Acute and long term effects of sport diving on respiratory function

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Introduction

Divers are exposed to decompression stress, increased work of breathing and hyperoxia that might impair respiratory function. Many studies have shown that deep saturation dives and professional compressed air dives can cause acute and long lasting changes in lung function. During past few years, we got some evidence about similar respiratory impairments even in the population of sport and recreational divers. Aim of this study was to evaluate the effects of active sport air scuba diving, using commonly practised dive profiles in this area, on pulmonary function.

Methods

A cross sectional study of pulmonary function in the group of 21 active sport scuba divers was compared with a matched subgroup of 21 healthy individuals who never dived. Mean time of diving experience was 16(6-30) years; mean time in the water was 1290(170-3000) hours; mean for greatest depth was 69(36-115) msw; mean time on the depths >30msw, given as percent of total time in the water, was 37(15-85) %. Divers were evaluated in their inactive period and in the water was 1290(170-3000) hours; mean for greatest depth was 69(36-115) msw; mean time on the depths >30msw, given as percent of total time in the water, was 37(15-85) %. Divers were evaluated in their inactive period and in the water was 1290(170-3000) hours; mean for greatest depth was 69(36-115) msw; mean time on the depths >30msw, given as percent of total time in the water, was 37(15-85) %.

Pulmonary function tests (PFT) : Spirometry, flow-volume loop, body plethysmography, single-breath transfer factor for carbon monoxide (TL,co,sb) with transfer coefficient (K co) measurement and predicted were performed according to the 1993 European Respiratory Society recommendations. Student's two group t test was used in the data analysis. After baseline, previously measured PFT, a subgroup of 7 divers performed the dive to 42 meters. The same PFT were repeated at 4 and at 24 hours after the dive. Bottom time was 22 minutes; ascent rate was 10m/min, with decompression stops on 6m/3minutes and 3m/11minutes. Student's paired t test was used in the data analysis.

Results

When we compared the results of PFT in the group of divers and healthy controls, we have found similar mean values of static and dynamic lung volumes, flows and airway resistance in the two groups. Mean values of TL,co,sb (p=0,007) and of K co (p=0,000) were significantly lower in the group of divers (83±22.1%pred. and 61±13.0% pred., respectively), than in the control group (100.1±15.4% pred. and 76.6±12.2% pred., respectively). After a single dive to 42 meters, we have found significant reductions of mean forced expiratory volume in 1 second (FEV1), before and 4 hrs after the dive (5,2±0,54 : 5,1±0,50, p=0,009), with recovery to pre dive values after 24 hours (5,3±0,68, p=0,26). We have also found significant reductions of mean forced expiratory flow rates at 75% of forced vital capacity (FEF75; 2,28±0,84 : 1,98±0,75, p=0,006), and mean TL,co,sb (11,89±1,84 : 11,45±1,52, p=0,041), 4 hours after the dive. Mean FEF75 remained significantly reduced 24 hours after the dive (2,04±0,7, p=0,028), and mean TL,co,sb (11,9±1,8, p=0,9) returned to values similar to the baseline.

Discussion/Conclusion

We have found acute as well as long term changes in pulmonary function in our study. It is well known that asymptomatic Doppler monitored bubbles could be found in many commonly practised dive profiles. According to DAN Europe studies, bubbles were present in 37% of all monitored recreational dives. It was stated earlier, that reduction in TL,co may reflect effects of venous gas microemboli (VGM) after air dives in which oxygen exposure was small, as it was in our study. The presence of VGM could be the cause for acute reduction of TL,co in our study, while the time for recovery to pre dive values took less than 24 hours. Disfunction of small airways which lasted more than 24 hours and was presented in significant reduction of FEF75, could be provoked by inflammatory reaction and even by capillary stress failure. Few studies have indicated raised exhaled NO and endothelin 1 plasma concentrations even after short hyperbaric air exposures, commonly practised by recreational divers. We concluded that intensive sport diving could impair diffusion of gases from alveoli to pulmonary capillaries. Underlying mechanism, including microembolization, inflammation, subclinical pulmonary edema and certain degree of hyperoxia are to be investigated. It is reasonable to suggest that, in addition to spirometry, pulmonary gas transfer should be regularly measured in sport and even recreational divers with longitudinal follow up of their pulmonary function.

References

