Auditory-Visual Synesthesia

Sound-Induced Photisms

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- Nine patients with visual loss due to lesions of the optic nerve or chiasm experienced photisms induced by sound. Descriptions of these varied from simple flashes of white light to complicated colorful hallucinations likened to a flame, a petal of oscillating lines, a kaleidoscope, or an ameba; they always appeared within a defective portion of the visual field as demonstrated by perimetry. The provoking sounds were usually those of normal daily life, ranged from soft to loud, and always seemed to be heard by the ear ipsilateral to the eye in which the photism was seen. Sound-induced photisms occurred under circumstances that would promote a startle reaction to sound, and each patient was startled when the photisms occurred. Visual evoked responses demonstrated partial deafferentation of the eye in which photisms were seen in seven patients tested. The phenomenon may occur when the patient has a partially deafferent visual pathway is startled by sound.

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The inherent potential of the normal brain for "cross-talk" between the auditory and visual senses is demonstrated by rare, healthy individuals who sometimes see lights and colors when they hear certain sounds. However, the occurrence of such auditory-visual synesthesias as a manifestation of disease of the CNS is not generally appreciated. We describe nine patients with visual loss due to lesions of the anterior portions of the visual pathways who experienced sound-induced photisms, and we present data suggesting that the effects of an acoustic startle reaction on a partially deafferent visual system may be important in the development of the phenomenon.

CLINICAL OBSERVATIONS

The cases are summarized in Table 1. The site of the lesion causing visual loss was the optic nerve in seven patients and the optic chiasm in two patients. A definite diagnosis was made in every patient except in case 9. Multiple sclerosis (MS) was diagnosed in three patients (cases 1 to 3) who had clear histories of exacerbations and remissions of disseminated neurologic deficits prior to the onset of visual symptoms. The patients with MS had unequivocally positive hot-bath tests and visual evoked responses (VERs) consistent with demyelination. Each had elevated serum total protein content, and two had elevations of y-globulin fraction in the CSF. Each patient with MS improved with adrenocorticotropic hormone therapy. Tumors of the sella turcica-juxtasellar regions were diagnosed (by tomographic radiographs, angiography, and computerized tomography [CT]) in cases 4 and 5. Craniotomy revealed a tuberculum sellae meningioma in case 5; the tumor type was unknown in patient 4, who refused surgery. Temporal arteritis was diagnosed in cases 6 and 7 by elevated ESIs and examination of temporal artery biopsy specimens. Patient 8 had been amaurotic on the right side for 25 years since surgery for removal of neurofibroma at the orbital apex. Probable vascular occlusive optic neuropathy was diagnosed in patient 9, a chronically hypertensive woman with atherosclerotic heart and peripheral arterial occlusive disease. Her cerebral angiogram revealed luminal narrowing and plaques of both cerebral arteries and right-sided intracavernous carotid stenosis. Fluorescein angiography revealed narrowed retinal arterioles, but no definite arteriolar occlusion. The onset of visual loss associated with photisms was sudden (seconds) in cases 8 and 9; over four to seven days in cases 1 to 3, 6, and 7, and gradual (six months and two years) in cases 4 and 5. The photisms were seen within one to three days after the first symptom of visual loss in eight patients, the only exception being patient 8, in whom photisms were first experienced three months after the onset of right-sided amaurosis. The first photism experienced by each patient was of the same degree of brightness as subsequent ones. There was no gradual "buildup" of photism intensity over time. Optic atrophy and Marcus Gunn pupillary reactions were present in the eye in which the photisms were seen in seven patients.

Photisms

The most commonly described photisms were small to large flashes of white light in the scotoma (Table 2). The sounds of walls "crackling" as they cooled at night, an
extinguished television set “scintillating, cracking” as it cooled, a digital clock changing numbers, and a pencil striking a desk induced a large, round flash of light surrounded by a pale blue rim in the upper peripheral portion of her right superior quadrant achromatopsia. Rarely, the flash of light was homogeneously colored as in patient 9, in whom the sounds of a gas furnace turning on, a door slamming, or voices induced a solid pink photism that filled the entire visual field (scotomatous and non-scotomatous portions) of the right eye.

Other photisms were more complex. An illuminated white, yellow, orange, and red flame photism filled the nasal scotoma of patient 1 when she heard “sharp” noises to her left (Fig 2). On other occasions, similar sharp sounds induced a plain white flashbulb hallucination in an identical location. The sounds of a furnace turning on (“muffled”), a dog bark, a tray crashing to the floor, and a woman’s voice on the right eye appeared when the same sounds were heard on other occasions. Each patient’s photisms lasted “a split second, an instant.”

Five patients (cases 3, 5, 6, 8, and 9) experienced a single stereotyped photism (always the same in appearance and location), patients 1 and 4 experienced two, and patients 2 and 7 experienced three to five different photisms.

**Sounds**

The different provoking sounds that could be identified (Table 2) ranged from two (cases 2, 5, and 8) to five (cases 3 and 9). They fell into five categories as follows: (1) soft clicks (eg, electric-blanket thermostat); (2) thuds that were of soft (eg, digital clock), medium (eg, CT gantry, pencil striking desk), or loud intensities (eg, door, fist, book slam); (3) soft cracklings (eg, walls and television cooling); (4) loud, harsh (eg, dog bark, tray crash, engine roar); and (5) voices that were of soft or medium intensities (eg, nurse speaking, voices from television or radio) or loud (hospital paging system). All of the provoking sounds seemed to be of mixed frequencies of wide range (door slam, 250 to 1,500 Hz; digital

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**Table 1.**—Clinical Features of Nine Patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Site</th>
<th>Disease</th>
<th>Field Defect</th>
<th>Visual Acuity</th>
<th>OA Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/25/F</td>
<td>70</td>
<td>F</td>
<td>Optic nerve</td>
<td>MS</td>
<td>Nasal, OS</td>
<td>OD 20/20</td>
<td>OS 20/70</td>
</tr>
<tr>
<td>2/38/F</td>
<td>20</td>
<td>F</td>
<td>Optic nerve</td>
<td>MS</td>
<td>Nasal, superior temporal, OD</td>
<td>OD 20/400-20/40</td>
<td>OS 20/20</td>
</tr>
<tr>
<td>3/25/M</td>
<td>70</td>
<td>M</td>
<td>Optic nerve</td>
<td>MS</td>
<td>Full-field scotoma, OD</td>
<td>OD LP</td>
<td>OS 20/20</td>
</tr>
<tr>
<td>4/44/F</td>
<td>50</td>
<td>F</td>
<td>Chiasm</td>
<td>Tumor</td>
<td>Superior temporal, OD</td>
<td>OD 20/50</td>
<td>OS 20/20</td>
</tr>
<tr>
<td>5/52/F</td>
<td>30</td>
<td>M</td>
<td>Chiasm</td>
<td>Tumor</td>
<td>Bilateral constriction</td>
<td>OS 20/30</td>
<td>OS 20/30</td>
</tr>
<tr>
<td>6/61/F</td>
<td>80</td>
<td>F</td>
<td>Optic nerve</td>
<td>Arteritis</td>
<td>Full-field scotoma OD</td>
<td>OD 20/800</td>
<td>OS 20/40</td>
</tr>
<tr>
<td>7/49/M</td>
<td>50</td>
<td>M</td>
<td>Optic nerve</td>
<td>Arteritis</td>
<td>Full-field scotoma, OD</td>
<td>OD LP-20/25</td>
<td>OS 20/20</td>
</tr>
<tr>
<td>8/71/M</td>
<td>50</td>
<td>F</td>
<td>Optic nerve</td>
<td>Postsurgical</td>
<td>Amaurosis, OD</td>
<td>OD NLP</td>
<td>OS 20/20</td>
</tr>
<tr>
<td>9/70/F</td>
<td>20</td>
<td>F</td>
<td>Optic nerve</td>
<td>Vascular occlusive(?)</td>
<td>Superior altitudinal nasal scotoma, OD</td>
<td>OD 20/50</td>
<td>OS 20/30</td>
</tr>
</tbody>
</table>

*OA indicates optic atrophy; MS, multiple sclerosis; LP, light perception only; NLP, no light perception; +, present; —, absent.

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**Table 2.**—Characteristics of Photisms and Sounds That Induced Them

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Photism Appearance</th>
<th>Color</th>
<th>Location</th>
<th>Sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Flame, flashbulb</td>
<td>Red-orange, white</td>
<td>In scotoma</td>
<td>Not sure (sharp)</td>
</tr>
<tr>
<td>2</td>
<td>Spray, microscope light, kaleidoscope, spot, pollywogs</td>
<td>White, pink, red, black, green</td>
<td>In and out of scotoma</td>
<td>Clap, computerized tomography gantry</td>
</tr>
<tr>
<td>3</td>
<td>Flash</td>
<td>White-yellow</td>
<td>In scotoma</td>
<td>Walls cracking, digital clock, pencil striking desk, television scintillating and cracking</td>
</tr>
<tr>
<td>4</td>
<td>Light bulb</td>
<td>White-blue</td>
<td>In scotoma</td>
<td>Car or motorcycle engines, others (loud)</td>
</tr>
<tr>
<td>5</td>
<td>Flash</td>
<td>White</td>
<td>In and out of scotoma</td>
<td>Digital clock</td>
</tr>
<tr>
<td>6</td>
<td>Flashbulb</td>
<td>White</td>
<td>In scotoma</td>
<td>Electric blanket, digital clock</td>
</tr>
<tr>
<td>7</td>
<td>Petal, ameba, goldfish</td>
<td>Pink, white, yellow</td>
<td>In and out of scotoma</td>
<td>Furnace, dog bark, tray crash, voices</td>
</tr>
<tr>
<td>8</td>
<td>Plaid</td>
<td>Green</td>
<td>In scotoma</td>
<td>Book or flat slamming desk, others (loud)</td>
</tr>
<tr>
<td>9</td>
<td>Flashbulb</td>
<td>Pink</td>
<td>In and out of scotoma</td>
<td>Furnace, door slam, television, radio, voices</td>
</tr>
</tbody>
</table>
Fig 1.—Case 3. Top, Large, irregularly bordered right central scotoma (motion) partially surrounded a chromatopsic periphery where light and motion could be perceived. Visual field of left eye (not shown) was normal. Bottom, "Flash" photism.

Fig 2.—Case 1. Top, Macula sparing nasal defect of left visual field. Visual field of right eye (not shown) was normal. Bottom, "Flame" photism induced by "sharp" sounds.

Fig 3.—Case 7. Left, Defect of right visual field including nasal crescentic anopsia, nasocentral achromatopsia, and temporal dyschromatopsia. Visual field of left eye (not shown) was normal. Center, "Petal" photism. Right, "Ameba" photism.

Fig 4.—Case 2. Left, Nasal, temporal, and superior defect of right visual field. Visual field of left eye (not shown) was normal. Right, "Spray" photism.
sound had startled him. The others were drowsiness. Patient 8 was certain that the uncertain as to whether the sound or the resulted in arousal from early sleep or occurred were similar in seven patients and fell asleep; this period never exceeded 18 minutes per week for 18 months in one patient (case 8), five months (approximately once every three to four weeks) changing to two to three per day during the next six months for patient 4, and three times per year for 25 years for patient 8. The photisms disappeared in patient 7 after five months but have persisted in the other patients. In patients who saw more than one photism, the same sound that induced a given photism on one occasion might induce a different one on another occasion. The sound of a moving CT gantry might induce a spray photism in the right eye of patient 2 on one occasion and a microscopelight photism on another occasion. In all patients, a given sound might induce a photism on one occasion but fail to do so on numerous other occasions.

Circumstances of the Photisms and Associated Phenomena

The conditions in which the photisms occurred were similar in seven patients (cases 1 to 3, 5 to 7, and 9). They were always startled, drowsy, or dozing in a quiet, dark and dimly illuminated room with their eyes closed. The photisms occurred between the time they entered the dark and fell asleep, this period never exceeded one hour. Photisms were not experienced when they awakened later in the night. Patients 4 and 8 differed from the others in that they experienced photisms in the light, (but not in the darkness of the room) with their eyelids opened and never experienced them in the dark. Patient 4 was relaxed (but not drowsy) and patient 8 was "tense, anxious, concentrating" when sounds induced the photisms.

Each patient was startled when the photism occurred. Frequently, the startling was associated with a gross body jerk that resulted in arousal from early sleep or drowsiness. Patient 8 was certain that the sound had startled him. The others were uncertain as to whether the sound or the photism had startled them. Each patient stated that the sound, the photism, and the startle seemed to occur simultaneously. Patients 1, 2, 4, and 7 described the experience as "seeing the sound on the inside of their eyelids."

Patients 1, 2, 4, and 7 saw spontaneous (not sound-induced) photisms in the involved eye as well as those induced by sound. Patient 7 began experiencing spontaneous photisms at a time when vision in the right eye had improved to near normal and sound-induced photisms were ceasing. They continued for one month after sound-induced photisms had ceased completely. In cases 1, 2, and 4, spontaneous photisms began at approximately the same time as those induced by sounds and have continued (approximately 20% as frequently as those induced by sound). Spontaneous photisms occurred under identical conditions and were of similar characteristics as those induced by sound. When spontaneous photisms occurred, they were not related to eye movement, sudden lid closure, or any other identifiable maneuver. Photisms could not be induced by tapping or application of a vibrating tuning fork to the orbital bone or globe (lids closed) or vigorous deviations of the eyes.

Neurophysiologic Studies

Visual evoked responses (black-white checkerboard reversals) and auditory evoked responses (AERs: binaural and monaural stimuli, broad band clicks, using standard Nicolet, 1170 averager) were performed on patients 1, 2, and 5 to 9. All seven patients had normal AERs but abnormal VERs (bilateral, case 1, unilateral, others). The mean latency to P1 (second positive deflection on derived EEG after stimulus onset) was 142.2 ms (129.8 to 170.4 ms) in the eye in which the photism was seen and 108.4 ms (101.9 to 129.5 ms) in the opposite eye (Fig 5). The range of normal P1 latencies for our laboratory is 85 to 110 ms. The VER amplitudes from the involved eyes were reduced to 50% to 60% of normal.

Photism induction was attempted in cases 1 to 3, 6, 7, and 9. Tones of 200 ms at frequencies of 250, 500, 1,000, 1,500, 2,000, and 3,000 Hz were presented monaurally through earphones at intensities of 60, 70, 80, 85, and 95 dB, sound pressure level (SPL) to each patient immediately, after five minutes, and then at successive ten-minute intervals after they had entered a pitch-black (but not soundproofed) laboratory. Patient 2 saw an irregularly edged petal photism (Fig 3, center) in his right eye on two occasions (second positive deflection on derived EEC 170.4 ms) in the eye in which the photism was seen. The VERs revealed that visual deafferentation was incomplete in each patient tested, including patient 8, who had been subjectively blind in the right eye (in which photisms were seen) for 25 years. The VERs elicited on stimulation of the scotomatus eyes revealed conductive delays (prolonged P1 latencies) and reduced evoked potential amplitudes. Unilateral VER abnormalities (six of seven patients tested) were always observed on stimulus of the eye in which photisms were seen. Bilateral VER abnormalities observed in case 1 are accounted for by an episode of right-sided retrobulbar neuritis four years before the onset of left papillitis associated with photisms. The VER pattern recorded in these patients has most often (although not exclusively) been associated with demyelination of the visual system, which would be expected in our three patients with MS and which may be a component of pathologic changes in the compressive and ischemic diseases of the other six patients. The possible relevance of demyelination to the development of the phenomenon is also suggested by another report of nine patients with optic neuritis due to MS who experienced hallucinations of light with eye movement. Regardless of specific etiology, the propensity of the partially deafferented, defectively conducting visual system to produce photisms is evidenced by the fact that four of our...
patients (three with the demonstrated VER abnormality) experienced spontaneous as well as sound-induced photisms.

Each patient had evidence of a lesion involving the presynaptic portions of the third-order neuron (axons of retinal ganglion cells) of the visual pathway. Partial deafferentation at this site might produce a heightened state of excitation of the fourth-order neuron (lateral geniculate nucleus [LGN]) since impulse and chemical trophic influences from the third-order neuron on the LGN would still be partially intact (albeit reduced). The LGN supersensitivity might particularly be expected in purely or predominantly demyelinating lesions with axons left intact. Supersensitive states of postjunctional elements following partial deafferentation have been demonstrated in the autonomic and peripheral nervous systems and the CNS (including visual). Partial rather than complete deafferentation has been stressed as the necessary element for the development of such postjunctional supersensitivity.

The LGN, which is normally responsive to sounds as well as visual stimuli, sound stimuli elicit transient discharges that can be recorded from the LGN of normal experimental animals. In such instances, postsynaptic elements may develop new receptive fields (intracellular recordings) that are heightened in responsiveness to novel inputs.

The LGN is normally sensitive to sounds as well as visual stimuli. Photisms and fields of these discharges (pentogeniculo-occipital spikes) induced by startling sounds in monkeys. The exaggerated startling effect on an already supersensitive LGN may be the critical element resulting in a threshold discharge for vision on a sound stimulus that produces photisms. The briefness of the sound-induced photisms and their tendency to habituate (subjectively reported by all patients, experimentally demonstrated in cases 2 and 7) are features that the phenomenon shares with the startle reaction to sound.

The startle reaction also results in abrupt eyelid blinking and deviation of the eyes toward the offending sound, both of which might induce photisms. However, we were unable to induce photisms by forced blinking, eyelid closures, globe-orbit tapping, or eye movement at any time, including the period when photisms were being induced experimentally by sounds delivered through earphones.

The requirement for the sound to be heard by the ear ipsilateral to the eye in which photisms were seen (subjec-


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