THE TROUBLE WITH BUBBLES

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Recent debate amongst some sections of the diving community has concerned the relative benefits of using new ascent and decompression strategies based on calculations of the presence and behaviour of microbubbles. This debate has extended to concern over the relative safety of standard entry level agency decompression/no stop tables. The concern stems from the fact that recent research and opinion regarding the formation of microbubbles on ascent from depth and their role in predisposing towards decompression sickness (DCS), would urge greater caution and, at the very least, slower ascent rates than those recommended in standard diver training. As such, it would seem prudent to review the information that is available in order to have an objective view of the subject of decompression stress and microbubble formation such that informed opinions can be developed prior to recommending any changes in diving practice and/or training principles. Whilst this is certainly relevant to the way our own club views things, obviously the ramifications are broad and need to be viewed in the context of the way the diving community as a whole views and reacts to the current trends.

Whilst it is beyond the scope of this article to deal with the theories of decompression and microbubble dynamics in great detail, it is nevertheless necessary to touch on some of the basic principles in order to understand how recent research is changing the way people are thinking at the leading edge of diving. These are the people who are involved in research and in extreme technical diving who need to be able to evolve new and safer techniques in order to push the decompression envelope. It is after all, the empirical testing of these new principles that eventually make our recreational sport diving safer and more enjoyable through the development of better decompression models. Where necessary, the interested reader will be referred to more in depth and informed articles.

Part I
CAUSES: THE PHYSICS AND PHYSIOLOGY OF DECOMPRESSION.

Factors Predisposing to DCS.

Before going on to discuss the background with respect to decompression models, it serves to first give a brief outline of DCS and its predisposing factors. Decompression illness (DCI) is basically caused by bubble formation in the blood and/or tissues due to a rapid reduction in ambient pressure usually as a result of too rapid an ascent or incomplete decompression to the extent that overt symptoms result, ranging from mild to severe and even life threatening. Additionally, pulmonary aveolar rupture can occur with resultant gas bubbles entering the arterial circulation, a condition known as arterial gas embolism (AGE). The two conditions (DCI and AGE) are collectively referred to as DCS. It is sometimes difficult to distinguish between severe DCI and cerebral gas embolism. The two conditions may co-exist in a diver who has ascended rapidly from a prolonged dive and developed pulmonary barotrauma. In any case, for all practical

purposes the on-site treatment is the same anyway-oxygen therapy followed by evacuation to a recompression facility. DCS is a multi-system condition. Limb pain is the most common complaint, with pain in the elbow, shoulder, hip and knee joints being the most prevalent sites. The skin may be involved, displaying a mottled appearance. Bubbles in the lymphatic system may result in regional lymphoedema. Whilst lymhatic bends are very rare, I personally know of someone who has had one ! (known affectionately as “Bendy Bob”). DCS limited to the musculoskeletal, skin and lymphatic system are referred to as Type I, or mild DCS. More severe cases may involve the brain, spinal cord or cardiopulmonary system. Neurological manifestations may include sensory deficits, hemiplegia, paraplegia, parasthesias and peripheral neuropathies. Possible cardiopulmonary manifestations include massive pulmonary gas emboli (chokes) or myocardial infarction. Systemic responses can present as severe hypovolemia and shock. DCS with these symptoms is referred to as Type II, or severe DCS. It is worth bearing in mind that it is now thought that some cases of mild DCS involving limb pain may actually be a result of less severe neurological or spinal hits!

So, what are the predisposing factors ? The presenting symptoms of DCS are influenced by the depth of the dive; the bottom time; the inert gas breathed; the ascent rate; the adequacy of decompression and the delay to presentaion. Apart from the obvious, like inadequate decompression, violation of no-decompression limits, flying after diving (12-24 hours) and inadequate surface intervals, intracardiac septal defect, or patent foramen ovale (PFO) is probably the single most important factor predisposing to cases of DCS that occur with ‘no obvious cause’ ie. within prescribed decompression limits. Since the lungs normally act as a filter for bubbles, any condition that allows bubbles to pass from the venous to the arterial side (right-left shunting), bypassing the lungs such as PFO or pulmonary barotrauma, is likely to result in DCS. Apart from this, it is now recognised that dehydration is probably the most important of the predisposing factors. Adequate water intake (ie. about a litre per day) is necessary to counteract the dehydration effect of compressed air and diuresis caused by immersion. On top of this, tea, coffee, alcohol (especially alcohol) and certain medications can contribute. Other differences in individual physiology that may predispose include lung or heart disease; scar tissue from a previous injury (decreased diffusion or areas of increased or decreased blood flow); previous DCS (neurological); gender (women have slightly higher incidence); obesity (nitrogen has high lipid solubility and fat tissue has a poor blood supply, therefore poor gas exchange); fatigue; age and poor fitness (fitness increases perfusion and gas exchange). Environmental factors may include cold water (hypothermia and vasoconstriction decreases gas exchange); exertion (the vacuum effect in which tendon use causes gas pockets and exercise at depth increases nitrogen uptake) and heated diving suits (may cause dehydration). Divers who have become chilled on decompression dives and take hot showers risk bubble formation, as do divers who engage in heavy exercise after diving. Interestingly, mild exercise during the ascent and whilst performing safety stops is helpful in off-gassing [see Campbell].

Finally, we must consider that differences in individual physiology (apart from things like PFO) probably play a major role in predisposition to DCS. Standard decompression models cannot possibly take this into account, apart from adding in arbitrary ‘conservatism’ factors, as some computers now do. One aspect of physiology that researchers now recognise as a complicating factor(s) in DCS are changes in blood

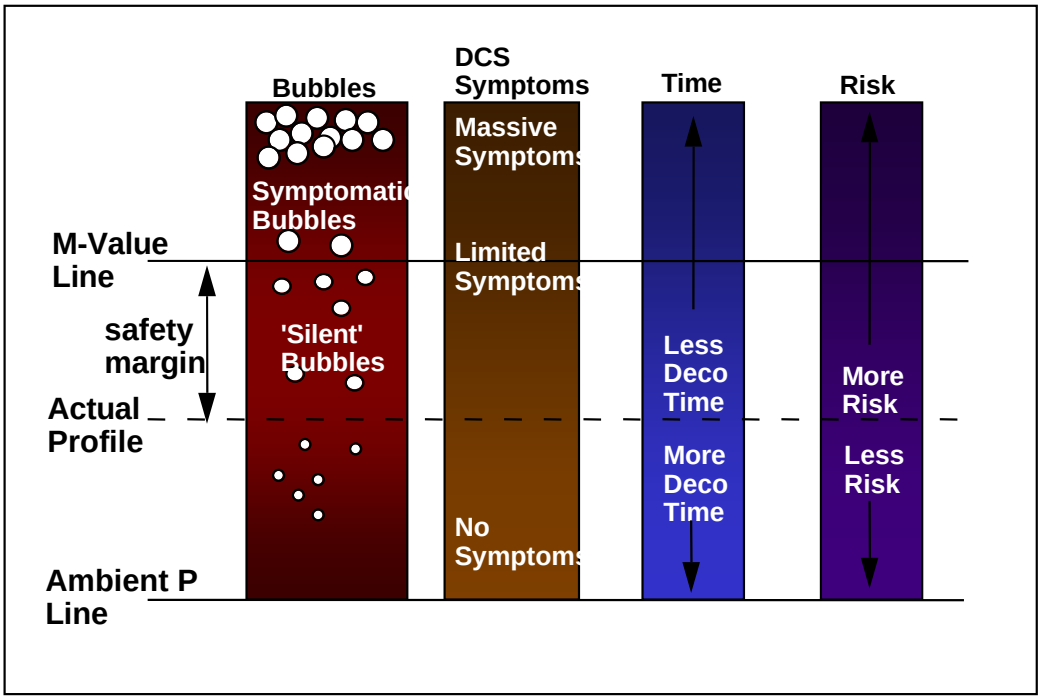
chemistry and clotting factors. Thus the presence of microbubbles probably induces an inflammatory/immune response, the severity of which will vary enormously from individual to individual. In fact, the possible benefits of anti-inflammatory agents such as steroids has been discussed in the diving medicine literature. Microbubbles are thought to cause platelet activation and aggregation [Softeland et al; Bakken et al; Thorsen et al (1, 2)] and also complement activation and thus, may contribute to microinfarction of tissue caused by the formation of microemboli.

The Basics of Decompression Theory

The decompression algorithms that are used in the standard decompression tables and wrist computers that most of us use in our normal sports diving activities are based on Haldanian, or rather neo-Haldanian, principles. That is to say that they use the concept of inert gas loading into a number of different hypothetical tissue compartments which are referred to as 'fast' or 'slow' tissues. Fast tissues are generally those which are highly perfused (ie. have a large blood supply), such as skeletal and cardiac muscle, liver etc. and which take up and eliminate gas relatively quickly. Slow tissues, such as adipose tissue, nerve tissue and bone take up and release gas more slowly (due to their relative under perfusion) but also take longer to saturate. In the 1980's/early 1990's Professor Albert Bulhmann introduced various refinements to the theory which resulted in him publishing a decompression set based on the tissue half-times of up to sixteen different tissue compartments. The B and C sets were introduced because the original A set, empirically derived using actual decompression trials, was considered not to be conservative enough. A such the ZHL16C set is commonly used in many diving software applications. The general trend has been to become slightly more conservative, which reflects a more rigorous validation process including the use of techniques such as Doppler ultrasound monitoring of divers to detect 'silent bubbles' ie bubbles that are detectable in the circulation but are not necessarily associated with overt symptoms of decompression illness.

An important point, is that these algorithms are based on the concept of 'M values' introduced by Robert Workman in the 1960's. These M values basically describe the tolerated overpressure limits (or critical tensions) for the inert gas (ie. nitrogen or helium) in the breathing mixture, in the different tissue compartments. Workmann realised that the faster compartments had greater overpressure ratios than the slower compartments and that for all compartments the tolerated ratios became less with increasing depth. The 'M-value' then, refers to the maximum tolerated partial pressure of nitrogen or helium for each hypothetical tissue compartment. The importance of the concept of 'M-values' will (hopefully) become more apparent later on when we discuss 'deep stops'. Bulhmann also used this concept when he developed his decompression models in which the M-values describe a linear relationship between the ambient pressure and the tolerated inert gas pressure in the hypothetical tissue compartments. The major difference was that Bulhmann's values are based on absolute pressure, rather than gauge pressure and thus can be used for altitude as well as sea level diving [the reader is referred to Baker and to Figures 1 and 2].

Figure 1



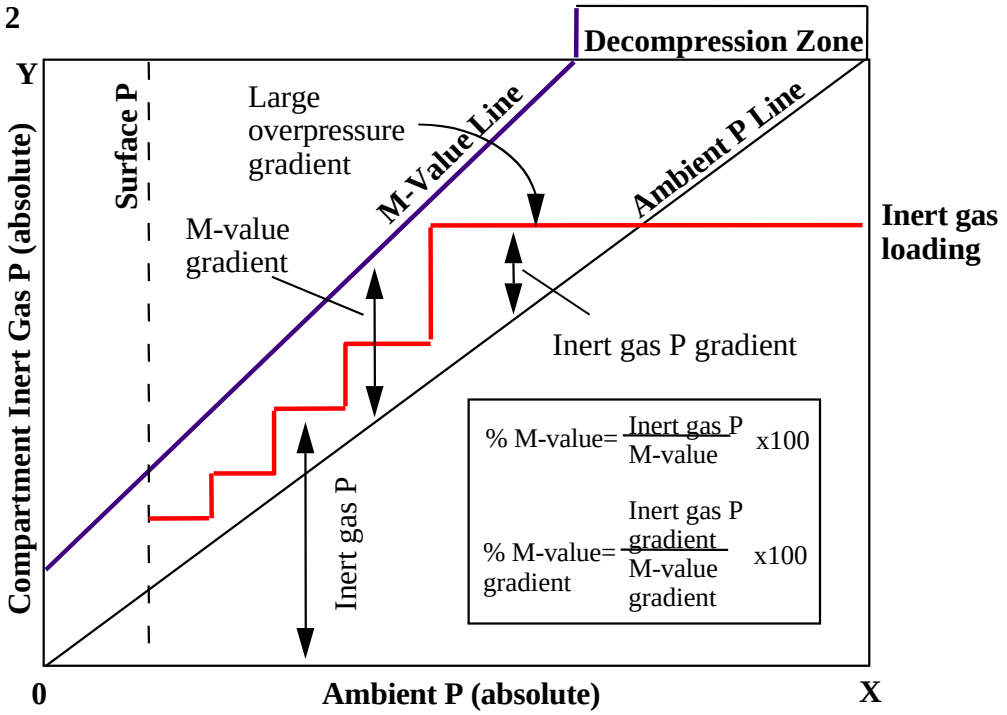
The M-value concept: A representative threshold beyond which a high frequency of symptoms of decompression sickness (DCS) can be expected in the majority ofdivers.

[Adapted from Baker (1)]

The slope of the M-value line will be slightly different for each tissue compartment due to the different half-times and represents the overpressure gradient for that tissue. Since the slope of the ambient pressure line is always 1.0, the M-value line will be greater than 1.0 with faster half-time compartments having a greater slope, therefore faster compartments can tolerate greater overpressure than slower compartments. The ambient pressure line on the graph shown in Figure 2 has a slope of 1.0 and simply reflects when the compartment inert gas loading will be equal to the ambient pressure. Thus, when the inert gas loading goes above this reference line, an overpressure gradient will be created. This is important for two reasons: (1) The M-value line represents the safe limit for the tolerated overpressure gradient and (2) during an ascent it is precisely this overpressure gradient that drives 'off-gassing' (decompression) or bubble formation if the overpressure limit (M-value) is exceeded or the ascent too rapid. This brings us neatly to the concept of a safe 'decompression zone' which, naturally, lies between the two lines (ie. between the ambient pressure line and the M-value, or overpressure, line). This simply means that the diver has to cross over the ambient pressure line [ie. from the right hand side of the graph (deeper) towards the left (shallower)] in order to generate a gradient for off-gassing without entering the danger

zone for bubble formation ie. without crossing the M-value line. It therefore stands to reason that the most efficient area of the graph to be in, in terms of off-gassing whilst remaining within safety is in the middle, between the two lines.

Figure 2



The M-value concept: A representative threshold beyond which a high frequency of symptoms of decompression sickness (DCS) can be expected in the majority of divers. Note the large overpressure gradient generated on ascent to the first stop which is out of proportion to the rest of the profile (Adapted from Baker).

[Adapted from Baker (1)]

Whilst this is a good model which has undoubtedly increased the relative safety of dives performed on this basis, to my mind at least, there are two obvious problems with this that actually create the very issues that this article/debate is really about. The first is that the tissue compartments are hypothetical and that the model assumes that during decompression all the gas is in the soluble phase. Since Doppler monitoring of divers has, in fact, revealed the presence of free phase bubbles in divers both during and following diving, this is clearly not the case. As such, recent opinion suggests that these models/assumptions are inadequate and that the free (gas) phase should be taken into consideration in decompression models.

The Gas Phase.

This second point brings us on to the issue of 'silent bubbles' or microbubbles. For most of the dives we do in the sport diver range, the fast tissues govern our decompression profile. The model assumes that during decompression all the gas is initially in the soluble phase as it diffuses from the tissues into the blood (down its concentration and pressure gradients) and only passes into the gas phase once it has crossed the alveolar membrane in the lungs. This has the effect of putting our conventional decompression stops at a shallower depth than would be necessary to avoid any microbubble formation. However, these profiles may be provocative in that at least some microbubble formation may be inevitable (see below).

As experienced divers, we have learned to live with the knowledge that when we make an ascent we are probably 'fizzing' a little. This has certainly been born out by research, even since the sixties, and Doppler monitoring has revealed the presence of gas phase bubbles in divers ascending even from relatively shallow dives. We have also all experienced symptoms such as headache and fatigue in the immediate post-dive phase which we have put down to overexertion on exiting the water etc. In more recent years, we have been told that these symptoms are those of subclinical DCI and we have lived with these, perhaps naively, thinking that because they are not associated with overt symptoms of DCI, they are not causing any damage and are nothing to worry about. However, enter the iceberg principle. Nine tenths of the damage may be invisible and cummulative. It is true that there is a great deal of plasticity within the human physiological system and that some of the damage maybe reversible. However, it is also true that some recent and some not so recent research suggests that some insidious cummulative injury may occur in divers without overt symptoms of DCI. For instance, examination of diver's retinas has revealed that the eye, ie. the cornea, tear membranes, retinal microvessels and humours within the eye, may be primary sites of bubble formation on ascent. This has the result that in a study of divers evidence of retinal microvessel damage was revealed [Polkinghorne; Mekjavic et al]. In another study using magnetic resonance imaging (MRI) on a cohort of German divers who regularly undertook deep, repetitive dives, evidence of permanent cerebral lesions was detected [Knauth; Wilmshurst]. Interestingly, in the same group of divers, evidence of damage to the cervical vertebral discs was noted in some. This latter pathology could conceivably be associated with repetitive lifting of heavy objects (ie. diving cylinders) rather than be a direct result of microbubles. Nevertheless, such soft tissue or bone injuries in divers are known to predispose to bubble formation and DCI, in some cases.

In another recent study by German researchers who used cranial MRI on 88 divers they discovered brain lesions in 12 divers. Of those 12, PFO was detected in 5 who had a total of 37 brain lesions. 4 had up to 16 lesions. Both depth and length of dives were associated with the risk of brain lesions. All the divers with lesions had 600 to 800 logged dives [Reuters Health]. PFO occurs in about one in four people and whilst it is harmless under most conditions, it is believed to contribute significantly to unexplained incidences of DCS. So, if our club has 80 members, up to 20 could have a PFO. These workers recommend that echocardiography be used to check for PFO as part of the certification process for divers. Whilst these examples are alarming, one has to accept that these studies have limitations. Large prospective trials are needed to properly

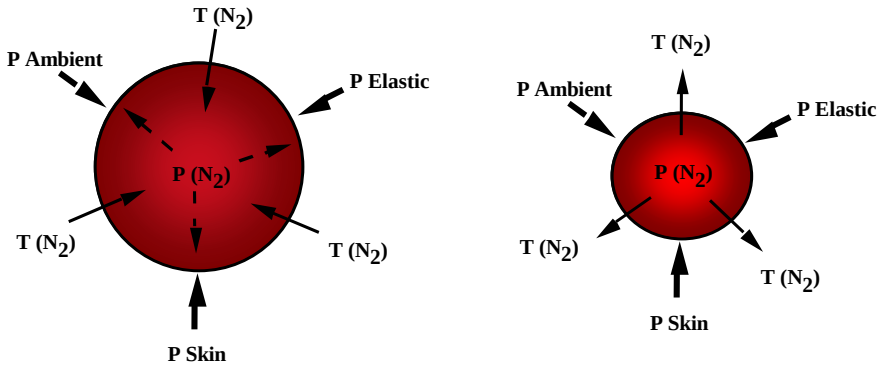
ascertain whether permanent damage is rigorously and statistically associated with deep, repetitive diving and microbubble formation.

Bubble Formation

The development and evolution of gas phases and bubble formation involves a number of overlapping steps. These include nucleation and stabilisation (free phase inception); supersaturation (dissolved gas build-up); excitation and growth (free-dissolved phase interaction); coalescence (bubble aggregation) and deformation and occlusion (tissue damage and ischaemia). Where bubbles form or lodge, how they migrate, how they evolve and dissolve or the complete spectrum of physico-chemical insults that results in DCS are not known in great detail. Bubbles may form *de novo* at supersaturated sites on decompression or may grow from pre-formed seed nuclei excited by compression-decompression. Bubbles may then migrate to critical sites elsewhere, or stick at the birth site. They may grow locally to the point where they cause deformation of the tissue and nerve endings beyond the pain threshold. They may dissolve locally by gaseous diffusion to surrounding tissue or blood or they may be broken down or eliminated by the pulmonary filter of the lungs.

Bubbles may form intravascularly (in blood) or extravascularly. Intravascular bubbles may block small blood vessels causing ischaemia or block the pulmonary filters, may cause blood sludging, adverse biochemical changes or mechanical nerve deformation. They may leave the circulation to lodge in tissues as extravascular bubbles. Intravascular bubbles have been detected in both the venous and arterial circulation, but in far greater numbers on the venous side. The lungs act as an efficient filter for most of the venous bubbles above a certain size. Venous bubbles are suspected to form in fat tissue due to the 5 times higher solubility of nitrogen in lipids and pass into the veins draining these tissues. Veins, being thinner than arteries, are more susceptible to extravascular gas penetration. Large volumes of extravascular gas can induce vascular haemorrhage, depositing both fat and bubbles into the circulation and breach the blood-brain barrier causing focal cerebral oedema [Hills; Hills and James; Nohara and Yusa].

Figure 3



Bubble grows if tissue tension (T) is greater than internal P of bubble.

Bubble shrinks if tissue tension (T) is smaller than internal P of bubble.

[Adapted from Maiken]

Also, lipid tissues, possessing fewer nerve endings may be a repository for undetected bubble formation which can form critical sites.

Spontaneous bubble formation in pure liquids is extremely unlikely and would require huge depressurisations (hundreds of atmospheres). However, the reason bubbles do form at relatively low depressurisations (a few atmospheres) is due to nucleation, or bubble seeding on particles and surfaces in contact with the dissolved phase. Once formed, bubbles are then stabilised by the presence of surfactants, either already present in blood and tissue, or released by tissue and vascular damage. Bubbles grow from micron size and although inherently unstable, they resist collapse due to elastic skins formed by surfactants or by a reduction in surface tension at tissue interfaces (see figure 3). Once formed, very large pressures are needed to crush them (tens of atmospheres). This is one reason why sawtooth profiles are bad, because the bubbles formed on each ascent phase are not crushed again but accumulate, such that the final ascent and decompression phase is started with a pre-existing bubble load leading to a provocative situation [see Maiken; Weinke (1)]. Since it is now recognised that the lifetime of a bubble can be measured in days, this has implications for repetitive and multi-day diving, in particular. Bends often occur towards the end of multi-day dive trips, when bubble loads have been allowed to built up to a critical point. Obviously, one way to reduce bubble loads for repetitive dives is to increase the surface interval between dives. It has been suggested that whilst it takes about 48 hours to achieve complete desaturation, it takes about 4 to 8 hours to get rid of most of the bubbles. Thus increasing surface time between dives to between 4 to 8 hours could decrease the risk of an event [Knauth].

Obviously these are complex processes which escape complete elucidation. What is clear is that both separated and dissolved gas phases must be taken into account in order to evolve safer and better decompression models. This is discussed below.

Part II
THE PREVENTION: DEEP STOPS; BUBBLE MODELS; OPENING THE OXYGEN WINDOW

Summary of Bubble Dynamics

A bubble will remain stable in size (at equilibrium) if the internal pressure of the bubble balances the outside pressure. In this case the sum of the hydrostatic mechanical pressures pushing inwards will balance the sum of the partial pressures of the free gases within the bubble pushing outwards. The mechanical pressures are due to tissue elasticity, the bubble's skin tension and the ambient pressure (proportional to depth). The pressure of gas dissolved in the tissues is referred to as tissue tension. The bubble will grow or shrink depending on whether the tension of gases in the surrounding tissue is greater or smaller than the internal pressure of the bubble (see figure 3). In either case, a pressure gradient (G) across the skin of the bubble drives the flow of gas. When G is positive, tissue tension is greater than bubble pressure and the bubble will grow due to inflow of gas. G is negative when tissue tension is less than bubble pressure leading to outflow of gas and bubble shrinkage. The objective of bubble decompression models is to keep G negative (or zero) by setting ascent stage depths and choosing gas mixtures to encourage bubble out gassing to the tissues. Thus, deep stops keep the ambient pressure

large, which keeps the bubble pressure large and G negative to force out gas. By utilising the appropriate ascent gas breathing mix (ie. to open the 'oxygen window') tissue tension is further reduced helping to keep G negative.

Deep Stops

The information in this section is, basically, a summary of Eric C. Baker's article on deep stops. The best treatment for the prevention of DCS is to complete a sufficient decompression profile. Technical divers have observed that many of the symptoms associated with subclinical DCS can be eliminated by including deep stops in their profiles. This was first described anecdotally by deep marine ichthyologist Richard Pyle, hence deep stops are sometimes referred to as 'Pyle stops'. A closer examination of decompression models reveals that this practice has a sound basis and serves to reduce or eliminate excessive overpressure gradients. With this knowledge, the model can be modified to provide precise control of the gradients and stops can be calculated within the decompression zone to the depth of the 'deepest possible decompression stop'. This is discussed below in the next section and is an important point, since it is necessary to perform this accurately to achieve optimal decompression. Putting in deep stops arbitrarily doesn't always work and may even make matters worse by increasing loading, particularly in the 'slow' tissue compartments. Analysis of the empirical observations of divers that have led to arbitrary methods for introducing deep stops have revealed potential problems. For instance, stops may be made too deep and there is inadequate extension of the shallow stops to compensate for the increased gas loading caused by the deep stops.

As mentioned above, conventional decompression models put the stops as shallow as possible, within the limits of the M-values. This is to maximise off-gassing of the fast tissues, whilst minimising on-gassing in the slow tissues. We are used to the idea of getting off the bottom quickly. On most dives we do, the inert gas loadings in the fast tissue compartments will be at or near saturation, whilst the slow tissues will only be partially loaded. In this case, the fast tissues will dictate the initial ascent profile, or will 'lead', since their inert gas loading will be closer to their respective M-values on ascent. The first stop is determined by when the inert gas loading in the leading compartment is close to its M-value.

As we've discussed above, we know that bubbles are probably present after most dives, so we don't have to exceed an M-value to produce bubbles in the absence of DCS. Because the M-values for the faster compartments permit larger overpressure gradients than slower compartments and lead the ascent profile, a large and rapid overpressure gradient is created on ascent to the first stop. This is out of proportion with the smaller over pressure gradients permitted during the rest of the profile when the slower compartments take over. Although an M-value may not have been exceeded, symptoms of decompression stress such as fatigue, malaise, drowsiness etc. could well be the result. It now becomes easy to see why putting in deep stops would prevent this large overpressure gradient during the initial ascent and probably most of the symptoms.

So, if we've decided deep stops are a good thing, where do we put them ? They can't be too deep, because they have to be within the decompression zone. As with conventional models, we still need to achieve efficient off-gassing, whilst limiting on-gassing in slow compartments. Therefore, within the constraints of the dissolved gas

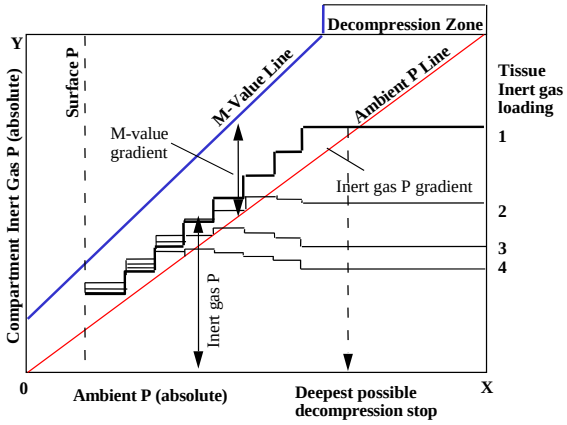
model, we need to calculate where the ‘deepest possible decompression stop’ would be. Generally, this is defined as the next standard stop depth above the point where the gas loading for the leading compartment crosses the ambient pressure line (see figure 4). This simply represents the beginning of the decompression zone ie. the point where at least one tissue enters the decompression zone. This is useful to know, even if the actual first stop is above the deepest possible stop. Profiles where the first stop is a few standard stop depths above this may be adequate to control excessive overpressure gradients, but the diver should at least slow down the ascent to 10 msw/min or less when this point is reached. When it comes to the issue of actually introducing the deep stops, it becomes slightly more complicated, depending on the method used. Basically, even if done using a desktop decompression program (such as the ones published by Richard Pyle) one has to be careful to increase the conservatism factor for the slow tissues at the shallower stops in order to avoid them exceeding their M-values due to increased loading at the deep stops. The reader is strongly urged to refer to other more in depth articles [see Baker]. It is recommended that the way to assess this is to calculate maximum percent M-values and percent M-value gradients across all compartments at each stop. These ‘gradient factors’ thus control the overpressure gradients across the entire profile. The addition of deep stops in a profile will generally increase the time required at the shallow stops as well as the overall decompression time. However, if a truly sufficient decompression is the result, then the general principle of economic decompression is not really compromised [Baker].

Bubble Decompression Models

We can see from the information above, that whilst deep stops seem to be a good thing (at least for long, deep dives and certainly for any extended range diving), they are not necessarily simple to implement without a decompression computer program that can handle it. Dr Bruce Weinke is a computer modeller who works at the Los Alamos National Laboratory and has been working with the Abyssmal Diving Team to implement his latest decompression model into the Abyss software. What I will do here is to simply summarise how this model differs from the conventional models and refer you to his articles in the online Abyss Technical Diving Library [see Weinke (2, 3)]. Of note is that both Suunto and Abyssmal Diving have released products which incorporate these modern phase algorithms, called Reduced Gradient Bubble Models (RGBMs). RGBMs incorporate modelling of both dissolved and free gas phase build-up and elimination. The Suunto Vyper is an RGBM-based diving computer (decometer) for recreational divers (including nitrox) and is to my knowledge a field leader in this respect. Also the Abyss/RGBM is a licensed Abyssmal Diving software product. References are given within Weinke (2) which give a full description of these products.

Figure 4

An extension of the M-value concept showing the deepest possible decompression stop, ie. the next standard stop depth above where the inert gas loading for at least one tissue (leading) compartment crosses the ambient pressue line and enters the decompression zone. Note that when the leading compartment crosses the ambient pressure line and begins decompression, other slower compartments may not have yet entered the decompression zone and their inert gas loading might actually increase slightly. However, also note that the large overpressure gradient on ascent to the first stop is eliminated (Adapted from Baker). This diagram (and that in figure 2) is a purely hypothetical and stylised profile and not derived from a 'real' decompression algorithm.



A review of some of the salient features that the model includes serves to illustrate how bubble calculations have been incorporated:

1) Standard Bulhmann no-stop limits
2) Restricted repetitive exposures beyond 100ft (30m) based on a reduction of permissible bubble diffusion gradients within 2 hour time spans
3) Restricted yo-yo and spike dives based on excitation of new bubble seeds
4) Restricted deeper-than-previous dives based on excitation of very small bubble seeds over 2 hour time spans
5) Restricted multi-day diving based on adaptation and re-growth of new bubble seeds
6) Smooth coalescence of bounce and saturation limit points using 32 tissue compartments
7) Consistent treatment of altitude diving, with proper zero-point exptrapolation of limiting tensions and permissible bubble gradients
8) Algorithm linked to actual diving data, Doppler bubble and micronuclei experiments.

Figure 5

DecoPlan (WKPP) (example a) 21%O2/79% N2

Depth	Time	Start	End	PpO2	Gas Reqd	M-Value%
50	15	0	15	1.26	2052	
21	1	18	19	0.65	59	62
18	1	19	20	0.59	53	65
15	1	20	21	0.53	48	69
12	1	21	22	0.46	42	74
9	3	22	25	0.4	108	81
6	17	25	42	0.34	547	85
0		43				92

(example b)

Depth	Time	Start	End	PpO2	Gas Reqd	M-Value%
50	15	0	15	1.26	1938	
30	1	17	18	0.84	152	
18	1	19	20	0.59	53	66
15	2	20	22	0.53	95	70
12	1	22	23	0.46	42	73
9	4	23	27	0.4	144	80
6	17	27	44	0.34	547	83
0		45				93

Global Underwater Exploration (GUE) (example c)

Depth	Time	Start	End	PpO2	Gas Reqd	M-Value%
50	15	0	15	1.26	1938	
21	1	18	19	0.84	152	
18	1	19	20	0.59	53	66
15	1	20	22	0.53	95	70
12	2	22	24	0.46	42	73
9	4	24	27	0.4	144	80
6	20	27	44	0.34	547	83
0		48				93

IANTD (Bulhmann) (example d)

Depth	Time	15	12	9	6	3	Deco	In-water	Group
51m	18	-	-	4	5	13	22	40	F
51m	21	-	3	4	7	18	32	53	G

In the Vyper and Abyss implementations of the model, perfusion (blood flow) is assumed to dominate the perfusion-diffusion gas transport process, which makes the model simpler and permits on-line calculations in real time. Also, the RGBM incorporates a feature which takes account of something called the “oxygen window”. This basically means that blood and tissues are naturally undersaturated with respect to ambient pressure at equilibrium. This is also referred to as “biologically inherent unsaturation”. But more of that later, as it is relevant to the use of oxygen as a decompression gas. The

RGBM assumes that there is always a size distribution of new bubble seeds and a certain number of these will be excited into bubble growth by compression-decompression. The methods employed for ascent staging thus controls the expansion rate of these bubbles in order that their collective volume never exceeds a phase volume limit point. Breathing mixtures containing nitrogen, helium and oxygen are calculated as having the same phase volume limit points, but contain bubble distributions of different sizes. The RGBM also assumes that bubble seeds have different gas permeabilities. Bubble skins are assumed permeable down to a crushing pressure of 10 atm and that beyond this they permit gas diffusion at a slower rate. The size of seeds excited into growth is inversely proportional to the supersaturation gradient. The RGBM assumes that bubble skins are stabilised by surfactants over unknown time scales, but that the seeds are persistent in the body. The RGBM assumes the size distribution is exponentially decreasing in size ie. that there are exponentially more smaller seeds than larger seeds. The RGBM incorporates a spectrum of tissue compartments (up to 32) with half-times ranging from 1 to 720 min, depending on the gas mixture. Phase separation and bubble growth in slower compartments is a central feature in calculations. The no-stop limits employed by the RGBM are based on recent studies involving Doppler measurements, conservatively reducing them along the lines of phase volume constraints. The Vyper implementation additionally penalises ascent violations by requiring additional safety stop time which is based on an ongoing risk analysis of the situation! The RGBM reduces the phase volume limit in multi-day diving by taking into account free phase elimination and build-up during surface intervals. This depends on time, altitude and depth of previous exposures. As such, repetitive, multi-day and deeper-than-previous exposures are affected by these critical phase volume reductions. The RGBM generates replacement bubble seed distributions on a time scale of days, adding new bubbles to existing bubbles in the calculations. As such, the critical phase volume limit points are reduced by the added effects of new bubbles.

So, that summarises the major differences between phase and dissolved gas models (phewww!!). What is really interesting about the examples that are given in Bruce Weinke's article, is the dramatic effects the RGBM has for dives involving deep staging. He shows comparative deep schedules for a trimix dive to 250ft incoporating a switch to air at 100ft and pure oxygen at 20ft. Deeper stops are noticeably requisite in the Abyss RGBM, but total decompression times are less than when using the Abyss/ZHL software. In short, the phase models recover dissolved gas models for short and nominal exposures, but require deeper stops and shorter overall decompression times for longer and exceptional exposures. Some examples of the treatment of a dive to 50m for 15 minutes on air by two deep stop models (Deco Planner and GUE) are shown in figure 5 in comparison to a conventional IANTD Bulhmann table. In each case the total in water time is about the same or slightly less than for the conventional table, but the result is more complete and efficient decompression. In contrast, putting in the same deep stops on a conventional table (ie. using an air computer) would give a much longer decompression penalty (see below and the hypothetical example in figure 6).

Increased Conservatism in Conventional Decometers.

This is particularly interesting given that, as mentioned above, in order to introduce deep stops we need to take into account increased gas loading into slower compartments. If done properly, we would expect this to increase the obligatory

decompression time at the shallower stops to take account of this. Some of us know from empirical experience that if we put in deep stops arbitrarily on a deep air dive (say a dive to somewhere between 35 to 50m involving necessary decompression) by reducing our ascent rate and stopping for, say one minute, at one or two conventional stops below the first required stop, that when using a conventional air computer, our overall decompression time would be extended by more time required at the shallower stops (see the hypothetical example shown in figure 6). If a proper RGBM model may reduce overall decompression time, this raises the question of how the newer conventional computers, which incorporate additional conservatism factors, calculate this additional conservatism. This will give us an idea as to whether using them in this mode (ie. by putting in deep stops) is viable and adds a safety factor to normal air diving over and above the conservatism already built in to the computer's algorithm. It would seem reasonable, providing you (and your colleagues already on the boat!) could live with the slightly longer in-water time.

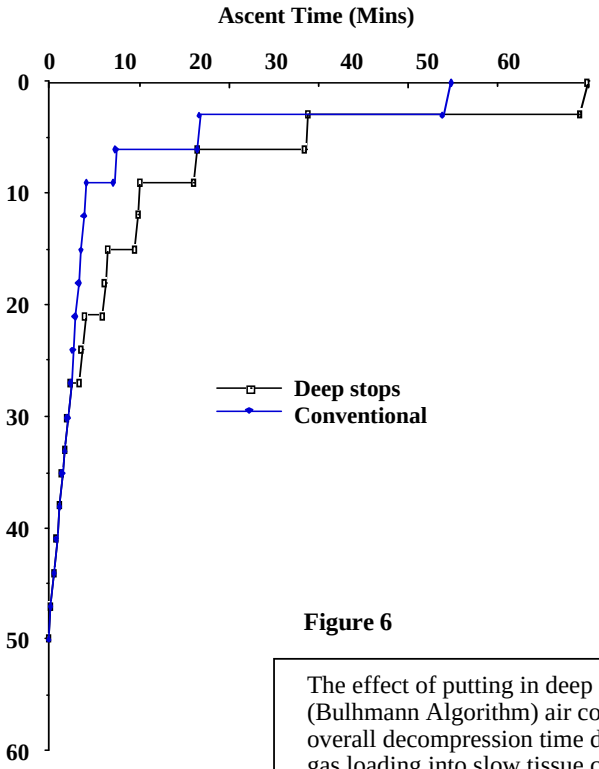


Figure 6

The effect of putting in deep stops on a conventional (Bulhmann Algorithm) air computer would be to extend overall decompression time due to the apparent increased gas loading into slow tissue compartments at the deeper stops

My own wrist unit is an Aladin Pro Nitrox. This model uses the ZH-L8 ADT algorithm which has 8 tissue compartments with nominal half-times of 5 to 640 minutes. It purportedly differs from other models by taking consideration of the fact that blood perfusion to the respective tissues is not constant, but depends on temperature. Particularly skin and muscle perfusion are sensitive to ambient temperature and workload, and as we have discussed above, the saturation tolerance of tissues is greatly

affected by perfusion. The ZHL-L8 ADT model takes this into account by providing the 'skin' and 'muscle' compartments with variable half-times and saturation tolerances. The main feature, therefore, seems to be an adjustment of these parameters based on water temperature, with the assumption that decreased water temperature and increased dive time means decreased skin temperature. It is also stated that it takes account of the divers behaviour on the dive, but I can't see how it can possibly take account of workload. The other feature is that it supposedly takes into account is the generation of free phase gas (microbubbles), which presumably *is* based on the divers behaviour. The Aladin Pro Nitrox calculates the formation of microbubbles depending on various assumed influences in arterial and venous blood. This appears to be mainly based on ascent rate and will penalise accordingly with increased decompression and 'no-fly' time and decreased bottom time in order to limit the growth of existing bubbles and increase elimination. The calculation of microbubbles will also result in a reduction of the ascent rate to 7 msw/min, again to limit formation of arterial microbubbles and limit growth of venous microbubbles both during and after the dive.

However, it is not clear precisely how the behaviour of microbubbles in this model is calculated. Since the penalty for putting in extra stops deeper than calculated by the unit is increased overall decompression time, it probably treats this behaviour as an extension of the bottom time and thus tissue loading and increased, rather than decreased, risk of microbubble formation. So, providing that you don't put in stops that are too deep, it's probably OK to do it given that it generates an added safety margin. As a rule of thumb, it is probably OK to use one or two conventional stop depths below the first necessary stop, rather than using 'half the bottom depth' and so on, as the conventional stop depths will smooth out the ascent profile and eliminate the large overpressure gradient on ascending to the first stop. For example, if your first stop is at 6m, stop for one minute at 9m (and possibly 12m). If you don't stop, at least reduce your ascent rate to 5-7 msw/min when entering the decompression zone (ie. between 12 and 9m). In combination with decompression on oxygen-rich mixtures (discussed below), this is probably a good practice.

The Oxygen Window

As mentioned above, the 'oxygen window' refers to the inherent biological unsaturation of tissue at ambient pressure. This concept is relevant to oxygen decompression and so is worth considering briefly here. Most of us know that it is generally considered a safer practice to decompress with an increased oxygen partial pressure (ie. using nitrox mixtures), since it aids the elimination of nitrogen from the tissues. Some of us use this as an added safety margin on deep air dives (ie. using a conventional wrist unit in 'air mode'), rather than to reduce overall decompression time. Increasing the ppO2 and thus decreasing the ppN2 in the breathing mixture, increases the diffusion gradient for the nitrogen coming out of the blood going through the lungs, therefore increasing elimination. Increasing the blood ppO2, will also displace nitrogen from the tissues.

The metabolism of tissues is such that the consumption of oxygen and production of carbon dioxide drops the tissue oxygen tension below the alveolar ppO2. This generates the inward and outward gradients for oxygen and carbon dioxide, respectively. Carbon dioxide tension rises only slightly, because it is 25 times more soluble than

oxygen. Blood and tissue ppO2 are considerably lower than alveolar ppO2. The degree of tissue unsaturation with respect to gases (ie. nitrogen, helium, oxygen) increases linearly with pressure for a constant composition breathing mixture and decreases linearly with the mole fraction of gas in the inspired mix. This window can be exploited in bringing divers to the surface by staging the ascent such that the inert gas (ie. nitrogen) just takes up this inherent unsaturation, thus keeping the total tissue tension equal to the ambient pressure. This is called a 'zero supersaturation' ascent. This inherent unsaturation can also be exploited by increasing the ppO2 in the inspired mix, since the total inert gas pressure in a tissue compartment is the sum of the partial pressures of the constituent gasses, even though different inert gasses have different half-times for the same compartment. Thus increasing the ppO2 will decrease the proportion of the total tissue tension taken up by nitrogen and reduce its gradient for on-gassing, whilst increasing its gradient for off-gassing. Put another way, the optimal strategy for inert gas elimination should effectively eliminate both free and dissolved phases. With this aim, it is best to stay at high ppO2 to encourage elimination of dissolved gas. By simultaneously keeping the external (ie. ambient) pressure as high as possible (to minimise bubble growth and maximise free phase elimination), the oxygen window is open to its fullest. This effectively means getting onto a higher ppO2 mix as early as possible during the ascent and keeping the ppO2 as high as possible within the limits of CNS oxygen toxicity (ie. around 1.4-1.6 bar working pressure) [Maiken; Weinke (3)]. In short, open the oxygen window as wide as possible, as early as possible. Without getting into the arguments of using an 80/20 nitrox mix versus 100% O2 for the shallowest stop, it's worth pointing out that the ppO2 of 80/20 at 6m is only 1.28 bar (less than optimal), whereas 100% is 1.6 bar (more realistic).

Before passing on from the benefits of oxygen decompression, there is one last point I would like to consider which came as something of a shock to me when reading some research abstracts on pulmonary gas exchange and gas phase formation during decompression on different mixes. As we know, or as discussed above, the lungs act as a very efficient filter for venous microbubbles above a certain diameter. Whereas the Doppler detection of venous return bubbles was associated as a risk factor for bends, during the oxygen-breathing portions of decompression, bubbles could not be detected precordially [Powell et al]. What is even more striking, is the effect on arterial blood gases when comparing air and 100% O2 decompression. Air decompression (AD) was associated with a significant drop in arterial ppO2. Furthermore, the pulmonary diffusing capacity for carbon monoxide (DLCO) was decreased in all divers with AD at 20, 40, 60 and 80 minutes after diving. There was a significant correlation between the maximum bubble grade and decreased DLCO, and decreased ppO2 with AD. Oxygen decompression abolished these changes. The conclusion is that the presence of venous bubbles causes pulmonary micrembolisation and a decrease in the pulmonary diffusing capacity [Dujic et al]. In layman's terms, venous microbubbles screw up gas exchange (ie. inert gas elimination) in the lungs, an effect which can be completely eliminated by decompressing on oxygen. So let's face it, if oxygen recompression therapy is the treatment of choice for DCS, why not treat ourselves *in situ* to prevent subclinical DCS, or decompression stress ? I think this is borne out by the above.

Summary and Recommendations

In this article we have considered the basics of decompression theory, why decompression illness occurs, the formation of microbubbles and so on. We have then considered ways of improving our diving practices on the basis of this knowledge, including the introduction of deep stops into dive profiles to keep gas in the dissolved phase, oxygen decompression to aid inert gas elimination and new decompression models which take into account bubble dynamics and which are the immediate future and will eventually find there way into our diving at every level. In recent years we have already witnessed many changes to our diving procedures which reflect the general trend towards increased conservatism. These include shorter no-stop time limits; slower ascent rates; discretionary safety stops; ascending repetitive profiles (ie. deepest-first); multi-level techniques; faster and slower repetitive tissues; variable compartmental gas loading; smaller critical tensions (M-values); longer surface intervals etc. In fact most wrist units (decometers) now available on the market now incorporate all or some of these modifications. What I find alarming, is that some entry level training agencies (not to mention any names), given all this recent knowledge and changing trends, haven't altered their tables since their inception. The question is whether as instructors we should alter our methods to take some of these things into account. I think that certainly as experienced divers we should adopt safer decompression strategies (see below). The recommendations for entry level training are obviously more limited, but I think we have a responsibility to adopt the safest possible methods for our trainees given the knowledge available.

Suggested **Recommendations:**

Entry level/training.

(i) Slow accent rates to minimum 7 msw/min and less (3 msw/min) within the 9-6 m zone and 1 msw/m between 6m and surface.

(ii) Do not conduct multiple ascents, even on shallow dives. Certainly not involving return to surface.

(iii) Conduct discretionary safety stops as a matter of course and staged, not just at 6m (ie. 9m, 6m, 3m).

Experienced/Deep Air.

(i) Slow accent rates to minimum 7 msw/min and less (3 msw/min) within the 9-6 m zone and 1 msw/m between 6m and surface.

(ii) Stage the ascent, incorporating deep stops (<u>according to an approved method</u>), accepting that using a conventional air computer this will increase overall decompression time (plan gas accordingly).

(iii) Use elevated ppO2 mixtures for decompression. Use the maximum permissible ppO2 for an operating ppO2 of 1.4-1.6 bar (ie. 100% at 6m,

depending on qualification); switch as early as possible on the ascent ie. “open the oxygen window as wide and as early as possible”.

(iv) Use a computer or tables incorporating an RGBM-particularly for mixed gases and extended range.

Footnote:

The information contained in this article solely represents the opinions of the author and is by no means an exhaustive review of all the available literature on decompression stress and microbubble decompression models. Much of the information is derived from from other more detailed articles and these are referenced throughout. The reader is urged to refer particularly to the articles of Eric Baker, Eric Maiken and Bruce Weinke for a more detailed handling of the subject material. As far as possible and within the limitations of the authors knowledge, the information contained herein is deemed correct. However, it is a personal interpretation and as such, may not wholly represent the opinions of everyone involved in this complex area of research.

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