Spinal cord multiple sclerosis and devic neuromyelitis optica in children.

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LETTERS

Spinal Cord Multiple Sclerosis and Devic Neuromyelitis Optica in Children

We read with interest the article by Glasier et al, "Clinical, Neurodiagnostic, and MR Findings in Children with Spinal and Brain Stem Multiple Sclerosis" (1). In this study, five of seven children with spinal multiple sclerosis (MS) had lesions that extended over three or more segments with concomitant cord swelling. This differs from our experience as well as that of previous authors. Typical plaques in the spinal cord are usually small, extend over fewer than two vertebral segments, involve less than one half the cross-sectional area of the spinal cord, and usually do not show cord enlargement (2–4).

We acknowledge that MS may present differently in children. It is known that acute MS plaques in children may be large and can simulate a brain tumor on magnetic resonance (MR) (5). However, we postulate that many of the cases described by Glasier et al may actually represent Devic syndrome. In their series, four of the five patients with swollen spinal cords and lesions extending for multiple segments presented with or subsequently developed optic neuritis. In two of these patients, the initial MR of the brain was negative. The lesions described in the spinal cords of these four patients bear a striking resemblance to a subacute necrotic myelopathy that is the characteristic spinal lesion in Devic neuromyelitis optica. The clinical syndrome is regarded as a form of MS or as a separate neurologic syndrome. Devic neuromyelitis optica is characterized by an extensive necrotizing myelopathy, associated with "optic neuritis" usually occurring before the onset of the myelopathy. Brain MR studies typically are normal initially, without evidence of typical MS plaques. Oligoclonal bands also are typically negative (4, 6).

In conclusion, the diagnosis of MS will continue to be a dilemma in children. However, we feel that the features described on the spinal MR studies in Glasier's article are most compatible with a necrotic myelopathy as opposed to atypical demyelinating plaques of multiple sclerosis. Very little has been written about Devic neuromyelitis optica in the literature, and its exact classification remains unclear. It also is not known whether there is an increased incidence of Devic neuromyelitis optica in children. We were very intrigued by the series presented by Glasier et al and expect it will elicit further study about MS and its related conditions in children.

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Reply

DeLara et al imply that the patients in our series probably have a necrotic myelopathy known as Devic neuromyelitis optica (DNO) rather than MS. As DeLara et al state in their letter, it is currently unclear whether DNO is an entity separate from MS. Strictly speaking, if one is to differentiate DNO as a clinicopathologic entity separate from MS, then clinical and neuroimaging findings should be isolated to the optic nerves and spinal cord (1). Indeed, none of our patients had evidence of disease limited to the optic nerves and spinal cord, although patient 7 has only spinal involvement without evidence of brain disease. This single patient with disease apparently localized to the spine has not developed optic neuritis and does not have evidence of necrotizing myelopathy clinically or at imaging. Patients 1 through 6 in our series all eventually had clinical and/or neuroimaging evidence of waxing and waning neurologic disease localized to more than one area of the central nervous system; in short, findings characteristic of MS.

The extensive cord swelling and enhancement as reported in our paper indeed has not been reported by previous authors and was meant to be one of the major points of our manuscript. DeLara et al imply that the lack of oligoclonal banding in the cerebrospinal fluid is characteristic of DNO; however, as noted in our paper, children with MS also have a low incidence of cerebrospinal fluid oligoclonal banding.

We do not dispute the fact that DNO may exist as a separate clinical syndrome in some patients. All the patients in our series, however, were diagnosed by experienced pediatric neurologists as having clinical MS. Our point was that demyelinating disease in children can present with confusing clinical and neuroimaging findings

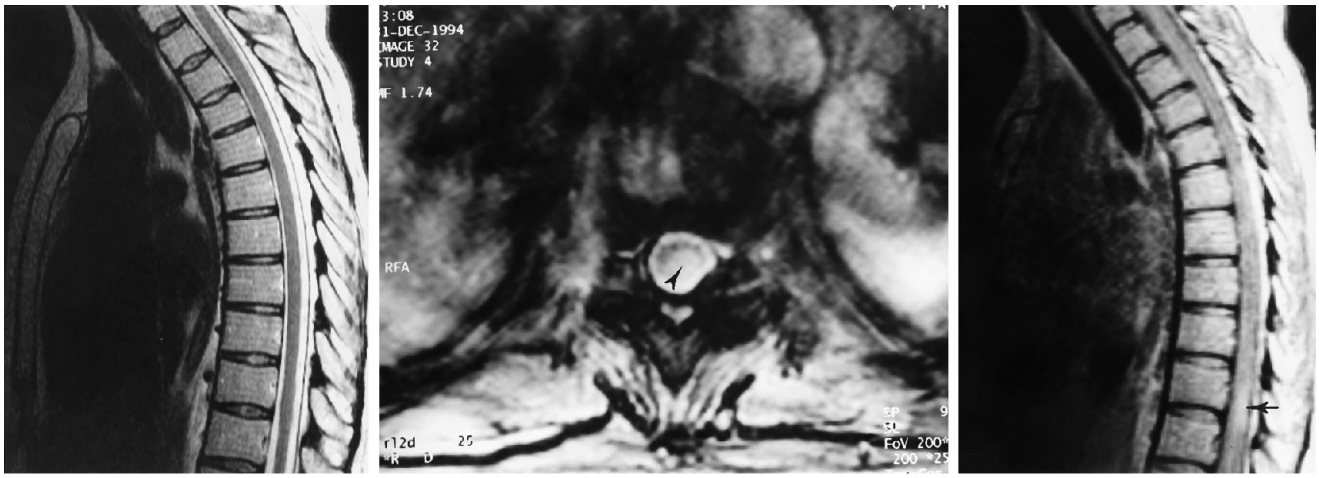

Fig 1. *A*, In this patient with demyelinating cerebral lesions and suspicion of intramedullary lesion, sagittal T2-weighted FSE MR image (4000/128 [repetition time/echo time]) was normal.

B, Because this patient presented symptoms and signs referable to lower thoracic spinal cord lesion, supplemental T2*-weighted gradient-echo axial sections (700/22; flip angle, 25°) were obtained on the lower thoracic spinal cord and disclosed a hyperintense intramedullary lesion (*arrowhead*).

C, A proton density–weighted CSE sagittal image confirmed the presence of this lesion (*arrow*).

and can be confused with neoplastic involvement of the brain stem and spinal cord. Further research into the etiology of MS and other demyelinating diseases of childhood is necessary to define and categorize these entities further and, it is hoped, to develop new treatment modalities.

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1. Mandler RN, Davis LE, Jeffery DR, Kornfield M. Devic's neuromyelitis optic: clinicopathological study of 8 patients. *Ann Neurol* 1993;34:162–168

Are Sagittal T2-Weighted Fast Spin-Echo MR Images Sufficient for Routine Spine Imaging?

Fast spin-echo (FSE) is a recently developed technique for the rapid acquisition of MR images with contrast that is similar, although not identical, to conventional spin-echo (CSE) (1, 2). For routine brain imaging, dual-echo (proton density– and T2-weighted) FSE images have been shown to have a sensitivity similar to dual-echo conventional spin-echo sequences (1, 3), with a shorter scanning time. In the diagnosis of vascular spinal cord abnormalities, the superior spatial resolution of FSE pulse sequences has been emphasized (4).

FSE sequences also appeared as accurate as conventional sequences in the evaluation of intradural disease of the spine in the sagittal plane (5). The use of multiarray coils and T2-weighted FSE sagittal sequences has proved to allow rapid and sensitive detection of spinal cord lesions in MS (6). Unlike these last two studies (5, 6), which used dual-echo FSE sequences, Jones et al (1) used single-echo (T2-weighted) sagittal FSE sequences for routine spine imaging. In our institutions, we also have replaced dual-echo CSE with single-echo (T2-weighted) sagittal FSE sequences for routine spine imaging.

We wish to report one patient with demyelinating cerebral lesions and clinical evidence of intramedullary lesion: T2-weighted FSE sagittal images showed no spinal cord lesion (Fig 1A). Because the patient presented symptoms and signs referrable to lower thoracic spinal cord lesion, we obtained axial T2*-weighted gradient-echo images that demonstrated a hyperintense intramedullary lesion at the level of T10-11 (Fig 1B). Proton density–weighted CSE sagittal images (Fig 1C) confirmed the existence of this

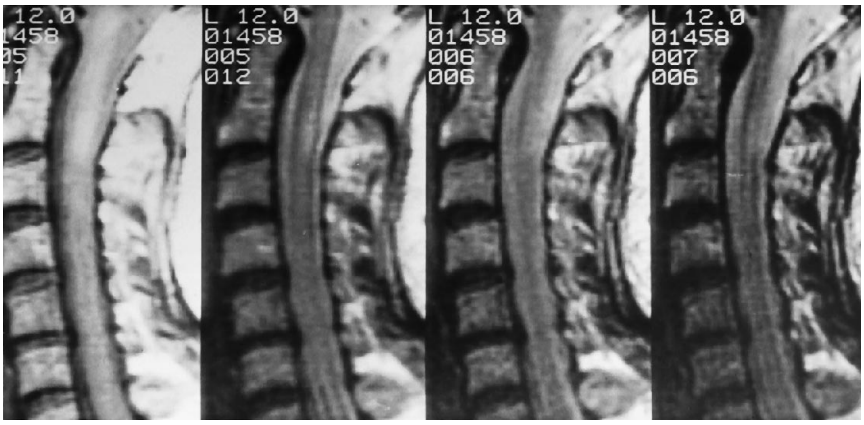

Fig 2. Patient with an MS plaque imaged four consecutive times using increasing TEs, with TR 2000 msec, echo train length 8, and echo spacing 14. The TEs are 84, 98, 112, and 126 msec. Note that the MS plaque posterior to the C-2 vertebral body is most visible at intermediate TEs rather than prolonged TEs.

lesion, which was barely visible on T2-weighted CSE images.

This observation suggests that pathologic changes within the spinal cord may be missed with the use of single echo (T2-weighted) sagittal FSE sequences. This is not attributable to resolution, because the pixel size used in FSE sequence was smaller than the one used in CSE sequence. Because the lesion was barely visible on the T2-weighted CSE sequence, it was probable that its T2 relaxation time was short. There are theoretical reasons that small objects (two pixels in size), especially those with short T2 relaxation time, may be lost on FSE sequences (7). But this loss of small objects' contrast is found to be more prominent on proton-density (short effective echo time [TE]) sequences (7). Nevertheless, because the lesion disclosed in our observation was larger than two pixels in size and was visible on proton density–weighted CSE sequence, it was possible that dual-echo FSE imaging could have disclosed the lesion. In conclusion, single-echo (T2-weighted) sagittal FSE sequences do not seem to be as accurate as conventional dual-echo sequences in the detection of intramedullary lesions. When such lesions are suspected, it is better to use dual-echo conventional or fast long-repetition-time (TR) sequences.

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Comment

Dr Miaux has presented a case in which T2-weighted (proton density not performed) FSE sagittal images showed no spinal cord lesion, but proton-density CSE sagittal images and gradient-echo axial images demonstrated a hyperintense lesion consistent with a plaque.

Several issues are pertinent here. First, very heavily T2-weighted images can be insensitive to small lesions of the cord (G. Sze, "Fast Spin-Echo Imaging of the Spine," presented at the annual meeting of American Society of Neuroradiology, St Louis, Mo, 1994; G. Sze, "Optimization of Fast Spin Echo Parameters," presented at the annual meeting of the Society of Magnetic Resonance, San Francisco, Calif, 1993). Although the use of FSE gives the ability to obtain extremely heavily T2-weighted images routinely with superb myelographic effect, for example, with an effective TE of 150 or 160 msec, we do not use them because such heavily T2-weighted images do not show cord lesions very well ("Fast Spin-Echo," 1994;

"Optimization of Fast Spin Echo," 1993). As an illustration of this concept, Figure 2 shows a patient with an MS plaque studied with multiple TEs (84, 98, 112, and 126 msec). As you can see, the plaque is less visible at very long TEs.

I believe that a similar mechanism was at work in the sagittal T2-weighted FSE image in this case. The parameters (4000/128) are heavily T2 weighted. Typically, for spine imaging in which we suspect cord lesions, we try to use a TR of 2000 to 3000 and a TE of less than 110. I believe that the lesion would have been seen with this sequence.

Second, our protocols always include a proton-density, as well as a T2-weighted, sagittal sequence if a cord lesion is suspected. This, of course, follows the example of routine brain imaging. Confirmation of a lesion on an alternatively weighted sequence is always useful. In this case, the authors used a CSE proton-density image to confirm the existence of a lesion. It is virtually certain that if they had performed a proton-density FSE image they would have seen the lesion as well.

The general acceptance of FSE makes it more important for users to understand any new influences that the routine parameters, such as TR and TE, may have, and to explore the new parameters, that is, echo spacing and echo train length. Optimization of all parameters can be important for lesion detection and for image optimization (1).

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Reference

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The Potential of Magnetization Transfer MR to Differentiate Fibrillary Gliosis from Fibrous Gliosis

Magnetization transfer contrast is a recently introduced method of creating a new tissue contrast in MR imaging (1–3). It is based on the transfer of magnetization between free protons in water and bound protons associated with macromolecules such as proteins. In a magnetization transfer sequence, an off-resonance pulse is applied before a standard imaging sequence to saturate the magnetization of bound protons that have a broad resonance peak (because of their immobility) (1), and the effect is signal suppression in all tissues to varying degrees in relation to macromolecular structure, water content, and efficiency of the protein/water interactions.

In the September 1994 issue of *AJNR*, there are two interesting papers on the potential of magnetization transfer to estimate the age of cerebral infarcts (4) and the age of MS lesions (5). In the final stages of infarction, fibrillary gliosis develops, and there is relatively little protein-bound water remaining and more mobile protons than in the acute stages of infarction. This results in lesser magnetization transfer effect in the chronic stage than what occurs in the acute stage (4). In the chronic stages of MS lesions, fibrous gliosis develops, resulting in higher values of magnetization transfer effect than what occurs in younger lesions (5).

Because the two papers were originating from the same radiology department and used the same MR 0.1-T unit, I concluded too rapidly that fibrillary gliosis and fibrous gliosis were two opposite "pathologico-anatomo-radiologic" entities concerning the magnetization transfer effect values. These differences could be related to the use of different sequences; in the first paper (4), the sequence used was a gradient-echo sequence, and in the second paper it seems to be a spin-echo sequence (5). The TR and TE were identical (1700/30), and the parameters for the off-resonance pulse also seemed to be identical (offset and duration). Nevertheless amplitude was 0.35 G in the first paper and 0.35 μT (0.0035 G) in the second, but I think that these differences in amplitudes were probably attributable to a typing error.

I would like to ask some questions:

Is fibrillary gliosis so different from fibrous gliosis?

Are the sequences used in the two papers actually different, or are they probably both gradient-echo sequences as in other studies (3, 6)?

If the sequences used are identical and fibrillary gliosis not very different from fibrous gliosis, how do we explain the very different values of magnetization transfer effect in the chronic stages of MS lesions and infarction?

Could these differences be attributable to the influence of water content?

In summary, I took out a subscription to *AJNR* in the hope of getting some transfer of knowledge, and I am a little disappointed concerning magnetization transfer, gliosis, and their relationships.

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Reply

Dr Miaux is correct that the sequences in the two studies were identical gradient-echo sequences with an amplitude of 0.35 G. The terms *fibrillary gliosis* and *fibrous gliosis* describe the same pathologic entity, and we did not mean to imply a difference. Dr Miaux nevertheless raises an interesting question: whether magnetization transfer effect (MTe) can distinguish different cellular processes, such as tumor from infarction. The answer unfortunately seems to be no. We have previously reported our experience that the MTe values of different pathologic processes overlap significantly and do not appear to aid in differentiation (J. M. Prager, J. D. Rosenblum, R. E. Halbach, S. Verma, S. Mojtahedi, R. G. Ramsey, "Magnetization Transfer Contrast in Pathologic Conditions at 0.1T in the Human Brain," presented at the annual meeting of the Radiological Society of North America, 1990). The trends we most recently reported in MTe values were in opposite directions for infarction and for MS. We believe this is attributable to changes in free water content related to the different pathologic processes in each disease process. With infarction, the changes in MTe most likely represent changes in water content of the tissue. In areas of early infarction, there is an inflammatory response with an influx of inflammatory cells that may transiently decrease the relative proportion of free water, whereas in the chronic stage of encephalomalacia, the area of abnormality is predominantly free water. With MS, the primary difference between normal and abnormal areas is a lack of myelin with an increased number of astrocytes. To reiterate, changes in MTe are not tissue specific but reflect the changes in water content with the evolution of the pathologic process. Our intent was to report the use of MTe to assess the age of specific pathologic processes. We hope this additional knowledge transfer will resonate well with Dr Miaux.

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Radiation Dose to the Lens from Cerebral Arteriography

In a recent article in *AJNR*, Professor Bushong recommends pulse-progressive fluoroscopy with freeze-frame imaging in all newly installed neuroangiography equipment to minimize radiation dose to patients and operators (1). We would like to report the entrance skin dose over the lens from angiography performed with such equipment, in 22 patients with subarachnoid haemorrhage.

Lithium fluoride 4.5 × 0.9-mm thermoluminescent dosimeters (TLDs) were used for dosimetry, after initial annealing and batch calibration. A TLD packet was taped over each closed upper eyelid at the beginning of the angiogram and read at the end, using a Toledo 654 reader (Vinten Instruments, Weybridge, United Kingdom). The entrance skin dose = (TLD reading − background reading)/batch calibration factor.

The angiography equipment was an IGE Advantx/DX Hiline (General Electric CGR, Paris, France) biplane screening unit. Pulse-progressive fluoroscopy was always used. The image-intensifier entrance-dose measurement during fluoroscopy is 550 mGy/s (manufacturer's data). During image acquisition, the operator controls the peak kilovoltage, milliamperes, and dose. The Advantx calculates an exposure time for the selected dose. The DX Hiline sets the aperture opening. All angiograms were acquired with digital subtraction angiography on dose setting C, which is 0.0176 mGy per exposure (2.02 mR per exposure, manufacturer's data).

There were 7 male patients (mean age, 43 years; range, 27 to 60 years) and 15 female patients (mean age, 49 years; range, 17 to 68 years). Nine patients had three-vessel angiography, and 14 patients had four-vessel angiography. Table 1 gives the fluoroscopy time and number of exposures. Table 2 gives the results of dosimetry.

We found the entrance skin dose over the lens was much lower than previously reported for conventional angiograms: 600 to 700 mGy (2); 174 ± 36 mGy (3); and 318 mSv at the glabella (4). As in other studies, the right TLD chip always read higher because this is the side of the lateral tube. One factor we did not quantify was the effect

TABLE 1: Fluoroscopy time and number of exposures

	Mean	Standard Deviation	95% Confidence Interval for the Mean
PA screening time, min	11.2	7.0	8.2 to 14.2
Lat screening time, min	3.3	5.5	0.8 to 5.9
Number of PA images	78	21	68 to 86
Number of lat images	49	17	41 to 56

Note.—PA indicates posteroanterior and lat, lateral.

TABLE 2: Dosimetry results

	Mean Lens Dose, mGy	Standard Deviation	95% Confidence Interval for the Mean, mGy
Right	30.9	27.2	19.1 to 42.6
Left	10.2	4.1	8.4 to 12.0

of the lateral tube-intensifier distance, which is normally 130 cm but can be brought in to 95 cm.

We agree with Professor Bushong that staff training is vital for proper radiation protection and suggest that lens dosimetry may be a useful method for radiologic audit.

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Comment

The casual reader may not appreciate the time, effort, and attention to detail that is required to obtain the type of patient dose data reported by Maclennan, Hadley, and Sloss. I believe their use of thermoluminescent dosimetry is the most appropriate way to measure such dose, although the use of an internal transmission ionization chamber is easier and becoming more available.

We need more data like those reported by these authors to allow sounder judgments to be made on what represents optimum use of radiation in these procedures. The lens doses reported ranged to a maximum of approximately 45 mGy. This is clearly an acceptable dose level, matching that observed in computed tomography (CT) and orders of magnitude less than that suspected to be cataractogenic.

Other investigators should be encouraged to measure and report their results in a similar fashion so that a more representative and complete database of patient entrance skin exposures and organ doses can be assembled. To develop more sophisticated dose optimization procedures, such data are essential.

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Differentiating Cerebral Lymphoma from Nonmalignant Central Nervous System Lesions in Patients with AIDS

The accurate differentiation of infectious from malignant cerebral lesions in patients with acquired immunodeficiency syndrome (AIDS) is important for the determination of appropriate therapy. Primary central nervous system (CNS) lymphoma and toxoplasma encephalitis may have similar findings on CT and MR. Ruiz et al in the November issue of *AJNR* (1) demonstrate the accuracy of thallous chloride Tl 201 (Tl-201) brain single-photon emission CT (SPECT) imaging in differentiating cerebral lymphoma from toxoplasma encephalitis. The 12 patients who had CNS lymphoma had abnormal Tl-201 accumulation, and the 25 patients who had toxoplasmosis or tuberculosis had negative Tl-201 studies.

We have shown that primary CNS lymphoma is hypermetabolic on fludeoxyglucose F 18 (FDG) position emission tomography (PET) imaging (2). The accumulation of FDG in primary CNS lymphoma is similar to that of anaplastic gliomas and significantly greater than that of low-grade astrocytomas. We subsequently reported the results of FDG PET imaging in 11 subjects with CNS lesions and AIDS (3). The lesions included lymphoma (n = 5), toxoplasmosis (n = 4), syphilis (n = 1), and progressive multifocal leukoencephalopathy (n = 1). Qualitative and semiquantitative analysis demonstrated that the patients who had lymphoma had greater FDG accumulation than subjects with nonmalignant lesions. Thus, FDG PET imaging also is highly accurate in the differentiation of malignant from nonmalignant lesions in patients with AIDS.

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Reply

Drs Hoffman and Coleman's comments are welcome. Unfortunately, although FDG PET imaging seems to be accurate in the differentiation of malignant versus nonmalignant brain lesions in patients with AIDS (1), the cost of this procedure and the fact that it is not available to many medical centers dealing with AIDS patients limit its utility.

Unless FDG PET imaging becomes more available and less expensive (2), Tl-201 brain SPECT will have a greater role in the differentiation between primary brain lymphoma and CNS infection in AIDS patients, because Tl-201 brain SPECT can easily be done in any hospital with a dedicated triple-head gamma camera (3).

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Midbrain Lesions and Third-Nerve Palsy

I had the pleasure of reading a recent Radiologic-Clinical Correlation in *AJNR* by Shuzo Shintani (1). Although the lesion demonstrated on CT scan and MR imaging certainly involves the region of the third nerve and surrounds, the explanation for the eye movement abnormality illustrated is incorrect.

A nuclear third-nerve lesion causes an ipsilateral third-nerve palsy with ptosis and a contralateral superior rectus palsy and ptosis. Nuclear third-nerve palsies, therefore, cause bilateral ptosis and a contralateral superior rectus palsy. Fascicular lesions, on the other hand, cause ipsilateral third-nerve palsies including ptosis; however, the contralateral superior rectus is not involved. In their patient, they illustrated unilateral ptosis, which is ipsilateral to the lesion and as well, an ipsilateral third-nerve palsy. This is correlated with the lesion on MR imaging which involves the ocular motor fascicles. To explain a contralateral upgaze deficit without concomitant ptosis on the basis of involvement of the ocular motor nucleus is anatomically incorrect. Although they do not show an eye movement photograph in the action of the contralateral inferior oblique, they make a blanket statement that elevation of that eye was limited. I propose that the elevation deficit is therefore caused by pressure from brain stem swelling on the tectum. It would have been interesting to know what the pupillary examination findings were in regard to light near dissociation and whether there was any convergence retraction nystagmus on attempted up-gaze.

I would ask that the author address this discrepancy. Thank you.

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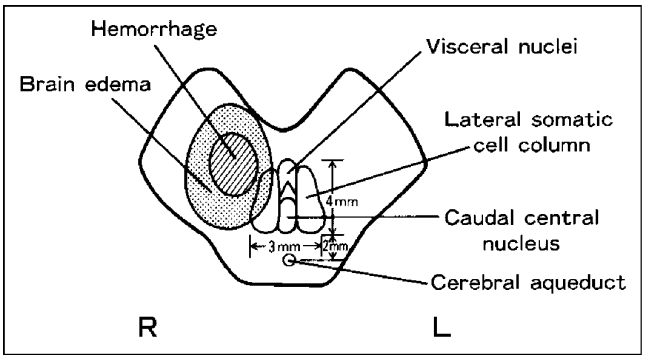

Fig 3. Schema of location of column and nuclei in the oculomotor nuclear complex. This complex consists of visceral nuclei, a caudal central nucleus, and a lateral cell column innervating the pupillary sphincter muscle, eyelid elevating muscle, and extraocular muscles, respectively. The brain edema around the hemorrhage transiently involved the right lateral somatic cell column but spared the caudal central nucleus.

Reference

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Reply

We thank Dr Strominger for his careful review of our manuscript. He provides important information about the eyeball movements associated with midbrain lesions. We agree with his opinion that nuclear third-nerve palsies cause bilateral ptosis and a contralateral superior rectus palsy, and fascicular lesions cause ipsilateral third-nerve palsies including ptosis. However, the contralateral superior rectus is not involved.

In our patient, the *hemorrhage* itself involved the distal portion of the right oculomotor fascicles and spared the oculomotor nucleus. However, as shown in Figure 3B in our article, brain *edema* around the hemorrhage transiently involved the oculomotor nuclear complex. The anatomic distribution of subnuclei of the oculomotor nuclear complex (1) suggested that the brain edema involved the right lateral somatic cell column innervating extraocular muscles including the left superior rectus, but spared the caudal central nucleus innervating the levator palpebrae (Fig 3 here). This resulted in the contralateral (left) superior rectus palsy without concomitant contralateral (left) ptosis in our patient. When the patient was discharged, the left superior rectus palsy had resolved.

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Editor's note. Preceding letters were forwarded to Dr Louis Caplan. His comments follow.

Comment

MR has facilitated identification and location of lesions within the midbrain during life. Clinical-imaging correlation of midbrain lesions has taught clinicians about various functions including those of the third nerve. The take-home message for clinicians from the article of Shintani et al and the correspondence between the authors and Strominger is that lesions of the midbrain tegmentum that involve the oculomotor nucleus result in clinical signs different from lesions of the base that affect the fascicles of the nerve.

Recent studies, in addition to those of Shintani, indicate that most patients with mesencephalic hematomas or infarcts (1) involving the middle portion of the midbrain on one or both sides have oculomotor dysfunction. When the tegmentum is affected on one side, inclusion of the third nerve nucleus leads to a complete third-nerve palsy on the ipsilateral side and ptosis and superior rectus weakness of the contralateral eye. The two eyes work synkinetically in looking up and in eye opening so that yoking these functions together makes physiologic good sense. This is done at a nuclear level as well as a supranuclear level in the region of the posterior commissure.

The fascicles of the third nerve spread out rather widely in the midbrain base before exiting at the most medial extreme of the cerebral peduncle. If all of the fascicles are included or compressed by the lesion, then a unilateral complete third-nerve palsy results, but the contralateral eye remains normal. Because the fascicles are separated, some patients have had just involvement of the fascicles to the superior division of cranial nerve III affecting the levator palpebra superioris and the superior rectus, or involvement of the inferior division affecting the inferior and medial recti, the inferior oblique, and the iris sphincter (2–4). Some of these muscles and functions can be involved in isolation by small lesions (4). When the midbrain base is involved, especially in its dorsal and medial portions, then a hemiparesis and/or ataxia and incoordination of the contralateral limbs accompanies the third-nerve palsy. When the lesions are ventral and lateral, the third-nerve palsy often is isolated (1).

Knowing the anatomy of the area can help clinicians define the type of lesion and its location.

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Retained Sponge in the Neck: MR Appearance

A 53-year-old man presented with fever, soft-tissue swelling, and tenderness on the right side of the neck 3 weeks after surgery for larynx carcinoma. Ultrasonography demonstrated a highly echogenic mass with strong acoustic shadowing in the right posterior cervical region. MR examination (0.5-T Philips, Gyroscan) revealed a 2.5 $\times$ 3 $\times$ 4-cm heterogenous mass in the posterior cervical triangle. On T1-weighted images, the mass was of low signal intensity and showed peripheral enhancement after contrast administration (Fig 4 A,B). On long-TR images, the mass remained relatively hypointense centrally, and the peripheral zone was hyperintense (Fig 4C). Because the clinical condition was highly suggestive of abscess, the patient again underwent surgery, and a 7.5 $\times$ 7.5-cm sponge with surrounding inflammation was found.

Retained surgical sponges and towels are uncommon complications of surgery. They remain clinically silent; however, they can become evident as an inflammatory syndrome with or without abscess formation. Retained sponges may induce two types of foreign body reactions: an aseptic fibrinous response that creates adhesions and encapsulation, resulting in a foreign body granuloma; and the exudative response that leads to abscess formation with or without secondary bacterial infection (1). The retained sponges or towels may mimic tumor, pseudocyst, or hematoma, and the diagnosis may be difficult.

MR appearance of retained sponges in the pelvis, retroperitoneum, chest, and paraspinal region has been reported (1–4). A case with a retained sponge in the neck, reported from our institution, did not have MR examination (5). The minimum time elapsed since surgery was 2 years in three patients (1–3). The remaining case was a patient who was operated on 6 weeks before the examination in

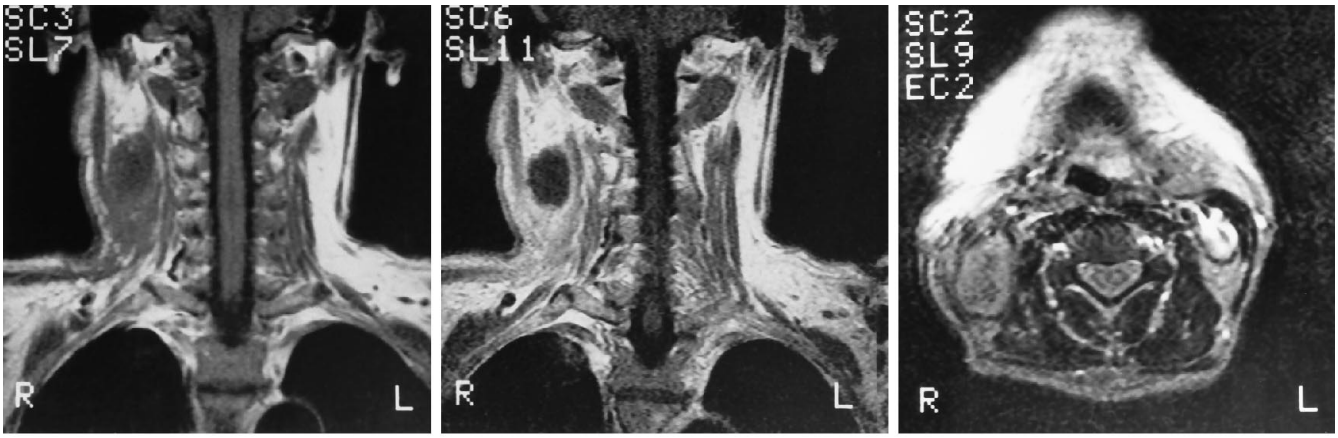

Fig 4. T1-weighted (450/20) coronal images before (*A*) and after (*B*) contrast administration demonstrate a hypointense mass with peripheral contrast enhancement.

C, T2-weighted (1900/80) axial image shows central hypointensity representing the retained sponge with surrounding hyperintense rim of inflammation.

which gelatin sponge, used for hemostasis, remained unabsorbed and mimicked residual or recurrent tumor (4).

In our case, the periphery of the mass was hyperintense on long-TR images and showed contrast enhancement (Fig 4 B, C), as in the case of Hoeffner (4). This finding differs from those of others (1, 3); they described a central area of increased signal intensity representing necrosis. The difference in time interval between surgery and the MR examination may explain the variation in the appearance. At the early stage, as in our case and in Hoeffner's patient, the peripheral hyperintensity on T2-weighted images probably represents the surrounding inflammation and the central area of low signal intensity, the retained sponge (Fig 4C). The central hypointensity is unlikely to be an abscess in the absence of air. However, in the long term, the fibrinous response and encapsulation take place, which may result in peripheral low signal intensity on all sequences (1, 3), and the necrotic center may show increased signal on T2-weighted images.

We conclude that retained sponges may have different MR appearances depending on the time between the surgery and the MR examination. In the early postoperative period, the retained sponge may be seen as a mass of heterogenous low signal intensity with surrounding hyperintensity on T2-weighted images. The absence of central T2 hyperintensity should suggest a retained foreign body, namely a sponge, rather than an abscess in the surgery site.

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