ABNORMALITIES IN CORTICAL AUDITORY RESPONSES IN CHILDREN WITH CENTRAL AUDITORY PROCESSING DISORDER

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Abstract-The main objective of the present study was to identify markers of neural deficits in children with central auditory processing disorder (CAPD) by measuring latency and amplitude of the auditory cortical responses and mismatch negativity (MMN) responses. Passive oddball paradigms were used with nonverbal and verbal stimuli to record cortical auditory-evoked potentials and MMN. Twenty-three children aged 9-12 participated in the study: 10 with normal hearing acuity as well as CAPD and 13 with normal hearing without CAPD. No significant group differences were observed for P1 latency and amplitude. Children with CAPD were observed to have significant N2 latency prolongation and amplitude reduction with nonverbal and verbal stimuli compared to children without CAPD. No significant group differences were observed for the MMN conditions. Moreover, electrode position affected the results in the same manner for both groups of children. The findings of the present study suggest that the N2 response could be a marker of neural deficits in children with CAPD. N2 results suggest that maturational factors or a different mechanism could be involved in processing auditory information at the central level for these children. © 2017 IBRO. Published by Elsevier Ltd. All rights reserved.

Key words: cortical auditory-evoked potentials, mismatch responses, central auditory processing disorder.

INTRODUCTION

Within the last decade, cortical auditory-evoked potentials have been used with clinical pediatric populations—with speech language impairment (Shafer et al., 2005, 2011), learning disabilities (Sharma et al., 2006), and hearing loss (Koravand et al., 2013)—to better understand how the central auditory system functions when listening to acoustic information. This valuable objective

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Abbreviations: CAPD, central auditory processing disorder; MMN, mismatch negativity.

measure is used to assess higher processing of auditory information in a specific time window (Cheour-Luhtanen et al., 1995). The benefit of these potentials is that they require less active participation than behavioral measures. These potentials are, however, underused in investigating the auditory functions of children with central auditory processing disorder (CAPD).

CAPD is characterized as difficulties processing auditory information despite having normal hearing (ASHA, 2005). In general, central auditory processing is clinically assessed using a number of behavioral tests. Unfortunately, the criteria for CAPD diagnosis are not universally accepted (Wilson and Arnott, 2013) and these tests can be affected by non-auditory factors such as attention (Gyldenkaerne et al., 2014; Riccio et al., 1994, 1996) and motivation (Silman et al., 2000). Tomlin et al. (2015) recently demonstrated that cognitive ability has a significant influence on CAPD test performance. Many children who scored low on auditory processing tests also showed poor cognitive results (Tomlin et al., 2015). One solution would be to use neurophysiological measurements to reduce the effect of cognitive ability on test results to a certain extent. Contrary to behavioral testing, these measurements of higher level auditory processing are less affected by attention especially in a passive listening task where attention is directed elsewhere.

explored studies objective Several have measurements of central auditory functions in children with CAPD using cortical auditory-evoked potentials (CAEPs) (Table 1). Jirsa (1992) conducted a study of 20 school-aged children (ages 9.5-12.5) diagnosed with CAPD who were matched with a group of 20 typically developing children. The children with CAPD were divided into two sub-groups: 10 children received individualized auditory training while the other 10 did not. The auditory training involved intensive listening exercises for auditory memory, auditory discrimination, attention, and language comprehension. The study examined CAEPs (N1 and P2) elicited by tone-burst stimuli at 65-dB nHL using the active oddball paradigm to record the P3 response (Pearce et al., 1989). The standard stimuli (1-kHz pure tone) occurred randomly with 80% probability and the target stimuli (3-kHz pure tone) occurred 20% of the time. To elicit the P3 responses, participants were instructed to keep a mental count of the number of target tones until a total of 300 trials were recorded in response to the target and standard tones. Latency (timing of stimulus processing, Luck, 2005) and amplitude (salience of processing, Luck, 2005) of the waveforms were analyzed.

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Pre-auditory training results, obtained from one electrode placed over the parietal vertex, showed a delay for N1, P2, and P3 latencies and a smaller P3 amplitude among the children in the CAPD group compared to the control group. Furthermore, only the experimental CAPD training group showed improved latencies and amplitudes at the post-training assessment. In contrast, the untrained CAPD children and the typically developing control group showed no change in auditory P3 latency or amplitude at the post-intervention assessment.

A study by Liasis et al. (2003) investigated cortical auditory responses in nine school-aged children (mean age: 9.5 years) with suspected CAPD who were matched with nine typically developing children using the passive oddball paradigm for recording mismatch negativity (MMN) (Näätänen et al., 1978). As with the P3 response, MMN is elicited by occasional deviant (target) stimuli embedded in a train of frequently presented standard stimuli (Näätänen, 1992); however, participants are instructed to disregard the both stimuli. MMN is obtained by subtracting the responses for standard stimuli from the results for the deviant responses (Näätänen, 1992). The subtraction process shows how the auditory system can automatically discriminate between the standard and the deviant stimuli (Alho, 1995). MMN, which is elicited passively, reflects the early sensory stages of sound processing and is ideal for investigating the mechanisms

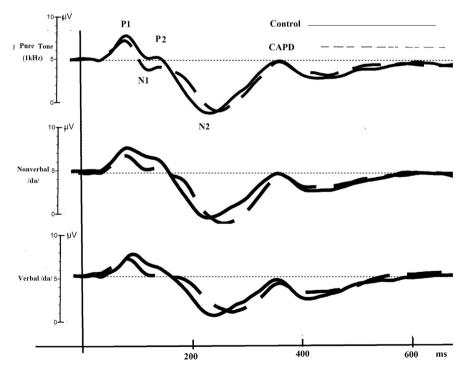


Fig. 1. Waveforms recorded at FCz electrode from two groups of children, 13 with normal hearing (Control) and 10 with central auditory processing disorder (CAPD), obtained with the standard stimuli, 1-kHz pure tone, nonverbal /ba/, and verbal /ba/. Stimuli were presented at 70 dB HL for normal hearing children (solid line) and for children with central auditory processing disorder (dashed line) in the passive listening paradigm. The top, middle, and bottom rows of waveforms display the four principal components, P1, N1, P2, and N2, observed with respectively the 1-kHz pure tone, the nonverbal /ba/, and the verbal /ba/ stimuli. The amplitude scale is 10 μ V and the latency range is -100 to 700 ms. The vertical gray bars in the waveforms are 200 ms time intervals. Linked mastoids were used as a reference electrode.

underlying auditory perception (Näätänen, 1992; Alho, 1995). In the Liasis et al. (2003) study, MMN was recorded with the standard stimulus (/ba/) with a 76% probability of occurrence and target stimulus (/da/) with a 24% occurrence. The MMN latency values revealed no significant differences between children with CAPD and their control peers. These results were replicated by Roggia and Colares (2008) in their study on children. ages 9-14 years old, with and without CAPD, using pure-tone stimuli (Table 1). However, unlike Liasis et al. (2003) and Roggia and Colares (2008) did not report the amplitude values of the cortical auditory responses. Using 20 electrodes, Liasis et al. (2003) analyzed the P1. N1. P2. and N2 latency of the auditory cortical responses recorded from the Fz electrode. Results showed that N1 latency was longer in the group of children with CAPD than the control group. P1, P2, and N2 latency values were similar in both groups, however, there were larger peak to peak amplitude of the P1-N1 and P2-N2 and smaller peak to peak amplitude of the N1-P2 for the CAPD children compared to their peers. Liasis et al. (2003) reported peak to peak amplitude rather than the amplitude values for each waveform. However, Fig. 1 in their study shows children with CAPD appear to have larger P1 and N2 waveforms-i.e. greater amplitude-than their typically developing peers.

Based on the findings of these studies, children with

CAPD show abnormal latencies for some cortical responses, however it still unclear whether the is amplitude of these components is affected. Passive change also detection, indexed by the MMN, durina passive auditorv discrimination shows no discrepancy between the values obtained in children with and without CAPD. However, the results of Sharma et al. (2006) contradict these findings. They revealed that the MMN amplitude was reduced in children with CAPD and a reading disorder in comparison with the value obtained for the control group. They used four electrode positions (F3, F4, Fz, and Cz) and several types of stimuli to elicit cortical auditoryevoked responses; the stimuli consisted of pure tones (1, 1.1, and 1.5 kHz), chords (a combination of the aforementioned pure tones), and verbal stimuli (/da/, /ga/, and / a/). They also showed that the four cortical auditory responses (P1, N1, P2, and N2) were generally similar for children within the experimental and control groups, regardless of stimulus type. Although the longer latency was recorded for the processing of verbal stimuli than the nonverbal, no differences were

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