No Detectable Major Changes in Gait Analysis After Soft Tissue Release in DDH

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Abstract The iliopsoas and adductor tendons are often soft tissue barriers obstructing relocation of the femoral head into the acetabulum and are frequently released to obtain reduction. We assessed whether posteromedial soft tissue release including sectioning of the adductor longus and iliopsoas tendons would lead to alterations in joint angles and moments of the hip joint or other major changes

in the gait pattern. We conducted 3-D quantitative gait analysis of 10 patients (mean age, 8.1 years) who had unilateral and surgically treated DDH before the age of 18 months. The mean single support time was shorter in the unaffected side of the patients than in the healthy control group. Mean pelvic excursions in both frontal and sagittal planes and maximum knee extension at stance of the affected and unaffected sides were higher in the patients than in the control group. Peak hip flexion moment during swing phase was somewhat reduced, and the hip moment crossover point from extension to flexion was slightly delayed in both the affected and unaffected sides. We could not identify an explanation for the slight deviations due to limited data. However, sectioning of the adductor longus and iliopsoas tendons in DDH patients under 18 months old did not appear to lead to major objective clinical gait alterations.

Level of Evidence: Level II, therapeutic study. See the Guidelines for Authors for a complete description of levels of evidence.

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Each author certifies that his or her institution has approved or waived approval for the human protocol for this investigation and that all investigations were conducted in conformity with ethical principles of research.

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Introduction

Developmental dysplasia of the hip (DDH) is associated with anatomic alterations of the acetabulum and to a lesser extent the proximal femur. Because of bony abnormalities, secondary adaptive soft tissue changes inevitably occur. The iliopsoas and adductor group tendons (especially the adductor longus) are the main extraarticular soft tissue barriers that obstruct relocation of the femoral head into the acetabulum [11, 21]. The inferomedial capsule itself, inverted labrum, hypertrophic pulvinar, elongated and hypertrophic ligamentum teres, and thick transverse acetabular ligament are the main intraarticular soft tissue

barriers [11, 21]. During the surgical treatment of DDH, all or some of these soft tissue obstacles are sectioned to obtain an anatomic reduction with or without an additional bony procedure [11, 21].

The adductor group muscles (adductor longus/brevis/magnus and gracilis) adduct the thigh and play an important role in the rhythmic lateral tilting of the pelvis in walking [8]. Among the adductor group muscles, the adductor longus muscle is the one most commonly released in DDH surgery. There seems no debate in complete sectioning of the adductor longus tendon when needed during DDH operations. The iliopsoas muscle formed by the psoas major and iliacus muscles is one of the principal hip flexors. Additionally, its tendinous portion helps stabilize the hip because it closely touches the hip capsule [8]. When it is shortened and tight as in DDH, it can interpose between the femoral head and acetabulum and prevent successful reduction [10]. However, in particular, it is suspected, the iliopsoas release from the lesser trochanter during the surgical treatment of DDH causes substantial hip flexor weakness. We have been sectioning the iliopsoas tendon from the lesser trochanter and the adductor longus tendon in almost all surgical procedures for DDH for many years because we believe both releases are important to obtain successful reduction.

We hypothesized posteromedial approach soft tissue release, including the sectioning of the adductor longus and iliopsoas tendons during the surgical treatment of DDH, would not lead to notable alterations in time and distance factors (velocity, step length, single support time), pelvic excursions, hip joint angles or hip joint moments in frontal and sagittal planes.

Materials and Methods

We compared the gait patterns of the unaffected and affected sides of patients with DDH to those of healthy subjects to ascertain whether the gait pattern of the unaffected contralateral side would also be affected at midterm followup in DDH. We determined the following criteria for inclusion of the quantitative gait analysis: (1) unilateral DDH for comparison of affected and unaffected sides; (2) no resubluxation, redislocation, or residual acetabular dysplasia to avoid the possible adverse effects of such complications on gait analysis; (3) no history of any type of osteonecrosis during the followup period to avoid the possible negative effects resulting from proximal femoral deformity and limb length shortening; (4) satisfactory radiographic outcome according to an objective classification system [15]; and (5) at least 5 years of age to obtain reasonable cooperation from the patient for making an effective and reliable quantitative gait analysis. We

examined the medical records of a group of 175 patients treated and prospectively followed from 1993 to 2001. Seventy-six patients met the inclusion criteria, and we invited 20 randomly selected patients from this group. The sample size was determined by using the anticipated difference in mean (0.19) and standard deviations (0.11) of walking velocity obtained from pilot data, as walking velocity was considered one of the most reliable parameters of gait in not only able-bodied subjects but in hemiparetic gait as well [22]. Power calculations indicated a sample of seven treated and control subjects would provide an 80% ($\beta = 0.20$) chance of detecting a 20% ($\alpha = 0.05$) difference between the groups. Among the invited patients, 10 agreed to quantitative gait analysis. All patients were female; the affected side was right in five patients and left in five. Mean age at the time of operation was 10.2 ± 4.8 months (range, 4–17 months) and during the gait analysis was 8.1 ± 2.4 years (range, 5.5–12.5 years). The minimum followup was 5 years (mean, 7.2 ± 2.2 years; range, 5–11 years). We obtained informed consent for participation in the study from the parents of all patients.

We (AB and YT) determined the clinical outcome according to the modified McKay's criteria [2] prior to the gait analysis. According to this categorical assessment an "excellent outcome" describes a painless hip with normal range of motion without limping or a positive Trendelenburg sign; a "good outcome" a painless hip with slight limping or insignificant decreased range of motion, but without a positive Trendelenburg sign; a "fair outcome" a hip with slight pain, a positive Trendelenburg sign and moderate stiffness; and a "poor outcome" a hip with considerable pain [2].

All patients were initially treated by the same surgical team (YT, AB, HA, HÖ) using the same soft tissue surgical procedure [4] before the age of 18 months. After adductor longus and iliopsoas tenotomies through a posteromedial approach [6], we performed intraoperative arthrography; in case of an arthrographically documented anatomic reduction, we performed no further surgery. If the femoral head was lateralized or outside the acetabulum on the arthrogram, then open reduction was made through the same incision at the same session.

After surgery, all patients were placed in a bilateral hip spica cast in the human position [11] excluding the ankles and feet for 3 months. After cast removal, both hips were put in a full-time rigid abduction brace in flexion and abduction for an additional 3 months [4]. A specific rehabilitation program was not instituted with the patients and all of them were allowed to walk freely following the immobilization procedure.

Quantitative gait data were collected using the Vicon 370 system (Vicon; Oxford Metrics Limited, Oxford, UK).

The VICON Clinical Manager (VCM) (version 3.2) software was used to calculate joint rotation angles between anatomically aligned reference frames associated with adjacent body segments. Anthropometric data including height, weight, leg length and joint width of the knee and ankle were collected. The standard clinical gait analysis (VCM) protocol uses the so-called Davis marker set, which consists of 15 passively reflective markers placed on specific anatomical landmarks: sacrum, bilateral anterior superior iliac spine, middle thigh, lateral knee (directly lateral to axis of rotation), middle shank (the middle point between the knee marker and the lateral malleoli), lateral malleoli, heel and forefoot between the second and third metatarsal head [5]. After the patients were instrumented with retroreflective markers, we instructed them to walk barefoot at a self-selected pace over a 10-meter-long walkway during which time data collection was completed. Five cameras recorded the quantitative spatial location of each marker as the subject walked. The trial in which all the markers were automatically and clearly identified by the system was determined the best data.

Two Bertec force plates (Bertec Corp, Columbus, OH, USA) were used to measure the three components of the ground reaction force at a sampling rate of 1000 Hz. Moments and powers of hip, knee and ankle joints were calculated in three dimensions using the Newton-Euler recursive inverse dynamics method [16].

Time-distance and selected kinematic (joint rotation angles of pelvis, hip, knee and ankle in frontal, sagittal and transverse planes) and kinetic (moment and powers of hip, knee and ankle in frontal and sagittal planes) parameters of the affected and unaffected sides were compared with the reference values of the gait laboratory obtained from 20 healthy children (40 sides) (Tables 1–3). There were 13 girls and seven boys with a mean age of 10.1 ± 2.7 years (range, 6–13 years) in the control group.

The t-test for independent samples was used for the comparison of the means of several time-distance parameters (velocity, single support time, step length), joint rotation angles of pelvis, hip, knee and ankle in frontal, sagittal, and transverse planes and kinetic parameters (moment and powers of hip, knee and ankle in frontal and

sagittal planes) between groups (SPSS 9.0 for Windows; SPSS Inc., Chicago, IL, USA).

Results

The clinical outcome of all hips at the latest followup was considered “excellent” (modified McKay criteria).

Mean velocity and step-length values of the affected and unaffected sides were similar to those of the control group ($p > 0.05$) (Table 1). Mean single support time of the “unaffected side group” was shorter ($p = 0.012$) than that of the “control group” (Table 1).

Mean pelvic excursions of the affected and unaffected sides in both frontal and sagittal planes were higher ($p = 0.018$ and $p = 0.021$, in frontal plane and $p = 0.011$ and $p = 0.010$ in sagittal plane) than those of the control group. Furthermore, maximum knee extension at stance was decreased ($p = 0.014$ and $p = 0.011$) in both the affected and unaffected sides when compared with the control group’s data (Table 2). However, we observed no differences in the mean hip flexion and adduction angles of the affected and unaffected sides when compared with the control group ($p > 0.05$) (Table 2).

Peak hip flexion moment during swing phase was diminished ($p = 0.053$ and $p = 0.041$) and the hip moment crossover point from extension to flexion was delayed ($p = 0.060$ and $p = 0.048$) in both the affected and unaffected sides (Table 3). We observed no alteration in the hip abduction moment in either the affected or unaffected side with respect to the control group ($p > 0.05$) (Table 3).

Discussion

Sectioning of the iliopsoas and adductor longus tendons during the surgical treatment of DDH is one of the key points for obtaining an anatomic surgical reduction during childhood. However, there is still debate over whether these releases have further adverse effects on gait pattern. We believe such releases do not have unfavorable effects, especially on the hip rotation angles and hip moment and

Table 1. Time-distance parameters

Time-distance parameters	Affected side in DDH group (n = 10)	Unaffected side in DDH group (n = 10)	Control group (n = 40)	p Value (affected versus control)	p Value (unaffected versus control)
Velocity (m/s)	0.99 ± 0.22	0.92 ± 0.21	1.06 ± 0.23	0.394	0.104
Single support time (seconds)	0.37 ± 0.06	0.35 ± 0.06	0.39 ± 0.05	0.219	0.012*
Step length (meters)	0.49 ± 0.12	0.48 ± 0.10	0.53 ± 0.09	0.199	0.127

Values are mean $\pm$ standard deviation; DDH = developmental dysplasia of the hip.

*Significant difference.

Table 2. Quantitative kinematic analysis

Kinematic analysis	Affected side in DDH group (n = 10)	Unaffected side in DDH group (n = 10)	Control group (n = 40)	p Value (affected versus control)	p Value (unaffected versus control)
Pelvic excursion, frontal (°)	6.67 ± 1.77	6.70 ± 2.18	5.03 ± 1.92	0.018*	0.021*
Pelvic excursion, sagittal (°)	4.18 ± 1.98	4.23 ± 2.12	2.76 ± 1.39	0.011*	0.010*
Pelvic excursion, transverse (°)	11.06 ± 12.23	8.15 ± 6.45	11.22 ± 5.13	0.950	0.115
Hip flexion angle at initial contact (°)	26.71 ± 5.89	26.68 ± 7.66	25.64 ± 6.62	0.643	0.669
Hip flexion at terminal swing (°)	27.52 ± 6.45	25.48 ± 5.89	26.88 ± 5.57	0.757	0.485
Hip extension at terminal stance (°)	-14.38 ± 7.06	-11.46 ± 6.58	-14.28 ± 7.51	0.968	0.283
Maximum hip adduction at stance (°)	7.69 ± 4.80	5.91 ± 3.83	5.68 ± 4.49	0.218	0.886
Maximal hip rotation at stance (°)	-1.17 ± 12.42	4.69 ± 22.96	-4.28 ± 16.32	0.577	0.159
Maximum knee extension at midstance (°)	11.15 ± 6.19	11.86 ± 8.49	6.25 ± 5.27	0.014*	0.011*
Maximum knee flexion at swing (°)	60.25 ± 6.32	57.40 ± 13.53	59.79 ± 7.13	0.851	0.441
Maximum ankle dorsiflexion at stance (°)	19.93 ± 3.26	15.89 ± 3.79	16.91 ± 5.39	0.098	0.575
Maximum ankle plantar flexion (°)	-3.00 ± 12.03	-2.57 ± 6.88	-5.13 ± 8.17	0.506	0.366
Foot progression at stance (°)	-6.90 ± 8.84	-6.10 ± 6.38	-5.08 ± 5.12	0.396	0.596

Values are mean ± standard deviation; DDH = developmental dysplasia of the hip.

*significant difference.

Table 3. Quantitative kinetic analysis

Kinetic analysis	Affected side in DDH group (n = 10)	Unaffected side in DDH group (n = 10)	Control group (n = 40)	p Value (affected versus control)	p Value (unaffected versus control)
Peak hip abduction moment (Nm/kg)	0.64 ± 0.22	0.58 ± 0.12	0.57 ± 0.13	0.221	0.795
Peak hip flexion moment during swing (Nm/kg)	0.67 ± 0.31	0.66 ± 0.24	0.94 ± 0.40	0.053	0.041*
Peak hip extensor moment at stance (Nm/kg)	0.24 ± 0.11	0.27 ± 0.24	0.23 ± 0.40	0.934	0.728
Peak knee extensor moment at stance (Nm/kg)	0.25 ± 0.27	0.32 ± 0.26	0.34 ± 0.13	0.326	0.806
Peak ankle plantar flexion moment (Nm/kg)	1.06 ± 0.16	1.04 ± 0.17	1.01 ± 0.17	0.386	0.612
First peak vertical ground force (N)	98.44 ± 9.70	102.22 ± 8.71	93.66 ± 16.43	0.384	0.120
Delay in the hip moment crossover point from extension to flexion (percent of gait cycle)	25.78 ± 12.02	26.44 ± 13.95	19.35 ± 8.72	0.060	0.048*

Values are mean ± standard deviation; DDH = developmental dysplasia of the hip.

*Significant difference.

power in frontal and sagittal planes. We therefore hypothesized soft tissue releases including the sectioning of the adductor longus and iliopsoas tendons during the surgical treatment of DDH would not lead to notable alterations in time and distance parameters of gait, pelvic excursions, hip joint angles or hip joint moments in frontal and sagittal planes.

The main limitation of the present study is the small sample size resulting from inclusion criteria and the parents' reluctance to allow further assessment of their children. Our study population included only girls, but we believe this would have no influence on the conclusions. Ideally one would have a longitudinal study comparing the same side of the same patient before and some substantial time after surgery, but this is not possible, because the

patients had surgery before or at the walking age. We evaluated a single cycle we considered representative rather than the mean of at least three to five cycles; averaged trials are generally used since a single trial is not necessarily representative. In this study all patients walked three to six times with at least five steps for each trial. The natural variation of the subjects was tested by consistency plots before deciding the best representing trial. We did not average the trials as representing trials were considered highly consistent.

Numerous studies report quantitative gait analysis of untreated or treated patients with DDH [17–19]. Many gait alterations correlating with the Harris hip score [9] were observed in a study of 21 adult patients with untreated symptomatic residual DDH. Pain and altered

proprioceptive input were considered the most important factors causing such gait deviations in these patients [19]. In another study performed in 14 adult women with hip dysplasia before periacetabular osteotomy, the insufficient cover of the femoral head and pain might explain both the kinematic and kinetic gait alterations seen in the ankle, knee, and hip joints at the second half of the stance phase [18]. The same study group observed considerable improvement in only the knee kinematics after the acetabular osteotomy in the same patients with hip dysplasia [17]. On the contrary, the patients in our study have been operated on under the age of 18 months before severe alterations in their gait patterns have occurred. Our kinematic and kinetic data suggest patients with DDH successfully treated in early childhood have patterns similar to those of healthy children with some slight deviations.

Release of the iliopsoas muscle at the pelvic brim is believed to lengthen only the psoas muscle, whereas release from the lesser trochanter of both psoas and iliacus muscles may weaken the hip flexor [1, 20]. Unwillingness of some surgeons to release the iliopsoas tendon from the lesser trochanter during DDH surgery seems to depend on the previously mentioned opinions and findings of some previous clinical studies carried out on patients with DDH and cerebral palsy. Release of the iliopsoas tendon from the lesser trochanter during DDH surgery was reported to have some considerable adverse effects on iliopsoas muscle mass and hip kinematics and kinetics in the sagittal plane [1, 12]. In addition, several studies recommend releasing the iliopsoas muscle at the pelvic brim, not from the lesser trochanter in patients with cerebral palsy [13, 14, 20]. One recent report suggested iliopsoas release from the lesser trochanter did not lead to a major change in hip flexor power generation in 25 patients with cerebral palsy at 1 year postoperatively [3]. Our data confirm those findings.

We believe the difference (0.04-second) between the mean values of single support time on the unaffected side and in the control group is irrelevant in clinical practice. Our findings suggest hip flexor moment decreases to some extent in both the affected and unaffected sides. We have also observed slight alterations (although still within the limits of the mean $\pm$ standard deviation range of the healthy population) in the kinematics of the pelvis in the frontal and sagittal planes, the kinematics of the knees in the sagittal plane, and the kinetics of the hips in the sagittal plane in not only the affected sides, but also in the unaffected sides. The alterations have been almost symmetric. If sectioning of the iliopsoas and adductor longus tendons or release of other soft tissues has had an effect on the gait pattern, these deviations should have been observed only in the affected side, but not in the contralateral unaffected side. There may be three explanations for the symmetric

alterations. First, these children may try to maintain symmetry by taking on a compensatory pattern on both sides since they have very good gastrosoleus muscles which can most likely be used in compensation for the weak hip flexors. However, it is very unlikely symmetric and primary pathology and compensatory movement would be observable in the same person at the same time. Also, we observed no increased ankle plantar flexion moment reflecting increased gastrosoleus muscle firing to compensate weak hip flexors. Apparently our patients did not require any compensation for weak hip flexor muscles. Secondly, these slight alterations may be due to postoperative prolonged immobilization in a cast followed by a brace for a total of 6 months. The locomotor control system finds a new motor pattern when the movements of the patient are restricted (eg, casting), and this is internalized and memorized somewhere in the motor control system [7]. This new retained pattern of movement continues for some time even after the restriction ends (cast removal) [7]. Within a certain time, there exists a gradual return to the old motor pattern [7]. However, it is difficult to ascertain if older children are still influenced by immobilization that occurred many years ago. Also, we do not have enough data to exactly analyze the long-term adverse effects of immobilization. On the other hand, the effects of these slight alterations on sports activities (especially heavy or contact sports) and the long-term outcome during adulthood remain still uncertain. Third, the alterations in the gait pattern may be due to persistent weakness or just due to pattern change from DDH, not related to the specific muscle release.

Based on our data derived from a small number of patients, we conclude posteromedial soft tissue surgery with release of the iliopsoas and adductor longus tendons, and in the absence of postoperative complications, does not cause major alterations in the gait analysis at midterm followup in DDH. However, these patients have some mild gait deviations of unknown cause which do not seem to adversely affect the clinical outcome.

Acknowledgments We thank Professor Süreyya Ergin, MD, from the Department of Physical Medicine and Rehabilitation, Ankara University Faculty of Medicine, Ankara, Turkey, for analyzing the data.

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