We recently treated a girl aged 1½ years with steroids; however, she did not show any clinical response. She eventually improved with 6-mercaptopurine in a dose of 75 mg/m². We suggest that these patients should be given an initial trial of steroid for four to six weeks. If the response is poor or inadequate, treatment with alkylating agents or antimetabolites is warranted.

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Cerebral arterial air embolism in experimental neonatal pneumothorax

Sir,

Fenton *et al* reported five cases of air embolism in ventilated very low birthweight infants with fatal outcome.¹ Two of the infants had pneumothorax, in one case on both sides. They have said, that 'air embolism represents the extreme end of the range of air leaks' in the perinatal period.

We have observed the pial-arachnoidal microcirculation intravitaly (Wild Photomicroscope) on anaesthetised, immobilised, and artificially ventilated newborn piglets during the course of induced bilateral pneumothorax.^{2,3} Up to now we have studied 30 animals and in three cases we have found artificial cerebral arterial air microembolisation with fatal outcome within minutes. The affected microvessels showed significant vasoconstriction with the rapid cessation of the blood flow (figure). Concomitantly,

Figure Intravital microphotographs of the parietal pial-arachnoidal microvessels in a newborn piglet (a) before and (b) 40 minutes after the induction of experimental pneumothorax. Arrows point to the arterial air emboli accompanied by severe vasoconstriction. Bar=1.0 µm.

air bubbles were seen in the umbilical arterial blood sample in all three cases.

Our *in vivo* observations suggest that air embolisation may occur more frequently than has been reported during neonatal air leaks and may also affect cerebral microcirculation.

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Recurrent cot death and suffocation

Sir,

The careful study by Beal and Blundell shows an incidence of sudden infant death syndrome (SIDS) 10 times greater in the previous siblings of an infant who has died of the syndrome compared with controls.¹ They show that the circumstances and the families, in which two or more siblings died from SIDS, differed from those in which only one infant died in several important ways, including a more variable and older age at death. In postulating that 'both genetic and environmental factors seem to contribute to recurrence of SIDS in families' it is a pity that the authors do not confront the issue more squarely and acknowledge that some of these deaths will have been caused directly by the mothers—that is, filicide or homicide. In order to understand the epidemiology of SIDS better, and also to prevent deaths, it is important to recognise that a small proportion of children labelled as 'SIDS' are killed by their parents, usually their mother. Emery has suggested that between 2% and 10% of cot deaths result from filicide,² and in his detailed study of 12 families in Sheffield who incurred two or more cot deaths, he concluded that in five of those families filicide was either certain or probable.³

From my current study of 21 families in which the mother has suffocated a young child it has become apparent how often that child has originally been labelled as SIDS and contributed to false national statistics concerning the syndrome. Within these families a recurrence of unexplained, or definitely homicidal, deaths in other siblings is high; it is noteworthy that for those children previously labelled SIDS their age at death is both more