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## **MR imaging of the cranial meninges with emphasis on contrast enhancement and meningeal carcinomatosis.**

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# MR Imaging of the Cranial Meninges with Emphasis on Contrast Enhancement and Meningeal Carcinomatosis

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MR imaging was used to investigate normal and abnormal meningeal enhancement, with an emphasis on meningeal carcinomatosis. Three groups of patients were studied on a 1.5-T system. In group 1, the normal meninges were examined in 20 patients and were found to show fine linear enhancement in short segments, especially in a parasagittal distribution. In group 2, all gadolinium-enhanced head scans were reviewed retrospectively. Abnormal meningeal enhancement was detected in 52 patients. In some of these, the enhancement was associated with pathologic conditions of the meninges, including leptomeningeal tumor and meningeal infections and other inflammatory conditions; in others the enhancement was adjacent to subdural hematomas, subacute infarcts, and skull lesions, such as metastases or postoperative defects. In group 3, 30 cases of meningeal carcinomatosis were studied prospectively. Enhancement was seen in approximately two-thirds of cases and usually was quite diffuse and applied to the inner table of the skull. Frank nodules were seen less often. Contrast-enhanced CT was equal to MR in the detection of nodules but was nearly always unable to show diffuse meningeal enhancement against the inner table of the skull.

Contrast-enhanced MR was more sensitive than contrast-enhanced CT in the examination of normal and abnormal meninges. Abnormal findings, such as meningeal carcinomatosis, were demonstrated more often by MR than by CT.

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Recent presentations [1-3] and anecdotal reports have discussed the sensitivity of gadolinium-enhanced MR imaging in the evaluation of suspected meningeal disease. Although meningeal disease can be difficult to evaluate on noncontrast MR [4, 5], after the administration of gadopentetate dimeglumine (gadolinium-DTPA), enhancement of meningeal processes is often easily detectable. The purpose of this article was threefold: (1) to establish the extent of normal enhancement of the meninges; (2) to document under which conditions abnormal meningeal enhancement might be seen; and (3) to test the sensitivity of meningeal enhancement in the detection of disease, especially with respect to meningeal carcinomatosis.

## Subjects and Methods

Three groups of patients were studied. In group 1, 20 patients without known primary meningeal or systemic disease were evaluated prospectively. These patients had nonspecific symptoms and signs, such as headache or seizure, and were not thought clinically to have processes that could cause meningeal disease. In addition, none of these patients had a previous history of skull disease or major trauma. The noncontrast and contrast-enhanced MR scans of these patients were interpreted as normal, except for a few small foci of high signal in the white matter in some patients. These foci did not enhance and were considered nonspecific in nature. Most of these patients were studied with the phase-encoding gradient horizontal and the frequency-encoding gradient vertical. However, a few patients were studied with both axes as described, and then with the direction of the frequency-encoding gradient switched.

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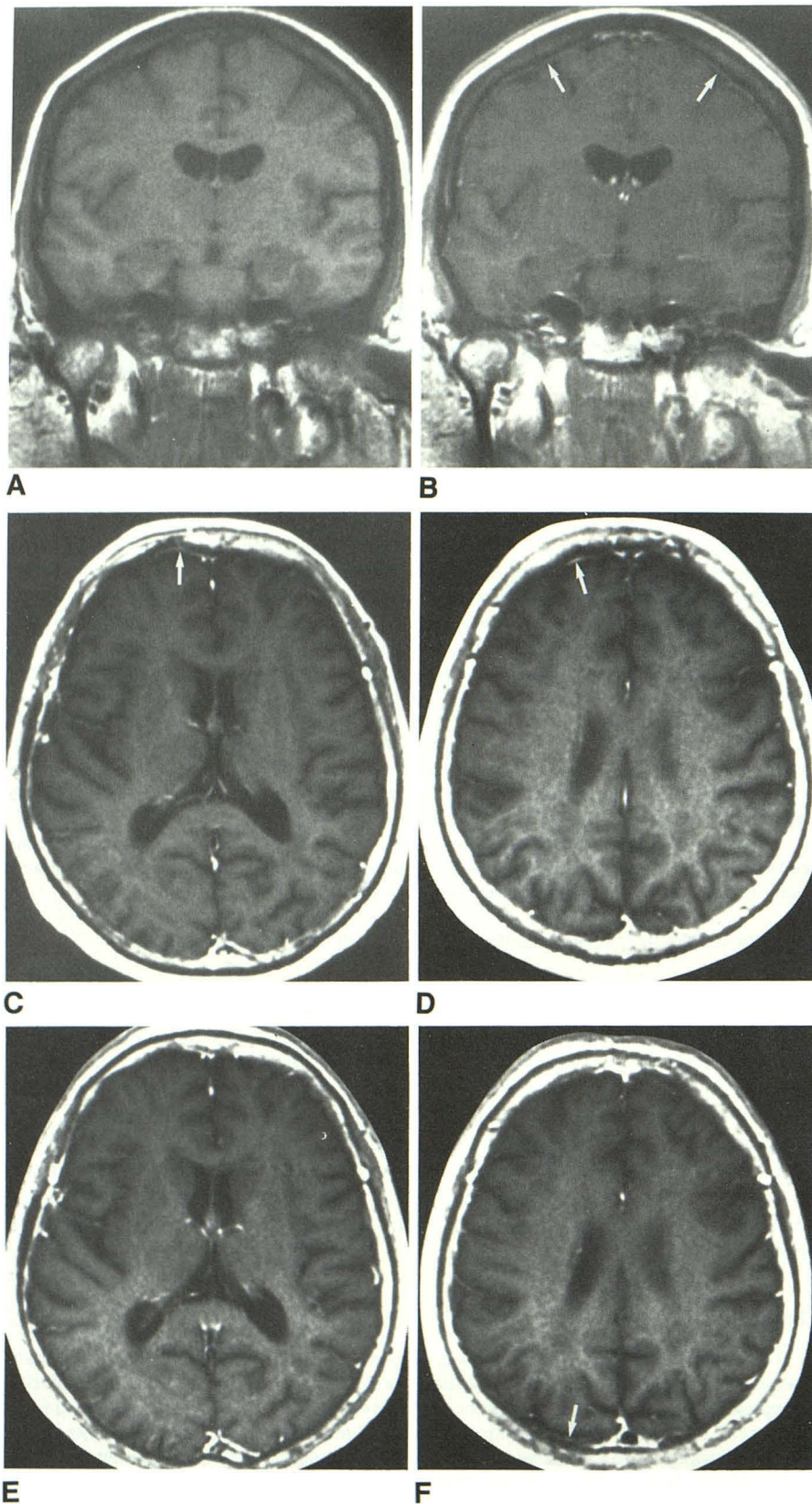


Fig. 1.—Normal meningeal enhancement. Note that short segments of meninges can be detected (arrows).

A and B, Short TR (600/20) coronal scans pre- (A) and post- (B) contrast.

C and D, Normal short TR (600/20) axial scans postcontrast, with frequency-encoding gradient oriented from inferior to superior. Enhancing meninges are better seen anteriorly than posteriorly.

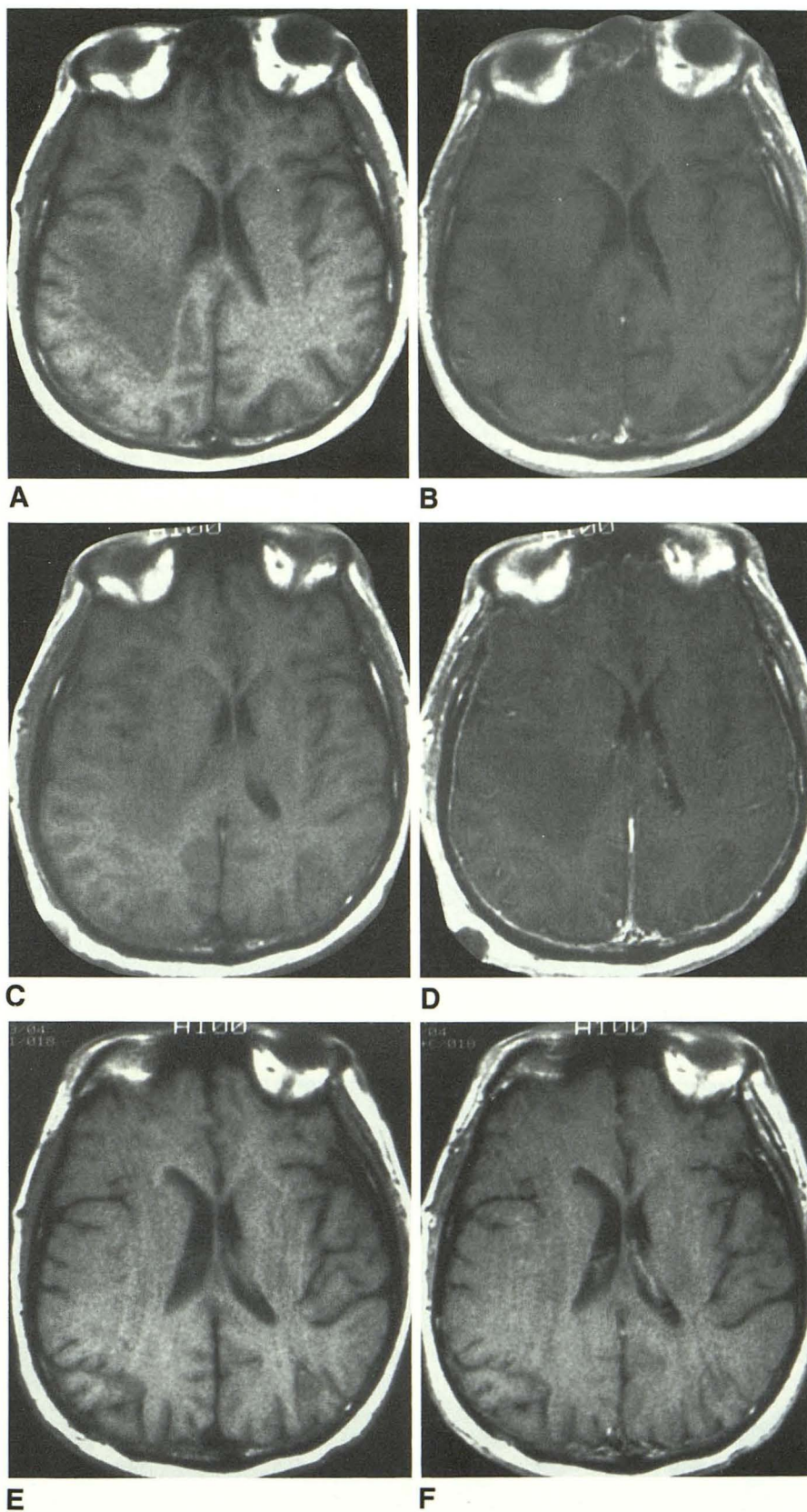
E and F, Normal short TR (600/20) axial scans postcontrast, with frequency-encoding gradient direction reversed. Note that marrow fat is now shifted inferiorly, revealing enhancing meninges better posteriorly.

Fig. 2.—60-year-old patient with lung carcinoma and a single brain metastasis.

A and B, Pre- (A) and post- (B) contrast short TR (400/20) axial scans before resection of metastasis. Little abnormal meningeal enhancement is seen.

C and D, Pre- (C) and post- (D) contrast short TR (400/20) axial scans 1 week after removal of metastasis show diffuse meningeal enhancement surrounding entire brain.

E and F, Pre- (E) and post- (F) contrast short TR (400/20) axial scans 4½ weeks postoperatively show resolution of previously noted meningeal enhancement. This patient was not found to have carcinomatous meningitis, and temporary diffuse meningeal enhancement was presumably reactive.





In group 2, all gadolinium-enhanced MR head studies were reviewed, and the scans of 52 patients in whom abnormal enhancement of the meninges was seen were studied retrospectively. The results of the first group of patients were used as the normal control. Specific attention was paid to the amount and pattern of enhancement and to the underlying disease. Particular care was taken to differentiate marrow fat in the cranial vault from enhancing meninges. Patients with meningiomas were excluded.

In group 3, 30 patients with suspected meningeal carcinomatosis were evaluated prospectively. These patients all had known primary or systemic tumors and had symptoms or signs localized to many regions of the neuraxis. The MR studies were correlated with other results. Three subgroups of patients were studied separately: (A) patients with positive CSF cytology with or without suggestive CT findings; (B) patients with typical contrast-enhanced CT findings of enhancing leptomeningeal tumor in whom the CSF was not examined or the CSF evaluation was negative; and (C) patients with a suggestive clinical picture associated with elevations of CSF protein but negative CSF cytology and no CT findings of leptomeningeal tumor.

CSF was obtained from a lumbar approach. Ten to fifteen milliliters of CSF were withdrawn at each examination. All patients in the third subgroup had lumbar punctures at least three different times in an effort to detect malignant cells in the CSF.

Imaging was performed on a superconductive magnet operating at 1.5 T. All sections were 5 mm thick, with an intersection gap of 1–2.5 mm. Short-TR, 400–600/20 (TR/TE), and long-TR, 2000/35,70, sequences were performed before the administration of contrast material in all patients. Gadolinium-DTPA (Magnevist, Berlex Laboratories, Wayne, NJ) was given in a concentration of 0.1 mmol/kg IV at a rate of approximately 15 ml/min. After the administration of contrast material, patients in groups 1 and 2 were studied with short TR scans only. The first three-fourths of the patients in group 3 were studied with short TR, long TR, and short TR acquisitions in that order. The final one-fourth were studied with short TR scans only. The matrix was  $256 \times 256$  in all cases. Two excitations were usually used for short-TR acquisitions, while one excitation was used for long TR-acquisitions. However, three patients in group 1 were studied with only one excitation during the short-TR acquisitions. In these

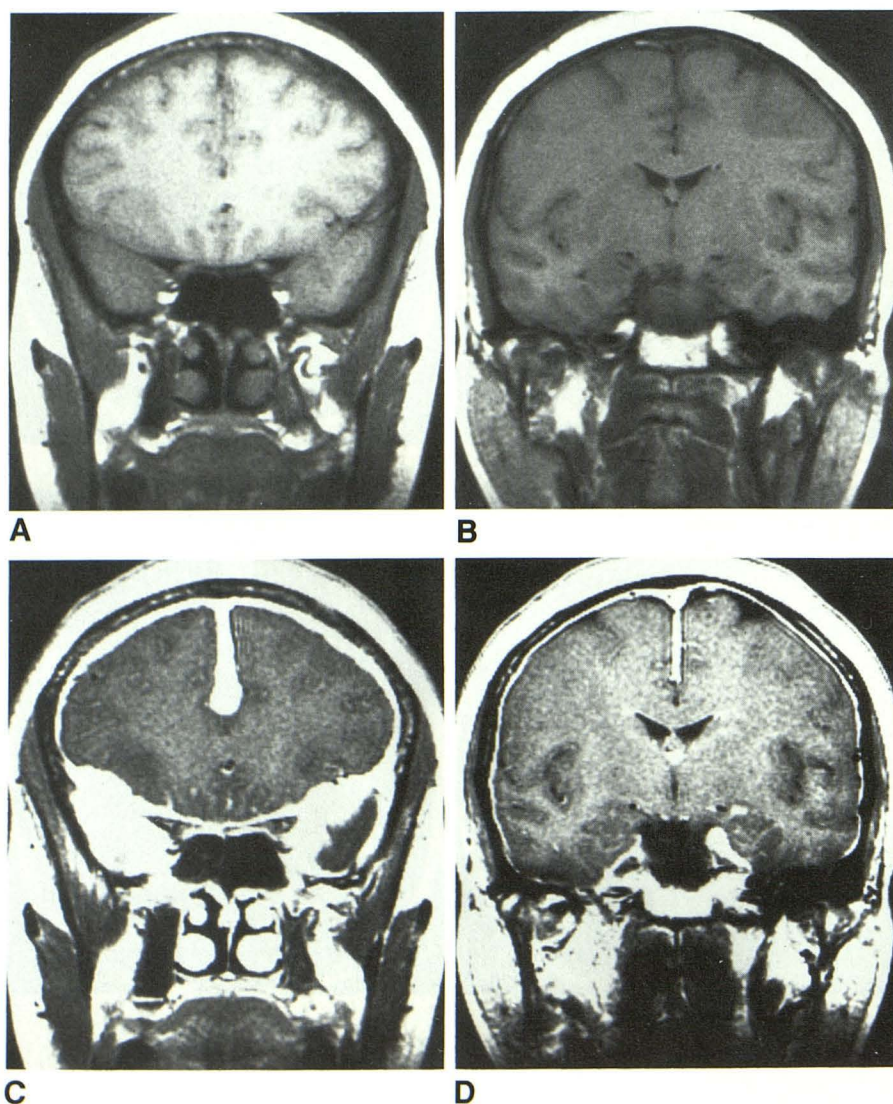


Fig. 3.—46-year-old patient with a 12-year history of sarcoidosis and 3-year history of headaches, treated as tension headaches or migraines.

A and B, Short TR (600/20) coronal scans. Thickening of meninges is difficult to visualize.

C and D, Postcontrast short TR (600/20) coronal scans show marked enhancement of thick meningeal sarcoid granulation tissue coating entire surface of brain.



Fig. 4.—64-year-old patient with posttraumatic subdural hematoma.

A, Short TR (400/20) axial scan shows large collection of subdural blood.

B, Postcontrast short TR (400/20) axial scan discloses marked enhancement of meninges, particularly along outer aspect of hematoma (arrows).

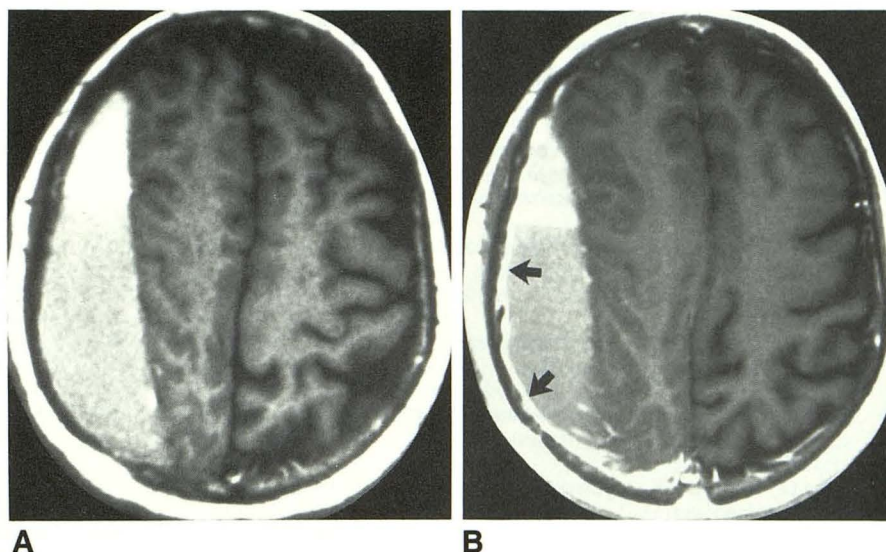
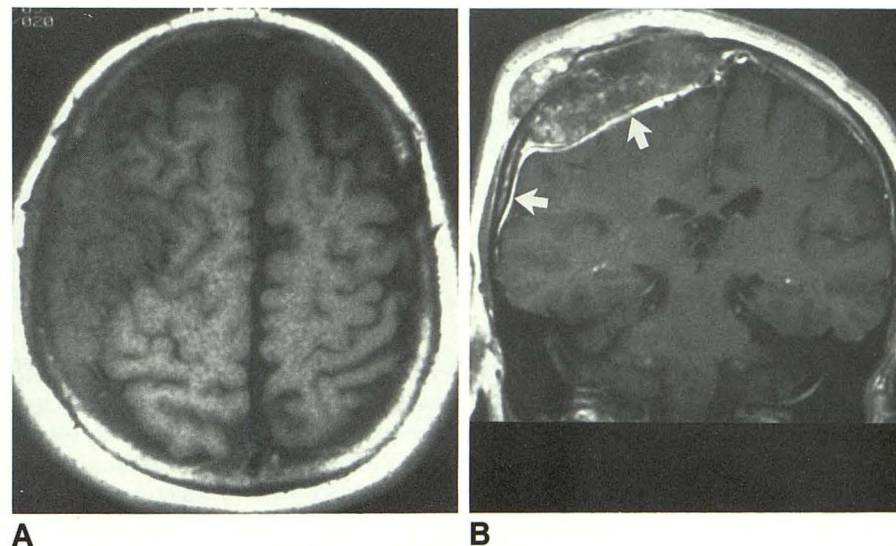


Fig. 5.—60-year-old patient with prostatic carcinoma skull metastases.

A, Short TR (400/20) axial scan shows large bony lesion with considerable mass effect. While underlying brain parenchyma appears to be primarily displaced, some invasion cannot be totally excluded.

B, Postcontrast short TR (400/20) coronal scan discloses enhancement of skull lesion and of underlying meninges (arrows). No evidence of meningeal carcinomatosis was found, and meningeal enhancement is most likely reactive, rather than neoplastic.



cases, contrast material was injected as a bolus as rapidly as possible after prescanning was completed in order to see if greater enhancement could be elicited.

In nearly one-half of the patients, contrast-enhanced CT scans were obtained within 1 week of the other examinations. Contrast-enhanced CT was performed in all except seven patients in group 3. The CT scans were obtained on a high-resolution scanner and consisted of 5- or 10-mm contiguous axial scans after the administration of 150 ml of 60% contrast material IV as a bolus, at a rate of approximately 50–70 ml/min.

## Results

### Group 1

Group 1 comprised 20 patients 11–80 years old. Indications for the MR scans were nonspecific and included seizure, headache, dementia, vertigo, and suspicion of pituitary ade-

noma, infarct, or multiple sclerosis. On unenhanced scans, short segments of the meninges could be detected faintly in most cases on short TR sequences as a thin line of signal mildly hypointense relative to brain. This thin line was visible against the hypointensity of surrounding CSF on one side and the signal void of bone on the other. It was never possible to delineate the entire meninges. On long-TR scans, it was difficult to visualize the meninges.

After the administration of gadolinium, mild enhancement of sections of the meninges was demonstrated in nearly all of the 20 patients (Fig. 1). This enhancement was most marked around the anterior portions of the temporal lobes and in the parasagittal region. Generally, at the convexity of the skull, a thin line of meningeal enhancement laterally would become increasingly visible and thickened as it progressed toward the dural reflections of the superior sagittal sinus. In none of the patients in group 1 could the enhancement be followed around the brain on the axial sections or down the entire convexity

TABLE 1: Summary of Patients with Suspected Meningeal Carcinomatosis

Subgroup/ Case No.	Age	Gender	Diagnosis	Protein	Glucose	Cytology	MR Findings	CT Findings
Positive CSF cytology								
1	11	F	Medulloblastoma	N/A	N/A	+	No abnormal meningeal enhancement	No abnormal meningeal enhancement
2	1½	F	Leukemia	7	65	+	Mild diffuse meningeal enhancement	N/A
3	55	F	Esophageal carcinoma	176	48	+	Several enhancing leptomeningeal nodules; no diffuse enhancement	Several enhancing leptomeningeal nodules
4	61	F	Breast carcinoma	30	120	+	Mild to moderate diffuse meningeal enhancement	No abnormal meningeal enhancement
5	7	M	Medulloblastoma	6	89	+	Moderate diffuse meningeal enhancement	Moderate diffuse and nodular sulcal enhancement
6	62	F	Breast carcinoma, lymphoma	19	60	+	No abnormal meningeal enhancement	No contrast-enhanced CT
7	58	M	Squamous cell carcinoma of head and neck	44	95	+	Several enhancing nodules invading brain	Several enhancing nodules invading brain
8	53	F	Esophageal carcinoma	36	N/A	+	No abnormal meningeal enhancement	Possible perimesencephalic enhancing nodule
9	59	F	Breast carcinoma	30	43	+	No abnormal meningeal enhancement	N/A
10	56	F	Lymphoma	27	45	+	Mild to moderate diffuse meningeal enhancement	No abnormal meningeal enhancement
11	53	F	Lymphoma	29	65	+	No abnormal meningeal enhancement	No abnormal meningeal enhancement
12	7	F	Glioblastoma of cord	3300	60	+	Mild diffuse meningeal enhancement	Mild to moderate perivascular, tentorial enhancement and at inner aspect of subdural collection
13	56	F	Leukemia	70	67	+	Large localized lesion invading brain	Infiltrative localized gyral enhancement
14	65	F	Lymphoma	230	67	+	Moderate diffuse meningeal enhancement	No abnormal meningeal enhancement
15	64	F	Lung cancer	53	82	+	Moderate localized meningeal enhancement	Very localized enhancement near posterior falx
16	4	F	Medulloblastoma	N/A	N/A	+(biopsy performed)	Prominent diffuse meningeal enhancement	Prominent perivascular and tentorial enhancement
17	77	M	Lymphoma	276	78	+	Moderate to severe meningeal enhancement along one hemisphere	Moderate to severe localized meningeal enhancement
18	42	F	Burkitt lymphoma	10	94	+	No abnormal meningeal enhancement	No abnormal meningeal enhancement
19	38	F	Lymphoma	256	114	+(atypical cells)	Moderate localized meningeal enhancement	N/A
20	4	F	Medulloblastoma	365	37	+(suspicious cells)	Moderate diffuse enhancement; several enhancing nodules	Several enhancing nodules
21	29	M	Medulloblastoma	265	60	+	Mild localized tentorial and peribrainstem enhancement	Mild localized sulcal and tentorial enhancement
CT positive for leptomeningeal tumor								
22	68	F	Breast carcinoma	N/A	N/A	N/A	Mild to moderate diffuse meningeal enhancement with a few nodules	A few superficial nodules with parenchymal invasion

Table 1 continues



TABLE 1—Continued

Subgroup/ Case No.	Age	Gender	Diagnosis	Protein	Glucose	Cytology	MR Findings	CT Findings
23	4	M	Medulloblastoma	N/A	N/A	N/A	Prominent diffuse meningeal enhancement, associated with extraaxial fluid collections	Prominent sulcal and cisternal enhancement with extraaxial fluid collections
24	70	M	Lung carcinoma	30	70	—	Multiple superficial nodules; moderate gyral enhancement superiorly	Multiple superficial nodules; moderate gyral enhancement superiorly
25	14	M	Brainstem tumor	N/A	N/A	N/A	Mild enhancement of meninges surrounding brainstem	Mild enhancement of meninges surrounding brainstem
26	21	M	Ependymoma	N/A	N/A	N/A	Several small enhancing subarachnoid nodules	Several small subarachnoid nodules seen with iohexol placed within subarachnoid space
27	73	F	Lymphoma	N/A	N/A	N/A	Prominent ependymal enhancement; minimal meningeal enhancement	Prominent ependymal enhancement
28	53	F	Breast carcinoma	N/A	N/A	N/A	Parasagittal mass with enhancement extending along inner table and falx	Parasagittal mass with enhancement extending along falx
Clinical diagnosis and indicative CSF								
29	58	M	Lymphoma	116	24	—	No meningeal enhancement	No meningeal enhancement
30	31	M	Adenocarcinoma of unknown primary	144	95	—	No meningeal enhancement	No meningeal enhancement

Note.—N/A = not available; + = positive; — = negative.

on coronal sections. On sagittal sections, enhancement was clearly visible only near the midline at the dural reflections around the superior sagittal sinus. Occasionally, short segments of enhancement could be seen just anterior and inferior to the tips of the temporal lobes. When gadolinium was given as a bolus and acquisition times were shortened, the extent and intensity of the enhancement of the meninges were increased.

Both with and without contrast material, the meninges were detected more easily anteriorly than posteriorly on most axial sections. With the frequency-encoding gradient in the vertical position, progressing from inferior to superior, the high intensity of the marrow fat was displaced slightly anteriorly owing to chemical shift. When the direction of the frequency-encoding gradient was switched, the meninges were better detected posteriorly (Fig. 1).

#### Group 2

Of the 175 gadolinium-enhanced MR head studies that were reviewed, abnormal enhancement of the meninges was seen on the scans of 52 patients. Twenty-two of the patients were believed to have carcinomatous meningitis; 30 had various other processes. The 22 patients with carcinomatous meningitis are included in group 3. The remaining 30 patients

are discussed here. The enhancement of the meninges in these patients was believed to be greater than that seen in group 1. However, the differences were occasionally subtle. In 24 of the 30 patients, the meninges were better detected more peripherally and laterally than would normally be expected, although no nodularity was seen. Longer continuous stretches could be followed, as compared with the short discontinuous segments in normal patients. Enhancement was generally visible as mild thickening of the meninges, often localized in extent. For example, meningeal enhancement too prominent and continuous to be normal, yet limited to a portion of the meninges covering one hemisphere, could be seen. In four of the 30 patients, diffusely enhancing meninges could be seen progressing around the entire brain on the axial section or down the entire convexity on the coronal section. In two more cases, nodularity of the meningeal enhancement could be demonstrated. This finding could be seen in nonneoplastic conditions, such as infections and other inflammatory conditions, as well as in the leptomeningeal tumors described later. In the most extreme examples, involvement of underlying brain parenchyma was also visible. Enhancement of the gyri was present, usually associated with edema and mass effect on the underlying brain parenchyma.

Excluding meningeal carcinomatosis, etiologies associated with abnormal meningeal enhancement consisted of infec-



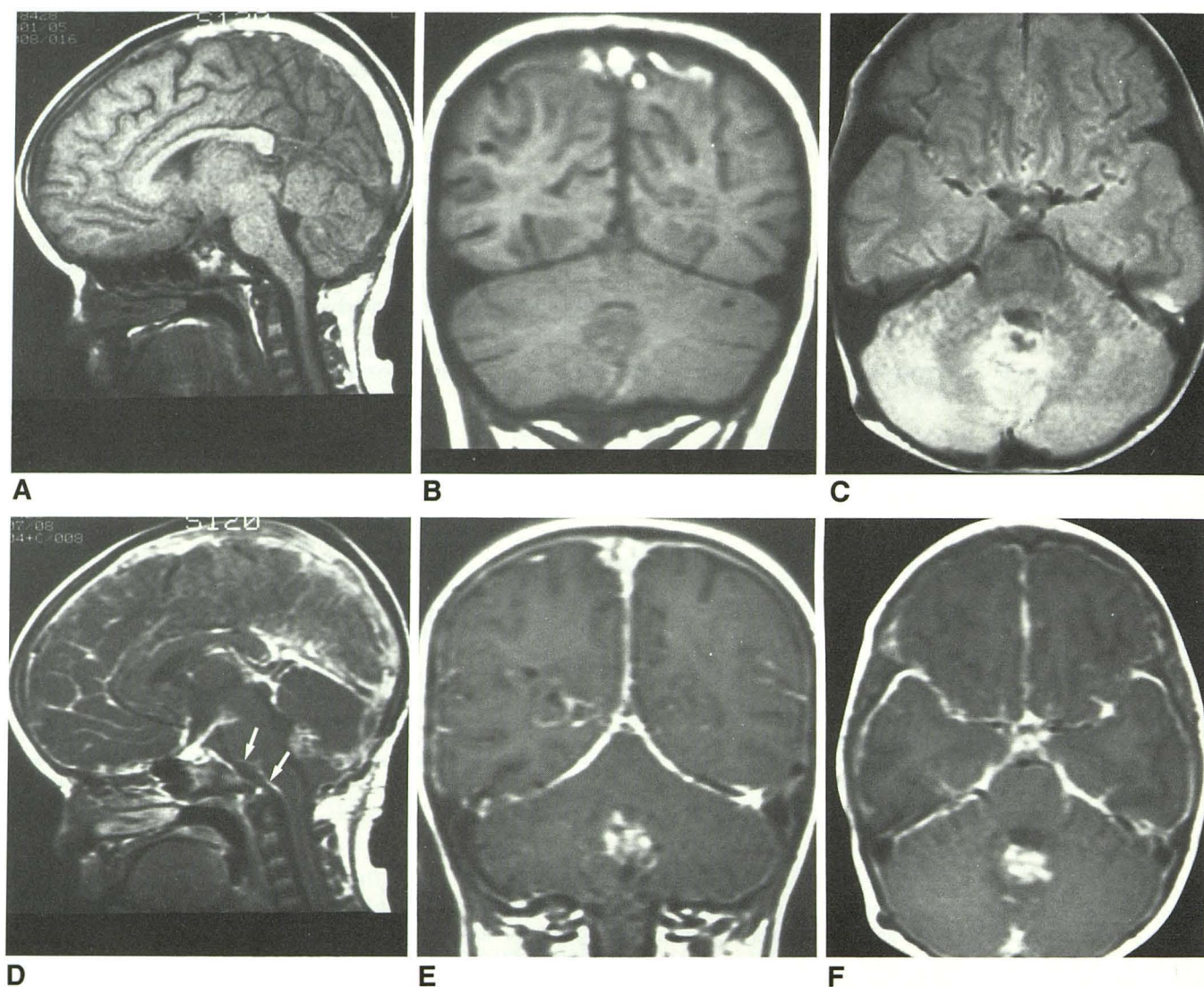


Fig. 6.—4-year-old girl with a history of medulloblastoma, now presenting with headache and cranial nerve palsies. A and B, Short TR (500/20) sagittal (A) and coronal (B) scans do not show substantial abnormality of meninges. Superior sagittal sinus thrombosis and posterior fossa tumor are noted. C, Long TR (2000/30) axial scan discloses mass in posterior fossa. No definite abnormality of meninges is detected. D–F, Short TR (500/20) sagittal (D), and coronal (E), and axial (F) scans show diffuse prominent meningeal enhancement. Note tumor extending along surface of brainstem (arrows).

tious and other inflammatory diseases, traumatic changes, subdural hematomas, posttreatment sequelae, subacute infarcts, and lesions of the overlying skull. In 14 of the 30 patients, changes due to previous trauma or treatment were visualized (Fig. 2). These included postoperative cases, which generally showed localized enhancement at the operative site but occasionally demonstrated diffuse enhancement. In none of these cases was there clinical suspicion of leptomeningeal tumor. In patients who had had radiation and/or systemic chemotherapy alone and not intrathecal chemotherapy, abnormal meningeal enhancement was not seen unless one of the other causes for enhancement listed above was present.

In six of the 30 patients changes were due to infectious or

other inflammatory disease. In four of these, fungal or bacterial meningitis was believed to be the cause. In two of the cases, sarcoidosis was believed to be the underlying disease (Fig. 3).

In three of the 30 patients, enhancement associated with subdural hematomas was seen (Fig. 4). In several other cases, subdural fluid collections that were not clearly hemorrhagic were also associated with enhancing meninges. In both situations, enhancement was nearly always more marked along the side of the fluid collection adjacent to the skull than on the side adjacent to the brain.

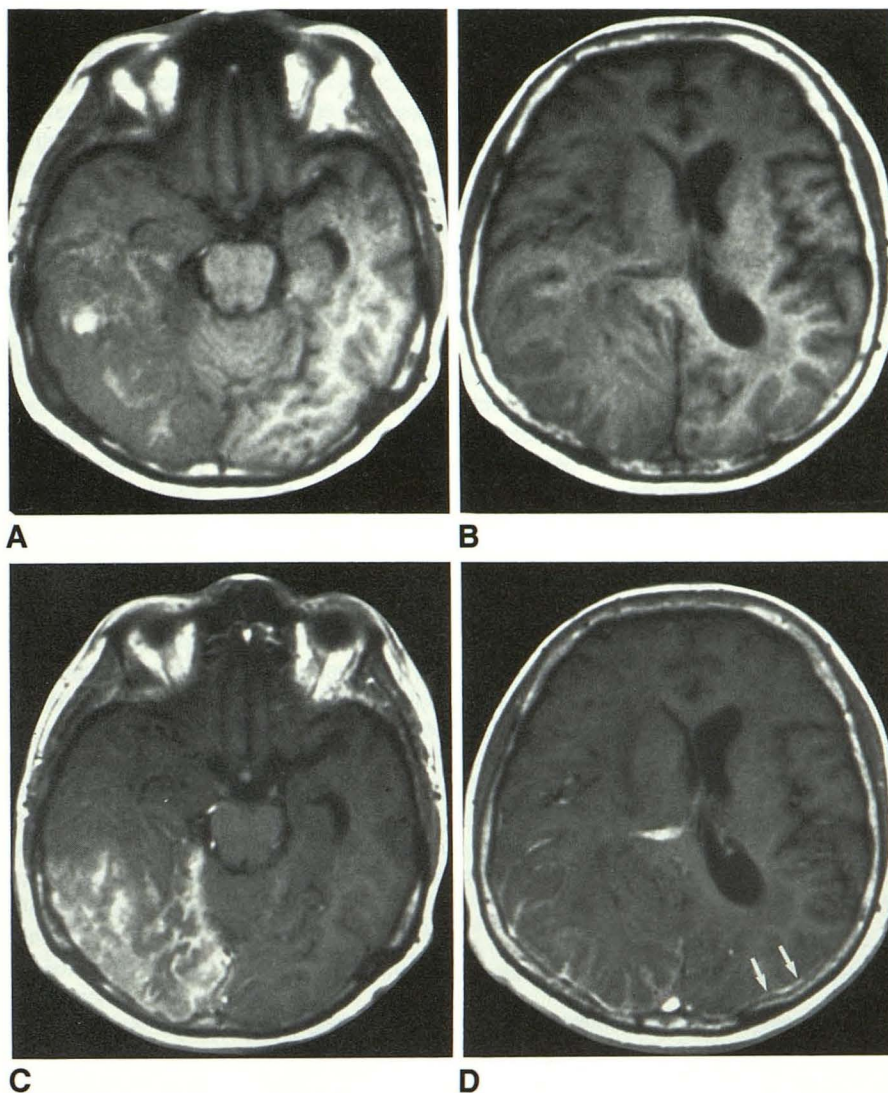
In three of the 30 cases, findings consistent with inflammation of the meninges adjacent to subacute infarct were



Fig. 7.—56-year-old woman with leukemia and positive CSF cytology.

A and B, Short TR (400/20) axial scans disclose area with substantial mass effect, edema, and small foci of hemorrhage.

C and D, Postcontrast short TR (400/20) axial scans show large mass with parenchymal infiltration. Enhancement pattern is still primarily superficial and gyral, although invasion into underlying parenchyma is present. Also note abnormal meningeal enhancement on contralateral side (arrows).



noted. In all of these cases, mild enhancement of the meninges overlying the region of infarct was seen.

In two of the 30 cases, enhancement of the meninges was seen adjacent to skull lesions (Fig. 5); these patients had large metastases to the skull, with impingement on the dura. In one of the 30 cases, mild enhancement of the meninges was detected adjacent to a superficial parenchymal metastasis. Finally, in one of the 30 cases, mild generalized meningeal enhancement was noted in a patient with diabetic neuropathy and elevated CSF protein, without other known cause.

### Group 3

Thirty patients qualified for inclusion in group 3. Twenty-two demonstrated abnormal meningeal enhancement. Comparison of relevant clinical data and MR findings is provided in Table 1. Of the 30 patients, meningeal carcinomatosis was proved by positive CSF cytology in 21. In seven, leptomeningeal tumor was demonstrated on contrast-enhanced CT. In two, presumptive meningeal carcinomatosis was based on suggestive clinical symptoms or signs and elevated CSF protein.

In 15 of the 21 cases with meningeal carcinomatosis proved by positive CSF cytology, enhancement of the meninges was seen. In most cases, this enhancement was mild and demonstrated primarily by sections of meningeal enhancement that were longer than those seen on normal scans. In some cases, diffuse enhancement of meninges surrounding the entire brain could be delineated (Fig. 6). As with subdural hematomas, when subdural effusions were present, enhancement was usually much more striking adjacent to the skull along the outer aspect of the fluid collection, rather than along the inner aspect adjacent to the brain parenchyma. In three of the 15 cases with positive CSF cytology and positive MR findings, nodular deposits of tumor were seen. In two of these three cases, nodular foci were not associated with diffuse meningeal enhancement. In two of the 15 cases, leptomeningeal tumor was demonstrated on contrast-enhanced CT. In two, presumptive meningeal carcinomatosis was based on suggestive clinical symptoms or signs and elevated CSF protein.

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ingeal tumor with parenchymal invasion was detected (Fig. 7). In these cases, edema and mass effect of the underlying parenchyma were prominent.

Of the seven cases demonstrated by previous CT and not by CSF cytology, six did not receive CSF examination. In these seven cases, the CT findings of enhancing tumor, especially sulcal and cisternal enhancement, were believed to be diagnostic of meningeal carcinomatosis. In four of the seven cases, contrast-enhanced MR not only confirmed the CT findings but also demonstrated additional meningeal enhancement against the inner table of the skull. This additional enhancement was prominent and generalized in two cases and mild and localized in two cases.

Of the two patients in whom leptomeningeal tumor was believed to be proved by clinical examination and elevated CSF protein, meningeal enhancement was seen in neither.

There was no definite correlation between CSF protein levels and meningeal enhancement. Unequivocal enhancement was seen in four patients with normal CSF protein. No abnormalities or only a few tumor nodules but no diffuse meningeal enhancement were seen in seven patients with markedly elevated CSF protein.

Cases in which nodular or invasive foci were seen generally were also positive on CT. Cases in which only diffuse meningeal enhancement along the inner table of the skull was seen were nearly always negative on CT. This latter group formed the preponderance of cases. Surprisingly, nodules that were easily seen on CT sometimes showed only mild enhancement on MR at the concentrations of gadolinium used in this study. In two cases, apparently small sulcal nodules could not be seen on MR.

## Discussion

The meninges comprise the dura and the underlying pia-arachnoid [6]. The dura, in turn, consists of two layers. An outer layer, or endosteal layer, is directly apposed to the inner table of the skull and forms the periosteum of the inner table. The outer layer is tightly fused for most of its length to an inner layer, or meningeal layer. These inner layers join to form the falx and tentorium. Between the inner layer of the dura and the arachnoid is the subdural space, which contains a thin film of fluid. Between the arachnoid and the pia is the subarachnoid space, filled with CSF. The arachnoid crosses over the sulci. The pia is closely applied to the surface of the brain and extends into the deepest sulci. The arachnoid and pia together are called the leptomeninges.

In normal meningeal enhancement, enhancement of the meninges is visualized as a thin, markedly discontinuous rim covering the surface of the brain. The enhancement is seen primarily in the dura and venous structures. The arachnoid is thin and avascular. Although vascular, enhancement of the normal, delicate pia is too subtle to visualize. When lesions of the skull occur, as in metastases to the skull or surgical trauma, the dura becomes thickened and fibrotic first. Lesions can also involve the dura alone, as in dural metastases or dural infiltration by sarcoid granulation tissue. These lesions can eventually invade the arachnoid to involve the CSF. When

lesions in the subarachnoid space occur, the pia-arachnoid is affected first. Deposits of tumor can proliferate in the subarachnoid space. When either the pia or arachnoid is thickened and inflamed, both can enhance. For example, enhancement extending down the surface of the brainstem, as in Figure 6, illustrates pathologic changes in the pia alone since the arachnoid is widely separated from the pia at this point. With invasive lesions, such as infections or neoplasms, penetration of the thin pia overlying the surface of the brain occurs. In these cases, the underlying brain parenchyma shows inflammatory or neoplastic changes.

On CT, normal meningeal enhancement is difficult to visualize. When detected, meningeal enhancement is usually due to meningeal carcinomatosis or to meningitis, particularly fungal and tuberculous. In these cases, spaces other than those directly adjacent to the inner table of the skull are often affected. Most often, enhancement is visualized along the vasculature, in the sulci, or in the basal cisterns. Enhancement is seen because it is not obscured by adjacent bony structures.

Because of its superior sensitivity, the introduction of contrast material in MR widens the spectrum of possible etiologies that often cause visible meningeal thickening. While more sensitive, contrast-enhanced MR is no more specific. Any process that causes meningeal irritation can cause meningeal enhancement. In group 2, multiple different etiologies can be postulated for meningeal enhancement. For example, transient enhancement in the postoperative state, as shown in Figure 2, could result from meningeal irritation caused by blood in the subarachnoid space as a result of the craniotomy. Longer-term localized enhancement at the operative site may result from fibrotic meningeal changes. Similarly, fibrotic meningeal deposits may be the reason for prominent meningeal enhancement often seen adjacent to subdural hematomas or other extraaxial fluid collections. This finding is especially common in patients who have received shunts. Because evidence of contrast enhancement is nonspecific, it may not always prove beneficial diagnostically. For example, the patient with a systemic tumor who has had previous resection of an intraparenchymal metastasis and now returns with possible carcinomatous meningitis may not be helped by the finding of mild thickening and enhancement of the meninges. Such changes could result from postoperative reaction, especially if the enhancement is localized, or from leptomeningeal tumor. Alternatively, a patient with a history of meningeal carcinomatosis, treated with intrathecal chemotherapy and radiation, might demonstrate persistent enhancement of thickened and fibrotic meninges even in the face of normal CSF findings and repeated negative CSF cytology.

When contrast enhancement does occur, it may be quite localized, regardless of the cause. When the primary inciting factor is a localized skull lesion or a subdural hematoma, this finding is not surprising. However, when the reason for the meningeal enhancement is meningeal carcinomatosis, this finding is less expected. To understand this, one must realize that tumor can enter the CSF in several ways [7]. First, tumor may invade the dura, growing into the CSF later. Second, tumor cells can be shed into the CSF from superficial paren-



chymal metastases. Often, these metastases incite an overlying fibrotic reaction that tends to limit their spread to the CSF. Both mechanisms may lead to localized enhancement and thickening. Third, and most common, tumor can spread hematogenously to the CSF via small meningeal vessels. Tumor can also involve the CSF by growth along nerve roots. Once in the CSF, tumor cells may deposit and proliferate in a localized sheetlike fashion, causing regional enhancement. Alternatively, they may coat the entire arachnoid diffusely, causing diffuse meningeal enhancement. Finally, they can also lodge in the sulci or on the brain surface, growing into focal nodules that are centered on the pia. Patterns of meningeal enhancement will reflect patterns of growth.

Contrast-enhanced MR appears to be far more sensitive than contrast-enhanced CT for meningeal enhancement, especially when most of the enhancement lies against the skull vault. The percentage of cases of meningeal carcinomatosis demonstrating positive MR findings was surprisingly high in this study, although by no means 100%. Either the coronal or the axial plane is the most useful in making the diagnosis; sagittal scans reveal enhancement less often. In addition, since the normal dural reflections near the superior sagittal sinus usually show considerable enhancement, examination of midline sagittal sections only can be misleading.

The frequency of positive MR findings suggests an important ancillary role for imaging in suspected leptomeningeal tumor, especially in cases in which lumbar puncture is contraindicated or in which CSF examination is equivocal. Of course, in some patients, meningeal carcinomatosis may not be suspected clinically and the finding of abnormal meningeal enhancement can prompt a search for the correct diagnosis. Finally, contrast-enhanced MR can also be useful in patients with known leptomeningeal disease in order to suggest response to therapy on follow-up examinations.

Although contrast-enhanced MR is more sensitive to leptomeningeal tumor than contrast-enhanced CT is, its use in making this diagnosis is still secondary to examination of the CSF. In nearly one-third of our cases, contrast-enhanced MR

was negative. These cases may have been early in their course or less severe. As the disease progresses, either diffuse or nodular meningeal enhancement can be visualized. Diffuse enhancement is best depicted by contrast MR and is difficult to visualize on contrast CT unless it is severe and extends to the tentorium and basal cisterns. Nodular enhancement, however, appears to be detected equally well by either technique. The apparent faintness of enhancement of some leptomeningeal nodules suggests a possible role for higher doses of gadolinium.

In conclusion, we examined normal and abnormal meningeal enhancement. The superior sensitivity of contrast-enhanced MR has increased the frequency of abnormal findings and widened the differential diagnosis when these findings are detected. Meningeal carcinomatosis, as well as other meningeal abnormalities, are demonstrated considerably more often with contrast-enhanced MR than with contrast-enhanced CT, and imaging studies should play an increasingly important role in this often difficult diagnosis.

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