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Title: An increased respiratory drive accounts for the severity of dyspnea in systemic sclerosis

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Body: Introduction Dyspnea in progressive systemic sclerosis (SSc) may originate from pulmonary hypertension or interstitial lung disease. Respiratory drive is a major determinant of dyspnea. Evaluation of the respiratory drive measured by mouth occlusion pressures and CO2 rebreathing may better relate to the magnitude of dyspnea than the severity of gas transfer or lung volume impairment. Methods In 73 SSc patients referred to a targeted outpatient health care program PFT as well as mouth occlusion pressures after 0.1 sec (P0.1) were measured while breathing room air at resting ventilation and during rebreathing of a gas mixture containing 7% CO2 and 93% O2. An abnormal V'E/P0.1 is defined as < 8 L/min/cmH2O (Scott GC, Burki NK. Chest 1990;98:900-06). Dyspnoea scores were assessed by the USCD dyspnoea scale (Eakin EG et al. Chest 1998;113:619-24). Results Mean P0.1 in patients with normal normal V'E/P0.1 (n=45) was 1.1 ± 0.04 and in patients with abnormal V'E/P0.1 (n=28) 1.6 ± 0.08 cmH20, p <0.001. ΔP0.1/Δ PetCO2 differed significantly between these groups (0.75 versus 0.45 cmH20/mmHg, P<0.001), as well as FEV1, FVC and DLCO. No significant difference was present in ΔV'E/Δ PetCO2. V'E/P0.1 showed the highest significant correlation with the USCD dyspnoea scale (r= -0.76, p <0.001). In a binary logistic regression model the USCD dyspnea scale was the only predictor for an abnormal V'E/P0.1 (OR 4.68, CI: 3.17-6.91). Conclusion In SSc with an abnormal V'E/P0.1, an increased respiratory drive to CO2 is present and accounts for the severity of dyspnea.