

was examined and found to contain amitriptyline three days after admission. The following day the mother was caught in the act of giving the child amitriptyline from a feeding beaker. It was later discovered that a sibling who had died at five months within 24 hours of discharge from a peripheral hospital had at necropsy an unexplained fracture of one humerus and a torn lingual frenum.

We concluded our initial report of the first case in 1968<sup>1</sup> by stating that the "mother gave her infant aspirin in the belief that it acts as a hypnotic but the borderline between deliberate and accidental poisoning is not always easy to define and in some cases it may have a similar aetiology to the 'battered baby syndrome,' the trauma having a chemical rather than a physical basis." This child is now of particular interest because although she was originally described as a case of drug poisoning, she has subsequently been physically battered on at least one occasion. Poisoning by parents before hospital admission despite parental denial is well documented,<sup>3,4</sup> but despite this experience and even having diagnosed amitriptyline poisoning in the second child we did not initially consider the possibility of repeated poisoning while she was in hospital. It now seems likely that this mother had physically battered and possibly killed one child and during the current admission was poisoning another. We emphasise that when children with a family background that puts them in an "at risk" physical battering category are admitted to hospital with unexplained disease, or have unexplained episodes of illness occurring while in hospital, deliberate poisoning should be considered in the differential diagnosis. We draw further attention to this manifestation of the "child abuse syndrome" so that social workers and lay magistrates as well as doctors may realise that it carries a significance just as serious as the more emotive physical battering.

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<sup>1</sup> Pickering, D, and Ellis, E, *Proceedings of the Royal Society of Medicine*, 1968, **61**, 1256.

<sup>2</sup> Pickering, D, *American Journal of Diseases of Children*, In press.

<sup>3</sup> Pickering, D, *Acta Paediatrica*, 1964, **53**, 501.

<sup>4</sup> *British Medical Journal*, 1968, **2**, 225.

SIR,—Dr D Rogers and others (3 April, p 793) report six cases of non-accidental poisoning as an extended syndrome of child abuse. I have seen two similar cases.

The first patient, a 14-year-old girl, presented with drowsiness and double vision. On examination she was lethargic but conscious and was ataxic. Her symptoms resolved spontaneously and she was discharged, but at follow-up she was found to have ataxia and nystagmus and to be drowsy. An electroencephalogram suggested barbiturate activity. Analysis of serum confirmed the presence of phenobarbitone and other unidentified drugs. Her father was an epileptic on combined phenobarbitone and phenytoin. He then admitted that the child's two siblings had had an identical illness at puberty and that he had administered the drugs.

An 8-year-old boy presented with drowsiness, vomiting, and ataxia. He had had headaches for two months. On examination he was drowsy and ataxic and had nystagmus. His stepfather was an epileptic on phenobarbitone and phenytoin. In view of our first experience blood was sent on

admission for toxicological examination, but before the results were available he was investigated by lumbar air encephalography as his signs were thought by a neurologist too convincing to be ignored. Analysis of the serum confirmed the presence of phenytoin.

I share Dr Rogers and his co-authors' opinion that this syndrome is more frequent than is at present realised and I emphasise the usefulness of the family history in both these cases. While I commend their paper, particularly for its suggestions on diagnosis and management, it must be remembered with older children that they may be self-administering the drugs and that the whole family needs to be carefully assessed. In my second case it never became clear who was responsible for administering the drugs.

I would like to thank Dr B D R Wilson for permission to report these two cases.

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### Induction and perinatal death

SIR,—The early studies of the Aberdeen school<sup>1-4</sup> confirmed the long-known<sup>5,6</sup> association of an increased perinatal mortality with increasingly prolonged pregnancy and also increasing incidence of difficult labour and traumatic delivery. In addition, however, and most important, those and later reports<sup>7</sup> demonstrated that in Aberdeen a large proportion of these deaths was associated with intrauterine or intrapartum anoxia and with the increase in difficult labour and traumatic delivery which was inevitable in primigravidae as pregnancy became prolonged.<sup>2,7</sup> It was therefore decided to select out primigravidae over the age of 25 who had proceeded at least seven days past certain dates (7% only of all primigravidae) and electively induce labour. There was developed in addition a greater awareness of the clinical significance of meconium and of slow and irregular fetal heart rates in labour, and especially postdates. The combined effect of induction, a greater awareness of the clinical syndromes of postmaturity, and the associated improvement in quality of care inevitably greatly reduced perinatal mortality in these groups, which were of course at that time the major clinical cause groups of perinatal death in primigravidae over 30.

The Aberdeen team of those days, now widely dispersed, and Baird in particular, have never claimed a direct relationship between induction and perinatal mortality in general but only in narrowly defined groups of special clinical causes of perinatal death.

It is quite impossible to analyse and interpret perinatal death results except in the broadest sense<sup>8,9</sup> without a careful clinical assessment of the circumstances of each individual death and an allocation to clinical cause groups.<sup>10,11</sup> Only by this method can there be any clear idea of the real "causes" and the clinical action necessary to improve results and care. Areas concerned with their own perinatal mortality by a failure to fall would be well advised to analyse their material in this way.<sup>12</sup>

Perinatal mortality rates of very low figures (table) should be attainable in most communities but only with high-quality care based on knowledge so obtained. Even then, however, there are vagaries in the incidence of fetal

Perinatal mortality rates (per 1000 births), single legitimate maternities, Dundee City, two contrasting groups

	1956-9	1968-73
Fetal deformity .. ..	8.8	5.1
Antepartum haemorrhage ..	5.7	2.5
Premature, cause unknown ..	5.3	3.0
Maternal disease .. ..	2.8	1.0
Toxaemia .. ..	2.5	0.2
Trauma .. ..	3.0	1.0
Mature, cause unknown ..	3.6	1.5
Other causes .. ..	1.1	1.3
Total .. ..	32.8	15.6

deformity which may play havoc from time to time with results. The perinatal death risk now is so small in general and the risk of traumatic or prolonged labour so slight that induction, except in the highly selected situation, could not of itself be expected or asked to reduce rates further.

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<sup>1</sup> McKiddie, J M, *Journal of Obstetrics and Gynaecology of the British Empire*, 1949, **56**, 386.

<sup>2</sup> Walker, J, *Journal of Obstetrics and Gynaecology of the British Empire*, 1954, **61**, 162.

<sup>3</sup> Stewart, D B, and Bernard, R M, *Journal of Obstetrics and Gynaecology of the British Empire*, 1954, **61**, 318.

<sup>4</sup> Turnbull, E P N, and Baird, D, *British Medical Journal*, 1957, **2**, 1021.

<sup>5</sup> Ballantyne, J W, *Journal of Obstetrics and Gynaecology of the British Empire*, 1902, **2**, 521.

<sup>6</sup> Clayton, S G, *Journal of Obstetrics and Gynaecology of the British Empire*, 1941, **48**, 450.

<sup>7</sup> Walker, J, *American Journal of Obstetrics and Gynaecology*, 1958, **76**, 6.

<sup>8</sup> Chalmers, I, et al, *British Medical Journal*, 1976, **1**, 735.

<sup>9</sup> Freidrich, J, and Yudkin, P, *British Medical Journal*, 1976, **1**, 738.

<sup>10</sup> Baird, D, et al, *Journal of Obstetrics and Gynaecology of the British Empire*, 1954, **61**, 433.

<sup>11</sup> Walker, J, *Transactions of the Edinburgh Obstetrical Society*, 1974 Session CXXVII.

<sup>12</sup> McIlwaine, G M, et al, *Health Bulletin*, 1974, **32**, 103.

### Nit-picking?

SIR,—I greatly enjoyed Dr John Apley's article on the pleasures of medical writing (24 April, p 999). I hope to have many happy years cribbing from it—as I hope he intended.

I wonder, however, if he would be kind enough to tell me where I can obtain one of these tooth-combs which he recommends for going through proofs and whether it was originally a dentist's tool?

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SIR,—How can Dr John Apley (24 April, p 999), who professes such respect for words, use the cliché "tooth-comb"? What is a comb without teeth? The original expression "with a fine-toothed comb" made sense; the modern "tooth-comb" is nonsense. Dr Apley should take his own advice: stamp on it, break its back, and kick it aside.

CHRISTOPHER HEATH

Truro

\*. Whatever Mr Heath may think of it, "tooth-comb" is certainly no neologism and is accorded respectability by inclusion in the *Oxford English Dictionary*, which defines it as "a small-tooth comb." *Chambers Twentieth Century Dictionary* describes it in more detail as "a comb with fine teeth placed close together." To obtain one Dr Whimster should apply to a parasitologist rather than a dentist.