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Effects of immersion and static lung loading on submerged exercise at depth

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Thalmann, E. D., D. K. Sponholtz, and C. E. G. Lundgren. 1979. Effects of immersion and static lung loading on submerged exercise at depth. Undersea Biomed. Res. 6(3):259–290.—The effects of static lung loading in the range +20 cmH_2O to −20 cmH_2O was investigated in 3 male subjects breathing air during submerged exercise in the prone position at pressures ranging from 1.45 ATA to 6.76 ATA. Both maximal and submaximal exercise was performed and dry controls were done at 1.45 ATA. A low-resistance bag-in-a-box breathing apparatus (<1.25 cmH_2O/liter/s at 8 g/liter density) was used. Static lung loading had little effect on maximal or submaximal $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, heart rate, or end-tidal PCO_2, while increased breathing gas density did affect these parameters to a larger extent. Imersion per se reduced the $\dot{V}E$ at a given level of $\dot{V}O_2$ and increased both the $\dot{V}T$ and $\dot{V}A$ at a given $\dot{V}E$. Increasingly positive static lung load increased VC and ERV both during rest and exercise. Exercise-induced dyspnea was experienced and scored. At submaximal $\dot{V}O_2$ levels up to 2.5 liter/min this dyspnea did not limit exercise at any depth, but during maximal exertion at 6.76 ATA ($\dot{V}O_2$ from 3.45–3.77 liter/min), dyspnea became work limiting in several cases. Static load had a marked effect on dyspnea and a load of +10 cmH_2O produced the least dyspnea, enabling all subjects to perform maximal exertions for 5 min at 6.76 ATA. The 15-s MVV was performed at all depths and static loads and neither it nor the $\dot{V}E$/MVV ratio correlated with the degree of dyspnea.

cardiorespiratory
dyspnea
heart rate
hyperbaric
hypercapnia
hyperoxia
maximum oxygen consumption
maximum voluntary ventilation
negative pressure breathing
oxygen consumption
positive pressure breathing
respiration
respiratory exchange ratio
tidal volume
ventilation
vital capacity

LIST OF SYMBOLS

ERV	Expiratory reserve volume
MVV	Maximum voluntary ventilation
Pa_{CO_2}	Arterial CO_2 partial pressure
$P_{A_{CO_2}}$	Alveolar CO_2 partial pressure
P_{Bal}	Chest reference balloon pressure
P_{CH}	Chamber atmosphere pressure
P_{CO_2}	Partial pressure CO_2
P_{ES}	Esophageal pressure
$P_{ET_{CO_2}}$	End-tidal CO_2 partial pressure
P_M	Gas pressure at the mouth
P_{O_2}	Partial pressure of oxygen
P_{TH}	Pressure at the mid-thoracic line
R	Respiratory exchange ratio ($\dot{V}_{CO_2}/\dot{V}_{O_2}$)
RV	Residual volume
VC	Vital capacity
$\dot{V}_A$	Alveolar ventilation
$\dot{V}_{CO_2}$	Carbon dioxide elimination
$\dot{V}_E$	Minute expired ventilation
$\dot{V}_{O_2}$	Oxygen consumption
$\dot{V}_{O_{2max}}$	Maximum oxygen consumption
V_T	Tidal volume

When exercising under water, divers are exposed to several conditions that are not present at the surface. From a cardiorespiratory point of view the two most important are the effects of increased gas density and immersion. The effects of increased gas density on the respiratory system have been extensively studied and include increasing turbulence in intrapulmonary flow patterns (Mead 1955; Bühlmann 1963; Maio and Farhi 1967; Murphy, Clark, Buckingham, and Young 1969; Anthonisen, Bradley, Vorosmarti, and Linaweaver 1971), alveolar-arterial oxygen gradient changes (Salzano, Overfield, Rausch, Saltzman, Kylstra, Kelley, and Summitt 1971; Wood and Bryan 1971), increased intrapulmonary flow resistance and work of breathing (Mead 1955; Maio and Farhi 1967; Wood and Bryan 1969; Broussolle, Chateau, Hyacinthe, LaPechon, Burnet, Battesti, Cresson, and Imbert 1976), and a decreased ventilatory response with CO_2 retention during exertion (Hamilton 1967; Miller, Wagensteen, and Lanphier 1971; Fagraeus, Hesser, and Linnarsson 1974). The CO_2 retention and decrease in ventilatory response to exercise can also be explained by the hyperoxia present in hyperbaric atmospheres, but evidence suggests that this can account for only a portion of the observed change (Fagraeus 1974; Fagraeus et al. 1974). Also, the narcotic effects of nitrogen do not seem to be responsible for the ventilatory depression (Fagraeus and Hesser 1970) that occurs while breathing air. Despite the changes described above, it has been possible for subjects exercising in the dry to perform maximum oxygen consumptions at pressures of 6 ATA on air (density of 7.4 g/liter) with end-expired CO_2 levels of up to 60 mmHg (Fagraeus, Karlsson, Linnarsson, and Saltin 1973; Linnarsson and Fagraeus 1976) although the dyspnea at these densities was greater than at 1 ATA.

The effects of immersion have been studied at rest and include increase in cardiac output (Arborelius, Balldin, Lilja, and Lundgren 1972a; Farhi and Linnarsson 1977), diffusional changes (Begin, Epstein, Sackner, Levinson, Dougherty, and Duncan 1976) changes in lung mechanics (Agostoni, Gurtner, Torri, and Rahn 1968; Hong, Cerretelli, Cruz, and Rahn 1969; Prefaut, Lupi-H, and Anthonisen 1976), decreases in FRC and RV (Craig and Ware 1967; Agostoni et al. 1968; Hong et al. 1969; Prefaut et al. 1976), increased pulmonary air trapping (Dahlbäck and Lundgren 1972, 1973), and changes in the ventilation perfusion ratio (Cohen, Bell, Saltzman, and Kylstra 1971; Arborelius, Balldin, Lilja, and Lundgren 1972b). These immersion effects are probably a result of the difference between the hydrostatic pressure surrounding the chest and the pressure at the mouth (static lung load) as well as the elimination of the hydrostatic gradients normally acting on the circulatory system in erect subjects. The effects of immersion on exercising subjects have been studied but usually no attempt was made to vary the static lung load (Dressendorfer, Morlock, Baker, and Hong 1976; Dwyer, Saltzman, and O'Bryan 1977; Spaur, Raymond, Knott, Crothers, Braithwaite, Thalmann, and Uddin 1977). The results of these studies have been inconclusive and give little information on the physiologic effects of static lung loading on exercising divers. Paton and Sand (1947) did, however, vary the static lung load on fully immersed subjects at 1 ATA in both the erect and prone positions during rest and exercise. They found that the most comfortable pressure (eupneic pressure) occurred when mouth pressure was at the same level as the suprasternal notch in the prone position. In the erect position, the eupneic pressure was 5–10 cm below the external auditory meatus at rest and 10–15 cm below the meatus with exercise. The exercise used in the above study was not quantitated and little information was gained about the effects of varying the static lung load. In addition, no attempt was made to determine what effect increased breathing gas density would have on the eupneic pressure.

Though the effects of immersion and increased gas density have been studied separately, studies of the effects of these variables together have been sparse. That the combination of these effects may have a profound effect on the exercising diver was demonstrated by Spaur et al. (1977), whose divers experienced severe work-limiting dyspnea exercising at oxygen consumptions of 2.0 liter/min breathing helium-oxygen at 50 ATA (8 g/liter density) with an inspired Po_2 of 0.3–0.5 ATA. No reason for the dyspnea was apparent from the data, the arterial blood gases showing no evidence of hypoxia or hypercapnia. The flow resistance of the breathing apparatus was felt to be low enough not to cause the dyspnea.

The present study was designed to investigate the effects of static lung loading and increased gas density on the submerged exercising subject breathing air. Both submaximal and maximal exercise levels were used at pressures up to 6.76 ATA. This maximum pressure was chosen because it is the maximum for most air diving.

METHODS

All experiments were done inside the hyperbaric chamber at the State University of New York at Buffalo. The subjects breathed air and used an electrically braked bicycle ergometer and low-resistance bag-in-a-box breathing apparatus, the details of which are described elsewhere (Thalmann, Sponholtz, and Lundgren 1978) (Fig. 1). In the submerged (wet) experiments, the subject was completely submerged and positioned behind a "Lanphier-Morin barrier" in the prone position, the position of his chest above, below, or at the free water interface determining whether he was subjected to positive, negative, or no-pressure breathing (Fig. 2). These conditions will be referred to as static lung loads. Water temperature was always kept at 30° ±1°C. Non-immersed (dry) studies were done either in the sitting position

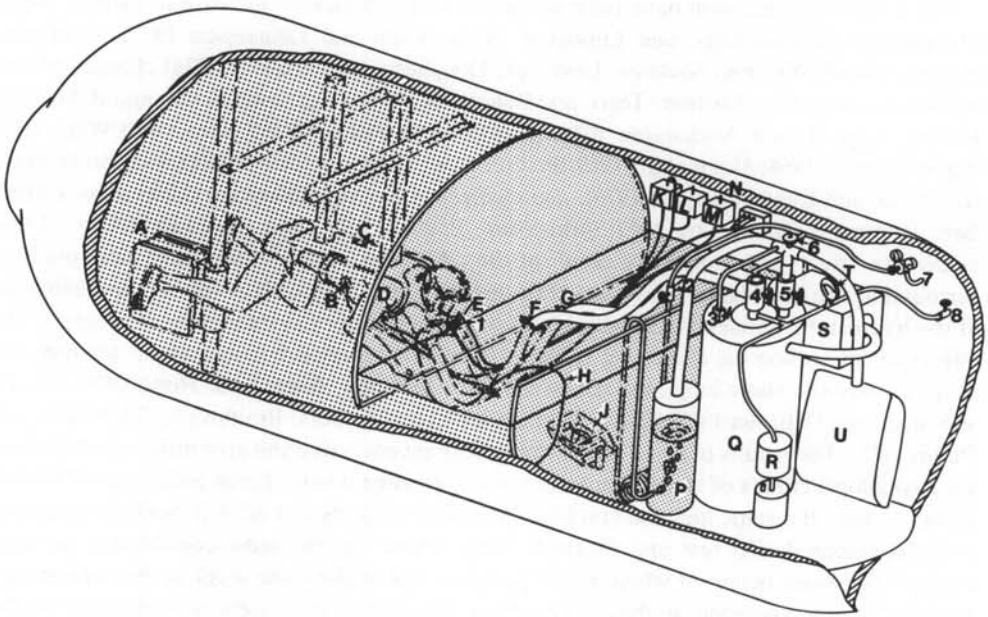

Fig. 1. Experimental setup inside SUNY hyperbaric chamber. *A*, electronically braked submersible ergometer; *B*, ECG lead; *C*, backplate and harness; *D*, chest reference balloon; *E*, full face mask and breathing valve; *F*, exhalation hose (2.25 in. i.d.); *G*, inhalation hose (2.25 in. i.d.); *H*, breath-by-breath gas sample line; *J*, remote ergometer speed indicator; *K*, esophageal pressure transducer (P_{ES}-P_M); *L*, mouth pressure transducer (P_M-P_{CH}); *M*, chest reference balloon pressure transducer (P_{BAL}-P_{CH}); *N*, instrument lead penetrator; *P*, breathing gas humidifier; *Q*, bag-in-a-box; *R*, water trap to prevent pressure in bag-in-a-box from exceeding -15 cmH_2O during emptying; *S*, rolling seal spirometer; *T*, vacuum-indicating gauge; *U*, breathing gas reservoir bag; *1*, breath-by-breath gas sample micrometering valve; *2*, breathing gas fill valve; *3*, bag-in-a-box fill valve; *4*, *5*, 3-way ball valves (2 in.); *6*, emergency breathing gas demand regulator; *7*, breathing gas supply regulator; *8*, overboard gas collection metering valve. For a detailed description of system operation, see Thalmann et al. (1978).

or semi-supine position. The latter position was chosen to place the legs approximately at the level of the heart. The 200-liter bag-in-a-box configuration was of a special low-resistance design (<1.25 cmH_2O/liter/s at a flow of 5 liter/s at 8 g/liter density on air) and continuously supplied the subject with fresh humidified air. The lung counter volume was provided by a rolling seal spirometer, and interface with the subject was provided by a full face mask that contained penetrations for esophageal ($P_{ES}-P_M$) and mouth (P_M-P_{CH}) differential pressure measurement as well as breath-by-breath O_2 and CO_2 analysis at the mouth. The dead space of the oronasal mask in the full face mask and valve housing was estimated to be 150 ml. The subject could be turned "on" or "off" the bag-in-a-box apparatus at any time so that several consecutive mixed expired volumes could be collected and analyzed. Whether on or off the system, the subjects' breathing gas was always supplied directly from the high pressure air banks that ensured that they never had to inspire chamber atmosphere, which would be contaminated with CO_2.

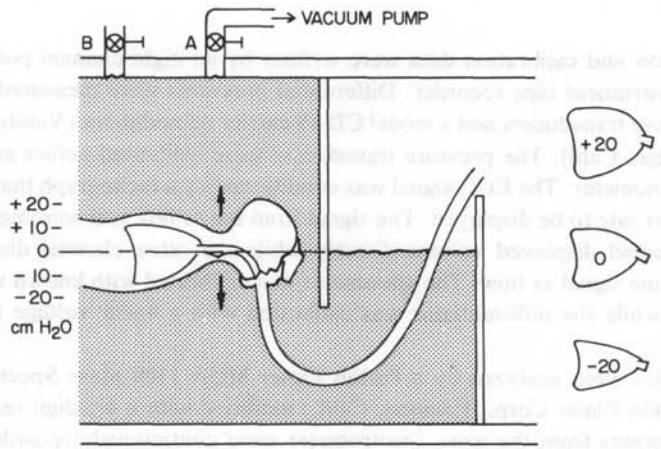

Fig. 2. Schematic of dual barrier system and method of producing various static lung loads. Valve *A* connects top of chamber to a vacuum pump to establish negative pressure necessary to keep water behind barrier at 1 ATA. Valve *B* allows any gas that might accumulate behind barrier to be vented to atmosphere when chamber is pressurized. By moving backplate and harness (Fig. 1) up and down, subject can be positioned so he is exposed to different hydrostatic pressures relative to chamber ambient pressure. Effects of positive or negative static loads are illustrated by size of the "lungs" at the right. Positive static loads increase lung volume, while negative loads decrease lung volume.

Subjects

The three subjects were all male non-smokers experienced in laboratory diving procedures, and had been physically examined and cleared for diving within the previous year. Their physical characteristics (height, weight, and age) were as follows: RJS (183 cm, 98 kg, 29 years); DKS (183 cm, 77 kg, 29 years); EDT (185 cm, 116 kg, 33 years). Subjects DKS and EDT are sport divers averaging 10–20 h per year of open water diving, while subject RJS is a semi-professional diver averaging 100–200 h per year of open water diving. Two of the three subjects (DKS, EDT) trained extensively before the study began by running and pedalling a bicycle ergometer. The third subject (RJS) entered the study after its inception and underwent some physical conditioning during his first few exercise runs. Once in the study, all three subjects continued training between experiments to ensure that no further changes in physical conditioning took place as a result of the experimental exercise runs.

During the experiments, the subjects were typically instrumented with ECG leads, an esophageal balloon, and a chest pressure reference balloon, before entering the chamber. The ECG leads were placed precordially to give the largest QRS complex for each subject. The esophageal balloon (10 × 1.2 cm) was passed through a nostril to a depth of 43 cm, as measured from the nostril, and inflated with 0.2 cc of air. The chest pressure reference balloon was secured to the chest at the suprasternal notch and used to set the static lung loads on the subject's chest in submerged experiments. The static lung loads were defined as the difference between the pressure in the mouthpiece and the hydrostatic pressure at the mid-thoracic line ($P_M - P_{TH}$) (Fig. 2). The mid-thoracic line was 10 cm shallower than the chest reference balloon in all subjects.

Instrumentation

Instrumentation and calibration data were written by an eight-channel polygraph and recorded on an instrument tape recorder. Differential pressures were measured with Validyne DP 15 TL pressure transducers and a model CD-19 carrier demodulator (Validyne Engineering Corp., Northridge, Calif). The pressure transducers were calibrated before each experiment with a water manometer. The ECG signal was conditioned by a tachograph that allowed either the ECG or heart rate to be displayed. The signal from the rolling seal spirometer was divided so that one channel displayed volume directly while the other channel displayed the differentiated volume signal as flow. The spirometer was calibrated with known volumes using a 2-liter syringe, while the differentiator was calibrated with a linear voltage ramp of known slope.

All gas samples were analyzed by a Perkin Elmer MGA 1100 Mass Spectrometer (Aerospace Div., Perkin Elmer Corp., Pomona, Calif.) modified with a 4½ digit readout. Oxygen, CO_2, and N_2 outputs from the mass spectrometer were continuously recorded by the polygraph as well as displayed digitally. Rigorous calibration techniques were used to obtain accuracies of ± 0.03% on all gas analyses using the mass spectrometer.

The expired gas volumes were collected in the bag of the bag-in-a-box and measured by venting the bag through a dry gas meter located outside the chamber. The dry gas meter had previously been calibrated against a 200-liter Tissot spirometer. The temperature of the mixed expired sample was measured with a thermistor probe at the inlet of the dry gas meter. Additional probes were used to monitor chamber and water temperatures, inhaled gas temperature at the box, and exhaled gas temperature at the bag of the bag-in-a-box.

All volumes were assumed saturated with water vapor at chamber depth, and it was assumed that water vapor behaved as an ideal gas during expansion to 1 ATA and that water vapor pressure was independent of absolute pressure. This allowed the water vapor pressure of the gas after expansion to 1 ATA to be calculated from the formula:

$$P_{H_2O_{Sur}} = P_{H_2OCH} (P_B/P_{CH})$$

where P_{H_2OCH} = water vapor pressure of saturated gas at chamber temperature; $P_{H_2O_{Sur}}$ = water vapor pressure of gas after expansion to 1 ATA; P_B = barometric pressure in the laboratory; and P_{CH} = absolute chamber pressure.

The $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ were then calculated using standard equations (Otis 1964).

Experimental schemes and procedures

Two types of exercise runs were performed; graded sub-maximal runs and maximum oxygen consumption runs. Wet submaximal runs were done at static lung loads of +20, +10, 0, −10, and −20 cmH_2O at pressures of 1.45, 2.81, 4.63, and 6.76 ATA (15, 60, 120, and 190 ft of seawater (fsw)). One subject exercised at loads of +30 and −30 cmH_2O at 1.45 ATA, but these loads were not used again because of the extreme discomfort they produced. Wet maximal runs were done only at static lung loads of +10, 0, and −10 cmH_2O and at pressures of 1.45 and 6.76 ATA. These depths and static loads were chosen because time constraints prevented us from using all depth/static load combinations employed in the submaximal study. Thus, we chose to do maximal runs only at depth/static load combinations that the submaximal studies indicated would be of greatest interest. Dry submaximal and maximal control experiments were done only at 1.45 ATA. The minimum pressure used in this study was 1.45 ATA, because that was the shallowest depth at which there was adequate flow through the gas sample lines.

Submaximal runs were done using ergometer settings of 50, 100, 150, or 200 watts, but the actual measured mechanical work rates at these settings, determined by the technique of Clark and Greenleaf (1971), were 47, 90, 141, and 197 watts. Unless otherwise stated, all work rates reported are the actual mechanical work rates, determined from the ergometer calibration curves. A pedalling frequency of 60 RPM was used for all studies.

After the subject had been instrumented, he and two tenders were compressed. At depth, the subject donned the full face mask and had the esophageal balloon inflated. For submerged studies, the subject went behind the barriers and positioned himself in the harness (Fig. 1), after which the static lung load was checked with the chest reference balloon and appropriate adjustments were made. The subject then began the appropriate wet or dry work schedule, which for graded submaximal runs is shown in Fig. 3. The subject was always "turned on" to the bag-in-a-box after expiring to RV and he performed a VC maneuver both before beginning and just prior to stopping exercise. These maneuvers enabled us to determine the ERV at several points during exercise. Just after completing the final work rate of a submaximal run

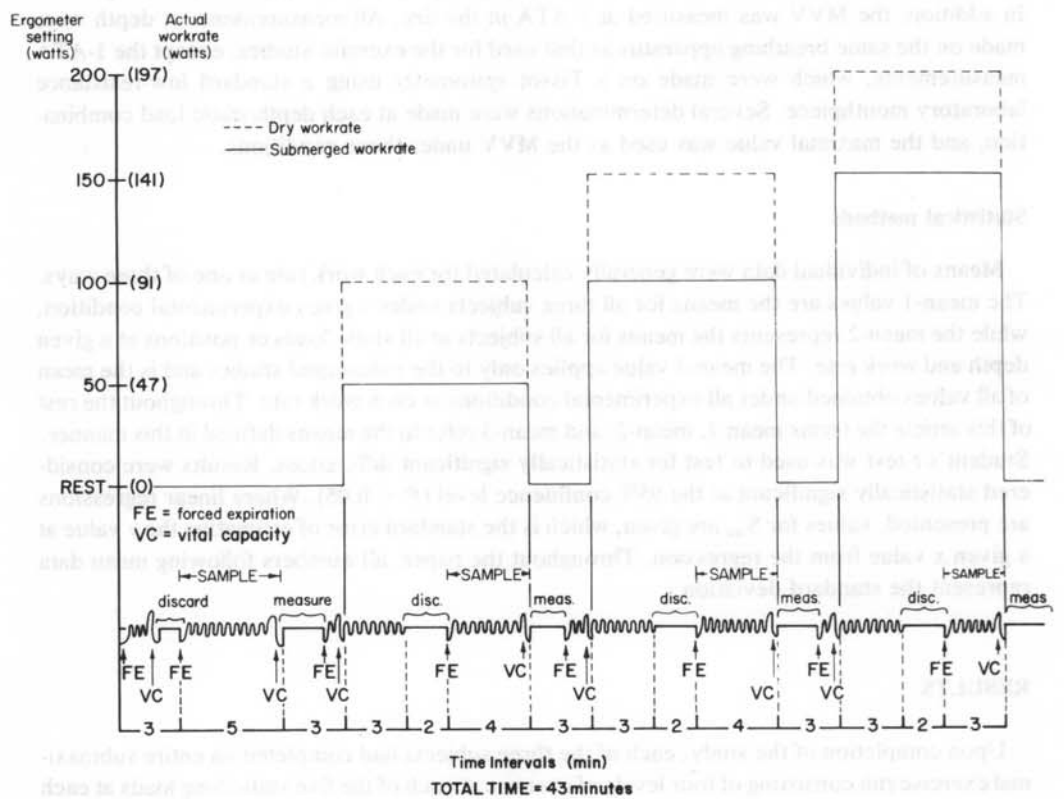

Fig. 3. Submaximum exercise profiles. Top plot graphs work rate vs. time; bottom graph depicts spirogram during exercise run, showing forced expirations, vital capacities, and tidal volumes. The only times the spirogram was not recorded during exercise was during the discard period when bag-in-a-box was being emptied in preparation for a sample. During periods labeled "measure," subject was at rest while collected sample was analyzed.

the subject indicated the amount of dyspnea he felt by signalling with none, one, or two fingers. No fingers indicated no dyspnea, one finger meant moderate dyspnea (a sensation of wanting more gas but not a strong enough sensation to interfere with exercise), and two fingers meant severe dyspnea (a sensation of wanting more gas that was severe enough to make the continuation of exercise uncomfortable).

The maximum work rate was chosen as the highest work rate that the subject could sustain for the full 5 min at 1.45 ATA and 0 cmH_2O static load in the wet studies and in the semi-supine position for the dry studies. Maximum oxygen consumption runs consisted of a 4-min warm-up at 100 watts, 2 min of rest, during which technical preparations were made, followed by 5 min of exercise at the maximum work rate. Measurements were made during the last two minutes of exercise. Subjects were connected to the bag-in-a-box after a forced expiration during maximal exercise, but no VC maneuvers were performed. In all cases, these maximal work rates produced venous lactate concentrations meeting the criteria of Åstrand (1976) for $\dot{V}O_{2\,max}$ (blood lactate >8 mmoles/liter). The 100-watt warm-up had separately been determined to be alactic.

In separate experiments, subjects performed 15-s MVV maneuvers at all four depths used for submaximal exercise, both standing in the dry and submerged at the five static lung loads. In addition, the MVV was measured at 1 ATA in the dry. All measurements at depth were made on the same breathing apparatus as that used for the exercise studies, except the 1-ATA measurements, which were made on a Tissot spirometer using a standard low-resistance laboratory mouthpiece. Several determinations were made at each depth/static load combination, and the maximal value was used as the MVV under those conditions.

Statistical methods

Means of individual data were generally calculated for each work rate in one of three ways. The mean-1 values are the means for all three subjects under a given experimental condition, while the mean-2 represents the means for all subjects at all static loads or positions at a given depth and work rate. The mean-3 value applies only to the submerged studies and is the mean of all values obtained under all experimental conditions at each work rate. Throughout the rest of this article the terms mean-1, mean-2, and mean-3 refer to the means defined in this manner. Student's *t*-test was used to test for statistically significant differences. Results were considered statistically significant at the 95% confidence level ($P < 0.05$). Where linear regressions are presented, values for $S_{y.x}$ are given, which is the standard error of estimating the y value at a given x value from the regression. Throughout the paper, all numbers following mean data represent the standard deviation.

RESULTS

Upon completion of the study, each of the three subjects had completed an entire submaximal exercise run consisting of four levels of exercise at each of the five static lung loads at each of the four pressures while submerged. They also each completed two dry runs while semi-supine and one while sitting. In addition, one to three maximum exercise runs were completed by each subject at each of the three static loads investigated at 1.45 and 6.76 ATA. The volume of data collected makes it impossible to present individual submaximal values. Therefore, linear regressions are presented for the most part, individual data being used only when necessary. Unless otherwise stated, submaximal data were obtained during the 3–4 min

sample period, as shown in Fig. 3, while maximal data were obtained during min 3–5 of the exercise run.

Oxygen consumption and carbon dioxide elimination

Static lung load had no effect on the $\dot{V}O_2$ response to submaximal exercise in the wet studies and position had no effect in the dry studies. When $\dot{V}O_2$ was regressed against pressure, the slopes of the regressions were found to increase linearly with increasing depth (Table 1). The $\dot{V}O_2$ increase at the greatest submaximal work rate (141 watts), though statistically significant, was only 0.08 liter/min in going from 1.45 ATA to 6.76 ATA. When all the wet $\dot{V}O_2$ data were pooled and the mean-3 $\dot{V}O_2$ plotted along with the dry data as a function of work rate (Fig. 4), the wet regression line was displaced 23–25 watts to the left of the dry regression line. This shift represented the additional work involved in moving the legs through the water (hydrodynamic work). Since the subjects pedalled at the same frequency during all wet experiments, this hydrodynamic work was assumed to be constant during all submerged exercise. Immersion increased the resting $\dot{V}O_2$ significantly by 0.03 liter/min (0.31 ± 0.01 liter/min dry, 0.34 ± 0.03 liter/min wet) and increasing the pressure to 6.76 ATA caused a further significant increase to 0.37 ± 0.02 liter/min. When the wet regression line was normalized to the same resting $\dot{V}O_2$ as the dry line, the increase in slope of the wet line gave calculated $\dot{V}O_2$ values 0.05 liter/min higher at 100 watts and 0.10 liter/min higher at 200 watts, both increases being statistically significant. This increase in slope of the 1.45 ATA wet $\dot{V}O_2$ response over the dry response means that for a given increase in mechanical work the increase in oxygen consumption was larger in the immersed than in the dry condition.

The $\dot{V}O_{2\,max}$ values are shown in Table 2. Though the subjects were always able to complete all of the submaximal work rates, two of the subjects were unable to complete some $\dot{V}O_{2\,max}$ runs because of intense dyspnea. In these cases, however, there was often sufficient time to measure the $\dot{V}O_{2max}$ before the subject stopped, and the time at which work was stopped is indicated in Table 2. The $\dot{V}O_{2max}$ data for each subject are plotted in Fig. 4. Each point

TABLE 1

$\dot{V}O_2$ LINEAR REGRESSION AGAINST WORK RATE

($\dot{V}O_2$ (liter/min STPD) ± $S_{y.x}$ = A+B • work rate (watts))

Condition	Pressure, ATA (fsw)	A	B	r	$S_{y.x}$
Dry	1.45(15)	0.33	0.0126	1.00	0.02
Wet	1.45(15)	0.62	0.0131	1.00	0.05
	2.81(60)	0.56	0.0132	1.00	0.05
	4.63(120)	0.58	0.0135	1.00	0.05
	6.76(190)	0.58	0.0140	1.00	0.01
Regression of mean values for all wet studies		0.59	0.0135	1.00	0.04

NOTE: Wet resting values were not used to compute wet regressions.

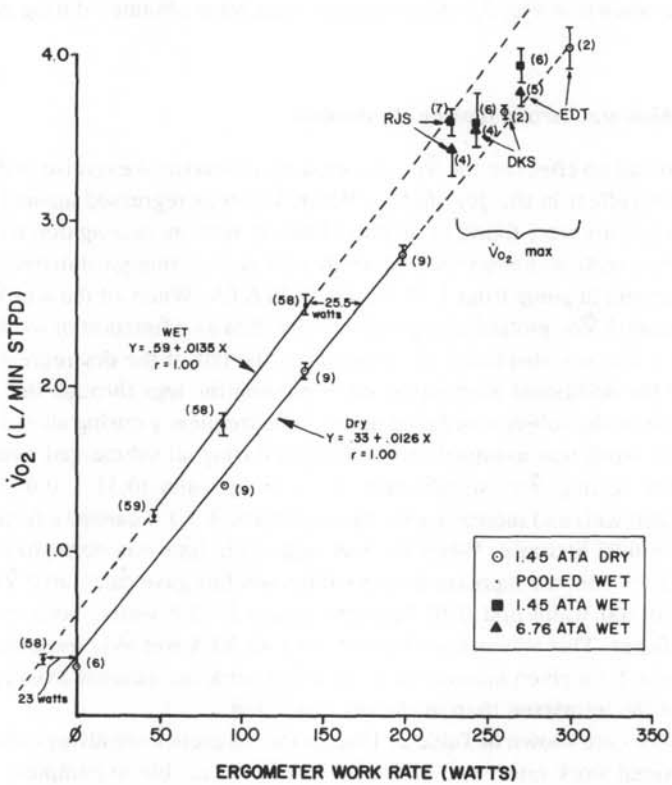

Fig. 4. $\dot{V}O_2$ vs. work rate. Regression lines were calculated using points that fall on solid lines. Numbers in parentheses are number of individual runs included in each data point. $\dot{V}O_{2max}$ points are labeled for each subject. Bars at each point indicate 1 SD. Horizontal displacement of wet regression to left of dry regression is indicated at rest and at 141-watt wet work rate.

represents the mean of all values at each pressure for the wet experiments and the mean of the sitting and semi-supine values for the dry experiments. Though the dry $\dot{V}O_{2max}$ values fall very close to the dry submaximal regression line, the wet values fall a little to the right of the wet submaximal regression line, indicating that the work rates during immersion were supramaximal. There appeared to be little effect of either position or static load on $\dot{V}O_{2max}$, but there was a statistically significant decrease in the wet mean values obtained at 6.76 ATA in two subjects (EDT, RJS) compared to the wet mean values obtained at 1.45 ATA (Table 2). Dry $\dot{V}O_{2max}$ data were obtained in only two subjects (EDT, DKS), and immersion had little effect on the $\dot{V}O_{2max}$ in either subject.

Submaximal $\dot{V}CO_2$ as a function of $\dot{V}O_2$ was linear under all conditions (Table 3) and was unaffected by either static load or position. In contrast to the slopes of the $\dot{V}O_2$ regressions, the slopes of the $\dot{V}CO_2$ regression lines decreased with immersion and further decreased with increasing depth. At a $\dot{V}O_2$ of 2.5 liter/min, the calculated $\dot{V}CO_2$ decreased significantly by 0.20 liter/min at 6.76 ATA compared to 1.45 ATA. Normalizing the wet and dry regression lines to the same resting $\dot{V}O_2$ showed that the calculated $\dot{V}CO_2$ decreased significantly by 0.12 liter/min with immersion at a $\dot{V}O_2$ of 2.5 liter/min. Immersion also decreased the resting $\dot{V}CO_2$ significantly by 0.07 liter/min (0.33 ± 0.03 liter/min wet, 0.26 ± 0.01 liter/min dry). Pressure

TABLE 2

$\dot{V}O_2$, $\dot{V}CO_2$, R, $\dot{V}E$, AND PET_{CO_2} DURING MAXIMAL EXERTION FOR EACH SUBJECT

Pressure, ATA (fsw) and Condition	Static Lung Load, cmH_2O or Position		Subject EDT (300 watts, Dry) (270 watts, Wet) $\dot{V}O_2$	$\dot{V}CO_2$	R	$\dot{V}E$	PET_{CO_2}	Subject RJS (– Dry) (226 watts, Wet) $\dot{V}O_2$	$\dot{V}CO_2$	R	$\dot{V}E$	PET_{CO_2}	Subject DKS (257 watts, Dry) (242 watts, Wet) $\dot{V}O_2$	$\dot{V}CO_2$	R	$\dot{V}E$	PET_{CO_2}
1.45 (15) (Dry)	Sitting		3.95	4.40	1.11	116	43.75	—	—	—	—	—	3.65	3.98	1.09	109	41.33
	Semi-supine		4.14	4.58	1.11	128	41.56	—	—	—	—	—	3.68	4.03	1.10	116	42.87
	Mean		4.05	4.49	1.11	122	42.66						3.67	4.01	1.10	106	42.10
	± SD		±0.13	±0.13		±9	±1.5						±0.02	±0.04	±0.01	±5	±1.09
	+10		3.80	4.12	1.09	118	39.28	3.49	4.09	1.17	125	37.32	3.73	4.64	1.24	143	37.15
			3.95	4.39	1.11	128	37.32	3.55	4.19	1.18	121	39.33	3.72	4.62	1.24	133	37.10
			4.13	4.40	1.07	127	39.28	3.72	4.52	1.21	139	37.00					
1.45 (15) (Wet)	0		3.92	4.13	1.10	130	38.10	3.64	4.29	1.18	124	39.18	3.52	4.32	1.23	147	35.13
			3.89	4.34	1.12	134	37.00	3.67	4.17	1.14	125	39.44	3.83	4.57	1.19	138	38.13
	−10		3.92	4.25	1.02	129	39.12	3.62	4.11	1.13	116	39.19	3.50	4.31	1.23	127	39.28
								3.61	4.22	1.17	119	39.28	3.40	4.15	1.22	118	41.37
	Mean		3.94	4.25	1.09	127	38.35	3.61	4.22	1.17	124	38.66	3.62	4.44	1.23	134	38.03
	± SD		±0.11	±0.13	±0.04	±5	±1.03	±0.08	±0.15	±0.03	±7	±1.07	±0.17	±0.20	±0.02	±11	±7.13
		*3/	3.86	3.69	0.96	80	61.34	*1/ 3.69	3.51	0.95	86	56.23	*2/ 3.45	3.66	1.06	74	61.35
	+10	6/	3.76	3.40	0.91	82	63.32						5/(4:43) 3.72	3.78	1.02	87	57.28
		7/(4:18)	3.65	3.35	0.92	82	57.18										
6.76 (190) (Wet)	0	4/(3:58)	3.78	3.56	0.94	80	63.38	2/ 3.40	3.52	1.03	81	61.34	1/ 3.65	3.88	1.06	83	61.36
		5/(4:44)	3.80	3.61	0.96	84	63.38						4/(4:34) 3.47	3.92	1.13	88	57.24
	−10	1/(3:30)	—	—	—	75†	55.34	3/ 3.34	3.24	0.97	75	61.28	3/(3:40) —	—	—	83†	—
		2/(3:30)	—	—	—	—	—	4/ 3.32	3.40	1.02	77	—					
	Mean		3.77	3.52	0.94	80	60.66	3.45	3.42	0.99	80	59.62	3.57	3.81	1.07	83	59.31
	± SD		±0.08	±0.14	±0.02	±3	±3.54	±0.17	±0.13	±0.04	±5	±2.93	0.13	±0.12	±0.05	±6	±2.36

Maximum work rates shown in parentheses below subjects' initials; † means $\dot{V}E$ estimated from spirogram; * numbers to left of the slash indicate order in which experiments were done. Numbers in parentheses to right of the slash represent time in min: s at which uncompleted runs were terminated. $\dot{V}O_2$ and $\dot{V}CO_2$ are in liter/min STPD, VE in liter/min BTPS, and PET_{CO_2} in mmHg.

TABLE 3
$\dot{V}CO_2$ LINEAR REGRESSION AGAINST $\dot{V}O_2$
($\dot{V}CO_2$ (liter/min STPD) ± S_{yx} = A+B • $\dot{V}O_2$ (liter/min STPD))

Condition	Pressure, ATA (fsw)	A	B	r	S_{yx}
Dry	1.45(15)	−0.13	1.06	1.00	0.11
Wet	1.45(15)	−0.08	1.01	1.00	0.09
	2.81(60)	−0.06	0.98	1.00	0.06
	4.63(120)	−0.04	0.95	1.00	0.07
	6.76(190)	−0.03	0.91	1.00	0.08

had no effect on the immersed resting $\dot{V}CO_2$, which had a mean-3 value of 0.33 ± 0.03 liter/min. The $\dot{V}CO_2$ values for maximal exertion are presented in Table 2, and although the wet values were uninfluenced by static lung load, they were significantly lower at 6.76 ATA compared to 1.45 ATA for all three subjects. Comparison of maximal wet and dry 1.45-ATA runs showed a mixed response: the $\dot{V}CO_2$ increased for one subject (DKS) and decreased for the other (EDT).

Respiratory exchange ratio

Values for the submaximal $\dot{V}CO_2/\dot{V}O_2$ ratio (R) are presented in Table 4 and maximal exertion values are found in Table 2. In the dry studies, R increased almost linearly with increasing work rate. The wet studies showed a rather high value of R at rest, which decreased when exercise began and then increased with a further increase in exercise level. To compare the wet and dry studies, dry-adjusted R values were computed from the regression of R against $\dot{V}O_2$ measured during the 1.45-ATA wet study (Table 4). Thus, the dry-adjusted R values represent the values that would have been obtained had the dry work rate been increased by the hydrodynamic work. Immersion was found to have no effect on R, while pressure tended

TABLE 4
RESPIRATORY EXCHANGE RATIO AS A FUNCTION OF SUBMAXIMAL WORK RATE AND PRESSURE

Condition	Pressure ATA (fsw)	Work Rate, watts: Rest	47	90	141	197
Dry	1.45(15)	0.83 ± 0.01	—	0.92* ± 0.02	0.96* ± 0.02	1.05* ± 0.03
Dry (adjusted)			0.90	0.94	1.01	
Wet	1.45(15)	0.97 ± 0.07	0.90* ± 0.02	0.93* ± 0.01	1.01* ± 0.01	—
	2.81(60)	0.89 ± 0.07	0.89 ± 0.02	0.94 ± 0.02	0.98 ± 0.03	—
	4.63(120)	0.96 ± 0.05	0.88 ± 0.05	0.91 ± 0.05	0.96 ± 0.07	—
	6.76(190)	0.96 ± 0.07	0.85*† ± 0.03	0.86† ± 0.03	0.90*† ± 0.03	—

Values are means ± SD; adjusted dry R values are those that would have occurred if the dry work rate had been increased by hydrodynamic work. *Value significantly different ($P < 0.05$) from value at the next lower work rate; †value significantly different ($P < 0.05$) from value at 1.45 ATA.

to decrease the value of R during exercise, the amount of decrease being greater at the larger work rates. No effect of pressure was seen on resting R values.

Figure 5 shows submaximal mean-2 $\dot{V}_{CO_2}$ as a function of mean-2 $\dot{V}_{O_2}$ at 1.45 and 6.76 ATA. Also shown are data points obtained at $\dot{V}_{O_{2max}}$ that are means for each subject for all runs and all static loads at 1.45 or 6.76 ATA wet and for both positions dry. The solid line represents an R of 1; points falling to the right of the line have an $R < 1$ and points falling to the left have an $R > 1$. All of the submaximal $\dot{V}_{O_2}$ runs had $R < 1$ except for the one obtained during the 197-watt work load in the dry. The 1.45 ATA wet and dry $\dot{V}_{O_{2max}}$ runs produced R values well above 1, while the 6.76 ATA wet $\dot{V}_{O_{2max}}$ runs produced R values scattered about a value of 1.

Ventilatory response

The $\dot{V}_E$ response to exercise was linear over the entire range of submaximal $\dot{V}_{O_2}$(Fig. 6). Neither static load nor position affected the $\dot{V}_E$ response, but both immersion and increasing pressure depressed the response during exercise. In Fig. 6, the response at 2.81 and 4.03 ATA

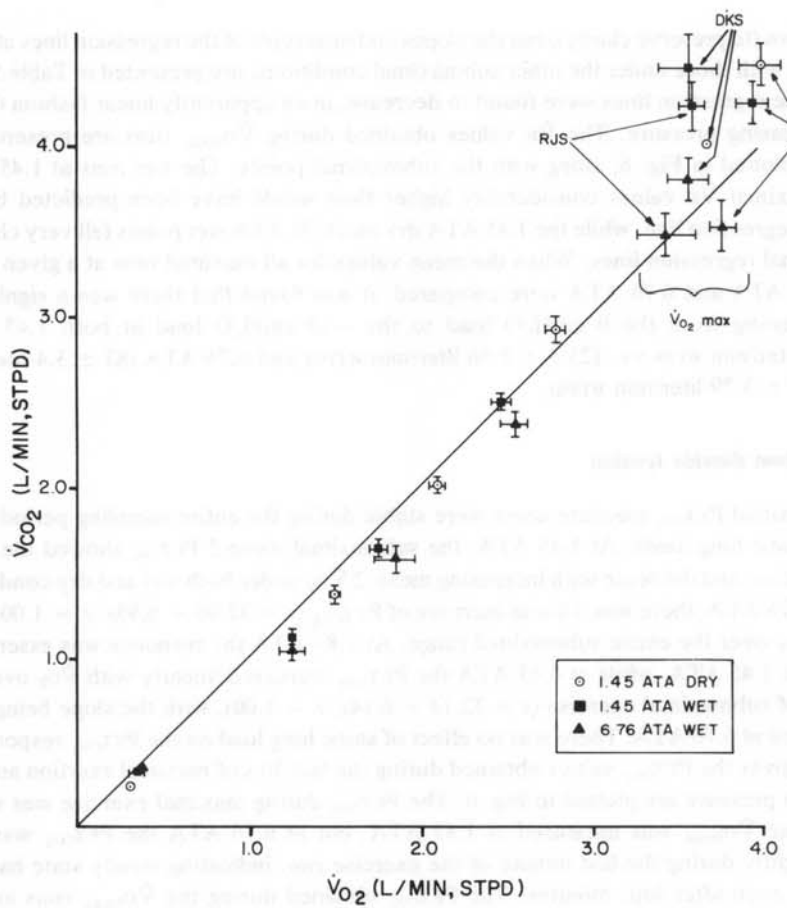

Fig. 5. $\dot{V}_{CO_2}$ vs. $\dot{V}_{O_2}$. Solid line represents a $\dot{V}_{CO_2}/\dot{V}_{O_2}$ ratio of 1.00. Each submaximum point represents mean ± SD at a given depth for all subjects at all static lung loads. $\dot{V}_{O_{2max}}$ points are means for each subject at a given depth at all static lung loads. Regression equations are given in Table 3.

TABLE 5
$\dot{V}_E$ LINEAR REGRESSION AGAINST SUBMAXIMAL $\dot{V}O_2$
($\dot{V}_E$(liter/min BTPS) ± $S_{y.x}$ = A+B • $\dot{V}O_2$(liter/min STPD))

Condition	Pressure ATA (fsw)	A	B	r	$S_{y.x}$
Dry	1.45(15)	−1.71	30.14	0.98	5.60
Wet	1.45(15)	0.26	25.96	0.99	3.34
	2.81(60)	2.50	24.12	1.00	1.35
	4.63(120)	4.24	21.13	1.00	0.85
	6.76(190)	6.70	18.71	1.00	0.53

Regression of Slope(B) vs. Pressure: B = 27.95 - 1.38 • Pressure (ATA); r = 0.99.

was now shown (to preserve clarity), but the slopes and intercepts of the regression lines at these depths, along with those under the other submaximal conditions, are presented in Table 5. The slopes of these regression lines were found to decrease, in an apparently linear fashion (Table 5), with increasing pressure. The $\dot{V}_E$ values obtained during $\dot{V}O_{2max}$ runs are presented in Table 2 and plotted in Fig. 6, along with the submaximal points. The wet runs at 1.45 ATA produced maximal $\dot{V}_E$ values considerably higher than would have been predicted by the submaximal regression line, while the 1.45 ATA dry and 6.76 ATA wet points fell very close to the submaximal regression lines. When the mean values for all maximal runs at a given static load for 1.45 ATA and 6.76 ATA were compared, it was found that there was a significant decrease in going from the 0 cmH_2O load to the −10 cmH_2O load at both 1.45 ATA (133 ± 8.67 liter/min BTPS vs. 123.3 ± 5.56 liter/min BTPS) and 6.76 ATA (83 ± 3.4 liter/min BTPS vs. 77.5 ± 3.79 liter/min BTPS).

End-tidal carbon dioxide tension

The submaximal $P_{ET_{CO_2}}$ measurements were stable during the entire sampling period at all depths and static lung loads. At 1.45 ATA, the submaximal mean-2 $P_{ET_{CO_2}}$ showed the same increase, plateau, and decrease with increasing mean-2 $\dot{V}O_2$ under both wet and dry conditions (Fig. 6). At 6.76 ATA, there was a linear increase of $P_{ET_{CO_2}}$ ($y = 32.00 + 6.95x$, $r = 1.00$) with increasing $\dot{V}O_2$ over the entire submaximal range. At 2.81 ATA the response was essentially the same as at 1.45 ATA, while at 4.63 ATA the $P_{ET_{CO_2}}$ increased linearly with $\dot{V}O_2$ over the entire range of submaximal exercise ($y = 32.14 + 6.14x$, $r = 1.00$), with the slope being 88% of that obtained at 6.76 ATA. There was no effect of static lung load on the $P_{ET_{CO_2}}$ response to $\dot{V}O_2$. Table 2 gives the $P_{ET_{CO_2}}$ values obtained during the last 30 s of maximal exertion and the means at each pressure are plotted in Fig. 6. The $P_{ET_{CO_2}}$ during maximal exercise was stable during the time $\dot{V}O_{2max}$ was measured at 1.45 ATA, but at 6.76 ATA the $P_{ET_{CO_2}}$ was still increasing slightly during the last minute of the exercise run, indicating steady state had not been reached even after four minutes. The $P_{ET_{CO_2}}$ obtained during the $\dot{V}O_{2max}$ runs at 1.45 ATA showed very little difference between the wet and dry studies and were about the same as those obtained at the highest submaximal work rate. At 6.76 ATA, the $P_{ET_{CO_2}}$ at $\dot{V}O_{2max}$ fell above the regression line established by the submaximal runs.

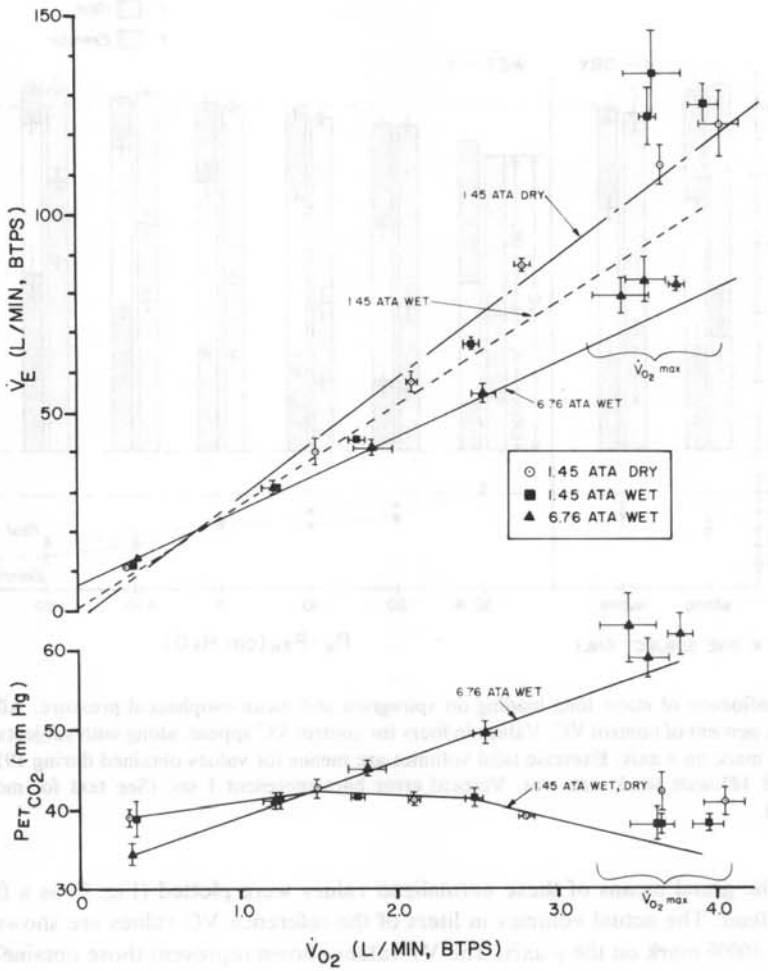

Fig. 6. $\dot{V}_E$ and $P_{ET_{CO_2}}$ vs. $\dot{V}_{O_2}$. Each submaximum represents mean ± SD at a given depth for all subjects at all static loads. $\dot{V}_{O_{2max}}$ points are means for each subject at a given depth at all static lung loads. Regression equation of $\dot{V}_E$ vs. $\dot{V}_{O_2}$ is given in Table 5.

Lung volumes

Spirogram data obtained during the submaximal exercise runs are presented in Fig. 7. Vital capacity maneuvers were performed three times by each subject before beginning exercise and five times after beginning exercise during each submaximal run (Fig. 3). In addition, there were three forced expirations performed before beginning exercise and five done after beginning exercise. The mean resting VC and ERV values for each subject at each depth, and the static load, were calculated from the pre-exercise maneuvers. Since the level of exercise did not influence the VC or ERV, the mean exercise values were calculated from all the maneuvers performed after beginning exercise. Since both the VC and ERV were unaffected by pressure, values at all depths for a given static load were averaged. Both the wet and dry VC and ERV values for the subjects were then expressed as a percent of their resting VC at 0 cmH_2O static

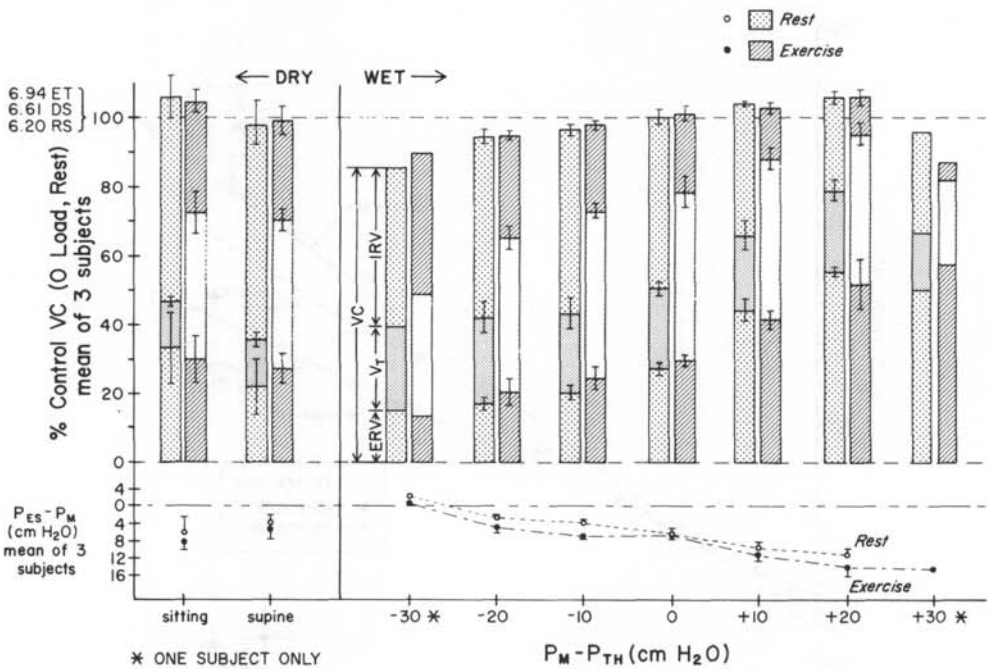

Fig. 7. Influence of static lung loading on spirogram and mean esophageal pressure. All values are presented as percent of control VC. Values in liters for control VC appear, along with subjects' initials, to left of 100% mark on y axis. Exercise tidal volumes are means for values obtained during 191-watt work rate dry and 141-watt work rate wet. Vertical error bars represent 1 SD. (See text for more detailed explanation).

load, and the grand means of these normalized values were plotted (Fig. 7) as a function of static lung load. The actual volumes in liters of the reference VC values are shown in Fig. 7 next to the 100% mark on the y axis. The V_T values shown represent those obtained either at rest or at the highest submaximal wet or dry work rate and are the normalized means for all subjects at all depths for a given static lung load. Mean esophageal pressures for all three subjects at all four pressures and each static load are also given in Fig. 7. The mean esophageal pressures were determined from the average of the maximum and minimum values during the last 30 s of each sample period.

As evident from Fig. 7, both the VC and ERV increased with increasing static load, except in the one experiment done at the +30 cmH_2O load. This was probably due to the unwillingness of the subject to take full inspirations because of the extreme discomfort at this static load. Resting values were not significantly different from exercise values. As the static lung load increased during the wet studies, mean esophageal pressure decreased as a reflection of the increased lung volume both during rest and exercise. During the dry studies the mean esophageal pressure varied little with positional change, while both the VC and ERV fell slightly during the shift from a sitting to a semi-supine position. During all submaximal studies the ERV was established on the first breath and did not change during the course of an exercise run, indicating that no intrapulmonary gas retention took place during submaximal exercise.

The VC was not measured during the $\dot{V}O_{2max}$ runs. Although subjects were always connected to the bag-in-a-box at the end of a forced expiration, the only reliable ERV determina-

tion that could be made occurred just prior to the beginning of exercise. The forced expiration done at the beginning of the sample period was too variable to be of use; however, changes in ERV during the sample period could be measured by noting the shift in the position of the VT on the spirogram as exercise progressed. Table 6 shows the ERV values obtained during both maximal and submaximal exertion at 1.45 and 6.76 ATA. Subjects DKS and RJS established their ERV after the first few breaths and maintained this level for the entire exercise run, whether maximal or submaximal. Subject EDT started his maximal exercise run at 6.76 ATA at a slightly lower ERV than during submaximal exercise and increased it, over the first two minutes, to a value greater than the submaximal value. These changes in ERV in subject EDT were accompanied by an average decrease of 7 cmH_2O in the mean esophageal pressure. During the sample period that occupied the last 2 min of the maximal exercise runs, no changes in ERV or mean esophageal pressure were noted in any of the subjects. The mean esophageal pressures during maximal exertion tended to be lower at the $+10$ cmH_2O static load than at the -10 cmH_2O load, although this was not always the case for every subject.

TABLE 6
COMPARISON OF ERV VALUES OBTAINED DURING MAXIMAL AND SUBMAXIMAL EXERTION

Subject	Static Load, cmH_2O	1.45 ATA		6.76 ATA		
		Submaximal	Maximal	Submaximal	Maximal	
DKS	+10	0.49(3.24)	0.29(2.58)	0.48(3.17)	0.36(2.38)	
	0	0.32(2.12)	0.31(2.05)	0.37(2.45)	0.29(1.92)	
	−10	0.24(1.59)	0.30(1.98)	0.31(2.05)	-	
EDT	+10	0.35(2.43)	0.28(1.94)	0.45(3.12)	0.31(2.15*)	0.45(3.12)*
	0	0.30(2.08)	0.22(1.53)	0.31(2.15)	0.30(2.08)	0.46(3.19)*
	−10	0.19(1.32)	0.29(2.01)	0.28(1.94)	0.20(1.30)	0.35(2.43)*
RJS	+10	0.34(2.11)	0.26(1.61)	0.37(2.29)	0.34(2.11)	
	0	0.23(1.43)	0.21(1.30)	0.30(1.86)	0.34(2.11)	
	−10	0.18(1.12)	0.16(0.99)	0.24(1.49)	0.23(1.43)	

Values are presented as a fraction of control VC (see Fig. 7); values in parentheses are actual volumes in liters. All values were obtained at beginning of exercise unless otherwise noted; *2 min after beginning exercise.

Respiratory frequency and tidal volume

Respiratory frequency was a linear function of $\dot{V}O_2$ for both wet and dry studies ($y = 6.45 + 5.54x$, $r = 1.00$ wet; $y = 8.61 + 7.72x$, $r = 1.00$ dry). Immersion decreased the respiratory frequency at a given $\dot{V}O_2$, indicated by the decrease in both the slope and the intercept of the regression line.

Mean VT values were computed by dividing the minute ventilation obtained during each sample period by the breathing frequency and were plotted as a function of $\dot{V}E$ (Fig. 8). Submaximal exercise produced a linear relationship between VT and $\dot{V}E$ under all conditions. Immersion caused an increase in VT at a given $\dot{V}E$, due mainly to a base-line shift. Increasing depth had little effect on the VT as a function of $\dot{V}E$. During the $\dot{V}O_{2max}$ runs at 1.45 ATA the

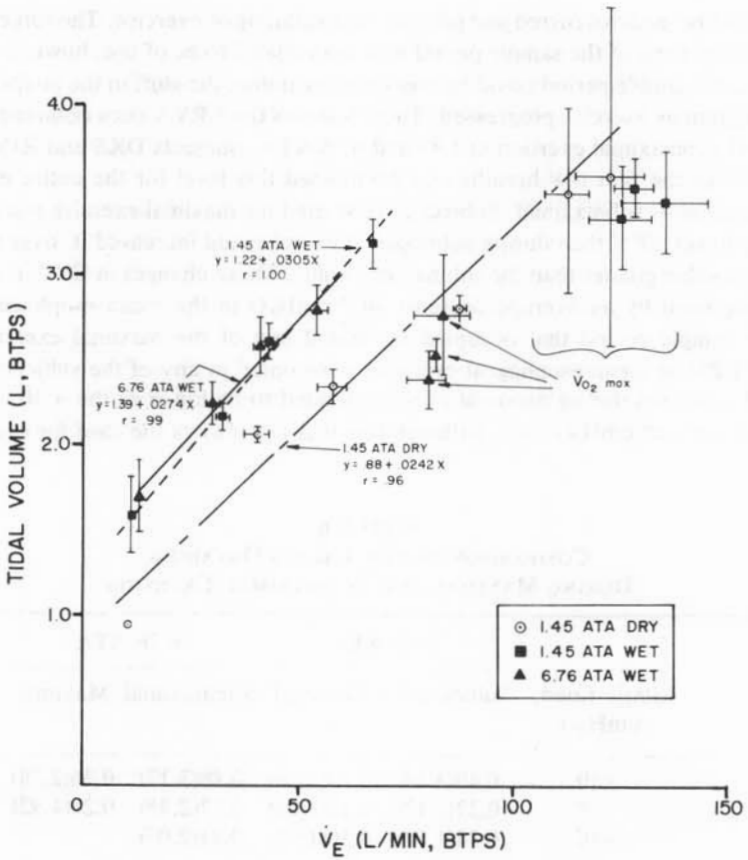

Fig. 8. Tidal volume vs. $\dot{V}_E$. Regression lines were calculated using submaximal points only. Each point represents mean ± SD at a given depth for all static loads. Note that one 1.45-ATA dry submaximal point occurs just to right of 6.76-ATA wet maximal points.

mean V_T values for each subject during wet and dry conditions were similar. During maximal exertion at 6.76 ATA, the V_T was significantly decreased, being reduced to values similar to those measured during the highest submaximal work load at this depth despite a much higher $\dot{V}_E$ (Fig. 6).

Dyspnea

Dyspnea after submaximal exercise was quantitated as described in METHODS and the results are presented in Fig. 9. At 1.45 ATA severe dyspnea was experienced during submaximal exercise on only one run done at the -30 cmH_2O load. This same subject did not quantitate his dyspnea at $+30$ cmH_2O, but he was very uncomfortable at this load. Though the occurrence of dyspnea was sporadic, Fig. 9 shows that both the frequency and intensity increased at negative static loads, at the deeper depths, and at higher work rates.

With maximal exertion, dyspnea was severe under all conditions studied. At 6.76 ATA, however, dyspnea caused premature termination of maximal exercise in two subjects (EDT, DKS, Table 2), who noted a sudden but small increase in dyspnea just before stopping. These

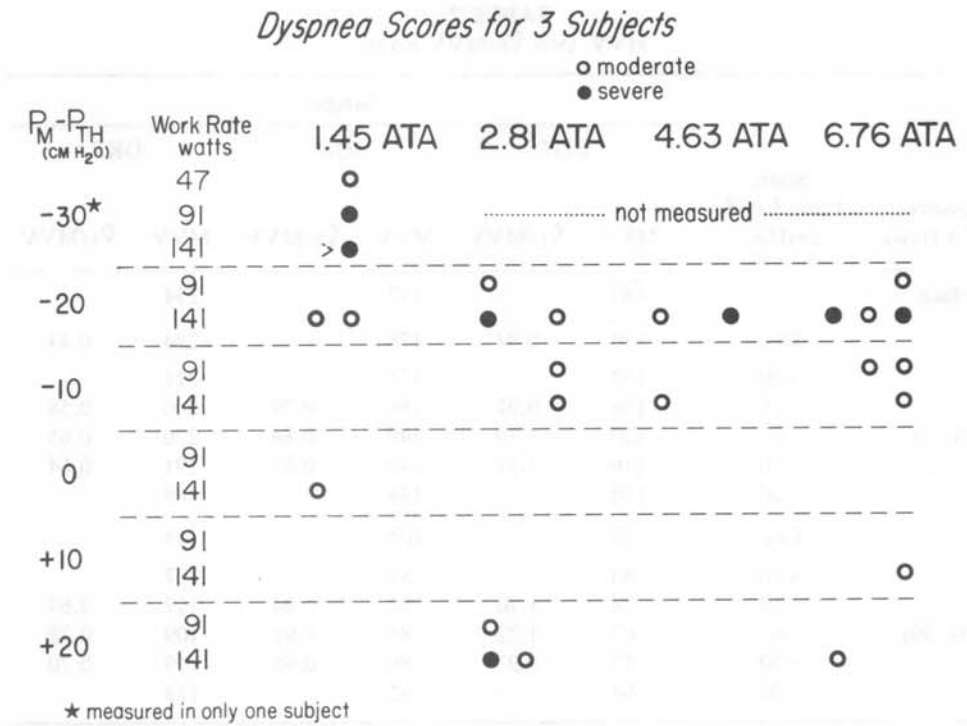

Fig. 9. Dyspnea scores during submaximal exercise. The 47-watt work load was not included for the +20 cmH_2O to −20 cmH_2O loads since no dyspnea ever occurred at that work rate. (See text for scoring method).

subjects were able to complete maximal exertion at positive static loads, but as the loads became more negative, the dyspnea worsened. It should be noted that the two uncompleted runs for these subjects at +10 cmH_2O were the last attempted and were not completed because of fatigue rather than dyspnea. One subject (RJS) was able to complete $\dot{V}O_{2max}$ runs at all three static loads and even completed two at the −10 cmH_2O load, although he thought the dyspnea was worse at this load. At 6.76 ATA the dyspnea was perceived by all subjects to consist of difficulty inspiring, and it did not abate immediately when the exercise was stopped. Instead, it remained constant or even increased slightly during the 30 to 60 seconds of uncontrollable hypernea that followed all $\dot{V}O_{2max}$ runs at 6.76 ATA. In subjects EDT and DKS, audible inspiratory and expiratory stridor was evident after all $\dot{V}O_{2max}$ runs, the intensity being greater after those runs that could not be completed. On auscultation, no wheezing or rhonchi were heard over the lung periphery.

Maximum voluntary ventilation

The MVV values obtained at 1 ATA, 1.45 ATA, and 6.76 ATA are presented in Table 7. The MVV was reduced both with increasing pressure and with decreasing static lung load. In addition to the MVV, the $\dot{V}E$/MVV ratios are presented. These were calculated on the basis of the means of all the $\dot{V}E$ values at a given static load, as presented in Table 4. As can be seen from Table 7, some of the subjects attained $\dot{V}E$ values during $\dot{V}O_{2max}$ runs that were greater

TABLE 7
MVV AND $\dot{V}_E$/MVV RATIO

Pressure, ATA (fsw)	Static Lung Load, cmH_2O	Subject: EDT MVV	EDT $\dot{V}_E$/MVV	RJS MVV	RJS $\dot{V}_E$/MVV	DKS MVV	DKS $\dot{V}_E$/MVV
Surface		186		192		234	
	Dry	130	0.94	158		244	0.44
	+20	132		173		211	
	+10	136	0.91	164	0.79	246	0.58
1.45(15)	0	120	1.10	149	0.84	220	0.65
	−10	116	1.11	142	0.83	191	0.64
	−20	110		144		188	
	Dry	78		103		147	
	+20	80		83		127	
	+10	74	1.10	83	1.04	117	0.69
6.76(190)	0	67	1.22	85	0.95	109	0.78
	−10	77	0.97	80	0.95	119	0.70
	−20	64		82		113	

$\dot{V}_E$ values are means obtained at maximal exertion at each pressure, as presented in Table 2; MVV values are in liter/min BTPS.

than their MVV at the same depth. Also, there appeared to be no correlation between the $\dot{V}_E$/MVV ratio and a subject's ability to complete a given maximum exercise run, the subject having the lowest ratios at 6.76 ATA (DKS) being one who could not complete all the maximum runs.

Heart rate

The response of heart rate to increasing $\dot{V}_{O_2}$ was linear under all experimental conditions for all subjects (Table 8). Values obtained at maximal and submaximal exertion at 1.45 ATA and 6.76 ATA are plotted in Fig. 10. Two subjects (DKS, RJS) had a lower heart rate during exercise in the dry compared to the wet 1.45-ATA studies; the third (EDT) showed no change. When the mean heart rates for all subjects were compared between the dry and wet studies at 1.45 ATA, only the 7 beat/min increase in resting heart rate was significant (Fig. 10). Static lung load had no consistent effect on the heart rate. In the dry studies, two subjects (DKS, RJS) showed a heart rate that was 13–16 beats/min higher in the sitting position than in the semi-supine, but the third subject showed no difference. When the slopes of the individual linear regressions of heart rate vs. $\dot{V}_{O_2}$ for each subject were examined, a consistent fall with increasing pressure was noted in only one subject (RJS); another subject (DKS) showed a decrease in response only in going from 4.03 to 6.76 ATA, and the third subject (EDT) had a mixed response with increasing pressure. To see what effect pressure had on the mean heart rate for all subjects, heart rates were computed for a $\dot{V}_{O_2}$ of 2.5 liter/min from the linear regressions (Table 8), and the only significant decrease occurred at 6.76 ATA.

TABLE 8

HEART RATE LINEAR REGRESSION AGAINST SUBMAXIMAL $\dot{V}O_2$

(Heart Rate (beats/min) ± $S_{y.x}$ = A+B • $\dot{V}O_2$ (liter/min STPD))

Condition	Pressure, ATA (fsw)	A	B	r	$S_{y.x}$	Heart Rate at $\dot{V}O_2 = 2.50$
Dry	1.45(15)	39.86	40.10	1.00	2.9	140.1
Wet	1.45(15)	48.34	37.26	1.00	2.5	141.5
	2.81(60)	47.09	37.50	1.00	2.3	140.9
	4.63(120)	44.80	37.06	1.00	2.6	137.5
	6.76(190)	44.04	34.93	1.00	2.3	131.4

During maximal exercise, all subjects showed a significant drop in heart rate at 6.76 ATA wet compared to 1.45 ATA wet, but the magnitude of the decrease varied from 4 to 10 beats/min. In the two subjects whose data for dry maximal runs are presented, one (EDT) showed no change in heart rate with immersion, while the other (DKS) had a 13 beat/min increase with immersion.

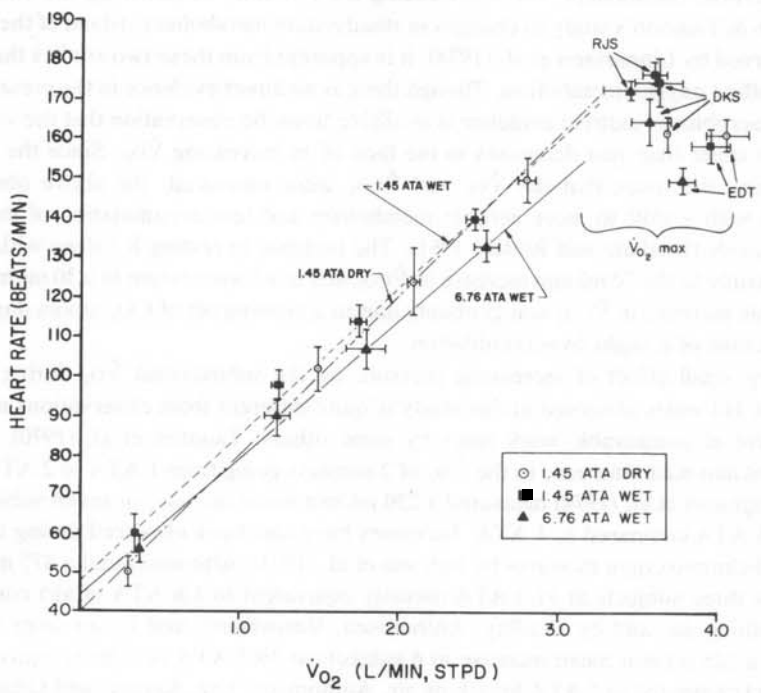

Fig. 10. Heart rate vs. $\dot{V}O_2$. Each submaximal point represents mean ± SD at a given depth for all subjects at all static loads. Maximal points are means for each subject at all static loads.

DISCUSSION

In the present study the changes in the $\dot{V}O_2$ response to submaximal exercise observed with immersion and increasing pressure, though statistically significant, were very small. The change in $\dot{V}O_2$ response in going from the dry to the wet condition could easily have been due to a metabolic increase caused by increased heat loss with immersion in 30°C water, or to a decrease in mechanical efficiency when pedalling in the submerged mode. During the immersed studies the ergometer-subject relationship and water temperature were kept constant, so another mechanism must be invoked to explain the depth-dependent $\dot{V}O_2$ increase. One such mechanism could be a density-dependent increase in the work of breathing, but this seems unlikely in view of the studies done by Cerretelli, Sikand, and Farhi (1969), Demedts and Anthonisen (1973), and Dressendorfer, Wade, and Bernauer (1977), in which the linear relationship between submaximal $\dot{V}O_2$ and work rate measured at 1 ATA breathing air showed no increase when subjects breathed through resistances that increased the power of breathing at a given level of exercise as much as threefold.

If the increased work of breathing at depth cannot explain the increase in $\dot{V}O_2$ at a given work rate, the only other mechanism that comes to mind is increased efficiency in oxygen utilization. Linnarsson, Karlsson, Fagraeus, and Saltin (1974) showed that performing 4 min of submaximal exercise at 1.4 ATA on air did affect the non-steady-state $\dot{V}O_2$ response and reduced the muscle lactate level as well as the oxygen debt. Taunton, Banister, Patric, Oforsagd, and Duncan (1970) observed both an increase in $\dot{V}O_2$, a decrease in R, and decreased acidosis after 5 min of exercise at 2 ATA on air or 100% O_2, compared to air at 1 ATA. These observations led these authors to speculate that the increase in inspired P_{O_2} at 2 ATA resulted in more aerobic metabolism. Notwithstanding the difficulty of ascribing the change in acid production in Taunton's study to changes in steady-state metabolism instead of the non-steady state observed by Linnarsson et al. (1974), it is apparent from these two studies that increased P_{O_2} does affect oxygen metabolism. Though there is no direct evidence in the present study for increased aerobiosis, indirect evidence is available from the observation that the value for R at work rates other than rest decreases in the face of an increasing $\dot{V}O_2$. Since the $P_{ET_{CO_2}}$ was stable during the times that the $\dot{V}O_2$ and $\dot{V}CO_2$ were measured, the above observation is consistent with a shift to more aerobic metabolism and less accumulation of metabolically produced acids (Issekutz and Rodahl 1961). The increase in resting R values with immersion was due mainly to the 70 ml/min increase in $\dot{V}CO_2$ and to a lesser extent to a 30 ml/min decrease in $\dot{V}O_2$. This increase in $\dot{V}CO_2$ was probably due to a blowing off of CO_2 stores during the rest period because of a slight hyperventilation.

The very small effect of increasing pressure on the submaximal $\dot{V}O_2$ during submerged exercise at 141 watts observed in this study is quite different from observations made during dry exercise at comparable work rates by some others. Taunton et al. (1970) measured a 300–400 ml/min mean increase in the $\dot{V}O_2$ of 2 subjects going from 1 ATA to 2 ATA breathing air, and Fagraeus et al. (1974) measured a 230 ml/min mean increase in seven subjects breathing air at 6 ATA compared to 1 ATA. Increases have also been observed during dry exercise breathing helium-oxygen mixtures by Salzano et al. (1971), who measured a 377 ml/min mean increase in three subjects at 31.3 ATA (density equivalent to 4.6 ATA of air) compared to 1 ATA breathing air, and by Bradley, Anthonisen, Vorosmarti, and Linaweaver (1971), who measured a 225 ml/min mean increase in 4 subjects at 19.2 ATA (a density equivalent to 3.7 ATA of air) compared to 1 ATA breathing air. Anthonisen, Utz, Kryger, and Urbanetti (1976) measured $\dot{V}O_2$ at 1, 4, and 6 ATA in seven subjects performing dry exercise while breathing a normoxic N_2–O_2 mixture and, although their data showed some scatter, no consistent in-

crease was observed in $\dot{V}O_2$ with increasing depth, and they proposed that the changes in $\dot{V}O_2$ observed by some others might be due to changes in thermoregulatory status. Though changes in thermoregulatory status could explain the increase in $\dot{V}O_2$ with depth in helium-oxygen studies, they do not offer a satisfactory explanation for the findings obtained in N_2-O_2 or air because of the much lower thermal conductivity at equivalent densities. The lack of increase in the study of Anthonisen et al. (1976) could have been due to the fact that the subjects breathed a normoxic gas mixture. As for the studies of Fagraeus et al. (1974) and Taunton et al. (1970), the fact that their data were obtained during dry exercise and that in the present study the data were obtained during immersion may account for the differences in the depth-dependent $\dot{V}O_2$ increases. The mechanism by which immersion attenuated the depth-dependent $\dot{V}O_2$ increase is unclear. In submerged exercise studies previously published, Spaur et al. (1977) found a 0.50 liter/min increase in $\dot{V}O_2$ at 50 watts at 49.5 ATA breathing helium-oxygen compared to 1 ATA on air, and Dwyer et al. (1977) found no change in $\dot{V}O_2$ between 1.66 ATA breathing air and 43.4 ATA breathing helium-oxygen, although their data showed more scatter than that of the present study.

The behavior of the $\dot{V}O_2$, $\dot{V}CO_2$ and R values during maximal exertion in this study follows the same pattern as that observed by Linnarsson and Fagraeus (1976), Fagraeus (1974), and Fagraeus et al. (1973) during dry studies breathing air to pressures of 6 ATA. They observed that the $\dot{V}O_{2max}$ was higher at 1.4 ATA than at 1.0 ATA and then decreased slightly at 3.0 and 6.0 ATA. Even with this decrease, the $\dot{V}O_{2max}$ at 6 ATA was greater than at 1 ATA. They postulated that the increased inspired P_{O_2} at 1.40 ATA provides a beneficial effect, while the detrimental effects of increasing gas density associated with further increases in pressure outweighed further increases in inspired P_{O_2}. The mechanism responsible for the decrease in $\dot{V}O_{2max}$ at the higher pressures remains a matter of conjecture. One possibility is that at sufficiently high gas densities the intrapulmonary diffusion of O_2 is interfered with during maximal exercise, with consequent limitations of O_2 delivery to the muscles. Although Spaur et al. (1977) observed no decrease in arterial P_{O_2} at $\dot{V}O_2$ levels around 2.0 liter/min and 49.5 ATA breathing 0.3–0.5 ATA O_2 in He (density very close to 6.76 ATA breathing air), Sponholtz, Thalmann, and Van Liew (1978) and Van Liew, Thalmann, and Sponholtz (1978) have recently shown that gas mixing in the lung is decreased with exercise at 1 ATA and at rest at 9.4 ATA breathing air. They did not investigate exercise at 9.4 ATA, but the indications from this work are that diffusion abnormalities may occur during high ventilatory rates at high gas density. It is generally agreed that at 1 ATA, the $\dot{V}O_{2max}$ is limited by the cardiac output, ventilation providing adequate O_2 delivery and CO_2 elimination. If the immersion-induced increase in cardiac output observed at rest (Arborelius et al. 1972a; Farhi and Linnarsson 1977) persists throughout exercise, the immersed $\dot{V}O_{2max}$ should be greater than the dry. The data from this study did not show an increase in $\dot{V}O_{2max}$ with immersion, suggesting that if a cardiac output increase did persist throughout exercise, it was either an insignificant one or its beneficial effect was countered by something else.

Interpretation of the $\dot{V}CO_2$ and R values during maximal exertion is difficult without knowing what the arterial pH and bicarbonate levels were. During heavy exercise both $\dot{V}CO_2$ and R will vary from minute to minute until a steady state is reached, which may take up to 10 min (Issekutz and Rodahl 1961; Wasserman, Van Kessel, and Burton 1967). During the period over which measurements were made in the present study (min 3–5), a true steady state had probably not been reached and there are no data from this study to indicate how this transient state is affected by pressure. Thus, how much of the large increase in $\dot{V}CO_2$ at 6.76 ATA compared to 1.45 ATA is due to shifts in metabolism, and how much is due to changes in total body CO_2 stores cannot be ascertained.

The decrease in $\dot{V}_E$ response to increasing $\dot{V}_{O_2}$ (Fig. 6, Table 5) with increasing pressure has been observed by others (Jarrett 1966; Hamilton 1967; Cook 1970; Salzano, Rausch, and Saltzman 1970; Taunton et al. 1970; Fagraeus and Linnarsson 1973; Fagraeus 1974; Anthonisen et al. 1976; and Linnarsson and Fagraeus 1976) during maximal and submaximal dry exercise studies. There are three traditional explanations for the reduction in exercise ventilation as depth increases, namely, increased inspired oxygen tension, increased gas density, and nitrogen narcosis. It is well known that an increase in inspired P_{O_2} during exercise at 1 ATA will reduce the $\dot{V}_E$ response to exercise (Bannister and Cunningham 1954; Asmussen and Nielsen 1958; Kozlowski, Rasmussen, and Wilkoff 1971; Pirnay, Marechal, Dujardin, Lamay, Deroanne, and Petit 1973). Another factor that decreases the $\dot{V}_E$ response to exercise at 1 ATA is an increase in external breathing resistance (Cerretelli et al. 1969; Demedts and Anthonisen 1973; Dressendorfer et al. 1977). As mentioned in the beginning of our paper, nitrogen narcosis is an unlikely explanation (Fagraeus and Hesser 1970). At increased air pressure, both increased P_{O_2} and breathing resistance contribute to the decrease in $\dot{V}_E$, as shown by Fagraeus et al. (1973), who estimated that at 4.5 ATA, 50% of the drop in $\dot{V}_E$ was due to the increase in inspired P_{O_2} and 50% to increased gas density during submaximal exercise.

The results of the present study show that the additive effects of increasing density and P_{O_2} depress the $\dot{V}_E$ as a linear function of pressure during submaximal exercise. Since depression of $\dot{V}_E$ by increasing P_{O_2} has been shown to occur even as the P_{O_2} is raised above 2 ATA (Pirnay et al. 1973), a depressant effect of P_{O_2} probably existed over the entire depth range of this study, because at 6.76 ATA the P_{O_2} was only 1.63 ATA.

Though increasing breathing gas density and inspired P_{O_2} can explain the depth-dependent decrease in $\dot{V}_E$, the decrease that occurred with immersion must be due to other factors since gas density and P_{O_2} remained constant. It can be calculated from the regressions in Table 5 that at a $\dot{V}_{O_2}$ of 2.5 liter/min the $\dot{V}_E$ decreases about 14% with immersion from a value of 74 liter/min in the dry to 65 liter/min submerged. *A priori* one might expect this decrease in $\dot{V}_E$ to be accompanied by an increase in $P_{ET_{CO_2}}$, but examination of Fig. 6 shows that this is not the case. Actually, the $P_{ET_{CO_2}}$ is not a function of $\dot{V}_E$ but rather is related to $\dot{V}_A$, $\dot{V}_{CO_2}$, and $P_{A_{CO_2}}$ in the following way

$$P_{ET_{CO}} - \Delta = \frac{\dot{V}_{CO_2} \cdot 0.863}{\dot{V}_A} \qquad (1)$$

where $\Delta = P_{ET_{CO_2}} - P_{A_{CO_2}}$. As a first approximation, Δ was taken as 0 and Eq. (*1*) solved for $\dot{V}_A$. The linear regression of these $\dot{V}_A$ values vs. $\dot{V}_{CO_2}$ are presented in Fig. 11, which shows a slight reduction in $\dot{V}_A$ at a given $\dot{V}_{CO_2}$ with immersion. Farhi and Linnarsson (1977) have shown that immersion increases the $P_{ET_{CO_2}} - Pa_{CO_2}$ gradient at rest by 1.3 mmHg, and as a second approximation, we assumed that immersion affected our subjects in the same way. The Pa_{CO_2} can usually be taken to be representative of the $P_{A_{CO_2}}$, which means that Δ would increase by 1.3 mmHg with immersion. Recalculating the immersed 1.45 ATA $\dot{V}_A$, taking this increase in Δ into account, increases its value so that the already small differences between the wet and dry 1.45 ATA regression lines in Fig. 11 all but disappear. This indicates that immersion reduced $\dot{V}_{CO_2}$ along with $\dot{V}_E$, and that CO_2 elimination was not impaired. Another striking effect of immersion is the large increase in V_T at a given $\dot{V}_E$ (Fig. 8), suggesting that $\dot{V}_A$ at a given $\dot{V}_E$ may actually increase with immersion. When the calculated $\dot{V}_A$ values, assuming $\Delta = 0$, are examined as a function of $\dot{V}_E$ (Table 9), it can be seen that the $\dot{V}_A$ at a given $\dot{V}_E$ increases with immersion. If Δ is assumed to be greater than 0, this increase would become greater, as discussed above.

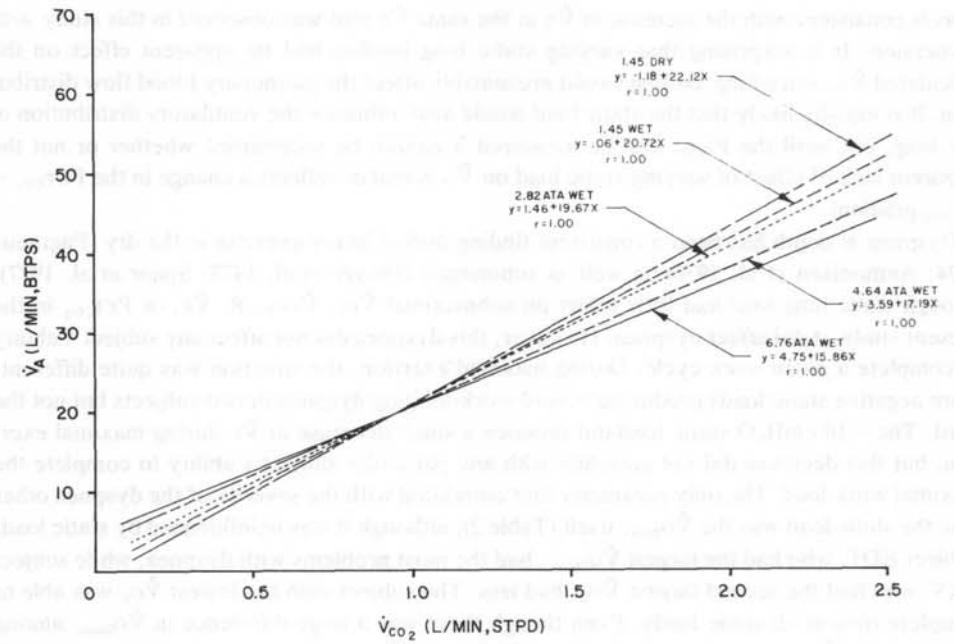

Fig. 11. Plots of $\dot{V}_A$ vs. $\dot{V}_{CO_2}$ linear regressions for submaximal exercise. $\dot{V}_A$ calculated using Eq. *1*, assuming $\Delta = 0$.

The effect of pressure on the $\dot{V}_A$ as a function of $\dot{V}_E$ was not so pronounced as the effect of immersion, and the static lung load had no effect on the $\dot{V}_A$. Immersion up to the neck seems to cause a larger disparity in $\dot{V}_A/\dot{Q}$ distribution (Cohen et al. 1971), with the pulmonary blood flow distribution becoming more even (Litman, Cerretelli, Chinet, Farber, Farhi, and Rennie 1969). This would imply an increase in the maldistribution of alveolar ventilation, and increases in pulmonary air trapping have indeed been observed with immersion (Dahlbäck and Lundgren 1972, 1973). Even with this increased air trapping, Arborelius et al. (1972b) found little change in the mean ventilation over other large areas of the lung using ^{133}Xe radiospirometry. This implies that the areas of underventilation that are responsible for the air trapping are localized and that areas of the lung that are ventilated have increased ventilation.

TABLE 9

$\dot{V}_A$ LINEAR REGRESSION AGAINST $\dot{V}_E$

($\dot{V}_A$ (liter/min BTPS) $\pm$ $S_{yx} = A + B \cdot \dot{V}_E$ (liter/min BTPS))

Condition	Pressure, ATA (fsw)	A	B	r	S_{yx}
Dry	1.45(15)	−2.57	0.78	1.00	0.34
Wet	1.45(15)	−1.64	0.81	1.00	0.12
	2.81(60)	−1.67	0.80	1.00	0.19
	4.63(120)	−0.55	0.78	1.00	0.64
	6.76(190)	−1.13	0.78	1.00	0.56

$\dot{V}_A$ calculated using Eq. *1*, assuming $\Delta = 0$.

This is consistent with the increase in $\dot{V}_A$ at the same $\dot{V}_E$ that was observed in this study with immersion. It is surprising that varying static lung loading had no apparent effect on the calculated $\dot{V}_A$, since lung loading would presumably affect the pulmonary blood flow distribution. It is equally likely that the static load would also influence the ventilatory distribution of the lung, but until the $P_{A_{CO_2}}$ can be measured it cannot be ascertained whether or not the apparent lack of effect of varying static load on $\dot{V}_A$ is real or reflects a change in the $P_{ET_{CO_2}} - P_{A_{CO_2}}$ gradient.

Dyspnea at depth has been a consistent finding during heavy exercise in the dry (Fagraeus 1974; Anthonisen et al. 1976) as well as submerged (Dwyer et al. 1977; Spaur et al. 1977). Though static lung load had little effect on submaximal $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, R, $\dot{V}_E$, or $P_{ET_{CO_2}}$ in the present study, it did affect dyspnea. However, this dyspnea did not affect any subject's ability to complete a given work cycle. During maximal exertion, the situation was quite different, more negative static loads producing severe work-limiting dyspnea in two subjects but not the third. The -10 cmH_2O static load did produce a small decrease in $\dot{V}_E$ during maximal exertion, but this decrease did not correlate with any particular subject's ability to complete the maximal work load. The only parameter that correlated with the severity of the dyspnea other than the static load was the $\dot{V}_{O_{2max}}$ itself (Table 2), although it was uninfluenced by static load. Subject EDT, who had the largest $\dot{V}_{O_{2max}}$, had the most problems with dyspnea, while subject DKS, who had the second largest $\dot{V}_{O_2}$, had less. The subject with the lowest $\dot{V}_{O_2}$ was able to complete runs at all static loads. Even though there was a large difference in $\dot{V}_{O_{2max}}$ among subjects, the $\dot{V}_E$ values were similar for all subjects and the $P_{ET_{CO_2}}$ showed little variation among subjects or with changes in static lung load. Spaur et al. (1977) theorized that the cause of dyspnea in their subject was lung overdistention from progressive intrapulmonary gas retention. Subject EDT did exhibit some intrapulmonary gas retention during the first two minutes of maximal exertion at 6.76 ATA and also had higher ERV values, but the ERV was lower at the 0 and -10 cmH_2O static load, where severe work-limiting dyspnea occurred (Table 6). Subject DKS experienced work-limiting dyspnea at the -10 cmH_2O static load, but an ERV was not obtained during this run. Subject RJS, who was able to complete all maximal exertions at 6.76 ATA, had some ERV values that were greater during maximal exertion than during submaximal. This inconsistent relationship between ERV and work-limiting dyspnea led us to believe that lung overdistention by itself could not explain the work-limiting dyspnea observed in this study. In support of our view is the fact that dyspnea during submaximal exercise was more pronounced at the negative static lung loads where the ERV was decreased. Wood and Bryan (1978) and Anthonisen et al. (1976) have suggested that the onset of dyspnea occurs with airway collapse at high ventilation rates. This would tend to make the dyspnea an expiratory phenomenon, but the subjects in this study agreed that the dyspnea was perceived as inspiratory difficulty. Furthermore, the subjects in the studies of Dwyer et al. (1977) and Spaur et al. (1977) perceived the exercise-induced dyspnea as difficulty inspiring. Hesser and Linnarsson (1977) have shown that at 6 ATA breathing air there is a marked decrease in inspiratory power and the dyspnea observed in their study was indeed felt during inspiration. This is compatible with results of the present study, and it may be that the advantage gained from a $+10$ cmH_2O static load stems from assisting inspiration.

It is notable that the subjects in the studies of Dwyer et al. (1977) and Spaur et al. (1977) experienced work-limiting dyspnea at $\dot{V}_{O_2}$ levels lower than those of our subjects. In Dwyer's study, the maximum $\dot{V}_{O_2}$ measured at 43.4 ATA was 2.36 liter/min and in Spaur's study the maximum $\dot{V}_{O_2}$ at 49.5 ATA was 1.92 liter/min. Both of these studies were done breathing helium-oxygen mixtures of density similar to that of 6.76 ATA of air (8 g/liter), as used in our study. However, there were several other factors that differed between the helium studies and the present experiments, although it cannot be said how these factors caused the disparity in

the levels of exertion required to induce work-limiting dyspnea. These factors included a 7-fold higher hydrostatic pressure and an inspired P_{O_2} approximately 1.2 ATA less in the helium studies than at the 6.76 ATA pressure of the present study. Also, Spaur's study was done with the subjects in the sitting position breathing from a closed-circuit breathing apparatus with their breathing bags at shoulder level. This may have introduced negative static lung loads that influenced the intensity of the dyspnea.

As important as the causes of the dyspnea are its consequences. The respiratory distress and uncontrollable hypernea after maximal exertion at depth would produce grave results if a breathing apparatus malfunction occurred at this time or some water was aspirated by the diver. Though it is not anticipated that divers normally would be working at their maximal capacity, emergency situations must be provided for. By applying a $+10\ cmH_2O$ static load to the diver in the swimming position, the dyspnea involved with maximal work would be reduced sufficiently to eliminate much of the potential hazard. In addition, our subjects found it more comfortable to breathe during submaximal exercise at the $+10\ cmH_2O$ static lung load. It should be noted that a static load of $+10\ cmH_2O$ is in agreement with the eupneic pressure described by Paton and Sand (1947).

The MVV measurements done in an attempt to evaluate the effect of static load on dyspnea served only to confuse the issue. Though decreasing static load did tend to decrease the MVV over the range of static loads from $+20$ to $-20\ cmH_2O$ at both 1.45 and 6.76 ATA, the change from $+10$ to $-10\ cmH_2O$ was small and in some instances was one of increase rather than decrease. In addition, it was discovered that $\dot{V}_E$/MVV ratios were greater than 1.0 in some cases. If one compares the MVV values obtained in this study at 6.76 ATA with those found by others at similar gas densities (Wood and Bryan 1971; Broussolle et al. 1976; Peterson and Wright 1976), it is found that the MVV values of subjects EDT and RJS are in good agreement, while those of subject DKS are slightly higher. Values obtained by Fagraeus and Linnarsson (1973) gave a mean MVV breathing air at 6.0 ATA of 94 liter/min for eight subjects, but extrapolation of their data to 6.76 ATA would decrease this mean to about 85 liter/min. Anthonisen et al. (1976) observed $\dot{V}_E$ values at depth that approached the MVV's, and noted that severe dyspnea was associated with $\dot{V}_E$ values approaching 95% of the MVV. This is in contrast to the present study, where there was no relationship between the MVV/$\dot{V}_E$ ratio and dyspnea and where some subjects completed maximal runs while ventilating at rates above the MVV.

In Table 10, the ventilatory characteristics during maximal exertion are compared with the MVV for all of the subjects at both 1.45 and 6.76 ATA. At 1.45 ATA all subjects opted for higher frequencies and lower tidal volumes during the MVV maneuvers, and the peak flow rates during the MVV were generally greater than those attained during maximal exercise. At 6.76 ATA, subjects EDT and RJS used frequencies during their MVV maneuvers that were close to those used during maximal exercise, while subject DKS chose much higher frequencies for his MVV. In all cases, at 6.76 ATA the magnitude of the P_{ES} excursions during the MVV were much greater than during maximal exertion, while the peak flow rates tended to be lower. This points to airway collapse as the limiting factor in determining the MVV at 6.76 ATA.

As is apparent from Table 10, the subjects differed quite a bit in their MVV values. However, having a high MVV did not seem to confer any particular advantage as far as exercise $\dot{V}_E$ goes, all three of our subjects showing similar $\dot{V}_E$ responses to submaximal and maximal exercise. If one accepts the MVV as indicative of the level of the maximum $\dot{V}_E$ attainable, one would predict that subject DKS would have a higher maximal $\dot{V}_E$ and lower CO_2 retention at depth, which was not the case. Evidently, the factors that regulate $\dot{V}_E$ are not the same as those that limit the MVV at 6.76 ATA.

TABLE 10

COMPARISON OF RESPIRATORY PATTERNS DURING $\dot{V}O_{2max}$ AND THE MVV MANEUVER

Pressure, ATA (fsw)		Subject EDT						Subject RJS						Subject DKS					
		+10		0		−10		+10		0		−10		+10		0		−10	
		$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV	$\dot{V}O_2$	MVV
1.45(15)	PES, cmH_2O	—	130	—	100	—	125	—	160	—	170	—	160	—	185	—	180	—	180
	VT, liter, BTPS	3.66	2.27	3.35	1.33	3.40	2.41	3.32	1.32	3.78	2.30	3.4	2.30	3.58	2.19	2.83	1.90	3.34	1.83
	Max. Insp. Flow liter/s, BTPS	9.0	11.0	10.0	9.0	8.0	10.0	8.5	10.0	7.5	10.0	8.5	9.0	9.2	14.0	8.8	11.0	7.6	10.0
	Max. Exp. Flow	8.5	10.0	8.0	10.0	7.5	7.0	8.0	9.0	7.0	9.0	8.0	9.0	8.8	13.0	8.8	12.0	6.8	11.0
	Frequency, breaths/min	35	160	40	88	38	48	42	124	33	64	38	60	40	112	52	116	38	104
	$\dot{V}E$, liter/min, BTPS	128	136	134	120	129	116	139	164	125	149	119	142	143	246	147	220	122	191
6.76(190)	PES, cmH_2O	54	160	60*	150	58*	200	—	160	40	90	60	90	70	190	65*	170	65*	190
	VT, liter, BTPS	2.48	2.31	2.50	1.70	2.50	2.13	2.68	2.24	2.38	2.40	2.26	2.20	2.73	1.33	2.85	1.04	2.31	1.24
	Max. Insp. Flow	5.0	6.0	5.5	4.8	4.8	5.5	4.0	5.5	5.2	4.2	4.8	4.4	4.8	7.0	4.5	5.0	—	6.0
	Max. Exp. Flow liter/s, BTPS	4.0	5.0	5.0	4.0	4.2	5.0	3.2	4.0	4.8	4.4	4.8	4.4	5.0	6.0	5.2	6.0	—	6.0
	Frequency, breaths/min	33	32	33	40	30	36	32	36	34	36	34	36	32	88	31	104	36	96
	$\dot{V}E$, liter/min, BTPS	82	74	84	67	75	77	86	83	81	85	77	80	87	117	88	108	83	119

*Uncompleted work rates and values presented are those obtained just before stopping work.

Immersion did not change heart rate during submaximal exercise in this study, which is in accord with the observations of Moore, Bernauer, Seto, Park, Hong, and Hayashi (1970), who compared exercise in 23°C air and immersion in 30°C water. The increase in resting heart rate with immersion seen in this study is at odds with the observations of Moore et al. (1970), but this increase may have been the result of an anticipatory reaction by our subjects. In addition, there were differences in position between wet (prone) and dry (sitting and semi-supine) experiments in the study that may or may not have affected the resting heart rate. Our observation of a reduction in heart rate with increasing pressure both during maximal and submaximal exercise is in accord with observations made by others, e.g., Linnarsson and Fagraeus (1970); Taunton et al. (1970); Fagraeus et al. (1973), in dry-air-breathing subjects. It is surprising that varying the static lung load, which is likely to influence intrathoracic blood volume and cardiac filling, did not alter heart rate.

APPLICATIONS

The results of this study paint a fairly detailed picture of the response of the respiratory system to exercise and point up the importance of static lung loading as a major factor in hyperbaric exercise tolerance. The submaximal exercise tolerance of the subjects was good and they had no problem in sustaining $\dot{V}O_2$ levels of 2.5 liter/min and $\dot{V}E$ levels of 50 liter/min at pressures of 6.76 ATA over a wide range of static lung loads. The fact that static lung load did not produce work-limiting dyspnea until $\dot{V}O_{2\,max}$ runs were attempted is probably one reason why the static load has not received the same attention in breathing apparatus design as flow resistance. During submaximal exercise, the subjects in the present study felt that the main feature of the +10 cmH_2O static load was increased comfort.

The physiological data obtained at this static load did not indicate any profound respiratory or metabolic changes other than shifts in the ERV. This, along with the fact that present day breathing apparatus expose the diver to a wide variety of static lung loads, shows that if exertion is limited, static lung load is not a critical parameter. Unfortunately, one cannot be assured that the diver will never be required to exert himself maximally and thus expose himself to the likelihood of becoming extremely dyspneic. One might speculate that by optimizing the static lung load the tolerance to increased flow resistances might be increased. The interaction between static lung loading and flow resistance as it affects the diver is critical in the design of underwater breathing apparatus (UBA), and elucidation of this interaction may ultimately provide designers with a set of optimum physiological design standards. In the meantime, the finding of an optimum static lung load of +10 cmH_2O in the prone position provides an immediately applicable piece of physiological information for UBA designers.

The work-limiting dyspnea during maximal exertion at 6.76 ATA was certainly the most spectacular phenomenon observed in this study, and the influence of static lung loading on this dyspnea was the most enigmatic. No single factor could be implicated as the cause of this dyspnea but it seemed to result instead from a combination of factors that included high $\dot{V}O_2$, increased gas density, CO_2 retention, and a relatively decreased ERV. How this work-limiting dyspnea should be approached awaits clarification of its causal mechanism, and one cannot say at this point whether optimization of static lung load will alleviate the problem under all conditions. At present, one should realize that this phenomenon could occur during operational dives and may be particularly distressing to divers who have not previously experienced it. Currently, education, training, and experience seem the only practical way to deal with this dyspnea.

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Requests for reprints should be addressed to Dr. C. E. G. Lundgren.

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Thalmann, E. D., D. K. Sponholtz, and E. G. Lundgren. 1979. Les effets de l'immersion et du chargeant statique pulmonaire sur l'exercice submergé à la profondeur. Undersea Biomed. Res. 6(3):259–290.—Les effets du chargeant statique pulmonaire dans la portée +20 cmH_2O a −20 cmH_2O ont été examines chez 3 sujets males respirant de l'air pendant des exercices submergés d'une position étendue aux pressions s'alignent de 1,45 ATA à 6,76 ATA. L'exercice maximale et sous-maximale et les contrôis sec ont été fait à 1,45 ATA. Un appareil de respiration sac-dans-une-boîte de résistance basse (<1,25 cmH_2O litre/s à 8 g/litre de densité) a été utilisé. Le chargeant statique pulmonaire a eu peu effet sur $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, maximales ou sous maximales, le taux de coeur, ou fin de marée (end-tidal) PCO_2, pendant que la densité augmentée du gaz respiré a affecté ces parametres d'une mesure plus grande. L'immersion per se a reduit le $\dot{V}E$ au niveau donné du $\dot{V}O_2$ et a augmenté le VT et le $\dot{V}A$, tous les deux, au niveau donné du $\dot{V}E$. Le chargeant statique pulmonaire de plus en plus positif a augmenté le VC et l'ERV pendant le repos et l'exercice. La dyspenée qui a été produit par l'exercice a été éprouvé et entaillé. Aux niveaux VO_2 sous-maximales jusqu'à 2,5 litre/min, cette dyspenée n'a pas limité l'exercice à n'importe quelle profondeur, mais pendant l'exertion maximale à 6,76 ATA ($\dot{V}O_2$ de 3,45–3,77 litre/min) la dyspenée a commencé de limité le travail dans certains cas. La charge statique a eu un effet prononcé sur la dyspenée et un charge de +10 cmH_2O a produit le moindre dyspenée, mettant tous les sujets en état de faire les exertions maximales pour 5 minutes à 6,76 ATA. Le 15-MVV a été fait aux toutes profondeurs et chargées statiques et ni celui-ci ni la proportion VE/MVV a été en relation avee la degré de dyspenée

cardiorespiratoire	respiration de pression negative
dyspenée	consommation d'oxygène
taux de coeur	respiration de pression positive
hyperbare	respiration
hypercapnie	proportion d'échange respiratoire
hyperoxie	volume de marée
consommation d'oxygène maximum	ventilation
ventilation voluntaire maximum	capacité vitale

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