An effect of CO_2 on the maximum safe direct decompression to 1 bar from oxygen-nitrogen saturation[1]

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Bell PY, Harrison JR, Page K, Summerfield M. An effect of CO_2 on the maximum safe direct decompression to 1 bar from oxygen-nitrogen saturation. Undersea Biomed Res 1986; 13(4):443–455.—An investigation into the maximum safe decompression step from oxygen nitrogen saturation to 1 bar was carried out with and without the presence of 0.02 bar carbon dioxide. The series, Islander 1, involved 13 teams of 5, fully informed, male volunteers carrying out simulated dives. One group of 6 teams carried out dives in an atmosphere of 0.4 bar oxygen, balance nitrogen (O_2-N_2); another group of 7 teams used an atmosphere of 0.38 bar oxygen, 0.02 bar carbon dioxide, balance nitrogen (O_2-N_2-CO_2). The dives consisted of a 48-h stay at 1.7 or 1.8 bar to saturate the tissues, followed by decompression to 1 bar air at 0.5 bar/min. Two decompression parameters were studied; the incidence of decompression sickness (DCS) in the 24 h postdecompression, and the incidence and grade of venous gas emboli (VGE) in the first 6 h postdecompression. The grade of VGE was assessed using the Kisman-Masurel scoring system which produces a bubble grade with the subject at rest and after movement. No significant difference was found in the incidence of DCS between the two groups. Twenty subjects were decompressed from 1.7 bar using each mixture, without signs or symptoms of DCS. However, after decompression from 1.8 bar there were 2 cases of DCS in 10 subjects in the O_2-N_2 group and 2 cases in 15 subjects in the O_2-N_2-CO_2 group. The incidence of detectable VGE was always lower in the O_2-N_2-CO_2 group at both saturation pressures; at 1.7 bar the VGE incidence was lower by 40% ($P < 0.05$) at rest and by 55% ($P < 0.001$) after movement. At 1.8 bar the reduction was 3% (NS) at rest and 30% (NS) after movement. The results indicate that decompression from 1.8 bar to 1 bar, with or without the presence of 0.02 bar carbon dioxide, is likely to produce more than 5% DCS.

carbon dioxide
decompression
nitrogen
saturation
submarine rescue
ultrasound
United Kingdom

Since Paul Bert (1) demonstrated that released gas was the cause of "caisson disease," considerable work has been carried out to determine the rules for the safe

return to 1 bar of men working in compressed gases. Despite this work, remarkably little data are available concerning the magnitude of the maximum direct decompression to 1 bar that will not produce decompression sickness (DCS) following a prolonged exposure to increased air pressure.

Boycott et al. (2) proposed that a pressure reduction ratio of 2:1 in inert gas was optimal, i.e., a reduction from 2 to 1 bar would be the maximum safe decompression step. Subsequent experience with caisson workers has indicated that DCS can occur even though the ratio does not exceed 2:1. Several small and poorly documented studies have investigated pressures between 1.7 and 2.0 bar (3, 4). Le Fur et al. (5) commented that in the Galathee experiment, carried out in 1979, they had determined that 1.6 bar was the maximum decompression step to 1 bar that would not produce venous gas emboli. They did not however report the incidence of DCS during their studies.

The size of the minimum decompression step likely to produce DCS is of great importance to survivors of a submarine accident. It is possible that they may have been exposed to pressures between 1 and 5 bar for an extended period before a rescue can be carried out. The rescue may involve a transfer under pressure in the Deep Submergence Rescue Vehicle or similar craft from the pressurized escape compartment of the disabled submarine to a mother submarine or surface craft. The transferred survivors would then be decompressed to 1 bar. In addition, because of the need to optimize the efficiency and duration of the carbon dioxide absorbent used to maintain a breathable atmosphere, the survivors will have been exposed to an elevated inspired carbon dioxide pressure, around 0.020–0.025 bar, before the rescue. The documented effects of such an inspired carbon dioxide level on any subsequent decompression are contradictory, ranging from no significant effect (6, 7) in aviators, to significant increases in DCS (8, 9) in caisson workers.

In this study, Islander 1, we attempted to determine the maximum safe decompression step to 1 bar from oxygen-nitrogen saturation with and without the presence of 0.02 bar carbon dioxide in the saturation gas mixture.

METHODS

Subjects

The subjects for these exposures were volunteer submariners with no previous diving experience other than the routine submarine escape training. All subjects were briefed in accordance with the Declaration of Helsinki on nontherapeutic clinical research, and an independent medical officer was available at all times during each trial. The protocol had been approved by the Ministry of Defence (Navy) Underwater Personnel Research Advisory Committee. The experiment was carried out in 2 phases and the anthropometric details obtained from the subjects used during each phase are summarized in Table 1. The values obtained from a study of a group of Royal Navy submariners are shown for comparison (Smith DJ, personal communication).

Procedures

The experiment was carried out in 2 phases. In phase 1, 5 subjects were compressed to either 1.7 or 1.8 bar using O_2-N_2 (P_{O_2} = 0.4 bar); and in phase 2, 5 subjects were

TABLE 1
ANTHROPOMETRIC DATA

		Phase 1	
		Volunteers (n = 30)	Royal Navy Submariner Study (n = 82)
Age	(yr)	28.6 ± 5.78	28.0
Weight	(kg)	77.4 ± 9.92	77.0
Height	(m)	1.76 ± 0.06	1.75
Total skinfold	(mm)	47.12 ± 18.5	
Percent body fat		18.61 ± 4.52	20.7
Lean body mass	(kg)	62.69 ± 6.11	60.8
Body surface area	(m^2)	1.93 ± 0.13	
FEV_1	(1)	3.98 ± 0.47	
FVC	(1)	4.77 ± 0.62	
		Phase 2	
		Volunteers (n = 35)	Royal Navy Submariner Study (n = 82)
Age	(yr)	27.5 ± 5.20	28.0
Weight	(kg)	75.0 ± 9.50	77.0
Height	(m)	1.78 ± 0.06	1.75
Total skinfold	(mm)	35.36 ± 12.3	
Percent body fat		15.2 ± 4.58	20.7
Lean body mass	(kg)	63.26 ± 5.98	60.8
Body surface area	(m^2)	1.92 ± 0.13	
FEV_1	(1)	4.00 ± 0.46	
FVC	(1)	5.08 ± 0.64	

Figures are shown ± 1 SD. Body surface area was measured by the method proposed by Dubois and Dubois (10) and the percent body fat was estimated using the method suggested by Durnin and Womersley (11).

compressed to 1.7 or 1.8 bar using O_2-N_2-CO_2 (P_{O_2} = 0.38 bar, P_{CO_2} = 0.02 bar). The subjects were held at the chosen pressure for 48 h and then decompressed at a rate of 0.5 to 1.0 bar/min. During decompression the chamber was flushed with air to reduce the high level of oxygen and/or carbon dioxide to near ambient levels. The subjects were then held in the chamber at exactly 1.0 bar for 24 h and were observed for signs and reported symptoms of DCS. (The need to hold the subjects at exactly 1 bar is outlined below.) If the subjects were clear of DCS after the observation period, the chamber pressure was equalized with the atmospheric pressure and the subjects released. The pressure profile is shown in Fig. 1.

Routine monitoring of the atmospheric pressure had revealed that it was possible that the atmospheric pressure could change by as much as 50 millibars (0.05 bar) over

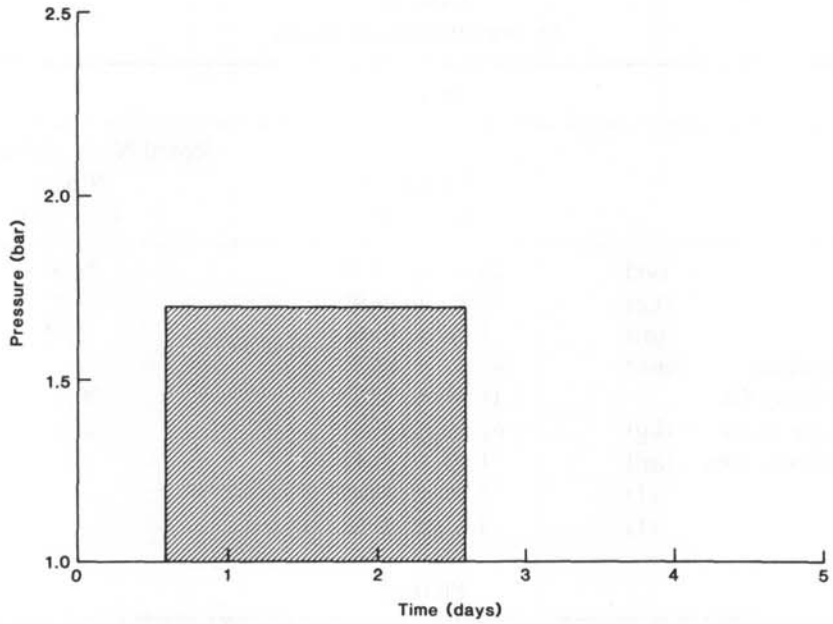

Fig. 1. Pressure profile used throughout the Islander 1 series. Compression rate was 1.5 bars/min and decompression was completed within 2 min.

a period of 3 d. Inasmuch as a change of this size could seriously affect the size of the decompression step at the end of the saturation phase it was decided that the effect of changes in atmospheric pressure should be removed if possible. Thus, in 2 of the early experiments the compression was carried out on top of the atmospheric pressure giving saturation pressures of 1.72 and 1.694 bar. The subjects were subsequently decompressed to 1.02 and 0.994 bar, respectively, giving a constant decompression step size of 0.7 bar. The successful maintenance of these low positive or negative pressures allowed us to change the routine to require compression to exact pressures and decompression to 1.0 bar regardless of atmospheric pressure.

The details of the pressures and composition of the gases in the 2 phases of the experiment are given in Tables 2*a,b*. The ratio describing the magnitude of the change in inert gas tension during decompression is also given in Table 2*a,b* as P_{N_2}/P_{N_2}sat (P_{N_2} divided by the P_{N_2} during the saturation period).

Decompression sickness monitoring

The safety of decompression was determined by the number of reported DCS symptoms that required treatment as assessed by the independent medical officer. The protocol governing the conduct of the series is given below and in Fig. 2.

a. Two experimental runs to be carried out at the required pressure, initially 1.7 bar.
b. A pressure to be deemed "safe" if 10 subjects (2 experimental runs) completed the profile at the chosen pressure with no signs or symptoms of DCS.
c. If the "safe" pressure criteria are satisfied, the experiment to be repeated with the saturation pressure increased by 0.1 bar.

TABLE 2a,b
SUMMARY OF PRESSURES AND P_{N_2} Values

Phase 1

Saturation			Holding			
Pressure	P_{N_2}	SD	Pressure	P_{N_2}	SD	$\Delta P_{N_2}/P_{N_2}$sat
1.720	1.299	0.006	1.020	0.784	0.006	0.396
1.694	1.284	0.007	0.994	0.760	0.009	0.408
1.800	1.384	0.010	1.000	0.767	0.012	0.446
1.800	1.369	0.006	1.000	0.783	0.005	0.428
1.700	1.273	0.007	1.000	0.785	0.008	0.383
1.700	1.276	0.006	1.000	0.775	0.004	0.393

Phase 2

Saturation					Holding			
Pressure	P_{N_2}	SD	P_{CO_2}	SD	Pressure	P_{N_2}	SD	$\Delta P_{N_2}/P_{N_2}$sat
1.700	1.289	0.010	0.0187	0.001	1.000	0.779	0.004	0.396
1.700	1.300	0.003	0.0195	0.001	1.000	0.779	0.003	0.401
1.800	1.395	0.003	0.0196	0.002	1.000	0.779	0.012	0.442
1.800	1.387	0.008	0.0192	0.002	1.000	0.777	0.003	0.440
1.800	1.380	0.005	0.0201	0.001	1.000	0.777	0.002	0.437
1.700	1.289	0.005	0.0191	0.002	1.000	0.779	0.004	0.396
1.700	1.288	0.005	0.0196	0.002	1.000	0.780	0.004	0.394

Values are the mean ± 1 SD of 48 samples during saturation period and 24 samples during holding period. All pressures are shown in bar.

d. A pressure to be deemed "unsafe" if 2 or more cases of DCS occurred within 20 subjects (4 experimental runs).
e. A pressure to be deemed "the maximum safe pressure" if:
   1) only 1 case of DCS occurred in 20 subjects (1 incident in 4 experimental runs);
   2) no DCS occurred in 20 subjects (4 experimental runs) following a decrease in the saturation pressure of 0.1 bar because the unsafe pressure criteria had been fulfilled.
f. If 1 case of DCS occurred within the first 2 experimental runs, 2 more experimental runs were carried out at the same pressure. On detection of a 2nd case of DCS the saturation pressure was decreased by 0.1 bar until the maximum safe pressure criteria were fulfilled.

The Sodelec ultrasonic bubble detector (DUG) was used in this study to monitor the production of venous gas emboli (VGE) in the right ventricle. The precordial monitoring was carried out at 30-min intervals during the first 4 h following the direct decompression to 1 bar and again after 6 h. The recorded signal was later scored according to the Kisman-Masurel (KM) code (12). This system produces two scores, one representing the VGE grade with the subject standing at rest and the other after movement (3 deep knee flexions).

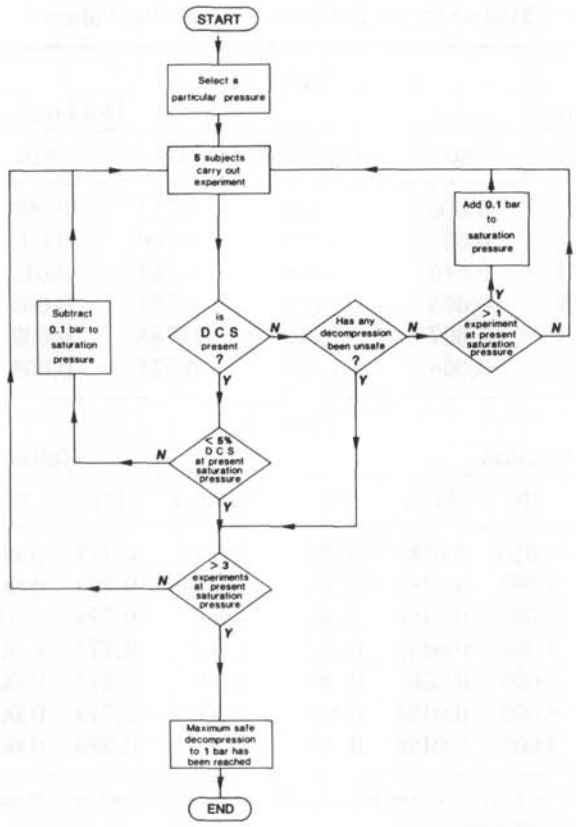

Fig. 2. Flow diagram illustrating the protocol to be followed throughout the trial.

Tests for significant differences were made by a test of independence utilizing the Fisher Exact Test of Independence (13). This tests the expected frequencies of events, based on the null hypothesis of independence, against the observed frequencies. The data were arranged in a 2 × 2 contingency table before processing.

RESULTS

Decompression sickness

The results are shown in Table 3. Four cases of DCS, all type I bends with no neurologic involvement, were recorded in 25 subjects following decompression from 1.8 bar. Two cases after O_2-N_2 dives during phase 1 and 2 cases after O_2-N_2-CO_2 dives during phase 2. All cases of DCS involved the knee, either unilaterally or bilaterally, and were reported after 265 and 305 min postdecompression in phase 1 and 380 and 275 min in phase 2. All cases were assessed by the independent medical officer and treated using Royal Navy Table 61, with full relief of symptoms on reaching 2.8 bar and no recurrence of pain during or after subsequent recompression. Only those cases

TABLE 3
SUMMARY OF THE DECOMPRESSION AND VGE RESULTS

		Phase 1		Phase 2	
		O_2-N_2		O_2-N_2-CO_2	
Saturation pressure (bar)		1.7	1.8	1.7	1.8
Total no. of subjects		20	10	20	15
Number of subjects with:					
Nonspecific aches		1 (5%)	2 (20%)	2 (10%)	3 (20%)
Pruritus		0	0	1 (5%)	0
Type I DCS		0	2 (20%)	0	2 (13%)
Number of subjects with VGE:					
At rest		9 (45%)	7 (70%)	3 (15%)	10 (67%)
With movement		17 (85%)	10 (100%)	6 (30%)	11 (70%)
Mean VGE onset time (min)		95	119	173	148
Range		(8–220)	(10–250)	(100–242)	(63–374)
Peak VGE grade:					
At rest	0	9 (45%)	3 (15%)	17 (85%)	5 (33.3%)
	1	5 (25%)	4 (20%)	2 (10%)	6 (40%)
	2	4 (20%)	7 (35%)	1 (5%)	2 (13.3%)
	3	2 (10%)	3 (15%)	0	2 (13.3%)
	4	0	3 (15%)	0	0
With movement	0	3 (15%)	0	14 (70%)	4 (25%)
	1	4 (20%)	4 (40%)	5 (25%)	7 (47%)
	2	7 (35%)	1 (10%)	1 (5%)	3 (20%)
	3	3 (15%)	2 (20%)	0	0
	4	3 (15%)	3 (30%)	0	1 (8%)

Figures are mean values ± 1 SD. Percentage values are given in parenthesis. The range of values is shown where SD are not appropriate.

requiring treatment in the opinion of the independent medical officer were recorded as suffering from DCS.

Eight subjects reported nonspecific symptoms, 6 with muscular aches and 2 with transient joint pain after decompression from 1.8 bar, suggesting that they were approaching their personal decompression limit. One isolated case of pruritus, which resolved in 2–3 h, was also reported.

Venous gas emboli

The incidence and grade of VGE detected in the subjects at rest and after movement are shown in Table 3.

During phase 1, no significant differences were found between the incidence of VGE following decompression from 1.7 or 1.8 bar. However, during phase 2 significant differences were evident both at rest ($P < 0.005$) and after movement ($P < 0.05$) (Fig. 3).

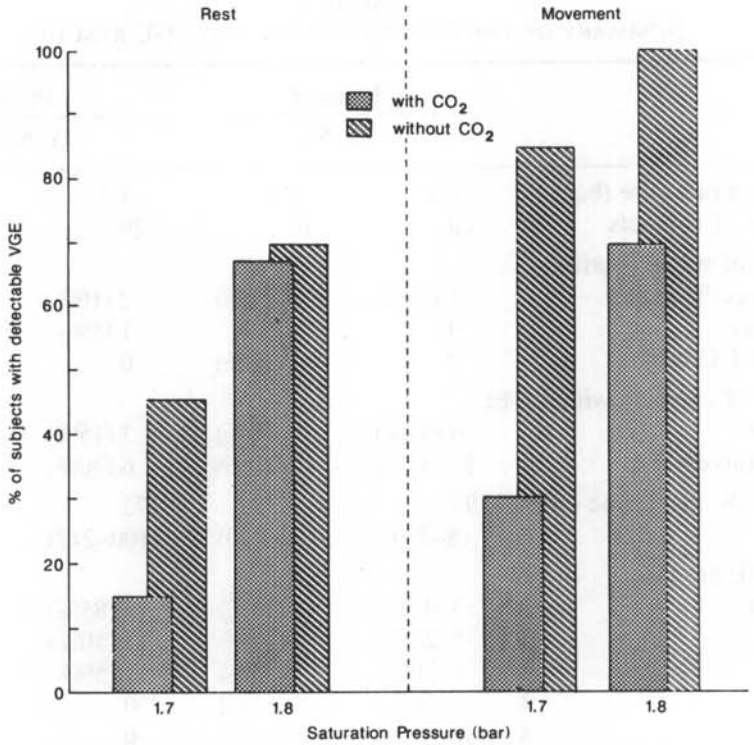

Fig. 3. The VGE incidence at rest and after movement following decompression from 1.7 and 1.8 bar during phases 1 and 2. In phase 1, without CO_2, there was a significant difference between the VGE incidence at rest and after movement following decompression from 1.7 bar ($P < 0.05$). In phase 2 this difference was not significant. The VGE incidence at rest and after movement following decompression from 1.7 bar during phase 1, without CO_2, was significantly higher ($P < 0.05$ and $P < 0.005$, respectively) than that following decompression from 1.7 bar during phase 2, with CO_2.

Figure 3 also allows comparison of the results from phases 1 and 2. This shows that the incidence of VGE was significantly reduced in phase 2 following decompression from 1.7 bar. The reduction in VGE incidence was evident both at rest ($P < 0.05$) and after movement ($P < 0.005$). However, following decompression from 1.8 bar there was no significant difference in VGE incidence at rest or after movement.

The mean onset times for detectable VGE in phases 1 and 2 are shown in Table 3. The considerable variability in the individual results make it impossible to observe any significant differences although the onset times in phase 2 seem greater than in phase 1. However, Fig. 4 showing the percent of subjects with detectable VGE at each of the monitoring periods reveals that the onset of detectable VGE is clearly longer in phase 2, particularly after decompression from 1.7 bar ($P < 0.005$) but also following decompression from 1.8 bar ($P < 0.05$).

The percentage of subjects with bubble grades 0 to 4 shown in Table 3 indicate the effect of increasing the pressure from 1.7 to 1.8 bar on the distribution of subjects within the 5 grades. Decompression from the larger pressures spreads the distribution and generally shifts the median value to the higher grades. Comparing phases 1 and

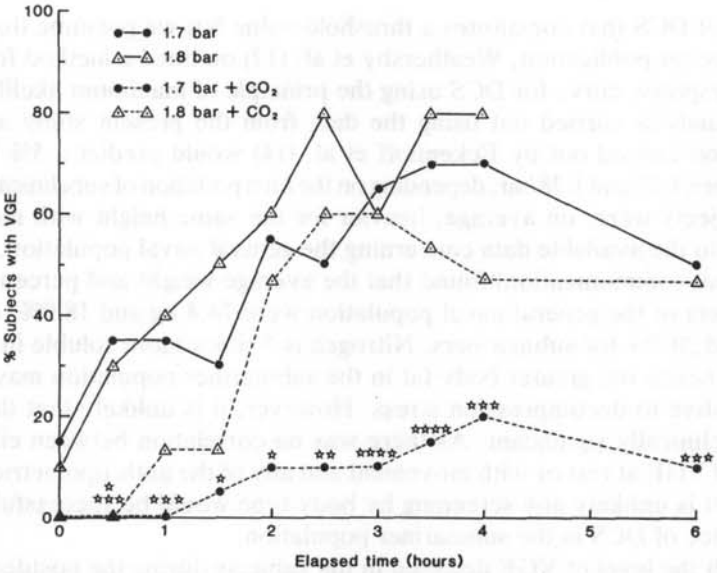

Fig. 4. The percentage of subjects with detectable VGE at each of the Doppler monitoring periods following decompression from 1.7 and 1.8 bar with and without the presence of 0.02 bar CO_2. Significant differences from the equivalent control group values are shown by the following: * = $P < 0.05$, ** = $P < 0.01$, *** = $P < 0.005$, **** = $P < 0.001$.

2, the same shift is evident but the starting distribution is more concentrated toward the lower grades in the presence of 0.02 bar CO_2.

Eckenhoff et al. (14) have previously reported that the shape of the curve produced by recording the percentage of subjects with detectable VGE against time is different following decompression from different depths. Figure 4 demonstrates that in phase 1 the curves were not significantly different following decompression from either 1.7 or 1.8 bar, but in phase 2, when 0.02 bar CO_2 was present, the curve following decompression from 1.7 bar is significantly different ($P < 0.05$) over its entire length from that obtained following decompression from 1.8 bar.

DISCUSSION

The type of DCS provoked, i.e., limb bends with no neurologic involvement, was in agreement with the results obtained in earlier studies (15). However, there was very little evidence among the other subjects of any other decompression-related phenomena, such as excessive fatigue, athralgia, and pruritus, previously reported by Eckenhoff et al. (14).

It is evident that a sharp distinction exists between the unsafe and safe decompression step, because while none of the 40 subjects decompressed from 1.7 bar recorded any signs or symptoms of DCS, 4 (16%) of the 25 subjects reported DCS following decompression from 1.8 bar. Although 0 out of 40 subjects does not guarantee a DCS incidence of less than 5%, 95% confidence limits (0–8.81%), 4 out of 25 subjects, 95% confidence limits (4.54–36.08%), would be almost certain to exceed the 5% limit. This finding is in agreement with the prediction of Hennessy and Hempleman (16) of a DCS threshold of 1.76 bar when the P_{O_2} is 0.4 bar. These authors do not specify the

incidence of DCS that constitutes a threshold value but we presume that it is below 5%. In a recent publication, Weathersby et al. (17) outlined a method for estimating the dose response curve for DCS using the principle of maximum likelihood estimation. An analysis carried out using the data from the present study and a similar investigation carried out by Eckenhoff et al. (14) would predict a 5% incidence of DCS between 1.75 and 1.78 bar, depending on the interpretation of subclinical symptoms.

The subjects were, on average, heavier for the same height with more body fat compared to the available data concerning the general naval population. Smith DJ et al. (personal communication) found that the average weight and percent body fat of 978 members of the general naval population were 74.4 kg and 18.9%, compared to 77.0 kg and 20.7% for submariners. Nitrogen is 5 times more soluble in lipid than in water and hence the greater body fat in the submariner population may make them more sensitive to decompression stress. However, it is unlikely that the difference would be clinically significant. As there was no correlation between either DCS or the level of VGE at rest or with movement and any of the anthropometric parameters recorded, it is unlikely any screening by body type would be successful in reducing the incidence of DCS in the submariner population.

Although the level of VGE detected in the subjects during the postdecompression monitoring period varied considerably, the difference in the VGE grade and incidence between the two decompressions and between the phases 1 and 2 groups illustrate an interesting effect.

The addition of CO_2:

1) significantly reduces the incidence of VGE following decompression from 1.7 bar but not after decompression from 1.8 bar;
2) modifies the distribution of bubble grades following decompression from 1.7 or 1.8 bar toward the lower values; and
3) significantly delays the onset of detectable VGE following decompression from 1.7 and 1.8 bar.

These observations suggest that the effect of CO_2 is to prevent the appearance of VGE and to lower the level of VGE once produced. The effect is more marked following decompression from 1.7 bar.

The reasons for the effect of the presence of CO_2 on the incidence and grade of VGE are not clear. Carbon dioxide is extremely soluble in water, suggesting that any decompression in which gas solubility governed the behavior of the gases would result in a rapid release of CO_2 from the aqueous tissues. Mano and D'Arrigo (9) suggest that this initial release of CO_2 would increase the size of any small bubble passing through the aqueous tissue, thus stabilizing the bubble long enough for nitrogen to diffuse into it. However, it is possible that the conjugation of CO_2 within the blood by the buffering systems may prevent the gas evolving too rapidly, and at the levels used in this study the action of CO_2 would appear to be an increase in both ventilation and cardiac output combined with an increased peripheral perfusion. These effects may be beneficial during decompression because by increasing the removal of inert gas from the periphery to the lungs they decrease more rapidly the concentration gradient formed between the tissues and the blood. This may have the effect of delaying the onset of VGE until the gas released from the tissues is greater than can be removed in solution. Once bubbles were formed, the interference they cause with the inert gas washout, by hemostasis, increased viscosity, and reduced blood flow (18) would allow the number of bubbles to increase to near normal levels.

Masurel and Guillerm (19) found that the presence of 0.05–0.07 bar of CO_2 in the diving gas mixture seemed to reduce the formation of bubbles although the difference was not significant. The CO_2 was present during the bottom phase of the dives that lasted over 5 h, and the bubble level was measured using chronically implanted Doppler probes around the pulmonary artery. An earlier study had assessed the effect on nitrogen washout of breathing a gas mixture of 0.05 bar carbon dioxide in oxygen commencing 10 min after decompression (20). Those authors found that at levels greater than 0.05 bar there was a significant increase in nitrogen efflux compared with control levels. The results of the present study seem to suggest that 0.02 bar CO_2 breathed over a 48-h period can produce similar effects on nitrogen efflux and subsequent bubble production.

Evidence exists that the effect of CO_2 on ventilation and cardiac output can persist for long periods with no adaption by the subjects. If the subjects adapted to the CO_2 stimulus, the proposed protective mechanism would not apply. However, Harrison and Smith (21) have shown that exposure to 0.015 bar CO_2 can increase ventilation by up to 20% and that there were no adaptive changes recorded during a 30-d exposure to 0.015 bar CO_2. They did record significant vascular changes on removal of the CO_2, which may be important during submarine rescue operations if the crew have been exposed to elevated CO_2 levels for a period approaching 20–30 d.

Another possible explanation for the effect of CO_2 on the incidence of detectable VGE could be if it reduced the bubble size to below the threshold for detection by the Doppler ultrasound unit. If the bubbles were below 80–100 μm they would pass undetected by the unit. However, if this were the case it is likely to be a beneficial effect, because the smaller the bubble, the less stable it would be and may be more easily removed by the lungs. In addition, it is always possible that the observer grading the VGE missed low amplitude bubbles. However, we have attempted to negate this effect by ensuring that the same observers graded all the recordings, thus giving their gradings the necessary consistency.

The results from this study indicate that decompression to 1 bar from 1.8 bar is likely to produce an unacceptable incidence of decompression sickness with or without the presence of 0.02 bar carbon dioxide. Carbon dioxide, when present in the saturation gas mixture in the concentration used in this study, delays the onset of detectable VGE and produces a lower incidence of detectable VGE following decompression. This reduction is particularly apparent following decompression from 1.7 to 1 bar.

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Bell PY, Harrison JR, Page K, Summerfield M. Un effet du CO_2 sur la décompression maximale directe sans danger à 1 bar d'une plongée à saturation à l'oxygène et l'azote. Undersea Biomed Res 1986; 13(4):443–455.—Une étude sur l'étape de décompression maximale sans danger à 1 bar d'une plongée à saturation à l'azote et l'oxygène fut conduite avec et sans la présence de 0.02 bar d'anhydride carbonique (CO_2). La série, Islander 1, comprit 13 équipes de 5 volontaires mâles, bien informés, pour effectuer les plongées simulées. Un groupe de 6 équipes plongea dans un atmosphère composé d'oxygène à une pression partielle de 0.4 bar avec de l'azote (O_2-N_2) comme complément; un autre groupe de 7 équipes employa un atmosphère composé d'oxygène à 0.38 bar, d'anhydride carbonique à 0.02 bar avec de l'azote (O_2-N_2-CO_2) comme complément. Les plongées consistèrent en un séjour de 48 h à 1.7

ou 1.8 bar pour saturer les tissus, suivi d'une décompression à 1 bar avec de l'air à la vitesse de 0.5 bar/min. Deux paramètres de décompression furent examinés: l'incidence de maladie de décompression (DCS) dans les 24 h suivant la décompression, ainsi que l'apparition et le grade des embolies gazeuses veineuses (EGV) dans les premières 6 h après la décompression. Le grade des EGV fut déterminé à l'aide du système de mesure Kisman-Masurel, lequel fournit un grade de bulles chez le sujet à l'état de repos et après un mouvement. Aucune différence significative ne fut trouvée dans l'incidence de la DCS entre les 2 groupes. Vingt sujets furent décomprimés à partir de 1.7 bar à l'aide de chaque mélange. Il n'y eut pas de signes ou de symptômes de DCS. Cependant, après la décompression de 1.8 bar, il y eut 2 cas de DCS chez 10 suiets du groupe O_2-N_2 et 2 cas chez 15 sujets du groupe O_2-N_2-CO_2. L'incidence des EGV détectables fut toujours moindre dans le groupe O_2-N_2-CO_2 pour les 2 pressions de saturation. A 1.7 bar, l'incidence des EGV était de 40% moindre ($P < 0.05$) au repos et de 55% ($P < 0.001$) après le mouvement. A la pression de 1.8 bar, la réduction était de 3% ($P > 0.05$) au repos et 30% ($P > 0.05$) après le mouvement. Les résultats indiquent que la décompression de 1.8 à 1 bar, avec ou sans la présence de CO_2 à 0.02 bar a des chances de produire plus de 5% de DCS.

saturation
anhydride carbonique
décompression
ultrason
azote
sauvetage de sous marin

United Kingdom

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