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Mathematical Modelling of Tissue Bubble Dynamics During Decompression

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It is well accepted that bubbles, through some mechanism, are the cause of marginal symptoms of decompression sickness (DCS). For this reason a theoretical model of extravascular gas bubble dynamics has been derived to aid in developing and evaluating decompression procedures. The model has been developed and refined over a number of years and has been aimed at both diving and aerospace decompressions.

The model assumes that gas nuclei exist, or through one or more processes form, in extravascular tissues during decompression. The model then describes the subsequent growth and resolution dynamics of a gas bubble during the course of decompression.

The model accounts for the diffusion of inert gas across the tissue/bubble interface, gas solubility and diffusivity, surface tension, tissue elasticity and a perfusion-limited inert gas exchange with the tissue. The model can account for multiple inert gas breathing mixtures, and variable ascent and descent rates. A graphic illustration of the model and a list of the assumptions and features is shown in Fig. 1.

Basic Assumptions

- Bubbles are the cause of marginal symptoms of DCS
- Gas nuclei exist or form in extravascular tissues during decompression
- The gas exchange between the extravascular bubble and the surrounding tissue is limited by diffusion
- The gas exchange between the blood and tissues is limited by the blood perfusion to the tissues

Advances in Underwater Technology, Ocean Science and Offshore Engineering, Volume 14: Submersible Technology

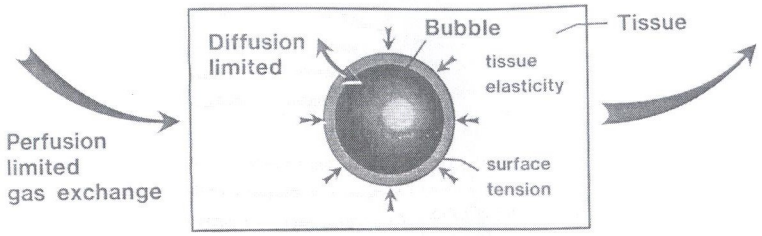

Fig. 1 Bubble dynamics model.

The model accounts for:

- Gas solubility and diffusivity
- The surface tension acting on the tissue/bubble interface
- The tissue elastic forces that would oppose bubble growth
- Multiple and sequential inert gas breathing mixtures
- Oxygen breathing
- Variable ascent and descent rates

The model does not account for the statistical nature of decompression sickness, or individual differences in the ability to withstand gas emboli. For this reason it is not thought that any critical theoretical bubble size would be predicted by the model that would always cause, or conversely always prevent, decompression sickness. Instead it is thought that the model could be used as one tool, in conjunction with empirical data and operational experience, to evaluate the relative severity of a variety of complex diving or aerospace decompressions.

The most basic assumption of the model is that gas nuclei exist or form in extravascular tissues during decompression. With this assumption, instead of trying to model the complex and poorly understood processes by which bubble nuclei form in tissues, their existence during decompression is assumed and used as a starting point for modelling the bubble growth and resolution phase.

This is a conservative and reasonable assumption supported by a wealth of data which suggest that bubble growth occurs at pressure differentials much less than the tensile strength of body fluids, thus implying the existence or formation of these bubble nuclei.

STANDARD DECOMPRESSION MODELS

Most standard diving and aerospace decompression models are based on modified Haldanian decompression concepts and incorporate a perfusion-limited inert gas exchange between the blood and the tissues. This assumption is common with the bubble dynamics model and allows the bubble model to be correlated with the vast database of information available from procedures developed with the Haldanian models.

The basic Haldanian model has been modified extensively over the years through a largely empirical process and has been adapted to both diving and aerospace applications. The primary difference between standard diving and aerospace models is not in the inert gas exchange, but in the criteria for a permissible decompression.

The commonly used diving models typically use a critical pressure difference, or supersaturation, between the calculated tissue tension and the ambient pressure.

Aerospace decompression models utilize a pressure reduction ratio, between the calculated tissue tension and the space-suit pressure, referred to as an *R*-value.

There is generally little direct correlation between the diving and aerospace models, because the diving models typically allow supersaturations of greater than one atmosphere for certain tissue compartments. This is clearly inconsistent with aerospace decompressions where supersaturations of much less than one atmosphere in all tissue compartments are necessary to protect against DCS. This is the reason the aerospace models rely on the pressure reduction ratios.

By modelling a gas bubble within a perfusion-limited tissue, the bubble dynamics model describes the bubble volume response to any change in pressure. For this reason, the model should be more continuous between diving and aerospace decompressions, allowing empirical data from both high pressure and low pressure environments to be analysed and correlated.

DERIVATION OF THE MODEL

For an extravascular stationary bubble, the flux of gas into the bubble is driven by the pressure gradient between the tissue and the bubble. The bubble then readjusts its pressure and volume to come to equilibrium. Neglecting convective effects, the flux of gas is given by Fick's law:

$$J = \frac{-\alpha D\,(P_T - P_B)}{RT\,h} \tag{1}$$

Where

J = Molar flux of gas
D = Diffusivity
h = Effective thickness of the diffusion barrier between the bubble and the tissue
P_B = Pressure of gas in the bubble
P_T = Tension of gas in the tissue
R = Ideal gas constant
T = Temperature
α = Solubility of gas in the tissues

Multiplying equation (1) by the area of the interface will give the number of moles of gas per unit time diffusing into the bubble:

$$J \cdot A = \frac{\alpha A D}{RTh}(P_B - P_T) = \frac{dn}{dt} \tag{2}$$

If the separated gas is assumed to be an ideal gas, then the equation of state for an ideal gas equation can be solved for dn/dt, the number of moles of gas diffusing into the bubble with time. This expression can be equated to the

equation (2) to yield an equation which describes the flux of gas into the bubble and the bubbles pressure/volume response (assuming constant temperature):

$$\frac{-\alpha AD}{h}(P_B - P_T) = P_B \frac{dV}{dt} + V \frac{dP_B}{dt} \quad (3)$$

Where P_T = the tension of inert gas in the tissues and P_B = the pressure of inert gas in the bubble.

The pressure of gas in the bubble will be equal to the sum of the pressure forces acting on the bubble. The three dominant forces would be:

- **Hydrostatic pressure** The pressure of surrounding environment.
- **Surface tension** The pressure due to the surface tension acting on the tissue/bubble interface would be equal to $2\delta/R$ where δ is the surface tension in dynes/cm and R is the bubble radius.
- **Tissue deformation** The pressure due to tissue deformation which will be equal to $\frac{4}{3}\pi R^3 H$, where H is the bulk modulus of the tissue expressed as $\Delta P/\Delta V$ (3).

Bubble expansion in tissues of high bulk modulus will result in greater increases in intra-bubble gas pressure than in tissues of lower bulk modulus. The increase in gas pressure within the bubble will tend to drive the gas out of the bubble, resulting in a smaller bubble radius.

The pressure of gas in the tissue P_T, can be calculated using equations derived from applying a mass balance to a perfusion-limited tissue. The result of this derivation is a form of the familiar equation for a perfusion-limited gas exchange (4)

$$P_{T_A} = P_T(0) + \{x(P_0 - vT) - P_T(0)\} \times (1 - e^{-\left(\dot{Q}\frac{\alpha_b}{\alpha_t}\right)t}) \quad (4)$$

Where,

P_T = Inert gas tension in the tissue
P_0 = Initial hydrostatic pressure
x = Mole fraction of inspired inert gas
$\dot{Q}$ = Blood perfusion
α_b = Gas solubility in blood
α_t = Gas solubility in tissue
t = Time
v = Ascent rate

The exponent in equation (4) can be related to the tissue half-time by:

$$\dot{Q}\frac{\alpha_b}{\alpha_t} = \frac{0.693}{\text{half-time}}$$

Where the half-time is the time for a tissue to reach half saturation with a given pressure.

The expressions for P_B and P_T are substituted into equation (3), and the resulting equation is solved for bubble radius as a function of time, using computer numerical solution techniques (second order polynomial extrapolation).

The equations can be modified to account for multiple gases by defining the total tissue gas pressure to be equal to the sum of the component gases. An effective gas diffusivity is also defined based on the component gases' diffusivity and concentration.

By varying the ambient pressure, breathing mixtures and ascent rates, it is possible to simulate the bubble dynamics for any complex decompression protocol. The computer solution is designed so that the final bubble radius from one exposure becomes the initial value for the next exposure. In this manner the results of decompressions, recompressions and gas switches can be simulated.

THEORETICAL SIMULATIONS AND DISCUSSION

The solution to the bubble radius equation is a plot of the bubble radius against time for any specified decompression protocol. The simulations discussed in this section are based on the theoretical model and *should not be construed as conclusive recommendations regarding the validity of any specific decompression protocol or collectively about any group of decompression tables.*

The simulations shown in Fig. 2 illustrate the theoretical bubble growth characteristics associated with a pressure reduction of one atmosphere. (From two to one atmosphere and from one to zero atmosphere.) The pressure reduction from one to zero atmosphere shows much greater bubble growth than the same pressure differential from two to one atmospheres.

Although this behaviour would be expected for an ideal gas undergoing an isothermal polytropic process, it is significant that the bubble model is able to distinguish between these two exposures. Standard diving models, based strictly on supersaturation concepts, would not predict any differences.

In the context of the bubble model, the theoretical tissue bubble would continue to grow until the tissues were completely desaturated with inert gas,

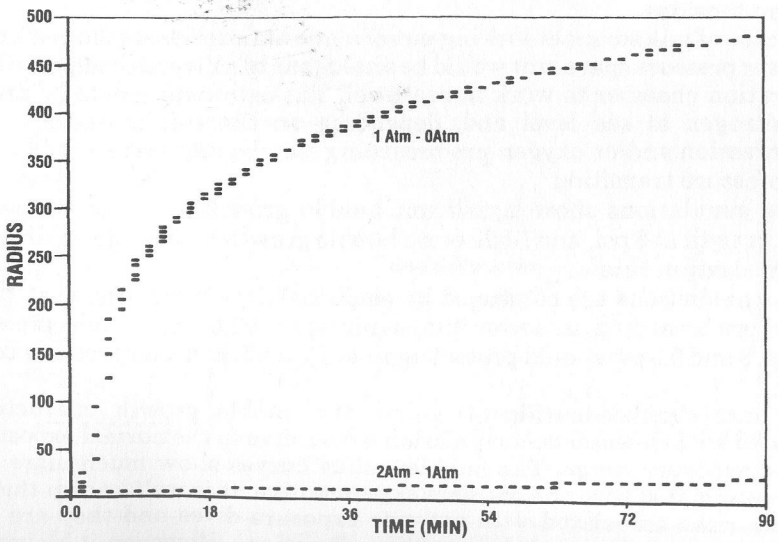

Fig. 2 Theoretical bubble growth associated with pressure reduction of one atmosphere.

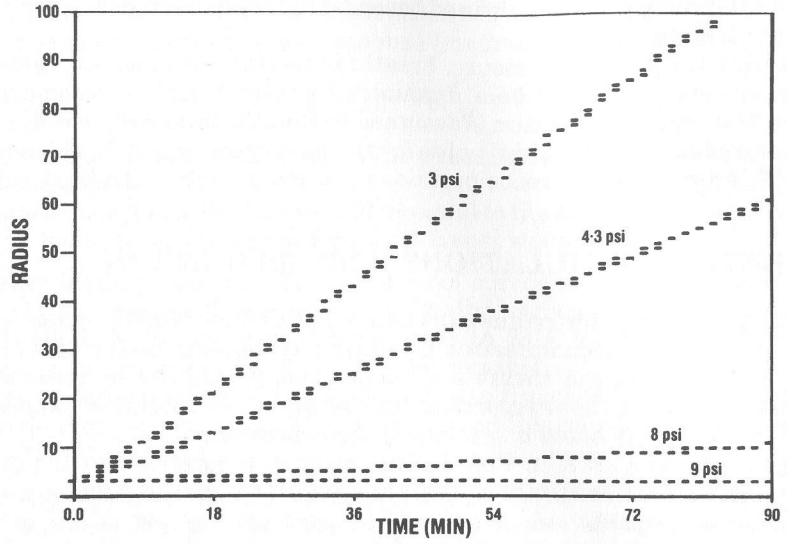

Fig. 3 Theoretical bubble growth for EVA excursions from one atmosphere to 3, 4.3, 8 and 9 psi with no O_2 prebreathing.

at which point the bubble would begin to decay under the resolving forces of tissue elasticity and surface tension, as illustrated in Fig. 2.

The next series of simulations (Fig. 3) were aimed at identifying the space-suit pressure (normoxic breathing mixture), which would protect against DCS, without the astronauts having to pre-breathe oxygen in order to lower tissue nitrogen tensions.

The case of an astronaut locking out of a one atmosphere space craft to work in a lower pressure space-suit would be analogous to a diver decompressing from a saturation chamber to work at sea level. The astronaut would be saturated with nitrogen at sea level and, depending on the suit pressure, a staged decompression and/or oxygen pre-breathing may be required to safely make a given pressure transition.

These simulations show significant bubble growth at 3 and 4.3 psi, some bubble growth at 8 psi, and little or no bubble growth at 9 psi, depending on the depressurization rate.

These predictions are confirmed by empirical data indicating that, without oxygen pre-breathing to lower tissue nitrogen tensions, a suit pressure of between 8 and 9.5 psi should protect against DCS when decompressing from sea level.

The next simulation (Fig. 4) shows the bubble growth characteristics associated with in-water decompression from a dive in the normal exposure and extreme exposure range. The bubble radius curves show much more bubble growth associated with the extreme exposure dive. It is well known that there are more risks associated with extreme exposure dives and they are almost universally not recommended for routine operations. However, it is significant that the bubble model is able to distinguish the relative severity of these dives, whereas an extrapolation of standard computerized diving models would not.

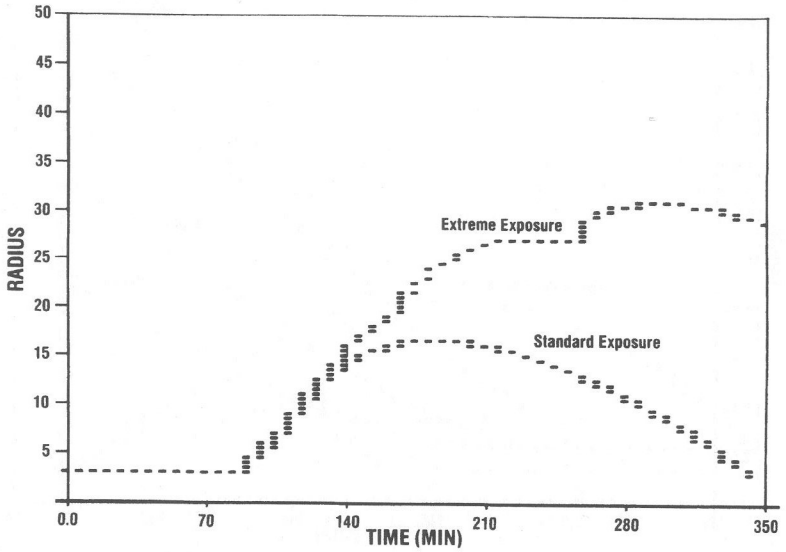

Fig. 4 Comparison of standard versus extreme exposure (in water decompression).

The next simulation (Fig. 5) shows the bubble growth characteristics associated with an air dive followed by surface decompression on oxygen. The two curves correspond to a modified and an unmodified procedure. Both curves show a period of bubble growth during the surface interval, followed by a period of bubble resolution during oxygen breathing under pressure in the chamber.

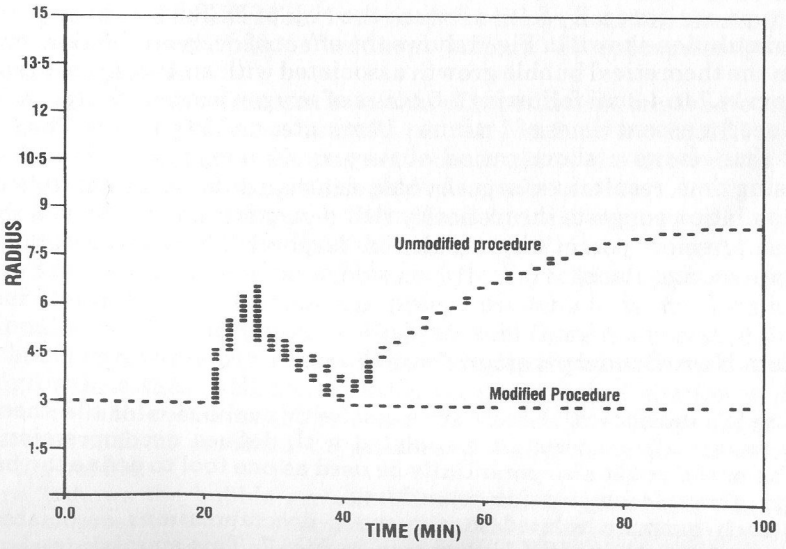

Fig. 5 Surface decompression on oxygen.

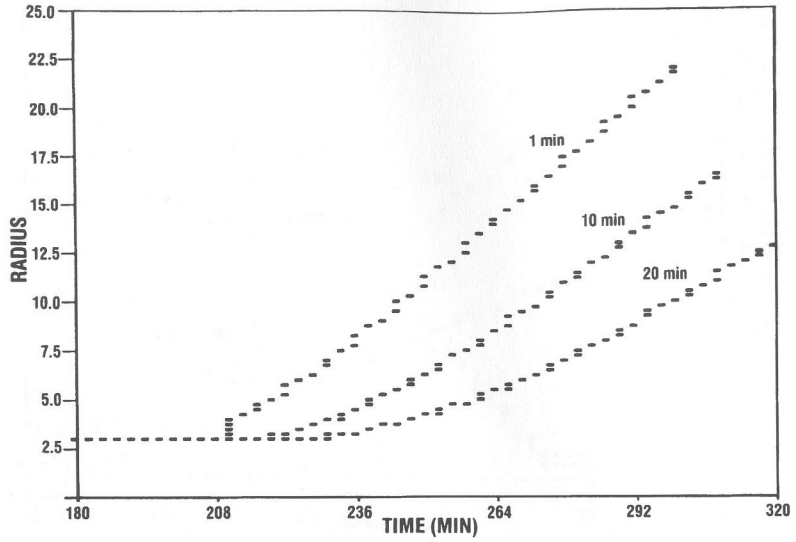

Fig. 6 Effect of depressurization rate for 1, 10, and 20 min ascents (14.7–4.3 psi) following 3.5 h of O_2 breathing.

For the unmodified procedure, the bubble is not completely resolved in the chamber and continues to grow after the ascent to the surface, reaching a maximum size in the time period generally associated with the onset of DCS symptoms, if they were to occur. For the modified procedure, which incorporates changes to the oxygen breathing and pressure protocol, the bubble is completely resolved during the chamber decompression period. Modifications of this type are generally felt to reduce the risk of DCS.

The simulation shown in Fig. 6 shows the effect of depressurization, or ascent rate, on the theoretical bubble growth associated with an aerospace decompression from 14.7 to 4.3 psi following 3.5 hours of oxygen pre-breathing. The curves shown are for ascent times of 1 minute, 10 minutes and 20 minutes. The 1 minute ascent time shows a short period of very rapid bubble growth which, with increasing time, results in a larger bubble size than does the slower ascent rates. This simulation suggests theoretically that a very fast ascent early in this type of decompression potentially results in larger bubbles much later in the decompression protocol.

Defining New Decompression Procedures

The simulations shown above have dealt with evaluation of the theoretical bubble growth characteristics associated with defined decompression protocols. The model could also potentially be used as one tool to define the basis for optimizing new decompression procedures.

One such example related to aerospace decompressions associated with extravehicular activity (EVA) is shown in Fig. 7. This simulation shows the effect of utilizing intermittent recompressions during the course of the EVA.

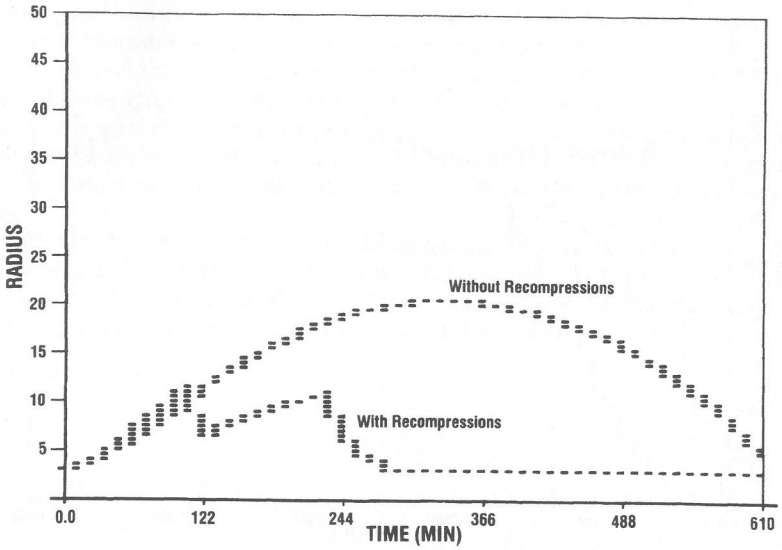

Fig. 7 EVA with and without intermittent recompressions.

The effect of the intermittent recompressions is to interrupt the gradual bubble growth that would normally be associated with this exposure. Since the EVA pressure is lower than the saturation pressure (sea level) and the simulated breathing gas is enriched with oxygen, continual off-gassing occurs during the course of the recompressions. The recompressions then help to resolve the bubble. The net effect is to keep the theoretical bubble smaller and resolve it sooner with only a minimal amount of appropriately chosen recompression periods.

This procedure is very different from that of repetitive diving where repeated exposures to higher pressures of inert gas can result in a cumulative effect, increasing tissue inert gas tensions and consequently the theoretical bubble size which would occur during subsequent decompressions.

This same theoretical concept illustrated for the aerospace case could potentially be applied to saturation diving.

Figure 8 shows the theoretical bubble growth characteristics associated with an older saturation decompression procedure which is no longer used operationally. The bubble dynamics illustrate that there is a period of slow but steady bubble growth early in the deeper stages of the decompression. The bubble growth is then well controlled in the longer and shallower phases; however, the bubble remains at a relatively large size. This is because there are no measures such as enriched oxygen breathing or recompressions to help resolve the bubble.

Figure 9 shows the bubble growth characteristics associated with a saturation decompression from the same depth, but this time incorporating minimal intermittent recompressions coupled with enriched oxygen breathing. The net effect is to control the theoretical bubble growth better.

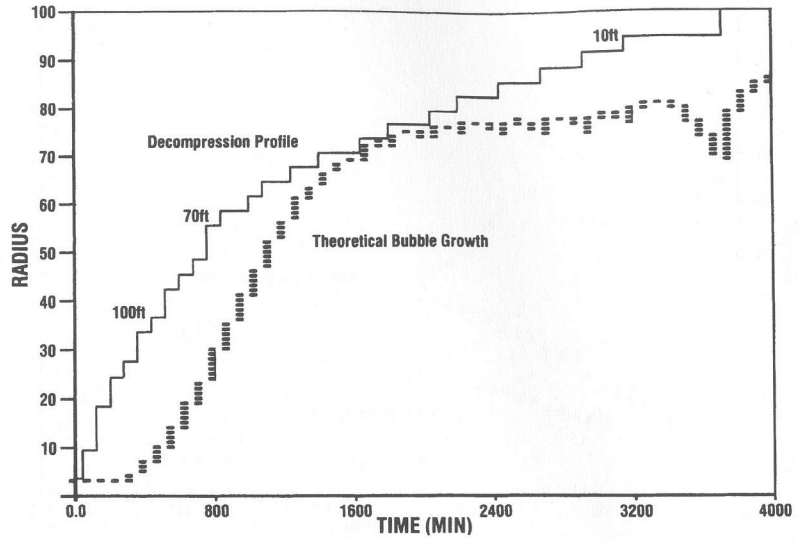

Fig. 8 Theoretical bubble growth: nitrox saturation decompression (schedule not approved for operations).

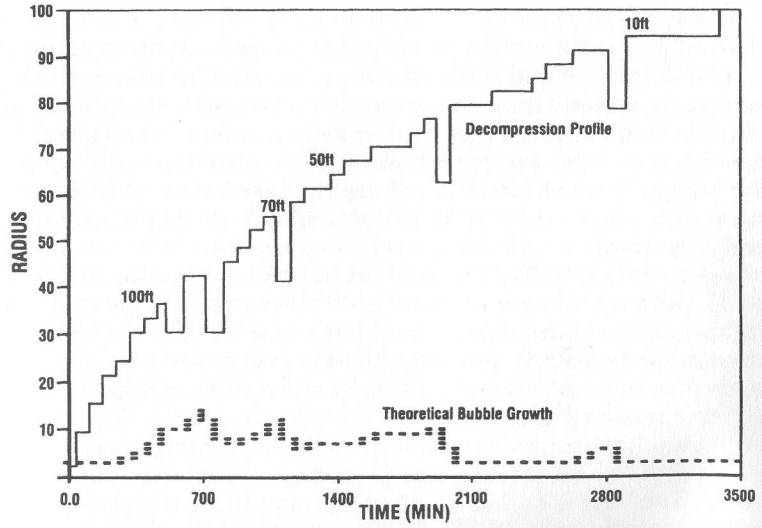

Fig. 9 Theoretical bubble growth: nitrox saturation decompressions (incorporated intermittent decompressions with enriched P_{O_2}).

CONCLUSIONS

The bubble dynamics model demonstrates qualitatively reasonable results which are generally consistent with a wide range of empirical diving and

aerospace decompression data. The model integrates perfusion and diffusion-limited inert gas exchange and gas pressure/volume response into a single time-dependent criterion, which is potentially applicable over a continuous range of decompressions from hyperbaric to hypobaric environments.

Since the model does not account for the statistical nature of decompression sickness or individual differences in the population, it is not thought that a critical bubble size exists that would always cause or prevent decompression sickness.

Instead it is thought the model could be a useful tool to be used in conjunction with standard decompression models and empirical data. In this manner, patterns of bubble growth and resolution could be used as one index to evaluate and develop a wide range of decompression procedures.

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Decompression Sickness in Commercial Air Divers

The Present Status of the Shields/ Lee Report

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At the request of the UK Department of Energy, a survey was made of all commercial air dives carried out in the UK sector of the North Sea during 1982 and 1983, to determine whether or not this form of diving resulted in an unacceptable incidence of Type II decompression sickness (DCS). The report of the survey (Shields and Lee, 1986) was submitted to the Department of Energy in mid-1986, and resulted in a Diving Safety Memorandum (DSM 7/86) which placed restrictions on the permissible bottom time at given depths.

The survey was based on a retrospective analysis of dive logs submitted by the diving contractors. In all, 25 740 man-dive records were examined and analysed under 15 parameters, such as dive depth and time, table depth and time, type of thermal protection, etc. Surface decompression diving accounted for approximately 60% of the dives, no-stop diving for 30%, and in-water decompression for only 10%. The dives using in-water decompression had, in general, a much less severe hyperbaric exposure than those using surfacing decompression.

During the survey period, 79 cases of DCS were recorded; 44 with Type I manifestations, and 35 with Type II (neurological) manifestations. Although the nature of the data made conventional statistical manipulation impossible, an attempt was made to determine the significance of four contributing factors: the severity of the hyperbaric stress; the decompression procedure used; the extent of safety-factoring; and the type of thermal protection used. An index of dive severity (the Decompression Penalty Index) was proposed, based on a notional decompression time required for any given exposure; a dive with a DP Index of less than 30 was considered to be modest, one with a DP Index of between 30 and 60 to be of moderate severity, and one exceeding a DP Index of 60 to be severe.

Of these contributing factors, the most powerful influence was the severity of the hyperbaric stress (that is, increasing depth and/or time of the dive) (Table 1). As dive severity increased, there was a greater incidence of DCS. When this was

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TABLE 1 *DCS related to hyperbaric stress and decompression procedure*

	Decompression penalty index					
	< 30		*30–60*		*> 60*	
	In-water decompression	*Surface decompression*	*In-water decompression*	*Surface decompression*	*In-water decompression*	*Surface decompression*
Number of dives	2 061	7 099	31	6 670	4	1 122
Number of cases of DCS	4	10	1	45	0	18
Incidence	0.19	0.14	3.22	0.67	0	1.60
Number of Type I cases	4	5	1	24	—	10
Number of Type II cases	0	5	0	21	—	1

TABLE 2 *Influence of the severity of hyperbaric stress and type of thermal protection on the incidence and type of DCS (surface decompression dives only)*

	Passive thermal protection			*Hot water thermal protection*		
	DP Index			*DP Index*		
	≤ 30	*31–60*	*> 60*	*≤ 30*	*31–60*	*> 60*
Number of dives	1 514	1 273	442	5 585	5 397	680
Number of cases of DCS	0	9	2	10	36	16
Incidence	0	0.7	0.5	0.2	0.7	2.4
Number of Type I cases	0	7	1	5	17	9
Number of Type II cases	0	2	1	5	19	7

controlled for, there was no obvious difference between the use of in-water stops and surface decompression, although this comparison was only possible on dives of modest severity. Thermal protection also had an effect on DCS, with a higher incidence resulting from the use of actively heated (hot-water) suits. This influence was only apparent, however, on dives of severe hyperbaric stress. Of particular concern was the increasing proportion of the Type II DCS on such dives, where hot-water suits were used (Table 2).

The conclusion from the report was that, although the overall incidence of DCS was commendably low, the total number of dives was such that an unacceptable number of individual cases of neurological DCS were accumulating. A 'no-Type-II' line was drawn on the depth/time plot of all the dives, to the left of which there would be an 'acceptable' incidence of Type II DCS (Fig. 1) and this line was used to determine a recommended bottom time at any depth. These times are the basis of DSM 7/86 (Table 3).

Although there were no cases of Type II DCS resulting from in-water decompression (possibly because 99% of such dives fell to the left of the DSM line in any case) it was decided to apply the restriction to this form of diving as well as to surface decompression diving. This was done because of a reluctance to encourage an increase in the commercial attractiveness of long-in-water-stop diving in the North Sea, with the potential dangers of long cold decompressions and the danger of pressure fluctuations on stops near the surface. It was recognized that these factors could be eliminated by the use of a closed diving bell, and that in these circumstances, the DSM 7 might not be appropriate. Indeed, evidence from other areas (Imbert and Bontoux, 1986) suggests that the use of conventional stage-decompression on long deep dives does not produce the same incidence of Type II DCS as occurred following such dives in the North Sea where surface decompression was used.

The imposition of the DSM was criticized by the diving industry on the grounds that it was based on information which was three years out of date, and

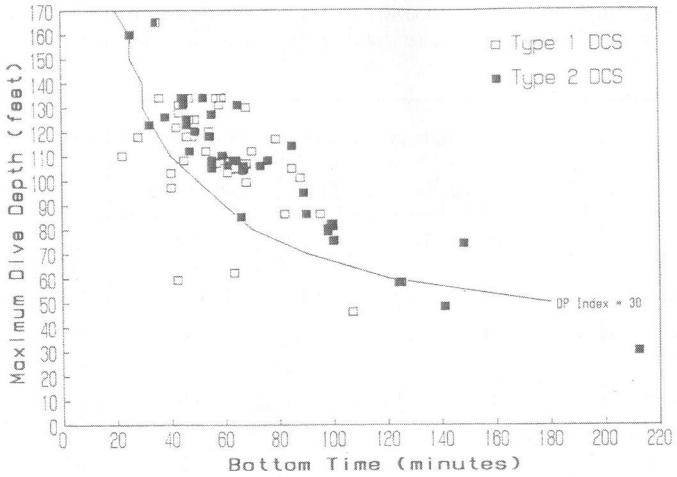

Fig. 1 Depth–time plot of all dives.

TABLE 3 *Maximum bottom times for dives within DP limits stated*

Depth (feet)	*Bottom time (minutes)*	
	DP limit of 30	*DP limit of 20*
40	360[a]	300[a]
50	180[a]	150[a]
60	120	100
70	90	80
80	70	60
90	60	50
100	50	40
110	40	35
120	35	30
130	30	25
140	30	25
150	25	20
160	25	20
170	20	15

[a]Note that these dives might exceed a maximum bottom-time limit.

that diving practices had been modified (and by implication, were producing fewer cases of Type II DCS) since then. The Department of Energy has therefore commissioned a continuation of this study, to bring the data up to date to the time of the DSM (August 1986) and to continue to monitor the effects of the DSM and of any dispensations given in terms of particular decompression schedules, for a further three-year period.

A large part of the information for 1984 and 1985 has now been collected, and a preliminary analysis of the 1984 data has been completed. At the date of preparing this paper, however, we are not at liberty to present these results publicly; the industry through the AODC Council has made strong representation to the Department of Energy that the results of the updated studies should not be revealed until there has been an opportunity for private discussion between the industry and the Department of Energy.

Analysis of data for 1985 will be completed by the end of 1987 and of those for 1986—up to the date of the DSM—shortly afterwards. These will again be discussed privately with the diving industry prior to any public disclosure, but hopefully the complete analysis from 1982 to August 1986, together with the initial results from the current year, will be available by mid-1988.

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