Carbon Dioxide Narcosis or Sleep Deprivation?

To the Editor:

Several assumptions were made in the Clinical Physiologist case presented by Lanks and colleagues (1) that are best avoided in the management of patients with Pickwickian syndrome. The patient was a 47-year-old man with decompensated obesity-related respiratory disease and 1 month of worsening symptoms. His baseline Pa_{CO_2} was unknown. Two liters of oxygen by nasal cannula were administered and later he was found to be somnolent. At that time, an arterial blood gas sample revealed a Pa_{CO_2} of 99 mm Hg. Three unwarranted assumptions followed this result: first, that the Pa_{CO_2} had increased since presentation; second, that the increased Pa_{CO_2} caused somnolence; and third, that the increased Pa_{CO_2} was caused by depression of the ventilatory drive from correction of hypoxemia. These assumptions are unfounded, for several reasons. High variability in both the incidence of hypercarbia after correction of hypoxemia *and* in the occurrence of mental status changes with hypercarbia has been recognized since at least 1950 (2), with some patients remaining completely lucid with Pa_{CO_2} levels as high as 175 mm Hg (3). In one recent study, application of 28% oxygen (approximately 2 L/min supplemental oxygen) to patients with obesity hypoventilation syndrome caused an average rise in CO_2 of only 2.5 mm Hg, and there is uncertainty as to whether these small increases resulted from reduced ventilation, increased dead space, or both (4). Thus, none of these three assumptions can be confidently defended by clinical experience or physiological reasoning. An alternative explanation is that the patient, exhausted from 1 month of symptoms and years of untreated sleep-disordered breathing, simply fell asleep when hypoxemia was relieved. This scenario of somnolence due to sheer exhaustion in patients with respiratory disease was described as early as 1992 (3) and is borne out by decades of subsequent clinical experience with increasingly obese patients. Thus, the patient's Pa_{CO_2}, if it indeed rose after presentation, may have risen not because of oxygen therapy but because of reduced ventilation during sleep. If any of these plausible and perhaps likely alternative narratives are correct, he could have been managed successfully with noninvasive ventilation and avoided mechanical ventilation with all its attendant risks. Because even extreme acute hypercarbia with Pa_{CO_2} as high as 250 mm Hg can be well tolerated physiologically (5), it may have been prudent to carefully observe the patient while considering other hypotheses and reducing supplemental oxygen, so long as he was otherwise stable and had a normal respiratory rate. A final assumption was that he could not be extubated because of a compensated respiratory acidosis with a Pa_{CO_2} of 82 mm Hg, and so acetazolamide was given. Clinical experience does not comport with this assumption, and such patients can frequently be extubated with compensated hypercapnia. Furthermore, in the DIABOLO (Effectiveness of Acetazolamide for Reversal of Metabolic Alkalosis in Mechanically Ventilated Chronic Obstructive Pulmonary Disease Patients) trial of ventilated patients with chronic obstructive pulmonary disease, acetazolamide administration did not significantly reduce duration of intubation (6). However, acetazolamide may foster extubation by improving the numbers on blood gas analysis if practitioners place undue emphasis on these numbers (7). Although we cannot know what would have happened to this patient in counterfactual circumstances, it behooves us and our obese patients to keep an open mind because, as the authors rightly emphasize, their ventilatory control may not comport with our expectations.

Author disclosures are available with the text of this letter at www.atsjournals.org.

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