LETTERS TO THE EDITOR

Recurrent cyanotic episodes with severe arterial hypoxaemia and intrapulmonary shunting: a mechanism for sudden death

SIR,—In a recent paper Dr Southall *et al* suggest that rapid shunting of unoxygenated blood through the pulmonary vascular bed is a cause of sudden and unexpected death in infants and children and that this may be causally related to the sudden infant death syndrome (SIDS).¹ This assertion is based on the occurrence of sudden death in eight of their 51 patients (16%), four of whom died during cyanotic episodes.

While the hypothesis and the physiological evidence for it are interesting and well presented, review of the fatal cases reveals that five of the eight had concurrent pathological processes that are independently associated with sudden death, and in some cases they had cyanosis: impacted intraoesophageal bolus of food in a patient with a repaired tracheoesophageal fistula ($n=1$), bronchopneumonia ($n=2$), bronchopneumonia with mucus within airways ($n=1$) and a brainstem glioma ($n=1$).^{2,3} It would strengthen the underlying hypothesis considerably if further information could be provided detailing how these well established causes of sudden death were eschewed in favour of intrapulmonary shunting as the prime mechanism responsible for death. Of even more importance is the need for clear documentation of the evidence for intrapulmonary shunting in the eight fatal cases, if this is to be accepted as the cause of death. It appears from the text that only one patient had contrast echocardiography (case 18) and that none had krypton infusion scans or postmortem injection studies. It would clarify matters considerably, therefore, if the antemortem and postmortem evidence for shunting in each of the fatal cases could be provided.

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- 1 Southall DP, Samuels MP, Talbert DG. Recurrent cyanotic episodes with severe arterial hypoxaemia and intrapulmonary shunting: a mechanism for sudden death. *Arch Dis Child* 1990;65:953-61.
- 2 Norman MG, Taylor GP, Clarke LA. Sudden, unexpected, natural death in childhood. *Pediatr Pathol* 1990;10:769-84.
- 3 Byard RW, Moore L, Bourne AJ. Sudden and unexpected death—a late effect of occult intraoesophageal foreign body. *Pediatr Pathol* 1990; 10:837-41.

Drs Samuels and Southall comment:

Dr Byard has raised important questions concerning our hypothesis that severe hypoxaemic episodes may cause sudden death in infants and young children. The presence of a structural abnormality at postmortem examination does not in itself always explain the cause of death. The cessation of cardiorespiratory and cerebral function must ultimately encompass a physiological mechanism. In our

patients, the findings of bronchopneumonia, an intraoesophageal impacted food bolus, and a brainstem glioma were alone insufficient, in our opinion, to explain their sudden and unexpected deaths. Two died during the night without any premonitory symptoms or signs, while the other three died during typical cyanotic episodes.

In all eight cases, our patients had suffered repeatedly from sudden and life threatening cyanotic episodes needing resuscitation. In the absence of physiological recordings during their deaths, we can only speculate as to the mechanism which caused their death based on the investigation of living infants undergoing similar hypoxaemic episodes having characteristics that could be described as life threatening. Although some of our patients did not undergo all the investigations required to confirm the development of an intrapulmonary shunt during their cyanotic episodes, their episodes were identical in all other respects to those occurring during krypton infusion scans or contrast echocardiography. Intrapulmonary shunting remains one of the best explanations for the sudden development of their life threatening hypoxaemia. How and where in the lungs this shunt occurs remains to be elucidated.

At present, an explanation for the final pathophysiological pathways leading to sudden and unexpected infant deaths, even in the presence of abnormal postmortem findings, remains unresolved.

Measles immunisation

SIR,—Active immunisation is efficient in preventing the central nervous system complications of measles.¹ The wisdom of current recommendations² on measles immunisation is illustrated by a case of progressive measles encephalitis after renal transplantation for treatment of the congenital nephrotic syndrome.

Immunosuppressive treatment after successful renal transplantation (age 6) was with azathioprine and prednisolone. The primary renal disease (congenital nephrotic syndrome) was considered to preclude pretransplant measles immunisation. At 10 years of age the child had a 'flu-like illness' with morbilliform rash. Three months later she suffered a second 'flu-like' illness and within days, developed myoclonic seizures involving arms, head, and subsequently legs. An electroencephalogram showed periodic and stereotyped bursts of high voltage slow components accompanying the clinical events. Seizures were absent during sleep; consciousness and intellect were preserved when awake. Anticonvulsant treatment was ineffective. Intrathecal synthesis of measles virus antibody was documented (serum titre 40, cerebrospinal fluid 4 in presence of normal blood cerebrospinal fluid barrier function; cerebrospinal fluid/serum albumin 2.4×10^{-3}). Rubella virus antibody (serum 640, cerebrospinal fluid <1, serum rubella virus IgM only weakly positive) was also found at this time but neither antibody was detectable in serial serum samples collected prior to the first 'flu-like' illness. Antibody titres were unchanged in a subsequent serum sample suggesting both measles and rubella to be temporally associated with the first illness (the second remained undiagnosed). Four months after the onset of seizures she developed chickenpox, lapsed into coma, and died six weeks later without regaining consciousness. Chickenpox probably acceler-

ated the clinical course but not by direct central nervous system invasion (serum Herpesvirus varicella 320, cerebrospinal fluid <1).

It is possible that rubella either triggered a latent measles virus infection (although we found no serological evidence of previous measles) or, interfered with the normal measles immune response (compounding the effects of immunosuppression) leading to progressive central nervous system infection.³ Whatever the mechanism, this case reinforces the view that all children going forward to renal transplantation should receive measles immunisation.

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- 1 Bloch AB, Orenstein WA, Stetler HC. Health impact of measles vaccination in the United States. *Pediatrics* 1985;75:524-32.
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- 3 Bhettay E, Kipps A, McDonald R. Early onset of subacute sclerosing panencephalitis. *J Pediatr* 1976;89:271-2.

Reflux vomiting

SIR,—It was kind of Dr Milla to refer to some of my publications in his article on reflux vomiting.¹ Unfortunately some of his interpretative conclusions require correction. Like many other writers on this subject he has totally misrepresented my 1959 observations on the natural history of vomiting infants with a partial thoracic stomach (hiatal hernia)² as being applicable to all infants with 'symptomatic gastro-oesophageal reflux'. This is most certainly not so. His quote relating to the incidence of gastro-oesophageal reflux is equally misleading as the hospital estimate of one in 500 to which he refers relates to the hospital attendance of children with a partial thoracic stomach and not to that of children with reflux per se as mentioned in his article. Clearly the number of infants attending hospital with reflux and no recognisable displacement into the thorax of the gastro-oesophageal sphincter would be greatly in excess of this figure. I am also perplexed by his statement that 'The finding of a hiatus hernia with or without an associated partial thoracic stomach is not of itself an indication for surgery'. I have tried substituting gastro-oesophageal reflux for hiatus hernia and for partial thoracic stomach but in neither instance does this alteration clarify the meaning of this sentence in the particular context of his article.

Largely as a result of the emphasis placed on the importance of gastro-oesophageal reflux per se and its detection by non-radiological means very much less attention is directed nowadays to the identification of a partial thoracic stomach, which is often regarded as having little clinical relevance. I have, however, found on long term clinical evaluation of many hundreds of infants with reflux that the presence or absence of a partial thoracic stomach serves as a very valuable clinical guide to prognosis.³ For whereas reflux in infants with a partial thoracic stomach is

associated with a significant morbidity, gastro-oesophageal reflux in the absence of this anomaly is a relatively benign self limiting condition, which in the case of vomiting infants can usually be treated successfully by appropriate and adequate thickening of feeds.⁴ I am therefore in no doubt as to the clinical importance of distinguishing infants with reflux and a partial thoracic stomach (in whom the antireflux contribution of the abdominal oesophagus is absent) from those with reflux as the only observed abnormality. Such reliance on the prognostic significance of a partial thoracic stomach is of course totally dependent on having an experienced paediatric radiologist as a colleague who is equally aware of the importance of carefully examining infants with reflux for a partial thoracic stomach.

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- 1 Milla PJ. Reflux vomiting. *Arch Dis Child* 1990; 65:996-9.
- 2 Carré IJ. The natural history of the partial thoracic stomach (hiatus hernia) in children. *Arch Dis Child* 1959;34:344-53.
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Growth after gut resection for Crohn's disease

SIR,—We read with interest the paper by McLain *et al*, and we would like to comment on both the methodology of their data collection and the authors' conclusions.¹ Firstly, this was a retrospective study, and thus has the major disadvantage of lacking standardisation in the collection of the auxological data. There is no mention in their paper of how height measurements were recorded and standardised, or on the accuracy of the staging of pubertal development.

Secondly, while agreeing with their findings that dramatic growth acceleration may occur after surgery for Crohn's disease, we disagree with their conclusion that 'catch up growth is not limited by the stage of puberty'. We have recently completed a large, prospective study investigating factors influencing growth after bowel resection for Crohn's disease in 42 children requiring surgery before their 17th birthday. All growth indices (including height measurements every three months) were performed prospectively by a clinical auxologist, and an accurate pubertal staging was performed in each patient at the time of surgery, together with the radiological bone age in the majority of cases. Our data (table) clearly show a strong relationship between height velocity in the first postoperative year and the pubertal status (Tanner breast and genital stage) at time of operation. The apparent lack of effect of pubertal status on growth in the study of McLain *et al* may merely reflect the very small number of children in advanced puberty (one), or the retrospective nature of their data collection. In our opinion, the timing of surgical intervention is vital, and should be performed before puberty becomes too advanced and the potential for catch up growth is lost. This is in direct contrast to the conclusion stated in their paper.

Finally, we monitor growth very carefully in all our children with Crohn's disease. Any

Mean preoperative and postoperative height velocities (cm/year) in 42 children undergoing bowel resection for Crohn's disease grouped according to pubertal status at time of operation

	Boys		Girls	
	Before surgery	After surgery	Before surgery	After surgery
Prepubertal: stage 1 (n=18)	1.85	7.40*	1.64	9.08*
Early puberty: stages 2 or 3 (n=14)	2.81	8.20*	2.23	7.14*
Late puberty: stages 4 or 5 (n=10)	3.32	4.50†	2.87	3.09†

*p<0.0001, †p=not significant.

patient whose growth appears to be suboptimal is referred to a joint paediatric inflammatory bowel disease/growth clinic every three months, which is staffed by a paediatric gastroenterologist, a paediatric endocrinologist, and a clinical auxologist, to determine the appropriate strategy for managing the problem (for example, surgery) at an early stage.

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- 1 McLain BI, Davidson PM, Stokes KB, Beasley SW. Growth after gut resection for Crohn's disease. *Arch Dis Child* 1990;65:760-2.

Drs Davidson and Beasley comment:

Thank you for the opportunity to reply to the correspondence of Evans *et al*. We agree completely with their comments regarding the relationship of the timing of surgery to puberty on catch up growth in Crohn's disease. Although we acknowledge that we have insufficient patients in advanced puberty to comment on the effect on growth of the stage of puberty, our data would suggest that catch up growth is not limited by the onset (as distinct from stage) of puberty. The one patient in advanced puberty clinically who exhibited catch up growth after surgery did not have his bone age assessed.

Like Evans *et al* we would emphasise the importance of careful ongoing growth measurements in these children. The use of a multidisciplinary team provides optimal management of children with Crohn's disease.

Finally, we are pleased to find that the prospective data of Evans *et al* support our retrospective observation.

SPRING BOOKS

The paediatric departmental library

What should be the priority for the paediatrician when financial considerations reign and access to current literature is still via books and journals? The rate of change in paediatric practice and slowness of publication in journals and especially in textbooks lead to such a rapid obsolescence that departmental libraries

are rarely of use for any in depth study. They provide a source to back up basic case presentations but at a considerable expense. It is impossible to shortlist key titles as every paediatrician has a favourite selection of titles that have proved to be of particular value. These titles have often been selected because the paediatrician has been asked to review that particular book or because a particular subject caught his/her eye from browsing through the unsolicited mailed catalogues or at bookshops at conferences. Most medical schools are persuading departments to adopt a centralised library system for the sake of more efficient lending schemes and security. So what does the paediatrician provide for the juniors who rarely have time to reach the central and sometimes distant library?

There must be the equivalent of a paediatric encyclopaedia covering most common subjects, such as the *Textbook of Paediatrics* by Forfar and Arneil or Nelson's *Textbook of Pediatrics*. Then a selection of system or disease based textbooks should be available. The exact proportion of the subjects will depend on the case mix of the unit. Most general paediatricians will have these books but whether they can be maintained in their current editions is unlikely at present. A cheap but effective way of maintaining a more up to date reference for junior staff is to organise a file of review articles from paediatric journals or free journals such as *Hospital Medicine*, *Medicine International*, *Update*, *Hospital Update*, *Prescriber's Journal*, and *Drugs and Therapeutics Bulletin*. The difficulty is the archiving and security. However the availability of photocopiers has made this easier, although care must be taken to avoid infringing the copyright laws. Another very useful practice is to incorporate a photocopy of a key article in the hospital notes of the patient with this particular diagnosis. It is important to keep updating this and to include this article in the departmental 'useful literature' file. This filing system is unlikely to succeed unless it is clearly one individual's responsibility and that should be at fairly senior level.

However, we are on the brink of the breakthrough in data retrieval which has had the Medline searches and now the CD ROMs as forerunners. When the computer terminal is as familiar and essential an item on the desk in consulting room and ward alike, we will be able to access up to date original articles and learned reviews at the touch of a few buttons. I can foresee the time when as the medical details (plus accounting data) is entered into the consulting room computer an automatic search will be made for relevant new literature on that child's condition. A summary of this will be available for inclusion in the word processed report that will be available for any of the child's professionals to access, as well as for inclusion in the hardcopy that the parents will be carrying. So the medical library will pass into the mists of memory as the stockmarket runners have and as the filing rooms in hospital will.

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A Paediatric Vade-Mecum. 12th Ed. Edited by Jack Insley. 12th Ed. (Pp 296; £13.95 paperback.) Edward Arnold, 1990. ISBN 0-340-52693-9.

Who needs a Filofax when he can have the vade-mecum? This remarkable little book must have a greater concentration of paediatric