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

The estimation of arterial carbon dioxide pressure (P_{aCO_2}) by Professor J Jordanoglou and colleagues (July 1990;45:520-4) assumes the equivalence of Bohr-Enghoff deadspace¹ with the helium deadspace by multiple breath washout (the "ventilatory deadspace" of Cumming and Guyatt²). In a letter to *Clinical Science* following their previous paper³ I pointed out the fallacy of this assumption.⁴

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 ($\dot{V}/Q$) mismatch, however unless due solely to ventilation-volume ($\dot{V}/V$) mismatch, will introduce such a discrepancy, and their patients with chronic bronchitis and asthma must be presumed liable to such $\dot{V}/Q$ non-uniformity. This doubtless accounts for much of the rather wide scatter in their figure 2. The 95% confidence interval about regression is about ± 1.54 kPa (11.5 mm Hg). Another difficulty is that the ventilatory deadspace for helium increases, during washout, with breath number if $\dot{V}/V$ mismatching is present. The choice of first breath deadspace by Professor Jordanoglou and colleagues is quite arbitrary.

This criticism is not merely about inaccuracy. The rebreathing method for oxygenated mixed venous carbon dioxide tension ($P_{\bar{V}CO_2}$)⁵ is inaccurate, 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 SF_6 are not equal; they differ systematically and very significantly,^{6,7} though this fact has no bearing on the question of whether helium and carbon dioxide deadspaces are equivalent.

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- 1 Enghoff H. Volumen inefficax. Bemerkungen zur Frage des schädlichen Raumes. *Uppsala Läkarforen Forh* 1938;44:191-218.
- 2 Cumming G, Guyatt AR. Alveolar gas mixing efficiency in the human lung. *Clin Sci* 1982; 62:541-7.
- 3 Jordanoglou J, Tatsis G, Bissiolli Z. Calculation of the V_D/V_T ratio by the helium washout technique. *Clin Sci* 1986;70:656-9.
- 4 Harris EA. Measurement of dead-space volume. *Clin Sci* 1987;73:239.
- 5 Campbell EJM, Howell JBL. The determination of mixed venous and arterial CO_2 tension by rebreathing techniques. In: Woolmer RF, ed. *A symposium on pH and blood gas measurement*. London: Churchill, 1959: 101-18.
- 6 Harris EA, Buchanan PR, Whitlock RML. Human alveolar gas-mixing efficiency for gases of differing diffusivity in health and airflow limitation. *Clin Sci* 1987;73:351-9.
- 7 Fuller GC, Harris EA, Withy SJ. Gas-mixing efficiency in a mathematical model of the lung. *Clin Phys Physiol Meas* 1990;11:149-58.

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) (wV_D/V_T) and of the mixed expired carbon dioxide and oxygen concentration (F_{ECO_2} , F_{EO_2}). 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 (V_D/V_T) was also applied. The comparison between these two indices showed that wV_D/V_T was well correlated with V_D/V_T . No assumption was made about the relation between these two ratios.

Theoretically, wV_D/V_T and V_D/V_T 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 wV_D/V_T ratio, measured by our technique, we calculated the alveolar carbon dioxide concentration or tension (wP_{ACO_2}). It is also mentioned here that no assumption was made for the calculation of wP_{ACO_2} .

wP_{ACO_2} was compared with P_{ACO_2} and it was found that there was a good correlation, as shown in the paper. The deviation of wP_{ACO_2} from P_{ACO_2} 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 wP_{ACO_2} is equal to P_{ACO_2} in general but we are entitled to say that wP_{ACO_2} is a good estimate of P_{ACO_2} for practical purposes, as the mean of the differences ($wP_{ACO_2} - P_{ACO_2}$) 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 al*¹ 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 also showed that the two compartment phenomenon, when there is doubt, can easily be recognised with a partial volume lung function manoeuvre.²

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- 1 Williams SJ, Pierce RJ, Davies NJH, Denison DM. Methods of studying lobar and segmental function of the lung in man. *Br J Dis Chest* 1979;73:97-112.
- 2 Roos CM, Braat MCP. End-inspiratory flow reduction in the forced flow-volume curve as a sign of unilateral bronchial disease. *Neth J Med* 1985;28:378-82.

I 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.¹

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- 1 Denison DM. Physiology. In: Clark TJH, ed. *Clinical investigation of respiratory disease*. London: Chapman and Hall, 1981:54.

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 flow-volume curves from one such patient support this contention.

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Jet and ultrasonic 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.¹

The technique used by us becomes inaccurate in circumstances where the mass output of the nebuliser is very small. Therefore—provided that the output of tracer is representative of the output of the solute under consideration—the tracer technique described by Dr Dennis and colleagues may be preferable when one is dealing with small mass outputs.

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- 1 Hamilton RD, Winning AJ, Guz A. Blockade of "alveolar" and airway reflexes by local anaesthetic aerosol in dogs. *Respir Physiol* 1987;67:159–70.

BOOK NOTICES

Asthma as an Inflammatory Disease. Paul M O'Byrne. (Pp 336; \$119.50.) New York: Marcel Dekker, 1990. ISBN 0-8247-8220-8.

This book is one of a series published by Marcel Dekker on allergic disease and treatment. The title is somewhat misleading as this volume is really an overview of the current concepts in asthma, with comparatively little emphasis on the inflammatory basis of the disease. The opening chapters deal with the pathology, epidemiology, and genetics of asthma and serve well as an introduction. The following six chapters include strong sections on airway hyperresponsiveness, atopy, and asthma and cough as a manifestation of asthma. The latter contribution I found particularly interesting as it is a subject often left out of books of this sort. The last four chapters are concerned with treatment and are probably the most interesting. They start with an excellent, comprehensive review of the pharmacology of antiasthma medication and end with a thought provoking contribution on variations in prescribing of prophylactic anti-inflammatory treatment throughout the world. Overall, I was pleasantly surprised by this book. The topics covered are important and well reviewed by an international team of contributors, resulting in a well balanced approach with a good mix of European and North American viewpoints. The chapters are well referenced and the references are up to date and comprehensive. My only reservation is the price: even at a favourable exchange rate, at \$119.50 this slim volume is more expensive than several equally up to date, more comprehensive reviews. Medical and paramedical newcomers to the field of asthma will, however, find much of interest in this book and I hope it finds its way on to a few library shelves.—IP

The Lung in Rheumatic Disease. GW Cannon, GA Zimmerman. (Pp 545; \$150.) New York: Dekker, 1990. ISBN 0-8247-8211-9.

This is the latest volume (No 45) in a long series of generally well received volumes entitled "Lung Biology in Health and Disease" under the executive editorship of the director of the National Heart, Lung, and Blood Institute in the United States. It is not before time that the involvement of the lung in the rheumatic disorders receives appropriate attention and the subject is generally well reviewed in this text. The authors are drawn almost entirely from the North American subcontinent with no European contribution and almost half of the contributors are rheumatologists. Seventeen chapters are divided into four sections; pathogenesis of lung and pleural lesions; methods of assessment of lung disease—physiology, imaging, biopsy and bronchoalveolar lavage, pathology; a section of chapters dealing with the individual entities; a pot pourri of chapters on sarcoidosis, cystic fibrosis, and hypertrophic pulmonary osteoarthropathy, disorders in which arthropathy may be a feature. It is pleasing to see good chapters on infection and drug induced diseases, complications that may be confused with the underlying lung problem; and other chapters worthy of mention include those on the pathogenesis of interstitial lung disease and pathology. In general, the book contains detailed, well referenced, and clearly illustrated chapters but it is particularly disappointing that there is no mention of high resolution computer tomography, a technique now well established in the assessment and early diagnosis of interstitial lung disease. Some of the contributions by those who are not lung specialists reflect a lack of the sort of detailed, in depth perception of the pulmonary problem that is more evident in the chapters by the lung experts. Aimed at the clinician and the basic scientist, it is likely to achieve only 50% success. The clinician will find it a good source of review and reference but the basic scientist will be disappointed by the depth of discussion on some of the basic mechanisms, particularly immunology and molecular biology. At \$150 this will not be a good buy for most individuals but would be a good acquisition for the library or the rheumatic lung disease aficionado.—RMB

Ventilation/Blood Flow and Gas Exchange. 5th ed. John B West. (Pp 120; £9.95.) Baltimore: Blackwell, 1990. ISBN 0-632-02855-6.

It was a pleasure to review this new edition of Professor West's classic monograph on the relation between blood flow and ventilation. He describes the physiological concepts concerned in gas transport from air to tissue with illustrations from original experiments, many of which he performed himself. The book begins by describing normal oxygen transport and the adverse effects on gas exchange of hypoventilation, impaired perfusion, shunt and ventilation-perfusion inequality. He then covers in detail the physiology of ventilation and perfusion

interrelationships in the normal lung, emphasising the effects of inequality of distribution of both ventilation and blood flow on regional gas exchange. For the faint hearted this section may be rather daunting, but it provides the basis for understanding abnormalities of gas exchange in the diseased lung. The pathophysiology of hypoxia and carbon dioxide retention are very clearly described, again with helpful experimental illustrations. Ventilation-perfusion inequality in various pulmonary and cardiac conditions is then illustrated, use being made of the multiple inert gas elimination technique. This approach emphasises the importance of both alveolar ventilation and cardiac output to the maintenance of normal gas exchange and transport. This short book is not easy reading. The subject matter is complex, but with perseverance comes a thorough understanding of the physiology of normal gas exchange and abnormalities caused by disease. The text is clear and logical. Mathematical formulae describing ventilation and blood flow are avoided in the text, but usefully included in the appendix. The figures and illustrations are helpful for understanding the text and only occasionally are too complex. This monograph is already a medical classic and the new edition well worth the investment. It can be recommended to all physicians caring for patients with abnormalities of gas exchange, to refresh understanding of this complex but very important aspect of human physiology.—SE

NOTICES

Symposium on breathlessness

The 1991 Campbell symposium, on breathlessness, will be held on 16–19 May 1991 in Hamilton, Ontario, to mark the retirement of Dr E J Moran Campbell and the 25th anniversary of the Manchester breathlessness symposium, which he chaired with Dr J B L Howell. Former students and colleagues of Dr Campbell are encouraged to attend. The number of participants will be limited by the accommodation available. Details from the conference coordinator IM10, McMaster University, 1200 Main Street West, Hamilton, Ontario, Canada L8N 3Z5 (fax (416) 521 0048).

Course on lung pathology

Lung pathology is the subject of a comprehensive course of lectures, hands on microscopy sessions, and a slide seminar to be held at the Brompton Hospital, London, on 10–12 June 1991. The programme will include J Wigglesworth on perinatal disease, M Dunnill on defence mechanisms and fibrosis, A Gibbs on pneumoconiosis, C Wagenvoort on hypertension, and various internal speakers on airway disease, infections, interstitial disease, angitis, and tumours. The fee is £150 (or US \$290). Applications should be sent to Professor B Corrin, Department of Histopathology, Brompton Hospital, London SW3 6NP.