Hypocapnia and increased ventilatory responsiveness in patients with idiopathic central sleep apnea

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We previously demonstrated that central apneas during sleep in patients with idiopathic central sleep apnea (ICSA) are triggered by abrupt hyperventilation. In addition, baseline PCO2 at the time of augmented breaths which triggered central apneas was lower than for augmented breaths which did not trigger apneas. These observations led us to hypothesize that patients with ICSA chronically hyperventilate maintaining their PCO2 close to the threshold for apnea during sleep owing to increased chemical respiratory drive. To test these hypotheses, we recorded transcutaneous PCO2 (PtcCO2) during overnight sleep studies on nine consecutive patients with ICSA and nine sex-, age-, and body-mass-index-matched control subjects. Daytime PaCO2 as well as rebreathing and single breath ventilatory responses to CO2 were also measured. Compared with the control subjects, the patients had significantly lower mean PtcCO2 during sleep (37.8 +/- 1.2 versus 42.7 +/- 10.9 mm Hg, p < 0.01) and lower PaCO2 while awake (35.1 +/- 1.3 versus 38.8 +/- 0.9 mm Hg, p < 0.05). Furthermore, patients with ICSA had significantly higher ventilatory responses to CO2 for both the rebreathing (3.14 +/- 0.34 versus 1.60 +/- 0.32 L/min/mm Hg, p < 0.005) and single breath methods (0.51 +/- 0.10 versus 0.25 +/- 0.04 L/min/mm Hg, p < 0.05). We conclude that: (1) patients with ICSA chronically hyperventilate awake and asleep and (2) chronic hyperventilation is probably related to augmented central and peripheral respiratory drive which predisposes to respiratory control system instability.