GADOLINIUM ENHANCED MAGNETIC RESONANCE IMAGING IN THE DIAGNOSIS OF ANTERIOR VISUAL PATHWAY MENINGIOMAS*

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INTRODUCTION

THE ROLE OF MAGNETIC RESONANCE (MR) IN DIAGNOSIS OF DISEASES OF the brain has become more clearly defined since its introduction for general clinical use less than 10 years ago. Compared to computed tomography (CT), MR is much more valuable in the diagnosis of some diseases of the brain and spinal cord. For example, it is far more sensitive in detection of multiple sclerosis plaques and in the early detection of low-grade astrocytomas (gliomas). In the detection of meningiomas, however, MR has been inferior to CT. This has been especially true in demonstrating extra-axial meningiomas that occur in the skull base at the orbito-cranial junction.[1-3]

Invasion of the optic canal and intracranial extension by perioptic sheath meningiomas has been particularly difficult to detect by both CT and MR.[4,5] Despite the introduction of surface coils and altering pulse sequences to manipulate tissue contrast, meningiomas of the anterior visual pathways continue to appear isointense and undistinguished from the surrounding brain on MR images (Figs 1A and B, 2A and B).

The need for a contrast agent to demonstrate differences in proton resonance of normal and abnormal tissue was apparent early in the clinical experience with MR. Experiments with gadolinium-diethylene triamine pentacetic acid (Gd-DTPA) as a contrast agent for use with MR began in 1984.[6] Following its introduction for clinical trials in 1986, Gd-DTPA has quickly proved useful in detection of small extra-axial lesions such as meningiomas, schwannomas, meningeal metastasis and granulomas.[7,8]

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We have used it in clinical practice since its approval by the FDA in July, 1988.

Because visual loss is often the first sign of these potentially life-threatening tumors, it is important for the ophthalmologist to be aware of this significant advance in diagnosis. This paper reports our initial experience with Gd-DTPA enhanced MR images in detection of meningiomas of the anterior visual pathway.

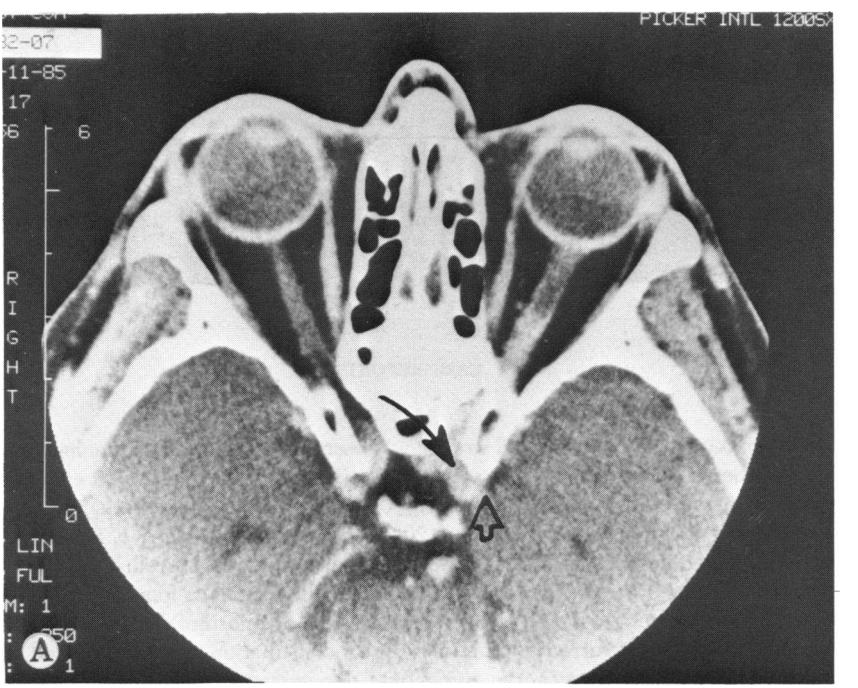
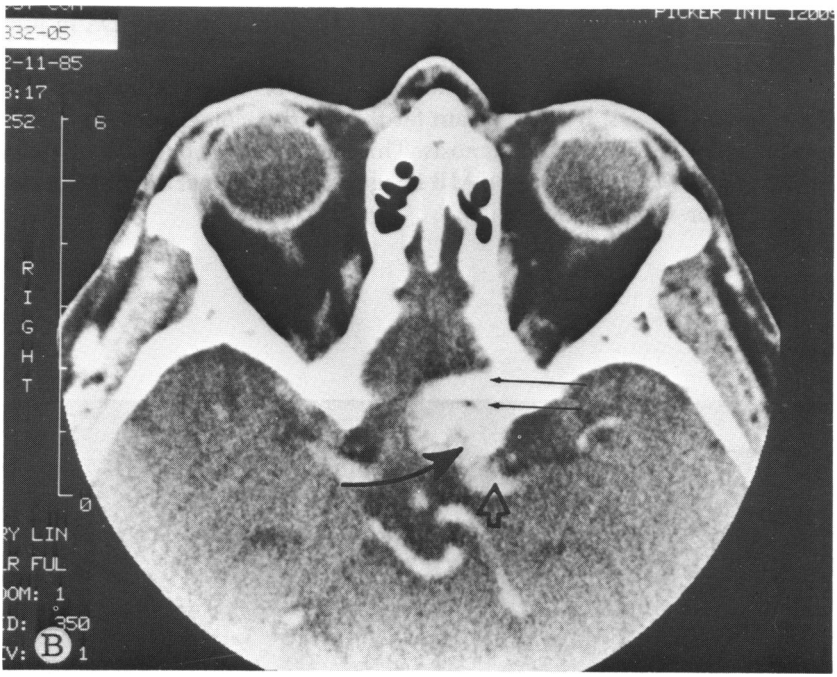


FIGURE 1

A: Case 3. A 76-year-old woman with long-standing blindness in the left eye. Enhanced axial CT scan. Left optic nerve/anterior clinoid meningioma with involvement of the optic canal and orbital apex is well seen (*curved arrow*). Intracranial extension lies adjacent to internal carotid artery. Middle cerebral artery (*open arrow*) is poorly differentiated from meningioma. Compare to Fig 2A and B. B: More superior enhanced CT scan than 1A demonstrates hyperostosis of left anterior clinoid process (*2 small arrows*). Optic nerve meningioma is seen adjacent to clinoid process (*curved arrow*). Middle cerebral artery (*open arrow*) is better defined than in 1A. Compare to Fig 2A and B.



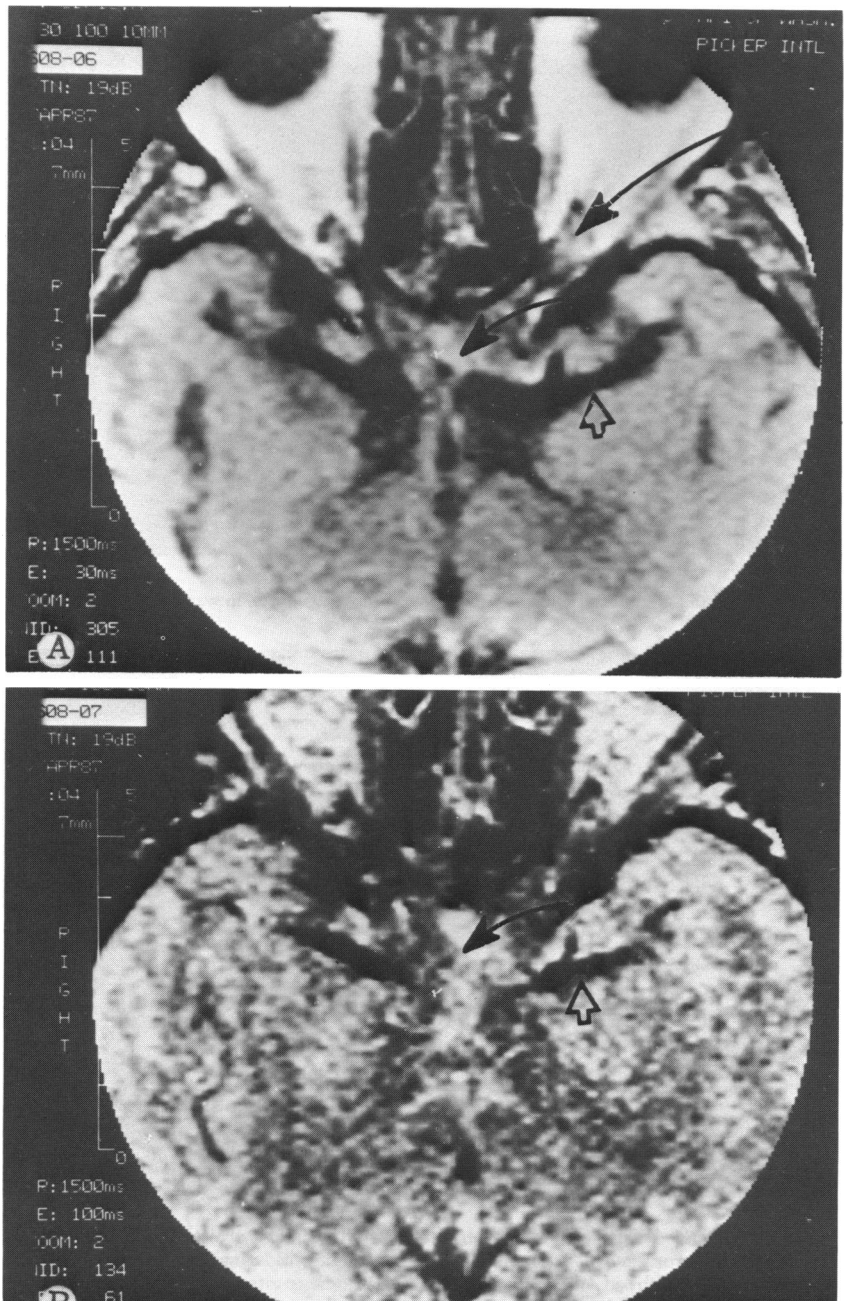
CASE REPORTS

CASE 1

LHF, a 26-year-old right handed woman was seen by her local ophthalmologist on September 17, 1988, because of a subconjunctival hemorrhage in her left eye. The patient attributed this to her contact lens. Upon examination, the ophthalmologist noted the following: vision, right eye, 20/20 with correction; left eye, 20/40 with correction. There was a left afferent pupillary defect. Pale disk swelling of the left optic nerve was present (Fig 3). An enlarged left blind spot was found upon visual field evaluation but the fields were otherwise full. A diagnosis of optic neuritis was made and she was referred to a neurologist for consultation. CT was ordered by the neurologist. An optic nerve tumor was demonstrated with probable intracranial extension (Fig 4). An MR image was obtained that demonstrated definite

FIGURE 2

A: Case 3. Axial MR T_1-weighted image shows a meningioma involving left orbital optic nerve, orbital apex, optic canal and intracranial optic nerve (*curved arrows*). Middle cerebral artery (*open arrow*) is well differentiated from meningioma. Tumor is isointense with brain tissue. Compare to Fig 1A and B. B: Axial MR T_2-weighted image. Meningioma (*curved arrow*) shows slightly increased intensity when compared to 2A. Middle cerebral artery is well defined. Compare to Fig 1A and B.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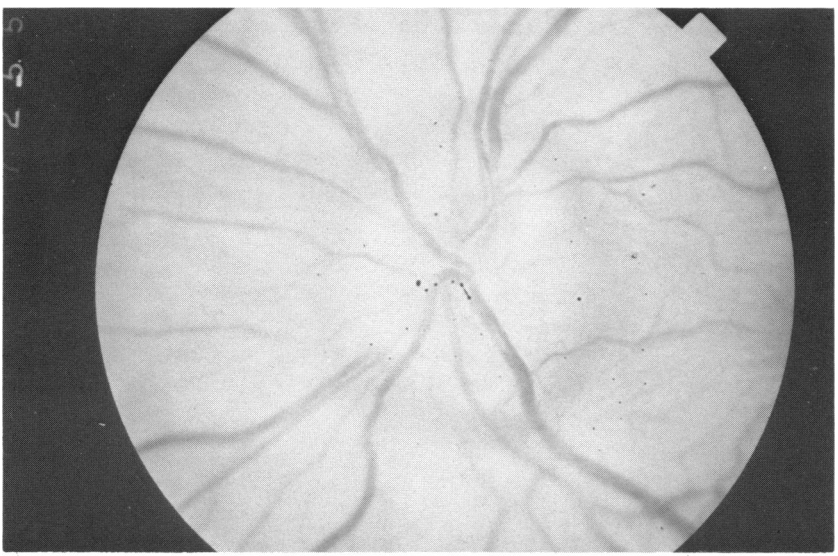


FIGURE 3
Case 1. Swollen pale left optic nerve in a 26-year-old woman. Vision was 20/40. Shunt vessels are absent.

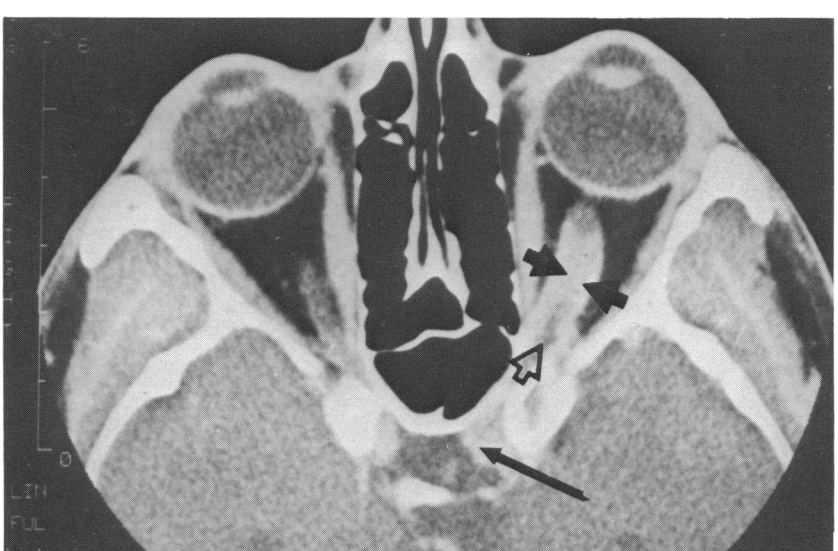


FIGURE 4
Case 1. Enhanced axial CT scan demonstrates left primary orbital optic nerve sheath meningioma (*arrowheads*). Tumor has invaded through optic canal into intracranial cavity (*single arrow*). "Railroad track" sign (*open arrow*) demonstrates optic nerve as a negative shadow surrounded by the enhanced tumor.

intracranial extension (Fig 5A). The extent of intracranial involvement was not fully demonstrated by either of these techniques. She was then referred for neuro-ophthalmologic consultation on November 17, 1988. Best corrected vision was noted as follows: right eye, 20/20; left eye, 20/40. Red color vision was 40% desaturated in the left eye as compared to the right. Color vision testing with HRR color plates and the Farnsworth dichotomous test was normal. A 2+ left afferent pupillary defect was noted. There was inability to fully elevate the left eye developing 6 prism diopters of right hypertropia on upgaze. Hertel exophthalmometry measured each eye at 16.5 mm with an interorbital distance of 95 mm. The left optic nerve demonstrated pale disk swelling with elevation of 3 diopters. Shunt vessels were absent. The right disk was normal. One cafe-au-lait spot measuring 1 cm was observed on the sole of the left foot. Lisch nodules were absent. The visual fields were full except for an enlarged left blind spot. There was no family history of neurofibromatosis or cafe-au-lait spots. Repeat ultra-fine 1.0 mm scans by CT with and without contrast were obtained. Optic nerve sheath tumor was noted in the left orbit extending intracranially to lie medial and superior to the left anterior clinoid process. Possible involvement of the optic chiasm on the left side was observed. A pre- and post-Gd-DTPA was obtained to further evaluate the intracranial portion of the tumor (Fig 5B). This study demonstrated intracranial extension along the medial portion of the anterior clinoid process measuring 10 mm. Compression of the intracranial optic nerve was caused by the nodular bulbous tumor between the anterior clinoid process and the optic nerve. The left internal carotid artery was encased at the origin of the left middle cerebral artery. The tumor remained adjacent to the optic chiasm but did not encase it. On December 19, 1988, she underwent a left frontal craniotomy. A large nodular extension of the tumor was removed from the lateral aspect of the optic nerve medial to the anterior clinoid process (Fig 6). The tumor was carefully removed from the internal carotid artery. The left optic nerve canal was unroofed with the air drill and the dural sheath divided. Large amounts of meningioma were removed (Fig 7). The dural sheath was cauterized wherever possible. Postoperative recovery was uneventful. Dilantin 300 mg daily was given. Vision returned to 20/20 in the left eye and visual fields were full. Optic nerve swelling subsided. Extraocular motility remained unchanged. Repeat MR imaging with and without gadolinium demonstrated that the left intraorbital optic nerve tumor persisted unchanged (Fig 8A and B). The intracranial portion, however, had been significantly resected. A minimal amount of residual enhancing tumor remained adjacent to the left clinoid process. The chiasm and left internal carotid artery appeared normal. The patient continues to take Dilantin 300 mg daily and will be followed every 3 months. The final pathologic diagnosis is meningothelial meningioma of the left optic nerve with intracranial extension.

CASE 2

HAG, a 47-year-old right handed attorney was seen on October 20, 1986, by his local ophthalmologist because of diminishing vision in his left eye for the previous 18 months. The following was observed at that time: vision right eye, 20/20; left

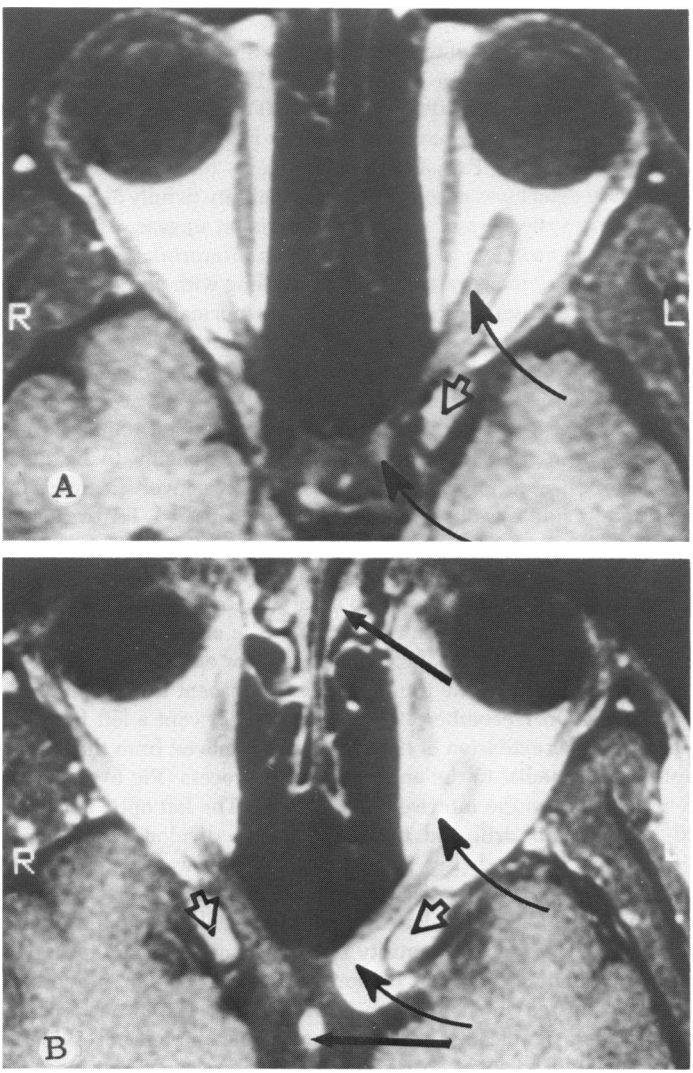


FIGURE 5

A: Case 1. Preoperative unenhanced axial MR T_1-weighted image. Note left orbital perioptic meningioma (*curved arrow*) invading intracranially through optic canal. Marrow fat of anterior clinoid process has an intense signal (*open arrow*). B: Preoperative axial MR T_1-weighted image enhanced by Gd-DTPA. Orbital and intracranial extension of the left perioptic meningioma (*curved arrow*) has an intense signal. Compare to unenhanced MR image in 5A. Note that signal of marrow fat of anterior clinoid process (*open arrow*) is isointense with the enhanced signal from the tumor. Note Gd-DTPA enhancement of the nasal mucosa and pituitary (*straight arrow*). This is a normal finding seen in MR images enhanced by Gd-DTPA.

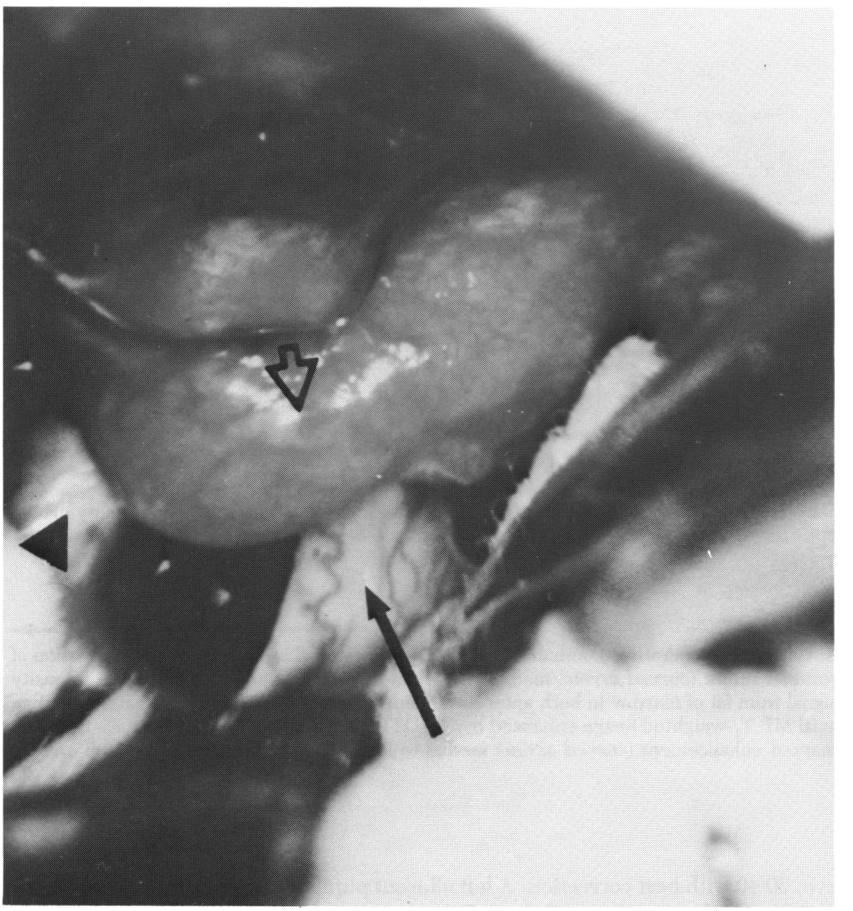


FIGURE 6
Case 1. Intraoperative photograph demonstrates left intracranial optic nerve (*arrow*) surrounded by meningioma (*open arrow*) which had invaded through the optic canal. Left internal carotid artery (*arrowhead*) is also encased by the tumor.

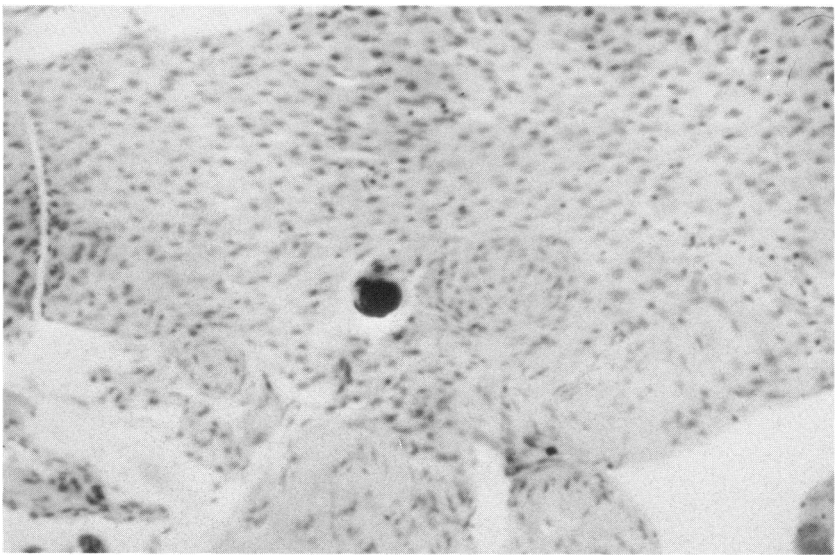
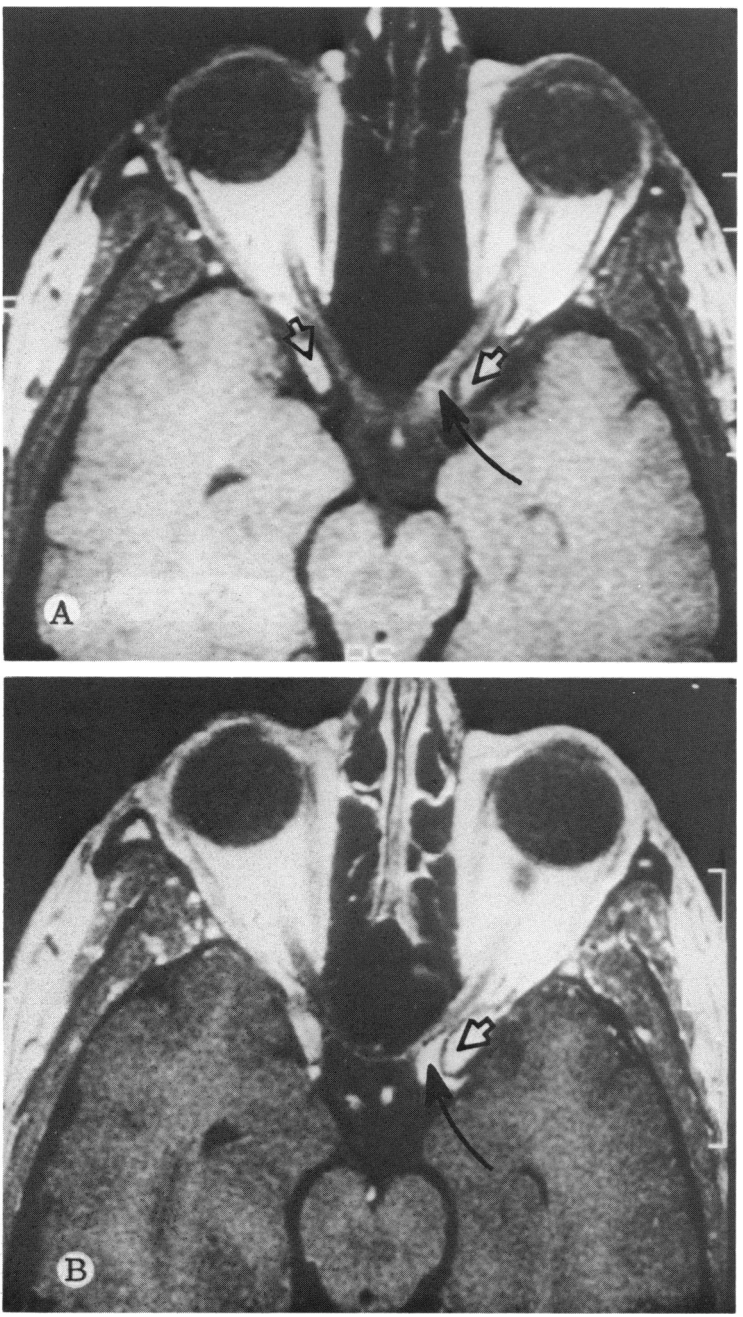


FIGURE 7
Case 1. Photomicrograph of tumor removed from intracranial extension of primary orbital optic nerve meningioma. Diagnosis is psammomatous meningothelial meningioma (magnification, ×28).

FIGURE 8 →
A: Case 1. Postoperative unenhanced axial MR T_1-weighted image. There is a suggestion of residual tumor (*curved arrow*) medial to left anterior clinoid process. Note high intensity signal from fat of marrow in both anterior clinoid processes (*open arrows*). B: Postoperative axial MR T_1-weighted image enhanced by Gd-DTPA. Presence of residual tumor is noted by marked enhancement (*curved arrow*) medial to left anterior clinoid process (*open arrow*).

eye, 20/40 with best correction. A left afferent pupillary defect was present. Three diopters of disk swelling of the left optic nerve, shunt vessels, and glistening refractile bodies were noted on the elevated surface of the disk (Fig 9A and B). The nerve appeared slightly pale. There was a left inferonasal visual field defect to large test objects (V_4) with sloping margins along the midline and absolute loss along the horizontal raphe. Central fixation was intact. He was referred to a neurologist for further evaluation. A CT scan and MR image demonstrated presence of a presumed perioptic nerve sheath meningioma confined to the left orbit (Fig 10). There was evidence of intracranial extension. An abnormal visual evoked response due to prolonged p100 was noted in the left optic nerve. The patient was then referred for neuro-ophthalmic consultation on November 20, 1986.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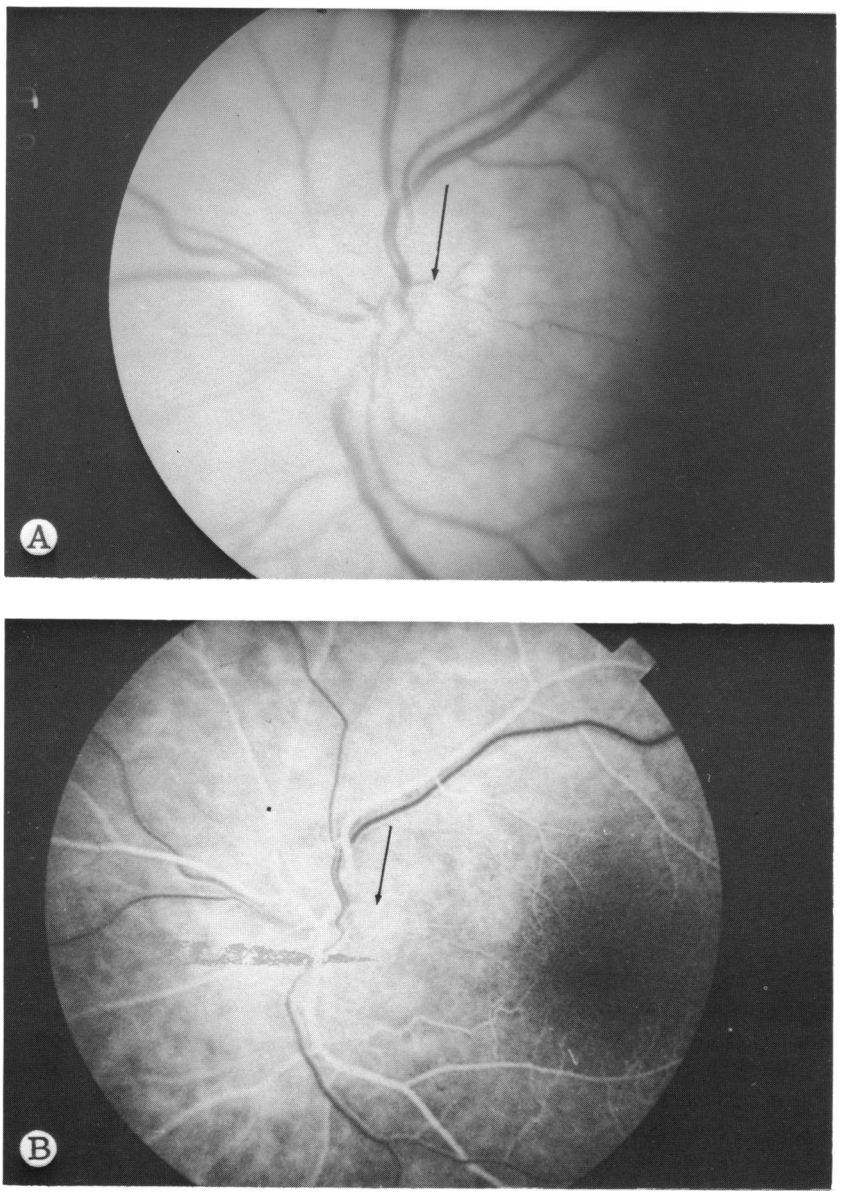

FIGURE 9
A: Case 2. Photograph of pale swollen left optic nerve in a 47-year-old man with slowly progressive loss of vision. Note shunt vessel (*arrow*) and refractile bodies on surface of disk.
B: Fluorescein angiogram demonstrates shunt vessel in A-V phase (*arrow*).

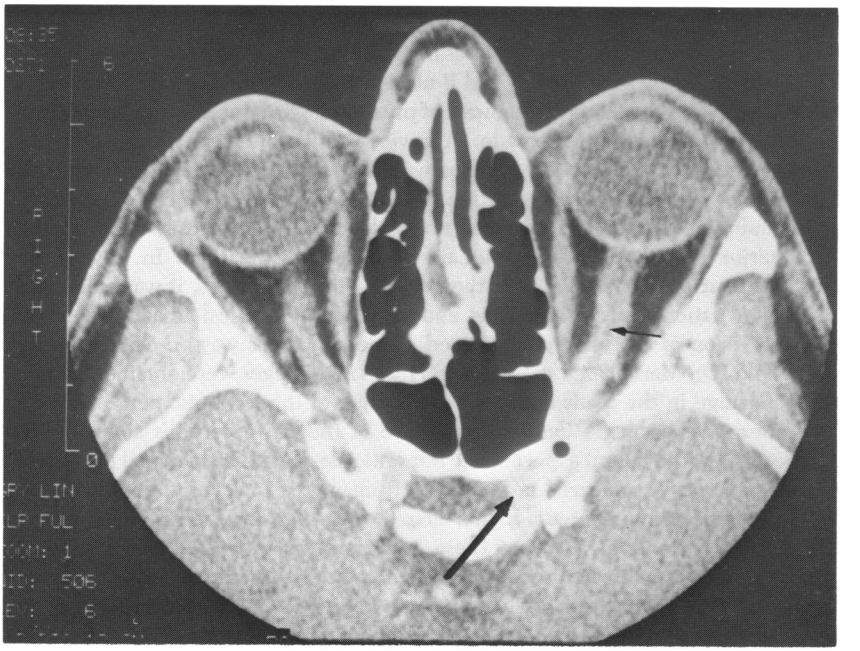

FIGURE 10
Case 2. Enhanced axial CT scan demonstrates primary orbital meningioma of left optic nerve. "Railroad track" sign (*small arrow*) is characteristic of optic nerve sheath meningioma. Note intracranial extension (*large arrow*).

Further history revealed that the patient was a former naval aviator and had had excellent vision in each eye until $2^1/_2$ years prior to presentation to the senior author (MGA). He had seen an optometrist who ordered a change in eye glasses. The patient was re-examined on three other occasions by optometrists with changes in glasses on each examination. For the previous 18 months, he had noted a decrease in peripheral vision of the left eye and occasionally bumped into objects on the left side when walking. Past medical history noted mild chronic obstructive pulmonary disease for the previous 3 years. Examination demonstrated the following: the patient is a right handed healthy man of stated age. Visual acuity: right eye, 20/20 with −0.50 sphere; left eye, 20/30 with +1.50 sphere. An afferent left pupillary defect was present. Red color vision was deficient in the left eye and normal in the right eye. Hertel exophthalmometry measurements were: right eye, 15.5 mm; left eye, 18.0 mm with an interorbital distance of 95 mm. Right hypertropia of 6 prism diopters developed on upgaze, but the patient was orthophoric in the downward and primary positions of gaze. The left optic nerve was slightly pale and elevated 4 diopters. Margins were

blurred and highly refractile bodies were noted on the elevated disk surface. Shunt vessels were noted. An inferonasal visual field defect was present in the left eye. The right eye was normal. A and B ultrasonography demonstrated a tumor of the left optic nerve. A clinical diagnosis of presumed perioptic sheath meningioma of the left optic nerve was made. A review of the medical imaging studies demonstrated a left optic nerve tumor with characteristics of meningioma confined to the left orbit. High resolution studies of the bony optic canal, sphenoid bone, and clinoid processes revealed no evidence of hyperostosis, although the left anterior clinoid appeared pneumatized more than the right. In view of the excellent central vision of 20/30 in the left eye, his sex and age, it was elected to follow the patient every 3 months and to perform medical imaging every 6 months. Vision of the left eye remained stable and there was no change in the visual field. On March 30, 1987, repeat CT and MR images were obtained. There was no change in the scans of the orbital portion of the left optic nerve. On CT scan, there was a suggestion of prominence of the left cavernous sinus and of hyperostosis of the lateral aspect of the left optic canal. The intracranial portion of the left optic nerve appeared normal on MR imaging. However, the left anterior clinoid process demonstrated a higher marrow content than the right clinoid and appeared more bulbous. Because these bony findings suggested an intracranial extension of the tumor, neurosurgical consultation was obtained. An arteriogram was performed. The internal cartoid artery appeared normal but there was a stain of the medial portion of the left cavernous sinus suggesting cavernous sinus invasion by the tumor. Radiotherapy was advised by the neurosurgical consultant. I considered the hazards of radiotherapy in the region of the intracranial optic nerve and chiasm too great a risk. On April 28, 1987, I referred the patient to Dr Edgar M. Housepin at Columbia University for another neurosurgical opinion. He reviewed all of the neurodiagnostic studies. Because the left optic canal was abnormal on the CT scans, he obtained polytomography of the optic canals and concluded that there was no enlargement or hyperostosis. He advised further observation and no radiotherapy. The patient was seen every 3 months without any clinical change in his vision or physical evaluation. On November 8, 1988, high resolution CT scans were obtained. Evidence of optic canal extension was observed (Fig 11A). MR images with and without Gd-DTPA, were obtained (Figs 11B to 12B). Gadolinium enhanced images showed extension of the tumor through the optic canal bulging into the suprasellar space just superior to the left anterior clinoid process. The bulk of the tumor appeared above the clinoid measuring 1.5 cm. It encased the supracavernous portion of the left internal carotid artery near the M1 segment of the middle cerebral artery. The patient has been scheduled for surgery, but it has not been performed at this writing.

CASE 3

AU, a 71-year-old right handed woman was first seen in consultation by the senior author (MGA) on June 17, 1980, at the request of her internist because of blindness in the left eye of undetermined cause. The patient first became aware of poor vision in her left eye by reason of a driver's test in December, 1970. There

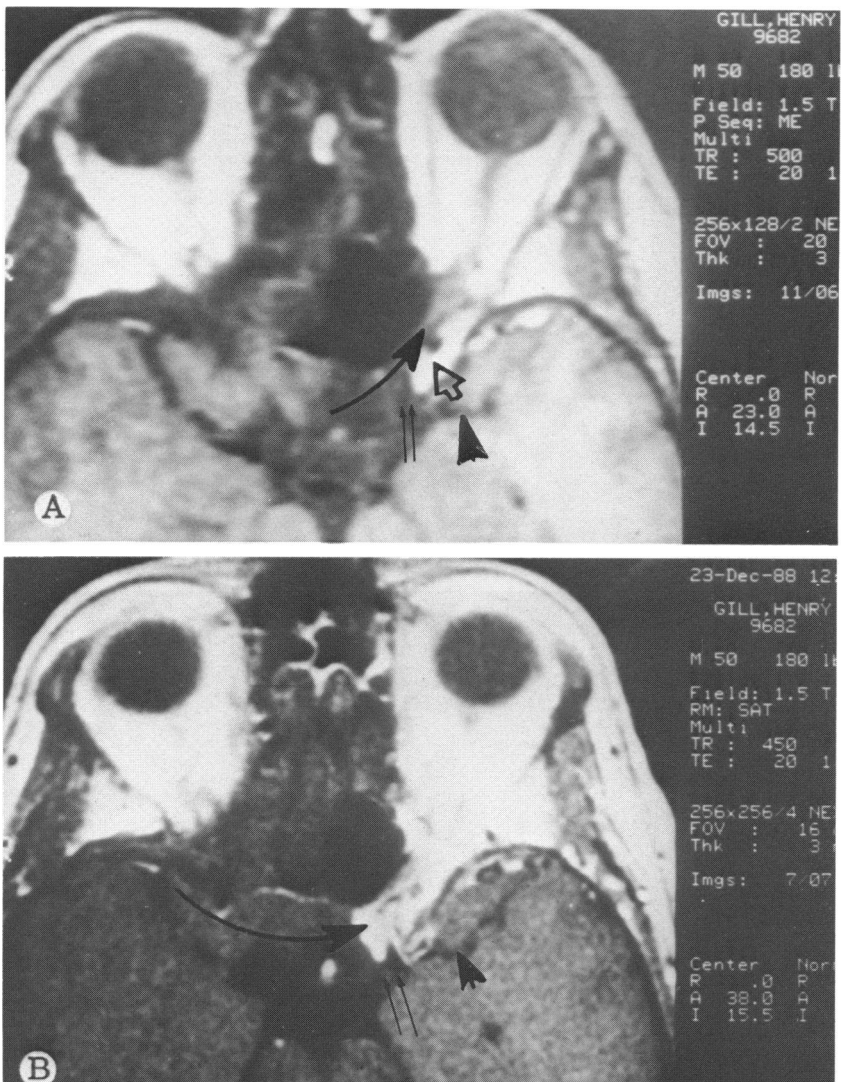

FIGURE 11

A: Case 2. Unenhanced axial MR T_1-weighted image demonstrates apparent intracranial extension of meningioma of left optic nerve (*curved arrow*). Tumor is isointense with brain tissue. Note intense signal from marrow fat of left anterior clinoid process (*open arrow*). Internal carotid artery (*2 small arrows*) and middle cerebral artery (*arrowhead*) are well differentiated. B: Axial MR T_1-weighted image enhanced by Gd-DTPA. Note markedly enhanced signal of the meningioma (*curved arrow*). Internal carotid artery (*2 small arrows*) and middle cerebral artery (*arrowhead*) are well differentiated. Enhancement of nasal mucosa and pituitary stalk are noted.

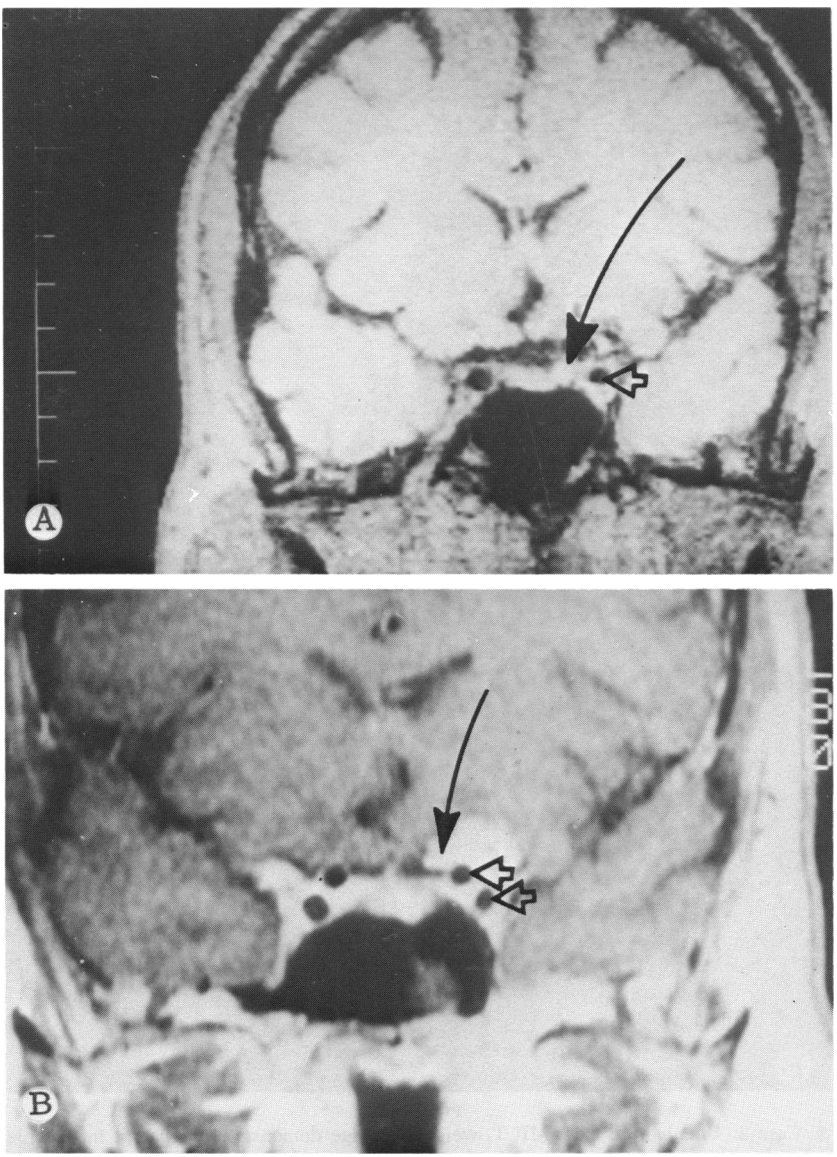

FIGURE 12

A: Case 2. Unenhanced coronal MR T_1-weighted image demonstrates left internal carotid artery (*open arrow*) with meningioma lying medially (*curved arrow*). Tumor is isointense when compared to brain tissue. B: Coronal MR T_1-weighted image enhanced by Gd-DTPA demonstrates meningioma (*curved arrow*) with an intense signal lying medial to and extending above the internal carotid artery (*2 open arrows*).

had been no pain, headaches, or diplopia. She was referred to an ophthalmologist by her internist for evaluation. On January 19, 1971, visual acuity was noted as follows: right eye, 20/20; left eye, 20/60. Bilateral Duane's retraction syndrome was noted as well as early posterior subcapsular lens changes. A chart review showed that no investigation was carried out for the poor vision in the left eye. On February 4, 1972, she was seen again by her ophthalmologist complaining of painless progressive loss of vision in the left eye. The following was observed: vision: right eye, 20/20; left eye, finger counting at 1 m. The ocular examination was unchanged except for blurred nasal disk margin of the left optic nerve and "edema in the macular and paramacular area." This was interpreted as retinal inflammation and a systemic workup was instituted. A bladder infection was found and treated. By April 10, 1972, the infection was cured, but the left eye vision remained counting fingers and the examination was unchanged. Routine skull x-ray was normal. Prednisone 100 mg every other day for 10 days was given without improvement. The patient sought further consultation at the Massachusetts Eye and Ear Infirmary where she was seen by Dr Shirley Wray. The following was observed: visual acuity: right eye, 20/30; left eye, counting fingers at 0.5 m in only the peripheral visual field. The visual field was full in the right eye, but in the left eye there was a large very dense central scotoma. There was a left afferent pupillary defect. Bilateral Duane's retraction syndrome was noted. The right optic disc was normal. The left optic disc was slightly elevated with blurred margins and gliosis. The macular region showed a pigment disturbance. All hematologic studies were normal. Skull x-rays including polytomography were normal; in particular, the optic canals, optic foramina, sphenoid ridges, and sella turcica showed no abnormalities. Technetium-99 brain scan was normal. A pneumoencephalogram was normal. Cerebrospinal fluid obtained at the time of the pneumoencephalogram was normal. A percutaneous carotid arteriogram was normal. A fluorescein angiogram demonstrated changes which were interpreted as an interpapillary drusen in the left eye. Dark streaks temporal to the left disk appearing like "small angioid streaks" were noted. The final impression of these neurodiagnostic studies was noncompressive left optic neuropathy. An inflammatory cause was suspected. Another course of prednisone 60 mg per day for 7 days was given without improvement. The patient was seen each year by her internist but not by an ophthalmologist until her visit to MGA on June 17, 1980. A note in the internist's records on April 11, 1977, however, observed that vision in the right eye remained unchanged, but that the left eye was blind. There were no further eye changes observed until the neuro-ophthalmology consultation of June 17, 1980, when the following was noted: visual acuity: right eye, 20/20 with best correction; left eye, no light perception. Bilateral Duane's syndrome was present. There was prolapse of each lacrimal gland. There was a 4+ left afferent pupillary defect. Pseudo-exfoliation of the left lens capsule was present. Intraocular pressure was 14 mm Hg by applanation tonometry. Gonioscopy demonstrated open anterior chamber angles. The right optic nerve was normal. The left optic nerve was atrophic with blurred margins; cilioretinal shunt vessels were present on the surface (Fig 13A). Drusen could not be identified. Striae were present across the

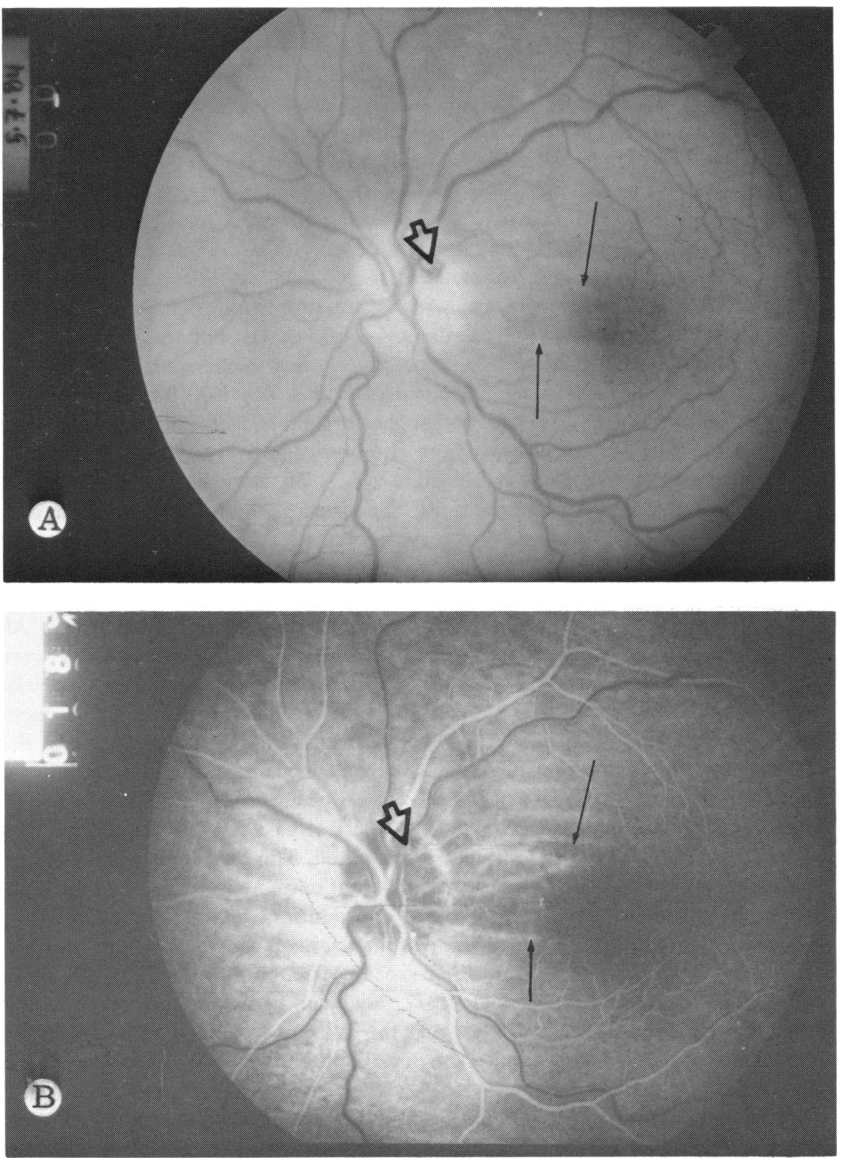

FIGURE 13

A: Case 3. Photograph of atrophic left optic nerve with shunt vessels on surface of disk (*open arrow*) and choroidal folds in posterior pole (*arrows*). Choroidal folds may be a sign of optic nerve meningioma. B: Fluorescein angiogram demonstrates choroidal folds across posterior pole (*arrows*) and shunt vessels on surface of optic disk (*open arrow*).

posterior pole and in the macula (Fig 13B). Hertel exophthalmometry measured right eye 16.0 mm; left eye 15.5 mm; interorbital distance 95 mm. Ultrasonography demonstrated that the left optic nerve was larger than the right. CT was performed in both coronal and transaxial planes with and without contrast infusion. The left optic nerve appeared wider than the right and a negative defect was present on coronal section. An enhanced density was seen at the superior aspect of the anterior clinoid process. Because initial scanning was done by 4 mm cuts, thinner sections were requested. Thin scans were than obtained employing 2 mm collimation. These scans showed an enhancing mass extending through the left optic canal into the area of the left anterior clinoid process and suprasellar region. The chiasm appeared uninvolved. The impression was "left optic nerve meningioma." Neurosurgical consultation was sought. It was agreed that this probably represented a long-standing meningioma and that surgery was not indicated in view of the patient's age. At first the patient was followed clinically every 3 months and CT scans were performed every 6 months. As new generations of CT scanners were obtained, images improved. It was felt that there was little if any growth of the tumor. Visual field examination of the right eye remained unchanged. Visits were extended to every 6 months. In 1986, CT scans were augmented by MR images to better view the chiasm. These examinations demonstrated that CT scans demonstrated the tumor better than unenhanced MR images in which the tumor appeared isodense with the surrounding brain (Fig 14A). A CT scan was obtained on August 12, 1988 that suggested extension slightly across the midline to the right without involvement of the chiasm or right optic nerve. On November 7, 1988, Gd-DTPA enhanced MR images were obtained that demonstrated the tumor apparently arising from the left anterior clinoid process or tuberculum. It crossed the midline to the right side and was sharply defined by the right optic nerve and chiasm. The right optic nerve and optic chiasm, however, were not infiltrated. The patient continues to be followed, without surgery (Figs 14B to 15B).

CASE 4

LW, a 58-year-old right handed woman was seen for neuro-ophthalmic consultation on August 1, 1980 because of painless loss of vision in her left eye for 3 months. For the previous 3 years, she had been aware of protrusion of the left eye. She was seen 1 month previously by her local ophthalmologist who referred her to a retinal specialist for evaluation of the macula. Examination revealed the following: visual acuity right eye, 20/25; left eye, 20/50 – 2 with best correction. Hertel exophthalmometry measured right eye, 18 mm; left eye, 22 mm with an interorbital distance of 95 mm. There was limited elevation of the left eye. Pupillary reactions were normal, but brightness was diminished in the left eye by 50% when viewing the light of an ophthalmoscope. The left optic nerve disk appeared pale compared to the right. There was no macular pathology. A relative central scotoma was present in the right eye. The left eye displayed dense paracentral arcuate defects. Routine skull x-rays with polytomography demonstrated hyperostosis of the left anterior clinoid process and the greater wing of the left sphenoid

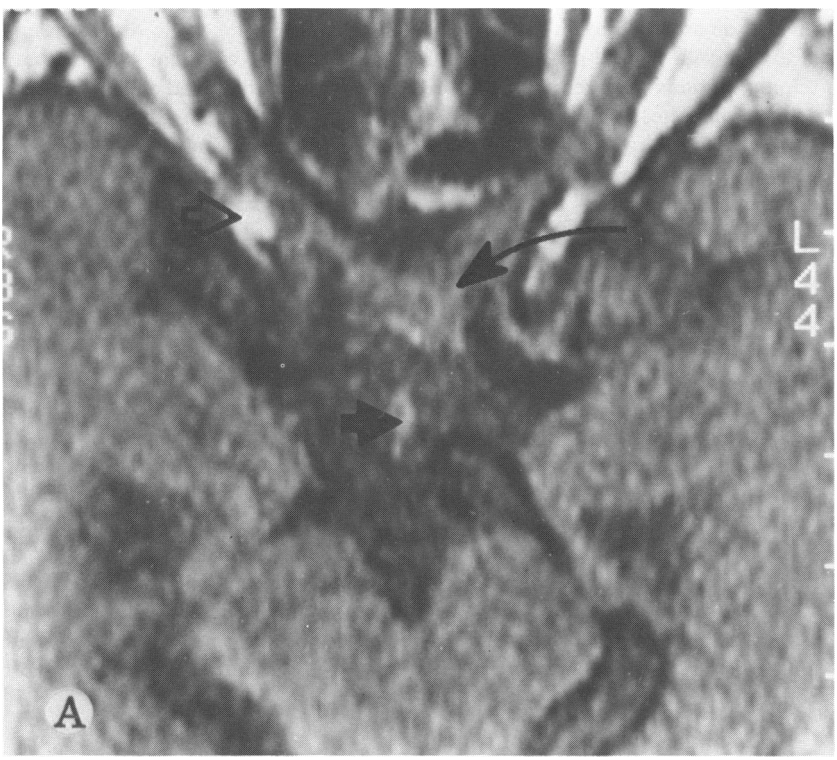

FIGURE 14

A: Case 3. Unenhanced axial MR T_1-weighted image. Meningioma (*curved arrow*) is isointense in comparison to brain tissue. Pituitary (*arrowhead*) has more intense signal than normal brain tissue. Compare with enhanced signal in 3B. Note normal fat in marrow of anterior clinoid (*open arrow*) is isointense with enhanced signal from tumor in 3B.

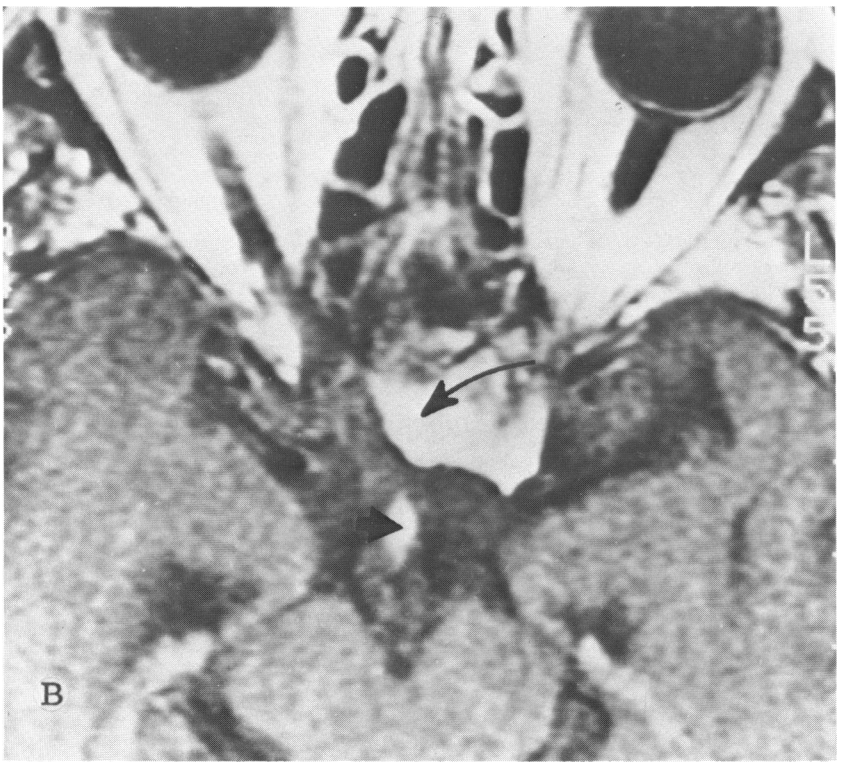

FIGURE 14 (CONT'D)
B: Axial MR T_1-weighted image enhanced by Gd-DTPA clearly differentiates meningioma (*curved arrow*) from surrounding brain tissue. Note normal enhancement of signal from pituitary and nasal mucosa.

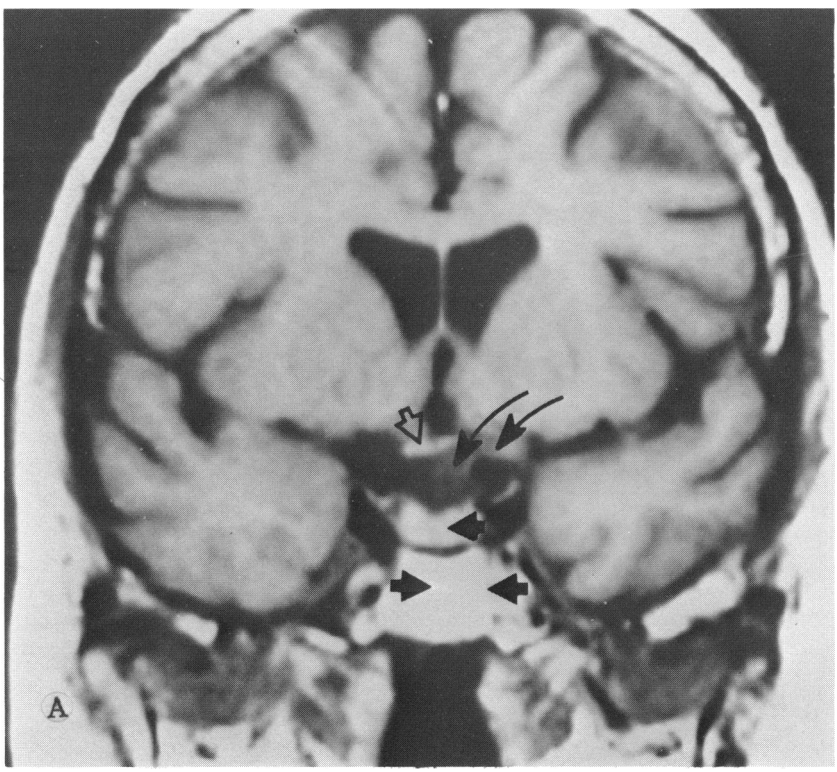

FIGURE 15

A: Case 3. Unenhanced coronal MR T_1-weighted image meningioma (*curved arrows*) is isointense in comparison to brain tissue. Note slightly distorted chiasm (*open arrow*). Note normal intense signal of marrow fat in sphenoid bone (*arrowheads*).

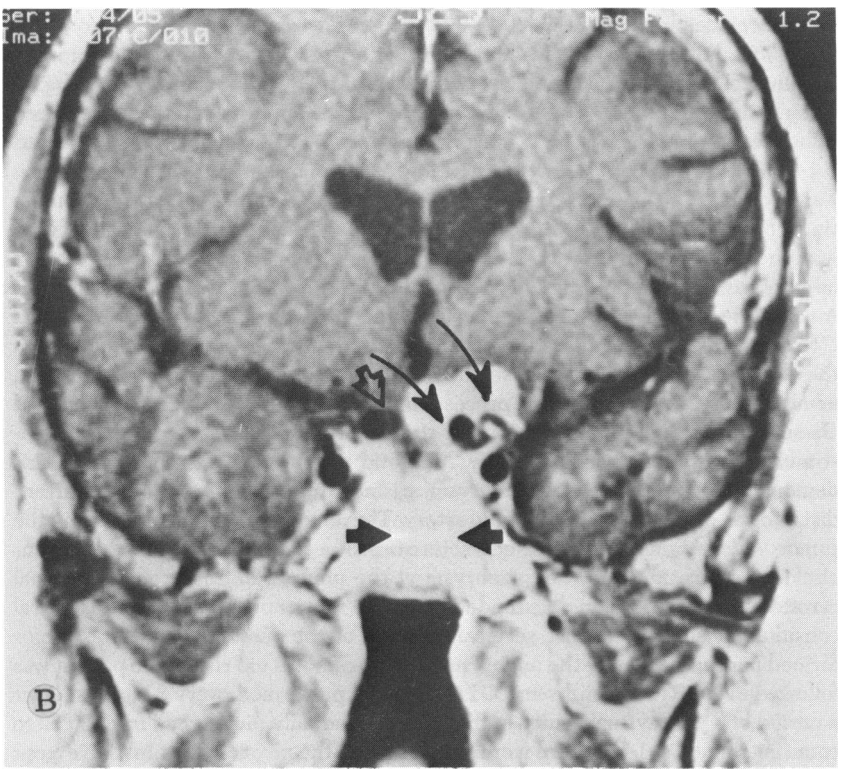

FIGURE 15 (CONT'D)
B: Coronal MR T_1-weighted image enhancced by Gd-DTPA. Meningioma is markedly enhanced (*curved arrows*). Note signal from marrow fat of sphenoid bone (*arrowheads*) is isointense with Gd-DTPA enhanced signal of the meningioma.

bone. A CT scan confirmed these findings. She was then referred to the senior author (MGA) for further study and management.

Neuro-ophthalmic evaluation elicited further past medical history. Hypertensive vascular disease was controlled by medication. A total thyroidectomy had been performed 18 years previously for a malignant nodule. She had been taking thyroid replacement therapy since. Examination revealed visual acuity of right eye, 20/20; left eye, 20/60. Swelling of the left lower lid was present. There was inability to elevate the left eye and retraction of the left upper eyelid developed when attempting upgaze (Collier's sign). Hertel exophthalmometry measured right eye, 16 mm; left eye, 19.5 mm with interorbital distance 100 mm. There was a 4+ afferent pupillary defect in the left eye. Diminished left corneal sensation was present. The patient carried her chin in an elevated position to maintain single binocular vision. Vertical diplopia developed in the primary position of gaze with the head straight and increased upon upgaze. The left optic disk was pale. An inferior paracentral scotoma to I_2 was present in the visual field of the right eye. An absolute inferior central scotoma was noted in the field of the left eye. Repeat CT scan with a fourth generation instrument demonstrated an enhancing mass in the left parasellar region that seemed to arise from the cavernous sinus and involve the left anterior clinoid process, the floor of the middle cranial fossa and Gasserian ganglion. Thyroid function studies were normal except for thyroid-stimulating hormone which was low. A carotid arteriogram was performed. This demonstrated presence of an aneurysm arising from the internal carotid artery distal to the origin of the ophthalmic artery. The aneurysm was surrounded by the tumor which stained with the characteristics of a meningioma. The tumor encircled the carotid artery and aneurysm at the point of entrance of the internal carotid into the skull and extended into the cavernous sinus. Neurosurgical consultation advised against surgical removal of the tumor because the tumor formed part of the wall of the aneurysm making removal too risky. The patient was followed every 3 to 4 months and CT scans were performed every 6 months. Over a period of 8 years visual acuity in the left eye gradually diminished from 20/60 to counting fingers at 1 m. There were no changes in the physical examination except for the visual field of the left eye. The central scotoma which had originally been most dense inferiorly gradually encompassed the central 20°. The vision and visual field of the right eye remained unchanged. Exophthalmometry and extraocular motility remained unchanged. CT examinations, however, showed slow growth of the tumor. Extension into the sella turcica was first noted on March 9, 1987. MR images were first obtained on January 22, 1985, but added very little to imaging of the progressive growth noted by CT scans. A CT scan performed on July 17, 1988 noted extension of the intracavernous mass into the sella turcica and planum anteriorly and into the retroclival region posteriorly. MR images confirmed this (Fig 16A and B). Pituitary function studies were normal. Neurosurgical consultation advised continued close observation since there was little to be gained by surgery except to save vision of the right eye. If evidence occurred to indicate loss of vision in the right eye surgery would then be advised. Gadolinium-enhanced MR imaging confirmed these findings and demonstrated greater exten-

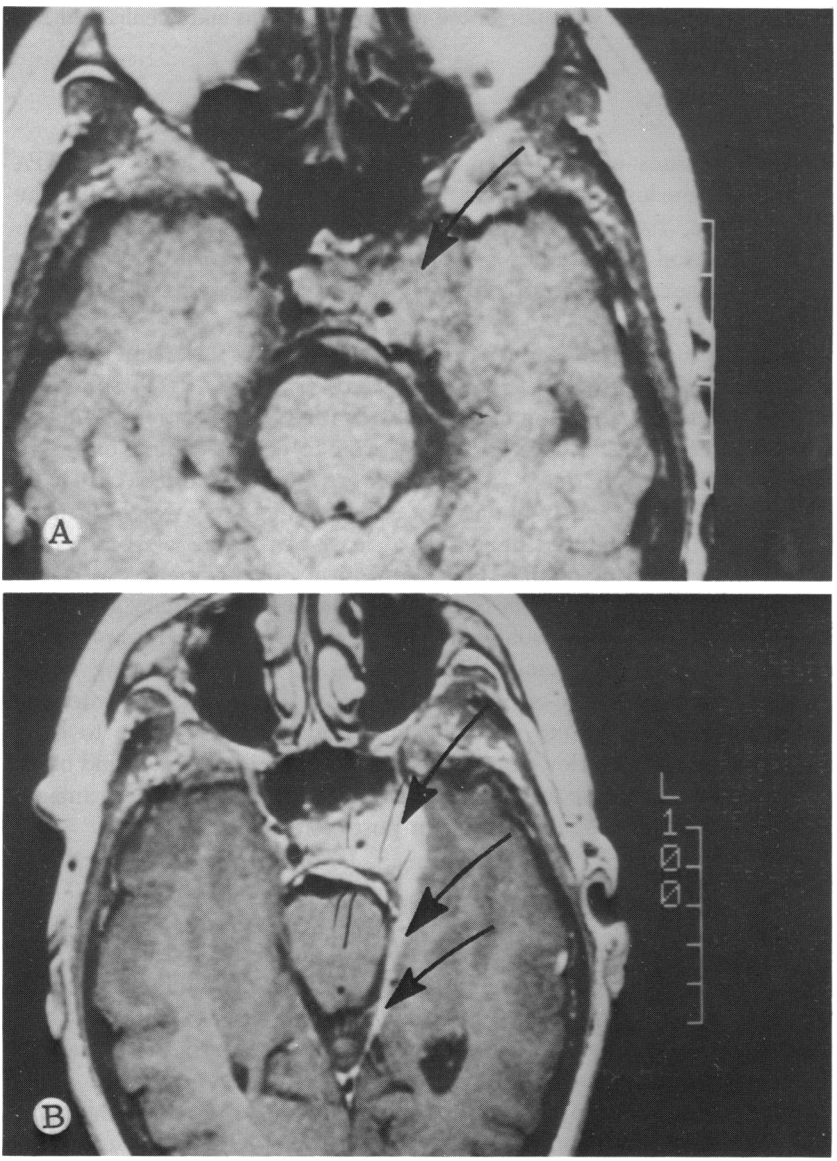

FIGURE 16

A: Case 4. Unenhanced axial MR T_1-weighted image. Parasellar tumor is identified (*curved arrow*). It extends across sella turcica and is isointense in comparison to brain tissue. B: Axial MR T_1-weighted image enhanced by Gd-DTPA. Note marked enhancement of meningioma with posterior "en plaque" extension along tentorial incisura (*curved arrows*). Also note extension across sella. Compare to 16A.

sion of the tumor than was appreciated from the CT scans and unenhanced MR images.

MATERIALS AND METHODS

Forty-seven cases of meningioma have been evaluated with Gd-DTPA (Magnavist, Berlex, Inc) since the release of the contrast agent by the FDA in June, 1988. These cases represent almost 10% of the 440 Gd-DTPA-enhanced MR studies performed. Fourteen of the meningiomas affected the anterior visual pathways. Four were selected as representatives to present in this report.

All MR examinations were performed using a 1.5 Tesla superconductive imager. T_2-weighted, T_1 weighted and "balanced" T_2/T_1 spin-echo pulse sequences were obtained without contrast in all patients. Images were generated in at least two planes prior to contrast injection (axial, coronal, sagittal or oblique). All sections were 3 to 5 mm thick. Gd-DTPA was injected intravenously in doses of 0.1 m Mol/kg body weight (usually 10 to 18 ml). T_1 weighted images in at least two planes were obtained immediately after injection. The adequacy of enhancement was determined by noting the intensity of enhancement of the pituitary gland and cavernous sinuses which usually enhance intensely. These areas provided a local enhancement "standard" to which abnormal enhancing tissue could be compared. Fat of bone marrow normally has a high intensity signal. Care was taken to note this high-signal intensity due to bone marrow fat in the anterior clinoid processes, tuberculum sellae and other nonaerated areas of the sphenoid bone and to distinguish these images from abnormally enhancing tissue in post-contrast images.

RESULTS

Analysis of four patients with meningiomas of the anterior visual pathways is reported from a group of 14 cases detected by gladolinium-enhanced MR images during the first 9 months of our experience with this new technique. These four cases were selected as representative of the various changes characteristic of enhanced MR images.

In case 1, an intraorbital optic nerve meningioma and intracranial extension had been demonstrated by CT scanning. The status of involvement of the chiasm and left internal carotid artery was demonstrated by gladolinium-enhanced MR images. The tumor encased the internal carotid artery at the origin of the middle cerebral artery but the chiasm appeared uninvolved. The optic canal was invaded by the tumor. Craniotomy and surgical exploration of the left anterior visual pathway demon-

strated the meningioma lying the exact position elicited by the enhanced MR images (Figs 6 and 7). Postoperative enhanced MR images demonstrated residual tumor (Fig 8A and B).

In case 2, CT scans and unenhanced MR images were inconclusive in demonstrating intracranial extension. Gd-DTPA-enhanced MR imaging showed intracranial extension and involvement identical to case 1. Surgery has been scheduled (Figs 11B and 12B).

Case 3 has had a long-standing tumor of the left optic nerve undetected by original studies in 1972. CT scanning with a fourth generation scanner demonstrated a tumor characteristic of meningioma of the left anterior clinoid process, optic nerve of the orbit, optic canal and anterior cranial fossa but did not reveal extension into the chiasm or right optic nerve. Gd-DTPA-enhanced MR imaging showed extension over the chiasm toward the right optic nerve (Fig 15A and B).

DISCUSSION

The purpose of this report is to call attention to the improved sensitivity of MR imaging in detecting meningiomas of the anterior visual pathways when enhanced with the paramagnetic agent Gd-DTPA.

Gadolinium is a rare metallic earth element which appears on the periodic table as atomic number 64 with atomic weight 157.25 (Table I). When chelated with DTPA it forms a nontoxic paramagnetic compound which when injected intravenously is normally restricted to the blood and interstitium.[9-12] It was developed for clinical use by Weinmann et al[6,13] in 1984 in the research laboratories of AG Schering, West Germany.

In healthy intra-axial brain tissue, the tight blood-brain barrier (BBB) prevents Gd-DTPA from leaving the vascular compartment. When the BBB is broken, however, as in infarction, tumor, inflammation or trauma, the unpaired electrons of this complex agent dampen the spins of the free protons with which it comes in contact. This results in reduction of T_1 and T_2 relaxation times enhancing MR signals in the area where the agent accumulates. It has also been shown to penetrate extra-axial brain lesions that lack a BBB.[7] It is this latter characteristic which makes Gd-DTPA so valuable in enhancing MR images of extra-axial meningiomas in the orbitocranial junction and skull base.

Extra-axial tumors such as meningiomas of the anterior visual pathways have signal characteristics which make them isointense with normal brain tissue on routine unenhanced MR imaging.[7] This factor together with their small size and plaque-like configuration have frustrated neuroradiologists in making a proper diagnosis. However, because these tumors

TABLE I: PERIODIC TABLE OF THE ELEMENTS

1a	2a	3b	4b	5b	6b	7b	8	1b	2b	3a	4a	5a	6a	7a	0	Orbit
1 +1 H −1 1.00794±7 1		KEY TO CHART Atomic Number → 50 +2 ← Oxidation States Symbol → Sn +4 1981 Atomic Weight → 118.69±3 2 8 18 18 4 ← Electron Configuration													2 0 He 4.00260 2	K
3 +1 Li 6.941±3 2-1	4 +2 Be 9.01218 2-2									5 +3 B 10.81 2-3	6 +2 +4 C −4 12.011 2-4	7 +1 +2 +3 +4 +5 −1 −2 −3 N 14.0067 2-5	8 −2 O 15.9994±3 2-6	9 −1 F 18.998403 2-7	10 0 Ne 20.179 2-8	K-L
11 +1 Na 22.98977 2-8-1	12 +2 Mg 24.305 2-8-2	Transition Elements							Transition Elements	13 +3 Al 26.98154 2-8-3	14 +2 +4 −4 Si 28.0855±3 2-8-4	15 +3 +5 −3 P 30.97376 2-8-5	16 +4 +6 −2 S 32.06 2-8-6	17 +1 +5 +7 −1 Cl 35.453 2-8-7	18 0 Ar 39.948 2-8-8	K-L-M
19 +1 K 39.0983 -8-8-1	20 +2 Ca 40.08 -8-8-2	21 +3 Sc 44.9559 -8-9-2	22 +2 +3 +4 Ti 47.88±3 -8-10-2	23 +2 +3 +4 +5 V 50.9415 -8-11-2	24 +2 +3 +6 Cr 51.996 -8-13-1	25 +2 +3 +4 +7 Mn 54.9380 -8-13-2	26 +2 +3 Fe 55.847±3 -8-14-2	27 +2 +3 Co 58.9332 -8-15-2	28 +2 +3 Ni 58.69 -8-16-2	29 +1 +2 Cu 63.546±3 -8-18-1	30 +2 Zn 65.38 -8-18-2	31 +3 Ga 69.72 -8-18-3	32 +2 +4 Ge 72.59 -8-18-4	33 +3 +5 −3 As 74.9216 -8-18-5	34 +4 +6 −2 Se 78.96±3 -8-18-6	35 +1 +5 −1 Br 79.904 -8-18-7	36 0 Kr 83.80 -8-18-8	-L-M-N
37 +1 Rb 85.4678±3 -18-8-1	38 +2 Sr 87.62 -18-8-2	39 +3 Y 88.9059 -18-9-2	40 +4 Zr 91.22 -18-10-2	41 +3 +5 Nb 92.9064 -18-12-1	42 +6 Mo 95.94 -18-13-1	43 +4 +6 +7 Tc (98) -18-13-2	44 +3 Ru 101.07±3 -18-15-1	45 +3 Rh 102.9055 -18-16-1	46 +2 +4 Pd 106.4 -18-18-0	47 +1 Ag 107.8682±3 -18-18-1	48 +2 Cd 112.41 -18-18-2	49 +3 In 114.82 -18-18-3	50 +2 +4 Sn 118.69±3 -18-18-4	51 +3 +5 −3 Sb 121.71±3 -18-18-5	52 +4 +6 −2 Te 127.60±3 -18-18-6	53 +1 +5 +7 −1 I 126.9045 -18-18-7	54 0 Xe 131.29±3 -18-18-8	-M-N-O
55 +1 Cs 132.9054 -18-8-1	56 +2 Ba 137.33 -18-8-2	57* +3 La 138.9055±3 -18-9-2	72 +4 Hf 178.49±3 -32-10-2	73 +5 Ta 180.9479 -32-11-2	74 +6 W 183.85±3 -32-12-2	75 +4 +6 +7 Re 186.207 -32-13-2	76 +3 +4 Os 190.2 -32-14-2	77 +3 +4 Ir 192.22±3 -32-15-2	78 +2 +4 Pt 195.08±3 -32-16-2	79 +1 +3 Au 196.9665 -32-18-1	80 +1 +2 Hg 200.59±3 -32-18-2	81 +1 +3 Tl 204.383 -32-18-3	82 +2 +4 Pb 207.2 -32-18-4	83 +3 +5 Bi 208.9804 -32-18-5	84 +2 +4 Po (209) -32-18-6	85 At (210) -32-18-7	86 0 Rn (222) -32-18-8	-N-O-P
87 +1 Fr (223) -18-8-1	88 +2 Ra 226.0254 -18-8-2	89** +3 Ac 227.0482 -18-9-2	104 +4 Rf (260) -32-10-2	105 Ha (260) -32-11-2	106 (263) -32-12-2											O P Q

*Lanthanides	58 +3 +4 Ce 140.12 -20-8-2	59 +3 Pr 140.9077 -21-8-2	60 +3 Nd 144.24±3 -22-8-2	61 +3 Pm (145) -23-8-2	62 +2 +3 Sm 150.36±3 -24-8-2	63 +2 +3 Eu 151.96 -25-8-2	64 +3 Gd 157.25±3 -25-9-2	65 +3 Tb 158.9254 -27-8-2	66 +3 Dy 162.50±3 -28-8-2	67 +3 Ho 164.9304 -29-8-2	68 +3 Er 167.26±3 -30-8-2	69 +3 Tm 168.9342 -31-8-2	70 +2 +3 Yb 173.04±3 -32-8-2	71 +3 Lu 174.967 -32-9-2	N O P
**Actinides	90 +4 Th 232.0381 -18-10-2	91 +5 +4 Pa 231.0359 -20-9-2	92 +3 +4 +5 +6 U 238.0289 -21-9-2	93 +3 +4 +5 +6 Np 237.0482 -22-9-2	94 +3 +4 +5 +6 Pu (244) -24-8-2	95 +3 +4 +5 +6 Am (243) -25-8-2	96 +3 Cm (247) -25-9-2	97 +3 +4 Bk (247) -27-8-2	98 +3 Cf (251) -28-8-2	99 +3 Es (252) -29-8-2	100 +3 Fm (257) -30-8-2	101 +2 +3 Md (258) -31-8-2	102 +2 +3 No (259) -32-8-2	103 +3 Lr (260) -32-9-2	O P Q

Numbers in parentheses are mass numbers of most stable isotope of that element.

lack a BBB and have a rich blood supply, they are ideal for identification with paramagnetic agents such as Gd-DTPA.

It is necessary to establish parameters of normal anatomy with routine MR imaging before undertaking interpretation of gladolinium-enhanced images. For example, normal marrow fat of the anterior clinoid process appears isointense with gladolinium-enhanced MR images. In addition, Gd-DTPA enhances certain normal tissues. This has been fully described by Kilgore et al.[12] In the orbit, enhancement of the choroid is evident, but *not* the optic nerve, retrobulbar fat or extraocular muscles. The signal intensity of the mucosa of the paranasal sinuses is reported to enhance markedly to an average increase of 150%. The pituitary gland and infundibulum are estimated to normally enhance an average of 60% to 80%. A similar degree of enhancement of the cavernous sinus is described. The choroid plexus in the lateral ventricles and fourth ventricle increase in sensitivity an average of 70%.

Knowledge of these findings enables the neuroradiologist to differentiate abnormal enhancement from that of normal tissues.

TABLE II: GADOLINIUM ENHANCED MR IMAGING IN MENINGIOMAS OF ANTERIOR VISUAL PATHWAYS

PATIENT NO.	SEX	AGE (YRS)	LOCATION
1	F	65	Right parasella, cavernous sinus, chiasm, optic tract
2	F	43	Right intraorbital optic nerve, optic canal, intracranial optic nerve
3	F	26	Left intraorbital optic nerve, optic canal, intracranial optic nerve, anterior clinoid process
4	M	60	Planum, right intracranial optic nerve
5	M	49	Left intraorbital optic nerve, optic canal, intracranial optic nerve, anterior clinoid process
6	F	49	Right intraorbital optic nerve, optic canal
7	M	69	Right parasella, cavernous sinus, chiasm, intracranial optic nerve, cerebello-pontine angle
8	F	42	Right intraorbital optic nerve, optic canal
9	F	52	Right parasella, cavernous sinus, chiasm, intracranial optic nerve
10	F	79	Left intraorbital optic nerve, optic canal, intracranial optic nerve, anterior clinoid process
11	F	62	Left intracranial optic nerve, optic canal, anterior clinoid process
12	F	66	Left parasella, cavernous sinus, intracranial optic nerve, optic canal, sella turcica, clivus, tentorium
13	F	51	Right intraorbital optic nerve meningioma with intracranial extension through optic canal
14	F	60	Meningioma right greater wing of sphenoid, right orbit, right optic nerve

TABLE III: COMPARISON OF CT AND MRI IN DETECTION OF MENINGIOMAS OF ANTERIOR VISUAL PATHWAYS

LOCATION OF MENINGIOMA	CT	MRI	MRI WITH GD-DTPA
Intraorbital optic nerve	Good	Poor	Poor
Intracanalicular optic nerve	Poor	Good	Excellent
Intracranial optic nerve	Good	Poor	Excellent
Chiasm	Good	Good	Excellent
Bone (hyperostosis)	Excellent	Poor	Poor

In this series of 14 meningiomas of the anterior visual pathways, identification of the tumor was made by routine unenhanced MR images in all cases, but the true extent of involvement was not fully appreciated. Gd-DTPA-enhanced images were striking on T_1 weighted images in all cases herein reported. Questions regarding chiasmal involvement, extension to the opposite optic nerve and spread to adjacent structures were defined in all cases after enhancement. Four cases from this group are reported in detail. They were selected to illustrate certain diagnostic points and to demonstrate the value of this new diagnostic points and to demonstrate the value of this new diagnostic technique (Table II).

In case 1, Gd-DTPA-enhanced MR images clearly demonstrated the intracranial extension of an optic nerve meningioma. Although the lesion was well-demonstrated on both CT (Fig 4) and unenhanced MR imaging (Fig 5A) the enhanced MR images (Fig 5B) revealed the degree of involvement better, and aided in planning the neurosurgical approach. Removal of the intracanalicular and intracranial portion of the tumor was accomplished to preserve life and to restore vision (Figs 6 and 7). The orbital portion of the tumor was left undisturbed, as is our custom. The patient will be re-evaluated clinically every 3 months and by Gd-DTPA-enhanced MR every 6 months. Postoperative enhanced MR imaging demonstrates residual tumor at this time (Fig 8A and B).

In case 2 (Fig 9A and B), efforts to demonstrate extension of the optic nerve sheath tumor through the optic canal were inconclusive with enhanced high resolution CT scans (Fig 10) and unenhanced MR images (Figs 11A and 12A). Arteriography demonstrated a stain of the medial portion of the cavernous sinus suggesting intracranial extension. Gd-DTPA-enhanced MR images showed intracranial extension (Figs 11B and 12B). Because Gd-DTPA was not available when the initial studies were performed in 1986, it is probable that intracranial extension existed when the patient was first seen. In this case, visual acuity is still good even though there is a visual field defect. The enhanced MR images demonstrate considerable intracranial involvement analogous to case 1. This

patient has been scheduled for a neurosurgical approach similar to that performed in case 1.

In case 3, a long-standing tumor was present for almost 20 years. At first, it was undetected by the current modalities of neuro-diagnostic medical imaging. An initial CT scan with third generation instruments (Fig 1A and B) demonstrated a presumed meningioma of the left optic nerve. Whether or not it arose intracranially and extended into the orbit was undetermined by the initial studies.

This tumor must have been present at the time of the initial eye examination in 1971. The retinal changes noted at that time have been demonstrated to be choroidal folds with striae in the posterior pole visible on more recent fluorescein angiography (Fig 13A and B). The fluorescein studies performed at Massachusetts Eye and Ear Infirmary in 1972 have been lost and are not available for study. However, the verbal description of Doctor Clement L. Trempe who interpreted the original findings states "dark streaks are around the disk and on the temporal side appear to be like small angioid streaks."

The senior author (MGA) has observed this phenomenon in other patients with meningiomas of the orbitocranial junction after having been made aware of the condition by Dr Bullock.[16] Recent studies imply that the tumor arose from either the anterior clinoid process or tuberculum sellae and invaded the optic canal secondarily (Fig 14A and B). The main issue at this time is whether or not the tumor is growing from the left intracranial optic nerve across the midline to involve the right optic nerve and/or the chiasm. Gadolinium-enhanced MR imaging has shown that this has not happened (Fig 15A and B). This is the best method for evaluating progression of the meningioma. Henceforth the patient will be followed with this technique.

Case 4 presented a complex management problem. The presumed meningioma was located in the cavernous sinus, a most difficult location for a surgical approach. This was complicated by the presence of an aneurysm of the left internal carotid artery which was encased by the tumor. Removal of the tumor, which formed part of the wall of the aneurysm, could not easily be accomplished and was considered too risky to attempt. CT scans and MR images have documented growth of the tumor. The more sensitive Gd-DTPA-enhanced MR images has shown marked intracranial involvement (Fig 16A and B). It will be most helpful in following this patient for possible involvement of the chiasm and right optic nerve.

In our experience, adverse reactions to intravenous injections of Gd-DTPA have been rare. Seven cases of nausea and 1 case of hives have

occurred in 444 cases. There has been no evidence for release of free gadolinium from the DTPA chelator reported either in the literature or extensive clinical trials.[6,9,10,13] The usual caveats of not using this substance in severe renal or hepatic disease would seem prudent. Because of possible vaso-occlusive complications patients with sickle cell anemia, hemoglobinopathies, and other hemolytic anemias should not be subjected to Gd-DTPA injections.

Development of new contrast media for MR imaging is undergoing extensive research. The properties for an "ideal" paramagnetic MR contrast agent have been defined.[9] These are:

1. Chemically stable - easily stored for clinical use.
2. Inexpensively synthesized from readily available material.
3. Water soluble.
4. Quickly excreted - primarily by renal route.
5. Highly paramagnetic - able to alter tissue contrast with lose doses.
6. Well-tolerated in diagnostic doses with high margin of safety between diagnostic and toxic doses.
7. Diagnostic and safe.

In the future these agents may be combined with specific tissue delivery vehicles such as liposomes or monoclonal antibodies MR may then assume a diagnostic ability that will enable this technique to identify specific pathological processes.[9,14,15]

CONCLUSIONS

Gd-DTPA-enhanced MR imaging highlights areas of alteration in the BBB.

Gd-DTPA is absorbed in extra-axial tumors such as meningiomas that have no BBB.

Gadolinium-enhanced MR images appear hyperintense in meningiomas on T_1-weighted images.

In the orbit, CT scans of optic nerve meningiomas are superior to enhanced and unenhanced MR imaging (Table III).

Enhanced MR images of meningiomas are superior to CT and unenhanced MR in both the optic canal and intracranial cavity (Table III).

Gadolinium-enhanced MR imaging of meningiomas of the anterior visual pathways is a safe and diagnostic procedure.

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DISCUSSION

Dr Ronald M. Burde. Firstly, I would like to apologize to the members of the AOS for being unable to join this Society for even part of this years 125th Anniversary celebration. Unfortunately, the obligations of assuming the Chairmanship place time constrictions on my ability to travel at certain times. Secondly, I would like to thank the authors, Doctor Alper in particular, for forwarding to me not only the manuscript but the accompanying slides in an appropriate time frame. Finally, I want to thank Doctor WR Green for presenting my discussion.

This paper serves to emphasize the key role that Doctor Alper has played in the continuing education of his neuro-ophthalmologic colleagues and members of this Society, as well as all other members of the ophthalmic community by his pioneering involvement in the field of neuroimaging. From the first coarse images shown at the Society meeting in 1974 through to the fourth generation CT scans and 1.5 Tesla MRI scans we were shown in 1986 and this morning, he has kept us at, what is best termed, the "cutting edge." For this, as a student, a colleague, and friend, I doff my hat and say "thanks, Mel."

In 1985, Doctor Zimmerman et al (*Am J Neuroradiology* 1985; 6:149-157) in

studying 28 patients with 32 meningiomas stated that "this benign treatable tumor was more clearly seen on computed tomography (CT) than MRI in over 50% of cases." "MRI did not demonstrate tumor calcifications but did demonstrate vascular encasement, displacement, and occlusion better than CT. . . ." Similar conclusions were drawn by Mawhinney et al who reviewed 2000 CT scans and identified 45 patients with suspected meningiomas. Thirty-two of these patients had subsequent MRI scans. They concluded that "computed tomography generally defined the meningiomas better than MRI." Only in the nonmeningioma group was the MRI superior to CT. In studying meningiomas, the inversion recovery sequence was superior for demonstrating supratentorial meningiomas but spin echo sequences were better for showing calcification.

A supporting statement is made by Berry et al (*Am J Radiology* 1986; 147:1231-1235) in an introduction to their paper *Gd-DTPA in Clinical MR of the Brain.* Extraaxial lesions offer a different challenge for MR than do intraaxial ones. Their location in the periphery of the brain and, as such, at the edge of an axial section, makes them less conspicuous and subject to partial volume averaging with CSF. More pointedly, in those meningiomas involving the afferent visual pathways, the problem is compounded by several factors: small size, plaque-like configuration, and the relative isointense characteristics of the tumor. The biology of these lesions including their typical vascularity and lack of a blood brain barrier make meningiomas ideal lesions for enhancement with conjugated gadolinium as Doctor Alper has demonstrated. Taking full advantage of the biologic characteristics of these lesions will lead to the development of even more specific markers.

I would request your indulgence for an extra minute to address the topic of what is an appropriate testing sequence in a patient presenting with acute, subacute, or chronic progressive visual loss. In the detailed case descriptions presented by Doctor Alper, most performed before being seen in Washington, patients were subject to the modern equivalent of the Greek elements; air, fire, and water, ie, ultrasound, VER, fluorescein angiography, CT scanning, MRI scanning, and angiography. I would submit the following guidelines:

1. Historical data should differentiate between the sudden onset and the sudden discovery of visual loss.

2. In the vast majority of cases of patients with sudden visual loss neuroimaging, at least initially, is unnecessary.

3. In patients without disc swelling or other signs suggesting anterior optic nerve disease, ultrasonography will be of little value.

4. Once the afferent visual system is compromised except to gain epidemiologic information, a VER has little diagnostic or therapeutic import.

5. The patient with progressive visual loss of some chronicity obviously requires neuroimaging studies. The pattern of the visual loss will determine the appropriate modality to be used.

    a. If the patient presents with unilateral visual loss the first study should be a high resolution CT scan. The question of whether to use contrast or not is not important at this junction. The important points that cannot be over emphasized are: (i) the x-ray request sheet to the radiologist must

inform him of what you are looking for, and, in this case, that "the lesion likely lies between the back of the globe and the chiasm, thus, clearly delineating the area that you want imaged." (ii) the ophthalmologist must personally review the scan with the radiologist in order to assure a careful re-evaluation of the area of concern.

b. If the CT scan is negative, a gadolinium-enhanced MRI scan should be obtained with emphasis placed on the intracranial opening of the optic canal and the intracranial optic nerve to the optic chiasm.

6. The physician should be aware that no modality yet available is capable of demonstrating intracanalicular changes consistently.

7. If the patient presents with visual dysfunction suggesting an intracranial lesion, a gadolinium-enhaned MRI scan should be ordered per primum. As mentioned before, we should inform the radiologist as to what we suspect and review the films with him.

8. Neither MRI or CT scanning is excellent for demonstrating lesions of the cranial base.

The question of when fluorescein angiography is appropriate and helpful in such cases, is enigmatic. Judgment suggests that fluorescein angiography is an abused modality, especially in terms of its diagnostic and therapeutic implication in many cases of visual loss. In the current social and political cataclysmic atmosphere in which medicine seems to be functioning, it is incumbent upon us to select those tests that will specifically lead us to provide both diagnostic and therapeutic help to a patient. We may, indeed, chose to perform other tests, eg, VER, but, in fact, are we justified in that case for so charging a patient?

I would close by once again congratulating Doctor Alper for a job well done.

Dr David Shoch. I have a question for Doctor Alper. Since the gadolinium stays in the vascular compartment, what happens to it? How long does it stay in the body? It is not radioactive and therefore doesn't decay. Is it cleared by the liver or the kidney?

Dr Thomas P. Kearns. Some of the older members; Doctors Blodi, Newell, Cogan, and a few others remember how Doctor Verhoeff would always get up at a meeting and call attention to a paper that he had written years before on the subject under discussion. I realize that I am no Freddy Verhoeff but I could not resist calling attention to one of my early papers on the diagnosis of meningiomas (slide - Kearns T, Wagener H: Ophthalmologic diagnosis of meningiomas of the sphenoid ridge. *Am J Med Sci* 1953; 226:221-228).

Doctor Blodi will notice that this article appeared in the journal that he mentioned in his presentation on the 1864 contributions to ophthalmology. This journal is still going strong and it seems appropriate to call attention to this interesting historical aspect.

More to the point, I am calling attention to this paper to point out just how far we have come in imaging techniques in the diagnosis of anterior visual pathway meningiomas. In this early paper, we found that 25% of these meningiomas were

not detected by routine x-rays of the head. Another way of thinking about this is to note that meningiomas were not at all visible using the ordinary x-ray examinations. Only the hyperostosis produced by these soft tumor masses was visualized hence the diagnosis of only 75% of these tumors.

The last slide that I wish to show is this one (slide - Baker HL, Kearns TP, Campbell JK, et al: Computerized transaxial tomography in neuro-ophthalmology. *Trans Am Ophthalmol Soc* 1974; 72:49-64). The historical aspects of this paper seemed worthy of attention. This was the first paper in the world literature on CT scanning in neuro-ophthalmology and the first paper in the world literature on CT scanning of orbital tumors. I am proud that I was able to have read this paper before the Society. Imaging techniques have come a long way since the time of this paper and Doctor Alper's paper today.

Doctor Alper, your paper is a fine contribution and I am glad that you have read it before the Society.

DR DAVID G. COGAN. A few weeks ago I had the pleasure of attending a conference in which Doctor Alper presented some of his material At that time Doctor Zimmerman showed the specimen of a highly pigmented melanoma of the iris. A gadolinium image had shown the tumor dramatically. My question relates to analogous demonstration of choroidal melanomas. Is there sufficient melanin in these choroidal tumors to differentiate them from amelanotic or hypomelanotic tumors?

DR W. RICHARD GREEN. Mr President, Mr Secretary. We studied two cases of optic nerve meningiomas with shunt vessels in the optic nerve head. The clinical features of one of the cases were reported by Imes, Schatz, Hoyt, et al in the January issue of *Archives of Ophthalmology*. We found that these vessels were venous shunts from the optic nerve head to choroidal veins. This finding suggests that the term optociliary shunts should be changed to optochoroidal venous shunts.

DR MELVIN G. ALPER. Mr President, Doctor Kearns. I wonder if it would be appropriate for my co-author who is here to show a few extra axial lesions? Doctor Sherman.

DR JOHN L. SHERMAN. Thank you. I have a couple of slides. We are just going to skip through these. I wasn't sure what questions were going to be brought up, so I prepared some material. Doctor Alper has already gone through our fact sheets for the use of gadolinium. This answers Doctor Shoch's question. Eighty percent of gadolinium is cleared in 3 hours and about 90% in 24 hours. It is water soluble which is one of the key requirements of a good contrast agent, and makes it clear rapidly. Because it is a relatively small dose it has a very small osmotic role. We have had not problems with renal clearance. There are no contraindications at this point except for theoretic ones based on sickle cell disease, where intravascular clotting could occur. That hasn't been proven yet.

I thought I would show just a couple of examples of some of the other things that gadolinium can do. Here is a case of an acoustic neuroma. T_2-weighted images in a noncontrast scan shows nothing; basically a normal study. There is the basal artery, in a noncontrast T_1-weighted scan. The patient is tilted slightly so we are not seeing the other side here. We see the 7th and 8th cranial nerves. This is fat. Here is the auditory canal. A gadolinium T_1-weighted scan shows the advancing edge of an acoustic neuroma which is most clearly shown by gadolinium. This is one of the key areas where gadolinium has been found useful. One other area here, talking about meningeal disease, is a patient with pain behind the eye. The patient on T_2-weighted noncontrast study showed edema. Otherwise the study was negative. This is significant edema but we don't know what is causing it. The gadolinium showed a mass lesion along the cavernous sinus. This is sarcoidosis with extension of the disease through the intravascular spaces into the brain.

In this patient with carcinomatous meningitis—a noncontrast T_1-weighted image and a noncontrast T_2-weighted image shows nothing. On the contrast T_1-weighted image diffuse intensity of the meninges is shown. This patient was also negative on CT. This is carcinomatous meningitis from breast disease. This is the first technique that we have been able to develop in neuroradiology that is diagnostic in carcinomatous meningitis. One last example of metastasis. The patient with a noncontrast T_2-weighted image showed only one metastatic lesion. A contrast study showed that metastatic lesion as well as a second lesion helping to make the diagnosis of metastatic disease. This is a patient with a T_2-weighted image of a tumor in the posterior fossa. Contrast T_1-weighted image showed the full extent of the tumor differentiating it from cyst.

Dr Melvin G. Alper. In answer to Doctor Shoch—we have answered that. In answer to Doctor Kearns about the senator in Washington, there is another Washington political saying, "read my lips," "gadolinium." Then Doctor Cogan asked the question about MRI of choroidal melanoma. Melanin has paramagnetic properties which if the tumor is loaded with melanin does show up quite well in nongadolinium-enhanced MRI. In the T_1-weighted image the melanoma appears white and the vitreous appears black. In T_2-weighted image the vitreous appears white and the melanoma is black. So melanin being paramagnetic created a white image in the T_1 image against the black vitreous and in the T_2 image (go to the third from the last slide) where the vitreous is white and the melanoma showed up black. Hemorrhage, on the other hand, shows a light signal as you can see there.

So if it is a pure melanotic melanoma it shows up this way and gadolinium would enhance that paramagnetic quality. May we have the lights please. In Doctor Green's astute observation on the choreoretinal shunts it is indeed on the venous side. I want to thank the discussors for their comments and Doctor Burde for sending his remarks as Doctor Green related them to this audience. We agree with all of Doctor Burde's observations. I am only sorry that circumstances prevented him from presenting them in person.

I would like to thank my co-author who has educated me in the middle of the night and on afternoons off.