Order, necessary to protect its life, may evoke strong feelings in that child after the break-up of what has previously been a close-knit relationship.[3]

We think the psychiatric aspects of this tragic family disorder differ in important respects from those of classical child abuse and that at present they are a long way from being fully understood. The analogy with the Munchausen syndrome[4] is valid as a description of the family's presentation and of the pathological cry for help, but it cannot explain the disturbed family relationship. We note that in Lorber's account[1] the father was strikingly present through a complete *absence* of any description at all, and also that the mother was described as 'a psychopathic personality and . . . a pathological liar' without reference to the family system she is, or was, a part of. Our cases have been managed with a psychiatric approach and psychiatric inpatient treatment for parent or child; this approach, rather than punitive legal action, seems to have been successful for an earlier patient reported from Sheffield.[5]

Much might be learnt from long-term follow-up of the many cases now known to paediatricians throughout the country, and we would like to co-operate with other workers to try to find the most appropriate course of action to take for subsequent management.

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Professor Lorber comments:

I agree with Rogers *et al.* that a psychiatric approach in the management of the mother was very important; a psychiatric colleague was directly concerned early on in the case and she was also an important part of the team dealing with the mother. However, the future risk to the child was so great that neither the social services nor the doctors felt that it was safe to leave the mother entirely in the hands of professionals without taking the matter to Court and asking for its decision.

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Hyperglycaemia in Lomotil poisoning

Sir,

I should like to draw your attention to the hitherto unreported association of transient hyperglycaemia with Lomotil (diphenoxylate and atropine) poisoning.

An 11-month-old boy, who had previously been well, was admitted as an emergency. He was comatose, had shallow respirations, constricted pupils, and peripheral cyanosis. He had been put to bed 5 hours earlier and had not drunk or eaten since that time. Two consecutive Dextrostix recordings, carried out immediately on admission, read '>13·9 mmol/l' (>250 mg/100 ml). His parents then admitted the possibility of Lomotil ingestion and he was given 0·1 mg naloxone with immediate clinical effect. A laboratory estimation of plasma glucose on an initial specimen, taken on arrival, was 30·8 mmol/l (555 mg/100 ml) with urea 4·5 mmol/l (27 mg/100 ml), sodium 135 mmol/l, potassium 4·3 mmol/l, total CO_2 20·5 mmol/l, and chloride 102 mmol/l. Three hours later (6 to 8 hours after ingestion) this had fallen to 5 mmol/l (90 mg/100 ml) with similar electrolytes. Urine, first obtained 8 to 10 hours after ingestion, showed 0·25 to 0·5% sugar on Clinitest testing. Subsequent testing of blood and urine showed neither hyperglycaemia nor glycosuria. The possibility that Lomotil might interfere with the assay of plasma glucose was investigated and excluded.

A search of the literature has not revealed a similar reported occurrence, and neither of the active components of Lomotil (diphenoxylate or atropine) has singly been associated with hyperglycaemia. It is difficult to understand the causative mechanism, although it may be suggested that the relative preponderance of sympathetic activity in atropine poisoning is contributory.

I suggest that Lomotil in overdosage should be considered in the unconscious child with hyperglycaemia in whom the clinical and biochemical findings are not those of diabetic ketoacidosis.

I thank Dr D B Horn for carrying out the biochemical assays and Dr A J Keay for permission to report this case.

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Clostridium difficile-associated colitis

Sir,

My 5-month-old son received a 5-day course of oral phenoxymethyl penicillin for pharyngitis. About one week later he passed blood-stained motions for 2 days, but appeared well. For the next 5 months he had frequent loose bowel actions but no more blood was seen and he remained well. At this stage a stool was tested and found to contain toxin neutralisable by *Clostridium sordellii* antitoxin, suggesting the presence of *Clostridium difficile* infection. Treatment with oral metronidazole caused the disappearance of the toxin within 24 hours and the loose frequent bowel actions ceased. Toxin was absent from