Diffusing capacity and spirometry following a 60-minute dive to 4.5 meters.

M. S. KOEHLE[1,2,3], A.N.H. HODGES[1,3], B.M. LYNN[1,3], M.F. RACHICH[1], D.C. MCKENZIE[1,2,3].

[1]Allan McGavin Sports Medicine Centre, University of British Columbia, 3055 Wesbrook Mall, Vancouver BC V6T 1Z3, Canada; [2]Department of Family Practice, University of British Columbia, 5804 Fairview Avenue, Vancouver BC V6T 1Z3, Canada; [3]School of Human Kinetics, University of British Columbia, 0-6081 University Boulevard, Vancouver BC V6T 1Z1, Canada

Koehle MS, Hodges ANH, Lynn BM, Rachich MF, McKenzie DC. Diffusing capacity and spirometry following a 60-minute dive to 4.5 meters. Undersea Hyperb Med 2006; 33(2):109-118.The purpose of this study was to assess the contribution of SCUBA to the pulmonary effects of diving to 4.5 meters depth in healthy subjects using a randomized crossover control condition. Ten healthy divers performed two 60-minute 'dives' using SCUBA in a swimming pool. The non-immersed 1 ATA SCUBA control exposure took place at ambient pressure in the laboratory. Thirty minutes prior to, and 30 and 90 minutes post-exposure, FVC (forced vital capacity), $FEV_{1.0}$ (forced expired volume), peak expiratory flow rate (PEFR), diffusing capacity (DL_{CO}), heart rate (HR) and temperature were measured. No significant differences were noted in HR, temperature or spirometry between the two conditions. A significant reduction in diffusing capacity occurred at 30 and 90 minutes after the pool dive (9.3% and 15.1%, respectively, $p< 0.05$). There was no concordant change in DL_{CO} following the non-immersed 1 ATA SCUBA control. Thus, a pool dive to 4.5 meters for 60 minutes causes a decrease in DL_{CO}, without a change in spirometry, while breathing from SCUBA equipment without immersion causes no significant change in lung function.

INTRODUCTION

Recreational diving is an enormously popular activity, with more than 5 million certified divers in the United States alone (1). Recreational divers have the potential to experience pulmonary problems as a direct consequence of diving. These include pulmonary barotrauma from trapped gases expanding on ascent and pulmonary edema from transudation from the pulmonary capillaries. Guidelines for divers with pulmonary conditions such as asthma are highly variable, reflecting a lack of understanding of the pulmonary effects of diving (2,3).

Recent studies of the pulmonary effects of diving have shown interesting changes in lung function following diving and "simulated" diving (we use the latter term to refer to diving in a wet pot in a hyperbaric chamber). Tetzlaff et al. (4) examined pulmonary function before and after dives to 10 meters and 50 meters in healthy divers (diver activity and position were not specified). The study demonstrated transient increases in airway resistance, forced expired volume in 1 second ($FEV_{1.0}$), and maximal-expiratory flow at 75% of vital capacity (MEF_{75}) following simulated dives to 10 meters and 50 meters (bottom time of 10 minutes). The changes were only significant following the dive to 50 meters. Diffusing capacity was not measured. In the only study of pulmonary function in divers conducted in the open sea, Skogstad et al.(5) examined FVC, $FEV_{1.0}$ and diffusing capacity (DL_{CO}), before and after surface-supply diving to 10 and 50 meters. Mean bottom time was 38 minutes,

the subjects did no work but body position was not specified. They found a transient decrease in all of the values post-dive. Reductions in FVC and $FEV_{1.0}$ were significantly greater following the 10-meter dive than after the 50-meter dive. The effect of depth is unclear from these studies. Another pulmonary consequence of diving is pulmonary edema of immersion (6). This phenomenon occurs infrequently in divers and open-water swimmers, usually after swimming in cold water. In divers, it has been reported to occur even in a swimming pool (7). Presenting symptoms include cough, dyspnea and haemoptysis. Physical examination often shows wheezes and crackles. When performed acutely, radiographic imaging is consistent with pulmonary edema. The condition can be fatal. A milder sub-clinical form of this edema may be occurring during regular dives, leading to a transient drop in diffusing capacity.

The SCUBA (self-contained underwater breathing apparatus) equipment used by the divers may play a role in the observed pulmonary function changes by two possible mechanisms. SCUBA tanks contain compressed unhumidified air that may be irritating to the airways of susceptible individuals. Secondly, by inspiring through a demand valve, there is a resistance to inspiratory flow inherent in SCUBA equipment. This resistance may also alter pulmonary function by causing more negative intrathoracic pressures on inspiration. No previous studies of pulmonary function and diving have used a control condition, thus it is difficult to determine whether observed changes in lung function are a result of immersion, breathing from the dive equipment, or a combination of the two.

The purpose of the present study was to assess the contribution of SCUBA to the pulmonary effects of diving in healthy subjects using a randomized crossover control condition. We hypothesized that pulmonary function changes would not be observed following the control exposure but that a 60-minute dive to 4.5 meters in a swimming pool would elicit alterations in pulmonary function similar to those observed at depths of 10 and 50 meters.

METHODS

Subjects

Following approval from the University of British Columbia Clinical Research Ethics Board, ten healthy sport divers (five men and five women) were recruited through the University SCUBA Club. Divers were previously certified in recreational SCUBA diving, and free from any history of respiratory disease. All subjects had normal baseline pulmonary function.

Design

The experiment consisted of a randomized crossover design, in which each subject acted as his or her own control. Subjects visited the lab a total of three times. On the first visit, informed consent was obtained; subjects were oriented to the lab, and familiarized with the pulmonary function testing protocols. On each of the subsequent two visits, subjects performed a 60-minute non-immersed 1 ATA SCUBA control exposure while breathing from SCUBA apparatus. The experimental dive occurred in the University of British Columbia swimming pool (at 4.5 meters) while the control exposure took place at ambient pressure in the laboratory. Exposures took place at least 48 hours apart.

Data Collection

Pulmonary function was measured 30 minutes before and 30 and 90 minutes post-exposure. Both spirometry and diffusion measurements were recorded using the same commercial apparatus (Collins DS/Plus II, Braintree MA). Measured variables included, FVC, $FEV_{1.0}$, peak expiratory flow rate (PEFR), forced expiratory flow rate between 25 and

75% of FVC (FEF_{25-75}) and diffusing capacity. The value from the better of two loops was used for each measurement. Lung diffusing capacity ($D_{L_{CO}}$) was measured using the single breath method according to American Thoracic Society Guidelines (8). A single diffusing capacity measurement was taken at each time point. Hemoglobin concentration was not corrected for. All apparatus was calibrated before each set of measurements.

On the days of data collection, subjects were instructed to refrain from exercise and caffeine intake. They were transported to the laboratory by automobile, to reduce pre-evaluation exercise. Upon arrival in the laboratory, subjects sat and rested for at least 15 minutes before any measurements were taken. To measure $D_{L_{CO}}$, seated subjects made a maximal inspiration (from residual volume) of a mixture comprising 20.9% oxygen, 9.7% helium and 0.3% carbon monoxide balanced with nitrogen. Subjects performed a 10 second breath hold before expiring maximally. The first expired liter was discarded, while the next 750mL was collected and considered to represent an alveolar sample. Carbon monoxide concentration was measured using an infrared analyzer (type 101, Morgan, Kent, U.K.). Criteria for an acceptable test included: total inspiration time less than 2 seconds, inspired volume of at least 90% of FVC, and breath hold time between 9 and 11 seconds. Data from the first acceptable test at each time point was used in the analysis.

Heart rate was recorded using a portable heart rate monitor (Timex, Middlebury, CT, USA), 30 minutes prior to, and 30 and 90 minutes post-exposure. Heart rate was also recorded during the pool dive and the non-immersed 1 ATA SCUBA control. Oral temperature was recorded at the same time as the pulmonary function measurements.

Divers used Conshelf 22 regulators (Aqualung, Vista CA) with an opening effort of 1.5-2.8 cmH_20 at 207 bar (0.6-1.1 in of H_20 at 3000 psi) at a flow effort of 25.5 m^3_n/hr (15 SCFM) at 965 kPa (140 psi) interstage pressure. Steel tanks were filled (with compressed air) to approximately 200 bar (3000 psi).

For the pool dive, the subjects were transported by automobile from the laboratory to the pool and not permitted to carry any of their equipment in order to minimize the exercise effect. Once at the pool, subjects donned their equipment (with assistance), including a mask, regulator, buoyancy compensation device, snorkel, fins, weight belt and wetsuit. The divers descended to the bottom of the pool (4.5m) and rested quietly in a seated position for 60 minutes. The pool temperature was approximately 27°C. At 60 minutes, subjects ascended to the surface, where they were assisted out of the pool and transported back to the laboratory for the post-dive testing. Subjects were permitted to drink ad libitum following both conditions.

During the control exposure, subjects remained in the laboratory. They breathed from a SCUBA tank and regulator while seated, watching movies for 60 minutes. The subjects were not wearing wetsuits during their control exposure.

Statistical Analysis

Heart rate, temperature, spirometry and diffusion measurements were examined by analysis of variance (ANOVA) with repeated measures over time (pre-test, 30 minute post-test and 90-minute post-test). The two experimental conditions (control exposure vs. dive) were compared at each time point. Post-hoc testing was performed using Tukey's HSD test. Diffusing capacity values were also compared between genders using Repeated Measures ANOVA. The change in diffusing capacity between the pre-test and the two post-tests was also compared between the control and experimental conditions using

repeated measures ANOVA. The level of significance was set at $p<0.05$ for all statistical comparisons.

RESULTS

Anthropometric data and baseline pulmonary function is presented in Table 1. Between the two genders, there was a significant difference in height, but not age or body mass. No significant differences were found between experimental conditions or time points for heart rate or temperature.

Spirometry values FVC, $FEV_{1.0}$, FEF_{25-75}, and PEFR are displayed in Table 2. No significant differences existed either between various time points or between the pool dive and the control conditions for any of these values.

Diffusing capacity values are presented in Table 3. In the control condition, the mean diffusing capacity remained unchanged at ~8.65 $mmol \cdot min^{-1} \cdot kPa^{-1}$ (25 $mL \cdot mmHg^{-1} \cdot min^{-1}$). In the experimental condition, DL_{CO} decreased at both 30 and 90 minutes post dive ($p< 0.05$). The mean change in DL_{CO} is presented in Table 3 and Figure 1 (individual values are presented in Table 4). In the experimental group, the diffusing capacity declined by 0.71 and 1.28 $mmol \cdot min^{-1} \cdot kPa^{-1}$ (2.05 and 3.71 $mL \cdot mmHg^{-1} \cdot min^{-1}$) at 30 and 90 minutes post-dive respectively. These values correspond to a decline of 9.3 and 15.1% over the pre-dive value. These differences are statistically different from the control condition ($p<0.001$). When *absolute* diffusing capacity was compared between male and female subjects, male subjects demonstrated a

Table 1. Age, anthropometric data and baseline pulmonary function for the ten subjects included in the study.

	Male		***Female***		***Total***	
	Mean	*Standard Deviation*	*Mean*	*Standard Deviation*	*Mean*	*Standard Deviation*
Age (years)	22.8	5.54	25.2	7.40	24.0	6.29
Height (cm)	175.0*	9.72	161.2*	6.42	168.1	10.64
Body Mass (kg)	70.62	11.93	58.48	10.19	64.6	12.26
	Mean (%Predicted)	*Range*	*Mean (%Predicted)*	*Range*	*Mean*	*Standard Deviation*
FVC (L)	5.07 (97%)	4.50-5.54	3.67 (100%)	3.13-4.32	4.37	0.83
FEV1.0 (L)	4.38 (100%)	3.93-5.00	3.17 (101%)	2.97-3.80	3.78	0.73
FEV%	86.0 (102%)	79-93	86.6 (101%)	80-95	86.3	5.3
FEF25-75 ($L \cdot s^{-1}$)	4.60 (97%)	3.55-6.02	3.83 (110%)	2.92-4.63	4.22	0.80
PEFR ($L \cdot s^{-1}$)	8.90 (94%)	7.56-10.97	7.21 (104%)	5.84-9.00	8.06	1.46

*-denotes statistically significant difference between male (n=5) and female (n=5) subjects ($p < 0.05$).

Table 2. Mean spirometry values for control and experimental conditions at various time points.

	Mean FVC (L) Control	*Mean FVC (L) Experimental*	*Mean $FEV_{1.0}$ (L) Control*	*Mean $FEV_{1.0}$ (L) Experimental*
Pre-Dive	4.36 ± 0.26	4.34 ± 0.93	3.71 ± 0.91	3.76 ± 0.78
Post-Dive 30 min	4.31 ± 0.89	4.34 ± 0.87	3.65 ± 0.72	3.67 ± 0.71
Post-Dive 90 min	4.32 ± 0.89	4.32 ± 0.84	3.65 ± 0.89	3.71 ± 0.84
	Mean FEF_{25-75} ($L \cdot s^{-1}$) Control	*Mean FEF_{25-75} ($L \cdot s^{-1}$) Experimental*	*PEFR ($L \cdot s^{-1}$) Control*	*PEFR ($L \cdot s^{-1}$) Experimental*
Pre-Dive	4.03 ± 1.04	4.16 ± 1.08	8.47 ± 1.76	8.43 ± 1.59
Post-Dive 30 min	3.90 ± 1.11	3.86 ± 0.96	8.18 ± 1.65	8.09 ± 1.60
Post-Dive 90 min	3.91 ± 1.11	4.03 ± 1.09	8.11 ± 1.68	8.08 ± 1.81

Variables displayed include forced vital capacity (FVC), forced expired volume in 1 second ($FEV_{1.0}$), mean forced expiratory flow between 25 and 75% of vital capacity (FEF_{25-75}) and peak expiratory flow rate (PEFR). Means are presented with standard deviations.

Table 3. Mean diffusing capacity values for control and experimental conditions at various time points.

	DL_{CO} ($mmol \cdot min^{-1} \cdot kPa^{-1}$) Control	*DL_{CO} ($mmol \cdot min^{-1} \cdot kPa^{-1}$) Experimental*	*Change in DL_{CO} ($mmol \cdot min^{-1} \cdot kPa^{-1}$) Control*	*Change in DL_{CO} ($mmol \cdot min^{-1} \cdot kPa^{-1}$) Experimental*
Pre-Dive	8.65 ± 2.48	8.96 ± 2.35	-	-
Post-Dive 30 min	8.66 ± 2.34	8.25 ± 2.91*	0.01 ± 0.39	-0.71 ± 0.97
Post-Dive 90 min	8.64 ± 2.45	7.68 ± 2.48*	-0.01 ± 0.51	-1.28 ± 0.83

Absolute values for diffusing capacity are displayed as well as the change in diffusing capacity. Means are presented with standard deviations. *-denotes statistically significant difference between control and experimental condition ($p < 0.05$).

Table 4. Individual values for the change in diffusing capacity post-intervention.

Subject Number	Percent Change in Diffusing Capacity (%)			
	30 minutes post-intervention		90 minutes post-intervention	
	Control	Experimental	Control	Experimental
1	+1.0	+0.8	-0.7	-7.2
2	+9.7	-12.0	+10.2	-19.7
3	+9.6	-16.0	+9.0	-23.9
4	-7.5	-38.0	-9.7	-36.9
5	-1.3	-10.2	-4.9	-13.6
6	-3.7	+6.0	+3.4	+2.3
7	+2.6	-11.0	+4.9	-12.3
8	-0.9	-11.2	-7.3	-10.5
9	+0.1	+2.8	-2.3	-14.8
10	-2.2	-3.7	0.0	-14.9
Mean	+0.7	-9.3	+0.2	-15.1
Standard Deviation	±5.4	±12.4	±6.6	±10.4

The change in diffusing capacity is displayed as a percent of the original value.

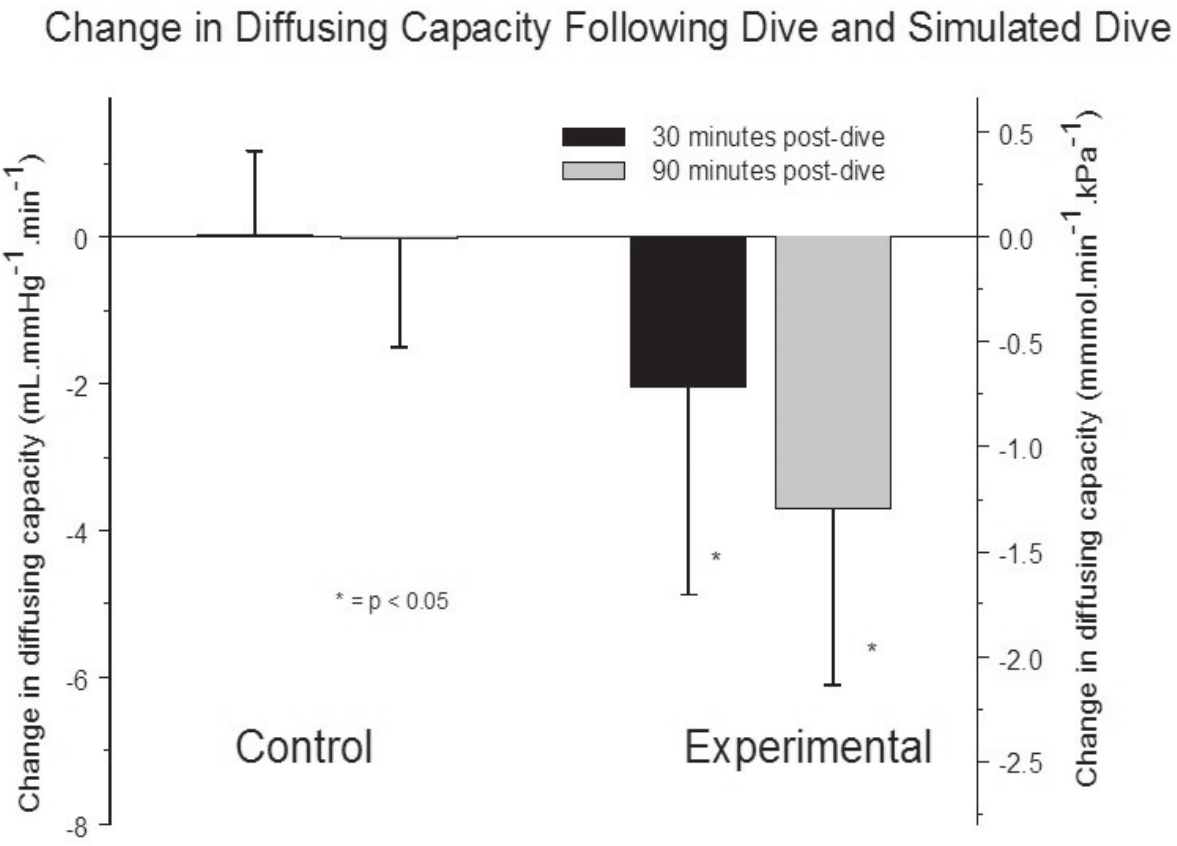

Fig. 1. The change in diffusing capacity following dive and control exposure ($mmol \cdot min^{-1} \cdot kPa^{-1}$). Data for the control condition are displayed on the left, pool dive data are displayed on the right. Error bars indicate standard deviation. *- denotes significant difference between experimental and control values ($p < 0.05$).

significantly higher diffusing capacity at all time points ($p<0.01$). With respect to *relative change* in diffusing capacity, there was no significant difference between the sexes.

DISCUSSION

Diffusing Capacity

The magnitude of the decrease in diffusing capacity observed in the present study is consistent with the 5 to 15% decrease observed in the previous studies of Dujic et al.(9) and Skogstad et al.(5) in dry and wet chamber exposures to 5.5 bar and 50 meters, respectively. Our study was unique to use a crossover design with the subjects acting as their own controls, and the only study to look at the diffusing capacity changes following the use of SCUBA equipment without submersion or hyperbaric exposure. These results indicate that the SCUBA used in the study itself does not cause the alteration in diffusing capacity. They also indicate that the altered $D_{L_{CO}}$ is possibly *not* depth dependant, as the magnitude of the effect was similar after a 4.5 meter dive to the previous results following dives simulating 10 and 50 meters. Previous evidence of the

absence of a depth effect on diffusing capacity was provided in the study by Skogstad et al. (5). These investigators compared diffusing capacity after dives to 10 and 50 meters and did not find evidence of an effect with depth.

Lung diffusing capacity (D_L) is a function of two factors: the membrane component (D_M), and the pulmonary capillary blood volume (V_c)[10]. Either component can provide resistance to the diffusion of gas from the alveolus to the capillary. In healthy individuals, each component contributes approximately equally to the total resistance to diffusion. Factors that can affect the membrane component include medical conditions such as pulmonary edema or fibrosis. The capillary blood volume component could be affected by fluid status or pulmonary embolism.

As the V_C component can be altered by exercise, the experimental design was careful to minimize exercise in the subjects. Heart rate was monitored to gauge the amount of exercise in each of the two conditions, and no significant difference was noted.

Similarly, body temperature was also monitored. Cold could affect lung function in a number of ways. Bronchoconstriction could reduce lung volumes and flows. Cold could also affect diffusing capacity through peripheral vasoconstriction leading to central pooling of blood. This would increase pulmonary capillary blood volume and diffusing capacity. While in the pool, the subjects were inactive and thus potentially prone to hypothermia. They therefore wore wetsuits for the pool dive. There was no significant difference in temperature between the two conditions at various time points during the data collection, ruling out the presence of hypothermia. Measuring oral temperature does not properly assess the extent of peripheral vasoconstriction in response to cold, so there could have still been some central pooling from the cold.

The actual cause of the diving-related diffusion impairment is not clear. The transient diffusing capacity alterations in the experimental group are consistent in magnitude and time course with previous studies, performed in open water (with surface gas supply) (5). Similar changes in diffusing capacity have also been seen with mock dives in a 'dry' hyperbaric chamber to 45 and 39 meters, respectively (9, 11). These dry chamber studies both used precordial Doppler ultrasound to assess the presence of venous gas microemboli in the pulmonary circulation. These microemboli are microscopic bubbles of nitrogen gas that have come out of solution as part of the decompression process. They have been demonstrated in divers after a dive within safe no-decompression dive limits, and have uncertain clinical significance. In both studies, the magnitude of the change in diffusing capacity correlated with the extent of the bubble signal on Doppler. The authors concluded that the observed changes in diffusing capacity were therefore a result of the microemboli interfering with pulmonary capillary blood flow, causing a limitation in $D_{L_{CO}}$. Eckenhoff et al. (12) examined the incidence of microbubbles using precordial and subclavian Doppler following 48 hour saturation exposures to 3 depths. They concluded that there was a 50% incidence of microbubbles after 48 hours at 135 kPa (3 meters seawater). According to their prediction curve, at the depth used in this study there would be an approximately 75% incidence of microbubbles following the 48 hour exposure. The subjects in this study dove for only one hour, so they would likely have a much lower incidence of microbubble formation.

As diffusing capacity depends on pulmonary blood volume, changes in total blood volume may play a role in the reduction in diffusing capacity. Following immersion there is an increase in venous return, activating atrial stretch receptors, which leads to an atrial natriuretic peptide-mediated diuresis. This immersion diuresis, by decreasing plasma

volume, may affect pulmonary capillary volume and hence diffusing capacity.

Sub-clinical pulmonary edema may be another mechanism for diving-related diffusion impairment. Several reports have described pulmonary edema in healthy individuals following diving and swimming (6, 13-17). Some cases have been linked to cold-water temperatures or strenuous exercise, but many cases have occurred in the absence of these factors. The proposed pathophysiology involves transudation and/or extravasation from the pulmonary capillary beds as a result of increased hydrostatic forces and capillary stress failure (17); (7, 16, 18). The central pooling (caused by immersion) can increase pulmonary blood volume and hence capillary hydrostatic pressure. Immersion in cold water would have a more profound effect, as peripheral vasoconstriction will further enhance the central pooling.

Another potential mechanism for the edema is the altered intrapulmonary pressures resulting from diving. Negative pressure pulmonary edema has been described as a result of breathing against a high resistance to flow (19). The hypothesized mechanism states that inspiration against resistance leads to more negative intra-alveolar pressures, and hence a higher transmural pressure gradient. This enhanced gradient causes transudation, leading to edema. Thorsen et al. (20) examined the effect of both immersion and inspiratory resistive loading on pulmonary function. They demonstrated that subjects who were *both* immersed (head-out) for 40 minutes *and* breathing against a resistive load, experienced a 7.3% reduction in diffusing capacity. Neither immersion nor resistive loading alone caused a significant decrease in diffusing capacity, indicating that each mechanism may have an additive effect on the other. In the present study, the control condition would be analogous to a mild resistive load, while the experimental condition would correspond to combined immersion and resistive loading. The reduction in diffusing capacity observed in our study may therefore have been caused by the addition of both resistive loading and immersion as in Thorsen's study.

Further evidence of an edema-like phenomenon occurred in one of the subjects who developed a mild cough after the pool dive. She denied aspiration. On auscultation, rales were present in both lung bases, which would be consistent with mild pulmonary edema. Thus, the observed changes in diffusing capacity could represent a subclinical presentation of pulmonary edema of immersion. Further study of these diffusing capacity changes may therefore provide valuable insight to the pathophysiology of clinical edema of immersion.

Spirometry

Our study failed to demonstrate any significant change in spirometry measurements (FVC, $FEV_{1.0}$, FEF_{25-75}, and PEFR) after diving and a control exposure. Only one previous study has looked at spirometry after SCUBA diving in healthy adults. Tetzlaff et al. (2001a), tried to examine the role of temperature in post-dive pulmonary function (4). They compared wet hyperbaric chamber dives under two temperature conditions. A small transient decrease in FVC, $FEV_{1.0}$, MEF_{75} was observed in the 'cold' group with no significant difference in the 'comfortable' groups. By attempting to control for temperature, the present study would be analogous to the 'comfortable' group, and thus we would not expect significant changes in pulmonary function. In the study by Tetzlaff et al. (2001) that demonstrated statistically significant changes in FVC and $FEV_{1.0}$ following diving, the measured difference was in the order of 3% or less, a magnitude that is not clinically significant. Thus, the present study adds support to the notion that any

transient changes in spirometry demonstrated in *healthy* divers are likely more related to the cold ambient temperature than to the diving process. By using a crossover design and a laboratory control, we are also able to confirm that breathing compressed air from a SCUBA tank does not affect the spirometry of healthy individuals.

Study Limitations

As previously mentioned, the subjects did not wear wetsuits while breathing from the SCUBA equipment during the control exposure. This difference was an effort to keep the subjects isothermic in both exposures. It is possible that the wetsuit itself might alter pulmonary function during the dive. Recent research (17, 21) has suggested that wetsuits produce little difference in lung volume. Therefore, the significance of this wetsuit discrepancy is likely negligible. Furthermore, in the control exposure, the subjects were not exposed to the same transportation stress as in the experimental exposure. This stress involved a slow walk to an automobile (about 100 meters) a 4-minute drive and then a 30-second walk to the lab. Subjects did not carry any of their equipment, and rested for ten minutes before pulmonary function testing was commenced. As heart rates were similar between control and experimental exposures, this transportation stress is not likely significant.

It would have been informative to have a third arm to the study in which subjects were immersed (with head out), but did not breathe from SCUBA equipment. This arm would have provided insight into the role of immersion alone in post-dive lung function. Additionally, fluid status was not assessed pre- and post-dive. Understanding the changes in fluid status would help determine the underlying mechanism of the reported phenomenon. More comprehensive assessment of hemoglobin concentration and fluid status are warranted in future studies.

CONCLUSION

Following a 60-minute dive to 4.5 meters in a pool, there is no evidence of increased airway obstruction in healthy divers. A decrease in $D_{L_{CO}}$ was observed in similar magnitude to previous hyperbaric chamber studies that simulated greater depths. This finding provides evidence that the diving-related impairment of diffusion capacity is not depth-dependent. This reduced diffusing capacity could be due to sub-clinical pulmonary edema, immersion diuresis or venous microemboli in the pulmonary circulation or perhaps a combination of these factors. This is the first study of pulmonary function and diving to use divers as their own crossover controls. No changes in either $D_{L_{CO}}$ or spirometry were observed after breathing from SCUBA equipment for 60 minutes without immersion, indicating that the compressed, dry air breathed by divers does not precipitate pulmonary function changes.

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