Doctoral Thesis

Advancing Clinical Movement Analysis: Spinal Kinematics are Fundamental for Understanding Normal and Pathological Gait

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Advancing Clinical Movement Analysis:
Spinal Kinematics are Fundamental for Understanding
Normal and Pathological Gait

A thesis submitted to attain the degree of

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(Dr. sc. ETH Zurich)

presented by
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2016
Statement of Originality

I hereby confirm that I am the sole author of the written work here enclosed and that I have compiled it in my own words. Parts excepted are corrections of form and content by the supervisors and/or co-authors of the published articles (Chapters 2-6).

Stefan Schmid
I would never have come so far without the support of many people that were involved in this project in one or the other way. First of all, I would like to express my deepest gratitude to my two advisors Dr. Dr. Silvio Lorenzetti and Prof. Dr. Reinald Brunner – you have been tremendous mentors for me. My sincerest appreciation is extended to Prof. Dr. William R. Taylor for being an excellent supervisor over the course of my doctoral studies. Also, I would like to thank Prof. Dr. Ralph Müller for his support in the early stages of the project.

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Abstract

Pathological gait is comprised of primary and secondary mechanisms, whereas primary mechanisms are a direct result of the pathology and secondary mechanisms occur as a consequence of the primary ones. While the distinction between these mechanisms is clear for most orthopedic conditions, this is not the case for pathologies of neurological origin. For this reason, a comprehensive literature review was conducted in the forefield of this thesis, aiming at the identification of secondary mechanisms during gait. The search resulted in 36 studies, whereof 31 studies were categorized as describing active (compensations) and 5 studies passive secondary mechanisms (physical effects). In addition, the analysis revealed several compensatory strategies that were reported for different patient populations and that therefore appeared to be independent from the underlying disease. However, while these studies were reporting on mechanisms in the lower extremities and the trunk, evidence of deviations occurring within the spine appears to be completely lacking. This is most likely due to the fact that spinal movements are difficult to measure and that the commonly used movement analysis systems regard the trunk as a rigid structure that does not allow any statements on spinal motion. A detailed knowledge of spinal motion is yet highly important in order to better understand complex spinal disorders, to prevent complications and to comprehensively evaluate treatment effects. Therefore, by introducing an advanced optical motion capture approach, the overall objective of this thesis was to gain an understanding of normal spinal motion and to investigate pathology-related primary and secondary spinal deviations during gait.

Using the previously introduced and on healthy individuals validated IfB marker model, normal spinal motion (sagittal and frontal plane spinal curvature angles) was investigated by analyzing 42 healthy individuals - categorized as adolescents, adults and elderly - while walking at a self-selected speed. The results implied that lumbar lordosis and thoracic kyphosis increase during the adolescent growth spurt, peak in adulthood and then flatten again with advancing age. Furthermore, this study highlighted the importance of interpreting kinematic measurements always in relation to spatio-temporal gait properties and the fact that investigations involving comparisons of data acquired in different laboratories and by different examiners might be subjected to additional error. The findings can serve as a normative basis for future explorations on pathologies affecting the spine.

Before including subjects with pathology, however, the validity of the curvature angle measurements had to be explored for a structurally deformed spine such as found in patients
with adolescent idiopathic scoliosis (AIS). For this reason, ten patients with main thoracic AIS were examined using standard biplanar radiography with radio-opaque markers placed on the designated spinous processes. The results demonstrated that in the sagittal plane, spinal curvature can be assessed with reasonable accuracy, while in the frontal plane, curvature angles were systematically underestimated due to the distinctive rotational deformities of the scoliotic vertebrae. In order to keep placement error minimal, markers should be applied by healthcare professionals with experience in palpation. Taking the limitations given by the non-invasive nature of the approach into account, the results suggested that the IfB marker model is suitable for the assessment of spinal motion in subjects with spinal deformities.

Therefore, 14 AIS patients and 15 healthy controls were investigated using the same approach as for the above quantification of normal spinal motion. Despite the expected sagittal and frontal plane deformations, patients exhibited altered motion patterns for the thoracic spine in the sagittal and for the lumbar spine in the frontal plane. When using standard trunk motion parameters such as the relative angles between a rigid thorax and pelvis segment, the two groups appeared to be no different from each other. This clearly emphasizes that advanced optical motion capture approaches are required when investigating spinal motion. Furthermore, these first data on the primary effects of AIS on the actual spinal motion might contribute to a better understanding of the pathogenesis of AIS as well as to the prevention of future complications and the improvement of current treatment methods.

One possibility of investigating secondary gait deviations is to assess individuals before and after correction of a known or assumed primary deviation. In adolescents with hemiplegic cerebral palsy (CP), for example, it was assumed that the observed spinal deviations are caused secondarily by foot equinus and leg length discrepancy (LLD) and would resolve spontaneously once lower extremity function was restored. Therefore, a group of 10 adolescents with hemiplegic CP were measured barefoot and with orthotic corrections of foot equinus by an ankle-foot-orthosis and LLD by a heel lift while walking at a self-selected speed. The results showed that although the orthotic corrections were able to restore the first rocker enabling a normal heel-to-toe gait, spinal motion was not influenced thereby. It is hence suggested that spinal gait deviations in adolescents with mild hemiplegic CP are not directly caused by foot equinus or LLD anymore, but probably by more proximal issues such as hip flexor contractures resulting as a long-term effect from foot equinus and LLD during the childhood. These findings provide a deeper understanding of spinal gait deviations in adolescent hemiplegic CP patients, which is a vital component in the prevention of complications in the adulthood as well as for a more effective treatment planning.
This doctoral thesis highlights the importance of using a validated advanced optical motion capture approach allowing the quantification of spinal kinematics for a better understanding of normal and pathological gait. Based on an altered spinal motion pattern in AIS, it has been suggested that segmental instability might play a role in the pathogenesis of AIS as well as in the development of secondary complications such as back pain. Furthermore, an unchanged spinal motion pattern following the correction of lower extremity function in hemiplegic CP patients indicated that the direct causes for the observed spinal deviations might over time have changed from primary problems such as foot deformity and LLD to structurally fixed secondary deviations like hip flexor contractures, which were shown to be involved in the development of back pain in this population.

These findings could serve as a basis for the derivation of non-invasive predictors of curve progression and back pain in AIS. Furthermore, adequate prevention and treatment protocols can be developed in order to address secondary complications in the above patient populations. Finally, this work lays the ground for future investigations on pathological mechanisms of the spine during dynamic everyday living tasks such as walking. In order to gain an even deeper insight into the biomechanics of the spine, the data can be used to drive sophisticated computer models allowing the simulation of segmental motion as well as internal spinal loading.
Zusammenfassung


Um die normalen Wirbelsäulen-Bewegungen (Kurvatur-Winkel in der Sagittal- und Frontalebene) zu erfassen, wurden 42 gesunde Versuchspersonen - kategorisiert als Jugendliche, Erwachsene und Senioren – während dem normalen Gehen gemessen, wobei das zuvor an gesunden Versuchspersonen validierte IfB-Markermodell verwendet wurde. Die Resultate deuteten darauf hin, dass sich die lumbale Lordose sowie die thorakale Kyphose während dem Jugendalter vergrössern, im Erwachsenenalter ihr Maximum erreichen und dann mit zunehmendem Alter wieder abflachen. Weiter zeigte die Studie auf, dass kinematische Messungen der Wirbelsäule immer unter Berücksichtigung der Raum-Zeit Gangparameter interpretiert werden sollten und dass Vergleiche von Daten, welche in verschiedenen
Zusammenfassung


Zusammenfassung


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<tr>
<td>AFO</td>
<td>Ankle-foot-orthosis</td>
</tr>
<tr>
<td>AIS</td>
<td>Adolescent idiopathic scoliosis</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of variance</td>
</tr>
<tr>
<td>AVG</td>
<td>Average</td>
</tr>
<tr>
<td>BMD</td>
<td>Bone mineral density</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
</tr>
<tr>
<td>CoA</td>
<td>Centers of area</td>
</tr>
<tr>
<td>CP</td>
<td>Cerebral palsy</td>
</tr>
<tr>
<td>CS</td>
<td>Cervical segment</td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
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<tr>
<td>d</td>
<td>Effect size for mean difference between two populations</td>
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<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>et al.</td>
<td>and others (&quot;et alii&quot;, Latin)</td>
</tr>
<tr>
<td>f</td>
<td>Effect size when using analyses of variance</td>
</tr>
<tr>
<td>f²</td>
<td>Effect size when using multiple regressions</td>
</tr>
<tr>
<td>Fron.</td>
<td>Frontal</td>
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<tr>
<td>GMFCS</td>
<td>Gross Motor Function Classification System</td>
</tr>
<tr>
<td>hCP</td>
<td>Hemiplegic cerebral palsy</td>
</tr>
<tr>
<td>IC</td>
<td>Initial contact</td>
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<tr>
<td>ICC</td>
<td>Intraclass correlation coefficient</td>
</tr>
<tr>
<td>LLD</td>
<td>Leg length discrepancy</td>
</tr>
<tr>
<td>LS</td>
<td>Lumbar segment</td>
</tr>
<tr>
<td>M</td>
<td>Marker</td>
</tr>
<tr>
<td>MCID</td>
<td>Minimal clinically important differences</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>PF</td>
<td>Plantarflexion</td>
</tr>
<tr>
<td>QA</td>
<td>Quality assessment</td>
</tr>
<tr>
<td>R²</td>
<td>Coefficient of determination</td>
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<tr>
<td>RO</td>
<td>Radio-opaque</td>
</tr>
<tr>
<td>RR</td>
<td>Retro-reflective</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>--------------</td>
<td>---------------------------</td>
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<tr>
<td>Sag.</td>
<td>Sagittal</td>
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<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>SP</td>
<td>Spinous process</td>
</tr>
<tr>
<td>STER</td>
<td>Sternum</td>
</tr>
<tr>
<td>TC</td>
<td>Thoracic curvature</td>
</tr>
<tr>
<td>TLC</td>
<td>Thoracolumbar curvature</td>
</tr>
<tr>
<td>Tran.</td>
<td>Transverse</td>
</tr>
<tr>
<td>TS</td>
<td>Thoracic segment</td>
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<tr>
<td>VB</td>
<td>Vertebral body</td>
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1. Introduction

1.1. Motivation

A possible reason for a pathological gait pattern to occur is a pathology-related process which compromises the normal function of the neuromuscular system. Thereby, deviations from the norm can be of primary or secondary nature, whereas primary deviations are a direct result of a pathology, while secondary deviations occur as a consequence of the primary deviations and resolve spontaneously once the primary deviations are addressed [1]. In addition, secondary mechanisms can be divided into passive and active mechanisms [2]. Passive secondary effects occur as a physical effect of a primary deviation, whereas active secondary effects take place in order to actively compensate for a primary or a passive secondary deviation [2].

In conditions where the primary problem can be clearly identified (e.g. tear of the anterior cruciate ligament), the distinction between primary and secondary mechanisms appears not to be a problem. However, in several pathologies, especially of neurological origin, it is still largely unclear which deviations are of secondary and which of primary nature. Such information is highly relevant for treatment planning in clinical practice, because the treatment of secondary deviations would most likely be inefficient or could even be counterproductive. For example, it has been shown that during gait, the plantarflexors can compensate for reduced knee and hip extensor strength in patients with and without neurological involvement [3-5]. Therefore, since this mechanism might enable a patient to walk independently, treating it can interfere with the ability to walk and might make the patient dependent on assistive walking devices. When addressing the primary deviation, i.e. the knee and hip extensor weakness, on the other hand, the activity of the plantarflexors would most likely normalize spontaneously. In case of a non-reversible pathology, the compensatory mechanism could even be promoted along with strategies in order to prevent secondary complications.

A comprehensive review of the literature (Chapter 2) identified several different secondary mechanisms that seemed not to be dependent on a specific pathology, i.e. not a direct result of pathology. However, none of these studies included an analysis of the spine. Considering that the spine consists of 24 vertebral bodies with the ability of moving in all three dimensions, it seems highly likely that in a loaded condition (i.e. with gravity acting), the spine passively reacts or actively compensates for deviations in the pelvis or the extremity joints, which, over time, could lead to complications. For example, a recent survey reported that 63% of adults
Chapter 1: Introduction

with cerebral palsy (CP) experienced episodes of chronic pain with the most common location being the lower back (71%) [6]. Furthermore, pain has been shown to be coincident with a lower activity level, which was linked to a higher risk for cardiometabolic complications, fragility and/or a higher mortality rate in this population [7]. Peterson et al. [7] thus stated that there is a critical need to identify predictors of secondary pathology and comorbidity in patients with CP.

Considering primary deviations of the spine such as observed in patients with adolescent idiopathic scoliosis (AIS), the literature lacks detailed biomechanical analyses. Even though it has been postulated that differences in spinal movement patterns during gait might be related to the pathogenesis of AIS [8, 9], no studies are available directly following-up on this. In addition, AIS has been associated with low back pain at a prevalence rate of twice as high as in non-scoliotic individuals [10].

For these reasons, a better understanding of spinal kinematics in such populations is of clear importance in order to better understand the pathogenesis of a disease as well as to prevent secondary complications.

1.2. Background

1.2.1. Morphological properties of the spine

The healthy human spine consists of 33-34 bony vertebrae. While the lower 9-10 of these vertebrae (5 sacral and 4-5 coccygeal) are mostly fused together forming a solid base, the upper 24 (5 lumbar, 12 thoracic and 7 cervical) are linked by fibrocartilagenous discs as well as synovial joints (facet joints) and can move in all major planes. In utero and just after birth, the spine is generally C-shaped with its convexity being dorsal (kyphosis). As the neonate starts to assume a more and more upright position (lifting head, sitting up and walk), the cervical and lumbar parts of the spine gradually develop a lordotic shape (ventral convexity), while the thoracic spine retains its kyphosis [11, 12]. With the exception of the atlas and axis (first and second cervical vertebrae), the anterior part of the vertebrae is composed of a cylindrically shaped body consisting of highly porous trabecular bone and covered by two endplates consisting of a thin layer of semiporous subchondral bone with an overlying cartilage layer [13]. The posterior part is made up of the vertebral arch, the superior and inferior articular processes that form the basis for the facet joints as well as the transverse and spinous processes, acting as lever arms for the muscles running over one or several spinal levels [13]. About 75% of axial spinal load are carried by the vertebral bodies, which increase
in size from cranial to caudal due to the load distribution in an upright posture [13]. Together with muscles and tendons, ligaments such as the anterior and posterior longitudinal ligaments provide passive stability and limit the range of movement, which is generally only small within single vertebral segments but considerable over the whole spine [11]. The flexion/extension movement is predominantly carried out by the cervical and lumbar spines, whereas the lateral-flexion movements occur mostly in the thoracic and rotation in the upper cervical and also thoracic spines [12]. These differences in mobility between regions are mostly due to the splinting effect of the rib cage as well as the shape and size of the articular and spinous processes [13].

Due to age or pathology, these morphological properties might be altered. In the aging spine, the mainly affected structures are the vertebral bodies and endplates as well as the intervertebral discs. It has been shown that from young adulthood onwards (20 to 40 years), men and women lose about 6-8% of their trabecular bone density per decade [14]. This loss of bone mineral density (BMD) goes along with decreased bone strength and has further been correlated with an increased risk for vertebral fractures [15]. The typical fracture due to reduced BMD takes place in the anterior part of vertebral body and leads to an increase in spinal kyphosis [13]. In addition, the loss of BMD causes an increase in endplate concavity, which results in a central expansion of the intervertebral disc and subsequently a loss of spinal stature [16]. However, the probably most important factor that influences posture and function of the spine with age is the degeneration of the intervertebral disc. It has been described that the intervertebral discs undergo a transition from a “fluid-like” to a “solid like” behavior with age, causing reduced energy dissipation in the disc that leads to a modified stress-strain environment in the nucleus pulposus and annulus [17]. Furthermore, it has been shown that the elastic properties of the main substance versus the insertion of the anterior longitudinal ligament change and the strength of the bone-ligament junction decreases between 20 and 80 years of age [18]. These structural alterations suggest that the posture and function of the spine might change as well with age. Dreischarf et al. [19], for example, showed an age-related decrease in total lumbar lordosis in asymptomatic elderly subjects, with the flattening occurring mostly in the middle part of the spine and the lower part retaining its lordosis. In addition, the total range of motion (ROM), especially in extension, was found to be reduced by about one third between the oldest and youngest cohorts, with the loss in ROM occurring mostly in the middle part, whereas the ROM next to the thoracic and sacral transitions remained nearly unchanged [19]. These age-related changes in sagittal curvature and ROM were also shown by other investigators [20, 21].
While age-related structural changes can be regarded as physiological adaptations occurring over time, the spine can also be affected by pathology. The most common spinal pathology in children and adolescents is idiopathic scoliosis, characterized by a frontal plane deformation of at least 10° and usually combined with an intrinsic rotational deformity of the vertebrae and a reduced thoracic kyphosis (Figure 1.1) [22, 23].

**Figure 1.1:** Illustration of the three-dimensional deformity of the spine of a patient with AIS using reconstructed CT scans (picture retrieved from Hong et al. [22]).

### 1.2.2. Clinical movement analysis

The term clinical movement analysis refers to the instrumented measurement of movement patterns and the associated interpretation in a clinical context [24]. When concerning the analysis of human gait, however, the term clinical gait analysis is commonly used. Much contemporary gait analysis is conducted for the purpose of clinical research to gain a deeper understanding of a condition affecting a group of patients or the effect of a treatment intervention [24]. It provides an objective record that is able to quantify the magnitude of pathological gait deviations and also to provide an explanation for these abnormalities [25]. Outcome parameters usually include spatio-temporal values (walking speed, cadence, stride length, etc.), kinematics (joint angles), kinetics (joint moments and powers) as well as muscle activation patterns [26]. Since the current thesis will focus on the assessment of kinematic
parameters as well as spatio-temporal values, measurement techniques for the assessment of kinetics and muscle activation are not further elaborated.

The nowadays most widespread technique used for the assessment of joint kinematics is optoelectronic stereophotogrammetry [27]. Thereby, retro-reflective markers are used in combination with specific cameras, detecting the reflection of infrared light that is produced by light-emitting diodes mounted around the lenses of these cameras [27]. With the position and orientation of the cameras known, the three-dimensional position of a marker can be established when the marker is detected by at least two cameras [24]. These markers are placed on anatomical or bony landmarks aiming at the representation of the underlying skeletal structures. Thereby, non-deformable rigid body segments are formed by at least 3 markers per segment, joint axes and segmental coordinate systems are defined and joint angles are calculated by the relative movements of these segments to each other [28].

Common sources of error when measuring with optoelectronic motion capture systems include instrumental errors and soft tissue artifacts [27, 29]. Instrumental errors are usually related to the set-up of the laboratory, including the number and placement of cameras, the size of the measurement volume and the calibration procedure [27]. The most significant source of error causing inaccuracies in human movement analysis, however, is soft tissue artifact. Researchers and clinicians should therefore be well aware of this phenomenon and its effects when interpreting relevant results or in clinical decision-making, respectively [29].

While the speed, accuracy and reliability of these optoelectronic measurement systems advanced considerably since the early 80’s, this cannot be said of the commonly used computer models applied to derive joint kinematics [24]. Most of the commercially available systems still rely on models, using a minimum number of markers possible to determine three-dimensional lower limb kinematics [24]. Furthermore, whereas models and analysis techniques for the lower body have at least certain standards, this is not the case for the upper body. Although the technical advances would allow exploring whole body movements in great detail, an understanding of the physiological mechanisms is lacking. Only few studies reported on pathological gait including a full body analysis [30-43], whereby most of these studies used a standard minimal marker configuration such as the Plug-in Gait full body marker model (Figure 1.2).
1.2.3. Trunk versus spinal motion

Standard marker models such as the Plug-in Gait full body (Figure 1.2) usually refer to spinal motion as the relative motion between a rigid trunk and pelvis segment [44]. However, considering that the spine contains 24 free-moving vertebral bodies, such simplifications are clearly not adequate to analyze spinal motion [45]. The rigid trunk segment approach only allows statements on the general position of the whole trunk in space or in relation to the pelvis segment but not on any movement taking place within the trunk or spine, respectively. Hence, when intending to investigate spinal motion patterns in patients with pathologies affecting the spine, these limited approaches appear not to be useful. For example, research on secondary effects of foot equinus during gait indicated passive deviations in hip and pelvis kinematics [2, 46]. It can therefore be assumed that the spine compensates for these pelvis deviations and that all of these proximal secondary deviations would resolve spontaneously once a normal ankle function is restored. Yet, a recent study addressing this issue did not find any differences in upper body kinematics (measured with the Plug-in Gait full body marker model) following the correction of ankle function in a group of hemiplegic CP patients, suggesting that upper body gait deviations are not caused as a secondary effect of foot equinus in this population [43]. Considering the above mentioned limited possibilities of such marker
models, however, it appears that possible kinematic changes in the spine might just have been missed.

In order to be able to detect spinal motion in adult athletes, a group of researchers at the ETH Zurich’s Institute for Biomechanics developed an enhanced trunk marker model (IfB trunk marker model) (Figure 1.3), which was first used for a preliminary analysis of the kinematics of trunk and spine during unrestricted and restricted squats [47]. By using multiple markers placed over the spinous processes, the researchers were able to quantify the sagittal spinal curvature and showed that the execution of unrestricted squats leads to a smaller ROM of the thoracic curvature and hence to lower stresses in the back [47].

![Figure 1.3: The IfB trunk marker model (picture retrieved from List et al. [47]).](image)

The assessment of sagittal plane curvature angles using the IfB trunk marker set was further validated using MRI scans of healthy subjects assuming three different sitting positions (neutral, flexion and extension) [48]. The results suggested that even though absolute values should be interpreted with caution, the marker set appears well suitable for the measurement of change in lumbar and thoracic spinal curvature angles in healthy adults [48]. To what extent this enhanced method could be used to assess spinal curvature angles in the frontal plane as well as in subjects with pathology, however, remained up to speculation at that time.
1.3. Specific aims

Evidence of spinal motion during activities of daily living such as walking is lacking throughout the scientific literature. One reason for this might be that spinal movements are difficult to measure, especially using non-invasive methods, and that the commercially available movement analysis systems regard the trunk simply as a rigid structure that does not allow any statements of the actual movement of the spine. However, a detailed knowledge of such is highly important in order to better understand the pathogenesis of complex spinal disorders, to prevent secondary complications and to comprehensively evaluate treatment effects. Using an advanced optical motion capture approach, the overall aim of this thesis was to get an understanding of normal spinal motion as well as to investigate the primary and secondary effects of pathologies on the spine during gait. In order to do so, the IfB marker model was adapted for the application in a clinical setting by further developing the curvature algorithm. Subsequently, normal spinal motion was quantified in healthy adolescents, adults and elderly individuals in order to take possible age-related effects into account. Prior to any measurements on patients with spinal pathologies, the marker model was validated for the assessment of spinal curvature in the sagittal and frontal planes using patients with AIS. Finally, spinal gait kinematics were investigated in patients with AIS as well as before and after orthotic correction of lower extremity function in patients with hemiplegic CP.

1.4. Outline of the thesis

This thesis is structured in nine chapters, whereby the chapters 2, 3, 4, 5 and 6 are each based on a peer reviewed original article published or in review for publication in an international scientific journal.

Chapter 1 contains the motivation, the background, the specific aims and the outline of the current thesis.

Chapter 2 provides a comprehensive overview of the available literature on secondary gait deviations. This chapter further identifies the lack of research on primary and secondary mechanisms of the spine during gait and highlights the importance of being able to quantify
spinal kinematics in order to comprehensively evaluate pathological gait patterns in regard to the distinction between primary and secondary deviations.

**Chapter 3** focuses on the spinal kinematics during gait in healthy adolescents, adults and elderly individuals and provides a normative dataset that can serve as a basis for future investigations on spinal pathologies.

**Chapter 4** aims at the validation of the IfB trunk marker set for the measurement of spinal curvature angles in patients with main thoracic AIS using standard biplanar radiography and static motion capture measurements.

**Chapter 5** reports primarily on the spinal curvature angles during gait in a cohort of patients with AIS compared to a group of healthy subjects and in relation to the severity of the deformity.

**Chapter 6** investigates the immediate effects of an orthotic correction of lower extremity function on secondary deviations of the spine during gait in a group of patients with spastic hemiplegic CP compared to group of typically developing adolescents.

**Chapter 7** discusses the main findings of the chapters 2, 3, 4, 5 and 6 and provides an overview of the limitations of the methods used in this thesis.

**Chapter 8** reports on planned or already ongoing follow-up studies that are based on the outcomes of the current thesis.

**Chapter 9** concludes the main findings of this thesis.
2. Secondary Gait Deviations in Patients with and without Neurological Involvement: A Systematic Review

adapted from:

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Permission for reprint granted from Elsevier on Nov 26, 2015 (Appendix E)
2.1. Introduction

Healthy human gait uses repetitive reciprocal limb motions in order to advance the body while simultaneously maintaining stance stability [49]. This is achieved by tightly regulated patterns of muscle activations and generated joint moments and powers. Part of the muscle work is done for acceleration and promotion, part is used to control external moments resulting from gravity and inertia. The appropriate use of these external moments is a major factor in the efficient management of human gait energy. Pathologies that lead to deformities (e.g. joint contractures or bone deformities), muscle weakness, sensory loss, impaired motor control or pain interfere with these tightly regulated patterns and hence, active compensatory strategies might be required in order to maintain proper function.

In addition, as the human body underlies to the laws of general physics, further passive segmental movements can follow from a primary deviation as a consequent physical effect. These passive physical effects, however, are often mistaken for active compensatory strategies. Only recently, researchers started to distinguish between those two kinds of secondary deviations [2, 50]. Brunner et al. [2] for example identified pelvic retraction and hip flexion in children with cerebral palsy (CP) as a passive physical effect of plantarflexor push under load, implying that there was no active compensatory strategy involved as it has been suggested by previous research [51, 52]. Physical effects can be considered passive secondary deviations. They result from gravity acting on the body while moving one or more segments, evoked simply by the anatomical coupling of segments. Hence, every biomechanical constraint following from a primary pathology implicates physical effects. In many cases, however, physical effects cannot be identified since the subjects might actively modify or hide them by exerting compensatory strategies in order to enable locomotion. Further (tertiary) deviations can occur as effects on secondary ones, but can be classified again like the secondary ones.

For all these reasons, gait deviations can be divided in principle into the following categories: 1) primary deviations that are directly due to the pathology; and 2) secondary deviations which split into a) passive secondary effects that follow as a physical effect to the primary deviation; and b) active secondary deviations (i.e. compensatory mechanisms / compensations) that act in order to actively offset primary deviations and secondary physical effects. A similar distinction was adopted by several authors investigating gait adaptations due to foot deformities [1, 46, 53, 54].
Distinguishing secondary from primary gait deviations is critical in clinical practice, e.g. when planning orthopedic interventions or physical therapy treatment. If the causes of gait abnormalities are identified incorrectly, unnecessary and/or ineffective treatment may be carried out [1]. As a consequence, primary abnormalities will be corrected when treated directly whereas secondary problems may resolve spontaneously once the primary issue is addressed [1, 46].

There are several different approaches to identify secondary problems. Most commonly, a pathologic gait pattern is compared to a healthy one and interpreted accordingly. However, this method does neither allow a clear distinction between primary and secondary deviations nor further distinctions between physical effects and compensatory mechanisms. It can only be speculated about the origin of the deviations. Other more reliable methods include comparisons to an additional condition. Stebbins et al. [1] for example assessed twelve children with CP prior to, and following surgery to correct foot deformity, along with a sample of healthy controls. This allowed the investigators to discriminate between the deviations that resolved spontaneously (secondary) and the deviations that persisted (further primary deviations). In another study, Romkes et al. [55] let healthy control subjects mimic the gait of given hemiplegic CP patients. Thereby, the investigators were able to distinguish between primary deviations in muscle activity as a direct consequence of the underlying neurological pathology and deviations due to the biomechanics of toe-walking (i.e. secondary deviations, demonstrated by both the patients and the healthy mimicking subjects). Further methods include in-vivo simulations of primary deviations, e.g. simulated restriction of joint range of motion [46], simulated shortening of the hamstrings [56] or simulated leg length discrepancy [57] as well as computer simulations [58-60].

In-vivo simulations allow researchers to better distinguish between primary and secondary deviations, since the primary pathology is artificially induced. However, in-vivo simulations do not involve subjects with real pathologies and therefore, data should be interpreted cautiously. The dynamic models used in computer simulation studies, on the other side, might be “fed” with real patient data and allow the researchers to make distinctions between primary and secondary deviations as well as between compensatory mechanisms and physical effects. The disadvantage with this method is the input bias, i.e. the models are “fed” with the data that is thought to be required, but necessary parameters such as rules of adaptations at a longer term are not known. In addition, computer simulations are dependent on the quantitative characteristics of human abnormal walking that have not yet been collected enough to be described as dynamic models [61]. Nevertheless, the possibility of modifying one single
parameter such as plantar flexion activity [2] could help decode the complexity of secondary deviations. Simulation studies should therefore be used in order to support the interpretations that are based on the studies involving real patient groups and a control group.

In the literature, deviations in proximal joints following from or compensating for constraints in distal joints and vice versa can be found. Davids et al. [53] and Stebbins et al. [1] for example identified abnormal pelvic transverse plane motion (pelvic rotation) and diminished hip extension during stance as secondary deviations of toe-walking in children with CP. Brunner & Romkes [3] and Matjacic et al. [5] reported plantarflexor hyperactivity during stance phase, which compensated for weak knee extensors in order to provide stance stability in patients with several different orthopedic pathologies.

When investigating gait compensations, it appears that the term “compensation” is usually linked to a specific pathological condition. However, in clinical practice very similar movement patterns can be seen in a variety of underlying disorders, questioning the principle that abnormal muscle activity is the direct result of a neurological disorder [3]. By investigating a group of orthopedic patients suffering from different orthopedic conditions, Brunner & Romkes [3] found two distinctive patterns of compensatory muscle activity, which were independent from the affected joint level, respectively the underlying pathology. They further concluded that these mechanisms corresponded to certain deviations observed in central nervous system (CNS) disorders and that CNS-patients probably do not compensate differently but may be using the same adaptations for muscle weakness as orthopedic patients or any human.

In the literature, the terms "compensation / to compensate" are widely overused and confusion can occur on whether the gait deviation is primary or secondary, respectively passive or active. Thereby, only a sparse amount of studies are concerned with the distinction between physical effects and compensatory mechanisms. Further, it is assumed that there might be general principles of compensation, i.e. compensations that are not directly related to a specific pathology. For treatment planning, a better and more comprehensive knowledge on secondary gait deviations is crucial and therefore, the purpose of the current systematic review was threefold: 1) to identify secondary gait deviations that have been described throughout the literature over the past three decades by means of marker-based three-dimensional gait analysis and involving a control group; 2) to distinguish between physical effects and compensatory mechanisms according to the currently available literature; and 3) to identify common secondary gait deviations that occur across different pathologies and therefore appear to be independent from the underlying disease.
2.2. Methods

2.2.1. Electronic database search

In order to provide a comprehensive overview on gait compensations, an electronic literature search was conducted within the databases MEDLINE, CINAHL, EMBASE, BIOSIS Previews, INSPEC and Journal Citation Reports using the search services Ovid, EbscoHost, EMBASE and ISI Web of Knowledge for the time period of January 1980 to October 2011. The search strategy targeted the categories title, abstract and keywords and included the following search terms: gait, walking, locomotion, compensation, adaptation, deviation, variation, alteration, changes, characteristics, strategy, mechanism, effect, pattern, function, movement, kinematics, motion analysis, gait analysis, motion capture, simulation, model, lower limb, lower extremity, lower body, leg, foot, feet, ankle, knee, hip, pelvis, upper limb, arm, thorax, upper body, upper extremity, joint, human, adult, adolescent, child, elder, patient, subject, woman, man, kid, girl, boy. Wildcard symbols were used to retrieve all possible suffix variations of the root words. The search was not restricted to specific languages.

2.2.2. Inclusion/exclusion criteria and screening

Title and abstract of each study were screened and full texts were retrieved subsequently and evaluated for definitive inclusion if they met the inclusion criteria (Table 2.1). Based on the advantages and disadvantages of the methods commonly applied for the identification of secondary gait deviations and in order to ensure the comparability of the retrieved studies, they were divided in two categories: 1) studies involving a group of subjects with a pathologic gait pattern as well as a group of normal control subjects, quantified by means of a marker-based three-dimensional motion capture system; and 2) studies involving in-vivo simulations of pathologic gait patterns in healthy subjects, computer simulations and single-case studies. The studies in the first category were considered “main outcomes” and were included in the systematic process of this review. The studies in the second category were considered supporting material in regard to the interpretation of the mechanisms. In addition, citation indexes of the included studies were searched in order to identify literature that could have been missed by the electronic database search.
Table 2.1: Inclusion and exclusion criteria for the title and abstract screening as well as for the full text evaluation.

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<tr>
<th>Category</th>
<th>Inclusion</th>
<th>Exclusion</th>
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<td>Type of studies</td>
<td>Original research, published in peer-reviewed scientific journals</td>
<td>Conference abstracts, non peer-reviewed publications, secondary literature or reviews</td>
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<td>Main outcomes</td>
<td>Clearly identified secondary gait deviations; data had to be retrieved from skin-mounted markers by means of at least two-dimensional kinematic data</td>
<td>Single video data without markers</td>
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<td>Subjects*</td>
<td>Human cohorts presenting a pathological gait pattern or a artificially induced restriction and a control group consisting of healthy subjects with group-average ages of greater than 6 years</td>
<td>Parkinson’s disease (due to bradykinesia), Down syndrome (due to complex cognitive impairments) or obesity (due to inaccurate placement of markers)</td>
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<tr>
<td>Measurement</td>
<td>Walking on level ground with a smooth surface and without any obstacles</td>
<td>Treadmill walking, stair climbing, walking up- or downhill, walking on uneven ground or a slippery surface</td>
</tr>
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<td>conditions</td>
<td>Subjects walking freely without any kind of walking aid at either normal (self-selected), fast, slow or default (e.g. paced) gait speed and either barefoot or in normal footwear (e.g. flat-heeled shoes)</td>
<td>Running studies, special footwear (e.g. MBT-shoes)</td>
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<td>Walking</td>
<td>Subjects walking freely without any kind of walking aid at either normal (self-selected), fast, slow or default (e.g. paced) gait speed and either barefoot or in normal footwear (e.g. flat-heeled shoes)</td>
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<td>characteristics</td>
<td>Subjects walking freely without any kind of walking aid at either normal (self-selected), fast, slow or default (e.g. paced) gait speed and either barefoot or in normal footwear (e.g. flat-heeled shoes)</td>
<td>Running studies, special footwear (e.g. MBT-shoes)</td>
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</table>

*except for computer simulations studies

2.2.3. Data extraction and quality assessment

Relevant information of the selected studies in the first category was retrieved using a customized data extraction form.

The quality assessment (QA) of the included articles was performed based on the checklist introduced by Downs & Black [62], which showed to have good inter-rater reliability ($r = 0.75$) as well as high internal consistency (KR-20: 0.89). Since, however, the included articles in the current study did not focus on treatment interventions, the checklist was adapted by discarding items 4, 8 and 19 on description and compliance of interventions as well as their adverse effects, items 9 and 17 on follow-up measures, items 14 and 15 on blinding, items 21 and 22 on different groups of intervention and items 23 and 24 on randomization. In order to ensure quality regarding the reproducibility of measurement procedures, a new item was added to the reporting section (type of devices, resolution, filtering techniques and exact marker and electrode placement are: 2: fully described; 1: partially described; 0: insufficiently described). In addition, item 27 on statistical power of the measurements was reduced from...
the originally six to three options (0: no power analysis done or power < 70%; 1: power = 70-80%; 2: power ≥ 80%). Finally, the QA checklist consisted of 17 items with a maximum score of 20 points, including the five different categories “quality of reporting” (8 items, maximum 10 points), “external validity” (3 items, maximum 3 points), “internal validity - bias” (3 items, maximum 3 points), “internal validity – confounding” (2 items, maximum 2 points) and “statistical power” (1 item, maximum 2 points).

The adapted checklist was cross-validated with four independent reviewers (S.S., K.S., S.L., R.B.) on three of the included articles to ensure reliable data extraction. Subsequently, the checklist was included in the data extraction sheet. All data, including QA, were extracted by two independent reviewers (S.S., K.S.).

2.2.4. Analysis

Percentage agreement and nominal kappa statistics with bootstrapped bias corrected 95% confidence intervals (CI) were used to ensure overall agreement of the two independent raters in the QA [63]. Kappa values were calculated using the command “kap” (STATA, Version 9.2, StataCorp, College Station, Texas, USA) and the user-written STATA-command “kapci” for the bias-corrected bootstrap confidence intervals [64]. Mean values along with standard deviations (SD) were calculated for the summarized scores in each of the QA categories to assess the overall quality of the included studies.

The extracted data were analyzed in a qualitative manner, since the incompatible form of the results did not qualify for a meta-analysis.

2.3. Results

2.3.1. Selection of studies

The electronic database search identified a total of 7805 papers. After removing duplicates, congress proceedings, non peer-reviewed publications, secondary literature and reviews, 4080 studies were included for the title and abstract screening. Following this step, 148 full texts were retrieved and evaluated, whereof 57 articles met all the inclusion criteria (Table 2.1). After dividing the selected publications in the two categories, 35 fell into the first (subjects with pathology and control group) and 22 into the second category (9 in-vivo simulation, 9 computer simulation and 4 single-case studies). The subsequent citation index search for the first category identified another 2 publications, adding up to a new total of 37 studies in this
category. However, one of the already included studies in this category had to be excluded afterwards due to inconsistency of data, explaining the final total of 36 studies included in the qualitative synthesis of the first category.

2.3.2. Data quality

The analysis revealed a percentage agreement of 89.05% and a kappa value of 0.83 (95% CI: 0.78 - 0.87), indicating an “almost perfect” agreement among the two independent raters [65]. The 36 included studies scored in total an average (SD) of 10.9 (2.9) out of 20 points, with a range from 4 to 17 points. The mean score for reporting was 7.0 (1.9) out of 10, for external validity 1.1 (0.9) out of 3, for internal validity bias 1.9 (0.9) out of 3, for internal validity confounding: 0.8 (0.5) out of 2 and for statistical power 0.1 (0.4) out of 2. Scores by each of the two raters for each study and each category are presented in Table 2.2. The numbers of studies that follow are all based on a 100% agreement between the two independent reviewers.

For 15 studies [1, 3, 4, 35, 51, 53, 55, 66-73], respectively 18 studies [3, 4, 35, 52, 53, 55, 69, 71, 73-82], the items reporting on principal confounders (i.e. gender, age, height, body mass) and reproducibility of measurements had to be rated as only “partially described” due to lacking information. In addition, eleven papers [1, 3, 35, 67, 69, 70, 72, 79, 81, 83, 84] did not describe any random estimates of variability such as standard deviation or interquartile range of the main outcome variables and twenty papers [1, 3, 4, 51, 66-70, 72, 74-77, 80, 81, 83, 85-87] did not provide actual probability values.

Regarding the identification of the source population and the proportion of the subjects asked and the subjects that agreed, most of the studies (23 studies [4, 51, 52, 55, 67, 70, 72, 74-76, 78, 80-84, 86-92], respectively 29 studies [1, 3, 4, 51-53, 55, 66-70, 72, 74, 76, 78, 80-92]) had to be rated as “unable to determine”. Another weakly scored item was the one reporting on staff, places and facilities of the measurements. In eleven papers [52, 67, 70, 75, 78, 82, 84-88], it was not identified where the measurements took place and what the profession of the examiner was, even though the item was already scored as 1 when a laboratory was mentioned in the article or in the affiliation.

Finally, only two studies [88, 91] reported on the inclusion of a power analysis (a priori or post hoc) and the respective effective power-values.
### Table 2.2: Results of study quality rated by the reviewers S.S. and K.S.

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<td>7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Sarwahi, V. [72]</td>
<td>6</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Stebbins, J. [11]</td>
<td>7</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Theologis, T. N. [73]</td>
<td>7</td>
<td>7</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Torry, M. R. [91]</td>
<td>10</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Westhoff, B. [82]</td>
<td>9</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Yavuzer, G. [92]</td>
<td>9</td>
<td>9</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>
2.3.3. Methodological data

The extracted data (subject characteristics, methodological data and compensatory mechanisms / secondary deviations) are presented in Table A.1 (Appendix A). The included studies contained a variety of several different neurological and orthopedic. The patient group average ages were between 6.6 years [76] and 65 years [84], with an overall average group age of 26.7 years.

Regarding the measurement conditions, four studies reported that the gait speed of the patients and the control group subjects was intentionally matched [70, 85, 86, 90], eleven studies reported that the patients walked slower than the control group subjects [4, 53, 66, 71, 72, 75, 80, 82, 88, 89, 92] and nine studies reported that there was no difference in gait speed between the patients and the control group subjects [5, 67, 69, 73, 76, 78, 84, 87, 91]. The remaining twelve studies provided no information on walking velocity. Further, 14 studies indicated that the subjects walked barefoot [93], five studies described specific and one study non-specific footwear [70, 84, 85, 87, 88, 91] and the remaining 16 studies provided no information on this subject.

In addition to the kinematic parameters, 31 studies also evaluated kinetic and nine studies electromyographic (EMG) parameters. Only twelve of the 23 studies measuring patients with unilateral pathologies evaluated, apart from the affected side, also the unaffected side. 13 studies provided no clear information on which side was evaluated. Thirty-three studies included the evaluation of the ankle, 34 the knee and 35 the hip joint, 26 the pelvis and eight the trunk. None of the studies evaluated the upper extremities. Overall, two studies were evaluating on two joint levels (e.g. ankle and knee), eleven studies on three levels and 23 studies on four or more levels.

2.3.4. Compensatory mechanisms and physical effects

Based on the definition of Brunner et al.[2] and Gaston et al.[50], five studies were identified describing solely physical effects [51-53, 73, 81] and two studies describing both physical effects and compensatory mechanisms [1, 71], leaving a total of 31 studies including a description of compensatory mechanisms. Thereof, 24 studies (77%) [1, 4, 5, 35, 55, 66, 68-72, 74-76, 78, 80, 82, 83, 86, 88-92] of the 31 studies investigating kinematics, 19 (68%) [4, 5, 67-69, 71, 75, 77-80, 82, 84, 85, 87-91] of the 28 investigating kinetics, and seven (100%) [3, 5, 55, 67, 68, 74, 91] of the seven investigating EMG parameters identified compensatory mechanisms. From the nine studies involving bilateral measurements, six studies (67%)
reported them occurring on both sides [1, 69, 74, 75, 79, 92] and two (22%) only on the affected side [78, 80]. The remaining study [82] did not specify the side of compensation. Overall, eight studies (26%) [70, 74, 83, 84, 87-90] of the 31 selected studies described compensations on only one level, twelve (39%) [1, 3, 35, 55, 67, 72, 75, 76, 78, 79, 85, 91] on two levels and eleven (35%) [4, 5, 66, 68, 69, 71, 77, 80, 82, 86, 92] on three or more levels. From the 27 studies that included the measurement of the ankle, knee and hip joints, nine studies (33%) [1, 3, 5, 55, 66, 68, 69, 79, 86] reported compensations on the ankle, 15 (56%) [3, 4, 55, 66-69, 72, 76, 77, 80, 85, 86, 91, 92] on the knee and 22 (81%) [1, 4, 5, 66-72, 75-80, 85-87, 89, 91, 92] on the hip level. Compensations on the pelvis, respectively trunk level were described in eleven studies (50%) [4, 5, 35, 68, 75, 78, 80, 82, 83, 90, 92] of the 22 studies that included the measurement of the pelvis, respectively in five studies (63%) [5, 35, 77, 82, 84] of the eight including the measurement of the trunk. A summary of compensatory mechanisms in relation to the biomechanical constraints of the primary pathologies as well as the identification of common compensatory mechanisms is presented in Table 2.3. In addition, Table 2.4 provides a list of the identified physical effects.
Table 2.3: Summary of compensatory mechanisms and identification of common compensatory mechanisms that appear to be independent from the underlying pathology (based on the 36 systematically reviewed papers).

<table>
<thead>
<tr>
<th>Biomechanical constraints due to primary pathology</th>
<th>Compensatory mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip extensor weakness</td>
<td>Posterior trunk extension [35](^a)</td>
</tr>
<tr>
<td>Hip abductor weakness</td>
<td>Duchenne limp [35, 77, 78, 82](^{*,a})</td>
</tr>
<tr>
<td>Knee extensor weakness / Quadriceps avoidance</td>
<td>Hip extensors (hamstrings) for knee extensors [3, 67, 74, 87, 91](^{*,a,c})</td>
</tr>
<tr>
<td></td>
<td>Center of mass anterior of knee joint by:</td>
</tr>
<tr>
<td></td>
<td>Increase activity of plantarflexion/knee extension couple [3-5](^{*,a,c})</td>
</tr>
<tr>
<td></td>
<td>Hip flexion [69, 70, 92](^{*a})</td>
</tr>
<tr>
<td></td>
<td>Anterior pelvic tilt [90]</td>
</tr>
<tr>
<td></td>
<td>Knee hyperextension [76]</td>
</tr>
<tr>
<td>Ankle plantarflexor weakness</td>
<td>Eccentric work of hip flexors for progression in stance [85]</td>
</tr>
<tr>
<td></td>
<td>Hip and knee extensors in stance [68, 85](^{*a})</td>
</tr>
<tr>
<td></td>
<td>Hip flexors (pulling) in preswing [68, 75](^{*a})</td>
</tr>
<tr>
<td></td>
<td>Hip extensor torque strategy in late stance (loading of flexor tissue) [88](^a)</td>
</tr>
<tr>
<td></td>
<td>Internal rotation of trunk and pelvis on contralateral side [35]</td>
</tr>
<tr>
<td></td>
<td>Larger symmetrical hip power generation [79]</td>
</tr>
<tr>
<td>General leg weakness</td>
<td>Hyperactivity ankle plantarflexors [3, 5](^{*,a})</td>
</tr>
<tr>
<td></td>
<td>Co-contraction around knee [3]</td>
</tr>
<tr>
<td></td>
<td>Prolonged activity of contralateral hip abductors (weight acceptance) [5]</td>
</tr>
<tr>
<td>Reduced foot clearance</td>
<td>Pelvic up tilt on unaffected side [75]</td>
</tr>
<tr>
<td></td>
<td>Pelvic hike [35, 68, 83, 88, 92](^{*,b})</td>
</tr>
<tr>
<td></td>
<td>Circumduction, hip abduction, hip external rotation [68, 70, 71, 76, 92](^*)</td>
</tr>
<tr>
<td></td>
<td>Hip flexion and/or knee flexion [76, 86](^*)</td>
</tr>
<tr>
<td></td>
<td>Increased plantarflexion on unaffected side (vaulting) [1, 79, 86, 92](^{*,b,c})</td>
</tr>
<tr>
<td>Limited hip extension</td>
<td>Lumbar lordosis [78]</td>
</tr>
<tr>
<td></td>
<td>Knee flexion to allow the pelvis to progress forward [80]</td>
</tr>
<tr>
<td>Loss of lumbar lordosis (Center of mass anterior)</td>
<td>Hip hyperextension [72]</td>
</tr>
<tr>
<td>Rotational knee instability / increased medial knee load</td>
<td>Crouch gait [72](^b)</td>
</tr>
<tr>
<td>Patella &quot;out of line&quot; (Q-angle increased)</td>
<td>Lateral shift of center of mass (e.g. pelvic hike) [67, 84, 90](^{*,b})</td>
</tr>
<tr>
<td>Leg length discrepancy</td>
<td>Reduced hip internal rotation [89]</td>
</tr>
<tr>
<td>Initial toe-contact</td>
<td>Hip and knee flexion and ankle dorsiflexion on unaffected (longer) side [66](^b)</td>
</tr>
<tr>
<td></td>
<td>Early onset plantarflexors, reduced dorsiflexor activity [55]</td>
</tr>
</tbody>
</table>

\(^*\) indicate mechanisms that appear to be independent from the underlying pathology.

\(^a\) indicate mechanisms that were supported by computer simulation studies.

\(^b\) indicate mechanisms that were supported by in-vivo simulation studies.

\(^c\) indicate mechanisms that were supported by single-case studies.
2.4. Discussion

The current systematic review identified 36 papers describing active and passive secondary gait deviations in subjects with different pathologies, measured by means of marker-based three-dimensional gait analysis within the past three decades. In addition, 22 papers were identified as supporting the interpretation of the secondary gait deviations based on in-vivo and computer simulations as well as the analyses of single patient cases.

Since the topic “gait compensations” does not correspond to a clearly defined and delimited area in the field of gait analysis or at least to certain pathologies, we designed our electronic search strategy rather generally. This way, the chances of missing any article related to the topic were minimized. We also did not restrict the search to specific languages, intending to cover all relevant journals in the field.

Ninety-one studies were excluded after the full text scan because they did not involve a control group and comparisons were made to either the contralateral side or only qualitatively to previously published norm data. Other reasons were that the studies were not marker-based, did involve patients in an acute post-surgical state, or did not describe any compensatory mechanisms. A major factor in the exclusion process was attributed to the lack of a uniform terminology defining primary and secondary gait deviations. Gok et al. [94] for example stated that the abnormalities observed in their knee joint angle measurements seemed to reflect mechanical changes secondary to knee osteoarthritis rather than underlying factors involved in the pathogenesis. Considering our definition of primary and secondary changes, this would actually reflect a primary deviation due to the pathology. Such misinterpretations could be prevented in the future with the introduction of a uniform terminology defining primary and secondary gait deviations as provided in this review. Overall, however, no major secondary gait deviations have been missed out by the excluded papers.

2.4.1. Considerations when interpreting the results of the QA

The 36 included clinical studies showed large variations in data quality with scores varying from four to 17. In general, items 5, 5a, 7, 10, 11, 12, 13 and 27 were either poorly scored or rated as “unable to determine” and will therefore be partially discussed further.

In instrumented gait analysis, it is important to take into account factors such as gender, age, body mass and height since they can influence the outcome measures [95-98]. In addition, missing information on biomechanical models, type and sampling rate of measurement devices, locations of body-mounted markers as well as filtering and other data processing
methods impedes proper reproducibility. Lacking control of these factors can have an impact on the interpretation of the results and on the comparability of the parameters between the patient and control groups as well as between the different studies.

Other insufficiently reported items were the identification of the source population, the proportion between the number of recruited and actually participating subjects and information on staff and facilities where the measurements took place. These issues were also revealed and discussed more detailed in a recently published systematic review on gait characteristics of diabetic patients [63].

Finally, the majority of the included studies did not provide any information on whether a power analysis was conducted or not and therefore it is questionable whether the studies had sufficient power to detect a statistically important effect. Since, however, the current systematic review aimed to identify secondary gait deviations in a broad manner, statistical power issues will not be further discussed.

2.4.2. Methodological issues

There were several methodological issues that might have negatively influenced the results and the interpretation of the data throughout the 36 included clinical studies. Approximately one third of all studies reported that the patients walked significantly slower than the control group subjects and another third did not provide any information on gait speed. Several researchers showed that gait speed should be considered as a factor changing the gait pattern [99-104]. Therefore, in order to identify deviations in the kinematic, kinetic or EMG patterns, matching the gait speed of the control group subjects to that of the patients is highly important in order to avoid misinterpretations of deviations that are solely due to gait speed. On the other hand, it has to be taken into account that a reduced gait speed might already be considered a compensatory strategy.

Another factor that is known for having an influence on the gait pattern is footwear [105, 106]. Around half of the studies did not provide any information on the footwear of the subjects, indicating another weak point among the included studies. An exact description of the footwear or, if possible, barefoot walking can therefore only be highly recommended for future studies.

In order to investigate active compensatory mechanisms, it further appears that the kinematic and kinetic measurements should be accompanied by an actual measurement of muscle activity. However, only nine of the included studies involved the measurement of muscle activity by surface EMG. Many of the studies just speculated about the activity of certain
muscles by interpreting the internal joint moments accordingly. This method, however, is not very convincing, given that the internal joint moments are obviously due to the action of different groups of muscles and ligaments. Bulgheroni et al. [67] for example stated that a reduction in anterior tibial displacement can be obtained by a decrease in quadriceps activity (e.g. anterior trunk lean) but also by a co-contraction of the quadriceps and hamstring muscles. By conducting a synchronized analysis of kinetic and EMG variables, they concluded that the global decreasing of the internal knee extension moment was not due to a reduction in extensor activity (quadriceps), but to a more complex neuromuscular mechanism which caused an increase in both extensor and flexor contraction to assure the joint stability [67]. For future studies, it is therefore highly recommended to include EMG measurements of at least the major muscle groups involved in the respective movement.

The often missing evaluation of the unaffected side in patients with unilateral pathologies was regarded as another weak point of the reviewed papers. Considering that the majority of the studies investigating both sides (seven out of twelve) found compensations on the unaffected side [51, 52, 69, 74, 75, 79, 92], the studies which only evaluated the affected side might have missed out on compensatory mechanisms. A complete understanding of the gait deviation appears therefore to be critical.

Besides the 34 clinical studies evaluating three or more joint levels among the lower extremities and the pelvis, only eight included an investigation of the trunk as rigid segments and none of the studies evaluated the upper extremities. This points out a clear lack of knowledge in terms of secondary deviations in the upper body, especially within the spine and the upper extremities. Considering further that the trunk has been suggested to compensate for limited joint range of motion and muscle weakness in the lower extremity [5, 35, 77, 82, 84], future studies should focus on investigating secondary deviations in both lower extremities including the pelvis, throughout the spine and in both upper extremities.

2.4.3. Identification and interpretation of the compensatory mechanisms and physical effects

Besides the usually clear identification of secondary deviations, the distinction between an active compensatory mechanism and a passive physical effect seems to be predominantly lacking throughout the literature. As to that, only two studies could be found describing physical effects, i.e. secondary deviations that do not involve actively regulated neuromuscular processes such as compensatory mechanisms. Brunner et al. [2] showed that in
hemiplegic CP gait, often assumed compensatory mechanisms such as hip internal rotation and pelvic retraction were in fact physical effects resulting from a triceps surae muscle contraction and ankle equinus position under load. Gaston et al. [50] revealed a strong correlation between transverse plane rotation at the foot level and that at the hip and pelvis by investigating the gait in diplegic CP children with plano-valgus deformities. They concluded that femoral rotational abnormalities in these patients were potentially physical effects to the rotational abnormalities at the foot level. Considering these two physical reactions, five studies, that were assumed to describe active compensatory mechanisms, could be identified retrospectively as describing mainly passive physical reactions (Table 2.4). In particular, simulation studies can promote a clear distinction between active and passive secondary deviations. It is therefore possible that some of the compensatory strategies, as described in the following section of the current review, might be identified later on as simple physical reactions.

In total, only 31 out of the 36 included clinical studies were identified describing compensatory strategies, considering that the search was not restricted to specific pathologies, languages or age groups. Overall, the included studies covered a broad area in terms of pathologies and subjects’ ages, making it possible to conclude on which secondary deviations might be universal strategies that are independent from the underlying pathology or from a specific patient group.

Approximately half of the listed compensations (Table 2.3) are mechanisms, compensating for lower extremity muscle weakness or to avoid muscle contraction in the stance phase. One of the most common mechanisms that appeared to be independent from the underlying disease was the Duchenne limp [35, 77, 78, 82]. Thereby, the patients compensated for hip abductor weakness during stance with moving their center of mass towards the affected side by means of a lateral trunk lean. This mechanism was partially supported by the findings of a computer simulation study that has been conducted using a zero moment joint approach [61]. Other mechanisms identified across different patient groups compensated for a weak quadriceps muscle or to avoid quadriceps muscle contraction during stance. They included a hip extensor for knee extensor strategy, i.e. using the two joint characteristics of the hamstring muscles to move the knee towards extension [3, 67, 74, 87, 91], an increased plantarflexion / knee extension couple strategy, i.e. using the triceps surae muscle to move the knee towards extension [3-5] and a hip flexion strategy, i.e. using hip flexion to move the center of mass anterior and thereby to generate an external knee extension moment that moves the knee towards extension [69, 70, 92]. Computer simulation studies conducted by Catalfamo et al.
as well as a single-case experiment conducted by Siegel et al. [108] using induced acceleration analysis further verified the hip extensor (hamstrings) for knee extensor strategy as a compensatory mechanism for weakness of the knee extensor muscles. The plantarflexion / knee extension couple compensatory strategy was supported with computer simulations conducted by Higginson et al. [109] and Goldberg et al. [59] as well as the single-case experiment conducted by Siegel et al. [108]. The hip flexion strategy was further supported by Tagawa et al.’s [61] computer simulation experiment. An additional mechanism to compensate for weak knee extensor muscles was introduced with the single-case experiment by Siegel et al. [108], indicating that a prolonged contralateral plantarflexor activity supported the affected knee into hyperextension in early stance, resulting in a prolonged double limb support.

For the compensation of a reduced foot clearance in the swing phase, the most common strategies described in the clinical studies were: The pelvic hike, i.e. an elevation of the pelvis on the affected side by using the hip abductors on the unaffected side [35, 68, 83, 88, 92], the circumduction, i.e. hip abduction and hip external rotation, or isolated hip abduction or hip external rotation on the affected side [68, 70, 71, 76, 92] and vaulting, i.e. an increased ankle plantarflexion during the stance phase on the unaffected side [1, 79, 86, 92]. The pelvic hike as well as the vaulting strategies further corresponded to the results of two in-vivo simulation studies conducted by Nuzzo et al. [110] and Kerrigan et al. [111] investigating compensatory strategies based on an artificially induced unilateral knee immobilization. A single-case experiment involving a traumatic brain injury patient additionally identified the vaulting strategy as a compensatory mechanism [112].

Several further computer simulation, in-vivo simulation and single-case studies were found, supporting the compensatory mechanisms identified by the 36 in the current systematic review included clinical studies. Computer simulation studies provided additional evidence for the following compensatory mechanisms: Hip and knee extensors compensating for weak plantarflexors in stance [59, 113]; hyperactivity of the ankle plantarflexors compensating for general leg weakness in stance [59]; hip flexors compensating for weak plantarflexors in preswing by promoting the advancement of the limb [59]; and hip extensor torque strategy in late stance (i.e. loading the flexor tissue) compensating for plantarflexor weakness [60]. In addition, studies conducted on the basis of in-vivo simulations provided supporting evidence for the following compensations: Hip and knee flexion and ankle dorsiflexion compensating for leg length discrepancy [56]; crouch gait compensating for a loss of the lumbar lordosis [101]; a lateral shift of the center of mass compensating for an increased medial knee load.
[114]; and knee flexion on the longer leg side compensating for leg length discrepancy [110].

A single-case study conducted by Lee et al. [115] provided supporting evidence for the posterior trunk extension as a compensatory mechanism for hip extensor weakness.

In contrast to the common compensatory mechanisms that appear to be independent from the underlying disease, the results clearly indicate that there are also different compensatory strategies for the same biomechanical constraints. For example, a reduced foot clearance in the swing phase could be compensated by either a pelvic up tilt on the unaffected side, a pelvic hike, circumduction or vaulting. In addition, Siegel et al. [108, 116] provided evidence that weakness of both hip and knee muscles was compensated by several different strategies. Considering this, future studies should be more attentive when averaging their data over all subjects, since this step could eventually mask out compensatory mechanisms.

### Table 2.4: Summary of the identified passive physical effects that resulted from gravity during locomotion (based on the 36 systematically reviewed papers).

<table>
<thead>
<tr>
<th>Biomechanical constraints due to primary pathology</th>
<th>Physical effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equinus position of ankle (toewalking gait)</td>
<td>Anterior pelvic tilt (hip flexion), hip internal rotation and external pelvic rotation (pelvic retraction) [1, 51-53]</td>
</tr>
<tr>
<td>Internal rotation deformity of foot (intoeing gait)</td>
<td>Hip external rotation [71, 73, 81]</td>
</tr>
</tbody>
</table>

### 2.5. Conclusions

#### 2.5.1. General conclusions

Despite the relatively unrestricted search algorithm, the amount of identified studies describing secondary deviations matching our definition was astonishingly small. Considering further that in the early 90’s, Perry and Burnfield [49] already described many of the compensations we found within the last three decades’ scientific literature, one might get the impression that this topic is almost entirely exploited. However, given the introduction of general principles of compensatory mechanisms and the fact that certain presumed “compensations” were identified as simple passive physical effects [2, 50], we are convinced of the contrary. Compensatory mechanisms have to be further investigated.
Most importantly, thereby, attention has to be paid to a uniform terminology. In literature, the term “compensation” is often inappropriately used. For the sense of clarity, it is therefore suggested to uniformly adopt the following classification for gait deviations: 1) primary deviations that are directly due to the pathology; and 2) secondary deviations which split into a) passive secondary effects that follow as a physical effect to the primary deviation; and b) active secondary deviations (i.e. compensatory mechanisms / compensations) that act in order to actively offset primary deviations and secondary physical effects. A strict implementation of these terms will prevent from misinterpretations in future studies.

2.5.2. Clinical implications

Treatment planning should include a careful evaluation of the pathologic gait pattern by means of computerized three-dimensional gait analysis (including surface EMG) with special attention to possible compensatory mechanisms or passive physical effects. Respecting this information may avoid unnecessary treatment of functional deformities. Distinguishing between compensatory mechanisms and physical effects appears not to be the highest priority for surgical treatments, since both are secondary to the primary deviation and would resolve spontaneously once the primary deviation is treated successfully [1, 46]. For the planning of a physical therapy treatment, on the other hand, this distinction can be of higher importance, since compensations are active neuromuscular processes and might therefore be controlled by voluntary actions, whereas physical effects are given based on the laws of physics and might therefore not be corrected.

In case of non-curable damage of the neuromuscular system, proper compensatory strategies could even be instructed to patients in order to promote locomotion. Thereby, regular physical therapy consultations and preventive treatment methods (e.g. specific exercise therapy) could prevent from further deteriorations such as the degeneration of cartilage tissue due to misuse of the joints (osteoarthritis).

2.5.3. Research implications

The sparse amount of available evidence addressing the identification of compensatory mechanisms and physical effects during pathologic gait as well as the partially rather low methodological quality implicate that more research has to be conducted in this area. Especially in respect of the distinction between compensations and physical effects by means of computer simulation studies, more data have to be collected in order to be able to
sufficiently “feed” the dynamic models with the required quantitative characteristics of abnormal human walking. Thereby, researchers should, if applicable, always include the measurement of muscle activity by means of surface EMG, considering that compensatory mechanisms are active neuromuscular processes that can only be evaluated directly by measuring muscular activity. Further, enhanced research is needed on evaluating a full-body marker set, including, if applicable, a multi-segmental trunk model in order to capture possible compensations within the spine. The unaffected side should thereby always be included in the evaluation of patients with a unilateral pathology, to ensure that secondary deviations are understood in a more comprehensive context. Finally, researchers should focus on a higher methodological quality by better controlling factors such as gait velocity and footwear.
3. Spinal Kinematics during Gait in Healthy Individuals across Different Age Groups

adapted from:

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3.1. Introduction

In human locomotion, the importance of the upper body has been emphasized for over forty years [117]. Saunders et al. [117] identified six major determinants of gait, of which three have been related to the motion of the pelvis. Furthermore, they highlighted the motion of the pelvis and trunk to be essential determinants of bipedal gait. The involvement of the upper body in gait stability as well as a variety of changes in motion patterns due to aging and disease were further pointed out by McGibbon et al. [118]. In contemporary biomechanical research as well as in clinics, gait kinematics are usually measured using skin marker-based motion capture systems. Unfortunately, only few marker-based motion capture studies are available that provide normative data on within-trunk or spinal kinematics [45, 119-125], leaving us with a limited understanding of the biomechanical behavior of the healthy spine during gait (for a short overview of these studies, please refer to Table B.1 (Appendix B)).

In addition, it has to be considered that most of these studies were solely conducted on young adults and using either rigid trunk segments or very simple 2D projection angles. A major disadvantage of these methods is that they allow only a limited assessment of the actual curvature of the spine. To address this deficit, an enhanced trunk marker set (IfB marker set) was previously introduced and validated for the assessment of sagittal spinal curvature in healthy subjects as well as sagittal and frontal spinal curvature in patients with adolescent idiopathic scoliosis (AIS) [47, 48, 126]. The importance of measuring spinal curvature angles in addition to general trunk kinematics was further emphasized by Schmid et al. [127], showing clear differences in spinal curvature angles between healthy adolescents and patients with AIS using these techniques, but not in angles based on commonly used rigid trunk segments.

Walking speed is known to have an influence on pelvis and head movements in healthy adults, whereby increased walking speed corresponds to an increase in the magnitude and variability of the acceleration of these segments [124]. It has therefore been proposed that an individual’s comfortable walking speed is selected in order to minimize the level of acceleration variability as well as to ensure smooth and rhythmic pelvis and head movements [124]. When looking at differently aged populations, this comfortable walking speed was reported to decline with increasing age [128-130] suggesting that elderly individuals might present kinematic changes due to a decreased walking speed. However, walking speed seems not to be the only factor responsible for altered trunk kinematics with advanced age, since elderly subjects have been shown to exhibit different head and trunk motion patterns.
compared to younger subjects even when walking at equal speeds [121]. In addition, Van Emmerik et al. [125] reported altered movement amplitudes of the pelvis and trunk segments between young, adult and elderly individuals that were not related to walking speed. These non-walking speed-related changes might be explained by age-related morphological changes that lead to the previously observed decreases in maximal sagittal range of motion of the lumbar, thoracic and cervical spine in elderly individuals [21]. Although normal trunk motion during gait was described to be small (i.e. less than 5 degrees range of motion) [45], a reduced maximal range of motion might still have an influence on the neuromuscular control of the respective joints.

In order to be able to comprehensively investigate pathologies that directly or indirectly affect the spine, knowledge of the biomechanics of a healthy spine during gait with respect to age is necessary. Using a previously validated enhanced non-invasive optical approach, the primary aim of the current study was therefore to assess sagittal and frontal spinal curvature angles during gait in healthy adolescents, adults and elderly individuals and to provide a normative dataset as a basis for future investigations involving pathologies. In addition, spatio-temporal gait parameters and absolute and relative angles of a rigid pelvis, lumbar, thoracic and cervical segment were calculated in order to support the interpretation of the primary outcomes.

3.2. Methods

3.2.1. Participants

Forty-two healthy individuals, divided into the categories adolescents (10-18 years), adults (19-35 years) and elderly (≥ 65 years), participated in the current study (Table 3.1). Subjects were included if they were in good overall health, considered of normal weight (no overweight or obesity), presented no history of spine surgery and did not suffer from back problems that required medical consultation or treatment in the previous 6 months. The study protocol was approved by the responsible ethics committees and all subjects (as well as the legal guardians for the adolescents) provided written informed consent to participate in this study.
Table 3.1: Demographics of the healthy adolescents, adults and elderly groups expressed in mean and standard deviation (SD).

<table>
<thead>
<tr>
<th></th>
<th>Adolescents (n = 14)</th>
<th>Adults (n = 13)</th>
<th>Elderly (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years]</td>
<td>13.9 SD 1.5</td>
<td>27.0 SD 2.5</td>
<td>69.7 SD 1.8</td>
</tr>
<tr>
<td>Height [m]</td>
<td>1.61 SD 0.10</td>
<td>1.74 SD 0.06</td>
<td>1.70 SD 0.05</td>
</tr>
<tr>
<td>Mass [kg]</td>
<td>53.8 SD 10.2</td>
<td>68.6 SD 8.4</td>
<td>66.8 SD 6.8</td>
</tr>
<tr>
<td>Gender [male/female]</td>
<td>7/7</td>
<td>6/7</td>
<td>6/9</td>
</tr>
</tbody>
</table>

3.2.2. Data collection

Measurements of the adolescents were conducted in the gait analysis laboratory of the University of Basel Children’s Hospital using a 12-camera motion analysis system (type MXT20, Vicon, Oxford, UK; sampling frequency: 200 Hz), whereas the adults and elderly individuals were assessed in the movement analysis laboratory of the ETH Zurich’s Institute for Biomechanics using a 12-camera motion analysis system (type MXV612, Vicon, Oxford, UK; sampling frequency: 100 Hz). After the assessment of relevant anthropometric data, subjects were equipped with retro-reflective markers according to the configuration of the IfB trunk marker set [47] (Figure 3.1) in combination with the Plug-in Gait full body marker set [42] (adolescents) and the IfB full body marker set [47] (adults and elderly). Subsequently, all subjects were measured wearing their own comfortable shoes in a standing upright position for 2 seconds and during walking at self-selected speed on a 10 m level ground walkway until at least 4 valid trials were recorded.

3.2.3. Data reduction

The software Nexus (version 1.8.5, Vicon, Oxford, UK) was used for defining the gait events (i.e. initial contact and toe-off). Kinematic data were evaluated using a custom-built MATLAB-Routine (version R2012a, MathWorks Inc., Natick, MA, USA). Spinal curvature angles in the sagittal and frontal planes were calculated based on the circular segments established by the markers on the spinous processes of the vertebrae T11, L1, L2, L3, L4 and L5 (lumbar curvature) and T3, T5, T7, T9 and T11 (thoracic curvature). Considering the rotation of the thorax during gait, a dynamic coordinate system was used for the thoracic curvatures in order to minimize possible projection error. For the lumbar curvatures, however, the global coordinate system was used due to a missing meaningful anterior reference point.
Chapter 3: Spinal Kinematics in Healthy Individuals

Figure 3.1: Marker placement according to the IfB trunk model in combination with the Plug-in Gait full body model for the assessment of spinal curvature angles and segmental trunk kinematics. White circles with black filling represent markers that were used for both models, while black circles with white filling represent markers that were only used for the IfB model, and markers with black circles were only used for the Plug-in Gait full body model.

These procedures have been previously described and successfully implemented for the evaluation of spinal curvature angles during gait in patients with adolescent idiopathic scoliosis [127] and hemiplegic cerebral palsy [131].

In order to analyze overall trunk motion, rigid pelvis, lumbar, thoracic and cervical segments were defined (Figure 3.1). The three-dimensional position and orientation of these segments during gait was then calculated in relation to the standing measurement trial (absolute segmental angles) as well as between the adjacent segments (relative segmental angles) as described elsewhere [127].

The individual curvature and segmental angle curves were time normalized to a left gait cycle consisting of 101 points and averaged over the four trials per subject. The primary outcome parameters of this study were defined as the average and range of motion (ROM) values of
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the spinal curvature angles over one full gait cycle (expressed in degrees), whereas the spatio-temporal gait parameters (walking speed, cadence, stride length and stride time; all expressed as dimensionless numbers [132]) as well as the ROM values of the absolute and relative segmental angles over one full gait cycle (expressed in degrees [°]) served as secondary outcome parameters.

3.2.4. Statistical analyses

Statistical calculations were performed using the software packages SPSS 22 (SPSS Inc., Chicago, IL, USA) and G*Power 3.1.9.2 [133]. To verify normal distribution of the outcome parameters, the Shapiro-Wilk test was used (normally distributed: p>0.05). Comparisons for several outcome parameters between the adolescents, adults and elderly groups were carried out using one-way analyses of variance (ANOVA) with Tukey HSD post-hoc tests. Due to the explorative character of the study, however, the interpretation of the results was mainly based on the magnitude of the effects (Cohen’s f and d, respectively) rather than the p-values. Thereby, pairwise comparisons were carried out when the ANOVA yielded considerable effects (f≥0.1). Parameters were considered different between the groups when the respective pairwise comparisons showed large effect sizes (d≥0.8).

3.3. Results

3.3.1. Primary outcome parameters

Adults presented considerably higher average thoracic kyphosis and lumbar lordosis values than the adolescents (Figure 3.2) (Table 3.2). In the frontal plane, group differences for the average values were found between the adolescents and the adults in the lumbar spine and between the adolescents and both the adults and the elderly in the thoracic spine. The ROM of the lumbar frontal curvature angles were smaller in the adolescents as compared to both the adults and elderly. Figure 3.3 shows the lumbar and thoracic curvature angles over one gait cycle for illustrative purposes.
Figure 3.2: Bar graphs representing the primary outcome parameters (expressed in degrees) for adolescents, adults and elderly individuals. The asterisks indicate (*) a large effect with statistical significance (p≤0.05) and (**) a large effect without statistical significance.
### Figure 3.3: Curvature angles of the lumbar and thoracic curves in the sagittal and frontal planes over one complete gait cycle of the subjects’ left leg. The grey solid lines represent the adolescents, whereas the black solid and dashed lines represent the adults and the elderly, respectively (thick lines = means, thin lines = standard deviations).

#### 3.3.2. Secondary outcome parameters

Elderly individuals walked considerably faster than the adults, who in turn walked considerably faster than the adolescents (Table 3.2). In addition, the elderly displayed higher cadence and stride length as well as lower stride time than the adolescents.

Considering the absolute segmental angles (Table 3.2), the adolescents exhibited considerably lower ROM values for the lumbar segment in the frontal plane compared to the elderly individuals and for the thoracic segment in the transverse plane compared to the adults. Furthermore, the adults showed higher ROM of the cervical segment in the transverse plane compared to the elderly individuals. The relative angles between the thoracic and lumbar segments indicated higher sagittal and frontal plane ROM in adults and elderly individuals than adolescents (Table 3.2). Furthermore, adolescents showed higher transverse plane ROM between the cervical and thoracic segments than elderly individuals.
Table 3.2: Reported are mean, standard deviation (SD) and 95% confidence intervals (within the brackets) for the average (AVG) and range of motion (ROM) values of the spinal curvature angles and trunk segmental angles in the sagittal (Sag.), frontal (Fron.) and transverse (Tran.) planes over one gait cycle (all angles expressed in degrees [°]) as well as the spatio-temporal gait parameters (expressed as dimensionless numbers) in healthy adolescents, adults and elderly individuals. In addition, results for the group comparisons (one-way analyses of variance (ANOVA) and Tukey’s post hoc tests as well as effect sizes (Cohen’s f and d, respectively) are presented.

<table>
<thead>
<tr>
<th>Segment</th>
<th>Thoracic Curves</th>
<th>Lumbar Curves</th>
<th>Pelvis Segment</th>
<th>Thoracic Segment</th>
<th>Cervical Segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sag.</td>
<td>AVG 34.7 SD 8.3 (29.9, 39.5)</td>
<td>AVG -24.9 SD 11.7 (31.7, -18.2)</td>
<td>AVG 3.8 SD 0.9 (3.3, 4.3)</td>
<td>AVG 3.8 SD 0.9 (3.3, 4.3)</td>
<td>AVG 3.6 SD 1.1 (29.4, 42.2)</td>
</tr>
<tr>
<td>Fron.</td>
<td>3.3 SD 2.2 (2.1, 4.6)</td>
<td>6.1 SD 1.3 (5.4, 6.9)</td>
<td>7.1 SD 2.8 (5.4, 8.7)</td>
<td>4.8 SD 2.3 (3.5, 6.1)</td>
<td>3.6 SD 1.1 (29.4, 42.2)</td>
</tr>
<tr>
<td>ROM</td>
<td>4.4 SD 3.5 (2.3, 6.4)</td>
<td>5.8 SD 2.3 (4.5, 7.2)</td>
<td>7.1 SD 2.8 (5.5, 8.8)</td>
<td>6.2 SD 2.6 (4.8, 7.7)</td>
<td>7.1 SD 2.8 (5.4, 8.7)</td>
</tr>
<tr>
<td>AVG</td>
<td>AVG 34.7 SD 8.3 (29.9, 39.5)</td>
<td>AVG -24.9 SD 11.7 (31.7, -18.2)</td>
<td>AVG 3.8 SD 0.9 (3.3, 4.3)</td>
<td>AVG 3.8 SD 0.9 (3.3, 4.3)</td>
<td>AVG 3.6 SD 1.1 (29.4, 42.2)</td>
</tr>
<tr>
<td>Fron.</td>
<td>3.3 SD 1.0 (2.7, 3.9)</td>
<td>5.8 SD 2.3 (4.5, 7.2)</td>
<td>7.1 SD 2.8 (5.5, 8.8)</td>
<td>6.2 SD 2.6 (4.8, 7.7)</td>
<td>7.1 SD 2.8 (5.4, 8.7)</td>
</tr>
<tr>
<td>ROM</td>
<td>0.5 SD 3.7 (-1.7, 2.8)</td>
<td>0.6 SD 3.8 (-1.5, 2.7)</td>
<td>7.9 SD 2.2 (6.6, 9.2)</td>
<td>8.8 SD 3.6 (6.8, 10.8)</td>
<td>8.6 SD 3.2 (6.7, 10.4)</td>
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<tr>
<td>Sag.</td>
<td>AVG 39.9 SD 12.3 (31.4, 46.8)</td>
<td>AVG -28.8 SD 12.1 (-35.7, -22.0)</td>
<td>AVG 4.2 SD 2.3 (2.9, 3.4)</td>
<td>AVG 5.0 SD 1.2 (4.3, 5.7)</td>
<td>AVG 3.6 SD 1.1 (29.4, 42.2)</td>
</tr>
<tr>
<td>Fron.</td>
<td>3.4 SD 1.3 (2.7, 4.2)</td>
<td>7.4 SD 2.5 (6.0, 8.8)</td>
<td>6.2 SD 2.6 (4.8, 7.7)</td>
<td>6.2 SD 2.6 (4.8, 7.7)</td>
<td>7.1 SD 2.8 (5.4, 8.7)</td>
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<tr>
<td>ROM</td>
<td>0.6 SD 3.8 (-1.5, 2.7)</td>
<td>0.7 SD 1.9 (-1.3, 0.3)</td>
<td>3.8 SD 0.9 (2.8, 3.9)</td>
<td>5.0 SD 1.2 (4.3, 5.7)</td>
<td>7.1 SD 2.8 (5.4, 8.7)</td>
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<tr>
<td>Sag.</td>
<td>AVG 0.41</td>
<td>AVG 0.38</td>
<td>AVG 0.32</td>
<td>AVG 0.38</td>
<td>AVG 0.32</td>
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<tr>
<td>Fron.</td>
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<td>0.368</td>
<td>0.32</td>
<td>0.38</td>
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<tr>
<td>ROM</td>
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<td>0.040</td>
<td>0.32</td>
<td>0.32</td>
<td>0.26</td>
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<tr>
<td>Sag.</td>
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<td>0.49</td>
<td>0.32</td>
<td>0.38</td>
<td>0.26</td>
</tr>
<tr>
<td>Fron.</td>
<td>0.000</td>
<td>0.000</td>
<td>0.26</td>
<td>0.26</td>
<td>0.26</td>
</tr>
<tr>
<td>ROM</td>
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<td>0.000</td>
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Continued on next page
Table 3.2: continued

<table>
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<tr>
<th></th>
<th>1) Adolescents (n=14)</th>
<th>2) Adults (n=13)</th>
<th>3) Elderly (n=15)</th>
<th>ANOVA</th>
<th>Post hoc (Tukey HSD)</th>
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<tr>
<td></td>
<td>f</td>
<td>p</td>
<td>d</td>
<td>p</td>
<td>d</td>
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<td>Lumbar vs. Pelvis</td>
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<tr>
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<td>4.1 SD 1.4 (3.3, 4.9)</td>
<td>4.3 SD 1.5 (3.4, 5.2)</td>
<td>4.1 SD 1.4 (3.3, 4.9)</td>
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<td>0.901</td>
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<tr>
<td>Fron. ROM</td>
<td>4.0 SD 1.7 (3.0, 5.0)</td>
<td>3.2 SD 1.0 (2.6, 3.8)</td>
<td>3.3 SD 1.1 (2.7, 4.0)</td>
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<td>0.211</td>
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<tr>
<td>Trans. ROM</td>
<td>4.5 SD 1.5 (3.7, 5.7)</td>
<td>3.8 SD 1.1 (3.1, 4.4)</td>
<td>3.8 SD 1.1 (3.2, 4.4)</td>
<td>0.26</td>
<td>0.201</td>
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<td>Thoracic vs. Lumbar</td>
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<tr>
<td>Sag. ROM</td>
<td>2.5 SD 0.9 (2.0, 3.0)</td>
<td>3.7 SD 1.2 (3.0, 4.5)</td>
<td>4.3 SD 2.9 (2.7, 5.9)</td>
<td>0.39</td>
<td>0.045, 0.83</td>
</tr>
<tr>
<td>Fron. ROM</td>
<td>5.6 SD 1.3 (4.9, 6.4)</td>
<td>9.2 SD 2.5 (7.7, 10.8)</td>
<td>8.4 SD 2.5 (7.0, 9.8)</td>
<td>0.70</td>
<td>&lt;0.001, 1.39</td>
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<tr>
<td>Trans. ROM</td>
<td>9.6 SD 3.4 (7.7, 11.6)</td>
<td>12.0 SD 4.7 (9.1, 14.9)</td>
<td>12.3 SD 4.3 (9.9, 14.6)</td>
<td>0.29</td>
<td>0.197, 0.69</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>1.8 SD 0.9 (1.3, 2.3)</td>
<td>2.0 SD 0.6 (1.6, 2.4)</td>
<td>2.2 SD 0.7 (1.8, 2.6)</td>
<td>0.22</td>
<td>0.462, 0.50</td>
</tr>
<tr>
<td>Fron. ROM</td>
<td>4.1 SD 1.0 (3.5, 4.6)</td>
<td>4.2 SD 1.4 (3.3, 5.0)</td>
<td>3.9 SD 1.4 (3.1, 4.6)</td>
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<td>0.823, 0.16</td>
</tr>
<tr>
<td>Trans. ROM</td>
<td>3.8 SD 1.0 (3.2, 4.3)</td>
<td>3.0 SD 1.4 (2.1, 3.8)</td>
<td>2.6 SD 1.5 (1.8, 3.4)</td>
<td>0.38</td>
<td>0.088, 0.93</td>
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<tr>
<td>Speed</td>
<td>0.32 SD 0.03 (0.31, 0.34)</td>
<td>0.36 SD 0.06 (0.33, 0.40)</td>
<td>0.40 SD 0.03 (0.38, 0.41)</td>
<td>0.80</td>
<td>&lt;0.001, 2.67</td>
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<tr>
<td>Cadence</td>
<td>45.6 SD 1.9 (44.5, 46.7)</td>
<td>48.2 SD 4.9 (45.3, 51.2)</td>
<td>51.0 SD 3.1 (49.3, 52.8)</td>
<td>0.65</td>
<td>0.001, 2.08</td>
</tr>
<tr>
<td>Stride length</td>
<td>0.85 SD 0.07 (0.81, 0.89)</td>
<td>0.90 SD 0.08 (0.85, 0.95)</td>
<td>0.94 SD 0.05 (0.91, 0.97)</td>
<td>0.56</td>
<td>0.003, 1.49</td>
</tr>
<tr>
<td>Stride time</td>
<td>2.63 SD 0.11 (2.57, 2.70)</td>
<td>2.51 SD 0.26 (2.36, 2.67)</td>
<td>2.36 SD 0.15 (2.28, 2.44)</td>
<td>0.62</td>
<td>0.001, -2.04</td>
</tr>
</tbody>
</table>

A Difference with large effect (d≥0.8) and statistical significance (p<0.05)
B Difference with large effect (d≥0.8) but no statistical significance (p>0.05)
3.4. Discussion

The main aim of this study was to investigate spinal kinematics during normal walking in healthy adolescents, adults and elderly individuals and to provide a normative dataset that can serve as a basis for future investigations on spinal pathologies.

The analyses revealed that adults exhibit more distinct thoracic kyphosis and lumbar lordosis angles than the adolescents and elderly individuals. In addition, adults and elderly individuals showed generally greater sagittal and frontal movement in the thoracic and lumbar regions compared to adolescents, who in turn exhibited more rotational movement in the cervical region. Gait speed was thereby considerably higher in adults and elderly individuals. Furthermore, the adolescents displayed a slight systematic lateral offset in the thoracic and lumbar curvatures as compared to the other groups.

The observed average lumbar lordosis angles in adults and elderly individuals were highly comparable with the values established by Dreischarf et al. [19], who used two flexible sensor strips that were attached over the paravertebral muscles in order to assess the lumbar shape and its mobility as a function of age in 323 standing asymptomatic subjects. Their total lumbar lordosis angle decreased from 36.4° in 20-29 years old subjects to 29° in subjects aged 50 years and more. By taking a closer look at the lumbar shape, they further revealed that the lower part of the lumbar spine retained its shape and mobility, while the middle section flattened and became less mobile with increasing age [19]. The observed decrease in average lordosis angle in the current study, however, showed only moderate effects and can therefore only be regarded as a tendency. Looking at the comparisons between adolescents and adults, on the other hand, the results indicate a significant age-related increase in average lumbar lordosis. Considering these and previous findings, it can be postulated that lumbar lordosis increases throughout adolescence, reaches its peak during adulthood and then again decreases with further advancing age, although the latter is likely dependent on the highly variable effects of ageing. In addition, the thoracic kyphosis seemed to follow a similar pattern by showing significant increases between adolescents and adults and decreasing-tendencies between adults and elderly individuals.

In the frontal plane, unlike the expected average values of approximately zero for all curvature angles, the analysis surprisingly showed significant differences for the lumbar and thoracic spine between the adolescents and both the adults and elderly individuals. These differences were not only recognizable during walking but also in the static measurement trial (values not included in this article). Since it is not very likely that such group-specific frontal
plane differences are of physiological nature, a possible explanation could be found in the fact that the measurements took place in two different laboratories and were carried out by two different examiners (i.e. adolescents vs. adults and elderly). Gorton et al. [134], for example, investigated the variability of the measurements of one single healthy subject conducted in 12 different motion analysis laboratories. Thereby, while the motion capture systems themselves contributed a negligible amount to the overall variability, marker placement differences between examiners were shown to have the largest impact on variability [134]. In addition, previous research has shown only poor to moderate inter-examiner reliability for the localization of lumbar and thoracic spinous processes by palpation [135, 136]. Hence, slight differences in the palpation technique between the two examiners could have introduced a systematic lateral offset in the frontal curvature angles as observed in the current study. From a clinical point of view, however, the relatively small differences (<5°) could not be considered to be relevant.

Another rather surprising finding was the observed increased ROM of the frontal lumbar curvature angle as well as the sagittal and frontal lumbar and thoracic segmental angles in elderly individuals compared to adolescents. Considering the fact that the spine becomes less mobile with advancing age [19] and that segmental trunk motion is known to decrease in older adults [120], one could have expected a decrease in ROM of the spinal curvature angles in the elderly group. However, earlier experiments have shown that decreases observed in segmental trunk motion (especially in the anteroposterior axis) with advancing age were most likely not related to age but rather to a reduced walking speed [120]. In addition, a morphology-dependent decrease in maximum ROM of the spine should not impair the only small motion required for walking. For these reasons, the currently observed increased ROM in frontal lumbar curvature angle in adults and even more in elderly individuals might most likely be associated with an increased walking speed. The question thereby arises, why the participants in the current study increased their walking speed, while the literature clearly suggests a decrease in walking speed with advancing age [128-130]? A possible explanation for this might be found in a phenomenon called the “Hawthorne effect”, or more recently known as “research participation effect” [137]. This effect describes the possible impact on behavior that occurs in an experiment as a result of the awareness of being treated, studied or observed [138]. Looking at the elderly population in the current study, the attention given to them by asking them to be part of a biomedical research project in a laboratory with sophisticated technical equipment at a prestigious university seemed to have led to an overly motivated behavior to deliver their best possible performance, which
was most likely directly associated with the increased walking speed. The adolescents were surely not less motivated, since they were all excited to see the systems that are being used to create their heroes in animated movies and video games. However, the strict laboratory atmosphere and the fact that the laboratory was located in the basement of a children’s hospital seemed to have an overwhelming and almost slightly intimidating effect on some of the young participants, leading to a rather shy behavior and hence slower walking speeds. The increased ROM values in adults compared to the adolescents might be explained in the same way or as an indirect consequence of the greater lumbar lordosis angles.

Limitations of the current study included the fact that the adolescents were measured in a different gait analysis laboratory by a different examiner than the adults and elderly individuals. Hence, slightly different calibration protocols as well as palpation techniques of the examiners might have influenced the measurements. The atypically high gait speed, especially in the elderly, was considered another possible limitation. It is therefore very important for future studies aiming at the comparison of spinal kinematics between different groups to consider gait speed as a factor that could influence ROM measurements. A final limitation was the lack of a dynamic coordinate system for the lumbar curvature angles due to a missing anterior reference point. Therefore, projection errors for this part of the spine were more likely to occur than in the thoracic spine.

3.5. Conclusions

The current study revealed clear postural differences in the sagittal plane between adolescents, adults and elderly individuals, whereby the magnitude of lumbar lordosis and thoracic kyphosis seemed to increase throughout adolescence, reach their peak in adulthood and decrease again with advancing age. In addition, differences in frontal curvature angle ROM could be identified between the groups, but were most likely due to differences in gait speed. This normative dataset provides an insight into the age-related kinematic behavior of the healthy spine during gait. It can serve as a basis for future investigations on specific pathologies or treatment effects.
4. Using Skin Markers for Spinal Curvature Quantification in Main Thoracic Adolescent Idiopathic Scoliosis: An Explorative Radiographic Study

adapted from:

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4.1. Introduction

Upper-body kinematics during daily activities such as walking are known to be important for movement control [139]. A comprehensive assessment of gait function including within-trunk and spinal movements can therefore be beneficial in specific patient groups. However, evidence of spinal movement during gait, measured using skin marker-based motion capture systems is still limited to a handful of studies, of which the majority was conducted on healthy subjects and without an appropriate evaluation of the spinal curvature [36, 37, 45, 119, 120, 122, 123, 140-143]. Standard trunk marker sets used in clinical gait analysis such as the “Plug-in Gait full body marker set” [42, 43] do not allow the tracking of spinal curvature and are hence not suitable for the quantification of abnormal spinal movement in patients with complex spinal deformities such as adolescent idiopathic scoliosis (AIS) or pathologies that affect spinal movement in the context of passive or active secondary deviations [144].

For these reasons, an enhanced trunk marker set (IfB marker set) was introduced and validated for the assessment of sagittal lumbar and thoracic curvature in healthy adults [48]. Although the validity was reported as low for the measurement of absolute spinal curvature angles, the marker set appeared to be suitable for the reliable assessment of change in sagittal lumbar and thoracic curvature.

An important aspect to consider in AIS is that the spinal deformity is always three-dimensional with the basic components being intervertebral lordosis (sagittal plane), lateral inclination (frontal plane) and axial rotation (transverse plane), i.e. the vertebral bodies rotate toward the convex side and the spinous processes toward the concave side [22, 145]. In addition, AIS patients were shown to have a distinct asymmetrical intravertebral deformity with its maximum being in the apical region of the curve [146]. Taking these factors into consideration, it is reasonable to assume that a superficial tracking of the spinal curvature with markers placed on the spinous processes will underestimate the curvature formed by the vertebral bodies in the frontal plane. Evidence for this assumption was provided by Herzenberg et al. [147], who radiographically demonstrated that the angle derived from the spinous processes significantly underestimated the Cobb-angle. However, it is plausible that this underestimation of curvature is systematic throughout normal functional activities, thereby still allowing the tracking of spinal movement and the dynamic assessment of changes in curvature.

In addition to the malrotation of vertebral bodies, inaccurate marker placement might also contribute to an under- or overestimation of the spinal curvature. Studies showed that the rate
of accurately locating the spinous processes by palpation was somewhere between 45% and 83% with a mean distance of inaccurate identification either above or below the targeted level of 19.3±18.6 mm [148-151]. This suggests that the correct identification of the spinous processes is difficult. In addition, all of the mentioned studies have only quantified the accuracy of spinous process identification in the vertical axis. No evidence is available that describes the accuracy of spinous process identification in the horizontal axis, i.e. lateral displacement from the designated location, which is crucial for understanding scoliosis, particularly in dynamic situations.

Using biplanar radiography and marker-based motion capture techniques, the primary aim of the current study was to explore the static validity of skin marker-based measurements of sagittal and frontal plane spinal curvature angles in a group of patients with AIS. In a secondary aim, the accuracy of spinous process identification by palpation and the impact of inaccurate marker placement on curvature angle measurements in the frontal plane were addressed.

4.2. Methods

4.2.1. Ethics statement

This study was approved by the local ethics committee (Ethikkommission Nordwest- und Zentralschweiz (EKNZ), Ref.-No.: 33/13). All patients as well as their legal guardians provided written informed consent to participate in this study.

4.2.2. Subjects

A consecutive sample of ten patients with AIS participated in the current study (Table 4.1). All participants were scheduled for a routine radiographic examination and were therefore not exposed to additional radiation. Inclusion criteria were an age between 10 and 18 years and the diagnosis of an idiopathic scoliosis with a structural (major) main thoracic curve (types 1-3 according to the Lenke classification [152]), whereas exclusion criteria included any other types of scoliosis (e.g. of neurological origin), previous surgical treatments or injuries to the locomotor system which led to persistent deformities in the lower extremities and the trunk.
Table 4.1: Subject demographics and marker selection for the estimation of the curvature angles in the frontal plane.

<table>
<thead>
<tr>
<th>Subject</th>
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<th>Height [m]</th>
<th>Mass [kg]</th>
<th>Lenke type</th>
<th>Sagittal Curvature</th>
<th>Thoracolumbar/Lumbar Frontal Curvature</th>
<th>Thoracic Frontal Curvature</th>
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<td>±16.8</td>
<td>±10.1</td>
<td>±12.6</td>
<td>8 left</td>
</tr>
</tbody>
</table>

The bottom row contains counts and means ± standard deviations.
Chapter 4: Radiographic Validation in Scoliosis Patients

4.2.3. Instrumentation

Standard biplanar radiographic examinations (posterior-anterior and lateral images) of the entire spine while standing were performed, including spherical radio-opaque markers (diameter: 5 mm) that were attached to the skin in a configuration described below. Measurements in the motion analysis laboratory were carried out using standard retro-reflective markers (diameter: 9-14 mm) and a 12-camera motion capture system (Vicon, Oxford, UK) at a sampling rate of 300 Hz. Selected markers from a previously developed trunk marker set (IfB marker set) [47], placed on the spinous processes of C7, T3, T5, T7, T9, T11 as well as L1-L5, were considered.

4.2.4. Procedures

Spinous processes of interest were located and marked by an experienced physiotherapist using a skin-compatible pen and with the patient in an upright seated position. First, the spinous process of C7 was located using two different methods: palpation of the two most prominent cervical spinous processes and identification of C7 by 1) flexion and assisted extension of the cervical spine (spinous process that remained stationary) [136, 151, 153] and 2) flexion and assisted rotation of the head (greater movement of spinous processes of C6 and C7 than T1) [154]. In a second step, spinous processes were counted down until L5 was identified. Finally, locations were confirmed when the spinous process of L4 corresponded to the level of the imaginary line between the two iliac crests [136, 148] and all identified spinous processes still corresponded to the respective spinal levels in a standing position. Subsequently, radio-opaque markers were placed directly onto the skin and the participants underwent the radiographic examination. After the examination, the radio-opaque markers were replaced with retro-reflective markers and the participants underwent an upright standing static measurement for the period of 2 seconds in the motion analysis laboratory.

4.2.5. Data analysis/reduction

Radiographic images were processed using the software ImageJ (version 1.47, U. S. National Institutes of Health, Bethesda, MD, USA). Cobb-angles were determined as described in the literature [155]. The centers of area (CoA) of the vertebral bodies and the markers in both planes as well as the spinous processes in the frontal plane were calculated based on their manually identified shapes (Figure 4.1A & B). Possible errors resulting from the geometry of a diverging x-ray beam were not corrected because of too many unknown variables. The
positions of the retro-reflective markers in the sagittal and frontal planes were extracted as the mean values during the 2 seconds static trial using the software Nexus (version 1.8.5, Vicon, OMG, Oxford, UK). All subsequent calculations were carried out using a custom-built MATLAB routine (R2013b, MathWorks Inc., Natick, MA, USA).

Figure 4.1: Illustration of the data extraction procedures from the posterior-anterior radiographic images. A) Identification of the shapes of the marker, vertebral body and spinous process, B) calculation of the centers of area (CoA) of the marker (M), vertebral body (VB) and spinous process (SP) and C) calculation of the horizontal (ΔH) and vertical (ΔV) distance between the marker’s CoA and the spinous process boundaries.

Curvature angle calculation in the sagittal and frontal planes was based on the radiographic (vertebral bodies and radio-opaque markers) and motion capture data (retro-reflective markers). In the sagittal plane, the lumbar curve was defined by the vertebral bodies T12-L5 and the markers placed on T11, L1-L5, while the thoracic curve was defined by the vertebral bodies T3-T12 and the markers placed on T3, T5, T7, T9 and T11. In the frontal plane, thoracolumbar / lumbar and thoracic curves were defined by the vertebral bodies and markers that corresponded to the Cobb-angle boundaries (Table 4.1), with a minimum of four markers selected for each curve. Circular segments were then established using the combination of a second order polynomial and a circle fit function (Taubin method [156]) and curvature angles were calculated based on the central angle theorem.

The accuracy of marker placement by palpation was evaluated on the basis of the posterior-anterior radiographic images (spinous processes and radio-opaque markers). Due to the laterally deviated spine, the calculated CoA’s of the spinous processes and the markers were vertically aligned according to the tilt angle of the longitudinal axis of the respective spinous process (Figure 4.1C). Vertical accuracy was then determined by measuring the absolute distances (in mm) from a marker’s CoA to the upper and lower boundaries of the respective
spinous process and horizontal accuracy by the absolute distances (in mm) from a marker’s CoA to the lateral boundaries of the spinous process. If a marker’s CoA fell between the upper and lower and/or lateral boundaries, the spinous process was considered as correctly identified in the respective direction.

4.2.6. Statistical analyses

Statistical calculations were performed using the software package SPSS 21 (SPSS Inc., Chicago, IL, USA) and statistical significance was set at p=0.05. Primary outcomes: To determine the validity of skin marker-based curvature angle measurements, linear regression analyses among the parameters vertebral body (VB)-, radio-opaque marker (RO)- and retro-reflective marker (RR)-derived curvature angles were carried out. Secondary outcomes: The accuracy of marker placement by palpation was analyzed descriptively and expressed as numbers and percentages. In addition, absolute displacement values of the markers that were not correctly identified were presented. To explore the impact of inaccurate marker placement on curvature angle measurements in the frontal plane, linear regression analyses among the parameters spinous process (SP)-, RO- and RR-derived curvature angles were carried out with higher correlations indicating a smaller impact and lower correlations a greater impact.

4.3. Results

Primary outcomes: Statistically significant moderate to strong correlations between the VB- and RO- as well as RR-derived curvature angles were found for the sagittal as well as frontal curves (Figure 4.2). Considering the attributes of the fitted regression lines, the VB-derived curvature angles of the sagittal lumbar and thoracic spines showed no substantial over- or underestimation by the RO and RR markers with slope-values ranging from 0.913 to 1.252 and y-intercept-values of below 10 degrees. In the frontal plane, however, VB-derived curvature angles were systematically underestimated when derived from the markers with slope-values between 0.882 and 1.308 and y-intercept-values of 20.4 to 34.4 degrees. The qualitative consideration of the spread of the sagittal curvature angles in the lumbar spine indicated a more accurate estimation of values less than 40 degrees. In addition, frontal thoracic curvature angles showed a slightly increased underestimation by the RR- compared to the RO-derived values.
Figure 4.2: Scatterplots and regression equations illustrating curvature angle estimation accuracy. VB: vertebral body-derived curvature angles; RO: radio-opaque marker-derived curvature angles; RR: retro-reflective marker-derived curvature angles. The asterisks (*) indicate statistical significance at the level p≤0.05.

Secondary outcomes: Six of the 110 radio-opaque markers were not visible on the radiographs and could therefore not be included in the evaluation. A total of 57.7% of the spinous processes were palpated correctly in the vertical and 38.4% in the horizontal direction (Table 4.2). Mean displacement values indicated palpatory inaccuracies between 5-18 mm in the vertical and up to 9 mm in the horizontal direction. Spinous processes were generally identified below the designated locations and towards the concave sides of the curves.
Regression analyses between the SP- and skin marker-derived curvature angles in the frontal plane showed higher correlations for the thoracic than the thoracolumbar / lumbar spine (Figure 4.3). In addition, the attributes of the fitted regression lines for the thoracolumbar / lumbar curve indicated a tendency for underestimation of the SP-derived curvature angles, whereas for the thoracic curve, no substantial under- or overestimation could be found.

**Figure 4.3:** Illustration of the impact of marker placement error on curvature angle estimation. SP: spinous process-derived curvature angles; RO: radio-opaque marker-derived curvature angles; RR: retro-reflective marker-derived curvature angles. The asterisks (*) indicate statistical significance at the level $p \leq 0.05$. 

![Graphs showing curvature angle estimation](image-url)
Table 4.2: Accuracy of spinous process identification by palpation.

| Region   | Marker | Vert.+Horiz. correct | Only vert. displaced | Only horiz. displaced | Vert.+Horiz. displaced | Missing marker | N  | %  | N  | %  | N  | %  | N  | %  | N  | %  | Vertical Mean | SD | Horizontal Mean | SD |
|----------|--------|----------------------|---------------------|----------------------|-----------------------|-----------------|-----|----|-----|----|-----|----|-----|----|-----|----------------|----|----------------|----|
| Cervical | C7     | 0                    | 0                   | 5                    | 100.0                | 0               | 0   | 0.0| 0   | 0.0| 5   | 0.0| 1   | 22.2| 5.8 | 1.2             | -1.6 | 0.8             |
|          | T3     | 0                    | 0                   | 2                    | 22.2                | 1               | 5   | 100.0| 5   | 55.6| 2   | 22.2| 1   | 22.2| 1   | 6.3 | 8.1             | -3.4 | 2.3             |
|          | T5     | 3                    | 30.0                | 2                    | 20.0                | 2               | 20.0| 0   | 0   | 0.0| 0   | 0.0| 1   | 30.0| 6.3 | 14.1            | -8.8 | 7.2             |
|          | T7     | 0                    | 0                   | 2                    | 20.0                | 2               | 20.0| 0   | 0   | 0.0| 0   | 0.0| 1   | 20.0| 6.3 | 14.1            | -8.8 | 7.2             |
|          | T9     | 0                    | 0                   | 1                    | 10.0                | 3               | 30.0| 0   | 0   | 0.0| 0   | 0.0| 0   | 30.0| 6.3 | 14.1            | -8.8 | 7.2             |
|          | T11    | 1                    | 10.0                | 2                    | 20.0                | 3               | 30.0| 0   | 0   | 0.0| 0   | 0.0| 0   | 30.0| 6.3 | 14.1            | -8.8 | 7.2             |
|          | T3-T11 | 6                    | 12.2                | 7                    | 14.3                | 24              | 49.0| 12  | 24.5| 1   | -6.3 | 8.2 | -3.1 | 4.4 |
|          | L1     | 2                    | 20.0                | 1                    | 10.0                | 5               | 50.0| 2   | 20.0| 0   | -13.3 | 13.2 | 0.5 | 0.2 |
|          | L2     | 2                    | 20.0                | 1                    | 10.0                | 4               | 40.0| 3   | 30.0| 0   | -11.9 | 8.8 | 2.5 | 1.2 |
|          | L3     | 5                    | 50.0                | 2                    | 20.0                | 1               | 10.0| 2   | 20.0| 0   | -8.6 | 3.5 | 0.6 | 0.4 |
|          | L4     | 3                    | 30.0                | 2                    | 20.0                | 3               | 30.0| 2   | 20.0| 0   | -7.3 | 1.7 | 1.3 | 1.0 |
|          | L5     | 2                    | 20.0                | 2                    | 20.0                | 3               | 30.0| 3   | 30.0| 0   | -11.8 | 2.1 | -0.4 | 1.7 |
|          | L1-L5  | 14                   | 28.0                | 8                    | 16.0                | 16              | 32.0| 12  | 24.0| 0   | -10.6 | 2.5 | 0.9 | 1.1 |
| All      | C7-L5  | 20                   | 19.2                | 20                   | 19.2                | 40              | 38.5| 24  | 23.1| 6   | -8.2 | 5.9 | -1.3 | 3.5 |

Numbers and percentages: markers that were placed correctly in both directions and those that were displaced in only the vertical, only the horizontal and both the vertical and horizontal directions. Displacement values: vertical displacement: positive = above designated spinous process, negative = below; horizontal displacement: positive = towards the convex side of upper curve, negative = towards concave side of upper curve.
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4.4. Discussion

The current study aimed at validating skin marker-based measurements of spinal curvature angles in the sagittal and frontal planes in patients with main thoracic AIS. In addition, the accuracy of marker placement by spinous process palpation and the impact of inaccurate placement on spinal curvature angle measurements were addressed.

In the sagittal plane, lumbar and thoracic curvature angles could be estimated with a reasonable accuracy by both the RO- and RR-markers, whereas in the frontal plane, thoracolumbar / lumbar and thoracic curvature angles were systematically underestimated by the skin markers. About half of the spinous processes were not identified correctly by palpation in the vertical and another half in the horizontal direction, but with fairly low displacement values. Inaccurate placement of markers appeared to have a greater impact on curvature angle estimation in the thoracolumbar / lumbar than the thoracic spine. While the consequence of these results for assessing the kinematics of the spine during dynamic activities remains to be elucidated in further studies, these data suggest that it might be possible to perform a systematic correction of the marker data or to focus on movement patterns (relative angular differences) in order to provide clinical understanding to dynamic data.

Only few studies evaluated the validity of skin marker-derived spinal curvature measurements [48, 157, 158]. In the late 80’s, Bryant et al. [157] investigated the accuracy of estimating sagittal thoracic and lumbar spine curvatures using RO markers and upright standing lateral view radiographic images in healthy adolescents. Their results suggested accurate estimation of vertebral centroid curves by the RO markers. The thoracic kyphosis was more reliably measured than the lumbar lordosis, possibly due to a greater soft tissue thickness in the lumbar region. Despite the slightly different methodological approach, the current findings on AIS patients largely support these results. Moreover, the fact that a lumbar lordosis of more than approximately 40 degrees was less accurately estimated in AIS patients indicated that the issue of greater soft tissue thickness in the lumbar region seems to also depend upon the extent of the lordotic posture.

To investigate the validity of skin marker-based measurements for the quantification of spinal movement in the sagittal plane, Mörl and Blickhan [159] used MRI images of healthy adults in different seating postures and showed that lumbar vertebral position and spatial orientation could be estimated using skin markers. Using a similar method, Zemp et al. [48] examined soft tissue artifacts as well as estimation accuracies of skin markers (same placement as in the
current study) for the measurement of lumbar and thoracic curves. They concluded that skin markers were suitable for the assessment of change in the sagittal curvature angles, but that absolute values suffered from uncertainty. Considering the tendency for lower estimation accuracy with increasing lumbar lordosis as reported in the current study, their suggestions to use skin markers for the assessment of postural change rather than absolute angles should also be followed for measurements in AIS patients.

While the two MRI-based studies above only described spinal curvature measurements in the sagittal plane, Hashemirad et al. [158] validated spinal curvature measurements in the frontal plane using posterior-anterior fluoroscopy images in a neutral and a lateral bending position. Their results suggested that skin markers could be confidently used for the estimation of lumbar spine curvature during lateral bending. Therefore, even though frontal curvatures in the current study were clearly underestimated mainly due to the rotational deformities in AIS patients (i.e. axial rotation and intrinsic axial torsional deformity of the vertebrae), skin markers might still be used for the assessment of movement in this plane.

The current study further showed that VB-derived sagittal thoracic curvature angles could be better estimated by the RO than the RR markers. This might be explained by the fact that in the movement analysis laboratory, the standing position of some patients did not exactly match the posture during the radiographic examination. For the lateral view images, patients were required to hold on to a horizontal bar elevating their arms to the front, which might have caused slight positional differences especially in the upper thoracic spine. If possible, future validations should therefore aim to perform the radiographic and motion capturing measurements simultaneously.

Concerning the differences between RO- and RR-derived measurements, the slightly increased underestimation of the frontal plane curvature angles by the RR compared to the RO markers might be due the fact that the RR markers were mounted on a plastic socket that allowed slight tilting towards the concave side when placed on the paravertebral muscles in the area of the curvature’s apex. In general, regardless of the different types of markers, the underestimation of the curvatures in the frontal plane could be explained mainly by the pathology related structural deformity of the vertebrae as well as a systematic marker placement error towards the concave side of the curvatures. The fact that the palpable parts of the spinous processes were in most cases approximately one level below the position of the respective vertebral body (especially in the lower thoracic spine) might have led to an additional falsification of the actual frontal plane curvature.
Considering the available evidence on the identification accuracy of selected spinous processes, the results of the current study seem to be in agreement for the lumbar but not for the cervical spine. Harlick et al. [148] evaluated the identification accuracy of lumbar spine levels in adults (in the vertical direction), which were identified correctly in 47% of the cases (current study: 60%). The average absolute displacement of the markers that were not identified correctly was 19.3±18.6 mm (current study: 10.6±2.5 mm). Other studies on adult and elderly subjects showed palpatory accuracies of 36-61% for the L5 (current study: 50%) and 55-77% for the C7 spinous process (current study: 0%) [136, 149, 151]. The low accuracy for the identification of C7 in the current study might be explained by the fact that the discrimination between C6 and C7 might have been harder in children and adolescents as compared to adults due to the size of the structures. In addition, five out of six markers that were not visible on the radiographs applied to C7. The placement of the thoracic markers could not be compared to the literature since no studies were available investigating the identification accuracy of thoracic spinous processes.

4.5. Conclusions

Skin marker-based motion capture techniques can be used for the non-invasive assessment of spinal curvature angles in the sagittal and frontal planes in patients with AIS. However, while absolute values in the sagittal plane could be measured with reasonable accuracy, frontal plane angles were systematically underestimated, mainly due to the rotational deformities of the scoliotic vertebrae. Skin markers on the trunk should therefore be used for the assessment of movement and postural change (i.e. during dynamic tasks such as walking) rather than for the measurement of absolute angles.

Inaccuracy of marker placement by palpation had a greater impact on the determination of the thoracolumbar / lumbar than the thoracic curvature angles. In order to keep such inaccuracies minimal, only health care professionals with experience in palpation should place markers. Based on the current and previous findings, it is suggested that spinal curvature measurements are included in marker-based clinical gait analysis protocols. This would enable a deeper understanding of the behavior of the healthy and pathological spine in dynamic situations and would open up the possibility for a more comprehensive evaluation of treatment effects as movement seems to be detected reasonably well. In addition, the data can be used to drive complex spinal models in order to get an insight into the dynamic loading of the spine during movement.
5. Quantifying Spinal Gait Kinematics using an Enhanced Optical Motion Capture Approach in Adolescent Idiopathic Scoliosis

adapted from:

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Permission for reprint granted from Elsevier on Jan 07, 2016 (Appendix E)
5.1. Introduction

Adolescent idiopathic scoliosis (AIS) is a complex three-dimensional deformity of the spine that includes intervertebral lordosis, lateral inclination and axial rotation [145]. The overall prevalence of AIS is 0.47-5.2% with a greater incidence and higher curve severity in girls than boys. Most commonly, the pathology presents itself in the thoracic and thoracolumbar/lumbar regions [23]. To this day, however, the pathogenesis of AIS remains poorly understood [9, 160]. Based on a literature review, de Seze et al. [160] proposed four main pathogenetic mechanisms: asymmetric bone growth regulation, susceptibility of bones to deformation, abnormal passive spinal system maintenance and disturbed active spinal system maintenance. Another recent review concerning the pathogenesis of AIS revealed possible connections between AIS and impaired gait control as well as decreased bone mineral density [9]. These findings are consistent with the earlier postulation of Burwell et al. [8] (also known as “the Nottingham concept”), which stated that kinematic differences in the spine, pelvis and lower extremities during gait might contribute to the progression of AIS.

Several research groups investigated gait kinematics in AIS patients using opto-electronic motion capture systems [33, 38, 143, 161-165]. As a result, an understanding of the reduced frontal plane trunk motion [33, 163] combined with asymmetrical trunk motion in the frontal and transverse planes [38, 165] was established in patients with AIS. The only study focusing more on actual spinal movement evaluated solely the within-day measurement reliability and did not compare kinematic parameters between AIS patients and healthy controls [143]. However, while considering the trunk as a rigid segment has yielded some basic information on upper body movement, it is clearly insufficient to explain many of the important aspects of spinal motion. Using a more sophisticated approach that enables kinematic measurements of the actual spinal motion and not just the relative angles between rigid trunk segments, would provide useful information that could assist researchers and clinicians in the management of patients with AIS. In particular, treatment methods such as physiotherapy, orthotics or surgery (e.g. spinal fusion) could be evaluated in a more comprehensive manner by investigating not only the effects of secondary deviations (passive physical effects or active compensatory mechanisms [144]) but also of deviations resulting directly from the primary pathology. Furthermore, the data could be used for the development and as input parameters of more complex musculoskeletal models to simulate internal spinal loading and to gain an insight into the dynamics of the scoliotic spine.
Marker models that are commonly used in clinical gait analysis such as the “Plug-in Gait full body” consider the relative movements between a rigid trunk and pelvis segment, in which the spinal shape is not reflected [43]. For measurement of the actual curvature of the spine, especially in the thoracic region, such non-specific marker models are clearly inappropriate. Therefore, an enhanced trunk marker set, developed at the ETH Zurich’s Institute for Biomechanics in Switzerland (IfB marker set) [47], has been introduced and validated for the measurement of spinal curvature angles in healthy subjects [48] and patients with AIS [126]. The primary aim of the current study was to quantify spinal curvature angles during gait in a cohort of AIS patients and to compare these angles to a group of healthy subjects as well as to analyze them in relation to the severity of the deformity. A secondary aim was to investigate overall trunk motion and spatio-temporal gait properties in order to support the interpretation of the primary outcomes and to provide a basis for comparisons with previous studies.

5.2. Methods

5.2.1. Study population

A consecutive sample of 14 AIS patients and a cohort of 15 asymptomatic control subjects participated in this cross-sectional observational study (Table 5.1). Patients were recruited among the 10-18 year old AIS patients with both a thoracic and a thoracolumbar/lumbar curve component that were scheduled for a clinical examination including biplanar radiography at the University of Basel Children’s Hospital, Switzerland. Exclusion criteria were any other type of scoliosis, previous surgical treatment of the spine, locomotor system injuries that led to persistent deformities and neuromuscular disorders. The control subjects were included if they were between the ages of 10 and 18 years and did not present any pathologies, diseases or injuries affecting the locomotor system, obesity (> 95th BMI-per-age percentile) or leg length (anterior superior iliac spine to medial malleolus) inequalities of > 1% of body height. All participants as well as their legal guardians signed informed consent and the study protocol was approved by the local ethics committee.
Chapter 5: Spinal Kinematics in Scoliosis Patients

Table 5.1: Demographics of the AIS patients and healthy controls expressed as means and ranges. Group comparisons were conducted using independent samples T-tests (statistical significance p≤0.05).

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</tr>
<tr>
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<tr>
<td>Thoracic curve</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Cobb-angle [°]</td>
<td>43.7 (14.0-70.9)</td>
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<td>N/A</td>
</tr>
<tr>
<td>Convexity [right/left]</td>
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<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Thoracolumbar / lumbar curve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cobb-angle [°]</td>
<td>38.7 (9.2-55.6)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Convexity [right/left]</td>
<td>2/12</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

5.2.2. Data collection

Immediately prior to the gait analysis, AIS patients underwent a standard biplanar radiographic examination for the determination of the Cobb-angles. All participants were then equipped with 56 retro-reflective markers (combination of the Plug-in Gait full body and the IfB marker sets; Figure 5.1A & B) by a physiotherapist with experience in clinical gait analysis (same person for all measurements). Gait analysis measurements were conducted using a 12-camera motion analysis system (type MXT20, Vicon, Oxford, UK; sampling frequency: 200-300 Hz). Following a standing measurement trial in an upright position, participants were asked to walk at their self-selected speed on a 10 m level ground walkway until at least 5 trials were recorded.

5.2.3. Data reduction

The software Nexus (version 1.8.5, Vicon, Oxford, UK) was used to set heel strikes and toe-off’s. Kinematic data were processed using a custom-built MATLAB-routine (version R2013b, MathWorks Inc., Natick, MA, USA). Spinal curvature was determined based on the markers placed over the spinous processes (Figure 5.1C & D). Thereby, sagittal thoracic and lumbar curves of all participants were defined by the markers SPT1-SPT5 and SPT5-SPL5 (where SP=spinous process, T=thoracic, L=lumbar). In the frontal plane, thoracic and thoracolumbar/lumbar curves of the control subjects were defined by the markers SPT1-SPT5 and SPT5-SPL4. For the AIS patients, frontal curves were defined by the markers that
corresponded to the Cobb-angle boundaries, with a minimum of four markers selected for each curve. In order to avoid possible projection errors due to the thorax rotation during gait, dynamic sagittal and frontal planes were calculated for the thoracic curvatures based on the marker on the sternum (STER) and the marker closest to the apex of the scoliotic curve. For the thoracolumbar/lumbar curvatures, sagittal and frontal planes were defined according to the global coordinate system since a meaningful anterior reference point was missing. Circular segments were then established using the combination of a second order polynomial and a circle fit function and curvature angles were calculated based on the Central Angle Theorem, as described previously [126]. To quantify overall trunk motion, position and orientation of a rigid pelvis, lumbar, thoracic and cervical segment (Figure 5.1E) were determined relative to the reference segments defined by the standing trial using a least-squares fit of the corresponding marker point clouds [47] and expressed as absolute (segment versus global coordinate system) angles in the sagittal, frontal and transverse planes. In addition, relative angles between the thoracic and the pelvis segments in all three planes were calculated for comparative purposes.

All angles were time normalized to a right gait cycle (time between two consecutive right foot strikes) consisting of 101 points. Two AIS patients had a left sided convexity of the thoracic scoliotic curve and therefore the mirrored data of a left gait cycle were used. The primary outcome parameters were then defined as the average and range of motion (ROM) values of the spinal curvature angles over the full gait cycle. In the sagittal plane, positive values are referred to as kyphosis and negative values as lordosis. Secondary outcomes included the ROM values of the segmental angles over the full gait cycle and selected spatio-temporal gait parameters. The average values of the segmental angles were considered not to be meaningful parameters since these angles were calculated in relation to the static trial. Spatio-temporal gait parameters consisted of speed (horizontal distance of sacrum marker divided by stride time), cadence (two divided by stride time), left and right step length (horizontal distance of heel marker over one step) as well as left and right step time. All angles were expressed in degrees [°] and spatio-temporal gait parameters in dimensionless numbers [132].
Figure 5.1: A & B: Combination of the Plug-in Gait full body and the IfB marker sets for the modelling of within-trunk and spinal kinematics. White circles with black filling represent markers that were used for both models, while black circles with white filling represent markers that were only used for the IfB model, and markers with black circles were only used for the Plug-in Gait full body model (SPC1-3: spinous processes of C3, C5 and C7; SPT1-5: spinous processes of T3, T5, T7, T9 and T11; SPL1-5: spinous processes of L1-L5). C & D: Definition of the thoracolumbar / lumbar (TLC) and thoracic (TC) curvature angles in the sagittal (C) and frontal planes (D). E: Definition of the rigid pelvis, lumbar (LS), thoracic (TS) and cervical (CS) segments.

5.2.4. Statistical analyses

Statistical testing was carried out using the software package SPSS 21 (SPSS Inc., Chicago, IL, USA). Prior to any analyses, normality of the parameters was confirmed using the Shapiro-Wilk test (normal distribution: p>0.05). To determine whether to base the statistical calculations on the average of the five trials per subject or on a single representative trial,
intraclass correlation coefficients (ICC(2,1)), type consistency, and their 95% confidence intervals (CI) were calculated. ICCs of ≥0.85 were considered acceptable for kinematic gait data [166]. In case of generally inacceptable intra-session reliability, the principal component analysis-based Selection Method for a Representative Trial (SMaRT) [167] was applied. Group differences among several parameters were explored using independent samples T-tests and effect sizes (Cohen’s d). To determine the clinical relevance of the differences showing considerable effects (small: d≥0.2, moderate: d≥0.5 and large: d≥0.8) [168], minimal clinically important differences (MCID) were defined based on expert opinions and previously published data [169] (Table 3). Thereby, the differences were categorized as A) clinically relevant (95% CI of mean difference above MCID), B) possibly clinically relevant (95% CI of mean difference contained MCID) and C) not clinically relevant (95% CI of mean difference below MCID) [170, 171]. Results categorized as possibly clinically relevant were interpreted according to the magnitude of their effect sizes and considered as dependent on further data. The relationships between the curvature angle parameters and severity of scoliosis (main Cobb-angle) were investigated using linear regression analyses (coefficients of determination, $R^2$) and effect sizes (Cohen’s $f^2$). Correlations showing considerable effects (small: $f^2$≥0.02, moderate: $f^2$≥0.15 and large: $f^2$≥0.35) [168] were categorized as 1) statistically significant (p≤0.05) and 2) statistically not significant (p>0.05, further data needed). Results with trivial effects (Cohen’s d<0.2, Cohen’s $f^2$<0.02) [168] were not further explored and considered as conclusive.

5.3. Results

The AIS patients and healthy controls did not differ in age, height or mass (Table 5.1). However, the AIS patient group consisted mainly of females, whereas in the control group, males and females were equally distributed. The average severity of scoliosis (Cobb’s angle) was 44° and 39° for the thoracic and the thoracolumbar/lumbar curves.

Since the majority of kinematic parameters showed inacceptable intra-session reliability (Table 5.2), calculations were based on a representative trial, selected using the SMaRT method [167].

5.3.1. Primary outcomes

The analysis of thoracic curvature angles indicated on average 10.7° (4.2°,17.3°) less kyphosis but 4.9° (2.3°,7.6°) more ROM in the sagittal plane as well as 12.7° (6.2°,19.3°)
average lateral deviation to the left side in AIS patients (Table 5.3). In the thoracolumbar/lumbar spine, curvature angles indicated on average 3.3° (8.7°,15.3°) more lordosis as well as 22.5° (14.1°,30.8°) average lateral deviation to the right side and 2.4° (1.1°,5.8°) greater ROM in the frontal plane in AIS patients. In addition, positive correlations between the main Cobb-angle and the sagittal ROM ($R^2=0.503$) as well as frontal average ($R^2=0.360$) thoracic curvature angles and the thoracolumbar/lumbar sagittal and frontal average curvature angles ($R^2=0.122$ and $R^2=0.259$) were indicated. Although the sagittal average and ROM parameters of the thoracic curvature angles were categorized as possibly clinically relevant, these results were considered in the interpretation due to their large effect sizes (d=1.25 and d=1.53). The also as possibly clinically relevant categorized sagittal average and frontal ROM parameters of the thoracolumbar/lumbar curvature angles, however, were not considered due to only small to moderate effect sizes. Likewise, the statistically not significant correlation for the sagittal average thoracolumbar/lumbar curvature angle was not considered due to a small effect size. For illustrative purposes, a graphical representation of the curvature angles over time is provided in Figure 5.2.

5.3.2. Secondary outcomes

The segmental angle and spatio-temporal gait parameters did not indicate clinically relevant differences between the two groups (Table 5.3). A complete set of the segmental angles over a full gait cycle can be found in Figures C.1 and C.2 (Appendix C).
Table 5.2: Reported are the results of the reliability analyses using the intraclass correlation coefficients (ICC(2,1), type consistency) as well as the group means and standard deviations (SD) for the average (AVG) and range of motion (ROM) values of the curvature and segmental angles (expressed in degrees [°]) in the sagittal (Sag.), frontal (Fron.) and transverse (Tran.) planes and the spatio-temporal gait parameters (expressed as dimensionless numbers) in patients with adolescent idiopathic scoliosis (AIS, n=14) and healthy controls (n=15) during gait. The brackets contain the boundaries of the respective 95% confidence intervals.

<table>
<thead>
<tr>
<th></th>
<th>Intra-session reliability</th>
<th>Kinematic data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AIS</td>
<td>Controls</td>
</tr>
<tr>
<td>Thoracic Curves</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>0.984 (0.965,0.994) ^</td>
<td>0.949 (0.885,0.984) ^</td>
</tr>
<tr>
<td>ROM</td>
<td>0.710 (0.497,0.881)</td>
<td>0.192 (-0.017,0.550)</td>
</tr>
<tr>
<td>Fron. AVG</td>
<td>0.996 (0.992,0.999) ^</td>
<td>0.907 (0.798,0.970) ^</td>
</tr>
<tr>
<td>ROM</td>
<td>0.854 (0.716,0.945) ^</td>
<td>0.811 (0.627,0.935)</td>
</tr>
<tr>
<td>Thoraco-lumbar / lumbar Curves</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>0.987 (0.972,0.996) ^</td>
<td>0.909 (0.802,0.971) ^</td>
</tr>
<tr>
<td>ROM</td>
<td>0.339 (0.107,0.649)</td>
<td>0.366 (0.111,0.669)</td>
</tr>
<tr>
<td>Fron. AVG</td>
<td>0.995 (0.990,0.998) ^</td>
<td>0.834 (0.666,0.944)</td>
</tr>
<tr>
<td>ROM</td>
<td>0.766 (0.577,0.908)</td>
<td>0.805 (0.617,0.933)</td>
</tr>
<tr>
<td>Pelvis Segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.445 (0.200,0.728)</td>
<td>0.057 (-0.100,0.392)</td>
</tr>
<tr>
<td>Fron. ROM</td>
<td>0.694 (0.476,0.873)</td>
<td>0.871 (0.730,0.957) ^</td>
</tr>
<tr>
<td>Cervical Segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.549 (0.304,0.795)</td>
<td>0.564 (0.299,0.823)</td>
</tr>
<tr>
<td>Tran. ROM</td>
<td>0.420 (0.176,0.711)</td>
<td>0.320 (0.074,0.664)</td>
</tr>
<tr>
<td>Thoracic Segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.428 (0.098,0.640)</td>
<td>0.712 (0.478,0.895)</td>
</tr>
<tr>
<td>Tran. ROM</td>
<td>0.462 (0.216,0.740)</td>
<td>0.770 (0.563,0.919)</td>
</tr>
<tr>
<td>Cervical Segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.559 (0.314,0.801)</td>
<td>0.514 (0.246,0.795)</td>
</tr>
<tr>
<td>Tran. ROM</td>
<td>0.628 (0.393,0.840)</td>
<td>0.620 (0.362,0.852)</td>
</tr>
<tr>
<td>Thoracic vs. Pelvis Segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.828 (0.673,0.934)</td>
<td>0.911 (0.807,0.971) ^</td>
</tr>
</tbody>
</table>
### Table 5.2: continued

<table>
<thead>
<tr>
<th></th>
<th>Intra-session reliability</th>
<th>Kinematic data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AIS</td>
<td>Controls</td>
</tr>
<tr>
<td>Speed</td>
<td>0.867 (0.737,0.950) ^a</td>
<td>0.867 (0.723,0.956) ^a</td>
</tr>
<tr>
<td></td>
<td>0.828 (0.672,0.934)</td>
<td>0.767 (0.557,0.918)</td>
</tr>
<tr>
<td>Cadence</td>
<td>0.928 (0.851,0.974) ^a</td>
<td>0.887 (0.760,0.963) ^a</td>
</tr>
<tr>
<td></td>
<td>0.893 (0.785,0.961) ^a</td>
<td>0.860 (0.711,0.953) ^a</td>
</tr>
<tr>
<td>Step length</td>
<td>Right</td>
<td>0.763 (0.552,0.916)</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>0.767 (0.557,0.918)</td>
</tr>
<tr>
<td></td>
<td>Right</td>
<td>0.782 (0.580,0.924)</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>0.782 (0.580,0.924)</td>
</tr>
</tbody>
</table>

^a Acceptable ICC (≥0.85)
Table 5.3: Reported are the results of the group comparisons (absolute values of the mean differences with 95% confidence intervals in the brackets (curvature and segmental angles expressed in degrees [°], spatio-temporal gait parameters expressed as dimensionless numbers), effect sizes (Cohen’s d) and p-values) as well as the correlations with severity of disease (coefficients of determination ($R^2$), effect sizes (Cohen’s $f^2$) and p-values). To determine clinical relevance, minimal clinical important differences (MCID) were defined.

<table>
<thead>
<tr>
<th>Group comparisons</th>
<th>Mean differences (absolute values)</th>
<th>Correlations</th>
</tr>
</thead>
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<tr>
<td></td>
<td>AVG</td>
<td>MCID</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>10.7 (4.2,17.3) $^B$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>4.9 (2.3,7.6) $^B$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>12.7 (6.2,19.3) $^A$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.8 (-2.0,3.6) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>3.3 (-8.7,15.3) $^B$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>1.4 (-1.8,4.7) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>22.5 (14.1,30.8) $^A$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>2.4 (-1.1,5.8) $^B$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>0.1 (-0.6,0.9) $^*$</td>
<td>5</td>
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<tr>
<td>Sag. ROM</td>
<td>1.6 (-0.3,3.4) $^C$</td>
<td>5</td>
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<tr>
<td>Sag. ROM</td>
<td>0.9 (-2.1,3.8) $^C$</td>
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</tr>
<tr>
<td>Sag. AVG</td>
<td>0.0 (-0.9,0.8) $^*$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>1.8 (0.2,3.5) $^C$</td>
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<tr>
<td>Sag. ROM</td>
<td>1.4 (-1.3,4.0) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>0.8 (0.0,1.5) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.2 (-0.7,1.2) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>0.7 (-0.8,2.2) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. AVG</td>
<td>0.7 (-0.3,1.8) $^C$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
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</tr>
<tr>
<td>Sag. ROM</td>
<td>0.7 (-1.2,2.5) $^C$</td>
<td>5</td>
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<tr>
<td>Sag. AVG</td>
<td>0.6 (-0.9,2.0) $^C$</td>
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<tr>
<td>Sag. ROM</td>
<td>0.5 (-1.5,2.5) $^*$</td>
<td>5</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>1.4 (-1.4,4.1) $^C$</td>
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Continued on next page
<table>
<thead>
<tr>
<th>Group comparisons</th>
<th>Mean differences (absolute values)</th>
<th>MCID</th>
<th>Cohen’s $d$</th>
<th>p-value</th>
<th>$R^2$</th>
<th>Cohen’s $f^2$</th>
<th>p-value</th>
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<tbody>
<tr>
<td>Speed</td>
<td>0.01 (-0.02,0.04) $^C$</td>
<td>0.1</td>
<td>0.22</td>
<td>0.443</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cadence</td>
<td>1.6 (0.3,2.9) $^C$</td>
<td>8.5</td>
<td>0.96</td>
<td>0.015</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Step length</td>
<td>Right 0.02 (-0.04,0.08) $^C$</td>
<td>0.2</td>
<td>0.25</td>
<td>0.515</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Left 0.04 (-0.02,0.10) $^C$</td>
<td>0.2</td>
<td>0.35</td>
<td>0.227</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Step time</td>
<td>Right 0.08 (0.00,0.16) $^C$</td>
<td>0.4</td>
<td>0.80</td>
<td>0.044</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Left 0.08 (0.02,0.15) $^C$</td>
<td>0.4</td>
<td>1.13</td>
<td>0.013</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

$^A$ Clinically relevant difference  
$^B$ Possibly clinically relevant difference, more data needed  
$^C$ No clinically relevant difference  
$^D$ Statistically significant correlation  
$^E$ Statistically not significant correlation, more data needed  
$^*$ Trivial effect, results not further explored
Figure 5.2: Curvature angles of the lumbar and thoracic curves in the sagittal as well as thoracolumbar / lumbar and thoracic curves in the frontal plane over one complete gait cycle. The black lines represent the AIS patients and the grey lines the healthy controls (solid = mean; dashed = standard deviation).

5.4. Discussion

This cross-sectional observational study aimed to provide a deeper understanding of spinal kinematics during gait in AIS patients compared to asymptomatic controls. The results indicated postural differences in the sagittal and frontal planes as well as a Cobb angle-dependent altered motion of the thoracic spine in the sagittal plane. Commonly used “spinal motion” parameters such as the relative angles between a rigid thorax and pelvis segment did not show clinically relevant differences between the groups. Therefore, a specific spine model, as used in this study, is required before clinically relevant parameters can be extracted when investigating patients with spinal deformities during gait.

The AIS patients showed less average thoracic kyphosis than the healthy controls during gait. A diminished thoracic kyphosis (hypokyphosis), resulting from relative anterior spinal
overgrowth during the adolescent growth spurt, has been previously associated with thoracic AIS and was reported to be strongly related to curve progression, i.e. faster progression of scoliotic deformation was observed in patients with smaller thoracic kyphosis [172]. Since the sagittal thoracic curvature angle was previously reported to be validly measurable using skin markers [126], this might be a valuable non-invasive parameter for the prediction of curve progression.

Furthermore, the lateral deformation of the thoracic and thoracolumbar/lumbar spines in AIS patients during gait could be clearly identified. When compared to the radiography-derived Cobb-angles, however, the skin marker-based curvature angles were considerably lower. This issue has been previously described by Schmid et al. [126] as a systematic underestimation of skin marker-based frontal plane curvature angle measurements due to the rotational deformity of the scoliotic vertebrae. The authors therefore suggested focusing on the ROM of lateral-flexion movements which considers differences of absolute positions rather than on the absolute values. In this regard, frontal plane ROM during gait seemed not to be affected by thoracic AIS.

A somewhat unexpected outcome of the current study was the Cobb-angle dependent greater sagittal thoracic ROM in AIS patients. Considering the three-dimensional deformity of the scoliotic spine, this could be due to the manner in which the angles were calculated. Since the sagittal curvature angle was calculated based on the height of the circular segment established by the markers SPT1 and SPT5, a linearly increasing isolated lateral-flexion of a kyphotic spine would cause an exponentially increasing narrowing of these markers and an equally increasing sagittal curvature angle. A lateral-flexion of 5° in a straight spine would therefore have caused less narrowing of the markers than the same lateral-flexion in an already laterally-deformed spine. On the other hand, since the sagittal lumbar curvature angle showed only weak differences in ROM between the two groups, it might be possible that lateral-flexion of the scoliotic thoracic spine was indeed accompanied by an increased flexion-extension movement during gait. This movement was ascribed to a previously reported axial rotational instability caused by anterior spinal overgrowth in thoracic AIS [172]. In addition, Mahaudens et al. [163] described an increased duration of erector spinae muscle activation around heel-strike, possibly as an attempt to stabilize the instable spinal segments during moments of higher impact loads. Since their measurements were based on a group of patients with thoracolumbar and lumbar AIS, however, future studies should investigate whether this activation pattern also applies for thoracic AIS.
It has been previously shown that head and pelvis motion were positively related to gait speed [124]. For this reason, it is important to include an evaluation of spatio-temporal gait properties when investigating gait kinematics. In this study, altered kinematics in AIS patients due to gait speed could thereby be ruled out.

Compared to earlier studies that reported altered trunk motion and spatio-temporal gait parameters in AIS patients [33, 38, 163, 165], the current results seem to deviate. Reasons might be that in the current study, group differences were subjected to an evaluation of clinical relevance using a defined threshold, the cohort was more homogeneous and the spatio-temporal gait parameters were considered in relation the participants’ anthropometric properties.

The fact that the thoracolumbar/lumbar curvature angles were based on the global coordinate system might have increased the chances for projection error and was therefore considered a possible limitation of this study. A meaningful anterior reference point, as required for the definition of a dynamic coordinate system, however, was missing for this part of the spine. When looking at the assessment of frontal plane curvature angles, the current method was further limited by the previously documented systematic underestimation in AIS patients [126]. In addition, the lack of data on between-session reliability limits the assessment of spinal curvature angles in a test-retest manner, required when reporting disease progression or evaluating treatment effects. Even though the marker placement accuracy was previously shown to be high [126], data on the consistency of repeated markers application would provide additional information on the sensitivity of the method. Furthermore, the SMaRT method, chosen because of insufficient within-session reliability, lacked on validity for trunk and spine parameters. Therefore, further studies should include an evaluation on the validity of this method or explore the possibility of increasing the reliability by measuring more than five trails per session. And last but not least, the small sample size limited the conclusiveness of some of the parameters. More data could be generated by including the assessment of spinal curvature angles during gait into the standard clinical examination routine in AIS patients.

5.5. Conclusions

The results of the current study provide an enhanced insight into the biomechanical behavior of the spine in AIS patients during gait. When evaluating commonly used gait analysis parameters, AIS patients did not indicate any clinically relevant differences compared to
healthy controls. The analysis of spinal curvature angles, however, revealed postural differences in the sagittal and frontal planes as well as an altered sagittal movement pattern of the thoracic spine without showing any clinically relevant differences in the spatio-temporal parameters. This demonstrates that the dynamic functionality of the scoliotic spine can be assessed using advanced non-invasive optical approaches and that these should become standard in clinical gait analysis. Furthermore, curvature angle data could provide a useful basis for driving sophisticated computer simulation models in order to gain an insight into the dynamic loading behavior of the scoliotic spine during gait.
6. Orthotic Correction of Lower Limb Function during Gait does not immediately influence Spinal Kinematics in Spastic Hemiplegic Cerebral Palsy

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6.1. Introduction

Cerebral palsy (CP) results from a static lesion of the brain in utero, during delivery or within the first two years after birth [173]. The overall prevalence was reported to be 2.4 per 1000 children aged 3 to 10 years [173]. About 25% of all cases present an involvement of one side of the body and are classified as hemiplegic CP (hCP) [173]. The most common gait problem in hCP patients is the foot equinus, caused by calf-muscle spasticity and dorsiflexor weakness, which results in a toe-walking pattern on the affected side [55, 174]. Biomechanical studies showed that unilateral toe-walking leads to secondary deviations such as increased pelvic rotation and anterior pelvic tilt as a result from hip flexion, internal rotation and adduction [2, 46]. In addition, hCP patients often develop a structurally shorter leg on the affected side [175], which has been shown to cause pelvic obliquity in children without associated neuromuscular diseases [176]. Considering these effects, it is therefore not unreasonable to assume that the spinal deviations in hCP are caused secondarily due to foot equinus and leg length discrepancy (LLD). Primary deviations (resulting directly from the brain lesion) are unlikely since innervation of the trunk muscles is known to remain almost normal in hCP and patients develop good trunk control [177]. Unfortunately, evidence on upper body kinematics in hCP gait is scarce. Only two studies reported on differences in posture and motion of the trunk in this population [30, 43] and these findings are based on commonly used gait analysis methods which only allow the quantification of general trunk movements but not of the underlying spinal movement.

As an alternative to invasive treatment methods such as botulinum toxin injections and surgery, ankle-foot-orthoses (AFOs) are commonly prescribed early on in order to allow normal ankle function during gait [178, 179]. Studies on the effects of AFOs on hCP gait reported increased gait speed and step length as well as normalized ankle (first and second rocker), knee and hip motion during stance [43, 178-183]. In addition, wearing AFOs reduced energy cost during walking [180, 182]. Regarding general trunk and upper limb kinematics, AFOs were not considered to cause any clinically relevant changes [43]. Schmid et al. [127] earlier described that the measurements of general trunk motion during gait in patients with adolescent idiopathic scoliosis (AIS) did not show any clinically relevant alterations from healthy controls, whereas the specific measurement of spinal curvature angles, using the previously introduced [47] and validated “IfB marker set” [48, 126], revealed several differences between the two groups. It is therefore assumed that the methods applied by Schweizer et al. [43] might not have been sensitive enough to capture possible...
effects of AFOs on spinal motion. In order to understand spinal motion in hCP patients, the implementation of more sensitive assessment methods is therefore of high importance. Using the “IfB marker set”, the primary aim of this study was to quantify spinal gait kinematics in patients with spastic hCP and to investigate the immediate effects of an AFO and orthotic leg-length correction on spinal kinematics in this population. As a secondary outcome, spatio-temporal gait parameters, lower extremity joint and pelvis angles as well as general trunk movements were calculated to support the interpretation of the primary outcomes.

6.2. Methods

6.2.1. Study population

A consecutive sample of ten hCP patients and a group of 15 healthy adolescents (Table 6.1) participated in this study. Inclusion criteria for the patients were: aged between 10 and 18 years, diagnosis of spastic hCP (GMFCS levels I and II) and an initial toe contact on the affected side, able to walk at least 50 meters without any assistive device, no diagnosed structural deformities of the spine, no previous surgical or casting treatments of the foot as well as botulinumtoxin treatments within the preceding 6 months and no injuries of the locomotor system which led to persistent deformities.

All participants as well as their legal guardians signed informed consent and the study protocol was approved by the local ethics committee.

Table 6.1: Demographics of the hemiplegic CP patients and healthy controls expressed in mean, standard deviation (SD) and range in brackets. Group comparisons were conducted using independent samples T-tests.

<table>
<thead>
<tr>
<th></th>
<th>Hemiplegic CP patients (n = 10)</th>
<th>Controls (n = 15)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years]</td>
<td>12.0 SD 1.4 (10-14)</td>
<td>14.1 SD 1.7 (12-17)</td>
<td>0.003*</td>
</tr>
<tr>
<td>Height [m]</td>
<td>1.61 SD 0.29 (1.37-1.76)</td>
<td>1.62 SD 0.10 (1.46-1.84)</td>
<td>0.901</td>
</tr>
<tr>
<td>Mass [kg]</td>
<td>49.2 SD 18.1 (23.5-76.7)</td>
<td>54.2 SD 10.0 (40.3-70.4)</td>
<td>0.435</td>
</tr>
<tr>
<td>Gender [male/female]</td>
<td>9/1</td>
<td>8/7</td>
<td>-</td>
</tr>
<tr>
<td>Affected side [left/right]</td>
<td>3/7</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>GMFCS level [I/II]</td>
<td>9/1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Hemiplegic type [1/2/3/4]a</td>
<td>4/3/1/2</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*a Hemiplegic type according to Winters et al. [184]

* Statistically significant difference (p≤0.05)
6.2.2. Orthotic correction

The custom made AFOs were constructed as a posterior shell with a joint at the ankle which allowed dorsiflexion but no plantarflexion. An LLD and a fixed equinus were corrected by adding a respective heel lift on the affected side. If the correction overrode the LLD, another heel lift was added on the non-affected side. The shank axis of the AFO was set perpendicular to the sole of the shoe.

6.2.3. Data collection

After a comprehensive physical examination (CP patients only) and the assessment of relevant anthropometric data, participants were equipped with 56 retro-reflective markers (combination of the Plug-in Gait full body [43] and the IfB marker [47] sets) by physiotherapists with experience in clinical gait analysis. Measurements were conducted using a 12-camera motion analysis system (type MXT20, Vicon, Oxford, UK; sampling frequency: 200-300 Hz). Following a static measurement trial in an upright position, participants walked barefoot at their self-selected speed on a 10 m level ground walkway until at least 5 trials were recorded. The CP patients were then fitted with shoes, AFO and heel lifts, underwent another static measurement trial and were asked to walk again until 5 additional trails were recorded.

6.2.4. Data reduction

The software Nexus (version 1.8.5, Vicon, Oxford, UK) was used to set the gait events and to calculate the angles of the affected lower extremity joints and the pelvis according to the Plug-in Gait model, whereas general trunk and spinal data were processed using a custom-built MATLAB-routine (version R2013b, MathWorks Inc., Natick, MA, USA). Thoracic and lumbar curves in the sagittal and frontal planes were defined by the markers placed on the spinous processes of the vertebrae T3, T5, T7, T9 and T11 (thoracic) and T11, L1, L2, L3, L4 and L5 (lumbar), respectively. Circular segments were established and curvature angles were calculated as described previously [127]. General trunk movements in the sagittal, frontal and transverse planes were computed as relative positional angles of rigid lumbar, thoracic and cervical segments between gait and the standing measurement trial [127]. Angles were time normalized to one gait cycle (time between two consecutive foot strikes of the patients’ affected leg, respectively the controls’ left leg) consisting of 101 points and averaged over the five trials per subject.
The average and range of motion (ROM) values during the full gait cycle as well as the values at initial contact (IC) of the thoracic and lumbar spinal curvature angles in the sagittal and frontal planes were defined as primary outcome parameters. In the sagittal plane, positive average values were thereby denoted as kyphosis and negative average values as lordosis. The secondary outcome parameters included: ankle plantarflexion at IC, average ankle plantarflexion during the stance and swing phases, maximum ankle plantarflexion during the swing phase, average and ROM values of the pelvis as well as ROM values of the lumbar, thoracic and cervical segment angles in three dimensions during the full gait cycle and the spatio-temporal gait parameters (expressed in dimensionless numbers according to Hof [132]).

**6.2.5. Statistical analyses**

Statistical tests were conducted using the software packages SPSS 21 (SPSS Inc., Chicago, IL, USA) and G*Power 3.1.9.2 [133]. Normal distribution for the vast majority of the parameters was verified using the Shapiro-Wilk test (p>0.05). Demographics of the hCP patients and healthy controls were compared using independent samples T-tests (p≤0.05). To test for overall differences in the kinematic and spatio-temporal parameters among the hCP (barefoot), hCP (orthosis) and healthy control groups, one-way analyses of variance (ANOVA) and effect sizes (Cohen’s f) were used. Parameters showing considerable effects (f≥0.1) [168] were further explored using Tukey’s post hoc tests and effect sizes of the pairwise mean differences (Cohen’s d). In order to distinguish between clinically relevant and clinically not relevant findings, the 95% confidence intervals (CI) of the respective mean differences were considered in regard to the minimal clinical important differences (MCID) (Figure 6.1) as previously described [127]. Differences were thereby categorized as A) clinically relevant (CI above MCID), B) most likely clinically relevant (MCID within CI but large effect (d≥0.8)), C) probably not clinically relevant (MCID within CI and small to moderate effect (0.2≤d<0.8)) and D) clinically not relevant (CI below MCID).
6.3. Results

The subjects in the control group were on average 2.1 years older than the hCP patients (Table 6.1). Furthermore, the majority of the hCP patients were male and classified as level I on the GMFCS as well as type 1 and 2 according to Winters et al. [184].

6.3.1. Primary outcomes

The analysis of the sagittal curvature angles indicated clinically relevant increases at IC and for the average and ROM values of the lumbar spine as well as decreases at IC and for the average values of the thoracic spine in both the barefoot and orthosis conditions compared to the controls (Table 6.2) (Figure 6.1). In the frontal plane, lumbar and thoracic curvature angles in the barefoot and orthosis conditions showed increases in ROM as well as sideward tilting towards the non-affected (lumbar) and affected (thoracic) sides. No clinically relevant changes in curvature angles were indicated between the barefoot and orthosis conditions. For illustrative purposes, a graphical representation of the curvature angles over time is provided in Figure 6.2.

6.3.2. Secondary outcomes

The ankle angles showed clinically relevant increases for plantarflexion at IC and during swing in the barefoot condition as well as decreased peak plantarflexion during the swing phase in the orthosis condition compared to the control group (Table 6.3) (Figure 6.1). The orthotic corrections caused clinically relevant decreases for ankle plantarflexion at IC and during the swing phase (average and peak values). The pelvis, lumbar, thoracic and cervical segmental angles showed several clinically relevant differences (mostly increases) between the hCP patients and the control group, but not between the two hCP conditions. A complete set of lower extremity and trunk motion graphs can be found in Figures D.1 and D.2 (Appendix D). Spatio-temporal gait parameters showed no clinically relevant differences between groups and conditions.
Table 6.2: Primary outcome parameters: reported are mean, standard deviation (SD) and 95% confidence intervals (within the brackets) for the average (AVG) and range of motion (ROM) values over one gait cycle as well as the values at initial contact (IC) of the spinal curvature angles (expressed in degrees) in the sagittal (Sag.) and frontal (Fron.) planes in hemiplegic cerebral palsy patients (hCP) and healthy controls. Data of the patients (gait cycle of the affected leg) are presented for a barefoot and an orthosis condition. In addition, results for the group comparisons (one-way analyses of variance (ANOVA) and Tukey’s post hoc tests as well as effect sizes (Cohen’s f and d, respectively) are presented.

<table>
<thead>
<tr>
<th>Segment</th>
<th>1) hCP patients (barefoot)</th>
<th>2) hCP patients (orthosis)</th>
<th>3) Controls</th>
<th>ANOVA</th>
<th>Post hoc (Tukey) 3 vs. 2</th>
<th>1 vs. 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sag. AVG</td>
<td>26.2 SD 11.2 (18.2,34.1)</td>
<td>25.3 SD 11.5 (17.1,33.5)</td>
<td>34.1 SD 7.8 (29.8,38.5)</td>
<td>0.42</td>
<td>0.061</td>
<td>-0.85</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>6.0 SD 3.1 (3.8,8.3)</td>
<td>5.4 SD 2.0 (4.0,6.8)</td>
<td>3.2 SD 1.5 (2.4,4.0)</td>
<td>0.57</td>
<td>0.008</td>
<td>1.24</td>
</tr>
<tr>
<td>Sag. IC</td>
<td>26.9 SD 11.0 (19.0,34.8)</td>
<td>26.1 SD 10.9 (18.3,33.9)</td>
<td>34.6 SD 7.8 (30.3,38.9)</td>
<td>0.41</td>
<td>0.064</td>
<td>-0.84</td>
</tr>
<tr>
<td>Fron. AVG</td>
<td>0.8 SD 4.4 (-2.4,3.9)</td>
<td>0.6 SD 4.0 (-2.2,3.5)</td>
<td>3.5 SD 3.2 (1.7,5.3)</td>
<td>0.37</td>
<td>0.121</td>
<td>-0.73</td>
</tr>
<tr>
<td>Fron. ROM</td>
<td>11.2 SD 4.9 (7.7,14.7)</td>
<td>11.3 SD 4.1 (8.4,14.3)</td>
<td>8.2 SD 3.6 (6.2,10.2)</td>
<td>0.36</td>
<td>0.124</td>
<td>0.72</td>
</tr>
<tr>
<td>Fron. IC</td>
<td>-4.5 SD 5.6 (-8.4,-0.4)</td>
<td>-3.8 SD 4.6 (-7.1,-0.5)</td>
<td>-0.1 SD 3.6 (-2.1,1.9)</td>
<td>0.45</td>
<td>0.044</td>
<td>-0.98</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVG</td>
<td>-40.5 SD 12.7 (-49.6,-31.4)</td>
<td>-39.2 SD 13.1 (-48.6,-29.8)</td>
<td>-25.2 SD 11.9 (-31.7,-18.6)</td>
<td>0.58</td>
<td>0.007</td>
<td>-1.25</td>
</tr>
<tr>
<td>ROM</td>
<td>13.4 SD 4.7 (10.0,16.8)</td>
<td>12.7 SD 5.6 (8.7,16.7)</td>
<td>6.2 SD 2.0 (5.1,7.3)</td>
<td>0.83</td>
<td>&lt;0.001</td>
<td>2.16</td>
</tr>
<tr>
<td>IC</td>
<td>-36.9 SD 13.0 (-46.2,-27.5)</td>
<td>-35.8 SD 12.7 (-44.9,-26.7)</td>
<td>-24.1 SD 11.9 (-30.7,-17.5)</td>
<td>0.49</td>
<td>0.026</td>
<td>-1.04</td>
</tr>
<tr>
<td>Lumbar Curves</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVG</td>
<td>4.3 SD 4.3 (1.3,7.4)</td>
<td>3.0 SD 5.3 (-0.8,6.8)</td>
<td>-0.4 SD 3.3 (-2.2,1.5)</td>
<td>0.49</td>
<td>0.025</td>
<td>1.26</td>
</tr>
<tr>
<td>ROM</td>
<td>19.1 SD 8.3 (13.2,25.0)</td>
<td>19.8 SD 10.4 (12.4,27.2)</td>
<td>12.6 SD 3.9 (10.4,14.7)</td>
<td>0.45</td>
<td>0.039</td>
<td>1.08</td>
</tr>
<tr>
<td>IC</td>
<td>4.8 SD 5.3 (1.0,8.6)</td>
<td>3.5 SD 5.8 (-0.7,7.6)</td>
<td>-1.3 SD 3.3 (-3.1,0.6)</td>
<td>0.58</td>
<td>0.007</td>
<td>1.45</td>
</tr>
</tbody>
</table>

Positive AVG and IC values: kyphosis (sagittal), non-affected side (frontal); negative AVG and IC values: lordosis (sagittal), affected (frontal)
Figure 6.1: Results of the pairwise group comparisons (absolute values of the mean differences with 95% confidence intervals) for the spinal curvature angles, ankle joint and segmental trunk angles (expressed in degrees) as well as spatio-temporal gait parameters (expressed in dimensionless numbers). The dashed horizontal lines represent the minimal clinical important differences (MCID). Differences were categorized as A) clinically relevant, B) most likely clinically relevant, C) probably not clinically relevant and D) clinically not relevant. AVG: average; ROM: range of motion; IC: initial contact; PF: plantarflexion.
Figure 6.1: continued
Table 6.3: Secondary outcome parameters: reported are mean, standard deviation (SD) and 95% confidence intervals (within the brackets) for the sagittal ankle joint angles, the average (AVG) and range of motion (ROM) angles of the pelvis, lumbar, thoracic and cervical segments in the sagittal (Sag.), frontal (Fron.) and transverse (Tran.) planes (all angles expressed in degrees) as well as the spatio-temporal gait parameters (expressed as dimensionless numbers) in hemiplegic cerebral palsy (hCP) patients and healthy controls. Data of the patients (gait cycle of the affected leg) are presented for a barefoot and an orthosis condition. In addition, results for the group comparisons (one-way analyses of variance (ANOVA) and Tukey’s post hoc tests as well as effect sizes (Cohen’s f and d, respectively) are presented.

<table>
<thead>
<tr>
<th></th>
<th>hCP patients (barefoot)</th>
<th>hCP patients (orthosis)</th>
<th>Controls</th>
<th>ANOVA</th>
<th>Post hoc (Tukey)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value at IC</td>
<td>Value at IC</td>
<td>f</td>
<td>p</td>
<td>3 vs. 1</td>
</tr>
<tr>
<td>Ankle (affected side)</td>
<td>-18.4 SD 11.3 (-26.5,-10.3)</td>
<td>-5.9 SD 6.8 (-10.8,-1.0)</td>
<td>-1.9 SD 4.4 (-4.3,0.5)</td>
<td>0.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AVG in stance</td>
<td>-0.4 SD 10.4 (-7.9,2.1)</td>
<td>-0.3 SD 6.6 (-6.5,5.9)</td>
<td>3.2 SD 3.0 (1.5,4.9)</td>
<td>0.24</td>
<td>0.38</td>
</tr>
<tr>
<td>AVG in swing</td>
<td>-15.3 SD 11.9 (-23.8,-6.8)</td>
<td>-6.2 SD 1.6 (-10.6,-1.18)</td>
<td>-7.3 SD 3.4 (-12.2,-5.4)</td>
<td>0.51</td>
<td>0.017</td>
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<tr>
<td>Peak PF in swing</td>
<td>-25.8 SD 13.0 (-35.1-16.5)</td>
<td>-9.9 SD 5.6 (-13.9,5.9)</td>
<td>-28.3 SD 6.8 (-32.0,-24.5)</td>
<td>0.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pelvis Segment (affected side)</td>
<td>AVG</td>
<td>13.2 SD 6.1 (8.9,17.5)</td>
<td>12.2 SD 6.1 (7.8,16.6)</td>
<td>6.9 SD 2.6 (5.5,8.4)</td>
<td>0.59</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>6.5 SD 2.1 (5.0,8.0)</td>
<td>6.9 SD 1.5 (5.8,8.8)</td>
<td>3.3 SD 0.7 (2.9,3.7)</td>
<td>1.17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pelvis Segment (affected side)</td>
<td>AVG</td>
<td>10.1 SD 3.6 (7.5,12.7)</td>
<td>10.3 SD 3.6 (7.7,12.9)</td>
<td>9.6 SD 2.9 (8.0,11.2)</td>
<td>0.09</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>16.9 SD 4.5 (13.7,20.1)</td>
<td>16.8 SD 4.1 (13.9,19.8)</td>
<td>10.1 SD 3.8 (8.0,12.2)</td>
<td>0.82</td>
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<td>Lumbar Segment</td>
<td>AVG</td>
<td>7.3 SD 2.2 (5.7,8.9)</td>
<td>6.6 SD 1.9 (5.2,8.0)</td>
<td>3.4 SD 1.1 (2.8,4.0)</td>
<td>1.04</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>6.4 SD 2.1 (5.0,7.9)</td>
<td>6.4 SD 2.2 (4.8,8.0)</td>
<td>5.3 SD 2.1 (4.1,6.5)</td>
<td>0.26</td>
<td>0.31</td>
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<tr>
<td>Thoracic Segment</td>
<td>AVG</td>
<td>9.8 SD 3.5 (7.3,12.3)</td>
<td>10.4 SD 4.3 (7.3,13.5)</td>
<td>5.8 SD 1.8 (4.8,6.9)</td>
<td>0.67</td>
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<tr>
<td>Sag. ROM</td>
<td>6.1 SD 2.5 (4.3,7.9)</td>
<td>6.8 SD 2.4 (5.1,8.6)</td>
<td>3.5 SD 1.1 (2.1,3.9)</td>
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<td>&lt;0.001</td>
</tr>
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<td>Sag. ROM</td>
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<td>10.4 SD 4.3 (7.3,13.5)</td>
<td>5.8 SD 1.8 (4.8,6.9)</td>
<td>0.67</td>
<td>0.002</td>
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<td>Lumbar Segment</td>
<td>AVG</td>
<td>7.1 SD 2.6 (5.2,8.9)</td>
<td>8.6 SD 2.7 (6.7,10.6)</td>
<td>3.4 SD 1.4 (2.7,4.2)</td>
<td>1.04</td>
</tr>
<tr>
<td>Sag. ROM</td>
<td>10.9 SD 4.3 (7.8,14.0)</td>
<td>12.4 SD 6.4 (7.8,16.9)</td>
<td>6.5 SD 2.7 (5.0,8.0)</td>
<td>0.59</td>
<td>0.007</td>
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Continued on next page
Table 6.3: continued

<table>
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<tr>
<th></th>
<th>1) hCP patients (barefoot)</th>
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<th>ANOVA f</th>
<th>ANOVA p</th>
<th>ANOVA d</th>
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<td>0.013</td>
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<tr>
<td><strong>Step time</strong></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td>0.55</td>
<td>0.011</td>
<td>-0.52</td>
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<tr>
<td><strong>Affected side</strong></td>
<td>0.41 SD 0.04 (0.38,0.43)</td>
<td>0.45 SD 0.04 (0.42,0.48)</td>
<td>0.43 SD 0.04 (0.41,0.46)</td>
<td>0.38</td>
<td>0.062</td>
<td>-0.50</td>
<td>0.248</td>
<td>0.50</td>
<td>0.557</td>
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</tr>
<tr>
<td><strong>Non-affected side</strong></td>
<td>33.3 SD 1.3 (32.4,34.2)</td>
<td>32.2 SD 1.3 (31.3,33.2)</td>
<td>33.7 SD 1.5 (32.9,34.5)</td>
<td>0.45</td>
<td>0.040</td>
<td>-0.28</td>
<td>0.698</td>
<td>-1.05</td>
<td>0.032</td>
<td>-0.85</td>
</tr>
<tr>
<td><strong>Affected side</strong></td>
<td>0.72 SD 0.06 (0.68,0.77)</td>
<td>0.82 SD 0.08 (0.76,0.88)</td>
<td>0.76 SD 0.07 (0.72,0.79)</td>
<td>0.54</td>
<td>0.013</td>
<td>-0.60</td>
<td>0.538</td>
<td>0.81</td>
<td>0.070</td>
<td>1.41</td>
</tr>
<tr>
<td><strong>Non-affected side</strong></td>
<td>0.71 SD 0.07 (0.66,0.76)</td>
<td>0.82 SD 0.08 (0.76,0.88)</td>
<td>0.75 SD 0.08 (0.70,0.79)</td>
<td>0.55</td>
<td>0.011</td>
<td>-0.52</td>
<td>0.512</td>
<td>0.87</td>
<td>0.064</td>
<td>1.46</td>
</tr>
<tr>
<td><strong>Affected side</strong></td>
<td>1.88 SD 0.09 (1.81,1.94)</td>
<td>1.94 SD 0.10 (1.87,2.02)</td>
<td>1.78 SD 0.08 (1.74,1.83)</td>
<td>0.77</td>
<td>&lt;0.001</td>
<td>1.19</td>
<td>0.040</td>
<td>1.81</td>
<td>&lt;0.001</td>
<td>0.63</td>
</tr>
<tr>
<td><strong>Non-affected side</strong></td>
<td>1.74 SD 0.10 (1.67,1.81)</td>
<td>1.79 SD 0.08 (1.74,1.85)</td>
<td>1.78 SD 0.08 (1.74,1.82)</td>
<td>0.24</td>
<td>0.313</td>
<td>-0.45</td>
<td>0.446</td>
<td>0.13</td>
<td>0.923</td>
<td>0.55</td>
</tr>
</tbody>
</table>

Positive AVG and IC values: dorsiflexion (sagittal ankle), anterior tilt (sagittal pelvis), upward movement (frontal pelvis), internal rotation (transverse pelvis); negative AVG and IC values: plantarflexion (sagittal ankle), posterior tilt (sagittal pelvis), downward movement (frontal pelvis), external rotation (transverse pelvis)
Figure 6.2: Curvature angles of the lumbar and thoracic curves in the sagittal and frontal planes over one complete gait cycle of the patients’ affected and the controls’ left leg. The grey solid lines represent the healthy controls, whereas the black solid and dashed lines represent the hCP patients in the barefoot and AFO conditions, respectively (thick lines = means, thin lines = standard deviations).

6.4. Discussion

The current study aimed at the immediate effects of a unilateral AFO and orthotic leg-length correction on spinal curvature angles during gait in hCP patients. The results showed that the AFO was able to restore a heel-to-toe gait pattern, but not to influence spinal or general trunk kinematics, despite the correction of any LLD. Differences in spinal curvature or rigid segmental angles were only found between the patient and the control groups. The observed increases in the average and ROM values of lumbar lordosis angles most likely resulted as a logical consequence of the pelvic deviations in the sagittal plane. In the thoracic spine, hCP patients presented hypokyphosis, similar to patients with AIS [127]. Furthermore, the spine showed no compensatory reactions to pelvic retraction such as reduced rotation of the rigid trunk segments. It is therefore plausible that pelvic retraction was compensated by a
rotation in the upper cervical region. The sideward tilting tendency of the lumbar and thoracic spine was observed even in the absence of any pelvic frontal plane deviations that could have resulted from unequal leg length. A possible LLD in the barefoot condition was probably compensated for by the equinus foot [185]. The sideward tilting tendency of the spine might therefore be explained as a concomitant effect resulting from the combination of pelvic anterior tilt and pelvic retraction or as a simple projection error due to a non-dynamic lumbar spine coordinate system. Since none of these deviations changed when walking with the orthotic corrections, however, it seems that the spinal gait deviations found in these patients were not secondary effects of the equinus foot or a possible structural LLD. This mostly supports the research of Schweizer et al. [43], who investigated the effects of an AFO on upper body gait kinematics in a similar cohort of hCP patients. Compared to our study, they found very similar ankle angles as well as increased ROM values in general trunk parameters that were not affected by wearing an AFO. They concluded that restoring the first ankle rocker had no clinically relevant effects on general trunk kinematics and hence none of the observed upper body gait deviations seemed to be of secondary nature in patients who regularly wore an AFO [43].

In contrast, Brunner et al. [2] reported that excessive plantar flexion was responsible for flexion, internal rotation and adduction at the hip as well as pelvic retraction and pelvic upward motion using a forward dynamic modelling approach. Furthermore, Goodman et al. [46] found an increased anterior pelvic tilt in a group of healthy subjects after constraining one ankle into equinus using a unique taping method. And finally, Stebbins et al. [1] suggested that pelvic rotation was a compensation for foot deformity after measuring hCP patients prior to and following foot correction surgery.

A possible explanation for these contradictory findings might derive from Renshaw et al. [186], who linked hyperlordosis in CP patients with hip flexor contractures. The question thereby arises, whether hip flexor contractures could be a result of the currently observed pelvic deviations? Lee et al. [187] previously reported that hip flexion contractures seem to be generally caused by immobility and lack of stretching rather than by the initial disease or any directly associated impairment. Since walking with an increased pelvic retraction and pelvic anterior tilt might engender a lack of stretching of the hip flexors, this explanation becomes entirely plausible. Taking into account that hip flexor contractures are known to be correlated with increased anterior pelvic tilt and lumbar lordosis as well as increased pelvic motion in the frontal plane [78, 187], it can be assumed that the currently observed pelvic and lumbar spine deviations might actually have been a direct result of hip flexor contractures and not of
foot deformity. This assumption concurs with the observations of Stebbins et al. [1], who also found no changes in anterior pelvic tilt following foot correction surgery. It can therefore be postulated that the observed pelvic deviations are structurally fixed (permanent) secondary gait deviations that initially resulted from an equinus foot deformity. Brunner et al. [2] confirmed the result of their forward modeling study using a group of hCP patients. Goodman et al. [46], however, did not involve patients and both did not consider long-term adaptations of the structures, which might explain their contradictory findings. Reasons for the presence of secondary mechanisms in the patient group of Stebbins et al. [1] might be that their patients were slightly younger than those observed in the current study and might therefore have not yet developed permanent secondary deviations. In addition, factors such as different treatment protocols, patient compliance and severity of disease might have played an important role. Attias et al. [30], for example, reported that greater levels of impairment in hCP were associated with greater ROM of the thorax. Therefore, more severe cases of foot deformity might show more pronounced effects on spinal kinematics following a corrective treatment, even in the presence of hip flexor contractures, whereas less severe cases might exhibit little or no measurable changes. Unfortunately, this information was not provided by the mentioned study [1] and remains therefore speculative.

Another way of looking at the fact that the AFO did not influence spinal kinematics is that all patients were used to walking with an AFO and might have already adapted their barefoot walking patterns in a way that possible immediate effects of an AFO on spinal kinematics would no longer be visible [43]. It remains unknown how the locomotor system of these patients would have developed without wearing AFOs. This issue might be followed up in future studies by comparing spinal kinematics between patients with an obligatory unilateral toe-walking pattern and mimicking healthy subjects. Romkes et al. [55], for example, used this approach to distinguish between primary and secondary abnormalities in the activity of lower extremity muscles in hCP patients.

In future research and clinical practice, the distinction between primary and secondary deviations in hCP patients remains important for treatment planning, since only primary deviations should be treated [144]. Once a primary deviation is treated, secondary problems often resolve spontaneously [1]. Therefore, secondary effects should only be addressed in a preventive manner or if present as a fixed deviation, since any other treatment would most likely be not effective or even counterproductive.

A limiting factor of the current study includes the calculation of the lumbar curvature angles in relation to the global coordinate system. Considering the generally increased ROM of the
trunk segments, especially in the transverse plane, a static coordinate system might have caused projection errors. However, the definition of a dynamic coordinate system, such as applied for the thoracic spine, was not possible due to a missing meaningful anterior reference point. In addition, since most parameters were considered not fully conclusive (most likely clinically relevant and probably not clinically relevant), data of larger samples are required. Furthermore, the fact that the AFO blocked plantarflexion during the push-off phase was considered another limitation.

6.5. Conclusions

Spinal gait deviations in adolescent patients with mild forms of hCP seemed to occur not as a secondary effect of a spastic equinus foot or a LLD but more likely of proximal abnormalities such as hip flexor contractures, which might have been long-term structural adaptations due to passive secondary effects of foot deformity. The question remains, however, whether an orthotic treatment of the lower extremities in younger patients and/or more severe cases of hCP would have different effects on spinal kinematics. In addition, all patients were used to walking with an AFO and might therefore have already adapted their barefoot walking pattern such that possible effects of an AFO have been diluted. Future research should consider investigating long-term effects of an AFO treatment as well as the relation between spinal kinematics and severity of disease.
7. Discussion

Although a detailed knowledge of spinal kinematics during activities of daily living such as walking is of high importance for a better understanding of pathological mechanisms and evaluation of treatment effects, such evidence is lacking throughout the scientific literature. For this reason, the previously developed IfB marker model was introduced into clinical gait analysis, aiming at an understanding of normal spinal motion with respect to age as well as to the biomechanical behavior of the spine in patients with a primary spinal pathology such as AIS and at the effects of a lower extremity orthotic treatment on secondary spinal deviations in patients with hemiplegic CP.

As previously pointed out, being able to distinguish between primary and secondary gait deviations is crucial for treatment planning. Therefore, a comprehensive review of the literature was conducted in the forefield of this thesis, aiming at the identification of secondary mechanisms in pathologic gait (Chapter 2). The search resulted in 36 studies, whereof 31 studies were categorized as describing active (compensations) and 5 studies passive secondary mechanisms (physical effects). In addition, the analysis revealed several compensatory strategies that were reported for different patient populations and that therefore appeared to be independent from the underlying disease. However, only about 20% of the identified studies included an analysis of trunk motion, using only minimal marker configurations that did not allow any conclusions on the kinematic behavior of the spine. These findings clearly highlighted the importance for further research in the field of secondary gait deviations, especially involving the analysis of spinal kinematics.

In order to better understand normal spinal motion, 42 healthy individuals were categorized as adolescents, adults and elderly and analyzed using the IfB marker model while walking at a self-selected speed (Chapter 3). The results mainly suggested that during the adolescent growth spurt, lumbar lordosis and thoracic kyphosis increase in magnitude, reach their peak in adulthood and flatten again in the course of life’s third trimester. These observations can be ascribed to the age-related physiological changes of spinal morphology. Furthermore, this study highlighted the importance of considering kinematic results always in relation to spatio-temporal gait properties and the fact that investigations involving comparisons of data acquired in different laboratories and by different examiners might be subjected to additional error. These findings can serve as a normative basis for future explorations on pathologies affecting the spine.
Prior to stepping in this direction, however, an investigation on the validity of the IfB marker model for the quantification of spinal motion in subjects with pathology was required (Chapter 4). Thereby, ten patients with main thoracic AIS were examined using standard biplanar radiography with radio-opaque markers placed on the designated spinous processes. The results demonstrated that in the sagittal plane, spinal curvature can be assessed with reasonable accuracy. Due to the distinctive rotational deformities of the scoliotic vertebrae, however, marker-derived measurements indicated a systematic underestimation of the frontal plane curvature angles and should therefore always be interpreted accordingly. Marker placement accuracy was acceptable when carried out by a healthcare professional with experience in palpation. Considering the limitations given by the non-invasive nature of the approach, the results suggested that the IfB marker model can be used for the assessment of spinal motion in subjects with spinal deformities.

Following up on this, Chapter 5 reports on a study investigating spinal motion during gait in a cohort of 14 AIS patients using the IfB marker model. Compared to a group of 15 healthy adolescents, sagittal and frontal plane deformations could be observed in the scoliosis patients as expected. In addition, the patients showed altered motion patterns for the thoracic spine in the sagittal and for the lumbar spine in the frontal plane. When evaluating these patients using standard trunk motion parameters such as the relative angles between a rigid thorax and pelvis segment, the two groups appeared to be no different from each other. This is the first study providing data on the primary effects of AIS on the actual spinal motion during gait, which might contribute to a better understanding of the pathogenesis of AIS as well as to the prevention of future complications and the improvement of current treatment methods. Furthermore, the findings clearly emphasize the fact that advanced optical motion capture approaches such as the IfB marker model are required when investigating within-trunk or spinal motion.

Chapter 6 describes the analysis of spinal gait deviations in a group of 10 adolescents with hemiplegic CP. Based on previous research, it was assumed that these deviations might be caused secondarily by foot equinus and LLD and would resolve spontaneously once lower extremity function was restored. In order to test this assumption, patients were measured barefoot and with orthotic corrections of foot equinus by an AFO and LLD by a heel lift while walking at a self-selected speed. The results showed that although the orthotic corrections were able to restore the first rocker enabling a normal heel-to-toe gait, spinal motion was not influenced thereby. Differences were only found in comparison to a group of 15 healthy adolescents. It was therefore suggested that spinal gait deviations in adolescents with mild
hemiplegic CP are not directly caused by foot equinus or LLD anymore, but probably by more proximal issues such as hip flexor contractures resulting as a long-term effect of foot equinus or LLD during the childhood. These findings provide a deeper understanding of spinal gait deviations in adolescent hemiplegic CP patients, which is a vital component in the prevention of complications in the adulthood as well as for a more effective treatment planning.

This doctoral thesis can be considered a first step towards a more detailed understanding of the pathomechanics of the spine in dynamic everyday living situations. The results of the different experimental studies clearly demonstrate the potential of an advanced optical motion capture approach. Especially the fact that commonly used trunk motion parameters seem not to be useful when investigating spinal pathologies highlights the need for advanced methods. When looking at the literature investigating gait deviations of the trunk using standard marker models, it is therefore highly likely that possible compensatory mechanisms of the spine might have been missed. Moreover, studies that found compensations of the trunk might not have been able to fully explain these mechanisms due to lacking information on spinal motion. For example, several authors reported that hip abductor weakness was compensated by a lateral trunk lean, also known as a Duchenne limp [35, 77, 78, 82]. The goal of this mechanism is to reduce the demand for frontal plane moment produced by the hip abductor muscles during the stance phase by shifting the center of mass towards the affected side. It has been shown, however, that a Duchenne limp can be carried out in several different ways [188]. In case of irreversible pathologies such as myelomeningocele [35, 77], the goal of a treatment might not be a correction of a Duchenne limp by addressing the primary pathology but more likely the prevention of future complications due to the pathologic gait pattern. Hence, detailed information on the biomechanical behavior of the spine is crucial for the development of adequate and effective preventive strategies such as customized exercise programs.

A profound knowledge of spinal gait kinematics could further contribute to a better understanding of the pathological mechanisms that lead to the development of complex disorders such as AIS. Ongoing research on AIS focuses mainly on etiological factors of the disease. It has been shown, for example, that the development of AIS might be associated with different genes [189, 190], certain hormones contributing to postural control anomalies [191] as well as morphological vestibular asymmetries [192]. However, beyond etiology,
clinicians involved in the treatment of developing AIS would benefit more from advanced knowledge of the pathogenesis of AIS and consequent prognostic factors of disease progression, since this could lead to a reduction of repetitive exposure to radiation, unnecessary brace treatments, psychological implications, and costs-of-care related to follow-up in low-risk patients [193].

The results of the current thesis might provide valuable information in this direction, by suggesting that the previously reported rotational instability, caused by a relative anterior spinal overgrowth [172], could be responsible for an increased flexion-extension movement of the thoracic spine. This increased dynamic behavior might result in greater axial loads during the reversal of movement direction and thus an increased lateral force component, driving the apical vertebral body out of the midline [194]. It is therefore not farfetched to postulate that the degree of rotational instability might be positively related to the magnitude of sagittal ROM of the thoracic spine. The degree of rotational instability might therefore be a possible predictive factor for curve progression, quantified indirectly by the non-invasive measurement of sagittal thoracic ROM in AIS patients. However, these conclusions would be premature with the current state of evidence and are thus subject to further investigation. In addition, since the degree of kyphosis has been related to curve progression [172], the assessment of thoracic kyphosis during gait might be another valid non-invasive predictive parameter for curve progression.

The knowledge on spinal gait kinematics in AIS patients might also contribute to a better understanding of the gait pattern in this population. It is very likely that the spinal motion pattern observed in AIS patients during gait has secondary implications on pelvis as well as lower and upper extremity joint motion. Further studies might therefore include correlation analyses investigating the relationship between spinal kinematics and pelvis, lower and upper extremity joint motion.

Further implications of a deeper knowledge of spinal kinematics are the understanding and consequently the prevention of mechanisms leading to secondary complications such as back pain. Sato et al. [195], for example, reported for AIS patients significantly more severe pain in the upper and middle right back with a longer duration and more recurrences as for healthy adolescents. Considering that the disproportional growth of the anterior and posterior vertebral elements in AIS patients might result in rotational segmental instability (see above), an instability-related uncoordinated hyperactivity of the paraspinal muscles during gait could be a plausible explanation for these complications. For this reason, a possible strategy for
back pain prevention in this population might be the stabilization of passive rotational instability by specific stabilization exercises. In the lumbar spine, for example, segmental instability has been previously linked to the occurrence of low back pain [196]. In terms of treatment, specific core stabilization exercises in addition to general exercises were thereby more efficient than general exercises alone [197]. Moreover, several authors describe the occurrence of chronic musculoskeletal pain in adult CP patients with prevalence rates around 65% for pain lasting at least 3 months (versus 23% in the European general population [198]) and the most common pain site being the (low) back (59-72%) [6, 199-201]. In addition, prevalence rates were shown to increase significantly with advancing age [200, 201]. Patients with deterioration in walking function further reported significantly increased pain frequency, more pain sites, higher pain intensity, and more impact of pain on daily life and activities [201]. Considering the different types of CP, prevalence rates seem not to differ between unilateral and bilateral CP [6]. For patients with hemiplegic CP, the occurrence of back pain was reported to be 47% [200]. These numbers show that chronic low back pain is significantly more frequent in patients with CP compared to the general population and within CP patients the most observed musculoskeletal complication. Since pain in this population has been recently linked to lower activity levels and subsequently to a higher risk for cardiometabolic complications, fragility and/or a higher mortality rate [7], prevention, especially of low back pain, seems therefore highly indicated. In addition, common physical pain treatment methods were shown to be only moderately effective [6]. The analysis of spinal gait kinematics in hemiplegic CP patients in the context of this thesis suggested that the observed spinal deviations might be caused by a hip flexor contracture, which was hypothesized to be a long-term effect of foot equinus or LLD. Considering that increased psoas muscle tension (e.g. due to spasticity) was identified as a possible cause of low back pain in CP patients [202], it could be further hypothesized that an early prevention of hip flexor contractures might prevent the later development of low back pain in this population. However, based on the current state of evidence, such considerations are subject to speculation. Further research investigating the effects of foot equinus and LLD on spinal kinematics in younger cohorts (i.e. children with ages of <10 years) with hemiplegic CP is indicated. These examples show that a comprehensive understanding of pathological gait patterns by using advanced non-invasive optical approaches might lead to more successful prevention of secondary complications in subjects with pathologies affecting the spine.
The understanding of pathological mechanisms could benefit further from information on the internal loading conditions of specific vertebral segments in patients with spinal pathologies. Such information could be gathered by using real patient data to drive complex computer simulation models. Yoder et al. [203], for example, recently used the data of individuals with and without unilateral transtibial amputation to guide a muscle-actuated whole body model, which showed that amputation patients were exposed to atypical motion and loading within the trunk-pelvis region and that this could be a possible mechanism for the development of low back pain. However, despite of an enormous increase in the number of studies using musculoskeletal modeling over the past 25 years [204], an appropriate and validated whole body model allowing a detailed analysis of the lumbar as well as thoracic spine during gait is still lacking.

Finally, it can be suggested that advanced optical motion capture approaches such as the IfB marker model should be implemented into standard clinical gait analysis protocols. Clinicians might, however, respond skeptically to such suggestions since costs are always tightly restricted in a clinical environment. The application of additional markers as well as the analysis and interpretation of additional data clearly goes along with an increased time exposure for patients and examiners. However, when considering that a normal clinical gait analysis takes about 1.5-2 hours, while the additional effort for the analysis of spinal motion amount to approximately 10 minutes, it can be argued that the benefits of information on the spine justify the additional investment.

7.1. Limitations

Considering the interpretation of the current results from a critical point of view, some limitations have to be addressed.

Although the validity of spinal curvature measurements was explored, these findings are technically limited to the static assessment of spinal curvature angles in patients with AIS. Other pathologies with different kinds of deformities such as juvenile or neuromuscular scoliosis might, for example, not show the same degree of systematic frontal curvature underestimation. Furthermore, vertebral segments in AIS might show an altered movement behavior and should therefore ideally be validated in a dynamic manner and if possible even during gait. Such dynamic validations could be realized by means of a moving radio-fluoroscopy system, as previously used to assess knee implant kinematics [205]. However,
since these measurements would require exposing the participants to additional radiation, this
method had to be rejected due to ethical considerations. Non-invasive imaging methods such
as magnetic resonance tomography would only allow a static validation of different functional
positions and no dynamic validation. In addition, the patients would have to remain still in
these positions for about 5-10 minutes each in order to get images with an acceptable
resolution that allow the digital reconstruction of the respective structures. For these reasons,
the validation using posterior-anterior and lateral radiographs that were taken in the context of
a standard clinical examination was the best available solution. And moreover, it is assumed
that a static validation sufficed for the measurement of gait kinematics, since the ranges of
spinal motion are rather small during walking. Additional limitations such as the greater soft
tissue thickness in the lumbar region were already discussed in Chapter 4.
The measurements during gait were mainly limited by small sample sizes, the lack of a
dynamic coordinate system for the lumbar spine as well as lacking information on inter-
session reliability. These limitations were, however, already discussed within the respective
studies (Chapters 3, 5 and 6).
The fact that the position of the rigid trunk segments during gait were calculated in relation to
the static measurement trial was considered another limitation. While this method in fact
accounted for a certain amount of marker placement error, no statements on the absolute
positions of these segments could be derived. Therefore, a rotational offset of a certain trunk
segment would not have been detected with this approach. A possible solution for this
problem could be to perform the calculations of the trunk segment positions during gait
against a “standardized norm subject”. However, such a method would introduce additional
assumptions and should therefore first be evaluated in detail.
Finally, since some of the markers in the IfB trunk model, especially in the lumbar region, are
placed very close together, powerful measurement systems, preferably with 12 or more
cameras, are required. Not all gait analysis laboratories are equipped with such material and
therefore, this might restrict the widespread use of such advanced approaches.
8. Outlook

As this thesis highlights the importance of a deeper knowledge on the biomechanics of a healthy and pathological spine in dynamic everyday living activities, the need for further research in this field is undisputed. The following list contains some ideas for future projects.

- Since the evaluation of treatment effects using spinal kinematics might require a repeated marker placement over the course of several days, weeks or months, data on the inter-session / between-day reliability of the IfB trunk model are inevitable. This could be fairly soon achieved in the context of a master thesis or a student semester project by assessing a cohort of healthy young subjects at different time points separated by several days or weeks.

- In order to follow up on the simulation of internal loading conditions of the spine during walking or other functional activities involving the whole body, the gathered data could be used to drive a more sophisticated spine model such as the one developed at our institute in the context of Dominika Ignasiak’s doctoral thesis. It consists of a generic lumbar spine model as well as a newly developed model for the thoracic region, including 12 vertebrae, 10 rib fragments and a sternum [206]. However, despite its advanced capabilities, the current version of the model only allows the isolated analysis of the pelvis-spine complex and has therefore first to be implemented in a whole body model. Even though first attempts in this direction have already been conducted, more work is needed in order to fully achieve such an implementation.

- While the above introduction of a more sophisticated spine model primarily focuses on simulations in a non-deformed spine, a further step would be the adaptation of the model for simulations in pathologically deformed spines such as in AIS. A deformity could thereby be artificially induced or subject-specific spine models could be created based on three-dimensional reconstructions of individual MRI or CT images. This could be realized, for example, by using the host-mesh fitting technique, which allows the rapid morphing of pathological geometries using a few control points that were defined based on a normative dataset [207].

- At the same time, the IfB trunk model could be used as a standard trunk model in clinical gait analysis laboratories. Thereby, data on spinal gait kinematics of several
different patient populations could be fairly easily gathered and subsequently used to drive biomechanical simulation models such as described above.
9. Conclusions

The current work highlights the importance of using a validated advanced optical motion capture approach allowing the quantification of spinal kinematics for a better understanding of normal and pathological gait. Based on an altered spinal motion pattern in AIS, it has been suggested that segmental instability might play a role in the pathogenesis of AIS as well as in the development of secondary complications such as back pain. Furthermore, an unchanged spinal motion pattern following the correction of lower extremity function in hemiplegic CP patients indicated that the direct causes for the observed spinal deviations might over time have changed from primary problems such as foot deformity or LLD to structurally fixed secondary deviations like hip flexor contractures, which were shown to be involved in the development of back pain in this population. These findings could serve as a basis for the derivation of non-invasive predictors of curve progression and back pain in AIS. Furthermore, adequate prevention and treatment protocols can be developed in order to address secondary complications in the above patient populations.

Finally, this doctoral thesis laid the ground for future investigations on pathological mechanisms of the spine during dynamic everyday living tasks such as walking. In order to gain an even deeper insight into the biomechanics of the spine, the data can be used to drive sophisticated computer models allowing the simulation of segmental motion as well as internal spinal loading.
References


References


References


[93] !!! INVALID CITATION !!!


References


References


Appendix A: Included studies in systematic review (Chapter 2)
<table>
<thead>
<tr>
<th>Authors (year)</th>
<th>Diagnosis</th>
<th>Number of subjects (gender)</th>
<th>Age in years: Mean (SD), Range</th>
<th>Gait speed</th>
<th>Parameters evaluated</th>
<th>Side (level) evaluated</th>
<th>Compensatory mechanisms and secondary deviations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkjaer et al. (2000)</td>
<td>Early intensive treatment (at age of 3 months) for unilateral congenital clubfoot</td>
<td>P: 9 (9m/0f) C: 15 (15m/0f)</td>
<td>P: 19.7 (-), 18-23 C: 30 (-), 21-42</td>
<td>P = C (matched)</td>
<td>KM, KT</td>
<td>Affected (ankle, knee, hip)</td>
<td>1) COMP: To keep up forward propulsion in MST, hip flexors compensated for smaller work generated by weak plantarflexors by generating larger eccentric muscle work. 2) COMP: Larger internal knee and hip extensor moments compensated for smaller internal ankle moment throughout ST (PF/KE couple)</td>
</tr>
<tr>
<td>Allen et al. (2000)</td>
<td>Spastic hemiplegic cerebral palsy (CP) with leg-length discrepancy (LLD)</td>
<td>P (LLD &gt; 1.5 cm): 49 (31m/18f) C: 61 (32m/29f)</td>
<td>P: 10.5 (-), 4.1-17.7 C: 9.4 (-), 4.1-15.6</td>
<td>P &lt; C</td>
<td>KM</td>
<td>Unclear (ankle, knee, hip)</td>
<td>COMP: Excessive ankle DF in ST and increased knee and hip flexion throughout the gait cycle on the unaffected side compensated for the functional leg-length discrepancy, produced by increased hip and knee flexion on the affected side.</td>
</tr>
<tr>
<td>Aminian et al. (2003)</td>
<td>Spastic hemiplegic CP (Surgery subset: Proximal femoral derotation osteotomy with concomitant soft tissue surgeries)</td>
<td>P: 71 (41m/30f) (Surgery: 971) C: Normal pediatric database</td>
<td>P: 8.4 (-), 3.9-18.4 C: - (-), -</td>
<td>-</td>
<td>KM</td>
<td>Affected and unaffected (ankle, knee, hip, pelvis)</td>
<td>REACT: In ST, pelvic retraction on the affected side could have resulted from foot internal rotation to achieve slightly external foot progression angle. Pelvic retraction on the affected side also increased the unaffected limb’s step length, which might have been affected by limited hip extension attributed to decreased hip extensor strength on the affected side. The protraction on the unaffected side was balanced by external hip rotation of the hip to maintain a normal foot progression.</td>
</tr>
<tr>
<td>Berchuck et al. (1990)</td>
<td>Unilateral anterior cruciate ligament (ACL) deficiency (5/16: isolated ACL tear, 11/16: ACL tear and a minor meniscal lesion)</td>
<td>P: 16 (14m/2f) C: 10 (5m/5f)</td>
<td>P: 26 (9.5), - C: 26 (5), -</td>
<td>-</td>
<td>KM, KT</td>
<td>Affected and unaffected (knee, hip)</td>
<td>1) COMP: In early ST, increased external hip flexion moment (but not hip flexion angle) was consistent with greater external KE moment that tended to extend the knee at footstrike and thus reduced quadriceps contraction (seen in both legs). 2) COMP: In MST, HS-contraction on the affected side caused co-contraction in order to reduce the net quadriceps moment and thus anterior tibia translation.</td>
</tr>
<tr>
<td>Brunner &amp; Romkes (2008)</td>
<td>Several orthopedic conditions, but no underlying neurological disorders</td>
<td>P: 39 (17m/23f) C children: 13 (6m/7f), 10.1 (-), 7-16 C adults: 32 (-), 25-58</td>
<td>P: 17.8 (-), 6.8-50.4 C adults: 14 (7m/7f), -</td>
<td>-</td>
<td>KM, KT, EMG</td>
<td>Unclear (ankle, knee, hip, pelvis)</td>
<td>General mechanisms for loaded limb stabilization and as compensations for general weakness in ST: 1) COMP: Premature triceps surae activation and a switched-off tibialis anterior caused KE (PF / KE couple) and thus allowed control of second rocker and upright posture. 2) COMP: Co-contraction of knee extensors (for KE) and HS (for hip extension because of two-joint muscle).</td>
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| Bulgheroni et al. (1997) | Symptomatic ACL deficiency                                                | P: 10 (10m/0f)              | P: 27 (6), -                  | P = C      | KM, KT, EMG           | Affected (ankle, knee, hip) | 1) COMP: In loading response and toe-off, increased HS activity (increased external hip flexion moment) caused co-contraction at knee and therefore a reduction in net quadriceps moment (reduced external knee flexion moment).  
2) COMP: In entire ST, knee external rotation instability was compensated for by a lateral shift of the GRF, causing decreased external adduction moments at knee and hip and an increased stability of the lateral compartment of the knee. |
|                        |                                                                           | C: 10 (10m/0f)              | C: 25 (4), -                  |            |                      |                        | 1) COMP: Hip flexors compensated for weak plantar flexors when moving affected leg forward in SW (pulling vs. pushing-off the leg in terminal ST).  
2) COMP: Quadriceps weakness affected the knee pattern in loading response, when patients avoided flexion of the knee. Knee hyperextension later in ST represented an attempt to maintain body stability while compensating for the weak quadriceps. |
| Chen et al. (2003)     | Hemiplegia (6 months after first stroke, 2 groups: poor and good motor stage) | P poor: 17 (7m/10f)         | P poor: 59.4 (14.1), -        | P < C      | KM, KT               | Affected and unaffected (ankle, knee, hip, pelvis) | 1) COMP: In SW, excessive compensatory pelvic up tilt on the unaffected side allowed clearance of the affected foot (insufficient hip and knee flexion and ankle DF).  
2) COMP: Hip flexors compensated for weak plantar flexors when moving affected leg forward in SW (pulling vs. pushing-off the leg in terminal ST). |
|                        |                                                                           | P good: 18 (10m/8f)         | P good: 63.1 (11.2), -        |            |                      |                        | COMP: Knee hyperextension was linked to an increase of the PF/KE couple during MST: the GRF fell in front of the knee and generated an external extensor moment. |
| Cimolin et al. (2007)  | Hereditary spastic paraplegia (HSP) and mild spastic diplegia (SD) secondary to CP | C: 15 (7m/8f)               | C: 58.2 (9.3), -              | P < C      | KM, KT               | Affected (ankle, knee, hip, pelvis) | 1) COMP: Hip hike compensated for reduced hip and knee flexion of the affected limb in SW.  
2) COMP: In late ST, a hip extensor torque strategy ("loading" of the passive hip flexor structures) compensated for impaired plantarflexor strength on the affected side to generate forward propulsion. |
|                        |                                                                           | P HDP: 15 (-)               | P HDP: 10.1 (5.6), -          |            |                      |                        | 1) COMP: Hip flexors compensated for weak plantar flexors when moving affected leg forward in SW (pulling vs. pushing-off the leg in terminal ST).  
2) COMP: Quadriceps weakness affected the knee pattern in loading response, when patients avoided flexion of the knee. Knee hyperextension later in ST represented an attempt to maintain body stability while compensating for the weak quadriceps. |
|                        |                                                                           | P SD: 40 (-)                | P SD: 8.6 (4.3), -            |            |                      |                        | COMP: Knee hyperextension was linked to an increase of the PF/KE couple during MST: the GRF fell in front of the knee and generated an external extensor moment. |
|                        |                                                                           | C: 20 (-)                   | C: 10.5 (5.2), -              |            |                      |                        | 1) COMP: Hip hike compensated for reduced hip and knee flexion of the affected limb in SW.  
2) COMP: In late ST, a hip extensor torque strategy ("loading" of the passive hip flexor structures) compensated for impaired plantarflexor strength on the affected side to generate forward propulsion. |
| Cruz et al. (2009)     | Chronic stroke                                                            | P: 18 (12m/6f)              | C: 51.8 (9.4), -              | P < C      | KM, KT               | Unclear (hip, pelvis) | 1) COMP: Hip hike compensated for reduced hip and knee flexion of the affected limb in SW.  
2) COMP: In late ST, a hip extensor torque strategy ("loading" of the passive hip flexor structures) compensated for impaired plantarflexor strength on the affected side to generate forward propulsion. |
|                        |                                                                           | C: 8 (5m/3f)                | C: 51.8 (9.4), -              |            |                      |                        | COMP: Knee hyperextension was linked to an increase of the PF/KE couple during MST: the GRF fell in front of the knee and generated an external extensor moment. |
| D'Angelo et al. (2009) | Duchenne muscular dystrophy (2 groups: no treatment [NoT], treatment [T]) | P NoT: 11 (11m/0f)          | P NoT: 6.6 (2.8), -           | P = C      | KM, KT               | Unclear (ankle, knee, hip, pelvis) | 1) COMP: Hip hike compensated for reduced hip and knee flexion of the affected limb in SW.  
2) COMP: In late ST, a hip extensor torque strategy ("loading" of the passive hip flexor structures) compensated for impaired plantarflexor strength on the affected side to generate forward propulsion. |
|                        |                                                                           | P T: 10 (10m/0f)            | P T: 7.3 (1.9), -             |            |                      |                        | COMP: Knee hyperextension was linked to an increase of the PF/KE couple during MST: the GRF fell in front of the knee and generated an external extensor moment. |
|                        |                                                                           | C: 10 (10m/0f)              | C: 7.4 (1.2), -               |            |                      |                        | 1) COMP: Hip hike compensated for reduced hip and knee flexion of the affected limb in SW.  
2) COMP: In late ST, a hip extensor torque strategy ("loading" of the passive hip flexor structures) compensated for impaired plantarflexor strength on the affected side to generate forward propulsion. |
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<tr>
<td>Davids et al. (1999)</td>
<td>Spastic diplegic type CP with toe-walking gait pattern (in addition: control group mimicking toe-walking)</td>
<td>P: 15 (-)</td>
<td>P: 8.2 (-), 5.4-13.6</td>
<td>P &lt; C (sign.)</td>
<td>Unclear (ankle, knee, hip, pelvis)</td>
<td>REACT: Anterior pelvic tilt, increased pelvic transverse plane dynamic range of motion and diminished hip extension were identified as secondary deviations for toe-walking gait pattern.</td>
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<td>C: 32 (-)</td>
<td>C: 9.3 (-), 5.4-13.6</td>
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<td>Only foot drop: 1) COMP: In ST, increased passive ankle DF (delayed plantar flexor activation) in coordination with a greater hip extension and an additional antigravitational effort at the knee (prolonged knee extensor activity) was necessary to preserve body progression and balance (compensatory mechanism for flatfoot landing). 2) COMP: Reduced plantarflexor push-off was compensated by hip flexors to perform enhanced hip flexion in SW (important contribution to propulsive energy). Severe foot drop and PF failure: 3) COMP: Increased hip abduction and pelvic elevation on the swinging side (by prolonged activation of gluteus medium) were used to prevent tripping.</td>
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<td>P: 40 (14), 15-59</td>
<td>KM, KT, EMG</td>
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<td>Only foot drop: 1) COMP: In ST, increased passive ankle DF (delayed plantar flexor activation) in coordination with a greater hip extension and an additional antigravitational effort at the knee (prolonged knee extensor activity) was necessary to preserve body progression and balance (compensatory mechanism for flatfoot landing). 2) COMP: Reduced plantarflexor push-off was compensated by hip flexors to perform enhanced hip flexion in SW (important contribution to propulsive energy). Severe foot drop and PF failure: 3) COMP: Increased hip abduction and pelvic elevation on the swinging side (by prolonged activation of gluteus medium) were used to prevent tripping.</td>
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<td>P = C (matched)</td>
<td>KM, KT</td>
<td></td>
<td>Only foot drop: 1) COMP: Increase in hip and/or knee flexion in SW (steppage gait). Foot drop and push-off deficit: 2) COMP: Early ankle PF associated to an increase of PF internal moment and ankle power production in MST (vaulting gait).</td>
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<td>COMP: In ST, increased hip flexion caused anterior displacement of CoM and therefore reduced external flexor moment at the knee. In late ST, the CoP is on metatarsal area and thus knee flexory moment is reduced by reducing push-off force.</td>
</tr>
<tr>
<td>Frigo et al. (1996)</td>
<td>Juvenile Chronic Arthritis</td>
<td>P: 19 (4m/15f)</td>
<td>P: 11.8 (4), 6-19, C: - (-), 17-24</td>
<td>KM, KT</td>
<td>Affecting and unaffected (ankle, knee, hip)</td>
<td>COMP: In ST, hip abductor paresis was compensated for by lateral trunk sway to position CoM over the hip joint to avoid external hip adduction moment.</td>
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<td>C: 13 (-)</td>
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<td>P = C</td>
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<td>Only foot drop: 1) COMP: Increase in hip and/or knee flexion in SW (steppage gait). Foot drop and push-off deficit: 2) COMP: Early ankle PF associated to an increase of PF internal moment and ankle power production in MST (vaulting gait).</td>
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<td>COMP: In ST, increased hip flexion caused anterior displacement of CoM and therefore reduced external flexor moment at the knee. In late ST, the CoP is on metatarsal area and thus knee flexory moment is reduced by reducing push-off force.</td>
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<td>Gutierrez et al. (2005)</td>
<td>Myelo-meningocele</td>
<td>P: 31 (18m/13f)</td>
<td>P: 10.5 (2.6), C: 10.4 (2.5)</td>
<td>KM, KT</td>
<td>Affecting and unaffected (ankle, knee, hip)</td>
<td>COMP: In ST, increased hip flexion caused anterior displacement of CoM and therefore reduced external flexor moment at the knee. In late ST, the CoP is on metatarsal area and thus knee flexory moment is reduced by reducing push-off force.</td>
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<td>C: 21 (11m/10f)</td>
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<td>Only foot drop: 1) COMP: Increase in hip and/or knee flexion in SW (steppage gait). Foot drop and push-off deficit: 2) COMP: Early ankle PF associated to an increase of PF internal moment and ankle power production in MST (vaulting gait).</td>
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<td>COMP: In ST, increased hip flexion caused anterior displacement of CoM and therefore reduced external flexor moment at the knee. In late ST, the CoP is on metatarsal area and thus knee flexory moment is reduced by reducing push-off force.</td>
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2) COMP: Internal rotation of trunk and pelvis compensated for the lack of push-off power in the contralateral leg by providing forward progression.  
3) COMP: Posterior trunk extension moved the CoM behind the hip to compensate hip extensor weakness.  
4) COMP: With extensive DF weakness, pelvic hike became increasingly apparent in swing. |
| Huang et al. (2008)     | Bilateral OA in the medial compartment of the knee (2 groups: mild [MI] and severe [SE]) | P MI: 15 (6m/9f)            | P MI: 63.1 (11.9), -           | P = C      | KM, KT               | Affected (ankle, knee, hip, pelvis) | 1) COMP: In mild osteoarthritis, normal abductor moment was maintained mainly by lifting the pelvis of the swing side and thereby shifting the CoM towards the stance leg.  
2) COMP: By anterior pelvic tilting (forward displacement of CoM) external knee extensor moment was reduced. |
| Hurwitz et al. (1997)   | Unilateral OA of the hip                                | C: 15 (6m/9f)               | C: 63.2 (9.9), -               | P = C      | KM, KT               | Affected and unaffected (ankle, knee, hip, pelvis) | 1) COMP: Increase in lumbar lordosis compensated for inadequate hip extension in ST on the affected side.  
2) COMP: In ST, shifting CoM over affected hip joint (decrease in external hip adduction moment) decreased hip joint load and compensated for weakened hip abductors on the affected side.  
COMP: Patients compensated with pelvic elevation for insufficient flexion of the affected knee (<30°) in SW in order to assure foot clearance. |
| Laborde et al. (2003)   | Stroke with spastic right hemiplegia                    | P: 7 (4m/3f)                | P: 42.7 (18.4), -              | -          | KM                   | Affected (ankle, knee, pelvis) | COMP: Patients compensated with pelvic elevation for insufficient flexion of the affected knee (<30°) in SW in order to assure foot clearance. |
| Lehmann et al. (1987)   | Hemiparesis (strokes 3-13 years previously)             | C: 7 (3m/4f)                | C: 23.5 (2.1), 22-27           | P = C      | KM, KT               | Unclear (ankle, knee, hip) | 1) COMP: In MST, putting the CoM farther in front of knee by increased hip flexion explained increased external extension moment of the affected knee.  
2) COMP: In SW, circumduction of affected side compensated for reduced knee flexion and ankle DF (stiff-leg) |
| Matjačić et al. (2008)  | Spinal muscular atrophy, type III                      | P: 7 (4m/3f)                | P: 39.7 (11), -                | P = C      | KM, KT, EMG          | Affected (ankle, knee, hip, pelvis, trunk) | Compensations for weakness of knee and hip extensors:  
1) COMP: Premature activity of the soleus and gastrocnemius during loading response and MST to minimize external flexion moments at knee and hip by displacing the CoP along the foot earlier.  
2) COMP: Decreased rate of weight acceptance after foot contact by prolonged activity of contralateral hip abductors. |

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<tr>
<td>Mündermann et al. (2005)</td>
<td>Bilateral medial knee OA [less severe [LS] and more severe [MS]]</td>
<td>P LS: 19 (6m/13f) P MS: 23 (13m/10f)</td>
<td>P LS: 65.2 (12.5), 36-82</td>
<td>P = C</td>
<td>KM, KT</td>
<td><strong>Unclear (ankle, knee, hip, pelvis, trunk)</strong></td>
<td>COMP: In ST, lateral shift of the trunk to stance leg compensated to control and lower the load at the medial compartment of the knee by reducing the mediolateral distance between the CoM and the knee that resulted in a reduced GRF moment arm and therefore in a reduced knee adduction moment.</td>
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<td>C LS: 19 (6m/13f) C MS: 23 (13m/10f)</td>
<td>C LS: 61.7 (12.3), 39-86</td>
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<td>Nadeau et al. (1997)</td>
<td>Right chronic patellofemoral pain syndrome (PFPS)</td>
<td>P: 5 (2m/3f) C: 5 (2m/3f)</td>
<td>P: 28.4 (7.5), C: 25.5 (13.3), -</td>
<td>P = C</td>
<td>KM, KT</td>
<td><strong>Affected (ankle, knee, hip)</strong></td>
<td>COMP: In early ST, increased hip extensor moment in conjunction with decreased knee extensor moment as a strategy to reduce knee flexion and therefore loading on the affected patellofemoral joint.</td>
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<td>Newman et al. (2007)</td>
<td>Charcot-Marie-Tooth disease, types I and II</td>
<td>P: 16 (11m/5f) C: 40 (21m/19f)</td>
<td>P: 20.1 (13), 8-52</td>
<td>P &lt; C (sign.)</td>
<td>KM, KT</td>
<td><strong>Affected (ankle, knee, hip, pelvis)</strong></td>
<td>1) REACT: In early ST, knee and tibial segment internal rotation may have facilitated transfer of the bodyweight medially in the absence of normal foot pronation, while hip external rotation may have provided a rotational compensation for supinated varus feet and internally rotated knees to ensure adequate foot alignment. 2) COMP: Hip external rotation may also have improved ground clearance of the dropped foot in SW.</td>
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<td>Powers et al. (2002)</td>
<td>Patellofemoral pain syndrome (PFP)</td>
<td>P: 24 (-)</td>
<td>P: 25.4 (7.3), C: 27.6 (4.8), -</td>
<td>P &lt; C (sign.)</td>
<td>KM</td>
<td><strong>Affected (ankle, knee, hip)</strong></td>
<td>COMP: In MST, the reduced femoral internal rotation compensated to reduce the Q-angle and lateral force vector on the patella, bringing the patella more in line with the anterior superior iliac spine.</td>
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<td>Riad et al. (2008)</td>
<td>Spastic hemiplegic CP</td>
<td>C: 18 (-)</td>
<td>P: 8.4 (-), 4-19.8</td>
<td>-</td>
<td>KM, KT</td>
<td><strong>Affected and unaffected (ankle, knee, hip, pelvis)</strong></td>
<td>1) COMP: In early ST, the unaffected side showed increased PF moment to create vaulting for clearance of the affected limb and to generate a more symmetric pattern. 2) COMP: Larger symmetrical hip power generation might have compensated for decreased ankle power generation on affected side (potential power generation from unaffected ankle was not fully used and optimised, but symmetry was preferred as a way of dealing with the movement impairment).</td>
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<td>Romano et al. (1996)</td>
<td>Unilateral congenital dysplasia of the hip</td>
<td>P: 21 (6m/15f) C: 46 (-)</td>
<td>P: 48 (-), 25-71</td>
<td>P &lt; C (sign.)</td>
<td>KM, KT</td>
<td><strong>Affected and unaffected (ankle, knee, hip, pelvis, trunk)</strong></td>
<td>COMP: The range of motion of the hip, particularly extension, was reduced. As a consequence, the knee compensated with flexion to allow the pelvis to progress forward before toe-off.</td>
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<tr>
<td>Romkes &amp; Brunner (2007)</td>
<td>Hemiplegic CP</td>
<td>P: 12 (8m/4f) C: 10 (5m/5f)</td>
<td>P: 12.3 (4.1), - C: 29.5 (3.3), -</td>
<td>-</td>
<td>KM, KT, EMG</td>
<td>Affected (ankle, knee, hip, pelvis)</td>
<td>1) COMP: In terminal SW, early onset of gastrocnemius and reduced tibialis anterior activity might have compensated to prepare the foot for toe-heel instead of heel-toe gait. 2) COMP: At initial contact, the tensed gastrocnemius did not allow the knee to fully extend and therefore limited KE.</td>
</tr>
<tr>
<td>Sankar et al. (2009)</td>
<td>Recurrent deformity following treatment of idiopathic clubfoot</td>
<td>P: 35 (-) C: 31 (-)</td>
<td>P: 6.7 (-), 3.6-15.4 C: -(-), -</td>
<td>-</td>
<td>KM, EMG</td>
<td>Unclear (ankle, knee, hip)</td>
<td>REACT: 80% of children walked with intoeing gait and many (50%) showed external hip rotation in ST as a result.</td>
</tr>
<tr>
<td>Saraph et al. (2002)</td>
<td>Spastic CP (hemiplegic [HP] and diplegic [DP])</td>
<td>P: 22 (-) (14 HP, 8 DP) C: 20 (-)</td>
<td>P: 11.9 (-), 9.2-15.5 C: -(-), -</td>
<td>-</td>
<td>KM, KT</td>
<td>Affected and unaffected (ankle, knee, hip, pelvis)</td>
<td>REACT: In all phases of gait, the internal rotation deformity of the affected leg caused an external rotation of the hemipelvis, bringing the internally rotated limb in the direction of progression. The unaffected side showed corresponding internal rotation of the pelvis and external rotation at the hip. 1) COMP: The loss of lumbar lordosis resulted in anterior CoM translation and was compensated by hip hyperextension. 2) COMP: With advancing age this compensation was not applicable anymore (due to decreased muscle strength &amp; decreased overall joint flexibility). Then, subjects compensated the anterior CoM position by flexing hips and knees (crouched posture and gait).</td>
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<tr>
<td>Sarwahi et al. (2002)</td>
<td>Postoperative flatback</td>
<td>P: 21 (3m/18f) C: Mentioned but not specified</td>
<td>P: 53 (-), 36-83 C: 45 (-), 25-60 P &lt; C (sign.)</td>
<td>-</td>
<td>KM, KT</td>
<td>Affected (ankle, knee, hip, pelvis, trunk)</td>
<td>1) REACT: In ST, excessive pelvic rotation (protrusion on unaffected side), when accompanied by reduced extension of affected hip, was likely to be a secondary deviation to allow adequate step length. This is often accompanied by internal rotation of the affected hip and external rotation of the unaffected hip. 2) COMP: In SW, increased PF on unaffected side (vaulting) and increased hip flexion on affected side compensated for foot deformity in clearing the foot.</td>
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<tr>
<td>Stebbins et al. (2010)</td>
<td>Spastic hemiplegic CP with foot deformity on affected side</td>
<td>P: 12 (6m/6f) C: 15 (5m/10f)</td>
<td>P: 11.8 (-), 6-14 C: 9.5 (-), 6-14</td>
<td>-</td>
<td>KM, KT, EMG</td>
<td>Affected and unaffected (ankle, knee, hip, pelvis)</td>
<td>1) REACT: In ST, excessive pelvic rotation (protrusion on unaffected side), when accompanied by reduced extension of affected hip, was likely to be a secondary deviation to allow adequate step length. This is often accompanied by internal rotation of the affected hip and external rotation of the unaffected hip. 2) COMP: In SW, increased PF on unaffected side (vaulting) and increased hip flexion on affected side compensated for foot deformity in clearing the foot.</td>
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<tr>
<td>Theologis et al. (2003)</td>
<td>Patients with previously treated congenital talipes equinovarus (clubfoot)</td>
<td>P: 20 (15m/5f)</td>
<td>P: 9.8 (2.3), 6.9-14.6</td>
<td>P = C</td>
<td>KM, KT</td>
<td>Affected and unaffected (ankle, knee, hip, pelvis)</td>
<td>REACT: External rotation of the hip resulted from in-toeing gait (in-toeing persisted in approximately half of the subjects).</td>
</tr>
<tr>
<td>Torry et al. (2004)</td>
<td>Unilateral ACL deficiency (complete isolated rupture, greater than 2 years prior to measurement)</td>
<td>P: 16 (9m/7f)</td>
<td>P: 28.1 (12.7), -</td>
<td>P = C</td>
<td>KM, KT, EMG</td>
<td>Unclear (ankle, knee, hip)</td>
<td>COMP: In early ST, increased hip extension torque (increased HS activity) explained decreased net knee extensor torque as a compensation to reduce anterior tibial translation and ACL strain on the affected side by maintaining normal knee kinematics.</td>
</tr>
<tr>
<td>Westhoff et al. (2006)</td>
<td>Legg Calvé Perthes disease (LCPD)</td>
<td>P: 33 (24m/9f)</td>
<td>P: 8 (2), -</td>
<td>P &lt; C (sign.)</td>
<td>KM, KT</td>
<td>Affected and unaffected (hip, pelvis)</td>
<td>COMP: In ST, hip abductor weakness on the affected side was compensated by trunk lean to the stance limb with the pelvis stabilized (Duchenne gait), decreasing the hip abductor moment and producing a hip-unloading effect.</td>
</tr>
<tr>
<td>Yavuzer et al. (2001)</td>
<td>Hemiplegia (after stroke)</td>
<td>P: 46 (30m/16f)</td>
<td>P: 58 (7.5), -</td>
<td>P &lt; C (sign.)</td>
<td>KM, KT</td>
<td>Affected and unaffected (ankle, knee, hip, pelvis)</td>
<td>1) COMP: Greater than normal flexion of the affected hip during MST moved the CoM farther in front of the knee, explaining the increased external KE moment. 2) COMP: In SW, the lack of ankle DF and insufficient knee flexion on the affected side was substituted by contralateral pelvic/hip hiking with circumduction of the limb or vaulting on the unaffected side.</td>
</tr>
</tbody>
</table>

Abbreviations: COMP = Compensatory mechanism, REACT = Physical reaction, P = Patients, C = Controls, m = male, f = female, KM = Kinematics, KT = Kinetics, EMG = Electromyography, CoM = Center of Mass, CoP = Center of Pressure, ST = Stance, MST = Midstance, SW = Swing, PF = Plantarflexion, DF = Dorsiflexion, KE = Kneeextension, HS = Hamstrings
Appendix B: Overview of studies on within-trunk motion (Chapter 3)
Table B.1: Overview marker based gait studies quantifying within-trunk motion.

<table>
<thead>
<tr>
<th>Authors (year)</th>
<th>Title</th>
<th>N (m/f)</th>
<th>Age (y±SD)</th>
<th>Height (cm±SD)</th>
<th>Weight (kg±SD)</th>
<th>Method</th>
<th>Main outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crosbie et al. (1997)</td>
<td>Patterns of spinal motion during walking</td>
<td>50m</td>
<td>46.34 ±18.25</td>
<td>172±8</td>
<td>73.7 ±10.5</td>
<td>Video-based system (MAC, 60 frames/s), backless swimsuit, 13 body markers</td>
<td>Consistent patterns within and between segments and movements, with apparent consequential trunk motion following pelvic displacements. Spinal movements associated with walking are linked to the primary motions of the pelvis and the lower limbs.</td>
</tr>
<tr>
<td>Crosbie et al. (1997)</td>
<td>Age, gender and speed effects on spinal kinematics during walking</td>
<td>50m</td>
<td>46.34 ±18.25</td>
<td>172±8</td>
<td>73.7 ±10.5</td>
<td>Video-based system (MAC, 60 frames/s), backless swimsuit, 13 body markers</td>
<td>Increased range of motion in each segment with increased walking speed, few gender-related differences in patterns or range of motion and significant reduction in spinal range of motion with advancing age. Age-related changes are more rely to be step-length dependent than an intrinsic feature of aging.</td>
</tr>
<tr>
<td>Frigo et al. (2003)</td>
<td>The upper body segmental movements during walking by young females</td>
<td>18f</td>
<td>12.3 (10.6-18)</td>
<td>156 (134-170)</td>
<td>49.2 (29-69)</td>
<td>A model of kinematic computation in a protocol for multifactorial gait analysis. 11 retro reflective markers on the trunk</td>
<td>Small segmental movements (less than 5°) during gait, except the angle of proximal curvature in the frontal plane shoulder rotation and angle between shoulders and pelvis. All the measured angles were far below their possible ranges of motion.</td>
</tr>
<tr>
<td>van Emmerik et al. (2005)</td>
<td>Age-related changes in upper body adaptation to walking speed in human locomotion</td>
<td>4m*</td>
<td>23.3* ±4.0*</td>
<td>172.7* ±0.4*</td>
<td>69.0*</td>
<td>Treadmill walking, 7 cameras (Qualysys) 100 Hz. 3 trunk marker 3 pelvis marker. Systematically increased and decreased speed, between min. speed (0.2 m/s) max. speed (1.8 m/s or subject’s max. speed)</td>
<td>Pelvic motion in the sagittal plane (AP tilting) had a reduced amplitude in the older group at all walking velocities. Increased frontal plane counter rotation between head and trunk in middle and older age groups. In all three planes of motion reduced counter movement in the upper body segments in the older age group</td>
</tr>
<tr>
<td>Konz et al. (2006)</td>
<td>A Kinematic Model to Assess Spinal Motion During Walking</td>
<td>5m 5m</td>
<td>27±4</td>
<td>171±6</td>
<td>71.9 ±12.2</td>
<td>Measuring known angles from a mechanical model, comparing them to calculated angles validated the kinematic model. Spine motion data were collected from 10 able-bodied adults walking at 5 self-selected speeds. Results were compared to literature data.</td>
<td>The uniaxial angles measured on the mechanical model were within 5° of the calculated kinematic model angles, and the coupled angles were within 2°. A multi-segment kinematic spine model has been developed and validated for analysis of spinal motion during walking.</td>
</tr>
<tr>
<td>Leadini et al. (2011)</td>
<td>Multi-segment trunk kinematics during locomotion and elementary exercises</td>
<td>5m 5f</td>
<td>24.7 ±0.8</td>
<td>171.6 ±8.1</td>
<td>62.4 ±9.3</td>
<td>Fourteen trunk markers, self-selected walking speed, 10 repetitions measured with a Vicon system</td>
<td>Intra-subject repeatability over ten repetitions high for most of the measurements. Considerable subject-specific motion at each of the different trunk segments in all three anatomical planes.</td>
</tr>
</tbody>
</table>

* = young; ** = mid; *** = old
Appendix C: Segmental Trunk Angles in Scoliosis Patients (Chapter 5)
Figure C.1: Rigid segments versus global coordinate system in AIS patients (thoracic scoliotic curve: right convex).
Figure C.2: Rigid thoracic versus pelvis segment in AIS patients (thoracic scoliotic curve: right convex).
Appendix D: Segmental Trunk and Lower Extremity Angles in Cerebral Palsy Patients (Chapter 6)
Figure D.1: Rigid segments versus global coordinate system in hemiplegic CP patients (gait cycle of the patients’ affected and controls’ left leg).
Figure D.2: Lower extremity and pelvis angles according to the Plug-in Gait model in hemiplegic CP patients (gait cycle of the patients’ affected and controls’ left leg).
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Appendix F: Curriculum Vitae
Appendix F

Curriculum Vitae
Stefan Schmid

Personal Information

Date of birth: November 08, 1979
Place of birth: Bern, Switzerland
Nationality: Swiss
Marital status: Single
Languages: German and Swiss-German (Mother tongue)
             English (Fluent written and oral)
             French (Basic communication skills)
Personal interests: Skydiving, Motorcycles

Contact Information

Bern University of Applied Sciences
Health Division
Murtenstrasse 10
3008 Bern, Switzerland
P: +41 31 848 37 96
E: stefan.schmid@bfh.ch

ETH Zurich
Institute for Biomechanics
HCP H 23.2
Leopold-Ruzicka-Weg 4
8093 Zurich, Switzerland
E: sschmid@ethz.ch

Education

2012 – 2016
ETH Zurich, Department of Health Sciences and Technology,
Institute for Biomechanics (CH)
Doctoral Candidate
Thesis (Advisors: W.R. Taylor, PhD, S. Lorenzetti, PhD, DSc and R. Brunner, MD):
Advancing Clinical Movement Analysis: Spinal Kinematics are Fundamental for
Understanding Normal and Pathological Gait

2008 – 2009
New York University, Steinhardt School, Department of
Physical Therapy (USA)
Master of Arts in Pathokinesiology
Master-Thesis (Advisor: Marilyn Moffat, PT, DPT, PhD, DSc (hon)):
Effect of Cryotherapy on the Electromyographic Activity of Lower Extremity Muscles
during a Plyometric Exercise in Normal Healthy Adults

2001 – 2005
School of Physiotherapy Bern (CH)
Diploma in Physiotherapy
Diploma-Thesis (Advisor: Lorenz Radlinger, PhD):
Variability of Ground Reaction Forces in Healthy Subjects during Stair Climbing

1995 – 1999
Lehrwerkstätten der Stadt Bern (CH)
Certificate in Electronics Technology / Professional Maturity Certificate
## Continuing Education / Certifications

<table>
<thead>
<tr>
<th>Year</th>
<th>Institution</th>
<th>Details</th>
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<tbody>
<tr>
<td>2014</td>
<td>University of Bern, Clinical Trials Unit (CH)</td>
<td>Clinical Investigators I and II: Basic and advanced GCP</td>
</tr>
<tr>
<td>2010</td>
<td>Istituto Ortopedico Rizzoli, Bologna (IT)</td>
<td>ESMAC Gait Analysis Course</td>
</tr>
<tr>
<td>2007</td>
<td>The University of New South Wales, Institute of Languages (AU)</td>
<td>General Proficiency English (Level: Advanced; Result: Distinction)</td>
</tr>
<tr>
<td>2006 – 2008</td>
<td>Akademie für Medizinische Aus- und Weiterbildung (DE)</td>
<td>Post-Graduate Certification in Analytical Biomechanics (Sohier)</td>
</tr>
</tbody>
</table>

## Professional Appointments

<table>
<thead>
<tr>
<th>Year</th>
<th>Institution</th>
<th>Position</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010 – present</td>
<td>Bern University of Applied Sciences, Health Division, Bern (CH)</td>
<td>Research Associate, 80-100%</td>
</tr>
<tr>
<td>2009 – 2010</td>
<td>Manhattan Physical Medicine &amp; Rehab., New York NY, (USA)</td>
<td>Physical Therapist, 100%</td>
</tr>
<tr>
<td>2005 – 2008</td>
<td>Hirslanden Klinik Beau-Site, Bern (CH)</td>
<td>Physical Therapist, 80-90%</td>
</tr>
<tr>
<td>2003 – 2005</td>
<td>Physiotherapie Steinbach, Med. Trainingstherapie, Belp (CH)</td>
<td>Instructor / Supervisor, 10-20%</td>
</tr>
<tr>
<td>2000 – 2001</td>
<td>Ascom AG, Bern (CH)</td>
<td>Electronics Technologist, 100%</td>
</tr>
</tbody>
</table>

## Institutional Appointments

<table>
<thead>
<tr>
<th>Year</th>
<th>Institution</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015 – present</td>
<td></td>
<td>Co-Leader Clinical Specialization “General Physiotherapy”, MSc in Physiotherapy program, Bern University of Applied Sciences, Health Division, Bern (CH)</td>
</tr>
<tr>
<td>2005 – 2008</td>
<td></td>
<td>Clinical Internship Supervisor, Hirslanden Klinik Beau-Site, Bern (CH)</td>
</tr>
<tr>
<td>2000 – 2001</td>
<td></td>
<td>Teamleader ATM Light Ring Component Test, Ascom AG, Bern (CH)</td>
</tr>
</tbody>
</table>

## Grants and Scholarships

<table>
<thead>
<tr>
<th>Year</th>
<th>Grant Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>2013</td>
<td>Research grant, Swiss Physiotherapy Association (physioswiss), Sursee (CH) (CHF 6'000.-)</td>
</tr>
<tr>
<td>2009</td>
<td>Graduate scholarship, Swiss-American Society, Zurich (CH) (CHF 2'500.-)</td>
</tr>
<tr>
<td>2008</td>
<td>Graduate scholarship, Manuela-Geiger-Foundation, Aarau (CH) (CHF 16’000.-)</td>
</tr>
</tbody>
</table>
Research Support

2012 – present  The Measurement of Spinal Kinematics during Gait for the Quantification of Intervention Outcomes in Patients with different Pathologies (NCT01803256 / NCT01803243)
   Role: Co-Investigator
   Sponsor(s): BFH, physioswiss, UKBB, ETHZ

2010 – present  Influence of stochastic resonance therapy (SRT) on postural control, gait and strength performance capacity in patients suffering from stroke or traumatic brain injury (SNSF, 13DPD6_127280)
   Role: Co-Investigator
   Sponsor(s): Swiss National Science Foundation (CHF 70’263. -)

   Role: Co-Investigator
   Sponsor(s): Swiss National Science Foundation (CHF 101’964. -)

2010 – 2012  Swiss Physiotherapy Research Priorities – How are they defined and what is expected?
   Role: Co-Investigator
   Sponsor(s): PTW, physioswiss, BFH, ZHAW, SUPSI, HES-SO

2010 – 2011  Strength and patient care in elderly (BFH, 10158VPT_WGS)
   Role: Co-Investigator
   Sponsor(s): BFH (CHF 85’625. -)

Memberships in Professional Societies

2011 – present  European Society of Biomechanics (ESB) – # 1266
2011 – present  European Society of Movement Analysis in Adults and Children (ESMAC) – # ESM00246
2010 – present  Swiss Physiotherapy Association (physioswiss) – # 116143

Professional Service

Scientific Journal Peer-Reviewing

2013 – present  Ad Hoc Reviewer, Journal of Biomechanics
2013 – present  Ad Hoc Reviewer, Medical Engineering & Physics
2013 – present  Ad Hoc Reviewer, Research in Developmental Disabilities
2013 – present  Ad Hoc Reviewer, Age (Official J. of the American Aging Association)
Appendix F

Teaching Experience

Lectures, courses and seminars

Undergraduate Level:

2013 – present  
Lecturer, “Biomechanics”, M0011, BSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH), 50 students, 30h of teaching/year

Graduate Level:

2015  
Guest Lecturer, “Introduction to Movement Analysis of the Spine”, MAS in Functional Kinetic Science, University of Basel, Basel (CH), 20 students, 1h of teaching

2014  
Lecturer, “Principles of Biomechanics and Movement Analysis”, Summer School – MSc in Physiotherapy Preparation Course, Bern University of Applied Sciences, Bern (CH), 10 students, 8h of teaching

2010 – 2011  
Teaching Assistant, “Epidemiology”, MGP0430, MSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH), 25 students, 10h of teaching/year

2013 – present  
Lecturer, “Introduction to EndNote for Master-Students”, Bern University of Applied Sciences, Bern (CH), 20-40 students, 3h of teaching/year

2012 – 2013  
Lecturer, “Introduction to the Movement Laboratory for Master-Students”, MSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH), 5-10 students, 4-6h of teaching/year

Course Responsibility

Graduate Level:

2015 – present  
Module Coordinator, “Anatomy and Biomechanics of the Locomotor System”, MGP1101, MSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH)

2015 – present  
Module Coordinator, “Transfer Module 3”, MSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH)

2011 – present  
Module Coordinator (until 2014 Co-Cordinator), “Transfer Modules 1&2”, MGP0612&MGP0615, MSc in Physiotherapy Program, Bern University of Applied Sciences, Bern (CH)

Advisory and Supervisory Responsibility

Undergraduate Theses:

3. Dino Causevic, BSc in Mechanical Engineering, 2014, ETH Zurich, “Testung und Weiterentwicklung einer Auswertungsroutine zur Quantifizierung der Wirbelsäulen-Kinematik”  
(Advisor, thesis completed)
Appendix F

Graduate Theses:

7. Fabiola Angelico, MSc in Physiotherapy, 2015, Zurich University of Applied Sciences, “Upper limb movements during gait in children with Leg Length Discrepancy – a kinematic analysis” (Supervisor, thesis completed)

6. Angela Ehrhardt, MSc in Physiotherapy, 2015, Bern University of Applied Sciences, “Vergleich der Reflexaktivität von normalbeweglichen und hypermobilen Frauen beim Treppensteigen” (Co-Supervisor, thesis completed)

5. Michael Niederer, MSc in Physiotherapy, 2014, Zurich University of Applied Sciences, “Effects of an induced extension restriction in the knee on secondary gait deviations in healthy young adults” (Co-Supervisor, thesis completed)

4. Martina Furrer, MSc in Physiotherapy, 2014, Bern University of Applied Sciences, “Validation of a smartphone-based measurement tool for the quantification of level walking” (Supervisor, thesis completed)


2. Adrien Cerrito, MSc in Physiotherapy, 2013, Bern University of Applied Sciences, “Development and validation of an Android®-based measurement tool for the quantification of the sit-to-stand movement” (Supervisor, thesis completed)


Research Assistants / Interns:

07/15 – 09/15 Samar Almubarak, BS in Mechanical Engineering student, Northeastern University (USA), 9 week research assistantship at ETH Zurich (co-supervisor)

10/14 – 11/14 Yael Brudsche, MSc in Physiotherapy student, Bern University of Applied Sciences, 5 week scientific internship (supervisor)

10/13 – 12/13 Yvonne Brühlhart, MSc in Physiotherapy student, Bern University of Applied Sciences, 10 week scientific internship (supervisor)

11/12 – 02/13 Martina Furrer, MSc in Physiotherapy student, Bern University of Applied Sciences, 10 week scientific internship (supervisor)

11/11 – 02/12 Adrien Cerrito, MSc in Physiotherapy student, Bern University of Applied Sciences, 10 week scientific internship (supervisor)

11/11 – 02/12 Helene Moser, MSc in Physiotherapy student, Bern University of Applied Sciences, 10 week scientific internship (supervisor)
## Invited Talks

2. **Schmid S.** Quantifizierung der Wirbelsäulen-Bewegungen beim Gehen. Colloque Santé, Bern University of Applied Sciences, Bern (CH), November 25, 2014.


## Bibliography

### Refereed Journal Publications (peer reviewed)


Published Abstracts and Congress Proceedings (peer reviewed)


**Oral and Poster Presentations (Presenter)**


Non-peer reviewed Journal Publications


Media appearances

1. SRF, Swiss Public Television, February 20, 2012: Puls "Hypermobilität - Wenn Beweglichkeit zur Qual wird".