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Where do hemianopic patients fixate when they look straight ahead?

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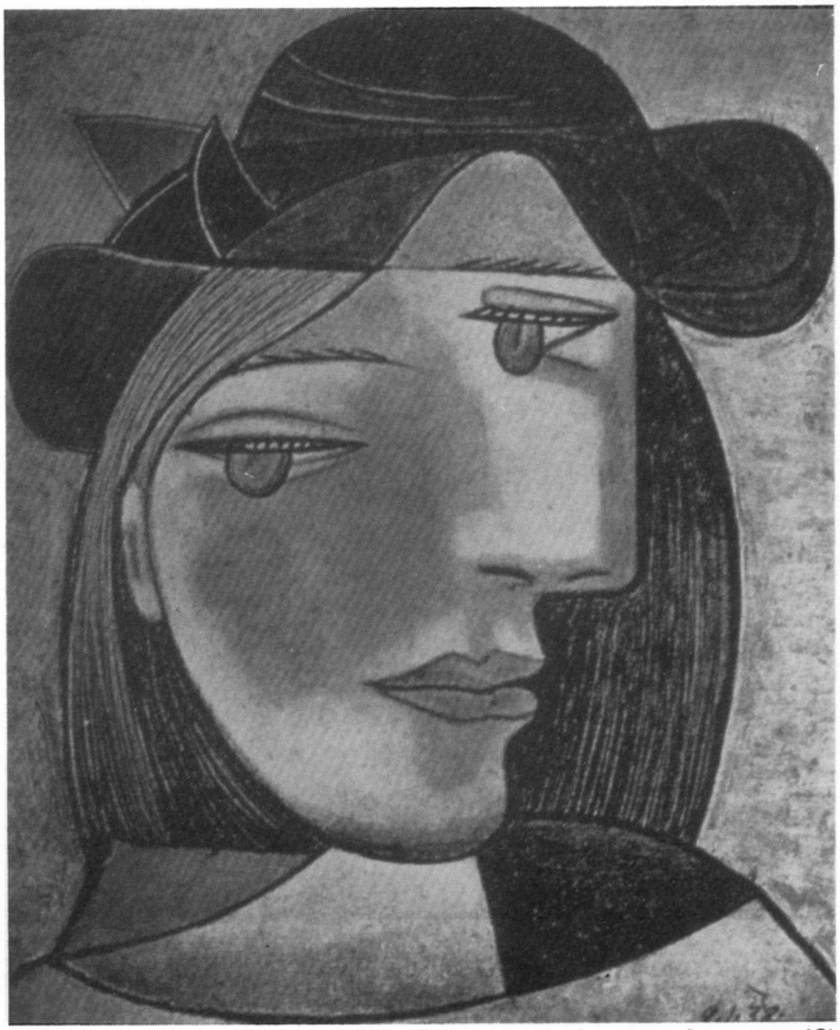

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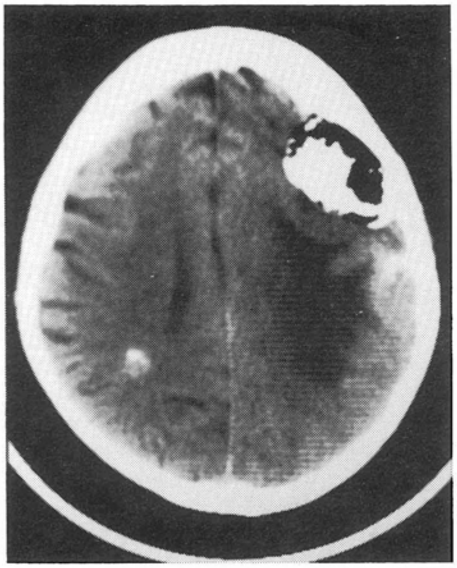

Figure 1a

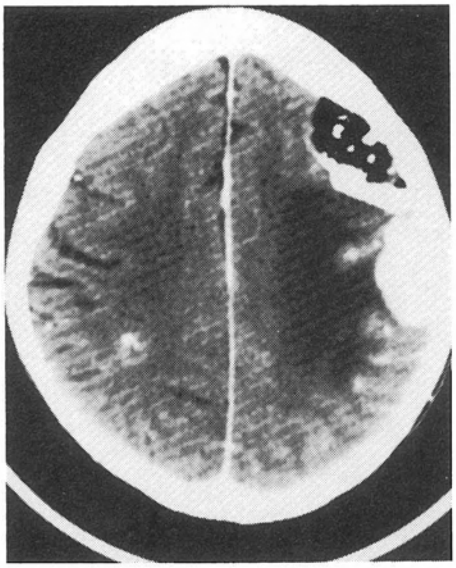

Figure 1b

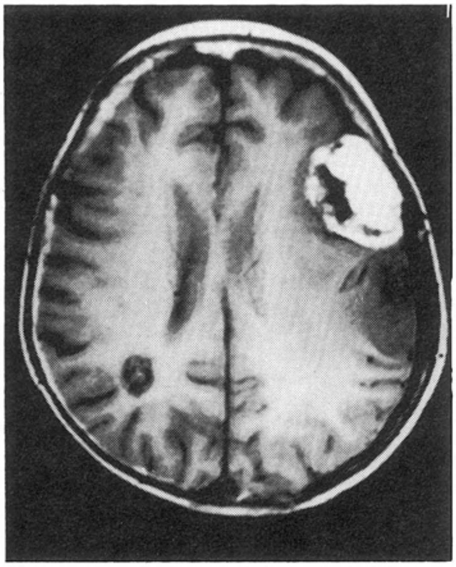

Figure 2

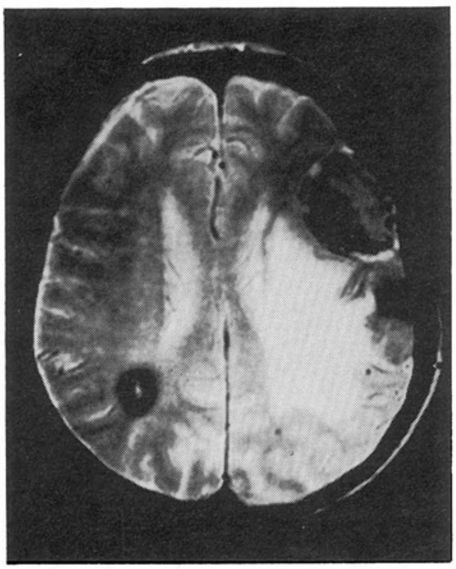

Figure 3

Xanthomatous Meningioma — An Uncommon Variant

D. TAMPIERI, M.D.; D. MELANÇON, M.D.

This 63 year old woman presented two episodes of dysphasia, nausea and vomiting lasting several hours. The neurological examination revealed right lower limb hypertonia, right Babinski and right facial spasm. The patient underwent CT plain and infused, MR with T1 and T2 weighted images and angiography.

The CT plain and infused (Fig 1a, b), showed two well defined space occupying lesions attached to the left fronto-parietal convexity. The lesion located anteriorly, partly calcified, presents a low density consistent with fatty deposits. This finding is confirmed by the MR imaging. In the T1 weighted image (Fig. 2) the fat tissue presents a high intense signal which becomes relatively low intense in the T2 weighted image (Fig. 3). On the other hand the bony metaplasia displays mixed signal intensity. The more posterior lesion, which enhances homogeneously on CT, has an isodense signal in both T1 and T2 weighted images consistent with meningioma. A cavernous angioma, as incidental finding, is visualized in the right parietal white matter. The patient underwent surgery and the pathology report described two meningiomas, syncitial and fibroblastic, with lipomatous and osseous metaplasia.

Fatty deposits can occur as a histologic variant of syncitial and fibroblastic meningiomas. The xanthomatous changes do not represent a degeneration but a storage phenomenon as demonstrated by the character of the lipid (anisotropic sudanophilic lipid) and the good preservation of the cell nuclei.

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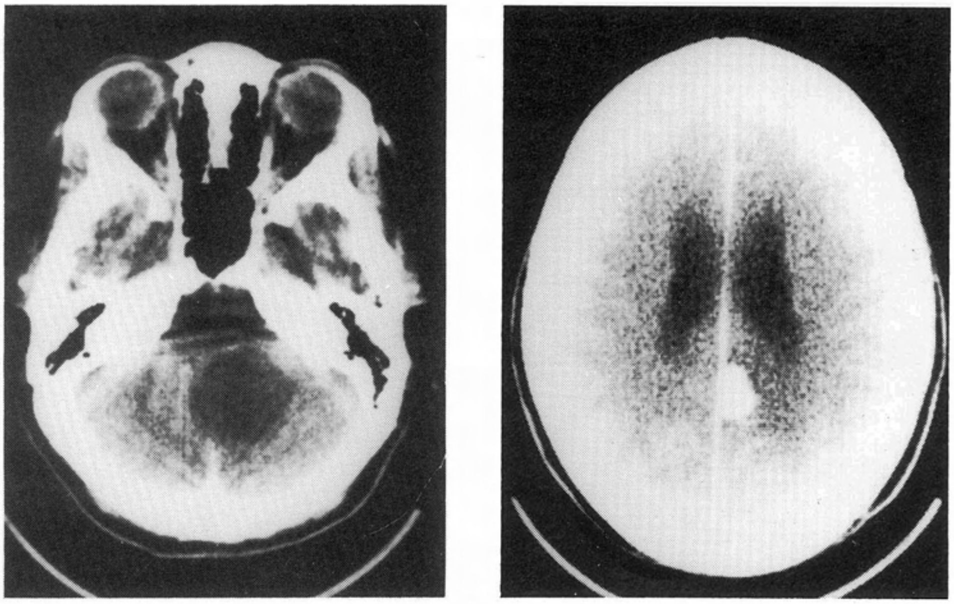
Intracranial and Intraspinal Hemangioblastomas

ROBERTO WEE, M.D., RAQUEL DEL CARPIO-O'DONOVAN, M.D.



▲ **Figure 1** — Contrast enhanced axial CT scan A) Left cerebellar cystic hemangioblastoma B) Left parietal parasagittal solid hemangioblastoma

A woman presented at age 25 with headaches and bilateral papilledema. CT scan after intravenous contrast injection (Fig. 1) demonstrated a small densely-enhanced lesion in the left parietal region. A larger low-density lesion containing an enhanced nodule was seen in the cerebellum. Vertebral angiography (Fig. 2) confirmed the presence of a vascular bean-shaped mass in the parietal region. The mass in the cerebellum was avascular with a mural nodule. The latter was typical of a cystic hemangioblastoma but the supratentorial lesion could not be distinguished from a meningioma. The tumours were removed in 2 separate procedures. They were both hemangioblastomas. At this time, search for other stigmata of the VHL complex was undertaken. There was no retinal angiomatous lesion. Ultrasonography of the abdomen failed to reveal any abnormality in the liver, pancreas, and kidneys. Six years later, this patient presented with progressive numbness of the left hand. Neurological examination localized the abnormality to the cervicothoracic cord. MRI showed a posterior intra-axial nodule at T2, associated with a long cystic cavity extending from C5 to T4. Edema was observed up to the lower medulla and at least to T5 caudally. Spinal arteriography confirmed the presence of a vascular mass fed by a spinal branch arising from the left 3rd intercostal artery. Smaller spinal branches from the deep cervical artery supplied the superior portion of the mass. At surgery, the mass which was located on the dorsal aspect of the cord at T2 was excised. The syringomyelic cavity was opened and clear fluid escaped. The histology of the mass was similar to that of the intracranial tumours removed previously.

▼ **Figure 2** — Vertebral angiogram: Left parietal vascular bean-shaped tumour, mural nodule in otherwise avascular cerebellar mass.

Discussion:

Hemangioblastomas account for 1.5-2.5% of all intracranial tumours and 7-12% of tumours in the posterior fossa[1]. Location in the supratentorial compartment of the skull is rare[2]. The tumours may occur sporadically as a single mass in the cerebellum or as a manifestation of a familial disorder: Von Hippel-Lindau's Disease. In the latter instance, the patient may present with multiple tumours in the neuraxis and neoplasms or cysts in the visceral organs. We report a patient with supratentorial and cerebellar hemangioblastomas who subsequently presented with a third hemangioblastoma involving the high thoracic spinal cord.

Hemangioblastomas account for only 1.6-2.1% of all spinal cord tumours. In a review of 80 cases of histologically demonstrated spinal cord hemangioblastomas, 32.5% occured in patients with VHL[3]. Syringomyelia was present in 66.7% of patients with intramedullary spinal hemangioblastomas.

The occurence of multiple hemangioblastomas in this patient seem to indicate that our patient had Von Hippel-Lindau's Disease inspite of the absence of other stigmata that accompany this disease and lack of familial clinical manifestations[4].

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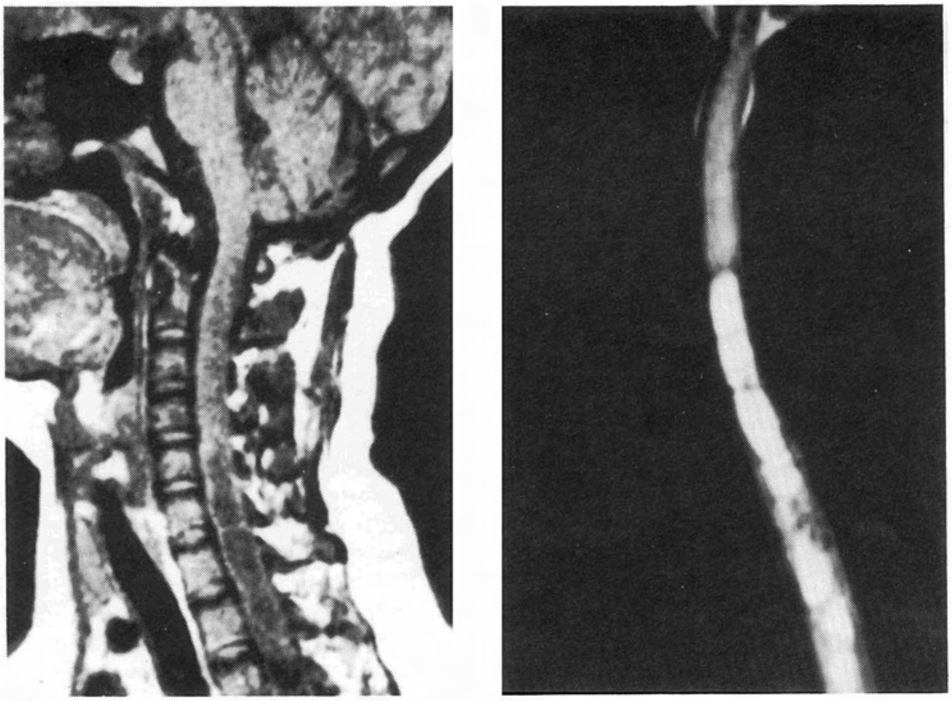

A) B)

Figure 3 — MRI of the Spine A) Sagittal T1-weighted image (SE 30/350). Hypointense nodule, associated C5 to T4 cavity.
B) Sagittal T2-weighted image (SE 180/2100). Hypointense nodule, hyperintense cavity and relatively hyperintense edema extending to the lover medulla.

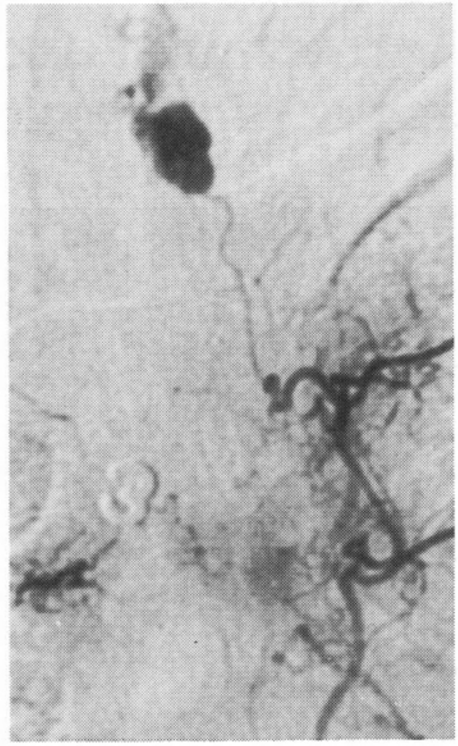

Figure 4 — Spinal angiogram: Vascular nodule fed by left 3rd intercostal spinal branch.

McRae Research Fund Montreal Neurological Institute

Friends and colleagues of Donald L. McRae may wish to show their gratitude for his influence and teaching in the field of Neuroradiology.

The proceeds of this fund will be used to promote education in this field, specifically, to develop new techniques in Interventional Radiology and to support the McRae Lecture in Neuro-Imaging.

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MRI in brainstem metastatic melanoma: A case report

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Melanoma is the third most common tumor to metastasize to the central nervous system, after lung and breast. It may present as a solitary or multiple lesion. Melanomas are asymptomatic in 12% of the cases. Until now, computed tomography offered the best and least invasive method of diagnosing metastasis to central nervous system. Angiography is inconclusive as it only reveals an avascular mass. Due to the inherent paramagnetic qualities or existence of free radicals of melanin, MRI may become an ideal investigative tool for the evaluation of the patient with presumed metastatic melanoma.

A 58 year old man was admitted to the Hartford Hospital with a six week history of slurred speech, difficulty swallowing and ataxia. A mass at the foramen magnum level was suspected. No past history of melanoma elsewhere was available at that time. Excision of a cutaneous melanoma came to light at the time of autopsy.

On autopsy, the brain stem was markedly abnormal with extensive replacement of the medulla by a well demarcated soft grey-yellow focally hemorrhagic and necrotic tumor measuring 2.2 cm in diameter which had destroyed most of the medulla except for the lateral and inferior portions of the olivary nuclei and medullary pyramids. MRI showed a discrete lesion, hypointense on first echo and hyperintense on second echo.

Melanomas are either melanotic or amelanotic and are also frequently hemorrhagic. In recent studies, melanotic melanomas were seen as intense lesions on short TR/TE and appeared isointense or hypointense on long TR/TE. On the other hand, the amelanotic melanoma appeared mildly hypointense or insointense on short TR/TE and mildly hyperintense to isointense on long TR/TE. However, hemorrhage within the tumor makes differentiation difficult. In our patient, the lesion in the medulla was clearly demonstrated from the rest of the brain stem and was hypointense on short TR/TE and mildly hyperintense on long TR/TE. Autopsy confirmed the melanoma as being amelanotic.

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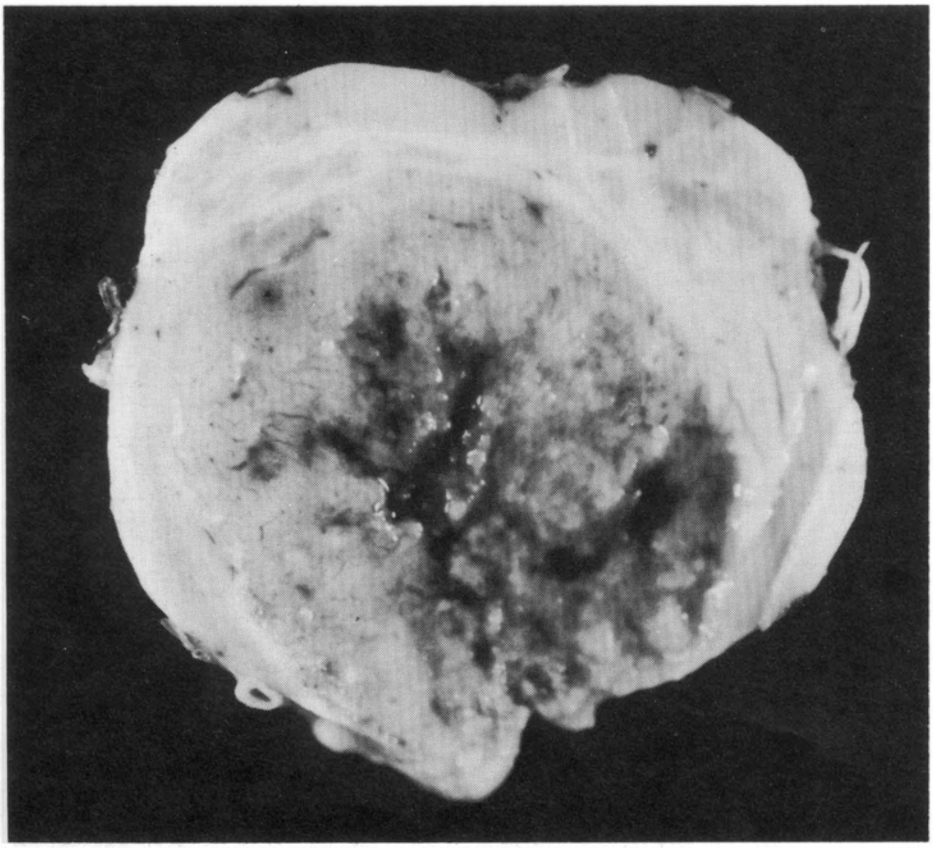
▼ **Figure 1** — MRI in sagittal plane showing discrete hypointense lesion, with a rim of isointense brain in the periphery (arrows) (Short TR/TE).



◄**Figure 2** — Section of the brainstem on autopsy showing the large spherical lesion with rim of normal brainstem (arrows).

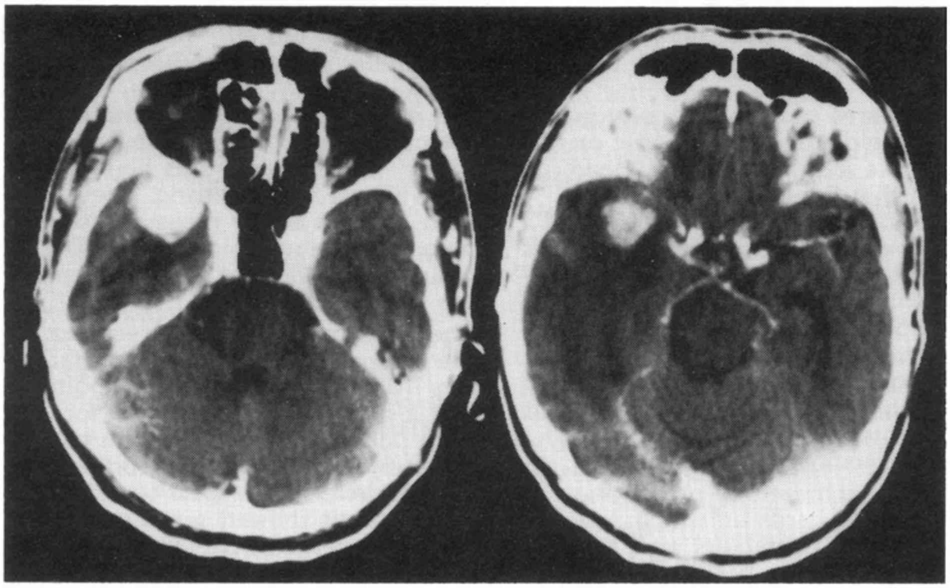


Figure 1

How a small meningioma can cause severe cerebral edema

DENIS MELANÇON, M.D., DONATELLA TAMPIERI, M.D.,

CASE PRESENTATION

The patient, a male of 74, was referred to our CT department to rule out a posterior fossa lesion. He had suffered from a 2nd division right (R) trigeminal neuralgia for 4 years and early CT scan had neglected the original small lesion. The lesion was still relatively small, enhancing homogeneously and located at the middle right (R) sphenoid ridge (Fig. 1), but associated with marked cerebral edema (Fig. 2). An additional lesion, such as a metastasis, was suggested by the clinician to explain the phenomenon. Having had previous experience of such a finding, we retained the diagnosis of meningioma of the sphenoid ridge and surgery was performed. The surgeon (Dr Gilles Bertrand) noted that the lesion was compressing the spheno-parietal sinus (Fig. 3) significantly and that after removal of the tumour better flow was reestablished. Following surgery also, the cerebral edema reduced significantly. (Fig. 4)

DISCUSSION

Cortical venous drainage can be predominant to three[3] main channels: the superior anastomotic vein (Trolard), the inferior anastomotic vein (Labbe) and the spheno-parietal sinus through the middle cerebral vein. Whenever one of these dominant channels is interfered with, one can expect to see more edematous changes. In the present case, the spheno-parietal route seemed to be dominant. (Fig. 3) Such a situation remains however a rare incidence.

In reviewing a recent series at the Montreal Neurological Hospital (200) we could find only 3 cases of small sphenoid ridge meningiomas causing severe cerebral edema. Only in this last case that we present does angiography support the hypothesis of Fine et al[2].

It seems reasonable to think that meningiomas will cause more edema if they are larger[1][3], but small ones will cause as much edema if located on a large venous pathway, and even more so if on a dominant pathway as postulated above.

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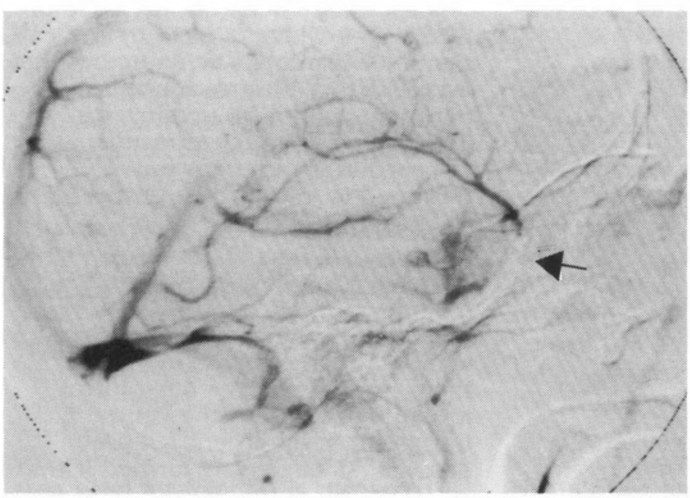
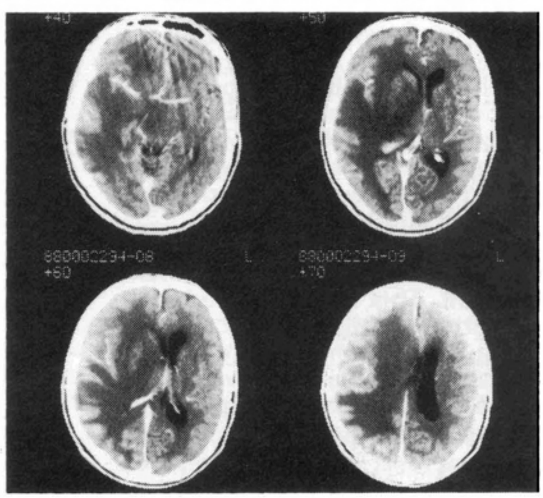


◄ Figure 3

► Figure 2



WHERE DO HEMIANOPIC PATIENTS FIXATE WHEN THEY LOOK STRAIGHT AHEAD?

Homonymous hemianopia refers to the loss of vision in one visual field resulting from the destruction of the optic tract, the geniculo-striate pathway or the striate cortex on one side of the brain. It is a negative symptom, characterized by an absence of sensation, and, as such, it is not "sensed" by the patients who are often unaware of this loss. Two main modifications of the visual system are inherent in homonymous hemianopia with macular splitting. The first, and the more obvious, is a reduction by half of the functional field of vision. The second concerns the splitting of the fovea, and it introduces two potential impairments in visual functions. One is that the size of the functional part of the fovea is halved, which reduces the area of highest acuity. The other is that the fovea, normally the center of fixation, is now directly adjacent to the blind field, which would place the object of attention at the extreme border of the field of vision.

While the first modification is generally well csompensated for by eye and head movements for the scanning of the whole visual scene, at least in patients without hemispatial neglect[7], it remains essentially unknown how hemianopic patients adapt to the second modification of their visual system, but it is generally assumed that they have no specific difficulty in fixating and no loss of visual acuity[6]. Yet early research on visual functions in hemianopia had led to the view that a new center of acuity, or "pseudofovea" was formed, displaced laterally from the anatomical fovea in the non-affected field[3, 4, 5]. Such a new "functional macula"[1, 2] was thought to result from "a redistribution of energy in the intact portion of the visual field about a new functional center of brightest vision"[5]. This suggestion implies that both the area of highest acuity and the center of fixation would be shifted away from the anatomical fovea in hemianopic patients. Although this view has been ruled out[9], and is no longer even alluded to in Textbooks[6], it is unclear how hemianopics cope with this aspect of their impairment. This was examined in two experiments that specifically aimed at determining the area of highest acuity in the intact visual field and the direction of fixation in hemianopic patients.

Area of highest acuity.

The first experiment sought to identify the area of highest acuity in the visual field when hemianopic patients are looking straight ahead. Given that Snellen acuity corresponds to the resolving power of the fovea, by requesting the patients to look straight ahead, and by presenting, on the horizontal meridian, alphanumeric characters of a size that only the fovea can resolve, the location of the identified character with respect to the fixation point would indicate the location of highest acuity in the visual field.

This was achieved by presenting, very briefly, cards on which 9 alphanumeric characters were printed on the horizontal meridian, 4 in the left visual field and 4 in the right visual field, 1° apart; the central character was displaced by .25° from the center toward the intact field, to avoid falling on the vertical meridian (see Fig. 1 for an example). The size of each character was adjusted for each patient and prorated as a function of his/her Snellen acuity and of the viewing distance.

The patient looked at the visual field through the aperture of a tachistoscope, with the head adjusted in a forehead-rest so that the eyes were at the level of the central fixation point. Twenty cards containing 9 characters each were presented one at a time in the tachistoscope, for a duration of 120 msec. so as to avoid eye movement during the presentation. The patient was told that several digits and letters would be briefly presented and that the task was to report the one that could be identified. A trial started by requesting the patient to fixate the bright dot in the center of the visual field. As soon as the patient said he/she was fixating the dot, a stimulus card was flashed and the patient reported the digit or letter that could be identified.

TABLE 1: CLINICAL DATA OF PATIENTS.

	Age	Sex	Time from onset of symptoms	Type of lesion	Side of lesion	Neglect
1.	28	F	5 years	Resection of glioma in striate cortex	Right	None
2.	73	M	3 months	Parieto-occipital hemorrhage	Left	Mild
3.	69	M	1 month	Large angioma in parieto-occipital area	Left	Mild
4.	49	F	6 months	Resection of glioma in temporo-parietal area	Right	Mild
5.	34	M	4 years	Resection of glioma in occipito-parietal area	Right	None
6.	57	F	6 months	Resection of glioma in temporo-parietal area	Right	Strong

The subjects were 6 unilaterally brain-damaged patients presenting with dense homonymous hemianopia with macular splitting. They had their Snellen acuity measured on 3 occasions before being tested. The main clinical data of these patients are presented in Table 1.

The main measure of interest was the distance of the identified characters from the central fixation point, and it is presented in Table 2 for each patient. By contrast with control subjects who always reported the character the closest to fixation, each patient showed systematic deviation away from the center, and always identified a digit or a letter that fell in the retinal periphery *within the anopic field*. There was little variation in the location of the identified characters within each patient's response, as indicated by the standard deviation of the mean distance which was in all cases inferior to 1°. All the patients reported that they were fixating accurately the central dot, and they were therefore unaware of the deviation of their eyes.

TABLE 2: MEAN DISTANCE (IN DEGREE OF VISUAL ANGLE) OF IDENTIFIED CHARACTERS FROM THE CENTRAL FIXATION POINT.

Patients	1	2	3	4	5	6	Mean	Controls
Distance	0.9	2.6	2.2	3.5	3.6	2.3	2.5	0

These results do not conform to what would be expected knowing the visual deficit of the patients. An identification of the central character, or of a character located in the intact visual field, would have been more consistent with the presence of hemianopa. Instead, eccentric fixation toward the blind field was systematic and points to a difficulty in directing gaze as intended. The results also suggest that the area of highest acuity does not correspond to the area which is used by the patients for fixation. The source of the eye deviation observed in this experiment cannot be unequivocally determined from these results, and the second experiment was designed to clarify this issue.

"Pseudofovea" or anatomical fovea?

Because hemianopics have difficulty fixating as intended, they were provided, in the second experiment, with landmarks that could guide them in directing the anatomical foveal area right on the central fixation point. If an eccentric "pseudofovea" is effectively formed within

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Temporal lobe seizures for 30 years: excision of amygdaloid sub-ependymoma detected by MRI.*

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Magnetic resonance imaging has resulted in a high detection rate and a detailed anatomical display of lesions associated with seizures originating from the mesial region of the temporal lobe (3, 5, 6, 7). The following case provides an example of the demonstration by MRI of a focal lesion in the amygdaloid region and illustrates also the surgical value of adding the sagittal view (1, 3) to the more routinely used axial and coronal planes. The findings further confirm the implication of the amygdaloid complex in the genesis of temporal lobe seizures (1, 2, 4) and the importance of excision of this structure in surgical treatment.

Case report

Patient RF was treated surgically at age 36 because of intractable seizures. At the age of three and a half years he had two grand mal attacks associated with a high fever but with a normal spinal fluid examination. From age 7 he had persistent seizures with a warning of a nauseous feeling in his stomach, sometimes an unpleasant smell, followed by an awareness of stiffening of his body. He might recover from these after 15 or 20 seconds or go on to a generalized attack lasting about 2 minutes. The EEG technician observed an attack; at the beginning he was unresponsive and staring at his hands before he had stiffening of his body. He was treated by medi-

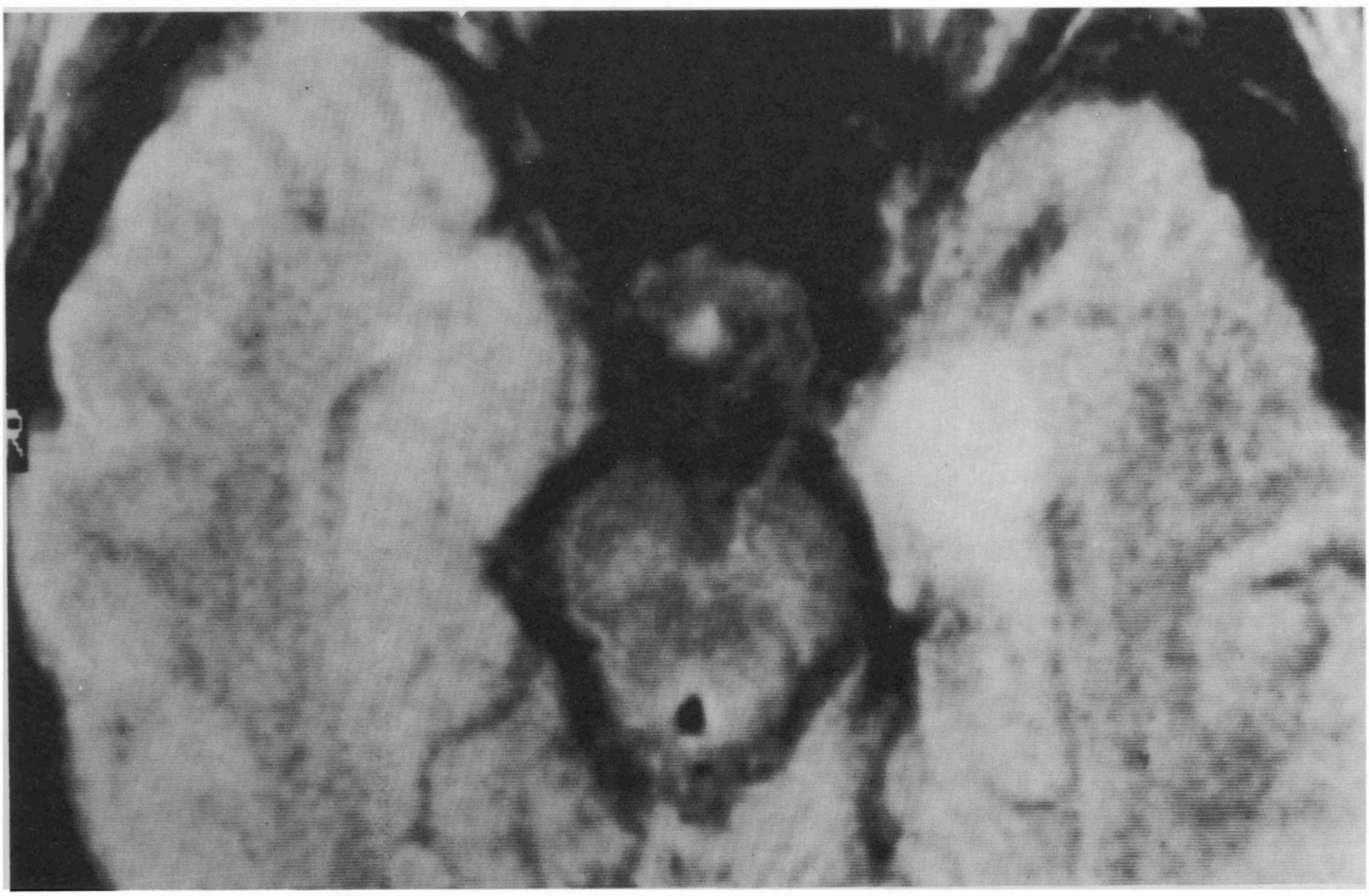

Figure 1 — Axial view showing the focal increase of signal intensity in the periamygdaloid region.

Figure 2 — The same on coronal view. Note again the atrophic gyri and smallness of the temporal lobe.

cation at first successfully, but then with inadequate control. In the 10 years before admission, he displayed at times difficult behavior with paranoid features and hostility toward members of his family. A year before admission, EEG showed a well-defined spike discharge from the anterior temporal region associated with mild focal slowing indicative of a structural lesion. Further studies showed bursts of sharp waves and slow waves with phase reversal at the sphenoidal electrode on the left side. PET with 18F-deoxyglucose showed a 15% lower glucose activity in the left temporal region and 37% lower activity in the left mesial temporal area as compared to the right (Dr. Lucas Yamamoto).

CT scan showed the sulci modestly larger in the left hemisphere than the right; no lesion was seen with infusion of contrast medium. On MRI, signal intensity was increased in a focal area corresponding to the amygdaloid nucleus and the anterior hippocampus (Fig. 1). The lesion extended superiorly into the claustrum and medially toward the lower margin of the pallidum (Fig. 2). The left temporal lobe was small with increased subarachnoid spaces and narrowing of the convolutions, especially the first temporal. The signal intensity increase on coronal and sagittal sections was bounded by the stem of the middle cerebral artery in the vallecula and the temporal tip of the ventricle (Figs. 3, 4 and 5).

At operation, active epileptic spiking was recorded from the anterior temporal cortex and from a depth electrode in the amygdala. The first temporal gyrus was narrow and increased in texture as it ran around into the uncus; microscopically, it showed severe subpial gliosis with focal marked neuronal loss mainly in the second cortical layer, extensive gliosis in the white matter and deposition of *corpora amylacea*.

The ventricular surface of the hippocampus was rough and dull compared to its usual smooth shiny coating; its anterior portion was tough and shrunken. The amygdala was firm and replaced in its posterior medial portion by pinkish tissue. On microscopic examination, the abnormal tissue showed spindle-shaped cells forming perivascular pseudo-rosettes, extension of the cells into the perivascular spaces and focal reactive gliosis (Fig. 6). The diagnosis of subependymoma was supported by the immunoperoxidase labelled antibody study that revealed astrocytic differentiation associated with few Flexner rosettes and typical tapering processes around vessels.

The final removal included the amygdala, the abnormal pink amorphous tissue, the anterior 1 cm of the pes and body of the hippocampus and adjacent hippocampal gyrus. Corticographic abnormalities from the lateral cortex were dealt with by resection of lateral cortex back to 4.5 cm from the temporal pole.

In the 3 years since operation, the patient reported no attacks, while on 200mg of dilantin and 150mg of phenobarbital each day. His father observed several episodes lasting up to 30 seconds when the patient was asleep, with stiffening of the limbs and watering of the eyes. None has occurred while he was awake.

Comment

Findings from MRI have confirmed the association of temporal lobe seizures with the involvement of the mesial temporal region (5) and particularly the amygdala (1,3) by small structural lesions. In addition,

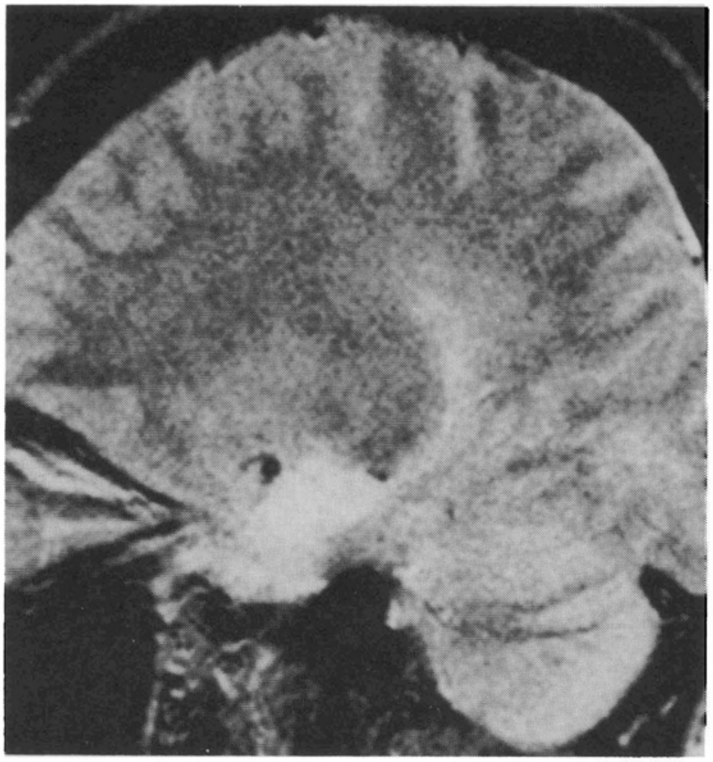

Figure 3 — Sagittal view showing the lesion between the middle cerebral artery and the temporal horn.

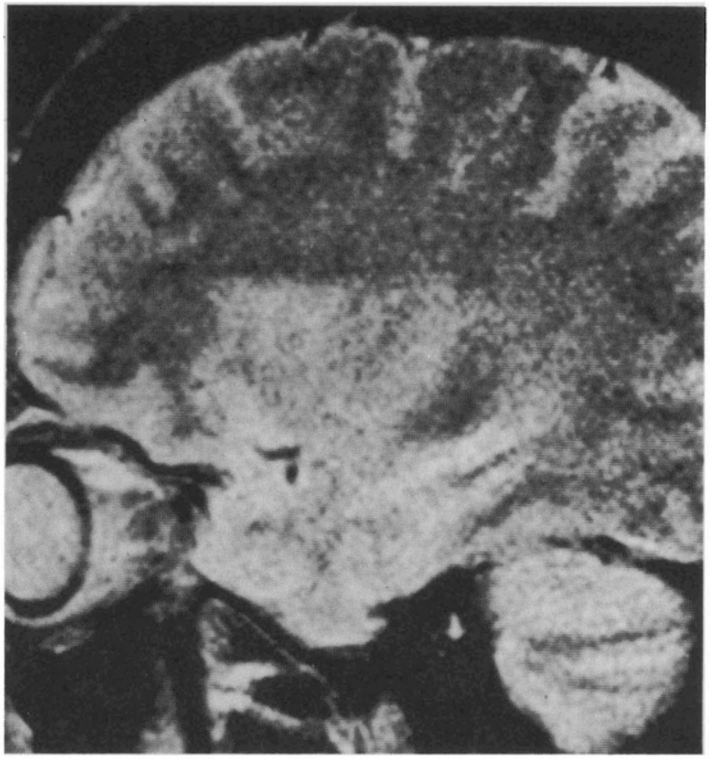

Figure 4 — Normal side for comparison.

gliosis and neuronal loss has been correlated with signal increase in the mesial temporal region on MRI in up to 70% of patients with well defined mesial temporal EEG localization. Brief tonic motor posturing noted earlier as a feature of temporal lobe seizures with automatism, has been produced by stimulation of the claustroamygdaloid complex in patients undergoing surgical treatment (1, 2).

Since the advent of MRI the incidence of small structural lesions in the anterior and mesial temporal region detected before operation has increased from 20% to over 25% in the MNI series.

In addition, MRI defines more exactly atrophic changes in the temporal lobe, including smallness of the lobe, shrunken gyri, especially the first temporal convolution, and the commonly (up to 70% of cases without small structure lesions) associated mesial temporal increase of signal intensity that correlates with gliosis and neuronal loss histologically, particularly in the amygdala and anterior hippocampus.

Finally, MRI provides precise definition of the extent of the surgical excision in this complex anatomical region to correlate with diagnostic, physiological and psychological findings. We recommend for this, triple planar imaging using an axial plane at a 20° angle below the horizontal cantho-meatal line (to give longitudinal sections of the amygdalo-hippocampal complex) plus sagittal sections that reveal the details of resection of the amygdala and anterior hippocampus.

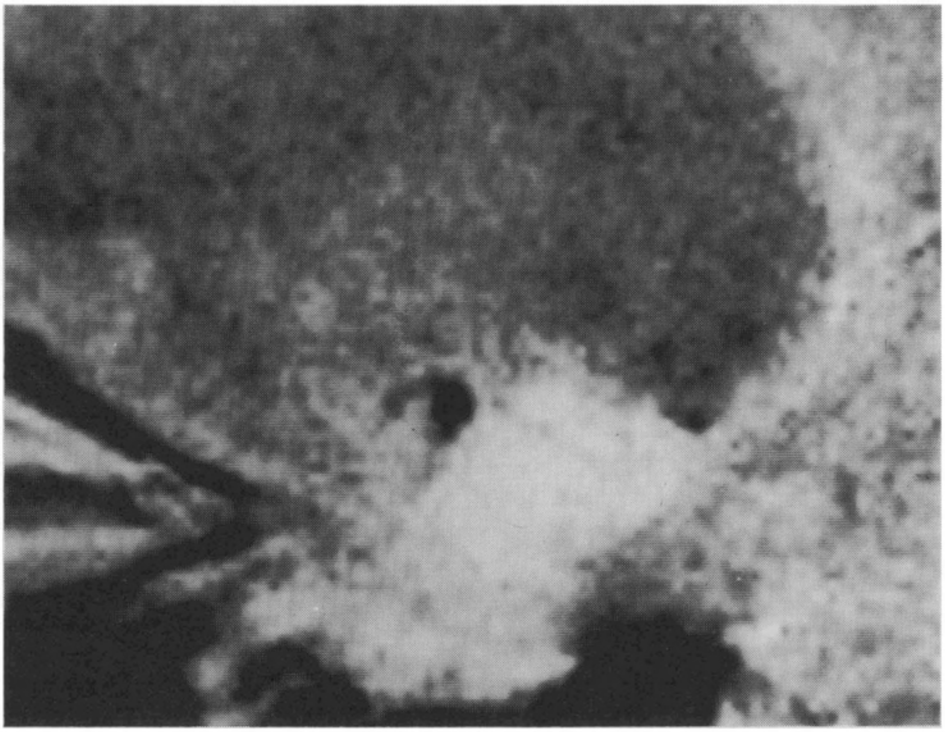

Figure 5 — Enlargement of the sagittal view with the increased signal intensity.

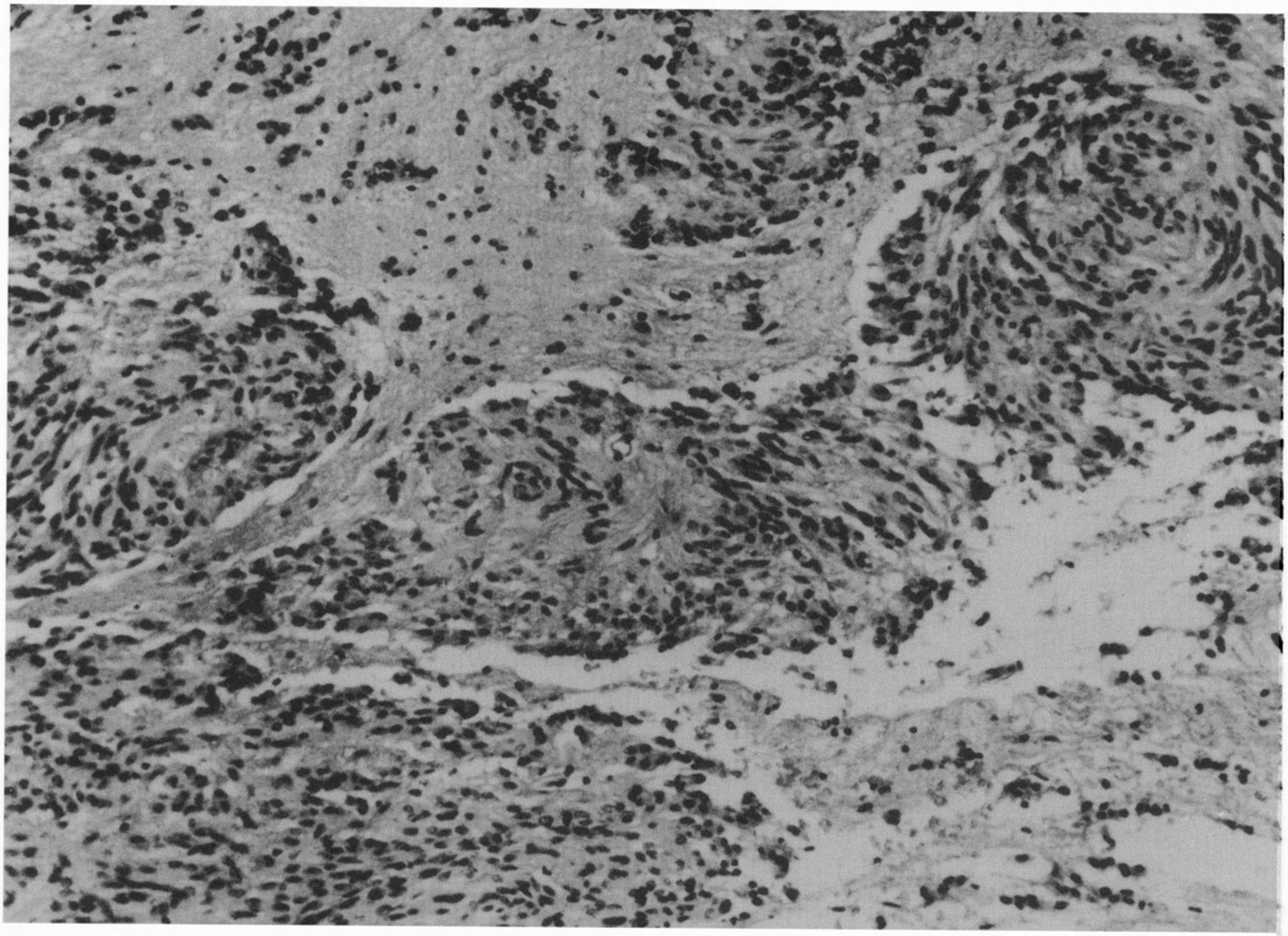

Figure 6 — Histology showing islands of tumor with spindle shaped cells in a streaming pattern and reactive gliosis on the margin.

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the intact field[1, 2, 5], the patients should identify characters falling on this new center of acuity. By contrast, if the anatomical fovea is the area of highest acuity, the patients should report the character closest to fixation.

The same patients participated in this experiment, and the same material was used, with one exception. The visual field contained landmarks that guided the patients in fixating as intended. These consisted in a division of the visual field into two halves of different colors, juxtaposed along the vertical meridian, without any line in between, so that they split the central fixation point. In addition, 3° below and above the fixation point, a square (side of .5°) was located within the intact field of the patient, so that the inner side of each square fell right on the vertical meridian. The patients were requested to fixate the central dot in such a manner that they see only one colour as well as the two squares. With this arrangement, the inner border of the patient's field of vision fell just on the vertical meridian, splitting the central fixation point.

All other aspects of the procedure were the same as in the first experiment, and each patient's Snellen acuity was again measured prior to the task to control for any change in visual acuity.

The patients reported no difficulty in following the instructions and adjusting their fixation as requested. The results were quite straightforward, as each patient now identified the character closest to fixation on all trials. By contrast with the results of the first experiment, there was no report of characters appearing in the anopic field, and the present results clearly indicate that the area of highest acuity corresponds to the anatomical fovea.

Discussion

This study was designed to examine the direction of fixation and the area of highest acuity in the functional visual field of hemianopic patients. The results suggest that hemianopics no longer use the anatomical fovea to focus on a target but they deviate their eyes toward the blind field so that the retinal area used for fixation is displaced from the anatomical fovea in the intact field. This finding may be consistent with the view that a "pseudofovea" is formed in hemianopics, shifted from the normal center to the periphery[1, 3, 5]. The results of the first experiment, in which the patients identified characters located in the affected field, conform to this view whereby the "pseudofovea" would have been used to fixate on the central dot and the anatomical fovea would have been directed toward the anopic field. However, contrary to this view, the results of Experiment 2 indicated that such a "pseudofovea" is not a new center of acuity, and the area of highest acuity remains the anatomical fovea. When hemianopic patients were provided with landmarks to guide their fixation, their performance was similar to that of control subjects.

Hemianopic patients are typically unaware of their deficit which is a negative symptom and to which they naturally adapt by resorting to mechanisms which they had developed prior to their illness. Thus, the reduction of the size of the visual field is readily compensated by eye and head movements, and when they fixate on a specific target, they continue to place the object of attention within its surrounding context rather than using the anatomical fovea which is now directly adjacent to the blind field. Eccentric fixation toward the anopic field in hemianopics may thus not reflect the formation of a "pseudofovea" but may represent an adaptive response to the restricted field of vision so that the object to which they attend can be perceived within its surroundings, as it was prior to their illness.

Several implications for the study of visual perception may follow from this eccentric fixation, as hemianopic patients may not be fixating as assumed by the examiner. For example, it was recently observed[8] that the phenomenon of perceptual completion is hemianopics was partly attribuable to a deviation of the eyes toward the blind field, although this factor was not sufficient to explain the whole phenomenon. The present finding thus leads to the view that visual fixation in hemianopic patients must be a complex affair, resulting from a compromise between achieving a sufficient level of resolution and obtaining sufficient information about the environment in which the object of attention is located. Such a compromise must involve interactions between the subject and the particular visual scene, which would indicate that there is no fixed area which has become the new center of fixation and that different areas may be used for fixation depending on the characteristics of the object of attention.

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Figure 1 — Example of the distribution of the stimuli on a stimulus card used in Experiments 1 and 2.

5 D 4 S 2• R 8 H M

Note: The central character was displaced from the exact center to the left for right hemianopics (as shown here) and to the right for left hemianopics.