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Effects of increased O_2–N_2 pressure and breathing apparatus on respiratory function

J.B. MORRISON, W.S. BUTT, J.T. FLORIO, and I.C. MAYO

Royal Naval Physiological Laboratory, Alverstoke, Hants, U.K.

Morrison, J.B., W.S. Butt, J.T. Florio, and I.C. Mayo. 1976. Effects of increased O_2–N_2 pressure and breathing apparatus on respiratory function. Undersea Biomed. Res. 3(3):217–234.—The ventilatory response of four subjects was measured at rest and various intensities of exercise. Experiments were conducted in a dry pressure chamber (1) at 1 ATA and 4 ATA with the subjects breathing from a low-resistance mouthpiece, and (2) at 4 ATA with the subjects breathing from open-circuit breathing apparatus (Royal Naval Swimmers' Air Breathing Apparatus). At 4 ATA there was significant hypoventilation and hypercapnia, together with an increased tidal volume and lower respiratory frequency. The use of the breathing apparatus tended to amplify these changes in ventilatory response. In addition, the extent of hypercapnia at 4 ATA was related to the exercise intensity. When subjects breathed from a low-resistance mouthpiece, oxygen uptake was significantly greater at 4 ATA than at the surface for the same ergometric work load, but when they breathed from the breathing apparatus, the increase in oxygen uptake was not significant in comparison to surface values. At 4 ATA bradycardia was evident at all levels of exercise but was not affected significantly by the presence of the breathing apparatus.

diving
exercise
ventilation
oxygen consumption
breathing apparatus
bradycardia
hypercapnia

At increased pressures there is evidence that ventilation is severely restricted during maximum oxygen uptake (Fagraeus 1974). Such ventilation limitation, attributed to the increased work of breathing denser gas mixtures, results in large increases in P_ACO_2, metabolic and respiratory acidosis. It is unlikely that such levels of exercise are normally attained in the underwater situation. Independent studies by Donald and Davidson (1954), Lanphier and Dwyer (1954), and Morrison (1973) indicate that it is possible for a fin swimmer to maintain an oxygen uptake of 2.5 to 3.0 liters/min STPD, and it is conceivable that individual values of up to 4.0 liters/min may be achieved. In contrast, the mean maximum oxygen uptake measured for booted divers operating in mud does not exceed 2.0 liters/min STPD (Donald and Davidson 1954). Nevertheless, when working under pressure it has been shown that at various activity levels requiring less than maximum oxygen uptake, the ventilatory response to exercise is considerably altered, resulting in hypoventilation and various degrees of carbon dioxide retention (Lanphier 1963; Hesser, Fagraeus, and Linnarsson 1968; Salzano, Rausch, and Saltzman 1970; Lambertsen, Gelfand, Lever, Bodammer, Takano, Reed, Dickson, and Watson 1973).

At present, information available on the combined effects of increased ambient pressure and underwater breathing apparatus is limited. In a cooperative underwater swimming project (C.U.S.P. 1953) divers performed static swimming against a 9-lb force while breathing air from open-circuit breathing apparatus. At 4 ATA the mean ventilation was decreased to 63% of the subsurface value. In a similar study by the U.S. Navy (also reported by C.U.S.P. 1953) involving weight lifting underwater it was found that mean ventilation at 4 ATA was some 20% lower than at 1 ATA.

Lanphier (1963) measured P_ACO_2 in divers breathing oxygen–nitrogen mixtures from underwater breathing apparatus. At 4 ATA an average P_ACO_2 of 55 mm Hg was recorded during moderate exertion and individual values of up to 70 mm Hg were measured. Lanphier showed hypercapnia to be due to a combination of elevated oxygen partial pressure and increased gas density, the latter being the more significant factor. The highest values of P_ACO_2 were found to be associated with a reduced respiratory response to inspired carbon dioxide, and it was suggested that certain divers might be classed as *carbon dioxide retainers*, although it was not possible to define them as a distinct group (Lanphier 1969).

From the experimental evidence available it would appear that the reduction of ventilation equivalent and the corresponding hypercapnia measured under hyperbaric conditions are very much augmented in the presence of underwater breathing equipment, although the variation in ventilatory response of divers observed by Lanphier (1969) and the different activity levels studied by the above investigators make it difficult to draw firm conclusions. The purpose of the present experiments was to examine the separate effects on the normal ventilatory response to exercise of (1) increased pressures of oxygen–nitrogen gas mixtures and (2) external respiratory resistance imposed by underwater breathing apparatus.

METHODS

The ventilatory response of divers was measured at rest and at various work loads. Experiments were conducted in a dry pressure chamber at 1 ATA breathing air, at 4 ATA breathing an oxygen–nitrogen mixture (P_{O_2} = 0.42 ATA), and again at 4 ATA breathing the same gas composition from open-circuit breathing apparatus. In addition to minute ventilations and oxygen uptakes, end tidal carbon dioxide partial pressures, tidal volumes, respiratory exchange ratios, and heart rates were also measured. The subjects exercised on a bicycle ergometer to which a constant, fixed friction could be applied independent of wheel speed. During the exercise the subject was supported in a semireclined position by a couch attached to the base frame of the ergometer. External work loads in the range 0 to 1000 kgm/min were undertaken.

The breathing apparatus used in the experiments was the Royal Naval Swimmers' Air Breathing Apparatus (SABA), consisting of a twin-hose, two-stage open-circuit demand type system. This apparatus is described in detail in the *Royal Naval Diving Manual* (1964). The inhalation pressure-flow characteristics (measured under static conditions) of this type of apparatus are described by Williams (1975). The relationship remains relatively constant over a wide range of cylinder pressures, but performance is dependent on absolute pressure.

A dynamic test of the pressure-flow characteristics of the particular breathing apparatus used in the present study was undertaken with a diver breathing from the apparatus at rest and at a work load requiring an oxygen uptake of approximately 2.0 liters/min. Results are summarized in Table 1. In addition, in tests of maximum voluntary ventilation (MVV) at 4 ATA it was found that divers could achieve a mean MVV of 96 liters/min from SABA, which represented 92% of their MVV without breathing apparatus at that pressure (Morrison and Butt 1972).

TABLE 1
The inspiratory resistance of SABA measured at peak flow rate (dry conditions) when subjects breathed at rest and during heavy exercise at 4 ATA.

SABA: O_2–N_2 Mixture at 4 ATA	Resting	Working
Ventilation (liters/min BTPS)	9.5	45.9
Peak inspiratory flow rate (liters/min BTPS)	40.8	116.6
Inspiratory pressure at peak flow rate (cm H_2O)	− 3.9	− 8.3
Expiratory pressure at peak flow rate (cm H_2O)	+ 1.3	+ 8.0
Inspiratory resistance at peak flow rate (cm $H_2O \cdot liters^{-1} \cdot s$)	5.8	4.3

To measure ventilation from the apparatus the inspiratory hose from the demand valve was split and a pneumotachograph inserted. The expiratory hose from the mouthpiece was connected to a second identical demand valve from which expired gas could be exhausted to a collection bag or atmosphere via a two-way tap. Inspiratory and expiratory resistances of the apparatus were thus simulated as closely as possible while allowing measurement of ventilation and analysis of expired gas. In the experiments without breathing apparatus the subjects breathed chamber atmosphere via a mouthpiece designed to maintain low-resistance characteristics. Both the low-resistance and SABA mouthpieces were fitted with end tidal gas sampling valves.

The pneumotachograph was connected to the inspiratory side of the mouthpiece to avoid problems of temperature variation and condensation associated with measurement of expired gas. Two sizes of pneumotachograph of the Fleisch type were employed to accommodate the full range of gas-flow rates encountered. The larger was specially manufactured and had a linear response to oxygen–nitrogen gas flows of up to approximately 400 liters/min measured at 4 ATA ambient temperature (Morrison and Butt 1972). The output from the pneumotachograph was recorded on magnetic tape (Phillips Analog 7) and subsequently analyzed by a computer (DEC LINC 8) programmed to calculate minute ventilation, mean tidal volume, and respiratory frequency. The accuracy of ventilation analysis was tested at 1 ATA and 4 ATA by simulating ventilation using an accurately calibrated syringe of variable volume. The analyzed ventilations were within −3.9% to +0.5% of the calibrated syringe values, with a mean discrepancy of −1.9%.

Samples of mixed expired and end tidal gas were collected outside the pressure chamber and the composition was analyzed by a gas chromatograph (Perkin Elmer F11) coupled to an integrator (Hewlett Packard). The gas chromatograph–integrator system was linear and typically had a repeatability of ±0.3% and an accuracy of ±0.5% of the reading (i.e. the measured component of the gas mixture). For each analysis all components of the gas were measured and the results summed. The analysis was rejected if the sum was outside the limits 100% ± 1%.

All four subjects were laboratory staff accustomed to working in pressure chambers under dry and wet conditions. Subjects AG and JT also had extensive sea-diving experience while the other two subjects had only limited experience of sea diving. Measurements were taken at rest and at four levels of exercise, except in the case of JM where five levels were studied. The subjects exercised for 10 min and measurements were taken during minutes 7 to 10, at which time the subject was assumed to be in a relatively steady state. Due to a change in the free-wheel friction of the bicycle ergometer occurring during maintenance, the no-load condition of both JB and JM represents a lower oxygen uptake ($\dot{V}_{O_2}$) than for the other two subjects. The ergometric loads of the four subjects are therefore not directly comparable.

RESULTS

Ventilatory Response to Exercise

The experimental results are presented in Tables 2–5. All four subjects exhibited an essentially normal respiratory response to exercise at 1 ATA. JB showed an increase in end tidal carbon dioxide partial pressure (P_{CO_2}) in relation to oxygen uptake resulting in a P_{CO_2} of 47 mm Hg at a $\dot{V}_{O_2}$ of 2.0 liters/min as shown in Table 4. This corresponds to a lower ventilation equivalent to oxygen at 1 ATA than the other subjects, particularly during heavy exercise. Ventilatory response to inspired carbon dioxide of all four subjects was found to be well within the normal range. In the case of JB, although the slope of the relationship is similar to that of the other subjects, the relationship is slightly displaced to the right of the others due to a higher intercept (P_{CO_2}) value (see Table 6).

Results show a reduced ventilatory response to exercise at pressure (Tables 2–5), in particular at an oxygen uptake in excess of 1.5 liters/min. At the maximum exercise level of each subject, ventilation ($\dot{V}_E$) is reduced between 7% and 18% despite a tendency for the corresponding oxygen uptake to be increased. When breathing from SABA at the maximum exercise level, ventilation is reduced between 14% and 34% in comparison to 1 ATA. Under resting conditions there is evidence of hyperventilation at 4 ATA, the mean ventilation equivalent for oxygen being 31.7 compared with 26.4 at the surface. When breathing from SABA, however, the mean ventilation equivalent is unaltered from the surface value.

The alteration of ventilation response to exercise observed at pressure is reflected in the corresponding end tidal carbon dioxide partial pressures. At a $\dot{V}_{O_2}$ of 1.5 liters/min or more, end tidal P_{CO_2} values measured at 4 ATA are in all cases greater than 40 mm Hg and increases of 5–7 mm Hg above corresponding measurements at 1 ATA are not uncommon. Further hypoventilation induced by use of the breathing apparatus resulted in even higher end tidal P_{CO_2} values in AG and JM but not in JT and JB. At the maximum exercise level attempted by the four subjects, the mean end tidal P_{CO_2} was 48 mm Hg when they were wearing SABA at 4 ATA compared with 39 mm Hg under normal conditions at 1 ATA, an increase of more than 20%.

Due to the small differences in oxygen uptake between experiments at 1 ATA and those at 4 ATA, it is difficult to quantify statistically changes in ventilation response to exercise. For this reason results at 1 ATA and 4 ATA have been compared on the basis of the change measured at a given activity level (i.e. rest or work load). The combined data of all subjects ($n = 21$) indicates that the reduction in $\dot{V}_E$ during activity at 4 ATA is significant ($P<.005$) when subjects breathe from SABA, but not ($P>.1$) when they breathe via the low-resistance valve. In contrast, a tendency to increased $\dot{V}_{O_2}$ noted at 4 ATA is significant only when subjects breathed via the low-resistance mouthpiece ($P<.005$). These statistics indicate a reduction in ventilation equivalent at 4 ATA both with and without breathing apparatus. This is supported by analysis of end tidal P_{CO_2} on the same basis. At 4 ATA there is a significant increase in end tidal P_{CO_2} ($P<.005$), both with and without breathing apparatus.

At 4 ATA, when subjects used SABA, $\dot{V}_E$ ($P<.005$) and $\dot{V}_{O_2}$ ($P<.01$) were both significantly less than when they used the low-resistance mouthpiece. End tidal P_{CO_2} tended to increase, although only at $P<.1$.

The responses of the four subjects are summarized in Table 7 and Figures 1 to 4 as follows. The relationship (for example, between $\dot{V}_E$ and $\dot{V}_{O_2}$) was approximated to a linear function and a regression line calculated for each set of experimental conditions. Because the number of experimental points varied among the four subjects, the regression line of each subject for a

TABLE 2
Experimental results: Subject JT

	Work load kgm/min	$\dot{V}_E$ liters/min BTPS	$\dot{V}_{O_2}$ liters/min STPD	$\dot{V}_E/\dot{V}_{O_2}$	End tidal P_{CO_2} mm Hg	Respiratory exchange ratio R	$\bar{V}_T$ liters	Respiratory rate breaths/min	Heart rate beats/min	Oxygen pulse ml/beat
Surface	*Rest*	9.7	0.37	26.3	34.9	0.76	0.66	14.8	49	7.5
	0	24.3	0.92	26.4	39.7	0.83	1.27	19.1	85	10.8
	250	38.0	1.51	25.2	43.1	0.84	1.99	19.1	107	14.1
	500	50.6	1.67	30.3	38.3	0.87	2.49	20.3	107	15.6
	750	67.2	2.35	28.6	37.0	0.93	2.96	22.7	124	19.0
4 atm	*Rest*	8.4	0.26	32.2	33.5	0.85	0.67	12.4	65	4.0
	0	30.8	1.25	24.6	40.0	0.86	1.93	16.0	83	15.1
	250	41.1	1.63	25.2	42.1	0.89	2.41	17.1	101	16.1
	500	44.8	1.92	23.3	42.2	0.82	2.65	16.9	113	17.0
	750	60.6	2.62	23.1	-	0.94	3.11	19.5	124	21.1
4 atm SABA	*Rest*	8.5	0.30	28.4	35.5	0.81	0.75	11.4	49	6.1
	0	24.7	1.05	23.5	38.9	0.83	1.79	13.8	84	12.6
	250	35.9	1.57	22.9	41.7	0.87	2.38	15.1	104	15.1
	500	43.9	1.90	23.1	41.3	0.88	2.68	16.4	109	17.4
	750	57.5	2.48	23.2	43.4	0.91	3.14	18.3	116	21.3

TABLE 3
Experimental results: Subject AG

	Work load kgm/min	$\dot{V}_E$ liters/min BTPS	$\dot{V}_{O_2}$ liters/min STPD	$\dot{V}_E/\dot{V}_{O_2}$	End tidal P_{CO_2} mm Hg	Respiratory exchange ratio R	$\bar{V}_T$ liters	Respiratory rate breaths/min	Heart rate beats/min	Oxygen pulse ml/beat
Surface	*Rest*	8.5	0.33	25.8	35.8	0.81	0.92	9.2	66	5.0
	0	24.3	0.94	25.9	35.3	0.81	1.43	17.1	99	9.5
	250	40.4	1.46	27.7	36.1	0.88	2.19	18.5	110	13.2
	500	56.4	2.00	28.2	39.2	0.93	2.35	24.0	140	14.3
	750	80.5	2.52	31.9	37.2	0.94	3.10	26.0	157	16.0
4 atm	*Rest*	9.9	0.33	30.0	36.2	0.80	1.28	7.7	65	5.1
	0	28.4	1.01	28.1	40.4	1.01	1.82	15.6	100	10.1
	250	41.2	1.47	28.0	41.0	0.95	2.74	15.1	109	13.5
	500	59.1	2.22	26.6	44.2	0.93	2.85	20.7	135	16.4
	750	66.8	2.63	25.4	44.1	0.96	3.61	18.1	141	18.6
4 atm SABA	*Rest*	9.2	0.38	24.2	38.0	0.91	1.67	5.5	64	5.9
	0	20.8	0.98	21.2	42.2	0.88	3.18	6.5	101	9.7
	250	28.7	1.55	18.5	49.0	0.88	4.20	6.8	111	14.0
	500	45.0	1.90	23.7	42.6	0.91	3.40	13.4	129	14.8
	750	53.3	2.59	20.6	50.1	0.93	3.29	16.2	146	17.8

TABLE 4
Experimental results: Subject JB

	Work load kgm/min	$\dot{V}_E$ liters/min BTPS	$\dot{V}_{O_2}$ liters/min STPD	$\dot{V}_E/\dot{V}_{O_2}$	End tidal P_{CO_2} mm Hg	Respiratory exchange ratio R	$\overline{V}_T$ liters	Respiratory rate breaths/min	Heart rate beats/min	Oxygen pulse ml/beat
Surface	*Rest*	5.7	0.22	26.0	34.7	0.93	0.77	7.4	68	3.2
	0	10.9	0.44	24.8	39.9	0.89	0.86	12.7	81	5.4
	250	23.3	1.05	22.2	43.1	0.91	1.93	12.1	99	10.6
	500	29.4	1.41	20.9	45.8	0.91	2.13	13.8	121	11.7
	750	44.1	2.01	21.9	46.6	0.93	2.47	17.9	142	14.2
4 atm	*Rest*	7.1	0.20	35.5	39.7	0.99	0.59	12.1	60	3.3
	0	10.7	0.41	26.1	39.2	0.94	1.27	8.5	67	6.1
	250	24.0	1.11	21.6	45.3	0.85	2.02	11.8	98	11.3
	500	25.2	1.39	18.1	54.5	0.86	2.71	9.3	109	12.8
	750	36.9	1.97	18.7	52.7	0.91	2.62	14.1	130	15.2
4 atm SABA	*Rest*	5.5	0.22	25.0	39.6	0.95	0.89	6.2	57	3.9
	0	10.9	0.40	27.3	40.5	0.90	0.72	15.2	79	5.1
	250	21.6	1.05	20.6	43.2	0.86	1.88	11.5	98	10.7
	500	26.7	1.34	19.9	47.9	1.01	2.32	11.5	116	11.6
	750	34.6	1.76	19.7	53.4	0.95	2.95	11.7	128	13.4

TABLE 5
Experimental results: Subject JM

	Work load kgm/min	$\dot{V}_E$ liters/min BTPS	$\dot{V}_{O_2}$ liters/min STPD	$\dot{V}_E/\dot{V}_{O_2}$	End tidal P_{CO_2} mm Hg	Respiratory exchange ratio R	$\bar{V}_T$ liters	Respiratory rate breaths/min	Heart rate beats/min	Oxygen pulse ml/beat
Surface	*Rest*	7.8	0.28	27.9	38.6	1.00	0.68	11.5	59	4.7
	0	13.5	0.48	28.1	38.1	0.96	0.88	15.3	75	6.4
	250	20.1	0.79	25.4	39.8	0.93	1.18	17.1	83	9.5
	500	31.0	1.24	25.0	41.4	0.94	2.05	15.1	100	12.4
	750	45.6	1.67	27.3	39.9	1.01	2.70	16.9	130	12.8
	1000	71.8	2.50	28.7	37.0	1.03	3.04	23.6	151	16.6
4 atm	*Rest*	7.8	0.27	28.7	39.5	1.04	0.95	8.2	54	5.0
	0	14.7	0.52	28.3	37.8	0.97	1.48	9.9	70	7.4
	250	23.5	0.88	26.7	40.6	0.95	1.82	12.9	83	10.6
	500	33.6	1.44	23.3	45.3	0.90	1.70	19.8	100	14.4
	750	41.4	1.73	23.9	46.1	0.95	3.17	13.1	121	14.3
	1000	66.4	2.59	25.6	42.4	0.97	3.33	19.9	151	17.2
4 atm SABA	*Rest*	5.9	0.21	28.1	39.8	1.14	1.28	4.6	57	3.7
	0	11.2	0.46	24.3	39.8	0.92	1.89	5.9	69	6.7
	250	17.1	0.83	20.6	43.1	0.89	2.53	6.8	77	10.8
	500	26.5	1.28	20.7	46.4	0.94	2.71	9.8	106	12.1
	750	42.7	1.86	23.0	44.5	0.95	3.37	12.7	124	15.0
	1000	55.7	2.42	23.0	50.0	1.05	3.31	16.8	153	15.8

TABLE 6

Relationship of ventilation to end tidal P_{CO_2} when subjects were breathing carbon dioxide-enriched gas mixture at rest.

Subject	B	S	*r*
AG	30.7	3.76	0.903
JT	31.7	3.42	0.976
JM	33.9	3.93	0.993
JB	39.2	4.03	0.911

B represents the intercept (P_{CO_2}); S, the slope (liters • min^{-1} $\dot{V}_E$/mm Hg end tidal P_{CO_2}); and *r*, the coefficient of correlation of the regression line.

TABLE 7

The slopes and coefficients of correlation of the regression lines calculated for each subject at each experimental condition, and describing the relationship of ventilation (liters/min BTPS), end tidal P_{CO_2} (mm Hg) and cardiac frequency to oxygen uptake (liters/min STPD), and ventilation to tidal volume (liters).

Subject	Experimental condition	$\dot{V}_E$: f($\dot{V}_{O_2}$)		P_{CO_2}: f($\dot{V}_{O_2}$)		$\dot{V}_E$: f($\overline{V}_T$)		HR: f($\dot{V}_{O_2}$)	
		S	*r*	S	*r*	S	*r*	S	*r*
AG	Surface	32.3	0.994	1.22	0.679	32.6	0.987	41.0	0.993
	4 atm	24.9	0.998	3.44	0.966	24.3	0.967	32.8	0.985
	4 atm SABA	20.7	0.983	4.88	0.817	7.7	1.000	35.6	0.979
JT	Surface	29.4	0.991	0.96	0.235	21.9	0.999	36.8	0.965
	4 atm	22.1	0.998	5.56	0.982	18.6	0.999	26.6	0.980
	4 atm SABA	22.4	1.000	3.59	0.973	17.9	0.995	31.5	0.965
JM	Surface	28.7	0.997	−0.40	−0.211	17.5	0.992	41.6	0.988
	4 atm	24.5	0.996	2.48	0.651	14.6	0.881	41.0	0.995
	4 atm SABA	22.7	0.997	4.30	0.922	12.7	0.937	43.1	0.995
JB	Surface	21.0	0.999	6.22	0.930	15.2	0.983	40.9	0.995
	4 atm	16.5	0.995	8.97	0.910	9.6	0.954	40.4	0.999
	4 atm SABA	18.3	0.998	8.57	0.961	12.1	0.974	43.4	0.981
Mean of four subjects	Surface	27.8	-	2.00	-	21.8	-	40.1	-
	4 atm	22.0	-	5.11	-	16.7	-	35.2	-
	4 atm SABA	21.0	-	5.34	-	12.6	-	38.4	-

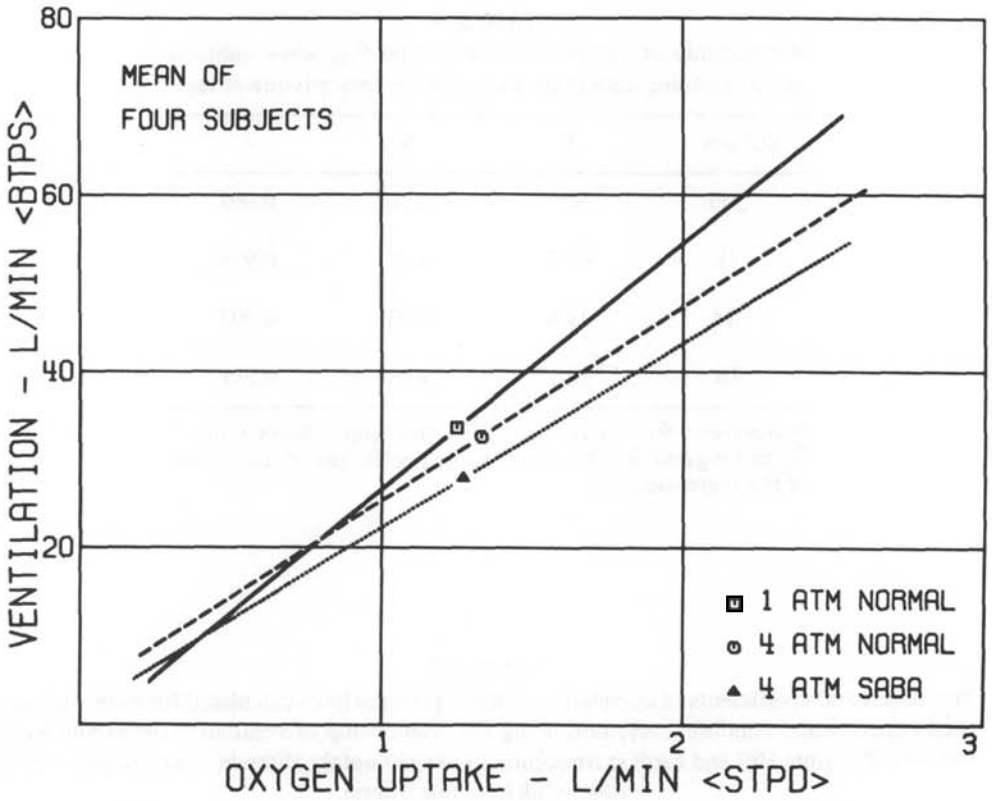

Fig. 1. Relationship of ventilation (liters/min BTPS) to oxygen uptake (liters/min STPD): 1 ATA breathing air; 4 ATA breathing oxygen–nitrogen mixture (P_{O_2} = 0.42 ATA); and 4 ATA breathing oxygen–nitrogen mixture from open-circuit breathing apparatus (SABA). Each regression line represents the mean results of four subjects.

given experimental condition was obtained first to avoid bias. The mean relationship was then derived by using the mean slope and center of gravity of the four regression lines. The slopes and correlations of the regression lines of each subject are shown in Table 7.

The mean relationship between $\dot{V}_E$ and $\dot{V}_{O_2}$ for each experimental condition is shown in Fig. 1 and may be defined by the following equations:

At 1 ATA	$\dot{V}_E = -1.2 + 27.8\,\dot{V}_{O_2}$	(1)
At 4 ATA	$\dot{V}_E = +3.3 + 22.0\,\dot{V}_{O_2}$	(2)
At 4 ATA (SABA)	$\dot{V}_E = +1.2 + 21.0\,\dot{V}_{O_2}$	(3)

The above analysis suggests that at an oxygen uptake of 2.0 liters/min $\dot{V}_E$ is reduced by 13% due to a four-fold increase in gas density and a two-fold increase in oxygen partial pressure and by a further 8% due to SABA, giving a total reduction of 21% in $\dot{V}_E$ compared with normal conditions (1 ATA).

At 1 ATA, the results of only one subject offer a correlation between end tidal P_{CO_2} and $\dot{V}_{O_2}$. At 4 ATA, however, the results of all subjects offer a fair correlation (r represents the correlation coefficient) when breathing from the low-resistance mouthpiece ($0.65 \leq r \leq 0.98$) and a good correlation when breathing from SABA ($0.82 \leq r \leq 0.97$). The data therefore

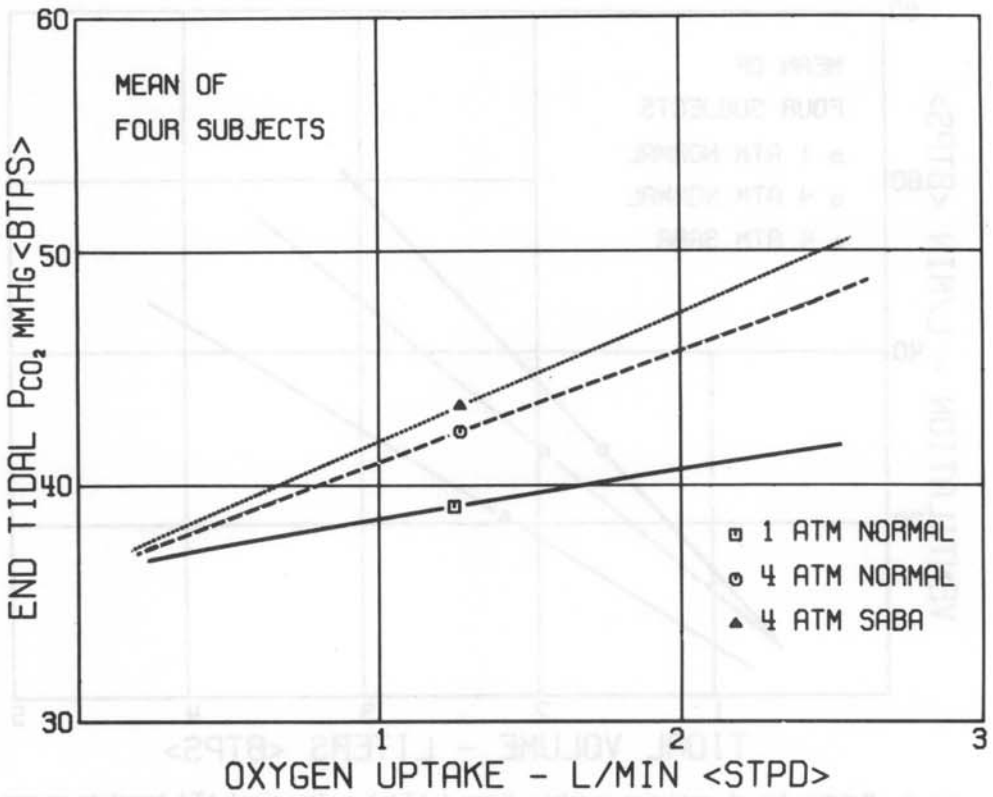

Fig. 2 Relationship of end tidal P_{CO_2} to oxygen uptake: 1 ATA breathing air; 4 ATA breathing oxygen–nitrogen mixture (P_{O_2} = 0.42 ATA); and 4 ATA breathing oxygen–nitrogen mixture from open-circuit breathing apparatus (SABA). Each regression line represents the mean results of four subjects.

support a dependency of end tidal P_{CO_2} on $\dot{V}_{O_2}$ at 4 ATA but not at 1 ATA. The mean regression lines are shown in Fig. 2. At 4 ATA the relationships are defined by the equations:

$$\text{At 4 ATA} \qquad P_{CO_2} = +35.8 + 5.1\,\dot{V}_{O_2} \tag{4}$$

$$\text{At 4 ATA (SABA)} \qquad P_{CO_2} = +36.6 + 5.3\,\dot{V}_{O_2} \tag{5}$$

The relationships represented in Figs. 1 and 2 are not truly linear functions (and in the case of end tidal P_{CO_2} offer a poor fit of the points). Nevertheless, it is considered that as a summary of the results these figures demonstrate clearly the trend in experimental data obtained and, hence, effectively summarize the separate effects of first, pressure and second, breathing apparatus on the respiratory response to exercise.

Oxygen uptake

Although there is a significant increase in oxygen uptake ($\Delta\dot{V}_{O_2}$) observed at pressure when breathing from the low-resistance mouthpiece, the scatter of experimental data makes it impossible to say whether the increase is proportional to activity level (i.e. oxygen uptake).

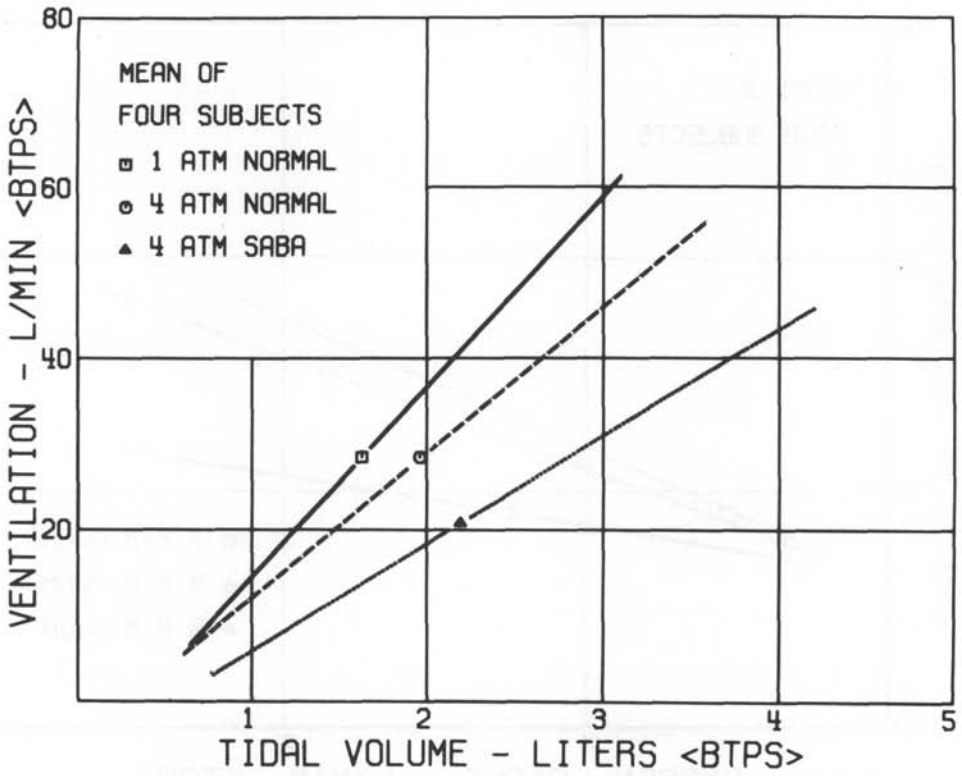

Fig. 3. Relationship of ventilation to tidal volume: 1 ATA breathing air; 4 ATA breathing oxygen–nitrogen mixture (P_{O_2} = 0.42 ATA); and 4 ATA breathing oxygen–nitrogen mixture from open-circuit breathing apparatus (SABA). Each regression line represents the mean results of four subjects.

The regression line of $\Delta\dot{V}_{O_2}$ on $\dot{V}_{O_2}$ representing the data of all four subjects is defined by the equation:

$$\Delta\dot{V}_{O_2} = -0.01 + 0.07\,\dot{V}_{O_2} \tag{6}$$

but has a coefficient of correlation of only 0.45. However, it should be noted that during exercise there is a significant increase of 0.11 liters/min in oxygen uptake ($P < .005$), (yet the reverse is true during rest ($\Delta\dot{V}_{O_2} = -0.03$). When subjects breathed from SABA at 4 ATA, mean $\dot{V}_{O_2}$ again tended to be lower when they were resting and greater during exercise than at 1 ATA, but these changes are not significant.

Respiratory exchange ratio

A comparison of data at all activity levels indicates no significant difference between respiratory exchange ratios (R) at surface and at pressure ($P > .05$). Considered separately, resting respiratory exchange ratios—although highly variable among the subjects—are significantly higher under hyperbaric conditions ($P < .005$). During exercise there was a tendency for R to increase in relation to activity level. However, due to the variability of the individual resting values, there was no distinct correlation between R and $\dot{V}_{O_2}$ either at surface or under pressure.

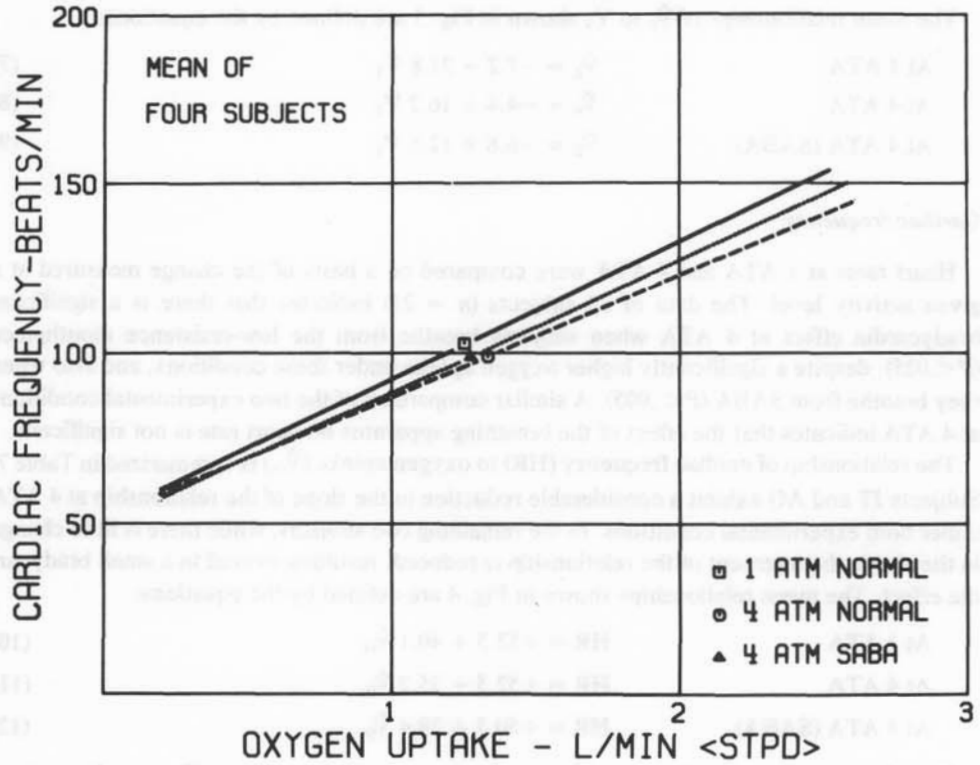

Fig. 4. Relationship of cardiac frequency to oxygen uptake: 1 ATA breathing air; 4 ATA breathing oxygen–nitrogen mixture (P_{O_2} = 0.42 ATA); and 4 ATA breathing oxygen–nitrogen mixture from open-circuit breathing apparatus (SABA). Each regression line represents the mean results of four subjects.

Tidal volume

The relationship of ventilation to mean tidal volume (V_T) has been demonstrated by Hey, Lloyd, Cunningham, Jukes, and Bolton (1966) to be linear up to a tidal volume of approximately 50% of vital capacity. At this point V_T tends to remain relatively constant and independent of further increases in ventilation. The relationship described by Hey et al. was also observed in this study. Hence, the regression line relating $\dot{V}_E$ to V_T of each subject was calculated initially by including only data points having a $V_T \leq$ 50% vital capacity. Data points having a $V_T >$ 50% vital capacity were then considered to be dependent if a good fit was obtained to the assumed relationship. If, however, a disproportionate increase in $\dot{V}_E$ was observed, the data points were rejected as independent of the relationship. A regression line was then calculated for all dependent data points.

There is considerable variation in the relationships exhibited by the four subjects (Table 7), but in all cases there is a good correlation between $\dot{V}_E$ and V_T ($0.88 \leq r \leq 1.0$). In each individual the slope of the relationship is reduced at 4 ATA indicating that ventilation is achieved by a larger tidal volume and lower respiratory frequency than at surface. At 4 ATA the addition of SABA resulted in a further decrease in slope in three subjects.

A comparison of tidal volumes at surface and under pressure using only the data which is ventilation-dependent indicates that there is a significant increase in tidal volume ($P < .005$) at 4 ATA and a further significant increase ($P < .05$) due to the breathing apparatus.

The mean relationships of $\dot{V}_E$ to V_T shown in Fig. 3 are defined by the equations:

At 1 ATA	$\dot{V}_E = -7.2 + 21.8\ V_T$	(7)
At 4 ATA	$\dot{V}_E = -4.4 + 16.7\ V_T$	(8)
At 4 ATA (SABA)	$\dot{V}_E = -6.8 + 12.6\ V_T$	(9)

Cardiac frequency

Heart rates at 1 ATA and 4 ATA were compared on a basis of the change measured at a given activity level. The data of all subjects ($n = 21$) indicates that there is a significant bradycardia effect at 4 ATA when subjects breathe from the low-resistance mouthpiece ($P<.025$), despite a significantly higher oxygen uptake under these conditions, and also when they breathe from SABA ($P< .005$). A similar comparison of the two experimental conditions at 4 ATA indicates that the effect of the breathing apparatus on heart rate is not significant.

The relationship of cardiac frequency (HR) to oxygen uptake ($\dot{V}_{O_2}$) is summarized in Table 7. Subjects JT and AG exhibit a considerable reduction in the slope of the relationship at 4 ATA under both experimental conditions. In the remaining two subjects, while there is little change in the slope, the intercept of the relationship is reduced, resulting overall in a small bradycardia effect. The mean relationships shown in Fig. 4 are defined by the equations:

At 1 ATA	$HR = +52.5 + 40.1\ \dot{V}_{O_2}$	(10)
At 4 ATA	$HR = +52.5 + 35.2\ \dot{V}_{O_2}$	(11)
At 4 ATA (SABA)	$HR = +50.3 + 38.4\ \dot{V}_{O_2}$	(12)

Equations 11 and 12 indicate that at 4 ATA there is a bradycardia effect at all levels of oxygen uptake, and that the magnitude of bradycardia (ie. beats/min) tends to increase with exercise intensity. Thus, at an oxygen uptake of 2.0 liters/min the heart rate is reduced by approximately 10 beats/min (7%) and 6 beats/min (4%), respectively, in comparison to surface values.

DISCUSSION

The principal feature of the results presented is the reduction in ventilation equivalent and elevation of end tidal P_{CO_2} occurring at 4 ATA. Hypoventilation and carbon dioxide retention under pressure has been shown to be caused partly by increased oxygen partial pressure and partly by increased ambient (or inert gas) pressure (Lanphier 1963; Hesser et al. 1968). In addition, Lanphier (1963) has shown that during exercise carbon dioxide retention due to inert gas pressure is related to the corresponding increase in gas density rather than the absolute pressure. For this reason, hypoventilation and carbon dioxide retention effected by increased inert gas pressures have been ascribed to the increased work of breathing (Lanphier 1969).

The further hypoventilation induced at 4 ATA when breathing from SABA most probably results from a further increase in the work of breathing due to added external resistance. This additional effect has been shown to be present during exertion at normal barometric pressures by Silverman, Lee, Plotkin, Sawyers, and Yancey (1951) and Tabakin and Hanson (1960); a similar tendency has also been noted by Lanphier (1963) and Salzano et al. (1970) under hyperbaric conditions when external resistance has been altered. In this study it is notable that the effect of the breathing apparatus on ventilatory response to exercise is variable; only two of the four subjects exhibited an increased degree of hypoventilation.

Although the increases in end tidal P_{CO_2} during exercise are of a similar magnitude to those measured by Lanphier (1963) and Hesser et al. (1968), it is notable that the absolute values in the present study tend to be considerably lower. This can be explained by the difference in ventilatory response to exercise at normal barometric conditions, where the divers in the above two studies exhibited considerable carbon dioxide retention. Such behavior may be a feature of certain divers, although existing evidence of this is inconclusive (Goff and Bartlett 1957; Froeb 1960; Lanphier 1969; Broussolle, Bensimon, Michaud, and Vegezzi 1970; Sterk 1972; Lally, Zechman, and Tracy 1974). Hence, although this study demonstrates the effects of increased pressures and breathing apparatus on the normal respiratory response to exercise, it may underestimate the degree of hypoventilation and carbon dioxide retention present in some divers who are described as "carbon dioxide retainers" (Lanphier 1969).

The increased oxygen uptake observed when exercising at 4 ATA is most probably due to the work of breathing a dense gas mixture. A similar effect has been shown at comparable gas densities but different pressures (Hesser et al. 1968; Salzano et al. 1970; Schaefer, Carey, and Dougherty 1970); the increased oxygen cost of breathing was estimated to be 0.1 to 0.25 liters/min during moderate to heavy exercise. At normal barometric pressure, Glauser, Glauser, and Rusy (1967) have estimated the work of breathing 7% CO_2 in air to be 2.6 ml O_2/liter $\dot{V}_E$. Assuming this value for air at 1 ATA, the results of the present study (from Equations 1 and 6) suggest the work of breathing the oxygen–nitrogen mixture at 4 ATA to be increased to approximately 5.1 ml O_2/liter $\dot{V}_E$. This value is somewhat lower than has been estimated by Glauser et al. (1967—7.7 ml O_2/liter $\dot{V}_E$ with a breathing mixture of 7% CO_2, 20% O_2, and 73% SF_6 at 1 ATA) and Salzano et al. (1970—8–10 ml O_2/liter $\dot{V}_E$ with a breathing mixture of 0.9% O_2, 99.1% helium at 31.3 ATA). However, the different gas mixtures and ambient pressures employed may account in part for the variance of the above results.

It is of interest to note that when subjects breathed from SABA at 4 ATA, the oxygen uptake at a given activity level was less than when they breathed with minimal external resistance at the same pressure and was not significantly greater than at 1 ATA. This result is somewhat contradictory, as the work of breathing was presumably greatest when the apparatus was used (this assumption is supported by the reduction of ventilatory response to exercise and corresponding hypercapnia shown in Figs. 1 and 2). Although there is no comparable study under hyperbaric conditions, it has been shown that during exercise at normal barometric pressure the imposition of external resistance to ventilation may cause a decrease in oxygen uptake (Silverman et al. 1951; Tabakin and Hanson 1960). In addition, Thompson and Sharkey (1966) have shown that when subjects breathe against the external resistance of a respiratory protective mask, the exercise heart rate is not significantly increased but there is a significant increase in recovery oxygen consumption; this suggests that oxygen debt contracted during exercise is greater than normal. It would seem odd that increases in internal and external resistance should have the opposite effects on oxygen uptake during exercise, but it is possible that the greater oxygen uptake measured when dense gases are breathed reflects only part of the increased work of breathing and the remainder is met by an increased oxygen debt, as would appear to be the case in the presence of external resistance. The subject requires further investigation, but from our results and from investigations at normal barometric pressure, it appears that use of an underwater breathing apparatus when subjects breathe dense gas mixtures under hyperbaric conditions may cause a reduction in oxygen uptake, an increased oxygen debt, and possibly higher levels of lactate concentration.

It has been shown by Hey et al. (1966) that the relationship of ventilation to tidal volume is essentially unaltered by changes in P_AO_2 or P_ACO_2. Thus, the alteration in respiratory pattern observed at 4 ATA would appear to be related to increased pressure or some related factor

(e.g. work of breathing). It is probable that the further reduction in the slope of the relationship effected by SABA also relates in some way to the increased work of breathing. Increases in tidal volume during exercise at pressure have been noted by Hesser et al. (1968) and Salzano et al. (1970), and Morrison and Florio (1971) have shown a possible correlation between gas density (or pressure) and the tidal volume selected at a given ventilation. Silverman et al. (1951) have shown that during exposure to increased external resistance, ventilation is achieved by a larger tidal volume and lower respiratory frequency.

Mead (1963) has postulated that respiratory frequency is selected to produce the smallest possible average force developed by the respiratory muscles and that this frequency may be slightly higher than that at which respiratory work is minimal, yet Otis, Fenn, and Rahn (1950) have suggested that frequency may be selected to minimize respiratory work. A third factor which may be involved is the ratio of work performed by the inspiratory and expiratory muscles. As an increase of gas density would not affect the elastic component of inspiratory work, expiratory work of breathing might increase disproportionately to inspiratory work. An increase in tidal volume and lower respiratory frequency would tend to redress this ratio.

The bradycardia effect observed in this study has been measured by other workers in experiments at various pressures. Although part of the bradycardia effect may be attributed to increased oxygen partial pressure, Hesser et al. (1968) and Schaefer et al. (1970) have shown that it results partly from increased inert gas pressure (or gas density), although the mechanism involved is not clear. The extent of bradycardia measured in the present study appears to be much smaller than in comparable studies in which an oxygen–helium mixture has been breathed at very high pressures (Salzano et al. 1970; Schaefer et al. 1970; Morrison and Florio 1971), although the oxygen partial pressures breathed in these studies ($0.3 \leq P_{O_2} \leq 0.6$) are relatively similar. Both Salzano et al. (1970) and Morrison and Florio (1971) have observed that the bradycardia seen at very high pressures does not appear to correlate linearly with absolute pressure. One possible explanantion is that the bradycardia is partly due to a decrease of skin temperature and peripheral vasoconstriction resulting from the high thermal conductivity of the oxygen–helium mixture of which the surrounding environment is composed (Raymond, Bell, Bondi, and Lindberg 1968). Alternatively, Schaefer et al. (1970) have suggested that the bradycardia noted during chamber diving is the result of a vagal stimulation produced by exposure to high pressures.

The increased oxygen pulse observed at 4 ATA indicates that either there is an increased oxygen extraction from a given volume of blood and cardiac output is reduced, or there is an increase in stroke volume with cardiac output equal to or greater than that at 1 ATA. An investigation by Spioch, Kobza, and Rump (1962) has shown that the imposition of external resistance to ventilation in the form of a respirator causes a significant increase in systolic blood pressure, stroke volume, and cardiac output, while heart rate and oxygen uptake remain relatively unchanged. These effects may result from the greater intrathoracic pressures necessary to maintain ventilation, and it is possible that similar changes occur under hyperbaric conditions, particularly in the presence of underwater breathing apparatus.

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Morrison, J.B., W.S. Butt, J.T. Florio, and I.C. Mayo. 1976. Les effets d'une pression accrue de O_2–N_2 et de l'appareil respiratoire sur la fonction respiratoire. Undersea Biomed. Res. 3(3): 217–

234.—La réponse ventilatoire a été mesurée chez quatre sujets en repos et à des intensités d'exercice diverses. Les expériences ont eu lieu dans une chambre de pression séche à l et 4 ATA pendant que les sujets respiraient à travers une embouchure à resistance basse, et à 4 ATA pendant que les sujets respiraient à l'aide du Swimmers' Air Breathing Apparatus de la Marine Royale. A 4 ATA il existait une hypoventilation significative et une hypercapnie, avec un volume respiratoire accru et une fréquence respiratoire diminuée. L'emploi de l'appareil respiratoire exaggérait ces altérations de réponse ventilatoire. De plus, l'hypercapnie à 4 ATA était en rapport direct avec l'intensité de l'exercice. Pendant la respiration avec l'embouchure à résistance basse, la consommation d'oxygéne était plus grande (différence significative) à 4 ATA qu'a la surface pour la même charge ergométrique, mais pendant la respiration avec l'appareil, l'augmentation de la consommation n'était pas significative par rapport aux valeurs témoins. A 4 ATA une bradycardie a été constatée à tous les niviaux ergométriques, sans rapport à l'appareil respiratoire.

plongée exercice — consommation d'oxygène
appareil respiratoire — hypercapnie
ventilation — bradycardie

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