medical. He was on anti-hypertensive treatment. He also had multiple medical problems with irritable bowel and back problems. In 1997 he had a motor vehicle accident where he had blunt chest trauma to his left upper chest, which is where the adhesions were found, but the chest X-ray done straight after the accident was negative.

### Similarities between the two cases

There are striking similarities between the two cases, both were novice divers but reported as being very experienced. Both were obese. Both had multiple medical problems. Both were sudden deaths on the surface of the sea from gas embolism. Both were initially thought to be a myocardial event but there was no cerebral vascular event or coronary artery disease found in either one. Both had bitten their tongues which to me indicates that both had had a convulsion. Both had pleural adhesions. They were both diving in a team, they probably had controlled ascents. Their dive medicals were probably inadequate and none.

Both were alone on the surface but I do not think that a buddy would have been any use at any stage during the rescue. Except perhaps they would have been able to tell us what really happened.

They were both recorded by the pathologist as being drowning deaths and on the second one the pathologist, even after we found air in the heart, said "I'll still put this down as a drowning death for insurance purposes". In this man I think his convulsion occurred when he went to inflate his buoyancy jacket. He was floating when he started to convulse and that is why his jacket over-inflated.

### Conclusions

These two divers died from drowning, but the cause of the drowning was air embolism, causing unconsciousness, after barotrauma in lungs tethered by pleural adhesions.

It is very important that an experienced diving doctor, with good reasoning abilities attends, every post-mortem after a diving death to integrate such oddities as bitten tongues into the pathologist's diagnosis.

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## EFFECT OF HYPERBARIC OXYGEN THERAPY ON LUNG VOLUMES IN NORMAL SUBJECTS AND HEAVY SMOKERS.

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**Key Words**
Hyperbaric oxygen, physiology, research, lung volume, airway obstruction

### Abstract

Decompression from hyperbaric conditions may be associated with air-trapping if the alveoli are unable to empty rapidly enough as ambient pressure decreases. In particular, such air-trapping may occur with the airway obstruction seen in emphysema and asthma. To determine whether this is clinically detectable, subjects with a heavy smoking history and airway obstruction were studied in a hyperbaric chamber.

Fifteen subjects underwent lung volume estimation by body plethysmography at sea level and again immediately after hyperbaric oxygen therapy at 240 kPa for 90 mins. Results were compared with those of seven non-smokers undergoing the same treatment, and five non-smoking staff breathing air.

There were no significant changes in lung volumes in any of the study groups in terms of their lung volumes. The smokers had more airway obstruction than the other groups, but this was mild despite a heavy smoking history. Mild airway obstruction does not appear to be associated with significant gas-trapping after hyperbaric therapy.

### Introduction

Patients receive hyperbaric oxygen therapy ($HBO_2T$) for a variety of medical indications, including osteoradionecrosis, necrotising infections and chronic ulcers secondary to diabetes and vascular insufficiency. An increasing number are elderly and have vascular insufficiency, or have a history of head and neck squamous cell carcinoma secondary to smoking. These smokers or ex-smokers may benefit from $HBO_2T$, but there are theoretical reasons why acute changes in lung volumes might occur in some subjects with airway dysfunction. Such dysfunction has not been documented, nor have adverse events been reported. It remains possible, however, that airway obstruction in smokers or asthmatic subjects might cause trapping of gas within the lungs as alveolar gas expands on decompression.

It was hypothesised that those with airway obstruction secondary to smoking would have a tendency

to trap air within their lungs during decompression, leaving them with an increased residual volume immediately after the treatment. The data from the limited number of studies that are available suggest that some changes take place during $HBO_2T$, although these studies were designed to look at chronic effects of these conditions. Thorsen et al. showed a small reduction in expiratory flow and diffusion with $HBO_2T$ (240 kPa 90 min daily for 21 days) in 20 subjects.[1] Clark et al., studying normal volunteers in a range of pressures up to 300 kPa, showed a slight fall in spirometry and diffusion capacity in 8 of 15 subjects over 5-18 hours.[2] These changes are thought to represent the effects of oxygen toxicity upon the lung.[3] Fractionating oxygen exposure appears to reduce the risk of toxicity.[2,4] On the other hand, Pott et al. were unable to demonstrate significant changes in spirometry, lung volumes, or carbon monoxide diffusing capacity in 18 subjects treated by $HBO_2T$ (240 kPa 90 min daily for 42 days), suggesting that the effects are not large.[5]

One aspect that has not been addressed specifically by the above studies is whether small airway obstruction could cause acute gas-trapping during decompression. If gas-trapping due to small airway obstruction occurs during decompression, there could conceivably be hyper-inflation of the obstructed lung units and an increased risk of pulmonary overpressure injury. This is one of the reasons why subjects with active asthma are advised not to dive. There are, however, no data to suggest that those who have asthma and who dive have a higher incidence of pulmonary barotrauma from this mechanism. It was hypothesised that $HBO_2T$ in those with a heavy smoking history and evidence of pre-existing gas-trapping with small airway obstruction would show an increase in residual volume after $HBO_2T$. Gas trapped during $HBO_2T$ would expand on decompression due to Boyle's Law but be unable to escape from the lungs because of small airway obstruction.

## Methods

The study was approved by the hospital ethics committee and informed consent was obtained. Patients currently smoking and receiving $HBO_2T$ underwent plethysmographic evaluation of lung volumes before and after a single $HBO_2T$ session. These results were compared with a control group of both non-smoking patients undergoing $HBO_2T$ and staff members who were compressed in the chamber breathing air. Participants included 15 smokers (>40 pack year exposure), 7 control subjects (non-smokers or 'light' ex-smokers, <10 pack years), and 5 non-smoking staff members who attended patients during treatment.

## $HBO_2$ treatment

All subjects and staff had been passed as 'fit to dive' according to current standards.[6] The chamber was pressurised to 240 kPa (equivalent to the pressure at 14 m of sea water) for 90 minutes, followed by a 20 minute decompression to 101 kPa (a routine profile frequently utilised at this facility).[7] Patients breathed 100% oxygen via a face mask throughout the procedure, while staff were breathing air during the 240 Pa period and 100% oxygen during decompression only.

## Body plethysmography and spirometry

Pulmonary function tests were measured using body plethysmography (Fennyves & Gutz, Basle, Switzerland) according to published methods.[8] The following values were derived: forced expiratory volume in the first second ($FEV_1$), maximal mid-expiratory flow (MMEF), forced vital

**TABLE 1**

**SUBJECT CHARACTERISTICS, REASONS FOR HYPERBARIC OXYGEN TREATMENT, AND BASELINE LUNG FUNCTION (SE).**

| | **Age** (range) | **$HBO_2T$ indication** | **FEV (L)** *% predicted* | **%FVC** | **FVC (L)** *% predicted* | **TLC (L)** *%predicted* |
|---|---|---|---|---|---|---|
| 15 Heavy smokers | 61.0 (33-81) | Soft tissue osteoradionecrosis: 8<br>Retinal vein thrombosis: 3<br>One each of: radiation myelitis, xerostomia, non-healing wound, and Crohn's abscess. | 2.82 (0.23)<br>*100.8 (8.9)* | 79.28 (1.79) | 3.55 (0.15)<br>*90.8 (8.0)* | 6.28 (0.25)<br>*97.6 (7.9)* |
| 7 Light or non-smokers<br>5 Staff (non-smokers) | 40.5 (25-71) | Soft tissue osteoradionecrosis: 4<br>One each of: retinal vein thrombosis, optic neuropathy, and diabetic ulcer. | 3.53 (0.01)<br>*108.2 (14.0)* | 84.34 (0.98) | 4.19 (0.17)<br>*99.51 (10.2)* | 6.12 (0.32)<br>*95.8 (8.8)* |

**TABLE 2**

**COMPARISON OF LUNG FUNCTION BEFORE AND AFTER HYPERBARIC OXYGEN THERAPY (90 MINS 100% OXYGEN AT 240 KPA), IN THOSE WHO HAVE A >40 PACK YEAR SMOKING HISTORY WITH THOSE WHO HAVE <10 PACK YEAR SMOKING HISTORY. COMPARISON OF % CHANGE IN HEAVY SMOKERS VERSUS CONTROLS USING UNPAIRED T-TESTS.**

| Lung volumes (% change) | Smokers (n=15) | Non-smokers (n=12) | p value |
|---|---|---|---|
| Total lung capacity % [se] | **-1.37** [0.72] | **-1.63** [0.87] | 0.42 |
| Pre v post (litres) | 6.28 (0.25) v 6.20 (0.25] | 6.12 (0.32) v 6.02 (0.24) | |
| Vital Capacity % [se] | **1.00** [1.94] | **0.95** [0.61] | 0.64 |
| Pre v post (litres) | 3.55 (0.15) v 3.57 (0.17) | 4.19 (0.17) v 4.21 (0.17) | |
| FRC % [se] | **0.00** [0.64] | **1.93** [2.25] | 0.28 |
| Pre v post (litres) | 3.77 (0.24) v.3.77 (0.23) | 3.22 (0.24) v 3.28 (0.22) | |
| $FEV_1$ % [se] | **0.48** [1.16] | **-1.61** [1.2] | 0.04 |
| Pre v post (litres) | 2.82 (0.23) v 2.83 (0.22) | 3.53 (0.01) v 3.47 (0.1) | |

capacity (FVC), total lung capacity (TLC) and hence residual volume (RV).

### Analysis

Results are expressed as mean percent change from baseline. Smokers >40 pack years were compared with those with non- and ex-smokers<10 pack years (including the staff members). Differences were analysed by unpaired t-tests between groups.

### Results

Subject characteristics are summarised in Table 1. There were no differences in terms of predicted lung function, between non-smoking staff members, who breathed air, and the patients classified as non- or 'light' smokers, therefore all analyses are reported with staff members in the non- or 'light' smokers group.

The smokers tended to be older than the control group, and had a male preponderance (M:F 10:5, versus control group 6:6). Although the differences between the groups were small, these were significant in terms of $FEV_1$, FVC and RV (Table 1), when expressed as percent predicted, thus controlling for differences in age and sex between groups.

Thus, the smoking group had a lower percent predicted FEV1, FVC and increased RV compared to the control group, consistent with a mild smoking-induced airway obstruction and gas-trapping. MMEF was not significantly different between the two groups.

### Lung function before and after $HBO_2$

Lung volumes measured as total lung capacity (TLC), functional residual capacity (FRC) and vital capacity (VC) did not change significantly, either between measurements or between the two groups (Table 2). There was a slight decrement in $FEV_1$ in normal subjects.

### Discussion

There were no significant differences between the groups of subjects, except for very small changes in $FEV_1$. Smokers showed a small improvement in $FEV_1$, while the other subjects showed a small decrease. These changes were not sufficient to be clinically meaningful.

There was no evidence that additional gas retention occurred in heavy smokers, and no difference was observed between heavy smokers and either control subjects having $HBO_2T$, or control subjects who were breathing air under conditions of increased pressure. This suggests that gas-trapping either does not occur, or is very transient, under these conditions in these subjects. One criticism of this study might be that only 15 heavy smokers were studied, however the changes in TLC showed a decrease in lung volumes rather than the postulated increase. As this is in the reverse direction of the hypothesised change, a power calculation would not be able to indicate a sample size that would show such an effect.

It is conceivable that trapped oxygen diffused rapidly out of bullae and poorly ventilated air spaces, and had dissipated before the lung tests were performed, or that our smokers did not have a sufficiently severe degree of small airway obstruction. Lung volumes were estimated and completed within 15 mins of exit from the chamber, suggesting that gas trapping would have to be very transient if it did occur. Extrapolating from the rate of decrease of air in pneumothoraces, which is estimated to be 1-2 % per day,[9] it would seem unlikely that this study would have missed an important degree of gas trapping, even though the rate of diffusion of oxygen from the lung to the blood stream is estimated to be approximately twice that of nitrogen.[10] No studies have been performed that would allow us to estimate the rate of oxygen uptake from emphysematous bullae.

These data do, however, suggest that some residual gas should have been present in the lungs of these subjects if gas-trapping was significant. While it is true that the smokers were not severely obstructed in terms of their spirometry, it would be difficult to study those most severely affected as they would not usually be considered for $HBO_2T$, unless the indication for this treatment was particularly pressing.

From these results, we conclude that heavy smoking in association with mild obstruction does not increase the risk of gas trapping while breathing hyperbaric oxygen, and there does not appear to be any appreciable risks of $HBO_2T$ in this regard. It is possible that mild asthmatic subjects with a similar degree of airway obstruction might not show any gas-trapping under hyperbaric conditions either, but such subjects were not involved in this study.

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