The effects of P_{IO_2} on pulmonary hemodynamics during immersed exercise at 122 fsw

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INTRODUCTION

Objective: To investigate the effects of hyperoxia on pulmonary hemodynamics during immersed exercise compared with normoxia

- Immersion pulmonary edema (IPE)
  - First described by Wilmshurst, et al. in cold water divers [1]
  - Acute noncardiogenic accumulation of fluid in lungs during diving and surface swimming
  - Occurs in otherwise healthy divers
  - Pathophysiology poorly understood
    - Proposed risk factors: hypertension, cold water diving, tight wetsuit, increased age
- High pulmonary vascular pressures may play a role in pathophysiology of IPE [2,3]
  - Pulmonary arterial system is usually low resistance and low pressure
  - In diving, exercise and immersion increase PA pressure
    - Exercise → increased cardiac output (CO)
      - Metabolic acidosis, catecholamines → vasoconstriction
    - Immersion and low ambient temperatures → central redistribution of blood from extremities, increased CO
  - O_2 is a vasodilator of pulmonary vessels.
- Ventilation has been shown to increase during prolonged immersed exercise (beyond 12 min duration) at surface and at depth of 55 fsw. This has been suggested to be due to respiratory fatigue and metabolic acidosis [4,5].
  - Acidosis would be expected to exacerbate high PA pressures

•**Hypothesis**: Pulmonary artery pressure will increase during prolonged immersed exercise, and this increase will be attenuated by hyperoxia compared to normoxia.

METHODS

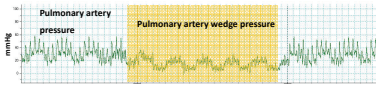
Subjects: After institutional approval and informed consent, we studied 10 volunteers: 8 male, 2 female, age range 20-44 with minimum VO_{2max} 30 ml•min^{-1}•kg^{-1} (female) or 35 ml•min^{-1}•kg^{-1} (male)

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_{IO2})
- Depth 122 fsw, 0.21 ATA P_{IO2}
- Depth 122 fsw, 1.75 ATA P_{IO2}



Above: Pulmonary artery tracing during exercise

Monitoring: EKG, pulmonary and radial arterial catheter

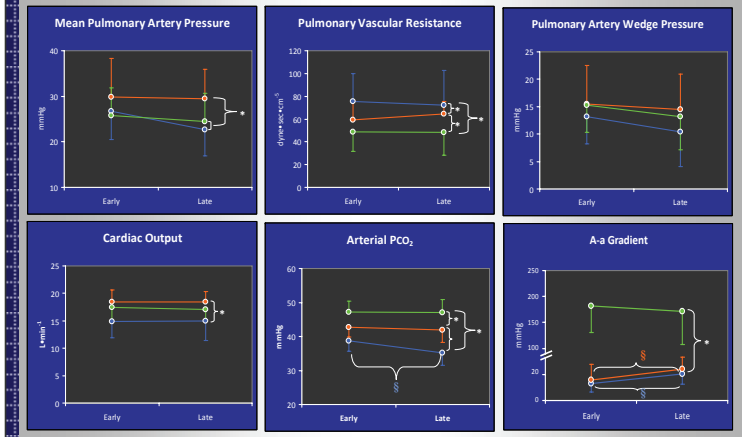
Measurements:

- Hemodynamics measured continuously
  - MPAP, PAWP, PVR, cardiac output
- Arterial and venous blood collected in early (6 min) and late exercise (16 min)
  - pH, P_{aCO_2}

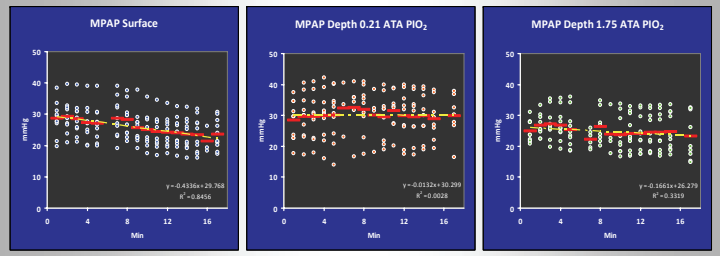
Data Analysis:

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 (§).



Above: Individual data points at Surface (○); Depth 122 fsw, 0.21 ATA P_{IO_2} (●); Depth 122 fsw, 1.75 ATA P_{IO_2} (●)
Mean for each time point (—)

- Mean pulmonary artery pressure (MPAP)
  - In normoxia, MPAP was lower at the surface compared with depth (24.7 surface vs. 29.4 mmHg depth, p=0.001); hyperoxia decreased MPAP at depth (25.1 hyperoxia vs. 29.4 mmHg normoxia, p=0.0027).
  - Continuous measurement of MPAP during exercise showed a trend of decreasing MPAP from early to late exercise, which was significant at the surface (p<0.0001) but not at depth.

RESULTS (continued)

- Pulmonary vascular resistance (PVR)
  - PVR was lower with hyperoxia at depth compared with normoxia and surface (48.5 hyperoxia vs. 60.6 dyne•sec•cm^{-5}, p=0.035; 48.5 hyperoxia vs. 72.7 dyne•sec•cm^{-5} surface, p<0.0001). PVR was lower at depth compared to surface in normoxia (60.6 depth with normoxia vs. 72.7 dyne•sec•cm^{-5} surface, p=0.035).
  - There was no difference in PVR from early to late exercise.
- Pulmonary artery wedge pressure (PAWP): no difference among the conditions, or from early to late exercise.
- Cardiac Output (CO)
  - CO was lower at the surface than at depth with normoxia (15.0 surface vs. 20.4 L•min^{-1} depth). There was no difference in CO from early to late exercise.
- PAO_2- Pao_2 difference (A-a gradient)
  - A-a gradient increased from early to late exercise at surface (12.8 early vs. 20.4 mmHg late, p=0.03) and depth with normoxia (15.6 early vs. 24.6 mmHg late, p=0.007). There was no significant difference between surface and depth in normoxia.
  - In hyperoxia, A-a gradient did not change significantly from early to late exercise.
- Acidosis
  - There was no significant difference in pH among the conditions (7.33 ± 0.05 surface, 7.31 ± 0.07 normoxia at depth, 7.32 ± 0.05 hyperoxia at depth, NS), or from early to late exercise (7.32 ± 0.05 early, 7.32 ± 0.06 late, NS).
  - 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 hyperoxia at depth vs. -5.66 mmol•L^{-1} surface, p<0.0001), and to a greater extent with hyperoxia compared with normoxia at depth (-0.94 hyperoxia vs. -3.54 mmol•L^{-1} normoxia, p = 0.0005).
- V_E 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.
- $Paco_2$
  - increased significantly from surface to depth (37.0 surface vs. 42.6 mmHg with normoxia, p<0.0001; 42.6 surface vs. 47.1 mmHg with hyperoxia, p <0.0001).
  - At depth, $Paco_2$ was higher with hyperoxia compared to normoxia (42.6 high vs. 47.1 mmHg low, 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 conditions.

CONCLUSIONS

Mean PA pressure in these healthy subjects varies substantially during prolonged immersed exercise to values that exceeded 40 mmHg. In normoxia, MPAP was increased at depth compared to surface. This effect was not seen with hyperoxia at depth.

The increase in V_E in late exercise at surface was not associated with a change in arterial pH.

Continuous measurement of mean PA pressure demonstrated a significant decrease over 16 min exercise at the surface. This effect was not seen at depth.

Although no manifestations of pulmonary edema occurred in these subjects, the increase A-a gradient in normoxia is consistent with an increase in lung water.

REFERENCES

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ACKNOWLEDGMENTS

Supported by NAVSEA contract #N0463A-07-C-0002 and NIH #TL1 RR024126