Effect of increased respiratory resistance on carbon dioxide levels and hemodynamics in the submerged exercising diver

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Introduction

During diving, hypercapnia occurs at rest as a result of high airway resistance due to increased gas density and the external resistance of the breathing apparatus[1]. Exercise while diving can significantly increase hypercapnia, which impairs exercise duration, exercise magnitude, and mental performance. End-tidal PCO_2 ($P_{ET}CO_2$) is conventionally used to evaluate hypercapnia during underwater breathing apparatus (UBA) testing. However, $P_{ET}CO_2$ underestimates arterial PCO_2 (P_aCO_2) when airway resistance is high (due to respiratory disease or in diving[2]). Increased airways resistance in these conditions is inhomogeneous and results in dilution of alveolar gas by gas from under-perfused and over-ventilated gas exchange units. This leads to a reduction in $P_{ET}CO_2$ relative to the arterial value (fig. 1b). On the other hand, during exercise $P_{ET}CO_2$ can exceed P_aCO_2[3,4]. Mixed venous PCO_2 during exercise is increased, and because of ongoing diffusion of CO_2 into the alveolus during exhalation, the end-tidal alveolar PCO_2 approaches that of the mixed venous blood and causes $P_{ET}CO_2$ to rise above P_aCO_2 (fig. 1c). Depending on which of these factors predominates, $P_{ET}CO_2$ in an exercising diver could be either higher or lower than P_aCO_2 (fig. 1d). This study examined the effect of varying levels of respiratory resistance during exercise at depth on work of breathing/volume (WOB/V), peak-to-peak mouthpiece pressures (Pm pk-pk), minute ventilation (V_E), cardiac output (CO), and the relationship between $P_{ET}CO_2$ and P_aCO_2. We hypothesized that 1) increased Pm pk-pk (and thus increased preload during inspiration) due to increased breathing resistance would cause an increase in CO, and 2) that $P_{ET}CO_2$ would underestimate P_aCO_2 during exercise at depth because the end-tidal to arterial gradient seems to be affected to a higher degree by exercise than by increased deadspace.

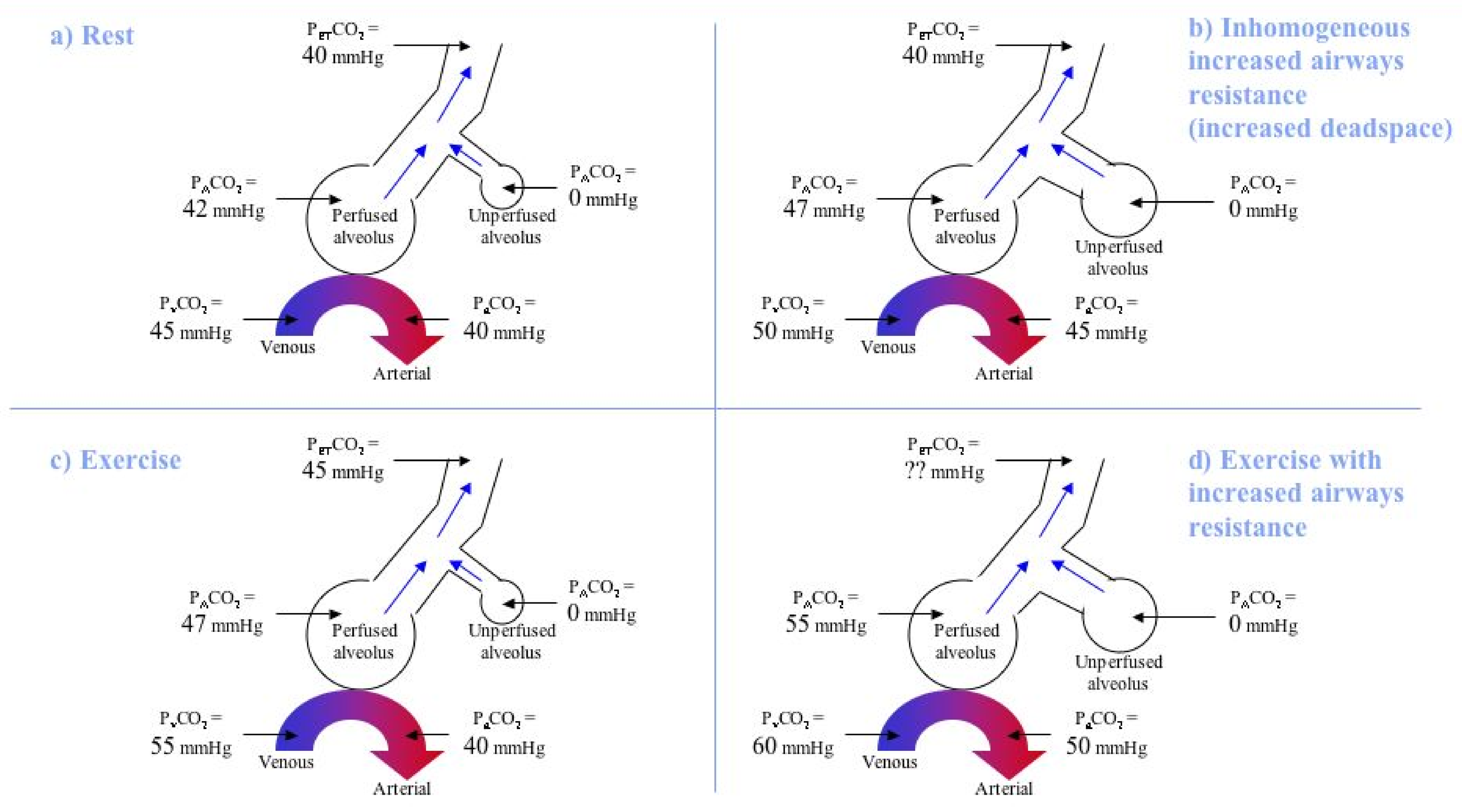

Figure 1. Schematic of the relative contribution of perfused and unperfused alveoli to the $P_{ET}CO_2$ under various conditions.

Methods

After institutional approval and informed consent volunteers were studied during moderate exercise while breathing air 1) upright and dry, then prone while submersed in thermoneutral water, 2) at the surface, and 3) with various levels of breathing resistance (to simulate an imperfect breathing apparatus) at a simulated depth of 122 feet of seawater (fsw) (fig.2). The order of surface dry, surface submersed, and depth submersed conditions was not randomized due to logistical concerns. The order of resistance levels at depth was varied. Twenty-seven subjects were studied: 19 at depth without external breathing resistance; 8 had added resistance, which was either inspiratory (I) or both inspiratory and expiratory (IE). Each subject was instrumented with an ECG and radial and pulmonary artery catheters. For each resistance, measurements of inspiratory/expiratory flow rates and pressures, inspired/expired gas concentrations, $P_{ET}CO_2$ and P_aCO_2 were made during the last minute of a 6 minute exercise.

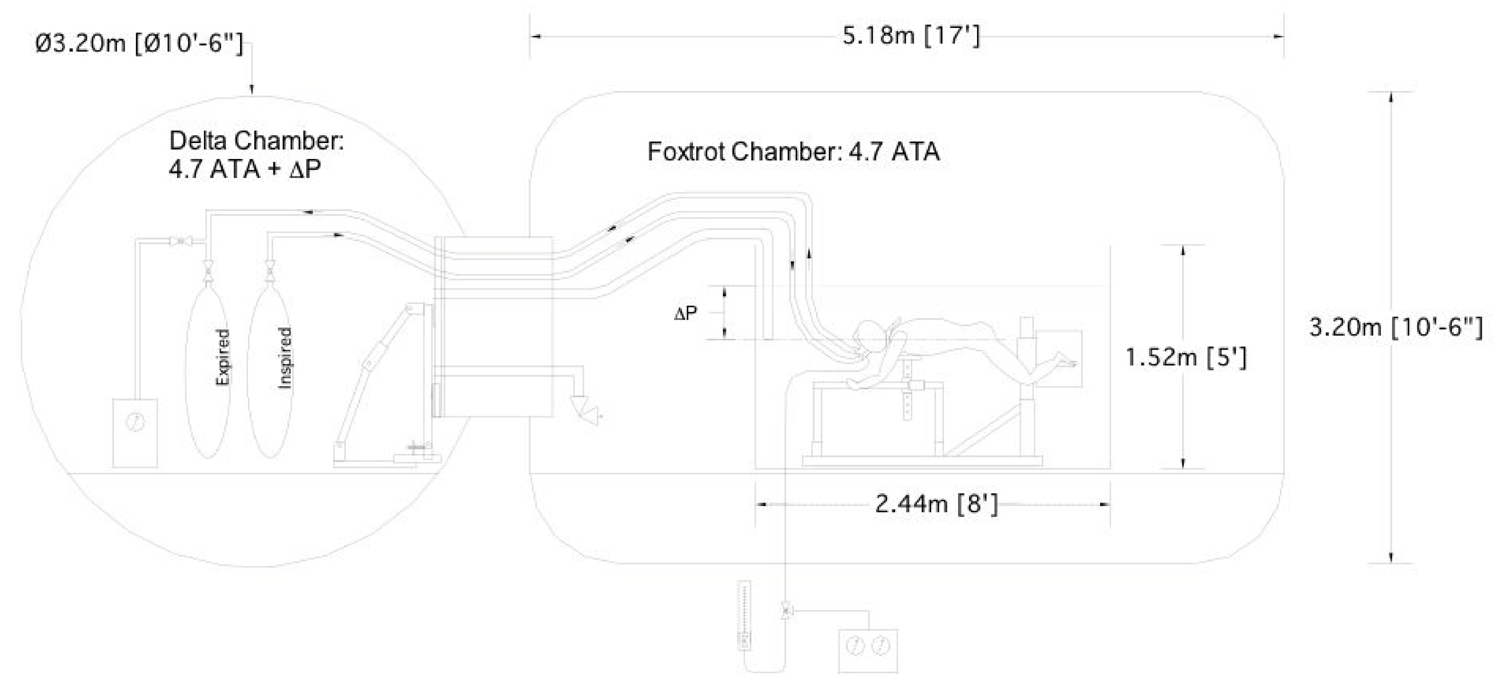

Figure 2. Subjects are placed prone in the pool and exercise using an electrically braked cycle ergometer. Breathing gas comes from separate chamber (Delta) which is at a pressure equal to the immersed subjects' pressure, Foxtrot pressure plus the extra pressure from the water.

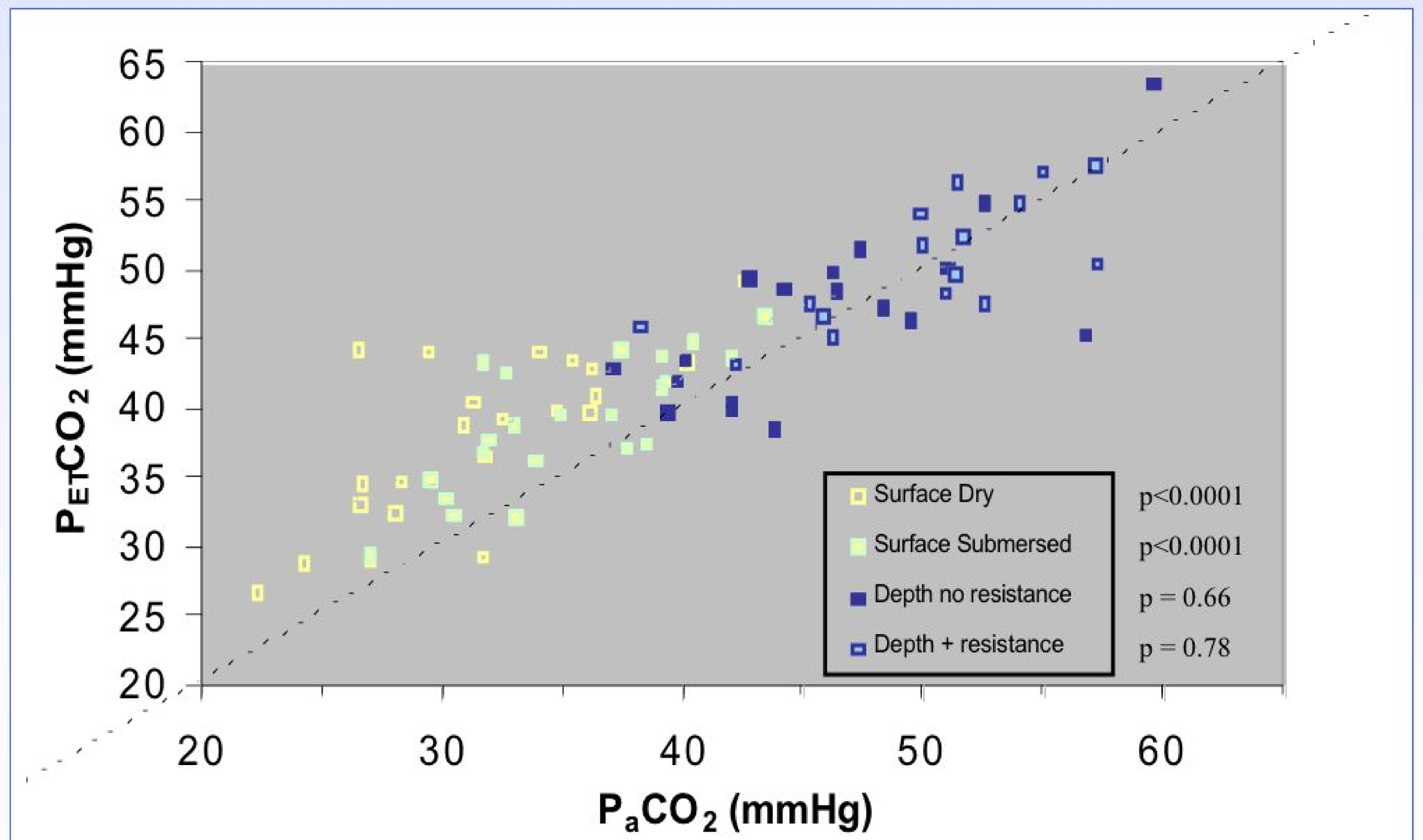

Figure 3. P_aCO_2 vs. $P_{ET}CO_2$ during exercise dry, immersed at the surface and at depth, and immersed at depth with added breathing resistance. P values are for comparison of the averages of $P_{ET}CO_2$ and P_aCO_2 via paired Student's t-tests.

Results

VO_2 during exercise was 30.1±5.1 mL/(kg*min) (mean ± SD). Pm pk-pk was 6.8±2.2 cmH_2O without external breathing resistance. With resistance (I or IE), Pm pk-pk ranged from 17.7 to 53 cmH_2O and WOB/V from 1.46 to 4.49 J/L.

- CO remained unchanged despite varying resistances
- P_aCO_2 increased during exercise with submersion, at depth, and with resistance at depth
- $P_{ET}CO_2$ higher than P_aCO_2 at the surface during dry and submersed exercise
- $P_{ET}CO_2$ and P_aCO_2 highly correlated during exercise at depth
- V_E decreased and V_d/V_t increased during exercise with submersion, at depth, and depth + resistance

Condition	P_aCO_2	P	$P_{ET}CO_2$	pH arterial	Fick CO	P	V_E	P	V_d/V_t	P
Surface dry exercise	31.6±5.2		38.0±6.1	7.29±0.05	16.8±3.6		89.7±23.1		0.18±0.13	
Surface sub exercise	34.2±4.9	0.02*	38.8±4.7	7.31±0.04	17.7±2.9	NS*	77.1±20.5	0.02*	0.23±0.08	0.03*
Depth exercise no resist	46.1±5.9	<0.005§	46.8±6.2	7.29±0.04	18.1±2.9	NS§	61.6±13.9	<0.005§	0.29±0.07	<0.005§
Depth exercise + resist	49.3±4.3	0.03–	50.2±5.3	7.29±0.03	17.7±3.3	NS–	53.4±8.0	0.01–	0.31±0.05	NS–

Table 1. Means and standard deviations of selected parameters. P values for the mean of P_aCO_2 vs. $P_{ET}CO_2$ appear in figure 3. P values for P_aCO_2, CO, V_E and V_d/V_t were determined by paired Student's t-tests for *submersed vs. dry exercise, §depth vs. submersed exercise, and by unpaired Student's t-test for –depth + resistance vs. depth exercise.

Conclusions

- With external breathing resistances that have been reported to be acceptable[1], moderate hypercapnia occurs in the exercising diver
- The effects of increased airways resistance and exercise on the relationship between $P_{ET}CO_2$ and P_aCO_2 appear to counteract one another
- Submersion, depth, and breathing resistance do not appear to affect CO during exercise
- Increased respiratory deadspace and decreased minute ventilation with increased respiratory resistance during diving are a likely cause of the hypercapnia observed

References

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