REPORT

Modifications to heliox tables to reduce extent of bubble formation

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SUMMARY

USl was asked to carry out a theoretical study of current heliox exucursion and decompression tables to evaluate the extent of bubble formation and to suggest changes which would reduce decompression bubbles. In order to complete the study quickly only the range of working depths required by the client in the immediate future have been studied and only maximum allowed excursions. Three depths have been considered, 75 msw, 100 msw and 130 msw, and the maximum downward excursion has been considered for each, 25 29 and 33 msw.

The volume of gas forming into bubbles in the brain can be reduced by 50% by slowing the return to the bell to 5.3 minutes (25-75), 6.5 minutes (29-100) and 7.7 minutes (33-130).

If decompression is started within an hour of the excursion the gas in bubbles can grow by up to 500%. A 6 hour hold, with an hour of 1.5 bar oxygen breathing, can almost halve that growth.

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1.0 INTRODUCTION

It has been known since 1997, with the publication of the analysis of data collected during the HADES project, that the incidence of decompression illness (DCI) is most closely related to the number of depth changes experienced by a diver during a saturation exposure (Jacobsen et al 1997).

Any reduction in depth, greater than a certain magnitude for which the normal physiology of the human body gives protection, can be expected to cause decompression bubbles. The magnitude of a safe ascent can vary between individuals, between different tissues of the body and even from time to time within an individual tissue. This means that both the occurrence of bubbles and the occurrence of symptoms have a statistical element. Definition of a safe depth change together with the time course of the change, the decompression profile, should be qualified by the level of risk accepted. It is striking that in commercial diving the level of risk accepted for depth changes following downward excursions during a saturation exposure appears to be very much higher than that found acceptable for decompression from the storage depth. An 8 hour bell run to a depth greater than the storage depth gives an increased body load of inert gas not far different from a saturation exposure to the same depth but, whereas return from saturation exposure may be required to take many hours, return from the excursion is allowed to be completed within 1 or 2 minutes. Even more surprising is the fact that excursions to a shallower depth to undertake work can also be made with the depth change completed in minutes.

Depth changes are a fact of life in all diving and in commercial diving are a necessity. This is not only a matter of commercial consequence; nobody would advocate moving a whole chamber full of divers to a new depth if the job requiring the depth change can be carried out by one or two divers making the change. Procedures should be defined together with the associated level of risk which is considered acceptable. Decompression hits interfere with efficiency; only once a properly evaluated risk assessment has been made does it become possible to maximise efficiency.

Decompression bubbles resulting from excursions are also a fact of life but a certain level can be acceptable. This level should be defined. Once that is done some limits currently in use may be seen to be more cautious than need be and diving companies should be free to accept a greater risk. Some limits will undoubtably prove to generate too many bubbles and diving companies, and their clients, should be discouraged from going to these limits or at least be enabled to understand the increased risks.

Unimed Scientific Limited has been asked to carry out this study with the objective of reducing the risk from excursions during heliox saturation diving.

1.1 BACKGROUND

The requirements for this particular study are restricted to a very limited range of saturation depths and working depths relevant to the needs of the client. Saturation depths ranging from 75 msw to 130 msw are considered and, in the interests of completing the study quickly, only maximum downward excursions have been considered.

The starting point for the work has been taken as the procedures which are currently used and the approach has been to evaluate changes in procedures which are both operationally acceptable and which reduce the amount of gas predicted to form into bubbles.

The study essentially divides itself into two parts because the rapid ascent from the working depth puts at risk different tissues from those at risk during the decompression from saturation. In considering the return from excursions the focus has been set on the formation of gas in the tissues of the brain and the consequential risk of CNS DCI. In consideration of decompression from saturation the focus is on the slower tissues in the body, most notably on the fat. However this does not mean that the two situations are entirely separate and independent. It is generally true that for most divers the long decompression from saturation would be bubble free; that they are not always so would seem to be due to the fact that the bubbles which result from the return to chamber pressure from working pressure are still in the body when the decompression starts.

The technique used is the mathematical model of decompression developed by USL and described in more detail in the Appendix. The model calculates the gas volume which would form into bubbles in each ml of tissue and of blood in equilibrium with the tissue. The brain consists of two types of tissue, so called white matter and grey matter, the white matter having a lower blood flow than the grey and therefore moving inert gas more slowly than the grey tissue. For the purpose of the present study the results for brain bubbles have been presented as a weighted mean of the two tissue types, equal weighting to each type.

The central venous blood is in effect a weighted mean of the blood flow from all of the tissues and as such forms a convenient, though not always very sensitive, indicator of the whole body bubble load. However the central venous blood, as it flows through the pulmonary artery to the heart, is one location at which ultrasonic techniques of bubble evaluation can be used. This makes the model calculation of gas carried in bubbles in the central venous blood a useful indicator of decompression risk.

Results are therefore presented either as the weighted means of the two types of brain tissue or, for the whole body, as the weighted means of the eight compartments into which the body tissues are grouped (see Appendix).

Three depths have been considered, 75 msw, 100 msw and 130 msw, and the maximum downward excursion has been considered for each, 25 29 and 33 msw. These are referred to in the report as 25-75, 29-100 and 33-130.

2.0 RESULTS

The results are calculated as volume of gas carried in bubbles per ml of tissue or blood. There is no attempt to estimate the number or size of the bubbles. In addition to calculating the gas in bubbles in tissues, an average for the whole body is determined by calculating a weighted mean of all tissue, weighted according to the weighting of blood flow which makes up the central venous blood. This is done because the central venous blood is the one site for which comparisons can be made between the predictions and the results of ultrasonic bubble counting techniques. This is the way in which the model predictions have been validated in the past.

For most of this report the predictions of gas volume in bubbles are given as a percentage of the predicted maximum following the return from excursion. This has been done to give a easier comparison with procedures as presently used.

2.1 CURRENT PROCEDURES

The results in Table 1 show the predicted volume of gas carried as bubbles in brain, as a weighted mean of the two types of brain tissue, and in central venous blood, representing a "whole body" estimate.

Table 1
Maximum gas carried in bubbles per ml of tissue or blood

	25-75	29-100	33-130
Brain	0.000056	0.000055	0.000052
Whole body	0.00039	0.00036	0.00034

These are the levels of gas which give the incidence of DCI seen after excursion but before decompression. Any change of procedure which reduces these levels must lead to a reduction in DCI incidence and a reduced risk of damage to all divers.

2.1 RETURN FROM EXCURSION

Four changes to the return from excursions depth have been considered:

two slower rates of ascent, 10 msw/min and 5 msw/min.

standard rate of ascent, 18 msw/min, but with a 3 minutes hold after a depth reduction of 9 msw

a slower rate of ascent, 10 msw/min, with a 3 minutes hold

As explained in the introduction, because of the high rate of decompression from excursions the central nervous system is a high risk tissue and the main focus of this part of the study has been on reducing bubble formation in the brain tissues.

2.1.1 Increased time to ascend

Table 2 shows the predictions of maximum gas in bubbles, expressed as a percentage of those in Table 1, i.e. the standard procedures, for both brain and the whole body weighted means. The stop has been taken after a 9 msw ascent.

The effect of the slower return is minimal on the whole body gas. This is as would be expected given that much of the body is made up of tissues with a blood flow so slow that the few extra minutes taken on the return would have little effect. However there is a worthwhile benefit to the brain and the slower return to the bell should results in fewer CNS hits.

The results in Table 2 have been plotted in terms of brain gas compared to time taken to return to the bell and this is shown in Figure 1. This figure makes it clear that either a slower rate of ascent or the standard rate with stops can be equally effective, what is important is the time taken. The relationships for 29-100 and 33-130 have been extrapolated slightly to allow calculation of the time required to reduce the brain gas in bubbles by 50%.

Table 2
Maximum gas expressed as a percentage of the values in Table 1

Profile	Duration (mins)	Whole body	Brain
25-75			
18 msw/min	1.4	100	100
10 msw/min	2.5	97.4	83.9
5 msw/min	5.0	96.2	55.4
18 msw/min + 3 mins	4.4	96.7	59.8
10 msw/min + 3 mins	5.5	95.9	46.4
29-100			
18 msw/min	1.6	100	100
10 msw/min	2.9	99.7	87.2
5 msw/min	5.8	98.6	58.7
18 msw/min + 3 mins	4.6	98.1	66.1
10 msw/min + 3 mins	5.9	97.5	56.9
33-130			
18 msw/min	1.8	100	100
10 msw/min	3.3	99.4	85.4
5 msw/min	6.6	97.9	60.2
18 msw/min + 3 mins	4.8	98.2	71.8
10 msw/min + 3 mins	6.3	97.6	62.1

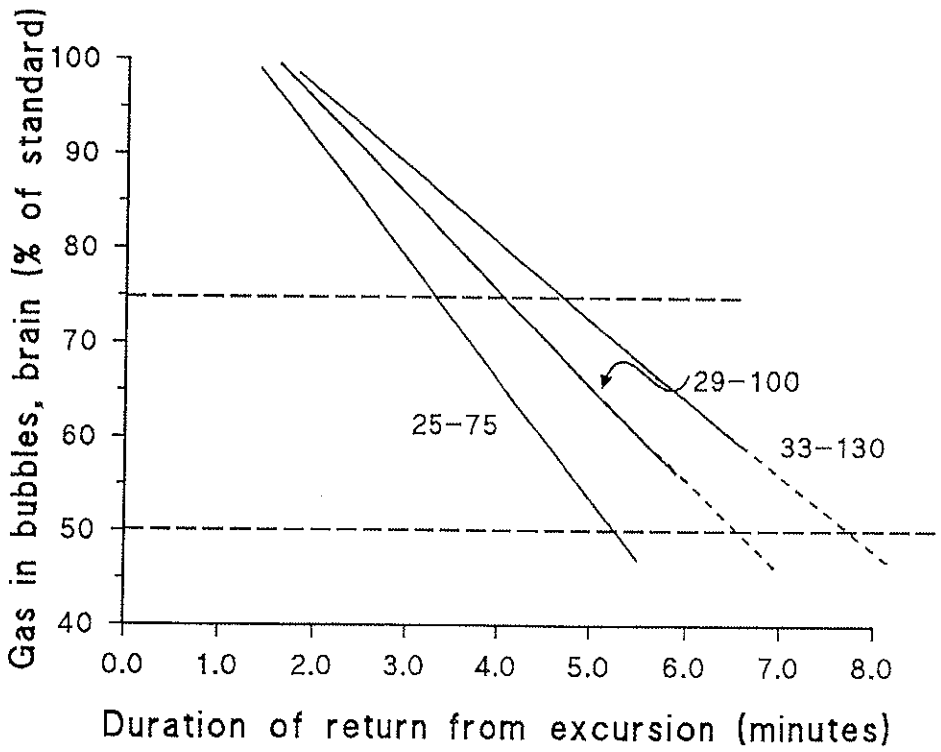

Figure 1 The relationship between predicted gas in bubbles and time taken to return to the bell.

Table 3 shows the time required for return from excursions in order to reduce the gas in brain by 25 and 50% following return to the bell.

Table 3
From Figure 1, the time required to reduce brain gas in bubbles

	Time required to reduce by	
	25%	50%
25-75	3.3	5.3
29-100	4.0	6.5
33-130	4.7	7.7

2.1.2 Bell taken to half excursion depth

For a diving system operating two bells an alternative option to reduce gas load would be to move the bell to a greater pressure at the end of the excursion time and to decompress the bell after it is finally locked on to the system at the end of the bell run. This would have a greater effect on the whole body gas in bubbles than simply taking longer to return to the bell.

The effect of this procedure has been examined by simulating the diver moving back to the bell (which has remained at chamber pressure) at the standard rate of 18 msw/minute, allowing 15 minutes at chamber pressure before compressing the bell to half the excursion depth, holding at that pressure for either 5 or 15 minutes to kill decompression bubbles then decompressing at a linear rate over 30 minutes back to chamber pressure. Once the divers are in the bell it can be moved to the surface and decompression can take place once the bell is locked back on to the system.

Because the move back to the bell is at the same rate as the standard procedure the gas in bubbles in the brain is not reduced but the bubbles exist only until the bell is compressed. The removal of brain bubbles by the bell compression should reduce the CNS hit rate. It would be possible to reduce the peak of bubbles in the brain by following a slower rate of move back to the bell or by reducing the 15 minutes before compressing the bell thus reversing the growth of bubbles before the peak is reached.

Table 4 shows whole body gas at end of bell decompression as percentage of that following the standard procedure. Figure 2 shows the pressure profile for the diver together with the gas carried as bubbles in the central venous blood. The reduction of gas in bubbles by bell compression is obvious. If the bell were held at pressure for longer than 15 minutes the reduction in bubbles would be greater.

Table 4
Effect of using a bell decompression as described in the text

	Bell depth (msw)	Duration of hold	
		5 mins	15 mins
25-75	12.25	91.3	88.7
29-100	14.5	93.8	91.7
33-130	16.5	93.5	91.8

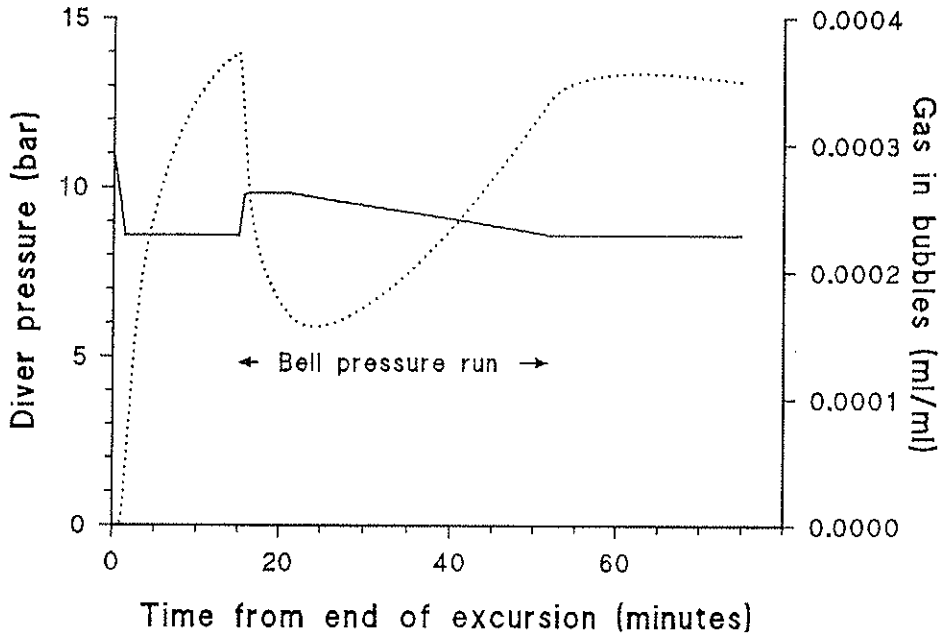

Figure 2 A bell decompression

The gains from the bell decompression as simulated are not great but the recompression could be very significant in preventing CNS hits in that it will compress any bubbles stuck in a critical place and cause them to move on. The procedures and pressure profiles for bell decompression could be refined and made more effective if this approach is considered to be operationally acceptable.

There are implications for the bellman using this procedure but simulation of the exposure which the bellman would experience indicates that the average diver would not have bubbles after being exposed to the bell decompression procedure.

2.2 DECOMPRESSION FROM SATURATION FOLLOWING EXCURSIONS

Simulation of the pressure profile used for decompression from heliox saturation shows that most divers would not have bubbles. Assuming a 20% range in the physiological parameters which determine gas movement, and assuming a normal distribution, less than 5% would be expected to bubble during a decompression.

The situation is very different if decompression begins whilst bubbles are still present following an excursion. Once formed bubbles take a long time to resolve because the inert gas is in effect grabbed by the bubbles; the solubility of gas in body tissues is low, the partition favours the gas staying in the gas phase. The main route for gas to be taken out of the tissue and out of the body is by solution in the blood. Once bubbles form the inert gas is passed back into solution very slowly. The mathematical predictions are that; following the 25-75 excursion, bubbles in muscle will survive for an average of 400 minutes, following 33-130 muscle bubbles will survive 530 minutes and in both cases bubbles in fat will survive in excess of 12 hours. In fact bubbles in fat can survive for several days.

If decompression is started whilst bubbles are present they will grow at a rate determined mainly by the rate of pressure change compared to the rate of blood flow removing the inert gas from the tissue. The more gas there is in bubbles at the start of the decompression the greater the growth. For this reason it is useful to introduce a delay between the end of the final excursion and the beginning of decompression. For this study 4 different conditions have been simulated;

decompression starting 1 hour after the return from excursion

decompression starting after a 6 hour hold

decompression starting after a 6 hour hold with three 20 minutes periods of breathing 1.5 bar oxygen during the last 75 minutes of the hold

decompression starting 12 hours after the end of the excursion.

The results are given in Table 5 as the peak value for the gas carried in bubbles, in each ml of the central venous blood, expressed as a percentage of the peak volume of gas in central venous blood at the end of the excursion (from Table 1). Only 2 exposures were chosen; 25-75 because that gave the greatest predicted volume of gas following the excursion and 33-130 because that decompression gives the longest time and greatest pressure drop, i.e. the greatest scope for bubbles to grow.

Table 5
Maximum gas in bubbles expressed as percentage of that following the excursion

	No hold	6 hr hold	6 hr hold, 1.5 bar O_2	12 hr hold
25-75	198	156	149	107
33-130	469	331	255	224

The benefits of a delay before starting decompression are obvious.

Figure 3 shows the pattern of bubble growth in the fat for 25-75 and 33-130, the reduced rate of growth during the slower decompression rate is apparent as is the benefit of the 4 hour stops in each 24 hours.

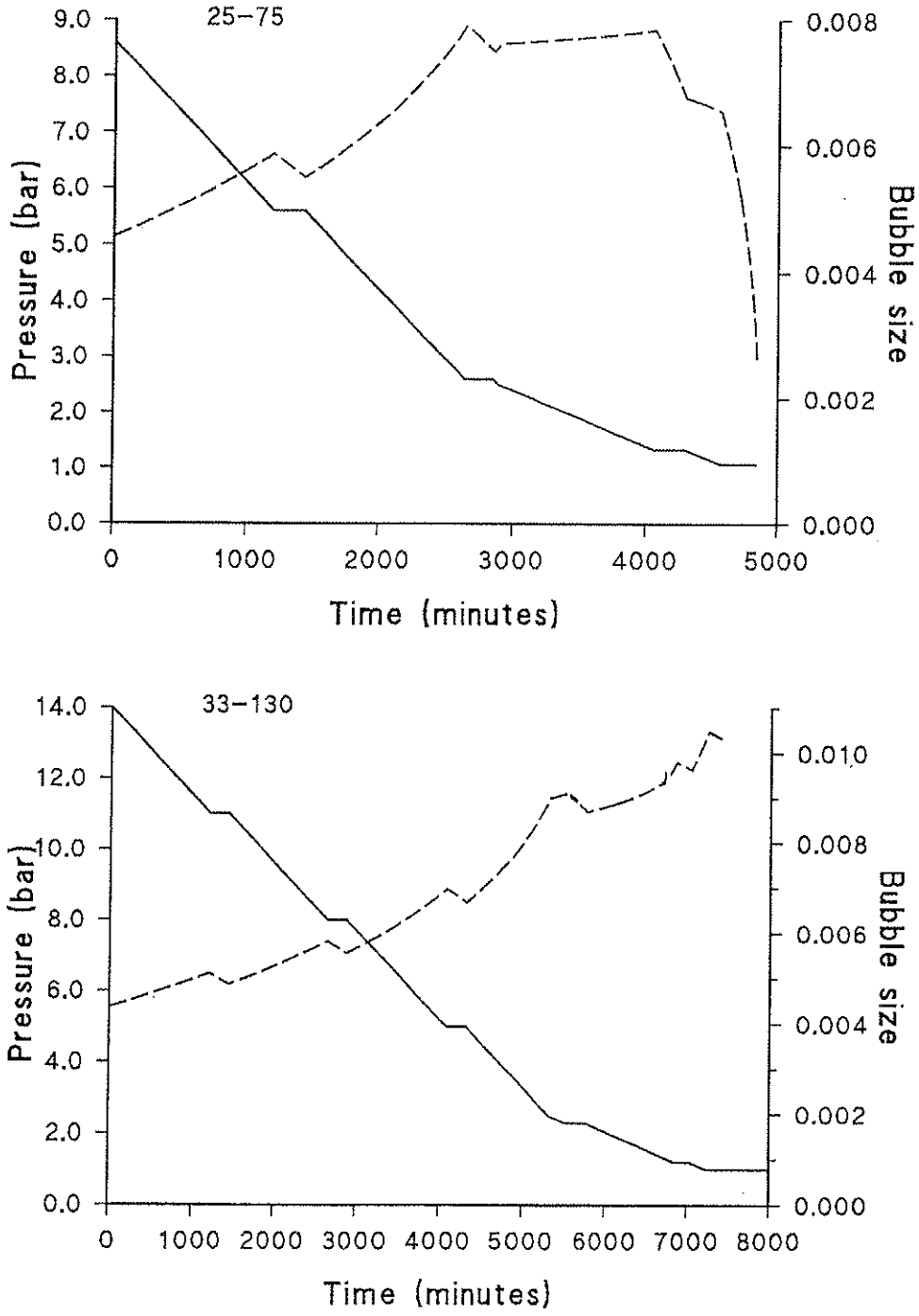

Figure 3 Time course of bubbles growth

In all cases the only tissue bubbling at the time of maximum gas in bubbles is the fat. This fits in with the conclusion from some of the very earliest work with the mathematical model in which the statistical analysis of the incidence of DCI, based on information in the HADES data base, were matched with model predictions. In that study it became apparent that fat was the critical tissue in the incidence of DCI. This does not mean that fat bubbles cause DCI, only that there is a relationship; more bubbles in fat - the decompression was such that there is greater chance of a hit (Flook and Brubakk 1997).

These results indicate that the duration of the hold, prior to starting decompression, should be longer the deeper the saturation depth.

3.0 DISCUSSION

The results show that whereas it is relatively easy to reduce the extent of bubble formation, and of the risk of CNS decompression sickness, immediately following excursions it is less easy to reduce bubble formation during decompression from saturation. The ideal would be to start the decompression bubble-free but this would require a hold of one or two days after the final excursion and a compromise is necessary. The combination of a slower return to the bell after the excursion and a hold prior to decompression, preferably with 1.5 bar oxygen during the last part of the hold, should reduce the volume of gas which forms into bubbles.

It should be remembered that the calculations refer to what will happen in the average man; by definition some divers will have more bubbles and some will have less. If we assume a 20% variation in the physiological and physical factors which determine gas dynamics and bubble formation, and a normal distribution about the mean, about 60% of divers will lie within ± 5% of the results quoted in this report. During the return to the bell somewhere between 90 and 98% of divers will develop bubbles in the brain 60% of whom will be within 5% of the predicted level.

It should also be remembered that there is not a direct link between the amount of bubbles formed and the incidence of DCI. There is a random, statistical, element to the occurrence of symptoms. It is generally true that the more bubbles which form the greater the risk of DCI but symptoms have occurred in divers in whom no bubbles were detected, see Nishi 1993 in which a 0.1% incidence of symptoms is reported in 715 divers who had no detectable Doppler bubbles at any site following short helium dives. It only takes a bubble to lodge in a sensitive site to cause symptoms and this is especially true of the brain. For this reason the use of a bell compression/decompression run would be expected to remove the bubble which has lodged in the wrong place and this addition to procedures could have a marked effect on the incidence of CNS DCI.

It is not possible to predict what will happen in any individual diver. The incidence of DCI hits in commercial diving is now so low that it would take thousands of man-dives to demonstrate a change. However, overt symptoms are only one end-point of the effect of bubbles; bubbles have been shown to have many adverse effects within the body, see for example Brubakk et al 1999, and any change in procedure which reduces the predicted amount of bubble formation should reduce the risk to the diver.

The recommendation from this study is that the return to the bell be made at a slower rate than in the standard procedures, the results in tables 2 and 3 give guidance here; there should be a minimum of 6 hour hold before the start of decompression preferably with 1.5 bar oxygen breathing during the last hour of the hold.

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APPENDIX 1

DETAILS OF CALCULATIONS

A1.1 MATHEMATICAL CALCULATIONS

The mathematical model used in this study is described below and is based on that of Mapleson (1963) which treats the body as eight parallel compartments having the physiological and anatomical characteristics of identifiable tissues. This model allows quantification of inert gas uptake and distribution. Bubble growth during and following decompression is simulated using the physical relationships given in Van Liew and Burkard (1993). In the gas dynamics section all tissues are handled concurrently. In the bubble growth section each tissue is handled independently for the duration of the bubbles. This allows quantification of the volume of gas which is carried in bubbles in each tissue. It is also possible to determine the volume of gas carried in bubbles in the pulmonary artery, central venous blood, by calculating a weighted mean of gas in bubbles in the venous drainage from each tissue.

A1.1.1 Gas dynamics

The uptake of inert gas during the time spent at maximum depth is calculated in the gas dynamics section of the model. The eight compartments are defined so that each contains all the tissues which have the same time constant. As described in Mapleson, the time constant for each tissue is the total capacity for the inert gas in the tissue divided by the rate of supply/removal of gas in the blood. This is expressed as:

$$\frac{(v_t \times \lambda_t) + (q_t \times \lambda_b)}{(\dot{q}_t \times \lambda_b)}$$

v_t, q_t and $\dot{q}_t$ are the volume of tissue, the volume of blood contained within the tissue

and the blood flow through the tissue respectively. λ_t and λ_b are the partition coefficients for tissue:gas and blood:gas.

Blood flow to each tissue can be changed to simulate different physiological conditions. Tissue volumes can be changed to allow simulations of different body shapes. Tissue groupings are listed in Table A1 together with the time constant for each compartment, for nitrogen and helium, for a standard 70 kg man at rest. The model can also be used with more compartments. For example a 9th compartment can be used to represent the proportion of the muscle which is involved in physical activity, or the skin to simulate conditions of thermal regulation of skin blood flow.

The values used for the partition coefficients are "best values" for the Ostwald solubility coefficient taken from the literature, notably from two reviews, Steward et al (1973) and Weathersby et al (1980). These are: for nitrogen in blood and all tissues except fat 0.0148 (units are ml/ml equilibration at 101 kPa volume measured at standard pressure and 37°C) for fat 0.066; for helium in blood and all tissues except fat 0.0092, for fat 0.015.

TABLE A1

Characteristics of each compartment. Time constant in minutes.

Compartment	Tissues	Time constant
1	Adrenals, kidneys, thyroid	0.86
2	Heart, brain grey matter	1.87
3	Liver plus portal system, other small glands and organs	3.07
4	Brain white matter	5.31
5	Red marrow	12.25
6	Muscle and skin	50.62
7	Nonfat subcutaneous	69.14
8	Fatty marrow and fat nitrogen helium	211.3 78.3

Uptake and washout of inert gas in each compartment is calculated using an exponential relationship. The calculation is reiterated at small time intervals.

This section of the model requires, as input, the pressure profile and the breathing gas mixture. At each time increment the model calculates:

the arterial oxygen and arterial inert gas partial pressure;

the venous oxygen partial pressure from a standard oxyhaemoglobin dissociation curve;

venous inert gas partial pressures.

Arterial and venous carbon dioxide pressures for the body at rest are assumed to be the textbook standards of 5.5 and 6.1 Kpa. In the standard format each tissue is assumed to be in equilibrium with the venous blood draining it and inert gas exchange at the lungs is assumed to be complete in a single passage of blood through the lungs.

If it is necessary to evaluate the predicted gas in bubbles in terms of Doppler bubble scores in central venous blood the mixed venous inert gas partial pressures are calculated as weighted means of the 8 contributory venous streams. The weighting factor for each is the ratio of blood flow to the compartment divided by the total cardiac output. Table A2 lists the values for the weighting factors for a body at rest.

TABLE A2

Weighting factors used to calculate mixed venous values from 8 separate venous values

Compartment	Tissues	Weighting factor
1	Adrenals, kidneys, thyroid	0.28
2	Heart, brain grey matter	0.17
3	Liver plus portal system, other small glands and organs	0.33
4	Brain white matter	0.03
5	Red marrow	0.02
6	Muscle and skin	0.13
7	Nonfat subcutaneous	0.01
8	Fatty marrow and fat	0.02

These values change when conditions change. For example increased physical activity results in an increase in muscle blood flow which, in addition to causing a reduction in muscle time constant, results in an increase in the proportion of pulmonary artery blood which derives from muscle.

Total venous dissolved gas partial pressure for each compartment is determined as the sum of the inert gas + oxygen + carbon dioxide + water vapour. A separated gas phase (bubbles) is assumed to occur whenever the total dissolved gas pressure exceeds the sum of environmental pressure plus surface tension forces in the bubble and this ratio; total dissolved gas pressure:bubble internal pressure, is calculated for each compartment and for the mixed venous blood at each time increment throughout the whole period of time which is being simulated. Simple inspection of this section of the model shows when the bubbles will start to grow in each tissue, i.e. when the ratio of dissolved gas pressure to bubble internal pressure exceeds 1. Thus the assumption is made that sufficient nuclei exist to enable bubble growth to go ahead without the requirement for any additional source of energy.

This section of the model can be run with multiple parallel calculations of inert gas movement to enable mixtures of inert gases to be studied.

A1.1.2 Bubble dynamics

The bubble dynamics section of the model is exactly as described by Van Liew and Burkard (1993) for multiple bubbles in tissue. An independent calculation of bubble dynamics is made for each compartment. For an 8 compartment simulation there are 8 parallel repeats of the Van Liew-Burkard model.

The equations in the Van Liew-Burkard model account for the physics of bubble growth and thereby take account of:

the effect of bubble density

gas diffusion between tissue and blood

the rate of removal of inert gas by blood

the effect of surface tension on internal pressure of the bubbles

The initial conditions are taken as the conditions at the last time increment before decompression starts. Thus the values for blood flow and time constant of the compartment, environmental pressure, arterial inert gas pressure, inert gas pressure for tissue and venous blood and venous oxygen pressure are provided from the gas dynamics section of the model. In this way mass balance is ensured on the switch from time at pressure to decompression. The calculations are reiterated for small time intervals throughout decompression. In this way the "fate" of the inert gas which is in the tissue at the start of decompression is followed throughout as it moves between blood, tissue and bubble during bubble growth and decay.

The starting radius of the nuclei on which the bubbles form is assumed to be 2 microns and this value is held constant until growth begins. This gives an initial values for internal surface forces of 0.5 bar so that the internal bubble pressure starts at environmental pressure plus 0.5 bar. If the initial size of the nucleus is

assumed to be bigger than 2 μm then in pressure due to surface forces is smaller than 0.5 bar. A slight increase in initial bubble size was used to start the bubbles for two of the profiles in section 3.1.3.

The diffusion coefficients used are: nitrogen, aqueous tissue 0.001302 cm^2/sec, fat 0.001038; helium 0.002802 cm^2/sec for aqueous tissue and 0.002172 for fat.

Time increments are chosen so that changes in them do not cause changes in the result. When used for a single inert gas the main problem relates to time increments which are too large. If the time increments are too big, so that the total volume of gas moving between bubble and tissue within the time interval is large, the change in bubble radius (and in all other calculated values) overshoots and oscillations are initiated. This is positive feedback and the calculation breaks down. Large movements of gas result from very large pressure changes, large numbers of bubbles changing size and slow removal of dissolved gas by the blood flow. Thus a large pressure drop in a tissue with a long time constant and high bubble density is more likely to result in oscillations and a failure to complete the simulation. The time increments must be reduced to a level at which no oscillations occur. Invariably the time increments required by this section of the model are much smaller than those required by the gas dynamics section and are usually dictated by the compartment which has been given the highest bubble density. The choice of bubble density is discussed in section A1.1.3.

Obviously such instability in the calculations can lead to gross error in the results. For example it is possible for the bubble radius to drop to zero during a period of oscillation whereas when corrected so that oscillations do not occur the bubble will last for a longer time. If the results are not studied carefully it would be possible to record a false early resolution of bubbles. Oscillations are manifest as sudden changes in inert gas partial pressures and in bubble radius; inert gas pressures may become negative. After every calculation inert gas pressures and bubble radius are displayed graphically and carefully inspected to look for such sudden changes. Time increments are adjusted until inert gas partial pressures change in a smooth continuous way consistent with what is happening to the environmental pressure and bubble. When this situation is reached the outcome must then be shown to be independent of time increment as long as oscillations do not recur. Only when these conditions have been fulfilled is the result

accepted. This is time-consuming but becomes less so with experience. All the results presented here have been subjected to this type of quality control.

Where pulmonary artery gas is to be calculated the 8 bubble dynamics sections are combined by calculating for each time interval the weighted mean values for:-

bubble radius

tissue and venous blood inert gas partial pressure

blood total inert gas concentration (the sum of dissolved gas and gas in bubbles)

gas volume carried in bubbles in the blood (assuming that bubbles have formed to the same extent in the blood and in the tissue)

total gas concentration (in bubbles and dissolved) in the tissue.

The weightings are as given in Table A2. Thus all parts of the model are linked and because of this there is a "seamless" join whenever there is a switch from one part to the other. In other words mass balance is automatically maintained.

A1.1.3 Bubble density

The number of bubbles which can form in any tissue is unknown. The energy required to generate a *de novo* bubble is very high and it seems unlikely that this is the normal mode of bubble initiation on decompression. A bubble can grow more easily if there is a nucleus of some kind available; nuclei can be gaseous or solid. It is not appropriate to make a detailed examination of the factors which determine the incidence of suitable nuclei here, the readers is referred to Vann (1989) for more information, a brief summary only is included here.

It is possible to argue that bubble density is very high throughout the body. Van Liew and Burkard (1993) suggested that the number of bubbles formed depends on the magnitude of the decompression move. It has also been argued that the number of bubbles formed depends on the level of supersaturation at the start of

decompression. This corresponds to the findings of Yount and Strauss (1976) in their studies of bubble formation in gelatin. In the original validation of the model high bubble density was one of the conditions studied.

The argument that bubble density relates to the magnitude of the decompression move or to supersaturation has a weakness in that the experimental evidence for high density depends on the methods used to visualise and count the bubbles. It may simply be that more bubbles grow large enough to be detected following a bigger decompression step. It is possible to argue the case that the number of bubbles formed is determined by the availability of nuclei.

Candidates for solid nuclei might be some of the larger molecules which are in the body, some protein molecules could be candidates. Gas nuclei can be formed in the body and there may be continuous formation on the arterial side of the circulation for example by the action of the heart valves (Fox and Hugh 1964); throughout the body by the action of ionising radiation (Evans and Walder 1978); by activity in skeletal muscle (Vann 1989). The simplest pattern of bubble density which follows from this reasoning is that all parts of the body have similar bubble density levels except skeletal muscle, in which tribonucleation may be caused by the muscle movement, fat (because of the large lipid molecules) and the arterial side of the circulation because of the action of the heart which may result in tribonucleation.

This simplistic approach was been used to determine an alternative low range of bubble density values. For the body at rest all compartments are assumed to have a bubble density of 100/ml except for muscle and fat. Muscle has been assumed to form 500 bubbles/ml to allow for the fact that the respiratory muscles and postural muscles are continuously active. Fat has been assumed to form 8,000 bubbles/ml, this density is in the region where precise bubble number has little effect on the outcome. This option is referred to as the "low" bubble density in the following section. These values were used throughout the work dealt with in this report and the justification for this choice is discussed in the next section.

A1.1.4 Validation of the model

The model used in this work was validated as part of a study undertaken for OMEGA, a consortium of Norwegian oil companies and the Norwegian Petroleum Directorate, and reported in Flook and Brubakk (1996). Several forms of predictions from the model were compared with the peak bubble counts as recorded by transoesophageal ultrasonic scanning in anaesthetized pigs. Fourteen different decompression types were involved and 106 animals contributed results.

Of various output values available from the model the peak predicted gas volume carried as bubbles in the pulmonary artery was the one which correlated best with measured peak bubble counts. The best correlation was for the "low" bubble density option; correlation coefficient $r = 0.91$, $P<0.001$. This very high, statistically significant correlation coefficient indicates that if appropriate physiological and anatomical values are used in the model it is a very reliable way to evaluate decompression stress. The relationship between the predicted peak volume of gas carried as bubbles in pulmonary artery blood and the peak measured bubble count in the pulmonary artery, is shown in figure A1. This relationship has been used to relate the model predictions to likely Doppler scores through the relationship between bubble counts and Doppler scores reported of Eftedal et al (1998).

The low bubble density case is now routinely used; that is with bubble density 00/ml in all tissues except muscle which has density 500/ml, and fat 8,000/ml.

Using the conditions as determined by the comparison with the experimental work the model has been calibrated for several conditions; for air exposures against trials of both air dives and compressed air work and for heliox with nitrogen short dives by comparison with mineclearance trials carried out by the Canadian Experimental Diving Unit.

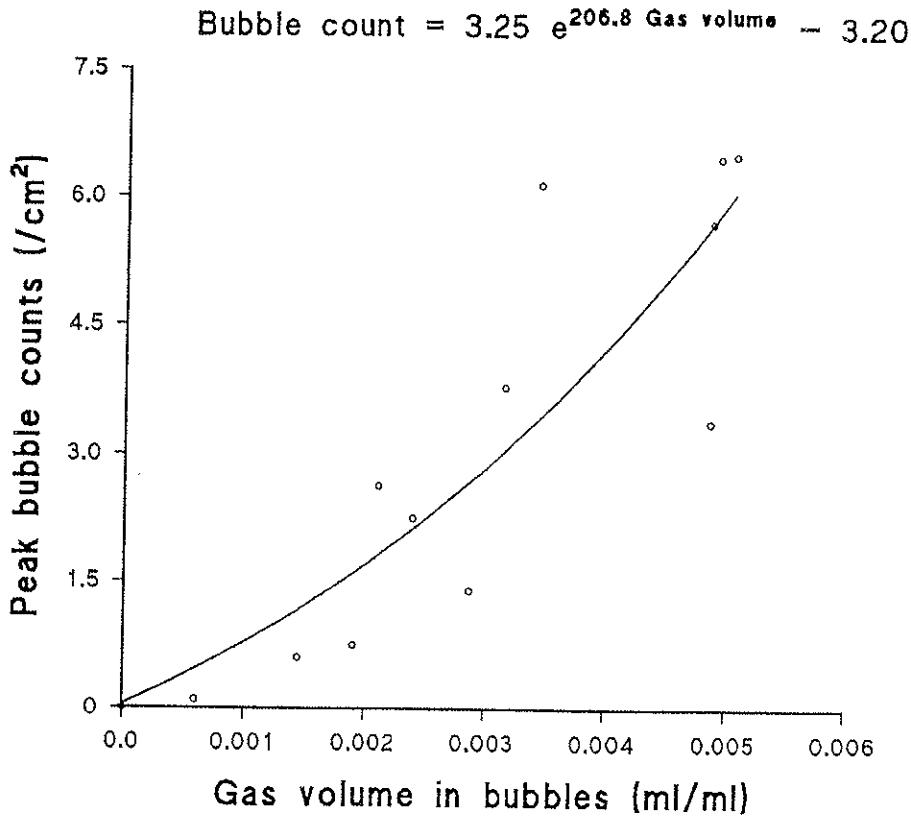

Figure A1 Relationship between predicted peak gas in bubbles in the mixed venous blood and measured pulmonary artery bubbles. See text for details