

# The Missing Temporal Crescent

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- **PURPOSE:** We studied clinically the representation of the monocular temporal crescent in the human visual cortex and noted the importance of using the perimetric techniques best suited to detect this visual field defect and to study patients in whom the temporal crescent is missing.
- **METHODS:** Goldmann perimetry and high-resolution magnetic resonance imaging were performed in two patients with vascular lesions located in the anterior striate cortex.
- **RESULTS:** A monocular visual field defect, the missing temporal crescent, was found on the side contralateral to the lesion.
- **CONCLUSIONS:** The perimetric-magnetic resonance imaging correlation is in exquisite agreement with recent information about the representation of the visual field in the human primary visual cortex. Reports of this specific perimetric finding are rare, in part because of underdetection with currently used perimetric techniques that concentrate on the central 30 degrees of the visual field.

**R**ETROCHIASMAL LESIONS OF THE VISUAL PATHWAYS affect the visual fields in a homonymous fashion. The only exception to this rule is the missing temporal crescent of one eye, for which no corresponding homonymous visual field defect can occur in the fellow eye. The nasal visual field extends to roughly 60 degrees on the horizontal meridian, as opposed to a 100-degree extension of the temporal

visual field. This well-known phenomenon of a monocular visual field defect caused by a retrochiasmal lesion is described theoretically in countless textbooks, but actual case studies are exceedingly rare. The last series was reported by Bender and Strauss<sup>1</sup> in 1937, and a single case was published by Walsh<sup>2</sup> in 1974. We studied two patients with purely monocular visual field defects involving the temporal crescent of one eye. Magnetic resonance imaging showed vascular lesions in the contralateral visual cortex.

## CASE REPORTS

• **CASE 1:** A 47-year-old woman had a ten-year history of migraine with visual auras occurring repeatedly in her right visual field. Over the years the frequency of the attacks increased. Computed tomography disclosed an arteriovenous malformation in the left parieto-occipital lobe, and the patient was referred for treatment. In May 1992 an embolization was performed, and the arteriovenous malformation was partially occluded. In November 1992 the patient was referred for neuro-ophthalmic examination. She gave a history of zigzag scintillations occurring in her right visual field, which were followed by headaches. Between such attacks there were no visual symptoms.

The patient's uncorrected visual acuity was 20/20 in each eye. Her pupils were equal and showed no afferent or efferent defect. Results of slit-lamp examination were normal. Goldmann perimetry of the right eye showed a peripheral defect, more pronounced in the upper than in the lower temporal quadrant, extending between 60 and 90 degrees along the horizontal meridian (Fig. 1). The visual field of the left eye was full, extending to 90 degrees temporally and to 60 degrees nasally. A dilated fundus examination was performed in both eyes, with special attention to the nasal retinal periphery of the right eye. No

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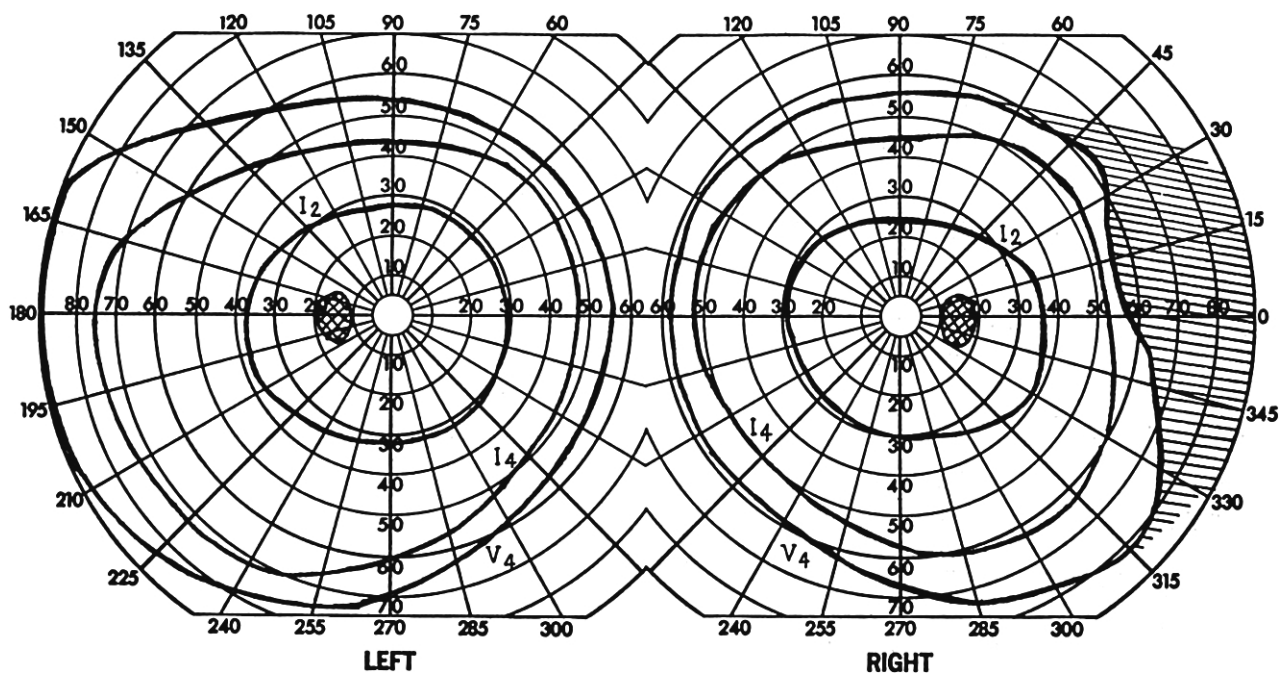


Fig. 1 (Landau, Wichmann, and Valavanis). Case 1. Right temporal peripheral defect, more pronounced in the upper than in the lower quadrant. No homonymous defect on the left side.

abnormality was found. Magnetic resonance imaging showed a large left temporo-occipital arteriovenous malformation (Fig. 2).

• **CASE 2:** A 27-year-old woman was seen at the Department of Ophthalmology for an emergency examination on May 28, 1993. Her medical history was remarkable for migraine headaches with visual aura since the age of 15 years. She was not taking any medication except for oral contraceptives. Two days before the examination the patient had an unusually severe attack of headache accompanied by nausea and photophobia. She had to lie down with closed eyes for several hours. Upon arising she immediately noted that the most peripheral part of the visual field on her right was missing. When this field defect persisted for the next two days, the patient consulted her general practitioner and was referred for a neuro-ophthalmologic examination.

Visual acuity was 20/15 in each eye, with slight astigmatic correction. Pupils were equal and showed no afferent or efferent defect. Slit-lamp examination was normal. Goldmann perimetry on the right showed a peripheral defect, more pronounced in the lower than in the upper temporal quadrant but

otherwise almost identical to the one seen in Case 1 (Fig. 3). The visual field of the left eye was full. Results of a dilated fundus examination were normal in both eyes. A 30-degree full-threshold static perimetry performed ten days after the initial examination was full. Computed tomography was normal, but on magnetic resonance imaging performed one month after onset of symptoms, a small enhancing lesion in the left upper anterior occipital cortex was seen (Fig. 4). It was interpreted as a subacute stroke. An incidental pineal cyst was also present.

## DISCUSSION

IN 1937 BENDER AND STRAUSS<sup>1</sup> PUBLISHED A SERIES OF ten cases of missing temporal crescent found among 100 perimetrically studied patients with brain tumors involving the optic radiations. On the basis of these ten cases, they concluded that temporal crescentic defects result from involvement of the distal optic radiation or anterior visual cortex and that they may represent the earliest sign of brain tumors. They postulated that "indeed, this defect may even be found to be the most frequent type of distortion in

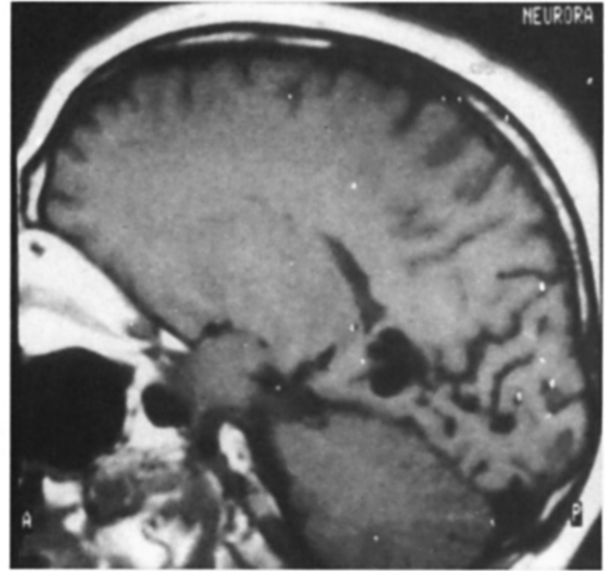
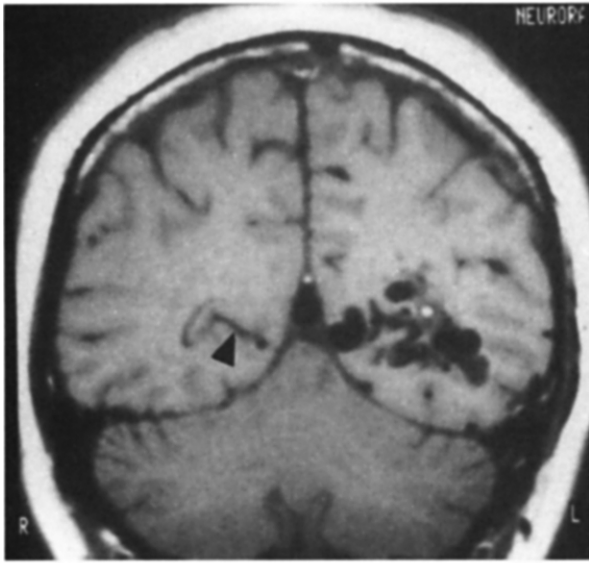


Fig. 2 (Landau, Wichmann, and Valavanis). Case 1. Left, Coronal 4-mm T<sub>1</sub>-weighted magnetic resonance image at the level of the anterior visual cortex, showing a left-sided arteriovenous malformation, draining by dilated veins. For orientation, an arrowhead marks the contralateral calcarine fissure containing a small vein. Right, T<sub>1</sub>-weighted parasagittal view of the arteriovenous malformation, located predominantly in the anterior part of the occipital lobe.

the fields of vision of patients with tumor of the brain." This prediction was not confirmed in subsequent decades. Reports about the missing temporal crescent are rare. Authors of perimetry books and

chapters describe the interesting phenomenon of the missing temporal crescent either entirely on theoretical grounds or in reference to Bender and Strauss.<sup>1</sup> In 1974 Walsh<sup>2</sup> studied two patients, one with a pre-

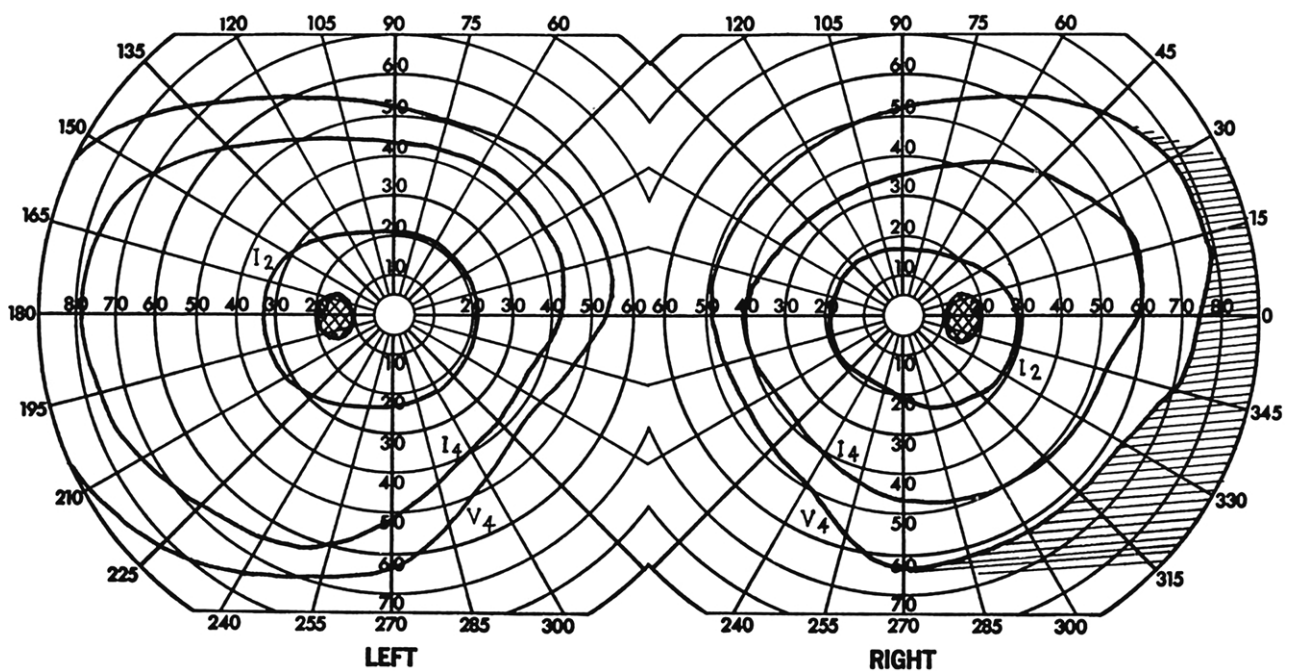
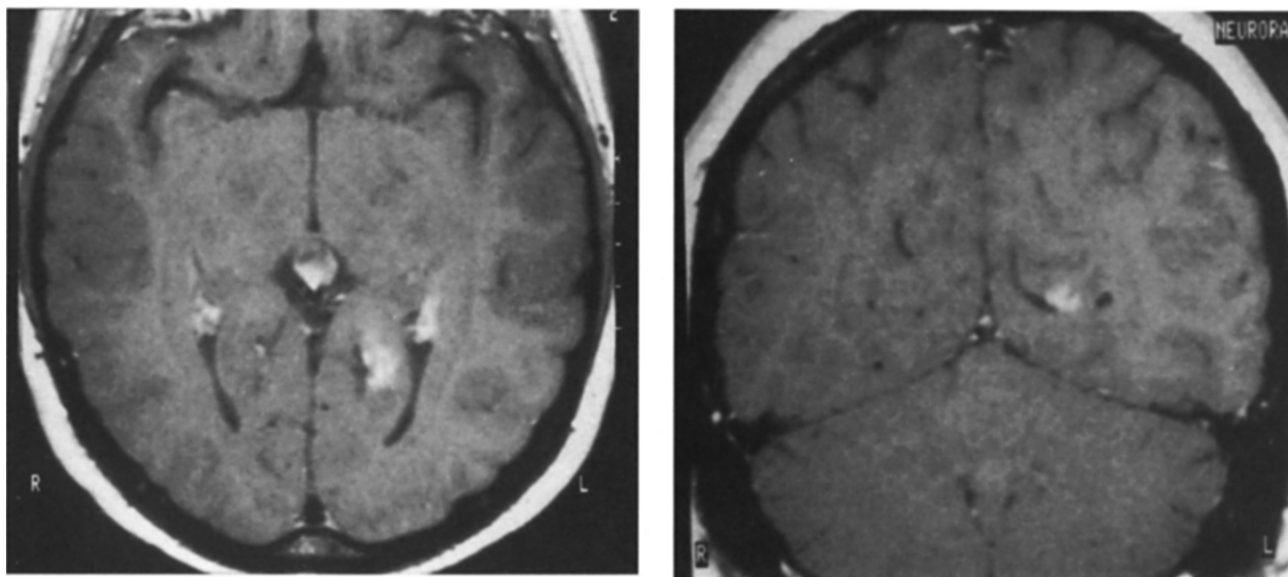


Fig. 3 (Landau, Wichmann, and Valavanis). Case 2. Right temporal peripheral defect, more pronounced in the lower than in the upper quadrant. No homonymous defect on the left side.



**Fig. 4** (Landau, Wichmann, and Valavanis). Case 2. Three-millimeter  $T_1$ -weighted gadolinium-enhanced magnetic resonance image, showing a minute subacute cortical infarction. Left, The axial view demonstrates that the lesion is located in the most anterior portion of the occipital cortex, measuring approximately  $13 \times 8$  mm. The distance between the bars on the right is 10 mm. Right, The coronal view demonstrates that only the cortex above the calcarine fissure is involved.

served monocular temporal crescent in the presence of a dense homonymous hemianopsia and the second with loss of the temporal crescent. Neuroradiologic studies were not available at that time. Clinicopathologic correlation was provided in a single case by Ask-Upmark<sup>3</sup> in 1932.

Our first patient demonstrates that large long-standing arteriovenous malformations may produce few neurologic deficits. On the basis of the neuroradiologic findings, this patient might have had any visual field defect, ranging from total right homonymous hemianopsia to no deficit at all. The missing temporal crescent in this patient may be a result of damage to optic radiation, cortical damage, or a combination of both.

Our second patient is of particular interest because the missing temporal crescent was caused by a purely cortical lesion. In a recent study by Horton and Hoyt,<sup>4</sup> in which a revised map of the representation of the visual field in human striate cortex was provided, the 40 degrees of the temporal crescent correspond to less than 10% of the total surface area of the striate cortex. This surface area is roughly equal to the area corresponding to the central 1 degree of vision. Horton and Hoyt<sup>4</sup> stated that testing the central 30

degrees of visual field by using full-threshold automated perimetry provides information on as much as 83% of the striate cortex. In the striate cortex, the most anterior portion that subserves the temporal crescent and gets input only from the contralateral eye has a distinct feature: it lacks ocular dominance columns. This feature allowed precise identification and measurement of this cortical region. The cortex subserving the monocular crescent in the case by Horton and associates<sup>5</sup> was found to have a dimension of  $154 \text{ mm}^2$ . The purely cortical lesion in Case 2 measured roughly  $13 \times 8$  mm, thus affecting an area of approximately  $100 \text{ mm}^2$ . If one takes into account that the temporal crescent was completely missing in the lower quadrant but was partially spared in the upper quadrant, then this discrepancy in size is easily reconciled. The magnetic resonance scan shows that the lesion involved the upper calcarine bank, which represents the lower temporal crescent. Thus, both the size and the location of the cortical lesion correspond to the perimetric defect and fit nicely with the revised map<sup>4</sup> of the representation of the visual field in the human striate cortex, as discussed previously.

Various reasons may account for the paucity of

reports on the missing temporal crescent. Such a defect may simply be unnoticed by the patient. In congenital lesions, even total homonymous hemianopsias can remain undetected for many years.<sup>6</sup> It is thus not surprising that our first patient was asymptomatic. In acquired lesions, the missing temporal crescent may be more readily appreciated if it occurs suddenly, as in Case 2 and in the patient studied by Walsh.<sup>2</sup> In slowly progressive lesions it may remain unnoticed by the patient because of little resulting handicap.

This type of visual field defect can be missed by the examiner. When performing Goldmann perimetry, the largest stimulus should be presented at the edge of the cupola, to include the temporal crescent. Another quick way to examine the temporal crescent is to move the examiner's hand slowly, starting behind the patient's head, anteriorly. This approach will show that the temporal visual field border extends beyond 90 degrees, with variations because of individual anatomic factors, such as deep or shallow orbits. Confrontation testing or finger counting in the four quadrants will not include all of the temporal peripheral field because the examiner's hand is not long enough to enter the temporal crescent out of the blind area. Computerized automated perimetry will not show a missing temporal crescent.

On the basis of a prospective study of 215 visual field defects caused by lesions of the visual pathways, Wellings<sup>7</sup> stated that such lesions are detectable within 30 degrees of fixation and that more-peripheral field testing is ineffective. We agree with Horton and Hoyt<sup>4</sup> that a lesion affecting only the most anterior 10% of the striate cortex must be truly rare. For most clinical situations it is sufficient to examine the central visual field when a retrochiasmal

lesion is suspected. However, the perimetrist should be aware that, although a 30-degree visual field provides information on 83% of the striate cortex, it does not provide information about the remaining 17%.

With this study, we draw attention to the missing temporal crescent, which may be a more common defect than is documented in the medical literature. A simple finger confrontation screening for such a defect will quickly enable the examiner to decide whether careful Goldmann perimetry should follow. We provide a magnetic resonance image of a missing temporal crescent caused by an isolated cortical lesion. It is in accord with the current information on the representation of the visual field in the human striate cortex.<sup>4,8</sup>

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## REFERENCES

1. Bender MB, Strauss I. Defects in visual field of one eye only in patients with a lesion of one optic radiation. *Arch Ophthalmol* 1937;17:765-87.
2. Walsh TJ. Temporal crescent or half-moon syndrome. *Ann Ophthalmol* 1974;6:501-5.
3. Ask-Upmark E. On the cortical projection of the temporal half-moon of the visual field. *Acta Ophthalmol* 1932;10:271-90.
4. Horton JC, Hoyt WF. The representation of the visual field in human striate cortex. *Arch Ophthalmol* 1991;109:816-24.
5. Horton JC, Dagi LR, McCrane EP, de Monasterio FM. Arrangement of ocular dominance columns in human visual cortex. *Arch Ophthalmol* 1990;108:1025-31.
6. Bajandas FJ, McBeath JB, Smith JL. Congenital homonymous hemianopia. *Am J Ophthalmol* 1976;82:498-500.
7. Wellings PC. Detection and recognition of visual field defects resulting from lesions involving the visual pathways. *Aust N Z J Ophthalmol* 1989;17:331-5.
8. McFadzean R, Brosnahan D, Hadley D, Mutlukan E. Representation of the visual field in the occipital striate cortex. *Br J Ophthalmol* 1994;78:185-90.