

the epileptics probably represented a postictal effect. Of the six epileptics who had a second EEG at least two weeks after the initial tracing, four had resolution of the delta focus; none had focal CT abnormalities. One of the two patients with persistent delta activity had a CT scan that showed a focal abnormality.

Masdeu et al<sup>6</sup> reported a 20% incidence of normal CT scans in cases of acute ischemic strokes and in our study the CT scan failed to demonstrate a focal abnormality in six of 32 patients (19%) with acute ischemic

infarctions. Thus, the EEG revealed dysfunction not yet detectable morphologically.

This study emphasizes the importance of continuous focal delta activity and its frequent association with a gross structural lesion, while demonstrating the complementary nature of the CT scan and the EEG in diagnosis.

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## Central Dazzle

### A Thalamic Syndrome?

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• A patient who experienced painless intolerance to light (dazzle) three months after a right posterior cerebral artery occlusion was shown by computerized tomography to have right occipital and right thalamic infarctions. His symptoms improved with amitriptyline hydrochloride and perphenazine therapy. The sensitivity to light, delayed onset, response to therapy, and presence of a thalamic lesion are analogous to the thalamic pain syndrome and suggest that central dazzle is a variant of the thalamic syndrome.

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Excessive sensitivity to light may be secondary to true photophobia or to photophobia due to dazzle.<sup>1</sup> True photophobia is pain on exposure to light typically associated with inflammatory conditions of the anterior segment of the eye. Dazzle is the painless

exaggeration of brightness as described by persons with toxic amblyopia, albinism, rod monochromatism, and corneal, lenticular, and vitreous opacities that disperse light. Sensitivity to light is unusual as a consequence of CNS disease. Walsh and Hoyt<sup>2</sup> mention its occurrence in trigeminal neuralgia, migraine, meningitis, subarachnoid hemorrhage, sellar and suprasellar tumors, during and after retrobulbar neuritis, and following head trauma. We describe a patient who experienced dazzle after an infarction in the territory of the right posterior cerebral artery (PCA). Clinical, radiologic, and therapeutic evidence suggests that a thalamic lesion accounts for the light intolerance.

#### REPORT OF A CASE

A 62-year-old right-handed man was well until November 1977, when without warning a "herringbone" or "tire tread" pattern appeared in the left lower portion of his vision and lasted for several seconds. There were no other associated symptoms. The pattern transiently recurred later that evening. The following day his vision seemed "blurry" or "fuzzy," and two days later he got lost on his way to work. He was seen by a neurologist and an ophthalmologist, who found a dense congruous left superior quadrantanopsia. No other cranial nerve, motor, sensory, or reflex abnormalities were noted. His mental status was unremarkable, except for a tendency to get lost in a hospital well known to him. A computerized tomographic (CT) scan of the head revealed an area of diminished densi-

ty in the territory of the right posterior cerebral artery.

In January 1978, three months after the initial event, the patient first noted an unusual sensitivity to bright lights. This condition progressed rapidly and became severe during the course of one week. The sensitivity was continuous, involved all the remaining visual field, and was uniform throughout. He described the light intolerance as disagreeable rather than truly painful and "like going into the sunshine from a dark room." He acquired five pairs of glasses of varying tints and wore a sun visor. He kept his shades drawn in the house and closed his eyes while outside in the daylight. Coincident with the development of the light sensitivity he also noted a "milky haze" that partially obscured his vision. He had difficulty adjusting to changes in illumination, and light contrasts were increased so that shadows seemed darker and lighted areas lighter. Colors appeared less distinct. A former amateur oil painter, he could no longer mix and match paints correctly. He continued to have mild difficulty finding his way in familiar areas, and he occasionally had trouble recognizing familiar people. On one occasion he "saw" his wife's arm in his blind field. No other hallucinations and no metamorphopsias were reported. Prior to his illness the patient had been employed for many years as an inventory controller for a large company. He described himself as a perfectionist, but there was no evidence of a premorbid psychiatric disorder.

The patient was referred for further evaluation of his visual disturbance. Mental status, motor, somatic sensory, and reflex examinations were normal. Color naming was intact. When tested under

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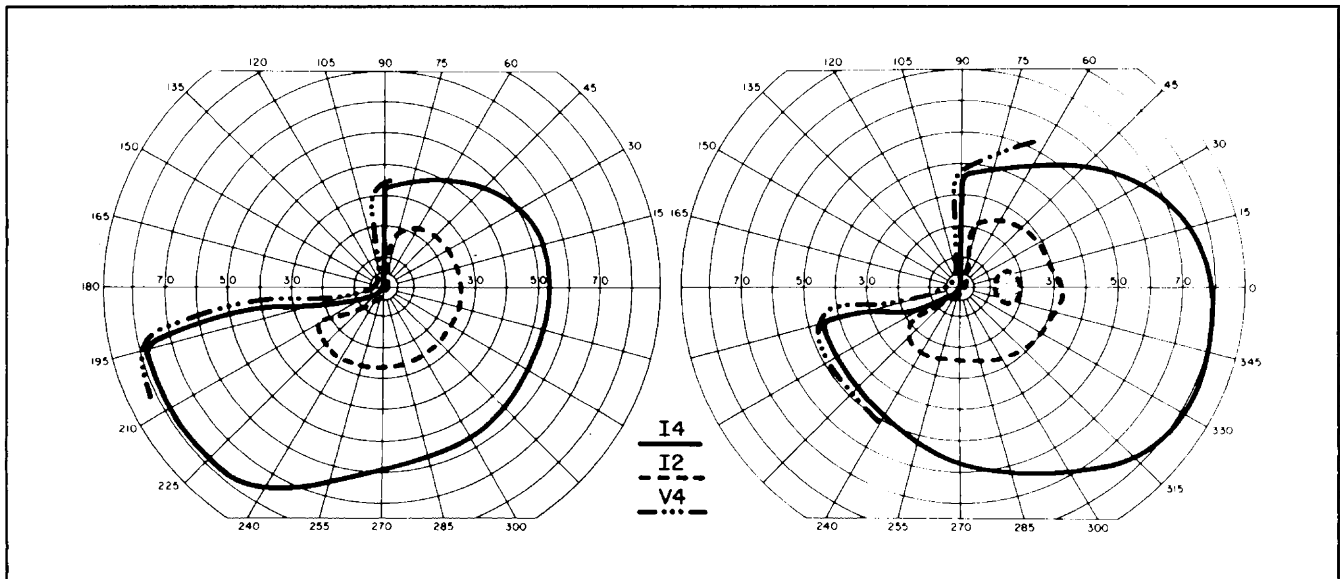


Fig 1.—Visual fields showing congruent left superior homonymous quadrantanopia.

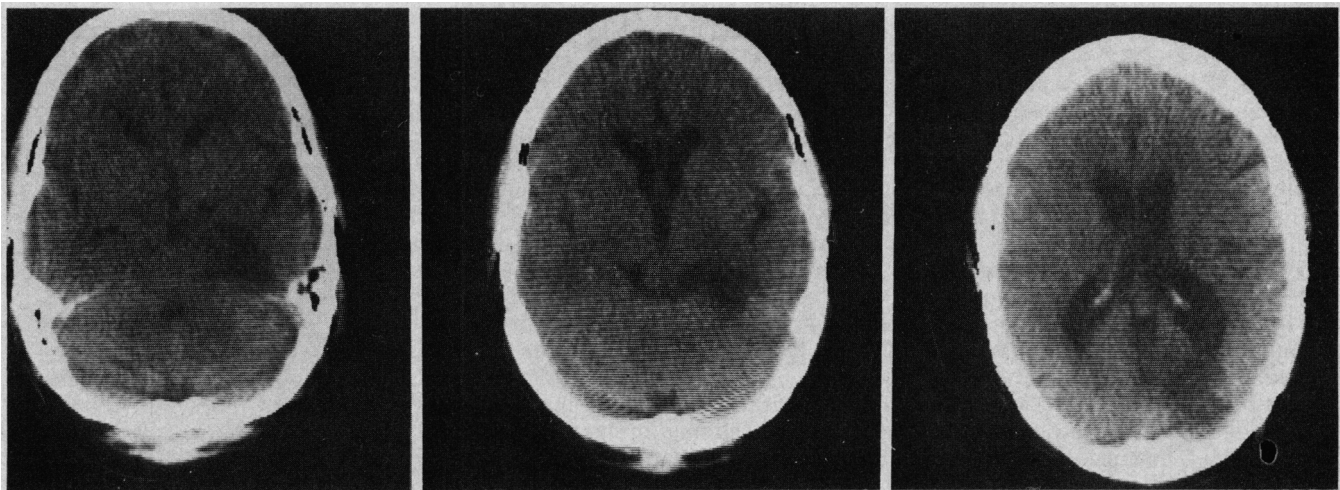


Fig 2.—Computerized tomograms showing infarction in distribution of right posterior cerebral artery. Central image demonstrates loss of substance in upper brainstem on right, producing concave indentation laterally at level of mesodiencephalic junction.

optimal conditions he had no difficulty matching faces with similar expressions or recognizing pictures of famous people or the faces of ward personnel. His performance on a battery of neuropsychological tests was average or above (verbal IQ, 130; performance IQ, 110; full-scale IQ, 123) except for a substantial impairment on the visual memory portions of the Wechsler Memory Scale.

Fundi, extraocular movements, optokinetic nystagmus, pupillary responses, and slitlamp examination results were normal. Visual acuity was 20/20 in each eye. Perimetry revealed a congruous left homonymous quadrantanopia (Fig 1). Stereoscopic vision was impaired (he failed to detect arcs of less than 100 s on the Titmus stereo tests). Color vision as tested with Ishihara's pseudoisochromatic plates was normal, but he scored in the impaired range on the

Farnsworth-Munsell 100 Hue Test of color hue discrimination (score, 132; normal, 20 to 100). Dark adaptation was normal in both eyes.

An EEG revealed asymmetric 9-Hz alpha activity of lower voltage on the right and an asymmetric response to photic stimulation with a smaller response amplitude on the right. An electroretinogram was normal. Pattern-reversal visual evoked potentials were unrecordable for 12-minute checks (stimulus squares subtending 12 minutes of visual angle) and normal for 48-minute checks. A CT scan (Fig 2) showed an area of diminished density in the occipital and posterior inferior medial temporal regions of the right hemisphere. There was also loss of substance in the right upper brainstem laterally at the level of the mesodiencephalic junction, with widening of the quadrigeminal cistern.

A variety of medications were prescribed in an attempt to modify the light intolerance. Therapeutic levels of phenytoin sodium and diethylcarbamazepine citrate had no effect. Ethosuximide produced a minimal ameliorating response. Partial relief was obtained with a combination of amitriptyline hydrochloride (150 mg/day) and perphenazine (6 mg/day). On this regimen, however, acute urinary retention developed. When the medications were stopped, the light sensitivity promptly returned. The patient then underwent a transurethral prostatectomy for benign prostatic hypertrophy. Following surgery, the combination of amitriptyline and perphenazine was reinstated and the excessive sensitivity to brightness again diminished. There was no change in the milky haze of his vision or in the difficulty adjusting to light contrasts.

## COMMENT

This patient presented a variety of disorders of higher visual function, including visual hallucinations, topographical disorientation, a history suggestive of prosopagnosia, and impaired color perception. His most disabling problems were marked light intolerance, the appearance of a milky haze obscuring his vision, and the exaggeration of light and dark contrasts. The latter abnormalities developed three months after an infarction in the distribution of the right PCA. Evaluation revealed a failure to elicit visual evoked potentials with small stimulus squares, impaired performance on the Farnsworth-Munsell color hue discrimination test, altered depth perception, and disturbed visual memory. The discomfort produced by light was partially ameliorated by treatment with a combination of amitriptyline and perphenazine. A CT scan demonstrated an occipital infarction and a lesion at the mesodiencephalic junction.

Several of the patient's deficits can be explained by the infarction in the distribution of the right PCA, whereas others suggest bilateral ischemic injury. Impaired visual memory,<sup>3</sup> defective depth perception,<sup>4</sup> visual hallucinations,<sup>5</sup> and failure on the Farnsworth-Munsell hue discrimination test<sup>6</sup> have been reported with unilateral right posterior lesions. Topographic disorientation, a common accompaniment of central achromatopsia<sup>7</sup> and of prosopagnosia,<sup>8</sup> occurs in patients with bilateral posterior cerebral damage. The milky haze and contrast alterations complained of by the patient have also been described in patients with bilateral occipital infarctions. Critchley<sup>9</sup> noted that some patients with visual agnosia-like syndromes saw things as "darker, smaller, and misty" or "as though there was a thin layer over it all." Similarly, the artist with visual agnosia described by Wapner et al<sup>10</sup> complained that "everything seemed dim, as if it were twilight." The lesions producing visual agnosia involve the region of the inferior longitudinal fasciculus bilaterally.<sup>11</sup>

The unusual light sensitivity experienced by this patient beginning several months after the PCA occlusion resembles closely the delayed onset of dysesthesia and hyperpathia following thalamic infarction and suggests that the observed mesodiencephalic lesion was responsible for the dazzle. The syndrome of thalamic infarction<sup>12</sup> is characterized by an acute transient

hemiparesis, disturbed sensation, and mild hemiataxia of the weak limbs followed several months later by severe persistent pains involving the previously paretic side. Sensory stimulation increases the pain (hyperpathia). Pathological study revealed infarction in the posterior and lateral nuclei of the thalamus in the distribution of the thalamogeniculate and thalamoperforate branches of the PCA.<sup>13</sup> In most cases of the thalamic syndrome, somatic sensation alone is involved. Occasionally, however, other sensory modalities are implicated. Riddoch<sup>14</sup> noted that a sudden bright light, a loud or unexpected noise, and less often intense smell or strong taste caused intense discomfort, and Head<sup>15</sup> described a patient who could no longer go to worship because he "could not stand the hymns on his affected side." Treatment of the pain in the thalamic syndrome is difficult, but recently some success has been achieved with the combination of a tricyclic antidepressant and a phenothiazine<sup>16</sup> used in this patient.

The contribution of the cortical damage to the light sensitivity cannot be determined with certainty. "Pseudothalamic" central pain after cortical injury has been described<sup>17,18</sup> but is infrequent, while dysesthesia and hyperpathia following thalamic lesions are common. Furthermore, patients with occipital infarctions have been extensively studied, and central dazzle rarely has been noted. The phenomenon was not mentioned in patients with cortical lesions producing agnosia, central achromatopsia, visual hallucinations, topographical disorientation, or in large series of patients with hemianopsias.<sup>5,6,8-11,19,20</sup> In a report by Meadows,<sup>7</sup> one patient with prosopagnosia complained that colors were excessively bright on sunny days, but the subcortical extent of the patient's lesion was unknown. Huber<sup>21</sup> described a patient with excessive light sensitivity who "would cover his eyes with a dark cloth, even indoors." The patient had a midbrain tumor, and Huber proposed a regulatory role for mesencephalic and thalamic structures with light intolerance resulting from disrupted regulation.

In summary, this patient had no ocular disease and no other disturbance known to cause photophobia or dazzle. The infarction in the cortical territory of the right PCA contributed to the complex visual syndrome, but by itself was an unlikely cause of the light intolerance. The sustained sensitivity to sensory stimulation, delayed onset, response to therapy, and pres-

ence of a diencephalic lesion are analogous to the thalamic pain syndrome and suggest that central dazzle is a consequence of thalamic injury.

Paul Vinger, MD, referred the patient; D. Frank Benson, MD, John Carter, MD, and Thedi Landis, MD, aided in the clinical evaluation; Edith Kaplan, PhD, and William Milberg, PhD, performed the neuropsychological assessment; Harvey Levine, MD, interpreted the CT scan; Samuel Sokol, MD, did the visual evoked potential studies; Lorne Drenfeld, MD, brought Head's cases to our attention; and Gayle Lawrence prepared the manuscript.

## Nonproprietary Names and Trademarks of Drugs

Amitriptyline hydrochloride—*Amavil*, *Amitid*, *Amitril*, *Elavil*, *Endep*.  
Ethosuximide—*Zarontin*.

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