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Title: Portioning out the contribution of dead space ventilation and low CO₂ set point to ventilatory inefficiency during exercise in chronic thromboembolic pulmonary hypertension

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Body: Rationale: The individual relevance of enlarged dead space ventilation as a fraction of tidal volume (VD/VT) and low CO₂ set-point in promoting exercise ventilatory inefficiency in chronic thromboembolic pulmonary hypertension (CTEPH) remains controversial. Objective: To determine the contributing role of VD/VT and PaCO₂ on the excessive ventilatory response to exercise in patients with CTEPH. Material and methods: 20 patients (47 ± 14 yrs, mean pulmonary artery pressure= 52 ± 10 mmHg) underwent an incremental exercise test and a steady-state test with arterial blood gases measurements. Results: Compared to normal standards, patients showed lower resting PaCO2 and peak V'O2 but greater Δ V'E/ Δ V'CO₂ slope (68 ± 16). Peak V'O₂ and Δ V'E/ Δ V'CO₂ were inversely correlated (r= -0.70; p<0.05). V'E/V'CO₂ ratio during the constant work rate test was also abnormally high (62 ± 15). VD/VT increased from 0.31±0.08 at rest to 0.39±0.11 during exercise; on the other hand, end-tidal partial pressure (PET) for CO₂ decreased despite a stable PaCO₂ (p>0.05). Consequently, P(a-ET)CO₂ became wider (4 ± 3 mmHg to 8 ± 5 mmHg), a finding related to greater V'E/V'CO₂ (r= 0.81; p<0.05). On a multiple regression analysis VD/VT (partial r = 0.89) and PaCO₂ (partial r = -0.37) explained up to 90% of V'E/V'CO₂ variability (125.9) VD/VT - 2.3 PaCO₂ + 77.1; p<0.001). Conclusion: Increased VD/VT is the main pathophysiological mechanism leading to an excessive ventilatory response to exercise in patients with CTEPH. Low CO₂ set-point responds for a relatively minor portion of this abnormal feature.