

Acute oxygen toxicity in a saturation diver working in the North Sea

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Crosbie WA, Cumming G, Thomas IR. Acute oxygen toxicity in a saturation diver working in the North Sea. *Undersea Biomed Res* 1982; 9(4):315-319.—A commercial diver taking part in a saturation dive (O_2 -He) was exposed intermittently to 1.4 b of oxygen for a total of 55 h. He developed the syndrome associated with oxygen pulmonary toxicity. Detailed pulmonary function spirometry tests before and after the incident showed that a significant decrease in the forced vital capacity and forced expired volume in 1 s occurred and full recovery was at this time not present 12 weeks after the incident, although by this time he demonstrated a high state of cardiopulmonary fitness. These findings indicate that the lung damage caused by acute oxygen toxicity take more than 12 weeks to disappear, but the condition does seem to be reversible.

oxygen lung toxicity
pulmonary function abnormalities

Breathing high concentrations of oxygen for some time damages the lungs of animals and man (1, 2).

An inspired O_2 tension of 0.5–0.6 b is generally accepted as the maximum limit for normal working conditions in the commercial diving field, other than for short periods as part of a decompression schedule. This report describes the circumstances and sequent lung changes that occurred in a commercial diver who was exposed to excessively high concentrations of oxygen when working in a saturation diving system in the North Sea.

HISTORY

Two divers left a saturation deck holding chamber in August 1979 in a diving bell that by mistake was being supplied oxygen at 1.4 b. Within 8–10 h they both noticed a tight retrosternal feeling followed by the development of an irritating cough. They continued working, but by 16 h their symptoms were worse, and they returned to the deck holding chamber. After a few hours they improved, and 8 h after the end of the first exposure they returned to the bell. Within 3–4 h the same symptoms returned, but they managed to complete the second 16-h shift. They reported their condition to the diving supervisor, but again the cough and chest pain subsided. Eight hours after the end of the second exposure they returned to the bell for

the next 16-h shift, only to have the symptoms recur with increasing severity. This time they developed progressive breathlessness. They somehow managed to complete the shift, but on return to the holding chamber they again reported their symptoms to the diving supervisor; by now their cough was productive of mucoid sputum. Again after an 8-h rest they returned to the diving bell, but within an hour their symptoms of cough, chest pain, and breathlessness redeveloped, and they were returned to the holding chamber. They asked for medical advice on their condition, and at this time it was discovered that the oxygen concentration being supplied to the bell was too high: they had been exposed intermittently for a total of 55 h to 1.4 b of oxygen. The oxygen partial pressure in the deck holding chamber during this entire period of time was 0.6 b. Arrangements were then made to decompress the men, and this was completed in 4.5 days. The breathlessness gradually subsided and the chest pain lessened, but the cough and expectoration were still present when they reached the surface.

Because one of the divers returned to the United States, we were unable to follow his progress. The other diver (age 29 at time of incident) is resident in the United Kingdom and his progress could be monitored. He reported that the chest pain disappeared within a week, but the cough and expectoration remained for a second week. Dyspnea on exertion persisted for about a month, but thereafter he could run and swim without undue breathlessness.

Five weeks after the incident spirometry showed that there was a reduction in the forced vital capacity (FVC) and forced expired volume in one second (FEV_1) compared with measurements made on the same spirometer 6 weeks before the incident (Table 1). Measurements repeated 10 weeks after the high oxygen exposure were still below the preincident level; hence he was referred for more detailed investigation. By good fortune detailed pulmonary function tests had been conducted in 1978 at the Midhurst Research Institute, where he had been referred for assessment of a low percentage of FVC expired in one second ($FEV_1/FVC\%$). The results at the time showed no evidence of abnormal lung function. There was no history of respiratory illness in the past. He started smoking when he was 15 years old, 5–10 cigarettes per day, and was smoking up to 40 cigarettes a day when he stopped at 22 years of age. He began scuba diving in 1970 when he joined the Royal Navy and became a ship's diver. He moved to commercial diving in 1978 following a course on mixed-gas diving, and for more than a year he worked in the North Sea without any diving problems until the incident described.

The serial measurements of this man are shown in Table 1. Six weeks before the oxygen toxicity exposure the forced vital capacity was within the normal range, and the forced expired

TABLE 1
SERIAL SPIROMETRIC CHANGES

Measurement	Unit	Predicted Value ^a	Time Before O ₂ Toxicity, 6 wk	Time After O ₂ Toxicity		
				5 wk	10 wk	12 wk
FVC ^b	liters	5.6	5.9	5.1	5.1	5.5
FEV_1 ^c	liters	4.3	4.0	3.4	3.4	3.5
$FEV_1/FVC\%$ ^d		74	68	67	67	64
$FEF_{0.2-1.2}$ ^e	liters/s	8.5	6.0	6.0	6.0	8.1
$FEF_{25\%-75\%}$ ^e	liters/s	4.5	2.1	1.9	2.2	2.0
$FEF_{75\%-85\%}$ ^e	liters/s	0.65	0.5	0.5	0.85	0.65

^aCotes (3). ^bFVC, forced vital capacity. ^c FEV_1 , forced expiratory volume in 1 s. ^d $FEV_1/FVC\%$, percentage of FVC expired in 1 s. ^eFEF, mean forced expiratory flow between two values.

volume in one second and the expiratory flows were reduced but considered normal, since they were similar to the levels for which he was investigated in 1978.

Five weeks after the incident there was a significant reduction in the FVC and FEV₁. By 10 weeks after the incident there had been little change, but by 12 weeks the FVC had increased, although the FEV₁ and expiratory flows showed less improvement. Table 2 provides further complementary information showing that the subdivisions of lung volume were close to measurements obtained 1 year prior to the high O₂ exposure. The total airway resistance was somewhat higher than the level before the insult and was in keeping with the FEV₁ and expiratory flow measurements. The gas transfer measurements of CO (Tco) and the transfer factor (per liter of alveolar volume) were close to those obtained before the episode of oxygen toxicity.

At the same time an exercise test (Table 3) confirmed that the diver showed a very good state of cardiopulmonary fitness. He showed a slow deep breathing pattern and reduced heart rate with normal oxygen uptake. Hence by 12 weeks after the high O₂ exposure it confirmed a good state of fitness, but the lungs had not returned completely to the preexposure level.

DISCUSSION

The history of exposure to high concentration of oxygen and the clinical events indicate that this diver sustained damage to his lungs from oxygen toxicity. While this syndrome is well recognized there is little information on the serial changes in the pulmonary function of such individuals. By chance, measurements of his lung function had been made prior to the incident and were available; they showed him to have normal lung function. The reduced expiratory flows were considered to be a normal variant present in some divers. No measurements were made at the time of the incident or when he was symptomatic, but 5 weeks after the exposure when he was asymptomatic there was evidence of a reduction in the FVC and FEV₁. This finding suggests that damaged lung tissue was still present. This may have been in the form of small areas of collapsed lung that were recovering but had not yet regained their full size.

Since the FEV₁/FVC \times 100 and expiratory flows were also reduced it seems that some airway narrowing or gas trapping may also have been present. Twelve weeks after the incident the FVC, lung volumes, and gas transfer characteristics had now returned to the levels measured before the injury. The FEV₁ and total airway resistance had not shown a similar

TABLE 2
DETAILED CARDIOPULMONARY FUNCTION RESULTS IN RELATION TO TIME OF OXYGEN TOXICITY EPISODE

Measurement	Unit	Predicted Value ^a	1 Year Prior to Toxic O ₂ Exposure	12 Weeks After Toxic O ₂ Exposure
TLC ^b	liters	7.4	7.6	7.4
RV ^c	liters	1.8	1.9	1.8
R _{aw} ^d	cmH ₂ O \cdot liter ⁻¹ \cdot s ⁻¹	1.7	1.55	1.7
Tco ^e	ml \cdot min ⁻¹ \cdot mmHg ⁻¹	35.3	39.6	39.1
DI/VA ^f	m (liter ⁻¹ \cdot min ⁻¹ \cdot torr ⁻¹)	6.4	6.9	6.9

^aPredicted value, Cotes (3). ^bTLC, total lung capacity. ^cRV, residual volume. ^dR_{aw}, airway resistance. ^eTco, transfer factor of CO. ^fDI/VA, transfer factor per liter of alveolar volume.

TABLE 3
RESULTS OF BICYCLE ERGOMETER EXERCISE TEST

Measurement	Unit	Work Load of 100 W		Work Load of 200 W	
		Measured	Predicted*	Measured	Predicted*
Minute ventilation	liters/min	38	42	62	76
Tidal breath	liters	2.5	1.7	4.1	2.5
Heart rate	beats/min	114	130	150	188
Oxygen uptake	liters/min	1.7	1.6	2.7	2.7

*Predicted value, Cotes (3). Maximum load achieved, 240 W.

recovery; hence some airway change may still have been present. Nevertheless an exercise test showed that the diver was not fit and did not have a good cardiopulmonary response to effort. The breathing pattern and slower than predicted heart rates are characteristic of fit healthy divers (4).

These results indicate that it takes at least 12 weeks for the lung damage produced by oxygen toxicity to recover, and the effects on the airways may persist longer. The history also shows that intermittent exposure to high concentrations of oxygen does produce lung damage. The diver who reports chest pain and cough in circumstances where increased exposure to oxygen can occur demands urgent review of this possibility, and the development of breathlessness should not be awaited before taking remedial measures. Removal from the high-exposure environment quickly leads to the disappearance of symptoms, and some functional abnormalities take longer to disappear. In 1945 Comroe et al. (1) showed that the vital capacity decreased after breathing 0.5–1.0 b of oxygen for 24 h. Caldwell et al. (5) reported that the vital capacity decreased by 16%–18% in the first hours of breathing oxygen at 1 b, but Clark and Lambertsen (2) showed that the decrease begins slowly, then becomes increasingly rapid as time progresses. Both groups of investigators have reported that most subjects have recovered the loss of vital capacity between 1 and 3 days after exposure, but individual subjects may take 2–3 weeks to full recovery. While these studies involved a period of continuous exposure to high concentrations of oxygen, in 1939 Soulie (6) showed that intermittent exposure had a similar but more delayed effect. Studies of pathological material taken from animal experiments on the effects of breathing high concentrations of oxygen have showed that the main effect is seen at alveolar level (7). The acute changes in the interstitium of congestions, cellular infiltration, fibrin deposition, and type 2 cell proliferation are all reversible, but later a stage of fibrinous infiltration can occur, with the laying down of permanent fibrous tissue.

Our findings show that the acute changes in lung function are reversible, and we have no evidence of permanent lung damage in this diver. Nevertheless the incident illustrates how easily fit, well-trained divers can be incapacitated and working schedules disrupted if insufficient attention is given to the possibility of oxygen toxicity damage to the lungs. Fortunately, the lungs react rather quickly to high concentration of oxygen, and the cough and chest pain, followed by breathlessness, should provide a more than adequate warning. It seems that removal from the high oxygen environment brings about relief from the symptoms, although the functional effects may require several months to disappear.

Crosbie WA, Cumming G, Thomas IR. Cas aigu de toxicité pulmonaire à l'oxygène chez une plongeur à saturation travaillant en Mer du Nord. *Undersea Biomed Res* 1982; 9(4):315-319.—Un plongeur professionnel qui participait à une plongée à saturation à l'oxygène-hélium s'est trouvé exposé de façon intermittente à 1,4 bar d'oxygène pour une durée totale de 55 heures. Il a développé le syndrome associé à la toxicité pulmonaire de l'oxygène. Les explorations fonctionnelles respiratoires complètes accomplies avant et après l'accident ont montré une diminution significative de la capacité vitale forcée et du volume expiré maximal en une seconde. Au moment où nous avons écrit ce rapport, 12 semaines après l'accident, une rémission complète n'était pas intervenue, bien qu'alors le malade présentasse déjà un bon état cardiopulmonaire. Ces observations indiquent que les lésions pulmonaires provoquées par l'oxygène demandent plus de 12 semaines pour disparaître, mais le trouble semble bien être réversible.

toxicité pulmonaire de l'oxygène
anomalies de la fonction pulmonaire.

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