1974, data for 1970-3 inclusive were taken from table 5, using the same codes. Although I was told that the data from table 5 were comparable to those from table 21 (ii) because the codes were identical, in fact they are not. Table 5 deals with all cases of specified injury whether accidental or undetermined. The undetermined group may in fact comprise accidental poisoning or intentional poisoning, or homicide by poisoning. Tables 5 and 21 (ii) are not comparable, so I have rechecked the data from table 5 only from 1970 to 1978 inclusively. My final graph is identical to that of Dr Jackson's above.

I therefore conclude that my supposition and conclusions drawn in my letter of 17 September were incorrect. In the light of the new graph as shown by Dr Jackson above I conclude that the child resistant containers have had an appreciable and significant effect on accidental poisoning since their introduction in 1976. I therefore support all the points made in their original paper (2 July, p 15).

My apologies.

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Family and community factors associated with infant deaths that might be preventable

SIR,—In the follow up to their recent paper in *Archives of Disease in Childhood*¹ Dr Elizabeth M Taylor and Professor John L Emery (24 September, p 871) have attempted to apply scientific method to investigating a possible relation between psychosocial factors and unexpected infant deaths. The study of this relation is important but exceptionally difficult as assigning retrospective scores to highly subjective factors is considerably different in infants who have died compared with surviving controls. Though it might have been possible to code data from deaths and controls and to subject the results to an appropriate panel of experts to score "blind" the risk of death, this vital procedure was not adopted. Moreover, the lack of comparability between results on deaths and on controls has been accentuated by unequal treatment of the two experimental groups in the simple matter of data collection. Thus in the group who died obstetric, paediatric, and casualty records, together with questionnaires from the family doctor, health visitor, midwife, and social worker, were examined at a case conference. In the control group the only data collected resulted from telephone calls to the infants' health visitors; even this information was not obtained in 28 of 130 cases (22%).

The principal conclusion of this study was that adverse psychosocial factors influenced the incidence of unexpected deaths from treatable disease. The fact that infant mortality increases with a decreasing ability to treat illness in a community is common knowledge, but the term "treatable" requires clarification. Most infants during their first year of life have one or more minor illnesses, but rarely die. Up to half the cases of sudden infant death syndrome have been reported to have minor illness in the week preceding death.² The problem is that, given these two observations, most pathologists have no way of knowing whether by treating minor disease more aggressively an unexpected death would have been prevented. The report by Dr Taylor and Professor Emery does not advance our know-

ledge in this direction because details of illness in the control group were not reported.

Finally, I must again take issue with the "gently battered" hypothesis which is offered to account for 10% of unexpected deaths in this study; their evidence for this suggestion (see previous correspondence³) is so subjective that Dr Taylor and Professor Emery should be taken to task over what, in my opinion, amounts to a "defamation of character" on the families involved.

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¹ Taylor EM, Emery JL. Two-year study of the causes of post-perinatal deaths classified in terms of preventability. *Arch Dis Child* 1982;57:668-73.

² Valdes-Dapena MA. Sudden infant death syndrome: a review of the medical literature 1974-1979. *Pediatrics* 1980;66:597-614.

³ Southall DP. Two year study of the causes of post-perinatal deaths classified in terms of preventability. *Arch Dis Child* 1983;58:75.

* * * We sent a copy of this letter to the authors, who reply below.—Ed, *BMJ*.

SIR,—The points raised by Dr David Southall are important because they illustrate different approaches. We are concerned with all causes of child death, but in particular the type of deaths that occur more frequently in Britain than in Sweden or Finland. Some of these deaths present as "unexpected" deaths and are registered as cot deaths.

We share Dr Southall's problem over the difficulties in obtaining control history—no control can have been subjected to the stress of a child death. He rightly points out that we have recorded information on only 102 of the 130 controls originally selected. We accepted information only from health visitors who had personal knowledge of the family at the relevant time and had access to full records of their contacts with the family. These criteria were not fulfilled for 28 cases. The data can under no circumstances be as full for controls as for cases; they were always based on personal knowledge and adequate records.

We disagree with him completely on our handling of our material. We are attempting to get deeper than the clinical recorded data—thus confidential information given over the telephone and across the room among the health visitor, general practitioner, social worker and ourselves in the family doctor's office is never going to be available for submission to an outside panel, and perhaps should not be.

Dr Southall mentions sudden infant death syndrome—we did not, or even attempt to discuss cot deaths as such. It is here that our approach differs most from his. We feel that the maintenance of sudden infant death syndrome as a mystical entity for which "the cause" needs to be sought, as for the Holy Grail, is now counterproductive for research. We accept completely (and continually study) many unexpected child deaths that are in our hands completely unexplained, but it would be irresponsible not to attempt to do something about the greater number where the possibility of modifying some circumstances exists.

We are sorry that David Southall thought it necessary to quote again a figure from a newspaper of possible infanticide among unexpected child deaths. In our paper we reported on our findings in one locality over a period of two years—we did not extrapolate from this, and do not think that appropriate. It almost

seems that he wishes to establish a figure to rub salt into the wounds of the very people whom he claims to champion.

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Legionella infection in Scotland 1981-2

SIR,—To complement the account of legionella infection in England and Wales in 1981 and 1982 presented by the Communicable Disease Surveillance Centre of the Public Health Laboratory Service (17 September, p 826), I wish to present similar information from Scotland, thus enabling a picture to be drawn of legionella infection in Britain as a whole.

The information for Scotland for the years 1977-81 inclusive has been presented elsewhere,¹ but the figures for 1981 are included for purposes of comparison and completeness. The laboratory diagnosis of all cases of legionella infection in Scotland in 1981-2 was made in this department, which carries out legionella examinations for most of Scotland. Eighteen cases were diagnosed in 1981 and 32 cases in 1982. *Legionella pneumophila* serogroup 1 was isolated from one patient in 1981. In two patients *L pneumophila* was shown by direct immunofluorescence in postmortem lung tissue. Another case of *L pneumophila* serogroup 1 was diagnosed both serologically and in fixed postmortem tissue. Thirty four patients had a fourfold or greater rise in immunofluorescent antibody titre to ≥ 128 . Fourteen had a static titre of antibody at a level of ≥ 256 . Forty two patients showed evidence of infection with *L pneumophila* serogroup 1, one with group 2, three with group 3, two with group 4, and one with group 5. A further patient showed a significant rise in titre of antibody to an atypical serogroup 4 strain (designated P185) originally isolated by Dr J O'H Tobin in Oxford. One patient in 1982 showed a significant rise in titre of antibody to *L gormanii* (*Fluoribacter gormanii*).

Forty of the cases were diagnosed as having pneumonia, one had an "influenza like" illness, five were reported as having a "chest infection," one "respiratory failure," and one "fever." One case had emphysema and another case (infected in England), who was part of a small point source outbreak, developed pseudobulbar palsy during the course of his illness. Figure 1 shows the age distribution of patients: most were aged 50-69. At

FIG 1—Age and sex of cases of legionella infection in Scotland 1981-2.