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Comment

to: Hyllienmark L, Maltez J, Dandenell A, Ludvigsson J, Brismar T (2005) EEG abnormalities with and without relation to severe hypoglycaemia in adolescents with type 1 diabetes. *Diabetologia* 48:412–419

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To the Editor:

I read with great interest the study by Hyllienmark et al. [1] on the risk factors for EEG abnormalities in adolescents with type 1 diabetes. Recurrent severe hypoglycaemia and poor metabolic control contribute to EEG abnormalities characterised by an increase in slow activity (delta and theta) and a reduction in alpha peak frequency.

We reported similar results 26 years ago [2], even though the methods we used were simplistic in comparison with those employed by Hyllienmark et al. We found that the incidence of abnormal EEGs was higher in patients with poor metabolic control. Moreover, using fluorescein angiography, which has high sensitivity, we demonstrated a clear correlation between incipient retinal angiopathy and EEG abnormalities. Eighty percent of our patients who experienced more than five severe hypoglycaemic attacks showed evidence of abnormal EEG. On the other hand, minor hypoglycaemic episodes had no effect on the EEG. The EEG abnormalities were classified as diffuse or focal. Paroxysmal sharp waves, spikes and spike-and-wave forms, as well as bursts of slow wave activity (theta or delta), were regarded as paroxysmal. Among the abnormal EEG patterns, six were diffuse (non-rhythmic, slowing)

and nine were paroxysmal (spike-and-wave in five, spikes in one, sharp waves in one, and bursts of delta waves in two). There were no focal abnormalities.

These EEG abnormalities are not necessarily related to cognitive dysfunction or impaired intellectual development in children and adolescents. Schoenle et al. [3] showed that long-term bad glycaemic control, rather than hypoglycaemic attacks, is a risk factor for impaired intellectual development. According to the DCCT [4], to avoid long-term complications, patients with type 1 diabetes must maintain blood glucose concentrations close to the normal range; this increases their risk of severe hypoglycaemia, but without changes in neurophysiological function. Thus, fear of hypoglycaemic attacks is not a good argument to stay in a bad metabolic control, even in children. Moreover, the Hvidore Study Group on Childhood Diabetes has shown that some centres are more successful than others at preventing severe hypoglycaemia, independent of the prevailing average HbA_{1c} level at the respective centre [5]. We have reported that in 144 unselected diabetic children and adolescents aged <18 years, with a mean HbA_{1c} level of 6.6% (upper normal limit: 5.5%) [6], the yearly incidence of severe hypoglycaemia was 0.2, which is three times less than in the intensive-therapy group of the DCCT [3].

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