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Impaired visual motion perception in the contralateral hemifield following unilateral posterior cerebral lesions in humans

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SUMMARY

Contrast thresholds for a number of tasks were measured in the contralateral and ipsilateral upper quadrants of the visual field (eccentricity = 10°) before and after an occipito-parietal surgical resection, in one patient, carried out for intractable epilepsy. Postoperatively the contrast thresholds for discriminating the speed of movement of drifting sine-wave gratings were elevated by greater than a log unit in the contralateral field with little or no change in the detection thresholds for the same stimuli. Contrast thresholds for opposite direction-of-motion (DOM) discrimination of a contrast modulated (CMod) grating (a 'non-Fourier' motion stimulus) were also elevated by about a log unit in the contralateral hemifield but thresholds for DOM discrimination of a sine-wave (luminance modulated, LMod) grating were unaffected. Contrast thresholds for orientation discrimination of stationary gratings (a non-motion task) were unaffected. This general pattern of results was found in two other patients following lateral occipital surgical resections. Eight other patients with occipito-temporal (two cases), parietal (three cases) and medial occipital lobe lesions (three cases) showed no difference between the two hemifields on any of the tasks. Comparison of the location of the lesions leads to the conclusion that damage to the lateral occipital gyri is responsible for the pattern of visual deficit observed. Damage to an extra-striate visual area concerned with motion perception (the human homologue of primate V5-MT) may have occurred. There has been no previous description of impairment of motion perception localized to a hemifield in humans. The characteristics of the residual motion perception in these cases is described further in the accompanying article [Plant and Nakayama (1993), *Brain*, **116**, 1337–1353].

INTRODUCTION

Evidence from primate anatomy and physiology suggests that projections from V1 (striate cortex) and from extra-striate visual area V2 to a cortical area designated the middle

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temporal area (MT) (Allman and Kaas, 1971) or V5 (Zeki, 1974) (referred to as V5-MT in this article) subserve a special role in the processing of visual motion. Also involved are the adjacent areas known as the medial superior temporal area (MST) (Allman and Kaas, 1974*a*; Desimone and Ungerleider, 1986) and the visual areas of the parietal lobe to which V5-MT and MST project. The physiological properties of neurons encountered in these areas are compatible with V5-MT having an important role in visual motion processing: neurons in V5-MT encode direction of motion (Dubner and Zeki, 1971; Zeki, 1974), and probably speed and velocity (e.g. Maunsell and Van Essen, 1983; Rodman and Albright, 1987). Maunsell and Newsome (1987) and Zeki (1990*a*) have provided recent reviews of this evidence. The location of an area in human cortex which may be the analogue of primate V5-MT was first identified on the basis of myeloarchitectonics (Clarke and Miklossy, 1990) and evidence of a similar functional specialization in human extra-striate cortex has been obtained in human PET studies (Corbetta *et al.*, 1990; Zeki *et al.*, 1991).

Examples of damage to the posterior cerebral hemispheres in humans giving rise to selective impairment of motion perception are rare (*see* Zeki, 1991, for a recent review). The most extensively studied case was first reported by Zihl *et al.* (1983) and has been the subject of further investigation (Hess *et al.*, 1989; McLeod *et al.*, 1989; Baker *et al.*, 1991; Zihl *et al.*, 1991). Vaina *et al.* (1990) have reported a case of bilateral parieto-occipital damage which resulted in impaired spatial localization and stereopsis who also showed impaired performance on a speed discrimination task. This case did not report the marked symptomatic perceptual difficulties experienced by the case described by Zihl *et al.* (1983). Both of these cases had sustained bilateral lesions and the anomaly was present throughout the visual field.

The purpose of our study was to better specify disorders of motion perception in humans. Specifically we have exploited the observation that, in primates, V5-MT is a relatively small visual area which contains a complete representation of the contralateral hemifield (Allman and Kaas, 1971; Gattass and Gross, 1981; Van Essen *et al.*, 1981; Desimone and Ungerleider, 1986; Fiorani *et al.*, 1989). Other extra-striate areas differ in this respect: in the case of the dorsal and ventral portions of V2 and V3, representations are found of the two contralateral quadrants of the visual field which are largely separate in cortex (Allman and Kaas, 1974*b*; Gattass *et al.*, 1981) or in the case of V4 the contralateral visual field is represented in a much larger strip of pre-striate visual cortex (Gattass *et al.*, 1988; *see* Zeki, 1990*b*, for a recent review). It therefore seemed plausible that impaired motion perception in a hemifield may occur in humans following discrete damage to a homologue of V5-MT or its afferent or efferent projections, provided that the pathway is interrupted at or before the extensive transfer of information to the opposite hemisphere through callosal connections. Such a lesion could occur more commonly than bilateral lesions responsible for impairment of motion perception and the ability to use the ipsilateral field as a control is a considerable experimental advantage.

We describe three patients with impaired speed discrimination restricted to a hemifield. The lesions responsible were surgical resections for (i) a medically refractory seizure disorder; (ii) a tumour; (iii) an occipital haematoma. In two we therefore had the unusual opportunity (in human studies) to examine them before and after the resections. Apart from the fact that the anomaly is localized to a hemifield there are other important differences from the perceptual deficit experienced by the case described by Zihl *et al.*

(1983) (*see* the accompanying article, Plant and Nakayama, 1993). In our investigation we have measured contrast thresholds for five basic visual parameters. These tasks have been selected to study the effects of the cerebral damage on basic and higher visual functions subserving motion perception. (i) Contrast detection in order to determine the integrity of mechanisms subserving the detection of the stimuli used. (ii) Direction-of-motion (DOM) discrimination of a luminance modulated (LMod) sine-wave grating in order to determine the integrity of mechanisms subserving the basic motion task of direction discrimination of a 'first order' stimulus, i.e. one in which information about motion can be derived from the contrast of the signal. (iii) Direction-of-motion discrimination of a contrast modulated (CMod) grating. This stimulus is 'second order', i.e. information about the motion of the stimulus cannot be obtained without a non-linear transformation. Unlike the bright and dark bars of the LMod grating, in this stimulus the motion is of regions of high and low contrast which have the same average luminance. Detection of motion in this stimulus may require additional processing in the brain in comparison with stimulus (ii). (iv) Speed discrimination (which is also likely to require additional processing in the brain). (v) Orientation discrimination (a non-motion task which employs the same stimulus as the other tasks).

Some of the findings have been published in abstract form (Plant *et al.*, 1990; Plant and Nakayama, 1991).

PATIENTS AND METHODS

Eleven cases of unilateral posterior hemisphere damage were studied. The clinical details of the patients and the results of perimetry are summarized in Fig. 1. Case 1 was the first patient to be studied and the abnormalities found determined the experimental protocol used subsequently. Informed consent was obtained from all the patients in accordance with the Declaration of Helsinki.

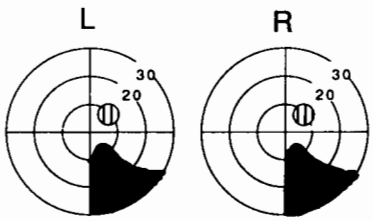
Cases 1 and 2 were tested before and at various time intervals following surgery. The other patients, who had established lesions when first seen, were tested at least 3 months following the acute event and in two sessions of 5−6 h each, separated by an interval of 2−3 days. The stimulus was masked to provide a circular aperture subtending an angle of 12° at a viewing distance of 114 cm. The aperture was centred at an eccentricity of either 10° or 30° of visual angle on the 45° oblique meridians in the quadrant of the visual field to be tested. Viewing was binocular. A chin and headrest were used to minimize head movements and the display itself was positioned directly in front of the subject at eye level. A fixation mark was positioned on a square white screen surrounding the display which measured 100×100 cm and the subject was instructed to fixate it by an eye movement and not by a head movement. This surround was illuminated to give approximately the same mean luminance and hue as the oscilloscope screen; no light fell directly on the oscilloscope display. During data collection the experimenter was positioned behind the screen so that he could observe the subject and reject responses to stimuli which may have coincided with an eye movement.

Visual stimuli

The visual stimuli employed in this study were sine-wave gratings generated digitally and displayed on an oscilloscope with a white phosphor (P4; Joyce screen) having a mean luminance of 200 cd/m^2 and a frame rate of 100 Hz (1024 lines displayed on each frame). The contrast response of the oscilloscope was linear up to 98% contrast. In all the experiments employing a sine-wave stimulus the spatial frequency of the grating was 0.5 cycles/degree and the orientation vertical (except where this was varied to set thresholds for orientation discrimination). One digital-to-analogue convertor (DAC) generated the sine wave, a second DAC controlled the contrast across the screen by a vertical one-dimensional raised cosine trapezoidal spatial window. In the case of the CMod gratings there was no spatial window and the first DAC was used to generate the carrier signal and the second to generate the modulating signal. The overall contrast of the pattern was controlled by a third DAC and the pattern was ramped on and off by controlling the contrast of the pattern by a raised

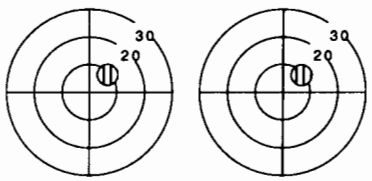
LATERAL OCCIPITAL LOBE LESIONS

CASE 1



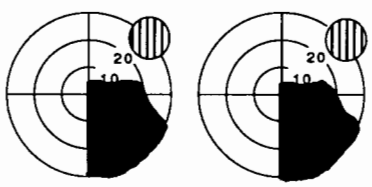
EXTENSIVE LEFT OCCIPITO-PARIETAL SURGICAL RESECTION FOR INTRACTABLE FOCAL EPILEPSY

CASE 2



LEFT LATERAL OCCIPITAL RESECTION FOR TUMOUR

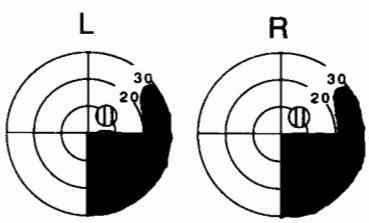
CASE 3



LEFT PARTIAL OCCIPITAL LOBECTOMY FOR HAEMORRHAGE

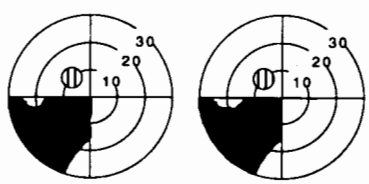
MEDIAL OCCIPITAL LOBE LESIONS

CASE 4



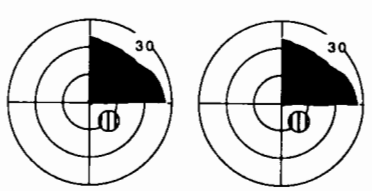
LEFT MEDIAL OCCIPITAL INFARCTION

CASE 5



RIGHT MEDIAL OCCIPITAL INFARCTION

CASE 6



LEFT VENTRO MEDIAL SURGICAL RESECTION OF TUBERCULOMA

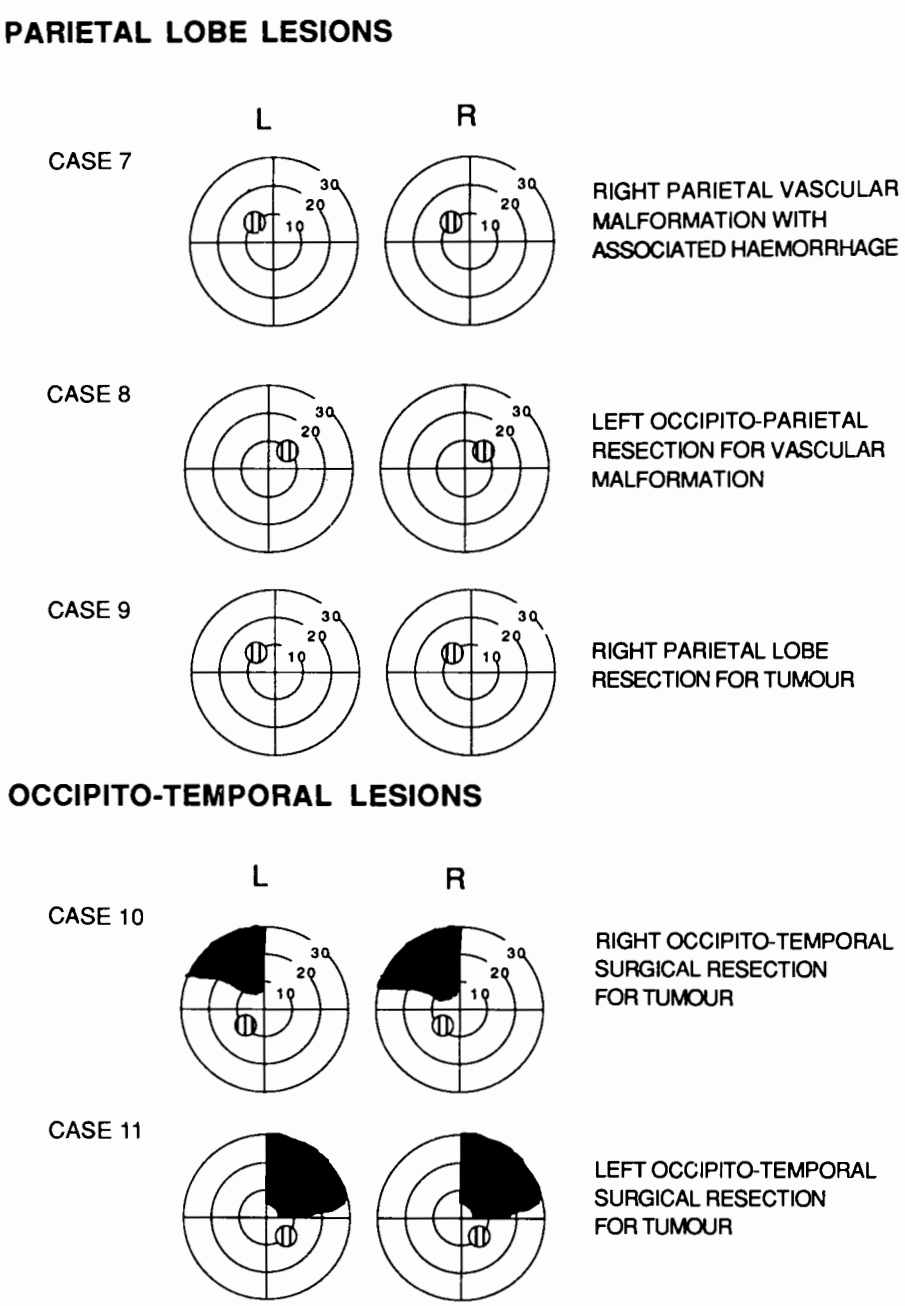


FIG. 1. The visual field defects and diagnoses are shown for the 11 patients who participated in this study. The field defect indicates an absolute field defect either to I4e on the Goldmann perimeter or to a target with an incremental contrast of 99% on the Humphrey automated perimeter. For each case the location of the test stimulus in the contralateral visual field is shown, the results obtained at that location have been compared with those obtained at the mirror symmetric location in the ipsilateral hemifield.

cosine trapezoidal function of time. These waveforms are shown schematically in Fig. 2. Stationary gratings were in spatial cosine phase; where temporal modulation was used, the spatial phase of the grating was changed at the start of each frame. In the case of the CMod grating the contrast of a 0.5 cycles/degree grating (the carrier, six cycles per 12° stimulus field in cosine phase) was modulated in space across the screen by a 0.083 cycles/degree cosine (the modulating signal, one cycle per stimulus field). This is trigonometrically equivalent to summing two cosine gratings of 0.417 cycles/degree and 0.583 cycles/degree each of half the contrast of the CMod grating and gives rise to an apparent 'beat' frequency of 0.17 cycles/degree (two cycles per stimulus field; *see* Fig. 2).

In preliminary experiments it was found that DOM discrimination was possible in control subjects at the eccentricity used when the modulating grating was drifted at a temporal frequency of 0.3 Hz. A very low spatial frequency of the modulating signal was necessary, otherwise DOM discrimination was not possible at this eccentricity. In this stimulus the carrier grating is temporally modulated in counterphase when the modulating grating is drifted and hence gives an equal rightward and leftward DOM signal. Therefore the subject must judge DOM from the direction of drift of the modulating waveform or 'beat' frequency. The contrast of the complex waveform was controlled in the same way as the sine-wave gratings by means of the third DAC and thus the contrast of the carrier and of the modulating grating were always equal.

Specific test procedures

Contrast detection thresholds (LMod and CMod gratings). A temporal two-alternative forced-choice staircase procedure was employed to measure contrast thresholds for stimulus detection. The stimulus was randomly assigned by the computer to one of two temporal intervals, the other interval being blank, and it was the subject's task to identify the interval in which the grating appeared. The subject's responses were in the form of button presses which were monitored by the computer. In these experiments a correct response resulted

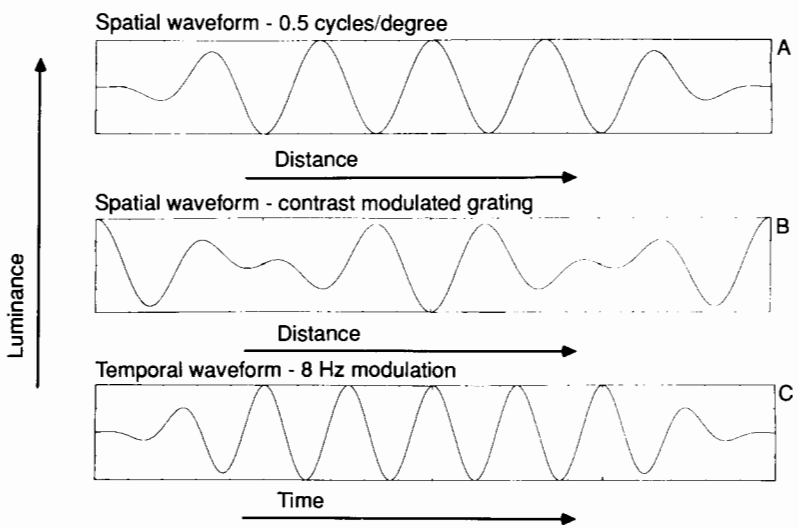


FIG. 2. The visual stimuli employed in the study are illustrated. The sine-wave luminance modulated (LMod) grating was a 0.5 cycles/degree vertical grating modulated in space by a vertical raised cosine trapezoidal window (A). The stimulus was presented in a circular aperture centred eccentrically in the visual field, usually at 10° from fixation. The contrast modulated (CMod) grating or 'beat' stimulus was a 0.5 cycles/degree cosine grating modulated in space by a 0.08 cycles/degree cosine-wave giving rise to an apparent 'beat' frequency of 0.17 cycles/degree (2 cycles per stimulus field) (B). When the modulating grating is drifted horizontally the 0.5 cycles/degree grating is modulated in counterphase and so information as to the direction of drift is available only from the motion of the envelope of the waveform. The stimulus presentation time was usually 800 ms. The stimulus was ramped on and off slowly by a raised cosine temporal modulation (C) this is similar to the spatial modulating function shown in (A).

in the next stimulus or stimulus pair being shown at a 20% lower contrast level and an incorrect response (a reversal) resulted in the contrast being increased by 100% on the next presentation of the stimulus. A brief preliminary test run was carried out to establish the approximate range of the thresholds to be measured and to give the subject practice at the task. Only in this preliminary run was feedback given to the subject (audible tones). The staircase proper was then commenced at a contrast level a factor of 10 higher than the predetermined approximate threshold. More than 40 observations were made in each staircase which was continued until at least seven reversals had occurred: the arithmetic mean of the contrast values at all the reversals except the first two (which were disregarded) was then computed and taken as the threshold for each stimulus. Each staircase was repeated at least once and the contrast sensitivity values presented below are the reciprocals of the means of two or three such values. Each observation interval was preceded by an audible tone and lasted 1 s: within that interval the stimulus duration was 800 ms with the maximum contrast being attained for 400 ms (the plateau of the temporal window). Contrast thresholds were measured for stationary and drifting sine-wave gratings of 0.5 cycles/degree. The drifting gratings were modulated at one of three temporal frequencies, 4, 8 and 10 Hz; the direction of drift being randomly assigned by the computer. In the case of the CMod grating the procedure was identical, except that the stimulus was the complex waveform described above drifting at 0.3 Hz in a randomly assigned direction. Following the two presentation intervals (total duration 2 s, giving an interstimulus interval of 400 ms) a third tone signalled the subject to respond. Unlimited response time was given during which the subject was encouraged to rest periodically. The next trial was initiated by the subject's response. These different stimuli were randomly interleaved in the staircase and the paired visual field locations were tested alternately.

Contrast thresholds for speed discrimination. Contrast thresholds for speed discrimination were estimated in an identical staircase to the detection thresholds, except that a pair of gratings differing in temporal rate of drift were presented in the two intervals and the subject reported the interval in which the grating drifting at the faster speed had appeared. The spatial frequency was 0.5 cycles/degree and the other stimulus parameters were identical to those employed in the threshold detection estimations. Direction of drift was randomly assigned by the computer and the stimulus duration was also chosen randomly from four preset values in the range 360−420 ms to minimize the possibility that the subject might utilize distance and duration cues in the speed discrimination task. Contrast thresholds for two temporal frequency pairs were determined: 8 and 10 Hz (i.e. velocities of 16°/s and 20°/s, representing a Weber fraction of 25%) and 4 and 8 Hz (velocities of 8°/s and 16°/s representing a Weber fraction of 100%). The two staircases were interleaved. The contrast detection thresholds had been previously determined for the three temporal frequencies employed in the two staircases and a pair of stimuli were always presented at the same contrast ratio as their respective detection thresholds in order to minimize any contrast cues in the discrimination task. Furthermore, as the contrast of each of the paired stimuli changed with every trial it is unlikely that the subjects were able to utilize contrast cues. The contrast threshold measured and used in the subsequent data analysis was in both staircases the contrast of the 8 Hz stimulus. The staircase procedure used to measure the contrast thresholds for speed discrimination was in all other respects identical to that employed in the threshold detection experiments described above.

Direction-of-motion discrimination (LMod and CMod gratings) and orientation discrimination. A single stimulus forced-choice procedure was used for DOM and for orientation discrimination. The drifting grating was shown in a single interval: the direction of drift was randomly assigned and the subject reported the direction of motion. The stimulus was either a 0.5 cycles/degree LMod sine-wave grating drifting at 8 Hz or the CMod grating described above drifting at 0.3 Hz. The two staircases were interleaved. The contrast thresholds for direction discrimination were determined in an otherwise identical staircase procedure to that used to determine detection thresholds. In the case of orientation discrimination in the single interval a grating was presented which was oriented either plus or minus 3° from vertical and the subject reported the direction of tilt. The contrast threshold was determined in the same manner as for DOM discrimination.

Cortical mapping

One patient (Case 1), as a standard procedure in the assessment of his epilepsy, underwent subdural implantation of an 8×8 array of stimulating and recording electrodes (stainless steel discs; 0.5 cm diameter, centre to centre separation 1 cm). A standard stimulation protocol was followed: 60 Hz, 0.2 ms width square pulse trains of 1 s duration. Stimulus intensity was increased until an effect occurred.

Neuro-imaging

All of the patients included in the study had had neuro-imaging studies as a part of their standard clinical work-up and these were MRI studies in all patients except Case 8 in whom X-ray CT images were available. The MRI studies described were carried out on a 1.5 tesla imager. Both T_1-weighted images with and without gadolinium-DTPA enhancement and T_2-weighted images were obtained in coronal, axial and parasagittal planes.

The authors consider that detailed case histories of the three cases showing abnormal motion perception are desirable to establish the anatomical features underlying the visual abnormalities in these cases, so that comparisons can be made with other cases. These case histories are given in the Results.

RESULTS

The first subject to be examined in this study is Case 1 in whom contrast thresholds were measured before and after surgery. The results obtained in this patient established the experimental protocol used in the remainder of the study.

Case 1: history

Case 1 was a 25-year-old male. Seizures were observed at age 7 years when he was described as having 'staring spells' lasting 1–2 min. At age 13–14 years there was an alteration in the character of his seizures which became brief tonic/clonic seizures preceded by an aura of 'blurred vision' and a lightheaded feeling. This remained the most common seizure type but he reported that on some occasions he experienced a right-sided or bilateral sensory aura. On other occasions he had observed a colourless 'fog' moving from the periphery of the right visual field and stopping abruptly at the midline. From his simulation in the form of a hand gesture this fog appeared to be travelling at ~40°/s. Objects in his visual field were never seen in motion and he was uncertain as to the extent that objects remained visible as the 'fog' passed across the right hemifield.

Clinical neurological examination was normal except for an uncertain right plantar response. Visual acuity was 20/20 bilaterally and performance on the Farnsworth–Munsell 100 Hue test and Goldmann perimetry were within normal limits. There was no evidence of parietal or pyramidal dysfunction and no hemiatrophy. A preoperative MRI scan (Fig. 3) revealed focal atrophy of the left posterior parietal convexity with an enlarged superficial vessel and appearances suggesting a left parietal heterotopy (Fig. 3, arrows). The remainder of the brain appeared normal with the exception of mild cerebral atrophy. Left temporal spike and sharp wave discharges were recorded on scalp EEG. Scalp telemetry recordings demonstrated the seizure onsets to occur from the left posterior quadrant. Concern over the risk to normal function located in this region resulted in a decision to improve the localization of the seizure onsets, as well as perform cortical mapping by means of a subdurally placed multi-contact grid.

A left fronto-parieto-occipital craniotomy was carried out (Fig. 4) with the infero-posterior limit of the craniotomy 1 cm from the occipital pole (Fig. 4, *upper panel*, asterisk). The enlarged superficial vein noted on MRI is indicated in Fig. 4 (arrow). The gyral anatomy was distorted. Electrocorticography revealed continuous epileptic activity in the occipito-parietal region with phase reversal at points labelled 'A' and 'B' in Fig. 4. Motor responses were observed following stimulation of points 1, 2, 3, 5 and 8 and this area represents the motor strip. It therefore seems that the large anterior vein is situated in an equivalent location to the posterior ramus of the lateral sulcus in a normal brain.

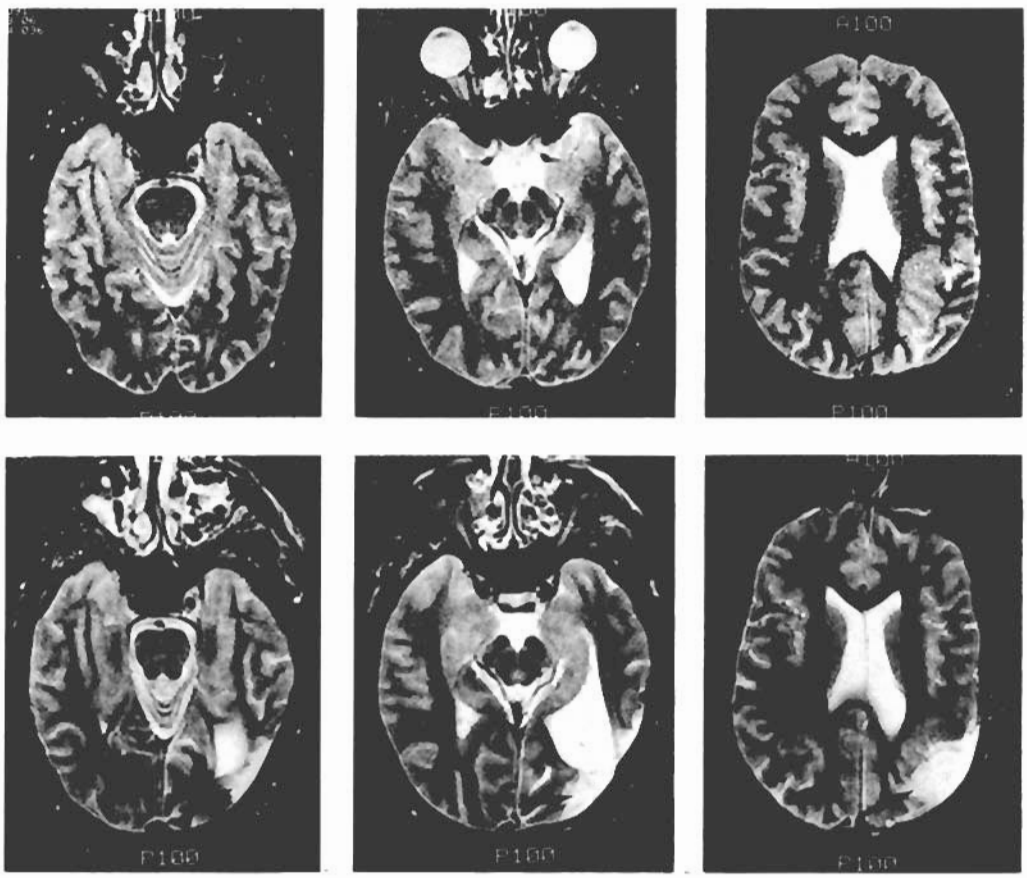


FIG. 3. T_2-weighted axial MRIs of Case 1. In the preoperative images (*upper row*) the left parietal developmental anomaly is seen (arrows) and in the lower cuts asymmetry of the occipital horns of the lateral ventricles with cortex of normal appearance. Postoperatively the extensive occipito-parietal resection is seen (arrows) together with the increased dilatation of the left lateral ventricle (*lower row*). *See also* Fig. 12.

The subdural electrode grid was positioned (Fig. 4, *lower panel*) and the dura and skull flap closed. The grid is shown diagramatically in Fig. 5 with the asterisk again marking the position of the occipital pole and the position of the large vein indicated.

The patient was allowed to recover from his craniotomy for 2 days before electrical stimulation was begun. During the period that the grid was in place both EEG and time-synchronized video monitoring were carried out. On electrical stimulation of electrodes marked 'M', movements of right wrist, fingers, forearm or right corner of his mouth were observed. On stimulation of electrodes marked 'S', a variety of somatosensory sensations were reported, some involving contralateral limbs, some bilateral. 'A' indicates a single auditory phenomenon. Unmarked electrodes produced no visible or subjective change when stimulated.

Visual phenomena occurred on stimulation of electrodes marked 'VB' when he reported

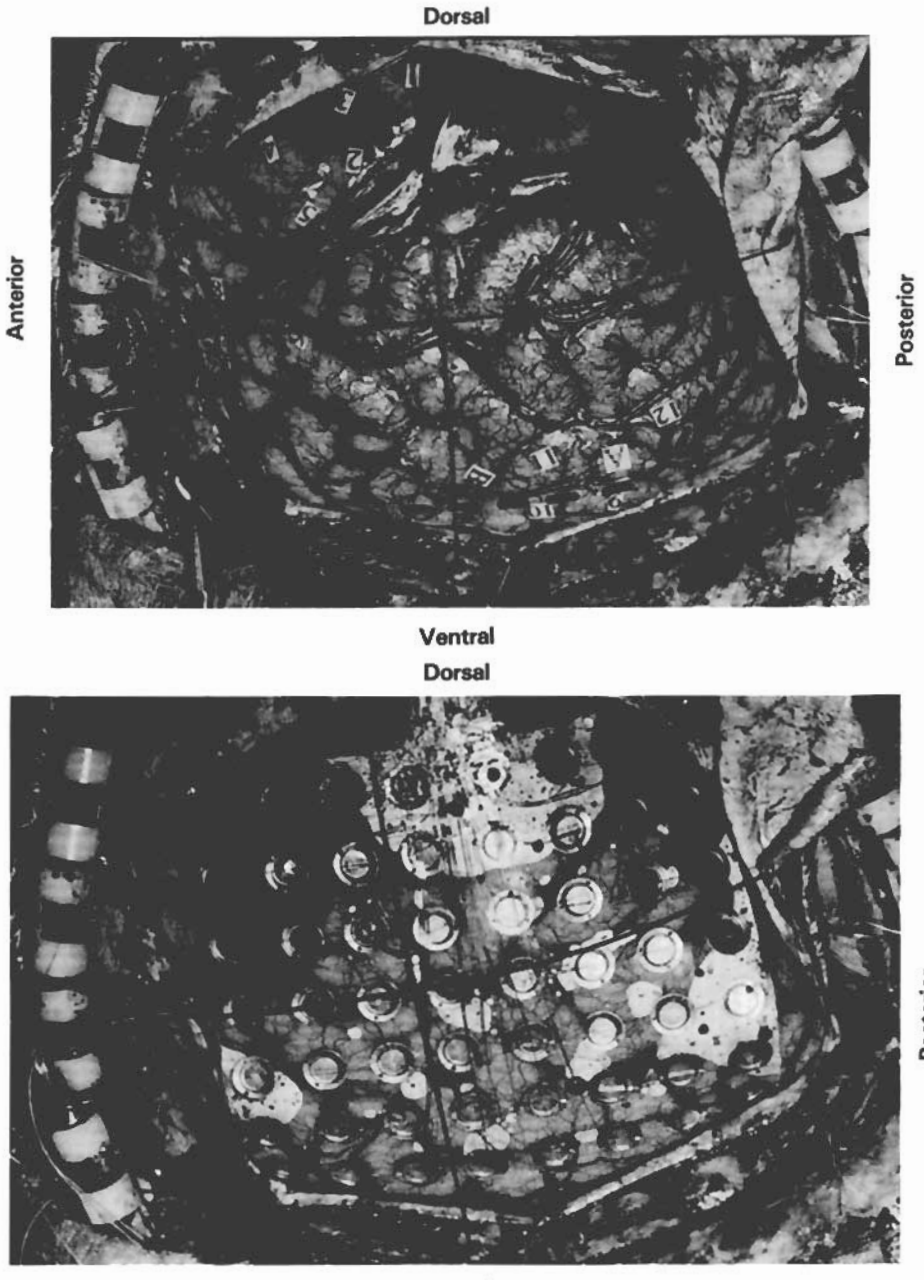


FIG. 4. *The upper photograph shows the left occipito-parietal region* of the brain of Case 1 exposed at craniotomy. Anterior is on the left and ventral is at the bottom of this and all subsequent operative photographs. The numbers 1–8 (inverted because the surgeon stood at the patient's vertex) mark regions which on stimulation generated motor responses in the contralateral face or arm. Other symbols mark locations at which visual sensations were generated. 'A' and 'B' are points where there is phase reversal. The large vein (arrow) continues anteriorly along the Sylvian fissure. The dashed line shows the limits of the resection which was subsequently carried out. The occipital pole was measured to be 1 cm from the posterior limit of the craniotomy (asterisk). The lower photograph shows the 8×8 array of electrodes implanted subdurally for stimulation and recording before the resection was carried out. The centre to centre separation of the electrodes is 1 cm. A schematic diagram showing the results of stimulation is shown in Fig. 5.

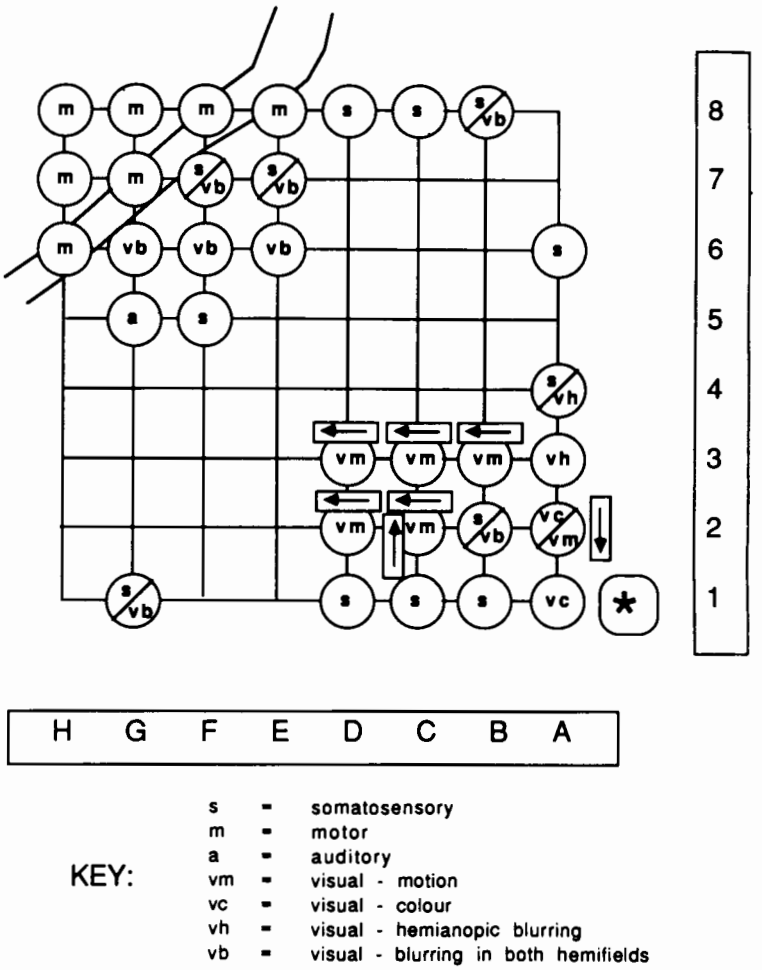


FIG. 5. Diagram of the subdural electrode grid. The position of the occipital pole is shown by the asterisk and the position of the large cortical vein is shown diagramatically at top left. Somatosensory responses, often mimicking symptoms occurring at the onset of seizures, were abnormally widely distributed throughout the occipito-parietal cortex. Electrodes at which stimulation gave rise to visual and motor phenomena are also indicated. The arrows denote the direction of motion of the evoked percept in the contralateral visual field. The cortical region which, on stimulation, evoked moving phosphenes in the contralateral field is located from 30 to 50 mm from the occipital pole.

non-specific visual blurring, 'VC' where he reported the appearance of a coloured spot in the contralateral field and 'VM' where he reported motion of a colourless fog. The latter experience was the same as the subject's aura mentioned above. In the case of the moving fog, and as was the case with his seizures, the patient reported that there was no motion of objects seen within the visual hemifield affected. Specific descriptions of the results of stimulation are given in the Appendix. In view of the difficulty in locating a precise epileptogenic focus, and having established that stimulation of none of the locations stimulated interfered with language functions it was decided to proceed with resection of all the cortex which, when stimulated, had reproduced symptoms associated with

seizures. The extent of this resection is shown by the dashed lines in Fig. 4 and it included lateral occipital and parietal cortex. In the postero-superior aspect of the resection there was found to be an abnormal area of cortical material beneath the most superficial grey matter. Pathological examination of the specimen was consistent with a migration defect. Postoperative MRIs are shown in Figs 3 and 12.

Case 1: experimental results

Post-operatively visual acuity remained 20/20 bilaterally. Goldmann perimetry was normal before surgery including isopters to coloured targets. The field defect 28 days postoperatively (to the I4e target) is shown in Fig. 1. It had been anticipated that a resection in this location was likely to damage the upper fibres of the underlying optic radiations and the field defect shown is typical of a radiation lesion: it is wedge shaped and shows minor incongruity. Comparison with larger targets revealed a 'sloping' lateral border to the field defect. There was slight depression of function in the upper quadrant also immediately postoperatively which subsequently recovered. Isopters to the identification of coloured targets were also preserved in the upper quadrants. Although he has continued to have seizures following the resection he has never again experienced the visual aura of motion in the right hemifield.

In view of the anticipated field defect, all testing was carried out in the upper quadrants with the stimulus centred at an eccentricity of 10°. Before surgery contrast sensitivity was determined for three tasks: detection and DOM discrimination of a 0.5 cycles/degree grating drifting at 8 Hz and discrimination of an 8 and 10 Hz grating pair. There was no difference between the results obtained at an eccentricity of 10° in either the left or right (ipsilateral or contralateral) upper quadrants (Fig. 6). In the immediate postoperative period these tests were repeated and it was found that there was a twentyfold rise in the contrast threshold for the speed discrimination task (open triangles in Fig. 6) and there was a slight increase in the threshold for DOM discrimination (by a factor of two) closed squares in Fig. 6). In the ipsilateral quadrant there was no change in the thresholds after surgery. In view of the marked impairment of speed discrimination for the 20% speed difference found postoperatively it was decided also to test, using 4 and 8 Hz as the pair to be discriminated (closed triangles in Fig. 6). The contrast threshold for this task was also elevated, being a factor of 20 higher in the contralateral than in the ipsilateral hemifield. These measurements were repeated at intervals over the following 7 months. The detection and DOM discrimination thresholds returned to normal. There was a substantial improvement in the threshold for discriminating 8 and 4 Hz but little change in the threshold for the more difficult speed discrimination task.

At 28 days postoperative, tests were carried out in addition to those plotted in Fig. 6. The purpose of these tests was to include a second DOM task using the CMod grating stimulus described in Patients and methods. It was also decided to include a task which did not depend upon intact motion sensitive mechanisms and the orientation discrimination task was chosen for this. These results and the 28 day results in Fig. 6 are replotted in Fig. 7 (*upper panel*), not as absolute contrast sensitivity values but as ratios of the discrimination to the detection contrast thresholds. The paired bars give the results for the ipsilateral field (solid bars) and the contralateral field (shaded bars). The small difference in the ratios for sine-wave DOM discrimination (Fig. 7, task 1) and the lack of any difference in the ratios for orientation discrimination (Fig. 7, task 5) should be compared

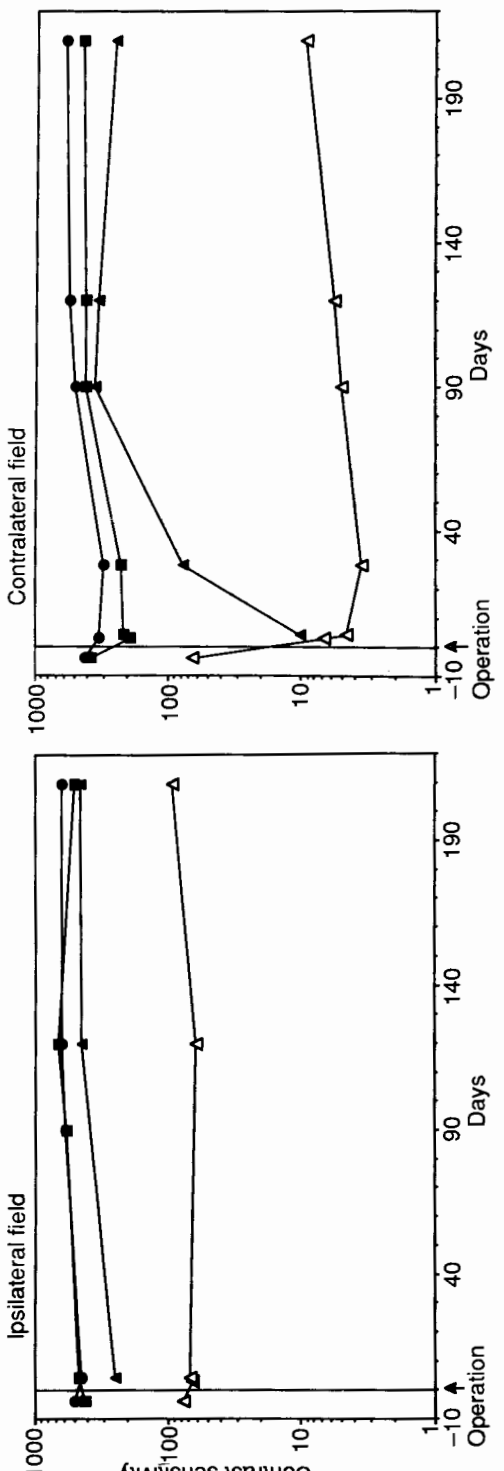


FIG. 6. Contrast sensitivity is shown for four tasks tested at an eccentricity of 10° (SF = 0.5 cycles/degree; drift frequency = 8 Hz) in the left (ipsilateral) and right (contralateral) upper quadrants of Case 1. In the ipsilateral quadrant there was no change in his contrast sensitivity for detection (closed circles), direction discrimination (closed squares) or speed discrimination (triangles: closed = 4 and 8 Hz; open = 8 and 10 Hz) following surgery. In the contralateral quadrant there was a small fall in sensitivity in the detection and direction discrimination tasks which subsequently recovered but there was a tenfold fall in sensitivity in his ability to discriminate two stimuli drifting at 8 and 10 Hz, respectively. Post-operatively we also examined his ability to discriminate 4 and 8 Hz and this was initially impaired but recovered (*see also* Fig. 7). Data in this and all subsequent figures are the means of at least three threshold estimates each of which was obtained from a 40 trial staircase.

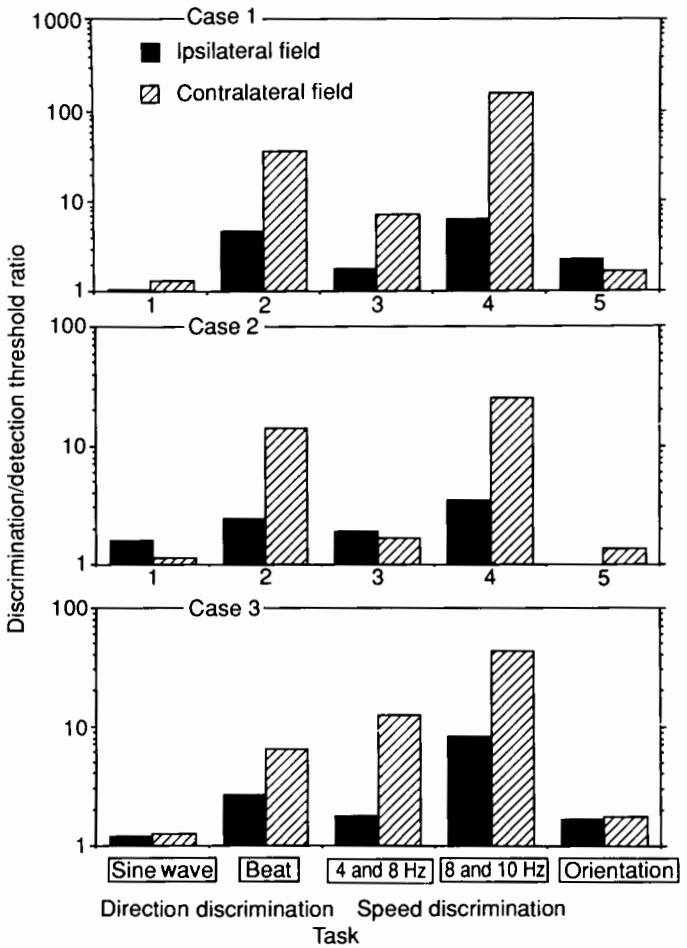


FIG. 7. These results are detection and discrimination threshold ratios for five tasks: direction discrimination of a sine wave and of a contrast-modulated grating (beat); speed discrimination of two gratings drifting at 4 and 8 Hz and 8 and 10 Hz; and orientation discrimination of a stationary grating. The results shown are obtained at an eccentricity of 10° in the ipsilateral and contralateral upper quadrants of Cases 1 and 2 and at an eccentricity of 20° in Case 3. In all cases the threshold ratios do not differ for the orientation discrimination and direction discrimination of sine-wave tasks. The ratios are greater (indicating a relative impairment in the ability to discriminate the quantity as opposed to detect the presence of the stimulus) in the contralateral field for the direction discrimination of the beat and the speed discrimination tasks. The results shown for Case 1 were collected 28 days postoperatively, subsequently there was an improvement in the ability to discriminate 4 and 8 Hz (*see* Fig. 6) by which time the deficit more closely resembled that shown by Case 2.

with the elevated discrimination to detection ratios for the other three conditions. Note from Fig. 5 that there was subsequent improvement in the thresholds for 4 and 8 Hz discrimination. The data in Fig. 7 are shown together because they were all obtained at the same test session (28 days).

It is of interest to note that Case 1 has not noticed any gross symptomatic consequences of the perceptual deficit that we have quantified here. When asked to view the same moving

stimulus in right and left hemifields he has reported only that the object moving in the right (affected) hemifield appears to be moving more slowly than when viewed in the unaffected hemifield. From a functional point of view he has not noticed any problem except when playing racketball: he has noticed that, if a ball passes from left to right across his visual field in a more or less tangential trajectory, it appears to slow down when it enters his right visual field and his subsequent forehand racket stroke (he plays racketball right-handed) is always inaccurate. For other shots, when he is able to track the ball or he is playing backhand shots he has noticed no difference in his performance.

Case 2: history

Case 2 was a 20-year-old woman who developed minor seizures at the age of 7 years. At the onset of her seizures her vision is blurred and observors have told her that her eyes deviate to the right. She may occasionally clench her teeth but is able to comprehend, talk and follow commands. The episodes last for 30−45 s and had occurred once or twice a month. On examination there were no abnormal neurological findings and Goldmann visual fields were normal. Scalp EEG revealed non-specific abnormalities only. Magnetic resonance images demonstrated a ventral occipito-temporal lesion that did not enhance with gadolinium-DTPA (Fig. 8). Electrocorticography following a left temporo-occipital craniotomy under general anaesthesia revealed occasional spikes, sharp waves and spike and wave discharges from the inferior lateral occipito-temporal surface. No electrical stimulation was carried out. On inspection of the brain a pale lesion could be seen at the base of the temporo-occipital region just behind a large temporo-parietal bridging vein (Fig. 9). As the bulk of the lesion arose from the ventral temporo-occipital region it was necessary to remove some cortex of normal appearance in the lateral temporo-occipital region. The extent of the removal is also shown in Fig. 9 and postoperative MRIs are shown in Fig. 8, the location of the normal-appearing lateral occipital cortex which was damaged is shown in Fig. 12 (*upper panels*, arrow). Note that the vein draining into the transverse sinus was preserved. The superior and medial aspects of the lesion blended imperceptibly with the surrounding tissue and a complete removal was not attempted in order to minimize damage to the optic radiations. Histological examination revealed a dysembryoplastic neuroepithelial tumour.

Case 2: experimental results

Postoperatively visual acuity remained 20/20 in each eye, there was no change in the Goldmann fields (Fig. 1) including isopters to coloured targets. When this patient was seen the results obtained in Case 1 were known and therefore the entire range of tests was carried out both before and after surgery in both upper quadrants. Figure 10 (*upper panel*) shows the pre- and postoperative results obtained in Case 2 for detection and DOM discrimination of a 0.5 cycles/degree grating drifting at 8 Hz presented at 10° eccentricity in the left and right upper quadrants of the visual field. Testing was carried out on the fourth and 28th days following surgery. The resection did not affect either threshold in either quadrant (closed circles and closed squares). Also plotted are the thresholds for speed discrimination of the 4 and 8 Hz pair (closed triangles) and the 8 and 10 Hz pair (open triangles), there was a tenfold elevation in the contrast threshold for discrimination of the 8 and 10 Hz pair in the contralateral field only.

The lower panel of Fig. 10 shows the results obtained in the orientation experiment

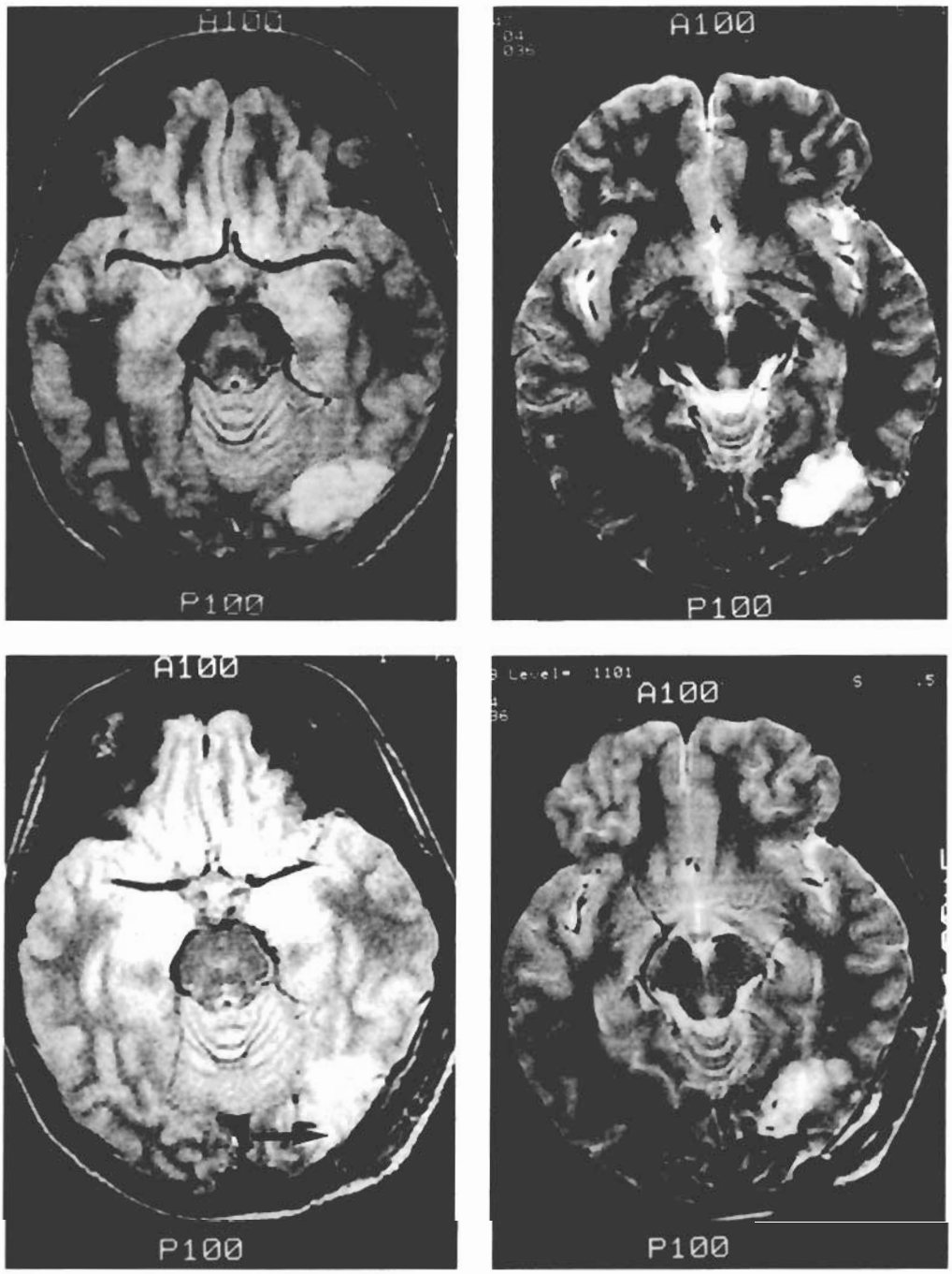


FIG. 8. Axial T_2-weighted MRIs of Case 2 are shown before (*upper pair*) and after (*lower pair*) surgery. The ventral occipito-temporal mass is shown together with the normal appearing lateral occipital cortex which was damaged at operation (arrow). *See also* Figs 9 and 12.

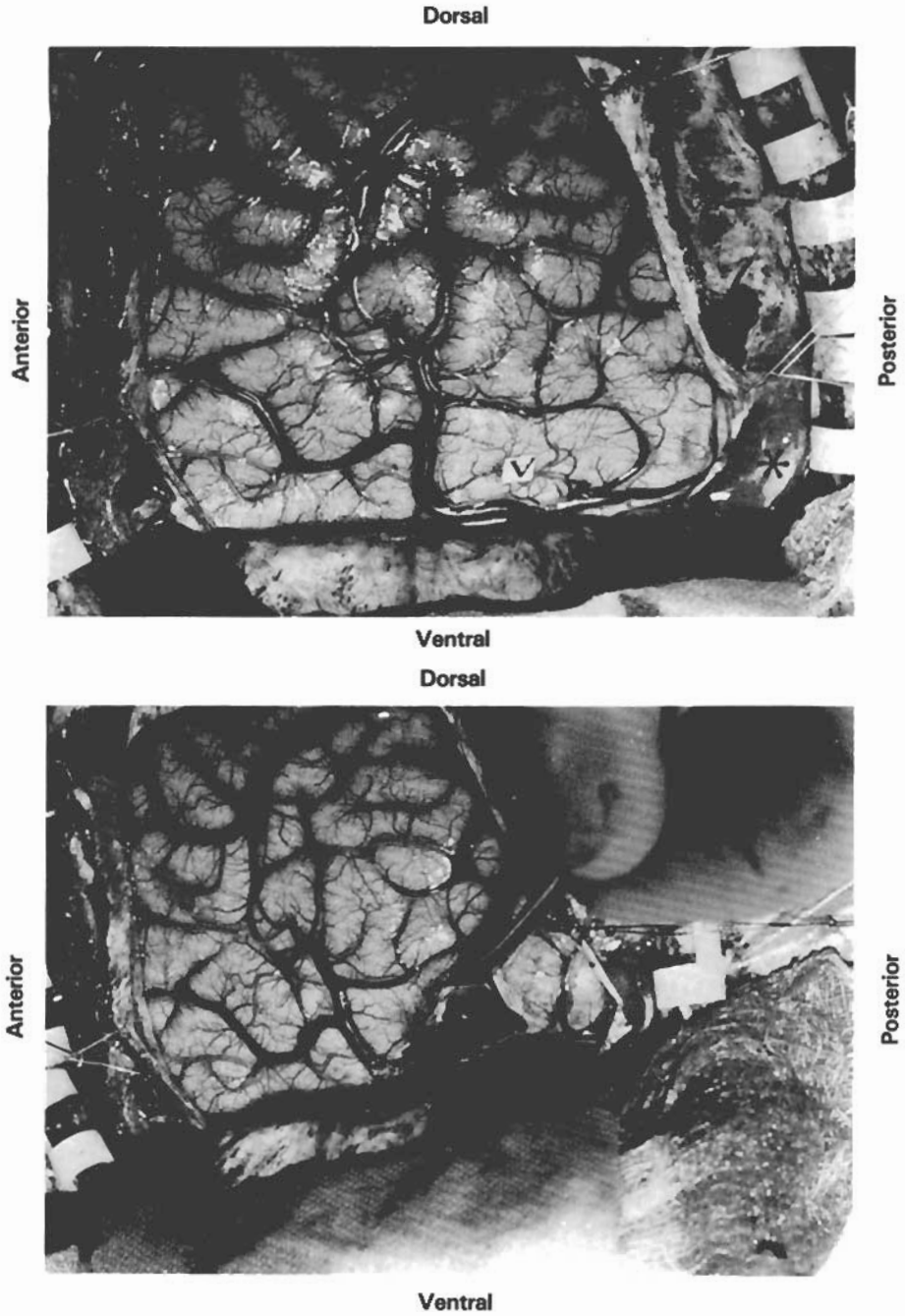


FIG. 9. The upper photograph shows the appearance of the brain at the left lateral temporo-occipital craniotomy in Case 2. Anterior is on the left and ventral is at the bottom of the photograph. The inverted letter 'A' marks the site at which spike discharges were recorded. The asterisk marks the position of the occipital pole. Because the tumour was largely ventro-medial it was necessary to remove some normal appearing cortex in one of the lateral occipital gyri as indicated by the surgeon in the lower photograph. The damaged region was 3–4 cm lateral to the occipital pole and 2 cm above the tentorium.

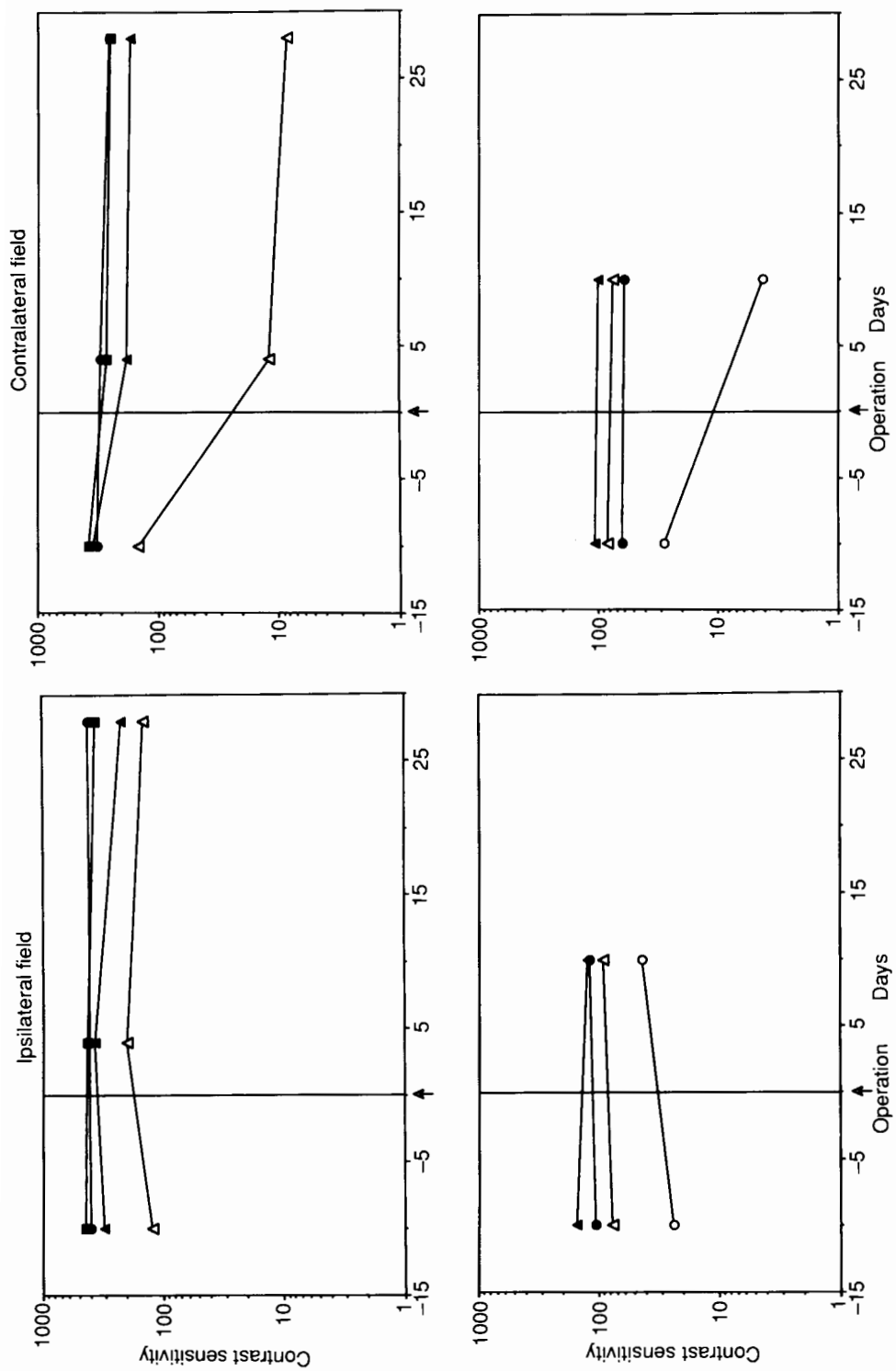


FIG. 10. *Upper panel.* Contrast sensitivity measurements (eccentricity = 10°; SF = 0.5 cycles/degree; drift rate = 8 Hz) for four tasks are plotted against time for Case 2. Postoperatively the only change was an elevation in the threshold for speed discrimination of 8 and 10 Hz drift rates in the hemifield contralateral to the surgical resection. Closed circles = detection; closed squares = direction discrimination; triangles = speed discrimination (closed = 4 and 8 Hz; open = 8 and 10 Hz). *Lower panel.* Contrast sensitivity measurements (eccentricity = 10°) for four tasks are plotted against time for Case 2. Postoperatively the only change was an elevation in the threshold for direction discrimination of the contrast modulated grating (beat) in the contralateral hemifield. There was no change in the threshold for direction discrimination of a sine-wave grating (*upper panels*). Closed circles = detection (beat); open circles = direction discrimination (beat); closed triangles = detection (0 Hz drift); open triangles = orientation discrimination (3°, 0 Hz).

and the DOM discrimination of the CMod grating. Results were obtained preoperatively and on the 10th postoperative day. As with Case 1, there was no change in the thresholds for detection or for orientation discrimination in the case of the stationary grating, and no change in the detection threshold for the CMod grating. There was, however, a tenfold elevation in the contrast threshold for discrimination of the direction of drift of the 'beat' or CMod grating.

The results obtained on postoperative days 10 and 28 have been replotted as discrimination : detection contrast threshold ratios in the centre panel of Fig. 7. Unlike Case 1 there was no threshold elevation for the 4 and 8 Hz discrimination task. In this respect the pattern of the deficit resembles that obtained in Case 1 several months after the resection (*see* Fig. 6).

Case 3: history

A 55-year-old man was unable to read upon awakening. Clinical evaluation shortly after this episode revealed a right, mainly lower quadrantic, homonymous field defect. Corrected visual acuity was 20/25 o.d. and 20/30 o.s. Isoptres to coloured targets were normal in the right upper quadrant on Goldmann perimetry. Magnetic resonance images of Case 3 were obtained shortly following the ictus. A left occipital lobe lesion was demonstrated which gave high signal on both T_1- and T_2-weighted images and which did not enhance following the administration of intravenous gadolinium-DTPA (Fig. 11). A diagnosis of occipital haemorrhage was made and an associated primary or secondary neoplasm was considered likely. A left occipital craniotomy was carried out and at operation a limited occipital lobectomy was performed extending superiorly ~ 3 cm off the tentorium and laterally 4 cm from the most lateral portion of the visible mass. Pathological examination of the resected material confirmed a haematoma without a neoplasm but there was evidence of amyloid angiopathy. Postoperative MRIs are shown in Figs 11 and 12. On MRI, involvement of medial occipital cortex was found to be limited to the upper calcarine area posteriorly. This is compatible with the most dense portion of the field defect which involves the lower quadrant and spared the peripheral field.

Case 3: experimental results

In view of the field defect which involved the central portion of the upper field, visual function was tested at an eccentricity of 30° in the upper quadrant and the diameter of the target was 24°. Unlike Cases 1 and 2, and in accord with the visual field changes and likely involvement of the optic radiations, there was a modest elevation of the detection threshold for the 8 Hz drifting grating. The contrast sensitivity to this stimulus was 220 in the ipsilateral quadrant and 128 in the contralateral quadrant. However, as can be seen from the discrimination : detection ratios plotted in the lower panel of Fig. 7, there was a disproportionate elevation in the contrast thresholds for DOM discrimination of the CMod grating (beat) and for the two speed discrimination tasks. There was therefore a similar pattern of loss to that shown in Cases 1 and 2.

Cases 4, 5 and 6: lesions principally affecting striate cortex

Magnetic resonance images of these cases are shown in Fig. 13. All had homonymous quadrantanopias due to medial occipital lobe lesions involving striate cortex and the terminal optic radiations (*see* Fig. 1). Any involvement of extra-striate cortex was limited to the

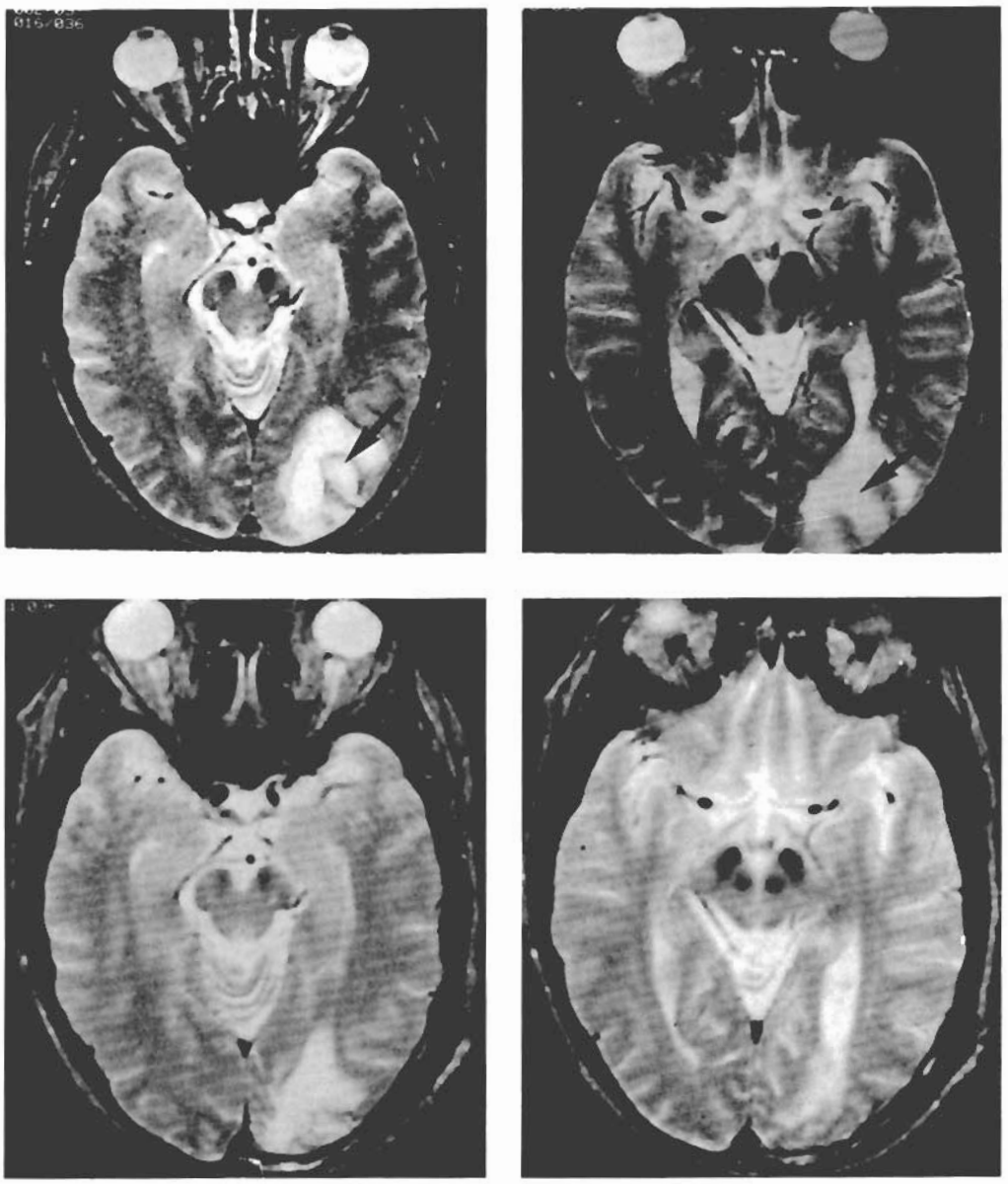


FIG. 11. Axial MRIs of Case 3 are shown. In the upper pair an acute occipital haematoma is shown (arrow). The lower pair were obtained following the lateral occipital resection described in the text in which lateral occipital cortex was removed (*see also* Fig. 12).

medial occipital lobes. Discrimination : detection threshold ratios were not elevated at locations in the residual contralateral hemifields when compared with the performance at mirror symmetric locations in the ipsilateral hemifields (*see* Fig. 14).

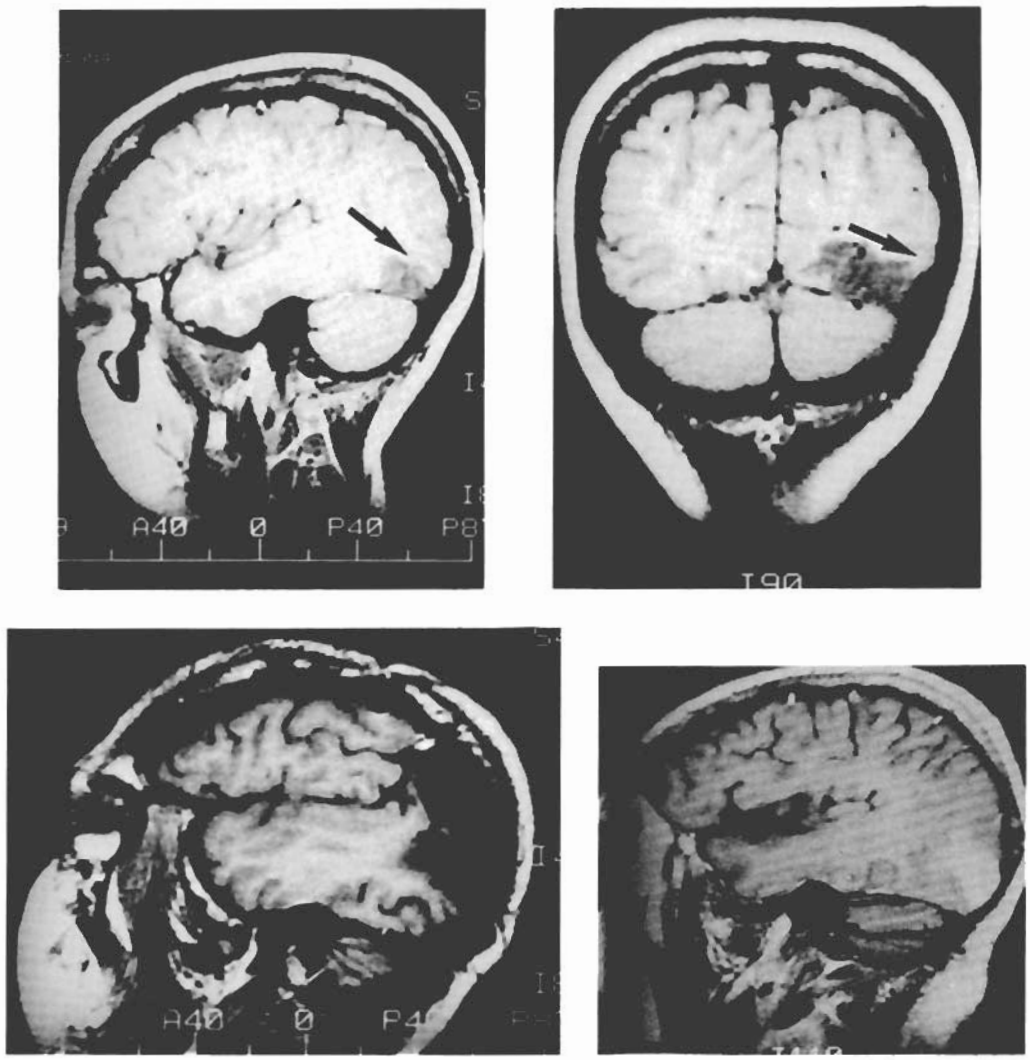


FIG. 12. Magnetic resonance images of Cases 1, 2 and 3, all of whom demonstrated impaired motion perception in the contralateral visual field, are shown. The upper pair are lateral parasagittal and coronal images in Case 2. These are pre-operative images and the region of normal-appearing lateral occipital cortex damaged at surgery is indicated by the arrow (*see also* Figs 8 and 9). The lower two images are lateral parasagittal images from Case 1 (*left*) and Case 3 (*right*). Although the lesion in Case 1 is very extensive, the major overlap with the other two lesions is in its ventral extent, involving the lateral occipital gyri.

Cases 7, 8 and 9: parietal and parieto-occipital lesions

Magnetic resonance images of Cases 7, 8 and 9 are shown in Fig. 15. These were all examples of parietal lobe damage without a visual field defect (Fig. 1). None showed evidence of impaired motion perception in the contralateral visual field (*see* Fig. 16).

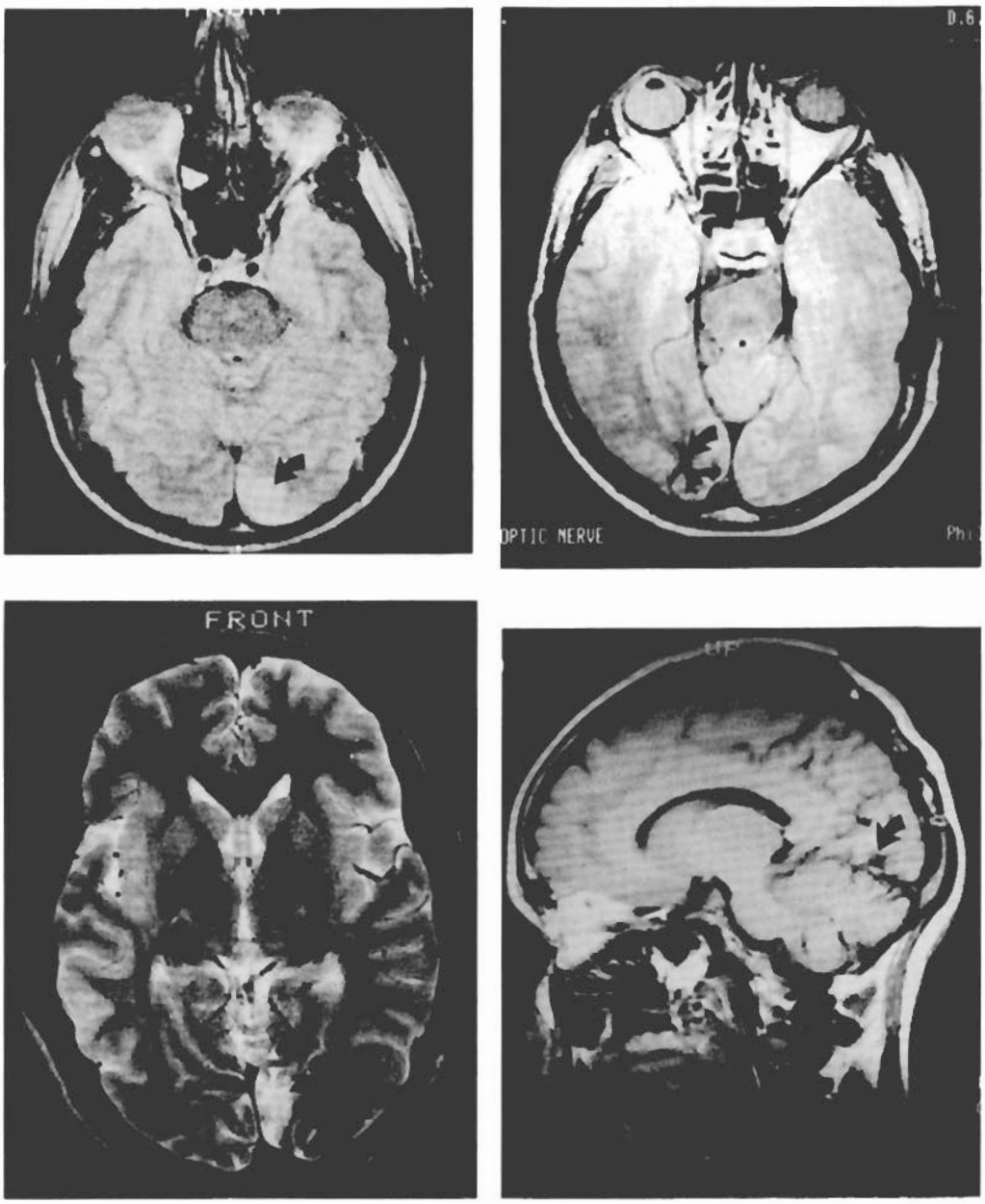


FIG. 13. Axial MRIs of Cases 4 (*upper left*), 5 (*upper right*) and 6 (*lower panels*) are shown. All had homonymous quadrantanopia due to medial occipital lobe lesions (arrows) involving striate cortex and the terminal optic radiations. If extra-striate cortex was also damaged then it was limited to the medial occipital lobes.

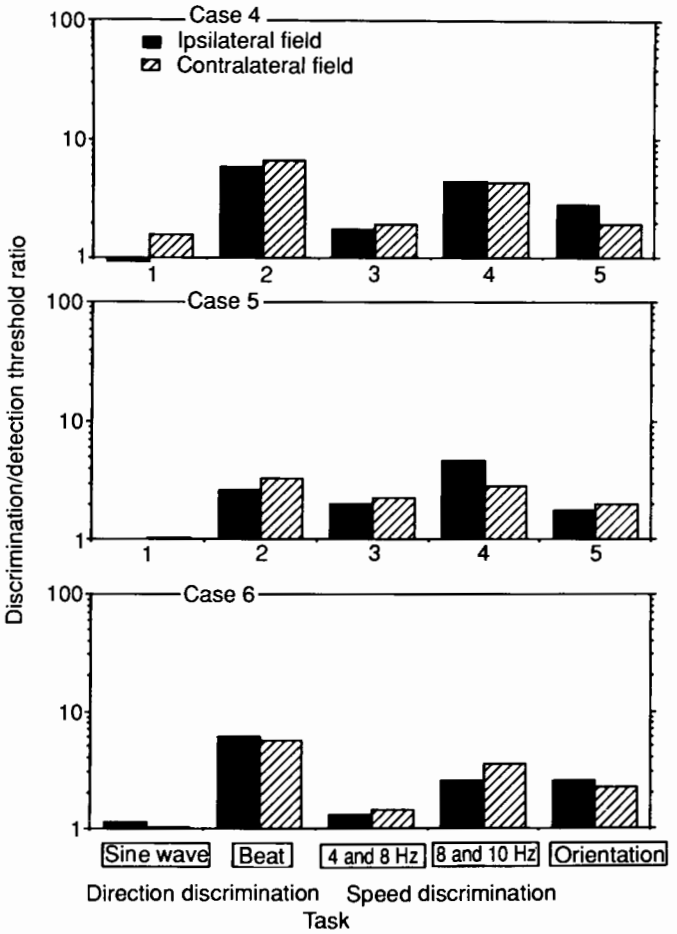


FIG. 14. *See* legend to Fig. 7. Cases 4, 5 and 6 all had field defects due to lesions of striate cortex and adjacent optic radiations, but the lateral occipital gyri were unaffected by the pathology, the discrimination/detection threshold ratios for all tasks were not elevated at locations in the residual contralateral hemifields when performance was compared at mirror symmetric locations in the ipsilateral hemifields.

Cases 10 and 11: occipito-temporal lesions

These two cases are of importance because the lesions are very close to those in Cases 1–3. Both were tumour cases and both had partial upper quadrantanopias due to involvement of the lower fibres of the optic radiations (Fig. 1). Magnetic resonance images of both cases and operative photographs of Case 11, indicating the extent of the resection, are shown in Fig. 17. Neither showed any abnormality in the contralateral hemifield in the tests performed (Fig. 18).

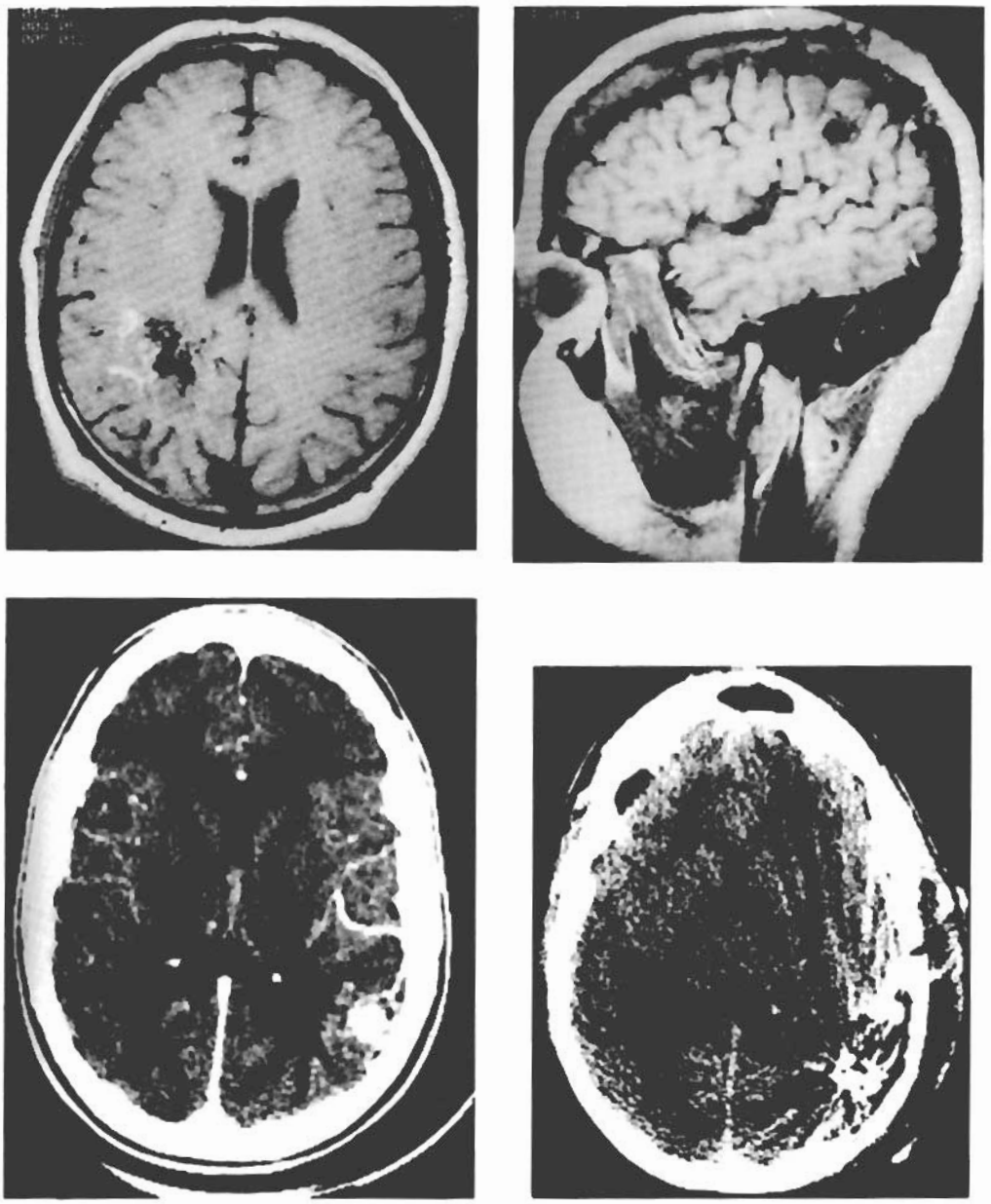


FIG. 15. Magnetic resonance images of Case 7 (*upper left*) showing a parietal vascular malformation with associated haemorrhage; Case 9 (*upper right*) showing the parietal lobe restion and CT images of Case 8 before (*lower left panel*) and following (*lower right panel*) surgical excision of a vascular malformation with associated damage to the occipito-parietal region (not well shown because of clip artefact). None of these lesions caused a visual field defect. None of these cases showed evidence of impaired motion perception in the contralateral visual field. The lesions coincide with much of the parietal damage which occurred in Case 1, reinforcing the view that it is the more ventral and posterior component of that lesion which was important in giving rise to the abnormality.

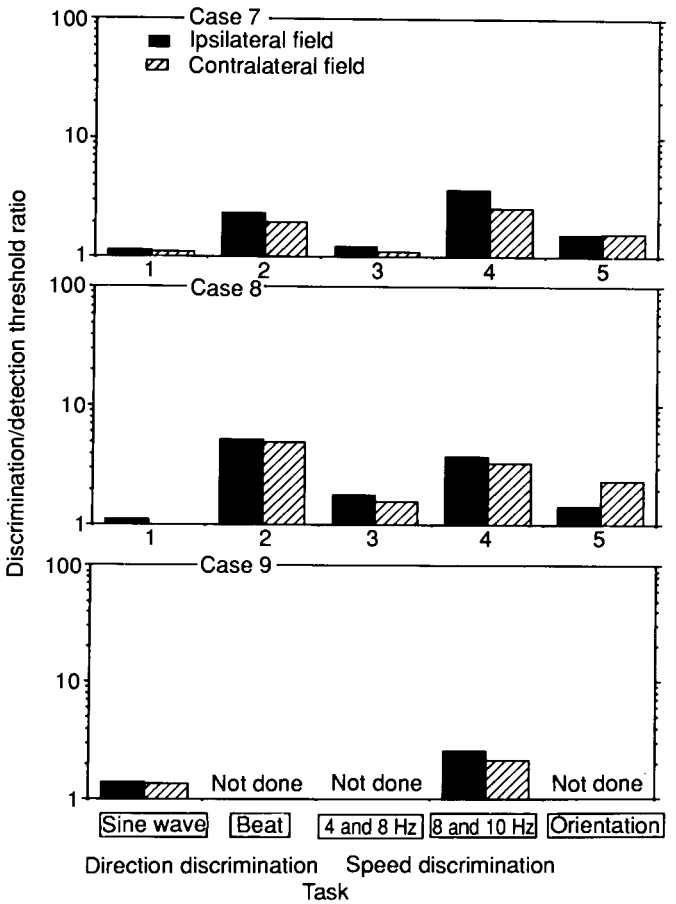
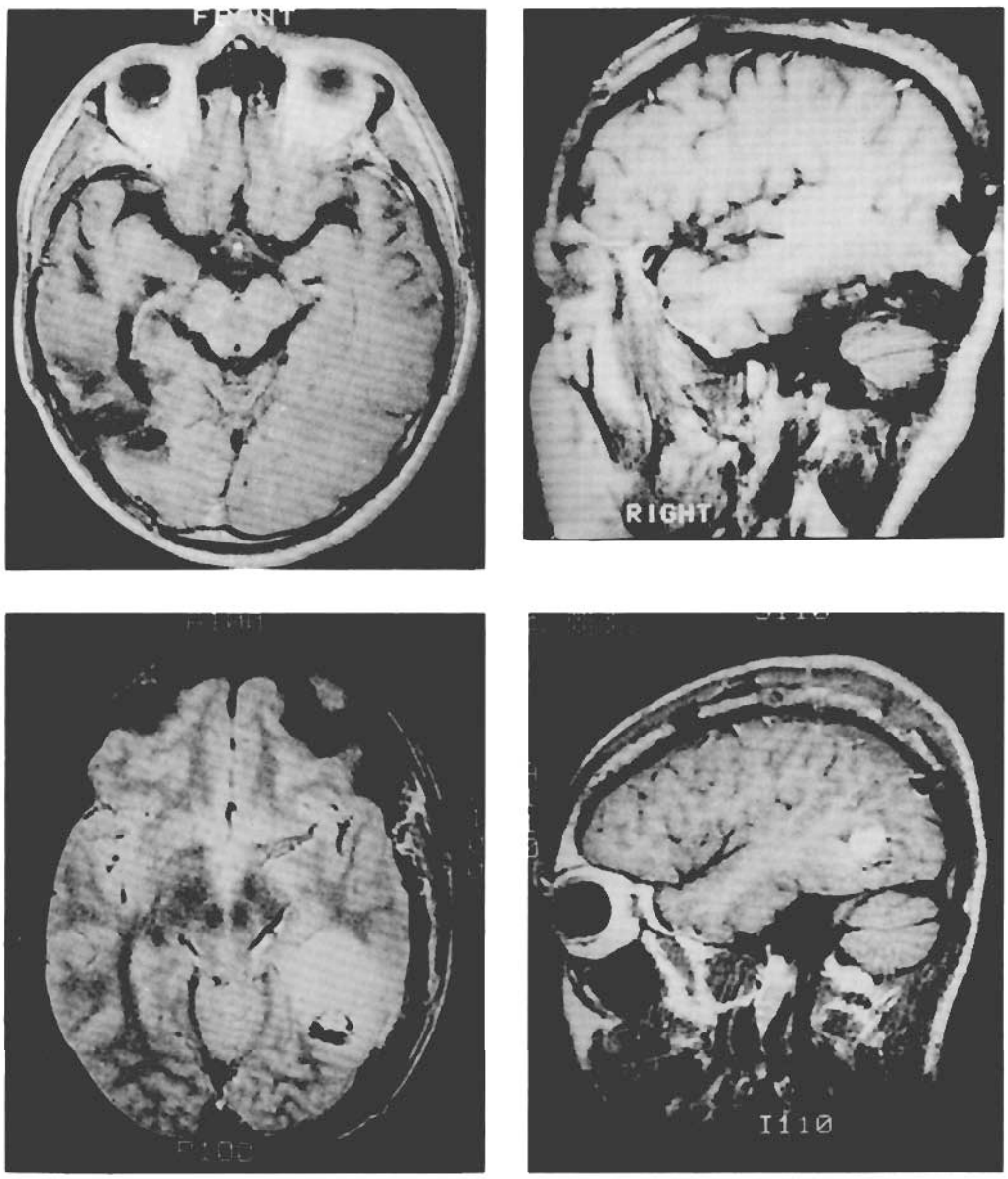


FIG. 16. *See* legend to Fig. 7 for explanation. Cases 7, 8 and 9 were cases of parietal lobe lesions which did not cause a field defect. There was no abnormality in the contralateral visual field on any of the parameters measured. A limited study was carried out in Case 9 as indicated.

DISCUSSION

In this study we have demonstrated that a motion deficit localized to a hemifield can occur following posterior hemisphere damage in humans. The deficit is observed using 'higher order' motion tasks (speed discrimination and DOM discrimination of a CMod grating) but not DOM discrimination of a LMod sine-wave grating or orientation discrimination. To our knowledge this has not been previously demonstrated, although impaired colour vision within a hemifield has been reported several times since the earliest case reported by Verrey (1888) [*see also* Zeki (1990*b*) and Plant (1991) for recent review articles]. The bidirectional ocular motor pursuit anomaly within a hemifield described by Thurston *et al.* (1988) may represent a similar phenomenon, although perceptual testing was not carried out in that case and we have not studied eye movements. In Cases 1

and 2 we were able to show that there was no impairment of motion perception in the ipsilateral hemifield after the resection. Case 3 did have a higher threshold for speed discrimination in the ipsilateral field (*see* Fig. 7) but we do not know what his performance would have been before the resection. He also was an older patient with generalized vascular disease and we cannot exclude contralateral cerebral damage not revealed on MRI. Case 3



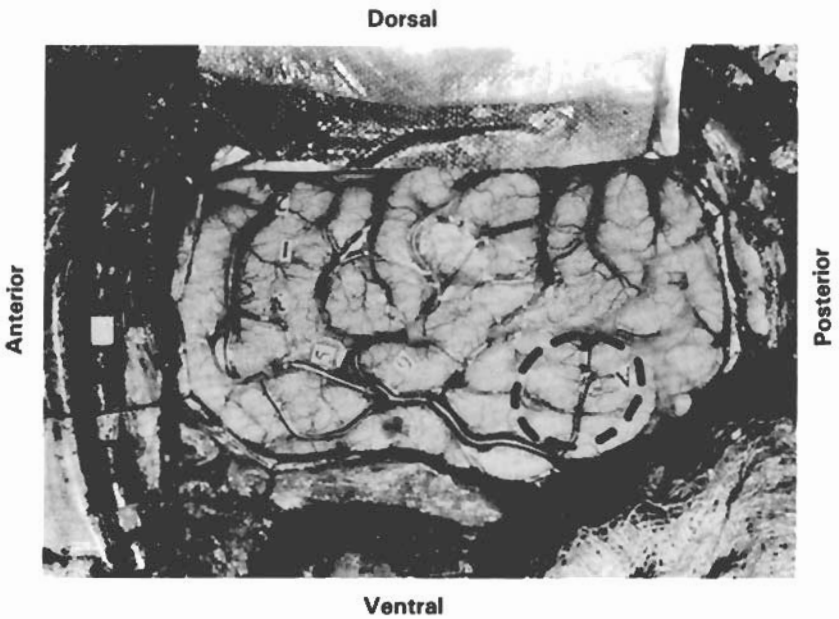


FIG. 17. Postoperative axial and lateral parasagittal MRIs of Cases 10 (*upper row, opposite*) and 11 (*middle row, opposite*). Both were cases of tumour and both had upper partial quadrantic hemianopia due to involvement of the lower fibres of the optic radiations (*see* Fig. 1). The operative photograph of Case 11 (*above*) indicates the extent of the occipito-temporal resection that was carried out (dashed lined). Anterior is on the left and ventral is at the bottom of the photograph. The labels numbered 1–5 indicate the location of the motor strip. Continuous spike and sharp wave activity was recorded from the location labelled 'A'. The occipital pole was measured to be 4 cm further posterior than the posterior edge of the craniotomy. These lesions are very close to those of Cases 2 and 3 being more anterior but neither showed evidence of impaired motion perception in the contralateral hemifield reinforcing the view that it was the damage to the lateral occipital gyri that was important in producing the abnormality found in Cases 1, 2 and 3.

is also a less 'pure' example than the other two cases in that threshold contrast sensitivity was slightly impaired at the tested location in the contralateral hemifield.

As far as the anatomical basis of the observed effects in our three patients is concerned, in Case 1 we are not necessarily dealing with normal anatomy. However, we do think it is significant to note that the region of extra-striate visual cortex which gave rise to a perception of motion in the contralateral hemifield on electrical stimulation (Figs 4, 5), reproducing his epileptic aura, was located on the lateral occipital cortex within 4 cm of the occipital pole. This cortical region is normal in appearance on the MRI (Fig. 3) and is not necessarily involved in the developmental anomaly. In the cortical region corresponding to the anomaly, electrical stimulation gave rise predominantly to non-visual effects (*see* the Appendix).

The lateral occipital region is relatively accessible compared with the ventral occipito-temporal or striate cortex: for this reason it was the visual area most commonly encountered by Penfield in his studies of the effects of electrical cortical stimulation of patients undergoing surgery for intractable epilepsy (Penfield and Rasmussen, 1950; Penfield and Jasper, 1954). Penfield's experience was that seizures originating in the lateral convexity of the occipital lobe gave rise most commonly to the perception of moving, twinkling or pulsating lights either in the contralateral visual field or directly in front of the patient

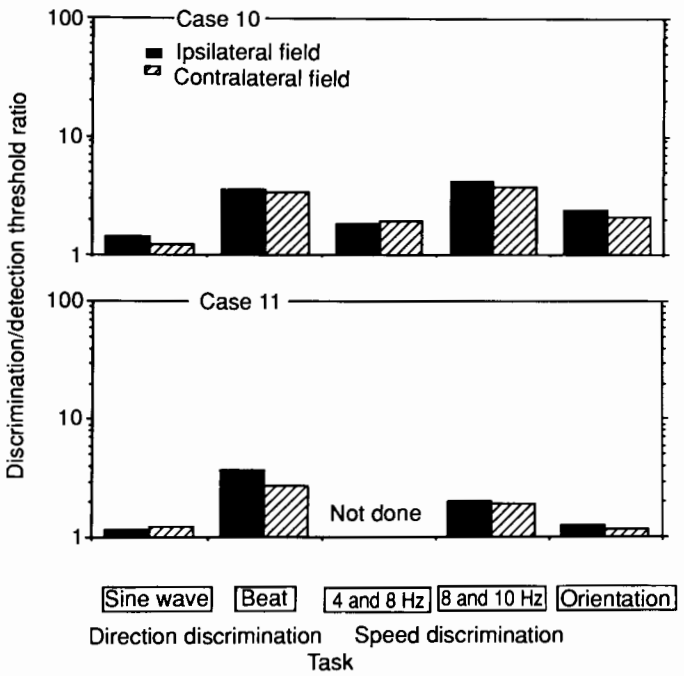


FIG. 18. Case 10 had an extensive temporo-parietal lesion (*see* Fig. 17) which caused an upper partial quadrantic field defect due to involvement of the optic radiations. The tests described previously were carried out in the lower quadrant of the hemifield contralateral to the lesion at an eccentricity of 10° and the results are compared above with those obtained at the mirror symmetric location in the ipsilateral hemifield. There was no evidence of a relative reduction in contrast sensitivity on the discrimination tasks in the contralateral field. Case 11 had a similarly situated occipito-temporal oligodendroglioma and following excision had an upper quadrantic field defect. Once again, however, there was no evidence of impaired motion perception in the contralateral lower quadrant.

(Penfield and Jasper, 1954). In a summary of the effects of electrical stimulation of the lateral occipital lobe (Penfield and Rasmussen, 1950, pp. 139–141) it was a general (although not absolute) observation that stimulation near the occipital pole and the calcarine fissure was more likely to produce coloured phosphenes, whereas stimulation more anteriorly was more likely to give rise to phosphenes which were colourless and moving, flickering or dancing.

We regard the lesion produced in Case 2 as being particularly important because the normally appearing cortex excised was 4 cm from the occipital pole and as such overlaps only the most inferior and posterior portion of the cortical resection in Case 1 (*see* Fig. 12); also corresponding to the region where moving phosphenes were induced on stimulation. The resected cortex in Case 3 included lateral occipital cortex extending anteriorly several centimeters from the occipital pole (4 cm anterior to the limit of the visible haemorrhagic change) and dorsally 3 cm above the tentorium. We suggest that it is also significant that our two examples of extensive occipito-temporal pathology (Cases 10, 11) are very close to the resections in Cases 2 and 3. These lesions are only 3–4 cm more anterior at the occipito-temporal junction and yet have produced no anomaly of motion perception identifiable by our test procedures. This, and the lack of any effect in our three cases

of parietal lobe pathology (Cases 7, 8, 9) reinforces our view that it is the damage to the lateral occipital gyri in Cases 1, 2 and 3 which was critical in producing the deficit in visual motion processing documented in this article.

From work carried out in many laboratories and conveniently summarized by Ungerleider and Desimone (1986), it is known that in the macaque V5-MT is the earliest visual area which is predominantly concerned with the projections to the parietal lobes mediating oculomotor and visuospatial performance (extra-striate areas V2 and V3 also process information destined for V4 mediating colour and form vision). Furthermore, V5-MT contains a retinotopic organization. Although there are dense callosal connections between left and right V5-MT and some representation of the ipsilateral field is found (Newsome and Allman, 1980; Van Essen *et al.*, 1981), an extensive representation of the ipsilateral visual field is not found until higher projection zones (e.g. in the floor of the superior temporal sulcus). Therefore to produce a motion deficit limited to a hemifield it may be necessary for a lesion to interrupt the inputs to V5-MT or damage V5-MT itself. We cannot distinguish these possibilities in our cases. It is possible that the closely located occipito-temporal lesions in Cases 10 and 11 may be further anterior than V5-MT itself, thus giving rise to damage after substantial callosal transfer of information has taken place. Lesions affecting higher visual areas than V5-MT in occipito-parietal cortex might be expected to produce a unidirectional pursuit anomaly throughout the visual field by analogy with the ocular motor impairment of ipsilateral pursuit (Thurston *et al.*, 1988). Such a deficit would not have been detected by the methods used here because the direction of motion of the stimuli was randomly varied in the speed discrimination task.

Striate cortex in the macaque and other non-human primates occupies a considerable portion of the lateral occipital lobe, whereas in the human it is located largely on the medial surface of the occipital lobe and in the calcarine fissure. Dorsal and ventral V2 are also likely to be located more medially in the occipital lobe if, in humans as in primates, these areas are contiguous with dorsal and ventral V1 along the representation of the vertical meridian (Horton and Hedley-Whyte, 1984). Dorsal and ventral V3 are, in turn, contiguous with V2 along the representation of the vertical meridian. If this is the case in humans, then the lateral occipital damage in our three cases is unlikely to have involved dorsal or ventral V2. The preserved chromatic fields make it very unlikely that V2 was involved in these lesions as these areas continue to process chromatic information (*see*, for example, Livingstone and Hubel, 1984; Zeki, 1990*b*). Indeed evidence has been presented suggesting that lesions of dorsal or ventral V2 in humans may give rise to dense lower or upper homonymous quadrantanopic field defects (Horton and Hoyt, 1991). Lesions which, in humans, give rise to loss of colour vision in the contralateral hemifield are located in ventral occipito-temporal cortex (Verrey, 1888; Kölmel, 1988), similar in location to the region activiated in PET studies (Corbetta *et al.*, 1990; Zeki *et al.*, 1991). Thus, in the human, the region damaged in cerebral achromatopsia may be located more ventrally than macaque V4.

These differences suggest that the human homologues of V2, V3 or V4 are unlikely to be located in lateral occipital cortex, although we do not know what more fundamental rearrangements may have taken place in the organization of extra-striate visual cortex in humans. Clarke and Miklossy (1990) have demonstrated a strikingly heavily myelinated region on the convexity of the lateral occipital cortex in human pathological material, the inferior margin of which received very dense callosal afferents. The region was centred

on the upper bank of the inferior lateral occipital sulcus in the two hemibrains studied. The posterior and anterior margins were, in one brain, 28 and 36 mm, respectively, from the occipital pole and its inferior border was 10–20 mm from the tentorium (*see* Figs 6 and 17 of Clarke and Miklossy, 1990). As such this region would certainly be included in the region of lateral occipital cortex included in the resections carried out in our cases. It also corresponds well with the region of lateral occipital cortex stimulation in which evoked moving phosphenes in Case 1 (Fig. 5). Beckers and Hömberg (1992) have shown that discrimination of direction of motion of random dot patterns presented in a background of uncorrelated random dots was impaired in the contralateral hemifield following transcranial magnetic stimulation. The critical location to produce this effect was 4–6 cm lateral to the medio-sagittal Nasion–Inion line; this result is compatible with our findings and those of Clarke and Miklossy (1990). However, stimulation of this type does not give rise to illusions or hallucinations of movement.

It seems likely that unilateral lesions of the type we have described may be considerably more common than bilateral lesions giving rise to selective impairment of motion perception. We would predict that cases of more anterior parietal lobe lesions showing impaired ipsilateral pursuit eye movements, an anomaly which is found throughout the visual field and which is likely to occur at a level where transcallosal transfer of information is extensive (Thurston *et al.*, 1988; Morrow and Sharpe, 1990), will not show anomalous motion perception in the contralateral hemifield. We further suggest that in all studies of anomalous motion perception the possibility of hemifield asymmetry should be explored. Without this information studies of unilateral posterior lesions under conditions of free fixation are difficult to interpret (e.g. Vaina, 1989). The paradigm employed in the present study (i.e. seeking examples of anomalous motion perception limited to a hemifield in unilateral lesions) is likely to provide the opportunity to investigate a larger number of human cases of impaired motion perception than has been possible hitherto. With contemporary imaging techniques a clearer idea of the locations of damage giving rise to visual motion perceptual deficits of varying severity should emerge. The characteristics of the motion anomaly in these three cases are further explored in the accompanying article (Plant and Nakayama, 1993) and are placed in the context of other human studies and studies involving cerebral lesions in animals.

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APPENDIX

Results of cortical stimulation in Case 1 (please refer to Fig. 5)

Electrode

A1 At 4V he immediately responded 'colour' and later described a red spot (the size of a coin at 10 feet) that had appeared in the right visual field.

A2 At 8V he saw a change in his vision which he described as a wave travelling downwards in the right hemifield.

A3 At 8V he noticed a slight change in his vision in the right visual field. He described this as blurring and said that it was similar to the onset of a seizure.

A4 Slight hemianopic visual disturbance and tingling in his right hand.

B3 Slight hemianopic visual disturbance.

C2 At 4V he reported a change in his vision like a wave moving from right to left. At 6V he reported, in addition to the visual disturbance, facial tingling.

C3 At 8V he reported a similar change in his vision like a wave moving from right to left and stopping abruptly at the midline.

C8 At 8V he was aware of tingling in his chest and right leg.

D1 At 3V he reported a sensation of warmth throughout his body.
D2 At 4V there was a change in his vision, moving as a wave from right to left. At 6V there was the same change in his vision, but stronger, associated with deviation of head and eyes to the right. These sensations were similar to those occurring at the start of seizure.
D3 At 8V there was a similar change in his vision travelling across to the midline.
E6, E7, F6, G1, G6. At these electrodes he reported visual blurring affecting both hemifields, sometimes associated with somatosensory phenomena.
E8, F8, G7, G8, H6, H7, H8. At these electrodes motor phenomena involving the contralateral arm and face.
G5 At 12V the patient reported the sound of a lady screaming in the distance with an associated echo.