

Online with the RGBM: A Modern Phase Algorithm and Diveware Implementation

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Both Suunto and Abysmal Diving have released products incorporating a modern phase algorithm, called 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 inclusive (altitude, repetitive, mixed gas, decompression, saturation, nonstop exposures), treating both dissolved and free gas phase buildup and elimination. NAUI Technical Diving employed the RGBM to schedule nonstop and decompression training protocols on trimix, heliox, and nitrox while also testing gas switching alternatives for deep exposures. The RGBM has its roots in the earlier work of the Tiny Bubble Group at the University of Hawaii, drawing upon and extending the so-called Varying Permeability Model (VPM) to multiding, altitude, and mixed gas applications. While certainly not radical, The RGBM is both 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.

The Suunto VYPER is an RGBM-based decometer for recreational divers (plus nitrox), while ABYSS/RGBM is a licensed Abysmal Diving software product. On the Internet, the sites <http://www.suunto.fi/diving.index.html> and <http://www.abysmal.com/index.html> can be visited for information and description. Both are first-time-ever commercial products with realistic implementation of a diving phase algorithm across a wide spectrum of exposure extremes. And both accommodate user knobs for additional conservatism.

Here, our intent is to (just) look at the underpinnings of both 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 recent technical diving interest in deep stop modeling, and concerns with repetitive diving in the recreational community, phase modeling is timely and pertinent.

Impetus

Recent years have witnessed many changes and modifications to diving protocols and table procedures, such as shorter nonstop time limits, slower ascent rates, discretionary safety stops, ascending repetitive profiles, multilevel techniques, both faster and slower controlling repetitive tissues, smaller critical tensions (M-values), longer flying-after-diving surface intervals, and others. Stimulated by observation, Doppler technology, decompression meter development, theory, statistics, or safer diving consensus, these modifications affect a gamut of activity, spanning bounce to multiday diving. Of these changes, conservative nonstop time limits, no decompression safety stops, and slower ascent rates (around 30 *fsw/min*) are in vogue, and have been incorporated into many tables and meters. As you might expect, recent developments support them on operational, experimental, and theoretical grounds.

But there is certainly more to the story as far as table and meter implementations. To encompass such far reaching (and often diverse) changes in a unified framework requires more than the simple Haldane models we presently rely upon in 99% of our tables and dive computers. To model gas transfer dynamics, modelers and table designers need address both free and dissolved gas phases, their interplay, and their impact on diving protocols. Biophysical models of inert gas transport and bubble formation all try to prevent decompression sickness. Developed over years of diving application, they differ on a number of basic issues, still mostly unresolved today

1. the rate limiting process for inert gas exchange, blood flow rate (perfusion) or gas transfer rate across tissue (diffusion);
2. composition and location of critical tissues (bends sites);
3. the mechanistics of phase inception and separation (bubble formation and growth);
4. the critical trigger point best delimiting the onset of symptoms (dissolved gas buildup in tissues, volume of separated gas, number of bubbles per unit tissue volume, bubble growth rate to name a few);
5. the nature of the critical insult causing bends (nerve deformation, arterial blockage or occlusion, blood chemistry or density changes).

Such issues confront every modeler and table designer, perplexing and ambiguous in their correlations with experiment and nagging in their persistence. And here comments are confined just to Type I (limb) and II (central nervous system) bends, to say nothing of other types and factors. These concerns translate into a number of what decompression modelers call dilemmas that limit or qualify their best efforts to describe decompression phenomena. Ultimately, such concerns work their way into table and meter algorithms, with the same caveats. The RGBM treats these issues in a very natural way, many gory details of which are found in the References.

Computational Algorithms And Issues

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

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

Over the years, much attention has focused on supersaturation. Recent studies have shed much light on nucleation, excitation and bubble growth, even though in vitro Bubble aggregation, tissue damage, ischemia, and the whole question of decompression sickness trigger points are difficult to quantify in any model, and remain obscure. Complete elucidation of the interplay is presently asking too much. Yet, the development and implementation of better computational models is necessary to address problems raised in workshops, reports, publications, disclaimers, and as a means to safer diving. The computational issues of bubble dynamics (formation, growth, and elimination) are mostly outside the traditional 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.

The playoffs are schematized in Figure 1. 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. Consider the issues and approaches taken in the VYPER and ABYSS implementations

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 2 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 linearly with pressure for constant composition breathing mixture, and decreases linearly 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 by diffusion. Depending on time scales and rate coefficients, one or another (or both) processes dominate the exchange. However, for both the VYPER 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.

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, as depicted in Figure 3.

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 extravascular 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 4 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 5 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.

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.

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 (somewhere near 10 atm) 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.

Under compression-decompression, gas nuclei may grow as bubbles, depending on their effective bubble radius. Below a certain critical radius, r_c , listed in Table 1 as a function of pressure according to the VPM, 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 $.8 \mu m$, smaller than living cells, having dimensions starting at a few μm .

Table 1. Micronuclei Excitation Radii.

pressure $P \sim$ (fsw)	excitation radius $r_c \sim$ (microns)	pressure $P \sim$ (fsw)	excitation radius $r_c \sim$ (microns)
13	.89	153	.49
33	.80	173	.46
53	.72	193	.44
73	.66	213	.41
93	.61	233	.39
113	.57	253	.37
133	.53	273	.33

Micronuclei can be broadly classified as homogeneous or heterogeneous, depending upon their composition and that of the surrounding media. If the composition of both micronuclei and parent media are essentially the same, the nucleation process is termed homogeneous. If the composition of micronuclei and parent media differ, the nucleation process is termed heterogeneous. Spontaneously bubble formation in pure supersaturated liquids under explosive decompression is mainly homogeneous, while bubble formation on dust particles in supersaturated fluids is mostly heterogeneous. Homogeneous nucleation and bubble formation usually require large decompressions (many tens of atmospheres), while heterogeneous nucleation and bubble formation processes transpire with very small decompressions (tenths of atmospheres). Homogeneous nucleation in body tissue under nominal and controlled conditions of decompression appears much less likely than heterogeneous nucleation, considering pressure change and host of organic and inorganic body substances.

An underlying point can be made here. If nucleation sites are extinguished, reduced in number, or ill-disposed to excitation, bubble formation and risk are commensurately reduced. Regeneration times for classes of micronuclei are estimated to be near a week, underscoring physiological adaptation to recurring pressure environments. The mechanics of nucleation, stabilization, and bubble growth are fairly complex, with stabilization mechanisms only recently quantified. Source and generation mechanisms before stabilization are not well understood. Some candidates include cosmic radiation and charged particles, dissolved gases in fluids we drink, lymph draining tissues into veins, collisional coalescence, blood turbulence and vorticity, exercise, the stomach, and the thin air-blood endothelium in the lungs. Once formed, micronuclei must stabilize very rapidly with surfactant material. Passing through the pulmonary filters of the lungs, only sub-micron sizes might survive. If nuclei are persistent, it is not clear that they populate all tissue sites, nor possess the same size distributions. Some can argue that gel findings are not relevant because biological fluids are formed, and contained, in a sealed environment (the body), but the Strauss and Yount studies confirm the existence of preformed gas micronuclei in serum and egg albumin. Nuclei seem to pervade all manner of fluids.

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. For a given difference between ambient and gas-vapor pressure, only one radius is stable. Changes in ambient, gas, or vapor pressures will cause the nuclei to either grow, or contract. But even if stable hydrostatically, bubbles and nuclei, because of constricting surface tension, will eventually collapse as gas and vapor diffuse out of the assembly. For instance, an air bubble of radius 10^{-3} cm will dissolve in saturated water in about 6 sec, and even faster if the water is undersaturated or the bubble is smaller. In saturated solutions, bubbles will grow by diffusion, and then tend to be quickly lost at free surfaces as buoyant forces raise them up. A 10^{-2} cm air bubble rises at the rate of 1.5 cm/sec in water. If nuclei are to persist in water, or for that matter, any liquid media, some mechanism must prevent their dissolution or buoyant exit.

The RGBM postulates bubble seeds with varying permeability. Bubble skins are assumed permeable down to 10 atm crushing pressure. The size of seeds excited into growth is inversely proportional to the supersaturation gradient. Beyond 10 atm, bubble seeds permit gas diffusion at a slower rate.

Surfactants

A number of possibilities have been suggested to account for the presence of persistent, or stabilized, nuclei in undersaturated liquids, liquids that have been boiled, or denucleated. Crevices in the liquid, or surrounding boundary, may exert mechanical pressure on gas nuclei, holding them in place. Microscopic dust, or other impurities, on which gas and vapor are deposited, are stabilized already. Surface activated molecules, (such as hydrogen and hydroxyl ions in water), or surface activated skins formed from impurities may surround the nuclei and act as rigid spheres, offsetting constrictive surface tension, preventing diffusion of gas out of the nuclei and collapse. In all cases, the end result is a family, or group of families, of persistent nuclei. Time scales for stabilization and persistence of nuclei would obviously equate to the strength and persistence of stabilizing mechanism. Experimentally, trying to differentiate stabilization modes is very difficult, because (eventual) growth patterns of nuclei are the same in all cases. The ultimate crumbling of surrounding shells, release of crevice mechanical pressure, removal of dust and impurity nucleation centers, and deactivation of surface chemicals leads to the onset of cavitation and bubble growth.

Water, gasoline, glycerin, and salad oil are clearly liquids. Pancake syrup, paster, eggwhite, silly putty, paint, glue, and soap are also liquids, that is, they flow on the application of stress, but border on classification otherwise. In mechanical response, the latter class differs from each other as much as they differ from solids. And the response is variable in time. Syrup becomes sticky as it dries. Dishwashing soap often dries into light flakes. Silly putty flows on tilt, but shatters on sudden impact. Airplane glue is springy and rubbery.

Substances in the latter category are called structured fluids, owing their distinctive and unusual properties to large polyatomic composites, many times the size of a water molecule. Fluids containing polyatomic structures manifest a wide variety of mechanical response and self organization. Body tissues and fluids host an uncountable variety of organic and inorganic matter, with many biochemical substances falling into structured fluid category. Among the structured fluids, a class of self assemblies, called surfactants, are very interesting, possessing properties which can stabilize microbubbles in various stages of evolution by offsetting surface tension.

A surfactant is a structured fluid which is ambiphillic, incorporating parts that assume preferential orientations at water-oil (immiscible) interfaces. A surfactant molecule usually consists of a bulky ion at one end, and a counter ion at the other. Isolated molecules cannot usually exist in one media type, or the other, but instead orient themselves into micelles, configurations in which like parts clump together, that is head in one substance and tail in the other. Micelles typically possess diameters near 10^{-3} μ m, and render the interfaces unlike anything measured in the components. Lipid-aqueous tissue interfaces potentially present favorable environments for surfactants.

Under certain conditions, a surfactant can reduce interfacial surface tension, allowing the interface to grow and wrap around itself. The result is a microbundle full of alternating surfaces and interfaces, spherical in structure to minimize thermodynamic energy constraints. Many substances may be bound up in the microbundle. If small gas nuclei, but typically much larger than a micelle, are in contact with the interfaces, or surfactants directly, a spherical gas micronucleus-microemulsion can develop, varying in size and surfactant content. The assembly is stable when the effective surface tension is zero, when surfactant skin pressure just balances mechanical (Laplace) surface tension. If the effective surface tension of the microbubble, γ , is not zero, the collection will grow or contract until stable, or disassemble. In the case of gas microemulsions, the surfactant is thought to coat the inside boundary layer mostly, with free gas in the interior. The actual picture is probably more complex, but such a picture can be drawn for computational simplicity. Surfactant stabilized micronuclei may theoretically destabilize under compression-decompression processes in diving, perhaps spawning bubble growth fueled by high gas tension in surrounding media. Microbubbles may remain at the interfaces, but probably migrate. Sources of initial gas nuclei, surfactant composition, and tissue sites await description.

The RGBM assumes bubble skins are stabilized by surfactants over unknown time scales, but that the seeds are 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.

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.

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 venous 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 suggested by Spencer (and others), but within the phase volume constraint. The VYPER implementation penalizes ascent violations by requiring additional safety stop time dictated by risk analysis of the violation.

Multidiving

Concerns with multidiving can be addressed through variable critical gradients, or equivalently 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. 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. And it replaces just slow tissue compartments in controlling multidiving.

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 deeper-than-previous exposures are tracked and impacted by critical phase volume reductions over appropriate time scales.

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 adaptation 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 adaptation process.

The RGBM generates replacement bubble seed distributions on time scales of days, adding new bubbles to existing bubbles in calculations. Phase volume limit points are also reduced by the added effects of new bubbles.

RGBM And Haldane Comparisons

So, having waded through the foregoing, a next question is how does the RGBM compare with classical Haldane models as far as staging ascents, limiting multiexposures, and treating mixed gases. Generally, for short nonstop air diving, the RGBM reproduces the Spencer limits. For multiday diving in spans shorter than 1-3 hrs, the RGBM reduces nonstop limits by 10% to 20% depending on surface interval, depth, altitude, and duration of present and previous dive. Multiday diving is impacted to lesser degree. Some comparisons appear in Table 2 for 3 days of repetitive air diving (120 fsw/10 min twice a day, separated by 45 min surface interval). Computer choices are illustrative, not indictive.

Table 2. Nonstop Limits For VYPER/RGBM And Haldane Air Multidiving

Computer/Algorithm	Dive 1 Mins	Dive 2 Mins	Dive 3 Mins	Dive 4 Mins	Dive 5 Mins	Dive 6 Mins
Vyper / RGBM	10	9	9	8	8	7
Delphi / Haldane	10	10	10	10	10	10
DC11 / Haldane	6	6	6	6	6	6
Aladin / Haldane	8	8	8	8	8	8
Source / Haldane	12	9	12	9	12	9

The VYPER/RGBM (first dive) nonstop limits (depth/time) are 150/6, 140/7, 130/9, 120/10, 110/13, 100/17, 90/22, 80/28, 70/36, 60/51, 50/69, and 40/120. In the mixed gas arena, Table 3 lists nonstop time limits for ranged trimix, that is, 13% to 17% helium, 61% to 53% nitrogen, and 26% to 30% oxygen, according to ABYSS/RGBM and ABYSS/ZHL (Buhlmann).

Table 3. Trimix Nonstop Limits For ABYSS/RGBM And ABYSS/ZHL (Haldane).

Depth (FSW)	Abyss/RGBM (min)	Abyss/ZHL (min)
80	28	26
90	23	22
100	19	18
110	16	15
120	14	13
130	12	11
140	11	10
150	10	9

These limits are used by NAUI Technical Diving for training purposes. While both sets of nonstop time limits are different in Tables 2 and 3, the more dramatic effects of the RGBM show up for deep staging, as seen in Table 4.

Comparative deep schedules for a trimix dive to 250 fsw for 30 min are contrasted, following a switch to air at 100 fsw and a switch to pure oxygen at 20 fsw on the way up. ABYSS/RGBM and ABYSS/ZHL are again employed, but with and without conservative safety knobs. In the case of ABYSS/ZHL, the outgassing tissue halftimes are increased by 1.5 in the conservative case, while for ABYSS/RGBM the bubble excitation radius is increased by 1.2 for purposes of comparison. Deeper stops are noticeably requisite in ABYSS/RGBM, but total decompression times are less than ABYSS/ZHL. The trimix is 33% helium, 51% nitrogen, and 16% oxygen.

Table 4. Deep Schedules According To ABYSS/RGBM And ABYSS/ZHL (Haldane)

Stop	Depth (FSW)	Abyss/ZHL (min) (Standard)	Abyss/RGBM (min) (Standard)	Abyss/ZHL (min) (Safer)	Abyss/RGBM (min) (Safer)
1	180	0	0	0	1
2	170	0	1	0	1
3	160	0	1	0	1
4	150	0	1	0	1
5	140	0	1	0	2
6	130	0	2	0	2
7	120	0	2	0	2
8	110	0	2	1	2
9	100	0	2	2	2
10	90	2	2	3	3
11	80	2	2	4	3
12	70	2	3	5	4
13	60	5	5	8	6
14	50	7	6	12	7
15	40	12	9	18	9
16	30	18	12	28	13
17	20	16	10	28	11
18	10	28	16	48	18
		93	77	147	88

That in a nutshell is a comparison of major differences between phase and dissolved gas models. The phase models recover dissolved gas models for short and nominal exposures, but require deeper stops and shorter decompression times for longer and exceptional exposures.

RGBM Software Package

1. A rundown of the software configuration of the RGBM used in full blown simulations is as follows. The package is under constant refinement and updating.
2. Module Three major routines (RGBMNX, RGBMHX, RGBMTMX) for nitrox, heliox, and trimix.
3. Source Code 1640 Lines
4. Language/Compiler FORTRAN 77/90, BASIC.
5. CRAY YMP Running Time 1 sec for deep trimix profile with 5 gas switches on way up.
6. Input altitude, bottom mixture, ascent/descent rate, switch levels and gas mixtures, pre-dive breathing gas, safety knobs, previous dive history.
7. Output controlling tissue compartments, stop depth and times, supersaturation gradient, permissible supersaturation, effective bubble and gas parameters, critical phase volume, dive profile.
8. \$3800

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BIOSKETCH

Bruce Wienke is a Senior Project Leader in the Nuclear 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 five monographs (Physics, Physiology And Decompression Theory For The Technical And Commercial Diver, High Altitude Diving, Basic Diving Physics And Applications, 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 Warfare Units, above and below water. Bruce heads Southwest Enterprises, a consulting company for computer research and applications in wide areas of applied science.

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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.

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Figure 1. Free And Dissolved Gas Gradients

Staging diver ascents is a mini-max problem. To eliminate dissolved gas, the diver is brought as close to the surface as possible. To eliminate free phases, the diver is kept at depth. Obviously, staging diver ascents with dual phase (free and dissolved gases) treatments is a payoff.

Figure 2. Inherent Unstauration

Tissue and blood are understaured with respect to ambient pressure, that is, summed partial tensions of oxygen, nitrogen, water vapor, and carbon dioxide in tissue and blood are always less than ambient pressure at equilibration. Carbon dioxide produced in metabolic processes is 25 times more soluble than oxygen consumed, and hence, by Henry's law, exerts a smaller partial pressure than the oxygen replaced. Tensions are listed in \$fsw\$ below.

Figure 3. Dual Phase Gas Diffusion Pathways

The hope in staging diver ascents is to eliminate both free and dissolved gas phases as rapidly as possible through the capillary blood flow. Dumping dissolved phases into existing free phases increases the separated volume, reducing diver ascent choices.

Figure 4 Bubble Pressure Balance

The total pressure inside a bubble equals the sum of the ambient pressure, P , plus effective surface tension, $2\gamma/r$, with r the bubble radius, shown below. Free gas pressures from oxygen, nitrogen, water vapor, and carbon dioxide (for air) provide the balance pressure. When surface tension, γ , is zero a bubble (or bubble seed) is stabilized and will not collapse spontaneously under just constriction. Surfactants can coat the inside of the bubble, accomplishing this, but allowing gases to diffuse into or out of the bubble.

Figure 5. Bubble Gas Diffusion

A bubble in hydrostatic equilibrium will grow or contract, depending on its size and relative pressure gradients between free gases on the inside, and free or dissolved gases on the outside. A critical radius, r_c , separates growing from contracting bubbles whenever gradients exist. Bubbles with radii larger than r_c will grow, while bubbles with radii less than r_c will contract. Surfactants coating the inside of the bubble may render the effective surface tension, γ , zero (resisting the constrictive effect of film tension), permitting gases to diffuse into or out of the bubble (permeable) or impeding the exchange to varying degrees (impermeable). The depiction below is for an air bubble.