Recreational technical diving part 2: decompression from deep technical dives

David J Doolette and Simon J Mitchell

Abstract

(Doolette DJ, Mitchell SJ. Recreational technical diving part 2: decompression from deep technical dives. *Diving and Hyperbaric Medine*. 2013 June;43(2):96-104.)

Technical divers perform deep, mixed-gas 'bounce' dives, which are inherently inefficient because even a short duration at the target depth results in lengthy decompression. Technical divers use decompression schedules generated from modified versions of decompression algorithms originally developed for other types of diving. Many modifications ostensibly produce shorter and/or safer decompression, but have generally been driven by anecdote. Scientific evidence relevant to many of these modifications exists, but is often difficult to locate. This review assembles and examines scientific evidence relevant to technical diving decompression practice. There is a widespread belief that bubble algorithms, which redistribute decompression in favour of deeper decompression stops, are more efficient than traditional, shallow-stop, gas-content algorithms, but recent laboratory data support the opposite view. It seems unlikely that switches from helium- to nitrogen-based breathing gases during ascent will accelerate decompression from typical technical bounce dives. However, there is evidence for a higher prevalence of neurological decompression sickness (DCS) after dives conducted breathing only helium-oxygen than those with nitrogen-oxygen. There is also weak evidence suggesting less neurological DCS occurs if helium-oxygen breathing gas is switched to air during decompression than if no switch is made. On the other hand, helium-to-nitrogen breathing gas switches are implicated in the development of inner-ear DCS arising during decompression. Inner-ear DCS is difficult to predict, but strategies to minimize the risk include adequate initial decompression, delaying helium-to-nitrogen switches until relatively shallow, and the use of the maximum safe fraction of inspired oxygen during decompression.

Key words

Technical diving, deep diving, trimix, decompression, decompression tables, models, decompression sickness, inner ear, review article

Introduction

Some scuba divers use gases other than air and specialized equipment configurations to explore deeper depths for longer durations than are possible with the single-cylinder, open-circuit air diving configuration typically used by recreational divers. These divers refer to themselves as 'technical divers'. In the preceding article, we provided an introduction to the methods used by technical divers.[1] Technical divers typically perform 'bounce dives'; that is, dives lasting minutes to hours in which a short period of descent and at the target depth ('bottom time') is immediately followed by decompression back to the surface, and in which any increase in bottom time results in increased absorption of inert gas and a longer decompression obligation. This distinguishes their activities from many occupational dives to similar depths in which the divers effectively live under pressure in a dry chamber environment, and make periodic excursions into the sea to perform underwater work. This is referred to as 'saturation diving' because, after a certain period, the inert gas in all tissues is in equilibrium with inspired inert gas partial pressures (saturated). The divers can subsequently spend an indefinite period under pressure and their decompression obligation will remain the same. In comparison, bounce diving is very inefficient in respect to the amount of time spent at depth versus the time spent decompressing. Not surprisingly, in technical diving there is invariable tension between the desire to spend more time at the target depth and a desire to minimize decompressions, which take place in the water, possibly exposed to cold, strong currents and other hazards. The optimisation of decompression from these deep bounce dives is, therefore, one of if not the most debated and controversial issues in technical diving. This paper focuses on decompression methods and selected controversies.

Decompression from technical dives

As discussed in the first paper in this series, helium-based breathing gas mixtures are used for deep diving to avoid the narcotic effects of high nitrogen partial pressures and to reduce gas density. Technical divers typically use trimix breathing gas ($He\text{-}N_2\text{-}O_2$) instead of heliox ($He\text{-}O_2$) for deep diving. The reasons for this are historical, financial and logistical. Early technical divers had limited infrastructure to handle and mix helium into high-pressure scuba cylinders.[2] Also, helium is expensive, a particular concern for open-circuit diving, but less so in rebreathers; and there is a perception that larger fractions of helium in the breathing gas result in an increased decompression obligation. These issues motivate some divers to use the minimum helium fraction consistent with managing nitrogen narcosis and gas density. The use of trimix breathing gas presents challenges. Whereas, recreational divers were able to adopt readily available military air decompression tables, which were validated against databases of dives with known outcomes, no such trimix tables were available to technical divers.

GAS-CONTENT DECOMPRESSION ALGORITHMS

The earliest technical divers used custom trimix decompression tables prepared for them by RW Hamilton using a proprietary software (DCAP) implementation of the Tonawanda II decompression algorithm and the 11F6 M-values matrix (see below).[2] Almost immediately thereafter, technical divers began implementing the Buhlmann ZH-L16 decompression algorithm, descriptions of which were readily available in the open scientific literature.[3–5] Both Tonawanda II and ZH-L16 are 'gas-content' decompression algorithms. Gas-content algorithms track the uptake and elimination of inert gas in notional tissue compartments with different gas kinetic properties, and schedule decompression stops according to ascent rules that limit the degree to which the sum of dissolved gas pressures in the compartment exceeds the ambient pressure: a state referred to as supersaturation. Supersaturation is a requirement for bubble formation from dissolved gas, and the principle of limiting supersaturation (and thereby bubble formation) to schedule decompression was introduced by Haldane and colleagues.[6] A widely used format for the ascent rules which define acceptable compartmental supersaturation is:

$$P_{tis.k} < a.k \cdot P_{amb} + b.k \qquad (1)$$

Where: P_{amb} is the ambient pressure; P_{tis} is the gas tension in k theoretical compartments that represent body tissues with different gas exchange rates, and a and b are constants for each of the k compartments.[7]

If P_{amb} is expressed in depth-of-water gauge pressure at specified decompression stops, the left-hand side of the equation is the maximum permissible tissue tension, or 'M-value', at that depth.[7] To apply the ascent rules, the depth/time/breathing-gas history of a dive is used to calculate P_{tis}, usually by assuming that the tissue-to-arterial inert gas tension difference declines mono-exponentially according to a half-time notionally determined by the blood flow to the tissue, and the relative solubility of the gas in the blood and tissue compartments. To accommodate trimix diving, each compartment may have a different half-time for helium and nitrogen and these gases are tracked independently. P_{tis} is compared to a matrix of M-values for each compartment at predefined decompression stop depths to determine if or when ascent to those depths is permissible.

BUBBLE DECOMPRESSION ALGORITHMS

There are two general classes of bubble decompression algorithms, although they have overlapping aspects. One class calculates bubble size using complex equations of bubble growth and resolution due to gas diffusion between bubbles and the surrounding tissue.[8,9] The second class of algorithms is much simpler, focusing on predictions of the number of bubbles that form during decompression.[10] These latter bubble-counting algorithms will be outlined here because they are widely available to technical divers.[11,12]

The smallest radius spherical bubble (R_{min}) that can grow for any particular supersaturation (P_{ss}) is given by re-arranging the LaPlace equation:

$$R_{min} = 2st/P_{ss} \qquad (2)$$

Since the growth of small bubbles requires a large supersaturation, it seems likely that bubbles in the body result from accumulation of gas into or around pre-existing gas nuclei. One theoretical form of gas nucleus is a spherical 'proto-bubble' coated with surface active agents that counteract surface tension and render the gas nucleus relatively stable. The varying permeability model (VPM) assumes a population of spherical gas nuclei and a theoretical distribution of their radii that, along with equation (2), is used to calculate the number of gas nuclei activated into growing bubbles by the maximum supersaturation encountered during decompression.[10] In the simplest form of VPM algorithm, decompression can be controlled by a predicted maximum allowed number of bubbles and, therefore, a maximum allowed supersaturation. Alternatively, the number of bubbles is converted to a simple index representing the number of bubbles and their growth by multiplying the number of bubbles by the time integral of supersaturation. The allowed supersaturation is that which, if sustained throughout the ascent, results in the target value of this bubble index. The parameters of the VPM algorithm were originally adjusted to give decompression times similar to existing military decompression tables.[10] Some of these parameters may be user-adjustable, resulting in longer or shorter decompression times, in computer implementations available to technical divers.

UNTESTED SCHEDULES

The decompression procedures promulgated by well-resourced organizations (e.g., the US Navy) are developed and validated in conjunction with human dive trials in which the conditions that influence the risk of decompression sickness (DCS) are well-documented and dive outcomes (typically DCS or not) are known. In the development phase, decompression algorithm parameters are found by prospective trial-and-error testing, or by formal statistical fit of decompression models to existing databases of dives. The final decompression algorithm is validated by comparison to other man-dives. The development and validation man-dives are conducted under conditions similar to the intended use of the procedures, both in terms of depth/time/breathing-gas histories and other DCS risk factors not accounted for in the algorithms (such as diver work rate and thermal status). The resulting decompression algorithms are embodiments of the development data and are not intended for and, indeed, do not extrapolate well to all types of diving.

Many decompression schedules used by technical divers are untested because they are generated using decompression algorithms that have not been developed and validated with the types of dives conducted by technical divers. Both the Tonawanda II – 11F6 and the ZH-L16 decompression algorithms were developed in conjunction with laboratory dive trials but had limited testing specific to trimix diving at the depth ranges typical for technical diving. Development of ZH-L16 included many man-dives although most were substantially shallower or deeper than the 60–90 metres' sea water (msw) typical of technical diving, and there were few trimix dives.[5] In a carefully monitored technical diving project using the ZH-L16 algorithm for decompression guidance, two cases of DCS requiring treatment occurred in the course of 122 trimix dives (95% confidence limits 0.2%, 5.8% incidence).[13] Anecdotally, other technical diving projects had similar incidences of DCS, and the ZH-L16 algorithm may be modified by the end-user to be more conservative.

A popular, but untested end-user modification is the use of 'gradient factors'. In this usage, gradient refers, unconventionally (because it is not a gradient), to the difference between ambient pressure and an algorithm M-value.[12,14] Supersaturation is limited to less than permitted by the original M-value by allowing only a fraction of the difference between ambient pressure and the original M-value. These fractions have come to be known as gradient factors.[14] Thus, if a diver elects to limit supersaturation to 80% of the usual difference between ambient pressure and the M-value, this is referred as 'gradient factor 80' (GF 80). Typical proprietary implementations of the gradient factor method require the diver to select two gradient factors: the first modifies permitted supersaturation at the deepest decompression stop and the second controls supersaturation at the point of surfacing. The algorithm then interpolates a series of modified M-values in between these two user-specified points. Not surprisingly, lowering the first gradient factor forces deeper stops to limit supersaturation in the fast tissues early in the ascent, and lowering the second will produce longer shallower stops to reduce supersaturation in the slower tissues in the latter phase of the ascent.

To our knowledge, there has been no formal testing of VPM-based technical diving decompression schedules; however, many thousands of dives have been conducted by technical divers using this algorithm (V-Planner Live & Multideco Dive Database [Internet]. Kingston (ON, CAN): HHS Software Corp. [2004]-2012 [cited 2012 Aug 01]. Available from: http://database.hhssoftware.com/database.html). This fact, of itself, is often used by proponents as evidence of the algorithm's efficacy. However, this sort of anecdote deserves very cautious interpretation. Firstly, there is concern over positive reporting bias for dives of good outcome. Secondly, many such dives are not conducted to the limits of the algorithm and therefore do not validate the algorithm: tabulated schedules assume the full bottom time is spent at the maximum depth whereas the actual dive may be shallower and shorter; the conservatism of the algorithm is user-adjustable and many divers indulge in idiosyncratic 'padding' of their decompression for extra safety.

Deep stops

A characteristic of bubble algorithms is that they typically prescribe deeper decompression stops than gas-content algorithms[10,15–17] The potential benefit of these bubble-algorithm-prescribed 'deep stops' has been hypothesized since the 1960s.[15] In simple terms, the aim is to limit supersaturation (below levels normally accepted by gas-content algorithms) early in the decompression in order to limit bubble formation.

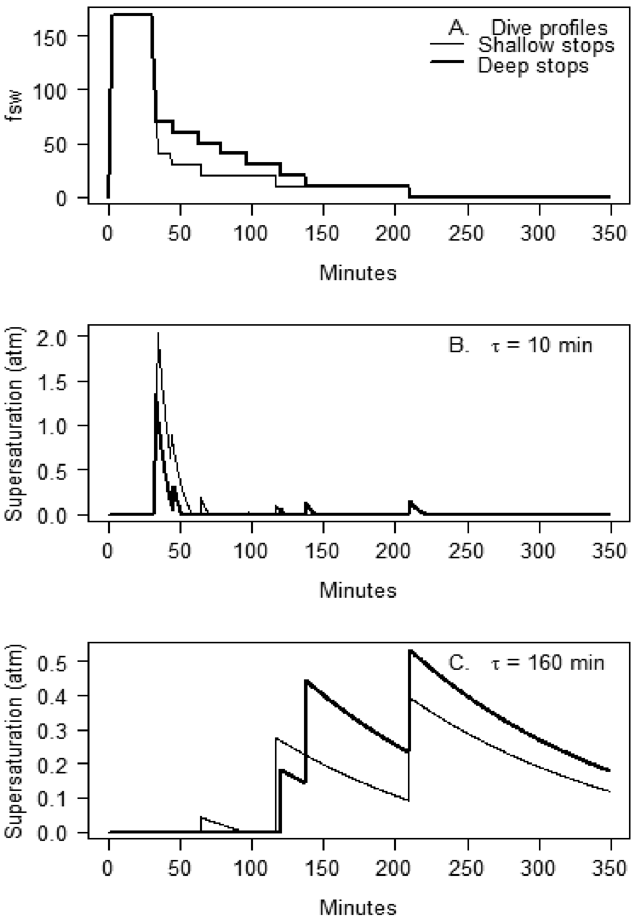
Deep stops came to the attention of early technical divers in the form of empirical 'Pyle stops', a practice serendipitously developed by ichthyologist Richard Pyle, arising from a requirement to vent the swim bladders of fish specimens collected at great depth before arriving at his first decompression stop.[18] There followed a strong trend toward the adoption of bubble algorithms, and also for the use of manipulation of gradient factors (see earlier) to force gas-content algorithms to impose deep stops. Based largely on supportive anecdote, there is a widespread belief among technical divers that deep-stop decompression schedules are more efficient than shallow-stop schedules. It is perceived that, compared to a decompression profile prescribed by a traditional gas-content algorithm, a deep-stop schedule of the same or even shorter duration has a lower risk of DCS. Recently, however, evidence has been accumulating from laboratory man-trials that shows deep stops are not more efficient than shallow stops for air or trimix dives.[17,19]

NEDU DEEP STOPS TRIAL

The largest of these trials was conducted at the US Navy Experimental Diving Unit (NEDU).[17] Submerged divers breathing surface-supplied air were compressed to 170 feet sea water (fsw, approx. 51.5 msw) for a 30-min bottom time, during which they performed 130 watt work on a cycle ergometer. They were then decompressed at 30 fsw per min^{-1} (approx. 9 msw min^{-1}) with stops prescribed by one of the two schedules shown in Figure 1A. Divers worked while on the bottom and were at rest and cold during decompressions. The shallow-stop schedule, with a first stop at 40 fsw (approx. 12 msw) and 174 min total stop time, was prescribed by the gas-content VVAL18 Thalmann algorithm. The deep-stop schedule, with a first stop at 70 fsw (approx. 21 msw), was the optimum distribution of 174 min total stop time according to the probabilistic BVM(3) bubble algorithm.[9] A higher incidence of DCS was observed on the deep-stop schedule (10 cases of DCS in 198 dives) than on the shallow-stop schedule (three cases

Figure 1

Supersaturation ($\Sigma P_{tis_j} \quad P_{amb} > 0$) in fast and slow compartments for the tested shallow-stop and deep-stop schedules.
A. Overlay of the two 170 fsw / 30 min air decompression dive profiles tested.
B. Supersaturation in a modelled compartment with fast (τ = 10 minutes) mono-exponential inert gas exchange.
C. Supersaturation in a modelled slow (τ = 160 minutes) compartment (redrawn from reference 17)



of DCS in 192 dives, P = 0.0489, one-sided Fisher's Exact test). Divers were also monitored for venous gas emboli (VGE) with trans-thoracic cardiac 2-D echo imaging, at 30 minutes and two hours after surfacing, both at rest and after limb flexion. The maximum VGE grade observed was significantly higher after the deep-stop schedule (median = 3) than after the shallow-stop schedule (median = 2; Wilcoxon rank sum test, W = 12967, $P < 0.0001$).

The BVM(3) bubble algorithm, used to produce the NEDU deep-stop schedule, predicts growth and dissolution of bubbles in three theoretical tissue compartments. It indicated substantial bubble growth on the shallow-stop schedule in the fast compartments (1- and 21-minute half-times) that required 'repair' with deep stops.[16] This is clearly at odds with the NEDU results. However, interpretation of the NEDU result simply requires a clear understanding of the relationship between tissue gas kinetics and bubble formation. In this regard, there are four important facts to keep in mind:

- bubbles form and grow only if the tissue is gas supersaturated, and the greater the supersaturation the more bubbles will form and the faster they will grow;
- supersaturated tissue has higher inert gas tension than the blood flowing into the tissue, so there is a net diffusion of inert gas from supersaturated tissue into the capillary blood, i.e., tissues that contain bubbles are losing, not taking up, inert gas;
- once inert gas washout has reduced inert gas partial pressure in tissue below that inside the bubble, the bubble shrinks;
- inert gas uptake and washout occurs at different rates in different body tissues. These different rates can be represented by compartments with different half-times.

Figure 1B shows gas supersaturation in a fast inert gas exchange compartment for the tested shallow- and deep-stop dive profiles illustrated in Figure 1A. This fast compartment (time constant, τ = 10 min, equivalent to half-time = 7 min) is notionally representative of all compartments that have comparatively fast gas exchange and in which an ascent to the shallow first decompression stop results in gas supersaturations greater than those produced by an ascent to a deeper first stop. The fast compartment in Figure 1B displays markedly lower and less sustained gas supersaturation (and therefore less driving force for bubble formation) during the deep-stop than during a comparable period of the shallow-stop schedule. This is consistent with the observation that a brief deep stop results in fewer Doppler-detectable VGE during decompression.[20] However, the NEDU results indicate that this reduction of gas supersaturation in fast compartments does not manifest in reduced DCS incidence. Put another way, the large ascent to the first stop in traditional schedules is not a flaw that warrants repair by deeper initial stops.

Figure 1C shows supersaturations in a notional slow compartment (τ = 160 min, half-time = 111 min) representative of all compartments having comparatively slow gas exchange and which are not gas supersaturated upon ascent to the deep first decompression stop. Inert gas will either wash out slowly or continue to be taken up into these slower compartments on the deep-stop schedule. Therefore, the deep-stop schedule results in greater and more persistent gas supersaturation in slow compartments on subsequent ascent than during the comparable period in the shallow-stop schedule. The observed differences in gas supersaturations in slower compartments late in the decompression are in accord with the present results from the tested dive profiles. The higher VGE scores and DCS incidence following the deep-stop schedule compared with the shallow-stop schedule may be a manifestation of bubble formation instigated by conditions in slower compartments.

Although the tested shallow- and deep-stop schedules are the optimal distributions of stop time under the VVAL18 Thalmann and BVM(3) algorithms, respectively, this does not mean that either schedule is the true optimal distribution of 174 minutes total stop time. Of interest is how alternative deep-stop schedules might have performed against the traditionally shaped shallow-stop schedule. Deep-stop schedules prescribed by VPM-based decompression software available to technical divers have deeper and shorter initial decompression stops than the tested deep-stop schedule (see, for instance, reference 10). However, analysis of half a million possible alternative distributions of stop times (which inevitably included profiles matching those more typical of technical diving) showed the same patterns as illustrated for the tested schedules in Figure 1: deeper stops reduced supersaturation in fast compartments at the expense of increased supersaturation in slow compartments compared to shallow-stop schedules.[17]

The impact of the technical diving practice of switching to a high fraction of oxygen for the decompression gas, or diving with a constant partial pressure of oxygen (PO_2) closed-circuit rebreather (see the first paper in this series) is relevant here. This accelerates decompression by faster washout of inert gas from all compartments, but will also result in less uptake of inert gas into slow compartments during deep stops. For instance, if the NEDU schedules in Figure 1 incorporated a switch to 50% O_2 / 50% N_2 at 70 fsw (21 msw), the supersaturations in slow compartments in panel C would be greatly reduced for both schedules. The risk of DCS would be reduced for both schedules, probably so that it would not be possible to distinguish a difference in DCS incidence between them.[16]

OTHER (NON-NEDU) DATA

Several air and trimix schedules with brief deep stops more like those conducted by technical divers have been compared to traditional gas-content algorithm schedules using venous gas emboli (VGE) counts as the endpoint in a limited number of man-dives. Despite longer decompression times, the deep-stop schedules resulted in the same or more VGE than the shallow-stop schedules, and some deep-stop dives resulted in symptoms of DCS.[19,21]

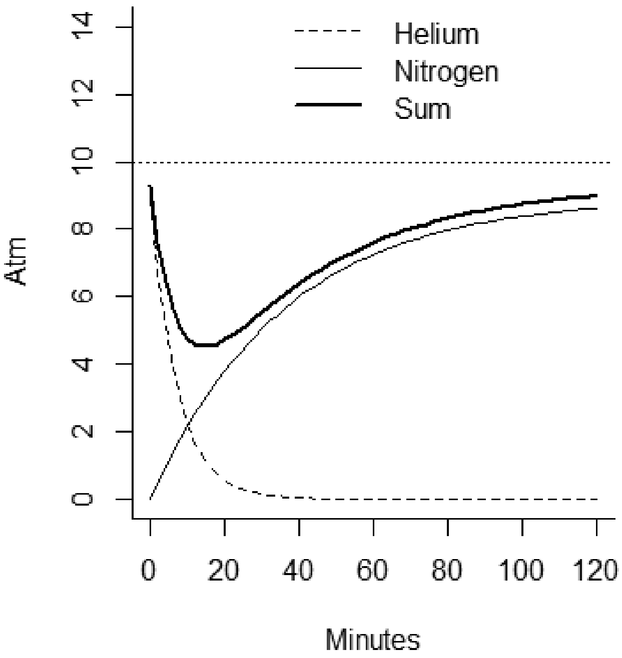
Diving with multiple inert gases

Owing to differing physicochemical properties, in some body tissues, helium is taken up and washed out faster than nitrogen. This difference can be seen in the whole-body washout of helium or nitrogen and appears to be important in tissues with slow gas kinetics (probably fat).[22–24] This slower washout of nitrogen in slowly exchanging tissues is manifest in a slower required rate of decompression from nitrox saturation dives (where the body has completely equilibrated with the elevated nitrogen partial pressure) than from heliox saturation dives of similar depth.[25–27]

A similar phenomenon is thought by some to be relevant in all bounce diving. In many decompression algorithms, helium is assumed to have faster exchange than nitrogen

Figure 2

Isobaric exchange of helium and nitrogen in a compartment with faster half-time for helium than nitrogen. Simulation of a compartment at equilibrium with 90% He/10% O_2 inspired gas at 10 atm abs ambient pressure and a switch to 90% N_2/10% O_2 inspired gas at time zero. Dashed and thin lines indicate partial pressures of helium and nitrogen. The thick line indicates the sum of both inert gases and metabolic gases. The compartment is transiently undersaturated while the sum of gases is below the equilibrium value (adapted from reference 24).



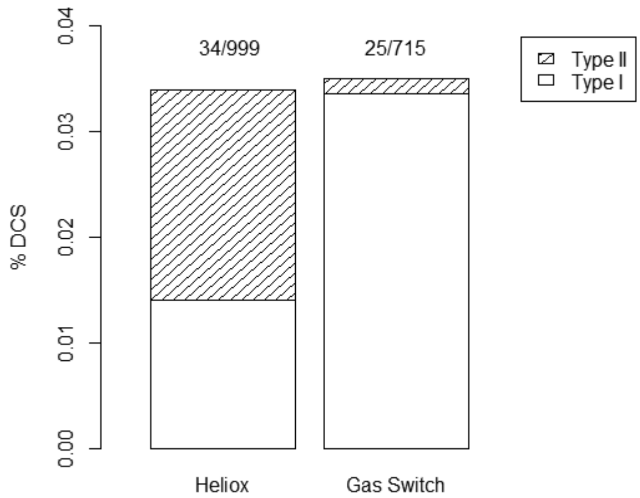
in all compartments. For instance, in the Buhlmann ZH-L16 gas-content decompression algorithm each of the 16 compartments has a half-time for helium that is 2.65 times shorter than the corresponding nitrogen half-time.[4] These, or similar, compartment half-times are used in most decompression algorithms available to technical divers. As a result of these compartment half-times, such decompression algorithms will prescribe shallower decompression stops and less total decompression time for a bounce dive conducted breathing nitrox than for a dive conducted breathing trimix or heliox because of a slower uptake of nitrogen than of helium.[5] Similarly, such decompression algorithms will prescribe shorter decompressions if switching to nitrox breathing during decompression from a heliox or trimix dive.[5] The reason for this latter effect is illustrated in Figure 2, which shows that faster helium washout than nitrogen uptake in a compartment will result in a period of under-saturation (making the safe ascent depth shallower) following a heliox-to-nitrox gas switch.

HELIOX TO NITROX GAS SWITCH MAY NOT ACCELERATE DECOMPRESSION FOR BOUNCE DIVES

It is not clear that the apparent differences in bounce diving decompression resulting from different inert gases are real.

Figure 3

Comparison of incidences of Type I and Type II DCS during development of constant 1.3 atm inspired PO_2-in-helium decompression schedules:[30,31] left (Heliox) and during development of surface supplied (84/16 He/O_2) decompression schedules;[32] right (Gas Switch). Only the latter employs a switch to air breathing during decompression. Although there are substantial differences between these two types of diving, dives were across a similar range of depths and bottom times and resulted in similar overall incidences of DCS (actual number DCS/dives indicated above bars). Although these studies were not designed for this purpose, the different incidences of Type II DCS are noteworthy.



Direct measurement of helium and nitrogen exchange rates in faster exchanging tissues relevant to bounce diving indicates very similar rates of exchange for nitrogen and helium.[24] These latter data suggest heliox, nitrox, and trimix decompression from bounce dives of the same depth and duration should be similar. This is supported by the finding that wet, working no-stop dives to 60 fsw (18 msw) for bottom times of 70 to 100 minutes showed a similar incidence of DCS whether breathing air or heliox (21% oxygen), indicating a similar rate of helium and nitrogen uptake and similar decompression requirement.[28]

The scant data relevant to actual decompression diving can be interpreted as 'conflicting'. The oft-cited work supporting accelerated decompression by switching from heliox to nitrox in fact shows nothing of the sort.[29] This work presents several dives with changes in inert gas composition and increases in oxygen fraction up to 100% during decompression and compares decompression time to schedules from (or extrapolated from) the US Navy 1957 Standard Air Tables that were not actually tested. Later experiments comparing dives with heliox-to-air gas switching to dives with all heliox decompression are confounded by different decompression schedules and small numbers of dives, particularly on the schedules that provoked DCS.[5] On the other hand, a US Navy man-trial indicated that a heliox to nitrox switch does not accelerate decompression.[30] In that study, 32 man-dives to 300 fsw (approx. 90 msw) for 25 min breathing 1.3 atm inspired oxygen partial pressure (P_iO_2)-in-helium for the entire bottom time and throughout the 190 minutes of decompression resulted in only one case of DCS, whereas 16 man-dives with an identical depth-time profile and P_iO_2 but a switch to nitrox at the first decompression stop (110 fsw, approx. 33 msw) resulted in three DCS. This difference in incidence does not reach statistical significance, but there is a strong trend indicating no advantage (and perhaps even a disadvantage) for the gas switch.

In very long decompressions, nominally over 10 hours, a switch from heliox or trimix to nitrox late in decompression may accelerate decompression. This is because the long shallow stops of such 'sub-saturation dives' are governed by the slow compartments that govern saturation decompression in a similar way, in which helium exchanges faster than nitrogen. However, there is no direct experimental evidence to support this. Another possible advantage of a heliox-to-nitrox gas switch is that it may result in less Type II DCS (Figure 3).

Inner-ear decompression sickness

Although a heliox-to-nitrox breathing gas switch may accelerate decompression in a very long dive, and may result in less Type II DCS, such switches have also been associated with the onset of DCS involving the vestibulocochlear apparatus (inner ear).[33–36] Though infrequent, inner-ear DCS is of particular concern to technical divers because debilitating symptoms characteristically onset during decompression and are life-threatening for a scuba diver with substantial remaining decompression obligation.[36]

A physiological model of the inner ear indicates that at great depths, following a switch from a helium-based breathing mixture to one containing nitrogen, transient supersaturation can develop in the vascularized membranous labyrinth without any further change in depth, principally due to diffusion of helium from the endolymph and perilymph exceeding the counter-diffusion of nitrogen in the opposite direction.[36] However, the same model demonstrates substantial pre-existing supersaturation in the inner ear during decompression from typical technical bounce dives, and that the counter-diffusion of gases following a helium-to-nitrogen mix gas switch makes only a small contribution to the total supersaturation at the depths where such switches are usually made.[36] The actual contribution of gas switches to inner-ear DCS in this setting is, therefore, uncertain. During sub-optimal decompression of the inner ear, gas switches could conceivably act as "*the straw that broke the camel's back*". Otherwise, such switches will usually not result in problems. Indeed, heliox-to-air breathing gas switches at 30 msw during decompression have been demonstrated to have low risk of DCS.[37] Thus, inner-ear DCS that seems related to gas switching in technical divers is more likely to be due to inadequate decompression *per se* prior to the breathing gas switch.

Isolated inner-ear DCS has also been described early after surfacing in divers visiting more modest depths (50 msw or less).[38,39] Interestingly, among such cases there is an unexpectedly high prevalence (100% in one series of 9 divers) of major right-to-left shunting demonstrated using transcranial Doppler sonography after administration of venous bubble contrast.[38,39] The nature of the right-to-left shunt in these studies was not established, but right-to-left shunting of VGE through a patent foramen ovale (PFO), which is present in about 25% of the population, has been identified as a potential risk factor for some forms of DCS, including inner-ear DCS.[40–42] Indeed, the contribution of PFO to the risk of DCS is an article of faith among technical divers. However, recent evidence indicates that 30–60% of divers without PFO also shunt venous bubbles to the arterial circulation after routine asymptomatic dives, presumably via intrapulmonary routes.[43–45]

Whatever the means by which they reach the arterial circulation, if VGE reach the labyrinthine artery, they must also distribute widely in the brain because the labyrinthine artery is usually a tiny branch of the much larger basilar artery. Despite this, these divers frequently do not develop cerebral manifestations. The selective vulnerability of the inner ear in this setting may relate to slower inert gas washout, and therefore more prolonged supersaturation, in the inner ear than the brain. Under these circumstances, small arterial bubbles reaching the inner ear are more likely to grow and cause symptoms than bubbles reaching the brain.[46]

This mechanism may also be relevant to the onset of inner-ear DCS at depth during decompression, which is when inner-ear symptoms characteristically occur in technical diving. It is notable that arterial bubbles (presumably shunted from the veins) have been detected in 70% of divers following typical, asymptomatic VPM-planned technical dives.[45] It is not known if such arterial bubbles would also be detected during decompression, but, if so, the passage of bubbles into a supersaturated inner-ear microcirculation provides another possible explanation for DCS in this setting.

Supportive but admittedly circumstantial evidence for a role for arterial bubbles in inner-ear DCS during decompression can be inferred from saturation diving. In one study, arterial bubbles were detected in all six participating divers between one and four hours following 10 msw min^{-1} upward excursions from a 300 msw saturation storage depth to 250 msw.[47] There were no symptoms of serious DCS in these divers, but isolated inner-ear symptoms are an infrequent but characteristic manifestation of DCS following rapid (8–18 msw min^{-1}) upward excursions (38–50 msw) from deep (200–430 msw) heliox saturation dives.[48] The previously described model of the inner ear indicates these excursions could produce sufficient supersaturation in the inner ear to cause local tissue bubble formation.[36] Nevertheless, the presence of arterial bubbles raises the possibility that isolated inner-ear DCS following upward excursions from heliox saturation could also result from passage of arterial bubbles into a supersaturated inner-ear microcirculation.

Although inner-ear DCS occurs relatively unpredictably, the putative pathophysiology described above suggests several goals to mitigate the risk of inner-ear DCS during decompression from technical dives. First, the chosen algorithm should adequately decompress the inner ear which has a half-time of about 8.8 minutes.[36] Second, if gas switches are made, they should be made at points during the decompression that avoid peak supersaturation of the inner ear and the shallower the better with no undue risk found with switches at 30 msw.[37] Third, since the right-to-left shunting of bubbles via either a PFO or the trans-pulmonary route is associated with high-grade VGE, the chosen algorithm should not consistently produce high VGE grades. Finally, since trans-pulmonary passage of VGE is potentially reduced by oxygen breathing, utilization of the highest safe inspired oxygen fraction at all times, and particularly after gas switches is likely to be beneficial.[36,44]

Summary

Technical divers use a variety of decompression algorithms although many are modifications of the ZH-L16, Tonawanda II and VPM algorithms. These modifications have generally not been validated by conduct of human dive trials comprising well-documented dives and outcomes. Many thousands of dives have been conducted safely in the field but the incidence of DCS in technical diving is unknown, and it is unknown if technical diving decompression procedures are optimal. Scientific evidence supports some technical diving procedures although not always for the reasons they were originally adopted.

References

1 Mitchell SJ, Doolette DJ. Recreational technical diving: part 1: an introduction to technical diving methods and activities. *Diving Hyperb Med*. 2013;43:86-92.

2 Hamilton RW, Turner P. Decompression techniques based on special gas mixes for deep cave exploration [abstract]. *Undersea Biomed Res*. 1988;15(Suppl):70.

3 Bühlmann AA. Experimentalle grundlagen der risikoarmen dekompression nach überdruckexpositionen. *Schweiz Med Wochenschr*. 1982;112:48-59. German

4 Bühlmann AA. Die berechnung der risikoarmen dekompression. *Schweiz Med Wochenschr*. 1988;118:185-97. German

5 Bühlmann AA. Decompression - decompression sickness. Michel GP, translator. Berlin: Springer-Verlag; 1984. *English translation of Dekompression – dekompressionkrankheit,* first published 1983.

6 Boycott AE, Damant GCC, Haldane JS. The prevention of compressed-air illness. *J Hygiene (Lond)*. 1908;8:342-443.

7 Workman RD. American decompression theory and practice. In: Bennett PB, Elliott,DH, editors. *The physiology and medicine of diving and compressed air work*, 1st ed. London: Ballière, Tindall, and Cassell; 1969. p. 252-90.

8 Gernhardt, ML. *Development and evaluation of a decompression stress index based on tissue bubble dynamics* [PhD thesis]. Philadelphia: University of Pennsylvania; 1991. 313 p.

9 Gerth WA, Vann RD. Probabilistic gas and bubble dynamics models of decompression sickness occurrence in air and N_2-O_2 diving. *Undersea Hyperb Med*. 1997;24:275-92.

10 Yount DE, Hoffman DC. On the use of a bubble formation model to calculate diving tables. *Aviat Space Environ Med*. 1986;57:149-56.

11 Yount DE, Maiken EB, Baker EC. Implications of the varying permeability model for reverse dive profiles. In: Lang MA, Lehner CE, editors. *Proceedings of the reverse dive profiles workshop*; *1999 Oct 29-30; Washington DC*. Washington DC: Smithsonian Institution; 2000. p. 29-60. [cited 2013 Mar 3] Available from: http://www.decompression.org/maiken/VPM/RDPW/rdpw.pdf

12 Wienke BR. Reduced gradient bubble model. *Int J Biomed Comput*. 1990;26:237-56.

13 Doolette DJ. Decompression practice and health outcome during a technical diving project. *SPUMS Journal*. 2004;34:189-95.

14 Baker EC. Clearing up the confusion about "deep stops". *Immersed*. 1998;3:23-31. [cited 2013 Mar 3] Available from: http://liquivision.com/docs/deepstops.pdf

15 Hills, BA. *A thermodynamic and kinetic approach to decompression sickness* [PhD thesis]. Adelaide: The University of Adelaide; 1966. p. 370.

16 Gerth WA, Doolette DJ, Gault KA. Deep stops and their efficacy in decompression. In: Vann RD, Mitchell SJ, Denoble PJ, Anthony TG, editors. *Technical diving conference proceedings 2008*. Durham (NC): Divers Alert Network; 2009. p. 138-56. [cited 2012 Mar 3 login required] Available from: http://www2.dan.org/fastaccess/2008TechnicalDiving.aspx

17 Doolette DJ, Gerth WA, Gault KA. *Redistribution of decompression stop time from shallow to deep stops increases incidence of decompression sickness in air decompression dives. Technical Report*. Panama City (FL): Navy Experimental Diving Unit; 2011 Jul. Report No.: 11-06.

18 Pyle RL. The importance of deep safety stops: rethinking ascent patterns from decompression dives. *SPUMS Journal*. 1997;27:112-5.

19 Blatteau J-E, Hugon M, Gardette B. Deeps stops during decompression from 50 to 100 msw didn't reduce bubble formation in man. In: Bennett PB, Wienke BR, Mitchell SJ, editors. *Decompression and the deep stop*. Undersea and Hyperbaric Medical Society Workshop; 2008 Jun 24-25; Salt Lake City (UT). Durham (NC): Undersea and Hyperbaric Medical Society; 2009. p. 195-206. [cited 2013 Mar 3] Available from: http://underseaandh960.corecommerce.com/WORKSHOPS-AND-OTHER-REPORTS/Decompression-and-the-Deep-Stop-Workshop-Proceedings-p13.html

20 Neuman TS, Hall DA, Linaweaver PG. Gas phase separation during decompression in man: ultrasound monitoring. *Undersea Biomed Res*. 1976;3:121-30.

21 Blatteau J-E, Hugon M, Gardette B, Sainty J-M, Galland F-M. Bubble incidence after staged decompression from 50 or 60 msw: effect of adding deep stops. *Aviat Space Environ Med*. 2005;76:490-2.

22 Behnke AR, Willmon TL. Gaseous nitrogen and helium elimination from the body during rest and exercise. *Am J Physiol*. 1940;131:619-26.

23 Duffner GJ, Snider HH. *Effects of exposing men to compressed air and helium-oxygen mixtures for 12 hours at pressures of 2-2.6 atmospheres. Technical Report*. Panama City (FL): Navy Experimental Diving Unit; 1958 Sep. Report No.: 1-59.

24 Doolette DJ, Mitchell SJ. Hyperbaric conditions. *Compr Physiol*. 2011;1:163-201.

25 Eckenhoff RG, Vann RD. Air and nitrox saturation decompression: a report of 4 schedules and 77 subjects. *Undersea Biomed Res*. 1985;12:41-52.

26 Summitt JK, Alexander JM, Flynn ET, Herron JM, Kulig JW. *Repetitive excursions from saturated depths on helium-oxygen mixtures. Phase II: saturation depth 200 feet; saturation depth 150 feet. Research Report*. Washington DC: Navy Experimental Diving Unit; 1970 Sep. Report No.: 6-70.

27 Summitt JK, Kulig JW. *Saturation dives, with excursions, for the development of a decompression schedule for use during Sealab III. Research Report*. Washington DC: Navy Experimental Diving Unit; 1970 Sep. Report No: 9-70.

28 Hamilton RW, Thalmann ED, Flynn ET, Temple DJ. No-stop 60 fsw wet and dry dives using air, heliox, and oxygen-nitrogen mixtures. *Data report on projects 88-06 and 88-06A. Technical Report*. Silver Spring (MD): Naval Medical Research Center; 2002 Jul. Report No.: 2002-002.

29 Keller H, Bühlmann AA. Deep diving and short decompression by breathing mixed gases. *J Appl Physiol*. 1965;20:1267-70.

30 Survanshi SS, Parker EC, Gummin DD, Flynn ET, Toner CB, Temple DJ, et al. *Human decompression trial with 1.3 ATA oxygen in helium. Technical Report*. Bethesda (MD): Naval Medical Research Institute; 1998 Jun. Report No.: 98-09.

31 Gerth WA, Johnson TM. *Development and validation of 1.3 ATA PO2-in-He decompression tables for the MK 16 MOD 1 UBA. Technical Report*. Panama City (FL): Navy Experimental Diving Unit; 2002 Aug. Report No.: 02-10.

32 Tikuisis P, Nishi RY. *Role of oxygen in a bubble model for predicting decompression illness. Report*. North York (ON, CAN): Defence and Civil Institute of Environmental Medicine; 1994 Jan. Report No.: 94-04.

33 Farmer JC, Thomas WG, Youngblood DG, Bennett PB. Inner ear decompression sickness. *Laryngoscope*. 1976;86:1315-27.

34 Bennett PB, Vann RD, Roby J, Youngblood D. Theory and development of subsaturation decompression procedures for depths in excess of 400 feet. In: Shilling CW, Beckett MW, editors. *Underwater physiology VI. Proceedings of the 6th Symposium on Underwater Physiology*. Bethesda (MD): Federation of American Societies for Experimental Biology; 1978. p. 367-81.

35 Bühlmann AA, Gehring H. Inner ear disorders resulting from inadequate decompression – "vertigo bends". In: Lambertsen CJ, editor. *Underwater Physiology V. Proceedings of the 5th Symposium on Underwater Physiology*. Bethesda: FASEB; 1976. p. 341-7.

36 Doolette DJ, Mitchell SJ. A biophysical basis for inner ear decompression sickness. *J Appl Physiol*. 2003;94:2145-50.

37 Doolette DJ, Gerth WA, Gault KA, Murphy FG. *Safe inner ear inert gas tension for switch from heliox to air breathing at 100 fsw during decompression*. Panama City (FL): Navy Experimental Diving Unit; forthcoming. Report No.: 12-04.

38 Klingmann C, Benton PJ, Ringleb PA, Knauth M. Embolic inner ear decompression illness: correlation with a right-to-left shunt. *Laryngoscope*. 2003;113:1356-61.

39 Cantais E, Louge P, Suppini A, Foster PP, Palmier B. Right-to-left shunt and risk of decompression illness with cochleovestibular and cerebral symptoms in divers: case control study in 101 consecutive dive accidents. *Crit Care Med*. 2003;31:84-8.

40 Moon RE, Camporesi EM, Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet*. 1989;1:513-4.

41 Wilmshurst PT, Ellis BG, Jenkins BS. Paradoxical gas embolism in a scuba diver with an atrial septal defect. *BMJ*. 1986;293:1277.

42 Bove AA. Risk of decompression sickness with patent foramen ovale. *Undersea Hyperb Med*. 1998;25:175-8.

43 Ljubkovic M, Dujic Z, Møllerløken A, Bakovic D, Obad A, Breskovic T, et al. Venous and arterial bubbles at rest after no-decompression air dives. *Med Sci Sports Exerc*. 2011;43:990-5.

44 Ljubkovic M, Zanchi J, Breskovic T, Marinovic J, Lojpur M, Dujic Z. Determinants of arterial gas embolism after scuba diving. *J Appl Physiol*. 2012;112:91-5.

45 Ljubkovic M, Marinovic J, Obad A, Breskovic T, Gaustad SE, Dujic Z. High incidence of venous and arterial gas emboli at rest after trimix diving without protocol violations. *J Appl Physiol.* 2010;109:1670-4.

46 Mitchell SJ, Doolette DJ. Selective vulnerability of the inner ear to decompression sickness in divers with right to left shunt: the role of tissue gas supersaturation. *J Appl Physiol*. 2009;106:298-301.

47 Brubakk AO, Peterson R, Grip A, Holand B, Onarheim J, Segadal K, et al. Gas bubbles in the circulation of divers after ascending excursions from 300 to 250 msw. *J Appl Physiol*. 1986;60:45-51.

48 Thalmann ED. Testing of revised unlimited-duration upward excursions during helium-oxygen saturation dives. *Undersea Biomed Res.* 1989;16:195-218.

Submitted: 06 January 2013
Accepted: 23 February 2013

David J Doolette, PhD, is a research physiologist at the Navy Experimental Diving Unit, Panama City, Florida, USA.
Simon J Mitchell, PhD, FANZCA, is Associate Professor and Head, Department of Anaesthesiology, University of Auckland, Auckland, New Zealand.

Address for correspondence:
Dr David Doolette
Navy Experimental Diving Unit
321 Bullfinch Road
Panama City Beach, Florida 7012
USA
Phone: *+1-(850)-230-3100*
E-mail: *<david.doolette.as@navy.mil>*