Correspondence

Adverse effects of nasogastric feeding tubes and the management of recurrent apnoea

Sir,
The role of miconazole in the treatment of systemic candidiasis was clearly described by Tuck in his report of 2 cases. However, his paper also contained examples of the iatrogenic hazards of neonatal intensive care and these deserve comment.

In each case intravenous feeding was given through a centrally placed Silastic cannula because of recurrent apnoea. In Case 1 the sequence of events is clearly documented. From the time that nasojejunal feeds were introduced apnoeic episodes occurred, and on day 29 the baby collapsed after the passage of a nasal feeding tube. Earlier in 1980 Stocks described work done at the Hammersmith Hospital which showed that nasal resistance in white infants was increased 101–138% when a feeding tube was passed through one nostril. She also confirmed the common observation that a nasally-passed tube causes not only physical obstruction but also irritation to the nasal mucosa which results in increased resistance. As most preterm babies are unable to mouth breathe even when both nostrils are fully obstructed the adverse effects of nasally passed tubes on respiration, if overlooked, may lead to further complications of management as described by Tuck.

He states that there is an increase in the use of intravenous feeding in the management of preterm infants and that systemic candidiasis can be expected to become more common. This and other complications of intravenous feeding should be avoided. The experience in Oxford suggests that intravenous feeding can be restricted almost exclusively to the few babies in whom necrotising enterocolitis is suspected, and that in preterm infants and others with respiratory distress feeding tubes should always be passed orally. Although apnoeic attacks may occasionally be the result of regurgitation of gastric contents this has never been taken to be an indication to resort to intravenous feeding through a centrally placed catheter.

References

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Dickens’s children’s hospital

Sir,
Douglas Gairdner wondered whether 'the children's hospital' mentioned in Our mutual friend was The Hospital for Sick Children, Great Ormond Street. That this was so is an opinion which has strong support, but there was another hospital which Dickens greatly admired and this was the East London Children's Hospital in Ratcliff. It was the subject of the sketch 'A small star in the East', written for All the year round and appearing later in The uncommercial traveller.

I have been unable to find the date of publication of ‘A small star in the East’ but most of the essays and sketches in The uncommercial traveller had appeared by 1865, which is the year in which Our mutual friend was completed; therefore it is possible that it is the East London Children's Hospital which is referred to in Dickens's last great novel.

References

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Possible pitfalls in the interpretation of inspiratory flow volume curves

Sir,
Smith and Cooper demonstrated residual upper airways obstruction in 7 of 20 children who had laryngomalacia in infancy. Certainly the non-invasive recording of an inspiratory flow volume curve is a useful method in the assessment of extrathoracic airways obstruction.

It is doubtful if a shallow inspiratory curve alone (and the resulting high midvital-capacity ratio) is sufficient proof for the existence of an obstructing lesion. In the case of patent upper airways the inspiratory flow is mainly effort dependent. In contrast to the expiratory flow volume curve which is determined by dynamic intrathoracic airways compression, suboptimal effort alone can produce a seemingly abnormal inspiratory curve and consequently a low inspiratory flow measurement at 50% of the vital capacity. Some children are consistent in their level of suboptimal effort, thereby faking reproducibility in repeated recordings.

There is a satisfactory way to differentiate suboptimal inspiratory effort from true flow limitation by stenosis: a extrathoracic upper airways obstruction produces a
Reproducibility of skin prick reactivity in cystic fibrosis

Sir,

Holzer et al.\textsuperscript{1} reported considerable variation in responses to allergen skin testing over a few months in a small group of children with cystic fibrosis (CF). However, we found only minor fluctuations in the skin test reactions of 62 patients with CF. These children and adolescents attended our laboratory on at least two occasions for allergen skin prick tests.\textsuperscript{2} The average time between the first and last study was 19 months (range 3–26), while the mean age of the group when last tested was 10.7 years (range 4.1 to 18.8). We defined atopy as a weal of 3 mm diameter greater than a negative control to one of 5 allergens.\textsuperscript{3}

Thirty-seven children remained non-atopic, and 15 remained atopic, thus, 52 (84\%) out of 62 CF patients had reproducible results. Furthermore, 8 of the 10 subjects who showed varying responses became atopic, which is in keeping with the increase in prevalence of skin test reactivity during childhood. It is important to note that half of the children who converted to atopic by our criteria had at least one allergen weal of 2 mm or greater when first tested and one of those who reverted to non-atopic still had 2 allergen weals of 2-5 mm.

Our data do not support Holzer et al.\textsuperscript{1} since we found that atopy, as defined by skin prick reactivity, was reproducible in the majority of our patients.

Mr Holzer and co-workers comment:

From the data it is obvious that there is little difference between our figures and those of Henry et al. Fifty-two (84\%) out of 62 of their subjects had reproducible results (consistently positive or consistently negative) while 10 (16\%) were variable. Our figures of 19 (76\%) out of 26 patients with reproducible results and 6 (24\%) with variable results were not significantly different from theirs ($X^2 = 0.30$).

It is interesting that 48\% of our patients were consistently positive while only 24\% of their patients were always positive. Part of this difference may be explained by the consistently positive reactions to Aspergillus fumigatus in children with more severe lung disease in our group. Henry et al. do not comment on the clinical spectrum of their patients. The important message from both studies is that there is a group of children with cystic fibrosis who show variable results to allergen skin testing and that to define atopy on the basis of tests done on one occasion can be misleading.