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Patterns of hip migration in non-ambulant children with cerebral palsy: a prospective cohort study

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Abstract

Background. In children with cerebral palsy (CP), we have little information on when hip migration (HM) starts, what causes hip displacement, how HM changes over time, and how to halt this migration to avoid surgery.

Objectives. We aimed to estimate the prevalence of HM percentage (HMP) >40% in a homogeneous population of non-ambulant children with CP and model the changes in HMP over a 2.6-year mean follow-up.

Methods. From September 2009 to September 2015, this observational, prospective, multicenter cohort study recruited 235 children from 51 centers who were 3 to 10 years old and had levels IV and V of the Gross Motor Function Classification System for CP. The outcomes were yearly HMP measurements by the Reimers index. Only children with at least one hip with HMP \leq 40% at baseline were included in trajectory modeling. Comparisons of children's characteristics between trajectory groups were adjusted by the false discovery rate method.

Results. The prevalence of children with at least one hip with HMP >40% was estimated at 24.3% (95% confidence interval 18.6-30.0). Pelvic obliquity was observed in 51.4% and

24.4% of children with asymmetric and symmetric HMP ($p=0.002$). The trajectory modeling identified 3 types of MP changes over time. Many children (67.4% and 79.3% for the right and left hip) could be assigned to the “stable” trajectory group.

Conclusions. In non-ambulant children with CP, the prevalence of HM requiring surgery is low and most hips remain practically stable over time.

Keywords: cerebral palsy; children; hip migration; trajectory modeling; surgery

Introduction

Cerebral palsy (CP) is a group of disorders that occur in 2/1000 live births (1). This group includes permanent disorders of motor function (movement, muscle control, posture, and/or balance) due to a non-progressive injury or malformation that affects the immature brain before, during, or immediately after birth. Among the children who do not acquire the ability to walk (levels IV and V of the Gross Motor Function Classification System [GMFCS]) (2, 3), one third have a high risk of orthopedic contractures, especially hip dislocation (4). In fact, before complete dislocation, hip migration (HM) is progressive, may be uni- or bilateral, and is accompanied by pelvic obliquity, scoliosis, windswept attitude, difficulties with sitting, and obviously, pain. These events may be causes or consequences of HM (5-7).

The literature has reported various estimations of the prevalence of HM (0% to 66%). This very large range is mostly due to small sample sizes, wide age ranges, different disease severities, and different prevention programs. Another issue is the absence of a consensual definition of hip luxation (8, 9).

The number of systematic reviews on interventions for children with CP has exponentially increased in recent years (3 in 1993-1997 to 41 in 2008-2018). This knowledge improvement has allowed for establishing states of evidence for interventions for children with CP (10). However, with the quick improvements or successive changes in clinical

practices over time, determining the measures that would stop or slow HM in the long term is difficult. To our knowledge, no study has reported on changes in hip placement over a long period in a homogeneous cohort because most observations stop when surgery is considered with HM percentage (HMP) > 40%. We still do not know when HM starts, what causes hip displacement, how HM changes over time, and how to halt this migration to avoid surgery. We assume that monitoring HM over time and describing the temporal sequence of events will help to answer these questions.

The aim of the present study was to estimate the prevalence of children with at least one hip with HMP >40% in a homogeneous population of non-ambulant children with CP, compare this prevalence to that in other patient samples, and estimate the prevalence of HMPs for which surgery could be avoided with a better prevention program. Another aim was to model HM changes over a 2.6-year mean follow-up and identify possible factors that influence these changes.

Material and methods

Study design and setting

This was an observational, prospective, multicenter cohort study. The patients were recruited from 51 French healthcare facilities: university hospitals, general hospitals, and public centers for disabled children located in metropolitan France, Reunion Island, Martinique, and Tahiti. The study involved 67 investigators observing children.

Ethical approvals were obtained from the institutional review board “*Comité de Protection des Personnes Lyon Sud Est II*” (Agreement 00009118). In accordance with the Declaration of Helsinki, written informed consent was obtained from each participant or legal representatives.

Participants

The recruitment for a larger non-interventional “parent” survey (ClinicalTrials.gov identifier: NCT01840930) began in September 2009 and is still ongoing. To avoid sources of bias, the investigators were asked to consecutively include all eligible patients from their sites. The present ancillary cohort study enrolled children with non-progressive bilateral CP diagnosed before age 2 years, who were 2 to 11 years old at inclusion, and had CP with GMFCS level IV (mobility requires physical assistance or powered wheelchair) or V (use of manual or powered wheelchair in all settings and limited ability to maintain antigravity head and trunk postures and control leg and arm movements). The analysis concerned data for a subset of children with at least one hip with HMP >40%.

Data collection

At baseline and at each annual visit, the investigators collected medical and radiological data on a standardized form.

The medical data included history of prematurity, history of orthopedic surgery, presence of seizures (>1 crisis per month), ability to communicate (fluent speech, reliable yes/no code or none), mobility according to the GMFCS (2), body mass index (BMI), and treatment for spasticity (intrathecal or oral baclofene, botulinum toxin A [BoNTA], or selective dorsal rhizotomy). With BoNTA use, the investigators collected the dose and the muscles involved (proximal [iliopsoas, gluteus minimus, adductor, gracilis, rectus femoris, semitendinosus] or distal [gastrocnemius, soleus, tibialis posterior, fibularis, flexor digitorum longus]).

The radiological data included HMP as measured by physicians (rehabilitation specialists) with the Reimers index (11) despite limitations reported in a recent review and because it is the reference method used in CP. We tested the interrater reliability in a sample

of measurements suggesting good results as reported in the literature (12). HMP had to follow a standard measurement procedure on an anteroposterior pelvic radiograph taken with the child in the supine position (Fig. 1).

For assessing HMP change over time, only children with HMP $\leq 40\%$ on the left or right hip were included because of the need for surgical intervention at HMP $>40\%$ (13-15). The maximum follow-up was 5 years, with one HMP measurement per year and hip. Children with only one measurement were excluded and those who underwent hip replacement surgery under the cotyle were censored at the date of surgery.

Statistical analysis

The present study did not include a sample-size calculation. For this preliminary report on the first 235 patients with a 5-year follow-up (enrolled until 2014), the main objective was to estimate the cumulative incidence of HMP $>30\%$ at various ages. Nevertheless, the original study was powered to estimate HMP $>30\%$ after a 12-year follow-up with precision of 4% in 385 participants (4).

Participant characteristics are described with mean (SD) for quantitative variables and frequency (%) and relative frequency for categorical variables. The prevalence of children with at least one HMP $>40\%$ was estimated with its 95% confidence interval (CI).

The lines that depicted changes in HMP over time showed high heterogeneity between patients. The usual statistical approaches (e.g., mixed models) are able to show mean trends but not patterns. In the absence of a single (homogeneous) line or shape able to summarize the changes, we used group-based trajectory modelling (16). The objective of the latter approach was to estimate sets of parameters that define shapes for change trajectories and probabilities of patient contributions to each trajectory. Thus, differences in trajectories may be summarized by a small number of polynomial functions of time, each of which corresponds to

a trajectory group (i.e., patients following approximately the same changes). Models with various numbers (2, 3, or 4) of trajectory groups were fitted and the final number was statistically determined (16). The trajectory groups with their 95% CIs were graphed and the proportion of the whole patient population in each trajectory was calculated. HMP changes were separately modelled for right and left hips. Comparisons of patient characteristics (at inclusion) between the less favorable trajectory group and the others involved Student *t* test for quantitative variables and chi-square test for categorical variables. Multiple comparisons were corrected by controlling the false discovery rate.

All statistical analyses involved using SAS v9.3. Trajectory-group modeling involved use of SAS PROC TRAJ (17, 18). All tests for differences were two-tailed and $p < 0.05$ was considered statistically significant.

Results

The mean (SD) age of the 235 children (130 girls) was 6 years 4 months (range 2 years 4 months to 10 years 11 months) (Table 1). One third (31.2%) were at GMCSF level IV and two thirds (68.8%) level V (one missing datum). One half (48.9%) were non-communicating and one quarter (22.9%) had active epilepsy.

At inclusion, the prevalence of HMP >40% was estimated at 16.4% (37/225, 10 missing data) for right hips, 14.8% (33/223, 12 missing data) for left hips, and 6.4% (14/218, 17 missing data) for both hips. The prevalence of at least one hip with HMP >40% was estimated at 24.3% (53/218 [95% CI 18.6–30.0]).

For trajectory analysis, 144 patients participated in the analysis of right HM and 150 left HM. (see Fig. 2). Asymmetric HMPs were found in 17.9% of children (39/218, 17 missing data): 21 had HMP >40% on the right side and \leq 40% on the left side (vice versa in the 18 left). We found pelvic obliquity in 24.4% of children with symmetric HMP (43/176, 3

missing data) and 51.4% with asymmetric HMP (19/37, 2 missing data) ($p=0.002$). At inclusion, the median HMP varied by age: from 19% to 26% in the right hip and from 18% to 25% in the left hip. High HMPs were observed even in very young children (Fig. 3).

The trajectory modelling over a median follow-up of 2.7 years (range 0.4–6.3; mean 2.6) allowed for identifying 3 typical HMP changes over time in left and in right hips. Trajectory 1 represented children with HMP decreases over time (10.4% and 8.7% of children for the right and left hip; median HMP from 26% to 5% for the right hip and 26% to 0% for the left hip). Trajectory 2 represented children with stable HMP over time. Trajectory 3 represented children with HMP increases over time (22.2% and 12.0% of children for the right and left hip; median HMP from 12% to 30% for the right hip and 5% to 38% for the left hip) (Fig. 4). Many children (67.4% and 79.3% for the right and left hip) belonged to trajectory 2, and the median HMP was about 20% (Table 2). Children's characteristics by trajectory group are in Table 3. Trajectory 1 children did not differ from other trajectories in medical data.

Discussion

HM in children with CP has been widely studied, but to our knowledge, its prevalence measured by a well-defined criterion and its early and long-term follow-up in a homogeneous non-ambulant population has not been investigated. Here we found that 1) the prevalence of HMP >40% was 24.3% in our sample; 2) HMP >40% was not necessarily found in older children; 3) pelvic obliquity was linked to asymmetric HM; and, 4) we could distinguish 3 different trajectories of progression of HM.

In the 1980s, the prevalence of HM in non-ambulant children with CP ranged from 17/54 to 15/22 (5, 19, 20). It decreased progressively thereafter. The 24.3% prevalence of HMP >40% we found is close to values found in the international literature. In a study by

Hägglund et al. (21), 38/212 (18%) children aged 9 to 16 years had hip displacement with HMP >40% whatever the type or severity of CP, and this decrease was further confirmed in 2014.

Among the 3 patterns of HM progression we found, the stabilization pattern over 5 years concerned 2 of 3 children. This finding might be due to the care of non-ambulant children with CP according to current recommendations (10). One shortcoming of the study is that we could not determine one or more specific factors able to predict belonging to a specific progression pattern. For example, the use of BoNTA was tested because of its efficacy for spasticity in children with CP (10) but showed no significant outcome differences between groups. This observation may confirm the results of a previous randomized study (22) finding that BoNTA has very modest benefit and does not prevent HM in children with CP. A future randomized trial among “worsening HMP” patients (trajectory 3) may assess the effectiveness of BoNTA in preventing or halting HM. A recent retrospective analysis of medical records of 709 children showed that hip subluxation and coxa valga deformity were correlated with dynamic spasticity and shortening of hip adductor muscles (23). Future analyses of the current sample will have more power to explore the effect of the same covariates on HMP.

Hägglund et al. (21) reported that mean age at the first record of hip displacement was 4 years but that some hips showed HMP >40% at age 2 years. The present study confirms this observation because the median proportion of HMP did not change by age; thus, the risk of HM (whatever its severity) might occur well before age 3 years, which justifies screening for HM as early as age 2 years (10). This early screening would prompt treatments to avoid reaching the HMP threshold of >40%, which calls for surgery.

A goal of prevention seems not the luxation but rather migration >40%. An HMP of about 30% but <40% could be tolerable and would have no functional consequences provided

the migration is practically symmetric and stable over time. Then, early prevention programs should pay particular attention to early abduction in the seated position and to right and comfortable night and day postures and equipment. The barriers to and facilitators of early prevention should also be explored to improve implementation of such preventive actions (24).

HM should not be considered in isolation but should be integrated in a general setting of orthopedic dynamics. Specific attention should be paid to control asymmetric migration and prevent pelvic obliquity and the risk of scoliosis. Several authors have reported on the associations among HM, pelvic obliquity, and scoliosis and highlighted an increased risk of scoliosis in the presence of pelvic obliquity (5, 6, 25-27). The association between HM and pelvic obliquity demonstrated by Letts et al. (20) in 1984 has never been questioned. Here, we found HM asymmetry significantly linked with pelvic obliquity. In the specialized literature, screening programs for prevention concern mainly the prevention of hip luxation. A better knowledge of HMPs and their changes over time will help achieve this goal of integrated management and hopefully avoid bone surgery at HMP >40% (8, 9, 15, 28).

Study limitations

This was an intermediate study; a future study is already planned to explore the evolution of HMP >40% over 5 more years (up to 10 years). Treatment with BoNTA (the factor with an impact on the progression of HM) was considered only at inclusion, but BoNTA was administered at various doses, muscle sites, and frequencies. Its related results should be interpreted with caution and BoNTA use should be given specific attention in future research.

Other limitations are different practices across centers (including BoNTA use, musculoskeletal management), the lack of standardized measurement of HMP in this population, and the lack of power in establishing the profiles and predicting HMP evolution.

Finally, data on posture, seating systems, night systems, and verticalization were collected, but their continuous changes and the lack of information on compliance did not allow for reliable analyses.

Conclusion

Preventing HM in non-walking children with cerebral palsy should begin at an early age with the objective of maintaining HMP <40% as long as possible. In the present sample, we found a prevalence of at least one hip with HMP >40% of about 24%. We identified 3 patterns of HM progression in this population. Hips of most children remained stable over time. We failed to identify a factor responsible for belonging to a trajectory. To date, the processes that cause the appearance and progression of HM in these children are still unknown. Having shown the existence of a group of children in which HM is prone to aggravation, a future study should search for factors able to predict this aggravation.

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Conflicts of interest. None declared.

Legends

Figure 1. Standardization of hip migration measurement.

Figure 2. Study flow chart

Figure 3. Distribution of right and left hip migrations over 1-year age classes at inclusion.

The horizontal line indicates the median. The “+” indicates the mean. Box edges are the 25th and the 75th percentile; whiskers are range. The circles indicate outliers (points located at >1.5 times the interquartile range of the box).

Figure 4. Modelled trajectories of migration percentage changes over time (with 95% confidence intervals). A: right hip, B: left hip. Groups 1, 2, and 3: decline, stability, and aggravation of hip migration, respectively. Median follow-up of 2.7 years (range 0.4–6.3; mean 2.6).

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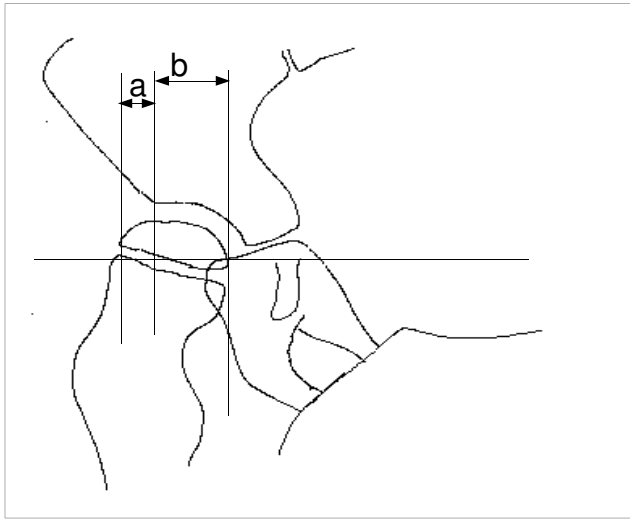
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Figure 1.



Drawing of an anteroposterior pelvic radiograph in supine position.
Migration percentage (MP) = $a / (a+b)$

Figure 2.

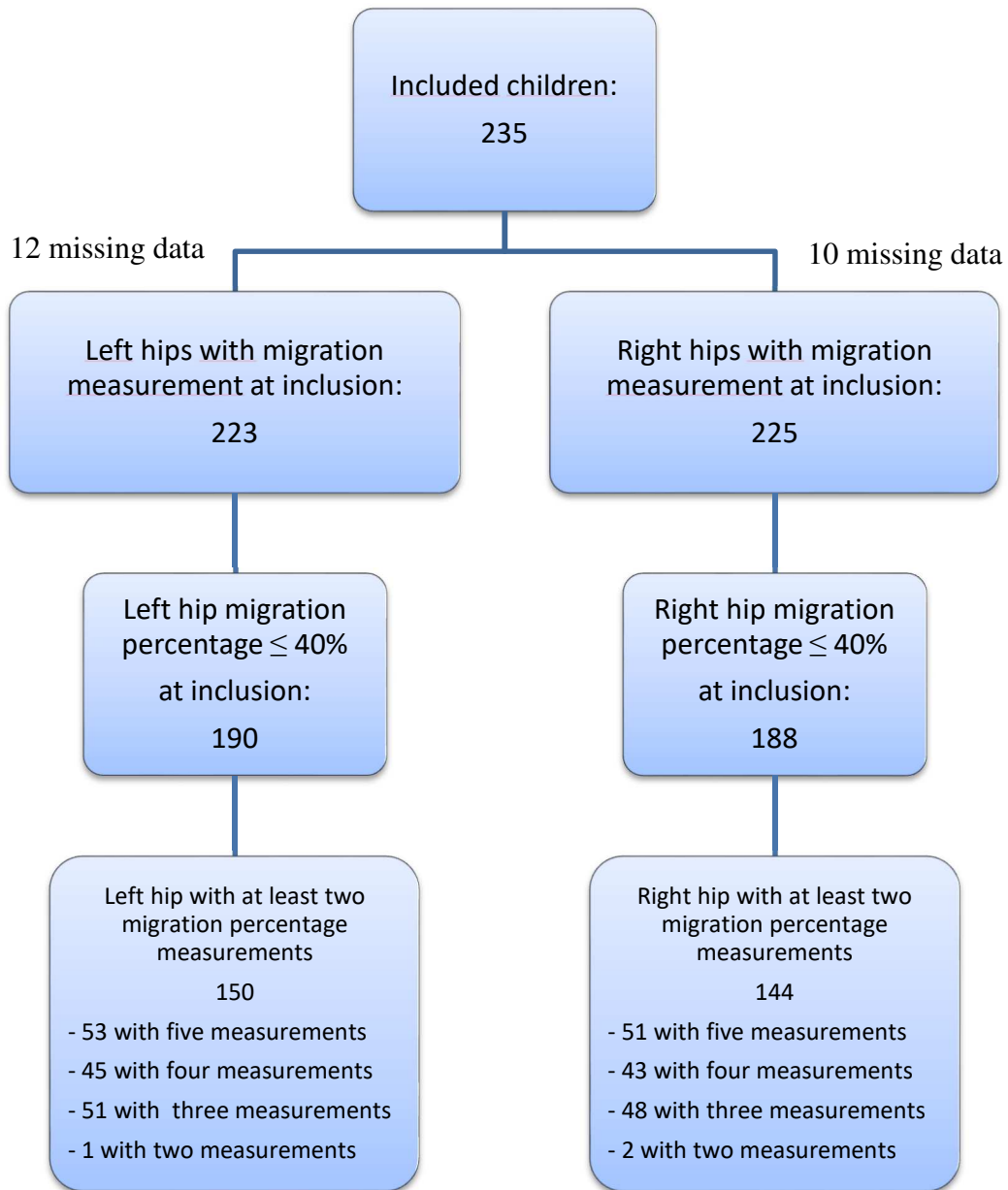


Figure 3.

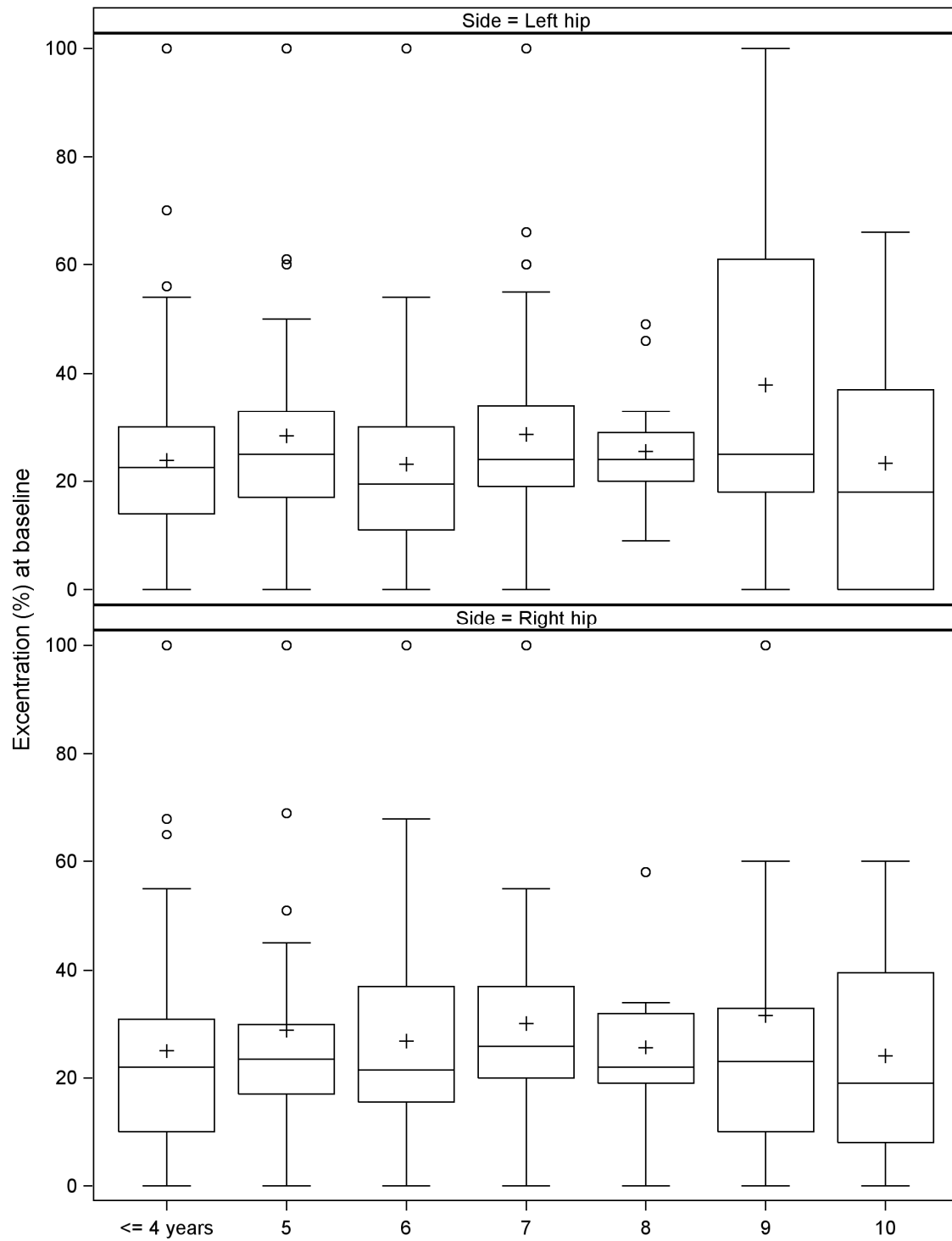


Table 1. Demographic and clinical characteristics at inclusion for children with cerebral palsy (n=235).

Characteristics	N (%) ¹
Sex	
Boys	130 (55.3)
Girls	105 (44.7)
Age (years.months)	
Mean (SD) (range)	6.4 (2) (2.3–10.11)
Prematurity (<37 WG)	100 (44.3)
Prenatal etiology	78 (33.2)
Cerebral malformations	51 (21.7)
Maternal pathology	11 (4.7)
Others	16 (6.8)
Perinatal etiology	129 (54.9)
Prematurity	86 (36.6)
Hypoxic ischemic encephalopathy	43 (18.3)
Others	10 (4.3) ³
Postnatal etiology	37 (15.7)
Epileptic encephalopathy	13 (5.5)
Infection	12 (5.1)
Others	13 (5.5) ⁴
GMFCS level ¹	
IV	73 (31.2)
V	161 (68.8)
Non-communicating children	115 (48.9)
Active epilepsy	1 (22.5)

¹ Percentage of whole cohort; 1 missing data

² GMFCS: Gross Motor Function Classification System (only levels IV and V were studied)

³ Of 235 children, 23 lacked data on perinatal etiology and 83 had missing data. Some children had several etiologies.

⁴ Of 235 children, 40 lacked data on postnatal etiology, and 158 had missing data. Some children had several etiologies.

WG, weeks' gestation

Table 2. Observed changes in hip migration over time among trajectory groups.

Hip	Stability (Group 2)				Aggravation (Group 3)				Improvement (Group 1)				
	n	med	min	max	n	med	min	max	n	med	min	max	
Right													
y0	97	20	0	40	32	12	0	36	15	26	10	39	
y1	95	21	0	44	32	21.5	0	60	15	15	0	47	
y2	61	21	0	39	24	28	5	46	9	6	0	31	
y3	32	20	0	41	12	29.5	14	56	7	5	0	21	
Left													
y0	119	21	0	40	18	5.5	0	33	13	26	21	38	
y1	118	22	0	50	18	21	0	48	13	0	0	25	
y2	77	23	0	41	14	25.5	0	55	7	0	0	31	
y3	41	22	0	47	10	38	14	100	2	0	0	0	

y0, at inclusion; y1, y2, y3, year 1, 2, 3 after inclusion

Table 3. Participant characteristics at inclusion per hip migration pattern.

	Decline (Gr. 1)		Stability (Gr. 2)		Aggravation (Gr. 3)		Decline or stability		Aggravation vs. other status
	% or mean (SD)	n	% or mean (SD)	n	% or mean (SD)	n	% or mean (SD)	n	<i>p</i> -value*
Right hip	10.4	15	67.4	97	22.2	32	77.8	112	-
Age (years)	6.61 (2.06)	15	6.40 (1.90)	97	5.38 (1.92)	32	6.43 (1.92)	112	0.15
Male	46.7	7	51.5	50	56.25	18	50.9	57	0.86
Born <37 WG	53.3	8	52.2	48	36.7	11	52.3	56	0.54
Non-communicating	46.7	7	44.3	43	46.9	15	44.6	50	0.93
Active epilepsy	6.7	1	16.5	16	25.0	8	15.2	17	0.54
BoNTA	40.0	6	49.5	48	37.5	12	48.2	54	0.64
Past M-T surgery	13.3	2	18.6	18	6.3	2	17.9	20	0.54
GMFCS Level 5	66.7	10	68.0	66	62.5	20	67.9	76	0.86
Unsuitable rehabilitation	26.7	4	33.0	32	21.9	7	32.1	36	0.69
BMI z-score	-0.47 (2.46)	15	-1.06 (1.87)	91	-1.29 (1.71)	32	-0.98 (1.96)	106	0.75
Left hip	8.7	13	79.3	119	12.0	18	88.0	132	-
Age (years)	5.70 (1.86)	13	6.24 (1.94)	119	5.69 (1.83)	18	6.18 (1.93)	132	0.75
Male	53.8	7	53.8	64	72.2	13	53.8	71	0.54
Born <37 WG	50.0	6	49.6	57	33.3	6	49.6	63	0.54
Non-communicating	61.5	8	42.9	51	61.1	11	44.7	59	0.54
Active epilepsy	30.8	4	21.0	25	22.2	4	22.0	29	0.99
BoNTA	46.2	6	47.1	56	33.3	6	47.0	62	0.64
Past M-T surgery	15.4	2	15.1	18	0.0	0	15.2	20	0.54

	Decline (Gr. 1)		Stability (Gr. 2)		Aggravation (Gr. 3)		Decline or stability		Aggravation vs.
	% or mean (SD)	n	% or mean (SD)	n	% or mean (SD)	n	% or mean (SD)	n	other status <i>p</i> -value*
GMFCS level V	69.2	9	66.4	79	72.2	13	66.7	88	0.93
Unsuitable rehabilitation	61.5	8	29.4	35	38.9	7	32.6	43	0.86
BMI z-score	-0.88 (2.62)	13	-1.04 (1.81)	113	-1.05 (1.89)	18	-1.02 (1.90)	126	0.93

WG, weeks' gestation; BoNTA, botulinum toxin A; M-T, muscle-tendon; GMFCS, Gross Motor Function Classification System; BMI, z-score of the body mass index in kg/m².

* After adjustment for multiple comparisons (false discovery rate methodology)

Figure 4.

