

Obesity hypoventilation syndrome: does the current definition need revisiting?

Obesity hypoventilation syndrome (OHS) has been conventionally (and to some extent arbitrarily) defined by the combination of obesity (body mass index (BMI) $>30 \text{ kg/m}^2$), daytime hypercapnia (arterial partial pressure of carbon dioxide (PaCO_2) $\geq 45 \text{ mm Hg}$ or 6 kPa) during wakefulness, and usually (but not always) the presence of 'sleep disordered breathing', such as obstructive sleep apnoea, rapid eye movement sleep hypoventilation or both.¹ The survival curve for untreated OHS is significantly reduced compared with the non-obese,² and so early identification and treatment for these patients is likely to be beneficial. Little is currently known about the true prevalence of OHS in ambulatory obese individuals, with estimates range from 0.3–0.4% of the general population,³ to around 30% of hospitalised patients with a BMI $>35 \text{ kg/m}^2$.² The combined medical costs associated with treatment of obesity associated diseases are estimated to increase by \$48–66 billion/year in the USA and by £1.9–2 billion/year in the UK by 2030,⁴ making OHS an increasingly common and expensive problem. The American Medical Association recently announced that obesity is now considered a 'disease', a step which may improve patient access to

obesity treatments and increase funding into obesity research.

The current definition, based as it is on a single one-off measurement of PaCO_2 , seems to us to be too restrictive; PaCO_2 is not constant and, rather like blood pressure where the white coat effect is well recognised, is subject to a variety of influences. For example, patient anxiety over arterial sampling is likely to 'falsely' lower the PaCO_2 , and many obese subjects hypoventilate overnight, but are able to return their PaCO_2 to within normal levels a short time following awakening.⁵ Calculated arterial standard bicarbonate (HCO_3^-) level from a conventional blood gas machine, in the absence of another influence on metabolic acid-base status, is a longer term guide to 24-h ventilation and, in some ways, can be viewed as the 'HbA1c' of CO_2 levels because it represents the renal retention of HCO_3^- that occurs in response to hypercapnia. Our combined experience in this area is that there are many patients with significant nocturnal hypoventilation who, during the day, have normal-to-high PaCO_2 levels, but have alkalotic (>7.40) pH values, and thus raised standard HCO_3^- (or base excess) levels. Serum venous bicarbonate concentration, which is increased by a currently raised PaCO_2 level and any renal compensation, has also previously been described to be a useful screening tool in identifying patients with OHS in a group of obese patients with sleep disordered breathing⁶ and might be used as a simpler alternative for screening.

In our view, the use of PaCO_2 alone in the definition of OHS may miss early disease, if used in isolation. Clearly delineating the OHS population is a key issue, for appropriate care, and for conducting unbiased clinical studies and determination of phenotypes associated with poor prognosis within the obese population. We propose that the definition of OHS should be based on obesity, plus a $\text{PaCO}_2 \geq 45 \text{ mm Hg}$ (6 kPa) OR an arterial base excess $>3 \text{ mmol/L}$ OR a standard $\text{HCO}_3^- >27 \text{ mmol/L}$ (in the absence of another cause for a metabolic alkalosis). Clearly, the BMI, the exact levels of PaCO_2 , and now the HCO_3^- , used in the definition of OHS are relatively arbitrary thresholds, and may benefit from refinement; but the addition of a raised arterial HCO_3^- criterion seems to add value to the identification of this increasing problem.

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