(4) That while we may regard health education as wholly desirable if it reduces true poisoning in children, this education in its present forms of commercial advertising and television programmes may also have the effect of generating poisoning scares. Parents are naturally anxious about the health of their children and these health education techniques may have heightened this anxiety. The consequence of this may be that some children have to suffer unnecessary clinical procedures in the hospital. Health education methods must therefore be examined in terms of their costs and benefits.

We are all agreed that suspected poisoning is not a random event but occurs under certain special social circumstances. We believe it to be useful to examine these circumstances and to recognize that those which produce a poisoning scare may be different from the circumstances which lead to a case of true poisoning. We expect then to be in a better position to reduce the latter without inflating the former. Policy until now has meant that parents have had to take full responsibility for the prevention of suspected poisoning in children and have had to suffer the consequences. However, evidence in the above correspondence suggests that in practice parents may have limited control over the events which lead to suspected poisoning. The responsibility for prevention must be shared and parents should be provided with preventive measures which have been shown to be effective through experiment and field studies.—We are, etc.,

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1 Calnan, M. W., *Community Health*, 1974, **6**, 91.

SIR,—Two points raised by Drs. A. W. Craft and R. H. Jackson in their letter (11 January, p. 95) about the research by Calnan[1] call for comment. I set up the research project.

By considering all the evidence it was possible to distinguish clearly between those children who developed symptoms and/or ill effects and those who did not. Those who had a quantity of a toxic substance retrieved from the stomach were classified as poisoned, even though no symptoms developed. It was found that all children admitted, irrespective of symptoms or ingestion, were recorded by the hospital as "poisoned" whereas children not admitted never reach the national statistics. Therefore nationally there appeared to be a gross overestimate of those actually poisoned and a considerable underestimate of poisoning scares. I do not think that distinguishing between true poisonings and poisoning scares will lead to an increase in the number of children dying; it can only clarify the situation and help more rational management.

The second point I wish to make is that no one, however experienced or otherwise, could hope to predict the onset of symptoms unless he first seeks evidence of possible ingestion. The following recent personal clinical experience illustrates my point. It involved an 18-month-old child found by the mother with an open bottle of aspirin and a few fragments in the mouth. Since the number of missing tablets was unknown, the simplest decision would have been to wash its stomach out and admit it, to be eventually classified as "poisoned." However, on close questioning of the mother it became quite evident that she had not left the child alone in the room with the bottle for more than one minute, which could have given the child enough time to open the bottle and put one tablet at the most in its mouth. I then reassured the mother, who had herself by then realized the possibilities with obvious relief.

The research demonstrated that the appropriate questions were not being asked and therefore admission appeared arbitrary, possibly reflecting clinical judgement without facts. Assessment of risks in management is accepted clinical practice. Only appropriate questions can determine the facts on which to decide whether a child with a head injury needs admission. Many children with such injury are sent home because intracranial complications are thought highly unlikely.

I find that even very anxious mothers can be adequately questioned when first seen and provide very reliable information. The demonstration of this to young doctors is very easy and they quickly grasp what it is all about. But it requires experienced people directly involved in the primary-care front line to lay down the guidelines.—I am, etc.,

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1 Calnan, M. W., *Community Health*, 1974, **6**, 91.

## Small Bowel Tumours

SIR,—Your leading article (18 January, p. 115) contains an ambiguous statement which is confusing. In referring to complications of adult coeliac disease you say: "Both lymphoma and carcinoma occur, but the incidence of carcinoma is reduced if the patient is treated with a gluten-free diet; this is as yet unproved for lymphomas. These findings justify the continued use of gluten-free diet for the whole of the patient's life."

Such a statement in an article on small bowel tumours implies that the lymphoma and carcinoma are situated in the small bowel. Though it is now well established that lymphomas of the small bowel may complicate adult coeliac disease,[1] the true relation of small intestinal carcinoma to adult coeliac disease is not known. Indeed, the same series of adult coeliac disease which established its association with abdominal lymphoma[1] contained no carcinomas of the small bowel. The carcinomas referred to were situated in the oesophagus and elsewhere in the gastrointestinal tract. You support your premise by reference to a paper by Holmes *et al.*,[2] which is from the same group and which presumably contains substantially the same cases. This paper refers vaguely to "all cancers" and to "carcinoma of the oesophagus and reticulosarcoma." No specific mention of small bowel carcinoma is made.

Some 16 cases of jejunal carcinoma associated with either steatorrhoea or histological villous abnormality have been described in the literature.[3-6] In all but one of these the mucosal histology was studied at the time of, or within months of, the carcinoma being resected. This makes them subject to the hypothesis that the mucosal abnormality is a result of the carcinoma. In the other case[3] the diagnosis of adult coeliac disease was established seven years before resection of the carcinoma. I studied a similar (unpublished) case in a woman who had suffered from steatorrhoea in early childhood and was diagnosed as having adult coeliac disease with jejunal biopsy evidence of subtotal villous atrophy at the age of 56 years. She went on to develop a jejunal carcinoma five years later.

I think it is likely that jejunal carcinoma may be a complication of adult coeliac disease, but as yet the case is supported by circumstantial evidence only and not proved as your leading article implies.—I am, etc.,

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1 Harris, O. D., *et al.*, *American Journal of Medicine*, 1967, **42**, 899.
2 Holmes, G. K. T., *et al.*, *Gut*, 1974, **15**, 339.
3 Kenwright, S., *Postgraduate Medical Journal*, 1972, **48**, 673.
4 Asch, T., and Seaman, W. B., *Radiology*, 1971, **100**, 271.
5 Blackwell, J. B., *Gut*, 1961, **2**, 377.
6 Joske, R. A., *Gastroenterology*, 1960, **38**, 810.

## Metoclopramide and Breast-feeding

SIR,—Paediatricians in developing countries are deeply involved in the battle against the decline of breast-feeding. It has recently been reported[1] that metoclopramide is a potent stimulator of prolactin release. This could be important in the prevention of the premature weaning that has serious consequences in preindustrialized areas. I have therefore tried the effect of giving 10 mg of metoclopramide three times daily to five lactating mothers who had suffered a severe decrease in milk volume for more than 72 hours. In every case the results were most encouraging. Both mother and child were put under close observation during the 7-10 days of treatment because metoclopramide might produce side effects in the mother[2] and I do not know how much of the drug is excreted in breast milk. In these five cases no side effects were observed.

I am at present investigating the clinical use of the drug in larger trials and trying to determine the amount of metoclopramide excreted in breast milk.—I am, etc.,

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1 McNeilly, A. S., *et al.*, *British Medical Journal*, 1974, **2**, 729.
2 Shaklay, M., Pinkhas, J., and de Vries, A., *British Medical Journal*, 1974, **2**, 385.

## Removal of Potassium from Fruit Juices by Ion Exchange

SIR,—Many attractive drinks are denied to patients with renal failure because of their high potassium content. The addition of resins to juices has been described,[1 2] potassium being exchanged for sodium or hydrogen. But these have the disadvantage of increasing the sodium intake or lowering