

Altitudinal Hemianopia Caused by Occipital Infarctions

Clinical and Computerized Tomographic Correlations

Richard P. Newman, MD; William R. Kinkel, MD; Lawrence Jacobs, MD

● Two patients with altitudinal homonymous hemianopia caused by bilateral occipital infarctions were studied. Their clinical-computerized tomographic correlations showed the anatomic representation of the superior and inferior visual fields in the lower and upper banks of the calcarine fissure, respectively. The patient with the superior-field defect was unusual because he retained normal color vision and facial recognition abilities, despite having bilateral lesions that have classically produced central achromatopsia and prosopagnosia.

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Altitudinal hemianopia caused by occipital infarctions is extremely rare. Only two cases have been reported with necropsy, both of which had inferior altitudinal visual field defects that appeared to have been caused by infarctions of the upper striate cortex; however, both cases had involvement of infracalcarine parenchyma as well.^{1,2} We report two additional cases of altitudinal hemianopia, one involving the inferior field

and one involving the superior field, caused by occipital infarctions. The clinical-computerized tomographic (CT) correlations showed the anatomic separation of the altitudinal fields in the occipital lobes. The patient with the superior-field defect also showed that, contrary to current concepts of central achromatopsia and prosopagnosia,^{3,4} color vision and facial recognition ability may sometimes remain intact, despite bilateral ventromesial occipitotemporal damage.

REPORT OF CASES

CASE 1.—A 66-year-old woman with Guillain-Barré syndrome suffered a hypoxic episode that resulted in cortical blindness (left eye, no light perception; right eye, light perception only and normal pupillary reactions to light). There was gradual improvement during the next seven days, during which vision returned in the superior field, where she could perceive light, color, and objects.

Visual field testing showed a complete macula-splitting, inferior altitudinal homonymous hemianopia (Fig 1, top). In the superior field, color was perceived normally, but perception of form was defective. The patient was able to identify and count fingers and recognize faces and a variety of other objects in the superior field, but they seemed blurred, displaced downward, and smaller than normal (approximately one half of normal size). On other occasions, objects in view seemed larger than they really were. She had difficulty reading because of "blurriness losing place" and also had difficulty judging distances (objects seemed farther away than they actually were). The patient made frequent

errors in judging which of the examiner's two index fingers was farther away when they were held at different distances in front of her. She made similar errors in estimating her distance from various objects when looking around her room or down the hall. She bumped into walls and furniture as she walked down the hall. The patient stated that she saw the objects but did not realize they were in her way, ie, she misjudged their positions.

Perception of sizes and shapes was also inaccurate. When shown a square block and instructed to outline its shape, the patient indicated a rectangle with horizontal components approximately twice as long as they really were. A comb, 12.7 cm in length and held 25.4 cm in front of her, appeared to be 38.1 cm long and approximately 50.8 cm away from her.

No optokinetic responses could be elicited. The flight of colors⁵ was absent in the right eye, and only green was seen in the left eye. Eye movements (studied by serial bedside observation and electro-oculography) were normal in the horizontal plane and the upward direction; downward pursuits were interrupted by saccades and often did not follow the target through its full range of downward motion. Flashes of light alternately presented in the superior and inferior fields induced normal upward but not downward saccades. Usually, the eyes were directed upward to the position where the flash in the superior field had been presented after it had been extinguished and a new flash had been presented in the inferior field. Auditory cues that the light had flashed in the inferior field resulted in irregular downward searching movements. Still, the patient could move her eyes upward and downward normally on command when no targets were presented. Visual evoked response

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From the Dent Neurologic Institute (Drs Newman, Kinkel, and Jacobs) and Departments of Anatomy (Dr Kinkel) and Physiology (Dr Jacobs), Division of Neurobiology, State University of New York School of Medicine, Buffalo.

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Reprint requests to Dent Neurologic Institute, Millard Fillmore Hospital, 3 Gates Circle, Buffalo, NY 14209 (Dr Newman).

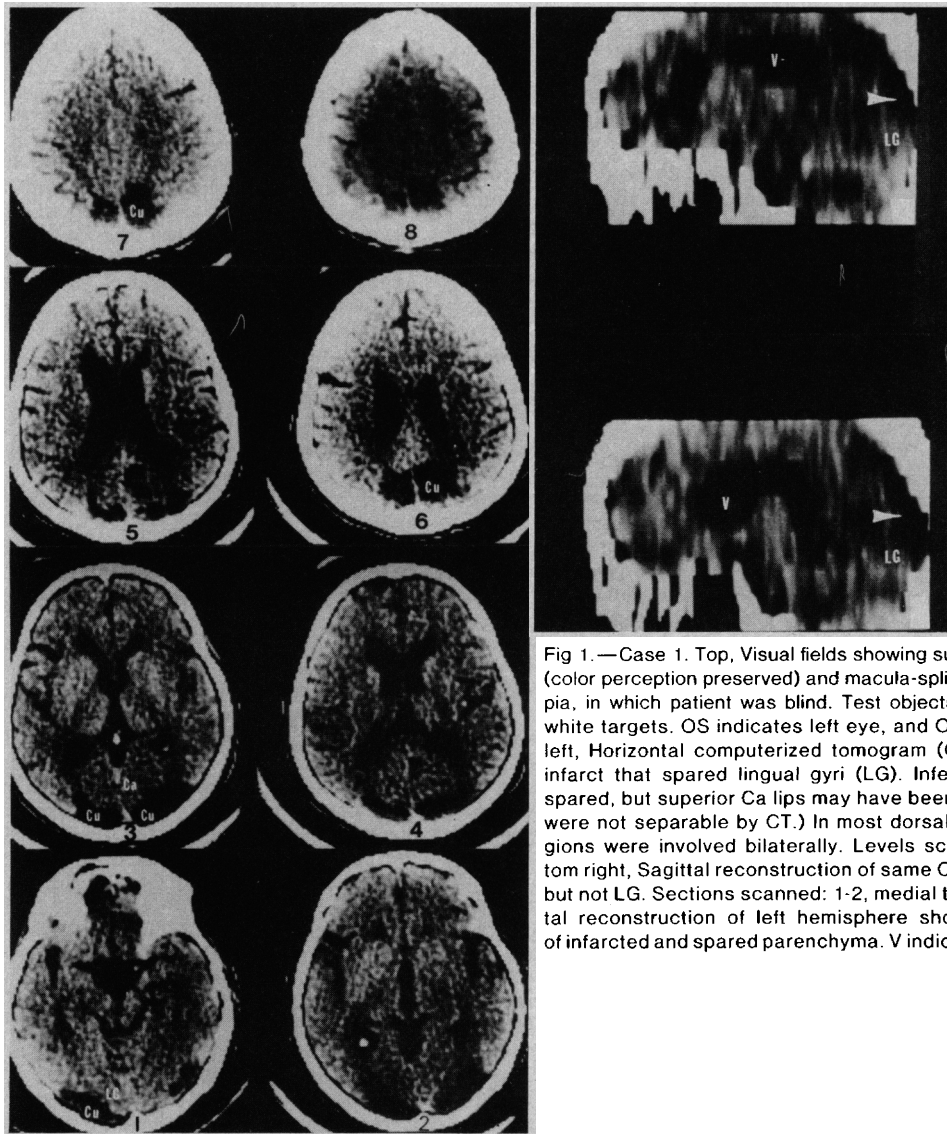
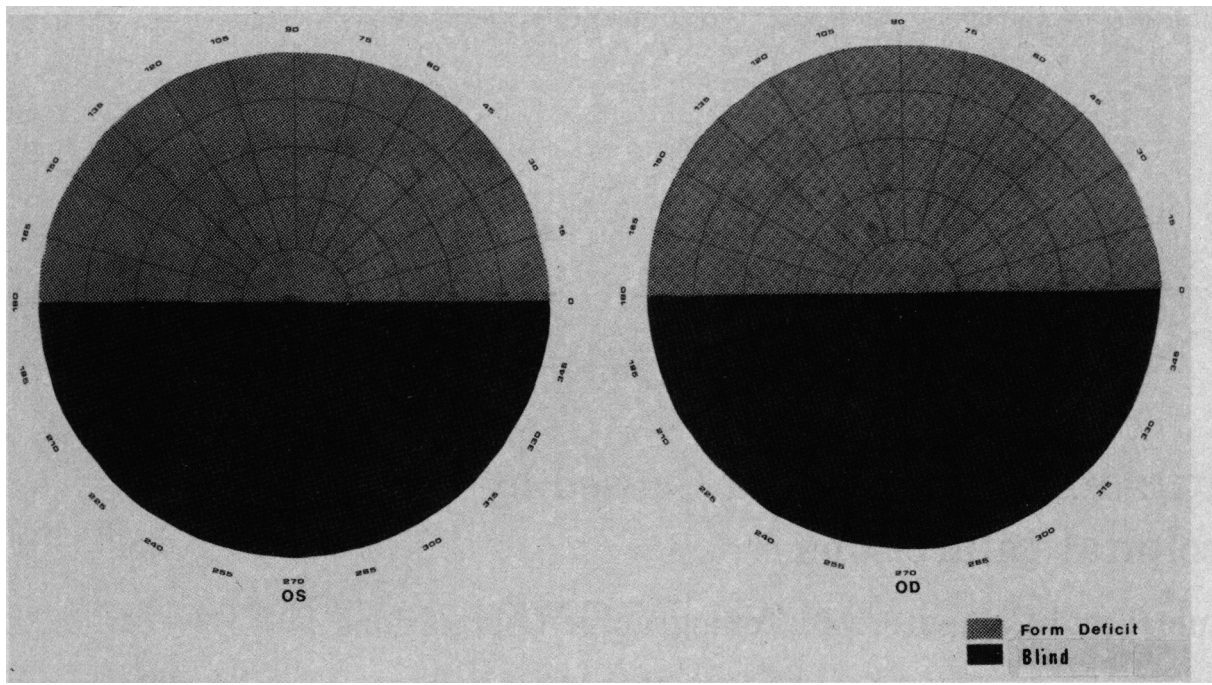


Fig 1.—Case 1. Top, Visual fields showing superior-field altitudinal defect for form (color perception preserved) and macula-splitting inferior-field altitudinal hemianopia, in which patient was blind. Test objects were 6-, 12-, and 15-mm red and white targets. OS indicates left eye, and OD, right eye (tangent field). Bottom left, Horizontal computerized tomogram (CT) showing bilateral cuneus (Cu) infarct that spared lingual gyri (LG). Inferior calcarine (Ca) lips were also spared, but superior Ca lips may have been involved. (Cu and superior Ca lips were not separable by CT.) In most dorsal sections (7 and 8), precuneus regions were involved bilaterally. Levels scanned: 1-8, ventral to dorsal. Bottom right, Sagittal reconstruction of same CT. Infarct (arrowheads) involved Cu but not LG. Sections scanned: 1-2, medial to lateral of right hemisphere. Sagittal reconstruction of left hemisphere showed essentially identical regions of infarcted and spared parenchyma. V indicates right lateral ventricle.

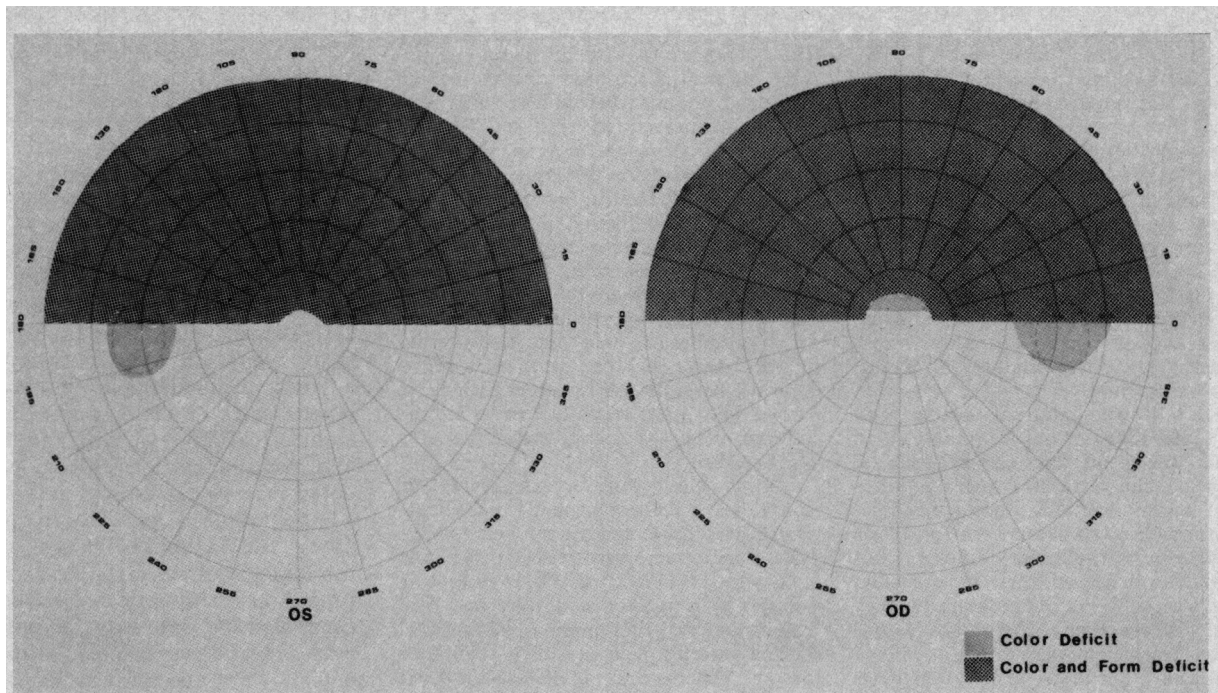
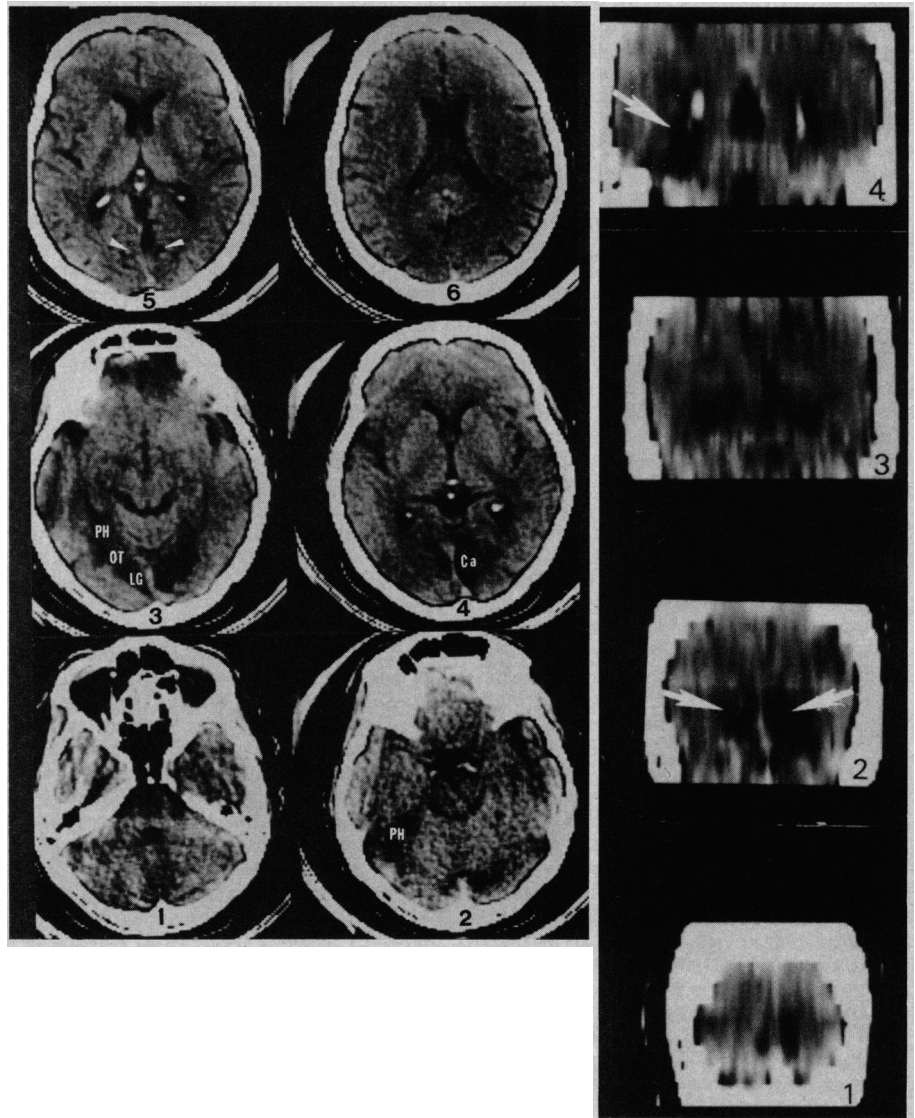


Fig 2.—Case 2. Top, Visual fields showing superior altitudinal macula-sparing monochromatism. Form perception was absent but motion was perceived normally. Test objects were 1-, 6-, and 15-mm red and white targets (tangent field). Identical field defects were shown by automated perimetry using 4-, 16-, and 64-sq mm targets at 1,000 apostilbs intensity. OS indicates left eye, and OD, right eye. Bottom left, Horizontal computerized tomogram (CT) showing infarcts of lingual (LG), occipitotemporal (OT), and parahippocampal (PH) gyri bilaterally, with some involvement of inferior lip of calcarine cortex (Ca) on right. Upper lips of Ca (arrowheads) appeared to be spared bilaterally. Levels scanned: 1-6, ventral to dorsal. Bottom right, Coronal reconstruction of same CT. Infarcts (arrows) were located inferolaterally to occipital horns and extended posteromedially to involve occipital poles (1 and 2). Left-sided lesion extended farther anteriorly into PH gyrus than that on right. Glomeruli of choroid plexus within occipital horns of ventricles is visible (4), as is third ventricle in midline (3 and 4). Sections scanned: 1-4, posterior to anterior.



(VER) testing (black and white checkerboard; full-field and separate altitudinal superior- and inferior-field stimulations) showed that cortex-dependent responses were present but so poorly developed that further interpretation was not possible.

The patient had amblyopia in the right eye, first identified in early childhood, and had worn corrective lenses ever since. Visual acuity at the time of this examination was 20/800 OD and 20/30 OS (unchanged from measurements taken during the previous 15 years). There were mild cataract formations bilaterally. Mental status was normal.

The CT showed bilateral, nearly symmetric infarctions of the occipital lobes in the territories of the parieto-occipital branches of the posterior cerebral arteries (Fig 1, bottom left). The narrow bands of the infarct involved parenchyma posterior and superior to the calcarine fissures but not inferior to them. Their most medial extent was to the midline; laterally, they extended to the area of the parieto-occipital sulci. The lesions were directly confined to the cunei from the level of the quadrigeminal cistern ventrally to the supraventricular level dorsally. Near the convexities, the lesions extended into the precuneate gyri bilaterally. The inferior calcarine lips appeared to be spared, but the upper calcarine cortices may have been involved, as it was impossible to identify them separately from the infarcted cunei. Sagittal reconstruction showed sparing of lingual gyri and the dorsal extent of the lesions into the precuneate regions bilaterally (Fig 1, bottom right).

CASE 2.—A 57-year-old man had episodes (lasting seconds to minutes) during which time he saw "white lights" moving from left to right without obscuring vision. These episodes had recurred numerous times for four years. Seven months prior to our examination, the frequency and intensity of the episodes increased, and the position of the lights changed so that they occupied the superior altitudinal portion of his visual field, where they took on "geometric" designs, such as "prisms" and "diamonds," still moving from left to right. The patient suddenly began to miss traffic signals while driving and could not see his hair or forehead when looking in a mirror. At this time, the geometric designs disappeared. He complained that visual objects viewed in the superior portions of the visual field appeared "whitish" and "spongy."

A variety of objects (eg, pictures, bills, coins, books, and clothing) presented in the patient's superior visual field could not be seen because they were obscured by a "washed-out, spongy" appearance. When the objects were moved, he could perceive the motion but was unable to identify the moving object, ie, its size, shape, texture, or color, until it was moved into the inferior field.

Vision in the inferior field (including color perception) was completely normal. The patient had no difficulty recognizing faces as long as he could use the inferior field, at least partially. When he looked at

the examiner's face (or his own in a mirror) and fixated the nose without moving his head to use the inferior field, the forehead appeared "spongy" and "washed out"; the hair could not be seen. The patient adjusted for this by slight extension of his neck, facilitating vision with the inferior field. Flashes of light and other stationary targets presented to his superior field induced searching movements and failure to arrive on target. However, moving targets (eg, wiggling fingers) presented in the superior field induced normal upward saccades that were on target. Upward pursuits were normal as long as the targets were initially fixated in the inferior field. Otherwise, eye movements were normal.

Visual field testing revealed complete, congruous, macula-sparing (2° to 3°) superior altitudinal homonymous hemianopia, where motion was perceived but form and color were not (Fig 2, top). Color perception (shown by a variety of objects tested in bright and dim illumination) was normal in the inferior field, as were Ishihara's design charts and pseudoisochromatic plate interpretations. Hue discrimination (similarity ordering of 12 variations of the four primary colors)⁶ was normal in the inferior field. The patient could name from recollection the colors of a variety of common objects (eg, lemon and fire engine). Reading was normal in the inferior field; so was writing. There was no dysnomia or other evidence of dysphasia. Mental status was normal. Visual acuity was 20/25 in the right eye, and 20/30 in the left eye, with corrective lenses. For five years following ictus, the patient reported being able to see some objects in the peripheral temporal portions of the superior fields, especially in bright light. However, serial visual field tests failed to show any change from the original tests.

The CT showed bilateral areas of infarction involving the lingual, occipitotemporal, and parahippocampal gyri (Fig 2, bottom left) in the territories of the posterior temporal branches of the posterior cerebral arteries. The infarctions appeared to destroy totally the lingual gyri bilaterally. The medial aspects of the occipitotemporal (fusiform) gyri were also involved symmetrically. The parahippocampal gyri were involved bilaterally, but asymmetrically, the left-sided infarction extended further anteriorly than that on the right. There was a wedge-shaped infarct of the striate cortex on the right side only. The hippocampal, cuneate, and lateral occipital gyri were spared. Preserved parenchyma of the medial and lateral occipital pole regions (Fig 2, bottom left, section 3) appeared to be composed of lateral occipital gyri and posterior caudal extents of cunei bilaterally. The only tissues that appeared to be preserved at the anteromedial aspects of the lingual infarct were those of the tentorium. Coronal reconstruction showed the ventrodorsal and anteroposterior extents of the lesions, from ventrolateral to occipital horns of the lateral ventricles to the occipital pole bilaterally (Fig 2, bottom right).

Although some authors have suggested that altitudinal hemianopia is usually a manifestation of a chiasmatic or prechiasmatic lesion,^{7,9} Bender¹⁰ found that this visual field defect is most commonly caused by bilateral occipital lesions, as seen in our patients. Still, the rarity with which occipital lesions produce such field defects is attested to by the fact that there were only five patients with altitudinal hemianopia in the combined series of Holmes and Lister,¹¹ Holmes,¹² Teuber et al,¹³ and Money and Nelson.¹⁴ All of these authors noted inferior altitudinal defects presumably caused by bilateral lesions above the calcarine fissure, though the location of the responsible lesions (upper posterior optic radiations) was confirmed by necropsy in only one case.¹² No superior-field altitudinal defects were encountered in patients with trauma, probably because the occipital parenchyma that would have had to be involved to produce that field defect (inferior to the calcarine fissure) lie adjacent to large venous structures (eg, the straight sinus) and penetrating wounds in that region would have been likely to cause fatal hemorrhage.

While the vascular supply of the occipital lobes has been described in detail¹⁵ and a variety of visual defects resulting from discrete occlusions have been reported, there have been, to our knowledge, only two patients with altitudinal hemianopia due to vascular lesions of the occipital lobes who have undergone autopsy.^{1,2} Both patients had complete altitudinal hemianopia of the inferior visual field, combined with some impairment of the superior field. Their defects were attributed to bilateral infarcts of the occipital parenchyma above the calcarine fissure, but neither case was pathologically "clean," as both had some involvement of the infracalcarine parenchyma as well.

The method of CT provides an excellent opportunity to correlate visual defects with occipital vascular lesions. The CT patterns of abnormalities that correspond to different branch occlusions of the posterior cerebral arteries are known.¹⁶ In addition, the scans are performed during life, when immediate correlations with visual defects can be performed and serial scans can be compared with changes in visual defects. However, to the best of our knowledge, there have only been two CT reports (of four cases of vascular lesions) correlating altitudinal hom-

onymous hemianopia with occipital lesions.^{17,18} The CT information provided in both reports was insufficient to make more than a general correlation between the occipital anatomy involved and the visual field defects produced. A patient with incongruous superior altitudinal hemianopia, central achromatopsia, and prosopagnosia had bilateral lesions of the "inferoposterior aspects of the cerebral cortex extending anteriorly into the temporal lobe and posteriorly to the occipital pole."¹⁷

Only one CT section (showing the ventral aspect of a left-sided lesion) was published; other sections were reported to show an infarct in similar inferior regions on the right. Judging from this limited information, the infarcts may have involved the lingual, occipitotemporal, parahippocampal, and, possibly, inferior temporal gyri, but this could not be determined with certainty. The anatomic correlations of three other patients with altitudinal homonymous hemianopia (one in the superior field, two in the inferior field) were limited to the levels of CT sections (relative to the pineal body) where the occipital lesions were best visualized. No other anatomic information on these patients was provided.¹⁸

Our patients' clinical-CT correlations clearly showed the representation of altitudinal visual fields in the occipital lobes above and below the calcarine fissures. In patient 1, the infarcts in the distribution of parieto-occipital branches of the posterior cerebral arteries produced discrete involvement of the cunei bilaterally. This finding confirmed results of previous necropsy studies that correlated inferior altitudinal hemianopia with bilateral lesions above the calcarine fissures. The macular-splitting nature of this patient's hemianopia suggested that the upper striate cortex (calcarine branches) was also involved bilaterally. However, this could not be shown with certainty because the upper parts of striate cortices could not be distinguished from the inferior aspects of infarcted cunei with currently available CT techniques. Vision was normal in patient 1's preserved homonymous superior field (return of premonitory acuity; light and color perception), except for defective form perception, which we attributed to the dorsal extension of her lesion into the parietal lobes. There was no CT evidence for involvement in the region of the inferior calcarine cortex or infracalcarine parenchyma.

Patient 2's infarcts in the distribu-

tion of the calcarine branch on the right and of posterior temporal branches of the posterior cerebral arteries bilaterally clearly showed that bilateral lesions inferior to calcarine fissures caused superior altitudinal hemianopia. The lesions appeared to destroy completely the lingual gyri bilaterally and involved large portions of the occipitotemporal and parahippocampal gyri bilaterally (on the left more than on the right). The congruous macular-sparing nature of the patient's hemianopia led us to postulate that the involvement of the right inferior striate cortex was incomplete, but confirmation of this postulate was beyond the resolution of CT.

The visuopsychic phenomena associated with the field defects of these patients deserve comment. Patient 1 experienced cortical blindness that persisted for seven days before resolving into a permanent altitudinal inferior-field defect. This defect has been accompanied by a behavioral abnormality best classified as visual disorientation,¹⁹ which also appeared to be permanent (present for four years since ictus). The errors in absolute and relative localization of objects in space; difficulty avoiding objects when walking and finding her way about; defective stereopsis (including teleopsia), sometimes with micropsia, other times with macropsia; and trouble reading are components of visual disorientation with metamorphopsia that might be expected, in view of the dorsal extension of her lesions into the precuneus regions. The phenomenon of visual disorientation has classically been associated with such bilateral parietal lesions.¹⁹

Patient 2 is noteworthy because of the absence of permanent visuopsychic abnormalities, despite lesions that should have produced two such deficits. He experienced occipital irritative phenomena in the form of moving photisms, which ceased when his superior-field defect appeared. No other visuopsychic disorder has developed during five years of follow-up since ictus. The patient has retained normal color vision in the inferior visual field, and his ability to recognize faces has been unaffected, despite the presence of bilateral lesions that characteristically produce central achromatopsia and prosopagnosia.

There is a body of evidence, including four autopsy cases,²⁰⁻²³ and CT analysis of two cases of central achromatopsia,³ that indicates that the total cortical representation of color perception from one homonymous

visual field is contained in the contralateral lingual and occipitotemporal gyri (analogous to area V4 of the macaque).^{24,25} A review of available literature indicated that when lingual and occipitotemporal lesions are bilateral, prosopagnosia can result (usually accompanied by achromatopsia).⁴ The ten autopsies and three CT-examined patients with prosopagnosia had bilateral lesions of these mesial occipitotemporal regions (sometimes extending into the parahippocampal and inferior temporal gyri).⁴

Why our patient 2 retained normal color vision and facial recognition abilities is unknown. The lingual gyri appeared to be completely destroyed bilaterally. Both occipitotemporal and parahippocampal gyri were also extensively involved, but the lesions were asymmetric, with that on the left extending farther anteriorly than that on the right. Possibly, the preserved anterior portion of the right parahippocampal gyrus was critical for retention of color vision, despite the presence of bilateral lesions. The relative superiority of the right over the left hemisphere in the process of facial recognition has recently been stressed in a study of patients with a surgically split corpus callosum.²⁶ Most of the preserved parenchyma at the occipital poles appeared to be those of lateral occipital gyri. Conceivably, those gyri, combined with the preserved right parahippocampal gyrus, were sufficient for retention of color vision and facial recognition abilities. However, the lateral occipital gyri have not been implicated previously as important structures for these abilities.

Previous reports of normal VERs in blind persons attributed the preserved potentials to intact striate cortices when association areas were destroyed,²⁷ or to preserved association areas when striate cortices were destroyed.²⁸ It is possible that both the striate cortex and association areas must be involved before occipital lesions will cause a loss of VER. Thus, the VER findings in our patients (case 1, nearly absent; case 2, normal) might be explained by involvement of the striate cortex and visual association areas in patient 1, but only visual association areas in patient 2. The nearly absent VER of patient 1 might also have been related to her limited ability to recognize and be attentive to the checkerboard pattern on the screen, because of visual disorientation.

The nearly absent flight of colors in case 1 may have been due to bilateral

supracalcarine and infracalcarine involvement caused by the patient's initial hypoxic episode, although only the supracalcarine lesions were visible by CT. The normal flight of colors in

case 2 showed the intactness of cerebral structures that generate colorful afterimages.⁵ Their absence was expected in this patient's hemianopic superior field, where all perception

except that of motion had been lost.

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