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Application of an Advanced Physiological Model of Decompression in the Evaluation of Decompression Stress

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Application of an advanced physiological model of decompression in the evaluation of decompression stress.

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This is the final report of a study in which a physiological model of decompression was used to predict the level of decompression stress from 12 different hyperbaric exposures and the predictions compared with the results of trials in which the level of central venous bubbles were determined by precordial Doppler measurements. In the early stages of the work it became apparent that direct comparison of the trials was not possible because of uncertainty about the levels of physical activity undertaken at maximum pressure on each trial and because of apparent discrepancies between the Doppler results. These problems were handled by grouping the trials, keeping those with the same level of physical activity and same Doppler procedures in a group. The model predictions and estimated bubble numbers agreed within each group.

This study has shown that the model can be used with some confidence to determine relative decompression stress for any type of decompression profile. Of particular interest are the facts that the model; identified the trial most likely to cause decompression symptoms in skin; shows the complexity of the factors which determine bubble growth when inert gas switches are used; demonstrates the need to design trials with more attention to the activity of the divers throughout the exposure; demonstrates the difference in decompression stress between hyperbaric exposures in a gaseous environment and those in water.
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1.0 INTRODUCTION

The main purpose of the work described in this report was to evaluate a physiological model of decompression, created by the author of this report, as a means of determining the decompression stress likely to arise from any particular hyperbaric exposure. This was to be done by comparing the amount of gas which this model predicted would be carried as bubbles in the central venous circulation with the recorded bubbles as determined by Doppler monitoring of the pulmonary artery of subjects taking part in a series of chamber trials. The underlying aim of the work was to validate the theoretical approach as a means of assessing the safety of a decompression.

This work forms a natural development of earlier work sponsored by the Health and Safety Executive which mainly attempted to evaluate decompression stress by analyzing the incidence of decompression symptoms in operational procedures. This earlier work is reviewed by Robertson and Simpson (1997). They point out the need to move away from DCI incidence as an end-point for two reasons:

- the success of the earlier work has resulted in DCI becoming a rare event in operational diving to the extent that new procedures would need many thousands of exposures before the level of decompression stress can be evaluated.

- increasing evidence indicates that decompression bubbles can cause long-term changes in the body, whether symptoms have been present or not, and improving safety in the future must mean designing decompression profiles which result in fewer bubbles.

Previous chamber trials have examined the effectiveness of new and modified dive procedures in reducing gas emboli in the pulmonary artery of divers (Brooke et al 1993, James and Lafferty 1993, Lambertz 1996, Robertson and Simpson 1997). These studies included assessment of decompression profiles designed at Institute for Environmental Medicine (IFEM) at the University of Pennsylvania. It was the intention of the current study to assess the validity of the Flock model by using it to predict gas scores for a range of dive profiles and procedures, and to compare the predictions with known results from the earlier chamber trials. A subsidiary objective was to use this model to predict gas phase, size and duration of bubbles arising following decompression on the IFEM tables. The requirement for this aspect of the work stemmed from a need to examine why, during the trials, some decompressions using the IFEM tables gave more bubbles at the end of the decompression than was predicted by the IFEM model on which the tables were based.

In the course of the current study twelve profiles were simulated of which 5 were IFEM profiles. The model predictions were compared with the Doppler scores from several series of chamber dives using the same 12 profiles.
2.0 MATHEMATICAL MODEL USED IN THIS STUDY

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.

2.1 GAS DYNAMICS

The uptake of inert gas during the time spent at maximum depth is calculated in the gas dynamic 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:

\[
\frac{(v_i \times \lambda_i) + (q_i \times \lambda_i)}{(q_b \times \lambda_i)}
\]

where \(v_i\), \(q_i\), and \(q_b\) are the volume of tissue, the volume of blood contained within the tissue and the blood flow through the tissue respectively. \(\lambda_i\) and \(\lambda_b\) 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 1 together with the time constant for each compartment, for nitrogen, for a standard 70 kgm man at rest. All work presented here has used this grouping of tissues but the time constants have been adjusted to take account of the level of physical activity during the time spent at maximum depth, during decompression and during 2 hours following return to the surface. 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. For most of this work this has not been done because the only bubble data available for comparison with model predictions were for pulmonary artery bubbles; therefore the appropriate comparison is with predicted pulmonary artery gas phase volume. However one of the trials gave a high incidence of skin decompression symptoms and this has been simulated with a 9th compartment representing skin.
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. The effect of temperature on the solubility of each gas has been evaluated in accordance with the data given in Allott et al (1973).

**TABLE 1**

**Characteristics of each compartment. Time constant in minutes.**

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Tissues</th>
<th>Time constant</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Adrenals, kidneys, thyroid</td>
<td>0.859</td>
</tr>
<tr>
<td>2</td>
<td>Heart, brain grey matter</td>
<td>1.869</td>
</tr>
<tr>
<td>3</td>
<td>Liver plus portal system, other small glands and organs</td>
<td>3.066</td>
</tr>
<tr>
<td>4</td>
<td>Brain white matter</td>
<td>5.313</td>
</tr>
<tr>
<td>5</td>
<td>Red marrow</td>
<td>12.25</td>
</tr>
<tr>
<td>6</td>
<td>Muscle and skin</td>
<td>50.621</td>
</tr>
<tr>
<td>7</td>
<td>Nonfat subcutaneous</td>
<td>69.143</td>
</tr>
<tr>
<td>8</td>
<td>Fatty marrow and fat</td>
<td>211.3</td>
</tr>
</tbody>
</table>

The Mapleson parameter values are for resting man; during periods of activity the parameter values have been adjusted in accordance with information derived from the literature. These are described at the appropriate stage in this report.

Uptake and washout of inert gas in each compartment is calculated using an exponential relationship. The calculation is reiterated at small time intervals. It is important to work with time intervals which are small enough so that the result is independent of the size of the increment.

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.

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 2 lists the values for the weighting factors for a body at rest.

**TABLE 2**

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

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

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 environmental pressure and this ratio, total dissolved gas pressure:environmental 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 environmental 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.
2.2 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 in each compartment are taken as the conditions at the last time increment before decompression starts, i.e. the end of the time at maximum depth. Thus the values for blood flow and time constant of the compartment, chamber 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 each time interval throughout decompression and the following two hours. 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.

Time increments are chosen so that changes in them do not cause changes in the result. 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 2.3.

Each compartment has bubble growth and decay calculated throughout the lifetime of the bubble or for the length of time after surfacing for which information is required, which ever is the shorter. In the present study the simulations have included the first 2 hours after the end of decompression. The starting volume of the nuclei on which the bubbles form is assumed to be 2 microns and this value is held constant until growth begins. For some tissues, during some profiles, bubbles grow and decay and grow again. The starting size, 2 microns, is held throughout the period between the end of the first decay and the subsequent re-establishment of the bubbles. This cycle, of bubble growth and decay, may be repeated several times for a complex decompression profile for the very fast compartments.
Figure 1 shows how the characteristics of the compartment affect bubble formation and growth. Bubble radius is shown for bubbles in compartments 1, 2, and 3, all having the same initial gas loading and differing only by the time constant (blood flow) for each. The lower the blood flow through a compartment the earlier a bubble can start to grow and the larger it can grow because less gas is removed by the blood during the early stages of growth.

![Graph showing bubble growth in compartments 1, 2, and 3](image)

**Figure 1**

Bubble growth in compartments 1, 2 and 3
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 2. 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.

### 2.3 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. The model was validated using a "high" bubbles density case drawn from Van Liew and Burkard. 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. The difference between these two arguments is relevant only for tissues which have not been saturated by the start of the decompression and which would therefore have a lower bubble density. For much of the work described in this report this would apply only to compartments 5, 7 and 8, i.e. red marrow, non-fat subcutaneous tissue and fatty marrow and fat. In the original validation of the model high bubble density was one of the conditions studied and this will be considered in a later section of this report.

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 tritonucleation 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 tritonucleation.

This simplistic approach was 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. A greater density in muscle should perhaps be used if there is physical activity during decompression. Fat has been assumed to form 10,000 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 bubble density values were used throughout the work dealt with in this report and the justification for this choice is discussed in the next section.

2.4 PREVIOUS 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. The relevant parts of this work are summarised here.

The mathematical model generates information about gas dynamics and bubble size which can be used in several alternative ways as a means of predicting decompression stress:

1. The potential for bubbles to form, which relates to the inert gas loading at the end of the time spent at maximum depth. This takes no account of the subsequent decompression procedure and is equivalent to the type of data analysis reported by Robertson and Simpson (1996) in figures 1-6. These figures show attempts to relate the incidence of DCI with maximum depth and time spent at that depth;

2. The mean mixed venous bubble radius, which is calculated as the weighted mean of bubble radius in each compartment. The standard weighting factors were used. The calculation is made for each time interval throughout decompression and 2 hours following return to surface. This approach has justification only in that bubble radius should relate to the volume of gas in bubbles supplied to the mixed venous blood.

3. The predicted gas volume carried as bubbles per ml of mixed venous blood. This is a weighted mean of the gas volume carried in bubbles in each tissue. The calculation is made for each time interval used to calculate bubble growth. This approach involves the assumption that within each tissue bubbles form just as easily in the venous blood within the tissue as in the tissue itself.
The predicted total gas concentration in the venous blood derived from a weighted mean of that for each tissue. Thus the total of gas carried in bubbles and gas remaining in solution contribute to this. Again the calculations are made for each time interval used in calculation of bubble growth.

Predicted pulmonary artery gas partial pressure derived as the weighted mean of inert gas partial pressure i.e. gas left in solution, at each time interval during decompression.

Peak values for these five parameters were compared with peak bubble counts determined by trans-oesophageal ultrasonic scanning in pigs subjected to a wide range of hyperbaric exposures in the laboratory in Trondheim during the years 1990 to early 1996. Peak bubble count is determined as bubble number per unit area of field of view of the transducer, bubbles/cm². Each comparison was made for both the high and low bubble density case. Fourteen different decompression profiles contributed to the analysis, these included non-stop decompressions, staged decompressions, decompression with a constant rate of pressure reduction, Sur-D decompressions and a staged decompression in which air was changed to heliox for part of the decompression. For each series of experiments the average peak bubble count was determined for all animals in the series.

The potential for bubbles to form (1) and the predicted total gas concentration (4) both relate to the time spent prior to decompression and although the total gas concentration during the decompression is also influenced by the decompression procedure neither of these approaches resulted in a significant correlation with measured bubble counts for either high or low bubble density. Neither did the predicted bubble radius (2), nor radius cubed (equivalent to bubble volume).

The peak predicted gas volume in bubbles (3) and the predicted pulmonary artery gas partial pressure (5) both correlated significantly with measured bubble counts; pulmonary artery partial pressure only for the high bubble density case, correlation coefficient \( r = 0.84, P<0.001 \). The best correlation was for the predicted peak gas volume carried in bubbles for the low bubble density case, 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 2.

Based on this earlier work the decision was taken to use the low bubble density case throughout the current study; that is with bubble density 100/ml in all tissues except muscle which has density 500/ml, and fat, 10,000/ml.

Validation of the model as presented in figure 2 compares model predictions of peak volume of gas carried as bubbles in the mixed venous blood with mean peak bubble counts for groups of 6 or more pigs. Given that physiological and anatomical parameter values used in the model are average values, derived usually from a large number of subjects, the model cannot predict what might happen in either an individual or a very small number of individuals. Six subjects should be taken as the minimum number to be compared with model predictions. Three of the 12 trials in this study had less than 6 subjects.
To predict bubble count from model

\[
\text{Bubble count} = 3.25 \cdot e^{20.8 \cdot \text{Gas volume}} - 3.20
\]

Figure 2

**Relationship between predicted peak gas in bubbles in the mixed venous blood and measured pulmonary artery bubbles in pigs.**

Some of the decompression profiles studied in this work resulted in predicted peak gas in bubbles in the human subject much greater than for any of the animal experiments. This leads to extrapolation beyond the limits for which the model has been validated. It is also possible to argue against using the relationship given in figure 2 because continuous transoesophageal scanning, which produced the bubble numbers in figure 2, is more sensitive, more reproducible and allows identification of a true peak of bubble numbers compared to intermittent transthoracic bubble detection techniques used in the human trials which are the basis of this work. Therefore all the results for the main body of the work presented here are given as predicted gas volume carried in mixed venous blood rather than as predicted bubble numbers.
2.5 PRELIMINARY USE OF THE MODEL

In a preliminary study the model was used to predict the peak volume of gas carried as bubbles for six trials, A to F, and to compare the prediction with the outcome as determined by Doppler scores for pulmonary artery bubbles. The reader is referred to Section 3.1, to Table 3 and to Figures A1-A11 in the Appendix for details of the decompression profiles. In several trials decompression procedures were identical but the divers were engaged in different levels of activity or experienced different thermal conditions. It will be apparent from consideration of sections 2.1 and 2.2 that gas uptake and bubble formation and growth are most strongly influenced by the time constant for each tissue and, through that, by the gas solubility. The time constant for each tissue is determined largely by the blood flow through the tissue; the gas solubility is influenced by tissue temperature. If blood flow and gas solubility are not measured during the course of the experimental work the simulations have to be made using estimates. These can only be based on information available in the literature relating the effect of exercise to muscle blood flow and temperature changes and relating the influence of external temperatures on tissue temperatures.

The information which was provided about the experimental work was not sufficient to define the levels of activity with any degree of confidence. Whereas it may be reasonable to assume that average conditions, derived from the literature, might apply to all divers in a single trial, i.e. all runs using the same procedures, it is not possible to have confidence in comparisons of activity levels between trials. The purpose of the preliminary study was to evaluate how strongly the predicted outcome depended on the values chosen for muscle blood flow and solubility changes.

Profiles A to F were initially simulated "blind". The person making the simulations had no information about the Doppler bubble scores. Profiles C and E were simulated twice, once for a level of physical activity slightly higher than that assumed for trials A, D and F; the second time with the same of activity as for trials A, D and F. When ranked in ascending order of predicted volume of gas in bubbles the order was B, C, A, F, E and D.

When the Doppler scores were evaluated the profiles ranked in order C, B, E, A, D, and F. Trial D, as will be discussed later, has to be set aside as a special case because all divers taking part were treated for DCI symptoms and were therefore recompressed before the maximum Doppler scores were determined. B, A and F were ranked by the simulations in the correct order but both C and E were too high although ranked correctly relative to each other. Thus it is possible to conclude that the level of physical activity assumed for trials C and E is too high. When the simulations were repeated using muscle blood flow for C and E set to 25% that used for A, D and F the predictions ranked all 5 trials in the correct order.

Thus the predictions from the model are shown to be critically dependent on the estimates of physical activity. Precise details of the final physiological changes used in the simulations are given in section 3.6.

The facility to build onto the model simulations of changing muscular activity, of changing body surface temperature and of the effect of immersion is unique to a model which has a physiological basis. Probabilistic models which rely on use of a large data base to set base-line parameter values cannot be used in this way. Thus despite the apparent difficulty to determine an appropriate level of physical activity it is possible to use this model to compare the effect of different conditions on decompression stress.
3.0 THE TRIALS

Twelve profiles were used in this study; 9 were Sur-D profiles, trial J was a staged decompression, K and L were staged decompressions with gas switches to heliox and oxygen. Table 3 lists the essential details of each trial together with the number of the figure in Appendix 1 which shows the decompression profile of each. Each trial is described in more detail in the following sections.

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>DEPTH (Metres)</th>
<th>TIME (Minutes)</th>
<th>DIVER CONDITIONS at maximum depth</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>36.4</td>
<td>40</td>
<td>Wet. Light work</td>
</tr>
<tr>
<td>B</td>
<td>36.4</td>
<td>40</td>
<td>Dry. No work</td>
</tr>
<tr>
<td>C</td>
<td>36.4</td>
<td>40</td>
<td>Wet. 10 mins on/off work, lifting</td>
</tr>
<tr>
<td>D</td>
<td>45.5</td>
<td>40</td>
<td>Wet. Light work</td>
</tr>
<tr>
<td>E</td>
<td>45.5</td>
<td>40</td>
<td>Wet. 10 mins on/off work, lifting</td>
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<td>F</td>
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<td>Wet. 10 mins on/off work, cycling</td>
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<td>K</td>
<td>40</td>
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<td>Wet. Moderate work</td>
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<tr>
<td>L</td>
<td>40</td>
<td>90</td>
<td>Wet. Moderate work</td>
</tr>
</tbody>
</table>
3.1 BRIEF DESCRIPTION OF EACH TRIAL

3.1.1 Trial A

This exposure, 40 minutes at 36.4 metres, was followed by an in-water decompression without stops and a 38 minute chamber decompression on oxygen with an air break. Final decompression, from 12.1 metres, was carried out on oxygen and took 10 minutes. The profile is shown in figure 3.

Trial A involved a diver wearing a hot water suit throughout the major part of the time at maximum depth. This diver engaged in light activity during the bottom time.

One of the significant characteristics of this trial is that at the end of the time at maximum depth in water the diver performed an upward excursion of 3 metres to a dry chamber, followed by a recompression of 3 metres in a dry chamber, followed by a stop at 36.4 metres during which he removed the suit. Thus the in-water decompression was, in fact, carried out in a dry chamber. The surface interval involved no activity. The chamber decompression was undertaken at rest in a dry chamber at thermo-neutral temperatures.

3.1.2 Trial B

Trial B involved a bellman on a similar exposure to that for Trial A, except that 35 minutes were spent at 33.3 metres prior to compression to 36.4 metres for 5 minutes.

He was at rest in a thermo-neutral environment. Following the compression to 36.4 metres this trial was identical to Trial A and is shown in figure 3.

![Graph showing decompression profiles for Trials A and B](image-url)
3.1.3 Trial C

Following 40 minutes at 36.4 metres, the in-water decompression was carried out without stops, chamber decompression was the same duration as trials A and B but the pattern of oxygen and air breathing was different. The final decompression was on oxygen and the profile is shown in figure 4.

Trial C diver wore a wet suit. This diver carried out a pattern of activity which was 10 minutes physical work followed by 10 minutes rest, repeated, during the 40 minutes at maximum depth. The overall level of physical activity during the 10 minutes work periods has been taken as lower than that for Trial A because of the intermittent nature of the work, lifting weights, and the fact that it was carried out in 10 minute work:rest cycles. It has been assumed that the wet suit kept the diver in thermo-neutral conditions.

This diver did not make an excursion and carried out the in-water decompression still in a wet suit.

The wet suit was removed some time during the surface interval which would involve the diver in some activity during that time.

![Figure 4: Decompression profile for Trial C](image-url)

**Figure 4**
Decompression profile for Trial C
3.1.4 Trial D

After 40 minutes at 45.45 metres the in-water decompression was carried out on air with 4 stops totalling 17 minutes. The chamber decompression, as originally supplied to the author, lasted 33 minutes and was on oxygen with one air break. Decompression was on oxygen and at the same rate as the previous trials. This profile is shown as figure 5a.

Trial D diver wore a hot water suit during 35 minutes at the bottom. As in Trial A this diver performed an upward, in-water, excursion of 3.3 metres followed by a compression in a dry chamber back to 45.45 metres for 5 minutes during which time the hot water suit was removed. The diver performed light physical activity during the bottom time. The in-water decompression was in thermo-neutral conditions and the surface interval was at rest in thermo-neutral conditions.

The chamber decompression was, as in all the trials, at rest in thermo-neutral conditions.

"Trial D is not strictly comparable with any of the others because all 4 divers taking part in this trial were treated for DCI symptoms and 2 of the 4 were decompressed on a more conservative profile.

In addition, during the final stages of preparation of this report, after completion of all the simulations, it was discovered that an error had been made in reading the records of this trial. The author was asked to simulate a chamber decompression of 33 minutes whereas in fact it lasted 63 minutes in agreement with the requirement of the decompression table for this exposure. The correct profile is shown in figure 5b. However the fact that all four divers taking part in this trial were treated for symptoms before the maximum bubble score could be determined is the critical factor and this trial should not be compared with the other trials no matter which decompression procedure is simulated. To draw attention to this fact D is written as "D in the tables."
Figure 5
Decompression profile for Trial D as simulated (5a) and the correct decompression (5b).
3.1.5 Trial E

After 40 minutes at 45.45 metres the in-water decompression was the same as in Trial D. The chamber decompression was longer than that simulated for Trial D, 64 minutes with 2 air breaks, but see note above. Decompression was on oxygen at the standard rate figure 6.

In Trial E the diver was in a wet suit during the bottom time carrying out the same pattern of activity as for Trial C. This trial was also similar to Trial C in that the wet suit was removed during the surface interval, no in-water excursion was performed and the in-water decompression was carried out in a wet suit.

![Graph showing decompression profile for Trial E]

**Figure 6**
Decompression profile for Trial E
3.1.6 Trial F

This trial was identical to Trial A up to the start of decompression. The in-water decompression was slower than for Trial A; decompression rate 25 feet/min compared to 30 feet/min. The chamber decompression was considerably shorter; 16 minutes at 40 feet on oxygen with 2 minutes decompression on oxygen, figure 7.

As for Trial A the in-water decompression was carried out in a dry chamber with the diver at rest.

![Graph showing decompression profile](image)

**Figure 7**
Decompression profile for Trial F
3.1.7 Trial G

This trial had a 50 minutes exposure to 36 metres. During the time at maximum depth the diver worked in -- ten minutes on/ten minutes off -- cycles to 65% of maximum heart rate using a bicycle ergometer. The diver wore a dry suit with a woolly-bear undersuit.

Decompression in-water had two stops. The chamber decompression was on oxygen and was 42 minutes. Final decompression to the surface took 2 minutes. The diver was at rest throughout the decompression, figure 8.

![Graph showing pressure changes over time for Trial G](image)

**Figure 8**
Decompression profile for Trial G
3.1.8 Trial H

This trial was 40 minutes at a maximum depth of 72 metres; the diver activity was as for Trial G. The in-water decompression had 8 in-water stops, at 3 metres intervals. The chamber decompression lasted for 114 minutes, oxygen was breathed with air intervals after each 30 minutes. Final ascent to the surface took 2 minutes on oxygen, figure 9. Diver activity and thermal protection was as for Trial G.

![Graph showing decompression profile for Trial H](image)

**Figure 9**
Decompression profile for Trial H
3.1.9 Trial I

This trial was a 30 minute exposure to 63 metres; the diver activity was 5 minutes on/5 minute off cycles at 70% of maximum heart rate. Thermal protection was as in trials G and H. Five in-water stops were used before the surface interval. The chamber decompression lasted 60 minutes on oxygen with air breaks, figure 10.

Figure 10
Decompression profile for Trial I
3.1.10 Trial J

This trial was for 60 minutes at 40 metres. The diver wore a hot water suit whilst in the water and engaged in relatively light activity moving weights and undoing bolts. There is no physiological information which would allow an estimate of the level of activity.

Decompression was carried out partly in the water (the first move) and then in a dry chamber. Oxygen breathing commenced at the third stop. Stops were at 3 metres intervals. Total decompression time was 150 minutes plus a 10 minute ascent on oxygen. Total oxygen breathing time was 60 minutes, figure 11.

![Graph](image.png)

**Figure 11**
Decompression profile for Trial J
3.1.11 Trial K

Diver dress and activity during the time at maximum depth were the same as for Trial J. Maximum depth was 40 metres and duration of exposure was 60 minutes.

The first decompression ascent was carried out in water; thereafter decompression was in a chamber. A 50:50 heliox mixture was breathed from the second stop, at 20 metres, until arrival at 10 metres when the breathing gas was switched to oxygen, figure 12.
3.1.12 Trial L.

Maximum depth was 40 metres with a 90 minute exposure. Diver activity and clothing were as for trials J and K.

The first decompression move was carried out in water; thereafter decompression was in a dry chamber using air, 50:50 heliox and oxygen as the breathing mixtures, figure 13.

---

Figure 13
Decompression profile for Trial L.
3.2 A PRELIMINARY COMPARISON OF THE TRIALS

It is possible to make some comparisons between trials based simply on the exposures, decompression times and level of activity of the divers. For example Trial B might be expected to produce less separated gas than Trial A because the diver was at rest, spent most of the time 3.3 metres shallower than Trial A and was not immersed. Thus inert gas uptake during the time at maximum depth should be less. Calculations of inert gas uptake show that there is slightly less gas in all tissues except muscle where the difference is large. At the end of the bottom time divers in Trial A might be expected to have muscle nitrogen partial pressure 359 kPa compared to 225 for divers in Trial B. This alone would result in less bubble formation on decompression in Trial B.

The divers in Trial D experienced the same conditions as those for Trial A but at a greater depth. The faster tissues were all equilibrated at nitrogen partial pressure 437 kPa and the muscle gas loading was 429 kPa. Thus if this exposure is to result in the same bubble count as Trial A the decompression has to be more effective. The total time from leaving maximum depth to the end of the chamber decompression is 54 minutes for trial A compared to 68 minutes as simulated for trial D. This is 25% extra time to remove a 20% extra gas load which would be sufficient were it not for the effect of bubbles on inert gas washout, i.e. if inert gas uptake and washout were governed by the same physical principles, a condition which has been assumed in almost all decompression profile design since Haldane. As shown in figure 15, section 6.1, this is an erroneous assumption. It should be remembered that Trial D gave trouble in terms of DCI incidents, and that 2 of the 4 divers were decompressed on a more conservative schedule.

Trial F was sufficiently similar to Trial A to give the same inert gas loading but with a total decompression time of only 23 minutes might be expected to give rise to more bubbles.

Trials C and E were assumed to have lower overall muscular activity than Trials A and D because of the intermittent nature of the work carried out, see section 2.5. Thus in trial C the muscle inert gas load will be 241 kPa and this trial might be expected to give less gas in bubbles than Trial A. Despite the fact that this trial gives a similar muscle loading to Trial B it is not possible to make a similar comparison with Trial B because there were also small differences in visceral and coronary gas loading in the resting diver compared to the working diver, muscle is not the only tissue to have inert gas levels influenced by activity.

Trial E, at 45.45 metres, has almost 18% more gas in muscles than Trial C and slightly more than 80% longer decompression time. Once again it is necessary to calculate the effect of bubbles on inert gas washout to predict accurately the effect on bubble numbers.

Trials G, H and L have very similar levels of activity but different hyperbaric exposures. Overall activity level is relatively low because of the intermittent nature of the work. Final muscle inert gas loadings are 293, 480 and 395 kPa, total decompression times 63, 192 and 100 minutes. However there are complicating factors in that the different in-water decompression times, which were undertaken with passive thermal protection, will lead to different degrees of cooling. The in-water decompression times are 14, 72 and 32 minutes. It is therefore difficult to make a simple prediction of the ranking of these 3 profiles.
Trials J, K and L have similar levels of activity but the picture is made complex because of the switch of inert gas during decompressions for K and L. Trial J might be compared with Trial A; there is a very low level of activity but a longer time at 40 metres results in a slightly higher gas loading, 393 kPa compared to 359 kPa in Trial A. Trial J has longer decompression, 162 minutes, but with air breathing for the last 70 minutes. Trials K and L have higher inert gas loadings than e.g. Trial A but have very much longer decompressions, 185 and 250 minutes respectively with very much more oxygen. A simplistic analysis indicates they should result in fewer bubbles than Trial A.

3.3 PHYSIOLOGICAL CHANGES DURING EXERCISE

One of the most difficult aspects of this study was to try to quantify the degree of physical activity undertaken by each diver. The degree of activity has the greatest single influence on the predictions from the model after the influence of the hyperbaric exposure itself. Changes in blood flow which occur in working muscle and in other parts of the body during the physical activity have a direct effect on the time constant of the tissue and therefore on the gas loading during the time spent at the bottom. Figure 14 shows the relationship between muscle blood flow and nitrogen partial pressure in muscle at the end of time spent at 40 metres for a range of bottom times. Blood flow is given as percentage increase over the resting value. It can be seen from this figure that the value chosen for muscle blood flow is critical for inert gas loading especially at low levels of activity and short bottom times.

![Graph showing the relationship between blood flow and nitrogen partial pressure in muscle at the end of time spent at 40 metres for a range of bottom times.](image)

**Figure 14**  
Effect of blood flow changes on muscle inert gas loading
An additional complicating factor is that different types of activity have different effects on blood flow. For example whereas cycling causes an overall increase in flow, even though at some phases of the pedal cycle the muscular contraction actually closes down blood flow through the muscle (Åstrand and Rodahl 1986), weight lifting causes prolonged interruption of flow through the working muscles and a compensatory surge of blood occurs when the muscle contraction is relaxed. This makes it particularly difficult to estimate muscle blood flow for trials C and E in which the activity was weight lifting. Section 2.5 described the effect of changing the assumed values for muscle blood flow for these 2 trials on the outcome of the preliminary study.

A further complicating factor is that at the beginning of a period of activity blood flow takes some time to build up to the level appropriate for the activity. The duration of this approach to steady state depends on several things, most notably on physical fitness of the worker. For light and moderate work the average person will achieve steady state in about 5 minutes when exercising in air but nothing is known about the duration of this build-up when working in water. The effect of immersion on central blood volume, see section 3.5, could change the dynamics of the non-steady state. The delay in approaching steady-state will be of particular consequence when activity is intermittent as in trials C, E, G, H and I.

Because of the uncertainty about comparative levels of physical activity the trials should be grouped according to the type of activity carried out. Thus A, D and F can be compared without too many assumptions, however trial D presents problems because of the 100% incidence of DCI; C and E can be compared; G, H and I can be compared; K and L can be compared; trials B and J stand alone either because of the physical activity undertaken or because of the bubble scoring system used, see section 4.4.

It has to be recommended that future comparative trials of decompression profiles be carried out either with the divers at rest or undertaking some low level whole body exercise of a type which will cause a generalised increase in blood flow without periods of extreme muscle contraction. For trials carried out with divers immersed in water this could be leisurely swimming. If any level of physical activity is used the minimum physiological data which must be recorded continuously is heart rate and minute ventilation. However direct comparison between decompression procedures is best made in trials in which the divers are at rest in a dry environment.

### 3.4 Effect of Temperature

The effect of changes in body temperature are twofold. Changes in temperature anywhere in the body change inert gas solubility and diffusivity. A change in temperature of 1°C causes 1.2% change in nitrogen solubility, an inverse relationship (Allott et al 1993). In addition changes in skin temperature cause changes in skin blood flow, the normal mechanism to maintain thermal equilibrium. Superficial vasodilatation also results from increased body temperature, physical activity at a level of about 50% of maximum oxygen consumption increases muscle temperature by about 2°C (Åstrand and Rodahl 1986) which can lead to a vasodilatation in the skin. These effects are further complicated by a reciprocity between skin blood flow and flow in the underlying muscle (Hensel 1981).

The very complex arrangements for thermal regulation in the body means that comparative trials are best carried out in thermo-neutral conditions.
3.5 EFFECT OF IMMERSION IN WATER

On immersion in water most of the blood which is normally pooled in the venous circulation of the lower parts of the body is returned to the central circulation as the hydrostatic pressure compresses the large veins. This results in an increase in cardiac output of about 30%, resulting almost entirely in increased stroke volume with little change in heart rate (Flook 1987). This changes the blood flow through all the tissues, resulting in a change in time constant and in gas dynamics. More gas is taken up during an hyperbaric exposure in which the diver is immersed compared to an exposure in dry conditions.

3.6 PHYSIOLOGICAL PARAMETER VALUES USED IN THE MODELLING

This section gives details of the physiological conditions used in the simulation of these trials.

Trials A, D and F have been simulated with an increase in skin blood flow because of the hot water suit and an increase in muscle blood flow because of the physical activity. The blood flow per unit volume in compartment six has been increased from 0.66 litres/min to 3.2 litres/min with a corresponding increase in cardiac output and in blood volume in compartment 6. The time constant for compartment 6 has thereby been reduced from 50.6 minutes to 10.7 minutes during work.

Trial B was simulated for rest in thermo-neutral conditions.

Trials C and E were simulated with muscle blood flow increased by 50%, the time constant for compartment 6 was reduced to 42 minutes. These relatively small changes are intended to reflect the intermittent nature of the work, lifting weights, which also only happened in 10:10 minute on:off cycles. Section 2.5 described preliminary work which influenced the final choice of muscle blood flow used for these trials.

Trials G, H and I provided much more information about the work conditions. These were at 65% and 70% of maximum oxygen consumption but in on:off cycles. Reference to text books (Ãstrand and Rodahl 1986, Ruch and Paton 1974) suggest that these levels of activity will result in an oxygen consumption of 1.6 litre/min in average man, cardiac output of 14.3 litres/min, muscle blood flow per unit of tissue 7.2 litres/min and skin blood flow 2.4 litres/min. These changes would result in a 14.5-fold increase in blood flow through compartment 6. However the intermittent nature of the work combined with a possible skin vasoconstriction during periods of no activity (thermal protection being passive) means that overall this level of change would be too high. The changes which have been used are 30% of the changes which would relate to steady state in a thermoneutral environment. This reduction takes account of both the period of time spent with no activity and the period during which blood flow changes were increasing towards the level appropriate for continuous activity.
A further adjustment has been made to take account of the time spent in cold water during decompression. This adjustment has been made to inert gas solubility; there has been no attempt to estimate the appropriate level of vasoconstriction. Trial H has been assumed to result in a 1°C change in average skin temperature (following discussions with Dr Peter Tikuisis at DCIEM) leading to a 1% change in nitrogen solubility. This resulted in a 3% increase in gas in bubbles for trial H. To take account of the different times spent in the cold water on profiles G and I the corresponding increase in the predicted volume of gas in bubbles has been taken as 0.2% for profile G and 0.5% for profile I.

For trials J, K and L the activity has been assumed at a level which would give a heart rate of about 100 beats/minute leading to a muscle blood flow increase of 2 litres/min, skin blood flow increase of 0.3 litres/min and an increase in skin shunt blood of 1 litre to take account of the high surface temperature in the hot water suit. These changes resulted in a time constant for compartment 6 of 11.6 minutes.
4.0 DECOMPRESSION BUBBLES

4.1 DOPPLER SCORES

The data for pulmonary artery bubble numbers generated in each diver taking part in the trials, was provided in the form of scores relating to some form of Doppler bubble detection. Bubble scores were recorded at intervals following return to normobaric conditions at the end of decompression. The interval between recordings was rarely less than 20 minutes and sometimes as high as 50 minutes. This means it is not possible to identify the peak score for comparison with the predicted peak volume of gas carried in bubbles. The comparison can only be made with the maximum recorded bubble score.

Bubble scores were determined under two conditions: at rest, that is following a short period of inactivity as the subject stood whilst recordings were made; after exercise, following movement usually in the form of knee bends. It is considered necessary to make recordings following movement because it is known that bubbles can adhere to the walls of blood vessels. The transient increase in blood flow which results from movement flushes these forward into the central venous system. The acknowledged authority on Doppler bubble recording is the laboratory at the Defence and Civil Institute for Environmental Medicine (DCIEM) in Toronto, Canada. Here a very strict protocol is followed in that following a period of recording at rest the subject is asked to make one knee bend and the resultant flush of bubbles is scored. The subject is then allowed to stand at rest until the Doppler score returns to the resting level before being asked to make a second knee bend. Three such movements are made giving 3 replicate scores. The results are reported as Kisman-Masurel (KM) scores (Kisman and Masurel 1983).
4.2 QUANTIFICATION OF DOPPLER SCORES

Doppler scores are not numerical values and cannot be treated as such because the interval between one score and the next highest is not identical for all such intervals. Neither is the relationship between the intervals known. In order to handle scored data statistically it is necessary that the interval from score I to score II is of the same importance as the interval from score II to score III (Altman 1991). When this condition is not fulfilled it is not possible to determine an average maximum score for a group of divers; neither do either median or mode properly measure the centre for the group. Doppler scores were therefore converted to bubble counts using the relationship described by Brubakk and Eltedal (1994) which is presented in Table 4. This relates the scoring system and bubbles counts derived from a comparison of scores with output from the ultrasonic scanning and counting system in use in the SINTEF Unimed decompression laboratory. The relationship between KM scores and bubble counts is close to being logarithmic if the assumption is made that the KM scoring points are evenly spaced. There is no obvious justification for that assumption but the main conclusion remains: the scoring system is not linear.

<table>
<thead>
<tr>
<th>KM Grade</th>
<th>Bubble Count Bubbles/cm²</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>I-</td>
<td>0.02</td>
</tr>
<tr>
<td>I</td>
<td>0.05</td>
</tr>
<tr>
<td>I+</td>
<td>0.1</td>
</tr>
<tr>
<td>II-</td>
<td>0.2</td>
</tr>
<tr>
<td>II</td>
<td>0.4</td>
</tr>
<tr>
<td>II+</td>
<td>1.0</td>
</tr>
<tr>
<td>III-</td>
<td>2.0</td>
</tr>
<tr>
<td>III</td>
<td>4.0</td>
</tr>
<tr>
<td>III+</td>
<td>8.0</td>
</tr>
<tr>
<td>IV-</td>
<td>12.0</td>
</tr>
<tr>
<td>IV</td>
<td>16.0</td>
</tr>
</tbody>
</table>
For each diver the maximum Doppler score at rest and the maximum following movement were both converted to bubble counts using Table 4. These counts were then used to calculate the average maximum count, at rest and following movement, for all divers in each trial. The average values ± standard deviation are given in Table 5.

**TABLE 5**

Mean bubble count ± standard deviation (number)

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>AT REST</th>
<th>AFTER MOVEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.84 ± 1.22 (12)</td>
<td>4.53 ± 2.99 (9)</td>
</tr>
<tr>
<td>B</td>
<td>0.38 ± 1.02 (18)</td>
<td>2.36 ± 3.78 (4)</td>
</tr>
<tr>
<td>C</td>
<td>0.008 ± 0.02 (6)</td>
<td>0.41 ± 0.89 (5)</td>
</tr>
<tr>
<td>D</td>
<td>2.10 ± 2.2 (4)</td>
<td>7.20 ± 8.02 (4)</td>
</tr>
<tr>
<td>E</td>
<td>0.52 ± 1.21 (9)</td>
<td>4.32 ± 6.75 (9)</td>
</tr>
<tr>
<td>F</td>
<td>3.18 ± 2.41 (8)</td>
<td>8.73 ± 7.03 (6)</td>
</tr>
<tr>
<td>G</td>
<td>0.14 ± 0.43 (23)</td>
<td>0.89 ± 1.98 (23)</td>
</tr>
<tr>
<td>H</td>
<td>1.37 ± 1.78 (21)</td>
<td>2.31 ± 3.23 (21)</td>
</tr>
<tr>
<td>I</td>
<td>1.24 ± 1.74 (20)</td>
<td>1.90 ± 2.29 (20)</td>
</tr>
<tr>
<td>J</td>
<td>0.57 ± 1.40 (22)</td>
<td></td>
</tr>
<tr>
<td>K</td>
<td>1.14 ± 2.18 (53)</td>
<td>2.96 ± 5.25 (40)</td>
</tr>
</tbody>
</table>

**D** Of the 4 divers which began this trial all reported symptoms of DCI and were treated, thus the true maximum bubble score may not have been recorded before treatment was started. In addition, because of the high incidence of DCI, two of the divers were actually decompressed on a more conservative table. This trial should not therefore be considered in the same way as the others.
4.4 DIFFERENCES BETWEEN TRIALS

Because the absolute sensitivity and the detection threshold of the recording system will influence the number of zero scores it is important only to compare trials in which the same systems were used. The trials can then be grouped A, B, D, F, K and L together; C, E, G, H and I together; trial J used a third system. Trials K and L could be regarded as a subgroup because these trials involved a gas switch during decompression and are not comparable in any way with the type of procedures carried out in the other trials. There are gross differences between the Doppler scores of the two main groups which may indicate a different threshold sensitivity of the counting system. In the first group, in trials A, B, D and F no divers scored zero after movement; in the second group all trials resulted in some zero scores after movement, in trials G H and I there were at least half of the divers with zero scores after movement yet in those divers with bubbles the range of grades was as wide as those in the first group.

In the first group either a multifrequency pulsed wave Doppler system was used or the Doppler signal was derived from an ultrasonic scanning system with spatial resolution. Measurements in the second group were made using a standard ultrasonic Doppler probe. There is no information available about comparative threshold sensitivity of these systems but there is no reason to suppose that they will all be the same and therefore the only possible way to handle the data is by grouping it into sub-sets as suggested above.
5.0 PREDICTIONS FROM THE MODEL

The twelve trials have been simulated using the mathematical model described in section 2.0 and the physiological conditions as described in section 3.6. The volume of gas carried as bubbles in the pulmonary artery has been calculated at each time interval throughout the decompression and for a period of time following return to the surface. The peak values for gas carried in pulmonary artery bubbles are listed in Table 6 together with the mean bubble count from Table 5.

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>GAS VOLUME/ML BLOOD</th>
<th>BUBBLE COUNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.00382</td>
<td>4.53 ± 2.99 (9)</td>
</tr>
<tr>
<td>B</td>
<td>0.00213</td>
<td>2.36 ± 3.78 (4)</td>
</tr>
<tr>
<td>C</td>
<td>0.00203</td>
<td>0.41 ± 0.89 (5)</td>
</tr>
<tr>
<td>D</td>
<td>0.00793</td>
<td>7.20 ± 8.02 (4)</td>
</tr>
<tr>
<td>E</td>
<td>0.00280</td>
<td>4.32 ± 6.75 (9)</td>
</tr>
<tr>
<td>F</td>
<td>0.00387</td>
<td>8.7 ± 7.03 (6)</td>
</tr>
<tr>
<td>G</td>
<td>0.00363</td>
<td>0.89 ± 1.98 (23)</td>
</tr>
<tr>
<td>H</td>
<td>0.00489</td>
<td>2.3 ± 3.23 (21)</td>
</tr>
<tr>
<td>I</td>
<td>0.00452</td>
<td>1.90 ± 2.29 (20)</td>
</tr>
<tr>
<td>J</td>
<td>0.00513</td>
<td></td>
</tr>
<tr>
<td>K</td>
<td>0.00122</td>
<td>1.14 ± 2.18 (53)</td>
</tr>
<tr>
<td>L</td>
<td>0.00230</td>
<td>2.96 ± 5.25 (40)</td>
</tr>
</tbody>
</table>

5.1 PREDICTIONS COMPARED TO BUBBLE COUNTS

In comparing the model predictions of peak volume of gas carried in the pulmonary artery with the pulmonary artery bubble counts derived from Doppler data, only the Doppler scores following movement have been considered. As explained earlier it is usual to ask divers to make a movement in order to release any bubbles which may be adhering to the blood vessel walls. The mathematical model does not make any allowance for bubbles being held back in this way; it assumes all intra-vascular bubbles move into the central venous circulation without hindrance, therefore the appropriate comparison is with Doppler grades following movement.
Taken as a complete group of 12 trials the predicted and measured bubbles do not rank in the same order. However, as explained in earlier sections, because of differences in the techniques for recording Doppler scores and because of lack of information about the true physiological status the twelve trials should be sorted into distinct groups. Section 2.5 describes how small changes in the way in which physical activity is simulated can influence the ranking of predicted bubble counts. Taking account of both the uncertainty about relative levels of activity between trials and of possible differences in Doppler sensitivity the trials fall into 4 main groups as follows.

Trials A, D and F were probably evaluated by common Doppler techniques and have similar levels of physical activity (Group 1). Trials K and L (Group 2) had the same Doppler technique as the first group but different level of physical activity and a gas switch during the decompression. Trials G, H and I had the same Doppler evaluation (which differed from that for Groups 1 and 2) but were probably consistent in physiological status (Group 3). Trials C and E (Group 4) go with Group 3 for Doppler evaluation but with a different level of activity. Trials B and J stand alone in that trial B was for a diver at rest and trial J seems to have had a Doppler evaluation which may be different from all others.

The following comparisons are made between predictions and measurements for the trials grouped in this way. In some instances it is possible also to make comparisons between groups and where these are valid they have been made.

5.1.1 Group 1 — A, D and F.

If these trials are ranked in order of increasing predicted gas in bubbles in the pulmonary artery the order is A, F and D. When ranked according to bubble count the order is A, D and F. However as has already been pointed out all divers taking part in Trial D suffered DCI and were recompressed for treatment before the maximum bubble count had been reached; also 2 of the 4 divers were subjected to a more conservative decompression than that modelled.

5.1.2 Group 2 — K and L.

The model predictions order these two trials in agreement with the Doppler bubbles. The Doppler recordings are compatible with Group 1. The model predicts fewer bubbles than for any of the Group 1 trials; both gave fewer Doppler bubbles than the Group 1 trials.

So with the exception of trial D, where true maximum bubble count was not determined, the model ranks all trials in Group 1 and Group 2 correctly.

The effect on bubble growth of exchanging helium for nitrogen will be discussed in section 6.5.

5.1.3 Group 3 — G, H and I.

The model predictions rank these in ascending order as G, I and H. This matches the Doppler bubble ranking.
5.1.4 Group 4 — C and E.

Once again the model predictions and Doppler counts order these two profiles in the same way but the model predicts less difference between them. However the scatter on the bubble data for these trials is too great, and the numbers of subjects too small, to allow any degree of confidence in the evaluation of relative bubble counts.

5.1.5 Trial B

Trial B is effectively trial A without physical activity and could be compared with it. Both Doppler bubbles and model predictions indicate it to be a procedure with lower risk than trial A.

5.1.6 Trial J

This trial stands alone in that it is the only trial for which all the Doppler recordings are known to have been made with the multi-frequency pulsed Doppler system. Unfortunately there is no information about Doppler recordings made after movement.

5.3 EFFECT OF IMMERSION AT MAXIMUM DEPTH.

One condition which can change gas loading, but has not been used in the modelling as presented, is the effect of immersion. It is, however, useful to look at the effect of immersion as it may explain some of the differences between trials or between divers on the same trial. This study was made for trial B, the only trial undertaken without physical activity.

The effect which immersion has on the redistribution of blood is to reduce the time constants of all compartments, section 3.5. This has an effect on gas loading for all compartments which are not fully equilibrated by the end of the bottom time in the dry exposure. These are compartments 5 to 8 of which 6 to 8 have bubbles at the end of the decompression. Time constants for these three compartments under immersed conditions are 39, 53 and 161 minutes compared to those given in Table 1. Gas loading is increased by 110, 111 and 109% respectively. The predicted peak volume of gas in bubbles goes up to 0.00235, an increase of 10.3%, resulting from immersion.

The difference in recorded bubble counts between trials A and B was 92%, trial A with exercise and immersion had the higher bubble count. The difference in predicted gas in bubbles was 79%, 13% more than the difference in bubble counts. Simulations took account only of the exercise in trial A. The simulation of the effect of immersion alone is 10.3% which is very close to the missing 13% difference between Doppler bubbles and predictions.
6.0 GENERAL DISCUSSION

The original intention for this work was to compare the level of decompression stress which the model predicted for all twelve profiles with the level of decompression stress as indicated by the extent of bubble formation determined by Doppler monitoring. The simplest way to make such a comparison is to rank all profiles in the order of decompression stress according to the model prediction and to compare this ranking with that derived from bubble counts. It has not proved possible to do that for several reasons, mainly related to apparent differences in bubble counting procedures and uncertainty about the exact level of physical activity in divers during the time spent at maximum depth. As stated in section 5.2 the level of physical activity has the greatest single influence on the predictions from the model after the influence of the hyperbaric exposure itself.

As a consequence of these uncertainties the trials were grouped as described in section 5.1. Groups 1-4 contain more than one trial and within each group the model predictions rank the trials in the same order as bubble counts. Furthermore where comparisons between groups can be made, group 1 and 2, the model predictions agree with the bubble counts for all 5 trials. Trial B, which has not been included in any group, should give less gas than trial A because of the lower level of activity of the subjects. It does give less gas as determined by bubble counts and the model predicts it to give less gas. That the model does distinguish so clearly between trial A and trial B is an indication of the sensitivity of the model as a means of predicting decompression stress. It should be noted that the range of bubble counts between divers within each trial is so great that Doppler bubbles can not be used to distinguish between these two profiles. The sensitivity of the model is also apparent in that the model has allowed quantification of the differences between trial A and trial B due to immersion, see section 5.3.

Quantification of decompression bubbles is now the method of choice for making an objective measurement of decompression risk. The incidence of DCI is no longer considered an acceptable means of evaluating stress and in most circumstances is so low as to require very large numbers of trials to evaluate it. However there is great variability in bubble numbers both between divers taking part in identical profiles and for a given diver experiencing identical profiles on different occasions. It has to be said that a similar wide range of bubble counts are seen in animal experiments in which breathing gas, decompression profile and animal activity are all closely controlled. It is worthwhile to look at the bubble scores recorded in these trials to demonstrate how difficult it can be to get a clear-cut evaluation of decompression stress from them.

6.1 VARIABILITY IN BUBBLE COUNTS

Trial L gives a clear example of the range of bubble scores which can arise in a group of divers undergoing apparently identical hyperbaric exposure. Of 40 divers 14 were recorded as grade 0, 26 had non-zero grades and these ranged from I to IV*. It is naive to assume that a zero bubble score means that the diver formed no decompression bubbles. The safest assumption is that divers taking part in identical hyperbaric exposures have very similar levels of gas loading and, therefore, of potential to form bubbles. A zero score only means that bubbles were not detected in the mixed venous blood. It is possible that there were bubbles in the pulmonary artery but that they
were below the level of detectability of the system used. It is also possible that there are bubbles somewhere in the body even though none are detected in the mixed venous blood.

The values quoted in Table 5 are the means of all subjects, including divers who recorded zero bubble counts.

There is enough evidence in this report to indicate that one source of the variability in bubble counts between divers, taking part in apparently identical decompressions, may be the level of activity or more correctly the effect of that activity on muscle blood flow. At the levels of activity used in the trials and with the relatively short bottom times used, even small differences in blood flow response to activity during the time at maximum depth will account for quite large differences in bubble numbers and in decompression stress as shown in figure 2.

The variability in detected bubble numbers may be largely a result of the site of measurement, the central venous blood. Pulmonary artery blood is made up from blood returning from all parts of the body. Venous blood from tissues with a sufficient inert gas load to form bubbles will bring a high concentration of nitrogen, blood from tissues with lower inert gas load and no bubbles will contribute less nitrogen. Whether or not bubbles form in the mixed venous blood will depend on the relative contributions of blood with bubbles to blood without. This itself will depend on whether bubbles have formed in the tissue itself or in the vascular system of the tissue.

It is possible that in some tissues in some divers bubbles form preferentially in the tissue rather than in the vascular system of the tissue. Fox and Hugh (1964) have shown that some people form a separated gas phase by cavitation at the aortic valves, such people might more easily form intravascular bubbles, the cavitation having ensured a supply of gas nuclei in the blood. People with fewer intravascular gas nuclei may have a greater proportion of tissue bubbles. These tissue bubbles can only transfer gas to the blood by diffusion, down a partial pressure gradient. The formation of bubbles has the effect of pulling down the partial pressure of inert gas in tissue and venous blood draining that tissue. Thus blood leaving such a tissue, where bubbles have formed in the tissue but not in the blood, will have a relatively low inert gas load and the presence of tissue bubbles will be unrecorded because the volume of gas transported to the central venous system will not reflect the volume of gas in the tissue.

Once tissue bubbles have formed the gas held within these bubbles is then released slowly and venous partial pressure remains below that which can cause intravascular bubbles. This trapping of gas in the tissues is a consequence of the very large gas:tissue partition coefficients for most diving gases. The gas:tissue partition for nitrogen in an aqueous tissue is 68:1 and for helium 109:1.

The driving force for gas to be removed from the body is always the difference between arterial and venous levels. This is true of small blocks of tissue and of the body as a whole. Figure 15 shows the effect which bubble formation has on venous inert gas partial pressure. This shows the arterial nitrogen partial pressure during a staged decompression, inert partial pressure in tissue when bubbles have not been allowed to form and when bubbles form. The bubbles grow following each move. The partial pressure gradient, arterial to venous, which determines gas removal from the tissue, is very much reduced once bubbles have formed.
Thus it is apparent that one source of inter-individual variability could be a different distribution of tissue or vascular bubble formation. One source of difference could be the tendency to produce cavitation at the heart valves. Equally, small local changes in blood flow could result in bubble formation in small blocks of tissue. The great effect which even a small number of bubbles have on the subsequent removal of inert gas will magnify the effect of small differences in tissue bubble numbers.
6.2 EVALUATION OF DECOMPRESSION STRESS FROM BUBBLE NUMBERS

If decompression stress relates at all to decompression bubbles then the more gas that is carried in bubbles the higher the stress. Whether or not decompression stress relates directly to signs and symptoms, DCI, is not an issue appropriate for this report. However some of what will be said below indicates a rather loose link between decompression stress and the incidence of symptoms. The success of statistical techniques such as Maximum Likelihood Analysis in the design of decompression procedures aimed at reducing the incidence of DCI perhaps also indicates that the relationship is not tight.

The only way to properly evaluate decompression stress following an hyperbaric procedure is to evaluate the total volume of gas which is carried in bubbles. For theoretical work, for example using the model described here, this is technically possible though very time consuming and expensive of computing facilities. Most of the profiles which are the subject of this study give rise to gas bubbles over a considerable period of time. If the bubble dynamics calculations are continued until all the bubbles in the body have gone the total time which has to be simulated can extend up to two or three days. For most practical purposes an alternative method of evaluating decompression stress has to be sought. Two options are possible. One would be to evaluate the gas carried as bubbles over a finite period of time, by integrating between time limits the gas in bubbles at each time interval. The second option would be to work with the predicted peak gas volume on the basis that in gross terms at least, higher peak volumes of gas will arise when total gas carried as bubbles is greater. This second option has been used throughout this report and this section gives some of the reasoning behind that decision.

Practical assessments of decompression stress are even more difficult than theoretical assessment. Until recently decompression stress was evaluated in terms of DCI incidence. As a result of the relatively infrequent occurrence of DCI in commercial diving, together with increasing knowledge of the possible long term adverse effects of bubbles, interest has shifted to concern about the number of bubbles generated by a procedure. Although it has long been accepted that DCI is less likely to occur in divers with lower Doppler scores, the data from the trials considered here do not entirely uphold that view.

Decompression symptoms were experienced by divers in trials D (4 of 4), G (2 of 23), K (4 of 53) and L (9 of 40). Ranked in terms of percentage hits, lowest first, these would be K, G, L and D with all other trials coming before K. Ranked in terms of average bubble counts the order is G, K, L and D with trial F exceeding trial D in bubble counts. Of the individual divers with DCI two in Trial D had reached a only grade II before treatment; in Trial G one had zero Doppler bubbles the other had maximum grade III; in Trial K 3 divers had maximum grade II and one maximum grade IV; in Trial L 1 had grade I the remainder having grade III or higher. The data from Doppler scores is of course subject to possible sensitivity differences in counting techniques between the groups.

The difficulties associated with the use of maximum bubble scores have been indicated at various stages in this report. First, because of the intermittent nature of the recordings true peak bubble numbers are not usually ascertained. Secondly, maximum bubble grades which have the kind of distribution as those discussed for trial L, see section 6.1, cannot distinguish unequivocally between more and less stressful procedures. Thus trial A cannot be distinguished from trial B in terms of maximum bubble counts as given in Table 5, neither can trial D be distinguished from trial F though trial D caused symptoms in every diver.
Given sufficient computing capacity it is obviously possible to integrate total gas carried in bubbles over some part of the time for which bubbles exist. It is not so easy to make a proper integration using data derived from intermittent bubble counting. Some of the problems are discussed below.

6.2.1 Inadmissibility of integration of gas volume for time less than the total duration of bubbles.

It is mathematically incorrect to attempt to compare two conditions by integrating an area under a curve unless the whole curve is used or unless the proportion of the curve used for the integrations represents the same proportion of the whole for both conditions.

Figure 16 shows two records of bubble counts, both derived from predictions made by the model. A was predicted for a decompression profile used experimentally, B has been derived from A in a way which changed the shape but not the total area under the curve. Total duration of bubbles is almost the same for both, 159 minutes for A, 165 minutes for B. The integrated area under the whole curve is almost identical 476.8 and 477.6. If integration is carried out over less than the total time for which bubbles exist differences emerge which are not related to the actual curves. The integral from time zero to 140 minutes is 468.6 for A, 440.5 for B. The integrals from 20 to 140 minutes are 425.8 and 403.8. Thus the apparent total gas volume carried as bubbles is less for profile B than for A; in fact both profiles give the same decompression stress.

![Graph showing bubble counts over time](image)

**Figure 16**

Figure to illustrate inaccuracies in integration, see text.
In practice the time course of bubble counts can vary greatly. The speed at which peak bubbles is reached and the rate of decay both depend on which tissues are contributing gas to the bubbles and this depends on the maximum depth and time at that depth as well as on the decompression profile.

Using the same example to consider integration of bubble counts, as distinct from integration of predicted gas in pulmonary artery blood, can lead to even greater errors. The less sensitive the detection system the greater the potential for errors. The line marked with the arrow on figure 16 represents the level of detectability for an imaginary counting system. The two curves A and B, with similar bubble durations in fact, appear to have different bubble durations when bubbles are viewed with this detection system. This leads to further inaccuracies when integration is carried out between fixed times.

Integration of Doppler grades, which are recorded at relatively infrequent intervals, will result in even greater errors. Integration carries the implicit assumption that the value used for integration between 2 fixed points is the value which applies for the whole of the line between these two fixed points. If line A in figure 16 were replaced by Doppler recordings made a 20 minute intervals, say at 20, 40, 60, 80 etc minutes, then carrying out the integration by multiplying by the 20 minute time interval the grade recorded at 40 minutes implies that that grade applies to the whole 20 minutes. In fact the grade would be dropping during the whole 20 minutes. Normally this is handled mathematically by using an average of the value for 40 minutes and that for 60 minutes. However this technique cannot be used for Doppler grades because the interval between one grade and the next is not identical for all intervals, average values have no meaning. (Section 4.2).

The wide range of shape of curves relating gas in bubbles and time, together with the effect of limitations of the bubble detection system mean that integration of gas in bubbles between fixed time limits can lead to even greater errors than shown here. It is a practice which should be discouraged as it can mislead and result in incorrect conclusions about the relative severity of different diving procedures.

6.2.2 The case for using peak bubbles as a measure of decompression stress

Given the probable adequate availability of suitable nuclei on which bubbles can form, see section 2.3; given the fact that once a separated gas phase has formed these relatively small, easily diffusible, gas molecules partition preferentially into the gas phase: some measure of maximum volume of gas in bubbles would seem to be the best assessment of decompression stress. This could be peak volume of gas in bubbles in any tissue or in the body as a whole. In order to relate the predictions from the model to recorded Doppler bubbles the peak volume of gas carried in bubbles in the pulmonary artery is the most useful comparison to make. That this relates to both initial gas loading and the subsequent decompression is shown in the example below in which the model was used to predict bubble formation for three different decompression schedules following identical exposures to pressure (1) and for one shorter exposure (2).
Following a 70 minute exposure to 3.4 bar breathing air, which results in total body inert gas loading at the end of bottom time 2.72 times gas loading in normobaric air, 3 different decompressions were simulated:

a. A rapid decompression taking 2 minutes breathing air.

b. A Sur-D decompression taking a total of 22 minutes with 15 minutes chamber decompression breathing oxygen.

c. A Sur-D decompression taking a total of 38 minutes with 27 minutes chamber decompression breathing oxygen.

Following a 50 minute exposure to 3.4 bar breathing air, giving total body inert gas loading at the end of bottom time 2.5 times gas loading in normobaric air:

a. A rapid decompression taking 2 minutes breathing air.

Table 7 shows the predicted peak pulmonary artery gas volumes derived from the model.

<table>
<thead>
<tr>
<th>Profile</th>
<th>Peak gas in bubbles (ml/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>0.0041</td>
</tr>
<tr>
<td>1b</td>
<td>0.0018</td>
</tr>
<tr>
<td>1c</td>
<td>0.0017</td>
</tr>
<tr>
<td>2a</td>
<td>0.0032</td>
</tr>
</tbody>
</table>

Profile 1 has the same bottom time, same inert gas loading, but a, b and c are in order of increasing decompression time and might therefore be expected to result in lower decompression stress. The predictions show the same ranking; predicted peak gas in bubbles relates correctly to decompression stress.

Profile 2 results in a lower gas loading but having identical decompression to 1a might be expected to produce less gas in bubbles. Predicted peak values show this. In fact the 20 minute reduction in bottom time which gave 22 kPa reduction in gas loading, shows up as 22% reduction in predicted gas in bubbles.

The differentiation between different levels of stress is apparent when peak values are used. This example once again shows how sensitive the model is to small differences in procedure.
6.3 COMPARISON OF PROFILES AT REST

In earlier sections of this report we have shown how difficult it is to determine accurate parameter values for different levels of physical activity. This was a major reason why the trials have had to be grouped for comparison with model predictions. It has been suggested several times in this report that future comparative trials would be better carried out with the divers at rest. It is therefore useful to look at the model predictions for peak gas in bubbles for some of these profiles as if they had been carried out at rest in dry conditions. The results are presented in Table 8 together with \( P\sqrt{t} \) values which give a useful indication of the potential stress arising from the time spent at maximum depth.

For this part of the work the simulation of trial D has been repeated using the correct decompression profile. As this section does not involve comparison with bubble counts it therefore is not influenced by the uncertainty about true maximum bubble counts for trial D and this trial can be compared with the other trials.

Given that inert gas loading and the formation of gas bubbles are both determined largely by physiological and physical factors the order of ranking of a group of decompression profiles should remain the same whether the exposure to maximum pressure is undertaken with the divers at rest or with physical activity provided the level of physical activity is identical for each. It is therefore informative to compare the trials as if all had been undertaken with the divers at rest during the exposure to maximum pressure.

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>GAS VOLUME/ML BLOOD</th>
<th>( P\sqrt{t} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.00218</td>
<td>29.3</td>
</tr>
<tr>
<td>B</td>
<td>0.00213</td>
<td>27.6</td>
</tr>
<tr>
<td>C</td>
<td>0.00160</td>
<td>29.3</td>
</tr>
<tr>
<td>D</td>
<td>0.00330</td>
<td>35.1</td>
</tr>
<tr>
<td>E</td>
<td>0.00280</td>
<td>35.1</td>
</tr>
<tr>
<td>F</td>
<td>0.00292</td>
<td>29.3</td>
</tr>
<tr>
<td>G</td>
<td>0.00235</td>
<td>32.5</td>
</tr>
<tr>
<td>H</td>
<td>0.00421</td>
<td>51.9</td>
</tr>
<tr>
<td>I</td>
<td>0.00251</td>
<td>40.0</td>
</tr>
<tr>
<td>J</td>
<td>0.00226</td>
<td>38.7</td>
</tr>
<tr>
<td>K</td>
<td></td>
<td>38.7</td>
</tr>
<tr>
<td>L</td>
<td></td>
<td>38.7</td>
</tr>
</tbody>
</table>
Some of these figures are worth commenting on. In trial B the divers were at 3.3 metres less than trial A for much of the time at depth; with an identical decompression to trial A, trial B represents a lower stress.

A comparison between trial A and trial C reveals very interesting information. In terms of physiological status, gas loading, total decompression time and time spent on oxygen these two trials should be identical but the predictions indicate a lower stress on trial C. The difference between the two decompressions is in the placing of the 5 minute air break during the chamber decompression: trial A has this just before the final decompression whereas trial C has it immediately on reaching the 12 metre stop with the oxygen breathing to follow. Figure 17 shows the difference between these two decompressions for muscle bubble growth. Figure 17A shows total pressure and muscle nitrogen partial pressure for each trial. For trial C nitrogen partial pressure is dropping throughout except for the 5 minutes of air breathing which comes early in the final chamber stop; for Trial A the increase in nitrogen partial pressure comes just before the last move. Figure 17B shows the bubble radius for both profiles. For trial A the late air breathing causes sufficient increase in nitrogen loading for surface tension forces to be overcome and new bubbles to form after the final move. Trial C the inert gas level does not get sufficiently high: in fact the tissue is protected by the oxygen window. Compartment 7 and 8 are similarly affected by the late air break on profile A. These are the only 3 compartments which bubble on the final move in trial A so the effect of the late air break is marked.

A comparison between trial D and trial E reveals the same effect, of the late air break. However, this should not be taken as an indication that late air breaks always have such a marked effect; in these examples the position of the break relative to the state of the bubbles in muscle is the important determinant. The fact that under some conditions the position of the air break can affect the level of decompression stress does mean that tables should be followed exactly; chamber support staff should have no freedom to move these air breaks to "more convenient" parts of the decompression procedure.
Figure 17
Comparison of tissue nitrogen partial pressure and bubble size in muscle for trial A and trial C
Trial J presents a similar story. The chamber decompression ends with 70 minutes of air breathing at 6 metres i.e. just greater than the depth for complete protection by the oxygen window in average tissue. When the trial was simulated with physical activity at the maximum depth bubbles were still present in muscle when air breathing was started and the nitrogen in the inspired air delayed the removal of nitrogen from the bubbles. This is the main reason why the predicted gas volume for the trial was so high. In the simulation of the trial without activity the muscle gas loading was much less; muscle bubbles were resolved before air breathing started and the inert gas level did not rise enough during the long period of air breathing to regenerate bubbles on the final move. Under these conditions, with no physical activity, the decompression stress of trial J is much closer to that of trial A than was the case when the trials were simulated with activity during time at maximum depth.

6.3.1 Effectiveness of the decompression procedure

In Table 8 trials A, C and F have the same values for $P\sqrt{t}$ but are predicted to have different peak volume of gas in bubbles. The only difference between these trials, when all are compared for standard conditions at rest, is the decompression profile. Profile C is more effective than A which is more effective than F. Ideal, well designed, decompression profiles would result in the same volume of gas carried as bubbles no matter what the $P\sqrt{t}$. The fact that it has been possible in the past to relate DCI incidence to $P\sqrt{t}$ and to reduce DCI by observing an upper limit for $P\sqrt{t}$ in operational diving indicates that we do not yet have decompression procedures which properly take account of all factors contributing to decompression stress.

Combining $P\sqrt{t}$ information with the mathematical model predictions for gas in bubbles after decompression gives a useful means of evaluating the relative success of decompression procedures. Figure 18 shows these data drawn from Table 8 with the addition of an extra point; no exposure to pressure results in no separated gas phase. Those trials which fall below the line of best fit plotted through the data can be taken to have more effective decompressions than average; those above are less effective decompressions. The vertical distance from the line can be taken as a measure of the effectiveness compared to average.

Two things are evident when the profiles are presented in the format of figure 18. The profile which had the highest value for $P\sqrt{t}$, H, had a decompression profile only slightly less effective than average. This explains why it gave relatively little trouble in bubble numbers and DCI despite having such a high $P\sqrt{t}$. On the other hand profile D, with a much lower $P\sqrt{t}$, is seen to be a decompression profile which is worse than most of the others. This profile had 100% incidence of DCI and as noted earlier a true maximum bubble count which may have been the highest of all.
This form of analysis, $\sqrt{P/t}$ against predicted gas in bubbles, could be useful in the design of new profiles. The model could be used to simulate gas in bubbles for existing decompression procedures for a range of values of $\sqrt{P/t}$; the line representing the average could be constructed through these. New profiles which fall above that line could be expected to give more problems when used operationally, those which fall below to give less problems than current practice. The relationship between $\sqrt{P/t}$ and predicted gas in bubbles should hold over a very wide range of $\sqrt{P/t}$ values allowing design of profiles for exposures which are no longer allowed. An alternative approach could be to compare new profiles with the best. In figure 18 this would mean comparing all profiles with trials C and I. If profiles A, B, D, E, F, G, H, J were all redesigned to fall on the same line relating predicted gas in bubbles to $\sqrt{P/t}$ as a line drawn between zero, C and I they should give equally low levels of decompression stress as trials C and I.
6.4 TRIAL D

It has been mentioned several times that trial D was a problem in that it gave 100% DCI rate despite the fact that 2 of the 4 divers were decompressed on a more conservative profile. Figure 18 shows the decompression for trial D to be less effective than the other decompressions.

The most common form of DCI seen with trial D was skin bends and extra simulations have been carried out to try to understand what might be happening in the skin. As described in section 2 it is possible to deal with any of the tissues of the body separately. A separate skin compartment has been run for trials A, D and F. All of these trials were carried out with the diver wearing a hot water suit. The skin temperature was not known but the temperature of the water at supply was 43°C in all trials. Trial J was also carried out with a hot water suit but has not been simulated.

The consequence of increased skin temperature is vasodilation to an unknown extent. This has the effect of reducing the time constant of the skin, thereby increasing the inert gas uptake during the time at maximum pressure. There is no way of knowing what real skin blood flows were. The situation is complicated in that heat induced vasodilation increases not only metabolic, and therefore gas exchanging, flow but also increases shunt flow as the main route for removing heat. The proportion of the total blood flow which serves each category is not known and may indeed be variable to serve the requirements of thermal regulation. A further complication arises in that skin in different parts of the body responds to heat stress with different levels of blood flow increase. The reader is referred to Hensel (1981) for more information.

For the simulations gas exchanging blood flow was taken to increase from 60 ml per minute to 240 ml per minute. Table 9 gives information about the inert gas loading during time at maximum depth and the subsequent peak gas carried as bubbles in skin for trials A, D and F. It should be stressed that in order to make an uncomplicated comparison the divers were assumed to be at rest in air throughout.

| TABLE 9 |

| Inert gas load and gas carried as bubbles for trials using hot water suits |
| Inert gas load | Peak gas in bubbles |
| normal | hot | normal | hot |
| Trial A | 2.34 | 3.49 | 0.00211 | 0.0154 |
| Trial D | 2.74 | 4.15 | 0.00808 | 0.0264 |
| Trial F | 2.34 | 3.49 | 0.01002 | 0.0248 |

From these figures it is quite obvious that when a hot water suit is used trial D gives rise to more skin bubbles than the other trials. Trial J also used a hot water suit, the gas load for this trial is calculated to be 3.90, i.e. less than for trial D. Figure 7 shows trial J to be a more effective decompression and therefore, on both counts, trial J should result in fewer skin bubbles than trial D. Of the 10 trials which did not involve a gas switch trial D is the one most likely to result in skin DCI symptoms.
6.5 GAS SWITCH PROFILES

The model was adapted to handle two gases simultaneously during decompression in order to simulate profiles K and L. It takes account of interaction between the two gases in that helium, diffusing more quickly through tissue into pre-existing nitrogen bubbles, can build up more quickly than nitrogen can leave, thereby generating a change of bubble volume which itself reduces nitrogen partial pressure over and above any change arising from nitrogen movement. This slows down the removal of nitrogen from the bubble. This is described more fully in Burkard and Van Liew (1995) and Van Liew and Burkard (1996). The two gas model also ensures that total mass balance is maintained for each gas. However this was a new development of the model and it is not possible to have the same level of confidence in the predictions as for the other ten profiles.

The gas switch profiles were originally designed in the expectation that helium, having a lower solubility in tissue and blood, would result in less inert gas available for bubbles when it was used in place of nitrogen. This is a simplistic view of the factors which govern bubble formation. Obviously the total volume of gas in the tissues is the absolute maximum that can go into bubbles, but how much goes in depends also on the rate at which the gas can diffuse through the tissue, the gas:blood partition, the rate at which the gas is being removed by the blood and on the precise decompression schedule. At any moment the bubble size is a dynamic equilibrium influenced by all factors.

It should be noted at this point that, because tissue time constants are determined not by the solubility of the gas in the tissue but by the tissue:blood partition, and this is equal to 1.0 for nitrogen and helium for all tissues except fat, the gas switch does not result in a change of time constant for any tissue except fat. It is a common misconception in decompression literature that all tissues have shorter time constants for helium compared to those for nitrogen. Table 10 lists the time constants for the tissues for both helium and nitrogen.

<table>
<thead>
<tr>
<th>COMPT</th>
<th>NITROGEN Time constant</th>
<th>HELIUM Time constant</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.86</td>
<td>0.86</td>
</tr>
<tr>
<td>2</td>
<td>1.87</td>
<td>1.87</td>
</tr>
<tr>
<td>3</td>
<td>3.07</td>
<td>3.07</td>
</tr>
<tr>
<td>4</td>
<td>5.31</td>
<td>5.31</td>
</tr>
<tr>
<td>5</td>
<td>12.25</td>
<td>12.25</td>
</tr>
<tr>
<td>6</td>
<td>50.62</td>
<td>50.62</td>
</tr>
<tr>
<td>7</td>
<td>69.14</td>
<td>69.14</td>
</tr>
<tr>
<td>8</td>
<td>211.3</td>
<td>78.37</td>
</tr>
</tbody>
</table>
Because the two gas model has not been validated in any way it was decided to try to demonstrate the effect of all these inter-relating factors by studying two simple hyperbaric exposures rather than using the more complex profiles used in the trials. Both simple exposures have been simulated for air and for normoxic heliox using the single gas model i.e. no gas switch.

The simplest exposure was 90 minutes at 40 metres with a two minute decompression to surface. Either air or heliox was used throughout until the moment of return to surface when the breathing gas became air. Bubble size and gas volume in bubbles is recorded for the moment of return to surface not at time of peak bubble size. To study the latter would have required using the two gas model for the return to air breathing on the surface and this would have introduced the assumptions inherent in the two gas model.

Table 11 lists, for each compartment, the total concentration of inert gas (gas volume/ml of tissue) in the tissue at the start of the decompression, the bubble radius and the percentage of the initial total volume which is held in the bubbles at the end of decompression.

<table>
<thead>
<tr>
<th>COMPT</th>
<th>TOTAL VOL</th>
<th>RADIUS</th>
<th>% IN BUBBLES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N₂</td>
<td>He</td>
<td>N₂</td>
</tr>
<tr>
<td>1</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0052</td>
</tr>
<tr>
<td>2</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0075</td>
</tr>
<tr>
<td>3</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0083</td>
</tr>
<tr>
<td>4</td>
<td>0.0585</td>
<td>0.0362</td>
<td>0.0089</td>
</tr>
<tr>
<td>5</td>
<td>0.0584</td>
<td>0.0353</td>
<td>0.0093</td>
</tr>
<tr>
<td>6</td>
<td>0.0506</td>
<td>0.0306</td>
<td>0.0080</td>
</tr>
<tr>
<td>7</td>
<td>0.0458</td>
<td>0.0277</td>
<td>0.0071</td>
</tr>
<tr>
<td>8</td>
<td>0.1237</td>
<td>0.0456</td>
<td>0.0078</td>
</tr>
</tbody>
</table>

For all compartments the total volume of inert gas, per unit volume of tissue, at the start of decompression is smaller when the procedure was carried out using helium than for nitrogen, a result of the lower solubility. However the bubbles grow to a larger size on helium because the lower solubility means a higher gas:tissue partition coefficient, a greater tendency for gas to move into bubbles. As a result a greater percentage of the initial total helium load is taken into bubbles compared to nitrogen. It is interesting to note how small the proportion of available gas which goes into bubbles in all tissues, particularly aqueous tissue. It should be stressed that bubble growth continues after return to surface. This would follow a gas switch to air for the heliox procedure so this example makes no comment about the final size of the bubbles.
The decompression in that example was very short, 2 minutes, and therefore the effect of the higher diffusibility of helium would be minimised. In order to study the effect of diffusion a second simulation was carried out. Following 90 minutes at 40 metres decompression was carried out in two stages; following a move to 20 metres in 1 minute there was a 20 minute stop before completion of the decompression in a further minute. The 20 minute stop might be expected to show up the effect of the greater diffusibility of helium. The results, for conditions immediately on reaching surface, are presented in Table 12.

<table>
<thead>
<tr>
<th>COMPT</th>
<th>TOTAL VOL</th>
<th>RADIUS</th>
<th>% IN BUBBLES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N₂</td>
<td>He</td>
<td>N₂</td>
</tr>
<tr>
<td>1</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0036</td>
</tr>
<tr>
<td>2</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0045</td>
</tr>
<tr>
<td>3</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0048</td>
</tr>
<tr>
<td>4</td>
<td>0.0585</td>
<td>0.0363</td>
<td>0.0051</td>
</tr>
<tr>
<td>5</td>
<td>0.0584</td>
<td>0.0363</td>
<td>0.0137</td>
</tr>
<tr>
<td>6</td>
<td>0.0506</td>
<td>0.0315</td>
<td>0.0140</td>
</tr>
<tr>
<td>7</td>
<td>0.0458</td>
<td>0.0285</td>
<td>0.0107</td>
</tr>
<tr>
<td>8</td>
<td>0.1237</td>
<td>0.0445</td>
<td>0.0081</td>
</tr>
</tbody>
</table>

There are several important comparisons to be made between this example and the previous one. The first 4 compartments produce bubbles during the first move which are then resolved during the stop. Thus at the beginning of the final move the inert gas partial pressure is lower and therefore the final bubbles are smaller and represent a smaller percentage of the total gas load. This situation only arises when the duration of the stop is long compared to the time constant of the tissue.

The other four compartments have bubbles throughout the decompression, including the stop, resulting in larger bubbles and a higher percentage of the gas load in bubbles at the surface compared to the no-stop decompression.

The no-stop decompression is compared to the staged decompression in table 13 which shows the ratio, staged decompression:no-stop decompression (derived from tables 11 and 12), for bubble radius and for the percentage of total gas load carried in bubbles. The nitrogen and helium ratios are the same for compartments 1-4, because the bubbles are resolved during the 20 minutes stop the different diffusivities of the two gases has no effect. There are no bubbles at the start of the final move so as far as bubble growth is concerned the staged decompression is effectively the second 20 metre move in 1 minute and does not differ from 40 metres in 2 minutes which was the no-stop decompression.
TABLE 13

Comparison of staged decompression to no-stop decompression

<table>
<thead>
<tr>
<th></th>
<th>RADIUS</th>
<th>% IN BUBBLES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N₂</td>
<td>He</td>
</tr>
<tr>
<td>1</td>
<td>0.69</td>
<td>0.69</td>
</tr>
<tr>
<td>2</td>
<td>0.60</td>
<td>0.60</td>
</tr>
<tr>
<td>3</td>
<td>0.58</td>
<td>0.56</td>
</tr>
<tr>
<td>4</td>
<td>0.57</td>
<td>0.57</td>
</tr>
<tr>
<td>5</td>
<td>1.47</td>
<td>1.39</td>
</tr>
<tr>
<td>6</td>
<td>1.75</td>
<td>1.47</td>
</tr>
<tr>
<td>7</td>
<td>1.51</td>
<td>1.45</td>
</tr>
<tr>
<td>8</td>
<td>1.04</td>
<td>0.98</td>
</tr>
</tbody>
</table>

For the slower compartments the bubbles continue to grow during all or most of the 20 minutes stop. The results in table 13 show that the stop has a slightly greater effect on nitrogen bubbles than on helium. This must result from the different diffusivities of the two gases, all other conditions being the same for both types of bubble at the beginning of the stop.

Thus, despite the lower solubility of helium in tissue and blood, for identical exposures helium bubbles grow larger than do nitrogen bubbles. This is because of the higher gas-tissue partition for helium and is true for no-stop decompressions and for staged decompressions. However for tissues in which bubbles do not resolve during a decompression stop the increase in bubble size due to the presence of the stop is greater for nitrogen than for helium. The relatively lesser effect of the stop on helium bubbles is a result of the greater ease with which helium diffuses through the tissue.

6.6 COMPARISON OF THE PREDICTIONS REPORTED HERE WITH THOSE OF THE IFEM MODEL

The predictions, from the IFEM model which was used in the design of the IFEM tables, was that there should be very few bubbles at the end of the procedure, that all the decompressions should have similar levels of bubble growth and that the risk of DCI should be very small. In the event the results of the trials showed the decompressions to give very much more decompression stress than was predicted by the IFEM model. There are two possible explanations for the failure of the IFEM model; it did not use parameter values drawn from the literature, instead parameter values were set by comparison of the output from the model with DCI incidence from a data base. This approach could only result in design of bubble-free decompression tables if no DCI also
meant no bubbles. This is not a realistic assumption as can be seen from the Doppler data recorded during the trials. Trials A, B, C, E, F, H, I and J gave no DCI but did cause bubbles as shown in Table 5. It could be argued that the numbers of subjects in these trials were too low to show up the incidence of DCI but H I and J had over 20 subjects and trial G with 2 cases of DCI had only 23 subjects.

Average bubble numbers for the IFEM procedures were amongst the highest for the 12 trials which seems to indicate that the IFEM prediction of low bubble numbers has not held. This may be the result of the fact that IFEM decompressions were considered to be low-bubble if the Bubble Growth Index did not exceed a certain value. In other words bubble growth was accepted up to a certain level. It seems from these trials that this level has been set too high. As stressed at an earlier stage in this report a mathematical model can only predict what will happen in the average subject. This means that even if a model predicts no bubbles following a particular decompression procedure some subjects will have bubbles. For example, in terms of the model used in this study, a decompression procedure which keeps the ratio, total dissolved gas pressure to environmental pressure just below 1.0 so that no bubbles will form in the simulation (see section 2.1), will probably result in bubbles in just under half the subjects. The level of bubbling should be low but there will be bubbles. By setting a non-zero acceptable level of bubble growth the IFEM model has failed to take an account of the fact that some subjects, probably half, will exceed the so called acceptable level.
7.0 CONCLUSIONS

One of the most difficult aspects of this study was to try to quantify the level of physical activity undertaken by each diver. This work has shown that, after the decompression profile itself, the single most important determinant of decompression stress, as indicated by the volume of gas carried in bubbles, is the level of activity of the divers during exposure to maximum pressure. This is true for relatively low levels of activity, such as those used in the trials presented here, and for relatively short bottom times. If the level of activity is higher and/or the bottom time longer, muscle will saturate with inert gas and the effect of activity becomes constant. The problems of guessing the level of activity involved in each trial, together with the indication that there may be differences in threshold of bubble detection and in the way Doppler recordings were carried out, has meant that the original plan to deal with all 12 profiles as a group has had to be modified. The trials have been grouped so that those with identical bubble counting techniques and similar work patterns have been grouped together.

Once this is done there is good agreement between the Flook model predictions for peak volume of gas carried as bubbles and maximum precordial bubble counts averaged for all divers in the trial. The predictions have identified the decompression profile D as the most stressful of those studied and this is in agreement with experience in that in trial D all divers suffered DCI and were recompressed for treatment before maximum bubble scores were attained.

The model has clearly demonstrated that the occurrence of skin bends in the divers on trial D would be predicted from both gas load and subsequent decompression profile. The cause of this high predicted incidence of skin bends is the vasodilatation simulated to match the effects of a hot water suit.

This study has shown that the model can be used with some confidence to determine relative decompression stress for any type of decompression profiles. The range of exposures for which it can be used is limitless because the parameter values used in the model have been evaluated over a much wider range of conditions than would be encountered in diving. Thus this gives the opportunity for designing new profiles across a wide range of exposure extending beyond any currently in use.

The model used in this study has come much closer to agreement with the Doppler findings for IFEM profiles than did the predictions from the IFEM model. This is probably because this model sets no threshold for bubble formation. The assumption that no DCI means no bubbles effectively sets a threshold for bubble formation. Because a model can only predict what might happen in the average subject even a prediction of no bubbles can mean bubbles in a small proportion of subjects. Thus by setting a acceptable level of bubble growth the IFEM model has accepted a rather high level of bubble formation.

Of particular note is the success of the model in the evaluation of gas switch profiles in which helium is used to replace nitrogen during decompression. In particular it has shown that, because of the multiple factors which influence bubble growth, gas switch profiles can not be designed by giving consideration to only one or two of the physical properties of the gases. Thus profiles which were expected to result in low decompression stress because the lower solubility of helium means less is available for bubbles, were not successful in reducing bubble numbers. This is study has shown this to be because bubble size is determined by gas/tissue partition, which is the inverse of solubility. The proportion of available gas which goes into bubbles is shown to be a very small proportion of that available within the tissue, particularly in aqueous tissues.
A consequence of this study is a recommendation that in future any trials of new decompression profiles should be carried out first with the divers at rest in thermo-neutral conditions and with a direct comparison, under the same conditions, with profiles which are already accepted for use. If this is done it is safe to assume that a new profile which has been proved less stressful than the existing profiles under these conditions will also be less stressful under any conditions. Should it be impossible to plan trials in which the divers are at rest then the model could be used to help to design the trials and to identify the factors which will have greatest influence on the outcome of the trials.

This study has shown that the physiological model used here can be used to compare the relative levels of decompression stress of new decompression procedures and it is recommended that it should be used prior to human trials. The model could be used with equal confidence to design new decompression procedures over a wide range of hyperbaric exposures. The physiological basis of the model makes it possible to define critical tissues in any exposure and to design decompression to keep bubbles in those tissues to a minimum. However it must always be stressed that the model in the standard format simulates conditions in the average diver. It is possible to design for a subject other than the average but this would require physiological data to be available.
REFERENCES


Bruhak AO, Efstedal O. A system for quantifying gas bubbles after decompression. SINTEF Unimed. (no report number).


James PB, Lafferty CF. Heliox and oxygen in decompression from air dives to 40 metres. OTO 93010, 1993.


Lambertsen CJ. Doppler monitoring in the Gulf of Mexico. Health and Safety Executive OTO 96024 1996.


Robertson DH, Simpson ME. OSD sponsored research towards safer decompression. Health and Safety Executive. OTO 96053, 1996.


