Alveolar partial pressures of carbon dioxide and oxygen measured by a helium washout technique

The estimation of arterial carbon dioxide pressure (Paco2) by Professor J Jordanoglu and colleagues (Journal of Physiology 1990;45:530-4) assumes the equivalence of Bohr-Enghoff deadspace1 with the alveolar deadspace by multiple breath washout (the "ventilatory deadspace") of Cumming and Guyatt2. In a letter to Clinical Science following their previous paper1 I pointed out the fallacy of this assumption.3

I note that in their Thorax paper the authors mention pulmonary embolism as a cause of discrepancy and took steps to exclude this in their patients. Any kind of ventilation-perfusion (V/Q) mismatch, however unequal solely to ventilation-volume (V/V) mismatch, will introduce such a discrepancy, and their patients with chronic bronchitis and asthma must be presumed liable to such V/Q non-uniformity. This doubtless accounts for much of the rather wide scatter in their figure 2. The 95% confidence interval about regression line is about ±1.5 kPa (11.5 mm Hg). Another difficulty is that the ventilatory deadspace for helium increases, during washout, with breath number if V/V mismatching is present. The choice of first breath deadspace by Professor Jordanoglu and colleagues is quite arbitrary.

This criticism is not merely about inaccuracy. The rebreathing method for oxygenated mixed venous carbon dioxide tension (Pvco2)3 is not accurate, as all methods are. But the target is the intended one. The authors shoot at a physiologically different target on the pretext that it often coincides with the one they wish to hit.

There are other statements in this paper with which I do not agree. Right to left shunts, unless enormous, will not affect the relation between the two deadspaces at rest. Membrane diffusion defects will, in theory, but in practice the effect would never be measurable. The ventilatory deadspaces for helium and SF6 are not equal; they differ systematically and very significantly,4 though this fact has no bearing on the question of whether helium and carbon dioxide deadspaces are equivalent.

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We also showed that the two compartment phenomenon, when there is doubt, can easily be recognised with a partial volume lung function manoeuvre.5


AUTHOR'S REPLY The calculation of the alveolar carbon dioxide and oxygen concentration during quiet breathing presumes the measurement of the physiological deadspace: tidal volume ratio by an inert gas washout method (helium (Vhel)/VT) and that of the mixed expired carbon dioxide and oxygen concentration (FEnvCO2, FEnvO2). The helium washout method, as developed in our laboratory, was applied in healthy subjects and in patients. In these subjects the classical carbon dioxide method for measuring the physiological deadspace: tidal volume ratio (Vhel/VT) was also applied. The comparison between these two indices showed that Vhel/VT was well correlated with Vhel/VT, the assumption was made about the relation between these two ratios.

Theoretically, Vhel/VT and Venv/VT are equal to each other when the alveolar carbon dioxide concentration is used in the Bohr equation, as explained in the paper. So by transformation of this equation and using the Vhel/VT ratio, measured by our technique, we calculate the alveolar carbon dioxide concentration or tension (wPaco2). It is also mentioned here that no assumption was made for the calculation of wPaco2.

wPaco2 was compared with Paco2, and it was found that there was (wVco2 correlation, as shown in the paper. The deviation of wPaco2 from Paco2 may reflect the real difference between these two measures in the patients studied and/or to some extent an error in the measurement of these two terms. Accordingly, we do not postulate that wPaco2 is equal to Paco2, in general but we are entitled to say that wPaco2 is a good estimate of Paco2, for practical purposes, as the mean of the differences (wPaco2-Paco2) is 0.01 kPa, the standard deviation of the differences is 0.7 kPa, and the limits of agreement are ±1.4 kPa.

The first breath deadspace is not referred to anywhere in the paper. The helium washout deadspace as measured by our technique may be equal to the ventilatory deadspace of Cumming and Guyatt, as Dr Harris mentions in his letter. These two tests differ from each other, however, in theory and method.

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The biphasic spirogram: a clue to unilateral narrowing of a mainstem bronchus

Dr A D Gascoigne and his colleagues (August 1990;45:637-8) confirm our findings of the two compartment phenomenon, caused by unilateral airflow obstruction and manifested as end inspiratory (and end expiratory) slowing of the maximum inspiratory flow-volume curve. The phenomenon was first described by Williams et al6 in a patient with severe stenosis of the left main bronchus. We described two patients; one with almost complete obstruction of the left main bronchus caused by bronchial carcinoma and the other with unilateral lung emphysema (Macleod's syndrome), as suggested by Dr Gascoigne and colleagues.

We greatly enjoyed the article by Dr A D Gascoigne and others (August 1990;45:637-8) on the biphasic spirogram, which the authors thought had not been described previously. They will find an earlier example in a book edited by Tim Clark.7

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AUTHORS' REPLY We thank Drs Braat and Roos and Professor Denison for drawing our attention to further examples of maximum flow-volume curves in individuals with stenosis of a mainstem bronchus; we acknowledged in our report that such appearances had been described previously. In most lung function laboratories, however, flow-volume curves are not obtained routinely from all patients and the main aim of our paper was to draw attention to the shape of the forced expiratory spirogram—that is, the volume-time curve in unilateral bronchial narrowing. Although this shape can be predicted on theoretical grounds, we are not aware that examples have been published previously and we hope that our report will alert the observer to the possible implication of such a pattern. We speculated that a similar appearance might be seen in unilateral emphysema and it is helpful to note that the volume-time curves from one such patient support this contention.

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Jet and ultrasound nebuliser output: use of a new method for direct measurement of aerosol output

We thank Dr JH Dennis and colleagues (October 1990;45:728-32) for highlighting the considerable limitations in using the weight loss of a nebuliser as an index of the amount of solute (for example a drug) released in an aerosol. We agree that it is necessary to measure the amount of aerosol which is leaving the nebuliser directly and have used such a technique where the sampling filters were weighed after drying to determine the weight of solute nebulised.8