

Defining obesity hypoventilation syndrome

The definition of obesity hypoventilation syndrome (OHS) proposed by Hart *et al*¹ raises two questions that we have tried to answer briefly in this letter.

First, previous definitions of OHS included obstructive sleep apnoea (OSA); Hart *et al* have excluded it. Why? OSA is present in most patients with OHS and is partly responsible for the hypercapnia.² We believe that it is useful to include it in the definition or classification of the condition in a manner similar to narcolepsy: OHS with OSA and OHS without OSA, as in narcolepsy with cataplexy and narcolepsy without cataplexy. Such classification has mechanistic and therapeutic implications.^{3,4}

Second, are high bicarbonate and base excess enough to establish the presence of chronic respiratory acidosis? For detecting OHS, a bicarbonate level cut-off of 27 mmol/L has a sensitivity close to 90%, but a specificity as low as 50%.^{5,6} Thus, a bicarbonate level less than 27 mmol/L excludes OHS, whereas a higher level only suggests it and must be followed by measurement of arterial blood gases.

Additionally, a high bicarbonate level indicates a metabolic alkalosis but does not differentiate a primary from a compensatory one. Because obese patients are frequently hyperaldosteronemic and are often on diuretics, corticosteroids, or both, they are prone to develop primary metabolic alkalosis that triggers mild compensatory hypercapnia.^{7,8} Thus, when OHS is suspected but the pH and the partial pressure of carbon dioxide are found to be slightly elevated, the physician should avoid attributing this result to hyperventilation from the pain of an arterial puncture and should consider interpreting it as a primary metabolic alkalosis.⁹

In summary, OSA and careful interpretation of arterial blood gas are critical for the definition of OHS. Obesity and elevated bicarbonate are merely triggers to look for it.

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