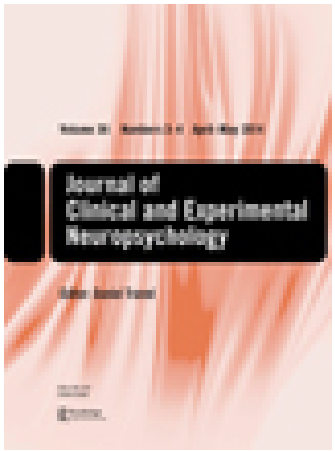

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Vanja Nenadović[ab], Miodrag Stokić[ab], Mile Vuković[c], Sanja Đoković[c] & Miško Subotić[b]

[a] Institute for Experimental Phonetics and Speech Pathology, Belgrade, Serbia

[b] Life Activities Advancement Centre, Belgrade, Serbia

[c] Faculty for Special Education and Rehabilitation, University of Belgrade, Belgrade, Serbia

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Cognitive and electrophysiological characteristics of children with specific language impairment and subclinical epileptiform electroencephalogram

Vanja Nenadović[1,2], Miodrag Stokić[1,2], Mile Vuković[3], Sanja Đoković[3], and Miško Subotić[2]

[1]Institute for Experimental Phonetics and Speech Pathology, Belgrade, Serbia
[2]Life Activities Advancement Centre, Belgrade, Serbia
[3]Faculty for Special Education and Rehabilitation, University of Belgrade, Belgrade, Serbia

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The current study is a preliminary examination of cognitive profiles and cortical distribution of the spectral power of different electroencephalogram (EEG) rhythms in children with specific language impairment and subclinical epileptiform discharges. Although a number of empirical studies point to higher incidence of abnormal EEGs in children with specific language impairment, only a few studies were found examining electrophysiological characteristics, such as locus of discharges and connections with cognitive functioning in this population of children. The sample included 12 children with specific language impairment (SLI) and abnormal EEG who underwent testing of cognitive functioning using the Wechsler Intelligence Scale for Children (WISC). The control sample included 13 children with specific language impairment and regular EEG. Results point to lower scores on several subtests of the performance scale for children with abnormal EEG than for the group with regular EEG. Detailed EEG analysis of cortical distribution of the spectral power of different EEG rhythms partially confirms the results of neuropsychological assessment, pointing to abnormal function of frontal and temporal regions. Higher values of spectral power of the delta brain rhythm in frontal regions are associated with lower results on the WISC performance scale. Results are discussed in the context of subgroups of the population of children with SLI.

Keywords: Electroencephalogram; Specific language impairment; Cognitive profile; Sharp and slow waves; Brain rhythms.

Abnormal neural discharges in the brain, marked by typical spike-wave complexes on the electroencephalogram (EEG) underlie epileptic seizures. However, EEG activity of a similar kind, manifested as spike and wave or sharp wave activity can appear without clinical manifestations. These events are known as "subclinical," "larval," or "interictal" discharges. One of the first studies undertaken by Schwab in 1939 showed that this type of discharge could be accompanied by impairments in cognitive functions that were of a transitory nature (Schwab, 1939, in Aldenkamp & Arends, 2004). This was demonstrated by reaction time tasks, showing a slowing of reaction time in a number of patients with subepileptiform discharges. Aarts, Binnie, Smith, and Wilkins (1984) have named this phenomenon *transitory cognitive impairment*. It has been debated in literature

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Address correspondence to: Vanja Nenadović, Gospodar Jovanova 35, 11 000 Belgrade, Serbia (E-mail: vilhi@sezampro.rs).

whether this type of event is subclinical or can be interpreted as a brief seizure (Binnie, 2003). Rugland (1990) has also measured simple and choice reaction time in patients with subclinical discharges, with discharges lasting under 3 s. His study showed a slowing of reaction time. In another study by Aldenkamp and Arends (2004), cognitive slowing (measured by simple visual or auditory reaction time) was also shown in direct association with an epoch characterized by subclinical epileptiform discharges. These authors found a correlation between type of discharge—that is, generalized type of discharge and cognitive slowing.

Empirical data show that about 10% of children without a diagnosis of epilepsy have subclinical EEG (Selassie, Viggedal, Olsson, & Jennische, 2008). However, little is known of the relationship between epileptiform activity described as subclinical and children with different types of developmental delay without epilepsy, due to the paucity of studies undertaken on this population. According to Ballaban-Gil and Tuchman (2000), the term *epileptiform disorder with cognitive symptoms* should be used to describe individuals who have no seizures, but do have epileptiform discharges. These could cause cognitive, speech and language, or behavioral dysfunction. Deonna and Roulet-Perez (2005) have used the term *cognitive epilepsy* for this condition.

Specific language impairment (SLI in further text) is a developmental disorder represented as a language delay, which is not a result of known neurological, sensory, intellectual, or emotional deficit. It is usually marked by a combination of normal intelligence and language impairment. The normal intelligence measure is usually operationalized through a performance IQ of 85 or more, with the language measure 1.25 standard deviations below average, according to Tomblin, Records, and Zhang (1996) or 1 standard deviation according to Tallal (Tallal & Benasich, 2002).

Most studies point to higher incidence of epilepsy or abnormal EEGs in children with SLI. A study by Tuchman (1994) points to 8% incidence of epileptiform abnormalities in children with specific language impairment, without a history of seizures, compared to 1% in the typical population. Some authors point that these findings are state dependent and that higher rates were found in SLI children, on which all night recordings were made (Echenne et al., 1992). In a study by Maccario, Hefferen, Keblusek, and Lipinski (1982), abnormalities in the form of spike and wave and sharp wave activity were found on a small sample of SLI children aged 2 to 5 years, also without seizures. In a sample of 50 SLI children (Fabbro, Zucca, Molteni, & Renatto, 2000), 8% of children have shown EEG abnormalities in the wake state, with 56% showing paroxysmal abnormalities in their sleep EEG. Interestingly, almost all (91%) of receptive language disorders in this sample showed abnormal EEG. Wheless, Simos, and Butler (2002) have reviewed studies showing that interictal discharges have an effect on speech and language in epileptic syndromes. In a study conducted by Picard, Cheliout Heraut, and Bouskraoui (1998), focal discharges predominantly in the left hemisphere, as well as generalized paroxysmal activity, were found in EEG during sleep, on a sample of children with SLI aged 4 to 11 years. These authors concluded that paroxysmal discharge itself can be a contributing problem to language deficits. Although named "transitory," cognitive impairment could have adverse effects when we are discussing cognitive and language structures under development. In other words, if subclinical discharges cause transitory cognitive impairment, there can be a cumulative effect of these mild impairments over time on stable cognitive structures (Aldenkamp & Arends, 2004).

Concerning the issue of locus of focal discharges, studies generally point to more errors in verbal tasks, in participants with left-sided focal spiking, while right-sided ones usually show impairment in processing of nonverbal material. In a recent study by Levy-Rueff et al. (2012), EEG abnormalities were registered in 49% of the sample of children diagnosed with SLI. This study is of interest also due to its focus on locus of discharges in the brain. Most abnormalities were located in the left side of the brain and in two specific regions: the temporo-occipital and frontorolandic.

Several studies have examined EEG during language processing or cognitive performance, focused on the analysis of the task-induced oscillations (Csibra, Davis, Spratling, & Johnson, 2000; Kaufman, Csibra, & Johnson, 2005; Southgate, Csibra, Kaufman, & Johnson, 2008). Benasich, Gou, Choudhury, and Harris (2008) examined individual differences in the distribution of power spectra across the scalp during resting (spontaneous) EEG epochs, in relation to cognitive ability. These data are valuable because they represent a relatively uncharted territory of association among power in the various frequency bands and cognitive and linguistic performance.

Webster and Shevell (2004) point to the fact that the population of children with SLI is highly heterogeneous. This could point to biological heterogeneity. They argue that factors that lead to language impairment also affect other neurologic processes

and are not specific as determined by this terminology. This can be seen in studies on increased incidence in motor abnormalities (Bishop, 2002; Trauner, Wulfeck, Tallal, & Hesselink, 2000; Vuković, Vuković, & Stojanovik, 2010) or "soft" neurological signs. When considering language not as an isolated system, but connected to general-purpose cognitive systems, this interpretation then seems plausible, because different aspects of language could be affected by a deficit (Dennis, 2010). One of the research pathways for examining the "specificity" of SLI is further exploration of possible neurophysiologic abnormalities at the level of EEG. This issue still remains to be tackled by new studies.

Most of the studies within this field of research have viewed this phenomenon in the context of possible predictive factors for epilepsy or treatment options in case of chronic adverse effects of abnormal EEG activity. However, not many studies were focused on examining whether this population is characterized by specific cognitive profiles. In other words, can a link be established between abnormal EEG and specific deficits in cognitive functioning? Examination of cortical distribution of different EEG rhythms (their spectral power as a measure of neuronal synchronous activity) may also help to clarify the link between cognitive profile and electrophysiological features. If a preliminary association is established, studies with stronger designs should follow, aimed at uncovering possible causal connections by use of simultaneous EEG and cognition testing on carefully selected clinical subgroups of children with SLI.

This study is a preliminary examination of the tentative association between the EEG and cognitive functioning. Our aim was to describe the cognitive and electrophysiological characteristics of children with specific language impairment in the context of uncovering whether resting state EEG patterns are linked with measures of cognitive skills within this clinical population. The study was conducted on several levels. We have selected a subgroup of children with abnormalities of the EEG, but without overt seizures. The control group consisted of children diagnosed with SLI who had regular EEG. Specifically, we wanted to examine whether children with subclinical discharges would have lower performance on the verbal and/or nonverbal aspects of cognition. The next level consisted of EEG analysis. This study's exploratory goal determined registration and reporting of cortical distribution of delta (2–4 Hz), theta (4–8 Hz), low alpha (8–10 Hz), high alpha (10–13 Hz), low beta (13–20 Hz), and high beta (20–30 Hz) during the awake state with eyes open. Next, we hypothesized that an association existed between brain regions where discharges were registered (mostly frontal and temporal regions as noted by a neurologist) and scores on specific cognitive subtests. We expected that burdening of these brain regions with slow waves could be associated with poor achievement on tasks involving executive functions and executive attention, as well as specific verbal tasks. Differences between groups were expected for slow brain rhythms. Also, we expected to confirm findings from literature concerning faster brain rhythms (Schmid, Tirsch, & Scherb, 2002; Thatcher, North, & Biver, 2005). Specifically, the beta rhythms are connected to logical reasoning and high alertness and would therefore be associated with higher scores on the WISC.

In sum, although some associations were found in earlier studies, concerning fast and slow brain rhythms and cognitive performance, these studies were mostly carried out on the typical population. When abnormal brain activity is concerned, our hypothesis was partially based on data showing the link of localization of focal spiking and type of cognitive performance.

METHOD

Participants

The participants were 12 children with specific language impairment and subclinical EEG discharges, which represented the experimental group (E group). Thirteen children with specific language impairment and regular EEG represented the control group (C group). The mean age of the E group was 6 years and 7 months (range from 5 years to 11 years 2 months). The mean age of the C group was 6 years and 6 months (age range from 5 years to 9 years 8 months). Both groups were referred to the Institute for Experimental Phonetics and Speech Pathology due to speech and language delay. These children received the diagnosis of specific language impairment based on a battery of tests and detailed anamnestic interview mapping early language development, medical history, and a family history of developmental and speech and language disorders. The diagnosis of speech and language impairment was given by a qualified speech and language therapist. None of the children had ever experienced a seizure, nor had ever received medication. A detailed anamnestic interview revealed cases with developmental delays of language at an early age (approximately 30 months). Cases with regression in language development were excluded. Written informed consent for participation was received from all parents.

TABLE 1
Mean values, standard deviations and group comparisons for scores on WISC, including VIQ, PIQ, and each subtest

WISC	*E group (n = 12) Mean (SD)*	*C group (n = 13) Mean (SD)*	*Independent samples (t test) (p)*
VIQ	78.15 (12.85)	74.5 (13.28)	.492
Information	6.77 (2.59)	7 (2.45)	.821
Comprehension	6.38 (3.75)	4.92 (2.78)	.281
Arithmetic	7 (2.97)	7.25 (3.17)	.840
Similarities	5.46 (1.94)	5.08 (1.88)	.626
Short-Term Memory	6.85 (2.44)	5.50 (2.39)	.178
PIQ	104.31 (15.86)	93.08 (12.21)	.061
Picture Completion	11.08 (2.66)	9.25 (1.29)	.042
Picture Arrangement	12.69 (2.66)	9.42 (2.43)	.004
Block Design	9.54 (3.89)	8.83 (3.56)	.642
Object Assembly	10.46 (3.55)	9.92 (1.97)	.644
Coding	9.38 (3.57)	7.25 (2.76)	.110

Note. WISC = Wechsler Intelligence Scale for Children; E = experimental group; C = control group; VIQ = verbal IQ; PIQ = performance IQ.

The study was performed in accordance with the ethical standards laid down in the Declaration of Helsinki, and the protocol was approved by the local human ethics committee.

This study used Stark and Tallal's (1981) term of specific language impairment, based on the exclusion criteria. Children with neurological disorders, as well as those with autistic spectrum disorder (according to *Diagnostic and statistical manual of mental disorders–Fifth Edition, DSM–V*, American Psychiatric Publishing, 2013, criteria), intellectual disability, and hearing impairment, were excluded from the study. Also, a discrepancy criterion was included, as typical of SLI studies: a difference of at least 1 standard deviation between verbal IQ (VIQ) and performance IQ (PIQ). Participants with full-scale IQ (FIQ) below 70 were excluded from the study as intellectual disability.

Both groups were compared based on gender, parents' education, and mono- or bilingualism using nonparametric statistics (test for trends in contingency tables), and no statistically significant differences were found.

An experienced child neurologist examined the EEG to identify and describe epileptiform activity. The EEG recording was then classified as *normal*, showing amplitude and frequency adequate for the participant's age and conscious state (Stern, 2004), or *epileptiform*, marked by sharp waves and excessive slow activity.

Neuropsychological assessment

The complete experimental and control groups were assessed by a psychologist using the Serbian standardization of Wechsler's Intelligence Scale for Children (WISC), REVISK (Biro, 1998). The results of this assessment are given as VIQ, PIQ, and separate subtest scores (Table 1). Each participant was tested in optimal conditions, which consisted of a session (60−80 min) in a quiet, plain room with the examiner.

EEG recordings

During the EEG recording, the participants were placed in a comfortable sitting position in a sound and electrically shielded room. The participants were isolated from visual and auditory stimuli using white curtains arranged in a box-shaped space in a soundproof room.

EEG was recorded using the Nihon Kohden Corporation, EEG 1200K Neurofax apparatus with Electrocap (model number 16 755) International, Inc., silver/silver chloride (Ag/AgCl) ring electrodes filled with electroconductive gel, providing 16 EEG channels. Electrodes were positioned according to the 10/20 placement system in longitudinal, monopolar montage. The reference electrode was set offline to A1 and A2 (ear lobes). The horizontal and vertical electrooculograms (EOGs) were recorded in order to detect eye-blinks and eye movements. The heart rate and hand movement sensors and electrodes for jaw muscle activity were used for offline artifact removal. The AC filter was on. The sampling rate was 200 Hz.

Impedance was kept below 5 kΩ, lower filter was set on 0.53 Hz and upper filter on 35 Hz in order to select frequency bands of interest and to cut off higher frequencies that might be muscle artifacts. According to the International 10/20 system of electrode positioning, the following cortical regions

were analyzed: Fp1–Fp2 (frontopolar), F3–F4 (midfrontal), F7–F8 (inferior frontal, anterior temporal, frontal-temporal), T3–T4 (midtemporal), T5–T6 (posterior temporal), Fz (frontal midline central), P3–P4 (parietal), Pz (parietal midline central), and O1–O2 (occipital).

The spontaneous (resting) EEG was recorded for 10 minutes. The participants' task was to keep their eyes open. Participants were asked to minimize their movements (eye blink, head and limbs movement) as much as possible in order to eliminate artifacts from the raw EEG trace. Resting state was used to determine whether there were neurological disorders as well as to determine mean spectral power values for delta (2–4 Hz), theta (4–8 Hz), low alpha (8–10 Hz), high alpha (10–13 Hz), low beta (13–20 Hz), and high beta (20–30 Hz) in frontal, temporal, parietal, and occipital cortical regions.

Before analysis, we first removed the data segments that contained obvious eye blinking, high-amplitude, high-frequency muscle noise, and other irregular artifacts, as identified by visual examination. In our study, we used fast Fourier transform (FFT) in order to separate brain rhythms from the raw EEG trace. The first task in signal analysis was to choose artifact free epochs from the recorded 10 min of the resting state. Before computing FFT, each epoch was multiplied by an appropriate windowing function (using Hanning window) in order to avoid border problems (leakage). Then FFT was computed in order to create spectrograms and amplitude maps of the selected epoch. Out of the total of 600 seconds (100%), 76.09% of EEG data was used for the control group for further spectral power analysis and 74.46% for the experimental group. No statistically significant differences were found in the amount of data between the control group (M = 456.54, SD = 39.83) and the experimental group (M = 446.75, SD = 33.06). Result of the t test has shown the following, $t(23) = 0.665$, $p = .512$. In this way, six brain rhythms (delta, theta, low alpha, high alpha, low beta, and high beta) were obtained, and their spectral power was analyzed. For each rhythm, the mean spectral power (averaged for epochs for each participant) in Fp1, Fp2, F3, F4, F7, F8, Fz, T3, T4, T5, T6, P3, P4, Pz, O1, and O2 was further used in statistical analysis.

Statistical analysis

The data were analyzed using the IBM SPSS Statistics 22. The t test was used to analyze results of neuropsychological assessment as well as the nonparametric test for trends in contingency tables, for comparison of categorical variables. Spectral power data for each rhythm (delta, theta, low alpha, high alpha, low beta, and high beta) were normalized by log10 function and were averaged across brain regions into four variables (frontal, temporal, parietal, and occipital). A 2 (group: control, experimental) × 4 (region: frontal, temporal, parietal, occipital) × 6 (rhythm: delta, theta, low alpha, high alpha, low beta, high beta) multivariate analysis of variance (MANOVA) was used to examine effect of group, region, and rhythm on spectral power. Normalized spectral power mean values for each rhythm were also carried out for regions Fp1, Fp2, F3, F4, F7, F8, Fz, T3, T4, T5, T6, P3, P4, Pz, O1, and O2 between E and C groups using a series of post hoc t tests.

RESULTS

Neuropsychological findings

The first level of analysis consisted of results from the neuropsychological assessment. As can be seen in Table 1, independent-samples t tests revealed no statistically significant differences for the verbal and performance scales of WISC, respectively. However, group differences were found for two subtests of the performance scale. Picture Completion showed statistical significance in scores, between E group (M = 9.25, SD = 1.29) and C group (M = 11.08, SD = 2.66), $t(23) = 2.15$, $p = .042$. Picture Arrangement scores also differed between the E group (M = 9.42, SD = 2.43) and the C group (M = 12.69, SD = 2.66), $t(23) = 3.21$, $p = .004$. The E group had lower scores on the remaining subtests of the performance scale, but none reached statistically significant difference. It should be noted that group differences between E group (M = 93.08, SD = 12.21) and C group (M = 104.31, SD = 15.86) on the PIQ were close to significance, $t(23) = 1.97$, $p = .061$.

EEG findings

Differences in spectral power values averaged across brain regions (Table 2) were first examined with a 2 (group: control, experimental) × 4 (region: frontal, temporal, parietal, occipital) × 6 (rhythm: delta, theta, low alpha, high alpha, low beta, high beta) MANOVA. The MANOVA revealed a significant effect of region, $F(3, 596) = 10.393$, $p < .001$, and rhythm, $F(5, 594) = 308.081$, $p < .001$. There was no significant main effect for group.

TABLE 2
Averaged EEG spectral power for the experimental and control groups across brain regions and brain rhythms

Brain rhythm	*Frontal*		*Temporal*		*Parietal*		*Occipital*	
	C	*E*	*C*	*E*	*C*	*E*	*C*	*E*
δ	0.50 (0.05)	0.65** (0.08)	0.45 (0.04)	0.57** (0.12)	0.58 (0.07)	0.61 (0.24)	0.55 (0.11)	0.64 (0.12)
θ	0.49 (0.02)	0.64** (0.11)	0.50 (0.02)	0.55* (0.09)	0.53 (0.07)	0.64* (0.15)	0.54 (0.04)	0.59* (0.09)
α1	0.43 (0.06)	0.31** (0.06)	0.38 (0.07)	0.35 (0.10)	0.41 (0.06)	0.44 (0.15)	0.53 (0.15)	0.44 (0.12)
α2	0.18 (0.02)	0.15* (0.04)	0.19 (0.06)	0.18 (0.04)	0.30 (0.10)	0.29 (0.14)	0.36 (0.13)	0.32 (0.07)
β1	0.29 (0.04)	0.20** (0.08)	0.26 (0.08)	0.22 (0.12)	0.20 (0.08)	0.25 (0.15)	0.31 (0.02)	0.29 (0.12)
β2	0.27 (0.04)	0.14** (0.07)	0.26 (0.09)	0.22* (0.11)	0.07 (0.04)	0.06 (0.04)	0.13 (0.07)	0.12 (0.11)

Note. Spectral power in μV^2. EEG = electroencephalogram; δ = delta rhythm; θ = theta rhythm; α1 = low alpha rhythm; α2 = high alpha rhythm; β1 = low beta rhythm; β2 = high beta rhythm; E = experimental group; C = control group. Standard deviations in parentheses.

*p < .01–.05. **p < .00–.01.

However, significant interactions were the following: the interactions between group and rhythm, $F(5, 552) = 12.555$, $p < .001$, and region and rhythm, $F(15, 552) = 6.888$, $p < .001$, and group, region, and rhythm intercept, $F(15, 552) = 1.678$, $p = .051$.

A post hoc one-way analysis of variance (ANOVA) showed significant effect of group on spectral power when frontal regions are concerned (electrode locations: Fp1, Fp2, F3, F4, F7, F8, and Fz) in delta, $F(1, 173) = 28.782$, $p < .001$, theta, $F(1, 173) = 19.752$, $p < .001$, low alpha, $F(1, 173) = 30.139$, $p < .001$, high alpha, $F(1, 173) = 6.835$, $p = .015$, low beta, $F(1, 173) = 14.365$, $p < .001$, and high beta, $F(1, 173) = 31.521$, $p < .001$. Regarding frontal regions, series of post hoc t tests showed no significant differences between E and C groups in delta rhythm in electrode locations F8 and Fz. All other electrode locations had statistically significant differences ($p < .01$). In theta rhythm, differences were obtained between E and C groups in all electrode locations ($p < .01$). In low alpha rhythm no significant differences between E and C groups were obtained in electrode locations F8 and Fz. All other electrode locations had statistically significant differences ($p < .05$). In high alpha rhythm, no significant differences between E and C groups were obtained in all electrode locations. In low beta rhythm, no significant differences between E and C groups were obtained in electrode locations F4, F7, F8, and Fz. All other electrode locations had statistically significant difference between E and C groups ($p < .05$). In high beta rhythm, no significant differences between E and C groups were obtained in electrode locations F4, F8, and Fz. All other electrode locations had statistically significant difference between E and C groups ($p < .05$).

Table 2 shows that the statistically significant differences in frontal regions were most prominent for slow brain rhythms (delta and theta). Children from E group had significantly higher spectral power of slow brain rhythms in prefrontal brain regions than C group. In addition, higher brain rhythms (low beta and high beta) had lower spectral power in children from E group.

A one-way ANOVA showed significant effect of group on spectral power when temporal regions are concerned (electrode locations: T3, T4, T5, and T6) in delta, $F(1, 98) = 10.787$, $p < .01$, theta, $F(1, 98) = 5.248$, $p = .02$, and high beta, $F(1, 98) = 7.766$, $p < .01$. Regarding temporal regions, series of post hoc t tests showed no significant differences between E and C groups in delta rhythm in electrode location T6. All other electrode locations had statistically significant differences ($p < .05$). In theta rhythm, no differences were obtained in electrode locations T4, T5, and T6. T3 electrode location had significant difference ($p < .01$). No differences were obtained in low alpha, high alpha, and low beta rhythm between E and C groups. In high beta rhythm, no differences were obtained in electrode location T6. All other electrode locations had statistically significant differences ($p < .01$).

A one-way ANOVA showed significant effect of group on spectral power when parietal regions are concerned (electrode locations: P3, P4, and Pz) in theta, $F(1, 73) = 6.289$, $p = .02$, and occipital regions (electrode locations: O1 and O2) in theta, $F(1, 23) = 4.169$, $p < .01$. No differences were obtained in delta, low alpha, high alpha, low beta, and high beta rhythms. Regarding parietal and occipital regions, series of post hoc t tests showed no significant differences between E and C groups in theta rhythm in electrode locations P4, Pz, and O1. All other electrode locations had statistically significant differences ($p < .01$).

The obtained results indicate frontal, temporal, parietal, and occipital increase of spectral power of slow brain rhythms and decrease of spectral power

of higher brain rhythms in the E group, when compared to the C group.

Correlational analysis of WISC performance and EEG

The final level of analysis included calculation of correlation coefficients in order to assess the relationship between performance on WISC and EEG results. In order to reduce the number of comparisons, the EEG data averaged across regions were used in the computation, for all four brain regions: frontal, parietal, temporal, and occipital. Correlation was computed for each of the six brain rhythms: delta, theta, low alpha, high alpha, low beta, and high beta. Results of the analysis so far uncovered differences between the experimental and control groups on some subtests of the performance scale. Also, we ran a correlation between the four averaged sites and PIQ, as well as each subtest (Table 3), for all of the six brain rhythms. Statistically significant correlations were found between frontal brain regions and results on the performance scale of WISC, for the delta, low alpha, high alpha, and high beta brain rhythms. The results are presented in the next section for each of the noted brain rhythms.

In the next level of analysis with delta rhythm, the average spectral power in the four brain regions was correlated with PIQ score and scores for each subtest using Pearson's product–moment correlation coefficient. The correlation coefficients are presented in Table 3. Statistically significant moderate negative correlations were found between frontal regions and PIQ, $r(23) = -.586$, $p = .002$. Also, statistically significant moderate negative correlations were found between frontal regions and the Picture Arrangement subtest, $r(23) = .594$, $p = .002$, and Code subtest, $t(23) = -0.514$, $p = .009$. No statistically significant differences were found for correlations computed between other brain regions and PIQ with performance scale subtests. Greater average spectral power in frontal regions, within the delta rhythm, is associated with lower achievement of the performance scale in full, as well as lower scores on the Picture Arrangement and Code subtests.

Analysis for low alpha yielded statistically significant moderate positive correlations between average spectral power in frontal regions and scores on the Picture Arrangement subtest, $t(23) = 0.413$, $p = .040$. Analysis for high alpha showed statistically significant moderate positive correlations between average spectral power in frontal regions and scores on the full performance scale, $t(23) = 0.413$, $p = .040$. Also, correlations were found for high beta. Moderate positive correlations that were statistically significant were found between frontal regions, the PIQ, $t(23) = 0.532$, $p = .006$, and Picture Arrangement, $t(23) = 0.504$, $p = .010$. Greater average spectral power in frontal regions, in low alpha, high alpha, and high beta, is associated with higher scores on the performance scale and the Picture Arrangement subtest.

No statistically significant correlations were found for the other brain regions. Also, no statistically significant correlations were found for the theta rhythm.

Following this study's results concerning the measure of association between the EEG and cognitive functions, a larger sample would enable more sophisticated statistical testing of the nature of this association in the form of multiple regression analysis, aimed at uncovering the possible predictive value of brain regions for scores on specific cognitive tasks.

TABLE 3
Correlations between average EEG power for each brain region, PIQ, and each performance scale subtest within the delta rhythm

Performance scale	*Frontal*	*Temporal*	*Parietal*	*Occipital*
PIQ	–.586**	–.426**	–.132	–.130
VD	–.172	–.077	.226	.000
PA	–.594**	–.336	–.131	–.158
BD	–.378	–.276	–.071	–.087
OA	–.311	–.214	–.033	–.080
C	–.514**	–.391	–.085	–.101

Note. EEG = electroencephalogram; PIQ = Performance IQ; VD = Visual Discrimination; PA = Picture Arrangement; BD = Block Design; OA = Object Assembly; C = Coding.
*$p < .05$. **$p < .01$.

Localization of EEG discharges

Sharp waves and excessive slow activity in the experimental group of children were dominantly expressed over frontal and temporal regions: Seven children had bilateral excessive slow activity—higher spectral power of delta and theta rhythm—over frontal regions, three children had excessive slow activity in the left frontal-temporal region, while two children had left temporal-parietal excessive slow activity. Sharp waves (combined with ongoing slow activity) were detected in almost all cases of the E group, in left frontotemporal regions (except one child that had sharp waves in the temporal-parietal region).

DISCUSSION

The aim of our study was to examine cognitive profiles in children with specific language impairment and abnormal EEG, in order to determine whether different patterns of specific verbal and nonverbal cognitive functions existed, when compared to children with SLI and regular EEG. We also analyzed the spontaneous (resting) EEG recording of SLI children with abnormal EEG in order to determine localization of brain rhythms and their spectral power compared to SLI children with regular EEG.

Our results did not show differences in cognitive profiles between SLI children with subepileptiform discharges and SLI children with regular EEG, when full verbal and performance IQs are taken into account. However, SLI children with subepileptiform discharges had lower performance on some subtests of the performance scale. A more detailed examination of differences across subtests has shown that scores of the experimental group were lower for the Picture Completion subtest and the Picture Arrangement subtest.

The Picture Completion subtest measures visual perception, as well as the ability of the child to differentiate essential from inessential detail. Difficulties in resolving this task can be ascribed to problems with visual input and inability to note visual detail. This is an interesting finding in the context of discussing the potential difficulties that define the population of children with SLI. Some authors claim that deficits on nonverbal tasks could be tied to use of immature strategy and poor planning rather than difficulties in visuospatial processing per se (Akshoomoff, Stiles, & Wulfeck, 2006). However, there are studies showing that SLI children have difficulties with visual discrimination (Hill, 2001; Powell & Bishop, 1992). Our findings are in line with this group of studies. If studies on larger samples of SLI children could corroborate this finding—simultaneous EEG with testing of visuoperceptual skills and visual discrimination on the population of children with SLI—this would be an important step towards more precise marking of likely neuropsychological profiles.

We should be cautious when interpreting the results of the Picture Arrangement subtest, because it has been estimated by authors as complex, consisting of visuospatial ability, including a motor response in the form of matching visual material by rotating one piece a number of times (Carroll, 1993). Performance on this subtest also includes the operations of sequencing and planning. The latter processes are an integral part of higher order thinking. Clinical evidence points to phases in successful completion of this subtest: Usually, the child is at first able to correctly retell the picture story by looking at it. However, the verbal response is not accompanied by a motor response (i.e., arranging the cards in the correct order). As the child gains success on this subtest, he or she is able to coordinate a complex set of operations consisting of visual perception analysis, sustained attention and shifting of attention, sequencing, motor planning, and execution. The child becomes more competent in self-regulating his or her behavior. Children with subclinical discharges have shown lower results than the control sample. In other words, these children had difficulties in tasks requiring higher order thinking. This has been corroborated by the EEG findings in our study and are discussed in the next section. Lower scores on both the Picture Completion and the Picture Arrangement subtests point to problems in visual input and inability to note visual detail. There were no statistical differences between the two examined groups on other subtests of the performance scale. The result of the *t* test aimed at assessing group differences on the PIQ came close to significance. Studies on larger samples would be necessary to test the tendency found in this study. Studies designed to more specifically probe different nonverbal skills, especially visuospatial, fine motor skills, and executive functions, would help answer the question of whether a specific neuropsychological profile could be delineated for this subpopulation of SLI children. Also, longitudinal studies are needed to examine the dynamics of language development and effects of speech therapy.

A review of studies that have tried to link EEG results and cognitive testing shows adverse findings. Parry-Fielder et al. (2008) have shown a significant association between abnormal sleep EEG and PIQ in their clearly defined sample of children with severe language impairment. These authors also point to subaverage scores on lexical diversity and length of utterance for these children. However, a fuller conclusion could not be made based on these results, due to lack of a control sample. The control group in our study consisted of children with SLI, and comparisons were made when aspects of language are concerned. The two examined groups were selected according to diagnosis of specific language impairment. Therefore, relatively similar measures of VIQ were expected. However, a more detailed examination of verbal profiles was conducted in order to establish whether EEG irregularities would result in different patterns of specific verbal abilities in the tested

groups of children. This was not confirmed in our study: Both groups showed a subnormal VIQ with relatively even subaverage scores across subtests.

Maccario et al. (1982) gave an assessment of intelligence of SLI children with abnormal EEG and showed normal nonverbal intelligence. However, the sample age (2 to 5 years) did not allow more thorough testing or stable measures.

It needs to be noted here that selection criteria in our study for the sample of children with abnormal EEG excluded a number of children with even lower PIQ scores. These cases could not be defined as SLI, according to Stark and Tallal (1981) criteria, because the discrepancy between VIQ and PIQ was less than 15 units or FIQ was under 70. However, it needs to be noted that this population of children, who could not be strictly defined as SLI, also exhibited diverse language deficits combined with deficits of visuospatial and fine motor skills. When discussing the VIQ−PIQ discrepancy, numerous authors claim that visuospatial skills are also impaired in the SLI population (Hick, Botting, & Conti-Ramsden, 2005; Johnston, 1994). It is possible that abnormal EEG causes complex deficits, manifesting in verbal as well as nonverbal skills. There is ample evidence connecting low PIQ with learning disabilities. However, only one study was found linking EEG abnormalities with learning impairments (Koch & Duane, 2010).

Is there an association between EEG and nonverbal skills?

Our study showed that SLI children with subepileptiform discharges had an increase in spectral power for slow brain rhythms (delta and theta) in prefrontal and frontotemporal regions and decrease of faster brain rhythms, when compared to their peers. Using a correlational analysis, we tried to assess the relationship between slow wave activity and performance on specific subtests. Results point to relevant correlations within the delta brain rhythm, between frontal regions and performance on the Picture Arrangement task and Coding. Were aspects of frontal functioning tapped in these subtests, as described earlier (Fuster, 2002; Romine & Reynolds, 2005)? If frontal regions are burdened by slow-wave activity, a drop in performance should be expected. Also, it needs to be noted that the Picture Arrangement and Coding are amongst the WISC subtests that place the highest demand on attention. This is compatible with our other finding of a moderate positive association between low alpha and the score on the Picture Arrangement task.

Correlational analysis did not confirm our hypothesis on the association of theta brain rhythm and scores on the performance scale.

The burden of slow activity over frontal regions in the group of children with abnormal EEG, associated with the drop in performance on cognitive tasks, can only be further explored by a detailed examination of the multidimensional construct of executive function with simultaneous EEG recordings. There is a growing field of executive function testing incorporating observational, behavioral, and self-report data (Demakis, 2004). Swingler, Willoughby, and Calkins (2011) have made progress in a specialized battery of executive function tests for preschool-aged children, which were EEG monitored during task execution. They show significant changes from baseline to task completion, in EEG coherence at specific prefrontal site pairs, within the 6–9 Hz frequency band.

We point now to an interesting case study that shows a connection between frontotemporal subclinical epileptiform discharges and executive functions and performance IQ in an adult (Licht, Jacobsen, & Fujikawa, 2002). This was a study conducted on a case of chronic and disruptive EEG activity, as characterized by the authors. The patient was on antiepileptic medication for a number of years. He was followed for a period of 16 years with 81% of his total EEG recordings burdened with subclinical epileptiform activity, mostly in the form of spike and wave discharges frontotemporally. Great variability in IQ scores on the Wechsler Adult Intelligence Scale–Revised (WAIS–R) was registered, with an improvement in test scores after introducing adequate medication. Deficits of frontal or executive functions were registered in this patient. Examination of his VIQ and PIQ results points to gradual increase in the VIQ, but not the PIQ. The authors point to cumulative effects from chronic cerebral disruption that could create a sustained encephalopathy, which persists even in stages of recovery and absence of discharges. Returning to our study, the same connection between locus of discharges and PIQ and performance subtests was found, with different implications, because we have tapped developing functions. The resting state delta rhythm can be viewed from a maturational point. Resting state activity shows gradual reduction in the amplitude of slow-wave activity—delta and theta—rhythms, while faster rhythms increase over childhood (Knyazev, 2012). This opens up an interesting dilemma for studies of the SLI population. Could slower progress in treatment of subgroups of SLI children be ascribed to neuropsychological immaturity or pathological processes? Our study has

examined profiles of SLI population showing associations with higher spectral power of low alpha and high alpha, as well as high beta—which could be interpreted as a more mature EEG—and better scores on the performance scale.

As previously noted, no differences were found on the verbal scale between the tested groups, and it is possible that only finer neuropsychological assessment would uncover differences as shown on the EEG, such as assessment of auditory processing and word recognition in particular. We also assume that subaverage language profiles, as seen in our sample, could have different underlying sources. This hypothesis could be tested in a longitudinal study aimed at follow-up of treatment outcome for this subpopulation. Persistent or severe language impairments could have abnormal EEG as one of the factors causing slow progress.

Our results from a population of SLI children burdened with sharp waves and excessive slow brain activity show a possible complex clinical picture pointing to specific deficits in the domain of frontal functioning and perceptive organization, in addition to language deficits. Studies on larger samples need to be carried out in order to further examine these findings. A more detailed neuropsychological assessment should be aimed at executive functions. Direct exploration of the electrophysiological level of functioning could possibly answer the question of why the SLI population is heterogeneous, as we have pointed out earlier. In addition, longitudinal studies are needed on this specific subpopulation of children, aimed at examining length of speech and language treatment as well as treatment outcomes in order to fully map the adverse effects of abnormal EEGs.

REFERENCES

Aarts, J. H. P., Binnie, C. D., Smith, A. M., & Wilkins, A. J. (1984). Selective cognitive impairment during focal and generalized epileptiform EEG activity. *Brain*, *107*, 293–308.

Akshoomoff, N., Stiles, J., & Wulfeck, B. (2006). Perceptual organization and visual immediate memory in children with specific language impairment. *Journal of the International Neuropsychological Society*, *12*(4), 465–474.

Aldenkamp, A., & Arends, J. (2004). The relative influence of epileptic EEG discharges, short non-convulsive seizures and type of epilepsy on cognitive function. *Epilepsia*, *45*(1), 54–63.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Ballaban-Gil, K., & Tuchman, R. (2000). Epilepsy and epileptiform EEG: Association with autism and language disorders. *Mental Retardation and Developmental Disabilities Research Reviews*, *6*, 300–308.

Benasich, A., Gou, Z., Choudhury, N., & Harris, K. (2008). Early cognitive and language skills are linked to resting frontal gamma power across the first three years. *Behavioral Brain Research*, *195*(2), 215–222.

Binnie, C. D. (2003). Cognitive impairment during epileptiform discharges: Is it ever justifiable to treat the EEG? *The Lancet Neurology*, *2*, 725–730.

Biro, M. (1998). *REVISK manual* (2nd rev. ed.). Belgrade: Drustvo psihologa Srbije [Serbian Psychological Society].

Bishop, D. V. M. (2002). Motor immaturity and specific language impairment: Evidence for a common genetic basis. *American Journal of Medical Genetics*, *114*, 56–63.

Carroll, J. (1993). *Human cognitive abilities*. Cambridge: Cambridge University Press.

Csibra, G., Davis, G., Spratling, M. W., & Johnson, M. H. (2000). Gamma oscillations and object processing in the infant brain. *Science*, *290*, 1582–1585.

Demakis, G. (2004). Frontal lobe damage and tests of executive processing: A meta-analysis of the Category Test, Stroop Test and Trail Making test. *Journal of Clinical and Experimental Neuropsychology*, *26*(3), 441–450.

Dennis, M. (2010). Language disorders in children with central nervous system injury. *Journal of Clinical and Experimental Neuropsychology*, *32*(4), 417–432.

Deonna, T., & Roulet-Perez, E. (2005). *Cognitive and behavioral disorders of epileptic origin in children*. London: McKeith Press.

Echenne, B., Cheminal, R., Rivier, F., Negre, C., Touchon, J., & Billard, M. (1992). Epileptic electroencephalographic abnormalities and developmental dysphasias: A study of 32 patients. *Brain Development*, *14*, 216–225.

Fabbro, F., Zucca, C., Molteni, M., & Renatto, B. (2000). EEG abnormalities during slow sleep in children with developmental language disorders. *Child Development and Disabilities*, *16*(1), 39–46.

Fuster, J. M. (2002). Frontal lobe and cognitive development. *Journal of Neurocytology*, *31*, 373–385.

Hick, R., Botting, N., & Conti-Ramsden, G. (2005). Cognitive abilities in children with specific language impairment: Consideration of visuo-spatial skills. *International Journal of Language and Communication Disorders*, *40*(2), 137–149.

Hill, E. L. (2001). Non-specific nature of specific language impairment: A review of the literature with regard to concomitant motor impairments. *International Journal of Language and Communication Disorders*, *36*(2), 149–171.

Johnston, J. R. (1994). Cognitive abilities of children with language impairment. In R. V. Watkins & M. L. Rice (Eds.), *Specific language impairments in children* (pp. 107–121). Baltimore, MD: Paul H. Brookes.

Kaufman, J., Csibra, G., & Johnson, M. H. (2005). Oscillatory activity in the infant brain reflects object maintenance. *Proceedings of the National Academy of Sciences of the United States of America*, *102*, 15271–15274.

Knyazev, G. G. (2012). EEG delta oscillations as a correlate of basic homeostatic and motivational processes. *Neuroscience and Biobehavioral Reviews*, *36*, 677–695.

Koch, H. A., & Duane, D. D. (2010, October). *Rate of persistent EEG abnormality in non-epileptic ADD/learning disability students*. Poster session presented at the 135th Annual Meeting of the American Neurological Association, San Francisco, CA.

Levy-Rueff, M., Bourgeois, M., Assous, A., Beauquier-Maccota, B., Boucheron, E., Clouard, C., & Robel, L. (2012). Abnormal electroencephalography results and specific language impairment: Towards a theoretical neurodevelopmental model? *L'Encephale*, *38*(4), 318–328. doi:10.1016/j.encep.2011.12.008

Licht, E. A., Jacobsen, R. H., & Fujikawa, D. G. (2002). Chronically impaired frontal lobe dysfunction from subclinical epileptiform discharges. *Epilepsy and Behavior*, *3*, 96–100.

Maccario, M., Hefferen, S. J., Keblusek, S. J., & Lipinski, K. A. (1982). Developmental dysphasia and electroencephalographic abnormalities. *Developmental Medicine and Child Neurology*, *24*, 141–155.

Parry-Fielder, B., Collins, K., Fisher, J., Keir, E., Anderson, V., Jacobs, R., & Nolan, T. (2008). Electroencephalographic abnormalities during sleep in children with developmental speech and language disorder. *Developmental Medicine and Child Neurology*, *51*(3), 228–234.

Picard, A., Cheliout Heraut, F., & Bouskraoui, M. (1998). Sleep EEG and developmental dysphasia. *Developmental Medicine and Child Neurology*, *40*, 595–599.

Powell, R. P., & Bishop, D. V. (1992). Clumsiness and perceptual problems in children with specific language impairment. *Developmental Medicine and Child Neurology*, *34*(9), 755–765.

Romine, C. B., & Reynolds, C. R. (2005). A model of development of frontal lobe functioning: Findings from a meta-analysis. *Applied Neuropsychology*, *12*(4), 190–201.

Rugland, A. L. (1990). Neuropsychological assessment of cognitive functioning in children with epilepsy. *Epilepsia*, *31*, 41–44.

Schmid, R. G., Tirsch, W. S., & Scherb, H. (2002). Correlation between spectral EEG parameters and intelligence test variables in school-age children. *Clinical Neurophysiology*, *113*(10), 1647–1656.

Selassie, G. R., Viggedal, G., Olsson, I., & Jennische, M. (2008). Speech, language and cognition in preschool children with epilepsy. *Developmental Medicine and Child Neurology*, *50*, 432–438.

Southgate, V., Csibra, G., Kaufman, J., & Johnson, M. H. (2008). Distinct processing of objects and faces in the infant brain. *Journal of Cognitive Neuroscience*, *20*, 741–749.

Stark, R., & Tallal, P. (1981). Selection of children with specific language deficits. *Journal of Speech and Hearing Disorders*, *46*, 114–122.

Stern, J. M., & Engel, J. (2004). *An atlas of EEG patterns*. Philadelphia, PA: Lippincott Williams & Wilkins.

Swingler, M. M., Willoughby, M. T., & Calkins, S. D. (2011). EEG power and coherence during preschoolers' performance of an executive function battery. *Developmental Psychobiology*, *53*, 771–784.

Tallal, P., & Benasich, A. A. (2002). Developmental language learning impairments. *Development and Psychopathology*, *14*, 559–579.

Thatcher, R. W., North, D., & Biver, C. (2005). EEG and intelligence: Relations between EEG coherence, EEG phase delay and power. *Clinical Neurophysiology*, *116*(9), 2129–2141.

Tomblin, B., Records, N., & Zhang, X. (1996). A system for the diagnosis of specific language impairment in kindergarten children. *Journal of Speech, Language and Hearing Research*, *39*, 1284–1294.

Trauner, D., Wulfeck, B., Tallal, P., & Hesselink, J. (2000). Neurological and MRI profiles of children with developmental language impairment. *Developmental Medicine and Child Neurology*, *42*, 470–475.

Tuchman, R. F. (1994). Epilepsy, language and behavior: Clinical models in childhood. *Journal of Child Neurology*, *9*, 95–102.

Vuković, M., Vuković, I., & Stojanovik, V. (2010). Investigation of language and motor skills in Serbian speaking children with specific language impairment and in typically developing children. *Research in Developmental Disabilities*, *31*, 1633–1644.

Webster, R. I., & Shevell, M. I. (2004). Neurobiology of specific language impairment. *Journal of Child Neurology*, *19*, 471–481.

Wheless, J. M., Simos, P. G., & Butler, I. J. (2002). Language dysfunction in epileptic conditions. *Seminars in Pediatric Neurology*, *9*(3), 218–228.