Cortical Auditory Evoked Potentials in Children with Developmental Dysphasia

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Abstract: Like all auditory evoked potentials, the cortical auditory evoked potentials are nonspecific for the disease, but they provide information about the auditory system function. It appears that the cortical auditory potentials can be used to study the disorders of speech comprehension and their pathology is related to the role of the temporal processing of the auditory stimuli. Cortical auditory potentials were studied in children with developmental dysphasia (DD) to examine maturation of the central auditory pathways. Study 1 (group of 6-7 yrs. old children with DD): the responses to verbal stimuli (P3 waves) were recorded with prolonged latencies from the left dominant hemisphere. Study 2: the latencies of P2 waves (to tonal stimuli) were being shortened within age - comparison of groups of 6-7 and 9-10 yrs. old children with DD. Great variability in P2 and P3 latencies, and their prolongation, compared to normal healthy children, reflects functional changes in the central hearing function. Latency differences may be related to a common temporal deficit to be one of the possible underlying factors in developmental dysphasia. The underlying phenomenon may be connected to cortical auditory processing.

Introduction

Developmental dysphasia (DD) denotes inability to acquire normal expression and comprehension of language in the absence of peripheral hearing impairment, neurological disorder and mental retardation. The use of the term of developmental dysphasia implies that the child's perceptual abilities for auditory speech events underlie his impairment for the acquisition of auditory symbols. At the heart of developmental dysphasia, there is disorder of auditory perception [1]. Central auditory deficit may result in/or coexist with difficulties in other CNS-based skills, such as speech-language impairment, attention deficit, learning and developmental disabilities.

Concerning developmental dysphasia, the typical clinical picture is delayed speech-language development, with specific disorders within all structures; disorder of the distinctive features of phonemes, disorders in the sequential arrangement of syllables (transpositions and reductions), problems with grammar (with word categories and with syntax), and problems with semantic and association language functions. These children experience typical speech comprehension problem, varying in intensity; they also have typical auditory decoding deficits, integration deficit, associative deficit and out-put organisation deficit of speech. Many of these children are unable to recognize acoustic contours and to identify keywords from a spoken message. These children often behave as if peripheral hearing loss was present, despite normal hearing. In some children, problems with perception are so conspicuous that the children appear as having a hearing disorder as they do not understand common conversation and elicit impression of the disorientation [2].

Like all auditory evoked potentials, the cortical auditory evoked potentials (CAEPs) are nonspecific for disease, but provide information about auditory system

function. CAEPs were studied in children with speech-language disorders. The author [3] explains the P3 (300) positive peak as a first phase of the language processing and he also considers the manifestation of the hemisphere dominance (Figure 1: normal characteristics of CAEPs=LAEPs, long latency auditory evoked potentials). The electrophysiological changes recorded correspond with the character of the respective speech disorders: it is apparent that the long latency auditory evoked potentials can be used to study the disorders of speech comprehension and their pathology is related to the role of the temporal processing of the auditory stimuli.

All studies presuppose that the children suffering from developmental speechlanguage disorders have also a comprehension disorder related to the defective temporal processing of the speech stimuli. Recently, the examination techniques for auditory evoked potentials have been increasingly used in connection with a monitoring of high-risk groups of children, including those with developmental speech-language disorders [4, 5].

Currently, the authors are mainly focused on monitoring of cortical auditory potentials. Analyzing the differences in latencies and amplitudes, Leppanen [6] published the cortical potential results obtained during the examination of children with developmental speech disorders and of patients with dyslexia. He attributed the changes to deficiencies in timings and he discussed the findings in relation to higher cognitive functions. He concluded that the brainstem auditory evoked potentials (BAEP) can be useful in the early identification of hearing impairments, but they are not effective in identifying infants at risk for later developmental problems like language impairment. He concluded that the cortical potentials are more important due to the capability to monitor abnormalities which can be explained by perception speech disorders.

The variability in topographic maps and in responses could be explained by a lack of synchronization, or by immaturity (maturation theory) or defective interconnection among the cortical regions and deeper structures. Korpilahti and

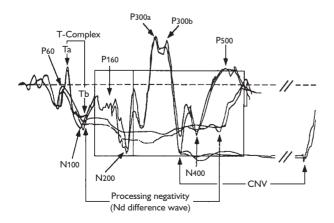


Figure 1 – Normal characteristics of CAEPs (=LAEPs); Mc Pherson: Late potentials of the auditory system. Singular Publishing Group, Inc., San Diego, 1996.

Lang [7, 8] studied the P3 waves and they revealed longer latencies, changes in the amplitude, impaired sensitivity, or impaired perception of the afferent system as being the possible defects related to sensory memory functions in developmental speech disorders. Tonnquist-Uhlen, Borg, and Spens [9, 10, 11] obtained topographic maps for the individual components of auditory evoked long-latency potentials in normal healthy children and in children with developmental language impairment; they focused on asymmetry in the cortical auditory potentials distribution and prolonged latencies in the left dominant hemisphere. The different results of auditory cortical responses in children with developmental language problems were attributed to a central diffuse affection of the CNS and to changes in the maturation of the central auditory pathways [12].

Speech-language comprehension disorders are typical in all patients with central auditory processing disorder. It is important to realize the matter of perception of the acoustic signal in connection with time. Temporal processing is a decisive factor for the myriad of auditory perceptions, including speech and music perception [13]. Temporal relations in the speech are the features, which are necessary for distinguishing the fine stimuli, such as voicing, and other. In other words, they are necessary for distinguishing between the distinctive features of the phonemes or to distinguish between similar words. The difficulties within final realization of sounds in dysphasic children persist despite improvement of their auditory differentiation – later on, the children have difficulties predominantly in the semantic domain (not being able to follow the story line and reproduce even a short story) [2].

Material and Methods

Cortical auditory potentials were studied in children with developmental dysphasia (DD) to compare the maturation of speech and tonal-evoked components. All individuals were audiologically normal according to pure-tone audiometry and tympanometry. Children were being best diagnosed in view of the fact of possibility to accomplish the widest possible spectrum of examinational methods. Majority of children underwent detailed phoniatric examination (analysis of language and speech functions, of phonological awareness, of auditory evoked potentials and dichotic central tests).

The next studies 1 and 2 concerned of different groups of children with DD. All results were compared with results of children with normal speech-language development and as statistical method was used t-pair test.

Study 1: In this study 30 subjects with developmental dysphasia from 6 to 7 years of age were studied. We used verbal stimuli and we recorded especially P3 cognitive potential (positive peak). The stimuli consisted of Czech words ("mama", "auto"; duration 400 ms), on 70 dB SPL, sweep time 1000 ms, input filter low (0.25–15 Hz), 1 impulse per 2 s, 50 repeats. Evoked potentials were investigated by the 2-channel ERA Madsen 2250 with monoaural, ipsilateral stimulation. Surface

electrodes (Ag/Cl) were applied to the vertex (+), ipsilateral mastoid (-) and to forehead (ground).

Study 2: In this study 33 subjects with developmental dysphasia from 6 to 10 years of age were examined with cortical auditory potentials. These subjects were divided to 2 groups – group A: 6–7 yrs. old children with developmental dysphasia, group B: 9–10 yrs. old children with developmental dysphasia. We used tone burst 2 kHz stimuli; equipment Evoselect ABR diagnostic system with the similar technical conditions as in study 1.

The equipment (for recordings of cortical auditory waves), used in these studies, was different. This ERA Madsen 2250, which was used in the first study, has the opportunity of speech stimulation. The Evoselect ABR from the second study is indeed technically better, but it lacks the mentioned possibility of stimulation by words.

Results

Study 1: The responses to verbal stimuli revealed delayed P3 latencies, almost in the left dominant hemisphere. When testing the hypotheses on differences between the values of the P3 latencies in children with developmental dysphasia (the P3 was chosen for testing as the most important response with respect to the stimulation by verbal stimuli), the following relationship was found: in the case of verbal stimulation – statistically significant differences were confirmed in the records of the P3 latencies from the left and right sides between the studied group and the control group (at 1% significance level). The record from the left hemisphere was statistically the most significant (p < 0.001) – Table 1.

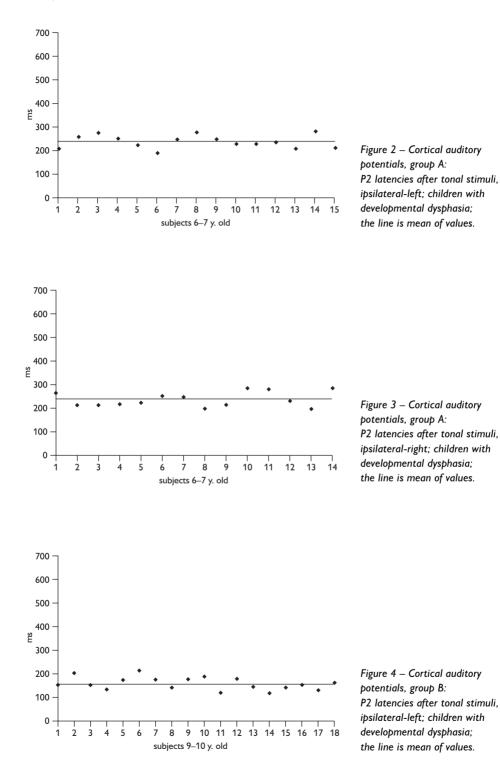
Study 2: We obtained average cortical waveforms in the response to 2 kHz tone burst; recorded latencies are shown in graphs of above mentioned developmental dysphasia groups A and B according to age. The positive of P2 waves was significant for both groups; P2 was the most distinctive. These waves were already stable without side differentiation. The latencies were being shortened within age and wave P2 appeared as the most important in 6–7 yrs. old children (group A) with developmental dysphasia. With increasing age (group B: 9–10 yrs. old children with DD) converged to the adult

Table 1 – CAEPs (cortical auditory evoked potentials) – results of latencies (in ms) of wave P3 after verbal stimulation

Group	Latency – dx	n	Latency – sin	n	
Control	321 ± 22	10	311 ± 28	10	
Dysphasia	410 ± 68	27	440 ± 62	31	

Mean and standard deviation (SD \pm) of values – monoaurally stimulation – right (dx), left (sin); statistically significant difference: p<0.001

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characteristics of cortical auditory evoked responses. P2 latency decreased from 236 ms at 6–7 yrs. of age to 163 ms at 10 yrs. The recorded waves P1, P2 and P3 were clearly visible from results of older children with developmental dysphasia.

All results of both groups A and B are presented in Figures 2–5. We recorded P2 latencies: group A (Figures 2 and 3) average: left side 236.8 ms, right side 235.3 ms; group B (Figures 4 and 5) average: left side 162.0 ms, right side 164.5 ms. Comparisons of recorded latencies are in Table 2 with statistically significant difference (p<0.001).

Recorded cortical auditory potentials as examples: Figure 6 – results of 6 yrs. old boy with developmental dysphasia: delayed peak of wave P2; Figure 7 – results of 10 yrs. old boy with developmental dysphasia: all waves P1, P2, P3 are stabilized. Control groups of 10 children with normal speech-language development had stabilization of P2 latencies (to tonal stimuli) sooner; that means in age of 6–7 years.

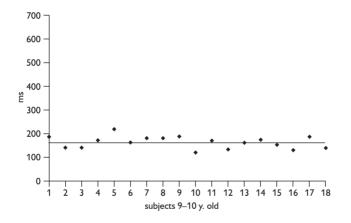


Figure 5 – Cortical potentials, group B: P2 latencies after tonal stimuli, ipsilateral-right; the line is mean of values.

Table 2 – CAEPs – latencies (in ms) of wave P2 after tonal stimulation 2 kHz

Group	Latency P2 dx	Ν	Latency P2 sin	Ν	
Control	156.5 ± 14.8	10	157.0 ± 15.4	10	
Dysphasia – group A	235.3 ± 28.5	14	236.8 ± 27.9	15	
Dysphasia – group B	164.5 ± 25.5	18	162.0 ± 28.0	18	

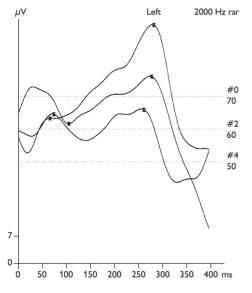
Mean and standard deviation (SD \pm) of values – monoaurally stimulation – right (dx), left (sin); comparison of results: control group: results of 6–7 y. old children with normal speech-language development; group A: results of 6–7 y. old children with developmental dysphasia; group B: results of 9–10 y. old children with developmental dysphasia; statistically significant difference (p<0.001)

Discussion and Conclusion

Our study was conducted to document and compare evolution of children with developmental speech-language impairment. Our results indicate the relationship between developmental dysphasia and central auditory processing disorder (CAPD). Central auditory processing disorders are associated with problems of comprehension, immediate or short-term memory and distraction from what is being provided or learned [14]. Many of these children are unable to recognize acoustic contours and to identify keywords from a spoken message. These children often behave as if peripheral hearing loss was present, despite normal hearing. With auditory training, common language skills of these children can be improved [15].

The recorded electrophysiological changes correspond with the character of developmental dysphasia; it appears that the auditory evoked long latency potentials can be used to study the disorders of speech comprehension (phonemic hearing disorders, disorders of basic phonemic comprehension during the speech perception) and their pathology is related to the role of the temporal processing of the auditory stimuli, especially more complex acoustic signals.

The occurrence of cortical auditory waves is limited and is influenced by the age. Great variability in P2 and P3 latencies, and their prolongation, compared to normal healthy children, was also found within the group of children with central



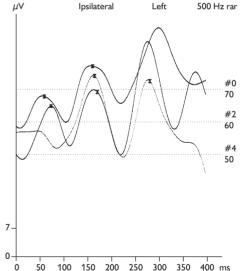


Figure 6 – CAEPs – example of the results: 6 y. old boy with developmental dysphasia; delayed peaks of waves, especially P2.

Figure 7 – CAEPs – example of the results: 10 y. old boy with developmental dysphasia – all peaks of waves P1, P2, P3 are stabilized in latencies.

auditory processing disorder. Despite the great variability in the clinical findings the long latency auditory evoked potentials reflect functional changes in the central hearing function [3, 5, 7]. It is possible to compare value of P3 cognitive wave with extend of developmental dysphasia. We can observe improvement in these older DD children. It concerns of school children with DD, who experienced learning disabilities very often (dyslexia, dysortographia).

The judgement of temporal order does not occur at the ear, it rather represents a central auditory function. Using a variety of acoustic stimuli, an inter-stimulus interval of only 2 ms is required for the normal listener to perceive two sounds instead of only one. However, this interval must be 17 ms long to identify correctly which sound appeared as the first one. If the listener needs more than 15 to 20 ms to realize the sequence of two consecutive stimuli, the examiner should check the central auditory system for pathologic changes. The first 100 to 250 ms of an auditory stimulus presentation is the most critical consequence for stimulus recognition [16]. Investigation of cortical auditory potentials seems appropriate to this knowledge.

The potentials display prolongation of the P2, P3 (P3 as cognitive potential) especially after stimulation with verbal stimuli. Evaluation of cortical potentials can contribute to the comparison of maturation of the central auditory system. The differentiation of cortical waves improved with age in children with developmental dysphasia, as well. The recording of these auditory waves is appropriate in older dysphasic children as a nonspecific sign of the disease, and it provides information about the central auditory system function.

Great variability in the responses is probably related to the variation in the bioelectrical activity, which can be seen also in abnormal native EEG records; this can be found in the results. Children with developmental dysphasia have difficulties in processing the speech signal. Ethiopathogenesis is unclear, however, a disorder of integration auditory and speech connections due to the defect in myelinization, delayed maturation of the unknown origin, or due to the relationships of pre- and postsynaptic elements is expected [12].

The shift of latencies in verbal and tonal stimulation is related to the action of endogenous factors within the dominant speech hemisphere. The shift might confirm the disorder of cognitive functions in children with developmental dysphasia, in particular speech perception disorder related to temporal processing disorder, delayed transmission and processing of the speech signal. The delayed auditory responses apply to higher auditory functions [13, 16]. The recordings confirmed delayed maturation of the central auditory pathways of these children.

As a result of our study we suppose that the very important matter for speech perception in children with developmental dysphasia is a disorder in temporal processing of acoustic signals. This presumption has also been confirmed by the results of examinations of the presented cortical auditory evoked potentials.

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