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Multiple Sclerosis in Adolescents: CT and MR Findings

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The MR and CT findings in 12 adolescents with multiple sclerosis were compared with reported findings in adults. The adolescent group showed a more striking female predominance, more severe disease characteristics, and more frequent infratentorial involvement. Cortical atrophy and abnormal iron accumulation in the basal ganglia were uncommon in the adolescents. Neither group demonstrated a correlation between symptom severity and either extent or location of disease as delineated by MR imaging. MR was more sensitive than CT in detecting demyelinating plaques.

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Multiple sclerosis (MS) is a rare disorder in children and adolescents, with this age group accounting for less than 1% of all cases [1]. Vague symptoms and an often erratic clinical course in this age group made it difficult to establish a diagnosis prior to the advent of MR imaging. While a few scattered case reports have appeared in the literature [2-4], no series has delineated the appearance of childhood MS on high-resolution CT and MR studies. We present the imaging spectrum based on a series of 12 documented cases.

Subjects and Methods

MR images obtained during the period from January 1985 to April 1989 of all children and adolescents either referred to the University of Utah for examination because of clinically suspected MS or whose scans themselves were considered suggestive of demyelinating disease were reviewed. Fourteen such cases were found; 12 had CT/MR findings that suggested MS. Ten of the 12 patients were female (Table 1), and five were 17 years old or younger at the time of initial imaging. Seven patients had symptom onset in their mid to late teens but were 18 or older at the time of imaging. Only two patients, among the oldest in the series, had a definite clinical diagnosis of MS prior to MR scanning. Two other patients were referred for suspected MS but were found to have nondemyelinating disorders (one had a high cervical disk herniation with cord compression and the other had progressive idiopathic arteriopathy of childhood). The remaining 12 patients either had CSF studies consistent with MS (presence of oligoclonal banding or evidence of increased immunoglobulin synthesis rates), abnormal visual or somatosensory evoked potentials, or a subsequent relapsing-remitting or chronic-progressive course consistent with clinically definite MS.

Eleven patients had both plain and contrast-enhanced high-resolution axial CT scans. A double-dose delayed scan was obtained in one patient. Axial and sagittal T1- and T2-weighted axial MR scans were obtained at 1.5T in all 12 patients. In one patient, contrast-enhanced MR images were also obtained. MR scans were evaluated for foci of abnormal signal in the periventricular regions, both hemispheres, cerebellum, brainstem, and (in four patients) the spinal cord. The thalamus, putamen, and caudate nuclei were assessed for the presence of abnormally decreased signal on T2-weighted images. The MR index developed by Uhlenbrock et al. [5] was modified and used to assess disease severity in each case (Table 2).

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TABLE 1: Case Material

Case No.	Sex	Age at First Symptoms	Age at First Scan	Description
1	F	16	17	18-month history of intermittent right-sided numbness, diffuse weakness
2	F	14	15	Vague neurologic complaints of diffuse numbness, upper extremity weakness
3	F	17	20	3-year history of bilateral upper extremity tingling
4	F	16	16	2-week history of right arm and leg numbness and tingling, weakness of left arm and leg (CSF was positive for oligoclonal banding)
5	F	17	21	Transient left facial numbness for 4 years; 3-week history of ataxia, diplopia
6	F	16	22	5-year history of progressive neurologic deficits, clinical diagnosis of MS; new onset of temporal visual field defects
7	F	18	19	Visual symptoms only
8	F	17	21	4-year history of visual difficulties, extremity numbness, speech difficulties, loss of bowel and bladder control; clinical diagnosis of MS; recent-onset seizures
9	F	16	22	6-year history of intermittent, vague neurologic complaints
10	F	17	17	"Flu" followed by lethargy; facial numbness, hand incoordination
11	M	17	18	Seizures, bilateral Babinski reflexes; CSF positive for oligoclonal bands
12	M	15	15	10-day history of left upper lip, cheek, face, scalp numbness; CSF positive for oligoclonal bands

Results

CT

Eleven patients had plain and contrast-enhanced brain CT scans (Table 3). When present, lesions were typically hypo- or isodense on plain scans, with variable enhancement after contrast administration. Differing degrees of plaque enhancement were often seen in the same patient varying from no enhancement in some low-density lesions to ring or inhomogeneously increased density in others. Double-dose delayed scanning was performed in one patient and disclosed additional lesions not identified on the immediate postcontrast study (Fig. 1). The three patients with spinal cord lesions who had negative brain MR studies also had normal CT brain scans. In all patients, more lesions were seen on MR than on CT (Table 3).

MR Imaging

Four patients had symptoms of a spinal cord lesion and thus had cord scans; all four had cord lesions (Table 3). Three

TABLE 2: Modified MR Index for Assessing MS Plaques*

A. Periventricular plaques
A0. No plaques
A1. No more than five plaques, with single plaque not larger than 8 mm, or one plaque larger than 8 mm and no more than three plaques; no confluence
A2. More than five plaques, more than three plaques with one plaque larger than 8 mm, or two or more plaques larger than 8 mm; in addition or exclusively, there is periventricular confluence but the bodies of the ventricles are mainly unaffected
A3. More than 12 plaques and/or periventricular confluence, including the bodies of the ventricles
B. Hemispheric plaques
B0. No plaques
B1. No more than five plaques
B2. More than five plaques
C. Cerebral atrophy
C1. White matter atrophy
C2. Focal cortical atrophy
C3. Diffuse cortical atrophy
C4. White matter and cortical atrophy
D. Cerebellar plaques
D0. No plaques
D1. One or more plaques
E. Brainstem plaques
E0. No plaques
E1. One or more plaques
F. Spinal cord plaques
F0. No plaques
F1. One or more plaques
G. Abnormal iron deposition in thalamus, putamen, and/or caudate nuclei
G0. Normal signal
G1. Decreased signal

* After Uhlenbrock et al. [5]; category added for abnormal nonheme iron deposition.

of these patients had a solitary focus of increased signal in the cervical cord and normal brain studies (Fig. 2); CSF was positive for oligoclonal bands in all three. The fourth patient had both thoracic and cervical cord lesions in addition to multiple lesions in the periventricular deep white matter characteristic of long-standing MS. Two of the patients had mild mass effect associated with their cord lesions.

The remaining eight patients had multiple brain scans, but no spinal cord studies were performed. All but one (case 11) had supra- and infratentorial lesions (Fig. 3) with most having severe disease as evidenced by multiple confluent periventricular plaques and extensive white matter involvement (Tables 2 and 3). Lesions were typically iso- or hypointense on T1-weighted images and hyperintense on T2-weighted studies (Fig. 4). Beveled or ring lesions with mixed signal on T1-weighted scans and various degrees of increased signal on proton-density scans were also common, as were ovoid plaques (Fig. 1D) that have recently been described in MS [6]. In the one patient who received MR contrast material, additional lesions were identified on the postcontrast study that were not seen on proton-density or T2-weighted studies.

One patient with long-standing severe disease had cortical atrophy as well as abnormally decreased signal in the thalamus, putamen, and basal ganglia on T2-weighted images (Fig. 5); none of the other 10 cases had evidence of these associated abnormalities. One patient had a biopsy-proved hypothalamic glioma in addition to florid MS.

TABLE 3: Radiologic Findings

Case No.	CT Findings	MR Findings	MR Index*
1	Brain: normal	Brain: normal Cord: solitary intramedullary focus of increased signal on T2-weighted image at C6; mild mass effect	A0, B0, C0, D0, E0, F1, G0
2	Brain: single lesion with mild enhancement	Brain: multiple confluent periventricular lesions with deep white matter and brainstem lesions. Cord: thoracic cord lesions, T2-T4	A3, B2, C0, D0, E1, F1, G0
3	Brain: normal	Brain: normal Cord: solitary intramedullary focus of increased signal at C3-C4; no mass effect	A0, B0, C0, C0, E0, F1, G0
4	Brain: normal	Brain: normal Cord: single midcervical intramedullary lesion; mild mass effect	A0, B0, C0, D0, E0, F1, G0
5	Brain: none	Brain: multiple large brainstem, cerebellar, and confluent periventricular lesions, iso- or hypointense on T1-weighted image, hyperintense on T2-weighted image	A3, B2, C0, D1, E1, G0
6	Brain: multiple enhancing plaques, deep cerebral white matter; enhancing suprasellar mass	Brain: brainstem, cerebellar, and confluent periventricular lesions, iso- or hypointense on T2-weighted image, hyperintense on T2-weighted image; hypothalamic glioma	A3, B2, C0, D1, E1, G0
7	Brain: single enhancing pontine lesion	Brain: multiple confluent periventricular and brainstem lesions of variable signal on T1-weighted image, hyperintense on T2-weighted image	A3, B2, C0, D1, E1, G0
8	Brain: multiple enhancing lesions, centrum semiovale	Brain: severe confluent periventricular, brainstem, cerebellar, and medullary plaques; generalized cortical atrophy; corpus callosum atrophy; low signal in basal ganglia	A3, B2, C0, D1, E1, G1
9	Brain: multiple periventricular low-attenuation lesions, variable enhancement	Brain: discrete brainstem and periventricular lesions, hypointense on T1-weighted image, hyperintense on T2-weighted image	A2, B2, C0, D1, E1, G0
10	Brain: multiple iso- or hypointense lesions with variable enhancement on double-dose delayed scans	Brain: mixed-signal lesions, deep white matter, brainstem, and cerebellum	A3, B2, C0, D1, E1, G0
11	Brain: normal	Brain: scattered foci of increased signal in periventricular white matter; two to three lesions not seen on T2-weighted image enhanced after contrast; posterior fossa normal	A1, B1, C0, D0, E0, G0
12	Brain: normal	Brain: scattered supratentorial periventricular nonconfluent foci of increased signal; solitary lesion at left fifth cranial nerve root in posterior fossa	A1, B2, C0, D0, E1, G0

* See Table 2 for descriptions.

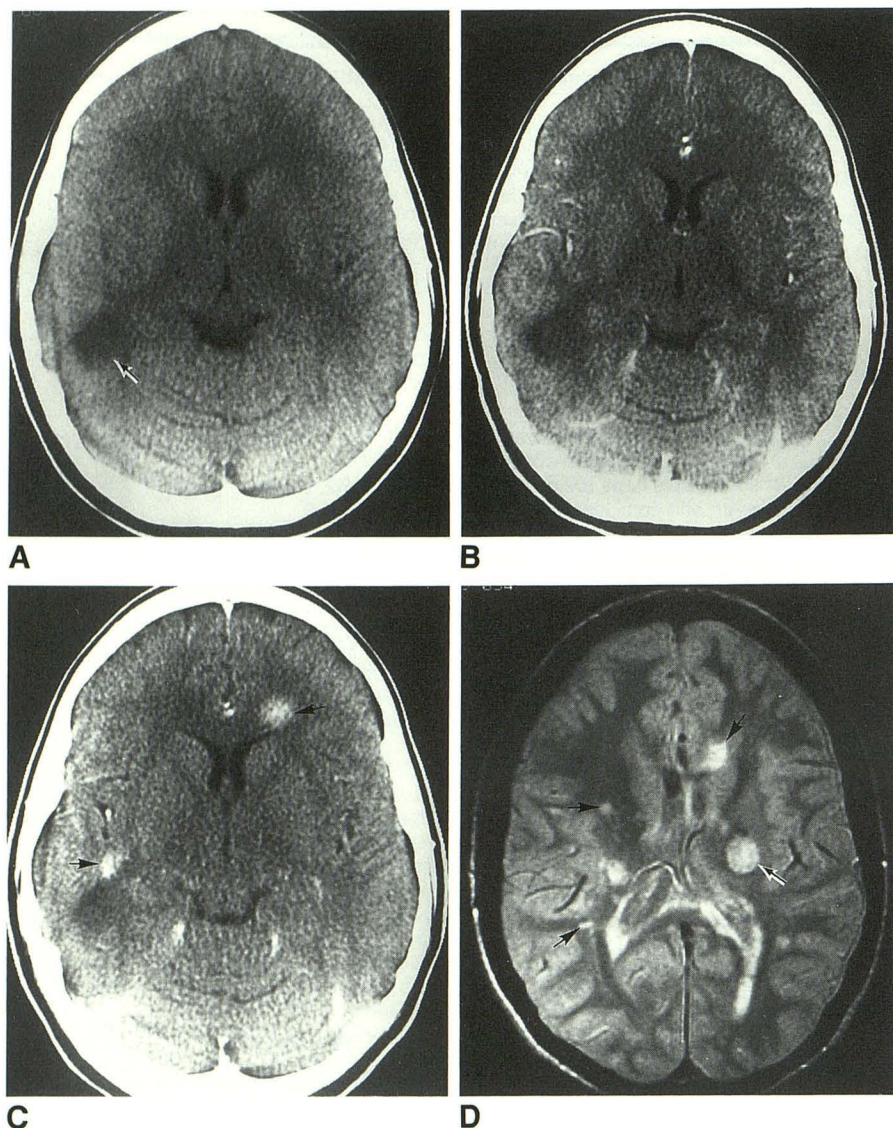


Fig. 1.—Case 10: 17-year-old girl with facial numbness and hand incoordination.

A, Axial CT scan without contrast shows low-density lesion in posterior right temporal lobe (arrow).

B, CT scan immediately after contrast administration shows no change in attenuation of the posterior temporal lobe lesion.

C, Double-dose delayed CT scan shows additional lesions (arrows) not apparent in A or B.

D, MR image (2700/30/1) through midventricular level shows multiple lesions (black arrows) not seen on CT. Note "beveled" or "target" appearance of lesion in posterior limb of left internal capsule (outlined arrow). The ovoid plaque may represent perivascular demyelination as described by Horowitz et al. [6]. MR revealed numerous other plaques in this patient that were inapparent on CT, even with double-dose contrast enhancement. (Case courtesy of Dr. A. Wirthlin, Salt Lake City, UT.)

Discussion

Several striking differences between MS in adults and in our series of adolescents were readily apparent. The female to male ratio in most adult MS series varies from 1.4:1 to 1.9:1. By contrast, 10 of our 12 patients with disease onset in adolescence were female.

In the large series of adult cases reported by Uhlenbrock et al. [5], lesion severity as rated on their MR index varied widely from normal to severe. While three of our four adolescents with spinal cord lesions had normal brain MR studies, all but two of the nine cases with positive brain scans had severe disease characterized by the presence of either more than 12 plaques or confluent periventricular lesions (both males in our series had mild disease).

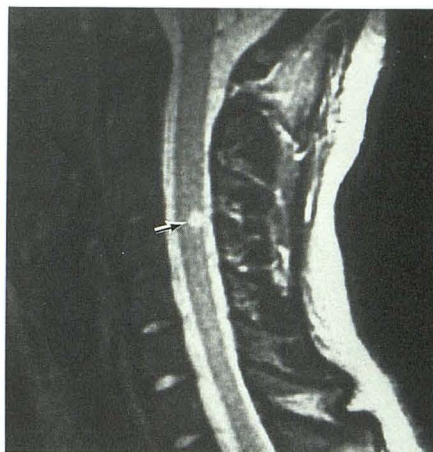
While cerebellar and brainstem lesions were seen in only one third and 14% of adults, respectively, in the Uhlenbrock series, seven of the 10 adolescents had cerebellar plaques and eight of 10 had brainstem disease even though symptoms

of posterior fossa involvement were often lacking. In contrast, cortical and white matter atrophy were seen in only one of the adolescents, manifested by sulcal enlargement and thinning of the corpus callosum. The relative lack of brain atrophy may reflect comparatively short disease duration in spite of its severity (30–70% of all patients with MS require at least ambulatory assistance 15 years after symptom onset) [7].

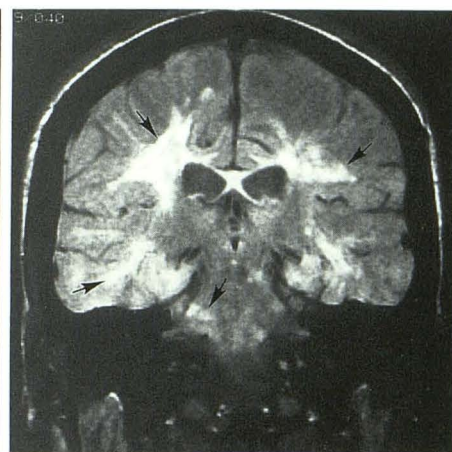
Other investigators report the presence of abnormally decreased signal intensity in the thalamus or putamen on T2-weighted images in 90% of adults with MS [7], probably secondary to nonheme iron deposition. Only one of the 12 adolescents had such findings; this patient had the most severe, long-standing disease in our series.

While we did not actually grade clinical impairment by using the Kurtzke score, severity of symptoms in our series did not correlate with disease extent as seen on MR scans (Tables 1 and 3). Some patients (e.g., case 7) with severe disease on MR had minimal symptoms. This lack of correlation between clinical data and imaging findings is consistent with the results reported for adults [8, 9].

Fig. 2.—Case 3: 20-year-old woman with 3-year history of bilateral upper extremity paresthesias; normal brain scan. Sagittal MR image (2500/80/1) shows solitary focus of increased signal in midcervical cord (arrow).



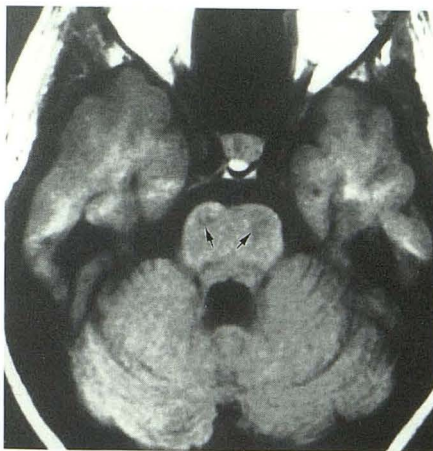
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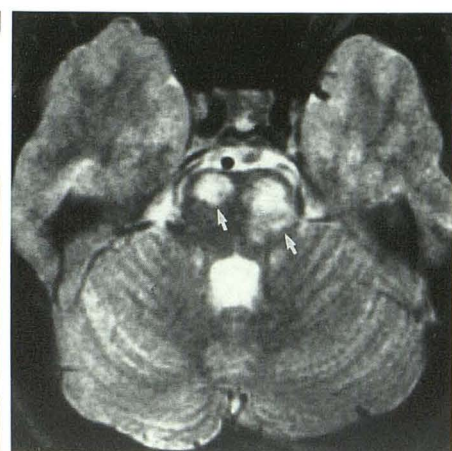
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Fig. 3.—Case 6: 22-year-old woman with 5-year history of facial numbness; recent onset of ataxia, diplopia, and visual-field defects. Coronal MR image (2500/20/1) shows extensive confluent periventricular deep white matter and brainstem disease (arrows).

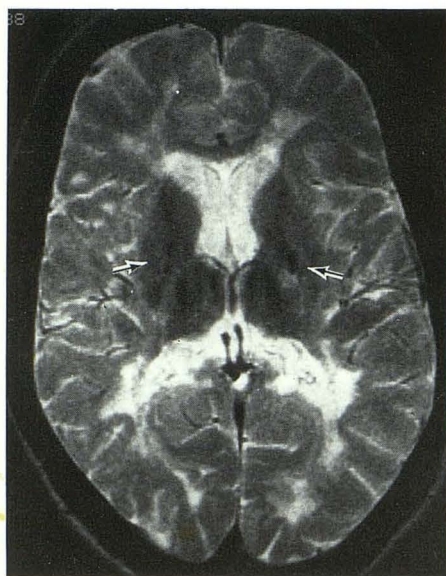
Fig. 4.—Case 9: 22-year-old woman with 6-year history of vague neurologic complaints.
A, Axial MR image (600/20/2) shows low signal foci in brainstem (arrows).
B, Hypointense lesions seen in A become hyperintense (arrows) on this T2-weighted scan (2800/70/1).



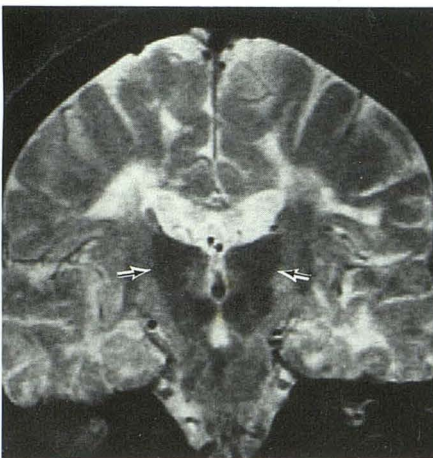
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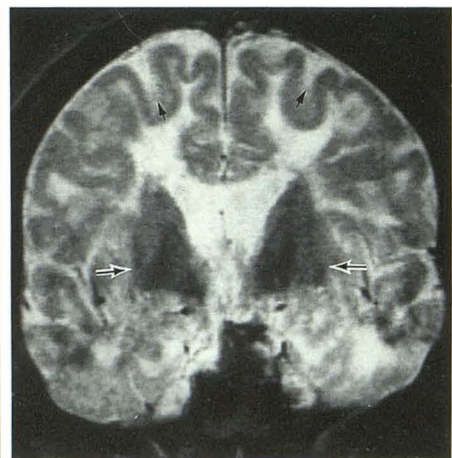
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A



B



C

Fig. 5.—Case 8: 21-year-old woman with 4-year history of progressive visual difficulty, upper extremity numbness, and urinary urgency and frequency; recent onset of seizures. Presumptive diagnosis of MS (CSF positive for oligoclonal bands) was questioned because seizures are uncommon in MS.
A and B, Axial (A) and coronal (B) MR scans (2500/70/1) show extensive confluent periventricular and deep white matter lesions. Note striking low signal in basal ganglia (outlined arrows in A-C).
C, More anterior coronal section shows cortical atrophy with dilated sulci (black arrows). Note marked thinning of the corpus callosum on B and C.

CT was less sensitive than MR in all patients studied with both methods (Table 3). Five of our patients had solitary or multiple low-density foci on CT with variable enhancement of at least one lesion. In the one patient who had a double-dose scan, enhancing foci were identified on the delayed scan that were not apparent on the immediate postcontrast study. Other authors have also noted that acute lesions display enhancement on delayed scans after administration of high-dose contrast material while chronic lesions remain unchanged. In all cases more lesions were seen on MR than on CT.

Of interest are the two other adolescents whom we scanned for clinically suspected MS. One, an 18-year-old woman with a 3-year history of intermittent upper extremity weakness and paresthesia, had a normal MR brain scan. The sagittal T1-weighted series disclosed C3–C4 disk herniation with cord compression. Discectomy resulted in complete relief of symptoms. A 13-year-old girl with left upper extremity weakness had numerous dilated vascular channels seen on MR. Idiopathic progressive arteriopathy of childhood (Moya-Moya-type pattern) was confirmed at angiography. MR is extremely helpful in distinguishing these and other clinical MS mimics from true demyelinating disease.

Conclusions

On the basis of this small series of 12 adolescents, we conclude that female predominance as well as the frequency

of MR-demonstrated lesions in the cerebellum and brainstem appear to be higher in adolescents than that usually encountered in adults. Atrophy and abnormal iron deposition were uncommon in the adolescents.

REFERENCES

1. Gall JC, Hayles AB, Siekert RG, Keith HM. Multiple sclerosis in children: a clinical study of 40 cases with onset in childhood. *Pediatrics* **1958**;21:703–709
2. Golden GS, Woody RC. The role of nuclear magnetic resonance imaging in the diagnosis of MS in childhood. *Neurology* **1987**;37:689–693
3. Di Mario FJ Jr, Berman PH. Multiple sclerosis presenting at 4 years of age: clinical and MRI correlations. *Clin Pediatr* **1988**;27:32–37
4. Nowell AM, Grossman RJ, Hackney DB, Zimmerman RA, Goldberg HI, Bilaniuk LT. MR imaging of white matter disease in children. *AJNR* **1988**;9:503–509
5. Uhlenbrock D, Seidel D, Gehlen W, et al. MR imaging in multiple sclerosis. *AJNR* **1988**;9:59–67
6. Horowitz AL, Kaplan RD, Grewe G, et al. The ovoid lesion: a new MR observation in patients with multiple sclerosis. *AJNR* **1989**;10:303–305
7. Drayer B, Burger P, Hurwitz B, Dawson D, Cain J. Reduced signal intensity on MR images of thalamus and putamen in multiple sclerosis: increased iron content? *AJNR* **1987**;8:411–419
8. Brainin M, Reisner T, Neuhold A, Omasits M, Wicke L. Topological characteristics of brainstem lesions in clinically definite and clinically probable cases of multiple sclerosis: an MRI study. *Neuroradiology* **1987**;29:530–534
9. Paty DW, Oger JF, Kastrukoff LF, et al. MRI in the diagnosis of MS: a prospective study with comparison of clinical evaluation, evoked potentials, oligoclonal banding, and CT. *Neurology* **1988**;38:180–185