



Abdominal wall tension after spinal deformity correction compromises postoperative biomechanics and may contribute to proximal junctional kyphosis

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Abstract

Purpose Proximal junctional kyphosis (PJK) is a common complication after fusion surgery for adult spinal deformity (ASD). Tissue adaptation to deformity may cause abdominal wall shortening/stiffening. This study evaluated, using musculoskeletal modeling, the effect of these adaptations and sarcopenia on trunk muscle effort required to maintain postoperative alignment in PJK patients versus controls.

Methods ASD patient data was grouped by mechanical complication status: PJK (N=44), other (N=56), none (N=260). Spinopelvic landmarks were annotated in pre-op, post-op, and follow-up X-ray images. Patient-specific musculoskeletal models of corresponding alignments were built. Forces due to stretching of the abdominal wall beyond pre-op length (assumed slack) were applied, representing abdominal wall stiffness. Sarcopenia was implemented by reducing paraspinal muscle strength based on patient age and gender. Inverse-static simulations predicted overall muscle effort by summing muscle activities.

Results Postoperatively, the abdominal wall was more elongated in the PJK group (+8.4%[-0.3;20.0]) versus the no-complication group (+2.4%[-4.9;10.2], $p < 0.01$) due to larger preoperative deformities and greater surgical correction. This elongation correlated more with pelvic tilt change ($r = -0.53$) than lumbar lordosis correction ($r = 0.16$). Greater muscle effort was estimated for post-op alignment in the PJK group (12.40[6.42;28.6]) versus the no-complication group (8.42[4.34;13.3], $p < 0.05$). Muscle effort was reduced at follow-up in groups with mechanical complications.

Conclusion Alignment restoration tensions abdominal structures, requiring increased extensor muscle forces to maintain postoperative alignment. Patients might develop PJK to reduce unsustainable muscle effort or due to spinal structure failure. More attention should be given to pelvic reciprocal changes to improve surgical planning and perioperative rehabilitation.

Keywords Adult spinal deformity · Proximal junctional kyphosis · Fusion surgery · Musculoskeletal model · Abdominal wall stiffness

Introduction

Multilevel spinal fusion is a common surgical treatment for adult spinal deformity (ASD), a condition expected to affect an increasing number of patients as the population ages [1]. One frequent complication is proximal junctional kyphosis and failure (PJK), with an incidence ranging from 17 to 46% [2]. Patients with PJK often experience heightened symptomatic pain and functional impairment [3–5]. The etiology

of proximal junctional complications is multi-factorial and remains poorly understood. From a biomechanical perspective, increased spinal loads due to sagittal malalignment [6], lower thoracolumbar muscularity and higher fatty degeneration [7, 8] have been speculated to play a role, but what seems to be neglected is that the postoperative spine biomechanics might be compromised by the prior adaptation of tissues to spinal deformity, which can hinder surgical outcomes.

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The deformity that develops and progresses for years before surgery [9] influences the functional length of the connective tissue structures and muscles responsible for stabilizing the spine in an upright standing posture. Animal studies have shown that the muscle bellies adapt to their functional lengths [10–12] and the prolonged muscle shortening leads to connective tissue accumulation [13, 14] and remodeling (fiber re-arrangement) [14] and a reduction in the number of sarcomeres in series [15–17]. These changes result in muscle shortening, loss of muscle mass and strength, and increased passive resistance (stiffening) [13, 17]. In scoliosis, paravertebral muscle stiffness at the apex of the thoracic curve has been reported to be significantly greater on the concave side (where the muscles are shortened) compared to the convex side [18]. While it is not definitely proven that these adaptations result from muscle shortening, it is believed that scoliotic deformities alter the mechanical environment of the paravertebral muscles, leading to adaptive changes [18, 19]. Similar adaptations of muscle shortening and stiffening might affect abdominal wall in patients with sagittal plane deformity. In fact, patients with degenerative kyphoscoliosis have been found to exhibit significantly smaller longitudinal diameters and volumes of the abdominal cavity compared to age-matched controls [20].

The altered properties of the abdominal muscles due to long-term deformity-induced shortening could play a role in the development of PJK, by applying forces resisting the surgically restored alignment and— either alone or exacerbated by age-related weakness (sarcopenia) of the extensor muscles— increasing the muscular effort needed to sustain the postoperative posture. While previous musculoskeletal modelling studies have investigated spinal loads in the context of spinal fusion [6, 21–25] and the general effects of sarcopenia on the muscular effort needed to maintain posture [26], to our knowledge, abdominal muscle adaptation to long-term spinal deformities as a potential factor in the etiology of PJK has not been yet explored.

The aim of this study was to evaluate the effect of adaptation of the abdominal wall passive properties to spinal deformities on the trunk muscle effort required to maintain postoperative alignment in patients who developed PJK during follow-up, compared to controls. A secondary objective of the study was to analyze the individual and combined effects of abdominal muscle adaptation and sarcopenia of spine extensors on the trunk muscle effort, aiming to better understand their relative roles and interaction.

We hypothesized that surgical restoration of sagittal alignment is related to postoperative elongation of abdominal structures in patients who later develop PJK. The resulting tension in the abdominal wall would create unfavorable bending moment on the spine, requiring increased extensor

muscle forces to support the posture. We also hypothesized that postoperative muscle effort is the greatest when both the effects of abdominal tissue adaptations and sarcopenia are considered.

Methods

The effect of adaptation of the abdominal wall passive properties to spinal deformities on the trunk muscle effort required to maintain postoperative alignment were investigated using an established musculoskeletal model of the spine, personalized to represent spinal alignment of 360 patients surgically treated for adult spinal deformity.

Study sample

Radiographic and clinical data of patients with ASD were retrospectively retrieved from a prospective multi-center database. The inclusion criteria for this database were age ≥ 18 years and at least one of the following conditions confirming spinal deformity: coronal Cobb angle $\geq 20^\circ$, sagittal vertical axis ≥ 5 cm, pelvic tilt $\geq 25^\circ$, or thoracic kyphosis $\geq 60^\circ$. The specific inclusion criteria for this study were instrumentation with upper instrumented vertebrae (UIV) $\leq T2$, available sagittal and coronal annotations of standing X-ray images including landmarks from T1 to S1 and femoral heads at three time points (pre-operation, post-operation < 100 days before complication development, and follow-up with the most recent X-ray before potential re-operation).

After exclusions from the 1294 patients with X-ray data in the database (Fig. 1), a final study cohort consisted of 360 patients, grouped by mechanical complication status: PJK ($N=44$), other ($N=56$), or none ($N=260$). The PJK group consisted of patients with two characteristics: (1) a proximal junctional sagittal Cobb angle $\geq 10^\circ$, and (2) at least 10° greater than the preoperative measurement. The “Other mechanical complication” group included rod breakage, distal junctional kyphosis, sagittal malalignment, screw breakage, screw pull-out, screw/rod/hook dislodgement, adjacent segment degeneration, pseudarthrosis, coronal malalignment (> 3 cm), and shoulder imbalance (> 2 cm).

Musculoskeletal modeling

A validated thoracolumbar spine model established in AnyBody Modeling System (AnyBody Technology A/S, Aalborg, Denmark, version 7.4.0) [31–35] was used with few modifications to perform static upright-standing simulations of patient alignments at preoperative, postoperative and follow-up time points. In this model, intervertebral joints were

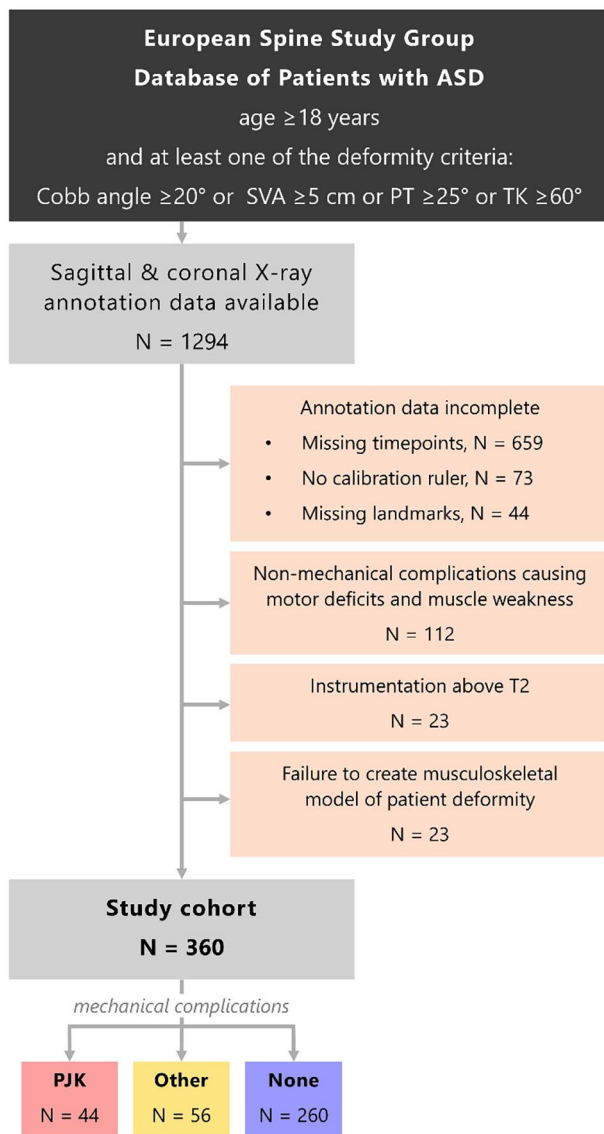


Fig. 1 Flow diagram of patient inclusion and exclusion criteria. (Non-mechanical complications that could cause motor deficits and muscle weakness— one of the exclusion criteria - were e.g., radiculopathy, screw malposition, paraparesis, and dural tear [27–30])

modeled as spherical joints with a traveling center of rotation by implementing reaction forces in translational directions. This allowed relative translation between vertebrae, assuming that the joint transfers forces in these directions, which reflects constraints imposed by stabilizing structures and joint anatomy (such as facet impaction [36]). Assuming neutral positions of intervertebral joints in each posture, segmental stiffness was not included. As in the original model, the ribcage was represented by passive stiffness elements from ex-vivo measurements [37, 38] and an intra-abdominal pressure model was included in the lumbar region [39, 40]. Rotations between ribs and vertebrae were estimated using

the force-dependent kinematics method [41], which allows to compute unknown displacements under given load conditions. The sternum position was delimited by the first rib, with its rotation averaging those of the 10 pairs of non-floating ribs. All major trunk muscles were represented by over 600 fascicles, based on anatomical descriptions (details provided in the Supplementary material 2— Model muscle description). Each fascicle was modeled as a force element transmitting force along the muscle path—either directly for short muscles, or via intermediate nodes to account for muscle wrapping in longer muscles. Tendon properties, force-length, and force-velocity relationships of the muscle function were not considered.

Model individualization with subject-specific inputs

Generic spine model was adjusted to represent relevant patient characteristics. Subject-specific parameters: body weight and height [42] were used to scale the model mass and geometry. Patient-specific alignment was represented by adjusting pelvic parameters as well as vertebral positions and orientations according to the radiographic data, and assuming horizontal gaze. Information about upper and lower instrumented vertebrae were used to model spinal fusion location accordingly. Spinal fusion was modeled by applying external moments compensating for 90% of the gravity-related moment of each fused vertebra to represent non-rigid instrumentation behavior, with assumption of small (10%) contribution of posterior muscles to balancing gravity loads. Muscle strength, i.e. maximum force generating capacity, proportional to muscle PCSA, was scaled using a strength factor non-linearly related to patient body weight [42]. The models individualized in this way were simulated for all patients in 3 configurations: (1) muscle loss modeled based on patient age and gender (Sarcopenia), (2) abdominal wall tension modeled based on the postoperative changes to spinal alignment (Abdominal stiffness), and (3) both (Sarcopenia + Abdominal stiffness).

Sarcopenia

Muscle degeneration modelling was implemented to reflect the deteriorated contractile capacity of the lumbar erector spinae and multifidus in the model based on age, gender and spinal level-specific data [43, 44]. As sarcopenia is manifested by fat infiltration and muscle atrophy, PCSA was reduced depending on %fat infiltration and a %loss in PCSA: $PCSA_{reduced} = PCSA_{normal} * (100\% - Fat[\%]) * (100\% - PCSA_{loss}[\%])$, similarly to previous work by Ignasiak et al. [26].

Estimation of abdominal wall elongation

Abdominal wall length was estimated from kinematic (position) analysis of patient-specific models, due to the challenges of assessing the abdominal landmarks from radiographic images (outside the field of view or difficult to identify). The patient-specific models reflected positions and orientations of individual lumbar and thoracic vertebrae (T1 to L5) derived from endplate coordinates in the sagittal and coronal X-rays of patients in an upright position. To account for uncertainties in X-ray data annotation [45], sagittal plane coordinates were linearly rescaled for each patient across the three time points to match the average endplate length, which varies negligibly over time [46]. Coronal coordinates were similarly rescaled by minimizing their longitudinal axis differences with sagittal data (see Supplementary Material 1 for details). Vertebral centroid (i.e., center of four vertebral endplate landmarks), vertebral angle (i.e., average inclination of inferior and superior endplates relative to the horizontal line), and spinopelvic parameters (i.e., pelvic incidence, sacral slope, pelvic tilt, and pelvic obliquity) in the sagittal and coronal planes were incorporated into the model for the three time points. The distance between the xiphoid process and pubic symphysis in 3D space was found as a measure of abdominal wall length (Fig. 2, left). Its changes in postoperative and follow-up alignment (L_f) from preoperative length (L_0 , assumed slack) were considered as abdominal strain ($\lambda = L_f/L_0$).

Passive muscle adaptations

Forces due to abdominal wall stretching beyond pre-op length were applied in post-op and follow-up models. The constitutive law used to estimate the passive force based on abdominal wall elongation $f(\lambda)$ was derived from Kriener et al. [47], who measured the tensile properties of a composite specimen including the linea alba, rectus abdominis, rectus sheath, and peritoneum (Fig. 2, middle). The stress in MPa was multiplied by the total physiological cross-sectional area (PCSA) of these soft tissues, estimated from laser scanning microscopy and cadaver studies: 20 mm² for the linea alba [48] and 600mm² for the rectus abdominis [49]. The PCSA of the rectus sheath, 80mm² was estimated using the dimensions of the rectus abdominis as well as the thickness of the rectus sheath [48, 50]. For the inverse-static analysis using sagittal model, the estimated tension force produced by the abdominal wall was projected onto the sagittal plane (Fig. 2, right).

Inverse static simulations

Inverse-static simulations were performed to predict muscle activities (exerted muscle force divided by a maximum force it could generate), and the sum of all muscle activities served to estimate overall muscle effort. By solving Newtonian equations of motion with the known gravity moments, the unknown muscle forces are calculated. A polynomial optimization method minimizing the sum of cubed muscle activities (i.e., muscle active state as a fraction of maximum voluntary contraction) as an objective function, was used to solve the problem of muscle recruitment caused by

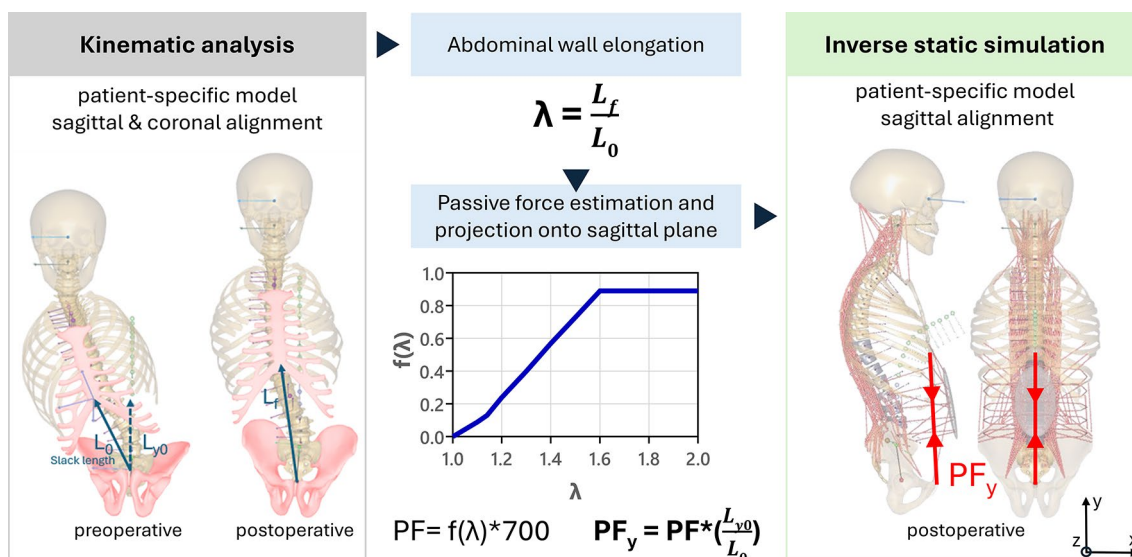


Fig. 2 Calculation of abdominal wall elongation and related passive force applied during inverse-static analysis. λ : strain/abdominal wall elongation, L_f : abdominal wall length in the post-operation or follow-

up posture, L_0 : slack length/abdominal wall length in the pre-operation posture, PF: passive force

muscle redundancy [51] (Supplementary material 2– Muscle recruitment equation). This method mimics physiological muscle recruitment by favoring muscle synergy while reducing energy expenditure [52].

The simulations were performed for all patients across three patient groups: PJK, other mechanical complications, and no complications. Each patient case was simulated with three model conditions: sarcopenia, abdominal wall stiffness, and their combination to predict muscle effort. In this way, the respective roles of abdominal wall adaptation to deformity and age-related muscle degeneration could be discerned.

Statistical analysis

Muscle effort, abdominal wall elongation, and spinopelvic parameters were compared between patient groups using the Kruskal-Wallis test and Dunn's test with Bonferroni correction for non-normally distributed data, and analysis of variance with Tukey's HSD for normally distributed data. Since muscle effort data were not normally distributed, comparisons across postures and modeled conditions (i.e., sarcopenia, abdominal wall stiffness, both) within each patient group were made using the Friedman test and pairwise comparisons [53]. For each test, the *r*-type effect size [54] was calculated: small ($0.1 < r \leq 0.3$), medium ($0.3 < r \leq 0.5$), and large ($r > 0.5$; [55]). Pearson's correlations (*R*) between the amount of correction of spinopelvic parameters and abdominal wall elongation, and Spearman's correlations (ρ) between the elongation and muscle effort were assessed.

Results

Patient sample

A sample of 360 patients with ASD was analyzed, grouped by a mechanical complication status: PJK ($N=44$), other ($N=56$), or none ($N=260$). The two groups with mechanical complications were statistically older and had higher body weight compared to the No Complication group ($p < 0.01$, Table 1). Additionally, their preoperative deformities and the magnitude of postural correction during surgery (changes in spinopelvic parameters) were greater for these two groups compared to the No Complication group for SVA, GT, SS, PT, and LL ($p < 0.05$, Table 1). ~85% of patients had fusion to the pelvis in the two groups with mechanical complications vs. 39% in the No Complication group.

Abdominal wall elongation and relationship with spinopelvic parameters

In the PJK group, the postoperative abdominal wall elongation was found significantly greater compared to the no-complication group (median[Q1;Q3]=+8.4%[-0.3; 20.0] vs. median=+2.4%[-4.9; 10.2]; $r=0.17$, $p < 0.01$; Fig. 3). During follow-up (i.e., 859 ± 596 days, min. 9 weeks, max. 121 months), it was significantly shortened relative to preoperative length in both other mechanical complication and PJK groups compared to the group without complications (median[Q1;Q3] = -3.6%[-15.8; 3.3], $r=0.15$ and median = -5.7%[-14.1; 2.7], $r=0.14$ vs. median = -0.6%[-7.1; 6.2], $p < 0.05$; Fig. 3). All three groups showed a significant shortening of the abdominal wall from postoperative alignment to follow-up, with a smaller effect size observed in the no-complication group ($r=0.32$ vs. $r=0.70$ and $r=0.80$). The change in abdominal wall length was most strongly correlated with Δ PT and Δ GT ($R = -0.53$, Table 2).

Muscle effort

Postoperatively, simulations with abdominal wall stiffness modelled in combination with sarcopenia (Abd+Sarc) predicted greater muscle effort in all three groups compared to the sarcopenia condition alone (*r* ranging from 0.45 to 1.14, $p < 0.001$; Fig. 4). This effect between the Sarc and Abd+Sarc conditions was the greatest in the PJK group.

In the simulated Abd+Sarc condition, greater muscle effort was estimated for post-op alignment in PJK group vs. no-complications group (median[Q1;Q3]=12.40[6.42; 28.6] vs. median=8.42[4.34; 13.3], $p < 0.05$; Fig. 5), but not anymore at follow-up posture. For this condition, both groups with mechanical complications reduced their muscle effort at the follow-up alignment (-4.74 i.e., -42% and -8.14 i.e., -65%; $p < 0.05$), which was not the case for the no-complication group, for whom muscle effort remained statistically similar (Fig. 5). A strong polynomial relationship was found between postoperative muscle effort and the modification of abdominal wall length for the conditions where passive forces were added (Spearman's $\rho=0.65$, $p < 0.001$; Fig. 6).

Discussion

Findings

Our study aimed to evaluate the effect of the abdominal wall adaptation to spinal deformity on trunk muscle effort required to maintain postoperative alignment. We compared estimated abdominal wall elongation and muscle effort at

Table 1 Anthropometric and clinical data of the three investigated groups. PJK: proximal junctional kyphosis, BM: body mass, BH: body height, SVA: sagittal vertical axis, GT: global tilt, PI: pelvic incidence, SS: sacral slope, PT: pelvic tilt, LL: lumbar lordosis, *: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$

	Whole sample	1. PJK	2. Other mechanical complications	3. No complications	<i>p</i> -value (test if groups 1-2-3 are different)
N	362	44	56	260	
N Males	77	6	15	55	
N Females	285	38	41	205	
Age	54.3±18.2	67.9±7.9	61.0±12.8	50.5±19.0	*** 1-3 2-3
BM [kg]	66.3±13.0	69.7±10.4	70.2±13.5	64.9±13.1	** 1-3 2-3
BH [cm]	162.9±8.6	160.2±7.3	162.5±9.7	163.4±8.5	n.s
Surgical details					
UIV	T2-L5	T2-L2	T2-L5	T2-L3	
LIV	T10-Iliac	T10-Iliac	T12-Iliac	T11-Iliac	
Fused to pelvis	186 (51%)	38 (86%)	45 (80%)	103 (39%)	
Radiographic parameters Pre-operation					
SVA [mm]	40.2 61.2	75.3±63.2	67.8±59.5	28.3±57.1	*** 1-3 2-3
GT [°]	29.3 18.7	39.1±16.9	39.1±16.9	25.0±17.6	*** 1-3 2-3
PI [°]	57.9 14.0	62.5±16.0	61.2±16.2	56.4±12.8	n.s
SS [°]	32.7 11.4	28.8±12.0	29.9±10.4	33.9±11.3	** 1-3
PT [°]	25.2 13.1	33.7±11.5	31.3±12.1	22.5±12.5	*** 1-3 2-3
LL [°]	41.6 20.6	33.3±18.8	34.2±16.4	44.6±20.9	*** 1-3 2-3
L4-S1 [°]	30.7 14.8	29.6±15.1	25.9±15.0	31.9±14.5	* 2-3
T2-T12 [°]	41.2 18.6	43.3±18.1	39.3±14.4	41.2±19.5	n.s
T2-T5 [°]	9.7 8.5	10.2±8.0	8.2±7.5	9.9±8.8	n.s
T10-L2 [°]	12.3 19.2	15.7±20.1	11.5±20.3	11.8±18.8	n.s
Change from pre-op to post-op					
ΔSVA [mm]	-31.1±55.0	-54.6±55.0	-39.4±58.0	-25.4±53.3	*** 1-3
ΔGT [°]	-7.4±15.0	-15.2±14.1	-11.9±15.0	-5.2±14.5	*** 1-3 2-3
ΔSS [°]	2.3±9.2	4.5±9.7	4.0±11.5	1.4±8.5	** 2-3
ΔPT [°]	-3.31±10.3	-8.1±10.1	-6.5±10.8	-1.8±9.9	*** 1-3 2-3
ΔLL [°]	9.8±18.8	18.3±18.3	14.2±19.3	7.4±18.2	*** 1-3 2-3
ΔL4-S1 [°]	0.4±14.0	2.1±13.0	2.6±16.2	-0.4±13.6	n.s
ΔT2-T12 [°]	7.9±13.4	12.2±14.3	10.3±11.6	6.7±13.4	* 1-3
ΔT2-T5 [°]	3.2±8.8	3.6±9.2	2.2±6.6	3.3±9.1	n.s
ΔT10-L2 [°]	-3.1±17.5	-2.5±17.6	-2.8±18.5	-3.3±17.4	n.s
X-ray date with respect to surgery date [days]					
Pre-operation	-106±134	-168±174	-98±102	-98±131	* 1-3
Post-operation	51±28	34±28	50±30	54±26	*** 1-2 1-3
Follow-up	859±596	953±628	1070±601	797±579	** 2-3

postoperative and follow-up time-points between patients who develop: PJK vs. other mechanical complications or no mechanical complications. Our results indicate that greater postoperative elongation of abdominal wall (due to deformity correction) together with age-related sarcopenia is related to significantly higher muscular effort in patients who develop PJK. This increased effort is primarily due to abdominal elongation, as indicated by our regression analyses (Fig. 6). Since the passive forces applied in the model are determined by abdominal wall elongation (Fig. 2), they need to be counterbalanced by increased extensors muscle activity, driving the overall muscle effort. Consequently, PJK might be developed as an energy-conserving posture or due to structural failure under increased muscular and segmental loads. While increased muscle activity in the lumbar region has been observed post-fusion in scoliosis patients

[56], no prior studies, to our knowledge, have explored its correlation with surrounding tissue stiffness.

The greater abdominal wall elongation in the PJK and other mechanical complication groups is related to greater preoperative deformities, necessitating more correction during surgery. As a result, the abdominal wall is shorter preoperatively and significantly more stretched postoperatively when alignment is restored, leading to increased muscle effort. These findings are consistent with studies showing that preoperative sagittal malalignment [57, 58] and the magnitude of correction [59–61] are risk factors for PJK and other mechanical complications [62]. Correlations between abdominal elongation and several sagittal alignment parameters found in the current work suggest that careful surgical planning might limit tension on stabilizing tissues and minimize PJK risk.

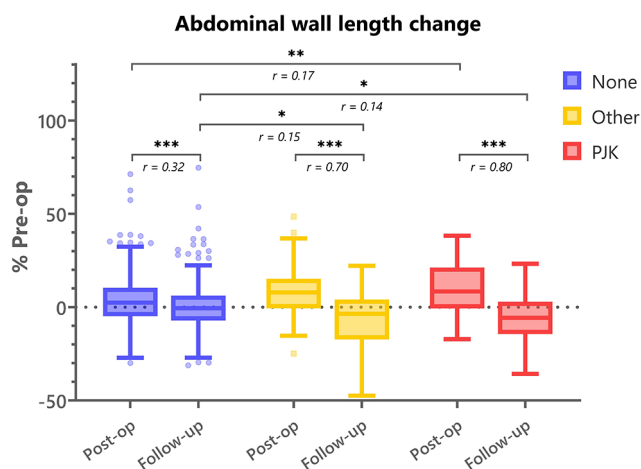


Fig. 3 Change in abdominal wall length (as % of pre-op) in postoperative (post-op) and follow-up alignments. The no complication group is marked in blue, the group with other mechanical complication in yellow and the PJK group in red. PJK: Proximal Junctional Kyphosis, r: r-type effect size, *: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$

Table 2 Correlation coefficients (R) between change in sagittal alignment parameters and change in abdominal wall length, data combined for postoperative and follow-up timepoints and all patients. SVA: sagittal vertical axis, GT: global tilt, PI: pelvic incidence, SS: sacral slope, PT: pelvic tilt, LL: lumbar lordosis, ***: $p < 0.001$

	R
Δ SVA [mm]	-0.33***
Δ GT [°]	-0.53***
Δ PI [°]	-0.26***
Δ SS [°]	0.39***
Δ PT [°]	-0.53***
Δ LL [°]	0.16***
Δ L4-S1 [°]	n.s
Δ T2-T12 [°]	-0.21***
Δ T2-T5 [°]	0.13***
Δ T10-L2 [°]	-0.34***

However, abdominal elongation was only very weakly influenced by lumbar lordosis correction ($R = 0.16$), slightly more by T10-L2 change ($R = 0.34$), and not at all by L4-S1 correction. Thus, the greater postoperative change in LL seen in patients with PJK does not affect abdominal length directly. Instead, it is driven by a reduction in pelvic tilt and global tilt, as they are more strongly correlated with abdominal elongation ($R = -0.53$). This is expected, as the rectus abdominis muscle runs vertically from the pubic crest to the costal cartilages [63]. This is consistent with previous studies showing that PJK patients have a higher PT change ratio (31% vs. 16%) postoperatively compared to no-PJK patients, despite similar global sagittal alignment [64].

While reciprocal change of pelvic compensation may seem clinically favorable by reducing hip flexor effort, our findings indicate that it increases abdominal tension, demanding more effort from trunk extensors. As hip muscles

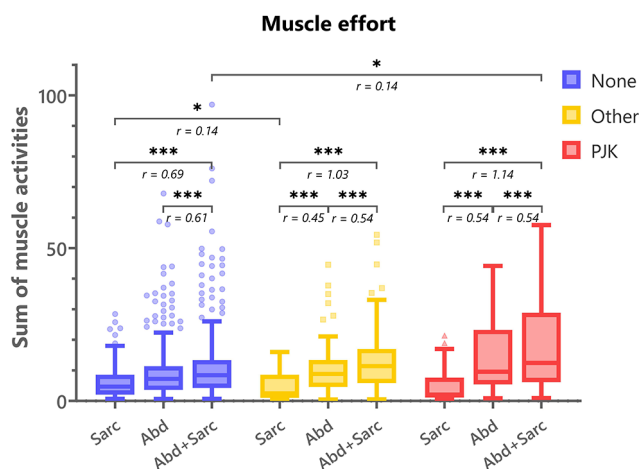


Fig. 4 Postoperative muscle effort across the three simulated conditions: Sarc–sarcopenia, Abd– abdominal wall stiffness, Abd+Sarc – both. The no complication group is marked in blue, the group with other mechanical complication in yellow and the PJK group in red. PJK: Proximal Junctional Kyphosis, r: r-type effect size, *: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$

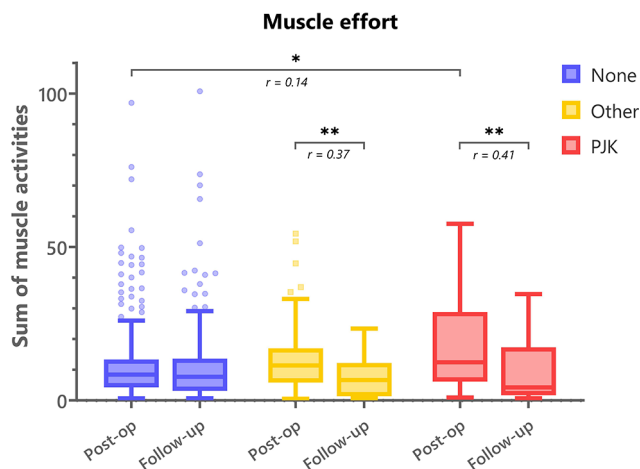


Fig. 5 Change in muscle effort in postoperative (post-op) and follow-up alignments, when both abdominal stiffness and sarcopenia are considered (Abd+Sarc). The no-complication group is marked in blue, the group with other mechanical complication in yellow and the PJK group in red. PJK: Proximal Junctional Kyphosis, r: r-type effect size, *: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$

may have adapted to prolonged pelvic compensation (shortened hamstrings and lengthened, weaker hip flexors), it could still be more ergonomic for patients to return to pelvic retroversion. Considering the majority (87% in our study) has been fused to pelvis, they might then develop PJK to maintain trunk balance over the hips. Indeed, studies show that patients with PJK return to their preoperative pelvic tilt with an increase in PJA [64]. This highlights the need for further research into spine-hip biomechanics, its adaptation to deformity and then disruption by surgical correction.

Simulations of abdominal wall stiffness alone, without modeling age-related muscle degeneration, did not show

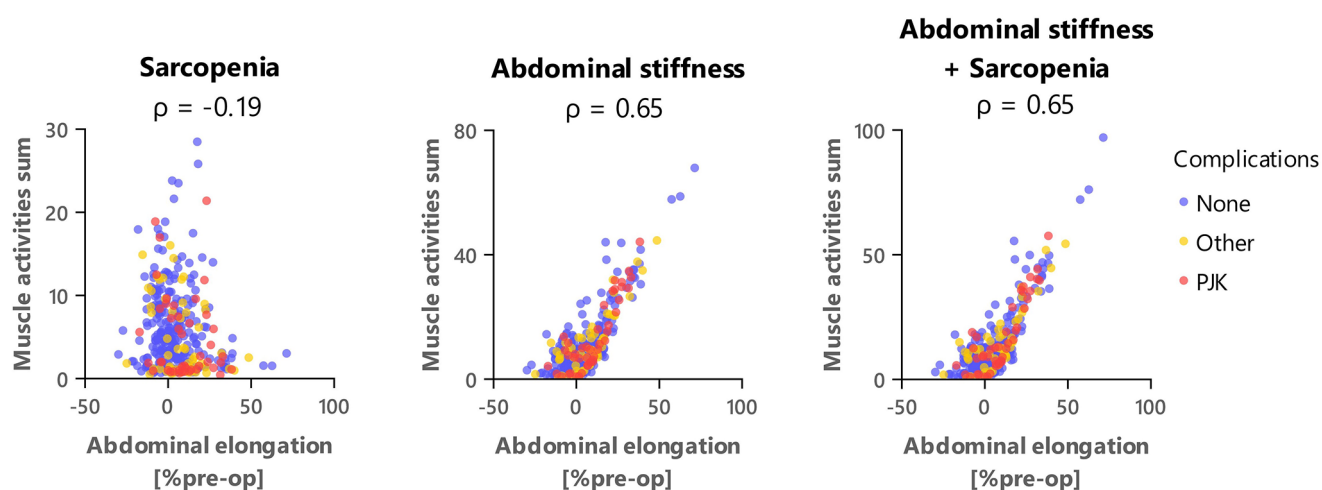


Fig. 6 Spearman's correlation analysis between muscle effort and post-operative abdominal wall elongation for the three tested conditions. The no-complication group is marked in blue, the group with other

mechanical complication in yellow and the PJK group in red. PJK: Proximal Junctional Kyphosis, ρ : Spearman's correlation coefficient. All correlations were significant at $p < 0.001$

differences in postoperative muscle effort between groups, suggesting that stronger muscles might have a preventive effect, and indicating that PJK might result from multiple muscular factors. Psoas CSA [65], fatty infiltration ratio, and CSA of paraspinal muscles at the UIV [66, 67] are in fact significant independent predictors of PJK. Weak trunk extensors are associated with sagittal malalignment in static position [68] and increase mechanical stress on vertebrae due to anterior upper torso shift above the UIV, potentially leading to fractures [66]. During follow-up, the abdominal wall in the PJK group shortened to below the preoperative value, and muscle effort reduced, becoming similar to the no-complication group. This suggests postural collapse of PJK may develop to reduce unsustainable metabolic effort and/or from structural failure, as the abdominal tension on the ribcage could increase compression forces and potentially lead to fractures at the adjacent segment [69]. In the no-complication group, despite a significant reduction in abdominal wall length during follow-up (possibly due to postural adjustments to the new load distribution on the spine), muscle effort did not decrease. At low elongation, tissues are in the “toe” region where stiffness is low due to crimped fibers gradually straightening [70], thus length modifications have minimal effect on muscle effort.

Novelty in the context of previous works

While studies have primarily focused on the active component of muscle force, passive biomechanical properties of skeletal muscles can significantly impact age-related diseases, particularly the balance of the spine in the sagittal plane. Previous musculoskeletal models have incorporated passive muscle forces to study spinal alignment using the Hill-type model [71, 72], which includes passive properties

of the tendon and muscle itself [73–79]. However, the effect of passive muscle properties in the context of spinal fusion and postoperative complications have not been yet investigated. Our model, which represents passive properties of the abdominal wall, as adapted to deformity, showed a substantial impact on spine biomechanics, which can have clinical implications. Clinical studies often focus on muscle elasticity, stiffness, and asymmetry in scoliosis e.g [18, 80]. This study should prompt further investigation into the effects of muscle stiffness and its postoperative evolution in other contexts, particularly complications following spinal fusion.

Additionally, a study demonstrated that following spinal fusion or bracing in scoliosis patients, muscle stiffness changes within six months [80]. Specifically, paraspinal muscle stiffness decreased significantly on both concave and convex sides, with a more pronounced reduction on the concave side (-5.9 kPa, $p=0.025$), likely due to surgical stretching. Passive stretch studies report muscle length growth [81], reductions in muscle stiffness and increased flexibility with daily passive stretching [82–84]. Therefore, while rehabilitation is typically performed postoperatively, our study suggests that prehabilitation involving abdominal wall stretching and spine extensor strengthening could be beneficial. Although this approach is not commonly implemented, it has demonstrated positive effects on patient pain, disability, self-efficacy, psychological behaviors, and satisfaction [85].

Limitations and outlook

The present study had the following limitations:

This study involved a small number of patients in the PJK and other mechanical complication groups, with differing

patient characteristics, including anthropometric features and the aggregated analysis of various fusion constructs. Studies have shown that instrumentation type and the number of fused levels can affect spinopelvic parameters and clinical outcomes [86], as well as muscle effort [3, 87].

Since the abdominal wall was not visible in the radiographs, its length was estimated using a musculoskeletal model, from the rib cage to the pelvis, without further personalization. Its stiffness was then derived from the estimated elongation and data from a mechanical testing study, which may introduce inaccuracies. We assumed that the tissues fully adapt to preoperative deformity, which might be an overestimation. However, only abdominal wall shortening was considered, not the stiffening observed in previous studies on long-term muscle shortening [17, 88]. Thus, our study may have underestimated the stiffness and passive forces linked to muscle fibrosis, already observed in patients with degenerative lumbar disease (i.e., 22% of 171 fiber bundles showing elastic moduli up to 20 times greater than normal [89]). Future studies should measure abdominal wall stiffness and length pre- and post-surgery for more faithful patient modeling.

This study only accounted for the stiffness of the abdominal wall. However, in a static standing position, patients with ASD and high PT exhibit significantly shorter hamstring lengths compared to controls: biceps femoris at 52% vs. 56% of lower limb length, semitendinosus at 61% vs. 64%, and semimembranosus at 52% vs. 55% [90]. Future studies should model the adaptations of other stabilizing structures of the spine and pelvis (e.g., spine extensors and hamstrings) to evaluate their effects.

We modeled sarcopenia only as a function of age and gender. As the severity of sarcopenia can be influenced by other factors, in the future muscle properties should be personalized based on patient imaging data. Additionally, muscle-skeleton interactions were simplified by assuming active force transmission along the muscle path, neglecting more complex factors such as anisotropic passive properties [91] and the force-length relationship [92]. Due to lack of data, body morphology was not individualized in the model, although variations in body shape and tissue composition can influence mass distribution and spine biomechanics [93].

Finally, it needs to be emphasized this study analyzed biomechanical consequences of certain patient factors as one possible mechanism involved in PJK development, which does not elucidate the causes of PJK. The relevance of abdominal wall adaptations for PJK development remains to be investigated, as it is possible that other factors and mechanisms are more important. In the future, the role of abdominal elongation among other known risk factors should be explored, employing a multivariable

logistic regression or a mediation analysis with confounders, respecting many interdependencies between predictors (e.g. age, deformity severity, fusion site, muscle quality), mediating factors (e.g. abdominal tension, loading of adjacent segment), and outcomes. This would allow to untangle complex causal relationships between patient and radiographic factors, biomechanics and complication risk.

In conclusion, differences in abdominal wall elongation following ASD correction were identified between patients who develop PJK and those who did not. Associated differences in muscle effort might affect a patient's ability to maintain postoperative alignment in the long term. Because abdominal wall elongation and muscle effort were estimated from biomechanical analyses of patient inputs, such as alignment, the results suggest a potential mechanism underlying known PJK risk factors, including preoperative deformity severity and the magnitude of correction. However, given the multifactorial nature of PJK as well as modeling limitations, these findings do not imply causality between abdominal muscle adaptation and PJK risk. Future work should further investigate this relationship, with particular focus on pelvic reciprocal changes, as it might have implications for surgical planning and perioperative rehabilitation aimed at preventing PJK.

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Data availability Our clinical data come from a prospective multicenter database and cannot be shared openly in order to protect patient privacy.

Declarations

Competing interests The authors declare no competing interests.

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