What is the best definition of the term “hyperventilation”?

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TO THE EDITOR: In the context of a discussion about a patient with chronic obstructive pulmonary disease (COPD), a second-year medical student recently asked: What is the definition of hyperventilation? My initial response was that, when the CO₂ production rate is constant, an individual who is hypocapnic (i.e., has a lower than normal CO₂ tension in arterial blood) must have hyperventilated. This left me with the question as to whether hypocapnia is an essential feature of hyperventilation. In pulmonary physiology, the term “ventilation” is used to refer to the volume of gas flowing into the respiratory system per unit time. (5) Comroe et al. (1) defined ventilation as a cyclic process of inspiration and expiration in which fresh air enters the alveoli and an approximately equal volume of gas leaves the alveoli. Alveolar ventilation is defined as respiratory minute volume (or minute ventilation) minus ventilation of the anatomic dead space (5).

However, historically, hypocapnia (a very likely consequence of an increase in alveolar ventilation) has been included as an essential feature of hyperventilation and alveolar hyperventilation (1, 3, 5). This is confusing because it does not logically follow the manner in which the term ventilation has been defined. Comroe et al. (1) defined hyperventilation as an increase in ventilation in excess of that required to maintain a normal PO₂ and PCO₂ in arterial blood (PAO₂ and PACO₂, respectively). Plum (5) defined hyperventilation as a greater volume of ventilation than required by the metabolic demands of the body and suggested “one cannot call overbreathing hyperventilation so long as a patient breathing room air has either an arterial PCO₂ above 40 mmHg or an arterial PO₂ below 70 mmHg.” Whipp (5) defined hyperventilation as “an increase in ventilation out of proportion to any increase in metabolic VCO₂, therefore resulting in a low arterial PCO₂. Hypocapnia is therefore a criterion by which to determine whether a subject is hyperventilating or not.” [Here, VCO₂ is expired CO₂.] According to these definitions by Comroe et al. (1), Plum (3), and Whipp (5), a patient with COPD who has a PAO₂ of 60 mmHg, PACO₂ of 46 mmHg, and whose respiratory minute volume is twice normal is not hyperventilating, and the increase in respiratory minute volume during exercise (hyperpnea) in a normal subject does not constitute hyperventilation if PACO₂ remains within the normal range. These definitions of hyperventilation are grounded in the notion that the functional significance of the process of ventilation is in part to maintain a PCO₂ of 40 mmHg (in humans). In contrast, when hypocapnia is held as an essential feature of voluntary hyperventilation, it is grounded in a different teleological notion, which is that the functional significance of ventilation is to maintain CO₂ elimination at the same rate as CO₂ production without regard to PACO₂ and pH.

Ingram et al. (2) studied the effect of voluntary “hyperventilation” (a term used by authors of this paper) on PACO₂ in patients with COPD and found that a small subset of patients had a PACO₂ > 40 mmHg despite a substantial increase in minute ventilation (see Table 2 in Ref. 2). The observed values of minute ventilation during voluntary hyperventilation were higher compared with baseline minute ventilation in eucapnic young adults. The high PACO₂ was associated with no change or a further increase in the ratio of Bohr’s dead space and tidal volume. The observed PACO₂ values in hypocapnic patients after voluntary hyperventilation may or may not have been clinically significant; however, it is clear that when there is a significant mismatch between alveolar ventilation and pulmonary capillary perfusion, not only is O₂ uptake by lungs impaired but CO₂ elimination becomes less effective as well, despite a high respiratory minute volume and a presumed increase in alveolar ventilation.

To summarize, my view is it is preferable that definitions for hyperventilation and hypoventilation follow the definition of ventilation in pulmonary physiology rather than be grounded in one of two implied teleological notions. Second, considering a broad range of physiological states and pathological states, including patients with COPD and substantial ventilation-perfusion mismatch, it is clear that an increase in respiratory minute volume does not necessarily result in a decrease in PACO₂. Third, as noted by West (4), defining hyperventilation as an increase in effective alveolar ventilation (i.e., alveolar ventilation that contributes to gas exchange) is not constructive since the latter is not measurable. Therefore, I suggest that the term hyperventilation be limited to refer to an increase in respiratory minute volume or total minute ventilation (i.e., the product of tidal volume and respiratory frequency) compared with baseline minute ventilation in age-matched healthy adults, regardless of changes in PACO₂.

The more specific term alveolar hyperventilation is also best defined as an increase in alveolar ventilation in reference to resting alveolar ventilation in age-matched healthy adults, regardless of changes in PACO₂. Although measurements of pulmonary ventilation and anatomic dead space and therefore the calculation of alveolar ventilation are not feasible in routine clinical practice, practically, it is important to remember that when a patient with chronic bronchitis or emphysema whose breathing is labored and tachypneic also happens to be hypocapnic, it is suggestive of gross ventilation-perfusion mismatch. As previously suggested by West (4), in any given patient, determining whether hypocapnia is due to an absolute reduction in alveolar ventilation, absolute reduction in pulmonary blood flow, or gross ventilation-perfusion inequality is critical for appropriate management.

DISCLOSURES

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REFERENCES


