Auditory-Visual Synesthesia

Report of a Case With Intact Visual Pathways

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Transformation of a sound stimulus to a visual experience, auditory-visual synesthesia, is a curious phenomenon reported in patients with acquired visual loss involving the anterior visual pathways. We describe a patient in whom a striking auditory-visual synesthesia developed ipsilateral to a large mass involving the medial temporal lobe and the adjacent midbrain. This patient’s neuro-ophtalmologic and neurophysiologic examinations did not disclose any evidence of visual dysfunction. The synesthesia disappeared after removal of the mass.

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REPORT OF A CASE

A 25-year-old man complained of dull left-sided headaches, weakness and numbness of the left side, and intermittent double vision, which had increased in intensity during a four- to five-week period. During the past two years, he had experienced several episodes during which he saw moving spots of light “like comets” in front of both eyes. General physical examination revealed no abnormality. The neurological examination showed a diminished corneal reflex on the left side, a left-sided peripheral facial weakness, decreased pain sensation on the left side of the face and body, and a mild left hemiparesis. He performed tandem gait poorly and displayed appendicular ataxia that was more pronounced on the left. His visual acuity was 20/15 OU. No afferent pupillary defect was seen. Color plate discrimination and results of the Farnsworth 100-hue test were normal. Direct ophthalmoscopy demonstrated full nerve-fiber bundles. The optic discs and macular reflexes were normal. Tangent screen examination with multiple white and red test objects and Goldmann perimetry showed full visual fields. Saccadic and pursuit eye movements appeared normal. Slit-lamp examination with a HRuba lens showed no evidence of vitreous detachment.

Patterner reversal visual evoked potentials (VEPs) and brain-stem auditory evoked potentials (BAEPs) were normal. During the BAEP study, at intensities above a 65-dB sensation level (SL), the patient noted a pronounced visual experi-

References

in the left eye. It occurred only with sound presentation to the left ear and increased in intensity with an increasing click rate (Table). The patient did not demonstrate startle response during the BAEP study. There was no notable diminution of the eyes toward the auditory stimulus, and eye movements in the absence of sound produced no visual experience. We later exposed him to different types of loud sound. He reported a less intense but somewhat similar experience in the left eye after left ear stimulation. An audiogram was normal. A computed tomographic scan showed a large, radiolucent mass within the medial temporal lobe on the left side extending to the midbrain (Figure). A left temporal craniotomy exposed a cystic tumor, and the mass was excised. Histological examination of the tissue disclosed gliosis but no evidence of malignancy. Results of repeated BAEP study one month after surgery were essentially unchanged. The patient did not experience auditory-visual synesthesia during repeated testing.

**COMMENT**

Auditory-visual synesthesia can occur in normal persons during hypnotic states. Bender drew attention to the pathological importance of this phenomenon. His patient, a 53-year-old man with a nine-month history of optic neuritis and central scotomas, saw blue lights in the shape of the scotomas each time he heard a loud sound. Lessell and Cohen reported the occurence of sound-induced phosphene in optic neuropathy, optic nerve compression and following keratopathy. They attributed this phenomenon to either postdenervation supersensitivity of bimodal neurons or ephaptic transmission of neural impulses between auditory and visual axons. A more detailed report described nine patients with auditory-visual synesthesia: seven with optic neuropathy and two with chiasmal lesions. All patients displayed evidence of visual field defect on perimetric examination and had abnormal VEPs. Jacobs et al believed synesthesia resulted from postdenervation haversensitivity of lateral geniculate neurons.

Our patient described his visual experience as always in the eye ipsilateral to the ear that received the auditory stimulus. This relationship was puzzling, since present anatomic knowledge dictates extensive crossing of the auditory fibers in the brain stem and arrangement of postchiasmal visual pathways by field rather than eye. We noted, however, that 11 of 12 patients of the last two previously reported communications described their visual experiences similarly. In fact, as with our patient, Jacobs et al experimentally reproduced the ear-eye ipsilaterality in two of their patients.

The data derived from the BAEP test during the past decade indicated existence of an ipsilateral auditory pathway in the human brain stem. Unilateral BAEP abnormalities frequently represent dysfunction of the brain stem on the side of the ear being stimulated. We believe that the sound stimulus responsible for auditory-visual synesthesia reaches the upper brain stem through the same ipsilateral pathway. There, through intercollicular or intergeniculate connections, it contacts the visual system, where it stimulates the bimodal neurons. Those bimodal neurons receiving primarily this ipsilateral auditory input may receive their major visual input from the ipsilateral retina. Under pathological conditions, direct irritation of these neurons or their supersensitivity due to deafferentation results in ipsilateral auditory-visual synesthesia.

Our patient demonstrated the following three special features:

1. The patient's auditory-visual synesthesia was caused by a tumor on the same side. This finding supports the notion of ipsilateral convergence of auditory and visual stimuli at the mesencephalic-diencephalic region.

2. Unlike previously described patients, he showed no evidence of visual system dysfunction on neuroophthalmologic testing.

3. Increasing the rate of aural stimulation clearly increased the intensity of his visual experience, a finding that indicates presence of "rate responsive" auditory-visual units in human beings. Our patient's earlier visual experiences can be best explained as spontaneous photisms, resulting from either disturbance of temporal cortex or mesencephalic-diencephalic regions.

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