Divers' pulmonary function after open-sea bounce dives to 10 and 50 meters

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National Institute of Occupational Health, Pb 8149 Dep., N-0033 Oslo, Norway; Norwegian Underwater Technology Centre A/S, P.O. Box 6, N-3034 Ytre Laksøy, Norway; Section of Medical Statistics, University of Oslo, P.O. Box 1122 Blindern, N-0317 Oslo, Norway; and Norwegian Radiation Protection Authorities, P.O. Box 55, N-1345 Østerås, Norway

Skogstad M, Thorsen E, Haldorsen T, Melbostad E, Tynes T, Westrum B. Divers' pulmonary function after open-sea bounce dives to 10 and 50 meters. Undersea Hyperbaric Med 1996; 23(2):71-75.—We have studied pulmonary function before and 2 h after open sea dives to 10 and 50 m and 24 h after the dive to 10 m. Nine trainee divers participated in the dive to 10 m and 17 in the dive to 50 m. Mean time in water was 33 (32-62) min for the 10-m dive and 38 (26-76) min for the 50-m dive. Assessment of lung function included dynamic lung volumes and flows and transfer factor for carbon monoxide (TlCO). There were significant reductions (P < 0.05) in forced vital capacity of 5.8% (SD = 3.9) and 1.8% (SD = 2.8), in forced expired volume in 1 s of 6.6% (SD = 3.5) and 2.7% (SD = 2.4), in forced mid-expiratory flow rate of 10.3% (SD = 7.8) and 5.2% (SD = 6.5), and in TlCO of 11.3% (SD = 7.9) and 12.8% (SD = 5.9) 2 h after the 10- and 50-m dive, respectively. Our results indicate that factors related to submersion and increased breathing resistance contribute to changes in pulmonary function in the first hours after open-sea bounce dives.

decompression, diving, hyperoxia, pulmonary function

In surface-oriented diving with air as breathing gas the lungs are exposed to hyperoxia, decompression stress with the possibility of venous gas microembolism, and mechanical loads due to submersion, breathing apparatus, and increased gas density. All these factors of exposure have been shown to contribute to changes in pulmonary function after deep saturation dives (1-4). Few studies have looked upon effects of open-sea bounce dives. Catron et al. (5) did not find any changes in pulmonary mechanical function in 10 divers after a simulated air dive to 87 m. Studies on pulmonary gas transfer function were not included in that study. Dujic et al. (6) found a reduction in transfer factor for carbon monoxide 20-80 min after a simulated air dive to 45 m relating their findings to the load of venous gas microemboli. These experimental dives were, however, done in hyperbaric chambers with the divers being exposed in dry environment, without submersion in water.

In this study pulmonary function was measured before and after open-sea dives to 10 and 50 m, comprising all factors associated with diving exposure.

MATERIAL AND METHODS

The divers were all male students attending a course at a school for professional divers situated in the Oslo area. A total of nine students participated in the first study (Table 1).

Six divers were non-smokers and three were daily smokers. Seventeen male students (Table 1) participated in the second experiment. Five were daily smokers and one was a former smoker. No diver participated in both dives. Smoking was prohibited 1 h before the dive and during the follow-up period. There was no difference in age, smoking habits, height and weight, and in expected lung function between the groups that participated in the two dives.

The dives (exposure) to a depth of 10 meters of sea water (msw) took place the same day for all participants. The mean time spent in water was 53 (32-62) min. The divers reached depths to 8-11 msw. The dives to a depth of 50 msw took place on 2 consecutive days. The mean time spent in the water during the dive was 38 (26-76) min. All subjects, except one, reaching 38 m, dived down to 50 m. The divers swam along a line in open sea to the 50-m mark and returned to a basket situated at 9 m which was connected to an elevator. Inside the basket the divers were elevated gradually and had a controlled decompression according to the Norwegian decompression tables (7). The divers did no work during the dive. Nine divers underwent 15 min of decompression and eight divers underwent 25 min or more of decompression, depending on bottom time. The mean oxygen load, expressed as the Unit Pulmonary Toxic Dose (UPTD), based on the pulmonary oxygen tolerance curves in normal
Table 1: Age, Height, Weight in Nine Male Divers Participating in a Single Open-sea Dive to 10 m and 17 Male Divers Participating in a Single Dive to 50 m

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th></th>
<th>Standard Deviation</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10-m dives</td>
<td>50-m dives</td>
<td>10-m dives</td>
<td>50-m dives</td>
</tr>
<tr>
<td>Age, yr</td>
<td>27.1</td>
<td>26.5</td>
<td>5.1</td>
<td>3.8</td>
</tr>
<tr>
<td>Height, cm</td>
<td>178.3</td>
<td>180.5</td>
<td>7.2</td>
<td>5.3</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>79.6</td>
<td>80.8</td>
<td>12.1</td>
<td>9.3</td>
</tr>
</tbody>
</table>

men according to Clark and Lamberts (8), was 52 (SD = 16). The divers used air as breathing gas.

All divers wore umbilical-type air supplied demand equipment, a Kirby Morgan band mask 18B or a Kirby Morgan super lite helmet 17B and neoprene suits.

Pulmonary function testing was performed using the Jaeger MasterLab (Erich Jaeger GmbH & Co KG, Wuerzburg, Germany). The spirometer was calibrated with a 2-liter syringe twice daily and test gas calibrations were also performed twice daily using the instruments automatic calibration program. The best results according to ATS criteria, of at least three flow-volume maneuvers performed before and after the dives, were used for analysis (9,10). The forced vital capacity (FVC), forced expired volume in one second (FEV1), and forced mid-expiratory flow rate (FEF25-75%) were measured before and 2 h after the dives to 10 and 50 m and before, and 24 h after the dive to 10 m. The transfer factor for carbon monoxide (TlCO) was measured by the single breath-holding method (11). Two measurements of TlCO were made on three occasions: before the dives to 10 and 50 m, 2 h after the dive to 50 m and 2 and 24 h after the dive to 10 m. The average of the two measurements was used for analysis. Effective alveolar volume (Vd) was measured simultaneously by helium dilution, and the transfer factor per unit effective alveolar volume (Kco) was calculated.

Student's paired t test, Student's two group t test, and correlation analysis were used in the data analysis. Simple linear regression analysis was used to find a possible effect of time on change in FVC and FEV1 in each group of divers. A P value of less than 0.05 was considered significant. All data are expressed as means (SD).

RESULTS

The changes in the lung function variables comparing pre- and post-dive values in the dive to 10 m are shown in Table 2 and Figs. 1 and 2. A statistically significant reduction in FVC, FEV1 and FEF25-75% were apparent 2 h after the dive to 10 m of 5.8% (SD = 3.9), 6.6% (SD = 3.5), and 10.3% (SD = 7.8), respectively. These changes were back to base-line 24 h after the dive. The reduction in TlCO was not back to base-

line values the day after the dive, the reduction being 11.6% (SD = 6.2) 24 h post-dive.

Dynamic lung volumes and flows also changed significantly in the 50-m dive, the fall in FEF25-75% being the largest (Fig. 1). Single-breath TlCO measured 14.5 mmol · min⁻¹ · kPa⁻¹ (SD = 2.2) before the dive to 50 m, with a 13% reduction (SD = 5.9) 2 h after the dive. The results are illustrated in Fig. 2. There was no significant difference between smokers and non-smokers. Eight of the divers included in the dive to 50 m underwent decompression for 15 min, while the remaining nine divers completed a decompression period of 25 min. During the 2-h follow-up period there was no difference in TlCO between the two groups having different length of decompression. There was no correlation between change in TlCO and oxygen exposure (P = 0.7) in terms of UPTDs. There was a small reduction in VA (P < 0.05), but the changes in KCO showed the same pattern as in TlCO. The pattern of changes in the subject reaching only 38 m did not differ from the others.

There was a significant difference between the results of FVC (P = 0.006) and FEV1 (P = 0.003) obtained in the 10-m dives when these results were compared with the 50-m dive, the results giving a significant larger reduction for the 10-m dive.

There was no effect of time in the change in FVC and FEV1 in each group. For the change in FVC the estimated coefficients were 0.024 (SE B = 0.12) and 0.016 (SE B = 0.06) and for the change in FEV1, it was -0.055 (SE B = 0.11) and 0.05 (SE B = 0.05) in the 10- and 50-m dive, respectively.

DISCUSSION

We have found a significant reduction in the dynamic lung volumes and flows as well as in TlCO, after dives to 10 and 50 m. There were significant greater reductions in FVC and FEV1 in the dive to 10 m compared to the dive to 50 m. Exposure factors not being related to pressure may have contributed to the reduction in pulmonary function. The pattern of changes in pulmonary function indicate a combination of both obstructive and restrictive components. This pattern is...
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<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Mean (SD)</th>
<th>2 h After Mean (SD)</th>
<th>Before Mean (SD)</th>
<th>2 h After Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (1)</td>
<td>6.07 (1.0)</td>
<td>-5.8 (3.9)*</td>
<td>6.26 (1.0)</td>
<td>-1.8 (2.8)*</td>
</tr>
<tr>
<td>FEV₁ (1)</td>
<td>5.17 (1.0)</td>
<td>-6.6 (3.5)*</td>
<td>5.13 (0.9)</td>
<td>-2.7 (2.4)*</td>
</tr>
<tr>
<td>FEF₉₅-₇₅ (liter/s)</td>
<td>5.66 (1.7)</td>
<td>-10.3 (7.8)*</td>
<td>5.09 (1.2)</td>
<td>-5.2 (6.5)*</td>
</tr>
<tr>
<td>TlCO (mmol · min⁻¹ · kPa⁻¹)</td>
<td>13.9 (2.7)</td>
<td>-11.3 (7.9)*</td>
<td>14.5 (2.2)</td>
<td>-12.8 (5.9)*</td>
</tr>
<tr>
<td>KCO (mmol · min⁻¹ · kPa/liter)</td>
<td>1.8 (0.3)</td>
<td>-6.3 (8.9)*</td>
<td>2.0 (0.3)</td>
<td>-11.4 (4.5)*</td>
</tr>
<tr>
<td>V₅₃ (1)</td>
<td>7.58 (1.1)</td>
<td>-5.3 (1.8)*</td>
<td>7.21 (1.1)</td>
<td>-2.00 (3.2)*</td>
</tr>
</tbody>
</table>

*Values are shown as pre-values and change in percent (n = 9 in the 10-m dive and n = 17 in the 50-m dive). *

*Significantly changed from pre-dive values (P < 0.05).

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**FIG. 1** — Change in percent in maximal mid-expiratory flow (FEF₉₅-₇₅₉₃) in 9 and 17 divers in dives to 10 and 50 m.

**FIG. 2** — Change in percent in TlCO in 9 and 17 divers in dives to 10 and 50 m.
different from the changes seen after saturation dives (1-3,12) and simulated air dives (5,6) in which only changes in pulmonary gas transfer function is seen, without changes in pulmonary mechanical function. It is also different from the long-term effects of diving on pulmonary function, which is a pattern of changes indicating small airways dysfunction (4).

The exposure to hyperoxia was very small in the dives to 10 and 50 m. Changes in pulmonary function after oxygen breathing of long duration in both normobaric and hyperbaric conditions have been described (13-16). In terms of Units Pulmonary Toxic Dose (UPTD), the oxygen load was zero in the dive to 10 m and was on average 52 in the 50-m dive. There are no studies indicating that this oxygen exposure should result in change in lung function, and definitely not in vital capacity (17). Thus, exposure to hyperoxia has probably not contributed to the changes in lung function seen in this study.

Monitoring for venous gas microemboli was not done in this study, and can therefore not be excluded as a contributing factor. A supersaturation of only 135 kPa may result in formation of venous gas microemboli (18) and could therefore have occurred in both the 10- and 50-m dive. There was, however, no relationship between changes in the lung function variables and the duration of the 50-m dive. More bubbles would be expected in the dive with the longer bottom time, and more bubbles would be expected in the 50-m dive than in the 10-m dive. In simulated dry air dives in which venous gas microemboli were formed, there were no changes in the dynamic lung volumes and flows (5,6), but in one study there was a relationship between reduction in $Tl_{CO}$ and load of venous microemboli (6). Thus, venous microemboli may have contributed to the reduction in $Tl_{CO}$, but probably not to the reduction in the dynamic lung volumes and flows. The submersion in cold water combined with physical work during the bottom time would favor bubble formation, but if so, more in the 50-m dive than in the 10-m dive.

The major differences between open sea dives and simulated dry dives in hyperbaric chambers are the exposure to submersion and the respiratory load of breathing apparatus. Submersion results in a hydrostatic imbalance on the lung which adds to the respiratory mechanical load due to increased gas density and breathing apparatus, and results in redistribution of blood volume and body fluid (19). Cold water would have an additional effect on blood volume distribution causing cutaneous vasoconstriction (20), and would also cool the breathing gas. Breathing cold and dry gas results in bronchoconstriction (21,22). Airways resistance will also increase with increasing gas density (23). This response is operative in the larger airways, and does not usually result in reductions in forced vital capacity as seen in the present study, but the bronchoconstrictive effect may have contributed to the changes seen in the 10- and 50-m dives. We would expect respiratory heat loss and mechanical load to be a function of time. We found, however, no association between change in lung function in each group and time in water.

A mechanism that would explain all changes seen after these dives, including the reduction of $Tl_{CO}$, even 24 h after the dive to 10 m, would be that of subclinical pulmonary edema (24). Pulmonary edema of clinical significance has been demonstrated in swimmers and scuba divers (25), but the mechanism is not clear. Edema may also be the mechanism for reduced $Tl_{CO}$ seen after exhausting physical exercise in athletes (26). During a dive several mechanisms would favor edema formation. The combined effect of submersion with pooling of blood in the pulmonary circulation and exercise with increased cardiac output, will increase pulmonary arterial and capillary pressure. Increased breathing resistance will increase transpulmonary and thereby transcapillary pressure gradients at least during parts of the breathing cycle. Although attractive, the hypothesis of pulmonary capillary stress failure has to be followed up by measurements of vascular and pulmonary pressures. We may however conclude that factors related to submersion and increased breathing resistance contribute to changes in pulmonary function in the first hours after open-sea bounce dives.

This study was financially supported by Statoil's, "Fund for Research in Occupational Medicine., Oslo, Norway. We thank the Norwegian Commercial Diving School for excellent assistance and support.-Manuscript received December 1995; accepted April 1996.

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