Functional Analysis of Children with Hip Joint Developmental Dysplasia

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ABSTRACT

Purpose: This study was designed to compare the functional abilities of children with unilateral developmental dysplasia of the hip joint (DDH) with normal children. Also, to compare between the treated and untreated DDH children. Methods: Thirty eight ambulant children with unilateral DDH were studied with respect to Gait analysis, oxygen consumption, muscle strength, and hip abduction range of motion as indicative for the child functional abilities. According to the treatment delivered to DDH children, they were classified into treated and untreated groups. Results: Comparing between DDH and normal children, results showed significant decrease in step length, cadence, speed of walking, isometric hip abductors muscle strength, and active hip abduction range as well as significant increase in energy cost of walking and oxygen uptake in DDH children. When DDH groups were compared, the treated cases were better than the untreated cases with respect to step length, foot angle, muscle strength, and passive hip abduction range, while no significant difference was found with respect to step width, cadence, speed of walking, VO₂ uptake, VO₂ cost, and active hip abduction range. Conclusion: Children with hip dysplasia were impaired during walking, not only because of the accompanying gait deviations and pain, but also because the energy required for walking was increased. Furthermore, they demonstrated limitation of hip abduction range and muscle strength on the affected side. The functional abilities of the treated cases of DDH were better than the untreated cases with regard to step length, foot angle, muscle strength, and passive hip abduction range. Key words: DDH; gait; oxygen consumption; muscle strength; ROM.

INTRODUCTION

The term developmental dysplasia of the hip DDH has replaced congenital dislocation of the hip because it more accurately reflects the full spectrum of abnormalities that affect the immature hip. DDH is a developmental (ongoing) process, variable in manifestation and not always detectable at birth. It involves a dysplasia, or abnormal formation of the hip joint occurring between fetal life and maturity as a result of instability. Hips that are found to be normal at birth (and even in the first few months of life) can subsequently be found to be abnormal later. The majority of children with DDH have ligamentous laxity-looseness of the fibrous bands connecting bones together in joints. This predisposes to hip instability; instability allows the hip to slip out of position; and certain mechanical factors such as breech presentation can aggravate the problem. Maternal hormones associated with pelvic relaxation around the time of birth also aggravate the instability of the newborn hip joint by allowing softening and stretching of the baby's hip ligaments.

DDH can predispose a child to premature degenerative changes, painful arthritis, abnormal gait, unequal leg length, and decreased agility. Careful physical examination is recommended as a screening
tool; early diagnosis helps improve treatment results and decrease the risk of complications\(^1,5\). Once a child is walking, gait abnormalities related to leg length discrepancy, asymmetric intoeing or outtoeing (one foot points in or out significantly more than the other) are the hallmark for identifying an abnormal hip. If the hip abnormality still remains undiagnosed, pain or more noticeable gait asymmetry ultimately draws attention to the hip as the child becomes older\(^4,6,7\). In older or walking children, complaints of limping, waddling, increased lumbar lordosis, toe walking, and leg-length discrepancy may indicate an unrecognized DDH\(^6,7,8\).

If an unstable hip is recognized at birth, treatment consists of maintaining the position of the hip in flexion (knee up towards the head) and abduction (knee away from the centerline) for about 1-2 months. The Pavlik harness is the most widely used device. It maintains proper position of the femoral head and allows for "tightening up" of the ligamentous structures as well as for stimulation of normal formation of the hip socket. The treatment must be continued until the hip is stable and x-rays or ultrasound examinations are normal\(^9,10,11\). From 1-6 months, true dislocations may develop. As a consequence, treatment is directed toward reduction of the femoral head into the socket (acetabulum), usually with the Pavlik harness or similar device. The harness pushes the femoral head toward the socket, and usually, relocation of the femoral head will occur within 3-4 weeks. The Pavlik harness is approximately 95% successful in dysplastic or subluxated hips and 80% successful in true dislocations. If a spontaneous reduction does not occur by splinting, then a surgical closed reduction (manipulation under anesthesia) is done\(^12,13\).

In the older infant from 6-18 months, surgical closed reduction (manipulation under general anesthesia) is the major method of treatment\(^12,13\). After 18 months of age, the progressive deformities become so severe that major open surgical intervention is necessary to realign the hip. As the child gets older more secondary deformities develop which can be grouped under 3 major pathological entities, acetabular dysplasias, subluxation of the femoral head with femoral neck-shaft antvertion deformities and secondary soft tissue contractures\(^14,15\). Many treatment options have been proposed for developmental dysplasias of the hip in older children. Among these are (1) Closed reduction\(^12,13\), (2) Closed reduction combined with pre-operative traction\(^16\) or adductor tenotomy\(^15\). (3) Open reduction\(^17\), (4) Open reduction combined with femoral\(^18\) and/or pelvic osteotomy\(^19\). (5) Open reduction combined with Salter's osteotomy\(^20\). All these protocols have advantages and disadvantages.

The purposes of this study, therefore, were 1) To compare the functional abilities of children with unilateral developmental dysplasia of the hip joint (DDH) with a group of normal healthy children. 2) To compare the functional abilities of the treated and untreated DDH children. Gait analysis (foot print), maximal oxygen consumption, muscle strength, and hip abduction range of motion were considered indicatives for the child functional abilities.

**MATERIALS AND METHODS**

**Subjects**

We tested thirty eight ambulant children with unilateral DDH. They were operated one year ago (from June 2003 to June 2004) or just diagnosed at the Paediatric Orthopaedics and Traumatology Department, Jordan University.
Hospital, Amman, Jordan. Twenty seven were girls and the other 11 were boys. The age limit selected was ranged from five to eight years. The mean age, height, and weight were calculated. All of them were able to walk independently without any of the assistive devices. Thirty two of them were complaining of mild to moderate pain during walking. According to the treatment delivered to those children, they were classified into two groups. Treated group included children who underwent an open reduction combined with femoral and/or pelvic osteotomies or an open reduction combined with Salter's osteotomy. Untreated group included children who were diagnosed late and received no treatment till the study date and they are scheduled to have the same types of interventions as in the treated group.

Fifteen matched healthy children with similar age, gender, and body size (height and weight) were chosen and were considered as the control group. They had no known muscular or skeletal abnormalities of the locomotor system. A written consent was obtained from parents of all children participated in the study.

Evaluation
Assessment of the functional abilities was done through:

Gait analysis
It was done through evaluating five major gait parameters. These parameters were (step length, step width, foot angle, cadence, and speed of walking). Footprint, as a tool for gait evaluation, was used as follows:

Chalk paper, as a straight walkway and colored powder were used. The walkway is about 10 meters long and about 50-cm width. The middle 6 meters of these walkways is red to be the area of measurement, as the first 2 meters and the last 2 meters were neglected during measurements. This walkway sheet was located in the gait evaluation area and was fastened from both ends to prevent slipping off the ground. Stop watch, tape measurement, and protractor were used to measure the outcome of gait parameter tests.

Oxygen consumption
Fox et al., protocol was conducted to measure oxygen consumption. The procedure required the child to walk unassisted on a level treadmill. Treadmill speed was carefully checked both before and during each walking trial. The subject was asked to mouthly breath while applying a nose clip, so the expired air passed through the gas meter and the amount of oxygen consumed will be computed and printed out. The resting VO₂/Kg/min was determined while the subject standing on the immobile treadmill. The cycling reeve of the treadmill was adjusted at the least speed. No oxygen consumption measurements were taken at this time, since the steady state period has not been reached yet. Once the steady state is reached (usually within 3 to 4 minutes after walking is started) oxygen consumption was measured over 2 minutes. The difference between the VO₂/Kg/min value at the steady state and the VO₂/Kg/min at the resting state was considered as the amount of oxygen consumption per kilogram per minute (oxygen uptake--VO₂/Kg/min).The amount of oxygen consumed per kilogram per meter was calculated and was defined as (oxygen cost--VO₂/Kg/meter).

Muscle strength
This was done through evaluating the isometric muscle force (in Newton) of hip abductors. We measured muscle strength using a hand-held electronic dynamometer (HHD). Three attempts were made for the hip abductors with the make test technique, where resistance is gradually built up for about 5
seconds. Fifteen minutes for rest was given between trials to allow muscular recovery. Children were tested in supine lying position with extended hips and knees. Stabilization was applied manually on the contralateral pelvis by the examiner as well as by asking the child to hold on to the bench. The HHD was placed distally on the femur at a place that was comfortable for the child. Encouragement to maximum effort was given in a standardized way.

**Hip abduction range of motion**

While the child was in supine lying position, active and passive hip abduction ranges were measured using a protractor goniometer with the knee in maximum extension. Fulcrum was placed in line with the anterior superior iliac spine. The moving arm of the goniometer was aligned with the midline of the patella, the stationary arm with the anterior superior iliac spine of the opposite side. The child was asked to move his leg out to the side as far as he can.

The same evaluation protocol of functional abilities was conducted for every child in DDH groups as well as the control group. Recording data was based on an average of three measurements, conducted by three physiotherapists who were well trained in this evaluation protocol rather than the researchers.

**Statistical Analysis**

The collected data was statistically treated to show the mean, range, standard deviation, and standard error of mean for all sets of measurements, in all groups (DDH and normal children). Least-significant difference (LSD) one way analysis of variance (ANOVA) was used to analyze all data and to show differences in all parameters between all groups. P-value <0.05 was considered significant.

**RESULTS**

Table 1 presents a summary of demographic data and clinical characteristics of all children. The groups were well matched for age, gender, height, weight, and sides to be evaluated and no significant differences were detected in respect to these data.

**Gait parameters**

When the treated and untreated DDH groups compared to the normal children, results showed statistically significant decrease in step length, cadence, and speed of walking in both DDH groups (P = 0.000). No significant difference was detected between DDH groups (treated and untreated) and the control group in respect to step width (P = 0.11, P = 0.79 respectively). Measurements of foot angle showed significant increase in the untreated DDH group (P = 0.000) and no difference in the treated DDH group (P =0.44) when compared to normal children (Table 1). On comparing between the treated and untreated DDH groups, there was a statistically significant increase of step length and decrease in foot angle in the treated DDH group (P = 0.001, P = 0.000 respectively). No significant difference was detected between both groups in respect to step width, cadence, and speed of walking (P = 0.07, P = 0.013, P = 0.14 respectively) (Table 1, Figure 1).
Table (1): Clinical data of DDH and normal children.

<table>
<thead>
<tr>
<th>Variables</th>
<th>DDH Children</th>
<th>Normal Children</th>
<th>P Treated</th>
<th>P Untreated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (n)</td>
<td>25</td>
<td>13</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>7/18</td>
<td>4/9</td>
<td>-</td>
<td>4/11</td>
</tr>
<tr>
<td>DDH side (Right/Left)</td>
<td>6/19</td>
<td>3/10</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Evaluated side (Right/Left)</td>
<td>6/19</td>
<td>3/10</td>
<td>-</td>
<td>4/11</td>
</tr>
<tr>
<td>Age Months mean (SD)</td>
<td>74±10</td>
<td>72.6±11.6</td>
<td>0.91</td>
<td>73.3±9.3</td>
</tr>
<tr>
<td>Weight Kg mean (SD)</td>
<td>21.7±4.3</td>
<td>22.3±5</td>
<td>0.79</td>
<td>20.1±4.8</td>
</tr>
<tr>
<td>Height Cm mean (SD)</td>
<td>115±12</td>
<td>112±10</td>
<td>0.82</td>
<td>114±9</td>
</tr>
<tr>
<td>Step length Cm mean (SD)</td>
<td>41.1±4.6</td>
<td>35.8±2.4</td>
<td>0.001*</td>
<td>47.2±4.8</td>
</tr>
<tr>
<td>Foot angle Mean (SD)</td>
<td>8.8±3.1</td>
<td>17.8±2</td>
<td>0.000*</td>
<td>8±3</td>
</tr>
<tr>
<td>Step width Cm mean (SD)</td>
<td>7.9±2</td>
<td>9±1.2</td>
<td>0.07</td>
<td>8.8±1.6</td>
</tr>
<tr>
<td>Cadence Steps/min mean (SD)</td>
<td>89±6.3</td>
<td>84.3±6.2</td>
<td>0.13</td>
<td>139.7±13.4</td>
</tr>
<tr>
<td>Speed (meter/minute) mean (SD)</td>
<td>56.1±3.3</td>
<td>53.8±3.4</td>
<td>0.14</td>
<td>66.8±6.7</td>
</tr>
<tr>
<td>VO₂ uptake (ml/Kg/min) mean (SD)</td>
<td>11.7±1.4</td>
<td>12.3±1.2</td>
<td>0.89</td>
<td>9.7±1.9</td>
</tr>
<tr>
<td>VO₂ cost (ml/Kg/meter) mean (SD)</td>
<td>0.32±0.03</td>
<td>0.34±0.02</td>
<td>0.98</td>
<td>0.27±0.04</td>
</tr>
<tr>
<td>Muscle strength ²N mean (SD)</td>
<td>145.5±8.9</td>
<td>137.9±5.1</td>
<td>0.004*</td>
<td>184.2±5.4</td>
</tr>
<tr>
<td>Passive range mean (SD)</td>
<td>44.6±0.8</td>
<td>40.2±4</td>
<td>0.000*</td>
<td>45±1.5</td>
</tr>
<tr>
<td>Active range mean (SD)</td>
<td>37.3±4.7</td>
<td>36.5±4.6</td>
<td>0.53</td>
<td>45±1.5</td>
</tr>
</tbody>
</table>

(P) Alpha level of significance when comparing between the treated and untreated DDH children.
(P Treated) Alpha level of significance when comparing between the treated DDH and normal children.
(P Untreated) Alpha level of significance when comparing between the untreated DDH and normal children.
(² N) Newton.
(*) Values significant at P<0.05.

Oxygen consumption
Children with untreated DDH displayed higher energy cost of walking and oxygen uptake (mean 0.34±0.02 ml/kg/meter, 12.3±1.2 ml/kg/minute respectively) than treated DDH children (mean 0.32±0.03 ml/kg/meter, 11.7±1.4 ml/kg/minute respectively) or normal children (mean

Fig. (1): Comparison between measurements of Cadence and speed of walking in all children.

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0.27±0.04 ml/kg/meter, 9.7±1.9 ml/kg/minute respectively). Least significance post-hoc analyses of variance revealed significant differences between each of the DDH groups and the group of normal children in respect to the energy cost of walking and oxygen uptake (P = 0.000), while no significant difference was detected between the two DDH groups (P = 0.98, P = 0.89 respectively) (Table 1, Figure 2).

**Muscle strength**

Our results revealed marked decrease of isometric hip abductors muscle strength in the untreated DDH children (mean 137.9±5.1 Newton) compared to the treated DDH children (mean 145.5±8.9 Newton) or compared to the normal children (mean 184.2±5.4 Newton). Analyses of variance revealed that the difference exist between each of the DDH groups and the group of normal children was statistically significant (P = 0.000). Similarly, the difference exist between the two DDH groups was found also to be statistically significant (P = 0.004) (Table 1, Figure 3).

**Hip abduction range**

Comparing between DDH groups and control group, passive hip abduction range was significantly lower in the untreated DDH group with mean degrees of 40.2±4 compared to mean degrees of 45±1.5 in normal children.
Passive hip abduction range was significantly higher in treated DDH cases than in untreated cases with mean degrees of 44.6±0.8 and 40.2±4 respectively (P = 0.000). On the other hand, the active hip abduction range was significantly low in treated and untreated DDH groups with mean degrees of 37.3±4.7 and 36.5±4.6 respectively when compared to mean degrees of 45.0±1.5 in normal children (P = 0.000). While no significant difference in active hip abduction range was detected between the treated and untreated DDH groups (P = 0.53) (Table 1, Figure 4).

![Bar Chart](image)

**Fig. (4): Comparison between measurements of passive and active hip abduction range in all children.**

**DISCUSSION**

Several studies approved that gender, age, weight and height may influence the kinematics of gait, oxygen consumption, and muscle strength and this necessitated that the control group to be comparable to the DDH groups with respect to these parameters. Furthermore, our data analysis did not show any significant difference between these groups in respect to these data.

Gait parameters Our study showed that important kinematics parameters (step length, foot angle, cadence, and speed of walking) measured during walking differ significantly in patients with DDH and normal children. It has been speculated that pain and pathologic changes caused the gait deviations in the DDH children. In agreement with our results, Cavagna et al., and Tesio et al., stated that painful hip conditions like hip dysplasia or arthritis may produce asymmetry in stride (two successive steps), especially in unilateral hip pathologies. Also, they concluded that pain may increase muscle tone and compel the patient to shorten the length of his step.

Similar to our results of increased foot angle, in the untreated DDH children only, Matovinovic et al., attributed the asymmetric intoeing or outtoeing observed in his study to the medial and lateral rotation in the extended hip as a result of abnormal femoral anteversion. Our results revealed decreased cadence in both DDH groups and this was in contradiction with Cavagna et al., and Tesio et al., findings which states that patient compensates for short step by increasing the stepping frequency at a given speed. Additionally, only the step length and foot angle was significantly improved in the treated DDH group compared to the untreated group. It is probable that femoral and/or pelvic surgeries e.g. periacetabular osteotomy and Salter’s osteotomy performed to our treated

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DDH group can partly normalize the gait pattern of those children. However, this hypothesis remains to be adequately tested in a future studies.

Oxygen consumption Bowen et al., 29 reported oxygen cost to be a more reliable oxygen-use measurement of energy expenditure than VO$_2$ alone. The oxygen cost measure takes into account the oxygen requirement on a per-meter basis and provides a means for comparing individuals or the same individual over time despite differences in walking velocity. In accordance with other studies, our DDH children has been shown to consume much more oxygen than normal children 30,31. A study conducted by Veicsteinas et al., 31 recorded an increase of up to 20% in the energy cost of locomotion in patients affected by minor foot lesions with an apparently normal gait. Based on clinical observation of our DDH children, we can attribute this increase in energy cost to pain experienced during walking as well as to gait deviations but not to limitation of hip joint range of motion or to joint pathogenesis evaluated radiographically 30. The mechanism by which pain and gait deviations contribute to an increased energy cost of locomotion could be explained as follow:

Walking is characterized by a continual transformation of potential energy (a rise in the center of gravity) into kinetic energy. This transformation yields a substantial saving of energy with each step. When, for various reasons, this transformation is impaired, an increase in muscle activity is necessary; hence, a greater quantity of oxygen is consumed 30,31. Asymmetry in stride length results is an unbalanced transformation from potential to kinetic energy and consequently an increase in the external mechanical work performed by the unaffected limb 26,27. As a consequence of pain and gait deviations (short step; increased foot angle; decreased cadence and speed) the DDH child attempts to correct these deviations actively by increasing the amount of activity per minute during walking resulting in increased energy expenditure and oxygen consumption 26,27,31.

Muscle strength and hip abduction ROM Comparing the ROM data in DDH and normal children, our untreated DDH children did demonstrate limitation in active and passive hip abduction range while the treated children demonstrated limitation in active range only. This was in agreement with Jari et al., 32 who confirmed that unilateral limitation of hip abduction is a valuable clinical sign for DDH. Jari et al., reported that dislocated hips seen after the age of six months, presented with unilateral limitation of hip abduction. His results revealed that all major (Graf type III) and 44.5% of minor (Graf type II) dysplastic hips presented with this sign. Information about strength issue or muscle weakness in DDH children is still limited however, our results revealed significant decrease of isometric hip abductors muscle strength in DDH children compared to normal children. The reduction of muscle strength observed in our DDH groups could be attributed to the mechanical disadvantage of the gluteus medius muscle. This mechanical disadvantage is caused by reducing the distance between the muscle origin and insertion in the dislocated hip.

It is important to mention that, in our study no significant difference was found between the treated and untreated DDH groups with respect to step width, cadence, speed, VO$_2$ uptake, VO$_2$ cost, and active hip abduction range. This may support the claim of Ryan et al., 33 who reported that the natural history of untreated dislocation is more favorable than history of treated dislocation by open reduction with or without osteotomy.
In conclusion children with hip dysplasia were impaired during walking, not only because of the accompanying gait deviations and pain, but also because the energy required for walking was increased. Our findings also demonstrated limitation of hip abduction range and muscle strength on the affected side in DDH children. Treated cases of the DDH were better than the untreated cases in respect to step length, foot angle, muscle strength, and passive hip abduction range.

REFERENCES