The Hazards of Measuring the Drive to Breathe

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In this issue of *Respiration*, Moura et al. [1] report on the effects of consecutive nights of nasal continuous positive air pressure on the occlusion pressure and ventilatory response to CO₂ in 12 patients with obstructive sleep apnea in an attempt to determine changes in ventilation drive, i.e. chemoreceptor responses.

Theoretically, in hypercapnic patients, nasal continuous positive air pressure might improve sensitivity to CO₂ by reducing resting levels of PCO₂, which in turn would lead to a fall in bicarbonate levels. In normocapnic patients, such as those studied by Moura et al. [1], relief of sleepiness might improve CO₂ responses [2]. The results of Moura et al. [1] were inconclusive; the occlusion pressure increase with CO₂ was greater but the ventilatory response was not.

Moura et al. [1] state that the occlusion pressure response (the pressure generated at the mouth during inspiration in the first 100 ms after airway occlusion) is a more sensitive measure of drive than ventilation. However, there is little or no evidence to support this. The occlusion pressure response was conceived as a way to measure chemosensitivity in the face of impaired lung mechanics, since poor mechanics could depress the response to ventilation even when the sensitivity of the chemoreceptors was normal [3–5]. The occlusion pressure was assumed to reflect the ‘net respiratory pressure’ generated by the respiratory muscles under isometric conditions, and this is less affected by increased airway resistance than ventilation. However, in the study of Moura et

al. [1], lung function is reported to be normal. Occlusion pressure does depend on the ability of the respiratory muscles to generate force and this in turn depends on their length. A decrease in functional residual capacity with the treatment of sleep apnea could give rise to an increase in occlusion pressure, but there is no reason why this should have occurred, and in fact, resting occlusion pressure was unchanged.

The real problem in measuring chemosensitivity, I believe, is the difficulty in measuring the drive to breathe by any technique and particularly by the technique used in this study. Great care is needed to obtain reliable measurements.

In this study, 8% CO₂ in 42% O₂ was breathed and measurements were made before and between the 4th and 5th minute of CO₂ breathing. To be valid, both before and after measurements have to be made in the steady state. There is no evidence that this is true for either one [6].

Initial measurements were made with the subjects apparently breathing room air and not with 42% O₂. The enriched O₂ mixture breathed might have affected breathing, albeit slightly. More importantly, it has been known for some time that a steady state is not achieved during CO₂ breathing for well over 20 min, particularly with the high concentration of 8% used in this study. Also, the rate of equilibration will vary not just with the dynamics of the ventilatory response but will also depend on the magnitude and dynamics of the systemic circulatory and cerebral blood flow response. More accurate and reproducible

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measurements probably could have been achieved using a rebreathing technique.

However, even with the rebreathing method, care must be taken to perform measurements in the same state, i.e. at the same time of the day, at the same time after meals and making sure to the greatest possible extent that environmental and psychological disturbances are minimal.

Despite the inconclusiveness of the results from the above-mentioned study, data on the drive to breathe as sleep apnea is treated can be extremely valuable in sorting out differences in the response to treatment and in selecting the appropriate treatment for different individuals.

References

- 1 Moura SMT, Bittencourt LR, Bagnato MC, Lucas SR, Tufik S, Nery LE: Acute effect of nasal continuous positive air pressure on the ventilatory control of patients with obstructive sleep apnea. *Respiration* 2001;68:243–249.
- 2 Stradling JR: Sleep-related breathing disorders. 1. Obstructive sleep apnoea: Definitions, epidemiology, and natural history. *Thorax* 1995; 50:683–689.
- 3 Altose MD, Kelsen SG, Stanley NN, Levinson RS, Cherniack NS, Fishman AP: Effects of hypercapnia on mouth pressure during airway occlusion in conscious man. *J Appl Physiol* 1976;40:338–344.
- 4 Whitelaw WA, Derenne JP, Milic-Emili J: Occlusion pressure as a measure of respiratory center output in conscious man. *Respir Physiol* 1975;23:181–199.
- 5 Lederer DH, Altose MD, Kelsen SC, Cherniack NS: Comparison of occlusion pressure and ventilation responses. *Thorax* 1977;32:212–220.
- 6 Lambertsen CJ, Owen SG, Wender H, Stroud MW, Lurie AA, Lochner W, Clark GF: Respiratory and circulatory control during exercise at 1.4- and 2.0-atmospheres inspired PO_2 . *J Appl Physiol* 1959;14:966–982.