Correspondence

to the unrecognisable sudden onset fever (4% in his experience).\textsuperscript{2} Hence the recurrence rate of Knudsen (mean 12\%) is somewhat higher than possible and considerably higher than mine (4\%).\textsuperscript{3} I think that in everyday practice the signals for starting diazepam administration must be less dictated by a protocol and more personalised, so enabling the complete efficiency of this method. Those signals I have used since 1973 for oral prophylaxis (I reserve rectal administration for home treatment of overt seizures) are in my experience very useful. The symptoms can be as trivial as: sudden excessive thirst, listlessness, changes of mood, reduced scrotal turgidity, halitosis, yawning, feeling cold (without shivering), etc.\textsuperscript{3} They are variably assembled in the single child, well known to his mother,\textsuperscript{3} and are nothing more than a short preceding step in the way of discovering the stated fever.

Paediatricians must be grateful to Knudsen for showing once again that the disturbing recurrences of simple febrile convulsions are preventable by intermittent treatment with diazepam and for re-stressing also the invaluable importance, to this purpose, of doctor-parents cooperation.\textsuperscript{1,3}

\textbf{GIANFRANCO DIANESE}
\textit{Divisone Peditrca,}
30027–San Dona' di Piave, Italy

Dr Knudsen comments:
I am grateful to Dr DiAnese for raising this important question. I agree that short term diazepam prophylaxis should be as personalised as possible, but it is equally important that the number of diazepam doses given to the child is kept as low as possible. In our clinic the parents are told to be aware of symptoms of illness in their children and take their temperature if necessary but not to let the thermometer rule the family. The parents are informed both verbally and in writing that recurrent febrile convulsions often take place very early in the feverish illness and that the first dose of diazepam is the most important one and should be given as early as possible, but only when the temperature is above 38.5°C. The method of prophylaxis proposed by Dr DiAnese will—according to my data—only result in a minor therapeutic gain (4\%), as compared with the conventional one. After rectal administration in solution diazepam is absorbed very rapidly, and the anticonvulsant concentration is reached within 2–4 minutes. From a pharmacokinetic point of view oral administration may justify more early prophylaxis, because anticonvulsant plasma concentration of diazepam may not be reached so rapidly, especially when we are dealing with feverish children. To start prophylaxis on the vague, initial symptoms mentioned by Dr DiAnese might result in overtreatment when administered by anxious mothers. I agree that a high degree of parental compliance is probably the most essential prerequisite for effective prophylaxis.

References
\textsuperscript{1} Knudsen FU. Recurrence risk after first febrile seizure and effect of short term diazepam prophylaxis. \textit{Arch Dis Child} 1985;60:1045–9.

Weight gain and height velocity during prolonged remission from acute lymphoblastic leukaemia

Sir,

We have read with great interest an article on weight gain and height velocity during prolonged first remission from acute lymphoblastic leukaemia.\textsuperscript{1} The authors claim that none of the previous studies on growth after acute lymphoblastic leukaemia has reported data on weight gain. It might therefore be appropriate to point out that in our study of 27 children treated for acute lymphoblastic leukaemia\textsuperscript{2} a similar phenomenon of excessive weight gain during the first year after stopping treatment was observed. This resulted in a higher than normal attained weight at four, five, and six years after the onset of disease; later on, however, weight became normal. Weight gain during the maintenance therapy observed by the Cardiff group\textsuperscript{3} might be explained by the cumulative weight gain from recurrent steroid pulses. Our patients were treated without 'reinductions'; and no such weight gain was noticed. The weight gain after stopping of treatment is more difficult to explain. In some of our patients it was so excessive that central nervous system leukaemia with hypothalamic obesity was suspected, but never proven.

In the analysed material\textsuperscript{1} there was no difference in weight gain between those children who had received cranial irradiation and those who had not. It is therefore unlikely that a hypothalamic damage induced by irradiation is responsible for the weight gain. We are, as the authors of the report, inclined to believe that psychological factors are likely to lead to weight gain. An additional factor may be the stopping of treatment with methotrexate, a drug that often causes malabsorption during the maintenance therapy.\textsuperscript{2} When enteropathy is no longer present excessive consumption of high energy foods contributes to relatively greater weight gain.

\textbf{CHRISTIAN MÖLL, STANISLAW GARWICZ, ULF WESTGREN, AND THOMAS WIEBE}
\textit{Department of Pediatrics, University Hospital, S–221 85 Lund, Sweden}

Drs Sainsbury, Newcombe, and Hughes comment:
We are grateful to Dr Möll and his colleagues for drawing our attention to their paper on weight gain in 27 children treated for acute leukaemia.\textsuperscript{2} The results from both studies show excessive weight gain after the stopping of treatment. We agree that stopping all cytotoxic treatment together with continued excessive food intake is the probable cause.
Chronic lung disease—home management with an oxygen concentrator

Sir,

The reduced mortality seen in ventilated very low birthweight infants has led to an increase in the incidence of chronic lung disease and the need for prolonged supplemental oxygen. Such infants require lengthy hospital stays, leading to inhibition of their physical and emotional development. Recently, the administration of low flow oxygen through a nasal catheter has been shown to be effective in the home management of these babies, but such a system is dependent on oxygen cylinders. The use of an oxygen concentrator is established in adult practice but has not been described in the management of the chronically oxygen dependent infant.

A survivor of a twin pregnancy born at 25 weeks' gestation was discharged from hospital after a prolonged and complicated stay, assisted ventilation having been necessary for a number of months. Low flow continuous oxygen was begun through a size 6FG feeding catheter, and after a period of adjustment based on clinical and transcutaneous oxygen monitoring he was discharged home on 0.5 l/minute. No hospital admissions have been necessary for the last five months and developmental catch up is being achieved. Supplemental oxygen was stopped after 161 days.

As the 'Concentrator' (DeVilbiss DeVo/44 Oxygen Concentrator, DeVilbiss Health Care Division, Feltham, Middlesex) is on wheels it could be moved easily about the house, although with suitable lengths of oxygen tubing this became unnecessary. The changing of filters was undertaken by the parents, who found this system more acceptable than one requiring cylinders. A small back up oxygen cylinder was needed, however, for possible breakdowns and hospital and social visits.

The cost of an oxygen concentrator is considerable (£966 excluding VAT); nonetheless its capital cost, installation, and maintenance would be offset after 75 days of continual use when compared with the average cost of cylinder oxygen at 68 pence/hour. In our patient there was a saving of roughly £1400 over a five month period.

I suggest that oxygen concentrators should be considered for the home management of oxygen dependent infants not only because of their convenience but also from the considerable savings resulting from their use.

References


DAVID J LLOYD
Department of Neonatal Paediatrics, Aberdeen Maternity Hospital, Aberdeen AB9 2ZA, Scotland

Secular trends in head growth

Sir,

The paper from Dr Ounsted and her colleagues1 prompted us to examine whether secular trends in head growth had also taken place in Hong Kong children.

Two sets of data from children aged 7 to 18 years were compared. The first was a mixture of cross-sectional and longitudinal data collected between 1961 and 1965 by Chang and his colleagues of several thousand children from schools and colleges in Hong Kong.2 (These data, along with those for weight and height, continue to be widely used as growth reference standards in Hong Kong.) The second were very recently published data from Fung and his colleagues from about 2500 normal children from schools on Hong Kong island and the Kowloon peninsula.3 There is a pronounced positive secular trend in both sexes. In boys, this difference is greatest between 10 and 13 years: in girls between 7 and 10 years. The pattern of head growth is different in both sexes with girls reaching their maximum head growth velocity before boys in keeping with Oun-