REVIEW SERIES

The pulmonary physician in critical care: towards comprehensive critical care?

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Thorax 2002;**57**:77–78

This overview of intensive care medicine in Europe and the United States is an introduction to the review series on "The pulmonary physician in critical care" which starts in this issue of *Thorax*.

In Europe intensive care medicine has been one of the most recent clinical disciplines to evolve. During a polio epidemic in Denmark in the early 1950s mortality was dramatically reduced by the application of positive pressure ventilation to patients who had developed respiratory failure, concentrating them in a designated area with medical staff in constant attendance. This focus on airway care and ventilatory management led to the gradual introduction of intensive care units (ICU), principally by anaesthesiologists, throughout Western Europe. The development of sophisticated physiological monitoring equipment in the 1960s facilitated the diagnostic role of the intensivist, extending their skill base beyond anaesthesiology and attracting clinicians trained in general internal medicine into the ICU. Moreover, because respiratory failure was (and still is) the most common cause of ICU admission, pulmonary physicians, particularly in the USA, were frequently involved in patient care. Many advances in the care of the critically ill have been made since the last series on intensive care medicine was published in *Thorax* in 1992,[1] and we have attempted to summarise some of these. The number and range of contributions to this series has therefore increased considerably and the current series will run over 17 issues. We have attempted to reflect the growing subspecialty interest of respiratory physicians in managing patients in ICU and high dependency facilities, as well as the large number of respiratory diseases causing or complicating critical illness that may require a respiratory opinion.

TRAINING IN INTENSIVE CARE MEDICINE

It would be satisfying to conclude that increased understanding of the pathophysiology of critical illness alone has been responsible for improvements in ICU outcome. However, improved clinical training and organisational changes have undoubtedly played their part. The global status of intensive care medicine is evolving. Board certification is established in the United States, albeit through either respiratory medicine or anaesthesia as base specialties. Furthermore, intensive care medicine is now a recognised specialty in two European Union member states (Spain and the UK). Where available, training in intensive care medicine is of variable duration and its accessibility to clinicians of differing base specialties varies. In Spain 4 years of training are required to achieve specialist status, 2 years of which is in intensive care medicine. In France, Germany, Greece, and the UK 2 years of training in intensive care medicine is required in addition to that needed for the base specialty (usually anaesthesiology, pulmonology, or general internal medicine). In Italy only anaesthesiologists may legally practise intensive care medicine. Currently, there is considerable variation between member states of the European Union regarding the amount of exposure to intensive care medicine in the training of pulmonary physicians as a mandatory (M) or optional (O) requirement: France and Greece 6 months (O), Germany 6 months (M, as part of general internal medicine), UK 3 months (O), Italy and Spain none. Respiratory specialist registrars who want to develop an interest in this area should be encouraged to know that 6 months of anaesthesia and 6 months of intensive care medicine may contribute to their training in general internal medicine and respiratory medicine, respectively.

DOES INTENSIVE CARE WORK?

Does intensive care work and does the way in which it is provided affect patients' outcomes? A higher rate of attributable mortality has been documented in patients who are refused intensive care, particularly on an emergency basis.[2] Clinical outcome is improved by the conversion of so-called "open" intensive care units to closed facilities in which patient management is directed primarily by intensive care specialists.[3 4] Superior organisational practices emphasising strong medical and nursing leadership can also improve outcome.[5] The emergence of intermediate care, high dependency, or step down facilities emphasises the growing gap between clinical practice in the ICU and the general wards. Hence, the time at which patients are discharged from intensive care affects their outcome.[6] Early identification of patients at risk of death—both before admission and after discharge from the ICU—may decrease mortality.[7] Patients can be identified who have a low risk of mortality and who are likely to benefit from a brief period of high dependency care.[8] The impact of specialist retrieval teams in moving critically ill patients between specialist units may also be relevant.[9] Finally, long term follow up of the critically ill as outpatients following discharge from hospital may identify problems of chronic ill health that require active management and physical/mental rehabilitation.[10]

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Table 1 Proposed classification of critical illness[11]

Level 0	Patients whose needs can be met through normal ward care in an acute hospital
Level 1	Patients at risk of their condition deteriorating, or those recently relocated from higher levels of care, whose needs can be met on an acute ward with additional advice and support from the critical care team
Level 2	Patients requiring more detailed observations or intervention including support for a single failing organ system or postoperative care and those "stepping down" from higher levels of care
Level 3	Patients requiring advanced respiratory support alone or basic respiratory support together with support of at least two organ systems. This level includes all complex patients requiring support for multiorgan failure

FUTURE DEVELOPMENT OF INTENSIVE CARE MEDICINE

The changing requirements and increased need for provision of intensive care were recognised in the UK in the late 1990s by the Department of Health which commissioned the report entitled "*Comprehensive Critical Care*" produced by an expert group to provide a blue print for the future development of intensive care within the NHS.[11] A central tenet of the report is the idea that the service should extend to the provision of critical care throughout the hospital, and not merely to patients located within the traditional confines of the ICU. To this end, the adoption of a new classification of illness severity based on dependency rather than location was recommended. Traditionally, the critically ill were defined according to their need for intensive care (delivered at a ratio of one nurse to one patient) and those requiring high dependency care (delivered at a ratio of one nurse to two or more patients). The new classification is based on the severity of the patient's illness and on the level of care needed (table 1). The report therefore represents a "whole systems" approach encompassing the provision of care, both before and after the acute episode within an integrated system.

How should the respiratory physician react to these changes? Firstly, we hope this series will increase awareness of the range of clinical problems likely to be encountered in the ICU. Secondly, we suggest that an attachment in intensive care medicine for all respiratory trainees is increasingly necessary. Indeed, specialty recognition and the increased availability of training opportunities should encourage some trainees from respiratory medicine to seek a certificate of completion of specialist training (CCST) combined with intensive care medicine. Thirdly, we suggest that changes in the organisational and administrative structure of intensive care services heralded by the publication of "*Comprehensive Critical Care*" are likely to impact most heavily on respiratory physicians. For example, respiratory support services using non-invasive ventilation are particularly attractive in providing both "step up" (from the general wards) and "step down" (from the ICU) facilities. In the USA, respiratory physicians have for a long time been the major providers of critical care. In the UK, given appropriate resources and training, the pulmonary physician is ideally suited to become an integral and vital component of the critical care service within all NHS trusts.

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PostScript

LETTERS TO THE EDITOR

BTS guidelines on CAP

The new BTS guidelines on the management of community acquired pneumonia (CAP) in adults[1] are welcome if they lead to improved diagnosis of pneumonia, better assessment of severity of illness, and thus more appropriate treatment according to clinical needs. It is widely accepted, however, that inappropriate implementation of the previous guideline contributed to large increases in unnecessary use of broad spectrum antibiotics with resultant increases in antibiotic resistance and *Clostridium difficile* infection. The authors acknowledge this, but the new guidelines seem likely to continue this unfortunate trend.

Firstly, there is no mention of the use of oral penicillin for treatment of mild cases of CAP. This is a first line choice in Scandinavian countries which have a commendably restrained history of antibiotic use (and consequently low rates of resistance).[2] The new BTS guideline recommendation for widespread use of the broader spectrum amoxicillin cannot help current antibiotic resistance problems. The pharmacodynamic arguments favouring amoxicillin may be important in those areas having problems with penicillin intermediate and resistant pneumococci, but in many areas of the UK—including much of Scotland—these strains are rare.[3] Did the authors consider oral penicillin as an option for mild cases?

Secondly, for treatment of severe pneumonia there is no mention of parenteral penicillin. The recommendation of co-amoxiclav or cefuroxime for this condition, while covering uncommon Gram negative pathogens and methicillin sensitive *Staphylococcus aureus* (MSSA), may lead to inadequate treatment of CAP due to penicillin resistant pneumococci. Surely benzyl penicillin is an option in young previously healthy people with severe CAP (the majority of whom will have pneumococcal infection).[4] Then, if there is a reasonable risk of infection with a pneumococcus with reduced susceptibility to penicillin, the dose of benzyl penicillin can be raised accordingly.

Thirdly, the recommendations for macrolide use in the first version of the guideline have probably been the main reason for the doubling of macrolide consumption in our local hospital since the previous guidelines were introduced (unpublished observation). If this observation is indicative of a more widespread trend, it may well be contributing to the current national problem with MRSA and other macrolide resistant organisms. To what benefit I wonder? Certainly, a laboratory diagnosis of atypical pneumonia is rare in our population. Isn't this another case for stratifying patients according to risk rather than treating all severely ill hospitalised patients with a macrolide?

I appreciate the huge body of evidence considered by the authors and the disappointing number of studies which were helpful in guiding best recommendations for treatment. Nevertheless, at a time when there is widespread concern about inappropriate antibiotic use, much of it with broad spectrum agents, it is crucial that new guidelines urge restrained prescribing unless the risks (inadequate spectrum) clearly outweigh the benefits (reduced ecological damage). At the same time, severe cases require the best treatment and this should not be compromised out of a desire to do the impossible and cover all conceivable (but unlikely) pathogens all of the time.

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Transudates and exudates

Joseph *et al* have made a valuable contribution to the evaluation of pleural effusions.[1] However, we would like to sound a note of caution. Throughout all the literature, including the study by Joseph *et al*, one message remains the same: no single test is diagnostic for transudates or exudates.[2] Thus, overreliance on such a test can be very misleading and lead to either under or over-investigation.

Rarely in the literature is there any discussion regarding the *place* of pleural fluid protein or lactate dehydrogenase (LDH) estimation. Specifically, how does it alter management? Does the finding of a transudate obviate the need for further investigation? The main problem is that a significant number of malignant effusions are classified as transudates, whichever method is used.

The cause of a transudate is usually clinically obvious. If, however, there is no obvious underlying cause, surely cytological and/or histological examination should still be sought, as for an exudate?

Estimation of pleural fluid protein or LDH is also irrelevant if the fluid is bloodstained, as here further investigation for possible malignancy is warranted anyway.

We propose that the principal use for pleural fluid protein or LDH measurement is when a probable underlying cause for a transudative effusion is apparent, such as heart failure or hypoalbuminaemia, and the fluid is not bloodstained. In this situation the finding of a transudate may help to reassure that no further investigation is necessary except observation, and that a trial of treatment with, for example, diuretics may be of help.

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Authors' reply

We appreciate the comments by Quantrill and Dabal on our recent paper[1] and would like to clarify the issues raised by them. By definition, when the pleural fluid is classified as a transudate, it indicates that a pathological process does not involve the pleural surface and that an effusion is formed because of a hydrostatic imbalance. If the pleural fluid is bloodstained, it therefore suggests disruption of the pleural membrane by an inflammatory or malignant process and hence cannot be classified as a transudate, which obviates the need for estimation of fluid LDH or protein estimation for diagnostic classification. However, as suggested by Quantrill and Dabal, an occasional malignancy may present as a transudate, in which case the mechanism is usually an effusion from collapse of a lobe causing an increase in the negative pleural pressure. Whatever the mechanism, if clinical suspicion for malignancy is high, further appropriate investigations need to be carried out.

Furthermore, Quantrill and Dabal state that hypoalbuminaemia is an apparent cause for transudative effusions.[2] However, recent literature shows that hypoalbuminaemia per se may not cause pleural effusions.[3] In our paper we have provided the positive likelihood ratios of the various tests so a clinician armed with the pretest probability for any individual patient and the positive likelihood ratio can work out the post-test probability using a standard nomogram.[4 5] In light of the above, we suggest that fluid LDH and total protein ratio are useful in the diagnostic separation of pleural effusions.

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Mycobacterium xenopi

We read with interest the report by Bachmeyer *et al* on *Mycobacterium xenopi* pulmonary infection manifesting in an HIV infected patient after receiving highly active antiretroviral treatment (HAART).[1] The diagnosis was made based on clinical, radiological, and histological findings of a granuloma in addition to one sputum specimen growing *M xenopi*. We think that the patient may meet the criteria set by the ATS for diagnosis and treatment of disease caused by non-tuberculous mycobacteria (NTM) but, according to this guideline, these recommendations fit best for *M avium* complex, *M kansasii*, and *M abscessus*. Too little is known about other NTM (such as *M xenopi*) and how applicable these criteria are to them.[2] This case may be one of those situations where it is difficult to make a definitive diagnosis.

M xenopi is usually a non-pathogenic coloniser of airways that has occasionally been associated with nosocomial outbreaks related to growth in hospital hot water systems.[3 4] A recent publication showed the incidence of *M xenopi* isolates in a large urban hospital and its pathogenicity to be low.[5] Tuberculosis would have the same clinical/radiological presentation and would have improved with the same treatment given to the patient.[6 7] The persistent negativity of tuberculin skin testing (TST) despite the increase in CD4 cell count cannot be used to exclude tuberculosis. TST has a high false negative rate even among non-HIV infected patients with confirmed tuberculosis.

While the management of this case would not have differed had the patient been treated as a presumed case of tuberculosis, it is important to keep in mind the need for contact investigation and appropriate public health interventions for tuberculosis cases.

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Authors' reply

We thank Drs Salazar-Schicchi and Nachman for their interest in our paper and their valuable comments. However, we consider that *Mycobacterium xenopi* was responsible for the patient's disease despite the fact that the microbiological diagnosis was not "definitive". Indeed, the criteria of the American Thoracic Society were not fulfilled.[1] These criteria do not seem to be applicable to *M xenopi* in HIV infected patients, in whom two positive cultures of *M xenopi* and no other cause of symptoms have been proposed as criteria for the diagnosis.[2] Our patient also did not fulfil these criteria. Indeed, we were concerned about the possible role of other pathogens—especially *M tuberculosis*—since coexistent pulmonary infections due to other pathogens had been reported.[3] However, no other pathogens were found and a search for *M tuberculosis* in the three sputum samples and bronchoalveolar lavage fluid was negative on direct microscopic examination and culture. This is rare in cavitatory tuberculosis and makes this diagnosis unlikely.

Mycobacterium xenopi may be found in hospital water taps, hot water storage tanks, and contaminated bronchoscopes.[4] Environmental contamination seemed unlikely since *M xenopi* was not isolated from samples in the microbiology laboratory during the period of management of our patient.

We conclude that *M xenopi* can be the cause of a lung disease in HIV infected patients that resembles tuberculosis and clinicians should not disregard the significance of this organism when isolated from respiratory specimen, even from only one.

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NOTICE

Pharmacology of Asthma

A course on the "Pharmacology of Asthma" suitable for physicians or scientists with an interest in the pharmacology and therapeutics of asthma organised by Professor Peter Barnes will be held on 25–28 November 2002. For futher details contact the Postgraduate Education Centre, National Heart & Lung Institute, Faculty of Medicine, Imperial College, Dovehouse Street, London SW3 6LY, UK. Telephone: 020 7351 8172. Fax: 020 7351 8246. E-mail: shortcourses.nhli@ic.ac.uk.

CORRECTIONS

Critical care training in Spain

In the review entitled "The pulmonary physician in critical care: towards comprehensive critical care?" by M J D Griffiths and T W Evans which appeared in the January issue of *Thorax* (2002;**57**:77–8), it was incorrectly stated that: "In Spain 4 years of training are required to achieve specialist status, 2 years of which are in intensive care medicine". This should have read: "In Spain **5** years of training are required to achieve specialist staatus, **3** years of which are in intensive care medicine".

Low dose of inhaled steroids and prevention of asthma death

In the paper by J C Kips and R A Pauwels entitled "Low dose inhaled corticosteroids and the prevention of death from asthma" which appeared in the 2001 Year in Review published as Supplement II in September 2001 (*Thorax* 2001;**56**(Suppl II):ii74–ii78), an error occurred in the abstract of the Introductory article by Suissa *et al* (*N Engl J Med* 2000;**343**:332–6). In the Results section it is stated that "... the rate of death from asthma decreased by 2% with each additional canister of inhaled corticosteroids used in the previous year ...". This should have read "... the rate of death from asthma decreased by **21%** with each additional canister of inhaled corticosteroids used in the previous year ...". The publishers apologise for this error.