

Bonke, E. M., Bonfert, M. V., Hillmann, S. M., Seitz-Holland, J., Gaubert, M., Wiegand, T. L.T., De Luca, A., Cho, K. I. K., Sandmo, S. B., Yhang, E., Tripodis, Y., Seer, C., Kaufmann, D., Kaufmann, E., Muehlmann, M., Gooijers, J., Lin, A. P., Leemans, A., Swinnen, S. P., Bahr, R., Shenton, M., Pasternak, O., Tacke, U., Heinen, F., Koerte, I. K. (2023). Neurological soft signs in adolescents are associated with brain structure. *Cerebral Cortex*, 33(9), 5547-5556.
<http://dx.doi.org/10.1093/cercor/bhac441>

Dette er siste tekst-versjon av artikkelen, og den kan inneholde små forskjeller fra forlagets pdf-versjon. Forlagets pdf-versjon finner du her:
<http://dx.doi.org/10.1093/cercor/bhac441>

This is the final text version of the article, and it may contain minor differences from the journal's pdf version. The original publication is available here:
<http://dx.doi.org/10.1093/cercor/bhac441>

Neurological soft signs are associated with reduced medial-lateral postural control in adolescent athletes

Elena M. Bonke^{1,2,3}, Amanda Clauwaert⁴, Stefan M. Hillmann⁵, Uta Tacke⁶, Caroline Seer^{4,7}, Eukyung Yhang⁸, Yorghos Tripodis^{8,9}, Stian B. Sandmo^{10,11}, Tim L.T. Wiegand^{1,3}, David Kaufmann^{1,12}, Elisabeth Kaufmann^{1,13}, Sutton B. Richmond^{4,14}, Malo Gaubert^{1,15,16}, Johanna Seitz-Holland³, Alexander Leemans¹⁸, Stephan P. Swinnen⁴, Roald Bahr¹⁰, Ofer Pasternak^{3,17}, Florian Heinen⁵, Inga K. Koerte^{1,2,3,19}, Michaela V. Bonfert^{5 +*}, Jolien Gooijers^{4,7 +}

+ last authors contributed equally

¹ cBRAIN, Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, University Hospital, LMU, Munich, Germany;

² Graduate School of Systemic Neurosciences, LMU, Munich, Germany;

³ Psychiatry Neuroimaging Laboratory, Department of Psychiatry, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA;

⁴ Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Leuven, Belgium;

⁵ Division of Pediatric Neurology and Developmental Neuroscience, Department of Pediatrics at Dr. von Hauner Children's Hospital, University Hospital, LMU, Munich, Germany;

⁶ University Children's Hospital (UKBB), Basel, Switzerland;

⁷ KU Leuven, Leuven Brain Institute, Department of Movement Sciences, Movement Control and Neuroplasticity Research Group;

⁸ Department of Biostatistics, Boston University School of Public Health, Boston, MA, USA;

⁹ Alzheimer's Disease and CTE Centers, Boston University School of Medicine, Boston, MA, USA;

¹⁰ Oslo Sports Trauma Research Center, Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway;

¹¹ Division of Mental Health and Addiction, Oslo University Hospital, Oslo, Norway;

¹² Department of Diagnostic and Interventional Radiology and Neuroradiology, University Hospital Augsburg, Augsburg, Germany;

¹³ Department of Neurology, LMU, Munich, Germany;

¹⁴ Department of Applied Physiology and Kinesiology, University of Florida, Gainesville, FL, USA;

¹⁵ Radiology Department, CHU Rennes, Rennes, France;

¹⁶ Inria, CNRS, Inserm, IRISA UMR 6074, Empenn ERL, University of Rennes, Rennes, France;

¹⁷ Department of Radiology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA;

¹⁸ Image Sciences Institute, University Medical Center Utrecht, Utrecht, the Netherlands;

¹⁹ Department of Psychiatry, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA.

* Corresponding author:

Michaela V. Bonfert, MD Michaela.Bonfert@med.lmu.de

Division of Pediatric Neurology and Developmental Neuroscience, Department of Pediatrics at Dr. von Hauner Children's Hospital, University Hospital, LMU, Lindwurmstraße 4, 80337 Munich, Germany.

Phone: +49 152 54923717

Authors' contact information:

Elena M. Bonke, MSc

Elena.Bonke@med.uni-muenchen.de

cBRAIN, Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, University Hospital, LMU, Nußbaumstraße 5, 80336 Munich, Germany.

Amanda Clauwaert, PhD

Amanda.Clauwaert@kuleuven.be

Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Tervuursevest 101, 3001 Leuven, Belgium.

Stefan M. Hillmann

stefan.hillmann@med.uni-muenchen.de

Division of Pediatric Neurology and Developmental Neuroscience, Department of Pediatrics at Dr. von Hauner Children's Hospital, University Hospital, LMU, Lindwurmstraße 4, 80337 Munich, Germany.

Uta Tacke, MD

Uta.Tacke@ukbb.ch

University Children's Hospital (UKBB), Spitalstraße 33, 4056 Basel, Switzerland.

Caroline Seer, PhD

caroline.seer@kuleuven.be

Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Tervuursevest 101, 3001 Leuven, Belgium.

Eukyung Yhang

eyhang@bu.edu

Department of Biostatistics, Boston University School of Public Health, Crosstown Building 801 Massachusetts Avenue 3rd Floor, Boston, MA 02118, US.

Yorghos Tripodis, PhD

yorghos@bu.edu

Department of Biostatistics, Boston University School of Public Health, Crosstown Building
801 Massachusetts Avenue 3rd Floor, Boston, MA 02118, USA.

Stian B. Sandmo, MD, PhD

stianbs@nih.no

Oslo Sports Trauma Research Center, Norwegian School of Sport Sciences, PB 4014 Ullevål
Stadion 0806 Oslo, Norway.

Tim L.T. Wiegand

Tim.Wiegand@med.uni-muenchen.de

cBRAIN, Department of Child and Adolescent Psychiatry, Psychosomatics and
Psychotherapy, University Hospital, LMU, Waltherstraße 23, 80337 Munich, Germany.

David Kaufmann, MD

David.Kaufmann@uk-augsburg.de

Department of Diagnostic and Interventional Radiology and Neuroradiology, University
Hospital Augsburg, Stenglinstraße 2, 86156 Augsburg, Germany.

Elisabeth Kaufmann, MD

Elisabeth.Kaufmann@med.uni-

muenchen.de

Department of Neurology, Ludwig Maximilians University, Marchioninistraße 15, 81377
Munich, Germany.

Sutton B. Richmond, PhD

sutton.b.richmond@gmail.com

Department of Applied Physiology and Kinesiology, University of Florida, 1864 Stadium
Rd., Gainesville, FL, USA.

Malo Gaubert, MSc

malogaubert@gmail.com

Radiology Department, CHU Rennes, 2 Rue Henri le Guilloux, 35000 Rennes, France.

Johanna Seitz-Holland, MD jseitz@bwh.harvard.edu

Psychiatry Neuroimaging Laboratory, Department of Psychiatry, Brigham and Women's Hospital, Harvard Medical School, 1249 Boylston St., Boston, MA 02215, USA.

Alexander Leemans, PhD A.Leemans@umcutrecht.nl

Image Sciences Institute, University Medical Centre Utrecht, Heidelberglaan 100, 3584 CX Utrecht, the Netherlands.

Stephan P. Swinnen, PhD stephan.swinnen@kuleuven.be

Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Tervuursevest 101, 3001 Leuven, Belgium.

Roald Bahr, MD, PhD roaldb@nih.no

Oslo Sports Trauma Research Center, Norwegian School of Sport Sciences, PB 4014 Ullevål Stadion 0806 Oslo, Norway.

Ofer Pasternak, PhD ofer@bwh.harvard.edu

Psychiatry Neuroimaging Laboratory, Department of Psychiatry, Brigham and Women's Hospital, Harvard Medical School, 1249 Boylston St., Boston, MA 02215, US.

Florian Heinen, MD Florian.Heinen@med.uni-muenchen.de

Division of Pediatric Neurology and Developmental Neuroscience, Department of Pediatrics at Dr. von Hauner Children's Hospital, University Hospital, LMU, Lindwurmstraße 4, 80337 Munich, Germany.

Inga K. Koerte, MD Inga.Koerte@med.uni-muenchen.de

cBRAIN, Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, University Hospital, LMU, Nußbaumstraße 5, 80336 Munich, Germany.

Jolien Gooijers, PhD

Jolien.Gooijers@kuleuven.be

Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Tervuursevest 101, 3001 Leuven, Belgium.

Conflicts of interest:

YT receives funding from ERA-NET Neuron [01EW1707] and from the National Institutes of Health [NIH R01 HL141774-02] and [NIH R01 HD090191]. EK receives royalties for book chapters and received speaker honoraria and travel support from Medtronic, UCB, Livanova, and Eisai and has participated in clinical trials for Medtronic, UCB and Precisis, all unrelated to the submitted work. JSH receives funding for a Fellowship Award from Harvard Medical School Livingston and for a Young Investigator Grant sponsored by Mary and John Osterhaus and the Brain & Behavior Research Foundation. IKK receives grant funding from the National Institutes of Health (USA), the European Research Council, and the German Ministry of Education and Research. IKK receives funding for a collaborative project and serves as a paid scientific advisor for Abbott. She receives royalties for book chapters. Her spouse is an employee at Siemens AG.

Funding:

Funding was provided through the framework of ERA-NET Neuron [01EW1707]. The individual national funding agencies are the German Ministry for Education and Research (Germany), the Research Foundation Flanders [G0H2217N] and Flemish Government (Sport Vlaanderen, D3392), Slovak Academy of Sciences and Ministry of Education of Slovak Republic [APVV-17-0668], the Dutch Research Council (NOW), the Norwegian Research Council (NFR), and the Ministry of Health, Israel [#3-13898]. This work was also supported by the European Research Council [ERC Starting Grant 804326].

Author's contribution:

CS, YT, SBS, DK, AL, SPS, RB, OP, FH, IKK and JG were involved in the study planning. EMB, AC, SMH, UT, CS, SBS, TLTW, DK, EK, FH, IKK, MVB and JG were involved in the data acquisition. EMB, AC, SMH, UT, EY, YT, SBR, FH, MVB, and JG were involved in

data analysis and/or statistical analysis. EMB, AC, MVB, and JG drafted the manuscript and created figures and tables. EMB, AC, SMH, UT, CS, EY, YT, SBS, TLTW, DK, EK, SBR, MG, JSH, AL, SPS, RB, OP, FH, IKK, MVB, and JG critically edited the manuscript, and approved the final version of the manuscript.

Acknowledgements:

Consortium members:

Sylvain Bouix, Martha E. Shenton, Alexander P. Lin, Fanny Dégeilh, Alexandra Gersing, Felicitas Heinen, Leonard Jung, Janna Kochsiek, Marc Muehlmann, Paul S. Raffelhueschen, Paula C.M. Schorlemer, Bettina Schwarz-Moertl, Alexandra A. C. Silva, Lisa Umminger, Alberto de Luca, Serafien D’Hooghe, Doron Elad, Thor Einar Andersen, Erling Hisdal, Audun Holm Torgersen, Martin Cente, Igor Jurisica, Katarina Matyasova, Sara Porubska and Jozef Hanes, Nir Sochen.

Abstract

Introduction: Neurological soft signs (NSS) are minor deviations from the norm in motor performance that are commonly assessed using neurological examinations. NSS may be of clinical relevance for evaluating the developmental status of adolescents. Here we investigate whether quantitative force plate measures may add relevant information to observer-based neurological examinations.

Methods: Male adolescent athletes (n=141) aged 13-16 years from three European sites underwent a neurological examination including 28 tests grouped into six functional clusters. The performance of tests and functional clusters was rated as optimal/non-optimal resulting in NSS+/NSS- groups and a continuous total NSS score. Participants performed a postural control task on a Balance Tracking System measured as path length, root mean square and sway area. ANCOVAs were applied to test for group differences in postural control between the NSS+ and NSS- group, and between optimal/non-optimal performance on a cluster- and test-level. Moreover, we tested for correlations between the total NSS score and postural control variables.

Results: There was no significant overall difference between the NSS+ and NSS- group in postural control. However, non-optimal performing participants in the diadochokinesis test swayed significantly more in the medial-lateral direction than optimal performing participants. Moreover, a lower total NSS score was associated with reduced postural control in the medial-lateral direction.

Conclusion: Our findings demonstrate that NSS are related to postural control in adolescent athletes. Thus, force plate measures may add a quantitative, objective measurement of postural control to observer-based qualitative assessments, and thus, may complement clinical testing.

Keywords: Neurological Soft Signs; Postural Control; Adolescent Athletes; Diadochokinesis; Maturation; Minor Neurological Dysfunction

1 Introduction

Neurological soft signs (NSS) are minor deviations from the norm in motor performance and sensory-motor integration [1]. In contrast, major (also called *hard*) signs are rated as pathological and include findings such as hyperreflexia and spasticity [2,3]. Even though not considered pathological, and also present in healthy adults [4], investigating NSS in adolescents is clinically relevant because it was found to be associated with an increased vulnerability for mental disorders [5,6]. More specifically, there is initial evidence for NSS being a non-specific marker for developing developmental deficiencies such as attention-deficit hyperactivity disorder and schizophrenia spectrum disorder [7–9].

NSS are typically assessed using a clinical neurological examination [10]. Commonly used rating systems are qualitative and include the Neurological Examination Scale (NES) [11], the Cambridge Neurological Inventory (CNI) [12], the Heidelberg NSS Scale (HS) [13], and the age-dependent assessment of Minor Neurological Dysfunction (MND) [14,15] (for review of NSS rating systems see [16]). With some minor differences, most rating systems comprise tests of coordination, fine motor skills, and postural control [17]. Importantly, these assessments are based on the examiners' observation and therefore are qualitative rather than quantitative by nature. Particularly when it comes to subtle alterations as it may be the case for physically trained adolescent athletes, the addition of instrumented investigations such as force plate measures assessing postural control, which are considered to be the “gold standard” for assessing postural stability [18], may provide valuable insights by quantitatively detecting variability in performance that might not be detected visually.

Postural control is considered a milestone of physiological development and allows individuals to engage in various static and dynamic activities [19]. It can be described as the maintenance of the center of pressure inside the base of support and is defined by Bartlett and Birmingham as the ability to maintain internal stability (e.g., to stand upright) and to

anticipate and avoid instability in response to an external perturbation [20]. Generally, motor performance and postural control development take place according to two stages. First, young children, approximately until the age of five years, follow an *open-loop strategy*, characterized by the explorative activity of the nervous system exploring all motor possibilities. This strategy is also referred to as the ballistic strategy and is characterized by a greater displacement of the center of pressure (COP) and thus greater instability [21]. Around the age of eight years, children transfer to using a *closed-loop strategy* (or sensory strategy) where they use various movement possibilities in each specific situation, resulting in better postural control [21,22]. As children age further, their nervous system adapts, and multisensory integration is improved. More specifically, older children integrate information from different modalities (e.g., visual and somatosensory system) more successfully to control posture, making them better equipped to function in a changing environment [22,23]. However, any disturbances of this maturation process will likely result in less optimal or non-optimal postural control. Of note, postural control has been shown to mature later along the ML compared to the AP axis [21–23]. Currently, there is no clear consensus in the literature regarding at what age adolescents reach their peak performance in postural control. Some studies suggest that adolescents have not yet reached an adult-like performance in postural control at the age of 14 years [24] while others found that processes underlying the maintenance of an optimal postural stability are mature much earlier [25]. In adolescent athletes, this developmental process was reported to be ongoing until the age of 19 years as shown by normative Balance Tracking System (BTrackS) data of more than 10 000 adolescent athletes between 8 and 21 years [26].

To the best of our knowledge, no study has yet used a combined approach of observer-based neurological examination and quantitative assessment of postural control to comprehensively assess motor maturation between the ages of 13 and 16 years. Thus, to date it remains unknown whether the findings of both assessments are interrelated. Therefore, this

study aims to provide information on the additional use of force plate measures in a clinical setting. It is expected that a lower qualitative NSS score derived from a neurological examination is reflected in reduced postural control performance. The use of force plates as an objective measure will allow for a detailed quantification of higher-order motor functioning in addition to clinical observer-based assessments. This will add relevant information when investigating motor functioning in a clinical setting.

2 Materials and Methods

2.1 Study design and participants

Participant data were extracted from the multi-site study REPIMPACT (Repetitive Subconcussive Head Impacts – Brain Alterations and Clinical Consequences; 2017-2020). The study enrolled male adolescent athletes aged 13-16 years between July 2017 and April 2020 from three study sites (Oslo, Norway; Leuven, Belgium; Munich, Germany). Included athletes participated in various sports that were primarily soccer, but also swimming, track and field, cross-country skiing, cycling, tennis, biathlon, and rowing. Participants and their legal guardians at all three data acquisition sites provided written informed consent in accordance with the local ethics board approvals and the Declaration of Helsinki.

In short, inclusion criteria for participating in the study were: (1) 13–16 years of age, (2) male, (3) participation in sports at a competitive level with training at least three times per week in the respective sport, and (4) fluent language skills of the respective country of citizenship (i.e., German, Dutch, or Norwegian). For more details on the REPIMPACT study design, see [27].

Participants were excluded from the analysis in case of (1) history of serious medical condition (history of encephalitis: $n = 3$), (2) incidental finding on a magnetic resonance imaging scan (periventricular gliosis: $n = 1$, subependymal heterotopia: $n = 1$), (3) premature

birth (i.e., < 37 weeks of gestation) (n = 3), (4) diagnosed attention deficit disorder (n = 1), (5) neurological hard signs as evident by neurological examination (n = 0), or (6) no performed neurological examination as part of the REPIMPACT project (n = 17). This resulted in an included sample of 141 participants (Table 1). The neurological examination and the postural control assessment were performed at the same visit.

Table 1. Cohort Characteristics

	NSS+ (n = 26)	NSS- (n = 115)	Statistical test
Study site (n)	B (2), G (5), N (19)	B (32), G (38), N (45)	$X^2 = 8.540$, df = 2, $p = .014 *$
Age [years] <i>Mean (SD)</i>	14.70 (0.67)	15.14 (0.74)	$t (139) = -2.798$, $p = .006 *$
Height [cm] <i>Mean (SD)</i>	170.32 (10.01)	173.62 (7.50)	$t (135) = -1.861$, $p = .065$
Weight [kg] <i>Mean (SD)</i>	58.19 (10.35)	60.39 (8.55)	$t (134) = -1.130$, $p = .260$
Total NSS score <i>Median (SE)</i>	21.50 (0.55)	25.30 (0.19)	$t (139) = -7.912$, $p < .001 *$

Note. * Indicates significant difference between groups at $p < .05$.

Abbreviations. B = Belgium, G = Germany, N = Norway, NSS = neurological soft signs, SD = standard deviation, SE = standard error, X^2 = Chi Square

2.2 Neurological examination assessing NSS

A standardized pediatric neurological examination based on the *William DeMyer's Neurological Examination* was performed [28]. This examination included the evaluation of NSS in line with the distinct age-related rating framework published within the MND concept [14]. We decided to follow the MND concept because it has proven useful when investigating developmental cohorts [29–31]. Of note, compared to other assessments such as the NES, CNI, HS, the MND concept considers the developmental status of a child and assesses performance with respect to age [14,32]. Using an age-dependent concept allows to detect even subtle changes which is particularly helpful in a cohort of physically trained adolescent athletes that are expected to perform well in tasks assessing motor performance.

The MND examination consists of 64 tests grouped as eight functional clusters. More details are published elsewhere [15]. In our examination, 28 out of these 64 tests were performed, assessing six functional clusters: (1) *Fine motor skills* (e.g., *finger-opposition test*: tapping the tip of the thumb with each finger of the same hand in a specific sequence), (2) *Coordination & balance* (e.g., *diadochokinesis test while standing*: quick antagonistic pro- and supination movements of the forearm), (3) *Posture & tone* (e.g., posture while sitting, standing, or walking), (4) *Involuntary movements* (e.g., sitting/standing choreatic movements or tremor) (5) *Associated movements* (e.g., in diadochokinesis or finger-opposition test), and (6) *Sensory function* (e.g., graphesthesia, kinesthesia).

The performance of every test was rated as either optimal or non-optimal based on the neurological optimality score (NOS), indicating how many items are performed in an optimal way [15,33]. Optimal performance is based on predefined criteria, meaning that failing a test indicates not meeting the specific predefined criteria of the respective test.

A non-optimal performed cluster is defined as a cluster that includes a number of non-optimal performed tests that exceeds a specific threshold. If one or more clusters were rated as

non-optimal, a participant was assigned to the group with NSS (NSS+ group), and otherwise in the group without NSS (NSS- group). In addition to the overall group categorization, participants were categorized as optimal/non-optimal performing on the cluster-level and on the test-level, respectively.

In a second step, an additional continuous NSS score was calculated because this qualitative clinical categorization approach does not allow to differentiate between participants with higher and lower NSS scores within a specific group. This score is in alignment with the NOS and is referred to as the *total NSS score*. For instance, a score of 26 indicates that the participant performed optimal in 26/28 tests. Consequently, a higher NSS score indicates better neurological functioning.

In Germany, the assessment was performed by experienced pediatric neurologists. Examiners from Norway and Belgium were trained by experienced pediatric neurologists from Germany before performing the examination independently. In addition, examinations from Norway and Belgium were videotaped and recorded and were then re-assessed independently by three pediatric neurologists from Germany.

2.3 Assessment of postural control using BTrackS

Postural control was assessed at all study sites using a validated portable force platform, BTrackS (Balance Tracking Systems Inc., San Diego, CA, USA) [18,34,35], consisting of a 0.4m x 0.6m force platform. A 25 Hz sampling rate was used to capture the ground reaction forces of the postural control assessments, which was registered as resulting COP and was filtered with a second-order, low-pass Butterworth filter (cut-off frequency of 4 Hz) prior to export. Postural control was tested both on a rigid and a compliant (foam) surface. The compliant surface consisted of a 6 cm thick Airex Balance Pad (Alusuisse Airex AG, Switzerland) placed on the force plate.

Participants performed all postural assessments without shoes and with socks. Following plate calibration, participants were instructed to stand on the force plate with their head in an upright position and as still as possible with eyes closed, hands on the hips, and feet apart at shoulder width. The start and end of each trial were indicated with an auditory tone. Each trial lasted 20 seconds and was repeated to obtain a total of three trials per condition (i.e., rigid and foam) per participant. All participants performed the rigid trials first, followed by the foam trials. A trial was repeated if a participant did not perform the task correctly (e.g., when a participant kept his eyes open).

The mean/total displacement of the COP, in addition to anterior-posterior (AP) and medial-lateral (ML) directional components, were used to quantify postural stability among participants. Utilizing COP-based equations developed by Thomas Prieto and colleagues in a custom MATLAB [36] script [37], three traditional postural metrics were derived for each axis (Mean/AP/ML): (1) path length (PL), which informs about the COP trajectory magnitude, (2) root mean square (RMS) amplitude deviations which represents the standard deviation of the displacement of the COP, and (3) sway area, or the COP displacement between the beginning and end of trial across axes [38]. The three dependent variables were chosen to describe the spatial-temporal and variability aspects of postural performance, respectively. For all measurements, higher values are indicative of poorer static postural control.

2.4 Statistical analyses

Statistical analyses were performed using the software R (version 4.0.1) [39]. In the first analysis, a categorical approach using a cut-off criterion based on clinical diagnostic practices was used. Participants were categorized into two groups: a group with and without NSS (NSS+ and NSS-) and groups with optimal/non-optimal performance in the *fine motor skills* and *coordination & balance* cluster, as well as in the *finger-opposition test* and the

diadochokinesis test. We selected these two clusters and two tests (1) because based on clinical experience, the performance of fine motor skills and coordination is highly relevant and commonly assessed, and (2) they were the most often non-optimal performed clusters/tests in our cohort. The sample size of participants that performed non-optimal in the other clusters/tests was too small ($n < 20$) to categorize reasonable groups. It was evaluated whether these groups differ in performance on the postural control tasks. However, while clinically relevant, this approach does not allow for a differentiation between participants with higher and lower NSS scores within a specific group. Therefore, for the second analysis, the continuous total NSS score was correlated with the postural control performance.

2.4.1 Differences in demographical variables between the NSS+ and NSS- group

Chi-Square tests were used to evaluate whether the distribution of participants from the three study sites was similar in the NSS+ and NSS- group. Moreover, to test whether the NSS+ and NSS- group include similar populations, demographical variables such as age, height, and weight and the total NSS score were assessed using independent t-tests.

2.4.2 Differences in postural control between groups

Data from the three acquired trials for each condition (rigid and foam) and each postural control variable (PL, RMS, sway area) were averaged. Outliers were visually inspected and removed if they were most likely caused by a technical error, resulting in an exclusion of one trial of $n = 1$ participant in the rigid condition. In this case, the remaining two trials were averaged.

Between-group differences (NSS+ and NSS-) in postural control performance in the rigid and foam condition for the variables of interest path length, root mean square, and sway area were calculated using ANCOVAs with age at the time of the neurological examination as a covariate. Additional analysis including study site as a covariate (not shown here), did not change the results. Multiple comparisons were corrected for 14 variables (PL (Mean/AP/ML),

RMS (Mean/AP/ML) and sway area in both rigid and foam condition) using the Bonferroni method. The level of significance was set to $\alpha = .05$.

2.4.3 Correlation between postural control and total NSS score

Spearman correlation coefficients were used to test whether the total NSS score was associated with postural control performance in the rigid and the foam condition. Spearman correlation was used because the total NSS score was not normally distributed, with more participants scoring on the higher end, meaning better overall performance.

3 Results

3.1 Demographic differences between the NSS+ and NSS- group

Table 1 summarizes the demographic information for both groups (NSS+ and NSS-).

Based on the neurological examination, 26 (18.4%) participants were categorized as NSS+ and 115 (81.6%) participants as NSS-. Of the 141 participants, 115 (81.6%) participants performed optimal in all six clusters (NSS- group), 24 (17.0%) performed non-optimal in one cluster, 1 (0.7%) performed non-optimal in two clusters, and 1 (0.7%) in four clusters. The cluster that most often was performed non-optimal was *fine motor skills*, performed non-optimal by 24 participants (17.0%), followed by a non-optimal performance of 3 participants in *associated movements* (2.1%), of 2 participants in *coordination & balance* (1.4%) and of 1 participant (0.7%) in *posture & tone*. Of note, none of the participants performed non-optimal in the *sensory function* cluster.

There was a significant between-group difference for study site ($p = .014$) with a greater proportion of participants in the NSS+ group coming from Norway compared to Belgium and Germany. Furthermore, there was a significant between-group difference for age ($p = .006$), with the NSS+ group being significantly younger than the NSS- group. In addition, there was a significant between-group difference for the total NSS score ($p < .001$). Height and weight

did not differ significantly between the NSS+ and NSS- group (all $p > .05$). Moreover, a-priori analyses revealed no statistically significant between-group differences regarding the type of sports (soccer versus non-contact) and concussion history (no previous concussion, probable concussion, physician-diagnosed concussion) in the prevalence of NSS (all $p > .05$) and in the postural control performance (for each dependent variable and condition; all $p > .05$).

3.2 No difference in postural control between the NSS+ and NSS- group

There were no significant differences in postural control for any of the variables of interest, neither in the rigid nor the foam condition between NSS+ (n = 26) and NSS- group (n = 115) (all $p > .05$; see Table A 1).

3.3 No difference in postural control between groups performing optimal/non-optimal on the cluster-level

There were no significant differences in postural control for any of the variables of interest, neither in the rigid nor the foam condition between the groups of participants with optimal and non-optimal performance in the *fine motor skills* cluster or the *coordination & balance* cluster (all $p > .05$).

3.4 Significant difference in postural control between groups performing optimal/non-optimal on the test-level

There were no significant differences in postural control for any of the variables of interest, neither in the rigid nor the foam condition, between the groups of participants with optimal (n = 105) and non-optimal (n = 36) performance in the *finger-opposition test* (all $p > .05$). The group that performed non-optimal in the *diadochokinesis test* (n = 33), however, had significantly higher sway area ($p = .003$), mean path length ($p = .022$), path length in the ML

direction ($p = .002$), mean root mean square ($p = .044$), and root mean square in the ML direction ($p = .002$) in the rigid condition compared to the group that performed optimal in this test ($n = 108$). Path length and root mean square postural performance in the AP direction, as well as all variables in the foam condition, were not significantly different between groups after correcting for multiple comparisons ($p > .05$). Differences in postural control between the groups performing optimal/non-optimal in the *diadochokinesis test* are illustrated in Figure 1 and listed in Table 2.

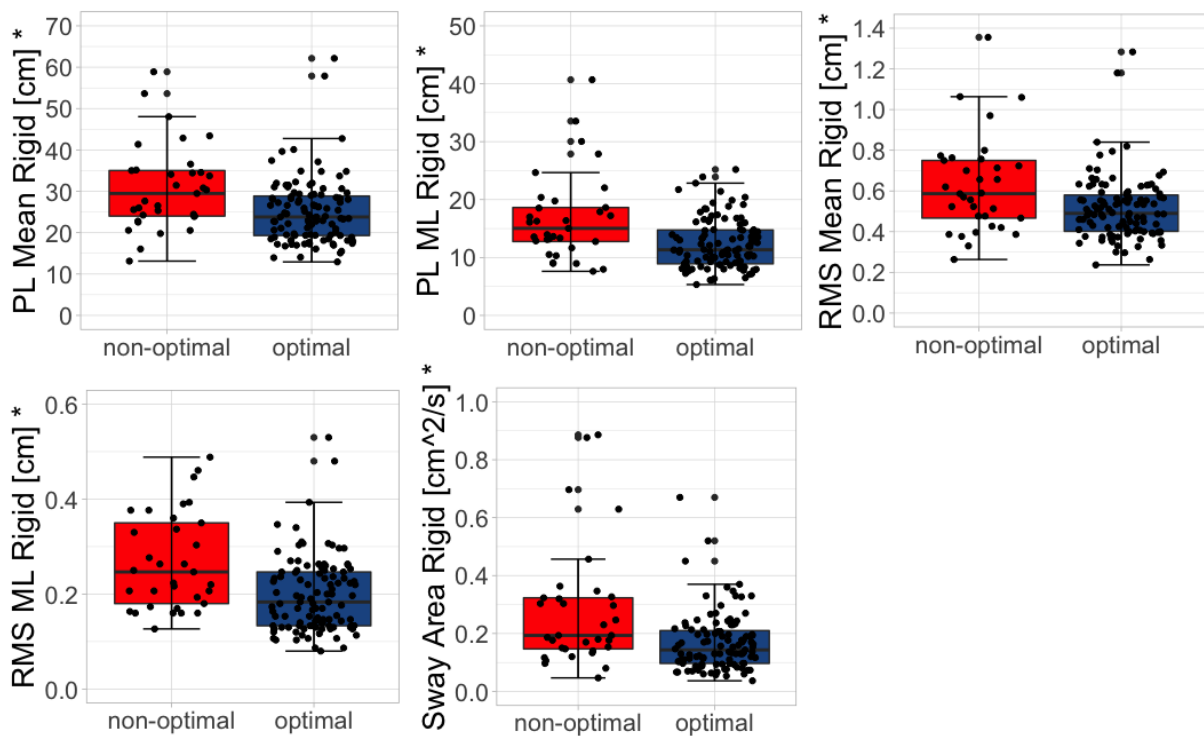


Figure 1. Significant difference between groups performing optimal/non-optimal in the *diadochokinesis test* in the rigid condition. Higher values indicate worse performance in postural control. Variable y-axis scaling was used for better visualization.

Note. * All p-values were corrected for 14 variables at $p < .05$.

Abbreviations. ML = medial-lateral, PL = path length, RMS = root mean square

Table 2. Difference in Postural Control between Groups Performing Optimal/Non-Optimal in the Diadochokinesis Test

Postural control	Non-optimal diadochokinesis <i>Mean (SD)</i>	Optimal diadochokinesis <i>Mean (SD)</i>	<i>F</i> (1, 136) = <i>p</i>
<hr/>			
Rigid			
PL	30.82 (10.34)	25.11 (7.93)	<i>F</i> (1, 136) = <i>p</i> = .022 10.429 *
PL ML	16.77 (7.55)	12.34 (4.28)	<i>F</i> (1, 136) = <i>p</i> = .002 15.651 *
PL AP	21.79 (6.97)	18.99 (6.67)	<i>F</i> (1, 136) = <i>p</i> = .526 4.410
RMS	0.63 (0.24)	0.51 (0.16)	<i>F</i> (1, 136) = <i>p</i> = .044 9.060 *
RMS ML	0.27 (0.10)	0.20 (0.08)	<i>F</i> (1, 136) = <i>p</i> = .002 15.185 *
RMS AP	0.56 (0.23)	0.47 (0.15)	<i>F</i> (1, 136) = <i>p</i> = .187 6.279
Sway area	0.28 (0.21)	0.17 (0.10)	<i>F</i> (1, 136) = <i>p</i> = .003 14.756 *
<hr/>			
Foam			
PL	66.41 (20.72)	60.96 (13.95)	<i>F</i> (1, 136) = <i>p</i> > .999 2.093
PL ML	30.73 (8.90)	27.71 (7.89)	<i>F</i> (1, 136) = <i>p</i> > .999 2.515

PL AP	52.46 (17.34)	48.38 (11.17)	$F (1, 136) = p > .999$ 1.754
RMS	1.31 (0.36)	1.25 (0.26)	$F (1, 136) = p > .999$ 0.652
RMS ML	0.63 (0.17)	0.61 (0.18)	$F (1, 136) = p > .999$ 0.170
RMS AP	1.14 (0.34)	1.07 (0.24)	$F (1, 136) = p > .999$ 0.781
Sway area	1.17 (0.62)	1.00 (0.43)	$F (1, 136) = p > .999$ 2.313

Note. * All p-values were corrected for 14 variables at $p < .05$.

Abbreviations. AP = anterior-posterior, ML = medial-lateral, PL = path length, RMS = root mean square, SD = standard deviation

3.5 Significant correlation between postural control and total NSS score

Figure 2 and Table 3 show the correlations between postural control and the total NSS score. The correlations were statistically significant for path length ($p = .016$) and root mean square in the ML direction in the rigid condition ($p = .011$), and for path length in the ML direction in the foam condition ($p = .025$). No significant correlations between postural control in the other variables were detected (all $p > .05$).

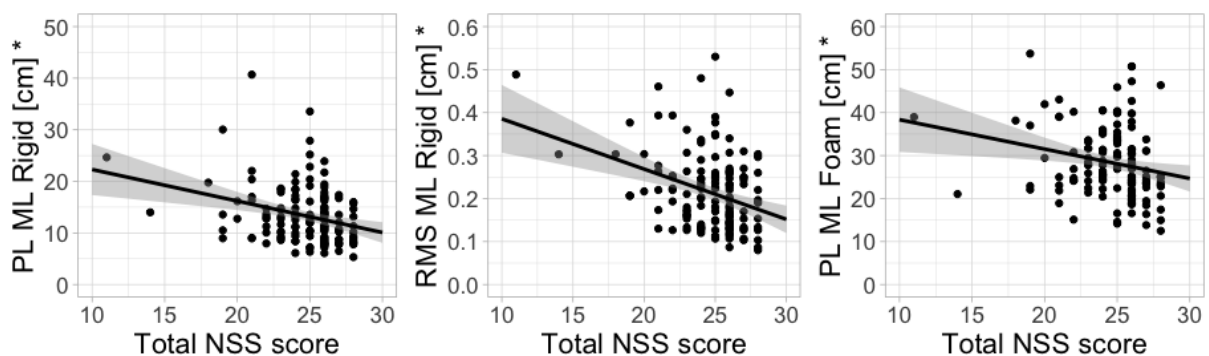


Figure 2. Scatterplots showing negative correlations between postural control and the total NSS score. Lower NSS scores and higher postural control variables indicate lower performance.

Note. * All p-values were corrected for 14 variables at $p < .05$. Variable y-axis scaling was used for better visualization.

Abbreviations. AP = anterior-posterior, ML = medial-lateral, NSS = neurological soft signs, PL = path length, RMS = root mean square

Table 3. Correlation between Postural Control and Total NSS Score

NSS	Postural control	<i>Rho</i>	<i>p</i>
Total NSS score	PL Rigid	$r_s(139) = -.195$	$p = .290$
Total NSS score	PL ML Rigid	$r_s(139) = -.271$	$p = .016 *$
Total NSS score	PL AP Rigid	$r_s(139) = -.132$	$p > .999$
Total NSS score	RMS Rigid	$r_s(139) = -.156$	$p = .906$
Total NSS score	RMS ML Rigid	$r_s(139) = -.279$	$p = .011 *$
Total NSS score	RMS AP Rigid	$r_s(139) = -.097$	$p > .999$
Total NSS score	Sway area Rigid	$r_s(139) = -.222$	$p = .114$
Total NSS score	PL Foam	$r_s(139) = -.141$	$p > .999$
Total NSS score	PL ML Foam	$r_s(139) = -.260$	$p = .025 *$
Total NSS score	PL AP Foam	$r_s(139) = -.189$	$p = .357$
Total NSS score	RMS Foam	$r_s(139) = -.064$	$p > .999$
Total NSS score	RMS ML Foam	$r_s(139) = -.093$	$p > .999$
Total NSS score	RMS AP Foam	$r_s(139) = -.055$	$p > .999$
Total NSS score	Sway area Foam	$r_s(139) = -.182$	$p = .424$

Note. * All p-values were corrected for 14 variables at $p < .05$.

Abbreviations. AP = anterior-posterior, ML = medial-lateral, NSS = neurological soft signs,

PL = path length, RMS = root mean square

4 Discussion

This study aimed to investigate the association between an objective quantitative assessment of postural control using force plates and an observer-based neurological examination in a cohort of adolescent athletes. Such associations have not been assessed before, but are of critical importance for clinicians as it provides a more comprehensive assessment of motor maturation between the ages of 13 and 16 years. It was assumed that lower NSS scores would be reflected in reduced postural control performance.

Our results revealed that participants performing non-optimal in the *diadochokinesis test* showed significantly reduced postural control. More specifically, this effect was only observed in the rigid condition of the postural control measurement, and primarily in the ML direction. No significant between-group differences in postural control were found in the foam condition.

Moreover, we found that the continuous total NSS score was, indeed, correlated with postural control performance. The results revealed a significant correlation between total NSS score and postural control in the ML direction, suggesting that those with lower performance in the neurological examination also tended to produce more ML COP movement in the force plate assessment.

4.1 A multi-method approach to NSS evaluation

In the clinical context, postural control is commonly assessed using observer-based tools such as classical neurological tests (i.e., Romberg stance, one-legged stance, tight rope walk). Particularly when it comes to subtle alterations, instrumented tools such as force plates may add additional and objective information to what even an experienced clinician can detect by eye. Using force plates in addition to a neurological examination may, thus, support clinicians by providing a more comprehensive and personalized assessment of motor maturation. Indeed, in the current study, the instrumented assessment of postural control revealed that

reduced performance in postural control along the ML axis was associated with a lower total NSS score. Nonetheless, more research is needed to determine the common underlying neurodevelopmental substrates of the quantitative and qualitative findings and whether other instruments or test conditions can quantitatively reflect other tasks derived from the NSS assessment.

4.2 Association of NSS with postural control in medial-lateral direction

Our findings of lower postural control in the ML direction being significantly correlated with a lower total NSS score, and being significantly reduced in the group of participants performing non-optimal in the *diadochokinesis test* is in alignment with previous literature on motor development [21,22,40]. It has been shown that AP and ML postural adjustments follow different maturation processes. More specifically, there is evidence that postural control along the ML axis matures later than along the AP axis, possibly being caused by (1) earlier maturation of antigravity muscles responsible for AP control, (2) daily life experience that is expected to be greater in the AP direction, and (3) earlier development of the ankle strategy involved in AP postural control than the hip strategy involved in ML postural control [21,22,40]. We did not observe an association between postural control in the AP axis and NSS scores. While the existence of such an association cannot be ruled out with certainty based on these data, the pattern that we observed is compatible with the possibility that the maturation along the AP axis is already fully developed in our sample.

Moreover, among the neurological tests, the performance of antagonistic forearm movements in quick succession, as examined in the *diadochokinesis test*, has been shown to mature later than other tests assessing upper-limb motor proficiency [41]. Of note, the two tasks are based on similar underlying functional mechanisms. Both tasks involve higher-order motor networks that control the alternation and timing of muscle activity in agonist and antagonist muscles as a result of cortical excitatory and inhibitory neuronal activity [41].

Thus, our unique finding of reduced postural control along the ML axis in a group of participants that performed non-optimal in the *diadochokinesis test* aligns with already existing knowledge. The performance in postural control and diadochokinesis movements may thus serve as indicators of motor maturation.

Adolescent competitive athletes receive extensive physical training during a crucial period of physiological motor development. Accordingly, one may expect that adolescent athletes will be more skilled in motor tasks, including postural control, than the general adolescent population. Indeed, it has been demonstrated that increased sensorimotor experience in sensitive developmental periods can improve motor proficiency [42,43]. Interestingly, Bieć & Kuczyński have shown that in sports disciplines such as soccer which requires bodyweight transfers along the ML axis, 13-year old soccer players had better postural control in the ML direction in comparison with a control group [43]. Moreover, a recently published study found a positive effect of special sensorimotor training in kicking sports on medio-lateral performance in postural control (REFERENCE). Thus, differences in postural control performance depending on the participants' athletic background may be assumed. Unfortunately, we were unable to compare sport disciplines (e.g., soccer, swimming, etc.) in our sample due to insufficient sizes of subsamples. It may be interesting for future studies to evaluate the association between postural control along the ML axis and NSS in athletes from different disciplines and compare with a non-athlete population.

4.3 Group differences in the rigid but not foam condition

We detected group differences only in the rigid, but not the foam condition. Moreover, fewer variables in the foam condition compared to the rigid condition were significantly correlated with the total NSS score. While the rigid condition is a well-validated method that is often used in research [35], the foam condition may have resulted in a greater variability, or in other words random noise, of postural control performance e.g., due to differences in the ability to

deal with less stability. Indeed, our data showed a greater variance of postural sway between participants and within the trials of individual participants in the foam condition compared to the rigid condition. Thus, the effects detected in the standardized, rigid condition may have been absent in the foam condition due to individual differences in response to the alteration of tactile inputs (decrease plantar pressure sensations) and stimulation of proprioceptive inputs (instability). Moreover, the foam condition was always performed after the rigid condition and thus, may have been affected more by potential training effects. However, we do not anticipate strong learning effects as only 3 trials of the rigid condition were performed.

4.4 Strengths and limitations

It is important to note that in our sample, the NSS+ group was significantly younger than the NSS- group. Preliminary evidence shows that by reaching puberty, the prevalence of NSS decreases due to several factors, including hormonal changes and brain maturation processes [32,44]. Therefore, we explicitly controlled our group analyses for a potential age effect. Correcting or not correcting for age resulted in similar findings. This additional analysis confirmed that in our sample, the age difference between groups did not explain the differences in postural control. Moreover, we replicated our analysis in a sub-sample only including participants from Germany and Norway and excluding participants from Belgium that turned out to be older (~5 months) than the rest of the cohort. Again, our results did not change compared to our main analyses, further confirming that our findings were not merely the result of an age difference between groups.

The fact that we performed an adapted version of the neurological examination to maximize the cost-benefit ratio, leaves the possibility that we may have missed potentially relevant tests in one of the clusters, in particular, the *coordination & balance* cluster. Such tests, in addition to the performance in the *diadochokinesis test*, may have correlated with the objective postural control assessment. Moreover, given that the sample size of some non-

optimal performed clusters and tests was too small for further analyses, we cannot interpret the association of these motor functions with postural control performance.

In addition to investigating an understudied population, another strength of the current study is that we were able to recruit athletes from various disciplines and three different study sites, resulting in a comparably large sample of this specific population. While this multi-site approach required differences in the NSS assessment setup (i.e., video vs. in-person evaluation), this decision allowed for relying on a comprehensive multi-rater approach to score the video examinations.

5 Conclusion

The current study demonstrated that the presence of NSS, and in particular non-optimal performance in the *diadochokinesis test*, is related to objective postural control performance in male adolescent athletes. Our findings reveal new knowledge by showing that a quantitative measurement of postural control may complement observer-based qualitative neurological examinations of NSS by capturing more detailed quantitative information. Future research should include larger samples of the general (healthy) population, with the inclusion of female participants, and participants of additional age ranges. This will ensure to capture a more complete picture of motor development, its variance, and its value to identify at-risk individuals with altered developmental trajectories.

References

- [1] P. Dazzan, R.M. Murray, Neurological soft signs in first-episode psychosis: A systematic review, *Br. J. Psychiatry.* 181 (2002) 50–57.
<https://doi.org/10.1192/bjp.181.43.s50>.
- [2] I. Martins, M. Lauterbach, P. Slade, H. Luís, T. Derouen, M. Martin, A. Caldas, J. Leitão, G. Rosenbaum, B. Townes, A longitudinal study of neurological soft signs from late childhood into early adulthood, *Dev. Med. Child Neurol.* 50 (2008) 602–607.
<https://doi.org/10.1111/j.1469-8749.2008.03043.x>.
- [3] R.D. Sanders, M.S. Keshavan, The neurologic examination in adult psychiatry: From soft signs to hard science, *J. Neuropsychiatry Clin. Neurosci.* 10 (1998) 395–404.
<https://doi.org/10.1176/jnp.10.4.395>.
- [4] M.A. Kennard, Value of equivocal signs in neurologic diagnosis, *Neurology.* 10 (1960). <https://doi.org/10.1212/WNL.10.8.753>.
- [5] M. Mayoral, I. Bombín, J. Castro-Fornieles, A. González-Pinto, S. Otero, M. Parellada, D. Moreno, I. Baeza, M. Graell, M. Rapado, C. Arango, Longitudinal study of neurological soft signs in first-episode early-onset psychosis, *J. Child Psychol. Psychiatry.* 53 (2012) 323–331. <https://doi.org/10.1111/J.1469-7610.2011.02475.X>.
- [6] J.E. Obiols, F. Serrano, B. Caparrós, S. Subirá, N. Barrantes, Neurological soft signs in adolescents with poor performance on the continuous performance test: Markers of liability for schizophrenia spectrum disorders?, *Psychiatry Res.* 86 (1999) 217–228.
[https://doi.org/10.1016/S0165-1781\(99\)00039-6](https://doi.org/10.1016/S0165-1781(99)00039-6).
- [7] R.C.K. Chan, T. Xu, R.W. Heinrichs, Y. Yu, Y. Wang, Neurological Soft Signs in Schizophrenia: A Meta-analysis, *Schizophr. Bull.* 36 (2010) 1089–1104.

<https://doi.org/10.1093/SCHBUL/SBP011>.

- [8] P. Whitty, M. Clarke, O. McTigue, S. Browne, M. Gervin, M. Kamali, A. Lane, A. Kinsella, J. Waddington, C. Larkin, E. O’Callaghan, Diagnostic specificity and predictors of neurological soft signs in schizophrenia, bipolar disorder and other psychoses over the first 4 years of illness, *Schizophr. Res.* 86 (2006) 110–117. <https://doi.org/10.1016/J.SCHRES.2006.04.012>.
- [9] V.C. Patankar, J.P. Sangle, H.R. Shah, M. Dave, R.M. Kamath, Neurological soft signs in children with attention deficit hyperactivity disorder, *Indian J. Psychiatry.* 54 (2012) 159–165. <https://doi.org/10.4103/0019-5545.99540>.
- [10] A. Ojagbemi, Neurological Soft Signs, *Encycl. Personal. Individ. Differ.* (2017) 1–5. https://doi.org/10.1007/978-3-319-28099-8_782-1.
- [11] R.W. Buchanan, D.W. Heinrichs, The neurological evaluation scale (NES): A structured instrument for the assessment of neurological signs in schizophrenia, *Psychiatry Res.* 27 (1989) 335–350. [https://doi.org/10.1016/0165-1781\(89\)90148-0](https://doi.org/10.1016/0165-1781(89)90148-0).
- [12] E.Y.H. Chen, J. Shapleske, R. Luqued, P.J. Mckennac, J.R. Hodgese, S.P. Callowayc, N.F.S. Hymasc, T.R. Dening, G.E. Berrios, The Cambridge Neurological Inventory: A clinical instrument for assessment of soft neurological signs in psychiatric patients, *Psychiatry Res.* 56 (1995) 183–204. [https://doi.org/10.1016/0165-1781\(95\)02535-2](https://doi.org/10.1016/0165-1781(95)02535-2).
- [13] J. Schröder, R. Niethammer, F.J. Geider, C. Reitz, M. Binkert, M. Jauss, H. Sauer, Neurological soft signs in schizophrenia, *Schizophr. Res.* 6 (1991) 25–30. [https://doi.org/10.1016/0920-9964\(91\)90017-L](https://doi.org/10.1016/0920-9964(91)90017-L).
- [14] M. Hadders-Algra, K.R. Heineman, A.F. Bos, K.J. Middelburg, The assessment of minor neurological dysfunction in infancy using the Touwen Infant Neurological

- Examination: Strengths and limitations, *Dev. Med. Child Neurol.* 52 (2010) 87–92.
<https://doi.org/10.1111/j.1469-8749.2009.03305.x>.
- [15] M. Hadders-Algra, *Examination of the Child with Minor Neurological Dysfunction* (3rd Ed.), Wiley, London, 2010.
- [16] A.A. Chrobak, A. Krupa, D. Dudek, M. Siwek, How soft are neurological soft signs? Content overlap analysis of 71 symptoms among seven most commonly used neurological soft signs scales, *J. Psychiatr. Res.* 138 (2021) 404–412.
<https://doi.org/10.1016/j.jpsychires.2021.04.020>.
- [17] I. Bombin, C. Arango, R.W. Buchanan, Assessment tools for soft signs, *Psychiatr. Ann.* 33 (2003) 170–176. <https://doi.org/10.3928/0048-5713-20030301-06>.
- [18] S.B. Richmond, K.D. Dames, D.J. Goble, B.W. Fling, Leveling the playing field: Evaluation of a portable instrument for quantifying balance performance, *J. Biomech.* 75 (2018) 102–107. <https://doi.org/10.1016/j.jbiomech.2018.05.008>.
- [19] D. Dinkel, K. Snyder, V. Molfese, A. Kyvelidou, Postural Control Strategies Differ in Normal Weight and Overweight Infants, *Gait Posture.* 55 (2017) 167.
<https://doi.org/10.1016/J.GAITPOST.2017.04.017>.
- [20] D. Bartlett, T. Birmingham, Validity and Reliability of a Pediatric Reach Test, *Pediatr. Phys. Ther.* 15 (2003) 84–92. <https://doi.org/10.1097/01.PEP.0000067885.63909.5C>.
- [21] M. Blanchet, F. Prince, J. Messier, Development of postural stability limits: Anteroposterior and mediolateral postural adjustment mechanisms do not follow the same maturation process, *Hum. Mov. Sci.* 63 (2019) 164–171.
<https://doi.org/10.1016/j.humov.2018.11.016>.

- [22] N. Kirshenbaum, C.L. Riach, J.L. Starkes, Non-linear development of postural control and strategy use in young children: A longitudinal study, *Exp. Brain Res.* 140 (2001) 420–431. <https://doi.org/10.1007/s002210100835>.
- [23] R.J. Peterka, Sensorimotor integration in human postural control, *J. Neurophysiol.* 88 (2002) 1097–1118. <https://doi.org/10.1152/jn.2002.88.3.1097>.
- [24] S. Barozzi, M. Socci, D. Soi, F. Di Berardino, G. Fabio, S. Forti, A.M. Gasbarre, D. Brambilla, A. Cesarani, Reliability of postural control measures in children and young adolescents, *Eur. Arch. Oto-Rhino-Laryngology* 2014 2717. 271 (2014) 2069–2077. <https://doi.org/10.1007/S00405-014-2930-9>.
- [25] C. Rival, H. Ceyte, I. Olivier, Developmental changes of static standing balance in children, *Neurosci. Lett.* 376 (2005) 133–136. <https://doi.org/10.1016/J.NEULET.2004.11.042>.
- [26] D.J. Goble, M.J. Rauh, H.S. Baweja, Normative data for the btracks balance test concussion-management tool: Results from 10045 athletes aged 8 to 21 years, *J. Athl. Train.* 54 (2019) 439–444. <https://doi.org/10.4085/1062-6050-178-18>.
- [27] I.K. Koerte, R. Bahr, P. Filipcik, J. Gooijers, A. Leemans, A.P. Lin, Y. Tripodis, M.E. Shenton, N. Sochen, S.P. Swinnen, O. Pasternak, REPIMPACT - a prospective longitudinal multisite study on the effects of repetitive head impacts in youth soccer, *Brain Imaging Behav.* 16 (2022) 492. <https://doi.org/10.1007/S11682-021-00484-X>.
- [28] J. Biller, G. Gruener, P.W. Brazis, DeMyer's: The Neurologic Examination. A Programmed Text (7th Ed.), New York City, 2016.
- [29] H.K. Kikkert, C. De Jong, M. Hadders-algra, Early Human Development Minor neurological dysfunction and cognition in 9-year-olds born at term, *Early Hum. Dev.*

- 89 (2013) 263–270. <https://doi.org/10.1016/j.earlhumdev.2012.10.001>.
- [30] M. Galić, A. Mikov, S. Sekulić, A. Kopitović, I.P. Starčević, Minor neurological dysfunction in children aged 5 to 7, *Vojnosanit. Pregl.* 75 (2018) 815–819. <https://doi.org/10.2298/VSP160629389G>.
- [31] M. De Jong, M. Punt, E. De Groot, R.B. Minderaa, M. Hadders-Algra, M.D.E. Jong, M. Punt, E.D.E. Groot, R.B. Minderaa, Minor neurological dysfunction in children with autism spectrum disorder, *Dev. Med. Child Neurol.* 53 (2011) 641–646. <https://doi.org/10.1111/j.1469-8749.2011.03971.x>.
- [32] M. Hadders-Algra, Two distinct forms of minor neurological dysfunction: Perspectives emerging from a review of data of the Groningen Perinatal Project, *Dev. Med. Child Neurol.* 44 (2002) 561–571. <https://doi.org/10.1017/S0012162201002560>.
- [33] C. De Jong, H.K. Kikkert, V. Fidler, M. Hadders-Algra, The Groningen LCPUFA study: No effect of postnatal long-chain polyunsaturated fatty acids in healthy term infants on neurological condition at 9 years, *Br. J. Nutr.* 104 (2010) 566–572. <https://doi.org/10.1017/S0007114510000863>.
- [34] D.J. Goble, E. Khan, H.S. Baweja, S.M. O’Connor, A point of application study to determine the accuracy, precision and reliability of a low-cost balance plate for center of pressure measurement, *J. Biomech.* 71 (2018) 277–280. <https://doi.org/10.1016/j.jbiomech.2018.01.040>.
- [35] S.M. O’Connor, H.S. Baweja, D.J. Goble, Validating the BTrackS Balance Plate as a low cost alternative for the measurement of sway-induced center of pressure, *J. Biomech.* 49 (2016) 4142–4145. <https://doi.org/10.1016/j.jbiomech.2016.10.020>.
- [36] The Math Works Inc, MATLAB, (2020) Computer Software.

- [37] T.E. Prieto, J.B. Myklebust, R.G. Hoffmann, E.G. Lovett, B.M. Myklebust, Measures of postural steadiness: Differences between healthy young and elderly adults, *IEEE Trans. Biomed. Eng.* 43 (1996) 956–966. <https://doi.org/10.1109/10.532130>.
- [38] R.M. Palmieri, C.D. Ingersoll, M.B. Stone, B.A. Krause, Center-of-pressure parameters used in the assessment of postural control, *J. Sport Rehabil.* 11 (2002) 51–66. <https://doi.org/10.1123/jsr.11.1.51>.
- [39] R Core Team, A Language and Environment for Statistical Computing, R Found. Stat. Comput. (2021). <http://www.r-project.org>.
- [40] M. Hadders-Algra, Variation and variability: Key words in human motor development, *Phys. Ther.* 90 (2010) 1823–1837. <https://doi.org/10.2522/ptj.20100006>.
- [41] U.M. Fietzek, F. Heinen, S. Berweck, S. Maute, A. Hufschmidt, J. Schulte-Mönting, C.H. Lücking, R. Korinthenberg, Development of the corticospinal system and hand motor function: Central conduction times and motor performance tests, *Dev. Med. Child Neurol.* 42 (2000) 220–227. <https://doi.org/10.1017/S0012162200000384>.
- [42] V.B. Penhune, C.J. Steele, Parallel contributions of cerebellar, striatal and M1 mechanisms to motor sequence learning, *Behav. Brain Res.* 226 (2012) 579–591. <https://doi.org/10.1016/J.BBR.2011.09.044>.
- [43] E. Bieć, M. Kuczyński, Postural control in 13-year-old soccer players, *Eur. J. Appl. Physiol.* 110 (2010) 703–708. <https://doi.org/10.1007/s00421-010-1551-2>.
- [44] R.J. Soorani-Lunsing, M. Hadders-Algra, H.J. Huisjes, B.C.L. Touwena, Minor neurological dysfunction after the onset of puberty: association with perinatal events, *Early Hum. Dev.* 33 (1993) 71–80. [https://doi.org/10.1016/0378-3782\(93\)90174-s](https://doi.org/10.1016/0378-3782(93)90174-s).

Appendix

Table A 1. Differences in Postural Control between the NSS+ and NSS- Group

Postural control	NSS+	NSS-	<i>F</i>	<i>p</i>	Note. All p-values were corrected for 14 variables and conditions at $p < .05$. Abbreviations: AP = anterior-posterior, ML = medial-lateral, PL = path length, RMS = root mean square, SD = standard deviation
Rigid					
PL	27.27 (10.95)	26.32 (8.34)	$F(1, 138) = 0.078$	$p > .999$	
PL ML	15.20 (7.86)	12.98 (4.79)	$F(1, 138) = 1.347$	$p > .999$	
PL AP	19.02 (6.69)	19.85 (6.87)	$F(1, 138) = 0.299$	$p > .999$	
RMS	0.56 (0.23)	0.54 (0.18)	$F(1, 138) = 0.052$	$p > .999$	
RMS ML	0.25 (0.12)	0.21 (0.08)	$F(1, 138) = 3.074$	$p > .999$	
RMS AP	0.49 (0.21)	0.49 (0.17)	$F(1, 138) = 0.034$	$p > .999$	
Sway area	0.23 (0.21)	0.19 (0.12)	$F(1, 138) = 1.098$	$p > .999$	
Foam					
PL	65.05 (20.83)	61.54 (14.53)	$F(1, 138) = 0.247$	$p > .999$	
PL ML	29.92 (8.71)	28.02 (8.08)	$F(1, 138) = 0.317$	$p > .999$	
PL AP	51.37 (17.97)	48.85 (11.51)	$F(1, 138) = 0.178$	$p > .999$	
RMS	1.30 (0.34)	1.26 (0.27)	$F(1, 138) = 0.033$	$p > .999$	
RMS ML	0.62 (0.16)	0.62 (0.18)	$F(1, 138) < 0.001$	$p > .999$	
RMS AP	1.13 (0.33)	1.08 (0.25)	$F(1, 138) = 0.073$	$p > .999$	
Sway area	1.14 (0.61)	1.01 (0.45)	$F(1, 138) = 0.489$	$p > .999$	

Highlights

- Neurological soft signs are related to postural control in male adolescent athletes
- Quantitative postural control assessments may complement neurological examinations
- Non-optimal performance in diadochokinesis is related to more medial-lateral sway