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Abstract

Background context: Radiographic evaluation in adult spinal deformity (ASD) offers no information on spinopelvic alignment and compensation during dynamic conditions. Motion analysis offers the potential to bridge the gap between static radiographic and dynamic alignment measurement, increasing our understanding on how ASD impacts function.

Purpose: This study aimed to explore the changes in sagittal alignment and compensation strategies in ASD between upright standing and walking, compared to control subjects and within different sagittal alignment groups. Ten patients were measured pre- and six months post-operatively to explore the impact of surgical alignment correction on gait.

Study design: Prospective study

Sample size: Full protocol: 58 ASD and 20 controls; Spinal kinematic analysis: 43 ASD and 18 controls; Post-operative analysis: 10 ASD

Outcome measures: Standing and walking sagittal spinopelvic (thoracic kyphosis (TK), lumbar lordosis (LL), sagittal vertical axis (SVA), pelvis) and lower limb kinematics, spinopelvic changes between standing and walking (Δ i.e. difference between mean dynamic and static angle), lower limb kinetics, spatiotemporal parameters, balance (BESTest), patient-reported outcome scores (SRS-22r, ODI and FES-I) and radiographic parameters.

Methods: Motion analysis was used to assess the standing and walking spinopelvic and lower limb kinematics, as well as the lower limb kinetics during walking. All parameters were compared between controls and patients with ASD, divided in three groups based on their sagittal alignment (ASD 1: decompensated sagittal malalignment; ASD 2: compensated sagittal malalignment; ASD 3: scoliosis and normal sagittal alignment). 10 patients were reassessed 6 months after spinal corrective surgery. Continuous kinematic and kinetic data were analyzed through statistical parametric mapping.

Results: All patient groups walked with increased forward trunk tilt (ΔSVA=41.43mm, $p<0.001$) in combination with anterior pelvic tilt (ΔPelvis=2.58°, $p<0.001$) compared to standing, as was also observed in controls (ΔSVA=37.86mm, $p<0.001$; ΔPelvis=1.62°, $p=0.012$). Patients walked with increased SVA, in combination with decreased LL and alterations in lower limb kinematics during terminal stance and initial swing, as well as altered spatiotemporal parameters. Subgroup analysis could link these alterations in gait to sagittal spinopelvic malalignment (ASD 1 and 2). After surgical correction, lower limb kinematics and spatiotemporal parameters during gait were not significantly improved.

Conclusions: To compensate for increased trunk tilt and pelvic anteversion during walking, patients with sagittal malalignment show altered lower limb gait patterns, which have previously been associated with increased risk of falling and secondary lower limb pathology. Since surgical correction of the deformity did not lead to gait improvements, further research on the underlying mechanisms is necessary to improve our understanding of how ASD impacts function.

Key words

Adult spinal deformity, sagittal alignment, compensation strategies, static, dynamic, gait pattern, kinematics, kinetics, motion analysis

1. Introduction

Adult spinal deformity (ASD) is characterized by a three-dimensional malalignment of the spinal curvature [1], often leading to severe disabilities such as axial back and/or leg pain [2,3], neurological deficits [1], difficulties to keep an upright posture [4], balance impairments [5–7] and increased risk of falling [8], resulting in decreased quality of life (QOL) [9–11]. The current ASD clinical evaluation is mainly based on static radiography during upright standing to quantify spinal malalignment and associated compensation strategies used by patients to maintain horizontal gaze and keep the body balanced within its support surface. These compensation strategies include pelvic retroversion, knee flexion, ankle dorsiflexion, decreased lordosis and increased kyphosis [12–17]. In addition, patient-reported outcome scores (PROMs) are used to assess the impact of spinal deformity and its surgical correction on QOL and disability [1,18,19]. However, both radiography and PROMs fail to obtain objective information on the patient's functional abilities and the impact of ASD on daily activities, given the known decreased balance of patients with ASD [5–7]. Although the introduction of balance scales contributes to a more comprehensive functional assessment, these scales do not provide any objective information on spinal alignment during dynamic activities. Motion analysis offers the potential to bridge this gap between static radiographic and dynamic alignment measurements by objectively quantifying the patient's dynamic biomechanical profile and could therefore increase our understanding on how ASD impacts function [20–22].

Motion analysis has been used previously to assess sagittal spinopelvic alignment and associated compensation strategies during standing and walking in ASD [20,22–25]. Compensation strategies used during upright standing were found to not be preserved during walking, as trunk tilt increased during gait, combined with pelvic anteversion. [24,25]. Patients with decompensated sagittal malalignment were also found to walk with tilted trunk and flexed knees (i.e. crouch gait) during terminal stance. This crouch pattern disappeared after spinal corrective surgery, suggesting that the sagittal malalignment was mainly responsible for this altered lower limb behaviour during walking [23]. However, these studies lacked a control group and reported the kinematic results as mean values over the gait cycle. Since walking is characterized by a complex interaction of largely changing joint positions over time, analysis of these joint positions over the entire gait cycle instead of mean values, would allow to link gait impairments to specific gait phases. Existing studies have predominantly focused on movement analysis of the trunk, lacking a complete evaluation of the compensation strategies of the lower limbs, including kinetics. In addition, these kinematic studies have mostly modelled the trunk as one rigid segment; this assumption is inadequate to provide information on kinematic changes within the spine. [20,23–25]

To increase our knowledge about how ASD impacts gait, this study explored how sagittal spinopelvic alignment and associated compensation strategies changed between upright standing and walking in patients with different sagittal alignment, and compared it to controls. Three hypotheses were formulated: 1) static compensation strategies are not preserved during walking [24,25]; 2) patients with sagittal malalignment, one of the main drivers of pain and disability [26], show more impairments in terms of kinematics, kinetics and spatiotemporal parameters during walking compared to controls, than patients with only coronal malalignment; 3) pre-operative gait impairments normalize after spinal corrective surgery [23].

2. Materials and methods

2.1 Ethics statement

This study was approved by the ethical committee of the university hospitals Leuven (S58082) and all subjects provided written informed consent.

2.2 Participants

Fifty-eight patients, with de novo degenerative scoliosis, progressive adolescent idiopathic scoliosis into adulthood, hyperkyphosis and/or flat back deformity, were recruited from our outpatient spinal clinic between February 2016 and March 2019. All patients were receiving conservative care, consisting of general physiotherapy and/or pain control through analgetics, when entering the study. Patients had to be older than 18 years and be able to walk at least 50 meters without a walking aid. Exclusion criteria were iatrogenic spinal deformity or previous spinal fusion, post-traumatic cause of spinal deformity and conditions which might compromise walking such as severe lower extremity musculoskeletal disorders (hip or knee arthroplasty) or neurological conditions. A control group of 20 age-matched healthy adults was recruited through online and poster advertising in the hospital and university. Inclusion criteria for controls were: age older than 18 and able to walk at least 1000 meters independently. A current history of back pain and lower extremity musculoskeletal or neurological conditions led to exclusion.

The total ASD group was further subdivided in three groups according to their sagittal alignment, based on 2D spinopelvic alignment parameters of the SRS-Schwab classification [27]:

1. **ASD 1**: Patients with decompensated sagittal malalignment (SVA>4cm with PI-LL>10° and/or PT>20°) ± coronal deformity;
2. **ASD 2**: Patients with compensated sagittal malalignment (SVA<4cm with PI-LL>10° and/or PT>20°) ± coronal deformity;

3. **ASD 3**: Patients with coronal malalignment (Cobb angle ≥20°) and non-pathological sagittal alignment.

A subset of ten patients (ASD 1: 5, ASD 2: 3, ASD 3: 2) received spinal corrective surgery and were re-evaluated six months post-operatively. Indications for surgery were uncontrollable pain irresponsive to conservative care, QOL decrease and/or curve progression. Surgical procedures for spinal correction included spinal instrumentation (± interbody fusion) with iliac fixation, corrective osteotomy and/or spinal decompression. The upper instrumented vertebra varied between L2 and T4, while the lower instrumented level was the sacrum. All patients received iliac fixation, except for one.

Due to limited marker visibility during biplanar imaging and motion analysis, approximately 20% of the subjects were excluded from the spinal kinematic analysis. Exact sample sizes for each analysis is shown in the flow chart (Fig 1).

2.3 Data collection

2.3.1 Biplanar radiographic exam

Biplanar radiographic images (EOS, EOS Imaging, Paris, France) were taken to measure 2D spinopelvic alignment according to the SRS-Schwab classification [27], necessary for subgroup division. Coronal alignment was measured by the deviation of the C7 plumbline from the coronal sacral midline (referred to as coronal vertical axis (CVA)) [28]. The exam was performed with the skin markers for motion analysis attached, to determine the relative 3D position of the markers and the vertebral bodies, necessary for subject-specific spinal marker position correction (See 2.4 and Fig 2B).

2.3.2 Patient-reported outcome scores

All subjects completed the Scoliosis Research Society outcome questionnaire (SRS-22r) [29], the Oswestry Disability Index (ODI) [30] and the Fall Efficacy Scale – International (FES-I) [31].

2.3.3 Balance assessment

Balance performance was measured using the Balance Evaluation Systems Test (BESTest) [32].

2.3.3 Kinematics and kinetics

Subjects were instrumented according to the lower limb Plug-in-gait (PiG) marker model (Vicon Motion systems, Oxford Metrics, UK) [33] in combination with a spinal marker model consisting of 6 single markers (C7, T5, T9, T12, L3 and on sacrum, in the middle between left and right posterior superior iliac spine), and 6 clusters, of three markers each, in between (T1, T3, T7, T11, L2, L4) [22]

(Fig 2.A). In the motion lab, all subjects were asked to stand still for three seconds, without shoes and with the arms alongside the body, for model calibration and capture of their static posture. After familiarization, three overground walking trials at self-selected speed on a 10m walkway were recorded. Marker trajectories were recorded using a 10-camera motion capture system (VICON Motion systems, Oxford Metrics, UK), sampling at 100 Hz. Ground reaction forces during walking were recorded at 1500 Hz with an embedded force plate (AMTI force and motion, Watertown, MA, USA).

2.4 Data analysis of kinematics and kinetics

For each subject, three left gait cycles (heel strike to heel strike) were recorded, processed and averaged. Kinematic and kinetic data were filtered using a low-pass Butterworth filter at 100 Hz and resampled at 51 samples per gait cycle, using custom-made MATLAB software (version 9.7, The Mathworks Inc., Natick, MA, USA).

Lower limb sagittal plane kinematics were calculated for the pelvis, hip, knee and ankle according to the PiG model [33] (Fig 2.A.2). Sagittal internal net joint moments, i.e. joint kinetics, of the hip, knee and ankle were calculated during walking using inverse dynamics based on the measured ground reaction forces. The following spatiotemporal parameters were calculated: cadence, step length, step time, velocity, stride time, stride length, duration of single and double support phase and foot-off timing.

Intersegmental spinal kinematics were evaluated with a previously validated subject-specific method [22]. Briefly, this method fits a polynomial through anatomy-corrected marker positions to measure thoracic kyphosis (TK), lumbar lordosis (LL) and sagittal vertical axis (SVA). The subject-specific spinal deformity of the patient was taken into account by correcting the marker positions to the true anatomical positions of the corresponding vertebral bodies, as measured on biplanar images (Fig 2.B). The resulting 3D offsets between the markers and vertebral bodies were then used as an invariant correction term for the marker positions in the motion trial. A polynomial was fitted through these corrected markers. Angles between the levels instrumented with a marker were then defined by the normals to this polynomial in the sagittal plane (Fig 2.B.3). TK was measured between T1 and the level closest to the inflection point of the curve. LL was measured between the level closest to the inflection point and the sacrum marker. The SVA was defined by the distance between the sacrum marker and the vertical projection of the C7 marker on the sacral transverse plane. (Fig 2.B.3) [22]

Sagittal kinematics for all joints were calculated for both upright standing and the entire gait cycle. Ranges of motion (ROM) during walking were also obtained. The mean angles of pelvis, TK, LL and

SVA over the gait cycle were measured to calculate the changes in spinopelvic alignment between upright standing and walking (Δ i.e. the difference between the mean dynamic and the static angle).

2.5 Statistical analysis

A Shapiro-Wilk test was used to test the normality of the discrete data: demographic, radiographic, balance, PROMs, spatiotemporal, static kinematic, ROM parameters and the alteration in spinopelvic alignment between upright standing and walking (Δ). Since most discrete data were found to be non-normally distributed and the sample size in the individual subsets was small, non-parametric statistics were used for all discrete parameters. An independent-samples Wilcoxon signed-rank test was performed to compare the ASD group, as well as pre- and post-operative conditions, to controls. For the ASD subgroup analysis, a Kruskal-Wallis test with post-hoc Bonferroni correction for multiple testing was used. Pre- to post-operative analysis was performed with the related Wilcoxon signed-rank test. All statistics on discrete parameters have been performed in SPSS 26 (IBM Corp. Armonk, NY, USA).

Continuous kinematic and kinetic data during walking were analyzed using Statistical Parametric Mapping (SPM1d version 0.4) [34]. Since the assumption of normality was fulfilled, parametric unpaired t-tests were performed to compare curves between controls and the total ASD group and between controls and the pre- and post-operative conditions. ANOVA was used for subgroup analysis and paired t-tests to compare pre- to post-operative conditions.

Significance level was set at 0.05 for all analyses. Due to the explorative character of the study, correction for multiple testing of different parameters was deliberately not performed to maximize the power and avoid increasing the risk for false negatives (type II error) [35].

3. Results

3.1 Participants

ASD and control subjects did not differ in age, weight, gender and BMI, although controls were taller ($p=0.003$). The total ASD group (ASD: 18.8; Control: 8.6; $p<0.001$), as well as ASD 1 and 2 and surgical patients before and after surgery, showed significantly larger CVA compared to controls. ASD scored significantly worse on SRS-22r (ASD: 3.2; Control: 4.6; $p<0.001$), ODI (ASD: 31.0; Control: 0.0; $p<0.001$) and FES-I (ASD: 25.0; Control: 17.0; $p<0.001$), but between ASD subgroups no significant differences were found. Post-operatively, SRS-22r improved significantly (Pre-op: 2.8; Post-op: 3.7; $p=0.005$) in contrast with ODI and FES-I. The sagittal ASD patients (ASD 1 & 2) performed worse on BESTest compared to controls, in contrast with ASD 3 (Controls: 94.0%; ASD 1: 81.0%, $p<0.001$; ASD

2: 86.1%, p=0.005). Balance performance did not improve significantly six months post-operatively (p=0.594). (Table 1)

3.2 Kinematics – kinetics

3.2.1 Upright standing

The total ASD group demonstrated increased SVA during upright standing compared to controls (ASD: 33.3mm; Controls: -0.1mm, p<0.001) in combination with increased knee and ankle (dorsi)flexion and decreased LL (ASD: 46.8°; Control: 54.7°; p=0.033). Subgroup analysis showed that this increased SVA (ASD 1: 69.2mm) and increased knee and ankle (dorsi)flexion were mainly observed in ASD 1 and that sagittal patients (ASD 1 & 2) also showed pelvic retroversion compared to ASD 3 (ASD 1: 1.2°; ASD 2: -0.3°; ASD 3: 7.0°; p=0.042/0.044). SVA decreased post-operatively and did not significantly differ from control values (Pre: 45.6mm; Post: 19.5mm; Controls: -0.1mm; $p_{pre\text{-}post}$=0.028; $p_{post\text{-}control}$=0.265), in contrast with lower limb kinematics at the level of the hip, knee and ankle. (Fig 3A and table A1)

3.2.2 Upright standing versus walking

All subject groups increased trunk tilt during walking compared to standing, as represented by an increased SVA (Control: ΔSVA=37.9mm, p<0.001; ASD: ΔSVA=41.4mm, p<0.001) and pelvic anteversion (Control: ΔPelvis=1.6°, p=0.012; ASD: ΔPelvis=2.6°, p<0.001). Controls and the total ASD group also increased TK (Control: ΔTK=0.6°, p=0.043; ASD: ΔTK=0.9°, p=0.010) and decreased LL (Control: ΔLL=-3.9°; p=0.008; ASD: ΔLL=-3.2°, p<0.001), but subgroup analysis showed that these differences were not significant in every group, nor in the pre- and post-op conditions. (Fig 3B and table A1)

3.2.3 Kinematics and kinetics during walking

ASD subjects walked with increased SVA over the entire gait cycle, in combination with a decreased LL, compared to controls (Fig 4A). This increased SVA was especially present in the decompensated ASD 1 group compared to all other groups (Fig 4B). ASD 2 showed decreased LL over the entire gait cycle compared to controls, in combination with an increased pelvic retroversion compared to ASD 3 (Fig 4C). During terminal stance, ASD subjects showed a decreased knee flexion moment compared to controls (Fig 4A). The subgroup analysis revealed a decreased knee flexion moment at the end of midstance going into terminal stance in ASD 1, in combination with an increased knee flexion angle (Fig 4B). During toe off going into initial swing, a decreased knee flexion in ASD subjects compared to controls was observed (Fig 4A), especially in ASD 2 (Fig 4C).

Pre-operative patients showed increased SVA over the entire gait cycle and a decreased internal knee flexion moment and increased ankle dorsiflexion during terminal stance and initial swing (Fig 4E). Increased knee and hip flexion and ankle dorsiflexion, in combination with decreased internal knee and hip moments, were observed post-operatively from terminal stance to initial swing (Fig 4F).

Detailed kinematic and kinetic curves are included in figures A1 to A4 of the appendix.

3.2.4 Range of motion

ASD subjects showed decreased ROM of the hip (ASD: 41.2°; Control: 45.7°; p=0.001) and knee (ASD: 54.5°; Control: 58.1°; p=0.001), mainly observed in ASD 1 & 2. Subgroup analysis revealed also significantly increased ROM for LL in ASD 2 & 3 compared to ASD 1 and controls (ASD 1: 6.8°; ASD 2: 10.8°; ASD 3: 9.9°; Control: 7.0°; p=0.003) and for TK in ASD 3 (ASD 3: 3.8°; Control: 2.0°; p=0.040). (Table 2)

Post-operatively, patients showed decreased spinal ROM in both LL (Control: 7.01°; Post: 2.73°; p=0.001) and TK (Pre: 2.51°; Post: 1.78°; p=0.047). Pre-operative decreased lower limb ROM at the level of hip (Pre: 35.91°; Control: 45.71°; p=0.001) and knee (Pre: 51.76°; Control: 58.10°; p<0.001) did not change post-operatively (Hip: Post: 37.74°, p<0.001; Knee: Post: 52.75°, p=0.007). (Table 2)

3.2.5 Spatiotemporal parameters

All spatiotemporal parameters, including velocity (Control: 1.32m/s; ASD: 1.06m/s; p<0.001), step length (Control: 0.64; ASD: 0.56; p<0.001), and duration of double support (Control: 0.24s; ASD: 0.28s; <0.001), were impaired in ASD, except for single support duration (p=0.158) compared to controls. Subgroup analysis revealed that significant differences were present in ASD 1 and ASD 2, but not in ASD 3. Spatiotemporal parameters did not significantly change post-operatively. (Table 3)

4. Discussion

This study investigated how sagittal spinopelvic alignment and compensation strategies in patients with ASD changed between upright standing and walking, compared to healthy controls and within ASD subgroups with different sagittal alignment. To assess the impact of surgical alignment correction on gait patterns, ten patients were re-evaluated six months after spinal corrective surgery.

Similarly to previous results in the literature [24,25], trunk tilt and pelvic anteversion increased between standing and walking for all patient groups. However, prior studies describing these strategies in young healthy adults [36,37] as well as the fact that our control group adopted the same strategies, indicate that forward trunk tilt and pelvic anteversion are normal biomechanical strategies

necessary to allow the center of gravity to progress forward, instead of a loss of static compensation as was hypothesized [24,25]. Nevertheless, this further increase of their statically increased trunk tilt might put these patients further at risk of falling during dynamic conditions [8], also reflected in their increased self-reported fear of falling (FES-I) and decreased balance capacity (BESTest).

To compensate for this further increase in SVA during walking, patients with ASD adopt compensation strategies during parts of the gait cycle, which correspond to those observed during upright standing. More specifically, in the total ASD group a decreased knee moment during terminal stance, suggesting crouch gait, and decreased LL over the entire gait cycle were observed. The subgroup analysis revealed that these patterns were mainly observed in the sagittal deformity groups (ASD 1 and 2). Coronal deformity patients (ASD 3), with normal sagittal alignment, showed no differences in gait patterns, nor in spatiotemporal parameters and balance (BESTest), compared to controls, confirming the hypothesis that mainly sagittal malalignment is associated with impaired gait.

Increased trunk tilt in combination with a lower limb crouch pattern, including increased knee flexion and decreased knee flexion moment, during terminal stance was mainly observed in the ASD 1 subgroup. These findings correspond to the results on trunk and knee kinematics in sagittally decompensated patients by Gottipati et al. [23] and Kim et al. [38]. Research on able-bodied gait showed that increased trunk flexion directly resulted in crouch gait [39] and altered lower-limb kinetics [40], confirming this crouch pattern served to allow upright balance during walking. However, it might lead to secondary lower limb pathology. The persisting internal knee extension moment during stance phase, observed in ASD 1, which is necessary to maintain this knee flexed position, requires increased energy expenditure, leading to early muscle fatigue [40], and in the longer term might lead to increased knee joint loading, and consequently degeneration [41]. The observed altered spatiotemporal parameters, such as slower walking and decreased step length, might serve to protect lower limb joints from this increased loading [39].

The ASD 2 subgroup showed decreased LL over the entire gait cycle compared to controls, in combination with a pelvic retroversion compared to ASD 3, confirming that also compensated patients adopted compensation strategies during walking. During initial swing a stiff knee pattern was also observed in ASD, and especially in the ASD 2 subgroup. This pattern is characterized by a decreased knee flexion which compromises foot clearance, and might therefore lead to tripping, the main cause of falls in elderly [42]. Previous research found that stiff knee gait is associated with altered activity of the quadriceps [43] and decreased force production of iliopsoas [44]. Future research should therefore investigate whether the observed spinopelvic compensation strategies

during walking in ASD, namely decreased LL and pelvic retroversion, lead to alterations in spinopelvic muscle activities during initial swing, possibly causing stiff knee gait.

In accordance with the post-operative static profile, the crouch pattern during terminal stance was still present or even worsened post-operatively. This is in contrast with the findings of Gottipati et al. [23] and therefore the hypothesis that spinal correction surgery would normalize the gait pattern is rejected. Since sagittal alignment was surgically corrected, other underlying mechanisms might contribute to these observed alterations in lower limb behavior. Decreased lower limb strength [45], persisting contractures in the lower limb joints and muscles or associated joint degeneration due to increased loading have previously been associated with crouch gait [41]. Future research should investigate whether these factors contribute to crouch gait in ASD and if specific treatments targeting these underlying mechanisms, such as lower limb strengthening [45], stretching of shortened muscles [46] and gait retraining [47], could ultimately improve gait efficiency in ASD, both in the conservative as well as the pre- to post-operative setting.

There are some limitations associated with this study. Firstly, the heterogeneity of the surgical group, in terms of surgical indication and instrumented levels, might have influenced the results. An explorative analysis could not clearly show the influence of different fusion lengths or pre-operative alignment on gait pattern differences; however, the small sample size (n=10) does not allow for reliable statistical analysis. Nevertheless, future research should further investigate the specific effect of surgical parameters, preferably through longer follow-up to investigate how these gait deviations change over time. Secondly, due to inferior marker visibility in the gait lab and on radiography, not all subjects were included in the spinal kinematic analysis (Fig 1). Changing camera positions could improve marker visibility in the gait lab, but due to multidisciplinary use this was not possible. Thirdly, only left gait cycles were analyzed, since pilot work showed mainly symmetrical gait in ASD. However, asymmetrical gait has been reported [48], and therefore kinematic and kinetic differences might have been underestimated. Fourthly, this study mainly focused on the impact of sagittal alignment on sagittal gait parameters. However, differences between groups on coronal alignment (CVA) suggest that future research should also investigate the specific impact of coronal imbalance on gait, since it has previously been related to pain and dysfunction [28]. Lastly, although other conditions possibly affecting gait, such as lower limb arthroplasty or neurological pathologies, led to exclusion, the study did not control for the presence of stenosis, which is known to lead to gait impairments [49]. While this might have influenced our results, a study of Kim et al. [38] showed that positive sagittal imbalance has greater impact on gait parameters than stenosis. Nevertheless, the impact of combined sagittal imbalance and stenosis warrants further investigation.

Conclusions

Increased trunk tilt and pelvic anteversion during walking might challenge patients with static sagittal malalignment even more to keep upright balance. Therefore, these patients adopt spinopelvic and lower limb compensations during parts of the gait cycle, resulting in specific gait patterns, such as crouch and stiff knee gait, which have previously been associated with increased risk of falling and secondary lower limb pathology. Since surgical correction of the deformity did not lead to gait improvements, spinal malalignment might not be the only underlying mechanism. Future research on gait alterations and possible underlying mechanisms, including altered muscle activity and weakness, could further increase our understanding on how ASD impacts function. Ultimately, this information can be used to develop specific treatments targeting these underlying mechanisms, to improve gait efficiency in ASD.

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Figure 1. Flow chart.
ASD: Adult spinal deformity; PROMs: Patient-reported outcome scores; BESTest: Balance Evaluation Systems Test; SVA: Sagittal vertical axis; TK: Thoracic kyphosis; LL: Lumbar lordosis.

Figure 2. Measurement methodology for kinematic analysis.
Figure A describes the spinal marker protocol (A.1), the Plug-in-gait lower limb angle definitions (A.2) and the motion analysis protocol (A.3). Figure B displays the polynomial method, with the definition of the 3D position of markers and vertebral bodies during biplanar x-rays (B.1), the marker position correction towards anatomical body positions (B.2) and the polynomial fit and spinal angle definitions (B.3)
a. normal to the polynomial; b. inflection point of the curve.
(Figure 1B edited from P. Severijns et al. Spine J 2020. doi:10.1016/j.spinee.2020.02.004.)

Figure 3. Static and dynamic sagittal profile of patients with ASD compared to controls.
Figure 3A shows the static profile for each group. In figure 3B alterations in spinopelvic profile from standing to walking are presented by arrows indicating where differences occur (red: increase: blue: decrease). Exact values on static alignment and alterations between standing and walking can be found in Table A1 of the Appendix. Δ: Difference between the mean dynamic angle and the static angle; SVA: Sagittal vertical axis; TK: Thoracic kyphosis; LL: Lumbar lordosis.
Medians and interquartile ranges (IQR) are reported. Significance level: $p < 0.05$.

Figure 4. Sagittal spinopelvic and lower limb kinematics and kinetics during overground walking in patients with ASD compared to controls.
For each patient group (A-F) the sagittal kinematic profile at each event of the gait cycle is presented. Arrows and lines (red: increase; blue: decrease) indicate in which joints significant differences occur. Kinetic differences are indicated with bars underneath the kinematic profiles. The tables on the right show during which percentages of the gait cycle and to which groups these differences occur, as well as the p-values of each significant difference, obtained through Statistical Parametric Mapping. Significance level: $p < 0.05$.

Table 1. Demographics, radiographic parameters, patient-reported outcome scores and balance assessment.

	A. Control vs ASD			**B. Pre- vs post-op**		*p -value*		
	Control	ASD total	*p-value*	Pre-op	Post-op	*Pre vs Post*	*Pre vs Control*	*Post vs Control*
A. Demographics								
Age (year)	64.5 (15.0)	62.0 (14.3)	0.775	64.15 (8.8)	64.8 (9.7)	**0.018**	1.000	0.812
Height (cm)	167.5 (7.8)	160.3 (9.0)	**0.003**	161.25 (12.3)	162.7 (11.9)	**0.011**	**0.031**	0.074
Weight (kg)	66.9 (14.1)	63.2 (13.2)	0.591	62.25 (21.1)	63.3 (17.7)	0.123	0.880	0.983
BMI (kg/m^2)	23.9 (3.4)	24.5 (5.3)	0.137	24.91 (7.9)	24.8 (7.4)	0.859	0.475	0.373
Gender (F/M)	14F/6M	49F/9M	0.156	7F/3M	7F/3M	1.000	1.000	1.000
B. Radiographic parameters								
PT (°)	21.4 (12.7)	21.9 (13.4)	0.464	19.8 (17.2)	19.3 (14.7)	0.721	0.948	0.650
SVA (mm)	11.5 (36.0)	35.4 (52.5)	**<0.001**	40.0 (117.7)	24.0 (42.4)	0.169	**0.001**	0.109
PI minus LL (°)	4.1 (22.1)	10.7 (27.9)	**0.002**	12.8 (31.2)	-2.4 (18.6)	**0.007**	**0.005**	0.650
Coronal (D/T/L/N)	20 N	20D/33L/5N	**<0.001**	3D/5L/2N	10 N	**<0.001**	**<0.001**	1.000
CVA (mm)	8.6 (11.9)	18.8 (27.5)	**<0.001**	25.1 (49.9)	17.4 (27.3)	0.575	**0.024**	**0.017**
C. PROMs								
SRS-22	4.6 (0.4)	3.2 (1.1)	**<0.001**	2.8 (1.0)	3.7 (0.9)	**0.005**	**<0.001**	**<0.001**
ODI	0.0 (5.9)	31.0 (26.5)	**<0.001**	48.0 (20.5)	37.9 (30.3)	0.169	**<0.001**	**<0.001**
FES-I	17.0 (3.0)	25.0 (17.8)	**<0.001**	25.0 (16.0)	27.5 (21.8)	0.833	**0.002**	**<0.001**
D. Balance assessment								
BESTest (%)	93.98 (6.25)	86.11 (11.57)	**<0.001**	80.56 (14.81)	81.48 (7.87)	0.594	**<0.001**	**<0.001**

	C. Between group analysis					*Post-hoc differences between groups*					
	Control	ASD 1	ASD 2	ASD 3	*p-value*	C vs 1	C Vs 2	C Vs 3	1 Vs 2	1 Vs 3	2 Vs 3
A. Demographics											
Age (year)	64.5 (15.0)	66.0 (14.8)	63.0 (14.0)	58.0 (14.0)	0.401						
Height (cm)	167.5 (7.8)	159.8 (9.4)	162.0 (11.0)	160.0 (12.0)	**0.028**	0.024					
Weight (kg)	66.9 (14.1)	62.3 (8.4)	63.4 (17.6)	65.0 (13.8)	0.949						
BMI (kg/m^2)	23.9 (3.4)	24.6 (5.8)	24.2 (4.4)	24.6 (5.2)	0.495						
Gender (F/M)	14F/6M	21F/3M	14F/5M	14F/1M	0.228						
B. Radiographic parameters											
PT (°)	21.4 (12.7)	27.1 (19.4)	26.1 (10.2)	17.2 (4.3)	**0.004**					0.014	0.005
SVA (mm)	11.5 (36.0)	94.1 (64.8)	25.1 (25.9)	16.1 (13.4)	**<0.001**	<0.001			<0.001	<0.001	
PI minus LL (°)	4.1 (22.1)	24.9 (23.1)	13.6 (14.5)	-3.3 (8.0)	**<0.001**	<0.001	0.014			<0.001	0.003
Coronal (D/T/L/N)	20 N	5D/16L/3N	7D/10L/2N	8D/7L	**<0.001**	<0.001	<0.001	<0.001			
CVA (mm)	8.6 (11.9)	20.4 (30.4)	24.6 (19.9)	13.8 (18.3)	**0.001**	0.002	0.009				
C. PROMs											
SRS-22	4.6 (0.4)	3.1 (1.2)	2.9 (1.3)	3.5 (0.4)	**<0.001**	<0.001	<0.001	<0.001			
ODI	0.0 (5.9)	32.7 (30.8)	32.0 (26.0)	24.0 (18.0)	**<0.001**	<0.001	<0.001	<0.001			
FES-I	17.0 (3.0)	25.0 (22.5)	28.0 (19.3)	22.0 (9.0)	**0.001**	0.002	0.011				
D. Balance assessment											
BESTest (%)	94.0 (6.3)	81.0 (14.8)	86.1 (11.6)	90.7 (5.6)	**<0.001**	<0.001	0.005			0.005	

Medians and (interquartile ranges) are reported; Significance level: $p < 0.05$.

BMI: Body Mass Index; F: Female; M: Male; PT: Pelvic tilt; SVA: Sagittal vertical axis; PI: Pelvic incidence; LL: Lumbar lordosis; Coronal: SRS-Schwab Coronal classification; D: Double; T: Thoracic; L: Lumbar; N: No Major Coronal Deformity; CVA: Coronal vertical axis; PROMs: Patient-reported outcome scores; SRS-22r: Scoliosis Research Society Outcomes Questionnaire; ODI: Oswestry Disability Index; FES-I: Falls Efficacy Scale-International; BESTest: Balance Evaluation Systems Test.

Table 2. Range of motion during walking.

	A. Control vs ASD			B. Pre- vs post-op		*p -value*		
	Control	ASD total	*p-value*	Pre-op	Post-op	*Pre vs Post*	*Pre vs Control*	*Post vs Control*
SVA (mm)	21.0 (8.4)	23.5 (9.4)	0.282	22.0 (17.8)	18.6 (9.9)	0.445	0.555	0.382
TK (°)	2.0 (1.5)	2.8 (3.9)	0.064	2.5 (4.0)	1.8 (1.7)	**0.047**	0.208	0.524
LL (°)	7.0 (4.2)	8.2 (5.4)	0.094	6.9 (3.1)	2.7 (2.9)	**0.005**	0.869	**0.001**
Pelvis (°)	3.0 (1.3)	3.4 (1.4)	0.514	3.6 (1.8)	2.9 (1.9)	0.333	0.914	0.422
Hip (°)	45.7 (5.1)	41.2 (9.3)	**0.001**	35.9 (9.6)	37.7 (7.0)	0.646	**0.001**	**<0.001**
Knee (°)	58.1 (3.4)	54.5 (7.56)	**0.001**	51.8 (4.7)	52.8 (6.2)	**0.028**	**<0.001**	**0.007**
Ankle (°)	28.1 (5.6)	28.4 (9.5)	0.891	25.7 (6.6)	27.5 (8.4)	0.508	0.286	0.502

	C. Between group analysis					*Post-hoc differences between groups*					
	Control	ASD 1	ASD 2	ASD 3	*p-value*	C vs 1	C Vs 2	C Vs 3	1 Vs 2	1 Vs 3	2 Vs 3
SVA (mm)	21.0 (8.4)	23.4 (8.6)	28.8 (16.1)	21.4 (7.4)	0.216						
TK (°)	2.0 (1.5)	2.6 (2.8)	2.2 (3.7)	3.8 (3.5)	**0.040**			0.024			
LL (°)	7.0 (4.2)	6.8 (3.1)	10.8 (7.4)	9.9 (6.2)	**0.003**		0.016	0.006		0.030	
Pelvis (°)	3.0 (1.3)	3.2 (1.5)	3.5 (1.3)	3.3 (1.2)	0.561						
Hip (°)	45.7 (5.1)	37.4 (10.5)	42.9 (7.0)	41.6 (9.6)	**0.001**	<0.001					
Knee (°)	58.1 (3.4)	52.5 (8.8)	54.5 (9.4)	56.1 (5.6)	**0.002**	0.001	0.031				
Ankle (°)	28.1 (5.6)	27.2 (8.5)	27.7 (6.5)	33.9 (11.9)	0.483						

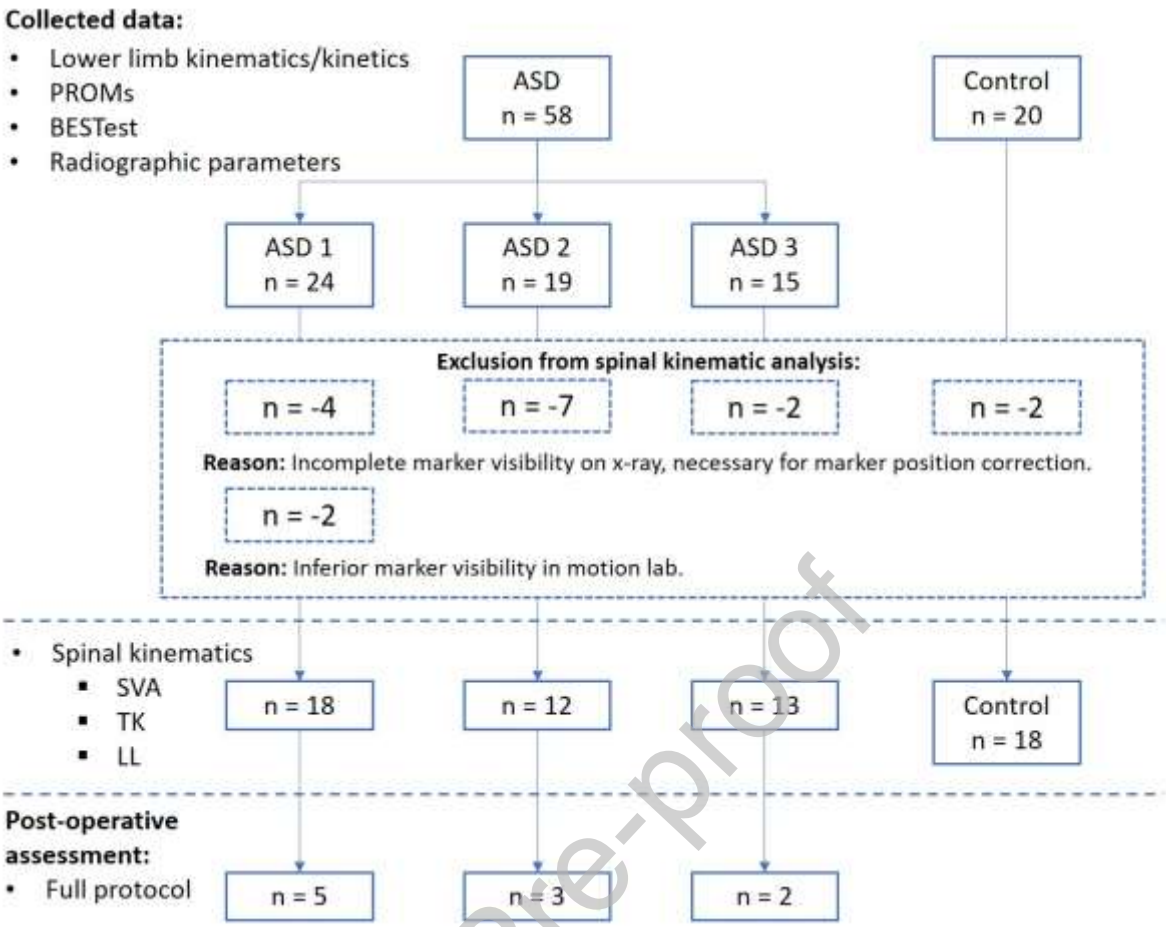
Medians (and interquartile ranges) are reported. Significance level: $p < 0.05$.
SVA: sagittal vertical axis; TK: thoracic kyphosis LL: lumbar lordosis; C: Control; 1: ASD 1; 2: ASD 2; 3: ASD 3.

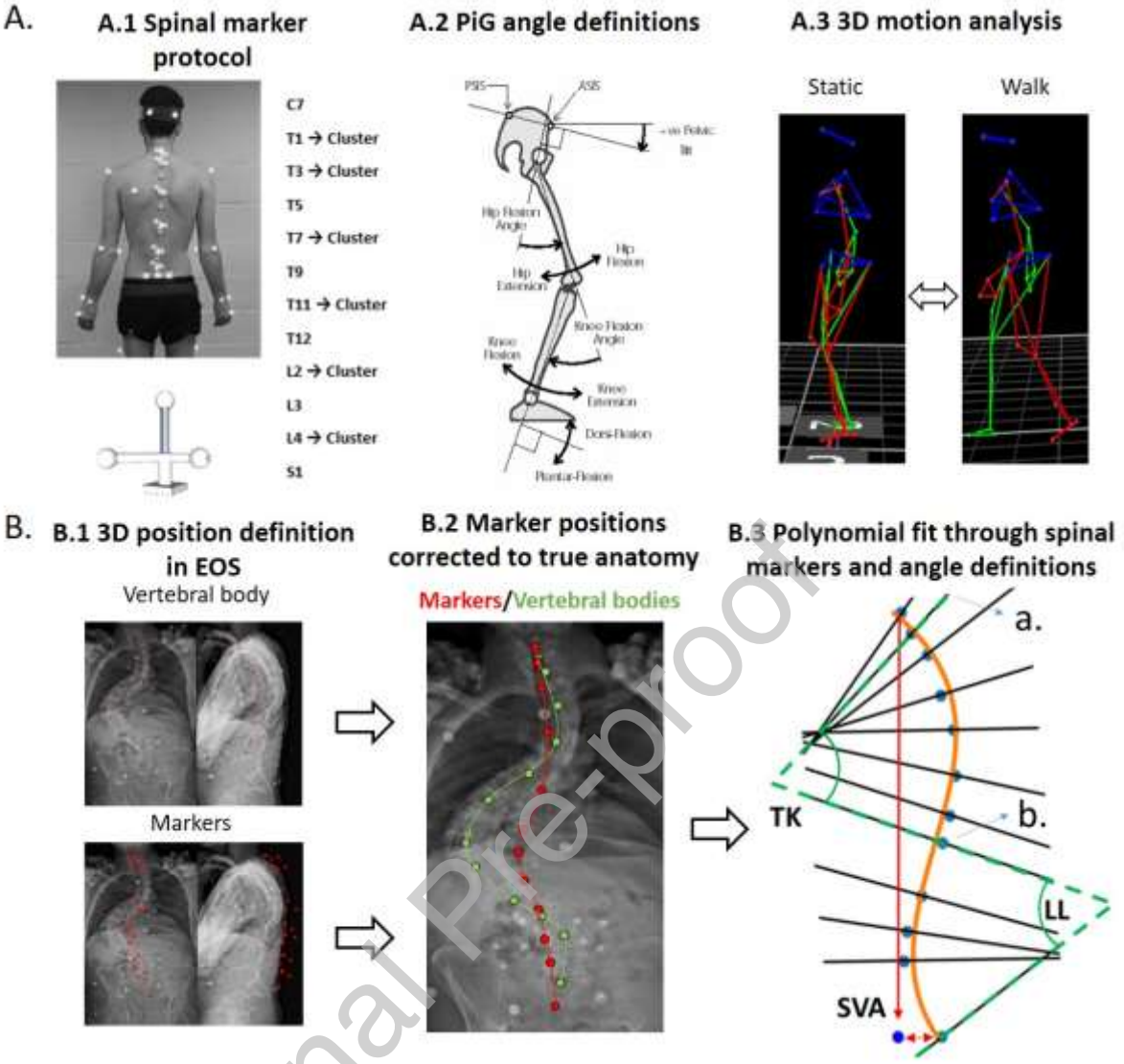
Table 3. Spatiotemporal parameters during walking.

	A. Control vs ASD			B. Pre- vs post-op		p -value		
	Control	ASD total	*p-value*	Pre-op	Post-op	*Pre vs Post*	*Pre vs Control*	*Post vs Control*
Cadence (/s)	2.0 (0.2)	1.8 (0.3)	**0.001**	1.8 (0.3)	1.9 (0.4)	0.959	**0.005**	**0.024**
Step length (m)	0.6 (0.1)	0.6 (0.1)	**<0.001**	0.5 (0.1)	0.5 (0.1)	0.575	**<0.001**	**<0.001**
Step time (s)	0.5 (0.1)	0.5 (0.1)	**0.001**	0.6 (0.1)	0.6 (0.1)	0.575	**0.001**	**0.010**
Velocity (m/s)	1.3 (0.2)	1.1 (0.2)	**<0.001**	1.0 (0.3)	1.0 (0.2)	0.878	**<0.001**	**<0.001**
Stride length (m)	1.3 (0.1)	1.1 (0.2)	**<0.001**	1.0 (0.1)	1.1 (0.1)	0.878	**<0.001**	**<0.001**
Stride time (s)	1.0 (0.1)	1.1 (0.1)	**0.001**	1.1 (0.2)	1.1 (0.3)	0.878	**0.005**	**0.028**
Single support (s)	0.4 (0.0)	0.4 (0.0)	0.158	0.4 (0.1)	0.4 (0.1)	0.646	0.169	0.120
Double support (s)	0.2 (0.1)	0.3 (0.1)	**<0.001**	0.3 (0.1)	0.3 (0.1)	0.314	**0.001**	**0.044**
Foot-off (%)	62.1 (1.7)	63.2 (2.6)	**0.003**	63.9 (3.7)	62.0 (2.5)	0.203	0.131	0.746

	C. Between group analysis					*Post-hoc differences between groups*					
	Control	ASD 1	ASD 2	ASD 3	*p-value*	C vs 1	C Vs 2	C Vs 3	1 Vs 2	1 Vs 3	2 Vs 3
Cadence (/s)	2.0 (0.2)	1.9 (0.2)	1.8 (0.3)	2.0 (0.3)	**0.003**	0.044	0.002				
Step length (m)	0.6 (0.1)	0.5 (0.1)	0.6 (0.1)	0.6 (0.1)	**<0.001**	<0.001	0.017				
Step time (s)	0.5 (0.1)	0.5 (0.1)	0.6 (0.1)	0.5 (0.1)	**0.010**		0.011				
Velocity (m/s)	1.3 (0.2)	1.0 (0.3)	1.1 (0.2)	1.1 (0.2)	**<0.001**	<0.001	<0.001				
Stride length (m)	1.3 (0.1)	1.1 (0.2)	1.2 (0.2)	1.2 (0.2)	**<0.001**	<0.001	0.029				
Stride time (s)	1.0 (0.1)	1.1 0.1)	1.1 (0.2)	1.0 (0.1)	**0.002**	0.044	0.002				
Single support (s)	0.4 (0.0)	0.4 (0.0)	0.4 (0.1)	0.4 (0.0)	**0.041**						
Double support (s)	0.2 (0.1)	0.3 (0.1)	0.3 (0.1)	0.3 (0.1)	**<0.001**	<0.001	0.004				
Foot-off (%)	62.1 (1.7)	64.0 (3.0)	62.8 (2.3)	62.5 (3.2)	**0.005**	0.002					

Medians (and interquartile ranges) are reported. Significance level: $p < 0.05$. C: Control; 1: ASD 1; 2: ASD 2; 3: ASD 3.

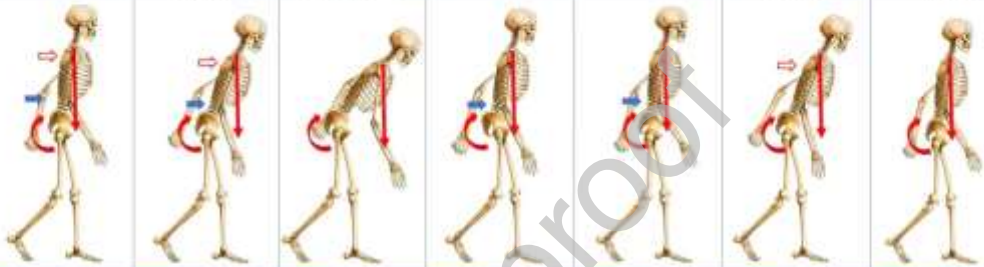




	Control	ASD	ASD 1	ASD 2	ASD 3	Pre-op	Post-op
A. Static profile							

Parameter	Different from	Different from	Different from	Different from	Different from	Different from	Different from
SVA		Control	Control / ASD 3			Control / Post-op	
TK							
LL		Control					
Pelvis			ASD 3	ASD 3			
Hip							Control
Knee		Control	Control / ASD 2			Control	Control
Ankle		Control	Control / ASD 2			Control	Control

B. Alterations from standing to walking



Parameter	Median (IQR)	p-value	Median (IQR)	p-value	Median (IQR)	p-value	Median (IQR)	p-value	Median (IQR)	p-value	Median (IQR)	p-value	Median (IQR)	p-value
Δ SVA	37.9 (41.4)	**<0.001**	41.4 (35.2)	**<0.001**	40.6 (34.7)	**0.006**	52.0 (32.9)	**0.008**	30.6 (33.6)	**0.002**	59.0 (66.0)	**0.013**	38.5 (26.2)	**0.005**
Δ TK	0.6 (1.9)	**0.043**	0.9 (4.6)	**0.010**	1.3 (6.2)	0.078	0.9 (4.5)	0.084	0.5 (3.3)	0.279	1.8 (4.6)	**0.013**	1.2 (2.3)	0.074
Δ LL	-3.9 (6.2)	**0.008**	-3.2 (6.6)	**<0.001**	-0.11 (6.0)	0.557	-3.9 (6.6)	**0.028**	-5.8 (5.7)	**0.001**	-1.6 (6.6)	0.203	-0.1 (3.5)	0.646
Δ Pelvis	1.6 (2.6)	**0.012**	2.6 (4.9)	**<0.001**	3.3 (5.6)	**0.001**	2.4 (4.1)	**0.005**	2.0 (5.3)	**0.047**	3.1 (2.4)	**0.005**	3.5 (3.8)	**0.013**

Legenda

- Increased thoracic kyphosis
- Decreased lumbar lordosis
- Pelvic anteversion
- Pelvic retroversion
- Increased sagittal vertical axis
- Increased hip flexion
- Increased knee flexion
- Increased ankle dorsiflexion