

Epileptiform activity in children with developmental dysphasia: quantification of discharges in overnight sleep video-EEG

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ABSTRACT – We present results of analysis of overnight sleep video-EEG in 8 patients with developmental dysphasia and rolandic discharges. We evaluated the incidence of epileptiform discharges (expressed as paroxysmal activity density) at one or more electrodes in different sleep stages in three different periods of the night (after falling asleep, around midnight and before awakening). The difference of paroxysmal activity density was never higher than 21%, indicating that quantifying the discharges in the whole night recording is not necessary. We also showed that two independent foci may differ in the frequency of discharges. We propose a scheme for evaluation of EEG reflecting both frequency and distribution of discharges.

Key words: developmental dysphasia, sleep EEG, epileptiform activity

Developmental dysphasia (DD) is defined as a disrupted ability to acquire normal language skills adequately to age, with intact peripheral hearing, normal intelligence and absence of gross sensori-motor deficit or orofacial malformation and absence of behavioral disorder or negative social factors. Terms Developmental Language Disorder (DLD) and Specific Language Impairment (SLI) are also used in the literature. The exact diagnostic criteria are lacking. The impairment of language sometimes overlaps with other developmental disorders.

Several authors reported higher incidence of epileptiform discharges in children with dysphasia (Tuchman *et al.* 1991; Echene *et al.* 1992; Duvelleroy-Hommet *et al.* 1995; Picard *et al.* 1998; Chéliout-Hérault *et*

al. 1999). The incidence of discharges ranges from 9 to 94%. The degree of EEG abnormality varies from focal sporadic discharges to generalized epileptiform activity fulfilling the criteria of ESES (electric status epilepticus in slow sleep). The reported samples differ in age structure and inclusion criteria (strictness of diagnostic criteria and inclusion of children with history of epileptic seizures). The used methodology plays an important role: wake EEG or overnight sleep EEG, number of evaluated channels, recording on one or several occasions. With respect to EEG characteristics, authors refer to “paroxysmal activity”, “epileptiform activity” or “epileptiform abnormality”. The type of discharges is not always well defined, e.g. whether the described activity had a form of benign

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sharp waves. In the population of healthy children the incidence of this type of sharp waves is reported to be lower than 2% (Dooze, 2003).

The important question is whether the epileptiform activity has negative effect on language development, whether it is the primary cause of dysphasia or an epiphenomenon.

There is a lack of evidence on correlation between EEG abnormality and severity of speech disorder. The opinions on using antiepileptic medication are not unified.

Some authors propose using antiepileptic medication according to the frequency of discharges. However, the distribution of discharges is not usually taken into consideration. The exact evaluation of whole night EEG records is extremely time consuming and its relevance either for clinical or for experimental use is questionable. Searching for relationships between epileptiform activity and language impairment is a significant challenge.

In this paper we present preliminary results of our ongoing study on overnight sleep video-EEG in children with developmental dysphasia. We aimed to evaluate intraindividual variability of epileptiform activity in the course of the night and between different sleep stages. Furthermore, our aim was to suggest effective way of quantification of epileptiform EEG respecting both frequency and distribution of discharges.

We present 2 case reports of children followed prospectively from the age of 2 years and 10 months, resp. 3 years and 3 months. We also compare the incidence of epileptiform activity in our sample with studies by other authors and discuss the impact of the overnight sleep video-EEG on clinical practice.

Methods

The population was 35 children referred for suspected DD by our outpatient child neurology centre, community neurologists and speech therapists. Twenty eight of them (age: 3 years 5 months – 9 years 1 month, mean 5 years 6 months) fulfilled the diagnostic criteria of DD. Epileptiform discharges in the whole-night sleep video-EEG were found in 11 of these children. In 10 cases the discharges had typical 5-component structure of focal sharp waves typical for benign idiopathic epilepsies (Dooze, 2003), one patient had biphasic sharp waves. In wakefulness, the discharges were present in 10 patients. Now we present a detailed analysis of EEG of 8 children (age: 4 years 6 months – 6 years 8 months, average 5 years 4 months, 4 boys/4 girls). We only evaluated EEG records with whole night data available. In one case, not presented in detail, there were only sporadic discharges at Cz during sleep (with density lower than 1%), they were not present in wakefulness.

Neurological examination showed no abnormality. Motor milestones were within normal range. None had hearing

impairment. One patient had a history of febrile convulsions but no child had experienced afebrile seizures. None was receiving antiepileptic medication. No child was diagnosed with pervasive developmental disorder or other dominating behavioral problems. None had a history of language or other cognitive regression.

Psychological evaluation

Children were assessed by clinical psychologist, using Gesell developmental diagnosis (Knobloch *et al.* 1980), Stanford-Binet Intelligence Scale-IV revision (Thorndike *et al.* 1995) and an additional test standardized for Czech language consisting of sound differentiation test, world differentiation test, auditory analysis and synthesis test (Matejcek, 1993). Spontaneous talk was also evaluated. Children were included if PIQ ≥ 70 , there was disturbed phonemic discrimination and/or disturbed language on various levels – phonologic, syntactic, lexical, semantic and pragmatic.

EEG

Overnight sleep video-EEG monitoring was performed using 19 channels with international 10-20 electrode placement in each child on the second night of stay. The duration of the recording was 9-16 hours, including at least 1 hour of wakefulness.

By visual inspection of all records discharges were classified according to the criteria proposed by Saltik *et al.* (2005) for idiopathic partial epilepsies with evolution to ESES. We only applied the criteria for localization and spreading of epileptogenic foci, and evaluated only the discharges occurring in sleep:

- 1) single epileptogenic focus (SEF),
- 2) bilateral synchronous epileptogenic foci (BSEF),
- 3) bilateral asynchronous epileptogenic foci (BAEF),
- 4) multiple epileptogenic foci (MEF),
- 5) spreading focal epileptogenic discharges (SFEDs),
- 6) focal slowing (in this study we did not evaluate this abnormality).

By visual inspection of recordings, for each patient we selected the channel with the highest frequency and the highest amplitude of discharges. In these channels the presence of epileptic discharges was quantified as a paroxysmal activity density (Autret *et al.* 1987), here referred to as PAD. PAD is expressed as percentage of seconds containing paroxysmal activity over a given time period. We counted the number of seconds containing spikes at each 10-second EEG page, then we averaged this number over one minute intervals (6 consecutive EEG pages), and then over the given time periods as defined below. For 2 patients, we selected additional channels. The discharges were counted using source derivation montage.

For each patient the PAD was evaluated in three one-hour long periods of EEG recording representing different night times (these will be referred to in the text as “different night

Table 1. Sleep characteristics of evaluated night time intervals and classification of discharges.

Cases	No. 1	No. 2	No. 3	No. 4	No. 5	No. 6	No. 7	No. 8	
Classification of discharges	SFEDs	SFEDs	SEF	SFEDs	SFEDs	MEF (sec. bilat. synchronization)	BAEF	BAEF	
Focus 1	T5	C4	P4	Cz, Pz	Cz	C3, P3, F7	T3, C3, F7	T6, P4, Pz, Cz, F8	
Spreading	P3, O1	F4, T4, T6, Cz, Pz		C3, C4, P3	F3, C3				
Focus 2						O1	O2, T6, P4	O1, P3, T3	
Focus 3						C4, P4, T6			
Characteristics of selected night time intervals									
1. Starting 10-30 min after falling asleep	NREM I-II min (%)	18 (30)	15 (25)	8 (13.3)	0 (0)	20 (33.3)	0 (0)	43 (71.7)	x ^a
	NREM III-IV min (%)	42 (70)	45 (75)	52 (86.7)	60 (100)	40 (66.7)	60 (100)	17 (28.3)	x ^a
2. Starting between 23:30-00:30	NREM I-II min (%)	38 (63.3)	40 (66.7)	38 (63.3)	24 (40)	28 (46.7)	45 (75)	46 (76.7)	x ^a
	NREM III-IV min (%)	22 (37.7)	20 (33.3)	22 (36.7)	36 (60)	32 (53.3)	15 (25)	14 (23.3)	x ^a
3. Starting between 4:000-5:000	NREM I-II min (%)	54 (90)	32 (53.3)	60 (100)	38 (63.3)	60 (100)	20 (33.3)	60 (100)	x ^a
	NREM III-IV min (%)	6 (10)	28 (46.7)	0 (0)	22 (36.7)	0 (0)	40 (66.7)	0 (0)	x ^a

^a In this patient we were not able to differentiate the sleep stages.

time intervals"): 1) *beginning of sleep* (starting 10-30 minutes after falling asleep), 2) *middle of the night* (starting \pm 30 minutes around midnight and 3) *early morning* (starting between 4-5 am). These periods were not continual in most cases, as we excluded pages containing artefacts, awakenings or REM sleep.

In each period we scored sleep stages NREM I-II and NREM III-IV (these will be referred to in the text as "sleep stages"), with exception of patient No. 8, whose sleep structure was disturbed to such an extent that we were unable to clearly differentiate the sleep stages. The sleep characteristics of the selected time periods are detailed in *table 1*.

In each individual, we first compared the PAD between the three different night time intervals, then between NREM I-II and NREM III-IV averaged from all three intervals. Finally, we averaged number of seconds containing discharges in different night time intervals for each sleep stage separately.

Results

This section summarizes the main findings from the analysis of 8 overnight video-EEGs taken and evaluated in our centre as part of this study. Summative descriptions of

EEGs records with respect to the aims of our study are presented in the first instance, followed by two case reports.

Classification of discharges

According to Saltik's criteria, we found the following types of discharges: SEF was found in 1 case, 4 patients had SFEDs, 2 patients were classified as having BAEF, and 1 patient had MEF with secondary bilateral synchronization. For classification and location of discharges in each patient, see *table 1*.

Paroxysmal activity density

For each of the eight patients we counted PAD in three different night time intervals and in different sleep stages. PAD in different 1-hour intervals varied from the minimum of 23.5% in case No. 1 to 65.1% in case No. 8. In the overall NREM I-II, PAD varied between 22.4 and 42%. In the overall NREM III-IV, PAD varied between 27.8 and 52.3%. These results are detailed in *table 2*.

The box plots in *figure 1* show time intervals (in seconds) containing discharges averaged over 1 minute (not expressed in %) for each patient in different night time intervals for each sleep stage separately. The durations of these periods are detailed in *table 1*.

Table 2. PAD in different 1 hour long night time intervals and in different sleep stages (taken from all three night time intervals). In patients No. 1 and No. 7 two EEG channels were evaluated.

PAD (%)	No. 1		No. 2	No. 3	No. 4	No. 5	No. 6	No. 7		No. 8
	T5	P3	C4	P4	Cz	Cz	P3	T3	O2	T6
1. Starting 10-30 min after falling asleep	26.6	16.5	45.6	25	53.4	41.7	45.9	45.3	47.6	61.1
2. Starting between 23:30-00:30	26.8	20.6	40.5	27.4	48.9	30.7	24.9	28.1	29.8	65.1
3. Starting between 4:00-5:00	23.5	15.1	46.3	26.2	34.9	28.4	32.5	38.4	30.9	51
NREM I-II min	25	17.1	42	25.2	33.2	28.1	22.4	37.7	34.2	X
NREM III-IV min	27.8	18.2	46.2	27.7	52.3	41.9	41.2	35.1	45.3	x

In comparison to the rest of the group, patients No.1-3 presented smaller variability of PAD within the whole night course as well as between the sleep stages. These are all cases of single epileptogenic foci with or without spreading.

The patients No.4-6 presented higher degree of variability when comparing different periods of the night or different sleep stages. The difference in PAD did not exceed 21% in any case. All 3 patients had higher PAD in NREM III-IV. Two patients had focal discharges with spreading, one had multifocal discharges.

The patient No. 7 (bilateral asynchronous discharges) showed almost equal PAD in both sleep stages (for the channel T3, 37.7% and 35.1%, respectively). PAD in the course of the night showed variability with maximum at the beginning of sleep, minimum in the middle of the night, and then increase again in the early morning.

Patient No. 8, with the highest frequency of discharges (bilateral asynchronous discharges), presented relatively constant PAD with slightly lower value in the 3rd interval. However, the variability within each 60 minute period was high (*figure 1H*).

Comparison of two channels

Patient No. 1 had focal discharges with maximum at T5, spreading to P3 and O1.

PAD at O1 was lower than at T5 at any part of night, regardless of the sleep stage (*table 2*).

On contrary, such tendency is not seen in patient No. 7, classified as having two independent foci (bilateral asynchronous). Here we see that O2 is more affected compared to T5 in NREM III-IV, while the difference between the two channels in NREM I-II is not so evident. We also found that PAD averaged over all three 1-hour periods was actually higher at O2 than at T3.

Case report No. 1

This boy was born from an uneventful pregnancy and delivery, normal motor milestones, and unremarkable family history. Speech therapy was initiated at the age of 3 years as there was an evidence of language delay (not specified as DD). He had generalized clonic seizure during bronchopneumonia in association with fever at the age of 4 years and 7 months.

Evaluation at our centre showed:

- aged 2 years and 10 months,
- sporadic bilaterally asynchronous discharges with centro-temporal foci, maximum at C4, less frequently generalized.

No medication was recommended.

At 5 years DD was confirmed, EEG showed bilateral asynchronous frontocentrotemporal foci, maximal at T3, in NREM I-II continual. Oral diazepam was started.

At 5 years + 3 months (on diazepam), EEG showed no epileptiform discharges.

At 5 years + 7 months, after a slow withdrawal of diazepam, EEG showed bilateral asynchronous FCT foci, sporadically generalized. A simple partial seizure (hemifacial) occurred one week after finishing diazepam.

Case report No. 2

A boy, born from uneventful pregnancy, spontaneous delivery and uncomplicated perinatal period. His father, the father's mother and cousin had delayed speech development (not specified). There was no history of epilepsy. Motor milestones were within normal limits. Until the age of 1.5 years he suffered from frequent otitis media.

On evaluation at our centre, at the age of 3 years and 3 months (9/05), the sleep EEG showed sporadic sharp waves at Fz and Cz bilaterally with focal slowing. Intensive speech therapy was initiated.

At the age of 4 years (for the purpose of the study patient No 8). For detail of the EEG characteristic, see *tables 1, 2* and *figure 1H*. Treatment with diazepam started.

At 4 years 3 months on diazepam discharges completely disappeared

At age 4 years and 7 months, 2 weeks after gradual finishing of diazepam treatment, the EEG in wakefulness and drowsiness showed sporadic generalized SW discharges with maximum at FCT, no focal discharges were seen in either wakefulness or sleep.

Discussion

Investigating epileptiform EEG in sleep in developmental disorders is a significant challenge. Furthermore, methodological differences between published studies investigat-

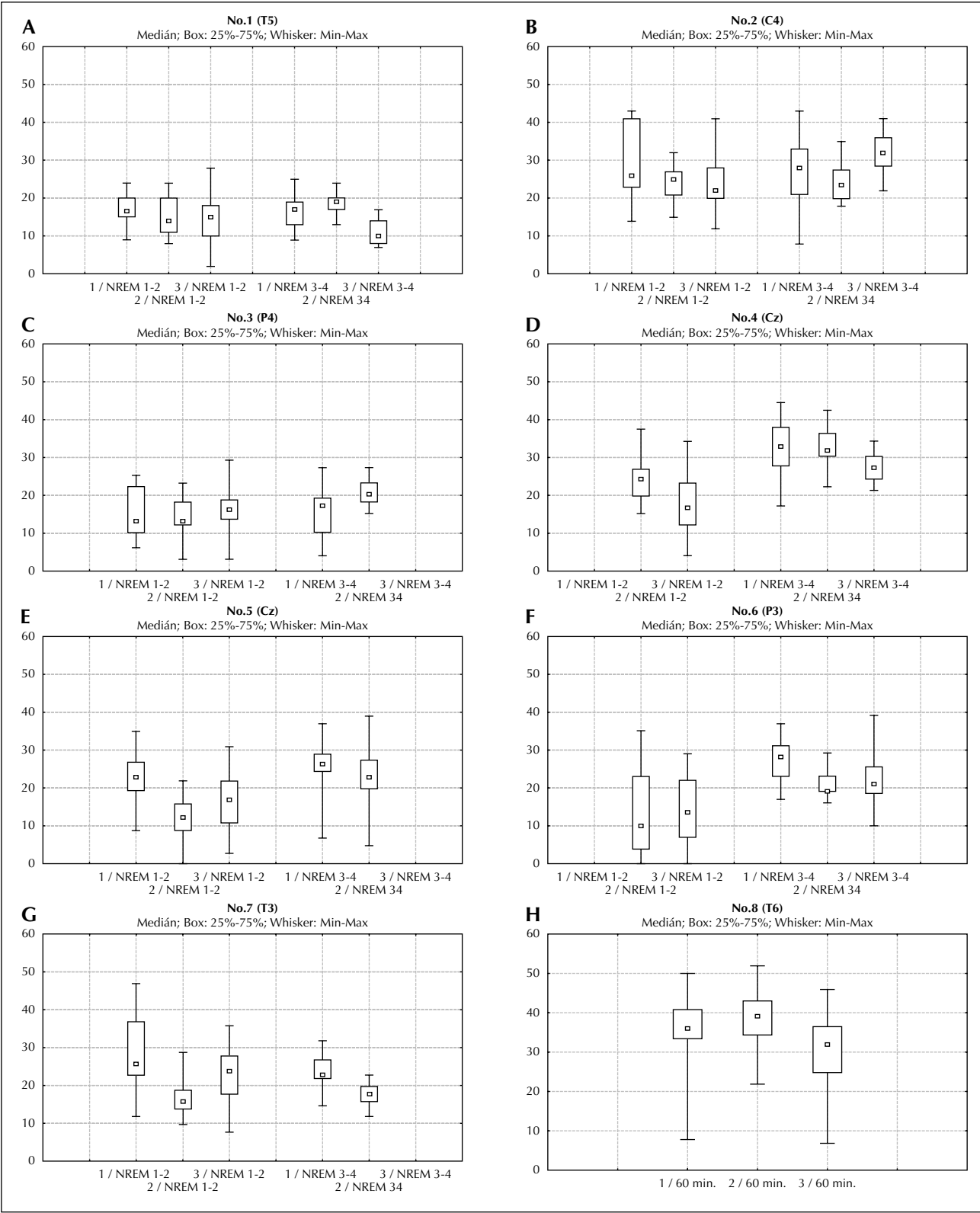


Figure 1. The box plots represent, for each of the reported patients, range of number of seconds containing discharges averaged over 1 minute for NREM I-II and NREM III-IV in different night time intervals separately.

ing epileptiform EEG in sleep in developmental disorders make comparison of results published by various authors almost impossible. In our study, we asked whether it is necessary to read the whole night EEG of children with developmental dysphasia or whether, for clinical purposes, it is sufficient to examine shorter periods of a sleep EEG recording.

Precise criteria defining developmental dysphasia are lacking in the literature. Picard (1998) included in his study children with full IQ above 90. Duvelleroy-Hommet (1995) included only patients with expressive dysphasia, with PIQ equal to or above 80. In both studies a range of verbal tests were used to confirm the diagnosis. We believe that the diagnosis of developmental dysphasia should be distinguished from simple delay of language development (slower development with no deviation from normal speech). The presence of speech disruption at various levels, including morphology, phonology, syntax, lexical semantic and pragmatic skills and disturbed phonemic perception is mandatory for the diagnosis of developmental dysphasia. We assume that children with borderline intellect should not be excluded from studies, if there is marked difference between verbal and nonverbal skills and the speech has typical marks of dysphasia. In our study we included children with PIQ equal or superior to 70. The question is whether it is possible to distinguish "pure" developmental dysphasia and whether epileptiform activity is specific to this particular type of developmental disorder. Various authors highlighted comorbidities of SLI other than epilepsy (Westerlund *et al.* 2002; Selassie *et al.* 2005). Some of our patients also presented with hyperactivity and/or attention deficit. On the other hand, higher rates of epileptiform activity in EEG have been reported in children with ADHD (Holtmann *et al.* 2003) and developmental coordination disorder (Scabar *et al.* 2006). Cognitive and behavioural problems were reported in children with BECTS (see review by Nikolai *et al.* 2006). However, benign rolandic traits have also been described in healthy children by Okubo (Okubo *et al.* 1994). Okubo also showed that these discharges are age-dependent, the foci may be migrating and transition from focal to generalized discharges is possible.

Doose introduced the concept of Hereditary Impairment of Brain Maturation (HIBM) (Doose and Baier, 1989) that unifies the whole range of pathologies: epileptic seizures, typical EEG traits and various developmental disorders as different manifestations of common underlying genetically determined mechanism.

Incidence of rolandic discharges in sleep EEG in our sample of 28 children with developmental dysphasia was 39% and this is within the range reported by other authors. Duvelleroy-Hommet *et al.* (1995) found the discharges in 9 out of 24 (38%) cases, Picard (1998) in 26 out of 52 (50%) cases, and Echenne *et al.* (1992) in 30 out of 32 (93%) cases. We admit certain inclusion bias. The differences in the number of children with discharges in sleep

EEG in various studies might be caused by several factors, such as inclusion of children with epilepsy, age structure of the sample, different diagnostic criteria of developmental dysphasia and different methodology of EEG recording (wake EEG, polysomnography with limited number of channels *versus* 10/20 system). Echenne (1992) prospectively followed 32 children. Thirteen of them had a history of epileptic seizures, and this makes his series incomparable to the one reported in this paper. He found epileptiform activity in overnight sleep in 30 cases. Tuchman *et al.* (1991) found epileptiform abnormality in 58% of dysphatic children with epilepsy and in 9% without epilepsy. Not all their patients had sleep EEG recorded. As we showed in the case report No. 1, in some patients initially considered as non-epileptic, the diagnosis of epilepsy might be confirmed later. In case of a study, this may happen after the study had finished. We assume that excluding children with seizures typical for BECT is not justified.

The relationship between the severity of EEG findings and the severity of language impairment has not yet been clarified. Picard's study indicates that the paroxysmal activity is more often present in children with mostly receptive difficulties. He used polysomnography with only four channels to record the EEG. Therefore, the study brings only limited information on the distribution of discharges. According to our experience, only careful evaluation using source derivation montage can sometimes distinguish real generalised discharges from focal discharges with wide spreading.

Regarding to the widely discussed relationship between epileptiform activity and language impairment, we present evidence in our case reports, that frequency of discharges may worsen from the time when language deficit is already marked (though the precise diagnosis of DD can be established later). This finding speaks against causal relationship. Whether the discharges might have additional negative effect on language development is still unclear.

We retrospectively studied psychological assessments of children with confirmed diagnosis of DD and who had had overnight sleep video-EEG at our centre between years 2000-2005. The VIQ in both groups (with and without discharges) varied from the level of severe mental retardation to almost normal values (in some of the older children after intensive speech therapy). There also was high interindividual variability in PIQ in both groups. This finding does not support the hypothesis about the negative effect of discharges on cognitive functioning. A previous study on children with autism revealed correlation between mental functioning and epilepsy but not epileptiform EEG (Hrdlicka *et al.* 2004).

Sufficient evidence exists to confirm that the incidence of epileptiform activity in children with developmental dysphasia is higher than that of normally developing children. We believe that the overnight sleep video-EEG monitoring (with 10-20 electrode placement) in patients with DD is

important for better understating of the problem. However, the impact of this on the clinical practice remains uncertain. The use of antiepileptic therapy is still controversial. Picard suggested using antiepileptic therapy in children with paroxysmal activity higher than 8% of total sleep time. Duvelleroy-Hommet reserves the treatment only for children with continuous spikes and waves in slow sleep. According to our experience, the medication has beneficial effect in some children. We accept the use of the antiepileptic drugs if the child makes minimal progress even with intensive speech therapy. Parents must be informed about the fact that this treatment is not evidence-based and the effect is not guaranteed. In general, we do not recommend the treatment if the child has strictly localized infrequent discharges that do not disturb the overall sleep structure.

We believe that in order to identify correlation between EEG findings and speech impairment, quantification of paroxysmal activity reflecting both spatial and temporal aspects is necessary. We find simple determination of laterality of discharges insufficient. However, counting discharges for each electrode separately in the overnight recording is not a realistic requirement.

We are uncertain about how representative for a given patient one night only record really is, as overnight recording on several consecutive sessions is usually impossible. The main aim of this study was to evaluate intraindividual variability of paroxysmal activity during night course and to propose an effective way of EEG quantification reflecting both frequency and distribution of discharges. In other words, we aimed to find the most appropriate balance between the length of recording and the number of channels in which the frequency of discharges should be evaluated.

The main limitation of the study is that we did not count PAD in all channels in every patient. Therefore, reported values of PADs do not represent paroxysmal activity over the whole brain (except from the patients with single foci with or without spreading) and cannot be compared to the results obtained by other authors. All channels were visually inspected in the whole length. Another limitation concerns the use and definition of 1-hour intervals for EEG evaluation.

Our data suggest that the frequency of discharges within time intervals of several minutes may show high degree of fluctuation. The absolute difference in PAD values obtained at different night intervals and averaged over 1 hour long intervals reached the maximum of 21% in one patient. The difference was less than 20% in the other seven cases. There was a difference in PAD between the sleep stages in three patients, presenting with higher values NREM III-IV. Two different foci may show different degree of fluctuation and even different maxima according to the sleep stage.

Supposing there is a possible link between the discharges location and cognitive deficit, we believe that, under

experimental conditions, evaluation of each electrode separately, or at least of each independent focus according to the most affected channel, is necessary. We find the classification of discharges according to Saltik's criteria very useful. We suggest following definition of time periods: 1) 40-60 minutes of each sleep stage (NREM I-II, NREM III-IV) within the first 120 minutes after falling asleep; 2) 40-60 minutes of each sleep stage (NREM I-II, NREM III-IV) during a later period of the night. This period may be set individually depending on the time falling asleep and waking up; 3) 40-60 minutes of NREM I-II within 120 minutes before waking up.

From the practical point of view, we do not think that exact quantification of discharges in a whole night records is now justified in clinical practice, as it is still not confirmed that more discharges mean greater chance for successful response to antiepileptic treatment.

If quantification of discharges is used for decision-making about starting medication, we think that evaluation of 1 hour of NREM I-II and 1 hour of NREM III-IV, starting from the beginning of the sleep, for all channels together, is sufficient. The objective evaluation of the effect of antiepileptic treatment is the goal of our ongoing prospective study. □

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References

- Autret A, Lucas B, Laffont F, Bertrand P, Degiovanni E, De Toffol B. Two distinct classifications of adult epilepsies: by time of seizures and by sensitivity of the interictal paroxysmal activities to sleep and waking. *Electroencephalogr Clin Neurophysiol* 1987; 66: 211-8.
- Chéliout-Héraut F, Picard A, Bouskraoui M, Lacert P. Anomalies EEG, modifications du sommeil et dysphasies de développement. *Neurophysiol Clin* 1999; 29: 277-89.
- Doose H. EEG in Childhood Epilepsy. Initial Presentation and Long-term Follow-up. 1st english edition. Paris: John Libbey Eurotext, 2003.
- Doose H, Baier WK. Benign partial epilepsies and related condition: multifactorial pathogenesis with hereditary impairment of brain maturation. *Eur J Pediatr* 1989; 149: 152-8.
- Duvelleroy-Hommet C, Billard C, Lucas B, et al. Sleep EEG and developmental dysphasia: lack of a consistent relationship with paroxysmal eeg activity during sleep. *Neuropediatrics* 1995; 26: 14-8.
- Echenne B, Cheminal R, Rivier F, Negre C, Touchon J, Billard M. Epileptic electroencephalographic abnormalities and developmental dysphasia: study of 32 patients. *Brain Dev* 1992; 14: 216-25.
- Holtmann M, Becker K, Kentner-Figura B, Schmidt MH. Increased frequency of rolandic spikes in ADHD children. *Epilepsia* 2003; 44: 1241-4.

Hrdlicka M, Komarek V, Propper L, *et al.* Not EEG abnormalities but epilepsy is associated with autistic regression and mental functioning in childhood autism. *Eur Child Adolesc Psychiatry* 2004; 13: 209-13.

Knobloch H, Stevens F, Malone AF. Manual for developmental diagnosis. The administration and interpretation of the revised Gesell and Amatruda developmental and neurologic examination. Hagerstown: Harper and Row, 1980.

Matejcek Z. Dyslexie – specifické poruchy čtení. Praha: H+H, 1993.

Nikolai J, Aldenkamp AP, Arends J, Weber JW, Vles JSH. Cognitive and behavioral effects of nocturnal epileptiform discharges in children with benign childhood epilepsy with centrotemporal spikes. *Epilepsy Behav* 2006; 8: 56-70.

Okubo Y, Matsuura M, Asai T, *et al.* Epileptiform EEG discharges in healthy children: prevalence, emotional and behavioral correlates, and genetic influences. *Epilepsia* 1994; 35: 832-41.

Picard A, Cheliout-Heraut F, Bouskraoui M, Lemoine M, Laceret P, Delattre J. Sleep EEG and developmental dysphasia. *Dev Med Child Neurol* 1998; 40: 595-9.

Saltik S, Uluduz D, Cokar O, Demirbilek V, Dervent A. A clinical and EEG study on idiopathic epilepsies with evolution into eses spectrum disorders. *Epilepsia* 2005; 46: 524-33.

Scabar A, Devescovi R, Blason L, Bravar L, Carroozzi M. Comorbidity of DCD and SLI: significance of epileptiform activity during sleep. *Child Care Health Dev* 2006; 32: 733-9.

Selassie GR, Jennische M, Kyllerman M, Viggedal G, Hartelius L. Comorbidity in severe developmental language disorders: Neuropediatric and psychological considerations. *Acta Paediatr* 2005; 94: 471-8.

Thorndike RL, Hagen EP, Sattler JM. Standfordský Binetův inteligenční test – IV. Revize. Brno: Psychodiagnostika, 1995.

Tuchman RF, Rapin I, Shinnar S. Autistic and dysphasic children II: Epilepsy. *Pediatrics* 1991; 88: 1219-25.

Westerlund M, Bergkvist L, Lagerberg D, Sundelin C. Comorbidity in children with severe developmental language disability. *Acta Paediatr* 2002; 91: 529-34.