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 long-term, 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 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. In contrast, a child trial of β agonists and the reponse may demonstrate a sort of child a trial of β agonists. It has a success rate of approximately zero. I have rationalised that this cough is due to airways inflammation, mucus, 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 on 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 not be the scientific purist it certainly please 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 child-hood 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 and to help clarify the degree of cough 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: 450-3.

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. Anecdot 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 on 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 bronchial reactivity in 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.1 Their welcome fall in infant death syndrome has shown the complexity 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 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 adrenocortico- trophic 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 has accompanied the 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 0·5–1·1% 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 potentially vulnerable infants, mother-infant contact throughout the night may be protective. Human evolution studies of infants, animal data on human behaviour, and studies of mother-infant interactions can give important insight into normal human development.

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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. Firstly, 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 classification of these superfamilies, 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 (α4β1) nor the β6 integrins are identical with LPAM-1, the lymphocyte Peyer’s patch adhesion molecule-1. LPAM-1 (α4β7) shares the α4 chain with VLA-4, however, the β chain is different (β7 versus β1 or β6) and therefore LPAM-1 belongs to the β7 integrins. This is critical for the regulation of lymphocyte homing, the role of LPAM-1 specifically mediates the binding of lymphocytes to the mucosa associated lymphoid tissue of the gut. LPAM-1 has expressed on T cells as well as on B lymphocytes and binding to CD44 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. However, it is identified with LPAM-1 that Peyer’s patch adhesion molecule-2, which has erroneously been listed as being VLA-2.