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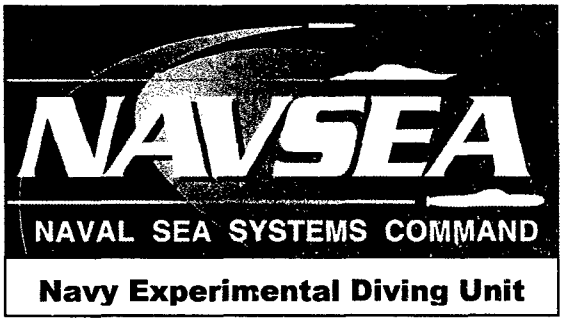
PULMONARY EFFECTS OF SIX-HOUR DIVES: IN-WATER OR DRY CHAMBER EXPOSURE TO AN OXYGEN PARTIAL PRESSURE OF 1.6 ATM



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Pulmonary effects of exposure to hyperbaric oxygen in the water were compared to those for similar exposure in a dry chamber. For six hours, 34 subjects breathed 100% oxygen at 20 feet of seawater, in either the wet pot or a dry chamber of the Navy Experimental Diving Unit Ocean Simulation Facility, with 28 individuals participating in both dives and 12 able to dive only once. Dives were two months apart. Forced flow-volume loops, diffusing capacity measurements, and reported respiratory and other symptoms were recorded before and after the dives. A few mild changes in pulmonary function were seen in seven subjects at any time after dry dives and in four any time after wet dives, with one subject affected after both dives. Dive-related symptoms, mostly mild, were more common than such pulmonary changes in 13 subjects any time during or after dry dives, in 14 during or after wet dives, and in 9 after both dives. Two subjects reported moderate symptoms, one after a dry dive and one after a wet dive. One diver left the water after three hours because of nausea. Except for Draeger ear, which may have been more common after wet dives than after dry exposures (six subjects versus two, $p = 0.078$), there were no differences in effects. We conclude that dry exposures can be used to predict in-water pulmonary oxygen toxicity at the same oxygen partial pressure.

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INTRODUCTION

Experiments in which subjects are exposed to oxygen at elevated pressure can be performed under greater control and with less risk in hyperbaric chambers than in open water. Even in hyperbaric chambers, exposures in a dry environment are safer than those in the water if the risk of seizure provoked by hyperbaric oxygen is significant. Furthermore, dry exposure durations can be extended because sanitary facilities, food, and water can be provided, and thermal comfort is easy to maintain in a dry chamber. Additionally, measurements can be made during the exposures, not just after surfacing. A large body of pulmonary data has been collected from chamber oxygen exposures.[1–10]

Whether respiratory symptoms or changes in pulmonary function after dry exposures are comparable to those after immersed exposures has not been evident. The prevailing wisdom is that central nervous system (CNS) oxygen toxicity is prevalent at lower oxygen partial pressures (Po_2) in the water than in dry exposures. At one atmosphere (atm), oxygen breathing during water immersion has been shown to cause a large acute decrease in vital capacity, while oxygen breathing alone does not.[11] The mechanical factors of water immersion — translocation of blood into the thorax from the abdomen and legs and compression of the chest wall — may contribute to symptoms or to signs of apparent oxygen toxicity. Thus, in one protocol[12] under the task *Pulmonary Oxygen Toxicity after Repeated Diving with Elevated Oxygen Partial Pressures*,[13] we measured pulmonary effects of six hours with 100% O_2 at 20 feet of seawater (fsw) (Po_2 = 1.6 atm) in the water and in the dry chamber to compare the two conditions.

We measured pulmonary function and assessed symptoms before diving, immediately after diving, and for several days after those exposures. The pulmonary function variables we considered were forced vital capacity (FVC), forced expired volume in one second (FEV_1), peak expired flow or maximum forced expired flow (FEF_{max}), mid forced expiratory flow (FEF_{25-75}), and diffusing capacity of the lung for carbon monoxide (D_LCO). The lower limits of normal for pulmonary function variables were defined as decreases from baseline of 2.4 times the coefficient of variation found for the Navy Experimental Diving Unit (NEDU) population — namely, 7.7% for FVC, 8.4% for FEV_1, 16.8% for FEF_{max}, 17.0% for FEF_{25-75}, and 14.2% for D_LCO.[14] We defined decreases of these magnitudes, the lower 95% confidence bands for each variable, as the lower limits of normal.

METHODS

GENERAL

We recruited 34 divers for each arm of the study, with 28 able to participate in both. Subjects dove under one condition, wet or dry, in February 2005, and under the other condition in April 2005. During an exposure, six subjects were in one of the dry chambers of the NEDU Ocean Simulation Facility (OSF) at a pressure of 20 fsw, while

another six were submerged in the OSF wet pot about 3 feet beneath the surface and with the chamber pressure at 17 fsw. The dives were controlled and supervised by qualified NEDU personnel. In both sets of dives, subjects were at rest and breathed dry gas open circuit, from either the MK 20 underwater breathing apparatus in the water or the built-in breathing system (BIBS) mask in the chamber. Both gas delivery systems use demand valves.

EXPERIMENTAL DESIGN AND ANALYSIS

Before the study, subjects had not been diving while breathing air or mixed gas for one week or while breathing oxygen for two weeks. Except for the experimental dives, they refrained from diving throughout the testing period. Each subject's smoking behavior and history of respiratory allergies were noted. General health and use of medications also were recorded during the studies; all subjects were generally in good health, but some had mild symptoms of upper respiratory infection (URI).

Table 1.
Subject characteristics

Median (range)	In-water			Dry chamber		
Age (Yr)	36 (24–44)			35 (24–46)		
Height (in)	70 (66–72)			71 (66–78)		
Weight (lb)	190 (145–225)			190 (145–262)		
Smoking: (#)	*never*	*former*	*current*	*never*	*former*	*current*
	21	11	2	21	10	3
Respiratory allergies (#)	9			10		
Medication (#)						
anti-inflammatory	7			6		
antiallergy	2			2		

The subjects performed pulmonary function tests (PFTs) several days before the test dives, immediately before diving, within 60 minutes of leaving the water, and on working days until the third or fourth day after the test dives. Variables were compared to those from the baseline measurements. If pulmonary function variables on the last of the regular measurement days were below the 95% confidence bands of baseline, pulmonary function was measured again the next day. We used Fisher's Exact Test to assess the incidence of changes in pulmonary function and of symptoms between the two conditions.

Each pulmonary function measurement session involved acquiring three flow-volume loops, with the tests performed and repeatable according to American Thoracic Society standards.[15] FVC, FEV_1, FEF_{max}, and other variables were read from the flow-volume

loops. The sessions also included three single-breath D_LCO measurements made with a 10-second breath hold. The variables used to obtain D_LCO were calculated from the gas concentrations before and after the breath hold. Adjustments were made for carboxyhemoglobin and hemoglobin concentrations,[16] and the samples were chosen to ensure that the analyzer signal was stable when measurements were recorded.[17] However, because hemoglobin and carboxyhemoglobin measurements were questionable on one day when the laboratory was hotter than the upper operating temperature of the carbon monoxide (CO) oximeter, values with and without correction were examined throughout the study.

Divers were questioned about specific symptoms (Table 2) each hour while they were at pressure and at each pulmonary function measurement session. Half of the divers in each arm of the study also provided samples of expired condensate, urine, and blood for *Oxidative Stress and Pulmonary Injury in U.S. Navy Divers*, a study conducted by Captain Charles Vacchiano of the Naval Aerospace Medical Research Laboratory (NAMRL).

Table 2.
Symptoms list

During the dives:	After the dives:
Vision changes	Inspiratory burning
Ringing or roaring in ears	Cough
Nausea	Chest pain or tightness
Tingling or twitching	Shortness of breath
Light-headedness or dizziness	Lowered exercise tolerance
Chest tightness	Unreasonable fatigue
Shortness of breath	Visual complaints
Rapid shallow breathing	Ear problems
Burning on inspiration	
Cough	

EQUIPMENT AND INSTRUMENTATION

The Collins CPL and Collins GS Modular Pulmonary Function Testing System instruments (Ferraris Respiratory; Louisville, CO) were used to measure pulmonary function. The test gas used to measure D_LCO contained 0.3% CO and 0.3% methane. A CO oximeter (Instrumentation Laboratory; Lexington, MA) determined the pretest carboxyhemoglobin and hemoglobin concentrations from a venous blood sample.

PROCEDURES

In each set of dives twelve subjects dove in a single day, six in the wet pot and six in the dry chamber. Six divers entered one side of the chamber complex, were pressed on air to 20 fsw, and then donned BIBS masks and rested in the dry chamber. A second

group of six then entered the other side of the OSF, descended to stand on the platform in the wet pot, and held their masks in their hands with their heads out of the water. Pressed on air to 17 fsw, they then donned their MK 20 masks, submerged themselves, and rested on the platform about 3 feet underwater. The groups of divers were brought back to surface pressure at the end of their 6-hour dives.

While in the chambers, subjects relaxed and watched movies. Subjects were permitted to breathe chamber air and to eat and drink for no more than five minutes per hour. Those in the water were permitted to surface and remove their masks. Those in the dry chamber were permitted to remove their masks and move around. Water temperature was 88 ± 5 °F (31 ± 3 °C). Divers were dressed for comfort. Because the BIBS system does not readily allow humidification of the gas, dry oxygen was supplied to both systems.

After divers surfaced, they were escorted to the laboratory for blood draws, for collection of the other samples for the associated study, for pulmonary function testing, and for recording of symptoms. On the days after diving, the measurements were repeated. Visual refraction was checked before and after diving.

RESULTS

Problems

Some data were lost because the gas analyzer in one of the pulmonary function machines became unstable. After the problem was identified, we discarded diffusing capacity measurements from four subjects at baseline, from four subjects after surfacing, and from four subjects on Day + 1. All these measurements were from subjects who were diving in the wet pot. For the four subjects without valid baseline data, we compared postdive diffusing capacity measurements to those from the last measurement day after diving.

Pulmonary function

Few changes in pulmonary function were seen after either dive, and only some of the changes were accompanied by symptoms. The pulmonary function changes are detailed in Tables 3a (dry) and 3b (wet), where the subjects are arbitrarily identified by a letter code. (The same subject identifiers are used in Tables 4a and 4b.) Immediately after his dry dive, Subject A reported mild inspiratory burning that cleared before the next measurement session, despite the persistent mild deficits in flow-volume parameters. Subject B also experienced mild inspiratory burning after surfacing from his dry dive, as he did after surfacing from his wet dive when he did not have measurable changes in pulmonary function. Subject C had a cough immediately after the dry dive and early on the first day after the dive, but Subjects D and E had no symptoms. Subject F participated despite a mild sore throat and cough before his dry dive, symptoms which persisted until two days after the dive. Subject G had symptoms only

after his wet dive, when he had mild inspiratory burning and chest tightness on surfacing. Subject H reported mild shortness of breath and inspiratory burning on the second day after his wet dive — that is, the day after his measured pulmonary function deficit. Subjects I and J did not report any symptoms, but Subject J thought he had a mild upper respiratory infection at the time of his lowered FEV_1. Subject C did not dive wet, and Subject E had to abort the wet dive for nonpulmonary reasons.

The only subject with pulmonary function changes after both dives, Subject G, showed similar FEF_{25-75} changes two days after diving each time.

Table 3a.
Pulmonary function changes after the dry dives
* if subject also had symptoms
† if subject had PFT changes after the other dive

ID	Dive day	Day + 1	Day + 2	Day + 3
A*	FVC −8.8% FEV_1 −8.5%	FVC −9.4% FEV_1 −11.5% FEF_{25-75} −18.7%	FVC −8.5% FEV_1 −8.5%	
B*	FEF_{max} −17.5%	FEF_{25-75} −19.4%		
C*		FEF_{25-75} −20.1%		
D		D_LCO −16.6%		
E			FVC −12%	
F* *URI*				FEF_{max} −18.4%
G†			FEF_{25-75} −20.5%	

Table 3b.
Pulmonary function changes after the wet dives
* if subject also had symptoms
† if subject had PFT changes after the other dive

ID	Dive day	Day + 1	Day + 2	Day + 3
G*†		FEF_{25-75} −19.7%	FEF_{25-75} −20.0%	
H*		FVC −9.4% FEV_1 −8.7%		
I			FVC −12.8%	
J				FEV_1 −8.6%

Respiratory symptoms

On the dive days, 12 subjects, either dry or wet, had respiratory symptoms not in conjunction with other symptoms of a mild URI (Tables 4a and 4b); eight of them had respiratory symptoms both times, and two who had symptoms after the wet dive did not dive dry. After the dry dive an additional subject developed symptoms later, and two subjects had symptoms probably related to respiratory infections, but 19 had no respiratory symptoms. Similarly, after the wet dives one subject had symptoms probably related to a respiratory infection and two developed respiratory symptoms later, but 19 were free of respiratory symptoms.

Table 4a.
Symptoms during and after the dry dives
Symptoms in **bold** were moderate; those in roman were mild; and those in *italics* were probably not related to the oxygen exposures.
* if subject also had PFT changes
† if subject had symptoms after the wet dive

ID	**Dive day**	**Day + 1**	**Day + 2**	**Day + 3**
A*	Inspiratory burning			
B*†	Inspiratory burning			
C*	Cough	Cough, shortness of breath		
F	*URI – cough*	*URI – cough*	*URI – cough*	
K†	**Inspiratory burning**, cough			
L†	Inspiratory burning	Cough, shortness of breath	cough	
M†	Inspiratory burning, cough	*URI – cough*	*URI – cough*	*URI – cough*
N	*URI – cough*		*URI – cough*	
O†	Chest tightness from hour 4			
P		**Cough**		
Q†	Inspiratory burning from hour 5			
R†	Inspiratory burning			
S†	Inspiratory burning, shortness of breath			
T†	Inspiratory burning	**Low exercise tolerance**	Low exercise tolerance	
U	Chest tightness			

Table 4b.
Symptoms during and after the wet dives

Symptoms in **bold** were moderate; those in roman were mild; and those in *italics* were probably not related to the oxygen exposures.

* if subject also had PFT changes
† if subject had symptoms also after the dry dive
‡ if subject had PFT changes after the dry dive

ID	Dive day	Day + 1	Day + 2	Day + 3
B†‡	Inspiratory burning, cough			
D‡	*URI – cough*	*URI – cough*	*URI – cough*	
G*	Inspiratory burning, chest tightness			
H*			Shortness of breath, cough	
K†	Inspiratory burning			
L†	Inspiratory burning, cough	Inspiratory burning, cough	Cough, low exercise tolerance	Cough, low exercise tolerance
M†	Inspiratory burning	Inspiratory burning, chest tightness		
O†	Inspiratory burning from hour 5			
Q†	Chest tightness, moderate at hour 6, becoming mild			
R†	Inspiratory burning	Inspiratory burning		
S†	Cough, shortness of breath			
T†		Low exercise tolerance		
V	**Cough**, **shortness of breath**, inspiratory burning, chest tightness			
W	Inspiratory burning			
X			Inspiratory burning	
Y	Inspiratory burning from hour 5	Cough, low exercise tolerance	Cough	

Subjects V and X dove only wet.

Two of the subjects who reported symptoms after both dives, Subjects L and R, were suffering symptoms of allergies, and both had taken antihistamine medications. Subject R was also using a nasal steroid spray. Two of the others with symptoms both times, Subjects B and M, and one, Subject G, with pulmonary function changes related to the dive, have seasonal allergies but had no symptoms at the time of the dives. Subjects K, M, and T, all of whom had symptoms after both dives, are former smokers, K and T having quit smoking about five years before the dives, and M about 12 years before these dives. Of the subjects with postdive pulmonary function deficits, C was an occasional smoker and G had last smoked 25 years ago. Subject V had quit smoking about 10 years before this dive.

Other symptoms

One subject terminated his in-water dive because he was nauseated after the third hour. He felt better soon after leaving the chamber. Among the divers in the dry chamber and the other 33 in the water, all at Po_2 = 1.6 atm, no symptoms suggesting CNS oxygen toxicity were seen.

Only two divers who had been in the water and one who had been in the dry chamber complained of reduced exercise tolerance in the days following this single dive. Two subjects reported symptoms of middle ear gas absorption (Draeger ear) after dry diving and six did so after wet diving, proportions that may differ (p = 0.078). We saw at least a 10% increase in hemoglobin concentration in one subject after his dry dive and in two subjects after their wet dives. No subject showed a myopic shift after these single 6-hour dives at 20 fsw.

DISCUSSION

We saw no significant differences in pulmonary effects between dry and in-water exposures to Po_2 = 1.6 atm for six hours, an observation indicating that the effects of the oxygen supersede the effects of water immersion. Because divers breathed from demand regulators, the gas pressure in the mask during inspiration was equal to the surrounding pressure at the depth of the face. For divers seated in the water, hydrostatic loading was identical to that of head-out water immersion, while divers seated in the dry chamber had no hydrostatic gradient around their chests. However, many divers chose to recline on the platform underwater, a position greatly reducing the hydrostatic gradients. Furthermore, major effects of head-out water immersion — vascular engorgement of lung tissue and changes in the mechanics of breathing — are highly transient, and others — closure of dependent airways and atelectasis in the lung bases — can be cleared rapidly in healthy, uninjured lungs by deep sighs or vital capacity maneuvers. Since we had from 25 minutes to two hours between surfacing and measuring, and since we required three consistent vital capacity maneuvers before we concluded a test, short-term effects of the immersion were eliminated, with only effects of lung or airway injury remaining. Immersion does not appear to have caused or exacerbated lung injury in these exposures.

Pulmonary Function Changes

Few subjects experienced pulmonary function changes overall (Tables 3a, 3b). No subject had deficits in pulmonary function on surfacing from a dive in the water, while two subjects did after surfacing from dry dives. Four divers showed pulmonary function change at any time after the wet dives, in contrast to seven subjects, one of whom reported having a URI after the dry dives. The slight differences in incidence of pulmonary function change may have been a result of humidity; although both gas streams were dry, the divers in the water would have had incidental humidification if they had had water in their masks.

The only change in D_LCO seen in this study, that in Subject D, was asymptomatic and was evident a full day after his dry dive. Some of the four subjects for whom we are missing dive day D_LCO values or the other four missing D_LCO values for the first day after diving dry may have had values that were reduced from baseline, but we have data without significant change from 33 subjects dry and from 26 wet. In general, D_LCO is reduced temporarily if the diffusion path from alveolar gas to blood is increased by edema or if the surface area for diffusion is decreased by redistribution of pulmonary ventilation away from blood flow or of blood flow away from the lung. Pulmonary edema is expected to generate cough and chest tightness, but perhaps symptoms were so slight that the subject failed to notice them. The measurement was long enough after the dive that transient effects were eliminated. However, the decrement seems real, in that it is evident in both the hemoglobin-corrected and the uncorrected values and was not associated with a change in calculated alveolar volume. Also, Subject D showed a D_LCO decrease of 13% from baseline one day after his wet dive, a value just inside the normal variation.

All other pulmonary function changes were in flow-volume parameters. Acute decreases in vital capacity and FEV_1 could be caused by edema, pleural effusion, small airway closure, redistribution of blood to the lungs, or atelectasis. Since Subject A, who had reductions in FVC and FEV_1 after his dry dive, also reported inspiratory burning after the dive and showed flow-volume deficits for three days, redistribution of blood is not a likely cause. Because the deficits increased on the first day after diving and the FEF_{25-75} had also decreased beyond its normal variability that day, we can postulate that some alveolar or small airway injury followed by inflammation required time to heal. A similar process may have been active in Subject H after his wet dive, but with injury that was below detectable limits on the day of the dive.

Decreases in FEF_{max} (in Subjects B and F) were likely caused by increases in laryngeal resistance; peak expiratory flow is limited by the rate of expiratory muscle shortening[18] and the large airway resistance, about 45% of which is at the larynx during mouth breathing.[19] After his wet dive, Subject B reported inspiratory burning and cough, while after his dry dive Subject F had a cough that he attributed to a URI that may have masked symptoms caused by the diving exposure.

Mid forced expiratory flow, the average flow from 25% volume expired to 75% volume expired, indicates the conditions of small airways. For most of the descending portion of a forced flow-volume curve, the flow is determined by the elastic recoil of the inflated lung and the diameters of the airways upstream of the point where the pressure inside the airways equals the pressure outside of them, the equal pressure point (EPP).[20] Mid forced expiratory flow thus decreases acutely if small airway resistance increases acutely — for example, because of fluid cuffing following oxygen injury.

The slope of the descending limb of the flow-volume curve is determined by lung elasticity divided by the resistance of the small airways upstream of the EPP.[20] A concave descending limb can be interpreted to mean either that small airway resistance is increasing as volume decreases or that different lung regions with varying mechanical properties are emptying sequentially. Some of our subjects developed concavities in flow-volume curves. In those subjects where small airway resistance increased sometime after diving, the changes developed one or more days after the diver surfaced. The resistance increase appears to comprise a secondary effect of the oxygen injury.

Respiratory Symptoms

Some individuals seem more likely than others to react to oxygen exposure; 8 of the 28 subjects who dove under both conditions had respiratory symptoms both times, and 8 had symptoms neither time, not even symptoms probably related to URIs. Indeed, half of the subjects with respiratory symptoms after both dives also had respiratory allergies, and half of them were smokers or former smokers. Airway reactivity may be a factor in the development of oxygen symptoms. However, four of the ten subjects who reported that they had allergies did not have symptoms after either dive.

Other symptoms

Model predictions of CNS events during or after in-water exposure to 100% oxygen at 20 fsw are based on data that stop at 240 minutes.[21] Extrapolation with that model gives the probability of 0.986 that a 6-hour dive at Po_2 = 1.6 atm will be free of dive-stopping CNS events. In this series, 33 subjects spent six hours in the water at that oxygen partial pressure without any symptoms of CNS disturbance, and one subject experienced dive-stopping nausea that may or may not have been oxygen related. These results are not at odds with the model prediction.

Reduced exercise tolerance and unreasonable fatigue are complaints reported frequently after repeated 6-hour dives with Po_2 = 1.35 atm, and whether the repeated immersion, the repeated oxygen exposure, or both the immersion and the oxygen are responsible is unclear. In this series of single dives, one subject reported reduced exercise tolerance for two days after a single 6-hour dry dive. In that case, at least, immersion was not a factor.

Draeger ear may be slightly more prevalent after in-water oxygen breathing than after oxygen breathing in the dry exposures. Fluid shifts during the dive may leave divers with nasal congestion that impedes ear clearing for some time after surfacing.

Significance of blood analyzer problems

Carbon monoxide present in the blood reduces the driving pressure for the transfer of CO during the breath holding maneuver. The correction for carboxyhemoglobin takes the carbon monoxide partial pressure from the hemoglobin–carbon monoxide dissociation curve and then subtracts that from the apparent driving pressure for gas transfer to yield the real driving pressure.

With the measurement units in which D_LCO is to be expressed,

$$D_LCO_{raw} = [V_A / (t\,(P_{tot} - P_{water})] \cdot \mathit{ln}\,\{[F_ACO_{initial} - P_{CO}/P_{tot}] / [F_ACO_{final} - P_{CO}/P_{tot}]\},$$

where D_LCO_{raw} is uncorrected for hemoglobin concentration, V_A is alveolar volume, t is breath hold time, P_{tot} is total pressure, P_{water} is partial pressure of water in the alveolar gas, *ln* is the natural logarithm, F_ACO is the alveolar mole fraction of CO, P_{CO} is the partial pressure of CO in the pulmonary capillary blood (assumed to be constant and the same as that in the blood sample drawn), and "initial" and "final" refer to the start and end of the breath hold, respectively. A non-zero P_{CO} increases the logarithmic term to give a higher reading of D_LCO than would have been obtained with the same measured quantities and no CO present in the blood.

Increased hemoglobin concentration increases D_LCO by providing more of a "sink" for carbon monoxide even if the gas-blood contact area and the pathway for diffusion through the membrane are unchanged. Because the contact area and diffusing pathway are the factors we wanted to monitor, we adjusted the diffusing capacity figures for changes in hemoglobin concentration.

For men, in whom a hemoglobin concentration of 14.6 g/dL is considered standard,

$$D_LCO_{Hb} = D_LCO_{raw} \cdot [(10.22 + Hb) / 1.7Hb],$$

where D_LCO_{Hb} is the diffusing capacity normalized to Hb = 14.6 g/dL, and Hb is hemoglobin concentration expressed in g/dL.[16] D_LCO_{Hb} is used as D_LCO in this report.

The elevated temperature in the laboratory caused the CO oximeter to give abnormally high readings of both hemoglobin and carboxyhemoglobin from the standard solutions. We therefore assume that the readings from blood samples were also biased high. Using an incorrectly high hemoglobin concentration decreases the reported diffusing capacity, while using an incorrectly high carboxyhemoglobin value increases the reported value. It is possible that the errors at least partially cancelled each other. In any case, the values uncorrected for hemoglobin and carboxyhemoglobin were examined in addition to those for which the corrections had been applied.

CONCLUSIONS

Pulmonary function changes after 6-hour exposures to Po_2 = 1.6 atm were infrequent and mild: in seven subjects at any time after dry dives and in four subjects any time after wet dives. Symptoms, more common than pulmonary function changes, were reported by 12 of 34 subjects on the days of diving either wet or dry, with one subject reporting moderate symptoms after each exposure and all others reporting mild symptoms. All toxic effects resolved spontaneously.

We saw one possible incidence of CNS toxicity — nausea in the water — and no definite CNS toxicity after 34 subject exposures in the dry chamber and 34 subject exposures in the wet pot (among a total of 46 individuals). This outcome was consistent with predictions made by extrapolation using an existing CNS risk model.[21]

A 6-hour exposure to Po_2 = 1.6 atm has similar pulmonary effects in the water or in a dry chamber. Thus, results obtained in dry hyperbaric chambers can be pooled with data collected in the water to determine pulmonary oxygen toxicity limits. Draeger ear may be more common after in-water exposures than after dry exposures.

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