FUNDAMENTALS OF HYPERBARIC MEDICINE

NATIONAL ACADEMY OF SCIENCES
NATIONAL RESEARCH COUNCIL

FUNDAMENTALS OF HYPERBARIC MEDICINE

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COMMITTEE ON HYPERBARIC OXYGENATION
DIVISION OF MEDICAL SCIENCES
NATIONAL ACADEMY OF SCIENCES – NATIONAL RESEARCH COUNCIL

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FOREWORD

The term "Hyperbaric Medicine" has been coined to delineate the medical aspects of man's reactions to gaseous environmental pressures exceeding that of the natural atmosphere. Since such environments are of medical interest only if they are compatible with life, and, if by their manipulation medical benefit can be derived, the focus of this book is primarily on the effects of oxygen enrichment of inspired gases under elevated pressures.

The biological effects of hyperbaric oxygenation were matters of only academic medical interest until oxygen became available in quantity early in this century. Even then the thought that high pressures of oxygen might have therapeutic potentialities was dampened by the well-known fact that pressures of oxygen exceeding one atmosphere elicited toxic manifestations in animals. Only in the last two decades, and with gathering momentum in the last ten years, has hyperbaric oxygenation been systematically explored as a therapeutic measure in a variety of diseased conditions.

Responding to this wave of interest there was established, in 1963, a Committee on Hyperbaric Oxygenation within the Division of Medical Sciences of the National Academy of Sciences-National Research Council. The purpose of this Committee was to review the needs and opportunities for research in its field of interest. Its members immediately set to work to prepare a brief and succint critique of principles and practice in the administration of oxygen to man at elevated pressures, and the problems of selection and operation of equipment and of the training of personnel. This document, which was entitled "Hyperbaric Oxygenation: Potentialities and Problems," (NAS-NRC, Washington, D.C.), was published within the year.

Meanwhile plans were being laid for a more comprehensive publication. These have materialized in the book that is now in the reader's hands. The Table of Contents indicates its scope and depth.

The Division of Medical Sciences and, indeed, the community of the medical profession, is deeply indebted to the members of the Committee for their unselfish gift of much time and thought in the preparation of their individual contributions. The members, in turn, would, I am sure, wish to acknowledge the scrupulous editorial services of Drs. Leon Greenbaum and Sam Seeley.

Finally the Chairman of the Division expresses his particular appreciation of the courtesy of the Department of the Navy in granting permission to reproduce extensive sections of the U.S. Navy Diving Manual.

It is our hope that this book will be helpful not only to physiologists, clinical investigators and oxygen therapists but also to engineers who design and operate equipment and to those who are responsible for the management of diving and caisson operations.

R. Keith Cannan
Chairman
Division of Medical Sciences

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INTRODUCTION

Sam F. Seeley

The concept of the "breath of life" is, of course, a very old one. Yet the notion that the air breathed has an active part in the living process was not seriously entertained until the 17th century. It was in 1667 that Robert Hooke (1), demonstrating before the Royal Society, affirmed that

> "An aerial something, whatever it may be, essential to life, passes into the mass of the blood. Thus, the air driven out of the lungs, these vital particles having been drained from them, is no longer fit for breathing again."

A few years later, in 1674, John Mayow (2) identified this "aerial something" that supports combustion as a specific component of air that he called "spiritus nitro-aereus."

If, indeed, an "aerial something" was essential to life, it was natural to conclude that appropriate modifications in the concentration of air breathed by patients might be effective in the treatment of their diseases. As early as 1662, Henshaw (3) designed a chamber in which acute disease would be treated with increased pressures and chronic diseases with reduced pressures of air.

Interest quickened as technological progress provided means by which man could work under increased pressures in caissons, tunnels, diving bells, and diving dress. In 1887 Arntzenius (4) collected some 300 references on pneumatic therapy. Scores of chambers were built in Europe, Canada and the United States, but it was not until the mid-1930's that grandiose claims bordering on quackery faded out with the abandonment of the largest chamber, Cunningham's steel sphere of six stories containing 72 rooms. From all of this effort, although some practical lessons were learned with regard to decompression and many of the physiological responses to increased pressures were measured with the laboratory tools available at the time, only in the treatment of bends and air embolism was compressed air found to be of unmistakable therapeutic value.

Oxygen did not qualify for therapeutic use until the 1920's simply because it was not available in quantity till then and its role when administered at ambient pressure conditions is now well established. Its first use at high pressures was to prevent bends (5,6) by speeding the elimination of helium during surfacing from dives. In 1950 an attempt was made to exploit the toxic action of oxygen at increased pressures by inducing oxygen convulsions as a safer therapy than electroshock for treating schizophrenia (7,8). Since 1955 there have been continuous efforts to use high oxygen environments to potentiate radiation effects on human neoplasms (9).

The use of high oxygen pressures in cardiac surgery was proposed in 1955 (10) on the principle that "drenching" patients with oxygen should prolong the tolerance of the myocardium and the brain to cirulatory arrest. Although this application has not, as yet, succeeded in providing a useful degree of protection (8,11), it has received wide publicity in technical journals and especially in the lay press, as have the more recent use of high pressures of oxygen in the treatment of gas gangrene (12) and in experimental coronary artery occlusion (13). This surge of interest has excited the imagination of workers on diversified problems of medicine and surgery, including the respiratory distress of the newborn, tumor irradiation, organ transplantation, anaerobic infections, cardiac and peripheral vascular surgery, and congenital and acquired conditions in which oxygen transport or tissue oxygenation is impaired.

During the past 10 years clinical activity in this field has grown at a pace limited to a great extent by the high cost of installing and operating hyperbaric facilities. The hazards of bends, air embolism, oxygen toxicity, and fire assumed importance to laboratory workers and clinicians unfamiliar with the safeguards well known to workers in compressed air, diving and submarine medicine. Early enthusiasts exposed patients to great risk in improvised chambers of compressed air containing hydrocarbon vapors

from combustion motors and without provisions for treatment of accidental decompression. Patients with a wide variety of conditions have been compressed in the very few properly constructed chambers, but in insufficient numbers for any given disease to permit statistical comparison with those treated by conventional methods. In very few cases have the pressures of oxygen been precisely controlled and the physiological effects explored in depth.

The clinician who expects to find in this book a catalog of medical conditions in which hyperbaric oxygen has been shown to be an effective treatment will be disappointed. Some studies do indeed look promising, but work in this field is still in the experimental stage. The authors contributing to this volume reviewed the vast amount of information on the exposure of animals and man to compressed air and the limited information on exposures to increased ambient pressures of oxygen, and have sorted out the basic physiological principles and the problems and opportunities for research.

Investigations in hyperbaric oxygenation are expensive and time-consuming. Conditions are created for which codes of safety and operation have not been established. Not only are there physical hazards to attendants and patients but it must be anticipated that there are strict biological limitations on the use of hyperbaric oxygen. There may be a rather narrow margin between therapeutic benefit and irreversible damage to the retina, lungs, or central nervous system.

Hyperbaric chambers are now being established at a number of medical centers, and systematic, controlled studies are being undertaken by multi-disciplined staffs. This concentration of scientific inquiry should effectively bridge the transition from a period of experimentation to one in which there can be a valid definition of the therapuetic usefulness of hyperbaric oxygenation.

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Chapter I

THE COMPRESSED-GAS ATMOSPHERE

C.J. Lambertsen

INTRODUCTION

In the chapters that follow, the effects and toxicity of oxygen will be discussed in detail, along with indications, precautions, and procedures for the use of high oxygen pressures in therapy. This chapter will have a much more restricted purpose, namely, to consider the physiological implications of the compressed-air environment to which patients and individuals aiding in the therapeutic utilization of high oxygen pressures will be exposed. This compressed-air environment will exert physical pressure, change gas volumes, alter gas density and viscosity, increase the tensions of nitrogen to narcotic levels, and cause increased amounts of the inert gas to dissolve in body fluids. Each of these consequences of altered ambient air pressure deserves careful study. This chapter will serve to call attention to the nature of the problems that will be encountered, and will indicate sources in this volume and elsewhere where the necessary detailed information can be obtained.

PRESSURE-VOLUME RELATIONSHIPS, PARTIAL PRESSURES, AND THE GAS LAWS

In the rational use of positive pressure for the therapeutic administration of oxygen, it is necessary to consider the relation of increasing pressure to the behavior of a gas mixture, of any one gas in the gas mixture, and of gases dissolved in body fluids. The behavior of gases, whether in a pressure chamber, in a body cavity in the lungs, or in the tissues is understandable on the basis that the individual molecules of a gas are in motion at high velocity and along straight pathways. Thus, the pressure exerted by a gas in any of the situations mentioned is the sum of the inertial forces exerted by these fast-moving molecules, whether this be from the external auditory canal against the tympanic membrane, against the wall of a chamber air lock, or represents the diffusion gradient from a tissue capillary. Anything that increases the number of impacts or the velocity of movement will increase the gas pressure. The following laws of gases, describing the relationships among the factors concerned with total and partial pressures of the respiratory gases, are all pertinent to the high-pressure environment.

Effects of Pressure on Volume

As a gas is compressed, its volume is decreased in direct proportion to the increase in pressure (Boyle's Law). This, and its converse, are of obvious importance during changes in total pressure. The change from one pressure and volume to a second pressure and volume are expressed, for an ideal gas,

$$P_1V_1 = P_2V_2.$$

In ordinary situations of pressure change that can be tolerated by man, the failure of a real gas to behave as an ideal gas, due to forces of attraction among molecules and the space occupied by the gas molecules themselves (1), is not important. Therefore, as shown in Figure 1, the 4-liter pulmonary volume of a breath-holding subject who is compressed from the 1 atmosphere pressure at sea level to 4-atmospheres pressure in a chamber will be reduced to 1 liter. Since this is less than the normal pulmonary residual volume, the volume change will result in gross distortions of lung tissue and large hydrostatic pressure gradients across the delicate lung membrane, and pulmonary hemorrhage will result. This is the "squeeze" referred to in diving manuals (2,3). It is similarly responsible for the eardrum damage and middle-ear hemorrhage that occurs when blockage of the eustachian tubes prevents equilibration of middle-ear pressures during compression.

Figure 1 also illustrates the converse situation in which an increase in trapped gas volume occurs on decompression. A gas volume of 4 units at 4 atmospheres will expand to 16 units on return to 1 atmosphere. This large volume

change causes no problem in the middle ear, since a patent eustachian tube acts as a one-way valve to allow free exit of gas on decompression. However, air trapped in a bleb of an emphysematous lung or beyond a bronchial obstruction, air in the pleural cavity, air in the gastrointestinal tract, or even air in cerebral ventricles will expand on decompression and cause serious harm. Breath-holding during decompression, even by normal individuals ascending from a depth no greater than 15 feet, has led to overdistension of the lungs, disruption of lung tissue, entrance of air into the circulation, and death from aeroembolism (3).

Finally, it is evident that as the normally breathing subject is compressed from 1 to 4 atmospheres, lung volume will not change, but the lungs will be occupied by four times as many molecules of gas as at sea level.

Partial Pressure of Gases in Gas Mixtures

In a gas mixture, the pressure exerted by each gas in a space is independent of the pressures of other gases in the mixture (Dalton's Law).

Since each gas behaves as though it were the only gas in a space and distributes itself uniformly, total gas pressure is the sum of the partial pressures of each of the individual gases present. For example, in the pulmonary alveoli

$$\text{Total pressure} = P_{H_2O} + P_{CO_2} + P_{N_2} + P_{O_2}.$$

The partial pressure or "tension" of one gas in a mixture is therefore directly proportional to the percentage of the gas in the mixture and to the total pressure of the gas mixture. Thus, oxygen partial pressure (P_{O_2}) in a dry gas mixture containing 10 per cent oxygen and inspired at 1.0 atm ambient pressure (760 mm Hg) is

$$P_{O_2} = 10\% \times 760 \text{ mm Hg} = 76 \text{ mm Hg}$$

and 100 per cent dry inspired O_2 at 1 atmosphere exerts a partial pressure of 760 mm Hg.

In the calculation of partial pressure of a gas in a mixture, water vapor must be considered as one of the gases present. Most gas-analysis procedures determine percentage composition of dry gas. Therefore, to determine the partial pressures of a particular gas in the lungs where alveolar gas is saturated with water vapor, the partial pressure of alveolar water vapor must be deducted from the value of total ambient pressure to obtain the total pressure of "dry gases" (4, 5a). The pressure of water

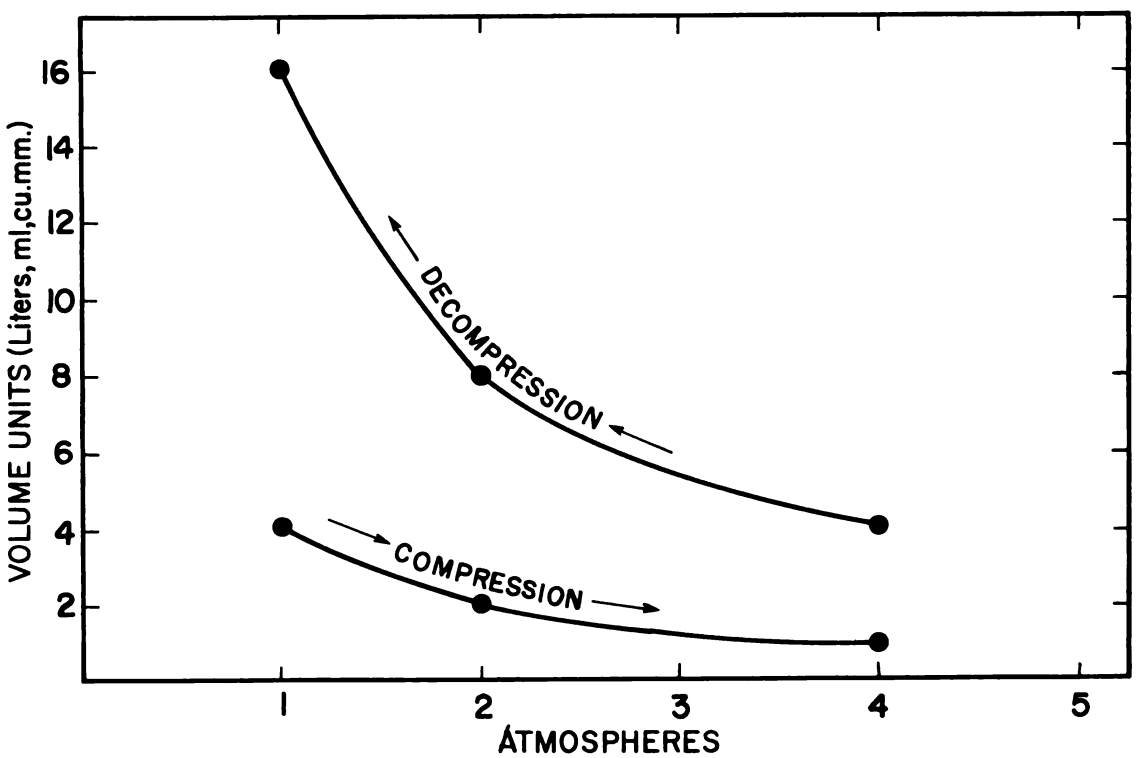

Figure 1.

vapor (P_{H_2O}) is a function of temperature only and at normal body temperature is 47 mm Hg. Therefore, after measuring the percentage of oxygen in alveolar gas, the alveolar P_{O_2} would be determined as follows:

P_{O_2} = %O_2 in dry gas x total pressure of dry gases

P_{O_2} = %O_2 x (total ambient pressure - P_{H_2O} 37° C).

Breathing air at 3.5 atmospheres (6)

P_{O_2} = 20.94% x (2660 mm Hg - 47 mm Hg)

P_{O_2} = 547 mm Hg.

During oxygen breathing at this same pressure (6)

P_{O_2} = total pressure - P_{CO_2} - P_{H_2O}

P_{O_2} = 2660 - 33 - 47

P_{O_2} = 2580 mm Hg.

Partial Pressures of Gases in Liquids

The degree to which a gas enters into physical solution in body fluids is in direct proportion to the partial pressure of the gas to which the fluid is exposed (Henry's Law).

Gases enter physical solution in body fluids independently of each other and tend to behave independently in solution.

When a gas is dissolved in a liquid, it continues to exert a partial pressure, which can be regarded as the force with which the gas molecules are diffusing through the liquid. Since molecular movement is altered by change in temperature, any alteration of the temperature of body fluids will also change the partial pressures of gases already in solution.

The sum of the partial pressures of gases dissolved in a body fluid does not have to equal the total ambient pressure. For example, at a pressure of 3.5 atmospheres (2660 mm Hg) the gas pressures found in brain venous blood (7) are

$P_{H_2O} + P_{CO_2} + P_{O_2}$ = total gas pressure in blood

47 + 53 + 75 = total gas pressure in blood

175 mm Hg = total gas pressure in blood.

This represents a drop of over 2000 mm Hg in total gas pressure across the brain. This large drop is due to removal of oxygen from the blood as it passes through the actively metabolizing brain.

When physical solution alone is involved, the relationship between partial pressure of a gas and the amount of gas dissolved in a body fluid is expressed numerically in terms of α, the Bunsen solubility coefficient. This is "the amount of gas in ml which will dissolve in 1 ml of the fluid per atmosphere (760 mm Hg) of gas pressure." The solubility coefficient is temperature dependent, solubility increasing with fall in temperature. Values for physical solubility of oxygen in plasma and in whole blood are 0.0214 and 0.0214 + (0.000108 x vol % O_2 capacity) ml/ml/atm at 37° C, respectively (8). The value for whole blood is greater than that for plasma due to a higher physical solubility of oxygen in red cells than in plasma. The solubility coefficient permits estimation of physically dissolved O_2 if P_{O_2} is known, and of P_{O_2} if physically dissolved O_2 is measured.

Thus,

Physically dissolved O_2 content (in vol %) = total O_2 content (in vol %) - HbO_2 capacity (in vol %)

and

$$P_{O_2}\ (\text{mm Hg}) = \frac{\text{dissolved } O_2 \text{ content} \times 760 \text{ mm Hg}}{\alpha (\text{vol \%})}$$

Conversely, when oxygen partial pressure is known, the physically dissolved O_2 concentration in a body fluid can be determined by

$$\text{Physically dissolved } O_2\ (\text{vol \%}) = \frac{P_{O_2} \times \alpha}{760}$$

It is necessary to emphasize partial pressures of gases in dealing with problems of hyperbaric therapy. The partial-pressure gradient for oxygen determines the rate and limits of diffusion from alveoli to blood and from blood to the tissue cell. Moreover, the toxicity of oxygen and the narcotic properties of nitrogen are related to the partial pressure of these gases in cells, not to their concentration in inspired air.

RESISTANCE TO BREATHING AT HIGH PRESSURES

As the pressure of a respirable gas is increased to the extreme levels encountered in deep-sea diving, resistance to breathing pro-

gressively rises. This appears to be due primarily to an increase in nonelastic resistance to air flow in the bronchioles and to be related to an increase in turbulence of air in the respiratory passages (9). The degree of turbulence of air in the small passages such as the bronchioles is dependent upon gas density (10); because of this, a helium-oxygen mixture can flow faster than air can through a small tube before turbulence develops (11). When studied by the maximum-breathing-capacity method, interference with ventilation can be detected even at pressures of 2, 3, and 4 atmospheres. While it should therefore be expected that ventilation in vigorous exercise at 3 or 4 atmospheres will be restricted (12), there is not yet any indication of detectable ventilatory impairment at the lower alveolar ventilations found in normal resting subjects (6) or in subjects during ordinary mild exercise. Studies will now have to be made of problems of ventilation at rest when the lung has been modified by pathological, obstructive processes.

INERT-GAS NARCOSIS

The "inert" gases such as nitrogen exert prominent effects upon cellular function (13), even though they do not strictly enter into chemical metabolic processes. The depressant or "narcotic" influences of nitrogen and other inert gases probably do not have a true threshhold for effect, but bring about their depressant phenomena by an action that progesses as the partial pressure in the tissue increases. Interest is increasing in the inert gases, and, though this report is largely limited to circumstances expected in medical uses of hyperbaric oxygenation, it is worth pointing out that excellent sources of information on inert gases other than nitrogen exist (14).

Nitrogen at high pressure does not merely produce a euphoric sensation in humans; it presumably can affect any type of cellular function if the pressure is raised high enough. It can produce actual unconciousness (13), prevent the convulsions of electroshock in mice (15), impair mentation and motor performance (16), affect visual reaction time (17), block conduction in nerve fibers (15), block synaptic transmission in nerve pathways (15, 18), slow the metamorphosis of insects (19), diminish the rate of maturation of plant seedlings (13), and interfere with the oxygen-dependent radiosensitivity of plant, microbial, and tumor cells (20). These and other influences of inert gases (13) indicate the diverse patterns of inert-gas effects. It is possible that many of the effects of nitrogen upon cellular function are produced by a single mechanism of action. The mechanism of inert-gas narcosis and the important related question concerning the mechanism of anesthetic action have been the subject of much experiment and thought over many years. The major theories have been critically reviewed on several occasions (1, 15, 21, 22, 23). In all such discussions, it is agreed that many of the biological effects of nitrogen and the other inert gases can be produced by physical actions. Ionic or covalent bonding with cellular metabolic constituents is not required to cause depression of metabolism or other functional activity. Apparently, the physical presence of molecules, probably concentrated at particular sites on the surface of structures within the cell, can lead to the interferences collectively called narcosis.

A number of different but related theories have been proposed to explain narcosis as the result of physical actions of inert gases. Each is based upon demonstration of a correlation between anesthetic or narcotic activity and a physical characteristic of the inert agent. The Meyer-Overton theory, best known of such correlations (24), considers the narcotic action to be related to lipoid solubility and oil-water partition coefficients of the inert substances and, hence, to a concentrating of molecules in important cellular lipid elements. However, anesthetic activity also correlates equally well with partial molal free energy of the gas molecules (21, 25), lowering of surface tension of water (26), molecular weight (13), molecular refraction (1), boiling point (1), polarizability of molecules (13), the dissociation pressure of hydrates (22, 27), and Van der Waals forces (1). In the face of these many proposals, it is important to recognize that most of the properties found to correlate well with anesthetic action are not in fact independent of each other (25), and that high correlation does not imply mechanism. Quite possibly, the common factor will prove to be one of distribution of gas molecules at water-lipid interfaces, such as the surfaces of mitochondria.

Whether or not the mechanism of inert-gas narcosis can be selected from among these many possibilities, it is a real phenomenon and one that has practical importance at high air pressures. The nitrogen in compressed air produces detectable subjective effects even at pressures of about 3 atmospheres (28). These effects are not great at this low pressure, and it is possible at 3 to 4 atmospheres to

perform intricate, timed, experimental procedures such as measurement of brain blood flow (7) and to perform surgical procedures (29,30,31). Apparently, objective evidence of a performance decrement can be obtained at air pressures of 4 to 5 atmospheres (17,32), but the decrement in performance appears to be extremely small both at 5 atmospheres of air pressure (17) and in other narcotic states where subjective effects are prominent (33). Thus, practical trial over the years has shown that competent, purposeful individuals can work accurately and effectively at air pressures up to 4 to 5 atmospheres. This should not be taken to mean that any individual will perform any procedure without difficulty under a pressure of 0.80 x 5 atmospheres of nitrogen. Judgement, comprehension, awareness, and skill will hardly be improved by exposure to this environment (33). As pressure is increased, or as P_{CO_2} rises (12,17,34), or as a gas having an increased level of oxygen pressure as well as increased nitrogen pressure is breathed (17), the degree of narcosis and the associated detrimental effects will increase. These all may be encountered in the course of treating a bends or aeroembolism patient at the high pressure of 6 atmospheres.

UPTAKE AND ELIMINATION OF NITROGEN BY BODY FLUIDS: DECOMPRESSION SICKNESS

The tissues of an individual living at sea level are in equilibrium with the partial pressure of nitrogen in his alveolar gas (about 573 mm Hg P_{N_2}). On exposure to an increasing ambient pressure of air in a pressure chamber, the alveolar P_{N_2} will increase directly and immediately with the rising pressure of respired air.

It should be expected that, due to the extreme efficiency of alveolar-blood gas exchange, the arterial P_{N_2} will follow "instantly" the rise in arterial nitrogen pressure. Thus, within one lung-to-tissue circulation time, every capillary will be presented with a P_{N_2} approximately equal to that in the pressurized atmosphere. Nitrogen tension of the various tissues throughout the body will not rise instantly to this high value; the cells will come into equilibrium with the arterial nitrogen at an infinite number of different rates. Theoretically, the cells will approach equilibrium exponentially, but the rate of equilibration will vary with the solubility of nitrogen in the constituents of a particular tissue (nitrogen is about 5 times more soluble in fat than in water) and with the volume rate of blood flow through the tissue region (35). Within a particular organ or tissue that has a rapid mean rate of circulation and low index of solubility for nitrogen, there may be a discrete mass of material that has a limited circulation and different physicochemical characteristics (such as high lipid concentration) than the rest of the tissue. Even normal tissues and organs should therefore not be considered homogeneous in regard to inert-gas exchange; the existence of pathological states will further modify the rate of nitrogen uptake and release.

If the exposure to increased pressure is prolonged, the P_{N_2} in all tissues and cells will come essentially into partial-pressure equilibrium with the high nitrogen pressure. In normal decompression, as a gradient of nitrogen pressure from tissues to capillary blood is established, the excess dissolved nitrogen diffuses into the blood, is then transported to the lungs in solution, and is readily eliminated into the alveoli and from the lungs.

Decompression sickness, or "bends," occurs when the rate of decompression is too fast to permit the desired, gradual diffusion of excess nitrogen from the tissue fluids into the blood. As indicated above, the tissues are not homogeneous in their uptake or release of nitrogen. Thus, on decompression, the pulmonary capillary blood may be cleared of nitrogen to the new level of alveolar P_{N_2} in a single passage through the alveolar circulation, and arterial blood may enter a tissue with no excess of nitrogen. However, again depending upon the amounts of dissolved nitrogen to be released by a tissue mass, the diffusion distance, and the rate of perfusion by blood, some tissues will be rapidly and safely cleared at a rate of decompression that results in dangerous residual nitrogen supersaturation in others.

The rate of nitrogen elimination from the whole body has been studied; it is described by a complex curve made up of the many overlapping rates of nitrogen elimination (35,36). For convenience only, and to aid in understanding the practical implications of the decompression procedures used to prevent bends, this complex curve has been empirically divided into rate functions described as "slow," "moderately fast," and "fast" tissues with respect to inert-gas uptake and elimination. It is important to realize that these do not describe anatomically identifiable, gross masses of tissue; the designations are mathematical descriptions of groups of cells in scattered regions having similar characteristics of inert-gas

exchange. It is probable that only in extremely rapid decompression or in severe decompression sickness are bubbles likely to appear in the arterial blood. However, if the decompression rate is extreme, it is believed that bubbles in the returning venous blood may become trapped in the pulmonary capillaries to produce the acute respiratory distress called diver's "chokes." Bubbles that succeed in passing the pulmonary filter are free to enter and obstruct the capillary beds of vital organs, including the brain and heart.

The occurence of severe circulatory-system obstruction by bubbles, as a form of decompression sickness, is fortunately not common. More usual is the development of pain in extremities, especially about the joints. This pain may be extreme. Skin involvement may result in rash and itching. The development of bubbles in the central nervous system, whether brain or spinal cord, is extremely serious and even with treatment may lead to long-lasting disability. The practical aspects of prevention and treatment of these severe problems are considered in Chapter VI and elsewhere (3).

Discussion of the decompression advantages of helium-oxygen and of nitrogen-oxygen mixtures other than air are presented elsewhere (3, 13, 37). For present purposes it should be pointed out that nitrogen not only has a higher solubility in water and in fat than does helium, but diffuses less rapidly in body fluids. For both these reasons, helium is more rapidly eliminated from the body than is nitrogen following exposure to a given high partial pressure of inert gas. This is true regardless of whether the exposure has been brief or has approached saturation.

ANOXIA AND OXYGEN

The aim of hyperbaric therapy with oxygen is usually to relieve an hypoxic state, which may be localized or general. Hypoxia implies only a deficiency of oxygen; the ultimate cause of damage or death is failure of oxidation within tissue cells. Rational employment of higher than ambient pressures of oxygen to relieve cellular hypoxia depends upon an awareness of the mechanism and extent of the interference with cellular oxygenation and upon the quantitative gains in cell oxygenation that can be accomplished by use of oxygen at increased partial pressure.

Detailed discussions of causes and effects of anoxia in disease are available (5b, 38) and will not be repeated here. Table 1 summarizes the various types of anoxia on the basis of precipitating causes and in relation to the consequent alteration of P_{O_2}. Together with special problems, such as treatment of bends and the use of oxygen to improve radiosensitivity of tumor tissue, this group of pathological situations includes some of the conditions that may be relieved in part by administration of oxygen at 1 or more atmospheres pressure. Chapters II and III describe the physiological and toxic effects of oxygen itself. The particular advantages and limitations of high oxygen pressures in treating various types of anoxia are considered in Chapter IV and elsewhere (29, 30, 38). It should be evident that administration of oxygen at increased ambient pressure is an extension of the extremely valuable procedure of oxygen therapy at 1 atmosphere, and that the use of oxygen at sea level, of drugs, and of hypothermia should continue to be fully exploited in efforts to protect against the damaging effects of hypoxia.

TABLE 1
Classification of Anoxia

Suggested terminology		Characteristics	Occurrence	Corresponding term in older classification
Anoxemia (diminished O_2 in blood)	Hypotonic anoxemia	Decreased volume and tension of O_2 in arterial blood; chemoreceptor activation by diminished P_{O_2}; cyanosis when Hb concentration is adequate	Any condition leading to lowered arterial P_{O_2} (respiratory obstruction, decreased alveolar ventilation, drowning, respiratory obstruction, lowered inspired P_{O_2}, asphyxia, transpulmonary shunt, reduced permeability of alveolar membranes)	Anoxic anoxia
	Isotonic anoxemia	Arterial O_2 content low but P_{O_2} normal; chemoreceptor stimulation not a feature; cyanosis absent; may be skin color of methemoglobinemia, carboxyhemoglobinemia	Anemia poisoning by CO and by drugs which cause methemoglobinemia (aniline, acetanilid, nitrophenol, chlorates, methylene blue)	Anemic anoxia
Hypokinetic anoxia (diminished blood flow) — Ischemic	Local	Subnormal local arterial supply; local cyanosis or pallor; pain	Arterial embolus, thrombosis, spasm, obliteration, damage; high extravascular pressure (e.g., CSF)	Stagnant anoxia
	General	Subnormal arterial supply to the entire body; hypotension, pallor, syncope, air hunger, metabolic acidosis	Acute circulatory collapse (spinal anesthesia, depressor drugs, acute cardiac failure)	
Congestive	Local	Impediment to venous return; edema, capillary engorgement, and cyanosis	Thrombosis, external pressure	
	General	High systemic venous pressure with capillary and venous engorgement; cyanosis	Questionable—perhaps congestive heart failure or polycythemia	
Overutilization anoxia (excessive O_2 requirement)		Demand for O_2 (local or general) increased relative to supply; may be local or general	Convulsions, oxygen debt in exercise, angina, intermittent claudication	None
Histotoxic anoxia (failure of metabolism)		Acute depression or inactivation of cellular oxidative systems	Poisoning by cyanide, sulfide, oxygen, etc.	Histotopic anoxia

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Chapter II

PHYSIOLOGICAL EFFECTS OF OXYGEN INHALATION AT HIGH PARTIAL PRESSURES

C.J. Lambertsen

INTRODUCTION

Time is required for the development of oxygen toxicity. However, promptly upon beginning oxygen breathing and during the safe or symptom-free latent period of useful exposure before overt oxygen toxicity occurs, oxygen produces a number of physiologically important effects. These chiefly involve respiration, gas uptake, gas transport and tissue gas exchange. Although harmless, they bear heavily on the rate of development of oxygen toxicity and on the degree of benefit to be expected from hyperbaric oxygenation in the disease state. The more important of these effects will be considered here with emphasis on actual experimental observations that can be extrapolated to unusual clinical situations.

EFFECT OF OXYGEN PRESSURES ON METABOLISM

Early it was considered that, as for a candle flame, the oxidative processes of body metabolism would be accelerated by exposure to very high oxygen tensions. This is not the case, because neither oxygen consumption of isolated tissues (1,2) nor the oxygen metabolism of the intact human brain (3) is increased by oxygen at a pressure of several atmospheres. If a pathological situation of subnormal tissue oxygenation exists with lowered tissue oxygen consumption due to hypoxia, then elevation of P_{O_2} should be expected to increase oxygen metabolism toward normal.

PULMONARY OXYGEN UPTAKE

In a subject with normal lungs, administration of oxygen at a pressure of several atmospheres will result in the same rapid rate of elevation of alveolar oxygen concentration as will oxygen administration at sea level (4). Nitrogen washout from the alveolar gas of a normal lung occurs at an exponential rate and should be about 98 per cent complete in about seven minutes (4,5). Concurrently, there is a rise in alveolar P_{O_2} nearly to the inspired level.

Alveolar P_{O_2}

The characteristics of any alveolar gas in terms of total and partial pressures can be expressed as follows:

Total alveolar gas pressure =
$P_{O_2} + P_{CO_2} + P_{\text{inert gas}} + P_{H_2O}$.

Total alveolar gas pressure, equal to the ambient pressure at which the individual breathed, is thus the sum of the partial pressures of the individual gases. Normally, the partial pressures of two of these gases are linked to regulatory processes and are not passively modified by alteration of ambient pressure. One is P_{CO_2}, which is held close to 40 mm Hg by the respiratory control system. The other, P_{H_2O}, depends only on body temperature. Thus, in normal subjects who have breathed oxygen long enough to eliminate most of the nitrogen from the lungs, the description of alveolar gas at 1 atm can be expressed as:

Total pressure = $P_{O_2} + P_{CO_2} + P_{H_2O}$

and

673 mm Hg P_{O_2}
40 mm Hg P_{CO_2}
47 mm Hg P_{H_2O}
760 mm Hg

As inspired oxygen pressure is increased to 2,3, or more atmospheres, P_{CO_2} and P_{H_2O} will remain close to their sea-level values, while the oxygen tension of the alveoli will rise almost millimeter for millimeter with the rise in the pressure of inspired oxygen.

Oxygenation of the Blood in the Lungs

In the normal exchange of oxygen between alveolus and blood, the P_{O_2} of the pulmonary capillary blood comes to within a fraction of a millimeter of equilibration with the oxygen tension of the alveolar gas (6, 7a, 8). As alveolar P_{O_2} is increased to several atmospheres, a limitation of alveolar-pulmonary capillary oxygen transfer becomes evident and is reflected in an alveolar-arterial P_{O_2} difference as large as several hundred millimeters of mercury at 3 to 4 atm of inspired oxygen (3). This is reflected in a smaller than expected rise in arterial oxygen content (3, 9). The cause of the limitation of oxygen uptake in the lungs is not yet known. Either a great increase above the normal (approximately 2 per cent) shunt due to venous admixture (6) or a diffusion limitation must be postulated. Study of the latter possibility has revealed a progressive decrease in the pulmonary diffusion constant for CO as alveolar P_{O_2} is raised from 1 to nearly 5 atm (10). This limitation may well be due to the interference by high oxygen pressures with the combination of hemoglobin and carbon monoxide, but it does not in itself explain the observed interference with transpulmonary oxygen uptake.

GAS TRANSPORT TO THE TISSUES

Arterial hemoglobin saturation with oxygen is normally about 96 to 98 per cent, even during air breathing at sea level (7b). Elevating the alveolar P_{O_2} leads to a further increase in arterial oxyhemoglobin concentration until the hemoglobin is completely saturated with oxygen. While arterial oxygen tensions of many thousands of millimeters of mercury can be obtained by increasing the pressure at which oxygen is breathed, hemoglobin oxygenation is self-limited, and essentially complete saturation occurs at somewhat over 100 to 200 mm Hg (11). The physical solution of oxygen in the water of arterial blood is not limited and increases indefinitely in proportion to the rise in P_{O_2}.

Figure 1 shows the oxygen-uptake curve of arterial blood as studied in normal man with inspired-oxygen tensions up to 3.5 atm. (7b). Above complete oxygenation of hemoglobin, the slope of the oxygen-uptake line represents the physical solution of oxygen in the water of blood as P_{O_2} increases. The characteristics of this curve above where complete hemoglobin saturation has occurred illustrate why removal of a small amount of oxygen from the blood in the tissues can cause the partial pressure of oxygen in blood to fall by several thousand mm Hg in its transition from the arterial to the venous state (12). Exposure of arterial blood to high alveolar P_{O_2} increases both the oxygen tension gradient from blood to metabolizing cell and the volume rate of "oxygen flow" (amount of oxygen perfusing the tissue per minute) through a tissue. This explains why effective cellular oxygenation can sometimes be accomplished at very low rates of tissue blood flow when the arterial P_{O_2} is high. However, hyperbaric oxygenation offers no direct advantage in either removal of metabolites from the tissue or delivery of nutrients. Therefore the blood flow must be maintained at a level adequate to prevent both undesired changes in the cellular environment and failure of cellular metabolic processes caused by conditions other than hypoxia (14). The solubility of oxygen in the plasma at a body temperature of 37°C is approximately 0.0214 ml O_2/ml plasma/atm (15). Because the solubility of oxygen is greater in a given volume of red cells than in the same volume of plasma (16), it is necessary to take into account the hematocrit (or hemoglobin concentration or oxygen capacity) when estimating the physical solubility of oxygen in whole blood. This is done by adding to the solubility coefficient for oxygen in plasma a value to adjust for the physical solubility of oxygen in the hemoglobin water as follows:

$$\begin{aligned} \alpha O_{2_{blood_{37}}} &= \alpha O_{2_{plasma_{37}}} + (0.000108 \times O_2 \text{ capacity}) \\ &= 0.0214 + (0.000108 \times 20) \\ &= 0.0214 + 0.00216 \\ &= 0.0236 \text{ ml } O_2\text{/ml blood/760 mm Hg} \end{aligned}$$

At an oxygen capacity of 20 volumes per cent, the solubility of oxygen in the water of whole blood is about 0.0236 ml O_2/ml blood/atm. For each increase of 760 mm Hg in arterial oxygen pressure, approximately 2.4 ml of oxygen will dissolve in each 100 ml of the arterial blood. The solubility of oxygen in blood and plasma is affected by body temperature (17), which must be taken into account in estimating delivery of oxygen to the tissues.

At a sufficiently high arterial P_{O_2}, the oxygen requirements of the metabolizing tissue can be met entirely by the physically dissolved oxygen carried in the blood. The oxyhemoglobin will then pass unchanged from the arterial to the venous side of the circulation. It was estimated about thirty years ago that this physiologically important situation would exist

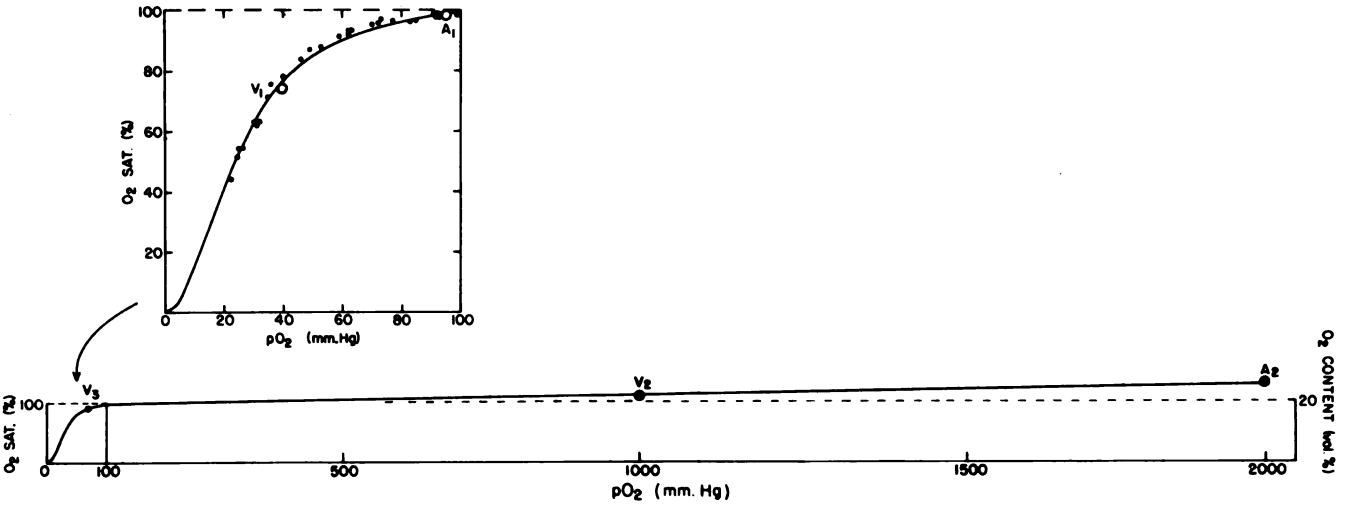

Figure 1. Oxygen-uptake and oxygen-liberation curves for blood at normal and high pressures of inspired oxygen (data from normal men at rest [12, 3, 13]).

In the upper diagram, A_1-V_1 indicates for air breathing at sea level the expected transition of P_{O_2} and per cent of Hb saturation from a measured arterial to an estimated level for mixed venous blood. The numerous points represent observations on arterial blood during the administration of air and gas mixtures low in oxygen content (13).

The lower diagram shows the additional oxygen uptake by arterial blood as inspired P_{O_2} is raised to 3.5 atm. Somewhat above 100 mm Hg P_{O_2}, hemoglobin becomes completely saturated; thereafter the slope of the oxygen-uptake curve represents the physical solubility of oxygen/mm Hg of P_{O_2}. A_2 - V_2 indicates the degree of change in P_{O_2} across the brain that would be predicted on the basis of normal (A-V) oxygen extraction.

A_2-V_3 shows the pattern of oxygen-liberation found by actual experiment. The greater magnitude of P_{O_2} fall is due to a considerable decrease in brain blood flow on oxygen administration (3).

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at inspired oxygen pressures above about 3.0 atm (18,19). This general statement should be true for tissues in which the ratio of "oxygen inflow" to oxygen consumption is high. Measurements of oxygen exchange across the human brain indicate that more than 3 atm of inspired oxygen or an increase in blood flow are required to ensure complete saturation of brain venous blood (3,12).

TISSUE-OXYGEN TENSIONS

Owing to the metabolic use of oxygen, an increase in the pressure of inspired oxygen does not produce an equal rise in the P_{O_2} of the tissue capillaries or cells. Figure 2 shows the results of experimentally imposed increases in pressure of inspired oxygen from 0.2 to 3.5 atm (14). Even the lowest of these levels is adequate to saturate arterial hemoglobin completely but not to supply the tissues entirely with physically dissolved oxygen. The induced rise in P_{O_2} along a brain-tissue capillary is not uniform from one end to the other unless oxygen flow is very high relative to demand. At the extreme arterial end, P_{O_2} will be elevated to nearly the pressure of the inspired oxygen. However, as physically dissolved oxygen diffuses along the P_{O_2} gradient to the metabolizing cells, P_{O_2} falls in the blood passing through a tissue capillary. As the P_{O_2} falls low enough to permit release of oxygen from oxyhemoglobin, large amounts of chemically bound oxygen become available to limit a further lowering of P_{O_2} in the capillary blood.

Only when the rise in the pressure of inspired oxygen or in tissue blood flow is so great or the rate of oxygen use is so small that oxyhemoglobin is not reduced at all in transit through a capillary can the P_{O_2} at all points along the capillary be expected to increase directly with the elevation of arterial P_{O_2} (see Figure 5, Chapter III). Even in this state, factors influencing the progressive removal of oxygen along the capillary may theoretically cause the change in P_{O_2} of one tissue cell to be much greater than the change in the P_{O_2} of others (12,14).

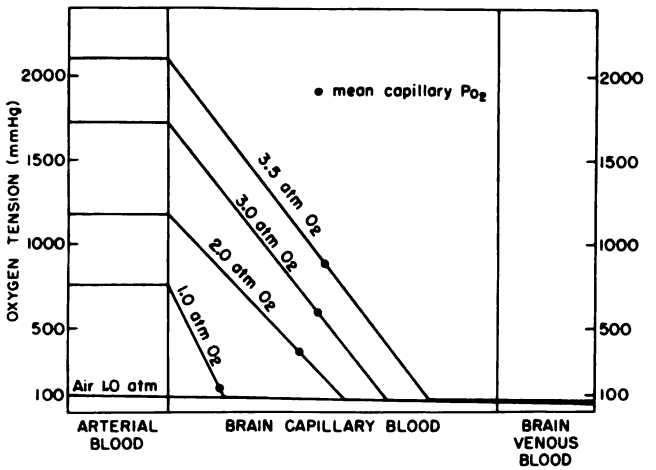

Figure 2. Effect of increased inspired P_{O_2} on the oxygen tensions of arterial, mean brain capillary and internal jugular venous blood (average values in normal men [9, 3, 20]).

The graph illustrates for each of several levels of inspired P_{O_2} the manner in which oxygen tension may fall as blood flows through the average brain capillary. The patterns of change in brain capillary P_{O_2} are calculated from experimentally measured levels of oxygen pressure in arterial and brain venous blood on the assumption of uniform oxygen loss. Measurements are for resting men at 0.2, 1.0 and 3.5 atm (3), and 3.0 atm (9). The values at 2.0 atm were obtained in exercising subjects (20).

As blood enters the capillary, a rapid fall in P_{O_2} occurs, owing to the use of physically dissolved O_2. The rates of fall of P_{O_2} at different levels of inspired P_{O_2} are nearly parallel, the small differences being due to slight inequalities in brain blood flow. When P_{O_2} falls to such a degree that oxygen is provided by the oxyhemoglobin, the fall of P_{O_2} is drastically slowed. Note that even at 3.5 atm the P_{O_2} of venous blood is raised relatively little by the administration of pure oxygen. The degree of rise of venous P_{O_2} and the pattern of P_{O_2} change across the capillary will both depend on blood flow and tissue metabolism (14).

Figure 3 compares the actual change in P_{O_2} found to occur across the brain circulation with predicted changes in the P_{O_2} of blood flowing through the capillary beds of various important tissues. The predictions, which are approximations for comparative purposes only, are based on known values of normal blood flow and oxygen consumption of each tissue (21), and on the conventional assumption that oxygen loss from the blood occurs uniformly along the capillary. It is clear that, even in the normal subject, for any particular pressure of inspired oxygen the "dose" of oxygen will differ from organ to organ and from tissue to tissue (14). Even within an organ or tissue, there should be local differences in P_{O_2} due to inequalities of blood flow and metabolism. In pathological states there should be greater discrepancies between one gross or microscopic region and another.

At a particular high inspired P_{O_2}, it can be assumed that the degree of oxygenation of cells can still be further increased by the administration of drugs that lower the rate of tissue oxygen consumption, by lowering body t.empera-

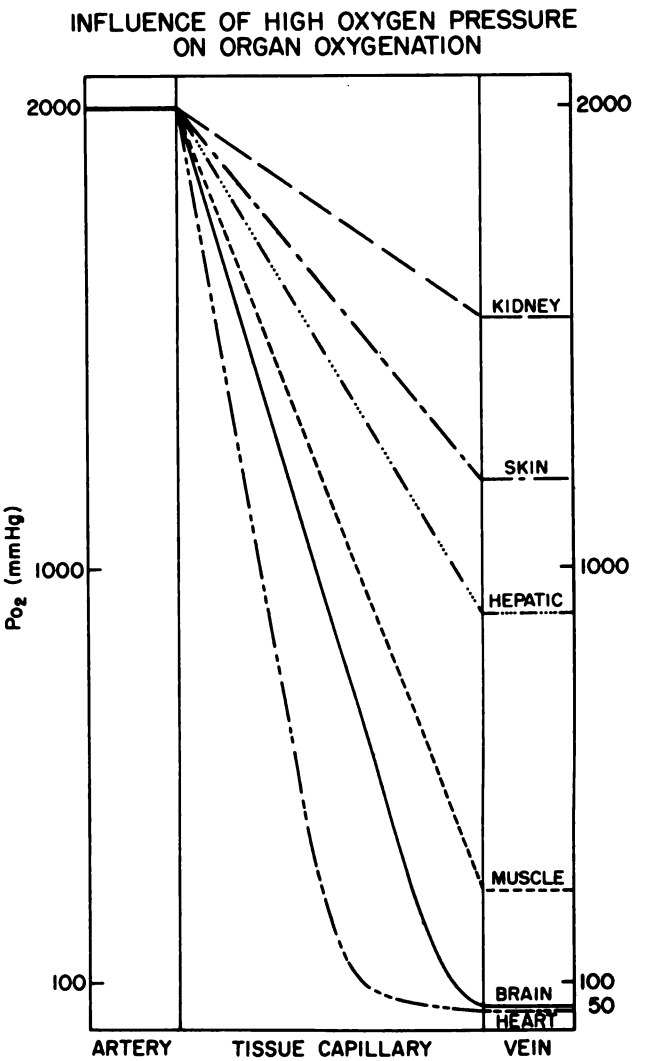

Figure 3.

ture or by both (14). The physical solubility of oxygen in blood is increased by approximately 10 per cent by a 5°C lowering of body temperature (17). This increase and the prominent depression of tissue metabolism produced by hypothermia should lead to an increase in the P_{O_2} of the cells in all tissues. Under such circumstances, a tissue in vivo should tolerate extreme reduction but not complete cessation of its blood flow. Although it should be possible to achieve a higher tissue P_{O_2} by superimposing hypothermia, narcosis, or both on the state of hyperbaric oxygenation, it is not now possible to predict how these conditons will influence cellular oxygen tolerance. Hypothermia and narcotic drugs may raise tissue P_{O_2} toward toxic levels, but conceivably they may also reduce or modify the cellular metabolic processes to such a degree that oxygen tolerance is actually increased over that in normal states (14).

INTERFERENCE WITH CARBON DIOXIDE TRANSPORT FROM THE TISSUES

In the normal air-breathing state, almost all of the oxygen supplied to tissue cells is derived from oxyhemoglobin in the capillaries. However, when oxygen is breathed at such a high partial pressure that it is supplied from physical solution, the oxyhemoglobin passes unchanged through the capillaries and serves no chemical function in oxygen transport. As hemoglobin fails to release oxygen, this also affects the transport of CO_2 from the tissues. Fully oxygenated hemoglobin is less effective than reduced hemoglobin as a buffer for hydrogen ions. For this reason, the transport of CO_2 and the hydrogen ions produced by its hydration in the tissue capillary occurs at higher than normal levels of P_{CO_2} and hydrogen-ion concentration. It is on this basis that an increase in arterial P_{O_2} above normal leads to a rise in tissue P_{CO_2} and acidity (19).

An important consideration in oxygen administration concerns the degree to which CO_2 will accumulate in the tissues (22). During air breathing at sea level, 90 per cent of the CO_2 molecules that diffuse into the tissue capillary blood become bound and are transported as bicarbonate or in the carbamino form. For each CO_2 molecule so bound, one hydrogen ion is liberated in the red cell. Again, in the normal air-breathing state the deoxygenation of hemoglobin makes available enough basic groups to transport the entire amount of CO_2 produced by a tissue with a respiratory quotient of 0.7 and to do so without change in pH (14,7c). It is this process that is inactivated in extreme hyperoxygenation. Because of the effect of oxygen on CO_2 transport, it has periodically been believed that an extreme degree of CO_2 retention should result from administration of oxygen above about 3.0 atm (18,19,23). However, the maximum effect of high oxygen pressure in decreasing the efficiency of CO_2 transport can be readily determined by nomogram (24) and by direct experiment (3). Such studies show that the maximum effect of using high oxygen pressures to completely prevent deoxygenation of hemoglobin in normal subjects is a rise of about 5 mm Hg in P_{CO_2} in such an organ as the brain (22). The central hypercapnia produced by oxygen is self-limited, but the 5 mm Hg increase in central P_{CO_2} is approximately equal to that produced by inhalation of 6 per cent CO_2 in 21 per cent oxygen at sea level. Where narcosis, inefficient pulmonary ventilation, respiratory failure, or other pathological states affecting CO_2 elimination exist, these can be expected to add their effects to the retention of CO_2 produced by failure of hemoglobin deoxygenation.

RESPIRATORY EFFECTS OF HIGH OXYGEN PRESSURES

In normal individuals, oxygen breathed at high partial pressures can affect respiration by any of several distinct mechanisms. At high altidues, with an anoxemia associated with low arterial P_{O_2}, the level of pulmonary ventilation is determined in part by exaggerated chemoreflex activity (7d). Administration of oxygen in such cases decreases chemoreflex respiratory drive, reduces pulmonary ventilation, and consequently restores arterial P_{CO_2} toward normal.

When oxygen is administered to normal subjects breathing air at sea level, a transient decrease in ventilation occurs that gives way within about a minute to a light respiratory stimulation (22,25). The slope of the ventilatory response to CO_2 inhalation decreases concurrently (22,26). Thus, stimulant and depressant effects of oxygen can coexist (27). The respiratory response to CO_2 is diminished more by oxygen inhalation at pressures of 2 and 3 atm than at 1 atm (22). In spite of its depressant effects on respiratory mechanisms, normal men breathing oxygen at a pressure of 1 atm or more demonstrate an overall increase in alveolar ventilation (22). This increased pulmonary ventilation is apparently a consequence of the rise in central P_{CO_2} and hydrogen-ion

concentration and occurs when high oxygen pressures interfere with transport of CO_2 from the tissues (22, 10e).

As mentioned elsewhere, the administration of oxygen to a patient whose respiratory mechanisms are depressed by drugs, injury, or disease should result in a prominent decrease in pulmonary ventilation, as the high P_{O_2} removes the sustaining hypoxemic, chemoreflex drive of respiration. Although respiratory depression will lead to elevation of arterial and tissue P_{CO_2} and hydrogen-ion concentration, a high degree of arterial oxygenation should nevertheless be sustained (28).

CIRCULATORY EFFECTS OF OXYGEN

At 1 atm the circulatory effects of oxygen administration to normal, resting subjects are most likely due to suppression of a "tonic" activity of peripheral chemoreceptors (28). The results at 1 atm or more include slight decreases of pulse rate and cardiac output (28, 29). The general systemic circulatory influences of the rise in central P_{CO_2} also produced by oxygen administration have not been seperately appraised, although it is known that a rise in central P_{CO_2} leads to stimulation of vagus centers (30).

Changes in the circulation of regional vascular beds are produced by oxygen. Cerebral and coronary vessels constrict as oxygen tension is raised (28, 29, 31). Similar vasoconstrictor phenomena occur in the adult eye (32, 33), the kidney (34), and possibly the skin (18). Over the years during which the vasomotor effects of oxygen have been studied, considerable emphasis has been given to whether the vasoconstrictor effect of oxygen breathing is due to a direct action of oxygen on vascular smooth muscle (7, 22). In addition to the possibility of such direct effects, the influences of central hypercapnia, neurogenic factors and local chemical influences have had to be considered. Existing information is inadequate for generalizations concerning whether important differences exist in the direct effects of oxygen on vascular beds throughout the body. Most of the available data bear on the circulation of the central nervous system, including the brain, meninges, and retina (7, 18, 22, 36). When arterial P_{CO_2} is held constant as oxygen is administered to normal men (7, 22) at 1.0 atm, cerebral vasoconstriction does not occur (see Figure 4).

It has therefore been proposed that the cerebral vasoconstriction in normal men is due to the arterial hypocapnia that accompanies the respiratory stimulation produced by oxygen (7, 14, 22, 27). Because this respiratory stimulation in itself appears to be due to a central accumulation of CO_2, the cerebral vasoconstriction during oxygen breathing can be considered an indirect result of the respiratory stimulant effects of oxygen administration.

Figure 5 shows the pattern of events that have been identified in stable-state experimentation thus far (14, 22), arranged to illustrate probable interrelationships of physiological factors.

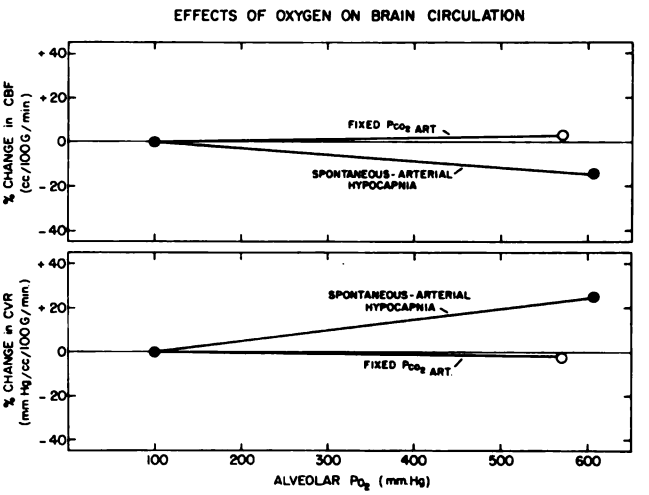

Figure 4. Effects of oxygen administration on cerebral blood flow and cerebral vascular resistance (average values in six and eight subjects [3, 35]). (From Lambertsen, C.J. [22]).

It must be emphasized that the sequences described in Figure 5 apply to normal men at rest. In pathological states of subnormal respiratory reactivity (e.g., narcotic poisoning and chronic pulmonary emphysema), the mechanisms involved should be the same but the sequence of events following oxygen administration will be different from those in normal men. The primary difference will relate to the failure to respond to the respiratory stimulant effect of CO_2 retention, and the suppression of respiration as chemoreflex activity is diminished (14, 28). In this situation, the respiratory-depressant actions of oxygen will dominate, arterial P_{CO_2} will rise, brain vessels will dilate owing to the elevated arterial P_{CO_2}, and the elevation of brain oxygen tension will be unopposed by cerebral vasoconstriction. Thus, the exposure of brain tissue to high P_{O_2} should

be greater in a depressed patient given oxygen than in a normal person.

If the subject is anoxemic and consequently has a cerebral vasodilitation, oxygen administration will restore brain vascular resistance to normal, even at a fixed arterial P_{CO_2} (28).

The existence of any type of oxygen effect on blood vessels, direct or indirect, certainly has considerable importance in attempts to deduce the probable gains in tissue oxygenation to be expected from administration of oxygen at high pressures for clinical purposes. Each tissue bed deserves to be studied experimentally for peculiarities that may negate the above-mentioned predictions. In such studies, the measurements most useful as indicators of the degree of oxygenation where some blood flow exists are the arterial and venous oxygen tensions. Because the arteriovenous P_{O_2} difference is affected both by metabolism and by blood flow, unexpected alteration of one or both of these factors can lead to gross inaccuracy in predicting the pattern of capillary oxygenation (14).

SEQUENCE OF ACUTE PHYSIOLOGICAL EFFECTS OF OXYGEN IN NORMAL MEN

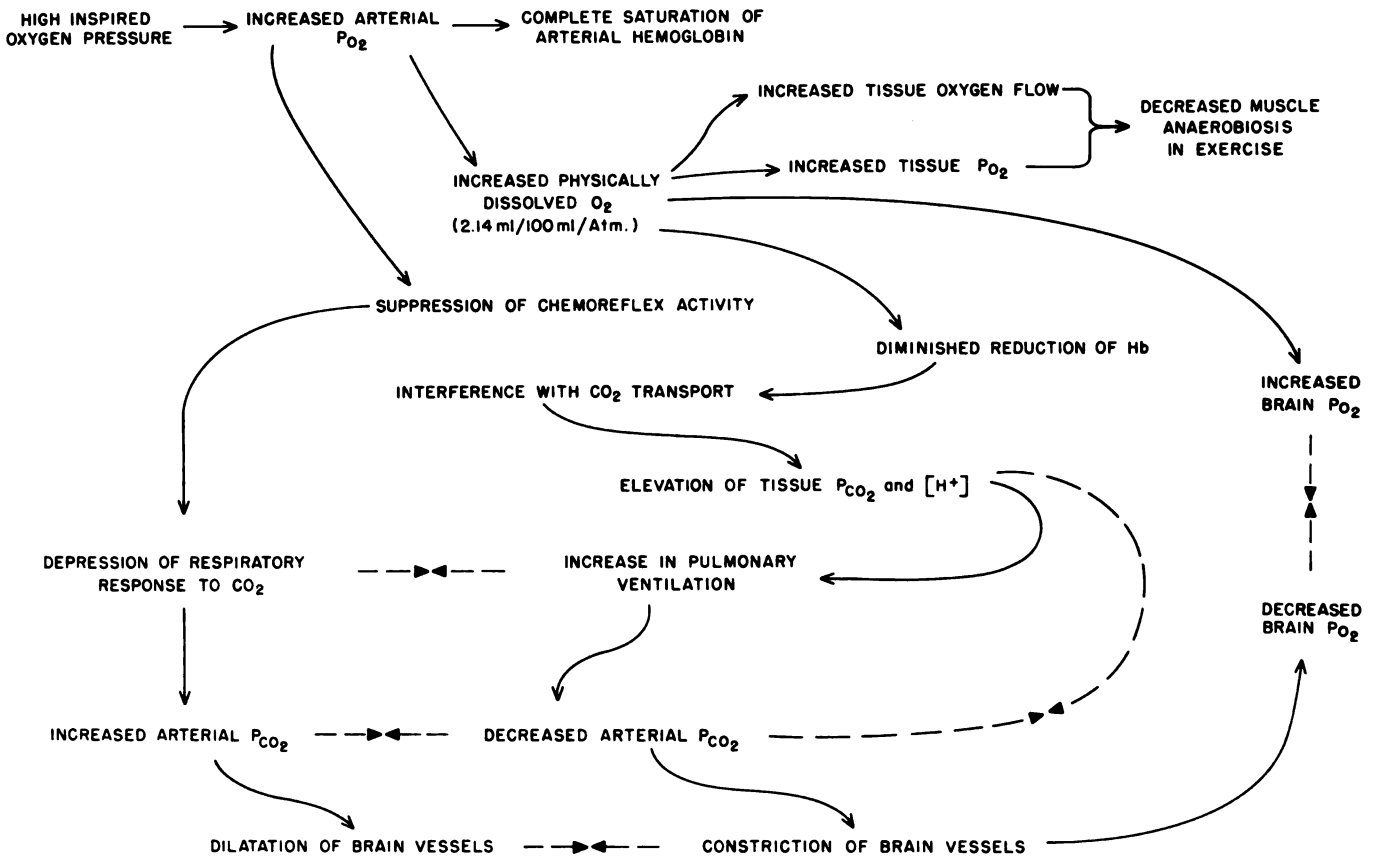

Figure 5.

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Chapter III

OXYGEN TOXICITY

C.J. Lambertsen

SUMMARY OF TOXIC EFFECTS

Inhalation of oxygen at high partial pressures can be accomplished without harm, but only if the duration of exposure is less than the "latent period" for development of a form of oxygen toxicity. Oxygen can have adverse effects on most tissues and the latent period for overt oxygen toxicity is different for various systemic forms of oxygen poisoning. The safe latent period becomes shorter as oxygen pressure is raised. It is highly variable and is modified by many known and by unknown factors. Because oxygen tolerance in man has been studied largely under special circumstances such as diving, space flight, and oxygen therapy at sea level, large areas of ignorance exist concerning the tolerance of various tissues to oxygen (Figure 1).

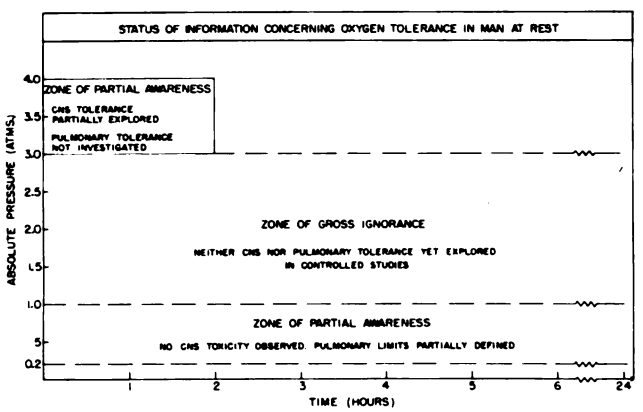

Figure 1.

Figure 2 cites several commonly recognized forms of oxygen toxicity (1) and suggests that other forms will emerge as the degree and duration of oxygen exposure are increased beyond the levels found safe for oxygen diving.

MECHANISMS OF OXYGEN TOXICITY

The causes of each of the toxic effects of oxygen on the intact animal are ultimely related to effects of oxygen on the chemical processes of cellular metabolism (2,3). Presumably any living cell can be affected, in that it has been shown that high oxygen pressures interfere with the metabolism of such varied cell types as the unicellular paramecium and the highly developed cells of the brain, liver, and heart (2,3,4,5,6, 7,8,9,10).

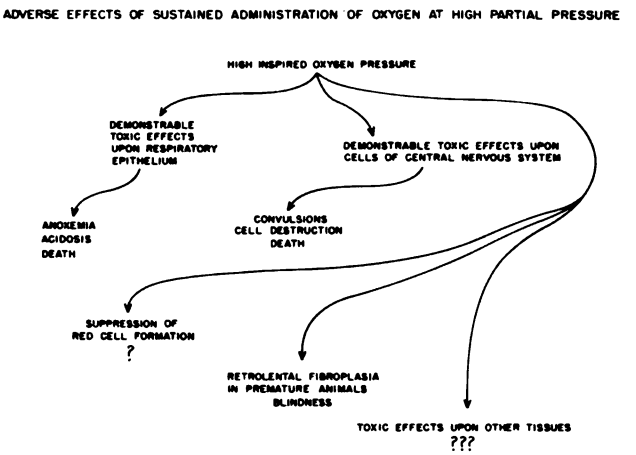

Figure 2. Above the safe level for sustained exposure, uninterrupted administration of oxygen produces adverse effects at a rate and to a degree proportional to the cellular P_{O_2}. Presumably, any cell can be affected if it is exposed to a high enough P_{O_2} for a long enough time. The most commonly recognized forms of gross oxygen toxicity include the generalized convulsions of CNS toxicity, pulmonary damage, and retinal damage in the premature infant. Other forms of toxicity should be searched for. (From Lambertsen, C.J., [1]).

The ultimate basis of the toxic action of oxygen within the cell may not be a single mechanism. There is now abundant evidence that excessive oxygen pressures interfere with oxidative reactions (2,3,8,9). However, this interference may be produced by way of more than one action and in more than one metabolic

site. One such effect is related to inactivation of the enzymes involved in the early stages of hydrogen transfer. These enzymes include several dehydrogenases that depend on sulfhydryl (SH) groups for activity (8). A second type of effect appears to be an interference with reactions involved in the tricarboxylic acid cycle. Other actions related to the formation of high-energy phosphate bonds may also be involved (3). Finally, it has been considered that the toxic action of oxygen is in fact related to an excessive production of free radicals in the affected tissues (6).

The basic mechanism of pulmonary and other superficial mucosal forms of oxygen poisoning (conjunctival, rhinopharyngeal) probably depends on the same enzymatic inhibitions as any other form of cellular oxygen intoxication. However, the consequences of pulmonary oxygen poisoning, including irritation, diffuse atelectasis, pulmonary edema and arterial anoxemia, are the peculiar result of the function played by the lungs as the fluid-gas interface of the air-breather. The initial manifestation of an oxygen effect in the conscious individual may be tracheobronchitis. Ultimately, if exposure to oxygen is extreme, failure of the functions of oxygen uptake or CO_2 removal occurs.

As in any tissue, the primary cause of oxygen toxicity in the central nervous system is an action of oxygen on cellular metabolic processes. In the neurons of the central nervous system these metabolic reactions are somehow linked to the biophysical processes involved in the generation of electrical impulses. Thus, cellular oxygen toxicity has as its early manifestation an uncoordinated form of electrical activity, the generalized convulsion. While the convulsion itself is not inherently harmful, if it is ignored or masked and oxygen administration is continued, lasting neurological damage can be expected to occur.

PULMONARY OXYGEN TOXICITY

The harmful effects of oxygen on the lung membranes and lung function are due both to chemical actions related to the increase in inspired P_{O_2} and to the physical consequences of excluding the inert carrier gas from the pulmonary passages. The following paragraphs outline the manner in which these distinct mechanisms interact to produce a composite biochemical-physical derangement of pulmonary function.

Chemical Effects of Oxygen on the Lungs

Pulmonary Tolerance to Oxygen Partial Pressures Not Greater than 1 atm

The rate of development and the degree of chemical damage to the respiratory passages and conjunctivae is proportional both to the dose of oxygen (P_{O_2}) and to the duration of exposure. The term "irritation" is a misnomer, in that the effect is undoubtedly a distinct form of biochemical toxicity involving the cells of all the exposed mucosal or serous surfaces. The effects upon the respiratory tract appear not only to have a threshold dose, but also to begin only after a long latent period (10, 11). Although the concept of a safe latent period is probably valid even on theoretical grounds, it must be realized that objective means of determining the presence and degree of pulmonary or epithelial irritation are not adequate for appraising pulmonary oxygen toxicity until it has become subjectively prominent.

Figure 3 shows most of the available information concerning pulmonary oxygen tolerance in man. In studies related to the evolving program for manned spaceflight, pure oxygen at a reduced ambient pressure of about 250 mm Hg has been breathed by normal men for up to 30 days without chemical or other pulmonary changes (12, 13, 14). Pulmonary, nasopharyngeal, and conjunctival irritation have been clearly observed in men exposed to pure oxygen at sea level (760 mm Hg) for 24 hours (11). Exposure for longer periods at the same partial pressure of oxygen has led to severe bronchopneumonia (10, 15, 16). An excellent summary of such studies is included in a symposium on gaseous environment for manned spacecraft (16).

Pulmonary Tolerance to Oxygen Partial Pressures Greater than 1 atm

Chemical damage of the lungs by oxygen occurs more and more rapidly as the partial pressure of oxygen is increased above 1 atm (10). Studies of this form of oxygen toxicity at increased ambient pressure have thus far been limited almost entirely to small animals. In spite of its great importance in diving and medicine, practically no information is available concerning the latency of pulmonary effects in man of breathing oxygen at pressures above 1 atmosphere. Because it is possible that oxygen will not produce central-nervous-system

or other forms of toxicity in resting men until the oxygen pressure is much higher than 1 atm, pulmonary oxygen toxicity may eventually prove to be the limiting factor in exposures of resting subjects or patients to oxygen at pressures between 1 and 3 atm. At present, in spite of the demonstrated advantage of intermittent exposures in delaying convulsions and death of animals from oxygen poisoning (18, 19), it is possible that pulmonary effects of very frequent administration of oxygen may be cumulative.

Physical Effects of Oxygen Breathing

It is emphasized above that breathing 100 per cent oxygen produces no biochemical toxicity when the ambient pressure is maintained low enough so that the alveolar P_{O_2} is not greater than normal (11, 12, 13, 14, 20). This situation exists in aviation and in an oxygen-filled altitude chamber or spacecraft where the total environmental pressure is 187 mm Hg. In such a state the alveolar gas composition will be: P_{H_2O} = 47 mm Hg, P_{CO_2} = 40 mm Hg, and P_{O_2} = 100 mm Hg. Although chemical oxygen toxicity under these circumstances is inconceivable, it has been considered possible for random blockage of bronchioles, even by normal secretions, to lead to a diffuse, progressive, and eventually severe pulmonary atelectasis due to rapid and complete absorbtion of the gaseous water, carbon dioxide, and oxygen from obstructed alveoli (11, 20, 21, 22). This physical consequence of oxygen breathing has been demostrated in aviators exposed to acceleration while breathing pure oxygen (23, 24) and studied in isolated lungs (22), but does not appear to occur in normal persons exposed to essentially pure oxygen at low ambient pressures for as long as 30 days (12, 13, 14). (Pulmonary atelectasis remains as a possible physical complication of oxygen breathing when the inspired P_{O_2} is high enough to produce a chemical pulmonary irritation, or when another factor, such as pulmonary infection, interferes with the patency of the terminal pulmonary passages (20, 59).)

Interaction of Physical and Chemical Factors to Influence the Severity of Pulmonary Oxygen Toxicity

Chemical effects on the pulmonary membranes, leading to hyperemia, edema, and loss of fluid into the air passages (10) also predispose the individual to pulmonary atelectasis by contributing to obstruction of the alveoli. Following obstruction, the absorption of oxygen, carbon

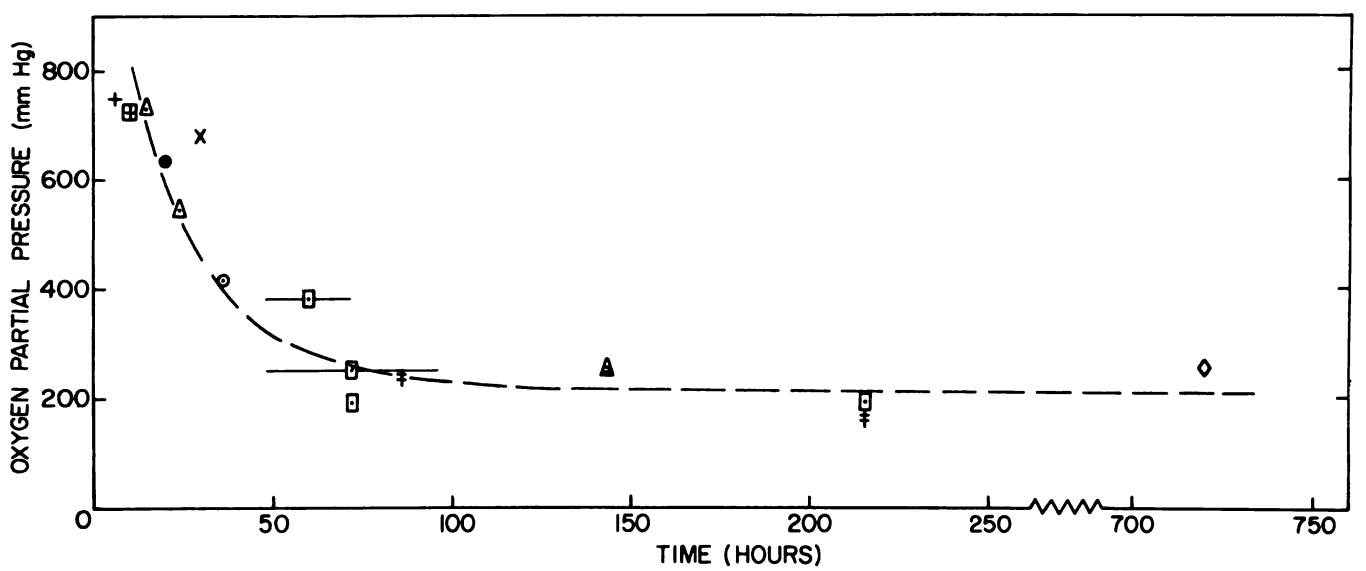

Figure 3. Pulmonary tolerance to oxygen. The symbols are those used by Welch and indicate a number of different studies described in a review of pulmonary effects of oxygen (17). Thus, the plotted points are taken from many studies and differ in the number of subjects involved. Some represent the average time of onset of symptoms and some the earliest indication of pulmonary irritation; the symptoms used to define the end points also vary in the different studies. (Data from Welch, et al. [17] modified by author [1]).

dioxide, and water proceeds rapidly. The physical atelectasis, but presumably not the chemical toxicity, can be partially delayed by periodic artificial insufflation of the lungs and by administration of atropine to diminish pulmonary secretions and bronchospasm (25). The composite inflammatory-atelectatic response to high oxygen pressures can be exaggerated by administration of carbon dioxide, sympathomimetic amines, and corticosteroids (26, 27, 28, 29).

End Results of Pulmonary Oxygen Toxicity

While short periods of oxygen breathing produce no demonstrable harm, continuous exposure for long periods must lead to severe damage. Limitation of alveolar ventilation (froth in airways), collapse of alveoli (atelectasis), passage of blood through non-ventilated alveoli, and slowed diffusion of gases between alveoli and blood (pulmonary edema) should all interfere with pulmonary gas exchange. This will be true even when oxygen at high pressures is breathed. The pulmonary damage leads progressively to a lowering of arterial P_{O_2}, elevation of arterial and tissue P_{CO_2}, and death from anoxia and acidosis. Because the inspired and alveolar P_{O_2} are initially high, the pulmonary damage may lead to CO_2 retention and acidosis, even while arterial oxygenation is sustained above the value normal for sea-level existence. Paradoxically, in this situation where there is a high alveolar-arterial P_{O_2} gradient, the patient may be kept alive only in the continued presence of the excessive P_{O_2} that is causing his death by progressive pulmonary damage. Removal to an air-breathing situation at sea level, where the oxygen gradient is inadequate for gas exchange, results in rapid death from anoxia. Presumably, oxygen therapy at sea level also would compound the toxicity.

CENTRAL-NERVOUS-SYSTEM OXYGEN TOXICITY

Convulsions Produced by Oxygen

The overt expression of CNS oxygen toxicity in vertebrates is a generalized convulsion. In man this resembles the convulsion of grand mal epilepsy (10). The convulsion is usually, but not always, preceded by the occurrence of localized muscular twitching, especially about the eyes, mouth, and forehead (1, 10, 30). Small muscles of the hands may also be involved, and an incoordination of diaphragm activity in respiration may occur. Once they begin, these phenomena increase in severity over a period that may vary from a few minutes to nearly an hour, with essentially clear conciousness being retained during the development of twitches. Eventually, an abrupt spread of excitation occurs, and the rigid "tonic" phase of the convulsion begins. This phase is accompanied by an abrupt loss of conciousness. Respiration ceases at this point and does not begin again until muscular coordination returns. This tonic phase usually lasts for about 30 seconds and is followed by vigorous clonic contractions of the muscle groups of the head, neck, trunk, and limbs which become progressively less violent over about one minute. As the uncoordinated motor activity stops, rhythmic respiration can again proceed. Following the convulsion, hyperpnea is marked, owing to accumulation of metabolic products during the period of vigorous muscular activity concurrent with breath holding. Respiration is complicated by soft-tissue obstruction and by the extensive secretions that result from what is probably an autonomic component of the central-nervous-system convulsive activity. Because a high alveolar P_{O_2} persists during the convulsive apnea, the individual remains well oxygenated throughout the convulsion itself. This is in sharp contradistinction to the epileptic patient who convulses while breathing air at sea level.

The Safe Latent Period

The convulsions of oxygen toxicity occur only after a "safe latent period" whose length is inversely proportional to the pressure of inspired oxygen (Figure 4). Considering the many factors that can affect not only the dose of oxygen at the cellular level, but also the chemical action of oxygen on intra-cellular enzymes and the inherent excitability of central neurons, it is not surprising that this latent period for the development of gross convulsions varies greatly from person to person and even within one person (10, 30, 31). Improved information concerning practical extension of the safe latent period is crucial to exploitation of high oxygen pressures for therapeutic purposes.

The Relation of Biochemical Effects to Convulsive Phenomena

Reference is often made to observations that, at a particular high oxygen pressure, the latent period for development of convulsions

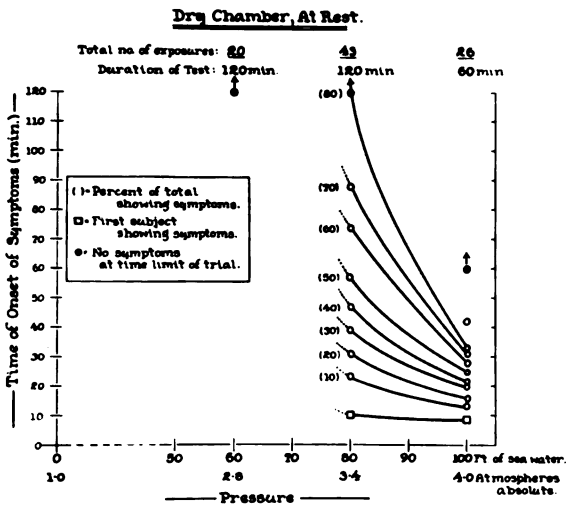

Figure 4. Latent period for development of oxygen toxicity by normal men at rest in a dry pressure chamber. Note the variability in times of onset of symptoms, even in the normal, resting subjects (30). Tolerance to the CNS effects of oxygen is decreased by factors such as increased carbon dioxide tension, underventilation, exercise, and certain drugs. (Graph prepared by author for Symposium on Underwater Physiology (18) from protocols of data obtained at U.S. Naval Experimental Diving Unit, Washington, D.C. [31]).

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in the intact animal is less than the time required for enzymic inhibition to alter metabolism in vitro (10,32,34). This is not at all surprising. It may indicate that extremely subtle chemical changes can affect the electrical activity of the highly organized central nervous system. It is also possible that effects of oxygen on membranes, not directly paralleled by changes in the oxidative metabolism of cell systems, are responsible for the convulsions. The discrepancy between in vitro and in vivo studies is seen to be still more pronounced when it is recognized that at a given elevated ambient P_{O_2} the cells of an in vitro homogenate are exposed to a rather uniform oxygen tension, whereas only a very small portion of the cells in the brain of an intact animal are exposed to this high oxygen tension (35,36).

Detection of Incipient Toxicity

Determination of the limits of oxygen tolerance of various organs is of the greatest practical importance to the use of oxygen in medicine. This need is especially great for pressures between 1 and 3 atmospheres at which there is little information concerning even pulmonary toxicity or central-nervous-system effects of oxygen (1). To determine these limits, human subjects must be studied until objective manifestations of toxicity appear. Also, to determine the range of variability in the population, each subject must develop signs of toxicity. Because convulsions and the risk of pulmonary damage are deterrents to studies of tolerance to hyperbaric oxygenation, it is necessary to develop objective indexes of early central-nervous-system or pulmonary involvement. Past suggestions for anticipating central-nervous-system toxicity, none of which has emerged as satisfactory, have included monitoring the electroencephalogram for preconvulsive signs (37) and monitoring the pulse rate (38). An index of early pulmonary change is also necessary, and thus far no better index than vital capacity has emerged. Maximal breathing capacity should be superior to simpler measurements such as vital capacity, although the latter is certainly useful. Alveolar arterial P_{O_2} difference does not appear to be a sensitive early warning.

TOXIC EFFECTS OF OXYGEN UPON OTHER TISSUES

There is a general tendency to consider that the brain cells are more "sensitive" to oxygen poisoning than are the cells of other organs or tissues. This is not always true. Because gross convulsions occur in vertebrates exposed to high oxygen pressures, it is quite natural to focus attention on the central nervous system. However, disruption of central-nervous-system activity is, by the very nature of the convulsion as a readily observable result, more easily detected than changes in vascular, endocrine, hepatic, renal, pancreatic, or even cardiac function. Toxic actions of oxygen probably can occur in most of the body cells that contain susceptible enzymes. In vitro studies have demonstrated that high oxygen pressures affect the metabolism of the lung, heart, liver, and testis (2,10,32,40). However, the effects of these actions may be minute when the pulmonary changes or the gross, convulsive expressions of central-nervous-system toxicity occur.

The Eye

The eye, as a component of the central nervous system, deserves special attention. The retinal circulation of premature infants is affected even by increased oxygen tensions no

greater than 1 atmosphere (33, 41, 42). Retrolental fibroplasia, causally linked to the therapeutic use of oxygen, involves extensive vascular obliteration and fibroblastic infiltration in the retina, resulting in permanent blindness. This unfortunate effect appears clearly related to the immaturity of the fetus, inasmuch as retinal damage does not occur in fully developed infants or children, and even oxygen pressure of many atmospheres does not appear to produce permanent residual disturbances in adults. Oxygen does seem to cause retinal vascoconstriction in mature humans (43, 44), and even at atmospheric pressure may lead to constriction of the visual fields (45).

The Blood

The recognized acceleration of hemoglobin formation at lower than normal P_{O_2} (46) raises the question of whether high tensions of inspired oxygen will lead to depression of erythropoiesis and hemoglobin synthesis. No reliable information concerning this question is yet available in man, although studies in mice are suggestive that such depression occurs (47). For short exposures, this should have little significance. In view of the relatively short latent period of central-nervous-system or pulmonary toxicity, determination of the effects of oxygen on bone marrow may prove impractical. Exposures to 0.5 atm of oxygen for 14 days have provided tenative indications, not that hemoglobin synthesis is suppressed, but that hemolysis may be accelerated (12). This may be exaggerated in some forms of congenital anemia (12).

Blood Vessels

Next to the respiratory passages, the cells exposed to the highest oxygen tensions are those lining the arterial tree and the left chambers of the heart. There is no information concerning the toxic effects of oxygen on the intima. If there are such effects, experience over many years of oxygen breathing in diving indicates that they should have little practical importance. It is of considerable theoretical and practical interest, however, whether oxygen at high pressures produces a direct constrictor effect on vascular smooth muscle. If a prominent direct constrictor action of oxygen exists, leading to the decreased blood flow seen in such organs as the brain, kidney, and eye, it would tend to limit the degree of exposure of tissues to elevated P_{O_2} (35).

FACTORS INFLUENCING THE TOXICITY OF OXYGEN

Carbon Dioxide

The safe latent period of oxygen breathing before the development of oxygen convulsions is prolonged by hyperventilation (10) and shortened by administration of low concentrations of CO_2 (10, 30). The basis for these effects is probably the indirect result of the influence of hypocapnia and hypercapnia on brain circulation, and hence on the dose of oxygen (P_{O_2}) delivered to brain cells (35, 36). The level of arterial P_{CO_2} accompanying exposure to high arterial P_{O_2} is of great practical importance, in that changes in brain blood flow are accompanied by large alterations of the P_{O_2} in the capillaries of the central nervous system (35) (Figure 5). In contrast

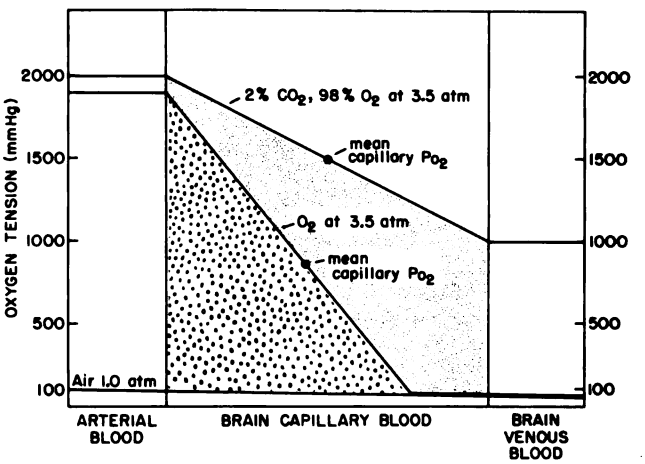

Figure 5. Effects of carbon dioxide upon cerebral venous and cerebral capillary P_{O_2} at high levels of arterial P_{O_2}. When oxygen is breathed at 3.5 atmospheres pressure, over 6.5 ml O_2/100 ml blood are present in physical solution. This is more than the normal arteriovenous O_2 difference across the brain. However, diffusion of oxygen from the capillary, together with additional oxygen loss related to decreased brain blood flow, results in a venous blood P_{O_2} that is only slightly elevated above the value found in air breathing at sea level (36). Addition of CO_2 to the inspired oxygen overcomes the cerebral vasoconstriction, speeds the flow of blood through the brain capillary, and results in less oxygen being removed per unit of capillary blood flow. The consequent extreme rise in capillary and venous P_{O_2} can be presumed to increase the dose of oxygen to which brain cells are exposed and the number of enzyme molecules exposed to toxic pressure of oxygen (1, 35).

to the situation while breathing air, it would be expected that breath holding while breathing oxygen at high ambient pressure would increase the dose of oxygen delivered to the brain.

It has often been proposed that CO_2 may predispose to oxygen toxicity by an action other than vasodilatation and increasing the exposure of brain cells to high P_{O_2}; however, no such effect has yet been demonstrated. The addition of CO_2 to oxygen at high pressure does not exaggerate the observable influences of oxygen on metabolizing tissues in vitro (5) or increase the toxic effects of oxygen on isolated nerve fibers (49). Although CO_2 itself is a convulsant, tensions of inspired carbon dioxide that are high enough to produce unconsciousness (20 to 30 per cent) are required to produce convulsions in normal subjects breathing air or oxygen at sea level (50). At very high partial pressures, depressant actions of CO_2 actually suppress oxygen convulsions (51).

Exercise

The rate of development of oxygen convulsions is also increased by exercise, a factor of great practical importance in diving. The basis for this influence is not yet established. Electrical activity of cerebral, cortical and other central neurons is increased in exercise, but how the exaggerated bombardment of neural systems may relate to the biochemical derangement is not at all clear. Understanding of the influence of exercise on oxygen tolerance (10, 30) is complicated by the possibility that, in exercise, a superimposed CO_2 retention may result from such factors as: interference with alveolar ventilation due to the increased resistance to gas flow in respiratory passages or in external breathing apparatus; the depression of alveolar ventilation produced by physiological effects of high oxygen pressures per se (52, 58); and inadequate methods of CO_2 absorption when closed rebreathing systems are used. At least one of these possibilities has existed in every study of oxygen tolerance in exercise (30, 31, 53), and it is therefore not yet known whether the neurophysiological effects of muscular exercise or an increased brain P_{O_2} due to some interference with CO_2 elimination is responsible for the shortened latent period for oxygen convulsions in exercise (54).

Other Factors

Still other factors are known to modify the duration of the safe latent period before the development of oxygen convulsions. Immersion (apparently in the vertical position) appears to shorten the latent period (30, 31), but it is not known whether this is an experimental artifact related to a circulatory adjustment due to the external hydrostatic forces, or is secondary to ventilatory changes.

Cold-blooded animals have long latent periods at low body temperatures, but become more susceptible to oxygen toxicity when their body temperature is raised (10). This finding has important implications for man and for warm-blooded animals exposed simultaneously to hyperoxia and hypothermia

Interruption of the exposure to high oxygen pressures provides a highly useful method of extending oxygen tolerance, apparently because the rate of recovery from developing toxicity is much higher than the rate of its initial development (18, 19).

RESIDUAL EFFECTS OF OXYGEN BREATHING

If the conditions of pressure and duration of an oxygen exposure do not cause acute manifestations of central-nervous-system or pulmonary toxicity, it is unlikely that important residual harmful effects will be produced. This is well illustrated by the extensive and successful employment of self-contained oxygen rebreathing apparatus by Italian, British, and U.S. divers in World War II, with exposures to high oxygen tensions repeated almost daily for many months.

Central Nervous System

Even the convulsions produced in man by exposure to oxygen pressures above 3 atm do not necessarily produce central-nervous-system damage under laboratory or treatment conditions. No physical, physiological, or psychometric evidence of harm, either acute or residual, has been detected in normal subjects (1, 30) or in several schizophrenic patients who were subjected to repeated, biweekly oxygen convulsions at an inspired oxygen pressure of 4 atm (J.E. Ewing, M.W. Stroud, and C.J. Lambertsen, unpublished observations). The convulsion apparently is a sensitive and early manifestation of central-nervous-system toxicity. Most likely, the development of toxic effects within central neurons has therefore not reached an irreversible stage by the time the diffuse electrical discharge of the convulsion occurs.

Even if it does not produce neuronal damage a convulsion due to oxygen can conceivably result in the same forms of physical trauma

associated with epileptic seizures or the convulsions produced by electroshock. Although recovery of conciousness is normally prompt, an instance of persistent convulsions has been reported (55). Residual neurological damage occurs in rats following severe oxygen poisoning (10,48), but has not been described in larger animals or man (37). Certainly the continuous exposure to high P_{O_2} beyond the latent period for convulsive seizures will eventually lead to irreversible effects, cell damage, and death. This is true even when the overt evidence of developing toxicity, the convulsion, is prevented by depressant drugs.

Pulmonary

Residual and cumulative effects of oxygen damage to the lungs should be expected to follow repeated extreme damage of the lungs by oxygen, as for any other form of chemical pulmonary injury (60). However, after injurious exposure that did not lead to the death of the subjects, essentially complete recovery occurred (15,17,57).

PREVENTION OF OXYGEN TOXICITY

Complete prevention of the direct, intracellular, enzymatic phenomena of oxygen toxicity is not now possible. However, it has proven feasible to prolong the latent period for the development of direct toxic effects both in vitro and in vivo. This has been accomplished especially by altering the concentrations of trace metals in the cells and administering agents that provide sulfhydryl groups (2,6,32). These protective influences are limited in degree and duration, however, and, in view of the fundamental importance of the oxidative reactions involved, it is very likely that it will never become possible to prevent cellular oxygen toxicity from occurring in the presence of high cellular oxygen tension.

At present, the practical approach to preventing cellular oxygen toxicity is to avoid excessive pressure and duration of exposure to oxygen. This is not the sole approach when dealing with the development of toxicity in the intact animal or subject. It has been possible to extend the safe period at a particular raised oxygen pressure. Hyperventilation has been shown to extend central-nervous-system oxygen tolerance in animals (10). Most likely, this effect is accomplished by reducing cellular oxygen tensions in the brain, since hyperventilation leads to arterial hypocapnia and cerebral vasoconstriction, which act to lower mean brain P_{O_2} and decrease the mass of brain tissue exposed to the toxic oxygen tensions (35,36). This gain must not be considered to be a general increase in tolerance; it merely indicates that development of convulsions can be delayed. Some of the brain cells, near the arterial ends of capillaries, remain exposed to P_{O_2} levels nearly as high as arterial levels (35), which suggests that toxic effects of oxygen on these cells would not be diminished by hyperventilation. Convulsions still develop eventually under these circumstances, even when only diffuse parts of the brain-cell mass are exposed to very high oxygen pressures. Recognition of this raised the question of whether toxic effects on a relatively minute fraction of an otherwise unaffected brain-cell population could produce the oxygen convulsion (35,36).

Use of drugs to prevent the central-nervous-system form of oxygen toxicity has been investigated extensively over the past several decades (2,8,10,32,56). The motor convulsions produced by excessive oxygen exposure can, of course, be completely prevented by central depressant agents, or by neuromuscular blocking agents. This does not, however, indicate that the underlying cause of the convulsion, the direct cellular poisoning by oxygen, has been prevented. It can be expected that cellular and enzymatic oxygen toxicity will continue to develop in animals and man, even if neither the electrical nor the motor expression of central-nervous-system toxicity is allowed to appear. It is therefore very important to recognize that ability to prevent oxygen convulsions does not imply ability to prevent oxygen toxicity.

One of the most practical measures available for extending ability to use high oxygen pressures is the carefully scheduled alternation of exposure to high and normal levels of P_{O_2} (1,18,19). Such purposeful, brief interruption of exposure to inspired oxygen of high pressures markedly extends oxygen tolerance in animals, and indicates that the rate of recovery from the direct toxic actions of oxygen is considerably greater than the rate of development of overt oxygen toxicity. The alternation of low and high inspired P_{O_2} offers a practical approach to increasing the duration of the total exposure to a particular high oxygen pressure within a given time period. Unfortunately, no systematic extension of this principle to man has been carried out. For its full exploitation, the

optimal relationships between the duration of exposure to high P_{O_2} and the length of the interruption of the exposure must be determined for various levels of inspired P_{O_2}. In addition, it must be recognized that the relationships of rate of development of toxicity and of recovery from it will undoubtedly be different for the pulmonary, the central-nervous-system, and other forms of oxygen toxicity (1). Study of these factors of latency and the influences of intermittent exposure should provide early and great returns in extending the clinical usefulness of oxygen at high pressure.

TREATMENT OF OXYGEN POISONING

Owing to the extreme rapidity of the metabolic utilization of oxygen in the cell, the P_{O_2} at a particular intracellular location should fall to natural, nontoxic levels within a few minutes of a pulmonary "oxygen washout time." Return to normal pressure of inspired oxygen is therefore the primary measure for aborting or interrupting the development of acute oxygen toxicity. There have been a few instances in man in which the toxicity has progressed to such a degree that convulsions occur a few seconds after the removal of the oxygen mask from the subject.

If a convulsion occurs, it is necessary to prevent the patient from injuring himself during the vigorous and generalized clonic contractions. Excessive restraint should be avoided. During the tonic phase at the onset of the convulsion the head becomes hyperextended and the lower jaw is strongly depressed so that the jaws are separated. During this period of about 10 seconds, a soft but firm "bite block" such as a padded tongue depressor can easily be inserted to prevent chewing of the tongue during the subsequent clonic jaw clamping.

During both the tonic and clonic phases there is severe interference with pulmonary ventilation, probably including laryngospasm as well as soft tissue oropharyngeal obstruction and incoordination of thoracic movements. It is therefore extremely important to avoid decompression during any part of the convulsion, since expanding pulmonary gas would then rupture the lung and produce a possibly fatal pulmonary embolism. Rhythmic breathing returns as the clonic convulsion ceases. It is at this stage that attention to the airway is especially important, At this time it is possible to proceed with decompression.

Following a convulsion and return to air breathing, the postconvulsive depression wears off and conciousness usually returns over a period of 5 to 10 minutes. During the state of gradually returning consciousness, the patient may be irrational and will at least require reassurance and gentle restraint. At times consciousness returns abruptly and the patient shows surprising mental clarity. Headache or nausea may occur, and muscular fatigue is to be expected. Consciousness and normal central-nervous-system function should return within a few minutes to an hour after the convulsion (unpublished observations of the author).

No specific measures are known to provide rational bases for treating cells damaged by the toxic process. If there are severe central-nervous-system effects and sustained convulsive or neurologic damage, these consequences should be managed as for other forms of convulsions or CNS injury. Until there has been more extensive study of the general pathology of central-nervous-system oxygen toxicity, recommendation for other than supportive therapy will not be meaningful.

Damage to the lungs leads to diffuse atelectasis, pulmonary edema, and bronchopneumonia. Because death will result from failure of pulmonary gas exchange, such measures as artificial ventilation and prophylactic use of antibiotics should aid the less seriously injured individual. In choosing the gas to ventilate the lungs, a concentration of oxygen not exceeding 60 per cent at sea level should be used, because higher tensions of oxygen would presumably aggravate the existing pulmonary damage (11). If the lungs of a patient have been so severely affected by pulmonary oxygen poisoning that anoxemia develops even at inspired oxygen pressures of several atmospheres, a quandry will exist that will certainly end only with the death of the patient (1). Treatment by lowering the oxygen pressure will result in further anoxemia. Treatment of the anoxemia with oxygen at normal or higher pressures will result in further pulmonary damage. Therefore, if such mechanical approaches as use of a heart-lung machine for extrapulmonary oxygenation are ignored as being impractical, no approach to reversal of severe pulmonary damage now appears feasible. It is on this basis that the pulmonary limits of oxygen tolerance deserve the most serious attention in therapy with high oxygen pressures.

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Chapter IV

THE PHYSIOLOGICAL BASIS OF HYPERBARIC THERAPY

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The physiological effects of high pressure, like those of most abnormal environments, are predominantly detrimental to man. Turning any of them to therapeutic advantage requires keeping the desired action within suitable limits while avoiding harm from simultaneous effects. Existing knowledge concerning the physiology of high pressure is far from complete, but making use of what is known can do much toward placing clinical applications of this entity on a logical and reasonably safe basis. The general effects and major problems of hyperbaric exposure are discussed further in subsequent chapters. The purpose of this chapter is to furnish more detailed information concerning physiological factors underlying potential therapeutic applications.

IMPROVEMENT OF OXYGENATION

The area of greatest recent interest in high-pressure therapy concerns elevation of inspiratory oxygen partial pressure as a means of overcoming or preventing various types of hypoxia. Although the laws of Dalton and Henry provide the physical basis for this application, they do not go very far by themselves toward providing a reasonable physiological basis. It is neither accurate nor useful, for example, to suggest that elevating the inspiratory oxygen pressure by a factor of 15 will elevate oxygen pressures by the same factor throughout the body. The relevant picture is complicated by the properties of hemoglobin, by rates of oxygen uptake and blood flow, and by uncertainties concerning the diffusion of oxygen from capillaries into tissues—to say nothing of imperfect knowledge of the pathologic physiology of the disease states at issue.

Many experiments and clinical studies remain to be conducted before true indications for hyperbaric therapy can be defined. Nevertheless, more than a few perplexities and false hopes have arisen purely from failure to apply available physiologic knowledge. Perhaps more important at this juncture, analysis of the problem in terms of such knowledge helps to define the real areas of uncertainty and suggests crucial experiments to illuminate them. Answers to a few fundamental questions will do much to improve our understanding and our ability to make useful predictions.

Understanding of the potential benefits and limitations of oxygen administered under high ambient pressure (OHP) is best sought by considering the stages in the process of oxygen transfer, the disorders in each that can result in hypoxia, and the beneficial effects that increased oxygen pressure may produce.

Alveolar Oxygen Pressure

Fresh air contains 20.94 per cent oxygen, the remainder being mainly nitrogen. At sea level with a barometric pressure of 760 mm Hg, the oxygen partial pressure (P_{O_2}) of inspired air is thus nearly 0.21 x 760 = 159.6 mm Hg. When air is inspired, however, it rapidly becomes saturated with water vapor at body temperature. Water vapor then accounts for 47 mm Hg of the total pressure, leaving only 760 - 47 = 713 mm Hg for the combined pressures of oxygen and other "true" gases. In the alveoli, pulmonary capillary blood removes oxygen and adds carbon dioxide in amounts governed by the metabolic activity of the body. The final composition of alveolar gas depends upon the relationship between these amounts and the volume of alveolar ventilation. With normal ventilation, the resulting P_{O_2} is a few mm Hg above 100 while the P_{CO_2} is about 40 mm Hg. Nitrogen, with small amounts of other inert gases that seldom need to be considered separately from nitrogen, then accounts for the remainder, or about 570 mm Hg. If alveolar ventilation is inadequate, the proportion of oxygen extracted from alveolar gas increases and P_{O_2} falls while P_{CO_2} increases.

Nitrogen continues to make up whatever remains of the total gas pressure.

Oxygen supplied from commercial cylinders is generally more than 99.5 per cent pure, so the inspiratory P_{O_2} at standard barometric pressure can potentially be elevated at least to .995 x 760 = 756.2 mm Hg— slightly less than five times the P_{O_2} of fresh air. Ventilation of the lungs with "pure" oxygen will shortly drop the alveolar nitrogen pressure to a negligible level. With only water vapor (47 mm Hg at 37°C), O_2 and CO_2 present in the alveoli, the P_{O_2} would be equal to the total alveolar gas pressure (760 - 47 = 713 mm Hg) minus the P_{CO_2}. With a normal PCO_2 of 40 mm Hg, an alveolar P_{O_2} of 713 - 40 = 673 is thus possible. Elevating the inspiratory O_2 concentration not quite fivefold thus can yield nearly a sevenfold increase in alveolar P_{O_2} over the air-breathing value of about 100 mm Hg.

Hypoxia due to inadequate pulmonary ventilation is best treated by restoring normal ventilation; unless this is done, respiratory acidosis will develop. However, the hypoxia itself can be overcome at any level of ventilation simply by replacing the alveolar nitrogen with oxygen. Once this is accomplished, a normal man's lungs can hold enough oxygen to supply resting metabolic needs for 10 minutes or longer without further ventilation (1). As oxygen is taken up, the volume present decreases, but the P_{O_2} falls only to the relatively small extent that the P_{CO_2} rises. In the procedure misnamed "diffusion respiration," a source of additional oxygen is provided, and oxygenation can be maintained almost indefinitely with no respiratory movements whatever (2). In both cases, accumulation of carbon dioxide becomes the limiting factor. This example illustrates an important principle: Prevention or correction of hypoxia does not in itself provide any direct benefit in problems of carbon dioxide retention. On the contrary, administration of oxygen, especially at high pressure, is likely to compound carbon dioxide problems to some degree.

A common cause of hypoxia in pulmonary disease is uneven distribution of alveolar ventilation and blood flow. In alveoli where ventilation is deficient compared to perfusion, the P_{O_2} falls to a low level, and poorly oxygenated blood is contributed to the arterial stream. At the same time, ventilation is in effect wasted upon other alveoli where blood flow is deficient compared to ventilation. The overventilation of these alveoli does almost nothing to compensate for hypoxia due to underventilation of the others.

The same mechanism may contribute significantly to the vicious circle of hypoxia in shock. It is postulated (3) that low pulmonary arterial pressure leaves higher portions of the lung with little perfusion while alveoli of the dependent portions must attempt to provide total metabolic gas exchange with small relative ventilation. Since the well-ventilated but unperfused alveoli constitute respiratory dead space, increases in ventilation will also largely be wasted.

Just as oxygenation can be maintained with oxygen in the absence of respiratory movements, the P_{O_2} of underventilated but open alveoli can be kept high with increased concentrations of oxygen at normal pressure. On the other hand, closure, collapse, or consolidation of large numbers of still-perfused alveoli produce a form of hypoxia in which oxygen at 1 atmosphere may be of little benefit. Blood flow from such alveoli constitutes venous admixture—in effect, a right-to-left shunt. This is likely to be the main mechanism accounting for hypoxia in conditions such as pneumonic consolidation and respiratory distress syndrome of the newborn. It can also be implicated in the atelectasis or frank lung damage produced by oxygen itself. Failure to relieve hypoxia with high concentrations at 1 atmosphere is almost diagnostic of shunt effects, and these form the principal indication for OHP in hypoxia of pulmonary origin.

Although relatively few pulmonary diseases require recourse to high pressure, it is necessary to consider the alveolar oxygen pressures that can be obtained by its use. First, the difficulty of achieving actual administration of "pure" oxygen must be emphasized. Although alveolar P_{O_2} values of 670 mm Hg or more should be obtainable at sea level, this requires exceptional measures to prevent leakage of air into the inspiratory stream. For example, a familiar type of oronasal mask may yield less than 50 per cent oxygen. The pitfalls are not confined to unsuitable or ill-fitting masks. Unexpected leakage may also come from sources like a poorly sealed demand unit, an exhalation valve that fails to close completely with the low negative pressure of quiet inspiration, or a perforated eardrum.

When administration of essentially pure oxygen to the patient's trachea is achieved and time allowed for washout of nitrogen from

the lungs, the alveolar P_{O_2} should be virtually equal to the ambient pressure, minus only the partial pressures of water vapor and alveolar carbon dioxide. At 3 atm abs, for example, the ambient pressure is 760 x 3 = 2280 mm Hg. With pure oxygen, normal body temperature, and normal ventilation, the alveolar P_{O_2} could thus be 2280 - (47 + 40) = 2193 mm Hg. This represents nearly a 22-fold increase over the P_{O_2} of air breathing at normal pressure. Subnormal ventilation will subtract from such values only to the extent of the rise in alveolar P_{CO_2}. For example, half-normal ventilation with a P_{CO_2} of 80 mm Hg would yield a P_{O_2} of 2153. Table 1 gives examples of alveolar P_{O_2} as ideally achieved at various pressures.

TABLE I

Alveolar oxygen pressures obtainable with oxygen administration at various ambient pressures.

Ambient Pressure		Alveolar P_{O_2}*
Atm abs	mm Hg	mm Hg
1	760	673
2	1520	1433
3	2280	2193
4	3040	2953
5	3800	3713
6	4560	4473

*Assuming that 100% O_2 reaches the patient's trachea, that his body temperature is 37° C (water vapor pressure = 47 mm Hg), and that ventilation is normal (P_{CO_2} = 40 mm Hg). Oxygen at 99.5% purity will yield very slightly lower values: e.g., about 2180 mm Hg at 3 atm.

Since the partial pressures of water vapor and carbon dioxide will remain essentially constant, each additional atmosphere of ambient pressure with pure oxygen should produce nearly 760 mm Hg increase in alveolar P_{O_2}. On the other hand, administration of oxygen concentrations significantly below 100 per cent will cause increasingly large discrepancies between ideal and actual values.

If the ambient pressure is accurately known and the inspiratory oxygen concentration is known to be very close to 100 per cent, alveolar P_{O_2} can be estimated with considerable accuracy. Alveolar gas samples can also be obtained and analyzed with relative ease, and this is a worthwhile procedure when arterial blood gas determinations are impractical. However, alveolar P_{O_2} itself is seldom of much interest except in relation to the arterial P_{O_2} and O_2 content that result from it.

Arterial Oxygen Pressure

Alveolar P_{O_2} is the principal determinant of arterial P_{O_2}, and these values are normally closely related. However, several factors prevent perfect agreement and can sometimes cause the arterial value to be well below the mean alveolar level. The main factor accounting for such differences is venous admixture. Since this is a prominent cause of hypoxia and thus an important entity in hyperbaric oxygenation, it deserves thorough discussion. This can best be accomplished when the basic subject of blood oxygen content has been covered.

Arterial Oxygen Content

One gram of hemoglobin can combine with 1.36 ml of oxygen. Since the normal concentration of hemoglobin is about 15 g per 100 ml of blood, the usual oxygen capacity of blood is in the order of 20 ml of oxygen per 100 ml of blood, usually expressed as 20 volumes per cent (vol %). At a normal arterial P_{O_2} of about 100 mm Hg, hemoglobin is approximately 97 per cent saturated with oxygen. At this P_{O_2}, whole blood also contains about 0.3 volumes per cent of unbound oxygen in physical solution in the water of the plasma and red cells. Increasing the P_{O_2} of blood beyond 100 mm Hg will complete the saturation of Hb, adding about 0.6 volumes per cent by the time a P_{O_2} of roughly 200 mm Hg is reached. Except for this, the blood oxygen content can increase only by addition of greater amounts of oxygen in physical solution.

The generally accepted coefficient of solubility ("alpha") for oxygen in normal whole blood at 37° C (4) is

0.0237 ml of O_2/ml of blood/760 mm Hg P_{O_2}, or

2.37 ml of O_2/100 ml of blood/760 mm Hg P_{O_2}, or

2.37/760 = 0.0031 vol %/mm Hg P_{O_2}.

The latter value is most commonly used but is often rounded to

0.003 vol %/mm Hg P_{O_2}, or expressed as

0.3 vol %/100 mm Hg P_{O_2}.

The relationships between P_{O_2} and the quantities of oxygen combined with Hb and in physical solution are most readily visualized by means of graphs such as Figures 1 and 2. For example, at 2000 mm Hg (ideally achieved by breathing 100 per cent oxygen at somewhat less than 3 atm abs of ambient pressure), the quantity of dissolved oxygen should be equal to 0.0031 x 2000 = 6.2 vol %. This is only about 30 per cent of the quantity carried by the hemoglobin of normal blood at only 100 mm Hg arterial P_{O_2}, which underscores the relative inefficiency of physical solution as a means of transporting oxygen. However, an additional 4.5 g of Hb/100 ml of blood would be required under normal conditions to match the total blood O_2 content achieved at 2000 mm Hg. In the same terms, an arterial P_{O_2} of 2000 mm Hg could compensate for the reduction of total blood oxygen capacity associated with loss of 4.5 g of Hb/100 ml of blood. Such a loss of Hb would be equivalent to loss of nearly one third of the total blood volume. Considering the normal margins of safety, an arterial P_{O_2} of 2000 mm Hg with maintenance of normal circulating fluid volume should permit survival in the face of extreme degrees of blood loss. We may also consider the fact that 6 volumes per cent of oxygen is at least equal to the normal blood oxygen extraction, or arteriovenous (a-v) oxygen content difference, of the body as a whole (5). Most tissues should therefore remain oxygenated under such conditions even with no circulating red cells. In the myocardium, which normally extracts much more than 6 volumes per cent (6), survival with only dissolved oxygen at 2000 mm Hg would require approximate doubling of

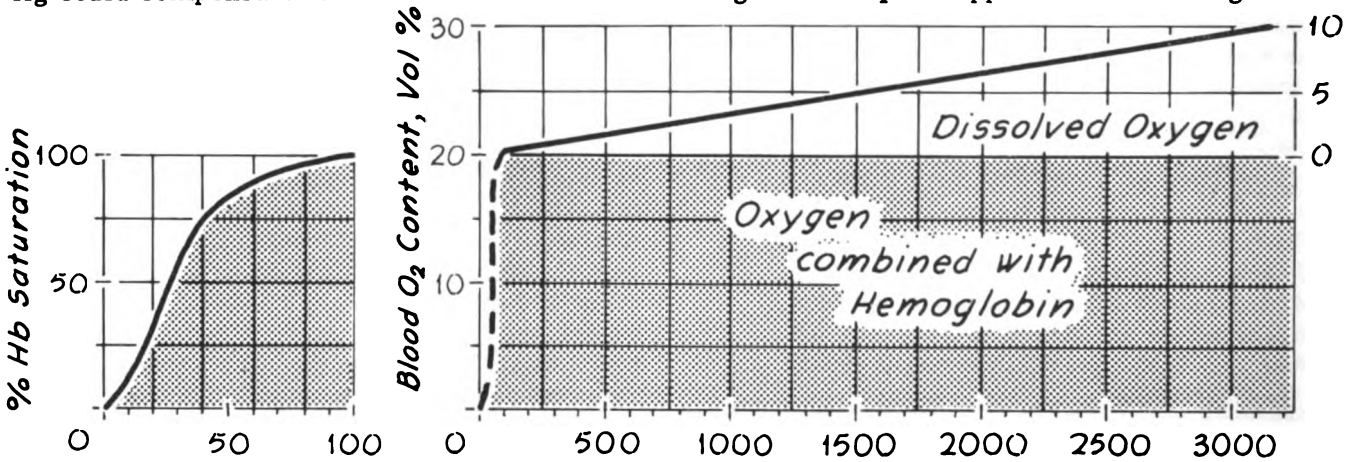


Figure 1.

Blood Oxygen Content at Various Blood Oxygen Pressures

The left-hand portion of the graph has a blood-P_{O_2} scale of 0-100 mm Hg and represents the oxyhemoglobin dissociation curve for normal human blood having an oxygen capacity of 20 vol % (ml O_2/100 ml blood). The simplifying assumption is made that full saturation of hemoglobin is achieved at 100 mm Hg (actually 97-98 per cent at this P_{O_2}).

The right-hand portion has a blood-P_{O_2} scale from 0 to over 3000 mm Hg and shows the increase in blood oxygen content in the form of oxygen physically dissolved in the water of plasma and red cells at various blood oxygen tensions. The dissolved fraction is assumed to be 0.31 vol % at 100 mm Hg and rises at the rate of 0.0031 vol %/mm Hg P_{O_2}. Oxygen will be dissolved in similar quantities whether the Hb concentration is normal or not. If the hemoglobin oxygen capacity is known, substitute the actual value for 20 vol % and add the dissolved fraction.

Assuming that 100 per cent oxygen is administered, that the body temperature is 37°C (H_2O vapor pressure 47 mm Hg), and that the alveolar P_{CO_2} is 40 mm Hg, the alveolar P_{CO_2} is determined by subtracting 87 from the ambient pressure in mm Hg (760 mm Hg/atm abs). In the absence of significant shunts or diffusion barriers, arterial P_{O_2} should have essentially the same value. However, this can seldom be assumed (see text).

Note that even at 3000 mm Hg (approximately equivalent to OHP at 4 atm abs) the quantity of dissolved oxygen is less than half that normally carried by hemoglobin at only 100 mm Hg. Note also, for example, that removal of 6 vol % of dissolved oxygen will drop the blood P_{O_2} approximately 2000 mm Hg. Removing the same quantity from combination with hemoglobin drops the P_{O_2} only about 65 mm Hg.

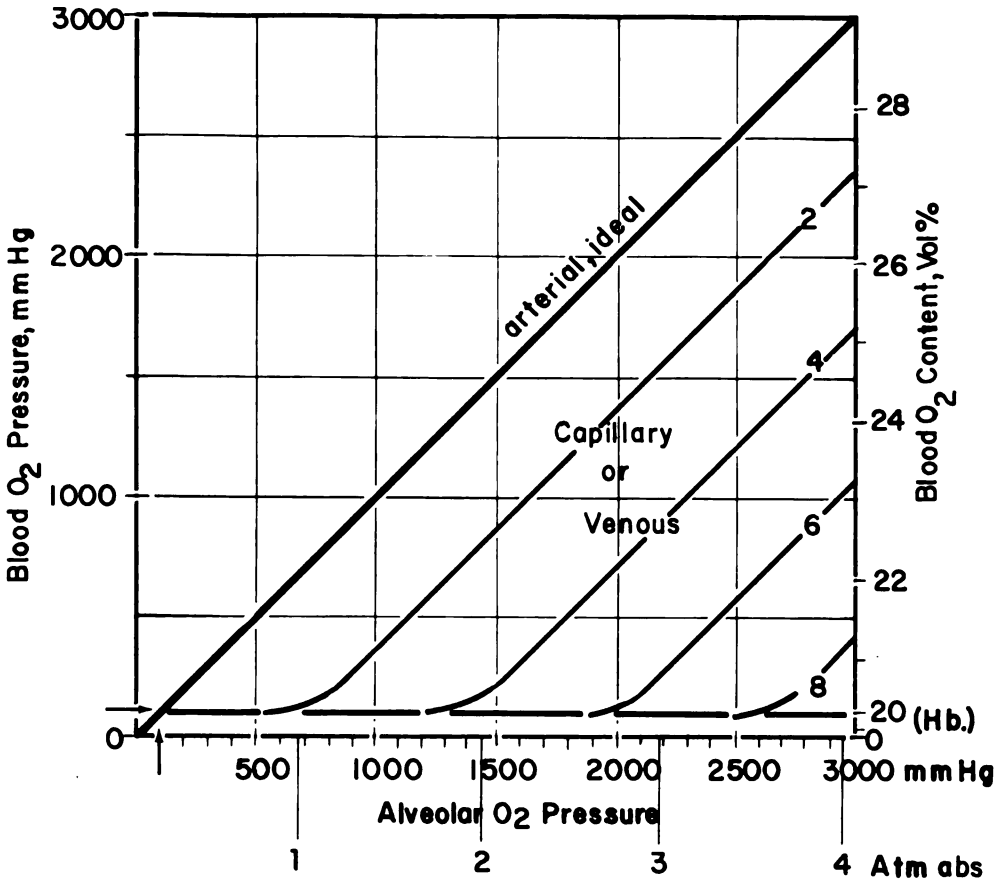


Figure 2.

Blood Oxygen Pressure and Contents Ideally Obtained at Various Alveolar Oxygen Pressures

The upper horizontal scale represents alveolar P_{O_2} in mm Hg, while the lower scale indicates chamber pressures (atm abs) at which such alveolar oxygen pressures would ordinarily be achieved with administration of 100 per cent oxygen. The vertical scales represent blood P_{O_2} (left) and blood oxygen content (right) assuming 20 vol % Hb O_2 at 100 mm Hg and total saturation of Hb at 200 mm Hg.

The diagonal solid line marked "arterial (ideal)" indicates equal values of arterial and alveolar P_{O_2}, not actually found even in normal individuals. This form of graph can be used for graphic comparison of actual values of arterial P_{O_2} with ideal values as in Figures 3 and 6, such comparisons having diagnostic significance (see text). For other applications, measured arterial values are simply plotted on the ideal line at the appropriate level, letting horizontal and vertical P_{O_2} scales both represent arterial P_{O_2}.

The lower diagonal lines marked "capillary or venous" indicate blood P_{O_2} and content with removal by the tissues of volumes per cent of oxygen as indicated by numbers on the lines. Consider, for example, a measured arterial P_{O_2} of 2000 mm Hg and place a point on the "ideal arterial" line at this point. Dropping directly down from this point, read off capillary blood P_{O_2} and content as 2, 4, 6, vol % of oxygen are removed. With normal blood flow in most tissues, the a-v oxygen content difference is about 6 vol %. If so, the "6" line would represent venous blood and indicates a P_{O_2} of about 130 mm Hg. If blood flow were reduced about 25 per cent, raising the a-v difference to 8 vol %, venous P_{O_2} would be below 100 mm Hg and can be determined by applying Figure 5. In this case, an arterial P_{O_2} of about 2500 mm Hg would be required to elevate venous P_{O_2} to 100 mm Hg.

Below 200 mm Hg, the altered direction of the "capillary or venous" lines indicates removal of oxygen from hemoglobin following the Hb dissociation curve. (The assumption of complete saturation of Hb at 200 mm Hg but with 20 vol % Hb oxygen at 100 mm Hg differs somewhat from the basis of Figure 1. The region below 100 mm Hg is enlarged in Figure 5.)

capillary flow. However, this may perhaps be accomplished readily with the low viscosity of a cell-free perfusate. At any rate, animals have been maintained for considerable periods with little or no active hemoglobin by means of oxygen at high pressure (7,8).

Ability to maintain blood oxygen capacity and transport in the face of massive loss of active hemoglobin suggests therapeutic applicability not only in hemorrhage but in conditions like carbon monoxide poisoning and methemoglobinemia. In the case of carbon monoxide poisoning, OHP has the additional advantage of hastening the removal of carbon monoxide from combination with hemoglobin (9).

Ability to elevate blood oxygen content above normal levels with no increase in the viscosity of circulating blood suggests that OHP may have unique value in conditions involving either local or generalized reduction of blood flow. This subject will be discussed in connection with capillary oxygen tensions, below.

Venous Admixture

Under normal conditions, the arterial P_{O_2} closely reflects the mean alveolar P_{O_2}. When it fails to do so, the cause is usually found in the entry of unusual amounts of poorly oxygenated blood into the arterial circulation (10). As has been discussed, gross underventilation of functioning alveoli can sometimes be implicated; but this source of hypoxia can be remedied by administration of oxygen at normal pressure. Such is not the case when actual venous admixture occurs through a "true" right-to-left shunt.

Normally, venous blood enters the arterial stream through the thebesian veins and probably to some extent through bronchial and pleural circulations (11). The exact quantities are not known but must be extremely small. An ingenious study by Lenfant (12) indicates that the "true shunt" in normal man is less than 1 per cent of the cardiac output. Pathological degrees of venous admixture may arise not only from congenital vascular malformations but also from continued perfusion of large numbers of collapsed or consolidated alveoli.

Venous admixture reduces mean arterial P_{O_2} and oxygen content by the transfer, in effect, of oxygen from well-oxygenated blood to the poorly oxygenated fraction of the stream. For example, consider a shunt involving 50 per cent of the cardiac output: If the normally oxygenated blood had an oxygen content of 20 volumes per cent and the venous blood contained 14 volumes per cent, the mixed arterial value would be 17 volumes per cent and the arterial P_{O_2} would be only about 50 mm Hg. (Under these conditions, a venous content of 14 volumes per cent would indicate an a-v oxygen content difference of only 3 volumes per cent, in turn suggesting a cardiac output of about twice the normal value.) If the venous oxygen content remained at 14, the oxygen content of oxygenated blood would have to be elevated to 26 volumes per cent in order to yield normal values of arterial P_{O_2} and oxygen content. This in turn would require an alveolar P_{O_2} of about 2000 mm Hg. In this case, the alveolar-arterial (A-a) P_{O_2} difference would have increased from 100 - 50 = 50 mm Hg with air at normal pressure to 2000 - 100 = 1900 mm Hg with OHP (Figure 3).

This example illustrates the unique ability of OHP to combat the hypoxia of venous admixture, but it also underscores the magnitude of oxygen pressures that may be required to produce normal—not to mention exceptionally high—arterial values in the presence of large shunts (Figure 4). Such hopes as "superoxygenating" the brains of cyanotic infants to permit circulatory arrest of significant duration during corrective surgery must be interpreted in this light. So must the problem of hyaline membrane disease and other conditions in which the effective shunt is of pulmonary origin and where prolonged exposure to effective oxygen pressures may compound the basic pathology (13).

In view of the large A-a P_{O_2} difference that may be encountered, it is always questionable to assume that an elevated alveolar P_{O_2} is fully reflected in arterial blood. In normal individuals breathing air at sea level, the usual A-a difference is 10 mm Hg or less. This is explained in part by normal degrees of both alveolar hypoventilation and true venous admixture.

High oxygen concentrations will eliminate the effect of alveolar hypoventilation upon the A-a difference but will magnify that of venous admixture. For example, consider a true shunt of 1 per cent of the cardiac output, assuming that the a-v oxygen content difference is 6 volumes per cent. The shunt will have the effect of lowering the arterial content by $0.01 \times 6 = 0.06$ vol % below the ideal value. At an abnormally low arterial P_{O_2}, the shape

of the oxyhemoglobin dissociation curve is such that the effect of this small change in oxygen content produces an almost undeterminable effect upon the P_{O_2}. Near a P_{O_2} of 100 it is associated with a drop of perhaps 5 mm Hg. The maximum effect is found at P_{O_2} levels where only dissolved oxygen is involved. Here 0.06 volumes per cent represents a P_{O_2} difference of 0.06/0.003 = 20 mm Hg. The A-a differences found by Lenfant (12) using 75 per cent O_2 at 2.6 atm in 8 subjects ranged from 21 to 126 mm Hg. The occurrence of values well above 20 led him to suggest an unusual amount of venous admixture through collapsed respiratory units in some of these men (Navy divers) at increased pressure.

Measured arterial P_{O_2} values reported by Brown and his associates (15) in healthy male subjects show much higher A-a P_{O_2} differences, and these increase progressively with increasing ambient pressure (Figure 6).

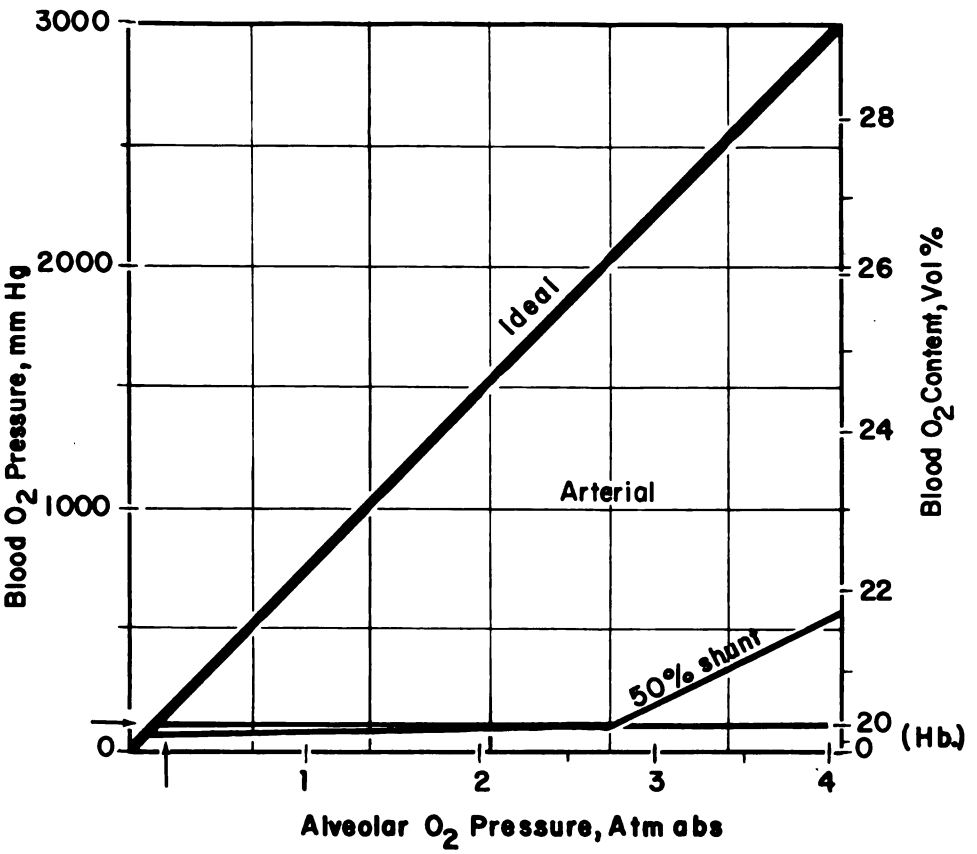


Figure 3.

Arterial versus Alveolar P_{O_2} in the Presence of a Large Hypothetical Right-to-Left Shunt (Venous Admixture)

This illustrates use of the graph form of Figure 2 to visualize the effect of OHP upon arterial P_{O_2} with massive venous admixture as in congenital vascular malformation. The calculations were based on these hypothetical values: (1) 50 per cent of the aortic flow consists of venous blood that has by-passed ventilated alveoli, (2) the remainder of aortic flow is blood that has been fully oxygenated at the P_{O_2} indicated on the horizontal scale, and (3) the mixed venous O_2 content is 14 vol % and remains at this level, implying that cardiac output is initially about two times normal and decreases as increasing values of arterial P_{O_2} are achieved.

Note that in the presence of a large shunt, several atmospheres of oxygen may be required merely to bring the arterial P_{O_2} to a normal level. Great elevation of arterial P_{O_2} may be extremely difficult to achieve. Rate of rise of arterial P_{O_2} above 100 mm Hg could be parallel to the "normal" arterial line if cardiac output remains constant in this range.

Finding of an arterial versus alveolar pattern of this sort is diagnostic of venous admixture, suggesting either congenital anomaly or continued perfusion of collapsed or consolidated respiratory units. The actual pattern will reflect size of shunt and level of mixed venous oxygen content.

Figure 4.

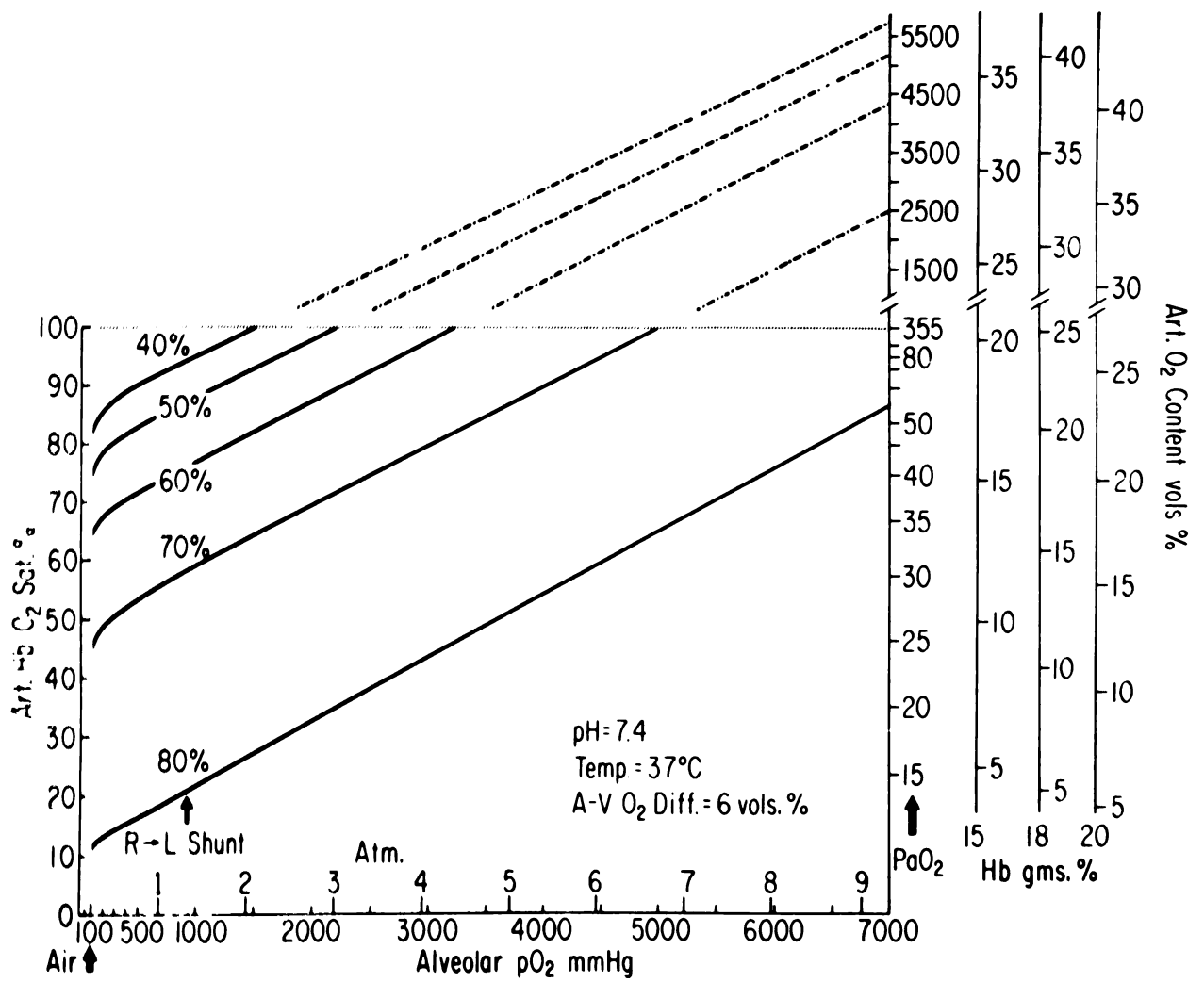
Arterial Blood Values with OHP in Various Degrees of Right-to-Left Shunt

This graph shows computed increases in arterial oxygen saturation, P_{O_2} and oxygen content (vertical scales) with increasing alveolar P_{O_2} (horizontal scale). Each diagonal line represents a different percentage shunt as labeled.

The calculations were based upon a blood pH of 7.4 and a body temperature of 37° C. A constant a-v oxygen content difference of 6 vol % was assumed, implying that the relationship between oxygen uptake and cardiac output remains constant. Since patients with such shunts are usually polycythemic, values of oxygen content are given for three different levels of hemoglobin concentration (right).

It is also assumed that the P_{O_2} of the oxygenated fraction of blood is equal to the alveolar P_{O_2}. If this is not the case (see text and Figure 6), values from this graph would be somewhat higher than those obtained clinically in otherwise identical circumstances. A similar graph has been published recently by Nelson and Reynolds (13).

Note, for example, that with a 70 per cent right-to-left shunt (not uncommon in severe cyanotic heart disease in infants), the arterial P_{O_2} with oxygen administration at 3 atm abs is only about 12 mm Hg greater than with air breathing at 1 atm. Although the arterial oxygen content is improved considerably at lower pressures, obtaining normal saturation in a 70 per cent shunt would require OHP at 6.5 atm —far beyond pressures now considered practical for hyperbaric surgery. The extremes of exposure required to obtain unusually high arterial oxygen values are indicated by pressures at which the values rise above 100 per cent arterial hemoglobin oxygen saturation (dashed lines).



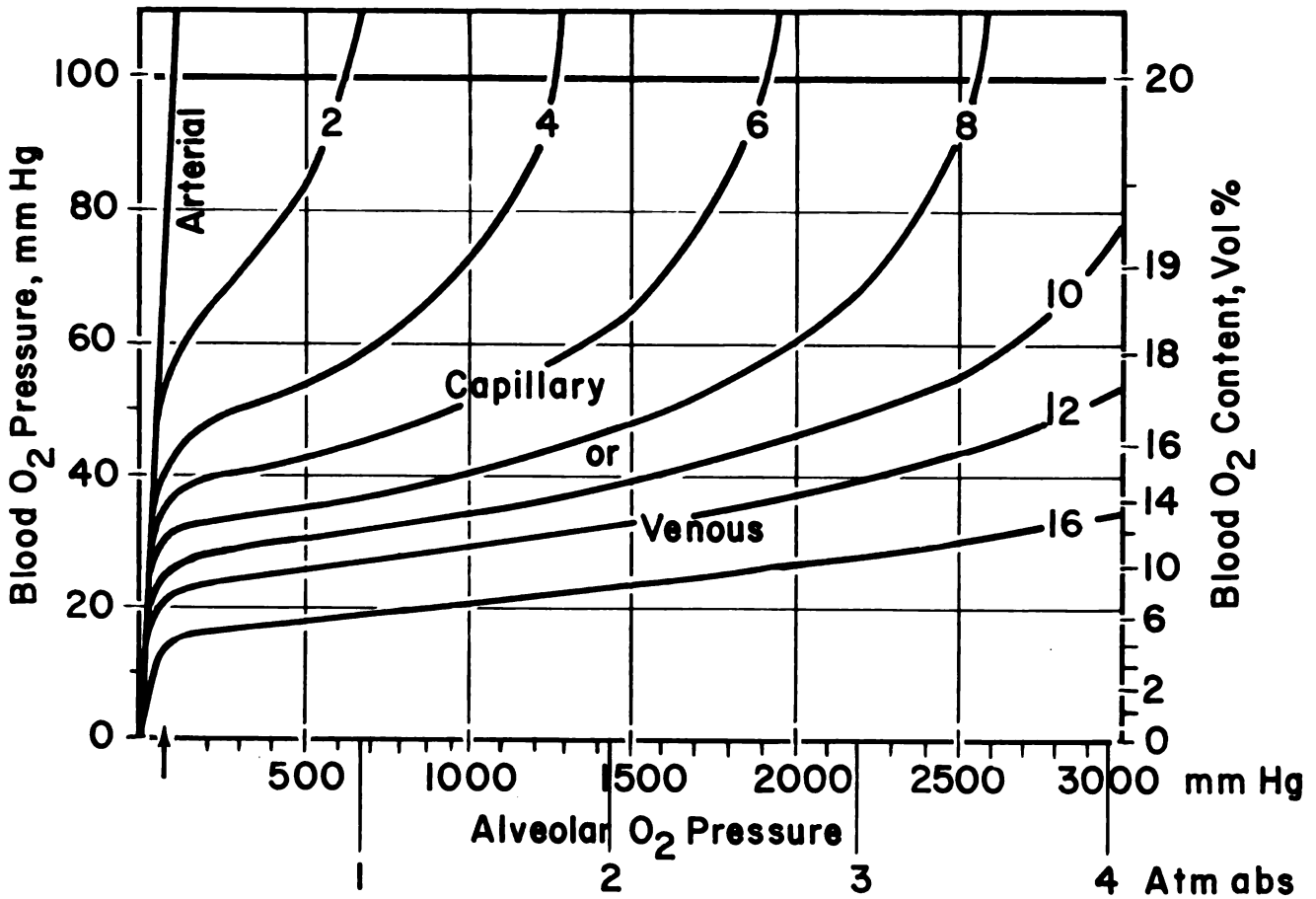


Figure 5.

Capillary and Venous Oxygen Pressure and Content versus Alveolar or Arterial P_{O_2} – (Lower Range)

This graph represents enlargement of the region below 100 mm Hg in Figure 2. The solid straight line at left is the continuation of the "ideal arterial" line. The P_{O_2} scale (left) is linear, hence the oxygen-content scale (right) must be alinear, representing the oxygen-hemoglobin dissociation curve. Hemoglobin oxygen content of 20 vol % at 100 mm Hg is assumed. The curves are based on the nomogram of Dill, Edwards, and Consolazio (14) for normal man at sea level. A respiratory quotient of about 0.8 and corresponding changes in blood pH and P_{CO_2} are assumed.

Where blood oxygen content is essentially normal, this graph can be used to estimate and visualize capillary and venous P_{O_2} and oxygen content values when these fall or remain below 100 mm Hg and 20 vol %. The curves are extensions of those in Figure 2 except for those representing quantities of oxygen extraction (beyond 8 vol %) too great to appear on that graph. The procedure for use of this graph is essentially as described for Figure 2, and Figures 2 and 5 will often be used together.

Consider, for example, a tissue that normally has an a-v oxygen content difference of 6 vol %. The normal venous P_{O_2} of this tissue with air breathing at 1 atm is indicated by the point of intersection (read on vertical P_{O_2} scale) of the "6" line with 100 mm Hg on the horizontal scale. If the blood flow of this tissue were cut in half, the a-v difference would increase to 12 vol % and the venous P_{O_2} would fall accordingly (find intersection of "12" line with 100 mm Hg). To determine the arterial P_{O_2} required to return venous P_{O_2} to its normal level, follow the "12" line up to that value (vertical scale) and read arterial P_{O_2} from horizontal scale. Assuming agreement of alveolar and arterial values of P_{O_2}, horizontal scale also indicates oxygen exposure required.

With actual measurement of arterial and venous values, use of this form of graph permits rapid estimation of a-v oxygen content differences and, hence, of relative changes in blood flow, plus evaluation of effects of OHP and pressures required to obtain desired capillary and venous values.

These men breathed oxygen from tight systems with mouthpiece and noseclip, but the mean measured arterial P_{O_2} at 3 atm abs, for example, was over 400 mm Hg below the computed alveolar P_{O_2}. This P_{O_2} difference represents an oxygen content difference of 400 x .0031 = 1.24 vol %, and the shunt required to account for this would be in the order of 1.24/6 = .21, or about 21 per cent of the cardiac output. Although breathing oxygen at high pressure probably favors the collapse of alveoli, such values are difficult to explain in the light of present knowledge. The matter obviously requires further study.

Although present means of determining blood gas values under increased pressure are by no means free of pitfalls, firm conclusions clearly cannot be reached from studies in which arterial P_{O_2} is completely unknown. The effect of unexpected failure to deliver the intended concentration of oxygen to the patient's trachea can be very large, and the influence of venous admixture is potentially enormous.

Capillary and Venous Blood Oxygen Values

When arterial blood enters a tissue capillary, it immediately begins to lose oxygen to the surrounding metabolizing cells. The quantity lost in transit depends upon the relationship between rates of capillary blood flow and tissue oxygen uptake. These values are difficult to measure, but the resulting average a-v oxygen content difference of blood supplying a given region or organ is often readily determined. Knowing the oxygen content values of both arterial and venous blood also permits drawing a profile of P_{O_2} and content at various points within a hypothetical average capillary in the tissue concerned. (This approach must of course be used with reservation in tissues where important a-v shunts are known to exist.)

When oxygen is being delivered from hemoglobin, the fall in P_{O_2} reflects the oxygen-hemoglobin dissociation curve and is relatively small. For example, extraction of 5 volumes per cent of O_2 from the blood will normally drop the P_{O_2} from about 100 mm Hg to about 40. On the other hand, delivery of oxygen from dissolved O_2 will drop the P_{O_2} at the rate of 1.00/.0031 = 323 mm Hg for each volume per cent of oxygen delivered, or 323 x 5 = 1615 mm Hg for 5 volumes per cent. This being the case under OHP, relatively small changes in the rate of oxygen uptake or blood flow, as reflected in the a-v difference, can make a very great difference in the P_{O_2} of the venous blood or at various points in the capillary network. The relationships are most readily visualized by graphical means as in Figures 2 to 8.

Many important potential applications of OHP depend upon its ability to compensate for decreases in blood flow. If we consider a tissue in which the a-v oxygen content difference is normally 5 volumes per cent, the P_{O_2} at the venous end of an average capillary should be about 40 mm Hg. Reduction of blood flow to half the original value (assuming no change in O_2 uptake) will increase the a-v difference to 10 volumes per cent as the same quantity of O_2 is now extracted from half as much blood. In consequence, the venous P_{O_2} will decrease to about 25 mm Hg (Figure 7). Restoring the venous P_{O_2} to 40 mm Hg will require an arterial O_2 content of 25 volumes per cent obtained at an arterial P_{O_2} of about 1600 mm Hg. Reduction of flow to 25 per cent of the normal value will increase the a-v difference to 20 volumes per cent and reduce the venous oxygen content and P_{O_2} to zero if the individual is breathing air at normal pressure. Restoration of the venous P_{O_2} to normal would now require adding 15 volumes per cent of dissolved O_2 to the arterial blood, requiring administration of oxygen at an ambient pressure in excess of 6 atm abs.

Complete restoration of normal venous P_{O_2} is probably not necessary to prevent serious hypoxic symptoms or damage when blood flow is reduced, but the actual requirements are not known. Studies based upon persistence of oxygen-dependent functions during complete cessation of circulation in the retina (16, 17) or brain (unpublished data) suggest that hypoxic effects may correlate with mean capillary or mid-capillary P_{O_2} rather than with venous values (Figure 11). Restoration of such capillary levels to normal is much more readily accomplished than normalization of venous levels, but the bearing of such observations on the problem of reduced blood flow is not yet certain. Good answers to such questions will greatly improve our ability to predict the effects and practicability of OHP and to assess its value in various conditions.

Tissue Oxygen Tensions

Oxygen tensions within the capillaries are of interest primarily because they can often be computed and must provide some index of oxygen tensions within the tissues. Direct

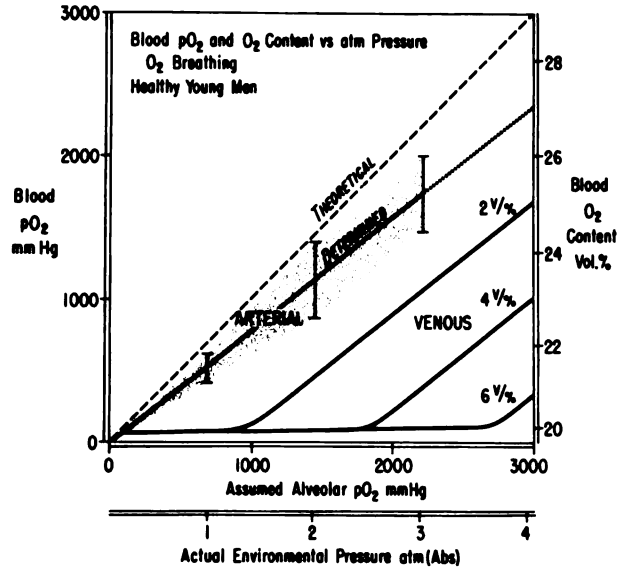


Figure 6.*

Measured Values of Arterial P_{O_2} versus Assumed values of Alveolar P_{O_2}

Here the form of Figure 2 is employed to show the contrast between ideal ("theoretical") arterial values and those obtained by oxygen electrode measurement in healthy young men by Brown et al. (15). The solid diagonal line ("determined") represents the mean of measured values obtained at the indicated assumed alveolar levels (horizontal scale). The brackets indicate plus or minus 2 standard deviations from the mean.

The capillary or venous lines ("venous") for 2, 4, and 6 vol % oxygen extraction have been drawn to conform to the slope of the determined arterial line.

Note that the mean of determined arterial values is approximately 400 mm Hg below the assumed alveolar value at 2000 mm Hg. Since the experiments were conducted with an optimal method of oxygen administration, the usual explanation of inspiratory air leaks is unlikely to be tenable. However, the "determined" line otherwise suggests venous admixture to the extent of about 20 per cent of the cardiac output (see text). Here, for example, elevation of the venous P_{O_2} to 100 mm Hg in a tissue with an a-v oxygen content difference of 6 vol % would require oxygen at nearly 4 atm abs instead of slightly over 2.5 as suggested by Figure 2. The actual cause of alveolar-arterial P_{O_2} differences of the magnitude shown here has not been determined.

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measurement of crucial tissue values, on the other hand, is frequently out of the question, and obtaining conclusively meaningful values by direct means is at present a difficult and questionable procedure (18). A measuring probe of significant size inserted into tissue must inevitably alter capillary blood flow by its mere presence. It must also yield a kind of "average" tissue value that may or may not be useful. In contrast, a sufficiently small probe must by definition indicate values from arterial down to well below the venous level within an extremely small range of movement—also yielding a questionable picture of the actual status of oxygenation. It is not even certain that changes in tissue-probe readings can be interpreted meaningfully on a relative basis. For such reasons, some investigators are more interested in establishing correlations between computed capillary-blood oxygen values and measurable oxygen-dependent functions.

Oxygen molecules reach and enter all body cells and fluids from the capillary blood solely by diffusion, and the difference in P_{O_2} between regions can be viewed as the driving force of diffusion. The higher the concentration of oxygen molecules in physical solution, the more frequently these molecules collide and the higher the resulting oxygen tension (P_{O_2}). If a region of lower oxygen tension exists nearby (lower tension meaning fewer molecules and fewer collisions), the net exchange of oxygen molecules between the two regions will be toward that with the lower tension. If no oxygen molecules were being taken up by chemical reactions in a tissue, the P_{O_2} would soon become homogeneous throughout. But with living cells taking up oxygen along the entire pathway of diffusion, the P_{O_2} falls progressively with increasing distance from the capillary. The number of oxygen molecules reaching the most distant cells may be barely sufficient for their needs under normal conditions, and anything that reduces the number available, the driving force of diffusion, or the effective diffusion distance may produce cellular hypoxia.

Administration of oxygen at high pressure not only increases the number of oxygen molecules in the blood, but the corresponding increase in P_{O_2} may greatly increase the ability of oxygen to diffuse from the capillaries to distant cells. At the same time, the P_{O_2} and oxygen content of tissue cells and fluids near the capillary must be increased. Both of these effects suggest corresponding clinical applications of great potential importance:

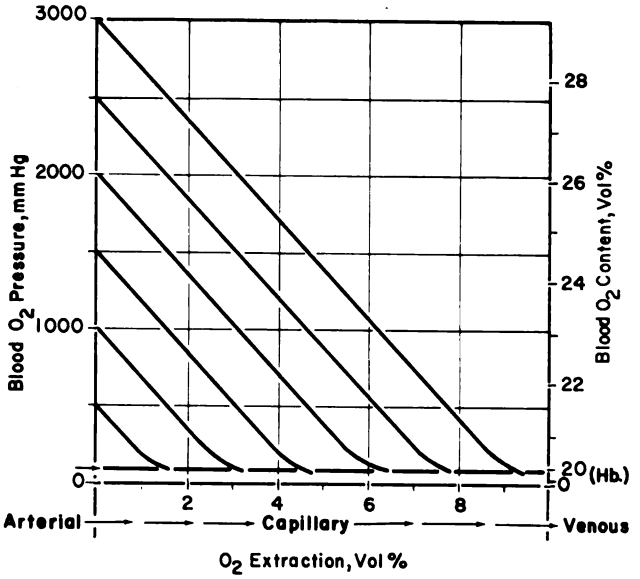

Figure 7.

Blood Oxygen Pressure and Content with Oxygen Extraction along a Capillary

The vertical scales are the same as those of Figure 2, but the horizontal axis now represents distance along a capillary in terms of volumes per cent of oxygen extracted by the tissue. The diagonal lines represent oxygen pressure and content starting with different arterial values as indicated by the intersection of each line with the left vertical scale.

The venous point in a given capillary will be determined by the total a-v oxygen content difference across that capillary. For example, an a-v difference of 5 vol % would place the venous end of the capillary at 5 on the horizontal scale, and the remainder of the graph to the right of that point would be ignored unless the a-v difference increased. Note, for example, that in such a tissue an arterial P_{O_2} of 1500 mm Hg would yield a venous P_{O_2} below 100 mm Hg. (The extension of these lines below 100 mm Hg is not shown here but is enlarged in Figure 8.)

If blood flow were halved in the same tissue (a-v difference now 10 vol %), elevation of the venous P_{O_2} to 100 would require more than 3000 mm Hg arterial P_{O_2}. (This conclusion would also apply, for example, to the myocardium, where the normal a-v difference may be 10 or more.) However, an arterial P_{O_2} of 3000 mm Hg would provide a mid-capillary P_{O_2} of nearly 1500 mm Hg as indicated by the position of the 3000 mm Hg line at the point of 5 vol % extraction.

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1. Increased oxygen content of the tissues themselves should provide a reservoir of oxygen that can be drawn upon to maintain life and function during interruption of circulation.

2. The increase in effective diffusion distance for oxygen should help maintain viability in tissues adjacent to functioning capillaries but deprived of their own blood supply (e.g., in acute stroke or myocardial infarction).

In both cases, the crucial question is quantitative: How long will life and function be extended? How much of an ischemic region can be kept viable?

Rough Predictions

In both cases, an indication of the order of magnitude can be derived from the type of information already presented if a few additional steps of reasoning are applied. For example:

1. The brain will continue to consume oxygen at the same rate as long as an adequate supply is available; then the usual pattern of hypoxic events will supervene with the usual time sequence.

2. In complete cessation of cerebral circulation, function and survival can therefore be prolonged only to the extent that additional oxygen is dissolved in brain tissue and in the blood remaining in cerebral capillaries—and for the time that this will support oxygen consumption at the usual rate for the temperature and other conditions extant.

3. Normal human whole-brain oxygen consumption at 37°C is in the neighborhood of 3.3 ml/min./100 g of brain tissue (19). The rate of consumption in gray matter, where hypoxic effects are likely to appear first, is probably higher; perhaps 5 ml/100 ml as assumed by Thews (20), or even greater (21).

4. Under OHP, the elevation of brain tissue P_{O_2} is unlikely to exceed the elevation of mean (or mid-) capillary P_{O_2}, nor is the quantity of dissolved oxygen per 100 g (or 100 ml) likely to differ greatly from that per 100 ml of blood at the same P_{O_2}. Putting one minute's worth of dissolved oxygen into solution should thus require approximately 5.0/0.0031 = 1600 mm Hg elevation in the mid-capillary P_{O_2}.

5. With a cerebral a-v difference of 6 volumes per cent, such elevation will require an arterial P_{O_2} of about 2500 mm Hg (Figure 7). This, in turn, will require administration of oxygen at a pressure above 3 atm abs. Even using the lower value for oxygen uptake (3.3 ml/min) would suggest that the prolongation of

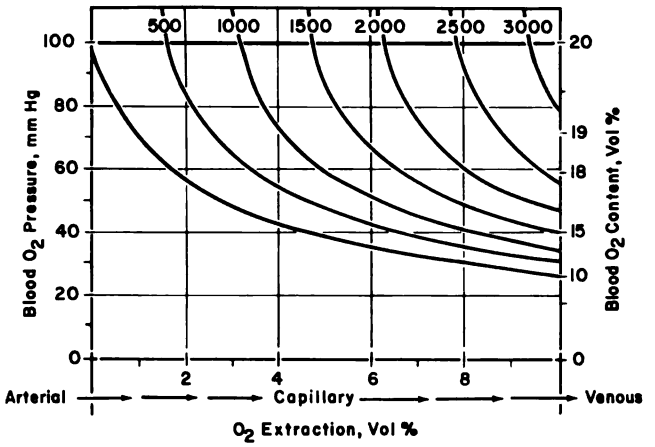

Figure 8.

Blood Oxygen Pressure and Content with Oxygen Extraction across a Capillary (Lower Range)

This graph presents the below-100 mm Hg region of Figure 7. The horizontal scale remains the same, but the vertical scales now represent 0-100 mm Hg and 0-20 vol % as in Figure 5. The upper curved lines are extensions of the diagonal lines of Figure 7, representing different values of arterial P_{O_2} as labeled. The lowest curve represents the normal situation breathing air at 1 atm (arterial P_{O_2} = 100 mm Hg). The basis of the curves is the same as for those of Figure 5 (14).

This graph helps to visualize for normal blood the consequences of diminished blood flow and the use of OHP to restore normal values. For example, with air at 1 atm, capillary blood of a tissue with a normal a-v difference of 5 vol % will follow the lower curve and show a venous P_{O_2} slightly below 40 mm Hg (oxygen content 15 vol %) and a mid-capillary P_{O_2} (at 2.5 vol % extraction) of 50 mm Hg. Halving the blood flow will move the venous end of the capillary from 5 vol % to 10 vol % on the extraction scale. Here the venous oxygen content is 10 vol % and the venous P_{O_2} about 25 mm Hg. Restoring the previous venous value will require an arterial P_{O_2} of about 1500 mm Hg as represented by the fourth curve from the bottom. This will elevate the mid-capillary P_{O_2} to about 90 mm Hg, or well above its previous value. Restoration of the previous mid-capillary P_{O_2} would require an arterial P_{O_2} of only about 700 mm Hg, almost achieved with oxygen at normal pressure. At the present time, it is not known whether relief of tissue hypoxia requires restoration of venous P_{O_2} to normal (or to some specific value) or whether some other index, as perhaps mean- or mid-capillary P_{O_2}, provides a more satisfactory reflection of effective tissue oxygen status (see Figure 11).

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cerebral function and survival could not be greater than, for example, two minutes at 4 atm OHP.

These are, of course, not optimistic predictions. While they are crude estimates, it is difficult to see how the actual values could differ greatly from them. One or two minutes extra time in complete circulatory arrest is considerably less extension of survival time than can be achieved with moderate hypothermia at normal pressure.

With hypothermia plus OHP, the prospect is improved. Hypothermia should increase the period of extension at a given arterial P_{O_2} in at least three ways. The decrease in rate of cerebral oxygen uptake will (1) extend the extra period provided by a given quantity of dissolved oxygen, and (2) decrease the cerebral a-v difference (unless blood flow decreases to exactly the same extent) and thus increase the capillary P_{O_2} values obtained with a given arterial P_{O_2}. In addition, (3) lower temperature will somewhat increase the solubility of oxygen, thus increasing the quantity of dissolved oxygen at a given tissue P_{O_2}.

Estimation of the distance of effective oxygen diffusion from a functioning capillary into a totally ischemic region requires less extensive reasoning: Most capillaries are believed to have a length of 1 millimeter or less. Yet across this small distance, even in capillary blood flowing at a normal rate, the drop in P_{O_2} of dissolved oxygen is extremely large (e.g., almost 2000 mm Hg with an a-v difference of 6 volumes per cent). Radial diffusion from a capillary should be more nearly equivalent to a state of no flow; but even using the value mentioned, we can predict that the P_{O_2} would reach zero in less than 1 millimeter of distance into ischemic tissue from the arterial end of a functioning capillary at 3 atm OHP, or in less than 0.5 mm from the functional mid-capillary point of the same vessel. The effective diffusion distance from the venous end of the capillary (assuming an a-v difference of 6 volumes per cent with venous P_{O_2} at about

100 mm Hg) should be only slightly greater than under normal conditions. Only by marked reduction of the rate of oxygen uptake by cells along the diffusion path as in hypothermia could the order of magnitude be increased.

Mathematical Models

The desirability of more sophisticated analysis of the problems led Brown and his associates (15) and Starmer, et al., (22) to apply mathematical models. Using Krogh's theoretical cylinder model of capillary oxygen diffusion and the more recent data of Thews (20) for cerebral gray matter, they calculated the oxygen-tension curve (extending between the venous ends of two adjacent capillaries) that might be expected in the steady state with OHP at 3 atm abs (Figure 9). The corresponding curves calculated by Thews for air breathing at 1 atm are shown below in the same figure. Only the cross-section of a cylinder surrounding the venous end of a capillary was considered since it would represent the area of lowest and presumably most critical oxygen tension. The relative values of P_{O_2} at the lowest points of the OHP (upper) and "normal" curves indicate that OHP at 3 atm should increase the P_{O_2} in these lowest zones of brain gray matter almost fourfold. Cells at the same distance from the arterial ends of the capillaries (not shown) should have an increase in P_{O_2} of over 17-fold at 3 atm OHP if the "determined" values of Figure 6 are used, or nearly 22-fold if the alveolar P_{O_2} were fully reflected in the arterial value.

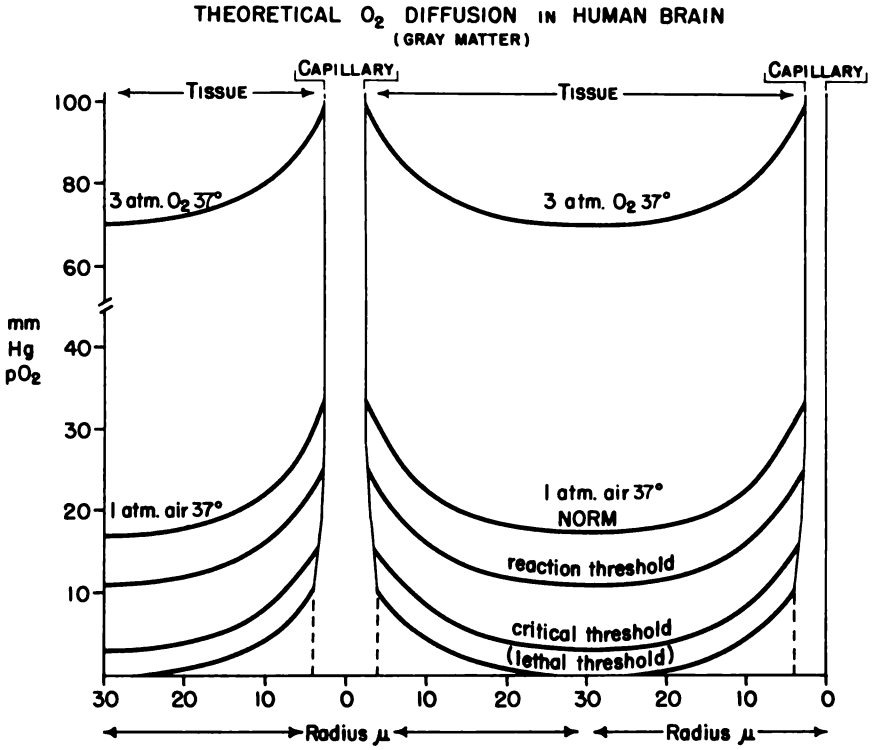

Figure 9.*

Theoretical Tissue Oxygen Pressures in Human Gray Matter at 37°C

The curves represent oxygen tensions (vertical scale) as computed for various distances (horizontal scale) from the venous ends of two adjacent capillaries. Capillary radius was assumed to be 2.5 microns; intercapillary distance 60 microns (average for human gray matter).

Uppermost curve was computed by Brown, et al. (15) and Starmer, et al. (22) for oxygen breathing at 3 atm abs based on an arterial P_{O_2} of 1750 mm Hg (see Figure 6) and an a-v oxygen content difference of about 5.5 vol %. The bottom four curves were calculated by Thews (20) and are reproduced for comparison. The curve labeled "norm" represents normal conditions breathing air at 1 atm. The critical threshold of 4 mm Hg is estimated for the level of oxygen tension at which consciousness is lost.

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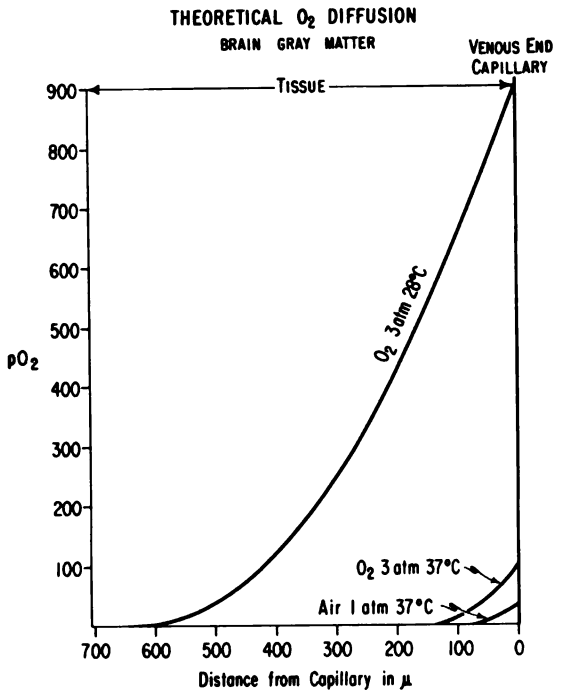

Figure 10.*

Computed Brain-Tissue Oxygen Pressures at 37° and 28°C

The curves represent oxygen tensions in human cerebral gray matter as computed by Brown, et al. (15) and Starmer, et al. (22), using the mathematical model of A.V. Hill for diffusion from a single capillary into a cylindrical solid.

Bottom Curve: Air breathing at 1 atm and 37°C body temperature. Here the critical P_{O_2} (4 mm Hg) is reached at 89 microns (0.098 mm) distance from the capillary wall if the distance between functioning capillaries is 180 microns or greater.

Middle Curve: Oxygen breathing at 3 atm and 37°C (arterial P_{O_2} 1750 mm Hg). Here the critical oxygen tension is reached at 150 microns (0.15 mm) from the capillary wall if the intercapillary distance is 300 microns or greater.

Top Curve: Oxygen breathing at 3 atm, 28°C. Tissue oxygen consumption is assumed to be half the normal rate but blood flow is unchanged (a-v oxygen content difference now about 2.75 vol %). Venous P_{O_2} rises to 900 mm Hg and rate of drop through tissues is decreased. Critical P_{O_2} is now reached at about 550 microns (0.55 mm) from the capillary wall.

The model corresponds roughly to a tissue in which a large proportion of the capillaries have been occluded.

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Thews has derived a critical threshold P_{O_2} value of about 4 mm Hg for the most vulnerable point in gray matter as indicated in Figure 9. Assuming this value to be correct, the time in which P_{O_2} would fall to 4 mm Hg during circulatory interruption was then computed. The resulting values were 1.7 seconds with air breathing at 1 atm and 4.8 seconds with 3 atm OHP.

NOTE: A problem affecting the validity of mathematical models as applied to oxygen under high pressure is the magnitude of longitudinal diffusion of oxygen through the theoretical cylinder of tissue. Under normal conditions, where the total difference in P_{O_2} between the ends of the capillary itself must be well below 100 mm Hg, this factor can probably be neglected. When the corresponding difference approaches 2000 mm Hg, it would cause the P_{O_2} about the venous end of the capillary to be higher than calculated. Another factor of potential importance is the orientation of the capillaries themselves. It is commonly assumed in mathematical models that adjacent capillaries are parallel to each other and that blood flow proceeds in the same direction in each. The intercapillary pattern of P_{O_2} distribution would be quite different if the venous end of one capillary were near the arterial end of another, as must often be the actual case. The apparent correlation of hypoxic end points with mean- or mid-capillary P_{O_2} may have its explanation in some such arrangement (Figure 11).

The question of effective oxygen diffusion distance was considered by means of A.V. Hill's model for diffusion of oxygen into a cylindrical solid. This model is roughly analogous to tissue cylinders surrounding functioning but widely separated capillaries in a cortical area rendered ischemic by obstruction (for instance by fat emboli) of a large portion of the normal capillary network. Tissue P_{O_2} values were then computed assuming no change in tissue oxygen consumption or in the rate of blood flow in the capillaries remaining open. The resulting curves (Figure 10) indicate relatively considerable extension of effective oxygen tension at 3 atm as compared to air at

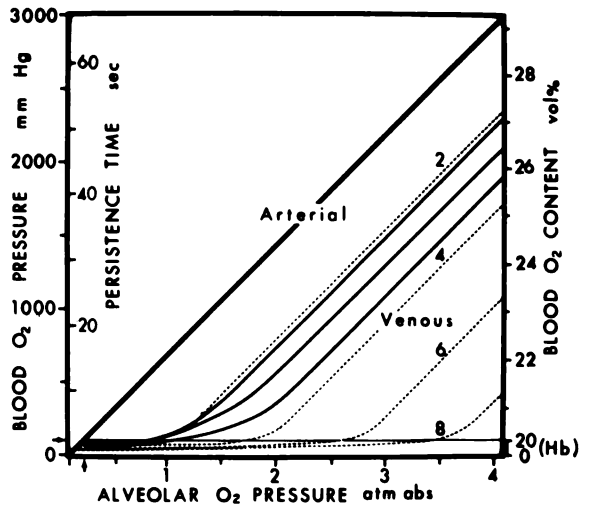

Figure 11.*

Persistence Time of Normal Vision in Retinal Ischemia with Computed Values of Retinal Capillary P_{O_2} at Various Alveolar Oxygen Pressures

Pressure on the eyeball sufficient to produce retinal ischemia causes dimming of vision in four to five seconds under normal conditions. Breathing oxygen at increased pressure allows normal vision to persist for periods up to 50 seconds at 4 atm abs. The three solid curved lines represent persistence times in three subjects, superimposed on a graph like Figure 2. Note that the curves for persistence time are remarkably similar to those for capillary or venous blood with 2-4 vol % oxygen extraction, suggesting that this oxygen-dependent function correlates with calculated blood P_{O_2} near the mid-capillary point. Figure taken from Carlisle, et al. (16). Similar findings have also been reported by Anderson and Saltzman (17).

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1 atm. Yet at 37°C this represents a tissue extension of only 73 microns (0.073 mm) for Thew's critical P_{O_2} of 4 mm Hg.

Under conditions of hypothermia, considering the favorable factors already discussed, the steady-state extension of the 4 mm Hg critical tissue P_{O_2} could be much farther: 544 microns (0.544 mm), as represented by the upper curve of Figure 10. This, however, assumes that blood flow remains constant while oxygen consumption decreases, thus elevating venous P_{O_2} very markedly.

Another unfavorable factor not taken into account above is that high arterial P_{O_2} can be accompanied by significant degrees of vasoconstriction in the brain and elsewhere as will be discussed below. This, of course, will increase the a-v oxygen content difference with potentially great lowering of capillary, venous, and tissue P_{O_2}. For example (Figure 7), a reduction of flow that increased the a-v difference from 6 volumes per cent to 8 could lower the venous P_{O_2} as much as 323 x 2 = 646 mm Hg—an effect not far from that of a 1 atm drop in exposure pressure. Cerebral blood flow can, however, presumably be maintained or increased by elevating the arterial P_{CO_2}.

Other Tissue Factors

Hyperbaric oxygenation cannot, as emphasized at the outset, improve the transport or elimination of carbon dioxide and may further impair it. However, the relative ease with which carbon dioxide diffuses, the ability of body fluids to buffer carbon dioxide, and the nature of its effects suggests that its retention in the tissues will seldom present problems comparable to those of hypoxia. OHP cannot be expected, except indirectly through improvement of general circulatory status, to be of any assistance in increasing capacity for, or transport of, any other ions or molecules required or produced by the cells. Maintaining adequate oxygenation should, however, yield incidental benefit in preventing anaerobic glycolysis and resulting local or systemic acidosis.

Whether factors such as lack of glucose or accumulation of metabolites can determine function or survival of tissues within the limits allowed by oxygen under hyperbaric conditions is not yet known. Thus far, it seems probable that the toxic effects of oxygen itself will be the greatest obstacle in the way of full realization of the potential benefits of hyperbaric oxygenation.

It is of interest to speculate upon tissue factors that may explain apparent persistence of benefit following periods of therapy. Clearly, tissue P_{O_2} must return rapidly to the previous hypoxic level. However, it is conceivable, as a possible example, that arrest of lactic acid production during the period of oxygenation permits tissue pH to return toward normal and that severe hypoxic symptoms do not return until this has again reached some critical level. It is also possible that a relatively brief period of therapy can sometimes interrupt a vicious circle that otherwise tends to perpetuate or

intensify the basic abnormality. Too little is known about factors beside hypoxia itself that must be involved in hypoxic symptomatology and in the ultimate death of cells.

Another question of interest in tissue hypoxia concerns "histotoxic" hypoxia. That OHP may be of some benefit here is suggested by a few observations in cyanide poisoning, and the possibility clearly deserves further investigation.

Conclusions

The foregoing discussion of the physiological basis for adminstration of oxygen under increased ambient pressure reveals important areas of uncertainty but leads to a number of tentative conclusions. These in turn can be related in a general way to proposed therapeutic applications.

1. Hypoxia of pulmonary origin should seldom require treatment with OHP. However, OHP provides a uniquely effective means of dealing with hypoxia due to continued blood flow through unventilated alveoli. (Here, however, the tendency of oxygen to produce atelectasis and frank lung damage may result in worsening of the basic pathology.)

2. The ability of OHP to increase, in effect, the oxygen capacity of blood suggests applicability to any condition in which hemoglobin is lost or inactivated.

3. Ability to increase the oxygen content of oxygenated blood provides the only known method of offsetting massive venous admixture to obtain normal or near-normal arterial values. However, presence of a large right-to-left shunt largely eliminates the practicability of elevating arterial oxygen to high supranormal levels.

4. Maintainance of significantly elevated arterial oxygen content provides an effective means of compensating for decreased blood flow in otherwise normal vascular beds. This suggests firm indications for OHP in shock and peripheral vascular trauma or disease. However, present lack of knowledge concerning critical levels of tissue P_{O_2} and their relation to capillary blood P_{O_2} prevents prediction of the decrements of blood flow that can be compensated at practical levels of OHP.

5. Hyperbaric oxygenation has relatively little to offer in extending the duration of tissue function or survival in temporary complete cessation of blood flow. The gains can be increased by combining OHP and hypothermia but may still remain well short of earlier optimistic predictions.

6. OHP extends the distance of effective oxygen diffusion but to such a limited extent that it is incapable of dealing with large regions of total ischemia. Its direct value in myocardial infarction, for example, may thus be negligible unless significant collateral circulation remains, or unless effects on the margin of the affected region are important—as perhaps in reducing the incidence of ventricular fibrillation. In stroke, OHP is likely to be of value only in forms involving critical reduction of blood flow without obstruction of end arteries.

7. With obstruction of a relatively small proportion of the capillaries in a given vascular network, or with limited increases in the diffusion pathway of oxygen as in tissue edema, OHP may be of real value. When OHP is combined with hypothermia, the degree of such abnormality amenable to treatment should increase considerably if reduction of blood flow with hypothermia is prevented.

8. Ability of OHP to elevate tissue P_{O_2} for such purposes as radiation therapy and the treatment of anaerobic infections may be highly variable, depending in each instance upon the extent of remaining local blood flow and the oxygen uptake of cells in the diffusion pathway.

9. The levels of OHP required for significant benefit are such that the toxicity of oxygen is likely to limit severely the safe duration of treatment. The clearest suggestions of possible benefit are thus in conditions where the need for OHP is of short duration, as where the basic defect can be remedied during a given exposure. In abnormalities of longer duration, its value will depend largely upon possible persistence of beneficial effects between relatively brief periods of repeated treatment.

OTHER APPLICATIONS OF HIGH PRESSURE

Compression and Absorption of Gas

One of the oldest applications of high pressure, and surely the most enduring one, is its use to reduce the volume of gas bubbles in decompression sickness and arterial air embolism. Since these conditions seldom arose except when an individual had previously been exposed to high pressure in the course of diving or tunnel work, this application has not been of widespread clinical interest despite its firm roots in Boyle's Law and its long and honorable history.

On the same grounds, increased pressure should be useful in treating air embolism that

occurs occasionally during open heart surgery (23) and through a few other iatrogenic mechanisms. To be of maximum value here, as in air embolism from diving, compression would have to be applied very promptly and to relatively high pressure. Boyle's Law operates to the patient's disadvantage when cardiac surgery is conducted under high pressure. A small bubble remaining in the left heart or arterial circulation might have little or no deleterious effect under normal conditions, but if the volume of such a bubble is increased by a factor of 3 or 4 by subsequent return from high to normal pressure, significant circulatory obstruction may occur. In such a case, immediate return to high pressure would be the only effective treatment. Problems can also arise in any situation in which gas is trapped in the bowel, thorax, or elsewhere during procedures conducted under pressure.

The hope of putting Boyle's Law to good use has also been one basis for other proposed therapeutic applications, as in Cross and Wangensteen's study of increased atmospheric pressure as a possible aid for decreasing distension and preserving gut viability in intestinal obstruction (24, 27). Yanda's use of high and low atmospheric pressures in the treatment of emphysema (26) may be another example, but the proposed mechanism is less readily understood.

Therapeutic utilization of Boyle's Law has evident shortcomings. One is that the decrease in absolute volume per unit of increase in pressure becomes progressively smaller. At the same time, any therapeutic benefit to be gained is also affected by the shape the enclosed gas assumes in the body. Ordinarily the shape assumed by distending intestinal gas is, like the gut itself, roughly cylindrical. Embolic gas in the vascular bed must be in the shape of spherical or cylindrical bubbles. The diameter of these masses of gas must often be more important than their volume. As illustrated in Figure 12, the decrease in diameter with increasing pressure may be disappointingly small.

Another aspect of the problem is that the patient must eventually be decompressed and that remaining gas must inevitably re-expand (45). For example, compression might provide dramatic relief of pneumothorax, but this relief would last only as long as the pressure was maintained. If the mass of gas in the thorax increased during the period under increased pressure, intervention would be required before the patient could safely be decompressed. A recurrent nightmare of diving medical officers concerns a patient with tension pneumothorax unwisely treated in a one-man chamber with no possibility of access.

Recompression is an effective treatment for decompression sickness and air embolism largely because high pressure not only reduces the size of the bubbles but also hastens their absorption and disappearance. Time required for this phase of the process in part explains

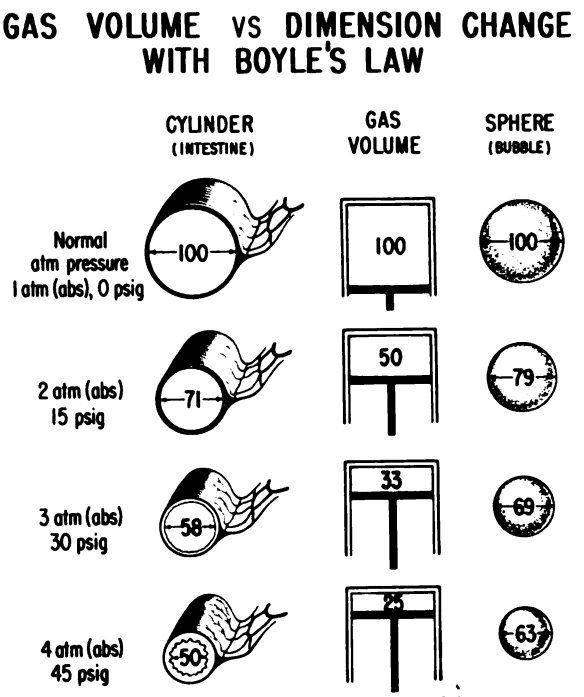

Figure 12.*

Changes in Diameter of Gas Pockets of Different Shapes with Compression

The reduction of volume per unit increase of pressure decreases progressively as shown here (center column). The corresponding reduction of diameter in cylindrical and spherical shapes is proportionally less from the outset as illustrated and becomes almost negligible at higher pressures.

Only in the length of a cylindrical mass of constant diameter (as perhaps a cylindrical bubble in an artery) will the reduction in an important dimension be equal to the decrease in volume with compression. From Brown, et al. (15).

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the long duration of standard recompression procedures. When large masses of gas are involved, as in intestinal distension, the time required for significant absorption of gas may be hopelessly long.

Analysis of factors influencing the rate of absorption of gas pockets (H.D. Van Liew, unpublished) indicates that the gradient for absorption of nitrogen is the principal determinant. The maximum nitrogen gradient is achieved by administration of oxygen at a pressure sufficient to keep the venous hemoglobin saturated with oxygen and thus maintain the maximum a-v P_{O_2} difference. Administration of oxygen at normal pressure will increase the rate of nitrogen absorption to a greater extent than will several atmospheres of compression with the patient breathing air. Even in the treatment of decompression sickness, new approaches place more emphasis upon oxygen breathing at moderate pressures than upon use of very high pressure (Chapter VII).

Use of high pressure for compression and accelerated absorption of collections of gas within the body will probably continue to be applied mainly, if not almost solely, in the treatment of decompression sickness and air embolism.

Potentiation of Gaseous Anesthetics

A venerable proposed application of high pressure concerns its use to increase the level of anesthesia obtainable with relatively weak but otherwise desirable gaseous agents. In France, around 1878, Fontaine became so enthusiastic about his experience with nitrous oxide in a mobile hyperbaric operating chamber that he contemplated construction of a pressurized surgical amphitheatre large enough to hold 300 observers.

In general, the fact that even nitrogen shows "narcotic" effects at increased pressure has been viewed as a problem rather than a potential benefit in high pressure work. On the other hand, it is true that certain biochemically inert anesthetics like nitrous oxide and particularly xenon (aside from its present great cost) lack little but high potency of being ideal agents. At 1 atm in concentrations of 70-80 per cent with oxygen, xenon produces rapid but light anesthesia in man with a minimum of disturbances in biochemical on physiological processes. Full recovery from the anesthetic effects usually comes within three to five minutes. It has already been established that with monkeys xenon under increased pressure can produce anesthesia to a depth of apnea and areflexia but with less suppression of electroencephalographic activity than with an equal depth of anesthesia with other agents (25).

With reasonably satisfactory anesthetic procedures available at normal pressure, it seems unlikely that high pressure would ever be used for the sake of anesthesia alone. At the same time, use of high pressure primarily for the sake of high P_{O_2} renders the use of weak anesthetic gases illogical. Any level of increased pressure that now seems clinically practical would not offer simultaneous benefits of inert-gas anesthesia and hyperbaric oxygenation. A further problem is suggested by the fact that inert gases having greater anesthetic potency than nitrogen appear to do so by virtue of greater solubility especially in lipids. Such solubility could be expected to predispose toward unusual decompression problems, as appears to be the case with nitrous oxide.

INCIDENTAL EFFECTS

The physiological effects of an entity like high pressure can seldom be confined to those desired for therapeutic purposes. From the patient's standpoint, the toxicity of oxygen itself (Chapter III) is by far the most significant of the "side effects" of hyperbaric therapy. From the physician's standpoint, decompression problems (Chapters V to VII) are probably the most troublesome.

Work of Breathing

Any increase in pressure is accompanied, in accordance with Boyle's Law, by an increase in the density of the gas involved. Even in normal individuals, increasing pressure produces an impressive increase in the work of moving air through the airways and a corresponding decrease in maximum breathing capacity (MBC) and related measures (28,29,30). MBC is decreased to about half the normal value at 4 atm, for example. With normal airways, however, the effects upon resting ventilation are negligible even at very high pressures. Such is not the case with preexisting respiratory pathology or intubation (Figure 13). Here, an attempt to increase the ambient pressure is capable of producing dyspnea, hypoventilation, and respiratory acidosis. Unless these can be overcome by mechanical assistance, use of larger tubes, or similar measures, attempts at hyperbaric therapy may have to be abandoned. Use of helium-oxygen mixtures is effective in combatting the increase in respiratory work; but if elevation of P_{O_2} is the aim of treatment, the addition of significant

concentrations of helium to the respired gas will largely defeat the purpose.

Retention of Carbon Dioxide

In addition to the possibility of respiratory acidosis from impaired ventilation, much has been made of the effect of hyperbaric oxygenation on carbon dioxide transport by blood. It is true that CO_2 transport is normally assisted by combination of carbon dioxide with reduced hemoglobin and by the greater buffering ability of Hb in its reduced state. However, the Haldane shift in the carbon dioxide dissociation curve of whole blood with normal degrees of hemoglobin desaturation is such that only a small rise in blood P_{CO_2} can be expected when hemoglobin remains fully saturated. Both theoretically and experimentally, the usual rise in cerebral venous P_{CO_2} is, for example, only about 6 mm Hg (32,33). Once venous Hb is fully saturated with oxygen, no further increase in P_{CO_2} can be expected with higher oxygen pressures as long as blood flow remains constant. Although P_{CO_2} elevation of this magnitude is not negligible in terms of resulting tissue P_{CO_2} and does have measurable effects, thus far no serious consequences appear to result from it (Chapter II).

The tendency toward carbon dioxide retention and acidosis under OHP is magnified by the presence of large right-to-left shunts (34). Here, the elevation of peripheral P_{CO_2} levels may require unusually large ventilation of the lungs in order to lower sufficiently the P_{CO_2} of the fraction of blood that goes through functioning alveoli. Use of intravenous buffering agents has also appeared desirable.

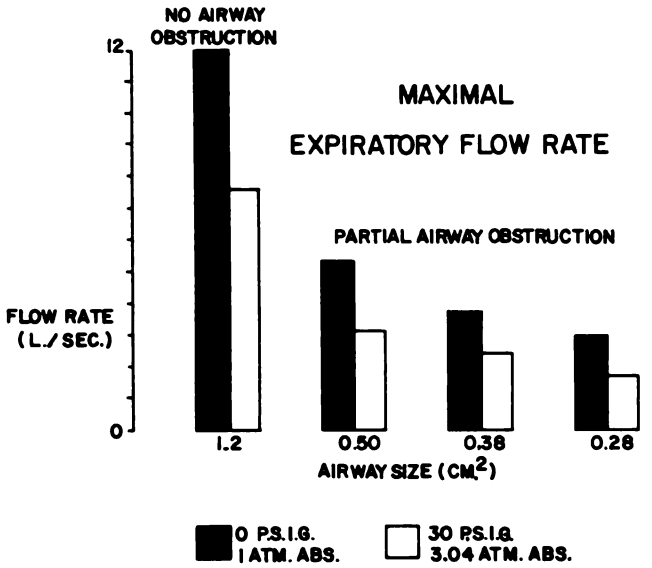

Figure 13.
Maximum Expiratory Flow Rates at 1 and 3 Atm Abs with Partial Airway Obstruction

The greater density of air at increased ambient pressure produces a measurable increase in the work of breathing and a corresponding reduction of breathing capacity and maximum flow rates. This is of no practical consequence in normal individuals at rest or during mild exertion at pressures presently used for hyperbaric therapy, but it can present serious problems in patients with dyspnea or decreased respiratory reserve.

Tracheotomy or endotracheal tubes and their connectors are an important source of excessive airway resistance in patients under hyperbaric conditions, and the largest possible sizes should be employed.

The values of maximal expiratory flow rate shown graphically here were obtained with tracheotomy tube connectors corresponding to tube sizes 8, 7, and 6. Note, for example, that the latter, when used at 3 atm abs, reduces maximal flow rate to less than one sixth of its normal value.

From Saltzman, et al. (31).

Changes in Cardiac Output and Local Blood Flow

Even at normal pressure, administration of oxygen produces a decrease in cardiac output (35,36), and it has now been shown that hyperbaric elevation of arterial P_{O_2} causes a reduction in cardiac output of 10-20 per cent in man (37). The change is primarily associated with bradycardia rather than reduced stroke volume, and arterial blood pressure remains essentially constant. The bradycardia can be prevented by vagotomy (38).

These observations suggest that the primary phenomenon is local vasoconstriction. This is known to occur in the brain (39,40) and retina (41,42). In dogs anesthetized with chloralose, Rennie and Knox (43) found that renal blood flow decreased to as little as 50 per cent of control values at 4 atm OHP. Rennie and Lanphier (unpublished data) demonstrated similar changes in man in preliminary experiments conducted at 2.2 atm of oxygen. Diminished glomerular filtration was also indicated in man. More recently, Schenk and Hahnloser (unpublished data) have studied dogs with electromagnetic flowmeters on the aortic arch

and renal arteries, and on the descending aorta below the renal arteries. They find diminished flow under OHP at all sites. Changes in the renal arteries and descending aorta, plus reported decreases in cerebral blood flow, do not fully account for the observed decrease in flow in the ascending aorta. It appears likely that vasoconstriction is a generalized phenomenon, greater in some organs such as the brain and kidneys but not confined to them.

As yet, no complications have been attributed to vasoconstriction under OHP. However, as discussed above, a decrease in blood flow of the order of 25 per cent as reported for human brain at 3.5 atm (40) and dog brain at 2.0 atm (44) can produce a large decrease in capillary, venous, and presumably tissue P_{O_2} below that expected in hyperbaric oxygenation assuming maintenance of normal blood flow.

The mechanism of decreased blood flow with oxygen is not known, but denervation of the kidney does not eliminate the phenomenon in that organ (43). This speaks against central vasoconstrictor activity but does not distinguish between purely local constriction and a possible hormonal mechanism. Lambertsen attributes the change in human cerebral blood flow to a complex sequence of events with ultimate lowering of arterial P_{CO_2}. How much reduction of cerebral blood flow by other mechanisms can be expected in man is not known.

SUMMARY

Physiological factors underlying potential therapeutic applications of high pressure have been reviewed. The most promising of these applications involve the use of oxygen under increased ambient pressure for the relief or prevention of hypoxia. There, the likelihood of benefit depends upon the nature and degree of the basic defect. For example, OHP can probably compensate for considerable reduction of blood oxygen capacity or blood flow when the number of functioning capillaries remains reasonably normal. It is not likely to provide much extension of the safe duration of circulatory arrest and offers little help for regions of total ischemia. Combination of hypothermia with hyperbaric oxygen offers increased benefit in most situations but is not likely to provide dramatic results in basically unpromising applications.

At the present time oxygen toxicity appears to be a serious obstacle to realization of legitimate expectations in OHP. Current understanding imposes severe limitations upon the duration of safe application of effective oxygen levels. As a result, the use of OHP may be limited to conditions in which the basic abnormality can be remedied in a short time or where benefit can be derived from intermittent therapy.

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Chapter V

DECOMPRESSION PROCEDURES

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The main factor likely to limit hyperbaric exposure in patients is the toxicity of oxygen as discussed in Chapter III. In many situations, a problem of almost equal magnitude concerns the decompression of medical personnel who accompany the patient but who breathe air during exposure.

The term decompression basically signifies nothing more than lowering the pressure that surrounds something. In diving and other forms of high-pressure work, however, the usual application of the word implies lowering an elevated pressure back to normal according to a predetermined pattern of controlled rates, specified stops, or both.

The need for decompression in this sense stems from familiar gas laws and almost self-evident facts of physics and physiology: When the body is exposed to increased pressure, added amounts of respired gas go into physical solution in the blood and tissues. (Metabolically inert gases like nitrogen are of primary concern.) The extra dissolved gas produces a state of supersaturation if the ambient pressure is then dropped. Formation of bubbles may follow and produce the undesirable consequences seen in decompression sickness.

Ill effects are avoided by keeping the ratios of dissolved gas pressure to ambient pressure within limits that will avoid consequential bubble formation. This can be accomplished by holding the exposure within certain limits of pressure and time to restrict the uptake of excess gas. It is also accomplished by following a decompression schedule that permits excess gas to leave via circulation and lungs at such a rate that critical pressure ratios do not develop as the ambient pressure is lowered.

Little beyond the basic outline of the decompression problem is as simple as this description suggests. The actual computation of decompression schedules is an involved process, and many factors remain uncertain and controversial. More detailed general discussions of the subject are available (1-4, 13), and virtually the entire published literature is covered by the Bibliographical Sourcebook of Compressed Air, Diving, and Submarine Medicine (8-10). Valuable recent information is found in naval reports that are difficult to obtain, but some of this is reflected in papers by Hempleman and Workman (11, 12).

AIR-DECOMPRESSION TABLES

Tentative decompression schedules can be computed using the data of experiment and experience in a framework of assumptions, but the results are always subject to the verdict of test and actual use. All satisfactory decompression procedures have been based on long and diligent work including large numbers of test exposures. The extent to which the resulting schedules can confidently be altered, extrapolated, or applied to unusual circumstances without extensive testing is very limited.

The needs of medical hyperbaric applications are not the same as those of diving and other older forms of high-pressure work. As a result, none of the existing decompression tables are perfectly suited even if satisfactory for their own purposes. Tables specifically designed for hyperbaric work should ultimately be developed, but the effort and time that this will require oblige us to proceed with the best that is now available.

Hyperbaric-chamber exposures are likely to resemble those of caisson and tunnel workers ("sandhogs"). The possibility of using tables designed for such work thus naturally suggests itself. Some of these tables also offer the greatest economy of decompression time. In these cases, however, examination of the tables and of the incidence of decompression sickness and late aseptic bone necrosis associated with their use argues strongly against their adoption.

On the other hand, some "sandhog" tables are notable for apparent safety and conservatism at least in certain ranges of pressure and duration of exposure. In this category are those

based upon principles set forth by Duffner (5) and embodied in the regulations of the states of New York (6) and Washington (7).

The New York tables generally require at least as much decompression time as the U.S. Navy tables. Unfortunately, they had to be designed to conform to the generally detrimental "split shift" approach demanded by New York labor and also embody arbitrary limitation of exposure time at increasing pressures. These features render them largely impractical for hyperbaric applications.

The new state of Washington standards provide schedules for decompression following exposure to pressures from 14 to 50 psig and for times from 30 minutes to presumed saturation. Decompression is conducted by continuous reduction of pressure at changing rates. The decompression times specified differ from the U.S. Navy diving schedules, being considerably longer for lesser pressures and shorter exposure times but markedly shorter for more extreme exposures. Forthcoming experience in the application of these schedules will be of great interest.

Analysis of navy diving tables rapidly narrows the choice to those of the United States and Great Britain. In the depths and times of exposure for which both provide schedules, the decompression times are quite similar. However, preference for the current U.S. Navy tables is readily justified by the greater range of exposures covered, by provision of a practical system for dealing with repetitive exposures, and by some saving of decompression time without an evident sacrifice of safety.

These tables are for pressure exposures in which air is the breathing medium throughout. At present, this is the usual situation for chamber personnel although other possibilities are important and will be discussed. In subsequent pages, the air decompression tables and accompanying information are reproduced, by permission, directly from the U.S. Navy Diving Manual (3). A commentary upon their application to hyperbaric-chamber operations is also provided. Other approaches to the problem of decompression are then taken up.

INTRODUCTION TO USE OF TABLES

The instructions and explanatory material that directly accompany the U.S. Navy air-decompression tables should be studied carefully. The following paragraphs are offered only as an introduction.

As far as decompression is concerned, there is little basic difference between an actual dive and an exposure to air under high pressure in a hyperbaric chamber. It is suggested that the principles be mastered in their original context as applied to relatively simple situations in diving. If this is done, the necessary modifications and added complexities of the hyperbaric situation can then be considered with relative ease.

The U.S. Navy tables consider all air-breathing dives under one of two headings: (1) single dives, where only one exposure to pressure needs to be considered, and (2) repetitive dives, where an individual has had earlier exposure to pressure within the previous 12 hours. Decompression from single dives is a simple matter, while repetitive dives are complicated by the fact that excess nitrogen remains in the body from previous exposure and must be taken into account.

In practice, the tables are applied in essentially the same way whether one is determining proper decompression for an exposure in progress or planning a procedure in advance. Where the pressure and duration of an exposure can be decided and fixed in advance, use of the tables in planning offers considerable advantage. Small differences in pressure or time sometimes make large differences in the length of decompression stops required. Basic steps in the use of the tables can be outlined as follows:

Single Dives

1. Express the pressure of exposure in feet of sea water and the "bottom time" in minutes. Note that the "bottom time" in diving is defined to include the entire period from the beginning of descent to the beginning of ascent. Hyperbaric procedures may require modification of this rule (see page 72).

2. Go to Table 1-5 (page 62). In the first column, "Depth," find the tabulated value equal to or next greater than the actual exposure. Example: 66 feet, use 70 feet.

3. In the section of Table 1-5 selected above, go to the second column (bottom time) and find the tabulated value equal to or next greater than the actual time. Examples: 45 minutes, use 50 minutes; 112 minutes, use 120.

4. Having thus located the appropriate line (schedule) in Table 1-5, follow it to the right to the column labled Decompression stops. The numbers at the head of this column (50, 40, 30, etc.) signify depths at which stops

may be required. Note the number(s) that appear on the schedule line. If the number is zero, as for example in the 70 feet/50 minute schedule, this means that no decompression stops are required. The numbers appearing in the 100 feet/120 minute schedule, for example, indicate that these decompression stops must be made on ascent: 30 feet, 12 minutes; 20 feet, 41 minutes; 10 feet, 78 minutes.

5. In the unlikely event that either the depth or the time is greater than can be found in Table 1-5 (note that this is in two parts and extends to 190 feet), go to Table 1-9 (page 67).

6. If addition of a "safety factor" is desired, do this in a systematic manner: use the next greater increment of depth or time (or both) beyond that required by the actual exposure.

7. In conducting the exposure, make sure that the rate of ascent (decrease of pressure) is appropriate. A standard rate of 60 feet/minute is specified for diving, but hyperbaric procedures may require modification of this rule (see page 71).

Repetitive Dives

The U.S. Navy system for determining proper decompression for a repetitive dive is based upon approximation of the amount of excess nitrogen remaining in certain tissues of the body. Different amounts of nitrogen are represented by arbitrary letters designating various repetitive groups. "A" represents the smallest amount considered; "Z" represents the greatest amount.

The operation of the system can be considered most readily with the help of examples. Let us say that a diver has just surfaced from a 100 foot/120 minute dive and was decompressed on the corresponding schedule of Table 1-5. The last column of this schedule indicates that he surfaced with "quantity Z" of excess gas. Another diver has just completed a 70 foot/50 minute dive. This required no decompression stops, and Table 1-5 refers us to Table 1-6 (page 63) for his repetitive group. Table 1-6 indicates that he retains "quantity J" of excess gas. We know that this excess will be lost gradually while the diver remains at the surface. Table 1-7 (page 64) tells us, for example, that the excess will drop from "quantity J" to "quantity F" if he spends more than 1 hour and 47 minutes at the surface, or to "quantity E" if he spends more than 2 hours and 20 minutes.

If a diver with "quantity F" then begins a second dive, we must determine what "quantity F" means in terms of time at the depth of this repetitive dive, let us say 60 feet. Table 1-8 (page 65) tells us that "quantity F" is equal to the amount of excess nitrogen taken up by a diver in 36 minutes at 60 feet. The diver thus begins his 60 foot dive in a state of nitrogen saturation equivalent to having spent, already, 36 minutes at that depth. In determining his decompression for that dive, he must therefore add 36 minutes to his actual bottom time. This yields the "equivalent single dive time," and this is used as the bottom time in applying Table 1-5. For example, an actual dive of 60 feet/55 minutes (which would normally require no decompression stops) demands, in this case, decompression for a bottom time of 55 + 36 = 91 minutes. The diver would thus use the schedule for 60 feet/100 minutes.

While the above paragraphs should serve as a useful introduction, the applicable tables and instructions must be understood thoroughly before repetitive exposures are attempted. Use of the Repetitive Dive Worksheet or an equivalent is strongly recommended.

One technicality deserves special emphasis: in some situations, simply adding the total time of successive dives yields shorter decompression than does the repetitive system. This approach is permissible only when the depth of the repetitive dive is the same as or greater than that of the previous dive.

APPLICATION OF U.S. NAVY AIR-DECOMPRESSION TABLES TO MEDICAL HYPERBARIC EXPOSURES

Decompression of Patients

Attendants and other personnel who have breathed air throughout a procedure conducted in a therapeutic chamber clearly require decompression according to the tables as indicated. A patient who has breathed pure oxygen for the entire period obviously does not. The properties of hemoglobin and the consumption of oxygen by the tissues tend to prevent very large quantities of oxygen from being dissolved in the body as a whole, and excesses tend to be utilized before significant bubble formation occurs. However, oxygen cannot be ignored as a source of decompression problems, and very rapid reduction of the pressure surrounding such a patient can only be condemned even as a supposed emergency procedure. The possibility of causing air embolism by excessively rapid decompression must be kept in mind. A maximum rate of 1-atmosphere reduction of pressure per minute is by no means too conservative.

SECTION 1.5 DIVING TABLES

1.5.1 GENERAL

The tables and procedures outlined herein have been developed to provide safety from the hazards of decompression sickness and oxygen toxicity described in section 1.3. At the same time, the tables have been made as efficient as possible in order that they will be the least possible hindrance to diving operations.

1.5.2 AIR DECOMPRESSION TABLES

General

(1) The air decompression tables comprise:

(*a*) Decompression Procedures (table 1–4).

(*b*) U.S. Navy Standard Air Decompression Table (table 1–5).

(*c*) "No Decompression Limits and Repetitive Groups" (table 1–6).

(*d*) Surface Interval Credit Table (table (1–7).

(*e*) Repetitive Dive Timetable (table 1–8).

(*f*) Standard Air Decompression Table for Exceptional Exposures (table 1–9).

(2) Regardless of the type of diving apparatus, for all dives where air is the breathing medium, use these tables as prescribed.

(3) Use these tables in conjunction with the Equivalent Air Tables (table 1–10) for dives where a nitrogen oxygen mixture is the breathing medium. (See art. 1.5.3 and sec. 3.6.)

Single dives

(4) A single dive is the first dive of the day. It is denoted by an exposure to a specific depth in feet for a specific time in minutes. An example would be 134 feet for 14 minutes. The depth is the maximum depth attained. The time is the actual bottom time. Bottom time is the elapsed time between leaving the surface in descent and leaving the deepest depth in ascent. A combination of depth and time listed in the decompression tables is called a dive schedule. All dives are included and covered in the next deeper and next longer schedule. Do not interpolate.

Repetitive dives

(5) Any dive performed within 12 hours of a previous dive is a *repetitive dive.* The period between dives is the *surface interval.* Decompression following a repetitive dive requires special consideration. This is because dissolved inert gas from the previous dive remains in the body at the *beginning* of the repetitive dive.

(6) A detailed consideration of all the factors involved would be prohibitively complicated. A simplified and workable solution is based on the degree of saturation of the "120 minute half-time tissue" (Experimental Diving Unit Research Report 6–57 documents the calculations and tests). The basic idea of this approach involves considering the previous dive, the surface interval, and the repetitive dive together as a whole to yield an *equivalent single dive.* For the *depth* of the equivalent single dive, the *actual* depth of the repetitive dive is used. But the *bottom time* is the sum of the actual time plus an additional amount of time to take into account the residual nitrogen from the previous dive and surface interval.

(7) Upon surfacing from a dive, the diver is catalogued by table 1–5 or 1–6 into one of 16 lettered *repetitive groups* in accordance with the amount of inert gas left in his body. During the surface interval the diver loses inert gas and is given "credit" for the loss by means of table 1–7 which shows the change from one group to another for various time intervals on the surface. For every depth of dive, there is a certain time of exposure that would bring the diver to the same degree of saturation as that represented by each repetitive group. This time, based on the residual inert gas from previous dive and surface interval, is called the *residual nitrogen time.* In table 1–8, residual nitrogen time is expressed as a number of minutes for various depths (in 10-foot increments) and for each repetitive group designation. The bottom time of the *equivalent single dive* is then obtained by adding this residual nitrogen time to the actual

bottom time of the repetitive dive being considered. The proper decompression for the ascent from the repetitive dive may then be found in the Standard Air Decompression Table (table 1–5) by using the actual depth of the repetitive dive and the equivalent single dive bottom time. Successive repetitive dives may be handled similarly.

U.S. Navy Standard Air Decompression Table

(8) The Standard Air Decompression Table (table 1–5) covers the normal range of diving. The depth limit is 190 feet and the bottom time limit for each depth is approximately 12,000 divided by the depth. This is an arbitrary time, but it is a good maximum for normal practice. Stay within the limits of this table for all routine air dives.

(9) Details on the use of the Standard Air Decompression Tables are:

(*a*) Time of decompression stops in the table is in minutes.

(*b*) Enter the tables at the listed depth that is exactly equal to or is the next greater than the maximum attained during the dive.

(*c*) Select the bottom time listed for the selected depth that is exactly equal or is next greater than the bottom time of the dive.

(*d*) Use the decompression stops listed on the line for the selected bottom time.

(*e*) For any repetitive diving, use the repetitive group designation listed on the same line (or if no decompression is required, obtain the repetitive group from table 1–6).

(*f*) Maintain the diver's *chest* as close as possible to each decompression depth for the number of minutes listed.

(*g*) The rate of ascent *between* stops is not critical. Commence timing each stop on arrival at the decompression depth and resume ascent when the specified time has elapsed.

(10) Specific examples of the use of the table are:

(*a*) You made a single dive to 82 feet for 36 minutes. You wish to determine the proper decompression procedure: The next greater depth listed in the table is 90 feet. The next greater bottom time listed opposite 90 feet is 40 minutes. The proper decompression procedure is therefore a 7 minute stop at 10 feet in accordance with the 90/40 schedule.

(*b*) You made a single dive to 110 feet for 30 minutes. You know that the depth did not exceed 110 feet. You wish to determine the proper decompression procedure: The exact depth of 110 feet is listed. The exact time of 30 minutes is listed opposite 110 feet. Decompress according to the 110/30 schedule unless the dive was particularly cold or arduous or conditions will prohibit accurate decompression. In any of these cases go to the 110/40, the 120/30 or the 120/40 schedule at your own discretion.

"No Decompression Table"

(11) The "No Decompression Table" is officially and more accurately titled "*No Decompression*" *Limits and Repetitive Group Designation Table for "No Decompression" Schedules.* It is a new table required by repetitive diving. It is no longer sufficient merely to know where decompression requirements begin. In repetitive diving you must know the amount of nitrogen remaining in the tissues from any dive, no matter how short or shallow. The repetitive group designations provide that information.

(12) Repetitive group designations are given for depths of 10 feet to 40 feet in 5-foot increments and for depths of 40 feet to 190 feet in 10-foot increments. Opposite each depth and each repetitive group is listed the maximum bottom time which will allow the diver to remain within the group. On the assumption that it is the operational limit, the times for 10 to 25 feet end at about 5 hours. From 40 feet on, the times end at the "no decompression" limit.

(13) The "no decompression" limits listed in this table for depths of 40 feet and greater are useful in planning operations. The diver may surface directly ("no decompression dive") as long as the bottom time is less than the maximum listed for the depth. For depths not greater than 33 feet, direct surfacing is permissible regardless of the bottom time.

GENERAL INSTRUCTIONS FOR AIR DIVING

Need for Decompression

A quantity of nitrogen is taken up by the body during every dive. The amount absorbed depends upon the depth of the dive and the exposure (bottom) time. If the quantity of nitrogen dissolved in the body tissues exceeds a certain critical amount, the ascent must be delayed to allow the body tissue to remove the excess nitrogen. Decompression sickness results from failure to delay the ascent and to allow this process of gradual desaturation. A specified time at a specific depth for purposes of desaturation is called a decompression stop.

"No Decompression" Schedules

Dives that are not long or deep enough to require decompression stops are "no decompression" dives. Dives to 33 feet or less do not require decompression stops. As the depth increases, the allowable bottom time for "no decompression" dives decreases. Five minutes at 190 feet is the shortest and deepest "no decompression" schedule. These dives are all listed in the No Decompression Limits and Repetitive Group Designation Table for "No Decompression" Dives, ("No Decompression Table" (table 1-6)) and only require compliance with the 60 feet per minute rate of ascent.

Schedules That Require Decompression Stops

All dives beyond the limits of the "No Decompression Table" require decompression stops. These dives are listed in the Navy Standard Air Decompression Table (table 1-5). Comply exactly with instructions except as modified by surface decompression procedures.

Variations in Rate of Ascent

Ascend from all dives at the rate of 60 feet per minute.
In the event you exceed the 60 feet per minute rate:
(1) If no decompression stops are required, but the bottom time places you within 10 minutes of a schedule that does require decompression; stop at 10 feet for the time that you should have taken in ascent at 60 feet per minute.
(2) If decompression is required; stop 10 feet below the first listed decompression depth for the time that you should have taken in ascent at 60 feet per minute.
In the event you are unable to maintain the 60 feet per minute rate of ascent:
(1) If the delay was within 30 feet of the bottom; add to the bottom time, the additional time used in ascent. Decompress according to the requirements of the total bottom time. This is the safer procedure.
(2) If the delay was above 30 feet from the bottom; increase the first stop by the difference between the time consumed in ascent and the time that should have been consumed at 60 feet per minute.

Repetitive Dive Procedure

A dive performed within 12 hours of surfacing from a previous dive is a repetitive dive. The period between dives is the surface interval. Excess nitrogen requires 12 hours to effectively be lost from the body. These tables are designed to protect the diver from the effects of this residual nitrogen. Allow a minimum surface interval of 10 minutes between all dives. Specific instructions are given for the use of each table in the following order:
(1) The "No Decompression Table" or the Navy Standard Air Decompression Table gives the repetitive group designation for all schedules which may precede a repetitive dive.
(2) The Surface Interval Credit Table gives credit for the desaturation occurring during the surface interval.
(3) The Repetitive Dive Timetable gives the number of minutes or residual nitrogen time to add to the actual bottom time of the repetitive dive in order to obtain decompression for the residual nitrogen.
(4) The "No Decompression Table" or the Navy Standard Air Decompression Table gives the decompression required for the repetitive dive.

U.S. NAVY STANDARD AIR DECOMPRESSION TABLE

INSTRUCTIONS FOR USE

Time of decompression stops in the table is in minutes.

Enter the table at the exact or the next greater depth than the maximum depth attained during the dive. Select the listed bottom time that is exactly equal to or is next greater than the bottom time of the dive. Maintain the diver's chest as close as possible to each decompression depth for the number of minutes listed. The rate of ascent between stops is not critical. Commence timing each stop on arrival at the decompression depth and resume ascent when the specified time has lapsed.

For example – a dive to 82 feet for 36 minutes. To determine the proper decompression procedure: The next greater depth listed in this table is 90 feet. The next greater bottom time listed opposite 90 feet is 40. Stop 7 minutes at 10 feet in accordance with the 90/40 schedule.

For example – a dive to 110 feet for 30 minutes. It is known that the depth did not exceed 110 feet. To determine the proper decompression schedule: The exact depth of 110 feet is listed. The exact bottom time of 30 minutes is listed opposite 110 feet. Decompress according to the 110/30 schedule unless the dive was particularly cold or arduous. In that case, go to the 110/40, the 120/30, or the 120/40 at your own discretion. (Rev. 1958)

TABLE 1-4.—*Decompression procedures.*

Reproduced from U.S. NAVY DIVING MANUAL

DEPTH (ft)	BOTTOM TIME (mins)	TIME TO FIRST STOP	DECOMPRESSION STOPS 50	40	30	20	10	TOTAL ASCENT TIME	REPET. GROUP
40	200						0	0.7	*
	210	0.5					2	2.5	N
	230	0.5					7	7.5	N
	250	0.5					11	11.5	O
	270	0.5					15	15.5	O
	300	0.5					19	19.5	Z
50	100						0	0.8	*
	110	0.7					3	3.7	L
	120	0.7					5	5.7	M
	140	0.7					10	10.7	M
	160	0.7					21	21.7	N
	180	0.7					29	29.7	O
	200	0.7					35	35.7	O
	220	0.7					40	40.7	Z
	240	0.7					47	47.7	Z
60	60						0	1.0	*
	70	0.8					2	2.8	K
	80	0.8					7	7.8	L
	100	0.8					14	14.8	M
	120	0.8					26	26.8	N
	140	0.8					39	39.8	O
	160	0.8					48	48.8	Z
	180	0.8					56	56.8	Z
	200	0.6				1	69	70.6	Z
70	50						0	1.2	*
	60	1.0					8	9.0	K
	70	1.0					14	15.0	L
	80	1.0					18	19.0	M
	90	1.0					23	24.0	N
	100	1.0					33	34.0	N
	110	0.8				2	41	43.8	O
	120	0.8				4	47	51.8	O
	130	0.8				6	52	58.8	O
	140	0.8				8	56	64.8	Z
	150	0.8				9	61	70.8	Z
	160	0.8				13	72	85.8	Z
	170	0.8				19	79	98.8	Z
80	40						0	1.3	*
	50	1.2					10	11.2	K
	60	1.2					17	18.2	L
	70	1.2					23	24.2	M
	80	1.0				2	31	34.0	N
	90	1.0				7	39	47.0	N
	100	1.0				11	46	58.0	O
	110	1.0				13	53	67.0	O
	120	1.0				17	56	74.0	Z
	130	1.0				19	63	83.0	Z
	140	1.0				26	69	96.0	Z
	150	1.0				32	77	110.0	Z
90	30						0	1.5	*
	40	1.3					7	8.3	J
	50	1.3					18	19.3	L
	60	1.3					25	26.3	M
	70	1.2				7	30	38.2	N
	80	1.2				13	40	54.2	N
	90	1.2				18	48	67.2	O
	100	1.2				21	54	76.2	Z
	110	1.2				24	61	86.2	Z
	120	1.2				32	68	101.2	Z
	130	1.0			5	36	74	116.0	Z
100	25						0	1.7	*
	30	1.5					3	4.5	I
	40	1.5					15	16.5	K
	50	1.3				2	24	27.3	L
	60	1.3				9	28	38.3	N
	70	1.3				17	39	57.3	O
	80	1.3				23	48	72.3	O
	90	1.2			3	28	57	84.2	Z
	100	1.2			7	23	66	97.2	Z
	110	1.2			10	34	72	117.2	Z
	120	1.2			12	41	78	132.2	Z
110	20						0	1.8	*
	25	1.7					3	4.7	H
	30	1.7					7	8.7	J
	40	1.5				2	21	24.5	L
	50	1.5				8	26	35.5	M
	60	1.5				18	36	55.5	N
	70	1.3			1	23	48	73.3	O
	80	1.3			7	23	57	88.3	Z
	90	1.3			12	30	64	107.3	Z
	100	1.3			15	37	72	125.3	Z

DEPTH (ft)	BOTTOM TIME (mins)	TIME TO FIRST STOP	DECOMPRESSION STOPS 50	40	30	20	10	TOTAL ASCENT TIME	REPET. GROUP
120	15						0	2.0	*
	20	1.8					2	3.8	H
	25	1.8					6	7.8	I
	30	1.8					14	15.8	J
	40	1.7				5	25	31.7	L
	50	1.7				15	31	47.7	N
	60	1.5			2	22	45	70.5	O
	70	1.5			9	23	55	88.5	O
	80	1.5			15	27	63	106.5	Z
	90	1.5			19	37	74	131.5	Z
	100	1.5			23	45	80	149.5	Z
130	10						0	2.2	*
	15	2.0					1	3.0	F
	20	2.0					4	6.0	H
	25	2.0					10	12.0	J
	30	1.8				3	18	22.8	M
	40	1.8				10	25	36.8	N
	50	1.7			3	21	37	62.7	O
	60	1.7			9	23	52	85.7	Z
	70	1.7			16	24	61	102.7	Z
	80	1.5		3	19	35	72	130.5	Z
	90	1.5		8	19	45	80	153.5	Z
140	10						0	2.3	*
	15	2.2					2	4.2	G
	20	2.2					6	8.2	I
	25	2.0				2	14	18.0	J
	30	2.0				5	21	28.0	K
	40	1.8			2	16	26	45.8	N
	50	1.8			6	24	44	75.8	O
	60	1.8			16	23	56	96.8	Z
	70	1.7		4	19	32	68	124.7	Z
	80	1.7		10	23	41	79	154.7	Z
150	5						0	2.5	C
	10	2.3					1	3.3	E
	15	2.3					3	5.3	G
	20	2.2				2	7	11.2	H
	25	2.2				4	17	23.2	K
	30	2.2				8	24	34.2	L
	40	2.0				5	19	33	59.0	N
	50	2.0				12	23	51	88.0	O
	60	1.8			3	19	26	62	111.8	Z
	70	1.8			11	19	39	75	145.8	Z
	80	1.7		1	17	19	50	84	172.7	Z
160	5						0	2.7	D
	10	2.5					1	3.5	F
	15	2.3					1	4	7.3	H
	20	2.3					3	11	16.3	J
	25	2.3					7	20	29.3	K
	30	2.2				2	11	25	40.2	M
	40	2.2				7	23	39	71.2	N
	50	2.0			2	16	23	55	98.0	Z
	60	2.0			9	19	33	69	132.0	Z
	70	1.8		1	17	22	44	80	165.8	Z
170	5						0	2.8	D
	10	2.7					2	4.7	F
	15	2.5					2	5	9.5	H
	20	2.5					4	15	21.5	J
	25	2.3				2	7	23	34.3	L
	30	2.3				4	13	26	45.3	M
	40	2.2			1	10	23	45	81.2	O
	50	2.2			5	18	23	61	109.2	Z
	60	2.0		2	15	22	37	74	152.0	Z
	70	2.0		8	17	19	51	86	183.0	Z
180	5						0	3.0	D
	10	2.8					3	5.8	F
	15	2.7					3	6	11.7	I
	20	2.5				1	5	17	25.5	K
	25	2.5				3	10	24	39.5	L
	30	2.5				6	17	27	52.5	N
	40	2.3			3	14	23	50	92.3	O
	50	2.2		2	9	19	30	65	127.2	Z
	60	2.2		5	16	19	44	81	167.2	Z
190	5						0	3.2	D
	10	2.8					1	3	6.8	G
	15	2.8					4	7	13.8	I
	20	2.7				2	6	20	30.7	K
	25	2.7				5	11	25	43.7	M
	30	2.5			1	8	19	32	62.5	N
	40	2.5			8	14	23	55	102.5	O
	50	2.3		4	13	22	33	72	146.3	Z
	60	2.3		10	17	19	50	84	182.3	Z

*See table 1-6 for repetitive groups in "no decompression" dives. (Rev. 1968)

TABLE 1-5.—*U.S. Navy standard air decompression table.*

Reproduced from U.S. NAVY DIVING MANUAL

DEPTH (ft.)	NO DECOMPRESSION LIMITS (Min.)	REPETITIVE GROUPS														
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
10	–	60	120	210	300											
15	–	35	70	110	160	225	350									
20	–	25	50	75	100	135	180	240	325							
25	–	20	35	55	75	100	125	160	195	245	315					
30	–	15	30	45	60	75	95	120	145	170	205	250	310			
35	310	5	15	25	40	50	60	80	100	120	140	160	190	220	270	310
40	200	5	15	25	30	40	50	70	80	100	110	130	150	170	200	
50	100	–	10	15	25	30	40	50	60	70	80	90	100			
60	60	–	10	15	20	25	30	40	50	55	60					
70	50	–	5	10	15	20	30	35	40	45	50					
80	40	–	5	10	15	20	25	30	35	40						
90	30	–	5	10	12	15	20	25	30							
100	25	–	5	7	10	15	20	22	25							
110	20	–	–	5	10	13	15	20								
120	15	–	–	5	10	12	15									
130	10	–	–	5	8	10										
140	10	–	–	5	7	10										
150	5	–	–	5												
160	5	–	–	–	5											
170	5	–	–	–	5											
180	5	–	–	–	5											
190	5	–	–	–	5											

(Rev. 1958)

INSTRUCTIONS FOR USE

I. "No decompression" limits

This column shows at various depths greater than 30 feet the allowable diving times (in minutes) which permit surfacing directly at 60 ft. a minute with no decompression stops. Longer exposure times require the use of the Standard Air Decompression Table (Table 1-5).

II. Repetitive group designation table

The tabulated exposure times (or bottom times) are in minutes. The times at the various depths in each vertical column are the maximum exposures during which a diver will remain within the group listed at the head of the column.

To find the repetitive group designation at surfacing for dives involving exposures up to and including the "no decompression limits": Enter the table on the exact or next greater depth than that to which exposed and select the listed exposure time exact or next greater than the actual exposure time. The repetitive group designation is indicated by the letter at the head of the vertical column where the selected exposure time is listed.

For example: A dive was to 32 feet for 45 minutes. Enter the table along the 35 ft. depth line since it is next greater than 32 ft. The table shows that since group "D" is left after 40 minutes exposure and group "E" after 50 minutes, group "E" (at the head of the column where the 50 min. exposure is listed) is the proper selection.

Exposure times for depths less than 40 ft. are listed only up to approximately five hours since this is considered to be beyond field requirements for this table.

TABLE 1-6.—*"No decompression" limits and repetitive group designation table for "no decompression" dives.*

Reproduced from U.S. NAVY DIVING MANUAL

REPETITIVE GROUP AT THE END OF THE SURFACE INTERVAL																
REPETITIVE GROUP AT THE BEGINNING OF SURFACE INTERVAL (FROM PREVIOUS DIVE)	Z	O	N	M	L	K	J	I	H	G	F	E	D	C	B	A
Z	0:10-0:22	0:34	0:48	1:02	1:18	1:36	1:55	2:17	2:42	3:10	3:45	4:29	5:27	6:56	10:05	12:00*
O		0:10-0:23	0:36	0:51	1:07	1:24	1:43	2:04	2:29	2:59	3:33	4:17	5:16	6:44	9:54	12:00*
N			0:10-0:24	0:39	0:54	1:11	1:30	1:53	2:18	2:47	3:22	4:04	5:03	6:32	9:43	12:00*
M				0:10-0:25	0:42	0:59	1:18	1:39	2:05	2:34	3:08	3:52	4:49	6:18	9:28	12:00*
L					0:10-0:26	0:45	1:04	1:25	1:49	2:19	2:53	3:36	4:35	6:02	9:12	12:00*
K						0:10-0:28	0:49	1:11	1:35	2:03	2:38	3:21	4:19	5:48	8:58	12:00*
J							0:10-0:31	0:54	1:19	1:47	2:20	3:04	4:02	5:40	8:40	12:00*
I								0:10-0:33	0:59	1:29	2:02	2:44	3:43	5:12	8:21	12:00*
H									0:10-0:36	1:06	1:41	2:23	3:20	4:49	7:59	12:00*
G										0:10-0:40	1:15	1:59	2:58	4:25	7:35	12:00*
F											0:10-0:45	1:29	2:28	3:57	7:05	12:00*
E												0:10-0:54	1:57	3:22	6:32	12:00*
D													0:10-1:09	2:38	5:48	12:00*
C														0:10-1:39	2:49	12:00*
B															0:10-2:10	12:00*
A																0:10-12:00*

(Rev. 1958)

INSTRUCTIONS FOR USE

Surface interval time in the table is in hours and minutes ("7:59" means 7 hours and 59 minutes). The surface interval must be at least 10 minutes.

Find the repetitive group designation letter (from the previous dive schedule) on the diagonal slope. Enter the table horizontally to select the listed surface interval time that is exactly or next greater than the actual surface interval time. The repetitive group designation for the end of the surface interval is at the head of the vertical column where the selected surface interval time is listed. For example – a previous dive was to 110 ft. for 30 minutes. The diver remains on the surface 1 hour and 30 minutes and wishes to find the new repetitive group designation: The repetitive group from the last column of the 110/30 schedule in the Standard Air Decompression Tables is "J". Enter the surface interval credit table along the horizontal line labeled "J". The 1 hour and 47 min. listed surface interval time is next greater than the actual 1 hour and 30 minutes surface interval time. Therefore, the diver has lost sufficient inert gas to place him in group "G" (at the head of the vertical column selected).

*NOTE: Dives following surface intervals of more than 12 hours are not considered repetitive dives. Actual bottom times in the Standard Air Decompression Tables may be used in computing decompression for such dives.

TABLE 1-7.—*Surface interval credit table.*

Reproduced from U.S. NAVY DIVING MANUAL

REPET. GROUPS	REPETITIVE DIVE DEPTH (Ft.)															
	40	50	60	70	80	90	100	110	120	130	140	150	160	170	180	190
A	7	6	5	4	4	3	3	3	3	3	2	2	2	2	2	2
B	17	13	11	9	8	7	7	6	6	6	5	5	4	4	4	4
C	25	21	17	15	13	11	10	10	9	8	7	7	6	6	6	6
D	37	29	24	20	18	16	14	13	12	11	10	9	9	8	8	8
E	49	38	30	26	23	20	18	16	15	13	12	12	11	10	10	10
F	61	47	36	31	28	24	22	20	18	16	15	14	13	13	12	11
G	73	56	44	37	32	29	26	24	21	19	18	17	16	15	14	13
H	87	66	52	43	38	33	30	27	25	22	20	19	18	17	16	15
I	101	76	61	50	43	38	34	31	28	25	23	22	20	19	18	17
J	116	87	70	57	48	43	38	34	32	28	26	24	23	22	20	19
K	138	99	79	64	54	47	43	38	35	31	29	27	26	24	22	21
L	161	111	88	72	61	53	48	42	39	35	32	30	28	26	25	24
M	187	124	97	80	68	58	52	47	43	38	35	32	31	29	27	26
N	213	142	107	87	73	64	57	51	46	40	38	35	33	31	29	28
O	241	160	117	96	80	70	62	55	50	44	40	38	36	34	31	30
Z	257	169	122	100	84	73	64	57	52	46	42	40	37	35	32	31

(Rev. 1958)

INSTRUCTIONS FOR USE

The bottom times listed in this table are called "residual nitrogen times" and are the times a diver is to consider he has already spent on bottom when he starts a repetitive dive to a specific depth. They are in minutes.

Enter the table horizontally with the repetitive group designation from the Surface Interval Credit Table. The time in each vertical column is the number of minutes that would be required (at the depth listed at the head of the column) to saturate to the particular group.

For example – the final group designation from the Surface Interval Credit Table, on the basis of a previous dive and surface interval, is "H". To plan a dive to 110 feet, determine the "residual nitrogen time" for this depth required by the repetitive group designation: Enter this table along the horizontal line labeled "H". The table shows that one must start a dive to 110 feet as though he had already been on the bottom for 27 minutes. This information can then be applied to the Standard Air Decompression table or "No Decompression" Table in a number of ways:

(1) Assuming a diver is going to finish a job and take whatever decompression is required, he must add 27 minutes to his actual bottom time and be prepared to take decompression according to the 110 foot schedules for the sum or equivalent single dive time.

(2) Assuming one wishes to make a quick inspection dive for the minimum decompression, he will decompress according to the 110/30 schedule for a dive of 3 minutes or less (27 + 3 = 30). For a dive of over 3 minutes but less than 13, he will decompress according to the 110/40 schedule (27 + 13 = 40).

(3) Assuming that one does not want to exceed the 110/50 schedule and the amount of decompression it requires, he will have to start ascent before 23 minutes of actual bottom time (50 - 27 = 23).

(4) Assuming that a diver has air for approximately 45 minutes bottom time and decompression stops, the possible dives can be computed: A dive of 13 minutes will require 23 minutes of decompression (110/40 schedule), for a total submerged time of 36 minutes. A dive of 13 to 23 minutes will require 34 minutes of decompression (110/50 schedule), for a total submerged time of 47 to 57 minutes. Therefore, to be safe, the diver will have to start ascent before 13 minutes or a standby air source will have to be provided.

TABLE 1-8.—*Repetitive dive timetable.*

Reproduced from U.S. NAVY DIVING MANUAL

(14) Other than the above uses to obtain "no decompression" limits, the only purpose of this table is to provide the repetitive group designation for "no decompression" dives. This knowledge is necessary to make repetitive dives after "no decompression" dives.

(15) Details and an example of its use to obtain the repetitive group designations are given directly on the table.

Surface Interval Credit Table

(16) The Surface Interval Credit Table is another requirement of the repetitive diving system. It is the real reason for the success and efficiency of the repetitive dive system.

(17) The diver continues to lose nitrogen while he is on the surface until he is completely desaturated. This requires 12 hours or more. In order to provide efficient decompression instructions, it is necessary to know the amount of nitrogen remaining in the tissues at the time a repetitive dive commences. This table provides that information.

(18) The repetitive groups are the measuring units. In this table, the loss of inert gas with increasing length of surface interval is reflected in the change from one group to another.

(19) Details and an example of its use are given directly on the table.

Repetitive Dive Timetable

(20) The Repetitive Dive Timetable lists the number of minutes at each depth that will build up the nitrogen partial pressure represented by each repetitive group.

(21) Knowing the diver's repetitive group designation, the system gives an arbitrary bottom time (the residual nitrogen time) that he must assume he has already completed when he starts his repetitive dive. This arbitrary bottom time and the actual bottom time of the repetitive dive are added to yield the bottom time of the equivalent single dive mentioned previously.

(22) Details and an example of its use are given directly on the tables.

(23) There is one exception to the table. It occasionally occurs when the repetitive dive is to the same or greater depth than the initial dives and the surface interval is short. Because of the necessity to account for the greatest exposure within a group, the arbitrary bottom time assigned may be greater than the sum of the actual bottom times of the previous dives. In such case, if the repetitive dive is to the same or greater depth than the previous dive or dives, add the actual bottom time of the previous dives to the actual bottom time of the repetitive dive.

Decompression for exceptional exposures

(24) The U.S. Navy Standard Air Decompression Table for Exceptional Exposures (table 1–9) includes only schedules of decompression for exceptional or emergency cases. Schedules are provided for "complete saturation" exposures up to 140 feet, and for extreme exposures up to 300 feet. Great demands are imposed upon the diver's endurance by emergencies which might necessitate use of the table. Therefore complete assurance of success of the schedules is impossible. They have, however, been tested to every practicable limit and found reasonably safe.

(25) Repetitive group designations are not given on the Table for Exceptional Exposures. Never follow a dive covered by that table with a repetitive dive. Make every effort to limit the equivalent single dive schedule of repetitive dives to the Standard Air Decompression Tables. The diving officer must be the one to weigh the need for any dive in the Table for Exceptional Exposures against the increased danger and demands on the diver's physical endurance.

Repetitive dive worksheet

(26) Figure 1–32 is a suggested worksheet for the selection of decompression schedules in repetitive diving. A systematic approach of this kind must *always* be used in applying the repetitive diving tables. (Fig. 1–32A can be removed from the manual and reproduced locally.)

(27) An example using figure 1–32 follows. A diver makes a dive to 105 feet with a bottom time of 24 minutes and decompresses properly according to the Standard Air Decompression Table. After being on the surface for 2 hours,

DEPTH (ft.)	BOTTOM TIME (Min.)	TIME TO FIRST STOP	DECOMPRESSION STOPS 130 120 110 100 90 80 70 60 50 40 30 20 10	TOTAL ASCENT TIME
40	360	0.5	23	24
	480	0.5	41	42
	720	0.5	69	70
60	240	0.7	2 79	82
	360	0.7	20 119	140
	480	0.7	44 148	193
	720	0.7	78 187	266
80	180	1.0	35 86	121
	240	0.8	6 53 120	179
	360	0.8	26 80 160	266
	480	0.8	59 107 187	354
	720	0.7	17 108 142 187	455
100	180	1.0	1 29 83 116	202
	240	1.0	14 42 84 142	283
	360	0.8	2 42 78 111 187	416
	480	0.8	21 61 91 142 187	502
	720	0.8	55 106 129 142 187	619
120	120	1.3	10 19 47 96	176
	180	1.2	5 27 37 76 137	283
	240	1.2	23 35 60 97 179	396
	360	1.0	15 45 64 83 142 187	550
	480	0.8	3 41 64 93 122 142 187	653
	720	0.8	32 74 100 114 122 142 187	772
140	90	1.5	2 14 18 42 88	166
	120	1.3	12 14 36 56 120	240
	180	1.3	10 26 33 54 94 168	386
	240	1.2	5 26 34 50 78 124 187	511
	360	1.0	9 32 42 64 84 122 142 187	683
	480	1.0	31 44 59 100 114 122 142 187	800
	720	0.8	16 56 88 97 100 114 122 142 187	923
170	90	1.8	12 12 14 34 82 190	332
	120	1.5	2 10 12 18 32 42 82 156	366
	180	1.3	4 10 22 26 34 50 78 120 187	535
	240	1.3	18 34 30 42 50 70 116 142 187	681
	360	1.2	22 34 40 52 60 88 116 132 142 187	873
	480	1.0	14 40 42 56 91 97 100 114 122 142 187	1006
200	5	3.2	1	5
	10	3.0	1 4	5
	15	2.8	1 4 10	15
	20	2.8	3 7 27	40
	25	2.8	7 14 25	49
	30	2.7	2 9 22 37	73
	40	2.5	2 8 17 33 53	113
	50	2.5	6 16 22 39 75	161
	60	2.3	2 13 17 34 51 80	199
	90	1.8	1 10 10 12 12 30 38 74 134	323
	120	1.7	6 10 10 10 24 28 40 64 96 160	479
	150	1.5	1 10 10 18 24 24 42 48 70 106 142 187	684
	240	1.3	6 20 24 24 38 42 54 68 114 122 142 187	841
	360	1.2	13 22 36 40 44 56 62 86 100 114 122 142 187	1067
210	5	3.3	1	5
	10	3.2	2 4	10
	15	3.0	1 5 15	22
	20	3.0	4 10 26	40
	25	2.8	2 7 17 27	56
	30	2.8	4 9 24 41	81
	40	2.7	4 9 19 26 63	124
	50	2.5	1 9 17 19 45 80	174
220	5	3.5	2	6
	10	3.3	2 5	11
	15	3.3	2 5 16	27
	20	3.0	1 3 11 24	43
	25	3.0	3 8 19 33	66
	30	2.8	1 7 10 23 47	91
	40	2.8	6 13 23 29 68	140
	50	2.7	3 13 17 18 51 86	190

DEPTH (ft.)	BOTTOM TIME (Min.)	TIME TO FIRST STOP	DECOMPRESSION STOPS 130 120 110 100 90 80 70 60 50 40 30 20 10	TOTAL ASCENT TIME
230	5	3.7	2	6
	10	3.5	1 2 6	13
	15	3.3	3 6 18	31
	20	3.2	2 8 12 26	49
	25	3.2	4 8 22 37	73
	30	3.0	2 8 12 33 51	99
	40	2.8	1 7 15 22 34 74	156
	50	2.8	5 14 16 24 51 89	203
240	5	3.8	2	6
	10	3.5	1 3 9	14
	15	3.5	4 8 21	33
	20	3.3	3 6 15 28	53
	25	3.2	1 4 9 24 40	82
	30	3.2	4 8 15 22 56	109
	40	3.0	3 7 17 22 39 75	168
	50	2.8	1 8 15 16 20 81 94	217
250	5	3.6	1 2	7
	10	3.7	1 4 7	16
	15	3.5	1 4 7 22	36
	20	3.5	4 7 17 27	59
	25	3.3	2 7 10 24 48	92
	30	3.3	6 7 17 23 59	116
	40	3.2	8 9 17 19 48 79	178
	50	2.7	4 10 10 10 12 22 36 64 126	297
	60	2.2	8 10 10 10 10 10 28 28 44 66 96 154	513
	90		(SEE EXTREME EXPOSURES BELOW)	
	120			
	150			
	240			
260	5	4.0	1 3	7
	10	3.8	2 4 9	19
	15	3.7	2 4 10 22	42
	20	3.5	1 4 7 20 31	67
	25	3.5	3 8 11 23 50	98
	30	3.3	2 6 8 19 26 61	125
	40	3.2	1 6 11 16 19 49 84	190
270	5	4.2	1 3	9
	10	4.0	2 5 11	22
	15	3.8	3 4 11 24	46
	20	3.7	2 3 8 21 38	74
	25	3.5	2 3 8 15 23 53	106
	30	3.5	3 6 12 22 27 64	138
	40	3.3	5 6 11 17 22 51 88	204
280	5	4.3	2 2	9
	10	4.0	1 2 5 13	26
	15	3.8	1 3 4 11 26	49
	20	3.8	3 4 5 23 39	81
	25	3.7	2 5 7 16 23 56	113
	30	3.5	1 3 7 13 22 30 70	150
	40	3.3	1 6 6 13 17 27 51 93	216
290	5	4.5	2 3	10
	10	4.3	1 3 5 16	30
	15	4.0	1 3 6 12 36	62
	20	4.0	3 7 9 23 43	89
	25	3.8	3 6 8 17 23 60	120
	30	3.7	1 5 6 16 22 36 72	162
	40	3.5	3 5 7 15 16 22 51 96	264
300	5	4.7	3 3	11
	10	4.3	1 3 6 17	32
	15	4.2	2 3 6 15 28	56
	20	4.0	2 3 7 10 23 47	104
	25	3.8	1 3 6 8 19 26 61	128
	30	3.8	2 5 7 17 22 39 75	171
	40	3.7	4 6 9 15 17 34 51 90	234
	60	3.0	4 10 10 10 10 10 14 28 32 50 90 167	466
	90		(SEE EXTREME EXPOSURES BELOW)	
	120			
	150			
	180			

(Rev. 1958)

EXTREME EXPOSURES – 250 AND 300 FT.

DEPTH (ft.)	BOTTOM TIME (Min.)	TIME TO FIRST STOP	DECOMPRESSION STOPS 200 190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10	TOTAL ASCENT TIME
250	120	1.8	5 10 10 10 10 16 34 24 36 48 64 94 142 187	682
	180	1.8	4 8 8 10 22 24 24 32 42 44 60 84 114 122 142 187	929
	240	1.8	9 14 21 22 22 40 40 42 56 76 98 100 114 122 142 187	1107
300	90	2.3	3 8 8 10 10 10 10 16 24 24 34 48 64 90 142 187	691
	120	2.0	4 8 8 10 14 24 24 24 34 42 56 68 102 122 142 187	887
	180	1.7	6 8 8 8 14 20 21 21 28 40 40 48 56 82 96 100 114 122 142 187	1168

TABLE 1-9.—*U.S. Navy standard air decompression table for exceptional exposures.*

REPETITIVE DIVE WORKSHEET

I. PREVIOUS DIVE:

24 minutes
105 feet
} see table 1-5 or 1-6 for repetitive group designation } Group H

II. SURFACE INTERVAL:

2 hours 0 minutes on surface
Group H (from I.)
} see table 1-7 for new group } Group E

III. RESIDUAL NITROGEN TIME:

145 feet (depth of repetitive dive)
Group E (from II.)
} see table 1-8 } 12 minutes

IV. EQUIVALENT SINGLE DIVE TIME:

12 minutes (residual nitrogen time from III.)

(add) 15 minutes (actual bottom time of repetitive dive)

(sum) 27 minutes

V. DECOMPRESSION FOR REPETITIVE DIVE:

27 minutes (equivalent single dive time from IV.)
145 feet (depth of repetitive dive)
} see table 1-5 or 1-6

☐ No decompression required

or

Decompression stops: 20 feet 8 minutes
10 feet 24 minutes
____ feet ____ minutes
____ feet ____ minutes

FIGURE 1-32.—Repetitive dive worksheet (filled in).

he is required to make a second dive, this time to 145 feet. It is anticipated that 15 minutes bottom time will be required to complete his work. The problem is to determine the proper decompression for this second or *repetitive dive*. Use the time and depth of his first or *previous dive* in worksheet part I. Table 1–5 indicates that he is in repetitive group "H" (according to the 110/25 schedule). During the surface interval of 2 hours he loses sufficient nitrogen to change from group "H" to group "E" according to the Surface Interval Credit Table (table 1–7). His residual nitrogen time may now be determined using the depth of his second or repetitive dive and the *new* group from the end of the surface interval by referring to the Repetitive Dive Timetable (table 1–8). This indicates that the diver's residual nitrogen time is 12 minutes. The 15 minute actual bottom time of the repetitive dive is added to the residual nitrogen time to obtain the *equivalent single dive time* which is 27 minutes. This is used, as indicated in worksheet part V, to select the decompression schedule for the repetitive dive; in this case from table 1–5, the 150/30 schedule.

More than one repetitive dive

(28) When one repetitive dive is to be followed by another, the procedure for selecting the proper decompression schedule for the first repetitive dive is *repeated*. The time and depth of the equivalent single dive of the *first* repetitive dive calculation becomes the time and depth of the "previous dive" of the *second* repetitive dive calculation. That is, the time and depth used in the worksheet part V (fig. 1–32) become the time and depth in part I of the following worksheet.

Patients who have not breathed pure oxygen, either by intent or because of poor methods of administration, or who have breathed air during part of their exposure, can present perplexing problems of decompression. If they are accompanied throughout by personnel breathing air, the decompression required by those individuals can be assumed to be adequate for the patient. Otherwise, some estimate of the patient's exposure to air must be made and an appropriate schedule adopted accordingly. Since such estimates may be subject to serious error, the only certain procedure is to decompress such a patient as if he had been breathing air for the entire period. Use of nitrous oxide for anesthesia can greatly complicate decompression of the patient.

Units of Pressure

The intended use of the U.S. Navy tables led naturally to their tabulation in feet of sea water, but there is no justification for adopting this unit of pressure for medical hyperbaric purposes. It is not yet clear what unit will come to predominate in hyperbaric practice, and it is doubtful that translation of the tables themselves into another unit would be worthwhile. This suggests that conversions will remain necessary and that actual chamber pressures will seldom correspond exactly to the increments of the tables.

The simple tables provided here (Pressure Equivalents) will assist determination of the U.S. Navy schedule to be applied for a given

PRESSURE EQUIVALENTS FOR SELECTION OF DECOMPRESSION SCHEDULES
when using decompression tables tabulated in feet of sea water.

ATMOSPHERES, ABSOLUTE		MILLIMETERS OF MERCURY (absolute pressure)		POUNDS PER SQ. INCH (gage pressure)	
When press. does not exceed...	Use schedules for...	When press. does not exceed...	Use schedules for...	When press. does not exceed...	Use schedules for...
2.2 atm	40 feet	1650 mm	40 feet	17 psi	40 feet
2.5 abs	50	1900 Hg	50	22 gage	50
2.8 (1)	60	2100 abs	60	26 (3)	60
3.1	70	2350 (2)	70	31	70
3.4	80	2600	80	35	80
3.7	90	2800	90	40	90
4.0	100	3050	100	44	100
4.3	110	3250	110	48	110
4.6	120	3500	120	53	120
4.9	130	3750	130	57	130
5.2	140	3950	140	62	140
5.5	150	4200	150	66	150
5.8	160	4400	160	71	160
6.1	170	4650	170	75	170
6.4	180	4900	180	80	180
6.7	190	5100	190	84	190
7.0	200	5350	200	89	200
7.3	210	5550	210	93	210
7.6	220	5800	220	97	220
7.9	230	6050	230	102	230
8.2	240	6250	240	106	240

Notes: (1) Rounded to 0.1 atm increment next below true equivalent pressure.
(2) Rounded to 50 mm Hg increment next below true equivalent pressure.
(3) Rounded to 1.0 psi increment next below true equivalent pressure.

Important: Except that equivalents have been rounded to a value slightly below true pressure in some cases, no "safety factor" is provided by these tables.

chamber pressure. Graphs, slide rules, and other devices can be prepared to expedite the process, or the foot indications of the tables can be replaced with appropriate values in the units employed, being careful that rounding off of numbers is done in the direction of safety.

Increments of Pressure

Table 1-5 is tabulated in 10-foot increments of pressure throughout, but part of Table 1-9 is in 20-foot steps. For example, exposure to air at 3 atm abs (66 feet) will require use of the 70-foot schedules of Table 1-5 for times up to 170 minutes; but beyond that duration of exposure the 80-foot schedules of Table 1-9 must be used. The extra decompression time required by the 4-foot "safety factor" afforded in the first instance should not be excessively burdensome and may prove to be highly desirable. The 14-foot discrepancy between actual chamber pressure and the 80-foot schedules of Table 1-9 is more serious in terms of added time although it, too, may prove appropriate.

Attempts at interpolation between the pressure increments of the tables are expressly forbidden in Navy diving and may have unfortunate results in any but skilled hands. If such interpolations appear desirable in the light of hyperbaric experience, it is hoped that the U.S. Navy Experimental Diving Unit will produce and promulgate them. Until such time, it is strongly recommended that the tables be used according to the instructions and that the resulting "safety factors" be accepted when they appear. The only alternative that can be suggested is to employ a chamber pressure that permits use of shorter schedules. In the example cited, using 2.8 atm abs instead of 3.0 would permit use of the 60-foot schedules, sacrificing a small amount of hyperbaric effect for the sake of much shorter, albeit less conservative, decompression time.

The 10-foot increments of the decompression stops should be observed as closely as possible. It is suggested that gages used for decompression be marked accurately for the stops most likely to be employed (probably 10-40 feet).

Increments of Time

The five- and ten-minute time increments of Table 1-5 should be adequate for all purposes. The much larger increments of Table 1-9, on the other hand, will inevitably impose unnecessarily burdensome decompression times under some conditions. For example, a 240-minute exposure using the 80-foot schedule will require 179 minutes of total decompression time, while 241 minutes of exposure require 280 minutes of decompression. As in the case of pressure increments, however, homemade interpolations will not "stand up in court" either literally or figuratively.

Until schedules with smaller increments of time are provided officially in these ranges, the best advice is to make every effort to avoid placing chamber personnel in the position of requiring decompression times of this magnitude in the first place; at least to avoid situations like that of the example.

Rate of Application of Pressure

The maximum rate of "descent" specified in the Diving Manual is 75 feet per minute. The "bottom time" is measured from the beginning of pressurization. In most hyperbaric installations, the rate will be limited by the capacity of the air system if not by equalization problems in patients or personnel. Barring equalization problems and assuming that pressurization can be stopped immediately if these develop, there is usually no need to specify a maximum rate. From the standpoint of barotrauma, however, pressure should not increase more than 0.1 atm from the time an individual signals distress. Problems of communication and control may make it necessary to limit the rate of pressurization in some installations on this basis.

Where the rate of pressurization is very slow, or where the process must be interrupted for some reason, it will seem unreasonable to include the entire period of pressurization in the "bottom time." No simple alternative will fit all such circumstances. However, no less than half of the period of compression at a uniform rate should be included. (The problem is discussed further under Multilevel Exposures.)

Rate of Decreased Pressure

The U.S. Navy tables are based upon reduction of pressure at the rate of 60 feet per minute (1 foot per second) to the first decompression stop. The instructions indicate that maintaining this rate is important and that deviations require compensatory measures. The reasoning behind this is that if the pressure

is reduced more rapidly, loss of gas during this phase will be less than anticipated in computation of the tables; that slower reduction of pressure may involve, in effect, a longer stay at pressures where some tissues may still be taking up gas. In practice, difficulties are not likely to arise unless the "bottom time" of the schedule being used is quite close to the actual exposure time; but the range of latitude is not readily defined.

In medical hyperbaric practice, rigid adherence to 1 foot per second reduction of pressure is indefensible. Slower rates are inevitably more practical, safer, and generally more desirable. The prescribed 60 feet per minute is by no means an excessively rapid rate in diving or chamber research, but it may readily be so in dealing with patients with unknown degrees of pulmonary obstruction, in whom closed gas pockets may have been created by some procedure, or who may be affected by some totally unforeseen problem of gas expansion. In situations of incomplete obstruction, the slower the rate of pressure drop the more likely the expanding gas is to escape harmlessly. In any case, more time is available for discovery and correction of a problem before serious harm results.

The most reasonable course is to make the rate of pressure reduction fit the circumstances, letting 60 feet per minute be the maximum if no problems can reasonably be expected and using very slow rates where real doubt exists. If a specific rate must be adopted for routine use, 0.3 atm per minute (10 feet per minute) is not an unreasonable figure. As in diving, however, compensation for alteration of the rate should be provided. This is especially necessary if any prolonged interruption of "ascent" occurs near the pressure of original exposure.

Bottom Time

In hyperbaric practice, there is much to recommend redefining "bottom time" as the elapsed time between the initial increase of pressure and reaching the first decompression stop or 30 feet (whichever is reached first) upon reduction of pressure. This definition necessarily requires commencing decompression sufficiently in advance of tabulated times to avoid being obliged to use a longer schedule than intended.

Direct reduction of pressure to normal can be tolerated after indefinite periods at 2 atm abs or less. At such pressures, therefore, no tissue will gain gas to reach a dangerous level of saturation; and there is no need to add to the "bottom time" periods spent at such pressures during the decompression phase. (On the other hand, periods spent at such pressures during compression must be taken into account since thay can add significantly to the total gas uptake during exposure. For similar reasons, an unusual prolongation of decompression will greatly complicate a subsequent repetitive exposure.)

Decompression Stops

As mentioned above, the specified pressures for the stops should be maintained as accurately as possible. The time, assuming that it is no shorter than specified, is less crucial. At any stop less than 40 feet, an indefinitely longer time may be spent without compromising the decompression process, although the complicating effect on repetitive exposures is a serious problem.

"Stage decompression," as decompression involving stops is known, is not the most efficient method of ridding the body of dissolved gas. Continuous reduction of pressure at changing rates maintains the greatest outward pressure-difference for gas elimination consistent with the risk of bubble formation and, thus, accelerates the process. The difference in times may be significant for longer exposures, but convenience favors the use of stops. It is much simpler to maintain a constant pressure for a specified period than to maintain accurate rates of continuous pressure reduction. However, the latter can be accomplished by special controls and may come to be considered practical for decompression procedures designed specifically for hyperbaric practice. At present, no acceptable tables using continuous reduction of pressure are available for hyperbaric use.

Repetitive Exposures

The procedure for determining the decompression required for repetitive dives is adequately described and less complex than it appears. It provides a safe method of dealing with repeated exposures with less than 12 hours between them. If the interval is greater than 12 hours, the standard tables are used.

When the pressure of a successive exposure is the same as that of the previous one, simple addition of the total time of both exposures will sometimes yield a shorter decompression

period than use of the repetitive-dive tables. If so, the shorter schedule can be used.

Occasionally, the same is also true when the second exposure is to a higher pressure than the first. Again, the most favorable schedule can be used. Note that the higher pressure of the two exposures must be used with the total time to determine the proper schedule. In no case may the lesser of two exposure pressures be used with the total time to determine the decompression schedule to be employed.

Note that the repetitive-dive tables can only be used if the preceding exposure falls within the limits of Table 1-5 or 1-6. If repetitive exposures are required beyond the limits of Table 1-5, the only safe procedure is to use the total time with the highest exposure pressure to determine the schedule to follow. This may require extremely prolonged decompression time that may be considerably in excess of the actual need, but there is no safe method for readily estimating the actual requirement. Consequently, such situations should be avoided if at all possible.

It is strongly recommended that the worksheet (Figure 1-32A) be reproduced in quantity, employed for every repetitive-dive computation, and filed with the chamber log. Repetitive-dive "computers" and such are useful for rapid determinations and for checking hand computations, but they cannot be viewed as a substitute.

It is important to note that the repetitive-dive tables are not to be employed when the "surface interval" is less than 10 minutes. In this case, use of the "total time—highest pressure" approach is the only authorized alternative. Whenever possible, it is desirable to extend the surface interval to 10 minutes to permit use of the repetitive-dive tables unless these offer no advantage over "total time—highest pressure."

Multilevel Exposure

Particularly difficult decompression problems are apt to be posed when a single exposure involves more than one level of pressure, and this is likely to be a frequent occurrence. For example, hyperbaric oxygenation in a particular patient might be started at 2 atm abs, but the exposure might then have to be carried to 4 atm in order to achieve the desired response. A longer period at 3 atm might then follow before decompression. If the same chamber personnel remained with the patient and breathed air throughout this procedure, they are faced with long decompression stops that they do not entirely need.

The U.S. Navy tables provide only one approved method of dealing with such an exposure: to assume that the entire period was spent at the highest pressure. The decompression time derived in this way will inevitably exceed the necessary minimum, sometimes to an obviously extreme degree. Unfortunately, the lack of more realistic provision for such situations simply reflects the difficulties involved.

It is extremely tempting in such situations to handle the periods at different pressures as a succession of repetitive dives without surface intervals between them. This of course would violate the rule concerning a minimum surface interval of 10 minutes, and there is at present no safe means of compensating for the difference. In many instances, the risk is probably acceptable; but no assurance of this can be given readily for any specific situation. Consequently, the application of repetitive-dive tables to multilevel exposures cannot be recommended here and should be employed, if at all, only as an emergency measure.

For the present, the best recommendation is simply to avoid multilevel exposures whenever possible; otherwise to rotate personnel in such a manner that the repetitive-dive tables can be used legitimately.

Exposures Not Covered by Tables

Extremely prolonged exposures can extend beyond the "bottom times" provided by the U.S. Navy tables, and no very satisfactory procedure for dealing with them can currently be provided. Needless to say, such exposures must be avoided if at all possible. The best rule now available for such circumstances is to follow Treatment Table 4 (page 97), entering it at a pressure not more than 40 feet shallower than that of the individual's exposure and never less than the tabulated 30-foot stop.

Omitted Decompression

Certain emergencies may interrupt or prevent observance of specified decompression procedures. These include uncontrollable loss of chamber pressure, temporary unavailability of a lock in an emergency, and other situations that are difficult to foresee in detail. The following discussion is based on U.S. Navy diving doctrine (Diving Manual, pp. 123-124).

If the omission results in symptoms of decompression sickness, immediate treatment by recompression is of course required.

REPETITIVE DIVE WORKSHEET

I. PREVIOUS DIVE:

___ minutes
____ feet
} see table 1-5 or 1-6 for repetitive group designation } Group___

II. SURFACE INTERVAL:

__hours___minutes on surface
Group___ (from I.)
} see table 1-7 for new group } Group___

III. RESIDUAL NITROGEN TIME:

____ feet (depth of repetitive dive)
Group___ (from II.)
} see table 1-8 ___minutes

IV. EQUIVALENT SINGLE DIVE TIME:

___ minutes (residual nitrogen time from III.)

(add) ___ minutes (actual bottom time of repetitive dive)

(sum) ___ minutes

V. DECOMPRESSION FOR REPETITIVE DIVE:

___ minutes (equivalent single dive time from IV.)
____ feet (depth of repetitive dive)
} see table 1-5 or 1-6

☐ No decompression required

or

Decompression stops: ____ feet ____ minutes
____ feet ____ minutes
____ feet ____ minutes
____ feet ____ minutes

FIGURE 1-32A.—Repetitive dive worksheet (sample for reproduction; see reverse side).

Reproduced from U.S. NAVY DIVING MANUAL

Tables for surface decompression (Tables 1-17 and 1-18, reproduced here) may be used only if (1) the emergency interval at normal pressure does not exceed 3.5 minutes and (2) the "water stops" required by the table have already been completed (or such stops were not required).

When the above conditions are not fulfilled, the individual's decompression has been compromised, and he requires recompression according to a treatment table whether evidence of decompression sickness is present or not. Take him to 100 feet (4 atm abs) in the chamber without delay and keep him there a minimum of 30 minutes. If no evidence of decompression sickness appears during this period, decompress according to Table 1 or 1A. If decompression sickness develops during or after this procedure, deal with it as a recurrence (see page 100).

If the available chamber does not permit pressurization to 100 feet, go to the maximum pressure and remain for 30 minutes. Then follow Table 3 for decompression. If decompression sickness occurs despite this procedure, return to maximum pressure for 30 minutes and follow Table 4 from that point.

Incidence of Decompression Sickness

It is impossible to predict what proportion of exposures in hyperbaric practice will result in decompression sickness when the U.S. Navy tables are employed. The incidence will probably be small, but it may still prove unacceptable when dealing with individuals who may be more susceptible than the divers upon whom Navy tests and experience are based. Further, an incidence that presents no problem in Navy diving may be prohibitive for various reasons among medical personnel.

In a large series of working, open-water dives in which Table 1-5 was employed without addition of safety factors, the incidence of decompression sickness was almost exactly 1 per cent (Lanphier, unpublished data, 1958). With Table 1-9, it would undoubtedly have been several times higher. The main reason for the division between these tables was the great difficulty of deriving practical schedules with very low incidence for exposures in the ranges covered by Table 1-9.

Experience with the research chamber at the University of Buffalo to date has involved nearly 700 exposures including about 250 requiring decompression stops, over 60 of which were on schedules from Table 1-9. A large proportion of the others were at or near "no decompression" limits. Within this experience, there have been no difficulties attributable to the tables. The zero incidence of decompression sickness may suggest that differences between diving and chamber exposure confer considerably greater safety in the latter, as might well be expected.

Whether extra safety factors should be added routinely in hyperbaric applications or not is a question that cannot be answered at this time. Doing so has the disadvantage of postponing the day when a true picture of the decompression problem in hyperbaric practice can be seen.

Breathing oxygen for a significant period during the stops of air-decompression tables confers a considerable margin of safety, but the decompression time cannot be shortened unless special oxygen-decompression tables are used (see page 77).

OTHER DECOMPRESSION PROCEDURES

Use of the U.S. Navy air-decompression tables as discussed above is a simple and logical starting point for decompression in medical hyperbaric work. However, decompression at best will impose serious limitations, prevent optimum utilization of personnel, and in general constitute one of the most troublesome problems of hyperbaric activity. This is nothing new. It has long been the case in diving and all kinds of work under significant pressures of air.

It is not likely that a low incidence of decompression sickness can be maintained in hyperbaric personnel with decompression times any less than those of the U.S. Navy tables if air is used throughout. The most promising approach to reduction of decompression times lies in limiting the uptake of inert gas during exposure, accelerating its elimination during decompression, or a combination of these objectives. The principal means of accomplishing either one is to decrease the amount of inert gas in the breathing medium by increasing the concentration of oxygen. The applicability of this method is limited by the toxicity of oxygen and the inadequacy of our information about tolerable exposures to oxygen under various conditions, but several approaches are available. All of them complicate matters, but in some cases the complications may be justified by the savings of time.

In this section, a number of different procedures will be discussed. Most are of interest from the standpoint of potential savings of decompression time, but a few are valuable for other reasons.

Oxygen Decompression

The elimination of inert gas can be hastened considerably and decompression time potentially shortened accordingly if oxygen is breathed during decompression. Oxygen breathing lowers the alveolar and arterial nitrogen pressures nearly to zero, thus increasing to a maximum the pressure difference or "outward gradient" that causes nitrogen to move from the tissues into the blood and then into the alveoli for elimination in the expired gas. At the same time, the likelihood of bubble formation at a given tissue gas pressure is unaffected or lessened. Critical ratios for bubbling depend upon both tissue gas pressure and ambient pressure, and the relative ambient pressure can be kept the same as during air decompression or maintained at a higher (safer) level. With oxygen breathing, gas elimination does not require progressive lowering of ambient pressure.

Oxygen decompression is part of the routine of current U.S. Navy helium-oxygen diving, but only one table provides schedules for oxygen decompression following exposure to compressed air. This is Table 1-17, which was designed primarily for surface decompression (see page 85).

Direct application of Table 1-17 to normal hyperbaric situations would be extremely cumbersome, so a new table for interim use (Table A) has been derived from Table 1-17 mainly by rearrangement of stops. (Table B, newly developed at the U.S. Navy Experimental Diving Unit, provides data for oxygen decompression after three-and four-hour exposures at 70 feet.) Table A can be applied in essentially the same way as the air-decompression tables, but several special notes apply:

1. Where the exposure time is less than that tabulated, use the appropriate air-decompression table instead. (The saving of time with oxygen in the lower range of time does not warrant the complications.)

2. Upon leaving the pressure of exposure, reduce chamber or lock pressure at the rate of 60 feet per minute (or use a slower rate and include the ascent time in the bottom time as discussed on page 72).

3. Make the stops indicated in the table, breathing oxygen. The rate of ascent between stops is not critical.

4. Upon completing the 30-foot stop, lower the pressure to normal over a period of two minutes, continuing to breathe oxygen.

5. Take note of the following considerations related to the use of oxygen:

(a) Safe decompression with this procedure requires that the individual receive pure oxygen without interruption throughout the stops and between them. Use suitable equipment for oxygen administration. Pay careful attention to mask fit and avoidance of air leaks from any source.

(b) The individual must remain at rest throughout decompression because of the accelerating effect of physical activity upon the onset of oxygen convulsions.

(c) If possible, the individual should have an attendant with him in the chamber or lock during oxygen breathing.

(d) Although the risk of oxygen poisoning should be extremely small when this table is properly used, signs or symptoms that could indicate its onset should be noted and heeded.

6. If oxygen breathing must be discontinued for any reason, decompression must be completed on the air-decompression table that applies to the pressure and duration of exposure. Subtract the time spent on oxygen from the total time of specified stops on the air schedule. Spend the remaining time on air at the appropriate stop depths.

Repetitive dives can be made following oxygen decompression. For the applicable repetitive-group-designation letter, consult the air-decompression schedule (in Table 1-5) for the depth and time of the exposure concerned.

Important Note:
If oxygen is breathed during decompression according to any procedure, inert-gas elimination will unquestionably be hastened; but unless a specific oxygen-decompression table is being followed, no shortening of decompression time can be specified. However, a considerable margin of safety is provided if the duration of oxygen breathing is significant, and this provides a simple means of reducing the likelihood of decompression sickness to the vanishing point. (Breathing oxygen during the brief period of direct ascent from "no decompression" exposures is probably a futile gesture and may as well be omitted.)

Decompression after Oxygen Breathing during Exposure

The minimal decompression needs of a patient who has been breathing oxygen while under pressure have already been discussed. This suggests a seemingly simple and effective means of reducing the decompression needs of

TABLE A.

DECOMPRESSION USING 100 PERCENT OXYGEN
(Derived from Table 1-17, U.S. NAVY Diving Manual)

Depth, ft.	Time, min.	Stop 30 ft. (O_2)	Approx. total time, min.(4)
	(1)		
	120	29	32
70	150	37	40
	180 }		
	240 }	See Table B.	
	(1)		
	100	32	35
80	115	37	40
	130	43	46
	150	50	53
	(1)		
	80	32	35
	90	37	40
90	100	41	44
	110	46	49
	120	50	53
	130	55	58
	(1)		
	70	33	36
	80	39	42
100	90	45	48
	100	51	54
	110	56	59
	120	63	66
	(1)		
	60	34	37
	70	41	44
110	80	49	52
	90	56	59
	100	64	67
	(1)		
	50	32	35
	60	42	45
120	70	51	54
	80	59	62
	90	69	72
	100	83	86

Depth, ft.	Time, min.	Stops(3) 40 ft. (O_2)	Stops(3) 30 ft. (O_2)	Approx. total time, min. (4)
	(1)			
	50		40	43
130	60		50	53
	70		60	63
	80	3	70	76
	90	8	79	90
	(1)			
	35		28	32
	40		34	38
	45		41	45
140	50		47	51
	55		53	57
	60		59	63
	65		66	70
	70	4	72	80
	(1)			
	35		35	39
150	40		45	49
	45		54	58
	50		63	67
	55	3	71	78
	(1)			
	30		31	35
160	35		45	49
	40		58	62
	45	2	68	74
	(1)			
	25		28	32
170	30		41	45
	35		55	59
	40	1	69	74

NOTES

(1) For shorter exposure times, use air decompression tables.

(2) Reduce pressure at 1 ft/sec or use slower rate and include ascent time (to first stop) in exposure time.

(3) Time between stops is not critical.

(4) Ascend from 30 feet to surface in not less than 2 min breathing oxygen.

TABLE B.

AIR EXPOSURE WITH OXYGEN DECOMPRESSION SCHEDULES FOR HYPERBARIC OXYGEN THERAPY

Depth	70 feet
Exposure	180 min.
Ascent	5 min.
30 feet	5 min. 100% oxygen
20 feet	20 min. 100% oxygen
10 feet	25 min. 100% oxygen
Total ascent	58 min.
Repetitive group	Z
Time to first stop is five minutes.	
Time between stops and surfacing from last stop is one minute.	
Depth	70 feet
Exposure	240 min.
Ascent	5 min.
30 feet	10 min. 100% oxygen
20 feet	25 min. 100% oxygen
10 feet	35 min. 100% oxygen
Total ascent	78 min.
Repetitive group	Z
Time to first stop is five minutes.	
Time between stops and surfacing from last stop is one minute.	

From: Workman, R.D. Oxygen decompression following air dives for use in hyperbaric oxygen therapy. (U.S. Navy Experimental Diving Unit Research Report 2-64, 1964.)
Workman, R.D. Standard decompression procedures and their modification in preventing the bends. Ann. N.Y. Acad. Sci., 1964, 117: 834-842, 1965.

chamber personnel. Theoretically, breathing oxygen for a time during exposure to high pressure will not only prevent uptake of inert gas during that period but will also cause elimination of some of the excess gas already present. On this basis, one could not only deduct oxygen time from total exposure time for purposes of decompression, but would be justified in applying the repetitive-dive tables, treating an oxygen period as a surface interval to obtain "credit" for desaturation.

The main obstacle in the way of applying this approach is the allowable period of oxygen exposure for active chamber personnel. At the present time, the only applicable guideline is the U.S. Navy oxygen depth-time limit table for working dives with oxygen as the breathing medium (Table 1-19).
Although this is hopefully more restrictive than necessary for chamber oxygen exposures, we presently have no other criterion. The primary rule is not to use oxygen for any dive deeper than 25 feet, and time limits for deeper exposures are provided only for exceptional circumstances. None of these limits permit oxygen breathing at pressures where it would be of worthwhile benefit in reducing the decompression requirements of hyperbaric work.

Preventing uptake of extra inert gas and eliminating some of the gas already taken up does not actually require breathing pure oxygen. It is sufficient if the nitrogen partial pressure is reduced to that of air at normal pressure. For example, breathing 79/3 = 26 per cent nitrogen would be satisfactory at 3 atm abs. The corresponding oxygen concentration of 74 per cent would yield an oxygen partial pressure equivalent to that of oxygen breathing at slightly more than 40 feet—still too high in terms of the Navy limits. However, the principle is valid for circumstances where it can be applied; and it may prove particularly valuable if it is found possible to use more liberal oxygen exposure limits for chamber personnel. For the present, the most practical approach of this sort is that described in the next section.

TABLE 1-19

OXYGEN DEPTH-TIME LIMITS

(Depth and time limits of exposure when breathing pure oxygen during working dives.)

1. NORMAL OXYGEN LIMITS

DO NOT DIVE DEEPER THAN 25 FEET

Observe these time limits:

Depth (feet)	Time (minutes)
10	240
15	150
20	110
25	75

2. LIMITS FOR EXCEPTIONAL OPERATIONS

Depth (feet)	Time (minutes)
30	45
35	25
40	10

3. EMERGENCY LIMITS

See article 1.5.7, paragraph (7), and figure 1-33.

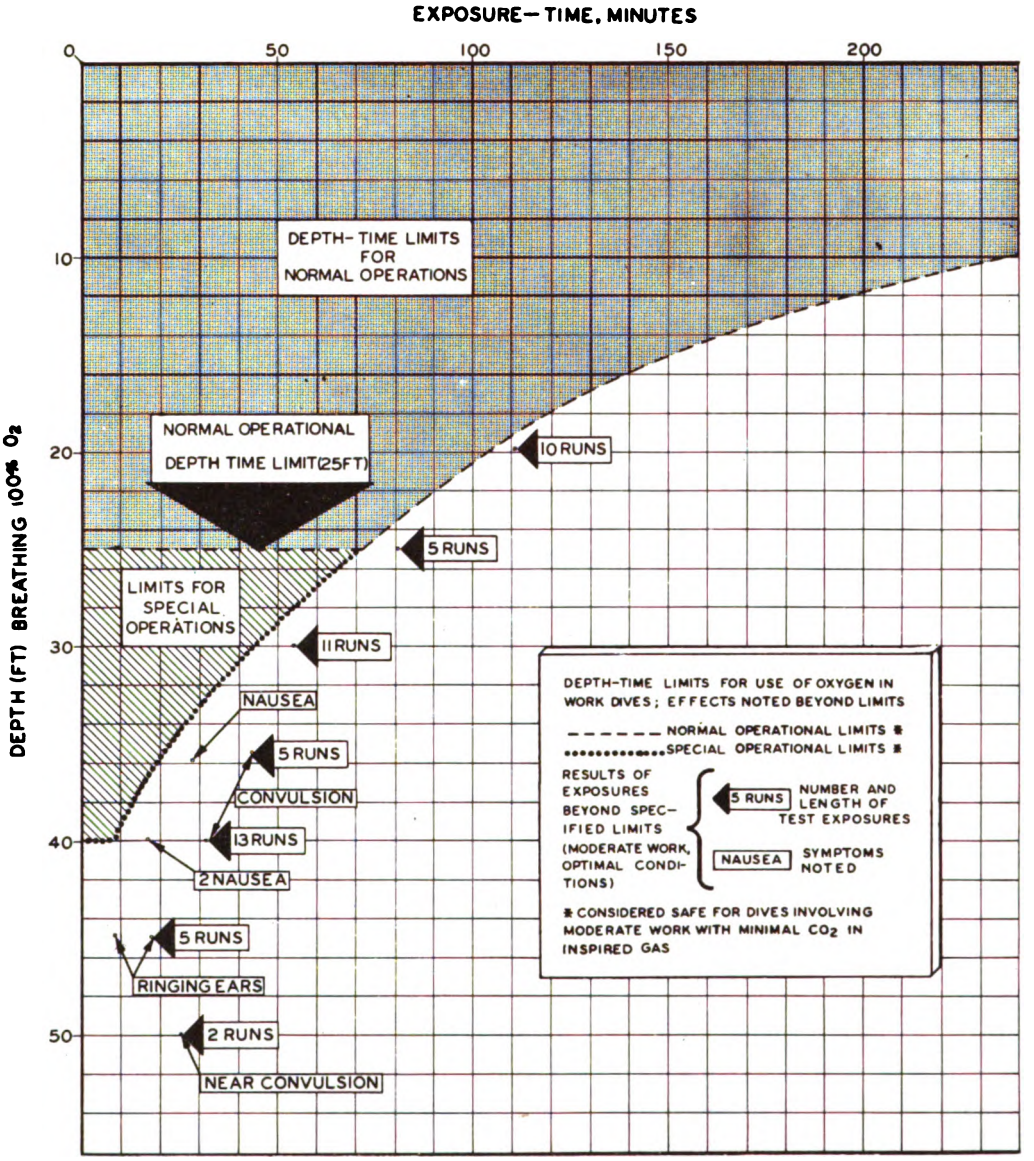

FIGURE 1-33.—Oxygen depth-time limits.

Reproduced from U.S. NAVY DIVING MANUAL

"Equivalent Air" Decompression after Use of Nitrogen-Oxygen Mixtures

Breathing a nitrogen-oxygen mixture that contains more than 21 per cent oxygen is at present the most practical method of reducing the uptake of inert gas during high-pressure exposure. Since the likelihood of decompression sickness is almost directly related to the partial pressure of nitrogen during exposure, keeping this at a lower level than with air permits using an air-decompression schedule for a lesser depth pressure.

The term "equivalent air depth" is often used in this connection. It signifies equating various nitrogen-oxygen mixtures and depths to air exposures on the basis of the nitrogen partial pressures involved. (The term is appropriate, but it invites confusion with the Diving Manual tables of "nitrogen-oxygen equivalent air depth" [Tables 1-10, 3-3, 3-4, 3-5 in Diving Manual]. These refer to the same principle but apply only to a specific type of breathing apparatus that is not likely to be used in medical hyperbaric work.)

Equivalent air depth can be computed according to the following formula (Diving Manual, 3.6.5 [14]):

$$E = \left[\frac{(1 - b) \times (D + 33)}{0.79} \right] - 33$$

where E = equivalent air depth (feet)
D = actual exposure depth (feet)
b = fraction of oxygen in the breathing medium

Example: Breathing a 50 per cent oxygen mixture at 70 feet

$$E = \left[\frac{(1 - 0.5) \times (70 + 33)}{0.79} \right] - 33$$
$$= \left[\frac{(0.5 \times 103)}{0.79} \right] - 33$$
$$= \frac{51.5}{0.79} - 33$$
$$= 65.2 - 33$$
$$= 32.2 \text{ feet}$$

The example itself makes clear the economy of decompression that can be effected by this means. Here, an exposure (3+ atm) that requires increasingly burdensome periods of decompression when air is breathed is reduced to one that should require no decompression stops no matter how long the time under pressure.

However, the problem of oxygen toxicity and the present limits for oxygen exposure must be considered at the same time. This involves the equally simple concept of relative oxygen depth, calculated by the formula

$$R = [b \times (D + 33)] - 33$$

where R = relative oxygen depth (feet)
D = actual exposure depth (feet)
b = fraction of oxygen in breathing medium

Using the same example as before:

$$R = [0.5 \times (70 + 33)] - 33$$
$$= (0.5 \times 103) - 33$$
$$= 51.5 - 33$$
$$= 18.5 \text{ feet}$$

Exposures to oxygen at 18.5 feet have not been known to cause either oxygen convulsions or lung damage, but very long periods at such pressures have not been studied. The limit specified by Table 1-19 is 110 minutes. Even with this restriction, the use of a high-oxygen mixture would permit spending 110 minutes at 70 feet with no subsequent decompression stops while the same exposure to air would have required about 44 minutes of decompression.

A serious drawback of this approach arises in the fact that obligatory extension of the time at 70 feet would require shifting to air for the remainder of the period and, for lack of a better method, decompressing as if the entire period had been spent on air. (The problem is the same as for multilevel exposures as discussed on page 73.) Thus, the discomfort of wearing a mask and the risk of possible oxygen complications during the first part of the exposure would simply have been wasted. If an exposure beyond 110 minutes had been foreseen, a lower oxygen concentration should have been selected.

Tabulation of the equivalent air and relative oxygen depths for a 60 per cent nitrogen - 40 per cent oxygen mixture may be of interest in connection with the applicable oxygen and no-decompression limits.

This table makes it evident that a "60-40" mixture is better for long procedures at 3 atm than a "50-50" mixture. It also has advantages at greater depths although the oxygen limit becomes increasingly restrictive. At 3 atm the no-decompression limit is passed at 100 minutes but the saving of decompression remains large. At the 240-minute oxygen limit, about 48-minutes decompression would be required compared to 179 minutes for a similar exposure to air.

Safe use of high-oxygen mixtures requires that the gas employed be of known and stable concentration and that affected personnel re-

Equivalents and Limits for a 60% N_2 - 40% O_2 Mixture

Actual Pressure		Equivalent Depths, Feet		Time Limits, Minutes	
Atm	Feet	Air	Oxygen	No-Decomp.	O_2
3.0	66	42	6.0	100	240
3.5	82.5	54.6	13.2	60	170
4.0	99	67	19.8	50	110
4.5	115.5	80	26.4	40	60*
5.0	132	92	33.0	25	30*

*From Diving Manual Fig. 1-33.

ceive it without interruption. Commercial gas mixtures must be analyzed by a reliable method prior to use.

Administration by mask is the most likely procedure, and the necessity for avoiding inward leaks of air cannot be emphasized too strongly. Even a small leak could cause the nitrogen partial pressure to be much higher than anticipated with a great increase in the likelihood of decompression sickness if the "equivalent air" schedule for the mixture were followed. In view of this, some systems are totally unsuitable. The best would be one in which a demand valve supplies the gas on inspiration and maintains a slight positive pressure in the mask throughout the respiratory cycle. Continuous gas monitoring at the mask is an alternative.

Maintenance of a high-oxygen chamber atmosphere is an attractive possibility since it would eliminate the need for masks. It is feasible with certain systems, particularly those that maintain the chamber atmosphere by recirculation with carbon dioxide removal and oxygen replenishment or those in which the chamber air is obtained by mixing oxygen and nitrogen from separate sources. Whether keeping the chamber oxygen concentration above 21 per cent can be justified is doubtful in view of the probable increase in fire hazard; but if this approach to the "equivalent air" principle were adopted, the need for precise regulation and monitoring of the chamber gas concentrations is obvious.

The possibility of ill-effects from high-oxygen exposure must be kept in mind even when observing the hopefully conservative Navy limits for working divers. Navy studies indicate that some individuals tend to retain carbon dioxide and become unusually susceptible to oxygen convulsions when exerting themselves during nitrogen-oxygen mixture breathing. Especially in individuals who are exposed repeatedly, the possibility of chronic effects from high oxygen must also be considered until the limits observed are known to be safe from this standpoint.

If long-term effects of repeated subconvulsive exposure can be ruled out, the applicability of high-oxygen mixtures can probably be extended considerably. For example, existing knowledge indicates that the acute toxic effects of oxygen disappear quite rapidly when the oxygen level is returned to a more normal value. This suggests that quite high levels could be tolerated on an intermittent basis and that appropriate schedules could reduce the mean effective nitrogen level of the whole exposure to a very low value.

Decompression after Use of Helium-Oxygen Mixtures

According to information still being copied from one textbook edition to another, the use of helium-oxygen mixtures practically eliminates the problem of decompression. This miracle is predicted on the basis of the low fat solubility and rapid diffusion of helium.

The truth of the matter is that differences between the decompression procedures required for helium and nitrogen exposures are relatively small and by no means always in favor of helium. The use of helium-oxygen mixtures in diving is based upon avoidance of nitrogen narcosis and not upon advantages in decompression. The same will probably be true in medical hyperbaric applications.

Nitrogen narcosis may produce significant impairment of thought and performance in a few individuals even at 3 atm abs. It is certain to be a problem if hyperbaric exposures exceed 4 atm. The problem is compounded by the fact that the individuals most affected may, as with

moderate doses of alcohol, consider themselves to be at the very peak of intellectual efficiency and manipulative dexterity.

U.S. Navy helium-oxygen diving procedures permit using the highest allowable oxygen concentrations during exposure, and oxygen decompression is routine. However, the decompression times for relatively short exposures to relatively low pressures are in general longer, according to the tables currently in use, than for comparable nitrogen-oxygen mixtures using the "equivalent air" approach. At the other end of the exposure-time scale, the current helium-oxygen tables are probably not sufficiently safe. (None of the helium schedules go beyond four hours in any case.)

Until more suitable helium-oxygen decompression tables become available, it is probably best for most hyperbaric procedures to concentrate upon the use of air or nitrogen-oxygen mixtures. (A high oxygen mixture will reduce narcosis to some extent, as illustrated by the fact that a 40 per cent oxygen mixture breathed at 5 atm is equivalent to air at 92 feet.) This does not preclude breathing a helium-oxygen mixture for limited periods when absolute freedom from narcosis is desirable. Such use would seldom require modification of the decompression procedure required by nitrogen-oxygen or air, provided that the period following helium-breathing (before decompression) is of equal duration.

A standard helium-oxygen mixture should be kept available for use in any hyperbaric installation where pressures above 4 atm can be employed, as for recompression in decompression sickness or air embolism. For most purposes, 80 per cent helium—20 per cent oxygen mixture is suitable. The oxygen concentration should not exceed 30 per cent.

Use of "Multiple Inert Gas" Mixtures

An often-suggested method of dealing with the decompression problem is to breathe a mixture that contains two or more different inert gases instead of the single one (nitrogen) that predominates in air. The underlying idea is that in this way one could readily keep any individual gas from reaching a critical tension from the standpoint of bubble formation. It is likely, however, that bubble formation depends mainly upon the total gas tension at a given site. Nevertheless, experimentation to date suggests that a significant decompression advantage can be gained through the "multiple inert gas" approach. The mechanism of such gains remains unclear, and practical tables for application of this principle have not yet been worked out.

The idea is worth pursuing not only because of decompression advantages, but because inclusion of a considerable fraction of helium would largely eliminate nitrogen narcosis as a problem in hyperbaric work even at higher pressures.

Alternation of Inert Gases

Another proposed procedure involving "multiple inert gases " is that of alternating, for example, between a nitrogen-oxygen mixture and a helium-oxygen mixture. In addition to the apparent advantages of using more than one inert gas as discussed above, another possible advantage is suggested by the fact that the first inert gas would have a high "outward gradient" and would be eliminated at a rather rapid rate while the second was being breathed. The relationship would be reversed during the next alternation and so on indefintely. However, the net result in terms of total inert-gas tensions should be no better than with a single gas.

Early experimentation has suggested that "inert-gas alternation" may be even more advantageous than use of "multiple inert-gas mixtures." As in that case, however, the theoretical basis has yet to be established and practical tables remain to be developed.

Oxygen Breathing Prior to Exposure

In aviation medicine, the problem of "altitude bends" can be eliminated by preliminary denitrogenation. This is accomplished by breathing oxygen for a period of hours prior to flight. It is natural to wonder if a similar procedure would not also be worthwhile prior to hyperbaric exposure.

The basic situation is of course different: Altitude bends result from formation of bubbles by nitrogen normally present in the body, and eliminating this solves the problem. In high-pressure exposure, it is the extra inert gas taken up during exposure that causes decompression sickness. Removal of some of the normally-present nitrogen still makes sense from the standpoint of reducing the total amount present at the end of exposure, but the potential benefits are not as great as might be supposed.

The uptake of inert gas depends not only upon the duration of exposure and the time constants of the tissues but also upon the

"inward gradient" of the gases concerned. This in turn depends not only upon the pressure of exposure to inert gas but upon the gas pressure in the tissues. If this has been lowered by denitrogenation, the "inward gradient" and the rate of gas uptake will be increased correspondingly and will in part offset the benefits.

In a hyperbaric exposure of reasonable duration, the tissues that saturate and desaturate rather rapidly will approach complete saturation whether denitrogenated initially or not. The slower tissues, which are mainly responsible for prolonged decompression times, will have usefully lowered nitrogen pressures only if the period of preliminary oxygen breathing has been lengthy.

In general, time is more efficiently spent in decompression following exposure than in oxygen breathing beforehand. There are no tables that permit taking advantage of preliminary oxygen breathing, and developmental efforts should probably be expended first in more promising directions.

Decompression in "Saturation Exposures"

If a hyperbaric chamber is to be operated on a 24 hour basis at more than 2 atm for indefinite periods, the decompression of personnel will inevitably be a very serious problem. One solution would be that now being explored for prolonged work at depth in diving.

This takes advantage of the fact that within about 24 hours at a given pressure, the body's saturation with inert gas reaches a new equilibrium. No significant further uptake of gas will take place no matter how long the exposure is continued. Thus, a maximum requirement for decompression is reached; and "ascent" will require no longer after a week or more of exposure than after about a day. The decompression time will inevitably be long, but the total man-hours spent in decompression could be vastly less than for repeated shorter exposures.

The feasibility of using this approach depends of course upon the doubtful willingness of personnel to live in the chamber for a matter of days, and a minimum of two men in the chamber at a time would be required to cover each 24-hour period. If suitable sleeping quarters were provided and enough compensatory free time allowed between exposures, this pattern of life would compare favorably at least with submarine duty.

Avoidance of chronic oxygen effects would require keeping the partial pressure of oxygen in the chamber at levels close to normal. The P_{O_2} of 21 per cent oxygen at 3 atm, for example, would probably not be satisfactory. If the need for a lower P_{O_2} meant that personnel had to wear masks for the entire period, the whole concept would become ludicrous. However, maintaining the chamber atmosphere below 21 per cent oxygen is by no means impossible. This would increase the "effective air depth" for anyone exposed, but it would have the advantage of reducing the fire hazard. It is also possible that keeping the "crew sleeping quarters" at a normal oxygen pressure would permit safe exposure to air during working hours.

Surface Decompression

If the tissue pressures of dissolved inert gas are not beyond certain critical limits at the time, the decompression process can be interrupted by a brief period at normal pressure and continued thereafter. In diving, this procedure is known as surface decompression because it permits a diver to minimize or eliminate decompression stops underwater and spend most of his decompression time in a chamber at the surface instead. A similar process known as decanting is used in caisson and tunnel work.

In hyperbaric practice, a few situations can be anticipated in which the principle of surface decompression might be applicable. These include an emergency with unavoidable return to normal pressure before decompression is completed, the need to transfer an individual from one chamber to another for decompression, or having to bring a lock to normal pressure for transfer of personnel or equipment while it was in use for decompression.

The U.S. Navy Diving Manual provides two tables for surface decompression following exposure to compressed air: Table 1-17 and Table 1-18. Table 1-17 involves oxygen breathing, which considerably reduces the time required. Table 1-18 uses air throughout and therefore requires more time, but it provides a more extensive range of exposure pressures and times. It is basically the same as Table 1-5 but with time added to compensate for the surfacing process.

Table 1-17 carries instructions that can readily be translated from diving to hyperbaric use if the "water stops" are interpreted as chamber stops made prior to reaching normal pressure. With that in mind, Table 1-18 is largely self-explanatory and is used basically in the same way as the other air-decompression tables.

1** Depth in feet	2** Time	3** Time (min.) at water stops breathing air at 60'	50'	40'	30'	4**	5** Time (min.) at 40' chamber stop oxygen	6**	7** Approximate total decompression time (min.)
70	52	0	0	0	0	SURFACE INTERVAL NOT TO EXCEED 5 MINUTES	0	2 MINUTE ASCENT FROM 40 FEET IN CHAMBER TO SURFACE WHILE BREATHING OXYGEN	3
	90	0	0	0	0		15		24
	*120	0	0	0	0		23		32
	150	0	0	0	0		31		40
	180	0	0	0	0		39		48
80	40	0	0	0	0		0		3
	70	0	0	0	0		14		23
	85	0	0	0	0		20		29
	100	0	0	0	0		26		35
	*115	0	0	0	0		31		40
	130	0	0	0	0		37		46
	150	0	0	0	0		44		53
90	32	0	0	0	0		0		4
	60	0	0	0	0		14		24
	70	0	0	0	0		20		30
	80	0	0	0	0		25		35
	*90	0	0	0	0		30		40
	100	0	0	0	0		34		44
	110	0	0	0	0		39		49
	120	0	0	0	0		43		53
	130	0	0	0	0		48		58
100	26	0	0	0	0		0		4
	50	0	0	0	0		14		24
	60	0	0	0	0		20		30
	70	0	0	0	0		26		36
	*80	0	0	0	0		32		42
	90	0	0	0	0		38		48
	100	0	0	0	0		44		54
	110	0	0	0	0		49		59
	120	0	0	0	3		53		66
110	22	0	0	0	0		0		5
	40	0	0	0	0		12		23
	50	0	0	0	0		19		30
	60	0	0	0	0		26		37
	*70	0	0	0	0		33		44
	80	0	0	0	1		40		52
	90	0	0	0	2		46		59
	100	0	0	0	5		51		67
	110	0	0	0	12		54		77
120	18	0	0	0	0		0		5
	30	0	0	0	0		9		20
	40	0	0	0	0		16		27
	50	0	0	0	0		24		35
	*60	0	0	0	2		32		45
120	70	0	0	0	4	SURFACE INTERVAL NOT TO EXCEED 5 MINUTES	39	2 MINUTE ASCENT FROM 40 FEET IN CHAMBER TO SURFACE WHILE BREATHING OXYGEN	54
	80	0	0	0	5		46		62
	90	0	0	3	7		51		72
	100	0	0	6	15		54		86
130	15	0	0	0	0		0		5
	30	0	0	0	0		12		23
	40	0	0	0	0		21		32
	50	0	0	0	3		29		43
	*60	0	0	0	5		37		53
	70	0	0	0	7		45		63
	80	0	0	6	7		51		76
	90	0	0	10	12		56		90
140	13	0	0	0	0		0		6
	25	0	0	0	0		11		23
	30	0	0	0	0		15		27
	35	0	0	0	0		20		32
	40	0	0	0	2		24		38
	45	0	0	0	4		29		45
	50	0	0	0	6		33		51
	*55	0	0	0	7		38		57
	60	0	0	0	8		43		63
	65	0	0	3	7		48		70
	70	0	2	7	7		51		80
150	11	0	0	0	0		0		6
	25	0	0	0	0		13		25
	30	0	0	0	0		18		30
	35	0	0	0	4		23		39
	40	0	0	3	6		27		49
	45	0	0	5	7		33		58
	*50	0	2	5	8		38		66
	55	2	5	9	4		44		78
160	9	0	0	0	0		0		7
	20	0	0	0	0		11		24
	25	0	0	0	0		16		29
	30	0	0	0	2		21		38
	35	0	0	4	6		26		49
	40	0	3	5	8		32		62
	*45	3	4	8	6		38		73
170	7	0	0	0	0		0		7
	20	0	0	0	0		13		26
	25	0	0	0	0		19		32
	30	0	0	3	5		23		44
	35	0	4	4	7		29		58
	*40	4	4	8	6		36		73

*These are the optimum exposure times for each depth which represent the best balance between length of work period, safety and amount of useful work for the average diver. Exposure beyond these times is permitted only under special conditions.

**Notes on columns.

Column 1. Depth—In feet, gage.

Column 2. Time—Interval from leaving the surface to leaving the bottom.

Column 3. Water stops—Time spent at tabulated stops using air. If no water stops are required use a 25 foot per minute rate of ascent to the surface. When water stops are required use a 25 foot per minute rate of ascent to first stop. Take an additional minute between stops. Use one minute for the ascent from 30 feet to the surface.

Column 4. Surface interval—The surface interval shall not exceed 5 minutes and is composed of the following elements:

(a) Time of ascent from the 30-foot water stop to the surface (1 minute).

(b) Time on surface for landing the diver on deck and undressing (not to exceed 3½ minutes).

(c) Time of descent in the recompression chamber from the surface to 40 feet (about ½ minute).

Column 5. During the period while breathing oxygen the chamber shall be ventilated.

Column 6. Surfacing—Oxygen breathing during this 2-minute period shall follow the period of oxygen breathing tabulated in Column 5 without interruption.

Column 7. Total decompression time—This includes

(a) Time of ascent from bottom to first stop at 25 feet per minute.

(b) Sum of tabulated water stops, column 2.

(c) One minute between water stops.

(d) Surface interval.

(e) Time at 40 feet in recompression chamber, column 4.

(f) Time of ascent, an additional 2 minutes, from 40 feet in the recompression chamber to the surface, column 5.

The Approximate Total Decompression Time may be shortened only by decreasing the time required to undress the diver on deck.

TABLE 1-17.—*Surface decompression table using oxygen.*

DEPTH (ft.)	BOTTOM TIME (Min.)	TIME TO FIRST STOP	TIME AT WATER STOPS 30	20	10	CHAMBER STOPS (AIR) 30	20	10	TOTAL ASCENT TIME
40	250	0.5			3			7	10.5
	260	0.5			3			11	14.5
	270	0.5			3			15	18.5
	300	0.5			3			19	22.5
50	120	0.7			3			5	8.7
	140	0.7			3			10	13.7
	160	0.7			3			21	24.7
	180	0.7			3			29	32.7
	200	0.7			3			35	38.7
	220	0.7			3			40	43.7
	240	0.7			3			47	50.7
60	80	0.8			3			7	10.8
	100	0.8			3			14	17.8
	120	0.8			3			26	29.8
	140	0.8			3			39	42.8
	160	0.8			3			48	51.8
	180	0.8			3			56	59.8
	200	0.7		3			3	69	75.7
70	60	1.0			3			8	12.0
	70	1.0			3			14	18.0
	80	1.0			3			18	22.0
	90	1.0			3			23	27.0
	100	1.0			3			33	37.0
	110	0.8		3			3	41	47.8
	120	0.8		3			4	47	54.8
	130	0.8		3			6	52	61.8
	140	0.8		3			8	56	67.8
	150	0.8		3			9	61	73.8
	160	0.8		3			13	72	88.8
	170	0.8		3			19	79	101.8
80	50	1.2			3			10	14.2
	60	1.2			3			17	21.2
	70	1.2			3			23	27.2
	80	1.0		3			3	31	36.0
	90	1.0		3			7	39	50.0
	100	1.0		3			11	46	61.0
	110	1.0		3			13	53	70.0
	120	1.0		3			17	56	77.0
	130	1.0		3			19	63	86.0
	140	1.0		26			26	69	122.0
	150	1.0		32			32	77	142.0
90	40	1.3			3			7	11.3
	50	1.3			3			18	22.3
	60	1.3			3			25	29.3
	70	1.2		3			7	30	41.2
	80	1.2		13			13	40	67.2
	90	1.2		18			18	48	86.2
	100	1.2		21			21	54	97.2
	110	1.2		24			24	61	110.2
	120	1.2		32			32	68	133.2
	130	1.0	5	36			36	74	152.0
100	40	1.5			3			15	19.5
	50	1.3		3			3	24	31.3
	60	1.3		3			9	28	41.3
	70	1.3		3			17	39	60.3
	80	1.3		23			23	48	96.3
	90	1.2	3	23			23	57	107.2
	100	1.2	7	23			23	66	120.2
	110	1.2	10	34			34	72	151.2
	120	1.2	12	41			41	78	173.2
110	30	1.7			3			7	11.7
	40	1.5		3			3	21	28.5
	50	1.5		3			8	26	38.5
	60	1.5		18			18	36	73.5
	70	1.5	1	23			23	48	96.5
	80	1.3	7	23			23	57	111.3
	90	1.3	12	30			30	64	137.3
	100	1.3	15	37			37	72	162.3

DEPTH (ft.)	BOTTOM TIME (Min.)	TIME TO FIRST STOP	TIME AT WATER STOPS 50	40	30	20	10	CHAMBER STOPS (AIR) 30	20	10	TOTAL ASCENT TIME
120	25	1.8					3			6	10.8
	30	1.8					3			14	18.8
	40	1.7				3			5	28	34.7
	50	1.7				15			15	31	52.7
	60	1.5			2	22			22	45	92.5
	70	1.5			9	23			23	55	111.5
	80	1.5			15	27			27	63	133.5
	90	1.5			19	37			37	74	168.5
	100	1.5			23	45			45	80	194.5
130	25	2.0					3			10	15.0
	30	1.8				3			3	18	26.8
	40	1.8				10			10	25	46.8
	50	1.7			3	21			21	37	83.7
	60	1.7			9	23			23	52	108.7
	70	1.7			16	24			24	61	126.7
	80	1.5		3	19	35			35	72	165.5
	90	1.5		8	19	45			45	80	198.5
140	20	2.2					3			6	11.2
	25	2.0				3			3	14	22.0
	30	2.0				5			5	21	33.0
	40	1.8			2	16			16	26	61.8
	50	1.8			6	24			24	44	99.8
	60	1.8			16	23			23	56	119.8
	70	1.7		4	19	32			32	68	156.7
	80	1.7		10	23	41			41	79	195.7
150	20	2.2				3			3	7	15.2
	25	2.2				4			4	17	27.2
	30	2.2				5			5	24	42.2
	40	2.0			5	19			19	33	76.0
	50	2.0			12	23			23	51	111.0
	60	1.8		3	19	26			26	62	137.6
	70	1.8		11	19	39			39	75	184.8
	80	1.7	1	17	19	50			50	84	222.7
160	20	2.3				3			3	11	19.3
	25	2.3				7			7	20	36.3
	30	2.2			2	11			11	25	51.2
	40	2.2			7	23			23	39	94.2
	50	2.0		2	16	23			23	55	121.0
	60	2.0		9	19	33			33	69	165.0
	70	1.8	1	17	22	44			44	80	209.8
170	15	2.5				3			3	5	13.5
	20	2.5				4			4	15	25.5
	25	2.3			2	7			7	23	41.3
	30	2.3			4	13			13	26	58.3
	40	2.2		1	10	23			23	45	104.2
	50	2.2		5	18	23			23	61	132.2
	60	2.0	2	15	22	37			37	74	187.0
	70	2.0	5	17	19	51			51	86	234.0
180	15	2.7				3			3	6	14.7
	20	2.5			1	5			5	17	30.5
	25	2.5			3	10			10	24	49.5
	30	2.5			6	17			17	27	69.5
	40	2.3		3	14	23			23	50	115.3
	50	2.2	2	9	19	30			30	65	155.2
	60	2.2	5	16	19	44			44	81	211.2
190	15	2.8				4			4	7	17.6
	20	2.7			2	6			6	20	36.7
	25	2.7			5	11			11	25	54.7
	30	2.5		1	8	19			19	32	81.5
	40	2.5		5	14	23			23	55	125.5
	50	2.3	4	13	22	33			33	72	179.3
	60	2.3	10	17	19	50			50	84	232.3

TIME ON SURFACE NOT TO EXCEED 5 MINUTES

TIME ON SURFACE NOT TO EXCEED 3.5 MINUTES

TABLE 1-18.—*Surface decompression table using air.*

Reproduced from U.S. NAVY DIVING MANUAL

Important precautions in the use of surface decompression procedures include:

1. Do not attempt to apply surface decompression unless the "water stops" have been, or can be, made properly if required. (This makes the procedure unsuitable for emergency interruptions of decompression in many situations. See "Omitted Decompression," page no. 73.)

2. Be sure that the allowed "surface interval" or "time on surface" is not exceeded. Note that the five-minute surface interval of Table 1-17 includes ascent from the last water stop and the return to pressure and that only 3.5 minutes are allowed for "landing diver on deck and undressing."

3. In using Table 1-17, take every precaution to assure that pure oxygen is administered; that the breathing system is suitable and functioning properly; that the mask fits properly and is kept on; that there are no sources of air leakage.

4. Note the specified rates of ascent to the first water stop: 25 feet per minute in Table 1-17; 60 feet per minute for Table 1-18. If slower rates are used, as is desirable if patients are involved, add the actual ascent time to the bottom time.

5. Note that either table can be applied following use of nitrogen-oxygen mixtures other than air if the nitrogen exposure is expressed as "equivalent air depth" and the table entered with this value (see page no. 82). The procedure for surface decompression after helium-oxygen exposure is different (see Diving Manual, 1.5.5 (16), page 125).

USE OF "DECOMPRESSION METERS"

The comings and goings of a number of people engaged in a complex hyperbaric procedure can add up to a very formidable problem in assuring that the correct decompression procedure is applied to each one. Repetitive and multilevel exposures complicate matters greatly. The same kind of problems arise in diving operations, and a promising solution is being developed in that connection: individual "decompression computers."

The basic principle of such a device is the use of "tissue analogs" whose response to pressure changes corresponds to the gain and loss of inert gas in body tissues. These analogs are utilized in such a way that the appropriate decompression is obtained by following an indicator.

Decompression computers for hyperbaric work could be of any practical size and would not have to be taken into the chamber if each was kept informed of the pressure to which its man was being exposed. The design should also permit input of the partial pressure of inert-gas exposure so that use of oxygen or mixtures could be taken into account. The computers could even be linked to automatic chamber controls in such a way that they would govern the lowering of chamber or lock pressure on the basis of the occupant's needs for decompression. A more remote possibility is a similar approach to the control of oxygen exposure for avoidance of oxygen poisoning.

In all such connections, however, it is well to remember that too great dependence upon automatic devices can sometimes have more devastating consequences than an occasional human failure.

SUMMARY

At the present time, the U.S. Navy air-decompression tables appear to provide the most satisfactory basis for decompression in medical hyperbaric work. The main drawback is the large amount of decompression time required for many exposures, but it is very doubtful that any significant reduction of time is possible if an acceptable incidence of decompression sickness is to be maintained in exposures where air is the breathing medium throughout.

Practical methods of reducing decompression time are those in which the uptake of inert gas is limited during exposure or its elimination hastened during decompression. Both objectives require the use of oxygen or high-oxygen mixtures, and the problem of oxygen poisoning limits the extent to which this can be done safely. The use of helium-oxygen mixtures appears useful, as in diving, for elimination of inert-gas narcosis rather than shortening of decompression. Procedures involving use of more than one inert gas show promise but are not yet practical. Use of saturation exposures is a possible approach to the problem of decompression in 24-hour chamber operation.

Surface decompression has limited applicability to hyperbaric work, but familiarity with the procedure is worthwhile. Decompression computers may prove of great value in solving some of the problems of decompression particularly in complex exposures.

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Careful review and criticism of this section by Captain R.D. Workman, MC, USN, was extremely helpful and resulted in numerous beneficial changes.

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Chapter VI

DECOMPRESSION SICKNESS

Edward H. Lanphier

Decompression sickness usually takes the form of localized pain; but other manifestations are possible, and some of these can be extremely serious in nature. Keeping exposures to pressure within "no decompression limits" or employing standard decompression procedures will largely prevent decompression sickness, but a few cases are inevitable whenever large numbers of exposures to pressures above 2 atm abs take place. Hyperbaric facilities can thus expect decompression sickness to arise occasionally from their own work, and some facilities will also be called upon to deal with divers or compressed-air workers brought in with evidence of the condition. In any event, it is essential to be able to recognize the condition promptly and treat it adequately.

NOMENCLATURE

Long-standing synonyms of decompression sickness include variations of caisson disease and compressed-air illness. Confusing additions from aviation medicine include aeroembolism (readily mistaken for air embolism, a different entity) and dysbarism (a basket term for ill effects of altered pressures).

Terms that originated with divers or compressed-air workers themselves include the bends (strictly, cases in which localized pain predominates; but a welcome substitute for the longer terms), the staggers (disturbance of equilibrium), and the chokes (characteristic respiratory distress). Niggles (British) and inkles (U.S.) are useful terms for mild, ill-defined, inconstant, or poorly localized symptoms that may either be transient or premonitory.

Medical hyperbarists would do well to accept the better of these terms and avoid useless additions to the lexicon.

PREDISPOSING FACTORS

When decompression sickness develops after appropriate decompression, the cause is often obscure. However, it is certain that the likelihood of bends is increased by any factor that causes unusual uptake of gas during exposure or poor elimination during decompression, or that otherwise makes bubble formation more likely.

The role of the individual's physical condition is discussed in the chapter on Personnel Selection. A less obvious individual factor is the unexplained tolerance to decompression that develops with frequent repeated exposure (1). In caisson and tunnel work, it is probably this factor that permits otherwise inadequate decompression schedules to be employed with a relatively low incidence of bends; and a marked increase in incidence accompanies introduction of new workers or any significant break in the continuity of the work. It is not likely that the exposures of hyperbaric personnel will ever permit them to be decompressed safely on such schedules, but the phenomenon of development and loss of tolerance should be kept in mind.

Trauma before or during exposure, unusual muscular exertion or strain, cramped positions, and extremes of temperature are accepted predisposing factors. Any cause of circulatory impairment present during decompression, especially if not present at working pressure, can be expected to cause difficulty—as can readily be demonstrated with a tourniquet. "Skin bends" are very frequent when the temperature is high during exposure and low during decompression. This probably results from rapid cutaneous nitrogen uptake with vasodilitation and slow elimination with vasoconstriction. Any medical or surgical procedure that might have comparable effects should be approached with caution.

Exercise during or soon after decompression hastens elimination of nitrogen from some tissues, but any benefit is more than offset by promotion of bubble formation. The same is true of exposure to altitude, which is best avoided for at least 12 hours following decompression.

MECHANISMS

There is general agreement that decompression sickness results from bubble formation. Whether the bubbles are usually in the bloodstream or in the tissues is not settled and is seldom of practical significance. Another controversial issue is whether bubbles often or always form even in the absence of symptoms. Bubbles in many parts of the body could be "silent," and it is a matter of real concern to know whether cumulative damage or late effects may occur without being signaled at the time of insult. There is some reason to believe that this may be the case in aseptic bone necrosis. The possibility at least provides good reason for avoiding dubious decompression procedures even when these seem free of harmful effects.

Formation of Bubbles

Any carbonated beverage provides convincing demonstration that bubbles form upon reduction of pressure in a fluid that contains gas dissolved in it at higher pressure. However, the beverage also shows clearly that much gas remains dissolved in a state of supersaturation for a long time after the pressure is reduced and formation of bubbles begins. Such supersaturation must also exist in the body, but its extent is not known. We can say only that symptomatic bubble formation is extremely rare if the pressure of dissolved gas never exceeds the ambient pressure by more than a certain margin. Such a margin is illustrated by the fact that a man can be fully saturated with dissolved gas at 2 atm abs and then return directly to 1 atm with negligible likelihood of ill effects. However, the simple "2:1 ratio" relationship of this example does not apply to all tissues under all circumstances.

Once a bubble has formed, its fate depends upon the direction of gas diffusion through its surface. Under normal circumstances, any deposit of gas in the body will be absorbed because the total pressure of the gases within it is somewhat above the total tension of dissolved gas in its surroundings. As a result, gas continues to diffuse outward until the bubble disappears.

The situation is quite different if a bubble is surrounded by tissues containing dissolved gas at a pressure well above the existing ambient pressure. Now, gas diffuses into the bubble causing it to grow. How long it will grow and how large it will become depend largely upon the subsequent rate of fall of gas pressure in its surroundings. In a tissue that desaturates rapidly, bubbles could form initially but then begin to disappear having caused only transitory symptoms or none. This may explain the fact that "faster" tissues can be decompressed safely with ratios above 2:1, and it may account for "inkles" that do not materialize into symptoms requiring treatment.

If a bubble persists and grows sufficiently to produce symptoms, a very long time may elapse before it will begin to diminish in size spontaneously; and it may produce considerable discomfort and damage in the meantime. The need at this point is to reduce the bubble to harmless size and also cause it to be absorbed entirely. How this can be accomplished is discussed under recompression.

Manifestations

Experience with decompression sickness to date has been derived almost entirely from pressure exposure in either diving or caisson and tunnel work. Although the basic manifestations and their order of frequency remain essentially similar, many details appear to be modified by the nature of exposure and very likely by the decompression procedure employed. Even greater variations may appear in hyperbaric experience, especially since heavy exertion will be largely absent. This is prominent in the older forms of exposure and seems to be important in affecting the outcome. The following summary is based in large part upon three recent large series: Rivera, 935 cases in divers treated by the U.S. Navy (2); Slark, 137 Royal Navy diver cases (3); and Golding, et al., 685 cases from the Dartford Tunnel (1). An unpublished study of 539 earlier U.S. Navy cases (H.W. Gillen, personal communication) agrees in most essential respects with the findings of Rivera (2).

Major acute manifestations fall into two main categories:

1. Localized pain (bends)

   Present in over 90 per cent of cases; sole symptom in 95 per cent of tunnel-worker cases; accompanied by other symptoms in about one third of diver cases.

   Confined to legs in 85 per cent of tunnel cases.

   Upper-limb pain predominant in diver cases; pain elsewhere than in extremities (15-20 per cent) more frequent in divers

than in tunnel workers.

2. Neurological, pulmonary or circulatory effects.

Frequency highly dependent upon type of exposure; much more frequent among divers.

Neurological symptoms predominate and are extremely variable. Vertigo frequent in tunnel workers, muscular weakness and sensory defects most common in divers. (Other comparative studies indicate that paraplegia is much more frequent in compressed-air workers than in divers; that neurological manifestations in divers more commonly involve alterations in consciousness, brain stem or cortical symptoms—Gillen.)

Chokes present in 2 per cent of Rivera's divers; not mentioned in Slark's. Less than 1 per cent in tunnel workers.

Signs of shock noted in nearly half of tunnel cases with symptoms beside pain; reported in about 6 per cent of R.N. diver cases; not tabulated by Rivera. Probably too often overlooked (3).

Minor manifestations, not usually treated, include a variety of skin reactions such as itching, formication, rashes, mottling ("marbling"). Milder forms are frequent, often not reported, seldom significant, probably often related to skin temperature changes rather than general state of gas saturation. However, marbling is very often associated with symptoms requiring treatment (1). Unusual fatigue, far out of proportion to associated exertion, is common following dives; but its cause and relationship to decompression sickness are not certain.

Late or chronic effects are largely confined to:

1. Aseptic bone necrosis (4,5,6,7,8).
   Common among caisson and tunnel workers; not seen in naval divers.
   Seldom detected in less than a year following exposure; relationship to acute episodes often unclear.
   Usually asymptomatic except where undermining of an articular surface, as in head of femur, produces marked pain and disability.
2. Lasting neurological defects. Generally the result of lack, delay, or inadequacy of treatment in acute central-nervous-system involvement.
   Paraplegia due to spinal cord damage is most common.

Time of Onset

The latent period between decompression and onset of acute signs and symptoms is extremely variable and appears to depend not only upon the form of exposure and decompression but upon the type of symptoms impending. In general, very early onset—especially if before completion of decompression—signifies a serious problem regardless of the nature of initial symptoms.

The Dartford Tunnel experience is tabulated in terms of the type of symptoms:

Localized pain (650 cases):
- Average time of onset—3 hours
- Range—during decompression to 12 hours

Neurological, etc. (35 cases):
- Average time of onset—40 minutes
- Range—during decompression to 6 hours
- During or immediately after decompression—17 per cent
- Within one hour—91 per cent

Rivera's large U.S. Navy series (2) shows the following cumulative percentages of onset with time:

During decompression	9.5 per cent
Within 1 hour	57.2
1-2 hours	69.8
3-6 hours	90.2
7-12 hours	97.2
13-24 hours	99.7
24-36 hours	(1 case)

Slark's series (3) is tabulated differently but shows much the same distribution. The patient with latest onset developed symptoms between the 16th and 18th hours.

Although over half of cases will have onset of symptoms within the first 30 to 60 minutes according to naval experience, the three-hour mean of the tunnel bends cases is of interest. The possibility of long delays, even though their frequency is small, is important. Clearly, the time of onset is not of great help in establishing or ruling out the diagnosis of decompression sickness.

DIAGNOSIS

Making the diagnosis of decompression sickness may appear to be a simple matter. It can be so if rather characteristic findings develop not long after exposure and if the picture is not confused by other possibilities. In practice, such straight-forward situations sometimes seem to be the exception rather than the rule.

History

Obviously, a person who has not been decompressed cannot have decompression sickness. Symptoms that clearly had their onset before or during exposure are thus unlikely to represent bends unless decompression produced some notable change in symptomatology. Trauma, or even muscular strain, during exposure can confuse matters greatly. In such cases, pain may result from the injury itself or may represent bends that developed in the affected part as an indirect result of injury.

A certain exposure to pressure is required to produce decompression sickness; but even if the exposure did not exceed 2 atm abs, the possibility cannot totally be ruled out. Civilian divers are noted for underestimation of the depth and time of their exposures and often totally overlook the cumulative effects of repetitive dives. In this sense also, the history may be of relatively little help.

Severe central-nervous-system symptoms sometimes represent air embolism rather than bends (see page 116). This is particularly likely if the exposure was well within safe limits relative to decompression. Since air embolism also requires prompt recompression, the differential diagnosis does not modify the treatment. It is extremely important to note that central-nervous-system effects requiring recompression can seldom if ever be ruled out on the basis of a seemingly harmless exposure.

Signs and Symptoms

An individual with bends pain in an extremity will generally state that it is located in or near a joint, that is seems deep, and (if he has had bends before) that the sensation is typical. Unfortunately, the characteristic pain is not very readily described. It may begin as a dull ache; it seldom is characterized as a sharp pain. "Like something boring into bone" is reasonably apt. The intensity can range from an ill-defined inkle to real agony. Examination usually reveals next to nothing. Pain is often not influenced by motion, and even local tender ness is often absent. Color changes and edema are seldom seen except where trauma has confused the matter. Frequently, the intensity of pain will continue to increase until treatment is provided. It may be relieved temporarily by rubbing or hot applications or appear to respond to analgesics, but such relief is seldom complete or lasting.

Abdominal pain is infrequent, but it does occur and can present a noteworthy problem in differential diagnosis. The extent and duration of soreness that can follow delayed or omitted treatment indicate that decisive management is in order, but few useful guidelines can be provided. Not even dramatic relief of pain upon recompression is necessarily conclusive in these cases since "false relief" may result from compression of gas in a distended structure.

A cutting or stabbing pain in the lower thorax or girdle region is often followed by development of paraplegic spinal-cord involvement.

The findings in neurological manifestations of decompression sickness may cover almost the entire range of conceivable nervous-system defects, up to and including coma, convulsions, and death. It is particularly important to discover any signs of neurological abnormality that may be present in cases dominated by pain. It is extremely worthwhile, when dealing with a relatively small population of people who are being exposed to pressure, to have good records of previous careful examination so that signs normal to the individual will not be a source of confusion. During examination, such possible abnormalities as aphasias and personality changes should not be overlooked.

The symptom complex descriptively known as chokes is rare but has serious implications. It probably represents progressive embolization of the pulmonary capillaries by gas bubbles. An early symptom is substernal distress aggravated by deep inspiration (Behnke's sign) or by inhalation of tobacco smoke, often causing paroxysms of coughing. Onset may occur some hours after decompression. Although uncommon and sometimes transient, such symptoms must be viewed as the probable forerunner of serious difficulties. No individual showing them should be allowed to leave the immediate vicinity of the chamber since recompression may be required on very short notice. If the condition progresses and is not treated, marked dyspnea with rapid, shallow respiration and cyanosis may develop; and loss of consciousness, circulatory collapse, and death may follow.

Development of shock in decompression sickness may follow asphyxia or reduced cardiac output in chokes, or local circulatory obstruction due to massive bubble formation in the periphery. In some cases, bends pain may be sufficient to produce syncopal reactions.

A small proportion of individuals with altitude bends fail to be relieved by return to normal pressure or develop serious delayed reactions. In these, circulatory collapse may dominate the picture. It is now generally recognized that such patients require recompression as urgently as those injured by exposure to high pressure (9).

DECISIONS

Absolute certainty that decompression sickness does or does not exist may be impossible to achieve, and the diagnosis may be made only on the basis of the response to recompression. It is always worth remembering that there is much more to be lost by failing to give prompt and adequate treatment by recompression than by providing it needlessly or using longer tables than are actually required. As the U.S. Navy Diving Manual summarizes the situation, "When in doubt, treat by recompression," and "Remember that time and air are much cheaper than joints and brain tissue."

When neurological difficulties are evident, no delay in treatment whatever can be countenanced. When the symptom is a questionable pain not causing severe discomfort, there is no objection to an hour or so of observation at normal pressure with the individual breathing oxygen. Occasionally, even a true bends pain will regress and disappear spontaneously or with the help of oxygen administration. Narcotics should be used very sparingly if at all since they can seriously obscure the picture. It is reprehensible to suggest that the patient try a hot bath and some aspirin (or a stiff drink) and go to bed in the hope that pain will be tolerated until it subsides and that treatment can thus be avoided. If an individual with probable bends needs any form of relief, he deserves recompression. This will remain true until we are certain that bone necrosis does not have its origin in neglect of milder cases.

SUMMARY

Decompression sickness represents symptomatic bubble formation from inert gas taken up during exposure and inadequately eliminated during decompression. A variety of predisposing factors may result in its occurrence even when good decompression procedures have been followed. The most frequent and least serious manifestation is localized pain (bends), present in over 90 per cent of cases and the sole symptom in the majority. More serious symptoms involve the nervous system, the lungs, or the circulation. Neurological symptoms are the most frequent of these and are extremely variable in nature. Symptoms develop in 50 per cent of cases within three hours or less, and less than 10 per cent are likely to have their onset delayed beyond six hours after decompression. However, onset as late as 24 hours or more does not completely rule out the possibility of decompression sickness. Diagnosis is usually not difficult, but a number of factors can complicate the picture. Treatment by adequate recompression is in order even when the diagnosis is uncertain.

ACKNOWLEDGEMENTS

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Chapter VII

RECOMPRESSION

Treatment of Decompression Sickness and Air Embolism

Edward H. Lanphier

Almost from the beginning of compressed-air work, it was known that returning to pressure often relieved the pain of bends. In time, "medical locks" were provided at caisson and tunnel construction sites, recompression chambers were placed aboard diving ships, and specific procedures were developed for recompression. As the term is generally used, it refers not only to putting the victim back under pressure but also to subsequent decompression according to a schedule designed for therapeutic application. Recompression remains the only reliably effective treatment for decompression sickness or air embolism, and the need for it can be expected to arise in hyperbaric practice.

As has already been emphasized, recompression is indicated not only when the diagnosis of decompression sickness or air embolism is certain but in most questionable cases as well. The main decision remaining concerns the specific procedure to be used.

RECOMPRESSION PROCEDURES

Several different approaches to recompression are in use in various places at the present time, but the U.S. Navy Treatment Tables (Table 1-21) have displaced a number of older methods and are now the most widely accepted. In naval diving, for example, they are virtually the international standard.

In view of the status of the U.S. Navy procedure, use of any other approach in American hyperbaric installations requires exceptionally good justification. This is not to suggest that the U.S. Navy tables could not be improved or to deny that other methods can yield satisfactory results. At the same time, it is true that proving the adequacy and advantage of a new recompression procedure requires a body of experience unlikely to be gathered except in naval diving or major tunnel or caisson projects. Even in some of these, reported results must be scrutinized.

In this chapter, major emphasis is placed upon the U.S. Navy treatment procedure. However, two other methods that appear to have particular merit are presented in addition. A discussion of circumstances justifying their use and of the pros and cons of the matter is also provided.

U.S. NAVY TREATMENT TABLES

The U.S. Navy Diving Manual sets forth the procedure for recompression in Table 1-21 (the treatment tables themselves) and Table 1-22 (two pages of Notes on Recompression arranged for rapid reference). Both are reproduced here directly from the Diving Manual. They were intended to be intelligible to divers and hospital corpsmen as well as to physicians, and very little additional information is essential to adequate handling of recompression. However, useful supplementary discussion will be provided here.

Attempts to memorize the tables or notes are not suggested, but considerable familiarity with their content is clearly advisable well in advance of actual need. This is particularly true of the general outline of the tables and of all the points of decision that may arise in their use.

"Tests of Pressure"

The first point of decision is whether recompression is needed or not. As has been discussed, at least a trial of recompression is in order even in questionable cases. Dramatic relief in the process will establish the diagnosis (except in some cases of abdominal pain, as discussed) and will prove the need for full treatment. However, not all symptoms can be relieved promptly or completely, especially if they have been present for some time and involve residual damage. According to U.S. Navy standards, a "test of pressure" is not conclusively negative in suspected pain only cases unless recompression to 6 atm abs (165 feet) and 30 minutes at that pressure fail to provide any degree of relief.

Even when a "test of pressure" is considered

negative in such cases, Table 1-21 specifies that decompression should nevertheless be conducted according to Treatment Table 2 or 2-A. This is, of course, the safest procedure and should be followed if the test is at all equivocal. If the test is clearly negative, however, it is reasonable to apply ordinary decompression procedure. Note that unless 12 hours have elapsed since the original exposure, the test itself will represent a repetitive dive. In any event, the individual must be observed very closely during decompression.

Application of a "test of pressure" in the presence of symptoms in the serious category presents additional problems. Failure to relieve symptoms within 30 minutes at 165 feet does not necessarily warrant a negative diagnosis, although at least some measure of improvement will generally be noted within that period if decompression sickness or air embolism is present. If the results of the test are negative or equivocal, the decision to commence decompression after 30 minutes should not be made without due consideration of its implications. Decompression according to Table 2 or 2-A is indicated at the minimum.

Selection of Table

The pattern followed in deciding which of the treatment tables to follow is set forth clearly in Table 1-21, but decisions may be difficult despite their apparent simplicity. The importance of looking closely for neurological abnormalities in cases dominated by pain deserves reemphasis. This requires careful examination before recompression, and borderline abnormalities or positive findings that may be normal for the individual can complicate matters considerably. In general, the more conservative course must be followed if there are any neurological defects. The use of Table 1 or 1-A would be difficult to justify under such circumstances, but sometimes advantage can be taken of the fact that Tables 2 and 3 are identical until the 30-foot stop is reached during decompression. If a questionable neurological defect has not improved during the first part of the treatment and has not become more pronounced during the decompression phase, it can usually be discounted and treatment completed on Table 2 or 2-A.

The recommendation against careful preliminary examination when serious symptoms are obviously present reflects the risk involved in any delay of recompression and the fact that additional findings will not influence the initial course of treatment. In a physician's hands, this is a matter of clinical judgement; but the principle remains valid. The emphasis in this situation is placed upon careful examination at 165 feet, where the decision between Table 3 and Table 4 must be made. Examination should commence at least as soon as that pressure is reached. If a definite abnormality remains after 30 minutes, use of Table 4 is mandatory even if the problem could be attributed to residual damage. In this event, the next decision concerns the period of time to be spent at 165 feet before commencing decompression according to Table 4. By and large, it is desirable to remain either for the full two hours or at least for 30 minutes after complete relief has been obtained. When relief is not complete even after 2 hours, residual damage is the most likely explanation. The problem is discussed under "Modification of Tables," below.

If Table 3 is chosen despite questionable remaining abnormalities, these must be given special attention throughout the decompression phase. If they become more pronounced, it must be assumed that remaining bubbles are responsible, and returning to 165 feet is indicated. Table 4 must then be followed. If in the process the total time at greater pressure becomes longer than that provided for by Table 4, the possibility of further difficulties during decompression arises. Compensatory measures are discussed under "Modification of Tables."

If air embolism is the suspected diagnosis, either Table 3 or Table 4 must, of course, be used. Recent experience suggests that Table 4 is preferable in these cases even when satisfactory relief is obtained during compression or in less than 30 minutes at 165 feet.

Severe cases of "pain only" bends sometimes require treatment on tables longer than Table 2 or 2-A. This problem arises when there is no question about the etiology or response but when adequate relief has not been obtained within 30 minutes at 165 feet. Table 3 does not provide a longer period at maximum pressure, so Table 4 becomes the only possible choice.

DELAY IN TREATMENT

A primary cause of equivocal or incomplete relief, difficult decisions, and the need for long-table treatment is a significant delay between the onset of symptoms and the beginning of treatment. It might even be argued that treatment becomes pointless after a certain

Table 1-21. Treatment of Decompression Sickness and Air Embolism.

Stops		Bends—Pain only				Serious Symptoms	
Rate of descent—25 ft. per min. Rate of ascent—1 minute between stops.		Pain relieved at depths less than 66 ft. Use table 1—A if O_2 is not available.		Pain relieved at depths greater than 66 ft. Use table 2—A if O_2 is not available. If pain does not improve within 30 min. at 165 ft. the case is probably not bends. Decompress on table 2 or 2—A.		Serious symptoms include any one of the following: 1. Unconsciousness. 2. Convulsions. 3. Weakness or inability to use arms or legs. 4. Air embolism. 5. Any visual disturbances. 6. Dizziness. 7. Loss of speech or hearing. 8. Severe shortness of breath or chokes. 9. Bends occurring while still under pressure.	
						Symptoms relieved within 30 minutes at 165 ft. Use table 3	Symptoms not relieved within 30 minutes at 165 ft. Use table 4
Pounds	Feet	Table 1	Table 1-A	Table 2	Table 2-A	Table 3	Table 4
73.4	165	----------	----------	30 (air)	30 (air)	30 (air)	30 to 120 (air)
62.3	140	----------	----------	12 (air)	12 (air)	12 (air)	30 (air)
53.4	120	----------	----------	12 (air)	12 (air)	12 (air)	30 (air)
44.5	100	30 (air)	30 (air)	12 (air)	12 (air)	12 (air)	30 (air)
35.6	80	12 (air)	12 (air)	12 (air)	12 (air)	12 (air)	30 (air)
26.7	60	30 (O_2)	30 (air)	30 (O_2)	30 (air)	30 (O_2) or (air)	6 hrs. (air)
22.3	50	30 (O_2)	30 (air)	30 (O_2)	30 (air)	30 (O_2) or (air)	6 hrs. (air)
17.8	40	30 (O_2)	30 (air)	30 (O_2)	30 (air)	30 (O_2) or (air)	6 hrs. (air)
13.4	30	5 (O_2)	60 (air)	60 (O_2)	2 hrs. (air)	12 hrs. (air)	First 11 hrs. (air) Then 1 hr. (O_2) or (air)*
8.9	20		60 (air)	5 (O_2)	2 hrs. (air)	2 hrs. (air)	First 1 hr. (air) Then 1 hr. (O_2) or (air)*
4.5	10		2 hrs. (air)		4 hrs. (air)	2 hrs. (air)	First 1 hr. (air) Then 1 hr. (O_2) or (air)
Surface			1 min. (air)		1 min. (air)	1 min. (air)	1 min. (O_2) *

Time at all stops in minutes unless otherwise indicated.

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*See modifying comments in text (page 101).

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TABLE 1-22

NOTES ON RECOMPRESSION

Explanation: All references to TABLES *indicate parts of table 1-21 "Treatment of Decompression Sickness and Air Embolism."*

1. *General Considerations*
   a. Follow TREATMENT TABLES (table 1-21) accurately.
   b. Permit no shortening or other alteration of tables except on advice of trained *diving medical officer* or in extreme emergency.

2. *Rate of Descent in Chamber*
   a. Normal rate is 25 feet per minute.
   b. Serious symptoms: rapid descent is desirable.
   c. If pain increases on descent: stop, resume at a rate tolerated by patient.

3. *Treatment Depth*
   a. Go to full depth indicated by table required.
   b. Do not go beyond 165 feet except on decision of medical officer.

4. *Examination of Patient* (see article 1.6.2(14))
   a. If no serious symptoms are evident and pain is not severe, examine thoroughly before treatment.
   b. If any serious symptom is noted, do not delay descent for examination or for determining depth of relief.
   c. In "pain only" cases where relief is reported before reaching 66 feet, make sure it is complete before deciding on TABLE 1.
   d. On reaching maximum depth of treatment, examine as completely as possible to detect
      1) Incomplete relief
      2) Any symptoms overlooked
      NOTE.—At the very least, have patient stand and walk length of chamber.
   e. Recheck before leaving bottom.
   f. Ask patient how he feels before and after coming to each stop and periodically during long stops.
   g. Do not let patient sleep through changes of depth or for more than an hour at a time at any stop. (Symptoms can develop or recur during sleep.)
   h. Recheck patient before leaving last stop.

5. *Patient Getting Worse*
   a. Never continue bringing a patient up if his condition is worsening.
   b. Treat as a *recurrence during treatment* (see 6).
   c. Consider use of helium-oxygen as breathing medium for patient (see 8).

6. *Recurrence of Symptoms*
   a. *During* treatment:
      1) Take patient to depth of relief (but never to less than 30 feet; and not deeper
   * than 165 feet except on decision of medical officer).
      (If recurrence involves serious symptom not previously present, take patient to 165 feet.)
      2) Complete the treatment according to TABLE 4.
   b. *Following* treatment:
      1) Recompress to depth giving relief.
      2) If depth of relief is less than 30 feet,
         a) Take to 30 feet.
         b) Decompress from 30-foot stop according to TABLE 3.
      3) If relief occurs deeper than 30 feet,
         a) Keep patient at depth of relief for 30 minutes.
         b) Complete remaining stops of TABLE 3.
         NOTE.—If original treatment was on TABLE 3, use TABLE 4.
      4) Examine carefully to be sure no serious symptom is present. If the original treatment was on TABLE 1 or TABLE 2, appearance of a serious symptom requires full treatment on TABLE 3 or TABLE 4.

MOST FREQUENT ERRORS RELATED TO TREATMENT

1. Diver's failure to report symptoms early.
2. Failure to treat doubtful cases.
3. Failure to treat promptly.
4. Failure to recognize serious symptoms.
5. Failure to treat adequately.
6. Failure to keep patient near chamber after treatment.

ALWAYS KEEP DIVER CLOSE TO CHAMBER FOR AT LEAST 6 HOURS AFTER TREATMENT. (Keep him for 24 hours unless very prompt return can be assured.)

7. *Use of Oxygen*
   a. Use oxygen wherever permitted by tables unless
      1) Patient has not had oxygen tolerance test, or
      2) Is known to tolerate oxygen poorly.
   b. Be sure mask fits snugly.
   c. Take all precautions against fire (see table 1-29).
   d. Tend carefully, being alert for symptoms of oxygen poisoning such as
      1) Twitching 3) Nausea
      2) Dizziness 4) Blurring of vision
   e. Know what to do in event of convulsion. Have mouth-bit available.
   f. If symptoms appear, remove mask at once.
   g. If oxygen breathing must be interrupted—
      1) On TABLE 1, proceed on TABLE 1-A.
      2) On TABLE 2, proceed on TABLE 2-A.
      3) On TABLE 3, continue on TABLE 3 using air.
   h. At medical officer's discretion, oxygen breathing may be resumed at 40-foot stop. If this is done, complete treatment as follows:
      1) Resuming from TABLE 1-A: breathe oxygen:
         at 40 feet for 30 minutes
         at 30 feet for 1 hour
      2) Resuming from TABLE 2-A: breathe oxygen:
         at 40 feet for 30 minutes
         at 30 feet for 2 hours
      3) In both cases, then surface in 5 minutes still breathing oxygen.
      4) Resuming from TABLE 3: breathe oxygen:
         at 40 feet for 30 minutes
         at 30 feet for first hour
         (then finish treatment with air)

*See additional information in text (page 100).

TABLE 1-22.—Continued

NOTES ON RECOMPRESSION

Explanation: All references to TABLES *indicate parts of table 1-21 "Treatment of Decompression Sickness and Air Embolism."*—Continued

8. *Use of Helium-Oxygen*
a. Helium-oxygen mixtures (ratio about 80:20) can be used *instead of air* (not in place of oxygen) in all types of treatment and at any depth.
b. Use of helium-oxygen is especially desirable in any patient who
1) Has serious symptoms that fail to clear within a short time at 165 feet.
2) Has recurrence or otherwise becomes worse at any stage of treatment.
3) Has any difficulty in breathing.

9. *Tenders*
a. A qualified tender must be in chamber
1) If patient has had any serious symptom.
2) Whenever patient is breathing oxygen.
3) When patient needs unusual observation or care for any reason.
b. Tender must be alert for any change in patient, especially during oxygen breathing. (See 7, d-f.)
c. *Tender must breathe oxygen* if he has been with patient throughout TABLE 1 OR TABLE 2
TABLE 1: Breathe oxygen—
at 40 feet for 30 minutes
TABLE 2: Breathe oxygen—
at 30 feet for 1 hour
d. Tender in chamber only through oxygen breathing part of TABLE 1 or 2 gains safety-factor by breathing oxygen for 30 minutes of last stop, but this is not essential. Tender may breathe
* oxygen during use of TABLE 3 or 4 at 40 feet or less.
e. Anyone entering chamber and leaving before completion of treatment must be decompressed according to standard diving tables.
f. Personnel outside must specify and control decompression of anyone leaving chamber and must review all decisions concerning treatment or decompression made by personnel (including medical officer) inside chamber.

10. *Ventilation of Chamber*
(See art. 1.6.21, par. 18)
Rule 1. Volume of air required (volume as measured at chamber pressure—applies at any depth):
a. Basic requirement:
1) Allow 2 cubic feet per minute per man.
2) *Add* 2 cubic feet per minute for each man *not at rest* (as tender actively taking care of patient).
b. When using oxygen:
Allow 4 cubic feet of air *per man breathing oxygen* if this yields larger figure than basic requirement. (Do not add to basic requirement.)

Rule 2. Maximum interval between ventilations:
a. Not using oxygen:
$$\text{Interval (min.)} = \frac{\text{Chamber (or lock) volume (cu. ft.)}}{\text{Basic vent. req. (cu. ft./min.) (from rule 1)}}$$
b. Using oxygen:
$$\text{Interval (min.)} = \frac{\text{Chamber (or lock) vol. (cu. ft.)}}{\text{No. of men br. } O_2 \times 10}$$
a. Timing of ventilation:
1) Use any convenient interval shorter than maximum from rule 2.
2) (Continuous steady-rate ventilation is also satisfactory.)
b. Volume used at each ventilation:
1) Multiply volume requirement (cu. ft./min.) from rule 1 by number of minutes since start of last ventilation.
c. Use predetermined exhaust valve settings to obtain required volume of ventilation. (See article 1.6.21 (18), (b).)

11. *First Aid*
a. First aid measures may be required in addition to recompression. Do not neglect them.
b. See table 1-26 and *Standard First Aid Training Course*, NAVPERS 1-0081.

12. *Recompression in the Water*
a. Recompression without a chamber is difficult and hazardous. Except in grave emergency, seek nearest chamber even if at considerable distance.
b. If water recompression must be used and diver is conscious and able to care for himself:
1) Use deep sea diving rig if available.
2) Follow treatment tables as closely as possible.
3) Maintain constant communication.
4) Have standby diver ready.
c. If diver is unconscious or incapacitated, send another diver with him to control his valves and otherwise assist him.
d. If lightweight diving outfit or scuba must be used, keep at least one diver with patient at all times. Plan carefully for shifting rigs or cylinders. Have ample number of tenders topside and at intermediate depths.
e. If depth is inadequate for full treatment according to tables:
1) Take patient to maximum available depth.
2) Keep him there 30 minutes.
3) Bring him up according to TABLE 3 if he can tolerate exposure. (If patient has been taken beyond 100 feet, do not use stops shorter than those of TABLE 2-A.)

*See modifying comment in text (page 101).

period of delay. Rivera's analysis of 935 cases of decompression sickness (1) indicates that the prognosis is poor if the interval exceeds 36 hours, but he concluded that all individuals with symptoms deserve recompression regardless of the elapsed time. The chances of success are admittedly even poorer in delayed treatment of air embolism, but we have no basis for setting any limit of time. A patient with air embolism deserves treatment as long as he remains alive.

RECURRENCE OF SYMPTOMS

Recurrence of symptoms during the decompression phase has sometimes been attributed to the development of edema from the original injury rather than to the re-expansion or continued growth of bubbles. This explanation has merit especially in air embolism and the central-nervous-system manifestations of decompression sickness. However, we have no basis for assuming this to be the explanation of recurrence or deterioration of condition in any individual patient. It must therefore always be assumed that bubbles are responsible and that immediate return to higher pressure is essential. The U.S. Navy rules for management of recurrence are set forth in Table 1-22, part 6. Recurrence while still under pressure signifies a more serious situation than recurrence after treatment, and thus calls for more drastic management. Recurrence can generally be accepted as proof that the bubbles in question were not resolved in the usual manner by original application of pressure beyond that of relief. Reapplication of higher pressure is therefore not considered promising, and the additional uptake of "new" gas that this involves is best avoided. The approach is therefore shifted to spending longer periods at pressures close to that of relief. Here it is assumed that relief will be maintained while the bubbles gradually lose gas to their surroundings and the total body gas content equilibrates at a level that will permit safe return to normal pressure on the indicated treatment table. Recurrence during or following treatment on Table 4 requires repeating application of Table 4 at least from the depth of relief. If depth of relief exceeds 80 feet, repeat entire table from 165 feet.

MODIFICATION OF TABLES

The rigidity of the U.S. Navy treatment procedure as presented reflects the expectation that it would often have to be employed by relatively untrained personnel. An experienced Submarine/Diving Medical Officer is largely at liberty to modify it. In practice, however, a rational basis for modification seldom exists, except on the rare occasions when the standard procedure fails to produce satisfactory results.

The possible need for recompression beyond 165 feet arises when symptoms fail to respond adequately within a reasonable period at that pressure. However, the gains to be expected beyond 6 atm abs are relatively small; and the problems of subsequent decompression are compounded seriously. It is virtually always better to remain at 165 feet. Lack of adequate response at 165 feet, especially within the two hours allowed by Table 4, must usually be due to damage that cannot in any case be remedied by compression. However, going to a higher pressure is not likely to be criticized if it seemed to provide the only possibility of saving a patient's life. Fortunately, the question seldom arises.

If the patient's condition remains unsatisfactory after two hours have passed at 165 feet, one is faced with a very difficult decision: to remain at 165 feet for a longer period or to proceed with decompression according to Table 4. Unless definite improvement is taking place, the problem probably represents residual damage, and decompression is in order. If the problem is related to unresolved bubbles, worsening upon decompression will probably make this clear. If so, returning to higher pressure for a longer period will be required. If there is no particular change upon decompression, the process can continue.

Modification of the tables to meet unforeseen complications is made difficult by lack of crucial information as to the status of bubbles and gas tensions and also by lack of clear-cut precedents. However, a number of basic principles apply:

1. A patient whose condition deteriorates with decompression must be taken back to higher pressure without delay, regardless of the difficulties that this will entail. Only when deterioration is clearly caused by factors other than re-expansion of bubbles can decompression continue.

2. Prolonged exposure to moderate pressure is not likely to be harmful to the patient. He can be kept at pressures between 30 and 60 feet for days if necessary.

3. Oxygen administration will greatly hasten elimination of gas from bubbles and from the body as a whole. It can be employed to any extent compatible with the risk of producing

convulsions or lung damage. It is a valuable method of dealing with patients whose symptoms tend to recur when decompression is attempted, and it is one of the best means of compensating for unusual periods of time spent at higher pressure.

4. Although their value is much less certain than that of oxygen, helium-oxygen mixtures can be employed freely, except as a substitute for obligatory use of oxygen.

5. Decompression Table 1-9 (exceptional exposures) is not to be viewed as a treatment table, but its longest schedules provide some basis for extrication from difficult situations not entirely covered by Table 4, as when recompression has been carried to higher pressure. In no such case should decompression stops be shorter than those of Table 4, and it may be desirable to add time to the shallower stops of Table 4. For example, the "30-foot soak" can be extended to a full day.

USE OF OXYGEN

If oxygen breathing is employed to permit use of the much shorter treatment of Tables 1 and 2 (as opposed to 1-A and 2-A), it is imperative to ensure that the patient is actually receiving oxygen throughout the designated periods of oxygen breathing. This requires administration with a mask that permits no dilution of the oxygen by inward leaks of air. Where the patient's ability to tolerate oxygen is uncertain, it is desirable to postpone the beginning of oxygen breathing until the 40-foot stop is reached, then to follow the rules given in Table 1-22, section 7h. With this modification, it is desirable to employ oxygen at least to the full extent of optional use on Table 3 and 4. Especially on Table 4, additional periods—such as 30-minute periods on oxygen separated by intervals of one hour on air—are probably desirable during at least a portion of the long stops at 40 and 30 feet. It is now considered mandatory for both patients and tenders to breathe oxygen for the final hour of the 30-, 20-, and 10-foot stops of Table 4.

AUXILIARY MEASURES

Especially in a hospital setting, application of additional diagnostic and therapeutic measures is largely a matter of clinical judgement. In the case of preliminary diagnostic procedures, the main problem is the possibility of excessive delay in starting treatment. For example, chest X-rays are obviously desirable in cases of air embolism attributable to pulmonary obstruction. However, the delay involved in obtaining these before treatment could seldom be justified. If they could be obtained later, either within the chamber with special equipment or through a port, they are clearly indicated.

In the vast majority of patients with decompression sickness, no form of therapy other than recompression is required; but with very few exceptions, any form of additional treatment indicated by the patient's condition can and should be administered during recompression. The exceptions are primarily pharmacological. For example, narcotics not only obscure the response of symptoms to pressure but may have unexpected effects under higher pressures or during oxygen administration. Any drug whose actions under such conditions are not well known is best avoided. Detection and management of impending shock are particularly important. Fluid intake and bladder function must not be neglected.

The most urgent current questions concerning auxiliary therapy relate to the use of hypothermia and of agents designed to reduce cerebral edema in the treatment of air embolism and central-nervous-system decompression sickness. Routine application of these measures in such cases is certainly not mandatory in the light of present knowledge, but their use may well be in order in patients who do not respond fully to recompression or who show subsequent deterioration that is not reversed by a prompt return to higher pressure.

The arrival of adequate, appetizing meals can become very important to a patient who is in normal condition following relief of symptoms on long-table treatment. A normal diet need not be denied, except that liquid nourishment is preferable during phases when oxygen is administered at pressures greater than 30 feet. (Nausea and vomiting are among the more common forerunners of oxygen convulsions and may follow convulsion.)

SUCCESS OF TREATMENT

In most cases of decompression sickness, recompression is promptly and impressively effective. The matter of greatest concern is the possibility of recurrence of symptoms, requiring treatment to be repeated. Even in these cases, relief is generally restored rapidly by retreatment, and complete cure is achieved in the process.

Slark (2) reported that in his series of 137 cases of decompression sickness in Royal Navy divers, the recurrence rate was about 20 per cent regardless of the recompression procedure employed. The methods included the U.S. Navy Treatment Tables as well as the slightly modified version of these currently in use in the Royal Navy.

Rivera (1) analyzed the results of treatment in his series of 935 cases in terms of "true failures" of treatment when the U.S. Navy tables were properly used. A true failure was defined as a case in which symptoms recurred or full cure was not achieved. Cases eliminated from consideration were those in which complicating factors of certain types were present: incorrect initial treatment, delay over 24 hours, aggravation by air transport, or alcohol intoxication. On this basis, he found that the overall incidence of true failure was 6.7 per cent on first treatment and 1.7 per cent on treatment of recurrences. Incidence of failure of first treatment was also determined for each of the U.S. Navy tables:

Table Used	% True Failure	Table Used	% True Failure
1	5.5	3 (air)	6.5
1-A	6.5	4 (air)	15.1
2	6.0	4 (O_2)	25.0
3 (O_2)	7.2	4 (He-O_2)	25.0

Except for Table 4, the record is certainly satisfactory. Cases treated on Table 4 were relatively few in number and included individuals in whom irreversible damage was almost certainly present. However, inherent shortcomings of the Table are suggested by the fact that a number of tenders who accompanied patients through Table 4 treatment developed decompression sickness. Pending modification of the table, increased utilization of oxygen breathing by both patients and tenders is strongly advised. At minimum, the periods suggested should be utilized.

The results of treatment in air embolsim have not been analyzed, but it is not to be expected that the record would be as good as in decompression sickness. However, complete relief is not uncommon when treatment is prompt.

PERSONNEL REQUIRED IN TREATMENT

The total number of people required in the course of long-table treatment is large and will usually exceed the number available in the regular hyperbaric staff. This means that it is highly desirable to give a number of potential auxiliaries sufficient training to permit them to take over routine duties and provide the regular operating and medical staff some periods of relief. Estimation of the amount of manpower involved in treatment of a Table 4 case at Buffalo indicated that, all jobs considered, 320 man-hours were required.

Keeping tenders with the patient throughout treatment on Table 3 or 4 is a particular problem. If the chamber has only one compartment, those who originally accompany the patient must obviously remain; and this is often the most practical arrangement in any case. For example, a physician who accompanies the patient and fully evaluates the response to treatment will usually find that he has obligated himself to prolonged decompression. If the chamber has an access lock, the physician can get out; but it may well be unwise to tie up the lock with the lengthy decompression that he may require. It is often better in such a case for the physician to "ride out the table" with the patient and serve as his primary tender. If further assistance is required, this can be provided by sending others in for periods requiring only short decompression. Unless the chamber has more than one access lock, however, the "relay" approach does not become practical until pressures well below 165 feet have been reached.

Rules concerning review by personnel outside the chamber of any decisions made inside, and the control of chamber operation from outside, stem primarily from the demonstrated unreliability of even the most qualified individuals when they are affected by nitrogen narcosis at higher pressure. Examination of the patient at 165 feet is a case in point. Unless he breathes a helium-oxygen mixture during this period, the physician will do well to let every step of the examination be specified, and the findings noted, by personnel outside. Recording both sides of all conversations on tape is also desirable.

As has been mentioned, the occurrence of decompression sickness in some tenders who have "ridden out" Table 4 indicates that the suggested periods of oxygen breathing should be used by both patient and tender. Additional periods of oxygen breathing may also be in order. Whenever a tender breathes oxygen at pressures greater than 30 feet, an additional tender must be provided to observe him and take over his duties.

THEORETICAL CONSIDERATIONS

The practical side of recompression, as it appears at the present time, has been presented first as a matter of logical priority. However, some consideration of the theoretical side is necessary for constructive thought about possible alternatives and future developments.

Objectives

The purpose of recompression is to provide prompt and lasting relief of the signs and symptoms of decompression sickness and air embolism. To accomplish this purpose, any recompression procedure must be designed with three specific objectives in mind:

1. To reduce the bubble(s) to asymptomatic size in a short time.
2. To ensure that no bubble again becomes symptomatic upon subsequent decompression.
3. To conduct the decompression phase in such a way that new bubbles do not form in the process.

Maximum Pressure

The increase in ambient pressure in recompression causes compression of bubbles in accordance with Boyle's Law, and prompt relief of symptoms is often achieved on this basis. If the maximum pressure employed in treatment is reached without relief, then the relatively slow process of diffusion of gases out of a bubble must be relied upon to provide relief. Having to wait for relief is not a serious matter in milder cases of localized pain, but it is clearly undesirable in the presence of serious symptoms. Significant delay in relief could be fatal in air embolism. It follows that the maximum pressure employed in treatment should be as high as is practical.

The limit of practicality is not readily defined. For example, compression to 6 atm abs reduces the volume of a bubble to 1/6 (16.7 per cent) of its original value, while going to 7 atm brings it down only slightly farther—to 1/7 or 14.3 per cent. The decrease in diameter of a spherical bubble is even less impressive: from 55 per cent of the original value at 6 atm to 52.3 per cent at 7 atm. In developing the present U.S. Navy Treatment Tables, Van Der Aue, Duffner, and Behnke (3) concluded that recompression beyond 6 atm would serve mainly to increase the nitrogen content of the tissues (and thus delay return to a pressure at which oxygen could be breathed safely) without appreciably decreasing the size of bubbles. The same argument might have been applied at levels other than 6 atm, but this judgement can be accepted.

The considerations mentioned, and especially the fact that the USN tables have in fact been based upon recompression of most patients to 6 atm, make it highly desirable, if not imperative, for at least one compartment of a hyperbaric facility to have capability for operation at this pressure and means of reaching it rapidly.

Resolution of Bubbles

Ability to relieve symptoms promptly is not the only reason for using relatively high pressure. The U.S. Navy procedure uses 6 atm (except in very responsive bends) even if relief has occurred at a much lower pressure. The reasoning here concerns the resolution of bubbles: causing them to disappear entirely, or at least assuring that they will not readily return to symptomatic size.

Under normal conditions, any bubble containing normal atmospheric components will tend to disappear by outward diffusion of gas. The total gas pressure inside is bound to be greater than the total pressure of dissolved gas in the bubble's surroundings. When tissues are supersaturated with gas, this may no longer be true. The bubble may then grow by inward diffusion of gas from its surroundings. To assure that this can no longer occur, the ambient pressure must at least be equal to the pressure of dissolved gas in the surroundings of the bubble. When this point of balance is reached in recompression can never be certain. Under some conditions, it may not have been reached even at the pressure of relief.

The original pressure of exposure is only one of several factors influencing the size of a bubble and the pressure required for adequate treatment. The only real bearing of exposure pressure upon treatment pressure is that recompression to "working pressure" assures that the gas pressure around a bubble cannot be greater than that within the bubble. Even if prompt relief is not obtained, the bubble must stop growing and sooner or later must shrink to a size giving relief.

Since the process of gas diffusion from bubbles may be slow, methods of speeding it are desirable. The most effective method is administration of oxygen at increased pressure. This is probably most efficiently done at that pressure where the difference between arterial and venous oxygen pressures is at its maximum:

in normally perfused tissues, in the neighborhood of 3 atm abs. Above this point, the venous oxygen pressure, reflecting maximum saturation of hemoglobin, will tend to rise along with the arterial value, so no great further gain in rate of gas transfer will be obtained from oxygen breathing at higher pressure.

Practical application of oxygen breathing is limited, by oxygen toxicity, to pressures around 3 atm and this pressure may not be sufficient to provide reasonably prompt relief. Higher pressure is not only more likely to provide rapid relief but also tends to hasten the movement of gas out of bubbles through a chain of interrelated processes. One of these concerns the pressure produced within a bubble by its own surface tension. The smaller a bubble becomes, the higher the pressure within it. At a certain microscopic "critical size," this pressure becomes so great that rapid gas loss causes the bubble to disappear almost instantly. Compression only to the pressure of relief is not very likely to reduce a bubble to critical size. Higher pressure is at least more likely to do so.

The U.S. Navy Treatment Tables probably represent a reasonable balance among the various factors at issue, but there are many reasons to hope that they can be improved upon. One of the main objections to them in clinical hyperbaric work is the requirement for 6 atm pressure capability, but most would agree that this is highly desirable for treatment of air embolism and serious forms of decompression sickness when satisfactory relief cannot be obtained at lower pressure. (It is also likely that some clinical hyperbaric procedures will come to be conducted at higher pressures.) In many cases of decompression sickness, at least the milder ones, greater overall efficiency of treatment might be obtained by limiting the pressure to a lower level and thus avoiding uptake of large amounts of "new" gas. In effect, Tables 1 and 1-A provide such an approach and appear quite satisfactory when applied to appropriate cases.

The most interesting newer concept, presented in detail below as an alternative method of treatment, involves limiting the pressure and breathing oxygen from the start. Not only is bubble resolution accelerated, but no uptake of "new" inert gas occurs. It is not unlikely that such a method may come to be applied to the majority of cases in which very prompt relief is not required.

ALTERNATIVE PROCEDURES

If the pressure capability of a hyperbaric facility is limited to 4 atm abs, the U.S. Navy tables can be applied properly only to "pain-only" cases relieved at 3 atm or less (Table 1 and 1-A). Many cases from low-pressure exposure will fall into this category, but some surely will not. It is thus essential to have procedures of established worth that can be applied instead of the U.S. Navy procedure.

The methods developed, largely by Griffiths, and applied during construction of the Dartford Tunnel (4,5) proved essentially satisfactory and are in current use with slight modifications on the Blackwall and Tyne tunnel projects. It must be emphasized that the Dartford experience was limited to pressures not above 28 psi. Although the overall correlation between pressures of exposure and relief is poor, relief in tunnel workers is generally at lower pressures than in divers (1).

Low-Pressure Recompression Using Air

The following description and discussion of procedures for treatment of Type 1 (pain-only) and Type 2 (serious) decompression sickness is taken directly, by permission, from an excellent but unpublished manual prepared by Dr. Griffiths for his medical-lock attendants at the Blackwall Tunnel site in London:

TREATMENT

(Griffiths - Dartford)

"Treatment (recompression) is always done in a medical lock, which must contain two compartments. One lock should always be available when men are working at pressures over 16 psi.

TYPE 1. (Simple Bends)

"Pains in the arms and/or legs, much more frequently the latter. They may commence at any time up to twelve hours after decompression, the commonest time being 2-4 hours after. The intensity of the pain varies; it may be slight (niggles) or agonizing.

Standard Method

"Recompress to three pounds above working pressure.

"Hold that pressure for ten minutes after all the pains have completely gone.

"Decompress. Use the official man-lock decompression tables but stop the quick drop at a pressure two pounds higher than that

laid down and take 15 minutes for each of the remaining pounds.

"In the majority of cases the pains will disappear at the higher pressure and will not return. A number of cases are much more difficult in that the pains may not be relieved at the higher pressure or may return on decompression.

"There are no regulations as to treatment so, with difficult cases, and some can be extremely difficult, there are various experimental methods that can be tried; the object being to relieve the man permanently of his pain.

"In this country the minumum effective pressure treatment is used as opposed to the method used in the U.S.A. where much higher pressures are used. There is something to be said for both methods.

"If the pains are not relieved by the initial recompression a higher pressure must be used but patience should be exercised, waiting 10-15 minutes to see the effect of each rise of two pounds, before going higher. It is rare for more than ten pounds above working pressure to be required.

"Should the pains return towards the end of, or after the therapeutic decompression, the man must be recompressed again, either back to the original effective pressure, or recompressed slowly, stopping at the pressure where relief is obtained. On occasions this pressure may be quite low, twelve or fifteen pounds. Should this be the case, wait at that pressure for half an hour then decompress.

"Should a second recompression be necessary it may be advantageous to break the quick phase of the decompression with a 'soak' for half an hour at 12 psi. (Alternative.)

"In the initial recompression the pressure may be raised comparatively slowly, stopping when pains are relieved. On occasions this pressure may be considerably lower than working pressure. Wait half an hour then decompress.

* * * * * *

"The great majority of cases will be cured by one of, or a combination of, the above methods. An occasional case will not be relieved completely of symptoms in spite of multiple recompressions. It is this case that might better be treated by the American high pressure (up to 70 psi) treatment. Our medical locks are not usually made to withstand such high pressures. It is generally accepted that all cases should be treated by recompression. Nevertheless it will be found that the pains of simple bends will, without treatment, disappear within three days.

TYPE 2.

"These cases are much more serious. Recompression is URGENT. They must have prolonged and careful treatment.

"The symptoms usually commence early; sometimes during the last few pounds of decompression; generally within three quarters of an hour of decompression. Rarely they are delayed for some hours.

"Should a man faint or become ill during decompression in the man-lock with the shift, the whole shift must be recompressed at once and the patient transferred to the working chamber. He should remain there until symptom free when he can be decompressed slowly and transferred to the medical lock for further treatment.

"Should a man collapse on leaving the man-

Note: The following table, derived from British Work in Compressed Air Special Regulations, 1958 (6) (with the 2-psi addition indicated above), gives the pressure at which to stop the initial rapid "quick drop" phase of decompression: At the beginning of decompression, drop the pressure to the appropriate value (below) in not less than 2 minutes.

Maximum pressure of exposure or recompression (whichever is greater):	Pressure at end of "quick drop":
up to 22 psi	5 psi
26	7
30	9
34	11
38	13
42	15
46	17
50	19

Important: Drop the pressure from the "quick drop" level at one psi / 15 min unless USN decompression tables (viewing recompression as a repetitive dive) require a longer period before reaching a given pressure. E.H.L.

lock he should be recompressed at once in the man-lock or even in the muck-lock (providing the latter has suitable controls), rather than valuable minutes should be lost by transporting him to the medical lock.

"The basic principle of treatment of TYPE 2 cases is recompression to working pressure at once, using higher pressure if required, maintaining the effective pressure for half an hour after all signs and symptoms have disappeared and then decompressing very slowly.

"The majority of cases, particularly if caught early, respond dramatically to recompression. If response is poor then higher pressure must be used, even to the highest pressure available or that the lock will stand. (For this reason the air supply to medical locks must be from the high pressure compressors.)

"Recompression treatment should be continued so long as there is any improvement whatsoever and only as a last resort must a patient be transferred to hospital, where treatment can be only symptomatic.

"The method of decompression suggested below is prolonged but has, up to now, proved to be satisfactory.

Method of Decompression

"Reduce from the effective pressure to 15 psi at the rate of one pound every 15 minutes.

" 'Soak at this (15 psi)of 1-1/2 pressure for FOUR HOURS.

" 'Then reduce at the rate of one pound every half hour, giving 'soaks' of 1.5 hours at 8 psi, one hour at 4 psi, and one hour at 2 psi'.

"These reductions in pressure may be done in steps or may be done gradually, producing a curve on the chart. The last pound must always be done gradually.

"During these procedures the patient must be observed constantly and, should symptoms return, he must be recompressed at once. This recompression may not have to be back to the original effective pressure but it must be sufficiently high to remove all signs and symptoms. Then, after half an hour's wait at the requisite pressure, decompression is started again.

"Using this method a second recompression should rarely be necessary.

"After treatment is completed the patient should remain under observation for two hours before being transported home and he should be warned to return should symptoms return."

Although the differences from U.S. Navy procedure are striking in several respects, it is noteworthy that the methods described by Griffiths apparently prevented significant lasting damage (with the possible exception of bone necrosis, which may reflect duration of exposure more than any aspect of treatment) in over 1600 men affected by decompression sickness during construction of the Dartford and Blackwall Tunnels. The most trying case in that experience was one in which the patient had to be kept under pressure for nine days, but finally emerged recovered. Clearly, the Griffiths (Dartford) treatment procedures can be employed with good expectations of success at least for exposures of similar nature or where the U.S. Navy tables cannot be used for lack of pressure capability.

Low-Pressure Recompression Using Oxygen

As has been explained, there are good theoretical reasons for attempting to treat decompression sickness by means of oxygen administration at pressures in the neighborhood of 3 atm abs. Workman and Goodman (7) have put such an approach into experimental use at the U.S. Navy Experimental Diving Unit with highly encouraging results in a variety of types of the condition. Their method has now been authorized for testing in a limited number of other naval facilities.

The present forms of the procedure are shown in the accompanying depth-time graphs and require little additional explanation. The shorter treatment (Figure 1) is applied in cases having complete relief of signs and symptoms within 10 minutes. The risk of oxygen poisoning in the longer procedure (Figure 2) is reduced by providing intervals in which air (or 80 per cent helium-20 per cent oxygen) is substituted for oxygen.

The "low-pressure oxygen" procedure is presented here primarily as a reasonable method for use when absence of adequate high-pressure capability prevents application of the standard U.S. Navy treatment tables. In other circumstances, careful application by competent physicians in suitable patients can be considered a matter of medical discretion.

The possibility of better treatment results with shorter therapy in this method inspires hope that it may soon become a fully accepted standard. For the present, several notes of caution must be mentioned:

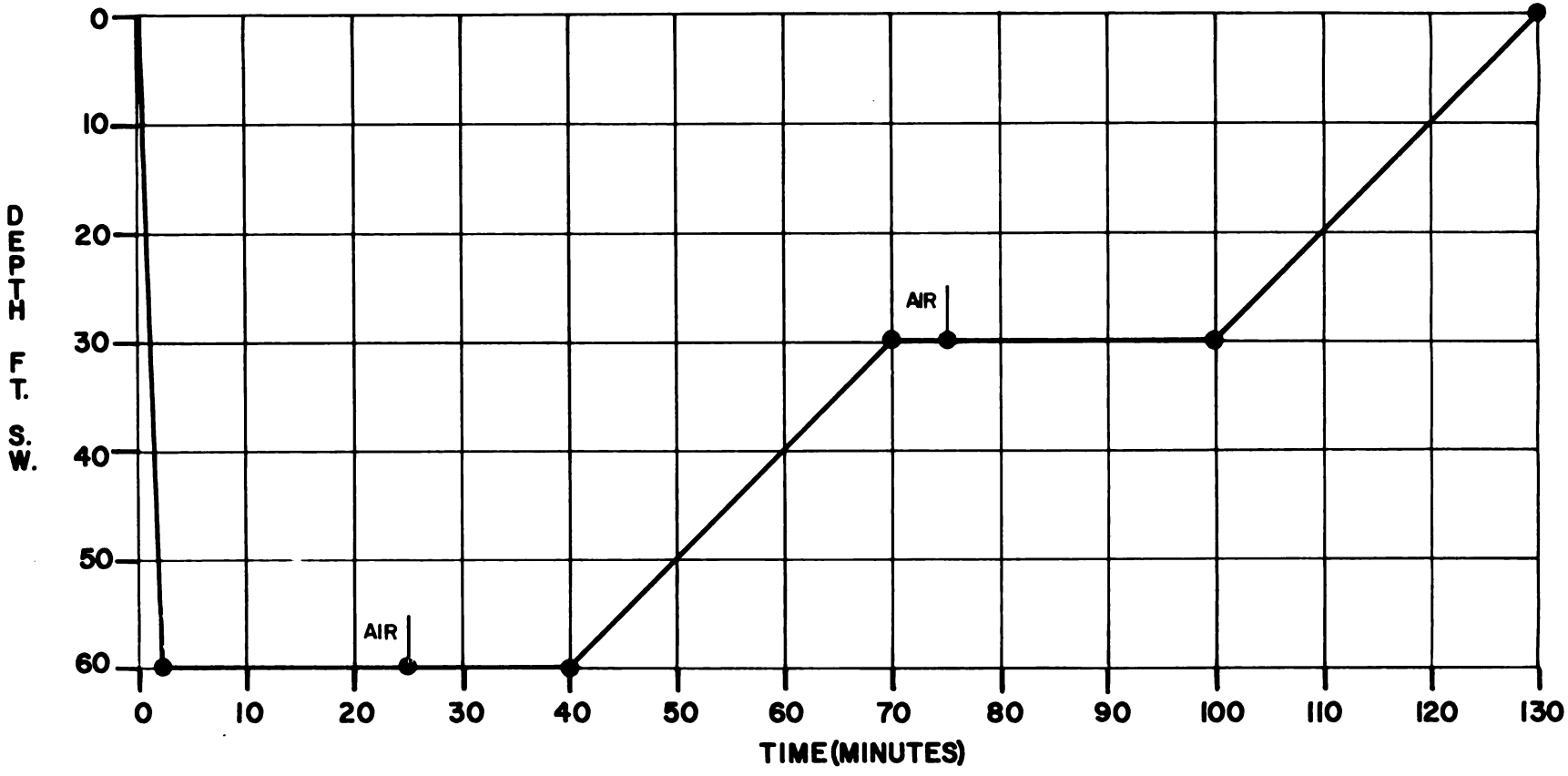

Figure 1.

Low Pressure Recompression Method Using 100% Oxygen
Method to be Used when Relief of Symptoms Occurs within 10 Minutes
at 60 Feet Depth

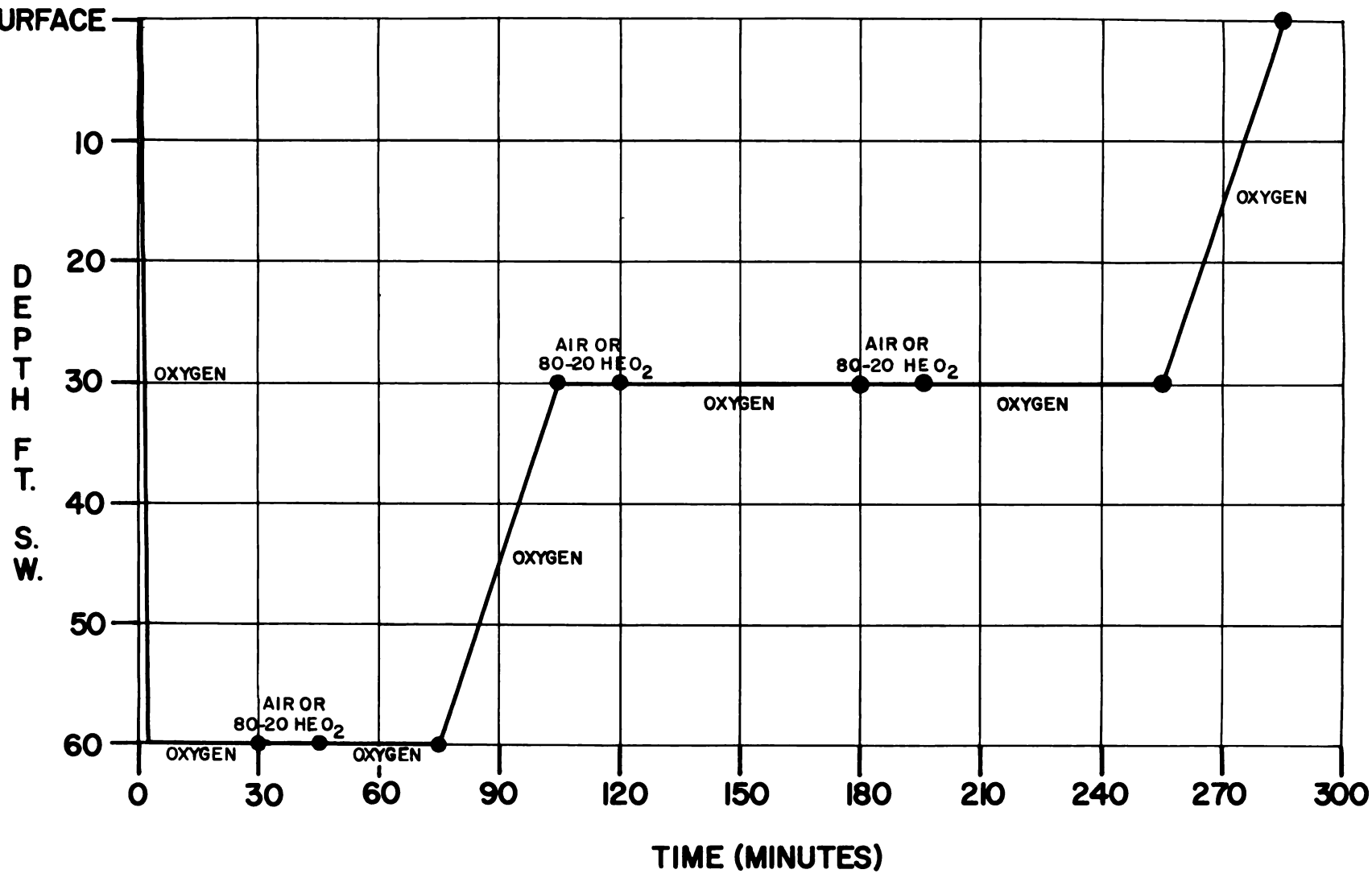

Figure 2.

Low Pressure Recompression Method Using Oxygen
Method to be Used when Relief of Symptoms is not Complete within 10 Minutes
at 60 Feet Depth

1. Experience with low-pressure recompression using oxygen remains limited compared to that with the older methods described. It is considered experimental even within the U.S. Navy, and lack of general acceptance could sometimes become important from a medico-legal standpoint.

2. Some cases of decompression sickness in the "serious" category (and virtually all cases of air embolism) involve disorders sufficiently alarming to demand the most rapid relief that can possibly be provided. If adequate pressure capability exists, waiting for relief at 60 feet would be difficult to justify in such a patient. In the presence of alarming signs or symptoms not relieved en route to 60 feet or within a very short time at that pressure, our present state of knowledge suggests going to higher pressure (breathing air or a suitable mixture) and proceeding with treatment acacording to the standard U.S. Navy treatment tables if pressure capability permits. If the working pressure of the chamber is greater than 60 feet but less than 165 feet, the Dartford procedure for "type 2" cases may be useful here. Current U.S. Navy experimentation includes a procedure for going initially to 165 feet for 30 minutes, then returning to 60 feet to complete decompression according to the longer oxygen table.

3. Use of 100 per cent oxygen is not advisable at 60 feet in all individuals. For example, if the patient has previously shown evidence of unusual susceptibility to oxygen poisoning at that pressure, another approach must be employed. One possibility is administration of oxygen at a lower pressure, but this involves further departure from procedures backed by experience.

ACKNOWLEGEMENTS

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Chapter VIII

OTHER MEDICAL PROBLEMS ASSOCIATED WITH EXPOSURE TO PRESSURE

Robert D. Workman

EFFECTS OF PRESSURE

Increased barometric pressure exposes man to forces and physiological effects not encountered in his normal environment. These not only impose limitations on his activities, but can also give rise to serious risk to life. The safety of personnel so exposed depends on their familiarity with these factors and willingness to respect them.

With each 2-foot increase in depth, the pressure increases by almost 1 psi. Each 33 feet of descent increases the pressure by an additional atmosphere (i.e., 14.7 psi, or 760 mm or 30 in. Hg).

The effects of pressure on man may be divided into two main categories: (1) those that are direct and mechanical, and (2) those that come about because of changes in the partial pressure of respired gases.

Direct Effects of Pressure During Descent

Man can tolerate enormous pressures provided they are uniformly distributed throughout the body. However, when outside pressure exceeds that inside body air spaces, the difference in pressure "squeezes" the involved tissues, causing barotrauma, the earliest signs of which are edema and capillary dilatation. As the pressure difference increases, capillaries rupture and hemorrhage ensues. Pressure in such spaces as the sinuses and the middle ear must be equalized by the admission of additional air on descent, or destructive pressure differences will develop across their walls. Once equalized at a given depth, the air must also vent freely during decrease of pressure.

The Ears

Ear squeeze may be prevented by teaching personnel how to equalize pressure through the Eustachian tubes. Successful maneuvers employed are swallowing, yawning, or exhaling against closed mouth or nostrils. When Eustachian tube blockage prevents the equalization of pressure in the middle ear, varying degrees of barotrauma may affect this structure, causing painful aerotitis with possible rupture of the eardrum. Pain will be experienced in the first few feet of descent. Further descent will increase the pain, stretch the eardrum, and dilate and eventually rupture the blood vessels in the tympanic membrane and lining of the space. Rupture of the eardrum may be caused by as little as 10 feet of unequalized descent. Transudation and hemorrhage may occur in the sinuses, as they do in the middle ear.

Treatment of mild ear damage is syptomatic. Analgesics are indicated when pain is intense. Pain usually subsides gradually. If pain persists, thereby suggesting infection, systemic antibiotics and nasal and systemic vasoconstrictors are indicated to promote drainage and combat infection.

When the tympanic membrane is ruptured, blood drains through the external auditory canal. Retained blood is usually absorbed within a few days with no impairment of hearing. Local application of medication to the ear canal is ordinarily contraindicated, and care should be exercised to prevent water from entering the external auditory canal until healing is complete, which may take days or weeks, depending on the severity of the injury.

The Sinuses

Blockage of the sinus ostia, which results in aerosinusitis (sinus squeeze), can be prevented by avoiding exposure to increased pressure when there is nasal congestion due to allergy or infection. It is accompanied by

painful edema and hemorrhage. Vasoconstrictors may be helpful, as in aerotitis, but if infection develops. as indicated by persistent pain, systemic antibiotics may be required.

The Lungs

As long as one breathes normally and has an ample breathing supply, the lungs and airway will equalize pressure without difficulty. If the breath is held during pressure increase, no difficulty arises until the total volume of air in the lungs is compressed to less than the volume of the rigid airways. Pulmonary congestion, edema, and hemorrhage of lung tissue then occurs in what is generally called thoracic squeeze. Considerable injury can occur in the absence of pain. Hemoptysis may be the first clue to the presence of lung damage caused by squeeze. This form of trauma generally responds well to conservative treatment consisting of general supportive care, prevention of infection, and intermittent positive-pressure inhalation therapy. Nebulization of bronchodilators and aerosols, with postural drainage if hemorrhage or extravasation has been severe, may prove beneficial.

Pulmonary edema may follow the use of breathing apparatus with high inspiratory resistance. In an effort to maintain adequate pulmonary ventilation during moderate activity, high intrapulmonary negative pressure may result in capillary dilatation, transudation, and rupture. Cough and mild dyspnea are presenting symptoms. Roentgenograms of the chest may show patchy pulmonary infiltration, which clears within 24 hours without specific therapy. The need for properly designed and maintained breathing devices is apparent for use under pressure. Trauma to the lungs caused by compression is possible in pressure chambers if an individual becomes apneic during compression, either voluntarily by breath holding, or involuntarily by unconsciousness, laryngeal or tracheal obstruction, convulsions, etc.

The Teeth

Dental pain associated with increase or decrease of pressure is seen only infrequently. Whatever pain does occur is usually in maxillary teeth and associated with maxillary sinus changes evident on X-ray. Freedom from dental pathology has been frequent enough in these cases to suggest that maxillary-sinus squeeze is the causative factor. It is possible that the presence of small gas bubbles in the tooth pulp may permit the soft tissue to be squeezed during pressure increase and to expand during pressure decrease. However, this theory has not been confirmed by dental examination.

The Gastrointestinal Tract

Gas pockets in the gastrointestinal tract do not usually produce symptoms during pressurization, because the bowel walls are non-rigid and equalization is accomplished simply by compression of the gas.

Direct Effects of Pressure During Ascent

Consequences of Excessive Lung Pressure

During the pressure decrease of the chamber, the air contained in body cavities expands. Normally, the air vents freely and there are no difficulties. If breathing is normal during ascent, the expanding lung air is exhaled freely. However, if the breath is held, or there is a localized airway obstruction, the expanding air is retained causing overinflation and overpressurization of the lungs. For example, the air in the lungs at a depth of 66 feet gradually expands to three times its volume during ascent to the surface. The air volume can expand to the point of maximum inspiration safely, assuming the absence of airway obstruction. With further pressure decrease, overexpansion and, later, overpressurization of the lungs result with progressive distention of the alveoli. Such overdistension may be generalized with breath holding or too-slow exhalation, or localized because of partial or complete bronchial obstruction due to bronchial lesions, mucus, or bronchospasm.

Pneumothorax

Distended alveoli or emphysematous blebs may rupture the parietal pleura, causing pneumothorax. Under pressure, this is extremely dangerous, because trapped intrapleural gas expands with continuing pressure decrease, thus causing tension pneumothorax. The rapidity of development can cause sudden respiratory and cardiovascular embarrassment and death from shock and impaired cardiac function. Early diagnosis and prompt treatment with thoracentesis are essential. Recompression will benefit, but the subsequent decompression must be carried out with caution to prevent recurrence of this condition.

Mediastinal Emphysema

With rupture of proximal alveoli, which begins with an intra-pulmonary pressure of 40

to 50 mm Hg, air will dissect along the vessels and bronchi to the mediastinum, causing mediastinal emphysema and pneumopericardium. Gas trapped in the interstitial spaces may expand rapidly with continuing decompression, causing impaired venous return. The symptoms of mediastinal emphysema are pain under the sternum and, in extreme cases, shortness of breath or faintness due to interference with circulation as the result of direct pressure on the heart and large vessels. Treatment in mild cases of mediastinal emphysema is symptomatic. In more severe cases, oxygen inhalation may aid resolution of the trapped gas. For severe, massive mediastinal emphysema, recompression is indicated.

Subcutaneous Emphysema

Subcutaneous emphysema, which may be associated with mediastinal emphysema, is a result of air having been forced into the tissues beneath the skin of the neck extending along the facial planes from the mediastinum. Unless it is extreme, the only symptoms of subcutaneous emphysema are a feeling of fullness in the neck and a change in the sound of the voice. Oxygen breathing will accelerate the absorption of this subcutaneous air.

Air Embolism

The most disastrous result of pulmonary overpressurization is the dissection of alveolar gas into the pulmonary venous system. The gas is carried to the left heart, and then into the systemic circulation, resulting in gas emboli in the coronary, cerebral, and other systemic arterioles. Gas bubbles continue to expand with further decrease of pressure, increasing the severity of clinical signs.

The clinical features of traumatic arterial gas embolism may occur suddenly or be preceded by dizziness, headache, or great anxiety. Unconsciousness, cyanosis, shock, and convulsions follow quickly. The convulsions may be severe and recurrent, and may require heavy sedation. Motor and sensory deficits occur in various degrees and distribution. Death results from coronary or cerebral occlusion with cardiac arrhythmia, respiratory failure, circulatory collapse, and shock. Physical examination may reveal (1) air bubbles in retinal vessels, (2) Liebermeister's sign (a sharply defined area of pallor in the tongue), (3) marbling of the skin, (4) hemoptysis, (5) focal or generalized convulsions, or (6) other neurological abnormalities.

The only effective treatment of cerebral air embolism is recompression to reduce the size of the bubbles, force them into solution, and thus restore effective circulation. A recompression chamber capable of pressurization to 165 feet (6 atm abs) within a few minutes is required. Treatment pending recompression is merely symptomatic. The patient should be kept in the Trendelenberg position, which may help to keep more air bubbles from reaching the brain. Placing the patient on the left side helps to maintain cardiac output, which may be impaired because a large amount of air has decreased the efficiency of the pumping action of the heart. In nonfatal cases, residual paralysis, myocardial necrosis, and other ischemic injuries may occur if recompression is not immediately carried out, and may occur in adequately treated patients if there is a delay in initiating therapy. Although most proposed surgical and medical therapeutic pressure facilities will operate at 2 to 3 atm abs, they must have the capacity to reach 6 atm abs safely, to treat pressure casualties described above, in addition to persons with decompression sickness. Central nervous system decompression sickness is clinically indistinguishable from air embolism. Fortunately, the treatment is the same. One cannot predict when such a casualty will occur, but the hazard exists whenever personnel are exposed to pressure, and proper treatment must therefore be available. Other forms of treatment such as oxygen inhalation, body positioning, cardiac aspiration, and hypothermia without recompression are inadequate and palliative.

Overexpansion of the Stomach and Intestine

While one is exposed to pressure, gas formation may take place within the intestine, or air may be swallowed and trapped in the stomach. During ascent, this trapped gas expands and occasionally causes sufficient discomfort to require stopping until it can be expelled. Continuing ascent may cause marked discomfort and vasovagal effects. The causes of air swallowing, such as chewing gum during pressure exposure, should be avoided.

Indirect Effects of Pressure

Some important conditions result from the increased partial pressures of the gases breathed in a hyperbaric environment. The two most important, nitrogen narcosis and oxygen toxicity, are discussed below.

Nitrogen Narcosis

Air is about 80 per cent nitrogen; therefore, its partial pressure is approximately four-fifths of an atmosphere at sea level. Nitrogen narcosis is a state of light anesthesia occurring at levels probably beginning at 2 atm abs (33 feet), with typical symptoms first noticed between 50 and 100 feet. As the depth increases, the partial pressure of respired nitrogen is increased and the gas directly affects the brain. This produces a sensation similar to that of alcohol intoxication, with confusion and impaired judgment. Susceptibility varies with the individual, but in general the greater the depth the more intense the effect. Rapid descent, which results in carbon dioxide retention owing to inadequate pulmonary ventilation, tends to increase the narcotic level momentarily. A reduction of the narcotic level follows a decrease in retained carbon dioxide with re-establishment of adequate lung ventilation at depth.

Nitrogen narcosis disappears during ascent as nitrogen partial pressure decreases, and no treatment is required. The phenomenon can easily be prevented by avoiding excessive depths. For deep exposures, clarity of thinking and manual dexterity can be maintained by use of helium-oxygen mixtures, rather than air. This may be important during operative procedures, even at a depth of 66 feet (3 atm abs), although repeated exposures to air breathing under pressure indicate some measure of acclimatization to nitrogen effects.

Oxygen Toxicity

The partial pressure of oxygen in air at 1 atm is about 0.2 atm. At a depth of 132 feet, the total pressure is increased to 5 atm and the partial pressure of oxygen to 1 atm. Thus, personnel breathing air at depths greater than 132 feet are exposed to a higher partial pressure of oxygen than encountered in breathing pure oxygen at the surface.

Oxygen poisoning, discussed in detail in Chapter III, is a convulsive phenomenon that can result with the use of compressed oxygen at a depth sufficient to increase the partial pressure of the gas to approximately 2 atm. Symptoms frequently occur without warning. When prodromal symptoms occur they may include nausea, vertigo, muscle twitching, and visual and auditory disturbances. If exposure continues, unconsciousness and convulsions follow. Convulsions may be followed by a period of depression, lasting from 15 minutes to an hour or more and characterized by somnolence or unconsciousness, restlessness, and irrational behavior. Convulsions and other symptoms disappear rapidly when the partial pressure of oxygen is decreased with reduction of pressure. No treatment is required, but airway obstruction and injury must be prevented. It is important in preventing air embolism that chamber pressure not be reduced until the subsidence of the convulsive episode, during which breath holding occurs. Only removal of the oxygen mask is indicated to reduce the inspired oxygen partial pressure and terminate the convulsion. Persistence of the convulsive episode, or marked restlessness and agitation following it, may require parenteral administration of a barbiturate.

RESPIRATORY PROBLEMS UNDER PRESSURE

Hypoxia

Hypoxia is unlikely to occur in the use of pressure chambers ventilated with air in accordance with accepted procedures. Available oxygen in the chamber air at 2 or 3 atm abs is sufficient for several hours of normal personnel oxygen consumption before a deficient oxygen partial pressure would result, even if chamber ventilation had stopped. Hypoxia may result from breathing an oxygen-poor mixture, which may be supplied in error to breathing masks through the chamber gas manifold. This hazard is always present in the use of mixtures of inert gas and oxygen, which may be improperly mixed, analyzed, and labeled.

Carbon Dioxide Excess

Carbon dioxide intoxication may result from inadequate chamber ventilation. Although the oxygen available in the chamber atmosphere may be adequate to sustain life at increased pressure, sufficient carbon dioxide may accumulate in the chamber to cause progressive toxic manifestations in personnel. In the presence of elevated P_{N_2}, P_{O_2}, and air density, respiratory stimulation may not be sufficient for adequate lung ventilation, thus permitting progressive mental confusion and unconsciousness. The air ventilation volumes required are such that a P_{CO_2} exceeding 10 mm Hg is not permitted in the chamber atmosphere.

Added Respiratory Dead Space

Breathing apparatus in which excessive dead space is permitted will result in increased carbon dioxide retention and increased risk of oxygen toxicity with oxygen breathing. Oxygen-

breathing masks with inspiratory bags have caused such difficulty.

Carbon Monoxide Poisoning

Carbon monoxide poisoning may be encountered after ventilation of the chamber with contaminated air from faulty compressors. The site of the air inlet during use is most important when internal-combustion engines are used because exhaust fumes containing carbon monoxide may be drawn into the compressor. In addition, excessive temperature at the cylinder head of the compressor during operation may cause flashing of the lubricating oil and produce carbon monoxide in the air being compressed. Only small quantities of carbon monoxide are tolerable during prolonged exposure. Air containing as little as 100 ppm would produce perceptible effects within four hours. Toxic limits under conditions of increased total pressure and P_{O_2} have not been established, but it is safest to assume that additional toxic effects would be manifest under conditions of increased carbon monoxide partial pressure at depth.

Oxygen is essential in treatment, and where there is unconsciousness, artificial means of resuscitation may be needed. Breathing oxygen at 3 atm abs provides effective tissue oxygenation and accelerates the release of carboxyhemoglobin to offer the most efficient mode of treatment.

Oil-Vapor Contamination of Air Supply

Another risk that occurs with oil-lubricated air compressors is the introduction of oil vapor into the compressed air. Oil fumes give an unpleasant taste to the mixture breathed, and under pressure the concentration may be sufficient to cause pulmonary irritation, cough, and, in extreme cases, pneumonia. Avoidance of excessive oil vapor in compressed air requires careful and regular compressor maintenance, water-and oil-vapor condensors, and an effective filtering system. Some compressors using carbon or Teflon rings are not lubricated with oil and thus avoid this problem.

The amount of oil vapor in the air supply can be measured by passing a known volume of air through a Whatman filter or fiberglas paper filter and determining the weight increase due to absorbed hydrocarbons. Total hydrocarbon indicators are recommended for use with oil-lubricated compressors.

Excessive Resistance to Breathing

The amount of work that a man can do is limited by his ability to ventilate his lungs adequately to maintain oxygen uptake and carbon dioxide elimination. Any breathing apparatus increases the work of breathing to some extent. This is further accentuated by turbulence in the gas flowing through air passages and apparatus, which becomes greater under pressure due to increasing gas density.

Even if the breathing resistance does not cause the work of breathing to exceed a man's limit, respiratory muscles become fatigued and the amount of resistance that can be overcome decreases with time. As the work of breathing increases, the body appears to reach a point at which it will accept a rising arterial carbon dioxide tension, rather than do all the respiratory work necessary to keep the carbon dioxide level normal. In some, it is possible that excessive breathing resistance leads to carbon dioxide intoxication. It is thus important that the easiest breathing apparatus be used and kept in good condition. The work done must be limited to that necessary for adequate alveolar ventilation.

Chapter IX

ANESTHESIA AND RELATED DRUG EFFECTS

John W. Severinghaus

PHYSICS OF ANESTHETIC GASES AND VAPORS UNDER HYPERBARIC CONDITIONS

The partial pressure exerted by a liquid is unaffected by the total barometric pressure above the liquid. With most anesthetic agents, the anesthetic partial pressure in the alveolus approximates 2 per cent of the vapor pressure of the liquid anesthetic at body temperature. This also applies to cyclopropane and nitrous oxide, which are liquids only at high pressure. For this reason, the function of most anesthetic vaporizers tends to be unaltered by a total increase in barometric pressure. For example, a copper kettle produces a saturated vapor of constant partial pressure no matter what the total pressure. The percentage of anesthetic in this total gas decreases as the total pressure increases. When the output of the copper kettle is diluted with other gases, the partial pressure of the anesthetic is reduced by the same proportion as the dilution, exactly as at 1 atm. For example, halothane will issue from a copper kettle at 243 mm Hg at 20°C, irrespective of the total pressure. It will have a concentration of 32 volumes per cent at 1 atm abs and 10.7 volumes per cent at 3 atm abs. In either case, it is necessary to dilute each volume with 32 volumes of O_2 to produce an inspired concentration with a partial pressure of 7.6 mm Hg, which equals 1 per cent at 1 atm abs.

However, gas flowmeters and proportioning devices do not necessarily function normally at high pressures. In general, it is desirable for each user to calibrate his own flowmeters with a spirometer measuring the delivered volume at the pressure at which it is to be used. An example of the calibration of flowmeters is shown in Figure 1 (1). The proportioning devices that divide a flowing stream, permitting some of it to go through a wick vaporizer (e.g., a fluotec vaporizer), deliver approximately the same partial pressure of anesthesia at any total pressure. However, there are small changes, as depicted in Figure 2 (2). As total pressure increases, gas flow is likely to become more turbulent, and the resistances of small orifices will therefore increase more than those of large orifices. Rotameter flowmeters are apt to read slightly high, in terms of volumes delivered at ambient pressure, because the denser gas, even if not turbulent, exerts a greater force on the bottom of the float.

High pressures have no effect on such characteristics as heat of vaporization, critical pressure, and temperature of anesthetic agents. However, air at high pressure conducts slightly more heat. Therefore, the heat required to vaporize the liquids can be more easily supplied by the flowing gas and the surrounding air, resulting in a smaller decrease in the temperature of the vaporizer.

Reducing Valves

A reducing valve generally contains a spring-loaded diaphragm on one side of which the ambient air pressure plus the spring pressure are used to balance the high pressure on the other side. A reducing valve within a hyperbaric chamber will therefore provide gas whose pressure will rise in parallel with the chamber pressure. For example, a valve that delivers 50 psi at 1 atm abs (i.e., 50 psig) will deliver 50 psi above chamber pressure, or about 95 psig, at 3 atm abs when the valve is within the chamber. This is true only if the high-pressure supply to the reducing valve exceeds the downstream pressure sufficiently to operate the reducing valve. One-stage regulators ordinarily fail to regulate when the supply pressure approaches the delivery pressure. Two-stage regulators, which have two pressure drops within them, require a higher upstream operating pressure. Consequently, if reducing valves are used within pressure chambers, the line delivering compressed gas to the reducing valve ordinarily must operate at pressures exceeding 100 psi

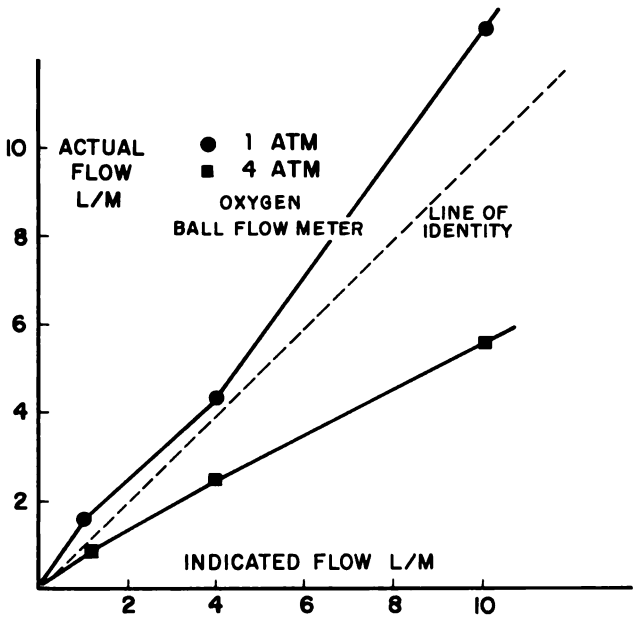

Figure 1.

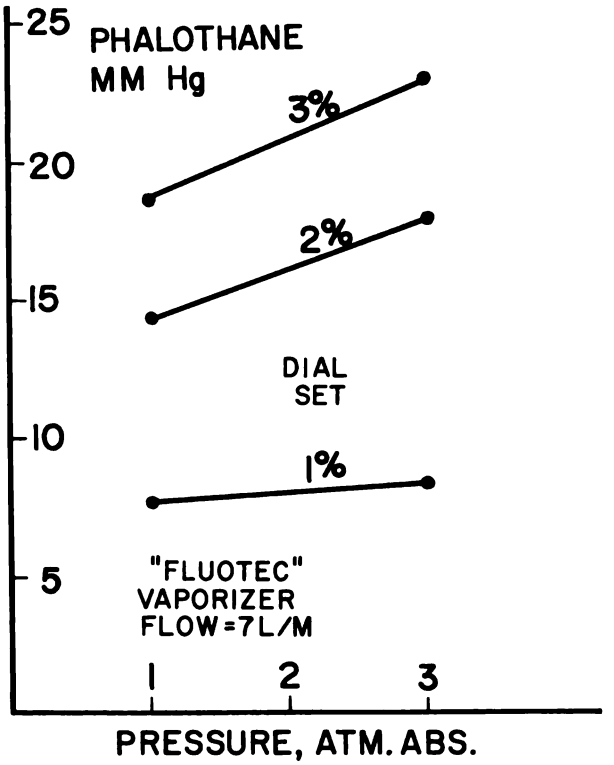

Figure 2.

but preferably not equaling cylinder pressure. It will thus be desirable for an outside regulator to reduce the cylinder pressure to some intermediate value, e.g., 250 psig. If, on the other hand, the reducing valve is entirely outside the hyperbaric chamber, as pressure is raised within the chamber the apparent delivery pressure will fall and the flow through orifices or flowmeters will decrease.

Needle Valves

The increased turbulence at high pressures increases resistance. For any particular setting of the valve, at a constant pressure gradient across the valve, a slightly smaller volume of gas measured at ambient pressure will be delivered through a needle valve at high pressure. Example: A needle valve delivers 4 liters per minute from a 50 psig line at 1 atm abs. At 3 atm abs and the same setting, again with 50 psig (95 psi abs), a flow of 3.5 liters per minute is delivered. This volume would be 10.5 liters per minute at 1 atm abs.

Volume Meters

Physical-displacement volume meters, such as spirometers and dry-test gasmeters, function normally at high pressures. No changes occur in calibration of the Monaghan meters.

Pneumotachygraph

Both screen and tube pheumotachygraphs will indicate higher than actual flows at high pressure if calibrated at 1 atm abs because of increased density and turbulence.

Pressure Transducers

Pressure-measuring devices, such as strain gauges, always determine the difference between inlet fluid or gas pressure and ambient pressure, unless they are differential-pressure gauges with separate outlets for the back side of the gauge. In some strain gauges, the back side of the gauge is connected to a tube, which is incorporated in the lead wire with its open end near the plug. This is true of many of the Statham transducers. If one were to incorporate the lead wire in a gasket leading through the wall of a hyperbaric chamber, the back side of the transducer would be exposed to outside pressure and the front side of the transducer would be exposed to chamber pressure. The gauge could be destroyed when the chamber pressure was elevated. This may be prevented by removing the back side tubing from the lead wire before passing the wire

through the chamber wall. Transducers should be open to the air or to the pressure being measured during compression and decompression. If the front chamber of a transducer is left closed, the diaphragm may rupture as ambient pressure changes on the back side.

Gas Mixtures

For some physiological purposes, mixtures of oxygen, CO_2, and helium are prepared. This can be done with flowmeters at ambient pressure if the three gases are delivered into the chamber separately. However, it is sometimes desirable to have prepared a known mixture in advance for use in the chamber. The physiological effect of inspiring 2 per cent CO_2 at 3 atm abs slightly exceeds the effect of inspiring 6 per cent CO_2 at 1 atm abs (.02 x [2280 - 47] = 44.7, whereas .06 x [760 - 47] = 42.8).

PHYSIOLOGICAL EFFECTS OF HIGH PRESSURE

This section contains only a sketch of effects relevant to anesthesia; more complete information is presented in preceding chapters.

Central Nervous System

Inert-Gas Narcosis

Nitrogen is only 5 per cent as soluble in lipids as N_2O, and has an anesthetic activity in keeping with this solubility. The lowest alveolar partial pressure of N_2 at which narcotic effects are evident is approximately 1400 mm Hg (2.4 atm abs or 21 psig) equivalent to breathing 10 per cent N_2O at sea level (3). In terms of judgement disturbance, this may be roughly equated to the effect of 10 mg of morphine. At this point it must be assumed that judgement is altered, euphoria may exist, laughing spells occur in some individuals, and the easy way out of difficult situations may be chosen when reason would suggest harder ways. At a nitrogen partial pressure (P_{N_2}) of about 2000 mm Hg, many individuals will experience tingling, distortion of sounds, feelings of disassociation, and distinctly bad judgment. Consciousness is gradually reduced as pressure rises, with complete loss of consciousness occurring at a P_{N_2} of between 5000 and 10,000 mm Hg (average approximately 7000 mm Hg). Therefore, personnel working at 3 atm abs probably should either breathe helium-oxygen mixtures or work under the direction of someone outside the chamber who makes or approves all decisions.

Oxygen Toxicity

Acute oxygen toxicity at 3 atm abs or above is first manifested as cerebral irritability, progressing to convulsions. Many of the symptoms resemble those of acute hypoxia or ischemia. Anesthesia may obscure the signs of central toxicity, but it has not yet been shown in man whether the damage itself is lessened by the anesthesia. Pentobarbital and urethane were shown to have no effect on the brain-tissue P_{O_2} of rats at 4 atm abs of O_2 (4). Many anesthetics reduce the pulmonary damage and forestall convulsions but, at least in rats, with barbiturates, brain damage resulted in a spastic paralysis (5) after nonlethal exposures to OHP. Halothane reduces cerebral vascular resistance (12), even when the reduced oxygen consumption is considered, so that tissue oxygen tension is elevated by halothane (13). Pupillary dilitation, sweating, increasing pulse rate, pallor, and, in awake man, nausea appear to be early signs of toxicity. In animals, hypothermia affords some protection against oxygen toxicity. Sympathetic blockade protects at least against pulmonary damage in animals. Thus it is not yet possible to state whether deep anesthesia and anticonvulsant drugs are desirable for prolonged exposure to high oxygen pressure, or whether they merely mask the evidence that tissue damage is occurring. Hyperventilation is believed to protect the brain, and hypercapnia leads to earlier convulsions. Dead space and CO_2 rebreathing are to be avoided. High CO_2 may mask signs but cause death.

Regulation of Respiration

Breathing of air at 3 atm abs has little effect on regulation of respiration. The alveolar oxygen tension is elevated to about 420 mm Hg, which has a slight depressant effect on the carotid body; the alveolar P_{CO_2} might therefore be expected to rise slightly. The end tidal P_{CO_2} of 40 mm Hg will, of course, approximate 2 per cent rather than 6 per cent. The administration of pure oxygen at 3 atm abs results in some hyperventilation and a small reduction in CO_2, but at the same time oxygen at this pressure exerts a rather pronounced depressant effect on the chemoreceptors such that the CO_2 response slope is decreased by 50 per cent (6). The degree of depression due to oxygen is greater than might be expected from chemoreceptor depression alone, and suggests an additional direct depression possibly at the respiratory center, similar to the known

central depressant effect of hypoxia. The effect of anesthetics on the regulation of respiration under hyperbaric conditions has not been studied but, in view of the above, it may be anticipated that elevation of P_{CO_2} will be, if changed at all, more pronounced at comparable anesthetic levels.

Gas Exchange and Blood Gases

The removal of CO_2 at normal arterial tensions requires the ventilation of alveoli with a normal volume of gas, no matter what the ambient pressure. This means that the number of molecules of oxygen, or other gas, inspired per minute increases in proportion to the total pressure and the fractional concentration of CO_2 in the expired gas decreases as pressure rises. A nonrebreathing system in this circumstance becomes more and more wasteful of oxygen or other gases. For example, to provide ventilation of 6 liters per minute at 3 atm abs requires 18 liters per minute of flow when the volume is expressed at 1 atm abs. The distribution of inspired gas presumably will become less uniform due to the higher density and increased turbulence at high pressures. Carbon dioxide transport is impaired because the blood leaving tissues is more nearly saturated with oxygen, leaving fewer hemoglobin sites available as hydrogen-ion buffers. Therefore, tissue P_{CO_2} tends to rise because more of the metabolically produced CO_2 must be carried in physical solution instead of as bicarbonate. In awake normal man at 3.5 atm abs P_{O_2}, brain tissue P_{CO_2} tends to rise about 3 mm (7). Arterial P_{CO_2} falls about 3 mm due to an increase in ventilation produced by the tissue P_{CO_2} rise at the medullary respiratory CO_2 chemoreceptor. At 3 atm abs of oxygen, the total oxygen demand of many of the tissues can be supplied from dissolved oxygen. This is not true of the myocardium or liver and is usually not true of the brain, unless cerebral blood flow is increased by elevating arterial P_{CO_2}. An increase in arterial P_{CO_2} greatly increases oxygen toxicity by increasing the oxygen tension in the cerebral tissue. The total gas pressure in venous blood and in tissue tends to be much lower than the ambient pressure when pure oxygen is being breathed. For this reason, sudden decompression does not ordinarily produce bends in patients breathing oxygen, whereas it may in personnel breathing air (see Chapter IV).

Pulmonary Effects

Mechanics

At high pressures the increased density results in an increased work of breathing, owing principally to increased resistance resulting from increased turbulence. Patients with destructive airway disease (emphysema) are particularly subject to airway closure due to the pressure gradients during expiration. Partial obstructions in anesthetic equipment will become more significant at high pressures, in the presence of turbulence particularly.

Airway Irritation

In many animals the tracheobronchial tree is rapidly damaged by less than 24 hours of oxygen at 1 to 2 atm abs, resulting in hyperemic mucosa, secretions, and atelectasis. Anesthetic agents are said to diminish this irritation (8). There are no reports of pulmonary damage following brief OHP in man, and it is probable that central-nervous-system toxicity precedes pulmonary damage at a P_{O_2} of 2 to 3 atm abs. The role of atropine and other drying agents in preventing and treating secretions arising in the tracheobronchial tree has not been evaluated.

Atelectasis, Surfactant Denaturation

Oxygen toxicity results in airway closure owing to plugs of the increased secretions. When alveoli contain no insoluble inert gas, such as N_2, collapse of the lung units rapidly follows. Terminally, a loss of alveolar surfactant has been demonstrated (9). At present it is thought that this loss of surfactant is secondary to the atelectasis and tissue irritation.

Dead Space

Whether the anatomic dead space may be decreased by bronchoconstriction, edema, or both resulting from oxygen damage has not been determined. Oxygen breathing produces a difference between the end tidal and arterial P_{CO_2} (10), a difference further increased by anesthesia itself for unknown reasons. Thus, it is not usually possible to assess accurately the arterial P_{CO_2} by observing the end tidal gas P_{CO_2}.

Diffusion

Diffusion will be significantly affected if the capillary bed exposed to gas is decreased or if

atelectasis occurs as a result of breathing oxygen. Patients with limited diffusing capacity may be treated with hyperbaric oxygen. If diffusing capacity were so severely limited that hyperbaric oxygen were required for oxygenation, it is conceivable that significant diffusion gradients would exist for CO_2 and anesthetic agents.

Humidity

There is some evidence that adequate humidification protects the airway against oxygen toxicity. In anesthetic systems, high humidity is most easily provided in a to-and-fro rebreathing system where the canister is near the airway, allowing the warmth of the CO_2 absorbent to prevent condensation of the respired gases. Humidification is most difficult in the nonrebreathing system. Usually, saturation of the inspired air can be provided only if both heat and nebulization are used.

Intestinal Gas

If the patient swallows air, or the anesthetist forces gas into the stomach at high pressure, the expansion of this gas during decompression may disrupt an abdominal incision, cause great pain or regurgitation, force the diaphragm up limiting respiration, or even rupture the stomach or bowel. This can be largely avoided by passing a nasogastric tube at the beginning of anesthesia.

Pneumothorax

If thoracic surgery is done, or if a pneumothorax has been induced or has been present, gas in the pleural space will expand during decompression, collapsing the lung, resulting in anoxia, and producing tension pneumothorax. Great care needs to be exerted to keep drain tubing open during decompression.

Miscellaneous Effects Relevant to Anesthesia

Heart rate and cardiac output are depressed, probably by homeostatic mechanisms. The heart itself appears to be much more tolerant than the central nervous system to prolonged hyperoxia. In unanesthetized man at the onset of oxygen toxicity, the bradycardia is reversed and systolic and diastolic pressures rise (11). In dogs under Dial Urethane on the other hand, a fall of blood pressure was reported to be the earliest sign of O_2 toxicity at 4 atm abs. Cortisone augments oxygen toxicity. With the onset of oxygen toxicity, the sympatho-adrenal system is stimulated.

PHARMACOLOGY

Very little is known about the effect of drugs on oxygen toxicity and about the effect of OHP on the action of drugs.

Premedication

Tranquilizing drugs, narcotics, and anticonvulsants are believed to forestall the onset of hyperoxic convulsions. Atropine might be considered undesirable, and it has been suggested that it has an increased potency. A hypothetical reason for this might be that the decreased cardiac output and decreased heart rate that occur during hyperbaric oxygenation are homeostatically induced by increased vagal tone. Atropine might therefore appear to have a more pronounced effect operating in a condition of increased vagal tone.

Anesthetic Agents

Nitrous Oxide, Xenon, and Ethylene

One early use suggested for high-pressure chambers was to permit the N_2O partial pressure to be elevated sufficiently to produce general anesthesia without other drugs. In dogs, the alveolar P_{N_2O} required to prevent movement on painful stimulus is approximately 1400 mm Hg (Eger, personal communication). At this point, dogs continue to exhibit some spontaneous movements and are not relaxed. Man appears to reach general anesthesia at a P_{N_2O} of about 800 mm Hg according to Faulconer and others (17). A serious difficulty with its use under these conditions is the enormous amount of dissolved gas in tissues (approximately 100 liters at 3 atm) that gives rise to bubble formation during decompression, unless there is prolonged washout of the N_2O at high pressure accomplished first. If decompression were begun with N_2O present, its outflow into the alveoli would displace O_2 and potentially result in a profound "diffusion anoxia." Furthermore, any air-containing cavities within the body will increase their volume owing to the inward diffusion of N_2O, causing severe distension during decompression. For example, pulmonary bullae expand and might rupture, and intestinal gas volume may multiply many fold, interfering with abdominal surgery and with respiration and venous return from the lower extremities. Because N_2O requires a high partial pressure, its use in any concentration reduces the oxygen tension delivered to the patient, or requires a higher total ambient pressure to which the attendant personnel are exposed. It has essentially no

use in attempting to achieve hyperbaric oxygenation. Similar reasoning applies to xenon and ethylene.

Cyclopropane

The flammability of cyclopropane in OHP has not been determined. Until this information becomes available, it should be assumed that the hazard is greatly increased.

Halothane

Halothane appears to be used most commonly under hyperbaric oxygenation. The partial pressure required for anesthesia is unaffected by the total barometric pressure. In man, the minimal concentration for anesthesia is an alveolar partial pressure of about 6 mm Hg. Respiratory arrest occurs at about 15 mm Hg and circulatory arrest between 18 and 20 mm Hg. The partial pressure delivered by most vaporizers stays approximately constant, as described in the first section of this chapter, when pressure is increased. It has been determined that halothane decomposition is not accelerated by exposure to OHP, with or without copper (14). It is now recognized that many so-called "inert" anesthetic agents are in fact metabolized to some degree by the liver. Whether this metabolism is altered and whether the toxic actions of some anesthetics on tissues is affected in the presence of OHP have not been investigated. Halothane is flammable under hyperbaric conditions if mixed with O_2 and N_2O in certain concentrations. The manufacturers state that there appears to be no risk at pressures below 2.5 atm abs, if no N_2O is used and cautery is avoided. The safety of the use of cautery with halothane and OHP remains to be evaluated.

Ether

The potency of ether is such that the alveolar partial pressure rarely enters the explosive range, and the propagation of flame in ether is so poor that explosions appear not to occur within the lung, although they do occur in anesthetic equipment. Because ether may find some use under hyperbaric-oxygen conditions, its explosive limits and its propagation of flame under high pressures should be determined.

Other Halogenated Anesthetics

It has not yet been determined whether agents that are poorly flammable at 1 atm of oxygen become more flammable at high pressures. In general, the lower limit of flammability partial pressure rises as the total pressure rises, although not necessarily proportionally. Thus an agent such as fluroxene (Fluoromar), which is not flammable below 28 mm Hg at 1 atm abs, will not be flammable at higher total pressures in the clinically useful concentration range (8 to 24 mm Hg). The possibility of oxidation of these anesthetics by high-pressure oxygen has not been investigated. Trichloroethylene, since it reacts with CO_2-absorbent lime, requires partial or nonrebreathing circuits. It is not flammable and may be used at high pressures by techniques used at 1 atm abs. Methoxyflurane vapor is not flammable at any oxygen pressure currently contemplated for use. It should function normally under hyperbaric conditions.

Intravenous Anesthetics

In view of OHP, the added difficulty and hazards associated with inhalation anesthesia in intravenously administered drugs may be especially useful. The only apparent impact of OHP on barbiturates, narcotics, and tranquilizers is the added respiratory depression associated with high oxygen pressures (see "Regulation of Respiration," page 117). Because rising P_{CO_2}, which accompanies respiratory depression results in increased cerebral blood flow and earlier onset of cerebral oxygen toxicity, artificial hyperventilation may be desirable, particularly with combinations of narcotic barbiturates and OHP. The usual disadvantages of using these agents for general anesthesia, viz., irreversibility and long action, apply equally in OHP. Some inhalation agents can be given intravenously, permitting the rapid elimination of such agents through the lungs. Of the clinically used anesthetics, ether, halothane, Fluroxene, and methoxyflurane exist as liquids. Intravenous injection of these pure liquid anesthetics may result in hemolysis, but if they are suitably diluted or mixed with solvents, this hazard may be overcome. Ether is the most water-soluble of the above agents and intravenous ether anesthesia has been demonstrated as feasible. The highest ether concentration that will not produce hemolysis is 5 volumes per cent. Such a mixture must be given at a rate of 1 to 2 liters per hour to produce anesthesia. Obviously, this limits its usefulness to short procedures.

Some of the remaining anesthetics are highly soluble in fat. This fact has been used as the basis for increasing the concentration

of intravenous methoxyflurane. The use of an emulsion of lecithin as the vehicle produced anesthesia with about 500 ml per hour. If similar mixtures of the other anesthetics could be prepared, they too might be suitable for use in a pressure chamber. These volatile compounds may form bubbles when given intravenously. If nitrogen or other inert gas is present at high tension in the blood, it will diffuse into the bubble and greatly delay its reabsorption. This will not occur after a few minutes of oxygen breathing.

Relaxants

All the usual relaxants have been used with OHP and appear to function normally. When paralyzing doses of relaxants are used, the motor signs of a convulsion are masked, this being an additional reason for monitoring the EEG.

Vasopressors

The blood needs of tissues during hyperbaric oxygenation are reduced because of the increased dissolved oxygen. Accordingly, peripheral resistance is increased by local homeostatic vasoconstriction, and there is a resulting decrease in cardiac output with a normal pressure. This does not imply, however, that the venous reservoirs are constricted, inasmuch as the predominant central circulatory control causes a reduction in cardiac output, cardiac slowing, and venous distention. Although it has not been adequately documented, one may expect to find increased sensitivity to small doses of vasopressers under these conditions. The circulatory system is apparently quite tolerant of oxygen toxicity and continues to function apparently normally, well beyond the point of convulsions and apnea.

Treatment of Acidosis

When hyperbaric oxygenation is used in association with a period of total ischemia, as in some forms of cardiac surgery, metabolic acidosis has been treated by some workers with either prior or subsequent administration of Tris buffer (Tham). In the rat, alkalization with Tris buffer confers some protection against pulmonary oxygen toxicity (15).

TECHNIQUES OF ANESTHESIA

Inflowing Gases

In most chambers, the only gas supplied to the anesthetist is oxygen, which generally enters the chamber at a pressure sufficiently above the maximum chamber pressure to ensure flow at all times. As chamber pressure changes, the flow from a needle valve within the chamber will therefore change. This can be avoided by having a second reducing valve within the chamber, an arrangement that increases the safety by maintaining constant flow. In some chambers, it is also possible to mix CO_2, helium, and nitrogen with the oxygen administered. These comments about inside reducing valves apply to these gases. If small cylinders of gases are to be brought within the chamber, the increased hazard of doing this should be recongized. Occasionally a cylinder that is tipped over will be broken at the neck. It will not only jet propel itself around the chamber, but will deliver a large volume of gas in a very short time into the closed chamber. This will change the composition of the atmosphere and the pressure within the chamber very rapidly. If cylinders are to be brought into high-pressure chambers, the pressure system of the tank probably should include some form of automatic control that could handle a sudden introduction of high-pressure gas into the chamber, and they should be firmly mounted and anchored.

Vaporizers

The function and calibration of vaporizers were discussed in the first section. If the vaporizer is of the type in which oxygen bubbles are forced through a liquid anesthetic, it should be recognized that as pressure is raised the liquid anesthetic will be forced back up the inlet gas line. This can be avoided either by maintaining a very low flow of oxygen through the vaporizer as pressure is raised, by not filling the vaporizer until after pressure is reached, or, as in some anesthetic machines, by opening the inflowing line to the atmosphere where it is not connected to allow oxygen to flow through the anesthetic. Some vaporizers have a one-way valve on the exit to prevent backflow of anesthetic gases during compression of the rebreathing bag. This check valve will prevent the equilibration of pressure within the vaporizer and might result in collapse of some portion of the vaporizer, which can be avoided either by opening the filling port or by having a low flow of oxygen through the vaporizer during compression. Most surface vaporizers used on older anesthesia machines close off both inflow and outflow ports when the anesthetic is not being used. If these ports are, in fact, sealed tight the danger of

implosion again exists with these vaporizers during compression.

For some installations, the vaporizer may be mounted outside the chamber. This is most likely to be the case in animal experimental work where the anesthetist is outside the chamber. No suitable vaporizers are commercially available for this purpose. The simplest method for devising such a vaporizer in the absence of commercially available devices is to use a motor-driven syringe to force the liquid anesthetic into the inflowing oxygen line, in which a copper or metal gauze has been placed to provide a surface from which the anesthetic may be vaporized. The calculations involved in determining the liquid inflow are as follows for halothane: 1 ml of halothane becomes 230 ml of vapor at room temperature and 1 atm abs. Therefore, each 0.20 ml of halothane delivered per minute will form 46 ml of vapor, which will result in halothane partial pressure of 7 mm Hg in the flowing gas if the flow is 5 liters per minute at any ambient pressure. The concentration would be 0.9 per cent at 1 atm abs and 0.31 per cent at 3 atm abs.

Breathing Systems

When the anesthetist is in the chamber, any of the ordinary rebreathing systems or nonrebreathing systems may be used. Space limitations may suggest bringing only a CO_2 absorber, valve system, vaporizer, and rebreathing bag into the chamber. It is generally considered desirable to vent the overflow gases to the outside; this is best accomplished by leading the overflow gases through tubing to the immediate vicinity of the chamber exhaust valve, making very certain that there is no possibility of a direct connection between the outflow orifice and the rebreathing system. Any direct connection of this sort with the possibility of becoming occluded could result in a very serious application of negative pressure to the pulmonary tree of the patient. For example, it should be considered dangerous to connect the tail of the rebreathing bag to a valve that vents to the outside if this valve is triggered by filling of the rebreathing bag. This is not a fail-safe device, because if the valve were to be jammed in the open position, the lower pressure of outside would be applied to the patient's airway. Many overflow valves in anesthetic systems do not have a direct connection to permit collection of overflow gases. When this is the case, it is possible to connect overflow tubing to the tail of the rebreathing bag and lead it to the vicinity of the outflow from the chamber. It is not necessary to be concerned that all the overflow anesthetic gas be vented to the outside, because the amount given to a patient is far from sufficient to cause any physiological symptoms in the remaining personnel in even the smallest chamber.

It is sometimes desirable to collect expired gases for analysis of concentration. The instantaneous tracheal-gas concentration can be monitored by allowing flow through a small tube connected to the endotracheal tube to outside, provided this flow is carefully limited to be incapable of suddenly evacuating the rebreathing system and the patient's lungs. If mixed expired gas from a nonrebreathing system is to be collected and transferred to the outside, the arrangement should avoid any possibility of sudden application of negative pressure to the subject.

Ventilators

Compressed-Air-Powered Ventilators

Most compressed-air-powered ventilators require 50 psi above ambient pressure. If they are in operated chambers, the supply-line pressure must be elevated accordingly. If a reducing valve is mounted in the chamber and set to 50 psi it can be supplied with a higher pressure from oxygen or air outside the chamber. When the ventilators are used with pure oxygen at 3 atm abs, extraordinary caution must be taken to ensure that no oil or other combustible substance is present in the portion of the respirator exposed to high oxygen. It should be noted that, at 3 atm abs, a respirator in general will require three times as much air or oxygen to operate it as at 1 atm abs. Provided the supply pressure is kept at the usual 50 psi above ambient, presently available respirators will function adequately, although with somewhat lower peak flow rates. If the respirator is used to ventilate the patient with oxygen, it is generally desirable to vent it near the gas exhaust from the chamber, to avoid raising the oxygen pressure in the chamber. If the ventilator is used to compress a rebreathing bellows or bag, it is generally better to power the ventilator with compressed air to obviate collecting the exhausted O_2 and conducting it to the outside.

Motor-Driven Ventilators

No motor has yet been approved by the NFPA for use in a hyperbaric chamber. Three

alternative approaches are available. (1) The motor may be enclosed in a relatively airtight box through which nitrogen is purged and bled to the vicinity of the chamber exhaust. If this approach is taken, it would probably be desirable to interlock the motor power supply with the nitrogen supply. (2) The motor may be mounted outside the chamber with a pressure-sealed shaft entering the chamber to operate the ventilator piston. This approach has been used particularly in small chambers for animal experiments. (3) The electric motor may be replaced with an air motor, which can be operated by the vacuum available by venting the downstream side of the motor to the outside air or to a suction line for operation before compression. Two types of air motors are available, one a turbine* that is said to be available with a speed governor; the other of the reciprocating-piston type. Devices such as windshield-wiper motors can be used and develop a great deal of force at 3 atm abs.

Inflatable Devices

All devices containing trapped air, such as endotracheal cuffs, Foley catheters, masks, tourniquets, and blood pumps, will collapse when the pressure is elevated; if they are filled at high pressure they will expand or explode when pressure is dropped. It is therefore necessary to fill them with water before entering the hyperbaric stage or to make certain that they are open to ambient pressure, except when pressure is stable in the chamber.

Intravenous Fluids

Several problems arise with the administration of fluids in chambers. In some chambers, the ceiling is so low that insufficient gravitational force can be obtained and it may be necessary to use a blood-pump set to administer ordinary fluids. Many of the intravenous-administration bottles have an air inlet, which bubbles air below the surface of the water. When pressure is decreased after hyperbaric chamber use, the water in the bottle will be forced out through the air inlet. If the air inlet has a one-way valve, as many of them do, the bottle may explode or infuse its contents rapidly into the patient and then air-embolize the patient. This is particularly true if the bottle is nearly empty when decompression occurs. Some manufacturers provide intravenous fluid with air-inlet pipes extending above the fluid level. These are probably more suitable for use in the chamber. The drip chambers also contain trapped air, which will vanish on compression, and which if replaced during the high-pressure stage, may air-embolize the subject on decompression. The amount of air involved is generally small but is undesirable. Blood, whether in bottles or in plastic bags, appears to offer no problems in the hyperbaric chamber, inasmuch as all blood sets have air inlets that extend above the level of the blood when the bottle is inverted.

* Harvard Apparatus Company, Dover, Mass.

Bottles and Vials

When a rubber-capped vial is pressured, the rubber cap may be forced into the bottle. If there is a needle hole through the top, the application of ambient pressure may force contaminants into the bottle. If the vial is upside down, on decompression the contents may be forced out through needle holes in the cap. Plastic bottles will collapse when the pressure rises and, if closed tight with a high pressure, may explode on decompression. Bottles of liquid anesthetic contain a vapor phase above them, and, if the bottle is opened within the chamber and then closed tight, it may explode on decompression. Vials and ampules have been known to implode during compression. An imploding ampule will not do any harm if it is kept in a box or kit somewhere to avoid flying glass.

Suction

Suction devices for surgical purposes and for anesthetists' use in caring for the airway need careful consideration. The safest procedure is to use a pressure-reducing suction regulator in the chamber. These devices are inexpensive and available from supply houses and are customarily used to limit the negative pressure in suction devices used for airway cleansing. If these are mounted in the chamber, the negative pressure applied to the end of a suction catheter will be independent of the total chamber pressure. If, however, a suction line flows directly to outside the chamber, it is possible very easily to tear off mucosa with the suction tube. Having the suction regulator in the chamber requires that the trap bottle also be in the chamber. Some traps have ping-pong ball floats to prevent overfilling. These will implode at high pressure, but that can be avoided by drilling a small hole in the ball and

injecting enough melted wax to keep the punctured portion up. It is not possible to allow the fluid contents to flow through the suction regulator, nor is it possible to allow the fluid contents to flow through a needle valve to the outside, because the tiny orifice needed for regulating the gas flow will become completely occluded with blood or mucus. The only alternative is to have a pressurized suction trap and suction regulator outside the chamber. In this case, the suction regulator should be coupled in such a way that it regulates suction with reference to chamber pressure rather than to atmospheric pressure. This is a much more difficult technique and requires a suction trap that is heavily constructed to avoid explosion.

Syringes

Sterile syringes, both plastic and glass, are often supplied with a cap over the tip. When pressure is applied there will be a tendency for outside air to be forced into the dead space of the syringe. In the case of a glass syringe, this may force contamination down the barrel into the syringe. The plastic syringes are not apt to leak around the plunger, but the negative pressure may make it more difficult to remove the plastic protecting tip, and, unless care is taken, contamination from the fingers may enter the dead space at the moment of removing the tip. Arterial blood samples taken in syringes will foam if the syringe is taken to atmospheric pressure from high pressure (see "Blood-Gas Monitoring," page 125).

Combined Extracorporeal Circulation and/or Hypothermia and Hyperbaric Oxygenation

Special problems relevant to anesthesia in OHP with these added interventions have not been explored sufficiently to form a report. If anesthesia is given into an oxygenator, the same considerations apply as when given to the lungs. However, one possible reason for combining OHP and bypass may be to decrease the size of the oxygenator. The enormous driving pressure of oxygen may permit full oxygenation with gas exposure, which would not permit equilibration with the anesthetic, such that a higher concentration of anesthetic might be required when administered via an oxygenator. When the patient has been reasonably well equilibrated with the anesthetic, a normal concentration will be required.

Hypothermia increases the solubility of gases. The association with OHP would not appear to alter the ordinary clinical anesthetic management further. Because hypothermia protects against oxygen toxicity, there may be a demand for anesthesia to permit hypothermia and thus permit prolonged therapy at high oxygen pressures.

Apparatus for cooling and warming and for pumping blood is generally electrical and contains motors and switches. It may require considerable redesign to permit its safe use in hyperbaric chambers.

MONITORING

Electroencephalogram

In addition to the usual monitoring during anesthesia, it is generally desirable to monitor EEG because of the possibility of oxygen convulsions. Spike discharges may be visible before any physical signs of convulsive activity occur; in the paralyzed subject the EEG is the only evidence of convulsive activity. The conventional monitoring of EEG is by oscilloscope. It is not yet known what pressures various cathode-ray oscilloscope tubes will withstand before imploding. Several anesthesiologists have used a commercially available explosion-proof unit.* This unit is enclosed in a steel cylinder capable of withstanding explosive forces of 600 psi. It is reasoned that even if the cathode-ray tube imploded (the case is not completely hermetically sealed), the damage would be confined. It may be possible to vent the inside of this case to the outside to prevent exposing the oscilloscope to high pressure.

No official statement of the safety of this procedure has been received from the manufacturers or from any testing agency. Furthermore, some authorities believe the high voltage needed to operate an oscilloscope is itself a fire hazard. An alternative is to mount the oscilloscope outside a window in the chamber, visible to the anesthetist, or to project optically a directly written EEG onto a screen in the chamber. Another alternative is to use other forms of monitoring the EEG. Unfortunately, no other suitable devices are commercially available.

Electrocardiogram

Comments are largely the same as for EEG. There are battery-operated monitors that dis-

* Electronics for Medicine OMR-1

play the EKG on a moving meter arm and by audible signals, which are useful under hyperbaric conditions. The large vacuum tubes in some amplifiers may be subject to implosion. Direct-writing recorders all have motors not yet approved. If it is necessary to mount a direct-writing recorder in a chamber, the motor housing should be purged with nitrogen.

Esophageal Stethoscope

The lumen of the catheter should be open when pressure is changing, because the balloon would greatly expand during decompression if air were trapped in the line.

Ventilation Monitoring

Direct volume-indicating devices (Ventimeter) operate normally in the chamber. Other devices, such as pneumotachygraph, the Wright flowmeter and the Monaghan flowmeter, must be recalibrated (see "Volume Meters," page 116).

Temperature Monitoring

The ordinary battery-operated thermister-probe meters are not affected by high pressure.

Alveolar-Gas Sampling

Carbon Dioxide

The infrared CO_2 analyzer is most easily used by mounting the head and amplifier outside the chamber, bleeding a continuous flow of gas at approximately 50 to 100 ml per minute from the endotracheal tube via a very small tube through the wall of the chamber into the microcatheter sampling cell, and allowing it to vent from the sample cell to the atmosphere. Calibration should be done by sending compressed gas through the cell at the same rate, also venting to the atmosphere, rather than using suction. The restricting orifice should be as near the endotracheal tube as possible for several reasons: it results in expansion of the gas at the restricting orifice with consequent higher flow rate through the remainder of the tubing, and the decompression avoids water-vapor condensation in the tubing. One suitable approach is to use an adjustable-needle orifice, such as is used with the nitrogen meter. If the anesthetist wishes to moniter CO_2 concentration, a remote meter in the chamber can be connected to the amplifier outside the chamber.

Oxygen

The same general considerations apply to oxygen monitoring. Generally, there will be no need for oxygen monitoring, because the patient is breathing pure oxygen diluted only by a small amount of anesthetic. If mixtures are used, however, the oxygen may be continually monitored by bleeding a small sample through a paramagnetic oxygen analyzer outside the chamber. These devices respond to the partial pressure of oxygen, and none is available commercially with scales exceeding 760 mm Hg. High pressure may implode the glass dumbells in the sensing element. Oxygen electrodes may be used in the chamber and several battery-operated devices are available with direct-reading meters suitable for monitoring the oxygen partial pressure in a rebreathing system.

Anesthetic Concentration Monitoring

Infrared and ultraviolet analyzers are available for most anesthetics in use. In general, the same considerations apply as described for use of the infrared CO_2 analyzer. The concentration read on the outside must be converted to partial pressure in the chamber by multiplying by the total chamber pressure.

Blood-Gas Monitoring

Arterial blood will contain total gas pressures approximately equal to chamber pressure and will foam if taken through a lock, or delivered through tubing, to the outside. It will be no longer possible to measure the oxygen or CO_2 pressure in the blood, because the gases will come out into the foam. Venous blood, on the other hand, generally does not become fully saturated with oxygen at 3 atm abs and has no nitrogen if the subject is breathing pure oxygen. It may be taken outside the chamber for analysis. It is possible to bring most of the blood-gas monitors into the chamber. Oxygen electrodes have no air vent for the space under the oxygen-permeable membrane. If an air bubble is trapped in the electrode, this will collapse and perhaps tear the membrane during compression, and may cause the membrane to explode during decompression. Care should be used, therefore, to assemble the electrode without any air bubbles. This can best be accomplished by boiling the electrolyte before assembling it and holding the oxygen electrode with the active end down in a pool of electrolyte while assem-

bling the membrane. The CO_2 electrode has an air vent and is not affected by alteration in total pressure. Calibration gases in general will have to be especially prepared for use in the hyperbaric chamber, because the concentration roughly equivalent to arterial tension at 3 atm abs is 2 per cent CO_2. With most instruments, an oxygen electrode can be made to read on scale even at 3 atm of oxygen. If not, the simplest modification is to use 2 or 3 layers of polyethylene membrane over the electrode. Generally, pH electrodes have a sealed air space within the glass electrode. In the case of bulb electrodes, the very thin glass membrane may implode under hyperbaric conditions. This is less likely to occur with capillary electrodes. Some electrodes have a relatively loose seal and may be destroyed by the hyperbaric conditions. Tar may be forced down into the electrode or the electrolyte may be forced out as pressure changes. Calomel reference electrodes likewise often have a sealed air bubble within them, which should either be vented to the air or filled completely with saturated KCl. The amplifiers for electrodes require checking for implodable tubes and spark-producing switches. One instrument* is known to be suitable in hyperbaric chambers, because it consists entirely of solid-state materials.

Clinical Monitoring

The anesthetists should, in addition to the usual clinical monitoring, monitor the following: (1) the ear drums, for congestion due to failure of the eustachian tubes to permit entrance of air into the middle ear if prophylactic needle myringotomy has not been done; (2) Chvostek's sign, as an early sign of oxygen toxicity; (3) pulse and blood pressure, because a rise in either is an early sign of oxygen toxicity, at least in awake man; and (4) the pupils, for dilatation as a sign of oxygen toxicity.

Tissue P_{O_2} has been monitored with the polyethylene-covered-needle oxygen microelectrode by Brummelkamp and Boerema (16) as an indication of effectiveness of hyperbaric O_2. In clostridial infection, for example, at 3 atm O_2, P_{O_2} rose to 330 mm Hg in the infected area. The electrode may locally compress tissue blood supply where inserted, so absolute values are difficult to document.

* Beckman Spinco

methemoglobinemia. Planning should permit the anesthetist to have available in the chamber all the equipment and drugs that might be necessary. Chambers with service locks permit equipment to be passed in during hyperbaric treatment. For example, the above list of possible emergencies might suggest a need for large intravenous needles, dextran, resuscitation equipment for newborn infants, and methylene blue.

Because haste so often leads to accidents in anesthesia, full-scale rehearsals of the handling of likely emergencies should be considered.

Postoperative Checks

Postoperative checks should include testing of peripheral vision, which may be impaired owing to oxygen toxicity, and checking the eardrums for hemorrhage and rupture.

SPECIAL ANESTHETIC PROBLEMS IN DISEASE

Rapid transfer of patients to a hyperbaric-oxygen environment may be indicated in a variety of conditions, including shock, pulmonary embolism with planned embolectomy, signs of fetal distress during labor, carbon monoxide or cyanide poisoning, or massive methemoglobinemia. Planning should permit the anesthetist to have available in the chamber all the equipment and drugs that might be necessary. Chambers with service locks permit equipment to be passed in during hyperbaric treatment. For example, the above list of possible emergencies might suggest a need for large intravenous needles, dextran, resuscitation equipment for newborn infants, and methylene blue.

Because haste so often leads to accidents in anesthesia, full-scale rehersals of the handling of likely emergencies should be considered.

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Chapter X

THE HYPERBARIC FACILITY

James V. Harrington and John C. Carter

The hyperbaric-oxygenation facility is a complex of pressure vessels and systems that permit the administration of oxygen to human or animal subjects under pressures varying between 1 atmosphere and the number of atmospheres for which the facility has been designed.

Facilities can range in size and complexity from a small, bench-type unit capable of accomodating an experimental animal to a large unit capable of taking a human patient and a team of doctors, nurses and attendants sufficient to perform the most complex surgical operations, or to accommodate a number of medical patients and attendants.

The equipment required will consist of one or more pressure vessels suitably connected, a pressurization system, a piping system, air-flow controls, electrical systems as necessary, including in some cases automatic-control systems, temperature and humidity regulation, and noise-suppression systems.

The pressurization system may be of several types or combinations. The possibilities include: (1) high-pressure supply, (2) low-pressure compressors and (3) cryogenic supply. Such systems will be described under appropriate headings.

Because of the wide diversity in physical size and end use of these facilities, ranging from the animal experiment on the one hand to the full surgical team and patient at the other extreme, each of the above-mentioned aspects of the facility will bear different emphasis in the different designs. It will be the purpose of this discussion to provide an adequate description of each part of the hyperbaric complex without becoming involved to the point that the pros and cons of the approach of various equipment vendors are discussed.

THE PRESSURE VESSEL

The pressure vessel is a container that can be made gas tight with the structural strength necessary to contain the internal pressure for which it was designed. In the case of an experimental-animal chamber it is large enough to contain the animal that is the subject of the experiment; whereas, in the case of an operating or treatment room, it must be sufficiently large to contain the human patient, his attendants, doctors and nurses, and all of the equipment that will normally be used during the course of the planned operation.

For most efficient structural design, pressure vessels are normally made either cylindrical or spherical in shape. Cylindrical vessels usually have dished heads at the ends, although some are made with flat heads, either stayed or unstayed on the ends.

Vessels for this service may be either of the single or multiple compartment varieties, depending upon the service they are to render. Small animal chambers are most often of the single lock variety. As many as three or four locks may be found in human surgical facilities.

Any facility designed to contain human beings using gas other than pure oxygen under pressure must have at least one compartment designed for a minimum working pressure of 6 atm abs. This is mandatory to permit the treatment of cases of decompression sickness and air embolism in accordance with standard practices developed and established by the U.S. Navy (6).

Design of the chamber should conform throughout to Section VIII of the American Society of Mechanical Engineers (ASME) Boiler and Pressure Vessel Code—Unfired Pressure Vessels. In addition, all rules established by the state and municipality in which the facility is located concerning pressure vessels, air tanks, etc., should be complied with (1).

The ASME Code clearly specifies both the method of design and the method for testing pressure vessels for structural integrity. The standard hydrostatic pressure prescribed for this test is one and a half times the design

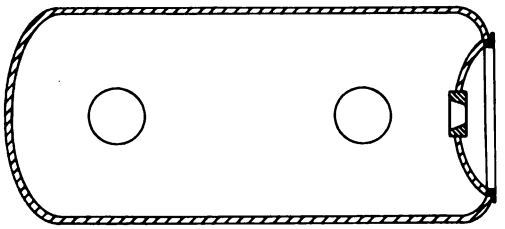

Figure 1. Typical Cross Section through Single Lock Pressure Chamber

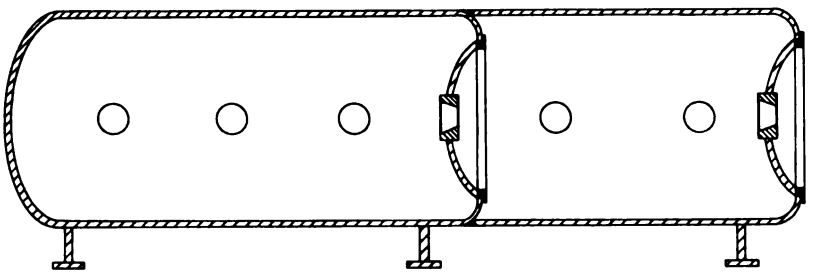

Figure 2. Typical Cross Section through Two Lock Pressure Chamber

working pressure. In addition, the qualification of service inspectors and periods and methods of inspection are also specified, based upon the type of service and method of installation.

Doors

All pressure vessels will be fitted with access doors to admit the subject or the patient and attendants. All doors should preferably seal with pressure to prevent accidental opening when the chamber is pressurized. Where the pressure can be applied to an opening from either side, such as an internal door between locks, two doors should be provided, one closing from each chamber, so that the door on the side seeing the higher pressure is sealed with it. Doors should be designed for the maximum allowable working pressure of the compartment they seal. Where double doors are used between chambers, care should be exercised to insure that the space between them does not form an additional chamber that could contain a pressure different than the chambers on the two sides of the doors.

Windows

Chambers should be fitted with viewing ports so that the attendant outside the chamber can see the activities on the inside.

Service Lock

For facilities intending to contain an attendant, a service lock or pass-through should be provided so that equipment and instruments may be passed into the chamber without using the main access locks. This lock should be large enough to pass the largest piece of equipment requiring handling during the course of the operation.

It should be recognized that the service lock must be equalized so that the pressure inside the lock is equal to the pressure on the opening side before the door is opened.

Vents must be provided on both sides of the lock for this purpose, and the designer of the facility should consider whether or not an interlock between the venting mechanism and the door-opening mechanism should be provided. Unlocking a door with pressure behind it can cause a serious accident.

For large chambers containing a number of people, a floor structure on the inside is usually necessary. It is suggested that these floors be designed for a minimum load of 60 pounds per square foot and be covered with a suitable non-sparking, conductive material as used in operating rooms.

Penetrations and Fittings

During the design of the chamber, a certain number of penetrations for piping and electrical services will be required. Additional capped-off penetrations should be provided. During the life of the chamber additional services may be required, and the difficulty of providing additional penetrations should be avoided if possible. Interior mounting pads and clips should be provided in the design of the vessel to eliminate the need for welding them on later. Since no general recommendation as to the number needed can be made, it is suggested that the designer consider their need in the light of the intended use of each facility that he is designing.

Foundations

Chambers other than portable types must be provided with suitable foundations to support and position them rigidly. Movement of sources of gas and air for its operation. The

air supply should be designed to accomplish the following objectives:

1. Have a ready source of supply to bring the chambers from atmospheric pressure to the operating pressure in a reasonable time period.

2. Have a sufficient high-pressure reserve supply to be able to bring the recompression treatment chamber from atmospheric pressure to the operating pressure a minimum of two times without additional charging of the high-pressure supply.

3. Have gas capacity and flow capability sufficient to pressurize the recompression treatment compartment from 1 atm to 6 atm abs in a period not to exceed two minutes.

4. Have adequate capacity to ventilate the chamber continuously at a satisfactory rate at the allowable working pressure. For the analysis of "satisfactory rate," the following criteria apply, and the method of calculating satisfactory rate is described in Appendix B.

   a. The total partial pressure of CO_2 under any circumstances should not exceed 10 mm Hg.

   b. The total percentage of oxygen in the atmosphere should not exceed 25 per cent under any circumstances.

   c. The concentration of other atmospheric contaminants should be maintained to values that will be set from time to time.

AIR AND GAS SYSTEMS

The safety and comfort of the occupants of this complex depend upon adequate and reliable chambers after they are operating is undesirable because this imposes undue stress and strain on rigid connections such as pipe, conduit, and other interconnections. Small experimental chambers are usually supplied with foundations or stands that are adequate for the purpose.

Large chamber complexes consisting of a series of units should be mounted on integral saddles, and the substructure preferably should be a block of concrete or other monolithic structure.

When chambers are connected together through tunnel-type openings, care should be exercised in their assembly to insure that axial alignment is properly attained by leveling and shimming before connections are made. Any attempt to force alignment by flange bolts or other means may unduly overload the connection.

In the design of the foundation, it should be determined whether subsequent hydrostatic test of the facility will be necessary. Very often the weight of the test liquid will exceed the weight of the facility, and a foundation designed for the facility might not support it during hydrostatic testing.

Adequate space between saddles and between the tank and adjoining structures should be left so that proper inspection of all parts of the tank can be made as required.

Foundations for rotating and reciprocating machinery should be sound, and vibration isolated from the building structure where operation of such machinery would cause disturbances to the surrounding area.

Ventilation Systems

Facilities may be provided either with a low-pressure air supply system or a high-pressure air supply system, or both. All piping for the air supply systems shall conform to Section 2, Industrial Gas and Air Piping Systems, Code for Pressure Piping ASA B 31.1-1955, and to other state and local codes and ordinances as applicable (2).

Breathable-Gas Supply Systems

Piping systems for oxygen, air, and at least one other breathable gas should be provided in each chamber. All system components containing oxygen or other therapeutic gases should conform to applicable National Fire Protection Association specifications (3, 4, and 5).

Low-Pressure Air Supply Using Compressor

A low-pressure air supply system for a chamber complex may consist of one or more continuously running low-pressure compressors. The system should consist of the compressor fitted with inlet air filters, mufflers as required, pressure-regulating valves and relief valves, and aftercoolers fitted with water separators and traps.

Low-pressure compressors are available in either oil-lubricated or non-oil-lubricated types. Since oil in the atmosphere of a hyperbaric facility is undesirable from many standpoints, non-oil-lubricated compressors are highly preferable for this system. These may be reciprocating compressors with either Teflon or carbon ring packing and piston rings. Certain rotary compressors are also suitable.

If oil-lubricated compressors are used, they must be equipped with adequate oil separators to remove oil from the atmosphere

before it is supplied to the chamber. It is also suggested that a total hydrocarbon indicator be provided for the detection of oil vapor in the air supply line. Units of this type, operating on the principle of hydrogen flame ionization of carbon atoms, are available. Sensitivity of these units depends upon sample concentrations and sampling conditions, but is in the region of 0-4 parts per million full scale.

The discharge of the compressor should generally be led to a volume tank or air receiver. From the receiver, the air will go to the chamber via suitable flow-and pressure-control systems that will be described later.

Since compressed air cannot be stored efficiently at low pressure, no storage other than the very small amount in the receiver is provided, and this system must run continuously to ventilate the chamber. A check valve should be provided between this system and the chamber complex to prevent backflow in the event of compressor failure.

The quality of air supplied to the chamber should conform to reasonable and acceptable safety standards.

High-Pressure Air Supply

Where a low-pressure system is used, a backup high-pressure system should be provided. High pressure in this instance would generally be from 2250 to 3000 psi. Sufficient air should be stored in cylinders to supply at least two fillings of the recompression portion of the chamber complex under maximum operating pressure.

The air for the high-pressure system may be purchased in cylinders and piped to the system, or a compressor capable of delivering a suitable volume of air at the maximum storage pressure of the system may be used to charge a system of storage banks. In the event that a compressor is installed, it should be fitted with an intake filter and muffler, an after-cooler, and a discharge separator and filter to remove vapors and water from the compressor discharge. The compressor, if installed, will discharge air to the storage flasks. As in the case of low-pressure compressors, a non-oil-lubricated type is preferred.

Air from the high-pressure system shall be carried to the mains supplying the chamber complex through a reducing station that reduces the pressure to a suitable value above maximum chamber operating pressure. Two reducing valves in parallel, each capable of full system flow, are desirable in this system.

Low-Pressure Cryogenic Air Supply

A system using liquid oxygen and liquid nitrogen may be used for supplying air to the chamber. Such a system would consist of cryogenic containers of each gas, a suitable vaporizer for each gas, and a suitable mixing system consisting of valves designed to mix the required volumes of each gas in the proportions of normal air at adequate pressure to be supplied to the chamber.

Sensors capable of measuring the concentration or partial pressure of oxygen should be installed in the chamber, and a suitable display of this value should be provided.

Visual and audible alarms should be installed to provide a warning if the oxygen partial pressure deviates from acceptable values.

Chamber Air Supply

All air should be supplied to the chamber service valves at a pressure suitably above the maximum chamber operating pressure. Each lock of the chamber must have a manually operable supply valve, operated from the outside for pressurizing and ventilating lock. In addition, controls can be provided to permit the personnel in the chamber to control the rate of pressurization and the ventilation rate. In this instance, the controls inside the chamber must be capable of being overridden by controls on the outside.

If automatic pressure and ventilation control is to be provided, provision should be made for manual override of the automatic system. Manual override in this instance should not depend upon an outside power source for its operation.

Chamber exhaust should be constructed for operation in a manner similar to the inlet system, permitting exhaust control by either attendants on the outside, by personnel on the inside, or by automatic means. Final manual control must be outside. The exhaust from all of the locks of the chamber complex should be run to a vent stack. Each lock of the chamber must be supplied with a relief valve in conformance with the ASME code requirments. In addition, drain valves in the bottom of each chamber are suggested as an aid in wash down. Drains should be run to a suitable sump. Metal damage-control plugs should be installed in the drain outlets, and be in place at all times except when washing down.

Oxygen Systems

In small animal chambers of the single-lock variety, the sole source of gas may be either a series of oxygen cylinders or a cryogenic oxygen source capable of bringing a chamber to maximum operating pressure and maintaining the pressure for a period of time sufficient to conduct the experiment as planned. Unless the system is also adequate to provide chamber ventilation, chemical CO_2 removal is required. Among others, lithium hydroxide is a good material for the removal of CO_2. One must remember that combustible materials, even the fur on an experimental animal, will burn extremely vigorously in an oxygen atmosphere. Extreme care must be taken when operating with an oxygen atmosphere to eliminate both sources of ignition and combustible materials. Oil and grease must be excluded in all forms.

Chambers for human occupancy that are used with an atmosphere of 100 per cent oxygen are available. They will accomodate one patient or experimental animal, but will not also admit an attendant, due to small size.

Several special problems exist with these units, among which are increased fire hazard, oxygen toxicity of the subject, and the subject's isolation in a manner that the attending physician cannot lay hands on him. Operators of such chambers must be alert to these factors and be thoroughly trained in the prevention of accidents as well as in the handling of any emergencies that might arise.

The most meticulous care should be taken to eliminate combustible materials, spark sources, and all other fire initiators and propagators from a chamber used with a human patient.

Chamber Gas Supply

Chambers for human occupancy should be provided with auxiliary sources of gas supply. Piping systems inside each chamber compartment should be supplied for pure oxygen for therapeutic use and for at least one other breathable gas such as a nitrogen-oxygen or helium-oxygen mixture. The systems should be sized to permit a flow at least equal to the maximum demand of all persons occupying the chamber inspiring simultaneously. In addition, piping systems for compressed air must be provided with multiple outlets in each chamber. Properly fitting masks with demand regulators should be supplied in each compartment in quantities sufficient for the total number of occupants, and spares should be provided in reasonable quanitity. The special breathing-air piping system is provided for use in the event of fire-contaminated atmosphere and for general purposes. Adequate flow rate through reducing valves to insure adequate supply to the compartments should be assured in the design phase. Placing reducing valves inside the chamber provides automatic regulation of over-ambient pressure.

Noise Control

When air is moving at high velocity, the turbulence in the air results in high noise levels. Suitable mufflers installed at points of large air-pressure drops may be required to reduce the noise. For inlets that are intermittent or used for pressurization only, higher noise levels may be permitted. For continuous operation, a noise level below 80 decibels should be attained.

CHAMBER HEATING AND COOLING

Chamber air should be cooled either by precooling incoming air or by recirculation through cooling coils to maintain both a comfortable temperature and reasonable (50 to 60 per cent) humidity.

Air flow in surgical chambers should enter at the top and exhaust at the bottom to facilitate the removal of dust and toxic agents that are usually heavier than the air itself.

Standard principles of heat transfer and air conditioning apply to the heating, cooling, and humidity problems associated with this type of chamber.

It is recommended that only chilled water, hot water, or low-pressure steam be used in coils entering the compartments. The use of other refrigerants or fluids could introduce toxic contaminants in the event of a leak.

ELECTRICAL EQUIPMENT

The electrical supply available to the chamber complex should be an ungrounded system, with all enclosures and conduits grounded to the chamber as specified in Sections 517-1 to 517-9 of the National Electrical Code (7).

An approved type of ground detector including visual indication and an audible alarm should be installed to monitor continually the circuits in the chamber. The reason for an ungrounded system is that if one wire of the circuit becomes grounded for any reason, no

short-circuit sparks will result. The circuitry on the outside of the chamber can be a grounded system.

All external electrical installations shall conform to the applicable sections of the Standard of the National Board of Fire Underwriters for Electrical Wiring and Apparatus as recommended by the National Fire Protection Association (7). All equipment supplied shall be in accordance with NEMA standards.

Switches, Receptacles, and Fittings

The National Electrical Code (7) specifies that equipment in atmospheres containing combustible anesthetics falls in group C of Section 500-2 of the code, and it is necessary that equipment be approved not only for the class of location but also for the specific gas, vapor, or dust that will be present.

All equipment approved as group C in manufacturers' catalogs is expected to be explosion proof, i.e., an internal explosion will not propagate outside the equipment when containing a stoichiometric mixture of the specified gas and air at atmospheric pressure. However, no known equipment has been tested for explosion-proof service when containing stoichiometric mixtures of gases at pressures of several atmospheres, and therefore the existing approvals for group-C equipment cannot be said to ensure the same degree of safety under hyperbaric circumstances that they ensure at atmospheric pressure.

It is hoped that manufacturers and vendors of electrical equipment for operating-room use will expeditiously have their equipment approved for service in hyperbaric facilities.

Lighting Systems

The chambers should have a general lighting system capable of supplying 60-100 footcandles of general illuminations. All lighting fixtures should be permanently installed and of the enclosed type. All general lighting shall be controlled from outside the chamber.

In addition, an operating-room light or lights as required should be permanently installed in the chamber, it should be pressure tested to a minimum of two times the intended maximum working pressure both in the off and full-on positions. In this connection, a small pressure test facility for components would be a useful adjunct to a large chamber.

Service Systems

Service systems as required should be supplied. These should consist of at least two power receptacles in the locks of any peripheral chambers and a minimum of six receptacles in the surgical chamber. Suction devices as required should be installed. All electrical service systems installed in the chambers should conform to class C of the National Electrical Code, insofar as possible.

In the installation of service systems, one should consider providing additional penetrations in the chamber walls in excess of those to be used immediately, since both additional instrument and power leads, including shielded instrument leads, may be required with time and the experience of use. All switches and circuit breakers should be mounted outside the chamber, and each circuit should have a pilot light to indicate whether it is on or off.

Power System

Large facilities for human occupancy in which air is continuously circulated for ventilation purposes will require substantial amounts of electrical power. This power will be required to run compressors, cooling water pumps, and for the operation of all other running equipment. An adequate amount of standby power should be available to maintain minimum necessary services in the event of failure of the primary supply. It is frequently unwise to rely upon the hospital's emergency power system for this purpose.

AUTOMATIC VENTILATION- AND PRESSURE-CONTROL SYSTEM

In addition to manual controls, which must in any event be installed, it is possible to control both the pressure and the flow through these chambers automatically. Either solenoid-operated, motor-operated, or pneumatically operated valves may be installed in the inlet and exhaust lines to the chamber.

As one example, potentiometers suitably connected can serve as valve controls. The potentiometer on the inlet valve can be used to preset a valve opening or rate of flow for ventilation. A potentiometer in conjunction with a slide-wire pressure transducer and control amplifier can be used to provide pressure control at the exhaust valve.

In this system, a constant rate of flow through the inlet valve is maintained, and the exhaust valve opening is controlled so as to maintain constant pressure in the chamber.

A chart recorder suitable for working with a conductive curve and curve follower may be provided as a substitute for the potentiometer

manual pressure control. In this manner, a pressure-versus-time control of the chamber pressure may be established, and a preprogrammed decompression schedule may be run.

COMMUNICATION SYSTEMS

At least two separate means of communication should be provided for each lock of the chamber to permit communication between locks, and from any lock to the outside of the chamber. The first method should consist of a wall-mounted sound-powered telephone. One such telephone should be installed in each lock of the chamber complex.

In addition, for more general and more effective use, a nonsparking, non-flammable amplified system of voice communication should be provided with speaker microphones in a convenient location to provide voice communication among various stations as required. Communication from inside to outside should not require the speaker to go to a microphone or operate any controls.

It is also desirable to install a telephone connected to the local telephone exchange in the chamber for outside communication, but the dialing and switching must be done outside of the chamber, with only the handset installed in the compartment. Explosion-proof handsets are available and should be used.

Electrical, electronic, or pneumatic failure in any part of the control system should cause all valves to fail safe in the closed position so as to maintain whatever pressure is in the chamber at the time.

INSTRUMENTATION

Each lock of the chamber complex shall be fitted with a caisson gage (or equivalent) covering the full range of pressure for which the chamber has been designed. Each operating station should be supplied with a pressure gage to read the pressure in the chamber.

Internal and external indications of temperature and humidity in the chambers should be provided.

For those chamber complexes using oil-lubricated compressors, a total hydrocarbons indicator should be provided to monitor the air supply.

Depending upon the uses to which the chamber is to be put, a wide variety of other instruments may be used within it from time to time. Reliable information is needed about the behavior of many standard instruments under pressure higher than atmospheric, and it will be necessary to obtain information as to their performance, reliability, and safety of use under hyperbaric conditions. In general, this information should be obtained from other researchers working in the field or from the manufacturer, having ascertained that the problems of hyperbaric use are appreciated by him.

An approved type of ground detector including visual indication and an audible alarm should be provided to detect any grounding of the ungrounded portion of the electrical system.

Pressure gages for monitoring the pressure in air banks, air receiver, and supply lines should be supplied. Monitoring of the discharge temperature of compressors should also be done. All such permanent system monitors should be outside the chamber, convenient to the chamber operator.

PAINTING

Only fire-resistant paint should be used inside the chamber complex. Painting should be kept to a minimum, and before applying new paint the old paint should be removed. After painting, be sure to thoroughly ventilate the chamber and allow sufficient drying time to eliminate volatile vapors.

List of Applicable Codes and Useful References

1. Section VIII - Unfired Pressure Vessels
   ASME Boiler and Pressure Vessel Code
   American Society of Mechanical Engineers
   29 West Thirty-Ninth Street
   New York 18, N.Y.
2. American Standard - Code for Pressure Piping
   American Society of Mechanical Engineers
3. NFPA No. 56 - Code for the Use of Flammable Anesthetics
   National Fire Protection Association
   60 Batterymarch Street
   Boston 10, Mass.
4. NFPA No. 565 - Non-Flammable Medical Gas Systems, 1962
   National Fire Protection Association
5. NFPA No. 566 - Standard for Bulk Oxygen Systems at Consumer Sites
   National Fire Protection Association
6. U.S. Navy Diving Manual
   Superintendent of Documents
   U.S. Government Printing Office
   Washington 25, D.C.
7. NFPA No. 70 - National Electrical Code
   National Fire Protection Association

Chapter XI

MAINTENANCE OF HYPERBARIC FACILITIES

John C. Carter

GENERAL

The maintenance program for any hyperbaric facility must emphasize three important areas: personal safety to the occupants of the hyperbaric chamber, cleaning techniques that are compatible both with chamber materials and with the hospital standards of cleanliness, and reliable performance of pressurization components and controls.

Safety in a hyperbaric facility involves factors not normally encountered where high environmental air pressures are not present. Some materials normally considered that are only slightly combustible will exhibit increased flammability when exposed to the greater oxygen partial pressure in pressurized air, and moderately flammable materials may burn violently under the same conditions. Fire becomes increasingly hazardous and is complicated by the inability of persons within the hyperbaric chamber to leave quickly in case of a fire. It is therefore imperative that highly combustible materials be prohibited for use in hyperbaric chambers, and moderately combustible materials be kept to an absolute minimum. All firefighting and emergency breathing apparatus must be maintained in good repair and ready for immediate use at all times.

HOUSEKEEPING

1. The cleanliness and sanitary standards of the hospital shall be maintained in the hyperbaric chamber.
2. Cleaning techniques and materials shall be approved by the manufacturer of the chamber to ensure compatibility with materials of construction and to prevent accidental damage to devices requiring special handling.
3. The hyperbaric chamber must not be used for storage of hospital equipment and supplies, except as specifically approved by the hospital administrators.
4. Materials that are flammable or can produce toxic vapors must not be used within the hyperbaric chamber.
5. Sealable glass containers must not be approved for use or storage within the chamber, and such approved sealable containers for use or storage must be capable of withstanding safely the maximum chamber pressure when applied both externally with the inside depressurized, and internally with the outside at ambient pressure.

PREVENTIVE MAINTENANCE

Painting

Surfaces of steel chambers must be protected from rusting. All paints used within the chamber must be selected for minimum flammability and for non-toxic properties. Paint should not be applied too heavily nor allowed to build up, as a heavy buildup increases the hazard of flammability. Painted surfaces shall be well sanded down before additional paint is applied.

Filters and Absorbers

Air filters and moisture absorbers shall be cleaned or replaced regularly and scheduled to maintain suitable atmosphere control within the chamber. Schedules should be based on conservative intervals as determined from initial system performance and as recommended by the manufacturer.

Windows

Where transparent plastic is used for windows or enclosures, care must be exerted at all times to prevent scratching the surface. Keep all abrasive substances away from the plastic and avoid wiping it when dry. Use only non-abrasive, mild soap solutions for washing and flush thoroughly with clean, warm water if

possible. If the viewing surface receives a scratch, it should be polished out or replaced. Yellowing or crazing of plastic windows indicates weakening that is justification for replacement. Inspections every six months are recommended. Glass windows must be replaced at the first sign of any hairline crack or other sign of failure.

Doors

Door seals should be replaced when they no longer seal well.

Foundations and Substructure

Periodically, the structure should be checked to ensure that differential settlement of the foundation supporting the chamber complex has not induced deflections in the steel vessel such that stresses are induced by foundation settlement. Checks of deflection may be made with a theodolite or surveyor's transit (readily available instruments). Should such settling occur, shimming under the chamber supports may be resorted to in order to correct the situation.

PRESSURIZATION AND PRESSURE-CONTROL EQUIPMENT

Compressors

Where air compressors are used to pressurize or ventilate a hyperbaric chamber, they must be maintained in prime condition by following all servicing recommendations of the manufacturer, and periodic inspections must be carried out. Air filters and moisture traps shall be inspected and cleaned or replaced frequently. The discharge piping shall be inspected occasionally for signs of oil contamination, and the compressor serviced if the contamination rate is above the manufacturer's specifications. Reliability is of great importance, and worn or weak components must be replaced as soon as detected.

Cryogenic Pressurization

Where pressures are produced by cyrogenic means (vaporization of liquid oxygen-liquid nitrogen mixtures), all equipment associated with the pressurization system must be strictly maintained to the system manufacturer's specifications. Repair and adjustment must be made only by personnel specifically trained in the servicing of cyrogenic equipment and with a thorough knowledge of the safety and cleanliness requirements for liquid oxygen and liquid nitrogen. Modifications must be made only by the system manufacturer. Pressure-relief and safety valves should be inspected for proper settings periodically. All valve packings, seats, and gaskets that can contact the cyrogenic liquid or cold vapor therefrom must be made from material suitable for liquid-oxygen service. All flammable materials must be stored at least 25 feet from liquid oxygen.

Compressed-Air Cylinders

The Bureau of Explosives requires that all high-pressure cylinders be hydrotested every five years and that accurate records of test results be kept. If the cylinders are removed and filled by a vendor, adequate inspections shall be performed by the vendor at the time of filling. If cylinders are filled by system air-pumping equipment, a regular program of maintenance must be followed, based on approved industrial practice. This will include frequent inspections for oil contamination and water accumulation in cylinders and for serviceability of the cylinders and all associated regulators, valves, connections, and pipes. When the five-year inspection period arrives, the cylinders shall be removed from service and hydrostatically tested by a competent, authorized cylinder-inspection facility.

Pressure Indicating and Regulating Devices

Regulators, gauges, relief valves, and similar apparatus used to measure or control pressure shall be checked frequently for accuracy by comparison with standard reference gauges. They shall be recalibrated, readjusted, repaired, or replaced when inaccuracies or erratic actions are noted.

Manual Valves

Manual valves shall be repaired in accordance with valve manufacturer's recommendations. Valve packing, disk, and seat materials shall be as prescribed by the system manufacturer. All parts shall be kept clean and free from any traces of oil.

Chapter XII

EQUIPMENT FOR USE IN HYPERBARIC CHAMBERS

R. Adams Cowley, Ivan W. Brown, Jr., and James V. Harrington

INTRODUCTION

Safety is the prime consideration in the design and selection of equipment for use in hyperbaric chambers. Serious fires have taken place in both hyperbaric and altitude chambers for a variety of reasons, and each may be traced to a prior hazardous condition. With increased size, number, and complexity of instruments involved in medical hyperbaric chambers, hazardous conditions may be introduced unless meticulous care is paid to the nature of every material and the design of each piece of equipment used. Broadly speaking, equipment with excessive heat output, with exposed leads and connections that might spark or short-circuit, or that may burn and release toxic products or dense smoke should be considered as potential hazards. The use of lubricants, equipment requiring oil or grease lubrication, or oil-filled hydraulic equipment requires careful consideration. Oil and grease are known to ignite spontaneously in the presence of high oxygen concentrations.

Another source of hazard is the introduction of volatile substances in a hyperbaric chamber. Volatile anesthetics, hydrocarbons, alcohols, and many other chemicals can produce conditions that lead to either flash fires or toxic hazards.

Equipment that is not vented or that is affected by variations in chamber pressure should be viewed with concern. Light bulbs, vacuum tubes, ampoules, closed cans and bottles all have the capability of imploding or exploding with pressure variations—particularly rapid pressure variations where slow leaks cannot relieve the pressure difference in slowly venting devices.

Equipment reliability must be evaluated because the repair and replacement of equipment or parts will be extremely difficult, if not impossible, under operating conditions. Where necessary, standby systems should be provided for all vital equipment. In the repair of devices, no temporary make-shift repairs should be allowed. Repair personnel should be fully aware of the use of such equipment and the types of hazards to be guarded against.

A small test chamber, able to withstand several times the pressure of the main hyperbaric facility and to contain components to be used in the main chamber, is a useful adjunct to the hyperbaric facility in checking, under operating conditions, the suitability of equipment to go in the main chamber.

Floor area should be conserved to a maximum degree to allow free access to all parts of the chamber, and equipment should be designed to use the vertical dimension to the fullest possible extent to minimize the use of the limited floor area. Permanently installed equipment should be accessible at all times, so that fire or other potential hazards may be instantly coped with. Electrical leads and piping runs should be kept close to the walls or ceiling and out of the way of passages and walking spaces.

Underwater technologists have been developing equipment to withstand submergence in the ocean for long periods of time at great depth. Television and other types of cameras, as well as detecting and recording equipment for other purposes, have been developed, tested, and have proven acceptable under pressure. When selecting equipment for use in hyperbaric facilities, some equipment suitable for use in the ocean depths may prove applicable to the problem and should be considered for use in hyperbaric chambers.

This section discusses the various equipment necessary in hyperbaric facilities and their safety and operating features under hyperbaric conditions.

CHAMBER MONITORING EQUIPMENT

The basic instrumentation needed for monitoring the operation of a pressure chamber has been outlined. In addition, it would be useful to obtain and record certain other data during operation or experiments. For example:

1. Chamber-ventilation rate
2. Rate of rise and fall of pressure
3. Chamber pressure
4. Atmospheric temperature
5. Relative humidity
6. Oxygen partial pressure
7. Carbon dioxide concentration or pressure
8. Flammable-gas concentration and identification

Pressure, temperature, relative humidity, and air flow can be measured very accurately with standard, commercially available gauges and transducers. Oxygen partial pressure of effluent air can be measured with commercially available instruments. Carbon dioxide can be continuously analyzed by an infrared CO_2 analyzer sampling the effluent air. Detectors capable of measuring concentrations of explosive gases are commercially available for use in mines, and should give satisfactory service in the area of a hyperbaric facility.

A detailed permanent record of information should be kept for each operation or experiment, and recorded. Standard single or multichannel recording devices capable of making continuous recordings from the types of sensors mentioned are available commercially.

GENERAL AND SURGICAL LIGHTING

The subject of general chamber lighting was covered previously (Chapter IX) where it was pointed out that no available fixtures for lighting have been approved for use under pressure greater than 1 atm abs. This applies both to general and surgical lighting systems. Therefore, a judicious engineering selection of the lighting for these chambers must be made.

Several systems for lighting may be considered. In the first, lights are installed inside the chamber and are subject to the pressure in the chamber. In the second, lights are installed outside the chamber and shine through glass safety ports. The second system, however, has one immediate disadvantage: the lights are fixed and their positioning is not directly under the control of the surgeon. The hazard of having lights positioned too close to the glass port and cracking the glass exists.

For general lighting, the use of enclosed bulbs is suggested, so that if a bulb breaks the fragments will be contained and isolated to a certain degree from the chamber.

The surgical lighting fixture of the operating area should provide a maximum degree of flexibility of position, and the lamp should have a removable, sterilizable aluminum handle to permit positioning by the surgeon.

There are units available that have large-diameter aluminum reflectors with reflectivity as high as 85 per cent and diminution of reflectivity of not more than 2 per cent over the life of the unit. Color-correcting, heat-absorbing globes are also available that can provide a color temperature of approximately 4000° and a beam temperature within acceptable limits. The lamp assembly should be such that at any point 20 inches or more below the rim of the reflectors, the heat rise will be no more than 3°F over ambient temperature per 1000 footcandles of intensity per hour of continuous operation.

It is recommended that light bulbs be operated at a pressure of at least three times the maximum chamber operating pressure before they are installed in the hyperbaric facility to ensure that defective bulbs will not be used.

It must be recognized, of course, that this is not an absolute assurance of safety, but it is probably the best safety precaution that can be taken. All bulbs must be suitably enclosed to prevent the scattering of fragments and to isolate the bulb as much as possible from the chamber atmosphere.

OPERATING TABLES

Most of the operating tables in present use depend on a hydraulic system for positioning various parts of the table and changing elevation. Hydraulic oils and other fluids in a hyperbaric facility can be the source of fires and toxic substances. For this reason, great care must be exercised in selecting operating tables and the systems associated with them. It would be considerably better, if one could be found, to select an operating table that is positioned by mechanical linkages operated either manually or by a closed air motor. In any event, suitable operating tables for hyperbaric service must be selected with great care to ensure safety at all times.

It is recognized that flame-resistant, nontoxic hydraulic fluids may be used to replace

oil in operating table systems. Care should be taken to ensure that fluids are not selected that will act as paint removers on surrounding surfaces in the event of leakage.

PUMP OXYGENATORS AND EQUIPMENT FOR ASSISTED CIRCULATION

Equipment for extracorporeal circulation within the chamber require power for pump drives and for moving gases. These devices have three sources of power: (1) a closed air motor, (2) an electrical motor, and (3) a closed hydraulic system. Of the three systems, it is believed that the closed air motor system introduces the least hazard. The device can be controlled by needle valves and mechanical speed changers between the motors and the blood pumps. Such a unit may be adequately soundproofed, particularly if the air system is closed and the exhaust is not admitted to the chamber.

All waste gases from the pump oxygenator should be vented through the anesthesia discharge hood in accordance with current operating practice.

Operation of the pump oxygenator under hyperbaric conditions will greatly increase efficiency of oxygenation and will allow both the use of smaller oxygenators and the ultra-miniaturization of equipment. Carbon dioxide removal will not be facilitated under hyperbaric conditions, but this will not prevent miniaturization of currently employed efficient disk oxygenators because they provide a generous safety factor for CO_2 removal.

It appears that hyperbaric oxygenation will not aid in miniaturization of membrane oxygenators because these instruments are limited in size at present, owing primarily to the difficulty of eliminating CO_2 which, in turn, is caused by the smaller difference in partial pressure available for diffusion.

HYPOTHERMIA EQUIPMENT

There is a variety of hypothermia equipment on the market. The most suitable for hyperbaric use appears to be one in which a cooling fluid is circulated in a hypothermia blanket. The cooling system in this case can be mounted on the outside of the chamber and fluid connections made through the chamber wall and connected to the blanket. Care must be exercised in using this equipment to ensure that the fluid cooling lines are properly vented, because the entrainment of air or other gases will cause the blanket to swell and rupture as pressure in the chamber is reduced. Brine is the preferred cooling medium, because it is nontoxic and nonflammable.

INDIVIDUAL BREATHING MASKS FOR PATIENTS AND CHAMBER WORKERS

Under some circumstances, such as fire with emanation of toxic contaminants or spills of volatile materials, it may be necessary for chamber workers and patients to breathe an atmosphere other than that of the chamber itself. An independant gas supply to the chamber with connections for individual breathing apparatus at least equal to the maximum number of patients and workers the chamber will contain has been specified. The preferable system consists of a high-pressure air or mixed-gas supply and individual demand regulators feeding each mask in the chamber. A breathing mask that covers nose, mouth, and eyes is the most desirable. Eye irritation, unless the eyes are enclosed in the mask, will result in case of noxious fumes and smoke.

The separate air or mixed-gas supply should have a design flow capacity equal to the demand of all individuals in the chamber inspiring simultaneously. Extra masks should in all cases be supplied in the event of a unit malfunctioning.

SURGICAL INSTRUMENTS, DRAPES, AND GOWNS

It is customary to lubricate most surgical instruments. As shown previously, hydrocarbon lubricants have been known to ignite spontaneously in atmospheres containing a high concentration of oxygen. Therefore, the minimum necessary quantity of lubricant should be applied to these instruments. The development of instruments having Teflon-coated moving parts should be encouraged to obviate hydrocarbon lubricants.

It is suggested that all gowns and drapes used in the hyperbaric facility be treated with a solution of tetrakishydroxymethyl phosphonium chloride or some other suitable chemical in order to reduce inflammability. At the present time, there is no effective method of treating fabrics that will make them flame-proof. Any nontoxic solution that would reduce inflammability is desirable.

PATIENT MONITORING EQUIPMENT

During the course of operations and medical treatment, it will occasionally be desirable to monitor various activities associated with the patient's systems. The most likely character-

istics to be monitored are:

1. Electrocardiograph
2. Arterial pressure
3. Venous pressure
4. Electroencephalograph
5. Electroretinogram
6. Esophageal and rectal temperatures

Usually it will be desirable to install the recording equipment outside the chamber and run the signal cables through penetrations in the chamber walls. This will both minimize the amount of equipment in the chamber and eliminate certain hazards. In any event, the transducers associated with the patient must be in the chamber, and each such transducer should be tested to ensure safe service under hyperbaric conditions. One must also remember that the calibration of some transducers will change with variations in pressure, and the characteristics of these transducers must therefore be known.

INTRAVENOUS AND INTRA-ARTERIAL FLUID THERAPY EQUIPMENT

No difficulty is anticipated with standard vented intravenous therapy bottles. Intra-arterial therapy equipment may be driven electrically, hydraulically, or pneumatically. As in the case of the pump oxygenation equipment, the preferable drive is now a closed air system because it would result in the least hazard. An alternate method of driving such a device would be to have the drive mounted outside the chamber with a rotating shaft seal through the chamber wall. Such an arrangement, i.e., using a rotating seal with the drive outside the chamber, may be impossible in some instances. In some cases, the wall of the chamber may be too far from the operating area, and the installation of rotating seals in the chamber wall will always represent a potential source of gas leakage. Of the two systems discussed, it is believed that the drive associated directly with the unit, all of which is within the hyperbaric chamber itself, is probably the most satisfactory arrangement.

GAS-VENTING PATIENT HOOD

A gas-venting hood, or similar device, should be provided to the anesthesiologist for the immediate venting of all surplus oxygen and anesthetic vapor, in order that it may be immediately swept out of the chamber. This device should be so designed that it would be impossible to impose a static differential on the patient's respiratory system.

A vent hood for this purpose can be easily constructed so that it is completely safe and the immediate discharge of waste gases will significantly protect chamber workers.

CHAMBER CLEANING AND STERILIZATION

Proper chamber construction includes drain plugs and removable floors to facilitate cleaning. Soap and water scrubbing is desirable for cleaning.

Beta-propiolactone vapor makes an excellent chamber-sterilizing agent. The vapor should be left in the chamber for a period of 12 hours or more, and then flushed out with air.

FRESH-WATER SUPPLY AND WASTE DISPOSAL

A supply of drinking and wash water should be provided in a vented container. A tank should be supplied for the temporary collection of waste.

SANITARY FACILITIES

Small sanitary units are available commercially.

Chapter XIII

SAFETY FACTORS IN CHAMBER OPERATION

George F. Bond

FIRE HAZARDS

Of the many hazards associated with operation of hyperbaric chambers, by far the most serious is that of accidental fire. The reason for this statement becomes clear when the complexities of the hyperbaric environment are considered.

First, the physical features of the functional pressure chamber multiply the hazards of even a small fire. The most obvious action to be taken in the event of fire in the occupied operating area would be to vacate that particular lock and isolate the personnel in an external space where protection against fire and noxious gases could be afforded by securing a hatch. In many instances, however, this action cannot be taken. Often the condition of the patient, under anesthesia and in process of surgical manipulation, will be such that he cannot be moved with dispatch; under such conditions the therapy team would probably elect to remain with the patient, risking death of all of the chamber occupants. In less extreme cases, the patient might be removed with the treatment team; but even under best possible circumstances, some additional delay is certain, with increased hazard to all chamber occupants.

Second, few people will be free to fight fire in the hyperbaric chamber. The therapeutic team will always be small, with each individual assigned to multiple tasks essential to the patient's condition. Thus, seldom more than one person will be available for the critical task of fire fighting; and he is not likely to be the most effective member of the team.

Although a compressed-air atmosphere will not greatly enhance flame propagation, since this is primarily a function of oxygen percentage rather than partial pressure, chamber pressures are very significant when noxious flame products are considered. The two principal toxic gases produced by fire are carbon monoxide and carbon dioxide. The noxious effect of carbon monoxide is a function of relative concentration rather than partial pressure; hence, its toxicity would not be significantly enhanced by a hyperbaric environment. The toxicity of carbon dioxide, however, is in direct proportion to the partial pressure of the gas, and thus to the ambient pressure of the chamber. Furthermore, depending on the particular material being consumed by fire, a host of other lethal gases, ranging from hydrogen cyanide to nitrogen dioxide, may be evolved. Although very promising work is currently underway in fireproofing textiles suitable for use in hyperbaric chambers, complete success has not been achieved. Certainly, development of totally fireproof textiles will be an important safety factor in future hyperbaric chamber operation.

Finally, unless especial efforts are made to remove all oxygen-rich exhalations of the patient from the treatment chamber, some increase in oxygen concentration is inevitable. This enrichment will augment the possibility of ignition and flame propagation.

FIRE FIGHTING

In general, only two basic methods of fire fighting can be considered for use in hyperbaric chambers: quenching with gas and with water. Both methods, however, may pose severe problems.

Carbon dioxide extinguishers are compact, efficient against all classes of fire, and relatively inexpensive. Unfortunately free release of CO_2 gas in the breathable atmosphere of a hyperbaric chamber will require special protection for all members of the therapy team. Such protection can best be afforded by a built-in breathing system (BIBS), with the view to providing masks, regulators, and proper mani-

fold connections for potential occupants of the chamber. Because such a breathing system is desirable in all hyperbaric chambers, it would seem likely that CO_2 extinguisher systems will be standard in these complexes. It must be emphasized that, in the absence of a BIBS, CO_2 extinguishers cannot safely be employed in a hyperbaric chamber. It should also be borne in mind that the fit of any face mask is critical to safe BIBS operation; toxicity must be considered wherever an intake leak could result in inhalation of a dangerous amount of CO_2.

In a search to replace CO_2 as a gaseous fire-quenching agent, both nitrogen and helium have been considered and tested. Neither appears of value in fire fighting because significant gas concentrations cannot be maintained. On account of their narcotic properties, the gases of heavier molecular weight cannot be considered for such use under pressure.

Perhaps as a last resort, it should be remembered that rapid dilution of the chamber atmosphere with an adequate quantity of either helium or nitrogen will reduce the oxygen to a concentration that will not support combustion. If a sufficient quantity of one of these gases is available, such an emergency measure deserves consideration. In hyperbaric chambers of the future, where captive and synthetic atmospheres are employed, it will probably be possible to keep oxygen concentration at operating pressures below 10 per cent with virtual elimination of the fire hazard, while providing an adequate partial pressure of oxygen for physiological needs.

Water-quenching systems deserve equal consideration. To fight combustion of some classes, water drenching may be preferable to CO_2. The small, isolated carbonaceous flame, for example, could be handled without recourse to a complicated and hampering BIBS. Basically, two water systems are available: single-stream hose delivery, and overhead fixed-drench or fog delivery. Ideally, both systems should be available in the chamber. Of the two, hose delivery appears preferable for most cases, in that it can be manually directed and offers a choice of water jet or fog delivery. Nevertheless, there may be occasions in which a general drenching system would be mandatory, e.g., for flash combustion of flammable gases throughout the lower flats of the chamber.

Finally, because many of the fires that may occur in the hyperbaric chamber will be small and well localized, the time-honored sand and water buckets are required in every such installation.

FIRE PREVENTION

The consequences of fire in a hyperbaric complex would be catastrophic. In view of the manifest difficulties of fighting fire in this situation, great effort must be taken to prevent its occurrence. To this end, all potentially sparking electronic equipment should be outside the chamber, or if that is impossible, encapsulated and surrounded with a purging atmosphere of inert gas, preferably nitrogen or helium. Such a purging system is feasible and should be incorporated into the hyperbaric-chamber design.

Of the greatest importance in preventing or managing fire in a hyperbaric complex is a comprehensive fire-safety bill, tested by frequent casualty drills. The plan must be clearly understood by all members of the operating team. With daily advances in medical instrumentation and pharmaceuticals, it is inevitable that new material will regularly be introduced into the hyperbaric chamber. Such material, all of which is potentially dangerous, must be subject to inspection and test, with an eye to regular revision of the safety bill.

ATMOSPHERIC HAZARDS

To some degree, all volatile substances are toxic, narcotic, or both, in direct proportion to the partial pressures of the evolved gases. Within a closed vessel, such as a hyperbaric chamber, much of the incarcerated material may be assumed to be volatile, hence toxic. For example, oil-base paints continue to emit potentially toxic hydrocarbon vapors many months after application; spilled mercury, which is nearly impossible to recover completely, will vaporize for years; and accidental release of the toxic fluids of the commonly used aromatic compounds might quickly result in disastrous atmospheric contamination.

With the advent of the nuclear submarine and later the space capsule, it became apparent that the toxic potential of many commonly accepted materials required reexamination. It was likewise obvious, because of the long human-exposure times involved, that current industrial medical standards did not apply to the closed ecological situation imposed on the submarine occupants. For example, nearly 200 potentially toxic elements have now been identified in the respirable atmospheres of

nuclear submarines, despite rigid control of material introduced into the closed system.

Although occupancy of the modern submarine involves continuous breathing of these noxious contaminants for periods of 60 days or more, as opposed to the intermittent exposures anticipated in the hyperbaric chamber, the physiological hazard of low-level toxic substances in the chamber should not be minimized. All captive atmospheres are subject to toxic contamination, and the operational requirements of the hyperbaric chamber will surely offer a large and ever-changing spectrum of these contaminants. Over the course of several years of clinical work, inside operators will accumulate hundreds of exposure hours, increased considerably in effect by multiplication of partial pressures. Therefore, it is conceivable that repeated exposures under conditions of increased ambient pressure might be equivalent to long-term continuous exposure. Future research will undoubtedly shed light on the problem, but it is important, pending the acquisition of definitive data, to eliminate, reduce, or at least control all potentially toxic substances within the hyperbaric chamber.

Of the many potential toxic contaminants, the hydrocarbons deserve primary consideration. Depending on chemical structure, some volatility or oxidation can be predicted of any hydrocarbon. Many of these compounds cannot be removed from the atmosphere by known techniques. Thus, methanol, acetone, toluene, and a host of similarly volatile materials must be excluded, or at least rigidly controlled, for use in the hyperbaric chamber. Mercury is difficult to remove after a significant spill and can produce mercury poisoning in individuals subject to daily exposure. Any electronic device with a spark gap will eventually produce a significant quantity of ozone and reaction by-products of nitrogen and oxygen; all must be detected and eliminated.

Hyperbaric chambers with closed atmospheric systems will require examination of all sources of potential atmospheric contamination because human exposures will be continuous and long. Even if the known toxic contaminants could be eliminated or accounted for in such a captive atmosphere, the normally innocuous atmospheric components would have to be considered. For long-term exposures in a high-pressure synthetic gas, the safe proportions of inert gases and oxygen will have to be defined. The inert gas chosen should probably be helium which offers relative immunity from narcosis and poses only a small density problem at the pressures contemplated. Oxygen, however, will require rigid control because high partial pressures at a prolonged exposure to oxygen will probably result in pulmonary damage.

Thus, a careful control of the atmosphere of the hyperbaric chamber is desirable today; in the future it will be mandatory. The problem cannot be ignored.

Chapter XIV

SELECTION AND TRAINING OF OPERATIONAL PERSONNEL

George F. Bond

The selection of personnel who will work regularly in the hyperbaric complex is a matter of considerable importance. Even under routine operating conditions, working exposures in the pressure chamber are hazardous, physically demanding, and productive of physiological and psychological stress. In abnormal situations, e.g., in the treatment of pressure accidents or in therapy requiring extreme exposures, environmental stresses may become almost intolerable. Because the failure of individual personnel under operating conditions might endanger all chamber occupants, the criteria of personnel selection deserve careful consideration.

PSYCHOLOGICAL REQUIREMENTS

It is generally believed that claustrophobic tendencies and overt reactions constitute the sole psychological selection criteria for work in hyperbaric chambers. This is not exactly the case. Although both frank and latent claustrophobia are very significant factors in personnel selection, motivation, maturity, and interpersonal compatibility deserve equal consideration.

Overt claustrophobia is almost always recognized by the afflicted individual, and those so affected are not likely to accept employment in a pressure chamber. Not infrequently, however, an individual normally unaffected by closed environments may react severely to the situation in a hyperbaric chamber where the sense of complete incarceration is very real. Ordinarily, one or two exposures to actual operating conditions will reveal this unrecognized defect and result in disqualification of the potential worker. More difficult, however, is the case of a person whose latent claustrophobic tendencies become apparent only under prolonged confinement or extrahazardous environmental conditions. Such a person may function adequately for long periods under routine hyperbaric conditions, only to break dangerously at a critical point of the pressure operation. Unfortunately, this latent response pattern can rarely be predicted and is revealed only at a time of extreme exposure. Nevertheless, it is a possibility to which chamber operators must be alert.

For sustained, efficient performance in a hyperbaric chamber, motivation is a most important factor. Working conditions are relatively harsh, with long periods of boring inactivity, frustrating physical restraints, and constant hazard. Clearly, an individual must possess a high and sustained level of motivation to render efficient performance under such conditions. In the case of professional personnel, adequate motivation may be assumed by their voluntary participation in the hyperbaric program. Paramedical personnel of lower echelons, however, may not be so well motivated, and the adverse or neutral attitudes of such people may jeopardize team morale and dangerously reduce operational efficiency.

Finally, the environmental conditions imposed by hyperbaric chamber operations will necessitate long periods of physical proximity without opportunity of escape. Unlike the situation of a conventional operating room or therapy ward, it will be virtually impossible for any therapy team member to avoid continuous and close contact with other chamber personnel. It is therefore essential that all team personnel be possessed of an unusual degree of maturity and compatibility. In the selection of therapy teams, all possible factors contributing to smooth impersonal relationships must be carefully weighed.

PHYSICAL REQUIREMENTS

Under hyperbaric conditions, even simple existence poses a considerable stress on the exposed individual. The increased density of the compressed-air atmosphere imposes the multiple problems of increased work of

breathing, nitrogen narcosis, and disturbed metabolic processes, common to all caisson work. With the added, nearly continuous physical and mental effort required for effective work in therapy pressure chambers, the total physiological load may become severe. In consequence, the physical criteria for personnel under these circumstances are of considerable importance. Basically, prime consideration must be given to the general areas of pulmonary function, central-nervous-system effects, cardiovascular integrity, otolaryngological complications, and skeletal health.

Pulmonary Function

Medical and paramedical personnel working in air compressed to 7 atm will inevitably encounter considerable breathing resistance (Wood, Leve, and Workman, 1962; and Lord, Bond, and Schaefer, 1964), with concomitant increase in the work of breathing. Although this additional workload is acceptable in the case of a healthy individual, any person with destructive or bronchospastic airway disease may be unequal to the environmental stresses of hyperbaric work and should be excluded from the active program. It is recommended that all potential workers in hyperbaric facilities be subjected to initial and annual pulmonary-function tests to detect possible decrements of pulmonary capacity and function.

Of great importance is the matter of excluding from all hyperbaric work those individuals who might be subject to localized air trapping in alveolar spaces. The presence of generalized or bullous emphysema should be an absolute contraindication to work under increased ambient pressures. Obviously, this interdiction cannot apply to the variety of patients requiring therapy; however, the medical therapists, who will daily undergo pressure exposures, must be carefully screened. There is considerable evidence that any pathological condition of generalized or local air trapping will predispose to the development of air embolism during decompression. Prudent selection of chamber workers will certainly reduce the incidence of this potentially fatal pressure accident.

Central Nervous System

Although no specific central-nervous-system effects have been observed from exposure to compressed air, excepting nitrogen narcosis, it is in order to consider nervous-system integrity in selection of personnel for work in such an environment. Preemployment physical screening should be particularly designed to reveal any central or peripheral nervous-system defect in the prospective worker, since late discovery of such disorders may raise serious question as to the etiology and chronology of the neurological lesion. It should be remembered that any pressure accident may result in a nervous-system defect, whether temporary or permanent. Therefore, discovery of a central or peripheral nervous-system lesion after a period of employment in a hyperbaric facility will inevitably raise the question of preexistence of the defect. In conclusion, it should be mandatory to rule out all potential convulsive-disorder subjects for work in the OHP facility, since unexpected occurrence of a seizure would not only create unwarranted physical confusion and division of effort, but might as well raise reasonable doubts in the minds of other workers with respect to the purity of the atmosphere.

Cardiovascular Integrity

Although specific cardiovascular effects of the compressed-air environment have not been demonstrated, some general considerations are applicable to the selection of chamber workers. Because the work of breathing may be considerably increased, with a concomitant reduction in vital capacity, some secondary cardiovascular stress is to be expected in hyperbaric work. Such stress, ordinarily acceptable, may become quite severe if strenuous physical exertion is required, as in the treatment of a serious pressure casualty. Although the exact mechanism is not known, so-called "divers' fatigue" is a very real entity. This syndrome, which results from exposure to compressed air and is independent of muscular exertion, may represent an end result of disturbed metabolic function. Thus, it may be seen that exposure to a hyperbaric environment is accompanied by a host of minor but definite physiological alterations, all of which, singly and in combination, must be carefully considered in prediction of cardiovascular stress.

Otolaryngological Conditions

If, because of acute or chronic pathological conditions, communication of the paranasal sinuses or middle ear with the oropharynx is partially or totally occluded, pressure equal-

ization of these enclosed spaces will be inadequate or impossible. Under such circumstances, exposure to increased ambient pressure will not be tolerated, owing to the pain and tissue trauma that accompany sinus or middle-ear "squeeze." Although this inability to equalize may occur from time to time in any individual, only about 5 per cent of the general population are consistently inadequate in this regard and, thus, unsuited for work in a pressure chamber. In this area of personnel selection, it should be borne in mind that the ability to equalize always improves with practice, and initial failures do not necessarily lead to final disqualification. When a vital member of the pressure-therapy team experiences persistent difficulties of equalization, examination may reveal lymphoid hyperplasia near the eustachian ostia, resulting in partial occlusion of the orifice. Such cases will usually be benefited by X-ray or radium treatment of the affected areas. Generally, the beneficial results of such treatment are not apparent for about three months. It should be emphasized that an adverse otolaryngological examination or a history of chronic sinusitis need not disqualify the applicant for hyperbaric work. The best criterion in all cases will be a series of tests of pressure that should be the determining criterion of selection.

Skeletal Health

Inasmuch as aseptic bone necrosis, a common occupational disease of caisson workers, is a distinct pathological possibility in hyperbaric-chamber personnel, it requires consideration as a factor in selection. This condition, when it involves an articulating joint, may result in a permanent, disabling, and compensable condition. Because radiologic evidence of the disorder may be indistinguishable from that of degenerative forms of osteoarthritis, it is desirable, if not mandatory, that all potential hyperbaric workers have pre-employment radiologic joint surveys of hip and shoulder articular areas, with repeated evaluations at annual intervals. Although it is not known whether previously existing bone disease or traumatic disorders may predispose to the aseptic bone necrosis of pressure exposure, it might be advisable to eliminate persons with such pathological lesions from daily employment in a hyperbaric program.

Age and General Physical Condition

Ideally, working personnel of the hyperbaric complex should be young, lean, stable, "bends" resistant, and in perfect health. In the case of U.S. Navy divers, this ideal is approached. With caisson workers, "bends" resistance and phlegmatic disposition become basic requirements, with the other factors waivable. The average working occupants of a clinical hyperbaric facility, however, rarely meet these criteria. Top-flight surgical or medical therapists are rarely young, seldom in excellent physical condition, and almost never phlegmatic; furthermore, "bends" resistance is always an unknown factor in the group. With nursing personnel, an added and poorly evaluated factor of sex is introduced; and, in the case of orderlies, desirable physical attributes are not likely to be found. It is evident that in selecting personnel for work in a pressure chamber, a compromise must be reached. Generally speaking, the selection of lean, reasonably healthy individuals in the middle decades of life and of even temperament should be a prudent goal. Because the physical condition of the least-acceptable member of the hyperbaric-facility team will probably dictate the limits of exposure and may well establish decompression times, considerable attention must be devoted to persons whose contribution to the overall team effort may be minimal.

Finally, and of some importance, is the matter of "bends" resistance. Although no experimental data are available, statistical evidence from caisson operations clearly indicates not only that such resistance is demonstrable, but that it may also result from repeated exposures to compressed-air atmospheres. In general, it may be assumed that the obese and older persons will be more susceptible to decompression sickness than leaner and younger persons. In the last analysis, it is clear that the selection of personnel for chamber work will depend largely on repeated exposures and subsequent evaluation and choice.

The selection of personnel for regular work in the hyperbaric complex is difficult. The psychological and physiological factors involved are ill-defined, but deserve careful attention. Although it is recognized that the ideal cannot be realized under normal requirements of operation, there are reasonable yardsticks of selection factors.

TRAINING OF OUTSIDE OPERATORS

If the criteria for selecting personnel to engage in daily work in hyperbaric facilities are important, the training of chamber-operating personnel deserves at least equal emphasis.

Outside operators will require longer and more technical training than those persons whose responsibilities will be limited to the clinical management of patients within the chamber. Depending on the sophistication of the treatment facility, the team of two or more outside operators will require intimate knowledge of piping layout, environmental control systems, monitoring devices, compressor operation, communication, and electronic circuitry for safe and efficient operation of the complex. In addition to these requirements, all outside operators must be sufficiently knowledgeable in the medical aspects of exposure to compressed air to guarantee safe operation under normal and emergency conditions. Inasmuch as few hyperbaric-treatment facilities are currently in operation, and no two units are likely to be identical, familiarity with any given facility requires early employment of outside operators to ensure the necessary on-site training. Such a plan, although time-consuming and relatively expensive, would seem mandatory in case of the initial installations in the United States.

Ideally, the senior member of the outside operating team should have a broad background in mechanical, stationary, and electrical engineering. The progressive sophistication of chamber operations and advances in the state of the art make it mandatory that the senior operator be trained to extract maximum efficiency from the complex to guarantee the highest level of professional competence on the part of the entire therapy team. It is of critical importance that the senior operator have basic responsibility for the safe maintenance and successful functioning of the facility at all times. He must therefore be aware of and agreeable to any of the various equipment modifications required by newly developing therapy situations. In this connection, it is of great importance that the senior outside operator be thoroughly familiar with all national, state, and local codes as they apply to the major components of the hyperbaric facility. Regular inspections of the equipment by authorized fire, building, and insurance inspectors should be anticipated; and compliance with all applicable codes will be a rigid requirement for approved operation. The senior operator should have no additional responsibilities outside the complex, i.e., his job should be a full-time one. Certainly, in this instance, the laborer is worthy of his hire.

Subordinate outside-chamber operators may not require a proportional academic background, but considerable experience with the hardware systems involved is mandatory. Basically, these operators should be capable of safe normal operation of the complex, for they will bear the greatest responsibility for repair and preventive maintenance. In addition, they should be sufficiently indoctrinated in the basic theory of pressure physiology to be considered reliable members of the therapy team.

Availability of Trainees

With these general requirements in mind, it is reasonable to ask where such people are to be found and how they are to be trained. Unfortunately, these questions are not readily answered. At present, a very limited supply of trained personnel is available by virtue of annual retirement of naval petty officers who have worked in diving activities and with the associated naval hyperbaric facilities. Less abundant are hospital corpsmen with diving experience whose paramedical training is of particular value. The availability of such trained and dedicated persons is limited, and they may not meet the numerical demands of multiplying civilian installations. Furthermore, in the case of more-complex chamber installations, individuals with formal training in bio-engineering will be required for optimum use of the facilities. Such scientific personnel, who would serve as senior operators, are available, but must "grow up" with the conceptual engineering installation and will require additional instruction in the medical aspects of hyperbaric procedures.

Training Courses

No formal courses are yet available for the final training of outside-chamber operators and medical and paramedical inside workers. The need for such courses, however, is being anticipated by teaching institutions, and suitable short training programs will probably be available to hyperbaric operators. These programs, nonetheless, must be considered as postgraduate instruction, designed for a limited number of professional persons in the fields of medicine and bio-engineering, and supplementary

workshops may be desirable. Ultimately, it will probably be necessary that industry arrange for on-the-job training of hospital personnel in the aspects of hyperbaric-facility operation requiring less formal education.

Finally, it must be emphasized that safe, efficient, and productive use of any hyperbaric facility is a function of the adequate training of all individuals concerned. Inside ancillary operators require little more than a sound understanding of the principles of pressure physiology, and these fundamentals can be taught through any accepted hospital training program. The more extensive education required for advanced members of the team can be provided only by university-oriented workshops directed to this specific need.

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Chapter XV

THE ADMINISTRATION AND OPERATION OF A MEDICAL HYPERBARIC FACILITY

Ivan W. Brown, Jr.

ORGANIZATION

Although interest in clinical applications of hyperbaric oxygenation will vary from one institution to another, the development and successful operation of any medical hyperbaric facility require a large and continuing investment of money and talent. Furthermore, the role and scope of hyperbaric therapy are still largely undefined. Therefore, a multidisciplinary approach, bringing together investigators with diverse interests and skills, will ensure the most efficient and productive use of a medical hyperbaric facility.

The hyperbaric research program in one institution* has been organized around an Interdepartmental Operating Committee. The committee consists of a project director, assistant director, and six participating hyperbaric investigators, each representing a department or a clinical discipline. The project director and the assistant director are both active investigators interested in their own hyperbaric research problems and devote at least 75 per cent of their time to the hyperbaric project. The director is responsible to the institutional officials for overall administrative matters, records, and the budget. He is also in charge of personnel training. The assistant director is responsible for chamber operations, equipment, safety, and maintainance. Both are appointed by a special administrative committee consisting of the dean of the medical school and the chairmen of the departments participating in the project. The administrative committee reviews all major fiscal and policy decisions made by the operating committee. By limiting the length of appointments to the operating committee and by monitoring all decisions relating to major institutional commitments, the department chairmen retain an adequate measure of control. However, from the practical standpoint, the operating committee administers the hyperbaric research program. It meets frequently to consider budgetary matters, the purchase of equipment, appointment of technical and professional personnel, and all research protocols submitted by investigators proposing to use the hyperbaric facility. It also has jurisdiction over the allotment of chamber time to the various projects and departments to ensure equitable distribution of the necessarily limited available time.

In the institutional setting under discussion, this arrangement has promoted harmony among the various departments and disciplines and has resulted in considerable cross-fertilization of ideas and successful joint projects. In other institutions such an arrangement might not be feasible or desirable. What is important is that every clinical hyperbaric facility have a competant physician or group of physicians trained in hyperbaric physiology and clinical investigation who can be responsible for its administration and safe operation.

In a multidisciplinary project, efficiency can probably be accomplished best by developing a core of personnel trained to operate the chamber and meet technical assignments common to many groups, while requiring that each investigator bring to the facility skills and equipment unique to his particular study. The chamber personnel would assist, modify equipment to establish technologic compatibility, and perform the technical tasks for which they are trained.

Priorities must be established for clinical and experimental time assignments. Patient treatment must be considered more crucial than experimentation. An appropriate investi-

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gative approach to patient treatment can best be ensured by requiring that each hyperbaric treatment be evaluated and approved by at least two members of the operating committee. Inasmuch as established experimental programs would suffer from prolonged diversions to patient treatment, conflicts of interest inevitably arise. The practical solution is to extend the chamber day to such a length that experimentation and patient treatments can both be undertaken. Twenty-four-hour operational capacity would be ideal for this purpose, but, from a practical point of view, a 168-hour week is enormously expensive, as well as inefficient if it exceeds the demand. Another approach is to begin with an eight-hour, five-day week (plus 24-hour emergency capability) and then to extend the routine operating day to 12, 16, and then 24 hours as demand warrants. By this means, personnel training can proceed gradually and economically.

Nonprofessional skills of a high order are needed for a successful hyperbaric operation. The most essential members of the nonprofessional team are experienced chamber operators who combine a versatile technical proficiency and a recognition of their great responsibility for life. Experienced Navy diving-chamber operators are ideally suited. Machining, pipefitting, and electronic skills are also virtually essential for good chamber operation. In this environment, the chief chamber operator is the overall supervisor of the chamber facility, with responsibility for the work of technicians, machinists, and even nurses. He has the authority delegated to him for time assignments, function assignments, vacation schedules, 24-hour emergency schedules, and personnel-performance ratings. The cohesive nature of a hyperbaric facility generates a unity of purpose and enthusiasm among personnel if the direction is good at both the nonprofessional and the professional levels.

RECORDS

Adequate and well-kept records are essential to the proper operation of any clinical hyperbaric facility. The chamber operator's working record (rough notes) of each "dive" is the most important. A printed form facilitates the keeping of orderly notes that are easy to interpret. One such form for this purpose is illustrated by Figure 1. This record, made at the operator's control station, lists the chamber occupants including the patient, the date, and the purpose of the "dive." It also lists the exact times and depths from leaving surface through decompression stops to return to the surface, the bottom depth of the "dive," the total bottom time, the number of U.S. Navy table used for decompression, the total decompression time, and the total time of the "dive." Initial and final pressures of oxygen supply tanks, reserve air tanks, and nitrogen "purge" tanks are also listed for the operator's information. Although the chamber operator's working record of a dive notes the repetitive-group letter (Navy Tables) assigned to each chamber occupant at the end of the "dive" for residual nitrogen, the repetitive-dive worksheet (page 66, U.S. Navy Diving Manual) is extremely useful for calculating and recording the necessary decompression for a repetitive dive.

Detailed and accurate records of all cases of decompression sickness must be kept. The U.S. Navy form "Report of Decompression Sickness"(Navmed-816), or some modification, is recommended as a permanent record for this purpose.

The working notes of the chamber operator become a part of the permanent records, but an individual hyperbaric log form should also be kept for all chamber personnel who undergo compression. This individual log need not be elaborate; its purpose is to serve as a ready reference to the hyperbaric exposures to which a given person has been subjected. A sample form of one such log is shown in Figure 2.

Because hyperbaric-oxygenation therapy is not without risk and is not yet widely accepted and conventional, the wise clinical investigator will obtain signed permission both for treating patients and for using normal human subjects in experiments. Sample forms for these purposes are illustrated in Figures 3 and 4.

Records of the physical fitness of personnel (Chapter XIV) must also be kept and certain essential examinations must be repeated every 6 to 12 months, not only for the protection of the personnel involved, but also for the legal protection of the institution responsible for the operation and administration of the hyperbaric facility.

CHECKLIST FOR CHAMBER COMPRESSION

Probably no two medical hyperbaric facilities will have identical systems of operation or maintenance. However, some principles of operation apply to all. One of those is the use of a checklist for compression and decompression to ensure that the facility is in proper working order and that all safety and emergency

equipment and supplies are present and ready for use. Although such checklists will vary according to the type of chamber and equipment in use, a sample checklist is reproduced below:

CHECKOUT PROCEDURE PRIOR TO OPERATION OF A HYPERBARIC FACILITY

A. Air System
   1. Compressors
      a. Check oil levels
      b. Check alarm system
      c. Start up
      d. Check condensation drains
      e. Check cooling water flow to drains
   2. Reserve air banks
      a. Check and tag all banks
         (1) Empty
         (2) In use
         (3) Standby
      b. Cut bank to be used into manifold
         (1) Check regulators—interstage 600 psig, final 100 psig
   3. Oxygen system
      a. Check and tag banks
         (1) In use
         (2) Standby
      b. Cutbank to be used in manifold
         (1) Set reducing regulator to desired pressure
         (2) Set inside regulator to to desired pressure
   4. Anesthetic gases
      a. Open cylinders and set pressures if to be used

B. Chamber
   1. Check condition of all control valves
      a. Dual control valves open outside
      b. Dual control valves closed inside
      c. Outside control valves closed
      d. Bilge drain valves closed
   2. Pass-through locks
      a. Bolted inside and outside
      b. Valves
         (1) Open outside
         (2) Closed inside
   3. Air conditioning
      a. Air on to motors
      b. Air on to humidifiers
      c. Temperature set
   4. Expiratory exhaust system
      a. Air on to controller
      b. Exhaust hoses connected
   5. Emergency breathing system
      a. Air on
      b. Masks checked
   6. Fire sprinklers
      a. Pressure up
      b. Cutoff valves open
   7. Electric power ungrounded
      a. Alarm checked and working
   8. Inside check
      a. All containers vented
      b. All inflammables out
         (1) Cigarette lighters, matches
         (2) All personnel in proper dress
         (3) All trash cans emptied
      c. Watches and pens out
      d. Emergency life-saving devices checked
         (1) Defibrillator blades
         (2) Emergency medical kit and drugs
         (3) Respirator
      e. Nitrogen purging system
         (1) Lines connected
         (2) Nitrogen on at desired pressure
   9. Recorders
      a. Pressure, temperature, humidity, O_2 partial pressure, and CO_2 partial pressure recorders balanced and in working condition
      b. All clocks synchronized
   10. Communication system
      a. On and warmed up
      b. Functioning

C. Physiological Recorder
   1. All channels recording

D. Blood-Gas Analyzer
   1. On and standardized

E. Vacuum System
   1. On with regulator set

F. Command—Standby for Pressure
   1. Pressure valve open and clocks started
   2. Exhaust valve set to selected ventilation

EMERGENCY MEDICAL KIT

In addition to the routine medical supplies within the chamber, which may vary with type of patient and treatment, each compartment of a hyperbaric medical chamber should contain

a basic medical kit to be kept within the chamber at all times in case of an emergency. Suggested equipment and drugs for these kits are as follows:

Equipment

Stethoscope
Sphygmomanometer
Sterile tracheostomy tubes, set
Syringes—30 cc, 20 cc, 10 cc
Needles
Three-way stopcocks
Tongue blades
Percussion hammer
Tuning fork
Ophthalmoscope
Oral airway
Endotracheal tubes
Thoracocentesis needles
Hemostats
Tourniquets
Intracardiac needles
Suction catheters
Sterile gloves
Oxygen masks
Flashlights
Defibrillator paddles—external and sterile internal

Drugs

Metaraminol Bitartrate (Aramine)
Levarterenol Bitartrate (Levophed)
Epinephrine (Adrenalin)
Phenylephrine Hydrochloride (Neosynephrine)
Cedilanid
Methoxamine Hydrochloride (Vasoxyl)
Isoproterenol Hydrochloride (Isuprel)
Calcium Chloride
Calcium Gluconate
Diacetylcholine Chloride (Anectine)
Sodium Bicarbonate
Sodium Lactate
Protamine Sulfate
Heparin Sodium
Digoxin (Lanoxin)
Procaine Hydrochloride

RECORD OF DIVE

Name	Group Letter	Date	Purpose
SANDRA FOSTER 6811		11/13/63	HEART OPERATION
DR. I.W. BROWN		Bank Pressures	
DR. C.R. STEPHEN		Compressed Air	Oxygen	Nitrogen
DR. F.M. MAUNEY		Start / Finish	Start / Finish	Start / Finish
DR. R.P. FUSON		2200 / 2100	1575 / 1550	950 / 925
DR. J. WARSHAW		Depth	Total Bottom Time	Table
LAURA BALLARD		85	79	90/80
NORMA MANN		Total Decompression Time	Total Time of Dive	Group Letter
		60	139	N

LS	1047
R 68	1056
L 68	1112
R 85	1114
L 85	1201
L 30	1206
R 20	1207
L 20	122
R 10	1221
L 10	1301
RS	1306

Remarks

DE Cameron
Operator

Figure 1.

HYPERBARIC LOG FORM

NAME_____ Duke History # _____
(last, first, middle)

Date	Depth	Bottom time	Inhalation mixture	Table used for decompression	Adverse reaction	Initial

Figure 2.

DUKE UNIVERSITY MEDICAL CENTER

PATIENT
PERMISSION FOR HYPERBARIC
OXYGENATION PROCEDURE

Date_____

Time_____

I authorize Dr._____ and such assistants as he may designate to administer hyperbaric oxygenation treatment to _____. The nature of this treatment and the risks involved despite precautions have been thoroughly explained to me. I voluntarily accept the risks involved and agree that the above-named physician, his assistants, Duke Hospital, and its personnel shall assume no responsibility for the results of this treatment or its interpretation.

_____
Patient or Person Authorized
to Consent for Patient

Witness:

_____

Figure 3.

VOLUNTEER PERMISSION FOR HYPERBARIC
OXYGENATION PROCEDURE

Date_____

I understand that this research is for experimental purposes, and results cannot be fully foreseen. Preliminary tests have been made and indicated precautions to protect volunteers have been taken. The hospital does not represent that any injury will necessarily be avoided in every instance even when these precautions are followed. Nevertheless, I voluntarily assume the risk involved, in order to advance medical knowledge. I will carefully follow instructions given for the conduct of the experiment. I will not make any claim or demand upon the hospital or its personnel for injury, if any arises from the experiment. This does not relieve the hospital of negligence in the performance of the experiment.

I agree that data obtained from this experiment may be used for medical or other scientific purposes, including publication, but my identity will not be revealed unless I expressly consent thereto.

I also authorize my admission to the Clinical Research Ward if deemed advisable by my physician.

_____
Signature

Figure 4.

APPENDIX A

CONVERSION TABLE FOR EXPRESSION OF DEGREES OF PRESSURE

John C. Carter

The following conversion table relates four commonly used measurements for the expression of degrees of pressure experienced in hyperbaric medicine and underwater exposure.

Column I

Expresses the values in mm. Hg and is the preferred "standard."

Column II

Presents pressure equivalents to mm. Hg in terms of absolute atmospheres. One atmosphere absolute is equal to the pressure of air at sea level (760 mm. Hg).

To convert to mm. Hg:

Multiply psig x 51.7 + 760
Multiply atm. abs. x 760
Multiply ft. sea water x 23.0 + 760

To convert to atm. abs.:

Multiply psig x 0.68 + 1
Multiply mm. Hg x .00132
Multiply ft. sea water x .0303 + 1

Column III

Utilizes feet below the surface of sea water as compared to mm. Hg.

Column IV

Expresses pressure in terms of pounds per square inch absolute, up to the pressure of air at sea level (14.69 psi). Following this, all expressions are in terms of pounds per square inch guage or the readings that will show on normal gas pressure guages.

Four formulas are presented for converting each of the values to the other three.

To convert to ft. sea water:

Multiply psig x 2.25
Multiply atm. abs. x 33 - 33
Multiply mm. Hg x 0.043 - 760

To convert to psig:

Multiply atm. abs. x 14.696 - 14.696
Multiply mm. Hg x .0193 - 14.696
Multiply ft. sea water x .445

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CONVERSION CHART

mm. Hg	Atm. Abs.	Ft. Sea Water	psia
250	.3290	_____	4.834
500	.6579	_____	9.668
760	1.0	_____	14.69
			psig
990.3	1.303	10.0	4.453
1000	1.316	10.42	4.640
1221	1.606	20.0	8.906
1277	1.680	22.5	10.0
1451	1.909	30.0	13.36
1500	1.973	32.13	14.31
1520	2.0	33.0	14.70
1681	2.212	40.0	17.81
1794	2.361	45.0	20.0
1912	2.515	50.0	22.30
2000	2.632	54.0	23.98
2142	2.820	60.0	26.72
2280	3.0	66.0	29.40
2311	3.041	67.5	30.0
2372	3.121	70.0	31.2
2500	3.290	75.5	33.65
2603	3.424	80.0	35.63
2833	3.730	90.0	40.0
3000	3.950	97.26	43.31
3040	4.0	99.0	44.09
3063	4.03	100.0	44.53
3294	4.333	110.0	48.99
3346	4.402	112.5	50.0
3500	4.605	119.0	52.99
3524	4.64	120.0	53.44
3754	4.94	130.0	57.90
3800	5.0	132.0	58.78
3863	5.082	135.0	60.0
4000	5.263	140.6	62.65
4380	5.763	157.5	70.0
4500	5.921	162.4	72.32
4560	6.0	165.0	73.48
4897	6.443	180.0	80.0
5000	6.580	184.1	81.99
5320	7.0	198.0	88.18
5414	7.124	202.5	90.0
5500	7.24	205.8	91.66
5931	7.8	225.0	100.0
6000	7.9	227.5	101.3
6080	8.0	231.0	102.8
6449	8.5	247.5	110.0
6500	8.552	249.2	111.0
6840	9.0	264.0	117.6
6979	9.165	270.0	120.0
7000	9.21	270.9	120.6
7483	9.845	292.5	130.0
7500	9.868	292.6	130.3
7600	10.0	297.0	132.2
8000	10.53	314.3	140.0
8157	11.20	337.5	150.0

APPENDIX B

ERRATA: Because of errors appearing in Appendix B of the first edition of Fundamentals of Hyperbaric Medicine, pages 157 and 158 have been completely reprinted. Please cancel the pages which have been bound into the volume and substitute these corrected pages.

VENTILATION ANALYSIS IN HYPERBARIC FACILITIES

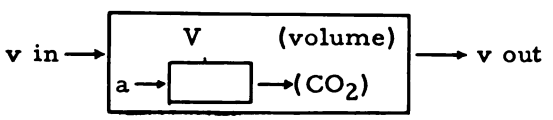
James V. Harrington

Ventilation-rate analysis for hyperbaric facilities is based on the rate of addition of contaminants to the chamber air, the total allowable concentration of contaminants, the chamber volume, and the time history of the addition of the contaminants.

Two special cases are of interest to the operators of hyperbaric facilities, one of which is the rate of buildup of CO_2, and the other, the rate of buildup of oxygen when a patient is breathing pure oxygen. Each of these situations is worked out and a typical example given.

Analysis of CO_2 Level in Hyperbaric Facility

A tank contains V cubic feet of air. Carbon dioxide is manufactured at a certain rate in this tank using some of the air in the process. The tank is being ventilated or flushed with air at a certain rate. Complete mixing of air and CO_2 in the tank is assumed. What is the relationship between the CO_2 level in the tank and the ventilation rate?



In the diagram,

v = air flushing rate cu ft/sec
V = volume of tank cu ft
a = rate of manufacture of CO_2 in tank cu ft/sec
X_c = mole fraction of CO_2
t = time, sec

Then the differential equation becomes

$$\frac{dX_c}{dt} + \frac{v}{V} X_c = \frac{a}{V} \quad (X_c = 0 \text{ when } t = 0)$$

Which has the solution

$$X_c = \frac{a}{v}\left[1 - e^{-\left(\frac{v}{V}\right) t}\right]$$

And at $t = \infty$ $v = a/X_c$

Typical Example:

CO_2 production per person 0.000161 cu ft/sec at STP*

CO_2 partial pressure should not exceed 10 mm Hg

Allowable X_c at different pressures is shown in Table 1 and the ventilation rate per person is also shown.

Table 1

$$X_c = \frac{10}{\text{mm Hg (total pressure)}} \qquad v = a/X_c$$

1.315% at 1 atm	0.735 cfm/person
0.658% at 2 atm	1.470 cfm/person
0.438% at 3 atm	2.20 cfm/person
0.329% at 4 atm	2.94 cfm/person
0.263% at 5 atm	3.67 cfm/person
0.219% at 6 atm	4.40 cfm/person

It should be noted that this computation is based upon the maximum allowable value of X_c and represents minimum tolerable ventilation, not a desirable level of ventilation.

As shown in Table 2, a computation has been worked out for the time required to attain maximum allowable CO_2 concentration for the following conditions:

Intervals of 10; 100; 1000; 10,000; 20,000; 30,000; 40,000; and 50,000 seconds, with a pressure of 4 atm in the chamber and six people. From Table 1 we see that a minimum flow of 2.94 cfm per person is required at pressure.

This becomes 2.94 x 6 = 17.64 cfm at pressure, and 4 x 17.64 = 70.6 scfm.

It may be seen from the computation that the chamber will reach the maximum allowable CO_2 level under these conditions and maintain it after approximately 14 hours.

*Corresponds to 16.4 liters per hour at STP

Table 2

COMPUTATION OF CO_2 VERSUS TIME

Time Sec	X_c	Hr	Min	Sec
10	0	0	0	10
100	0.0038	0	1	40
1,000	0.032	0	16	40
10,000	0.205	2	46	40
20,000	0.283	5	33	20
30,000	0.322	8	7	0
40,000	0.328	10	53	40
50,000	0.329	13	40	20

Oxygen Addition to Atmosphere

V = volume of tank cu ft
v_1 = rate of gas entering tank cu ft/min
v_2 = rate of gas leaving tank cu ft/min
v_o = rate of oxygen entering in ventilating gas cu ft/min
v_n = rate of nitrogen entering in ventilating gas cu ft/min
a = rate of oxygen administered to patient cu ft/min
X_o = mole fraction of oxygen in tank
t = time, min

Then, for steady state conditions

$$v_2 = v_1 + a = a + v_o + v_n$$

Instantaneous changes in concentration of gases in the tank atmosphere, assuming perfect mixing, are as follows:

$(a + v_o)\,dt$ = rate of oxygen entering tank

$\left[X_o\, v_2/V\right] dt$ = rate of oxygen leaving tank, and the differential equation becomes

$$dX_o = \left[\frac{(a + v_o)}{V} - X_o \frac{(a + v_o + v_n)}{V}\right] dt$$

which has the solution

$$X_o = \left[\frac{v_o}{v_o + v_n} - \frac{(a + v_o)}{a + v_o + v_n}\right] e^{-\left[\frac{(a + v_o + v_n)t}{V}\right]} + \frac{a + v_o}{a + v_o + v_n}$$

$$\text{at } t = 0 \quad X_o = \frac{v_o}{v_o + v_n}$$

$$\text{and at } t = \infty \quad X_o = \frac{a + v_o}{a + v_o + v_n}$$

Typical Computation

rmv (respiratory minute volume) 0.71 cfm (20 liters/per minute)

O_2 partial pressure should not exceed 25 per cent of total pressure

Assume a chamber volume of 3000 cu ft

$X_o = 0.25$ (max. allowable)

$a = 0.71$

$v_o = 0.21\, v_1$

$v_o + v_n = v_1$

Then

$$0.25 = \frac{0.71 + 0.21\, v_1}{0.71 + v_1}$$

and $v_1 = 13.3$ cfm, the required ventilation rate to attain a maximum X_o of 25 per cent.

The rate of buildup of oxygen concentration in this chamber becomes:

$$X_o = \left[0.21 - \frac{0.71 + .21\, v_1}{0.71 + v_1}\right] e^{-(0.71 + v_1)t} + \frac{0.71 + 0.21\, v_1}{0.71 + v_1}$$

and substituting 13.3 cfm for v_1 this may be further reduced to

$$X_o = \left[0.21 - \frac{3.503}{14.01}\right] e^{-0.00467\, t} + \frac{3.503}{14.01}$$

or

$$X_o = -.04\, e^{-0.00467\, t} + 0.25$$

Table 3

OXYGEN CONCENTRATION WITH TIME

Time (t) min	X_o	Per Cent O_2 (100 X_o)
0	0.21	21
10	0.212	21.2
100	0.225	22.5
500	0.246	24.6
1,000	0.249	24.9
10,000	0.250	25.0

APPENDIX B

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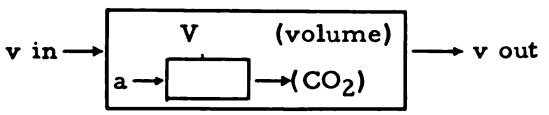

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