Original articles and case reports

Lung function following exposure to Royal Navy treatment table 62

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Key words
Pulmonary oxygen toxicity, hyperbaric oxygenation, vital capacity, unit pulmonary toxic dose

Abstract
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Objective: To quantify any change in vital capacity (VC) measured as forced vital capacity (FVC) in humans, as an index of pulmonary oxygen toxicity (POT) following the Royal Navy treatment table 62 (RN 62) protocol for the treatment of decompression illness (DCI). We assessed whether the observed decrement in FVC was closer to the VC decrement as predicted by the unit pulmonary toxic dose (UPTD) (-2%), or the VC decrement previously described following a similar oxygen exposure (-12%).
Methods: We conducted a prospective, observational study with 20 subjects. Six underwent hyperbaric oxygen therapy (HBOT) for the treatment of DCI, while 14 were healthy volunteers. All subjects underwent the RN 62 protocol and expiratory spirometry was performed immediately before and after this exposure. Participants completed a simple questionnaire after exposure to evaluate symptoms related to POT. The primary outcome was percentage change in FVC following the hyperbaric exposure. Secondary outcomes included changes in other lung function parameters such as peak expiratory flow (PEF) and forced expired volume in one second (FEV_1), and estimation of symptom severity related to POT.
Results: Mean change in FVC after HBOT for all subjects was a decrease of 8.3% (SD 9.4%, P = 0.001). DCI patients showed a significantly greater reduction in VC post exposure than the healthy volunteers (P = 0.04). There was no association between POT-related symptom severity and change in FVC.
Conclusions: Observed decrement in FVC exceeded the VC decrement predicted by the UPTD for the RN 62 HBOT protocol and was closer to the decrease in FVC observed after a similar oxygen exposure in a previous study. Symptoms related to POT do not correlate with decrement in FVC.

Introduction

Pulmonary oxygen toxicity (POT) is a recognised risk of hyperbaric oxygen therapy (HBOT).[1] Early changes resulting from oxygen toxicity include alveolar and interstitial oedema, alveolar haemorrhages and proteinaceous exudates. Further exposure amplifies the inflammatory reaction and leads to a proliferative phase during which type II epithelial cells and fibroblasts proliferate and collagen is deposited.[1,2]

In the development of POT, lung mechanical function is impaired earlier and to a greater extent than gas exchange.[3] The degree of POT can be effectively measured by decrement in vital capacity (VC), where VC is the maximum volume of air that can be exhaled or inspired during a forced (FVC) or slow (SVC) manoeuvre.[1,3-7] Various mechanisms by which POT reduces VC have been proposed, including bronchoconstriction secondary to increased vagal tone and atelectasis,[2-4] but VC changes are probably caused by multiple interacting factors.[3] After a single experimental hyperoxic exposure, any difference in lung function has been found to be reversible within 24 to 72 hours.[2,3,8]

POT is best monitored by serial pulmonary lung function tests but in practice this is difficult and costly.[9] The concept of the 'unit pulmonary toxic dose' (UPTD) was therefore developed in an attempt to quantify the predicted degree of POT as measured by decrement in VC from a single hyperoxic exposure[10,11] (see methodology "Derivation of the UPTD"). The greater the UPTD, the greater the average predicted decrement in VC and severity of POT. The relationship between the predicted decrement in VC and UPTD is summarised in Table 1. Examples of HBOT tables and their designated UPTD and VC predicted decrements are given for clinical relevance.

Despite UPTD calculation being based on observed VC decrements, subsequent studies have shown that the observed changes in VC do not always equal those predicted by the UPTD. For example, when Clark and Lambertsen exposed subjects to continuous 100% oxygen at 2.0 and 2.5 atmospheres absolute (ATA), the observed decrease in VC tended to exceed the decrement predicted by UPTD.[3]

The calculated UPTD for the Royal Navy table 62 (RN 62, Figure 1) is 637 and predicts a 2% decrease in VC (Table

1).[12] To date, observed changes in VC following a series of RN 62 schedules have not been reported. When Clark and Lambertsen exposed subjects to 2.5 ATA (253 kPa) for 3.1 and 4.9 hours they observed a change in VC of -4.9% and -12.2% respectively.[3] Given the P_IO_2/t profile of RN 62, one might therefore expect the subsequent change in VC to lie somewhere between these figures rather than at -2% as predicted by the UPTD.

This study was designed to test the hypothesis that the measured decrease in FVC following an RN 62 treatment schedule significantly exceeds that predicted by the UPTD. In addition we examined any relationship between symptoms of POT and the measured decrement in FVC.

Materials and methods

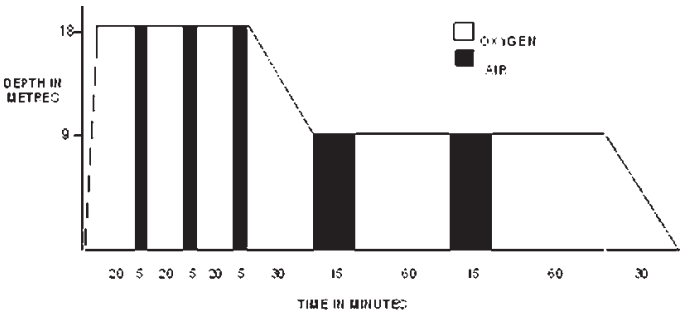
The study was approved by the South Eastern Sydney Area Health Service Research and Ethics Committee prior to volunteer enrolment. Subjects were eligible for entry if over 18 years of age and both qualified to dive with at least an 'open water' certification and not suffering from any temporary or permanent contraindication to compression. We accepted both healthy volunteers and patients who were diagnosed with DCI and for whom an RN 62 treatment table had been independently prescribed by their diving physician. Potential subjects were excluded if they were unable to effectively perform lung function testing or had symptoms suggestive of significant pulmonary disease.

Expiratory spirometry was performed immediately before and after hyperbaric treatment (PneumoCheck™, Welch Allyn, Skaneateles Falls, NY) and following instruction by experienced staff. One of the investigators (JL) performed all spirometry and recorded FVC, FEV_1 (forced expired volume in litres in one second following a maximal inspiration), FEV_1/FVC (FEV_1 expressed as a percentage of FVC), FEF_{25-75} (the average forced expired flow in litres over the middle half of expiration), and PEF (peak expiratory flow, the maximal expiratory flow rate $l.sec^{-1}$ achieved on a forced expiratory manoeuvre). The best effort from three attempts was recorded for each examination.

The predicted 'normal' FVC for each subject, based on subject's height, age and sex, was calculated by the spirometer and recorded. The spirometer was calibrated before every test in accordance with the manufacturer's guidelines.

All subjects were exposed to the RN 62 HBOT schedule in a multi-place chamber. This schedule mandates oxygen breathing at 2.8 ATA (283 kPa) with air breaks, a controlled decompression over 30 minutes to 1.9 ATA (193 kPa) on oxygen, further oxygen breathing periods with air breaks at that pressure and a final 30-minute decompression to the surface to a total time of 4 hours 45 minutes (Figure 1). The breathing system consisted of an airtight mask and demand valve regulator as shown in Figure 2.

Figure 1
Graphical representation of Royal Navy Table 62 HBOT schedule



Total elapsed time 4 hours 45 minutes
(can be extended up to 6 hours 50 minutes)

Following hyperbaric exposure and prior to lung function testing, subjects were asked to record pulmonary-related symptoms using a questionnaire designed for this study. Subjects were asked to rate four symptoms (pain behind the breast bone, tight feeling in chest, shortness of breath, dry cough), each on a scale of 0 to 3 (absent = 0, mild = 1, moderate = 2, severe = 3). If any symptom was exacerbated on inspiration, the subject scored an extra point. The four symptom scores were summed to give a total score for each subject. For example, a subject experiencing mild substernal pain, moderate chest tightness, severe shortness of breath and severe dry cough on inspiration would have a total symptom score of 1 + 2 + 3 + 3 (+1) = 10. This attempt at quantifying symptoms was similar to the method utilised by Clark and Lambertsen.[3]

Table 1
Unit pulmonary toxicity dose (UPTD) values and average expected decrement in Vital Capacity (VC) (adapted from Wright [11])

UPTD	Approximate % decrement in VC	Example HBOT table
270	<2	P_IO_2 1.4 ATA, t = 1.5 hrs
615	2	
637	2	P_IO_2 2.8 to 1.9 ATA, t = 4.45 hrs (RN 62)
860	4	P_IO_2 2.8 to 1.9 ATA, t = 5.45 hrs (extended RN 62)
1035	6	
1230	8	
1425	10	
1815	15	
2061	17	University of Pennsylvania Institute of Environmental Medicine table 7A (50/50 nitrox/air table at 3.0 to 2.2 ATA)

Figure 2
Oxygen mask demand breathing apparatus

Derivation of the UPTD

The UPTD is calculated as the equivalent oxygen exposure at one ATA (101 kPa) in minutes.[5] The UPTD must be described with respect to both partial pressure of oxygen (P_IO_2) and duration of oxygen exposure (*t*) since POT increases with both parameters. Estimation of the UPTD is usually made with reference to a family of hyperbolic curves.[2,4–6,13] These curves describe the rate of development of POT with increasing P_IO_2 in an individual with 'average' susceptibility and are constructed from VC measurements during oxygen breathing at 2.0 ATA (203 kPa), 1.0 ATA (101 kPa) and 0.83 ATA (84 kPa).[2,4,5,14,15] From these curves a mathematical equation was derived which may be used to convert any P_IO_2 – *t* combination to an equivalent 1.0 ATA exposure; that is, the UPTD for that exposure.

POWER AND STATISTICAL ANALYSIS

Sample size calculation suggested we recruit 18 subjects in order to have an 80% chance of detecting a predicted reduction in FVC of 12% following hyperbaric exposure, at a significance level of 0.05. We used Student's paired t-test (two-tailed) to quantify the significance of any differences in lung function for each subject, and the independent t-test for estimations made between subgroups. Any correlation between symptoms of POT and FVC decrements were investigated using Kendall's rank correlation (non-parametric equivalent of simple linear regression). All statistical analyses were made using StatsDirect statistical software, version 1.611, Iain Buchan, 2000.

Table 2
Demographic data for the study subjects

Male/female ratio	16/3
DCI patients/healthy volunteers	5/14
Smoker/non-smoker	3/16
Average age (years)	35.8 +/- 7.5
Average height (cm)	178.9 +/- 7.6

Results

Twenty subjects were recruited from November 2002 until February 2003. One subject was excluded from all analysis because spirometry data after RN 62 exposure had become corrupted. The demographic data for the remaining 19 subjects are summarised in Table 2.

There was a statistically significant reduction in mean FVC following the RN 62 protocol of 8.3% (SD 9.4%, P = 0.001). Sixteen out of 19 subjects demonstrated a decrease in FVC while in three subjects FVC increased. There was no statistical difference between FVC measurements in DCI patients and healthy volunteers before exposure to the RN 62 protocol (P = 0.804). However, DCI patients sustained a decrease in FVC of 15.5% (SD 8.2, P = 0.01, 95%CI = 5.4 to 25.7), while healthy volunteers sustained a smaller reduction in FVC of 5.7% (SD 8.6, P = 0.02, 95%CI = 0.7 to 10.7). This difference between subject types was statistically significant (P = 0.04, 95%CI = 0.5 to 19.2). There was no significant difference in FVC reductions between those who smoked and those who did not. The mean FVC decrease in smokers was 6.2% (SD 4.6%, P = 0.14), and in non-smokers 8.8% (SD 10.1, P = 0.003), the difference not being significant (P = 0.69, 95%CI = -5.2 to 17.6). FVC data for all subjects, DCI patients and healthy volunteers, are summarised in Table 3.

FEV_1 for all subjects decreased by a mean of 2.4% (SD 37.4 %). This change was not statistically significant (P = 0.78, 95%CI). Because the decrease in FEV_1 was less than the decrease in FVC, the FEV_1/FVC ratio improved following HBOT. The measured changes in FEF_{25-75} and PEF were highly variable (variance 30%), and there were no significant changes before and after HBOT (Table 4).

Eleven subjects complained of symptoms that might be related to POT. Ten complained of a mild dry cough only, one of a moderate dry cough and one of several symptoms including cough, chest tightness and retrosternal chest pain on inspiration. There was no correlation between symptom severity and change in FVC (Kendall's rank test, P = 0.697).

Discussion

VC, measured as FVC, showed a greater reduction following an RN 62 protocol than that predicted by the pre-existing UPTD calculation. The FVC decrement roughly correlated

Table 3
Forced vital capacity (FVC; mean +/- SD) before and after RN 62 HBOT exposure

Group (n)	Pre RN 62 FVC (l)	% predicted	Post RN 62 FVC (l)	% predicted	% change	P 95% CI
Total (19)	5.19	106.2	4.74	96.1	-8.3	0.001
(SD)	(+/- 0.93)	(+/- 23.1)	(+/- 0.79)	(+/- 15.8)	(+/- 9.4)	-3.8 to 12.8
DCI patients (5)	5.10	105.1	4.3	88.0	-15.5	0.01
(SD)	(+/- 0.82)	(+/- 25.6)	(+/- 0.69)	(+/- 18.6)	(+/- 8.2)	-5.4 to -25.7
Volunteers (14)	5.23	106.5	4.89	99.0	-5.7	0.02
(SD)	(+/- 0.99)	(+/- 23.2)	(+/- 0.79)	(+/- 14.3)	(+/- 8.6)	-0.7 to -10.7

with the observed change shown previously by Clark and Lambertsen.[3]

We enrolled patients suffering with DCI on an opportunistic basis during the healthy volunteer recruitment period and did not attempt to enrol equal numbers of each subject type for comparison. A somewhat unexpected finding was that the decrements observed were significantly greater in individuals suffering with DCI than in healthy volunteers. This finding might be due to increased exposure to oxygen prior to the hyperbaric exposure in the study, both from 100% oxygen administration on the surface and increased P_IO_2 during the in-water dive that precipitated the episode of DCI. It may also have been due to pulmonary bubble injury in the DCI patients. There was, however, no statistical difference between FVC measurements before exposure to the RN 62 protocol, and a number of the healthy volunteers had also dived in the days prior to the study. One further possibility is that the DCI patients underperformed during their spirometric evaluation after treatment. The healthy volunteers were enthusiastic and treated in a motivating group environment, while DCI patients were mostly treated late in the evening, alone apart from the attendant, and were often asked to perform effort spirometry after a long and stressful day.

We chose to use changes in FVC as our primary measure of POT. A reduction in VC has previously been shown to be one of the most sensitive and consistent manifestations of POT, and is progressive throughout oxygen exposure.[2–6,9] It is relatively simple to estimate FVC with acceptable accuracy and reproducibility using easily available hand-held devices such as that employed in this study. An alternative measure of POT is the volume of air able to be inspired from rest (inspiratory capacity or IC, the inspiratory component of VC). Some authors have suggested IC might be more sensitive than VC as an index of POT, since the relative change in IC was greater after HBOT exposure.[2,4]

FEV_1 was not significantly reduced in our study, a similar result to that reported by Clark and Lambertsen, but reductions have previously been observed after HBOT without a fall in FVC.[3,4,16-19] FEV_1 is a measure of small airway conductance. Since the reduction in FEV_1 in our study was relatively less than the reduction in FVC, the FEV_1/FVC ratio appeared to increase. FEF_{25-75} is a more sensitive measure of small airway function than FEV_1 and it has been shown to decrease with increasing oxygen exposure.[3,16,17] However, its normal range is wide, it is less reproducible than FEV_1 and it can also be difficult to interpret.[7] The wide variation in results for FEF_{25-75} in our study confirms this. PEF results were also extremely varied with changes between +229% and -30%. Other studies have shown that PEF decreases or remains unchanged.[8,20]

Our symptom questionnaire was based on previous observations of POT-related symptoms following hyperbaric oxygen exposure.[2–4,12,13] These are likened to the symptoms

Table 4. FEV_1, FEV_1/FVC, FEF_{25-75} and PEF changes for all subjects before and after RN 62 HBOT exposure (FEV_1 – forced vital capacity in 1 sec; FVD – forced vital capacity; PEF – peak expiratory flow; FEF – forced expiratory flow)

Lung function	Pre RN 62	Post RN 62	Absolute change	% change
FEV_1 (l)	3.8 (+/- 0.8)	3.7 (+/- 0.8)	-0.3 (+/- 1.5)	-2.4 (+/- 0.4)
FEV_1/FVC (%)	74.5 (+/- 12.0)	76.2 (+/- 8.1)	+1.6 (+/- 7.6)	+3.9 (+/-13.1)
FEF_{25-75} (l)	3.3 (+/- 1.5)	3.1 (+/- 1.3)	+0.1 (+/- 0.9)	+13.0 (+/- 55.5)
PEF ($l.sec^{-1}$)	9.0 (+/- 2.6)	8.7 (+/-1.7)	+0.3 (+/-2.3)	-7.1 (+/-55.6)

of a tracheobronchitis, originating in the carina and spreading to the bronchial tree.[2,4] Symptoms usually begin as a mild throat irritation accentuated by inspiration or a dry cough and progressing to a tight chest, uncontrollable coughing, painful inspiration and dyspnoea. Ultimately POT culminates in severe asphyxia and death in the experimental animal.[2] Symptoms rapidly diminish within the first few hours following exposure and have usually completely reversed over the next 24 to 72 hours.[2,3,4] We did not document recovery in our study. The lack of any apparent association of respiratory function decrement and symptoms related to oxygen toxicity in individuals confirms previous reports,[3,13] although a general trend towards increasing symptom severity with decreasing VC has been shown.[2,3,4]

By predicting the decrement in VC following a hyperoxic exposure, the UPTD allows the comparison of pulmonary effects of different HBOT treatment schedules and has been widely employed to provide safe guidelines for pulmonary hyperoxic exposure.[5] Our study suggests that the UPTD for the RN 62 schedule significantly underestimates POT as indicated by the immediate effect on VC. The mathematical derivation of the UPTD is one possible source of this underestimation. The UPTD is based on predictive curves from observations of normal, healthy males exposed to a P_IO_2 of 2.0 ATA (203 kPa) and below.[2,5] This, and Clark and Lambertsen's study,[3] used exposures above 2.0 ATA, so using the UPTD in this context is extrapolating outside the test data. However, these rectangular hyperbolae and hence the UPTD have been used in practice to predict the estimated reduction in VC following exposure to a P_IO_2 greater than 2.0 ATA (203 kPa). The curves are drawn with the implicit assumption that there are asymptotes at $t = 0$ and $P_IO_2 = 0.5$ ATA.[2,5]

Conclusions

VC, measured as FVC, decreased after exposure to the RN 62 protocol. Patients with DCI showed larger decrements in FVC than healthy volunteers. The decrease in both groups exceeded that predicted by the UPTD for that oxygen exposure, suggesting that the concept of the UPTD as a predictor of POT is not without limitations. FEV_1, FEV_1/FVC, FEF_{25-75}, and PEF did not change significantly and there was no correlation between symptoms related to POT and observed change in FVC.

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Declaration

The authors declare they have no financial interest in any commercial product involved in this research and received no financial assistance for the conduct of this study.

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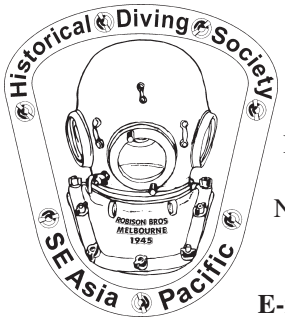

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