Letters to the editor

‘Neuropsychological deficiencies as a mediator between CNS dysfunction and inattentive behaviour in childhood epilepsy’

SIR—A recent paper in ‘Developmental Medicine and Child Neurology’ presented evidence for an increased comorbidity of childhood epilepsy with attention-deficit–hyperactivity-disorder (ADHD), predominantly of the inattentive type. The authors suspect that the underlying neurological dysfunction may cause both seizures and attention problems with the precise pathways remaining unclear. Clarifying mediators connecting central nervous system (CNS) dysfunction to inattentive behaviour in childhood epilepsy, however, may be an important next step in tailoring more specific intervention.

We suggest that evidence from neuropsychological research both in epilepsy and in ADHD can contribute to a more profound understanding of the valuable data provided. We recently performed a study on neuropsychological functioning in a sample of children with epilepsy characterized by an identical sample size (n = 175), and very comparable clinical features. We identified significant deficiencies in sequential cognitive information processing (SEQ), assessed via the Kaufman Assessment Battery for Children (K-ABC), as a specific neuropsychological deficit present across all subtypes of epilepsy and levels of intelligence. Deficits in SEQ predicted best reduced performance in the Achievement Scale of the K-ABC and thus learning problems. Impairment of SEQ comprises deficiencies in selective and sustained attention and in the short term, respectively working memory functioning. Our results are in line with the finding of selective attention deficit in childhood epilepsy assessed via a computerized neuropsychological test of reaction times.

Interestingly, recent studies on neuropsychological functioning in ADHD have provided corresponding evidence for equivalent deficiencies concerning attention and memory functioning in ADHD. Working memory dysfunction is one of four major factors involved in ADHD according to the theory of Barkley. Despite the different neurological aetiologies of epilepsy and ADHD, both conditions share common neuropsychological impairments leading to observable inattentive behaviour. We therefore assume that the specific neuropsychological impairments in childhood epilepsy act as a mediator between the underlying neurological aetiology and the symptom of inattention and are presumably responsible for the increased prevalence of inattentive behaviour.

In the face of the high general prevalence of ADHD among children and adolescents in the normal population we have to differentiate between concurrent versus successive comorbidity of epilepsy and ADHD. Concurrent comorbidity refers to the aetiologically independent coincidence of both disorders. Following the data provided by Dunn et al., most children with the hyperactive or combined type of ADHD will belong to this kind of ‘true’ comorbidity. Successive comorbidity refers to the inattentive behaviour secondary to deficient attention and memory functioning on the basis of CNS disturbance in childhood epilepsy. Despite different trajectories of pathogenesis, the clinical pattern of inattentive behaviour may look very similar among children with concurrent versus successive comorbidity, reflecting an example for the principle of equifinality in developmental neuropsychology and psychopathology (i.e. similarity in outcome despite differences in aetiology).

For clinical reasons, differentiation of the aetiological source of inattention in affected children with epilepsy (e.g. by exploring the temporal sequence of manifestation of epilepsy and ADHD), however, may have important implications for treatment strategy. Inattention mediated by neuropsychological impairment due to the origin of epilepsy may require, in the first line, efforts to optimize antiepileptic therapy with special consideration of neuropsychological and hence behavioral outcome parameters. In case that inattention is resulting from concurrent comorbidity, a combined approach of antiepileptic treatment and ADHD therapy is required. Potential interaction effects between stimulant and antiepileptic medication (e.g. reduction of seizure threshold) should be monitored but are not regularly to be expected. In both cases, patients, respectively parents should be informed and counselled on the suspected origin of inattentive behaviour development in the individual case to improve the understanding for the specific intervention strategy chosen.

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References
References

SIR–Comments on the letter by Dr Al-Dahhan and his colleagues regarding neonatal hyponatraemia with reference to our article ‘Developmental risks and protective factors for influencing cognitive outcome at 5½ years of age in very-low-birthweight children’. At the end of 1980s, when we started our study, frequent check-ups from the day of birth and necessary supplementation for hyponatraemia (<130mmol/l) were already routine in our clinical practice. Therefore, hyponatraemia was not included as a risk factor for adverse neuropsychological development in the preterm born children.

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‘Böhm et al. reply’

Developmental risks and protective factors for influencing cognitive outcome at 5½ years of age in very-low-birthweight children

SIR–One possible risk factor for poor cognitive outcome not mentioned in the study by Böhm et al.1 is neonatal hyponatraemia, a common event in very-low-birthweight infants unless appropriate salt supplements are given.2,3,4 In a recently published study5 we showed that very-low-birthweight infants who had received salt supplements from the 4th to the 14th postnatal day had significantly better neurodevelopmental outcome at 10–13 years of age than otherwise similar infants who had not been so supplemented, and in whom the incidence and severity of hyponatraemia was therefore greater.2 In another study,6 hyponatraemia was found to be a risk factor for cerebral palsy (odds ratio 6.8, range 1.9–24.2, compared with infants without a history of hyponatraemia — a higher odds ration than that of any other variable studied). Additionally, hyponatraemia and aminoglycoside exposure were identified7 as the two most significant risk factors for sensorineural deafness in preterm infants. These findings are consistent with the results of numerous experimental animal studies in which sodium depletion during early development impairs brain growth, as well as that of other organs.8 It would be interesting to know whether hyponatraemia was an adverse risk factor for cognitive function in the infants studied.1 If this question was not addressed, as seems likely, perhaps it would be possible to extract the necessary information from the records in order to determine whether infants who had hyponatraemia (at least one plasma sodium concentration <130mmol/l) had a significantly poorer outcome than those who did not. The question is of more than academic interest because, unlike some of the other risk factors identified,1 hyponatraemia is a potentially preventable complication of extreme prematurity.

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