



Excursion tables in saturation diving - decompression implications of current UK practice

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Excursion tables in saturation diving - decompression implications of current UK practice

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Unimed Scientific Limited has undertaken, on behalf of UK Health and Safety Executive, a study of the decompression risks of saturation diving with particular reference to the risks which arise from depth changes during the work shifts, excursions. Decompression safety has increased over the decades for which this type of diving has been used in the North Sea, and the risk of decompression sickness to the individual is low. As in any industry, further improvements can be made and this work has shown possible options for improvement.

Decompression bubble formation has been evaluated using a mathematical model of decompression validated by comparison with Doppler bubble scores and DCI incidence during both trials and operational hyperbaric conditions.

The main conclusion from the work is that neither excursions alone, as currently used, nor the decompression procedures alone, are likely to cause decompression problems. The risks result from the combination of excursions followed by decompression before the bubbles have totally resolved. Starting decompression whilst bubbles are present is the single significant risk factor. This finding explains the limited success of earlier attempts to reduce risk by either reducing the allowed excursion depths or prolonging the decompression from saturation.

A secondary conclusion is that by taking an approach which considers excursions and decompression together further reductions in risk could be achieved. The reductions could be considerable and, from the work reported here, it seems likely that these improvements could be made with an increase in operational efficiency.

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EXECUTIVE SUMMARY

Unimed Scientific Limited was asked by UK Health and Safety Executive to evaluate the risk of decompression bubble formation arising from the use of excursions to depths greater or less than the living depth during heliox saturation diving. Decompression illness is not unknown following decompression from saturation exposures despite what appear to be conservative decompressions. One of the factors which can contribute to the extent of decompression bubble formation is depth changes during the course of bottom time.

Decompression safety has increased over the decades for which this type of diving has been used in the North Sea. As in any industry, further improvements can be made and this work has shown possible options for improvement, but the possibility to reduce risk does not imply that current levels of risk are dangerous to the individual. The level of risk described in this report is the level currently found acceptable; the fact that this study has been made indicates an intention to reduce risk further. The hyperbaric exposures dealt with in this report represent current practise.

The calculations were made using the USL model of decompression. This model has been validated by comparison of the predictions made by the model, of the volume of gas carried as bubbles, with precordial Doppler scores recorded in trials and under operational conditions, for a wide range of hyperbaric exposures used in diving and compressed air (CA) work. It has also been validated by comparison with the incidence of decompression illness symptoms (DCI) for compressed air exposures using the HSE database which contains results from about half million exposures collected over several decades. This comparison is especially relevant to the current work as the duration of CA exposures is similar to the duration of excursions.

USL has details of the allowed procedures for several diving companies covering both the UK and Norwegian sector of the North Sea. For each saturation depth considered, the excursions to be studied were selected in the following way:

- the maximum excursion for the company which allowed the greatest depth change,
- the special case maximum depth change allowed by a second company,
- the standard maximum depth case for that company,
- the maximum depth change allowed in the Norwegian sector.

This gave a good spread of allowed exposures for inclusion in the study.

The work reports bubbles formation following excursions, bubble decay between excursions and the effect of the final decompression on bubble formation.

All aspects of operational saturation diving have been considered. These include;

both upward and downward excursions,

the effect of different rates of depth change

the effect of different hold times between the last excursion and the final decompression

The predictions for bubble formation following each exposure are presented in several forms;

the volume of gas which will form bubbles in the central venous blood

the percentage of divers who may be expected to form bubbles somewhere in the body,

the volume of gas which will form into bubbles in the brain tissue,

the percentage of divers who will form bubbles in the brain.

The main conclusion from the work is that there are bubbles formed on excursions but that neither the excursions currently used nor the decompression procedures are likely to cause decompression problems. The risks result from the combination of excursions followed by decompression before bubbles have totally resolved. Starting decompression whilst bubbles are present is the single significant factor.

This is a significant finding because over the years excursions have been seen as the main cause of problems and as a result depth changes have been restricted. Similarly over the years changes have been made to decompression schedules, in general slowing down the rate of change of pressure in an attempt to reduce the DCI rate, without reference to the fact that the trouble is more likely to have originated with the preceding excursions. This study has shown that excursions and decompression must be considered together. This introduces the possibility that by considering excursions and decompressions together changes can be made which will reduce risk without increasing operational costs. Indeed preliminary work suggests that the improvements could be made with a considerable increase in operational efficiency.

1.0 INTRODUCTION

Unimed Scientific Limited has been asked by UK Health and Safety Executive to evaluate the risk of decompression bubble formation arising from the use of excursions to depths greater or less than the living depth during heliox saturation diving.

Decompression illness is not unknown following decompression from saturation exposures despite what appear to be conservative decompressions. One of the factors which can contribute to the extent of decompression bubble formation is depth changes during the course of bottom time. Jacobsen et al (1997) reported on the relationship between the incidence of decompression illness (DCI) and the pressure profile of 2,622 saturation dives in the Norwegian sector between 1983 and 1990. They concluded that there was a positive relationship between DCI rate and the number of depth changes during the saturation period.

A common form of depth change is an excursion, to allow divers to work at depths different from the storage depth of the living chambers. This allows greater flexibility and excursions can be a cost effective move in that the divers are allowed to work at a greater depth than main storage without the increased decompression time which would be required from the greater depth. An upward excursion allows divers to carry out work at shallower depths without subjecting the whole dive crew to a pressure reduction. Excursions are usually for 6 to 8 hour and as such excursions represent a significant change of gas load in all tissues of the body. A 6 hour excursion to a depth 15 metres deeper than storage depth will increase gas load by the same amount as a 6 hour dive to 15 metres from surface. Nobody would consider doing a no-stop decompression from 6 hours at 15 metres whereas that is what is done at the end of a 6 hour 15 metres excursion.

Most diving companies define limits to the depth changes which can be undertaken as an excursion and the thinking behind the limits is based on Boyle's Law, that it is not the pressure change *per se* which determines the magnitude of bubbles growth but the relative pressure change. Lesser bubble growth for a given pressure change if the overall depth is greater. This approach presupposes the formation of decompression bubbles on all excursions.

The present study has been carried out in order to look at the likely risk of decompression bubble formation related to excursions, to evaluate the extent of bubble growth and to look at the effect on the bubbles of the subsequent decompression from saturation.

This report is concerned with the risk to health and safety of divers carrying out saturation heliox dives. Decompression safety has increased over the decades for which this type of diving has been used in the North Sea. As in any industry, further improvements can be made and this work has shown possible options for improvement, but the possibility to reduce risk does not imply that current levels of risk are dangerous to the individual. The level of risk described in this report is the level currently found acceptable; the fact that this study has been made indicates an intention to reduce risk further. The hyperbaric exposures dealt with in this report represent current practise.

2.0 CALCULATIONS

The calculations were made using the USL model of decompression which has been described fully elsewhere (Flook 2004). This model has been validated by comparison of the predictions made by the model, of the volume of gas carried as bubbles, with precordial Doppler scores recorded in trials and under operational conditions, for a wide range of hyperbaric exposures used in diving and compressed air (CA) work. It has also been validated by comparison with the incidence of decompression illness symptoms (DCI) for compressed air exposures using the HSE database which contains results from about half million exposures collected over several decades. This comparison is especially relevant to the current work as the duration of CA exposures is similar to the duration of excursions.

The model uses an iterative procedure to solve numerically the equations which track gas movement, both in solution in the tissues and blood and as a separated gas phase in bubbles, throughout the whole of an exposure from compression to the end of decompression and for several hours after the end of decompression. The time increments used for the iterations in the current study range from 0.1 minute for the decompression from excursions to 1 minute for the decompressions from saturation. The individual tissues of the body are grouped together, forming eight groups each defined by the time constant for inert gas movement. At each iteration the model determines the total volume of gas, that carried in the bubbles plus the volume remaining in solution, in the tissues and the blood draining those tissues. The volume of gas carried as bubbles in each compartment, or group of tissues, provides the means to compare different decompressions. The volume of gas in the compartments is also combined, using a weighted mean of the 8 compartments, to give the volume of gas carried as bubbles in the central venous, pulmonary artery (PA), blood. This value, in addition to being used to compare decompressions, relates to the Doppler precordial bubbles score and to the DCI incidence. Thus the model makes predictions which can be used with reference to individual tissues and with reference to whole body risk.

Obviously the model uses values for tissue size and blood flow which describe the average person. Therefore all predictions are for the average result which might be expected for a group of individuals undergoing identical exposures. This means that 50% of individuals will have the predicted level of gas or more in bubbles and 50% will have the predicted level or less. Working with the average is a useful approach because any hyperbaric exposure which is safer on average will also be safer for the individual. However the level of risk to the individual can not be predicted in the absence of measurements of tissue blood flow in that individual during the exposure.

It is possible to make some predictions for the extreme ends of the physiological range, for the individual who is far from average. For most physiological and anatomical parameters 99.9% of individuals are within $\pm 20\%$ of the average value. The model has been used to calculate the outcome for an individual at the extreme end of the range, in the direction which would lead to greater decompression bubble formation, for a decompression from saturation and the implications of this are considered in this report.

2.1 EXCURSIONS STUDIED

Time constraints made it necessary to select excursions for inclusion in the study. USL has details of the allowed procedures for several diving companies covering both the UK and Norwegian sector of the North Sea. For each saturation depth considered, the excursions to be studied were selected in the following way:

the maximum excursion for the company which allowed the greatest depth change,

the special case maximum depth change allowed by a second company,

the standard maximum depth case for that company,

the maximum depth change allowed in the Norwegian sector.

This gave a good spread of allowed exposures, as shown in Table 1. Only at one saturation depth did the maximum excursion allowed in Norwegian waters differ from the standard already selected.

Table 1 includes both upward and downward excursions; one company has lower maximum depth changes allowed for upward excursions. Depth changes which are only allowed in the downward direction are marked * and followed (in brackets) by the corresponding depth change for upward excursions.

TABLE 1 Saturation and excursion depths,
upward and downward excursions (see text)

Saturation depth	Excursion Depth			
180 msw	38* (34)	30	15	13
150 msw	35* (31)	26	13	
120 msw	31* (28)	24	12	
90 msw	26* (25)	20	10	
60 msw	23* (19)	18	9	
30 msw	17* (14)	12*	6	

All excursions were assumed to last a maximum time of 8 hours.

Between the companies there is a range of allowed rates of return to living pressure, from 18 msw/min to 10 msw/min, both these rates were used in the calculations. An additional slower rate of 5 msw/min was also included as USL has experience that this slower rate will be beneficial in reducing bubble formation in the brain.

There is also, between the companies, a small range of oxygen levels allowed during excursions. Because of the effect of the oxygen carriage by haemoglobin this difference in inspired oxygen becomes insignificant at tissue level so a single value for inspired oxygen was used for the excursions, this was 0.7 ATA.

2.2 DECOMPRESSION FROM LIVING DEPTH

There are differences in decompression procedures between the companies. Mathematical simulation of a decompression from saturation can use well over a quarter million iterative calculations and therefore it was not possible, within the constraints of the study, to follow more than one procedure. The alternative decompressions from 180 msw were compared and the procedure which gave the shortest time was used. The same company's procedures were used for decompressions from other depths. The lowest oxygen levels allowed at the saturation depth and during decompression were chosen. The differences between those used by different companies is insignificant in terms of decompression bubble formation. The combination of fastest decompression and lowest oxygen should mean that the worst case was simulated.

The decompression profile used was 1.5 msw/hour to 15 msw, thereafter 0.5 msw in every 50 minutes with a 4 hour hold in every 24 hours. Inspired oxygen during saturation was taken as 0.35 ATA and during decompression as 0.5 ATA with the appropriate adjustment closer to the surface.

According to model predictions this procedure will not cause bubbles in the average diver but may cause bubbles in divers at the extreme end of the normal range. The significance of this is dealt with where appropriate in this report.

2.3 SIGNIFICANCE OF THE PREDICTIONS

As mentioned above it is possible to interpret the model predictions in terms of precordial Doppler scores and of likely incidence of DCI. The UK Health and Safety Executive, at an international workshop organised by USL under the sponsorship of the HSE, accepted the recommendations of the assembled scientists that Doppler scores higher than Grade 2 were connected with a greatly increased risk of DCI.(Simpson 1999). From the calibration of the model predictions against measured Doppler scores, Grade 2 corresponds to a central venous (PA) gas in bubbles of 4 μ l/ml.

Figure 1 shows the relationship between model predictions and DCI rate drawn from the compressed air data base. The two completely different ways of validating the model coincide, and fit with the recommendations of the workshop. Doppler Grade 2 corresponds to 4 μ l/ml which is shown on the figure to be the point at which the DCI incidence begins to be a measurable quantity based on a half million exposures in the CA database.

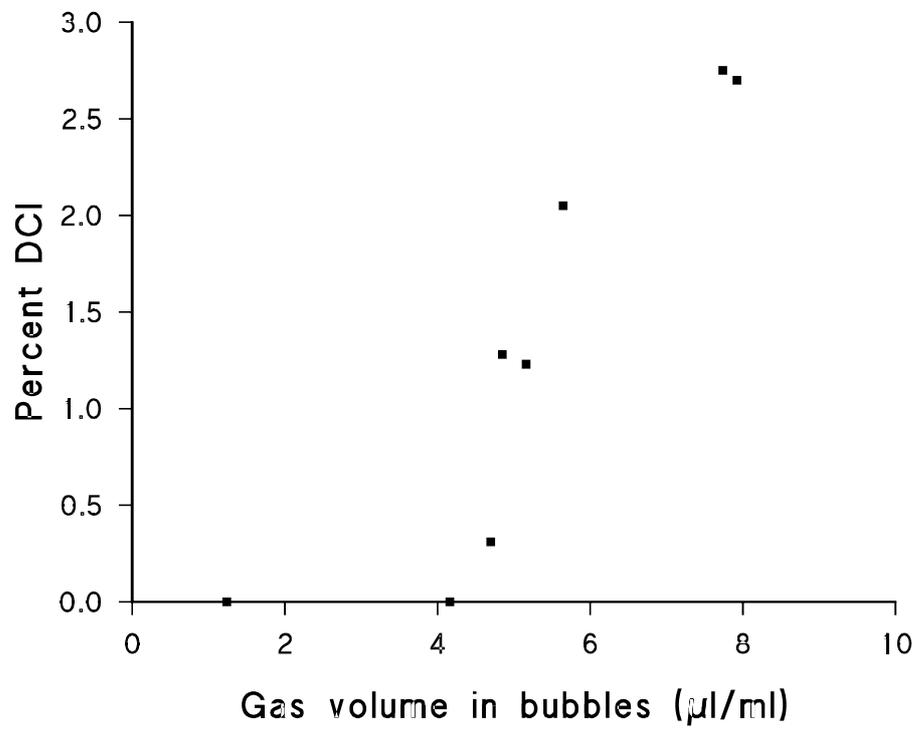


Figure 1 Relationship between model predictions and known DCI rate

3.0 DOWNWARD EXCURSION RESULTS

As described above, the model can be used to determine predicted bubble levels in the whole body, presented as gas in bubbles in pulmonary artery (PA) blood, and also to look at the risk for individual tissues. From experience it is known that the tissue most at risk during pressure reduction at the rates considered here, is the brain. The predictions relating to downward excursions are therefore presented both for the pulmonary artery gas and the brain.

3.1 PULMONARY ARTERY GAS

Table 2 shows the predicted maximum value for gas in bubbles in the pulmonary artery for all excursions using 10 msw/min as the rate of return. The predictions are the average for all divers and are given as volume of gas carried as bubbles in each ml of central venous blood, $\mu\text{l/ml}$. Taking $\pm 20\%$ as the normal physiological variation it is possible to calculate the percentage of individuals who would have some bubbles somewhere in the body and these figures are given in brackets in the table.

TABLE 2 Predicted PA gas ($\mu\text{l/ml}$) and percentage who will bubble

Saturation depth	Excursion Depth			
	38 msw	30 msw	15 msw	13 msw
180 msw	0.43 (99)	0.31 (95)	0.10 (63)	0.07 (56)
150 msw	0.47 (99)	0.30 (93)	0.09 (56)	
120 msw	0.59 (100)	0.33 (95)	0.09 (56)	
90 msw	0.50 (99)	0.33 (93)	0.00 (44)	
60 msw	0.61 (100)	0.45 (96)	0.00 (37)	
30 msw	0.67 (99)	0.33 (69)	0.00 (5)	

It must be remembered that the predictions are the average for all divers; where the average gas in bubbles is 0.0 µl/ml fewer than 50% of divers will have bubbles, as shown by the number in brackets.

Reference to section 2.3 shows that these levels of bubble formation are low and should not cause DCI symptoms in the average diver. The level below which bubbles would be undetectable in most divers using either Doppler or ultrasonic scanning is about 2 µl/ml.

The columns correspond each to a single source for the excursion depths; column 2 relates to the maximum allowed by one company, column 3 to special exposures for another company and column 4 to the standard exposures. This is of interest because it might be expected that any single company would use, as maximum excursions, depth changes chosen to give a similar level of risk; another company might be expected to design to a different level of risk. The different levels of bubble formation between columns is clear but there is no evidence that there is constant risk within any column. For example the allowed excursions from 60 msw give a greater level of bubble formation than those for other depths in both columns 2 and 3.

3.1.1 Effect of rate of ascent

Three rates of ascent were studied, 18, 10 and 5 msw/min. Table 3 shows the range of predicted bubbles for each excursion, the fastest rate of ascent giving the most bubbles. The ranges are small, different rates of ascent have relatively little effect.

TABLE 3 The effect of ascent rate on predicted PA gas in bubbles (µl/ml)

Saturation depth	Excursion Depth			
	38 msw	30 msw	15 msw	13 msw
180 msw	0.439 - 0.423	0.312 - 0.303	0.098 - 0.096	0.071 - 0.063
150 msw	35 msw	26 msw	13 msw	
	0.472 - 0.454	0.303 - 0.296	0.087 - 0.085	
120 msw	31 msw	24 msw	12 msw	
	0.593 - 0.569	0.335 - 0.328	0.090 - 0.088	
90 msw	26 msw	20 msw	10 msw	
	0.508 - 0.489	0.332 - 0.326	0.000	
60 msw	23 msw	18 msw	9 msw	
	0.618 - 0.596	0.448 - 0.440	0.000	
30 msw	17 msw	12 msw	6 msw	
	0.674 - 0.661	0.332 - 0.324	0.000	

3.2 GAS IN BUBBLES IN BRAIN TISSUE

Table 4 shows the predicted volume of gas carried as bubbles ($\mu\text{l/ml}$) in brain tissue and venous blood draining the brain together with the percentage of divers who would have bubbles in the brain. For most excursions most divers will form bubbles in the brain tissue on return to living depth.

TABLE 4 Gas in bubbles ($\mu\text{l/ml}$) in brain and percentage with bubbles

Saturation depth	Excursion Depth			
	38 msw	30 msw	15 msw	13 msw
180 msw	0.112 (90)	0.065 (84)	0.008 (56)	0.004 (50)
150 msw	0.120 (93)	0.059 (84)	0.006 (50)	
120 msw	0.164 (96)	0.066 (84)	0.005 (50)	
90 msw	0.121 (93)	0.059 (84)	0.000 (38)	
60 msw	0.150 (90)	0.088 (87)	0.000 (32)	
30 msw	0.146 (90)	0.036 (50)	0.000 (4)	

Model predictions of the volume of brain gas in bubbles are not easily compared with any known measurements. Neuropathologists have some experience of the extent to which the contents of the skull can increase in volume before death is the inevitable outcome. A figure of 4%, 4 ml per 100 ml, 40 $\mu\text{l/ml}$, is sometimes quoted as the limit. This compares well with a death from decompression bubbles which was not preventable by recompression treatment, following an explosive decompression which the model predicted to give 35.5 $\mu\text{l/ml}$ for gas in bubbles in the brain. Trials of military diving, for which there is a great deal of Doppler data and information about the DCI rate, used procedures predicted by the model to give a maximum volume of gas carried as bubbles in the brain of 11.6 $\mu\text{l/ml}$, so this can be taken as a level which can be survived. No CNS DCI cases were reported in those trials. These number can be used to put the results in Table 4 into some kind of perspective.

3.2.1 Effect of rate of ascent

Table 5 shows the effect of the three ascent rates on brain bubbles. The range is shown, the fastest rate causing the highest volume of gas in bubbles. The benefits of slowing the ascent are obvious. Taking an extra few minutes over the return reduces the brain gas to 75% or less.

Although the volumes shown in Table 4 would appear to carry no risk it must be remembered that they are for the average. It must also be remembered that brain blood flow is very labile and the increased levels of oxygen during an excursion may cause local transient reductions in flow during which that portion of the brain will not be able to offload gas and bubbles may form. Once formed they will not resolve when blood flow returns to normal. Given the few minutes which the slower return would add it should always be good practice to give the brain this extra protection.

TABLE 5 The effect of ascent rate on brain gas ($\mu\text{l/ml}$)

Saturation depth	Excursion Depth			
	38 msw	30 msw	15 msw	13 msw
180 msw	0.122 - 0.090	0.071 - 0.053	0.009 - 0.006	0.005 - 0.003
150 msw	35 msw	26 msw	13 msw	
	0.131 - 0.097	0.066 - 0.049	0.006 - 0.004	
120 msw	31 msw	24 msw	12 msw	
	0.179 - 0.132	0.073 - 0.054	0.006 - 0.000	
90 msw	26 msw	20 msw	10 msw	
	0.133 - 0.099	0.065 - 0.048	0.000	
60 msw	23 msw	18 msw	9 msw	
	0.167 - 0.123	0.097 - 0.071	0.000	
30 msw	17 msw	12 msw	6 msw	
	0.162 - 0.116	0.042 - 0.000	0.000	

Figure 2 compares the effect of the different ascent rates on pulmonary artery gas (dotted line)

and brain (solid line) for the ascent from a 38 msw excursion from living depth of 180 msw.

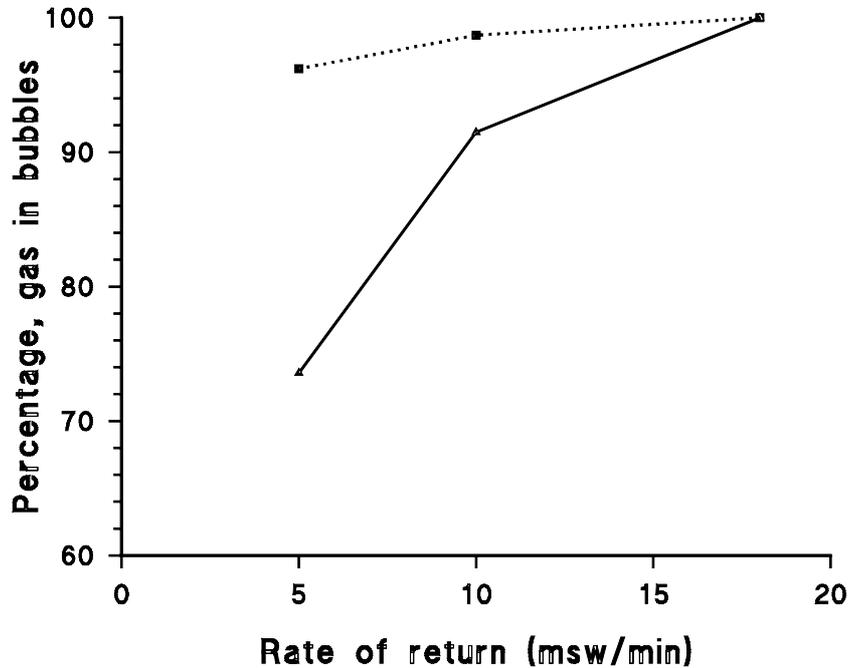


Figure 2 Effect of ascent rate on bubbles in pulmonary artery blood (dotted line) and in brain (solid line)

3.3 EFFECT OF DURATION OF HOLD

The diving companies all specify a period of time for which the divers, following a downward excursion, should remain at the living pressure before the start of decompression. The intention is to allow any bubbles formed during the return to be resolved. Table 6 shows the amount of gas carried as bubbles in the pulmonary artery blood at the end of holds of 6, 12 or 24 hours, expressed as a percentage of the volume immediately on return to living depth. An empty cell in the table indicates an excursion which produced no bubbles. The reduction in bubbles, even from a 6 hour hold, is valuable but only after the mildest of excursions are the bubbles resolved during a hold as long as 24 hours.

The operational procedures on the duration of this hold were written long before it was realised how long bubbles could last.

TABLE 6 Percentage of original bubbles remaining at the end of the hold

Saturation Depth	Excursion	Duration of hold (hours)		
		6	12	24
180	38	69.5%	43.9%	8.3%
180	30	26.5%	17.8%	1.62%
180	15	13.4%	0%	0%
120	31	29.1%	19.7%	6.32%
120	24	24.1%	12.0%	0%
120	12	1.1%	0%	0%
60	23	51.5%	10.8%	0%
60	18	37.5%	4.0%	

3.4 EFFECT OF REPEAT EXCURSIONS

Divers are expected to do a work shift each day which means that excursions are repeated with approximately 16 hours between each. Any bubbles remaining from the previous excursion would be compressed on the return to the deeper depth and the gas leaving the bubbles would add to the gas load in solution. When the previous excursion has lasted 8 hours and the second excursion is to the same depth, for the same time, the gas load at the end of the second excursion, in all except the slowest tissues, is the same as that for the first excursion because most tissues are saturated by an 8 hour excursion. The exception is the slowest tissue, the fat. The first 8 hour excursion brings this tissue to 99.81% saturation whereas the addition of the remaining gas load to the uptake of the second excursion brings it to 99.96% saturation. This results in an increase in gas in bubbles, following the second excursion, of less than 0.02%

The difference between first and second excursions, the risk of build up of gas as the number of excursions increase, would be greater than quoted above if the excursion duration were less than 8 hours. However the gas levels can only increase to saturation so the gas in bubbles will not go above the values quoted in this report by more than 0.02%.

3.5 EFFECT OF FINAL DECOMPRESSION

In section 2.2 it was stated that in the average diver the decompression from saturation will not cause bubbles to form. From section 2.3 it is apparent that the bubbles formed following excursions, as quoted in the tables above, do not reach levels in the average diver which might

cause DCI. Cases of DCI do occur in saturation diving and according to Jacobsen et al (1997) the incidence relates to the number of depth changes undertaken by the individual.

Table 6 shows that gas bubbles can continue for over 24 hours after many excursions and therefore the decompression is likely to be started with bubbles present. These bubbles will simply expand as the pressure is reduced no matter how small they were at the start of the decompression. Whether or not the bubbles persist throughout the decompression is dependent on the rate at which the blood flow through the tissue can remove the gas from the bubbles compared to the rate of change of pressure. If the blood flow is not sufficient to remove enough gas to limit the volume increase as the pressure falls then bubbles will grow throughout the whole decompression.

The effect of the final decompression has been studied for three depths, 180, 120 and 60 msw. The decompression has been started following holds of 6, 12 or 24 hours and the return from the excursion was carried out at 10 msw/min. Table 7 gives the maximum volume of gas in bubbles ($\mu\text{l/ml}$) in the central venous blood. For some combinations of excursion and decompression the maximum gas in bubbles occurs on return to surface, for others it occurs earlier as the rate of removal of inert gas by the blood surpasses the effect of the pressure decrease. In Table 7 the * indicates where the maximum volume of gas in bubbles occurs before the end of decompression. For these results the value given in brackets is the depth (msw) at which the maximum occurs. Blank cells indicate both excursions which produced no bubbles and excursions for which the bubbles were resolved during the hold prior to the start of decompression.

TABLE 7 Maximum PA gas in bubbles ($\mu\text{l/ml}$) after decompression (see text)

Saturation Depth	Excursion	Duration of hold (hours)		
		6	12	24
180	38	3.14	2.24	1.41
180	30	2.10	1.68	1.29* (9)
180	15	1.37* (9)		
120	31	1.44	1.39* (9)	1.0* (12.9)
120	24	1.19* (12.6)	1.01* (12.9)	
120	12	0.80* (13.2)		
60	23	0.95* (13.4)	0.54* (13.5)	
60	18	0.59* (13.5)	0.41* (13.8)	

Some of the volume of gas carried as bubbles given in Table 7 reach levels which will give

Doppler detectable bubbles in most divers and may cause symptoms in some divers. A few divers with slower inert gas removal than the average might well grow bubbles to 4 $\mu\text{l/ml}$ following the 38 msw excursion from 180 msw. At that level, given the random element in the occurrence of symptoms, 1 in 1000 of these divers might have symptoms.

The maximum depth at which the peak bubbles occur is 13.8 msw which fits very well with the fact that symptoms occur usually towards the end of the decompression.

It is interesting to look at what happens to the gas formed following the 23 msw excursion from 60 msw. This had almost the greatest volume of bubbles (Table 2) 0.61 $\mu\text{l/ml}$. The volume was reduced during the 6 hour hold by about 50% leaving a volume almost exactly equal to that following the 6 hour hold after the 38 msw excursion at 180 msw. The smaller decompression from 60 msw increased the volume to 0.95 $\mu\text{l/ml}$ compared to the increase on the 180 msw decompression, to 3.14 $\mu\text{l/ml}$.

Figure 3 shows the growth and decay of bubbles for an exposure for which the peak bubble volume occurs before reaching the surface. The decompression profile, from 120 msw, is shown as the dashed line. The excursion was to 24 msw with a 6 hour hold prior to decompression.

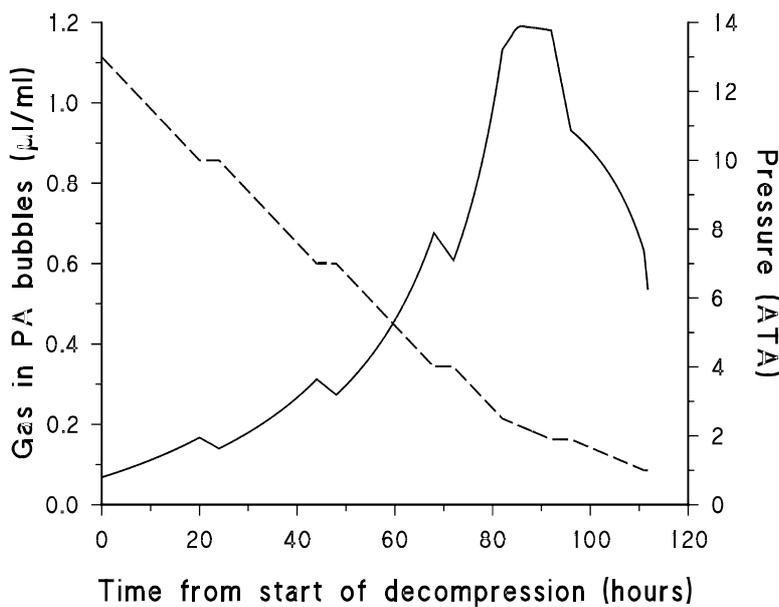


Figure 3 Growth and decay of excursion bubbles during final decompression

Table 8 presents the results in Table 7 expressed as a percentage of the volume of gas which

was in bubbles at the end of the excursion. This gives a very clear picture of the magnification of existing gas when decompression is started before bubbles are resolved. Obviously the general rule must be that the greater the pressure change required for final decompression the bigger the increase in bubbles left over from the excursion.

TABLE 8 Amplification of bubbles during the final decompression

Saturation Depth	Excursion	Duration of hold (hours)		
		6	12	24
180	38	725%	518%	325%
180	30	678%	544%	417%
180	15	1407%		
120	31	246%	237%	171%
120	24	359%	307%	
120	12	898%		
60	23	155%	89%	
60	18	132%	93%	

This leads to the conclusion that less deep excursions should be allowed the deeper the saturation depth. If the decompression amplification of bubbles is to be greater, because decompression is from a greater depth, then it would be well for the gas in bubbles resulting from the excursion to be less. This is completely the opposite to the thinking behind the current practice, however it would only apply to the last excursion undertaken by a diver. One way in which the final excursions could be reduced in operational practice would be for the living depth to be increased during the final 24 hours to minimise the bubbles formation during the relatively rapid return from excursion. This would increase the total decompression time but that will be set against the alternative option which would be to increase the duration of the pre-decompression hold.

3.6 BUBBLE GROWTH IN THE NON-AVERAGE DIVER

Section 2.2 deals with the concept of the average result and introduces the fact that not all divers will have average physiology. The main interest to the study of decompression, must be the diver who has lower than average blood flow in part or all of his body. A lower blood flow means a slower removal of dissolved gas and more gas left to form bubbles. The effect of this on decompression from saturation is of particular interest because that decompression has the major influence on final outcome.

Decompression from 120 msw has been simulated for a diver at the outer extent of the normal range, 20% below the average. Statistically this would account for 1 in a 1000 divers, assuming divers to be representative of the population as a whole. This diver would form bubbles during the decompression even though he had not undertaken any excursions. The maximum gas in bubbles in the central venous blood for this diver is predicted to be 3.1 $\mu\text{l/ml}$, detectable by Doppler. If we assume that of a 1000 such divers only 1 will develop symptoms we are left with an estimated DCI rate of 1 in a million for decompression from 120 msw, having done no excursions and following the decompression procedures used in this study.

Decompression from greater depths would result in more gas in bubbles for this diver, decompression from a smaller pressure would give less gas in bubbles.

However if that diver carried out an excursion and started the decompression with his excursion bubble load the situation becomes much worse. To the 3.1 $\mu\text{l/ml}$ resulting from the decompression would have to be added at least the value given in Table 7 for his excursion. If the excursion had been to 24 msw and the hold 6 hours then the total gas in bubbles on surface would be at least 4.3 $\mu\text{l/ml}$. Of course, that 1 in 1000 diver would also grow bigger bubbles following the excursion. Thus it becomes possible to see how the incidence of DCI found in saturation diving in the North Sea can arise.

Intermittent reduction of blood flow

One more special case has been studied; should a diver experience a period of reduced blood flow whilst the pressure is dropping, bubbles would form during that time and though these would grow during the subsequent decompression they would grow less rapidly once the blood flow was restored. A possible scenario for this would be a diver sleeping very soundly for several hours with his softer fat layers compressed on a mattress or with a limb bent so that some flesh is compressed. Normally the resultant reduction in blood flow would cause pain and wake the diver. This example could be particularly relevant to the situation of an injured diver under sedation.

Figure 4 shows what might happen in such a case. The decompression profile is shown as the dashed line. The upper curve is the 1 in a 1000 diver who is at the extreme edge of the normal range and has reduced flow throughout the decompression. The lower curve is the diver who has suffered a short term reduction in flow. The benefit of the restored flow results in a lower final volume of gas in bubbles, 1.1 $\mu\text{l}/\text{ml}$ and, as the figure shows, the bubbles are decaying by the time surface is reached and would resolve within a few hours.

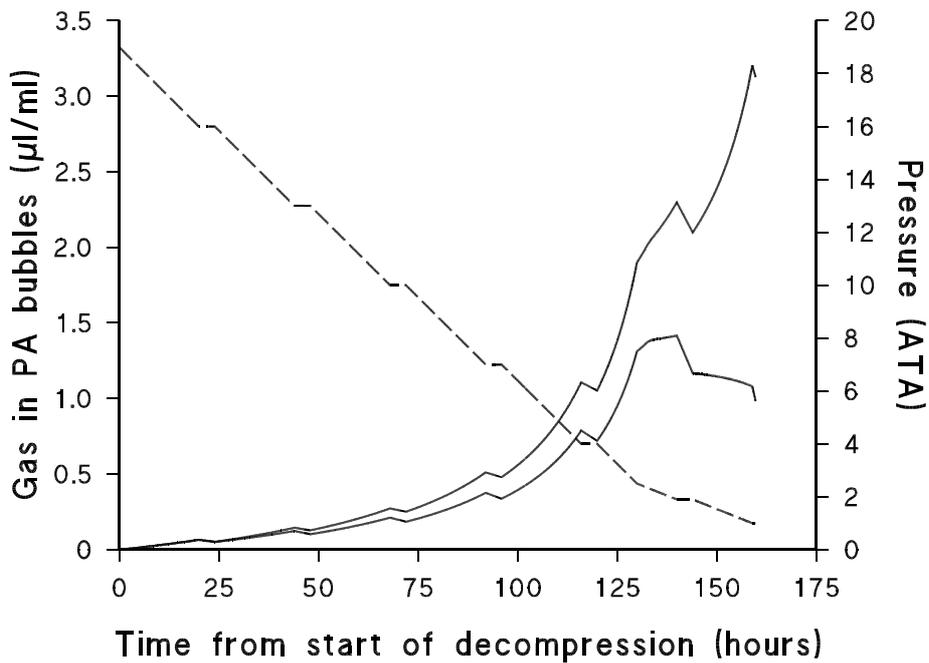


Figure 4 Bubble growth in divers with reduced blood flow (see text for details)

4.0 UPWARD EXCURSIONS

Bubble formation on an upward excursion affects the diver during the work shift and could influence performance of tasks and attention to safety. Return to the living pressure will cause the bubbles to resolve and, provided the living depth has not been reduced during the excursion, the gas made available from the bubbles will not cause an inappropriate gas loading. Thus the concern about upward excursions relates only to the duration of the excursion.

4.1 PULMONARY ARTERY GAS

Table 9 shows the maximum volume of gas carried as bubbles in the pulmonary artery blood during the upward excursions simulated in this study, depth change at 10 msw/min. As for Table 2 the values refer to averages for all divers and the percentage who would have bubbles somewhere in the body is given in brackets. Most divers will form bubbles during most upward excursions. The duration of the bubbles might be expected to be as for the return from downward excursions so that for an 8 hour excursion bubble decay will be something between the 6 and 12 hour hold given in Table 6. For most excursions bubbles will persist until return to living pressure.

TABLE 9 Predicted PA gas ($\mu\text{l/ml}$) and percentage who will bubble

Saturation depth	Excursion Depth			
	34 msw	30 msw	15 msw	13 msw
180 msw	0.43 (99)	0.35 (96)	0.09 (62)	0.07 (56)
150 msw	0.46 (99)	0.34 (96)	0.08 (62)	
120 msw	0.51 (100)	0.38 (97)	0.08 (56)	
90 msw	0.60 (100)	0.39 (96)	0.00 (44)	
60 msw	0.58 (99)	0.52 (98)	0.00 (32)	
30 msw	0.70 (95)		0.00 (3)	

4.1.1 Effect of rate of ascent

Table 10 shows the range of gas volume in bubbles for the three ascent rates ranging from 18 msw/min to 5 msw/min. As with downward excursions the ascent rate does not have a very large effect on central venous bubble formation.

TABLE 10 The effect of ascent rate on predicted PA gas in bubbles ($\mu\text{l/ml}$)

Saturation depth	Excursion Depth			
	34 msw	30 msw	15 msw	13 msw
180 msw	0.433 - 0.424	0.347 - 0.345	0.096 - 0.094	0.066 - 0.065
	31 msw	26 msw	13 msw	
150 msw	0.460 - 0.456	0.342 - 0.338	0.082 - 0.081	
	28 msw	24 msw	12 msw	
120 msw	0.512 - 0.506	0.390 - 0.384	0.085 - 0.084	
	25 msw	20 msw	10 msw	
90 msw	0.602 - 0.594	0.391 - 0.387	0.000	
	19 msw	18 msw	9 msw	
60 msw	0.583 - 0.576	0.520 - 0.513	0.000	
	14 msw		6 msw	
30 msw	0.711 - 0.694		0.000	

4.2 GAS IN BUBBLES IN BRAIN TISSUE

Table 11 shows the predicted volume of gas carried as bubbles ($\mu\text{l/ml}$) in brain tissue and venous blood draining the brain, together with the percentage of divers who will have bubbles in the brain. Again most divers will form bubbles in brain tissue during most excursions.

TABLE 11 Gas in bubbles ($\mu\text{l/ml}$) in brain and percentage with bubbles

Saturation depth	Excursion Depth			
180 msw	34 msw	30 msw	15 msw	13 msw
	0.087 (93)	0.061 (87)	0.005 (63)	0.002 (50)
150 msw	31 msw	26 msw	13 msw	
	0.091 (93)	0.054 (87)	0.003 (56)	
120 msw	28 msw	24 msw	12 msw	
	0.100 (95)	0.062 (90)	0.003 (50)	
90 msw	25 msw	20 msw	10 msw	
	0.117 (96)	0.055 (87)	0.000 (38)	
60 msw	19 msw	18 msw	9 msw	
	0.093 (93)	0.075 (90)	0.000 (26)	
30 msw	14 msw		6 msw	
	0.110 (79)		0.000 (2)	

The work on downward excursions showed that, on average, bubbles in the brain decay within a 6 hour hold at constant pressure. It might be expected therefore that by the end of an 8 hour upward excursion most divers will have resolved brain bubbles though those in the slower tissues will remain until compression on return to living pressure.

Table 12 shows the effect of the different ascent rates on the formation of brain bubbles. As in the case of downward excursions the effect of rate of ascent is much more marked for bubble formation in the brain than in the body as a whole.

The amount of gas which forms bubbles in the brain is in general less for upward excursions than for the equivalent downward excursion but the difference is very small.

TABLE 12 The effect of ascent rate on brain gas ($\mu\text{l/ml}$)

Saturation depth	Excursion Depth			
180 msw	34 msw	30 msw	15 msw	13 msw
	0.095 - 0.075	0.065 - 0.053	0.006 - 0.004	0.003 - 0.001
150 msw	31 msw	26 msw	13 msw	
	0.098 - 0.079	0.058 - 0.047	0.004 - 0.002	
120 msw	28 msw	24 msw	12 msw	
	0.108 - 0.086	0.067 - 0.053	0.003 - 0.000	
90 msw	25 msw	20 msw	10 msw	
	0.126 - 0.100	0.060 - 0.045	0.000	
60 msw	19 msw	18 msw	9 msw	
	0.101 - 0.076	0.082 - 0.060	0.000	
30 msw	14 msw		6 msw	
	0.123 - 0.082		0.000	

5.0 CONCLUSIONS

The results from this study show the relationship between the magnitude of the excursion, the pressure from which the excursion starts and the volume of gas which is predicted to form into bubbles. The numbers given are meaningless until they are set into context. For the industry this means relating the numbers to the incidence of DCI; for those concerned with the possible damage to health of symptomless bubbles this means relating the numbers to Doppler bubbles scores. USL has attempted to give perspective to the predictions by drawing on experience gathered over recent years in which model predictions have been compared to measurements; to Doppler scores and to DCI rate for different types of hyperbaric exposure.

The mathematical analysis results in the conclusion that the excursions studied, though causing bubbles, are unlikely to cause either detectable bubbles or DCI in the average diver and that detectable bubbles might occur at a level of a few divers in a thousand. The results also show that decompression from saturation is unlikely to cause any bubbles in the average diver though something like 1 in a 1000 may have detectable bubbles. DCI is likely to be a rare event following either an excursion or a decompression from saturation. The risk increases considerably when an excursion is followed by decompression; when a decompression is preceded by an excursion.

This is a significant finding because over the years excursions have been seen as the main cause of problems and as a result depth changes have been restricted. Similarly over the years changes have been made to decompression schedules, in general slowing down the rate of change of pressure in an attempt to reduce the DCI rate, without reference to the fact that the trouble is more likely to have originated with the preceding excursions. This study has shown that excursions and decompression must be considered together. This introduces the possibility that by considering excursions and decompressions together changes can be made which will reduce risk without increasing operational costs.

Already several possibilities become apparent:

risk could be reduced by increasing the time for which divers are held at constant pressure before beginning decompression, the extra time being offset by a reduction in the time spent on decompression;

risk could be reduced by taking account of the amplification of bubbles by the decompression and working so that during the last 24 hours before decompression the deeper the living depth the smaller the excursion, changing living depth to make that possible without adding time to the final decompression;

the ideal would be to start the decompression with no bubbles remaining from excursions. If this were so there would be no justification at all for slowing decompression closer to the surface. This would mean using an appropriate constant decompression rate, slow enough to prevent bubbles formation in any tissue. The result would be a considerable reduction in total decompression time.

Following through on this approach could result in a considerable reduction in risk combined with simpler and more cost effective operations.

The main conclusion from the work is that neither the excursions currently used nor the decompression procedures are likely to cause decompression problems. The risks result from the combination of excursions followed by decompression before bubbles have totally resolved. Starting decompression whilst bubbles are present is the single significant factor.

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