BUBBBBB DECOMPRESSION

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Background, Theory, and Application

INTRODUCTION

Unlike abstract frontiers of knowledge, the boundary between the known and unknown is clearly visible underwater-often beginning near 130ft (40m). In addition to exploring this edge, technical divers also extrapolate beyond the known with their decompression practice. Examples include the use of exotic ascent gasses (such as argox and neox) and the advent of modern rebreathers (with the attendant possibilities for gas mixtures and durations at depth). In the face of change, it is important to pause and critically examine decompression methods before conducting hyperbaric experiments on yourself.

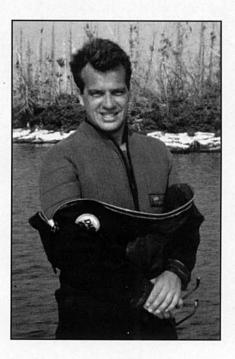
Most civilian decompression

meters, software, and tables are derivatives of neo-Haldane calculation methods, often modeled after Buhlmann's work. Ascents are controlled by limiting tissue supersaturation in a set of gas-loaded compartments (hypothetical tissues). In the laboratory, the current focus in decompression modeling involves formulating ascent schedules that report the statistical confidence in a stated probability of an incidence of decompression illness (DCI) (eg: they might state that you can be 95% sure that the given profile will have a bends incidence of 2%). Statistical models form reliable predictions within the "known." However, they cannot be used to extrapolate to untested regions (new combinations of depths and bottom times, gas mixtures and switches, etc.) This is both a strength and a weakness, for only by grounding tables in experimental data can a meaningful definition of what is "known" be made. Yet, senior researchers insist that they are not confident that they even have a handle on air diving-let alone the use of multiple inert gases. It will be a long time before decompression calculation methods of universal validity become available. A fundamental, first-

f universal validity become available. A fundamental, first-principles model may never be developed. Meanwhile, divers will go deeper for longer durations, using whatever map of the territory is

available.

Any diver who has been routinely monitored with a



Doppler meter can tell of times they bubbled—even after following a conservative ascent schedule. As unnerving as the experience is, the first thing that you wonder after hearing low-grade bubbles is "why don't I feel bent?" The answer may be that the body has the ability to handle small amounts of bubbles without undue stress. There is substantial evidence that the body's tissues contain cavities and bubble nuclei before making a dive. These preexisting voids are activated into growth with the application and reduction of pressure associated with diving. Bubbles are especially prone to growth when surrounding tissue is heavily loaded with dissolved gas, such as might be expected from long, deep or repetitive dives.

It is possible to adopt decompression strategies to minimize the forma-

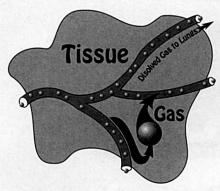


Fig 1. Dissolved gas can diffuse from the tissue into either the circulatory sytem or into bubbles. Gas in circulation is easily eliminated in the lungs. Gas in bubbles can present problems by greatly increasing outgassing times, especially if the diver does not take consistent steps to eliminate the bubbles.

tion and growth of bubbles. While this might be the objective of all decompression schedules, the surprising result is that bubble elimination strategies are often contrary to the recommendations of traditional diving tables and therefore seem counter-intuitive. For instance, decompression stops called for by bubble models are much deeper (often within a few ata of bottom) than corresponding neo-Haldane tables. We will see why this is so when we consider the physics of bubbles and look into the details of some bubble models.

ABBREVIATED HISTORY OF THE **BUBBLE MODELS**

The idea that divers could develop bubbles, yet not display overt symptoms of the bends is nearly as old as the sport. Behnke, in the early 1950s, termed these asymptomatic cases silent bubbles. A decade later, Brian Hills of Australia introduced a method for minimizing the formation and growth of bubbles by advancing the thermodynamic calculation of decompression tables, culminating in the publication of the classic Decompression Sickness in 1977. Therein, he gave clear discussions of concepts such as the inherent unsaturation of tissue (oxygen window) in addition to detailing his method for decompressing divers at zero supersaturation. Hills' tables stipulated initial stops far deeper than the US Navy (USN) profiles. Hills believed that the USN's tables encouraged

formation of bubbles via a long ascent to a relatively shallow first stop. He asserted that the long ten-foot stop served as therapy to reduce bubbles formed by the extreme first pull from the bottom to the first stop.

Though Hills' ideas for decompression methods were firmly based in experiment, experience and theory, the resulting unconventional ascent schedules met with derision (especially from workers at the USN). The introduction of Doppler monitoring techniques in the late 1960s substantiated that divers were in fact bubbling on the USN tables. Although hopes that Doppler might be used as real-time feed-back for decompression haven't materialized, the idea still holds that the prevention of bubbles will minimize risk of DCI.

Following Hills, researchers at the University of Hawaii postulated that a common basis of all DCI might be that the insult initiated in aqueous tissues that comprise the bulk of living creatures. They conducted a series of experiments that resulted in the formulation of the Varying Permeability Model (VPM). As in Hills' model, VPM tables call for deep first stops to keep gas in solution so that it can be eliminated through the circulation rather than flow into bubbles. Wienke, extended the VPM to include repetitive and multi-day diving in the Reduced Gradient Bubble Model3 (RGBM). A major equipment manufacturer slated the RGBM for implementation in diving meters in the early 1990s, though a product was never released.

Currently, a number of modeling efforts incorporate bubble-mechanical principals into the calculation of decompression schedules. A manufacturer (Abyss) of desktop decompression software has contracted with Wienke to implement a version of the RGBM (as of 1996 the full bubble model has not been incorporated). Models under development at Duke, NASA, and commercial diving firms incorporate both bubble mechanics and statistical analysis of DCI data. Technical divers already employ decompression procedures consistent with bubble models ad-hoc in their planning (Ref. Richard Pyle's prescription for deep stops in an

earlier issue of DeepTech). Yet, the justification of these methods is often given in terms of operational concerns and neo-Haldane viewpoints rather than bubble mechanics.

PHYSIOLOGICAL ISSUES

Divers are not passive systems like the inanimate gelatin used in laboratory models for developing the VPM. The ability of the body to react to stimuli and stresses hinders deterministic decompression models. The physiological complexities of this feedback cycle are daunting. For instance, the biochemical reactions to the presence of gas bubbles can compound problems facing a DCI victim by initiating clotting and complement activation. The bubbles themselves can form obstructions in arteries, with severe consequences to the nervous system. On a less urgent level, even high partial pressures of oxygen can trigger responses such as the reduction of blood perfusion. The reduced flow to tissues diminishes outgassing to the circulatory system, resulting in larger tissue tensions, encouraging bubble growth.

A comprehensive approach to building effective decompression tables necessarily requires input from a wide range of specialties. Even if we step back from the goal of building a universal model, it is still possible to make recommendations for decompression procedures that are consistent with minimizing the occurrence and growth of bubbles. This is where the fundamental perspective of physics can provide guidance for avoiding bubble formation and growth. Most importantly, recommendations based an a mechanistic perspective must be weighed against physiological, medical, and operational concerns.

During decompression, there are competing pathways for the flow of gas that has been absorbed by tissues while diving. (FIG 1). The challenge to table designers (and ultimately decompressing divers) is to keep tissue gas in solution while ascending so it can be eliminated by diffusion to the circulation. Assumptions inherent in neo-Haldane decompression models are inappropriate if gas has formed free phases in a diver's body. At best,

Equilibrium: External P = Internal P $P_A+P_c+P_F=PN^2+(PO^2+PCO^2+PH^2O)$

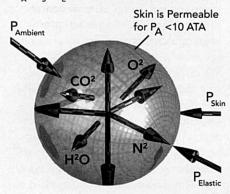


Fig.2 Bubbles will grow or shrink, depending on whether the surrounding tissue tension is greater or smaller than the bubble's internal pressure. In either case, a pressure gradient G = (T - P_b) exists across the skin of the bubble, driving the flow of gas. When G is positive, tissue tension is greater than bubble pressure, leading to bubble growth. G is negative if tissue tension is less than bubble pressure, leading to bubble shrinkage. It is the objective of bubble models to keep G negative (or zero) by appropriately setting ascent stage depths and choosing gas mixtures to discourage growth.

decompression schedules that ignore free phases lead to bubble growth and ineffective gas elimination. For example, consider that DCI victims (who are likely bubbling) are compressed to relieve their symptoms. From this painfully practical example, we see that if a diver bubbles, the quickest way out of the water is to stay deep (pressurized), rather than pull stops as close to the surface as possible as standard decompression methods encourage

BUBBLE PHYSICS

A glass of your favorite carbonated beverage will come in handy for this section. An intuitive understanding of how bubbles work can be gained from observing a glass of cola, champagne or beer. Other models provide further insights that guide us in designing procedures for keeping bubbles as small as possible.

A bubble has an internal pressure that is generally different from the tension of inert gas in surrounding Bubble Grows if T>P_b (P_b=P_{amb}+P_{xs}) $G = (T-P_b) \text{ is } \underline{Positive}$ T = Tissue Tension CAS P_b

Bubble Shrinks if $T < P_b (P_b = P_{amb} + P_{xs})$ $G = (T - P_b) \text{ is } \frac{\text{Negative}}{\text{T = Tissue Tension}}$

Fig.3. A bubble will remain stable in size if pressure balances in and outside of the bubble. In equilibrium, the sum of the external pressures, pressing inward will balance the summed partial pressures of the gases within the bubble. A nitrox diver's bubbles could contain nitrogen, carbon dioxide, oxygen, and water vapor, all of which will diffuse in or out of the bubble independently to balance internal partial pressures with external partial tensions.

tissue. A bubble's size is key to determining whether it will shrink or grow. Bubble size is closely linked to its internal pressure through effects such as skin tension, the elasticity of surrounding tissue, and Boyle's law. The most important factor in minimizing bubble growth is keeping internal bubble partial pressures greater than (or equal to) tissue tensions. This is accomplished by setting stages deep and astutely choosing breathing gases. A common approach in bubble models is to limit the volume of gas freed from solution during ascent rather than set tissue tensions limits (as M values do in neo-Haldane calculations). The conditions under which bubbles grow or shrink (FIG 2) can be studied by

considering what factors affect changes in bubble size (FIG 3).

The mathematical description of how gas flows between tissue and bubble is given by a diffusion equation, which is distinct from the simple rate equations that yield exponential in/out gassing. A conceptual diffusion equation is easy to write down and solve. All we need do is account for those factors that make bubbles grow and those that make them shrink. The rate of change of a bubble radius is proportional to:

A: The surface area of the bubble. As in opening window wider in a breeze, the greater A, the more flow is possible.

D: The diffusivity of gas in tissue.

A measure of how fast gas flows to equalize pressure differences.

S: The solubility of gas in tissue.

The more soluble the gas, the higher the concentration of gas that is available to be absorbed by the bubble from the tissue or visa-versa.

G: The gradient of pressure across the bubble surface.

 $G = T - P_b$, where T is tissue tension and P_b is the pressure inside the bubble. G is positive if tissue tension exceeds bubble partial pressure, causing bubble growth. G is negative if bubble partial pressure exceeds tissue tension causing bubble shrinkage (fig 4). The magnitude of a negative G is essentially a measure of the oxygen window⁴.

And inversely proportional to: p: The density of diffusing gas. V: The volume of the bubble.

The driving force eliminating free phases is opposite from dissolved gas. It is the oxygen window rather than the tissue tension T that controls free phase elimination. To eliminate bubbles, keep G negative by staying as deep as possible to force P_b to be larger than T. Think: G = growth—and negative growth is shrinkage (Fig. 5).

The quickest route out of the water from a neo-Haldane viewpoint is to minimize your depth (within the limits set by M values). An extreme version of this would be to ascend on a dive

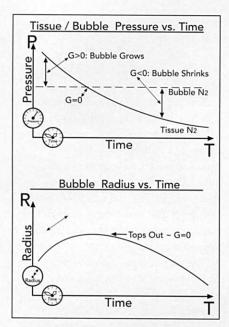


Fig. 4. The two diagrams illustrate how a bubble responds to differences between its internal pressure (red) and the tension of gas dissolved in the surrounding tissue (green). While the tissue tension of gas is greater than the bubble, G is positive, leading to bubble growth. The bubble shrinks once the tissue has off-gassed sufficiently for the tension of the inert gas to fall below the bubble pressure.

computer's ceiling alarm rather than at conventional 10-foot increments. From this viewpoint, a minimum depth causes maximum elimination gradient between gas dissolved in tissues and arterial gas tension. That is, if dissolved N₂ tension T decreases at a rate proportional to the difference between arterial tension

 ppN_2 (= $f_{N2}x(1 + D/33)$) and T, then off-gassing will increase as (ppN_2 - T) becomes more negative.

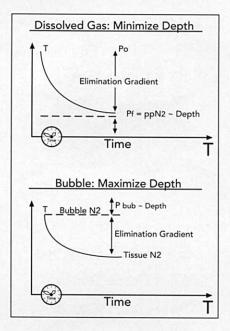


Fig. 5. The optimal strategy for the rapid elimination of inert gas depends on whether the gas is dissolved in tissues or present as a free phase in bubbles.

This occurs as a diver ascends because ppN_2 decreases (upper Fig. 5).

In the case of bubbles, the opposite holds. The most efficient elimination of free phases occurs at maximal depth (lower Fig. 5). The rate of bubble collapse is proportional to G, the gradient between tissue tension and bubble internal pressure: (T - P_b). With bubble internal pressures increasing with depth

 $(P_b = P_{ambient} + skin\&volume effects),$ a negative G occurs if P_b exceeds T. So, we go deep to shrink bubbles.

Reality is in between the two extremes (what goes down must come up). The optimal strategy for inert gas elimination should effectively eliminate both free and dissolved gas. With this aim, it is best to stay at high ppO₂ (within CNS toxicity limits) to encourage elimination of dissolved gas. By simultaneously keeping external pressure maximized, the O² window is open to its fullest. We might well eliminate the 10-foot stop altogether in meeting this goal.

We are now prepared to make some practical recommendations for decompression strategies based in bubble mechanics. This is where part II of this series will pick up the story. There, bubble ideas will be applied to the real water-world by considering practical recommendations that arise naturally from bubble models. Topics up for consideration include:

•The best (though usually ignored) reason for pulling your 10-foot stop at 20 feet on oxygen.

 The superiority of Nitrox 80/20 over Oxygen as a mix for shallow stops.

 The strong (physical) argument in favor of in-water-recompression.

 Inert gas counter-diffusion (why argon suit-inflation gas shouldn't cause problems)

 Bubble amplification due to gas switches at constant pressure.

 Why rapid deep ascents are a bad idea for dives deeper than nine ata.

So there you have the background. Above all, I hope this article encourages critical discussion in the technical diving community regarding current decompression practice. As the popular advertisement (DiveRite) infers, it's your ass on the line.

BIO

The author is not a professional decompression modeler—just a critic. He does some decompression diving, setting deep stops on the basis of bubble models. He has a Ph.D. in Physics and works for the SONY Corporate Research Laboratory in Yokohama, Japan.

