

Minor illness in infants — still a major worry for parents?

WIDESPREAD and understandable anxiety may have been caused by a recent television programme (*The Cook report*, Central Television, 17 November 1994) in which cot mattress materials were implicated in the sudden infant death syndrome (SIDS). Although this particular controversy remains to be resolved^{1,2} it has again brought the spectre of sudden infant death syndrome to centre stage. There has, nonetheless, been a gratifying decline in deaths from sudden infant death syndrome, which mostly affects babies between the ages of two and six months, from a peak in the United Kingdom of about 1.8 females and 2.7 males per 1000 live births in the late 1980s to well under half that figure in 1992.³ This improvement has been attributed to the campaign against the prone sleeping position for babies.⁴⁻⁸ It is still however a cruel lottery as sudden infant death syndrome strikes silently and indiscriminately, affecting apparently healthy babies. Sudden infant death syndrome cannot reliably be prevented because the cause(s) remain largely undefined, although there may be some measures that are prudent to take. Small wonder that many parents of young babies feel insecure and apprehensive, particularly when the manifestation of illness in this age group is so limited in range of expression.

This is an emotive subject, particularly if there might be some way in which death could be prevented if parents or doctors were alert enough to recognize predisposing symptoms. As Holme describes in this issue of the *Journal*,⁹ while links with supposed presaging symptoms such as fast or noisy breathing, poor feeding, drowsiness and irritability have been suggested,¹⁰ such a possibility seems unlikely.

Although not specifically a study of sudden infant death syndrome, the importance of Holme's study is that data on the natural history of these and other symptoms were collected prospectively from a sample of babies from a general healthy population. Previous general practice studies have been retrospective or only of unwell infants presenting for medical consultation. There were no deaths from sudden infant death syndrome in Holme's sample: an impracticably large study of this kind would be required to capture sufficient deaths from sudden infant death syndrome to study possible presaging symptoms in a definitive way. It is for this reason that most studies have depended on case-control methods. Although case-control comparisons are currently the only feasible method for studying such rare occurrences, there is increasing recognition that the method is fraught with possible unidentified bias, primarily because the study and control groups necessarily come from different samples (rather than being randomized from the same sample as in 'gold standard' clinical trial design).¹¹ Holme is therefore right to say that his results provide the context in which these issues should be re-examined. Specificity refers to the ability of a test to detect a target disorder, in this case the ability of any of the given symptoms to predict sudden infant death syndrome. Holme's study leads to the inevitable conclusion that the symptoms involved are so common and the target event (sudden infant death syndrome) so rare that specificity is bound to be negligible.

The earlier approach to the epidemiology of sudden infant death syndrome is typified by the conclusion of a brief review of research in a standard text of 15 years ago:

'The incidence of these deaths is highest in socially deprived families characterized by low parental intelligence,

poor maternal efficiency and a multiplicity of problems — illegitimacy, cohabitation or a tense marital situation. In this group too there is evidence of failure by the parents to recognize serious illness in their baby.'¹²

These earlier studies may have identified groups of children that were at higher risk and they may have helped to generate theories about the possible pathophysiology of sudden infant death syndrome, but both parents and doctors had little or no control over these factors in individual cases, leaving an unhelpful sense of hopelessness. In fact, recent evidence suggests that these broad epidemiological 'risk factors' are not associated with true sudden infant death syndrome, where no detectable pathology is found, but with sudden explicable or possibly explicable deaths which have been misclassified according to the strict definition of sudden infant death syndrome.¹³⁻¹⁵

More recently the emphasis has been on environmental factors and child care practices. The prone sleeping position, overheating and maternal tobacco smoking (both pre- and post-natally) have all been implicated. As Fleming points out the importance is that these factors are amenable to change.¹⁶ Although the cause(s) of sudden infant death syndrome remain to be fully elucidated, this more pragmatic approach may have resulted in a major reduction in rates of sudden infant death syndrome in a number of countries.^{8,17,18}

For day to day general practice, the importance of Holme's study perhaps lies in his unique description of the natural course of these symptoms in previously healthy babies. Although he shows that in the vast majority of cases there was, in retrospect, no cause for alarm we should take this as reinforcing, not lessening, the need for vigilance. The main difference between children, particularly babies, and adults is the rapidity with which an apparently healthy child can deteriorate through initially minor illness to, albeit rarely, become dangerously ill. The limited range of symptoms and signs which can be detected in babies adds to the difficulty.

Nonetheless, Holme also shows that parents managed 67% to 99% of these episodes themselves and often delayed seeking medical advice for four or five days although behavioural changes (such as unusual drowsiness) and fever led to more urgent consultation. As general practitioners, we may think that many consultations, for babies in particular, could in retrospect be classified as minor or unnecessary but a high screening burden is necessary to maximize the detection of rare events of serious illness. If doctors find it difficult to differentiate minor from serious causes of a common and limited range of symptoms, how much more difficult must it be for parents?

ROSS J TAYLOR

Senior lecturer in general practice, University of Aberdeen

References

1. Fleming PJ, Cooke M, Chantler SM, Golding J. Fire retardants, biocides, plasticisers and sudden infant deaths [editorial]. *BMJ* 1994; **309**: 1594-1595.
2. Morgan B. Cot death TV probe sparks row [news item]. *The Times Higher Education Supplement* 1995; 6 January: 3.
3. Staples B, Pharoah POD. Child health statistical review. *Arch Dis Child* 1994; **71**: 548-554.

4. Davies DP. Cot death in Hong Kong: a rare problem? *Lancet* 1985; **2**: 1346-1349.
5. Englebarts AC, de Jonge GA. Choice of sleeping position for infants: possible association with cot death. *Arch Dis Child* 1990; **65**: 462-467.
6. Dwyer T, Ponsonby AL, Newman NM, Gibbons LE. Prospective cohort study of prone sleeping position and sudden infant death syndrome. *Lancet* 1991; **337**: 1244-1247.
7. Bolton DP, Taylor BJ, Campbell AJ, *et al*. Rebreathing expired gases from bedding: a cause of cot death. *Arch Dis Child* 1993; **69**: 187-190.
8. Court C, Roberts J, Essex C, *et al*. Cot deaths [news item]. *BMJ* 1995; **310**: 7-10.
9. Holme CO. Incidence and prevalence of non-specific symptoms and behavioural changes in infants under the age of two years. *Br J Gen Pract* 1995; **45**: 65-69.
10. Valman B. Preventing infant deaths [editorial]. *BMJ* 1985; **290**: 339-340.
11. Sackett DL, Haynes BR, Guyatt GH, Tugwell P. *Clinical epidemiology*. Boston, MA: Little, Brown and Company, 1991.
12. Richards IDG. The epidemiology of disease in childhood. In: Mitchell RG (ed). *Child health in the community*. Second edition. Edinburgh: Churchill Livingstone, 1980.
13. Bartholomew SE, Macarthur BA, Bain AD. Sudden infant death syndrome in south east Scotland. *Arch Dis Child* 1987; **62**: 951-956.
14. Taylor EM, Emery JL. Categories of preventable unexpected infant deaths. *Arch Dis Child* 1990; **65**: 535-539.
15. Haas JE, Taylor JA, Bergman AB, *et al*. Relationship between epidemiologic risk factors and clinicopathologic findings in the sudden infant death syndrome. *Pediatrics* 1993; **91**: 106-112.
16. Fleming PJ. Understanding and preventing sudden infant death syndrome. *Curr Opin Pediatr* 1994; **6**: 158-162.
17. de Jonge GA, Burgmeijer RJ, Englebarts AC, *et al*. Sleeping position for infants and cot death in the Netherlands 1985-91. *Arch Dis Child* 1993; **69**: 660-663.
18. Mitchell EA, Brunt JM, Everard C. Reduction in mortality from sudden infant death syndrome in New Zealand 1986-92. *Arch Dis Child* 1994; **70**: 291-294.

Address for correspondence

Dr R J Taylor, Department of General Practice, University of Aberdeen, Foresterhill Health Centre, Westburn Road, Aberdeen AB9 2AY.

Through audit to quality: the future of clinical audit in primary care

OVER the last year, the primary health care clinical audit working group of the Clinical Outcomes Group has been considering the future of clinical audit in primary care, under the chairmanship of Sir Donald Irvine. The actual remit for the working group was to 'explore the development of clinical audit in primary health care and to make recommendations' regarding ways in which audit can be developed in primary care, methods to involve management in audit within primary health care teams, and use of clinical audit as a tool for quality improvement in primary health care.

The group has now published its deliberations.¹ Although the paper is not yet a National Health Service Executive policy document, it clearly reflects some aspects of central thinking. The original health circular,² which introduced medical audit advisory groups, has been extended until 31 March 1996 according to a letter of 16 May 1994 from Dr Rosemary Field at the NHS Executive headquarters. The suggestions in this report to the Clinical Outcomes Group are likely to determine future arrangements for clinical audit in primary care in England. Staff from the Welsh Office have been taking an interest in the proposals and, although audit is organized through the clinical resource and audit group in Scotland at present, Scottish Office thinking may well be influenced also. However, the report contains assumptions and implications concerning the whole structure and funding of primary care far beyond the institution of universal clinical audit as an integral part of professional good practice. There is no denying the attraction of its vision of high quality care assured through peer review, explicit standards (especially in the development and implementation of appropriate clinical guidelines) and self-critical awareness.

There are several themes underlying the direction of the paper which lead to unresolved tensions and inconsistencies. It is clear that the whole concept of primary care embraces more than just general practice but there is no attempt to reconcile the differences in provision of care from independent contractors and managed units. The bald statement that 'it is in principle desirable to incorporate audit agreements directly into a contract with each practice unit' presupposes practice based contracts in

advance of professional negotiations and a change from current General Medical Services remuneration. The onus is to be placed on joint commissioning authorities to make explicit the link between audit and service development and to 'bring a managerial dimension to a professionally-led process'. The paper is keen to emphasize local solutions and initiatives and to avoid a single imposed audit infrastructure for primary care. At the same time, there is a lack of guidance within which to conduct local negotiations in order to produce a coherent strategy for quality assurance within the NHS. It may prove difficult to reconcile long-term investment in quality with immediate demands for efficiency savings and value for money.

Although the preface to the report advocates an evolutionary approach from medical to clinical audit to involve the whole primary care team, the most significant recommendation is the abolition of medical audit advisory groups, as presently constituted. This is an example of the discontinuous change which is currently militating against self-development in the NHS and leading to individual initiative being subsumed in reacting to externally imposed change.

It is clear that we have moved on from medical to clinical audit and that the original purpose of medical audit advisory groups to advise family health services authorities, educate practitioners and promote the institution of regular and systematic medical audit is due for review. The report pays tribute to the success of most medical audit advisory groups to date but fails to appreciate their recent development to embrace clinical audit. The extent of their activities has been well evaluated by Humphrey and Berrow.³ Johnson demonstrated the difficulties in quantifying improvements in care as a result of a medical audit advisory group programme in such a short timescale.⁴ By March 1993, most groups had adopted the attitude that general practice audit involved the whole practice team⁵ and over a third of medical audit advisory groups had already expanded their membership to include practice managers and nurses (Birmingham Medical Audit Advisory Group national MAAG survey 1992-93). In December 1993, the National Primary Care Audit Group recommended that MAAG should now stand for 'multidisciplin-