Suspected poisoning in childhood

Sir,

All new information on the problem of ingestion of poisons in childhood is valuable. However, it would be damaging if a sense of complacency were engendered by the work of Calnan, Dale, and de Fonseka (1976). It is undisputed by anyone who has worked in this field that the majority of children coming into hospital come to no harm, and in retrospect the incidents seem to be trivial. However, it would be dangerous to assume that these children are aetiologically different from those who suffer serious side effects from ingesting poisons. The child who takes his grandmother’s antibiotics is no different, aetiologically speaking, from a child who takes his grandmother’s digoxin. However, one would be classified by Calnan as a poisoning scare and the other as a true poisoning.

There must be doubt also about the method of Calnan et al. of classifying their ‘poisoning scares’ and ‘true poisonings’. It is the aim of treatment to empty the stomach as soon as possible to prevent symptoms. How many of Calnan’s children would have developed symptoms had the stomach remained unemptied? The majority of children’s departments throughout the United Kingdom now use Ipecacuanha emesis. This is an effective and humane method of emptying the stomach (Boxer, Anderson, and Rowe, 1969), and its use prevents many of the problems of hospital admission that Calnan and his colleagues fear.

There is one commonly taken poison for which blood levels are easily available. This is aspirin. The vast majority of children who present to casualty after aspirin ingestion have detectable salicylate in their blood, which suggests that children are not commonly presented to casualty with a suspicion of taking poisons who have in fact not taken the poison at all. If children who have probably not taken sufficient amounts of poison are sent home there will occasionally be disasters. I can do no better than quote from the report of Medical Defence Union in 1975 where the death of a child from aspirin poisoning is described. He had been sent home from hospital having been thought to have taken insignificant amounts of junior aspirin. The Medical Defence Union emphasized the importance of admitting all such children to hospital. It has been shown that stress is an important factor in the aetiology of ingestion of poisons in childhood (Sibert, 1975; Julyan and Kuzemko, 1975). Parents may not be able to give accurate information at such times. When the work of Calnan, Dale, and de Fonseka was published previously (Calnan, 1974) in the correspondence which followed in the British Medical Journal, de Fonseka (1975) said that even very anxious mothers can be adequately questioned when first seen, and can provide very reliable information. I believe this not always to be the case.

In addition, there may always be the possibility of nonaccidental poisoning (Rogers et al., 1976). The diagnosis here seems always to be difficult and would be almost impossible to make if the children were sent home. Accidental child poisoning may be a symptom of a variety of family problems. It is usually impossible to find out about these and to alert the appropriate agency if the child is not admitted to hospital.

It may be that in time, with the improvement of liaison between general practice, the community services, and the hospital, children who have ingested poisons will be sent home for adequate observation. However, at the present time safety demands the majority of children should be admitted to hospital.

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REFERENCES

Dr. J. W. Dale comments as follows:

Thank you for the opportunity of replying to Dr. Sibert’s letter. It was not our intention to introduce a sense of complacency about the number of children brought to hospital each year with suspected poisoning. We wished to distinguish between poisoning scares not requiring treatment and true poisoning that may require active treatment, to avoid the types of death referred to by Dr. Sibert while at the same time not increasing the load on hospital facilities.

The aetiology of a disease is the study of factors which cause the disease; our paper made little reference to
causes of child poisoning and concentrated on the process by which true poisonings are distinguished from poisoning scares in the home and in casualty. We do not know whether the factors causing poisoning scares are similar to the factors causing poisoning.

Dr. Sibert was right to draw attention to the need to study stomach contents and blood levels; these need to be related to parental evidence so that we can improve our discrimination. We have no wish to imply that parents are overanxious in seeking medical help. Nevertheless, though the occasional parent is unable even after questioning to provide reliable information, and this should be recognized by the Casualty Officer, many mothers can reply to appropriate questions by the Medical Officer.

The child who has taken a dangerous dose of digoxin differs from the child who has swallowed a few tablets of penicillin; the one child may die without treatment, the other will certainly survive. We disagree with the view that the aim of treatment for a child brought to hospital is to induce vomiting, however effective or humane the methods used, if this is not a necessary procedure in the interest of the child's health.

As we recorded in our paper, in only 22% of cases did the child develop symptoms, and in a further 13% was there no evidence that the child had swallowed a toxic substance. We offered no figures on the risks to the child's life. However, only one child in this study suffered permanent damage—from swallowing caustic soda.

In the light of Dr. Sibert's evidence on the relation of stress to suspected poisoning, we must support again the introduction of childproof containers which should make the evidence of true poisoning even easier to determine.

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Sir,

We were interested in the article by Griffiths, Bull, and Dykes (Archives, 51, 321, 1976) concerning the effect of gestational age on albumin content of meconium. In a similar study performed on 183 newborn infants of less than 38 weeks' gestational age, a BM-Test Meconium as described by Stephan et al. (1975) was performed. According to the latter authors, the test is positive if the albumin concentration is at least 20 mg/g dry weight of meconium. In our series the incidence of positive BM-tests on the first meconium sample is 15·3% and in agreement with the high incidence (8%) found by Griffiths et al. in infants of less than 37 weeks' gestational age. Taking into account both gestational age and weight (Lubchenco, Hansman, and Boyd, 1966), the Table shows that the incidence of raised meconium albumin levels is particularly high (36·4%) among small-for-dates infants with birthweight below the 10th centile for gestational age (Lubchenco et al., 1966).

In a previous study (Eggermont, 1966), we were able to show that the decrease of protein content in meconium according to fetal age is not due to dilution by increased accumulation of mucopolysaccharides but to the proteolytic activity which, however, is not entirely due to pancreatic proteases. These observations should be kept in mind when meconium albumin tests as a screening technique for cystic fibrosis are performed in preterm infants.

**TABLE**

Results of BM-Tests Meconium for albumin in the first meconium samples of 183 preterm infants of <38 weeks' gestational age and consecutively admitted to the newborn nursery

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Positive BM-Test Meconium</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. (%)</td>
</tr>
<tr>
<td>All infants</td>
<td>183</td>
<td>28 (15·3)</td>
</tr>
<tr>
<td>Weight groups</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;P 90</td>
<td>7</td>
<td>0 (0)</td>
</tr>
<tr>
<td>P 10-P 90</td>
<td>143</td>
<td>16 (11·2)</td>
</tr>
<tr>
<td>&gt;P 10</td>
<td>33</td>
<td>12 (36·4)</td>
</tr>
</tbody>
</table>

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**REFERENCES**


Dr. A. D. Griffiths comments:

Thank you for allowing us to reply to the letter from Professor Eggermont. His findings are in agreement with ours, confirming that preterm infants have raised levels of albumin in their meconium. This can lead to an unacceptable number of false-positive results when screening for cystic fibrosis using a method based on the albumin content of meconium. It may be possible, however, to increase the specificity of the screening procedure by subjecting those meconium samples with raised protein levels to further tests such as described by Ryley et al. (1975) and Antonowicz, Ishida, and Shwachman (1976).
Dr. J. W. Dale comments as follows

J. W. Dale

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Updated information and services can be found at: http://adc.bmj.com/content/51/11/900.2.citation

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