Correspondence

Gaviscon bezoar was first described in 1976 by Hewitt and Benham and similar complications have also been reported in the neonate from the use of aluminum hydroxide gel. If our experience is typical the condition may be much more common than has been suspected, and may occur after very brief periods of treatment. Sinaasappel et al removed much of the mass with a gastroscope and noted improvement in their patient's condition after this intervention. Our experience in the two patients where repeat barium studies were performed and that reported elsewhere suggest that such active intervention is unnecessary. The only action required for spontaneous resolution of the bezoar is to stop Gaviscon treatment.

Dr Bankier and co-workers comment:

We thank Dr Reynolds for drawing attention to the potential problems of neurotoxicity after chronic ingestion of high doses of pyridoxine. The only report of neurotoxicity in man postdated our paper on pyridoxine dependent seizures. In reply to Dr Reynolds' comments:

1) We know of no evidence to suggest that a single dose of pyridoxine is harmful even if the child is not pyridoxine dependent. Pyridoxine given as a diagnostic test in a fitting child is likely to be less harmful than the obvious danger of missing a treatable disorder.

2) There was no evidence of deterioration after pyridoxine treatment in any of our patients. The developmental delay reported was present at the time of the first administered dose. In fact the one patient (case 5) treated from day 1 had an "intelligence quotient in the low to normal range" at 5 years of age. None of these children had peripheral neuropathy. Case 3 developed signs consistent with spinomuscular atrophy. This is an inherited condition which had affected her sibling who had never been exposed to pyridoxine.

References

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Pyridoxine dependent seizures

Sirs,

In a recent article in this journal Bankier et al described case reports of four infants with a variety of seizures that responded to large intravenous or oral doses of pyridoxine.

The high doses used by these investigators are well within the range that has been shown to be neurotoxic in mature adults and substantially above the lowest dose observed to result in adult neuropathies (H Schaumburg, personal communication).

Bankier et al administered doses of 100 mg pyridoxine intravenously and then maintained the infants on 75 mg orally. Some form of subsequent impaired mental development was noted in all infants. The ages ranged from 1 day (case 5) to 11 months (case 3). In case 5, the birthweight was 3300 g and pyridoxine was given four hours after birth at a dose of 100 mg intravenously. That is 30-3 mg pyridoxine/kg body weight intravenously in an infant who is undergoing massive and rapid development of the central nervous system. The minimum adult oral dosage shown to result in neuropathies is approximately 7 mg/kg body weight or approximately a quarter of the dose given to these infants. Bankier et al reported in case 3 that 'The results of investigations for weakness and hypotonia were consistent with spinal muscular atrophy . . . , symptoms which are consistent with those reported for toxicity in adults' and in beagle dogs.

I maintain that the recommendation proposed by Bankier et al that 'A neonate with seizures, even with documented birth asphyxia, should be given 100 mg of intravenous pyridoxine . . . ' is both potentially harmful to the infant and irresponsible unless there is substantial evidence that potentially toxic doses are absolutely necessary for the survival of the infant. Bankier et al did not investigate the efficacy of maintenance doses less than 75 mg/day on the infants, and their treatment may, in fact, have contributed substantially to the impaired mental development noted by the authors.

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Figure Two Gaviscon bezoars present in the stomach.
Pyridoxine dependent seizures.

R D Reynolds

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