Undersea Biomedical Research, Vol. 18, No. 2, 1991

Decrease in the single-breath diffusing capacity after saturation dives

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Suzuki S, Ikeda T, Hashimoto A. Decrease in the single-breath diffusing capacity after saturation dives. Undersea Biomed Res 1991; 18(2):103–109—Before and after saturation dives, we measured lung volumes and diffusing capacity (DL_{CO}/V_A) with a single-breath method on 12 divers (6 divers per dive) who participated in 300-msw saturation dives (total dive time of 15 days with P_{O_2} = 0.42 atm abs) and on 6 divers who were engaged in a 320-msw saturation dive (6 days of bottom time with P_{O_2} = 0.42 atm abs and 12 days of total decompression time with P_{O_2} = 0.495 atm abs. In all divers, vital capacity right after surfacing did not significantly decrease compared with predive values. In the 300-msw saturation divers after surfacing, DL_{CO}/V_A adjusted for hemoglobin (Hb) changes did not significantly decrease, but in the 320-msw saturation divers Hb-adjusted DL_{CO}/V_A was significantly ($P < 0.001$) decreased by 0.70 (mean) ± 0.21 (SD) ($ml \cdot min^{-1} \cdot mmHg^{-1} \cdot liter^{-1}$). These observations indicate that the diffusing capacity is a more sensitive index of pulmonary oxygen toxicity than vital capacity that is traditionally used, and that oxygen partial pressure less than 0.5 atm abs can induce a decrease in pulmonary function.

oxygen toxicity
prolonged oxygen breathing
pulmonary function
saturation dive

It is well known that exposure to hyperoxia produces progressive lung injury (1, 2). With regard to the short-term toxic effects of oxygen, many papers (3–6) have reported reduction of vital capacity as an index of pulmonary oxygen toxicity. In those studies, humans exposed to oxygen partial pressure (P_{O_2}) of under 0.5 atmospheres absolute (atm abs) have been considered at no risk for pulmonary oxygen toxicity. To investigate the long-term effect of oxygen toxicity on the lung after saturation dives ($P_{O_2} < 0.5$ atm abs), we measured both lung volumes and diffusing capacity.

METHODS

The subjects for saturation dives were 18 healthy, male, active-duty uniformed divers. Twelve divers (6 divers per dive) did saturation dives to 300 meters of sea water (msw) and 6 divers did a 320-msw saturation dive. Physical characteristics of

the subjects are given in Table 1. These dives were conducted in the deep diving simulator (DDS) at the Japan Maritime Self-Defense Force (JMSDF) Undersea Medical Center (one 300- and one 320-msw saturation dive) and in the deep diving system on board JDS *Chiyoda* (AS 405) (one 300-msw saturation dive).

All divers engaged in both simulated and open-water saturation dives underwent pulmonary function tests as a part of health check procedures so that data taken from all divers were compiled for analysis.

In all cases, divers were decompressed according to the Royal Navy saturation decompression schedule for the 300-msw saturation dives (7) and the modified Royal Navy saturation decompression schedule for the 320-msw saturation dive (8). In the 300-msw saturation dives (Fig. 1), total time of dive was 14 days and 17 h, and P_{O_2} was maintained at 0.42 atm abs throughout the dives. Oxygen partial pressure was made up before each stage of decompression. In the 320-msw saturation dive (Fig. 2), total time of dive was 16 days and 17 h, and P_{O_2} was kept at 0.42 atm abs for 5 days and 5 h of the bottom stay and 0.495 atm abs for 11 days and 12 h of the decompression period. Oxygen was added after decompression of each stage to make up $P_{O_2} = 0.495$, so that P_{O_2} was kept below 0.5 atm abs throughout the decompression phase. The reason for increasing P_{O_2} to 0.495 atm abs during decompression was to reduce the risk of decompression sickness (DCS) (9).

Lung volumes including residual volume (RV), functional residual capacity (FRC), total lung capacity (TLC), and vital capacity (VC) and pulmonary diffusing capacity for carbon monoxide (DL_{CO}/V_A) were measured with an automated system (CHES-

TABLE 1
PHYSICAL CHARACTERISTICS[a]

	Height, cm	Weight, kg	Age, yrs
300 msw, $n = 12$	170.2±3.5	68.8±6.7	32.9±7.3
320 msw, $n = 6$	170.5±5.1	66.8±6.9	27.5±5.0

[a]Values are mean ± SD.

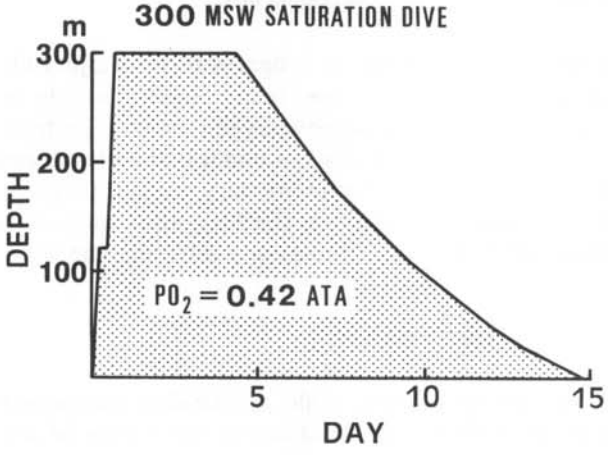

Fig. 1. The profile for 300-msw saturation dives.

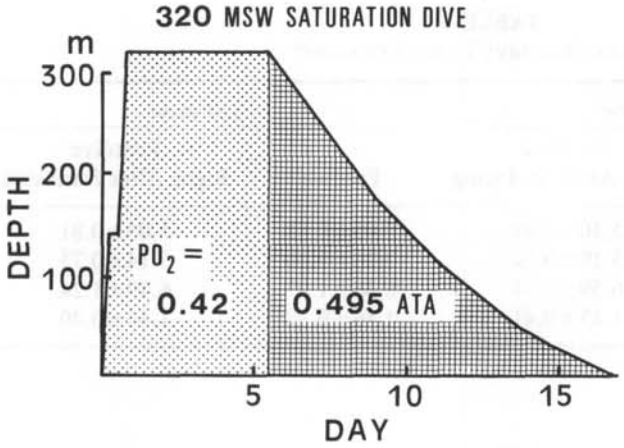

Fig. 2. The profile for 320-msw saturation dive.

TAC-25V model; Chest Ltd., Tokyo, Japan), which allowed the investigator to encourage the subject while monitoring the result. DLCO measurement was carried out by single-breath technique. Alveolar volume (VA) was calculated by adding the inspiratory volume to the RV that was measured just before the measurement of the inspiratory volume (10). All measurements were performed by the same investigator at least 3 times for each subject at each measurement in the sitting position. DLCO measurement was repeated until three values were obtained whose intervariation was within 5%, and the median value was used for analysis. The largest VC from three measurements was taken for comparison. The unit of lung volumes was converted into body temperature, ambient pressure, saturated with water vapor, BTPS.

For the 300-msw saturation dives, measurements were carried out at pre- and postdive (0 day after surfacing) conditions, and for the 320-msw saturation dive were at pre- and postdive (0, 4, 13, and 48 days after surfacing). In the latter case, there was no measurement between 13 and 48 days after surfacing because the divers were not available during that period.

At pre- and postdive conditions, hemoglobin (Hb) concentration was measured from venous blood using an E-3000 (Toa Medical Electronics, Tokyo, Japan). Taking account of changes in Hb after the saturation dives, the measured DLCO was adjusted to standard values of Hb = 14.6 g/dl as described below (10):

$$\text{Hb adjusted DLCO} = \text{measured DLCO} \times \{(10.22 + \text{Hb})/(1.7 \times \text{Hb})\}$$

In this study, M-mode ultrasonic detection of microbubbles in the right heart was performed right after surfacing to investigate the effects of decompression-induced bubbles on lung function. In the 320-msw saturation dive, ultrasonic examination was conducted in the DDS during the decompression period as well. These methods have been described in detail elsewhere by Ikeda et al. (11–13).

Paired Student's *t* tests were employed for statistical analysis.

RESULTS

Vital capacity right after surfacing did not significantly decrease in all divers in comparison with predive values (Table 2). No significant change occurred in other lung volumes (RV, FRC, and TLC) (Table 2).

TABLE 2
PRE- AND POSTDIVE LUNG VOLUMES[a]

	300 msw		320 msw	
	Predive	Postdive Right After Surfacing	Predive	Postdive Right After Surfacing
VC[1]	5.14 ± 0.46	5.10 ± 0.48	5.05 ± 0.88	5.03 ± 0.81
FRC[1]	3.37 ± 0.52	3.19 ± 0.64	3.28 ± 0.60	3.14 ± 0.75
TLC[1]	6.67 ± 0.65	6.59 ± 0.74	6.48 ± 1.07	6.49 ± 1.24
RV[1]	1.57 ± 0.28	1.45 ± 0.41	1.46 ± 0.32	1.44 ± 0.40

[a]Values are mean ± SD.

DLCO/VA was significantly ($P < 0.01$) decreased by 0.25 (mean) ± 0.27 (SD) (ml · min^{-1} · mmHg^{-1} · liter^{-1}) (4.3%) in the 300-msw saturation divers, and the further decrease by 0.89 ± 0.23 ($P < 0.001$) (15%) was observed in the 320-msw saturation divers immediately after surfacing (Table 3).

Hemoglobin was significantly decreased after all saturation dives (Table 3). Hb adjusted DLCO/VA did not significantly decrease in the 300-msw saturation divers, but still showed a significant decrease in the 320-msw saturation divers by 0.70 ± 0.21 ($P < 0.001$) (13%) (Table 3 and Fig. 3). In the 320-msw saturation divers, the decreased DLCO/VA was still observed even 13 days after surfacing; the decrease was insignificant 48 days later (Fig. 4).

In the bubble detection we observed few microbubbles except in 1 diver who participated in 1 of the 300-msw saturation dives (12). There were no remarkable microbubbles during decompression and after the 320-msw saturation dive (8).

TABLE 3
PRE- AND POSTDIVE DIFFUSING CAPACITY[a]

	300 msw		320 msw	
	Predive	Postdive	Predive	Postdive
DLCO, ml · min^{-1} · mmHg^{-1}	28.0 ± 2.70	26.1 ± 2.31[b]	30.3 ± 4.12	25.4 ± 3.56[c]
VA, liter	5.18 ± 0.59	5.07 ± 0.65	5.21 ± 0.85	5.17 ± 0.93
DLCO/VA, ml · min^{-1} · mmHg^{-1} · liter^{-1}	5.46 ± 0.74	5.21 ± 0.68[b]	5.88 ± 0.85	5.00 ± 0.83[c]
Hb, g · dl^{-1}	16.1 ± 1.15	15.4 ± 1.39[b]	17.2 ± 0.93	16.0 ± 0.79[d]
DLCO, adj, ml · min^{-1} · mmHg^{-1}	27.0 ± 2.82	25.6 ± 2.54[d]	28.4 ± 3.81	24.5 ± 3.39[c]
DLCO/VA, adj, ml · min^{-1} · mmHg^{-1} · liter^{-1}	5.26 ± 0.68	5.11 ± 0.66	5.51 ± 0.73	4.82 ± 0.80[c]

[a]Values are mean ± SD; [b]$P < 0.01$; [c]$P < 0.001$; [d]$P < 0.05$ from comparable predive value; adj = values adjusted to standard levels of Hb.

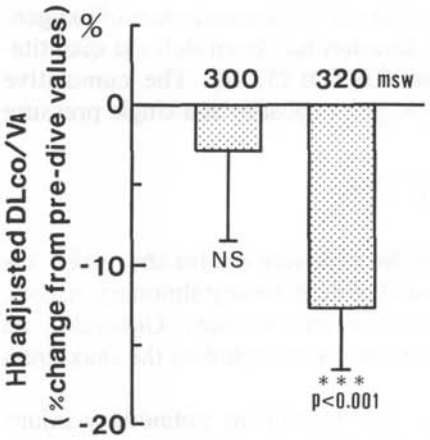

Fig. 3. Change of Hb-adjusted DLCO/VA right after surfacing. Values are means ± SD.

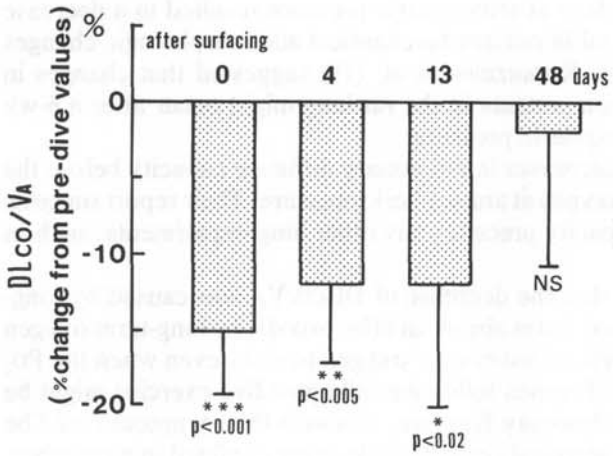

Fig. 4. Change of DLCO/VA in the 320-msw postdiving periods. Values are means ± SD.

Right after surfacing there were no complaints of cough, chest tightness, chest pain, and dyspnea at rest. Some divers in this study however complained of slight dyspnea after mild postdive exercise.

DISCUSSION

Our results indicated that P_{O_2} less than 0.5 atm abs could induce a decrease in pulmonary function (DLCO/VA) during prolonged oxygen exposure even when the decrease in Hb, which is often seen in hyperoxic exposure, was taken into account. What is the reason for this decrease?

The absence of DCS and scarcity of detected microbubbles in our cases suggest little relationship between the decompression-induced microbubbles or DCS and the decrease of DLCO/VA. Consequently, it is unlikely that the reduced DLCO was caused by microbubble insult in the lung.

It is widely accepted that exposure to hyperoxia produces progressive lung injury (1, 2). This injury becomes progressively more severe as the inhaled P_{O_2} and exposure

duration increase (3). To aid continuous tracking of the toxic consequences of oxygen, exposure to different combinations of P_{O_2} and duration has been defined quantitatively in terms of a unit pulmonary toxic dose (UPTD) (5, 14). The cumulative pulmonary toxic dose (CPTD) for a continuous oxygen exposure at a single pressure in UPTDs is expressed as

$$\text{CPTD} = t \cdot \sqrt[-1.2]{0.5/(P-0.5)}$$

where t = exposure time in minutes, $P = P_{O_2}$ of the exposure in atm abs, and 0.5 is an approximate pressure asymptote for development of detectable pulmonary effects. In this equation, P_{O_2} under 0.5 atm abs is not taken into account. Generally, an inspired O_2 tension of 0.5–0.6 atm abs for infinite time is accepted as the maximum limit for humans (3, 15, 16).

Hayatdavoudi et al. (17) reported, however, that significant pulmonary injury occurred in rats exposed to 60% O_2 at 1 atm abs for 7 days and that the pulmonary site of injury was the pulmonary capillary endothelium. Holm et al. (18) demonstrated that exposure to 60% O_2 for 21 days at atmospheric pressure resulted in a decrease in arterial P_{O_2} and induced several important biochemical and morphologic changes in alveolar type II pneumocytes. Kutsuzawa et al. (19) suggested that changes in water distribution and protein components in the rat lung might occur after a 6-wk exposure to 50% O_2 under atmospheric pressure.

Caldwell et al. (20) observed decreases in pulmonary diffusing capacity before the fall in VC in men breathing 98% oxygen at atmospheric pressure. Their report suggests that the decrease in diffusing capacity precedes any other lung impairments, such as decreased VC.

Therefore, we conclude first, that the decrease of D_{LCO}/V_A was caused by long-term exposure to oxygen close to 0.5 atm abs or, in other words, by long-term oxygen toxicity. We must consider the risk of pulmonary oxygen toxicity even when the P_{O_2} is below 0.5 atm abs. The slight dyspnea following mild postdive exercise might be due in part to this decrease of pulmonary function, although this symptom could be partly attributable to the lack of physical activity while being confined in a chamber. Second, the diffusing capacity is a more sensitive index of pulmonary oxygen toxicity than the VC that is traditionally used. D_{LCO} test is preferable in postdive tracking or regular routine health examination of saturation divers.

Manuscript received March 1990; accepted January 1991.

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