Thorax 1985;**40**:879–880

Correspondence

Role of automatic staplers in the aetiology of bronchopleural fistula

SIR,—In their article in January 1985 (**40**:27–31) Mr M Hakim and Mr BB Milstein reported on the incidence of bronchopleural fistula following pneumonectomy. They suggest that the use of the TA-55 Premium instrument contributes to a significantly higher rate of bronchopleural fistula following pneumonectomy than the original style TA-55 stapler, and indicate that the differences are related to differences in design and function of the two staplers.

The authors state that the TA-55 Premium instrument "achieves closure by a toggle mechanism which is capable of generating large compression forces." This is incorrect. The fact is that the "toggle" mechanism of the TA-55 Premium instrument delivers 18–50% *less* tissue compression force than does the screw threaded original TA-55 instrument over comparable tissue thickness.

The authors state that the slotted hinge pin of the TA-55 Premium instrument cartridge is "an attempt to reduce the compression forces near the hinge." In fact, the design intent of the slotted hinge is to produce a parallel type of closure comparable to that of the original style TA instrument.

Additionally, the authors state that the movement of the hinge pin in the slotted hole produces "a mobile fulcrum." This is only true before the cartridge is loaded into the instrument. When the "toggle" lever is closed over tissue, the cartridge approximates to an even 2 mm across the entire length of the cartridge.

The authors claim that this study "confirms that closure of the bronchus with a parallel jaw stapler (TA-55) is uniform and independent of the forces applied on firing the staples." Actually, complete staple formation is almost exclusively dependent on the force exerted on the movable handle during firing. An incomplete handle squeeze could result in incomplete staple formation in either the TA-55 or the TA-55 Premium stapler.

Dr R Maurice Hood, professor of clinical surgery at New York University Medical Center, has related to us his experiences with both the original and the TA-Premium instruments. In over 900 lobectomies and pneumonectomies using both types of staplers Dr Hood and associates have experienced only three fistulas, two of which developed at six and nine months from recurrent carcinoma and one of which was associated intraoperatively by other means.

Additionally, we have had the opportunity to examine the two TA-55 Premium instruments used as the basis of the article by Mr Hakim and Mr Milstein. In both instances the instruments were found to be damaged in such a manner as to produce malformed staples, the malformation being such as to be quite possibly responsible for the fistulas which were observed.

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*** This letter was sent to Mr Hakim, who replies below.

SIR,—We are grateful for this opportunity to comment on Mr Hennig's letter.

1 Our measurements of the strain generated at the front jaw of the instruments using SGA 800 digital strain gauge monitor (CIL Electronics, Worthing) showed 200 strain units near the hinge and 125 strain units near the retaining pin with the Premium TA-55, compared with 20 strain units for the old style TA-55. Our results directly contradict the statement that the Premium TA-55 stapler delivers less tissue compression force.

2 The reasons for the design of the slotted hinge may be more than the manufacturer envisaged. Mr Hennig does not deny that the compression force at the hinge is thereby reduced.

3 We do not agree that when the TA-55 stapler is closed, the cartridge closes to "an even 2 mm across the entire length . . . ." Our measurements showed that the gap varied from 2 mm near the retaining pin to 3 mm at the hinge.

4 If it is true that complete staple closure is "almost exclusively dependent on the force exerted on the moveable handle . . ." why is it that this vital point is nowhere mentioned in the very detailed instructions for use? What explanation can we offer for the very different results with the two instruments?

5 With reference to the unpublished series of Dr Hood, we feel that we cannot comment in the absence of essential facts such as the number of pneumonectomy cases in which the Premium TA-55 stapler was used.

6 In response to Mr Hennig's comment concerning possible damage to the instruments, we have now tested the Premium TA-55 instruments which have been used at two other units. We encountered the same problem of incomplete and non-uniform staple formation.

7 Finally, we wonder why, if the design of the Premium TA-55 instrument is entirely satisfactory, "the more modern" instrument replacing it (TA II-55) is a return to the parallel closure TA-55 instrument.

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Role of airway receptors in the breathing pattern of patients with chronic obstructive lung disease

SIR,—We read with interest the paper on the study by Dr AG Fennerty and others (April 1985;**40**:268–71) in which they found changes in resting ventilation, measured by the use of a mouthpiece, in chronic obstructive lung disease following upper airway anaesthesia. A similar result has previously been reported in normal subjects, with the same method of measurement.[1] As Dr Fennerty and his colleagues comment, the use of a mouthpiece in patients with respiratory impairment is an unsatisfactory method of measuring the respiratory cycle. Because of concern that the breathing pattern of normal subjects and patients with chronic airways obstruction is affected by the presence of a mouthpiece and nose clip,[2] we have recently examined the effect of such anaesthesia on the resting respiratory pattern of normal subjects, using inductance plethysmography.

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.

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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 reasonable 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 subjects[1] 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.

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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.