Dyspnea in divers at 49.5 ATA: mechanical, not chemical in origin


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Spaur, W. H., L. W. Raymond, M. M. Knott, J. C. Crothers, W. R. Braithwaite, E. D. Thalmann, and D. F. Uddin. 1977. Dyspnea in divers at 49.5 ATA: mechanical, not chemical in origin. Undersea Biomed. Res. 4(2): 183–198.—Pulmonary function was studied in six divers living in a hyperbaric chamber at a pressure nearly fifty times normal (49.5 atmospheres absolute (ATA), equivalent to 488 m or 1600 ft seawater (fsw)). As expected, ventilatory function was reduced. At 49.5 ATA, maximum voluntary ventilation (MVV) was 45% less than the control value. Instantaneous rates of gas flow during forced expiration were similarly reduced, especially those flow rates measured high in the lung volume. These reductions occurred despite an apparent increase in functional residual capacity (FRC) and the use of transpulmonary pressures considerably greater than those exerted during the same maneuvers at normal (sea-level) pressure. During underwater work at 49.5 ATA, the divers rapidly became exhausted at moderate levels of oxygen consumption (1.9 liters/min), showing severe dyspnea and impending syncope. These symptoms were not due to retention of carbon dioxide, nor to hemodynamic or metabolic causes. Thus, dense gas breathing, like asthma, exemplifies a state in which severe dyspnea may occur with normal or low arterial carbon dioxide and normal oxygen transport. The physiological adjustments the divers employed were similar to those seen in acute asthma, imposing an elastic load in addition to the flow-resistive work of breathing a gas mixture eight times as dense as air. Although men can do moderate work under conditions similar to those of this experiment, they will have only a limited physiological reserve available to meet the possibilities of emergencies or respiratory infections.

Dyspnea in divers at 49.5 ATA: mechanical, not chemical in origin

Technical advances in the past four decades have made it possible to study man’s performance at pressures more than 60 times normal (equivalent to 2000 ft seawater (fsw)) using helium–oxygen breathing mixtures. Such studies are generally done in hyperbaric chambers ashore, but open-sea dives deeper than 1100 fsw have recently been made. This sort of diving has commercial, scientific, and military uses, but the safe depth limits for human exposure are not yet known, nor are the factors which may set such limits. One limiting factor may be the ability of the respiratory system to support metabolic processes at great depths. Even with helium–oxygen breathing mixtures, gas density exceeds a value seven times normal (air, 1 ATA) at 1600 fsw.
We have considered the question of respiratory function at great depths, and this study reports the results of measurements made in a hyperbaric chamber complex at a maximum pressure near 50 ATA. This pressure was selected because we wished to study man’s ability to work underwater, and therefore needed to avoid the more serious neurological disturbances which occur at greater pressures (Hunter and Bennett 1974).

MATERIALS AND METHODS

Six males were selected from healthy U.S. Navy divers who volunteered for the experiment. Two were hospital corpsmen, two were physicians, and two were equipment specialists. Their mean age was 31 yr, height 183 cm, weight 85.3 kg, and vital capacity 5.4 liters. After a period of training and base-line examinations, the divers were gradually subjected to increasing pressure by the addition of helium to a hyperbaric chamber complex in which they lived for 32 days. Chamber Pₐ was continuously monitored by a conventional fuel cell, and was maintained at 0.30 to 0.35 ATA by continuous addition of oxygen. The first week of the dive was spent in gradual compression to 49.5 ATA. During the second week, the divers performed experiments, including work on a bicycle ergometer in an adjoining chamber while immersed in 35°C water. During immersion, a mixture of helium and oxygen (49 ATA helium, 0.3–0.5 ATA O₂) was supplied from a U.S. Navy Mark 10 closed-circuit underwater breathing apparatus (Majendie and Lady 1970). Inspired gas was not humidified directly, but was probably close to being saturated with water vapor because of the recirculating nature of the Mark 10, and because of the chemical reaction of expired carbon dioxide with the absorbent, releasing water vapor. Inspired gas temperature was not measured, but it was probably close to 35°C since the entire Mark 10 apparatus (including breathing hoses) was immersed in water at that temperature.

The general design of the experiments has been described earlier (Spaur 1974; Raymond, Thalmann, Lindgren, Langworthy, Spaur, Crothers, Braithwaite, and Berghage 1975). This study presents the measurements of pulmonary function in the divers at 49.5 ATA. Three kinds of measurements were made. First, lung volumes and gas flow rates, the flow-volume relationship (V/Vol), and maximum voluntary ventilation (MVV), were measured in the usual way with a wedge spirometer (Med-Science Electronics, St. Louis) and Model 1000 direct-writing X-Y and 2-channel-time recorders (Hewlett Packard Co., Mountain View, CA.). All recorders were located outside the hyperbaric chambers, and outputs from transducers were hard-wired through bulkhead fittings. Second, quasi-static measurements of the pulmonary pressure-volume relationship were made, to define the mechanical behavior of the lungs in this unusual environment (Milic-Emili, Mead, Turner, and Glauser 1964; Raymond and Severinghaus 1971). Transpulmonary pressure was also recorded during V/Vol and MVV maneuvers and at rest. This group of measurements was made while the divers were unencumbered by immersion and its attendant need for breathing apparatus.

A third set of measurements dealt with gas exchange during immersed exercise at 49.5 ATA. Prior to immersion, the diver underwent cannulation of a radial artery under local lidocaine anesthesia. He then had electrocardiograph leads placed, donned the Mark 10 underwater breathing apparatus, and climbed down a ladder (Fig. 1) into a fiberglass tank which had been installed in a lower chamber of the hyperbaric complex. Completely immersed, the diver mounted a bicycle ergometer and extended his cannulated wrist through a sleeve which penetrated the wall of the tank. A protective surgical glove was removed. A physician stationed outside in the dry area adjacent to the tank connected the radial artery cannula to a pressure transducer, from which heart rate and blood pressure (and pulsus paradoxus) could
be continuously recorded. Heart rate was also recorded from the ECG electrodes. Respiratory rate was recorded from a thermistor in the diver’s oral-nasal mask and from fluctuations in blood pressure with breathing. Pressure in the oral-nasal mask was also recorded.

Arterial blood samples were taken in heparinized glass syringes during rest, exercise, and recovery at 49.5 ATA. During decompression, arterial blood was also obtained from all six divers during supine rest while they breathed the chamber atmosphere without artificial breathing devices. For the supine conditions, alveolar P\textsubscript{O\textsubscript{2}} was estimated by assuming a respiratory quotient (R) of 0.83, and subtracting the value of arterial P\textsubscript{CO\textsubscript{2}}/R from the inspired P\textsubscript{O\textsubscript{2}}, in the manner usually employed when collections of expired gas or end-tidal samples are not available. The use of R = 0.83 for resting divers in the postabsorptive state is supported by observations from the experiment of Raymond and his co-workers (1975) and from earlier ones (Raymond, Bell, Bondi, and Lindberg 1968) at lesser pressures.

Electrodes for the determination of arterial blood P\textsubscript{O\textsubscript{2}}, P\textsubscript{CO\textsubscript{2}}, and pH were set up in the upper chamber, using methods developed at the F. G. Hall Laboratory (Duke University Medical Center, Durham, N.C.) and calibrated by the methods of Overfield, Saltzman, Kylstra and Salzano (1969). Methods for sample preparation and analysis for plasma lactate and pyruvate concentrations were adapted from those of Hoborst (1965).

**Characteristics of the submersible ergometer**

The relationship between oxygen consumption and work rate with the submerged bicycle ergometer requires clarification. The settings of the device (Warren E. Collins, Inc., Braintree, MA) are accurate for nonimmersion work, but the submerged user does additional work against the surrounding water, against his diving suit, and against suit buoyancy under some
conditions. These factors cause $\tilde{V}_o$, for the nonpeaking condition (rest) to be considerably higher than expected for a resting subject. They also cause $\tilde{V}_o$, to be a nonlinear function of the ergometer setting during immersed work. This relationship has been clarified by unpublished measurements made in our laboratory (Table 1). Immersion increased $\tilde{V}_o$, by about 38% in 4 air-breathing subjects at 1 ATA. For comparison, the results of $\tilde{V}_o$, measurements during ergometer work at 49.5 ATA are also shown in Table 1. In these unpublished measurements, $\tilde{V}_o$, was obtained by collection and analysis of mixed-expired gas by the usual methods. For the 49.5 ATA measurements, in which expired-gas collections were not available, $\tilde{V}_o$, was obtained in another manner. While the divers wore the Mark 10 breathing device, their inspired $P_0$, was kept constant by a matrix of oxygen sensors which sampled the gas stream every 2 s, and activated an oxygen-addition solenoid if any sensor was exposed to a $P_0$, of less than 0.4 ATA. Since solenoid activity was recorded by an event marker on a strip-chart recorder, the steady-state firing rate of the solenoid provided a measure of $\tilde{V}_o$, because all leaks had been corrected as part of the pre-dive checkout. The solenoid was therefore physiologically calibrated at 49.5 ATA by displacement techniques, by collecting its oxygen output at representative firing rates in a carefully degassed 13-liter Tissot spirometer and by timing the rate of bell excursion. A calibration of $\tilde{V}_o$, vs. firing rate was constructed after conversion of volumes to STPD conditions.

**Characteristics of the underwater breathing apparatus**

The breathing equipment used during immersed work in this study was the U.S. Navy Mark 10 Mod 4 mixed-gas apparatus. The basic Mark 10 design has been described by Majendie and Lady (1970). However, its use in an earlier dive similar to that of this study (but with a maximum depth of 1000 fsw) was attended by an unacceptable level of carbon dioxide retention and acidosis at heavy work rates. The Mark 10 was therefore modified to minimize the

**TABLE 1**

Oxygen consumption during bicycle ergometer work at various ergometer settings and test conditions

<table>
<thead>
<tr>
<th>Test conditions</th>
<th>Oxygen consumption, liter/min, STPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ergometer setting, kgm min⁻¹</td>
</tr>
<tr>
<td></td>
<td>0  150  300  450  600  750</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Position</th>
<th>Pressure</th>
<th>Gas</th>
<th>Breathing apparatus</th>
<th>Ergometer setting, kgm min⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry</td>
<td>1 ATA</td>
<td>Air</td>
<td>None</td>
<td>0.35 0.55 0.97 1.22 1.44 1.82</td>
</tr>
<tr>
<td>Immersed, head out</td>
<td>1 ATA</td>
<td>Air</td>
<td>None</td>
<td>0.58 1.31 1.66 2.09 2.44</td>
</tr>
<tr>
<td>Immersed, head out</td>
<td>1 ATA</td>
<td>Air</td>
<td>Mark 10</td>
<td>0.46 0.95 1.41 1.88 2.50 3.14</td>
</tr>
<tr>
<td>Immersed fully</td>
<td>49.5 ATA</td>
<td>He–O₂</td>
<td>Mark 10</td>
<td>0.62 1.80 1.92 – – – –</td>
</tr>
</tbody>
</table>

Values are means; $n = 4$ at 1 ATA, $n = 3$ at 49.5 ATA. SEM ranged from 0.04–0.18.
breathing resistance, in particular by using larger breathing hoses and by streamlining elbows and other abrupt geometric transitions in the hoses. The design changes and associated changes in breathing characteristics of the Mark 10 Mod 4 device are described in detail by Cetta and Radecki (1975). Of special interest are the results of measurements of pressure changes in the M-10 oral-nasal mask of the modified Mark 10 when tested with a breathing machine at 49.5 ATA, which simulated human use at that pressure. In this test, the breathing machine manikin generated a tidal volume of 2.0 liters at a breathing rate of 20 breaths per min using a gas mixture of helium with 0.4 ATA oxygen. During inspiration, the oral-nasal mask pressure was 12 cmH₂O below ambient at its minimum and during expiration it was 23 cmH₂O above ambient at its maximum. As the Results section below will show, the fluctuations in pressure within the oral-nasal mask during actual use by the divers in the 1600-fsw chamber dive reported in this study were considerably less than the values described by Cetta and Radecki (1975).

RESULTS

Experimental findings will be presented in two main sections: those related to exercise during immersion, and those from ventilatory maneuvers done in the dry, resting state without the encumbrances of breathing apparatus, diving suit, immersion, and arterial cannulation.

Exercise during immersion

The ability of the divers to work was greatly impaired at 49.5 ATA. Of the three men in whom arterial cannulation was successfully accomplished, all were able to complete 6 min of work on the submerged ergometer at the work rate setting of 150 kgm • min⁻¹ (Fig. 2, 150 kgm • min⁻¹). Only one man was able to complete the scheduled 6 min of work at the higher ergometer setting (300 kgm • min⁻¹, \( \dot{V}_{\text{O}_2} = 1.92 \) liter/min). The other two divers stopped work early, due to severe breathlessness and a sense of incipient syncope. The same symptoms occurred in the diver who completed 6 min of 300-kgm • min⁻¹ ergometer work, leaving him unwilling to attempt work at the 450-kgm • min⁻¹ setting. In contrast, the divers who used this apparatus at lesser pressures (1–10 ATA) prior to the 49.5 ATA studies routinely completed successive 6-min work periods at loads up to 750 kgm • min⁻¹ or higher, producing arterial lactate levels of 80–100 mg/dl. In the 1–10 ATA work, the men experienced only mild dyspnea and no presyncopal symptoms.

The divers characterized the sense of breathlessness which limited exercise at 49.5 ATA as an intense drive to inspire. The sensation was mild at the start of work, but became more intense with time at a given work rate. Near exhaustion, it increased with inspiration but persisted into the expiratory phase, as if the full breath had not satisfied some stimulus. Some divers found the symptom alarming in intensity, but noted that it disappeared within 30 s of the end of work, reaching a peak during the 10 s after stopping exercise. Arterial blood gas values at the time of exhaustion (Fig. 2) showed that CO₂ retention was not the limiting factor. Hypoxemia was also excluded, since direct measurements showed that arterial P₂O₂ exceeded 180 mmHg in all samples. Cellular hypoxia was not absolutely eliminated as the cause of breathlessness, but seems unlikely for two reasons: arterial pH remained normal, and plasma lactate levels increased only mildly with exercise, falling to the pre-exercise range within the 6-min recovery period. Heart rate and blood pressure responded normally to exercise (Table 2), minimizing the possibility of hemodynamic abnormalities being the work-limiting factors.
Fig. 2. Responses to underwater work at 49.5 ATA. Severe breathlessness and incipient syncope limited exercise to a maximum ergometer setting of 300 kgm $\cdot$ min$^{-1}$ (see Tables 1 and 2 for other results). Mean values (± SE) are given for divers 2, 5, and 6.

### TABLE 2

Cardiorespiratory responses to exercise at 49.5 ATA

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Rest</th>
<th>150</th>
<th>300</th>
<th>3</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen consumption, liter/min, STPD</td>
<td>0.62</td>
<td>1.80</td>
<td>1.92</td>
<td>0.80</td>
<td>0.68</td>
</tr>
<tr>
<td>Heart rate, min$^{-1}$</td>
<td>87±7</td>
<td>130±4</td>
<td>137±7</td>
<td>99±7</td>
<td>96±6</td>
</tr>
<tr>
<td>Blood pressure, mmHg</td>
<td>126/82</td>
<td>150/85</td>
<td>134/79</td>
<td>132/80</td>
<td>128/83</td>
</tr>
<tr>
<td>Respiratory rate, min$^{-1}$</td>
<td>13±2</td>
<td>21±1</td>
<td>29±1</td>
<td>20±2</td>
<td>18±4</td>
</tr>
<tr>
<td>Pulsus paradoxus, mmHg</td>
<td>24±6</td>
<td>27±1</td>
<td>32±7</td>
<td>24±5</td>
<td>23±6</td>
</tr>
<tr>
<td>Pressure fluctuations in oral-nasal mask, cmH$_2$O</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspiration</td>
<td>-2.2±0.1</td>
<td>-5.4±0.6</td>
<td>-5.2±0.6</td>
<td>-3.0±0.1</td>
<td>-2.3±0.1</td>
</tr>
<tr>
<td>Expiration</td>
<td>4.5±0.2</td>
<td>10.9±1.3</td>
<td>10.5±1.3</td>
<td>6.0±0.2</td>
<td>4.7±0.2</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 3.
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However, the blood pressure recordings showed that an abnormal degree of pulsus paradoxus (greater than 10 mmHg) was present during both rest and exercise, suggesting that substantial obstruction to gas flow was present in the divers' airways, or in the breathing apparatus (Rebuck and Pengelly 1973). The rather small fluctuations in oral-nasal mask pressure (Table 2) appear to exonerate the breathing apparatus as the source of the obstruction. It should be pointed out in this regard that breathlessness was reported during even mild exertion during this experiment in the dry, unencumbered state at depths beyond about 1200 fsw. This was noted by all 6 divers, and at 1600 fsw, this sensation often limited conversation to phrases of 5-6 words between breaths.

Spirometry and blood gas composition under dry, resting conditions

More direct evidence of obstruction in the tracheobronchial tree is available from the $\dot{V}/\text{Vol}$ and MVV measurements. Peak expiratory flow was reduced by almost one-half at 49.5 ATA (Table 3). Flow rates were reduced at all lung volumes as ambient pressure was raised (Fig. 3), despite driving pressures which were much greater than the control values (Table 3). For example, peak expiratory pressures during $\dot{V}/\text{Vol}$ maneuvers at 49.5 ATA ranged between $+140$ and $+265$ cmH$_2$O, and peak inspiratory pressures ranged between $-80$ and $-96$ cmH$_2$O.

<table>
<thead>
<tr>
<th>Pulmonary function measurement</th>
<th>Atmosphere breathed and total pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air, 1 ATA</td>
</tr>
<tr>
<td>Lung volumes</td>
<td></td>
</tr>
<tr>
<td>Vital capacity, liters</td>
<td>6.0–6.2</td>
</tr>
<tr>
<td>Expiratory reserve volume, liters</td>
<td>2.0–2.2</td>
</tr>
<tr>
<td>Maximum voluntary ventilation</td>
<td></td>
</tr>
<tr>
<td>Transpulmonary pressure, cmH$_2$O</td>
<td></td>
</tr>
<tr>
<td>Inspiration</td>
<td>$(34–36)$</td>
</tr>
<tr>
<td>Expiration</td>
<td>$(32–35)$</td>
</tr>
<tr>
<td>Tidal volume, liters</td>
<td>2.7–2.9</td>
</tr>
<tr>
<td>MVV, liter/min</td>
<td>178–182</td>
</tr>
<tr>
<td>Forced expiratory flow-volume curve</td>
<td></td>
</tr>
<tr>
<td>Transpulmonary pressure, cmH$_2$O</td>
<td>$(35–65)$</td>
</tr>
<tr>
<td>Peak expiratory flow, liter/s</td>
<td>12.4–12.8</td>
</tr>
<tr>
<td>Resistance, cmH$_2$O per liter/s</td>
<td>2.8–5.2</td>
</tr>
<tr>
<td>Static compliance, liter/cmH$_2$O</td>
<td>0.20–0.28</td>
</tr>
</tbody>
</table>

Values are means for divers 5 and 6, in whom esophageal pressure was measured; similar lung volume, MVV, and flow-volume relationships were found for divers 1–4, whose esophageal pressures were not measured. All volumes corrected to BTPS.
Fig. 3. Analysis of maximal expiratory flow-volume curves observed at chamber pressures equivalent to 0–1600 fsw; values are means for 6 divers.

For both inspiration and expiration, these peak pressures were about four times the respective predive values in air at 1 ATA. These measures achieved flow rates only half as great as the control values, however. Similar results were obtained with MVV maneuvers, although the divers adapted to the 49.5 ATA condition and performed MVV in a manner which differed from the predive, 1 ATA state. For example, at 49.5 ATA, they were already breathing higher in the lung volume, since expiratory reserve volume (ERV) was increased from 2.2 to 2.8 liters (Table 3). During MVV, they breathed with smaller tidal volumes (Fig. 4, $V_T = 1.2$ vs. 2.9 liters, predive), and tended to accumulate additional gas within the lungs in the course of the brief MVV maneuver. Peak values of transpulmonary pressure with MVV were almost twice as large as the predive control values. All variables were remeasured at the end of decompression (Table 3, He–O₂, 1.2 ATA). These measurements showed the expected supranormal values in $V_E$ and MVV (Fig. 4) which would be anticipated with the respiration of a less-dense-than-air medium, such as helium with 30% oxygen at a nearly normal pressure (1.2 ATA).

In addition to the arterial blood gas determinations during immersed work, arterial $P_{O_2}$, $P_{CO_2}$, and pH during supine rest were measured on repeated occasions during the 3-wk decompression from 49.5 ATA. As was true during immersed work (Fig. 2), there was no evidence of respiratory acidosis in any diver. On the contrary, the tendency was to respiratory alkalosis (Table 4), but neither arterial $P_{CO_2}$ nor pH was systematically related to depth. These low values of resting $P_{CO_2}$ reflect the results in two subjects at 600–1500 fsw reported by Morrison, Bennett, Barnard, and Eaton (1976). The high values of arterial $P_{O_2}$ at all depths (Fig. 5) reflected the hypoxic chamber atmosphere. The alveolar-to-arterial oxygen difference ($AaD_O$) during rest ranged between 16 ± 8 (SE) and 51 ± 1 mmHg, as might be expected during hyperoxic breathing (Cole and Bishop 1967). The value of 18 mmHg at 1600 fsw agrees well with the findings of Overfield et al. (1969) from a 1000-fsw chamber dive, when the greater
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Fig. 4. Breathing pattern (tidal volume vs. time) (upper panels) and transpulmonary pressure (lower panels) during maximum voluntary ventilation (MVV). Control measurements were made in air at normal pressure (left). During MVV at 35 ATA (center), diver began MVV maneuver with an already increased expiratory reserve volume, and retained additional gas during MVV, staying progressively above functional residual capacity (FRC), shown by dashed lines, as the maneuver proceeded. The individual breaths (tidal volumes) at 35 ATA were smaller, despite greater efforts shown by wider swings in transpulmonary pressure. At end of 19-day decompression (panels at right), breathing pattern during MVV had become nearly normal. Recordings are from diver 6. Results were same at 49.5 ATA (see Table 3), but recordings did not reproduce satisfactorily.

degree of hyperoxia in this study is taken into account. Like the arterial $P_{CO_2}$ and pH, the $AaDO_2$, was not systematically related to chamber pressure (gas density). The high value found at 1000 fsw in this study is not readily explained.

<table>
<thead>
<tr>
<th>Chamber Depth, fsw</th>
<th>1,378</th>
<th>1,192</th>
<th>1,000</th>
<th>808</th>
<th>424</th>
<th>232</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial $P_{CO_2}$</td>
<td>38 ± 1.0</td>
<td>39 ± 4.0</td>
<td>36 ± 2.0</td>
<td>37 ± 2.0</td>
<td>37 ± 2.0</td>
<td>34 ± 2.0</td>
</tr>
<tr>
<td>pH</td>
<td>7.47 ± .03</td>
<td>7.41 ± .03</td>
<td>7.42 ± .03</td>
<td>7.43 ± .02</td>
<td>7.45 ± .01</td>
<td>7.42 ± .01</td>
</tr>
</tbody>
</table>

Values are means ± SD; $n = 6$. 
Fig. 5. Alveolar and arterial $P_O_2$, in supine males undergoing decompression from 49.5 ATA maximal pressure. $AaDO_2$ was not a function of gas density (chamber pressure). Mean values ($\pm$ SE) are for $n = 6$ at the three greatest depths, and for $n = 3--4$ at the lesser depths.

DISCUSSION

When diving mammals dive, the gas in their airways becomes more dense the deeper the dive. Man is no exception to this maxim, so in 1926, Sayers and Yant (1926) introduced the newly discovered light gas, helium, as a breathing medium for divers. Even with helium, however, there probably exists some limiting depth (pressure) at which gas density may render the respiratory system inadequate to support metabolic processes (Fagraeus 1974; Lanphier 1976; Lambertsen 1976). In the present study, we measured some indices of pulmonary function in the dry chamber atmosphere at 49.5 ATA and attempted to relate them to measurements of cardiorespiratory function during maximal exercise in the immersed state at the same pressure. Other, nonpulmonary, factors which might limit performance in open-sea diving were excluded by selecting experienced, healthy divers, and by providing them with comfortably warm gas and water temperatures. Hypoxia and extreme hyperoxia were also excluded. The external resistance to breathing imposed by the Mark 10 Mod 4 system was mild (Cetta and Radecki 1975). The work task was simple, employing apparatus and procedures with which the divers and investigators were familiar at lesser pressures. There is little likelihood that hemodynamic or metabolic factors were responsible for the limited exercise tolerance of the divers (Rodkey, Raymond, Collison, and O’Neal 1974; Raymond, Sode, Spaur, Uddin, Johnsonbaugh, Bauer, Knott, and Crothers 1974).

This study showed that carbon dioxide retention and its accompanying acidosis were not responsible for limiting the ability of the divers to do underwater ergometer work at 49.5 ATA. In showing that $O_2$ and $CO_2$ exchange did not limit exercise tolerance at this pressure, we
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extended the observations of Overfield et al. (1969), who found no hypercapnia and only small increases in $\text{AaD}_{\text{O}_2}$ at 31 ATA. This is in keeping with the more recent findings of Peterson and Wright (1976) and Lambertsen (1976), whose subjects appeared capable of performing greater work loads while breathing considerably denser breathing media with apparent ease. The relative limitation in our divers compared to those of Lambertsen (1976) is puzzling to us. One explanation might be a difference in work task or breathing equipment, but such would not account for the dyspnea which all 6 of our divers noticed with mild exertion or animated conversation at depths below about 1200 fsw, but which occurred in the Lambertsen subjects only at high work rates breathing Ne–$\text{O}_2$ at 1200 fsw (equivalent to He–$\text{O}_2$ at 5000 fsw). The only major difference between the two groups of subjects was age, which seems a weak explanation for such a large discrepancy in exercise tolerance.

The lack of alternative explanations for the exercise intolerance and exertional dyspnea in our divers focuses attention on possible changes in mechanics of breathing as the cause of their symptoms and reduced performance. Such changes might have resulted from either restrictive processes leading to increased lung stiffness, or from obstruction to gas flow in the lungs. With regard to the first possibility, no evidence of lung stiffness was found in the two men in whom pressure-volume curves were measured, both of whom were subjects in the exercise tests. We concluded, therefore, that structural changes in the lungs were not a factor in the limiting symptom of dyspnea in this study. Instead, dyspnea in these divers appears to have resulted from their efforts to overcome the resistance to flow of a gas seven times the density of air at 1 ATA (He–$\text{O}_2$ at 49.5 ATA). From measurements made in the dry, resting state, we inferred that the working divers were breathing higher in their lung volume, and exerting unusually great transpulmonary pressures to maintain adequate alveolar ventilation, in the face of increasing $\text{CO}_2$ production during work. Since it was not possible to measure esophageal pressures in the divers while they worked underwater at 49.5 ATA (they were already encumbered with underwater breathing apparatus, radial artery cannulation, and electrocardiographic leads), we recognize that our esophageal pressures from the $\dot{V}/\dot{V}_{\text{vol}}$ and MVV maneuvers provide only indirect assessment of what happened to lung mechanics during immersed exercise. However, the fact that the qualitative impairment, less flow despite greater driving pressure, was the same in both the $\dot{V}/\dot{V}_{\text{vol}}$ and MVV measurements, makes it reasonable to suppose that these maneuvers offer a useful insight into the origin of the divers’ dyspnea during immersed exercise. Clearly, if the men breathed during work as they did during the MVV maneuvers (Fig. 4) marked dyspnea would be expected to impair function at least as rapidly as what we observed. Additional studies with more detailed measurements of lung mechanics during exercise should help clarify some of the questions raised by the present observations.

Results of spirometric measurements

The high gas density encountered at the maximum chamber pressure in this study was expected to reduce gas flow rates in the airways in accordance with the findings of Schilder, Roberts, and Fry (1963). Since their study, others have made observations on the changes in $\dot{V}/\dot{V}_{\text{vol}}$ relationships caused by increased density. For example, Broussolle, Chouteau, Hyacinthe, Le Pechon, Burnet, Battesti, Cresson, and Imbert (1976) included such measurements in a chamber dive similar to ours, but at a greater pressure. The $\dot{V}/\dot{V}_{\text{vol}}$ changes in our divers (Fig. 6) are quite similar to those which they reported. In a more elaborate study of $\dot{V}/\dot{V}_{\text{vol}}$, Anthonisen, Bradley, Vorosmarti, and Linaweaver (1971) varied both pressure and gas composition to yield relative densities (air, 1 ATA = 1.0) from 0.4 to 15.0. Interpolating among their high-density values, we again note substantial agreement of our data with theirs.
Fig. 6. Comparison of flow-volume (V/V) relationship, air at 1 ATA vs. He–O₂ at 49.5–51 ATA. There was close agreement between present (n = 6) results and those of Broussolle et al. (1976) (n = 2).

Fig. 7. Reduction in MVV with increasing pressure (adapted from Lanphier (1976)). Hysteresis, higher values during decompression than during earlier, compression measurements, probably reflects a training effect; values are means ± SE for n = 6.
MECHANICAL ORIGIN OF DYSPNEA AT 49.5 ATA

MVV has been measured in a larger number of studies over a wide range of pressures and gas densities. The use of MVV as an index of pulmonary function can be criticized on a number of grounds, of course, but it should probably continue to be measured in studies such as this, since it provides continuity among diverse sets of observations. In our divers, MVV decreased as a hyperbolic function of ambient pressure in a manner similar to the initial predictions of Laapheier (1976), which have recently been reviewed. Similar results were more recently reported by others (Broussolle et al. 1976; Peterson and Wright 1976). A direct comparison of the results of MVV measurements by different groups is made difficult by discrepancies in age, size, training, and other characteristics of the subjects and equipment used in testing. For example, not all reports give control (air, 1 ATA) data for comparison. Nevertheless, it is apparent that a similar hyperbolic fall-off in MVV with increasing ambient pressure has been shown by most of the He – O2 studies (Fig. 7). An unexpected finding in the present study was that of hysteresis in the MVV-depth relationship such that MVV values for any given depth were higher in the decompression phase of the dive than for the initial measurement. Most of this increase in MVV is probably attributable to training (Leith and Bradley 1976), but other factors cannot be excluded.

Relationship between MVV and exercise ventilation

The relationship between MVV and minute volume of ventilation (\(\dot{V}_E\)) during exercise should also be considered. This relationship is not clearly established for man at 1 ATA. Since MVV is a brief, somewhat exhausting maneuver, it is obvious that ventilation at this level can not be long sustained (Leith and Bradley 1976). While the actual ratio of maximal exercise-\(\dot{V}_E\) to MVV is therefore less than 1.0, the actual values vary quite widely (Fagraeus 1974; Lapheier 1976). Like Broussolle et al. (1976), we wished to estimate the ratio of \(\dot{V}_E\) during maximal exercise to MVV in our divers. To do so required a few assumptions, since \(\dot{V}_E\) was not measured directly in this study. First, it was assumed that \(\dot{V}_{CO_2} = \dot{V}_{O_2}\) (respiratory exchange ratio = 1.0), as is usually true with moderate exercise associated with a small rise in lactate production. If one also assumes that alveolar P\(_{CO_2}\) is equal to the measured arterial P\(_{CO_2}\), then one can solve equation (1) for the value of alveolar

\[
\dot{V}_A = \frac{863 \cdot \dot{V}_{CO_2}}{P_{A CO_2}}
\]

ventilation, \(\dot{V}_A\). In our divers at the limit of exertion, we found \(\dot{V}_{CO_2} = 1.9\) liter/min STPD, and P\(_{A CO_2}\) = 36 mmHg (the constant 863 is needed to yield values of \(\dot{V}_A\) in BTPS units), and thus computed \(\dot{V} = 45\) liter/min, BTPS. Since \(\dot{V}_E = \dot{V} + \dot{V}_D\), we computed \(\dot{V}_D\) from the observed respiratory rate and an assumed dead space (\(\dot{V}_D = 400\) ml), such that \(\dot{V}_D = 29 \times 0.4 = 12\) liter/min, and \(\dot{V}_E = 57\) liter/min, BTPS, at 49.5 ATA. This value of \(\dot{V}_E\) turns out to be about 56% of MVV at the same pressure (mean MVV = 102 liter/min).

From the data reviewed by Lapheier (1976), this level of \(\dot{V}_E\), expressed as a percentage of the MVV, can probably be sustained for only about 15 min under optimal conditions. One must then recall that MVV in our study was measured with only the imposed resistance of the measuring apparatus, a balanced wedge spirometer. For the immersed diver with his suit, surrounding water and Mark 10 breathing equipment, even though the measured breathing-resistance was low, it seems reasonable to expect that the duration for which \(\dot{V}_E\) could be
sustained at 50–60% of MVV might be considerably less than 15 min, although we did not make direct measurements of duration in this study. However, we believe that the limitation to exercise in our divers was one of mechanical origin, related to the work of breathing. The precise source of such limitation is not clear, however. The main possibilities seem to be as follows. First, there is the tendency of the divers to breathe at abnormally high lung volumes (Fig. 4), resulting in the addition of elastic loading superimposed on a recognizably high resistive impedance. A second possibility is that the high density of the breathing mixture resulted in intense stimuli from shearing or impaction forces (or both) on mucosal surfaces, especially during exercise. A corollary of this idea is that respiratory muscles were subjected to unaccustomed loads at maximal levels of exercise, and they responded by generating adverse stimuli. A third notion is similar to the first two but deals with a more elusive proposition: that perfusion of the respiratory muscles becomes inadequate in these unusual circumstances to support the unique metabolic requirements of the high resistive and elastic loads of these experimental conditions. The design of the present study does not allow us to choose among these (and other) possibilities, but the results seem vulnerable to further experimental attack.

**Perspective**

The physiological changes exhibited by the divers include some of the adjustments seen in acute attacks of bronchospasm in asthmatic humans (Permatt 1973). These include hyperinflation, dyspnea in the absence of hypoxemia or CO₂ retention, impaired flow rates, and increased pulsatia paradoxus. In regard to the clinical diagnosis of asthma or exercise-induced bronchospasm, we point out that auscultation of the chest on repeated occasions before and after exercise showed no evidence of airway dysfunction. Whether the similarities of the responses in our divers to dense gas breathing vis-a-vis those of asthmatics are merely coincidental or are indicative of a similarity in mechanism at some level must remain a matter of continuing inquiry. "For a long time, it has been assumed that the increase in density of the respired gases might be a limiting factor in underwater exploration" (Varene, Vieillefond, Lemaire, and Saumon 1974). We agree that density may limit underwater performance, though the mechanism of the limitation remains unclear. Uncertainty in this regard, however, will serve as a catalyst rather than a damper for further study, at least for the physiologist and diving medical officer.

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mesurées pour les volumes pulmonaires plus grands. Ces réductions se sont produites malgré une augmentation apparente de la capacité fonctionnelle résiduelle, et l’emploi de pressions transpulmonaires supérieures à celles qu’on observe au cours des mêmes exercices à une pression normale (au niveau de la mer). Pendant le travail subaquatique, les sujets se sont fatigués rapidement à des taux de consommation d’oxygène modérés (1,9 l/min); la fatigue était accompagnée d’une dyspnée importante et de la syncope imminente. Nous n’attribuons pas ces symptômes à la rétention de CO₂, ni à des causes hémodynamiques ou métaboliques, mais plutôt à la respiration d’un gaz dense, ce qui représente, avec l’asthme, une condition dans laquelle peut survenir une dyspnée importante accompagnée d’un taux normal ou même sous-normal de CO₂ artérielle et un transport d’oxygène normal. Les adaptations physiologiques employées par les plongeurs ressemblent à celles qu’on observe chez l’asthmaticique aigu. Les hommes peuvent accomplir un travail modeste sous les conditions de cette expérience, mais la réserve physiologique, qui permettrait aux plongeurs de faire face aux urgences ou aux infections respiratoires éventuelles, se trouve diminuée.

asthme  exercise

dioxyde de carbone  hélium

REFERENCES


