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Title: Cerebral oxygenation in highlanders with high altitude pulmonary hypertension

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Body: Objective High altitude pulmonary hypertension (HAPH) is associated with sleep apnea. Since sleep apnea may impair cerebral tissue oxygenation (CTO) and cerebrovascular autoregulation we evaluated whether CTO and cerebrovascular response to changes in FiO2 are reduced in highlanders with HAPH. Methods 17 highlanders with HAPH (mean±SD pulmonary artery pressure, mPAP, 35±3 mmHg) and 17 age-matched controls (mPAP 24±4 mmHg, P<0.01 vs. HAPH) were studied in Aksay, Kyrgyzstan, at 3250m. CTO by near-infrared spectroscopy, finger pulse oximetry (SpO2), end-tidal PCO2, and ECG were recorded while subjects breathed room air and 100% oxygen for 20 min each, in random order. Results In highlanders with HAPH on FiO2 0.21, SpO2 was 89±2%, CTO 68±3%. In controls SpO2 was 91±3% (P=0.01 vs. HAPH), CTO 67±4% (P=0.34). In highlanders with HAPH, breathing FiO2 1.0 increased SpO2 by 9±3% (P<0.01), CTO by 5±2% (P<0.01) and reduced cerebral hemoglobin concentration (CHb), a surrogate of changes in cerebral blood volume by -1.4±1.6 relative units (P<0.01). In controls, breathing FiO2 1.0 increased SpO2 by 7±2% (P<0.01 vs. FiO2 0.21; P=0.01 vs. HAPH) and CTO by 3±1% (P<0.01 vs. FiO2 0.21; P=0.11 vs. HAPH), and decreased CHb by -1.4±2.1 ru (P=0.03 vs. FiO2 0.21; P=0.73 vs. HAPH. Younger age and higher CTO on FiO2 0.21 were significantly correlated with hyperoxia-induced changes in CHb but changes in blood pressure and end-tidal PCO2 were not. Conclusions In highlanders with HAPH arterial oxygen saturation is reduced but cerebral oxygenation and the cerebrovascular response to alterations in FiO2 are not impaired. Thus, compensatory mechanisms may prevent cerebral hypoxia in HAPH patients. Grants: OPO Foundation, Zurich Lung League.