

Pulmonary mechanical function and diffusion capacity after deep saturation dives

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Abstract

To assess the effects of deep saturation dives on pulmonary function, static and dynamic lung volumes, transfer factor for carbon monoxide (Tl_{CO}), delta- N_2 , and closing volume (CV) were measured before and after eight saturation dives to pressures of 3.1–4.6 MPa. The atmospheres were helium-oxygen mixtures with partial pressures of oxygen of 40–60 kPa. The durations of the dives were 14–30 days. Mean rate of decompression was 10.5–13.5 kPa/hour. A total of 43 divers were examined, six of whom took part in two dives, the others in one only. Dynamic lung volumes did not change significantly but total lung capacity (TLC) increased significantly by 4.3% and residual volume (RV) by 14.8% ($p < 0.05$). CV was increased by 16.7% ($p < 0.01$). The Tl_{CO} was reduced from 13.0 ± 1.6 to 11.8 ± 1.7 mmol/min/kPa ($p < 0.01$) when corrected to a haemoglobin concentration of 146 g/l. Effective alveolar volume was unchanged. The increase in TLC and decrease in Tl_{CO} were correlated ($r = -0.574$, $p < 0.02$). A control examination of 38 of the divers four to six weeks after the dives showed a partial normalisation of the changes. The increase in TLC, RV, and CV, and the decrease in Tl_{CO} , could be explained by a loss of pulmonary elastic tissue caused by inflammatory reactions induced by oxygen toxicity or venous gas emboli.

During deep saturation dives, the lungs are exposed to an artificial atmosphere with high density and usually a raised partial pressure of oxygen of 40–60 kPa during decompression to facilitate inert gas elimination. The increased work of breathing imposed by the increased density of the breathing gas is depth related¹ and the ventilatory capacity will eventually become a limiting factor for physical work

capacity. During long exposures, toxic effects of oxygen in this concentration range cannot be excluded and during decompression the venous gas microemboli filtered in the pulmonary circulation may also induce inflammatory reactions in the lungs and gas exchange abnormalities.^{2,3}

An increase in vital capacity has in some cases been reported after saturation dives, attributed to a training effect of respiratory muscles.^{4,6} Hyacinthe *et al*⁷ and Cotes *et al*⁶ have also shown reduced transfer factor for carbon monoxide (Tl_{CO}) immediately after two deep dives to a pressure equal to 3.1 MPa. This could be an effect of oxygen toxicity but effects of microembolisation cannot be excluded. Probably all decompressions from those depths will produce venous gas emboli filtered in the pulmonary circulation but not necessarily associated with clinical decompression sickness.⁸

The possibility of long term effects on pulmonary function of professional diving is still controversial. The cross sectional studies of divers' lung function by Watt,⁹ Davey *et al*,¹⁰ and Crosbie *et al*¹¹ show larger than predicted vital capacities of divers but lower than predicted maximal flow rates at low lung volumes. This may indicate an airflow limitation but it is not known whether it is an adaptive response to diving or a progressive deterioration of lung function.

We have measured pulmonary function before and after eight deep saturation dives in Norway during the period 1983–6 to evaluate the magnitude and functional significance of changes in pulmonary function.

Methods

THE DIVES

Eight dives numbered 1 to 8 (table 1) in the depth range equivalent to a pressure of 3.1–4.6 MPa (300–450 metres of sea water—msw)* were studied. Two were open sea dives (dives 5 and 6) whereas the others were simulated in the NUTEC onshore hyperbaric chamber complex. Welding trials were performed in dives 2 and 8. Equipment and operational procedures were tested both in the dry and wet. The durations of the dives were 14–30 days. The mean rate of decompression was 10.5–13.5 kPa/h. The atmosphere was

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*1 MPa = 100 msw = 10 bar, 100 kPa = 10 msw = 1 bar.

helium-oxygen mixtures with partial pressures of oxygen of 40–60 kPa. Table 1 gives the characteristics of each dive. The number of divers in each saturation dive was four to nine, giving a total of 49 man dives. The diving procedures and the protocol for medical and physiological monitoring of the divers were approved by the Regional Ethical Review Committee (dives 1–5) and the ethical committee of the Norwegian Research Council for Science and the Humanities (dives 6–8).

THE DIVERS

Forty three professional divers participated in the dives. Their average age was 30.3 years (range 23–39), weight 78.3 kg (range 67–91), and height 180.2 cm (range 168–193). Ten were current smokers, four ex-smokers, and 29 non-smokers. Their experience as saturation divers was on average 5.8 years (range 1–9) and their total number of days in saturation on average 270 days (range 5–600). On the first examination before the dives, they had all passed the annual medical examination required by the Norwegian Directorate of Public Health for offshore diving. The time since their last routine saturation dive (less than 1.9 MPa) was at least four weeks. Six divers participated in two of the deep saturation dives described here and the time between their deep dives was from five months to two years.

PROTOCOL

The divers were first examined four to six weeks before the dives (pre-dive) and re-examined one to three days (first post-dive) and four to six weeks (second post-dive) after the dives. Three divers were examined at other institutes pre-dive and their results are not included in this study. A second post-dive examination was not done after dives 1 and 5. From 1983 to 1984 (dives 6–8) the examinations included clinical examination, dynamic lung volumes, and diffusion capacity. Later (dives 1–5), static lung volumes and distribution of ventilation were also included. Chest radiographs were taken in connection with dives 1 and 4 only.

Table 1 Characteristics of the dives

Dive	No of divers	Pressure (MPa)	Duration* (days)	Partial pressure of oxygen† (kPa)
1	6	3.7	2+3+13	40–50
2	6	3.7	2+10+13	40–50
3	6	3.7	2+12+13	40–50
4	6	4.6	2+10+18	40–50
5	4	3.1	1+3+10	40–50
6	9	3.1	1+9+9	40–60
7	6	3.6	2+11+11	40–60
8	6	3.6	2+6+11	40–50

*Compression time + bottom time + decompression time.

†Bottom phase and decompression phase.

ASSESSMENT OF PULMONARY FUNCTION

Static lung volumes

The multibreath nitrogen washout technique was used to measure functional residual capacity (FRC). Combined with the measurements of expiratory reserve volume (ERV) and inspiratory vital capacity (IVC), total lung capacity (TLC) and residual volume (RV) were calculated.

Dynamic lung volumes

A minimum of three satisfactory forced vital capacity manoeuvres were performed.¹² The forced expiratory vital capacity (FEVC), forced expired volume in one second (FEV₁), and peak expiratory flow rate (PEF) were taken as the highest readings obtained. The forced mean mid-expiratory flow rate (FEF_{25–75%}) and forced expiratory flow rates at 50% and 75% of FEVC expired (FEF_{50%}, FEF_{75%}) were taken as the highest readings from flow volume curves not differing by more than 5% from the highest FEVC. The forced inspiratory vital capacity (FIVC), forced inspired volume in one second (FIV₁), forced inspiratory flow rate at 50% of FIVC (FIF_{50%}), and peak inspiratory flow rate (PIF) were taken as the highest readings obtained. Maximum voluntary ventilation (MVV) was measured as the highest ventilation sustained for 12 seconds.

Diffusion capacity

Tl_{CO} was measured by the single breath holding method.¹² Effective alveolar volume (V_A) was then measured simultaneously by helium dilution and transfer per unit effective alveolar volume (K_{CO}) was calculated. Tl_{CO} was corrected to a haemoglobin concentration of 146 g/l.

Distribution of ventilation

During the multibreath nitrogen washout test for measuring FRC, the nitrogen washout time (NWT) (time to bring expired N₂ concentration below 2%) and the lung clearance index (LCI) (volume ventilated to bring expired N₂ concentration below 2% relative to FRC) were measured. The slope of phase 3 of the single breath O₂ test—delta N₂—was also measured along with closing volume (CV) at the point of inflection between phases 3 and 4. Closing capacity (CC) was calculated as the sum of RV and CV.

The pre-dive and post-dive examinations were performed with the same equipment and technicians on each occasion at least two hours after breakfast without tea or coffee and with no smoking in the last two hours before the examination. Volume and test gas calibrations were done before each test and the results were corrected to the BTPS condition.

STATISTICS

For comparison of results between pre-dive and

postdive examinations paired Students *t* test was applied. Least squares linear regression analysis was done for correlation analysis. Differences between examinations were calculated as difference from the mean. All data are expressed as mean \pm 1SD. A *p* value less than 0.05 was considered significant.¹³

Results

On the predive examinations none of the divers reported pulmonary symptoms and the clinical examinations of the heart and lungs were considered normal. Immediately after the dives retrosternal discomfort was reported by 20 divers, including nine with a non-productive cough provoked by deep inspirations. Otherwise non-specific symptoms of weakness, general fatigue, and insomnia were reported. Clinical examinations were still normal and in the two dives where chest radiographs were taken they were also normal. All symptoms had gradually disappeared during the first two weeks after the dives.

STATIC LUNG VOLUMES

In 24 divers diving to 3.7 and 4.6 MPa there was a significant increase in TLC of 4.3%, FRC of 11.6%, and RV of 14.8% from the predive to first postdive examination. The IVC and ERV were unchanged (table 2). On the second postdive examination the TLC and its subdivisions were partially normalised (fig 1).

DYNAMIC LUNG VOLUMES

There were no changes from the predive through the first and second postdive examinations when all dives

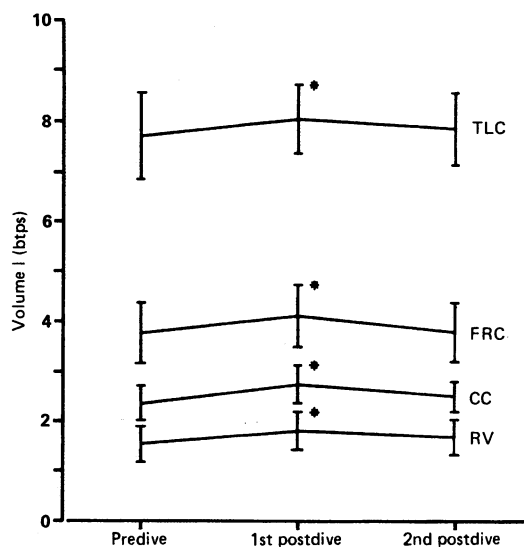


Fig 1 Static lung volumes on predive and postdive examinations. *Significantly different from predive.

were pooled (table 2). In dives 2, 3, and 4 FEV₁ increased significantly from the predive to first postdive examination by 3.8%, 5.6%, and 8.1% respectively, whereas FEV₁ and flow rates at other lung volumes were unchanged (fig 2). As shown in figure 3, the outline of the flow volume curves did not differ, only their positions related to absolute lung volume, which means that at the same absolute lung volume, flow rate was lower postdive. The change in FEV₁ (all dives) did not correlate with diver's age

Table 2 Results of selected pulmonary function tests. *n* refers to number of examinations

	Predive (<i>n</i> = 46)	First postdive (<i>n</i> = 46)	Second postdive (<i>n</i> = 38)
Dynamic lung volumes:			
FVC (l)	6.10 \pm 0.68	6.17 \pm 0.64	6.16 \pm 0.60
FEV ₁ (l)	4.81 \pm 0.55	4.78 \pm 0.56	4.69 \pm 0.55
FEF _{25-75%} (l/s)	4.57 \pm 1.13	4.41 \pm 1.15	4.54 \pm 1.20
PEF (l/s)	11.95 \pm 1.43	11.71 \pm 1.48	11.77 \pm 1.37
FIV ₁ (l)	5.52 \pm 0.85	5.25 \pm 0.78	5.31 \pm 0.80
PIF (l/s)	8.81 \pm 2.27	8.40 \pm 2.20	8.63 \pm 2.34
MVV (l/min)	174 \pm 19	182 \pm 24	171 \pm 24
Static lung volumes:			
TLC (l)	n = 24 7.70 \pm 0.85	n = 24 8.04 \pm 0.67*	n = 24 7.86 \pm 0.70
FRC (l)	3.77 \pm 0.74	4.11 \pm 0.61*	3.79 \pm 0.68
RV (l)	1.56 \pm 0.36	1.81 \pm 0.37*	1.69 \pm 0.34
CV (% VC)	11.0 \pm 1.6	13.0 \pm 1.8*	11.2 \pm 2.1
CC (l)	2.35 \pm 0.41	2.74 \pm 0.37*	2.50 \pm 0.40
Diffusion capacity:			
Tl _{CO} (mmol/min/kPa)	n = 46 13.00 \pm 1.62	n = 46 11.82 \pm 1.66**	n = 38 12.62 \pm 1.66
K _{CO} (mmol/min/kPa/l)	1.60 \pm 0.20	1.44 \pm 0.17*	1.53 \pm 0.21
V _A (l)	8.13 \pm 0.80	8.20 \pm 0.73	8.24 \pm 0.84
Hb (g/l)	147 \pm 11	138 \pm 15	139 \pm 13
Packed cell volume (volume fraction)	0.44 \pm 0.02	0.42 \pm 0.02	0.42 \pm 0.03

*Significantly different from predive *p* < 0.05.

**Significantly different from predive *p* < 0.01.

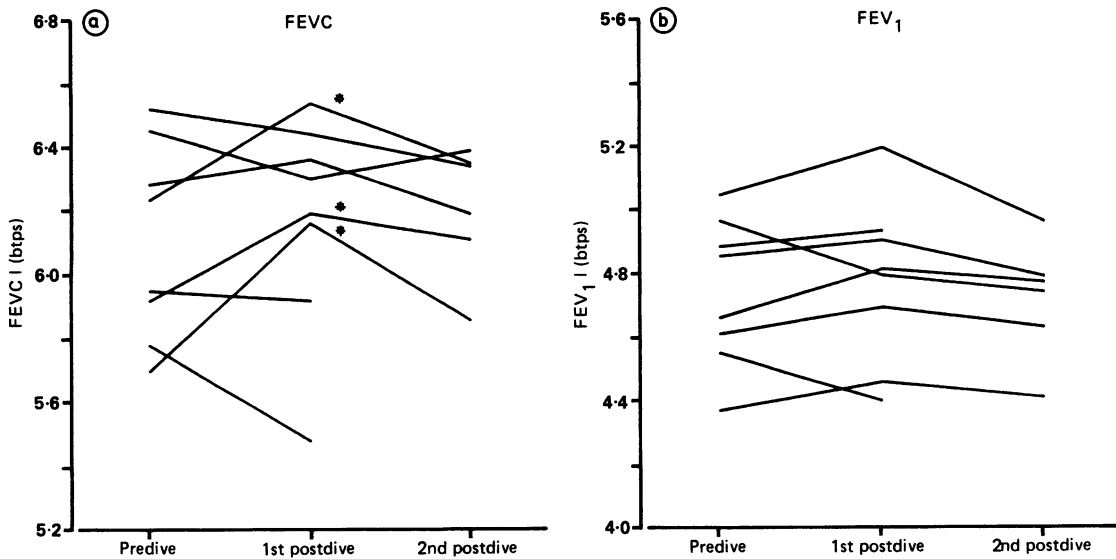


Fig 2 FEVC and FEV₁ pre-dive and post-dive. Means for each dive, Nos 1-8, are plotted. *Significantly different from pre-dive ($p < 0.05$).

($r = 0.139$) or depth ($r = 0.196$) or rate of decompression ($r = 0.224$) but correlated positively with duration of the dives ($r = 0.359$, $p < 0.02$). The dives where welding was performed showed no specific trends compared with the other dives.

DISTRIBUTION OF VENTILATION

There were no changes in the distribution indices delta-N₂, LCI, or nitrogen washout time. The clos-

ing volume was significantly increased from 11.0 ± 1.6 to $13.0 \pm 1.8\%$ of VC ($p < 0.05$). Since both RV and CV were increased, closing capacity was also increased (table 2).

TRANSFER FACTOR FOR CARBON MONOXIDE

Tl_{CO} was significantly reduced by 9.5% immediately after the dives. K_{CO} was reduced by the same magnitude (9.0%). Figure 4 shows the results of Tl_{CO}

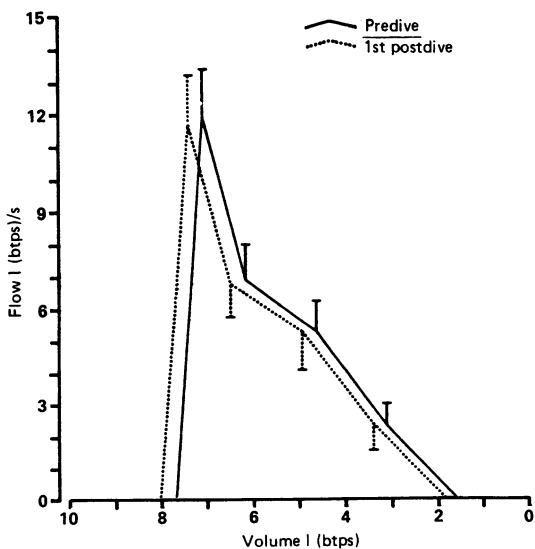


Fig 3 Flow volume loops at pre-dive and first post-dive examinations related to absolute lung volume ($n = 24$). 1 SD is shown in only one direction.

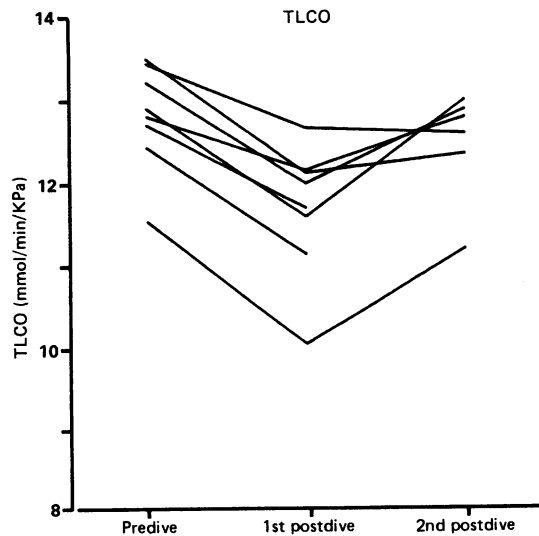


Fig 4 Tl_{CO} pre-dive and post-dive. Means for each dive, Nos 1-8, are plotted. All means at first post-dive examination differ significantly from pre-dive ($p < 0.05$).

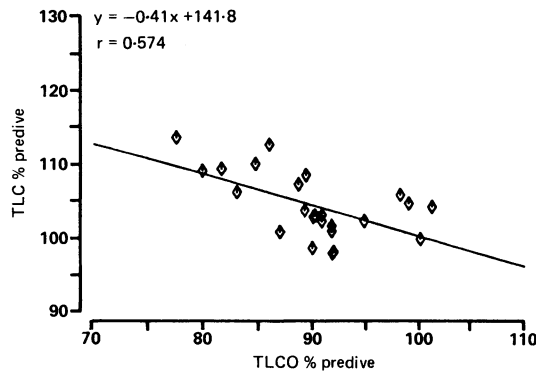


Fig 5 Correlation plot between change in TLC and Tl_{CO} from predictive to first postdive examination.

in each dive. The reduction in Tl_{CO} did not correlate with duration of the dive ($r = 0.123$), diver's age ($r = -0.060$), or depth ($r = 0.099$) but there was a significant negative correlation between change in TLC and change in Tl_{CO} ($r = -0.574$, $p < 0.02$) (fig 5). Tl_{CO} was still reduced on the second postdive examination four to six weeks after the dives (dives 1 and 5 not examined), the reduction being 5.6% (NS). The reduction in Tl_{CO} of divers decompressed at a partial pressure of oxygen of 50 kPa and 60 kPa were 9.7% and 9.1% respectively (NS).

Discussion

Twenty of the 43 divers reported chest symptoms persisting for up to two weeks after the dives. The symptoms were consistent with a tracheobronchitis that may be induced by raised partial pressure of oxygen. A reduction in VC, which is a characteristic finding in oxygen toxicity, was not shown. The results of the assessment of static and dynamic lung volumes indicate an expansion of the lungs. Some reports indicate an increase in VC immediately after deep dives⁵ but this was seen in only three of our dives. The TLC and VC may be increased by a training effect because of raised breathing resistance. The results of training of respiratory muscles by loaded breathing¹⁴ and swimming¹⁵ have shown an increase in FRC, TLC, and VC but unchanged RV. An increase in residual volume has also been shown after a saturation dive to 1.86 MPa.⁴ The significant correlation between duration of the dive and increase in FEVC could reflect a training effect. There was no increase in PEF, MVV, or maximal inspiratory flow rates, which are effort dependent, to support this. Maximal respiratory pressures were not measured in this study but Cotes *et al* did not find changes in maximal respiratory pressures after the dive to 3.1 MPa where there was an increase in FEVC of 6.8%.⁶

The capillary endothelial cells are probably the most vulnerable structure to hyperoxic injury with

disruption of the endothelial lining and occlusion of capillaries and small arterioles.¹⁶ Venous gas embolisation will also result in a microvascular injury with occlusion of capillaries and increased permeability of the endothelium.^{17,18} Reduced static lung volumes, which are characteristic of oxygen toxicity, will not be seen unless the lymphatic drainage capacity of the lungs is overloaded, forming oedema. Existing oxygen tolerance tables indicate an oxygen concentration of 50 kPa as being harmless,¹⁹ but there exists no experience to support this when exposure time is more than two weeks. These tables are based on changes in vital capacity as the measure of oxygen toxicity but other lung function variables such as the Tl_{CO} would probably detect changes at an earlier stage.

The inflammatory processes associated with oxygen toxicity and gas embolism injury may destroy pulmonary elastic tissue through the mechanisms of oxygen radicals. In a study by Riley *et al* in mice exposed to hyperoxia a degradation of collagen was shown with a reduced pulmonary recoil pressure and histopathological evidence of an emphysematous lesion eight weeks after exposure.²⁰ Oxygen radicals are also involved in the process of injury after air embolisation.^{21,22} The pattern of changes in static lung volumes in our study may be explained by that mechanism resulting in a loss of pulmonary elastic tissue. The increase in closing capacity and reduction of Tl_{CO} is also consistent with this.

The characteristics of divers' lung function described by Crosbie *et al* indicates a slight bronchial obstruction in divers.¹¹ It is not known whether it is a real obstruction or a result of the divers' significantly higher than predicted lung volumes. Our study indicates that a slight obstruction might be induced by the dives when looking at both the increase in RV and TLC and maximum flow rates at absolute lung volumes. At a given absolute lung volume, maximum expiratory flow is lower postdive.

The functional significance of changes in pulmonary function after deep dives is indicated by the striking reduction of Tl_{CO} . The transfer of CO from alveoli to haemoglobin depends on the area available for diffusion, the condition of the membrane over which diffusion takes place, the capillary area with its blood volume, and haemoglobin concentration. The measurement of static lung volumes as well as V_A show that the lung volume and thereby alveolar area available for diffusion is almost unchanged. The haemoglobin concentration has been corrected for and does not explain the difference between predictive and postdive results. The explanation is then reduced to an increased thickness of the diffusive membrane or a reduced capillary area available for diffusion.

Reductions in Tl_{CO} have been shown after exposure to both normobaric and hyperbaric oxygen^{6,7,23,24} and the time for complete recovery may

be several weeks. In the study by Puy *et al* the TI_{CO} was partitioned into the membrane and blood components, and the main reduction in TI_{CO} was in the blood component, indicating changes in the pulmonary capillary bed.²⁴ Hyacinthe *et al* have shown a 13% reduction of TI_{CO} after a decompression from 3.1 MPa at a partial pressure of oxygen of up to 80 kPa.⁷ This is definitely a toxic oxygen concentration. The TI_{CO} was still reduced at two weeks after this dive. In the study by Cotes *et al* after a dive to 3.1 MPa, a reduction in TI_{CO} of 9.6% was shown and the recovery was complete at four weeks postdive.⁶ Our data indicates a recovery time of more than five weeks depending on the initial reduction. TI_{CO} does not significantly correlate with duration of the dives, but seems in some way to be related to the change in TLC as reflected by the negative correlation between change in TI_{CO} and TLC.

Other significant correlations between changes in lung function parameters and characteristics of the hyperbaric exposure or the divers themselves were not found, such as age, depth, rate of decompression, or partial pressure of oxygen. The individual response to the environmental challenges differ considerably and with so many aetiologic factors acting simultaneously makes it difficult to draw conclusions. The range of variation for oxygen concentrations and rates of decompression in these dives are also small. Systematic studies of routine operational saturation diving to less than 2 MPa should be carried out, making the correlations over a wider range for the independent variables.

It may be concluded that significant pulmonary changes after deep saturation diving are induced by the dive and that the recovery may take several weeks. The findings of this study support the findings in cross sectional studies which indicate the development of airflow limitation in professional divers. So long as effects on long term health are mostly unknown, efforts should be taken to assure that a complete recovery has taken place before other dives are done, not only deep dives but also routine diving. The TI_{CO} test and static lung volumes should be considered in the follow up examinations of divers.

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