Correspondence

Non-invasive measurement of cardiac output by a single breath constant expiratory technique

Sir,—We were interested in the report by Dr U Elkayam and colleagues (February 1984;**39**:107–13) on estimation of cardiac output using a single breath technique but wish to draw attention to certain problems which are not fully considered in the paper.

Firstly, as the authors briefly mention, their technique bears some resemblance to that developed by Denison and colleagues, but we were surprised at the very limited comparison of these methods in the discussion and at the lack of reference to other publications on the theoretical and practical aspects of this type of measurement.[1][2] The technique applied by Dr Elkayam and his colleagues appears to depend on completely even distribution of ventilation and assumes a perfectly horizontal plateau of expired helium concentration, whereas the method of Denison *et al* attempts to take account of the almost inevitable maldistribution of ventilation in such patients by comparing the inert and soluble gas traces to allow calculation of an "effective" pulmonary blood flow. We have shown[3] in a small group of patients with no pulmonary disease that this latter method gives values close to those obtained at cardiac catheterisation by the direct Fick technique. Dr Elkayam and his colleagues admit that in three patients with airflow obstruction the results were not reproducible; but, since spirometric data were available in only 12 of their 23 patients the influence of appreciable airway narrowing may have been considerably underestimated. We presume that the majority of their patients had very uneven ventilation, since the calculated venous admixture exceeded 10% of the cardiac output in 10 of the 14 for whom the data are given. In our experience the record of expired helium against time in such patients is unlikely to be close to the horizontal. The effect should be to underestimate cardiac output and it is therefore surprising that no such tendency is evident in the data presented.

Secondly, the authors appear to ignore any influence of inspiratory flow rate; this may be important since there is evidence that a more rapid inspiratory flow produces more even regional distribution of inspired gas.[4][5]

Thirdly, in the equation for derivation of pulmonary capillary blood flow the Bunsen solubility coefficient for acetylene in blood should presumably appear in the denominator.

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[1] Denison DM, Davies NJH, Brown DJ. A theoretical comparison of single breath and rebreathing methods of studying soluble gas exchange in the lung. In: Cumming G, Bonsignore G, eds. *Pulmonary circulation in health and disease.* New York: Plenum Press, 1980:139–66.
[2] Denison DM, Waller JF. Interpreting the results of regional

single breath studies from the patients point of view. *Bull Eur Physiopath Respir* 1982;**18**:339–51.
[3] Corris PA, Kendrick AH, Hawkins T, Gibson GJ. Measurement of unilateral pulmonary blood flow in patients with carcinoma of the bronchus. *Clin Sci* 1984;**66**:54P (abstract).
[4] Robertson PC, Anthonisen NR, Ross D. Effect of inspiratory flow rate on regional distribution of inspired gas. *J Appl Physiol* 1969;**26**:438–44.
[5] Connolly T, Bake L, Wood L, Milic Emili J. Regional distribution of a ¹³³Xe labelled gas volume inspired at constant flow rates. *Scand J Respir Dis* 1975;**56**:150–9.

Sir,—We read with interest the paper by Dr U Elkayam and his colleagues on the non-invasive measurement of cardiac output by a single breath constant expiratory technique (February 1984;**39**:107–13). We welcome their valuable contribution to work in this field by their direct comparison of their method to thermodilutional measurements of cardiac output. We have been using a similar approach to studying both whole and regional lung function in this laboratory for the past six years, using argon and freon 22. While we would agree with Dr Elkayam that such techniques are for application to those with near normal pulmonary function, we have demonstrated that this does not preclude its application to patients with more than mild airways obstruction. Indeed, we have preferred to make a virtue of the effects that regional maldistribution of function has on such traces and measures. Consequently, we apply the term effective pulmonary blood flow ($\dot{Q}p$) to the value derived by our techniques, thus recognising that it measures only that part of total cardiac output which comes into contact with ventilated lung. As such, reduced $\dot{Q}p$ provides a valuable index of the severity of cardiopulmonary disease.

We should also like to point out that, contrary to the statement made in their discussion of our work,[1] our technique involved the measurement of pulmonary capillary blood, over a similar volume change to that used by Dr Elkayam and his coworkers. To that extent their work is not as original as their paper suggests.

Finally, our experience indicates that reproducible values for effective pulmonary blood flow may best be obtained by repeating single breath tests at 3·5 minute intervals and it is unnecessary and undesirable to wait for complete washout of the soluble gas between estimations.

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[1] Denison DM, Davies NJH, Meyer M, Pierce RJ, Scheid P. Single exhalation method for study of lobar and segmental lung function by mass spectrometry in man. *Respir Physiol* 1980;**42**:87–99.

⁎⁎ These letters were sent to the authors, who reply below.

Sir,—In response to the thoughtful letter of Drs Corris and Gibson we would like to offer the following response.

Firstly, the differences between our technique and that

of Denison and colleagues are not major (see below). Our technique is based on a general evaluation of gas behaviour during the several phases of respiration.[1] Hence all equations are absolute closed solutions for each phase of breathing. Like the approach of Denison and colleagues, we correct for uneven volume to function by dividing instantaneous absorbable gas concentrations (carbon monoxide, acetylene) by simultaneous inert gas (helium) concentration. Mild to moderate maldistribution does not affect the accuracy of the technique. In the nine patients in whom single breath nitrogen values were measured the mean (SD) values were 4·11% (2·84%)/1 (range 0·5–8·4%) in seven (of 20) who had reproduceable values and 6·8% and 16·4%/1 in two (of three) who did not have reproduceable values.

Secondly, while it is quite true that high inspiratory flow rates lead to more even distribution of boluses of gas inspired at several lung volumes,[2 3] the effects of this phenomenon on gas absorption test results may not be easy to predict for near vital capacity breaths, particularly in the presence of "normal" regional non-uniformity of blood flow.[4] In fact, the mean (SD) inspiratory flow rate of our patients was 0·60 (0·22) 1/s. This flow rate is moderately low when compared with the data in the reference.[2 3]

Thirdly, we apologise for the typographical error in the equation on p 108 of our article. The first α_t should read α_b (Bunsen coefficient for blood).

Finally, the article by Dr Corris and his associates, unfortunately, has not, as yet, reached California. We look forward to reading this work with great interest.

We wish to thank Professor Denison and Dr Waller for their comments. They have highlighted several interesting and important points which we would like to try to address. After carefully reading the paper of Denison and colleagues[5] we agree that their technique has a large number of similarities to our technique, including the choice of gases, respiratory manoeuvres, and, perhaps, the theoretical approach. Equation 3 in their paper is:

$$\frac{-dP_A}{dt}\,\beta g\;V_A(t) = G\,P_A,$$

where G is pulmonary blood flow, βg a comparative term, and $V_A(t)$ alveolar volume at any time. Later in the paper they state that initial gas volume must be corrected for an equivalent lung tissue volume which has reached equilibration with end inspiratory gas. In our theoretical paper[6] we are more explicit (equation 9, rearranged to be similar to that of Denison and colleagues):

$$\frac{dF_A}{dt}\,(V_A + {}_tV_t) = \alpha_{\,b}\dot{Q}_cF_A;$$

the symbols are defined in both the earlier theoretical paper[1] and our more recent validation paper.[6] One apparent difference between the approaches is that the term for gas equivalent volume of tissue, α_tV_t, is utilised at all alveolar volumes in our technique and, perhaps, only at end inspiratory gas volume in their technique. For further comparison between techniques, it would be useful to know what solution of the differential equation they utilise for actual calculation of $\dot{Q}c$. Nevertheless, Denison and Waller are quite correct in their assertion of similarity.

Our experiences with these similar techniques have, however, not been identical. We have noted progressive systematic reduction of Qc values when repeat estimations are performed after less than a 15 minute wait; we allow an additional five minute wait to be sure that the rate of absorption of acetylene will not be reduced by retained gas. Finally, we have not felt confident in applying this technique to patients with severe airways obstruction and markedly abnormal distribution of volume, ventilation, and perfusion. We have frequently noted curvilinear relationships in these patients and feel that validation by direct measurement is indicated before the technique can be widely applied in such patients.

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[1] Martonen TB, Wilson AF. Theoretical basis of single breath gas absorption tests. *J Math Biol* 1982;**14**:203–20.
[2] Robertson PC, Anthonisen NR, Ross D. Effect of inspiratory flow rate on regional distribution of inspired gas. *J Appl Physiol* 1969;**26**:438–44.
[3] Connolly T, Bake L, Wood L, Milic Emili J. Regional distribution of a ¹³³Xe labelled gas volume inspired at constant flow rates. *Scand J Respir Dis* 1975;**56**:150–9.
[4] West JB. *Ventilation* 2nd ed. Oxford: Blackwell, 1970.
[5] Denison DM, Davies NJH, Meyer M, Pierce RJ, Scheid P. Single-exhalation method for study of lobar and segmental lung function by mass spectrometry in man. *Respir Physiol* 1980;**42**:87–99.
[6] Elkayam U, Wilson AF, Morrison J, Meltzer P, Davis J, Kosterman P, Louvier J, Henry WJ. Non-invasive measurement of cardiac output by a single breath constant expiratory technique. *Thorax* 1982;**39**:107–13.

Riedel's thyroiditis with multiple organ fibrosis

Sir,—In 1981 we reported a man with Riedel's thyroiditis who had extensive fibrosis in the upper parts of both lungs.[1] We now record the postmortem findings.

He presented in 1962 with a small lump in the thyroid and a diagnosis of Riedel's thyroiditis was made after biopsy. A chest radiograph was reported to show apical pleural thickening and by 1968 there were large opacities in the upper parts of both lungs. These increased in size and he became increasingly short of breath. He eventually developed right heart failure and he died with bronchopneumonia in December 1983 at the age of 80.

At postmortem examination the thyroid was replaced by dense fibrous tissue, which was constricting the trachea and infiltrating the strap muscles. The upper lobes, the right middle lobe, and the apical segment of the lower lobes were also replaced by fibrous tissue, which obliterated the adjacent pleural space (fig). Sections of this tissue show coarse interweaving bundles of collagen fibres, containing occasional fibroblasts, small lymphocytes, and small blood vessels. There was no continuity of fibrosis between the thyroid and the lungs. The caudal areas of lungs showed