Physiological effects of rapid reduction in carbon dioxide partial pressure in submarine tower escape

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Abstract

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Introduction: The objective of this study was to determine whether adverse effects from a rapid drop in inspired carbon dioxide partial pressure (P_iCO_2) in the breathing gas could hinder or prevent submarine tower escape.

Methods: A total of 34 male volunteers, mean (SD) age 33.8 (7.5) years, completed the trial. They breathed air for five minutes then 5% CO_2/16% O_2, 79% N_2 ($5CO_2/16O_2$) for 60 minutes before switching to breathing 100% O_2 for 15 minutes and then returned to air breathing. Breathing gases were supplied from cylinders via scuba regulators and mouthpieces. Blood pressure, cerebral blood flow velocity, electrocardiogram and end-tidal CO_2 and end-tidal O_2 were monitored throughout. Subjects were asked at intervals to indicate symptom type and severity.

Results: Symptoms whilst breathing $5CO_2/16O_2$ included breathlessness and headache. Following the switch to 100% O_2 seven subjects reported mild to moderate faintness, which was associated with a significant drop in cerebral blood flow compared to those who did not feel faint ($P < 0.02$). No subject vomited or fainted following this breathing-gas switch.

Conclusions: This study shows that the risk of fainting, sudden collapse or vomiting on switching to 100% O_2 following acute exposures to hypercapnia at a P_iCO_2 of up to 5.0 kPa is less than 8%.

Key words

Hypercapnia, oxygen, cerebral blood flow, Doppler, physiology, submarine

Introduction

In a scenario where the crew of a UK Royal Navy (RN) submarine is unable to surface their vessel, they may attempt escape. The escape tower is an air-lock supplied with diving quality air. The submarine crew member will switch from breathing a possibly hypercapnic and hypoxic atmosphere in the distressed submarine (DISSUB) to a normocapnic (approximately 0.0395 kPa inspired carbon dioxide partial pressure, P_iCO_2) and normoxic atmosphere in the escape tower. Subsequent pressurisation during tower escape means that the escaper will also be exposed to a hyperoxic atmosphere, with the inspired oxygen partial pressure (P_iO_2) reaching as high as 380 kPa at the maximum permitted escape depth (180 metres' sea water, msw).

An early study reported that switching from breathing a hypercapnic gas (6% CO_2) to 100 kPa O_2 resulted in nausea and vomiting in three of six subjects.[1] The authors indicated that the work could have been better controlled. To our knowledge only one other study has examined the effect of this gas switch; using 7 kPa P_iCO_2, it was found that two of 12 subjects vomited shortly after the switch to oxygen breathing.[2] Cerebral hypoperfusion has been associated with nausea.[3] The reduction in cerebral blood flow associated with a rapid reduction in P_iCO_2 combined with a rapid elevation in P_iO_2 could induce nausea and vomiting, in addition to the risk of fainting. Vomiting during the pressurisation or ascent phase of submarine escape would likely result in pulmonary injury and possibly death. The term 'carbon dioxide-off' effect refers to any symptoms that might be experienced by an individual who has been exposed to a high level of CO_2 (a hypercapnic atmosphere) and then switches to breathing a normal (normocapnic) or reduced (hypocapnic) level.

Fainting can be provoked by anything that endangers cerebral perfusion.[4] The switch from breathing a hypercapnic gas in the DISSUB to a hyperoxic gas whilst stood in the submarine escape tower might lead to cerebral hypoperfusion, which could in turn result in fainting. A crew member who faints in the escape tower presents an additional obstacle for fellow crew members to negotiate and furthermore his airway could be compromised.

The purpose of the present study was to determine the risk to escapers with P_iCO_2 of approximately 5.0 kPa and P_iO_2 of approximately 16.0 kPa that might exist in the DISSUB when a switch is made to breathing 100 kPa O_2 (the maximum P_iO_2 that can be delivered under normobaric conditions and equivalent to that experienced in tower escape breathing air at approximately 40 msw tower depth).

Methods

The study was carried out at the QinetiQ Hyperbaric Medical Unit, St. Richard's Hospital, Chichester, UK and was conducted in accordance with the principles of the declaration of Helsinki.[5] An ethical protocol for the study was reviewed and approved by the QinetiQ Research Ethics Committee (approval number: SP774v2.3).

A power calculation (single-sample binomial test, two-tailed, power = 0.8 and $P = 0.05$) showed that 34 subjects would

need to complete a switch from a hypercapnic and hypoxic breathing gas to 100% O_2, without vomiting or fainting, to demonstrate the underlying risk to be less than 8%.

SUBJECTS

Volunteer subjects were requested to fast from 2000 h and to refrain from alcohol for 24 h prior to the morning of the test. They were asked to drink only clear liquids other than taking their usual caffeinated drink in the morning and not to consume any liquids for two hours prior to the test.

PROCEDURE

All tests were carried out at normobaric ambient pressure. A nose clip was worn throughout the test. Each subject sat at rest breathing air from a scuba mouthpiece for 5 min while baseline measurements were taken. The subject then breathed room air for a short period. The subject then commenced breathing a hypercapnic, hypoxic mixture for 60 min. The composition of the mixture was 5% CO_2, 16% O_2, 79% N_2, referred to here as $5CO_2/16O_2$.

A subjective symptoms questionnaire was administered each minute for the first 5 min of breathing $5CO_2/16O_2$, then after a further 5 min and then at 10 min intervals. The subject was required to rate their level of discomfort on a five-point scale – as none, mild, moderate, severe or intolerable – for four symptoms – nausea, breathlessness, faintness and headache.

At 50 min the subject was asked to stand. At 60 min the breathing gas was switched to 100% O_2 and the subjective symptoms questionnaire administered at 1 min intervals for 5 min, then at 2 min intervals. At 75 min the breathing gas was switched to air and the subject asked to sit. At 80 min the test was ended.

INSTRUMENTATION AND MEASUREMENTS

PCO_2 and PO_2 were measured continuously at the centre of the scuba mouthpiece (AMIS 2000 respiratory mass spectrometer, Innovision Denmark). Mean blood flow velocity in the middle cerebral artery ($MCAv_{mean}$) was measured continuously using transcranial Doppler (TCD) (TC-Pioneer EME/Nicolet Vascular), the probe being located at the temporal region above the zygomatic arch. Insonation of the MCA was adjusted to the angle resulting in the highest recorded blood velocity and best-quality Doppler signal.

At 1 min intervals for 5 min after changing breathing gas and 5 min intervals thereafter, mean arterial pressure (MAP) was measured with an automated sphygmomanometer (DINAMAP® Pro 1000, General Electric) at the brachial artery of the right arm. Electrocardiogram (ECG) was continuously displayed on two ECG monitors. The 3-lead monitor of the DINAMAP® Pro 1000 was used to allow display of the lead I signal and a 5-lead monitor (LifePulse10, HME Ltd.) was used to display the lead II signal.

TEST TERMINATION CRITERIA

The test would be terminated:

- at the subject's request;
- on a subjective questionnaire response of 'intolerable' to any aspect;
- on failure of any equipment used to monitor withdrawal variables;
- on recording end-tidal carbon dioxide ($ETCO_2$) > 8.5 kPa for more than five consecutive breaths;
- if the subject began to vomit;
- if the subject requested assistance as feeling severely faint or the subject fainted;
- on subjective signs of impending panic or
- if BP, measured by DINAMAP was greater than either a systolic of 180 or a diastolic of 110 mmHg, sustained for over 1 min.

STATISTICS

The relative percentage changes in respiratory rate, heart rate, MAP, $ETCO_2$ and $MCAv_{mean}$ were calculated for the minute pre-switch to the minute post-switch to 100% O_2. A boxplot was used to determine whether any of these data warranted further statistical analysis. Where this was the case, subject data were grouped according to symptoms and differences between groups tested using the unpaired, unequal variance *t*-test. Differences were considered significant if $P \leq 0.05$.

Results

SUBJECT DETAILS

A total of 39 male volunteers participated in the trial. The procedure was stopped in six subjects; three because they exceeded the upper BP limits, two whilst breathing $5CO_2/16O_2$ and one on 100% O_2 (the last subject's data were included in the analysis, however); two for increasing ventricular ectopics (not present on their pre-trial ECGs) whilst breathing $5CO_2/16O_2$ and one who was entraining room air around the mouthpiece during the test. The mean (SD) age of the 34 volunteers whose data were used was 33.8 (7.5) years; height 180.7 (5.7) cm; body mass 82.8 (9.1) kg.

SYMPTOMS

No subject vomited, fainted or was incapacitated on the switch to 100% O_2 breathing. Six subjects reported no symptoms throughout the test. Eleven subjects reported mild to moderate headache. Only three subjects reported a headache that developed after the switch to 100% O_2, as opposed to eight whose headache developed whilst breathing $5CO_2/16O_2$; three of these eight found their symptoms of headache resolved following the switch to 100% O_2.

Seven subjects reported mild to moderate faintness occurring only after the switch to breathing 100% O_2, while six reported

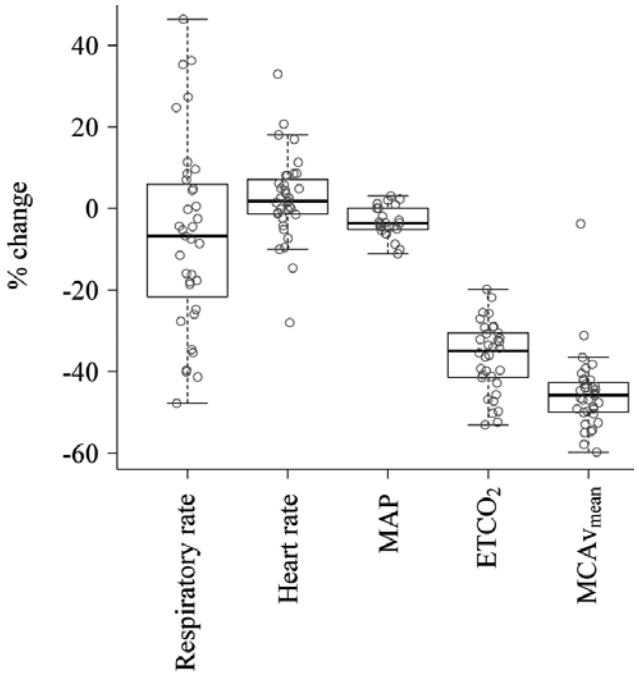
Table 1

95% confidence intervals on absolute values of physiological variables ($n = 34$); MAP – brachial BP was measured once per min, other signals were recorded continuously; * value taken over 1 min; † value taken over 5 min; $MCAv_{mean}$ – mean middle cerebral artery blood flow velocity

	Air baseline*	First min* $5CO_2/16O_2$	First 5 min† $5CO_2/16O_2$	After 30 min* $5CO_2/16O_2$	Final min* $5CO_2/16O_2$	First min* 100% O_2	First 5 min† 100% O_2	Final min* 100 % O_2	Final min air*
Respiratory rate (breaths·min^{-1})	8 ± 1.2	8.9 ± 1.2	9.1 ± 1.2	10.2 ± 1	12.7 ± 1.2	11.6 ± 1.4	9.8 ± 1.4	9.5 ± 2	8.5 ± 1
MAP (mmHg)	94 ± 2	99 ± 4	100 ± 4	97 ± 4	106 ± 4	102 ± 2	99 ± 2	98 ± 4	95 ± 2
Heart rate (beats·min^{-1})	66 ± 4	67 ± 4	67 ± 4	69 ± 4	82 ± 4	84 ± 6	82 ± 6	76 ± 4	65 ± 4
$MCAv_{mean}$ (cm·s^{-1})	52 ± 4	73 ± 6	77 ± 6	71 ± 6	72 ± 8	39 ± 4	38 ± 4	43 ± 6	45 ± 6
$ETCO_2$ (kPa)	5.2 ± 0.2	6.6 ± 0.2	6.9 ± 0.2	6.9 ± 0.2	6.8 ± 0.2	4.3 ± 0.2	4.1 ± 0.2	4.1 ± 0.4	4.4 ± 0.4

Figure 1

Percentage change in physiological variables taken over 1 min before and after switching to 100% O_2 ($n = 34$); heavy lines denote median values; box extents show interquartile range; whiskers denote data values within 1.5 times the interquartile range from upper/lower quartiles



mild faintness whilst breathing $5CO_2/16O_2$. Faintness was the only symptom which developed in an appreciable number of subjects following the switch to 100% O_2. One subject reported mild nausea after the switch to 100% O_2 and one reported mild nausea whilst breathing $5CO_2/16O_2$. Eighteen subjects reported mild to severe breathlessness whilst breathing $5CO_2/16O_2$. Three of these continued to experience breathlessness on the switch to 100% O_2, one of whom rated it as moderate.

CHANGE IN PHYSIOLOGICAL VARIABLES ON SWITCH TO 100% OXYGEN

The values of physiological variables are summarised in Table 1. Figure 1 shows a boxplot of the percentage changes in respiratory rate, heart rate, MAP, $ETCO_2$ and $MCAv_{mean,}$ taken from 1 min pre-switch to 1 min post-switch to 100% O_2. There was a significant difference in the percentage drop in $MCAv_{mean}$ on the switch between subjects experiencing faintness following the switch ($n = 7$, mean reduction in $MCAv_{mean}$ 51%) compared to those who did not ($n = 27$, mean reduction in $MCAv_{mean}$ 44%) ($P < 0.02$, unpaired, unequal variance *t*-test). The boxplot demonstrates no significant change in the mean respiratory rate, heart rate or MAP.

Discussion

SYMPTOMS WHILST BREATHING $5CO_2/16O_2$

Increased cerebral blood flow and headache and possible nausea related to increased intracranial pressure were anticipated in subjects whilst breathing $5CO_2/16O_2$, the symptoms of headache in eight subjects and one reported case of mild nausea were in accord with the findings of an earlier study.[1] Cerebral blood flow increases in the order of 50% when breathing 5% CO_2.[6] At 2.5% CO_2, there is no effect; at 3.5% a significant effect has been reported and at 7% the effect is far greater than at 5%.[6,7] After 5 min of breathing $5CO_2/16O_2$ in the present study, the $MCAv_{mean}$ increased by 49%, in agreement with these previous studies.

Moderate hypertension was recorded in all subjects whilst breathing $5CO_2/16O_2$, which has also been reported by others investigating the effect of an increased P_iCO_2.[8]

EFFECTS OF THE SWITCH TO 100% OXYGEN

Nausea and vomiting

No subjects vomited. One reported mild nausea which

developed after the switch to 100% O_2. Since one subject reported mild nausea whilst breathing $5CO_2/16O_2$, there is little or no evidence of a difference in the apparent effects of breathing $5CO_2/16O_2$ and the switch to 100% O_2 in terms of inducing nausea.

Some studies have not shown any evidence of incapacitation when switching from breathing a hypercapnic gas to air. Exposure to CO_2 at a concentration of 7% has been used as a tool to investigate panic and fear.[9] Neither sudden collapse nor vomiting was reported, although headache was, on return to air breathing. In another study, subjects were exposed inside a chamber to a PCO_2 of 1.3–5.6 kPa for 5 days, coming out of the chamber once each day to breathe air for 30 min. The study did not report any adverse effects on the subjects of switching between hypercapnia and air.[10] In the studies where adverse effects were reported, the P_iCO_2 was higher.[1,2] It appears that a CO_2-off effect that causes vomiting when switching to 100% O_2 following acute (~ 1 h) exposures to hypercapnia may only become apparent when switching from a P_iCO_2 above 5.0 kPa and that the severity may rapidly increase with only slight further increases in P_iCO_2.

Headache

In the current study, only three subjects reported headache that developed after the switch to 100% O_2, with the majority of subjects that experienced headache (8 of 11) having symptoms developing whilst breathing $5CO_2/16O_2$. Thus the exposure to $5CO_2/16O_2$ was more likely to induce headache than the switch to 100% O_2. The resolution of symptoms in three subjects following this switch suggests that it was at least as likely to reduce as to provoke headache.

Faintness

Faintness (mostly mild) was the most frequent symptom reported following the switch from hypercapnia to breathing 100% O_2. This occurred in seven subjects where faintness was not reported prior to the switch. There is some controversy over whether administration of 100% O_2 can maintain cerebral oxygenation in spite of hypoperfusion. It has been argued that hyperoxic hyperventilation and hypocapnia could decrease cerebral blood flow in excess of the effect of the increased O_2 content of breathing gas and paradoxically diminish O_2 delivery to the brain.[11] However, other authors have presented evidence that any likely effect of hypoperfusion (such as inducing fainting) caused by breathing 100% O_2 would be offset by the increased blood O_2 tension.[12] A clear independent cerebral vasoconstrictive effect of hyperoxia across a wide range of arterial PCO_2 has been demonstrated in at least one study.[13] Therefore, the decrease in cerebral blood flow observed in the present study when subjects switched to breathing 100% O_2 is likely to have been caused by cerebral vasoconstriction due to hyperoxia and the associated hypocapnia.

Several studies using TCD to measure $MCAv_{mean}$ have demonstrated a drop in values in association with pre-syncope and syncope. Passive head-up tilt in healthy subjects reduces $MCAv_{mean}$ and cerebral O_2 saturation and pre-syncopal symptoms appear when there is a reduction of about 50% in $MCAv_{mean}$.[14–16] Similar percentage drops in $MCAv_{mean}$ associated with symptoms of faintness have been observed in the present study.

Signs of imminent syncope have been associated with reductions in $MCAv_{mean}$ of 62% and 68% induced by sudden cold water immersion.[17] $MCAv_{mean}$ has also been measured in one study after acute hypercapnia reversal.[18] Subjects rebreathed from a bag containing 5% carbon dioxide in O_2 up to an $ETCO_2$ of 10% or to the limit of tolerance. When rebreathing ceased, there was a rapid decline in $MCAv_{mean}$ within 42 s, followed by a further rapid decline to below baseline, $MCAv_{mean}$ falling by 31% in total.[18]

Another study found reductions in $MCAv_{mean}$ of 44% and 69% respectively and concluded this decrease to be more important as a predictive factor of syncope than the MAP.[19] This is in agreement with the present study where a mean percentage decrease in $MCAV_{mean}$ of 51% was associated with pre-syncopal symptoms (sensation of mild or moderate faintness) while decrease in MAP was not associated with the group of subjects who experienced faintness developing following the switch.

LIMITATIONS OF THE STUDY

Use of a demand valve (DV) regulator for the mouthpiece

Subjects who were inexperienced in the use of a DV made comment on the difficulty of breathing. It is known that breathing systems have an effect on the depth, flow and pattern of breathing.[20,21] The use of a DV regulator could be avoided in future trials by supplying the subjects' breathing gases from pre-filled Douglas bags.

Duration of the test and effects of raised pressure

It should be noted that survivors waiting in the DISSUB may be exposed to raised ambient pressure and wait for up to seven days before rescue or escape. Investigation of prolonged (chronic) exposure to hypercapnic gas at raised pressure and the effects that acid-base balance, buffering and compensation may have on the response to a switch from hypercapnia to hypocapnia and/or hyperoxia was outside the scope of the current study. Effects of a switch to air or 100% O_2 following prolonged exposure to raised PCO_2 and/or hyperbaric exposure remain as possible topics for future investigation.

Possible additional effect of Valsalva

The Valsalva manoeuvre is carried out during the compression phase of escape in order to equalise pressure across the tympanic membrane, preventing otic barotrauma. During Valsalva, the $MCAv_{mean}$ can drop by about 35% when supine, and by around 50% when standing.[22] Thus, Valsalva may partially compromise cerebral perfusion and this may be compounded by any CO_2-off effect during escape. This issue is currently under investigation.

Conclusions

On undergoing a switch from breathing $5CO_2/16O_2$ to breathing 100% O_2, a significant difference was observed in percentage drop in $MCAv_{mean}$ between subjects who had symptoms of faintness that developed after this switch and those who did not, suggesting that feeling faint is linked to the drop in cerebral perfusion. The risk of incapacitation owing to fainting, sudden collapse or vomiting on switching to 100% O_2 following acute exposures to hypercapnia at a P_iCO_2 of up to 5.0 kPa is less than 8%. The relative mildness of symptoms observed does not indicate that a change to current procedures is necessary. However, the limitations of the current study suggest that the possibility of worse symptoms in some DISSUB scenarios cannot be ruled out. Evidence from other studies suggests that the severity of symptoms will increase if P_iCO_2 rises above 5.0 kPa.[1,2]

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