

Familial predisposition to snoring

We read with interest the paper by Douglas and coworkers (July 1993;48:719–21) on the possible inheritance of sleep disturbed breathing. Investigating 40 first degree relatives of patients with sleep apnoeas, they found a high prevalence of sleep apnoeas/hypopnoeas (25%), several times higher than the prevalence in a random sample of the British population (5%).¹ The authors concluded that a familial predisposition to sleep apnoeas/hypopnoeas is probable.

If such a predisposition is inheritable, so must be the first stage of the syndrome – that is, habitual snoring.² To test this hypothesis we analysed the results of a questionnaire survey completed in a centre of preventive medicine in north east France. The parents of children aged four to 12 years who attended this centre in August and September 1990 answered a physician administered questionnaire on sleeping habits, and personal and familial medical history. Complete data were obtained from 487 children (51% girls), 30 of whom (6.2%) snored habitually. In univariate analysis habitual snoring was associated with a history of mouth breathing, habitual snoring in a family member, a personal history of allergy, hypermotility during sleep, enuresis, and a history of recurrent "colds". Strong further associations with a history of laboured breathing during sleep, stopping breathing during sleep, and night sweating could not be interpreted as the number of subjects was too limited. When these variables were entered in a multiple logistic regression procedure mouth breathing ($\chi^2=16.9$, $p<0.0001$), snoring by a family member ($\chi^2=5.1$, $p=0.02$), and enuresis ($\chi^2=4.8$, $p=0.03$) emerged as factors independently significantly associated with habitual snoring. In as much as mouth breathing is a clinical correlate of upper airway obstruction and enuresis a consequence of sleep disruption, habitual snoring by a family member appears as the main risk factor for snoring in this study. We believe our results are in favour of a "familial factor" for habitual snoring, as suggested by Douglas *et al.*

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- 1 Stradling JR, Cosby JH. Predictors and prevalence of obstructive sleep apnoea and snoring in 1001 middle aged men. *Thorax* 1991;46:85–90.
- 2 Lugaresi E, Cirignotta F, Gerardi R, Montagna P. Snoring and sleep apnea: natural history of heavy snorers disease. In: Guilleminault C, Partinen M, eds. *Obstructive sleep apnea syndrome*. New York: Raven Press, 1990:25–36.

Formoterol dry powder in asthma

The paper by Dr A Wallin *et al* (June 1993;48:611–4) on the action of formoterol causes some concern in the way the authors

express the duration of bronchodilatation. The method quoted of using the median time with 20% or more of the maximum achieved bronchodilatation means that fairly minor degrees of effect, probably without clinical relevance, are used for the criteria for duration of action. For example, from their fig 2 the baseline FEV₁ would seem to be approximately 1.8 l. The maximum FEV₁ achieved for 24 µg formoterol was approximately 2.2 l, a bronchodilatation of 0.4 l, 20% of which is 0.08 l. Therefore bronchodilatation of from 1.8 l to 1.88 l or 4.4% of baseline was taken to estimate duration of action. This compares with a bronchodilatation of 0.27 l if the criterion of a 15% improvement from baseline is used. If this later value is employed then formoterol only produces bronchodilatation for approximately 4–5 hours, which may not be statistically different from salbutamol.

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AUTHORS' REPLY We are grateful to Dr Bright for his comments and calculations on the duration of bronchodilatation. If you use the definition time with FEV₁ > 15% over baseline we have no disagreement with him. However, as mentioned in the discussion we did not use this definition because of the moderate reversibility in our data with patients on inhaled steroids. The mean reversibility of FEV₁ was 23%, the median 19%, and the lowest 15%. If you use the definition of duration as time with FEV₁ > 15% over baseline, you cannot usefully express duration if the patient's maximum bronchodilatation was 15%. Even with a median reversibility of 19% the duration calculated in this way remains misleading. Another problem with defining duration based on change of FEV₁ in relation to baseline is the natural variability of lung function in asthma.

In fig 3 in our paper the FEV₁ in the salbutamol and placebo groups declined spontaneously. For those treated with placebo the decline was from 1.8 l (baseline) to 1.59 l at 12 hours. If we take formoterol 24 µg as an example the FEV₁ was 1.88 l 12 hours after dosing, which was 18% over the placebo baseline (1.59 l) at that time. For formoterol 12 µg at 12 hours the corresponding value was 11%. When deciding if a drug has any clinical effect one should consider the natural variation of asthma. In this study we have done that by calculating duration as time with >20% of maximum achieved bronchodilatation. This method of measuring duration of action on inhaled long acting β₂ agonists has been advocated by Arvidsson *et al* and has recently been described in detail elsewhere.¹

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- 1 Arvidsson P, Larsson S, Löfdahl C-G. Objective and subjective bronchodilatation over 12 hours after inhaled formoterol; individual responses. *J Asthma* 1993;30:459–65.

BOOK NOTICES

Cystic Fibrosis. PB Davis. (Pp 560; \$185.00). New York: Marcel Dekker, 1993. 0 8247 8815 X.

A year ago this reviewer was bemoaning the dearth of books published on cystic fibrosis. Since that time four books on this subject have been sent to me for consideration. The fourth of these books is the biggest at 550 pages and is the most expensive. It is multiauthored by American experts but, unusually, the in-depth literature review has embraced a considerable amount of work done outside North America.

The book is essentially descriptive and consists of 13 chapters. The first two chapters are devoted to genetics and scientific advances. The information is set out concisely, clearly, and is easy to read. The remaining chapters largely consider cystic fibrosis as a disease of different organ systems and the treatment thereof. Several unusual aspects are considered and are discussed in depth – for example, the chapter on drug disposition in cystic fibrosis is essential reading as intravenous antibiotic therapy is the cornerstone of treatment, and recognition of adulthood is considered in a chapter entitled "Cystic fibrosis and the reproductive system" which even considers the advantages and disadvantages of breast feeding in cystic fibrosis. More conventional subjects considered are infection and inflammation of the lung in cystic fibrosis, which deals in considerable length with host responses. It is an illustration of how far we have to go before we can understand with clarity the complex interacting cellular and humoral responses. Chapters on the treatment and complications of pulmonary disease are dealt with in considerable depth and provide an extremely useful reference source for all cystic fibrosis clinicians.

The strength of the book lies in the diversity and depth with which it deals with the overall subject of cystic fibrosis. Very little is left out. There are, however, some omissions – for example, only half a page is given to the role of exercise and very little consideration is given to the patient as a person and the difficulties in complying with the complex process of self care. One considerable virtue of the book is its author index which consists of 52 pages and references major and minor publications over the last 20 years. It is invaluable for checking references and for writing articles. The few weaknesses lie in its poverty stricken subject index which consists of only five pages, and the overlap of the subject matter in individual chapters – one reference appears repeatedly in six different chapters. However, these are trivial criticisms when the book is considered overall. This is undoubtedly the most clinically useful book presently available on the subject of cystic fibrosis. It is expensive but well worth the cost, and is an essential purchase for every paediatric and adult physician who has an interest in cystic fibrosis and needs to deal with the complexity and pathogenesis of the disease. It is unlikely to be outdated because of its clinical content and should be on every library shelf. – AKW