

Tidal volume and inspiratory and expiratory times were measured for individual breaths over five minute periods before and after control and bupivacaine aerosol inhalations, and the data analysed by two way analysis of variance. We were unable to demonstrate an effect of the local anaesthetic aerosol on mean levels of tidal volume or inspiratory and expiratory times, though the variability of tidal volume was increased.³

In our experience the 5% bupivacaine aerosol used in that study provides a more profound and longlasting airway anaesthesia than a 4% lignocaine aerosol. Unfortunately, in Dr Fennerty's study anaesthesia at the end of the period of measurement was not established.

The small though statistically significant differences in resting ventilation found following airway anaesthesia may therefore be attributable to a method of measurement that affects the breathing pattern, a different degree of airway anaesthesia, and differences in the statistical treatment of data.

AJ WINNING
RD HAMILTON
A GUZ

*Department of Medicine
Charing Cross and Westminster Medical School
London W6 8RF*

- 1 Savoy J, Dhingra S, Anthonisen NR. Inhaled lidocaine aerosol changes resting human breathing pattern. *Respir Physiol* 1982;50:41-9.
- 2 Gilbert R, Auchincloss JH, Brodsky J, Boden W. Changes in tidal volume, frequency and ventilation induced by their measurement. *J Appl Physiol* 1972;33:252-4.
- 3 Winning AJ, Hamilton RD, Shea SA, Knott C, Guz A. The effect of airway anaesthesia on the control of breathing and the sensation of breathlessness in man. *Clin Sci* 1985;68:215-25.

* * This letter was sent to the authors, who reply below.

SIR,—We accept that the method used for measuring the breathing pattern in our patients was not ideal. To compensate for this the study was randomised and placebo controlled, and to check that our results were not artefactual three patients had their breathing frequencies measured by a non-stressful technique. Since the patients using mouthpieces were clearly under stress, it is reason-

able to propose that the results were biased against finding significant differences in resting ventilation following lignocaine. That this was the case is suggested by the fact that the baseline breathing frequency was lower, and the reduction following lignocaine larger, in those three patients using the non-stressful technique than in the seven patients using mouthpieces.

Duration of anaesthesia was tested before the study, and was found to be effective for a minimum of 15 minutes, the measurements being completed within 10 minutes of lignocaine inhalation.

The statistical point raised is valid and we have repeated our analysis using two way analysis of variance. The reduction in breathing frequency and the increase in expiratory time remains significant ($p = 0.01$ and 0.03 respectively).

It is known that stretch receptors do not influence inspiratory time at resting tidal volumes in normal subjects¹ and we were unable to observe an effect of these receptors on the breathing pattern of patients with chronic obstructive lung disease. The reduction in breathing frequency in our study was due to an increase in expiratory time rather than in inspiratory time, as occurred in the study of Savoy *et al*, and was probably due to blocking of irritant receptors. All our patients had evidence of bronchial inflammation, being chronic sputum producers. There is no reason to suppose that irritant receptors are activated in normal subjects, and this may explain the difference between our findings and those of Dr Winning and colleagues.

We do not feel that the points raised detract from our conclusion that stimulation of irritant airway receptors, while influencing breathing frequency, is not responsible for the alveolar hypoventilation, due to a reduction in inspiratory time, in patients with chronic bronchitis and obstructive lung disease.

A FENNERTY
J BANKS
C BEVAN
AP SMITH

*Llandough Hospital
Penarth, S Glam CF6 1XX*

- 1 Clark FJ, Van Euler C. On the regulation of depth and rate of breathing. *J Physiol* 1972;222:267-95.

Notice

Fleischner Society sixteenth annual symposium

The Fleischner Society will hold its 16th annual symposium on chest disease from 28 February to 3 March 1986 at the Marriott Hotel, Maui, Hawaii. Lectures, refresher courses, and panel discussions will be used to discuss imaging, anatomy, physiology, pathology, and clinical aspects of chest disease. Emphasis will be placed on the use of

imaging modalities and correlative studies. The registration fee is \$375 before 3 January 1986 and \$400 thereafter. The fee for residents in training is \$250. Further information may be obtained from the Fleischner Society Conference Coordinator, 3770 Tansy, San Diego, California 92121, USA.