

Walking pattern in adults with congenital hip dysplasia

14 women examined by inverse dynamics

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Background Knowledge of the gait dynamics in patients with hip dysplasia may help to understand the consequences of the mechanical changes in the hip.

Patients and methods We evaluated the walking pattern of 14 women with hip dysplasia (median age 39 (24–50) years) before periacetabular osteotomy and compared it with a control group of 12 healthy women (median age 35 (24–56) years). Data were collected using five video cameras and two force plates. We used an inverse dynamics approach to calculate sagittal joint angles, moments and power in the stance phase.

Results The patient group showed increased dorsiflexion of the ankle and knee flexion in the second half of the stance phase. The hip flexor joint moment in the patient group was both delayed and reduced in the second half of the stance phase. Moreover, the angular impulse of the hip flexors was significantly reduced in the patient group.

Interpretation The pathological deformities of hip dysplasia may explain both the kinematic and kinetic gait deviations between the patient group and the control group in the second half of the stance phase. We suggest that insufficient cover of the femoral head leads to a reduced hip flexor moment and an increased flexion in the ankle, knee and hip joints.

(Trousdale et al. 1995). The pathological changes consist of reduced covering of the femoral head proximally, anteriorly and laterally and therefore an insufficient load-bearing surface of the femoral head (Hadley et al. 1990, Murphy et al. 1990). Several studies assert that osteoarthritis in close to 50% of cases is caused by dysplasia of the hip (Aronson 1986, Weinstein 1997).

Gait analysis is commonly used for evaluation of the walking pattern in various patient groups, including patients with hip osteoarthritis (Romano et al. 1996, Hurwitz et al. 1997, Lai et al. 1997, Catani et al. 1998, Tanaka 1998, Watanabe et al. 1999, Perron et al. 2000). In most of these studies, the underlying cause of osteoarthritis is not mentioned. Knowledge about the dynamics of gait in patients with dysplasia of the hip is important for an understanding of the consequences of the mechanical changes in the hip joint. Moreover, determining which gait compensations these patients adopt is relevant for management and evaluation of various treatments, such as physical therapy and periacetabular osteotomy.

We analyzed the walking pattern of a group of adult women with dysplasia of the hip before periacetabular osteotomy. Their walking pattern was compared with that of a group of healthy women. Both joint angles and inverse dynamics calculations of joint moments and power around the ankle, knee and hip joints in the sagittal plane were used to quantify the walking patterns in the two groups. We also evaluated any possible associations between changes in selected gait param-

Congenital dysplasia of the hip is a disease characterized by pathological changes in the hip joint. If diagnosed early and treated according to schedule, the prognosis is good, but if diagnosed late or left untreated it will lead to early osteoarthritis

eters, the radiographic signs of degeneration and the CE-angle.

Material and methods

Subjects

We analyzed the gait pattern of 14 women with known dysplasia of the hip who had a median age of 39 (24–50) years and compared it with a control group consisting of 12 healthy women having a median age of 35 (24–56) years. All 14 women were patients in the Department of Orthopedics, Aarhus University Hospital, Denmark. During 7 months from June 2000 to January 2001, 19 women diagnosed with dysplasia of the hip and scheduled for periacetabular osteotomy were contacted. 14 agreed to participate in our study. Patients with secondary dysplasia due to other hip diseases, such as Calvé-Perthes or epiphysiolysis, were excluded. The median height and weight in the patient group were 169 (154–174) cm and 70 (50–83) kg, respectively. The median CE-angle was 8 (–13–18) degrees. We used the hip score developed by Tönnis (1984) to measure joint degeneration. 3 women had 0 osteoarthritis, 7 scored I (mild osteoarthritis) and 3 scored II (moderate osteoarthritis). Information about osteoarthritis was missing for 1 woman. 6 women had unilateral and 8 had bilateral dysplasia. In the latter cases, gait analysis was performed on the hip where periacetabular osteotomy was planned. When the patient group had been selected, the control group was recruited among students at the University of Copenhagen and employees at Copenhagen University Hospital. The median weight and height in the control group were 67 (54–81) kg and 169 (162–173) cm, respectively. The subjects in the control group stated that they had no known muscular or skeletal abnormalities of the locomotor system, but they were not examined clinically or radiographically. All subjects gave their informed consent to participation in the study, which was approved by the local ethics committee.

Gait analysis

The subjects were asked to walk across two force platforms (AMTI, OR6-5-1) at a speed of 4.5 km/h.

The speed was controlled by photocells, which made it possible to teach the subjects to walk at approximately 4.5 km/h. 15 small reflective spherical markers (12 mm diameter) were placed on the subjects according to the manufacturer's set-up described by Vaughan et al. (1992). The markers were placed on the fifth metatarsal, the heel, the lateral malleolus, the tibial tubercle, the lateral femoral epicondyle, the greater trochanter, the anterior superior iliac spine and sacrum. All subjects wore lightweight flexible shoes with a thin flat sole. We used 5 video cameras (Panasonic WV-GL350) operating at 50 Hz to record the movements. Synchronization between the video signals and the force plate signals was obtained electronically with a custom-built device. This device put a visual marker on one video field from all cameras and at the same time triggered the analogue to digital converter that sampled the force plate signal at 1,000 Hz. The subjects triggered the data sampling and synchronization when they passed a photocell. The video sequences were digitized and stored. 16 non-coplanar points on a standard calibration frame (Peak Performance 5) were digitized to calibrate the video sequences. The calibration frame was placed in the measuring volume and covered both force plates. Three-dimensional coordinates were then reconstructed by direct linear transformation, using the Arial Performance Analysis System (APAS). Before calculation, the position data were digitally low-pass filtered by a fourth-order Butterworth filter with a cut-off frequency of 6 Hz, and the 1,000 Hz force plate signals were reduced to fit the 50 Hz video signals.

Calculations

Joint angles of the ankle, knee and hip joints were calculated to describe the movements in the sagittal plane. In a neutral upright position, the angle between the foot and shank is 90 degrees and the angles between the shank and thigh and between the thigh and pelvis are 180 degrees. Joint angle values lower than 90 and 180 degrees indicate dorsiflexion of the ankle and flexion of the hip and knee joints, respectively. Internal flexor and extensor joint moments about the ankle, knee and hip were calculated, using the three-dimensional inverse dynamics approach described by Vaughan et al. (1992). Joint moments were considered posi-

tive when the pulling direction was counterclockwise, meaning that ankle dorsiflexor, knee extensor and hip flexor moments were positive. Internal net joint moments provide information about whether flexor or extensor muscles dominate the joints. The angular impulse (Nm.s) was calculated by integrating the area under the joint moment curves. The angular impulse quantifies the total contribution of a joint moment towards producing movement. Joint power was calculated by multiplying the joint moments and the joint angular velocity. Power reflects whether the muscles generate (positive) or absorb (negative) energy.

Normalization and data reduction

Data of the affected limb in the patient group and the right limb of the control group were analyzed. 6 gait cycles were normalized and averaged in each subject. The gait cycles were normalized to 500 sample points by interpolating each cycle. Only the stance phase was analyzed. Ensemble averages were then calculated for the patient group and the control group, using the individual subject means.

Statistics

To detect differences between the two groups, peak values of joint angles, joint moments and power were tested statistically, using the Student's t-test. The level of significance was set at 5%. The results are presented as means (CI). The correlation between statistically significant peak values for the joint angles and joint moments, the radiographic signs of degeneration and the CE-angle were analyzed by use of Spearman's rank correlation.

Results

There were no differences in walking speed between the two groups ($p = 0.99$). The walking speeds were 4.5 (0.02) km/h in the patient group and 4.5 (0.04) km/h in the control group.

Joint angles (Figure 1)

We tested the peak values A1 and A2 in the ankle, K1 to K3 in the knee and H1 in the hip for statistical significance (Table). Statistically significant differences were seen in the ankle and knee joint angles. The mean knee angle for the entire stance phase

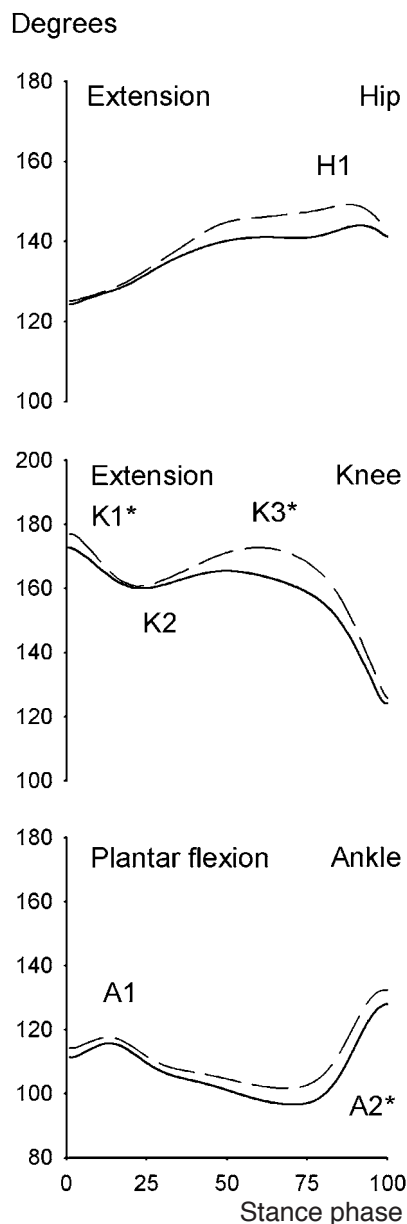


Figure 1. Average joint angles (degrees) of the ankle, knee and hip in the patients (solid line) and controls (dashed line). Zero on the x-axis indicates heel strike and 100 toe-off. Increases in joint angle values indicate plantar flexion and knee and hip extension. The points A1, A2, K1 and K2, etc., represent the various peak values used in the statistical analyses. Asterisks indicate statistically significant differences in peak joint angles between the two groups.

was 159 (2) degrees in the patient group and 163 (3) degrees in the control group ($p = 0.01$). The mean angle of the ankle and hip joint during the entire

stance phase showed no significant differences between the two groups ($p = 0.06$ for the ankle joint and $p = 0.4$ for the hip joint). We found no correlation between the statistically significant peak values A2, K1, K3 and the clinical parameters related to hip dysplasia. The correlation coefficients for A2 and the CE-angle and the Tönnis score were $r = 0.001$ ($p = 1$) and $r = -0.07$ ($p = 0.8$), respectively. As regards the knee joint angles, the correlation coefficients between the significant peak values and the CE-angle were: K1 $r = 0.05$ ($p = 0.8$) and K3 $r = 0.002$ ($p = 1$). The correlation between the Tönnis score and the same peak values were: K1 $r = 0.03$ ($p = 0.9$) and K3 $r = -0.14$ ($p = 0.6$).

Joint moments (Figures 2 and 3)

There were statistically significant differences between the two groups in the ankle and hip joint moments (Table). In addition to the reduced peak hip flexor moment, the patient group also shifted from extensor dominance to flexor dominance later in the stance phase, as compared to the control group ($p = 0.02$). The hip extensor-flexor moment shift was observed at 53 % of the stance phase in the patient group, while it was observed at 43 % of the stance phase in the control group.

As regards the angular impulse for the ankle and knee joint, there were no differences in either the total flexor or the total extensor joint moments during the stance phase (Figure 3). The angular impulse of the hip joint moment curve showed a significant difference between the two groups ($p < 0.001$). The angular impulse of the total flexor joint moment was 7 Nms (2) in the patient group and 13 Nms (2) in the control group. As for the joint angles, no correlations were found between the radiographic signs of degeneration, the CE-angle and the statistically significant peak values for the joint moments and the angular impulse for the hip flexor moment. The correlation coefficients for the CE-angle and the peak values were: A1 $r = 0.45$ ($p = 0.10$) and H2 $r = 0.56$ ($p = 0.04$). The correlation coefficients for the Tönnis score and the peak values were: A1 $r = -0.15$ ($p = 0.61$) and H2 $r = -0.19$ ($p = 0.50$). The correlation coefficient between the angular impulse of the hip flexor moment, the CE-angle and Tönnis score were $r = 0.03$ ($p = 0.09$) and $r = 0.22$ ($p = 0.44$), respectively.

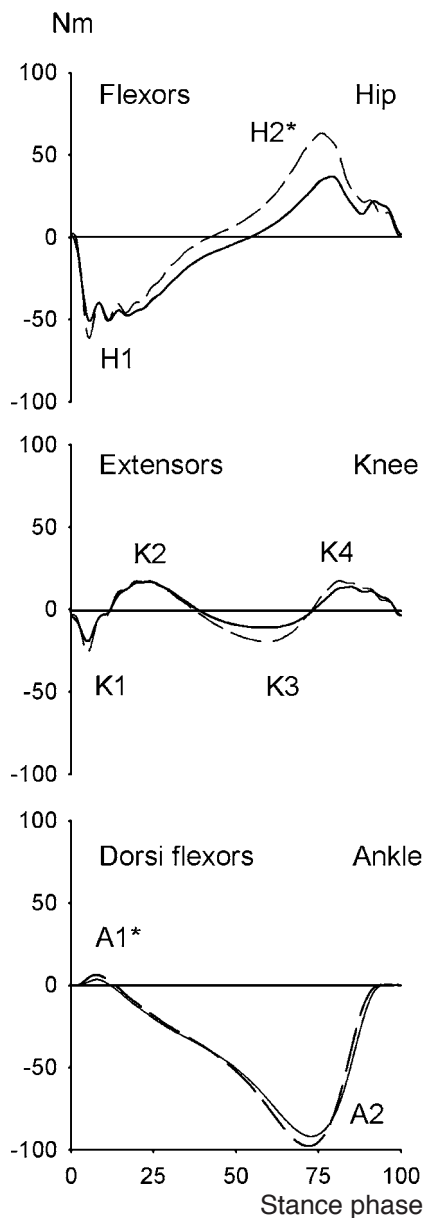


Figure 2. Average ankle, knee and hip joint moments in the patients (solid line) and controls (dashed line). Zero on the x-axis indicates heel strike and 100 toe-off. An increase in the joint moment values indicates dorsiflexor dominance in the ankle joint, extensor dominance in the knee joint and flexor dominance in the hip joint. The points A1, A2, K1 and K2, etc., represent the various peak values used in the statistical analyses. Asterisks indicate statistically significant differences in peak joint moments between the 2 groups.

Power (Figure 4)

In the ankle joint, we found no differences in peak values between the patient group and the control

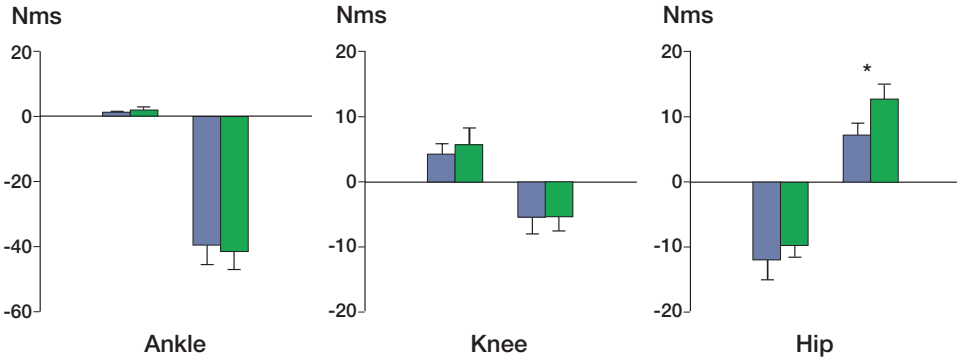


Figure 3. Angular impulse values means (CI) in the patients (blue bars) and controls (green bars). Asterisks indicate statistically significant differences between the two groups. Positive values indicate the total amount of dorsiflexor dominance in the ankle, extensor dominance in the knee and flexor dominance in the hip. Negative values indicate the total amount of plantar flexor dominance in the ankle, flexor dominance in the knee and extensor dominance in the hip.

Peak values of joint angles, joint moments and power in patients and control subjects

Peak joint angles (degrees)							Peak joint moments (Nm)							Peak power (Watts)						
Patients			Controls				Patients			Controls				Patients			Controls			
a	b	c	a	b	c	d	a	b	c	a	b	c	d	a	b	c	a	b	c	d
Ankle																				
A1	117	3	A1	118	2	0.8	A1	4	2	A1	7	3	0.03	A1	−50	13	A1	−45	10	0.5
A2	96	3	A2	102	4	0.02	A2	−91	11	A2	−97	9	0.3	A2	247	43	A2	245	49	1.0
Knee																				
K1	173	2	K1	177	2	0.01	K1	−21	4	K1	−26	4	0.08	K1	38	8	K1	62	11	<0.001
K2	159	5	K2	161	2	0.4	K2	15	11	K2	18	8	0.7	K2	−66	13	K2	−93	23	0.03
K3	166	3	K3	173	3	0.001	K3	−12	5	K3	−20	8	0.05							
							K4	17	5	K4	20	5	0.3							
Hip																				
H1	145	8	H1	150	6	0.3	H1	−59	10	H1	−63	7	0.5	H1	66	16	H1	53	10	0.1
							H2	40	7	H2	64	9	<0.001	H2	−23	7	H2	−25	8	0.7
														H3	19	5	H3	34	7	0.001
a peak, b mean, c CI, d p-value																				

group (Table). In the knee joint, significant differences were seen at K1 and K2. In the hip joint, differences were seen in H3 in the second half of the stance phase.

Discussion

Several factors such as walking speed and footwear, affect the time course pattern and the magnitude of the joint moments during walking (Kirtley et al. 1985, Bergmann et al. 1995, Cooper et al. 1996, Bohannon 1997, Holden et al. 1997). Very often subjects are allowed to walk

at a self-selected speed and scanty information is given about their footwear. These factors must be standardized if gait analysis can be used as a clinical tool. When evaluating gait patterns in people with gait disturbances, it is therefore important to select a velocity both patients and controls can adapt to. We selected 4.5 km/h because it is neither too fast for the patients nor too slow for the controls. Moreover, gender, age, weight and height may influence the dynamics of gait (Murray et al. 1970, Wu 1994, Bohannon 1997, DeVita and Hortobagyi 2000), which implies that a control group should be comparable to the intervention group with respect to these parameters. In general,

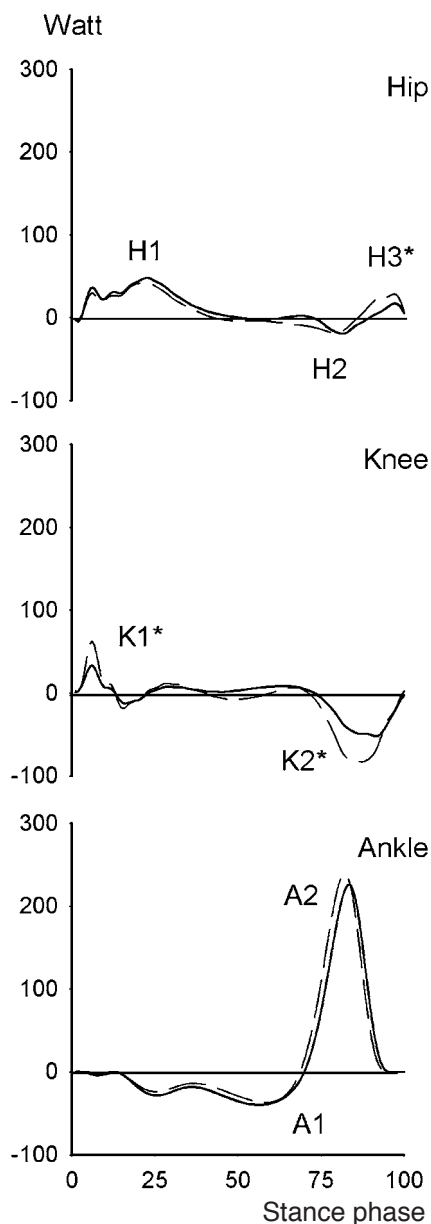


Figure 4. Average ankle, knee and hip power in the patients (solid line) and controls (dashed line). Zero on the x-axis indicates heel strike and 100 toe-off. Positive values indicate that the muscle generates energy and negative values that the muscle absorbs energy. The points A1, A2, K1 and K2, etc., represent the various peak values used in the statistical analyses. Asterisks indicate statistically significant differences between the two groups.

when evaluating the gait pattern in subjects with various hip diseases the intervention groups consist of elderly patients. The importance of these

different parameters makes a comparison between studies that evaluate gait patterns in other patient groups difficult.

In 3d inverse dynamics, the estimates of the joint centers are based on mathematical models that depend on marker placements, assumptions about body segments and anthropometric measurements. Each of these has some elements of uncertainties, which limit the 3d inverse dynamics approach. Moreover, individual anatomical variations affect the estimates. These limitations are inevitable, regardless of the gait analysis system used, but as they are systematic, they will affect both patients and controls. The pathological changes in the hip joint, however, do not affect the estimate of the joint center. The narrowing of the joint space which is found in Tönnis score I and II is included in the estimate of the hip joint center. Furthermore, the narrowing of the joint space represents only a few millimeters and is therefore not so important as the individual variations mentioned above.

So far as we know, only a single study has evaluated the gait pattern of patients with osteoarthritis secondary to hip dysplasia (Romano et al. 1996). Even though the subjects in that study were slightly older (mean age 48 years) and walked at a self-selected speed significantly slower than a control group, Romano et al. (1996) observed considerable differences in both kinematic and kinetic parameters between a group with congenital dysplasia of the hip and a control group, like that found in the present study. We noted that adult women with dysplasia of the hip appeared to walk with increased flexion in the ankle and knee joints and reduced hip flexor moment and power in the second half of the stance phase. Furthermore, Romano et al. (1996) observed that the total range of motion of the hip joint was reduced. This reduced hip extension during the entire stance phase. We found no statistically significant reduction in hip extension. But the increased flexion of the ankle and knee joints in the second half of the stance phase that we noted can not have occurred without a decrease in hip extension.

The pathological deformities of hip dysplasia may explain the differences in the second half of the stance phase. In general, the hip muscles cause a flexor moment in the second half of the stance phase, which controls extension of the hip.

In patients with dysplasia of the hip, the load-bearing surface in the hip joint is reduced because of the insufficient cover of the femoral head. This joint will therefore be painful when it is loaded. Therefore, the reduced hip flexor moment in the patient group can be interpreted as an attempt to unload the hip joint and thereby lessen the pain. Another consequence of this attempt to unload the hip joint is the hesitation observed in extending the hip joint with a resultant increase in dorsiflexion of the ankle joint and knee flexion in the second half of the stance phase. The patient group stayed more flexed than the control group during the entire stance phase, possibly to delay and reduce hip extension. The amount of power, generated or absorbed by the knee and hip joints was reduced in the second half of the stance phase. These findings can be viewed as resulting from the general reduction in the joint moments about these joints, but they can also be a result of various gait compensations. It seems likely that the patient group, due to the pathological changes in their hip joints, has less propulsion of their affected limb. Such changes may also reduce power.

In addition to the findings in the dynamic parameters, Romano et al. (1996) found that the kinematic and kinetic differences correlated with functional signs of impairment measured with the Harris Hip Score. In our study, we used correlation analysis between the dynamic differences, the CE-angle and the Tönnis score. The CE-angle is the most important indicator in diagnosing hip dysplasia. Although all women in the study had a poor covering of the femoral head, no correlation was found between the dynamic differences and the CE-angle. The Tönnis score is used to quantify the radiographic signs of degeneration of the hip. Of the 14 women, 10 had a measurable degeneration of the hip joint, but we observed no correlation between the dynamic parameters and the Tönnis score. Therefore, it seems that the CE-angle and the radiographic signs of degeneration of the hip joint can not predict the degree of gait compensations a patient with hip dysplasia will resort to. Most probably, this lack of applicability is due to other factors, which are more important for gait compensations. Since all women in our study experienced pain as a consequence of hip dysplasia, this might be a cause. With this method

one can not say whether pain or the pathologic changes caused the gait deviations in the patient group. However, since the clinical indicators of hip dysplasia did not correlate with the gait deviations and all patients felt pain, pain can be a likely cause.

Our study showed that important dynamic parameters measured during walking differ significantly in patients with painful hip dysplasia and healthy subjects. In our opinion, it is probable that surgery—e.g., periacetabular osteotomy—can partly normalize the gait pattern of the patients. However, this hypothesis remains to be tested in a future study.

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No competing interests declared.

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