Abstract

Qualitative abnormalities of spontaneous motor activity in newborns and young infants are early predictive markers for later spastic cerebral palsy. Aim of this research was to identify which motor patterns may be specific for later dyskinetic cerebral palsy. In a large, prospectively performed longitudinal study involving four European hospitals we identified twelve cases with the relatively rare condition of dyskinetic cerebral palsy and compared their early motor development with twelve spastic cerebral palsy cases and twelve controls. From birth to the fifth month post-term, all infants were repeatedly videoed and their spontaneous motor patterns, including general movements, were assessed. Until the second month post-term, the infants that later became dyskinetic displayed a poor repertoire of general movements, “arm movements in circles” and finger spreading. Abnormal arm and finger movements remained until at least five months and were then concurrent with a lack of arm and leg movements towards the midline. Later dyskinetic infants share with later spastic infants the absence of fidgety movements, a spontaneous movement pattern that is normally present from three to five months. Qualitative assessment of spontaneous motor patterns enabled us to identify infants at high risk for dyskinetic cerebral palsy early in life. Additionally, we were able to discriminate them from those infants at high risk for later spastic cerebral palsy. This is a matter of significant clinical relevance because the two types of cerebral palsy ask for different management and early intervention.

Key words
General Movements · Cerebral Palsy · Dyskinetic Movements · Newborns · Fidgety Movements · Young Infants

Abbreviations
ATNR asymmetrical tonic neck response  
CP cerebral palsy  
CT computer tomography  
FM fidgety movements  
GM general movements  
MRI magnetic resonance imaging  
PMA postmenstrual age  
UIPE unilateral intraparenchymal echodensity  
US ultrasound

Introduction

The prevalence of cerebral palsy (CP) (about 2‰) has changed very little over the past forty years in spite of the many technological advances in perinatology that have reduced mortality in compromised neonates [9]. The spastic form of CP is the more common form while dyskinetic CP has a frequency of only 10 to 15% of all CP forms [15, 29]. The dyskinetic group comprised syndromes of choreo-athetosis as well as the more disabling dystonic forms [15]. As long as there is no neonatal magnetic resonance

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imaging (MRI) easily available, it is still difficult to predict early, which infant will be affected by CP, and it is even more difficult to predict its specific form. A number of signs might raise the index of suspicion for developing CP. Infants who are hypotonic and open their mouths excessively [9], who have attacks of opisthotonus [23], who are less active than expected, who move one limb less than the others, who keep their hands fistled after three months of age [32], who are late in beginning to smile [18], or who have a persistent asymmetric tonic neck response [29] might be suspect. However, none of the motor development scales such as the Movement Assessment of Infants, the Motor Scale of the Bayley Developmental Test, Paine’s Protocol, or the Assessment of a Delay in Motor Milestones ever improved on a sensitivity of more than 70% [2, 12, 16, 28]. By contrast, the number of cases correctly identified in the first weeks of life by more comprehensive neurological examinations [3, 10, 31] is remarkably high, but the number of false positive results, however, is not low enough [7, 8, 21].

A new technique for assessing the spontaneous motor activity has been recently introduced, suitable to identify and distinguish between those infants who require early surveillance and intervention for neurological abnormalities and those who do not [24, 26]. The quality of general movements (GMs), gross movements involving the whole body in a variable and complex sequence and occurring from early fetal age onwards until maximally six months after term [25], is assessed. The absence of fidgety movements (FM s), small, circular and elegant movements of neck, trunk and limb [24], observable in the three- to five-month-old infant, is a reliable and specific marker for CP. In addition, infants who develop CP also have abnormal GMs before their third month. Usually cramped-synchronised GMs precede the absence of FMs [24]. These rigid GMs, which lack the normal smooth and fluent character, have already been reported to be highly predictive, particularly for the spastic forms of CP [13]. Returning to the data of the individual developmental trajectories of the GMs reported in the multi-centre study by Prechtl et al [24], we found that the one infant who later developed dyskinetic CP followed another general movement trajectory than the 48 infants who later developed spastic CP. During the first months of life, prior to the absence of FMs, the GMs of this infant lacked their normal complexity and variability although they had not been considered as cramped-synchronised.

The considerable difference in the quality of life and in the type of management of patients with spastic versus dyskinetic forms of CP makes it important to search for early specific signs. This investigation was possible within the context of our large, prospectively collected longitudinal data which included twelve dyskinetic cases.

Subjects and Methods

Subjects

This study is part of a large ongoing research project in which neurological and developmental data from more than 300 cases were prospectively collected in four European centres, i.e., Graz, Groningen, Modena and Pisa. The twelve infants (Group D) for whom the development of dyskinetic CP was reliably known, were recruited from this study population. The four girls and eight boys were born between 1986 and 1999. For comparison, 24 other infants were selected according to their postmenstrual age (PMA) at birth, birth weight and number of movement assessments. Twelve of them had developed spastic CP (Group S). These three girls and nine boys were born between 1988 and 1994. The severity of motor impairment, assessed with the Gross Motor Function Classification System [22] was similar between the dyskinetic (mean = 3.7, SD = 1.4) and the spastic group (mean = 3.5, SD = 1.4). The other six girls and six boys, born between 1984 and 1990, were neurologically at risk because of pre- or perinatal complications, but had developed neurologically normal (Group N). For all 36 infants repeated observations of their spontaneous motor activity at various ages until 26 weeks post-term, results of brain ultrasound scans, and standardised repeated neurological examinations, were available. In all three groups eight infants were born at term, and four were born preterm (26 to 33 weeks PMA). The infants’ birth weights ranged from 820 g to 3800 g (median: 2945 g, P25: 1723 g, P75: 3480 g) in Group D; from 950 g to 4150 g (median: 2540 g, P25: 1375 g, P75: 3355 g) in Group S; and from 800 g to 4350 g (median: 3140 g, P25: 1255 g, P75: 3723 g) in Group N. Details are given in Table 1.

Neuroimaging scans (cranial ultrasound [US], brain computer tomography [CT] or magnetic resonance imaging [MRI]) were carried out in all infants. In the neonatal period all infants had serial US scans. Neonatal MRI was performed in three infants of the dyskinetic group and in one of the spastic group. Most of the CP cases had an MRI scan during their first year of life, but in a few cases, the first MRI was carried out in their second year of life. This heterogeneity is due to the large scatter in the sampling of the cases. In addition, different MRI systems and protocols were used in the different centres. Only one infant with normal outcome had an MRI scan, performed at 6 months of age. All CP cases had signs of brain lesions. In the dyskinetic group, five infants showed basal ganglia lesions (associated with diffuse white matter lesions in two cases) and the other seven infants had periventricular white matter changes (associated with focal occipital infarct in one infant). In the spastic group, seven infants showed diffuse white matter lesions (associated with basal ganglia lesions in two cases), four infants had periventricular white matter damage, and one a unilateral periventricular lesion. US scans of the normal infants either were completely normal or showed very mild intraventricular haemorrhage or short-lasting transient periventricular density.

All parents had given their informed consent and had agreed to participate in the follow-up study. The procedure followed during this study was in accordance with the requirements of the ethical committees of the centres involved.

Observation of spontaneous movements

During video recording, the infants were partially dressed (e.g., wearing a “body” only), lying supine, at least half an hour after a feed, and during periods of active wakefulness (for details of the recording technique see [11]). The very young infants were recorded in the incubator. After discharge from the hospital, the infants were recorded in the outpatients’ clinic or at home. From birth until the end of the second month post-term, at most ten video recordings were made (median: 2). From all but two infants, four to 70 minutes of recording were available for further analysis (median: 21 minutes, P25: eight minutes, P75: 28 mi-
Two infants who later developed dyskinetic CP were not recorded before three months. During the age of three to five months, when FMs are normally observed, two recordings were made on average (range: 1 to 4) to ensure that the presence of FMs as well as concurrent movements and their quality (normal or abnormal) could be assessed correctly. For this age six to 65 minutes (median: 28 minutes; P25: 13 minutes; P75: 42 minutes) could be used for further analysis.

**Assessment of general movements**

During the playback of video recordings the quality of GMs was assessed by global judgement concerning normal or abnormal by C.E., G.C., F.F., and A.B. at their respective centres (for review of the methodology see [11]). In addition, those of us who were blind as to the infants' clinical histories and outcomes also independently assessed the GM quality (C.E., G.C., F.F., A.B., H.F.R.P.). The interscorer agreement was 92% and hence, similar to the reliability data previously published for the method on qualitative assessment of GMs [11].

From birth until the end of the second month, normal GMs are gross movements involving arms, legs, the neck and trunk in a variable sequence. They wax and wane in intensity, force and speed, and they have a gradual beginning and end. The majority of sequences of extension and flexion movements of arms and legs are complex, with superimposed rotations and often slight changes in the direction of the movement. These additional components make the movements fluent and elegant and create the impression of complexity and variability [25]. Types of abnormal GMs before the third month are “poor repertoire” and “cramped-synchronised” movements. An infant’s movement repertoire is considered poor when a sequence of successive movement components is monotonous and movements of the different body parts do not occur in the complex way seen in normal GMs. Cramped-synchronised GMs look rigid and lack the smoothness and fluency which are a characteristic feature of normal GMs; all limb and trunk muscles contract and relax almost simultaneously [13, 24].

Normal FMs are elegant circular movements of small amplitude and moderate speed and variable acceleration of neck, trunk and limbs in all directions [24]. They differ significantly from involuntary dyskinesia, which appears forced. FMs are a transient phenomenon. They gradually emerge at the beginning of the third month, come to full expression and then taper off again gradually at the end of the fifth month in both term and preterm infants at corrected age. Usually they coexist with other movements. If FMs are not observed at all during the third to fifth month, this abnormality is called “absent FMs”.

**Assessment of other movement patterns**

FMs are usually concurrent with other movements, such as head, arm and leg movements towards the midline; mutual manipulation of fingers; fiddling of clothing; reaching and touching; kicking; legs lift with flexion or extension at knees very often with hand-leg (knee) contact; trunk rotation; and axial rolling [17]. We also noted if spontaneous head turning was followed by an asymmetric tonic neck response (ATNR), and if this response could be overcome (with spontaneous flexion of the jaw-arm) or if it was obligatory. The presence and quality (normal or abnormal) of these motor patterns were assessed by C.E. and G.C. The former was unaware of the infants’ clinical histories. Furthermore, any other abnormal motor pattern was carefully noted. The interscorer agreement between G.C. and C.E. was 97% (kappa: 0.94).

**Neurological follow-up examination**

Neurological assessments were carried out repeatedly. After the neonatal period (preterm age: [10]; term age: [10, 27]) the neurological condition was assessed by an age-adequate neurological

| Table 1: Postmenstrual age at birth (in weeks), birthweight (in increasing order in grams), and neurological outcome (age at last assessment) of the 36 study cases | Case | PMA Birthweight | Outcome | Case | PMA Birthweight | Outcome | Case | PMA Birthweight | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|
| D9 | 27 | 820 | dyskinetic TP (5 ys) | S9 | 26 | 950 | spastic DP (6 ys) | N9 | 26 | 800 | Normal (5 ys) |
| D1 | 29 | 1400 | dystonic TP (10 ys) | S1 | 28 | 1340 | spastic DP (7 ys) | N1 | 27 | 850 | Normal (8 ys) |
| D10 | 29 | 1710 | dystonic TP (5 ys) | S10 | 28 | 1250 | spastic DP (10 ys) | N10 | 27 | 1100 | Normal (4 ys) |
| D12 | 32 | 1760 | dystonic TP (5 ys) | S12 | 30 | 1480 | spastic DP (6 ys) | N12 | 33 | 1920 | Normal (8 ys) |
| D11 | 37 | 2700 | Bilat. HP, dyskinetic movs (5 ys) | S11 | 37 | 2280 | spastic HP (11 ys) | N11 | 37 | 2180 | Normal (11 ys) |
| D6 | 40 | 2850 | choreo-athetoid TP (12 ys) | S6 | 40 | 2650 | spastic DP (11 ys) | N6 | 40 | 2980 | Normal (6 ys) |
| D7 | 40 | 3040 | choreo-athetoid TP (10 ys) | S7 | 38 | 2430 | spastic DP (3 ys) | N7 | 39 | 3300 | Normal (4 ys) |
| D8 | 40 | 3250 | dystonic TP (11 ys) | S8 | 40 | 3870 | spastic DP (11 ys) | N8 | 40 | 3300 | Normal (5 ys) |
| D3 | 40 | 3420 | dystonic TP (5 ys) | S3 | 40 | 3220 | spastic TP (3 ys) | N3 | 40 | 3550 | Normal (5 ys) |
| D4 | 39 | 3500 | dystonic TP (4 ys) | S4 | 41 | 3050 | spastic TP (12 ys) | N4 | 40 | 3780 | Normal (3 ys) |
| D2 | 40 | 3630 | dystonic TP (3 ys) | S2 | 37 | 4150 | spastic TP (14 ys) | N2 | 41 | 4230 | Normal (4 ys) |
| D5 | 40 | 3800 | dystonic TP (5 ys) | S5 | 40 | 3400 | spastic TP (8 ys) | N5 | 40 | 4350 | Normal (5 ys) |

Group D: Children with dyskinetic cerebral palsy; Group S: Children with spastic cerebral palsy; Group N: Children with a neurologically normal development; PMA: postmenstrual age; TP: tetraplegia; DP: diplegia; HP: hemiplegia; ys: years; bilat: bilateral; movs: movements.
We hardly ever observed normal movement patterns in Group D. Other movement patterns were "arm movements in circles" and long-lasting finger spreading. "Arm movements in circles" occur unilaterally or bilaterally. These forward shoulder rotations are monotonously slow and of large amplitude. The arms are either slightly flexed or fully extended. From birth until the end of the second month these movements could be observed in eight out of ten infants (p < 0.01). These infants, and one additional case, showed these abnormal arm movements from the third to the fifth month as well (p < 0.005). In case D9, however, the speed of the movements was faster in comparison to the others. In addition, this infant had small amplitude zig-zag arm movements.

From the third month onwards, only four infants from Group D had self-initiated movements towards the midline (p < 0.01), but three of them never showed any fiddling of clothes or mutual hand manipulation. Only Case D11 displayed the normal variety of movements towards the midline. Turning now to the specific movement patterns towards the midline, it was surprising to note that apart from Case D11, who showed normal foot-foot contact (plantar to plantar touching), only two other infants had foot-foot contact but of an abnormal quality, namely contact on the tibial side of the feet (p < 0.01).

Finally, during the third month normal leg lifting was only observed in one out of twelve infants who later developed dyskinetic CP (Case D11; p < 0.005). "Arm movements in circles" is not characteristic for later spastic cases. These movements were occasionally observed in two cases only; in one infant during preterm age (p < 0.05 versus Group D; n.s. versus Group N) and in another at three months (p < 0.005 versus Group D; n.s. versus Group N). Another difference between the two CP groups concerned finger spreading. In Group S, only one infant had a very short period of finger spreading during preterm age (p < 0.05 versus Group D; n.s. versus Group N). Movements towards the midline, including foot-foot contact were observed in all infants in Group S (p < 0.01 versus Group D; n.s. versus Group N). However, these movements were restricted to the lower extremities in five infants. They did not show any head or hand movements towards the midline. One infant also showed abnormal foot-foot contact similar to Group D (p < 0.05 versus Group D; n.s. versus Group N). Only three out of twelve infants had legs lifting (n.s. versus Group D; p < 0.05 versus Group N).

In all twelve infants in Group N, the rich repertoire of age specific normal movements was observed. The quality of these movements was normal. Only one infant did not have any leg lifting before five months.

**Quality of general movements from three to five months**

None of the infants who later developed CP ever displayed FMs. This holds true for both Groups D and S (p < 0.001). Again, all 12 infants who later developed normally also had normal FMs.

**Assessment of the spontaneous asymmetric tonic neck response (ATNR)**

This postural pattern was present in ten out of twelve infants in Group D until the end of the fifth month. However, in all infants the ATNR could be overcome with spontaneous flexion of the jaw-arm. Thus, ATNR was not obligatory in any of the dyskinetic cases.

All infants in Group S displayed ATNRs until five months. In half of them the ATNR was obligatory during their third to fifth month (n.s.). In Group N an ATNR, which could be overcome, was observed in three out of twelve infants until four to five months; none had an obligatory ATNR.

**Results**

**Quality of general movements from birth until the end of the second month**

All infants who later developed CP had abnormal GMs. In Group D, nine out of ten infants had a poor movement repertoire (p < 0.005). GM quality of the remaining infant could not be assessed because it was not in the required behavioural state. In Group S, cramped-synchronised GMs (p < 0.005) were observed in all infants but one. However, in four cases a poor repertoire preceded the cramped-synchronised quality of movements. The remaining infant had a poor movement repertoire but was only observed once at two weeks post-term. All twelve infants who later developed normally displayed normal GMs (p < 0.001).

**Quality of general movements from three to five months**

None of the infants who later developed CP ever displayed FMs. This holds true for both Groups D and S (p < 0.001). Again, all 12 infants who later developed normally also had normal FMs.

**Other movement patterns**

We hardly ever observed normal movement patterns in Group D. By contrast, their most conspicuous abnormal motor patterns were "arm movements in circles" and long-lasting finger spreading. "Arm movements in circles" occur unilaterally or bilaterally. These forward shoulder rotations are monotonously slow and of large amplitude. The arms are either slightly flexed or fully extended. From birth until the end of the second month these movements could be observed in eight out of ten infants (p < 0.01). These infants, and one additional case, showed these abnormal arm movements from the third to the fifth month as well (p < 0.005). In case D9, however, the speed of the movements was faster in comparison to the others. In addition, this infant had small amplitude zig-zag arm movements.

Long-lasting finger spreading with fully extended or semiflexed fingers was frequently observed. The normal, variable finger movements were absent. Instead, the infants spread their fingers unilaterally or bilaterally, sometimes followed by short fisting and spreading once again. Eight out of ten infants in Group D had finger spreading even before the end of the second month (p < 0.01). This feature remained in nine out of twelve infants during the third to fifth month (p < 0.01).

In all twelve infants in Group N, the rich repertoire of age specific normal movements was observed. The quality of these movements was normal. Only one infant did not have any leg lifting before five months.
The main differences and similarities of early motor patterns among the three groups are summarised in Table 2.

**Discussion**

By means of a functional assessment technique, the present study is the first of its kind to document early markers specifically predictive for the development of dyskinetic CP. Besides a poor repertoire of GMs in most of the infants who later developed dyskinetic CP, “arm movements in circles” and finger spreading were observed as early as during the first two months of life. Characteristically, these abnormal arm and finger movements remained until at least five months post-term. A lack of movements towards the midline, particularly foot-foot contact, was an additional specific sign of dyskinetic cases. Common to both dyskinetic and spastic cases during their third to fifth month, was the absence of FMs and antigravity movements, i.e., leg lifting. By contrast, as reported earlier [18, 29, 32], an obligatory asymmetric tonic neck response before the sixth month did not discriminate between infants who developed normally and those who developed CP.

The abnormal unilateral or bilateral “arm movements in circles” cannot be confused with normal writhing or swiping arm movements, which are typical for the first two to three months after term. The normal writhing character of GMs consists of ellipsoid arm movements of variable speed, intensity and amplitude, whereas swiping arm movements are ballistic, fast, of large amplitude and high speed [17]. In contrast to these normal movements, the abnormal “arm movements in circles” are monotonous, slow forward rotations from the shoulder. Particularly the monotony in speed and amplitude is the most characteristic quality of these stereotyped “arm movements in circles”. Similar arm movements patterns were mentioned by Prechtl [27] in fullterm neonates, while Dubowitz et al [10] refer to “cycling movements” in infants with hypoxic-ischaemic encephalopathy and Amiel-Tison and Grenier [3] describe “wind-milling arm movements” during the first year of life. However, these authors did not relate this phenomenon to dyskinetic CP. The same holds true for the description by Thompson et al [30] of “intermittent bicycling movements of the limbs”. This abnormal pattern in the neonate increases the risk for later unspecified neurological abnormalities. “Bicycling movements of the limbs” were described to be concurrent with fisting of fingers, while our findings showed that “arm movements in circles” were usually accompanied by finger spreading. In later dyskinetic cases we also observed short periods of fisting but they were predominated by long-lasting finger spreading. As far as the spastic cases of our study are concerned, we confirmed the findings of Konishi and Prechtl [19], who reported that later spastic children showed all kinds of finger movements during their early infancy and were not restricted to tight fisting.

In agreement with Cioni et al [6], the one hemiplegic case with significant dyskinetic components had normal movements towards the midline including normal foot-foot contact during its third to fifth month. This infant was the only exception among the dyskinetic cases. Besides the lack of normal foot-foot contact, the majority of them neither displayed hand-hand contact nor hand-mouth contact. This lack of movements towards the midline was described in the context of an obligatory and persistent asymmetric tonic neck response [5]. In our dyskinetic cases, however, the lack of movements towards the midline could not be caused by any asymmetrical postural reaction. Surprisingly, all but one infant who later became spastic showed mutual hand-hand as well as foot-foot contact. In this respect, spastic and normal infants did not differ.

What dyskinetic and spastic infants had in common was the lack of antigravity movements and the absence of FMs. The latter is of particular interest. Prechtl [26] suggested a specific central pattern generator for FMs located, most likely, in the brainstem. The absence of FMs in both forms of cerebral palsy and hence in different brain lesions indicate that intact cortico-spinal fibres as well as the output from the basal ganglia and the cerebellum are necessary to generate normal FMs.

A poor repertoire of GMs during the first months after birth may precede the absence of FMs. This observation suggests that a poor repertoire of GMs should not merely be considered as a mild abnormality, as has been recently suggested [14]. A poor repertoire of GMs and cramped synchronised GMs can both be followed by the absence of FMs, and can lead to CP. As early as

<table>
<thead>
<tr>
<th>From birth until the end of the 2nd month post-term</th>
<th>Abnormal GMs</th>
<th>Arm movements in circles</th>
<th>Finger spreading</th>
<th>ATNR obligatory</th>
<th>From 3 to 5 months post-term</th>
<th>Abnormal FMs</th>
<th>Arm movements in circles</th>
<th>Finger spreading</th>
<th>Absent or abnormal foot-foot contact</th>
<th>Absent leg lift</th>
<th>ATNR obligatory</th>
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<tr>
<td><strong>Group D</strong> (dyskinetics)</td>
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<td><strong>Group S</strong> (spastics)</td>
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<td>9/12</td>
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<tr>
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</tbody>
</table>

Table 2: Conspicuous early signs as markers for the development of later dyskinetic and spastic forms of cerebral palsy.

Group D: Children with dyskinetic cerebral palsy; Group S: Children with spastic cerebral palsy; Group N: Children with a neurologically normal development; GMs: general movements; PR: poor repertoire of general movements; CS: cramped-synchronised general movements; FMs: fidgety movements; ATNR: asymmetric tonic neck response.

* Two cases not recorded in this period, and another one not scorable for GMs.
during the first two months after term, the coexistence of three 
neurological signs, namely a poor repertoire of GMs, “arm move-
mements in circles” and finger spreading, provides a clear marker 
for later dyskinetic CP. The clinical relevance is significant, since 
the two types of CP, the spastic and the dyskinetic form, have a 
different natural history and require different management and 
intervention regimens.

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