PART ONE: DECOMPRESSION SCIENCE

I. INTRODUCTION: PHILOSOPHY OF DECOMPRESSION TABLE DEVELOPMENT

The DCAP program is concerned with decompression table development, a process that involves physiology and biophysics, medicine, and diving operations, and a good bit of philosophy.

A. Background

When a person is exposed to a hyperbaric environment, the elevated gas pressure that must be breathed causes an increase in the quantity of gas dissolved in his (or her: The female diver is always implied in this manual if not always stated) body. Subsequent ascent to a lower environmental pressure can cause this gas to come out of solution, and this can produce, either directly or indirectly, a malady called decompression sickness.

The evidence that bubbles (or gas that has come out of solution) are the cause of decompression sickness, though indirect, is overwhelming. Decompression sickness (DCS) may be manifested as rash, pain, sensory disturbances, paralysis, or under exceptional circumstances, even death. It can be avoided by controlling the ascent of the diver in such a way that the dissolved gas is released slowly, slow enough that it does not form more bubbles or "gas phase" than the body can handle. The plan or procedure for the regulation of an ascent profile in order to control the formation or growth of bubbles is called a decompression "table." In other usage the profile for a single dive is called a "schedule" and a "table" covers several dives having different depths and/or bottom times. A decompression procedure involves primarily a time-pressure-gas profile, but many factors are involved in determining a procedure and in whether or not it is effective in returning a diver to shallower depths and to the surface without ill effects. These factors, as well as the aspects of diving operations that affect decompression, are discussed in Chapters II and III.

Two things need to be pointed out about the process of decompression table development. First, although many general physiological and biophysical aspects of decompression and decompression sickness are well known, many aspects of the actual mechanisms are poorly understood. Thus the development and final evaluation of decompression procedures must at this time be made from a strictly **empirical** process.

Second, the occurrence of decompression sickness is a "statistical" or "stochastic" event, one based on **probability**. Decompression procedures attempt to reduce this probability to an acceptable level, but no practical procedures can reduce the probability of DCS to zero. This has to be considered in assessing the effectiveness of a set of tables, and it has the further meaning that all diving operations must be able

to deal with DCS should it occur. Although this requirement for medical resources is a part of all diving, normal

procedures for reliable decompression should be regarded as **operational physiology** and not **medical practice**.

The description of the use of DCAP in this manual is primarily oriented toward diving. Other situations also involve decompression, and DCAP can be used for predictive calculations and analysis in most or all of them. These include tunnel and caisson work, medical practice involv-ing hyperbaric oxygen therapy, escape from distressed submarines, flight in high altitude aircraft, and space flight, as well as the research and training necessary to support these endeavors.

B. Validation of decompression tables

One highly complex philosophical issue deserves mention, the matter of **validation** of decompression tables once they are produced. Fortunately a recent workshop held by the Undersea and Hyperbaric Medical Society (Schreiner and Hamilton, 1989; references for Part One are found as Chapter XI at the end of Part Two) has addressed this question, and the proceedings of that Workshop provide the most current consensus thinking of experts in this field.

The procedures discussed by the Workshop reflect current practice in decompression table development, which includes the familiar steps of computation based on available experience, laboratory testing, provisional use at sea, and operational acceptance, all threaded with several feedback loops. There has always been a problem determining when enough testing has been done, and more specifically on who should make the decisions and how to make them. The Workshop made two significant contributions; the first was to point out that the developing organization had the responsibility for the development process, and the second was to define a mechanism for taking the steps that require judgement, such as answering the difficult question of when to move from one phase to another. The Workshop was clear that decisions should be made by the agency developing the tables (a diving company, a navy, etc.), not by government or some central authority. The judgement could come from a body within the organization, a small group or committee that is charged with the responsibility for making the decisions and has the expertise to do so; this might be called a "Decompression Decision Board." Experts from out-side the organization has the responsibility and stands by the decisions.

II. OVERVIEW OF DECOMPRESSION PHYSIOLOGY AND BIOPHYSICS

The objective of a proper decompression is to avoid decompression sickness (DCS). This is accomplished primarily by limiting the formation and growth of bubbles (or separated gas) in the body by making the ascent slow enough and by manipulating the breathing gases. This section deals with the management of gases in the body during decompression. It covers the basic mechanism as well as other factors related to diving operations that are used in planning decompression strategy. It touches on the physiology and biophysics that are the basis for the mathematical models used for making decompression calculations, conditions that cause deviations, and some consequences of the hyperbaric exposure, specifically oxygen toxicity and treatment of decompression sickness.

It is important to point out that the various "models" used in decompression computation are **HIGHLY** theoretical and hypothetical. Assumptions about tissues, supersaturation, diffusion and perfusion, bubble formation and growth, etc., etc., while they may be based on valid physical, chemical, and biological principles, are in reality only gross generalizations about what is really going on in the body. They are useful, but they are probably not real.

A. Gas uptake and elimination

DCAP, when properly configured, can handle different "models" of gas exchange on which the computation of decompression tables is based. The primary one is the Haldane-Workman-Schreiner model, a modern version of the classical Haldane model which is also called "neo-Haldanian" (Schreiner and Kelley, 1971). This section discusses the biophysical background of that model and serves as a basis for others; the models themselves are covered in Chapter IV.

Gases enter or leave body tissues depending on whether the partial pressure of the gases in arterial blood is greater or less than their partial pressure in the tissues. The rate at which a gas is taken up or eliminated by any tissue depends—primarily—on the partial pressure gradient of the gas between the blood and tissue, the respective solubilities of the gas in blood and the tissue, the rate of blood flow through the tissue, and the diffusion rate and path of the gas into and within the tissue. There is still some slight controversy over whether one or the other of the latter two factors, perfusion or diffusion, limits the rate of gas exchange, but most sentiment today leans toward perfusion as the major limiting factor. At any rate, it now seems established that this distinction is not very important.

It is generally considered that gas uptake and elimination proceed in an exponential fashion, taking place rapidly when gas pressure gradients are high and more slowly when gradients are low. There are several variables that disturb this relationship, and evidence is mounting that the process is not symmetrical, that elimination may be slower than uptake.

Because of differences in tissue characteristics such as lipid-water content, blood flow, and diffusion distances, there are a wide range of gas uptake and elimination patterns in man. A tissue with a gas solubility nearly equal to that of blood, a high blood flow and a high capillary density will have a relatively

rapid gas uptake and elimination profile. A tissue with a high gas solubility relative to blood, a low blood flow and a low capillary density will have a slow exchange pattern.

For purposes of this discussion the blood is assumed to become equilibrated with the gases in the lung alveoli during its passage through the lungs. This alveolar gas reflects the composition of the inspired gas mixture, but differs from it slightly due to water vapor and the metabolic exchange of oxygen and carbon dioxide. In actual fact this equilibration may be far from complete, and it may be affected by various factors.

The amount of gas taken up by the body during a hyperbaric exposure depends on the partial pressures of the inspired gases (a function of the depth—pressure—of the exposure and the gas composition), the duration of the exposure, the solubility of the gas, and how much of that gas is already present. The deeper the diving depth the greater the gradient for gas uptake and the greater the maximum amount of gas that can be taken up. This applies to all gases, but we are primarily concerned here with inert gases, those that do not take part in body metabolism. The longer an exposure, the closer each tissue will come to reaching equilibrium with the composition of the gas being breathed. Once equilibrium between the breathing gas and tissue has been reached, however, no additional gas will be taken up even if the exposure is extended in time. This condition is called "saturation." In diving a practical saturation limit is reached somewhat before biophysical saturation.

If the sum of gas partial pressures dissolved in a tissue is greater than the ambient pressure, the tissue is said to be supersaturated. This can occur by a reduction in ambient pressure, or in some situations by "counterdiffusion" of different gases.

The many factors influencing perfusion and diffusion of body tissues can affect gas uptake and elimination rates in diving. Among these are temperature, circulatory obstruction or autoregulation, activity level, blood oxygen and carbon dioxide levels, and available blood volume.

B. Micronuclei and bubble formation

Bubbles will form in biological systems only if gas micronuclei are present. Bubbles do not form in experiments in which all other conditions for bubble formation are present but where micronuclei have been eliminated (for example, by filtration or compression to extreme pressure). Micronuclei exist normally in biological systems and they probably take several forms. The cause of their formation is not certain, but they may form in microscopic crevices or be due to such things as large local mechanical stresses in joints, etc. The very high levels of supersaturation that are necessary to produce *de novo* nucleation compared with the relatively low supersaturations that occur as a result of decompression are strong evidence that gas micronuclei are preexisting and not formed by the act of decompression itself.

A liquid that contains micronuclei cannot sustain supersaturation for more than a short time without bubble growth occurring. Therefore, if during decompression the pressure of the gases dissolved in a tissue exceeds the sum of the pressures opposing it (these are the ambient pressure, the surface tension at the separated gas phase, and the mechanical forces resisting deformation within the tissue itself) bubble "formation" and growth will occur. This is accelerated by rapid "stirring" of the fluids involved, hence by normal circulation and body movement.

DCAP Manual. II. Overview of decompression physiology and biophysicsPart One.C. Use of different inert gases

Some phase separation most likely occurs in all practical decompressions. Doppler ultrasonic monitoring has shown that bubbles do exist during decompression, even in the absence of symptoms of DCS. Bubbles are no doubt directly involved in causing the symptoms of decompression sickness; they definitely damage the blood, and they also appear to cause disturbances in the linings of the blood vessels. A bubble—the air-fluid interface—is regarded as a foreign body, and it can invoke all the appropriate reactions. Bubbles are normally trapped by the lungs, preventing damage to the brain and other organs, but their presence in lungs causes release of biologically active substances that can have secondary effects, and it appears that some bubbles might pass into the arterial side of the circulation through a patent *foramen ovale*, an opening between the right (venous) and left (arterial) sides of the heart. About 15% of normal people have a potentially open foramen ovale; evidence is mixed as to whether this is an important factor in decompression sickness or arterial gas embolism

C. Use of different inert gases

In the following discussion about inert gases, although it is not always mentioned, oxygen is always present in any suitable breathing gas.

1. Gas properties

Because of the different physical properties and biological effects of several inert gases—nitrogen, helium, neon, and hydrogen—each has a use or potential use in professional diving. Differences in thermal conductivity, speech intelligibility, narcotic potency, and effects on pulmonary mechanical function are all important, but from the standpoint of decompression the differences in the solubilities and exchange rates of the inert gases are most important. Argon, while not of much value as a diving gas because of its high solubility and density, also has to be considered because it is used in hyperbaric welding as a shield gas and thus may be breathed by divers, and it may be formed by oxygen generation systems on high performance aircraft.

The characteristics determining the exchange rate of a gas are its diffusivity and its solubilities in fat and water. The relative importance of these characteristics depends on the rate-limiting factor, its perfusion or diffusion in the tissue. Because of their different physical properties, some gases—helium for example—are taken up and eliminated from the body relatively quickly. Other gases such as nitrogen have slower exchange rates. For this reason, the choice of the sequence of inert gases used in a dive can greatly influence the decompression time. Hypothetically, for a deep dive with long bottom time the slow elimination rate of nitrogen necessitates a longer decompression than helium. For a shorter dive, however, the slow uptake rate of nitrogen might produce less decompression obligation than would helium. It is important to note that although these factors can be calculated in some cases with great precision, the calculations are extraordinary oversimplifications and may not necessarily work out in the same quantitative manner in real life; such calculations can be relied upon only when there is some experience to back them up.

2. Gas switching and sequencing

Differences in the exchange rates of inert gases also allow decompression time reduction through the switching of inert gases during the dive. By changing the inspired inert gas to one taken up by the tissues

more slowly than the first is eliminated, the calculated decompression times can be reduced. This practice is commonly followed in commercial diving where decompression from helium-oxygen dives incorporates a switch to air or a nitrogen-oxygen mixture during the decompression.

If a switch is made from a slowly exchanging gas to a rapidly exchanging gas during a dive, the opposite situation can occur. That is, the tissue inert gas pressure can rise and with it the decompression obligation. If such a switch were made during a decompression, tissue gas pressures could increase and decompression sickness could result, even without further ascent.

These gas-specific factors can be manipulated by the computational models, but how valid the models are is difficult to determine empirically or experimentally. One general principle is that gas changes may take place instantly in the computer, but they certainly do not change so quickly in the body, and this has to be taken into account in table computation.

3. Counterdiffusion

The effect just mentioned, of an excess of gas and the possibility of bubbles developing from a specific gas switch, is one aspect of a phenomenon known as *isobaric counterdiffusion*. Counterdiffusion effects may also be observed if a diver breathes a mix containing a slowly diffusing inert gas such as nitrogen while surrounded by a more rapidly diffusing inert gas such as helium; the differences in gas flux across his skin can result in the formation of bubbles even if there has been no change in depth. The usual manifestation is intense itching of the skin, with rash and possibly "blebs" of gas on exposed areas of skin. If this process is allowed to proceed for too long a time, injury or death can result from gas emboli within the circulatory system.

The practice followed routinely in commercial diving is to keep the inspired and surrounding inert gases the same. Even so, derangement of the vestibular organs of the inner ear has been observed to follow an abrupt switch in the inspired inert gas from helium to nitrogen. It is possible that those vestibular problems are caused or intensified by counterdiffusion between the residual helium in the middle ear and the newly-acquired nitrogen carried by the blood. Thus it is recommended that switches from one gas to another be made gradually. There is evidence also that other forms of decompression sickness may follow an abrupt gas switch; these too are felt to be controlled or reduced by avoiding abrupt switching.

D. The role of oxygen

The use of oxygen in hyperbaric exposures can increase the efficiency and safety of decompression procedures. This is due in large part to its replacement of inert gas in the breathing mixtures, but in some cases the improvement in decompression (and treatment of DCS) due to increased oxygen levels seems to be greater than can be accounted for solely by its replacement of inert gas. Excessive exposure to oxygen can be toxic to the lungs and central nervous system. Thus an important objective in planning diving operations is to take full advantage of the usefulness of oxygen while avoiding its toxicity.

DCAP Manual. II. Overview of decompression physiology and biophysicsPart One.D. The role of oxygen

1. The "oxygen window"

The oxygen fraction in the inspired gas replaces a portion of the inert gas constituting the balance of the mixture. It is the inert gases dissolved in the body that pose the primary hazard during decompression; oxygen is not generally considered to contribute significantly to the formation and growth of bubbles because continual oxygen consumption in cellular metabolism maintains a low oxygen partial pressure (PO_2) in the body tissues. Displacing inert gas with oxygen during a dive reduces the potential inert gas uptake in the body, and increases the rate of inert gas elimination during decompression by increasing the inert gas gradient between the tissue and blood.

Another way of expressing the advantage of oxygen is in terms of the gradient it can provide for inert gas to be eliminated, without the venous gas tension exceeding the ambient pressure. The space for this gradient, called by Dr. A.R. Behnke the "oxygen window" (1967), exists because cell metabolism continuously removes oxygen from body tissue and arterial blood. The oxygen window is a gas tension difference equivalent to the fall in oxygen pressure from the arterial to the venous blood. In operational practice it can be considered approximately equal to the partial pressure of oxygen in the inspired gas. At normal inspired oxygen pressures, (e.g., sea level air), most of the oxygen supplied to the tissues comes from that which is bound to hemoglobin; hemoglobin can deliver oxygen without a large change in PO₂, so the arterio-venous drop in PO₂ is not great. At the higher oxygen pressures that can exist in hyperbaric environments, the metabolic needs are supplied first by the oxygen dissolved in the blood (rather than on hemoglobin), and the fall in oxygen partial pressure and therefore the size of the oxygen window can be much greater.

2. Oxygen physiology

From the decompression standpoint there are two potentially adverse effects of breathing increased levels of oxygen. These are its physiologic modification of circulation, and its toxic effects. Exposure to increased oxygen pressures reduces cardiac output and heart rate and causes vasoconstriction in at least some vascular beds. The resulting decreased blood flow could slow the removal of dissolved gas from the tissues during decompression. Even so, the net effect of breathing oxygen at levels up to a partial pressure of about 2 atm is clearly beneficial to decompression.

Another physiologic result of exposure to oxygen at elevated levels (at a partial pressure of more than 0.6 atm) for several weeks is a decrease in circulating red blood cells. This is a normal adaptive response and should not be regarded as a result of toxicity, but it can result in a temporary anemia.

3. Oxygen toxicity

In addition to its essential role in energy metabolism, oxygen at high pressure affects basic functions common to all cells; eventually these effects become toxic. A number of them have been proposed as the basis for the toxicity of oxygen. They include enzyme inhibition, membrane damage, increased production of chemically reactive species (such as free radicals), neurotransmitter inhibition, and increased intracellular acidity. No single hypothesis of the mechanism of oxygen toxicity is yet accepted, but free radicals are considered certainly to be involved. It is quite likely that the mechanism differs in various tissues.

a. Types of O_2 toxicity

There are two main manifestations of oxygen poisoning of importance in diving. These are central nervous system (CNS) involvement, which can lead to convulsions, and a more generalized "whole-body" or "somatic" toxicity that has its main manifestation in lung irritation. These effects vary depending on the oxygen pressure and duration of exposure. For example, at pressures above two to three atmospheres absolute CNS effects are of the greatest concern and appear first; CNS toxicity is usually the limiting factor in exposures above about 1.5 atm. The potential for lung damage and whole-body symptoms limits the maximum tolerable oxygen exposure when oxygen partial pressures are in the range below about 1.5 atm. As oxygen pressure increases, less time is required to produce symptoms of oxygen poisoning. In fact, oxygen toxicity follows a classical pharmacological dose-response curve.

b. CNS toxicity

CNS oxygen toxicity results after minutes to tens of minutes of exposure to levels of oxygen above about 1.5 to 1.8 atm (the necessary exposure is a complex matter). The main manifestation is a convulsion similar to a grand mal epileptic seizure. The exposed individual may have lip or facial twitching or ringing of the ears prior to the convulsion, but there often is no warning whatsoever. A convulsion can be extremely dangerous to a diver in the water when it can lead to drowning, and it can lead to tongue biting or other physical trauma in a chamber situation. In the absence of mechanical problems the effects of a convulsion itself are apparently benign and reversible, with a headache and a brief period of amnesia the most obvious sequelae. Experimental subjects encountering oxygen convulsions are often willing to continue the work; they may have no unpleasant memories of the occasion. There is no specific treatment for a convulsion except to prevent injury; it is prudent to remove the source of high oxygen, but the convulsion will generally run its course in a minute or two in any case.

Susceptibility to CNS oxygen poisoning is increased by exercise, dense gas, breathing restrictions, and other factors that increase brain blood flow or the brain level of carbon dioxide; different individuals vary considerably in their susceptibility, and a person's sensitivity may change from time to time.

c. Whole-body and lung toxicity

As long as the oxygen level is kept below that which will produce CNS effects (this is the common practice), pulmonary irritation has been considered the most noticeable effect and one which can act as a monitor of the development of oxygen toxicity. It is normally measured by changes in **vital capacity** of the lungs. The degree of lung irritation is a relevant parameter to use in evaluating the oxygen exposure of a dive schedule or other hyperoxic exposure. Symptoms are chest pain, coughing, and accumulation of fluid in the lungs.

Another category of oxygen toxicity that may develop after about one to several days of exposure is a generalized syndrome of effects we have called somatic or "whole body" oxygen toxicity. This is seen as paresthesias of the skin, tingling, numb fingertips, nausea, headache, effects on vision, and a reduction in aerobic capacity.

Apparently the effects of somatic or "pulmonary" oxygen toxicity are quickly and completely reversible if the duration of exposure is not too long. However, continuing a single exposure or repeated exposures with insufficient recovery intervals can cause more long-lasting effects (requiring weeks for complete recovery). These effects seen from diving should not be confused with a hospital-related "chronic" pulmonary oxygen poisoning that takes weeks to develop and may cause permanent damage.

Recovery from oxygen poisoning begins as soon as non-toxic levels of oxygen are breathed. Only limited quantitative information is available on the rate of recovery from oxygen poisoning and how this is affected by the level and duration of oxygen breathing during the toxic and non-toxic phases. It has been shown experimentally that recovery can take place if the oxygen level is below 0.5 atm.

4. Managing the effects of oxygen toxicity: Oxygen tolerance

In order to live with oxygen's toxicities in diving and still get the maximum benefit from oxygen during decompression (and treatment of decompression sickness, if needed) special tactics are used. These are based on physiological principles, but their quantitative application is determined primarily from experience. They consist mainly of controlling the extent and pattern of the exposure to oxygen. DCAP monitors oxygen exposure in a calculated dive and can print warnings on the table if preset limits are exceeded; the approach is reviewed in Chapter IV and operational details are given in Part Two.

While the extensive use of high oxygen in decompression is recommended, care must be taken not to push this exposure too close to the limit. Since high oxygen breathing is critical to the treatment of decompression sickness, operational decompression schedules should not make use of such high doses of oxygen that proper **treatment** of decompression cannot then be performed. Thus the ability to predict the degree of oxygen poisoning during any exposure sequence is of great practical utility. DCAP limits CNS exposure by incorporating published tables of limits, and uses the accumulation of "**Oxygen Tolerance Units**," **OTU**, for limiting exposures in the whole-body range.

The interruption of high oxygen breathing with periods of normal or non-toxic (0.5 atm or less) oxygen breathing safely extends the duration of tolerable exposure to oxygen, and this **intermittent** breathing pattern is the basis of most procedures used in diving. The most thoroughly studied oxygen breathing patterns and those most commonly used in diving call for 20 or 25-minute periods or "cycles" breathing a high oxygen level separated by 5-minute periods breathing a normal or subtoxic level of oxygen.

While some drugs can delay the onset of symptoms of oxygen poisoning, their efficacy is not well enough accepted to recommend drugs for operational use. Valium (diazepam) is given by some physicians who place a patient on oxygen breathing for extended periods during treatment

E. Environmental and physiological factors in decompression

A number of environmental factors can affect decompression obligation and results. Among these are the type of inert gas, and the inspired inert gas and oxygen levels. Others are the temperature, inspired carbon dioxide, and environmental humidity (in a chamber); these may affect decompression through their influences on local circulation. The effects of these factors are not uniformly detrimental; the results depend on when and where they act. A recent workshop report discusses many of these in great detail (Vann, 1989).

1. Cold

Diving experience has shown in general that when divers are cold they are more susceptible to DCS. The detrimental effect of cold is worse if the diver is cold during decompression than during the gas uptake phase. Some companies use a more conservative decompression if the water is very cold. It is important to note that it is the temperature of the **diver**, not the **water**, that matters. Also, the diver's subjective feeling of cold—cold skin, etc.,—seems to be a better measure of the effect cold will have on his decompression than the diver's core temperature. It is important to note that this physiological phenomenon works in both directions. It has been shown convincingly that divers wearing hot water suits have a higher incidence of DCS than those diving with passive insulation. The diver who is warm while working on the bottom evidently picks up more gas, and this effect is particularly prominent if he lacks the warming during decompression.

2. Immersion

Immersion causes a shift in the pattern of circulation, and gas exchange is increased. As with other physiological factors affecting decompression effectiveness, the time on a dive during which the diver is immersed influences this factor. Presumably the best situation would be for the diver to do his bottom work in a gas environment and decompress immersed, but the opposite is what usually happens.

3. Physical activity

The activity or exercise level during a dive is known to affect the outcome of a decompression. Hard work during the high pressure parts of a dive increases circulation and thus gas delivery to the body. A working diver will take up more inert gas during a bounce dive than a non-working diver. It would seem that exercise during decompression would be valuable to speed the elimination of inert gas from the tissues, and to a very limited degree this is true, but strenuous physical work during decompression increases the incidence of DCS.

Cramped chambers and restricted freedom of movement during decompression are decidedly detrimental.

4. Sleep

Some investigators believe sleep slows gas elimination and account for this in their procedures, but others believe no special precautions need be taken. Likewise, some believe that divers in a chamber should move around frequently during a long decompression, and never allow circulation to be restricted to a limb, but others feel it is better for the divers to sleep comfortably. One factor favoring occasional movement during the night, especially during a stressful non-saturation decompression, is that it permits more frequent checking for symptoms.

F. Diver-related factors

Many individual factors can influence a diver's susceptibility to bends.

1. Age and condition

Age and poor physical condition affect circulatory function. Obesity results in storage of excess inert gas and a slower gas elimination. These factors are detrimental to trouble-free decompressions. There is little doubt that a high level of physical fitness is beneficial. Conversely, a diver who is "not well" seems to have an increased susceptibility.

2. Fluid balance and alcohol

Dehydration is considered to increase susceptibility to DCS. Food and water intake influence fluid volumes and thus the volume of blood circulation. Alcoholic consumption affects both circulation and fluid volume; the effects of alcohol on decompression are complex and controversial. One factor on which there is agreement is that dehydration, which might be induced by excessive alcohol consumption, is probably detrimental. Diet affects fluid balance, but also may change the level of lipid (fat) in the blood. Unfortunately neither the direction nor importance of these latter factors, or the role of dehydration for that matter, is well established experimentally.

3. Individual susceptibility

Individuals differ in their susceptibility to decompression sickness, for reasons that have not yet been explained by any of the factors just mentioned. While it may be difficult to determine why some individuals should be more susceptible to DCS than others, it is a well established fact that this is so. It does not yet seem practical to design diver's decompression tables for the most susceptible elements of the population. Such divers tend to select themselves out or be selected out of diving programs (or others which lead to decompression, such as compressed air work). In cases where a wide range of "unselected" individuals are to be decompressed it may be wise to use more conservative procedures.

4. Recent diving history: The repetitive dive

Previous diving history has definite and paradoxical effects on the incidence of decompression sickness. Later exposures in a sequence of dives tend to produce fewer cases of bends than those dives in which the divers have not been recently exposed to pressure. Field diving experience, however, has also shown that if an exposure is made repeatedly, day-after-day, eventually decompression sickness may result when using tables that are quite safe for the first few dives. More information is needed before this situation can be understood, and the individual diving operation should keep records that can help keep track of these effects. Procedures to make multi-day diving more conservative should be used. A 1991 workshop has addressed these issues (Lang and Vann, 1992).

DCAP Manual. II. Overview of decompression physiology and biophysics Part One. F. Diver-related factors

5. Gender

According to a few reports females tend to be more difficult to decompress, but data are limited on the nature and extent of this difference. One study showed about the same incidence of DCS in male and female groups of comparable age and fitness, but the females had more serious DCS that was harder to treat. Fetuses are probably more resistant than their mothers, but the consequences may lead to birth defects. Current recommendations are that females who are pregnant or who could be pregnant should not dive.

III. OVERVIEW OF DIVING OPERATIONS: DECOMPRESSION ASPECTS

This section covers the various types of decompression in common diving practice. Some of the equipment mentioned here is explained in more detail in the reference by Haux (1982).

A. Short duration or "bounce" diving

Various types of dives made from the surface followed by immediate ascent to the surface have similar decompression considerations. Whether a dive uses scuba equipment or surface-supplied breathing gas, or is made with a diving system (bell and deck chamber), from a physiological viewpoint the decompres sion obligation depends primarily on the bottom depth (pressure), duration of the dive, and the gases breathed. The longer the decompression required for an exposure, the less convenient it is to conduct that decompression in the water with a self-contained gas supply. Thus the use of surface gas supplies and diving bell and deck chamber systems increases the practicality and safety of a dive when lengthy decom pression obligations are encountered. Inwater decompression stations, sometimes called "habitats," effectively afford protection from cold and other risks and inconveniences of decompressing in the water.

1. Surface-supplied dives

Both air and mixed gas (that is, helium-oxygen or heliox) diving may be carried out from the surface. The diver is supplied with pre-mixed gas by means of a hose to the surface, and may wear a wide variety of gear, including the traditional free flowing "hard-hat," or perhaps "demand" breathing apparatus. Pressurization results from the diver's descent into the water. He may swim, be hoisted up and down on a "stage," or may use an "open" bell which stays at ambient pressure but which affords some protection and may have a source of breathing gas. Usually only one diver is in the water; the "tender" is at the surface, as well as a standby diver. In a mixed-gas dive the divers may breathe air during most of the descent, or only to a certain point where the switch is made to heliox. This is necessary because a proper heliox mix may contain a reduced oxygen fraction, and may not provide normal PO₂ when breathed at the surface; if the gas is, say, 10% oxygen and the shift is made at 12 msw or 2.2 bars, the diver then gets an adequate PO₂ of 10% of this pressure or 0.22 bars.

2. Surface decompression

One method which has been devised to minimize or eliminate decompression stops in the water is called "surface decompression" or "sur-d." In surface decompression, the diver, perhaps after making some stops in the water, ascends directly to the surface before he has completed an adequate decompression for his dive. He then gets into a deck decompression chamber (DDC), is recompressed to the pressure equivalent of a depth a little deeper than the one from which he began his final ascent; usually ascent is from 9 msw (30 fsw) and recompression is to 12 msw (40 fsw), and from that point he completes his decompression in the chamber. The equivalent term in caisson work is "decanting." Surface decompression has the inherent danger that the risk of DCS might be increased as a result from too slow a transfer

DCAP Manual. III. Overview of diving operations: Decompression aspects Part One. A. Short duration or "bounce" diving

to the chamber or failure to recompress the diver, but it offers a sensible alternative to extended decom pressions in the water

where a bell is not used. It permits the diver to get out of the cold water and avoid its hazards, and allows other work to progress which cannot be carried out with the diver in the water. When combined with oxygen breathing in the chamber ("sur-d/ O_2 "), this method usually results in successful and efficient decompressions. It has been subjected to criticism in the North Sea, due in part to inadequacies in the tables used for that situation, and because only the more stressful decompressions are done with sur-d.

3. Diving bells and deep diving systems

The safest method for deep diving is to use a **diving bell** (or "submersible decompression chamber," SDC) which connects to a deck chamber, a "deep diving system." This increases the accuracy with which a decompression schedule can be followed, increases the options for breath-ing gas to use in decompression, protects the diver from the more hostile environment of the water, permits other work to commence or the vessel to move, and allows for immediate and adequate treatment of DCS should it occur. A shallow-water version of this system is being introduced into the North Sea to get around the problems of surface decompression; this is called "transfer under pressure," or TUP (Hamilton, 1984).

4. No-decompression or no-stop dives

Another consideration of bounce diving is the "no-decompression" range. For all depths at which air can be used as the breathing gas and for the shallower depths at which helium-oxygen mixtures might be used, there is a maximum bottom time at each depth for which no decom-pression stops are necessary. This time becomes shorter as the depth becomes greater. A dive made within this envelope is called a "no-decompression" or more properly a "no-stop" dive. After such an exposure the diver may return directly to the surface at a specified, continuous rate. While such dives are uncomplicated in themselves, they still must be considered in repeti-tive dive sequences and in flying after diving.

The term "no-decompression" is well entrenched, but it is preferable to call such a dive a "no-stop" dive, to avoid the implication that there is such a thing as a dive with no decompression obligation. There is always some obligation, but it may be trivial in some cases.

5. Cave and "technical" diving

Cave diving is a sport wherein divers explore water-filled caves. It is in a sense a competitive sport, and the participants strive for "firsts," whether it be the first to be in a particular cave, or the first to accomplish dive of unprecedented depth and/or distance. They are generally highly trained and have a good understanding of diving physiology and operations, with particular emphasis on equipment used and management of gas supplies. Cave diving is regarded as being extremely dangerous when performed by divers lacking the special training, but properly trained and equipped cave divers have a good record.

Other divers with primarily recreational, scientific, or "entrepreneurial" motivations (as opposed to strictly commercial or military diving) may use similar technology-based approaches to expand their operational capabilities. These may include special breathing mixtures, perhaps with helium to combat narcosis, but

nearly always with optimal oxygen profiles. These divers may use diver propulsion vehicles ("scooters"), dry suits with argon as the insulating gas, and even recirculating breathing equipment ("rebreathers").

Some of the more sophisticated cave divers use trimix (oxygen, nitrogen, and helium) or heliox to reduce narcosis. For long dives they may have to "stash" or "stage" tanks along the way; these are put in place before the main dive on preliminary setup dives. Often the decompressions are with a different mix having optimal oxygen, and these tanks are usually staged in the appropriate places. A creative cave dive will require meticulous planning and preparation, and will often require a custom decompression table that follows the profile of the cave.

B. Repetitive diving

Decompression from a dive performed shortly after an earlier dive is affected by the earlier dive. In such cases some adjustment is made in the decompression—usually of the second dive or of the interval between them.

The U.S. Navy considers any dive made within a 12-hour period after a previous dive to be a repetitive dive. For others such as the DCIEM tables the repetitive dive interval may be 18 or more hours. Procedures for repetitive diving have been computed by the same models and tested by the same methods used for other decompression schedules, and because they are based on experience as well, they usually work (Lang and Vann, 1992).

The theoretical situation in repetitive diving is by no means clear. On the one hand, it seems likely that gas loadings build up over time. Divers working for several successive days on tables proven to be effective when used singly may occasionally develop DCS on the 5th or 6th day of this "multi-day diving" regimen.

On the other hand, the behavior of bubbles and/or gas nuclei is not so clear. The first dive may clean out bubble nuclei, or it may cause them to grow or proliferate. The former may explain why tunnel workers experience most of their DCS after being off work for a few days or when starting to work at a new pressure, and why divers feel a "workup" dive makes them less susceptible.

Some evidence, mentioned in the report on NOAA's Repex project (Hamilton, Kenyon, Peterson et al, 1988), suggests that if anything the effect of an earlier dive on bubbles may be beneficial, removing more nuclei than it creates. If this is the case then the repetitive dive may be calculated on the basis of gas loading. This is the method most often used. Another twist on this is that one would expect that the "worst case" repetitive dive would be one that is the same as the dive just done, since that would tend to leave the highest gas loadings in the same compartments.

One point that comes through all this is that development of repetitive dive procedures calls for a judicious application of practical experience to whatever computations are done. As an example, an established rule for recreational divers is to make the deeper dive first; the evidence suggests—for reasons not yet clear—that doing a deep dive after a shallow one may prejudice a clean decompression. DCAP users should be alert to the fact that DCAP allows such dives to be calculated without prejudice and should apply experience to such computations.

The role of dive computers in repetitive diving has been reviewed in a UHMS Workshop (Hamilton, 1995).

C. Saturation diving

If an extended diving time is required to complete a job, saturation rather than surface bounce diving is the most efficient method, from the standpoint of decompression time. Once a diver has been in a hyperbaric condition long enough so that no appreciable additional inert gas is being taken up by his body, no further decompression obligation will be incurred no matter how extended the exposure. Saturation diving takes advantage of this by keeping a diver pressurized to the depth of a job so that he may work for long periods without decompression, and he then will have only a single decompression at the end of the dive. Saturation diving is carried out with a diving (or chamber) system or out of an underwater habitat.

Decompression efficiency is not the only reason for using saturation. In current commercial practice saturation methods are used by many companies for dives to the 150 msw range and deeper because they do not possess satisfactory procedures for decompressing from short-duration ("bounce") dives. An additional incentive, not a trivial one, is that the earnings of both the divers and the contractor from a saturation dive may be much greater, making saturation the preferred method if the client is willing to pay.

Saturation decompression from heliox takes roughly one day of decompression per 30 metres of depth, and decompressions are relatively trouble free. Some additional risk is involved, how-ever, by the fact that saturated divers cannot leave the pressure chamber in the event of fire or shipwreck, and they are at very high risk in the unlikely event of a pressure loss.

Decompression from saturation may be by linear ascent ("slow bleed") or by small (e.g., 1/2 msw) stages that approximate a linear ascent. Rates in the range of about 45 min/msw are used for heliox, and up to 180 min/msw for decompression from nitrox saturation. The oxygen partial pressure is (normally) maintained at 0.4 to 0.5 bars. Sometimes there is a shift to air and/or oxygen breathing near the end. Decompression may be stopped during sleep periods, but the necessity for this is not universally accepted.

D. Saturation-excursion diving

Saturation-excursion diving involves "bounce-type" dives by a saturation diver, whereby the diver is saturated at a "storage" depth and excurses to a deeper depth for work. This method is useful in cases where work periods are brief or unscheduled and there is no need to saturate the entire crew at the working depth. It thus can reduce the final decompression time substantially, and allows the divers to be stored at a shallower and hence safer depth. This is also a primary dive mode for work from a subsea habitat.

Decompression results from the excursions in heliox saturation-excursion diving have been good. Studies by the British and United States Navies have proven that the decompression obligation for a given excursion is reduced with increasing depth, at least to the 300-metre range. For example, a diver saturated at 250 msw has unlimited excursion privileges for 50 msw deeper and 44 msw shallower with no required decompression stops (U.S. Department of the Navy, 1993); these values are reduced due to field experience

from earlier values tested in the laboratory. For a given excursion the decompression obligation is less than that required for a similar dive from the surface. Practices for the exploitation of this phenomenon for unlimited-duration excursions in direct-decompression helium-oxygen diving have been developed by the U.S. Navy Experimental Diving Unit.

Less experience is available for time-limited heliox excursions (those requiring stops or having a limited excursion period), but some successful excursions have been done in chambers.

The situation with excursions from nitrox saturation is more highly developed but there is considerably less field experience. The second edition of the NOAA Diving Manual (Miller, 1979) includes procedures which have recently been extended to include repetitive excursions and excursions with stops in the Repex procedures (Hamilton, Kenyon, Peterson, 1988). These excursions have been uniformly successful, including a series which tested the repetitive excursion algorithm (Hamilton, 1990), and more recently, excursions involving helium (Hamilton, Shi, et al, 1996).

E. Use of rebreathers

Although they actually predated open-circuit scuba, closed-circuit, mixed gas, rebreathers have been developed in the last 20 years to eminently workable forms, with the primary development and experience belonging to the U.S. Navy. A significant series of projects has been carried out by the Naval Experimental Diving Unit to develop algorithms for calculating decompression tables for use with rebreathers. These projects led to the new and highly successful Exponential-Linear computational model (Thalmann, 1983).

However, the Navy dives its rebreathers at a constant PO_2 of 0.7 atm. This is a safe value for most Navy missions, but is relatively inefficient from the decompression point of view. Work with levels in the range of 1.4 atm has established that this is a nearly optimal oxygen for decompression efficiency and yet is below the level of CNS toxicity; this has been exploited by entrepreneurial operations (Hamilton, Kenyon, and Clough, 1988).

F. Altitude and hypobaric decompression

Most diving work is oriented to sea level pressure as the starting and ending point. Aircraft, spacecraft, mountains, and hypobaric chambers all may create pressures less than one atmosphere. These are considered in the following sections, and many aspects have been reviewed in a recent workshop (Pilmanis, 1992).

Although altitude is part of the same pressure continuum as hyperbaric pressures the units are different and somewhat more difficult to handle. For one thing, the atmosphere is slightly nonlinear because gas is compressible; a given change of altitude at high altitude causes a smaller pressure change than the same differential at lower altitude. The atmosphere has been well studied and its pressure can be easily fit with an equation. International air traffic is controlled in thousands of feet, with occasional translation to metres. Barometric pressure may be measured in inches or millimeters of mercury, and occasionally in millibars. NASA insists on measuring its spacecraft pressures in pounds per square inch, psi.

1. Diving at high altitude

Ascents from pressures higher than sea level (i.e., excursions from saturation) have been shown to be less demanding than return to sea level, and the converse situation, decompression to altitude, as might be expected is more demanding. The reduced ambient pressures of altitude conditions must be considered when a dive is made in a mountain lake.

Decompression when diving at altitude requires that the diver return to a barometric pressure less than normal, but at the same time a given dive will have caused him to take up a nearly normal amount of additional inert gas. Several algorithms for dealing with this problem have been proposed; the most practical approaches seems to be those of Prof. A.A. Bühlmann in Zürich (Bühlmann, 1984; Vann, 1989), which are essentially ascents based on gas loading with constraints adjusted to compensate for the reduced pressure.

One important aspect of calculating tables for diving at altitude is the equilibration of the diver to altitude; if the diver is equilibrated then his body holds less gas at the beginning of the dive. Tables have to be adjusted for the state of equilibration.

As with other aspects of decompression, practical experience is an essential requirement to the development of safe procedures.

2. Flying after diving

Flying after diving exposes the diver to a further reduction of ambient pressure which may cause bends if a significant residual of inert gas or decompression bubbles accumulated during the dive remains in his body at the time of the flight. Consequently, definite surface intervals must separate diving and flying. Recommendations for this delay range from arbitrary periods of 2 to 24 hours, or may use intervals computed on the basis of the dive profile and gases used. Sometimes effects of flying after diving are seen well beyond what can be explained by gas loading theory, indicating that the effects are probably due to stable, residual bubbles. Well over 24 hours may be required following saturation or treated decompression sickness. The UK Diving Medical Advisory Committee has issued interim flying-after-diving procedures recommended for commercial diving (Hollobone, 1982). A more recent workshop on flying after diving offers more recommendations; these are primarily oriented toward recreational divers (Sheffield, 1989).

DCAP allows calculation of flying after diving by setting a limit in a special matrix of ascent limits and decompressing to altitude.

3. Aviation and space: Ascent to altitude or exposure to reduced pressure

Another altitude-related decompression is encountered by aviators and others exposed in a short time to reduced pressure, whether or not they have been exposed to hyperbaric pressure beforehand. This does not usually apply to mountain climbers because the time required for a climb provides, in effect, a decompression. Astronauts on extravehicular activity in a space suit are affected (Pilmanis, 1992).

For a person exposed to an ambient or aircraft "cabin" pressure of less than about 50 kPa (about 1/2 atmosphere) the possibility of DCS exists. This risk is affected by many factors, including particularly the exposure pressure, duration of exposure, prior exposure of the individual, and degree of exercise.

Aviation-type exposures can get into the DCS-provoking range in unpressurized aircraft, or in aircraft that fly so high that even with a differential cabin pressure the resulting pressure is too low. Most exposures to these pressures are for military reasons and in military aircraft. Throughout most of the altitude decompression range the pressure is so low that oxygen has to be breathed, either at ambient pressure or in some cases with pressure breathing. Civilian passenger aircraft fly high enough, but because of the lack of oxygen breathing equipment, if there is a cabin pressure loss it is necessary to reduce the altitude below the DCS range quickly so there is minimal chance for DCS.

For the astronaut the planned low pressure exposure is during extra vehicular activity (EVA). The differential pressure of the NASA space suits is 30 kPa (4.3 psi). This pressure is low enough to produce decompression sickness in most individuals within the time span of a normal EVA mission. This decompression sickness is reduced to an acceptable level by means of a partial step decompression and oxygen "prebreathing" prior to EVA.

The countermeasure most frequently used by both aviators and astronauts is oxygen prebreathing or denitrogenation. This is the breathing of 100% oxygen (usually by mask) to reduce the gas loading of inert gas (usually nitrogen) in the person's body prior to reduction of pressure. High altitude pilots and astronauts both use this technique. Decompression patterns similar to those used for diving are physiologically possible but are usually operationally impractical. However, astronauts planning to make EVA from the Space Shuttle are decompressed overnight at about 70 kPa, and prebreathe oxygen for about one hour before exiting the spacecraft. With this "stage decompression" about one hour of prebreathing is needed, without it about four hours. Exercise during prebreathing may shorten this time.

The amount of prebreathing necessary for a given exposure (pressure, prior exposure, duration, and activity) is worked out empirically. NASA scientists have developed an algorithm for predicting prebreathing. It is based on the inert gas pressure ratio in the 360 min Haldanian tissue compartment, which gave the best match to experience. This has led to the development of satisfactory procedures for EVA activities (Horrigan, et al, 1989; Conkin, et al, 1990). Given a proper analysis of previous low pressure exposure experience, DCAP should be quite effective for altitude decompression.

G. Other decompression requirements

Although diving is the main field requiring decompression and is the archetype for this manual, it is by no means the only endeavor which can result in the need for decompression. Among these other ventures are tunnel and caisson work, and certain medical treatments. These are briefly mentioned in this chapter and compared with diving.

1. Compressed air work: Tunnels and caissons

One of the oldest reasons for exposing people to high pressure is the us of compressed air to prevent the intrusion of water into construction sites below the water table. These are most frequently caissons which are sunk straight down, or tunnels which go under a river or harbor. In cases where the soil is too loose or leaky to hold out the surrounding water, the use of air pressure can make it possible to maintain a "dry"

work environment. Workers enter and leave the pressurized space through a "lock," which performs the same function (and was the predecessor for) the lock on a deck decompression chamber or diving system. The normal pattern is for workers to enter the lock at the end of the work period, then for pressure to be reduced gradually until they reach surfaced pressure. Depressurization or decompression may be in a gradual linear bleed or in stages. For several reasons the decompression procedures are usually mandated by law, although in Japan the caisson master performs the decompression according to his own (empirical) experience.

Sometimes the workers are reduced directly to surface pressure and then reenter a lock or chamber, where they are recompressed and subsequently decompressed according to a protocol or table. This is the corollary of surface decompression in diving. In compressed air work it is called "decanting."

Compressed air work exposures are usually quite long compared to those of diving. A limit seems to be to complete the work and decompression within a single shift, usually 8 hr. This is often required because the next shift has to go to work using the same equipment. The pressure or depth usually ranges from a metre or two to perhaps 10 msw, but work has been done to the 40 msw range. Contractors will try all sorts of tricks such as pumping the water table down or making the soil impervious to water with "grout" in order to avoid the complications of working in compressed air. Better decompression procedures might open up more options.

Whatever differences may be cited between compressed air work and diving, the basic physiology and biophysics of decompression are the same. Compressed air workers are often decompressed on schedules that would be unacceptably fast for divers. Explanations for why this is tolerated call on some sort of acclimation or accommodation (also called "adaptation," but this is not the best use of that term) from the frequent exposures. There seems to be a difference in tolerance, such that air workers are in fact not adequately decompressed and may suffer from high levels of dysbaric osteonecrosis (bone necrosis) and probably also from frequent untreated pain-only DCS.

Recently gas mixtures containing helium have been used for caisson work at higher pressures (Takashima et al, 1996).

2. Submarine escape and rescue

Two rather different decompression situations are encountered in escape or rescue from military submarines. One is free ascent, where the brief exposure to pressure during ascent from a distressed submarine can be enough to make decompression a consideration, the other is rescue and recovery of the crew by means of a small rescue submersible such as the U.S. Navy's DSRV or the Swedish Navy's URF. In the latter case transfer is normally at atmospheric pressure unless the distressed submarine happens to be under pressure, in which case the saturated or nearly saturated crew will have to be decompressed. Pressure in the submarine can also complicate the decompression aspects of free ascent.

a. Free ascent

Free ascent has been developed to a high degree of sophistication by the Royal British Navy. Escape "towers" on their subs allow crew members to lock out one at a time. The submariner enters the escape compartment, pressurizes it with water and compressed air, then opens the hatch and ascends to the surface. He wears a protective suit with a hood allowing breathing (air) on the way to the surface. Time at

maximum pressure is 5 sec. Ascent is limited by the terminal velocity of the person and his gear, usually at about 2.5 to 3 msw/sec. Compression is roughly a doubling of pressure every 4 seconds. This set of factors combines to allow escapes with low enough incidence of DCS for it to be feasible to conduct training to depths of 150 msw. Escape from deeper depths is possible, but will result in casualties. As mentioned, when the submarine is under even a little extra pressure the DCS-free envelope shrinks considerably.

Another complication is for submarines such as those of the German Navy, which do not have escape towers. These have to use "compartment" escape, whereby a group of several crew pressurize together in a compartment, flood it, then each person goes out in turn. This forces a bottom time of a couple of minutes, which limits the maximum depth from which an escape can be made.

We have "modelled" the free ascent pattern with DCAP, using criteria from training and laboratory exposures of humans as well as animal data to establish a suitable matrix of ascent limiting values. This allowed the comparison of various options, such as oxygen breathing, slowing ascent at the end, and the effects of initial pressure (Hamilton, 1990).

b. Pressurized rescue

A crew exposed to a pressurized submarine atmosphere faces a problem when rescued by the USN DSRV. The submarine atmosphere would be a nitrogen-oxygen mixture, but the DSRV can only pressurize with heliox. This poses a counterdiffusion problem on the crew, to switch from saturation with nitrox to immersion in and breathing heliox. Various staging and gas mixture manipulations are being tried to help solve this problem. It can be modelled with DCAP.

3. Medical treatment: HBO₂

A growing field in modern medicine is hyperbaric oxygen therapy. This is the administration of oxygen to a patient under pressure. The main disease entity for which hyperbaric oxygen is the primary treatment of choice is gas lesion disease, or the familiar set of decompression sickness, gas embolism, and the less frequently encountered counterdiffusion sickness. Treatment of these conditions is covered in the last section of this chapter, III.I. Other diseases for which HBO₂ is effective adjunctive therapy are gas gangrene, carbon monoxide poisoning, soft tissue necrosis, problem wounds, thermal burns, and a number of others.

Treatments are most frequently carried out at low pressures, in the range of 1.5 to 2.8 atmospheres absolute, and usually involve the patient breathing oxygen for most or all of the time under pressure. As a result the HBO₂ patient usually does not encounter much in the way of a decompression obligation. Attendants, however, who breathe air during the treatment might require decompression. One area where DCAP is of great value in practice is for managing extended and unusual treatments. Normal ascent constraints may not be valid for a person with a decompression injury but the ability to track the gas loading and oxygen exposure can be extremely helpful in managing a difficult treatment.

4. Other activities requiring decompression

There are a few other situations which do not fit any of the previously described categories that require decompression. One of these is testing a containment vessel for a nuclear reactor, whether a commercial

power plant or a nuclear submarine. The containment vessel has to operate under pressure on the order of one or two extra atmospheres and involves a crew being exposed to such pressures for several hours. Decompression procedures are relatively simple for this situation, but contingency planning is also often necessary.

H. Dive computers

A recent development based on modern electronics is the diver-carried decompression computer, most widely used in the recreational diving community. These devices monitor the exposure of the diver (actually the environment of the computer) and maintain a running calculation of the decompression obligation. The diver is provided with the maximum no-stop time, and if necessary, with the decompression stops needed to ascend to the surface with a low risk of DCS. There are a number of dive computers on the market; these all seem to provide adequate decompression, but may not limit dive patterns as much as they should (Hardy and Schuster, 1993). A workshop dedicated to dive computers offers guidelines on how best to use them (Lang and Hamilton, 1989).

DCAP can be used to mimic some of the behavior of dive computers, but the orientation is different. Dive computers monitor a varied profile and constantly provide a "safe ascent depth" or more properly a "ceiling" on a real time basis during the course of a dive; the diver can follow the ceiling to the surface. DCAP can deal with a varied pressure exposure, but normally provides a predictive profile with stops or linear ascent, not on a real time basis. (However, a version of DCAP is used in hyperbaric chamber operations for monitoring exposures and logging profiles.)

I. Treatment for DCS and embolism

It is quite clear from the present knowledge of decompression phenomena that it is never going to be completely safe to conduct diving or pressure chamber operations (to pressures of about 0.7 bars or greater) without some provision for treatment of decompression sickness (DCS) and/or embolism. No matter what the degree of conservatism in a decompression table there is always the possibility that DCS or embolism will occur, even if only as a result of a medical anomaly in the individual or if appropriate procedures are not followed correctly.

Accordingly, along with other provisions for decompression, it is essential that the capability to "treat" always be available. The main factors needed in treatment are the ability to recompress the affected diver using the proper gases, and to be able to supply him with oxygen or mixtures rich in oxygen to breathe. Also needed are intravenous fluids and drugs for use in the more difficult treatments. Perhaps the most important item to have on hand is the knowledge of what to do and the ability to make the decisions—particularly the early ones—promptly. A preexisting arrangement with a diving medical doctor is highly recommended. If no doctor is immediately available the DAN network, (919)684-8111, can be contacted, or the U.S. Navy Experimental Diving Unit, (504)234-4351. If there is a delay before treatment the patient should be kept well hydrated and given 100% oxygen to breathe by a demand mask or mouthpiece.

Still another function will need to be performed in any treatment. Once the diver has been recompressed, etc., it will be necessary to return him to surface pressure. This is usually done with more conservative

profiles than the diving tables used. Many treatment procedures for symptoms developing at or near the surface also include the final decompression as part of the procedure. For deeper treatments a saturation decompression is usually used.

DCAP can be used to provide a decompression profile following treatment by programming the diver's exposure history and completing the decompression using a profile more conservative than the original dive. A supplementary document, *Chamber treatment of decompression sickness and gas embolism*, rovides detailed information on both DCS and the use of DCAP in its treatment.

IV. DCAP CALCULATIONS

This chapter covers some of the more important computations performed by DCAP. The equations solved are given here; how to use them is covered in Part Two. The chapter defines the units generally used with DCAP and the calculations for oxygen exposure, and describes the computational models used with DCAP. The latter are referred to in this manual as "models," but they are not true models as that word is normally used. A model is used to emulate, with mathematics and other tools, the behavior of a system, usually with the objective of better understanding and predicting its behavior.

The DCAP "models" are used to carry out an operation, and thus they could more properly be called computational methods or algorithms. The term "model" has been used throughout the development of DCAP and is widely used in this context by others; we have chosen to continue using it.

In earlier DCAP materials the word "model" was used to refer to a set of half times. Thus a Haldane calculation would be defined by a mod have elected here to refer to any specific subset of the Haldane (or Tonawanda II) model as a "half-time set," and reserve the word "model" theoretical approaches.

Before discussing computations and models it is appropriate to describe the philosophy of units used by DCAP, and to review oxygen exposure calculations; these apply to all models used with DCAP. A few of the DCAP terms used here are defined in more detail in later chapters.

A. Pressure units

DCAP allows computations to be done in virtually any pressure unit. Those units commonly used for diving and in DCAP examples are defined here. Typically the ordinary depth units used in diving are either metres of sea water (msw), the "metric" or SI unit, or feet of sea water (fsw), the English or Imperial unit. Note that these are actually units of **pressure**, but they look for all the world like units of **length**; this is a source of great confusion and misunderstanding, and the conversion between these units is often done incorrectly. DCAP allows units to be defined in essentially any way the user wants, but because of this flexibility it is pertinent to define the two most common usages, the ones used in the examples in this manual.

The units msw and fsw are defined independently and arbitrarily:

1 msw I 1/10 bar = 10 kPa = 10⁴ pascals (Nm⁻²)

This conforms to a specific gravity for sea water of 1.027.

1 fsw I 1/33 standard atmosphere = $3.0705 \text{ kPa} = 3.0705 \text{ x} 10^3 \text{ pascals} (\text{Nm}^{-2})$

This conforms to a specific gravity for sea water of 1.020.

Sea water ranges in specific gravity between about 1.020 and 1.030. Both depth units were chosen to be convenient yet stay within these limits. The density of the water (or the depth for that matter) does not matter if the **pressure** of the diver is measured and used to determine the proper decompression. Thus a dive in fresh water involves no adjustment as long as the diver's **pressure** is measured with the same gauge calibration as is used for decompression. An adjustment for fresh water would of course be required if the depth were measured as a linear distance, using for example a shot line or the survey of a dam to determine the depth.

Incidently, the conversion values between fsw and msw, as defined here, are **3.2568 fsw/msw** and **0.30705 msw/fsw**. The conversion values between feet and meters are 3.2808 feet/meter and 0.3048 meters/foot, as linear units. Use of these latter "linear" factors to convert the pressure units causes a discrepancy of about 1.3%. As mentioned earlier, it is quite common for this conversion to be made incorrectly. When it is essential to ensure correct communication the pressure should be referenced to SI units (pascals) or atmospheres.

The definition of a foot of sea water as exactly 1/33 atm is the most widely used and the most convenient. The most significant deviation from this is that of the U.S. Navy, which uses the definition of 1 fsw = 1/33.08 atm; this has also been picked up by the leading journal covering diving physiology, *Undersea and Hyperbaric Medicine*.

Another technicality related to units needs to be mentioned. The unit "msw" is based on the bar (1000 mb), and users of msw units usually consider atmospheric pressure at sea level to be 1 bar or 1000 mb. The Standard Atmosphere, however, is 1013.25 mb or 1.01325 bar. This means that the two systems have "bases" that differ by 13.25 mb or 1.3%. It would be possible to relate msw to the standard atmosphere (and DCAP makes provision to do this), but the convenience achieved by maintaining the familiar European usage of 10 msw=1 bar far outweighs the trivial difference. Also, daily barometric pressure may fluctuate more than this. This difference becomes even more insignificant when all calculations are made against the same base and units are kept consistent.

It should be noted that DCAP can be configured to handle dives in any of these units or conversion factors, or in mmHg or psi or what have you. DCAP's use of units is covered in more detail in Part Two.

B. Oxygen exposure calculations

DCAP provides methods for monitoring both CNS and whole-body (or pulmonary) oxygen toxicity on dives and dive analyses computed by DCAP. This section defines the calculations made by DCAP; how to use them is covered in part in the section on oxygen breathing and explicitly in the section on oxygen tolerance, both in Chapter VIII in Part Two. References to DCAP's methods for managing oxygen tolerance are Kenyon and Hamilton, 1989, Hamilton, 1989, Bohrer and Hamilton, 1993.

1. CNS toxicity

Limits on oxygen exposure to avoid central nervous system (CNS) oxygen toxicity are generally expressed as a set of **time limits**, each an exposure duration limit for a given PO_2 . These are empirical, but because

relevant data is scarce and human variability is great, they rarely provide a method of dealing rigorously with multiple exposures to different partial exposures. Even so, this is one of few available methods, and DCAP's method is based on it. DCAP assumes that an exposure of the full "allowed" time for a given PO_2 is equal to unity (e.g., 1.0), and that this may be made up of a series of fractional exposures. The fractional exposures at each partial pressure (fraction of the total allowed time at that PO_2) are summed, and when the sum reaches 1.0 a warning is printed on the table. The flexibility in this system resides in DCAP's ability to use different empirically determined exposure limits. This principle is discussed in the paper by Kenyon and Hamilton (1989) and its use is covered in Part Two, Chapter VIII.

Although it is useful in its simplicity, we acknowledge that this method has significant weaknesses; we are watching for the development of a suitable operational algorithm for tracking CNS O_2 toxicity. A major difficulty preventing this is the wide variability in sensitivity to oxygen of different individuals and especially of the same individual at different times (Donald, 1992). Other approaches include the "superposition" concept advocated by Brian Hills (1976) and recently investigated by Fairbairn and Morrison (1990), which reduces the impact of each element of exposure as it recedes into the past; more modern are empirically-based statistical approaches by Vann (1988) and Harabin (Harabin et al, 1995).

A precautionary note to the user is a reminder that CNS oxygen toxicity is somewhat like decompression sickness, in that it is highly variable and may not occur just because limits are exceeded, but staying within the limits by no means guarantees that an individual will not be affected. This is particularly important since a convulsion may be dangerous to a diver in the water

2. Whole-body or pulmonary toxicity

a. The Pennsylvania method: UPTD and CPTD

A method was worked out some years ago by researchers at the University of Pennsylvania to quantify oxygen exposure in order to compare the effects of different exposures on the lung. The method, referred to both by its aggregate dose CPTD (Cumulative Pulmonary Toxicity Dose) or its individual units UPTD (Unit Pulmonary Toxicity Dose), keeps track of the dose of oxygen exposure of the individual's lungs (Bardic and Lambertsen, 1970; Clark and Lambertsen, 1971; Wright, 1972).

The basic "unit" is defined as the toxic effect of an exposure of 1 minute at an oxygen partial pressure of 1 atmosphere (or bar). Using changes in vital capacity of the lungs as an index, the Pennsylvania workers developed an empirical formula by a "curve fit" to the data to account for the observation that at doses higher than 1 bar the rate appears greater than proportional, and that below 1 bar it seems to taper down to a threshold such that exposures below 0.5 bars do not contribute to the toxic effects. That is, at exposures greater than 1 atm the effects on vital capacity are greater than 1 unit/min, and that at levels below 1 atm the effects are less than 1 unit/min. These units, while defined mathematically in precise terms, are no more than a good estimate of empirical observations that vary widely between individuals.

A formula was worked out by the Pennsylvania group using curve fitting to take these factors into account in determining Oxygen Tolerance Units:

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$$\mathbf{OTU} = \mathbf{CPTD} = t \left(\frac{\mathbf{PO}_2 - 0.5}{0.5} \right)^{0.63}$$

where t = dtration in minutes of exposent to a particular PO₂ in atmospheres, and 0.5 is the "threshold" below which toxicity is minimal.

This Oxygen Tolerance Unit is called OTU in DCAP tenninology. DCAP can calculate OTUs in the process of calculating each dive, and can display the value at each stop, in comments, and/or at the end of each dive. Procedmes and lookup tables for calculating CPTD and thus OTU "by hand" are given in the *Undenvater Handbook* (Shilling et al., 1976) as well as in the to repolts cited at the beginning of this section.

The concept of oxygen exposme limits is an extremely important one in diving physiology. As originally proposed **it** suffered from some limitations. First, the only dose limit proposed for use with the method is 615 units, which causes a decrease of 4% in vital capacity and which by implication is an acceptable oneday limit. The method does not take multiday exposmes into account, nor does it allow a calculation of recovely by the hmgs when the diver is not breathing a high level of oxygen. Also, it is based on lun g toxicity only— ach ally only vital capacity, since this can be measmed relatively easily— yet other effects of long dtrration, low level hyperoxic exposmes may be just a.s prominent (Sterk, 1986).

We feel there is justification to change away from the terms UPTD and CPTD to OTU, Oxygen Tolerance Units, in palt because of confusion between the telms CPTD and UPTD (the unit is the same but CPTD is the cumulative total), but mainly because the effects of long-dmation oxygen exposme are not limited to the hmg. Another minor point is that "tolerance" seems to offer a better outlook than "toxicity."

b. The Repex approach

A new method is now available for managing long dtration oxygen exposme, ba.sed on a method derived for the NOAA Repex project. The Repex algorithm shows that on a daily basis the total tolerable exposme limit (in OTU's) is a function of the **duration**, in days, of the **total period** of exposure to elevated oxygen. Thus a single daily dose which is preceded and followed by days with no exposure to hyperoxia can be much higher than a daily dose where the individtial ha.s been exposed to high oxygen for several successive days. The multiday approach thus indirectly takes recovery into account. The proposed method and some provisionallimits for using it are given in a pair of NOAA publications on the "Repex" ha.bitat diving tables (Harnilton, Kenyon, Peterson, 1988; Hamilton, Kenyon, Peterson et al, 1988). A "cookbook" of the Repex oxygen tolerance procedmes has also been published (Hamilton, 1989). Some recommended limits and how to use them with DCAP are given in Chapter VIII.

This approach provides a concephial method of dealing with recovery. The Repex method described and referenced above shows a higher level of tolerance in a "fresh" diver, but that after about 10 days the predicted tolerance (number of OTUs tolerated per day) levels out at a more or less linear rate. That rate, the slope of the "units per day" line, can be regarded as the steady state rate of recovery, and for a person in a steady state exposme over several weeks the tolerance can be predicted to the extent that these obselvations are valid.

c. The Harabin approach

Recent statistical work by Andrea Rarabin and colleagues at the U.S. Naval Medical Research Institute with a larger set of high oxygen and vital capacity data shows that a more linear (not exponential) relationship matches the data better (Rarabin et al, 1987; Rarabin and Smvanshi, 1990). Although there are huge individual differences in oxygen sensitivity and some pitfalls in its determination, vital capacity was used because it is something that can be measured reliably and there was alarge data base. Rarabin worked out a more direct method of predicting the average reduction in vital capacity as a result of a specific hyperoxic exposure. This equation is also solved by DCAP, and an estimate of the predicted percentage of reduction in vital capacity can be printed on a table or comment. The Rarabin equation for reduction of vital capacity is:

% VC drop = 0.011(PO, - 0.5)t

where PO_2 is the partial pressure in atmospheres of the oxygen exposure over time t in minutes. Although some long-duration exposures were used in Rarabin's data base, the method does not take recovely into account and is most useful for a single, isolated exposure.

d. Calculation of oxygen partial pressures

DCAP calculates a range of oxygen values for each gas mixture, both a "high" and a "low" PO_2 . The high value is used for tolerance estimates, and the low value is used for decompression calculations. The high PO_2 value is based on total absolute ambient pressure and the oxygen fraction in the mixture, while the low value considers CO_2 and water vapor in the ltmgs (see note tmder IV.C.2.b, alveolar gas adjustment, below). This helps solve the matter of defining a range of oxygen levels in a mixture.

C. The Haldane-Workman-Schreiner Tonawanda Ila model

DCAP'S traditional calculation model has been the popular Raldane-Workman-Schreiner or Tonawanda IIa model. It is covered in this chapter in a general way. Depending on the configuration, DCAP is not limited to this model, but it is the one most fully developed and on which most DCAP experience is based.

In general, with this and most other computational models the main calculation perfmmed is to keep track of the inert gas in the body, to compare that with previously determined limits, and to decompress the diver so as not to violate those limits. A more extensive coverage of the rationale for the pure neo-Raldane computations (the original 'Tonawanda II'' model) can be formd in the reference by Schreiner and Kelley (1971). The ''Tonawanda IIa'' model used with DCAP is a further development of the original ''pragmatic'' model of Schreiner and Kelley, which specified a single set of half times based on physiological factors; the DCAP application can use different half-time sets tmder different circumstances.

1. Gas transport mechanism assumptions

For its calculations the Tonawanda Ila system assmnes "perfusion limitation," that blood is fully equilibrated with the alveolar gas in the lungs, and that it equilibrates with tissue "instantly" as well. That

is, the limitation to gas transport is blood flow. This is a characteristic of the Haldane approach. It has a relatively minor effect on the actual tables produced, but it is a matter of concern among theorists. There is good experimental evidence to support the choice of this "perfusion limited" approach.

Another assumption made by the Haldane method is that uptake and elimination are symmetrical; that is, that the same parameters prevail in both directions. This assumption is not so sound, as will be seen, and living with it requires a rather close match between the experience on which a calculation is based and

the resulting new dive. DCAP also allows unsymmetrical uptake and elimination calculations to be used, for example with the gas elimination at a slower rate than the uptake.

2. Alveolar gas

The operational assumptions of DCAP are based on inspired gas values, but Tonawanda II uses internal or "alveolar" levels for the computations. This is a major difference between DCAP and some other decompression computational programs.

a. Rationale for alveolar gas adjustment

Inspired gas is diluted by CO_2 and water vapor in the lungs. This is assumed to be at partial pressures of 40 and 47 mmHg (53 and 63 mb) respectively. These are not particularly important in the pressures used in diving, but they can become significant in altitude calculations where they make up a greater fraction of the total pressure. At a respiratory quotient assumed to be 0.8, the Alveolar Ventilation Equation yields an alveolar inert gas P_A (see Schreiner and Kelley, 1971):

$$P_{A} = F_{I} (P_{B}-37) \text{ mmHg}$$

3 where F_I is the fraction of inert gas in the inspired gas and P_B is ambient "barometric" pressure (that is, the divers ambient pressure). Converting to atmospheric and depth units:

37 mmHg = 0.0493 bar = 0.493 msw 37 mmHg = 0.0487 atm = 1.607 fsw

The appropriate value is subtracted from barometric pressure as indicated during DCAP's calculation of alveolar gas (next section).

b. Units of alveolar gas adjustment

The alveolar ventilation equation, using the above assumptions, yields the following equation that is solved by DCAP. Barometric (atmospheric) rather than depth units are used. If pressure units are bars:

$$P_0 = F_1 (P_D + P_B - 0.0493)$$
 4-

4 where,

 $P_0 = Corrected$ inspired inert gas partial pressure used in calculations, bars

 F_{I} = Fraction of inspired inert gas

 P_D = Ambient pressure at depth, in bars

 P_{B} = Barometric pressure at the surface (normally 1 bar).

Therefore, an entry of **0.0493 bars** is made in the Initialization file or Basecase for the CO2&H2O.FACTOR for tables in msw, or **0.0487 atm** when fsw are used.

NOTICE

When DCAP calculates the "high PO_2 " value it does not subtract the CO_2 and H_2O factor. This is in order to give the "worst case" value and to match partial pressure calculations made by hand. The "low PO_2 " value is calculated in the normal way described here, after subtracting the CO2&H2O.FACTOR, and is the value used in making decompression calculations. "Low" and "high" PO_2 values are parameters that can be used in table displays. The low PO_2 value in DCAP graphics is presented in depth units, not atmospheric units.

3. Gas loading compartments

a. Half times

The model assumes that gas is taken up into a number of "tissues" or "tissue compartments." A compartment is defined as all parts of the body which handle gas at the same uptake and elimination rates. The term "compartment" is preferred over "tissue" to avoid the suggestion of a discrete anatomical entity.

Half times, min					
Cpt#	N2	Не	H2	Ne	Ar
1	5	5	5	5	5
2	10	10	10	10	10
3	25	20	20	20	30
4	55	40	45	40	60
5	95	60	70	65	110
6	145	80	110	85	165
7	200	100	150	110	225
8	285	130	205	145	320
9	385	160	270	180	435
10	520	200	360	225	590
11	670	240	455	275	755

Figure 4-1. Half times, 11 compartment Ton-IIa model, (used in IF1100 and IM1100). Argon values developed in collaboration with R.E. Peterson.

Gas uptake and elimination follows an exponential decay pattern and is normally handled as if it were symmetrical; that is, the same half time is used for uptake and elimination. (If a different assumption is desired there are features in DCAP for making some adjustments, such as might be used for dealing with variables such as cold.) Each compartment handles different inert gases differently and independently. Traditionally the number of compartments used for a particular computational model has varied between 6 and 16. The set of half times is the main characteristic of a different computational "model." A model commonly used with DCAP uses 11 compartments. The half times for the most frequently used 11 compartment models (e.g., IF1100; IM1100) are shown in Figure 4-1. Other models can of course be used.

The half times are normally entered in the Initialization file, which also contains the

definitions of the units to be used.

b. Pi values

The fundamental parameter which the computer calculates is the partial pressure of each inert gas in each compartment. These are known as Pi (or 8) values (Schreiner and Kelley, 1971). The change in partial pressure Pi for a given gas in a given compartment over the time interval t is (in depth units, e.g., msw):

$$Pi = 8 = P_0 + R(t - t_h / \ln 2) - (P_0 - Pi_0 - Rt_h / \ln 2) e^{kt}$$
4-5

where:

Pi or 8 is partial pressure of a gas in a half-time compartment t_h

P₀ is the inspired inert gas partial pressure corrected for CO₂ and water vapor

Pi₀ is the Pi at the beginning of the time interval

R is a constant rate of pressure change in units of depth per min during time interval t

- t_h is half time of the compartment, in minutes
- ln 2 is the natural log of 2, equal to 0.693
- k is the exponential decay constant in units of min⁻¹ that relates the classical "time constant" (based on 1/e) to the half time used here; $k = \ln 2/t_h$.

In cases where computation is made over a short time interval (less than 1 min) or when pressure is constant then R can be considered as zero and Pi is computed by:

$$Pi = 8 = P_0 + (Pi_0 - P_0)e^{kt}$$
.

4-6

Gas partial pressures (Pi values, in units of depth, e.g., msw) for a given compartment are stored as values for the individual gases because they are needed for subsequent computations. However, constraints are based on the total or sum of individual gas partial pressures within each compartment. The computed total gas loading of all the different inert gas partial pressures in a compartment is referred to as the "sum of Pi."

4. Ascent constraints: M-values and matrixes

During descent and bottom time the buildup of gas loading in each compartment, the "sum of Pi", is calculated.

a. Staging and stage steps

Once ascent from the bottom begins it proceeds until a stop is called for, and from that point the diver ascends in "stages" or "stops" to the surface. (The stops are for operational convenience; a linear ascent can be used as well, even for short dives.) A convenient staging interval or step of 3 msw (or 10 fsw) is used for msw dives near the surface, but 5 msw may be used during the deep part of the dive. Making

stage increments much greater than this will cause the diver's ascent to be faster, and the resulting table may be unreliable unless it can be supported by experience. Most of the experience with DCAP has involved stage intervals of 3 msw (or 10 fsw), and it is recommended that stops larger than this be used only when they are well justified.

b. Ascent limits

Ascent or pressure reduction is controlled by the relationship between the gas loadings and a set of predetermined limits of maximum tolerable partial pressure known as M-values. Ascent is stopped when, because of the reduction of ambient pressure, the sum of Pi (the gas loading) in any compartment exceeds the M-value for that compartment at that stop depth. On a normal ascent depth is reduced faster than gas can be unloaded, so each compartment soon has a gas loading greater than the current depth. As soon as a depth is reached where the inert gas loading exceeds ambient pressure the diver is held at that depth until the gas loading again falls below the M-value in all compartments. These calculations are made at each preselected stop depth or staging interval.

c. M-values

The M (for maximum tolerable) values have been empirically determined from previous dives. They represent a fuzzy limit beyond which the risk of decompression sickness is unacceptable for the situation.

An M-value is a gas loading, expressed in absolute units of gas partial pressure. As with some of the other partial pressures it is in depth units, e.g., msw. It will be a few msw larger than the absolute partial pressure for the depth in question; this difference represents the hypothetical "tolerable supersaturation" in that compartment at that depth.

d. The matrix of M-values

Historically a "matrix" or chart of specific M-values for each compartment at each staging depth has been used. In the present model an alternative method is used to define the matrix, whereby each compartment's ascent limiting constraint is calculated when it is needed. An equation for a straight line (of the form y = mx + b) is used in which the M-value at the surface has an intercept or "base" (B) and a slope (S) which yields a proper M-value at each stop depth (D). D_B is base depth, normally zero (i.e., sea level) but it can be changed as desired.

$$\mathbf{M} = \mathbf{S} \left(\mathbf{D} - \mathbf{D}_{\mathbf{B}} \right) + \mathbf{B}$$

This method performs in a conservative manner the same function as a "matrix" having a specific M-value at each depth.

DCAP interpolates the M-values and allows intermediate staging intervals to be used. As mentioned above, it is important that the staging intervals used be quite similar to those which have been determined empirically to be effective.

e. Evaluation of M-values: Other approaches

One way in which DCAP running the Tonawanda IIa model may differ from other similar programs is in the way M-values are evaluated. Tonawanda IIa here allows ascent to begin once all M-values are cleared **at the present depth**. The other approach, which is used by similar neo-Haldanian systems

(e.g., Lambertsen's PADUA, the University of Pennsylvania program; this is an in-house program which has been used by some others but which has not been published or released), is to **look ahead** and predict that the diver can make it to the next stop without exceeding the M-values for that stop. Thus there is an offset between those systems and Tonawanda IIa of one staging interval (a "stage step," usually 3 msw or 10 fsw), such that Tonawanda IIa's —values at the last stop (3 msw or 10 fsw) look like PADUA's values at the surface. There are advantages to each of these approaches and the results are similar. The important thing is that it may not be correct to use a matrix from another system in DCAP without an appropriate offset (or vice versa).

D. The Exponential-Linear model

For many years the Haldane approach just described has—in several forms—been the leading method used for the calculation of decompression tables. Although it is probably the most practical model, it occasionally does not allow a wide range of empirical results to fit a single set of parameters. For example, a set of half times and M-values that agree with experience well in very short dives may not give correct values for a long dive.

One approach to bridging this gap with a single model, the Exponential-Linear model, uses different assumptions about gas transport. It assumes the exponential gas uptake described above, but for gas elimination it uses a slower "linear" exchange. The linear exchange rates are constant throughout the gas exchange period rather than proportional to the difference in gas partial pressures; linear exchange rates are also adjusted by means of empirically determined parameters.

This model was developed by E.D. Thalmann, working at the U.S. Navy Experimental Diving Unit in Panama City, Florida. Because both the algorithm, which is designated "EL-MK 15/16 DCM," and the testing of it have been described in great detail by Dr. Thalmann's reports, the coverage is brief here (1980; 1983; 1984; 1985; 1986). Comparisons to "DCAP" and DCAP's functions are based on the Tonawanda IIa neo-Haldanian model.

The equations for DCAP's gas uptake using the E-L model are the same as those used by the Tonawanda IIa model (Equations 4-5 and 4-6). The equation used by DCAP with the E-L model for outgassing when inspired PO_2 is constant is:

$$Pi = Pi_0 + (ATM.FACTOR \times 0.085 - P_1O_2) (IGR) \times (t - t_0)$$
4-8

where P_1O_2 is the inspired oxygen partial pressure and 0.085 is a correction factor (in atm) incorporating venous oxygen and arterial and venous CO_2 . IGR is the Inert Gas Ratio, and the other factors are as above. This equation is used whenever the gas loadings (Pi) approach ambient pressure (within about 0.1 atm). The term ATM.FACTOR is the unit definition, the number of depth units per atmosphere. An additional term accounts for the rate of change of P_1O_2 , which was not used by Thalmann. The variables have where possible been put in DCAP form.

Also, because DCAP handles multiple gases the inert gas portion of the equation is multiplied by a factor IGR (inert gas ratio). IGR is the ratio of the inert gas in use to all the inert gas.

A problem was encountered in adapting the E-L model to work under DCAP. DCAP is made to handle several inert gases with different properties (the individual gases are usually characterized in DCAP by their half times). The E-L model (at least in the version used here) handles only one gas at a time. It can

do either nitrogen or helium, but not at the same time. E-L with heliox uses the same set of half times for each gas, and deals with gas behavior by adjustments in the matrix and SDR. Also, the nitrogen matrix used by Thalmann is 9 compartments and his helium matrix uses only 6.

The model and its behavior are illustrated in Figure 4-2, from Thalmann, 1984. This shows, in slightly different symbols, the rise in tissue inert gas tension (solid line) with a step increase in arterial inert gas tension from P_1 to P_2 , followed by a linear decrease. The dotted and dashed line shows how the "exponential-exponential" or traditional Haldane model would handle the same outgassing. The curved tail is the result of tissue N₂ decreasing below venous N₂.



Figure 4-2. Gas transport example of Exponential-Linear model. (Thalmann, 1984)

There is another other twist to the E-L model when used for heliox diving. As soon as the diver begins to decompress the half times change slightly. The values of these changes are determined empirically. This is managed by a term "SDR," the saturation-desaturation ratio. The SDR is a value of 1.0 or slightly less, and the half time of each compartment is divided by this value as soon as outgassing begins. This increases the half times and slows the ascent. The SDR is not used with the nitrogen model described here, but is used for a later version with matrix VVAL59 (Thalmann, 1986).

The E-L model makes assumptions slightly different from those of Tonawanda IIa about the effect of the water vapor and CO_2 in the lung, and it can consider standard arterial and venous blood gas values as well. Although they are used in the derivation, no physiological values other than the standard ones have been used in normal calculations.

The E-L model has been highly refined over several years at NEDU. While this program has resulted in improvements in the calculation methods, the important thing is that **several thousand test dives** were carried out as part of this development effort. This has resulted in a high level of reliability in dives calculated with this procedure—the objective has been to validate a **method** more than a specific set of tables. The type of dive of primary interest has been one that uses a closed-circuit rebreather, covers several depths over a long dive period, may be with either heliox or nitrox, and uses a constant oxygen partial pressure of 0.7 atm. The use of this low PO_2 is physiologically acceptable but it generates rather inefficient decompressions.

In implementing the E-L model in DCAP we first included Thalmann's equations as he used them, and adjusted the program so that DCAP would calculate the same values, exactly in most cases but always within 1 or 2 minutes; these small differences we regard as due to rounding practices, and we do not consider them meaningful physiologically. This showed that DCAP could reproduce the E-L results accurately, but it required special computations (in the subroutine INSET) regarding the assumptions about CO_2 and water vapor in alveolar gas, and these did not fit the DCAP format. The CO_2 and water vapor during uptake in the original E-L model acts only on the inert gas fraction, but not on oxygen. This differs slightly from the way it is handled in the standard DCAP gas transport algorithm, where the CO_2 and H₂O are deducted from the total pressure and the gases are all affected proportionately.

Because DCAP has long relied on rigorous gas transport equations in the Tonawanda IIa model, it seemed appropriate to use this approach for the uptake of the E-L model as well. Therefore we made the decision to make the calculations in the standard DCAP manner so that the DCAP user could use the E-L model in a familiar way. DCAP assumes that the exponential (uptake) part of an E-L dive is done with the standard DCAP uptake algorithms. This makes differences of a few minutes in long tables. With the standard CO₂ and H₂O factors the DCAP results are in the order of 5% more conservative (decompression is longer) than Thalmann's published results for nitrogen and a PO₂ or 0.7 atm. Preliminary testing showed that this difference may be slightly greater with long helium dives, but again it is on the conservative side. It appears that by some refinement of the CO2&H2O parameter a closer fit can be obtained, still without having to change the M-values. Schmidt (1997) has worked out a set of M-values that matches the published E-L tables within the limits of rounding.

We investigated whether realistic nitrogen half times could be used for the gas present in the diver's body at the start of a helium-oxygen dive. The E-L pattern in effect calls for the diver to be saturated with helium at the beginning of a dive. That is, there is no difference between helium and nitrogen half times. When nitrogen half times are tried it slows the decompression considerably (because the initial load of nitrogen is slow in washing out). If a user wants to regard helium and nitrogen as having different properties in the same dive, it will be necessary to adjust other parameters to compensate. It does not appear that USN is interested in undertaking this task.

This is the "original" of the E-L Mk 15/16 decompression models. The initial E-L model was able to handle constant oxygen partial pressures but not constant fractions, so that air dives could not be calculated. A revised version using two new models, including the VVAL59 mentioned above (Thalmann, 1986), allows this; it uses a more sophisticated method of dealing with gases in the body, including the use of hemoglobin dissociation curves. This specific version is not yet implemented in DCAP. The E-L Mk15/16 DCM-II model's method of accounting for CO_2 and H_2O is probably closer to the method used by DCAP than the earlier version, but we have not made the comparison. Since VVAL59 is entered into DCAP as a matrix it can be tried without further modification of the DCAP program.

The E-L computational method with its extensive empirical foundation and novel approach to computation has been used by one operational group in a modified form to produce more efficient decompressions. Whereas the USN tests were with divers breathing 0.7 atm PO_2 , high levels of oxygen can be used, up to an operational limit of 1.4 atm PO_2 . This has proven to be a highly efficient—virtually optimal—oxygen level that at the same time offers minimal risk of oxygen toxicity (Hamilton, Kenyon, and Clough, 1988).

It is worth noting that the U.S. Navy has more or less switched their approach to decompression development from this "deterministic" approach to one based on statistical analysis of dive experience. DCAP currently does not perform this type of calculation, but the maximum likelihood assessment of existing tables or profiles uses some of the same techniques.

E. The Kidd-Stubbs or DCIEM model

1. Development of the series-compartment model

A pneumatic analogue decompression computer was developed for the Canadian Forces in the early 1960's by Wing Commander R.A. Stubbs and Surgeon Commander D.J. Kidd (Stubbs and Kidd, 1965a; 1965b) at the laboratory that is now the Defence and Civil Institute of Environmental Medicine, Toronto. The device assumes that the body consists of four compartments in series; gas is assumed to pass between the compartments as a function of differences in partial pressure. The original pneumatic device used 4 bellows connected in series with small orifices; each compartment had a pressure gauge. Gas passed into and out of each compartment as a function of its pressure in the adjacent compartments, and of course of the orifice sizes, density, temperature, etc. The original device consisted of four small metal bellows connected by orifices drilled through sapphires, with linkages and needles to display the pressures. The device was empirically refined so as to generate suitable decompression tables for air dives if pressure limits in the compartments were not exceeded. It was built originally to mimic as well as possible the US Navy air tables, but to make it possible to manage multilevel ("non-square") decompressions. For "mechanical" reasons the pneumatic devices were never very successful when carried by a diver or even when used in the field, but they worked well on chambers. Quality control was difficult, and two units that should have been equivalent often did not give the same decompression profile; this was regarded as a serious defect by some experts. Even so, considerable successful experience accumulated on these computers in chamber applications (usually more than one of them was used on each chamber).

While the Kidd-Stubbs model has some attractive features, it is not really a physiological model. Some gas transport in the body acts as if it were compartments in series, but this is certainly not the case everywhere. Further, the constants for gas flow through an orifice do not apply to body gas transport. On the other hand, the inherent asymmetry of this model matches quite well what seems to be necessary for long, deep dives.

The analogue was for many years only a real-time device and there were no printed tables to go with it. When digital computers were becoming more accessible, DCIEM set out to develop a digital analog of this pneumatic analog. In 1971 a computer program was written that permitted the calculation of tables (Nishi and Kuehn, 1973). The simultaneous differential equations for flow in and out of the various compartments do not lend themselves to easy, exact solutions, but were solved numerically. Nishi and Kuehn used a Runge-Kutta iteration algorithm which proved successful and which has been incorporated

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into DCAP. We do not go into great detail here about the mathematics of this model but refer the user to the original DCIEM reports (Nishi and Kuehn, 1973; Nishi and Lauckner, 1984).

In 1983 DCIEM released a set of air tables based on computer emulations of the Kidd-Stubbs model (Lauckner and Nishi, 1984). This set of tables was an outstanding example of decompression table development. The team set out to correct known deficiencies in the pneumatic models and in the leading air tables, those of the U.S. and Royal British Navies. An iterative process alternated between well-controlled laboratory tests and a revised algorithm, and resulted in a set of tables that has become highly regarded.

As a real-time calculator and even in early forms of the computer emulator the main target was a safe ascent depth. The diver ascended to the indicated "SAD" and then gradually ascended as the gases unloaded as the SAD was reduced. The term "ceiling" is better than "safe ascent depth" since nothing involving decompression can be guaranteed to be "safe."

One aspect of the Kidd-Stubbs model that was troublesome in the process of converting from a real-time computer to tables was the calculation of no-stop or "no-decompression" dives. Some of the dives tended to be more conservative than ones on which vast experience had accumulated and that were working well. It was eventually decided that for the Canadian Forces the no-stop times would be arbitrarily adjusted to match practical, operational tables (Department of National Defence-Canada, 1986).

Another problem of the pneumatic model was that it treats the whole mixture as a single gas. This meant that oxygen had no special effect. As long as diving was always on air only, this made no difference, but when the intent was to expand the diving to include things like surface decompression with oxygen and oxygen decompression it was necessary to make changes. DCIEM did this by "removing" the oxygen fraction internally and continuing the calculations with the inert gases only. DCAP focuses on the inert gas, next section.

2. Kidd-Stubbs parameters

The Kidd-Stubbs model as presently used has four compartments. The gas is monitored in all of the compartments, but only the first two are used with constraints. The third and fourth compartments are important because they affect both the uptake and elimination of gases in the first two compartments. This is what gives the Kidd-Stubbs model the ability to deal with rather long dives with only four compartments.

Four different parameters are used (Nishi and Kuehn, 1973; Nishi and Lauckner, 1984). The first two are familiar and we have adapted them to DCAP terminology. These two are a pressure **offset** (P_{OFF}) and a dimensionless **ratio** (R), as used in the DCIEM reports.

Two other parameters are called A and B by DCIEM; we call them Kidd-Stubbs A and Kidd-Stubbs B. These are parameters related to gas flow through the orifices. A has a value in the range of 10⁻⁴ and B has a value in the range of 100 to 300 or so. These have been worked out for other gases, but the values that we have used are those appropriate for nitrogen. Raising either of these parameters causes an increase in the decompression time, although it is not necessarily a uniform increase; both of them tend to affect the shorter and faster dives more than the longer ones. DCAP Manual. IV. DCAP calculations Part One. G. The t-delta-P model

An additional set of numbers are needed for the Kidd-Stubbs computation. These are a series of 6 numbers which act as "seeds" for the Runge-Kutta iteration. These are not related to decompression but are handled by DCAP as variables. They are called Kidd-Stubbs Z. We have not explored the effect of changing these parameters.

In adopting the Kidd-Stubbs model to DCAP we have had to make some small compromises. For one, the original Kidd-Stubbs pneumatic analogue measured the flow of **air** rather than just the inert gas. Because DCAP is equipped to deal with the inert gases only we have adjusted the published R and offset values to deal with the inert gases only (by dividing by 0.791, the nitrogen fraction in air). Also there is no provision in the DCIEM use of the model for carbon dioxide and water vapor; when these are included the results do not exactly match the published DCIEM tables but they are quite close and follow the same pattern.

DCIEM has recently done a set of heliox decompression tables. This has required modifications of the parameters on the Kidd-Stubbs model. It should be possible to include these parameters in DCAP and to make calculations of heliox tables when they become available. Tables with more than one inert gas, e.g., "trimix," require adjustments of the program itself. This is done by proportioning the gases.

F. The Tiny Bubble model

This model was developed by Dr. David Yount and colleagues at the University of Hawaii. It was inspired by some meaningful observations in the laboratory, primarily the behavior of bubbles in gelatin, that tied in with clinical data. The model is more properly called the varying-permeability model (VPM). Because the model is described in some detail in published reports (Yount and Hoffman, 1986), it is covered only briefly here. The model's basis is the observation that bubble formation in aqueous media following naturally occurring pressure changes seems to require the existence of stable micro nuclei. The gas tensions in the body tend to dissolve bubbles, so some explanation is needed for the persistence of microtubules. Two factors which could bring this about are an impermeable "skin" on the bubble, or a very low surface tension at the gas-fluid interface. The postulated varying permeability membranes can be permeable to gas, but this permeability is reduced when the bubbles are subjected to large compressions.

In order to refine the bubble model Yount made the assumption that existing decompression tables could be treated as valid data (a practical but not very physiological approach). The model is looking for a "critical bubble volume," but the "bubbles" are dynamic, constantly exchanging gas during the course of a dive and decompression. Some bubbles are tolerated and trapped by the lung in the usual way. The number of supercritical nuclei can exceed a "safe" limit temporarily. The total gas released continues to increase until some time after the dive when the level of supersaturation returns to zero. At this point the maximum volume has to be less than the critical volume.

The computation specifies 6 nucleation parameters. These are the surface tension (gamma), the nuclear skin compression (gamma.c), the minimum initial radius (r.min), the time constant for the regeneration of nuclei crushed in the initial compression (tau), and a composite constant (lambda) that is related to the critical volume and the degree the bubble count can exceed the "safe" limit. The model involves preliminary estimation of a minimum supersaturation level able to produce a "safe" number of bubbles. Then the nuclear radius, which changes with changes in pressure, is allowed to regenerate according to a algorithm based on statistical mechanics. A preliminary table is calculated, with a total decompression time. Using this total time a new supersaturation level is estimated, from which a new radius is estimated.

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Part One. G. The t-delta-P model

This is done for each "tissue" compartment. This process is iterated until total decompression time and supersaturation pressure "converge," implying that the relation between actual bubbles and the safe level is now tolerable.

The first 5 of the parameters are specified in DCAP specifically as Tiny Bubble parameters; supersaturation is the conventional one. Using values given by Yount, DCAP will calculate a table essentially the same as the samples in his report. The report describes the function of the parameters so adjustments should be possible, but we have no experience in doing this.

G. The t-delta-P model

It is well established that the Haldane computational model tends to provide inadequate decompression for longer, deeper dives when set to provide an efficient decompression for shorter and shallower exposures. This problem has been addressed in a variety of ways. Among these are new approaches such as the E-L model and the use of conservatism "factors" for longer exposures as used for Swen 88, (Hamilton, Muren et al, 1988). Another approach is the t)P or t-delta-P model. This is based on the concept that the risk of DCS increases with the duration of exposure to supersaturation. This idea has been around for some time (Peterson and Greene, 1976; Greene et al, 1978; Hamilton et al, 1980) and has been implemented by Peterson and Hamilton (1987); this reference provides an operational description of the method. The method was developed to account for the observation that ascent from saturation on nitrogen-oxygen mixtures requires slower average ascent rates as the **depth** of saturation increases.

Briefly, t)P performs bookkeeping on two parameters. One is the traditional gas loading, which is calculated by the exponential method and constrained by M-values in the familiar way. The other item "monitored" by the method is an integration of supersaturation over

time, the sum of the incremental product of supersaturation over Supersaturation is the excess gas loading above ambient pressure. In use, the t)P method "keeps books" on the t)P integral for each halftime compartment in the same manner that the Tonawanda IIa or Haldane model monitors gas loading; gas loading is monitored as well, and ascent is halted whenever its limits exceed the matrix limit in one or more compartments.

The t)P calculation is an iteration, because the total t)P value cannot be known until the dive is complete. In practice DCAP uses the "Vann k" parameter to choose an acceptable ascent rate that will not exceed the empirically determined t)P allowable. Vann k is a method of adjusting saturation decompression ascent rate to account for changes in oxygen partial pressure of the breathing mix. The actual unit is fsw/hr-atm PO₂ (Vann, 1984). DCAP sets a high value for Vann k, performs the decompression, checks the resulting t)P's against the limits, and if there is no violation adjusts the rate of ascent by changing Vann k to a smaller number (less conservative, in increments of 0.1) and tries again. Once the limit is reached the last Vann k value that did not violate is saved and used for the decompression. Table 4-I. t)P values for nitrox saturation diving. Values are derived from HydroLab and commercial experience (Peterson and Hamilton, 1987).

Half time	Maximum t) P fsw-min		
5	0.5		
10	1		
25	2		
55	7		
95	19		
145	32		
200	84		
285	2000		
385	9000		
520	25000		
670	45090		

A list of t- P limits used with DCAP is given in Table 4-1. These have been derived from experience with nitrox sahrration decompression (Peterson and Hamilton, 1987). This is the only application for which reliable experience is provided, but the method should be able to work with other kinds of decompression as long as data from successful dives is available. The tmits of t• P are fsw-minutes; these are the original tmits derived by Peterson, and are computed by DCAP in these tmits regardless of the tmits used for the dive.

H. The Bühlmann algorithm

Swiss Professor Albelt A. Biililmann began working in decompression in 1960. Although his main vocation was as a cardiologist, he worked in many areas of decompression, with palticular emphasis on commercial deep bell, deep sahrration, recreational, and altihtde diving. His impact on the field was enormous, but became palticularly intense and international in recreational diving by his publishing a small book in 1984 in English that gave the mathematical algorithms for calculating decompressions (Bühlmann, 1984). These have been applied by numerous dive computers (most of the existing ones) and do-it-yourself decompression computation programs for personal computers used by technical divers. These were updated by subsequent editions in Ge1man, one in 1993, and a 4th edition published posthumously in 1995 with a colleague, Emst Völlm, and his son Thomas (Biililmann, 1995). An English translation of the 4th edition is in preparation. The methods used by DCAP are based on these books, and references are to page numbers in the 4th edition.

The Biililmann model has a different approach to the calculations, but the decompression method is strictly Haldanian in its operation.

The inelt gas pressmed differential, the gradient, is the driving force for the gas transport between alveoli, blood, and tissues. As with Tonawanda Ila and other neo-Haldanian methods, gastranspolt takes place exponentially and symmetrically for uptake and elimination (Biililmann 1995, p. 64).

The resulting exponential pressurization of inert gases is calculated by means of half-times:

$$P_{t} \underline{\boldsymbol{r}}_{\boldsymbol{\theta},\boldsymbol{y}} = P_{t} \underline{\boldsymbol{r}}_{\boldsymbol{\theta},\boldsymbol{y}} + P_{t} \underline{\boldsymbol{r}}_{\boldsymbol{\theta}} - P_{t} \underline{\boldsymbol{r}}_{\boldsymbol{\theta},\boldsymbol{y}} \cdot \left[1 - 2^{-\frac{t_{\star}}{T_{\star}}} \right]$$

where

p tE)= inert gas pressure in the tissue at the end of the exposure,p tO)= inert gas presstu-e in the tissue at the beginning of the exposure,Piig= inert gas presstu-e in the (Inspired) breathing gas • PAig in the alveoli,tE= exposure time in min,T y,= half time in min.

The numerical value of the first bracket represents the effective inert gas paltial pressure gradient between the inspired gas and the body tissues. The numerical value of the second bracket, depending upon the exposure time and the half time, gives the fraction of the gradient that must be added to and/or subtracted from the initial inelt gas paltial pressure in the tissue.

This equation is essentially the same as the one used by DCAP's TIIa, with one exception. Tonawanda IIa has a te1m dealing with inert gas transpOlt during changes of pressure, using the rate of change of pressure. This makes it necessary, when making comparisons between TIIa and this and other algorithms, to account for gas changes during pressure changes.

If air is breathed the gas uptake for N_2 is calculated with the P_1N_2 resulting from the (Inspired) gas being breathed at the ambient pressure. However, in cettain conditions the PAN₂ (alveolar nitrogen pattial pressure), which is decisive for the gastranspOlt between blood and tissues, can occasionally be considerably lower or higher than the P_1N_2 , and this must be considered in the interpretation of experimental results and the calculation of decompression profiles (p. 64).

The analysis of precisely recorded decompression profiles, with and without symptoms of decompression sickness, is the only practicable way to the goal of determining the symptom-free tolerated inert gas overpressure in the tissue. These boundaries are verified **if** it can be shown that numerous symptoms of decompression sickness appear when the botmdaty is exceeded by some small increment (p. 75).

With longer dive times and on deeper dives, the ascent to the surface must be slower. This is done by empirical means, by determining what inert gas overpressure in the tissue is tolerated trouble-free by a representative collection of people for each rapid reduction of the ambient pressure (p. 78).

At no point during the decompression, regardless of whether it takes place continuously or in steps, should the tolerated inert gas overpressure be allowed to exceed the respective ambient pressure. The assumption of a practically **linear** relationship between ambient pressm.e and tolerated inert gas overpressure in the tissue is sound. This linear relationship can be formulated mathematically (p. 78):

$$P_{tiat}ig = \langle P_{abb}/b \rangle + a,$$
 4-10

or

The numerical value of Coefficient a is dependent upon the pressure unit. Coefficient **b** has no dimension; it determines the slope of the relationship between ambient pressure (Pamb) and tolerated inelt gaspresstue in the tissue (Puotig). The second equation shows the maximum tolerated ambient pressure (Pambtot) for a given tissue inett gaspartial pressm·e (P_ig).

In studying the possibilities, Bühlmann fotmd a surprisingly simple solution for a direct mathematical derivation of Coefficients a and **b** using a f01mula for a linear relationship between ambient pressure and tolerated N_2 overpressure (p. 86):

Coefficient
$$a = 2.0$$
 bar $(TyN_2[min])$ -a. 4-12

Coefficient
$$b = 1.005 - (1 \circ (T_y, N_2[min]) \cdot Y_{.}).$$
 4-13

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Example: For a $T_{\frac{1}{2}}N_2$ of 27.0 min, Coefficient a = 2 • (27)^{-1/3} = 0.6667 bar (the exponent is -1/3). Coefficient b = 1.005 - (1 • (27)^{-1/2}) = 0.8126 (the exponent is -1/2) (p. 86).

With increasing half-time, Coefficient a becomes smaller and Coefficient b becomes larger.

The lines of the relationship between ambient pressure and the tolerated N_2 -pressure in the tissue converge with decreasing ambient pressure. The lines remain separate up to a vacuum (zero absolute pressure), however (p. 86).

DCAP deals with the Bühlmann algorithm by means of Coefficients a and b for each of the gases handled. Although DCAP could have been programmed to calculate a and b using these equations, in order to provide more flexibility DCAP requires that these a and b values be specifically set; this is usually done in the Init file.

DCAP calculates the ascent limits from **a** and **b** by converting them to M-values as described by Equations 4-10 and 4-11, above.

$$M = (depth at stop/b) + (a * UNIT.DEFN)$$
4-14

Where

M = M-value at the stop depth, maximum tolerable inert gas partial pressure
b = Coefficient b
a = Coefficient a
UNIT.DEFN = the DCAP parameter defining the number of depth units in an atmospheric unit (i.e., 10 for msw and 33 for fsw operations).

The resulting M values are handled as others are by DCAP, and the calculated values can be displayed in a Loadings file. Otherwise the operation is the same as for other Haldanian models such as Tonawanda IIa. DCAP methods are given in Chapter VII.

It can be observed that as **a** gets larger the M-value gets larger and the dive gets less conservative. As **b** gets larger M gets smaller and the dive becomes more conservative.

XI. REFERENCES AND BIBLIOGRAPHY

- Bardin H, Lambertsen CJ. 1970. A quantitative method for calculating cumulative pulmonary oxygen toxicity: Use of the Unit Pulmonary Toxicity Dose (UPTD). Philadelphia: Institute for Environmental Medicine, University of Pennsylvania.
- Behnke AR. 1967. The isobaric (oxygen window) principle of decompression. In: Steer RC, ed. The new thrust seaward, Transactions of the Third Annual Marine Technology Society Conference. Washington: Marine Technology Society. Pp 213-228.
- Bell RI, Borgwardt RE. 1976 Mar. The theory of high-altitude corrections to the U.S. Navy Standard Decompression Tables. The Cross corrections. Undersea Biomed Res 3(1):1-23.
- Bohrer CR, Hamilton RW. 1993. A provisional method of oxygen exposure management for a recreational dive computer. J Undersea & Hyperbaric Med 20(Suppl):72.
- Bühlmann AA. 1984. Decompression: Decompression sickness. Berlin: Springer-Verlag.
- Bühlmann AA. 1993. Tauchmedizin: Barotrauma, Gasembolie, Dekompression, Dekompressionskrankheit. Berlin: Springer-Verlag.
- Bühlmann AA. 1995. Tauchmedizin: Barotrauma, Gasembolie, Dekompression, Dekompressionskrankheit, Dekompressionscomputer. Fourth ed. Berlin: Springer-Verlag.
- Clark JM, Lambertsen CJ. 1971. Pulmonary oxygen toxicity: A review. Pharmocol Rev 23(2):37-133.
- Conkin J, Edwards BF, Waligora JM, Stanford Jr J, Gilbert Jr JH, Horrigan Jr DJ. 1990 Aug. Updating empirical models that predict the incidence of decompression sickness and venous gas emboli for shuttle and space station extravehicular operations. NASA Technical Memorandum 100456 (Update). Houston: NASA JSC.
- Conkin J, Van Liew H. 1992 Nov. Failure of the straight-line DCS boundary when extrapolated to the hypobaric realm. Aviat Sp Environ Med 63(11):965-970.
- Department of National Defence Canada. 1986 Apr. Canadian Forces air diving tables and procedures (Change 1 included). Downsview, ON: Defence and Civil Institute of Environmental Medicine.
- Donald K. 1992. Oxygen and the diver. Hanley Swan, Worc., UK: The SPA Ltd. (Images Booksellers & Distributors Ltd/The Self Publishing Association Ltd.)
- Egi SM, Brubakk AO. 1995 Sep. Diving at altitude: A review of decompression strategies. J Undersea & Hyperbaric Med 23(3):281-300.
- Fairburn SM, Morrison JB. 1990. A dynamic model of CNS O₂ tolerance based on depth, workrate, and exposure time. Undersea Biomed Res 17(Supp):166.

- Flynn ET, Catron PW, Bayne GC. 1981. Diving Medical Officer student guide. Course A-6A- 0010. Panama City, FL: Naval Diving and Salvage Training Center.
- Gernhardt ML. 1991. Development and evaluation of a decompression stress index based on tissue bubble dynamics. Ph.D. thesis, University of Pennsylvania. Ann Arbor, MI: University Microfilms International Dissertation Information Service.
- Greene KM, Peterson RE, Lambertsen CJ. 1978. Decompression from saturation exposures. In: Lambertsen CJ, Gelfand R, Clark JM, eds. Work capability and physiological effects in He-O₂ excursions to pressures of 400-800-1200-1600 fsw. Philadelphia: Institute for Environmental Medicine, University of Pennsylvania.
- Hamilton RW, ed. 1976. Development of decompression procedures for depths in excess of 400 feet. WS: 2-28-76. Bethesda, MD: Undersea Medical Society.
- Hamilton RW. 1984. Man made diving environments. In: Schilling CW, Carlston CB, Mathias RA, eds. The physician's guide to diving medicine. New York: Plenum. Chapter I, sec D, pp. 12-33.
- Hamilton RW. 1989 Dec. Tolerating exposure to high oxygen levels: Repex and other methods. Marine Tech Soc J 23(4):19-25.
- Hamilton RW. 1990 May. Evaluation of submarine free ascent options using gas loading analysis. Presented at International Symposium on Naval Medicine, Kiel, May 25-27.
- Hamilton RW, ed. 1995. Effectiveness of dive computers in repetitive diving. UHMS 81(DC)6-1-94. Bethesda: Undersea Hyperbaric Medical Society.
- Hamilton RW, Kenyon DJ, Clough SJ. 1988. Development of a new diving method based on a constant PO₂ rebreather. Undersea Biomed Res 15(Suppl):95.
- Hamilton RW, Kenyon DJ, Peterson RE. 1980. Effect of duration of exposure to M-values on their validity.In: Berghage TE, ed. Decompression theory. 29WS(DT)6-25-80. Bethesda, MD: Undersea Medical Society.
- Hamilton RW, Kenyon DJ, Peterson RE. 1987. Development of decompression procedures for undersea habitats: Repetitive no-stop and one-stop excursions, oxygen limits, and surfacing procedures. In: Bove AA, Bachrach AJ, Greenbaum Jr LJ, eds. Underwater and hyperbaric physiology IX. Bethesda, MD: Undersea Hyperbaric Med Society.
- Hamilton RW, Kenyon DJ, Peterson RE. 1988 May. Repex habitat diving procedures: Repetitive vertical excursions, oxygen limits, and surfacing techniques. NURP Technical Report 88-1B. Rockville, MD: NOAA, U.S. Department of Commerce.
- Hamilton RW, Kenyon DJ, Peterson RE, Butler GJ, Beers DM. 1988 May. Repex: Development of repetitive excursions, surfacing techniques, and oxygen procedures for habitat diving. NURP Technical Report 88-1A. Rockville, MD: U.S. Department of Commerce.

- Hamilton RW, Muren A, Röckert H, Örnhagen H. 1988. Proposed new Swedish air decompression tables.In: Shields TG, ed. XVth annual meeting of the EUBS: European Undersea Biomedical Society.Aberdeen: National Hyperbaric Center.
- Hamilton RW, Schane W. 1990 Oct. Chisat I, extension and validation of NOAA's new Repex procedures for habitat diving: A Chinese-American collaboration. Report 90-1. Silver Spring, MD: NOAA National Undersea Research Program.
- Hamilton RW, Shi ZY, Gerth WA, Zhuang Q. 1996 Mar. Chisat II: Procedures for excursing with helium breathing mixtures from a nitrogen-based undersea habitat. NOAA Award No. NA46RU0506. Tarrytown, NY: Hamilton Research, Ltd.
- Harabin AL, Homer LD, Weathersby PK, Flynn ET. 1987. An analysis of decrements in vital capacity as an index of pulmonary oxygen toxicity. J Appl Physiol 63(3):1130-1135.
- Harabin AL, Survanshi SS. 1990. Risk of human toxicity under hyperbaric conditions. Bethesda, MD: Naval Medical Research Institute.
- Harabin AL, Survanshi SS, Homer LD. 1995 May. A model for predicting central nervous system oxygen toxicity from hyperbaric oxygen exposures in humans. Toxicol Appl Pharmacol 132(1):19-26.
- Hardy J, Shuster B. 1993 Feb. Computer age. Rodale's Scuba Diving 50-57, 102-107.
- Haux G. 1982. Subsea manned engineering. San Pedro, CA: Best Publishing Co.
- Hills BA. 1976. A cumulative oxygen toxicity index allowing for the regression of effects at low inspired oxygen partial pressures. Rept. 4/76. London: Royal Navy Physiological Laboratory.
- Hollobone TA, rapporteur. 1982 Mar. Recommendation for flying after diving. TAH-chb-D37. London: Diving Medical Advisory Committee.
- Horrigan Jr DJ, Waligora JM, Gilbert J, Edwards B, Conkin JH, Stanford J. 1989. Decompression in space.
 In: Vann RD, editor. The physiological basis of decompression. 75(Phys)6/1/89. Bethesda, MD: Undersea Hyperbaric Med Society. Pp. 425-437.
- Kenyon DJ, Hamilton RW. 1989 Sep. Managing oxygen exposure when preparing decompression tables.In: Bitterman N, Lincoln R, eds. Proceedings XVth Meeting EUBS. Haifa: Israeli Naval Hyperbaric Institute.
- Lang MA, Hamilton RW, eds. 1989 Jan. Proceedings of the American Academy of Sciences Dive Computer Workshop. USCSG-TR-01-89. Costa Mesa, CA: American Academy of Underwater Sciences.
- Lang MA, Vann RD, eds. 1992. Proceedings of Repetitive Diving Workshop. AAUSDSP- RDW-02-92. Repetitive Diving Workshop. Costa Mesa, CA: American Academy of Underwater Sciences.
- Lauckner GR, Nishi RY. 1984 Dec. Decompression tables and procedures for compressed air diving based on the DCIEM 1983 decompression model. DCIEM 84-R-74. Downsview, ON: Defence and Civil Institute of Environmental Medicine.

- Miller JW, ed. 1979. NOAA diving manual. Second edition. Rockville, MD: NOAA, U.S. Department of Commerce.
- Muren AM, Adolfson J, Örnhagen H, Gennser M, Hamilton RW. 1984. Nisahex: Deep nitrox saturation with nitrox and trimix excursions. In: Bachrach AJ, Matzen MM, eds. Underwater Physiology VIII. Bethesda, MD: Undersea Medical Society.
- Nishi RY, Kuehn LA. 1973 Jan. Digital computation of decompression profiles. DCIEM 884. Downsview, ON: Defence and Civil Institute of Environmental Medicine.
- Nishi RY, Lauckner GR. 1984 Sep. Development of the DCIEM 1983 decompression model for compressed air diving. DCIEM 84-R-44. Downsview, ON: Defence and Civil Institute of Environmental Medicine.
- Peterson RE, Hamilton RW, Curtsell I. 1980. Control of counterdiffusion problems in underwater dry welding. In: International Diving Symposium '80. Gretna, LA: Association of Diving Contractors.
- Peterson RE, Greene K. 1976. Current work at the Institute for Environmental Medicine. In: Hamilton RW, ed. Development of decompression procedures for depths in excess of 400 feet. Report WS 2-28-76. Bethesda, MD: Undersea Medical Society.
- Peterson RE, Hamilton RW. 1987. Development of saturation decompression procedures for nitrogen-oxygen and air habitat diving operations. In: Bove AA, Bachrach AJ, Greenbaum LJ Jr, eds. Underwater and hyperbaric physiology IX. Bethesda, MD: Undersea and Hyperbaric Medical Society.
- Pilmanis AA, ed. 1992 Jun. Proceedings 1990 Hypobaric Decompression Sickness Workshop. AL-SR-1992-0005. Brooks Air Force Base, TX: Air Force Systems Command.
- Rogers R. 1991 Apr-Jun. The development of the recreational dive planner. SPUMS J 21(2):98-106.
- Schmidt TC, Wellborn M, November S. 1997 Mar. Provisional decompression procedures for pressurized submarine rescue using elevated atmospheric PO₂ levels and mask oxygen breathing. Report #RV-R-2453, Rev. A. Contract N00024-93-C-6100 to Lockheed Martin, San Diego. Arlington, VA: Naval Sea Systems Command, Department of the Navy.
- Schreiner HR, Hamilton RW, eds. 1989 May. Validation of decompression tables. UHMS 74(VAL)1-1-88. Bethesda, MD: Undersea Hyperbaric Medical Society.
- Schreiner HR, Kelley PL. 1971. A pragmatic view of decompression. In: Lambertsen CJ, ed. Underwater Physiology IV. New York: Academic Press.
- Sheffield PJ, ed. 1989 Dec. Flying after diving. 77(FLYDIV)12/1/89. Bethesda, MD: Undersea Hyperbaric Med Society.
- Shilling CW, Werts MF, Schandelmeier NR, eds. 1976. The underwater handbook: A guide to physiology and performance for the engineer. New York: Plenum Press.

- Sterk W. 1986. Intermittent hyperoxia in operational diving: What are the safe limits. In: Schrier LM, de Jong MH, Sterk W, eds. Proceedings of the XIIth Annual Meeting of the European Undersea Medical Society. Rotterdam: Foundation EUBS. Pp. 55-64.
- Stubbs RA, Kidd DJ. 1965a. 65-RD-1. Downsview, ON: Canadian Forces Institute of Aviation Medicine.
- Stubbs RA. Kidd DJ. 1965b. 65-RD-8. Downsview, ON: Canadian Forces Institute of Aviation Medicine.
- Takashima R, Sterk W, Nashimoto I, Mochizuki T. 1996. Use of trimix breathing in deep caisson work for the construction of the Meiko West Bridge. Undersea & Hyperbaric Medicine 23(Supplement):34.
- Thalmann ED. 1983 Jan. Computer algorithms used in computing the Mk 15/16 constant 0.7 ATA oxygen partial pressure decompression tables. NEDU Rept. 1-83. Panama City, FL: US Navy Experimental Diving Unit.
- Thalmann ED. 1984 Jan. Phase II testing of decompression algorithms for use in the US Navy underwater decompression computer. NEDU Report 1-84. Panama City, FL: US Navy Experimental Diving Unit.
- Thalmann ED. 1985 Apr. Development of a decompression algorithm for constant 0.7 ATA oxygen partial pressure in helium diving. NEDU Report 1-85. Panama City, FL: US Navy Experimental Diving Unit.
- Thalmann ED. 1986 Aug. Air-N₂O₂ decompression computer algorithm development. NEDU Report 8-85. Panama City, FL: US Navy Experimental Diving Unit.
- Thalmann ED, Buckingham IP, Spaur WH. 1980 Aug. Testing of decompression algorithms for use in the US Navy underwater decompression computer: Phase I. Report 11-80. Panama City, FL: US Navy Experimental Diving Unit.
- Tikuisis P, Nishi RY, Weathersby PK. 1988 Jul. Use of the maximum likelihood method in the analysis of chamber air dives. Undersea Biomed Res 15(4):301-313.
- US Department of the Navy. 1993 Feb. US Navy Diving Manual. Volume 1 (Air Diving) Revision 3. 0994-LP-001-9110. Washington: Navy Department. [Supersedes 15 Dec 1988 NAVSEA 0994-LP 9010, Rev 2.]
- US Navy Diving Manual. 1995 Jun. NAVSEA 0994-LP-001-9010. Volume I. Rev. 1. Washington: Navy Department.
- Vann RD. 1984. Decompression from saturation dives. In: Cox FE, ed. Proceedings 3rd Annual Canadian Ocean Technology Congress. Toronto: Underwater Canada.
- Vann RD. 1988 May. Oxygen toxicity risk assessment. Final report under ONR Contract N00014-87-C-0283. Durham, NC: FG Hall Laboratory, Duke University.
- Vann RD, editor. 1989. The physiological basis of decompression. 75(Phys)6/1/89. Bethesda, MD: Undersea and Hyperbaric Medical Society.

- Weathersby PK, Homer LD, Flynn ET. 1984. On the likelihood of decompression sickness. J Appl Physiol 57(3):815-825.
- Weathersby PK, Survashi SS, Hays JR, MacCallum ME. 1986 Jul. Statistically based decompression tables III: Comparative risk using U.S. Navy, British, and Canadian standard air schedules. NMRI 86-50. Bethesda, MD: Naval Medical Research Inst.

Wienke BR. 1993. Diving above sea level. Flagstaff, AZ: Best Publishing Co.

- Wright WB. 1972. Use of the University of Pennsylvania, Institute for Environmental Medicine procedure for calculation of cumulative pulmonary oxygen toxicity. Report 2-72. Washington: US Navy Experimental Diving Unit.
- Yount DE, Hoffman DC. 1986. On the use of a bubble formation model to calculate diving tables. Aviat Space Environ Med 57:149-56.