Neonatal Cerebral Infarction and Neuromotor Outcome at School Age

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ABSTRACT. Objective. The aim of this study was to assess neuromotor function at school age in children who had cerebral infarction on neonatal magnetic resonance imaging (MRI).

Design. Twenty-two children with evidence of cerebral infarction on neonatal brain MRI (18 with arterial infarction and 4 with border-zone lesions) were assessed at school age with a structured neurologic examination and the Movement Assessment Battery for Children, a battery of tests designed to assess motor function.

Results. Of the 22 children, 6 (30%) had hemiplegia and a further 7 (30%) showed some neuromotor abnormality such as asymmetry on the neurologic examination (n = 4) or poor scores on the neuromotor test without any sign of asymmetry (n = 3). The remaining 9 children had a normal motor outcome. Hemiplegia was found only in children who had concomitant involvement of hemisphere, internal capsule, and basal ganglia on brain MRI. Children with involvement of the internal capsule, associated either with basal ganglia or hemispheric lesions, did not show hemiplegia but still had motor difficulties.

Conclusions. Our results suggest that although hemiplegia occurs in a relatively small proportion of children with neonatal cerebral infarction, other signs of neuromotor impairment can be present, and these become more obvious at school age when a more specific assessment can be performed. These results also suggest that the involvement of the internal capsule on neonatal MRI can predict the presence of these abnormalities. Pediatrics 2004;113:95–100; Mov ABC, MRI, hemiplegia, neonatal cerebral infarction.

ABBREVIATIONS. MRI, magnetic resonance imaging; MCA, middle cerebral artery; Mov ABC, Movement Assessment Battery for Children; PLIC, posterior limb of the internal capsule; ALIC, anterior limb of the internal capsule.

H emiplegia is the most feared and frequent motor sequela associated with neonatal cerebral infarction. The majority of studies on perinatal focal infarction report a very high incidence of ensuing hemiplegia,1–5 but in more recent years it has become obvious that not all the infants with such lesions have an abnormal motor outcome (see refs. 6–10 and, for review, ref. 11). These differences almost certainly reflect the different populations studied. Although earlier studies mainly included infants with severe neonatal clinical presentation, recent studies have also included infants who had serial cranial ultrasound and/or brain magnetic resonance imaging (MRI) following the onset of seizures in the first days following birth and were found to have an infarction. Our experience, which is largely obtained from infants presenting with seizures, is that only 20% of full-term infants with infarction develop a hemiplegia.11

Most of the studies published thus far, however, including ours, focused on the presence of hemiplegia and the length of follow-up has been relatively short.1–8 We now report the neuromotor follow-up at early school age of 22 children who were found to have cerebral infarction on neonatal MRI. The aims of this study were 1) to evaluate the spectrum of neuromotor difficulties and 2) to correlate the type and extent of motor impairment, when present, with the type, size, and side of infarction on neonatal brain MRI. We have also compared the motor outcome in these children, as assessed at 2–3 years, with that obtained in this study.

SUBJECTS AND METHODS

Ethical permission for this study was obtained from the Hammersmith Hospital (London, United Kingdom) Research Ethics Committee. The children described in this study are part of a large prospective cohort of term infants born at or referred to the Hammersmith Hospital for MRI between 1991 and 1996. As part of this study, all the infants who present with birth asphyxia and/or neonatal seizures undergo neonatal brain MRI.

Twenty-four full-term infants who showed evidence of cerebral infarction on neonatal MRI were enrolled. All the infants were investigated following the onset of convulsions between days 1 and 3. In all, the lesions were obvious on both cranial ultrasound and brain MRI by the end of the first week of life. As part of this prospective study, all patients are followed regularly at 6-month intervals until the age of 3 and at yearly intervals after that irrespective of whether they show any sequela on short-term follow-up. The results of the short-term follow-up of these children have been reported already.8 We were not able to personally assess 2 of the 24 children at school age, because they moved outside the United Kingdom; however, from clinical letters we know that they do not have hemiplegia. The remaining 22 have been followed until primary school age. They all were tested between 5.6 and 6.6 years, with the exception of one child who was tested at 9 years.

MRI

The infants were imaged on a 1.0-T Picker HPQ system using conventional T1-weighted spin-echo (860/20 milliseconds) inversion recovery (3800/30/950 milliseconds) and T2-weighted spin-echo (3000/120 milliseconds) sequences. Two different classifica-
tions were used, according to 1) the arterial distribution of the lesions and 2) the degree of involvement of cortical and subcortical structures.

**Classification According to Arterial Territory**

Based on the location, extent, and shape of the lesions, infarcts were characterized as being in the territory of the main arteries or in a border-zone distribution, i.e., in the watershed area between the end fields of main arterial territories. Border-zone lesions are usually multiple and bilateral, involving the posterior convexities and, less frequently, the anterior lobes.

The infarcts in the territory of the main arteries were first classified according to the main artery involved. The infarcts in the territory of the middle cerebral artery (MCA) were subdivided further according to a modified version of the criteria suggested by de Vries et al.\(^1\) into main branch, cortical branches, and lenticulostriate branches.

In addition to these 3 categories, we classified separately the infarcts in the territory of the MCA that were associated with contralateral lesions.

**Classification Based on the Type of Cerebral Structures Involved**

This classification was based on the site and concomitant involvement of hemispheric and subcortical structures. The lesions were classified according to the possible combination of the involvement of the hemispheres and/or subcortical structures as internal capsule, basal ganglia, thalamus, and brainstem.

**Follow-Up Assessment**

In the early years, global neurodevelopment was assessed by using a structured neurologic examination for infants\(^1\) and the Griffiths neurodevelopmental scales.\(^2\) Details of the early assessments have been published already.\(^3\)

At school age the children’s neurologic status, motor competence, and cognitive ability were assessed as described below.

**Neurologic Status**

The Touwen’s Examination of the Child with Minor Neurologic Dysfunction\(^5\) was administered by two of the examiners (L.D. and E.M.). The examination evaluates performance in 9 areas: sensorimotor apparatus, posture, balance of the trunk, coordination of the extremities, fine manipulative ability, (dys)kinesia, gross motor function, quality of motility, and associated movements. Special attention was paid to asymmetry of posture, tone, power, and movements. Hemiplegia, when present, was graded according to the degree of involvement of upper and lower limbs. Children who only had a mild asymmetry of tone but no clear hypertonia or loss of function or who had minimal contractures on one side but with a normal gait were reported as “asymmetrical.”

**Motor Competence**

The Movement Assessment Battery for Children (Mov ABC)\(^7\) was used to assess the children’s performance on a range of functional tasks. The 3 manual-dexterity items, 2 ball-skill items, and 3 balance items were scored according to the manual. The total scores were expressed as percentiles based on age-specific normative data. Scores ≤50th percentile were considered to be indicative of a motor problem.

**RESULTS**

Of the 22 children assessed at school age, all but 3 had normal cognitive development (intelligence quotient >80) on the Revised Wechsler Preschool and Primary Scale of Intelligence.\(^1\) These were the only 3 children who also needed remedial teaching. None of them have developed epilepsy thus far.

**MRI**

Details of the extent of the lesions on MRI are given in Table 1.

**Arterial Territory: Cerebral Artery Infarcts**

In 18 of the 22 infants, the infarcts were in the territory of the MCA.

Three of these 18 children had changes in the territory of the main branch, 13 in one of the cortical branches, and 2 in the territory of the lenticulostriate branch.

Two of the 3 children with main branch distribution also had small ischemic lesions in the contralateral hemisphere and 1 had a small lesion in the contralateral posterior limb of the internal capsule (PLIC). Two of the 13 children with a cortical branch infarction also showed small lesions in the contralateral white matter.

**Arterial Territory: Border-Zone Lesions**

Four children showed changes in the watershed areas that were consistent with border-zone lesions.

**Hemispheric and Subcortical Involvement**

Eight of the 22 children had hemispheric involvement only with sparing of the internal capsule and basal ganglia. Another 5 had hemispheric lesions and involvement of either the PLIC or basal ganglia, whereas a further 2 had normal hemispheres but involvement of basal ganglia and internal capsule. In these 2 children, however, the internal capsule involvement was in the anterior limb (ALIC) rather than the PLIC. The remaining 7 children had involvement of hemispheres, basal ganglia, and PLIC.

**Clinical Examination at School Age**

**Neurologic Examination**

Six children had clear signs of hemiplegia, and a further 4 were found to have some asymmetries. Three of the 4 had a mild asymmetry of tone but no clear hypertonia, and one had minimal contractures on one side but a normal gait. None of the 4 had clear asymmetry of function, sensory abnormalities, or neglect. The remaining 12 had a completely normal and symmetrical examination. Table 1 shows details of the neurologic findings.

**Mov ABC**

Twenty-one of the 22 children were within age band 1 (4–6 years old) and 1 was in age band 3 (9–10 years old). Seventeen (67%) had total scores >50th percentile. Five other children (23%), 3 with hemiplegia and 2 with normal neurologic examinations, had total scores <50th percentile. Details are provided in Table 1.

**Motor Outcome and MRI: Arterial Distribution**

**Main Cerebral Artery Infarcts**

Tables 1 and 2 show details of the correlation between arterial distribution and neuromotor findings.

Three children had an infarction of the main branch of the MCA. All 3 had hemiplegia, and 2 of 3 had poor scores on the Mov ABC.

Thirteen children had unilateral infarction in the territory of a cortical branch of the MCA. Two of the 13 developed hemiplegia, 2 showed some asymmetry in tone on neurologic examination, 2 had a nor-
| No. | Age  | Infarct | Site       | Basal Ganglia/Thalami | Internal Capsule | Neurological Examination | Mov. ABC  
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12 d</td>
<td>Lenticulostriate b</td>
<td>Normal</td>
<td>L lentiform/caudate</td>
<td>L decreased SI ALIC</td>
<td>Asymmetry</td>
<td>Normal (45)</td>
</tr>
<tr>
<td>2</td>
<td>10 d</td>
<td>Lenticulostriate b</td>
<td>Normal</td>
<td>L lentiform/caudate</td>
<td>L decreased SI ALIC</td>
<td>Normal</td>
<td>Abnormal (3)</td>
</tr>
<tr>
<td>3</td>
<td>10 d</td>
<td>Cortical b</td>
<td>R F,P</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (75)</td>
</tr>
<tr>
<td>4</td>
<td>8 d</td>
<td>Cortical b</td>
<td>L F,P</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (65)</td>
</tr>
<tr>
<td>5</td>
<td>3 d</td>
<td>Cortical b</td>
<td>L P</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (60)</td>
</tr>
<tr>
<td>6</td>
<td>6 d</td>
<td>Cortical b</td>
<td>L P,T,O</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (93)</td>
</tr>
<tr>
<td>7</td>
<td>5 d</td>
<td>Cortical b</td>
<td>L P,T,O</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (62)</td>
</tr>
<tr>
<td>8</td>
<td>4 d</td>
<td>Cortical b</td>
<td>L P,O</td>
<td>L lentiform</td>
<td>Normal</td>
<td>Asymmetry</td>
<td>Normal (79)</td>
</tr>
<tr>
<td>9</td>
<td>4 d</td>
<td>Cortical b</td>
<td>L F,P</td>
<td>L lentiform/caudate</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (72)</td>
</tr>
<tr>
<td>10</td>
<td>7 d</td>
<td>Cortical b/contralateral</td>
<td>L P,T</td>
<td>L lentiform R thalamus</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (72)</td>
</tr>
<tr>
<td>11</td>
<td>6 d</td>
<td>Cortical b</td>
<td>L P</td>
<td>Normal</td>
<td>L decreased SI PLIC</td>
<td>Asymmetry</td>
<td>Normal (16)</td>
</tr>
<tr>
<td>12</td>
<td>6 d</td>
<td>Cortical b</td>
<td>L F,P</td>
<td>Normal</td>
<td>L decreased SI PLIC</td>
<td>Normal</td>
<td>Normal (4)</td>
</tr>
<tr>
<td>13</td>
<td>4 d</td>
<td>Cortical b</td>
<td>L P,T,O</td>
<td>L lentiform/thalamus</td>
<td>L decreased SI PLIC</td>
<td>Normal</td>
<td>Normal (1)</td>
</tr>
<tr>
<td>14</td>
<td>4 d</td>
<td>Cortical b</td>
<td>L P,O</td>
<td>L lentiform/caudate</td>
<td>L loss of SI PLIC</td>
<td>R hemi (Mod*)</td>
<td>Normal (18)</td>
</tr>
<tr>
<td>15</td>
<td>10 d</td>
<td>Cortical b/contralateral</td>
<td>R P,O; L O</td>
<td>R thalamus</td>
<td>R decreased SI PLIC</td>
<td>L hemi (Mod*)</td>
<td>Abnormal (2)</td>
</tr>
<tr>
<td>16</td>
<td>7 d</td>
<td>Main b/contralateral</td>
<td>L F,P,T,O</td>
<td>L lentiform</td>
<td>L decreased SI PLIC</td>
<td>R hemi (Mod*)</td>
<td>Normal (6)</td>
</tr>
<tr>
<td>17</td>
<td>5 d</td>
<td>Main b/contralateral</td>
<td>R F,P,T,O</td>
<td>R lentiform/thalamus</td>
<td>R decreased SI PLIC</td>
<td>L hemi (Mod*)</td>
<td>Abnormal (3)</td>
</tr>
<tr>
<td>18</td>
<td>5 d</td>
<td>Main b/contralateral</td>
<td>L F,P,T,O</td>
<td>L lentiform/thalamus</td>
<td>Bilat. decreased SI PLIC</td>
<td>L hemi (Mild*)</td>
<td>Abnormal (3)</td>
</tr>
<tr>
<td>19</td>
<td>9 d</td>
<td>Border zone</td>
<td>Bilat P,O</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (50)</td>
</tr>
<tr>
<td>20</td>
<td>6 d</td>
<td>Border zone</td>
<td>L F,P,T,O; R O</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal (18)</td>
</tr>
<tr>
<td>21</td>
<td>5 d</td>
<td>Border zone</td>
<td>Bilat P,O</td>
<td>Normal</td>
<td>Normal</td>
<td>Asymmetry</td>
<td>Normal (28)</td>
</tr>
<tr>
<td>22</td>
<td>6 d</td>
<td>Border zone</td>
<td>Bilat P,O (L&gt;R)</td>
<td>L lentiform R lentiform</td>
<td>Bilat decreased SI PLIC</td>
<td>R hemi (Mild*)</td>
<td>Abnormal (3)</td>
</tr>
</tbody>
</table>

b indicates branch; Bilat, bilateral; SI, signal intensity; F, frontal; P, parietal; T, temporal; O, occipital; L, left; R, right; Mod, moderate.

* Classification according to Claey et al.26
mal neurologic examination but poor scores on the Mov ABC, and 7 had a normal motor outcome. Two children had lenticulostriate infarction. Neither had hemiplegia, but both showed other signs of motor impairment; one had a mild asymmetry, and the other had poor scores on the Mov ABC.

**Borderzone Lesions**

One of the 4 infants with border-zone lesions had a hemiplegia and poor scores on the Mov ABC, and 3 had a normal motor outcome.

**Hemispheric and Subcortical Involvement**

Table 3 shows details of the correlation between the site of lesions and outcome.

In 7 children there was involvement of hemispheres, basal ganglia, and internal capsule (more specifically, of the PLIC): only 1 of the 7 had a normal neurologic examination but had poor scores on the Mov ABC, and the other 6 all had a hemiplegia. In 1 of the 7 there was a concomitant involvement of the 3 structures but also a small lesion in the contralateral PLIC. This child has hemiplegia, but it was contralateral to the small internal capsule lesion.

Four children had involvement of internal capsule (2 in the ALIC and 2 in the PLIC) and either the hemisphere or basal ganglia: none had a hemiplegia, but 3 of the 4 were asymmetrical and 1 had poor scores on the Mov ABC.

Eleven children had hemispheric lesions with or without basal ganglia involvement but sparing of the internal capsule: 9 of the 11 had a normal motor outcome and 2 were asymmetrical.

Details of this correlation are shown in Table 3.

**Early and Late Motor Follow-Up**

Tables 2 and 3 also show details of the comparison between the assessment performed at school age and the results of the previously published short-term follow-up.\(^{11}\)

None of the children who were found to have hemiplegia at school age were considered as normal on short-term follow-up: 5 of the 6 already had clear signs of hemiplegia, and 1 was classified as asymmetrical.

Of the children without hemiplegia, the 3 with poor scores on the Mov ABC and the 3 with asymmetries at school age were all classified as normal on short-term follow-up.

**DISCUSSION**

The results of this study show that, when examined at school age, <30% of the children in our cohort who were born full-term and had neonatal infarction on brain MRI showed clear signs of hemiplegia. The aim of this study, however, was primarily to assess whether children who do not develop hemiplegia and are regarded as normal on short-term follow-up may still show minor signs of motor impairment at an older age. Using a structured neurologic examination and a battery of tests specifically designed to assess motor function in children at school age, we found that a further 7 children (28%) with neonatal infarction showed either asymmetry on the neurologic examination \((n = 4)\) or had poor scores on the test assessing neuromotor function \((n = 3)\).

All the children in the study have been followed longitudinally since birth, and the short-term outcome has been published already. In that study we focused on motor outcome between 18 and 36 months, because only 3 of the 24 children included were >3 years old. When we compared the neuromotor findings at school age with the findings of the shorter-term follow-up, we found that with one exception, hemiplegia, when present, had been diag-

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**TABLE 2.** Motor Outcome at School Age in Infants Subdivided According to the Arterial Distribution: Correlation With Previously Published Short-Term Follow-up in the Same Patients\(^a\)

<table>
<thead>
<tr>
<th>Arterial</th>
<th>School Age</th>
<th>Short-Term Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lenticulostriate branch</td>
<td>Ø</td>
<td></td>
</tr>
<tr>
<td>Cortical branch</td>
<td></td>
<td>Ø</td>
</tr>
<tr>
<td>Cortical branch with contralateral changes</td>
<td>Ø</td>
<td>Ø</td>
</tr>
<tr>
<td>Main branch with contralateral changes</td>
<td>-Ø</td>
<td>-Ø</td>
</tr>
<tr>
<td>Border zone</td>
<td>-Ø</td>
<td>-Ø</td>
</tr>
</tbody>
</table>

Ø indicates normal; Ø, poor results on MovABC, no hemiplegia; Ø, asymmetrical; Ø, hemiplegia; –, not tested.

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**TABLE 3.** Motor Outcome at School Age in Infants Subdivided According to the Involvement of Cortical and Subcortical Structures: Correlation With Previously Published Short-Term Follow-up in the Same Patients\(^8\)

<table>
<thead>
<tr>
<th>Involvement</th>
<th>School Age</th>
<th>Short-Term Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemispheric only</td>
<td>Ø</td>
<td>Ø</td>
</tr>
<tr>
<td>Hemispheric and BG</td>
<td>ØØ</td>
<td>ØØ</td>
</tr>
<tr>
<td>Hemispheric and PLIC</td>
<td>-Ø</td>
<td>-Ø</td>
</tr>
<tr>
<td>BG and ALIC</td>
<td>ØØ</td>
<td>ØØ</td>
</tr>
<tr>
<td>Hemispheric, PLIC and BG</td>
<td>ØØ</td>
<td>ØØ</td>
</tr>
</tbody>
</table>

Ø indicates normal; Ø, poor results on MovABC, no hemiplegia; Ø, asymmetrical; Ø, hemiplegia; BG, basal ganglia; –, not tested.
nosed already on the early assessment. The remaining patient who was found to have hemiplegia at school age had already showed some asymmetries on neurologic examination at 2 years of age. It is of interest that this is also the case with a predominant lesion in the main branch of the MCA but also a discrete lesion in the contralateral PLIC. In this child the hemiplegia is ipsilateral to the main lesion and contralateral to the discrete internal capsule involvement.

None of the children who were diagnosed as normal before the age of 3 developed hemiplegia, but a proportion showed some minor neuromotor difficulties or asymmetries at school age. The difference between the early and late findings may reflect the fact that, at school age, a more detailed neuromotor assessment including evaluation of diadochokinesia and repetitive independent finger movements can be performed. It may also be caused by the late onset of minor abnormalities such as mild joint contractures, which can cause asymmetry of posture or walking.

The 3 children (15%) with no asymmetry but with poor scores on the tests assessing motor function and coordination (Mov ABC) had either mild mental retardation or an intelligence quotient just >5th percentile and were described as “clumsy” by parents and teachers. Two of these children were also slightly dysmorphic, but extensive investigations for genetic and metabolic syndromes were all negative. Although mental retardation has been reported previously in children with hemiplegia who were found to have an extensive neonatal cerebral infarction, there are no previous descriptions of this pattern of abnormal motor coordination with mild mental retardation not associated with hemiplegia. de Vries et al12 reported 3 cases with periventricular infarction with global delay, but all 3 cases were born preterm (28–31 weeks’ gestation) and had other risk factors.

We also wished to examine whether motor abnormalities, if present, were related to a specific pattern of lesions on neonatal brain MRI. In our previous study we found that infants with a combination of hemispheric, basal ganglia, and internal capsule involvement were at high risk of developing hemiplegia but that the involvement of the internal capsule did not predict hemiplegia per se. The results of our current study at school age confirm our earlier observation. It is of interest, however, that all the children who had internal capsule involvement and did not develop hemiplegia had minor motor abnormalities at school age, whereas only 2 of the 11 children without internal capsule involvement had abnormal motor signs. These results are in keeping with the observation that the internal capsule plays an important role in the prognosis of motor outcome in children with perinatal lesions.19–21 When we looked at the part of the internal capsule involved, we found that, in children with hemiplegia, it was always the PLIC that was involved, whereas in children with minor motor signs it could be either the PLIC or ALIC. This suggests that although minor motor signs may stem from different parts of the brain, hemiplegia will only result when there is specific involve-ment of that part of the internal capsule that carries the primary motor and sensory tracts.

Another study has reported the association of hemiplegia and unilateral internal capsule involvement in prematurely born children with periventricular hemorrhagic infarction.20 The comparison with that study, however, is difficult because both the type of lesions and the time when the scans were performed are different. In our full-term infants the most common lesion was an arteriole infarct in the territory of the main branch or in one of the cortical branches of the MCA, whereas in the study on preterm infants the lesions were mainly periventricular hemorrhagic infarctions. We and others have previously reported that, in symptomatic full-term infants, arterial infarction is of perinatal onset, as demonstrated by using early and serial magnetic resonance images, including diffusion-weighted imaging.22 In the present study, all the full-term infants had MRI scans within the first week, and the signal abnormalities in the internal capsule were already obvious on these early scans, suggesting that the internal capsule was primarily involved. In contrast, in the preterm infants in the de Vries et al study, the scans were performed many weeks following the insult at term-age equivalent, and in most cases it is likely that the internal capsule involvement was the consequence of the hemispheric or basal ganglia lesion rather than being involved primarily as part of the lesion.

CONCLUSIONS

Our results suggest that although hemiplegia occurs in <30% of the children with neonatal infarction, a similar percentage of children will show minor signs of neuromotor impairment at early school age, which could affect their everyday performance. Our findings suggest that the involvement of the internal capsule on neonatal MRI is a good predictor for motor abnormalities. In a proportion of infants with abnormal internal capsule, however, the short-term follow-up may be normal because more minor motor abnormalities may only become obvious at school age when a more detailed assessment can be performed. Further studies are in progress to assess other aspects of neurodevelopment such as language, memory, or behavior, which have been found to be affected in children with cerebral infarction.23,24

ACKNOWLEDGMENT

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**KEEP INFANTS AWAY FROM MIRRORS!**

“The fear of souls being captured in mirrors was widespread in antiquity . . . Similar beliefs explained why a sick person should avoid looking into a mirror . . . infant souls were particularly susceptible to harm, so folklore warned parents to keep children under a year of age away from mirrors.”

Pendergrast M. *Mirror Mirror*. Basic Books; 2003

Submitted by Student