VII

Decompression Sickness

A. *Introduction*

Man first began to spend substantial amounts of time under increased pressure soon after the development of the air compressor, early in the nineteenth century. The majority of such exposures occurred in European tunneling and caisson work. The process of decompressing took some time because of the cumbersome air locks and the general need to climb many steps back to the surface. During and after these decompressions men were noted to exhibit symptoms of distress but this was generally attributed to the unhealthy gas environment that resulted from poor ventilation, candle-burning, and tunneling under marshes where trapped sulfurous gases were often found.

As machinery and methods improved, the tunnels became longer and were often much deeper. The decompression was shortened because improved air locks permitted rapid decompression. These changes in work habits resulted in a major increase in the incidence of crippling and fatal sequelae. Since air quality in the work areas had been substantially improved, attending physicians had to look elsewhere for the causative factor; they soon decided that the decompression was too short for the men to become reacclimated to the lower pressure. Indeed, prolonged decompression did substantially reduce the incidence of severe symptoms.

The salient features of decompression sickness (DCS) were established during the period from 1870 to 1910. Hoppe's early suggestion that sudden death in compressed air workers was due to liberation of gas from blood and tissues was confirmed by the work of Paul Bert. In 1878 Bert performed the first definitive studies oriented toward understanding the processes involved in reacclimation. He observed that bubbles formed within animal tissues following rapid decompression and that bubbles, if present in adequate numbers, could kill or paralyze the animals. The higher the pressure and the longer the exposure, the greater was the ensuing volume of bubbles. Slow decompression suppressed bubble formation. These findings have been repeatedly confirmed and form the basis of our understanding of the malady that we now call *decompression sickness*.*

* Other names used in recent times include dysbarism and "bends."

The requirement of a slow decompression represents a major limitation on man's ability to dive safely to great depths. This limitation is always contrary to the goal of the planned activity. Traumatic injuries at diving depths represent a major hazard because treatment must await the completion of a slow decompression. Thus, enormous effort has been expended to develop the most rapid and safe decompression methods.

J. S. Haldane and his associates (Boycott *et al.* 1908) attempted to generalize the required decompression schedule in terms of the exposure depth and bottom time* so as to be able to calculate the so-called "decompression schedule" for any set of diving circumstances. Using goats, they found that so long as the pressure was less than 2 ATA, the goat could return to the surface directly without any ill effects.

The Haldane generalization postulated that the body absorbs excess nitrogen when exposed to increased pressure and that this is the source of gas needed to generate bubbles during decompression. A slow decompression permits the exhalation of the excess nitrogen, thereby inhibiting bubble formation. Nitrogen is distributed throughout body tissues according to the relative solubility and blood flow to that tissue.

Rapid decompression slows nitrogen elimination and thus allows more nitrogen to remain in the body for bubble formation. Neither Haldane nor anyone since has successfully validated or repudiated the model. The concept has been extremely useful in guiding research, and the model has been extensively used to correlate empirical diving results.

The military and commercial demand for shorter decompression times has been met with the development of procedures that result in an acceptably low incidence of decompression sickness. In order to achieve this low incidence, diving medical officers have also evolved an extensive set of limitations on the divers and the equipment they use. The evaluation of these procedures and limitations has involved empirical data based on extensive human experimentation.

The recent availability of scuba equipment has led to an enormous amount of diving for sport or science that is not subject to the same controls. It is a considerable testimony to the existing procedures that they have worked so well for scuba divers. Table VII-1 lists a series of hyperbaric exposures that result in demands for decompression procedures that pose special problems in the use of existing methods. It would be fortuitous if the empirical techniques that are now available could be extrapolated to all diving situations; but it is highly improbable.

In view of this situation, this chapter is organized to provide the basic information needed to understand existing decompression procedures and their limits. Diagnosis and treatment of decompression accidents are detailed and sources of advice and assistance are identified. This chapter is outlined to help in planning a program of hyperbaric exposure so as to avoid a decompression accident. Such planning inevitably leads to a restriction on the amount of exposure one would like to have in order to accomplish a job. Yet experience has shown that a philosophical acceptance of this limitation is highly preferable to the risk of a severe decompression catastrophe.

This chapter will deal only with decompression sickness. Other forms of barotrauma, such as air embolism, are discussed in Chapter III.

It will become evident that equipment design and performance play a key role in

* See the section on prevention, this chapter, for a detailed definition of these terms.

Table VII-1
Hyperbolic Exposures for Which Decompression Schedules Have Not Been Expressly Developed

Hyperbaric exposure	Special features not tested in available schedules
Hyperbaric medicine	a. Patients with altered circulation and no experience in hyperbaric exposure and excessive O_2 exposure
	b. Physicians and professional staff who may be older and in poorer physical condition than normal divers and therefore not able to tolerate the same risks
Scuba sport diving	a. Age and physical condition of divers
	b. Lack of appropriate treatment facilities (chambers, physicians, extra gas, medicine)
	c. Variable pressure exposure
	d. Increased hazard of underwater accident
Scientific diving	a. Lack of appropriate treatment facilities due to (generally) lower financial support
	b. Variable pressure exposure
	c. Increased bottom time
	d. Increased hazard of underwater accident due to (1) use of scientific equipment; (2) need to observe dangerous life forms in more dangerous environments (caves, canyons, etc.)
Deep water diving	a. Depths are so great that pressure, per se, may alter biological response to decompression
	b. Need to use habitats (gas-phase decompression)
	c. Appearance of decompression sickness at depths greater than the normal treatment depths
	d. Existence of pressure-related symptoms that complicate diagnosis of decompression sickness (compression arthralgia, HPNS, narcosis)

the prevention of decompression sickness. The provision of a diver with a consistent life support system (respiratory, thermal, guidance, propulsive, etc.) is an excellent way to minimize the risk in any given diving operation.

B. *Factors Relevant to the Pathogenesis of Decompression Sickness*

Decompression sickness (DCS) occurs when man is subjected to reduced environmental pressure that causes bubbles of inert gas to form and grow within tissues or the vascular space. Physical factors relevant to the nucleation and growth of bubbles are of importance, as they permit one to assess the environmental conditions that would lead to the greatest risk of DCS (pressure, temperature, noise). Biological factors that influence the transport of inert gas throughout the body can be characterized. Responses of the body to the insult of bubble formation are less well known.

This section will discuss the underlying physical and biological factors that have been shown to be relevant to the process of bubble formation, growth, and dissolution

as a result of hyperbaric exposures. Biological factors relevant to the pathology of DCS are well described in the medical literature.

Note that the extensive studies on decompression sickness that have been made in association with altitude (hypobaric) exposure are not always relevant to hyperbaric exposures. They will be cited only where the factors have been shown to be of importance in hyperbaric environments.

1. *Physical Factors*

a. Inert Gas Solubility

During hyperbaric exposure, the partial pressure of respired inert gases is generally different than it is at atmospheric pressure with air ($P_{N_2} = 0.79$ ATA). As shown in Chapter III, the partial pressure of an inert gas in a breathing mixture is related to the environmental pressure P_{abs} by

$$P_x = F_x P_{abs} \tag{1}$$

where F_x is the fraction of the inert gas in the gas mixture. It is generally assumed that the ideal gas law $PV = nRT$ is applicable to inert gases in diving environments, validating Eq. (1). See Chapter IV for further details on the properties of inert gases in hyperbaric breathing mixtures.

All the inert gases (He, H_2, N_2, Ne, Ar) dissolve in blood and tissue according to Henry's law, viz.

$$C_x = \alpha_x P_x \tag{2}$$

where C_x is the dissolved concentration [generally expressed as cm^3 (STP)/cm^3 fluid] and α_x is the absorption constant [cm^3(STP)/cm^3 fluid atm] for the inert gas denoted by the subscript x. Thus, when an individual is exposed to an elevated partial pressure of inert gas, the inert gas tends to be absorbed into the body through the lungs and to dissolve into the tissues of the body until they are equilibrated with the respired gas.

After a period of time, equilibration occurs [as per Eq. (2)] and no more gas may dissolve. Thus, at any elevated pressure there is a maximum of gas that may dissolve in the body tissues.

The Bunsen solubility coefficient α_x depends on the temperature, type of tissue or fluid, and type of inert gas. Over the normal range of diving, it is *not* a function of absolute pressure (Poynting effect), partial pressure of the inert gas, or the presence of other inert gases.*

The temperature dependence of the solubility coefficient α_x is given by

$$\alpha_x = \alpha_{x,0} e^{-\Delta H/RT} \tag{3}$$

where ΔH is the heat of solution, R is the gas constant, and T is the absolute temperature in degrees Kelvin (T °K = 273 + T °C). Table VII-2 presents the preexponential

* While these statements have been experimentally validated for water at depths as deep as 50 ATA, they have not been fully tested for biological fluids; therefore, at the recently attained depths of 50 ATA there may be some influence of pressure on solubility. Its significance in developing decompression procedures is probably negligible.

Table VII-2
Solubility Parameters for Various Gases in Water
$\alpha_x = \alpha_{x,0} e^{-\Delta H/RT}$

Gas x	$\alpha_{x,0}$, cm³ (STD)/cm³ ATA	ΔH, kcal/g mole	α_x(37.0°C)	Reference
N_2	2.210×10^{-4}	2,465	12.30×10^{-3}	Douglas 1964
He	8.193×10^{-9}	8,520	8.66×10^{-3}	Weiss 1971
H_2	1.830×10^{-8}	8,422	16.70×10^{-3}	Morrison 1952
Ne	5.317×10^{-10}	10,256	9.61×10^{-3}	Douglas 1964
Ar	2.721×10^{-4}	2,777	25.90×10^{-3}	Douglas 1964
O_2	2.645×10^{-4}	2,757	23.60×10^{-3}	Douglas 1964

factor $\alpha_{x,0}$ and ΔH for the various inert gases in water. Since ΔH is a positive number, the solubility decreases at elevated temperatures.

The solubility of inert gases is greater in fat tissue than in lean tissue, due to the tendency for greater solubility in lipoidal fluids. Table VII-3 tabulates inert gas solubility for blood, lean tissue, fat tissue, and for a lipoidal fluid (olive oil) at 37°C. Tissues behave very similarly to fluids in their solubility characteristics. The solubility increases with increasing molecular weight of the gas, except for H_2. Similarly, the partition coefficient between olive oil and water ($\lambda = \alpha_{0.0.}/\alpha_{water}$) also increases with molecular weight of the gas.

Thus, fat tissues represent large reservoirs for all the inert gases and the absolute amount held increases with the molecular weight of the gas being inhaled. On the other hand, the partitioning of the gas from the gas phases into the liquid requires that few molecules go into the liquid in order to saturate it. For example, the volume of bubbles that could form if one saturates 100 cm³ of fat tissue with air is 4.2 cm³. Haldane and others (Boycott *et al.* 1908) have established that a dive involving a 1-atm change in pressure is apparently safe for man. We may thus surmise that: (a) this volume loading of gas is tolerable, (b) that bubbles do not form, or (c) there is some variant in between these extremes. The question of bubble nucleation and growth is presented in the next subsection.

Table VII-3
Solubility Coefficients for Inert Gases in Biological Fluids at 37°C

Gas	Fluid			
	Blood[a] α	Lean tissue α	Fat α	Olive oil α
H_2	—	—	—	0.0484
He	0.0159	—	—	0.0159
N_2	0.0130+	0.012+	0.062+	0.067
Ar	—	—	—	0.14
Ne	—	—	0.020	0.019
O_2	0.0223	0.023	—	0.112
CO_2	0.488	—	—	1.25

[a] 150 g Hb/100 ml.

b. Bubble Nucleation and Growth

In order to transpose dissolved gas within a pure liquid into a bubble within that liquid, the physical environment of the liquid must be changed so that the bubble-containing state can form spontaneously. Thermodynamic arguments guide this process. A few examples serve to introduce the problem.

In Figure VII-1(a) a liquid is in equilibrium with N_2 gas at 2 ATA within a cylinder that is isolated by a diaphragm that transmits ambient pressure. The valve is closed in (b); the concentration of dissolved gas in the liquid is then equal to 2α and the system is at equilibrium.

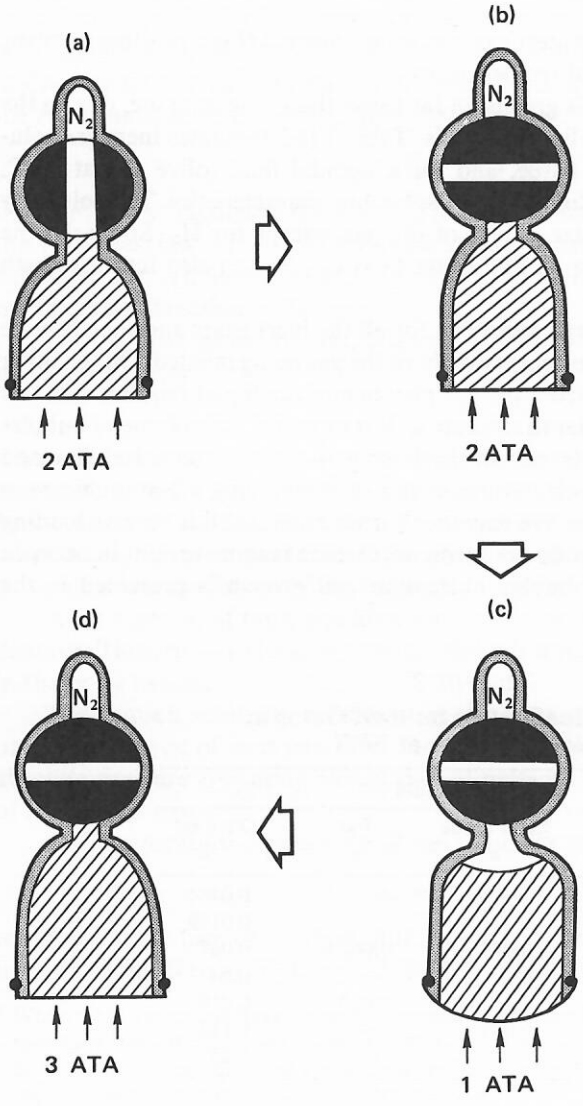


Figure VII-1. The equilibrium between an inert gas and water in a closed system. The two-phase system is isolated from the exterior environment by the container. Pressure is transmitted to the lower end across an impermeable diaphragm. The valve may be rotated to isolate the liquid from the gas phase.

If the ambient pressure is reduced to 1 ATA [Figure VII-1(c)], bubbles form and grow by the process of N_2 diffusion from the liquid. The bubble will grow until the concentration of dissolved N_2 is α; at that point equilibrium is reestablished. The diaphragm distends to compensate for the increased volume below the valve.

If the ambient pressure is then raised to 3 ATA [Figure VII-1(d)], the bubbles will dissolve in the liquid. When totally dissolved, the concentration of N_2 is again 2α. The system is less than saturated but at pressure equilibrium so long as the valve remains closed. If the valve is now opened so that the liquid is in contact with the gas, gas will dissolve in the liquid until it is saturated (i.e., the concentration is 3α).

Thus, Figure VII-1 demonstrates the principle that when the absolute pressure on a fluid is decreased below the partial pressure of the dissolved gas, there is a tendency for the gas to leave the solution going into the gas phase which exists at a lower pressure. It will do this until the partial pressure in the gas phase equals the dissolved partial pressure (1 atm, in this case). It also shows that bubbles may be dissolved by raising the ambient pressure to a value where the equilibrium state results in subsaturation.

There are other ways to produce supersaturation. If we equilibrated a liquid with pure N_2 at 1 atm at 25°C in the equipment shown in Figure VII-1(a), the dissolved gas concentration would be

$$C_{N_2} = \alpha_{N_2}{}^{25^\circ} = 0.02 \text{ cm}^3\text{(STP)/cm}^3$$

If the valve were then closed and the temperature raised to 37.0°C, the partial pressure of N_2 in the liquid would now be

$$P_{N_2}(37^\circ) = C_{N_2}/\alpha_{N_2}{}^{37^\circ} = 0.02/0.0127 = 1.5 \text{ ATA}$$

but the absolute pressure is still only 1 ATA. So this solution is also supersaturated [in the same sense as supersaturation was produced in Figure VII-1(c)]; a gas phase would again tend to form so that enough N_2 would diffuse out of the liquid to reduce its partial pressure to 1 ATA. So nucleation of a gas phase tends to occur whenever the N_2 partial pressure in the water exceeds the ambient pressure, regardless of how we attained this state of affairs.

Yet, nucleation and growth are kinetic processes (i.e., they proceed at a finite rate). Thus, through equilibrium considerations we can describe the final state of a process that involves nucleation and growth. But we must use kinetic equations to describe the rate of these processes. The body is only transiently supersaturated, so it is essential to estimate nucleation and growth rates if we are to be able to assess the potential hazard of a decompression procedure.

Since all kinetic processes are described by a general equation, we may seek to characterize nucleation and growth of bubbles in this way. The general kinetic equation is

$$\text{rate of a process} = \frac{\text{driving force for the process}}{\text{resistance to the process}} \tag{4}$$

or

$$J_i = \frac{\Delta E_i}{R_i} \tag{5}$$

where i denotes the process.

In bubble nucleation processes the driving force is represented by the difference between the dissolved-gas partial pressure and the absolute pressure $P_x - P_{abs}$. By convention, whenever the difference is positive, the rate of bubble nucleation is positive and bubbles will continue to form.

The resistance to nucleation RN cannot be theoretically predicted, because of our imprecise knowledge of the thermodynamic states of water. However, it is known empirically that several factors do influence the process; it occurs much more rapidly at elevated temperatures, and it occurs much more easily at a liquid–surface interface. This is especially true if the interface is hydrophobic.*

De novo bubble formation rates have been estimated. Under diving conditions the rates would appear to be so slow as to preclude bubble formation. The process may be greatly accelerated by ultrasonic energy, as it acts to transiently produce substantial reductions in the local fluid pressure. This occurs because the driving force for the nucleation rate is $P_x - \bar{P}_{abs}$, and $\bar{P}_{abs}$, the local fluid pressure, is now given by

$$\bar{P}_{abs} = P_{abs} + P_u \sin(\omega t) \tag{6}$$

where P_u is the amplitude of the ultrasonically produced pressure wave of frequency ω^{-1}. Whenever P_u is large compared to P_{abs}, the nucleation rate is enhanced. This occurs because the negative segment of the acoustic wave produces bubble growth at a greater rate than that of the bubble dissolution produced by the positive segment.

Sources of ultrasonic energy are abundantly available in the diving environment; noise and vibration are predominant external sources. The acoustic inhomogeneity of the body makes it an excellent site for acoustic focusing effects that would tend to amplify a normally weak ultrasonic wave.

As the dissolved gas concentration increases, the energy needed for cavitation decreases. Figure VII-2 shows the cavitation threshold for water and benzine; 100% saturated fluid at 1 ATA total pressure requires only 1 atm of ultrasonic energy to induce cavitation.

As the hydrostatic pressure is increased on a fluid saturated at 1 ATA, the ultrasonic energy required for cavitation increases proportionately (Figure VII-3). Cavitation is *not* strongly temperature dependent. As the ultrasonic frequency increases, the required cavitation energy increases exponentially. Thus, one is generally concerned with sound sources below 100 kHz; higher frequency sources are generally too weak to cause cavitation.

Transient ultrasonic pulses can induce nucleation, but generally more energy is required. Figure VII-4 shows that a 0.1-sec pulse will cavitate a liquid, but it requires about ten times the power required under constant-irradiation conditions.

Viscous fluids and tissues (*gels*) require more ultrasonic energy for bubble formation. Figure VII-5 shows that the power requirements increase as the logarithm of the fluid viscosity.

Some investigators have suggested that bubble nucleation rates would never be a rate-limiting step in bubble formation because there are always micronuclei that exist. Thus, bubble appearance merely depends on the growth of the micronuclei in the

* It also appears the RN is a function of the degree of supersaturation. Thus, at low degrees of supersaturation the process is exceedingly slow; at higher values, it increases rapidly and then the process is substantially proportional to the degree of supersaturation.

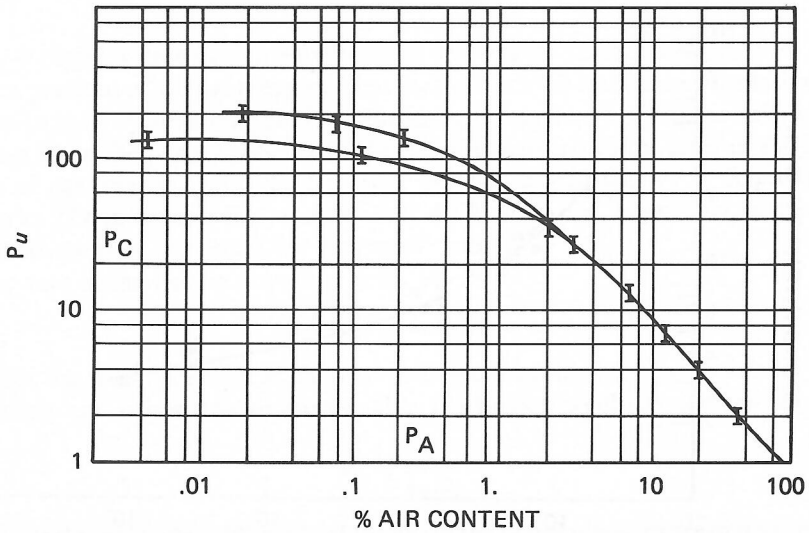


Figure VII-2. Cavitation threshold of water (upper curve) and benzine (petroleum ether) (lower curve) as a function of percentage of gas concentration at a hydrostatic pressure of 1 atm and temperature of 22°C. P_u is the cavitation threshold in bars.

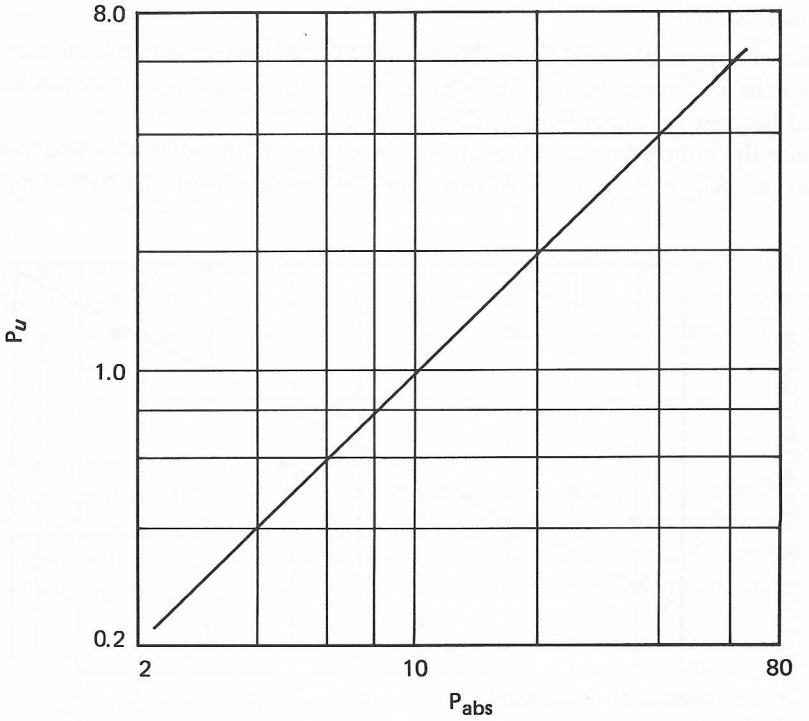


Figure VII-3. Cavitation threshold of air-saturated water as a function of hydrostatic pressure. P_u is the cavitation threshold in bars; P_{abs} is the atmospheric pressure in bars. $T = 22°C$.

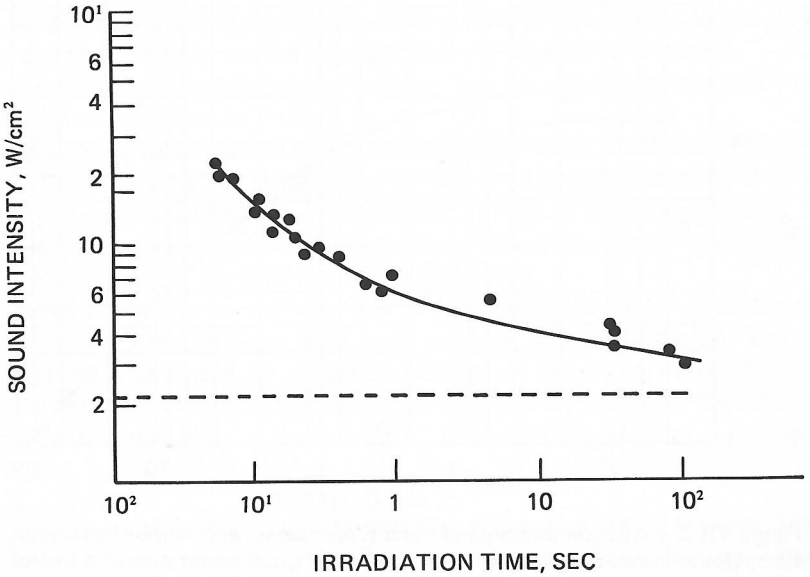


Figure VII-4. Average sound intensity as a function of time between switching instrument on and start of cavitation. Excitation frequency is 365 kHz in tap water. The intensity below which no cavitation appears in 15 min of irradiation is shown as a dashed line.

presence of a supersaturated tissue. Harvey (1951) demonstrated that such micronuclei do not exist in human blood. Their presence in peripheral tissue has recently been inferred but not yet unequivocally demonstrated.

Once the bubble forms and reaches a size of about 0.1 μm, it is viewed as a stable bubble (i.e., $2\gamma/r < P_x - P_{abs}$). At this point the pressure inside the bubble P_B is given

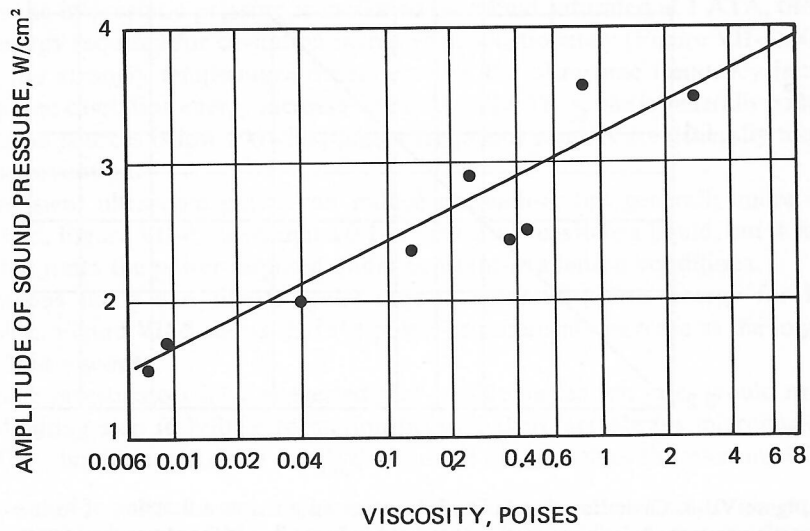


Figure VII-5. Amplitude of cavitation sound pressure against viscosity of liquid.

by $P_{abs} + (2\gamma/r)$, where r is the bubble radius and γ is the surface tension. If $P_B < P_x$, there is a gradient of partial pressure from the liquid to the bubble and the bubble will continue to grow by absorbing gas from the surrounding liquid. This process of growth will cease when the partial pressure in the liquid is reduced to the level of P_B (by the process of gas transport into the bubbles). Now $P_B \simeq P_{abs}$ when $r \geq 20\ \mu$m (i.e., in water, $\gamma \simeq 72$ dyn/cm and $P_{abs} - P_B = 0.07$ ATA when $r = 20\ \mu$m). So, surface tension effects are only important for very small bubbles.

The rate of the bubble growth is given by

$$J_G = (P_x - P_B)/R_G \tag{7}$$

Normally, the resistance term R_G is governed by diffusion of the gas molecules in the liquid phase and is inversely proportional to the Bunsen solubility coefficient

$$R_G = \frac{K_G}{\alpha_x D_x} \tag{8}$$

where D_x is the molecular diffusivity of the gas in the liquid phase, expressed as cm^2/sec. The constant of proportionality K_G is dependent on the bubble geometry, the presence of other bubbles, and the location of the bubble in the tissue. Table VII-4 gives the diffusivity D_x of various gases in fluids and tissues.*

It is not possible to predict diffusivities for liquid solvents based on molecular considerations. Tissue diffusivities are unavailable. However, one can estimate diffusivities based on the well-known Stokes–Einstein equation, $D = kT/6\pi\mu r$, where k is the Boltzmann gas constant, T is the absolute temperature, μ is the viscosity of the solvent, and r is the molecular radius of the solute molecule.

The temperature dependence of D is based on the ratio of T/μ. Since the logarithm of viscosity is generally inversely dependent on absolute temperature (i.e., $\ln \mu = B/T + A$), diffusivity will increase with absolute temperature, but in a complex manner.

The diffusivity of gases in tissue is normally determined by measuring the steady-state flux through a flat piece of tissue of known thickness. If the gas solubility is known, one can estimate diffusivity by use of the linear diffusion form of Eq. (7):

$$J_G = \frac{DA\alpha}{t} \Delta P \tag{9}$$

where A and t are the area and thickness of the tissue section that is exposed to a gas pressure gradient of ΔP. The diffusivity determined from Eq. (9) is a bulk average value. The relative contribution of diffusion through cells or between cells is unknown.

The product $D\alpha$ is often called the "permeability" of the tissue.† This is a useful term for heterogeneous media because of the uncertainties in estimating α.

* There are some reports in the literature that D_x in tissue may be much less than in plasma. The argument revolves around the methods whereby the diffusion coefficient is measured (steady-state vs dynamic methods). Presentation of the data in Table VII-4 is offered to show the influence of gas molecular weight and tissue type, using the same measurement method.

† Many early investigators referred to permeability as the ratio $J/(A\ \Delta P)$. This is really the "specific flux density" and is of little use because the tissue thickness is seldom reported in these studies.

Table VII-4
Diffusion Coefficients of Gases in Fluids and Tissues $T = 37°C$

Gas	Fluid/tissue	$D \times 10^5$ cm^2/sec
H_2	Water	3.04
H_2	Serum	2.68
N_2	Water	1.32
N_2	Serum	1.15
O_2	Water	1.82
O_2	Lung	1.38
O_2	Connective tissue	0.58

Notice that if nucleation must occur to create bubbles, at least one dissolved gas must be present in a supersaturated form. Recent results at great depth where pressure was not altered, but He and N_2 were exchanged in the breathing mix, resulted in symptoms normally associated with subcutaneous bubbles (see the section on diagnosis, this chapter). The gases were switched in such a way that the total dissolved gas partial pressure ($P_{N_2} + P_{He}$) would have been substantially elevated in subcutaneous tissue. If micronuclei existed, bubbles could have resulted by increased growth rates. No such anomalous appearance of symptoms has ever been reported in subsaturation, low-pressure diving where only one inert gas was involved.

In conclusion, bubble nucleation or growth may be important in the production of *in vivo* bubbles during decompression procedures. No generalizations can be made that will assist in determining which factor is the dominant one for a given type of dive. Yet dive procedures are often selected that would be safe based on the dominance of nucleation as the rate-limiting step, yet would be unsafe for a growth-dominated process of *in vivo* bubble production.

2. *Biological Factors*

The whole process of inert gas dissolution into body tissues begins at the interface with the environment. Inert gases enter the body mainly through the lungs; transdermal transport, while finite, is probably only important when one is concerned with subcutaneous bubbles. Absorption of gas from trapped gas (e.g., intestinal) does occur, but is generally not a factor because of the limited amount of gas (see Chapter III for complications of trapped-gas pockets during decompression).

Dissolved gases are carried by blood flow from the lungs to the peripheral tissues, where they diffuse into tissue cells. During decompression, the process is reversed.

Ambient pressure is transmitted throughout the body. During compression and decompression certain (relatively) isolated anatomical zones may not equilibrate instantly, but the time lag for equilibration is probably minimal. One may assume, then, that by relating a gas partial pressure in a tissue to the environmental pressure one has the correct gradient for bubble nucleation and growth.

The biologically important factors are the dynamic processes of (a) pulmonary exchange; (b) blood flow and distribution between and within tissues; and (c) diffusion and convection in peripheral tissues.

a. Pulmonary Exchange

Inert gases are absorbed in the lungs by transalveolar diffusion into blood. The amount of inert gas needed to fully equilibrate the blood with the gas in the alveolar space is a small fraction of that present in the lungs under normal inflation. For example, the maximum rate of N_2 transport across the lung would be about 160 cm^3 (STD)/min. This is a small fraction of the 8 liters/min air flow in the lungs under normal conditions (see Chapter IV for a detailed discussion of respiratory exchange). Thus, inert gas absorption occurs without influencing pulmonary gas volumes; that is, the process of O_2 and CO_2 exchange is considered to be independent of inert gas exchange:

$$P_{A_x} = P_{abs} - P_{A_{O_2}} - P_{A_{CO_2}} - P_{H_2O}{}^{37^\circ} \tag{10}$$

One can estimate the inert-gas partial pressure in the alveoli from a knowledge of the respired gas mixture. The maximum contribution of $P_{A_{CO_2}}$ is 60 mm Hg, and this is partly compensated by $P_{A_{O_2}}$ being somewhat less than $F_{O_2} \times P_{abs}$. A reasonable approximation of Eq. (10) would be to neglect all factors but those due to the inert gas and O_2:

$$P_{A_x} \simeq F_x \times P_{abs} \tag{11}$$

where F_x is the inert gas fraction in the breathing gas. Equation (11) becomes even more valid at greater depths.

For the purposes of developing safe decompression schedules, we need to estimate the absorption of inert gases using assumptions that yield a degree of absorption that is equal to or greater than the actual amount. Equation (9) provides such a conservative estimation.

The oscillatory nature of respiration has little or no effect on decompression estimates. This is a valid approximation because the breathing frequency is 16 min^{-1} or more, while the highest frequency associated with inert gas transport within the body is about 0.2 min^{-1}; therefore, the respiratory oscillations are thoroughly damped by the much slower transport frequencies of the cardiovascular system.

The diffusional resistance of alveolar epithelium is so small that pulmonary venous blood is in essential equilibrium with alveolar gas (which, as we stated above, is in essential equilibrium with inspired gas). This seems to be a valid approximation for inert gas uptake; again, it represents a conservative estimation of gas absorption.

b. Transdermal Exchange

Inert gases are exchanged across the skin by diffusion based on the following equation:

$$J_x = \frac{D_x \alpha_x A}{t} (F_x P_{abs} - P_x) \tag{12}$$

where A is the whole body area, t is the average skin thickness, and P_x is the mixed venous inert gas partial pressure. When a diver is submerged, the surrounding fluid has a low inert gas pressure (for N_2, 0.79 atm; for other inert gases, $\sim$0 atm). In these environments inert gas is lost from the diver.

Chamber decompressions that involve breathing a gas mixture from a mask (e.g., pure O_2) provide the setting where transdermal exchange may play a dominant role. The appearance of local skin reactions in divers who were breathing one inert gas while living in a habitat of another inert gas has been explained as being due to the additive effect of the two inert gases diffusing in opposite directions.

There is enough gas transport that Behnke (1969) was able to measure transcutaneous N_2 transport by measuring N_2 washout from the lungs with only a 1-ATA gradient in N_2 partial pressure.

During decompression in a chamber (where the skin is surrounded by high inert gas partial pressure), O_2 breathing may not be as useful as it would be in the water (where $P_i \sim 0$), because of transcutaneous gas transport. In general, experience shows that safe decompressions in such a chamber environment will often lead to unsafe schedules when subsequently tested in the cold water (whether effects are due to reduced local blood flow or gas diffusion is not known).

In most other diving environments transdermal exchange is neglected in the development of decompression procedures.

c. Blood Flow and Distribution between and within Tissues

During hyperbaric exposure the arterial blood carries inert gas to all the tissues of the body. Each tissue is presented with a limited amount of gas per minute, which is (in cm^3 of gas delivered to tissue i per minute) $J_i = C_a Q_i$, where Q_i is the blood flow rate into tissue i.

In the previous section we showed that $C_a = F_x P_{abs} \alpha_x$; thus

$$J_i = F_x \alpha_x P_{abs} \cdot f_i Q \tag{13}$$

where f_i is the fraction of the cardiac output $Q(cm^3/min)$ that is delivered to tissue i. The tissue cannot saturate any faster than inert gas is supplied by blood flow. If a tissue's rate of saturation is controlled by Eq. (13), it is called a perfusion-limited tissue.

The process of achieving a maximum of tissue saturation is shown schematically in Figure VII-6. The entering dissolved gas totally distributes between blood and tissue so the partial pressure of venous blood is identical to the average value of the tissue $P_{i,x}$. The ratio of $\alpha_x/\alpha_{i,x}$ is the partition coefficient for blood and tissue, λ_i, and is generally less than 1.0. A fatty tissue, where $\lambda_i \sim 0.2$, would saturate more slowly than a lean tissue with the same degree of blood supply because of the need to deliver more moles of gas to achieve equilibrium. Figure VII-6 also includes the dynamic mass balance model for inert gas within the tissue.

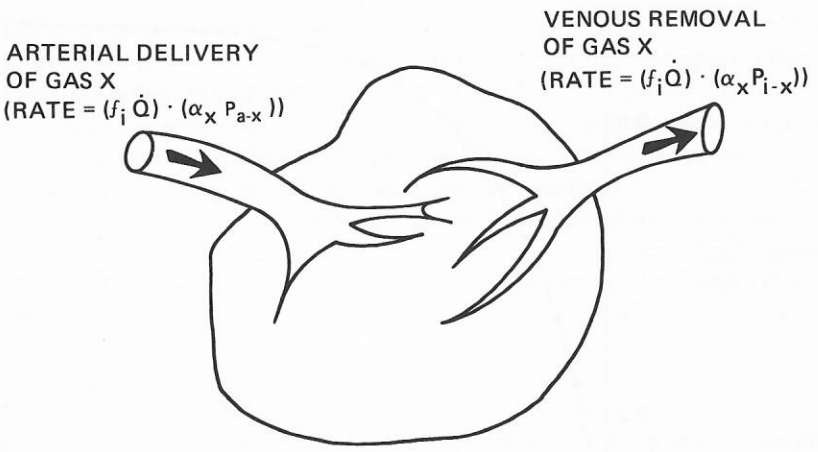
For the case where absorption occurs while the external pressure is constant over a long period of time, the tissue absorbs gas according to the following general equation (see Figure VII-7):

$$\left[\frac{P_a - P_i(t)}{P_a - P_i(0)}\right]_x = 1 - e^{-t/\tau_x} \tag{14}$$

where

$$\tau_x = \left(\frac{\alpha_i}{\alpha}\right)_x \left(\frac{\rho_i W_i}{1000 f_i Q}\right)$$

is the time constant for the tissue.



RATE EQUATION FOR UPTAKE & WASHOUT:

$$\frac{dP_{i-x}}{dt} = \left(\frac{1000 f_i \dot{Q}}{\rho_i W_i}\right)\left(\frac{\alpha_x}{\alpha_{i-x}}\right)\left(P_{a-x} - P_{i-x}\right)$$

Figure VII-6. Inert gas exchange in a perfusion-limited tissue.

In subsequent sections the time constant will not be directly expressed. Rather, we will use the more traditional factor, called the half-time of the tissue. It is defined as

$$t_{\frac{1}{2}x} \equiv (\ln 2) \times \tau_x \tag{15}$$

Any tissue in the body will absorb inert gas at either the rate defined for the perfusion-limited tissue or at a slower rate. The blood flow term ($\rho_i W_i / 1000 f_i Q$, expressed as cm^3 of tissue $\cdot$ min^{-1}/cm^3 of blood flow) has been measured for many tissues in the body by a variety of techniques. Table VII-5 is a summary of such flow rates based on the compilation of Jones (1951).

Tissue perfusion rates depend on exercise, inspired P_{O_2}, temperature, conditioning, nutritional state, subject size, and even (perhaps) the rate of change of environmental pressure. These influences have been generalized as follows:

1. Exercise increases muscle perfusion proportional to muscular work but causes a concomitant decrease in skin blood flow.
2. Elevated arterial O_2 partial pressures depress blood flow in almost all tissues that have been measured.
3. Decreased ambient temperature has little effect on central tissue blood flow but drastically reduces peripheral perfusion rates.
4. An aquanaut in good condition performs work at lower than normal levels of cardiac output.

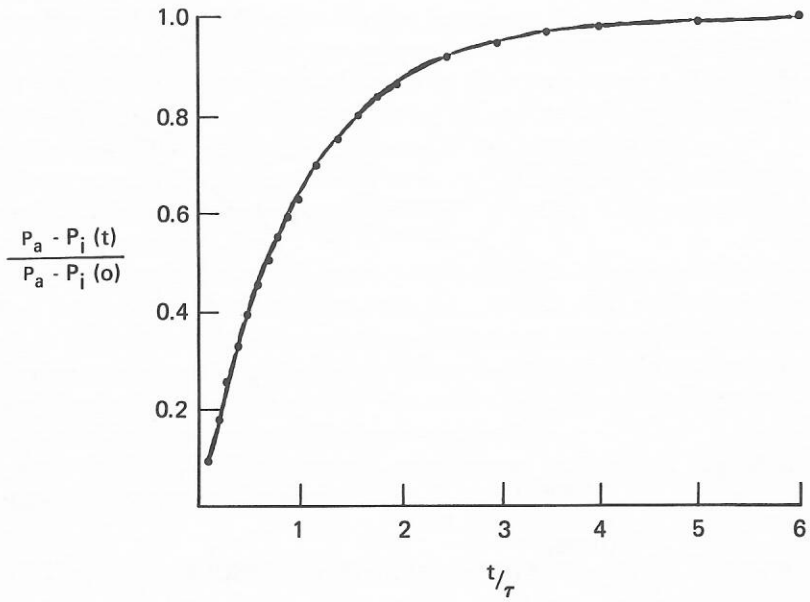


Figure VII-7. Generalized plot of inert gas absorption by a perfusion-limited tissue [see Eq. (14) in the text].

5. Overly obese and slender people exceed the normal range of cardiac output per kg BW and therefore probably have altered tissue perfusion rates (in addition, the obese subject has a higher fat content, which is reflected in an altered partition coefficient).
6. Rapid compression has been shown to drastically reduce local blood flow rates (bone and peripheral tissue), although this may not influence overall tissue perfusion rates.

Table VII-5
Tissue Perfusion Rates for a 70-kg Man (1.85 m^2) under Rest Conditions with an Expected Cardiac Output of 5.8 liters/min

Tissue	Blood flow rate, cm^3 of blood/cm^3 of tissue·min^{-1}	Tissue volume cm^3
Lungs	4.8	1,200
Kidney	4.9	270
Heart	0.5	300
Adrenals, testes, prostate	1.0	80
Brain	0.54	1,400
Marrow	0.21	1,400
Hepato-portal	2.1	3,145
Muscle connective tissue, skin	0.022–0.0089	45,000–55,000
Fat	0.013–0.039	15,000–5,000

While there is great temptation to consider the process of inert gas exchange to be influenced by only these factors, the situation is really far more complex. Ultimately, we evaluate the adequacy of decompression schedules in terms of the existence of symptoms which are really only partially related to blood flow. Because of this uncertainty in predicting the influence that a hyperbaric exposure will exert on blood flow, decompression procedures should be tested under conditions closely simulating the actual environment. Also, schedules should be used in only those circumstances where one is sure that hemodynamic factors will not lead to lower than normal tissue perfusion. (For example, it is hazardous for an obese person to dive with U. S. Navy procedures that were tested on slender men.)

d. Site of Bubble Nucleation and Growth

The other role of the cardiovascular system in decompression is as a possible site for bubble nucleation or growth. The vascular tree is a compliant space (more so than tissue), so a rapid volume expansion due to bubble nucleation would result in little mechanical resistance. In addition, turbulence at heart valves or around rigid arterial bifurcations could result in the formation of short-lived micronuclei. These would normally dissolve. If they were carried into tissues where gas depots could provide a supersaturated environment, they could grow to a size greater than the critical radius. With these nuclei lodged in venules, they would be in the most supersaturated tissue regions. Bubbles so positioned would not represent a serious resistance to venous flow. Thus, they could persist and grow to considerable size before being dislodged into the the central venous circulation. Harvey and his associates (Harvey 1951) demonstrated that such micronuclei do not persist in circulating venous blood. This does not preclude their existence in peripheral vascular beds or in tissue spaces.

The severe cases of decompression sickness involve circulating bubbles that somehow bypass the lungs. When this condition occurs, the normal blood flow is drastically altered. The ability to transport inert gas from peripheral tissues is greatly reduced because the induced state of circulatory shock will result in an almost complete cessation of blood flow in the peripheral tissues. Even after therapeutic recompression completely dissolves the bubbles, the damage to the circulatory system (release of clotting factors, lipoproteins, alteration in platelet function, edema, etc.) may require days to recover, during which time the blood distribution will be abnormal. Thus, divers are normally kept from diving for at least a week after experiencing DCS.

e. Diffusion and Convection in Tissues

The concept of perfusion-limited tissues has been reasonably well established for the central tissues of the body, such as the CNS, heart, liver, kidney, and muscles. But in the peripheral tissues the assumptions are less well founded. In these tissues the cells have evolved so as to endure a more variable blood flow and one that is regulated by other functionalities, such as heat balance, response to trauma, and response to external stimuli (thermal and mechanical). In these tissues, the capillary morphology is irregular and, often, large avascular regions exist (e.g., cartilage, synovial fluid). Lymph flow in these regions may be a dominant means of nutrient transport. The

distance between capillaries may be so great that diffusion will play a dominant role in the process of saturating the tissue with an inert gas.

Since oxygen must somehow reach these isolated cells, one can assume that inert gases do likewise (having similar solubilities and diffusivities). But if the process of diffusion dominates the delivery of inert gas to these regions, then there must be substantial gradients in partial pressure between these cells and the nearest vascular sections. During decompression these peripheral tissues would have inert gas partial pressures substantially greater than the average vascular levels, resulting in the peripheral tissues being a site of maximum supersaturation.

During a hyperbaric exposure, these tissues would all exhibit saturation times greater than those of the perfusion-limited tissues. The estimation of saturation times for these tissues is dependent on both the gas diffusivity (Table VII-4) in these tissues and the geometry of the tissue. Based on realistic anatomical considerations, saturation time constants of 300 min have been estimated, where

$$\tau = \delta^2/D \tag{16}$$

where δ is some characteristic diffusion distance (e.g., mean intercapillary separation).

It is important to note that shifting from one gas to another will change a diffusion-limited time constant in a different manner than it will for a perfusion-limited tissue. A change in breathing gases implies a change in the rate of tissue saturation in a perfusion-limited tissue based on the relative gas solubilities, while it depends on the relative gas diffusivities in a diffusion-limited tissue.

One must be careful not to assume that these long saturation times necessarily correlate with empirical whole-body saturation times for developing decompression schedules. The *slow* tissue defined by Eq. (14) may not be a suitable site for bubble nucleation, due to its low compliance to distention (e.g., a gelled liquid has a greater resistance to cavitation than does the ungelled liquid). These tissues do, however, represent a reservoir from which gas slowly emerges such that it could contribute to local vascular bubble growth long after decompression is complete. [Unfortunately, there are tissues that are neither perfusion-limited nor diffusion-limited, but are partially controlled by both processes. In these tissues a 30% change in blood flow might result in only a 10% change in the rate of inert gas absorption.

Whole-body washout studies. Measurement of gas exchange within the body yields evidence for the simultaneous existence of many rates of gas exchange. If a subject breathes a gas mixture devoid of N_2 and the excreted nitrogen is measured, a multi-exponential curve of N_2 excretion vs. time is obtained. Analysis of this curve yields evidence for at least four tissue domains characterized by a volume and a time constant. Other than providing direct experimental evidence for the spectrum of time constants required to adequately characterize man, these measurements are of little utility in defining the rate-controlling processes in inert gas exchange at tissue level, where bubbles presumably originate.

The fast time constants will correlate with the perfusion-limited time constants, but the slow time constants will not. Measurement of the time constant for a diffusion-limited tissue is complicated by the small rate of gas evolution from the tissue.

3. *Summary*

1. Gas solution and dissolution *in vivo* follow expected physicochemical laws.
2. Bubble nucleation and growth rates can be characterized by physical models; but the rates of these processes are unpredictable from first principles and depend strongly on the details of the local site for determining the controlling factors.
3. Inert gas absorption into body tissues requires anywhere from a few minutes to over 24 hr to reach a state of equilibrium with the gas environment.
4. While the laws of physics predict that bubbles will form at the site of maximum supersaturation, experimental conditions do not permit the assessment of whether this occurs *in vivo*.
5. The site of symptoms may not correlate with the site of bubble formation or the site of bubble growth, due to the involvement of the circulatory system.

C. *Diagnosis*

The basis for diagnosing decompression sickness is an interactive physical examination of the subject. The symptoms are qualitative impressions of discomfort and can often be confused with minor accidents that occur during the dive. While one could be tempted to wait for confirmation of the severity of these symptoms, it is far wiser to treat the first symptoms, if at all possible.

1. *Incidence of Decompression Sickness and Appearance of Symptoms*

Because of the limited testing of decompression schedules, there remains a finite incidence of the disease even when all instructions have been followed. There is no way to determine which exposures give the greatest risk. One always has to be aware of the possibility of suffering this disease.

The symptoms of decompression sickness are generally classed as mild (Type 1) or serious (Type II). They may occur any time during the decompression or soon thereafter. A review of U. S. Navy records of subsaturation showed that:

50% of the symptoms had occurred within 30 min of surfacing
85% of the symptoms had occurred within 1 hr of surfacing
95% of the symptoms had occurred within 3 hr of surfacing

Similarly, Figure VII-8 shows the time of onset of both Type I and Type II symptoms for 127 cases of decompression sickness recorded from a Canadian diving experience. Notice that 42 cases (33%) occurred prior to surfacing. Saturation diving results in a higher incidence (86%) of bends at depth. (This fact may only be due to the experimental nature of virtually all decompressions from saturation.)

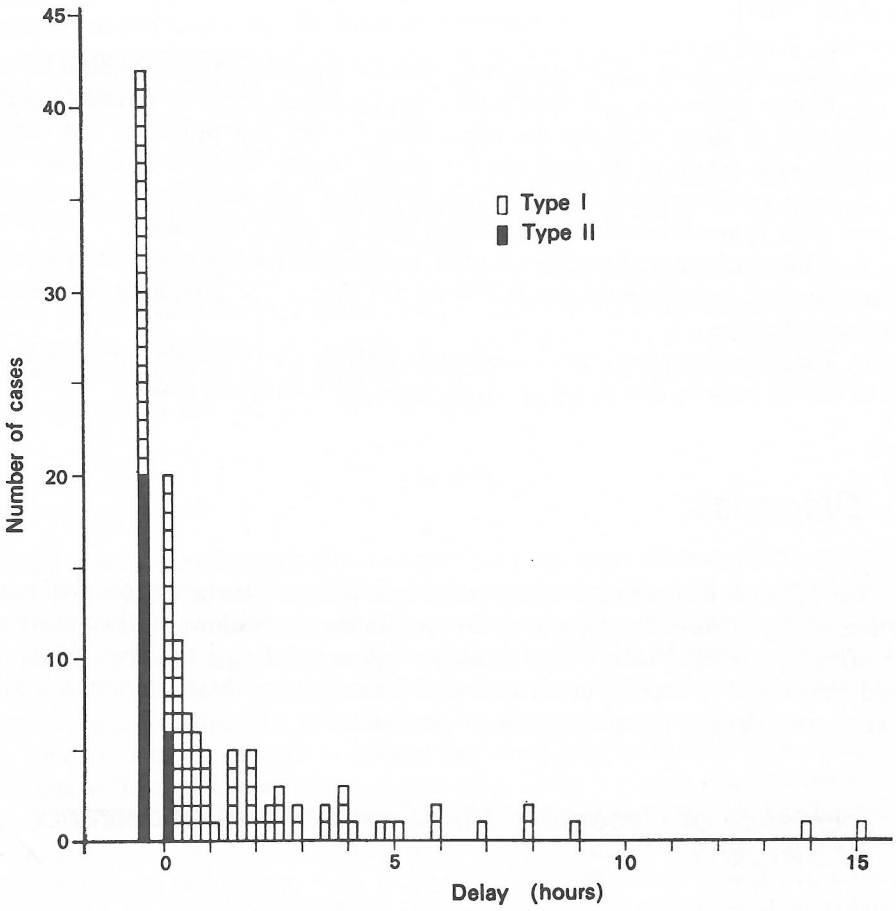


Figure VII-8. Decompression sickness—time of onset of cases in relation to the time of reaching surface. A total of 127 cases from the Canadian Forces Institute of Aviation Medicine and Royal Canadian Navy Diving Establishments, 1962–1967. The 42 cases that occurred during decompression are shown together before the time of surfacing. [From Kidd and Elliott (1969) by permission of Bailliere and Tindall, London.]

Military dives deeper than 10 ATA result in substantially more bends at depth (46%) than do dives at less than 10 ATA (10%). There are no comparable figures available for civilian diving. Tunnelers experience symptoms both at depth and after surfacing, with the mean time for symptoms being 2 hr.

2. *Major Symptoms*

The major symptoms of decompression sickness are (Type I) pain, dizziness, paralysis, shortness of breath, extreme fatigue, and collapse. These symptoms are best described for laymen (who must often make the first diagnosis) in the *U. S. Navy Diving Manual* (1970):

Occasionally the skin may show a blotchy and mottled rash. There may be small red spots that vary in size from a pinhead to the size of a dime. Sometimes mottling is so pronounced that the skin takes on an appearance like that of pink marble, and the term of "marbeling" is applied.

A typical case of decompression sickness may begin with itching or burning of a localized area of the body. This symptom may spread and then finally become localized again. There may be a feeling of tingling or numbness of the skin. In rare cases, the man may have a sensation of ants crawling over him.

Pain, which is the most frequent and predominating symptom is of a deep and boring character. Divers describe it as being felt in the bone or in the joint. Usually the pain is slight when first noticed and then becomes progressively worse until it is unbearable. The pain usually is not affected by movement of the area, but it may be temporarily relieved by vigorous rubbing or hot applications. The most frequently confused situation is that of a diver suffering a muscle strain or a joint sprain during a dive. However, this condition can usually be distinguished by the fact that strains and sprains are painful to touch and motion, while pain in a joint from decompression sickness is generally not. Swelling and discoloration usually occur with a sprain but are rare in uncomplicated cases of decompression sickness. A diver who has pain that might be a symptom of decompression sickness should have *treatment* by recompression, even though the pain may turn out to have been from a strain or sprain. WHEN IN DOUBT, TREAT BY RECOMPRESSION. Failure to treat doubtful cases is the most frequent cause of lasting injury.

Abdominal pain after a dive has frequently been followed by symptoms and signs of spinal cord involvement (i.e. weakness or paralysis of legs; burning, tingling, or numbness of legs or feet). It is important to recognize that the onset of abdominal pain in decompression sickness is a serious symptom requiring immediate recompression *treatment* as such. Careful examination of the diver upon his arrival at depth in the chamber should determine the extent of spinal cord involvement and the time necessary for complete resolution of the involvement in order to select the proper table for treatment.

When dizziness occurs, the diver feels that the world is revolving about him and that he is falling to one side. Frequently, he will have ringing in the ears at the same time that dizziness occurs. History and physical examination become important when these symptoms occur because they also can follow middle-ear damage, as from squeeze.

Serious symptoms are those caused by bubbles in the brain, spinal cord, or lungs. These symptoms require longer *treatment* than the pain-only type, and it is very important not to overlook them when they are present. Many of the serious symptoms are so well defined that the diver is certain to notice and report the symptoms, or the signs are so obvious that his tenders cannot miss them. However, it is quite possible to miss some of the less obvious signs and symptoms or to fail to recognize the milder disorders such as simple weakness, partial paralysis, or defective vision. Do not let a serious case be treated inadequately simply because no one bothered to check! For example, occasionally a diver who complains only of pain in an arm or a leg will also be found to have weakness or partial paralysis when he is examined thoroughly. It is also important to know *all* that is wrong with the patient so that you can be sure he is relieved of all his symptoms during treatment.

The *U. S. Navy Diving Manual* (1970) also provides guidance on performing the diagnosis:

The following are the most important things to check when examining a man prior to treatment or when trying to determine whether all symptoms have been relieved:

(a) *How does he feel:* (Ask him.)

1. Pain—where and how severe? Changed by motion? Sore to touch or pressure? Bruise marks in the area?
2. Mentally clear?
3. Weakness, numbness, or peculiar sensations anywhere?
4. Can he see and hear clearly?
5. Can he walk, talk, and use his hands normally?
6. Any dizziness?

(b) *Does he look and act normal:* (Don't merely take his word if he says that he is all right.)

1. Can he walk normally? Any limping or staggering?
2. Is his speech clear and sensible?
3. Is he clumsy or does he seem to be having difficulty with any act of movement?
4. Can he keep his balance when standing with his eyes closed?

(c) *Does he have normal strength:* (Check his strength against your own and compare his right side with his left.)

1. Normal handgrip?
2. Able to push and pull strongly with both arms and legs?
3. Able to do deep knee bends and other exercises?

(d) *Are his sensations normal:*

1. Can he hear clearly?
2. Can he see clearly both close (reading) and distant objects? Normal vision in all directions?
3. Can he feel pinpricks and light touches with a wisp of cotton all over his body? (Note that some areas are normally less sensitive than others—compare with yourself if in doubt.)

(e) *Look at his eyes:*

1. Are the pupils normal size and equal?
2. Do they close down when you shine a light in his eyes?
3. Can he follow an object around normally with his eyes?

(f) *Check his reflexes* if you know how.

Note that it should not take a great deal of time to examine a man reasonably well. Especially when you are under pressure in the chamber, there is seldom time to waste, but do not shortchange the patient. If there is real need for haste, having him walk and do a few exercises will usually show (or call to his attention) the more serious defects. In all cases where there is any doubt, treat the diver as though he is suffering from decompression sickness. If you are not sure that he is completely free from *serious* symptoms, use the longer table. Remember that time and air are much cheaper than joints and brain tissue.

Tables VII-6 and VII-6B present the diagnosis process in such a manner that one can differentiate among possible causative factors. (Gas embolism, pneumothorax, and mediastinal emphysema are discussed in Chapter III.)

These symptoms occur with the following frequency, as summarized by the U. S. Navy (1970): pain, 89% (leg, 30%/arm, 70%); dizziness, 5.3%; paralysis, 2.3%; and shortness of breath, 1.6%. The Canadians have found pain associated with 75% of their cases. Reports on tunnelers indicate a higher incidence of pain in the legs than in the arms.

The skin is often involved in Type I symptoms (20% of the cases reported). The visual symptoms are always preceded by intense itching, generally about the trunk of the body. Local vasodilation yields a red spot with a cyanotic, blue center, indicative of local stasis. These symptoms most often appear after long, shallow dives.

Table VII-6A
Diagnosis of Decompression Sickness[a]

Signs and symptoms	Bends		Serious decompression sickness		Gas embolism CNS symptoms		Other barotraumas	
	Skin bends	Pain only	CNS bends	Chokes	Brain damage	Spinal cord damage	Pneumo-thorax	Mediastinal emphysema
Pain: Head					××			
Back			×					
Neck								××
Chest			×	××		×	××	×
Stomach			××			×		
Arms/legs		××				×		
Shoulders		××				×		
Hips		××				×		
Unconsciousness			××	×	××	×	×	
Shock			××	×	××	×	×	
Vertigo			××		×			
Visual difficulty			××		××			
Nausea/vomiting			××		××			
Hearing difficulty			××		××			
Speech difficulty			××		××			
Balance lack			××		××			
Numbness	×		××		××	×		×
Weakness		×	××		××	×		
Strange sensations	×		××		××	×		
Swollen neck								××
Short of breath			×	×	×	×	×	×
Cyanosis				×	×	×	×	×
Skin changes	××							
Eye tracking			××		××			

[a] ×× Probable; × possible cause.

Table VII-6B
Diagnosis of Decompression Sickness: Confirming Information

Diving history	Yes	No
Decompression obligation?	___	___
Decompression adequate?	___	___
Blow-up?	___	___
Breath-hold?	___	___
Nonpressure cause?	___	___
Previous exposure?	___	___

Patient examination	Yes	No
Does diver feel well?	___	___
Does diver look and act normal?	___	___
Does diver have normal strength?	___	___
Are diver's sensations normal?	___	___
Are diver's eyes normal?	___	___
Are diver's reflexes normal?	___	___
Is diver's pulse rate normal?	___	___
Is diver's gait normal?	___	___
Is diver's hearing normal?	___	___
Is diver's coordination normal?	___	___
Is diver's balance normal?	___	___
Does the diver feel nauseous?	___	___

For tunnelers and divers decompressing in chambers, "skin bends" are often encountered. They are not useful as predictors of subsequent symptoms and are transient in nature. Treatment is seldom provided. Behnke (1970) reported that 25% of the Type II cases and 12.5% of the Type I cases of decompression sickness in the BART project exhibited skin symptoms. Lymphatic occlusion occurs in a small percentage of cases. It is manifested by local swelling and soreness, with the skin over the affected area having a *pigskin* appearance due to pitting edema.

The symptoms associated with Type II decompression sickness are more random in occurrence, probably because they reflect severe systemic involvement caused by bubbles that have been discharged into the central circulation. The bubbles (or their hematologic sequelae) may lodge in any of the central organs.

Table VII-7 reports the incidence of Type II symptoms reported for Canadian divers. Note that the severe pulmonary and CNS symptoms may be due to pulmonary barotrauma (see Chapter III) as well as decompression sickness. Diagnosis of the source of the symptoms should wait until treatment has been initiated. This differential diagnosis will be necessary in carrying through the treatment and planning for the use of auxiliary measures (e.g., with severe decompression sickness, one may wish to use plasma expanders and heparin, which are not indicated for acute treatment of pulmonary gas emboli).

3. *New Methods of Diagnosis*

In recent years there has been substantial research into the development of new methods of diagnosing decompression sickness. These methods center around direct detection of the bubbles or the biochemical concomitants to the bubbles. These

Table VII-7
The Distribution of Type II Symptoms[a]

Symptomatology	Number	Percentage
Dizziness	9	31
Confusion	2	
Disorientation	2	
Nystagmus	1	
Eye signs: diplopia	2	7
tunnel vision	1	
Sensory impairment: hyperaesthesine	4	25
paraesthesiae	7	
Motor weakness or loss	9	25
Dysarthria	2	
Nausea	2	4
Respiratory: dyspnoea	3	9
coughing	1	
Total	45	

[a] From Bennett and Elliott (1969) by permission of Bailliere and Tindall, London. Type II manifestations presenting symptoms may occur in more than one category.

methods have been developed to assist in the testing of experimental decompression schedules by providing a more quantitative end point to the disease. A general finding with these newer methods has been the realization (long suspected) that many processes occur well before the subject experiences the conventional symptoms. In many cases the symptoms never occur, even though the new method reveals a valid alteration in the measured parameter, indicating a disturbed homeostasis.

a. Physical Methods

Major advances in ultrasonic instrumentation have resulted in several *real-time* systems for bubble detection. Almost all the methods use frequencies greater than 1 MHz; at these frequencies they utilize the fact that ultrasound cannot penetrate air bubbles, but will penetrate tissues. The method involves purposely introducing ultrasonic energy and measuring the energy that either penetrates the tissue (*transmission* mode) or is reflected back off the bubbles (*reflected* mode). Some methods can detect stagnant bubbles lodged in peripheral tissues (the presumed source of bubbles) and some can only detect the circulating bubbles.

The ultrasonic energies utilized are presumed to be safe, based on information published concerning ultrasonic cavitation in supersaturated liquids. Figure VII-9 shows that the energy needed to induce cavitations rises sharply with frequency, so

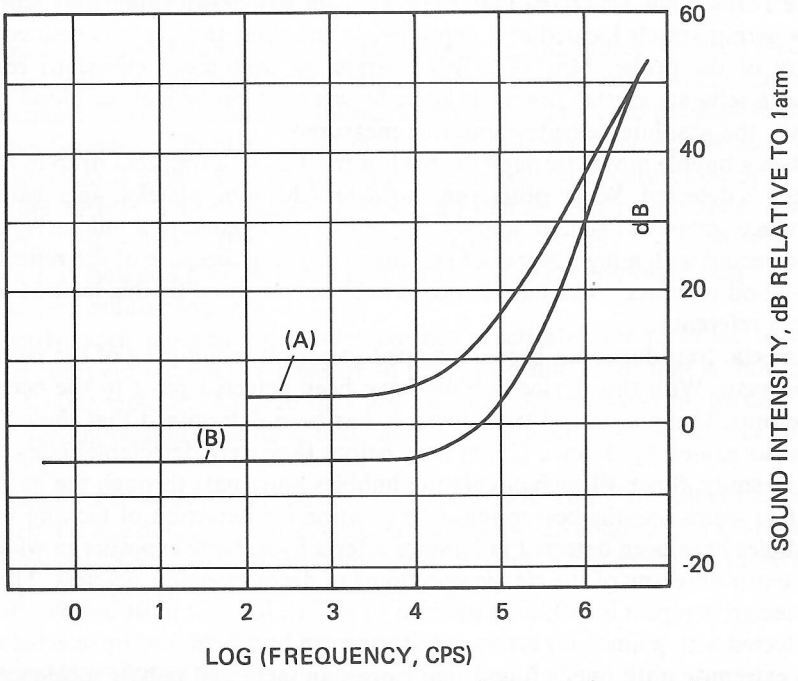


Figure VII-9. Cavitation threshold as a function of ultrasonic frequency. Frequency dependence of onset of cavitation in a liquid. (A) Distilled, filtered, and degassed water; (B) tap water containing 20% air. [From Galloway (1954) by permission of The American Institute of Physics.]

that the estimated sound pressure requirements at 1 MHz are over 1000 atm relating to 1 ATA. Although this power intensity is substantially higher than anyone has used to date, little work has been done to account for the possible focusing or resonance-producing possibilities that could occur in the inhomogeneous environments in the body (both conditions are known to occur *in vivo* and could result in local sound energies well in excess of the energy source). Until such time as the safety has been well demonstrated, these instruments should only be utilized by trained investigators.

The Doppler Ultrasonic Flowmeter (DUF) was first used to detect intravascular bubbles as an air emboli monitor in open-heart surgery. The device was subsequently used to detect bubbles in peripheral veins of divers after surfacing from dives in which no other symptoms were seen.

The DUF detects moving objects by the principle of detecting the Doppler shift of ultrasound that is produced. The Doppler shift is related to the velocity of reflecting particles v by

$$\Delta f = \frac{2fv \cos \theta}{c}$$

where f is the ultrasonic frequency, c is the speed of sound in the fluid, and θ is the angle between the incident ultrasonic beam and the vector of the velocity v.

This frequency shift is normally monitored audibly because it falls in the audible range for all but the highest of particle velocities.

The Transcutaneous DUF (TDUF) permits one to measure qualitative blood-flow changes within vessels located at a depth below the skin; the depth is defined by the geometry of the probe. Most TDUF's operate by continuous emission; reception occurs at a separate crystal. Since the angle between the probe and the blood vessel is unknown, the absolute velocity cannot be measured.

When a bubble moves through the field of the TDUF a transient drop in Doppler frequency is detected. While other vascular *debri* (thrombi, platelet, aggregates, etc.) can produce somewhat similar sounds, the bubbles do generate a unique signal that can be detected with a high degree of certainty (probably because of the reflection off the air–blood interface). The bubble size cannot be measured by this method without an *in vivo* reference.

A special transducer has been developed to permit monitoring of the pulmonary artery in man. With this device bubbles have been detected prior to the occurrence of symptoms. Using a second transducer, it has been determined that these "silent" bubbles [so-named by Behnke (1955) long before they were detectable] come from a single extremity. Since all such circulating bubbles must pass through the pulmonary artery, this seems like the best monitoring location for detection of moving bubbles.

Bubbles have been detected in humans after a hyperbaric exposure in which they did not experience any of the classic symptoms of decompression sickness. They have been detected in repeat breath-hold dives to 15 m. The locus of most bubbles that have been detected with pulmonary artery monitoring can be determined by selected motion of each extremity until one is found that causes an increased bubble incidence in the pulmonary artery. Confirmation is then made by use of a peripheral vein TDUF monitor placed over the appropriate vein.

Rubissow (Rubissow and MacKay 1971) has reported on a scanning ultrasonic

system that has a 2–5 μm detection limit for bubbles. He has shown that the amplitude of the imaged reflected bubble may be confused with reflection from a multiplicity of smaller bubbles. With the use of a step recompression he was able to measure the actual bubble size. This system could be developed for field use in much the same way as a portable X-ray machine.

Martin *et al.* (1973) have reported detecting bubbles in human trials of a novel transmission ultrasonic system. The unit utilizes the fact that ultrasonic reflection off a bubble is nonlinear and tends to generate higher harmonics of the incident ultrasound. They used a pair of 115-kHz transducers mounted on each side of the thigh so the beam transected the great saphenous and femoral veins, as well as the skin and musculature. By filtering the received signal at the second harmonic (230 kHz) they identified a characteristic *signature* of bubbles. Their studies indicate that the unit could be used in a hyperbaric environment. However, the subjects need to be motionless, and the identification of the bubble signature is subject to interpretation. Often, the subjects experienced symptoms before any bubbles were detected, unlike the results of the DUF studies.

Acoustic-optical imaging is able to detect stagnant bubbles (Buckles and Knox 1969). This method produces a direct optical image of the acoustic energy either reflected from or transmitted through the tissue. It is at a very early stage of development, and would generally only be available to large research or treatment centers.

b. Biochemical Methods

Measurements of hematological changes during decompression are based on one of two concepts: (a) that bubbles will initiate measurable changes; or (b) that the stress of inadequate decompression will induce alterations that would predispose the blood to bubble formation. Bubbles could induce hematological changes because the gas–blood interface is highly active and could lead to protein denaturation or alterations in clotting factors. Extravascular bubbles can disrupt cell membranes, releasing a wide variety of enzymes that are indicative of tissue trauma. Alternatively, bubbles growing in vessels could block blood flow and result in the release of enzymes or other indicators of cellular hypoxia.

Clearly, such biochemical studies are not practicable for real-time monitoring. However, they do represent a measure of decompression *insult* that is more sensitive than clinical symptoms. As such they are useful as determinants in the development of new decompression schedules that should have much lower incidence of clinical symptoms.

Studies have shown marked elevations of CPK and haptoglobins, which are measures of tissue damage, in divers exposed to saturation conditions and subsequently decompressed. No reports of decompression sickness were made by these subjects. Others have shown that there is an increase in circulating lipids.

D. *Prevention*

Decompression sickness is prevented by proper preparation for a dive and use of decompression procedures. Some divers still suffer decompression sickness even though

they adhere to schedules. This is because the tables were developed to permit a finite incidence of decompression sickness (if made 100% safe, they would be prohibitively long). While this is acceptable to divers who have good treatment facilities nearby, this risk may be excessive for others who are less well equipped. For example, a successful decompression schedule for compressed air workers in the U. K. is one that reduces the incidence of decompression sickness to below 2% for exposures over 4 hr between depths of 40 and 90 ft (2.2 and 3.7 ATA).

In this section the development and use of decompression procedures will be presented. When the requirements of a dive exceed the range of available tables, the change in procedures will be indicated, where known. The decompression schedules discussed here are presented together at the end of this chapter.

1. *Preparation*

Diver selection (see Chapter XI) is necessary so as to prohibit from diving those participants who will probably have difficulty in decompressing or be especially prone to decompression sickness. The decompression schedules have generally been developed with experienced, conditioned subjects who range from 19 to 45 yr in age and meet military standards of physical fitness. Increasing age and body weight increases one's susceptibility to decompression sickness. Subjects who have suffered diseases that impaired their circulatory or respiratory system are also high-risk subjects.

Tunneling schedules are somewhat different in order to take into account the generally greater variance in subject physical characteristics. Behnke (1970) has reported that adequate selection criteria are necessary to maintain an acceptably low incidence of decompression sickness and avoid aseptic bone necrosis. The selection physical examination must be given by a physician trained in this field of environmental medicine; the examination should include: age, weight (correlated against general body structure), confirmation of auditory tube patency and activity, pulmonary pathology, radiographic survey of long bones, equilibrium disturbances, and response to a suitable cardiovascular stress test (e.g., Step-Test Exercise). See Chapter XI for further details. Schedules have been validated only on men, even though many women have been safely decompressed using the U. S. Navy air decompression tables.

Comparison of *safe* decompression schedules developed in different countries suggests that there may be national and racial differences in susceptibility to DCS. This factor has never been tested. The observed differences could also reflect variations in aquanauts' or tunnelers' response to pain or the diving officers' selection of subjects (e.g., in order to validate a schedule some investigators will use a subject who is known to be highly susceptible to DCS while others would reject such a person from the protocol).

Most subjects in preparation for testing decompression schedules are *worked-up*; that is, they make several exposures to safe pressures near the one to be tested, within a period of days before the test dive. Worked-up divers are more resistant to DCS; in fact, in tunneling work it is well known that most of the occurrences of decompression sickness are limited to men who are returning to work after a layoff.

The majority of accidents occur to the relatively inexperienced subjects. Proper equipment selection and maintenance are also critical to the prevention of decompression sickness. Equipment failures at depth (especially breathing equipment) are a major cause of accelerated decompression. Each set of diving equipment is designed for use in a certain environment in order to maximize safety. For example, a standard scuba tank, used at depths shallower than 60 ft (the recommended limit) will safely limit one's bottom time so as to never require decompression stops.

In summary, careful preparation involves several general rules:

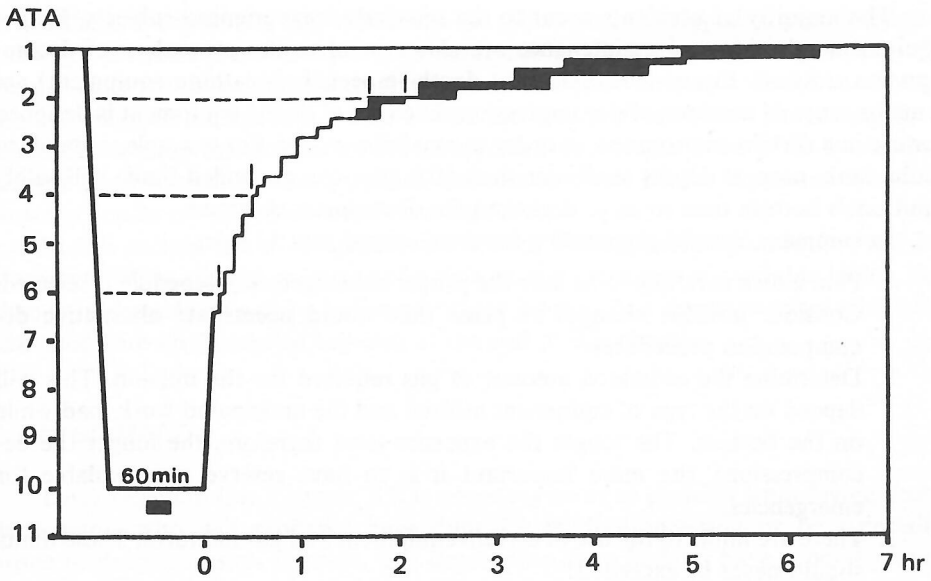
1. Plan a dive carefully to be sure the proper decompression schedule is selected. Consider possible changes in plans that would necessitate alternative decompression procedures.
2. Determine the estimated amount of gas required for the mission. This will depend on the type of equipment utilized and the anticipated work load while on the bottom. The longer the exposure (and therefore, the longer the decompression), the more important it is to have reserve gas available for emergencies.
3. There are limits to the use of certain equipment and gas mixtures. These limits should never be exceeded!
4. ANY TIME ANYONE SKIPS A SCHEDULED DECOMPRESSION STOP, PRESUME HE WILL SUFFER DECOMPRESSION SICKNESS AND SEEK TREATMENT IMMEDIATELY.

2. *Operational Factors that Influence Decompression Procedures*

During a hyperbaric exposure, one exercises at a certain work level. Then, during decompression, one normally minimizes exercise. So decompression procedures are normally developed with subjects performing a certain level of work consistent with the anticipated use of the schedules. Schibli and Bühlmann (1972) have shown that the amount of decompression one needs is directly related to the level of work performed at depth. This undoubtedly relates to the increased inert gas absorption that would occur during exercise. Figure VII-10 shows this increased decompression time for a subsaturation He–O_2 dive profile. This increased decompression need will be greatest for short dives, where the perfusion-limited tissues dominate and are not saturated. No simple rule is available to adjust decompression schedules for extreme exercise, mainly because of the empirical nature of the models. This variability is nicely avoided in saturation diving.

On the other hand, moderate exercise during decompression, especially to counter the influence of a cold water environment, should enhance inert gas washout and lower the risk of decompression sickness. There is, however, no conclusive proof that this is true. Exercise should be avoided after surfacing from a satisfactory decompression.

Oxygen breathing is often used during decompression to accelerate inert gas washout. While this does increase the inert gas partial pressure gradient from tissues to gas, it also tends to reduce blood flow rate. Decompressions using oxygen are generally faster than air decompressions; however, there are no general rules on how



ATA	% O_2	He	N_2
10,0-6,0	10	82	8
6,0-4,0	20	-	80
4,0-2,0	50	-	50
2,0-1,0	100	-	-

Figure VII-10. The influence of exercise on safe decompression time following subsaturation diving. Decompression schedules from a 60-min, 10-ATA (297-fsw) dive on an 82–8–10 mixture of He–N_2–O_2. The divers performed 15 min of exercise at an 80-W rate; there were 15 dives without work and 39 dives with work included. The darkened area indicates the extension in decompression time required to compensate for the exercise. [From Schibli and Bühlmann (1972) by permission of *Helvetica Medica Acta*, Schwabe and Co., Basel.]

to alter a standard air decompression schedule for oxygen breathing. In addition, there is the risk of oxygen toxicity (see Chapter IV). Studies on O_2 breathing during decompression have utilized resting divers and are therefore not applicable to scuba diving. In tunneling, O_2 breathing has been used, but one has to educate the workers as to its explosive hazards.

Similarly, there are many vasoactive drugs that one might consider using to enhance gas washout during decompression. But the influence of drugs on decompression schedules has not been evaluated. (The use of drugs in the treatment of decompression sickness is discussed later in this chapter under treatment.)

3. *Decompression Schedules*

Decompression schedules are tables and instructions that indicate how to safely decompress a diver from a hyperbaric exposure utilizing specific gas mixtures and life support equipment. The schedules are generally presented as a function of three variables: (1) depth of the dive, (2) time spent at depth (bottom time), and (3) available breathing gas.

These schedules are developed for use with specific equipment and generally have been developed for specific commercial or military needs. They carry medical–legal validity only within the context for which they are developed; most carry disclaimers of liability from use of the tables by anyone other than the designated user.

The schedules are generally developed in response to a specific need, such as when new equipment becomes available or an operational task must be carried out for which existing schedules are inadequate. Guided by the principles put forth earlier, it is possible to develop a model based on existing tables for calculating new schedules.* Most new tables are so complex that computers are utilized.

A matrix of test dives is calculated with the model that spans the desired depth and bottom time required. Then the new schedules must be validated experimentally using human volunteers and (generally) a test chamber capable of simulating the operational environment. If the schedules are found to be unsafe, they are modified empirically so as to achieve a certain number of symptom-free dives. Once such a matrix has been validated, the model is adjusted to reflect the necessary empirical modifications that occurred during testing. Then the modified model is used to calculate other schedules within the matrix limits of depth and bottom time.

The diving schedules are finally tested in the operational environment. This often requires further refinement of the schedules and the model.

a. Models Utilized to Compute Decompression Schedules

Models used to develop and correlate schedules must be viewed as empirical tools for curve-fitting. Ever since Haldane suggested that the parameters of the model had physiological and anatomic relevance, there has been an ongoing attempt to validate that concept. While such a goal is laudable, it is hardly necessary for the model to be useful for the purpose described above. As will be seen below, any model that is useful for schedule correlation is so complex as to make it exceedingly difficult to ever prove that it is an accurate reflection of actual *in vivo* processes. The literature thoroughly documents the failures of models that are used to extrapolate to new environments.

All models must have certain general characteristics. First, the process of inert gas absorption into body tissues during the hyperbaric exposure must be characterized, both for the projected dive profile and for the to-be-determined decompression exposure. During decompression, the tissue partial pressures must be compared to the environmental pressure in order to assess the state of supersaturation. Then, the process of bubble formation/growth must be described as a function of this supersaturation and some criteria of acceptability utilized. That is, one must decide what degree of supersaturation or bubble size should be viewed as hazardous. The model permits computation of a decompression depth–time profile that never exceeds this limit, yet minimizes the decompression time. Since most of the diving in this century has utilized the stage method of decompression (i.e., decrease the pressure rapidly to the next stage and hold at that pressure until the next step), the empirical values for permissible supersaturation include the safety inherent in this procedure. The results are *not* directly applicable to continuous decompressions, as discussed below.

* The general goal is to develop one model capable of handling all diving environments; less ambitious models can be more empirical, resulting in great mathematical simplifications.

During staged decompression, one remains at the depth until the tissue tensions reduce to a level such that the next step in pressure may be accomplished. That is, before the pressure is reduced, the tissue tension must be at the level acceptable after the pressure reduction; this implies that the tissue tension during most of the holding period is substantially less than the tolerable limit.

Models that have been most successfully utilized in the development of decompression schedules are introduced in the following subsections.

(i) Inert Gas Transport. Because the decompression schedule is dependent on so many factors, the model must be written in order to account for the important practical factors* encountered in the diving environment.

All models must be capable of representing inert gas exchange throughout the time domain represented by the dive. Earlier in the chapter the dynamics of inert gas exchange were shown to fall into three general time domains: (a) *early stages*, dominated by perfusion-limited tissues; (b) *late stages*, where peripheral tissues with long time constants ($\sim$300 min) contribute most of the expired gas; and (c) *intermediate stages*, where all tissues are substantial contributors to the expired inert gas. A single model that covers all three zones is the most general, but also the most complex to manipulate.

A generalized way to handle this mass transfer process is shown in Figure VII-11. Blood flow through the lungs results in equilibration of arterial blood with inspired gas (i.e., $P_{A_x} = P_x = F_x P_{abs}$). This blood is carried to a variety of capillary beds, where exchange with tissues occurs. Tissues 1 through n are perfusion-limited. Tissue r is perfusion-limited; it is broken into several zones, chosen such that r is a perfusion-limited tissue and r', r'', etc., are diffusion-limited tissues.

It is well established that one must compute the inert gas partial pressure in body tissues. However, British investigators and others have adhered to the older Haldane simplification of equating the inert gas absorption in a tissue to the total ambient pressure. Thus, for a diver on air at 33 ft of sea water (2 ATA) Haldane said a tissue could absorb 33 ft of excess nitrogen, when in fact it can only absorb 25.4 ft of excess nitrogen ($0.8 \times 33 = F_{N_2} \times P_{abs}$).

The Haldanian approach is adequate for dives where a single gas mixture is utilized (e.g., air). The resulting conclusions about permissible supersaturation need only be systematically modified to use either description. But for diving with inert gas mixtures that change during the dive, it is preferable to utilize real inert gas partial pressures. Gas partial pressures will be utilized throughout this chapter.

Most empirical models are simplifications of the general model in Figure VII-11. The Workman model (Workman 1965) considers only perfusion-limited tissues; the spectrum of time constants is much greater than for the previous Haldane model, upon which it is based. This is necessary because the Haldane model was based on short dives of shallow depths; Workman's model had to encompass experience with much longer dives, including saturation exposures.

* A more restricted use of modeling has been proposed by Barnard (1967) to assist in the interpolation between decompression profiles from a series of experimentally successful dives where all variables were constant except for depth (saturation dives). The technique is perfectly valid and will be discussed further.

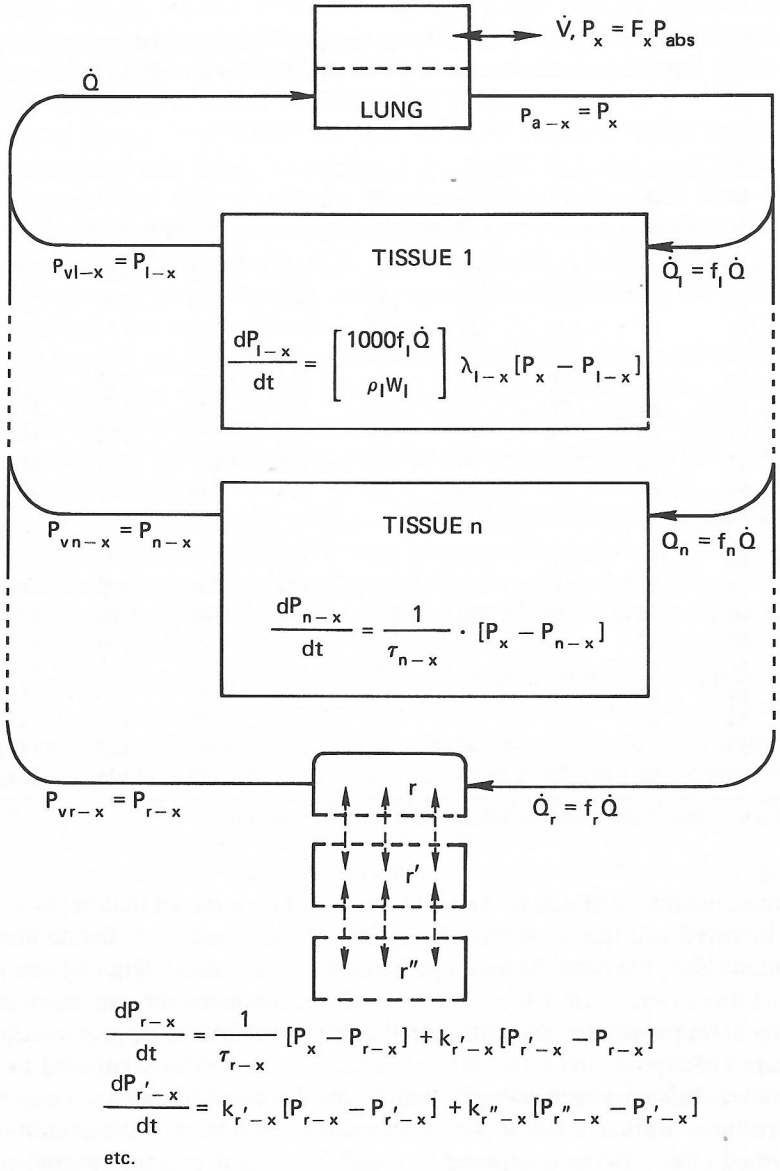


Figure VII-11. Generalized model for inert gas exchange between tissues and respired gases.

Table VII-8 lists the half-times (t_i/1.44) used by Workman (1965) to characterize inert gas transport. Values are given for both N_2 and He, the two common inert gases used in modern diving. Each tissue was considered to exchange gas with arterial blood independent of other tissues; during the dive, the arterial inert gas partial pressure is specified as a function of time and each tissue achieves a certain gas loading based on this imposed forcing function $P_x(t)$.

Table VII-8

Table of Maximum Allowable Tissue Tensions (M Values) of Nitrogen and Helium for Tissues of Various Half-Times (Workman 1965) [a]

$t_{1/2}$ min	Depth of decompression stop, ft									
	10	20	30	40	50	60	70	80	90	100
Nitrogen										
5	104	122	140	158	176	194	212	230	248	266
10	88	104	120	136	152	168	184	200	216	232
20	72	87	102	117	132	147	162	177	192	207
40	56	70	84	98	112	126	140	154	168	182
80	54	67	80	93	106	119	132	145	158	171
120	52	64	76	88	100	112	124	136	148	160
160	51	63	74	86	97	109	120	132	143	155
200	51	62	73	84	95	106	117	128	139	150
240	50	61	72	83	94	105	116	127	138	149
Helium										
5	86	101	116	131	146	161	176	191	206	221
10	74	88	102	116	130	144	158	172	186	200
20	66	79	92	105	118	131	144	157	170	183
40	60	72	84	96	108	120	132	144	156	168
80	56	68	80	92	104	116	128	140	152	164
120	54	66	78	90	102	114	126	138	150	162
160	54	65	76	87	98	109	120	131	142	153
200	53	63	73	83	93	103	113	123	133	143
240	53	63	73	83	93	103	113	123	133	143

[a] M values are in feet of sea water equivalent. $t_{1/2}$ is the tissue half-time.

Decompression schedules in the U. K. are based on a model that implies only one tissue is involved and that inert gas absorption in that tissue is diffusion-controlled. Hempleman (1967) has been the major proponent for this model. Since he was working with short dives (less than 100 min) he could approximate the gas absorption dynamics by a simple square root of time factor (a well-known approximation of an infinite series of exponential terms taken at times that are short compared to the saturation time). This approach does not require the direct calculation of tissue inert gas partial pressures. Rather, a factor $\phi(t)$ is computed that relates to the quantity of inert gas absorbed after a tissue is exposed to a unit increase in external partial pressure.

$$\phi(t) = 1 - \frac{8}{\pi^2}\left(e - Kt + \frac{1}{9}e^{-9Kt} + \frac{1}{25}e^{-25Kt} + \cdots\right) \tag{17}$$

The empirical value of K (based on evaluating experimental data) proposed by Hempleman (1969) is

$$K = 0.007928$$

when t is expressed as minutes. The total gas absorbed during a dive is computed by dividing the dive into a series of step changes in depth and using the superposition

principle to sum the results. When decompressing, the value K is increased by 50% because Hempleman suggests that inert gas transport is impeded during decompression. Decompression tables for both caisson and tunnel workers and deep sea divers have been developed with this model and are in use.*

Exercise during a dive has a profound influence on gas exchange and presumably on decompression schedules. Bühlmann (1969) found that exercising divers required more decompression time than when they rested during the same dive. His model (based on a modified Haldane concept) was modified by increasing the half-time of the fast tissues during the exercise period. Workman's (1965) model was based on dives in which hard work was performed; so the empirical constants that are used implicitly contain the influence of exercise.

When oxygen breathing is used, it is customary to make the following assumptions: (a) Neglect the reduction in blood flow to most tissues (i.e., no change in half-time spectrum of tissues) and (b) include a 20% leak of inert gas around the mask (i.e., the fraction of inert gas is 0.20, not zero as would occur if perfect seals could be made).

There is no fully satisfactory way to account for breathing inert gas mixtures (so-called tri-mix gas) or for switching between inert gases during the dive. One may choose to account for them separately, but ultimately they must be combined so as to estimate the total inert gas loading of each tissue. There are no decompression procedures that permit one to randomly change inert gas during the dive.

In recent years *saturation diving* has gained great favor because it prolongs bottom time without increasing the decompression requirements. The concept is based on the assumption that if a diver stays at a certain depth he will ultimately become saturated with the inert gas he is breathing. From then on he does not incur any more decompression obligation. Clearly, the diver must be provided extensive life support means to perform a saturation dive (food, shelter, heat, etc.).

The time required to reach saturation can be inferred from the inert gas models described above. Workman utilizes a slow tissue with a half-time of 240 min. This tissue will be saturated (to within 1%) after six half-times or 24 hr. Recent data on air saturation diving indicate that there may be slow tissues with 500-min half-times. These tissues would require 48 hr to saturate.

(ii) Safety Criteria. Haldane proposed that the body could tolerate dissolved inert gas so long as the decompression is carried out by reducing the pressure in successive steps where the ratio of computed tissue inert gas tension to final pressure never exceeds a value of 2.0. This conclusion was based on subsaturation studies where the final pressure was always 1 ATA; Haldane (Boycott *et al.* 1908) said this ratio should not be used at depths in excess of 165 ft.

Hawkins *et al.* (1935) were the first to point out that the Haldane ratio was too conservative for the fast-half-time tissues and not conservative enough for slow tissues.

* Hempleman's model is presented as an example of a simplified model that has little potential for extrapolation to diving environments outside the limits within which it has been tested (e.g., greater depths or mixed inert gases). The fact that the model has empirical utility is testimony to the hazards of applying the criterion of rigorous anatomical relevance to a model such as Workman's.

These concepts were utilized in developing the revised U. S. Navy Standard Air Decompression Tables (U. S. Navy 1943).

Studies on deep diving carried out during the late 1940's clearly indicated that the Haldane ratio must be reduced at elevated pressures. This fact is consistent with the notion that the ratio is a measure of the tendency for bubbles to form under the influence of supersaturation (see this chapter, physical factors). It is also consistent with the alternative theory that bubble growth is controlled by minimizing the degree of supersaturation. That is, the concept of a permissible degree of supersaturation is perfectly sound even though we have not proven whether bubble nucleation or growth is the process that controls decompression.

It has been determined empirically that the Haldane ratio decreases as absolute pressure increases in a parabolic fashion. Figure VII-12 shows this dependence as it has been determined by Bühlmann (1969). Two curves are presented; the shorter half-time tissues can utilize a higher supersaturation ratio at all depths.

Schedules were computed using the data from Figure VII-12 such that the permissible ratio is determined at each stop depth according to the computed values of tissue inert gas tension.

The U. S. Navy air decompression schedules incorporate this depth dependence of supersaturation. The schedules were the first ever computed on a digital computer. They utilize a tissue half-time spectrum of 5, 10, 20, 40, 80, and 120 min. The permissible supersaturation was selected for each tissue as a unique function of the tissue's nitrogen tension at the end of the dive. This permissible supersaturation value was used throughout all subsequent stops up to 10 fsw. Surfacing from this stop was controlled by the surfacing ratios originally proposed by Van der Aue *et al.* (1951). Figure VII-13 depicts both the surfacing values and the ratios used at depth for the U. S. Navy air schedules. These same values have been used to compute the U. S. Navy repetitive dive schedules.

Early studies with He as a breathing gas indicated that the helium Haldane ratio could be increased over the value used for N_2, permitting shorter decompressions from

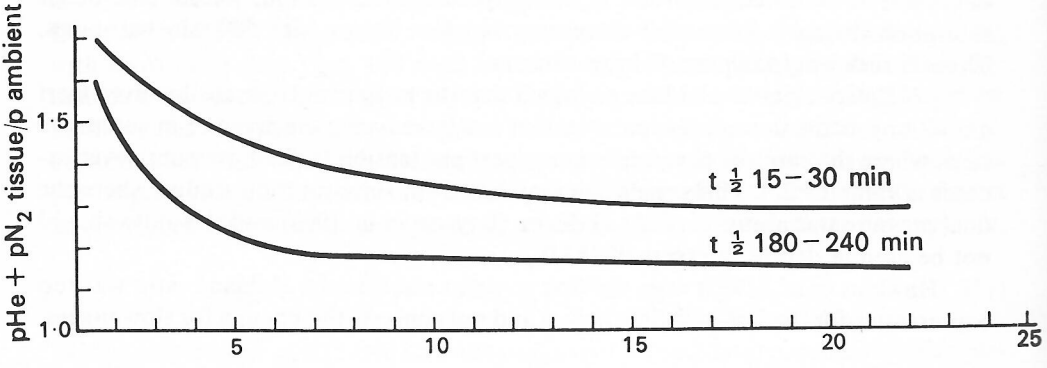


Figure VII-12. The Haldane ratio as a function of depth (N_2–He–O_2 mixtures). The permissible supersaturation of N_2 and/or He for the half-time spectrum used by Bühlmann in the calculation of human decompression schedules. The tables used stage decompression (see Figure VII-10) and can be used for any gas mixture at sea level or altitude. [From Schibli and Bühlmann (1972) by permission of *Helvetica Medica Acta*, Schwabe and Co., Basel.]

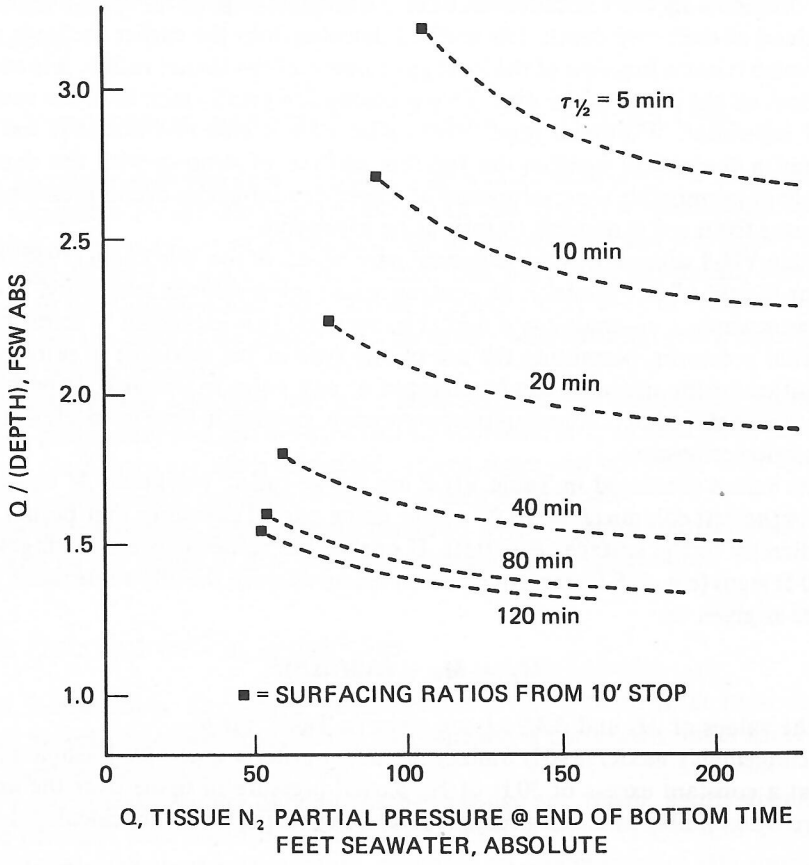


Figure VII-13. Permissible supersaturation ratios utilized in the U. S. Navy's air decompression schedules (des Granges 1956).

equivalent dives. Ever since, investigators have continuously been modifying the values as helium dives have progressed to deeper depths for longer exposure times. The data of Bühlmann are probably the most comprehensive available because of his extensive experience with very deep dives. Figure VII-12 shows how the Haldane ratio decreases at great depth. Bühlmann (1969) used tissue half-times that cover the range of 15–240 min, in essential agreement with Workman (1965).

Workman suggested that the Haldane tables were also compatible with the concept that there is a permissible partial pressure difference which governs decompression rates. He proposed that each tissue can sustain a certain amount of inert gas partial pressure and that this quantity M increased linearly with depth; at all depths, $M > P$. A review of published data for both air and $He–O_2$ dives yielded initial estimates of these M values for each of the presumed tissue constants.*

* Both Rashbass (1955) and Duffner *et al.* (1959) had used this "pressure head" concept for $He–O_2$ dives where the difference was independent of depth and only one tissue was considered. Their experimental data were subsequently used by Workman for establishment of the M values.

Workman's method requires that the permissible supersaturation difference be determined at each stop depth. His method deviates from the earlier methods in that the M value is not a function of the inert gas content of the tissue; rather, it is uniquely dependent on the depth of the dive. This presumption greatly simplifies the computation of schedules. Workman (and others who now utilize this method) has never presented a theoretical justification for this method of dealing with the depth dependence of permissible supersaturation of staged or continuous decompressions ranging in time from a few minutes to saturation exposures.

Table VII-8 summarizes the empirical parameters of the Workman (1965) model. A major benefit of this model is its generality and mathematical simplicity. It can be easily programmed on analog and digital computers. It is expressed in terms of inert gas partial pressures, permitting the use of any type of gas mixture; in principle, the composition of the mixtures can be changed at any point in the dive. It permits the calculation of staged or continuous decompression, ranging in time from a few minutes to saturation exposures.

The values presented in Table VII-8 are the so-called Workman M values. The values in the first column ($P = 10$ ft) are the tissue partial pressures that permit one to come directly to the surface ($P = 0$ ft). If one wishes to decompress in stages other than 10-ft steps (e.g., 0.5-ft steps, or *quasicontinuous* ascent), the allowable tissue partial pressure is given by

$$M_P = M_0 + (\Delta M/\Delta P)P \tag{18}$$

when the values of M_0 and $\Delta M/\Delta P$ are given in Table VII-8.

Hempleman's model (1967) utilizes the safety criteria urged by Rashbass (1955), i.e., that a constant excess of 30 ft of N_2 partial pressure in tissue over the ambient pressure is acceptably safe. The present Royal Navy diving tables are calculated on this basis.

The use of this constant allowable supersaturation is too conservative for long dives of medium depth, especially as it applies to compressed air workers. Hempleman (1969) has adopted a modified Haldane ratio that varies with depth and is given by

$$r = 400/P_{\text{abs}} + 180 \tag{19}$$

where P_{abs} is the absolute pressure in pounds per square inch. Tables based on this ratio and the diffusion-controlled inert gas transport (equal uptake and washout rates) have yielded a 1% incidence of decompression sickness when used between 79 and 95 ft (over 40,000 exposures) by U. K. compressed air workers.

Clearly the development of safety criteria for use in the calculation of decompression tables involves medical, ethical, and legal considerations. The limited experimental data, combined with the wide range of operational environments encountered in undersea activities, make scientific judgment only a partial contribution to the selection of such criteria. Extrapolation of any criterion beyond the bounds within which it has been tested is exceedingly hazardous. The models described in this section are presented because they are the basis for most of the decompression procedures used in the noncommunist world.

The following section is a guide to decompression procedures that can be used

for a wide variety of undersea exposures. The actual tables are given at the end of this chapter.

b. Decompression Schedules Available for Human Use

A decompression schedule consists of: (a) a table of depth–time relations; (b) a set of instructions for the selection and use of (a); and (c) a set of limitations that define the diving environment in which the schedule has been tested and is intended for use. In this section the schedules will be detailed that have been developed, tested, and reported in the published literature. Where schedules have been developed in several countries for the same environment (e.g., air diving with hard-hat gear), the American schedules will be reported.

Decompression schedules that have been developed by commerical diving firms are often proprietary and not available for public disclosure. In emergency situations, however, these firms are often contacted for assistance and will freely offer advice on suitable decompression procedures based on their experience.

The decompression schedules are classified by the type of inert gas (air, mixed gases) and by the length of dive exposure (subsaturation, saturation).

c. Air Decompression Schedules

(i) Subsaturation Exposures. Subsaturation diving with air is the most common form of hyperbaric exposure. Literally all tunneling is performed under these conditions; most scuba and hard-hat diving is done with air. The preponderance of nonmilitary diving tasks require only air (harbor salvage and repair, sports diving). This type of diving is subdivided into three environmental zones (see Figure VII-14): (I) *shallow* diving with short bottom times, requiring no decompression, (II) *normal* dives requiring decompression, and (III) *exceptional* or *emergency* dives requiring the use of relatively untested decompression schedules. These subsaturation air schedules are the most complex type of available tables because the diver must decompress in the water where many other risk factors are encountered. Consequently, there is great motivation to minimize the decompression schedule. In addition, there are a variety of emergency procedures available that permit the dive master to cope with special problems that may occur during decompression.

Many subsaturation air diving operations call for repetitive dives within a 12-hr period in order to optimize the use of personnel. There is always the risk that residual excess N_2 will persist in the diver from the first dive, enhancing his risk of decompression sickness following the second dive. Procedures have been developed to account for this excess risk, based on a knowledge of the first dive (depth and bottom time) and the time between dives (surface interval).

The U. S. Navy decompression schedules are the procedures of choice for all such air diving (Schedules 1.1–1.7 at the end of this chapter). The no-decompression limits are defined in Schedules 1.1 and 1.2. Repetitive dives require the assignment of a group designator that reduces the available bottom time in the second dive; this group designator is obtained by the use of Schedules 1.2 and 1.3.

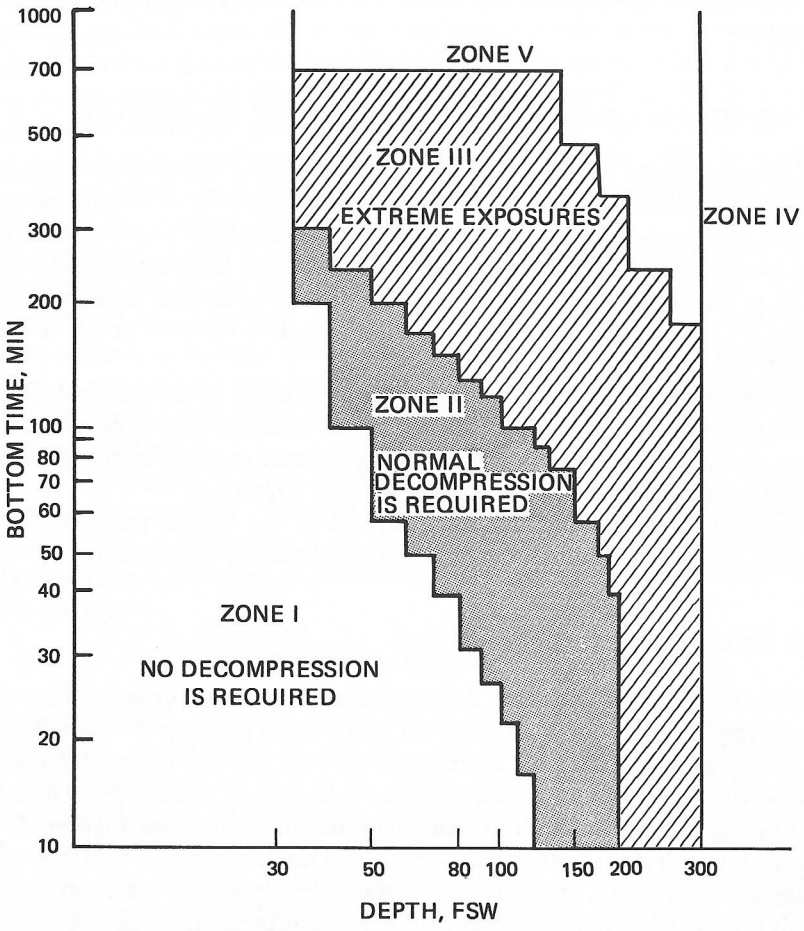


Figure VII-14. This figure permits one to determine the decompression risk associated with a dive that consists of a selected depth and bottom time. Such a dive may require: no decompression (I), use of normal decompression (II), or use of extreme exposure decompressions (III); dives in zone IV ARE NOT RECOMMENDED. Decompressions from zone V are covered under saturation decompression (Figure VII-15) (U. S. Navy 1971).

If one elects to minimize in-the-water decompression, one can have the diver breathe oxygen, directly surface, and transfer to a decompression chamber where he is recompressed to a suitable depth and subsequently decompressed. Schedule 1.6 is used for this procedure; if either oxygen breathing is not available or the diver suffers O_2 toxicity, use Schedule 1.5. These schedules have, for the most part, never been actually tested. They were prepared using the same parameters that were used to compute the normal tables.

Figure VII-15 outlines these air decompression tables and the sequence in which they are utilized for repetitive dives. Table VII-9 lists the general rules governing the

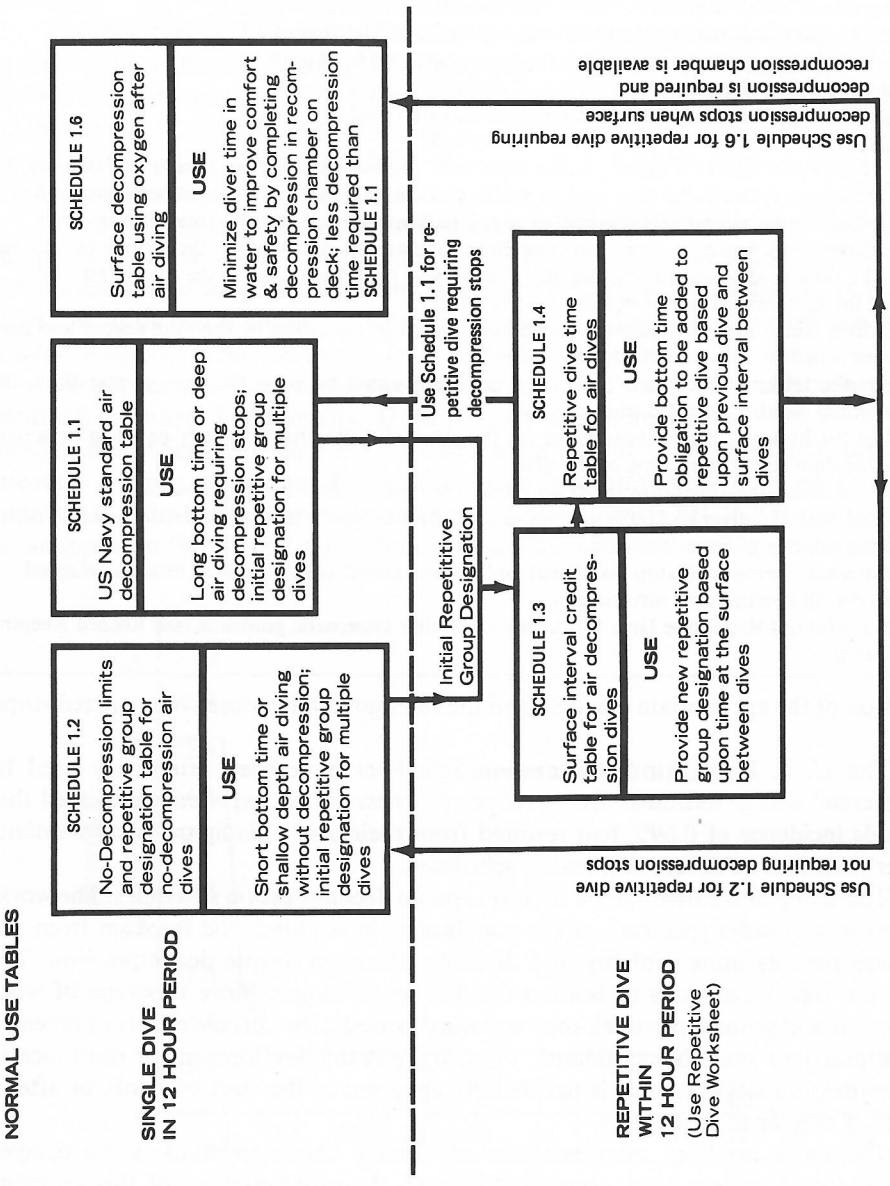


Figure VII-15. A guide to the use of the U. S. Navy Air Decompression Tables (Schedules 1.1–1.7).

Table VII-9
Limitations to U. S. Navy Air Decompression Schedules

Relevant terminology: see Decompression Schedules 1.1–1.7 at the end of this chapter
Equipment suitable for diving:
Open-circuit scuba (normal: 60 ft/60 min; exceptional: 130 ft/20 min)
Deep-sea gear (normal: 190 ft/40 min; exceptional: 250 ft/40 min)
Lightweight gear (normal: 60 ft/60 min; exceptional: 90 ft/60 min)
Variations in rate of ascent
1. Ascend from all dives at 60 ft/min unless otherwise noted
2. If unable to maintain the ascent rate of 60 ft/min:
   A. If the delay was at a depth greater than 50 ft, increase the bottom time of the dive by the difference between the time used in ascent and the time that should have been used at a rate of 60 ft/min; decompress according to the requirements of the new total bottom time
   B. If the delay was at a depth less than 50 ft, increase the first stop by the difference between the time used in ascent and the time that should have been used at the rate of 60 ft/min

Specific instructions for use of decompression tables
1. All dives which are not separately listed are covered in the tables by the next deeper and next longer schedule; do not interpolate
2. Enter the tables at the listed depth that is exactly equal to, or is the next greater than, the maximum depth attained during the dive
3. Select the bottom time of those listed for the selected depth that is exactly equal to or is next greater than the bottom time of the dive
4. Use the decompression stops listed on the line for the selected bottom time
5. Ensure that the diver's chest is maintained as close as possible to each decompression depth for the number of minutes listed
6. Commence timing each stop on arrival and resume ascent when specified time has elapsed
7. Observe all special table instructions
8. Always fill out Repetitive Dive Worksheet or similar systematic guideline (see Record Keeping Section)

selection of the appropriate schedule and the rules governing ascent rate, missed stops, etc.

The U. S. Navy Air Subsaturation Schedules have been extensively used by commercial and government divers. A recent review of fleet experience indicated that a bends incidence of 0.69% had resulted from their use. This figure is substantially better than that achieved with earlier schedules.

Tunneling and caisson work requires special decompression schedules. The workers cover a broader spectrum of the population; in addition, the freedom from immersion permits more mobility and diversity of action during decompression. The exposure time (analogous to bottom time) is much longer. Since this type of work leads to five exposures per week over sustained periods, the chronic effects of repeated decompressions must be considered. First, there is the development of resistance to decompression sickness; this is particularly apparent at the start of a job, or after a layoff of greater than ten days.

The more insidious manifestations of chronic decompressions is the delayed appearance of aseptic bone necrosis. Although the exact etiology of this crippling disease is still uncertain, definitive evidence exists that it arises due to decompressions from the especially long exposures that characterize compressed air work. Thus, a major impetus for the development of better decompression schedules for compressed air workers is the reduction of the incidence of aseptic necrosis.

The wide variability in age, weight, and training of compressed air workers demands that they be subjected to a stringent physical examination as an employment condition. Behnke (1970) reported that 18.5% of the examinees for work on the San Francisco tunnels were disqualified, "chiefly because of excess weight and pulmonary pathology." All men found with juxta-articular lesions were disqualified.

The incidence of decompression sickness with modern decompression procedures is generally less than 2% in compressed air work. Behnke (1970) reported an incidence of only 135 cases out of the 80,360 man-decompressions in the BART project; this is an incidence of 0.17%. In spite of the earlier indications that obese older men should be more susceptible to decompression sickness, no such distinctions could be drawn in the San Francisco experience.

More striking is the reduction in incidence of bone lesions that accompanied the adoption of the so-called "Washington State Tables" (Schedules 2.1–2.5). No bone lesions have been found in either the Washington State or San Francisco tunneling. This low incidence is consistent with the studies that have been carried out in the United Kingdom using the newly devised Blackpool Tables.

Decompression from compressed air work (tunnels and caissons) occurs in an air-filled chamber. Traditionally, U. S. workers decompress by a series of constant venting maneuvers, changing the decompression rate at preassigned pressures. The pressure is expressed in pounds per square inch (gauge), psig. The British procedure differs in that the Haldanian staging method is utilized. Figure VII-16 is the comparative decompression (U. K. and U. S.) following a 4-hr work shift at 24 psig (2.63 ATA).

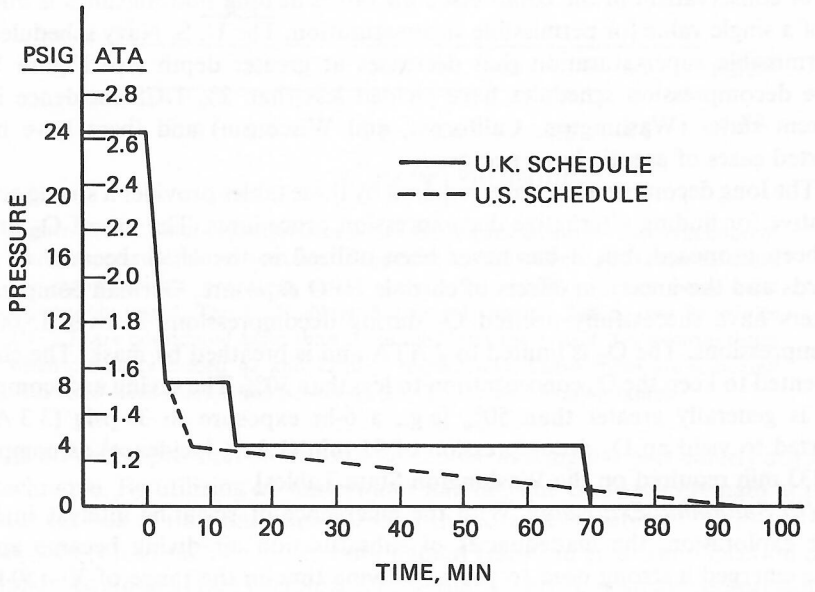


Figure VII-16. Decompression schedules for compressed air workers, resulting from a 4.0-h shift at 24 psig (2.63 ATA). The breathing gas is air. [Wisconsin (1971) and Decompression Sickness Panel (1973). Reprinted by permission from the Department of Industry, Labor and Human Relations, State of Wisconsin.]

Decompression schedules for this environment are generally developed by contractors for the insurance companies. The recent enactment of the Occupational Safety and Health Act (OSHA) resulted in the adoption of a national standard tunneling decompression procedure [the Washington State Tables (Schedules 2.1–2.5)]. These schedules were originally calculated by Duffner (1962), who utilized a three-tissue, perfusion-limited model (30, 60, 120 min half-times).* The use of a *split shift* (decompress to 1 ATA for lunch) was abolished on the grounds that it doubled the decompression hazard to the worker.

The permissible supersaturation in the Washington State Tables was based on U. S. Navy experience at that time; the 30- and 60-min tissues had surfacing N_2 partial pressures of 21 psig and that for the 120-min tissue had to be less than 18.6 psig N_2 pressure. The first decompression *stage* was at a rate of 5 psi/min (0.33 atm/min) and could not exceed a 16-psi drop in pressure or approach closer to the surface than 4 psi. The rate is then adjusted for the next stage based on a permissible supersaturation for the mean depth of the stage. This is repeated until reaching the 4-psi stop, where the final stage is commenced at the slowest rate of ascent consistent with arriving at the surface with safe supersaturation criteria.

Figure VII-17 indicates the total decompression time required for compressed air work at varying pressures and over the normal working time. The shaded area indicates the zones where the schedules have been extensively utilized. The dotted lines indicate the extreme exposures. The heavy bisecting lines separate the region where these tables are more conservative than the U. S. Navy air schedules from the region where the Extreme Exposure U. S. Navy air table is more conservative. This lack of conservatism in the compressed air tables at long bottom times is due to the use of a single value for permissible supersaturation. The U. S. Navy schedules utilize a permissible supersaturation that decreases at greater depth (see Figure VII-13). These decompression schedules have yielded less than 2% DCS incidence in three different states (Washington, California, and Wisconsin) and there have been no reported cases of aseptic bone necrosis.

The long decompression times required by these tables provides a strong economic incentive for finding alternative decompression procedures. The use of O_2 breathing has been proposed, but it has never been utilized in the U. S. because of the fire hazards and the uncertain effects of chronic HPO exposure. German compressed air workers have successfully utilized O_2 during decompressions in over 15,000 man-decompressions. The O_2 is limited to 2 ATA and is breathed by mask. The chambers are vented to keep the O_2 concentration to less than 30%. The saving in decompression time is generally greater than 50% [e.g., a 6-hr exposure to 35 psig (3.3 ATA) is reported to yield an O_2 decompression of 91 min (1.94% incidence) as compared to the 233 min required on the Washington State Tables].

(ii) Saturation Exposures. With the emergence of scientific interest in shallow water exploration, the inadequacies of subsaturation air diving became apparent. There emerged a strong need to prolong diving time in the range of 30–150 fsw and permit greater flexibility in diving profile. Saturation diving offers the opportunity to

* Duffner used absolute pressures in his model; for consistency they have been converted here to N_2 partial pressures, since air is used throughout the tables.

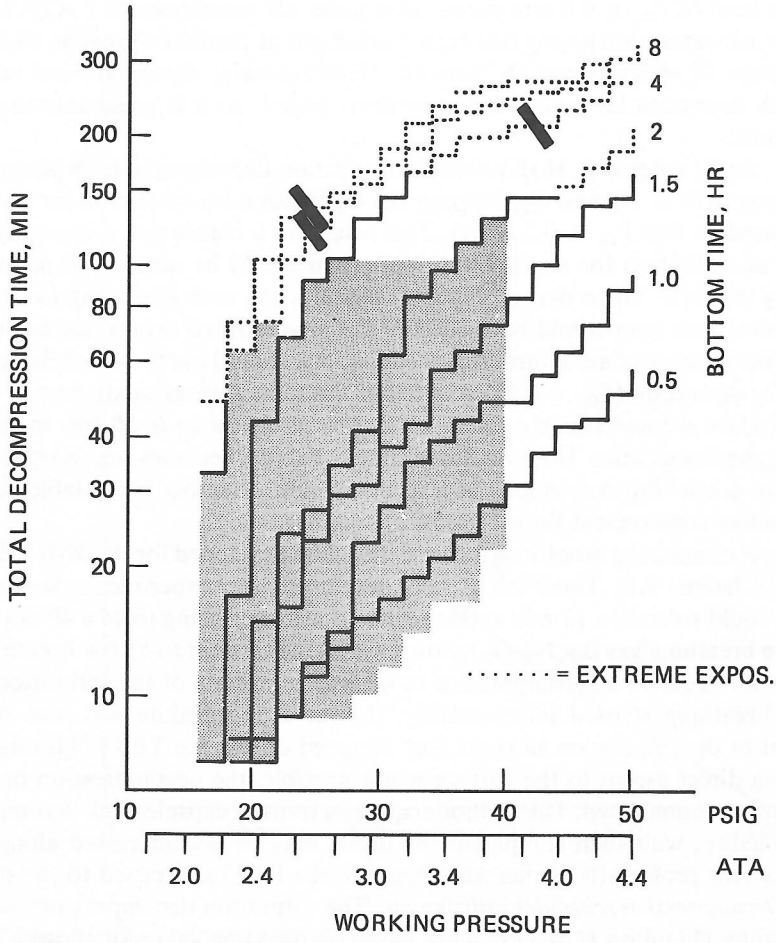


Figure VII-17. Total decompression time for compressed air work as a function of working pressure and bottom time (Schedules 2.1–2.5). The shaded zone indicates the recommended operating zone wherein the schedules have been most thoroughly tested. The U. S. Navy Extreme Exposure Tables (see Schedules 1.1–1.7) are considered safer than these schedules for 4- and 8-h bottom times when pressures exceed 42 and 22 psig, respectively. These zones are indicated with solid slash marks. See Schedules 2.1–2.5 for the detailed tables.

extend the no-decompression limits to these depths, making it a particularly attractive diving technique. By utilizing an underwater habitat, one could also remain at depth for greater periods of time and thus accomplish more useful work.*

Also, using air in such an underwater habitat results in great simplification of the diver-support equipment (see Chapter IX). All that is needed is an air compressor that can ventilate the habitat so as to keep the P_{CO_2} below tolerable limits. So long as the oxygen partial pressure is maintained less than 0.4 atm, oxygen toxicity is unlikely.

* Saturation diving was first developed to permit deeper diving on He–O_2 gas mixtures.

This limit of $P_{O_2} \leq 0.4$ atm presumably limits air saturations to 2 ATA (33 fsw). However, air-saturation diving has been carried out at depths as great as 99 fsw with no apparent ill effects from O_2 toxicity. Unfortunately, decompression schedules from such exposures have not been extensively tested; so it is preferable to retain a 2-ATA limit.

The most extensive shallow-water saturation-decompression experience has utilized mixtures of N_2 and O_2, keeping the O_2 fraction below that in air (generally, it is adjusted so that $P_{O_2} \simeq 0.2$ ATA). This results in a higher tissue inert-gas partial pressure at saturation for any habitat depth than would be achieved if air were the breathing mixture. These decompression schedules for such dives require more decompression time than would be needed for an air-saturated dive to the same depth. Thus, in the absence of air-saturation schedules, one should use the schedules available for N_2–O_2 saturation ($F_{O_2} < 0.21$) selected at the same saturation depth.

1. Surface decompression from air saturation at depths up to 50 fsw. In the event an emergency evacuation from an air-saturated habitat becomes necessary, one may surface for a brief interval as long as a recompression chamber is available on-site (a normal safety requirement for all saturation diving).

The recommended procedure is to use the tables developed for the 50-ft TEKTITE mission (Schedule 3.1). These tables were developed after experiments demonstrated that one could tolerate a 15-min surface interval after surfacing from a 40-fsw habitat where the breathing gas is a N_2–O_2 mixture (91/9) (equivalent to 51 fsw breathing air). Schedule 3.1 requires a recompression to 60 fsw, regardless of the saturation depth. Oxygen breathing is used intermittently. (Note: This schedule was also used for treatment of decompression sickness that occurred during the TEKTITE missions.)

When direct ascent to the surface is not possible, the decompression procedure is much more complicated. The method requires a transfer capsule-deck decompression (DDC) facility; with such equipment the divers may be decompressed along a continuous ascent profile after being transferred to the DDC and raised to the surface.

2. Decompression schedules utilizing air. The saturation decompression schedules developed by Hamilton *et al.* (1973) are based on the same values of allowable supersaturation as will be recommended below for excursion diving; therefore, they are recommended for use up to the depths to which they have been tested, 60 fsw. The general procedure involves decompressing at a constant rate of 1/6 ft/min to a given depth; from that depth to the surface a slower and continually changing rate of ascent is used. The only variable factor in the decompression is the depth at which one switches from 1/6 ft/min to the slower rate. Figure VII-18 shows the decompression from 60 fsw. The numbers along the curve indicate the saturation depth that results in an intersection with the continuous curve. Always use the saturation depth value at the 5-ft increment deeper than the saturation depth. Table VII-10 indicates the rate of ascent in 2- and 3-ft increments from 30 fsw to the surface, as is shown in Figure VII-18.

Decompression from saturation diving in the open sea poses several technical problems. If the saturation depth is less than 33 fsw (2 ATA), one can theoretically ascend directly to the surface at a reasonable rate. Because of the population variability in response to this decompression, the use of oxygen breathing in the habitat prior

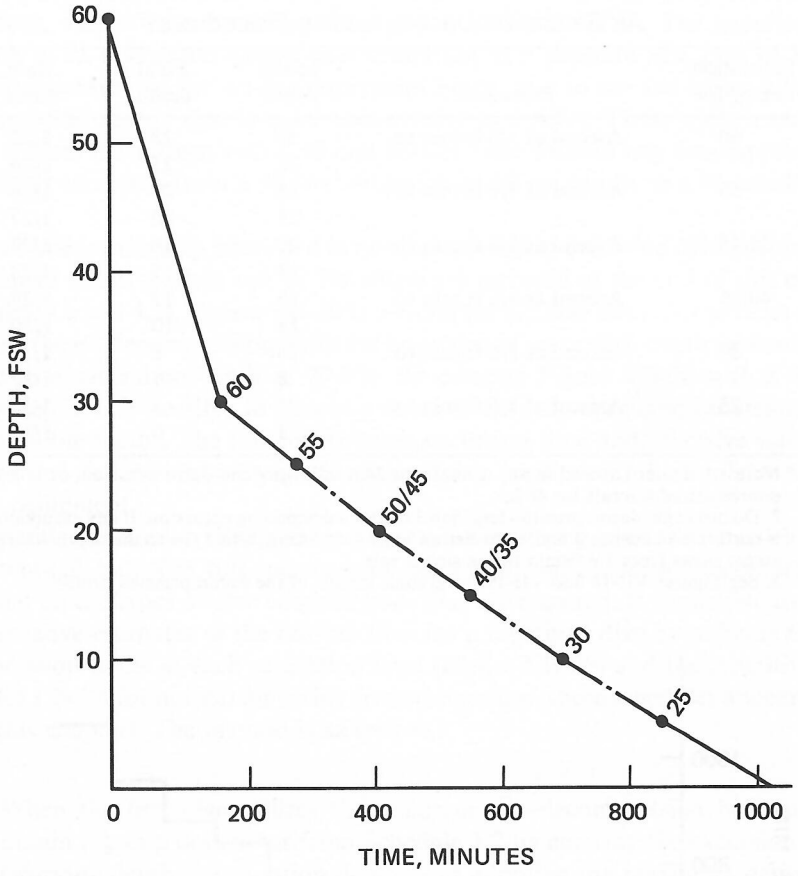


Figure VII-18. Decompression profile for saturation diving with air at depths up to 60 fsw. The numbers next to the curve indicate the point on this profile where decompressions from the depths below 60 fsw occur. See Table VII-10 for further details. (Hamilton *et al.* 1973.)

to ascent is probably warranted. Saturation diving should never be undertaken without the advice of a trained physician.

Figure VII-19 permits one to estimate the decompression time required from air saturation at any depth up to 60 fsw.

3. Saturation-excursion diving. Seldom is the ocean floor level around a habitat. In most habitat operations it has been necessary to undertake excursions from the saturation depth to accomplish one's goals. Generally, these excursions are short enough so that no decompression is necessary upon returning to the habitat. With increased exposure time, however, decompression stops are required. Excursions to shallower depths must be restricted so as to avoid inducing decompression sickness while at the excursion depth.

Table VII-10
Air Saturation Decompression Procedures[a]

Saturation depth, fsw	Procedure	Initial depth	Final depth	Rate, ft/min
60	Ascend at 1/6 ft/min to:	30	28	1/22
		28	25	1/24
50	Ascend at 1/6 ft/min to:	25	23	1/26
		23	20	1/27
50/45	Ascend at 1/6 ft/min to:	20	18	1/27
		18	15	1/28
40/35	Ascend at 1/6 ft/min to:	15	13	1/29
		13	10	1/30
30	Ascend at 1/6 ft/min to:	10	8	1/31
		8	5	1/32
25	Ascend at 1/6 ft/min to:	5	3	1/33
		3	0	1/34

[a] Notes: 1. Do not ascend in any aircraft for 24 h following end decompression, or in an unpressurized aircraft for 48 h.
2. Do not begin decompression less than 3 h after a descending excursion. If any excursion is performed between 3 and 12 hr before beginning ascent, add *5 fsw* to the depth where ascent slows from 1/6 ft/min to the slower rate.
3. See Figures VII-18 and VII-19 for graphic details of the decompression profile.

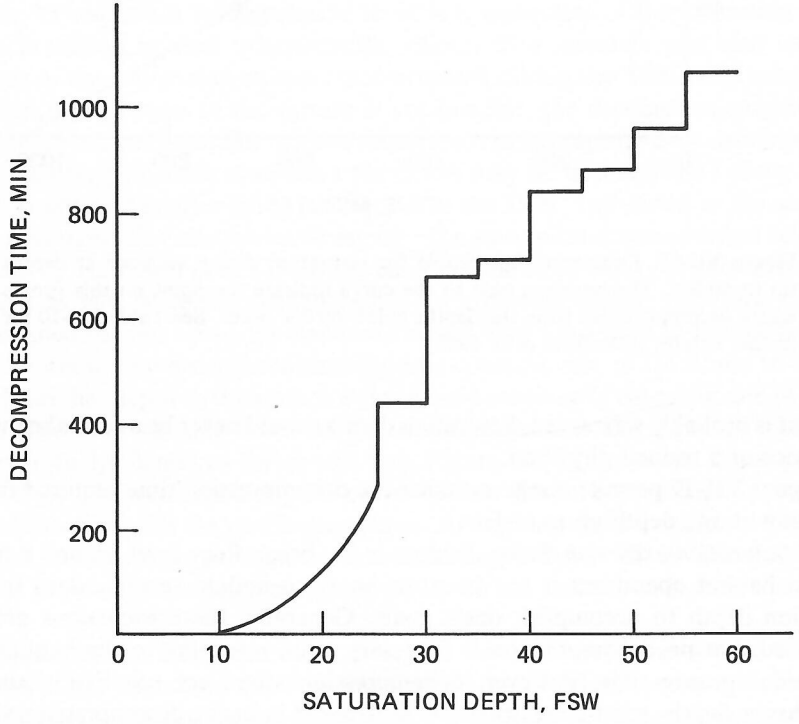


Figure VII-19. Total decompression time for air saturation diving at depths up to 60 fsw. See Table VII-10 for further details.

Descending excursion dives from saturation are entirely analogous to dives from the surface, where we are initially saturated at 0 fsw (1 ATA). The experience of Hamilton *et al.* (1973) has shown that saturation at a pressure in excess of 1 ATA results in extension of the no-decompression limits, due to the use of an allowable supersaturation matrix that is most conservative at 1 ATA. Thus, while a man can dive 80 ft from the surface with a 40-min bottom time without any decompression, a similar 80-ft excursion from a 30-fsw saturation platform results in a 68-min bottom time.

Since one is primarily interested in no-decompression excursion diving from habitats, detailed tables worked out by Hamilton are included at the end of this chapter (Schedules 4.1 and 4.2). Figure VII-20 is a crossplot of these data that permits one to determine the no-decompression limits for any value of saturation depth and excursion distance to a saturation depth of 70 fsw. By entering Figure VII-20 with a desired working depth, it is possible to choose a series of no-decompression excursion times and saturation depths. The tradeoff between excursion time and excessive saturation depth is often dependent on the goals of the diving mission and the available surface support equipment.

Repetitive excursion diving from air saturation diving platforms has not been simply worked out. The full spectrum of tissues can influence the decompression, depending on the types of dive combinations that are required. It is possible to arrive at conservative estimates of the bottom time for a repetitive dive by utilizing the no-decompression limits at each saturation level (Figure VII-20) and the repetitive dive Schedules 1.2–1.4 for normal air diving from the surface (these schedules appear at the end of this chapter). The method is as follows:

1. When the first dive utilizes the maximum no-decompression bottom time, obtain a group designator from Schedule 1.2 by entering the excursion depth (working depth − saturation depth) and adopting the maximum designator. Then use the surface interval, Schedule 1.3, to reduce this designator according to the time spent in the habitat at the saturation pressure. Then obtain the residual nitrogen time from Schedule 1.4, using the excursion depth; this time must be subtracted from the no-decompression time available for the second dive (obtain this no-decompression time from Figure VII-20).
2. When the bottom time in the first dive is less than the no-decompression limit, a *surface equivalent* excursion time (SEET) must be calculated for entry into Schedule 1.2. This SEET is obtained by multiplying the real excursion time (ET) by the ratio of maximum ET at the surface to maximum ET at the saturation depth D_{SAT}; i.e.,

$$\text{SEET} = \text{ET} \times \frac{\text{ET}_{MAX} \text{ at } D_{SAT} = 0}{\text{ET}_{MAX} \text{ at } D_{SAT}}$$

Use this SEET and the excursion depth to obtain a group designator from Schedule 1.2. After correcting for the surface interval, obtain the residual nitrogen time from Schedule 1.4 and subtract it from the maximum no-decompression ET at the excursion depth of the repetitive dive.

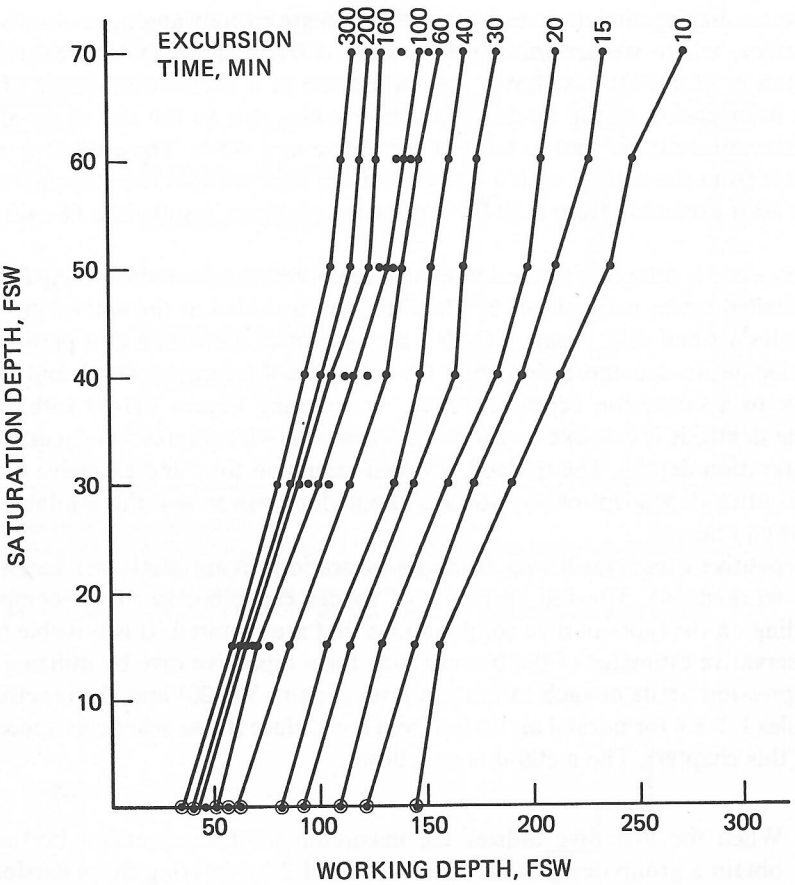


Figure VII-20. The air saturation depth as a function of the desired working depth and excursion time. No decompression is required for the excursion times. (Hamilton *et al.* 1973.)

Decompression schedules for repetitive diving beyond the no-decompression limits have not been developed. *Such procedures should be avoided!* In an emergency, use the U. S. Navy Decompression Tables, substituting excursion depth (working depth – saturation depth) for depth in the tables.

Excursion to depths shallower than the saturation depth carry the attendant risk of inducing decompression sickness while the diver is in the water. There was an incident in the TEKTITE dive of symptoms occurring when a diver made an excursion shallower than the habitat *after* having made a descending excursion earlier in the day. The NOAA instructions (Hamilton *et al.* 1973) accompanying the ascending excursion table (see Schedule 4.2) are worth quoting:

> Timing: Begin timing on arrival at excursion depth (bottom time does not include transit times). Rates: Ascend at 10 to 30 fpm. Descend at 60 fpm or faster if desired. If bends symptoms are noted, descend immediately to habitat. If descent to habitat is held up by ear problems, it is preferable to discontinue descent (or even momentarily ascend a bit) and clear the ear, rather than incur ear damage in order to adhere strictly to the table.

The ascent excursion schedules are based on a limited set of data, except in the region where there is no time limit. This latter region is based on the use of the same matrix of allowable supersaturations that was used for the descending excursion and decompression tables mentioned earlier. Notice that the tables suggest that one may decompress directly to the surface and safely remain there 36 min before returning to the habitat.

(iii) Air Decompression Schedules for Fresh Water Diving. Scientific and sport diving is often carried out in fresh water lakes and quarries. One may use the normal schedules given in the preceding sections, but corrected for depth. That is, 1 atm of pressure is equivalent to 33 ft of salt water and to 30 ft of fresh water. So a dive in fresh water to x feet is equivalent to a salt water dive of $(33/30)x$ feet. By entering the diving tables at this hypothetical depth, the appropriate decompression schedule can be selected. Each stop depth must be converted to the equivalent fresh water depth [i.e., stop depth in ffw = stop depth in fsw × (33/30)].

(iv) Air Decompression Schedules for Personnel and Patients within a Hyperbaric Chamber. Patients who are treated with high-pressure oxygen (HPO) are generally not at risk during decompression. The pressures are generally limited to less than 6 ATA and much of the exposures are spent breathing pure O_2. It is the attendants who are at risk; they are usually exercising, breathing air, and are most anxious to leave the chamber when the treatment is complete.

Modifications of the U. S. Navy air tables (Schedules 1.1–1.7) are probably the best decompression schedules for these purposes. Decompression rates between stops should be slowed to 15 ft/min (the usual 60 ft/min is appropriate for a diver anxious to leave the water). The last 10 ft (0.3 atm) should take 5 min. Finally, most centers utilize Behnke's (1967) recommendation that personnel breathe pure oxygen (readily available!) for 15 min during the decompression, while resting, and preferably at or about 60 fsw (2.82 ATA).

d. Mixed-Gas Decompression Schedules

Mixed-gas decompression schedules refer to diving environments in which gases other than air or O_2 are being utilized. Historically, the first deviation from air breathing was the use of helium to replace nitrogen in an attempt to reduce the decompression time required with air. Helium–oxygen mixtures were successfully utilized in the rescue and subsequent first U. S. Navy salvage of a sunken submarine, the SQUALUS. At present, there is a variety of gas mixtures that have been successfully used in operational diving.

The use of gas mixtures substantially complicates safe decompression. Since no simple procedures have been developed to correlate N_2 and He dives that are performed at the same depth for the same bottom times, He–O_2 decompression schedules must be developed from diving experience. However, there are substantially fewer controlled He–O_2 dives than there are air dives and the problem is further complicated by changing gas mixtures during the dive (e.g., from He–O_2 to air) or in utilizing a third inert gas, such as neon (tri-mix).

This section will, therefore, report only on fully tested schedules and refrain from speculation on the schedules to be utilized with novel gas mixtures. Any gas mixture

complicates the logistics of a dive and should only be utilized by qualified professionals who have successfully tested their decompression schedules in chambers. The recent observation of bends occurring with a change in inert gases, *without a decrease in pressure*, is an ominous comment on the state of knowledge regarding mixed-gas decompressions. It is always desirable to have a physician and treatment chamber available when utilizing mixed gases.

Mixed-gas decompression procedures utilize the concept that inert gas partial pressures are the determinant variables in computing decompression schedules. The relevant supersaturation parameters are the inert gas partial pressures in each tissue and the local absolute hydrostatic pressure.

One of the major reasons for using gas mixtures is to keep the P_{O_2} within tolerable limits (see Chapter IV for a discussion of O_2 toxicity). At a P_{O_2} in excess of 0.4 atm, one is limited in the time he may breathe O_2; this time decreases as the diver increases his work effort. The toxic results may be pulmonary or CNS abberrations. The generally accepted P_{O_2} time limits for working divers are given in Chapter IV.

Working with mixed gases greatly complicates life-support equipment. No longer can a simple air compressor be utilized. Extensive backup procedures and equipment must assure an adequate supply of the mixed gas. Emergency procedures should be available in case the mixed-gas source is inadequate and decompression must be completed using air. Everything possible must be done to conserve the gas. Toward this goal, many sophisticated systems recirculate the gas to the diver after passing it through a CO_2 scrubber. This recycled gas is mixed with fresh gas from the gas supply (see Chapter IV, discussion of breathing mixtures, for details of such equipment). Because the recycled gas has less O_2 than the fresh supply, the diver breathes a gas mixture that has a lower P_{O_2} than the supply. In computing decompression schedules, one must use the actual P_{O_2} (or an approximation) that the diver is breathing.

The following paragraphs divide mixed-gas decompression procedures into those utilized for He–O_2, N_2–O_2, and tri-gas mixtures.

(i) Helium–Oxygen Gas Mixtures. The use of helium as a respired inert gas was first proposed by the chemist Hildebrand (Sayers *et al.* 1925) because it is so much less lipid-soluble than is nitrogen. He reasoned that the long-half-time tissues in the body are really fat depots that store considerable quantitites of nitrogen. Helium, being much less soluble, ought to be rapidly removed; in fact, the half-time of a perfusion-limited tissue (see biological factors earlier in this chapter) ought to be reduced by the ratio of fat/blood partition coefficient between the two inert gases. In fact, this concept has been experimentally validated for other inert gases of higher molecular weight, so it surely does apply to helium and nitrogen.

Unfortunately, diving experience with helium has not reduced decompression time as predicted by the He–N_2 partition coefficient. The probable explanation is that the really slow tissues are not purely perfusion-limited, but also include some diffusion dependence. Alternatively, the slow tissues could be aqueous, rather than lipoidal, where the partition coefficients are not so different between He and N_2.

Helium diving has two major advantages: *The diver does not suffer inert gas narcosis*, so he may safely dive deeper than on air (the lower density of He–O_2 also makes breathing easier at depth), and *the decompression time is shorter* than the equivalent air decompression time (Table VII-11). The major disadvantages (other

Table VII-11
Comparative No-Decompression Limits for Scuba Diving with Air as He–O_2

	No-decompression time limit	
Depth, fsw	He–O_2[a]	Air
40	260	—
60	130	60
80	60	40
100	35	25
120	25	15
140	15	10
160	10	5
180	5	5

[a] U. S. Navy Schedules for Mk VI Semiclosed scuba with 68% He, 32% O_2.

than logistic complications) are that *the high thermal conductivity of helium* results in heating requirements and/or elevated respiratory heat loss (see Chapter IV, discussions of breathing mixtures and of cold) and *the alteration in speech* that accompanies He–O_2 breathing makes communications more difficult (see Chapter X). Oxygen toxicity is an ever-present hazard in He–O_2 diving because of the longer and deeper exposures. Table VII-12 lists the ten rules of safety for He–O_2 diving.

1. *Hard-hat schedules.* The first extensive use of He–O_2 was carried out in the late 1930's at the U. S. Navy Experimental Diving Unit. The schedules for hard-hat diving were then extensively tested during the salvage of the U. S. S. SQUALUS. These schedules were subsequently recalculated and retested by Molumphy (1950), resulting in the publication of the schedules that are still in use today for hard-hat diving (see Schedules 5.1–5.4 and 7.1 at the end of this chapter). These schedules were calculated with a Haldanian model that included the following assumptions:

Breathing gases

1. Constant F_{O_2}(0.14–0.21).
2. Oxygen breathing at 60 and 50 ft (assume $F_{O_2} = 0.8$).
3. Express depth in terms of helium partial pressure, so that

$$P_{He} = (d + 33)[1.0 - (F_{O_2} - 0.02)]$$

where d is the depth and F_{O_2} is the oxygen fraction.

Helium uptake and washout:

1. Assume a tissue spectrum where $t_{\frac{1}{2}} = 5, 10, 20, 30, 40, 50, 60, 70$ min.
2. Use twice the actual bottom time to calculate the helium loadings in each tissue (to account for exercise at depth).
3. Treat the nitrogen that is equilibrated at surface (26 ft) as helium.

Permissible supersaturation:

A constant ratio of tissue partial pressure to local barometric pressure was selected as (1.7:1.0). This was not depth dependent, nor did it vary throughout the tissue spectrum.

Table VII-12
The Ten Rules for He–O_2 Breathing, to Avoid Oxygen Toxicity Problems

Loss of He–O_2 *Supply*
Deeper than 50 ft
1. Shift to air, come all the way out in accordance with Emergency Air Table (Schedule 5.4); no surface decompression

Loss at 50-ft stop
2. Shift to air (or He–O_2); complete stop in accordance with Emergency Air Table (Schedule 5.4) (or He–O_2, Schedule 5.3); can surface decompress after 30 ft stop; O_2 time is good time

Loss at 40-ft stop
3. Not within surface decompression or emergency surface decompression limits: same treatment as above
4. Within emergency surface decompression limits: Surface-decompress diver; double missed time of required water stop for surface decompression and add to chamber stop
5. Within normal surface decompression limits: Surface-decompress normally

O_2 *Toxicity Symptoms*
Symptoms at 50-ft stop
6. Ascend to 40-ft stop; shift to air (or He–O_2); can surface-decompress after 30-ft stop; disregard missed time at 50 ft

Symptoms at 40-ft stop
7. Not within surface decompression or emergency surface decompression limits: Ascend to 30 ft stop; shift to air (or He–O_2); can surface-decompress after 30 ft stop; disregard missed time at 40 ft
8. Within emergency surface decompression limits: Surface-decompress diver; double missed time of required water stop for surface decompression and add to chamber stop
9. Within normal surface decompression limits: Surface-decompress normally
10. Symptoms during chamber stop: Remove mask; complete decompression in accordance with Emergency Air Table (Schedule 5.4); O_2 time is good time

The decompression tables available for hard-hat diving (Schedules 5.1–5.4 and 7.1) are summarized in Figure VII-21. As with air diving, there are no-decompression limits, normal diving (limited by O_2 toxicity hazards), exceptional exposure tables, emergency tables (with and without He–O_2), and surface decompression procedures. Repetitive diving is NOT permitted within a 12-hr period.

The schedules have been used extensively by the U. S. Navy and many commercial firms. The U. S. Navy incidence of decompression sickness has been reported to be 0.83% when using the schedules.

2. *Mixed-gas scuba schedules.* The U. S. Navy developed He–O_2 decompression procedures for modified scuba equipment. They were developed in order to reduce the risk of CO_2 retention, oxygen toxicity, nitrogen narcosis, and the overexertion of breathing denser air. The equipment involves a rebreathing circuit; Figure VII-22 is a schematic of the U. S. Navy Mk. VI semiclosed diving apparatus. (Commerical diving systems are available that utilize the same decompression procedures.) The gas mixture has an elevated F_{O_2} (0.32 is normal and 0.40 is permitted) into the system at a rate that is dependent on the depth and exercise level of the dive. After mixing with the recycled gas in the inhalation bag, the F_{O_2} is lowered to a predictable level. By carrying a separate bottle of pure oxygen and a crossover switch, divers can decompress at 30 and 20 fsw on pure oxygen.

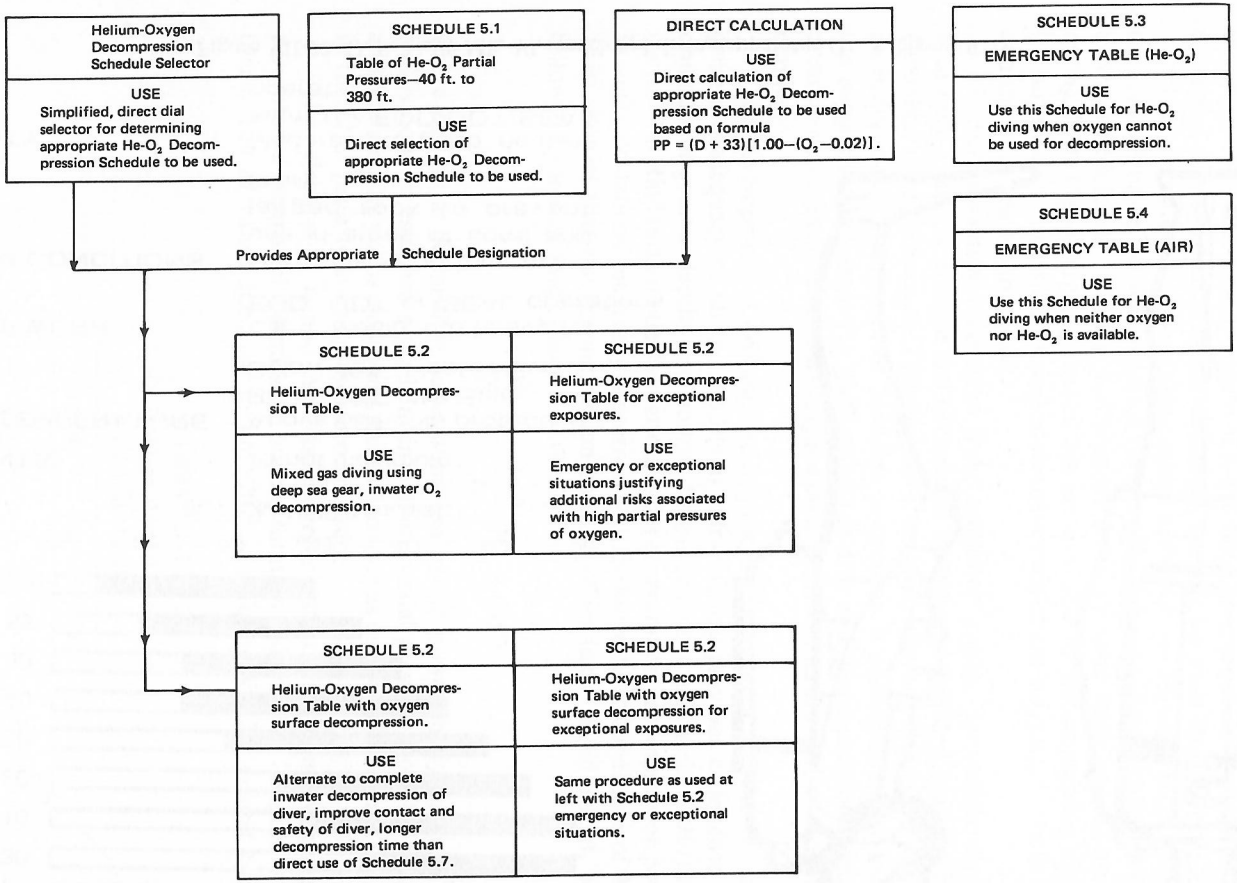
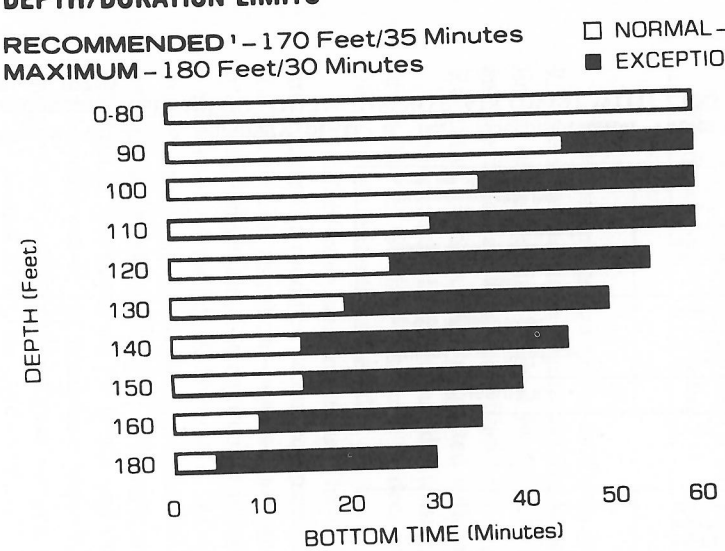


Figure VII-21. A summary of U. S. Navy Decompression Tables for the use of He–O_2 breathing while diving with deep sea diving equipment. REPETITIVE DIVING IS NOT ALLOWED WITH LESS THAN A 12-HR SURFACE INTERVAL (see Schedules 5.1–5.9). (U. S. Navy 1971.)

DEPTH/DURATION LIMITS

RECOMMENDED [1] – 170 Feet/35 Minutes
MAXIMUM – 180 Feet/30 Minutes



CURRENTS	1 knot maximum
WATER TEMPERATURE	Above 68°F: No protection Below 68°F: Wet Suit Below 45°F: Heated Suit
TYPE OF WORK	Light; Medium to heavy for EOD, UDT or SEAL operations
BOTTOM CONDITIONS	Hard, clean bottom; avoid use in areas of coral and jagged rock to prevent injury
VISIBILITY	Moderate to good, no minimum for EOD/UDT/SEAL operations

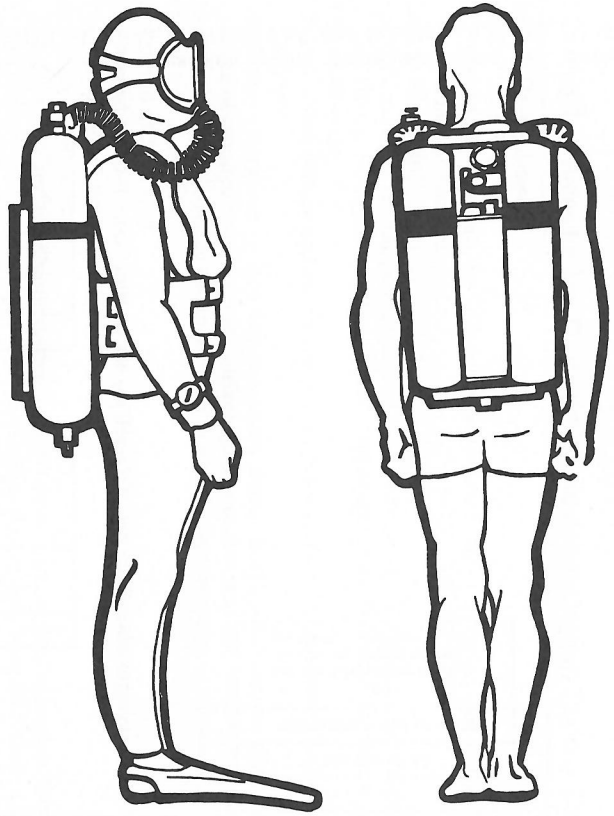


Figure VII-22. U. S. Navy Mk. VI semiclosed diving apparatus (U. S. Navy 1971).

The mixed-gas scuba decompression schedules (Schedules 5.5–5.9 at the end of this chapter) were developed by several U. S. Navy investigators. They were completed (including the repetitive schedules) by Workman and Reynolds (1965). The model utilized a tissue half-time spectrum of 5–120 min. The permissible supersaturation was expressed as a constant pressure difference, varying for each tissue (Table VII-13).

The procedures for using these tables are shown in Figure VII-23. Initial tables are selected based on the projected dive profile and equipment available. Repetitive diving schedules are selected and computed on the same basis as was used for air diving. Note that emergency procedures are available for decompressing with air and for surface decompression. The schedules have been extensively tested in both chambers and the open sea environment.

3. *Decompression procedures from saturation diving.* Saturation diving while breathing a He–O_2 mixture has become an operational reality during the last 10 yr. Chamber tests as deep as 1700 fsw have been carried out and operational diving procedures exist for depths of 1000 fsw. Saturation is accomplished by maintaining the oxygen at a constant P_{O_2} throughout the dive and decompression. Since decompression occurs inside chambers, oxygen breathing from masks is often used at shallow depths.

The development of decompression procedures for great depths has shown the general inadequacy of all the models in extrapolating previous experience into new domains of greater depths and bottom times. Schreiner and Kelly (1967) articulate the general Haldanian model for various types of saturation decompression procedures. In such calculations one is only concerned with the longest half-time tissue of the model. They showed that the decompression consists of an initial rapid ascent, or "pull," followed by a slow rate of ascent, given by

$$\frac{dP}{dt} = -\frac{\ln 2}{t_{\frac{1}{2}}}(P_{O_2} + \Delta P) \tag{20}$$

where ΔP is the permissible supersaturation of the slowest body tissue. The initial pull is done to establish a gradient in helium partial pressure from the tissue to the ambient air. The pull is a constant amount so long as no excursion diving has occurred in the 12 hr prior to starting the decompression.

The selection of $t_{\frac{1}{2}}$ and ΔP has undergone steady revision. Workman (1965) presented a matrix of permissible supersaturation values that included a 240-min tissue. The M values [i.e., ΔP in Eq. (20)] (see Figure VII-24) exhibit two general

Table VII-13
Safe Helium Partial Pressure upon Surfacing

$t_{\frac{1}{2}}$, min	$P_{He} - 33$, fsw
5	83
10	59
20	41
40	29
80	23
120	20

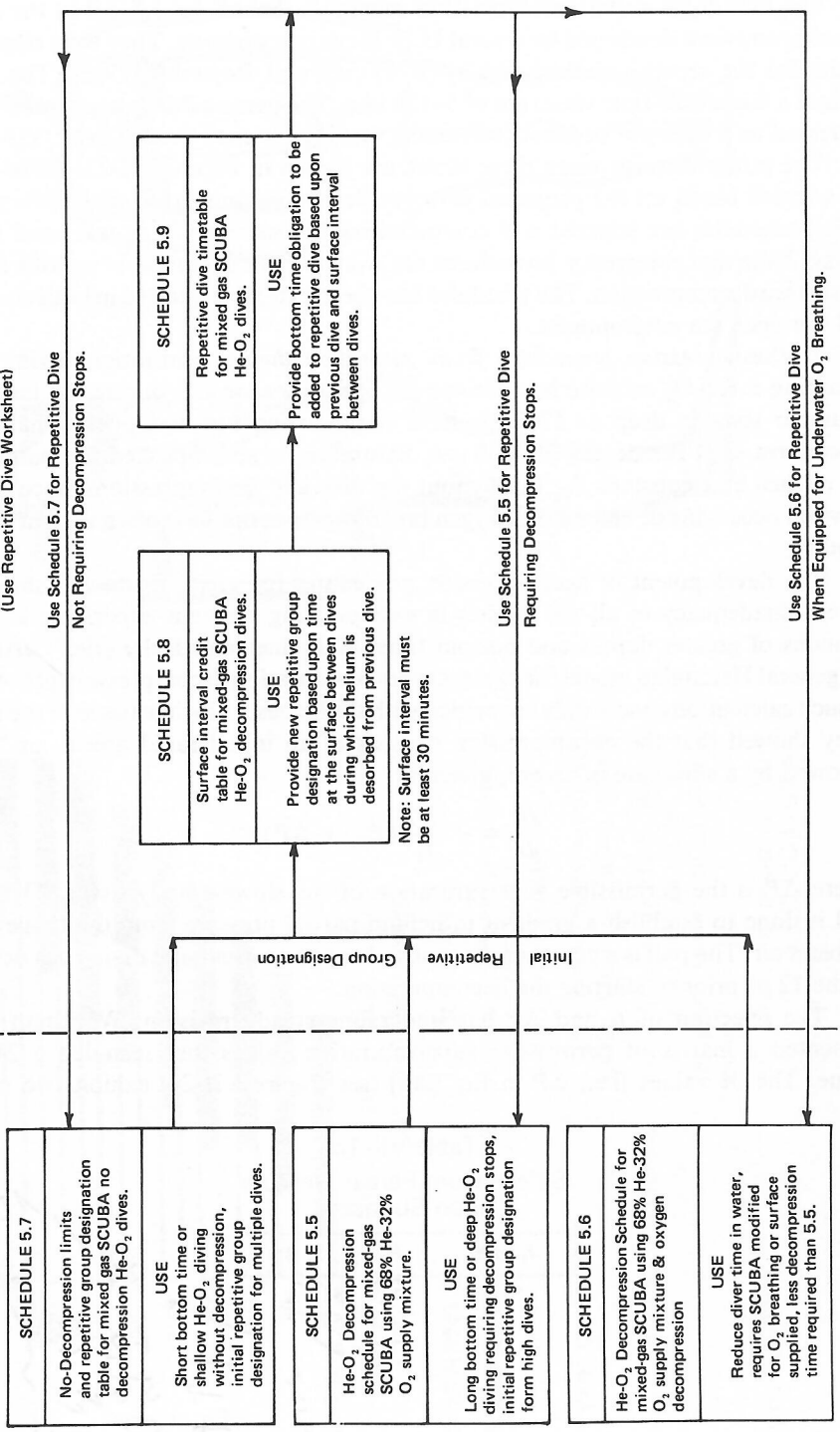


Figure VII-23. A summary of U. S. Navy Decompression Schedules for use with mixed-gas scuba (U. S. Navy 1971).

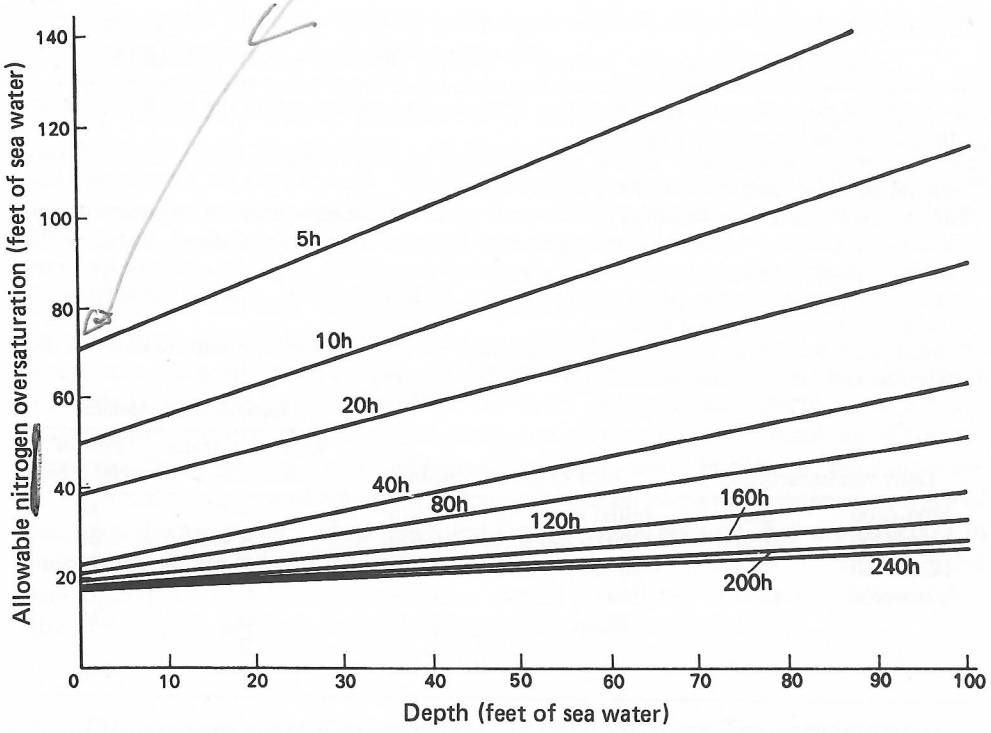


Figure VII-24. The permissible supersaturation of helium partial pressure as a function of depth and tissue half-time [data of Workman and Reynolds (1965)].

characteristics that have been extensively validated: (1) As $t_{\frac{1}{2}}$ increases, M decreases, (2) as depth increases, M increases linearly (except for the M value of the slowest tissue, which is independent of depth). Thus, one would project from the above equation for dP/dt that a linear rate of ascent would yield a safe decompression schedule.

During testing of these schedules, several compromises were made that ultimately yield a much slower decompression than these considerations suggest is necessary. Overnight stops were instituted so as to permit the divers time to rest. Because the divers are relatively secure in chambers, the need to achieve a speedy decompression was deemed secondary to a safe procedure. So these slower schedules have never really been tested against the model and should not be interpreted as implying that a much longer tissue half-time is required.

The U. S. Navy procedures utilize a decompression curve that is a series of ascent rates, gradually slowing down as the surface is approached. The decompression begins with a 30-fsw pull. Table VII-14 summarizes the procedure.

4. *Excursion diving from saturation.* One may ascend up to 30 fsw from a saturation pressure once within a 24-hr period. Descending excursions depend on the saturation depth and the bottom time that is desired. Schedules 6.1–6.3 (at the end of this chapter) indicate permissible bottom times and a repetitive group designation used for repetitive excursion diving. Schedules 6.3 may be used to compensate for time in the

Table VII-14
General Instructions for Saturation Diving Decompression Schedules

Divers should normally remain at saturation depth for 24 h subsequent to the last excursion for the purpose of reequilibrium of tissues

Chamber oxygen environment should be maintained at 0.30–0.32 atm; carbon dioxide levels should be maintained less than 0.5% surface equivalent

"Initial ascent" represents the initial rapid ascent rate at the commencement of decompression to establish a gradient for inert gas elimination; to find the initial ascent distance, find from the habitat internal credit table the repetitive group designation of the diver who has the most residual helium; the initial ascent distance is given in the table below, opposite the appropriate repetitive group designation letter

To adequately decompress from a saturated condition the diver must follow both the daily routine schedule and rate of ascent schedule given below

Daily routine schedule		Rate of ascent schedule		Initial ascent distance	
				Repetitive group designation	Feet of initial ascent
2400–0600	Stop	Initial ascent	10 ft/hr	—	30
0600–1400	Ascend	1000 ft–200 ft	6 ft/hr	A	25
1400–1600	Stop	200 ft–100 ft	5 ft/hr	B	20
1600–2400	Ascend	100 ft– 50 ft	4 ft/hr	C	15
		50 ft– 0 ft	3 ft/hr	D	10
				E	5
				F	0

habitat between dives. These excursion tables have been extensively tested by the U. S. Navy.

5. *Emergency abort schedules from saturation habitats.* Abort schedules were calculated using the matrix from Figure VII-24. They have not been extensively tested. The schedules are included at the end of this chapter (Schedule 7.1).

(ii) N_2–O_2 Gas Mixtures. Generally nitrogen–oxygen mixtures are only used for saturation diving in shallow depths. The mixture is used to reduce the P_{O_2} to avoid toxic responses. Decompression procedures have been developed for both excursion diving from saturation and decompression to sea level. The schedules are based on extensive compilations of Hamilton *et al.* (1973) (Schedules 4.1 and 4.2). The matrix of tissue half-times and permissible supersaturations was previously discussed under air saturation diving.

E. *Treatment*

In all diagnosed cases of decompression sickness, the patient must be moved rapidly to a recompression chamber where he may be treated. All planning for diving activities should include the identification of the nearest treatment facility and a plan for being able to get to the facility rapidly in the case of an accident.

There are two phases in the treatment of DCS: *diagnosis* and *emergency first aid* during transportation to a facility and *treatment* in a recompression chamber. The first phase is generally supervised by nonmedical personnel. The second phase is generally directed by a medically trained chamber operator with a local physician being available