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MR of Spastic Tetraplegia

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PURPOSE: To characterize the MR findings in children with spastic tetraplegia by gestational age at birth and perinatal history. **METHODS**: Thirty-four children, 19 boys and 15 girls, with spastic tetraplegia whose brain damage occurred in the prenatal or perinatal period were included in the study. Eighteeen were born at term or later and 16 were premature. Axial proton density– and T2-weighted images and sagittal and coronal T1-weighted images were obtained on a 0.5-T MR imaging unit. **RESULTS:** All patients had abnormal MR findings, with a high prevalence of congenital anomalies (62.5%) in term patients who had experienced no adverse perinatal events. Term patients who had suffered detrimental perinatal events had a wide variety of brain lesions. A high frequency (75%) of periventricular leukomalacia was characteristic of preterm patients. **CONCLUSION:** MR imaging is useful for evaluating structural abnormalities in the brain and the extent of brain injury in patients with spastic tetraplegia.

Index terms: Cerebral palsy; Children, diseases

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Cerebral palsy is a clinical syndrome of nonprogressive motor deficits of central origin with onset in the prenatal or perinatal period or during early infancy (1). Although a wide spectrum of neurologic manifestations are included in this category, spastic tetraplegia (quadriplegia) is the most severe type of cerebral palsy, and it has been defined as a condition with pareses of the upper limbs of the same degree as or more severe than those of the lower limbs, which is analogous to the definition of bilateral hemiplegia (2). Computed tomographic (CT) studies of patients with spastic tetraplegia (3, 4) have shown cerebral atrophy with or without ventricular dilatation, cerebral infarction, porencephaly, and congenital malformations, but the percentage of such patients with abnormal CT findings was relatively low: 59% in the study by Kolawole et al (3) and 67% of all spastic palsy patients reported by Taudorf et al (4). Magnetic resonance (MR) imaging is more sensitive than CT, not only for the assessment of brain myelination but also for the detection of subtle brain malformations and mild white matter changes (5). Several MR studies of cerebral palsy have included patients with spastic tetraplegia (5–8). We conducted an MR study of 38 patients with stable spastic tetraplegic during the period from January 1990 to October 1994 with the aim of defining the MR characteristics of brain lesions in these patients.

Subjects and Methods

Thirty-eight children (22 boys and 16 girls) in whom spastic tetraplegia had been clinically diagnosed by pediatric neurologists were examined by MR imaging at least 4 months after birth. Four patients, whose brain damage occurred after 1 month of age and who had experienced no adverse perinatal events, were excluded. The remaining 34 patients, whose brain damage occurred during intrauterine life, intrapartum, or in the early neonatal period (within 1 month of birth), constitute the subjects of this study. Their ages at the time of the MR examination ranged from 4 months to 14 years (median age, 1 year). Seventeen patients were born at term (patients 1 to 17), one was

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TABLE 1: Clinical data for 34 children with spastic tetraplegia

Case	Sex/Age at MR	Gestational Age at Birth, wk	Weight at Birth, g	Prenatal and Perinatal history
Term and postterm patients (n = 18)				
1	M/11 y	39	3360	None
2	M/2 y	38	2884	None, microcephaly
3	M/14 y	39	2630	CS
4	F/4 mo	40	2894	None, microcephaly
5	M/2 y	39	2524	Breech delivery
6	F/9 mo	39	3756	Consanguinity
7	M/2 y	40	3720	CS
8	F/10 mo	40	2338	CS, twin, microcephaly
9	M/6 y	41	3470	Severe asphyxia, microcephaly
10	M/4 mo	Normal	3860	Convulsion, asphyxia
11	M/4 mo	37	2744	Seizure
12	M/9 mo	41	Normal	Fetal distress, premature detachment of placenta, birth asphyxia
13	F/7 mo	38	1794	Small for age (low birth weight), hypoglycemia, thrombocytopenia
14	M/4 y	38	1730	Fetal distress, CS
15	M/3 y	39	2360	Fetal distress, CS, microcephaly
16	F/4 mo	Normal	Normal	Fetal distress, apnea, microcephaly, subarachnoid hemorrhage
17	M/10 mo	38	2470	CS, cerebral hemorrhage, microcephaly
18	M/1 y	42	3998	Birth asphyxia, cerebral hemorrhage, clavicular fracture
Preterm patients (n = 16)				
19	F/2 y	36	1630	RDS, twin, microcephaly
20	F/3 y	28	1300	Premature
21	F/4 y	29	1620	RDS, birth asphyxia, VSD
22	M/8 y	34	2200	Intubation for apnea at 7 days
23	M/9 mo	35	1750	Fetal distress, CS, hypoglycemia, microcephaly
24	F/11 mo	24	598	Breech, RDS, sepsis, hyperbilirubinemia, microcephaly
25	F/8 mo	34	1844	CS, twin, birth asphyxia
26	F/4 mo	32	1950	CS
27	M/1 y	29	1370	CS, breech delivery
28	F/8 mo	28	1308	CS, birth asphyxia
29	F/3 y	28	1106	Twin
30	M/2 y	28	1442	CS
31	F/2 y	26	942	Premature rupture of membranes, CS
32	M/8 mo	30	1476	Triplets CS, RDS, PVL at sonography
33	M/6 mo	36	1940	CS, microcephaly, 23 days old: DIC, intracranial hemorrhage
34	F/11 mo	28	1370	Cerebral hemorrhage, apnea, respiratory distress

Note.—CS indicates cesarian section; RDS, respiratory distress syndrome; PVL, periventricular leukomalacia; DIC, disseminated intravascular coagulation; and VSD, ventricular septal defect.

born postterm (patient 18), and 16 were premature (patients 19 to 34). Details are provided in Table 1.

The MR examinations were performed on a superconducting MR unit operating at 0.5 T. First, the midline sagittal plane was scanned with a field-echo pulse sequence (50/14/1 [repetition time/echo time/excitations], flip angle of 60°), then axial proton density– and T2-weighted images were obtained using a spin-echo sequence (2000–2300/30,120/2), and, finally, sagittal and coronal T1-weighted images were obtained using a spin-echo (500/30/2) or field-echo (250/14/2, flip angle of 90°) sequence. The section thickness was 10 mm for axial and coronal images and 7.5 or 10 mm for sagittal images. The matrix size was 256 × 256.

Two experienced radiologists familiar with pediatric neuroradiology reviewed the MR images with knowledge of the clinical information and arrived at their diagnoses by consensus after discussion. Findings in the lateral ventricles, basal ganglia and thalami, cerebral cortex, corpus callosum, cerebral white matter, pons, and cerebellum were assessed subjectively as abnormalities of focal signal, size, and volume, and compared with normal configurations. The MR diagnosis in any patient with multiple abnormal findings was based on the dominant finding. *Cerebral atrophy* was diagnosed when diffuse sulcal widening of the cerebrum with symmetical ventricular dilatation without periventricular signal abnormalities was observed. *Minimal change* was diagnosed when thinning of the corpus callosum was the only abnormal finding on any MR images. *Periventricular leukomalacia* (PVL) was diagnosed in patients who had ventriculomegaly with irregular outlines of the body and trigone of the lateral ventricle, a reduced quantity of periventricular white matter, deep prominent cerebral sulci, and periventricular signal abnor-

malities of low intensity on T1-weighted images and high intensity on T2-weighted images (9).

Results

The MR findings in all 34 patients are listed in Table 2. Eight of the 18 term and postterm patients (patients 1 to 8) had an uneventful perinatal experience, and their MR findings included lobar type holoprosencephaly (n = 2), nonlissencephalic cortical dysplasia (cerebral pachygyria, n = 2), agenesis of the corpus callosum (n = 1), bilateral basal ganglia infarction with diffuse cerebral atrophy (n = 1), minimal change (n = 1), and bilateral cerebral porencephaly (n = 1). Two of these patients had no MR findings that could account for their tetraplegia: patient 1 had right-sided diffuse cerebral cortical dysplasia, but the left side of the cerebrum appeared normal; patient 7, who had thinning of the corpus callosum, was considered to have minimal changes. We speculated that in the latter, a very small periventricular injury could have occurred in utero and then been completely absorbed with no resulting periventricular lesion or ventricular deformity.

Ten patients (patients 9 to 18) had had various adverse perinatal experiences, and seven of them (patients 9 to 15) had perinatal abnormalities other than intracranial hemorrhage. The MR findings in these children included multicystic encephalomalacia (n = 1), bilateral basal ganglia infarction with or without cerebral atrophy (n = 2), PVL (n = 2), cerebral atrophy with chronic subdural hematoma (n = 1), and cerebral atrophy with ulegyria of the left occipital lobe (n = 1). Three patients (patients 16 to 18) had had intracranial hemorrhage, and their MR findings were multicystic encephalomalacia with bilateral basal ganglia infarction and chronic subdural hematoma (n = 1), porencephaly and cerebral atrophy (n = 1), and cerebral watershed infarction with chronic subdural hematoma (n = 1).

Fourteen of the 16 preterm patients (patients 19 to 32) did not have intracranial hemorrhage, and their MR findings consisted of PVL (n = 11), cerebral atrophy (n = 2), and bilateral cerebral cortical infarction of watershed regions (n = 1). The MR findings of the other two patients who had a history of intracranial hemorrhage (patients 33 and 34) included PVL (n = 1) and cerebral atrophy associated with chronic subdural hematoma (n = 1).

The frequencies with which various brain sites were involved are as follows: 97% for the corpus callosum and cerebral white matter, 85% for the lateral ventricle, 53% for the cerebral cortex, 32% for the pons, 24% for the basal ganglia and thalamus, and 12% for the cerebellum. The corpus callosal changes consisted of diffuse or focal thinning and agenesis; but in patients with severe brain atrophy, such as multicystic encephalomalacia, the corpus collosum was just traceable. Various degrees of diminished cerebral white matter, depending on the underlying disease, were found. In patients with PVL, not only was the volume of white matter diminished but periventricular hyperintensity on T2-weighted images was observed frequently, and in several such patients, tiny periventricular cavities persisted. Most of the ventricular changes were symmetrical dilatation; the basal ganglia and thalamic changes consisted of infarction, abnormal signal intensity, and atrophy in patients with severe cerebral damage; while the most commonly observed cerebral cortical changes were cortical atrophy and diffuse and focal atrophy. In patients with PVL, the cerebral cortex was well preserved in comparison with that in patients with other diseases. The pontine and cerebellar changes consisted of size diminution, and were found in patients with severe brain atrophy.

Discussion

In a recent article, Krägeloh-Mann et al (8) reviewed the MR findings of 56 patients with bilateral spastic cerebral palsy, but they did not separate the MR features of the relatively small number of tetraplegic patients from those of the diplegic patients, and they discussed them collectively. In our study, we noticed that the abnormal findings in the brain were more severe and more extensive in tetraplegic patients than in diplegic patients, and, therefore, we thought it meaningful to discuss exclusively the MR features of brain lesions in children with tetraplegia. We found a 100% rate of occurrence of abnormal MR findings, although in two patients (patients 1 and 7) the abnormal brain findings were not severe enough to have caused tetraplegia.

It is well known that MR imaging can isolate several definite pathologic changes in the brain relating to the time the injury occurred, and that MR findings in patients with perinatal asphyxia vary according to the degree of asphyxia and

TABLE 2: MR findings in 34 children with spastic tetraplegia

Case/Sex	Lateral Ventricles	Basal Ganglia/Thalamus	Cerebral Cortex	Corpus Callosum	White Matter	Pons	Cerebellum	MR Diagnosis
Term and postterm patients (n = 18)								
1/M	Dilatation	Normal	R cortical dysplasia	Very thin	R patchy T1 and T2 prolongation	Small	Small	R cerebral cortical dysplasia
2/M	Dilatation	Normal	Frontal fusion	Agenesis	Diminished, frontal fusion	Normal	Normal	Holoprosencephaly, lobar type
3/M	Marked dilatation	Normal	Bilateral dysplasia	Thin	Diminished	Normal	Small	Bilateral cerebral cortical dysplasia
4/F	Dilatation	Normal	Frontal fusion	Agenesis	Frontal fusion	Normal	Small	Holoprosencephaly, lobar type
5/M	Dilatation	Normal	Atrophy	Agenesis	Diminished	Normal	Normal	Agenesis of corpus callosum
6/F	Dilatation	High signal bilaterally on T2WI	Atrophy	Normal	Diminished	Normal	Normal	CA, bilateral BG infarction
7/M	Normal	Normal	Normal	Slight thinning	Normal	Normal	Normal	Minimal change
8/F	Normal	Small	Bilateral porencephaly	Just traceable	PVH, diminished	Small	Normal	Bilateral porencephaly
9/M	Marked dilatation	Small	Multicystic change	Just traceable	Almost vanished	Normal	Normal	Multicystic encephalomalacia
10/M	Normal	Normal	Atrophy	Thin	PVH, diminished	Normal	Normal	CA, CSH
11/M	Normal	High signal on T2WI	Normal	Thin	PVH	Normal	Normal	Bilateral BG infarction
12/M	Dilatation	Normal	Atrophy, focal ulegyria	Thin	Diminished	Normal	Normal	Cerebral atrophy, L occipital ulegyria
13/F	Dilatation	Spotty high signal on T2WI	Normal	Thin	PVH, diminished	Small	Normal	PVL
14/M	Dilatation	Normal	Normal	Thin	PVH, diminished	Small	Normal	PVL
15/M	Dilatation	Bilateral T1 and T2 prolongation	Atrophy	Thin	Diminished	Small	Normal	CA, bilateral BG infarction
16/F	Dilatation	Normal	Atrophy	Just traceable	Diminished	Normal	Normal	CA, CSH
17/M	Dilatation	Normal	R porencephaly	Just traceable	Diminished	Small	Normal	Porencephaly, CA
18/M	Marked dilatation	Low signal bilaterally on T1WI	Multicystic change	Just traceable	Markedly diminished	Small	Small	Multicystic encephalomalacia, bilateral BG infarction, CSH
Preterm patients (n = 16)								
19/F	Marked dilatation	Small	Atrophy	Just traceable	PVH, diminished	Small	Normal	PVL
20/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
21/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
22/M	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
23/M	Dilatation	Normal	Watershed infarction	Thin	Bilaterally diminished in occipital lobe	Normal	Normal	Cerebral watershed infarction
24/F	Dilatation	Normal	Atrophic	Thin	PVH, diminished	Normal	Normal	CA
25/F	Marked dilatation	Normal	Bilateral occipital atrophy	Just traceable	PVH, diminished	Small	Normal	CA with ventricular dilatation
26/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Small	Normal	PVL
27/M	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
28/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
29/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
30/M	Normal	Normal	Normal	Thin	PVH	Normal	Normal	PVL
31/F	Marked dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL
32/M	Dilatation	Normal	Normal	Thin	PVH, diminished	Small	Normal	PVL
33/M	Dilatation	Normal	Normal	Just traceable	Diminished	Normal	Normal	CA, CSH
34/F	Dilatation	Normal	Normal	Thin	PVH, diminished	Normal	Normal	PVL

Note.—T2WI indicates T2-weighted images; T1WI, T1-weighted images; PVH, periventricular hyperintensity on T2-weighted images; CA, cerebral atrophy; CSH, chronic subdural hematoma; PVL, periventricular leukomalacia; and BG, basal ganglia.

the gestational age at birth (10–13). However, there is still a large knowledge gap, not only between the MR findings during the neonatal period and the time when cerebral palsy is confirmed clinically, but also between the neuroimaging features during the neonatal period and clinical outcome. In this study, we performed MR imaging after spastic tetraplegia was established clinically. We could not compare these MR findings with those obtained during the neonatal period, since the latter were not available.

The brain lesions found in children with tetraplegia who were delivered at term and who experienced no adverse perinatal events indicated that this group had a higher frequency of congenital anomalies (62.5%), such as cortical dysplasia, agenesis of the corpus callosum, and holoprosencephaly (Fig 1), than did those who suffered such events or those who were born prematurely. Porencephaly may be included as a congenital lesion that arises during intrauterine life (patient 8), but we classified it separately because it was also found as an end-stage feature of neonatal intracerebral hemorrhage (patient 17). A survey of the literature revealed that congenital malformations such as schizencephaly, lissencephaly, polymicrogyria, and hydranencephaly can cause spastic tetraplegia.

MR images in the 10 term patients who experienced adverse perinatal events (patients 9 to 18) showed a variety of brain lesions. Most of these patients had had severe perinatal asphyxia and/or hypoxic ischemic encephalopathy. Hypoxic-ischemic encephalopathy in term patients caused parasagittal or watershed cerebral injury, selective neuronal necrosis, focal and multifocal ischemic cerebral necrosis, and status marmoratus of the basal ganglia and thalamus (14). Of these five pathologic entities, parasagittal cerebral injury and focal and multifocal ischemic cerebral necrosis can cause spastic tetraplegia. Among our patients, those with focal or multifocal ischemic cerebral injury, characterized by injury to all cellular elements in a vascular distribution and resulting from either venous or arterial occlusion, usually with a middle cerebral arterial distribution, had porencephaly and multicystic encephalomalacia (Fig 2). Parasagittal cerebral injury was associated with bilateral, often symmetrical, wedge-shaped cerebral cortical and subcortical infarctions that occurred in a characteristic superomedial distribution between the major arterial distributions (patients 16 and 23), although the latter patient was born prematurely (Fig 3).

MR imaging disclosed various brain lesions, including multicystic encephalomalacia, cerebral infarction, cerebral atrophy, and PVL, with no particular dominant pattern. In the 10 term patients who experienced adverse perinatal events, cerebral atrophy was the dominant imaging feature in two patients (patients 10 and 12). Cerebral atrophy was substantial in both, and in one was associated with chronic subdural hematoma, which implied that the brain shrinkage after brain ischemia was severe. Three other patients had significant cerebral atrophy with bilateral basal ganglia infarction (patients 6 and 15) and porencephaly (patient 17).

PVL was the dominant imaging feature in preterm patients with spastic cerebral palsy, but it was also found in two of our term patients. Krägeloh-Mann et al (8) found PVL in 53% of term children with no clinical evidence of perinatal/neonatal antecedents. They speculated that PVL was acquired prenatally in the absence

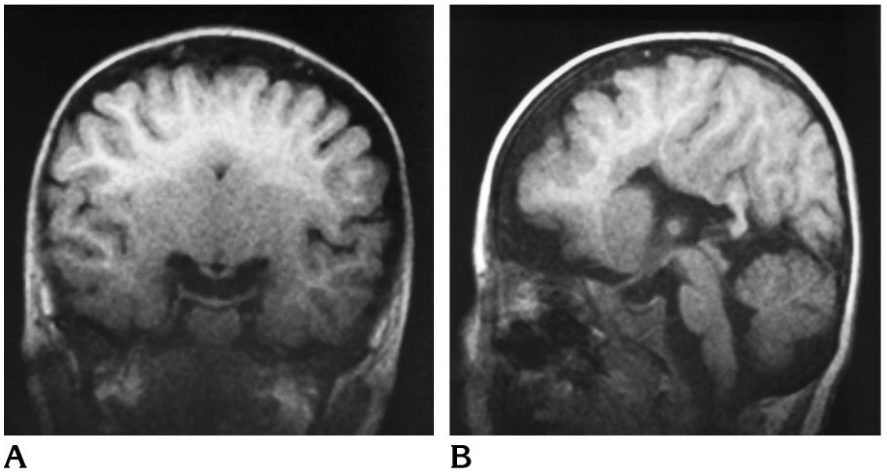

Fig 1. Patient 2: 2-year-old boy born at 38 weeks (birth weight, 2884 g) with microcephaly and spastic tetraplegia. His mother had neither inheritable disease nor illness during pregnancy.

A and *B*, T1-weighted MR images show fused frontal lobes, hypoplasia of the corpus callosum, and separate thalami. The diagnosis was lobar-type holoprosencephaly.

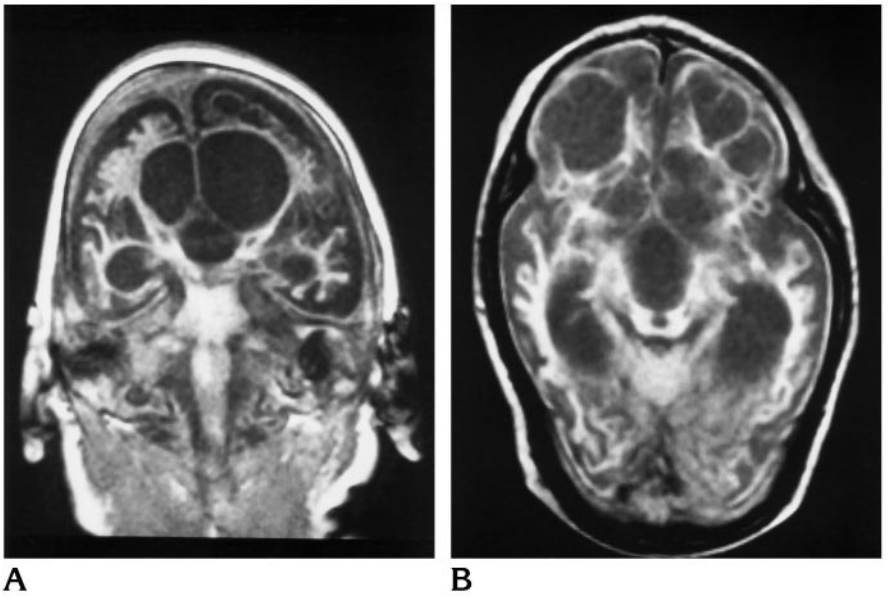

Fig 2. Patient 18: 1-year-old boy born at 42 weeks (birth weight, 3998 g) in whom delivery was complicated by clavicular fracture, birth asphyxia, and intracerebral hemorrhage. After stabilizing, he has been vegetative and tetraplegic with no voluntary movements.

A and *B*, MR images show marked diminution of cerebral white and gray matter with diffuse cystic changes as well as high-density fluid collection in the bilateral subdural spaces on T1-weighted image (*A*). Bilateral linear low-intensity bands are apparent in the bilateral corticospinal tracts on T1- (*A*) and proton density–weighted (*B*) images. These findings are thought to represent a severe type of multicystic encephalomalacia.

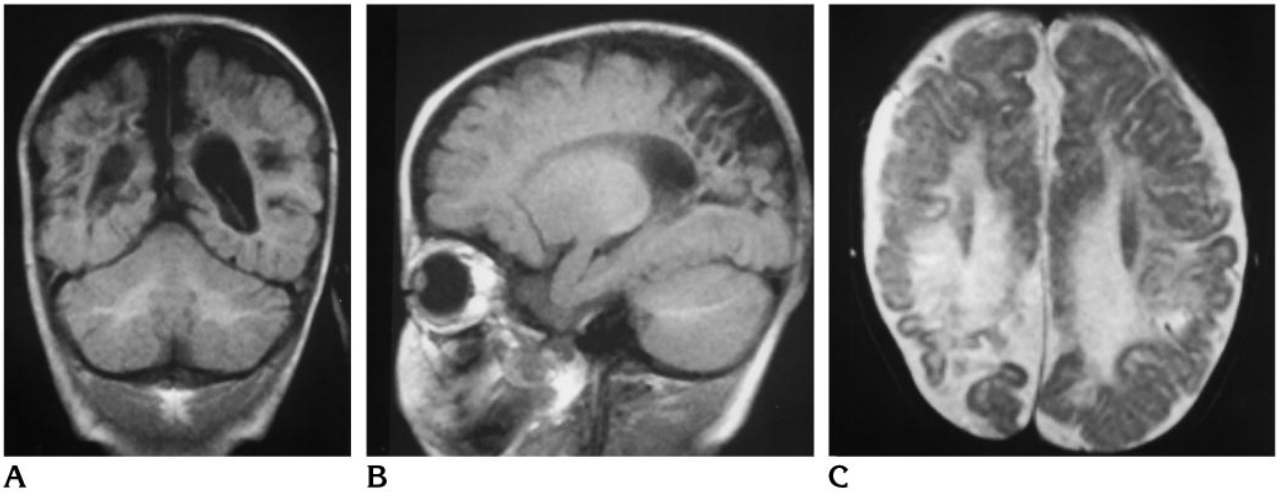

Fig 3. Patient 23: 9-month-old boy born at 35 weeks (birth weight, 1750 g) whose delivery was complicated by fetal distress, necessitating cesarian section and resulting in hypoxic-ischemic encephalopathy. Hypoglycemia was present during the neonatal period.

A–C, MR images show hypointense cystic areas in the bilateral temporooccipital region and dilated lateral ventricles on T1-weighted images (*A* and *B*). Axial T2-weighted image (*C*) shows bilateral large hyperintense area extending from the frontal to the occipital white matter. The diagnosis was bilateral cerebral watershed infarctions, reflecting the parasagittal cerebral injury.

of compromising perinatal/neonatal events, and a prenatal pathogenesis was suggested by various clinical findings, such as preeclampsia, microcephaly, and small size for gestational age. Moreover, they found a correlation between PVL and clinical outcome and concluded that PVL seemed to be associated with more severe disability in term than preterm infants.

Rademaker et al (7) described a pattern of brain damage known as *central cortico-subcortical cerebral damage,* characterized by bilateral cortical and subcortical lesions bordering the central sulcus with or without bilateral thalamic lesions, in seven term infants. These lesions were caused by perinatal and postnatal asphyxia and produced the clinical outcome of spastic tetraplegia.

In another study of preterm infants (15), hypoxic-ischemic encephalopathy was found to cause periventricular hemorrhagic infarction, including germinal matrix and intraventricular hemorrhage, and PVL. The subsequent neuroimaging results included PVL, cerebral atrophy, cerebral infarction, hydrocephalus, and poren-

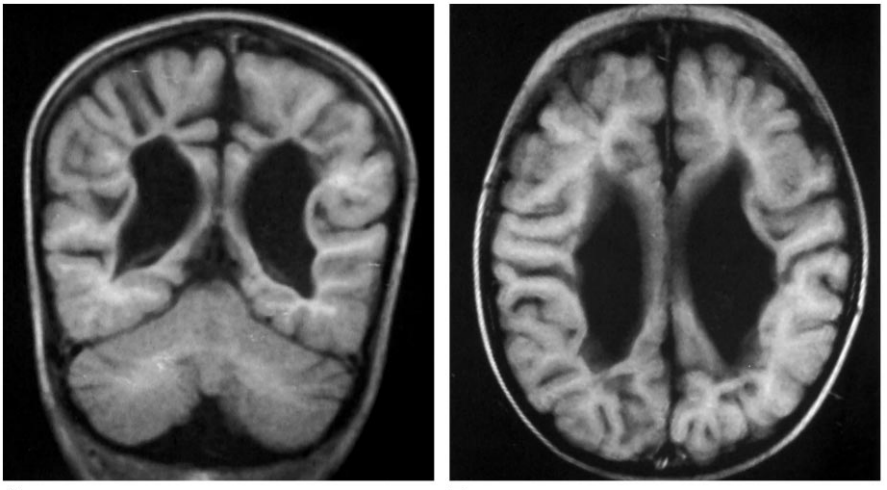

Fig 4. Patient 21: 4-year-old girl born at 29 weeks (birth weight, 1620 g) who had neonatal asphyxia and respiratory distress syndrome complicated by congenital heart disease (ventricular septal defect).

A and *B*, MR images show marked diminution of cerebral white matter volume (*A*) and irregular ventricular dilatation (*B*). The diagnosis was PVL.

cephaly due to periventricular hemorrhagic infarction. In our series, 12 (75%) of the 16 preterm patients had PVL (Fig 4), but in four of them (patients 26, 27, 29, and 30), PVL was disclosed by MR imaging with no apparent hypoxic-ischemic encephalopathy, suggesting that a not inconsiderable percentage of the PVL lesions had a prenatal origin (8).

We noted associated chronic subdural hematoma in the patients with cerebral atrophy and multicystic encephalomalacia, and these patients tended to have severe and destructive brain changes. Subdural hematoma may be a result of venous tearing due to rapid brain shrinkage caused by destructive processes in the brain parenchyma, and such a subdural hematoma may not be reparable, despite a shunt procedure, because the brain has already shrunk.

In conclusion, we found that the MR imaging features of 34 patients with spastic tetraplegia comprised a wide variety of brain lesions, reflecting various pathologic entities. Abnormal MR findings were universal in these patients and the extent of the brain injuries was relatively severe. We also noticed that several definite pathologic changes in the brain, relating to the time the injury occurred, could be found using MR imaging, except in term and postterm patients who had experienced adverse perinatal events, a group in whom no dominant imaging features were present. MR imaging is a useful technique for evaluating structural abnormalities in the brain and the extent of brain injury in patients with spastic tetraplegia.

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