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Address for correspondence. Dr Christopher S Butler MB BS, DipDHM, 21 Lindsay Street, Ashgrove, Queensland, 4060, Australia.

ON NO-STOP TIME LIMITS, SAFETY STOPS AND ASCENT RATES

Bruce Wienke

Introduction

The past ten years, or so, have witnessed a number of important changes in diving protocols and table procedures, such as shorter no-stop time limits, slower ascent rates, discretionary safety stops, repetitive dive profiles requiring all dives to be shallower than the one before, multi-level techniques, both faster and slower tissue half-times controlling repetitive dives, lower critical tensions (M-values) and longer flying-after-diving surface intervals. Stimulated by

Doppler technology, decompression meter development, theory, statistics, or safer diving concerns, these modifications affect a gamut of activity, spanning bounce to multi-day diving. Of these changes, conservative no-stop time limits, non-decompression safety stops and slower ascent rates (less than the standard 18m (60 ft)/min) are much in vogue, and deserve a closer look. As it turns out, there is good support for shorter no-stop limits, safety stops, and slow ascent rates on practical, experimental and theoretical grounds.

Discretionary protocols

Spencer[1] poineered bubble counting by the use of Doppler. His results, showing many bubbles at no-stop limits, led him to suggest reductions in the no-stop limits of the US Navy (USN) tables. A reduction in the M value, of the order of a repetitive group or two at each depth in the tables (1-4 ft in critical tensions), was recommended to lower bubble counts. Others have made similar recommendations over the past 15 years.

Smith and Stayton[2] noted marked reductions in precordial bubbles when ascent rates were cut from 18 m (60 ft)/min to 9 m (30 ft)/min. In similar studies, Pilmanis[3] and Neuman, Hall and Linaweaver[4] witnessed an order of magnitude drop in venous gas emboli (VGE) counts in divers making short safety stops following bounce exposures to 30m (100 ft).

An American Academy of Underwater Sciences (AAUS) workshop on repetitive diving, recorded by Lang and Vann,[5] and Divers Alert Network (DAN) statistics[6] suggest that present diving practices become riskier with increasing exposure time and pressure (depth) This evidence has encouraged the development of ancillary safety measures for multi-level, repetitive and multi-day diving. Dunford, Wachholz, Huggins and Bennett[7] noted persistent Doppler scores in divers performing repetitive, multi-day diving, suggesting the presence of VGE in divers, all the time, under such loadings.

Ascent rates, safety stops, decompression computers and altitude diving were also the subject of extensive discussion at workshops and technical forums sponsored by the American Academy of Underwater Sciences and the Undersea and Hyperbaric Medical Society (UHMS) and have been summarized by Lang and Hamilton,[8] Lang and Egstrom[9] and Sheffield.[10] The discussions culminated in a set of recommendations, based on standard Haldane[11] table and meter procedures, even for exposures not exceeding time limits nor critical tissue tensions.

The upshot of these studies, workshops, discussions and tests is a set of discretionary protocols, not necessarily endorsed in all diving sectors, but which might be summarized as follows:

1 reduce no-stop time limits a repetitive group, or two, below the standard USN limits;
2 keep ascent rates below 18 m (60 ft)/min, preferably slower and required to be slower at altitude;
3 limit repetitive dives to a maximum of three per day, none exceeding 30 m (100 ft);
4 avoid multi-day, multi-level, or repetitive dives to increasing depths;
5 wait 12 hours before flying after no-stop diving, 24 hrs after heavy diving (taxing, near decompression, or prolonged repetitive) activity, and 48 hrs after decompression diving;
6 avoid multiple ascents to the surface and short repetitive dives (spikes) with surface intervals less than 1 hour;
7 surface intervals of more than an hour are recommended for repetitive diving;
8 safety stops for 2-4 minutes in the 3-9 m (10-20 ft) zone are advisable for all diving, but particularly for deep, near 30 m (100 ft), repetitive and multi-day exposures;
9 do not dive at altitudes above 10,000 ft using modified conventional tables, or linear extrapolations of sea-level critical tensions;
10 in short, dive conservatively, remembering that tables and meters are not bends-proof.

Procedures such as those above are prudent, theoretically sound and accepted safe diving practice. Ultimately, they can all be linked to bubble decompression models, and our interests here are no-stop limits, safety stops and ascent rates. In considering these items, a quick look at bubbles and related dynamics is first necessary.

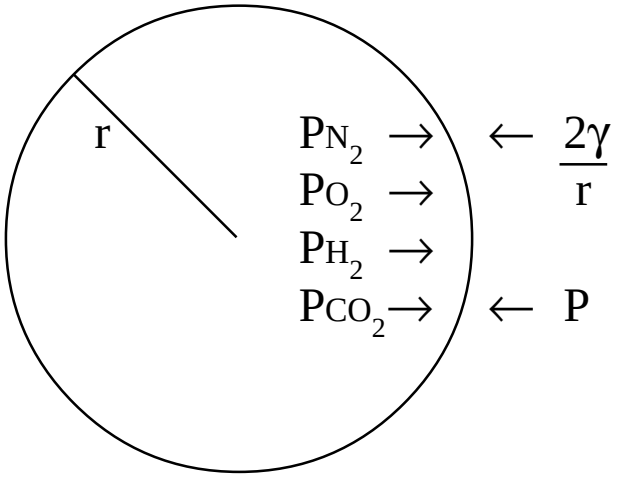
Bubble dynamics

Internal pressures in bubbles exceed ambient pressures by amounts equal to the effective surface tensions of the bubbles (Figure 1). To eliminate bubbles, or reduce their growth, increased ambient pressure is required, not only to restrict the size, but also to drive gas out of the bubble by diffusion, and across the tissue-bubble interface (Figure 2). The shorter the desired time of elimination, the greater must be the ambient pressure. Experiments conducted in decompressed gels, notably by Yount and Strauss,[12] Kunkle and Beckman,[13] and Yount,[14,15] have been illuminating, showing that the smaller the bubble, the shorter the dissolution time (Figure 3).

The implications for diving are clear. In the presence of even asymptomatic bubbles increased off-gassing pressure is prudent. At any pressure, the length of time required to dissolve bubbles of 250 micron diameter is significantly shorter than that required to dissolve larger bubbles. Immediate recompression, within less than 5 minutes, is adequate treatment for bubbles less than 100 microns in diameter and forms the basis for Hawaiian emergency in-water recom-

FIGURE 1

BUBBLE PRESSURE BALANCE



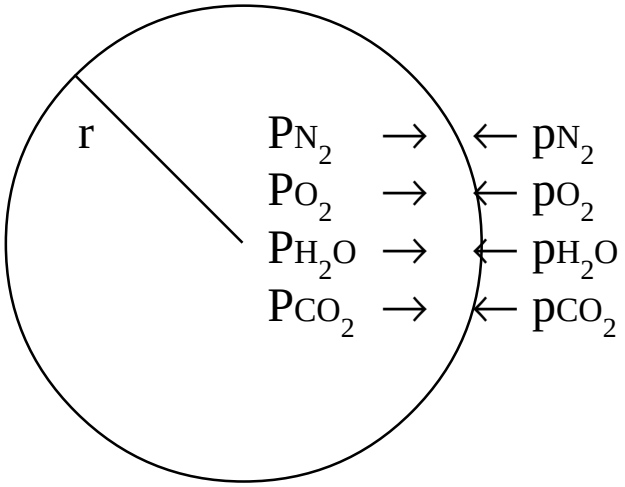
The total gas pressure, P_t within an air bubble equals the sum of ambient pressure, P, plus effective surface tension, 2γ/r, according to,

$$P_t = P + 2\gamma/2$$
$$P_t = PO_2 + PN_2 + PH_2O + PCO_2$$

At small radii, surface tension effects are large, while at large radii effects of surface tension vanish. Effective surface tension is the difference between Laplacian (thin film) tension and skin (surfactant) tension. Stabilized nuclei exhibit zero effective surface tension, so that total gas pressures and tensions are equal. When nuclei are destabilized (bubbles), any gradients between free and dissolved gas phases will drive the system to different configurations, that is, expansion or contraction, until a new equilibrium is established.

FIGURE 2

BUBBLE GAS DIFFUSION



An air bubble in hydrostatic equilibrium will grow or contract, depending on its size and any relative gradients between free gas in the bubble and dissolved gas in tissue. Gradients are inward if tissue tensions exceed bubble gas presures and outward if bubble gas pressures exceed tissue tensions. A critical radius, r_c, separates growing from contracting bubbles for a given set of pressures. The critical radius depends on the total tension, p_t, ambient pressure, P, and effective surface tension, γ,

$$r_c = \frac{2\gamma}{p_t - P}$$
$$p_t = p_{N_2} + p_{O_2} + p_{H_2O} + p_{CO_2}$$

where growth occurs for $r>r_c$ and contraction for $r>r_c$. Some stabilized gas micronuclei in the body can always be excited into growth by pressure changes (compression-decompression).

pression procedures. If one assumes that gel and tissue bubbles respond to pressure in much the same manner, these facts support the arguments for safety stops when conventional tables are pushed to the limits of times or on repetitive dives.

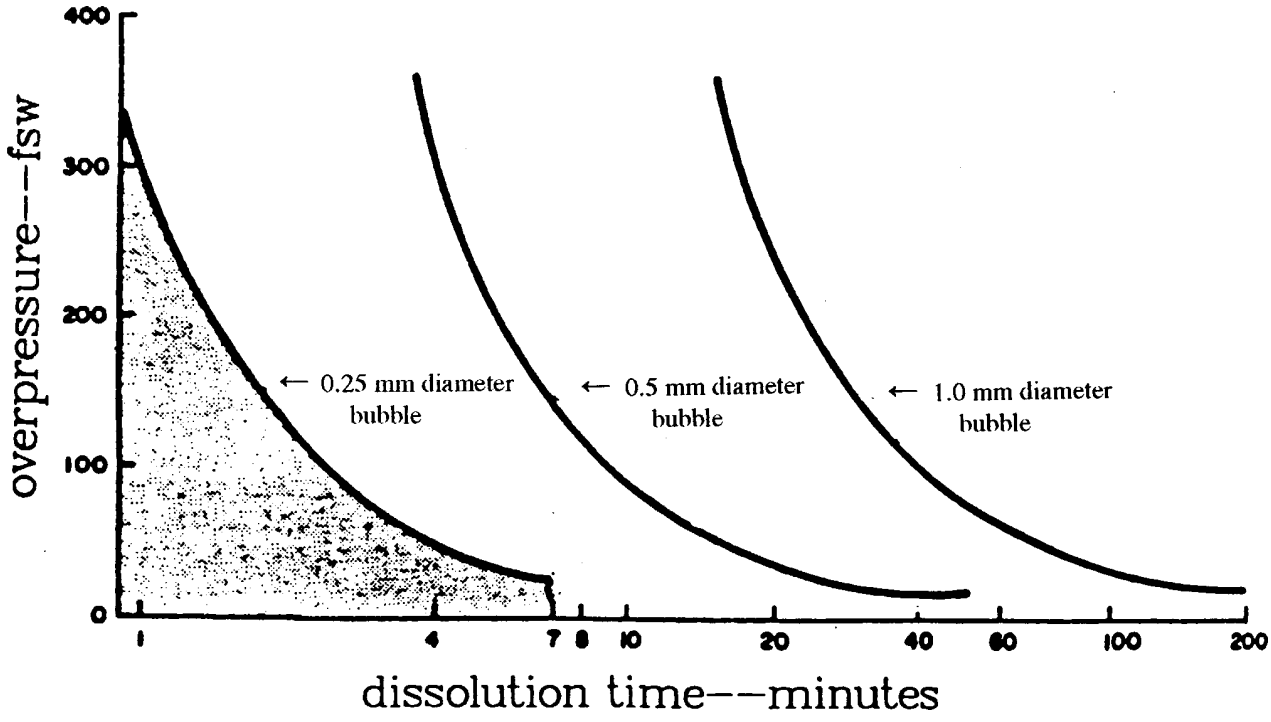
Bubbles, which are unstable, might grow from micron size gas nuclei, formed and stabilized over short periods of time and resisting collapse due to permeable skins of surface-activated molecules (surfactants), or possibly by reduction in surface tension at tissue interfaces or crevices. Gas nuclei seem to pervade all manner of fluids and their existence in blood serum and egg albumin has been established by Yount and Strauss.[12] Families of micronuclei vary in size and surfactant content.

Micronulcei theoretically are small enough to pass through the pulmonary vascular bed filters, yet dense enough not to float to the surfaces of their environments, with which they are in both hydrostatic (pressure) and diffusion (gas flow) equilibrium. When nuclei are stabilized their net surface tension is zero. Then all pressures and gas tensions are equal.

However, on decompression, these stable pockets, which have had extra gas diffuse into them during the period of compression, can be destabilized by the reduced ambient pressure, so that net surface tension is no longer zero, and subsequently they can enlarge into bubbles, which will expand as surrounding gas diffuses into them. The rate at which bubbles grow, or contract, depends directly on the difference between tissue tension and the local ambient pressure, effectively the supersaturation gradient. At some point in time, a critical volume of bubbles, or separated gas, is established and bends symptoms become statistically more probable.

FIGURE 3

DISSOLUTION TIME FOR GRADED BUBBLES



Bubbles develop and grow over longer time scales than nuclear stabilization. Yet, the rapid dissolution of bubbles in decompressed saturated gelatin (and the body proper) requires immediate and adequate repressurization. The absolute length of time required to dissolve bubbles with given overpressure is directly proportional to the size of the bubble. Obviously, the smaller the bubble, the shorter the time needed to dissolve that bubble at any overpressure. The bubbles studied in this experiment by Kunkle and Beckman grew to approximately 1 mm in 5 hours, starting from stabilized micronuclei. Such experiments have provided vital information, corroborating nucleation and bubble theories in vitro.

Nucleation

Nucleation-bubble theory is consistent with various diving observations. Divers can significantly increase tolerance against bubble formation and therefore bends, by following three simple practices, originally suggested by Strauss,[16] Evans and Walder,[17] and many others:

1 make the first dive a deep, short (crush) dive, to compress micronulcei down to a smaller, safer size;
2 make succeeding dives progressively shallower, that is diving within the crush limits of the first dive and so minimizing excitation of smaller micronuclei;
3 make frequent dives (like every other day), to deplete the number of micronulcei available to form bubbles.

The mechanics of nucleation, stabilization and bubble growth are fairly complex, with stabilization mechanisms only recently quantified. Source and generation mechanisms before stabilization are not well understood. Some candidates include cosmic radiation and charged particles, dissolved gases in the fluid we drink, lymph draining from tissues into veins, collisional coalescence, blood turbulence and vortices, exercise, the stomach and the thin air-blood endothelium in the lungs. More direct methods of bubble formation are also certainly possible. Cavitation, produced by the rapid tearing or moving apart of tissue interfaces, is a candidate, as well as surface friction (tribonucleation). Crevices in tissues may form or trap gas phases, with later potential for release. Vortices in blood flow might cause small microbubbles. Whatever the production and stabilization mechanisms of micronuclei, once destabilized the ensuing bubbles follow the dynamic growth and contraction patterns shown in Figures 1 and 2.

Stable or unstable, the presence of copious microbubbles in the venous circulation would affect dissolved gas elimination adversely, possibly impairing the lungs or escaping into the arterial network. The presence of bubbles in the arterial circulation might result in emboli. The

chokes, a serious form of decompression sickness, is thought to be due to bubbles clogging the pulmonary circulation. Cerebral decompression sickness is believed by some to be due to arterial emboli. Microbubbles in the venous circulation would render gas uptake and elimination more asymmetrical than it normally is by slowing down elimination. Displacing blood, microbubbles would reduce the effective area and volume for tissue-blood gas exchange.

Sites

Bubbles may hypothetically form in the blood (intravascular) or outside the blood (extravascular). Once formed, intravascularly or extravascularly, a number of critical insults are possible. Intravascular bubbles may induce blood sludging and chemistry changes. Circulating gas emboli may clog the pulmonary filters, and occlude the arterial flow. Extravascular bubbles may remain locally in tissue sites, enlarging by diffusion from adjacent supersaturated tissue, and compress nerves or compress a blood vessel and occlude it causing ischaemia. Extravascular bubbles can also pass through capillary walls and so enter veins, at which point they become intravascular bubbles.

Many doubt that bubbles form in the blood directly, but intravascular bubbles have been seen in both the arterial and venous circulation after very rapid decompression, with vastly greater numbers detected in venous flow known as venous gas emboli(VGE). Ischaemia resulting from bubbles caught in the arterial network has long been invoked as a cause of decompression sickness. Since the lungs are effective filters of venous bubbles, arterial bubbles must either form in the arteries or have bypassed the lung. The more numerous venous bubbles are suspected to form first in lipid tissues drained by the veins. Lipid tissue sites also posses very few nerve endings, possibly masking critical insults. Veins being thinner than arteries, are more susceptible to extravascular gas penetration.

Extravascular bubbles may form in aqueous (watery) or lipid (fatty) tissues in principle. For all but extreme, or explosive, decompression bubbles are seldom observed in muscles or liver tissue. Most gas is seen in fatty tissue, not surprisingly considering the five-fold higher solubility of nitrogen in lipid tissue compared to aqueous tissue. Since fatty tissue has few nerve endings, tissue deformation by bubbles is unlikely to cause pain locally. On the other hand, formations of large volumes of extravascular gas which then enters the capillaries could induce vascular damage, depositing both fat and bubbles into the circulation as has been seen in animal experiments. If mechanical pressure on nerves is a prime candidate for the critical insult, then tissues with high concentrations of nerve endings, such as tendon or spinal cord, are candidate structures. The spinal cord with high nerve density and much lipid insulating axons and a high blood flow is a good environment for bubble formation and growth as well as an obvious site for mechanical insult.

VGE

Sound reflected off a moving boundary undergoes a shift in acoustic frequency, the so-called Doppler shift. The shift is directly proportional to the speed of the moving surface (component in the direction of sound propagation) and the acoustic frequency of the wave and inversely proportional to the sound speed. Acoustic signals in the megahertz range, termed ultrasound, have been directed at moving blood in the pulmonary artery, where blood flow is fastest (near 20 cm/sec), with resulting Doppler shifts, in the form of audible chirps, snaps, whistle, and pops, noted and recorded. Sounds heard in divers have been ascribed to VGE as all venous blood passes through the pulmonary artery. In vitro simulations have established minimum bubble detection size as a function of blood velocity. Coalesced lipids, platelet aggregates and agglutinated red blood cells formed during decompression also pass through the pulmonary circulation, but are less reflective than bubbles, and are usually smaller. Bubbles with radii in the 20 micron range represent the smallest dectable by Doppler using signals of a few megahertz.

As blood constitutes no more than 9% of the total body capacity for dissolved gas, the volume of the venous circulation cannot account for the amount of gas detected as VGE. VGE are not the direct cause of bends per se, unless they block the pulmonary circulation, or pass through the pulmonary filters and enter the arterial system to lodge in critical sites. The likely culprits are bubbles forming in fatty tissues surrounding nerves or in poorly perfused tissues such as tendons. Intravascular bubbles probably first form at extravascular sites. According to Hills[27] electron micrographs have shown bubbles breaking into capillary walls from adjacent lipid tissue beds in mice. The Lambertsen[28] studies of vascular disruption, subcutaneous bruising and venous emboli point to bubble formation in tissues as the culprit. Fatty tissue, possessing few nerve endings, is thought to be an extravascular site of bubble formations.

No-stop limits

Ultrasound techniques for monitoring moving gas emboli in the pulmonary circulation are popular today. Silent bubbles, a term applied to the VGE detected in sheep undergoing bends-free USN table decompressions by Spencer and Campbell,[18] were a first indication that asymptomatic free gas was present in blood, even under bounce dive loadings. Similar results were reported by Walder, Evans and Hempleman.[19] After observing and contrasting VGE counts for various no-stop exposures at depth, Spencer[1] suggested that no-stop limits be reduced below the USN (Workman) table limits. These shorter times produced a 20% drop in VGE counts compared to the USN limits. The newer no-stop limits, t, satisfy a reduced Hempleman relationship,[20,21] that is, $dt^{1/2} \leq 465$ ft min,$^{1/2}$ where d is depth.

TABLE 1

COMPARATIVE NO-STOP TIME LIMITS IN MINUTES

Depth		Workman	Spencer	Bühlmann	Wienke-Yount and Hoffman
msw	fsw				
9	30		225	290	250
12	40	200	135	125	130
15	50	100	75	75	73
18	60	60	50	54	52
21	70	50	40	38	39
24	80	40	30	26	27
27	90	30	25	22	22
30	100	25	20	20	18
33	110	20	15	17	15
36	120	15	10	15	12
39	130	10	5	11	9

Table 1 compares no-stop time limits according to the Workman,[22] and more recent Spencer,[1] Bühlmann,[23] and Wienke-Yount/Hoffman[24,25] algorithms. Further reduction in time limits would seem to increase safety. Limits much below the Spencer, Bühlmann and Wienke-Yount-Hoffman times would restrict repetitive diving, but at the expence of bounce diving.

Statistics gathered by Gilliam[26] suggest that divers using conservative time limits (Bühlmann based diving computer) have compiled an enviable track record, with an incidence of decompression sickness below 0.01% in combined table and meter usage. Many regard such an incidence rate as acceptable.

Another way to restrict repetitive and multi-day diving, suggested by bubble models employing the critical phase volume trigger point, is to reduce the permissible supersaturation tensions on successive dives. This does not restrict no-stop time limits for single bounce dives. The permissible, or critical, tensions are the maximum dissolved gas partial pressures allowed in each tissue compartment and the critical phase volume is the maximum allowable separated gas volume present in all the compartments. The reduced gradient bubble model[24] (RGBM) is one such dual phase model. It systematically reduces critical tensions on repetitive dives by constraining both dissolved and free phase gas build-up.

Table 2 lists the corresponding maximum (critical) surfacing tensions (M_0) for four algorithms. Three, the Workman, Spencer and Bhülmann, have fixed Haldane-model values. The fourth is the variable bubble model (RGBM). The critical tensions in the latter three algorithms are smaller, by some 0.3-1.2 msw (1-4 ft), than the Workman (USN) values, effectively shortening the no-stop time limits a group, or two, compared with the USN tables.

The numbers of VGE detected with ultrasound Doppler techniques can be correlated with no-stop limits and the bubble free limit can then used to fine tune the critical tension matrix for select exposure ranges. However fundamental issues are not necessarily resolved by VGE measurements.

What has not been established is the link between VGE, possible micronuclei and bubbles in critical tissues. Any such correlations of VGE with tissue micronulcei would unquestionably require considerable first-hand knowledge of nuclei size distributions, sites and tissue thermodynamic properties. Recent Doppler studies and correlations by Powell and Rogers,[29] Eckenhoff,[30] and Sawatzky and Nishi[21] do hint that the variability in gas phase formation, is probably less than the variability in symptom generation.

Whatever the origins of VGE, procedures and protocols which reduce gas phases anywhere in the body deserve attention, on the assumption that venous bubbles are a reflection of tissues bubbling. The moving Doppler bubble may not be the bends bubble, but perhaps the difference may only be its site. The numbers of VGE may reflect the state of critical tissues where decompression sickness does occur. Studies based on Doppler detection of VGE are still the only viable means of monitoring free gas phases in the body.

Ascent rates and stops

The effects of slower ascent rates and safety stops, in the context of dissolved gas models, are consistent with bubble mechanics. Both reduce bubble growth rate and bubble formation because of greater off-gassing at the end of the dive. That is a strong endorsement for the practice. Some regard slower ascent rates, safety stops and increased bubble off-gassing pressures as treatment for bubbles, par-

TABLE 2

COMPARATIVE SURFACING CRITICAL TENSIONS (M_o)

Half-time minutes	Workman Mo (fsw)	Spencer M_o (fsw)	Buhlmann M_o (fsw)	Wienke-Yount and Hoffman M_o (fsw)
5	104	100	102	100-70
10	88	84	82	81-60
20	72	68	65	67-57
40	58	53	56	57-49
80	52	51	50	51-46
120	51	49	48	48-45

ticularly near the surface where ambient pressure reduction enhances bubble growth. Gas nucleation theory and experiments show that on any given dive (compression-decompression), families of micronuclei, larger than a critical (minimum) size, are excited into bubble growth, so one must pay attention to free phase (bubble) development throughout the dive. Experiments and calculations suggest that slow ascent rates and shallow, short stops not only reduce bubble build-up, but also reduce dissolved gas in faster tissues. Reducing fast tissue dissolved gas is important for deeper diving. The reasons are rooted in nucleation and bubble mechanics, but some empirical diving practices deserve attention before we turn to illustrative phase model calculations.

Diving practices

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

Pearling fleets, operating in the deep tidal waters off northern Australia, employed Okinawan hard hat divers who regularly dived to depths of 55 m (180 ft) for as long as one hour, twice a day, six days per week and for ten months a year. Driven by economics and not by science, these divers developed decompression schedules empirically. As reported by LeMessurier and Hills,[32] deeper decompression stops, but shorter decompression times than required by Haldane theory, were characteristics of their profiles. Such profiles are entirely consistent with minimizing bubble growth and the excitation of nuclei. Being pulled up by hand they had slow ascent rates. There was a high incidence of decompression sickness, but less than would have been expected. Years later Dr Carl Edmonds, an Australian, devised a simple, but very effective, in-water recompression procedure for use in isolated places. The diver is taken back down to 9 m (30 ft) on oxygen for a minimum 30 minutes in mild cases, or longer in severe cases, and decompressed at 1 m every 3 minutes. The increased pressure helps to compress bubbles, while breathing pure oxygen maximizes inert gas washout (elimination).

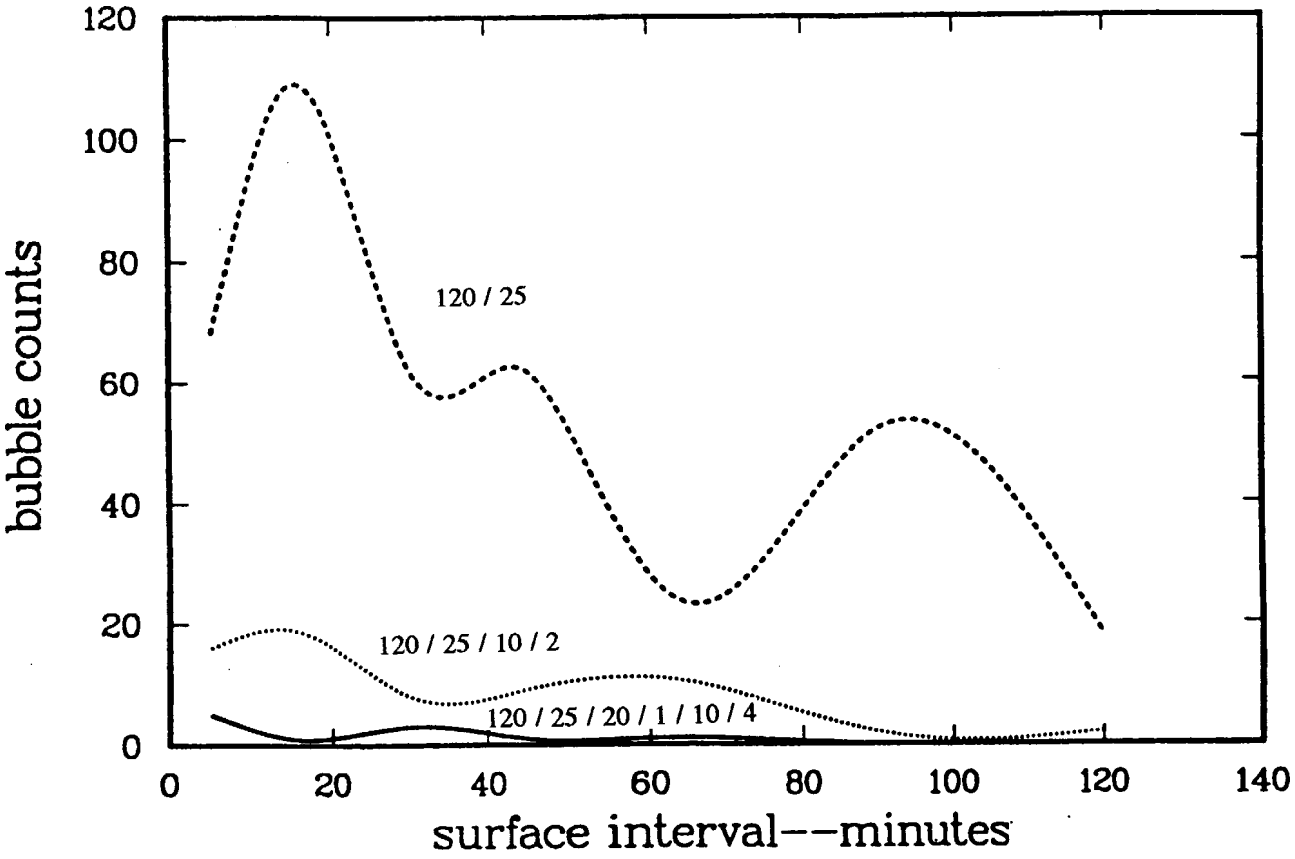
Somewhat similar schedules have evolved in Hawaii, among diving fishermen, according to Farm, Hayashi and Beckman.[33] Harvesting the oceans for food and profit, Hawaiian divers make between 8 and 12 dives a day to depths beyond 105 m (350 ft). Profit incentives induce divers to take risks by exceeding the bottom time in conventional tables. Three repetitive dives are usually necessary to net a school of fish. Consistent with bubble and necleation theory, these divers make their deep dive first, followed by shallower excursions. A typical series might start with a dive to 66 m (220 ft), followed by 2 dives to 36 m (120 ft) and culminate in 3 or 4 more excursions to less than 18 m (60 ft). Often, very short or zero surface intervals are clocked between dives. Such profiles are incompatible with Haldane tables, but, with proper reckoning of bubble and phase mechanics, appear possible. With ascending profiles and suitable application of pressure, gas seed excitation and any bubble growth are constrained within the body's capacity to eliminate free and dissolved gas phases. In a broad sense, the final shallow dives have been tagged as prolonged safety stops and the effectiveness of these procedures has been substantiated in vivo (dogs) by Kunkle and Beckman.[13] If the diver develops decompression sickness immediate in water recompression, using air, is undertaken.

Wet and dry tests

While the above practices developed by trial-and-error, VGE measurements, performed off Catalina by Pilmanis[3] on divers making shallow safety stops, fall into a

FIGURE 4

REDUCTION IN DOPPLER BUBBLE COUNTS FOLLOWING SAFETY STOPS



Safety stops have considerable impact on Doppler sounded VGE measurements, according to Pilmanis. Following a dive to 30 m (100 fsw) for 25 minutes, the top curve registers VGE counts over increasing surface time. The lower two curves depict the count after a brief stop for 2 minutes at 3 m (10 fsw), and then 1 minute at 6 m (20 fsw) followed by 4 minutes at 3 m (10 fsw). Reductions by factors of 4-6 are apparent. Whether VGE correlate with susceptibility to DCS or not, bubble reduction in the pulmonary circulation is impressive with shallow safety stops.

more scientific category. Bubble counts following bounce exposures near 30 m (100 ft), with and without stops in the 3-6 m (10-20 ft) range, showed marked reductions (factors of 4 to 5) in VGE when stops were made (Figure 4). If, as some suggest, VGE in bounce diving correlate with bubbles in sites such as tendons and ligaments, then safety stops probably minimize bubble growth in such extravascular locations. In these tests, the sample population was small, but similar findings were also made by Neuman, Hall and Linaweaver.[4]

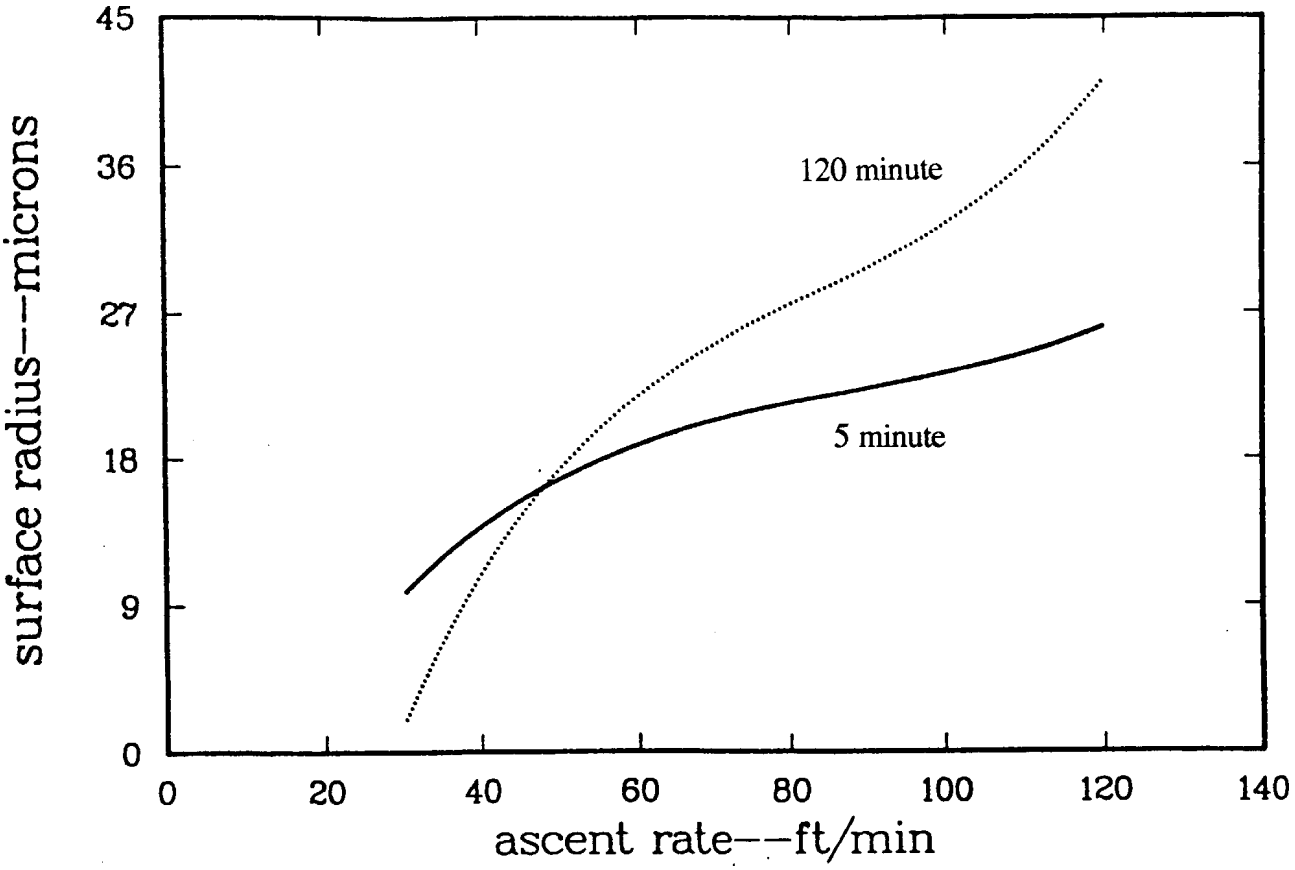
Smith and Stayton,[2] in goat studies, have shown that the incidence of precordial bubbles was greatly reduced when ascent rates were cut from 18 m (60 ft)/min to 9 m (30 ft)/min. Across a variety of decompression profiles, venous bubbles were greatly reduced by slower ascent rates and deeper initial decompression stops than are required by the USN tables. Venous bubbles eliminated during short, deeper stops probably originate in fast tissues. Eliminating these bubbles early in the decompression would allow more slowly exchanging tissues to desaturate safely, while also minimizing the number of arterial emboli possibly remaining after intracardiac shunting, or transpulmonary escape of VGE.

Phase calculations

Theoretically, growth minimization and free phase elimination also recommend slow ascents. Figure 5 plots surfacing radius of an initially small bubble (r = 0.36 microns), held in both fast (5 minute) and slow (120 minute) saturated compartments at a depth of 36 m (120 ft), as a function of constant ascent rate, employing a bubble growth

FIGURE 5

BUBBLE GROWTH WITH VARYING ASCENT RATE



The rate at which bubbles grow on ascent depends on their size and surface tension and the average difference between tissue tension and ambient pressure. For bubbles larger than a certain critical (cutoff or minimum) radius, faster ascents in the presence of elevated gas tensions in surrounding tissue sites tend to support growth, because average ambient pressure, P, is lessened by fast ascent. Increasing ambient pressure always tends to restrict simple bubble growth, since internal bubble pressure is always greater than ambient pressure by an amount, $2\gamma/r$. In this calculation, $2\gamma/r = 8.3$ fsw/micron and unit solubility, concentration and diffusivity employed for simplicity. One notes that the growth rate in the 5 minute compartment is less than in the 120 minute compartment. The faster compartment off-gases more rapidly during any ascent, presenting a lower average tension and weaker diffusion gradient for growth.

equation. The results plotted are also typical for actual bounce, multi-level and repetitive diving profiles and show growth minimization with slow ascent dueincreased average ambient pressure.

Using tissue bubble growth equations, Gernhardt, Lambertsen, Miller and Hopkins[34] have correlated bubble sizes with statistical risk of decompression sickness. One result of that analysis is a risk curve which increases with surfacing bubble radius, pointing to the efficacy of slow ascent rates and safety stops, which reduce surfacing bubble radii (Figure 5).

Discussions at the American Academy of Underwater Sciences Ascent Workshop,[9] suggested discretionary safety stops for 2-4 minutes in the 3-6 m (10-20 ft) zone. Supporting calculations, recorded by Wienke[35] and summarized in Table 3, support the bases of the suggestions. Relative changes in three computed trigger points,[21] tissue tension, separated phase volume and bubble radius, are listed for six compartment following a bounce dive to 36 m (120 ft) for 12 minutes, with and without a safety stop at 4.5 m (15 ft) for 3 minutes.

Stop procedures markedly restrict bubble and phase volume growth and dissolved gas build-up in the faster tissue compartments, while only creating insignificant dissolved gas build-up in the slow tissues. The reduction in

TABLE 3

RELATIVE CHANGES IN CRITICAL PARAMETERS AFTER SAFETY STOP

Tissue half-time	Tissue tension relative change	Critical volume relative change	Bubble radius relative change
5	-12%	-34%	-68%
10	-11%	-24%	-39%
20	-6%	-11%	-24%
40	-2%	-8%	-18%
80	1%	3%	-2%
120	2%	4%	1%

growth parameters far outstrips any dissolved gas build-up in slow compartments and faster compartments naturally eliminate dissolved gases and bubbles during the stop, which is important for deeper diving when the gas loads are greater. The calculations in Table 3 are illustrative of a broad category of no-decompression bounce and repetitive diving that has been analyzed.

Safety stop time can be added to bottom time for additional conservatism, but the effect of not doing so is small. A stop at 4.5 m (15 ft) for 2 minutes is roughly equivalent to more than halving the standard ascent rate at depths in excess of 36 m (120 ft). Procedures such as this, as well as conservative no-stop time limits, appear beneficial in multi-day, multi-level and repetitive diving. A safety stop near 4.5 m (15 ft) is easier than at 3 m (10 ft) in adverse water conditions, such as surge and surface disturbances. Slower ascent rates afford additional advantages, but safety stops in the 2-4 minute range are easier and theoretically more efficient. Ascent rates slower than 18 m (60 ft)/min and safety stops in the 6-9 m (10-20 ft) zone are becoming routine for recreational and scientific divers.

Generally, bubble growth and excitation are compounded at altitude because of reduced pressure. Bubbles grow faster as they get bigger and as ambient pressure drops. With decreased ambient pressure, bubbles will also expand in accordance with Boyle's Law. Bigger bubbles are not as constricted by Laplacian film tension, while reduced ambient pressure supports a faster rate of tissue gas diffusion into the bubble itself. At altitude, bubble mechanics theoretically exacerbate decompression risk.

The point to be made here is simple. Increased off-gassing pressures are likely to reduce bubble growth rates dramatically in shallow zones, while increasing dissolved gas build-up in the slowest compartments minimally. Fast compartments also off-load gas and bubbles during slow ascents and safety stops, important for deep diving. Slow ascent rates and stops are always advisable, particularly at altitude and in multi-level and multi-day diving.

Summary

A first-principles decompression theory is not available at present. One suspects shortcomings in present approaches and wonders how to enhance their effective implementation.

In the case of the Haldane (dissolved gas) algorithm, the basis of virtually all diving tables and meters up to 1983, there are two problem areas, free phase (bubble) dynamics and bends trigger points. Tissue tensions are not the same as gas pressures in bubbles and elimination gradients for dissolved phases are not the same as gradients for bubbles. With increased exposure, one observes lower tolerance levels to bubbles. With successively deeper profiles, one suspects that there is excitation of greater numbers of gas nuclei into growth, exceeding the body's capacity for bubble elimination.

These considerations may explain the slightly higher bends incidence, observed by hyperbaric specialists, for divers doing multi-day, repetitive and multi-level excursions, in that order of decreasing risk. Bounce diving is relatively free of risk these days, especially when diving within algorithms employing conservative time limits which restrict phase separation. The presence of increasing proportions of gas as bubbles alters and invalidates tables based on dissolved gas models.

Such changes are best assessed by nucleation and bubble models. Safety stops and slower ascent rates correlate in principle with bubble models and tests as effective procedures, restricting bubble growth. No-stop time limit reductions appear prudent, based on Doppler bubble counting experiments. However, further reductions in no-stop time limits, beyond those in current use, do not appear warranted, considering the low incidence of decompression sickness, less than 0.01% in populations employing recent tables and meters with conservative limits.

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Bruce Wienke PhD, is Director of the Computational Testbed For Industry, Los Alamos National Laboratory, Los Alamos, New Mexico 87545, U.S.A.

THE WORLD AS IT IS

In this new feature we print original papers which are neither scientific articles nor editorials but which are considered to be sufficently important or interesting that they should be brought to the attention of members.

IS THE AMA REALLY INTERESTED IN PREVENTIVE MEDICINE ?

These comments are provoked by a letter from Dr Bob Thomas, who was once the Editor of the SPUMS Newsletter and OIC the Royal Australian Navy School of Underwater Medicine, and the letter which he received from the Federal AMA, both of which appear below.

Diving Medical Centre
132 Yallambee Road
Brisbane
Queensland 4074

11th September, 1992

Dear Editor,

I though you would be interested to read the letter enclosed. Obviously the AMA is still playing games and believes that all doctors can be everything to everyone.

I feel that this letter should be passed to the SPUMS Committee for their information. It raises great concern about the suitability of any SPUMS member not appointed by the Executive (and especially one who espouses an AMA dictum opposite to the ideas of SPUMS at Standards Australia Committee meetings, and yet, supposedly, is himself in agreement with SPUMS views) sitting on Standards Committees concerning diving. How can two opposing hats be worn?

In fact, I can see no need whatsoever for the necessity of any AMA representative on such a diving Committee when the AMA represents no collective body of knowledge concerning diving.

I feel that all SPUMS members should be made aware of this AMA stupidity. Can you please publish this letter in the Journal?

Bob Thomas

Australian Medical Association Limited
42 Maquarie Street
ACT 2600

1st September, 1992

Dear Dr Thomas,

I am replying on behalf of the AMA to your letter of August 9, 1992 with which you enclosed an updated list of medical practitioners who have successfully completed a course of instruction conducted by the Diving Medical Centre (Australia) on diving medical examinations.

As you may know from previous correspondence, the AMA has not accepted that a need exists for such certification in the case of doctors who perform fitness examinations for candidates who wish only to undertake sports/recreational scuba diving. While such certification appears desirable in the longer term, the AMA notes the precedent of no <u>required</u> training for designated medical examiners who complete aircrew licensing medicals, even those for airline transport pilots. The AMA is aware of your views, also those of SPUMS, and is represented on the relevant SAA Committee by Dr I.L.Millar. The Association will keep this matter under review.

P.S. Wilkins
Assistant Secretary General
(Health Services)

The SPUMS position on diving medicals is that they should be compulsory before starting to use compressed air underwater and that they should be done by doctors with training in underwater medicine.

This attitude comes from bitter experience of the inadequacy of many medicals done by doctors who knew little, or more probably nothing, about diving medicine and passed people as fit to dive when they should have been told that it was extremely dangerous to go diving. Some of these people died as a result of this failure to assess them properly.