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Postoperative evaluation for intracranial recurrence of medulloblastoma: MR findings with gadopentetate dimeglumine.

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Postoperative Evaluation for Intracranial Recurrence of Medulloblastoma: MR Findings with Gadopentetate Dimeglumine

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PURPOSE: To characterize the gadopentetate dimeglumine–enhanced MR features of recurrent medulloblastoma. **METHODS:** The postsurgical gadopentetate dimeglumine–enhanced MR images of 48 patients (206 head examinations) with prior resection of medulloblastoma were retrospectively evaluated for enhancement in the brain parenchyma, meninges (dura, pia-arachnoid), and ventricles. **RESULTS:** Nineteen patients had recurrent tumor as determined by clinical course and positive imaging studies. Seventeen patients with recurrent disease had intracranial enhancement predominating in the pia-arachnoid (63%) or as a focal nodular brain lesion (26%). Three of these patients also had intraventricular metastases. None of the clinically healthy patients had these findings. One patient had recurrent tumor presenting within the fourth ventricle. Only 3 of 8 intraventricular lesions observed in the 4 patients initially enhanced with gadopentetate dimeglumine. Another patient with recurrent disease had extensive skeletal metastases without involvement of the central nervous system. Dural enhancement was observed in patients both with (42%) and without (38%) recurrent tumor. **CONCLUSION:** The MR findings of pia-arachnoidal or focal nodular brain enhancement are highly specific in the diagnosis of recurrent medulloblastoma. Pia-arachnoidal or focal nodular brain enhancement were also the most frequent patterns associated with recurrent tumor. Dural enhancement alone is not a reliable indicator of recurrent medulloblastoma. Not all intraventricular metastases enhance with gadopentetate dimeglumine, and careful evaluation for nonenhancing lesions within the ventricles should be made on postoperative MR examinations.

Index terms: Medulloblastoma; Magnetic resonance, postoperative; Magnetic resonance, contrast enhancement; Brain, magnetic resonance; Brain, neoplasms

Medulloblastomas represent a group of primitive neuroectodermal tumors that originate within the cerebellum (1–3). These neoplasms account for nearly one quarter of all intracranial neoplasms in infants and children (3, 4). Medulloblastomas generally grow rapidly and are known to be invasive and metastasize along cerebrospinal fluid pathways (1–3). It is because of these tumor characteristics that effective treatment of medulloblastoma depends on gross total surgical resection and subsequent craniospinal radiation therapy with or without adjuvant chemotherapy (3, 5–10).

The presence of disseminated disease dramatically reduces the 5-year survival rate of patients with medulloblastomas (5, 8). Postoperative surveillance is therefore critically important in the evaluation of local recurrent and/or metastatic disease. Magnetic resonance (MR) with gadopentetate dimeglumine has been recommended as the procedure of choice in the postoperative assessment of neoplasms of the central nervous system in pediatric patients (11, 12). Primary and recurrent medulloblastomas, however, have been shown to have highly

variable gadopentetate dimeglumine enhancement (13, 14). In some cases, the enhancement can be minimal or nonexistent (13, 14). In addition, gadopentetate dimeglumine enhancement may occur as a result of radiation therapy or surgery rather than recurrent disease (11).

The purpose of this study was to characterize the gadopentetate dimeglumine–enhanced MR features of recurrent medulloblastoma in comparison with nonmalignant postoperative changes.

Patients and Methods

Patient Group

We reviewed the tumor registry files and surgical and pathologic reports from three university medical centers and found records of 48 patients who had contrast-enhanced MR examinations (1988 through 1993) after resection of medulloblastomas. The group had 30 male and 18 female patients. At the time of initial resection and diagnosis, the patients ranged in age from 1 to 42 years (mean, 8.3). Forty-four tumors were histologically subclassified as classical medulloblastomas and the other four as medulloblastomas with desmoplasia. Two patients had preoperative MR findings of tumor spread beyond the primary lesion. One patient had a single metastatic lesion within a lateral ventricle, and the other had leptomeningeal tumor in the posterior cranial fossa. All patients received postoperative radiation therapy, which most often consisted of 36 Gy to the whole brain and spine as well as booster doses of 18 Gy to the posterior cranial fossa. Forty-two patients also received chemotherapy.

MR

MR was performed at 1.5 T for 44 patients and at 1.0 T for 4 patients. A total of 206 postsurgical MR studies of the head were performed. The number of postoperative MR studies per patient ranged from 3 to 10 (mean, 4). These examinations were obtained from 2 days to 11 years after surgery.

Multisection spin-echo pulse sequences were used in all MR studies and included short repetition time (TR)/echo time (TE) (430–800/11–30/1–2 excitations) and long-TR/first-echo TE, second-echo TE (2000–3200/15–30, 75–100/1–2) sequences. Short-TR/TE images were obtained in the axial and sagittal planes. Long-TR images were acquired in the axial plane. MR was performed after intravenous administration of gadopentetate dimeglumine (0.1 mmol/kg) using short-TR/TE sequences (430–800/11–30/1–2) acquired in the axial planes for all patients and in the coronal and/or sagittal planes for most patients. MR images were 5 mm thick with interimage gaps of 0.5 to 1 mm. The acquisition matrix ranged from 256 × 128 to 256 × 256.

The unenhanced and enhanced short-TR images were evaluated for enhancement in dural, pia-arachnoidal, parenchymal, encephalotomy, ependymal, and intraventricular locations. When applicable, enhancement at each site was evaluated with regard to degree (mild, moderate, or marked) and configuration (nodular, linear, or both).

Results

Patients with Recurrent Disease

Nineteen of the 48 patients had recurrent tumor as determined by clinical course, positive cerebrospinal fluid cytology (n = 14), or histologic evaluation of resected surgical specimens (n = 5). Twelve of these patients were dead 5

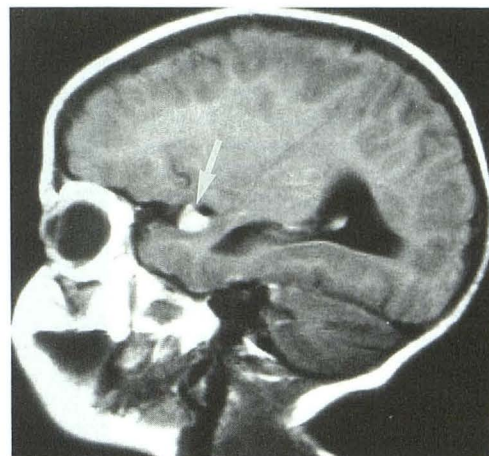Fig 1. MR images of a 2.5-year-old boy obtained 7 months after surgery.

A, Short-TR axial (560/30) and B, coronal (430/16) enhanced MR images show extensive pia-arachnoid enhancement representing leptomeningeal tumor.

A   **B**
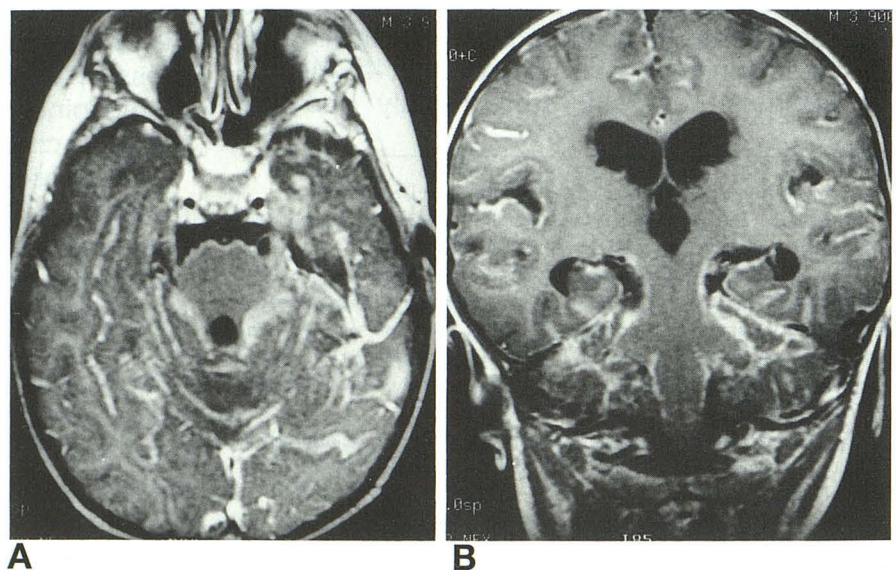
months to 10.8 years (mean, 2.7 years) after initial surgery. Patient age at the time of death ranged from 2.1 to 27.7 years (mean, 9.8 years). The 7 other patients with recurrent disease were alive 1.5 to 4.8 years after initial surgery.

Pia-arachnoidal enhancement or combined pia-arachnoidal with adjacent cortical enhancement were the initial patterns observed for 8 and 4 patients, respectively. Pia-arachnoidal enhancement appeared as enhancement along the gyral contours, including the deeper portions of the sulci (Fig 1). The degree of pia-arachnoidal enhancement was marked in all cases and was predominantly linear in configuration for 10 patients (Fig 1) or nodular in 2 (Fig 2). These enhancement patterns occurred 2.5 months to 9 years (mean, 25 months; median, 15 months) after surgery in 10 patients. One patient had pia-arachnoidal enhancement demonstrated on a preoperative MR examination obtained 1 day before surgery. Another patient had pia-arachnoidal enhancement demonstrated on an MR examination obtained 2 days after surgery. Both of these patients also had leptomeningeal enhancement on subsequent postoperative MR examinations. Ten patients were dead 1 month to 21 months (mean, 10 months; median, 12 months) after pia-arachnoidal enhancement was initially detected. The two surviving patients also have had deteriorating clinical courses. Six of the patients with only pia-arachnoidal enhancement subsequently developed cortical enhancement adjacent to the pial margins or deeper within the brain.

A single focal site of parenchymal enhancement was the initial pattern observed in five patients (Figs 3 and 4). These lesions had ovoid or spheroid configurations. Three were located near the sites of the primary tumors within the cerebella (Fig 3), and two involved the inferior frontal regions (Fig 4). The degree of enhancement was moderate in two cases (Fig 3) and marked in three (Fig 4). This enhancement type occurred 7 to 48 months (mean, 28 months) after surgery. Three of these patients were still alive 10 to 44 months (mean, 21 months) after this enhancement pattern was detected. Intraventricular metastases and leptomeningeal tumors subsequently developed in the other two patients; they were dead 2.5 months and 38 months, respectively, after the initial enhancement patterns were detected.

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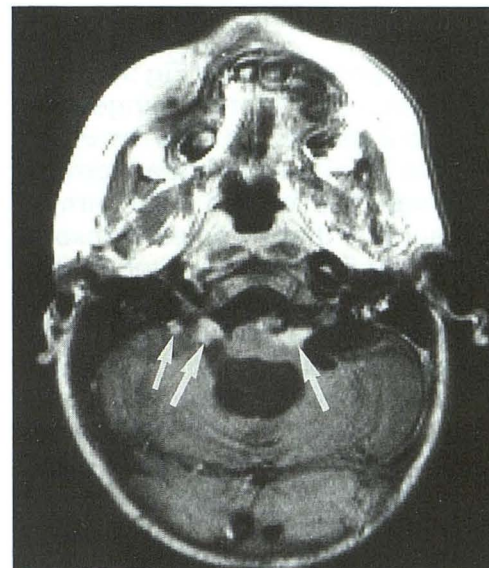
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Fig 2. MR images of a 4-year-old boy obtained 32 months after surgery.

A, Enhanced short-TR (620/20) sagittal MR image shows a nodular enhancing lesion in the pia-arachnoid (*arrow*).

B, Enhanced short-TR (760/20) axial MR image shows nodular enhancing lesions in the pia-arachnoid (*arrows*).

A total of eight intraventricular lesions were seen in four patients. The intraventricular lesions had nodular configurations (Figs 5–7). Only three of the eight lesions within the ventricles enhanced initially with gadopentetate dimeglumine (Figs 5 and 7). One patient had a single nonenhancing metastatic lesion within a lateral ventricle demonstrated on a preoperative MR examination 12 days before surgery (Fig 5). A subsequent MR examination 2 years later showed enlargement and enhancement of the intraventricular lesion and as invasion into the

Fig 3. MR images of an 11-year-old girl obtained 4 years after surgery.

A, Unenhanced and B, enhanced short-TR (600/12) axial MR images show moderately enhancing focal mass (arrows) near the site of the resected primary tumor. Four earlier postoperative MR examinations showed no recurrent tumor at this site.

A   B
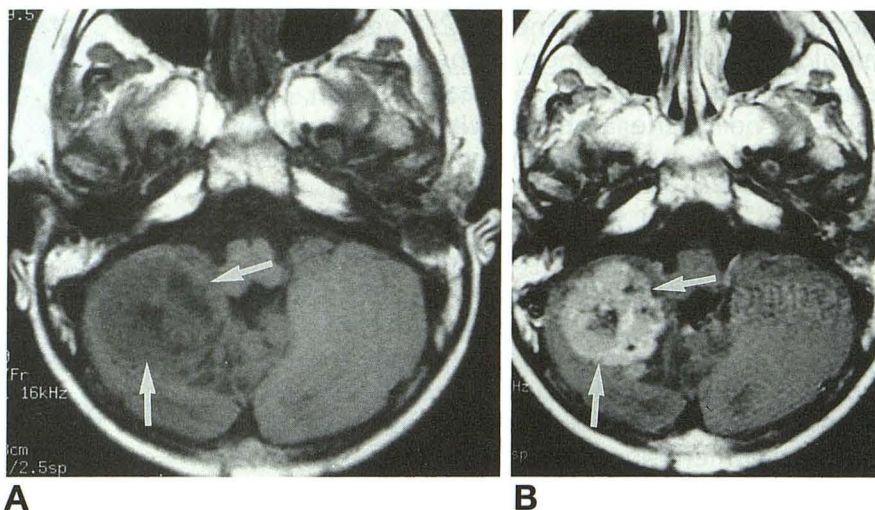
adjacent brain parenchyma (Fig 5). This patient also had a recurrent leptomeningeal tumor in the posterior fossa on the later exam. Another patient had a focal area of enhancement within the fourth ventricle on an MR examination 9 days after surgery that completely resolved on a follow-up study 5.5 months later (Fig 6). This patient, however, subsequently had an enhancing recurrent lesion within the fourth ventricle and adjacent cerebellum that was demonstrated on the seventh postoperative MR examination 40 months after surgery. Two other patients had intraventricular metastases that occurred after the earlier presentation of leptomeningeal or fo-

cal brain recurrence (Fig 7). Enhancement at the ependymal margin was observed in 10 patients and generally occurred late or in association with pia-arachnoidal or parenchymal enhancement.

Multiple skeletal metastases without recurrent tumor in the central nervous system was the initial pattern observed for one patient in our series (Fig 8). The skeletal lesions were detected 35 months after surgery. The metastases were shown by computed tomography to be sclerotic focal lesions scattered throughout the spine and pelvis. Contrast-enhanced MR of the brain and spine, performed 2 days after the

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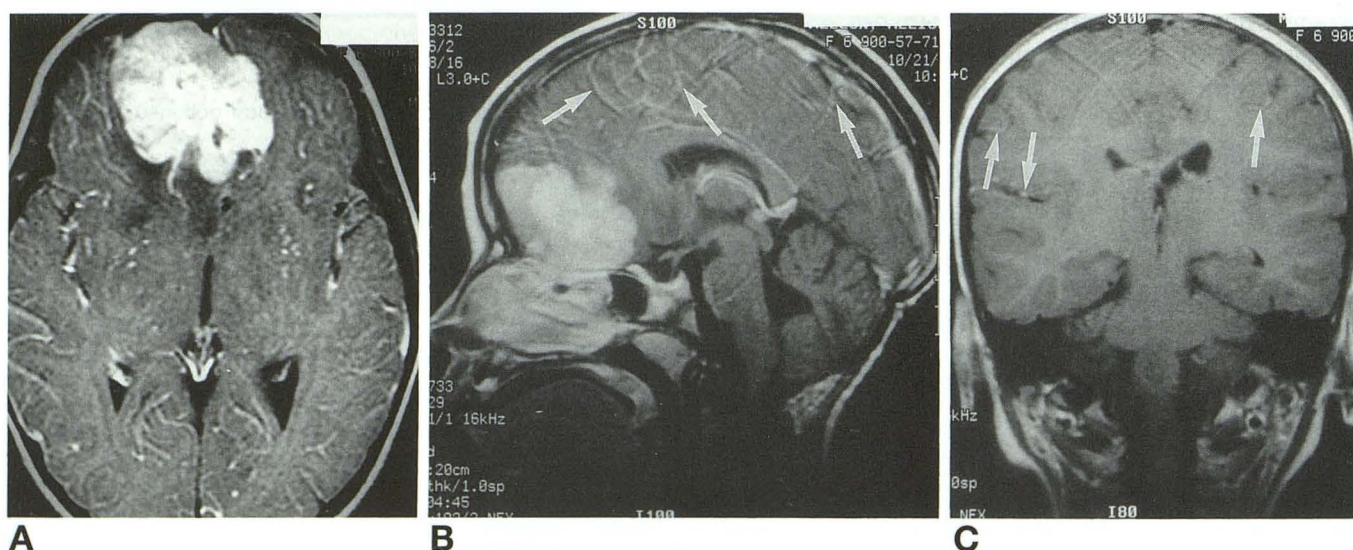
Fig 4. MR image of a 6-year-old girl obtained 2.3 years after surgery.

A, Enhanced short-TR (560/30) axial MR image shows a large markedly enhancing lesion in the inferior right frontal lobe with extension toward the left. Four earlier postoperative MR examinations showed no abnormalities in this location.

Linear enhancing structures in the sulci represent veins. This was confirmed on enhanced short-TR sagittal (733/29) (B) and coronal (433/16) (C) MR images on which vessels appeared as small round or thin curvilinear enhancing structures (arrows).
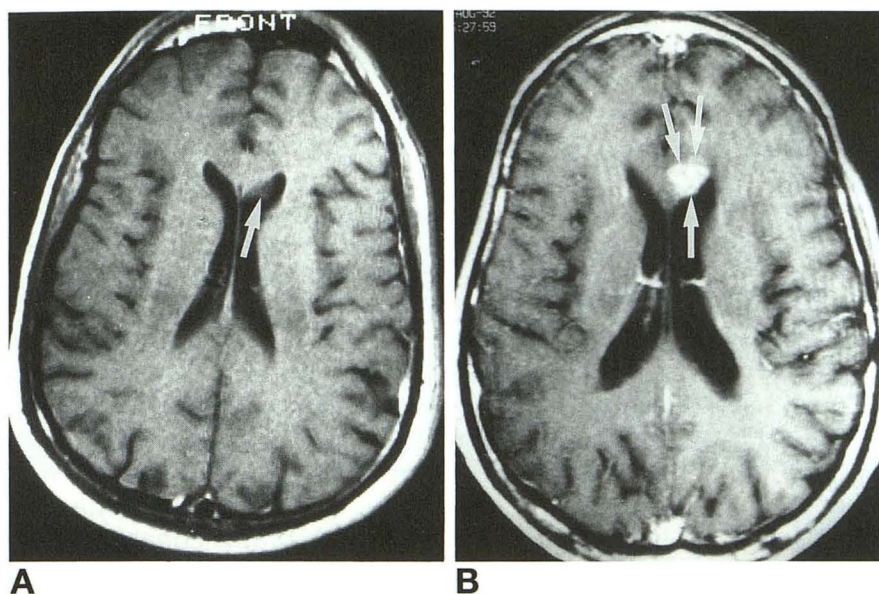
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computed tomographic examination, showed no lesions or abnormal enhancement involving the dura, leptomeninges, brain or spinal cord.

Dural enhancement was observed in eight patients, three of whom also had intraventricular shunts. The dural enhancement was linear in configuration, and was mild in degree (less than 2 mm thick) for five patients (Fig 9) and moderately marked (greater than 2 mm thick) in

three. Six patients with dural enhancement also had pia-arachnoidal tumor (Fig 9), and the other two had focal recurrent lesions within the brain parenchyma.

Patients without Recurrent Disease

Twenty-nine patients had no clinical evidence of recurrent disease. The mean and me-

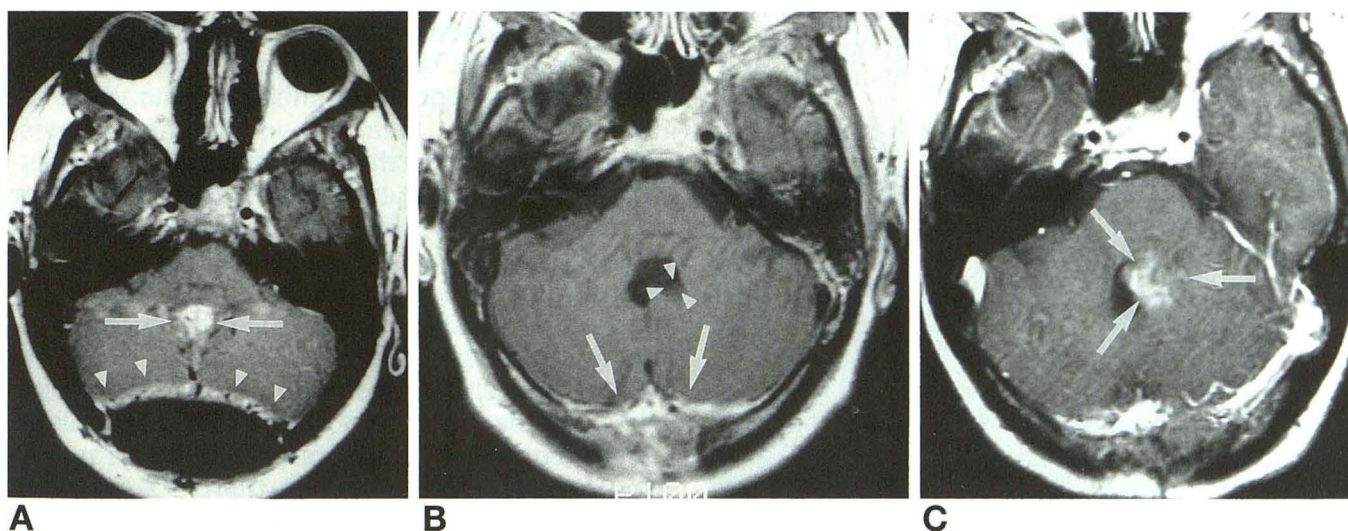**A**　　　　　　**B**　　　　　　**C**

Fig 6. Postoperative MR images of a 13-year-old girl.

A, Enhanced short-TR (600/20) axial MR image obtained 9 days after surgery shows an enhancing focus within the fourth ventricle (*arrows*). Enhancement and fluid collection are also noted at the meningogaleal complex (*arrowheads*) resulting from the craniectomy. The meningogaleal complex represents the surgical attachment of the dura to the overlying galea as described previously (15).

B, Enhanced short-TR (650/20) axial MR image obtained 5.5 months later shows resolution of the intraventricular enhancing focus and fluid collection at the craniectomy site. Prominent enhancement persists at the meningogaleal complex (*arrows*). Irregularity of the fourth ventricular contour is seen, as well as an equivocal nonenhancing intraventricular lesion (*arrowheads*).

C, Enhanced short-TR (500/29) axial MR image obtained 40 months after surgery shows an enhancing lesion within the fourth ventricle with invasion into the adjacent cerebellum (*arrows*).
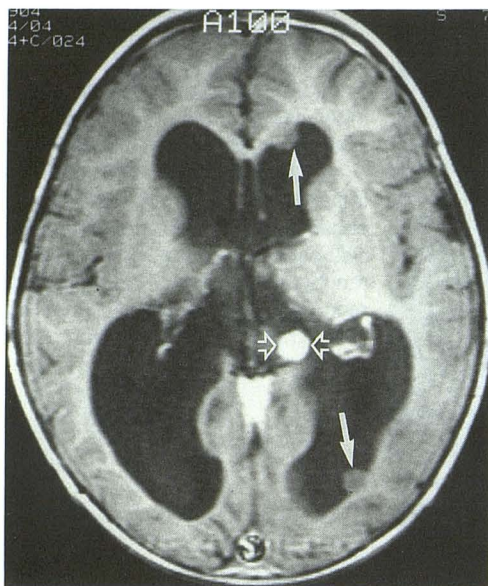
Fig 7. MR image of a 2-year-old boy obtained 7 months after surgery. Enhanced short-TR (500/20) axial MR image shows nonenhancing (*solid arrows*) and enhancing (*open arrows*) intraventricular metastases.

dian follow-up times for these patients were 50 and 46 months, respectively. None of the patients without recurrent medulloblastomas had pia-arachnoidal or focal nodular parenchymal enhancement. Linear enhancement at the encephalotomy margins was observed in 6 of 8 patients who had MR examinations within 3 weeks after surgery (Fig 10A). This type of en-

Fig 8. Enhanced axial CT scan of a 22-year-old man obtained 35 months after surgery shows multiple blastic metastases within the iliac bones and sacrum. This patient also had numerous blastic lesions within multiple vertebrae. No recurrent disease was identified within the central nervous system on enhanced MR images of the head and spine obtained 2 days later.

hancement was transient and was not present on follow-up examinations 2 to 4 months later (Fig 10B). Linear dural enhancement was observed in 11 patients, 6 of whom had intraventricular shunts. Five patients, with intraventricular shunts, however, did not have dural enhancement. The degree of dural enhancement was mild in 7 (less than 2 mm thick) and moderately marked in 4 (greater than 2 mm thick) (Fig 11).

The sensitivity, specificity, and accuracy of dural, pia-arachnoidal, and parenchymal enhancement patterns in the detection of recurrent medulloblastoma are listed in the Table. Dural enhancement alone yielded relatively low sensitivity, specificity, and accuracy values for recurrent tumors. Focal brain enhancement, other than the linear pattern along encephalotomy margins, and pia-arachnoidal enhancement were highly specific in the detection of recurrent disease. Pia-arachnoidal or focal nodular brain enhancement were also the most frequent signs associated with recurrent disease.

Discussion

Medulloblastoma is the most common type of the primitive neuroectodermal tumors involving the central nervous system (1, 2). The incidence of medulloblastoma is most frequent during the first decade, although a second smaller peak occurs in the third decade (3). Medulloblastomas that occur in childhood are most often midline in location within the vermes, whereas in adults, they are nearly equally distributed in either the cerebellar hemispheres or vermes (3, 13). Extension of these rapidly growing tumors into the fourth ventricles or regional leptomeninges is common and predisposes to metastatic disease via the cerebrospinal fluid pathways (3).

As a result, effective treatment generally consists of gross total tumor resection with subsequent high-dose radiation therapy to the posterior cranial fossa (greater than 50 Gy) and lower-dose prophylactic radiation therapy (25 to 45 Gy) to the rest of the brain and spine (5, 7–10). Adjuvant chemotherapy may be beneficial in some patients with advanced disease (9, 10). With combinations of these treatment methods, the 5-year event-free survival probability ranges from 50% to 59% and the 5-year survival probability from 53% to 68% (5, 7, 9, 10). Tumor size, neoplastic cells in cerebrospi-
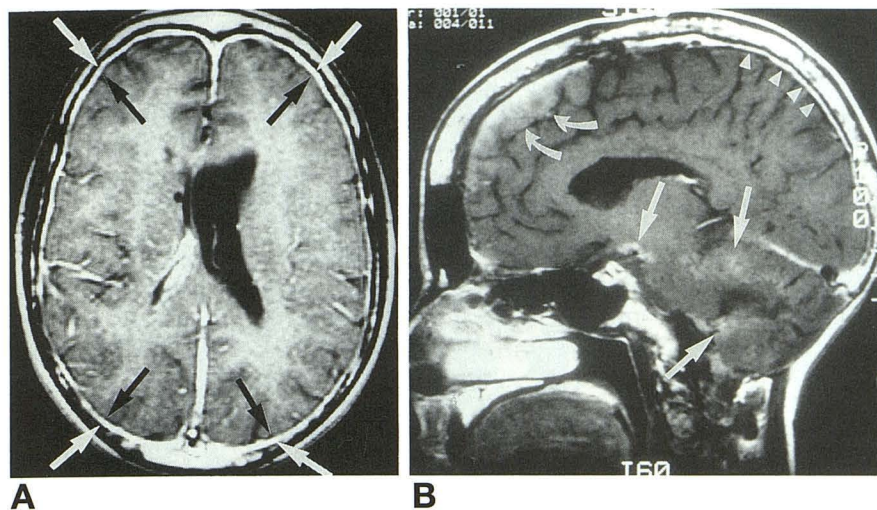
Fig 9. MR image of a 20-year-old man obtained 10 years after surgery.

A, Enhanced short-TR (500/20) axial MR image shows diffuse dural enhancement (*arrows*).

B, Enhanced short-TR (450/20) sagittal MR image shows dural enhancement (*arrowheads*), as well as pia-arachnoidal enhancement representing a leptomeningeal tumor involving the cerebellum and hypothalamic region (*arrows*). Volume averaging of the anterior portion of the falx is also noted (*curved arrows*).

nal fluid, and macroscopic subarachnoid tumors at diagnosis have been reported to be factors associated with poor prognoses (6, 9, 10).

Contrast-enhanced MR has been recommended as the method of choice for the postoperative assessment of the brain parenchyma and the different meningeal layers (ie, the dura mater [pachymeninges] and pia-arachnoid [leptomeninges]) (11, 12, 16). A functional blood-meningeal barrier exists in the leptomeningeal compartment because of tight junctions of the capillaries (11). Leptomeningeal contrast enhancement is usually associated with neoplastic infiltration or inflammation and infection (11, 16–23).

Pia-arachnoidal enhancement was the initial pattern observed in 63% of patients with recurrent medulloblastomas. All but two of the patients with leptomeningeal tumor had recurrent disease within 26 months of surgery. The two exceptions were patients who had late recurrence of pia-arachnoidal tumor 3.5 and 9 years after surgery. Late recurrences also have been reported for adults with medulloblastomas (24). Pia-arachnoidal enhancement was associated with a very poor prognosis. Eighty-three percent of these patients were dead within 21 months after this enhancement pattern was detected. Of the various tumors that result in leptomeningeal metastases, medulloblastoma has been reported to be the most frequent (22). None of the patients without recurrent medulloblastomas had pia-arachnoidal enhancement.

Dural (pachymeningeal) enhancement, however, was observed both in patients with (42%)

and without (38%) recurrent medulloblastomas. The low specificity of dural enhancement for recurrent neoplasms has been reported previously (11, 16, 17). Dural enhancement can be a common postsurgical finding that may result from perioperative meningeal inflammation and eventual fibrosis (11, 15–17). Prominent dural enhancement has been associated with postoperative subdural collections that become permeated with capillaries or fibrovascular tissue during the subsequent organization phase (11). This enhancement pattern is common in patients imaged within 3 months after surgery and has been reported to persist as long as 40 years (15).

A focal enhancing mass within brain parenchyma was the initial pattern of recurrent medulloblastoma in five (26%) patients. This enhancement type occurred in a mean postsurgical time of 28 months and as early as 7 months in one case. In our series, recurrent focal lesions typically occurred later and had different configurations than the linear pattern of nonmalignant enhancement observed along encephalotomy margins. Linear enhancement at the encephalotomy site was observed within 3 weeks after surgery but not on MR 4 months later. Elster and DiPersio (15) reported that enhancement along encephalotomy margins occurred in 10 of 15 patients (adults and children) imaged within 6 months after surgery but not after 1 year. The slightly greater duration of encephalotomy margin enhancement in their series than in ours may be related to their older patient population, more varied surgical sites, and different postoperative treatment protocols.
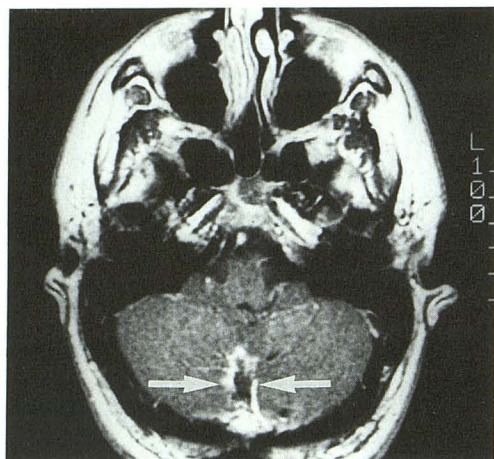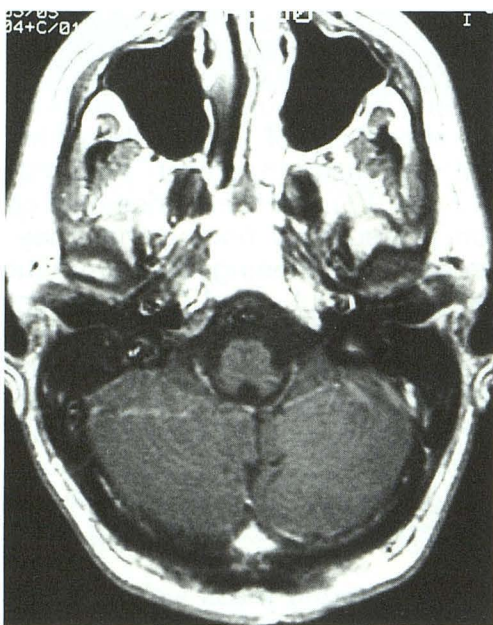
A

B

Fig 10. *A,* Enhanced short-TR (500/29) axial MR image of a 27-year-old man obtained 11 days after surgery shows linear enhancement at the encephalotomy site (*arrows*).

B, Enhanced short-TR (600/11) axial MR image obtained 2.5 months later showed no enhancement in this location.
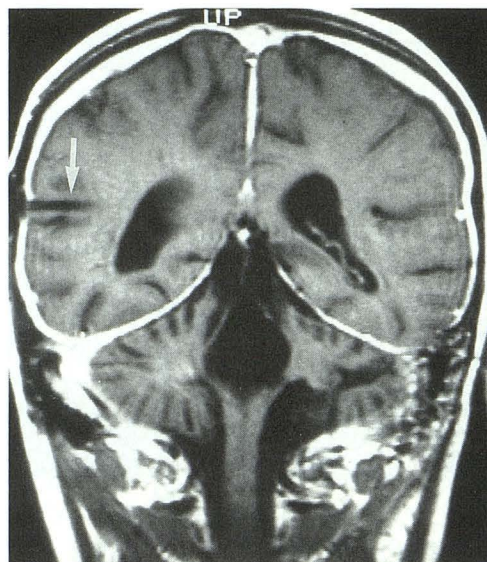

Fig 11. MR image of a 12-year-old girl obtained 9 months after surgery. Enhanced short-TR (760/20) coronal MR image shows prominent diffuse dural enhancement. A shunt tube tract is also seen (*arrow*).

diation therapy in an attempt to minimize ocular side effects (25).

Four patients in our series had intraventricular lesions. One patient had a single nonenhancing metastatic lesion in a lateral ventricle that was demonstrated on a preoperative MR examination. This patient subsequently developed leptomeningeal tumor and enhancement and progressive enlargement of the intraventricular lesion. Another patient had an enhancing lesion within the fourth ventricle as the initial site of recurrence. Two patients developed intraventricular metastases after the earlier presentation of leptomeningeal or focal parenchymal recurrence. Only three of the eight intraventricular lesions initially enhanced with gadopentetate dimeglumine. Rollins et al (14) reported that

In our study, three recurrent focal masses were located near the sites of the resected primary tumors, whereas two other lesions involved the inferior frontal regions. Donnal et al (25) reported that isolated subfrontal metastases can be the initial sites of recurrent medulloblastomas. They suggested that subfrontal metastases may result from tumor cells seeding this region because of the prone positioning of patients during surgery and underdosage of ra-

Relationship of recurrent medulloblastoma and MR enhancement patterns with gadopentetate dimeglumine

	Sensitivity, %	Specificity, %	Accuracy, %
1. Dural	42	62	54
2. Pia-arachnoidal	63	100	85
3. Focal parenchymal	26	100	71
4. Pia-arachnoidal or focal parenchymal	89	100	96

three of nine patients with recurrent medullo-blastomas had lesions that did not enhance on MR examinations after contrast administration. The nonenhancing recurrent lesions in their study were located within the ventricles or along the ependymal margins (14). The lack of en-hancement of metastatic lesions in the ventri-cles or ependymal margins may be secondary to tenuous or attenuated blood supplies to these lesions. Eventual enhancement of the intraven-tricular lesions may occur when they invade adjacent brain parenchyma (Fig 5).

Extraneural or systemic metastases have been reported to occur as the first sites of re-currence in 5% to 15% of patients with medul-loblastomas (26, 27). Bone is the most frequent location of extraneural metastases, occurring in 80% to 90% of these cases (26, 27). The skel-etal lesions are most often blastic, although lytic and mixed patterns also occur. Other sites of extraneural metastases include lymph nodes, liver, and lungs (26, 27). Multiple skeletal me-tastasis without recurrent tumor in the central nervous system was the initial pattern observed for one patient in our series. The metastases were shown by computed tomography to be sclerotic lesions scattered throughout the spine and pelvis (Fig 8). Contrast-enhanced MR 2 days later showed no abnormal enhancement involving the dura, leptomeninges, brain, or spinal cord. Tarbell et al (26) reported that the addition of chemotherapy to the standard treatment of surgery and craniospinal irra-diation has dramatically reduced the incidence of extraneural metastases. The patient in our series with osseous metastases was treated with surgery and radiation therapy, but not with chemotherapy.

In conclusion, the MR findings of pia-arach-noidal or focal parenchymal (nodular) brain en-hancement are highly specific in the diagnosis of recurrent medulloblastoma. Pia-arachnoidal or focal nodular brain enhancement were the most frequent initial patterns associated with recurrent tumors. The detection of pia-arach-noidal enhancement is associated with a poor prognosis. Dural enhancement alone is not a reliable indicator of recurrent tumor. Last, me-tastases within the ventricles may not enhance with gadopentetate dimeglumine. Careful scru-tiny for nonenhancing recurrent lesions in these locations therefore should be made.

References

1. Becker LE, Hinton D. Primitive neuroectodermal tumors of the central nervous system. *Hum Pathol* 1983;14:538–550
2. Rorke LB. The cerebellar medulloblastoma and its relationship to primitive neuroectodermal tumors. *J Neuropathol Exp Neurol* 1983;42:1–15
3. Russell DS, Rubinstein LJ. Medulloblastomas. In: *Pathology of Tumours of the Nervous System*. 5th ed. Baltimore: Williams & Wilkins, 1989:251–279
4. Arseni C, Ciurea AV. Statistical survey of 276 cases of medullo-blastoma (1935–1978). *Acta Neurochir* 1981;57:159–166
5. Hughes EN, Shillito J, Sallan SE, Loeffler JS, Cassady JR, Tarbell NJ. Medulloblastoma at the Joint Center for Radiation Therapy between 1968 and 1984: the influence of radiation dose on the patterns of failure and survival. *Cancer* 1988;61:1992–1998
6. Deutsch M. Medulloblastoma: staging and treatment outcome. *Int J Radiat Oncol Biol Phys* 1988;14:1103–1107
7. Levin VA, Rodriguez LA, Edwards MSB, et al. Treatment of medul-loblastomas with procarbazine, hydroxyurea, and reduced radia-tion doses to whole brain and spine. *J Neurosurg* 1988;68:383–387
8. Mazza C, Pasqualin A, Da Pian R, Donati E. Treatment of medul-loblastoma in children: long-term results following surgery, radio-therapy and chemotherapy. *Acta Neurochir* 1981;57:163–175
9. Tait DM, Thornton-Jones H, Bloom HJG, Lemerle J, Morris-Jones P. Adjuvant chemotherapy for medulloblastoma: the first multi-centre control trial of the Internal Society of Paediatric Oncology (SIOP I). *Eur J Cancer* 1990;26:464–469
10. Evans AE, Jenkin RDT, Sposto R, et al. The treatment of medul-loblastoma. *J Neurosurg* 1990;72:572–582
11. Hudgins PA, Davis PC, Hoffman JC Jr. Gadopentetate dimeglu-mine-enhanced MR in children after surgery for brain tumor: spec-trum of meningeal findings. *AJNR Am J Neuroradiol* 1991;12:301–307
12. Bird CR, Drayer BP, Medina M, Rekate HL, Flom RA, Hodak JA. Gd-DTPA-enhanced MR imaging in pediatric patients after brain tumor resection. *Radiology* 1988;169:123–126
13. Meyers SP, Kemp S, Tarr RW. MR imaging features of medullo-blastomas. *AJR Am J Roentgenol* 1992;158:859–865
14. Rollins N, Mendelsohn D, Mulne A, et al. Recurrent medulloblas-toma: frequency of tumor enhancement on Gd-DTPA MR imag-ing. *AJNR Am J Neuroradiol* 1990;11:583–587
15. Elster AD, DiPersio DA. Cranial postoperative site: assessment with contrast-enhanced MR imaging. *Radiology* 1990;174:93–98
16. Sze G. Diseases of the intracranial meninges: MR imaging fea-tures. *AJR Am J Roentgenol* 1993;160:727–733
17. Sze G, Soletsky S, Bronen R, Krol G. MR imaging of the cranial meninges with emphasis on contrast enhancement and menin-geal carcinomatosis. *AJNR Am J Neuroradiol* 1989;10:965–975
18. Chang KH, Han MH, Roh JK, Kim IO, Han MC, Kim C-W. Gado-pentetate dimeglumine–enhanced MR imaging of the brain in patients with meningitis: comparison with CT. *AJNR Am J Neu-roradiol* 1990;11:69–76

19. Mathews VP, Kuharik MA, Edwards MK, D'Amour PG, Azzarelli B, Dreesen RG. Gadopentetate dimeglumine–enhanced MR of experimental bacterial meningitis: evaluation and comparison with CT. *AJNR Am J Neuroradiol* 1988;9:1045–1050

20. Paakko E, Patronas NJ, Schellinger D. Meningeal Gd-DTPA enhancement in patients with malignancies. *J Comput Assist Tomogr* 1990;14:542–546

21. Phillips ME, Ryals TJ, Kambhu SA, Yuh WTC. Neoplastic versus inflammatory meningeal enhancement with Gd-DTPA. *J Comput Assist Tomogr* 1990;14:536–541

22. Lee Y-Y, Tien RD, Bruner JM, DePena CA, Van Tassel P. Loculated intracranial leptomeningeal metastases: CT and MR characteristics. *AJNR Am J Neuroradiol* 1989;10:1171–1179

23. Yousem DM, Patrone PM, Grossman RI. Leptomeningeal metastases: MR evaluation. *J Comput Assist Tomogr* 1990;14:255–261

24. Koci TM, Chiang F, Mehringer CM, et al. Adult cerebellar medulloblastoma: imaging features with emphasis on MR findings. *AJNR Am J Neuroradiol* 1993;14:929–939

25. Donnal J, Halperin EC, Friedman HS, Boyko OB. Subfrontal recurrence of medulloblastoma. *AJNR Am J Neuroradiol* 1992; 13:1617–1618

26. Tarbell NJ, Loeffler JS, Silver B, et al. The change in patterns of relapse in medulloblastoma. *Cancer* 1991;68:1600–1604

27. Olson EM, Tien RB, Chamberlain MC. Osseous metastasis in medulloblastoma: MRI findings in an unusual case. *Clin Imaging* 1991;15:286–289.