the previous infant(s) were killed by the parent, rather than dying of natural causes. Currently, there does not seem to be a mechanism for correcting the national childhood mortality statistics when later, correct diagnoses are made. For instance, in the 1990s, I am aware of at least 20 infants who were initially categorised as SIDS, but who in later years, after extensive child protection investigations, were deemed to have been killed, usually by smothering. Colleagues will know of other cases: the true number will be higher. It is unfortunate that the official statistics do not seem to be altered retrospectively, and remain a misleading figure for any research worker. I should add that, since none of the cases of parental killing of which I am aware involves twins, the conclusions of Platt and Pharoah are more likely to have been strengthened rather than weakened by such false diagnosis. However, as the number of SIDS continues to fall, it will become ever more difficult for research workers to compare small subgroups of SIDS within national mortality statistics unless the statistics are revised retrospectively in response to later correct diagnosis.

It is appropriate to warn of an additional hazard for research workers in this field. In the same issue of *Archives* there was an interesting letter from epidemiologists in Paris concerning the possibility of vagal overactivity as a cause of sudden infant death.[3] They referred to a "positive family history of SIDS". A particular hazard there is that, unless details of that family history are verified in considerable detail, mistakes may be made. In recent years I have been involved with families in which parents who have repetitively smothered or killed children have provided to paediatricians, genetic counselling services, and to SIDS research workers, a false family history of SIDS—for instance, mother saying that two of her own siblings "died of SIDS". Such statements invariably are taken at face value and become part of the medical history: they are included in family trees in the hospital notes, and they have been quoted and displayed in published research concerning SIDS, yet subsequent questioning of the relevant grandparent has revealed that no such infant deaths occurred. Presumably, the mother responsible for smothering or killing her child has invented the family history, either to gain more medical attention for herself, or as a cover to distract from her actions. A second reason for verifying the alleged previous infant deaths in more detail is that, even if a death has occurred, it is necessary to explore the extent of the contemporary investigation and pathological examination. In one of Professor Emery's studies of infants initially categorised as SIDS, detailed re-assessment pointed to either a definite natural cause, or abuse, in two thirds of cases.[4]

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Use of inhaled corticosteroids in children

I read with interest the article Survey of adrenal crisis associated with inhaled corticosteroids in the United Kingdom by Todd *et al* and the accompanying editorial in the December issue of *Archives*.

In the reported cases, the children had been administered substantially (up to 5 times) higher than the Glaxo SmithKline (GSK) Core Data Sheet recommended Flixotide dose of 400 mcg/day and use of fluticasone (FP) at such doses is certainly not endorsed by GSK. Within the recommended doses, there are a wealth of data from controlled clinical trials that Flixotide is a well tolerated and effective drug in adults and children.[1–5] There are a number of recent studies in children which identified no cases of adrenal crisis and no effect on growth following 12 months treatment with FP at licensed doses.[6–8]

There are also a number of methodological deficiencies in this survey, the most important being that the survey is not case-controlled and lacks information on true incidence against the overall FP use or exposure. In addition, it is unclear from the survey what attempts were made to closely monitor any adrenal suppression with increasing doses of FP or whether patients were down-titrated to the lowest effective FP dose, as routinely recommended.

The survey data also imply that fluticasone has been implicated in the great majority of cases of adrenal failure even though it is the least frequently prescribed form of inhaled corticosteroid. Prescribing data in relation to fluticasone from the UK DINLINK (Doctors Independent Network) database, shows that it is in fact the most commonly prescribed inhaled corticosteroid in children with moderate and severe asthma.[9] DINLINK is an amalgamated database of the anonymised computer records of a panel of 300 general practitioners spread across the UK selected to represent the demographic population of the UK.

In addition, the authors' contention that adrenal effects with FP are due to its greater lipophilicity and hence accumulation over prolonged periods is misconceived and inaccurate. There are studies to show that there is no accumulation of FP at a steady state.[10] It is the clearance value which determines the amount of FP in the body at steady state, and lipohilicity per se in not a relevant factor.[11]

I also wanted to take this opportunity to comment on the editorial by Dr Russell. The last line of the editorial recommends that if high dose inhaled corticosteroid is considered necessary, that it is advisable not to use fluticasone. The recent publication by the CSM "Current Problems in Pharmacovigilance"[12] states that adrenal suppression is a dose related class effect of inhaled steroids, and that all inhaled corticosteroids are associated with an increased risk of adrenal crisis when used at higher than licensed doses.

In conclusion, inhaled corticosteroids have an important place in asthma management throughout the world, and this paper by Todd *et al* should be reviewed in this context. Any inhaled corticosteroid used at such high doses has the potential to cause systemic effects, and paediatricians should be encouraged to treat their patients using the lowest effective dose, down-titrating as appropriate.

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