RGBM TECHNICAL UPDATE from PHASE MECHANICS AND DECOMPRESSION THEORY IN DEPTH

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ABSTRACT

Decompression theory and phase mechanics are detailed in a Seven Chapter series, with topics motivated and strategically developed in their relationship to diving. Topics span many disciplines and focus in a number of decompression arenas. Targeted audience is the commercial diver, instructor, hyperbaric technician, underwater researcher, and technical diver looking for greater detail, and especially the doctor, physiologist, physicist, chemist, mathematician, engineer, or biologist by training. Topics include energy and thermodynamics, pressure and density, flow mechanics and gas kinetics, free and dissolved phase transfer, nucleation and cavitation, bubbles and surfactants, mixed gases, statistics, risk and probability, binomial and related distributions, computing and models, and altitude effects. References are included. This monograph extends Basic Decompression Theory And Application, as well as Basic Diving Physics And Application and Technical Diving In Depth. The Appendices house sets of RGBM recreational and technical Tables.

Specifically, we cover a number of linked topics:

1. *basic physics and fundamental concepts;*
2. *basic statistics and risk analysis;*
3. *nucleation and cavitation, persistence, time scales, and metrics;*
4. *seeds, bubbles, equations of state, and material properties;*
5. *energy, thermodynamics, hydrodynamics, and pressure mechanics;*
6. *gas laws, flow dynamics, and phase transfer;*
7. *perfusion and diffusion limited processes;*
8. *critical tensions and phase volumes;*
9. *altitude similarity and protocols;*
10. *mixed gases, oxygen dose, deep stops, and decompression;*
11. *inert gas transport and isobaric counterdiffusion;*
12. *probabilistic decompression, statistical methods, and maximum likelihood;*
13. *staging, validation, and model testing;*
14. *dive tables, meter algorithms, and computational issues.*

Material presentation is phase mechanics first, followed by decompression theory. This facilitates continuity and discussion. New material is woven into previous material, and, as such, is necessary for further and extended development.

Pages – 184, Tables – 34, Figures – 40, References – 167

AUTHOR SKETCH

Bruce Wienke is a Program Manager in the Nuclear Weapons Technology/ Simulation And Computing Office at the Los Alamos National Laboratory (LANL), with interests in computational decompression and models, gas transport, and phase mechanics. He contributes to underwater symposia, educational publications, technical periodicals and decompression workshops, having authored seven monographs *(Technical Diving In Depth, Decompression Theory, Physics, Physiology And Decompression Theory For The Technical And Commercial Diver, High Altitude Diving, Basic Diving Physics And Application, Diving Above Sea Level, Basic Decompression Theory And Application)* and some 200 technical journal articles. Diving environs include the Caribbean, South Pacific, Asia, inland and coastal United States, Hawaii, and polar Arctic and Antarctic for sundry technical, scientific, military, and recreational activities. He functions on the LANL Nuclear Emergency Strategy Team (NEST), in exercises often involving Special Operations (SEAL, Delta), above and below water, and leads the Nuclear Countermeasures Dive Team. He started and heads Southwest Enterprises, a consulting company for research and applications in overlapping areas of applied science and simulation, contracts as an Expert Witness in diving litigation, and served SEAL

Wienke is an Instructor Trainer/Technical Instructor with the National Association Of Underwater Instructors (NAUI), serves on the Board Of Directors (Vice Chairman for Technical Diving, Technical and Decompression Review Board Member), is a Master Instructor with the Professional Association Of Diving Instructors (PADI) in various capacities (Instructor Review Committee), is an Institute Director with the YMCA, and is an Instructor Trainer/Technical Instructor with Scuba Diving International/Technical Diving International (SDI/TDI). Wintertime he hobbies skiing, coaching, and teaching as a Racing Coach and Instructor, certified United States Ski Coaches Association (USSCA) and Professional Ski Instructors of America (PSIA), and races in the United States Ski Association (USSA) Masters Series Competition, holding a 8 NASTAR racing handicap. Other interests include tennis, windsurfing, and mountain biking. He quarterbacked the 63 Northern Michigan Wildcats to an NCAA II Championship (Hickory Bowl).

Wienke received a BS in physics and mathematics from Northern Michigan University, MS in nuclear physics from Marquette University, and PhD in particle physics from Northwestern University. He belongs to the American Physical Society (APS), American Nuclear Society (ANS), Society Of Industrial And Applied Mathematics (SIAM), South Pacific Underwater Medical Society (SPUMS), Undersea And Hyperbaric Medical Society (UHMS), and American Academy Of Underwater Sciences (AAUS). He is a Fellow of the American Physical Society, and a Technical Committee Member of the American Nuclear Society.

Wienke, a former dive shop owner in Santa Fe, presently serves as a Consultant for decompression algorithms in the Industry. He has worked with DAN on applications of high performance computing and communications to diving, and is a Regional Data Coordinator for Project Dive Exploration. Scubapro, Suunto, Mares, Dacor, HydroSpace, Plexus, Abysmal Diving, and Atomic Aquatics engage him (or have) as Consultant for meter algorithms. He is the developer of the Reduced Gradient Bubble Model (RGBM), a dual phase approach to staging diver ascents over an extended range of diving applications (altitude, nonstop, decompression, multiday, repetitive, multilevel, mixed gas, and saturation). A number of dive computers (Suunto, Mares, Dacor, Plexus, HydroSpace, and others coming online) incorporate the modified and full iterative RGBM into staging regimens, for technical and recreational diving. Aggressive computers with RGBM for helitrox, trimix, heliox, nitrox, air, and combinations are in the pipeline. ABYSS, a commercial software product, features some of the RGBM dynamical diving algorithms developed by him for Internet users and technical divers. He is also Associate Editor for the International Journal Of Aquatic Research And Education, and is a former Contributing Editor of *Sources*, the NAUI Training Publication. NAUI Technical Training has adopted the RGBM for technical and recreational training, and employs RGBM trimix, heliair, nitrox, and air tables. Wienke is a Contributing Editor of *Advanced Diver* magazine.

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Quiescent Nucleation

Henry's law tells us that a gas will tend to separate from solution (pass from the dissolved state to the free state) if the tension of the gas in the dissolved state exceeds its partial pressure in the adjacent free state. And the opposite holds true if the gradient is reversed. Phase separation can be delayed if some remnant of a free phase does not already exist in the liquid, providing a pathway for the dissolved gas to *dump* over into the free state, rendering the dissolved gas *metastable* during the delay. The challenge in tracking phase separation is the presence and quantification of free phase precursors, or seeds, that facilitate gas transfer in a process called *nucleation*.

Metastable states are unstable thermodynamic states lying close to stable configurations, that is, separated by relatively small energy differences. A substance in a metastable state will eventually transition into a stable state. For instance, a supercooled vapor will eventually condense into a liquid, a supercooled liquid will eventually become solid, and a superheated liquid will eventually evaporate into a gas. Bubble formation can be a process in which a gas, or vapor, phase is initially formed from a metastable liquid environment, one that is usually supersaturated with dissolved gas.

Metastable phase transitions deposit an unstable phase onto a stable phase, with aggregates in the stable phase serving as *nuclei* for the transition. Liquid drops in a supercooled vapor, if sufficiently large, become centers of condensation of the vapor, for example. Nuclei will form in both phases because of statistical fluctuations, but the nuclei in the metastable phase will disappear in time, while those in the stable phase will remain. Such nuclei form statistically as a result of thermal fluctuations in the interior of the media, with a certain (small) number reaching *critical* radius for growth. If large enough, nuclei in the stable phase seed the continuing process of phase transitions from the metastable state. For each metastable state, there is a minimum size which nuclei in the stable phase must possess to afford more stability than the metastable state. This size is called the critical radius, r_c. Nuclei smaller than the critical radius will not support phase transitions from the metastable state, and will also disappear in time. In assigning a critical radius to nuclei, spherical aggregate symmetry is assumed, and is requisite to minimize surface energy.

Homogeneous nucleation processes occur in single component systems, while heterogeneous nucleation processes involve more than one component. To describe nucleation, a heterogeneous model, ascribed to Plesset, containing the homogeneous case as a subset, has been useful in applications. A solid hydrophobic sphere, of radius r_0, is surrounded by a concentric layer of vapor, out to a radius r. The instantaneous (Boltzmann) probability, dw, for the state depends on the difference in free energy, ζ, associated with the vapor phase,

$$dw = exp\ (-\zeta/kT)\ dG \quad ,$$

at temperature, T, for (Gibbs) free energy change, ζ,

$$\zeta = \frac{4}{3}\pi r^2 \gamma_{lv} + \frac{4}{3}\pi r_0^2\ (\gamma_{vs} - \gamma_{ls}) \quad ,$$

and γ_{lv}, γ_{vs}, and γ_{ls} surface tensions associated with the liquid-vapor, vapor-solid, and liquid-solid interfaces. The homogeneous case corresponds to $r_0 = 0$, that is, no solid and only liquid-vapor nucleation.

Tribonucleation

Cavitation

Simply, *cavitation* is the process of vapor phase formation of a liquid when pressure is reduced. A liquid cavitates when vapor bubbles are formed and observed to grow as consequence of pressure reduction. When the phase transition results from pressure change in hydrodynamic flow, a two phase stream consisting of vapor and liquid results, called a cavitating flow. The addition of heat, or heat transfer in a fluid, may also produce cavitation nuclei in the process called boiling. From the physico-chemical perspective, cavitation by pressure reduction and cavitation by heat addition represent the same phenomena, vapor formation and bubble growth in the presence of seed nuclei. Depending on the rate and magnitude of pressure reduction, a bubble may grow slowly or rapidly. A bubble that grows very rapidly (explosively) contains the vapor phase of the liquid mostly, because the diffusion time is too short for any significant increase in entrained gas volume. The process is called vaporous cavitation, and depends on evaporation of liquid into the bubble. A bubble may also grow more slowly by diffusion of gas into the nucleus, and contain mostly a gas component. In this case, the liquid degasses in what is called gaseous cavitation, the mode observed in the application of ultrasound signals to the liquid. For vaporous cavitation to occur, pressure drops below vapor pressure are requisite. For gaseous cavitation to occur, pressure drops may be less than, or greater than, vapor pressure, depending on nuclei size and degree of liquid saturation. In supersaturated ocean surfaces, for instance, vaporous cavitation occurs very nearly vapor pressure, while gaseous cavitation occurs above vapor pressure.

In gaseous cavitation processes, the inception of growth in nuclei depends little on the duration of the pressure reduction, but the maximum size of the bubble produced does depend upon the time of pressure reduction. In most applications, the maximum size depends only slightly on the initial size of the seed nucleus. Under vaporous cavitation, the maximum size of the bubble produced is essentially independent of the dissolved gas content of the liquid. This obviously suggests different cavitation mechanisms for pressure (reduction) related bubble trauma in diving. Slowly developing bubble problems, such as limb bends many hours after exposure, might be linked to gaseous cavitation mechanisms, while rapid bubble problems, like central nervous system hits and and embolism immediately after surfacing, might link to vaporous cavitation.

Gas Turbulent Nucleation

Chemical Nucleation

Ensemble Theory

PHASE MECHANICS AND DECOMPRESSION THEORY IN DEPTH
CHAPTER 2: MATERIAL PROPERTIES

Gases

Air is a mixture of inert and metabolic gases, composed of hydrogen and oxygen mainly, with variable amounts of carbon dioxide, water vapor, ozone, sulfur dioxide, and nitrogen dioxide, and fixed trace amounts of xenon, helium, krypton, argon, methane, nitrous oxide, hydrogen, and neon.

By volume, air is 78.1% nitrogen, 20.9% oxygen, and 1% everything else. Over nominal pressure and temperature ranges encountered in the Earth's atmosphere, air can be treated as an *ideal*, or dilute, gas.

Solids

Fluids

Compressibility And Cubical Expansion

Time Scales

We know from Doppler measurements in the body and laboratory experiments with bubbles that micronuclei and bubbles have finite lifetimes, ranging from minutes to hours. Seeds and bubbles are transients in all environments, but with virtually intractable time evolution in the body.

Bubble Metrics

Certainly we do not know the exact physical properties of gas seeds and bubbles in the body, but we can make some general comments based on known equation of state relationships. Phenomenological relationships fitted from laboratory experiments are also of interest.

Material Response

Under changes in ambient pressure (and temperature), bubbles will grow or contract, both due to dissolved gas diffusion and Boyle's law. An *ideal* change under Boyle's law is symbolically written. Denoting initial and final pressures and volumes with subscripts, i and f, we have,

$$P_i V_i = P_f V_f$$

with bubble volume,

$$V = \frac{4}{3}\pi r^3$$

for r the bubble radius. The above supposes totally flexible (almost ideal gas) bubble films or skins on the inside, certainly not unrealistic for thin skin bubbles. Similarly, if the response to small incremental pressure changes of the bubble skins is a smooth and slowly varying function, the above is also true in low order. Obviously, the relationship reduces to,

$$P_i r_i^3 = P_f r_f^3$$

for a ideal radial response to pressure change.

But for real structured, molecular membranes, capable of offsetting constrictive surface tension, the response to Boyle's law is modified, and can be cast in terms of Boyle modifiers, ξ,

$$\xi_i P_i V_i = \xi_f P_f V_f$$

with ξ virial functions depending on P, V, and T. For thin and elastic bubble skins, $\xi = 1$. For all else, $\xi \neq 1$. For gels studied in the laboratory, as an instance, surfactant stabilized micronuclei do not behave like ideal gas seeds with thin elastic films. Instead under compression-decompression,

their behavior is always less than ideal. That is to say, volume changes under compression or decompression are always less than computed by Boyle's law, similar to the response of a wetsuit, sponge, tissue bed, or lung membrane. The growth or contraction of seeds according to an EOS is more complex than Boyle's law. The virial expansions has for all P, T, V and mole fractions, n, for R the universal gas constant,

$$PV = nRT \sum_{i=0}^{N} \alpha_i \left[\frac{nT}{V}\right]^i$$

or, treating the virial expansion as a Boyle modifier, ξ,

$$\xi PV = nRT$$

across data points and regions. Symbolically, the radius, r, can be cast,

$$r = \sum_{i=0}^{N} \beta_i \left[\frac{nRT}{P}\right]^{i/3}$$

or, again introducing Boyle modifiers, ζ,

$$\zeta r = \left[\frac{nRT}{P}\right]^{1/3}$$

for α and β standard virial constants. Obviously, the virial modifiers, ξ and ζ are the inverses of the virial sum expansions as power series. For small deviations from thin film bubble structures, both are close to one.

Permeability Response

Observationally, though, the parameterization can take a different tack. In gel experiments, the EOS is replaced by two regions, the permeable (simple gas diffusion across the bubble interface) and impermeable (rather restricted gas diffusion across the bubble interface). In the permeable region, seeds act like thin film bubbles for gas transfer. In the impermeable region, seeds might be likened to beebees. An EOS of course can recover this response in both limits.

Accordingly, just in gels, the corresponding change in critical radius, r, following compression, $(P - P_i)$, in the *permeable* region, satisfies a relationship,

$$(P - P_i) = 2(\gamma_c - \gamma)\left[\frac{1}{r} - \frac{1}{r_i}\right]$$

with γ_c maximum compressional strength of the surfactant skin, γ the surface tension, and r_i the critical radius at P_i. When P exceeds the structure breakpoint, P_c, an equation for the *impermeable* region must be used. For crushing pressure differential, $(P - P_i)_c = P - P_c$, the gel model requires,

$$(P - P_i)_c = 2(\gamma_c - \gamma)\left[\frac{1}{r} - \frac{1}{r_c}\right] + P_c + 2P_i + P_i\left[\frac{r_c}{r}\right]^3$$

where,

$$r_c = \left[\frac{P_c - P_i}{2(\gamma_c - \gamma)} + \frac{1}{r_i}\right]^{-1}$$

is the radius of the critical nucleus at the onset of impermeability, obtained by replacing P and r with P_c and r_c above.

The allowed tissue supersaturation, $\Delta\Pi$, is given by,

$$\Delta\Pi = 2\frac{\gamma}{\gamma_c r}(\gamma_c - \gamma)$$

with, in the permeable region,

$$r = \left[\frac{(P - P_i)}{2(\gamma_c - \gamma)} + \frac{1}{r_i} \right]^{-1}$$

and, in the impermeable region,

$$r^3 - 2(\gamma_c - \gamma)r^2 - \frac{P_i}{\zeta} r_c^3 = 0$$

for,

$$\zeta = (P - P_i)_c - P_c + 2P_i + \frac{2(\gamma_c - \gamma)}{r_c}$$

Thus, allowed supersaturation is a function of three parameters, γ, γ_c, and r_i. They can be fitted to exposures and lab data. But Boyle expansion or contraction needs be applied ad hoc to the excited seeds. Additionally, nuclei regenerate over times scales, ω, such that,

$$r = r_0 + [1 - \exp\ (-\omega t)](r_i - r_0)$$

with r_0. the critical radius at initial time ($t = 0$). The fourth parameter, ω^{-1}, is on the order of many days (Chapter 4).

Discontinuities in types of materials and/or densities at surfaces and interfaces give rise to interfacial forces, called *surface tension*. Discontinuities in density produce cohesive gradients tending to diminish density at the surface region. At the interfaces between immiscible materials, cohesive forces produce surface tension, but adhesional forces between dissimilar materials tend to offset (decrease) the interfacial tension. Surface and interfacial tension are readily observed in fluids, but less readily in solids. In solids, very little stretching of the surface region can occur if the solids are rigid. Upon heating rigid solids to higher temperature, surface tension becomes a discernible effect.

Any two phases in equilibrium are separated by a surface of contact, the existence of which also produces surface tension. The thin contact region is a transition layer, sometimes called the $film$ layer. Phases can be solid, liquid, or vapor, with surface tension in each case different. The actual position, or displacement, of the phase boundary may alter the area of the phases on either side, leading to pressure differences in the phases. The difference between phase pressures is known as the surface, or film, pressure. The phase equilibration condition requires the temperatures and chemical potentials (Gibbs free energy) of phases be equal, but certainly not the pressures.

PHASE MECHANICS AND DECOMPRESSION THEORY IN DEPTH
CHAPTER 4: CRITICAL TENSIONS AND PHASE VOLUMES

Critical Phase Volumes

Another way to limit diving through critical parameters occurs through phase volume limits, often integral constraints across the full pressure schedule. A couple of approaches are plausible, and require tuning and correlations with actual diving exposure data. Consider the Wienke, Yount, and Hennessy approaches, that is, starting with the most recent analyses.

Reduced Gradient Bubble Model

A complete approach to imposing phase volume limits, incorporating both gas diffusion across tissue-bubble interfaces and Boyle expansion-contraction is used in the full blown reduced gradient bubble model of Wienke. The phase volume constraint equation is rewritten in terms of a phase function, $\dot{\phi}$, varying in time,

$$\int_0^\tau \frac{\partial \phi}{\partial t} dt \ \leq \ \Phi$$

with, simplifying notation,

$$\dot{\phi} = \frac{\partial \phi}{\partial t}$$

for Φ the separated phase, and τ some (long) cutoff time. Specifically, for Π total gas tension,

$$\dot{\phi} = \left[\frac{\partial V}{\partial t}\right]_{diffusion} + \left[\frac{\partial V}{\partial t}\right]_{Boyle} + \left[\frac{\partial V}{\partial t}\right]_{excitation}$$

for,

$$\left[\frac{\partial V}{\partial t}\right]_{diffusion} = 4\pi DS \int_r^\infty nr \left(\Pi - P - \frac{2\gamma}{r}\right) dr$$

$$\left[\frac{\partial V}{\partial t}\right]_{Boyle} = \int_r^\infty n \left(\frac{1}{P}\frac{\partial (PV)}{\partial t}\right) dr$$

$$\left[\frac{\partial V}{\partial t}\right]_{excitation} = \frac{\partial}{\partial t}\left(4\pi \int_0^\infty nr^2 dr\right)$$

with all quantities as denoted previously, and the bubble number integrand normalized,

$$\int_0^\infty n dr = 1$$

The temporal phase function, $\dot{\phi}$, depends on number of bubbles, n, stimulated into growth by compression-decompression, the supersaturation gradient, G, seed expansion-contraction by radial diffusion, $\partial r/\partial t$, Boyle expansion-contraction, PV, under pressure changes, and temperature, T, in general. The excitation radius, r, depends on the material properties, and is given for air (μm),

$$r = 0.003929 + 0.001467 \left[\frac{T}{P}\right]^{1/3} + 0.021183 \left[\frac{T}{P}\right]^{2/3}$$

with ranges for virial coefficients, aqueous to lipid materials, varying by factors of 0.75 to 4.86 the values listed above. Values of the excitation radii, r, above range from 0.01 to 0.05 μm for sea level down to 500 fsw. This is compared to excitation radii in other models (VPM and TBDM) which vary in the 1 μm range. Values for pure helium and nitrogen are recounted later. And the air expression above represents a good RGBM fit to exposure data across lipid and aqueous representations.

The phase integral for multiexposures is written, for any number of J dives, or dive segments,

$$\sum_{j=1}^{J} \left[\dot{\phi}\ t_{d_j} + \int_0^{t_j} \dot{\phi} dt\right] \leq \Phi$$

with the index j denoting each dive segment, up to a total of J, and t_j the surface interval after the j^{th} segment. For the inequality to hold, that is, for the sum of all growth rate terms to total less than Φ, obviously each term must be less the Φ. Assuming that $t_J \to \infty$, gives,

$$\sum_{j=1}^{J-1} \left[\dot{\phi}\ \left[t_{d_j} + \lambda^{-1} - \lambda^{-1} exp\ (-\lambda t_j)\right]\right] + \dot{\phi}\ (t_{d_J} + \lambda^{-1}) \leq \Phi.$$

Defining $\dot{\phi}_j$,

$$\dot{\phi}_j\ (t_{d_j} + \lambda^{-1}) = \dot{\phi}\ (t_{d_j} + \lambda^{-1}) - \dot{\phi}\lambda^{-1} exp\ (-\lambda t_{j-1})$$

for $j = 2$ to J, and,

$$\dot{\phi}_1 = \dot{\phi}$$

for $j = 1$, it follows that

$$\sum_{j=1}^{J} \dot{\phi}_j \ (t_{d_j} + \lambda^{-1}) \leq \Phi$$

with the important property,

$$\dot{\phi}_j \leq \dot{\phi}.$$

This implies we employ reduced phase functions extracted from bounce phase functions by writing,

$$\dot{\phi}_j = \xi_j \dot{\phi}$$

with ξ_j a *multidiving* fraction requisitely satisfying,

$$0 \leq \xi_j \leq 1$$

so that, as needed,

$$\dot{\phi}_j \leq \dot{\phi}.$$

The fractions, ξ, applied to $\dot{\phi}$ always reduce them. As time and repetitive frequency increase, the body's ability to eliminate load bubbles and nuclei decreases, so that we restrict the permissible bubble load in time by writing,

$$\dot{\phi}(t_{j-1}^{cum}) = N\beta r_i \left[1 - \frac{r(t_{j-1}^{cum})}{r_i}\right] = \dot{\phi} \ exp \ (-\lambda_r t_{j-1}^{cum})$$

$$t_{j-1}^{cum} = \sum_{i=1}^{j-1} \ t_i$$

with t_{j-1}^{cum} cumulative dive time. A reduction factor, η_j^{rg}, accounting for creation of new micronuclei is taken to be the ratio of present load over initial load, written,

$$\eta_j^{rg} = \frac{\dot{\phi}(t_{j-1}^{cum})}{\dot{\phi}} = exp \ (-\lambda_r t_{j-1}^{cum})$$

For reverse profile diving, the phase function is restricted by the ratio (minimum value) of the bubble load on the present segment to the bubble load at the deepest point over segments. The phase function reduction, η_j^{ex}, is then written,

$$\eta_j^{ex} = \frac{(\dot{\phi})_{max}}{(\dot{\phi})_j} = \frac{(rP)_{max}}{(rP)_j}$$

with rP the product of the appropriate excitation radius and pressure. Because bubble elimination periods are shortened over repetitive dives, compared to intervals for bounce dives, the phase function reduction, η_j^{rp}, is proportional to the difference between maximum and actual surface bubble growth rate, that is,

$$\eta_j^{rp} = 1 - \left[1 - \frac{\dot{\phi}^{min}}{\dot{\phi}}\right] exp \ (-\lambda_m t_{j-1})$$

with t_{j-1} consecutive total dive time, λ_m^{-1} on the order of an hour, and $\dot{\phi}^{min}$ the smallest $\dot{\phi}$.

Finally, for multidiving, the phase function reduction factor, ξ, is defined by the product of the three η,

$$\xi_j = \eta_j^{ex} \eta_j^{rp} \eta_j^{rg} = \frac{(\dot{\phi})_{max}}{(\dot{\phi})_j} \left[1 - \left(1 - \frac{\dot{\phi}^{min}}{\dot{\phi}}\right) \ exp \ (-\lambda_m t_{j-1})\right] \ exp \ (-\lambda_r t_{j-1}^{cum})$$

with t_{j-1} consecutive dive time, and t_{j-1}^{cum} cumulative dive time, as noted. Since bubble numbers increase with depth, reduction in permissible phase function is commensurate. Multiday diving is mostly impacted by λ_r, while repetitive diving mostly by λ_m.

Varying Permeability Model

The rate at which gas builds up in tissue depends upon both excess bubble number, Λ, and supersaturation gradient, G. The critical volume hypothesis requires that the integral of the product of the two must always remain less than some limit point, $\alpha\ V$, with α a proportionality constant. Accordingly this suggests for Yount, and his associated varying permeability model,

$$\int_0^\infty \Lambda G dt \leq \alpha V \quad ,$$

for bubble number excess, Λ, an approximately linear function of excitation seed radius (difference) on compression-decompression, ΔP,

$$\Lambda = N\beta(r_i - r)$$

with N, β seed constants, r_i, r seed sizes (Chapter 1, Table 1), and V the limiting gas volume. Assuming that tissue gas gradients are constant during compression-decompression, t_d, while decaying exponentially to zero afterwards, and taking limiting condition of the equal sign, yields for a bounce dive,

$$\Lambda G(t_d + \lambda^{-1}) = \alpha V \quad .$$

For compression-decompression, the excitation radius, r, follows from micronuclei growth experiments in gels, but not necessarily in tissue, and assuming equal supersaturation for sets of excitation radii,

$$\frac{2(\gamma_c - \gamma)}{r} - P = \frac{2(\gamma_c - \gamma)}{r_i} - P_i$$

where r and r_i are excitation radii at P and P_i, (Chapter 1, Table 1), are purely phenomenological, and based on laboratory observations and experiments in gels (only).

No accounting of gas transfer across bubbles films, nor Boyle expansion and contraction, enters the Yount (VPM) approach. But Boyle effects might be tracked using appropriate equations-of-state for the seed surfactants (many molecular layers of internal seed coatings). Assigning equations-of-state (EOS) to the lipid and aqueous substances forming the seed surfactants, we have more generally,

$$2(\gamma_c - \gamma) = 135.3 \left[\frac{P}{T}\right]^{1/4} + 73.6 \left[\frac{P}{T}\right]^{1/2} - 15.9 \left[\frac{P}{T}\right]^{3/4}$$

so, a virial expansion of the tension EOS suggests,

$$\frac{2(\gamma_c - \gamma)}{r} - P = \frac{2(\gamma_c - \gamma)_i}{r_i} - P_i$$

At sea level, Yount fits to gel data suggest that $r_i = 0.80\ \mu m$ for air. Of course, if Boyle expansion and bubble gas diffusion were treated in the VPM, the fits to the data would probably start at much smaller excitation radii, r, as in the RGBM, and such would be correspondingly reflected in r_i. Above, $r \leq r_i$, as, $P \geq P_i$, that is, smaller seeds grow on decompression.

With all exposures, the integral must be evaluated iteratively over component decompression stages, maximizing each G while satisfing the constraint equation. In the latter case, t_d is the sum of individual stage times plus interstage ascent times, assuming the same interstage ascent speed, v. Employing the above iteratively, and one more constant, δ, defined by,

$$\delta = \frac{\gamma_c \alpha V}{\gamma \beta r_i N} = 7500\ fsw\ min \quad ,$$

we have,

$$\left[1 - \frac{r}{r_i}\right] G(t_d + \lambda^{-1}) = \delta \frac{\gamma}{\gamma_c} = 522.3 \ fsw \ min \quad ,$$

from the Spencer bounce and Tektite saturation data.

Separated Phase Model

Before dual phase models, such as the two above, came online, Hennessy and Hempleman looked at the critical phase volume concept in a different manner, assuming a certain volume of separated gas, V, remained in equilibrium with all dissolved gases.

And it goes like this. Suppose a unit volume of tissue, V, is equilibrated with an inert gas at partial pressure, p, and ambient pressure, P. After rapid decompression to ambient pressure, Q, assuming that V is formed and filled by free phases, and that no gas is lost through blood nor tissues, and assuming that the partial pressure of the dissolved gas in the bends tissue, q, remains at the threshold for DCS, a simple mass balance requires,

$$Sp = Sq + Vq$$

with S the solubility of the inert gases. Hydrostatic equilibrium in the gas cavity, V, also requires,

$$q + \Pi = Q + \frac{2\gamma}{r} + \delta$$

for Π the sum of all gases (free) in the pocket (approximately constant), γ the surface tension, and δ the tissue deformation pressure in the pocket of radius r.

The above can be conveniently written

$$q = Q + \kappa$$

with κ a constant for a given tissue and released gas volume distribution. Eliminating q,

$$p = \left[1 + \frac{V}{S}\right] (Q + \kappa)$$

If the mixture is breathed at constant oxygen partial pressure, p_{O_2},

$$p = P - p_{O_2}$$

while if oxygen is a constant proportion, f, of the mixture,

$$p = fP$$

In both cases,

$$P = AQ + B$$

with, specifically for the constant oxygen case,

$$A = 1 + \frac{V}{S}$$

$$B = A\kappa + p_{O_2}$$

and for the constant oxygen proportion case,

$$A = \left[1 + \frac{V}{S}\right] f^{-1}$$

$$B = A\kappa$$

The critical pressure ratio, R, is the usual,

$$R = \frac{P}{Q}$$

For the US Navy Tables (240 minute compartment), $A = 1.375$, $B = 5.2\ fsw$, and for Swiss Tables (240 minute compartment), $A = 1.401$, $B = 4.7\ fsw$, while for the lipid and aqueous estimates (olive oil and water) of Hennessy and Hempleman, $A = 1.361$, $B = 3.4\ fsw$ and $A = 1.604$, $B = 4.0\ fsw$, respectively.

The above recovers a standard (M-value straightline) representation in the hyperbaric pressure regime, but not the asymptotically correct zero pressure intercept of the hypobaric regime (as we know it today). The approach is dissolved gas based, with no accounting of the microscopic features of bubble dynamics, and with those dynamics essentially buried in the constants, A and B.

The phase volume constants, Φ, αV, and V, in the above serve as limit points for staging diver ascents, replacing the critical tension M-values as limiting parameters. Imbedded in the first two are bubble dynamics which dramatically alter the staging regimens of all (just) dissolved gas schedules, as mostly imbedded in the third. The Hennessy model however was pivotal to modern decompression theory, helping to underscore the importance of bubble dynamics in staging divers.

Reduced Haldane Gradients

Within the Haldane framework of critical tensions, M, it is possible to fold phase volume constraints over M for multidiving exposures, thereby incorporating some bubble mechanics into time dependent definitions of critical tensions, M, or critical gradients, G. One set of Haldane gradients, G, appears in Table 2 below, and the gradient representation, G, of the usual form, is the starting point,

$$G = G_0 + \Delta G d$$

at depth, d. The set is routinely extracted from the Spencer nonstop limits (NDLs), and the approach is useful in decompression meters with existing Haldane algorithms and software, needing to properly limit diving with phase mechanics, but not able to process full blown phase models and associated physics.

Table 2. Spencer Critical Gradients.

halftime $\tau\ (min)$	threshold depth $\delta\ (fsw)$	surface gradient $G_0\ (fsw)$	gradient change ΔG
2	190	151.0	0.518
5	135	95.0	0.515
10	95	67.0	0.511
20	65	49.0	0.506
40	40	36.0	0.468
80	30	27.0	0.417
120	28	24.0	0.379
240	16	23.0	0.329
480	12	22.0	0.312

For repetitive diving, the gradients, G, above are replaced with a reduced set, $\bar{G}$, with the property,

$$\bar{G} \leq G \quad .$$

tending to reduce bottom time for repetitve activities and exposures. Because of this constraint, the approach is a reduced (Haldane) gradient model, It is important to note that this model is Haldane pseudo-bubble in nature, also termed a (modified) reduced gradient bubble model in publications. Others, in similar tacts, term the reduction process as a *gradient factor* method, though no formal methodology has been reported. Wienke, linking the reduction process to the full phase reduced gradient bubble model through maximum likelihood profile fits, suggested the following formally in 1990, against the background of the VPM,

$$\dot{\phi} = \Lambda G$$

but abandoning preformed nuclei and regeneration time scales of weeks. The excitation radius deduced from gel experiments (above) was a starting point for the retrofits to Haldane gradients, but had to be abandoned at an early stage for actual meter and table applications, and to fit the data.

The terms, $\Lambda\, G$ and $\Lambda \bar{G}$, differ by effective bubble elimination during the previous surface interval. To maintain the phase volume constraint during multidiving, the elimination rate must be downscaled by a set of bubble growth, regeneration, and excitation factors, cumulatively designated, ξ, such that,

$$\bar{G} = \xi G \quad .$$

A conservative set of bounce gradients, G, can be employed for multiday and repetitive diving, provided they are reduced by ξ. These same ξ are the *gradient factors* available in commercial diveware operationally, though explicit forms and applications do not necessarily map onto the set described below, formally.

Three bubble factors, η, reduce the driving gradients to maintain the phase volume constraint. The first bubble factor, η^{rg}, reduces G to account for creation of new stabilized micronuclei over time scales, ω^{-1}, of hours,

$$\eta^{rg} = exp\ (-\omega t_{cum}) \quad ,$$
$$2 \leq \omega^{-1} \leq 4\ hrs \quad ,$$

for t_{cum} the cumulative (multiday) dive time. The second bubble factor, η^{ex}, accounts for additional micronuclei excitation on reverse profile dives,

$$\eta^{ex} = \frac{(\Lambda)_{prev}}{(\Lambda)_{pres}}$$

for excitation radius, r, at depth, d, and the subscripts referencing the *previous* and *present* dives. Obviously, η^{ex} remains one until a deeper point than on the previous dive is reached. The third factor, η^{rp}, accounts for bubble growth over repetitive exposures on time scales, χ^{-1}, of hours,

$$\eta^{rp} = 1 - \left[1 - \frac{G^{bub}}{G_0\ exp\ (-\omega t_{cum})}\right] exp\ (-\chi t_{sur}) \quad ,$$
$$10 \leq \chi^{-1} \leq 120\ minutes \quad ,$$
$$0.05 \leq \frac{G^{bub}}{G_0} \leq 0.90 \quad ,$$

according to the tissue compartment, with t_{sur} the repetitive surface interval.

In terms of individual bubble factors, η, the multidiving fraction, ξ, is defined at the start of each segment, and deepest point of dive,

$$\xi = a\eta^{rg} + b\eta^{rp} + c\eta^{ex}$$

for a, b, and c constants,

$$a + b + c = 1$$

with surface and cumulative surface intervals appropriate to the preceeding dive segment. With η bounded by zero and one, ξ are similarly bounded by zero and one. Corresponding critical tensions, M, can be computed from the above,

$$M = \xi G + P \quad ,$$

with G listed in Table 2 above. Both G and ξ are lower bounded by the shallow saturation data,

$$G \geq G^{bd} = 0.303\ P + 11 \quad ,$$

for P ambient pressure, and similarly,

$$\xi \geq \xi^{bd} = \frac{0.12 + 0.18\ exp\ (-480\lambda_{bd})}{0.12 + 0.18\ exp\ (-\tau\lambda_{bd})} \quad ,$$

$$\lambda_{bd} = 0.0559\ min^{-1} \quad .$$

Tables And Meters

For purposes of continuity, a chronological ordering of models is taken below. Obviously, models get better in time, and as the list progresses. Time span across these models is roughly a century, and only the main ones appear.

1. Bulk Diffusion Model

2. Multitissue Model

   The multitissue model addresses dissolved gas transport with saturation gradients driving the elimination. In the presence of free phases, free-dissolved and free-blood elimination gradients can compete with dissolved-blood gradients. One suggestion is that the gradient be split into two weighted parts, the free-blood and dissolved-blood gradients, with the weighting fraction proportional to the amount of separated gas per unit tissue volume. Use of a split gradient is consistent with multiphase flow partitioning, and implies that only a portion of tissue gas has separated, with the remainder dissolved. Such a split representation can replace any of the gradient terms in tissue response functions.

   If gas nuclei are entrained in the circulatory system, blood perfusion rates are effectively lowered, an impairment with impact on all gas exchange processes. This suggests a possible lengthening of tissue halftimes for elimination over those for uptake, for instance, a 10 min compartment for uptake becomes a 12 min compartment on elimination. Such lengthening procedure and the split elimination gradient obviously render gas uptake and elimination processes asymmetric. Instead of both exponential uptake and elimination, exponential uptake and linear elimination response functions can be used. Such modifications can again be employed in any perfusion model easily, and tuned to the data.

3. Thermodynamic Model

   The thermodynamic model (TM) suggested by Hills, and extended by others, is more comprehensive than earlier models, addressing a number of issues simultaneously, such as tissue gas exchange, phase separation, and phase volume trigger points. This model is based on phase equilibration of dissolved and separated gas phases, with temporal uptake and elimination of inert gas controlled by perfusion and diffusion. From a boundary (vascular) thin zone, gases

diffuse into the cellular region. Radial, one dimensional, cylindrical geometry is assumed as a starting point, though the extension to higher dimensionality is straightforward. As with all dissolved gas transfer, diffusion is controlled by the difference between the instantaneous tissue tension and the venous tension, and perfusion is controlled by the difference beween the arterial and venous tension. A mass balance for gas flow at the vascular cellular interface, enforces the perfusion limit when appropriate, linking the diffusion and perfusion equations directly. Blood and tissue tensions are joined in a complex feedback loop. The trigger point in the thermodynamic model is the separated phase volume, related to a set of mechanical pain thresholds for fluid injected into connective tissue.

The full thermodynamic model is complex, though Hills has performed massive computations correlating with the data, underscoring basic model validity. One of its more significant features can be seen in Figure 11. Considerations of free phase dynamics (phase volume trigger point) require deeper decompression staging formats, compared to considerations of critical tensions, and are characteristic of phase models. Full blown bubble models require the same, simply to minimize bubble excitation and growth.

4. Varying Permeability Model

   The varying permeability model (VPM) treats both dissolved and free phase transfer mechanisms, postulating the existence of gas seeds (micronuclei) with permeable skins of surface active molecules, small enough to remain in solution and strong enough to resist collapse. The model is based upon laboratory studies of bubble growth and nucleation.

   Inert gas exchange is driven by the local gradient, the difference between the arterial blood tension and the instantaneous tissue tension. Compartments with 1, 2, 5, 10, 20, 40, 80, 120, 240, 480, and 720 halftimes, τ, are again employed. While, classical (Haldane) models limit exposures by requiring that the tissue tensions never exceed the critical tensions, fitted to the US Navy nonstop limits, for example, the varying permeability model, however, limits the supersaturation gradient, through the phase volume constraint. An exponential distribution of bubble seeds, falling off with increasing bubble size is assumed to be excited into growth by compression-decompression. A critical radius, r_c, separates growing from contracting micronuclei for given ambient pressure, P_c. At sea level, $P_c = 33\ fsw$, $r_c = 0.8\ \mu m$. Deeper decompressions excite smaller, more stable, nuclei.

   Within the phase volume constraint, a set of nonstop limits, t_n, at depth, d, satisfy a modified law, $dt_n^{1/2} = 400\ fsw\ min^{1/2}$, with gradient, G, extracted for each compartment, τ, using the nonstop limits and excitation radius, at generalized depth, $d = P - 33\ fsw$. Tables 2 and 7 summarize t_n, G_0, ΔG, and δ, the depth at which the compartment begins to control exposures.

Table 7. Critical Phase Volume Time Limits.

depth $d\ (fsw)$	nonstop limit $t_n\ (min)$	depth $d\ (fsw)$	nonstop limit $t_n\ (min)$
30	250.	130	9.0
40	130.	140	8.0
50	73.	150	7.0
60	52.	160	6.5
70	39.	170	5.8
80	27.	180	5.3
90	22.	190	4.6
100	18.	200	4.1
110	15.	210	3.7
120	12.	220	3.1

Gas filled crevices can also facilitate nucleation by cavitation. The mechanism is responsible for bubble formation occuring on solid surfaces and container walls. In gel experiments, though, solid particles and ragged surfaces were seldom seen, suggesting other nucleation mechanisms. The existence of stable gas nuclei is paradoxical. Gas bubbles larger than 1 μm should float to the surafce of a standing liquid or gel, while smaller ones should dissolve in a few sec. In a liquid supersaturated with gas, only bubbles at the critical radius, r_c, would be in equilibrium (and very unstable equilibrium at best). Bubbles larger than the critical radius should grow larger, and bubbles smaller than the critical radius should collapse. Yet, the Yount gel experiments suggest the existence of stable gas phases, so no matter what the mechanism, effective surface tension must be zero. Although the actual size distribution of gas nuclei in humans is unknown, these experiments in gels have been correlated with a decaying exponential (radial) distribution function. For a stabilized distribution accommodated by the body at fixed pressure, P_c, the excess number of nuclei excited by compression-decompression must be removed from the body. The rate at which gas inflates in tissue depends upon both the excess bubble number, and the supersaturation gradient, G. The critical volume hypothesis requires that the integral of the product of the two must always remain less than some volume limit point, αV, with α a proportionality constant.

5. Reduced Gradient Bubble Model

The RGBM departs from the VPM in a number of ways, abandoning gel parameterizations. Colloidal suspensions, such as gel, are far different than aqueous and lipid materials coating bubbles and seeds in the body. Additionally, typical gel-type micronuclei, with persistence time scales of tens of hours to days, have never been found in the body in any circumstance. Present wisdom suggests that seeds are produced by tribonucleation (tissue friction). The full blown RGBM treats coupled perfusion-diffusion transport as a two step flow process, with blood flow (perfusion) serving as a boundary condition for tissue gas penetration by diffusion. Depending on time scales and rate coefficients, one or another (or both) processes dominate the exchange. However, for most meter implementations, perfusion is assumed to dominate, simplifying matters and permitting online calculations. Additionally, tissues and blood are naturally undersaturated with respect to ambient pressure at equilibration through the mechanism of biological inherent unsaturation (oxygen window), and the model includes this debt in calculations.

The RGBM assumes that a size distribution of seeds (potential bubbles) is always present, and that a certain number is excited into growth by compression-decompression. An iterative

process for ascent staging is employed to control the inflation rate of these growing bubbles so that their collective volume never exceeds a phase volume limit point. Gas mixtures of helium, nitrogen, and oxygen contain bubble distributions of different sizes, but possess the same phase volume limit point.

The RGBM postulates bubble seeds with lipid or aqueous skin structure. Bubble skins are assumed permeable under all crushing pressure, unlike the VPM. The size of seeds excited into growth is inversely proportional to the supersaturation gradient. At increasing pressure, bubble seeds permit gas diffusion at a slower rate. The model assumes bubble skins are stabilized by surfactants over calculable time scales, producing seeds that are variably persistent in the body. Bubble skins are probably molecularly activated, complex, biosubstances found throughout the body. Whatever the formation process, the model assumes the size distribution is exponentially decreasing in size, that is, more smaller seeds than larger seeds in exponential proportions. The RGBM also employs an equation-of-state for the skin surfactants, linked to lipid and aqueous biophysical structures. Gas diffusion across the bubble film interface, and Boyle expansion and contraction under ambient pressure change are also tracked in the RGBM.

In tracking seed excitation and number, gas transport into and out of bubbles, and Boyle-like expansion and contraction under pressure changes, the RGBM incorporates a spectrum of tissue compartments, ranging from 1 min to 480 min, depending on gas mixture (helium, nitrogen, oxygen). Phase separation and bubble growth in all compartments is a central focus in calculations, over appropriate time scales, and the model uses nonstop time limits tuned to recent Doppler measurements, conservatively reducing them along the lines originally sugested by Spencer (and others), but within the phase volume constraint.

The Haldane folded RGBM reduces the phase volume limit in multidiving by considering free phase elimination and buildup during surface intervals, depending on altitude, time, and depth of previous profiles, Repetitive, multiday, and reverse profile exposures are tracked and impacted by critical phase volume reductions over appropriate time scales. The model generates bubble seed distributions on time scales of minutes to hours, adding new bubbles to existing bubbles in calculations. Phase volume limit points are also reduced by the added effects of new bubbles. In the Haldane folded algorithm, deep stops can be injected into staging procedures with a simple time-depth scaling law correlated with calculations from the full iterative RGBM model.

The modified (folded) RGBM extends the classical Haldane model to repetitive diving, by conservatively reducing the gradients, G. A conservative set of bounce gradients, G, can always be used for multiday and repetitive diving, provided they are multiplicatively reduced by a set of bubble factors, all less than one (Chapter 4). Three bubble factors reduce the driving gradients to maintain the phases volume constraint. The first bubble factor reduces G to account for creation of new stabilized micronuclei over time scales of days. The second factor accounts for additional micronuclei excitation on reverse profile dives. The third bubble factor accounts for bubble growth over repetitive exposures on time scales of hours. Their behavior is depicted in Figures 5, 6, and 7.

The RGBM (both versions) is a diveware implementation, accessible on the Internet at various sites. Additionally, the RGBM has been encoded into a number of commercial decompression meter products. Specific comparisons between RGBM and Haldane predictions for staging are summarized (Chapter 6), with resultants generic for phase versus dissolved gas models. NAUI uses RGBM Tables for trimix, helitrox, nitrox, and altitude dive training.

6. Tissue Bubble Diffusion Model

   The tissue bubble diffusion model (TBDM), according to Gernhardt and Vann, considers the diffusive growth of an extravascular bubble under arbitrary hyperbaric and hypobaric loadings.

The approach incorporates inert gas diffusion across the tissue-bubble interface, tissue elasticity, gas solubility and diffusivity, bubble surface tension, and perfusion limited transport to the tissues. Tracking bubble growth over a range of exposures, the model can be extended to oxygen breathing and inert gas switching. As a starting point, the TBDM assumes that, through some process, stable gas nuclei form in the tissues during decompression, and subsequently tracks bubble growth with dynamical equations. Diffusion limited exchange is invoked at the tissue-bubble interface, and perfusion limited exchange is assumed between tissue and blood, very similar to the thermodynamic model, but with free phase mechanics. Across the extravascular region, gas exchange is driven by the pressure difference between dissolved gas in tissue and free gas in the bubble, treating the free gas as ideal. Initial nuclei in the TBDM have assumed radii near 3 μm at sea level, to be compared with 0.65 μm in the RGBM.

As in any free phase model, bubble volume changes become more significant at lower ambient pressure, suggesting a mechanism for enhancement of hypobaric bends, where constricting surface tension pressures are smaller than those encountered in hyperbaric cases. As seen in Figure 12, the model has been coupled to statistical likelihood, correlating bubble size with decompression risk, a topic discussed in a few chapters. For instance, a theoretical bubble dose of 5 ml correlates with a 20% risk of decompression sickness, while a 35 ml dose correlates with a 90% risk, with the bubble dose representating an unnormalized measure of the separated phase volume. Coupling bubble volume to risk represents yet another extension of the phase volume hypothesis, a viable trigger point mechanism for bends incidence.

Under compression-decompression, gas nuclei may grow as bubbles, depending on their effective bubble radius. Below a certain critical radius, r, listed in Table 8 below as a function of pressure according to a bubble model (varying permeability), as fitted to gel experiments, bubbles tend to collapse on themselves, while at larger equilibrium radius, they grow as gas diffuses into them. Stabilized nuclei evolve into unstable bubbles when their effective surface tension is greater than zero, or a sufficient diffusion gradient exists to drive gas into, or out of, the nucleus. At sea level, the model excitation radius is near 0.8 μm, smaller than living cells, having dimensions starting at a few μm.

Table 8. Varying Permeability Model Excitation Radii.

pressure P (fsw)	excitation radius r (μm)	pressure P (fsw)	excitation radius r (μm)
13	0.89	153	0.49
33	0.80	183	0.45
53	0.72	283	0.35
73	0.66	383	0.29
93	0.61	483	0.24
113	0.57	583	0.21

However, the EOS excitation radii of the reduced gradient bubble model, Table 1 (Chapter 7), are much smaller than those of the varying permeability model above, certainly no surprise because lipid and aqueous tissues are not colloidal gel suspensions. Gels are not relevant because biological fluids are formed, and contained, in a sealed environment (the body). The Strauss and Yount studies suggest the existence of gas micronuclei in gels. Partially stable nuclei seem to pervade all manner of fluids. But gel nuclei would seem to share little with nuclei formed in the body, since the materials stabilizing body nuclei are not colloidal gel.

Abandoning preformed nuclei, other methods of instantaneous bubble formation are certainly possible. Cavitation, produced by the rapid tearing, or moving apart, of tissue interfaces, is a candidate, as well as surface friction (tribonucleation). Crevices in tissues may form or trap gas phases, with later potential for release. Vorticity in blood flow patterns might cause small microbubbles. Stable,

or unstable, the copious presence of microbubbles in the venous circulation would impact dissolved gas elimination adversely, also possibly impairing the lungs or the arterial network. The presence of bubbles in the arterial circulation might result in embolism. Bubble clogging of the pulmonary circulation is thought to relate to the chokes, a serious form of decompression sickness, while cerebral decompression sickness is believed due to emboli. Microbubbles in the venous circulation would render gas uptake and elimination asymmetric, with uptake faster than elimination. Displacing blood, microbubbles would reduce the effective area and volume for tissue-blood gas exchange.

PHASE MECHANICS AND DECOMPRESSION THEORY IN DEPTH CHAPTER 7: COMPUTING AND DECOMPRESSION ALGORITHMS

Computing Advances

Computational Algorithms

The models touched on (Chapter 4) address the coupled issues of gas uptake and elimination, bubbles, and pressure changes in different computational approaches. Application of a computational model to staging divers and aviators is often called a diving algorithm. Consider the computational model and staging regimen for 7 popular algorithms, namely, the perfusion limited, diffusion limited, thermodynamic, varying permeability, reduced gradient bubble (2), and tissue bubble diffusion algorithms:

Dissolved Phase Algorithms

Dissolved gas diving algorithms historically trace back to the original Haldane experiments in the early 1900s. They are still around today, in tables, meters, and diving software. That is changing, however, as modern divers go deeper, stay longer, decompress, and used mixed gases.

Dual Phase Algorithms

Dual phase diving algorithms are rather recent innovations, coming online in the past 20 years or so. They are more correct than dissolved gas algorithms, becasue they couple dissolved gases to bubbles, and lead to deeper staging as a result. Meters, tables, and software employing these algorithms do exist, and are supplanting traditional versions.

1. Thermodynamic

   The thermodynamic model couples both the tissue diffusion and blood perfusion equations. Cylindrical symmetry is assumed in the model. From a boundary vascular zone of thickness, a, gas diffuses into the extended extravascular region, bounded by b. The radial diffusion equation is given by,

$$D\frac{\partial^2 p}{\partial r^2} + \frac{D}{r}\frac{\partial p}{\partial r} = \frac{\partial p}{\partial t}$$

   with the tissue tensions, p, equal to the venous tensions, p_v, at the vascular interfaces, a and b. The solution to the tissue diffusion equation is given previously,

$$p - p_v = (p_i - p_v)\frac{4}{(b/2)^2 - a^2}\sum_{n=1}^{\infty}\frac{1}{\epsilon_n^2}\frac{J_1^2(\epsilon_n b/2)}{J_0^2(\epsilon_n a) - J_1^2(\epsilon_n b/2)} exp\ (-\epsilon_n^2 D t)$$

   with ϵ_n eigenvalue roots of the boundary conditions,

$$J_0(\epsilon_n a)\ Y_1(\epsilon_n b/2) - Y_0(\epsilon_n a)\ J_1(\epsilon_n b/2) = 0$$

for J and Y Bessel and Neumann functions, order 1 and 0. Perfusion limiting is applied as a boundary condition through the venous tension, p_v, by enforcing a mass balance across both the vascular and cellular regions at a,

$$\frac{\partial p_v}{\partial t} = -\kappa(p_v - p_a) - \frac{3}{a} S_p D \left[\frac{\partial p}{\partial r}\right]_{r=a}$$

with S_p the ratio of cellular to blood gas solubilities, κ the perfusion constant, and p_a the arterial tension. The coupled set relate tension, gas flow, diffusion and perfusion, and solubility in a complex feedback loop.

The thermodynamic trigger point for decompression sickness is the volume fraction, χ, of separated gas, coupled to mass balance. Denoting the separated gas partial pressure, P_{N_2}, under worse case conditions of zero gas elimination upon decompression, the separated gas fraction is estimated,

$$\chi \; P_{N_2} = S_c \; (p - P_{N_2})$$

with S_c the cellular gas solubility. The separated nitrogen partial pressure, P_{N_2} is taken up by the inherent unsaturation, and given by (fsw),

$$P_{N_2} = P + 3.21$$

in the original Hills formulation, but other estimates have been employed. Mechanical fluid injection pain, depending on the injection pressure, δ, can be related to the separated gas fraction, χ, through the tissue modulus, K,

$$K\chi = \delta$$

so that a decompression criteria requires,

$$K\chi \leq \delta$$

with δ in the range, for $K = 3.7 \times \; 10^4 \; dyne \; cm^{-2}$,

$$0.34 \leq \delta \leq 1.13 \; fsw.$$

Identification of the separated phase volume as a critical indicator is a significant development in decompression theory.

2. Varying Permeability

The critical radius, r_i, at fixed pressure, P_i, represents the cutoff for growth upon decompression to lesser pressure. Nuclei larger than r_i will all grow upon decompression. Additionally, following an initial compression, a smaller class of micronuclei of critical radius, r, can be excited into growth with decompression. If r_i is the critical radius at P_i, then, the smaller family, r, excited by decompression from P, obeys,

$$\frac{2\gamma}{r} - P = \frac{2\gamma}{r_i} - P_i$$

with P measured in fsw, and r in μm. Table 1 (Chapter 1) lists critical radii, r, excited by sea level compressions ($P_i = 33 \; fsw$), assuming $r_i = 0.8 \; \mu m$. Entries also represent the equilibrium critical radius at pressure, P.

The permissible gradient, G, is written for each compartment, τ, using the standard formalism,

$$G = G_0 + \Delta G d$$

at depth $d = P - 33\ fsw$. A nonstop bounce exposure, followed by direct return to the surface, thus allows G_0 for that compartment. Both G_0 and ΔG are tabulated in Table 2 (Chapter 4), with ΔG suggested by Buhlmann. The minimum excitation, G^{min}, initially probing r, and taking into account regeneration of nuclei over time scales τ_r, is (fsw),

$$G^{min} = \frac{2\gamma\ (\gamma_c - \gamma)}{\gamma_c\ r(t)} = \frac{11.01}{r(t)}$$

with,

$$r(t) = r + (r_i - r)\ [1 - exp\ (-\lambda_r t)]$$

γ, γ_c film, surfactant surface tensions, that is, $\gamma = 17.9\ dyne/cm$, $\gamma_c = 257\ dyne/cm$, and λ_r the inverse of the regeneration time for stabilized gas micronuclei (many days). Prolonged exposure leads to saturation, and the largest permissible gradient, G^{sat}, takes the form (fsw), in all compartments,

$$G^{sat} = \frac{58.6}{r} - 49.9 = 0.372\ P + 11.01.$$

On the other hand, G^{min} is the excitation threshold, the amount by which the surrounding tension must exceeed internal bubble pressure to just support growth.

Although the actual size distribution of gas nuclei in humans is unknown, experiments *in vitro* suggest that a decaying exponential is reasonable,

$$n = N\ exp\ (-\beta r)$$

with β a constant, and N a convenient normalization factor across the distribution. For small values of the argument, βr,

$$exp\ (-\beta r) = 1 - \beta r$$

as a nice simplification. For a stabilized distribution, n_0, accommodated by the body at fixed pressure, P_0, the excess number of nuclei, Λ, excited by compression-decompression from new pressure, P, is,

$$\Lambda = n_0 - n = N\beta r_i \left[1 - \frac{r}{r_i}\right].$$

For large compressions-decompressions, Λ is large, while for small compressions-decompressions, Λ is small. When Λ is folded over the gradient, G, in time, the product serves as a critical volume indicator and can be used as a limit point in the following way.

The rate at which gas grows in tissue depends upon both the excess bubble number, Λ, and the gradient, G. The critical volume hypothesis requires that the integral of the product of the two must always remain less than some limit point, $\alpha\ V$, with α a proportionality constant,

$$\int_0^\infty \Lambda G dt = \alpha V$$

for V the limiting gas volume. Assuming that gradients are constant during decompression, t_d, while decaying exponentially to zero afterwards, and taking the limiting condition of the equal sign, yields simply for a bounce dive, with λ the tissue constant,

$$\Lambda G\ (t_d + \lambda^{-1}) = \alpha V.$$

In terms of earlier parameters, one more constant, δ, closes the set, defined by,

$$\delta = \frac{\gamma_c \alpha V}{\gamma \beta r_i N} = 7180\ fsw\ min$$

so that,

$$\left[1 - \frac{r}{r_i}\right] \; G \; (t_d + \lambda^{-1}) = \delta \frac{\gamma}{\gamma_c} = 500.8 \; fsw \; min.$$

The five parameters, γ, γ_c, δ, λ_r, r_i, are five of the six fundamental constants in the varying permeability model. The remaining parameter, λ_m, interpolating bounce and saturation exposures, represents the inverse time contant modulating multidiving. Doppler experiments suggest that λ_m^{-1} is in the neighborhood of an hour. Discussion of λ_m follows in the next section (RGBM).

The depth at which a compartment controls an exposure, and the excitation radius as a function of halftime, τ, in the range, $12 \leq d \leq 220 \; fsw$, satisfy,

$$\frac{r}{r_i} = 0.90 - 0.43 \; exp \; (-\zeta \tau)$$

with $\zeta = 0.0559 \; min^{-1}$. The regeneration constant, λ_r, is on the order of inverse days, that is, $\lambda_r = .0495 \; days^{-1}$. Characteristic halftimes, τ_r and τ_h, take the values $\tau_r = 14 \; days$ and $\tau_h = 12.4 \; min$. For large τ, r is close to r_i, while for small τ, r is on the order of 0.5 r_i. At sea level, $r_i = 0.8 \; \mu m$ as discussed.

3. Reduced Gradient Bubble

Two versions exist. One is a Haldane folded (single phase) algorithm using phase factors from the full iterative model to limit Haldane repetitive, reverse profile, multiday activities, and flying after diving. The folded version is found in many decometers on the market today. The full (dual phase) version is the basis of released mixed gas technical tables and simplified no-group, no-calc recreational air and nitrox tables up tp 10,000 ft elevation. Meter implementations of the full RGBM are underway. Both modified and iterative RGBM are offered to users of ABYSS diveware.

Dual Phase

As mentioned the full RGBM employs a phase volume constraint across the total dive profile. The gel parameterization is replaced by flexible seed skins with appropriate EOS, permeable to gas diffusion at all pressures and temperatures. Gas diffuses across the bubble interface, and the bubble is subjected to Boyle expansion-contraction.

The phase volume constraint equation is rewritten in terms of a phase function, $\dot{\phi}$, varying in time,

$$\int_0^\tau \frac{\partial \phi}{\partial t} dt \; \leq \; \Phi$$

with, as before,

$$\dot{\phi} = \frac{\partial \phi}{\partial t}$$

for Φ the separated phase, and τ some (long) cutoff time. More particularly, for Π the total gas tension,

$$\dot{\phi} = \left[\frac{\partial V}{\partial t}\right]_{diffusion} + \left[\frac{\partial V}{\partial t}\right]_{Boyle} + \left[\frac{\partial V}{\partial t}\right]_{excitation}$$

for,

$$\left[\frac{\partial V}{\partial t}\right]_{diffusion} = 4\pi DS \int_r^\infty r \left(\Pi - P - \frac{2\gamma}{r}\right) dr$$

$$\left[\frac{\partial V}{\partial t}\right]_{Boyle} = \int_r^\infty n \left(\frac{1}{P} \frac{\partial (PV)}{\partial t}\right) dr$$

$$\left[\frac{\partial V}{\partial t}\right]_{excitation} = \frac{\partial}{\partial t}\left(4\pi \int_0^\infty nnr^2 dr\right)$$

with all quantities as denoted previously, and the bubble number integrand normalized,

$$\int_0^\infty ndr = 1$$

Thus the phase function, $\dot{\phi}$, depends on the number of bubbles, n, stimulated into growth by compression-decompression, the supersaturation gradient, G, seed expansion-contraction by radial diffusion, $\partial r/\partial t$, Boyle expansion-contraction, PV, under pressure changes, and temperature, T, in general. The excitation radius, r, depends on the material properties, and is given for nitrogen (μm),

$$r_{N_2} = 0.007655 + 0.001654\left[\frac{T}{P}\right]^{1/3} + 0.041602\left[\frac{T}{P}\right]^{2/3}$$

and for helium,

$$r_{He} = 0.001946 + 0.009832\left[\frac{T}{P}\right]^{1/3} + 0.016183\left[\frac{P}{T}\right]^{2/3}$$

with ranges for the virial coefficients, aqueous to lipid materials, varying by factors of 0.75 to 4.86 the values listed above. Both expression above represent fits to RGBM mixed gas data across lipid and aqueous bubble films, and are different from other phase models. Values of excitation radii, r, above range from 0.01 to 0.05 μm for sea level down to 500 fsw. This is compared to excitation radii in other models (VPM and TBDM) which vary in the 1 μm range. In the very large pressure limit, excitation radii (like beebees) are in the 1/1,000 μm range. Table 1 lists excitation radii (air) according to the RGBM.

Table 1. Reduced Gradient Bubble Model Excitation Radii

pressure P (fsw)	excitation radius r (μm)	pressure P (fsw)	excitation radius r (μm)
13	0.174	153	0.033
33	0.097	183	0.029
53	0.073	283	0.024
73	0.059	383	0.016
93	0.051	483	0.011
113	0.046	583	0.009

Single Phase

The following is specific to the ZHL implementation of the RGBM across critical parameters and nonstop time limits of the RGBM/ZHL algorithm. Extensive computer fitting of profiles and recalibration of parameters to maintain the RGBM within the ZHL limits is requisite here. ABYSS has implemented this synthesis into Internet diveware. Deep stops are not intrinsic in this limited, still basically Haldane approach, but can be inserted empirically as described earlier.

Haldane approaches use a dissolved gas (tissue) transfer equation, and a set of critical parameters to dictate diver staging through the gas transfer equation. In the Workman approach, the critical parameters are called M-values, while in the Buhlmann formulation they are called a

and b. They are equivalent sets, slightly different in representation but not content. Consider air, nitrox, heliox, and trimix in the ZHL formalism.

Overall, the RGBM algorithm is conservative with safety imparted to the Haldane ZHL model through multidiving f factors. Estimated DCS incidence rate from likelihood analysis is 0.01% at the 95% confidence level for the overall RGBM. Table and meter implementations with consistent coding should reflect this estimated risk. Similar estimates and comments apply to the ZHL mixed gas synthesis.

4. Tissue Bubble Diffusion

Bubbles shrink or grow according to a simple radial diffusion equation linking total gas tension, Π, ambient pressure, P, and surface tension, γ, to bubble radius, r,

$$\frac{\partial r}{\partial t} = \frac{DS}{r}\left[\Pi - P - \frac{2\gamma}{r}\right]$$

with D the gas diffusion coefficient, and S the gas solubility. Bubbles grow when the surrounding gas tension exceeds the sum of ambient plus surface tension pressure, and vice versa. Higher gas solubilities and diffusivities enhance the rate. Related bubble area, A, and volume, V, changes satisfy,

$$\frac{\partial A}{\partial t} = 8\pi r \frac{\partial r}{\partial t}$$
$$\frac{\partial V}{\partial t} = 4\pi r^2 \frac{\partial r}{\partial t}$$

Using Fick's law, a corresponding molar current, J, of gas into, or out of, the bubble is easily computed assuming an ideal gas,

$$J = -\frac{DS}{RTh}\left[\Pi - P - \frac{2\gamma}{r}\right]$$

for R the ideal gas constant, T the temperature, and h an effective diffusion barrier thickness. And the molal flow rate is just the molal current times the interface area, that is,

$$\frac{\partial n}{\partial t} = JA$$

for n the number of moles of gas. The change in pressure and volume of the bubble, due to gas diffusion, follows simply from the ideal gas law,

$$\frac{\partial (PV + 2\gamma r^{-1} V)}{\partial t} = R\frac{\partial (nT)}{\partial t}$$

for V the bubble volume.

Obviously, the above constitute a coupled set of differential equations, solvable for a wide range of boundary and thermodynamic conditions connecting the state variables, namely, P, V, Π, r, n, and T.

A bubble dose, based on the hypothetical volume of an expanding test bubble, is linked to decompression data for the exposure. Maximum likelihood regression is used to correlate bubble dose with DCS risk.

Diving models address the coupled issues of gas uptake and elimination, bubbles, and pressure changes in different computational frameworks. Application of a computational model to staging divers is called a diving algorithm. The Reduced Gradient Bubble Model (RGBM) is a modern one, treating the many facets of gas dynamics in tissue and blood consistently. Though the systematics of gas exchange, nucleation, bubble growth or collapse, and decompression are so complicated that theories only reflect pieces of the decompression sickness (DCS) puzzle, the risk and DCS statistics of staging algorithms can be easily collected and analyzed. And the record of the RGBM, just over the past 5 years or so, has been spectacular, especially so far as safe staging coupled to deep stops with overall shorter decompression times. This is important. Models are one thing, even with all the correct biophysics, and actual diving and testing are something else.

RGBM Motivation And Implementations

The RGBM grew from needs of technical divers to more efficiently stage ascents consistent with coarse grain dissolved gas and bubble dynamics, and not just dissolved gas (Haldane) constraints. And the depth, diversity, mix variation, and self consistency of RGBM diving applicability has satisfied that need. And safely.

The RGBM has gained tremendous popularity in the recreational and technical diving worlds in just the past 2 - 3 years, due to meter implementations, Internet software packages, specialized Table releases, technical word of mouth, NAUI training testing and adoption, Internet traffic, chamber tests, and, most of all, actual technical and recreational RGBM diving and validation. And the reasons are fairly clear.

Present notions of nucleations and bubbles suggest that decompression phase separation is random, yet highly probable, in body tissue. Once established, a gaseous phase will further grow by acquiring gas from adjacent saturated tissue, according to the strength of the free-dissolved gradient. Although exchange mechanisms are better understood, nucleation and stabilization mechanisms remain less so, and calculationally elusive. But even with a paucity of knowledge, many feel that existing practices and recent studies on bubbles and nuclei shed considerable light on growth and elimination processes, and time scales. Their consistency with underlying physical principles suggest directions for table and meter modeling, beyond parameter fitting and extrapolation techniques. Recovering dissolved gas algorithms for short exposure times, phase models link to bubble mechanics and critical volume trigger points. The RGBM incorporates all of the above in all implementations, and additionally supports the efficacy of recently suggested safe diving practices, by simple virtue of its dual phase mechanics:

- reduced nonstop time limits;
- safety stops (or shallow swimming ascents) in the 10-20 fsw zone;
- ascent rates not exceeding 30 fsw/min;
- restricted repetitive exposures, particularly beyond 100 fsw,
- restricted reverse profile and deep spike diving;
- restricted multiday activity;
- smooth coalescence of bounce and saturation limit points;
- consistent diving protocols for altitude;

- deep stops for decompression, extended range, and mixed gas diving with overall shorter decompression times, particularly for the shallow zone;
- use of helium rich mixtures for technical diving, with shallower isobaric switches to nitrox than suggested by Haldane stategies;
- use of pure oxygen in the shallow zone to eliminate both dissolved and bubble inert gases.

Bubble models tend to be consistent with the utilitarian measures detailed earlier, and have the right signatures for diving applications across the full spectrum of actvities. Or, said another way, bubble models are more powerful, more correct, and more inclusive. In terms of RGBM implementations, the mechanistics of dissolved gas buildup and elimination, inert gas diffusion across bubble interfaces, bubble excitation and elimination persistence time scales of minutes to hours from tissue friction, lipid and aqueous surfactant material properties, and Boyle expansion and contraction under ambient pressure change, are sufficient to address all of the above considerations.

So Mares, Dacor, Plexus, Suunto, HydroSpace, and Abysmal Diving developed and released products incorporating one such validated phase algorithm, the Reduced Gradient Bubble Model (RGBM), for diving. An iterative approach to staging diver ascents, the RGBM employs separated phase volumes as limit points, instead of the usual Haldane (maximum) critical tensions across tissue compartments. The model is tested and inclusive (altitude, repetitive, mixed gas, decompression, saturation, nonstop exposures), treating both dissolved and free gas phase buildup and elimination. NAUI Technical Diving employs the RGBM to schedule nonstop and decompression training protocols on trimix, helitrox, air, and nitrox, and will be releasing an exhaustive set of RGBM tables for those mixes shortly (some 500 pages of Tables). Included are constant ppO2 Tables for rebreathers. Mares, Dacor, and Plexus are also developing RGBM meters.

Suunto VYPER/COBRA/STINGER are RGBM meters for recreational diving (plus nitrox), while ABYSS/RGBM is a licensed Abysmal Diving software product. The HydroSpace EXPLORER is a mixed gas decompression meter for technical and recreational diving, as is the ABYSS/RGBM software vehicle. All are first-time-ever commercial products with realistic implementation of a diving phase algorithm across a wide spectrum of exposure extremes. And all accommodate user knobs for aggressive to conservative diving. Expect RGBM algorithms to surface in other meters and software packages on the Internet. NAUI Worldwide just released a set of RGBM no-group, no-calc, no-fuss recreational Tables for air and nitrox, sea level to 10,000 feet elevation.

The Countermeasures Dive Team at LANL employs the RGBM (last 8 years). Military, commercial, and scientific sectors are using and further testing the RGBM. And scores of technical divers are reporting their RGBM profiles over the Internet and in technical diving publications. There are presently other major RGBM implementation projects in the works for meters and software packages.

The RGBM extends earlier work of the Tiny Bubble Group at the University of Hawaii, updating missing physics and extending their Varying Permeability Model (VPM) to multidiving, altitude, and mixed gas applications. While certainly fundamental, the RGBM is also different and new on the diving scene. And not unexpectedly, the RGBM recovers the Haldane approach to decompression modeling in the limit of relatively safe (tolerably little) separated phase, with tolerably little a qualitative statement here. There is quite a bit more and different about the RGBM than other and related phase models. Differences focalize, in a word or two, on source generation mechanisms and persistence time scales for bubbles and seeds, bubble structural mechanics and materials, consistent treatment of all bubble expansion and contraction venues, and real world testing.

RGBM Underpinnings Here, our intent is to (just) look at the underpinnings of table, meter, and diveware implementations of the RGBM algorithm, one with extended range of applicability based on simple dual phase principles. Haldane approaches have dominated decompression algorithms for a very long time, and the RGBM has been long in coming on the commercial scene. With technical

diving interest in deep stop modeling, helium, and concerns with repetitive diving in the recreational and technical community, phase modeling is timely and pertinent.

The establishment and evolution of gas phases, and possible bubble trouble, involves a number of distinct, yet overlapping, steps:

- nucleation and stabilization (free phase inception);
- supersaturation (dissolved gas buildup);
- excitation and growth (free-dissolved phase interaction);
- coalescence (bubble aggregation);
- deformation and occlusion (tissue damage and ischemia).

The computational issues of bubble dynamics (formation, growth, and elimination) are mostly outside Haldane framework, but get folded into halftime specifications in a nontractable mode. The very slow tissue compartments (halftimes large, or diffusivities small) might be tracking both free and dissolved gas exchange in poorly perfused regions. Free and dissolved phases, however, do not behave the same way under decompression. Care must be exercised in applying model equations to each component. In the presence of increasing proportions of free phases, dissolved gas equations cannot track either species accurately. Computational algorithms tracking both dissolved and free phases offer broader perspectives and expeditious alternatives, but with some changes from classical schemes. Free and dissolved gas dynamics differ. The driving force (gradient) for free phase elimination increases with depth, directly opposite to the dissolved phase elimination gradient which decreases with depth. Then, changes in operational procedures become necessary for optimality. Considerations of excitation and growth invariably require deeper staging procedures than supersaturation methods. Though not as dramatic, similar constraints remain operative in multiexposures, that is, multilevel, repetitive, and multiday diving.

Other issues concerning time sequencing of symptoms impact computational algorithms. That bubble formation is a predisposing condition for decompression sickness is universally accepted. However, formation mechanisms and their ultimate physiological effect are two related, yet distinct, issues. On this point, most hypotheses makes little distinction between bubble formation and the onset of bends symptoms. Yet we know that silent bubbles have been detected in subjects not suffering from decompression sickness. So it would thus appear that bubble formation, per se, and bends symptoms do not map onto each other in a one-to-one manner. Other factors are truly operative, such as the amount of gas dumped from solution, the size of nucleation sites receiving the gas, permissible bubble growth rates, deformation of surrounding tissue medium, and coalescence mechanisms for small bubbles into large aggregates, to name a few. These issues are the pervue of bubble theories, but the complexity of mechanisms addressed does not lend itself easily to table, nor even meter, implementation. But implement and improve we must, so consider the RGBM issues and tacks taken in the Suunto, Mares, Dacor, Hydrospace, and ABYSS implementations:

1. Perfusion And Diffusion

   Perfusion and diffusion are two mechanisms by which inert and metabolic gases exchange between tissue and blood. Perfusion denotes the blood flow rate in simplest terms, while diffusion refers to the gas penetration rate in tissue, or across tissue-blood boundaries. Each mechanism has a characteristic rate constant for the process. The smallest rate constant limits the gas exchange process. When diffusion rate constants are smaller than perfusion rate constants, diffusion dominates the tissue-blood gas exchange process, and vice-versa. In the body, both processes play a role in real exchange process, especially considering the diversity of tissues and their geometries. The usual Haldane tissue halftimes are the inverses of perfusion

rates, while the diffusivity of water, thought to make up the bulk of tissue, is a measure of the diffusion rate.

Clearly in the past, model distinctions were made on the basis of perfusion or diffusion limited gas exchange. The distinction is somewhat artificial, especially in light of recent analyses of coupled perfusion-diffusion gas transport, recovering limiting features of the exchange process in appropriate limits. The distinction is still of interest today, however, since perfusion and diffusion limited algorithms are used in mutually exclusive fashion in diving. The obvious mathematical rigors of a full blown perfusion-diffusion treatment of gas exchange mitigate against table and meter implementation, where model simplicity is a necessity. So one or another limiting models is adopted, with inertia and track record sustaining use. Certainly Haldane models fall into that categorization.

Inert gas transfer and coupled bubble growth are subtly influenced by metabolic oxygen consumption. Consumption of oxygen and production of carbon dioxide drops the tissue oxygen tension below its level in the lungs (alveoli), while carbon dioxide tension rises only slightly because carbon dioxide is 25 times more soluble than oxygen. Figure 3 (Chapter 1) compares the partial pressures of oxygen, nitrogen, water vapor, and carbon dioxide in dry air, alveolar air, arterial blood, venous blood, and tissue (cells).

Arterial and venous blood, and tissue, are clearly unsaturated with respect to dry air at 1 *atm.* Water vapor content is constant, and carbon dioxide variations are slight, though sufficient to establish an outgradient between tissue and blood. Oxygen tensions in tissue and blood are considerably below lung oxygen partial pressure, establishing the necessary ingradient for oxygenation and metabolism. Experiments also suggest that the degree of unsaturation increases linearily with pressure for constant composition breathing mixture, and decreases linearily with mole fraction of inert gas in the inspired mix.

Since the tissues are unsaturated with respect to ambient pressure at equilibrium, one might exploit this window in bringing divers to the surface. By scheduling the ascent strategically, so that nitrogen (or any other inert breathing gas) supersaturation just takes up this unsaturation, the total tissue tension can be kept equal to ambient pressure. This approach to staging is called the zero supersaturation ascent.

The full blown RGBM treats coupled perfusion-diffusion transport as a two step flow process, with blood flow (perfusion) serving as a boundary condition for tissue gas penetration (diffusion). Depending on time scales and rate coefficients, one or another (or both) processes dominate the exchange. However, for the Suunto, Mares, Dacor, Hydrospace, Plexus, and ABYSS implementations, perfusion is assumed to dominate, simplifying matters and permitting online calculations. Additionally, tissues and blood are naturally undersaturated with respect to ambient pressure at equilibration through the mechanism of biological inherent unsaturation (oxygen window), and the RGBM includes this debt in calculations. Independent of perfusion or diffusion dominated gas transport, the RGBM tracks bubble excitation and number, inert gas transfer across the surfactant skin, and Boyle-like expansion and contraction of bubbles with ambient pressure changes.

2. Bubbles

We do not really know where bubbles form nor lodge, their migration patterns, their birth and dissolution mechanisms, nor the exact chain of physico-chemical insults resulting in decompression sickness. Many possibilities exist, differing in the nature of the insult, the location, and the manifestation of symptoms. Bubbles might form directly (de novo) in supersaturated sites upon decompression, or possibly grow from preformed, existing seed nuclei excited by compression-decompression. Leaving their birth sites, bubbles may move to critical sites elsewhere. Or stuck at their birth sites, bubbles may grow locally to pain-provoking size. They

might dissolve locally by gaseous diffusion to surrounding tissue or blood, or passing through screening filters, such as the lung complex, they might be broken down into smaller aggregates, or eliminated completely. Whatever the bubble history, it presently escapes complete elucidation. But whatever the process, the end result is very simple, both separated and dissolved gas must be treated in the transfer process.

Bubbles may hypothetically form in the blood (intravascular) or outside the blood (extravascular). Once formed, intravascularly or extravascularly, a number of critical insults are possible. Intravascular bubbles may stop in closed circulatory vessels and induce ischemia, blood sludging, chemistry degradations, or mechanical nerve deformation. Circulating gas emboli may occlude the arterial flow, clog the pulmonary filters, or leave the circulation to lodge in tissue sites as extravasular bubbles. Extravascular bubbles may remain locally in tissue sites, assimilating gas by diffusion from adjacent supersaturated tissue and growing until a nerve ending is deformed beyond its pain threshold. Or, extravascular bubbles might enter the arterial or venous flows, at which point they become intravascular bubbles.

Spontaneous bubble formation in fluids usually requires large decompressions, like hundreds of atmospheres, somewhere near fluid tensile limits. Many feel that such circumstance precludes direct bubble formation in blood following decompression. Explosive, or very rapid decompression, of course is a different case. But, while many doubt that bubbles form in the blood directly, intravascular bubbles have been seen in both the arterial and venous circulation, with vastly greater numbers detected in venous flows (venous gas emboli). Ischemia resulting from bubbles caught in the arterial network has long been implied as a cause of decompression sickness. Since the lungs are effective filters of venous bubbles, arterial bubbles would then most likely originate in the arteries or adjacent tissue beds. The more numerous venous bubbles, however, are suspected to first form in lipid tissues draining the veins. Lipid tissue sites also possess very few nerve endings, possibly masking critical insults. Veins, thinner than arteries, appear more susceptible to extravascular gas penetration.

Extravascular bubbles may form in aqueous (watery) or lipid (fatty) tissues in principle. For all but extreme or explosive decompression, bubbles are seldom observed in heart, liver, and skeletal muscle. Most gas is seen in fatty tissue, not unusual considering the five-fold higher solubility of nitrogen in lipid tissue versus aqueous tissue. Since fatty tissue has few nerve endings, tissue deformation by bubbles is unlikely to cause pain locally. On the other hand, formations or large volumes of extravascular gas could induce vascular hemorrhage, depositing both fat and bubbles into the circulation as noted in animal experiments. If mechanical pressure on nerves is a prime candidate for critical insult, then tissues with high concentrations of nerve endings are candidate structures, whether tendon or spinal cord. While such tissues are usually aqueous, they are invested with lipid cells whose propensity reflects total body fat. High nerve density and some lipid content supporting bubble formation and growth would appear a conducive environment for a mechanical insult.

To satisfy thermodynamic laws, bubbles assume spherical shapes in the absence of external or mechanical (distortion) pressures. Bubbles entrain free gases because of a thin film, exerting surface tension pressure on the gas. Hydrostatic pressure balance requires that the pressure inside the bubble exceed ambient pressure by the amount of surface tension, γ. Figure 2 (Chapter 3) depicts the pressure balance in a spherical (air) bubble. At small radii, surface tension pressure is greatest, and at large radii, surface tension pressure is least.

Gases will also diffuse into or out of a bubble according to differences in gas partial pressures inside and outside the bubble, whether in free or dissolved phases outside the bubble. In the former case, the gradient is termed free-free, while in the latter case, the gradient is termed free-dissolved. Unless the surface tension is identically zero, there is always a gradient tending to force gas out of the bubble, thus making the bubble collapse on itself because of surface

tension pressure. If surrounding external pressures on bubbles change in time, however, bubbles may grow or contract. Figure 3 (Chapter 3) sketches bubble gas diffusion under instantaneous hydrostatic equilibrium for an air bubble.

Bubbles grow or contract according to the strength of the free-free or free-dissolved gradient, and it is the latter case which concerns divers under decompression. The radial rate at which bubbles grow or contract depends directly on the diffusivity and solubility, and inversely on the bubble radius. A critical radius, r_c, separates growing from contracting bubbles. Bubbles with radius $r > r_c$ will grow, while bubbles with radius $r < r_c$ will contract. Limiting bubble growth and adverse impact upon nerves and circulation are issues when decompressing divers and aviators.

Bubbles grow or contract by gaseous diffusion across the thin film interface, due to dissolved gas gradients. Bubbles also expand or contract upon pressure changes according to Boyle-like equations of state (EOS), with the expansion or contraction rate a function of the material composition of of the surfactants coating the inside of the bubble. Material behavior can vary from thin elastic films to almost solid shell beebees,

depending on the coefficients and pressure regimes of the EOS. *The RGBM assumes that a size distribution of seeds (potential bubbles) is always present, and that a certain number is excited into growth by compression-decompression. An iterative process for ascent staging is employed to control the inflation rate of these growing bubbles so that their collective volume never exceeds a phase volume limit point. Gas mixtures of helium, nitrogen, and oxygen contain bubble distributions of different sizes, but possess the same phase volume limit point. Distributions have lifetimes of minutes to many hours, impacting repetitive, reverse profile, multiday, altitude, and gas mixes on varying time scales. Colloidal particles are not the stabilizing material inside seeds and bubbles.*

3. Bubble Seeds

   Bubbles, which are unstable, are thought to grow from micron size, gas nuclei which resist collapse due to elastic skins of surface activated molecules (surfactants), or possibly reduction in surface tension at tissue interfaces or crevices. If families of these micronuclei persist, they vary in size and surfactant content. Large pressures (not really known) are necessary to crush them. Micronuclei are small enough to pass through the pulmonary filters, yet dense enough not to float to the surfaces of their environments, with which they are in both hydrostatic (pressure) and diffusion (gas flow) equilibrium. When nuclei are stabilized, and not activated to growth or contraction by external pressure changes, the skin (surfactant) tension offsets both the Laplacian (film) tension and any mechanical help from surrounding tissue. Then all pressures and gas tensions are equal. However, on decompression, the seed pockets are surrounded by dissolved gases at high tension and can subsequently grow (bubbles) as surrounding gas diffuses into them. The rate at which bubbles grow, or contract, depends directly on the difference between tissue tension and local ambient pressure, effectively the bubble pressure gradient. At some point in time, a critical volume of bubbles, or separated gas, is established and bends symptoms become statistically more probable. On compression, the micronuclei are crunched down to smaller sizes across families, apparently stabilizing at new reduced size. Bubbles are also crunched by increasing pressure because of Boyle's law, and then additionally shrink if gas diffuses out of them. As bubbles get smaller and smaller, they probably restabilize as micronuclei.

   The RGBM postulates bubble seeds with lipid or aqueous surfactants. Bubble skins are assumed permeable under all anbient pressure, unlike the VPM. The size of seeds excited into growth is inversely proportional to the supersaturation gradient. RGBM excitation radii, r, start in the 0.01 μm range, far smaller than other dual phase models, because the RGBM tracks Boyle

expansion and bubble gas diffusion across the tissue seed interface (across the surfactant). At increasing pressure, bubble seeds permit gas diffusion at a slower rate. The RGBM assumes bubble skins are stabilized by surfactants over calculable time scales, producing seeds that are variably persistent in the body. Bubble skins are probably molecularly activated, complex, biosubstances found throughout the body. Whatever the formation process, the RGBM assumes the size distribution is exponentially decreasing in size, that is, more smaller seeds than larger seeds in exponential proportions. Skin response of the bubbles to pressure change is dictated by a material equation-of-state (EOS), again unlike the VPM. As stated, the RGBM diffuses gas from tissues to bubbles (and vice-versa) using a transfer equations across the film interface. This requires a mass transfer coefficient dependent on the gas solubility and diffusivity. The source of bubbles and seeds is probably tribonucleation due to muscle and tissue interfriction, and persistence time scales range from minutes to tens of hours.

4. Slow Tissue Compartments

   Based on concerns in multiday and heavy repetitive diving, with the hope of controlling staircasing gas buildup in exposures through critical tensions, slow tissue compartments (halftimes greater than 80 minutes) have been incorporated into some algorithms. Calculations, however, show that virtually impossible exposures are required of the diver before critical tensions are even approached, literally tens of hours of near continuous activity. As noted in many calculations, slow compartment cannot really control multidiving through critical tensions, unless critical tensions are reduced to absurd levels, inconsistent with nonstop time limits for shallow exposures. That is a model limitation, not necessarily a physical reality. The physical reality is that bubbles in slow tissues are eliminated over time scales of days, and the model limitation is that the arbitrary parameter space does not accommodate such phenomena.

   And that is no surprise either, when one considers that dissolved gas models are not suppose to track bubbles and free phases. Repetitive exposures do provide fresh dissolved gas for excited nuclei and growing free phases, but it is not the dissolved gas which is the problem just by itself. When bubble growth is considered, the slow compartments appear very important, because, therein, growing free phases are mostly left undisturbed insofar as surrounding tissue tensions are concerned. Bubbles grow more gradually in slow compartments because the gradient there is typically small, yet grow over longer time scales. When coupled to free phase dynamics, slow compartments are necessary in multidiving calculations.

   The RGBM incorporates a spectrum of tissue compartments, ranging from 1 min to 720 min, depending on gas mixture (helium, nitrogen, oxygen). Phase separation and bubble growth in slower compartments is a central focus in calculations over long time scales, and the same for fast tissue tissue compratments over short time scales, that is, scales over 2 or 3 times the compartment halftime.

5. Venous Gas Emboli While the numbers of venous gas emboli detected with ultrasound Doppler techniques can be correlated with nonstop limits, and the limits then used to fine tune the critical tension matrix for select exposure ranges, fundamental issues are not necessarily resolved by venous gas emboli measurements. First of all, venous gas emboli are probably not the direct cause of bends per se, unless they block the pulmonary circulation, or pass through the pulmonary traps and enter the arterial system to lodge in critical sites. Intravascular bubbles might first form at extravascular sites. According to studies, electron micrographs have highlighted bubbles breaking into capillary walls from adjacent lipid tissue beds in mice. Fatty tissue, draining the veins and possessing few nerve endings, is thought to be an extravascular site of venous gas emboli. Similarly, since blood constitutes no more than 8% of the total body capacity for dissolved gas, the bulk of circulating blood does not account for the amount of gas detected as venous gas emboli. Secondly, what has not been established is the link between ve-

nous gas emboli, possible micronuclei, and bubbles in critical tissues. Any such correlations of venous gas emboli with tissue micronuclei would unquestionably require considerable first-hand knowledge of nuclei size distributions, sites, and tissue thermodynamic properties. While some believe that venous gas emboli correlate with bubbles in extravascular sites, such as tendons and ligaments, and that venous gas emboli measurements can be reliably applied to bounce diving, the correlations with repetitive and saturation diving have not been made to work, nor important correlations with more severe forms of decompression sickness, such as chokes and central nervous system (CNS) hits.

Still, whatever the origin of venous gas emboli, procedures and protocols which reduce gas phases in the venous circulation deserve attention, for that matter, anywhere else in the body. The moving Doppler bubble may not be the bends bubble, but perhaps the difference may only be the present site. The propensity of venous gas emboli may reflect the state of critical tissues where decompression sickness does occur. Studies and tests based on Doppler detection of venous gas emboli are still the only viable means of monitoring free phases in the body.

The RGBM uses nonstop time limits tuned to recent Doppler measurements, conservatively reducing them along the lines originally sugested by Spencer (and others), but within the phase volume constraint. The Mares, Dacor, and Suunto implementations penalize ascent violations by requiring additional safety stop time dictated by risk analysis of the violation. All RGBM implementations supply user knobs for aggressive to conservative diving modifications, thru EOS in the full versions and M-values in the Haldane folded algorithms. Doppler scores over surface intervals are employed to calibrate RGBM bubble factors, both short and long intervals.

6. Multidiving

Concerns with multidiving can be addressed through variable critical gradients, then tissue tensions in Haldane models. While variable gradients or tensions are difficult to codify in table frameworks, they are easy to implement in digital meters. Reductions in critical parameters also result from the phase volume constraint, a constraint employing the separated volume of gas in tissue as trigger point for the bends, not dissolved gas buildup alone in tissue compartments. In the VPM the phase volume is proportional to the product of the dissolved-free gas gradient times a bubble number representing the number of gas nuclei excited into growth by the compression-decompression, replacing just slow tissue compartments in controlling multidiving. In the RGBM, the phase volume depends on the number of seeds excited and the Boyle and gas diffusion expansion-contraction of the seeds excited into growth.

In considering bubbles and free-dissolved gradients within critical phase hypotheses, repetitive criteria develop which require reductions in Haldane critical tensions or dissolved-free gas gradients. This reduction simply arises from lessened degree of bubble elimination over repetitive intervals, compared to long bounce intervals, and need to reduce bubble inflation rate through smaller driving gradients. Deep repetitive and spike exposures feel the greatest effects of gradient reduction, but shallower multiday activities are impacted. Bounce diving enjoys long surface intervals to eliminate bubbles while repetitive diving must contend with shorter intervals, and hypothetically reduced time for bubble elimination. Theoretically, a reduction in the bubble inflation driving term, namely, the tissue gradient or tension, holds the inflation rate down. Overall, concern is bubble excess driven by dissolved gas. And then both bubbles and dissolved gas are important. In such an approach, multidiving exposures experience reduced permissible tensions through lessened free phase elimination over time spans of two days. Parameters are consistent with bubble experiments, and both slow and fast tissue compartments must be considered.

The RGBM reduces the phase volume limit in multidiving by considering free phase elimination and buildup during surface intervals, depending on altitude, time, and depth of previous profiles,

Repetitive, multiday, and reverse profile exposures are tracked and impacted by critical phase volume reductions over appropriate time scales.

7. Adaptation

   Divers and caisson workers have long contended that tolerance to decompression sickness increases with daily diving, and decreases after a few weeks layoff, that in large groups of compressed air workers, new workers were at higher risk than those who were exposed to high pressure regularly. This acclimatization might result from either increased body tolerance to bubbles (physiological adaptation), or decreased number and volume of bubbles (physical adaptation). Test results are totally consistent with physical adaptation.

   Yet, there is slight inconsistency here. Statistics point to slightly higher bends incidence in repetitive and multiday diving. Some hyperbaric specialists confirm the same, based on experience. The situation is not clear, but the resolution plausibly links to the kinds of first dives made and repetitive frequency in the sequence. If the first in a series of repetitive dives are kept short, deep, and conservative with respect to nonstop time limits, initial excitation and growth are minimized. Subsequent dives would witness minimal levels of initial phases. If surface intervals are also long enough to optimize both free and dissolved gas elimination, any nuclei excited into growth could be efficiently eliminated outside repetitive exposures, with adapatation occurring over day intervals as noted in experiments. But higher frequency, repetitive and multiday loading may not afford sufficient surface intervals to eliminate free phases excited by earlier exposures, with additional nuclei then possibly excited on top of existing phases. Physical adaptation seems less likely, and decompression sickness more likely, in the latter case. Daily regimens of a single bounce dive with slightly increasing exposure times are consistent with physical adaptation, and conservative practices. The regimens also require deepest dives first. In short, acclimatization is as much a question of eliminating any free phases formed as it is a question of crushing or reducing nuclei as potential bubbles in repetitive exposures. And then time scales on the order of a day might limit the adapatation process.

   The RGBM generates bubble seed distributions on time scales of minutes for fast tissues and hours for slow tissues, adding new bubbles to existing bubbles in calculations. Phase volume limit points are also reduced by the added effects of new bubbles. Repetitive and reverse profile diving are impacted by bubble growth in the fast compartments, while flying after diving and multiday diving are affected by bubble growth in the slow compartments.