Undersea Biomedical Research, Vol. 10, No. 3, September 1983

Influence of exercise on maximal voluntary ventilation and forced expiratory flow at depth

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Hickey DD, Lundgren CEG, Påsche AJ. Influence of exercise on maximal voluntary ventilation and forced expiratory flow at depth. Undersea Biomed Res 1983; 10(3):241–254.—Four to six subjects performed maximal voluntary ventilation (MVV) and forced expirations during rest, exercise (50, 125, and 200 W), and inhalation of air and CO_2 and air at rest while submerged at pressures of 1.45, 2.82, 4.64, and 6.76 atm. Maximal expiratory flow (at 40% of vital capacity) and MVV at rest decreased as exponential functions of gas density, but the decrease was less than in some earlier studies. Independent of pressure, MVV increased by about 10%–17% at the heavier work loads and expiratory flow increased by 27%–48%; the increase in expiratory flow disappeared within 2 min after exercise. Exercise increased end-tidal CO_2 tension by up to 9 mmHg. Carbon dioxide inhalation increasing the end-tidal level by up to about 25 mmHg during rest had no effect on MVV and a slight to moderate effect on flow, increasing it by a maximum of 21% at 4.64 atm. The enhancing effect of exercise on MVV and expiratory flow at depth apparently was mainly due to modified autonomic nervous activity reducing pulmonary flow resistance, CO_2 accumulation playing an uncertain role, and passive distension of airways playing no role.

carbon dioxide
exercise
expiratory flow
gas density
immersion
maximal oxygen uptake
maximal voluntary ventilation
pressure
respiratory mechanics
respiratory resistance
submersion
ventilatory capacity

We were prompted to undertake this study by earlier occasional observations in exercising subjects who, at increased atmospheric pressures would exhibit ventilation levels that approached or even exceeded their maximum voluntary ventilation (MVV) levels during resting conditions (1, 2). It is known that exercise may enhance the ventilatory capacity at 1.0 atm as measured by for instance MVV, the suggested mechanism for this being a reduction in airway resistance brought about by autonomic control and/or carbon dioxide or other chemical factors (3, 4).

A diver's ventilatory situation at depth may be different than at 1.0 atm due to differences in breathing gas density and other pressure and immersion effects. Therefore this investigation

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was focused on the effects of exercise on the ventilatory capacity in simulated dives. The study included inhalation of carbon dioxide-enriched air to reproduce possible effects of carbon dioxide accumulation during exercise at depth. The experiments were performed in air-breathing subjects at simulated depths down to 57.6 m (6.76 atm). In part of the experiments the subjects were submersed in order to create a situation close to real in-sea conditions.

MATERIALS AND METHODS

For different parts of the study between 3 and 6 volunteer subjects were used. They were nonsmoking males between 21 and 30 years of age with prior scuba diving experience. They had passed physical examinations for diving, including pulmonary roentgenographs, and they had no relevant history of pulmonary disease.

The experimental apparatus has been described in detail elsewhere (5). Briefly, it consists of a pressure chamber with a wet compartment allowing intrapulmonary pressure to be set as desired relative to water pressure at the pressure centroid of the chest, which was determined by Paton and Sand (6) to be a point approximately 13 cm below the sternal notch. The location of the centroid, by definition, is such that if breathing gas pressure is adjusted to be the same as water pressure at this point, a normal functional residual capacity is preserved. In the present study breathing gas pressure was always kept the same as water pressure at the centroid. A small rubber balloon secured to the subject's chest at the pressure centroid and connected with a pressure transducer allowed careful control of the water pressure relative to mouth pressure (breathing gas pressure). The chamber pressure was 1.45, 2.82, 4.64, or 6.76 atm (accuracy better than ± 0.1%). The reason for selecting 1.45 atm as the lowest pressure was because that value was required for the proper function of the gas sampling system. Measurements of MVV and ventilation at maximal exertion ($\dot{V}_E$,W_{max}) were also made at 1.0 atm. Water temperature was maintained between 30°C and 32°C for immersed experiments. The subjects wore only swimming trunks.

A bag-in-box system coupled to a rolling-seal spirometer (Spirometer 822, Ohio Medical Products, Madison, WI) allowed monitoring of the diver's breathing frequency, tidal volume, expiratory reserve volume, and vital capacity. To reduce the impedance of the spirometer in high density gas the internal diameter of its hose connection had been increased to 58 mm, providing a 65% larger cross-sectional opening area than that of the standard instrument (cf. Ref. 7). The breathing resistance of the entire system was less than 1.25 $cmH_2O \cdot liter^{-1} \cdot s$ at a flow of 5 liters/s at 8 g/liter air density. The volume signal from the spirometer was differentiated for display of flow.

The subject was connected to the breathing system via a full-face mask with an oronasal mask, which, including the attached breathing valves, had a dead space of 150 cm^3.

Analysis of inhaled and exhaled gas was made with a Perkin-Elmer MGA 1100 Mass Spectrometer (Aerospace Division, Perkin-Elmer Corp., Pomona, CA), modified and rigorously calibrated to yield an accuracy of ± 0.03%. Sampling was made in the valve connection to the oronasal mask.

The MVV volumes were computed from the spirometer tracings, rather than deriving them from the gas volumes collected during MVV maneuvers, because of the possibility that inertial gas movement would increase the gas volumes delivered to the bag (8). All data were recorded on an 8-channel polygraph. The subject sat upright in a seat to which he was fixed by straps arranged so as not to impede chest and leg movements. Leg exercise was performed on an electrically braked ergometer modified for underwater use (5). The breathing gas was either compressed air or various mixtures of carbon dioxide in air (see *uppermost panel* in Fig. 2).

All breathing gases had been moisturized at ambient pressure and temperature (27°C–28°C) before inhalation.

The MVV maneuvers were performed for 15 s with free breathing frequency and tidal volume. Forced expired vital capacity (FEVC) maneuvers were made to obtain maximal expiratory flow at a lung volume equal to residual volume plus 40% of vital capacity ($\dot{V}_{E_{max}}$,0.4 VC). The reason for calculation of the flow at a lung volume of 0.4 VC was that the flow at this volume is effort independent (9) and therefore determined by pulmonary mechanics. The subjects had all practiced the maneuvers repeatedly over several days before the actual experiments. Because order effects and day-to-day variations have been shown to occur in measurements of nonelastic pulmonary resistance (10), only one experiment per day was performed in each subject and a control measurement was made as a part of each experiment. The order in which the experiments at the different pressures were performed varied randomly.

Separately, the ($\dot{V}_E$,W_{max} was recorded in connection with measurements of maximal oxygen uptake ($\dot{V}_{O_{2max}}$), the latter being done as described by Åstrand and Rodahl (11). These measurements were performed both during submersion and nonsubmersion at 1.00, 1.45, 4.64, and 6.76 atm. At 1.0 atm the maximal exercise performance ranged from 200 to 275 W with $\dot{V}_{O_{2max}}$ values from 2.6 to 3.6 liters/min.

EXPERIMENTAL PROCEDURES

After compression in the chamber to the desired pressure, the subject, wearing the face mask, entered the water and strapped himself to the seat. The hydrostatic pressure at the pressure centroid of the chest was adjusted so as to equal breathing gas pressure, i.e., chamber gas pressure.

While resting, the subject carried out a series of respiratory maneuvers, consisting of VC, FEVC, MVV, VC, and FEVC and requiring a total of about 1.5 min. Following this, leg exercise was performed at either 50, 125, or 200 W (including hydrodynamic work, cf. Ref. 2) during which the series of respiratory maneuvers was repeated. Measurements were made at 1.00, 1.45, 2.82, 4.64, and 6.76 atm; not all respiratory maneuvers and work loads were used at all pressures. The application of the three different work loads was made according to somewhat differing protocols as dictated by another study running in parallel with the present one. These protocols are presented in more detail:

50 W: Following the respiratory maneuvers at rest, exercise was begun, and 5 min later, while still exercising, the subject repeated the respiratory maneuvers.

125 W: Three 9-min exercise periods with loads of 75, 125, and 175 W and separated by 2-min rest intervals were used. Immediately following the 175-W period the work load was lowered to 125 W, 2 min into which the respiratory maneuvers were repeated.

200 W: Work was first performed at 100 W for 3 min immediately followed by a 200-W load, 4 min into which the respiratory maneuvers were repeated.

To investigate possible "off-effects" of exercise, three subjects repeated the FEVC maneuver 3 times at 2-min intervals after termination of the 125-W load. This was done at 1.45 and 2.82 atm.

In the experiments testing for CO_2 effects, two CO_2 mixtures were chosen for each depth so as to match or exceed the end-tidal CO_2 concentrations observed during exercise. The control measurements were first performed during resting conditions and air breathing. This was followed by open-circuit breathing of the gas mixture with the lower CO_2 concentration for 5 min, after which (while still on the gas mixture) the subject performed the standard array of breathing maneuvers. The mixture with the higher CO_2 content was then introduced, and 5

min later the measurements were repeated. This series was performed at 1.45 and 4.64 atm (5 subjects) and 6.76 atm (3 subjects). For safety reasons, subjects were not immersed during the CO_2 inhalation experiments at 6.76 atm.

RESULTS

Mean values of MVV as influenced by pressure and exercise are plotted in Fig. 1A, which for comparison also includes an adaptation of Lanphier's (12) graphical presentation of results from several other laboratories. The latter results were all obtained in nonsubmersed resting subjects, whereas our recordings were from fully submersed subjects. Increased air pressure depressed MVV although to a somewhat lower degree than in earlier studies, as is discussed later. The normalized, resting MVV values were 92% ± 11% (SE) at 1.45 atm, 79% ± 3% (SE) at 2.82 atm, 68% ± 3% (SE) at 4.64 atm, and 61% ± 10% (SE) at 6.76 atm. The most striking finding, however, was that exercise markedly increased MVV, as illustrated by the data points from measurements at work loads of 125 and 200 W. For comparison, Fig. 1A also shows the ventilation levels reached during maximal exertion and submersion.

Similar effects of high air pressures and exercise as on MVV were exerted on maximal expiratory flows ($\dot{V}_{E_{max}}$, 0.4 VC) measured at a lung volume of 40% of VC during FEVC maneuvers, as shown in Fig. 1*B*.

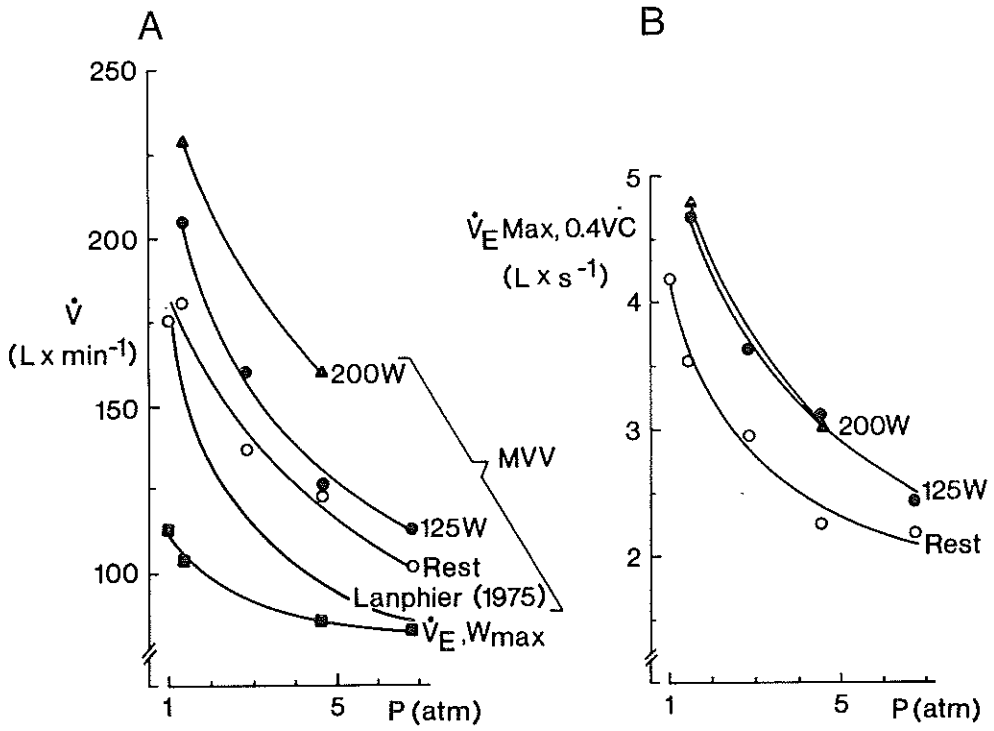

Fig. 1. *A:* Maximal voluntary ventilation (MVV) at increased ambient pressure (P) in submerged air breathing subjects (means from 5 subjects) at rest and during leg exercise at 125 and 200 W. The MVV curve, modified from Lanphier (12), is from four different studies in resting, nonimmersed subjects. The $\dot{V}_{E,W_{max}}$ curve represents mean spontaneous ventilation during maximal exertion in 5 subjects. *B:* Maximal expiratory flow at a lung volume of 40% of vital capacity ($\dot{V}_{E_{max}}$, 0.4 VC) under the same conditions as MVV measurements in *A*.

In Fig. 2 mean values and standard errors based on all individual values are presented. These means (with the exception of the mean of the end-tidal CO_2 tensions, or $P_{ET_{CO_2}}$) were calculated from data normalized for each subject relative to his resting results. Using a test based on the relationship between order statistics and empirical distribution, the data were determined to be normally distributed, and statistical evaluation was made by paired comparisons (t test). Comparisons between results from various exercise levels were based on mean values (t test). Relevant results of these comparisons are displayed in Fig. 2. All comparisons in this text refer to significant differences ($P<0.05$) unless otherwise stated.

While exercise at 50 W had no effect, exercise at 125 W increased the MVV, and even more so at 200 W, and this occurred to the same relative degree at all pressure levels. Thus at 1.45 atm, and with the 125-W work load, the MVV was 114% ± 5% (SE) of the resting value, and with 200 W it was 115% ± 4% (SE). The corresponding values at 2.82 atm were 115% ± 2%

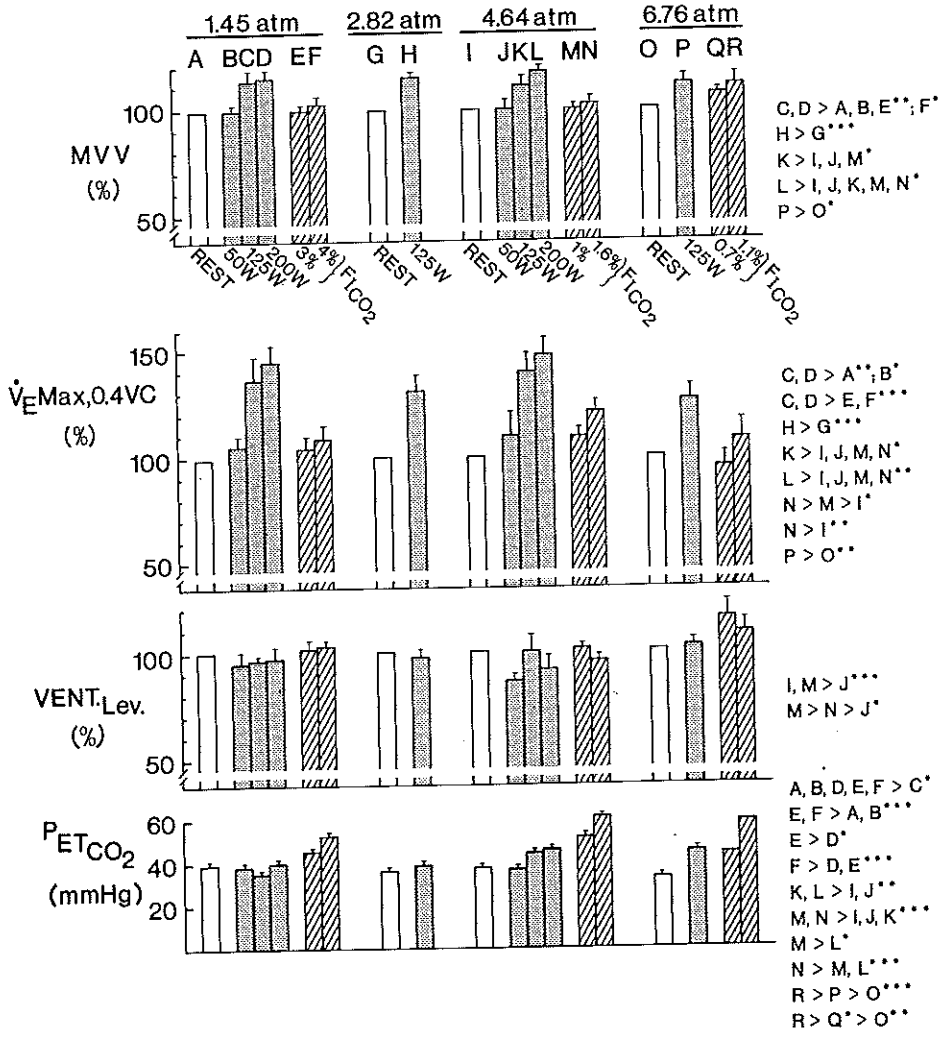

Fig. 2. MVV, $\dot{V}_{E_{max}}$, 0.4 VC, mean ventilatory level and pre-MVV end-tidal carbon dioxide tensions ($P_{ET_{CO_2}}$) at increased ambient pressure, and exercise or inhalation of mixtures of carbon dioxide and air. Mean values and standard errors (normalized to resting condition) from 4 or 6 subjects. Levels of significance: *$P<0.05$; **$P<0.01$; ***$P<0.005$.

(SE) (125 W only) and at 4.64 atm, 111% ± 4% (SE) and 117% ± 2% (SE), respectively. Only the 125-W work load was used at 6.76 atm, and the MVV was 110% ± 4% (SE).

The changes in expiratory flows were qualitatively the same as the MVV changes but more marked: at 1.45 atm and 125 W the flow was 136% ± 11% (SE) of the resting value and at 200 W, 144% ± 8% (SE); at 2.82 atm and 125 W the flow was 131% ± 7% (SE); at 4.64 atm, 140% ± 9% (SE) and 148% ± 11% (SE) for 125 W and 200 W, respectively; at 6.76 atm and 125 W it was 127% ± 8% (SE).

The mean ventilatory levels (expiratory reserve volume + 0.5 tidal volume) during rest at different pressures were normalized to the 1.0-atm values. They were 107% ± 5% (SE) at 1.45 atm, 102% ± 5% (SE) at 2.82 atm, 110% ± 9% (SE) at 4.64 atm and 67% ± 13% (SE) at 6.76 atm, and they did not differ significantly from the control values. Furthermore, the mean ventilatory levels were not changed by exercise (Fig. 2) except for a reduction at 4.64 atm and 50 W to 86% ± 2% (SE). Similarly, the expiratory reserve volumes during MVV at rest (not shown in Fig. 2) did not differ significantly between 1.0 atm and increased pressure, but the scatter in the data was large. The values were 133% ± 19% (SE) at 1.45 atm, 131% ± 15% (SE) at 2.82 atm, 166% ± 30% (SE) at 4.64 atm, and 172% ± 52% (SE) at 6.76 atm. The expiratory reserve volumes were not changed by exercise except for reductions to 86% ± 6% (SE) at 1.45 atm and 125 W, 88% ± 7% (SE) at 2.82 atm and 125 W, 81% ± 3% (SE) and 76% ± 8% (SE) at 4.64 atm and 50 W and 200 W, respectively.

The end-tidal carbon dioxide levels graphed in Fig. 2 were obtained during the steady state immediately preceding the respiratory measurement maneuvers. Those levels increased from an average of 36 mmHg at rest to about 45 mmHg with exercise, but only at the highest pressures (4.64 and 6.76 atm). As expected, carbon dioxide inhalation did increase the end-tidal concentrations; the highest level reached at 1.45 atm was 53 mmHg, at 4.64 atm it was 61 mmHg, and at 6.76 atm it was 60 mmHg (Fig. 2).

In contrast to exercise, inhalation of carbon dioxide-enriched air alone did not influence MVV at any pressure. The effects of carbon dioxide inhalation on $\dot{V}_{E_{max}}$, 0.4 VC were generally less than the effects of exercise. Thus, with 4% carbon dioxide at 1.45 atm, the flow was not different from the resting value; with the 1% mixture at 4.64 atm, it was 109% ± 4% (SE); with 1.6% carbon dioxide it was 121% ± 5% (SE); the carbon dioxide mixtures had no effect at 6.76 atm. The mixtures caused no changes in the mean ventilatory levels.

The results of $\dot{V}_{E_{max}}$, 0.4 VC measurements during and after exercise at 125 W are shown in Fig. 3. Because we had found that the influence of exercise was independent of pressure (Fig. 2), the data from a total of 6 experiments in 3 subjects at 1.45 and 2.82 atm were pooled. Evidently the enhancing effect of exercise on expiratory flow had already disappeared after 2 min. Expiratory flow 6 min after the exercise was actually about 15% lower than the flow obtained before the exercise.

The resting MVV maneuvers that were performed during submersion did not differ significantly from those obtained during nonsubmersion (Fig. 4*A*). The relationships between the ventilation at maximal exertion ($\dot{V}_E$, W_{max}) and MVV at rest and 200 W are illustrated in Fig. 4*B*. There were slight increases in exercise ventilation relative to MVV as pressure increased from 1.45 to 4.64 atm but no further change at 6.76 atm (only resting MVV was recorded).

DISCUSSION

High pressures had less of a depressant effect on maximal expiratory flows and resting MVV (Fig. 1*A*) than reported by some other authors. The maximal expiratory flow at lung volumes larger than 25% of VC has been shown by Wood and Bryan (13) to depend on the gas density

raised to an exponent of about −0.45, a figure that was confirmed by Hesser and co-workers (14). A computation of the density exponent from our flow measurements at 40% of VC yielded lower values: namely, −0.44 at 1.45 atm, −0.34 at 2.82 atm, −0.40 at 4.64 atm, and −0.31 at 6.76 atm—i.e., a mean of −0.37 ± 0.06 (SD).

If these values reflect less dependence of maximal expiratory flow on gas density, it may be inferred that the flow in the airways of our subjects would be more laminar and exhibit a lower amount of turbulence and convective acceleration connected with pressure losses related to gas density (cf., Ref. 13). It is possible, however, that the differences in results may be due to methodological differences, since pneumotachography was used in the two other investigations, whereas the present study employed the time derivative of a spirometric volume signal for flow measurements. Notwithstanding this difference, our resting MVV values, like those obtained by Hesser et al. (14), were larger at all pressures than what may be predicted based on the MVV at 1.0 atm and the density exponents (−0.45 and −0.37, respectively) derived from maximal expiratory flow measurements in the two studies. Thus a comparison in the present study of predicted and measured MVV values yields 87% vs. 102% at 1.45 atm, 68% vs. 79% at 2.82 atm, 57% vs. 68% at 4.64 atm, and 49% predicted vs. 61% observed at 6.76 atm. The difference between predicted and observed by 11 to 15 percentage points is in fair agreement with the differences of 7 to 9 percentage points reported by Hesser et al. (14). The mechanism behind these higher-than-predicted MVV values appears to be that expiratory flow is higher at depth than would be predicted from measurements at 1.0 atm. One explanation favored by Hesser et al. (14) is that expiratory reserve and mid-expiratory volumes may have increased. Thus the breaths would occur at a higher position on the VC range that is compatible with higher expiratory flows. However, while our measurements of resting expiratory reserve volume and mean ventilatory levels indicated no significant changes with pressure, a firm statement, based on our data, cannot be made about the feasibility of this mechanism. This is because of the considerable scatter in the expiratory reserve volume data due to variations in tidal volumes between individuals (mean individual ventilatory levels remaining stable).

The other explanation, also proposed by Hesser et al. (14), takes into consideration the fact that carbon dioxide in the hypocapnic range has been shown to influence pulmonary resistance (15). Because MVV is depressed at depth, less alveolar hypocapnia would develop and thus

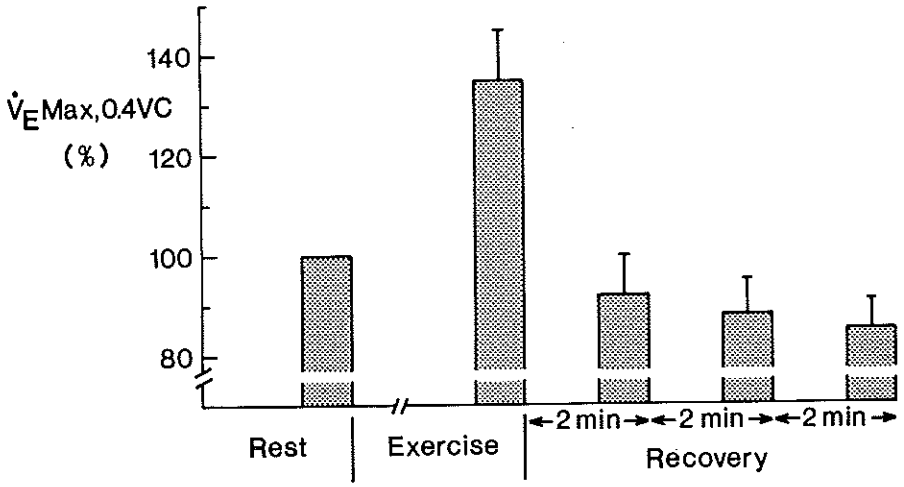

Fig. 3. Normalized $\dot{V}_{E_{max}}$, 0.4 VC values in 3 subjects before, during, and 2, 4, and 6 min after exercise. Means and standard errors of pooled data from measurements at 1.45 and 2.8 atm are shown.

there would be less increase in pulmonary resistance, allowing the MVV to be performed under more favorable flow conditions. It is quite possible that such a mechanism may have contributed to the higher-than-predicted resting MVV values at high pressures in the present study.

In this study 125–200 W exercise at pressures between 1.45 and 6.76 atm caused a pressure-independent increase of 10%–17% in the free-frequency 15-s MVV and the expiratory flow (at 0.4 VC) increased by 27% to 48%. These results, partially presented in an earlier preliminary report (16), were obtained in submersed subjects by spirometry. They are in excellent agreement with the results in nonsubmersed subjects studied with pneumotachography and reported on the same occasion by Hesser et al. (14). The latter authors observed increases in MVV of 11%–15% in response to heavy exercise (270–310 W) at pressures between 1.0 and 6.0 bar. These effects of exercise are equal to or larger than those observed in three other studies at 1.0 atm. Thus it may be inferred from the results of Lewis and Morton (3) that light exercise caused a 6.5% increase in maximal breathing capacity measured after the work. Likewise,

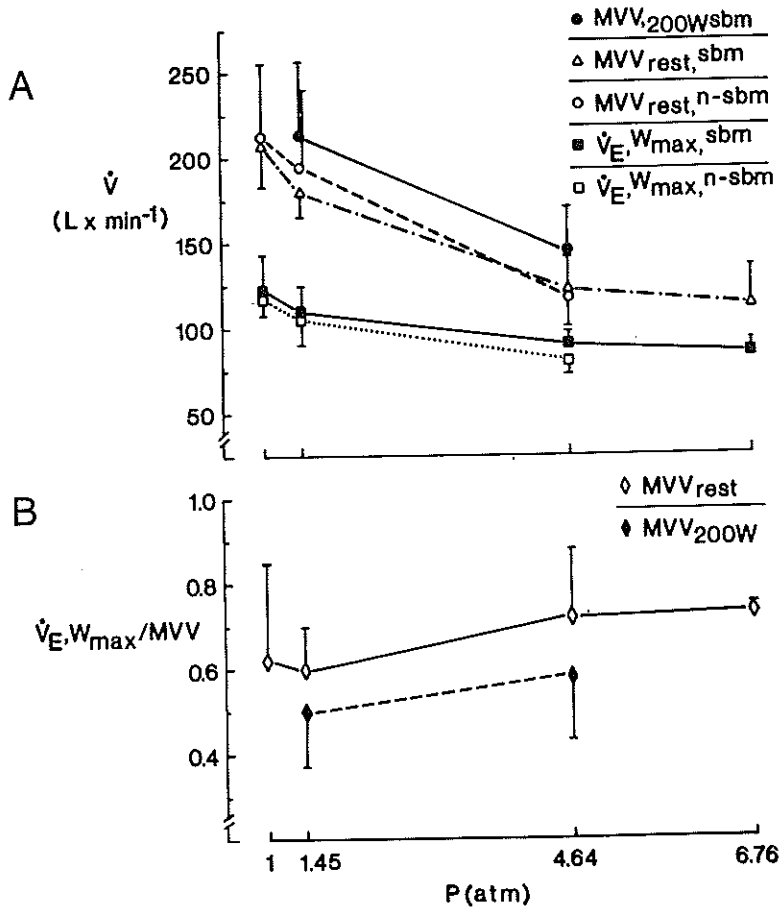

Fig. 4. *A:* Ventilation ($\dot{V}$) vs. pressure attained during MVV (rest and submaximal exercise) and during spontaneous ventilation (maximal exercise). Results from both submersion (sbm) and nonsubmersion (n-sbm) are shown. *B:* Ratio between $\dot{V}_{E,W_{max}}$ and MVV_{rest} and MVV during submaximal exercise vs. pressure. Data points in *A* and *B* are means ± SD from 4 or 5 subjects calculated on mean values from several measurements in each subject.

Shephard's (4) results show an increase in MVV of 14% in the final minute of a 5-min period of moderate work. However, moderate work for 20 min was connected with a 3.5% decrease in MVV. Lefcoe (17) applied forced expiratory vital capacity maneuvers during and after moderately heavy exercise to deduce FEVC 1.0 and maximum mid-expiratory flow. The latter index, being the most sensitive, showed increases during work that may be calculated to about 25% and returned to control values in 2–4 min. Kagawa and Kerr (10) found increases of between 12%–20% in specific airway conductance in subjects performing light to heavy exercise, and these changes had normalized in about 3 min.

Possible explanations for the increase in MVV during exercise include several factors that may influence flow resistance and also optimize (i.e., minimize) the combined flow resistive and elastic respiratory work. One consideration is that a change in breathing frequency, by itself, may alter MVV. It has been shown that MVV at 1.0 atm increases with respiratory frequency up to about 80 breaths per min while it is relatively stable at higher frequencies (18). A similar relationship held true in this study. This was ascertained by relating varying breathing frequencies in repeated runs with corresponding resting MVV values in each subject at each depth. Regression analysis of 19 sets of paired data revealed positive slopes for all but 3 (2 at 2.82 atm and 1 at 6.76 atm). The MVV breathing frequencies used during all exercise levels were generally lower than the MVV frequencies during rest. This is exemplified by the MVV data obtained with 125 W in Fig. 5. This tendency was more marked as pressure increased. Therefore, any frequency effect on MVV in the present experiments must have run contrary

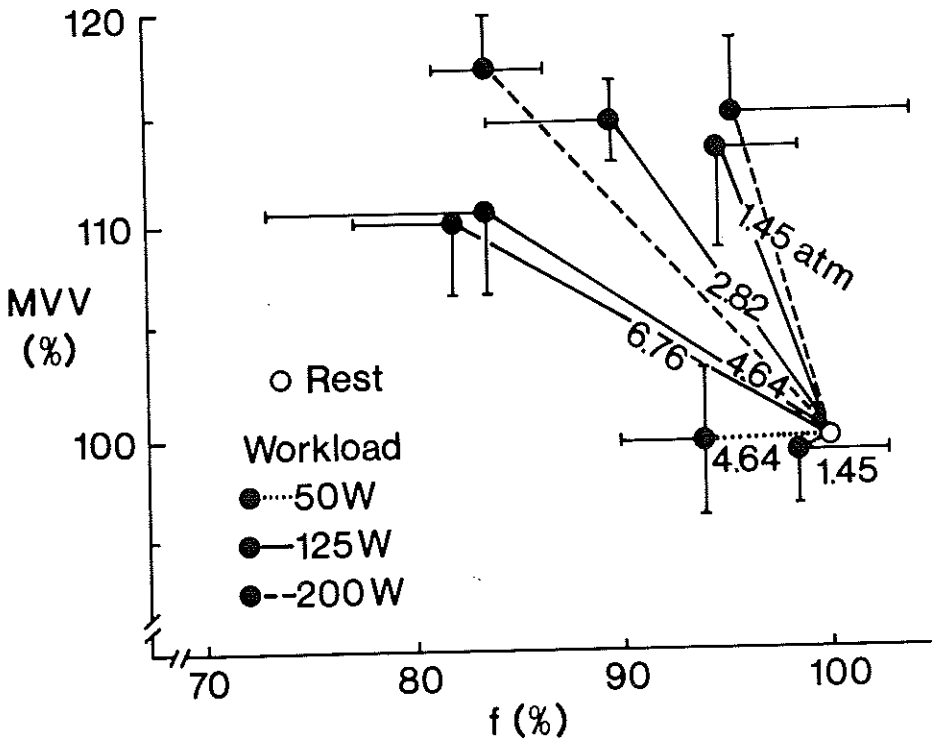

Fig. 5. Values of MVV and f (breathing frequency) during exercise at 50, 125, and 200 W while at 1.45, 2.88, 4.64, and 6.76 atm normalized to resting values of MVV and f at the respective pressures. Data points are mean values ± SE from measurements of 3–5 subjects.

to the observed enhancing effect of exercise on MVV, and it may be speculated that had the breathing frequency been unchanged, the exercise-induced changes in MVV might have become even more marked, especially with increasing pressure.

As far as gas flow resistance is concerned, the possibility must be considered that exercise may induce lung distension, which in its turn may cause distension of the airways and thus a lowered resistance. This explanation has been favored by Hesser et al. (14). In our measurements, however, the mean ventilatory levels remained remarkably stable throughout all types of experiments, and this speaks against passive changes in flow resistance. This interpretation of our data depends on the assumption that the residual volume remained unchanged in the different experiments. That this is a valid assumption gains some indirect support from the observation that there were no reductions and even a tendency to increases in vital capacities measured during exercise as compared with rest. In addition, Kagawa and Kerr (10) have reported observations indicating that exercise-induced increases in airway conductance occur over the entire vital capacity span.

The possibility should be considered that exercise may alter autonomic nervous activity and/or may increase catecholamine release so as to reduce airway resistance and allow for the increases in MVV and flow. It seems unlikely that exercise would enhance airway conductance by catecholamine release, for Kagawa and Kerr (10) have shown that administration of a beta blocker (propranolol) would not prevent the exercise effect. By contrast atropine did, which would indicate that the differences in airway conductance between rest and exercise are due to differences in parasympathetic tone.

The notion that exercise influences pulmonary flow conditions by changes in autonomic control of bronchomotor tone gains further support from observations that exercise effects may subside too rapidly to be commensurate with hormonal effects. Studies of catecholamine response to exercise at 125 W (19, 20) indicate that the catecholamine levels still remain elevated 3–5 min after cessation of exercise. Nevertheless, the increase in maximal expiratory flow (at 0.4 VC) had disappeared within 2 min after exercise in 3 of our subjects who were studied in this regard (Fig. 3). Likewise, a 25% increase in maximum mid-expiratory flow recorded by Lefcoe (17), as well as the 12%–20% conductance increase in Kagawa and Kerr's (10) subjects, had returned to control levels within 4–5 min. It is interesting to note that in the present study, one naive subject commented that he could feel that he attained higher expiratory flows during exercise, but that this was not so 2 min after the exercise.

For the sake of completeness it should be mentioned that other factors could have potentially changed during exercise so as to enhance the MVV. Increased muscular performance during inspiration and during the effort-dependent part of the expiration would be such a factor. It probably only played a minor role, however, since the flow enhancement during the effort-independent phase of the FEVC maneuvers was marked. Reduced flow resistance probably could account for most of the increase in MVV.

Increased pulmonary elastic recoil lowering pleural pressure, thereby inducing airway distension, is another factor that may enhance expiration in its entirety. Although Kagawa and Kerr (10) did record a slight increase in pulmonary elastic recoil during exercise, they concluded that it was too small to account for the observed changes in airway conductance.

Whether carbon dioxide may influence airway resistance is of particular relevance in the hyperbaric environment where exercise may induce considerable hypercapnia (12). This is in contrast to the conditions at 1.0 atm where moderate exercise has only a very slight effect, if any, and where heavy exercise may induce arterial hypocapnia (21). Hesser et al. (14) have indeed speculated that hypercapnia during exercise at depth may be a major factor for increasing airway conductance and thus MVV.

Our findings do not support that view. In our experiments at 1.45 and 2.82 atm there were no changes in end-tidal carbon dioxide levels at the work loads between 50 and 200 W, whereas at 4.64 atm both the 125- and 200-W work loads were accompanied by an increase in end-tidal carbon dioxide tension of 6.8 mmHg. Similarly, it was increased by 13.7 mmHg by exercise (125 W) at 6.76 atm. Despite the increases in end-tidal carbon dioxide during exercise at 4.64 and 6.76 atm, the relative increases in MVV and $\dot{V}_{E_{max}}$, 0.4 VC were the same as at the lower pressures with normocapnia. These observations indicate that carbon dioxide was not a major factor behind the exercise effects. Further support for this notion can be gathered from the experiments with inhalation of carbon dioxide-enriched air. To do so it is, however, first necessary to consider the possibility that the hyperventilation during the 15-s MVV maneuver caused some alveolar hypocapnia (the level of which could not be recorded because of too-long a mass spectrometer response time). This hypocapnia most likely would place the end-tidal carbon dioxide tension in the low end of the range between 20 and 50 mmHg within which Newhouse et al. (15) have shown pulmonary resistance to increase as $P_{ET_{CO_2}}$ is reduced. As a consequence, MVV might have been depressed because of hypocapnia in our resting control experiments. However, while inhalation of the carbon dioxide-enriched air mixtures without doubt increased the alveolar carbon dioxide concentrations during the MVV maneuvers as well as during the flow measurements, in no case did it increase the MVV above the resting control level and only at 4.64 atm was there a notable effect on flow (21% increase with the 1.6% CO_2 mixture). By contrast, exercise brought about substantial increases at all depths in both MVV (range 10%–17%) and expiratory flows (range 27%–48%). A possible explanation for the difference in carbon dioxide effects between the present study and that of Newhouse et al. (15) is that in their study, hyperventilation was carried on for several minutes while our subjects' MVV maneuvers only lasted 15 s. It is possible that this did not allow enough time for hypocapnia to increase flow resistance sufficiently to depress the MVV in our control experiments. Hence, inhalation of carbon dioxide mixtures would not be expected to have any marked effect on MVV. Even if hypocapnia during the MVV maneuver did not depress control MVV levels, the possibility remains that hypercapnia (such as may be encountered in diving) would reduce pulmonary resistance below normal levels. However, we did not (with one exception) see any differences in flow recordings as the end-tidal carbon dioxide levels were increased by inspiration of carbon dioxide mixtures.

The 20% increase in flow that our subjects showed with inhalation of 1.6% (7.4% surface equivalent) carbon dioxide at 4.64 atm was not paralleled by an increase in MVV. A tentative explanation for this might be that 3 of 5 subjects showed decreases (and 2 no changes) in breathing frequencies while performing MVV with the carbon dioxide mixture as compared to air breathing, and this may have biased the MVV downward as explained earlier.

Although it can be deduced from the data of Lewis and Morton (3) that they were able to induce a 10% increase in MVV by inhalation of 7.5% carbon dioxide, this is probably not of relevance for the 6.4% increase in MVV that they observed in response to light exercise because, as they recognized, the alveolar and arterial carbon dioxide tensions are not likely to be significantly increased during exercise at 1.0 atm.

One difference between the experiments of Lewis and Morton (3) in which they found an increase in MVV with inhalation of carbon dioxide-enriched air and our experiments where no carbon dioxide effect was seen is in the carbon dioxide contents. The former authors used a 7.5% carbon dioxide mixture, while our mixtures were leaner with the exception of 7.4% (1 atm equivalent) mixtures at 4.64 and 6.76 atm, respectively. It is noteworthy that the experiment at 4.64 atm is the only one associated with hypercapnia that had a positive effect on the ventilatory parameters. Therefore, while it is feasible that sufficiently high carbon dioxide

tensions in the inhaled air may enhance MVV, the carbon dioxide levels reached during exercise in the present study were probably not of major consequence for the exercise effect to enhance the MVV and the $\dot{V}E_{max}$, 0.4 VC.

There are reports of exercise ventilations under hyperbaric conditions approaching or even exceeding MVV levels (2, 13, 22, 23), while some authors hold the contrasting view that the $\dot{V}E$, W_{max}/MVV ratio roughly equals that at 1.0 atm (1, 24, 25). Our findings are in accord with quite a variation in ratios observed in other laboratories. As shown in Fig. 4*B*, the $\dot{V}E$, W_{max}/MVV ratio when calculated with resting MVV was 0.6 ± 0.11 (SD) at 1.45 atm, 0.72 ± 0.16 (SD) at 4.64 atm, and 0.73 ± 0.18 (SD) at 6.76 atm, whereas calculating it with the 200-W exercise MVV yielded 0.51 ± 0.12 (SD) and 0.59 ± 0.16 (SD) at 1.45 and 4.64 atm, respectively. The differences between these two pairs of ratios clearly depend on the exercise effect to enhance the MVV. In addition, however, it is noteworthy that the highest ratio attained in this study was 0.93 at 4.64 atm, whereas in an earlier study in this laboratory one subject reached 1.22 at 6.76 atm and 1.1 at 1.45 atm. There were considerable individual differences in the MVV levels attained, and these differences account for most of the variability in $\dot{V}E$, W_{max}/MVV ratios in this study because the $\dot{V}E$, W_{max} values showed considerably smaller variations (Fig. 4*A*).

There was no difference in $\dot{V}E$, W_{max} obtained during submersion and nonsubmersion and the same held for resting MVV. It may therefore be concluded that the inertia of the water surrounding the chests of the subjects was of no consequence for their ventilatory capacity; this is in agreement with observations by McKenna et al. (26) and Flynn et al. (27). The latter authors have also demonstrated that the hydrostatic effect of head-out immersion was related to a diminished functional residual capacity and a reduction in MVV by about 15%. Furthermore, they showed that this reduction may be offset by properly increasing intrapulmonary pressure and restoring lung volume. In harmony with those results was the close agreement between MVV in submersion and nonsubmersion in the present study, in which the hydrostatic pressure at the pressure centroid of the chest and the inhaled gas pressure were set to be the same.

Most of the depression of the MVV with increasing depth is clearly the result of higher gas densities and increasing dynamic airway collapse. However, in the light of the present observations, it appears that a higher ventilatory potential may be revealed under the influence of an exercise-induced flow-promoting factor. The optimization of gas flow during exercise is presumably obtained by changes in autonomic nervous control of bronchomotor tone.

Predictions of the ventilatory capacity of the working diver from respiratory flow measurements made at rest should take these considerations into account.

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The research reported here has been jointly funded by the Office of Naval Research and the Naval Medical Research and Development Command through Office of Naval Research Contract N00014-78-C-0205. A.J. Påsche was supported under the auspices of the Norwegian Underwater Institute by a Fellowship Grant from the Royal Norwegian Council for Scientific and Industrial Research (Grant No. KS 1850.7552).

The authors would like to especially thank P.J. Simonetti and R.J. Smith, who volunteered as subjects in addition to assisting in running the experiments. D.C. Marky and J.P. Fair gave valuable assistance inside the chamber, and B.S. Laraway's and W.J. Lawrence's help in technical and operational aspects is greatly appreciated—*Manuscript received for publication December 1979; expanded and revised manuscript received October 1982.*

Hickey DD, Lundgren CEG, Påsche AJ. Influence de l'exercice sur la ventilation volontaire maximale et le débit expiratoire forcé en profondeur. Undersea Biomed Res 1983; 10(3):241–254.— Quatre à six sujets ont accompli des manoeuvres de ventilation volontaire maximale (MVV) et

d'expirations forcées au repos, pendant l'exercise (50, 125, et 200 W), et l'inhalation d'air et de CO_2-air au repos et submergés à des pressions de 1.45, 2.82, 4.64, et 6.76 atm. Le débit expiratoire maximal (à 40% de la capacité vitale) et la MVV au repos diminuèrent de façon exponentielle avec la densité des gaz, mais la diminution était moindre que celle observée dans des études antérieures. Indépendamment de la pression, la MVV augmenta d'environ 10% à 17% et le débit expiratoire de 27% à 48% aux charges de travail les plus élevées. L'augmentation du débit expiratoire disparut en moins de 2 min après l'exercise. L'exercice augmenta la tension du CO_2 en fin d'expiration jusqu'à 9 mmHg. En dépit d'une élévation de plus de 25 mmHg de la tension du CO_2 en fin d'expiration, l'inhalation d'anhydride carbonique au repos n'affecta pas la MVV et produisit un effect de léger à modèré sur le débit expiratoire, l'augmentant au maximum de 21% à 4.64 atm. Apparamment, l'effet renforçant de l'exercice sur la MVV et le débit expiratoire en profondeur proviendrait surtout d'une modification réductrice de l'activité nerveuse autonome sur la résistance du débit pulmonaire, l'accumulation de CO_2 jouant un rôle incertain, et la distension passive des voies respiratoires ne jouant aucun rôle.

anhydride carbonique
exercice
débit expiratoire
densité des gaz
immersion
consommation maximale d'oxygène
ventilation volontaire maximale
pression
mécaniques respiratoires
résistance respiratoire
submersion
capacité ventilatoire

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