

to the unrecognisable sudden onset fever (4% in his experience).² Hence the recurrence rate of Knudsen (mean 12%) is somewhat higher than possible and considerably higher than mine (4%).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.³ They are variably assembled in the single child, well known to his mother,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 distressing 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.¹⁻³

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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 data¹—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

- ¹ Knudsen FU. Recurrence risk after first febrile seizure and effect of short term diazepam prophylaxis. *Arch Dis Child* 1985;**60**:1045–9.

² Knudsen FU. Effective short-term diazepam prophylaxis in febrile convulsions. *J Pediatr* 1985;**106**:487–90.

³ Dianese G. Treatment of febrile seizures. *J Pediatr* 1980;**96**:516.

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.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 leukaemia² a similar phenomenon of excessive weight gain during the maintenance therapy observed by the Cardiff group¹ 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 material¹ 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 radiation 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.³ When enteropathy is no longer present excessive consumption of high energy foods contributes to relatively greater weight gain.

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Drs Sainsbury, Newcombe, and Hughes comment:

We are grateful to Dr Moëll and his colleagues for drawing our attention to their paper on weight gain in 27 children treated for acute leukaemia.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.