LETTERS TO THE EDITOR

Guidelines for care during bronchoscopy

We read with interest the British Thoracic Society's guidelines for patient care during bronchoscopy (May 1993;**48**:584) and would like to offer some further relevant comments from the anaesthetist's point of view.

Pulse oximetry is important, but we would suggest that ECG and blood pressure monitoring should be included in all patients to assist in the early detection of cardiovascular complications. Diagnostic bronchoscopy has a mortality rate of 0·01–0·5% which is comparable to total anaesthetic and surgical mortality (0·042–0·63%) for which basic minimal monitoring requirements have already been recommended. This generally includes pulse oximetry, ECG, and blood pressure monitoring, and the Association of Anaesthetists of Great Britain and Ireland recommend that the same standards should be applied for sedation techniques and that additional monitoring is required when there is pre-existing medical disease.[1] It is logistically difficult to demonstrate the value of monitoring devices,[2] but to rely solely on one form of monitoring that may fail or give misleading information could be hazardous.

The BTS guidelines recommend that patients receiving drugs with potential respiratory depressant effects must have antidotes immediately available. Recently there has been interest in using the anaesthetic induction agent propofol to achieve sedation. Propofol may have advantages over opioid and benzodiazepine sedation in that recovery times are shorter.[3] It is, however, a powerful cardiorespiratory depressant and requires the presence of personnel experienced in airway control.

In our view joint bronchoscopy lists with anaesthetic personnel may be the best compromise. The patient will be more closely monitored and may receive better oxygenation and sedation, including the safer use of propofol; the physician gains exposure to airway management and resuscitation techniques and can concentrate on the procedure itself during difficult cases, and the trainee anaesthetist can become more familiar with awake techniques and with bronchial anatomy.

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1 Adams AP, Baird WLM, Sykes MK, Charlton JE. *Recommendations for standards of monitoring during anaesthesia and recovery*. London: The Association of Anaesthetists of Great Britain and Ireland, 1989.
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AUTHORS' REPLY We thank Drs Brimacombe and Berry for their interest in our guidelines for care during bronchoscopy. We agree that patients undergoing fibreoptic bronchoscopy should be monitored by pulse oximetry which allows for the detection of tachycardias, bradycardias, and pulse irregularity. Since we also emphasise the importance of having monitoring equipment readily available in the bronchoscopy suite and also of monitoring any patient with known cardiac problems or a history of dysrhythmias, we do not feel that ECG monitoring is necessary for every patient.

Blood pressure should be measured both before the procedure and during the recovery period, but we do not see a need for this to be undertaken in all patients whilst actually undergoing a bronchoscopy.

Respiratory physicians who wish to use intravenous sedation should use drugs with which they are familiar and to which an effective antidote is readily available. Propofol, an anaesthetic induction agent with powerful cardiorespiratory depressant effects, falls outside our guidelines and we agree that, if used, might necessitate the presence of extra personnel experienced in airway control.

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Computerised polysomnography

I read with great interest the paper by Drs H Biernacka and NJ Douglas evaluating a computerised polysomnography system (March 1993;**48**:280–3). The authors found that the CNS Sleep Lab is sufficiently accurate for use in clinical sleep studies in patients with mild sleep disordered breathing. This agrees with other studies evaluating computer aided sleep scoring.[1,2] It would be of interest to know the age and diagnosis of the patients investigated by the authors as automatic scoring results in higher agreement in subjects who are young or show a normal sleep pattern.[1,2] We have validated the CNS Sleep Lab in a faster but less interactive configuration in patients suspected of having obstructive sleep apnoea[3] and found a lower agreement for sleep stages and apnoea index in patients who were elderly (age 57 (7) years) and overweight (BMI 30 (4·3)). The agreement for sleep stages awake, IV, and REM was not significantly different, while the agreement for sleep stages I, II, and III was significantly better in 13 patients with BMI <30 compared with 14 patients with BMI ≥ 30. This is due to the poor signal quality in these obese patients. The mean (SE) apnoea + hypopnoea index was 29 (25) h for visual scoring and 21 (19) h ($p<0·05$) for computer scoring in all patients. The agreement for the apnoea + hypopnoea index was significantly correlated with the apnoea + hypopnoea index ($r=0·64$, $p<0·01$). Visual scoring took 186 (76) minutes and computer scoring took 53 (27) minutes ($p<0·05$). Thus care should be taken over extrapolating the results of Biernacka and Douglas to sleep studies in other patients and to sleep studies performed with a less interactive configuration of the CNS Sleep Lab. However, I fully agree that automated apnoea and sleep stage scoring at present is of only limited value.

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1 Stanus EL, Lacroix BP, Kerkhofs M, Mendlewicz J. Computer-aided sleep staging in clinical environment. *Neuropsychobiology* 1988;**20**:178–86.
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AUTHOR'S REPLY I noted the study by Andreas *et al* published two months before ours and performed on a smaller number of patients (27 *v* 43). Our patient mix was similar to theirs. I suspect the disparity in the apnoea indices between the two studies mainly relates to differences in the definition of what is an apnoea and what is a hypopnoea. Our criteria have been published and validated elsewhere and are referenced in our article.

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Chronic obstructive airways disease: terminology

Those of us who still have hypersensitivity reactions to terms such as "chronic obstructive airways disease" are now having to contend with a new phenomenon, "obstructive airways disease" (for example, the paper by Larsson *et al*, January 1994;**49**:41). What is this disease? If *Thorax* is prepared to publish papers about it, I feel you should define it for your readership. If, on the other hand, the authors simply mean airflow obstruction, why not say so?

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Endobronchial valves

We recently described a case of recurrent bronchopneumonia in a 12 year old child, caused by diverticulosis of the left main bronchus, demonstrated bronchographically (March 1993;**48**:187–8). The patient was seen again two years later because of another left lower lobe pneumonia. In fact, over the previous two years the patient had been admitted to another hospital four times for recurrent pneumonia in the left lung. Endoscopic examination showed a clockwise rotation of the left upper lobe bronchus and an inflamed stenosis of the left lower lobe bronchus, and bronchography showed a complete occlusion of the left lower lobe bronchus and bronchiectasis.

In view of the bronchographic picture a left lower lobectomy with removal of the larger diverticulum was performed. The anatomical findings confirmed the presence of the diverticulum and of numerous little valves in the left lower lobe bronchus and in its segmental bronchi, which we had observed at previous endoscopies. Histologically the valves consisted of connective and muscular tissue.

Bronchial diverticulosis is very rare and the cause is unknown. In our patient it was not associated with malformations in other major organs, and it presented as recurrent episodes of bronchopneumonia, always distal to the larger diverticulum. The other anomaly seen at fibreoptic bronchoscopy was numerous endobronchial valves in the lower lobe bronchi bilaterally. They were translucent in appearance and reduced the segmental bronchial lumen to about 10% of normal. The anatomical and histological examination of the resected lobe confirmed the presence of valves along the lobar and segmental bronchi. The association of bronchial diverticulosis and bronchial valves has not been previously described in a child.

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LTOT in COPD

The recent article by Dr M I Walters *et al* (November 1993;**48**:1170–7) provided a useful review of the use of long term oxygen therapy (LTOT) in chronic obstructive pulmonary disease (COPD), but it is disappointing that recommendations as to the appropriate use of LTOT remain indistinct. The UK Department of Health absolute indication (suitably hypoxic patients with COPD and a history of oedema) is unambiguous and will be based on the findings of the MRC study.[1] The "relative" indication of LTOT in the much larger group of hypoxic patients with COPD but no history of oedema is less clear, because the NOTT study[2] included a heterogenous group of patients with and without a history of oedema. This study did find that continuous oxygen therapy conferred a survival benefit to, particularly, hypercapnic patients ($PaCO_2$ > 45 mm Hg). It is, however, not clear from the NOTT study what proportion of these patients had a history of oedema, although one might speculate that this subgroup (hypoxic and hypercapnic patients) would have the highest incidence of oedema and thus be the patients most comparable to those included in the MRC study. Whether or not the findings from the NOTT study can be extrapolated to the *whole* population of patients with hypoxic COPD, with or without hypercapnia, is arguable.

Clinical experience suggests that only a few patients with hypoxic COPD develop cor pulmonale. The FEV_1 remains a powerful predictor of survival which continues to decline irrespective of whether the patient receives LTOT. It is thus possible that LTOT may be unable to prolong survival in patients in whom oedema has not occurred. In their review Walters *et al* quote a number of smaller studies which have examined physiological variables such as pulmonary artery pressure in such patients – often with conflicting conclusions. Indeed they state "even if reversal of pulmonary hypertension is confirmed, its relevance in terms of survival is not yet clear."

There are, of course, issues other than survival which need to be considered – and many might argue that LTOT could improve quality of life rather than survival, and should for this reason be given to hypoxic patients without oedema. This is an important issue. The prevalence of non-oedema hypoxic COPD is such[3] that many thousands of patients fulfil the vague "relative" criteria for the provision of LTOT. Surely there is still a need for a further study to examine specifically the role of LTOT in hypoxic patients with COPD but *no* history of oedema. The principal end point should be survival although "quality of life" issues could be addressed. It might even be possible to increase our limited understanding of the significance of nocturnal desaturation in these patients. This should not be beyond our means or enthusiasm; after all, the much quoted MRC report[1] only studied 87 patients.

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1 Report of the Medical Research Council Oxygen Working Party. Long-term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981;i:681–5.
2 Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxic chronic obstructive lung disease. *Ann Intern Med* 1990;**93**:391–8.
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We read with interest the review on long term oxygen therapy (LTOT) by Dr M I Walters *et al* (November 1993;**48**:1170–7). We would, however, like to make several additional points about current usage of LTOT in the UK.

While we agree that overall there is underprescription of LTOT, it is no longer true that LTOT is prescribed inappropriately and for inadequate durations in the majority of patients. In our study of 176 patients using LTOT in East London in 1991[1] the prescription for LTOT was recommended by a respiratory physician in 80% of patients and simplified criteria for the prescription of LTOT were fulfilled by 76% of patients. Furthermore, not only were 83% of patients prescribed 15 hours of LTOT or more daily, but 74% of patients used LTOT for more than 12 hours daily.

Hence, use of the guidelines for the prescription of LTOT has improved compared with earlier studies of the prescription of LTOT[2,3] which assessed adherence to the guidelines within the first two years of their introduction in December 1985. Nonetheless, use of LTOT could be further optimised. Firstly, communication about patients' respiratory status between hospital physicians and general practitioners needs to be improved. In our study[1] the general practitioner was only aware of the results of respiratory assessment in about 30% of patients. Secondly, we agree with Walters *et al* that regular reassessments are essential for the effective prescription of LTOT. We found that some patients were still being assessed for LTOT during an exacerbation and oxygen saturation was uncorrected by the concentrator in 17% of patients[1]. Reassessments would maximise the benefit of LTOT by ensuring optimal correction of hypoxaemia and that prescription is appropriate in all patients.

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1 Restrick LJ, Paul EA, Braid GM, Cullinan P, Moore-Gillon J, Wedzicha JA. Assessment and follow up of patients prescribed long term oxygen treatment. *Thorax* 1993;**48**:708–13.
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Sleep Apnea and Rhonchopathy. K Togawa, S Katayama, Y Hishikawa, Y Ohta, T Horie. (Pp 178; SFr 246, US$197.) Basel, Switzerland: Karger, 1993. 3 8055 5611 X.

This book reports the proceedings of the 3rd World Congress on Sleep Apnea and Rhonchopathy held in Tokyo, Japan on 21–23 September 1991. All five editors are Japanese and 23 of the 32 articles have Japanese authors. The 32 articles span 174 pages, so the average article is six pages in length. They amount therefore to little more than extended abstracts and occasionally one finds this frustrating. There are undoubtedly some interesting contributions including articles on the upper airways resistance syndrome from Stanford, driving in patients with sleep apnoea from Stockholm, changes in blood gas tensions during apnoeas from Tokyo, and the value of dynamic cephalometry from Sagamihara and Michigan. However, there are several articles which add little to the world knowledge of sleep apnoea.

I have to confess to a major bias against the publication of books of proceedings. Books of extended abstracts published two years after a meeting are out of date because any article containing good original science will already have been published in full in reputable journals. Indeed, this is the case for several of the articles in this book. Such volumes merely serve to make profits for publishers and pad curriculum vitae of contributors, and I personally would not advocate purchase of many of them. I certainly would not advocate purchase of this one, unless you have a burning desire to know what is going on in terms of sleep apnoea in Japan. – ND