

reasons. Two of the authors' cases (cases 1 and 3) were not managed in accordance with the guidelines, and in case 2 negative results of tuberculin tests six weeks apart meant that no chest x ray film was obtained, and primary tuberculosis was diagnosed shortly afterwards.

The 1994 code of practice superseded that of 1990, and the recommendations with regard to contact tracing were based on five contact studies (referenced in the code²) of a total of 22 971 contacts, including many children. These studies showed that contacts found to have clinical tuberculosis are largely unvaccinated close contacts of people with sputum smear positive disease and that detection usually occurs at the initial visit. Because of possible delays in tuberculin conversion, in children it is advised that the tuberculin test is repeated after six weeks if the initial test gives a negative result and that children under the age of 2 years are given prophylaxis with isoniazid pending the result of this repeat test.

Chest radiography regardless of the result of the first tuberculin test is also not justified for two reasons. Firstly, there is little evidence that non-pulmonary tuberculosis is infectious to others, including children. Secondly, unnecessary radiation is to be avoided in children. Many children could well have radiography for which there is insufficient justification.

The 1994 code of practice will be audited and its effectiveness monitored. However, as the recommendations were based on large studies from five separate centres, none of which found the problems reported by Clark and Cant, more would be required than evidence from a single case before any change to strongly evidence based recommendations could be justified. In view of the unfortunate delay in reporting the history of contact with tuberculosis in case 2 it might be sensible to advise parents of children in whom tuberculin tests persistently give negative results of the small possibility of subsequent tuberculosis and of the need to report relevant symptoms and the history of exposure.

For the Joint Tuberculosis Committee of the British Thoracic Society

PETER ORMEROD
Consultant chest physician

Blackburn Royal Infirmary,
Blackburn BBE 3LR

JOHN WATSON
Consultant epidemiologist

PHLS Communicable Disease Surveillance Centre,
London NW5 5EQ

CRAIG SKINNER
Consultant chest physician

Birmingham Heartlands Hospital,
Birmingham B9 5SS

1 Clark JE, Cant AJ. Pitfalls in contact tracing and early diagnosis of childhood tuberculosis. *BMJ* 1996;313:221-2. (27 July.)

2 Joint Tuberculosis Committee of the British Thoracic Society. Control and prevention of tuberculosis in the United Kingdom: code of practice 1994. *Thorax* 1994;49:1193-200.

Sudden infant death syndrome

More attention should have been paid to socioeconomic factors

EDITOR.—Chris Mihill and colleagues were right to complain¹ that the breaking of the embargo on the report on the confidential inquiry into stillbirths and deaths in infancy² and the accompanying papers in the *BMJ*^{3,4} meant that there was inadequate time for the full data in the report to be studied. Given more time, journalists might have noticed major differences between the socioeconomic and other circumstances of babies whose death was attributed to the sudden infant death syndrome and control babies. These were not mentioned in the Department of Health's press release and were

Table 1—Comparison of babies whose deaths were classified as being due to the sudden infant death syndrome and control babies from same health visitors' lists (source: ref 2). Figures are percentages of families except where stated otherwise

	Sudden infant death syndrome	Controls
Employment and income		
Neither parent employed	44.6	15.4
Neither parent had ever been employed	13.3	4.1
Family income:		
<£100 a week	41.0	20.4
<£200 a week	72.8	47.8
Receiving income support	66.6	28.2
No of families in sample	195	780
Education		
No educational qualifications:		
Mother	40.5	19.0
Partner	33.7	18.7
Parent with highest qualifications	24.3	11.0
A level or above:		
Mother	12.5	25.0
Partner	16.2	30.9
Parent with highest qualifications	19.7	37.7
No of families in sample:		
Mother	193	775
Partner	166	699
Parent with highest qualifications	193	776
Housing		
≥2 people per room	10.8	0.8
No of families in sample	194	778
≥6 people in household	21.6	9.9
No of families in sample	195	780
Damp or mould in baby's room	13.5	7.2
No of families in sample	193	778
Babies*		
Born before 37 weeks' gestation	18.0	4.3
Birth weight (g):		
<1500	3.6	0.6
<2500	21.1	4.2
No of babies in sample	195	778

*Figures are percentages of babies.

mentioned only in passing in the report's executive summary and the papers.

The two papers restricted their focus to the sleeping environment³ and to smoking and the use of alcohol and illegal drugs.⁴ The first paper mentioned that the "effect modifiers" included several markers of socioeconomic status that differed between the cases and controls and that the differences may underestimate the differences in the population as a whole because of the way in which the controls were selected. Data to show the extent of these major differences were published only in the report of the confidential inquiry.² Table 1 shows that many of the households in which sudden infant deaths occurred were in the more deprived sectors of society.

The authors' multivariate analyses showed differences in environmental and behavioural factors after adjustment for these social factors but did not explore possible associations further. Thus they did not question whether the strengths of the associations between the sudden infant death syndrome and risk factors varied between social groups. A recent study in Victoria, Australia, compared groups, defined by ethnicity and place of birth, that had different rates of the syndrome. It did not find the expected associations with rates of prone sleeping, bed sharing, breast feeding, and smoking by family members other than the mother or a straightforward association with smoking by mothers.⁵

Account needs to be taken of social and economic factors when possible strategies for prevention are explored. To dismiss the factors

by using the statistical term "effect modifier" is likely to understate their impact on people's lives, the problems that people may encounter in caring for a new baby, and the barriers to responding to advice about the hazards of smoking, alcohol, and illegal drugs.

The authors state that it is a good idea to avoid smoking. Should they have added that it is also a good idea to avoid being born into a poor household? Whatever the answers, the questions should have been publicly aired. The breaking of the press embargo ensured that this did not happen.

ALISON MACFARLANE
Medical statistician

National Perinatal Epidemiology Unit,
Radcliffe Infirmary,
Oxford OX2 6HE

- Mihill C, Pope N, Hunt L, Cooper G, Fletcher D. *BMJ's* embargo should not be broken. *BMJ* 1996;313:305. (3 August.)
- National Advisory Body for CESDI. *Annual report 1994*. London: Department of Health, 1996.
- Fleming PJ, Blair PS, Bacon C, Bensley D, Smith I, Taylor E, et al. Environment of infants during sleep and risk of the sudden infant death syndrome: results of the 1993-5 case-control study for confidential inquiry into stillbirths and deaths in infancy. *BMJ* 1996;313:191-5. (27 July.)
- Blair PS, Fleming PJ, Bensley D, Smith I, Bacon C, Taylor E, et al. Smoking and the sudden infant death syndrome: results from the 1993-5 case-control study for confidential inquiry into stillbirths and deaths in infancy. *BMJ* 1996;313:195-8. (27 July.)
- Potter A, Lumley J, Watson L. The 'new' risk factors for SIDS: is there an association with the ethnic and place of birth differences in incidence in Victoria, Australia? *Early Hum Dev* 1996;45:119-31.

Smoking is part of a causal chain

EDITOR.—Peter S Blair and colleagues suggest that over 60% of cases of the sudden infant death syndrome may be attributable to the effects of parental smoking.¹ This depends on the assumption that the association described is causal.

While smoking is undoubtedly harmful to babies, the magnitude of the risk is less clear. The close correlation between adverse socioeconomic circumstances and smoking and between risk of the sudden infant death syndrome and deprivation requires that the analysis should take careful account of potential confounding. The importance of the association between the syndrome and deprivation is suggested by the univariate odds ratios associated with social factors quoted in the full report of this study²—for example, receipt of family income supplement, 6.27; income of <£200/week, 3.57; living in rented accommodation, 3.81; and <0.5 rooms per person, 31.3. To avoid residual confounding, the measure of socioeconomic status used for adjustment in multivariate analyses needs to split the population into relatively homogeneous bands. To adjust for low socioeconomic status the authors seem to use receipt of family income supplement, dividing the population into two heterogeneous groups.³ Blackburn and Graham have shown, however, that even among women in receipt of income support (a more homogeneous group) the risk of smoking during pregnancy is strongly related to the degree of deprivation.⁴ This raises the potential for appreciable residual confounding.

While acknowledging that social variables remain significant after adjustment, the authors have chosen to concentrate on behavioural variables on the grounds that social variables are "not amenable to change." Between 1979 and 1987 the proportion of British children in families with less than half the mean household income increased from 12% to 26%.⁵ This suggests that social factors are amenable to change over quite short periods.

Smoking is harmful, but to lay responsibility for deaths due to the sudden infant death