pressure of oxygen in gas mixtures.
Oxygen tension.	The partial pressure of oxygen in tissues.
P_B.	The barometric pressure, as expressed in units of torr.
P_{O2}.	The partial pressure of oxygen, as expressed in units of torr.
P_{CO2}.	The partial pressure of carbon dioxide, as expressed in units of torr.
Torr.	A unit of pressure equal to $1/760^{th}$ of an atmosphere, or 1 mm Hg.

1. The question

Submarine crews would have more time to extinguish fires if the rate of combustion were retarded by low concentrations of oxygen (1). However, the Navy has been hesitant to permit the operation of submarines with reduced concentrations of oxygen. Members (2) of the COMMITTEE ON SUBMARINE FIRE PREVENTION AND CONTROL rationalized that diminished concentrations of oxygen would interact with background levels of carbon dioxide (ca. 1% CO_2) to degrade crew performance. The side effects could include impaired oxygen utilization, increased incidence of headaches, shortness of breath, and reduced peripheral vision (2). Therefore, an inquiry to the Bureau of Medicine and Surgery questioned the permissibility and physiological effect of maintaining oxygen levels at 19 $\pm$ 0.5%, with occasional reductions to 17% (3). The Bureau advised that unknown problems might result from long-term exposure to atmospheres containing 19% oxygen, trace contaminants, and 0.7-1.0% carbon dioxide. Furthermore, doubts were expressed concerning the capability of 19% atmospheric oxygen to significantly lower the fire risk in submarines (4). The final recommendation to the Naval Sea Systems Command was that submarine crews continue to operate in oxygen levels specified for nuclear powered submarines (6).

Current U.S. Navy directives require sufficient oxygen concentrations to maintain P_{O2} at 140-160 torr. Consequently, crews live in an atmosphere pressurized to 700-800 torr while breathing concentrations of oxygen below 21% (figure 1). The data on which these limits were established are not readily available. The policy of the Royal Navy is to limit decrements of P_{O2} to 137 torr aboard all submarines, since lower levels of oxygen are believed to cause deterioration of night visual acuity and decrement of mental performance (7). Naval officers know that nuclear submarines can operate effectively with oxygen

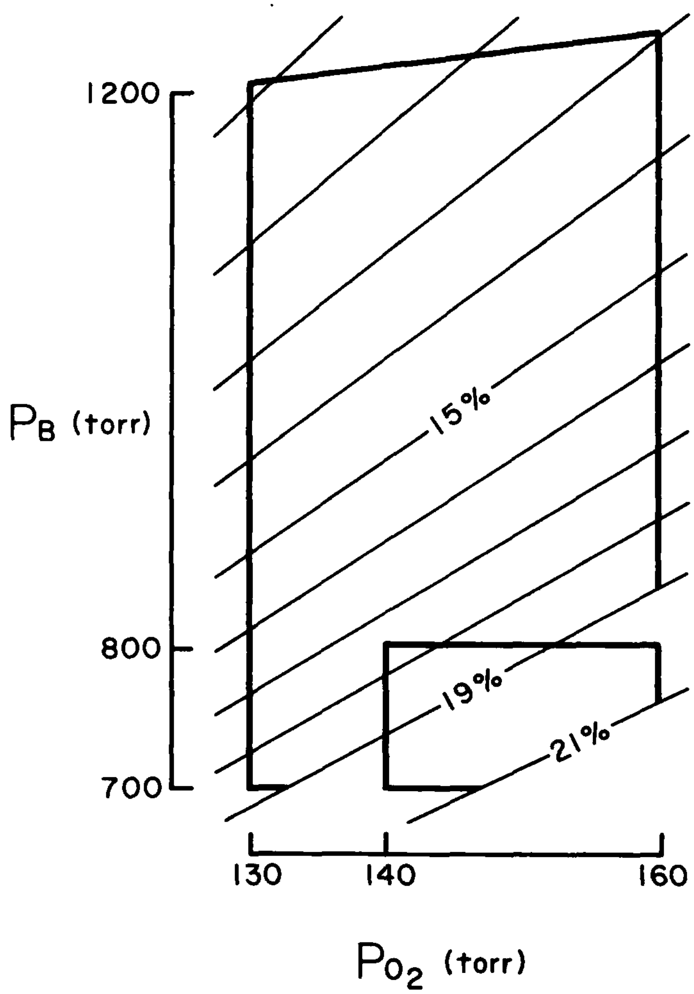

FIGURE 1: LIFE SUPPORT ZONE ABOARD SUBMARINES

Abscissa: sea-level P_{O2} is 159 torr; Denver's P_{O2} is 130 torr; and, P_{O2} at the U.S. Air Force Academy is 128 torr. Ordinate: sea-level pressure is 760 torr.

The heavily outlined pentagons depict two life support zones which are composed of nitrogen and oxygen. Submarine crews currently maintain their barometric pressure (P_B) and oxygen pressure (P_{O2}) within the perimeter of the smaller zone. The larger zone is designed to retard fires. The larger zone's upper border is an isobar for atmospheric nitrogen's partial pressure (P_{N2}) of 1.42 ATA. where; $P_{N2} = (P_B - P_{O2}) / 760$. The diagonal isopleths of oxygen concentration were constructed from the following formula; $\%O_2 = 100 (P_{O2} / P_B)$. The combustion of paper is incomplete when oxygen concentration is less than 15% (13).

concentrations close to 19%. For example, data from a 70-day cruise showed that mean oxygen concentration was 19.6%, with the 1^{st} and 99^{th} percentiles at 19.1% and 20.2%, respectively. Mean P_{O2} was 153 torr and mean P_B was 778 torr (figure 2). Other field studies indicated that submarine crews tolerate 17% oxygen with 3% carbon dioxide during prolonged rebreathing of the atmosphere (8,9,10, 11,12). The important question is, "What is the lowest concentration of oxygen permitting effective performance by submarine crews?"

2. Strategy of fire retardance aboard submarines

Ignition energy, flame size, and burning rate are dependant on oxygen concentration when barometric pressure is 1-1.5 ATA (1,13,18). Consequently, habitable atmospheres retard fires when oxygen is diluted with nitrogen and other noncombustible gases (19). The use of helium and CF4 for retardance is impractical from the aspects of cost and potential toxic effects on submarine crews. But dilution with nitrogen to 15% oxygen provides a considerable reduction in risk of fire damage. Class A fires burn incompletely in that environment (13) and kerosene will burn until the ambient oxygen concentration falls to 16% (10).

Scientists and engineers (7,13) use P_{O2} as the important parameter for assuring life support. Work at the Naval Submarine Medical Research Laboratory recently confirmed this principle by showing that men could live in 5% oxygen when hyperbaric nitrogen maintained P_{O2} at 160-228 torr (20). Therefore, one option of assuring crew performance is to maintain $P_{O2} \geq 140$ torr by diluting the oxygen concentration with hyperbaric nitrogen. A second option is to reduce P_{O2} below 140 torr by diluting oxygen with normobaric nitrogen. Submarine crews should be able to live in 130 torr oxygen since residents of Denver and the USAF Academy both live in an oxygen pressure of 128-132

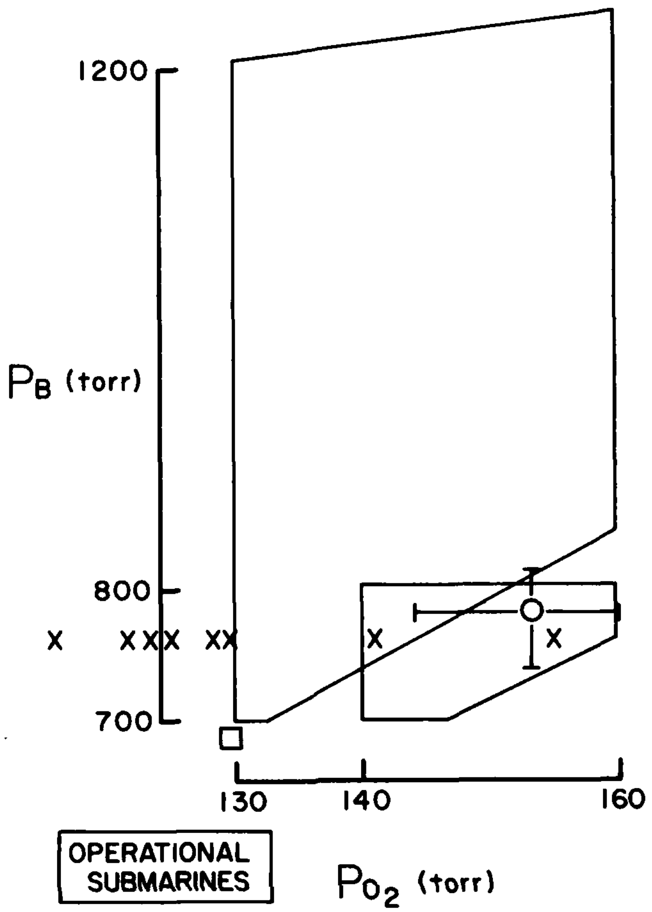

FIGURE 2: OXYGEN LEVELS ABOARD OPERATIONAL SUBMARINES

The pentagonal figures were explained in figure 1. Symbol "O" depicts the mean P_{O2} (153 torr) and mean P_B (778 torr) for 70 continuous days aboard a nuclear submarine. The bars indicate a range of values between the 1^{st} and 99^{th} percentiles of the tabulated data. The mean oxygen concentration was 19.6%, with the 1^{st} and 99^{th} percentiles at 19.1% and 20.2%, respectively.

Symbol "X" shows oxygen pressures aboard World War II submarines after prolonged submergences without replenishment of oxygen (8-12,14-17). The partial pressures of carbon dioxide were 14-30 torr when barometric pressure was assumed to be 760 torr. The times of exposure to 129, 141, and 155 torr oxygen were 38, 24, and 12 hours, respectively. Less than 1 hour was spent in oxygen pressures below 129 torr before changes were made in the oxygen concentrations.

Symbol ☐ represents the mean P_{O2} and P_B during 14 hours of snorkeling in a diesel-electric submarine (7).

torr. Fortunately, the concentrations and partial pressures of atmospheric oxygen can be regulated by the engineering plants of nuclear submarines and submersibles. It remains to determine whether crews perform well in 130 torr oxygen, since trace contaminants of the submarine atmosphere might impair oxygen transport to the brain (4).

Figure 1 depicts 2 zones of life support for submarine crews. The smaller zone is currently used to provide life support while protecting against conflagration. **The larger zone is proposed for retarding fires at sea.** The potential health hazards of operating in the larger zone are hypoxia, nitrogen narcosis, and decompression sickness.

3. Physiological Principles of Hypoxia

ACUTE AEROHYPOXIA. The physiological mechanisms of oxygen transport depend on a gradient of P_{O2}'s from sea-level air (P_{O2} 159 torr) to the brain (P_{O2} 1-15 torr) (figure 3). This gradient diminishes after transitions from sea-level to altitudes of 10,000 ft. or 22,000 ft. (21-23). Since cerebral metabolism is limited by the delivery of oxygen through arteries (24), humans tolerate sudden exposures to low P_{O2} by accelerating their ventilation and blood flow. Atmospheric P_{O2}'s exceeding 109 torr saturate 85% of the circulating hemoglobin with oxygen (25). Consequently, ventilatory and circulatory function are not stimulated by exposure of resting men to altitudes below 10,000 ft. (21). For example, at an altitude equivalent to 118 torr P_{O2}, arterial P_{O2} dropped 26 torr while oxygen saturation dropped only 4-7%. The 14% increment of minute-ventilation failed to reduce arterial P_{CO2} by more than 3 torr (26).

The physiological responses to altitude include shortness of breath, tachycardia, dizziness on exertion, light headedness, dimming of vision, and poor muscular coordination (27,28, table 1). Many of these responses

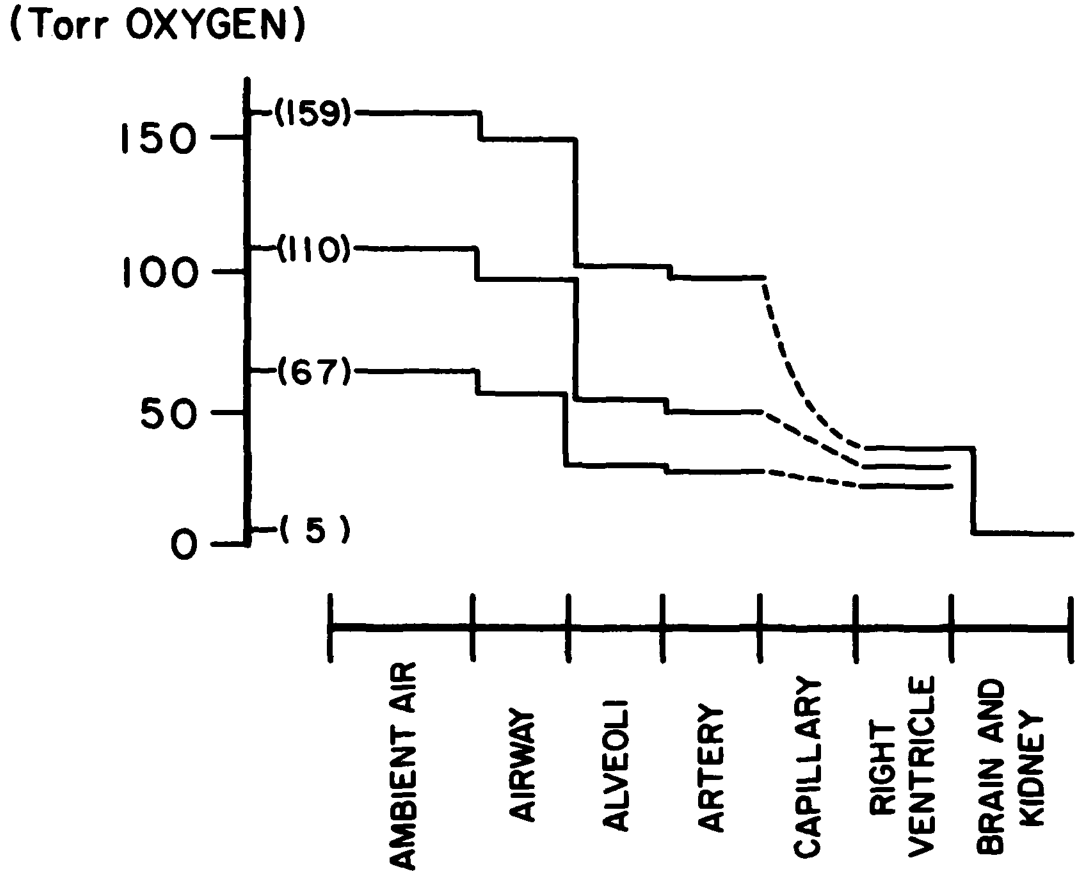

FIGURE 3. THE OXYGEN CASCADE

Oxygen pressures of 159, 110, and 67 torr respectively exist in air at sea-level, 10,000 ft. altitude, and 22,000 ft. altitude. The data for sea-level and 22,000 ft. altitude were adapted from figure 5 of reference 21. The data for 10,000 ft. were calculated from information in references 22 and 23.

TABLE 1. FUNCTIONAL DEGRADATION DURING ACUTE AEROHYPOXIA

Ref	Altitude, feet	Ambient P_{O_2}, torr	Effect
(21)	0-10,000	159-109	**INDIFFERENT PHASE**
(31)	4,000	136	Raised threshold for scotopic vision.
(21)	4,000	136	No change of respiration or blood circulation.
(29)	5,000	132	Impaired dark adaptation.
(31)	5,000	132	Impaired dual task performance.
(33)	5,280	130	Arterial P_{CO_2} 36 torr; arterial pH 7.41; minute ventilation 9 liters per minute.
(32)	0	129	Reduced night vision. Increased breathing volume. Tachycardia.
(25)	7,000	122	Deterioration of sensitive visual functions.
(32)	0	122	Dizziness. Impaired attention. Impaired judgment. Impaired coordination.
(32)	0	114	Intermittent breathing. Rapid fatigue. Loss of muscle control.
(21)	10,000	109	**COMPENSATED HYPOXIA**
(25)	10,000	109	Normal mental function. 85% saturation of arterial hemoglobin.
(29)	10,000	109	Potential onset of impaired concentration. Impaired short-term memory. Hyperventilation.
(34)	11,500	107	93% saturation of arterial hemoglobin. No decrement of vigilance. No change of heart rate or respiratory rate.
(35)	12,500	95	Tachycardia. Excretion of catecholamines unchanged.
(5)	13,000	93	Impairment of machinists' performance.
(32)	0	91	Very faulty judgment. Very poor muscular coordination.

			Loss of consciousness. Permanent brain damage.
(21)	15,000	89	**MANIFEST HYPOXIA**
(25)	15,000	89	Impaired hearing.
(29)	15,000	89	Potential onset of euphoria. Irritability. Hallucinations. Impaired critical judgment. Muscular incoordination.
(5)	18,000	80	Impaired tapping speed. Errors in performance of pursuitmeter task.
(36)	0	77	64% saturation of arterial hemoglobin. Slower response to reduced luminesence.
(5)	0	76	Loss of bone conducted hearing threshold.
(32)	0	76	Inability to move. Nausea. Vomiting.
(21)	20,000	75	**CRITICAL HYPOXIA**
(5)	20,000	75	Random performance of auditory localization task.
(29)	20,000	75	Potential for loss of consciousness.
(21)	23,000	61	Impaired writing.
(32)	0	46	Spasmatic breathing. Convulsive movements. Death in 5-8 minutes.
(37)	27,500	42	Rapid loss of logical thought.
(37)	35,000	27	Rapid deterioration of matching task.

[0 ft. altitude equals the absolute pressure of 760 torr.]

indicate impairment of function in the central nervous system despite attempts by the body to sustain the delivery of oxygen to neurons. The brain cannot function without oxygen since it derives energy from the oxidative metabolism of glucose (29). Mitochondrial NADH, a product of glucose catabolism, is the fuel for 80% of the oxygen consumption derived from glucose (30).

There are functional reservoirs of molecular oxygen in the alveolar-arterial compartment, venous blood, and cellular proteins which support metabolism during brief moments of oxygen deprivation (38-40). The shape of the oxyhemoglobin dissociation curve protects the arterial content of oxygen when reductions of alveolar P_{O2} fail to drop arterial oxygen tension below 40 torr. Reductions of arterial oxygen tension to the steep region of oxyhemoglobin's dissociation curve favor the release of oxygen to extravascular tissues. The mixed venous P_{O2} links cellular function to size of the body's oxygen reservoir (22). Measurements from selected organs indicated that cell function deteriorated when venous oxygen tension ($P_{v,O2}$) dropped below a minimum value called the "critical venous tension". Signs of brain malfunction appeared when cerebral $P_{v,O2}$ dropped below 19 torr (41). Myoglobin binds oxygen so tenaciously that only severly hypoxic muscle cells derive energy from cellular stores of oxygen (22,39,40).

It is generally believed that severe cerebral hypoxia impairs the production of ATP in brain cells. However, this mechanism may not impair learning and judgment when healthy men are exposed to moderate aerohypoxia. Tissue studies showed that the brain's high energy phosphates did not decline until arterial oxygen tension dropped below 35 torr. Moderate aerohypoxia may transiently impair metabolism of neurotransmitters in the brain, since animals experienced transient reductions of catecholamine synthesis in the brain when breathing 76 torr oxygen (29).

CHRONIC AEROHYPOXIA. The consequences of $\geq$ 2 weeks' exposure to high altitude are summarized in table 2. Oxygen uptake was not reduced in resting individuals who were acclimated to altitude (43). Chronic exposure to 15,000 ft. altitude (P_{O2} 89 torr) increased the blood volume (+25%), hematocrit (+55%), rate of iron turnover, myoglobin content, and respiratory minute ventilation. Residents of Denver also developed higher red blood cell volumes (+ 25%) and hematocrits than sea-level residents. The mild aerohypoxia (P_{O2} 130 torr) stimulated erythropoiesis without expanding plasma volume or raising the concentration of total serum proteins (48). These functional changes are believed to maintain diffusion of oxygen to tissues and cells in face of reduced diffusion gradients, resulting in improved work capacity at altitude (21,49).

Acclimatized individuals possess: (a.) higher pulmonary diffusion coefficients for oxygen, and (b.) greater effective diffusion areas in capillary beds. Biochemical changes increase the activity of the rate-limiting steps in cell respiration and improve storage capabilities of hemoglobin and myoglobin (43,27,50). Chronic hypoxia raised the tissue concentrations of DNA and high energy phosphate carriers, in addition to increasing cellular activity of glycolytic enzymes, succinoxidase, and the rate-limiting steps of respiration. The cardiac output, systemic blood pressure, and heart size did not change (27).

Long term survival without compensatory mechanisms may be possible when inspired P_{O2} exceeds 100 torr, assuming that a $P_{v,O2}$ of 30 torr is tolerable (22). Therefore, gradual reduction of oxygen pressure to 130 torr should allow time for development of homeostatic mechanisms to maintain normal brain function (29).

TABLE 2: CONSEQUENCES OF CHRONIC EXPOSURE TO HIGH ALTITUDE

Reference	System	Effect
42,27,43	Body Composition	Lower body fat content; Increased content of myoglobin in the heart and skeletal muscle; Increased content of high-energy phosphate carriers in the liver.
27,43,44	Pulmonary function	Hyperventilation; Higher alveolar P_{O2} and lower alveolar P_{CO2} than in unacclimated man.
43,27,45, 38,44	Circulation	No change of cardiac output during rest and submaximal work; Reduction of maximal cardiac output; Able to perform hard work; No change of arterial blood pressure; Increased venous pressure; Enlarged chambers of right heart; Increased vascularity of tissues; Increased red blood cell volume and hematocrit; No change of hemolytic index and increased concentration of total bilirubin; Increased turnover of iron.
44,46,47	Blood gases	Increased oxygen carrying capacity; Lower arterial oxygen tension; No improvement of the low $S_{a,O2}$ during sleep.
44,43	Blood acid-base	Compensated respi-

		ratory alkalosis; Reduced content of bicarbonate ion and reduced alkaline reserve.
43	Metabolism	No change of resting oxygen uptake.
43	Endocrine system	Transient hypertrophy of adrenal cortex.
43,44	Nervous system	Longer time of useful consciousness.
43	Gastrointestinal system	No delay in gastric emptying.
43	Reproductive system	Natives have normal fertility and reproduction.

4. Health effects of aerohypoxia

Submarine crews may experience accidental hypoxia in two situations. First, oxygen pressure can rapidly drop below 140 torr during snorkeling (7,14). For example, 7 men were incapacitated by hypoxia when the diesel engine dropped the barometric pressure in their compartment to 260 torr. Prompt remedial action by crew members in adjacent compartments prevented serious injury to the men (14). Second, failure of the engineering plant to replenish the ship's oxygen could asphyxiate an entire crew (8,51).

Acute mountain sickness occurs during the first few days of sudden exposure to aerohypoxia. A reduction of arterial oxygen tension must precede the symptoms of hypoxia (52) and many of the symptoms have been associated with respiratory alkalosis (27). Headache, nausea, vomiting, insomnia, and loss of apetite, develop at altitudes greater than 8,000 ft. (P_{O2}<117 torr; reference 28). However, patients with cardiovascular disease have experienced dizziness, nausea, and dyspnea upon sudden exposure to 6,000 ft. altitude ($P_{O2} \leq 127$ torr; reference 53).

Duration of consciousness can be shortened to 10 minutes at altitudes above 20,000 ft. ($P_{O2} \leq 75$ torr; references 52,29). Severe hypoxia caused irreversible effects when individuals were exposed to altitudes in excess of 28,000 ft. ($P_{O2} \leq 52$ torr) for longer than 5 minutes. Loss of consciousness may be followed by convulsions and death. Autopsies indicated circulatory failure, visceral congestion, and degenerative changes of ganglion cells in the central nervous system (27).

Acclimatization may involve adaptive diminution in the symptoms of acute mountain sickness as well as changes in physiological function. Adaptation to altitudes above 8,000 ft. can cause malaise, headache, insomnia, tachycardia, and dyspnea. Although these responses diminished with time, exercise seemed to enhance the process of acclimatization. Over acclimatization may lead to polycythemia and cor pulmonale (27).

5. Damage control in aerohypoxia

The ability to perform exercise in 130 torr oxygen is important when considering the work of controlling and repairing ship's damage. The exertion required to control flooding with hand pumps demands the consumption of oxygen at a rate of 1.9 liters/minute (54). Fire fighting may be characterized by bursts of heavy work during exposure to severe heat, with the uptake of oxygen estimated to be 1.9-3.0 liters/minute (55,56).

The exercising body compensates for reductions of ambient P_{O2} by raising minute ventilation and cardiac output above levels required for exertion in the normoxic environment. Heavy work at altitude tended to raise sea-level concentrations of muscle lactate and depress muscle content of high energy phosphates below resting levels (57). Relatively higher concentrations of blood lactate indicated that aerohypoxia retards the acceleration of aerobic metabolism at the onset of submaximal work (58,59), thereby lengthening the time required for subjects to achieve steady-state rates of oxygen uptake at submaximal work loads. However, aerohypoxia did not suppress the steady-state rate of oxygen uptake required for submaximal work (59).

The maximum rate of oxygen uptake was lowered in proportion to the degree of aerohypoxia, irrespective of whether the exposure was acute or chronic (58-60) (figure 4). Sudden exposure to 91 torr oxygen reduced maximal aerobic power by 28% (21,49). Exposure to 125 torr oxygen did not reduce maximal uptake of oxygen, but tended to reduce the duration of exhaustive work. Respiratory minute volume and blood lactate levels were higher during heavy

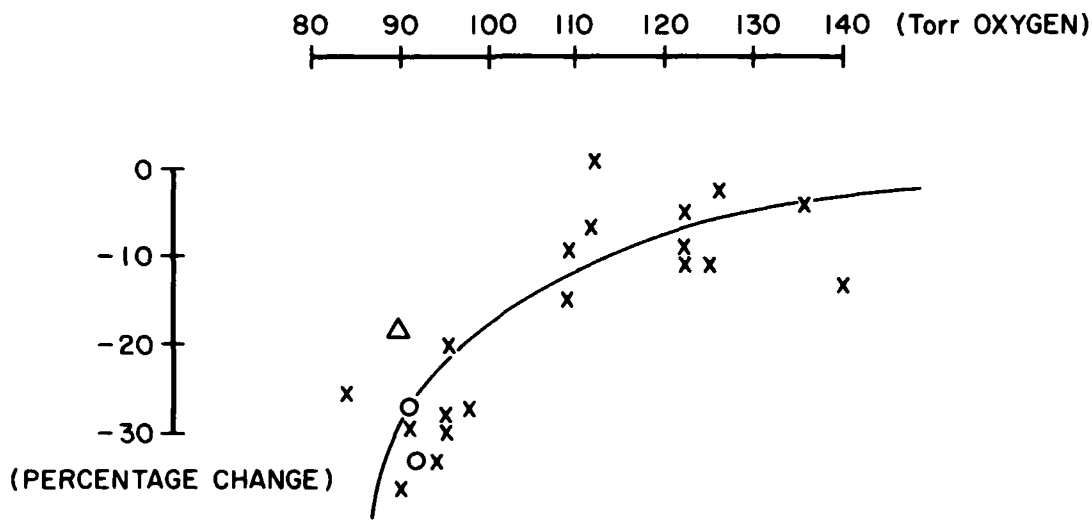

FIGURE 4: % CHANGE FROM SEA-LEVEL MEASUREMENTS OF MAXIMAL AEROBIC POWER

Acute and chronic exposures to aerohypoxia degraded the maximal aerobic power (ordinate) in proportion to the reduction of atmospheric P_{O2} (abscissa). SYMBOLS: "X" depicts exposures to hypobaric air or low concentrations of oxygen. "O" depicts the effect of inspired carbon dioxide on performance in aerohypoxia. △ demonstrates the effect of acetozolamide on exercise at altitude. The data in this figure, which were taken from references 62,61,60,59,63,64, 21,65,53,57, and 38, can be superimposed on older data published in figure 17-6 of reference 58.

exercise in 125 torr than in 159 torr oxygen. The lower P_{O2} blunted the rise of arterial $[H^+]$ during heavy work (61). Graded exercise in oxygen pressures of 124-140 torr (3280-6500 ft altitude) appropriately raised ventilatory rate in proportion to oxygen uptake (21,49). Linear regression analysis prompted Wagner, et al (60), to postulate that decrement of maximal aerobic power occurs when ambient oxygen pressure falls to 147 torr.

Figure 4 suggests that only 2 studies have evaluated the decrement of maximal aerobic power at 130-140 torr P_{O2}. The curve, which should intersect the abscissa at 159 torr oxygen, indicates that maximal aerobic power is degraded by less that 10% in 130 torr oxygen (60,38,58).

Impaired diffusion of oxygen at high altitude may limit oxygen transport by reducing the arterial oxygen tension (60). The reason for reduction of maximal aerobic power in oxygen pressures of 84-122 torr is not fully understood (60). Since mild reductions of arterial oxygen tension did not prevent saturation of arterial blood with oxygen, other mechanisms of oxygen transport were presumably impaired. Exercise capacity in moderate hypoxia may be limited by at least one of the following mechanisms: 1. Attainment of maximum cardiac output at lower work loads (59,60). 2. Recruitment of maximum ventilation at lower work loads (59). 3. Early depletion of muscle glycogen by anaerobic metabolism (59). 4. Acute reduction of plasma volume (60).

An elevated rate of anaerobic metabolism may reduce endurance time (63) and lengthen the time required to perform intense work. The endurance time of exhaustive work tended to fall in oxygen pressures of 122-126 torr and was significantly reduced in 91-109 torr oxygen (60,61).

The effects of aerohypoxia on repetitive bursts of work are not well documented. Heavy exertion may not be exhausting when performed in bursts of activity (58).

Competitive times in normoxic conditions were lengthened at altitude ($P_{O2} \geq 112$ torr) when athletes ran distances greater than 400 meters (53,58,64); but, the times for 400 meter runs were not lengthened. Competitive times were degraded when distances of 1500 meters, or longer, were run in 130 torr oxygen. At higher altitude, 121 torr oxygen prolonged the competitive times for distances of 800 meters (58). In all altitudes, single bursts of work are not degraded over 1 minute of maximal effort at altitude.

It is unlikely that 130 torr oxygen will impair the work of damage control by submarine crews. To summarize the previous paragraphs:

(a.) Maximal aerobic power was only reduced by 5% in 130 torr P_{O2}.

(b.) Aerohypoxia lengthened the time to achieve a steady-state rate of oxygen uptake, but did not suppress the oxygen uptake required for submaximal work.

(c.) The endurance time of exhaustive work was not significantly degraded at 130 torr P_{O2}.

(d.) Bursts of heavy work were not impaired in 130 torr P_{O2}.

Mild aerohypoxia is more likely to impair mental performance than manual labor, since the brain is more vulnerable to aerohypoxia than is skeletal muscle (22).

6. Field studies of watchstanding and psychomotor performance in aerohypoxia.

Navy policy required World War II diesel submarines to operate with oxygen concentrations $\geq$ 17% in order to prevent deterioration of mental work, judgment, and physical effort (11,12). There was also concern that the effects of oxygen concentrations < 17% might persist in the form of physical and nervous exhaustion (10,66). The Naval Submarine Medical Research Laboratory performed a series of field studies on the safe limits of respiratory gases aboard non-nuclear submarines (9). Crewmembers experienced malaise when exposed for periods of hours to concentrations of oxygen $\leq$ 19% combined with 3% carbon dioxide. Under these conditions, shortness of breath interfered with effective communications over the ship's internal communications system. Although crewmembers were able to learn new tasks, there were decrements of night vision, mental recall, and efficiency of physical exercise. The crews tended to feel ill during mild reductions of oxygen (129-133 torr P_{O2}) when associated with hypercapnia (23-30 torr P_{CO2}). Euphoria occurred when lower levels of oxygen (115-130 torr P_{O2}) were combined with 14-16 torr P_{CO2} (9). Symptoms of illness may have resulted from an interaction of atmospheric contaminants with low-level oxygen rather than from pure hypoxia. Today's diesel submarine crews seem able to tolerate mild aerohypoxia without distress. Crews of the Royal Navy have operated for 53 hours in oxygen pressures lower than 137 torr. Jolly (7) reported the successful operation of a diesel submarine for 17 hours with ship's oxygen pressures at 126-137 torr. Today's atmosphere monitoring capabilities and reduced levels of atmosphere pollutants may permit crews to tolerate aerohypoxia better than in World War II submarines.

Submariners may be able to perform well during exposure to oxygen partial pressures of 130 torr for up to 90 days since humans routinely perform complex tasks in Denver (P_{O2} 130 torr) and the U.S.A.F. Academy (P_{O2} 128 torr). Few studies, if any, have evaluated the performance of AWACS operators and air traffic controllers at altitude (67,68). However, the U.S. Air Force requires crews to perform in-flight missions while living for several days at a cabin-altitude of 7,000 ft. (P_{O2} 122 torr). Psychobiological measurements were performed on trained personnel during 30 continuous hours aboard the Advanced Airborne Command Post (69). Sixty-six crewmen maneuvered the aircraft, operated radios, maintained security of the

Airborne Command Post, performed repairs and cooked meals. Performance, safety, and crew morale were not compromised by the moderate fatigue at altitude.

The performance and output of machine operators was studied on-site at sea-level (159 torr oxygen), 9,000 ft altitude (114 torr oxygen), and 13,500 ft altitude (96 torr oxygen) (70). Machine tools were handled with nearly equal efficiency during work in 114 and 159 torr oxygen. Deterioration of performance in 96 torr oxygen was indicated by longer production times, inaccuracy of dimensions, and poor quality of surface finish. Certain machine operations lengthened production time in aerohypoxia. Information feedback, via the eye, appeared to be misleading during work in 96 torr oxygen. For example, operators informed that the tool often appeared to be in contact with the workpiece before actual contact. Removal of visual control from the manufacturing process reduced fumbling, hesitation, and delay in machine operation. Removal of tactile control by installing a positive stop relieved the operator from judging accuracy. Productivity at altitude reached sea-level standards when the operator based his judgment on acoustic and tactile stimuli rather than visual stimuli (70).

7. Effect of aerohypoxia on task performance

Acute exposures to altitude degraded performance in proportion to the desaturation of oxygen from arterial hemoglobin. Tests of memory and other cognitive functions were not degraded until ambient P_{O2} fell below 130 torr. Reduction of alveolar P_{O2} from 100 to 50 torr degraded the score for hand steadiness (71). The performance of cognitive tests was clearly unacceptable when physiological tolerance of hypoxemia approached unacceptable limits (23,72). Marked deterioration of performance terminated in unconsciousness as arterial oxygen staturation dropped below 60% (52,25,72). Neuromuscular incoordination was indicated by; (a.) increased body tremor during slow-onset hypoxia (P_{O2} 46 torr), and (b.) increased anteroposterior sway during rapid-onset hypoxia (P_{O2} 76 torr) (73).

The threshold altitude at which performance of a task was degraded varied inversely with the complexity of a task (5,74). Environments with oxygen pressures of 130 torr modified performance of complex psychomotor tasks (31,75,76). Response times to complex tasks were longer at a simulated altitude of 7,000 ft. (P_{O2} 122 torr) than at sea-level or 14,000 ft. (P_{O2} 91 torr). The time for decision, not speed of response, prolonged the response times (77). Learned tasks were less sensitive to aerohypoxia than novel tasks (72,35). The learning of new tasks may be impaired during sudden exposures to 118-138 torr oxygen, indicating that performance of uniquely difficult tasks may be impaired at 130 torr oxygen (78,79,72). In the absence of pre-training, there was deterioration of information-reduction tasks at 7,000 ft. (80). Exposure to 5,000 ft. altitude (P_{O2} 132 torr) may impair task performance during learning of the task, but not after practice of the task (31). Ernsting (75) recommended that the hypoxia associated with breathing air at 6,000 ft. (alveolar P_{O2} 70 torr) represent the safe limit for aircrew performance during routine flight.

The causes for impaired performance at 7,000 ft simulated altitude (P_{O2} 122 torr) may be attributed either to features of experimental design, failure of the body to maintain convective transport of oxygen, or failure in stimulation of the sympathetic nervous system (77). The novelty of experimentation may arouse apprehension which impairs performance until adjustments are made to the aerohypoxia (78,77). This could explain why trained subjects performed better in altitude chambers than untrained subjects (78,77,31,75). Supplemental oxygen did not improve performance at 8,000 ft. simulated altitude (78), indicating that performance was not degraded by reduction of oxygen transport to the brain. The performance of exercise prior to work at 12,500 ft. altitude (P_{O2} 99 torr) improved results of a tracking test (35).

8. Responses of the special senses to aerohypoxia

Degradation of sensory function is considered to be a reliable indicator of hypoxia. Measurements of distance judgment, range of the visual fields, accomodation, convergence, and retinal sensitivity, indicated no impairment of visual function during exposure to P_{O2}'s < 135 torr (72). Although impairment of dark adaptation began at P_{O2} < 138 torr, the magnitude of change may not be of practical concern until $P_{O2} \leq 110$ torr (75,21,25,31). Abrupt exposure to simulated high altitude degraded visual contrast discrimination when alveolar P_{O2} dropped from 100 to 50 torr (71).

Exposure to P_{O2} 93 torr impaired [the rate of] dark adaptation to green light, but not red light. The sensitivity-threshold for green light was elevated above normoxic values after the 3_{rd} day of continuous exposure to altitude. The decrement of dark adaptation was changed by transient elevation of P_{O2} to 108 torr, but not by exposure to P_{O2} 93 torr for 16 days. Although the investigators concluded that supplemental oxygen is necessary for essential operations involving night vision at altitude, their data (81) showed that 93 torr P_{O2} delayed, but did not prevent, dark adaptation. Aerohypoxia exerted a differential effect on the function of rods (dark vision) and cones (color vision), since sensitivity to color vision was degraded above 16,000 ft. altitude. Aerohypoxia may interfere with mediation of the visual process by the brain rather than with photochemical receptor activity (5,81).

Taste perception was not changed when P_{O2} exceeded 101 torr (5). Auditory function may be more resistant to aerohypoxia than visual function. Low frequency hearing loss was observed at P_{O2} < 75 torr, irrespective of ambient noise levels, but hearing sensitivity was improved at higher sound frequencies (5,82).

9. Sleep in aerohypoxia

Fragmented sleep was of restorative value, and prevented severe fatigue, when aircrews continuously operated at a cabin altitude of 7,000-8,000 ft. (122-117 torr P_{O2}) for 30 hours. Subjective fatigue was reported a few hours after completion of the mission and uninterrupted sleep was restorative (69). Whether or not mild aerohypoxia fragmented the crew's sleep at altitude could not be determined (69).

Insomnia is a complaint of tourists sojourning to altitudes of 8,000-12,000 ft. Humans at Pike's Peak experienced slight decrement in duration of deep, slow wave sleep, vairable decrement in REM sleep, and a higher number of arousals (83). Informal inquiries (84,85) led to speculation that healthy men experience restful sleep at Denver's low altitude, although sojourners might experience insomnia the first night (85).

The hyperventilatory response to high altitude imposes a state of respiratory alkalosis on healthy sojourners. A change in ventilatory pattern to periodic breathing may cause insomnia by fragmenting the sleep (84,86). The threshold P_{O2} for fragmented sleep may occur below 115-130 torr since hyperventilation and hypoxemia do not occur when P_{O2} is 115 torr (26,87,84). For example, bedrest in 118 torr oxygen reduced the arterial tension of oxygen by 26 torr without widening the arterial-alveolar difference in oxygen tensions. Decrement of hemoglobin saturation by 4% was inconsequential at this level of hypoxia (26). "Hypoxic insomnia" was observed in rats exposed to oxygen pressures below 115 torr (87,83). Time of wakefulness was prolonged when there was shortening of paradoxical (REM) sleep. The total time of slow wave sleep was unchanged, but most episodes of slow wave sleep were shortened by transitions to the awake state (87). Exposures to either 81 torr oxygen or 500 ppm carbon monoxide practically removed REM stage sleep and shortened the total time spent in non REM sleep. Acclimatization to aerohypoxia restored the non REM sleep within 2 weeks, but REM sleep was not even restored after 4 weeks. Partial recovery of sleep patterns may have resulted from improvement in arterial transport of oxygen. Full recovery did not occur until 24 hours after return to normoxia (83). Pappenheimer (83) postulated that humans experience unrestful sleep at altitude as a result of reductions in REM sleep and slow wave sleep.

10. Atmosphere contaminants in aerohypoxia

CARBON DIOXIDE. Figure 5 shows changes of physiological and psychological functions when humans were exposed either to low levels of oxygen (P_{O2} 20-90 torr, P_{CO2} 0-1 torr) or high levels of carbon dioxide (P_{O2} 115-139 torr, P_{CO2} 26-38 torr). Hypercapnia (P_{CO2} 18-38 torr) was observed to protect from changes of psychological function during exposure to aerohypoxia (P_{O2} 20-90 torr). Therefore, figure 5 raises questions of when and how ambient CO_2 protects human performance during exposures to aerohypoxic environments.

Hypercapnia may interfere with visual function at the periscope, since 3% carbon dioxide reduced night vision and visual sensitivity to green light (95). Five percent carbon dioxide reduced the critical flicker fusion frequency threshold (5). Residence in 23 torr carbon dioxide caused euphoria for 36 hours, followed by depression for 6 days. Other side effects included insomnia and deterioration of task performance (89). Retention of carbon dioxide in the body was favored by the

```
(AMBIENT  P_CO2,  torr)

      26.0+     A                        B     $
          I
          I
          I
          I
      18.4+     $                        $     $
          I
          I
          I
      12.2+
          I
          I
          I
       6.1+                        $     $
          I
          I
          I
        .0+     B                  $     $          A

             ++-------+------+------+-------+------++
             20      50     80     110     140    170

                    (AMBIENT  P_O2,  torr)
```

FIGURE 5: THE EFFECTS OF AEROHYPOXIA AND HYPERCAPNIA ON HUMAN FUNCTION (N=153)

The scatterplot represents changes of health (n=7), nervous system function (n=35), ventilation (n=44), cardiovascular function (n=14), metabolism (n=7), exercise/integrated functions (n=27), and psychological tests (n=19). SYMBOLS: A indicates "no change" from measurements of function at sea-level. B, which depicts "change" from sea-level measurements, represents a decrement or improvement in function. $ shows the overlap of A and B. The data were obtained from acute and chronic exposures to submarines (88,7,14-17) or other experimental environments (43,31,61, 89-95,65).

reduced gradient of P_{CO2}'s between ambient air and the alveoli. Since alveolar ventilation was less effective in excreting carbon dioxide when ambient P_{CO2} was 21 torr, the customary drop of arterial P_{CO2} did not occur during heavy exercise (91).

Respiratory acidosis was compensated after 5 days in 23-30 torr carbon dioxide, compared to 23 da in 11 torr carbon dioxide (89,90). This suggested that renal mechanisms compensated acidosis more quickly in higher levels of atmospheric carbon dioxide than occur in the submarine atmosphere. Compensated acidosis was characterized by elevated concentrations of bicarbonate ion in the cerebrospinal fluid and arterial blood. Constant hyperventilation explained the constancy of carbon dioxide tensions in cerebrospinal fluid and arterial blood (90,88).

Rapid reduction of arterial pH, and elevation of arterial P_{CO2}, displaced oxyhemoglobin's dissociation curve to the right (96,97). However, guinea pigs experienced leftward shift of the curve after 6 hours of exposure to high levels (114 torr) of carbon dioxide. The dissociation curve returned to its pre-exposure position after the third day of continuous exposure, when the guinea pigs experienced compensated respiratory acidosis (97).

It was postulated that chronic exposure to elevated concentrations of carbon dioxide impairs oxygen assimilation either by reducing the delivery of oxygen to tissues or impairing cellular utilization of oxygen. There was a divergence of opinion on whether leftward shift of the oxyhemoglobin dissociation curves aided or hindered delivery of oxygen to the cells. Schaefer (98) argued that leftward shift enhanced oxygen uptake in the lung, subsequently providing more oxygen to tissues as splenic contraction raised the red blood cell volume. Albers (41) suggested that leftward shift of the dissociation curve promotes hypoxia of the brain by narrowing the arterio-venous, oxygen-content difference at the critical tension of oxygen in cerebral venous blood. Respiratory acidosis may impair cellular utilization of oxygen since biochemical studies showed that reduction of intracellular pH impeded NADH synthesis. Although the logarithm of oxygen uptake was proportional to arterial pH in anesthetized dogs (41), respiratory acidosis may not impair oxygen assimilation in man. Chronic exposures to carbon dioxide (11-21 torr) did not alter oxygen uptake at rest or work (88,91). Nor was thermoregulatory elevation of oxygen uptake inhibited by the inhalation of 4% CO_2 (99).

Hypercapnia may protect individuals from incapacitation during exposures to aerohypoxic environments. In the early part of this century, DuBois (10) predicted that fainting would not occur in strong individuals during the forced submergence of a submarine crew until atmospheric oxygen reached 9%. If composition of the ship's atmosphere were maintained above 17% oxygen and below 2% carbon dioxide, until shortly before the end of a long submergence, then little danger should result from brief exposures to 3% carbon dioxide in 12-14 % oxygen before ventilating the boat (66).

At altitude, the performance of psychomotor tests was degraded by hypocapnia (71) and improved by hypercapnia (100). Hypoxic humans and subhuman primates improved their performance of psychomotor tasks when inhaling 3-5 % CO_2 (92,93,101). Inspiration of 4% CO_2 favored saturation of human hemoglobin with oxygen by raising the alveolar P_{O2} and respiratory minute volume. For example, supplemental carbon dioxide raised alveolar P_{O2} by 13 torr when ambient P_{O2} dropped from 159 to 132 torr (100).

During World War II, Gibbs et al (92) postulated that carbon dioxide aided survival of hypoxia in sealed compartments. Sudden exposures to reduced P_{O2} (<70 torr) required high levels of atmospheric P_{CO2} (23-38 torr) to prevent dizziness, maintain cerebral performance, and preserve an ordered electroencephalogram. This indicated that carbon dioxide approaches the importance of oxygen with respect to brain function when hemoglobin saturation approaches 30% in jugular venous blood (92,93). Naval scientists (11) conducted a series of human exposures to sealed environments in order to determine the highest percentage of carbon dioxide compatible with sustained efficiency during reductions in percentage of oxygen. Carbon dioxide was expected to render the ambient oxygen more effective in the lung by raising the ventilation; furthermore, the drop of pH in arterial blood should release oxygen from red blood cells for diffusion into extravascular tissues. The confinements reduced atmospheric oxygen concentration and raised atmospheric carbon dioxide concentration according to the respiratory exchange ratio's of the subjects (9,23). The crews safely tolerated 17% oxygen and 3% carbon dioxide for periods as long as 72 hours (8-11). Gradual exposure to 13% oxygen and 5% carbon dioxide caused headaches in 20% of the subjects, occasional nausea, and some impairment of performance. Symptoms of headache and dyspnea sharply increased during exercise when ambient concentrations of carbon dioxide exceeded 5% (11). Although 13% oxygen - 5% carbon dioxide could be tolerated without incapacitation, impairment of combat efficiency prevented the U.S. Navy from permitting such exposures aboard submarines (12).

Carbon dioxide may protect performance by supporting oxygen transport to the brain. First, carbon dioxide enhances the hypoxic stimulation of ventilation. At the same rate of oxygen consumption, hyperventilation improved the saturation of arterial blood by raising the alveolar P_{O2} (21,92,94). Second, carbon dioxide may enhance venous return to the heart by improving skeletal muscle tone in the legs (93). Third, CO_2 enhances cerebral blood flow by dilating the cerebral vasculature (93). Fourth, CO_2 may increase brain tissue P_{O2} by causing rightward shift of the oxyhemoglobin dissociation curve (92,65).

The running activity of rats was improved when carbon dioxide was added to an aerohypoxic environment (45). Furthermore, fasting rats did not suffer from fatalities, adrenal cortical hypertrophy, and accumulation of glycogen in the liver when inhaling supplemental carbon dioxide during severe aerohypoxia. The protective effect of supplemental CO2 was postulated to result from prevention of hypocapnia and alkalosis (102,103). The protective effect of carbon dioxide was absent in the extreme case where confined rats gradually depleted their supply of oxygen. At respiratory arrest, no correlation existed between lethal P_{O2} (21-38 torr) and environmental P_{CO2} (0-117 torr) (104).

Experimental evidence (61,94,65,105,99) indicated that carbon dioxide does not interfere with the assimilation of oxygen by human subjects. Aerohypoxia and hypercapnia affected the resting metabolism of patients in opposite, additive ways. Twenty minutes of aerohypoxia (P_{O2} 76 torr) reduced arterial blood's oxygen saturation from 93% to 59%. The slight decrement in rate of oxygen uptake, 0.01 liters/min/m^2, was accompanied by increased concentrations of serum lactate. Twenty minutes of hypercapnia (P_{CO2} 38 torr) caused a slight increment in rate of oxygen uptake (0.01 liters/min/m^2) and decreased the arterial blood pH without changing arterial oxygen saturation (105). The slight increment of oxygen uptake in hypercapnia may be attributed to extra work of breathing (99). Combination of aerohypoxia (P_{O2} 61 torr) with hypercapnia (P_{CO2} 30 torr) buffered the drop in oxygen saturation of arterial blood and prevented change in the rate of oxygen uptake. Nor were there changes in concentration of serum lactate and pH of arterial blood (105).

Adams and Welch (61) postulated that duration of performance is limited by a critical intracellular concentration of hydrogen ions rather than availability of oxygen. Aerohypoxia presumably speeds the rate of hydrogen ion accumulation in cells as a by-product of anaerobic glycolysis (61). If intracellular acidosis limits performance of exercise, then administration of carbon dioxide may impair performance of exhaustive work during aerohypoxia by raising tissue tensions of carbon dioxide. Higher tissue levels of carbon dioxide would in turn raise tissue concentrations of hydrogen ion. This prediction was examined during performance of graded exercise on the fifth day of residence in 92-95 torr oxygen. Although duration of exhaustive exercise was not reported, the addition of carbon dioxide did not enhance hypoxic impairment of maximum oxygen uptake (65). Arterial pH was lowered 0.06 units with the addition of 16 torr CO_2, suggesting that intracellular pH might also be lowered. Since PCO2 16 torr did not alter maximum aerobic power in 92-95 torr oxygen, carbon dioxide did not impair endurance of heavy work in reduced pressures of oxygen (65,94).

CARBON MONOXIDE. Trace amounts of carbon monoxide enter the bloodstream from biochemical conversions of heme molecules and methylene chloride; but, larger amounts may diffuse into blood across the alveolar membrane during exposure to polluted air. In either case, oxygen transport is impaired when carbon monoxide competitively binds the oxygen-binding sites of reduced hemoglobin to form carboxyhemoglobin (HbCO). Blood's release of oxygen is impaired when there is sufficient HbCO to shift the oxyhemoglobin dissociation curve to the left. A third mechanism by which carbon monoxide blocks aerobic metabolism is to bind mitochondrial cytochrome a3 (106, 107).

The formation of HbCO in-vitro depends on P_{CO}, P_{CO2}, and temperature. According to Haldane's first law (equation 1), the concentration of HbCO in total circulating hemoglobin depends on the ratio of arterial P_{CO} to arterial P_{O2}.

eq. 1. Haldane's first law.

$$HbCO/HbO_2 = M\ (P_{CO}/P_{O2})$$

where, M is the ratio of relative affinities of hemoglobin for carbon monoxide and oxygen. It is assumed that P_{CO} and P_{O2} are sufficiently high to minimize the amount of reduced hemoglobin.

Carbon dioxide inhibited the association of carbon monoxide with hemoglobin at low P_{CO}'s by shifting the HbCO saturation curve to the right and transforming the curve to a sigmoid shape (106,107). In the intact animal, carbon dioxide may enhance the rate of formation of HbCO by stimulating pulmonary ventilation (108). Blood levels of HbCO may be higher in Denver because of the lower P_{O2}. Denver fire fighter (n=35) contained carboxyhemoglobin levels of 5%, or less, with only a few blood samples containing 5-6.3% HbCO. Half of the firemen were tobacco smokers (109).

Carboxyhemoglobin reduced maximal aerobic power by impairing the arterial transport of oxygen (110,111). Maximal rate of oxygen uptake fell from 4.4 to 3.8 liters/min when the hemoglobin content of HbCO was 13%. The arterial transport of oxygen was diminished by a 7% reduction of maximal cardiac output combined with a 5% reduction of arterial oxygen content (110). An increment of HbCO from 0.3% to 4.3% degraded maximal oxygen uptake from 3.7 liters/min to 3.4 liters/min, indicating an 8% decrement of maximal aerobic power. The reduction of aerobic power occurred irrespective of whether HbCO concentration was raised by continuously breathing 100 ppm carbon monoxide for 38 minutes or absorbing several boluses of carbon monoxide during the first 10 minutes of a 36 minute time period. It was therefore predicted that continuous inhalation of 24 ppm carbon monoxide degrades maximal aerobic power when HbCO reaches 4% (111).

In aerohypoxia, P_{O2} determined the final equilibrium value of HbCO withcut changing the initial rate of carbon monoxide absorption (106,112). The thresholds for decrement of maximal aerobic power were estimated to be 4% HbCO and 1,600 m altitude (P_{O2} 130 torr). Above these thresholds, decrement in maximal rate of oxygen uptake may be proportional to the increment in blood content of HbCO and decrement of P_{O2}. This was tested by measuring maximal aerobic power in residents of Denver, where ambient P_{O2} is 130 torr. A bolus of 37-53 ml of carbon monoxide raised their hemoglobin concentration of HbCO from 1.0 to 5.1%. The decrement of oxygen uptake, from 44.3 to 42.8 ml/kg/min, was judged to impair maximal aerobic power to the same extent as at sea level. Linear regression analysis showed the following relationship between decrement of maximal aerobic power ($-V_{O2,max}$) and hemoglobin content of HbCO ([HbCO]);

eq. 2. $$-V_{O2,max} = 1.1\,[HbCO] + 0.35$$

Residence at low altitude (P_{O2} 130 torr) did not enhance the reduction of maximal aerobic power resulting from rapid elevation of HbCO content. Carbon monoxide reduced the anaerobic threshold and raised the minute ventilation at heavy work loads (113).

The effects of carbon monoxide and aerohypoxia may be additive since both reduce the saturation of arterial blood with oxygen. McFarland et al (114) observed decrements in sensitivity of cone vision when volunteers inhaled carbon monoxide or low concentrations of oxygen. The visual response to a given %HbCO was roughly the same as an equal loss of $\%HbO_2$ due to aerohypoxia. McFarland (114) postulated that carbon monoxide diminished visual sensitivity by displacing oxygen from hemoglobin. Hence, "physiological altitude" was the apparent increase in altitude resulting from inhalation of carbon monoxide. For example, inhalation of 50 ppm carbon monoxide at 6,000 ft altitude (P_{O2} 127 torr) may be physiologically equivalent to an exposure to 12,000 ft altitude (P_{O2} 100 torr). Inhalation of 100 ppm carbon monoxide at 6,000 ft should produce the effect of exposure to 16,000 ft altitude (P_{O2} 86 torr). The hypothetical increments of apparent altitude would diminish as inspired concentrations of carbon monoxide exceed 100 ppm (72,114).

Correlations have been made between content of HbCO and physiological effect of altitude. One percent HbCO lowered the altitude which impaired critical flicker fusion frequency by 400 ft (82). Consequently, 5-9% HbCO depressed the critical flicker fusion frequency at 5,000-6,000 ft. altitude (82). Submarine atmospheres should raise HbCO by 2%, since they are contaminated by 10 ppm carbon monoxide (115). Were submariners to reside in 127 torr oxygen, equilibration with 10 ppm carbon monoxide would only degrade sensitivity for cone vision to that in 122 torr oxygen (114). If submariners maintain HbCO levels as high as 8%, then visual sensitivity in 130 torr oxygen should noticeably degrade to that in 108 torr (114,116).

It is not universally accepted that low doses of carbon monoxide degrade the performance of visual tasks. Subjects with 9-13% HbCO did not experience degradation of scotopic sensitivity, visually evoked cortical potentials, reaction time, or eye movements (117).

Christensen et al (112) tested whether 2-hour exposures to carbon monoxide (114 ppm) and aerohypoxia (P_{O2} 129 torr) cooperatively changed the performance of a visual vigilance task. Surprisingly, the subjects identified the same percentage of signals when breathing carbon monoxide in aerohypoxia as when breathing sea-level air. Significantly fewer signals were detected when the subjects breathed the low content of oxygen. Although several possible explanations were given for paradoxical improvement of hypoxic performance with supplemental carbon monoxide, there was concern for protection by an "alarm reaction" to hypoxia. If so, then long periods of exposure might exhaust the individual's ability to maintain vigilance (112)!

Continuous inhalation of 80 ppm carbon monoxide at 10,000 ft. altitude (P_{O2} 109 torr) degraded visual discrimination when HbCO reached equilibrium values (82). It is not known whether reduction of P_{O2} to 130 torr promotes toxic effects from chronic exposure to low levels of carbon monoxide (approximately 10 ppm) aboard submarines. Will 130 torr oxygen eventually raise the quantity of carbon monoxide bound by hemoglobin and extravascular heme molecules to the point of degrading performance?

11. Hyperbaric nitrogen

Doell et al (118) postulated that inspiration of 4 ATA air blunts the ventilatory response to aerohypoxia by doubling gas density. Denser gas raised the work of breathing, but did not depress the ventilatory response to aerohypxia (110 and 76 torr oxygen) at alveolar P_{CO2}'s < 46 torr. Higher alveolar P_{CO2}'s (> 46 torr in 11-22 torr atmospheric P_{CO2}) depressed the ventilatory response to aerohypoxia (118).

Maximum pressures of hyperbaric air (1216-1672 torr) have been estimated to permit no-stop decompression of nitrogen-saturated men to sea-level air (119). Assuming 1383 torr air (27 FSWG) to permit safe, uninterrupted

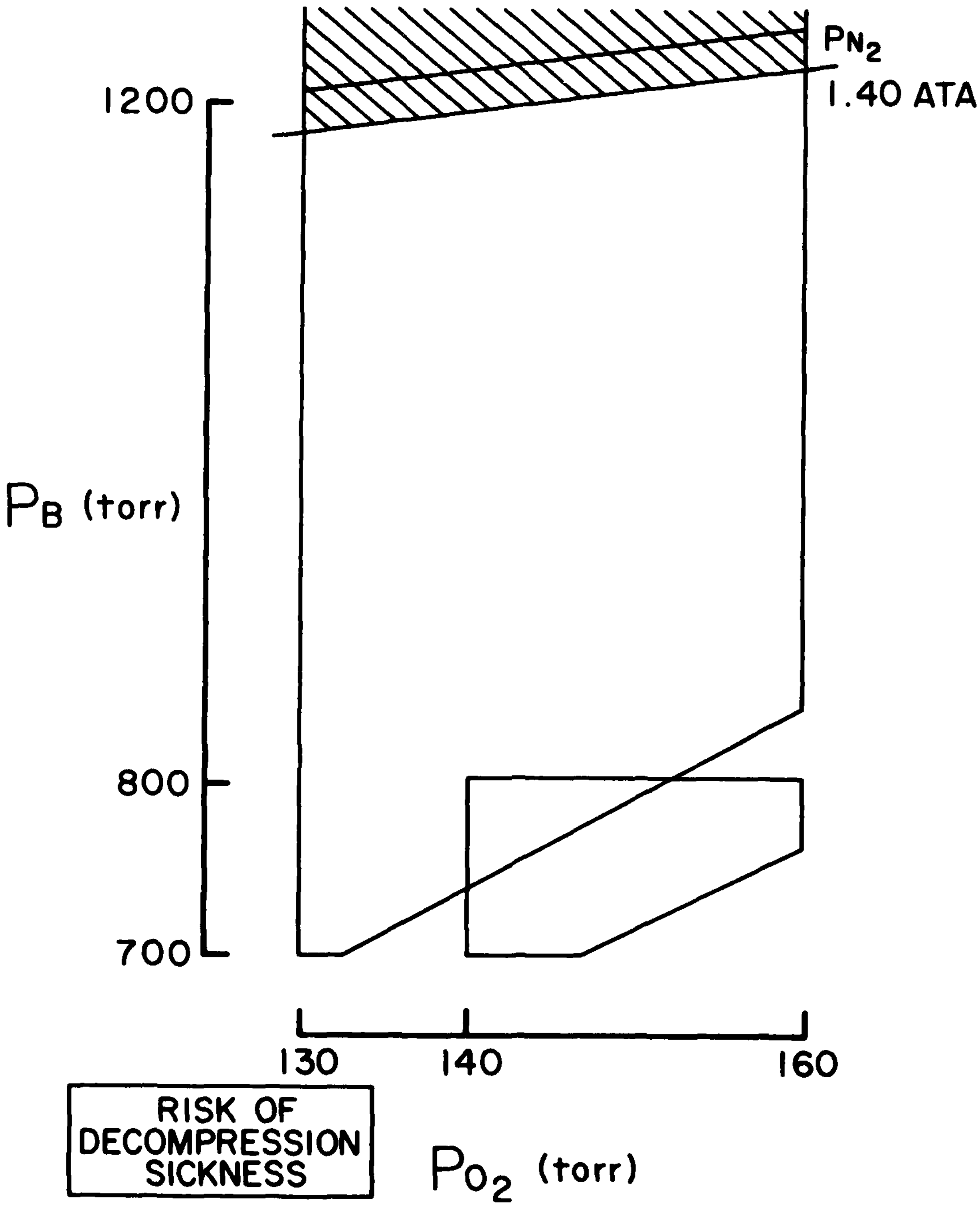

FIGURE 6. RISK OF DECOMPRESSION SICKNESS AFTER PATROL

The cross-hatched region warns of the possible development of decompression sickness when healthy men rapidly decompress to sea-level pressure after 2 or more days of residence in hyperbaric air.

decompression to sea-level pressure, the safe ambient nitrogen pressure is 1.44 ATA. In figure 1, the top edge of the larger life-support zone was an isobar for atmospheric nitrogen's partial pressure of 1.42 ATA. The preliminary findings of Eckenhoff et al (120) indicated that 1.40 ATA nitrogen is safe for abrupt decompression, but that 1.49 ATA nitrogen imposes a risk for decompression sickness. Nineteen men did not suffer symptoms of decompression sickness after living in 1.40 ATA nitrogen for 2 days. However, 4 of 15 men experienced mild symptoms of decompression sickness after living in 1.49 ATA nitrogen. The cross-hatched portion of figure 6 therefore depicts a zone of atmospheric P_{N2}'s which predispose crewmembers to decompression sickness. The chance that crewmembers would develop in-flight decompression sickness seems negligible. Submarine crews typically remain at sea-level pressure for several days after a patrol before flying in airplanes. Any medical evacuations at sea are accomplished by helicopters which fly at low altitudes.

It is unlikely that nitrogen narcosis results from living in nitrogen at a partial pressure of 1.4 ATM. Previous studies at the Naval Submarine Medical Research Laboratory (20) indicated that multiday exposures to 2 ATM nitrogen did not impair performance of arithmetic and tracking tasks.

Impure mixtures of hyperbaric nitrogen contain trace contaminants such as carbon monoxide, carbon dioxide, suspended particulates, and volatile organic compounds. Although Cohen (121) realized that "the effects of increased total pressure, per se, on the complex interaction between toxic substances and normal metabolic processes remain largely unknown", he concluded that compressed submarine atmospheres are safe for use as diver breathing air, provided that excess carbon dioxide and oil mist are removed before air consumption.

12. Potential engineering and operational problems

There are potential personnel problems arising from reducing the oxygen concentrations aboard submarines.

1. The crew might be incapacitated by hypoxia when the ship begins snorkeling with low concentrations of oxygen in an elevated barometric pressure (7,14). Sudden reduction of barometric pressure from 1000 torr to 760 torr, before raising oxygen concentration from 13% to 21%, would drop oxygen pressure from 130 torr to 99 torr. If the diesel engine were to draw a 6 inch vacuum, oxygen pressure would continue to drop to 79 torr. To avoid hypoxia, either the practice of snorkeling at weekly intervals (3) should be curtailed, the oxygen concentration should be raised before snorkeling, the crew should wear EAB's masks until oxygen pressure exceeds 140 torr, or the barometric pressure should not be elevated.

2. It is conceivable that the extinguishment of fires by improperly flooding a compartment with inert gas could induce hypoxia.

3. Smokers would not be able to burn tobacco. Crew morale might degrade when exposed to reduced oxygen concentrations for long periods of time. On the otherhand, non-smokers might enjoy the freedom from exposure to sidestream smoke.

4. Further reduction of oxygen concentration during a fire might impede performance before crewmembers begin using a respiratory device.

Perhaps there are engineering and operational concerns to be resolved before operating at sea with diminished concentrations of oxygen.

1. Will the benefits of reduced oxygen concentration apply to fires originating from electrical systems and flammable solvents?

2. Will the diesel engine operate in an atmosphere of diminished oxygen?

3. Although most machinery can withstand barometric pressures of 1.9 ATA (122), could the ship be incapacitated by damage to critical equipment when barometric pressure is raised to 1.4 ATA (1194 torr)?

4. Is the accuracy of the ship's analytical instruments sufficient to monitor the atmospheric P_{O2}?

5. What is the effect of lowering oxygen concentration and P_{O2} on the atmosphere control engineering plant?

6. The strategy of reducing oxygen concentrations aboard submarines does not apply fires originating from monofuels.

7. As a contingency, the ship may have to provide extra space for storing 21% oxygen in high pressure banks.

13. Summary and recommendations

The chances for successful submarine missions may be improved by operating with concentrations of oxygen below 19%, since fire damage would not be enlarged by conflagration. The reduced rate of burning (1,18) would certainly allow more time for effective damage control. Furthermore, a slower rate of burning should reduce radiant heat and improve visibility.

Submarine crews should continue the policy (123) of maintaining their ambient oxygen pressure at 159 torr when maneuvering in shallow water. It seems logical that they be required to breath 21% oxygen through a mask when correcting ship's casualties associated with fires, release of toxic gases, weapons' hazards, and radiation leaks. Otherwise, crewmembers should be able to operate in an oxygen pressure of 130 torr when P_B is 700-1000 torr.

Performance can be expected to degrade in proportion to the desaturation of oxygen from arterial hemoglobin. Since residents of Denver possess fully saturated hemoglobin, it is not surprising that the data in table 3 fail to indicate that P_{O2} 130 torr degrades human health and performance:

TABLE 3: FUNCTIONAL RESPONSES TO MILD AEROHYPOXIA

Ref.	P_{O2}, torr	Effect
(100)	159	alveolar P_{O2} 107 torr. alveolar P_{CO2} 39 torr.
(100)	159	4% CO_2 raised alveolar P_{O2} to 118 torr.
(95)	159	P_{CO2} 23 torr reduced night vision.
(88,91,99)	159	P_{CO2} 11-30 torr did not alter the uptake of oxygen.
(111)	159	4.3% HbCO degraded the maximum uptake of oxygen by 0.3 liters/min.
(117)	159	9-13% HbCO did not degrade visual tasks.
(12)	150	alveolar PO2 100 torr. alveolar PCO2 41 torr. oxygen saturation of arterial hemoglobin, 95%.
(9)	144	3% CO_2 caused shortness of breath to interfer with crew communications.
(21,25, 31,75)	138-110	slight impairment of dark adaptation.
(7)	137-126	successful operation of a submarine.
(9)	133-115	P_{CO2} 14-30 torr caused malaise or euphoria (in polluted air) aboard a submarine.
(100)	132	alveolar P_{O2} 85 torr. alveolar P_{CO2} 39 torr.
(100)	132	4% CO_2 raised alveolar P_{O2} by 13 torr.
(31)	132	slight impairment of novel tasks until completion of practice.

(12)	131	in P_{CO2} 17 torr: alveolar PO2 99 torr. alveolar PCO2 42 torr.
	$\geq$ 130	No impairment of memory.
(48)	130	Denver residents possess higher red blood cell volume.
(58)	130	competitive times prolonged for foot races $\geq$ 1,500 meters.
(113,109)	130	1% HbCO in the blood of non-smokers. < 6.3% HbCO in the blood of smokers.
(113)	130	5.1% HbCO degraded the maximum up-take of oxygen by 1.5 ml/kg/min.
(31,75,76)	130-122	training improved the performance of complex psychomoter tasks.
(112)	129	114 ppm CO did not degrade visual vigilance.
	128	altitude of the USAF Academy.
(72,114)	127	50 ppm CO should degrade the sen-sitivity of cone vision to that at P_{O2} 100 torr.
(61)	125	slight reduction of endurance for heavy work.
	122	cabin altitude for flight crews.
(112)	122	degradation of visual vigilance.
(12)	121	in 3% CO_2: alveolar PO2 95 torr. alveolar PCO2 42 torr. oxygen saturation of arterial hemo-globin, 95%.
(26)	118	arterial P_{O2} reduced by 26 torr. oxygen saturation of arterial blood only reduced by 4%.
(83)	117	threshold for hypoxic insomnia.
(28)	117	threshold for acute mountain sick-ness.

(70)	114	acceptable performance by resident machinists.
(21)	109	COMPENSATED HYPOXIA.
(25)	109	oxygen saturation of arterial blood, 85%
(5)	>101	normal taste perception and auditory function.
(21)	89	MANIFEST HYPOXIA.
(36)	77	oxygen saturation of arterial blood, 64%
(21)	75	CRITICAL HYPOXIA.

Experimental data should provide assurance that submarine crews can safely operate for 60-90 days in 130 torr oxygen without degradation of performance. The following questions need to be answered:

a. Does 130 torr oxygen interfere with effective work at the periscope and on watch?

b. Does the submarine environment degrade crew performance in 130 torr oxygen?

c. If there are decrements of night vision and performance of difficult tasks at 130 torr oxygen, do they reverse as individuals acclimate to the aerohypoxia?

d. What is the effect of hyperbaria on tolerance for oxygen pressure of 130 torr?

e. Will the combination of mild aerohypoxia with low concentrations of atmospheric carbon monoxide impair submarine crew performance?

f. Will the elevations of carbon dioxide encountered by submarine crews cause degradation of crew performance during exposures to mild aerohypoxia combined with trace concentrations of carbon monoxide?

At-sea trials must be performed before concluding that submariners can safely operate in reduced levels of oxygen. The objective of the field studies would be to show that atmospheric contaminants do not degrade crew performance during long-term residence in low concentrations of oxygen.

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