

# Magnetic Resonance Image Correlates of Hemiparesis After Neonatal and Childhood Middle Cerebral Artery Stroke

James P. Boardman, MSc, MRCPCH\*; Vijeya Ganesan, MD, MRCP‡; Mary A. Rutherford, FRCPCH, FRCR\*; Dawn E. Saunders, MD, FRCR§; Eugenio Mercuri, MD¶||; and Frances Cowan, PhD, MRCPCH||

**ABSTRACT.** *Objective.* Motor impairment after neonatal and childhood-onset ischemic stroke (IS) is common, although the prevalence and type of hemiparesis differs between the 2 age groups. Lesion topography is an important predictor of hemiparesis after neonatal IS, but it is not known if the same topographic predictors of adverse motor outcome apply to childhood-onset IS. We used a consistent approach to define lesion topography and evaluate motor outcome in both age groups to (1) investigate whether early topographic predictors of hemiparesis after unilateral middle cerebral artery–territory stroke are the same in neonates and older children and (2) compare the prevalence of dystonia and loss of independent finger movements between the 2 age groups.

*Design.* Twenty-eight patients with neonatal-onset IS (Hammersmith Hospital, London, United Kingdom) were studied together with 43 patients with childhood-onset IS (Great Ormond Street Hospital, London, United Kingdom). All patients had exclusive unilateral middle cerebral artery–territory IS. Lesion topography was studied by using the first magnetic resonance image acquired after the onset of symptoms and was coded for involvement of cerebral cortex (CC), posterior limb of the internal capsule (PLIC), basal ganglia (BG), and white matter. The primary outcome was hemiparesis, and secondary outcomes were dystonia and loss of age-appropriate independent finger movements.

*Results.* Hemiparesis was more common after childhood-onset IS (56%) than neonatal-onset IS (24%). In neonatal-onset IS, concomitant involvement of BG, CC, and PLIC predicts the development of hemiparesis (odds ratio: 99; 95% confidence interval: 5.2–1883.8), and no child with 1 or 2 of these structures involved developed hemiparesis. In contrast, in childhood-onset IS, concomitant BG, CC, and PLIC lesions tended to be associated with hemiparesis (9 of 11), but this adverse outcome was

seen also among patients with 1- or 2-site involvement. However, hemiparesis was less likely if the infarction involved BG only (odds ratio: 0.162; 95% confidence interval: 0.036–0.729). Dystonia was present in 15 of 24 in the childhood-onset group with hemiparesis but was not seen after neonatal-onset IS. In both age groups upper-limb impairment was more severe than lower-limb impairment, with frequent loss of independent hand function among hemiparetic patients.

*Conclusions.* In neonatal and childhood-onset IS, early magnetic resonance imaging provides useful prognostic information about subsequent motor outcome. There are differences in the functional response of the neuromotor system to injury between the 2 age groups that cannot be attributed to methodological differences alone. *Pediatrics* 2005;115:321–326; *ischemic stroke, magnetic resonance imaging, middle cerebral artery, childhood, neonatal.*

**ABBREVIATIONS.** IS, ischemic stroke; MCA, middle cerebral artery; BG, basal ganglia; PLIC, posterior limb of the internal capsule; MR, magnetic resonance; TE, echo time; TR, repetition time; WM, white matter; CC, cerebral cortex; OR, odds ratio; CI, confidence interval.

Neonatal ischemic stroke (IS) (cerebral infarction occurring within 28 days of birth) is recognized in ~1 in 4000 live births per year, with term-born infants more commonly affected than preterm infants.<sup>1</sup> Childhood-onset IS (28 days to 18 years) is as common as pediatric brain tumor and is 1 of the top 10 causes of childhood mortality.<sup>2</sup> In both age groups, infarction occurs most frequently within the middle cerebral artery (MCA) territory,<sup>3–5</sup> and its most common adverse consequence is motor impairment. There are striking differences in the prevalence of hemiparesis after neonatal and childhood IS: two thirds of those who survive childhood-onset stroke are affected,<sup>6–9</sup> compared with approximately one third of children born at term who have neonatal-onset IS, although the percentage varies with different studies, populations, and extensiveness of investigation.<sup>1,4,10–16</sup> There are also differences in the type of motor impairment between the 2 age groups: dystonia and loss of isolated finger movements on the paretic side frequently compound spastic hemiparesis after childhood-onset IS<sup>9,17–19</sup> but have not been reported after neonatal-onset IS.

Early prediction of motor outcome would enable clinicians to provide prognostic information, as well as to identify patients appropriately for possible

From the \*Robert Steiner MR Unit, Imaging Sciences Department, MRC Clinical Sciences Centre, and ||Department of Paediatrics, Faculty of Medicine, Imperial College London, Hammersmith Hospital Campus, London, United Kingdom; ‡Neurosciences Unit, Institute of Child Health, London, United Kingdom; §Department of Radiology, Great Ormond Street Hospital for Children, NHS Trust, London, United Kingdom; and ¶||Department of Child Neurology and Psychiatry, Catholic University, Rome, Italy.

Accepted for publication Jul 6, 2004.

doi:10.1542/peds.2004-0427

No conflict of interest declared.

Address correspondence to James P. Boardman, MSc, MRCPCH, Robert Steiner MR Unit, Imaging Sciences Department, MRC Clinical Sciences Centre, Imperial College London, Hammersmith Hospital Campus, DuCane Road, London W12 0HS, United Kingdom. E-mail: j.boardman@imperial.ac.uk

PEDIATRICS (ISSN 0031 4005). Copyright © 2005 by the American Academy of Pediatrics.

early intervention and rehabilitation. The De Vries et al<sup>16</sup> study of neonatal IS showed that infants with main-branch MCA infarction were more likely to develop spastic hemiparesis than those with distal-branch infarction. In a study of 24 neonates with IS we found that concomitant involvement of basal ganglia (BG), cerebral hemispheric tissue, and the posterior limb of the internal capsule (PLIC) was associated with hemiparesis, whereas involvement of only 1 or any 2 of these locations was associated with a good motor outcome.<sup>13</sup> In a study of 38 patients with childhood-onset MCA-territory infarcts, lesions involving >10% of the intracranial volume and bilateral lesions were associated with poor outcome. However, some children with small BG lesions had a similarly poor motor outcome.<sup>20</sup>

From neonatal studies it is clear that lesion topography is crucial for predicting motor impairment. The system used by Mercuri et al<sup>13</sup> to categorize lesion topography is simple to apply in a clinical setting and, if shown to have prognostic value in older children, is likely to prove useful to clinicians. The aims of this study were to (1) use a consistent approach to the evaluation of infarct topography and motor outcome to investigate whether early topographic predictors of hemiparesis after unilateral MCA-territory IS are the same in neonates and older children and (2) compare the prevalence of dystonia and loss of independent finger movements between the 2 age groups.

## METHODS

### Patients

The neonatal group consisted of 28 term neonates who were born at or referred to the Hammersmith Hospital (London, United Kingdom) in 1994–2001 (Table 1), had seizures between days 1 and 4 after birth, and had evidence of a recent unilateral MCA-territory infarction as evidenced by conventional T1- and T2-weighted magnetic resonance imaging (MRI) and on diffusion-weighted imaging in 24 of the 28 infants for whom this form of imaging was acquired. None of the infants died, and none had a known risk factor for neonatal stroke such as congenital heart disease, twin-to-twin transfusion syndrome, systemic illness or infection, extracorporeal membrane oxygenation, or intravascular cannula before the onset of their symptoms. Four infants were subsequently found to have a prothrombotic factor.<sup>21</sup> None of the infants have had a recurrence of IS. The study forms part of an ongoing longitudinal prospective project aimed at documenting the evolution of neonatal cerebral infarcts and has approval from the Hammersmith Hospitals Trust Research Ethics Committee.

The older group consisted of 43 children (4 weeks to 18 years old) seen at Great Ormond Street Hospital (London, United Kingdom) between 1991 and 2002 with exclusive unilateral MCA-territory IS evident on MRI. Risk factors for IS were identified in 18 children: 8 had recent chicken pox infection; 6 occurred in the

perioperative period; 2 had a febrile illness; and 2 had a prothrombotic disorder. All images were acquired as part of clinical evaluation. Patients who died in the acute period, those with bilateral infarcts, those with recurrent IS, and those with moyamoya syndrome or sickle cell disease were excluded due to potential confounding effects.

### MRI

The patients in the neonatal-onset IS group were imaged on a 1.0-T Picker system (Cleveland, OH) using conventional T1-weighted spin echo (SE 860/20), inversion recovery (IR 3800/30/950), and T2-weighted spin echo (SE 3000/120) sequences. After 1999, images were acquired on a 1.5-T Eclipse system (Philips Medical Systems, Cleveland, OH) and comprised conventional T1-weighted spin echo (SE 500/15) and T2-weighted fast spin echo (SE 4500/210) at 5-mm slice thickness. The infants were usually sedated for imaging with oral chloral hydrate (20–30 mg/kg), and pulse oximetry and electrocardiograph were monitored throughout the procedure.

The patients in the childhood-onset IS group were imaged on a 1.5-T Magnetom SP4000 (Siemens, Erlangen, Germany). Turbospin echo T2-weighted images (echo time [TE]: 90 milliseconds; repetition time [TR]: 4600 milliseconds), fluid-attenuated inversion recovery images (TE: 120 milliseconds; TR: 10 000 milliseconds; inversion time: 2500 milliseconds), and T1-weighted spin echo (TE: 15 milliseconds; TR: 550 milliseconds) were acquired at 5-mm slice thickness and a 2.5-mm slice gap in the >2-year-olds. Double echo short  $\tau$  inversion recovery (DESTIR) (TE: 5 milliseconds; TR: 3500 milliseconds; inversion time: 145 milliseconds) and T1-weighted spin echo (TE: 15 milliseconds; TR: 550 milliseconds) images were acquired in the <2-year-old group. A three-fourths field of view was used with matrix sizes of 135 × 236 for the DESTIR and 192 × 256 for the turbospin echo images.

### Image Analysis and Classification of Infarct Location

The first magnetic resonance (MR) image acquired after IS was used for analysis of lesion topography. Infarction was defined as tissue with abnormal high signal intensity on T2-weighted images with or without loss of gray matter/white matter (WM) differentiation on T1- and T2-weighted images. Infarct location was classified to include structures in the motor tract within the distribution of the MCA territory: BG, cerebral hemispheric tissue, and PLIC. In our original neonatal study we used the term “hemisphere” to indicate involvement of WM and cortical tissue. In this study we separated the cerebral hemispheric tissue into cerebral cortex (CC) and WM. Involvement of any part of the structure was coded as positive.

### Neuromotor Outcome

Outcome data were obtained as part of clinical evaluation, because all subjects are undergoing long-term neurodevelopmental follow-up (Table 2). The primary motor outcome was hemiparesis, defined as unilateral motor impairment with a severity greater than at least 1 of: reflex asymmetry or reduced isolated finger movements or abnormal posturing. Secondary motor outcomes were (1) dystonia and (2) complete loss of independent finger movements leading to no age-appropriate independent hand use. All infants were assessed by 1 of the authors (F.C.: neonatal group; V.G.: childhood group).

### Statistical Analysis

Logistic regression analyses were used to explore the relations between infarct site(s) and hemiparesis. The following were examined as covariates in these analyses: length of follow-up, because motor impairment may change with time; length of time from IS to MRI, because the MR signal from infarcted tissue

**TABLE 1.** Study Groups

|   | Neonatal Group  | Childhood Group  |
|---|-----------------|------------------|
| Number  | 28 (18 male)    | 43 (28 male)     |
| Median age at time of IS (range)              | 2 d (1–4)       | 3.6 y (0.3–15.5) |
| Median length of time from IS to MRI (range)* | 4.5 d (0–35)    | 2.0 d (0–268)    |
| Median length of follow-up (range)†           | 5.5 y (1.7–5.5) | 2.5 y (0.3–8.5)  |

\* No significant difference ( $P = .06$ ).

† ( $P = .002$ ).

**TABLE 2.** Motor Outcome After Neonatal and Childhood IS

|                                      | Neonatal Group | Childhood Group |
|--------------------------------------|----------------|-----------------|
| Hemiparesis                          | 7 (24%)        | 24 (56%)        |
| Dystonia                             | 0 (0%)         | 15 (39%)        |
| Spasticity                           | 7 (24%)        | 24 (56%)        |
| Loss of independent finger movements | 5 (17%)        | 15 (39%)        |

changes with time; and in the childhood group, age at time of IS was examined, because this has been suggested as a prognostic indicator in previous studies.<sup>8,22</sup>

## RESULTS

### Patient Characteristics and Motor Outcome

In both groups there was a male predominance of ~1.5 to 1, which is consistent with epidemiologic studies of neonatal and childhood stroke.<sup>23</sup> The neonatal group comprised term-born infants (median gestational age: 40.3 weeks; range: 38.0–42.3 weeks). The time of onset of seizures was taken to indicate the time of presentation of IS. In the childhood group, 42 patients presented with acute hemiparesis (with seizures in 3 cases), and 1 presented with an acute behavioral disturbance without motor symptoms.

Although there is a significant difference in the length of follow-up between the 2 groups, this did not significantly influence the relationship between lesion site and hemiparesis in either group.

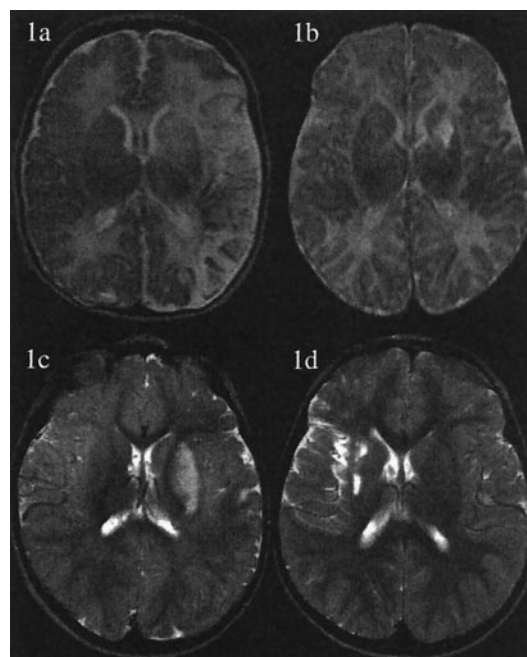
### Sites of Infarction

In the neonatal-onset group, 8 infants had IS affecting 3 sites (BG, CC, and PLIC), 5 infants had 2 sites affected, and 15 infants had 1 site affected. In the childhood-onset group, 11 had IS in 3 sites, 17 children had IS in 2 sites only, and 15 had IS in 1 site only. Some WM abnormality was seen commonly in both groups and was not discriminatory in isolation (see below). Three-site involvement occurred in 29% of neonates and 26% of children. BG involvement in isolation or with 1 other site occurred in 70% of the children but only 18% of neonates. In contrast, lesions involving CC without other sites were far more common in neonatal IS than childhood IS.

Seventy-five percent of neonatal-onset infarcts occurred in the left MCA territory (21 of 28), but the preponderance for left-sided infarction was not seen in the childhood-onset group (17 of 43 left-sided and 26 of 43 right-sided). There was no relationship between side of lesion(s) and hemiparesis in either age group (or dystonia in the childhood-onset group).

### Hemiparesis and Its Relationship With Infarct Location

Hemiparesis was more common after childhood IS (56%) compared with neonatal IS (24%; Table 3). In both age groups MCA IS tended to result in multiple areas of infarction within the arterial territory: 17 of 28 neonates and 28 of 43 children had >1 structure affected. The BG were frequently affected, especially



**Fig 1.** Axial T2-weighted images acquired after neonatal-onset (a and b) and childhood-onset (c and d) IS. In a, there is abnormal high signal intensity in left caudate and lentiform nuclei (BG), the PLIC, and the CC, with loss of gray matter/WM differentiation. This child has a hemiparesis, which characteristically occurred when there was concomitant involvement of BG, CC, and PLIC. In b, there is high signal intensity in the lentiform nucleus (BG) and anterior limb of the internal capsule, with no involvement of PLIC or CC. This child has no hemiparesis; no neonate with exclusive BG or CC involvement or BG and PLIC or BG and CC lesions developed hemiparesis. In contrast, c was acquired after childhood-onset IS and shows BG involvement only with sparing of the CC and PLIC, and this child does have a hemiparesis. d shows involvement of PLIC, BG, and CC after childhood-onset IS. Although this pattern of lesion site tended to result in hemiparesis (9 of 11), some children with this distribution, including the example shown here, did not develop hemiparesis.

after childhood IS, when 41 of 43 had involvement of these structures. Because multiple site involvement was common, it was not possible to examine the effect of single sites on outcome, so infarct sites were grouped according to involvement of  $\geq 1$  of BG, CC, and PLIC, and the relationship between these grouped sites and outcome was analyzed (Fig 1).

### Neonatal IS

In the neonatal group, involvement of BG, CC, and PLIC together was significantly associated with hemiparesis (odds ratio [OR]: 99; 95% confidence interval [CI]: 5.2–1883.8;  $P = .002$ ). This was not significantly influenced by age at follow-up (OR = 0.80;  $P = .55$ ). One infant with involvement of all 3 sites does not meet the criteria for hemiparesis used in this study but has reflex asymmetry. No other combination of sites was associated with hemiparesis. WM involvement was present in 26 of 28 neonates and was too common to be a discriminating factor for outcome.

### Childhood IS

Although BG, CC, and PLIC involvement resulted in hemiparesis in 9 of 11 childhood IS cases, hemi-

**TABLE 3.** Grouped Infarct Sites and Hemiparesis After Neonatal and Childhood IS

| Infarct Site(s)    | No. With Hemiparesis/Total Number With Lesion Distribution |           |
|--------------------|--|-----------|
|                    | Neonatal   | Childhood |
| BG and CC and PLIC | 7/8  | 9/11      |
| BG and CC          | 0/2  | 5/6       |
| BG only            | 0/3  | 4/13      |
| PLIC and CC        | 0/3  | 0/0       |
| CC only            | 0/12   | 1/2       |
| BG and PLIC        | 0/0  | 5/11      |
| Total              | 7/28   | 24/43     |



**TABLE 4.** Association Between Infarct Site and Hemiparesis After Childhood IS

| Infarct Location   | Regression Coefficient (b) | SE (b) | OR (Exp B) | 95% CI for OR | P Value |
|--------------------|----------------------------|--------|------------|---------------|---------|
| BG and CC and PLIC | 3.31                       | 1.75   | 27.470     | 0.89–849.38   | .06     |
| BG and CC          | 2.29                       | 1.20   | 9.86       | 0.94–103.95   | .06     |
| BG only            | –1.82                      | 0.77   | 0.162      | 0.036–0.729   | .02     |
| CC only            | –0.70                      | 1.59   | 0.496      | 0.02–11.21    | .66     |
| BG and PLIC        | –0.55                      | 1.01   | 0.58       | 0.08–4.18     | .59     |

paresis could occur after any pattern of infarction (Table 3). However, lesions involving BG only are less likely to result in hemiparesis than other combinations of site involvement ( $P = .02$ ; Table 4). WM was involved in 34 of 43 patients and did not significantly discriminate for motor outcome.

The length of time between IS and MR examination and length of time of follow-up did not significantly alter these relationships in either group, and age at time of IS in the childhood group did not affect outcome.

#### Dystonia and Loss of Independent Finger Movements

Dystonia was frequent after childhood-onset IS (15 of 24 children with hemiparesis) and occurred with each type of lesion distribution observed, but it was not seen among those with neonatal IS. Dystonia was not always associated with loss of independent finger movements in the childhood group ( $P = .08$ , Fisher's exact test). Loss of independent finger movements accompanied hemiparesis in 5 of 7 from the neonatal IS group and 15 of 24 children from the older group.

### DISCUSSION

This study used identical methods of image analysis and outcome evaluation to identify differences in the relationship between lesion topography and motor outcome after neonatal and childhood MCA stroke. By using this approach we reduced the influence of methodological differences in comparing the relationship between lesion topography and outcome between the 2 age groups. In neonatal unilateral MCA-territory stroke, concomitant involvement of the BG, CC, and PLIC strongly predicts hemiparesis. This lesion pattern was frequently associated with hemiparesis in childhood-onset IS (9 of 11 patients): although the association did not reach statistical significance, the 95% CIs of the OR are wide, which together with a  $P$  value of .06 suggests that a significant relationship might be detected in a larger study group.

The most striking difference is that some children with lesions in BG only (4 of 13), BG and CC (5 of 6), BG and PLIC (5 of 11), or CC only (1 of 2) developed hemiparesis when 0 of 20 children with lesions in 1 or 2 sites sustained in the neonatal period developed hemiparesis, suggesting that injuries acquired in the neonatal period may have a less deleterious effect on motor function. After early unilateral brain injury, there are abnormal motor projections from the unaffected side ipsilaterally to the paretic side indicating a reorganization of corticomotorneuronal projections.<sup>24–30</sup> The pattern of ipsilateral motor projection is different in subjects with antenatally compared

with postnatally acquired lesions, indicating that the maturational age of the nervous system at the time of insult influences the variability of cortical reorganization.<sup>29</sup> In normal postnatal corticospinal tract development there are similar responses to transcranial magnetic stimulation of ipsilateral and contralateral motor projections for the first 3 months of postnatal life, after which there is withdrawal of ipsilateral projections leading to contralateral dominance by 18 months, and in hemiparetic patients with perinatally acquired lesions, ipsilateral withdrawal is attenuated.<sup>31</sup> It is possible that the capacity for ipsilateral reorganization is maximal before corticomotor axonal withdrawal becomes established at 3 months. This study provides functional evidence for a difference in the effect of injury on sensorimotor pathways that is age-dependent and could be mediated by differences in the capacity for corticomotor tract reorganization.

In both age groups MCA-territory stroke commonly led to infarcts involving multiple structures, with a predisposition to BG injury that is most striking in the childhood group (41 of 43 children), and is consistent with other studies of childhood-onset IS.<sup>18,32</sup> In contrast, only two thirds of the neonatal-onset IS group had BG involvement. Secondary dystonia was frequent in the childhood group, as previously described,<sup>17,18,33,34</sup> but was absent in children who sustained injuries in the neonatal period. Observed differences in lesion topography and prevalence of dystonia could reflect the different etiologies underlying neonatal and childhood IS and/or the variations in tissue maturation, corticomotorneuronal connectivity, and plasticity within BG, which may be age-dependent. Additional characterization of differences in lesion topography within BG between the 2 age groups might help to identify neural correlates for dystonia. We have not identified dystonia after a median follow-up period of 5.5 years, but we continue to monitor for late-onset dystonia, which has been reported in a small selected group of children with neonatal hypoxic-ischemic injury.<sup>35</sup>

In those who developed a spastic hemiparesis, upper-limb function was commonly and severely affected in both groups, with the majority of patients having lost independent finger movements. The lower limb was affected less strikingly, and all children in the study were independently mobile.

The study is limited by difficulties in defining lesion site. We coded any abnormal high signal intensity on T2-weighted imaging as involvement of that structure. This classification scheme does not account for the extent of infarction within a structure; for example, lesions involving the whole of the BG, CC, or PLIC could have a different impact on out-

come than a smaller lesion in the same site. Systems of describing lesion topography based on MCA-branch territories or specific cortical regions could be useful in refining the definition of lesion site,<sup>36</sup> although this study indicates that large study groups would be required to identify significant relationships with outcome. For consistency we chose to compare the 2 groups by using the same tools that had proved useful in our neonatal studies and are simple to use on conventional, clinically available MRI studies. This study does not address the role of lesion volume on outcome. In adult MCA infarction there is a direct relationship between lesion volume on early conventional<sup>37</sup> and diffusion-weighted MRI<sup>38</sup> and neuromotor outcome. This relationship is not present in childhood stroke,<sup>20</sup> and there have been no studies relating lesion volume to outcome after neonatal-onset stroke. Additional studies incorporating diffusion-weighted MRI to help define lesion topography, together with volumetric studies to define lesion extent, are required in both age groups.

Early prediction of poor outcome groups after adult onset IS has helped to define suitable candidates for entry into trials of acute thrombolytic and neuroprotective therapies, which usually require starting treatment within 6 hours of IS (see ref 39 for review). The success of these trials is attributed in part to raised awareness of the potential benefits of early diagnosis and treatment together with service reorganization,<sup>40,41</sup> which have helped reduce the time from onset of symptoms to neuroimaging and intervention. However, the paucity of symptoms heralding the onset of neonatal IS and the frequent delay to diagnosis in childhood-onset IS<sup>42</sup> mean that there are currently difficulties in acquiring early neuroimaging in pediatric patients. With increased awareness of the symptoms of IS and the potential benefits of early detection, particularly among those who care for children with known risk factors for IS, detection could be expedited and these early imaging prognostic indicators used to help in the development of trials of acute intervention treatments in pediatric IS.

## CONCLUSIONS

Infarct topography is an important predictor of hemiparesis in unilateral MCA stroke occurring in the neonatal period. These data confirm our earlier findings that hemiparesis after neonatal stroke is likely with concomitant involvement of BG, PLIC, and CC. In contrast, after childhood unilateral MCA stroke, hemiparesis may occur with 1, 2, or 3 of these structures involved. The BG are affected in the majority of childhood stroke, but involvement of these structures alone is unlikely to result in hemiparesis. Severe upper-limb dysfunction is a frequent component of hemiparesis in both age groups, and secondary dystonia is common in older children but was not seen in those who had an infarction in the perinatal period. The principle followed in this study to predict motor outcome after IS could be applied to other areas of outcome such as visual function,<sup>43</sup> cognition, language development, later seizures, and behavior. Comparative studies of age-dependent

structure-function relationships for other neurodevelopmental outcomes after IS would benefit from larger study groups to detect significant differences, which will necessitate collaborative international studies and continued detailed follow-up of these children.

## ACKNOWLEDGMENTS

We thank the children and families who participated in the study; the radiographic staff involved in image acquisition; nursing and medical staff who supervised scanning; colleagues who made referrals; and the Medical Research Council (United Kingdom) and Action Medical Research for support.

## REFERENCES

- Estan J, Hope P. Unilateral neonatal cerebral infarction in full term infants. *Arch Dis Child Fetal Neonatal Ed.* 1997;76:F88–F93
- Fullerton HJ, Chetkovich DM, Wu YW, Smith WS, Johnston SC. Deaths from stroke in US children, 1979 to 1998. *Neurology.* 2002;59:34–39
- Satoh S, Shirane R, Yoshimoto T. Clinical survey of ischemic cerebrovascular disease in children in a district of Japan. *Stroke.* 1991;22:586–589
- Sran SK, Baumann RJ. Outcome of neonatal strokes. *Am J Dis Child.* 1988;142:1086–1088
- Volpe JJ. *Neurology of the Newborn.* Philadelphia, PA: W. B. Saunders Company; 1995:211–372
- Giroud M, Lemesle M, Gouyon JB, Nivelon JL, Milan C, Dumas R. Cerebrovascular disease in children under 16 years of age in the city of Dijon, France: a study of incidence and clinical features from 1985 to 1993. *J Clin Epidemiol.* 1995;48:1343–1348
- Schoenberg BS, Mellinger JF, Schoenberg DG. Cerebrovascular disease in infants and children: a study of incidence, clinical features, and survival. *Neurology.* 1978;28:763–768
- Hurvitz EA, Beale L, Ried S, Nelson VS. Functional outcome of paediatric stroke survivors. *Pediatr Rehabil.* 1999;3:43–51
- Ganesan V, Hogan A, Shack N, Gordon A, Isaacs E, Kirkham FJ. Outcome after ischaemic stroke in childhood. *Dev Med Child Neurol.* 2000;42:455–461
- Koelfen W, Freund M, Varnholt V. Neonatal stroke involving the middle cerebral artery in term infants: clinical presentation, EEG and imaging studies, and outcome. *Dev Med Child Neurol.* 1995;37:204–212
- Wulfeck BB, Trauner DA, Tallal PA. Neurologic, cognitive, and linguistic features of infants after early stroke. *Pediatr Neurol.* 1991;7:266–269
- Ment LR, Duncan CC, Ehrenkranz RA. Perinatal cerebral infarction. *Ann Neurol.* 1984;16:559–568
- Mercuri E, Rutherford M, Cowan F, et al. Early prognostic indicators of outcome in infants with neonatal cerebral infarction: a clinical, electroencephalogram, and magnetic resonance imaging study. *Pediatrics.* 1999;103:39–46
- Trauner DA, Chase C, Walker P, Wulfeck B. Neurologic profiles of infants and children after perinatal stroke. *Pediatr Neurol.* 1993;9:383–386
- Trauner DA, Mannino FL. Neurodevelopmental outcome after neonatal cerebrovascular accident. *J Pediatr.* 1986;108:459–461
- de Vries LS, Groenendaal F, Eken P, Van Haastert IC, Rademaker KJ, Meiners LC. Infarcts in the vascular distribution of the middle cerebral artery in preterm and fullterm infants. *Neuropediatrics.* 1997;28:88–96
- Dusser A, Goutieres F, Aicardi J. Ischemic strokes in children. *J Child Neurol.* 1986;1(2):131–136
- Jaap Kapelle L, Willemsse J, Ramos LMP, van Gijn J. Ischaemic stroke in the basal ganglia and internal capsule in childhood. *Brain Dev.* 1989;11:283–292
- Chuang C, Fahn S, Frucht SJ. The natural history and treatment of acquired hemidystonia: report of 33 cases and review of the literature. *J Neurol Neurosurg Psychiatry.* 2002;72:59–67
- Ganesan V, Ng V, Chong WK, Kirkham FJ, Connelly A. Lesion volume, lesion location, and outcome after middle cerebral artery territory stroke. *Arch Dis Child.* 1999;81:295–300
- Mercuri E, Cowan F, Gupte G, et al. Prothrombotic disorders and abnormal neurodevelopmental outcome in infants with neonatal cerebral infarction. *Pediatrics.* 2001;107:1400–1404
- deVeber GA, MacGregor D, Curtis R, Mayank S. Neurologic outcome in survivors of childhood arterial ischemic stroke and sinovenous thrombosis. *J Child Neurol.* 2000;15:316–324
- Deveber G. Stroke and the child's brain: an overview of epidemiology, syndromes and risk factors. *Curr Opin Neurol.* 2002;15:133–138

24. Maegaki Y, Maeoka Y, Ishii S, et al. Mechanisms of central motor reorganization in pediatric hemiplegic patients. *Neuropediatrics*. 1997;28:168–174
25. Nezu A, Kimura S, Takeshita S, Tanaka M. Functional recovery in hemiplegic cerebral palsy: ipsilateral electromyographic responses to focal transcranial magnetic stimulation. *Brain Dev*. 1999;21:162–165
26. Maegaki Y, Yamamoto T, Takeshita K. Plasticity of central motor and sensory pathways in a case of unilateral extensive cortical dysplasia: investigation of magnetic resonance imaging, transcranial magnetic stimulation, and short-latency somatosensory evoked potentials. *Neurology*. 1995;45:2255–2261
27. Macdonell RA, Jackson GD, Curatolo JM, et al. Motor cortex localization using functional MRI and transcranial magnetic stimulation. *Neurology*. 1999;53:1462–1467
28. Thickbroom GW, Byrnes ML, Archer SA, Nagarajan L, Mastaglia FL. Differences in sensory and motor cortical organization following brain injury early in life. *Ann Neurol*. 2001;49:320–327
29. Staudt M, Grodd W, Gerloff C, Erb M, Stitz J, Krageloh-Mann I. Two types of ipsilateral reorganization in congenital hemiparesis: a TMS and fMRI study. *Brain*. 2002;125:2222–2237
30. Muller RA, Rothermel RD, Behen ME, Muzik O, Chakraborty PK, Chugani HT. Plasticity of motor organization in children and adults. *Neuroreport*. 1997;8:3103–3108
31. Eyre JA, Taylor JP, Villagra F, Smith M, Miller S. Evidence of activity-dependent withdrawal of corticospinal projections during human development. *Neurology*. 2001;57:1543–1554
32. Brower MC, Rollins N, Roach ES. Basal ganglia and thalamic infarction in children. Cause and clinical features. *Arch Neurol*. 1996;53:1252–1256
33. Demierre B, Rondot P. Dystonia caused by putamino-capsulo-caudate vascular lesions. *J Neurol Neurosurg Psychiatry*. 1983;46:404–409
34. Giroud M, Lemesle M, Madinier G, Manceau E, Osseby GV, Dumas R. Stroke in children under 16 years of age. Clinical and etiological difference with adults. *Acta Neurol Scand*. 1997;96:401–406
35. Saint Hilaire MH, Burke RE, Bressman SB, Brin MF, Fahn S. Delayed-onset dystonia due to perinatal or early childhood asphyxia. *Neurology*. 1991;41(2 pt 1):216–222
36. Govaert P, Matthys E, Zecic A, Roelens F, Oostra A, Vanzielegheem B. Perinatal cortical infarction within middle cerebral artery trunks. *Arch Dis Child Fetal Neonatal Ed*. 2000;82:F59–F63
37. Saunders DE, Clifton AG, Brown MM. Measurement of infarct size using MRI predicts prognosis in middle cerebral artery infarction. *Stroke*. 1995;26:2272–2276
38. Engelter ST, Provenzale JM, Petrella JR, Delong DM, Alberts MJ. Infarct volume on apparent diffusion coefficient maps correlates with length of stay and outcome after middle cerebral artery stroke. *Cerebrovasc Dis*. 2003;15:188–191
39. Lees KR. Management of acute stroke. *Lancet Neurol*. 2002;1:41–50
40. Alberts MJ, Perry A, Dawson DV, Bertels C. Effects of public and professional education on reducing the delay in presentation and referral of stroke patients. *Stroke*. 1992;23:352–356
41. Harbison J, Massey A, Barnett L, Hodge D, Ford GA. Rapid ambulance protocol for acute stroke. *Lancet*. 1999;353:1935
42. Gabis LV, Yangala R, Lenn NJ. Time lag to diagnosis of stroke in children. *Pediatrics*. 2002;110:924–928
43. Mercuri E, Anker S, Guzzetta A, et al. Neonatal cerebral infarction and visual function at school age [published correction appears in *Arch Dis Child Fetal Neonatal Ed*. 2004;89:F187]. *Arch Dis Child Fetal Neonatal Ed*. 2003;88:F487–F491

## WHEN EVERY CHILD IS INCREDIBLE

“‘Winners can be victims of competition,’ said Denise Clark Pope, the author of *Doing School: How We Are Creating a Generation of Stressed Out, Materialistic and Miseducated Students*.

‘When learning becomes about competing with your peers to get ahead, what gets learned is how to compete and not how to learn,’ said Dr. Pope, a lecturer at Stanford University’s school of education ‘Kids learn to cheat, to raise their hands even when they don’t know the answers, to form alliances instead of learning the material we want them to understand.’

Her attitude is shared by some parents, especially ones whose children are frantically competing at exclusive private and suburban schools. But fans of competition complain that it’s been deemphasized for most students. Some schools have dropped honor rolls and class rankings, and the old practice of routinely segregating smart students in separate tracks has given way to the heterogeneous ‘inclusion classroom.’

Competition has long been out of fashion at education schools, as indicated in a 1997 survey of 900 of their professors of Public Agenda, a nonprofit public opinion research group. Only a third of the professors considered rewards like honor rolls to be valuable incentives for learning, while nearly two-thirds said schools should avoid competition.”

Broder JM. *New York Times*. November 21, 2004

Noted by JFL, MD

## Magnetic Resonance Image Correlates of Hemiparesis After Neonatal and Childhood Middle Cerebral Artery Stroke

James P. Boardman, Vijeya Ganesan, Mary A. Rutherford, Dawn E. Saunders, Eugenio Mercuri and Frances Cowan

*Pediatrics* 2005;115:321

DOI: 10.1542/peds.2004-0427

### Updated Information & Services

including high resolution figures, can be found at:  
<http://pediatrics.aappublications.org/content/115/2/321.full.html>

### References

This article cites 42 articles, 19 of which can be accessed free at:  
<http://pediatrics.aappublications.org/content/115/2/321.full.html#ref-list-1>

### Citations

This article has been cited by 17 HighWire-hosted articles:  
<http://pediatrics.aappublications.org/content/115/2/321.full.html#related-urls>

### Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):

#### **Radiology**

[http://pediatrics.aappublications.org/cgi/collection/radiology\\_sub](http://pediatrics.aappublications.org/cgi/collection/radiology_sub)

#### **Cardiology**

[http://pediatrics.aappublications.org/cgi/collection/cardiology\\_sub](http://pediatrics.aappublications.org/cgi/collection/cardiology_sub)

### Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:  
<http://pediatrics.aappublications.org/site/misc/Permissions.xhtml>

### Reprints

Information about ordering reprints can be found online:  
<http://pediatrics.aappublications.org/site/misc/reprints.xhtml>

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2005 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



# PEDIATRICS<sup>®</sup>

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

## **Magnetic Resonance Image Correlates of Hemiparesis After Neonatal and Childhood Middle Cerebral Artery Stroke**

James P. Boardman, Vijeya Ganesan, Mary A. Rutherford, Dawn E. Saunders,  
Eugenio Mercuri and Frances Cowan

*Pediatrics* 2005;115;321

DOI: 10.1542/peds.2004-0427

The online version of this article, along with updated information and services, is  
located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/115/2/321.full.html>

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2005 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN<sup>™</sup>

