LETTERS TO THE EDITOR

Nosocomial pneumonia during mechanical ventilation

We read with interest the editorial by Dr Brun-Buisson published in the November issue (1995;80:1128–30). Since part of our work was alluded to in several parts of the text, we feel it necessary to reply to avoid confusion to the readers interested in the field of the diagnosis of ventilator-associated pneumonia. Dr Brun-Buisson stated that, in one of our papers,1 we concluded that lung histological and microbiological findings on immediate necropsy biopsy samples are inadequate “gold standards” in patients receiving antibiotics. Actually, our conclusion was that quantitative cultures of immediate necropsy biopsies are not a good gold standard since they cannot discriminate the histological absence or presence of pneumonia (page 331, first paragraph, reference 2). Our recommendations for future studies were to use the histopathology of necropsy pulmonary biopsies as a gold standard, comparing patients with and without antibiotics and considering different time intervals (page 331, second paragraph, reference 2). The results of this study1 have been confirmed by others2-4 and only the recent work by Chastre et al5 differs with most of the necropsy studies performed upon humans. However, one can argue, as the author did, that our results and those of others could be biased by the presence of prior antibiotic treatment which is, on the other hand, a frequent and real situation when managing mechanically ventilated patients who develop VAP. For that reason, we repeated our necropsy study using a human model of multiple bilateral biopsy sampling with an average of 16 biopsy specimens per patient. This time we also included patients free from antibiotics (at least 48 hours before dying), and compared these patients with those who received antibiotics. The results were exactly the same as those of our previous work, irrespective of the presence or absence of antibiotics. Furthermore, when classifying pneumonia into four different evolution categories, quantitative cultures of lung biopsy specimens could not discriminate between the presence or absence of pneumonia. In view of this work and that of previous authors,1-4 we believe that histology (with positive or negative cultures) is the only acceptable gold standard of VAP. Obviously, experienced pathologists have to differentiate initial periods of VAP from initial periods of diffuse alveolar damage when using necropsy histology to validate diagnostic techniques for pneumonia.

In another part of the editorial Dr Brun-Buisson refers to the inaccuracy of quantitave cultures of endotracheal aspirates to diagnose VAP. This is a controversial issue in the literature. However, at least five studies2-6 support quantitative cultures of endotracheal aspirates for the diagnosis of VAP. One of these studies6 has been performed in a post mortem human model. We think that a sampling method which omits bronchoscopic techniques should be taken into account for the routine management of patients with VAP. We agree with Dr Brun-Buisson that the best way to study the specificity of diagnostic methods in the context of VAP is in patients without pneumonia as we have done previously.1,2 Dr Brun-Buisson suggested technical problems in our paper concerning oral contamination since high counts of non-pathogenic oropharyngeal organisms were reported. Although no explicit reference was made to the type of technical problems, these can be due to sampling error or oral contamination. With regard to sampling, and since diagnosis of VAP with bronchoscopic techniques is not an issue for amateurs, one experienced member of staff with a particular interest in the field (AT) personally performed most of the techniques for that study which removes the possibility of sampling errors.

With regard to oral contamination, our study2 shows that only two of 12 (16%) microorganisms in samples from prebronchoscopic brush samples of controls (with 10^6 cfu/ml) were non-pathogenic oropharyngeal flora. In bronchoalveolar lavage samples all but one isolate (10^6 cfu/ml) did not correspond to oropharyngeal flora. When carefully analysed, our results indicate that oral contamination was not a major problem in that study. Furthermore, a study of identical design concerning the specificity of the techniques2 confirmed our results in mechanically ventilated patients with suspected pneumonia. On the other hand, this study fits very well with the concept that mechanically ventilated patients frequently have colonised distal airways. Our necropsy studies have confirmed this.

We hope that our comments will help the reader to better understand the editorial comment by Dr Brun-Buisson.

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AUTHOR’S REPLY I agree with Torres and colleagues that lung pathology, when examined by experienced pathologists, should be considered as the test least subject to bias for determining the presence or absence of pneumonia. The purpose of my editorial was to highlight problems that arise when this technique is applied to patients who have received mechanical ventilation for some time, often have associated lung disease and have received multiple antibiotics, and eventually have died at an unknown time delay from prior or ongoing pulmonary infection. In this context, lung pathology appears to be especially difficult to use when attempting to assess the diagnostic value of various sampling techniques of respiratory secretions, which are being relatively substituted to lung biopsy cultures in intubated patients.

I hasten to add that the technical skills of Torres and coworkers to perform bronchoscopy. The problem I see with some studies that use lung pathology10 is that their results are contradictory to some well accepted principles – for example, that pulmon- ary infection (as one would expect from any bacterial infection) is associated with substantial growth (in the range of 10^5 cfu/g) from lung tissue,11 and that tracheal aspira- tes of ventilator patients are relatively snapshot time samples, which are a novel culture for pneumonia but a non-specific one during prolonged tracheal intubation5 because of frequent colonisation of the upper and even the lower airways. Clearly, the finding of pathological aspects “typical of pneumonia” is associated with no or insignificant growth from lung specimen cultures as reported by Torres et al and others11 raises questions. Obviously, there are problems with either the interpretation of lung biopsy cultures of endotracheal aspirates for the diagnosis of nosocomial pneumonia. Am Rev Respir Dis 1995;152:1982–91.

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lungs pathology and cultures of respiratory secretions.

Numerous studies have been performed in this area in recent years in an attempt to clarify the diagnostic approach to the so-called "idiopathic pneumonitis." It would be unfortunate if such efforts were wasted simply because of misinterpretation. The practical issue for physicians who care for patients on mechanical ventilation is to learn to recognize when reliance on the sampling techniques they routinely use when pulmonary infection is suspected. Taken together, the studies by Torres et al and others simply suggest that tracheal aspirates are just as good as other more "sophisticated" techniques using quantitative cultures and/or protected specimens, with or without bronchoscopy. I have already mentioned the potential impact of this approach on antibiotic usage in intensive care units and its consequences. But are we 25 years behind in the diagnosis of pulmonary infection?

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BOOK REVIEW

Silica exposure and risk of lung cancer

The objective review by Weil and McDonald (January 1996;51:97-102) of the role of silica exposure and the risk of lung cancer was long overdue and thoroughly needed. The authors endorse, albeit to a limited extent, the study by Checkoway et al of diatomite workers,1 but in doing so they do not mention a most important observation that casts doubt on the alleged excess risk of lung cancer in the diatomite cohort. Thus, the overall SMR for lung cancer was 1.43, a figure which suggests a mildly increased risk. Lacking a smoking history and in an effort to control the confounding effects of smoking, the authors calculated the SMRs for other cigarette smoke “induced” cancers – namely, those of the larynx, bladder, kidney, and oesophagus – and found no increase. While all of these cancers are related to cigarette smoking to some extent, the association is much more tenuous than it is for lung cancer. Other factors, in particular alcohol and diet, also have a significant effect. In contrast, the SMR for emphysema for the diatomite workers was 180, indicating a greater risk of dying from emphysema than from lung cancer. The cause and effect relationship between cigarette smoking and emphysema is as compelling, if not more so, than it is between lung cancer and smoking. Moreover, there is virtually no cause of disabling emphysema leading to death other than cigarette smoking.

Weil and McDonald correctly cast doubt on studies that rely on subjects selected from silicosis registries or from registries of those who have been compensated. One of the papers referred to is that of Ng et al who studied subjects with silicosis in Singapore.2 The paper stated that over 90% of their cohort were smokers, compared with 60% of the general population. It is difficult to understand how so many studies that rely on silicosis registries and their like find their way into print. The question as to whether exposure to silica per se or silicosis is associated with an increased rate of lung cancer cannot be answered by statistical manipulation of defective data; statistics obviously have a role to play but, as Bradford Hill pointed out many years ago, only in conjunction with other criteria, the most important of which is biological plausibility.

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This book, edited by two of the most eminent men in the field, is composed of 17 chapters by current contributors in the area of smoking and health.

The chapters on price and consumption of tobacco by Townsend, on tobacco and the developing world by Mackay and Crofton, and on children and smoking: the family circle by Charlton are particularly pertinent and well written. Epidemiological and mortalitity data are presented in two clear, easily digested chapters by Wald, Petco and co-authors. Sir Richard Doll deals with cancers weakly related to smoking. Authors from the Barr’s group spell out the harm from passive smoking and evidence of its effects on the respiratory and cardiovascular systems as well as during pregnancy.

The chapters by Reid on tobacco control, Chapman on advertising, and Pollock on the tobacco industry make stimulating, informative reading and are complemented by accounts of the current legal position of tobacco and of the industry’s tactics.

There are informative chapters on smokeless tobacco, the history of tobacco substitues, and the moves to reduce tar and nicotine levels. Baron provides a balanced discussion of the putative beneficial effects of nicotine and cigarette smoking, and a chapter is devoted to women as a vulnerable target group. The overview of cessation by Foulds is a competent account delivered from the viewpoint of a psychologist in the field, but perhaps more space could have been given to work and progress with patients.

This readable book is up to date, comprehensive, and well referenced. It is likely to be of interest to a wide audience than just medics, paramedics, public health experts, psychologists, and students who wish to be well informed. I recommend it not just to interested individuals, hospital and university libraries, but also to the general public.

NOTICES

International Lung Sounds Association

The 21st International Conference on Lung Sounds will be held in Chester, UK on 4—6 September 1996. For further information please contact Raymond L H Murphy Jr, Faulkner Hospital, 1153 Centre Street, Boston, MA 02130, USA (Telephone: 617 925-5800, x 1968; Fax: 617 522-4156) or John Earsie, Liverpool Medical Institution, 114 Mount Pleasant, Liverpool L 3 5SR, UK (Telephone: 151 709 1295; Fax: 151 709 2810). Internet address: HTTP://WWW. UMNITOBA.CA/FACULTIES/ MEDICINE/PEDIATRICS/CHILD HEALTH/ILSA/

4th International Congress on the Immune Consequences of Trauma, Shock and Sepsis

The 4th International Congress on the Immune Consequences of Trauma, Shock and Sepsis – Mechanisms and Therapeutic Approaches will be held in Munich on 4—8 March 1997. The deadline for abstracts is 30 October 1996. For further information contact Prof Dr med E Faist, Ludwig-Maximilians-University Munich, Klinikum Grosshadern, Dept of Surgery, Marchioninistrasse 15, 81377 Munich, Germany. Phone: 49-89-7095-3441/2461. Fax: 49-89-7095-2460.

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Nosocomial pneumonia during mechanical ventilation.

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Thorax 1996 51: 771-772
doi: 10.1136/thx.51.7.771

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