ABYSS/REDUCED GRADIENT BUBBLE MODEL: ALGORITHM, BASES, REDUCTIONS, AND COUPLING TO ZHL CRITICAL PARAMETERS

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Although the manifestations of DCI are statistically distributed, tables and meters employ deterministic staging models, with models broadly categorized as Haldane (dissolved phase) or as bubble (combination of dissolved and free phases). The Reduced Gradient Bubble Model (RGBM) is one such dual phase model developed for a very wide spectrum of diving activities (bounce, altitude, decompression, saturation, repetitive, multiday). Over appropriate diving ranges and exposures, the general features of the RGBM can be retrofitted to any more limited model, like a Haldane dissolved gas model, across the other model nonstop time limits and critical staging parameters (Workman M – Values, Buhlmann a and b) This extended writeup addresses the reduction and linkage of the RGBM to the ABYSS/ZHL (Haldane) meter algorithm, using the ABYSS/ZHL desired nonstop limits and critical tensions. The process involves both profile and parameter fitting in the synthesis, requiring fairly powerful computers. All techniques and model essentials are contained in this document, however, only the fundamental relationships are presented and discussed for simplicity and (hopefully) clarity. The full blown iterative ABYSS/RGBM is also described and discussed.

INTRODUCTION

Overview

We first discuss DCI risk and coupled statistics, then return to specific description of the RGBM, and the algorithm used in diving applications. The next section details the ABYSS/RGBM and synthesis linking the Haldane (ABYSS/ZHL) and phase (RGBM) models. A discussion of the mulitdiving fractions, f, and their relationship to the RGBM and ABYSS algorithms is also sketched. Parts 1 and 2 are general in content, focusing on decompression risk and the full blown RGBM. Part 3 is specific to the ABYSS/ZHL implementation of the RGBM, using the ABYSS critical parameters and exposure times, and details all linkages to the RGBM, profile and parameter fitting, and data employed in coupling analysis. Part 4 contrasts layman differences between phase (ABYSS/RGBM and RGBM) and dissolved gas models, focusing on software (ABYSS) and meter (Haldane) predictions for test profiles.

Models And Data

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 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 DCI puzzle, the risk and statistics of decompressing divers are straightforward. And folding of DCI risk and statistics over data and model assumptions is perhaps the best means to safety and model closure.

PART 1: STATISTICS AND RISK ANALYSIS

Decompression Risk And Statistics

Computational algorithms, tables, and manned testing are requisite across a spectrum of activities. And the potential of electronic devices to process tables of information or detailed equations underwater is near maturity, with virtually any algorithm or model amenable to digital implementation. Pressures for even more sophisticated algorithms are expected to grow.

Still computational models enjoy varying degrees of success. More complex models address a greater number of issues, but are harder to codify in decompression tables. Simpler models are easier to codify, but are less comprehensive. Some models are based on first principles, but many are not. Application of models can be subjective in the absence of definitive data, the acquisition of which is tedious, sometimes controversial, and often ambiguous. If deterministic

models are abandoned, statistical analysis can address the variability of outcome inherent to random occurrences, but so called dose-reponse characteristics of statistical analysis are very attractive in the formulation of risk tables. Applied to decompression sickness incidence, tables of comparative risk offer a means of weighing contributing factors and exposure alternatives. At the basis of statistical and probabilistic analyses of decompression sickness is the binomial distribution. The binomial distribution is the fundamental frequency distribution governing random events:

1. Binomial Distribution

   Decompression sickness is a hit, or no hit, situation. Statistics are binary, as in coin tossing. Probabilities of occurrence are determined from the binomial distribution, which measures the numbers of possibilities of occurrence and nonoccurrence in any number of events, given the incidence rate. Specifically, the probability, P, in a random sample of size, N, for n occurrences of decompression sickness and m nonoccurrences, takes the form,

$$P(n) = \frac{N!}{n!\ m!}\ p^n q^m \quad , \tag{1}$$

   with,

$$n + m = N \quad , \tag{2}$$

   p the underlying incidence rate (average number of cases of decompression sickness), and q,

$$q = 1 - p \quad , \tag{3}$$

   the underlying nonincidence. The discrete probability distributions, P, are the individual terms of the binomial expansion of $(p+q)^N$,

$$(p+q)^N = \sum_{n=0}^{N} P(n) = 1 \quad . \tag{4}$$

   In risk analysis, p and q are also the failure and success rates, gleaned, for instance, from random or strategic sampling of arbitrary lot sizes. Obviously, the larger the sample size, the better are the estimates of p or q. Once p or q is determined, the binomial statistics and probabilities are also fixed. The statistical mean, M, and variance, s, are given by,

$$M = \sum_{n=1}^{N} nP(n) = pN \quad , \tag{5}$$

$$s = \sum_{n=1}^{N} (n-M)^2\ P(n) = pqN \quad , \tag{6}$$

   the usual measures of a statistical distribution. The square root of the variance is the standard deviation. The cumulative probability for more than n cases of decompression sickness, $P_>(n)$, is written,

$$P_>(n) = \sum_{j=n+1}^{N} P(j) = 1 - \sum_{j=0}^{n} P(j) \quad , \tag{7}$$

   and the probability of less than n cases, $P_<(n)$, is similarly,

$$P_<(n) = \sum_{j=0}^{n-1} P(j) = 1 - \sum_{j=n}^{N} P(j) \quad . \tag{8}$$

   The probability of nonoccurrence in any set of N trials is simply,

$$P(0) = q^N \quad , \tag{9}$$

while the probability of total occurrence in the same number, N, of trials is given by,

$$P(N) = p^N \quad . \tag{10}$$

The binomial distribution is a special case of the multinomial distribution describing processes in which several results having fixed probabilities, p_l, q_l, for $l = 1, L$, are possible. Separate probabilities are given by the individual terms in the general multinomial expansion,

$$(p_1 + q_1 + ... + p_L + q_L)^N = \sum_{n_1,...,n_{L-1}=0}^{N} P(n_1, ..., n_{L-1}) = 1 \quad , \tag{11}$$

as in the binomial case. The normal distribution is a special case of the binomial distribution when N is very large and variables are not necessarily confined to integer values. The Poisson distribution is another special case of the binomial distribution when the number of events, N, is also large, but the incidence, p, is small.

2. Normal Distribution

The normal distribution is an analytic approximation to the binomial distribution when N is very large, and n, the observed value (success or failure rate), is not confined to integer values, but ranges continuously,

$$-\infty \leq n \leq \infty \quad . \tag{12}$$

Normal distributions thus apply to continuous observables, while binomial and Poisson distributions apply to discontinuous observables. Statistical theories of errors are ordinarily based on normal distributions.

For the same mean, $M = pN$, and variance, $s = pqN$, the normal distribution, P, written as a continuously varying function of n,

$$P(n) = \frac{1}{(2\pi s)^{1/2}} \, exp \, [-(n-M)^2/2s] \quad , \tag{13}$$

is a good approximation to the binomial distribution in the range,

$$\frac{1}{N+1} < p < \frac{N}{N+1} \quad , \tag{14}$$

and within three standard deviations of the mean,

$$pN - 3\,(pqN)^{1/2} \leq n \leq pN + 3\,(pqN)^{1/2} \quad . \tag{15}$$

The distribution is normalized to one over the real infinite interval,

$$\int_{-\infty}^{\infty} P dn = 1 \quad . \tag{16}$$

The probability that a normally distributed variable, n, is less than or equal to b is,

$$P_<(b) = \int_{-\infty}^{b} P dn \quad , \tag{17}$$

while the corresponding probability that n is greater than or equal to b is,

$$P_>(b) = \int_{b}^{\infty} P dn \quad . \tag{18}$$

The normal distribution is extremely important in statistical theories of random variables. By the central limit theorem, the distribution of sample means of identically distributed random variables is approximately normal, regardless of the actual distribution of the individual variables.

3. Poisson Distribution

The Poisson distribution is a special case of the binomial distribution when N becomes large, and p is small, and certainly describes all discrete random processes whose probability of occurrence is small and constant. The Poisson distribution applies substantially to all observations made concerning the incidence of decompression sickness in diving, that is, $p << 1$ as the desired norm. The reduction of the binomial distribution to the Poisson distribution follows from limiting forms of terms in the binomial expansion, that is, $P(n)$.

In the limit as N becomes large, and p is much smaller than one, we have,

$$\frac{N!}{(N-n)!} = N^n \ , \tag{19}$$

$$q^m = (1-p)^{N-n} = exp\ (-pN) \ , \tag{20}$$

and therefore the binomial probability reduces to,

$$P(n) = \frac{N^n p^n}{n!}\ exp\ (-pN) = \frac{M^n}{n!}\ exp\ (-M) \ , \tag{21}$$

which is the discrete Poisson distribution. The mean, M, is given as before,

$$M = pN \tag{22}$$

and the variance, s, has the same value,

$$s = pN \ , \tag{23}$$

because q is approximately one. The cumulative probabilities, $P_>(n)$ and $P_<(n)$, are the same as those defined in the binomial case, a summation over discrete variable, n. It is appropriate to employ the Poisson approximation when $p \leq .10$, and $N \geq 10$ in trials. Certainly, from a numerical point of view, the Poisson distribution is easier to use than than binomial distribution. Computation of factorials is a lesser task, and bookkeeping is minimal for the Poisson case.

In addition to the incidence of decompression sickness, the Poisson distribution describes the statistical fluctuations in such random processes as the number of cavalry soldiers kicked and killed by horses, the disintegration of atomic nuclei, the emission of light quanta by excited atoms, and the appearance of cosmic ray bursts. It also applies to most rare diseases.

Probabilistic Decompression

Table 1 lists corresponding binomial decompression probabilities, $P(n)$, for 1% and 10% underlying incidence (99% and 90% nonincidence), yielding 0, 1, and 2 or more cases of decompression sickness. The underlying incidence, p, is the (fractional) average of hits.

As the number of trials increases, the probability of 0 or 1 occurrences drops, while the probability of 2 or more occurences increases. In the case of 5 dives, the probability might be as low as 5%, while in the case of 50 dives, the probability could be 39%, both for $p = .01$. Clearly, odds even percentages would require testing beyond 50 cases for an underlying incidence near 1%. Only by increasing the number of trials for fixed incidences can the probabilities be increased. Turning that around, a rejection procedure for 1 or more cases of decompression sickness at the 10% probability level requires many more than 50 dives. If we are willing to lower the confidence of the acceptance, or rejection, procedure, of course, the number of requisite trials drops. Table 1 also shows that the test practice of accepting an exposure schedule following 10 trials without incidence of decompression sickness is suspect, merely because the relative probability of nonincidence is high, near 35%.

Questions as to how safe are decompression schedules have almost never been answered satisfactorily. As seen, large numbers of binary events are required to reliably estimate the underlying incidence. One case of decompression sickness in 30 trials could result from an underlying incidence, p, bounded by .02 and .16 roughly. Tens more of trials are necessary to shrink those bounds.

Table 1. Probabilities Of Decompression Sickness For Underlying Incidences

N (dives)	n (hits)	$P(n)$ $p = .01$ $q = .99$	$P(n)$ $p = .10$ $q = .90$
5	0	.95	.59
	1	.04	.33
	2 or more	.01	.08
10	0	.90	.35
	1	.09	.39
	2 or more	.01	.26
20	0	.82	.12
	1	.16	.27
	2 or more	.02	.61
50	0	.61	.01
	1	.31	.03
	2 or more	.08	.96

Biological processes are highly variable in outcome. Formal correlations with outcome statistics are then generally requisite to validate models against data. Often, this correlation is difficult to firmly establish (couple of percent) with fewer than 1,000 trial observations, while ten percent correlations can be obtained with 30 trials, assuming binomial distributed probabilities. For decompression analysis, this works as a disadvantage, because often the trial space of dives is small. Not discounting the possibly small trial space, a probabilistic approach to the occurrence of decompression sickness is useful and necessary. One very successful approach, developed and tuned by Weathersby, and others for decompression sickness in diving, called maximum likelihood, applies theory or models to diving data and adjusts the parameters until theoretical prediction and experimental data are in as close agreement as possible.

Validation procedures require decisions about uncertainty. When a given decompression procedure is repeated with different subjects, or the same subjects on different occasions, the outcome is not constant. The uncertainty about the occurrence of decompression sickness can be quantified with statistical statements, though, suggesting limits to the validation procedure. For instance, after analyzing decompression incidence statistics for a set of procedures, a table designer may report that the procedure will offer an incidence rate below 5%, with 90% confidence in the statement. Alternatively, the table designer can compute the probability of rejecting a procedure using any number of dive trials, with the rejection criteria any arbitrary number of incidences. As the number of trials increases, the probability of rejecting a procedure increases for fixed incidence criteria. In this way, relatively simple statistical procedures can provide vital information as to the number of trials necessary to validate a procedure with any level of acceptable risk, or the maximum risk associated with any number of incidences and trials.

One constraint usually facing the statistical table designer is a paucity of data, that is, number of trials of a procedure. Data on hundreds of repetitions of a dive profile are virtually nonexistent, excepting bounce diving perhaps. As seen, some 30-50 trials are requisite to ascertain procedure safety at the 10% level. But 30-50 trials is probably asking too much, is too expensive, or generally prohibitive. In that case, the designer may try to employ global statistical measures linked to models in a more complex trial space, rather than a single profile trial space. Integrals of risk parameters, such as bubble number, supersaturation, separated phase, etc., over exposures in time, can be defined as probability measures for incidence of decompression sickness, and the maximum likelihood method then used to extract appropriate constants:

1. Maximum Likelihood

   We can never measure any physical variable exactly, that is, without error. Progressively more elaborate experimental or theoretical efforts only reduce the possible error in the determination. In extracting parameter estimates from data sets, it is necessary to also try to minimize the error (or data scatter) in the extraction process. A number of techniques are available to the analyst, including the well known maximum likelihood approach.

   The measure of any random occurrence, p, can be a complicated function of many parameters, $x = (x_k, k = 1, K)$, with the only constraint,

$$0 \leq p(x) \leq 1 \ , \tag{24}$$

for appropriate values of the set, x. The measure of nonoccurence, q, is then by conservation of probability,

$$q(x) = 1 - p(x) \ , \tag{25}$$

over the same range,

$$0 \leq q(x) \leq 1 \ . \tag{26}$$

Multivalued functions, $p(x)$, are often constructed, with specific form dictated by theory or observation over many trials or tests. In decompression applications, the parameters, x, may well be the bubble-nucleation rate, number of venous gas emboli, degree of supersaturation, amount of pressure reduction, volume of separated gas, ascent rate, or combinations thereof. Parameters may also be integrated in time in any sequence of events, as a global measure, though such measures are more difficult to analyze over arbitrary trial numbers.

The likelihood of any outcome, Φ, of N trials is the product of individual measures of the form,

$$\Phi(n) = p^n q^m = p^n (1-p)^m \ , \tag{27}$$

given n cases of decompression sickness and m cases without decompression sickness, and,

$$n + m = N \ . \tag{28}$$

The natural logarithm of the likelihood, Ψ, is easier to use in applications, and takes the form,

$$\Psi = \ln \ \Phi = n \ \ln \ p + m \ \ln \ (1-p) \ , \tag{29}$$

and is maximized when,

$$\frac{\partial \Psi}{\partial p} = 0 \ . \tag{30}$$

In terms of the above, we then must have,

$$\frac{n}{p} - \frac{m}{1-p} = 0 \ , \tag{31}$$

trivially requiring,

$$p = \frac{n}{n+m} = \frac{n}{N} \ , \tag{32}$$

$$1 - p = q = \frac{m}{n+m} = \frac{m}{N} \ . \tag{33}$$

Thus, the likelihood function is maximized when p is the actual incidence rate, and q is the actual nonincidence rate. The multivalued probability functions, $p(x)$, generalize in the maximization process according to,

$$\frac{\partial \Psi}{\partial p} = \sum_{k=1}^{K} \frac{\partial \Psi}{\partial x_k} \frac{\partial x_k}{\partial p} = 0 \ , \tag{34}$$

satisfied when,

$$\frac{\partial \Psi}{\partial x_k} = 0 \ \ for \ k = 1, \ K \ . \tag{35}$$

In application, such constraints are most easily solved on computers, with analytical or numerical methods.

In dealing with a large number of decompression procedures, spanning significant range in depth, time, and environmental factors, an integrated approach to maximum likelihood and risk is necessary. Integral measures, $p(x,t)$ and $q(x,t)$, can be defined over assumed decompression risk, $\zeta(x,t)$,

$$p(x,t) = 1 - exp \ \left[- \int_0^t \ \zeta(x,t') dt' \right] \ , \tag{36}$$

$$q(x,t) = exp\ \left[-\int_0^t\ \zeta(x,t')dt' \right]\ , \tag{37}$$

with t' any convenient time scale, and ζ any assumed risk, such as bubble number, saturation, venous emboli count, etc. as mentioned. Employing $p(x,t)$ and $q(x,t)$ in the likelihood function, and then maximizing according to the data, permits maximum likelihood estimation of $\zeta(x,t)$. Such an approach can be employed in decompression table fabrication, yielding good statistical estimates on incidence rates as a function of exposure factors.

2. Saturation Bends Probability

Many factors contribute to bends susceptibility. Age, obesity, temperature, physical condition, alcohol, and cigarettes are a few. Whatever the contributing factors, the distribution of bends depths for saturation exposures has been characterized in terms of the saturation tension, Q, and ambient pressure, P, by Hills. This characterization is not only of academic interest, but is also useful in assigning formal risk to decompression formats.

The distribution of saturation bends depths, χ, fits a Weibull function. This is true for all breathing mixtures, nitrox, heliox, trimix, etc. If cumulative fraction of air bends cases up to G is χ, the survivor fraction, $1-\chi$, satifies,

$$\ln\ (1-\chi) = -\left[\frac{G-14.3}{25.1} \right]^{4.73} \tag{38}$$

for cumulative bends probability, χ, the usual integral over bends risk, ζ, as a function of gradient, G,

$$\chi = \int_0^G \zeta(G')dG' \tag{39}$$

with saturation bends gradient, G, measured in fsw,

$$G = Q - P \tag{40}$$

As the gradient grows, the survivor function approaches zero exponentially. The smallest bends gradient is 14.3 fsw, which can be contrasted with the average value of 26.5 fsw. The root mean square gradient is 27.5 fsw. At 27 fsw, the survivor fraction is 0.96, while 67% of survivors fall in the range, 26.5 ± 7.6 fsw, with 7.6 fsw the standard deviation. For gas mixtures other than air, the general form is given by,

$$\ln\ (1-\chi) = -\varepsilon \left[\frac{(P_f - 20.5)}{(P_i - 33.0)} - \frac{1}{f_i} \right]^{\delta} \tag{41}$$

where f_i is the total volume fraction of inert breathing gases, for $G = P_f - P_i$, and with ε, δ constants.

The efficiency of the Weibull distribution in providing a good fit to the saturation data is not surprising. The Weibull distribution enjoys success in reliability studies involving multiplicities of fault factors. It obviously extends to any set of hyperbaric or hypobaric exposure data, using any of the many parameter risk variables described above.

3. Risk Tables

A global statistical approach to table fabrication consists of following a risk measure, or factor p, throughout and after sets of exposures, tallying the incidence of DCI, and then applying maximum likelihood to the risk integral in time, extracting any set of risk constants optimally over all dives in the maximization procedure. In analyzing air and helium data, Weathersby assigned risk as the difference between tissue tension and ambient pressure divided by ambient pressure. One tissue was assumed, with time constant ultimately fixed by the data in ensuing maximum likelihood analysis. The measure of nonincidence, q, was taken to be the exponential of risk integrated over all exposure time,

$$q(\kappa,\tau) = exp\ \left[-\int_0^{\infty}\ \zeta(\kappa,\tau,t')dt' \right]\ , \tag{42}$$

$$\zeta(\kappa,\tau,t') = \kappa\,\frac{p(t') - p_a}{p_a} \quad , \tag{43}$$

with κ a constant determined in the likelihood maximization, p_a ambient pressure, and $p(t')$ the instantaneous Haldane tension for tissue with halftime, τ, also determined in the maximization process, corresponding to arbitrary tissue compartments for the exposure data. Other more complex likelihood functions can also employed, for instance, the separated phase volume according to the varying permeability and reduced gradient bubble models,

$$\zeta(\kappa,\xi,\tau,t') = \kappa\Delta n G(t') \quad , \tag{44}$$

$$\Delta = \left[1 - \frac{r}{\xi}\right] \quad , \tag{45}$$

with Δn the permissible bubble excess, r the bubble radius, G the bubble diffusion gradient (dissolved-free gas), and κ and ξ constants determined in the fit maximization of the data. Another risk possibility is the tissue ratio,

$$\zeta(\kappa,\tau,t') = \kappa\,\frac{p(t')}{p_a} \quad , \tag{46}$$

a measure of interest in altitude diving applications.

Hundreds of air dives were analyzed using this procedure, permitting construction of decompression schedules with 95% and 99% confidence (5% and 1% bends incidence). These tables were published by US Navy investigators, and Table 2 tabulates the corresponding nonstop time limits ($p = .05, .01$), and also includes the standard US Navy (Workman) limits for comparison. Later re-evaluations of the standard set of nonstop time limits estimate an underlying incidence rate of 1.25% for the limits. In actual usage, the incidence rates are below .001%, because users do not dive to the limits generally.

Table 2. Nonstop Time Limits For 1% And 5% Incidence Rates

depth $d\ (fsw)$	nonstop limit $t_n\ (min)$ $p = .05$	nonstop limit $t_n\ (min)$ $p = .01$	nonstop limit $t_n\ (min)$ US Navy
30	240	170	
40	170	100	200
50	120	70	100
60	80	40	60
70	80	25	50
80	60	15	40
90	50	10	30
100	50	8	25
110	40	5	20
120	40	5	15
130	30	5	10

Implicit in such formulations of risk tables are the assumptions that a given decompression stress is more likely to produce symptoms if it is sustained in time, and that large numbers of separate events may culminate in the same probability after time integration. Though individual schedule segments may not be replicated enough to offer total statistical validation, categories of predicted safety can always be grouped within subsets of corroborating data. Since the method is general, any model parameter or meaningful index, properly normalized, can be applied to decompression data, and the full power of statistical methods employed to quantify overall risk. While powerful, such statistical methods are neither deterministic nor mechanistic, and cannot predict on first principles. But as a means to table fabrication with quoted risk, such approaches offer attractive pathways for analysis.

Model Validation

Validation procedures for schedules and tables can be quantified by a set of procedures based on statistical decompression analysis:

1. select or construct a measure of decompression risk, or a probabilistic model;
2. evaluate as many dives as possible, and especially those dives similar in exposure time, depth, and environmental factors;
3. conduct limited testing if no data is available;
4. apply the model to the data using maximum likelihood;
5. construct appropriate schedules or tables using whatever incidence of decompression sickness is acceptable;
6. release and then collect use statistics for final validation and tuning.

Questions of what risk is acceptable to the diver vary. Sport and research divers would probably opt for very small risk (.01% or less), while military and commercial divers might live with higher risk (1%), considering the nearness of medical attention in general. Many factors influence these two populations, but fitness and acclimatization levels would probably differ considerably across them. While such factors are difficult to fold into any table exercise or analysis, the simple fact that human subjects in dive experiments exhibit higher incidences during testing phases certainly helps to lower the actual incidence rate in the field, noted by Bennett and Lanphier.

Certainly there is considerable latitude in model assumptions, and many plausible variants on a theme. Many models are correlated with diving exposure data, using maximum likelihood to fit parameters or other valid statistical approaches, but not all. Most have been applied to profiles outside of tested ranges, when testing has been performed, in an obvious extrapolation mode. Sometimes the extrapolations are valid, other times not.

PART 2: REDUCED GRADIENT BUBBLE MODEL

Bubble Dynamics

Crucial to all bubble models are the concepts of critical radii and bubble growth. The critical radius, r_0, at fixed pressure, P_0, represents the cutoff for growth upon decompression to lesser pressure. Nuclei larger than r_0 will all grow upon decompression. Additionally, following an initial compression, $\Delta P = P - P_0$, a smaller class of micronuclei of critical radius, r, can be excited into growth with decompression. If r_0 is the critical radius at P_0, then, the smaller family, r, excited by decompression from P, obeys in the range, $100\ fsw\ \mu m \leq \kappa \leq 200\ fsw\ \mu m$,

$$\frac{1}{r} = \frac{1}{r_0} + \frac{\Delta P}{\kappa} \tag{47}$$

with ΔP measured in fsw, and r in *microns*. Table 3 lists critical radii, r, excited by sea level compressions, $P_0 = 33\ fsw$, for $r_0 = .8\ \mu m$, and $\kappa = 160\ fsw\ \mu m$. Entries are the equilibrium critical radii at pressure, P.

Table 3. Micronuclei Excitation Radii

pressure $P\ (fsw)$	excitation radius $r_0\ (\mu m)$	pressure $P\ (fsw)$	excitation radius $r_0\ (\mu m)$
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	.36

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

$$G = G_0 + \Delta G d \tag{48}$$

at depth $d = P - 33\ fsw$. A nonstop bounce exposure, followed by direct return to the surface, thus allows G_0 for that compartment. One set G_0 and ΔG are tabulated in Table 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),

$$G^{min} = \frac{2\,\gamma\,(\gamma_c - \gamma)}{\gamma_c\,r(t)} = \frac{11.01}{r(t)} \tag{49}$$

with,

$$r(t) = r + (r_0 - r)\,[1 - exp\,(-\lambda_r t)] \tag{50}$$

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,

$$G^{sat} = \frac{58.6}{r} - 49.9 = .372\,P + 11.01. \tag{51}$$

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,

$$n = N\,exp\,(-\beta r) \tag{52}$$

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

$$exp\,(-\beta r) = 1 - \beta r \tag{53}$$

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

$$\Delta n = n_0 - n = N\beta r_0\,\left[1 - \frac{r}{r_0}\right]. \tag{54}$$

For large compressions-decompressions, Δn is large, while for small compressions-decompressions, Δn is small. When Δn 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.

Phase Volume Limits

The rate at which gas inflates in tissue depends upon both the excess bubble number, Δn, 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,

$$\int_0^\infty \Delta n G dt = \alpha V \tag{55}$$

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,

$$\Delta n G\,(t_d + \lambda^{-1}) = \alpha V. \tag{56}$$

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

$$\delta = \frac{\gamma_c \, \alpha \, V}{\gamma \, \beta \, r_0 \, N} \tag{57}$$

so that,

$$\left[1 - \frac{r}{r_0}\right] \; G \left(t_d + \lambda^{-1}\right) = \delta \, \frac{\gamma}{\gamma_c} \tag{58}$$

The five parameters, γ, γ_c, δ, λ_r, r_0, 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. Bubble growth experiments suggest that λ_m^{-1} is in the neighborhood of an hour. Discussion of λ_m follows.

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,

$$\frac{r}{r_0} = .9 - .43 \, exp \, (-\zeta \tau) \tag{59}$$

with $\zeta = .0559 \; min^{-1}$. 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_m, take the values $\tau_r = 14 \; days$ and $\tau_m = 30 \; min$. For large τ, r is close to r_0, while for small τ, r is on the order of $.5 \; r_0$. At sea level, $r_0 = .8 \; microns$ as discussed.

The phase (limit) integral for multiexposures is written,

$$\sum_{j=1}^{J} \left[\Delta nG \, t_{d_j} + \int_0^{t_j} \Delta nG dt\right] \leq \alpha \, V \tag{60}$$

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 αV, obviously each term must be less the $\alpha \, V$. Assuming that $t_J \rightarrow \infty$, gives,

$$\sum_{j=1}^{J-1} \left[\Delta nG \left[t_{d_j} + \lambda^{-1} - \lambda^{-1} exp \left(-\lambda t_j\right)\right]\right] + \Delta nG \left(t_{d_J} + \lambda^{-1}\right) \leq \alpha \, V. \tag{61}$$

Defining G_j,

$$\Delta nG_j \left(t_{d_j} + \lambda^{-1}\right) = \Delta nG \left(t_{d_j} + \lambda^{-1}\right) - \Delta nG \, \lambda^{-1} exp \left(-\lambda t_{j-1}\right) \tag{62}$$

for $j = 2$ to J, and,

$$\Delta nG_1 = \Delta nG \tag{63}$$

for $j = 1$, it follows that

$$\sum_{j=1}^{J} \Delta n \; G_j \left(t_{d_j} + \lambda^{-1}\right) \leq \alpha \, V \tag{64}$$

with the important property,

$$G_j \leq G. \tag{65}$$

This implies we employ reduced gradients extracted from bounce gradients by writing,

$$G_j = \xi_j \; G \tag{66}$$

with ξ_j a *multidiving* fraction requisitely satisfying,

$$0 \leq \xi_j \leq 1 \tag{67}$$

so that, as needed,

$$\Delta nG_j \leq \Delta nG. \tag{68}$$

The fractions, ξ, applied to G always reduce them. As time and repetitive frequency increase, the body's ability to eliminate excess bubbles and nuclei decreases, so that we restrict the permissible bubble excess in time,

$$\Delta n(t_{j-1}^{cum}) = N\beta r_0 \left[1 - \frac{r(t_{j-1}^{cum})}{r_0}\right] = \Delta n \; exp\; (-\lambda_r t_{j-1}^{cum}) \tag{69}$$

$$t_{j-1}^{cum} = \sum_{i=1}^{j-1} t_i \tag{70}$$

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

$$\eta_j^{reg} = \frac{\Delta n(t_{j-1}^{cum})}{\Delta n} = exp\; (-\lambda_r t_{j-1}^{cum}) \tag{71}$$

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

$$\eta_j^{exc} = \frac{(\Delta n)_{max}}{(\Delta n)_j} = \frac{(rd)_{max}}{(rd)_j} \tag{72}$$

with *rd* the product of the appropriate excitation radius and depth. Because bubble elimination periods are shortened over repetitive dives, compared to intervals for bounce dives, the gradient reduction, η_j^{rep}, is proportional to the difference between maximum and actual surface bubble inflation rate, that is,

$$\eta_j^{rep} = 1 - \left[1 - \frac{G^{min}}{G}\right] exp\; (-\lambda_m t_{j-1}) \tag{73}$$

with t_{j-1} consecutive surface interval time, λ_m^{-1} on the order of an hour, and G^{min} the smallest G_0 in Table 4.

Finally, for multidiving, the gradient reduction factor, ξ, is defined bt the product of the three η,

$$\xi_j = \eta_j^{exc}\; \eta_j^{rep}\; \eta_j^{reg} = \frac{(\Delta n)_{max}}{(\Delta n)_j} \left[1 - \left(1 - \frac{G^{min}}{G}\right) exp\; (-\lambda_m t_{j-1})\right] exp\; (-\lambda_r t_{j-1}^{cum}) \tag{74}$$

with t_{j-1} consecutive interval time, and t_{j-1}^{cum} cumulative interval time, as noted. Since bubble numbers increase with depth, reduction in permissible gradient is commensurate. Multiday diving is mostly impacted by λ_r, while repetitive diving mostly by λ_m. Obviously, the critical tension, M, takes the form,

$$M = \xi(G_0 + \Delta Gd) + P. \tag{75}$$

Table 4 tabulates a (sample) set of RGBM critical gradients, G_0 and ΔG.

Table 4. Critical Phase Volume Gradients

halftime $\tau\ (min)$	threshold depth $\delta\ (fsw)$	surface gradient $G_0\ (fsw)$	gradient change ΔG
2	190	151.0	.518
5	135	95.0	.515
10	95	67.0	.511
20	65	49.0	.506
40	40	36.0	.468
80	30	27.0	.417
120	28	24.0	.379
240	16	23.0	.329
480	12	22.0	.312

Parameter Ranges

Over a range of depths, exposures, repetitive frequency, and gas mixtures, the parameter sets of the RGBM are roughly limited as follows,

$$0.45\ \mu m \leq r_0 \leq 1.45\ \mu m$$

$$15\ dyne/cm \leq \gamma \leq 65\ dync/cm$$

$$160\ dyne/cm \leq \gamma_c \leq 290\ dyne/cm$$

$$6500\ fsw\ min \leq \delta \leq 8300\ fsw\ min$$

$$7\ days \leq \tau_r \leq 36\ days$$

$$20\ min \leq \tau_m \leq 140\ min$$

with nonstop, altitude, decompression, saturation, nitrox, heliox, trimix, and repetitive exposures down to 550 fsw included in the range analysis. Values of the these parameters are also consistent with biophysical estimates, experimental data, and theoretical models across aquaeous and lipid substances. Given our present state of knowledge, nothing is incompatible in the ranges listed.

PART 3: ABYSS RGBM (Critical Parameter) SYNTHESIS

Profile And Parameter Matching

The following is specific to the ABYSS implementation of the RGBM across critical parameters and nonstop time limits of the ABYSS/ZHL algorithm. Extensive computer fitting of profiles and recalibration of parameters to maintain the RGBM within the ABYSS/ZHL limits is requisite here.

1. Critical Parameters (a, b)

   Haldane approaches use a simple 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, just slightly different in representation, but not content, First consider the transfer equation, assuming air (.79/21 nitrox).

   Tissue tensions (nitrogen partial pressures), p, for ambient nitrogen partial pressure, p_a, and initial tissue tension, p_i, evolve in time, t, in standard fashion in compartment, τ, according to,

   $$p - p_a = (p - p_a)\ exp\ (-\lambda t) \tag{76}$$

for,

$$\lambda = \frac{.693}{\tau} \tag{77}$$

with τ tissue halftime, and, for air,

$$p_a = .79\,P \tag{78}$$

and with ambient pressure, P, given as a function of depth, d, in units of fsw,

$$P = d + P_0 \tag{79}$$

Staging is controlled in the Buhlmann ZHL algorithm through sets of tissue parameters, a and b, listed below in Table 4 for 14 tissues, τ, through the minimum permissible (tolerable) ambient pressure, P_{min}, by,

$$P_{min} = (p - a)b \tag{80}$$

across all tissue compartments, τ, with the largest P_{min} limiting the allowable ambient pressure, P_{min}. Recall that,

$$1\ bar = 1.013\,atm\ \ ,\ \ 1\ atm = 33\ fsw$$

as conversion metric between *bar* and fsw in pressure calculations. Linear extrapolations across tissue compartments are often used for different sets of halftimes and critical parameters, a and b.

Table 5. Nitrogen ZHL Critical Parameters (a, b)

halftime $\tau\,(min)$	critical intercept $a\,(bar)$	critical slope b
5.0	1.198	.542
10.0	.939	.687
20.0	.731	.793
40.0	.496	.868
65.0	.425	.882
90.0	.395	.900
120.0	.372	.912
150.0	.350	.922
180.0	.334	.929
220.0	.318	.939
280.0	.295	.944
350.0	.272	.953
450.0	.255	.958
635.0	.236	.966

In terms of critical tensions, M, according to the USN, the relationship linking the two sets is simply,

$$M = \frac{P}{b} + a = \Delta M\ P + M_0 \tag{81}$$

so that,

$$\Delta M = \frac{1}{b} \tag{82}$$

$$M_0 = a \tag{83}$$

in units of *bar*, though the usual representation for M is fsw. The above set, a and b, hold generally for nitrox, and, to low order, for heliox (and trimix too). Tuned modifications for heliox and trimix are also tabulated below.

Corresponding nonstop time limits, t_n, are listed in Table 6, and the nonstop limits follow the Hempleman square root law, roughly,

$$dt_n^{1/2} = 475\ fsw\ min^{1/2} \tag{84}$$

in a least squares fit. The square root law also follows directly from the form of the bulk diffusion transfer equation, but not from any Haldane assumptions nor limiting forms of the tissue equation.

Table 6. Air ZHL Nonstop Time Limits

depth $d\ fsw$	time $t_n\ (min)$
30	290
40	130
50	75
60	54
70	38
80	26
90	22
100	20
110	17
120	15
130	11
140	9
150	8
160	7
170	6
180	5
190	4
200	3

2. Likelihood Profile And Model Analysis

   Over ranges of depths, tissue halftimes, and critical parameters of the ZHL algorithm, approximately 2,300 dive profiles were simulated using both the RGBM (Part 2) and Haldane ZHL algorithms. To correlate the two as closely as possible to the predictions of the RGBM across these profiles, maximum likelihood analysis is used, that is, extracting the temporal features of three bubble parameters mating the RGBM and ZHL algorithms extending critical parameters of the ZHL Haldane model to more complete bubble dynamical framework and physical basis. These factors, f, are described next, with their linkages to a and b, and are the well known *reduction factors* of the RGBM.

3. Multidiving Fractions

   According to the RGBM fits across the ZHL profiles (2,300), a correlation can be established through multidiving reduction factors, f, such that for any set of nonstop gradients, G,

$$G = M - P \tag{85}$$

   a reduced set, G_f, obtains from the nonstop set, G, for multidiving through the reduction factors, $f \leq 1$,

$$G_f = fG \tag{86}$$

   so that,

$$M_f = \frac{P}{b_f} + a_f = G_f + P = fG + P \tag{87}$$

but, since,

$$fG = f(M-P) = f\left[\frac{P}{b} + a - P\right] \tag{88}$$

we have,

$$a_f = fa \tag{89}$$

$$b_f = \frac{b}{f(1-b)+b} \tag{90}$$

The new (reduced) staging regimen is then simply,

$$P_{min} = (p - a_f)b_f \tag{91}$$

using *reduced* critical parameters, a_f and b_f. Certainly, as $f \to 1$, then $a_f \to a$, and $b_f \to b$, as requisite. Now all that remains is specification of f, particularly in terms of repetitive, reverse profile, and multiday diving, as limited by the bubble dynamical RGBM. The full factor, f, depends on tissue halftime, τ, generally through the relationship (for nitrox),

$$f = (1 - f_0)\,\frac{\tau}{180} + f_0 \quad (f = 1,\ \ \tau \geq 180\ min) \tag{92}$$

as the tissue scaling up through the 180 *min* nitrogen compartment, with multdiving weighting,

$$f_0 = .45\ f_{rp} + .30\ f_{dp} + .25\ f_{dy} \tag{93}$$

where f_{rp}, f_{dp}, and f_{dy} are reduction factors for repetitive, reverse profile (deeper than previous), and multiday (time spans of 30 *hrs* or more) diving. These forms for multidiving f are dependent on time between dives, t_{sr}, ambient pressure difference between reverse profile dives, ΔP, ambient pressure, P, and multiday diving frequency, n, over 24 *hr* time spans. Specifically, they are written,

$$f_{rp} = 1 - .45\ exp\ \left[-\frac{(t_{sr} - \eta_{rp})^2}{\eta_{rp}^2}\right] \tag{94}$$

$$10\ min \leq \eta_{rp} \leq 90\ min \tag{95}$$

$$f_{dp} = 1 - .45\ \left[1 - exp\ \left(-\frac{\Delta P}{P}\right)\right]\ exp\ \left[-\frac{(t_{sr} - \eta_{dp})^2}{\eta_{dp}^2}\right] \tag{96}$$

$$30\ min \leq \eta_{dp} \leq 120\ min \tag{97}$$

$$f_{dy} = .70 + .30\ exp\ \left(-\frac{n}{\eta_{dy}}\right) \tag{98}$$

$$7\ days \leq \eta_{dy} \leq 36\ days \tag{99}$$

with t_{sr} measured in *min*, and n the number of consecutive days of diving within 30 *hr* time spans. These factors are applied after 1 *min* of surface interval (otherwise, previous dive continuation). The difference, ΔP, is the time averaged difference between depths on the present and previous dives (computed on the fly). Reduction factors are consistent (folded in maximum likelihood in the RGBM) with the following:

(a) Doppler bubble scores peak in an hour or so after a dive;

(b) reverse profiles with depth increments beyond 50 fsw incur increasing DCI risk, somewhere between 5% and 8% in the depth increment range of 40 fsw - 120 fsw;

(c) Doppler bubble counts drop tenfold when ascent rates drop from 60 fsw/min to 30 fsw/min;

(d) multiday diving risks increase by factors of 2 -3 (though still small) over risk associated with a single dive.

4. Nitrox

The standard set, a, b, and τ, given in Table 5 hold across nitrox exposures, and the tissue equation remains the same. The obvious change for a nitrox mixture with nitrogen fraction, f_{N_2}, occurs in the nitrogen ambient pressure, p_{aN_2}, at depth, d, in analogy with the air case,

$$p_{aN_2} = f_{N_2}\, P = f_{N_2}\,(d + P_0) \tag{100}$$

with P ambient pressure (fsw). All else is unchanged. The case, f_{N_2} = .79, obviously represents an air mixture.

5. Heliox

The standard set, a, b, and τ is modified for helium mixtures, with basic change in the set of halftimes, τ, used for the set, a and b, To lowest orderset, a and b for helium are the same as those for nitrogen, though we will list the modifications in Table 7 below. Halftimes for helium are approximately 2.65 times faster than those for nitrogen, by Graham's law (molecular diffusion rates scale inversely with square root of atomic masses). That is,

$$\tau_{He} = \frac{\tau_{N_2}}{2.65} \tag{101}$$

because helium is approximately 7 times lighter than nitrogen, and diffusion rates scale with square root of the ratio of atomic masses. The tissue equation is the same as the nitrox tissue equation, but with helium constants, λ, defined by the helium tissue halftimes. Denoting the helium fraction, f_{He}, the helium ambient pressure, p_{aHe}, is given by,

$$p_{aHe} = f_{He}\, P = f_{He}\,(d + P_0) \tag{102}$$

as with nitrox. Multidiving fractions are the same, but the tissue scaling is different across the helium set,

$$f = (1 - f_0)\,\frac{\tau}{67.8} + f_0 \quad (f = 1,\ \ \tau \geq 67.8\ min) \tag{103}$$

and all else is the same.

Table 7. Helium ZHL Critical Parameters (a, b)

halftime $\tau\,(min)$	critical intercept $a\,(bar)$	critical slope b
1.8	1.653	.461
3.8	1.295	.604
7.6	1.008	.729
15.0	.759	.816
24.5	.672	.837
33.9	.636	.864
45.2	.598	.876
56.6	.562	.885
67.8	.541	.892
83.0	.526	.901
105.5	.519	.906
132.0	.516	.914
169.7	.510	.919
239.6	.495	.927

6. Trimix

For trimix, both helium and nitrogen must be tracked with tissue equations, and appropriate average of helium and nitrogen critical parameters used for staging. Thus, denoting nitrogen and helium fractions, f_{N_2}, and f_{He}, ambient nitrogen and helium pressures, p_{aN_2} and p_{aHe}, take the form,

$$p_{aN_2} = f_{N_2}\, P = f_{N_2}\, (d + P_0) \tag{104}$$

$$p_{aHe} = f_{He}\, P = f_{He}\, (d + P_0) \tag{105}$$

Tissue halftimes are mapped exactly as listed in Tables 5 and 6, and used appropriately for nitrogen and helium tissue equations. Additionally,

$$f_{O_2} + f_{N_2} + f_{He} = 1 \tag{106}$$

and certainly in Tables 5 and 6, one has the mapping,

$$\tau_{He} = \frac{\tau_{N_2}}{2.65} \tag{107}$$

Then, total tension, Π, is the sum of nitrogen and helium components,

$$\Pi = (p_{aN_2} + p_{aHe}) + (p_{iN_2} - p_{aN_2})\; exp\; (-\lambda_{N_2} t) + (p_{iHe} - p_{aHe})\; exp\; (-\lambda_{He} t) \tag{108}$$

with λ_{N_2} and λ_{He} decay constant for the nitrogen and helium halftimes in Tables 5 and 6. Critical parameters for trimix, α_f and β_f, are just weighted averages of critical parameters, a_{N_2}, b_{N_2}, a_{He} b_{He}, from Tables 5 and 6, that is, generalizing to the reduced set, a_f and b_f,

$$\alpha_f = \frac{f_{N_2} a_{fN_2} + f_{He} a_{fHe}}{f_{N_2} + f_{He}} \tag{109}$$

$$\beta_f = \frac{f_{N_2} b_{fN_2} + f_{He} b_{fHe}}{f_{N_2} + f_{He}} \tag{110}$$

The staging regimen for trimix is,

$$P_{min} = (\Pi - \alpha_f)\beta_f \tag{111}$$

as before. The corresponding critical tension, M_f, generalizes to,

$$M_f = \frac{P}{\beta_f} + \alpha_f \tag{112}$$

Synthesis Summary

Overall, the ABYSS/RGBM algorithm is conservative with safety imparted to the Haldane ABYSS model through multidiving f factors. Estimated DCI incidence rate from likelihood analysis is .001% at the 95% confidence level for the overall ABYSS/RGBM. Table and meter implementations with consistent coding should reflect this estimated risk. Similar estimates and comments apply to the ZHL mixed gas synthesis.

PART 4: PHASE (ABYSS/RGBM) AND HALDANE CONTRASTS

ABYSS/RGBM For The Layman

The following discourse charts in layman terms the differences between phase models, such as the full RGBM and ABYSS/RGBM, and dissolved gas models, such as the ZHL of Buhlmann. Hopefully this Part aids in ABYSS marketing strategy and diver education.

Empirical Practices

Utilitarian procedures, entirely consistent with phase mechanics and bubble dissolution time scales, have been developed under duress, and with trauma, by Australian pearl divers and Hawaiian diving fishermen, for both deep and repetitive diving with possible in-water recompression for hits. While the science behind such procedures was not initially clear, the operational effectiveness was always noteworthy and could not be discounted easily. Later, the rationale, essentially recounted in the foregoing, became clearer.

Pearling fleets, operating in the deep tidal waters off northern Australia, employed Okinawan divers who regularly journeyed to depths of 300 fsw for as long as one hour, two times a day, six days per week, and ten months out of the year. Driven by economics, and not science, these divers developed optimized decompression schedules empirically. As reported by Le Messurier and Hills, deeper decompression stops, but shorter decompression times than required by Haldane theory, were characteristics of their profiles. Such protocols are entirely consistent with minimizing bubble growth and the excitation of nuclei through the application of increased pressure, as are shallow safety stops and slow ascent rates. With higher incidence of surface decompression sickness, as might be expected, the Australians devised a simple, but very effective, in-water recompression procedure. The stricken diver is taken back down to 30 fsw on oxygen for roughly 30 *minutes* in mild cases, or 60 *minutes* in severe cases. Increased pressures help to constrict bubbles, while breathing pure oxygen maximizes inert gas washout (elimination). Recompression time scales are consistent with bubble dissolution experiments.

Similar schedules and procedures have evolved in Hawaii, among diving fishermen, according to Farm and Hayashi. Harvesting the oceans for food and profit, Hawaiian divers make beween 8 and 12 dives a day to depths beyond 350 fsw. Profit incentives induce divers to take risks relative to bottom time in conventional tables. Three repetitive dives are usually necessary to net a school of fish. Consistent with bubble and nucleation theory, these divers make their deep dive first, followed by shallower excursions. A typical series might start with a dive to 220 fsw, followed by 2 dives to 120 fsw, and culminate in 3 or 4 more excursions to less than 60 fsw. Often, little or no surface intervals are clocked between dives. Such types of profiles literally clobber conventional tables, but, with proper reckoning of bubble and phase mechanics, acquire some credibility. With ascending profiles and suitable application of pressure, gas seed excitation and any bubble growth are constrained within the body's capacity to eliminate free and dissolved gas phases. In a broad sense, the final shallow dives have been tagged as prolonged safety stops, and the effectiveness of these procedures has been substantiated *in vivo* (dogs) by Kunkle and Beckman. In-water recompression procedures, similar to the Australian regimens, complement Hawaiian diving practices for all the same reasons.

While the above practices developed by trial-and-error, albeit with seeming principle, venous gas emboli measurements, performed off Catalina by Pilmanis on divers making shallow safety stops, fall into the more *scientific* category perhaps. Contrasting bubble counts following bounce exposures near 100 fsw, with and without zonal stops in the 10-20 fsw range, marked reductions (factors of 4 to 5) in venous gas emboli were noted when stops were made. If, as some suggest, venous gas emboli in bounce diving correlate with bubbles in sites such as tendons and ligaments, then safety stops probably minimize bubble growth in such extravascular locations. In these tests, the sample population was small, so additional validation and testing is warranted.

Only a handful of hard and fast conclusions about DCI can be drawn from present knowledge. So elementary as to be innocuous, they are stated:

1. bubble inception or phase separation is the primary event triggering simple decompression sickness;
2. prevention of decompression sickness amounts to prevention (as a limit) of bubble inception or phase separation;
3. gradual pressure reductions prevent bubble formation.

As known by many, after the above attempts at concensus usually diverge. Modelers and table designers must then supply, or assume, gas exchange models, trigger points, and safe diving protocols which prevent or, at least, minimize phase inception and bubble growth.

Present notions of nucleation and cavitation 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. Stochastic Monte Carlo bubble tracking methods are powerful, but only in supercomputer environments, due to the large number of events required for meaningful statistics over simulation time spans. Exchange models for entrained bubbles and coalescence dynamics are

similarly complicated. In all cases, more knowledge about gas micronuclei and size distributions, tissue sites, thermodynamics properties, stabilization, and excitation mechanisms is necessary before computing power can be leveraged to decompression modeling.

But even with a paucity of knowledge, many feel that empirical 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. Bubble and phase models support the efficacy of recently suggested safe diving practices, by simple virtue of dual phase mechanics:

1. reduced nonstop time limits;
2. safety stops (or shallow swimming ascents) in the 10-20 fsw zone, 1-2 *min* for dives in the 40-90 fsw range, 2-3 *min* for dives in the 90-240 fsw range;
3. ascent rates not exceeding 30 fsw/min;
4. restricted repetitive exposures, particularly beyond 100 fsw, based on reduction in permissible bubble excess over time;
5. restricted spike (shallow-to-deep) exposures based on excitation of additional micronuclei;
6. restricted multiday activity based on regeneration of micronuclei over longer time scales;
7. smooth coalescence of bounce and saturation limit points, consistent with bubble experiments;
8. consistent model treatment of altitude diving;

Bubble models also tend to be consistent with the utilitarian measures observed for diving practice. Conservatism may be downplayed in some meter implementations, yet medical authorities are becoming increasingly concerned about long term effects of breathing pressurized gases. On firmer principles, bubble models tend to corroborate safety measures in multidiving, and thus one might reasonably expect to witness their further development. Said another way, bubble models have the right physical signatures for diving application.

Phase Versus Haldane Profiles

Both SUUNTO and Abysmal Diving have released products incorporating a modern phase algorithm, the above 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 multidiving, 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 diving (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. Expect RGBM coded software to surface in other SUUNTO computers, like the COBRA and SPYDER.

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. And, of course, since the RGBM extends the VPM, much of the following applies to the VPM directly.

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 concensus, 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 natural way, gory details of which are found in the References.

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 and publications 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.

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 (and VPM) issues and tacks taken in the VYPER 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.

   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 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.

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. 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.

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.

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 (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.

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. 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.

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.

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 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 sugested 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.

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. 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 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 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.

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 multidiving 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 8 for 3 days of repetitive air diving (120 fsw/10 *min* twice a day with 45 *min* surface interval). Computer choices are illustrative, not indictive.

Table 8. Nonstop Limits For VYPER/RGBM And Haldane Air Multidiving

Computer/Algorithm	Dive 1 (min)	Dive 2 (min)	Dive 3 (min)	Dive 4 (min)	Dive 5 (min)	Dive 6 (min)
VYPER/RGBM	10	6	9	5	9	5
SPYDER/Haldane	10	9	10	9	10	9
DATA PLUS/Haldane	12	6	12	6	12	6
DELPHI/Haldane	10	10	10	10	10	10
DC11/Haldane	6	6	6	6	6	6
DC12/Haldane	9	7	9	7	9	7
ALADIN/Haldane	8	8	8	8	8	8
ALADIN PRO/Haldane	10	7	10	7	10	7
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 9 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 9. 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 3 and 4, the more dramatic effects of the RGBM show up for deep staging, as seen in Table 10. 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 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 10. 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	19
16	30	18	12	28	13
17	20	16	10	28	11
18	10	28	16	48	18
		93	77	147	98

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. A rundown of the software configuration of the RGBM used in full blown simulations follows. The package is under constant refinement and updating.

1. Module: Three major routines (RGBMNX, RGBMHX, RGBMTMX) for nitrox, heliox, and trimix.
2. Source Code: 1640 Lines
3. Language/Compiler: FORTRAN 77/90, BASIC.
4. CRAY YMP Running Time: 1 *sec* for deep trimix profile with 5 gas switches on way up.
5. Input: altitude, bottom mixture, ascent/descent rate, switch levels and gas mixtures, predive breathing gas, safety knobs, previous dive history.
6. Output: controlling tissue compartments, stop depth and times, supersaturation gradient, permissble supersaturation, effective bubble and gas parameters, critical phase volume, dive profile.
7. Cost: $3800

ADDITIONAL READING

References span a wide spectra of technical diving material and details, broaching historical to modern developments. Entries are alphabetically and chronologically listed.

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Wienke, a former dive shop owner in Santa Fe, presently works with DAN on applications of high performance computing and communications to diving, and is a Regional Data Coordinator for Project Dive Safety. Scubapro, Suunto, Abysmal Diving, and Atomic engage him 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). The Suunto VYPER dive computer incorporates the RGBM into staging regimens, particularly for recreational diving (including nitrox). 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.

Wienke received a BS in physics and mathematics from Northern Michigan University, an MS in nuclear physics from Marquette University, and a PhD in particle physics from Nothwestern University. He is a member of the Undersea And Hyperbaric Medical Society (UHMS), American Physical Society (APS), Society Of Industrial And Applied Mathematics (SIAM), and the American Academy Of Underwater Sciences (AAUS).

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