PULMONARY EFFECTS OF U.S. NAVY TREATMENT TABLE 6 HYPERBARIC EXPOSURE

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<td>TT6, Pulmonary oxygen toxicity, FVC, D.C.O, FEV1, diffusing capacity, recompression treatment, MK 25, LAR V</td>
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</table>

19. ABSTRACT: Eighteen healthy subjects underwent pulmonary function measurements after single experimental Treatment Table 6 (TT6) recompression exposures in which oxygen was delivered from MK 25 rebreather underwater breathing apparatus. Most subjects showed no pulmonary function changes, but three had mildly decreased flow-volume parameters on surfacing and one developed changes later, for a 22% incidence (95% binomial confidence interval 6 to 48%). All changes resolved spontaneously over the next several days. This incidence of decreased pulmonary function after rebreather TT6 may underestimate slightly the incidence after conventional oxygen delivery, because inspired oxygen fraction is lower and gas is more humid with the MK 25 than with conventional systems. Nonetheless, the pulmonary insult from a TT6 is minor in comparison to the benefit to be gained if recompression therapy is indicated.

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INTRODUCTION

Although the U.S. Navy Diving Manual states that pulmonary oxygen toxicity is unlikely to develop during a single Treatment Table 6 (TT6) exposure, the oxygen dose of the TT6 depicted in Fig. 1 is substantial.

### Treatment Table 6

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 10 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 22-5 A 11).
5. Table 6 can be lengthened up to 2 additional 25-minute periods at 60 feet or 2 additional 75-minute periods at 30 feet or 15 minutes on air and 60 minutes on oxygen or both.
6. Tender breathes 100 percent O₂ during the last 30 minutes at 60 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O₂ breathing at 30 feet is increased to 60 minutes. If the tender had a hyperbaric exposure within the past 12 hours an additional 60-minute O₂ period is taken at 30 feet.

The first periods on 100% oxygen at 60 feet of seawater (fsw) (green in the diagram) provide an oxygen partial pressure (PO₂) of 2.8 atmospheres (atm), while the air breaks (blue) have PO₂ = 0.6 atm. The average PO₂ during the ascent to 30 fsw is 2.4 atm. At 30 fsw, PO₂ with air is 0.4 atm, and with 100% oxygen PO₂ is 1.9 atm. The average PO₂ is 0.95 atm during the final ascent.

Although the potential benefits of the treatment outweigh the risks in anyone for whom a TT6 is indicated, adverse pulmonary effects can be expected in some patients who breathe this much oxygen. Because measurement of pulmonary function would delay treatment, the effects have not been documented in patients. However, an opportunity to quantify the pulmonary effects was presented during testing of the MK 25 rebreather as an oxygen supply for treatment. Because healthy U.S. Navy divers underwent TT6 exposures, pulmonary function could be measured before and after exposure without undue risk.
We recorded forced flow-volume loops to determine forced vital capacity (FVC), forced expired volume in one second (FEV₁), peak expired flow or maximum forced expired flow (FEFₘₐₓ), and average forced expiratory flow from 25% to 75% of expired volume (FEF₂₅⁻₇₅). Diffusing capacity of the lung for carbon monoxide (D\textsubscript{L}CO) was determined from single breath tests.

**METHODS**

**GENERAL**

Eighteen divers from Navy Experimental Diving Unit (NEDU) participated in TT6 dive profiles in the Ocean Simulation Facility. Divers used the MK 25 underwater breathing apparatus (UBA) as their oxygen sources. Details of the dives are given in a separate report.\textsuperscript{2}

Before ("baseline") and for at least two days following (longer if we saw changes), we measured flow-volume loops and diffusing capacity for carbon monoxide for 17 divers once a day. We considered averages from three consistent maneuvers. D\textsubscript{L}CO values from 10 s breath holds were adjusted for carboxyhemoglobin and hemoglobin concentrations,\textsuperscript{3} and samples were chosen to ensure that the analyzer signal was stable when measurements were recorded.\textsuperscript{4}

**EXPERIMENTAL DESIGN AND ANALYSIS**

Each subject's posttreatment values were compared to his baseline values. Variables were considered to have decreased if they were below the lower 95% confidence bands previously found for the NEDU population: namely, 7.7% for FVC, 8.4% for FEV₁, 16.8% for FEF\textsubscript{ₘₐₓ}, 17.0% for FEF₂₅⁻₇₅, and 14.2% for D\textsubscript{L}CO.\textsuperscript{5}

Confidence in estimates of the incidence of changes in pulmonary function with $\alpha = 0.05$ (95% confidence in the proportion) was obtained from the binomial distribution.

**EQUIPMENT AND INSTRUMENTATION**

The Collins CPL or GS Pulmonary Function Testing System instruments (Collins Ferraris Respiratory; Louisville, CO) were used to measure pulmonary function. The test gas used to measure D\textsubscript{L}CO contained 0.3% CO and 0.3% methane. A CO oximeter (Instrumentation Laboratory; Lexington, MA) determined carboxyhemoglobin and hemoglobin concentrations from a venous blood sample.
RESULTS

Immediately after surfacing, three subjects demonstrated reductions in several flow-volume parameters, with changes persisting for one to three days. A fourth subject showed a deficit in FEF\textsubscript{25-75} on the second postexposure day (Table 1, Figure 1). The incidence in four of eighteen subjects, or 22%, has 95% confidence bands from 6% to 48%.

An additional subject had greatly reduced FEF\textsubscript{max} after the TT6 exposure (Table 1, Figure 1), a reduction that was considered to be spurious (see DISCUSSION).

Table 1.
Decreases in Pulmonary Function when Outside Normal Variation

<table>
<thead>
<tr>
<th>Subject</th>
<th>Day</th>
<th>%ΔFVC</th>
<th>%ΔFEV\textsubscript{1}</th>
<th>%ΔFEF\textsubscript{max}</th>
<th>%ΔFEF\textsubscript{25-75}</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Exposure</td>
<td>-9.2%</td>
<td>-15.5%</td>
<td>-33.8%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+1</td>
<td>-10.0%</td>
<td>-12.7%</td>
<td>-24.6%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+2</td>
<td></td>
<td>-8.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Exposure</td>
<td>-11.7%</td>
<td>-12.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>+1</td>
<td>-8.6%</td>
<td>-9.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Exposure</td>
<td>-7.9%</td>
<td>-11.1%</td>
<td>-20.6%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+2</td>
<td>-11.8%</td>
<td>-17.6%</td>
<td>-24.0%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+3</td>
<td></td>
<td>-18.4%</td>
<td></td>
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<tr>
<td></td>
<td>+4</td>
<td>-9.7%</td>
<td>-17.5%</td>
<td>-25.8%</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>+2</td>
<td></td>
<td></td>
<td></td>
<td>-19.8%</td>
</tr>
<tr>
<td>5</td>
<td>Exposure</td>
<td></td>
<td></td>
<td></td>
<td>-38.7%</td>
</tr>
</tbody>
</table>

No subject showed a decrease in D\textsubscript{L}CO (Figure 2). Average changes in all variables remained within normal limits (Table 2).

Table 2.
Average changes in pulmonary function variables

<table>
<thead>
<tr>
<th>Day</th>
<th>%ΔFVC</th>
<th>%ΔFEV\textsubscript{1}</th>
<th>%ΔFEF\textsubscript{max}</th>
<th>%ΔFEF\textsubscript{25-75}</th>
<th>%ΔD\textsubscript{L}CO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure</td>
<td>-2.8%</td>
<td>-3.5%</td>
<td>-3.7%</td>
<td>-4.6%</td>
<td>2.7%</td>
</tr>
<tr>
<td>+1</td>
<td>-2.5%</td>
<td>-3.2%</td>
<td>1.4%</td>
<td>-5.5%</td>
<td>4.6%</td>
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<tr>
<td>+2</td>
<td>-1.1%</td>
<td>-1.6%</td>
<td>1.0%</td>
<td>-4.4%</td>
<td>3.1%</td>
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</table>
Figure 2. Changes from baseline, flow-volume parameters. Heavy horizontal lines indicate limits of variability. Each symbol represents a value for one subject, the comparison of the average of three measurements to the average of three at baseline.
Peak expiratory flow is strongly influenced by effort. Because poor effort is a more likely cause of the 39% reduction in $\text{FEF}_{\text{max}}$ (Entry 5 in Table 1) than is oxygen toxicity, we disregarded that value. No effort-independent flow-volume changes were measured, and the large decrement had disappeared by the following day. The inclusion of that value would increase the incidence of pulmonary oxygen toxicity from 22% to 28%.

If these healthy subjects are representative of the population, between 6% and 48% of patients undergoing U.S. Navy TT6 recompression therapy will experience decreased pulmonary function during the three days following such therapy. From these data it is impossible to predict the pulmonary effects of repeated recompression treatment, but the changes in pulmonary function after one exposure were less than twice the limits of normal variability; had they been participants in experimental series of repeated oxygen dives at NEDU, they would have been cleared to enter the water. Only one subject reported symptoms, and all pulmonary function deficits resolved spontaneously in a few days following treatment.

The effects measured when oxygen was delivered through a rebreather UBA may be less severe than those after standard oxygen delivery with a built-in breathing system (BIBS) mask, because the inspired oxygen during the experimental exposures ranged from 71% to 94%, while a BIBS mask provides close to 100% oxygen. Furthermore, the gas in a rebreather is humid while that in a BIBS may be dry, and breathing dry gas can reduce some indices of pulmonary function. Although inspired gas temperature and the work of breathing with the MK 25 may cause pulmonary insult that is not experienced
with the BIBS mask, none of these subjects reported that the gas was uncomfortably warm, and only two divers reported that the work of breathing the MK 25 was noticeable. 

**CONCLUSIONS**

Although even a single TT6 does cause pulmonary changes in some subjects, pulmonary oxygen toxicity is a minor risk for the benefit possible when recompression therapy is indicated. The pulmonary effects did not impact in these healthy subjects on the days following the exposure.
REFERENCES


