Exercise tolerance at 4 and 6 ATA

N. R. ANTHONISEN, G. UTZ, M. H. KRYGER, and J. S. URBANETTI

Respiratory Division, Department of Medicine,
Royal Victoria Hospital, McGill University, Montreal

Anthonisen, N. R., G. Utz, M. H. Kryger, and J. S. Urbanetti. 1976. Exercise tolerance at 4 and 6 ATA. Undersea Biomed. Res. 3(2): 95-102. —Seven normal male subjects performed 5-min bicycle exercise ranging from 50-100% maximum oxygen uptake at 4 ATA and three were also studied at 6 ATA. At all pressures, the subjects breathed 0.2 ATA O₂ plus nitrogen. All subjects were able to perform maximum work at all pressures. No pressure-dependent variations in heart rate, O₂ uptake, or CO₂ output were noted. At both 4 and 6 ATA, ventilation was decreased at exercise levels greater than 80% maximum O₂ uptake. The magnitude of the decrease was not great, however, and signified only minor CO₂ retention. In some instances exercise ventilation closely approached the 15-s maximum breathing capacity and these subjects noted severe dyspnea, possibly due to dynamic compression of large airways. In three subjects, respiratory frequency was measured as well as minute ventilation; this relationship did not change with depth. Subjects performing heavy exercise at 6 ATA noted disturbances of consciousness, presumably due to N₂ narcosis.

exercise performance
ventilation
gas density

From numerous studies of exercise tolerance at high pressure, some consistent patterns have emerged. Heart rate has been lower and O₂ consumption higher at depth than at the same exercise level at the surface (Salzana, Rausch, and Saltzman 1970; Salzana, Overfield, Rausch, Saltzman, Kylistra, Kelley, and Summitt 1971; Bradley, Anthonisen, Vorosmarti, and Linaweaver 1971). However, these studies were conducted in oxyhelium atmospheres, which introduce depth-related thermal variables. In addition, the inspired-O₂ tension has often increased with depth. Some studies have shown clear limitation of exercise ventilation consequent to increased gas density (Wood and Bryan 1971; Miller, Wangensteen, and Lanphier 1971), but these experiments involved few subjects and changing levels of inspired O₂. It therefore seemed appropriate to study exercise responses up to and including maximal exercise, at constant P[subscript]O₂ in a larger group of subjects.

MATERIALS AND METHODS

Seven young male subjects were studied (Table 1). All were physically fit but none were other than casual divers. All measurements were made on a mechanically braked bicycle ergometer inside a hyperbaric chamber. Because the ergometer fit the chamber rather tightly it was impossible to study tall subjects (Table 1). Heart rate was measured from an ECG.
TABLE 1

Physical characteristics of the subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>$\dot{V}_{O_2}$ max*</th>
<th>MBC†</th>
</tr>
</thead>
<tbody>
<tr>
<td>N.A.</td>
<td>40</td>
<td>175</td>
<td>70</td>
<td>3.60</td>
<td>200</td>
</tr>
<tr>
<td>M.D.</td>
<td>30</td>
<td>170</td>
<td>66</td>
<td>3.24</td>
<td>165</td>
</tr>
<tr>
<td>B.E.</td>
<td>31</td>
<td>184</td>
<td>84</td>
<td>3.60</td>
<td>205</td>
</tr>
<tr>
<td>O.H.</td>
<td>23</td>
<td>170</td>
<td>60</td>
<td>3.00</td>
<td>160</td>
</tr>
<tr>
<td>J.L.</td>
<td>26</td>
<td>172</td>
<td>80</td>
<td>3.90</td>
<td>—</td>
</tr>
<tr>
<td>G.U.</td>
<td>33</td>
<td>178</td>
<td>69</td>
<td>3.20</td>
<td>190</td>
</tr>
<tr>
<td>C.Z.</td>
<td>32</td>
<td>182</td>
<td>82</td>
<td>3.40</td>
<td>227</td>
</tr>
</tbody>
</table>

* Maximum $O_2$ uptake liters/min (STPD)
† Maximum breathing capacity liters/min (BTPS)

tracing of standard lead II, which was recorded outside the chamber. Subjects breathed through a low-resistance valve (Otis-McKerrow) with a dead space of 0.1 liter, connected by tubing (3.75-cm internal diameter) to meterologic balloons for inspired and expired gas. All exercise periods consisted of 5 min at a constant load. During the last minute, heart rate was measured, expired gas was collected, and in some instances respiratory frequency was counted. Expired minute volume was measured with a dry gas meter, and expired and inspired $P_{O_2}$ and $P_{CO_2}$ measured with $O_2$ and $CO_2$ electrodes (Instrumentation Laboratories), which were calibrated immediately before and after each measurement. From these values, $O_2$ uptake ($\dot{V}_{O_2}$), $CO_2$ output ($\dot{V}_{CO_2}$) and respiratory quotient were calculated. Analysis of volume and composition of expired gas was carried out immediately after collection, without change in environmental pressure.

All subjects were studied at 1 and 4 ATA and three were studied at 6 ATA. At 1 ATA subjects breathed room air and performed at 3-4 levels of exercise ranging from approximately 50% to 100% of maximum oxygen uptake. Two to four exercise runs were made per day, the lowest level always being first, the highest level last. Each exercise level was performed 3-5 times at 1 ATA to establish the range of variability of these control measurements. Maximum $O_2$ uptake ($\dot{V}_{O_2}$ max) was determined in each subject as the level of $\dot{V}_{O_2}$ at which $V_{O_2}$ did not increase as work increased. At 4 ATA, subjects breathed 5% $O_2$. Two 5-min exercise periods were accomplished on each exposure to 4 ATA, the most strenuous being last. Each subject performed at least once at each work load achieved at 1 ATA. Three subjects were studied at 6 ATA, breathing 3.5% $O_2$. Only one 5-min exercise period was attempted during each exposure to 6 ATA and most of these were at high levels (equivalent to 80-100% $\dot{V}_{O_2}$ max). During exercise at depth the subjects wore a loosely fitting plastic bag over their heads, which also encased the breathing valve. A flow of gas with the same composition as the inspiirate was maintained through this bag. In some of the subjects the bag was used during control runs at 1 ATA; it had no influence in the measurements we made. In addition, in six of the seven subjects the 15-s maximum breathing capacity (MBC) was measured twice at each atmospheric pressure.
EXERCISE TOLERANCE AT 4 AND 6 ATA

All decompressions from 4 and 6 ATA were accomplished without event; decompression was according to the U.S. Navy tables (1963), the 120-ft table for 4-ATA exposures and the 190-ft table for the 6-ATA exposures.

RESULTS

Inspired $P_{O_2}$ was 150 ± 15 mm Hg during all experiments. All subjects were capable of performing maximum steady-state exercise at both 4 and 6 ATA. Some subjects with maximum exercise ventilation approximating 100 liters/min experienced severe dyspnea at these exercise levels, two at 4 and one at 6 ATA; this dyspnea persisted for 3-5 min after exercise ceased. Two subjects had difficulty maintaining consciousness during heavy exercise at 6 ATA. In some subjects mixed expired-gas analysis during heavy work at depth gave unsatisfactory results in that calculated $O_2$ uptakes were much lower than those resulting from similar exercise at ATA. This was judged to be due to leaks in and around the mouthpiece and was rectified in all but one subject by surrounding the head and mouthpiece with inspired gas.

Typical results for subject O.H. are shown in Fig. 1, which plots oxygen uptake, heart rate, and ventilation against work load. Only the lowest work load at which maximum $O_2$ uptake was observed is shown in Fig. 1; higher work loads were not regularly attempted at depth. It can be seen that neither $V_{O_2}$ nor heart rate was affected by depth. At maximal work (100% $V_{O_2}$ max) ventilation at depth was slightly reduced.

These findings typified the response of other subjects. $V_{O_2}$ and $V_{CO_2}$ at a given work load were not changed by depth. Similarly (Fig. 2) heart rate at a given work load was the same at all three pressures. Ventilation, on the other hand, did decrease at high work levels at both pressures (Fig. 3). Work which demanded 80 liters/min ventilation at the surface and which utilized more than 80% $V_{O_2}$ max was uniformly associated with decreased ventilation. At $V_{O_2}$ max, $V_{E}$ averaged 109 liters/min at 1 ATA, and 89 liters/min at 4 ATA. Values at 6 ATA were not different from those at 4 ATA in the three subjects studied (Fig. 3).

Complete data for respiratory frequency and tidal volume were acquired in only three subjects. Figure 4 shows one of these; for a given ventilation, frequency and, therefore, tidal volume was unaffected by depth.

DISCUSSION

These experiments gave results which differed from some in the literature. We failed to observed an increase in $V_{O_2}$ at a given work level with depth. This may have been because our $V_{O_2}$ (Fig. 1) showed somewhat more scatter than those of other observers, notably those of Salzano et al. (1970). Though some studies showing increased $V_{O_2}$ with depth have involved depth-dependent increases of $P_{O_2}$, the latter was not large enough to influence the former (Salzano et al. 1970). These and other workers (Bradley et al. 1971) have attributed increased $V_{O_2}$ with depth to increased $O_2$ usage by respiratory muscles. However, in studies of the influence of external resistances on steady-state exercise (Ceretelli, Sikand, and Farhi 1969; Denadis and Anthonisen 1973), no increase in $V_{O_2}$ was observed with external resistances that almost certainly increased respiratory muscle work to a greater extent than depth. It may be, therefore, that the increase in $V_{O_2}$ observed by other workers was related to factors other than respiratory work, possibly including metabolic thermogenesis induced by high-pressure helium atmospheres.
Fig. 1. Exercise responses in O.H. Abscissa: Work load in kg·m/min. Ordinates: top, ventilation in liters/min (BTPS); middle, heart rate in beats/min; bottom, O₂ uptake in liters/min (STPD). Open circles represent data gathered at 1 ATA, closed circles 4 ATA, and x's 6 ATA.

Our failure to observe a decrease in heart rate for a given work load at depth may also have been related to the different thermal environment our subjects experienced. Further, most studies that have shown significant bradycardia at depth have also utilized higher P[O₂] at depth than at the surface and it was recognized (Salzano et al. 1970; Salzano et al. 1971) that increased P[O₂] might depress heart rate. In any event, the present results indicated that exercise bradycardia was not an effect of pressure per se, at least over the range of pressures we employed.
Fig. 2. Heart rate (beats/min) at depth compared to that at the surface (abscissa) at the same work load. Open circles are data gathered at 4 ATA, closed circles data gathered at 6 ATA. Data from all subjects are included.

Fig. 3. Ventilation (liters/min) at depth compared with that at the surface at the same work load. Open symbols (○, △) are data gathered at 4 ATA; closed symbols (●, ▲) are data gathered at 6 ATA. Circles (●, ○) are data gathered at work loads demanding less than 80% VO₂ max, triangles (▲, △) are data gathered at work loads demanding more than 80% VO₂ max. Data from all subjects are included.
Fig. 4. Effect of depth on breathing frequency in one subject. Frequency is measured in breaths/min, ventilation in liters/min BTPS. Open circles represent (○) data gathered at 1 ATA, closed circles (●) data at 4 ATA, and x's data at 6 ATA.

During heavy work, exercise ventilation decreased. Our observations were similar to those of Wood and Bryan (in press) whose two subjects had a greater limitation of exercise performance at 4 and 7 ATA than our subjects at 4 and 6 ATA. This was probably because their subjects had higher levels of ventilation for a given work load or O₂ uptake, presumably because they were less fit. The decreases in ventilation we observed probably resulted in increases of arterial CO₂ of only 5-6 mm Hg increases of non clinical significance. Wood and Bryan (1971, in press) showed that at high levels of exercise at 4 ATA, ventilation was limited by dynamic compression of airways, an event which did not occur at the surface. They reported that severe choking dyspnea was associated with dynamic compression and flow limitation; two of our subjects experienced this at 4 ATA and another at 6 ATA. The importance of dynamic compression of airways at depth may be inferred from the relationship between maximal exercise ventilation and the 15-s MBC (Fig. 5). At the surface, maximal exercise was associated with ventilations which were 50-75% MBC.

The MBC is limited by dynamic airway compression, although this does not occur at the lower ventilations associated with maximal exercise (Wood and Bryan in press; Olafsson and Hyatt 1969). At depth, however, maximum exercise ventilation may approach the MBC closely and both are limited by dynamic airway compression. The subjects in our series who experienced choking dyspnea were all breathing at least 95% of their MBC. It is not clear how dynamic compression induces severe dyspnea but it may be triggered by receptors in major airways. This was suggested by the fact that dyspnea at depth was of a different quality and more severe than that experienced as a result of external resistance (Demedts and Anthonisen 1973), though ventilatory limitation in the latter case was more severe. In
Fig. 5. Maximum exercise ventilation (MVE) as a percentage of 15-s maximum breathing capacity (MBC) as a function of depth (in ATA). Lines join data points derived in each of six subjects.

some of our subjects maximum exercise ventilation did not approximate the MBC at depth. Since all subjects hypoventilated at depth, those subjects who did not reach their MBC must have chosen to retain CO₂ rather than attain ventilations involving dynamic airway compression.

Our finding that depth did not affect breathing pattern is at variance with most other studies (Bradley et al. 1971; Miller et al. 1971; Salzano, et al. 1971). However, all have observed that changes in ventilatory pattern were not well-related to gas density. Further, previous experiments have used experienced divers as subjects, and they tend to employ relatively low breathing frequencies on the surface (Lally, Zechman, and Tracy 1974). Our results in naive subjects might indicate that changes in breathing pattern were to some extent learned. Also, during exercise on a bicycle ergometer breathing frequency tends to have a fixed relationship to leg motion; by forcing our subjects to pedal at 50 strokes/min, we may have obliterated adaptive changes of breathing frequency.

During exercise at 6 ATA, two of our subjects had difficulty maintaining consciousness, and the third felt “woozy.” Since none of them demonstrated enough hypoventilation to make CO₂ narcosis a reasonable possibility, we concluded that these symptoms were probably due to N₂ narcosis. When subjects were breathing N₂ at 5.8 ATA they were working hard and had mild CO₂ retention, both of which may potentiate N₂ narcosis (Bennett 1969).

These studies indicate that it is unlikely that respiratory limitations will be important in deep oxyhelium diving. Our subjects were able to perform maximum work at 6 ATA, a gas density equivalent to 0.4 ATA O₂ and 40 ATA helium.
This work was supported by grants from the Medical Research Council and Defence Research Board of Canada.

Received for publication September 1975; revised manuscript received October 1975.

Anthonisen, N. R., G. Utz, M. H. Kryger, and J. S. Urbanetti. 1976. La tolerance pour l'exercice à 4 et 6 ATA. Undersea Biomed. Res. 3(2): 95-102. —Sept sujets mâles normaux ont subi un test à bicyclette ergométrique avec une consommation d’oxygène de 50-100% à 4 ATA; trois des sujets ont été étudiés aussi à 6 ATA. A toutes les pressions, les sujets respiraient de l’oxygène à 0,2 ATA; le reste du mélange était de l’azote. Tous les sujets ont pu accomplir le travail maximum à toutes les pressions. Aucune variation de la fréquence cardiaque, de la consommation d’oxygène, ou de l’élimination du CO₂ dépendante de la pression n’a été observée. A 4 et 6 ATA, la ventilation se trouvait diminuée à des valeurs d’exercice audessus de 80% de la consommation maximale de l’oxygène. La chute était modérée, et ne signifiait qu’une utilisation légère de CO₂. Dans certains cas, la ventilation à l’exercice s’approchait du MBC de 15 s.; une dyspnée sèvere, due peut-être à une compression dynamique des grandes voies aériennes, a été notée chez ces sujets. Chez trois sujets, la fréquence respiratoire a été déterminée, aussi bien que la ventilation-minute; ce rapport n’a pas varié avec la profondeur. Les sujets qui ont accompli un exercice difficile à 6 ATA ont remarqué des troubles de la conscience, dus sans doute à l’intoxication à l’azote.

l’accomplissement de l’exercice
ventilation
densité d’un gaz

REFERENCES


