LETTERS TO THE EDITOR

Cough – but is it asthma?

EDITOR,—Although the scientific content of Sheila McKenzie's recent review is impressive,¹ I do not agree with her conclusions. There is a group of children that one often sees in clinic, usually preschool children who have had a chronic persistent nocturnal cough that may wake them three or four times a night. Although there may be a personal history of eczema and a family history of atopy in general, there is often nothing to suggest personal wheeze. In view of their age it is often difficult to demonstrate airways lability scientifically. Dr McKenzie's approach seems to offer these children and their parents little except tea and sympathy.

It used to be my practice to offer this sort of child a trial of β agonists. It had a success rate of approximately zero. I have rationalised this that cough is due to airways inflammation, mucous, and oedema and therefore not likely to respond to bronchodilators. It is also likely that the time course of action of short acting β agonists make them ineffective at the most vulnerable time of the night.

My current practice is to start such children, after a very thorough history and examination, on a six week course of inhaled steroids. In my experience the success of such practice is something like 80% and although it may upset the scientific purists it certainly pleases the parents.

Clearly more research is required to elucidate this problem. I feel that in the absence of wheeze, however, that tests based on bronchial hyper-reactivity are not likely to be useful. Histological evidence of airways inflammation from direct bronchoscopy would be interesting but currently there is so little known about the early changes in childhood asthma that such an approach at present would not produce useful results. A double blind randomised trial of the use of inhaled steroids in this situation would be one way forward and the measurement of breath nitric oxide to help clarify the degree of airway inflammation would also be one interesting approach for the future.²

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- 1 McKenzie S. Cough – but is it asthma? *Arch Dis Child* 1994; 70: 1–2.
- 2 Kharitonov SA, Yates D, Robbins RA, Logan Sinclair R, Shinebourne EA, Barnes PJ. Increased nitric oxide in exhaled air of asthmatic patients. *Lancet* 1994; 343: 133–5.

Dr McKenzie comments:

Antibiotics used to be prescribed for cough and sometimes still are. Parents often claim their benefit, as Dr Upton's parents do for steroids. Anecdote is no substitute for a good clinical trial.

Parents, of course, are pleased to be given a prescription and doctors are often delighted to write one, but sometimes parents are better pleased with an explanation. There is no

proof that any medication is better than no medication for cough. I have to admit that my own practice of a two week trial of β agonists is far from satisfactory.

I agree with Dr Upton that treatment of cough with inhaled steroids needs proper evaluation. There is not much at present to support their empirical use. My main concern is that too many children could be labelled asthmatic because their cough improves while they are taking steroids. This could be purely coincidental or it could be that steroids affect non-specific cough by a mechanism different from that in asthma. I suspect the understanding and treatment of cough in children is likely to be one of the growth industries of the next decade.

Ethnicity and the sudden infant death syndrome: important clues from anthropology

EDITOR,—Davies and Gantley question a number of entrenched views on infant care practices.¹ The welcome fall in sudden infant death syndrome rates which has accompanied the reduction in the prone sleeping position of infants in several countries after 'risk reduction' campaigns suggests that apparently small changes in infant care practices may have profound effects upon infant well being and survival.

At birth the human infant is the least neurologically mature primate, with the longest and most intense postnatal dependence upon the mother. Infant monkeys separated from their mothers for brief periods experience adverse physiological consequences, including increased adrenocorticotrophic hormone concentrations, reduced body temperature, cardiac arrhythmias, and compromise of the immune system.²

In the home environment, mothers are able to achieve a thermoneutral environment for their sleeping infants with remarkable accuracy.³ Studies of mothers and their 3 month old infants sleeping either in separate rooms or in the same bed ('co-sleeping') within a sleep laboratory on successive nights have shown the complexity of the mother-infant interactions during sleep.⁴ Co-sleeping mothers and infants continually induce mutual small arousals, the infants breast feed more frequently than when sleeping separately, and mothers actively monitor and regulate their infant's thermal environment. The infants' inspired air commonly contains 0.5–1% carbon dioxide from the mother when co-sleeping. Co-sleeping babies spend less time in the deeper stages of sleep. Co-sleeping mothers almost always place their infants next to them in a supine position, close to their nipples, so that the infants can feed. The supine position is the only one in which infant manipulation, control, and breast feeding is facilitated.⁵

Infant sleep, breathing, arousal, and thermoregulation all evolved in the context of continuous parental contact, and no evidence has been produced on the benefits of solitary sleeping arrangements.

Western values favour early autonomy and individualism and researchers inadvertently may have overemphasised the infant's physiological independence from its caregivers, confusing the infant's preparedness to adapt with actual adaptation.⁶

Anthropological studies of parents and infants provide the basis for postulating that for some, possibly small subclass of poten-

tially vulnerable infants, mother-infant contact throughout the night may be protective. Human evolutionary studies, cross cultural data on human behaviour, and studies of mother-infant interactions can give important insight into normal human development.

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- 1 Davies DP, Gantley M. Ethnicity and the aetiology of sudden infant death syndrome. *Arch Dis Child* 1994; 70: 349–53.
- 2 McKenna JJ. An anthropological perspective on the sudden infant death syndrome. The role of parental breathing cues and speech breathing adaptations. *Med Anthropol* 1986; 10: 9–53.
- 3 Wigfield RE, Fleming PJ, Azaz Y, et al. How much bedding do babies need at night? *Arch Dis Child* 1993; 69: 181–6.
- 4 McKenna JJ, Thomson E, Anders TF, et al. Infant-parent co-sleeping in an evolutionary perspective: implications for understanding infant sleep development and the sudden infant death syndrome. *Sleep* 1993; 16: 263–82.
- 5 Mosko S, McKenna JJ, Dickel M, Hunt L. Parent-infant co-sleeping: the appropriate context for the study of infant sleep and implications for sudden infant death syndrome. *Research Journal of Behavioural Medicine* 1993; 16: 589–610.
- 6 McKenna JJ, Mosko S. Evolution and infant sleep: an experimental study of infant-parent co-sleeping and its implications for SIDS. *Acta Paediatr* 1993; 389 (suppl): 31–6.

Leucocyte adhesion deficiency syndromes

EDITOR,—El Habbal and S Strobel recently gave an interesting overview of what is known about the leucocyte adhesion deficiency (LAD) syndrome and the molecular basis of adhesive events that occur in the immune response.¹

I would like to point out some details that need clarification. As the authors state the adhesion molecules can be subdivided into three superfamilies: the integrins, the selectins, and the members of the immunoglobulin superfamily. The tables of the review listing the adhesion molecules, their tissue distribution, and the supposed ligands contain a number of errors which might be misleading for the understanding of the regulation of adhesive interactions.

Members of the integrin family of adhesion molecules are heterodimers which are formed by the association of an α chain with a β chain. Neither VLA-4 ($\alpha\beta 1$) nor the $\beta 6$ integrins are identical with LPAM-1, the lymphocyte Peyer's patch adhesion molecule-1. LPAM-1 ($\alpha\beta 7$) shares the $\alpha 4$ chain with VLA-4, however, the β chain is different ($\beta 7$ versus $\beta 1$ or $\beta 6$) and therefore LPAM-1 belongs to the $\beta 7$ integrins.² This is critical for the regulation of lymphocyte homing because LPAM-1 specifically mediates the binding of lymphocytes to the mucosa associated lymphoid tissue of the gut. LPAM-1 is expressed on T as well as on B lymphocytes and the ligand is not CD28 (here the $\beta 7$ integrin has been mixed up with B7, one of the most important costimulatory molecules expressed on B lymphocytes and binding to CD28 and CTLA-4 on T cells). In contrast, the ligands so far identified for LPAM-1 are: MAdCAM-1, VCAM-1, and the extracellular matrix protein fibronectin.³ VLA-4, however, is identical with LPAM-2, the lymphocyte Peyer's patch adhesion molecule-2, which has erroneously been listed as being VLA-2.