EVENT RELATED BRAIN POTENTIAL (ERP) CORRELATES OF ACTIVATION AND EFFORT IN STATE REGULATION AMONG CHILDREN WITH ATTENTIONAL PROBLEMS

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Tässä opinnäytetyössä tutkittiin tarkkaavaisuushäiriöisten (attention deficit hyperactivity disorder, ADHD) lasten kahta aivojen herätevastetta, CNV:tä (contingent negative variation) ja P300:a, kahden esitystahdiltaan hitaan, jatkuvaa tarkaavuuden ylläpitoa vaativan antisipaatioparadigmaa mukailevan tehtävän eli CPT:n (continuous performance task) aikana. Vertailuryhminä toimivat normaalit ja lukihäiriöiset lapset. Tutkittavia lapsia oli yhteensä 59 ja he olivat iältään 8-16 vuotiaita. Tarkkaavaisuushäiriöisillä lapsilla oletetusti ilmenevää tilansäätelyn ongelmaa lähestyttiin Van der Meeren (1996) aktivaation/suorituspyrkimyksen toimintahäiriön hypoteesin ja näitä oletetusti vastaavien herätevasteiden kautta. Analyyseihin sisällytettiin käyttäytymisen mittareina keskimääräiset reaktioajat ja virheiden kokonaismäärä, joiden avulla oli mahdollista saada yksityiskohtaisempia arviointeja herätevasteiden suhteesta suoriutumiseen. Toiseen tehtävään liitettyjen motivaatiotekijöiden odotettiin kohottavan sekä CNV että P300 amplitudeja lisääntyvän aktivaation ja suorituspyrkimyksen kautta. Vain toisessa tehtävässä esiintyi määritelmän mukaista CNV:tä, mikä tukee olettamusta, että motivaatiotekijät lisäävät aktivaatiota. Havainnot ovat yhdenmukaisia aiempien CNV:stä tehtyjen tutkimusten kanssa, jotka pitävät vastetta aktivaation ja motorisen valmistautumisen heijasteena. P300 amplitudit osoittautuivat kuitenkin olettamusten vastaisiksi, sillä mitatut amplitudit olivat suuremmat ensimmäisessä kuin toisessa tehtävässä. Keskimääräiset reaktioajat pidentyivät toisessa tehtävässä tilastollisesti merkittävästi vain tarkkaavaisuushäiriöisten ryhmällä. Virheiden määrä väheni kaikilla ryhmillä. Tulokset tukevat osittain Van der Meeren hypoteesia aktivaation/suorituspyrkimyksen toimintahäiriöstä tarkkaavaisuushäiriöisillä lapsilla. Kyseenalaisena säilyy kuitenkin P300:n ja suorituspyrkimyksen välinen suhde.

Avainsanat: CNV, P300, CPT, tarkkaavaisuushäiriö, tilansäätely, aktivaatio, suorituspyrkimys, motivaatiotekijät

Event related brain potential (ERP) correlates of activation and effort in state regulation among children with attentional problems

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Two event related potential (ERP) components, contingent negative variation (CNV) and P300, were examined during two slow event-rate continuous performance tasks (CPTs) in three groups of children (n = 59) aged 8 - 16 years. The clinical group consisted of children suffering from attention deficit hyperactivity disorder (ADHD). Reading disabled (RD) along with normal children were used as reference groups. The supposed state regulation problem of ADHD children was approached by utilizing Van der Meere's (1996) hypothesis of activation/effort dysfunction and its assumed psychophysiological correlates. Behavioural measures in the form of mean reaction times (RTs) and total amount of errors were included in the analyses to evaluate the relation of the ERPs to performance in more detail. Motivational factors added to the second task were expected to enhance both slow wave and P300 amplitudes via increased activation and effort. Support for the research hypothesis was provided on behalf of CNV proper, which was found only in Task 2 in all groups. In the ADHD group the slow wave enhancement was the weakest. Results concerning the P300 amplitudes proved to be unexpected and contrary to the hypothesis. The amplitudes were larger in Task 1 than in Task 2. Mean RTs lengthened only within the ADHD group in the second task while the amount of errors decreased in all groups. These findings are congruent with previous studies of CNV as a reflection of activation and motor preparation and, in some parts, support Van der Meere's hypothesis of activation/effort dysfunction in ADHD children. The role of P300 as a correlate of effort remains questionable.

Keywords: CNV, P300, CPT, ADHD, State regulation, Activation, Effort, Motivational factors

Introduction

The objective of the present study was to investigate the underlying psychophysiological mechanisms of executive functioning and state regulation in learning disabilities placing special emphasis on attentional problems. These are becoming increasingly evident among school-aged children and clarification of the problematic factors underlying attention deficit hyperactivity disorder (ADHD) (DSM-IV, American Psychiatric Association, 1994) would provide better understanding and treatment of the disorder. Our research hypotheses were based on Van der Meere's (1996) theory of state regulation, which is derived from Sanders's (1983) and Mulder's (1986) model of cognitive-energetic information processing. We approached the theory from a psychophysiological point of view and concentrated on the brain's event-related potentials (ERPs) in electroencephalography (EEG), linking behavioural measures to them. Special emphasis was on two ERP components, the positive P300 and the slow waves, especially contingent negative variation (CNV). A slow event-rate continuous performance task (CPT), which is assumed to expose attentional problems, was used in the present experiment (Van der Meere, 1996). In addition, the effect of motivational factors was taken into account.

Executive functioning, which is considered to reflect the functioning of the prefrontal areas of the brain, is defined as goal-directed behaviour including, among others, planning, organized search, and impulse control (Welsh, Pennington & Groisser, 1991). A possible prerequisite of executive functioning is state regulation. It refers to energy mobilization, which is essential in order to change the current state of the organism according to situation and task demands (Van der Meere & Stemerdink, 1999).

A non-optimal arousal state has a long tradition as a physiological concept underlying attentional deficits (reviewed by Douglas, 1983). Dissatisfaction with this unitary state theory has led to the development of a variety of cognitive multi-state models, which argue that there are two distinguishable and different psychological states, arousal and activation. Pribram and McGuinness (1975), Sanders (1983) and Mulder (1986) have postulated also a third energetic system, i.e., the effort system, which is, in turn, under the control of an evaluation system. The subject's arousal and activation state is scanned by the evaluation system, and suboptimal state is compensated with effort. The cognitive-energetic model of information processing and state regulation from Sanders (1983)

and Mulder (1986) deals with the distinction between process and state. Processes mediate between the stimulus and response while state fluctuations are not directly involved in information processes but modulate the cognitive operations.

Van der Meere (1996) stresses that the information processing approach may be helpful in identifying the assumed cognitive deficits of hyperactive children since it unravels cognition into smaller components. Task inefficiency, a typical feature in hyperactivity disorder according to Van der Meere (1996), may be considered to be caused by limitations at the cognitive or at the state level of information processing, or in a combination of both levels. He also assumes that the task inefficiency in hyperactive children involves an activation/effort dysfunction.

Time-on-task effect in a CPT is considered to be a measure of sustained attention. Depending on the rate of stimuli in CPTs, the time-on-task exerts its influence on either arousal or activation (Van der Meere, 1996). No differences in sustained attention have become evident between hyperactives and normals when using fast event-rate and therefore, according to Van der Meere (1996), inadequate arousal state does not seem to explain the deficit. He states that a rapid presentation rate improves ADHD children's performance via increased activation. Slow event-rate, in turn, possibly underactivates ADHD children and causes their slow and inaccurate responding (Van der Meere, 1996; Van der Meere & Stemerdink, 1999). The slow motor preparation and execution processes are seen as manifestations of a state regulation deficit and hence, it has been concluded that a non-optimal state serves as a physiological basis of hyperactivity disorder (Van der Meere, 1996; Van der Meere & Stemerdink, 1999; Van der Meere, Stemerdink & Gunning, 1995*b;* Yordanova, Dumais-Huber & Rothenberger, 1996).

Referring to Van der Meere (1996), the effort mechanism is influenced by motivational factors such as knowledge of results, absence/presence of the experimenter and reward/non-reward condition. However, basing his arguments on Sanders (1983), Van der Meere claims that children with ADHD are relatively unaffected by the reward in the task but that external control may modify the level of their activation. He further suggests that external control, in itself, does not prevent the sustained attention deficit, but it improves task performance, which still remains below the level of normals'. ADHD children treated with a stimulant called methylphenidate do not show any attentional decrement compared to normal children neither in the experimenter-absent nor -present condition due to normalized activation level (Van der Meere, 1996; Van der Meere, Shalev, Börger & Gross-Tsur, 1995*a*).

Besides through behavioural expressions, activation and effort may also be manifested in the brain's electrical activity. The P300 component, a positive peak in an ERP, is assumed to reflect brain functions associated with context and memory updating and the amount of both voluntary and involuntary attention allocated to the stimulus processing (Donchin & Coles, 1988; Picton & Hillyard, 1988). The latency of P300 normally varies between 250 and 350 msec, but may sometimes reach even 600 msec being a possible representation of stimulus evaluation time. Coles, Gratton and Fabiani (1990) and Duncan-Johnson and Donchin (1977) have suggested that the amplitude is affected by task relevance of the eliciting event and the probability of the stimulus. Van der Meere (1996) instead considers larger P3b amplitude as a reflection of greater effort invested in the task.

There has been some evidence of smaller P300 amplitude in children suffering from ADHD (Kemner, Verbaten, Koelega, Camfferman & van Engeland, 1998; Klorman, 1991; Strandburg *et al.*, 1996), which according to Van der Meere (1996), is consistent with the effort hypothesis. Michael, Klorman, Salzman, Borgstedt and Dainer (1981) and Klorman (1991) on their behalf have shown that stimulant administration enlarges the amplitude and improves ADHD children's CPT performance, which could be interpreted as increased activation state. It is therefore questionable whether P300 amplitude is, among others, a sign of effort or, alternatively, a representation of activation.

A study by Holcomb, Ackerman and Dykman (1985) on P300 latencies showed that both children with ADHD and children with RD exhibited slower P300 latencies than nondisabled children. Studies by Sunohara, Voros, Malone and Taylor (1997) and Taylor, Voros, Logan and Malone (1993) on tasks that require high degrees of sustained attention have also revealed that P300 is at a longer latency in ADHD subjects without medication when compared to control and ADHD children with medication. Results have, however, been controversial in the literature.

In addition to P300, also CNV may reflect state regulation. The CNV is a slow negative potential in an ERP depending upon the association of two successive stimuli. It appears about 400 msec after the onset of a warning stimulus (S1) and terminates with an imperative stimulus (S2) to which the subject makes a motor response (Rockstroh, Elbert, Canavan, Lutzenberger & Birbaumer, 1989; Tecce, 1970). Hillyard (1973) has proposed different settings in which CNVs are generated, e.g., when holding a motor response in readiness, anticipating a reinforcer, or preparing for a cognitive decision. Van der Meere (1996) stresses the importance of CNV as a sign of activation and especially, motor preparation. The CNV consists of two independent components, an early orienting wave (O-wave) and a later expectancy wave (E-wave), when the inter-stimulus-interval is 3 seconds or more (Loveless & Sanford, 1974). The O-wave is claimed to be influenced by S1 parameters and related to orienting and activation (Weerts & Lang, 1973). The

E-wave in turn is assumed to be influenced by the S1-S2-response contingency (Loveless, 1977). Rohrbaugh and Gaillard (1983) have concluded that it is explicitly the later component that reflects preparation for optimal, effective motor responses. In their Master's Thesis, Kivijärvi and Saunamäki (1999) argue that motivational factors, such as pay-off, knowledge of results and experimenter's presence enhance the CNV frontally in a CPT.

Studies on CNV in hyperactive children are rather rare. With respect to the findings by Grünewald-Zuberbier, Grünewald, Rasche and Netz (1978), it appears that hyperactive children have smaller CNV after S1 and also lower pre-S2 rise than controls, whereas Strandburg and colleagues (1996) have found normal CNV in ADHD children. The existing results of CNV's associations to hyperactive and inattentive behaviour appear controversial and need further investigation.

Van der Meere (1996) has based his hypotheses of the role of CNV and P300 on earlier studies but has not confirmed them empirically. In our study, the focus is on viewing his activation/effort hypothesis from a psychophysiological standpoint. We apply two-stimulus anticipation paradigm CPTs with and without motivational factors and concentrate on the P300 and the late-CNV (E-wave) in the EEG in children with attentional problems, reading disabled (RD), and normal children. The children with attentional problems are not strictly diagnosed according to the DSM-IV, but they will be concidered as ADHD group below. The RD group is used as a reference group to see whether the outcoming differences are specific to ADHD or concern all learning disabled children. The two-stimulus paradigm is not commonly used in eliciting P300 and thus, our P300-like component, interpreted as P300, deviates slightly from the traditional P300 provoked by an oddball paradigm. The effort expected to increase in Task 2 is presumed to be reflected in higher P300 amplitudes. Improved motor preparation via increased activation in the second task is assumed to be seen in enhanced CNVs. Experimenter's presence, feedback and pay-off as motivational factors are expected to increase effort and/or activation in normal and RD children and we examine whether the same effect is visible in the ADHD group. Behavioural measures that are reaction time (RT) and the amount of errors are used in confirmation of conclusions concerning the ERPs.

Method

Subjects

All subjects of the present study participated in the experiment as rewarded volunteers. The ADHD and RD children were recruited from the local learning disability center, the Niilo Mäki Institute (NMI) and central Finland's MBD-association. The control group was obtained from the local primary school. All subjects were administered two sub-tests of the Wechsler Intelligence Scale for Children-Revised (WISC-R), block design and vocabulary, to ensure their normal intellectual abilities (Wechsler, 1984). Attentional problems were screened from the participants by their parents and teacher by administering the Finnish translation of the Conners scales (Goyette, Conners & Ulrich, 1978). To evaluate reading abilities, the subjects were tested for word recognition and comprehension of sentences (Lindeman, 1998), as well as spelling (Häyrinen, Serenius-Sirve & Korkman, 1999). On the basis of the results of these tests, some of the subjects were either regrouped or removed from the analysis. Consequently, three groups were formed.

All subjects scoring at least one point five standard deviation above local norms on the Conners hyperactivity factor in both questionnaires were placed in the ADHD group consisting of 13 children. The norms were based on the calculated means of 8 -12-year-old local school children. The clinical control group consisted of 14 reading disabled children including all children whose reading ability scores were on either one of the two lowest levels out of nine. Approximately 11 % of the population fall into this category. In case of overlapping ADHD and RD diagnoses the final placement was in the ADHD group. The ADHD group consisted of 11 males and 2 females, the mean age being 11.7 years and ranging from 8.8 to 15.8 years. The RD group comprised 11 males and 3 females, whose mean age was 13.6 years ranging from 8.7 to 16.6 years. Gender distribution in the control group was 15 males and 17 females. Their ages ranged from 8.2 to 13.2 years and the mean age was 10.8 years. Total number of subjects was 59.

Design and Procedure

The subjects performed the tasks in a sound attenuated and electrically insulated room. They were seated in front of a display by a table to which the response button was attached. Connection between the experimental cabin and the adjoining laboratory was arranged with a bi-directional communication system.

The set of experiments started with a 5-minute heart rate (HR) baseline recording and was followed by an 11-minute mismatch negativity (MMN) measurement. Subsequently, two 29-minute slow event-rate CPT's following the two-stimulus anticipation paradigm (S1-S2) were administered after which a HR-baseline was measured again. Results of the HR and MMN measurements will be considered elsewhere.

The CPTs were preceded by instructions emphasizing fast and accurate responses and informing the subjects to adopt a relaxed position and to avoid

unnecessary body movements. Subjects were to concentrate on S1, which was a "plus" symbol in the centre of the screen displayed during the whole course of the trial. The S2 was either an asterisk, the probability of which was 75 %, demanding a fast button press or, alternatively, a circle with a probability of 25 % requiring response inhibition. An approximately 5-minute training session with the experimenter preceded the tasks to ensure the subjects' full comprehension of the rules. A total of 240 trials were presented throughout both tasks. Every second trial was a fixed duration (6500 msec) and every second was variable (5500, 6500, 7500 or 8500 msec). Only the fixed duration trials were taken into account in the data analyses.

Task 1 and Task 2 were similar to each other with the exception that during the first task the subjects stayed alone in the cabin having no knowledge of their performance, whereas in the second task external control, feedback and pay-off were included as motivational factors. At the onset of Task 2 the subjects had 48 FIM and the experimenter was entitled to subtract 2 FIM per error. Omissions (misses) and commissions (false alarms) were considered as errors. A feedback bar indicating remaining money was provided during the variable trials.

The design and procedure as well as the electrophysiological recordings are described in more detail in Kivijärvi and Saunamäki's (1999) Master's Thesis.

Electrophysiological recordings

The control of experiments, presentation and timing of stimuli and storage of the behavioral responses was managed with an Amiga 2000 computer. Bio-Logic Brain Atlas-system was on the basis of the EEG-recording system. Data acquisition of electrophysiological responses was conducted with DSAMP software run on a 233 MHz Pentium PC.

The EEG was recorded by using an EEG-cap (ECI) and according to the international 10-20 system (Jasper, 1958) mid-sagittally at frontal (Fz), central (Cz) and parietal (Pz) electrodes. The lateral electrode placements were at C3 and C4. The electro-oculograph (EOG) was obtained with disposable electrodes positioned below the canthus of the right eye and above the canthus of the left eye approximately 2.0 cm from the pupil. Linked mastoids were used as references for both EEG and EOG recordings. The ERP-trial sampling was begun 2500 msec before the onset of S1 and ended 3000 msec after the S2 onset, resulting in a total trial length of 12000 msec.

Data Reduction and Analysis

Raw EEG data was managed with DSAMP program. EOG shifts exceeding ±70 μV in ERP trials were excluded from the CNV analysis. However, in the P300 analysis, trials including eye movements only during 100 - 1500 msec after the onset of S2 were removed. To investigate time-on-task effect, both task conditions were split into two 60 trial blocks for the CNV analysis. More trials remained in the P300 data and therefore it was possible to examine it in three 40 trial blocks, which allowed more specific inspection of time-on-task effect. Bad data quality or insufficient number of uncontaminated trials led to the discarding of 15 subjects out of the original sample.

When investigating slow wave potentials, statistically significant mean negative shifts during three intervals (1500 - 2000, 3750 - 4250 and 6000 - 6500 msec) were used in the definition of CNV. The simultaneous offset of S2 and the onset of S1 caused an overlapping effect on the early ERP components. Setting the ERP-baseline at the onset of S1 would have resulted in a baseline placed on the top of the responses evoked by S2, mainly P300. In order to avoid the effect's influence on the baseline, it was set at the onset of the S2 of the preceding trial. In statistical analysis of P300, the time window for Fz, C3, Cz and C4 channels was 100 - 1500 msec following the onset of S1. The overlapping S2-S1 effect was manifested most visibly parietally as the P300-like double wave. Hence, for the investigation of the two positive P300-like peaks at Pz, two separate time windows (100 - 600 and 600 - 1500 msec) were used in the analysis.

Multivariate analyses of variance (MANOVA) were performed using SPSS 8.0 for Windows software. When investigating the slow waves by MANOVA, the within subject factors were task (two levels; Task 1 and Task 2), block (two levels), and interval (three levels) and the between subjects factor was group (three levels). After the initial analysis, a one-way analysis of variance (ANOVA) was run in order to explore in which parts of the brain the CNV is reached at. To gain more specific information of the potential differences between the two tasks, a paired samples t-test was conducted for all groups to inspect the respective intervals in all EEG channels. A MANOVA design was also used for the P300 analysis investigating task (two levels; Task 1 and Task 2) and block (three levels) as the within subjects factors, and group (three levels) as the between subjects factor. Both the amplitudes and the latencies of the P300 were inspected but the emphasis was on the amplitudes. The latencies were used mainly for the inspection of associations with reaction times.

Behavioural measures were included in the analyses in the form of mean reaction times and the total amount of errors in the CPT. A MANOVA procedure was run for task (two levels; Task 1 and Task 2) and block (three levels) as the within subject factors and group (three levels) as the between subject factor. A t-test was conducted to examine whether the groups differed in mean reaction times and the total amount of errors in

both tasks. Pearson correlation coefficients were calculated between slow wave amplitudes (intervals 2 and 3) and RTs as well as between P300 amplitudes and RTs. Pearson correlation was also used to measure relations between P300 latencies and RTs. In addition, it was examined whether the amount of errors was associated with CNV and/or P300.

Results

MANOVA revealed no CNV proper in any group in Task 1. In Task 2, a significant increase in negativity indicating CNV was found in the control and RD groups at Fz ($F(2,30) = 18.8$, $p < .001$ and $F(2,12) = 15.8$, $p < .001$, respectively), Cz ($F(2,30) = 6.0$, $p < .01$ and $F(2,12) = 5.6$, $p < .05$, respectively), and at C3 ($F(2,30) = 5.1$, $p < .05$ and $F(2,12) = 7.0$, $p < .05$, respectively). In the ADHD group CNV was observed only at Fz ($F(2,11) = 4.7$, $p < .05$).

MANOVA did not show any clear time-on-task effects in CNV between the first and the second half of the tasks in any group and thus, only the task effects were investigated. Figure 1 presents the task averages of the control, ADHD, and RD groups. A significant main effect for task was found indicating level differences in slow wave potentials between the tasks in the control and RD groups at Fz ($F(1,31) = 12.1$, $p < .01$ and $F(1,13) = 8.3$, $p < .05$, respectively). The negativity was enhanced in Task 2. This effect was not visible in the ADHD group.

Level differences of slow negative potentials were examined with a paired-samples t-test between Task 1 and Task 2, and as noticeable in Figure 1 (a,b,c), the first interval (1500 - 2000 msec) shows no differences. In the control group the second (3750 - 4250 msec) and third (6000 - 6500 msec) interval at Fz ($t(31) = 4.3$, $p < .001$ and $t(31) = 5.2$, $p < .001$, respectively) and the second interval at Cz ($t(31) = 2.4$, $p < .05$) showed significant potential differences being more negative in Task 2. The RD group exhibited similar results on behalf of Fz ($t(13) = 2.8$, $p < .05$ and $t(13) = 2.7$, $p < .05$, respectively) and the third interval at C4 ($t(13) = 2.2$, $p < .05$). In the ADHD group the only statistically significant potential difference was reached at the third interval at Fz, which was also greater in negativity in Task 2 ($t(12) = 2.7$, $p < .05$).

In all groups, the P300 amplitude analysis yielded a main effect for task, which was seen as larger amplitudes in Task 1 than in Task 2. The task average curves and the amplitude (μV) values of channels that gained significance in the analysis are illustrated in Figure 2. In the control group the task effect was seen at all channels, being significant at Fz ($F(1,31) = 7.6$, $p < .01$), C3 ($F(1,31) = 17.5$, $p < .001$) and at the first time window of Pz ($F(1, 31) = 22.7$, $p < .001$). The same tendency was found in the RD and ADHD

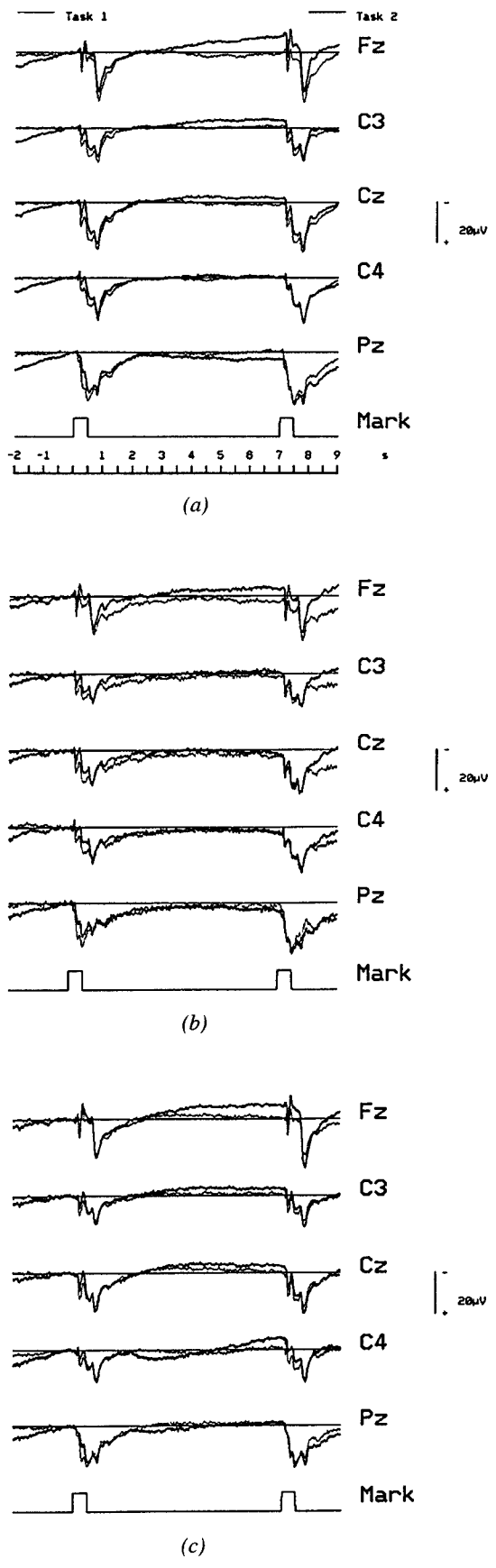

Fig. 1 Across (a) control (b) ADHD, and (c) RD subjects averaged slow wave responses during Task 1 and Task 2.

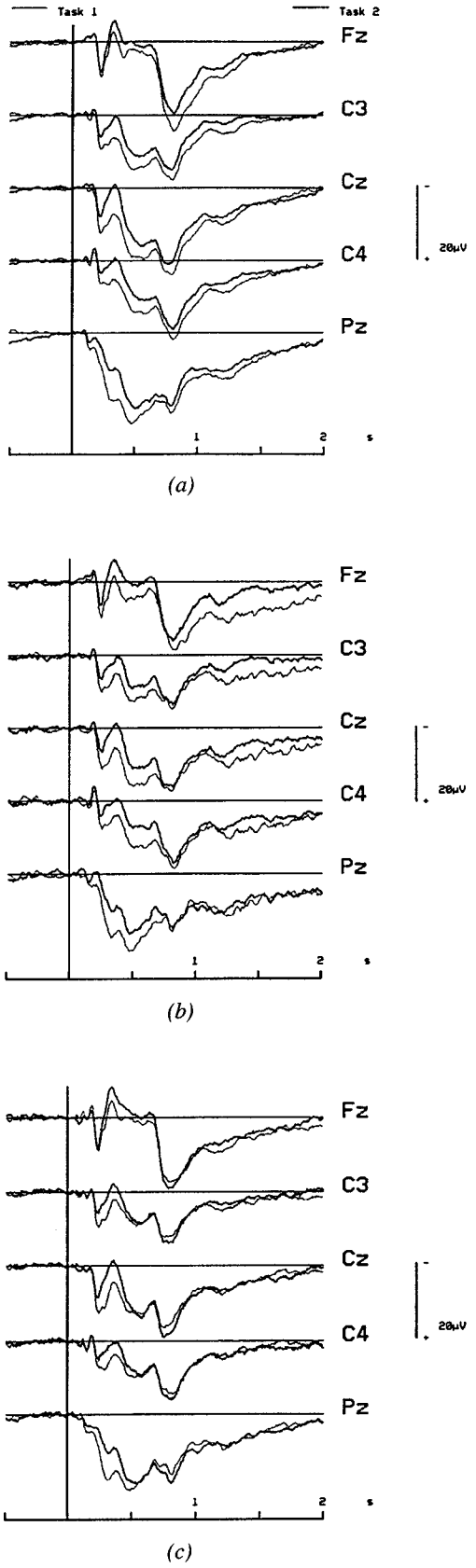

Fig. 2 Across (a) control, (b) ADHD, and (c) RD subjects averaged P300 trials. Significant amplitude differences found in Task 1 and 2 are (a) at channels Fz 30.0 and 29.2 μV, at C3 25.4 and 24.1 μV and at Pz 30.6 and 28.7 μV, (b) at Pz 26.3 and 22.3 μV, and (c) at Pz 24.8 and 22.2 μV, respectively.

groups, but the amplitude reduction was significant only parietally, at the first time window of Pz ($F(1,13) = 5.4$, $p < .05$ and $F(1,12) = 6.3$, $p < .05$, respectively). In the ADHD group there was also a main effect for block, that is, the P300 amplitudes decreased at all channels in the passage of time in both tasks. Significant difference was reached at Cz ($F(2,11) = 7.4$, $p < .01$), C3 ($F(2,11) = 11.5$, $p < .01$), C4 ($F(2,11) = 7.0$, $p < .05$) and at the first time window of Pz ($F(2,11) = 30.2$, $p < .001$). Between groups comparisons of amplitudes did not reveal any significant differences.

A main effect for task was found also in the P300 latency analysis. The latencies were slightly shorter in Task 1 than in Task 2. Significant differences were observed in the control group at Cz ($F(1,31) = 4.7$, $p < .05$) and at the first time window of Pz ($F(1,31) = 10.2$, $p < .01$). The same effect was seen in the RD group at C3 ($F(1,13) = 5.9$, $p < .05$) and in the ADHD group at the first time window of Pz ($F(1,12) = 5.4$, $p < .05$). No systematic group differences were found.

The results of the behavioural measures are illustrated in Table 1. The reaction times became significantly ($F(1,12) = 12.7$, $p < .01$) longer within the ADHD children in Task 2 while this was not observed in the two other groups. The amount of errors decreased in Task 2 in all groups. As can be seen in Table 1, the ADHD group's reaction times in both tasks were longer than the control and RD groups', but a t-test indicated that the difference was not statistically significant. The ADHD children also made more errors than the control and RD children, but statistical significance was reached only in Task 1 in comparison to the children of the RD group ($t(25) = -2.12$, $p < .05$).

Table 1 Mean reaction times (RT) and the amount of errors (ERR) in two blocks per task in three groups.

	Control	ADHD	RD
RT block 1	465.1	480.6	481.5
RT block 2	467.5	503.5	496.0
RT task 1	**466.3**	**492.0**	**488.8**
RT block 1	478.0	509.4	484.0
RT block 2	501.7	547.6	501.4
RT task 2	**489.9**	**528.5**	**492.7**
ERR block 1	8.8	12.3	7.1
ERR block 2	10.2	14.5	7.1
ERR task 1	**19**	**26.8**	**14.2**
ERR block 1	5.8	7.6	4.3
ERR block 2	5.3	6.5	2.9
ERR task 2	**11.1**	**14.1**	**7.2**

Note: RTs in msec.

The Pearson correlations between RTs and CNV revealed no relationship, but some associations were displayed between RTs and P300 amplitudes in the RD

and ADHD groups. No systematic correlations were found in the control group. In the RD group negative correlations emerged in Task 2 at channels C3, Cz, C4 and at the both time windows of Pz ($r = -.55$, $p < .05$; $r = -.71$, $p < .01$; $r = -.77$, $p < .01$; $r = -.70$, $p < .01$; $r = -.70$, $p < .01$, respectively). In the ADHD group associations were observed in both tasks at channels C3 ($r = -.57$, $p < .05$ in Task 1; $r = -.59$, $p < .05$ in Task 2) and Cz ($r = -.63$, $p < .05$ in Task 1; $r = -.58$, $p < .05$ in Task 2).

In Task 1, the control group exhibited positive correlations between RTs and P300 latencies at C3, C4, and at the first time window of Pz ($r = .42$, $p < .05$; $r = .45$, $p < .05$; $r = .44$, $p < .05$, respectively), whereas the RD and ADHD groups showed no such effect. In Task 2, positive correlations were found in all groups. In the control group they were manifested at C3 ($r = .36$, $p < .05$) and at C4 ($r = .37$, $p < .05$), and in the RD group at Cz ($r = .64$, $p < .05$) and at the second time window of Pz ($r = .56$, $p < .05$). In the ADHD group the correlation was significant only at the second time window of Pz ($r = .61$, $p < .05$).

Discussion

The results showed that no CNV was found in any group in the first task whereas in the second task statistically significant CNV was attained in all groups. The effect was manifested frontally and centrally in the control and RD groups and frontally among the ADHD children. No time-on-task effect was visible in either task. In significant respects, these results are in line with Kivijärvi and Saunamäki's (1999) outcomes of the resembling study. In all groups the P300 amplitudes were larger in the first task. Unlike in the RD and control groups, in the ADHD group the amplitudes decreased gradually within both tasks showing time-on-task effect. With ragard to P300 latencies, they were slightly shorter in the first than in the second task in all groups. The reaction time analysis revealed that only the ADHD group's RTs were significantly longer in the second than in the first task and the amount of errors diminished in all groups in the second task. The ADHD children made more mistakes in both tasks than the children in the RD and control groups. Although statistical significance was reached only in the first task when comparing the ADHD to the RD group, the results indicated overall poorer performance of the ADHD children. Correlational measures revealed some relations between RTs and P300 amplitudes in the second task in the RD group and in both tasks in the ADHD group, but no associations were found in the control group. The negative correlations perceived in these two groups indicated that slower RTs were related to decreased amplitudes. Latencies and RTs in turn were correlated in the control group in both tasks while in the RD and ADHD groups this phenomenon was significant only in the second task. Slower response speed was seen in lengthened latencies.

The findings of the present study gave support for the interpretation of CNV as a reflection of activation and motor preparation in previous literature (see for review Rockstroh *et al.*, 1989). The CNV found in Task 2 probably refers to the subjects' more activated state due to the motivational factors. Frontally and centrally manifested CNV of the control and RD children could reflect more widespread activation in their neuronal networks in comparison to the ADHD group's only frontally exhibited CNV. When interpreting this in the light of executive functioning, it could be concluded that the assumed non-optimal activation state in the ADHD children also deteriorates their cognitive functions. Difficulties in state regulation thus require more effort to compensate for the lower level of activation.

The amount of effort invested in the tasks was presumed to be reflected in the P300 amplitudes. Motivation in the second task was assumed to increase effort and consequently, also the amplitudes, but the results were contradictory and puzzling. Considering Van der Meere's (1996) hypothesis, the boring condition in the first task may have lowered the level of activation and the non-optimal state has been compensated with effort. The smaller P300 amplitudes in the second task could therefore be a sign of decreased effort and the observed time-on-task effect in the ADHD group might refer to declining effort during the tasks. The negative correlations between reaction times and amplitudes provide support for the effort dysfunction in the case of the ADHD children, but the same effect observed in the RD group speaks against the interpretation. Another proposition for the results is one where the role of effort becomes less meaningful. It could be possible that when the motivational factors are introduced, they directly raise the level of activation. Contrary to expected, the ADHD children did not exhibit significantly smaller P300 amplitudes than the control and RD children, which also questions Van der Meere's effort dysfunction hypothesis. However, on closer inspection of the results of the behavioural measures, it could be assumed that the ADHD children were inferior in the effectiveness of effort to the control and RD children.

The slower response speed in the second task among the ADHD children could be explained as a state regulation problem reflected in slow motor responding, or, it could express the subjects' response strategy applied in achieving incentives. Fast and accurate performance might have been too demanding and by slowing down they have tried to avoid wrong responses. In the second task the strategy was possibly employed also by the two other groups, but only in the ADHD group the course of action was not efficient enough. The decreased amount of errors in all groups in the second task stands up for the assumption of the

subjects' operations model. The performance decrement during the tasks was not linear in any group and thus, statistically significant time-on-task effect was not found. That sets a confounding argument to Van der Meere's (1996) definition of sustained attention deficit in ADHD children.

Despite the absence of time-on-task effect, the slow but still most inaccurate responding in the ADHD group is comparable with Van der Meere's (1996) idea of task inefficiency. The ADHD children had difficulties in concentration, presumably due to their under-activated state and were thereby incapable of maximizing their performance. The task inefficiency could also be a reflection of a deficit on the motor side of cognitive information processing chain, where failing executive processes and slow motor preparation are intertwined. Van der Meere (1996) postulates that the origins for the task inefficiency and motor problems is again, a dysfunction of activation/effort. The smaller CNV as a reflection of motor preparation supports the view. The decreased amount of errors in the second task refers to the motivational factors having had an improving effect on performance level.

Sanders (1983) has suggested that external control as a motivator may modify the level of activation. Van der Meere and his colleagues (1995*a*) have later supported and extended the theory and shown that external control has an improving effect on ADHD children's performance whereas incentives do not. In the present study, it was noted that the performance improved in all groups in the second task in which the motivational factors were included. However, it remains questionable as to whether it was the external control or the pay-off or both together that motivated the children.

Interpretation of the meaning of the P300 latencies in the present study could be based on their positive correlations with reaction times, which refer to the latencies' role as a possible index of stimulus evaluation time (Coles *et al.*, 1990; Rockstroh *et al.*, 1989). Contrary to earlier studies, the latencies were not systematically longer in the ADHD group than in the other groups (Holcomb *et al.*, 1985; Sunohara *et al.*, 1997; Taylor *et al.*, 1993).

When further considering the present results and the differences found between the groups, the age and gender distributions between and within the groups have to be taken into account. It should be noted that boring task conditions set high expectations for young children's attentional abilities. Van der Meere and Stemerdink (1999) have studied the development of response inhibition and state regulation trajectory of normal children and concluded that the younger age group demonstrates poorer impulse control. Referring to Cohen (1973), the CNV amplitudes increase throughout ages from 8 to 16 years and hence, the age groups differ in CNV due to maturational processes. Results from Klorman (1975) also point out that age groups (10-, 14- and 19-year-olds) differ especially on behalf of the E-wave, supporting the view of the maturation of the CNV. In the present study, the developmental aspects were not examined and the groups were not matched by age, which might have biased the results from the CNV analysis along with the behavioural measures. The least errors in the tasks were made by the RD children, whose mean age was also the highest. It could be expected that the older the child, the better his ability to control impulsive behaviour and to sustain attention. Although the ADHD children's mean age was higher than the control children's, their EEG slow waves and behavioural measures refer to results gained from younger children. It is thus possible that the development of executive functioning skills is delayed in ADHD children. Gender distributions within and between the groups in the present study were not equal. Although the groups were not matched by gender, it is unlikely that it had a remarkable effect on the results.

Pure ADHD diagnoses are very rare and the issue of comorbidity in the ADHD group of the present study represents a common situation. August and Garfinkel (1990) have found out that as much as 39% of ADHD children also demonstrate reading disability. Thus, the sample used in the present experiment adds to the external validity of the study.

The broad usage of the concepts arousal and activation should be noticed in the generalization of the results. They are usually difficult to separate and define due to their varying and overlapping meanings in literature and that can have a great impact on the comparability of results from different studies. Arousal is defined in the present paper as a physiological state affected by external stimulation, whereas activation is considered as a psychological state meaning mainly inborn preparedness for action.

Although the two-stimulus paradigm used in the present study is not traditional in eliciting P300, it seems that the character of the P300-like wave was consistent with genuine P300. However, the experimental setting applied in the present research raises a question whether the results can reliably be compared to studies that have used the oddball paradigm.

The outcomes of the present study partly supported the set hypotheses and previous research results, but on the other hand, raised new questions to be answered. It appears that children suffering from ADHD do have some problems in state regulation, especially in activation. The role of effort and its association with P300, however, remains rather unclear. Although the part of effort in compensation of non-optimal activation state seems vague, motivation clearly improved the children's performance – including the ADHD children. This implies that children with attentional problems might benefit from supervising and reward tokens also in everyday life. The concept of

state regulation deficit seems useful in studying and explaining the ADHD children's maladjusted behaviour on behalf of activation, but further research is needed to clarify the role of effort as a compensatory factor.

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References

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, D.C.

August, G.J., & Garfinkel, B.O. (1990). Comorbidity of ADHD and Reading Disability Among Clinic-Referred Children. Journal of Abnormal Child Psychology, 18, 29 - 45.

Cohen, J. (1973). The CNV in children with special reference to learning disabilities. Electro-encephalography and Clinical Neurophysiology, Suppl. 33, 125 - 133.

Coles, M.G.H., Gratton, G., & Fabiani, M. (1990). Event-related brain potentials. In J.T. Cacioppo, & L.G. Tassinary (Eds.), Principals of psycho-physiology: Physical, social and inferential elements (pp. 413 - 455). New York: Cambridge University Press.

Donchin, E., & Coles, M.G.H. (1988). Is the P300 component a manifestation of contest updating? Behavioral and Brain Sciences, 11, 357 - 374.

Douglas, V.I. (1983). Attention and Cognitive Problems. In M. Rutter (Ed), Developmental neuropsychiatry (pp. 280 - 329). New York: Guilford Press.

Duncan-Johnson, C.C., & Donchin, E. (1977). On quantifying surprise: The variation of event related potentials with subjective probability. Psycho-physiology, 14, 456 - 467.

Goyette, C.H., Conners, C.K., & Ulrich, R.F. (1978). Normative data on revised Conners Parent and Teacher Rating Scales. Journal of Abnormal Child Psychology, 6, 221 - 236.

Grünewald-Zuberbier, E., Grünewald, G., Rasche, A., & Netz, J. (1978). Contingent negative variation and alpha attenuation responses in children with different abilities to concentrate. Electro-encephalography and Clinical Neurophysiology, 44, 37 - 47.

Hillyard, S.A. (1973). The CNV and human behavior. A review. Electroencephalography and Clinical Neurophysiology, Suppl. 33, 161 - 171.

Holcomb, P.J., Ackerman, P.T., & Dykman, R.A. (1985). Cognitive event related brain potentials in children with attention and reading deficits. Psychophysiology, 22, 656 - 667.

Häyrinen, T., Serenius-Sirve, S., & Korkman, M. (1999). Lukilasse – Lukemisen, kirjoittamisen ja laskemisen seulontatestistö peruskoulun ala-asteen luokille 1-6 [Lukilasse – A screening test for reading and writing skills and arithmetics for grades 1-6]. Helsinki: Psykologien kustannus.

Jasper, H. (1958). Report on the committee on methods of clinical examination in electroencephalography. Electroencephalography and Clinical Neuro-physiology, 10, 370 - 375.

Kemner, C., Verbaten, M.N., Koelega, H.S., Camfferman, G., & van Engeland, H. (1998). Are abnormal event-related potentials specific to children with ADHD? A comparison with two clinical groups. Perceptual and Motor Skills, 87, 1083 - 1090.

Kivijärvi, M.T., & Saunamäki, M.I. (1999). A psychophysiological approach to Sanders's and Mulder's cognitive-energetical information processing model: Contingent Negative Variation (CNV) in healthy 8 - 9-year-olds. Master's Thesis in psychology, University of Jyväskylä, Jyväskylä.

Klorman, R. (1975). Contingent negative variations and cardiac deceleration in a long preparatory interval: a developmental study. Psychophysiology, 12, 609 - 617.

Klorman, R. (1991). Cognitive Event-Related Potentials in Attention Deficit Disorder. Journal of Learning Disabilities, 24, 130 - 140.

Lindeman, J. (1998). Ala-asteen lukutesti [A comprehensive school reading test]. Turku: Oppimistutkimuksen keskus.

Loveless, N.E. (1977). Event-related brain potentials in selective response. Biological Psychology, 5, 135 - 149.

Loveless, N.E., & Sanford, A.J. (1974). Effects of age on the contingent negative variation and preparatory set in a reaction-time task. Journal of Gerontology, 29, 52 - 63.

Michael, R.L., Klorman, R., Salzman, L.F., Borgstedt, A.D., & Dainer, K.B. (1981). Normalizing Effects of Methylphenidate on Hyperactive Children's Vigilance Performance and Evoked Potentials. Psychophysiology, 18, 665 - 677.

Mulder, G. (1986). The concept and measurement of mental effort. In G.R.J. Hokkey, A.W.K. Gaillard, & M.G.H. Coles (Eds.), Energetics and Human Information Processing (pp. 175 - 198). Dordrecht: Martinus Nijhoff Publishers.

Picton, T.W., & Hillyard, S.A. (1988). Endogenous event related potentials. In T.W. Picton (Ed.), Handbook of electroencephalography and clinical neurophysiology: human event-related potentials (pp. 361 - 426). Amsterdam: Elsevier.

Pribram, K.H., & McGuinnes, D. (1975). Arousal, Activation, and Effort in the Control of Attention. The control of attention. Psychological review, 82, 116 - 149.

Rockstroh, B., Elbert, T., Canavan, A., Lutzenberger, W., & Birbaumer, N. (1989). Slow cortical potentials and behaviour (pp. 1 - 38). Munich: Urban & Schwarzenberger.

Rohrbaugh, J.W., & Gaillard, A.W.K. (1983). Sensory and Motor Aspects of the Contingent Negative Variation. In A.W.K. Gaillard and W. Ritter (Eds.), Tutorials in ERP Research: Endogenous Components (pp. 269 - 310). Amsterdam: North-Holland Publishing Company.

Sanders, A.F. (1983). Towards a model of stress and human performance. Acta psychologica, 53, 61-97.

Strandburg, R.J., Marsh, J.T., Brown, W.S., Asarnow, R.F., Higa, J., Harper, R., & Guthrie, D. (1996). Continuous-processing-related event-related potentials in children with attention deficit hyperactivity disorder. Biological Psychiatry, 40, 964 - 980.

Sunohara, G.A., Voros, J.G., Malone, M.A., & Taylor, M.J. (1997). Effects of methylphenidate in children with attention deficit hyperactivity disorder: A comparison of event related potentials between medication responders and non-responders. International Journal of Psychophysiology, 27, 9 - 14.

Taylor, M.J., Voros, J.G., Logan, W.J., & Malone, M.A. (1993). Changes in event related potentials with stimulant medication in children with ADHD. Biological Psychology, 36, 139 - 156.

Tecce, J.J. (1970). Attention and evoked potentials in man. In D.I. Mostofsky (Ed.), Attention: Contemporary theory and analysis (pp. 331 - 365). Appleton-Century-Crofts, New York.

Van der Meere, J.J. (1996). The role of attention. In S. Sandberg (Ed.), Hyperactivity Disorders of Childhood (pp. 111 - 148). Cambridge University Press.

Van der Meere, J., Shalev, R., Börger, N., & Gross-Tsur, V. (1995*a*). Sustained Attention, Activation and MPH in ADHD: A Research Note. Journal of Child Psychology and Psychiatry, 36, 697 - 703.

Van der Meere, J., & Stemerdink, N. (1999). The Development of State Regulation in Normal Children: An Indirect Comparison With Children With ADHD. Developmental Neuropsychology, 16, 213 - 225.

Van der Meere, J., Stemerdink, N., & Gunning, B. (1995*b*). Effects of presentation rate of stimuli on response inhibition in ADHD children with and without tics. Perceptual and Motor Skills, 81, 259 - 262.

Wechsler, D. (1984). Manual for the Wechsler Intelligence Scale for Children – Revised. Helsinki: Psychological Corporation.

Weerts, T.C., & Lang, P.J. (1973). The effects of eye fixation and stimulus response location on the contingent negative variation (CNV). Biological Psychology, 1, 1 - 19.

Welsh, M.C., Pennington, B.F., & Groisser, D.B. (1991). A Normative-Developmental Study of Executive Function: A Window on Prefrontal Function in Children. Developmental Neuropsychology, 7, 131 - 149.

Yordanova, J., Dumais-Huber, C., & Rothenberger, A. (1996). Coexistence of tics and hyperactivity in children: no additive effect at the psychophysiological level. International Journal of Psychophysiology, 21, 121 - 133.