First Aid Oxygen Treatment for Decompression Illness in the Goat After Simulated Submarine Escape

Geoff A. M. Loveman; Fiona M. Seddon; Karen M. Jurd; Julian C. Thacker; Arran S. Fisher

BACKGROUND: Personnel responding to a distressed submarine incident require information on likely casualty levels and the severity and progression of decompression illness (DCI). Recompression may not be immediately available. First aid oxygen (FAO_2) can be administered; however, there is no direct evidence of its efficacy in this scenario.

METHODS: Trials were conducted between 2004 and 2006. Goats exposed to raised pressure for 24 h ('saturation') were either returned directly to atmospheric pressure (Phase A, $N = 40$) or exposed to simulated submarine escape at a depth of 656 ft (200 m; assumed seawater density $= 1019.72\ kg \cdot m^{-3}$; Phase B, $N = 39$). The pressure during saturation was selected to provoke 50% DCI. Cases of DCI were randomly assigned to receive FAO_2 or air.

RESULTS: DCI cases were: limb pain in 39 subjects, neurological in 6, respiratory in 4, and pulmonary barotrauma in 1 subject. In Phase A, 5/12 subjects in the FAO_2 group and 0/11 in the air control group achieved permanent resolution of DCI. In Phase B, 6/8 subjects in the FAO_2 group and 5/8 in the air control group achieved permanent resolution. In both Phases, levels of venous gas bubbles reduced sooner with FAO_2. Of three cases of neurological DCI receiving FAO_2, two showed permanent resolution. In total, four cases of respiratory DCI occurred; none of these resolved, with three being treated with FAO_2 and one in the air control.

DISCUSSION: Oxygen can be an effective first aid measure for DCI following submarine escape. However, it should not be used as a replacement for recompression therapy.

KEYWORDS: surface oxygen, Doppler, distressed submarine, DISSUB, decompression illness.

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Evacuation of a UK Royal Navy submarine is possible via a small airlock, the escape tower. The crew may be exposed to raised pressure within the distressed submarine and subsequently in the escape tower. Rapid return to normal atmospheric pressure puts the crew at risk of suffering decompression illness (DCI). Information on likely casualty levels and the severity and progression of DCI would be valuable for the medical team responding to a distressed submarine event should recompression facilities not be immediately available.

Following World War II, considerable work was carried out in order to determine rates of escape tower pressurization and subsequent ascent that might be safely achievable.[10] These early trials ignored the possible increase in likelihood and severity of DCI due to exposure to raised pressure within the submarine prior to escape. This issue was addressed in later trials described by Bell et al. using goats.[2] These later trials defined combinations of submarine pressure and escape depth which would result in a rate of occurrence of DCI of 50%. For example, Bell et al. state that exposing goats to a pressure of 2.0 bar (200 kPa) for 17 h, followed by a simulated submarine escape from 697.2 ft (212.5 m), resulted in DCI being observed in 50% of animals, although confidence limits were not given. In fact, the 50% DCI rate was based on 6 cases of DCI occurring in 12 subjects (unpublished data), which means the observed rate of DCI could have been stated as $50 \pm 30\%$ (Clopper Pearson 95% confidence interval for a sample from the binomial distribution). The UK Ministry of Defense required improved definition of the relationship between the distressed submarine internal pressure (the saturation pressure), the depth from which escape

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is made (the escape depth), and the risk of DCI. Subsequent trials investigating DCI in goats following simulated submarine escape have been previously summarized.[32,36] This paper presents details of a subset of those trials. Prior to conducting this subset of trials, data were available from 165 previously completed 24-h exposures of goats to raised pressures between 1.5 and 3.59 bar. Based on these data, direct return to 1.0 bar following 24 h spent at a pressure of 2.2 bar was predicted by logistic regression to give a probability of DCI of 50.7 ± 24% (95% CI).

Following submarine escape, the first external help that submariners are likely to receive will be from the Royal Navy Submarine Parachute Assistance Group, who are equipped to administer 100% oxygen as a first aid measure (FAO_2). In the absence of a recompression chamber, the administration of oxygen, fluid therapy, and an antiplatelet agent are generally indicated in the treatment of DCI.[6] A study carried out for the UK Health and Safety Executive concluded that there is evidence that normobaric oxygen protects divers against the progression of neurological DCI symptoms and may compensate for delays in administering hyperbaric oxygen.[33] In recreational diving, FAO_2 has been found to increase recompression efficacy and decrease the number of recompression treatments required if given within 4 h of surfacing.[25] At a workshop held to discuss management of mild DCI in remote locations, a consensus was reached that some patients with mild symptoms and signs after diving can be treated adequately without recompression[27] and administration of 100% oxygen has formed the basis of a nonrecompression treatment pathway for the management of scientific diving operations in the Canadian high Arctic.[31] However, the efficacy of FAO_2 for the treatment of DCI following submarine escape has not been previously reported.

The trials described here were aimed at determining the simulated saturation pressure which would result in a 50 ± 10% occurrence of DCI in the goat following direct return from saturation to 1.0 bar and also for saturation followed by a simulated submarine escape from a depth of 656 ft (200 m; assumed seawater density = 1019.72 kg · m^{-3}). This provided the opportunity to investigate the efficacy of treatment of the resulting cases of DCI using FAO_2 without recompression.

METHODS

Animals

This study was conducted under UK Home Office License according to the Animals (Scientific Procedures) Act 1986. The experimental protocol for the study was reviewed internally by an Ethics Review Committee (protocol number: 042218). The animals used were female or castrated male adult goats weighing 41.0 kg to 81.0 kg, mean (SD) 57.0 (10.4) kg. The goats were maintained under the surveillance of a veterinary surgeon and an animal care welfare officer and certified in good health prior to use.

Equipment

Pressure exposures took place in a purpose-built hyperbaric chamber designed to allow simulation of submarine escape. Audio Doppler bubble detection was carried out using TSI Doppler Bubble Monitor 9008 (Techno Scientific Ltd., Concord, ON, Canada). Mass spectrometry was carried out using a QP-9000 quadrupole mass spectrometer (Airspec/Morgan Medical Ltd., Rainham, Kent, UK). Expired respiratory gases and respiratory rates and volumes were monitored using Servomex fast-response oxygen and carbon dioxide analyzers (1400 series; Servomex, Sugarland, TX) and a flow meter (Kozak Turbine Compensator, KTC-3-D, KL Engineering, Van Nuys, CA) attached to an oro-nasal mask. The gas analyzers were dual-point calibrated using alpha gravimetric certified calibration gases supplied by BOC (Guildford, Surrey, UK). Calibration of the flow meter was carried out using a 3-L spirometer syringe.

Procedure

By conducting logistic regression on data already available and adding simulated data, it was predicted that 40 animal exposures would be required to reduce the width of the 95% CI on the predicted 50% DCI saturation exposure to ± 10%. Animals were exposed to raised pressure, initially of 2.2 bar, for 24 h and then either returned directly to atmospheric pressure (Phase A) at a rate of 1 bar · min^{-1} or exposed to a pressure profile simulating submarine escape at a depth of 656 ft (200 m; Phase B). The simulated 656-ft (200-m) escape consisted of pressurization from the saturation pressure to 21 bar in 28 s with a 4-s hold at the maximum pressure followed by a linear decompression to 1.0 bar at a rate of 0.275 bar · s^{-1}. **Fig. 1** shows example pressure/time profiles for Phases A and B.

For the first two exposures in each phase, animals were used in pairs. Following this, animals were exposed in groups of three. After each pressure exposure, a new 50% DCI saturation pressure was estimated based on the accrued data using logistic regression and was used for the next exposure.

A total of 63 goats were used in 79 animal exposures. There were 16 animals used in Phase A that were reused in Phase B. If

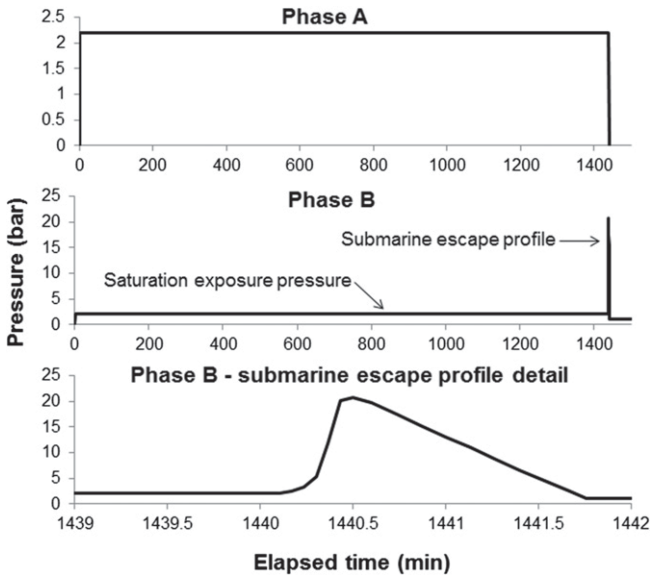

Fig. 1. Example of pressure/time profiles for Phases A and B.

an animal experienced DCI but made a full recovery, it was returned to the herd. There was a gap of 6 mo between Phases A and B.

Following the pressure exposures, animals were observed in an open pen next to the chamber while monitoring procedures were carried out. The following diagnostic criteria were used for the different types of DCI:

- Limb: Limb lifted off the ground, pawing or stamping, walking with a limp.
- Neurological/central nervous system (CNS): Unsteadiness, swaying, collapse, arching of back.
- Respiratory: Fast shallow breathing, increased heart rate, raspy breathing.
- Pulmonary barotrauma: Rapid onset collapse within 2 min, loss of consciousness.

Monitoring was performed for an 8-h period and further observations made intermittently for 24 h following a pressure exposure. Animals that showed signs of DCI were randomly assigned to a FAo_2 group or an air control group.

Air or 100% oxygen was delivered via plastic tubing to a transparent plastic hood with a latex neck seal (Sea-Long Medical Systems Inc., Louisville, KY). Mass spectrometry was used intermittently to give an indication of the actual levels of oxygen being administered. The animals wore the hoods for 1-h periods with the hood being removed for a 10-min air break between each hour. The air breaks allowed assessment of the animals' condition and walking gait, respiratory monitoring, and offering of rehydration fluids (Effydral™, Pfizer Ltd., New York, NY). Air or oxygen administration was continued for 1 h after resolution of all signs. If signs of DCI had not resolved after 3 h then the treatment was ceased: thus, the animals wore the hoods for treatment for a maximum of 4 h.

Careful and constant observation was maintained and all progression or resolution of signs recorded. Pain relief and/or sedation was available, but every attempt was made not to mask the progression of signs. If an animal's signs had not resolved after 3 h, it was humanely killed by a Home Office approved (Schedule 1) method.[20]

Audio Doppler bubble detection was carried out before and after each pressure exposure. The animals' left precordial area was shaved prior to the exposure to allow better contact of the Doppler transducer. Ultrasound transmission gel (Aquasonic 100™, Parker Laboratories Inc., Fairfield, NJ) was liberally applied to improve contact between the transducer and the skin. The precordial site was monitored and a bubble grade assigned according to the Kisman and Masurel method ('KM grade').[22] Doppler monitoring was completed at 2, 5, and 15 min, then at 15-min intervals to 2 h, and then every hour until 8 h post-exposure or until bubbles ceased. Respiratory monitoring was carried out at 20 min and 1, 2, 3, and 4 h post-exposure.

Statistical Analysis

Nonparametric data were compared using the median and Mann-Whitney *U*-test. Parametric data were compared using the mean and Student's *t*-test. The effect of FAo_2 compared to air breathing controls was compared using Fisher's Exact Probability Test. Differences were considered significant if $P < 0.05$. Where data were compared for individual time points the *P*-value for significance was adjusted using the Bonferroni correction.

RESULTS

A total of 40 animal saturation only exposures were carried out in Phase A. The range of saturation pressure was 2.2 to 2.35 bar, mean [SD] 2.28 [0.04] bar. Of these 40 animals, 23 showed signs of DCI: 12 animals were assigned to the Phase A FAo_2 group and 11 to the air control group. Oxygen level in the hoods during the FAo_2 treatments ranged from 91.5 to 99.3% with a mean of 96.6%.

Results are summarized in **Table I**. The shortest onset time for DCI was 4 min post-exposure and the longest was over 5 h,

Table I. Summary of Results for Trial Phases A and B.

	PHASE A - SATURATION ONLY		PHASE B - SATURATION AND 200-m ESCAPE		
Saturation pressure (bar)	2.28 (0.04)		2.14 (0.05)		
DCI/*N*	23/40		20/39		
Treatment type	**FAo_2 (*N* = 12)**	**Air (*N* = 11)**	**FAo_2 (*N* = 8)**	**Air (*N* = 8)**	**No treatment (*N* = 4)**
Body mass (kg)	59.2 (11)	62.7 (10)	52.2 (4)	53.0 (13)	56.2 (3)
Saturation pressure (bar)	2.29 (0.04)	2.30 (0.03)	2.16 (0.03)	2.15 (0.05)	2.20 (0.03)
DCI type	10 L 1 L + R 1 CNS + L	10 L 1 L + R	5 L 1 L + R 1 L + CNS 1 CNS + L + R	8 L	3 CNS 1 PBT
DCI onset (min)	38 (27)	88 (95)	30 (20)	28 (13)	11 (6)
Treatment delay time (min)	14 (12)	18 (8)	9 (3)	11 (6)	N/A
Duration of oxygen treatment (min)	156.9 (53)	N/A	166.7 (54)	N/A	N/A
Resolution (observed cases)	6	3	6	6	N/A
Time to resolution (min)	115 (52)	365 (162)	121 (70)	255 (135)	N/A
DCI recurrence	1	3	0	1	N/A

200 m = 656 ft. FAo_2 = first aid oxygen; L = limb pain; R = respiratory DCI; CNS = central nervous system DCI; PBT = pulmonary barotrauma.
Where mean values are given, the standard deviation is shown in parentheses; where multiple signs of DCI were observed in an individual animal, this is indicated in the row labeled 'DCI type,' for example: '1 CNS + L + R' indicates an individual animal with signs of CNS, Limb pain and Respiratory DCI.

with most signs appearing in the first hour. The most common sign noted was limb lifting associated with limb pain, with more than 1 limb affected in 10 animals. One animal displayed signs consistent with mild CNS DCI: it had a hunched posture and was clumsy and slow when changing from standing to lying positions. In two of the animals there were indications of pulmonary compromise, a sign of respiratory DCI. This was manifest as a rise in respiratory rate with a decrease in respiratory exchange.

Of the 12 animals who received FAO_2, 5 achieved permanent resolution to their signs. The remaining 7 had residual or recurring signs. Of the 11 air control animals, 3 showed a resolution of signs, but this was temporary and the signs recurred. There was a significant difference between the effect of FAO_2 compared to air on the permanent resolution of signs of DCI ($P = 0.037$, Fisher's exact probability test).

A total of 39 animal exposures were carried out in Phase B. The range of pressure for the saturation exposures was 2.05 to 2.2 bar, mean 2.14 (0.05) bar, followed by simulated escape from 656 ft (200 m). Of these 39 animals, 19 showed signs of DCI and there was also 1 case of pulmonary barotrauma. The most common sign noted was limb lifting, with more than one limb affected in four animals. Of the 19 with DCI, 3 presented with CNS signs that were severe enough to require an early humane end point without assignment to either the air or FAO_2 group. The remaining 16 animals were randomly assigned to the FAO_2 ($N = 8$) or air control group ($N = 8$). In two of the animals there were indications of respiratory DCI. In the FAO_2 group, 6/8 achieved complete resolution of their signs: the remaining two had residual respiratory signs. It is worth noting that CNS signs occurred in two animals in the FAO_2 group which resolved with treatment. In the air control group, 5/8 also showed complete resolution of signs. All DCI cases in the air group were limb pain only, two were unresolved and one recurred overnight. There was no significant difference in the level of resolution or mean time to resolution between the air and FAO_2 groups in Phase B.

Doppler measurements were continued after a diagnosis of DCI had been made and while the animal was being treated with FAO_2 or air. **Fig. 2** shows the median KM grades for Phase A. There was no significant difference between median KM grades for DCI versus no DCI. KM grades decreased to zero by 8 h post-exposure in the animals without DCI and in those in the FAO_2 group. The grades began to fall earlier in the FAO_2 group than the air control group, with a significant difference between the two groups at 90 min post-exposure ($U(21) = 111$, $z = -2.74$, $P = 0.006$, Mann-Whitney *U*-test, two-tailed).

Fig. 3 shows the median KM grades for Phase B. As in Phase A, median KM grades in Phase B were not significantly different for animals with DCI compared to those without. The KM grades had decreased to zero in 13 of the 19 animals without DCI by the end of the 8-h monitoring period. Fig. 3 shows that the median KM grades began to fall earlier in the FAO_2 group than the air control group. At 300 min there was a significant difference between the KM grades for the groups ($U(10) = 33.5$, $z = -2.4$, $P = 0.02$, Mann Whitney *U*-test, two-tailed). The

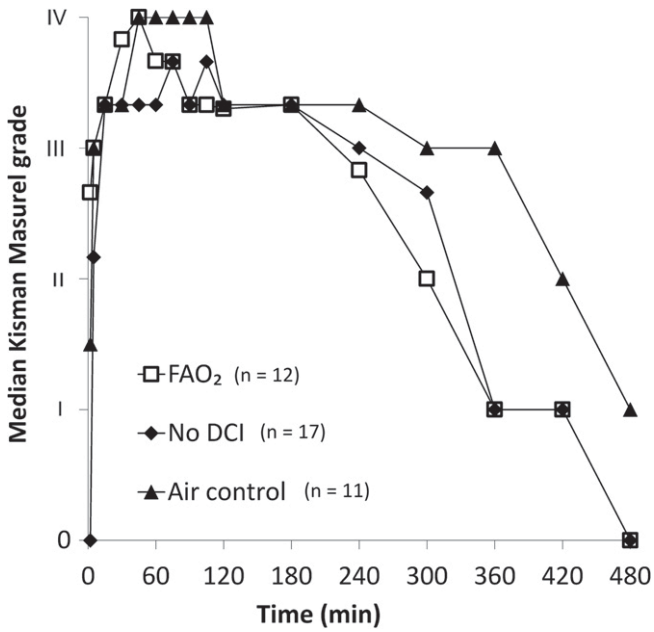

Fig. 2. Phase A median Kisman Masurel grade for no DCI, F_{AO_2}, and air control groups.

median KM grade had dropped to zero by 7 h in the group without DCI and in the FAO_2 group.

The mean onset times to the first signs of DCI were significantly shorter for Phase B than for Phase A ($t(25) = 2.32$, $P = 0.029$, Student's *t*-test, two-tailed, unpaired, unequal variance). There was a significant difference between the median KM grades at 2, 5, and 15 min in Phase A compared with those in Phase B [2 min: $U(63) = 856.5$, $z = -4.32$, $P < 0.0001$; 5 min: $U(71) = 1065$, $z = -4.4$, $P < 0.0001$; 15 min: $U(74) = 1061.5$, $z = -3.55$, $P = 0.0004$; Mann Whitney *U*-test, two-tailed].

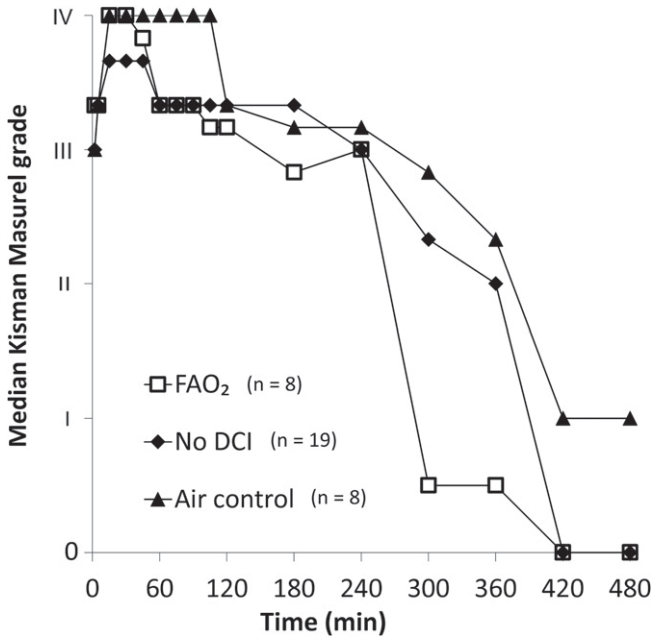

Fig. 3. Phase B median Kisman Masurel grade for no DCI, F_{AO_2}, and air control groups.

DISCUSSION

Based on the results obtained from the trials described here, a saturation pressure of 2.24 bar followed by direct ascent was predicted to provoke an incidence of DCI of 50 ± 10% based on logistic regression. The pressure which will provoke an incidence of DCI of 50 ± 15% after a 24-h exposure with a 656-ft (200-m) simulated submarine escape was estimated to be 2.14 bar. These data have been included in the calibration of a model for the estimation of risk of DCI following submarine tower escape, details of which have been previously published.[26]

No permanent resolution of DCI was seen in the Phase A air control group, whereas signs in the FAO_2 group completely resolved in five animals; this was statistically significant ($P = 0.037$). This indicates that oxygen is an effective first aid measure in resolution of DCI following direct return to surface pressure from shallow air saturation exposures.

It is worth comparing the types and onset times of DCI observed in the two phases of the trial. From the saturation only phase (Phase A), there was a single case of CNS DCI, which was treated with FAO_2 with no resolution. For saturation with a 656-ft (200-m) escape, there were five cases of CNS DCI, three of which were early onset and severe. The other two cases of CNS DCI were treated with surface oxygen, one of which resolved completely, and the other case saw resolution of the CNS signs, but also had respiratory DCI signs which did not resolve. The mean time to resolution of signs for the FAO_2 group in Phase A was 115 min, but there was no resolution in the air control group. In Phase B there was no significant difference between the mean time to resolution of signs in the FAO_2 group (120 min) and the air control group (255 min).

An explanation for the different types and severity of DCI is that deeper saturation exposures (2.24 bar) were required to provoke 50% DCI with no escape. The escape from 656 ft (200 m) increases the risk of DCI, therefore the saturation pressure must be slightly reduced (to 2.14 bar) to induce the same 50% risk. Unresolved limb pain was the predominant DCI type in Phase A, possibly due to the deeper mean saturation pressure of Phase A. Following direct ascent from saturation, a steep increase in likelihood of DCI with saturation pressure is well documented in the literature.[24] The lower mean saturation pressure of Phase B apparently gave rise to limb pain DCI cases that were more successfully treatable than those in Phase A, but the addition of the 656-ft (200-m) simulated escape profile increased the risk of CNS DCI. This effect might be due to uptake of nitrogen primarily in the CNS during the rapid pressurization to 21 bar and in the first part of the simulated ascent. Other, less well perfused tissues would not necessarily be affected, since they would take up little nitrogen in the relatively short duration of the escape exposure. The level of respiratory DCI observed was the same in both phases.

The mean onset times to the first signs of DCI were significantly shorter for Phase B than for Phase A ($P = 0.029$). KM grades in Phase B were also higher than those in Phase A for the first 15 min post exposure. These results suggest that the initial high level of circulating venous gas bubbles observed in Phase B was due to the effect of the 656-ft (200-m) escape exposure and it was this that gave rise to earlier cases of DCI and increased levels of CNS signs. Shorter latency times for detection of circulating bubbles have been associated with increased risk of DCI in other studies[21,23] and shorter onset times to signs of DCI are associated with a requirement for a greater number of recompression treatments and poorer long-term outcome.[15,16]

In both phases the KM grades decreased more rapidly after 240 min in the FAO_2 group. This was likely a result of the low partial pressure of inspired nitrogen giving rise to a faster washout of nitrogen from the body, an example of the effect of the 'oxygen window.'[19] Commencing oxygen breathing immediately upon surfacing, rather than waiting for signs of DCI, after simulated submarine escape has also been shown to reduce KM grades more rapidly than breathing either air or a 5% carbon dioxide in oxygen mixture.[18]

It is worth noting that for the three early onset cases of severe CNS DCI from Phase B, the Doppler technician reported in each case that the level of bubbling 2 min post-exposure (and before the CNS events occurred) was extremely high. Eftedal et al. have described a bubble grading system for use with ultrasound imaging in which the highest level of bubbles, where single bubbles cannot be discriminated in an image, is referred to as 'white-out.'[14] They state that they have observed near 100% mortality in animals with this level of bubbles. In our experience, a shortfall in the KM scoring system is that grade IV, the maximal grade that can be assigned, does not allow for differences in extremely high bubble grades which can be discerned by an experienced technician.

Initially selected for use due to their amenable nature and being the largest animal readily available, goats have been shown to display a range of easily recognizable signs of DCI which correspond with signs in man.[7] Interspecies response to decompression from saturation has been shown to be scalable using body mass as a scaling factor.[3] **Fig. 4** shows a logistic regression fit to DCI outcome data from the 205 goat saturation exposures available at the conclusion of the trials described here. (Data from 165 saturation exposures were available prior to the trials described here. Only the 40 exposures from Phase A were added in the regression). Saturation pressure and body mass were used as factors in the regression model. Coefficients of the regression model are given in **Table II**.

It should be noted that both the magnitude of the standard error and the *P*-value for the coefficient of body mass in the model indicate that inclusion of body mass as a factor did not significantly improve the fit of the model. Body mass has been included as a factor in the model nevertheless since, as already stated, risk of DCI has previously been shown to be scalable with body mass.[3] The regression plot shown in Fig. 4 was made for goats with a body mass of 80 kg (estimated 95% confidence limits are shown in gray). Also included in Fig. 4 is a plot of predicted risk of DCI using a model described by Van Liew and Flynn which was fit to DCI data from human no-stop decompression exposures.[34] Fig. 4 suggests that estimation of risk of DCI in humans following saturation exposures can reasonably

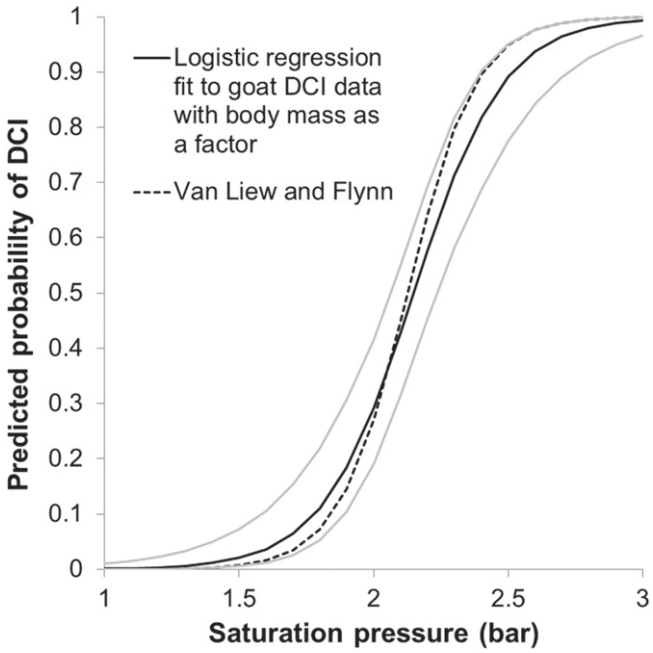

Fig. 4. Logistic regression fit to goat DCI data following direct ascent from saturation shown with a fit to human data from Van Liew and Flynn.[34] Body mass was included as a factor in the goat model and the plot shown is for an 80-kg animal; the gray lines show the extent of the estimated 95% confidence limits.

be made based on goat DCI data from similar exposures using body mass as a scaling factor.

This finding is in agreement with Lillo et al., who have additionally shown that combining data from higher risk exposures tested in animals with data from lower risk exposures in man allows improved prediction of the risk of DCI for humans where high risk data are sparse.[24] Details of the use of these principles to provide a model for the prediction of DCI following submarine tower escape based on the combination of available data for both man and goat have been previously published.[26] Comparison of DCI data in goats and humans for the rapid pressurization and decompression of a submarine escape exposure is more difficult due to the limited human data available. Histological examination has shown similar lesions in the spinal cord of man and goats following spinal DCI.[28] However, there is also evidence to suggest that the goat may suffer fewer decompression-induced cerebral lesions than man.[29] This may be due to differences in the cerebral vasculature between the two species or possibly due to a postulated higher prevalence of patent foramen ovale in man, which could allow gas bubbles to pass into the arterial circulation via venous-arterial shunt in the heart.[5]

Levels of circulating venous gas bubbles following direct ascent from a range of saturation pressures have been measured in man by Eckenhoff et al.[12,13] Based on these observations, Eckenhoff et al. generated models for predicting the probability of observing a peak KM grade greater than each of the bubble grades 0, I, II, and III. These models for predicting levels of venous bubbles in man may be compared with our measurements in the goat. **Fig. 5** shows the observed fraction of animals in Phase A (FAO_2 group excluded) with peak KM grades higher than grade II and the observed fraction with peak grades higher than grade III. All animals in this group ($N = 28$) had a peak KM grade higher than grade II. The two points are plotted at the mean saturation depth for these exposures, which was 2.24 bar. The maximum saturation pressure used by Eckenhoff et al. in their manned trials was 1.92 bar, as indicated by the dashed vertical line in Fig. 5. The extrapolated predictions for man at 2.24 bar are within the 95% binomial confidence limits of the two points plotted for the goat. This suggests that peak KM grades observed in the goat following direct ascent from saturation may follow a similar pattern to those observed in man.

It should be noted, however, that Eckenhoff et al. used KM grades observed in man both at rest and also following a 'flex' movement (a deep knee-bend) and took the peak of these, which generally occurred after the flex movement.[13] The goats were not induced to make any movement akin to the flex movement. Therefore, the peak grades for the goats might be anticipated to be somewhat lower than those observed in man.

Given the evidence as discussed, it would appear that the response of the goat to decompression stress is not grossly dissimilar, at least, to that observed in man. Assuming the response

Table II. Summary of Logistic Regression Fit to Goat DCI Data Following Saturation Exposures.

COEFFICIENTS	ESTIMATE	STD. ERROR	*z*-VALUE	$P(>\|z\|)$
Intercept	−14.6	2.4	−6.1	1.4e-09
Saturation pressure (bar)	5.99	0.97	6.2	7.2 e-10
Body mass (kg)	0.02	0.02	0.9	0.4

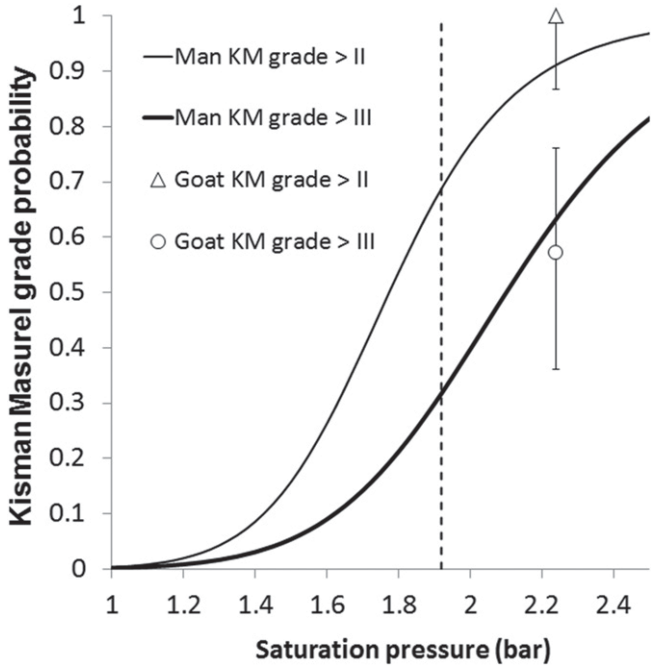

Fig. 5. Observed fraction of subjects in Phase A (FAO_2 group excluded) with peak KM grade > II ($N = 28$) and with peak KM grade > III ($N = 16$); error bars are the 95% exact binomial confidence limits. The solid lines are fits made to human venous gas bubble data from Eckenhoff et al.[12] The dashed line shows the upper limit of saturation pressure at which Eckenhoff et al. conducted manned trials.

to be similar, then the saturation pressure which elicits 50% DCI results in signs which the authors suggest would not be expected to cause death or prevent a submariner from boarding the one-man life raft that they are equipped with. These signs would be expected to resolve on recompression treatment in the majority of cases.[1] This finding contradicts to some extent the expectations described by Weathersby[35] that in the region of 50% DCI risk there would be a range of DCI that may lead to death in some and permanent disability without immediate recompression therapy in most people. Observations from our previous studies, both manned[21] and animal[32] indicate that while this looked unlikely for saturation alone, or saturation with a shallow escape, it may be true for saturation dives with deeper escapes. The cases of CNS DCI that we encountered in Phase B were severely disabling and probably would have prevented the successful rescue of some individuals. In retrospective studies, severe symptoms of neurological DCI have been found to be associated with long-term sequelae even when recompression is available, with time to treatment being found to have either no influence or at most a weak association with likelihood of full recovery.[4,17] In the distressed submarine scenario, therefore, if medical assistance and recompression were available then the likelihood of survival would be high, but possibly with some respiratory or neurological sequelae. However, it is also possible that some apparently severe neurological DCI following submarine escape may resolve spontaneously. A previous study has described this phenomenon, with cases of neurological DCI after simulated submarine escapes having been observed to resolve untreated after around 20 min.[11] The authors postulated that short-duration, high-pressure exposures typical in submarine escape might give rise to venous bubbles with a high oxygen content. This could occur since, with air as the breathing gas during the escape procedure, the inspired partial pressure of oxygen will be as much as 4.0 bar for escape at a depth of 590.6 ft (180 m). Since the procedure is so rapid, there would be little time for this oxygen to be metabolized and, therefore, it could act as a contributor to the gas content of any bubbles that form, a possibility that has been modeled by Parker et al.[30] These bubbles could resolve in a shorter time than bubbles with a higher inert gas content due to the metabolic use of oxygen in the tissues generating an increased gradient for oxygen diffusion out of the bubble. However, in a distressed submarine scenario, short-duration neurological DCI might still prove fatal, perhaps resulting in drowning, if support were not immediately available upon surfacing.

A further consideration in the context of a distressed submarine scenario is that the crew may be exposed to a raised partial pressure of oxygen within the submarine and that subsequent treatment with 100% oxygen on the surface or during recompression could exacerbate symptoms of pulmonary oxygen toxicity, which can be fatal.[8,9] Pulmonary oxygen toxicity remains an issue in both the treatment of DCI following escape and in the successful decompression of submariners following evacuation via a rescue submersible.

The trials described here show that oxygen can be an effective first aid measure in the treatment of DCI following submarine escape. However, the level of DCI signs that remained or recurred show that, although a useful adjunctive therapy, FAO_2 should not be considered as a replacement for recompression therapy as the gold standard in the treatment of DCI.

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