The effects of P_{IO_2} on respiratory control during immersed exercise at 122 fsw

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INTRODUCTION

Objective: To investigate the effects of hyperoxia on ventilation and P_{aCO_2} during immersed exercise compared with normoxia

•Hypercapnia poses a risk to divers as it can produce troublesome symptoms (headache, nausea, dizziness, loss of consciousness) and potentiate the effects of nitrogen narcosis and CNS oxygen toxicity.

•At 1 atmosphere absolute (ATA) respiratory drive usually maintains normocapnia in the face of varying gas exchange conditions.

• Ventilation during diving is usually lower than at the surface (thus arterial P_{CO_2} is higher).
  – Reduced lung compliance (immersion → redistribution of blood from extremities into pulmonary vessels) → higher work of breathing
  – Increased flow resistance (increasing depth → higher breathing gas density) → CO_2 retention

•Divers often breathe gas mixtures with higher proportions of O_2 to decrease the deleterious effects of inert gas at depth.

• While high inspired P_{O_2} (P_{IO_2}) can reduce respiratory drive as measured by the ventilatory response to hypercapnia, hyperoxia over the range 0.7-1.3 ATA has not been definitively shown to affect respiration during immersed exercise [1].

•Ventilation has been shown to increase during prolonged immersed exercise (beyond 12 min duration) at surface and at depth of 55 fsw, which has been attributed to respiratory fatigue and metabolic acidosis [2, 3].

Hypothesis 1: Ventilation during immersed exercise is lower with hyperoxia (1.75 ATA) compared with normoxia (0.21 ATA).

Hypothesis 2: This increase in ventilation during immersed exercise and accompanying metabolic acidosis are attenuated by hyperoxia compared with normoxia.

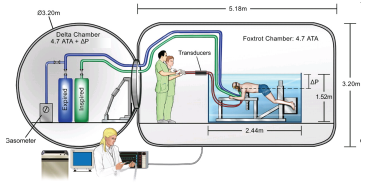
METHODS

Subjects: After institutional approval and informed consent, we studied 10 volunteers: 8 male, 2 female, age range 20-44 with minimum VO_{2max} 30 ml•min^{-1}•kg^{-1} (female) or 35 ml•min^{-1}•kg^{-1} (male)

Equipment:
- Electronically braked ergometer immersed in thermoneutral water in hyperbaric chamber
- Adjacent hyperbaric chamber houses breathing gases, pressurized to match depth of subject

Immersed exercise: 15-17 min moderate intensity (O_2 consumption typically 1.8-2.6 L•min^{-1}) in prone position
- Surface, room air (0.21 ATA P_{IO_2})
- Depth 122 fsw, 0.21 ATA P_{IO_2} (normoxia)
- Depth 122 fsw, 1.75 ATA P_{IO_2} (hyperoxia)

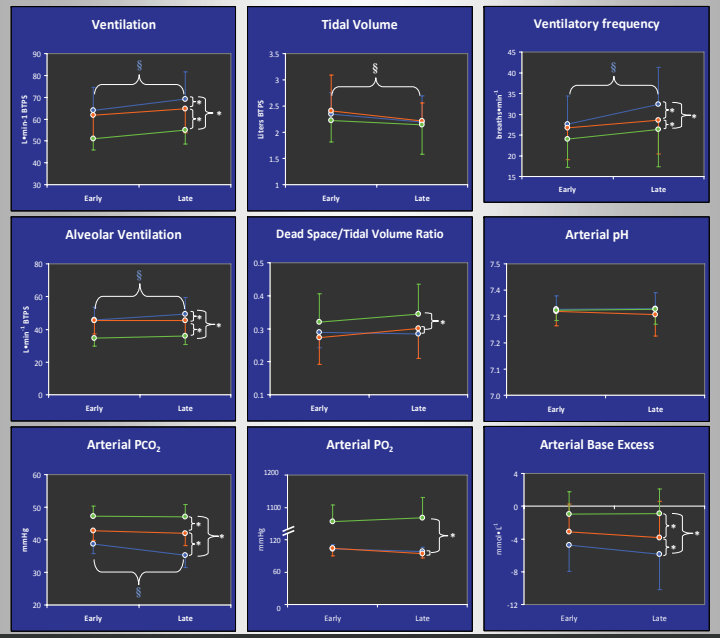
Monitoring: EKG, pulmonary and radial arterial catheter



Measurements:
- Expired gas collected in early (6 min) and late exercise (16 min)
  - V_E, V_A, f, V_T
- Arterial and venous blood collected in early and late exercise
  - pH, P_{aCO_2}, P_{aO_2}, arterial BE

Data Analysis:
Repeated measure general linear model with Tukey-Kramer *post hoc* tests for pairwise comparisons of significant predictors (SAS Enterprise Guide, Cary, NC)

RESULTS



Above: Mean ± SD shown for Surface (●); Depth 122 fsw, 0.21 ATA P_{IO_2} (●); Depth 122 fsw, 1.75 ATA P_{IO_2} (●)
$p < 0.05$ among conditions (*), $p < 0.05$ between early and late exercise (§).

- Ventilation (V_E)
  - In normoxia, V_E was reduced at depth compared to surface (63.5 depth vs. 67.9 L•min^{-1} surface, p=0.025). At depth, hyperoxia further reduced VE compared to normoxia (53.1 hyperoxia vs. 67.9 L•min^{-1}, p < 0.0001).
  - V_I increased from early to late exercise at the surface (64.1 early vs. 71.7 L•min^{-1} late, p= 0.016), but not significantly at depth. The increase seen at the surface is attributable to an increase f.
- Ventilatory frequency (f)
  - f was reduced at depth compared to surface (25.1 depth hyperoxia vs. 30.3 breaths•min^{-1} surface, p<0.0001, 28.0 depth normoxia vs. 30.3 breaths•min^{-1} surface, p=0.037). At depth, f was reduced by hyperoxia compared to normoxia (25.1 hyperoxia vs. 28.0 breaths•min^{-1} normoxia, p=0.008).
  - f increased from early to late exercise at surface (27.6 early vs. 33.0 breaths•min^{-1} late, p = 0.001), but not significantly at depth.
  - Tidal Volume (V_T) decreased from early to late exercise (2.34 L early vs. 2.19 L late, p = 0.044). No difference in V_T among the three conditions.

RESULTS (continued)

- Dead space/tidal volume ratio (V_D/V_T)
  - V_D/V_T was higher with hyperoxia at depth compared to normoxia and surface (0.33 depth hyperoxia vs. 0.29 depth normoxia, p=0.0032, 0.33 depth hyperoxia vs. 0.29 surface, p=0.0018). There was no effect of depth alone on V_D/V_T in normoxia.
  - There was no effect of exercise on V_D/V_T.
- Alveolar ventilation (V_A)
  - In normoxia, depth reduced VA compared to surface (45.1 depth vs. 48.3 L•min-1, p = 0.026). At depth, VA was further reduced by hyperoxia compared to normoxia (35.3 hyperoxia vs. 45.1 L•min^{-1} normoxia, p < 0.0001). V_A increased from early to late exercise at the surface (45.6 early vs. 51.0 L•min^{-1} late, p= 0.024), but not significantly at depth.
- P_{aO_2} was constant at the same P_{IO_2} regardless of depth or exercise.
- P_{aCO_2}
  - increased significantly from surface to depth (37.0 surface vs. 42.6 mmHg normoxia at depth, p<0.0001; 42.6 surface vs. 47.1 mmHg with hyperoxia, p <0.0001).
  - At depth, P_{aCO_2} was higher with hyperoxia compared to normoxia (42.6 low vs. 47.1 mmHg high, p <0.0001).
  - decreased slightly from early to late exercise at surface (38.7 early vs. 35.2 mmHg late, p = 0.013). No significant change at depth.
- Acidosis
  - There was no significant difference in pH among the conditions, or from early to late exercise.
  - The reduction in arterial base excess was attenuated in both conditions at depth (-3.54 normoxia at depth vs. -5.66 mmol•L^{-1} surface, p=0.0005; -0.94 depth hyperoxia vs. -5.66 mmol•L^{-1} surface, p<0.0001), and to a greater extent with hyperoxia compared with normoxia (-0.94 hyperoxia vs. -3.54 mmol•L^{-1} normoxia at depth, p = 0.0005).
  - There was no change in arterial BE from early to late exercise.

CONCLUSIONS

• V_E and V_A were reduced, and P_{aCO_2} and V_D/V_T increased by hyperoxia during immersed exercise at 122 fsw. While pH did not change during exercise, a reduction in arterial BE was reduced by hyperoxia at depth. We conclude that the reduced ventilation seen in hyperoxia at depth occurs by depressing respiratory drive, possibly via an amelioration of mild metabolic acidosis in combination with an effect on the peripheral O_2 chemoreceptors.

• The expected rise in V_E did not occur to a significant degree at 122 fsw.

REFERENCES

1. Cherry AD, Forkner IF, Frederick HJ, Natoli MJ, Schinazi EA, Longphre JP, Conard JL, White WD, Freiberger JJ, Stolp BW, Pollock NW, Doar PO, Boso AE, Alford EL, Walker AJ, Ma AC, Rhodes MA, Moon RE. J Appl Physiol 106:316-325, 2009.
2. Wylegala JA, Pendergast DR, Gosselin LE, Warkander DE, Lundgren CEG. Respiratory muscle training improves swimming endurance in divers. Eur J Appl Physiol 99:393-404, 2007.
3. Ray AD, Pendergast DR, Lundgren CEG. Respiratory muscle training improves swimming endurance at depth. UHM 35(3):185-196, 2008.

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