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Altitude Decompression Illness – The Operational Risk at Sustained Altitudes up to 35,000 ft

VM Lee and AE Hay,
Centre for Human Sciences,
DERA Farnborough,
Hampshire, GU14 0LX
UK

Summary

Altitude decompression illness (DCI) is generally considered to be a risk at altitudes in excess of 18,000 ft. UK military aircrew are therefore not routinely exposed to altitudes in excess of this, however, there are circumstances such as loss of cabin pressure, parachute operations, and high cabin altitudes in future aircraft, when exposure to altitudes in excess of 18,000 ft may be necessary. A series of experiments were carried out at the DERA Centre for Human Sciences to investigate the risk of venous gas emboli (VGE) and DCI symptoms at altitudes up to 35,000 ft. Subjects were exposed, for a maximum of four hours, to i) simulated altitudes between 20,500 ft and 25,000 ft breathing an oxygen/nitrogen gas mixture, ii) 25,000 ft breathing 100% oxygen with and without one hour of prior denitrogenation and iii) simulated altitudes up to 35,000 ft with one hour prior denitrogenation. It was concluded that VGE formation will occur at cabin altitudes that will be encountered by aircrew of future agile aircraft although only 7% of subjects developed symptoms. Exposure to 25,000 ft breathing an oxygen/nitrogen gas mixture resulted in VGE and symptoms significantly earlier than during exposures to 20,500 ft and 22,500 ft. Furthermore, exposure to 25,000 ft for four hours breathing either gas mix or 100% oxygen, without prior denitrogenation, incurred a substantial risk of developing symptoms of DCI. Denitrogenation, however, for one hour prior to decompression provided effective protection against development of symptoms of DCI at 25,000 ft for subjects at rest. Finally, subjects exposed to 35,000 ft developed VGE and symptoms of DCI significantly earlier than subjects exposed to 25,000 ft.

Introduction

A method of using compressed air to prevent mine shafts beneath water and submerged ground from flooding was first described in 1841 (Triger 1845). The first application of this technique took place in 1841 in France, and although it was considered a complete success, symptoms consistent with decompression illness (DCI) were described by the workers. A brief note suggests that two of the workers who had been compressed for seven hours suffered pain, one in the left arm and the other in the left shoulder and both knees, half an hour after the exposure (Pol & Watelle 1854). Later, during the building of the St Louis Bridge in the 1870s one hundred and nineteen caisson workers developed neurological symptoms and fourteen workers died (Woodward 1881). Paul Bert showed that decompression from a raised pressure gave rise to the evolution of bubbles in tissues and fluids and postulated that these were the cause of the clinical syndromes seen in caisson workers and divers (Moon *et al* 1995). The possibility of bubble formation on ascent to altitude, however, was not considered until 1901 when von Schrotter reported symptoms including difficulty of breathing, despite the use of oxygen, during a 15 minute exposure to 30,000 ft. Von Schrotter attributed the incident to bubble formation in accordance with Paul Bert's theory for the aetiology of symptoms experienced by caisson workers and divers (Fryer 1969).

Aviators ascending to altitude, like divers ascending from depth, are exposed to a reduction in pressure. When the pressure of the gases dissolved in the body tissues exceeds that of the atmospheric pressure the tissues become supersaturated with gas and there is potential for the formation of bubbles within the tissues. These bubbles are thought to be the cause of a myriad of clinical disorders from mild limb pain or paraesthesia through to more serious neurological or respiratory symptoms, and ultimately to complete circulatory collapse and death. The manifestation of symptoms may be caused simply by the physical presence of gas causing direct mechanical disruption of a tissue, tissue compression, or obstruction of a blood vessel. In contrast, symptoms may be caused

by a tissue response to the presence of bubbles which may stimulate activation of endothelial cells, platelets or biochemical pathways (Francis & Gorman 1993).

The minimum altitude at which bubble formation is sufficient to result in symptoms is not known, although 18,000 ft is generally considered to be the threshold for decompression illness (DCI). This threshold of 18,000 ft came from a theory put forward by J.S. Haldane who proposed that a pressure drop of one half of the original gas pressure, whatever the value, would be safe - ie: sea level to 18,000 ft. Altitude decompression illness however, was not considered when this theory was presented (Boycott *et al* 1908). This 2:1 rule is commonly held today despite the demonstration of decompression illness, albeit rarely, below this altitude. With this in mind UK military aircrew are not routinely exposed to altitudes in excess of 18,000 ft in order to minimise the risk of decompression illness. There are circumstances, however, when altitudes in excess of 18,000 ft will be encountered by UK military personnel. For example, following loss of cabin pressure a maximum altitude of 25,000 ft is recommended and some aircraft have the facility to remain at this altitude for longer than four hours. Parachute operations may expose individuals to altitudes in excess of 18,000 ft, and high altitude high opening operations will provide the greatest risk of decompression illness. Finally, future fast jet aircraft, such as Eurofighter (EF) will attain cabin altitudes in excess of 18,000 ft when the aircraft reaches its operational ceiling.

The reported incidence of DCI in RAF aircrew to date has been extremely low. Harding (1992) stated that only two cases of DCI had been reported by aircrew in the RAF between 1980 and 1990, although there have been several anecdotal incidents of unreported limb pain by Canberra crew members. A recent survey of Canberra aircrew found that more than 20% of respondents had experienced limb pain that was considered to be consistent with symptoms of DCI (Mitchell & Lee 2000). Similarly, a large discrepancy between the number of reported cases of DCI in aircrew (USAF) and the incidence of DCI in experimentally similar profiles has been highlighted by Pilmanis & Bisson (1992). These authors reported that while only 1- 2 cases of DCI are declared each year in pilots that fly at cabin altitudes between 28,000 ft - 30,000 ft (U-2 & TR-1), 73% of research subjects exposed to a similar altitude profile reported DCI. Furthermore, in an anonymous survey, this same population of pilots reported that 62% had experienced DCI at least once during a high altitude flight. These data suggest that reported cases of DCI are unlikely to reflect the true incidence during flight.

This paper describes a series of experiments that were carried out at the DERA Centre for Human Sciences to investigate the risk of developing venous gas emboli (VGE) and symptoms of DCI at altitudes up to 35,000 ft.

Methods

Fifteen male subjects were exposed, with Local Research Ethics Committee approval, to each of the profiles shown in Table 1 for a maximum period of four hours.

Altitude (ft)	Denitrogenation	Breathing Gas Composition
20,500	No	56% oxygen 44% nitrogen
22,500	No	56% oxygen 44% nitrogen
25,000	No	63% oxygen 37% nitrogen
25,000	No	100% oxygen
25,000	Yes	100% oxygen
30,000	Yes	100% oxygen
35,000	Yes	100% oxygen

Table 1. Experimental profiles indicating the simulated altitude, the use of denitrogenation with 100% oxygen for one hour prior to decompression, and the composition of the breathing gas from the initiation of decompression.

The subjects were fitted with an aircrew coverall, a cloth G type flying helmet and a modified RAF P/Q type oronasal oxygen mask. The mask was modified with the addition of a drinking facility, and subjects were encouraged to drink fluids throughout the experiment. Subjects were supplied with breathing gas from a Type

17 oxygen demand regulator with a positive pressure, of about 5 mmHg, to ensure no inward leaks of ambient air into the mask during inspiration.

No subject had been exposed to a hyper- or hypobaric environment within the 48 hours prior to participating in this study. For profiles that did not employ denitrogenation, subjects started to breathe the appropriate gas mixture as decompression to simulated altitude was initiated. For profiles employing denitrogenation subjects breathed 100% oxygen (preoxygenation) for exactly one hour before decompression to simulated altitude, and subjects remained at rest throughout the period of preoxygenation. Subjects were decompressed to simulated altitude in the CHS's hypobaric chamber, at a rate equivalent to 5,000 ft min⁻¹. Subjects remained at the required altitude for a maximum duration of four hours and remained at rest throughout the exposure to altitude.

On reaching the simulated altitude the subjects were monitored, every fifteen minutes, using 2D and Doppler echocardiography (SONOS 1500 or 2500 Hewlett Packard with a 2.5MHz transducer) for the presence of VGE (Olson *et al*; 1992). A four chamber view of the heart was obtained (Figure 1), with the subject in the seated position. Any VGE that were detected in the right cardiac chambers were graded by the investigator outside the hypobaric chamber on a 0 - 4 scale based on that described by Spencer (1976). During each echocardiograph the subject moved each limb in turn in such a manner that the limb joints were rotated and flexed to allow any bubbles present around the joint to be dislodged.

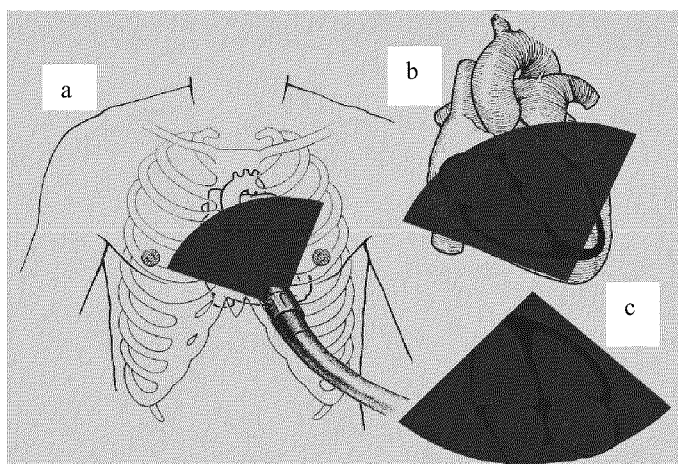


Figure 1. Diagram showing a) orientation of the echocardiograph transducer on the chest wall, b) plane of the echocardiograph through the cardiac chambers and c) view of 2D echocardiographs used for data analysis.

Subjects were briefed prior to exposure to altitude on the symptoms of decompression illness and instructed to report any symptoms or sensations experienced at altitude that were not present at ground level. In an attempt to avoid the reporting of spurious symptoms the subjects were encouraged to watch films throughout the exposure and, beyond the pre-exposure brief, subjects were not reminded again about symptom reporting.

If the subject reported limb or joint pain that was constant - Grade II pain according to Webb & Pilmanis (1992) - they were asked to rate the level of pain on a scale from 1-10 (0 being no pain and 10 being worst pain imaginable). The subject was then immediately recompressed to ground level at a rate of 5,000 ft min⁻¹ (unless a different rate was indicated). The subjects were also recompressed to ground level immediately if any other symptoms resulting from exposure to simulated altitude were reported by the subject or observed by the investigators, or if gas emboli were detected in the left cardiac chambers. During recompression, the simulated altitude at which the subject no longer experienced symptoms was recorded. All subjects were required to breathe 100% oxygen for a further two hours on reaching ground level unless their symptoms indicated that hyperbaric oxygen therapy was required.

The latency from reaching the final altitude to exhibiting any VGE, grade 4 VGE and reporting symptoms was calculated. These onset times were analysed using analysis of variance. Where no VGE or symptoms were exhibited during exposure to the final altitude it was assumed, for the purposes of statistical analysis, that VGE

or symptoms could have developed at 240 minutes. This does however, have the effect of biasing the results towards shorter, or worst case, mean onset times.

Results

Figures 2 and 3 show an example of an echocardiograph from a subject at 30,000 ft without and with VGE respectively. Table 2 shows the percentage of subjects that exhibited any VGE, and grade 4 VGE, with mean times to onset from reaching the final altitude. Table 2 also shows the percentage of subjects that reported symptoms consistent with DCI that resulted in recompression to ground level, and the mean time to initiation of recompression. Figures 4, 5 and 6 show the cumulative number of subjects, during each 30 minute period of each profile, exhibiting any VGE, grade 4 VGE, and symptoms, respectively.

Venous gas emboli, grade 4 VGE, and symptom development at 20,500 ft, 22,500 ft and 25,000 ft were compared (Figs 4(a), 5(a) & 6(a) respectively). Subjects exposed to 25,000 ft exhibited grade 4 VGE significantly earlier than subjects exposed to 20,500 ft and 22,500 ft ($p < 0.05$) (Fig 5(a)). Similarly, subjects exposed to 25,000 ft breathing a nitrogen/oxygen gas mixture exhibited symptoms significantly earlier than subjects exposed to 20,500 ft and 22,500 ft (Fig 6(a)).

Venous gas emboli, grade 4 VGE, and symptom development at 25,000 ft employing a nitrogen/oxygen breathing gas, 100% oxygen breathing gas, and 100% oxygen breathing gas with one hour prior denitrogenation have also been compared (Figs 4(b), 5(b) & 6(b) respectively). There was no significant difference in the onset time of VGE or symptoms between subjects exposed to 25,000 ft breathing either gas mix or 100% oxygen. However, subjects breathing the nitrogen/oxygen gas mixture at 25,000 ft exhibited grade 4 VGE significantly earlier than subjects breathing 100% oxygen ($p < 0.05$). Subjects who denitrogenated prior to exposure to 25,000 ft exhibited VGE, and grade 4 VGE, and symptoms significantly later than subjects exposed 25,000 ft without denitrogenation ($p < 0.001$, $p < 0.01$ & $p < 0.001$ respectively) (Figs 4(b), 5(b) & 6(b) respectively).

Venous gas emboli, grade 4 VGE, and symptom development at 25,000 ft, 30,000 ft and 35,000 ft with one hour prior denitrogenation have been compared (Figs 4(c), 5(c) & 6(c) respectively). Subjects exposed to 35,000 ft exhibited VGE and symptoms significantly earlier than subjects exposed to 25,000 ft with denitrogenation ($p < 0.01$ & $p < 0.01$ respectively). However, there was no significant difference in the onset of grade 4 VGE between 25,000 ft, 30,000 ft and 35,000 ft with denitrogenation (Figs 4(c), 5(c) & 6(c)).

Altitude (ft)	Preox	Breathing gas (% O ₂)	% with VGE	Mean time to onset (mins)	% with G4 VGE	Mean time to onset (mins)	% with symptoms	Mean time to recompression (mins)
20,500	N	56	80	77 ± 70	53	95 ± 30	7	192
22,500	N	56	67	54 ± 38	47	82 ± 37	7	172
25,000	N	63	100	51 ± 58	93	83 ± 49	53	138 ± 39
25,000	N	100	87	52 ± 60	60	89 ± 43	60	125 ± 35
25,000	Y	100	33	123 ± 53	20	162 ± 23	0	-
30,000	Y	100	60	111 ± 78	33	108 ± 68	40	148 ± 46
35,000	Y	100	93	102 ± 69	53	116 ± 51	53	106 ± 46

Table 2. Percentage of subjects undergoing each profile exhibiting any VGE, grade 4 VGE and symptoms, together with times (mins) to onset of VGE and times to recompression following symptom development (mean ± SD).

During these altitude exposures a number of subjects exhibited gas emboli in the left ventricle, (Figure 7 shows an echocardiograph from a subject with gas emboli in both the left and right ventricles). Gas emboli in the left ventricle will pass directly into the arterial circulation. Therefore, these subjects were immediately recompressed to ground level as the potential for development of neurological symptoms is considered to be greater in individuals with arterial gas emboli. Table 3 shows the conditions under which subjects developed arterial gas emboli (AGE) and times to onset. Five out of the six subjects exhibiting AGE were also exhibiting maximum grade (grade 4) VGE.

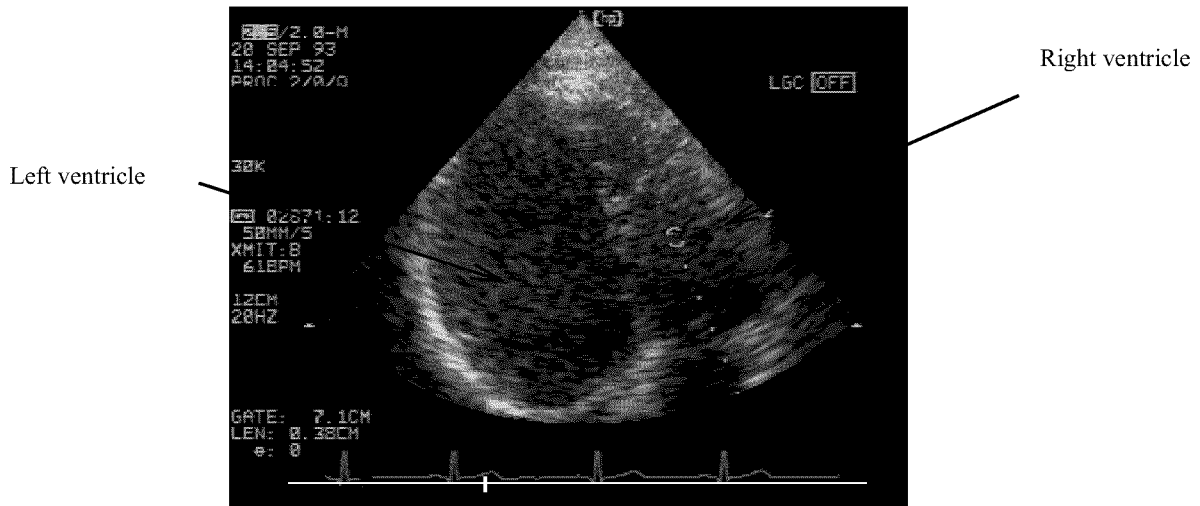


Figure 2. Echocardiograph from a subject at 30,000 ft breathing 100% oxygen following denitrogenation – no gas emboli are evident in this image.

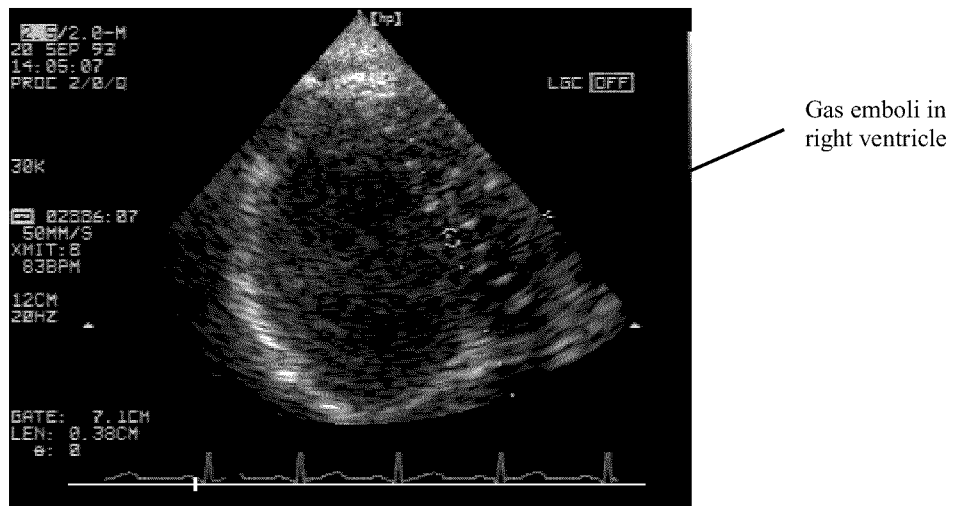


Figure 3. Echocardiograph from a subject at 30,000 ft breathing 100% oxygen following denitrogenation – several gas emboli are present in the right ventricle of this subject.

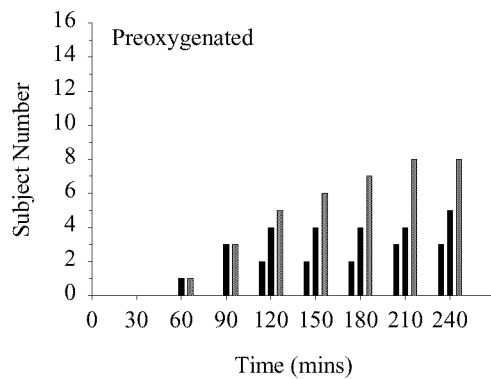
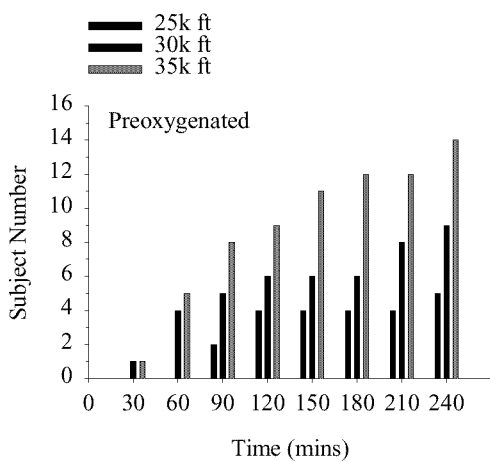
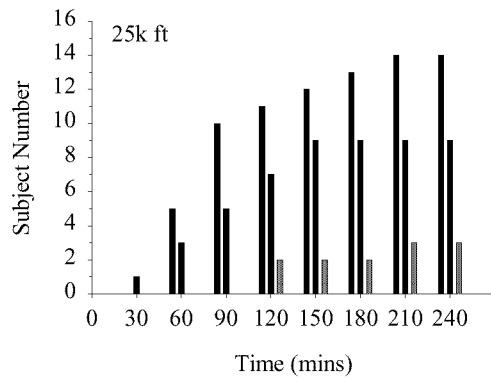
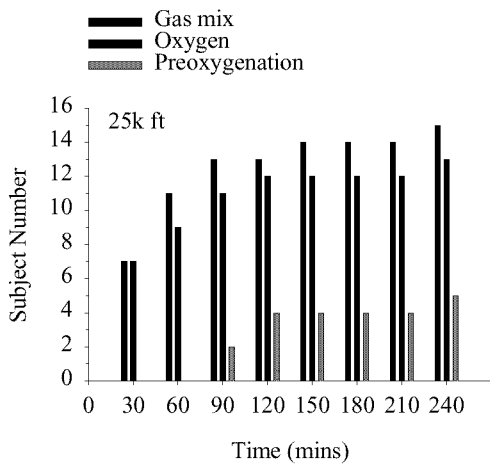
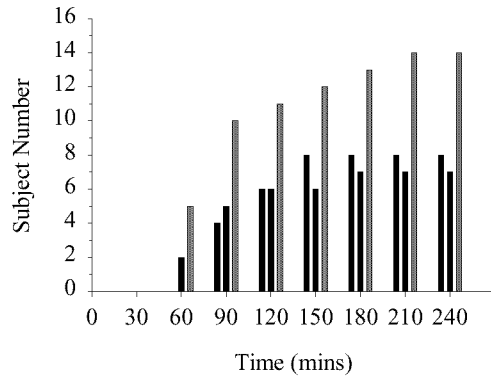
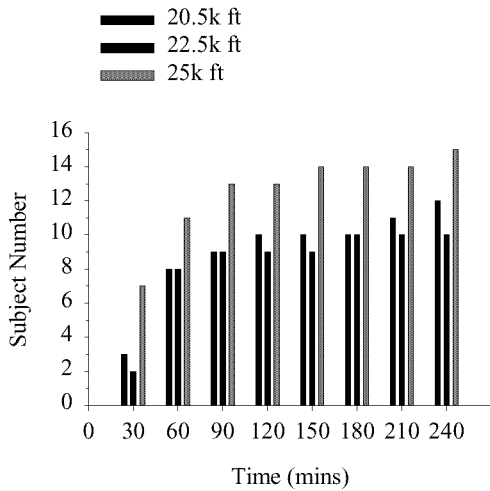


Figure 4. Showing the cumulative number of subjects developing VGE during each 30 minute period of the 240 minute exposure for a) profiles that subjects were breathing a nitrogen/oxygen gas mixture b) profiles that subjects were exposed to 25,000 ft and c) profiles where one hour of prior denitrogenation with 100% oxygen was employed.

Figure 5. Showing the cumulative number of subjects developing grade 4 VGE during each 30 minute period of the 240 minute exposure for a) profiles that subjects were breathing a nitrogen/oxygen gas mixture b) profiles that subjects were exposed to 25,000 ft and c) profiles where one hour of prior denitrogenation with 100% oxygen was employed.

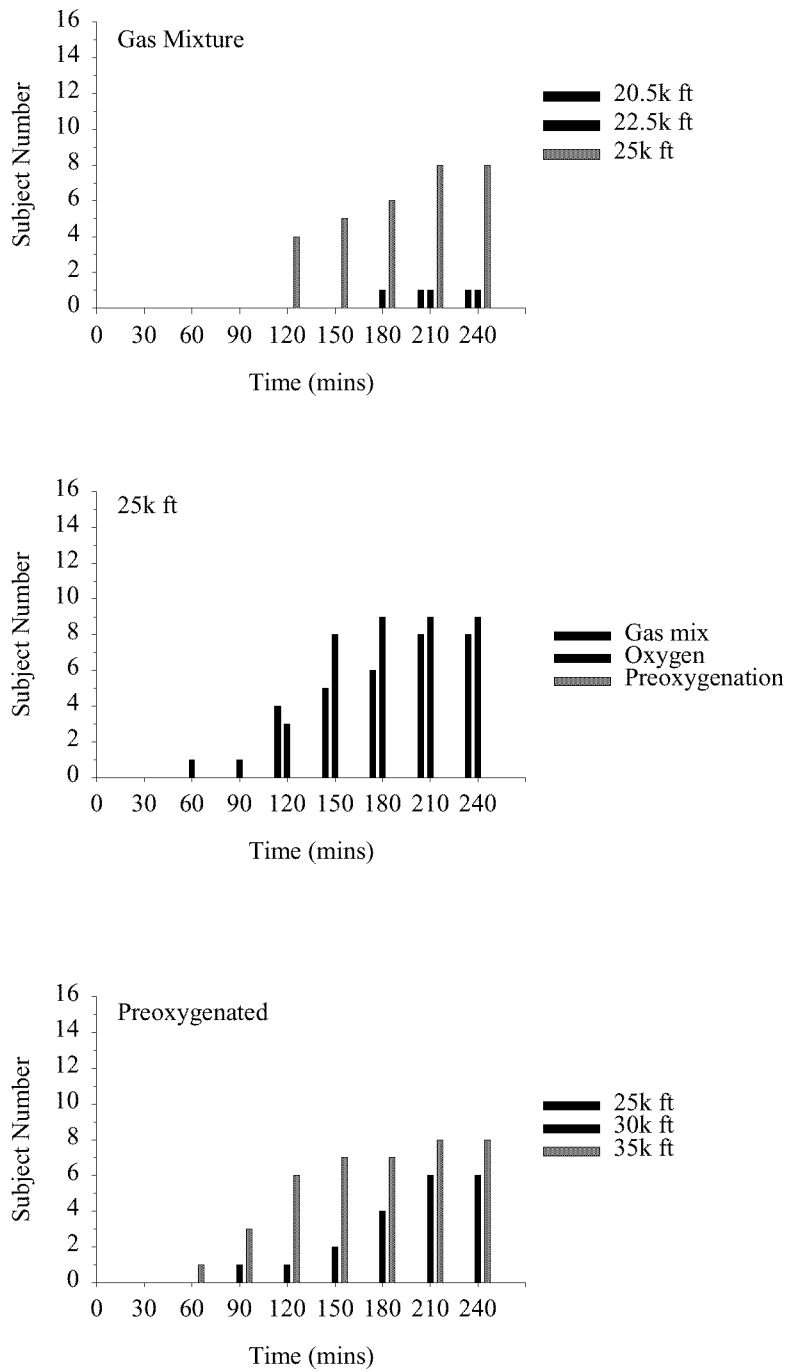


Figure 6. Showing the cumulative number of subjects developing **symptoms** during each 30 minute period of the 240 minute exposure for a) profiles that subjects were breathing a nitrogen/oxygen gas mixture b) profiles that subjects were exposed to 25,000 ft and c) profiles where one hour of prior denitrogenation with 100% oxygen was employed.

that this discrepancy in the incidence of DCI is due to the additional use, by Webb *et al* (1998), of three sets of mild arm exercises each hour, to simulate extravehicular activity. The effect of exercise on the incidence of DCI is discussed later.

The results also indicated that between 80% and 67% developed VGE at 20,500 ft and 22,500 ft respectively, with a corresponding mean onset time of 77 and 54 minutes. The presence of such non-symptomatic bubbles in the venous side of the circulation has been demonstrated in many other studies (eg Dixon *et al* 1986, Kumar *et al* 1992). Most commonly venous gas emboli are thought to circulate through the right cardiac chambers and be filtered out in the pulmonary circulation (Spencer 1971). Gas emboli can, however, enter the arterial circulation via intracardiac septal defects, pulmonary arteriovenous shunts, or through the pulmonary microcirculation. Indeed, in this study gas emboli in the left ventricle were detected during six out of one hundred and fifteen exposures (5.2%). The presence of arterial gas emboli present a considerable hazard as they have potential to result in impairment of neurological function and cerebral damage.

Intracardiac septal defects, in the form of a patent foramen ovale (PFO), appear to exist in around 20-30% of the human population and have been implicated in neurological DCI in divers (Moon *et al* 1989). In one study a five fold increase in risk of decompression illness was associated with individuals with a PFO evident under resting conditions (Moon *et al* 1991). In contrast, no correlation was found, in individuals undergoing USAF hypobaric training exposures, between those with a PFO and those that reported neurological DCI (Clark & Hayes 1991). Furthermore, in a single subject with a PFO evident under resting conditions, and exhibiting grade 4 VGE at 21,000 ft, no symptoms were reported and no evidence for gas emboli in the left side of the heart was seen (Powell *et al* 1995). Present evidence indicates that the incidence of serious DCI in divers, and in particular in aviators, does not approach the percentage of the population with a PFO. Individuals with a PFO may not experience symptoms of DCI as a reversal of the normal left to right heart pressure gradient is usually necessary for gas emboli to traverse a septal defect into the left side of the heart. Such reversals in pressure can however be established upon release of a Valsalva and cessation of either positive pressure breathing or the anti-G straining manoeuvre (Garrett 1992). It is apparent, therefore, that there is greater potential for such reversals of pressure in aircrew of future high performance aircraft which might allow for venous gas emboli, provoked by the altitude exposure, to cross into the arterial circulation.

In addition to intracardiac septal defects, high grades of VGE have also been shown to result in gas emboli in the arterial circulation even when a PFO is not evident (Butler & Hills 1985, Vik *et al* 1993). Indeed, Pilmanis (1995) reported, that an atrial septal defect could only be established by transoesophageal contrast echocardiography in two of three subjects who exhibited arterial gas emboli at altitude. Pilmanis (1995) also reported that all the subjects with AGE were exhibiting the highest VGE score (grade 4) prior to the presence of arterial gas emboli, this is in common with all but one of the subjects in this study with arterial gas emboli. Thus, more than one mechanism may be responsible for the cross over of bubbles from the venous to arterial circulation. This study shows that bubble onset can occur within twenty two and nineteen minutes of exposure to 20,500 ft and 22,500 ft respectively, and that grade 4 VGE onset can occur within sixty one and thirty six minutes respectively. Therefore, although the risk of developing symptoms at altitudes up to 22,500 ft while at rest appears to be small, the exposure of aircrew to these altitudes can result in significant bubble formation. These conditions, particularly for those with PFOs, increase the risk of transfer of gas emboli to the arterial circulation which in turn could result in development of serious neurological symptoms.

VGE & Decompression

The development of VGE at cabin altitudes that are likely to be encountered routinely in future aircraft provides the potential for the development of arterial gas emboli, with the consequences discussed above. The presence of non-symptomatic bubbles is also likely to have considerable consequences in the event of cabin depressurisation. It is likely that an individual who has developed VGE under pressurised conditions, and who undergoes rapid decompression, will be exposed to an increased risk of DCI as bubbles in the tissues and circulation will rapidly increase in size on decompression in accordance with Boyle's Law. This may lead to rapid onset of symptoms directly or may facilitate transfer of gas to the arterial circulation.

Effect of Breathing Gas

A greater number of subjects breathing a nitrogen/oxygen gas mixture than breathing 100% oxygen exhibited VGE at 25,000 ft. Although the mean onset times to VGE in these subjects were similar, statistical analysis did show that the gas mixture significantly reduced the time to grade 4 VGE formation of the whole group exposed to 25,000 ft. These results are similar to those reported by Webb & Pilmanis (1993) who found that 100% oxygen significantly reduced the number of subjects exhibiting VGE at 16,500 ft, and below, compared with those breathing a 50:50 oxygen/nitrogen gas mixture. In contrast, however, there was no significant difference in the development of symptoms between the groups exposed to 25,000 ft breathing 100% oxygen or nitrogen/oxygen gas mixture.

Effect of Preoxygenation

Denitrogenation, by breathing 100% oxygen, results in a downward gradient of the partial pressure of nitrogen between the bloodstream and the alveoli as the nitrogen content in the lung falls. Nitrogen diffuses out of the bloodstream into the lungs across the downward gradient. In turn, as nitrogen poor blood passes through the tissue with high nitrogen content nitrogen passes from the tissues into the bloodstream. Thus, as the nitrogen content of the tissues falls the risk of formation of nitrogen bubbles in the tissues at altitude is reduced, and in turn, this is considered to reduce the risk of DCI symptoms.

The use of oxygen breathing prior to decompression to reduce the incidence of decompression illness was first demonstrated in the 1940s (Boothby *et al* 1940 and Gray *et al* 1946). More recently, it has been demonstrated that the formation of bubbles in the circulation and the manifestation of symptoms of DCI can be substantially reduced by breathing 100% oxygen at ground level and at altitudes up to 16,000 ft (Pilmanis & Olson 1991). This study showed that denitrogenation for one hour prior to ascent to 25,000 ft significantly reduced the onset of symptoms – with 60% demonstrating symptoms without denitrogenation compared with 0% with denitrogenation. These results contrast with those of Webb & Pilmanis (1995) who predict that one hour of prior denitrogenation would reduce the incidence of DCI at 25,000 ft by just 15% (from 90% to 75%). This discrepancy, however, may be due to the slight differences in the protocol with regard to activity during the altitude exposure. The one hour of denitrogenation prior to exposure to 25,000 ft also reduced the number of subjects exhibiting VGE from 87% to 33% and increased the latency to the onset of VGE from a mean time of 52 to 123 minutes.

Effect of Altitude

Increasing altitude from 25,000 ft to 35,000 ft had a significant effect on the time to VGE and symptom onset (although not on grade 4 VGE onset). Figure 8 shows mean times to onset of symptoms at 25,000 ft, 30,000 ft and 35,000 ft with denitrogenation, and 25,000 ft without prior denitrogenation. As for all statistical analysis applied to these data, it has been assumed that for subjects who did not develop symptoms during altitude exposure, symptom onset could have occurred at 240 minutes. This of course represents the worst possible case. The linear functions have been fitted to the mean time to symptom onset with denitrogenation. It has then been assumed that this equation holds under conditions of no denitrogenation, and the linear function has been applied to the data from subjects exposed to 25,000 ft without denitrogenation. This provides an estimate of the mean times to the onset of symptoms at altitudes above 25,000 ft if no denitrogenation was employed. With all these assumptions it can be estimated that mean time to symptom onset at 30,000 ft and 35,000 ft, with no prior denitrogenation, would be 135 and 99 minutes respectively.

Pilmanis & Stegmann (1991) collated DCI data from the Armstrong Laboratory DCI database together with published results from between 1944 and 1951, all studies included employed no greater than mild exercise. They concluded that, with no denitrogenation, the mean onset time for decompression illness at 35,000 ft would be about 35 minutes. Pilmanis & Stegmann (1991) concluded further that with 1 hr of denitrogenation the mean onset time to DCI symptoms at 35,000 ft would be about 82 minutes. This compares with this study where a mean onset time with denitrogenation of 102 minutes was seen, and a mean onset time of 99 minutes without denitrogenation was predicted.

Extrapolation of the data presented by Pilmanis & Stegmann (1991) suggests that, with no denitrogenation, the onset of DCI symptoms would be instantaneous at 40,000 ft. While with denitrogenation extrapolation suggests that mean onset time at 40,000 ft would fall to about 45 mins while onset would be instantaneous at about 48,000 ft. This however, is not confirmed by Fraser *et al* (1994) who saw no cases of DCI in 8 subjects who were rapidly decompressed to an altitude of 60,000 ft which was maintained for 3 minutes. Similarly, Gradwell (1993) conducted 135 human rapid decompressions to between 45,000 ft and 60,000 ft with up to 45 minutes of denitrogenation and up to two minutes exposure to the final altitude with no reported cases of DCI. Despite these data from rapid decompressions there is no evidence to confirm the suggestion that there is a critical period of time, at any altitude, before which symptoms of decompression illness will not occur (Pilmanis & Stegmann, 1991).

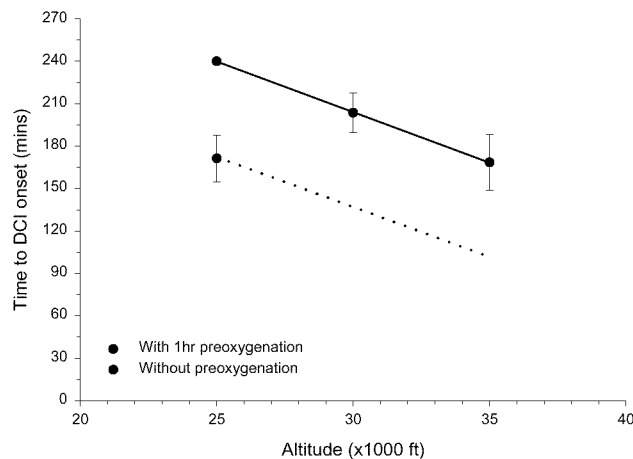


Figure 8. Linear regression applied to mean times to onset of DCI at 25,000 ft and above with 1 hour preoxygenation. An estimation has been made of times to onset of DCI using the mean time to DCI at 25,000 ft without preoxygenation.

Effect of Exercise

It has been demonstrated that even relatively modest increases in muscular activity increase the incidence of VGE and DCI symptoms. Krutz & Dixon (1987) demonstrated that five knee bends and five upward extensions of the arms with a 5lb weight every 15 minutes increased the number of subjects with symptoms at 30,000 ft, from 57% at rest (with limb flexion only) to 86%. Similarly, Ferris & Engel (1951) showed that while 55% of resting subjects reported symptoms at 35,000 ft, with a mean onset time of 61 minutes, 100% of subjects performing five knee bends every three minutes reported symptoms with a mean onset time of only 16 minutes. These authors also noted that the site of pain was often the site subjected to the maximum stress of exercise. Fryer (1969) concluded that exercise appears to have the equivalent effect, on DCI risk, of increasing the altitude by 3,000 ft - 5,000 ft.

In this study none of the subjects exposed to 25,000 ft at rest, with prior denitrogenation, exhibited symptoms of DCI. This is in contrast to Webb & Pilmanis (1995) who reported that 75% of subjects exposed to 25,000 ft, with limited arm exercises, reported DCI. Similarly, in comparison with the present study, Webb & Pilmanis (1995) predicted that 90% of subjects would report DCI within four hours at 25,000 ft with no preoxygenation, which is substantially greater than the 60% incidence of DCI reported in this study at 25,000 ft, with no preoxygenation. Such a difference in the incidence of DCI between these studies may be a result of the differing level of activity of the subjects. Indeed, the present data compare well with the incidence of DCI reported by Krutz & Dixon (1987) at 35,000 ft who found that 57% of resting subjects reported symptoms.

Conclusions

VGE formation occurs at cabin altitudes that will be encountered by aircrew of future agile aircraft. The presence of such VGE is likely to increase the risk of DCI in the event of cabin depressurisation and the presence of VGE *per se* has potential to result in arterial gas emboli and development of neurological symptoms.

Denitrogenation for one hour prior to decompression provides effective protection against development of symptoms of DCI at 25,000 ft for subjects under resting conditions.

Exposure to 25,000 ft for four hours breathing either gas mix or 100% oxygen, without prior denitrogenation, incurs a substantial risk of developing symptoms of DCI.

These data suggest that personnel exposed to 30,000 and 35,000 ft, at rest, are at risk of developing DCS even with one hour prior denitrogenation.

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