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Review

Secondary gait deviations in patients with and without neurological involvement: A systematic review

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ABSTRACT

Pathologies that lead to biomechanical restrictions in human gait interfere with the tightly regulated muscle activation patterns that control the external moments. In order to maintain proper function, secondary mechanisms are required. The aims of this systematic review were (1) to identify secondary mechanisms in pathologic gait that have been described throughout the scientific literature by means of instrumented gait analysis, (2) to distinguish between active compensatory mechanisms and passive physical effects and (3) to identify common compensatory mechanisms that appear to be independent from the underlying disease. A comprehensive literature search revealed 4080 citations for review, whereof 148 studies entered the full-text review. Thirty-six studies were included and the quality of these studies was assessed by two independent reviewers (kappa = 0.83). The quality of the included studies showed large variation and several methodological issues were identified. Five studies were further identified describing only passive physical effects, leaving a total of 31 studies reporting on compensations. The qualitative analysis revealed common compensations that appeared to be independent from the underlying pathology. In clinical practice, distinguishing primary from secondary gait deviations can be considered highly important since unnecessary treatment may be avoided. However, given the introduction of general principles of compensatory mechanisms and the fact that certain presumed "compensations" were identified as simple passive physical effects, secondary gait deviations have to be further investigated. Computer simulation studies are valuable, especially in respect of the distinction between compensations and physical effects. Furthermore, the need for a uniform terminology was highlighted.

1. Introduction

Healthy human gait uses repetitive reciprocal limb motions in order to advance the body while simultaneously maintaining stance stability [1]. 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 [2,3]. 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 [4,5]. 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

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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 [6–9].

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 [9]. As a consequence, primary abnormalities will be corrected when treated directly whereas secondary problems may resolve spontaneously once the primary issue is addressed [7,9].

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. [9], for example assessed 12 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. [10], 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 [7], simulated shortening of the hamstrings [11] or simulated leg length discrepancy [12] as well as computer simulations [13–15].

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 [16]. Nevertheless, the possibility of modifying one single parameter such as plantarflexion 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. [6] and Stebbins et al. [9] 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 and Romkes [17] and Matjacic et al. [18] 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 [17]. By investigating a group of orthopedic patients suffering from different orthopedic conditions, Brunner and Romkes [17] 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. Methods

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–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. 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 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

Table 1
Inclusion and exclusion criteria for the title and abstract screening as well as for the full text evaluation.

Category	Inclusion	Exclusion
Type of studies	Original research, published in peer-reviewed scientific journals	Conference abstracts, non peer-reviewed publications, secondary literature or reviews
Main outcomes	Clearly identified secondary gait deviations; data had to be retrieved from skin-mounted markers by means of at least two-dimensional kinematic data	Single video data without markers
Subjects[a]	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	Parkinson's disease (due to bradykinesia), Down syndrome (due to complex cognitive impairments) or obesity (due to inaccurate placement of markers)
Measurement conditions	Walking on level ground with a smooth surface and without any obstacles	Treadmill walking, stair climbing, walking up- or downhill, walking on uneven ground or a slippery surface
Walking characteristics	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)	Running studies, special footwear (e.g. MBT-shoes)

[a] Except for computer simulations studies.

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.

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 and Black [19], 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 $\geq 80\%$). Finally, the QA checklist consisted of 17 items with a maximum score of 20 points (Appendix A), including the five different categories "quality of reporting" (eight items, maximum 10 points), "external validity" (three items, maximum three points), "internal validity – bias" (three items, maximum three points), "internal validity – confounding" (two items, maximum two points) and "statistical power" (one item, maximum two 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.).

2.4. 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 [20]. Kappa values were calculated using the command "kap" (STATA, Version 9.2, StataCorp, College Station, TX, USA) and the user-written STATA-command "kapci" for the bias-corrected bootstrap confidence intervals [21]. 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.

3. Results

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 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 (nine in vivo simulation, nine computer simulation and four single-case studies). The subsequent citation index search for the first category identified another two 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.

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 [22]. 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. The numbers of studies that follow are all based on a 100% agreement between the two independent reviewers.

For 15 studies [4,6,9,10,17,23–32], respectively 18 studies [5,6,10,17,25,27,28,30,32–41], 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, 11 papers [9,17,24,27–29,31,38,40,42,43] did not describe any random estimates of variability such as standard deviation or interquartile range of the main outcome variables and 20 papers [4,9,17,23–27,29,31,33–36,39,40,42,44–46] 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 [4,5,10,24,25,29,31,33–35,37,39–43,45–51], respectively 29 studies [4–6,9,10,17,23–27,29,31,33,35,37,39–51]) 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 11 papers [5,24,29,34,37,41,43–47], it was not identified where the measurements took place and what the

Table 2
Results of study quality rated by the reviewers S.S. and K.S.

First author	Reporting		External validity		Internal validity (bias)		Internal validity (confounding)		Power		Total score	
	S.S.	K.S.	S.S.	K.S.	S.S.	K.S.	S.S.	K.S.	S.S.	K.S.	S.S.	K.S.
Alkjaer [44]	8	8	1	0	3	2	1	1	0	0	13	11
Allen [23]	3	2	1	2	2	1	0	0	0	0	6	5
Aminian [4]	7	7	1	1	1	2	1	1	0	0	10	11
Berchuck [33]	7	7	1	1	1	1	1	1	0	0	10	10
Brunner [17]	5	5	2	1	2	1	1	2	0	0	10	9
Bulgheroni [24]	6	5	0	0	3	2	1	1	0	0	10	8
Chen [34]	7	7	1	1	1	2	1	1	0	0	10	11
Cimolin [25]	7	6	1	1	2	2	1	1	0	0	11	10
Cruz [47]	10	10	0	0	1	2	1	0	2	2	14	14
D'Angelo [35]	6	6	1	1	1	1	1	1	0	0	9	9
Davids [6]	7	7	2	1	2	3	1	1	0	0	12	12
Don [26]	7	8	2	2	2	2	1	0	0	0	12	12
Ferrarin [45]	8	8	0	0	3	3	1	0	0	0	12	11
Frigo [27]	5	4	2	2	0	0	1	1	0	0	8	7
Gutierrez [36]	8	8	3	2	3	3	2	2	0	0	16	15
Gutierrez [28]	7	6	2	2	2	2	0	0	0	0	11	10
Hurwitz [37]	9	8	0	0	3	3	1	1	0	0	13	12
Laborde [42]	5	5	1	1	1	2	0	0	0	0	7	8
Lehmann [29]	5	5	0	0	1	1	0	0	0	0	6	6
Matjacic [18]	10	10	3	3	2	2	1	2	0	0	16	17
Mündermann [43]	8	8	0	0	2	2	1	1	0	0	11	11
Nadeau [46]	8	8	0	0	2	2	1	1	0	0	11	11
Newman [30]	8	8	2	1	3	3	1	0	0	0	14	12
Powers [48]	10	10	1	0	3	2	1	1	0	0	15	13
Riad [38]	6	7	3	3	1	2	0	0	0	0	10	12
Romano [39]	5	5	1	1	2	1	1	0	0	0	9	7
Romkes [10]	6	7	1	1	2	3	1	1	0	0	10	12
Sankar [40]	2	4	1	1	0	1	1	1	0	0	4	7
Saraph [5]	7	7	0	0	0	1	1	1	0	0	8	9
Sarwahi [31]	6	6	1	1	1	2	1	1	0	0	9	10
Shier-Chieg [49]	10	10	1	1	3	3	1	1	0	0	15	15
Stebbins [9]	7	5	1	2	1	0	1	0	0	0	10	7
Theologis [32]	7	7	2	3	3	3	1	1	0	0	13	14
Torry [50]	10	10	1	1	2	3	1	1	1	1	15	16
Westhoff [41]	9	9	0	0	3	3	1	0	0	0	13	12
Yavuzer [51]	9	9	1	1	3	3	1	1	0	0	14	14

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 [47,50] reported on the inclusion of a power analysis (a priori or post hoc) and the respective effective power-values.

3.3. Methodological data

The extracted data (subject characteristics, methodological data and compensatory mechanisms/secondary deviations) are presented in Table 3. The included studies contained a variety of several different neurological and orthopedic. The patient group average ages were between 6.6 years [35] and 65 years [43], 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 [29,44,45,49], 11 studies reported that the patients walked slower than the control group subjects [6,23,25,30,31,34,39,41,47,48,51] and nine studies reported that there was no difference in gait speed between the patients and the control group subjects [18,24,27,32,35,37,43,46,50]. The remaining 12 studies provided no information on walking velocity. Further, 14 studies indicated that the subjects walked barefoot [4,9,10,25–27,30,35,38,39,41,42,45,51], five studies described specific and one study non-specific footwear [29,43,44,46,47,50] 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 12 of the 23 studies measuring patients with unilateral pathologies evaluated, apart from the affected side, also the unaffected side. Thirteen 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), 11 studies on three levels and 23 studies on four or more levels.

3.4. Compensatory mechanisms and physical effects

Based on the definition of Brunner et al. [2] and Gaston et al. [3], five studies were identified describing solely physical effects [4–6,32,40] and two studies describing both physical effects and compensatory mechanisms [9,30] leaving a total of 31 studies including a description of compensatory mechanisms. Thereof, 24 studies (77%) [9,10,18,23,25–31,33–35,37,39,41,42,45,47–51] of the 31 studies investigating kinematics, 19 (68%) [18,24–27,30,34,36–39,41,43,44,46–50] of the 28 investigating kinetics, and seven (100%) [10,17,18,24,26,33,50] 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 [9,27,33,34,38,51] and two (22%) only on the affected side [37,39]. The remaining study [41] did not specify the side of compensation.

Overall, eight studies (26%) [29,33,42,43,46–49] of the 31 selected studies described compensations on only one level, 12 (39%) [9,10,17,24,28,31,34,35,37,38,44,50] on two levels and 11

Table 3
Overview of the 36 included clinical studies.

First author	Diagnosis	Number of subjects (gender)	Age in years: mean (SD), range	Gait speed	Parameters evaluated	Side (level) evaluated	Compensatory mechanisms and secondary deviations
Alkjaer [44]	Early intensive treatment (at age of 3 months) for unilateral congenital clubfoot	P: 9 (9 m/0 f) C: 15 (15 m/0 f)	P: 19.7 (–), 18–23 C: 30 (–), 21–42	P = C (matched)	KM, KT	Affected (ankle, knee, hip)	(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)
Allen [23]	Spastic hemiplegic cerebral palsy (CP) with leg-length discrepancy (LLD)	P (LLD > 1.5 cm): 49 (31 m/18 f) P (LLD < 1.5 cm): 99 (56 m/43 f) C: 61 (32 m/29 f)	P (LLD > 1.5 cm): 10.5 (–), 4.1–17.7 P (LLD < 1.5 cm): 8.2 (–), 3.2–22.8 C: 9.4 (–), 4.1–15.6	P < C	KM	Unclear (ankle, knee, hip)	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
Aminian [4]	Spastic hemiplegic CP (surgery subset: proximal femoral derotation osteotomy with concomitant soft tissue surgeries)	P: 71 (41 m/30 f) (Surgery: 9/71) C: normal pediatric database	P: 8.4 (–), 3.9–18.4 C: – (–), –	–	KM	Affected and unaffected (ankle, knee, hip, pelvis)	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
Berchuck [33]	Unilateral anterior cruciate ligament (ACL) deficiency (5/16: isolated ACL tear, 11/16: ACL tear and a minor meniscal lesion)	P: 16 (14 m/2 f) C: 10 (5 m/5 f)	P: 26 (9.5), – C: 26 (5), –	–	KM, KT	Affected and unaffected (knee, hip)	(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
Brunner [17]	Several orthopedic conditions, but no underlying neurological disorders	P: 39 (17 m/23 f) C children: 13 (6 m/7 f) C adults: 14 (7 m/7 f)	P: 17.8 (–), 6.8–50.4 C children: 10.1 (–), 7–16 C adults: 32 (–), 25–58	–	KM, KT, EMG	Unclear (ankle, knee, hip, pelvis)	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)
Bulgheroni [24]	Symptomatic ACL deficiency	P: 10 (10 m/0 f) C: 10 (10 m/0 f)	P: 27 (6), – C: 25 (4), –	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

Chen [34]	Hemiplegia (6 months after first stroke, 2 groups: poor and good motor stage)	P poor: 17 (7 m/10 f) P good: 18 (10 m/8 f) C: 15 (7 m/8 f)	P poor: 59.4 (14.1), – P good: 63.1 (11.2), – C: 58.2 (9.3), –	P < C (sign.)	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)
Cimolin [25]	Hereditary spastic paraplegia (HSP) and mild spastic diplegia (SD) secondary to CP	P HDP: 15 (–) P SD: 40 (–) C: 20 (–)	P HDP: 10.1 (5.6), – P SD: 8.6 (4.3), – C: 10.5 (5.2), –	P < C (sign.)	KM, KT	Affected (ankle, knee, hip, pelvis)	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
Cruz [47]	Chronicstroke	P: 18 (12 m/6 f) C: 8 (5 m/3 f)	P: 54.8 (9.9), – C: 51.8 (9.4), –	P < C (sign.)	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
D'Angelo [35]	Duchenne muscular dystrophy (2 groups: no treatment [NoT], treatment [T])	P NoT: 11 (11 m/0 f) P T: 10 (10 m/0 f) C: 10 (10 m/0 f)	P NoT: 6.6 (2.8), – P T: 7.3 (1.9), – C: 7.4 (1.2), –	P = C	KM, KT	Unclear (ankle, knee, hip, pelvis)	(1) 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 (2) COMP: In SW, excessive ankle PF was compensated by increased flexion and abduction of the hip to aid clearance
Davids [6]	Spastic diplegic type CP with toe-walking gait pattern (in addition: control group mimicking toe-walking)	P: 15 (–) C: 32 (–)	P: 8.2 (–), 5.4–13.6 C: 9.3 (–), 5.4–13.6	P < C (sign.)	KM, KT, EMG	Unclear (ankle, knee, hip, pelvis)	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
Don [26]	Charcot-Marie-Tooth disease (with foot drop and/or plantarflexion failure)	P: 21 (10 m/11 f) C: 21 (10 m/11 f)	P: 40 (14), 15–59 C: 40.1 (10.8), 14–60	–	KM, KT, EMG	Unclear (ankle, knee, hip, pelvis)	Only foot drop: (1) COMP: In ST, increased passive ankle DF (delayed plantarflexor 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
Ferrarin [45]	Charcot-Marie-Tooth disease type 1A with foot drop and/or push-off deficit)	P: 21 (12 m/9 f) C: 18 (9 m/9 f)	P: 11.9 (2.8), – C: 11 (3.3), –	P = C (matched)	KM, KT	Affected (ankle, knee, hip, pelvis, trunk)	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)
Frigo [27]	Juvenile chronic arthritis	P: 19 (4 m/15 f) C: 13 (–)	P: 11.8 (4), 6–19 C: – (–), 17–24	P = C	KM, KT	Affected and unaffected (ankle, knee, hip)	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

Table 3 (*Continued*)

First author	Diagnosis	Number of subjects (gender)	Age in years: mean (SD), range	Gait speed	Parameters evaluated	Side (level) evaluated	Compensatory mechanisms and secondary deviations
Gutierrez [36]	Myelo-meningocele	P: 31 (18 m/13 f) C: 21 (11 m/10 f)	P: 10.5 (2.6), – C: 10.4 (2.5), –	–	KM, KT	Unclear (ankle, knee, hip, pelvis, trunk)	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
Gutierrez [28]	Myelo-meningocele	P: 30 (–) C: 21 (–)	P: 10.3 (–), 6.8–15.8 C: 10.4 (–), 5.2–14.4	–	KM, KT	Unclear (ankle, knee, hip, pelvis, trunk)	(1) COMP: In early stance, leaning of trunk over the ST leg hip compensated for ipsilateral hip abductor weakness (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
Hurwitz [37]	Unilateral OA of the hip	P: 19 (12 m/7 f) C: 19 (12 m/7 f)	P: 60 (8), – C: 61 (8), –	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
Laborde [42]	Stroke with spastic right hemiplegia	P: 7 (4 m/3 f) C: 5 (5 m/0 f)	P: 42.7 (18.4), – C: 23.5 (2.1), 22–27	–	KM	Affected (ankle, knee, pelvis)	COMP: Patients compensated with pelvic elevation for insufficient flexion of the affected knee ($<30^{\circ}$) in SW in order to assure foot clearance
Lehmann [29]	Hemiparesis (strokes 3–13 years previously)	P: 7 (3 m/4 f) C: 7 (3 m/4 f)	P: – (–), 59–75 C: – (–), 57–73	P = C (matched)	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)
Matjacic [18]	Spinal muscular atrophy, type III	P: 7 (4 m/3 f) C: 9 (6 m/3 f)	P: 39.7 (11), – C: 33.1 (2.7), –	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
Mündermann [43]	Bilateral medial knee OA (less severe [LS] and more severe [MS])	P LS: 19 (6 m/13 f) P MS: 23 (13 m/10 f) C LS: 19 (6 m/13 f) C MS: 23 (13 m/10 f)	P LS: 65.2 (12.5), 36–82 P MS: 65 (8), 49–80 C LS: 61.7 (12.3), 39–86 C MS: 63.7 (9.2), 49–84	P = C	KM, KT	Unclear (ankle, knee, hip, pelvis, trunk)	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
Nadeau [46]	Right chronic patellofemoral pain syndrome (PFPS)	P: 5 (2 m/3 f) C: 5 (2 m/3 f)	P: 28.4 (7.5), – C: 25.5 (13.3), –	P = C	KM, KT	Affected (ankle, knee, hip)	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

Newman [30]	Charcot-Marie-Tooth disease, types I and II	P: 16 (11 m/5 f) C: 40 (21 m/19 f)	P: 20.1 (13), 8–52 C: 18.4 (8.5), 8–32	P < C (sign.)	KM, KT	Affected (ankle, knee, hip, pelvis)	(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
Powers [48]	Patellofemoralpainsyndrome (PFP)	P: 24 (–) C: 18 (–)	P: 25.4 (7.3), – C: 27.6 (4.8), –	P < C (sign.)	KM	Affected (ankle, knee, hip)	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
Riad [38]	Spastichemiplegic CP	P: 99 (60 m/39 f) C: 5 different age groups with each 20–25 subjects	P: 8.4 (–), 4–19.8 C: matched	–	KM, KT	Affected and unaffected (ankle, knee, hip, pelvis)	(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 optimized, but symmetry was preferred as a way of dealing with the movement impairment)
Romano [39]	Unilateral congenital dysplasia of the hip	P: 21 (6 m/15 f) C: 40 (14 m/26 f)	P: 48 (–), 25–71 C: 46 (–), 31–71	P < C (sign.)	KM, KT	Affected and unaffected (ankle, knee, hip, pelvis, trunk)	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
Romkes [10]	Hemiplegic CP	P: 12 (8 m/4 f) C: 10 (5 m/5 f)	P: 12.3 (4.1), – C: 29.5 (3.3), –	–	KM, KT, EMG	Affected (ankle, knee, hip, pelvis)	(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
Sankar [40]	Recurrent deformity following treatment of idiopathic clubfoot	P: 35 (–) C: 31 (–)	P: 6.7 (–), 3.6–15.4 C: – (–), –	–	KM, EMG	Unclear (ankle, knee, hip)	REACT: 80% of children walked with in-toeing gait and many (50%) showed external hip rotation in ST as a result
Saraph [5]	Spastic CP (hemiplegic [HP] and diplegic [DP])	P: 22 (–) (14 HP, 8 DP) C: 20 (–)	P: 11.9 (–), 9.2–15.5 C: – (–), –	–	KM, KT	Affected and unaffected (ankle, knee, hip, pelvis)	REACT: In all phases of gait, the internal rotation deformity of the affected leg caused an external rotation of the hemi-pelvis, 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
Sarwahi [31]	Postoperative flatback	P: 21 (3 m/18 f) C: mentioned but not specified	P: 53 (–), 36–83 C: 45 (–), 25–60	P < C (sign.)	KM, KT	Affected (ankle, knee, hip, pelvis, trunk)	(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 & decreased overall joint flexibility). Then, subjects compensated the anterior CoM position by flexing hips and knees (crouched posture and gait)

Table 3 (*Continued*)

First author	Diagnosis	Number of subjects (gender)	Age in years: mean (SD), range	Gait speed	Parameters evaluated	Side (level) evaluated	Compensatory mechanisms and secondary deviations
Shier-Chieg [49]	Bilateral OA in the medial compartment of the knee (2 groups: mild [MI] and severe [SE])	P MI: 15 (6 m/9 f) P SE: 15 (2 m/13 f) C: 15 (6 m/9 f)	P MI: 63.1 (11.9), – P SE: 63.1 (8.2), – C: 63.2 (9.9), –	P = C (matched)	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
Stebbins [9]	Spastic hemiplegic CP with foot deformity on affected side	P: 12 (6 m/6 f) C: 15 (5 m/10 f)	P: 11.8 (–), 6–14 C: 9.5 (–), 6–14	–	KM, KT, EMG	Affected and unaffected (ankle, knee, hip, pelvis)	(1) REACT: In ST, excessive pelvic rotation (protraction 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
Theologis [32]	Patients with previously treated congenital talipes equinovarus (clubfoot)	P: 20 (15 m/5 f) C: 15 (6 m/9 f)	P: 9.8 (2.3), 6.9–14.6 C: 10.7 (1.8), 7.7–13.1	P = C	KM, KT	Affected and unaffected (ankle, knee, hip, pelvis)	REACT: External rotation of the hip resulted from in-toeing gait (in-toeing persisted in approximately half of the subjects)
Torry [50]	Unilateral ACL deficiency (complete isolated rupture, greater than 2 years prior to measurement)	P: 16 (9 m/7 f) C: 8 (4 m/4 f)	P: 28.1 (12.7), – C: 28.2 (4.3), –	P = C	KM, KT, EMG	Unclear (ankle, knee, hip)	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
Westhoff [41]	Legg Calve Perthes disease (LCPD)	P: 33 (24 m/9 f) C: 30 (14 m/16 f)	P: 8 (2), – C: 8.1 (1.2), –	P < C (sign.)	KM, KT	Affected and unaffected (hip, pelvis)	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
Yavuzer [51]	Hemiplegia (after stroke)	P: 46 (30 m/16 f) C: 46 (30 m/16 f)	P: 58 (7.5), – C: 56.9 (12.6), –	P < C (sign.)	KM, KT	Affected and unaffected (ankle, knee, hip, pelvis)	(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 ipsilateral pelvic/hip hiking with circumduction of the limb or vaulting on the unaffected side

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, knee extension; HS, hamstrings.

Table 4
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).

Biomechanical constraints due to primary pathology	Compensatory mechanisms
Hip extensor weakness	Posterior trunk extension [28] [c]
Hip abductor weakness	Duchenne limp [28,36,37,41] [d,a]
Knee extensor weakness Quadriceps avoidance	Hip extensors (hamstrings) for knee extensors [17,24,33,46,50] [d,a,c] Center of mass anterior of knee joint by: Increased activity of plantarflexion/knee extension couple [17,18,25] [d,a,c] Hip flexion [27,29,51] [d,a] Anterior pelvic tilt [49] Kneehyperextension [35]
Ankle plantarflexor weakness	Eccentric work of hip flexors for progression in stance [44] Hip and knee extensors in stance [26,44] [d,a] Hip flexors (pulling) in preswing [26,34] [d,a] Hip extensor torque strategy in late stance (loading of flexor tissue) [47] [a] Internal rotation of trunk and pelvis on contralateral side [28] Larger symmetrical hip power generation [38]
General leg weakness	Hyperactivity ankle plantarflexors [17,18] [d,a] Co-contraction around knee [17] Prolonged activity of contralateral hip abductors (weight acceptance) [18]
Reduced foot clearance	Pelvic up tilt on unaffected side [34] Pelvic hike [26,28,42,47,51] [d,b] Circumduction, hip abduction, hip external rotation [26,29,30,35,51] [d] Hip flexion and/or knee flexion [35,45] [d] Increased plantarflexion on unaffected side (vaulting) [9,38,45,51] [d,b,c]
Limited hip extension	Lumbar lordosis [37] Knee flexion to allow the pelvis to progress forward [39]
Loss of lumbar lordosis (center of mass anterior)	Hip hyperextension [31] Crouch gait [31] [b]
Rotational knee instability/increased medial knee load	Lateral shift of center of mass (e.g. pelvic hike) [24,43,49] [d,b]
Patella "out of line" (Q-angle increased)	Reduced hip internal rotation [48]
Leg length discrepancy	Hip and knee flexion and ankle dorsiflexion on unaffected (longer) side [23] [b]
Initial toe-contact	Early onset plantarflexors, reduced dorsiflexor activity [10]

[a] Mechanisms that were supported by computer simulation studies.
[b] Mechanisms that were supported by in vivo simulation studies.
[c] Mechanisms that were supported by single-case studies
[d] Mechanisms that appear to be independent from the underlying pathology.

(35%) [18,23,25–27,30,36,39,41,45,51] on three or more levels. From the 27 studies that included the measurement of the ankle, knee and hip joints, nine studies (33%) [9,10,17,18,23,26,27,38,45] reported compensations on the ankle, 15 (56%) [10,17,23–27,31,35,36,39,44,45,50,51] on the knee and 22 (81%) [9,18,23–27,29–31,34–39,44–46,48,50,51] on the hip level. Compensations on the pelvis, respectively trunk level were described in 11 studies (50%) [18,25,26,28,34,37,39,41,42,49,51] of the 22 studies that included the measurement of the pelvis, respectively in five studies (63%) [18,28,36,41,43] 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 4. In addition, Table 5 provides a list of the identified physical effects.

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.

Table 5
Summary of the identified passive physical effects that resulted from gravity during locomotion (based on the 36 systematically reviewed papers).

Biomechanical constraints due to primary pathology	Physical effect
Equinus position of ankle (toe-walking gait)	Anterior pelvic tilt (hip flexion), hip internal rotation and external pelvic rotation (pelvic retraction) [4–6,9]
Internal rotation deformity of foot (in-toeing gait)	Hip external rotation [30,32,40]

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. [76], 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.

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 4 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 [53–56]. 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 [20].

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.

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 [52,57–62]. 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 [63,64]. 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. [24], 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 [24]. 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 [4,5,27,33,34,38,51], 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 [18,28,36,41,43], future studies should focus on investigating secondary deviations in both lower extremities including the pelvis, throughout the spine and in both upper extremities.

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. [3], 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 5). 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 4) 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 [28,36,37,41]. 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 [16]. 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 [17,24,33,46,50], an increased plantarflexion/knee extension couple strategy, i.e. using the triceps surae muscle to move the knee towards extension [17,18,25] 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 [27,29,51]. Computer simulation studies conducted by Catalfamo et al. [13], Liu and Maitland [65] and Goldberg and Neptune [14] as well as a single-case experiment conducted by Siegel et al. [66] 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. [67] and Goldberg and Neptune [14] as well as the single-case experiment conducted by Siegel et al. [66]. The hip flexion strategy was further supported by Tagawa and Yamashita's [16] computer simulation experiment. An additional mechanism to compensate for weak knee extensor muscles was introduced with the single-case experiment by Siegel et al. [66], 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 [26,28,42,47,51], the circumduction, i.e. hip abduction and hip external rotation, or isolated hip abduction or hip external rotation on the affected side [26,29,30,35,51] and vaulting, i.e. an increased ankle plantarflexion during the stance phase on the unaffected side [9,38,45,51]. 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. [68] and Kerrigan et al. [69] 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 [70].

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 [14,71]; hyperactivity of the ankle plantarflexors compensating for general leg weakness in stance [14]; hip flexors compensating for weak plantarflexors in pre-swing by promoting the advancement of the limb [14]; and hip extensor torque strategy in late stance (i.e. loading the flexor tissue) compensating for plantarflexor weakness [15]. 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 [11]; crouch gait compensating for a loss of the lumbar lordosis [72]; a lateral shift of the center of mass compensating for an increased medial knee load [73]; and knee flexion on the longer leg side compensating for leg length discrepancy [68]. A single-case study conducted by Lee et al. [74] 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. [66,75] 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.

4.4. 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 1990s, Perry and Burnfield [1] 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 [2,3], 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.

4.5. 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 [7,9]. 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).

4.6. 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.

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Conflict of interest statement

All authors state that there are no conflicts of interest which might have influenced the preparation of this manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.gaitpost.2012.09.006.

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