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Language in benign childhood epilepsy with centro-temporal spikes abbreviated form: Rolandic epilepsy and language

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Abstract

Although Benign Childhood Epilepsy with Centrotemporal Spikes (BECTS) has a good prognosis, a few studies have suggested the existence of language disorders relating to the interictal dysfunction of perisylvian language areas. In this study, we focused on language assessment in 16 children aged 6–15 currently affected by BECTS or in remission. An important proportion of children showed moderate or more severe language impairment. The most affected domains were expressive grammar and literacy skills. We found linguistic deficits during the course of epilepsy but also persistent deficits in children in remission, suggesting possible long-term effects. Our results support the hypothesis that BECTS may be associated with impairment to language and suggest the possibility of a direct link between epileptic activity and language development, and the existence of long-term consequences.

Keywords: Benign Childhood Epilepsy with Centrotemporal Spikes; Rolandic epilepsy; Language; Morphosyntax; Literacy skills; Critical period; Active phase; Remission; Long-term consequences

1. Introduction

The study of the links between epilepsy, language, and cognitive functions is complex, because of the existence of many combined factors such as neurobiological, pharmacological, and psychosocial factors (Tuchman, 1994). Gordon (2000) reviewed the effects of epilepsy on cerebral functions with particular attention to language and concluded that language disorders were currently established in the Landau–Kleffner syndrome (LKS) and in the Continuous Spike Wave in Slow Wave Sleep syndrome (CSWSS). The impact of epilepsy on language development in partial epilepsy with left frontal focus was shown as well by Cohen and Le Normand (1998) who found persistent language disorders in six right-handed children with partial epilepsy (left frontal focus). On the other hand, concerning the role of epileptic discharges on language in Benign Childhood Epilepsy with Centrotemporal Spikes, a frequent conclusion has been that further research is needed (see, for example, Gordon, 2000).

Benign Childhood Epilepsy with Centrotemporal Spikes (BECTS) is an idiopathic localization-related epilepsy, having its onset between 3 and 13 years. No significant brain lesion is described. Remission occurs before adulthood without systematic anti-epileptic treatment. The long-term medical and psychosocial prognosis is good (Loiseau, 1999; Wirrel, 1997). These distinctive features make it an interesting model to distinguish the effects directly related to the epileptic discharges themselves from effects related to cerebral lesion, treatment

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or psychosocial factors (D'Alessandro et al., 1990). As epileptic discharges are localized in the central or mid-temporal regions, BECTS is a suitable model to investigate the links between epileptic activity and language.

Although the absence of cognitive deficit is one of the criteria of BECTS (ILAE, 1989; Loiseau, Duché, & Cohadon, 1992), some studies have shown the existence during the course of epilepsy of neuropsychological impairment, such as attentional and visuo-motor abilities deficits (Baglietto et al., 2001; D'Alessandro et al., 1990; Heijbel & Bohman, 1975; Metz-Luz et al., 1999; Piccirilli et al., 1994; Weglage, Demsky, Pietsch, & Kurlemann, 1997), executive function and verbal short term memory impairment (Croona, Kihlgren, Lundberg, Eeg-Olofsson, & Eeg-Olofsson, 1999; Gündüz, Demirbilek, & Korkmaz, 1999; Laub, Funke, Kirsch, & Oberst, 1992; Weglage et al., 1997). Moreover, the localization of the epileptic focus in the perisylvian language areas would appear to suggest a specific impairment of this function. However, few studies have widely investigated language in BECTS. Some research has found language disruption such as moderate fluency troubles, naming deficits, and comprehension disorders (Baglietto et al., 2001; D'Alessandro et al., 1990; Gündüz et al., 1999; Laub et al., 1992). Staden, Isaacs, Boyd, Brandl, and Neville (1998) found specific language impairment in 13 of 20 children with BECTS during the active phase, affecting in particular reading, spelling, and expressive grammar. In a case study, Deonna, Roulet, Fontan, and Marcoz (1993) reported three patients with interictal oral dyspraxia, naming troubles, and literacy impairment, and Scheffer et al. (1995) described cases of speech dyspraxia and language dysfunction in a family study with autosomic dominant BECTS.

These results question the benign nature of this epilepsy, and suggested a direct link between paroxysmal anomalies and cognitive functions. Some studies support this hypothesis and have shown that interictal discharges could lead to transient cognitive impairment, dependant on the nature of the task (verbal vs visuospatial), according to the localization (left vs right) of the epileptic focus (Binnie, 1993, 1996).

Others studies have maintained the hypothesis that paroxystic anomalies may play a direct role on cognitive function, by studying the link between lateralized hemispheric function and topography of the epileptic focus. D'Alessandro et al. (1990) showed, in a group of 44 children assigned to three subgroups according to side of EEG focus (right, left, and bilateral), that patients with left focus performed worse on verbal tasks (naming, fluency, and comprehension). A few studies, using a dual-task procedure (Kinsbourne & Cook, 1971), have also suggested that left paroxysmal rolandic abnormalities could influence the cerebral functional lateralization for language, in right-handed children with left focal BECTS (Piccirilli, D'Alessandro, Tiacci, & Ferroni, 1988) and in right-handed young adults in remission of left focal BECTS (Hommet et al., 2001).

On the other hand, concerning the long-term outcome of cognitive functions in BECTS, most studies have concluded that neuropsychological deficits observed in the active phase disappeared when the patients were in remission for epilepsy (Baglietto et al., 2001; D'Alessandro et al., 1990; Metz-Luz et al., 1999). However, the study of Scheffer et al. (1995) mentioned above found persistent language deficits in adulthood in several patients after seizures had subsided (oral and speech dyspraxia, reduced speed of processing on complex structures such comparative and passive grammatical relation ships, naming deficit, literacy).

The aim of our study was to determine if patients with BECTS show identifiable language impairment, which domains are affected and if there is a link between clinical factors—such as lateralization of epileptic focus, age of epilepsy onset—and language disorders. Additionally, we expected that we might find critical period effects on language development. It is commonly held that active language development occurs within a limited time frame ending around age six or seven (Johnson & Newport, 1989, 1991; Lenneberg, 1967; Weber-Fox & Neville, 1996). During this sensitive period, it is a fact that circuits are establishing as a substrate for language (Gordon, 2000): the presence of epileptic activity within these areas specific to language may disturb parts of the network and these may be permanent as the critical period has passed. Therefore, we hypothesized that deficits observed might differ according to the age of onset of epilepsy and to the duration of its active phase. In addition, we wanted to determine if persistent language deficits can be found after remission of epilepsy.

2. Material and method

2.1. Subjects

All subjects from the Neuropediatric Unit of the university hospital of Tours (Hôpital Gatien de Clocheville) who fulfilled the ILAE (1989) criterion of BECTS were recruited. All patients were evaluated within the framework of follow-up visits with parental consent. The patients (6 boys and 10 girls) were 6–15 years old (mean age: 12 years 2 months). The initial epileptic focus was determined using an awake EEG for nine children, combined with night polysomnography for seven patients. Six patients had a bilateral focus, five a left focus, and five a right focus. Three children had never been treated, six still had treatment (carbamazepine or valproic acid), and seven had stopped it. Seven children fulfilled the criterion of remission: absence of seizure or EEG abnormalities for at least one year after treatment interruption. Handedness was right for nine children and