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Review Article

Gait parameters associated with untreated developmental dysplasia of the hip: a systematic review

Chao Xu[1*], Xinxin Wen[2*], Wei Wei[1*], Luyu Huang[1], Jian Wang[1], Yabo Yan[1], Wei Lei[1]

[1]Department of Orthopaedics, Xijing Hospital, The Fourth Military Medical University, Xi'an, China; [2]Department of Orthopaedics, No. 463 Hospital of Chinese PLA, Shenyang, China. [*]Equal contributors.

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Abstract: Developmental dysplasia of the hip (DDH) has been linked to functional disability and altered gait patterns in adults. However, specific gait alterations in patients with DDH are unclear. The objective of this study is to investigate gait parameters associated with untreated DDH in otherwise healthy adults. Electronic databases (PubMed and Embase) were searched systematically for articles reporting the gait patterns in patients with untreated DDH. Outcome measures assessed included spatiotemporal parameters, lower limb kinematics and kinetics, plantar pressure and electromyogram (EMG). Four studies were included consisting of 76 patients and 99 healthy controls. The quality of the studies varied, with Quality Index scores ranging from 9 to 15 out of a possible 17. Three-dimensional gait analysis and a repeated measures design were used in all included studies. Results revealed that patients with untreated DDH walked more slowly with shorter steps compared with controls. The untreated DDH patients also demonstrated a lower value of the maximum hip flexion angle, hip extension angle and external hip flexion moment in the diseased limb. Meanwhile, lots of gait parameters have been shown to correlate with the Harris Hip score. No included study attempt to investigate the dynamic EMG and plantar pressure parameters in patients with untreated DDH. Current evidence suggests the existence of specific gait deviations in patients with untreated DDH. Definitive conclusions are hampered by the heterogeneous nature of the participants and small sample sizes of the included studies. Future well-designed studies with a large sample size on this topic are warranted.

Keywords: Developmental dysplasia of the hip (DDH), gait, systematic review, biomechanics, motion

Introduction

Developmental dysplasia of the hip (DDH) is a common orthopedic disorder, referring to a wide spectrum of conditions ranging from mild dysplasia to irreducible hip dislocation [1].

Early detection and intervention for patients with DDH are essential to achieve good results [2, 3]. However, there are still lots of adult DDH patients who did not receive any treatment at their early age. Untreated DDH is closely related to long-term morbidities such as gait deviations, avascular necrosis of the femoral head, degenerative hip osteoarthritis (OA), muscular fatigue and chronic pain [4-6].

Gait analysis is commonly utilized for assessing the walking patterns in different patients [7-10]. These evaluations have been regarded as a useful supplement to clinical and radiologic assessment [7-10]. Analyses of gait patterns have been reported previously for patients who had received different operative treatments for DDH [11-18]. Most of them reported that the patients had an improvement in gait parameters after the therapy, but did not return to the normal level. To our knowledge, only a limited number of studies have investigated gait patterns in untreated DDH patients [19-23].

A systematic review and methodological appraisal is required, to make clear to physicians the current state of the evidence for gait deviations in patients with untreated DDH. Firstly, the information of the gait parameters will help clinicians to improve their understanding of motion performance related to untreated DDH [21] and the consequences of the pathological and mechanical changes in the hip. Secondly, awareness of the gait compensations these patients adopt is related to the treatment choice [19, 20, 22]. Thirdly, if the clinicians want to objectively monitor the progress after a certain treatment by gait analysis, the baseline data before the therapy will be necessary [17-

Table 1. Methodological quality assessment of the included studies

	Q1	Q2	Q3	Q5[a]	Q6	Q7	Q10	Q11	Q12	Q16	Q18	Q20	Q21	Q22	Q25	Q27[b]	Total
Romano et al. [19] (1996)	Y	Y	Y	Y	Y	Y	N	U	U	Y	Y	Y	U	U	Y	Y	12
Lai et al. [21] (1997)	Y	Y	Y	P	Y	Y	N	U	U	Y	Y	Y	U	U	N	N	9
Pederson et al. [22] (2004)	Y	Y	Y	P	Y	Y	Y	U	U	Y	Y	Y	Y	Y	N	Y	13
Jacobsen et al. [20] (2013)	Y	Y	Y	Y	Y	Y	Y	U	U	Y	Y	Y	Y	Y	Y	Y	15

This methodological quality assessment was performed by CX and YBY utilizing a modified version of Downs and Black Quality Index. Y (Yes) = 1, N (No) = 0, U (Unable to determine) = 0. The total score was out of 18. Q1: clear aim/hypothesis, Q2: outcome measures clearly described, Q3: clear patient characteristics, Q5: distributions of principal confounders in each group of subjects clearly described, Q6: clear main findings, Q7: estimates of the random variability provided, Q10: actual probability values provided, Q11: representative of population (asked), Q12: representative of population (prepared to participate), Q16: no data dredging, Q18: use of appropriate statistical tests, Q20: valid and reliable outcome measures, Q21: participants recruited from the same population, Q22: participants recruited over the same period of time, Q25: adjustment for confounding, Q27: sufficient statistical power. [a]This question was scored on the basis of P (partial) = l, Y (Yes) = 2 and N (No) = 0. [b]This question was converted to a yes or no answer to make the comparison simpler.

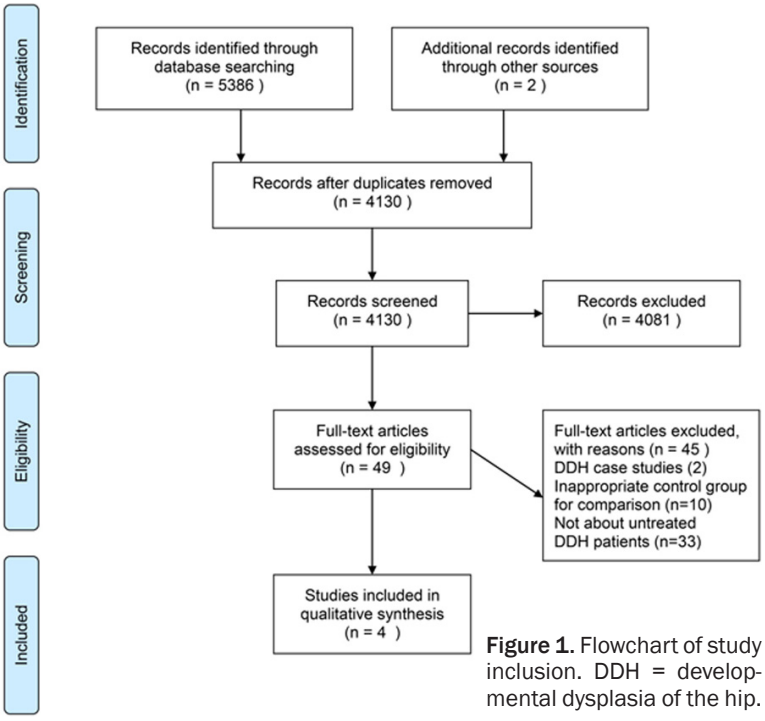

Figure 1. Flowchart of study inclusion. DDH = developmental dysplasia of the hip.

20, 22]. The comparisons of these gait parameters are valuable for doctors to plan, implement and evaluate the clinical managements. The aim of this systematic review was to study the gait parameters in otherwise healthy individuals with untreated DDH compared to healthy controls (HC).

Methods

Literature search strategy

In order to identify the key papers on this topic, comprehensive searches of electronic databases (PubMed and Embase) were performed by three authors (CX, XXW and WW) for all years available up to April 2017. The following search strategy was used in the PubMed database: (Gait[Mesh] OR gait* [All Fields] OR Walking [Mesh] OR walk* [All Fields] OR step [All Fields] OR stride [All Fields] OR cadence [All Fields] OR spatio-temporal [All Fields] OR Pedobarograph* [All Fields] OR electromyograph* [All Fields] OR EMG [All Fields] OR biomechanic* [All Fields] OR kinematic [All Fields] OR kinetic [All Fields] OR "plantar pressure" [All Fields] OR locomotion [All Fields] OR power [All Fields] OR motion [All Fields] OR "ground reaction force" [All Fields] OR moment [All Fields] OR compensation [All Fields] OR adaptation [All Fields]) AND ("Hip Dislocation, Congenital" [Mesh] OR "developmental dysplasia of the hip" [All Fields] OR "congenital dislocation of the hip" [All Fields] OR DDH [All Fields] OR CDH [All Fields] OR "hip dislocation" [All Fields] OR "hip joint instability" [All Fields] OR "congenital hip dislocation" [All Fields] OR "hip dysplasia" [All Fields]). Search strategy applied in Embase was derived from the PubMed search. We also screened the references from key papers to ensure that all key studies were included.

Selection of studies

Evaluation of study eligibility was performed by three authors (CX, XXW and WW). The titles and abstracts retrieved from all identified records

were screened for eligibility utilizing the question: 'Did the research discuss gait parameters in patients with untreated DDH?' Eligible full text articles were then obtained for detailed assessment for the final decision on inclusion according to the following criteria: 1) studies reporting gait parameters in clearly identified untreated DDH group in comparison to HC; 2) outcome measures including spatiotemporal parameters, kinematics, kinetics, plantar pressure parameters, dynamic instability or electromyogram (EMG) results; 3) study population without any systemic disease; 4) studies in the English language.

Assessment of methodological quality

A quality assessment index of 16 items (maximum score of 17) was used to assess the quality of the studies. The assessment was based on the Quality Index (27 items) developed by Downs and Black [24] and items which were not applicable to the included studies were omitted from the analysis [8, 25]. Two reviewers (CX and YBY) independently assessed the quality of each article. After the initial scoring, the assessors met and discussed any differences and obtained a final score (**Table 1**). The initial scoring system was scaled according to an overall score of 17. Therefore, a score of ≤ 7 was rated as low quality, 8-10 as fair quality and >10 as good quality [8].

Data collection

For all included studies, the relevant data were extracted by two authors (LYH and JW), including 1) general study information (author, year, publication type); 2) participant characteristics (sampling size, inclusion and exclusion criteria, population demographics); 3) characteristics of the study methodology (study purpose, study design, gait analysis method); 4) main findings of the gait analysis (spatiotemporal parameters, kinematics, kinetics, plantar pressure and EMG findings). Data were tabulated for ease of comparison and grouping of interested variables. Where data were missing or unreported, authors from the studies were contacted in an attempt to obtain relevant data.

Statistical analysis

Data were synthesized descriptively. Meta-analysis was not performed due to the included researches were lack of homogeneity of participants, study designs and gait parameters of interest.

Results

Search results

With our search we originally identified 4130 unique records. After screening the titles and abstracts, 49 articles were assessed for full text review. Of these, 4 were deemed suitable for inclusion [19-22] (**Figure 1**).

Quality assessment

The quality of the four studies varied, and they scored 9, 12, 13 and 15 respectively out of a possible 17 according to the modified quality assessment instrument (**Table 1**). One study was of fair quality [21] and three studies were of good quality [19, 20, 22]. All studies clearly described participant characteristics (e.g., age, body mass, sex). However, only one study reported sample size calculation [20]. All studies were deemed to correctly perform statistical tests to assess the main findings, but only two of them reported actual probability values (i.e. $P = 0.003$) rather than approximate values (i.e. $P < 0.05$) [20, 22]. It was hard to determine whether or not the participants recruited were representative of the source population in all the included studies. However, all the four studies [19-22] stated the inclusion and exclusion criteria clearly. Sex was reported as a relevant confounder in all the four papers. In addition, Pedersen et al. [22] and Jacobsen et al. [20] also considered age as a confounder, and reduced its effects by matching sex and age when analyzing the data.

Characteristics of included studies

The characteristics of included studies are summarized in **Table 2**. The included studies investigated spatiotemporal parameters (e.g., walking speed, length of stride or step, duration of subphases of the gait cycle), kinematics (e.g., joint angles of the hip, ankle or knee) and kinetics of the lower limb joints (e.g., ground reaction force (GRF), moment or power of the joints) in different phases of the gait cycle during walking or running. None of the included studies investigated the dynamic plantar pressure parameters and dynamic EMG of the lower limb muscles. Three studies performed evaluation of the gait patterns in walking [19, 21, 22]

Table 2. Selected characteristics of included studies

Authors (Year)	Study aim	Study design/Method of gait assessment	Pathological characteristics of the participants	Selection criteria	Measures collected
Romano et al. [19] (1996)	To investigate gait patterns in adult patients with residua of unilateral DDH compared to HC.	CC/Three dimensional gait analysis.	Subjects with different degree of dysplasia, from mild dysplasia to clear coxarthrosis Harris hip score ranging from 27 to 96.	DDH group: included if female patients with residua of untreated DDH; excluded if history of any bone or joint disability; Control group: excluded if history of any abnormality of locomotor system.	Spatiotemporal parameters: walking velocity, foot velocity, length of stride, duration of the stance phase and double support, length of step; kinematic: joint angles of the ankle, knee and hip joints; kinetics: GRF, sagittal and frontal plane moments and power of the ankle, knee and hip joints.
Lai et al. [21] (1997)	To investigate gait patterns in patients with untreated Crown group IV DDH compared to HC.	CC/Three dimensional gait analysis.	9/9 patients with Crown group IV DDH	DDH group: included if female patients with completed dislocation of the hip, no pain; excluded if history of any surgical treatment; Control group: included if healthy women.	Spatiotemporal parameters: walking velocity, step length, cadence, single support time; kinematic: angular changes of pelvic and hip joint; kinetics: GRF, moment of hip and knee.
Pederson et al. [22] (2004)	To investigate gait patterns in adult patients with DDH compared to HC.	CC/Three dimensional gait analysis.	3/14 patients with no, 7/14 with mild and 3/14 with moderate osteoarthrosis. 1/14 patient missing the information.	DDH group: included if female patients with DDH and scheduled for periacetabular osteotomy; Control group: excluded if history of muscular or skeletal diseases.	Spatiotemporal parameters: walking velocity; kinematic: joint angles of the ankle, knee and hip joints; kinetics: internal flexor and extensor joint moments, angular impulse and power of the ankle, knee and hip joints.
Jacobsen et al. [20] (2013)	To investigated differences in walking, running, and self-reported health between DDH and HC.	CC/Three dimensional gait analysis.	32/32 patients with osteoarthritis of grade 0-1.	DDH group: included if planned pelvis operation, osteoarthritis of grade 0-1, age between 18 and 60 years; excluded if history of operation, neurological or rheumatological diseases. Control group: excluded if history of hip, knee, ankle, or back diseases.	Spatiotemporal parameters: walking and running velocity; kinematic: hip extension angle in walking and running; kinetics: hip flexion moment in walking and running.

DDH = developmental dysplasia of the hip, HC = healthy controls, CC = case control study, GRF = ground reaction force.

Table 3. Characteristics of study participants in included studies

Authors (Year)	Cases									HC							
	DDH (n =)	Unilateral (n =)	Age (years)			Female (n =)	BM (kg)			HC (n =)	Age (years)			Female (n =)	BM (kg)		
			Mean	SD	Range		Mean	SD	Range		Mean	SD	Range		Mean	SD	Range
Romano et al. [19] (1996)	21	21	48.1	14.8	25-71	15	65.4	10.2	42-82	40	46	-	31-71	26	56.9	11.3	-
Lai et al. [21] (1997)	9	6	31.4	8.6	21-43	9	49.3	5.1	43-58	15	27.8	-	23-32	15	50.3	-	41-63
Pederson et al. [22] (2004)	14	6	39[a]	-	24-50	14	70[a]	-	50-83	12	35[a]	-	24-56	12	67[a]	-	54-81
Jacobsen et al. [20] (2013)	32	8	34	-	18-53	26	22[a,b]	-	15-29[b]	32	33	-	18-54	26	22[a,b]	-	16-31[b]

Values are expressed as means ± standard deviation. DDH = developmental dysplasia of the hip, HC = healthy controls, BM = body mass, - = not reported. [a]Values are median. [b]Values are body mass index.

Table 4. Main findings of the spatiotemporal parameters in untreated DDH compared with HC

Spatiotemporal parameters	Authors (Year)	DDH (n =)	HC (n =)	Main findings
Walking speed	Romano et al. [19] (1996)	21	40	DDH walked slower than HC.
	Lai et al. [21] (1997)	9	15	DDH walked slower than HC.
	Pederson et al. [22] (2004)	14	12	No significant difference (4.5 km/h required).
	Jacobsen et al. [20] (2013)	32	32	DDH walked slower than HC.
Running speed	Jacobsen et al. [20] (2013)	32	32	No significant difference between the DDH and HC.
Step length	Romano et al. [19] (1996)	21	40	Both the diseased and unaffected sides had shorter step length than HC.
	Lai et al. [21] (1997)	9	15	The diseased side walked with shorter step length than the unaffected side and HC.
Stride length	Romano et al. [19] (1996)	21	40	DDH walked with shorter stride length than HC.
Walking cadence	Lai et al. [21] (1997)	9	15	No significant difference between the DDH and HC.
Duration of stride cycle	Romano et al. [19] (1996)	21	40	No significant difference between the DDH and HC.
Duration of stance phase	Romano et al. [19] (1996)	21	40	No significant difference between the affected limb and HC. The unaffected side had a longer stance phase than the affected side and HC.
	Jacobsen et al. [20] (2013)	32	32	No significant difference between the affected limb and HC.
Double support time	Romano et al. [19] (1996)	21	40	The diseased side had a longer double support time than HC.
Single support time	Lai et al. [21] (1997)	9	15	The diseased side had a shorter single support time than the unaffected side and HC.
Foot velocity	Romano et al. [19] (1996)	21	40	Both the diseased and unaffected sides had slower foot velocity than HC.
	Romano et al. [19] (1996)	21	40	The diseased side had a slower foot velocity than the unaffected side.

DDH = developmental dysplasia of the hip, HC = healthy controls.

and the remaining one was in walking and running [20]. All studies utilized a case – control study design and clearly reported their study aims, pathological characteristics of the participants and selection criteria.

Characteristics of included participants

Table 3 presents age, body mass and sex characteristics of the research participants, including a total of 175 subjects (76 patients, 99 controls). In the included studies, the DDH group comprised predominantly women, and two studies only included female subjects [21, 22]. The included studies compared DDH patients with sex-matched or age- and sex-matched HC.

Spatiotemporal parameters

Table 4 outlines the main findings of the spatiotemporal parameters. Three of the four studies reported that DDH patients, who performed the trials at self-selected pace, walked slower than HC [19-21]. The remaining study required all participants to walk at approximately the same speed (4.5 km/h) [22]. Two studies reported DDH walked with shorter step length than HC [19, 21]. In addition, they also found that the diseased limb had a longer double support time and shorter single support time than HC. In term of the duration of the stance phase, two studies reported no significant difference between the affected limb and HC [19, 20].

Joint kinematics

All the included studies reported the lower limb kinematic results (**Table 5**). Each study focused on some phases of the gait cycle. Diseased limb of DDH exhibited reduced hip flexion angle [19, 21, 22] and hip extension angle [19, 20] when compared with HC. Meanwhile, the diseased limb also demonstrated greater knee flexion angle during the stance phase [22] and smaller knee flexion angle during the swing phase [19]. One study reported that the diseased limb had a greater maximum internal rotation angle of the hip during the gait cycle [21]. However, another study reported that the diseased hip had a greater maximum external rotation angle and the unaffected hip had a greater maximum internal rotation angle during the entire gait cycle than HC [19]. Lai et al. [21] investigated the pelvis kinematics and found that the patients had less maximum anterior tilting of pelvis. They also demonstrated that the diseased side of the pelvis in unilateral DDH stayed lower than the unaffected side throughout the gait cycle. Romano et al. [19] reported the similar result.

Table 5. Main findings of the kinematic parameters in untreated DDH compared with HC

Kinematic parameters (phases of gait cycle)	Authors (Year)	DDH (n =)	HC (n =)	Main findings
Maximum hip flexion angle (single limb stance phase)	Romano et al. [19] (1996)	21	40	The diseased hip had a smaller maximum flexion angle than HC.
Maximum hip flexion angle (entire gait cycle)	Lai et al. [21] (1997)	9	15	The diseased hip had a smaller maximum flexion angle than HC.
Maximum hip flexion angle (stance phase)	Pederson et al. [22] (2004)	14	12	The diseased hip had a trend toward smaller maximum flexion angle than HC, but did not reach the significant level.
Maximum hip extension angle (single limb stance phase)	Romano et al. [19] (1996)	21	40	The diseased hip showed a reduction of maximum extension angle than HC.
Maximum hip extension angle (stance phase)	Jacobsen et al. [20] (2013)	32	32	The diseased hip showed a reduction of maximum extension angle than HC when walking. No significant difference in the maximum hip extension angle between the affected hip and HC when running.
Maximum hip external rotation angle (entire gait cycle)	Romano et al. [19] (1996)	21	40	The diseased hip had a greater maximum external rotation angle than HC.
Maximum hip internal rotation angle (entire gait cycle)	Romano et al. [19] (1996)	21	40	The unaffected hip had a greater maximum internal rotation angle than HC.
	Lai et al. [21] (1997)	9	15	The diseased hip had a greater maximum internal rotation angle than HC.
Maximum hip adduction angle (entire gait cycle)	Romano et al. [19] (1996)	21	40	The diseased hip had a greater maximum adduction angle than HC.
Maximum knee flexion angle (swing phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a smaller maximum knee flexion angle than HC.
Maximum knee flexion angle (second half of stance phase)	Pederson et al. [22] (2004)	14	12	The diseased limb had a greater maximum knee flexion angle than HC.
Maximum ankle plantar flexion angle (swing phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a smaller maximum ankle plantar flexion angle than HC.
Maximum ankle dorsiflexion angle (second half of stance phase)	Pederson et al. [22] (2004)	14	12	The diseased limb had a greater maximum ankle dorsiflexion angle than HC.
Maximum pelvis tilting (entire gait cycle)	Lai et al. [21] (1997)	9	15	The diseased limb had less maximum anterior tilting of pelvis than HC.
Pelvis shift (entire gait cycle)	Romano et al. [19] (1996)	21	40	The diseased side in unilateral DDH patients showed an increased pelvis drop during the stance phase than the unaffected side.
	Lai et al. [21] (1997)	9	15	The diseased side in unilateral DDH patients showed an increased pelvis drop during the stance phase than the unaffected side.

DDH = developmental dysplasia of the hip, HC = healthy controls.

Joint kinetics

All the included studies reported kinetic variables (**Tables 6**). According to Lai et al.'s study [21], both the maximum external knee adduction moment and maximum external hip adduction moment were increased in the unaffected limb and were reduced in the diseased limb compared to HC. The diseased limb also showed a less maximum external extension moment of the hip [19, 20, 22] and less maximum external flexion moment of the knee than HC [19]. Notably, the results for maximum hip flexion moment were inconsistent. Romano et al. [19] reported a lower value in the diseased limb compared to HC; while Lai et al. [21] reported no difference between the two groups. Two studies investigated the GRF of the DDH patients and HC [19, 21]. The results demonstrated that the diseased limb had a shift of GRF to the lateral side in the mediolateral plane [21] and had a reduced GRF than controls [19]. In term of power, two studies reported the diseased limb had less peak hip and knee power than HC [19, 22].

Discussion

To our knowledge, this is the first systematic review of studies investigating the gait patterns exclusively in untreated DDH patients compared to HC. The aim of this review was to assess the gait pattern differences between untreated DDH and HC. Our findings indicate that individuals with DDH differ to HC on specific gait parameters, likely correlating to the pain, leg-length discrepancy (LLD), hip OA and initial pathologic changes [19-22, 26-28]. However, it

Table 6. Main findings of the kinetic parameters in untreated DDH compared with HC

Kinetic parameters	Authors (Year)	DDH (n =)	HC (n =)	Main findings
Maximum external hip flexion moment (stance phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a less maximum flexion moment of the hip than HC.
Maximum external hip flexion moment (entire gait cycle)	Lai et al. [21] (1997)	9	15	No significant difference between the diseased limb and HC.
Maximum external hip extension moment (stance phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a less maximum extension moment of the hip than HC.
	Pederson et al. [22] (2004)	14	12	The diseased limb had a less maximum extension moment of the hip than HC.
	Jacobsen et al. [20] (2013)	32	32	The diseased limb had a less maximum extension moment of the hip than HC when walking and running.
Maximum external hip adduction moment (entire gait cycle)	Lai et al. [21] (1997)	9	15	The diseased limb had a less and unaffected limb had a greater maximum adduction moment of the hip than HC.
Maximum external knee flexion moment (stance phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a less maximum flexion moment of the knee than HC.
Maximum external knee adduction moment (entire gait cycle)	Lai et al. [21] (1997)	9	15	The diseased limb had a less and the unaffected limb had a greater maximum adduction moment of the knee than HC.
GRF (stance phase)	Romano et al. [19] (1996)	32	32	The diseased limb had a reduced GRF than HC.
	Lai et al. [21] (1997)	9	15	The diseased limb had a shift of the GRF to the lateral side in the mediolateral plane.
Peak hip power (late stance-to-early swing phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a less peak of power absorption and generation in the hip joint than HC.
	Pederson et al. [22] (2004)	14	12	The diseased limb had a less peak of power absorption and generation in the hip joint than HC.
Peak knee power (middle-to-late stance phase)	Romano et al. [19] (1996)	21	40	The diseased limb had a less peak of power absorption in the knee joint than HC.
	Pederson et al. [22] (2004)	14	12	The diseased limb had a less peak of power absorption and generation in the knee joint than HC.
Peak ankle power (stance phase)	Pederson et al. [22] (2004)	14	12	No significant difference between the affected limb and HC.

DDH = developmental dysplasia of the hip, HC = healthy controls, GRF = ground reaction force.

must be emphasized that the findings should be interpreted with care due to the heterogeneous nature of the included participants and of the research methods utilized in the studies reviewed.

Regarding spatiotemporal gait parameters, three studies demonstrated that DDH patients walked slower than HC [19-21]. Meanwhile, Lai et al. [21] reported that the cadence was not significantly different between the two groups, which indicate that the decreased walking speed was mainly attributed to the shorter step length [19, 21]. In addition, Romano et al. [19] reported that the diseased limb had a longer double support time, slower foot velocity and shorter stride length than HC. All these findings indicate that the progression of the lower extremities in the patient group was not as speedy as that of the HC group. We assumed that these changes are a protective mechanism for relieving the loads and pain on the diseased hip [16, 29]. In Pedersen et al.'s study [22], the participants performed the gait test in a fixed speed. Some researchers believed that an altered gait pattern can occur when subjects are required to walk at a fixed speed [30, 31], which should be taken into account when interpreting the results.

In Jacobsen et al.'s study [20], the authors also investigated the gait parameters in running and found no difference in running speed, hip flexion moment and hip extension angle between the DDH patients and HC. The negative results may be attributed to the use of Bonferroni correction of the *P* value [20], different running patterns and paces of the participants, different magnitudes of the net joint moments between running and walking and smaller percentage of the running cycle taken up by the stance phase [32]. The authors admitted that evaluation of

Table 7. Main findings of the correlation between the gait parameters and Harris score in Romano et al.'s study

Gait parameters	Gait phase	r value	*P* value
Drop of pelvis on the diseased side	Stance phase	0.75	≤ 0.01
Difference between the duration of the stance phase on the diseased and unaffected side	Stance phase	-0.63	< 0.01
Foot velocity on the diseased side	Gait cycle	0.62	< 0.01
Difference between the foot velocity on the diseased side and unaffected side	Gait cycle	-0.67	< 0.01
Duration of the stance phase on the unaffected side	Stance phase	-0.53	≤ 0.05
Maximum range of motion about hip	Gait cycle	0.86	≤ 0.01
Maximum hip extension angle Maximum knee flexion angle	Gait cycle	-0.74	≤ 0.01
	Swing phase	0.79	≤ 0.01
Maximum ankle dorsiflexion angle	Swing phase	0.67	≤ 0.01
Maximum hip extension moment	Gait cycle	-0.8	≤ 0.01
Maximum knee flexion moment	Gait cycle	-0.78	≤ 0.01
Maximum hip power absorption	Middle-to-late stance phase	0.82	≤ 0.01
Maximum hip power generation	Late stance-to-early swing phase	0.85	≤ 0.01
Maximum knee power absorption	Gait cycle	0.77	≤ 0.01

gait patterns just in walking seemed to be sufficient and adequate in DDH patients [20].

Two studies [19, 20] observed a decreased hip extension angle in patients with DDH during walking. Romano et al. [19] also found a less maximum hip extension moment in the diseased limb compared to HC. The maximum hip extension angle and hip extension moment have been reported to be correlated with propulsion of the body [33, 34]. Less maximum hip extension angle is reflected in slower walking speed and shorter stride length [34], which were consistent with the above findings. As we know, the DDH patients have insufficient joint coverage which will be painful when it is loaded [35]. Therefore, the reduced hip extension angle and hip extension moment could be interpreted as an attempt to unload the hip joint and relieve the pain [20, 33, 36]. Pedersen et al. [22] demonstrated an increased dorsiflexion angle of ankle and flexion angle of knee joint during stance phase, which can possibly delay and reduce the hip extension angle [22] and may also be a pain-avoidance maneuver. A smaller knee flexion angle during the swing phase was reported by Romano et al. [19]. In fact, it is largely passive, an inertial by-product of reduced hip flexion angle during the swing phase [37]. In patients with low Harris scores in Romano et al.'s research [19], the diseased hip had a greater maximum external rotation angle during the entire gait cycle than HC. The authors deduced that the asymmetrical rotation of the pelvis, which kept the diseased side in front of the contralateral one, may result in a prolonged external rotation of the affected limb. However, in Lai et al.'s report [21], the diseased limb manifested a greater internal rotation angle throughout the gait cycle. The inconsistency may be attributed to the heterogeneity of the patients. In Lai et al.'s study [21], all the patients were Crown IV dislocation of the hip, the authors inferred that the dislocated hip becomes more stable and the progression becomes more efficient in the internal rotation position [21]. In addition, Romano et al. [19] and Lai et al. [21] reported that the diseased side showed an increased pelvis drop during the stance phase than the unaffected one in the patients with unilateral DDH. LLD in Lai et al.'s study (mean, 4.6 cm) may account for this inconsistence [29].

Some researchers have reported that the external hip extension moment was reduced postoperatively and attributed it to the surgical procedures involving hip flexors [16, 38-41]. While three included studies demonstrated a lower value of the maximum external hip extension moment [19, 20, 22] in the diseased limb of untreated DDH, which indicated that the changes of the hip movement in the sagital plane have already existed to some extent preoperatively. Romano et al. [19] interpreted that this deviation was a protective mechanism to diminish the loads on the diseased hip. Lai et al. [21] reported a lateral shift of GRF in the patient group, which was also observed by other researchers [29, 42]. The researchers explained it by the LLD. However, Chang et al. [16] demonstrated the existence of the lateral shift of GRF

in DDH patients without LLD. Thus, there may be other factors at work. Further studies are needed to clarify this topic. Romano et al. [19] reported a smaller GRF in the diseased limb and interpreted this deviation as a protective adaptation. Two studies reported a reduced peak power in the hip and knee of the diseased side [19, 22], which could be viewed as resulting from the reduced joint moments, slower walking speed and less propulsion of the diseased limb.

Pedersen et al. [22] investigated the correlations between the Tönnis score, the central-edge angle and the dynamic gait parameters, and no obvious correlation was found. As all patients in their study felt pain, the authors speculated pain was an important reason for gait deviations, which is consistent with Jacobsen et al.'s findings [20]. In another study, Romano et al. [19] found lots of gait parameters correlated with the Harris Hip score (**Table 7**), and concluded the pain and altered proprioceptive input were the main reasons for the gait compensations [15, 19, 37]. Patients complaining of pain were excluded from Lai et al.'s study and the mean LLD of their patients was 4.6 cm [21]; therefore, LLD was considered as the primary reason for the compensatory gait deviations in the study. We concluded that relieving pain and equalizing LLD may be effective to improve the gait performance. In addition, based on the experimental findings and theoretical analyses, strategies to improve hip muscle power and pelvis stability preoperatively and postoperatively will be necessary to achieve a favorable treatment outcome [19, 38, 43-45]. Further prospective research would be helpful to verify these hypotheses.

Maeyama *et al.* investigated the dynamic instability of the unilateral DDH patients by using triaxial accelerometry [23]. The results demonstrated that the overall magnitude of acceleration of the diseased hips was significantly greater than that of the contralateral hips, which meant a less stable gait pattern in the affected side. In addition, the authors found a significant association between hip instability and the degree of hip dysplasia. This study provided a new perspective for comprehensive understanding of the gait deviations in patients with untreated DDH. However, it was not included in our review because the data of untreated DDH group were not compared with that of the HC group. Conclusions drawn when taking the unaffected hip in unilateral DDH as normal control should be taken with some reserve [16, 19, 21, 35, 46].

To our knowledge, there are no published studies evaluating the gait patters in children or adolescent with untreated DDH. As lots of interventions are performed in these populations, the relevant gait information is important for the doctors to plan, implement and monitor the treatment. Future studies are warranted to address this issue.

The results of this systematic review should be interpreted in light of some limitations. Firstly, although extensive literature search was performed, the number of included studies and the sample sizes were small, and thus, the outcomes may have been due to lack of statistical power. Secondly, the participant resource was not described clearly in two of the four included studies [19, 21], which may affect their generalisability. Thirdly, the age and stage of pathological changes of the participants were heterogeneous among studies due to different inclusion and exclusion criteria. Fourthly, reporting of gait parameters was across the entire gait cycle in some studies and confined to subphases of the gait cycle in other studies. Finally, different systems were used for data collection and analysis, which may impact on the data comparability [7].

Current evidence from the literature indicates that DDH patients exhibit significant gait deviations compared to HC. We encourage future randomized controlled trial in DDH to assess the differences between the pre- and post-treatment gait parameters, which can help physicians to evaluate the effect of the treatment. Meanwhile, future studies investigating the dynamic EMG and plantar pressure parameters on a large sample size are required in order to comprehensive understand the gait characteristics of the untreated DDH patients.

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Disclosure of conflict of interest

None.

Address correspondence to: Dr. Yabo Yan and Wei Lei, Department of Orthopedics, Xijing Hospital, Fourth Military Medical University, 15 Changle West Road, Xi'an 710032, Shaanxi, China. Tel: +862984771011; Fax: +862984772132; E-mail: giamyimpoh@hotmail.com (YBY); leiwei@fmmu.edu.cn (WL)

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