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Postural instability in adult ADHD – A pilot study

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ABSTRACT

Background: Apart from inattention, hyperactivity and impulsivity, ADHD in childhood presents with an impairment of motor coordination and balance functions. Until now, literature is scarce about sensorimotor deficits in adult ADHD. This is a pilot study that identifies and quantifies the role of sensory, motor, and central adaptation mechanisms for adult ADHD patients' sensorimotor deficits in a systematic way, using postural control.

Methods: We analyzed spontaneous and externally perturbed stance in ten adult patients suffering from ADHD. Findings were compared to data from ten matched healthy subjects.

Results: Spontaneous sway amplitudes and velocities were larger in ADHD patients compared to healthy subjects. Furthermore, body excursions as a function of platform tilts were abnormally large in ADHD patients, specifically in the low frequency range. Based on simple feedback model simulations, we found that ADHD patients showed a larger time delay between platform tilts and body response, and a lower value of the integral part of the neural controller, which affects the long-term control of their posture. These postural abnormalities correlated well with the hyperactivity and impulsivity dimensions of the individual ADHD symptoms.

Conclusion: We conclude that adult ADHD patients' major postural deficit consists of an impairment of a stable, long-term sensorimotor behavior, which fits very well to the concept of impulsivity and hyperactivity.

1. Introduction

Attention-deficit/ hyperactivity disorder (ADHD) begins in childhood and may persist into adult life in a substantial subgroup of patients [1]. Adult ADHD patients seem to carry a higher risk of musculoskeletal trauma [2]. However, it is still unclear if the higher rate of trauma is accompanied by deficits in sensorimotor control. There are only very few reports about sensorimotor functions in adults suffering from ADHD. Recently, the group of Hove et al. [3] described larger body sway in adult ADHD and linked the postural control abnormalities to the cerebellar gray matter volume. Relatively smaller cerebellar volumes have been reported in adults suffering from ADHD before [4].

In childhood, it is well known that ADHD goes along with an impairment of coordination and balance functions [5–7]. Numerous studies found abnormal postural stability in ADHD children mostly expressed by larger body sway velocities or sway area (e.g.[7,8]). Moreover, active motor tasks seemed to be impaired in ADHD children [6], with an emphasis on timing of motor tasks, and greater variability in motor performance [9]. It has been shown that children with ADHD have lower sensory-motor abilities, motor coordination [5,10], as well as increased muscle activation latencies and amplitudes [11]. We hypothesize that these deficits may be related to balance abnormalities in adult ADHD.

In this pilot study, we examine postural stability of ADHD adults and control subjects during spontaneous sway and externally perturbed stance. A systematic, model-based approach delivers estimates for e.g. sensorimotor reaction time, use of sensory channels, error correction gain, and passive joint stiffness, which all may contribute to balance abnormalities with larger sway (see [12,13]).

2. Material and methods

Subjects were tested both using standardized ADHD diagnostic scales (clinical symptoms) and by recording their postural control on a motion platform (platform experiments).

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2.1. Subjects

The ADHD- group (N = 10) and the control group (N = 10) were matched by gender (60% male, 40% female) and age (+/−3y.). The ADHD-group was consecutively recruited from the outpatient department for ADHD of the University Medical Center of Freiburg, Germany. We compared ADHD patients without psychopharmacological treatment with a healthy control group. The control group was recruited from the social network of colleagues. Following international guidelines [14] the diagnosis of ADHD had been assessed by specialist psychiatrists (AP) with expertise in the diagnosis on the basis of a full structured clinical and psychosocial assessment concerning current and past symptoms, a developmental and psychiatric history, the DSM-IV-ADHD-Diagnostic-Checklist (ADHS-DC; [15]), the Conners-Adult-ADHD-Rating-Scale (Self-Report-Form, Long Version, CAARS- S:L; [16,17]) and the Conners-Adult-ADHD-Rating-Scale, Observer-Form, Long Version (CAARS-O:L; *x*). ADHD-symptoms in childhood were retrospectively assessed by using the German short form of the Wender-Utah-Rating-Scale (WURS-k; [18]), and school certificates.

Both the ADHD and the control group were seen by a psychiatrist and neurologist in an outpatient setting and were interviewed with respect to comorbidities, physical activity, and drug intake. Psychiatric comorbidities were assessed with the German versions of the Structured Clinical Interview for DSM-IV (SCID-I, [19]), depressive symptoms were additionally assessed by BDI-II (Beck Depression Inventory; Revision, BDI-II; [20]). The cut-off value for suffering from a depression was set at a value of 14 points according to [21]). In addition, the Body Sensations Questionnaire, (BSQ, [22]), the Agoraphobic Cognitions Questionnaire (ACQ, ([22]), and the Mobility-Inventory (MI, [23]) were assessed. Moreover, we used a visual analogue scale (VAS) to determine anxiety with regard to the experiment, i.e. fear of falling (0 = no anxiety, 10 = maximal anxiety, [24]). The VAS transfers feelings via emoticons into a chart.

In addition, both groups were tested with a urine drug screening. Care was taken that patients and control subjects did not use any medication with potential influence on the performance in the tests applied. The study was in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Freiburg University Medical Center, all subjects gave their informed written consent prior to study participation.

2.2. Platform experiments

Subjects stood upright on a custom built platform (Fig. 1A). Spontaneous sway was investigated on the stationary platform (2x with eyes open (eo) and 2x with eyes closed (ec), 1 min/ trial). Furthermore, we recorded the motor reaction to small platform rotations in anterior-posterior direction (6x eo and 6x ec; 1 min/ trial; frequencies: 0.05–2.2 Hz; peak-to-peak amplitudes of the tilt 0.5 or 1 deg). The axis of the rotation was located at the ankle joints. The computer-generated stimuli were based on a pseudorandom ternary sequence (PRTS). The two-dimensional spontaneous sway of the center of pressure (COP) was recorded by a force transducing platform (Kistler platform type 9286, Winterthur, Switzerland). In addition, the positions of the upper and the lower body in space were recorded with an optoelectronic system (Optotrack 3020, Waterloo, Canada) with active LED-markers attached to the hip and the shoulders. Software programmed in LabView (National Instruments, Austin, TX) recorded the output signals at a sampling rate of 100 Hz.

2.3. Data analysis

With software programmed in MATLAB (The MathsWorks Inc., Natick, MA) we evaluated the raw data. For comparison with clinical scales we used table calculation programs (Microsoft Excel). For assessing statistical significance we applied a statistical program (JMP, SAS Inc., Cary, NC, USA). The motor reactions following the stimulus were characterized by discrete Fourier-Transforms. The responses as functions of the stimulus were determined by transfer functions. Transfer function data consisted of GAIN, PHASE and coherence. GAIN is the angular excursion of the body as a function of the angular excursion of the platform. PHASE indicates the temporal relationship between platform and body angular excursion. The transfer functions were used as the experimental data basis for model simulations using a predefined model of upright stance.

2.4. Statistics

The statistical significance of the questionnaires between ADHD patients off-treatment and control subjects was tested by an analysis of variance (ANOVA) with group (ADHD, Control) as the between variable. In both conditions the influence of depressive symptoms was assessed using an ANOVA with the BDI as covariate. In order to test the differences of the self- and the observer-rating of the ADHD-symptoms we compared the CAARS-S:L and the CAARS-O:L of the ADHD group with a Pearson-product-moment-correlation.

The differences between the ADHD group and the control group in motor behavior were calculated by using a factorial ANOVA. We tested the effects of group (ADHD, Control) and its interactions with visual condition (eyes open, eyes closed) and segment (upper body, lower body) for all trials. In trials with platform rotation we also took into account the interactions between group and stimulus amplitude and stimulus frequency. Correlations between motor behavior and the clinical symptoms were analyzed by multivariate analyses. Bonferroni corrections were applied for multiple comparisons.

2.5. Model simulations

Using the transfer functions, which we derived from the relationship between stimulus and COM body angle, we extracted relevant parameters of postural control with a well-known model of balance control (e.g. [25]). This model includes a negative feedback loop that relates body excursion detected by sensors to a corrective torque via a controller with proportional, derivative and integral contributions (PDI-controller). Moreover, the model contains a lumped time delay, which represents the time between the stimulus and the motor reaction, and a sensory weighting mechanism, representing the coordinate frame of the body excursions (space coordinates vs. platform coordinates) as well as a biomechanics part, which depicts the elasticity and damping of the muscles and tendons. With the help of an optimization procedure (fmincon/ Matlab, Mathworks), we were able to fit model simulations to the experimental transfer functions under different stimulus frequencies, amplitudes and visual conditions.

3. Results

3.1. Subjects

The mean age was 35.5 years in both groups (ADHD: *SD* = 10.9 y, Range 18.0–52.0 y; controls: *SD* = 10.5 y, Range 19.0–49.0 y; $p = 0.8$). All ADHD subjects fulfilled the DSM IV criteria of the combined ADHD subtype. The ADHD group had an average of 0.7 psychiatric life-time-comorbidities (*SD* = 0.66; Min. 0.00; Max. 2.00), ie, six patients reported that they had suffered from a depression in the past. There was no current psychiatric comorbidity in the ADHD group. The control group did not report any comorbidity. One ADHD patient and one control subject had a positive urine screening for amphetamine after consuming weight-loss and sports nutrition supplements. The control urine screening prior to the platform experiments was negative. No subject or patient lost balance on the platform.

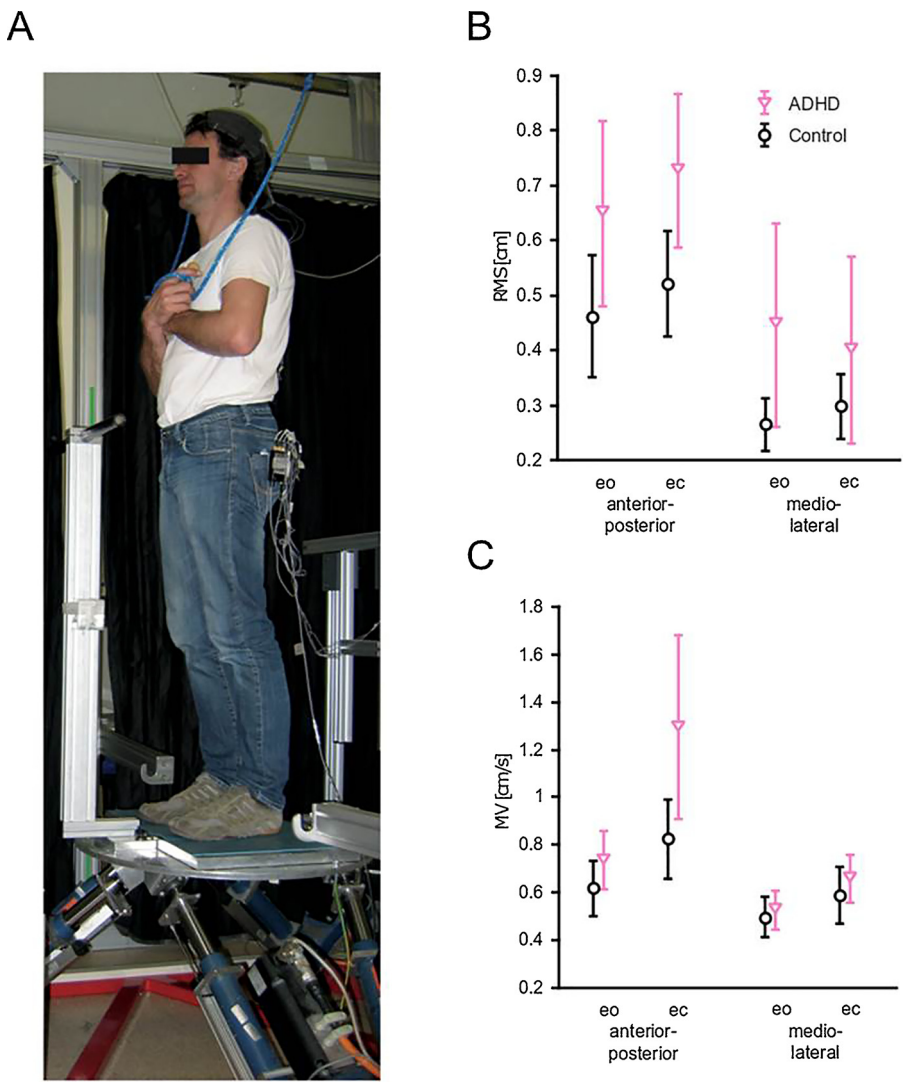

Fig. 1. A, experimental setup with subject standing on the hexapod platform. Subjects stood upright with a stance width of 7 cm (heel to heel). They held ropes which were fixed to the ceiling. In case of imminent falling, they could stabilize themselves by pulling the ropes. B, Spontaneous sway amplitude (root mean square, RMS), C, spontaneous sway velocity (mean velocity, MV), in ADHD patients off-treatmend and control subjects. eo, eyes open, ec, eyes closed.

3.2. Clinical scales

The results of the psychometric data are depicted in Table 1 (Supplementary material). All subscores of the CAARS-S:L (self) correlated significantly with the matched subscores of the CAARS-O:L (observer; each subscore, $p < 0.005$).

The covariance-analysis with the BDI-score as a covariate showed a significant difference of ADHD-symptoms on almost all subscales between patients and control subjects (except subscore D: problems with self-concept). General anxiety (questionnaires ACQ, BSQ and MI) and anxiety with regard to the experiment (VAS) did not differ significantly between the groups (ACQ: ADHD group: 1.5, control group 1.2; BSQ: ADHD group: 1.9, control group 1.4; MI-accompanied: ADHD group: 1.5, control group: 1.0; MI-alone: ADHD group: 1.7, control group: 1.1; each $p > 0.05$; VAS: all patients and control subjects 0 or 1). One patient reached the cut-off value of 14 points in the Beck Depression Inventory (mild depression with 14 points and more). This value was composed of high values in the sub scores concentrativeness, restlessness and disappointments in the past that can be interpreted in the frame of the known ADHD symptoms. The BDI values of the other patients and the control group did not reach the cut-off value for depression.

3.3. Spontaneous sway

The ADHD group showed a larger root mean square (RMS, sway amplitude; ADHD off-treatment: 0.60 cm; controls: 0.39 cm; $F = 8.9$; $p < 0.001$; Fig. 1B) and a larger mean velocity of sway than controls (MV, ADHD off-treatment: 0.80; controls: 0.63; $F = 5.7$; $p < 0.005$; Fig. 1C). The mean frequency (MF) was similar in ADHD patients and in control subjects ($F = 0.3$; $p = 0.48$, not shown).

3.4. Externally disturbed stance

Fig. 2 shows the average time series of the shoulder segment (upper body, A), and hip segment (lower body, B) as a function of anterior-posterior platform tilts (C) for ADHD patients, and for control subjects. We Fourier-transformed the time series into frequency functions, which relate the angles of the upper, and lower body to the platform angle in terms of GAIN and PHASE (see Fig. 3).

The ADHD group showed a significantly larger GAIN of the upper (Fig. 3A) and lower body (Fig. 3B) than the control group (ADHD: 1.84; controls: 1.71; $F = 6.6$; $p = 0.005$). Both in ADHD patients and control subjects, GAIN varied significantly as a function of stimulus frequency ($p < 0.0001$), visual condition (eyes open/eyes closed $p < 0.0001$, not shown), and stimulus amplitude (0.5° vs. 1°; $p < 0.0001$, not

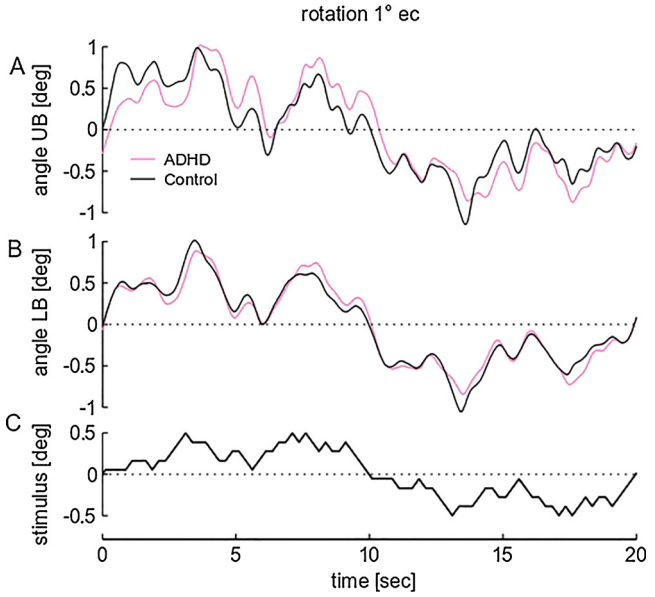

Fig. 2. Average time series of the upper body (UB; A) and lower body (LB; B) angles as a function of anterior-posterior platform tilts (stimulus; C) in the eyes-closed condition for ADHD patients, and for control subjects. Note that some differences between groups are visible. deg, degree, sec, seconds, ec, eyes closed.

shown). The ADHD group showed larger GAIN values specifically in the low frequency range (Fig. 3E, gain values were normalized with respect to the gain of control subjects). None of the interactions between factors and group designation were significant ($p > 0.2$).

PHASE relates to the latency of the response to the stimulus. PHASE in the ADHD group was slightly but significantly different from the control group ($F = 4.4$; $p < 0.05$, Fig. 3C, D). Whereas the upper body showed a slight phase lead with respect to the control subjects (Fig. 3C), the lower body displayed a slight phase lag (Fig. 3D). This is represented by the fact that segment and group significantly interact ($p = 0.0005$) and points to a slightly different inter-segmental coordination. Similar to GAIN, PHASE was significantly determined by stimulus frequency ($p < 0.0001$), and visual condition ($p = 0.005$, not shown). Amplitude had no significant effect ($p > 0.05$).

Coherence (reproducibility of the reaction to the stimulus) was significantly smaller in the ADHD group compared to control subjects (ADHD: 0.46, controls: 0.50; $F = 17.0$; $p < 0.0001$) representing a higher variability due to the increased spontaneous sway.

3.5. Model-based analysis

The model-based parameter extraction revealed that ADHD patients showed a significantly lower value of the integral part of the PDI-control mechanism that sums up the position errors over time and affects the low frequency postural response (Ki, in ADHD off-treatment: 73.5; control subjects: 81.3; $F = 4.7$, $p = 0.01$; Fig. 4). Moreover, we observed in ADHD patients a significantly larger time delay (ADHD off-treatment: 164 ms; control subjects 148 ms; $F = 4.2$, $p = 0.03$; Fig. 4). Other parameters of postural control, e.g. the proportional and the derivative gain of the neural controller were similar in ADHD patients and control subjects ($p > 0.2$; not shown). The sensory weighting factor (Wp), representing the percentage of proprioceptive, vestibular, and visual sensory information to the overall sensory feedback, was similar in ADHD patients as well as in control subjects ($p > 0.25$; Fig. 4).

3.6. Correlation between ADHD-symptoms and posturographic results

In a first step we calculated multivariate correlations across all parameters listed above, including clinical scores, parameters of

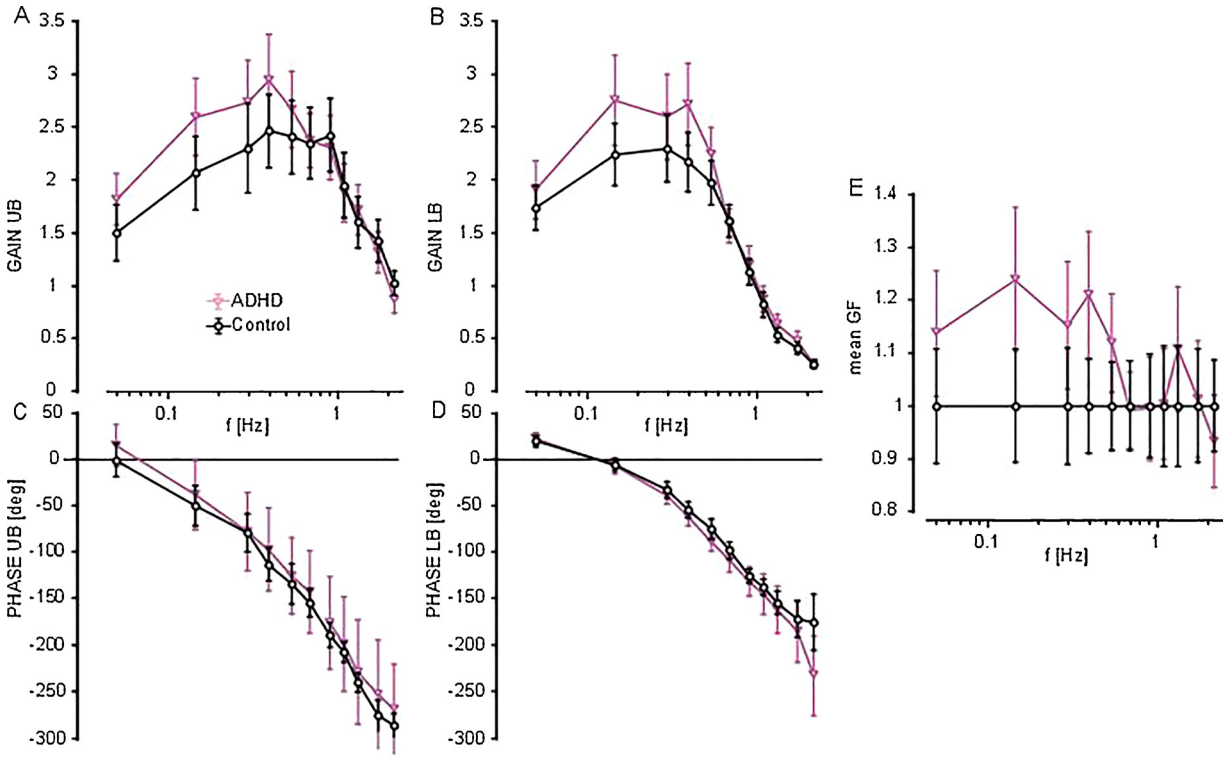

Fig. 3. Parameters of perturbed stance. GAIN (A, B), PHASE (C, D), and normalized GAIN (gainfactor, GF) with respect to the control subjects (E) for upper body (UB; A, C) and lower body (LB; B, D) excursions. Values are given as a function of frequency (Hertz, Hz). Note that the main GAIN differences are confined to the lower frequency range.

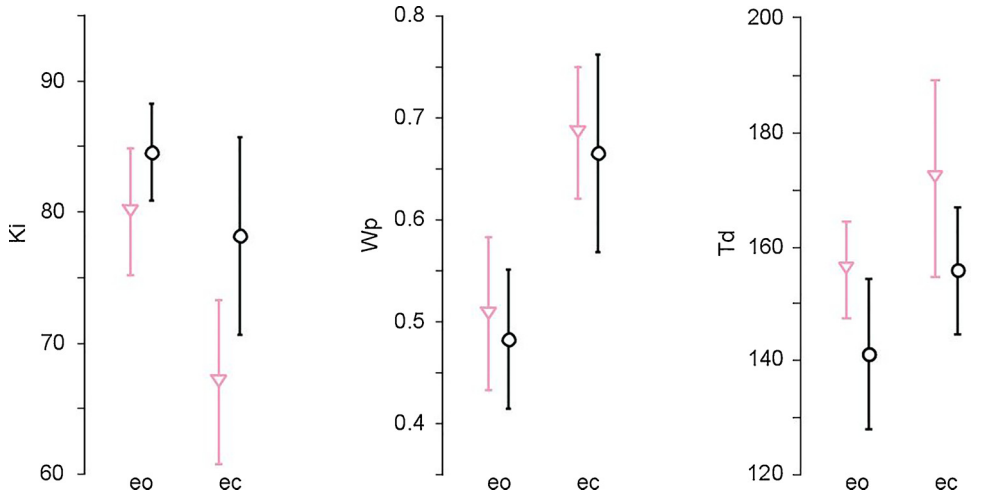

Fig. 4. Model-based parameter extraction. Ki (integral part of the neural controller, low frequency, i.e. long-term deficit of postural reactions), Wp (sensory weighting factor) and Td (time delay) as a function of visual condition. eo, eyes open, ec, eyes closed.

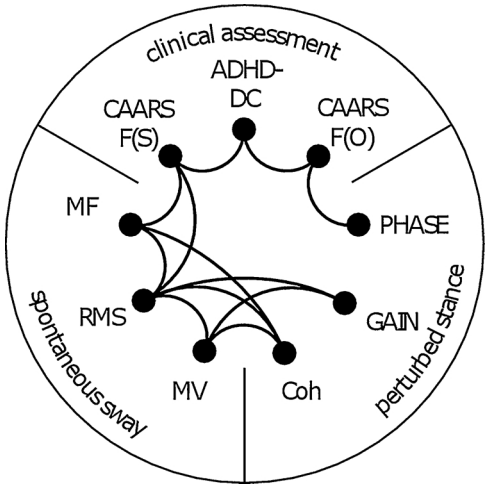

Fig. 5. Correlation between representative clinical scales of ADHD-symptoms and the results of the postural control experiments. ADHD-DC, modified DSM-IV-ADHD-Diagnostic-Checklist; subscores of CAARS-S:L (=S) and CAARS-O:L (=O), Conners-Adult-ADHD-Rating-Scale. MF, mean frequency, RMS, root mean square, MV, mean velocitiy, Coh, coherence.

spontaneous sway and of perturbed test, applying a Bonferroni correction. For didactic reasons, we focus in Fig. 5 on the three clinical assessment scores that correlated best with the postural control parameters. All the other subcores of CAARS-S:L, CAARS-O:L and ADHS-DC were significantly correlated to these three clinical assessment scores. The F-subscore of the CAARS-S:L (symptoms of hyperactivity and impulsivity of DSM IV, self) correlated significantly with RMS sway amplitude ($r = -0.51$, $p = 0.02$). Moreover, the F-subscore of the CAARS-O:L (observer) significantly correlated with PHASE ($r = 0.60$, $p = 0.005$). Interestingly, not all postural control parameters correlated with each other. For example, PHASE did not correlate with any other postural control parameters ($p > 0.08$). RMS sway amplitude ($r = 0.47$, $p = 0.03$) and MV of sway ($r = 0.62$, $p = 0.004$) significantly correlated with GAIN.

4. Discussion

The aim of this pilot study was to investigate underlying mechanisms of motor deficits in a representative sample of adult ADHD patients by using a very specific motor function test. During unperturbed stance we observed significantly higher spontaneous sway amplitudes (RMS) and velocities (MV) of ADHD patients compared to controls. This is in line with previous research ((8): larger sway velocity and sway area, [7]: larger sway area). In a recent study, larger body sway in adult ADHD was described and postural control abnormalities were linked to the cerebellar gray matter volume [3]. Due to the fact that measures of (enhanced) spontaneous sway just provide a very limited view on underlying postural mechanisms (see e.g.(25)), we also measured subject's reaction to external perturbations, i.e. small platform tilts. ADHD subjects had significantly larger response GAINs than controls across all body conditions, specifically in the low frequency range. Patients' PHASE curves pointed to a different inter-segmental strategy. In addition, coherence was significantly lower in ADHD subjects compared to controls. This represents a higher variability of the reactions and corresponds to the higher RMS and MV of spontaneous sway. Larger variabilities in motor tasks are a common finding in ADHD [9,26]

The model-based parameter extraction revealed that ADHD patients showed a significantly larger time delay between stimulus and response than control subjects. A higher time delay challenges postural stability in terms of a resonance tendency. Larger reaction time variability in ADHD children has been observed before [9,27]. The larger variability in time reproduction was interpreted as a dysfunction of the fronto-striato-cerebellar timing-system in ADHD [28]. In addition, we observed a significant lower value of the integral part (Ki) of the neural controller (PDI) in ADHD patients, which is related to their low frequency, i.e. long-term, postural deficits. As we assume that neural controller mechanisms are anatomically located within a loop involving the sensory and motor cortex, abnormalities in ADHD patients in this loop have been reported before [29]. To our surprise, the sensory weighting mechanism in ADHD seemed to be unaffected, which is inconsistent to other studies [30]. This may be related to the fact that these former studies used the sensory organization test (SOT), which relates any abnormality to either sensory or motor disturbances, while our approach includes, in addition, central control mechanisms.

Since impulsivity and hyperactivity measured with the self- and investigator-rated CAARS were significantly associated with several parameters of undisturbed and disturbed stance in ADHD, we interpret the postural abnormalities in ADHD in the context of hyperactivity/impulsivity rather than in the context of other dimensions of the disorder like inattention. Likewise, a positive link between motor impairments and deficits in attention and concentration has been recently doubted in children [6].

One limitation of this pilot study was the small sample size which might influence the statistical power. In addition, we recruited patients and healthy control subjects in two different ways, possibly inducing a

selection bias. Whereas patients were recruited consecutively from the outpatient clinic, control subjects were recruited from colleagues by matching age and gender of the patients. Relevant psychiatric comorbidities have been assessed with validated interviews. In addition, a neurological examination was performed. However, other comorbidities have only been assessed by interviews. Moreover, substance abuse or use has been evaluated during an interview and using an urine drug screening. No further analysis of e.g. blood samples for signs of substance abuse/ use or comorbidities was undertaken which might bias the data.

Altogether, our results demonstrate that postural deficits are present in ADHD adults and should therefore be specifically targeted by therapeutic interventions. More generally, we showed here that a perturbation-guided approach can precisely monitor and allocate sensorimotor deficits such as those in adult ADHD with or without therapeutic interventions. Further studies with larger sample sizes are needed to corroborate our findings.

Conflict of interest statement

A. Philipsen has been on the advisory boards of and has received lecture fees and travel assistance from Eli Lilly, Medice Arzneimittel Pütter GmbH, Novartis, Lundbeck, Servier and Shire. She is also the author of books and articles on psychotherapy published by Elsevier, Hogrefe, Schattauer, Kohlhammer, Karger, and Oxford Press. The other authors report no conflicts of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.gaitpost.2018.10.016.

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