Letters

To the Editor

Weight of all births and infant mortality
SIR—In response to the paper by Saugstad on weight of all births and infant mortality (September 1981, p 185), we would like to share our experience in India concerning the influence of birth weight and gestation on mortality.

Perinatal, neonatal, and infant mortality was studied in an urban cohort in Delhi in a population of 100 000.1 Birth weight and gestation were important factors which influenced the outcome, except in babies with a birth weight of less than 1500 g in whom outcome was poor irrespective of the period of gestation. Perinatal, neonatal, and infant mortality was 703·70, 461·54, and 615·38, respectively, in birthweight group 1001–1500 g and fell to 3·89, 4·88, and 21·62 in the weight group 2501–3000 g. Above 3000 g the figures were 4·08, 5·47, and 17·89 (table 1). When gestation was considered along with birth weight, there were remarkable differences within the same birthweight group (table 2).

The overall perinatal mortality rate was 38·8, neonatal mortality rate 21·18, and infant mortality rate 46·5.

Similar influence of birth weight was observed in a hospital study of 27 000 consecutive births,2 even though the overall perinatal death rate was much higher (75·6). The perinatal death rate was 901·03 in the weight group 1001–1500 g and fell to 16·71 in the weight group 3001–3500 g, after which it again rose to 28·24 and 54·79 in the weight groups 3501–4000 g and above 4000 g, respectively. In this study 31·3% of births occurred with a birth weight of 2500 g or less and 7·79% with a birth weight of 2000 g or less.

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References

Epidemiology of sudden infant death syndrome
SIR—The study of a large birth cohort from which cases who subsequently died from the sudden infant death syndrome (SIDS) have been identified is a potentially exciting adjunct to our knowledge of this perplexing disorder. Unfortunately, however, the study based on the Cardiff Birth Survey1 may suffer from inadequate case ascertainment.

It is a common misconception that all cases of SIDS occur at home. Certainly, comparatively few such deaths occur to infants who are hospital inpatients but many are not ascertained as being dead until they have reached hospital. Indeed, the Foundation for the Study of Sudden Infant Death actually suggests to the health personnel that this is the advisable course to take. In a study carried out in Oxfordshire and West Berkshire4 we found that 29% of the sudden unexpected infant deaths were registered as having occurred at or en route to hospital. A further factor presumably lacking in this study is the identification of any child actually dying outside Cardiff. We found that a not inconsiderable number of such deaths occurred while the parents were on holiday outside the region, or shortly after they had moved house.

How might such biases affect the published results? Obviously the reported incidence will be a pronounced underestimate, but that in itself would not affect the calculation of the relative risks, nor the computation of overall risk.

Nevertheless, for the patterns of relative risk to be meaningful it is vital that there be no bias in the distribution of maternal, social, and medical factors.
in the cases omitted. But it is quite feasible to suggest that parents who are quickest to get their child to a hospital may be of higher social class, more likely to have used the health services, etc.

Is it too late to ask the authors to reanalyse their data in the light of these comments and attempt a complete case ascertainment? Perhaps they could also determine whether any of the factors that might still be significant could be explained by any of the others. Would reanalysis confirm findings from other large prospective studies\(^1\) that breast-feeding is not a significant association once smoking and social class have been taken into account?

SIR—We thank Dr Golding for her helpful comments on our paper. We agree that many cot deaths are registered at hospital, although in most cases the death actually occurred at home. In our study we included infants brought to hospital and certified there, and as a result we believe we are missing very few cases.

We did not seek to identify those variables that would remain predictive when all other variables are taken into account. Following Dr Golding’s own work\(^2\) \(^3\) \(^4\) \(^5\) we examined explanatory variables in pairs, a process which led to the discarding of some variables in favour of others. In particular, the cross-product relative risk of artificial feeding is 2.32 (\(\chi^2 = 9.13\), df = 1, \(p < 0.005\)). Adjustment\(^*\) for smoking habits reduces the relative risk to 1.90 (\(\chi^2 = 5.28\)) and adjustment for social class gives 1.96 (\(\chi^2 = 5.61\)), both significant at the 0.025 level; the effect of the one adjustment to some extent encompasses that of the other. The case-control study of Naeye et al.,\(^6\) in which 125 cases of the sudden infant death syndrome were compared with 375 controls matched for social class, date and place of birth, gestational age, sex, and race, but not maternal smoking habits, gave a relative risk of 1.30 for artificial feeding, which is compatible with either unity or the value of 1.84 that we obtained.

Our article did not intend to imply that in Cardiff the necropsies on the bodies of infants who had died from SIDS were deficient in terms of additional investigations, especially histological examinations, and we apologise if such an implication could be inferred. In Cardiff, indeed, a particular interest is taken in the pathological aspects of SIDS as all infant deaths are regularly reviewed by a postperinatal death survey team, which includes several pathologists, one of whom is responsible for collating histological findings. Our remarks were intended to record that specialised research investigations such as analysis of vitreous humour were not routinely performed.

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References