The cortical evoked potentials in children with Developmental Coordination Disorder (DCD)

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Abstract

Purpose: Evoked potentials were recorded in patients with DCD to evaluate the integrity the afferent pathways and to rule out the presence of any neurological lesions.

Material and methods: Two boys: 5 and 16 years old with recognized DCD were examined. Battery of tests: short-latency somatosensory evoked potentials (SEP), pattern-reversal visual evoked potentials (VEP), cognitive event-related potentials (CERP) and EEG were recorded. CT and neuropsychological assessment were also performed.

Results: N20 and P25 latencies and also central conduction time of SEPs in both patients were longer. N9, N11, N13 latencies were normal. VEP, CERP, EEG and neuroimaging scans were normal.

Conclusions: Relationship among perceptual – motor skills, cognitive impairment and electrophysiologic findings in children with developmental dyspraxia are discussed. The disturbances of the integrity of the afferent pathways could to be one of many causal factors. Further researches are required to determine the specific source of the neurological deficit of clumsy children.

Key words: developmental dyspraxia, developmental coordination disorder, clumsy children, evoked potentials.

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Introduction

Developmental Dyspraxia has been recognized since very early twenty century, when Collier first described it as 'congenital maladroitness'. In 1937 Dr Samuel Orton declared it to be 'one of the six most common developmental disorders, showing distinctive impairment of praxis'. Since then it has been described and labeled by many, such as A. Jean Ayres, who in 1972 called it a disorder of Sensory Integration, or Dr Sasson Gubbay who in 1975 called it the 'Clumsy Child Syndrome' [1]. Other labels have included developmental disorder, sensorimotor dysfunction, minimal brain dysfunction, motor sequencing, minimal cerebral dysfunction, or sensory integration problems, clumsy children syndrome and most recently Developmental Coordination Disorder [2-4]. Although the diagnostic criteria appear to be similar, we are left with the question: are children who receive the diagnosis developmental coordination dyspraxia the same as those who receive the other diagnoses [3].

The American Psychiatric Association classifies these children and adolescents as having developmental coordination disorder (DCD), defined as "marked impairment in the development of motor coordination" [5]. It is estimated that 6% of children ages 5 to 11 in The United States have DCD [5] or 2-10% in Great Britain [6].

Dyspraxia can be defined as motor difficulties caused by perceptual problems, especially visual-motor and kinesthetic motor difficulties [7]. DCD is a disorder characterized by an impairment in the ability to plan and carry out sensory and motor tasks. Generally, individuals with the disorder appear "out of sync" with their environment. Symptoms vary and may include poor balance and coordination, clumsiness, vision problems, perception difficulties, emotional and behavioral problems, difficulty with reading, writing, and speaking, poor social skills, poor posture, and poor short-term memory. Although individuals with the disorder may be of average or above average intelligence, they may behave immaturely [2,7]. There is no consensus whether DCD is a physiological or developmental disorder or, if the disorder is physiological, whether it is mul-

Table 1. **SEP results of patient M.S.**

Latency (ms)	**N9**	**N11**	**N13**	**N20**	**P25**	**N9-13**	**N13-N20**
N.median right	7.85 ms	10.1 ms	11.0 ms	30.3 ms	38.1 ms	3.15 ms	19.3 ms
N.median left	9.75 ms	12.7 ms	15.6 ms	29.4 ms	34.9 ms	5.85 ms	13.8 ms

tisensory or unisensory. Children and adolescents with DCD may have problems with gross motor skills, fine motor skills, or both. Some have difficulty planning movements (dyspraxia) and executing them, others have difficulty planning movements but not executing them, and others have difficulty executing movements but not planning them. Children and adolescents with DCD should not be confused with those who do not perform motor skills as well as their peers. Children and adolescents with DCD have extreme difficulty acquiring new motor skills. Practice can help them, but it must be structured in specific ways to be effective. Motor skill development is slow for children and adolescents with DCD, and perceptual motor skills that are complex and/or require precise perception, such as writing between the lines on a sheet of paper, can be very difficult [2,3]. Children and adolescents with motor coordination problems are at risk for low academic performance, poor self-esteem, and inadequate physical activity participation. Unless there is intervention, their problems are likely to continue through adolescence. These children and adolescents are likely to avoid physical activity and experience frustration if they are forced to participate. Motor coordination problems do not resolve themselves, and children and adolescents do not outgrow them [8]. The assessment, etiology and treatment of DCD are discussed. Changes of cortical evoked potentials have been find in many disturbances. These electrophysiological tests seems to be useful also in children DCD. We recorded multimodal evoked potentials: visual somatosensory and cognitive event potentials in children with DCD to rule out the presence of any neurological lesions and to evaluate the integrity the afferent pathways.

Material and methods

Two boys 16 and 5 years old, with recognized DCD were examined. Neurological examination was normal. Assessment involved obtaining a detailed developmental history of the child and psychological profile. Neuroimaging was also performed. We recorded battery of electrophysiological tests including visual evoked potentials (VEP), short-latency somatosensory evoked potentials (SEP), cognitive event-related potentials (CERP) and electroencephalography (EEG).

Four-channel Sapphire Premiere MEDELEC was used for evoked potentials recording. Tests were performed according to standards accepted by most clinical laboratories [9-11]. In VEP checkboard pattern of black and white squares at reversal frequency of 2 Hz was presented. The responses were recorded from silver chloride electrodes on the scalp in occipital region in point Oz with referral electrode in Cz. During uninterrupted stimulation blocks of 128 responses were averaged. Peaks N1, P100 and P2 were analyzed [9,10]. In short latency SEP electrical stimulation of median nerve in at frequency 5 Hz and intensity ranges from 10 to 20 mV was used. The responses were recorded from electrodes located over ipsilateral Erb's point, the seven cervical vertebrae (C7), the second cervical vertebrae (C2) and contralateral somatosensory cortex (C3 or C4). The reference electrode was located in mid-front side (Fz). We used averaging 1000 responses. Latencies of potentials N9, N11, N13, N20 and P25 were analyzed [10-12]. In AudioCERP/P300 wave/stimuli frequent – 1000 Hz and rare – 2000 Hz, duration 50 ms and intensity 70 dBHL were presented in headphones. Rare stimuli were 15%, interstimulus interval 1.5 ms. The responses were recorded from active electrodes over the scalp: Cz and Fz with referral electrodes on earlobes. Peaks N1, P2, N2, P3 were analyzed [10,13].

Results

CASE 1. M.S., sixteen years old boy, from first normal pregnancy, delivery with caesarean section, 10 points of Apgar scale, body weight 3820 g. The psychomotor development and intellectual ability were normal, although from early childhood clumsiness, poor posture, coordination balance, walk awkward was observed. He also demonstrated difficulty in self-care tasks (dressing, using utensils) and academic tasks (handwriting, painting, organizing seatwork, gym class). No neurological and mental disorders were noted in his family.

Conventional neurological examination was normal. We observed slower movement time, clumsy movements of hands, difficulties in visual-motor coordination, problems with precise manual skills, rhythm of movements, repetition of learned tasks. He had difficulty in planning movements. Laboratory tests, computer tomography (CT) of CNS and EEG were normal. In psychologic assessment intellectual ability was average, visual-spatial disturbances without focal agnosia was found.

SEP results are presented in *Tab. 1*. The latencies of cortical component N20 and P25 were prolonged. Central conduction time (N13-N20 interlatency) was also prolonged. Latencies P100 component of VEP were slightly prolonged (left eye – 117.8 ms, right eye 114.8 ms), amplitudes (left eye – 7.69 μV, right – 7.66 μV) were low. CERP latencies: N1 – 116 ms, P2 – 203 ms, N2 – 258 ms and P3 (P300) – 333 ms were in normal range.

CASE 2. K.B., five years old boy was born from second normal pregnancy, delivery natural, 9 points of Apgar scale, body weight 3250 g. Hiperbilirubinenia since 4 day to 7 day of neonatal period, phototherapy was used. The motor development was late: sitting in 10 month, walking in 16 month of life. From early childhood he demonstrated clumsiness, difficulty in self-care tasks like dressing, using utensils and manual skills like

Table 2. **SEP results of patient K.B.**

Latency (ms)	**N9**	**N11**	**N13**	**N20**	**P25**	**N9-13**	**N13-N20**
N.median right	7.25 ms	8.65 ms	8.80 ms	16.90 ms	30.30 ms	1.55 ms	8.1 ms
N.median left	7.25 ms	8.70 ms	9.80 ms	18.90 ms	27.90 ms	2.55 ms	9.1 ms

throwing or catching a ball, holding properly a pencil, painting and drawing. He was confused about which hand to use.

Conventional neurological examination was normal. We found clumsy movements of hands, problems with catching of small things, lack of thumb opposition, difficulties in visual-motor coordination, problems with precise manual skills. Laboratory tests, computer tomography (CT) of CNS and EEG were normal. In ophthalmologic examination lower acuity of vision: Vod=5/10, Vos=5/6 (pictures) was detected. Abnormal ossification of lunar bone core in radiology of both hands was found. In psychologic assessment intellectual ability was above average (IQ=126 in Terman–Merrill scale), high developed verbal skills, manual and graphomotoric ability lower than average to his age were detected. In the drawings tendency to rotation was observed.

SEP results are presented in *Tab. 2*. The latencies of cortical component P25 were prolonged, other were in normal range. Central conduction time (N13-N20 interlatency) was also normal. Latencies and amplitudes P100 component of VEP were in normal limits (left eye – 102 ms, 8.94 μV right eye – 104 ms, 8.64 μV). CERP Latencies: N1 – 87 ms, P2 – 150 ms, N2 – 221 ms and P3 (P300) – 301 ms were in normal range.

Discussion

Early studies in this field explored a variety of methods for identifying and describing children with DCD. Since there are no clear-cut criteria which define clumsiness and there is no "generally accepted" level of motor proficiency, it was found that the characteristics of children who were identified as having DCD depended upon the source of referral, the professional discipline of the researchers, and the types of assessments used [14]. Identification and assessment continues to be a major source of debate in the field and is confounded by the use, in different disciplines, of terminology or assessment methods which imply causation: DCD has no known cause. All children with DCD have some impairment of motor skill, in the absence of other physical and intellectual disorders; however, they are certainly not an homogeneous group. The only characteristic that has been demonstrated consistently in empirical studies is that children with DCD have slower movement time, regardless of the type of task or how it is taught or measured [15,16]. A recent trend in the research is to attempt to define subtypes of children within the DCD classification [17], in the hope that this may contribute to our understanding of why many treatment methods have been largely ineffectual.

After conducting a series of research studies, Laszlo and colleagues [18] have argued strongly for kinesthetic dysfunction and Hulme and colleagues [19] for visual perceptual dysfunction as the underlying problem in children with DCD. The kinesthetic findings have since been called into question [17] and there is evidence that most children with DCD do not have any visual acuity or other ophthalmologic problems [20]. The possibility of visual processing difficulties, however, remains an area of controversy that is continuing to be investigated [16]. At this time, evidence is mounting which suggests that children with DCD may rely more heavily on visual feedback for movement control [21] and that they may not use rehearsal strategies to retain visual information in memory [22].

Diagnosis of DCD should be confirmed by The Movement Assessment Battery for Children (MABC), a ball-catching test, a jumping test, a timed response task to a visual moving stimulus and The Beery-Buktenica Developmental Test of Visual Motor Integration, incorporating copying, visual discrimination and tracing tasks [23]. Children with DCD performed significantly worse than the control group on all measures. The visual discrimination task did not correlated significantly with any of the motor tasks and visual timing task correlated significantly with the ball-catching test in the DCD group [23].

Evoked potentials could be recorded to evaluate the integrity of the sensory pathways and to rule out the presence of any neurological lesions. In our patients we recorded prolonged latencies of cortical components of SEPs and prolonged central conduction time in patient No. 1 M.S. It could suggest possibility of disturbances in somatosensory pathways. We didn't find any studies in literature in this field. Only in patient M.S. VEP latencies were slightly prolonged. In patient K.B. VEP were normal. In Mon-Wiliams and colleagues study [24] pattern onset VEP were recorded in 14 children with DCD aged between 5 and 7 years, and age-matched control group using pattern onset, high contrast stimuli. Inattention and movement artifact meant that VEPs were more difficult to record within the DCD group resulting in smaller amplitudes of the waveform but no significant differences in the implicit times were observed between the DCD groups and control [24]. Normal CERP results, particularly P300 wave latency did not show any disturbances of cognitive function and processing information in DCD patients.

Results of research studies concerning causes and mechanisms of DCD are inconclusive. Further researches are required to determine the specific source of the neurological deficits in DCD but a problem with the integrity of the afferent visual and sensory pathways does not appear to be the main causal factor. Focused research will lead to greater understanding of the characteristics and needs of children with DCD.

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