# Crossed-Quadrant Homonymous Hemianopsia

## The "Checkerboard" Field Defect

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### Abstract

A 70-year-old man with a history of hypertension and coronary artery disease suffered an abrupt loss of vision in June 1980. Neuro-ophthalmologic examination in August 1981 revealed 20/20+ acuity in both eyes, but quantitative perimetry disclosed a classic crossed-quadrant homonymous hemianopsia. This is known as the "checkerboard" visual field defect; a right upper quadrantanopsia as well as a left lower quadrantanopsia. A review of the eight previously reported cases is presented. A trial with "checkerboard" Fresnel prisms gave only a slight improvement in ambient field in this patient. The significance of that point is discussed. To our knowledge, this is the first patient with a "checkerboard" occipital lobe infarction pattern documented by computed tomography.

Crossed-quadrant homonymous hemianopsia, the "checkerboard" visual field defect, is the homonymous loss of two diametrically opposed quadrants of the visual field, is a rare occurrence. We report the case of a patient with this field defect documented by crossed-quadrantic computed tomographic findings, and describe eight cases from the literature in which similar fields are recorded. The clinical features and anatomico–pathological correlations of crossed-quadrant homonymous hemianopsia are discussed.

## Case Report

A 70-year-old, left-handed man presented to the Bascom Palmer Eye Institute on August 27, 1981, because of difficulty with his vision. Several years earlier he suffered occasional transient loss of vision in the right visual fields accompanied by flashing lights, but had not reported these to a physician. In June 1980, he suffered several epi-

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sodes of scintillations, sometimes synchronous with his pulse, covering the entire field of vision in both eyes. One morning while shaving, he was suddenly unable to see, experienced flashing lights to the right, and had a severe parieto–occipital headache. These complaints prompted admission to a hospital in Wilmington, Delaware. The patient had a 20-year history of hypertension, reportedly well controlled, and he had a myocardial infarction in 1978.

Physical examination recorded only a right homonymous hemianopsia with some left field loss in addition. A CT scan done at that time reportedly showed a left occipital infarction. An EEG showed decreased alpha activity on the left. The diagnosis of a posterior circulatory ischemic was made, and the patient was treated with ascriptin and persantine. He appeared to recover uneventfully and at the time of discharge from the hospital on June 14, 1980, a right homonymous hemianopsia, denser above, remained.

Neuro-ophthalmological consultation was performed on August 27, 1981. The patient considered that his visual function had remained stable since the 1980 hospitalization, but noted that he frequently bumped into objects up and right and down and left. He also complained of difficulty with his memory. By this he meant that he could no longer give directions while his wife was driving, as streets in his neighborhood always looked unfamiliar. He was vexed by the inability to recall names of friends and acquaintances, but denied difficulty with recall of past events, calculations, right-left discrimination, reading, writing, or balancing. There was no diplopia, dysarthria, weakness, numbness, or dizziness. There had been no scintillations or other visual hallucinations since 1980.

Examination revealed a corrected visual acuity of 20/20+4 and J-1 in the right eye and 20/15−1 and J-1 in the left eye. The blood pressure was 140/80. Visual field testing (Figs. 1–3, *a* and *b*) showed a crossed-quadrant homonymous hemianopsia, with the right upper defect extending
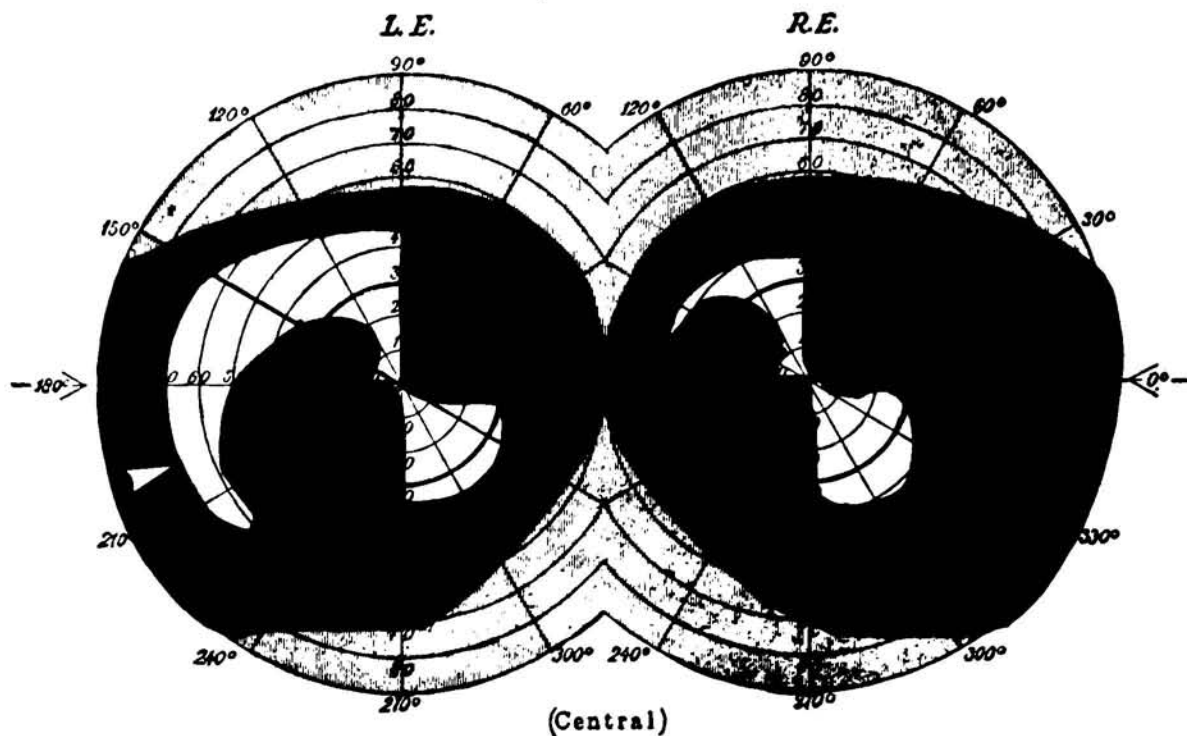
# VISUAL FIELDS
## (Peripheral)



**Figure 1.** Peripheral fields to 10/330 white on August 2b, 1981 (Aimark perimeter). Note intact monocular lower temporal crescent in left eye (white arrowhead).
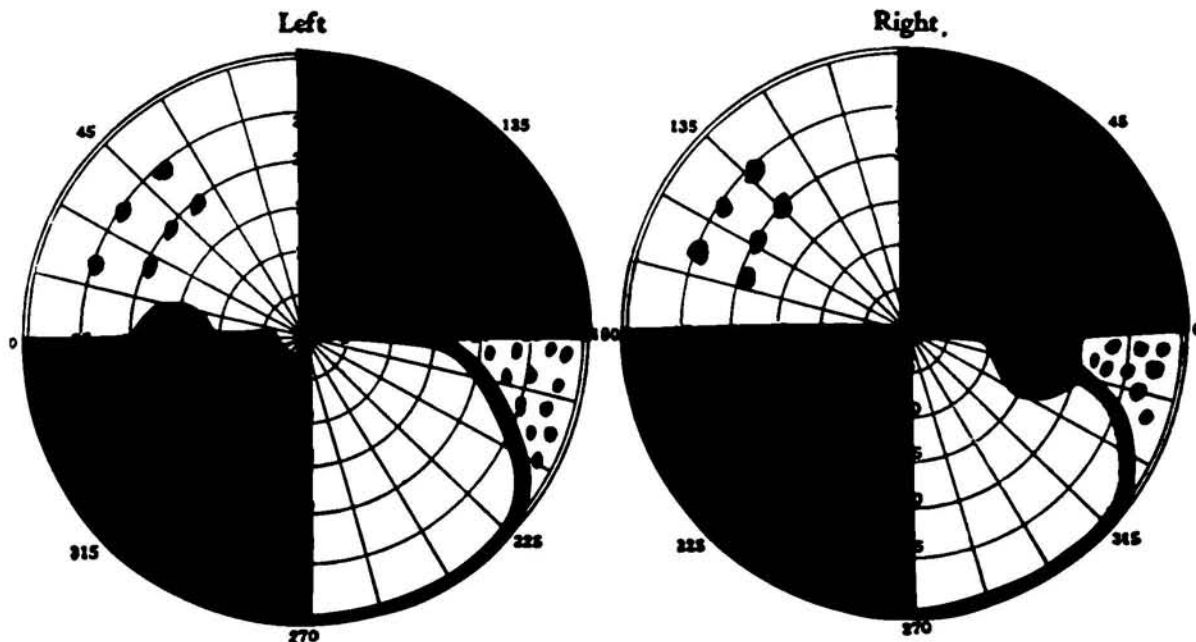


**Figure 2.** Central visual fields to 70/1000 white projection light on August 27, 1981. Note extreme congruity in central fields with the "checkerboard" defect (right upper quadrantanopsia and left lower quadrantanopsia). Note that extension into right lower quadrant was denser than the more subtle extension into left upper quadrant.
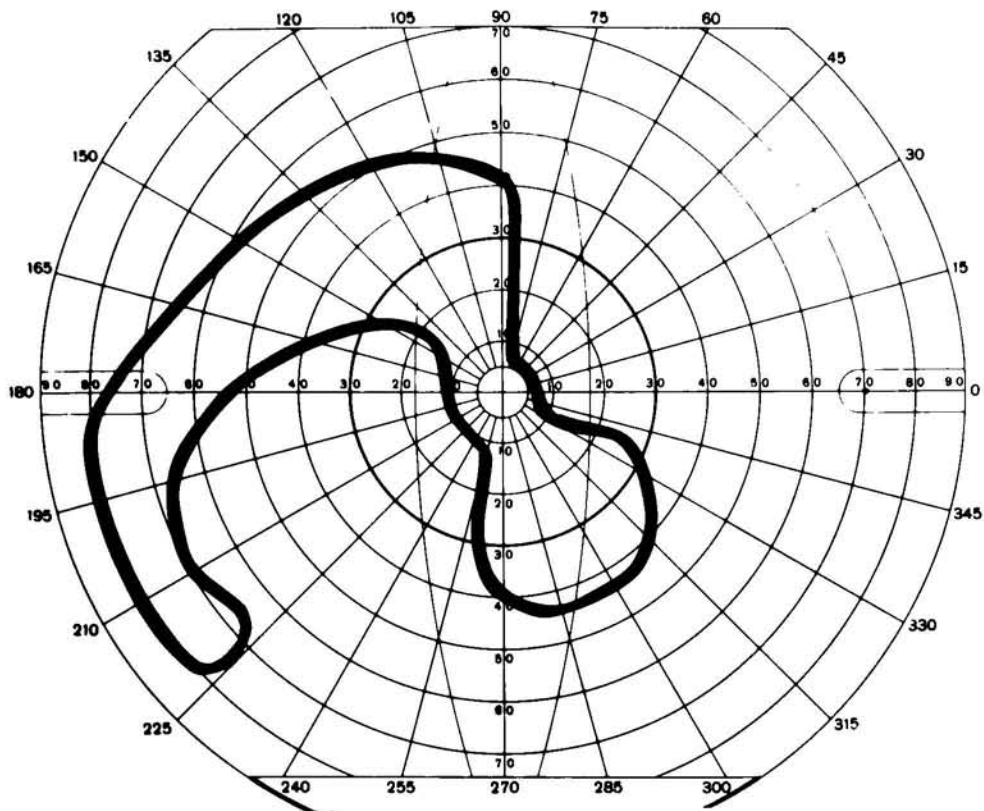
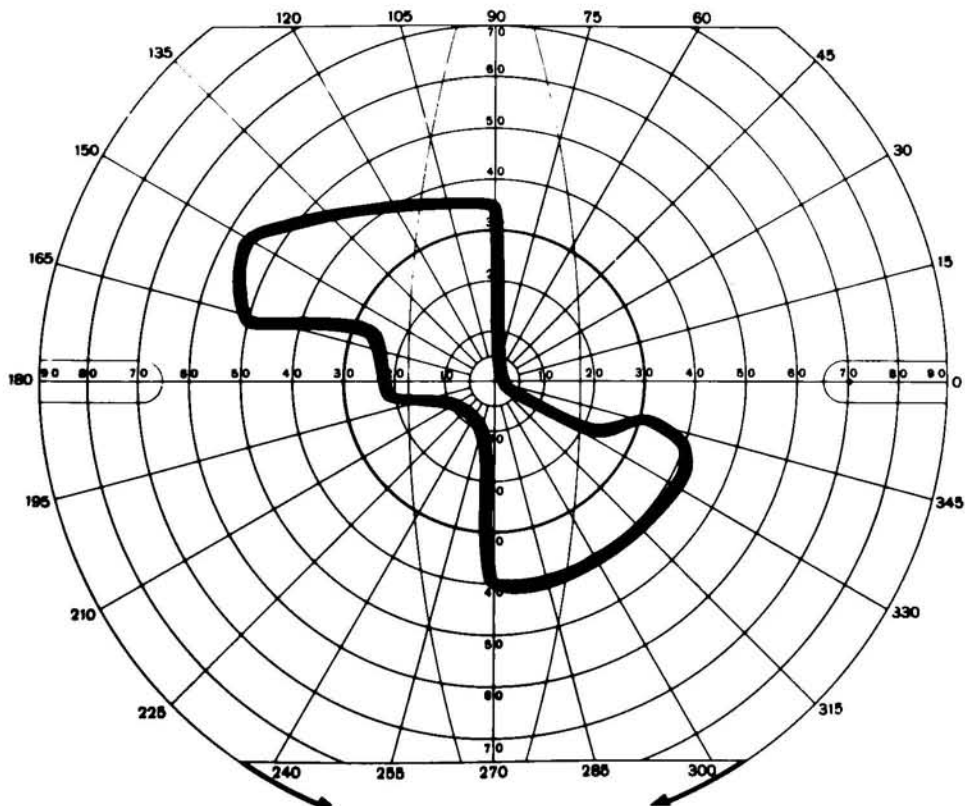**Figure 3a.** Goldmann visual field on left eye on July 6, 1981.



**Figure 3b.** Goldmann visual field on right eye on July 6, 1981. Six isopters were plotted in each eye. The largest isopter (V4e) was drawn here, since III4e and II4e were all essentially the same, showing that the field defect had absolute, and not sloping margins. This was consistent with old infarction. These fields were done by perimetric technicians and show "pseudoincongruity" at first glance. This is explained by the intact lower temporal crescent in left eye, and Riddoch phenomenon accounts for some differences in isopter with varying speeds of testing kinetic fields.

slightly below the horizontal meridian, but respecting the vertical meridian. Similarly, the left lower defect extended above the horizontal meridian, but again respected the vertical midline. An isthmus of partially intact central field connected the two intact quadrants. Fixation appeared to be split up and to the right, but in the inferior half of the field the presence of splitting versus sparing was difficult to determine. There was a subtle arcuate defect in the left upper quadrant. The monocular temporal crescent in the left eye was spared. On Amsler grid testing the patient reported a "missing" right upper quadrant in each eye. He did not report any abnormality in his left field. Optokinetic responses were 3+ and symmetrical with targets taken right, left, up, and down. Muscle balance, motility, external, slit lamp, and ophthalmoscopic examinations were within normal limits.

Neurological examination showed that the patient was alert and oriented. Immediate, recent, and remote memory were intact. Speech was fluent and without errors except for momentary hesitations and occasional inability to provide a noun in difficult naming tasks, such as naming the parts of a watch. The patient provided the names of political figures pictured in a magazine. Color naming, calculations, and right-left distinctions were well performed. Writing and reading were abnormal only for hesitation in the use of proper and common names. The patient's constructions included a floor plan of his house in which two rooms were reversed in position. A drawing of a clock and a map of Florida were well executed. The patient had claimed difficulty in recognizing familiar places, but this could not be demonstrated using photographs. Cranial nerves, motor, sensory, and cerebellar testing were normal. In particular, the patient did not extinguish on double simultaneous stimulation, and graphesthesia and stereognosis were intact.

A computed tomographic scan with axial cuts and specific coronal studies of the occipital lobes (Figs. 4–6) was performed on October 14, 1981. There was a small lucency high in the right calcarine cortex, and a larger lucency was seen low in the calcarine region and adjacent lingual gyrus on the left. Neither of these lesions enhanced. The findings were consistent with old infarctions.

Because the patient complained that he encountered unseen objects up and right, and down and left, we fitted his glasses with "checkerboard" Fresnel prisms (Figs. 7 and 8). A 30-diopter base out Fresnel prism was placed in the right upper quadrant of the right lens, and another 30-diopter base out prism was placed in the left lower quadrant of the left lens. Both prisms were trimmed so that they did not encroach upon fixation. The patient wore these prisms glasses for several months. He was not impressed with his ability to ambulate with them. Tangent screen testing, however, done
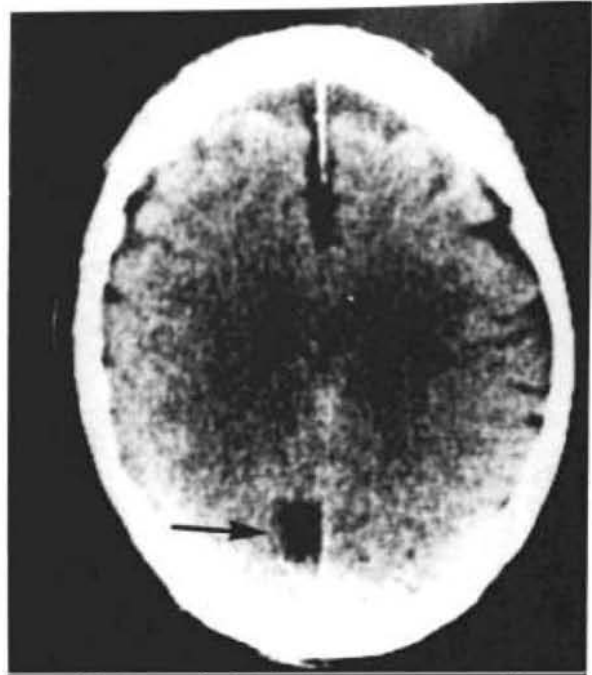


Figure 4. Axial CT scan made October 14, 1981. Note hypodense lesion high in right calcarine cortex (black arrow). This accounts for the left lower homonymous visual field defect in our patient.



Figure 5. Axial CT scan made October 14, 1981. On this cut, which is lower than that seen in Fig. 4, a larger hypodensity is seen in left occipital lobe (arrowheads). This accounts for the right upper homonymous visual field defect in our patient.
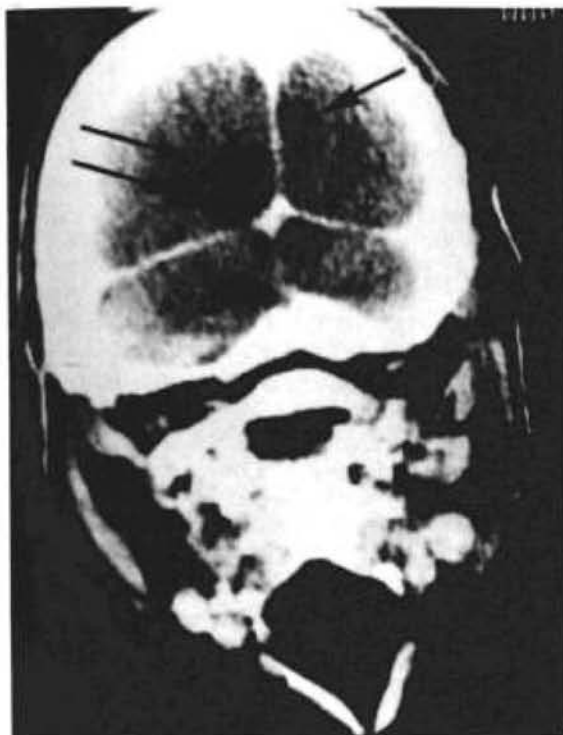
**Figure 6.** Coronal CT scan made October 14, 1981. The right upper calcarine infarction (single arrow) and the larger lower left calcarine infarction (double arrows), show the "checkerboard" anatomical correlate to the visual field defects.



**Figure 7.** Patient wearing "checkerboard" Fresnel prism glasses.



**Figure 8.** "Checkerboard" Fresnel prism glasses, closer view. Note patient is facing the camera and holding the glasses as he wears them.

without the glasses, and with the prism glasses on, showed that the field in the blind quadrants was increased by 10–15 degrees from the vertical meridian when wearing the glasses. It was thought that the intact monocular temporal crescent in the left eye and the presence of the Riddoch phenomenon in this patient probably accounted for the fact that he did not appreciate a more dramatic improvement from the Fresnel prisms.

**Comment.** This 70-year-old man with a history of hypertension and atherosclerosis may have suffered loss of some of the right visual field several years before his presentation, as simple hallucinations and intermittent visual loss had occurred in that field. However, catastrophic visual loss occurred at the time of the 1980 hospitalization, when he suffered an occipital headache and more hallucinations. The right visual field was significantly affected, a new anomia was present, and the left field loss along with right parietal deficits probably also dated from that hospitalization.

Several characteristics of the patient's visual field are of interest. Only the right field loss was specifically noted by the patient. The missing quadrants respected the vertical meridian, but encroached upon areas on the opposite sides of their respective horizontal meridians. An isthmus of relatively intact field connected the two intact quadrants. The "intact" areas of field showed subtle deficits. The monocular temporal crescent in the left eye was spared. Normal optokinetic responses favored an occipital location for the lesions.

## Literature Review

A search of the literature revealed that only eight cases of crossed-quadrant homonymous hemianopsia have been reported. Many of these cases suffer from inadequate clinical descriptions and lack of pathologic correlation. However, some of the reports contain clinical observations which are relevant to the problems we will discuss, and are recorded here in as much detail as was available.

### Case 1

In 1891, Groenouw[1] reported the case of a patient who had suffered a left hemiplegia and a left homonymous hemianopsia. Ten months later, he had a second apoplectic attack from which a crossed-quadrant hemianopsia was discovered. The field cut was not absolute.

**Comment.** If this patient's initial deficits are to be explained by a single lesion, the most likely location would be in the region of the right internal capsule and optic radiation.

### Case 2

Weymann's patient[2] had syphilis and a violent continuous headache. After an abrupt collapse with loss of consciousness, a right hemiplegia and a crossed-quadrant hemianopsia were found. The scomata were not absolute, and they did not change after antiluetic therapy.

**Comment.** In this case, the quadrantanopsias seem to have had simultaneous onset. Luetic vascular disease was their cause. Proximal posterior cerebral artery occlusion is likely, but deep parietal involvement, possibly in the middle cerebral artery, is also possible.

### Case 3

Felix[3] reported the case of a 63-year-old man who had atherosclerosis and cardiac disease. He awoke one morning completely blind, but unconcerned about his plight. Over several days his vision improved so that he could see large objects "as in a mist." Four months after the initial event, he complained that he could see small photographs more easily than large ones, and that reading was impossible. Examination of the visual fields revealed a crossed-quadrant hemianopsia. "The left upper scotomata take not only the left upper quadrants but also rather more than one-third part of the next lower quadrants (left lower quadrants). The right lower scotomata are somewhat larger than the quadrants there (extending into the right upper quadrants). The quadrant scotomata show a sparing of the macula and are absolute. The left lower quadrants of the visual fields are color blind. In the centre and in the right upper quadrants, which show a peripheral restriction, colours are recognized till about 15°—outside this there is colour blindness."

**Comment.** This patient had total occipital blindness which, upon recovery, resolved into a crossed-quadrant hemianopsia. The seeing fields were abnormal. First, the quadrantic defects extended into them. Second, perception of colors was abnormal within them. There was some sparing of central vision.

### Case 4

In 1896, Allyn[4] recorded the case of a 49-year-old man with bilateral homonymous hemianopsia. The patient suffered two episodes of loss of vision, each one resulting in a homonymous field defect. The first occurred while he was standing at his desk. The sight of both eyes left him and he remarked to a friend that he was blind. After a few minutes a slow and incomplete recovery commenced. Three weeks later, on formal field testing, a left homonymous hemianopsia was discovered. There was sparing of 5° around fixation. No other neurological deficits were noted.

Six months later the patient suddenly fell to the floor and remained unconscious for 2 hours. Within 36 hours, the vision in both eyes began to

fail rapidly and the patient was unable to see faces or find his way about a room. For a few hours, his speech was "thick." His wife thought that his hearing had been affected.

One week after his collapse, the visual acuity was hand motions. The fundi were normal. The patient complained of visual hallucinations. For example, he thought that his wife's nose was painted red. Another time, the patient thought that burglars were breaking into the house. In the subsequent months, his vision improved steadily. Serial visual field testing, hampered by the patient's inability to see the fixation target, showed a slowly improving crossed-quadrant hemianopsia. One year after the illness, central vision had significantly recovered, and crossed-quadrant hemianopsia remained.

**Comment.** The patient's first illness resulted in bilateral occipital blindness which resolved into a left homonymous hemianopsia. The second episode is suggestive of a basilar embolus which caused transient brain stem ischemia and then occipital cortex infarction. The hallucinations imply either posterior parietal ischemia or posterior limbic ischemia with peduncular hallucinosis.

### Case 5

Traquair[5] illustrated field defects found 10 weeks after an attack of necrotizing ependymomyelitis. Damage to the walls of the lateral ventricles had been ascertained by ventriculography. Impairment of memory was present, but dysphasia and other signs of cortical involvement were absent. On the right side, the upper quadrant and the superior part of the lower quadrant were affected. On the left side, the lower quadrant was affected. An isthmus of intact field connected the intact quadrants. In addition, there was general depression of the fields. The optic discs appeared normal. The vision in both eyes was 20/20.

**Comment.** This case, documented by ventriculography, again shows the development of a crossed-quadrant homonymous hemianopsia secondary to deep lesions in the radiations. The defects extended across the horizontal meridian, and central field was preserved.

### Case 6

Walsh and Hoyt[6] showed the field of a 70-year-old woman with basilar artery disease. Quadrantic defects were found in the right upper field and the left lower field with a narrow congruous isthmus near fixation. The left temporal crescent was spared and there were incongruities along the upper vertical meridian and the right horizontal meridian.

**Comment.** This case suggests that incongruity may occur in occipital defects. This question will be addressed in detail later. Central vision was relatively well preserved.

### Case 7

Janssens and Berthelon[7] reported two cases. The first was that of a 65-year-old woman who was admitted to hospital for what appeared to be total blindness and amnesia. She had a history of mitral valve disease and a congenital hemolytic anemia. The vision was 20/25 in both eyes. Confrontation fields disclosed a left homonymous hemianopsia and on the following day, a right hemianopsia. Two days after admission, ophthalmological examination revealed a left homonymous hemianopsia which was "very incongruent." Fifteen days later, a congruent homonymous crossed-quadrantic hemianopsia was discovered. A final visual field, performed $1\frac{1}{2}$ months after the initial examination demonstrated essentially full recovery of the inferior quadrant defect. Neurological examination was normal.

**Comment.** This case again raises the question of incongruity in the visual field in these lesions. A tenable explanation for the apparent abrupt change in the side of the hemianopsia is that both hemifields were initially involved and the Riddoch phenomenon was notably present.

### Case 8

The second of Janssens' and Berthelon's cases[7] was that of a 51-year-old man who complained of a black spot in his right visual field. A few days later he noted a defect in the right visual field accompanied by the sensation of light and paresthesias of the hands and the right side of the mouth. Three weeks later, when first examined by an ophthalmologist, the patient had a visual acuity of 20/200 on the right and 20/40 on the left. There was a congruous partial bilateral homonymous hemianopsia involving the right hemifield, particularly superiorly, and the left upper quadrant. Neurological examination disclosed a left facial weakness and right hemihypesthesia involving the trunk as well as the limbs. Stereognosis was diminished in the left hand. Examination 1 week later revealed an acuity of 20/20 in both eyes. The field defect persisted.

Arteriography of both carotid arteries and the right vertebral artery showed multiple thromboses in the terminal segments of the right sylvian branches, a probable thrombosis in the terminal segment of the right posterior cerebral artery, and a new thrombosis of the middle third of the left posterior cerebral artery.

**Comment.** This patient was reported to have had numerous thrombotic central nervous system lesions. Their distal locations suggest that they might actually have been embolic in origin.

## Discussion

Crossed-quadrant homonymous hemianopsia is a rare occurrence. In our survey of the literature,

including discussions focusing on occipital visual loss of all types, there were only eight reported cases. Our case is the ninth. Although clinical descriptions are incomplete, the data available do indicate some important features of the lesions and resulting visual field deficits and associated abnormalities. One case was studied by ventriculography (case 5) and one by arteriography (case 8). Our case was investigated by CT scanning. No autopsy material is available.

The visual fields in cases of crossed-quadrantic defects have several interesting characteristics:

1) The onset of the defects may be in two simultaneous quadrantanopsias, two successive homonymous hemianopsias each resolving into a quadrantanopsia, or simultaneous bilateral homonymous hemianopsias resolving into crossed-quadrantic defects.
2) The quadrantic defects usually extend across the horizontal meridian while the vertical meridian is always respected. This meridian may appear to be violated when there are defects in the seeing fields. In order to demonstrate its integrity, its entire extent must sometimes be explored in detail.
3) Central vision is relatively well preserved, with an isthmus of intact field connecting the two intact quadrants.
4) The monocular temporal crescent may be spared.
5) The "intact" quadrants are not necessarily normal. Small targets may not be detected in them. The Riddoch phenomenon may be present. Color vision may be abnormal. The defective areas of field may encroach upon the intact quadrants. Simple or complex hallucinations may appear in either the blind or in the seeing fields.

The question of incongruity in homonymous crossed-quadrantic field loss was raised by case 6 and case 7. Incongruity may be either apparent or true. There are two important factors that must be considered in occipital lobe field defects that can cause an apparent incongruity (or "pseudo-incongruity") in the field. These are the Riddoch phenomenon and the presence of the monocular temporal crescent. The first of these was reported in 1917 by Riddoch,[8] who described dissociation of visual perceptions in patients with occipital injuries. The ten patients studied fell into three groups. Those in the first group had homonymous hemianopsias with preservation of the ability to detect moving—but not static—targets in the blind field. Some had preservation of the monocular temporal crescent (to be discussed later), and in some of these movement could be detected in the crescent only, and not more centrally. In some cases, an object could actually be identified when seen in the crescent, but the identity would then be lost until it appeared in the intact hemifield. The second group of patients had homonymous hemianopsias which showed recovery. Field loss for static targets was always greater in extent than field loss for moving targets. The ability to detect motion in an area of field preceded the ability to

see static test objects. The third group of patients had homonymous field defects in which there was no dissociation between detection of moving and static targets. Benton et al.[9] have emphasized that preservation of the monocular temporal crescent often correlates with striking sparing of the ability to perceive moving stimuli. Riddoch's observations argue for the importance of testing patients with both static and kinetic targets when mapping the visual fields resulting from occipital lesions. It also provides one explanation for apparent incongruity, if a different velocity is used when testing isopters in the different eyes.

A second reason for apparent incongruity in the fields lies in the existence of the monocular temporal crescent itself. The crescent is that part of the visual field which represents unpaired peripheral nasal fibers. It is in the extreme temporal periphery, outside the field of binocular representation. When it is spared, the fields simply look incongruous. The unwary observer may map the temporal side on the perimeter as being full when only the crescent is present, or when the field central to the crescent is defective. Conversely, when only the central 30° of field is examined, as on the tangent screen, the presence of an intact crescent may be overlooked.

In some cases of crossed-quadrantic homonymous hemianopsias, the fields may be truly, not just apparently, incongruous. The associated neurological deficits of memory loss, right and left parietal defects such as anomia and spatial disorientation, motor weakness, and thalamic type sensory changes indicate that the lesions extend forward into the parieto–occipital white matter and the posterior perforated substance. A few of the lesions, in fact, may lie in the distal territory of the middle cerebral artery or the anterior perforators. The incongruity of the fields in these few cases may be accounted for by anterior optic radiation involvement. However, it should be emphasized that the crossed-quadrant hemianopsia is nearly always an occipital lobe sign.

The origin of bilateral hemianopsias, of which the crossed-quadrant homonymous hemianopsias are a subset, and the determination of whether the deficits are simultaneous or successive, has been addressed at length by Symonds and MacKenzie.[10] Material derived from 58 cases (29 with postmortem) is discussed by these authors. In their cases, the loss of vision was either sudden or gradual and both half-fields could be affected simultaneously or in succession. Simultaneous loss was more common. Frequent prodromal symptoms were attacks of vertigo and transient episodes of visual impairment.

The pattern of field loss was often such that the central 10° was preserved. In the more peripheral parts of the field the sectors adjacent to the vertical meridians were not infrequently spared. Selective

central visual loss was rare, but it did occur. Disorders of higher function including spatial disorientation, visual agnosia, and denial of blindness; visual hallucinations were inconstantly present, but sometimes conspicuous.

In cases in which the visual loss in the two half-fields was simultaneous, the most frequent case was embolization of the calcarine arteries, with the source of the emboli arising in the basilar or vertebral arteries (a frequent finding) or thrombi associated with valvular heart disease, atrial fibrillation, or myocardial infarction.

Although there is no case of crossed-quadrantanopsia with postmortem material, the cases recorded here, except for Traquair's case, appear to belong to an infrequently occurring subset of bilateral homonymous hemianopsia occurring with either thrombotic or embolic disease. Only Traquair's case was the result of an ependymomeningitis with damage to the optic radiations around the posterior horns of the lateral ventricles. A few cases, especially those in which there was a hemiparesis, may have had vascular lesions possibly in the middle cerebral artery territory.

Several radiologic studies have been undertaken to identify the relationship between visual field defects and arterial occlusion in the posterior circulation.[11, 12] Kaul et al.[11] compared the extent and severity of visual field loss in 14 patients, the occlusions of whose posterior circulations were verified angiographically. Atheroma, embolism, and migraine were the most common types of underlying disease. Occlusion of the main trunk of the posterior cerebral artery was associated with severe and permanent visual loss often with central sparing due to collateral blood supply from the middle cerebral artery territory. Single or multiple occlusions of main branches of the posterior cerebral artery caused variable amounts of field loss sometimes with considerable recovery. Occlusions of smaller branches of the calcarine artery caused loss of central vision which was usually severe.

Hoyt and Newton[12] recorded the angiographic changes seen in the occlusion of arteries that supply the visual cortex. Atheromatous emboli, tumor emboli from atrial myxoma, posterior cerebral artery aneurysm, focal vasculitis, and spasm after subarachnoid hemorrhage were the most common causes of abnormal findings. Most proximal occlusions of the posterior cerebral artery were embolic. More distal occlusions of the posterior cerebral artery were related to uncal herniation with compression of the artery as it passes over the tentorial edge. True narrowings of the artery occurred with subarachnoid hemorrhage, occipital contusions, migraine, and arteritis. Cortical branch occlusions were also usually the result of emboli, but cortical complications of migraine and polyarteritis also caused such occlusions. Angiographic findings were sometimes normal, even in the presence of a field defect, and they were occasionally abnormal with normal visual fields. The requirement for significant infarction seemed to be multiple emboli.

CT scan findings have been correlated with visual field defects in vascular lesions of the posterior visual pathways. McAuley and Russell[13] studied 39 patients with various types of homonymous hemianopsias. Most cases had low-density lesions within the distribution of the posterior cerebral artery. Most of the lesions were situated posteriorly and involved visual cortex or posterior radiations. In two patients the lesions spared this posterior area and involved only the anterior radiations. In both of these cases the defects were quadrantic only, and may have been within middle cerebral artery territory. In general, lesions giving rise to quadrantic defects were smaller than those causing total hemianopsias. Lower quadrantic defects tended to occur with superior CT lesions, and vice versa. Macular sparing was associated with survival of the occipital pole in some instances. Bilateral cases had a higher prevalence of associated neurological defects. Our case had crossed-quadrantic CT findings consistent with the patient's visual field loss and in agreement with the above observations.

Visual rehabilitation of patients with crossed-quadrant homonymous hemianopsias depends upon the extent of the deficits. One important factor is the ability to detect the presence of objects in the peripheral field. The advantage of preservation of the temporal crescent, and with it the ability to detect movement, has recently been documented in detail.[9, 14] Some patients, however, are seriously disturbed by the perception of movement in the blind field.[8]

One means of improving peripheral vision is by the use of mirrors or prisms. There are several reports in the literature[15-17] describing the use of mirrors attached to patients' spectacles and fixed at such an angle that objects in the blind half of the visual field are reflected by the mirror so as to be imaged on seeing retina. There are also reports of the use of prisms to increase functional peripheral vision.[18, 19] We have employed paste-on Fresnel prisms in more recent years, and have found them more practical in practice than hemianopic mirrors. A 30-diopter Fresnel prism is placed with the base towards the hemianopic side of the patient's glasses. The prisms are trimmed so that they do not interfere with fixation. In a patient with a total left homonymous hemianopsia, a 30-diopter Fresnel prism is placed base out (base left) on the temporal half of the patient's left spectacle lens. An example is seen in Figure 9.

Our patient was inconvenienced in ambulation by a tendency to bump into objects up and to the right, and down and to the left. We fitted him with Fresnel prisms in a "checkerboard" pattern. Only

**Figure 9.** Conventional hemianopic Fresnel prism as used in a patient with a total left homonymous hemianopia. Patient is holding glasses and the 30-diopter Fresnel prism is base left on left lens.

modest visual improvement was noted in our patient with these prisms, probably because of the integrity of the monocular temporal crescent in the left eye and also because of the Riddoch phenomenon. To our knowledge, this is the first case in which a "checkerboard" field defect has been documented by "checkerboard" CT findings, and in which "checkerboard" Fresnel prisms have been prescribed.

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