Effects of age and exercise on physiological dead space during simulated dives at 2.8 ATA


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Mummery, H. J., B. W. Stolp, G. deL. Dear, P. O. Doar, M. J. Natoli, A. E. Boso, J. D. Archibald, G. W. Hobbs, H. E. El-Moalem, and R. E. Moon. Effects of age and exercise on physiological dead space during simulated dives at 2.8 ATA. J Appl Physiol 94: 507–517, 2003. First published October 11, 2002; 10.1152/japplphysiol.00367.2002.—Physiological dead space (VDS), end-tidal CO2 (PETCO2), and arterial CO2 (PaCO2) were measured at 1 and 2.8 ATA in 10 older (58–74 yr) and 10 younger (19–39 yr) air-breathing subjects during rest and two levels of upright exercise on a cycle ergometer. At pressure, VD (liters BTPS) increased from 0.34 ± 0.09 (mean ± SD of all subjects for normally distributed data, median ± interquartile range otherwise) to 0.40 ± 0.09 (P = 0.0060) at rest, 0.35 ± 0.13 to 0.45 ± 0.11 (P = 0.0003) during light exercise, and 0.38 ± 0.17 to 0.45 ± 0.13 (P = 0.0497) during heavier exercise. During these conditions, PaCO2 (Torr) increased from 33.8 ± 4.2 to 35.7 ± 4.4 (P = 0.0059), 35.3 ± 3.2 to 39.4 ± 3.1 (P < 0.0001), and 29.6 ± 5.6 to 37.4 ± 6.5 (P < 0.0001), respectively. During exercise, PETCO2, overestimated PaCO2, although the absolute difference was less at pressure. Capnography poorly estimated PaCO2, during exercise at 1 and 2.8 ATA because of wide variability. Older subjects had higher VD at 1 ATA but similar changes in VD, PaCO2, and PETCO2 at pressure. These results are consistent with an effect of increased gas density.

departmental gas exchange; hypercapnia; hyperbaric; end-tidal CO2; arterial CO2; aging

Hypercapnia is a well-described consequence of hyperbaric exposure, the mechanisms of which are incompletely understood (31, 48). Many studies in the past half century have described elevated end-tidal PaCO2 (PETCO2) in exercising subjects, often higher than 60 Torr, at pressures of 3.0 atmospheres absolute (ATA) and greater (14, 19, 24, 37, 40, 54). Measurements of arterial CO2 tension (PaCO2), although less common, confirm that hypercapnia of varying degrees occurs during exercise at high atmospheric pressure (28, 47) and with dense gas breathing at 1 ATA (57). Hypercapnia is a potentially serious problem for divers, contributing to central nervous system O2 toxicity, inert gas narcosis, and loss of consciousness (31).

The cause of hypercapnia in divers is multifactorial. Mechanisms that may contribute include the following. 1) Hypoventilation secondary to increased work of breathing may be due to the effects of increased gas density (10). Expiratory flow is limited by an increase in airway resistance, and inspiratory elastic work is increased because of breathing at a higher lung volume (51). In immersed divers, the work of breathing is further increased by a redistribution of blood into the thorax, causing a decrease in lung compliance (1). 2) A blunted ventilatory response to exercise with inappropriate minute ventilation (VE) (28, 31) may be due to factors other than gas density. Possible causes of this phenomenon include self-selection among divers, an acquired adaptation to hyperbaric exposure, or an alteration in central regulatory mechanisms (15, 27). 3) Increased physiological dead space (VDS) (46, 47) may be possibly due to changes in the distribution of ventilation as a consequence of dense gas breathing. Increased anatomic VDS as a result of an enlarged functional residual capacity may contribute (51). Although increased VDs does not by itself lead to hypercapnia, it will do so if it is not accompanied by a compensatory increase in total ventilation.

Of these factors, the increase in physiological VDs is the least explored. Saltzman et al. (46) reported elevated Bohr VDs in resting subjects breathing dense gas mixtures at increased atmospheric pressure (up to 7.7 g/l at 7 ATA). The first evidence that changes in VDs may contribute to hypercapnia during hyperbaric exercise was reported by Salzano et al. (47), who found significant hypercapnia and very large increases in VDs and VDs-to-tidal volume (VT) ratio (VDs/VT) in divers performing exercise in a dry chamber at the extreme simulated depths of 460 and 650 m (pressures 47 and 66 ATA and gas densities 12.3 and 17.1 g/l, respectively). Increased VDs/VT and PaCO2 have also been observed in resting subjects breathing dense gas mixtures at 1 ATA (57). Measurements of VDs at shallower depths have not been reported.

If VDs were to increase at commonly encountered recreational depths, older divers especially might be at risk for hypercapnia.
increased risk of respiratory impairment. Aging is associated with increased VDS (5, 11, 38, 43), loss of respiratory muscle strength (21, 26), and decreased lung elastic recoil and chest wall compliance (21). Older subjects also exhibit decreased ventilatory responses to exercise and hypercapnia (8, 42). An increase in VDS could accentuate relatively minor changes in resting gas exchange in older divers and increase the risk of CO2 retention.

Furthermore, although P\textsubscript{ETCO2} closely estimates PaCO\textsubscript{2} in young, healthy individuals at rest (41, 44), its accuracy has not been assessed under hyperbaric conditions. As an estimate of PaCO\textsubscript{2}, capnography is falsely high during conditions associated with increased CO\textsubscript{2} production, such as exercise (44, 55), and falsely low in the presence of increased heterogeneity of ventilation or increased VDS (34, 35), which may occur during diving or with normal aging (5, 12). Therefore, under hyperbaric conditions, P\textsubscript{ETCO2} may be less accurate in all age groups as an estimate of PaCO\textsubscript{2}.

The primary goal of this study was to examine VDS at a relatively shallow depth of 18.3 m of sea water [60 ft of sea water (fsw)], equivalent to 2.8 ATA, and determine whether the changes in VDS observed at extreme pressures also occur at a simulated depth commonly encountered by recreational divers. Additional goals of this study were to establish the degree of hypercapnia experienced by both younger and older divers during moderate exercise at 2.8 ATA and to compare capnography and direct arterial measurement of CO\textsubscript{2} to evaluate the accuracy of P\textsubscript{ETCO2} as an estimate of PaCO\textsubscript{2} across a range of ages.

**METHODS**

**Subjects.** After institutional approval and informed consent, 20 subjects were studied. Subjects were divided into two groups by age [younger (Y), 19–39 yr; older (O), 58–74 yr] and screened for cardiac and pulmonary disease by medical history, physical exam, posterior-anterior and lateral chest radiographs, spirometry [forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV\textsubscript{1})], and treadmill electrocardiogram (ECG) (group O only). Subjects were excluded who had FEV\textsubscript{1}/FVC < 0.70, contraindications to diving, clinical evidence of heart or lung disease, or pregnancy.

**Apparatus.** The study took place in a dry hyperbaric chamber (volume 40.5 m\textsuperscript{3}). The temperature was adjusted for comfort between 22 and 24°C (surface) and 24 and 27°C (pressure). One physician and two attendants were present in the chamber and in communication with an outside attendant at all times. Standard hyperbaric safety procedures were observed.

Subjects sat on a mechanically braked bicycle ergometer (model 818, Monark Exercise, Vansbro, Sweden), which was fitted with electrical work rate and rpm outputs. After abrasive skin preparation, a three-lead ECG was applied and used to determine heart rate. At the beginning of the study day, subjects were instrumented in the radial artery with a 20-gauge arterial catheter with the use of sterile technique and local anesthesia (1% lidocaine). The catheter allowed periodic blood samples to be drawn and was connected to a transducer (model 42370-01, Abbott Critical Care, Morgan Hill, CA) placed 5 cm inferior to the sternal angle, from which systolic, diastolic, and mean pressures were monitored. To prevent a fall in the event of syncope, subjects wore a safety harness that was attached via a pulley system to the roof of the chamber.

Subjects breathed dry air from a 150-liter bag connected to a low-resistance mouthpiece with one-way valves to allow separation of expired and inspired gases (model 2700, Hans Rudolph, Kansas City, MO). The inspired loop included a Fleisch no. 4 pneumotachometer, which was used to record breathing frequency. Expired gas was collected in leak-tested 100-liter Douglas bags (WE Collins, Braintree, MA), which were opened and closed with a three-way stopcock (model 2100, Hans Rudolph). Components of the breathing loop were connected by leak-free tubing (1% in. ID, WE Collins). P\textsubscript{ETCO2} was measured by a high-flow capnograph (CapnocheckPlus, BCI International, Waukesha, WI; modified to increase sample flow rate to 250 ml/min), which withdrew gas from a port on the mouthpiece 2 cm from the subject’s mouth and returned the gas at a port distal to the expiratory valve. Inspired CO\textsubscript{2} level was monitored to confirm that rebreathing did not occur. Mouthpiece pressure was measured by a pressure transducer connected to the mouthpiece port. Nose clips were worn to ensure that ventilation occurred through the mouthpiece.

Spirometry was performed at the beginning of each surface and pressure session with the use of a rolling seal spirometer (Sensormedics, Anaheim, CA) with electrical output. Subjects performed forced maximal expirations, from which FVC, FEV\textsubscript{1}, and forced expiratory flow in the middle 50% of the exhalation (FEF\textsubscript{25–75}) were determined. The best value of four trials was used for analysis. Spirometer temperature and atmospheric pressure were recorded for each trial and used to correct volumes for body temperature, ambient pressure, and water vapor saturation (BTPS). A body temperature of 37°C was assumed.

All electrical outputs from inside the chamber were hard-wired through bulkhead fittings to preamplifiers outside the chamber. The amplifier for the blood pressure transducer and ECG signals was provided by a clinical monitor (model 514, Spacelabs, Hillsboro, OR). Output from the pneumotachometer was connected to a signal processor (model CD19A, Validyne, North Ridge, CA). All signals were digitized and recorded with MacLab (model 16/8, Analog Digital Instruments, Mountain View, CA) and Chart software (version 3.5, ADInstruments, Castle Hill, New South Wales, Australia) on a Macintosh computer (model 7600/120, Apple Computer, Cupertino, CA).

**Experimental protocol.** The experimental protocol consisted of trials performed first at the surface and then repeated after a 20- to 30-min break at a pressure of 2.8 ATA (corresponding to a simulated depth of 60 fsw or 18.3 m of sea water). We did not randomize the order of surface and pressure experimental protocols because of the potential for initiating decompression illness by performing heavy exercise immediately after decompression. After spirometry, subjects performed the following conditions while sitting upright on the bicycle: 1) rest (5 min); 2) exercise 1 (6 min); and 3) exercise 2 (6 min). Atmospheric pressure and chamber temperature were recorded for each condition.

Exercise levels were based on each subject’s level of physical fitness, such that exercise 1 could easily be maintained for 6 min and exercise 2 would be strenuous but would not exceed the subject’s maximal exercise capacity. For the purposes of this experiment, a value of 95% of the subject’s predicted maximum heart rate (estimated by 220 minus age in years) was used as a guide to exercise capacity. Determination of each subject’s maximal O\textsubscript{2} consumption (V\textsubscript{O2}) was not performed because the qualitative effects of exercise were
more important to the outcome variables than the exact level of exercise performed. To allow for a possible effect of exercise above the aerobic capacity on gas exchange variables during exercise 2, comparisons were also made for all variables during exercise 1. When possible, the same exercise levels were performed at 1 and 2.8 ATA, as determined by subject comfort and fatigue.

Work rates were achieved by adjusting the work load on the ergometer while the subjects pedaled at 80 rpm. Between each exercise level, subjects rested until heart rate had returned to baseline or for at least 10 min. Between trials at 1 and 2.8 ATA, subjects rested for 30–60 min.

At the third minute of rest, expired gases were collected for at least 2 min. At the fifth minute of exercise, two bags of expired gases were collected sequentially for 1 min each. Values from the fifth and sixth minutes were compared to verify steady state. Values from the sixth minute were used in all analyses. At the end of each recording session, gas samples were taken from the inspired and expired Douglas bags in 100-ml wetted, gas-tight glass syringes. The expired gas volume was measured by evacuating each bag to a pressure of 5 cmH2O through a calibrated gasometer (model DTM 325-4, American Meter, Nebraska City, NE). Gas analysis was performed on a gas chromatograph (model 3800, Varian, Walnut Creek, CA) for O2 and CO2 concentrations.

During the third minute of rest and the sixth minute of each exercise period, two arterial blood samples were collected anaerobically over 15–30 s in heparinized gas-tight glass syringes. One sample was analyzed immediately within the chamber for pH, arterial O2 tension (PaO2), and PaCO2, with a blood-gas analyzer calibrated for use at the ambient pressure of the chamber (model Synthesis 15, Instrumentation Laboratory, Lexington, MA). The second sample was stored on ice and analyzed within 30 min of collection for hemoglobin concentration and O2 saturation with a CO-oximeter (model 482, Instrumentation Laboratory) located outside the chamber.

The time at simulated depth ranged from 52 to 60 min. Decompression protocols were derived from a conservative modification of US Navy air decompression tables (following tables for 70 fsw and the next longest time). Decompression proceeded at a rate of 30 ft/min from the simulated depth of 60 to 30 fsw, and then at 10 ft/min to 10 fsw, where an 8- to 14-min stop occurred to ensure adequate N2 elimination. Decompression then continued at 2 ft/min from 10 fsw to the surface. All personnel (subjects and experimenters) breathed 100% O2 during decompression from a pressure equivalent to a depth of 50 fsw to the surface.

The blood-gas analyzer was calibrated at the beginning of each surface and pressure session by use of standard calibration gases. The capnograph was calibrated before each period of rest and exercise with gases of known CO2 fraction, from which the partial pressure was determined for each ambient pressure. Calibration gases for blood-gas, gas chromatograph, and capnograph analysis were calibrated by gas chromatography against a single reference gas. Arterial and mouthpiece pressure transducers were calibrated before each period of rest and exercise with an aneroid gauge. The gasometer was calibrated before each experiment and the spirometer and pneumotachometer before each session by use of a 3-liter calibration syringe (standard 3-liter, Collins).

Calculations. VT was determined from measurements of VE and ventilatory frequency (fV) and corrected to BTPS. Mixed expired PCO2 (PeCO2) was determined from the expired gas composition and corrected for water vapor pressure at a body temperature of 37°C. Alveolar ventilation (VA), alveolar O2 tension (PAO2), VO2, and CO2 elimination (VCO2) were determined from standard equations. VDS/VT was determined from simultaneous measurements of PaCO2 and PeCO2 by using the Enghoff modification of the Bohr equation

\[
\frac{V_{DS}}{VT} = \frac{P_{aCO2} - P_{eCO2}}{P_{aCO2}}
\]

PeCO2 was calculated as the mean of all capnograph peaks during the 60 s of expired gas collection and corrected for ambient water vapor pressure.

Statistical analysis. For data that were normally distributed, values are reported as means ± SD. Otherwise, values are median ± interquartile range. To compare surface (1 ATA) and pressure (2.8 ATA) measures, paired Student’s t-test or Wilcoxon’s signed-rank test was used, respectively, depending on whether the data was normally distributed or not. Normality was judged by the Shapiro-Wilk test. Marginal differences between groups Y and O were determined by two-sample t-test or two-sample Wilcoxon rank sum test for continuous data, and by Fisher’s exact test for binary data. Bland-Altman analysis (4) was used to compare the accuracy of PeCO2 as an estimate of PaCO2. Weighted Cohen’s kappa statistic was used to determine agreement between measurements during the fifth and sixth minutes of exercise as confirmation of steady state.

The measurements of a subject’s response under the six different conditions (rest and two levels of exercise at 1 and 2.8 ATA) are necessarily correlated. Thus each of these six measures constitutes a cluster. A mixed-model approach was adopted to account for the variation in the measurements that adjusts for interactions between factors such as age, work rate, and atmospheric pressure.

StatXact 4.1 (Cytel Software, Cambridge, MA) and SAS V8.2 (SAS Institute, Cary, NC) were used to analyze the data. A significant difference was defined as P < 0.05. Parameters at rest and exercise were treated as separate variables; therefore, P values were not corrected for multiple comparisons.

RESULTS

Subjects. Subject characteristics are presented in Table 1. Subjects in group O were heavier on average by 9.5 kg and had a higher past prevalence of smoking (50 vs. 10%); there were no current smokers. Spirometry values (FVC, FEV1, and FEF25–75) are those recorded at the surface; when corrected for height and age, percentage of predicted values was similar between groups.

Work performed. Work rates varied depending on each subject’s level of physical fitness and were identical at 1 and 2.8 ATA in all but two subjects (group O), who performed less work at 2.8 ATA during exercise 2 (Table 1). Percent maximum heart rate was similar at 1 and 2.8 ATA for each exercise, with mean values 73 ± 14 (Y) and 77 ± 11% (O) during exercise 1 and 86 ± 13 (Y) and 96 ± 9% (O) during exercise 2. During both exercises, average work rates were lower in group O, but VO2 was similar between groups.

During exercise at 2.8 ATA, VO2 was higher and VCO2 lower than surface values (Table 2). These differences were small but most pronounced during exercise 2 (change in VO2 of +0.14 l/min, P = 0.0037; change in VCO2 of −0.14 l/min, P < 0.0001). For both measure-
Table 1. Subject characteristics, spirometry values, and work performed

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<th>Age, yr</th>
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<th>Height, cm</th>
<th>Weight, kg</th>
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<th>FVC, liters</th>
<th>FEV1/FVC</th>
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Means ± SD 27.3 ± 6.5

Table 1 continued...

Group Y

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<th>FEV1/FVC</th>
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Means ± SD 63.1 ± 4.9

Group O

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<th>Weight, kg</th>
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<th>FEV1/FVC</th>
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<td>0.80</td>
<td>1.94</td>
<td>480</td>
</tr>
<tr>
<td>26</td>
<td>62</td>
<td>F</td>
<td>191.8</td>
<td>114.3</td>
<td>past</td>
<td>4.92</td>
<td>0.77</td>
<td>3.11</td>
<td>480</td>
</tr>
<tr>
<td>27</td>
<td>63</td>
<td>M</td>
<td>181.6</td>
<td>78.4</td>
<td>past</td>
<td>4.75</td>
<td>0.74</td>
<td>2.52</td>
<td>480</td>
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<tr>
<td>28</td>
<td>64</td>
<td>M</td>
<td>165.1</td>
<td>74.4</td>
<td>none</td>
<td>4.11</td>
<td>0.76</td>
<td>2.36</td>
<td>480</td>
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<tr>
<td>29</td>
<td>69</td>
<td>M</td>
<td>181.6</td>
<td>83.2</td>
<td>none</td>
<td>4.45</td>
<td>0.76</td>
<td>2.78</td>
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<tr>
<td>30</td>
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<td>F</td>
<td>168.3</td>
<td>72.3</td>
<td>past</td>
<td>3.31</td>
<td>0.94</td>
<td>4.35</td>
<td>240</td>
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<td></td>
<td></td>
<td>173.6 ± 13.5</td>
<td>81.7 ± 20.6</td>
<td></td>
<td>756 ± 196.40</td>
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<tr>
<td>Indices</td>
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<td>4.04 ± 0.71</td>
<td>0.80 ± 0.08</td>
<td>3.08 ± 1.28</td>
<td>(732 ± 173.9)※</td>
</tr>
</tbody>
</table>

Means ± SD 63.1 ± 4.9

Forced vital capacity (FVC), ratio of forced expiratory volume in 1 s (FEV1) to FVC (FEV1/FVC), and forced expiratory flow in the middle 50% of exhalation (FEF25–75) are at 1 ATA during rest. Group Y, younger; group O, older; Ex 1, exercise 1; Ex 2, exercise 2. ※Work rates in parentheses were at 2.8 ATA; all other subjects had identical work rates at 1 ATA and 2.8 ATA.
at 1 and 2.8 ATA separately for rest and exercise (Fig. 3), as well as for both conditions combined. Results are reported as means (lower limit of agreement, upper limit of agreement), where the limits of agreement are equal to the mean ± 1.96 SD. During rest and exercise at 1 ATA, $P_{\text{ACO}_2}/P_{\text{ETCO}_2}$ was $-3.73 \pm 9.31, 1.85$ ($P < 0.0001$) and $-5.13$ Torr ($-10.92, 0.65$) ($P < 0.0001$), respectively. During rest and exercise at 2.8 ATA, $P_{\text{ACO}_2}/P_{\text{ETCO}_2}$ was $2.21 \pm 2.04, 6.45$ ($P = 0.0002$) and $-2.46$ Torr ($-9.88, 4.95$) ($P = 0.0089$), respectively.

$P_{\text{ACO}_2}/P_{\text{ETCO}_2}$ exhibited wide variability under all conditions. When all six conditions were combined, the mean difference was $-2.79$ Torr ($-16.37, 10.79$). On average, $P_{\text{ACO}_2}/P_{\text{ETCO}_2}$ was closer to zero at 2.8 ATA only during rest and exercise. During rest, the variability was less at 2.8 ATA than at the surface. However, during exercise, the variability increased. Therefore, we conclude that the reliability of $P_{\text{ETCO}_2}$ as an estimate of $P_{\text{ACO}_2}$ is imperfect under all conditions, but worse during exercise at 2.8 ATA.

### Table 2. Respiratory parameters

<table>
<thead>
<tr>
<th>$P_{\text{ACO}_2}$ Torr</th>
<th>1 ATA</th>
<th>2.8 ATA</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td></td>
<td></td>
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<tr>
<td>Younger</td>
<td></td>
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<tr>
<td>Older</td>
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<tr>
<td>$P_{\text{ACO}<em>2}/P</em>{\text{ETCO}_2}$</td>
<td></td>
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<td>All</td>
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<td>Younger</td>
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<tr>
<td>Older</td>
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</tr>
</tbody>
</table>

Values are means ± SD, unless otherwise indicated. $P_{\text{ACO}_2}$: arterial $P_{O_2}$; $P_{\text{ETCO}_2}$: alveolar $P_{O_2}$; $P_{\text{ETCO}_2}$: end-tidal $P_{CO_2}$; $P_{\text{ACO}_2}$: arterial $P_{CO_2}$; $V_{O_2}$: $O_2$ consumption; $V_{CO_2}$: $CO_2$ elimination; $V_{E}$: minute ventilation; $V_{T}$: tidal volume; $V_{f}$: ventilatory frequency; $V_{D}$: dead space. ^$P < 0.05$ for Y vs. O, adjusted for work rate and pressure; †$P < 0.05$ for all values (Y and O), compared with 1 ATA; ‡$P < 0.001$ for all values (Y and O), compared with 1 ATA; §Data are not normally distributed; values are medians and interquartile range.

Alveolar and arterial $O_2$. After correction for partial pressure of water vapor at 37°C, inspired $P_{O_2}$ ($P_{O_2}$) was 112 Torr at 1 ATA and 383 Torr at 2.8 ATA (equivalent to inspired $O_2$ fraction of 57% at 1 ATA). Accordingly, $P_{O_2}$ and $P_{ACO_2}$ were significantly higher at pressure (Table 2). $P_{O_2}/P_{ACO_2}$ was lower in group O on average by 0.04 ($P = 0.013$). After adjustment for age and work rate, $P_{O_2}/P_{ACO_2}$ was lower at pressure on average by 0.06 ($P < 0.0001$). However, there was a significant interaction between work rate and pressure.
(P = 0.0002), such that the effect of pressure on PaO2/PaO2 depended on work rate and was greatest during rest. There was no functionally significant impairment of O2 exchange at 2.8 ATA, and O2 saturation exceeded 98% in all subjects.

**Pulmonary ventilation and Vd.** Compared with surface values, Ve at pressure was significantly decreased during both exercise 1 (−2.6 l/min BTPS, P = 0.0004) and exercise 2 (−13.1 l/min BTPS, P < 0.0001), caused by decrements in both Ve and VT (Table 2). In contrast, there was a slight but significant increase in Ve at rest of 1.06 l/min (P = 0.0192). Ve was unchanged at pressure during rest and, similar to Ve, reduced during exercise (Fig. 4).

After adjustment for work rate and pressure, Vd was slightly higher in group O during all conditions. Vd was significantly higher at 2.8 ATA in both groups during rest (+0.06 liter BTPS, P = 0.006), exercise 1 (+0.11 liter BTPS, P = 0.0003), and exercise 2 (+0.06 liter BTPS, P = 0.0497). Changes in Vd between 1 and 2.8 ATA were similar between groups (Fig. 5).

During both rest and exercise, Vd/VT was higher at pressure. After adjustment for work rate, changes in Vd/VT were significantly associated with changes in PaCO2-PETCO2 in the same direction (P < 0.0001) (Fig. 6). Pressure significantly increased the slope of this relationship (P = 0.0004).

**Age effects.** In group O, Vd was higher on average by 0.089 liter (P = 0.018), and PaO2/PaO2 was less on average by 0.04 (P = 0.013). After adjustment for work rate and pressure, groups Y and O had no significant differences in the following variables: PaCO2, PETCO2, PaCO2-PETCO2, DH, Ve, VT, Ve, Va, and Vd/VT.

**Gender differences.** Gender differences were not a primary outcome of this study, and, therefore, it was underpowered to detect a significant effect of gender while adjusting for age, work rate, and atmospheric pressure. A simple comparison (t-test) of Vd, Vd/VT, PaCO2, PETCO2, and PaCO2-PETCO2 revealed only slight differences in PaCO2 during exercise 2 at 1 ATA [28.90 ± 2.66 Torr (women), 33.24 ± 4.12 Torr (men), P = 0.014] and PETCO2 during exercise 2 at 1 ATA [32.66 ± 4.90 Torr (women), 38.42 ± 5.97 Torr (men), P = 0.032]. PaCO2-PETCO2 was similar in both groups during this same condition (P = 0.379).

## DISCUSSION

**Physiological Vd.** This study at 18.3 m (gas density 3.1 g/l) reports the first measurements of physiological Vd during rest and exercise at depths <460 m (gas density 12.3 g/l). Even at this moderate depth, Vd significantly increased and caused significant changes in PaCO2, PETCO2, and the relationship between them. These changes were evident during both rest and exercise and were affected little by age.

In a similar study (47) performed at 47 and 66 ATA (gas densities 12.3 and 17.1 g/l), Vd (liters BTPS) was 0.64 at rest and 0.99 during heavy exercise at 66 ATA, with similar values at 47 ATA. Changes in Vd/VT closely followed changes in Vd in both studies. These results suggest a progressively increasing effect of pressure or gas density on Vd during rest and exercise that is present even at 2.8 ATA and reaches a maximum by 47 ATA. Because the direct effects of atmospheric pressure on human physiology are minimal at 2.8 ATA, the increase in Vd in our study is most likely due to the effects of increased gas density on the respiratory system.

Physiological Vd, as measured by the Bohr equation, comprises all components of respiratory Vd-anatomical Vd (the volume of the conducting airways), lung units with ventilation-to-perfusion ratios (VA/Q > 1), and lung units that are ventilated but not perfused (VA/Q = ∞). Anatomical Vd or changes in VA/Q distribution are the only components of physiological Vd likely to contribute to the elevation observed in this study. Intra-alveolar diffusion limitation or a defect in CO2 transport, e.g., altered carbonic anhydrase activity (6, 7),

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**Fig. 1.** Arterial Pa (PaCO2) vs. P CO2 elimination (VCO2) for rest and each exercise level. Values are means ± 95% confidence interval. PaCO2 was elevated at 2.8 ATA during all conditions, and the decrease in PaCO2 during exercise 2 at 1 ATA was less pronounced at 2.8 ATA. *P < 0.01; **P < 0.0001.

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**Fig. 2.** End-tidal PCO2 (PETCO2) vs. PaCO2 at 1 and 2.8 ATA. The line is the line of identity.
could theoretically contribute to an increase in V̇O at extreme atmospheric pressure but would not be expected to contribute to V̇DS at the relatively modest pressure of 2.8 ATA. Hyperoxia and the Haldane effect have also been reported to increase V̇DS by affecting V̇A/Q̇mismatch.

Anatomic V̇DS is elevated by the effect of increased gas density on airways resistance, which consequently favors a higher lung volume (18). End-inspiratory lung volume has been shown to increase during maximal exercise from ~85% of vital capacity to 90% of vital capacity at 3 ATA (18). In a separate study, with an increase in end-inspiratory lung volume from 3.2 to 7.7 liters, anatomic V̇DS increased from 130 to 245 ml (49). By extrapolating the expected increase in anatomic V̇DS on the basis of these data and an average FVC at 1 ATA in this study of 4.48 liters BTPS, the corresponding increase in anatomic V̇DS at 2.8 ATA would be <0.006 liter BTPS. This effect is not large enough to account for >10% of the observed increase in physiological V̇DS.

Hyperoxia has been shown to increase P̄aCO₂ (32, 34) and V̇DS/V̇T (2, 34) in patients with lung disease. Theoretically, elevated P̄IO₂ could alter V̇DS by its effect on V̇A/Q̇ matching, resulting in part from the release of hypoxic pulmonary vasoconstriction and thereby increasing the proportion of lung units with effective shunt (V̇A/Q̇ > 1). However, the effect of hyperoxia on actual V̇A/Q̇ distribution, as measured by the multiple inert-gas elimination technique, is unclear (52, 53), and in healthy subjects there is no significant effect of hyperoxia on P̄aCO₂ or V̇DS/V̇T (3).

Additionally, the Haldane effect contributes to a small elevation in measured V̇DS/V̇T in response to increased P̄O₂ in hypoxemic patients (2, 17, 36). The Haldane effect describes the indirect relationship between CO₂ binding to hemoglobin and O₂ saturation, whereby oxygenated blood at any P̄O₂ has a lower CO₂

Fig. 3. Bland-Altman plots for arterial-to-end-tidal difference (P̄aCO₂-P̄ETCO₂) vs. their average. P̄aCO₂-P̄ETCO₂ demonstrated wide variability at 1 and 2.8 ATA, although the variability was greatest during exercise at 2.8 ATA. See text for further discussion.

Fig. 4. Minute ventilation (V̇E) and alveolar ventilation (V̇A) vs. O₂ consumption (V̇O₂) for rest and each exercise level. Values are means ± 95% confidence interval. At 2.8 ATA, V̇E was slightly higher at rest and decreased during each exercise. Changes in V̇A were similar but reached significance only during exercise. *P < 0.05.
content than deoxygenated blood at the same tension. The importance of this effect is directly related to the severity of the initial hypoxemia and hypercapnia (20, 34, 36), and, whereas the increase in gas density during diving simulates diffuse airway obstruction caused by structural lung disease, divers are neither hypoxic nor hypercapnic at 1 ATA. On the basis of a nomogram developed by Lenfant (34), the estimated elevation in PaCO2 during exercise 2 at 2.8 ATA due to the Haldane effect would be <1 Torr.

The most likely mechanism of increased VDS in response to hyperbaric exposure is a worsening of V/A/Q mismatch. Changes in V/A/Q matching are reflected indirectly by measurements of O2 and CO2 exchange, and dense-gas breathing has been shown to decrease the alveolar-to-arterial O2 gradient (PAO2-PaO2) while at the same time causing an increase in VDS/VT and PaCO2 (39, 46, 47, 57). The detrimental effect of gas density on V/A/Q matching is supported by studies utilizing the multiple inert-gas elimination technique, which assesses distributions of ventilation and perfusion independent of O2 or CO2 analysis (9). A maldistribution of ventilation in response to increased pressure would increase the proportion of lung units with V/A/Q > 1, therefore contributing to an increase in physiological VDS. We, therefore, hypothesize that increased gas density accounts for the majority of the increase in VDS at 2.8 ATA by its detrimental effect on V/A/Q mismatch.

**Pulmonary ventilation.** Salzano et al. (47) report a bimodal ventilatory response to exercise at 47 and 66 ATA that is similar to the findings of this study at 2.8 ATA: increased VE at rest, and decreased VE with heavier exercise. The decrease in VE during exercise at 2.8 ATA is caused by decrements in both Vf and VT, although changes in VT were only significant during exercise 2. VA was unchanged between 1 and 2.8 ATA at rest and decreased only with exercise. The decrease in VA during exercise is caused by both the decrease in VE and an increase in VDS/VT, which is elevated predominantly because of increased VDS. To explain these effects of atmospheric pressure on ventilation, we propose that increased VDS is the primary perturbation that leads to a compensatory increase in VE at rest or light exercise levels, but that inadequate VA results when the respiratory drive inadequately compensates for the increased VDS during heavier exercise.

It is striking that, in the setting of similar elevations at pressure in VDS and VDS/VT across work rates, changes in VE and VA were minimal at rest and profound during the heaviest exercise. The many proposed mechanisms for a decreased ventilatory drive during exercise at depth include 1) a conscious hypoventilation by trained divers (16, 25, 27); 2) a diminished ventilatory response to inhaled CO2 in certain subjects (40), possibly secondary to an acquired adaptation in divers (15); and 3) altered chemosensitivity to CO2 as a result of increased pressure (25) or the effects of increased partial pressures of O2 and N2 (30).

**PaCO2 during rest and exercise.** The reported effects of increased pressure or gas density on resting PaCO2 are variable (46, 47, 50). In this study, resting PaCO2 was normal but slightly higher at 2.8 ATA than at the surface (+1.8 Torr). However, at the surface, PaCO2 was abnormally low (33.8 Torr) and pH was slightly high (7.42), consistent with a mild respiratory alkalosis that may have been due to hyperventilation. The phenomenon of hyperventilation in naive subjects breathing on a mouthpiece has been observed before in this laboratory, especially during rest; this effect usually disappears with exercise. Because surface trials were always performed first, the increase in resting PaCO2 at pressure could simply represent a normalization of an
abnormally low surface value due to an increasing familiarity with the experimental apparatus. However, both Ve and Vds/Vt were slightly higher during rest at 2.8 ATA, and Vco2 was unchanged. In this circumstance, the only variable that increases paco2 is the increase in Vds/Vt.

Changes in respiration and paco2 were seen most clearly with heavier exercise. As expected, there was a decrease from resting values in both pH and paco2 during exercise 2 at the surface, along with a nonlinear increase in Ve, consistent with a mild exercise-induced acidemia. During exercise 2 at pressure, a further drop in pH and increase in paco2 are consistent with the development of a respiratory acidosis superimposed on the underlying metabolic acidemia. Furthermore, a decrease in Ve and increase in Vds/Vt both appear to contribute to the increase in paco2 during exercise. By calculating the expected value of paco2 if Vds/Vt had remained unchanged, 42% of the increase in paco2 during exercise 2 at 2.8 ATA (3.3 of 7.8 Torr) is explained by the decrease in Ve; the remaining difference is attributed to the increase in Vds/Vt.

Paco2-Petco2. Petco2 is a common, noninvasive estimate of paco2, the limitations of which are well known for rest and exercise at 1 ATA across a range of ages (22, 44, 55, 56). During spontaneous breathing at rest, the alveolar Vds fraction usually causes Petco2 to slightly underestimate true alveolar PCO2 (Paco2), and therefore Paco2, in both younger and older subjects (56), producing a positive Paco2-Petco2. During exercise, Petco2 generally overestimates Paco2 because of several factors, including: 1) an increase in average PAO2 due to increased VT, which has the effect of diminishing the contribution of alveolar Vds, 2) increased cardiac output, which increases pulmonary blood flow and lowers the number of alveoli with high Va/Q, thereby increasing Petco2, and 3) increased mixed-venous PCO2, which, during the course of exhalation, can cause a progressive rise in Paco2 and hence Petco2 (13). With increasing age, Petco2 during exercise is closer to Paco2 than it is in exercising younger subjects (56), probably because of the increase in Vds with age. The validity of Petco2 as an estimate of Paco2 under hyperbaric conditions has never previously been assessed.

In this study, the abnormally low values of Paco2, during rest at the surface were associated with higher than expected Petco2, and negative values of Paco2-Petco2. At pressure, this relationship reversed, and Paco2-Petco2 was positive during rest. After excluding any contribution from rebreathing, which can cause elevations in Petco2 above Paco2 (45), we considered the possibility that the unexpectedly negative Paco2-Petco2 at 1 ATA during rest may have been due to the observed slight respiratory alkalosis, caused by an unconscious hyperventilation. During hyperventilation, Petco2 may rise above Paco2 because of the lack of steady-state equilibrium and increased excretion of CO2. The elevation in VT that often accompanies hyperventilation favors this effect by promoting intrapulmonary gas mixing and lowering Vds/Vt, thereby lessening the contribution of the baseline alveolar Vds to Petco2. In support of this proposed effect of hyperventilation, we observed that, when Paco2-Petco2 was plotted against Paco2 for all subjects during rest at the surface, Paco2-Petco2 became increasingly negative as Paco2 decreased. There was no evidence for this effect during exercise or at pressure. The hypothesis that hyperventilation lowers Paco2-Petco2, was further supported experimentally during a separate session in a single subject who consciously hyperventilated at rest for 11 min. A Petco2 of 25 Torr was maintained by visual feedback from the capnograph, with all variables measured as during the experimental setup. Paco2-Petco2 was lowest during acute hyperventilation (–8.5 to –9.4 Torr) when Paco2 was also at a minimum (20.8 to 18.5 Torr). As discussed above, we suspect that the observed modest hyperventilation during rest at the surface was a product of unfamiliarity with the experimental setup that may have lessened during pressure trials. Therefore, we can make no conclusions about the effect of hyperbaric exposure on Paco2-Petco2 during rest.

Paco2-Petco2 during exercise at 1 ATA was consistent with previously published values (23, 55), becoming more negative compared with resting values as Petco2 rose and Paco2 decreased. At 2.8 ATA, Paco2-Petco2 was higher than at the surface. Changes in Paco2-Petco2 at pressure can be explained by the direct relationship between Paco2-Petco2 and Vds/Vt (Fig. 8). Just as the rise in Petco2 during exercise is partly explained by the accompanying decrease in Vds/Vt, the elevation of Vds/Vt at pressure can be expected to lower Petco2. Therefore, on average the increase in Vds/Vt at pressure reduced the magnitude by which Petco2 overestimated Paco2 during exercise.

Interestingly, increased pressure altered the relationship between Paco2-Petco2, and Vds/Vt, such that changes in Vds/Vt had more of an effect at 2.8 ATA, especially during rest (Fig. 6). This phenomenon is consistent with an increase in Va/Q mismatch at higher gas densities, with a greater proportion of lung units with Va/Q > 1 contributing to Vds/Vt. The slopes converge during exercise, which may reflect the decreasing contribution of Vds/Vt to Paco2-Petco2 relative to the effect of the increasing mixed-venous PCO2.

Although it is tempting to conclude that Petco2 more accurately reflects Paco2 under hyperbaric conditions, the wide variability of Paco2-Petco2 makes capnography an imprecise way to assess hypercapnia at depth. Large differences between Paco2 and Petco2 existed even at the surface, and at 2.8 ATA, Petco2 still overestimated Paco2 by 9–11 Torr in two subjects. Furthermore, elevated Petco2 did not predict arterial hypercapnia. In the two instances of Petco2 greater than 50 Torr, Paco2 did not exceed 45 Torr. These results were obtained in the setting of a relatively modest increase in gas density during moderate exercise; under more extreme conditions, the relationship between Petco2 and Paco2 may be even less certain. However, it is also important to emphasize that, in many diving studies, extreme elevations of Petco2 have been strongly corre-
lated with symptoms consistent with hypercapnia (29, 37, 40), even though PaCO₂ was not directly measured. Further studies in divers to correlate PETCO₂ with PaCO₂ would be useful, given the demonstrated variability of PETCO₂ under the conditions that are most relevant to working divers, i.e., exercise.

V̇O₂ and V̇CO₂. For a given work rate, V̇O₂ tended to be higher at pressure than at 1 ATA and V̇CO₂ tended to be lower, although these differences were small even at the highest work rates.

It is possible that V̇O₂ may have been elevated by the increased work of breathing at pressure, whereas V̇CO₂ may have been lowered by hypventilation and the consequent accumulation of CO₂. The conclusions of this study would not be altered by small perturbations of V̇O₂ and V̇CO₂.

Differences between older and younger subjects. As expected, V̇Ds was slightly higher in group O during all conditions. The higher V̇Ds was not associated with significantly higher values of V̇Ds/VT or PaCO₂ for group O during exercise or at pressure, nor was there a greater relative increase in V̇Ds at pressure. PaCO₂-PETCO₂ was similar in both groups under all conditions. The only other difference between groups was a slight but functionally insignificant decrease in PaO₂/PaO₂ in group O. The excellent performance of group O on every other measure of lung function and exercise ability make it unlikely that older divers in good health would experience significantly greater increases in PaCO₂ at 2.8 ATA, or that PETCO₂ would be less accurate in this particular age group.

In conclusion, rather than being an isolated phenomenon of extreme hyperbaric exposure, changes in physiological V̇Ds were apparent during both rest and exercise even at 2.8 ATA. The increase in V̇Ds most likely reflects changes in V̇A/Q matching due to the increased gas density, even though the increase in gas density was not large enough to cause dyspnea or discomfort. Because of an inadequate ventilatory compensation for an increase in physiological V̇Ds, PaCO₂ increased during rest and exercise. Significant arterial hypercapnia did not occur, even during the heaviest exercise.

The increase in V̇Ds/VT at 2.8 ATA contributed to a decrease in the expected elevation of PETCO₂ above PaCO₂ during exercise. Consequently, the absolute PaCO₂-PETCO₂ difference was less during exercise at 2.8 ATA. However, large intersubject variability at both 1 and 2.8 ATA makes PETCO₂ an imperfect estimate of PaCO₂ for monitoring individual divers.

Finally, this study in a dry hyperbaric chamber does not support the hypothesis that older divers in good physical condition with no underlying pulmonary or cardiovascular disease are at any increased risk of respiratory impairment during moderate exercise at 2.8 ATA.

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REFERENCES


