LETTERS

Role of low-dose theophyllines in exacerbations of COPD

In the paper by Cosio and colleagues on the effect of low-dose theophylline on the anti-inflammatory effects of steroids during exacerbations of chronic obstructive pulmonary disease (COPD), the authors concluded that recovery of forced expiratory volume in 1 s 3 months after the exacerbation and low mortality (although not statistically significant) in the theophylline group, in combination with the molecular effects described, provided a strong rationale for investigating further the potential clinical relevance of this therapeutic strategy in large randomised double-blind placebo controlled trials.[1]

In such trials, if the time of hospital discharge is taken as the end point instead of taking 3 months after the exacerbation as in the study by Cosio *et al*, it would better reflect the physiological events of a COPD exacerbation. In addition to addressing the limitations expressed by the authors in the article, consideration of clinical parameters such as symptoms, blood gases and the need for non-invasive ventilation in the outcome measures might add more strength to the evidence. Another factor which also needs to be considered is whether patients with COPD are on a pulmonary rehabilitation programme.

A B Palamarthy

Correspondence to Dr A B Palamarthy, All Wales Adult Cystic Fibrosis Centre University Hospital, Llandough Vale of Glamorgan CF64 2HR, UK; anandom999@yahoo.com

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1. **Cosio BG,** Iglesias A, Rios A, *et al*. Low-dose theophylline enhances the anti-inflammatory effects of steroids during exacerbations of COPD. *Thorax* 2009;**64**: 424–9.

Author's reply

We are grateful for Dr Palamarthy's comments on our paper. However, we disagree with his suggestion of taking the time of hospital discharge instead of 3 months as the endpoint because the aim of our intervention was to decrease inflammation and it is well known, as reported by Perera and colleagues,[1] that inflammation and symptoms may not recover to baseline even 35 days after an exacerbation. In trials that we are about to start, in which patients will be recruited while stable, we have considered clinical outcomes as well as inflammatory and mechanistic variables but, in our view, the time to first exacerbation and the rate of exacerbations would be more reflective of the mechanism of action that we are proposing for low-dose theophylline. Rehabilitation is important and needs to be considered for daily clinical practice, but both arms would eventually be exposed to it so it should not be a confounding factor.

B G Cosio

Correspondence to Dr B G Cosio, Department of Respiratory Medicine, Hospital Universitario Son Dureta, Andrea Doria 55, 07014 Palma de Mallorca, Spain; borja.cosio@ssib.es

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REFERENCE

1. **Perera WR,** Hurst JR, Wilkinson TM, *et al*. Inflammatory changes, recovery and recurrence at COPD exacerbation. *Eur Respir J* 2007;**29**: 527–34.

Risk disclosure prior to bronchoscopy

We read with great interest the recent article by Uzbeck *et al*[1] demonstrating increased anxiety in patients following detailed risk disclosure prior to bronchoscopy. This timely well-conducted study engages with the challenging area of informed consent, and the authors eloquently outline the difficulties in balancing patient autonomy and physician desire to minimise patient anxiety but also to avoid litigation. We agree that the small increase in patient anxiety following explicit consent "seems a price worth paying for most patients". We wish to comment, however, on a couple of methodological issues.

The study information sheets were provided on the day of bronchoscopy which allowed little time to rationalise the information and also discussed prominently the complications of transbronchial biopsy, although not all patients would have undergone this riskier procedure.

We have recently surveyed the consenting practices of 33 respiratory physicians in the north-east of England. Thirty-one respondents always provided procedural information prior to bronchoscopy and 2 often did so (verbal and written 26, verbal only 6, written only 1). Thirty respondents always, 2 often and 1 never explained the indication for performing bronchoscopy. There was a wide variation in individual consenting practices

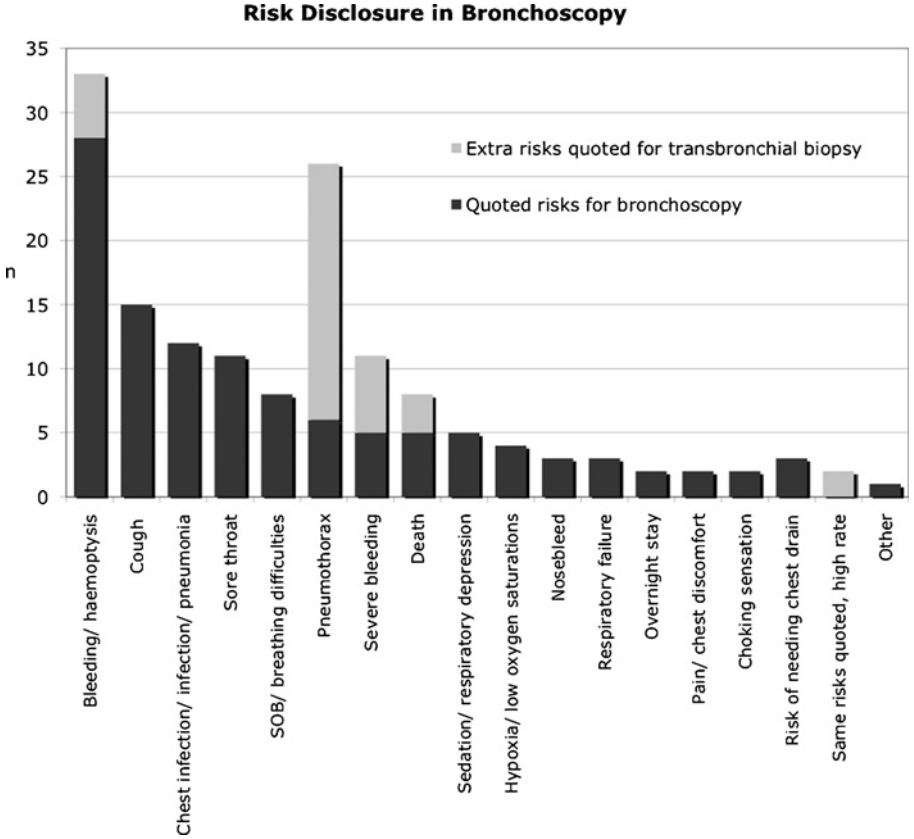

Figure 1 Risk disclosure in bronchoscopy.

with regard to disclosure of serious or frequent risks which was tailored in the majority if the patient underwent transbronchial biopsy (figure 1). Particular risks were represented in terms of an exact percentage in 15 and in 6 by means of words (eg, occasional, common). Fifteen respondents tailored their consent in the presence of significant co-morbidity. Risk of death was communicated always by 3 respondents, often by 1, sometimes by 15 and never by 14.

The current British Thoracic Society guidance on consent for bronchoscopy from 2001[2] reflected the legal and ethical standard at that time where doctors were "entitled to withhold information" if it was thought to be detrimental to patient health and were under "no duty … to point out remote risks". The ethicolegal landscape has been changed dramatically by rulings such as Chester v Afshar,[3] and this is reflected in the recent General Medical Council publication on consent[4] which states that a doctor "must tell patients if an investigation or treatment might result in a serious adverse outcome, even if the likelihood is very small".

We feel that all patients with capacity undergoing bronchoscopy should be offered detailed risk disclosure with documentation of the decision in those patients wishing to "opt out", and that this risk information should become standardised and individually tailored where possible.

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C Echevarria, G A Anwar, S Parker, R M Rutherford

Northumbria Healthcare Trust, Tyne and Wear, UK

Correspondence to Dr R M Rutherford, Northumbria Healthcare Trust, Tyne and Wear, UK; bobrutherford87@yahoo.com

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3. *Chester v Afshar* [2004] 4 All E.R. 587 (H.L.).
4. **Anon.** *Consent: Patients and doctors making decisions together*. General Medical Council, 2008.

Authors' reply

We are grateful to Dr Echevarria and colleagues for their interest in and comments on our study. We accept their criticism that the provision of information sheets only on the day of bronchoscopy and the inclusion for all patients of data on complications relevant only to those likely to undergo transbronchial biopsy may have affected our results. However, apart from the constraints imposed by the artificial environment of a randomised trial, we suspect that these practices are not far removed from what happens in real life. While the verbal discussion of the procedure should always put risks in a personal context, it will be difficult—given the workload involved in developing and agreeing even a single information sheet for a procedure—for any individual centre to develop multiple personally tailored information sheets.

We agree that rulings such as Chester v Afshar have changed the legal requirements for doctors so that even very small risks of serious outcomes such as death need to be discussed.[1] The authors' survey of consent practices for bronchoscopy among respiratory physicians in the north-east of England confirms for bronchoscopy the same startling variations found in the amount of risk disclosure by doctors for other procedures. Standardisation of risk information, even allowing for individual tailoring, would eliminate much of this variation. However, there is an inherent tension in the fact that informed consent does "double duty" as protection both for doctors and for patients. It is worth noting O'Neill's criticism that, while greater emphasis on patient autonomy in medicine is supposed to make doctors more responsible to patients' needs and wishes, it might have the opposite effect of encouraging a culture of back covering.[2] Thus, a legalistic approach to consent may lead to the doctor passing too much information and responsibility onto the patient, "muttering 'caveat emptor' under his breath", as Cowley put it.[3]

S T O'Keeffe, J J Gilmartin

Merlin Park University Hospital, Galway, Ireland

Correspondence to Dr S T O'Keeffe, Merlin Park University Hospital, Galway, Ireland; sokanc@iolfree.ie

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Upper and lower airway microbiology in cystic fibrosis

I thank Mainz *et al* for their recent paper on concordance of upper airway with lower airway microbiology in cystic fibrosis (CF) using nasal lavage.[1] However, their claim that their evidence supports a role for the upper airway in the "acquisition and persistence of opportunistic bacteria" in the lower airway does not stand up to scrutiny. Their work is of great interest and addresses an important and often neglected area of CF research: just how do bacteria gain access to the lower airways? They elegantly demonstrate the close association between the bacteria of the upper and lower airways. It is, however, a cross-sectional study and provides no information as to the direction of transfer of bacteria between the upper and lower airways. In subjects with a lower respiratory tract infection, as is quintessentially the case in CF, it is both general knowledge and scientifically well established that bacteria are expectorated in both sputum and fomites. Such knowledge is the basis of the cough swab or cough plate often used for lower airway microbiological surveillance in the paediatric CF population.[2] It would therefore be natural to assume in any patient with a "colonised" lower airway that fomites from the lower airway will lodge within the nasopharynx. This would result in concordant upper and lower airway bacterial strains, as has been found in this study. However, the evidence provided here does not demonstrate that the upper airway is a source of bacterial seeding to the lower airway, merely that genetically identical strains are found in both compartments and the walls between these compartments are flimsy at best. In order to ascertain the direction of bacterial travel between these two compartments, it would be necessary to undertake a longitudinal study. If such a study were to demonstrate early bacterial travel from the upper to the lower airways, it would open up new avenues of potential therapies for this devastating disease.

T Daniels

Correspondence to Dr T Daniels, Adult CF Unit, Southampton General Hospital, Hampshire SO16 6YD, UK; thomaswvd@hotmail.com

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2. **Cystic Fibrosis Trust**. *Standards for the clinical care of children and adults with cystic fibrosis in the UK*. 2001.

Authors' reply

We thank Dr Daniels for his comments on our paper and agree that "a cross-sectional study … provides no information as to the