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Visual Integration Disorders

By Nurhan Torun, MD, FRCSC

Ophthalmologists occasionally encounter patients who complain that they are not able to see well, yet they have relatively intact visual acuities and fields and an unremarkable examination. Such a scenario may be due to a disorder affecting higher cortical centers involved in the processing of visual information. In other situations (eg, anosognosia), patients may be unaware of visual deficits that are apparent to relatives who are escorting them to the examination. Since these patients are often initially referred to an ophthalmologist, it is important for ophthalmologists to be familiar with the symptoms of visual integration disorders because they may be misled by a seemingly unremarkable examination and miss an existing neurological disorder. In addition, ophthalmologists are occasionally consulted about patients with known neurological disease who have unexplained visual symptoms. In this context, it is helpful to know about the different manifestations of dysfunction in cortical processing centers. This issue of *Ophthalmology Rounds* presents an overview of the anatomy of the afferent visual system, disorders resulting from dysfunction of the occipitotemporal and occipitoparietal pathways, and the diseases that lead to these disorders.

Brief overview of anatomy

The afferent visual system consists of the anterior visual and geniculocalcarine pathways that deliver visual information to the striate cortex and the higher cortical association areas that process these data. The visual association areas are divided into ventral and dorsal pathways (Figure 1). The ventral, or occipitotemporal ("what") pathway, is mainly involved in contrast, colour, and object recognition. The dorsal or occipitoparietal ("where") pathway is involved in spatial orientation and motion perception. Another function of the dorsal pathway is visuospatial attention.

Table 1 lists the occipitotemporal and occipitoparietal pathway disorders that are reviewed in this issue of *Ophthalmology Rounds*. It should be emphasized that these disorders are not mutually exclusive and can be encountered simultaneously because of the proximity of the areas involved.

Occipitotemporal pathway disorders

Visual agnosia

Patients with visual agnosia do not recognize objects by sight alone, even though they have reasonably good vision, cognition, and language.[1] Patients are able to identify objects only if they touch or feel them, or listen to a description of what that object is used for, indicating that "object naming" is intact.[2] Thus, this diagnosis requires demonstration of sufficient vision, but an inability to recognize visualized objects unless there is nonvisual sensory input. This deficit is generally caused by bilateral medial inferior occipitotemporal lesions that disrupt the inferior longitudinal fasciculus, a white matter pathway connecting the striate cortex with visual association areas.[3] Visual agnosia usually occurs following bilateral posterior cerebral artery infarction that initially results in cortical blindness. Patients recovering from visual

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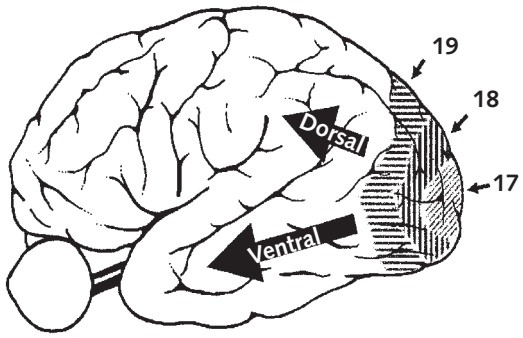
Figure 1: Diagram of occipitoparietal (dorsal) and occipitotemporal (ventral) pathways in relation to primary visual cortex (Brodmann's area 17) and visual association areas 18 and 19



(Adapted with permission from Liu GT, Volpe NJ, Galetta SL. *Neuro-ophthalmology: Diagnosis and Management,* Philadelphia: Saunders; 2001:346.)

Table 1: Visual integration disorders

Disorders of the occipitotemporal pathway	**Symptom/Finding**
Visual agnosia	Inability to identify objects by sight alone
Prosopagnosia	Impaired recognition of faces
Pure alexia	Being able to write, but not to read
Cerebral achromatopsia	Defective colour perception
Disorders of the occipitoparietal pathway	**Symptom/Finding**
Hemispatial neglect	Not acknowledging stimuli presented in one hemispace
Balint's syndrome	Inaccurate reaching under visual guidance, inability to make saccades to visual targets and piecemeal vision
Akinetopsia	Defective motion perception

agnosia often have a specific deficit called "visual anomia," where they cannot name the objects by sight, but are able to describe its function.[4]

Prosopagnosia

This condition describes the inability to recognize familiar faces or learn to recognize a new face. Patients have to use other clues such as stature, body movement, and voice to identify a familiar face.[5] Prosopagnosia is an interesting deficit in that the difficulty is not limited only to faces, as is commonly assumed; these patients are also unable to distinguish different elements under the same broad category, even though they have no problem distinguishing different categories of objects from one another. For example, a patient may be able to distinguish a cat from a dog, but not dogs from two different breeds that resemble each other. Similarly, patients may no longer be able to distinguish between different makes of cars, types of birds, etc.

Patients with prosopagnosia generally have lesions in the lingual and fusiform gyri, as well as the parahippocampal gyri and surrounding white matter. It is useful to remember that prosopagnosia is a relatively rare entity and patients who say they cannot recognize faces often have a psychiatric disorder or anterograde amnesia, rather than prosopagnosia. However, if a patient is unable to recognize the faces of famous people in photographs or family members by sight and can only recognize them by their voices, the diagnosis is likely prosopagnosia.[4]

Pure alexia

Patients with pure alexia, or alexia without agraphia, complain of suddenly not being able to read. This is usually associated with a complete right homonymous hemianopia. Other language functions (eg, spelling and writing) are spared, but these patients are unable to read what they have just written. This is in contrast to alexia *with* agraphia (central alexia), in which patients are also unable to write and sometimes have sensory aphasia as well. In pure alexia, the deficit is variable in that some patients can read individual letters, but cannot combine them to form words. The underlying lesion is most commonly caused by occlusion of branches of the left posterior cerebral artery and involves the occipital horn of the lateral ventricle.[4] It has been hypothesized that alexia without agraphia is a disconnection syndrome where the exchange of information between the visual cortex and the language center is disrupted.[6] Alternatively, it may represent an agnosia for lexical material.[7]

Cerebral achromatopsia

This is a rare acquired inability to discriminate colours. The term "cerebral achromatopsia" indicates complete loss of colour vision. "Cerebral dyschromatopsia," on the other hand, implies that some colour perception is present. Processing of colour vision takes place in both hemispheres and, therefore, cerebral achromatopsia involving the whole field of vision is possible only if lesions involve both hemispheres. Unilateral lesions in one hemisphere may produce colour loss in the hemifield opposite to the lesion.[8] Hemifield cerebral achromatopsia is the more common form. With a left hemispheric lesion, a right superior homonymous achromatic quadrantopsia is observed and this is usually associated with pure alexia. With a right hemispheric lesion, a left

superior homonymous achromatic quadrantopsia will be observed. Generally, the underlying etiology is an infarct in the territory of the occipitotemporal branch of the posterior cerebral artery.[4]

Occipitoparietal pathway disorders

Hemispatial neglect

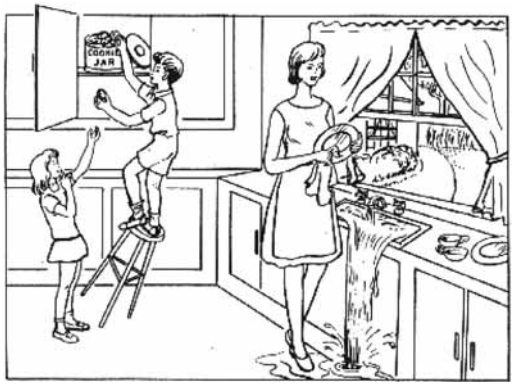
Hemispatial neglect is the inability to respond to stimuli presented contralaterally to a brain lesion that is not attributable to a sensory or motor defect.[9] This is lateralized to one hemispace. Hemispatial neglect affects all sensory modalities; auditory, tactile, and visual perception are all affected, although not always to the same extent. The visual component of hemispatial neglect can be mistaken for hemianopia; however, hemispatial neglect is body-centered or craniotopic; while hemianopia, in contrast, is visual field-centered or retinotopic.[10] Therefore, it is helpful to test the patient's visual fields in different directions of gaze to differentiate hemianopia from hemispatial neglect. For example, if a patient with left hemispatial neglect looks to the right, the area of neglect remains the same. On the other hand, if the patient with left hemianopia looks to the right, the blind left hemifield will shift to the right.[10] Hemispatial neglect is usually caused by unilateral lesions of the posterior parietal cortex; however, lesions in a variety of other locations (eg, dorsolateral frontal gyri, thalamus, and mesencephalic reticular formation) have also been reported to cause hemispatial neglect.[9] Hemispatial neglect is more severe and longer-lasting in patients with right-sided lesions.[11] This is explained by the hypothesis that the right cerebral hemisphere controls attention to stimuli presented in both hemispaces – left more than right – but the left hemisphere only controls attention to the contralateral hemispace. Accordingly, a right hemispheric lesion would eliminate attention to the left hemispace, whereas a left-sided lesion would still be compatible with some attention to right hemispace because of the intact right hemisphere.[10]

Balint's syndrome

In 1907, Rezso Balint reported a patient with "psychic paralysis of gaze," disordered spatial attention, and optic ataxia. This triad was named after him 47 years later.[12] The full syndrome consists of simultanagnosia, so-called "ocular motor apraxia," and optic ataxia.[13] Each of these elements, which can also be seen in isolation, are reviewed below:

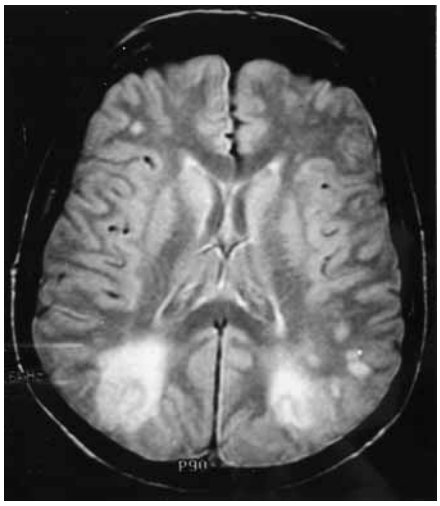
Simultanagnosia: This disorder is characterized by the inability to interpret complex visual arrays despite preserved recognition of single objects. It can be described as "piecemeal" vision. Patients are not able to detect more than one object at the same time and they often see with macular vision alone, which provides good visual acuity, but only captures a tiny fraction of the visual field. This deficit is easy to overlook unless the examiner specifically checks for it. This is easily achieved with the "Cookie Theft Picture" from the Boston Diagnostic Aphasia Examination kit (Figure 2). A patient with simultanagnosia is usually able to see only one person at a time and would not be able to tell that the sink is overflowing or that the boy is about to fall off the stool at first glance. Since the main defect is a failure to attend to more than one component of a visual array, the term "bilateral visual inattention" has been proposed instead of simultanagnosia.[10] Of the 3 elements in Balint's syndrome, simultanagnosia is most often seen in isolation.[14] In isolated simultanagnosia, interestingly, patients complain that stationary objects in the visual environment simply "disappear" from direct view. Rizzo and Hurtig[15] have documented that patients with simultanagnosia report intermittent disappearance of a target during fixation verified by electro-oculographic (EOG) recording. The authors relate this to a "defect of sustained attention." All of their patients had bilateral lesions of the superior visual association cortex.

Figure 2: Cookie Theft Picture from Boston Diagnostic Aphasia Examination for simultanagnosia testing



Optic ataxia: This is a striking impairment of coordinated voluntary hand movements in response to visual stimuli. The movements under proprioceptive control are spared. Optic ataxia can be confused with incoordination due to cerebellar or proprioceptive dysfunction. However, cerebellar and proprioceptive ataxias either remain unchanged or improve with visual guidance, while optic ataxia is *only* present with visually-guided limb movements. Optic ataxia, on the other hand, is *only* seen under visual guidance. In order to differentiate optic ataxia from other types of ataxia, the patient is asked to make a movement that requires proprioceptive input (eg, touching his ear or his other hand). If a patient has genuine optic ataxia, proprioceptive input markedly

Figure 3: Axial T2 weighted MRI of a patient with Balint's syndrome showing bilateral parieto-occipital lesions



improves the accuracy of the movement.[10] The diagnosis requires exclusion of motor, cerebellar, and somatosensory disturbances, apraxia, and visual field defects. Optic ataxia may be due to an inability to convert retinotopic (visual field-centered) coordinates into craniotopic (body-centered) coordinates, a conversion that is required to program a movement in response to a visual target and takes place in the posterior inferior parietal cortex. It may also be considered as a disconnection syndrome, with information from the visual cortex not reaching the motor cortex through the superior longitudinal fasciculus, an important pathway between the occipital and frontal lobes.[10]

Optic ataxia can be seen in isolation as well. In this scenario it may involve one or both hands and it may be present in one or both hemifields. Typically, patients are impaired in reaching with either hand for objects located in the visual hemifield contralateral to the lesion.

Impairment of visually-guided saccades (commonly known as ocular motor apraxia):

Previously termed "psychic paralysis of gaze," this eye movement disorder has, on occasion, been mistaken for a visual conversion reaction.[16] Patients with this condition are unable to voluntarily move their eyes toward an object even though they can move their eyes at random. This ocular motility defect has been referred to as "ocular motor apraxia" and, unlike congenital ocular motor apraxia, involves both horizontal and vertical eye movements.[17] However, in accord with the neurological definition of apraxia, it has been suggested that the term "ocular motor apraxia" be restricted to disorders of initiating voluntary saccades, when reflexive saccades and vestibular quick phases are intact.[18] A quantitative study has shown that the saccadic disorder in Balint's syndrome consists of inaccurate, misdirected, and delayed saccades to visual targets, whether volitional or reflexive. This supports the notion that there is no genuine saccadic apraxia in this disorder.[19]

Most cases of Balint's syndrome have bilateral lesions in the parieto-occipital regions. Figure 3 shows an axial T2 weighted MRI from a patient with Balint's syndrome secondary to posterior leukoencephalopathy that was caused by cyclosporine toxicity.

Akinetopsia

Akinetopsia is an acquired defect of motion perception. In 1983, Zihl et al[20] was the first to report a patient with defective motion perception due to bilateral cortical infarcts. These resulted from a sagittal sinus thrombosis that involved both parieto-occipitotemporal junctions, but spared the striate cortex. Patients with akinetopsia may complain that moving objects appear to jump from one spot to the next. Running water seems to be "frozen like a glacier." The disorder is diagnosed by verbal reports and impaired pursuit eye movements.[10] Akinetopsia has also been reported as a side effect to medication; Horton and Trobe[21] described 2 cases of akinetopsia secondary to use of nefazodone.

Diseases commonly associated with visual integration disorders

The visual integrative disorders discussed above are most commonly caused by a stroke involving the posterior cerebral artery or one of its branches, either unilaterally or bilaterally. Less common causes are tumours, intracerebral hemorrhage, trauma, demyelinating disease, infectious processes, and abscesses, and progressive multifocal leukoencephalopathy. In addition, posterior Alzheimer's disease and the Heidenhain variant of Creutzfeldt-Jakob disease (CJD) specifically affect visual association cortices.[14] Patients with posterior Alzheimer's disease, also called the visual variant of Alzheimer's disease, are relatively young at onset and, unlike most patients with Alzheimer's disease, they initially present to an ophthalmologist rather than a neurologist. CJD is a progressive dementia caused by prions, which are infectious proteins. Cortical visual loss is the predominant present-

ing symptom of the Heidenhain variant of this disorder. Prions may be resistant to standard surgical sterilization techniques and transmission of CJD via surgical procedures such as corneal grafts is well-documented, making early identification of the disease critical.[22]

Conclusion

Patients with visual integration disorders frequently present with vague complaints, such as, "I don't see well" or "Something is wrong with my eyes." Many make multiple visits to their optometrist and ophthalmologist, resulting in several changes to their eyeglass prescriptions. It is easy to see how some of these disorders may be mistaken for conversion reactions or even malingering. Making an accurate diagnosis of these disorders can be challenging and requires a high index of suspicion for some uncommon entities. Therefore, it is important for the ophthalmologist to be familiar with the different manifestations of these integrative disorders so that they can arrange appropriate investigations or referrals.

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Abstracts of Interest

Neural basis of prosopagnosia: an fMRI study.

HADJIKHANI N, DE GELDER B, BOSTON, MA.
Brain imaging research has identified at least two regions in human extrastriate cortex responding selectively to faces. One of these is located in the mid-fusiform gyrus (FFA), the other in the inferior occipital gyrus (IOG). We studied activation of these areas using fMRI in three individuals with severely impaired face recognition (one pure developmental and two childhood prosopagnosics). None of the subjects showed the normal pattern of higher fMRI activity to faces than to objects in the FFA and IOG or elsewhere. Moreover, in two of the patients, faces and objects produced similar activations in the regions corresponding to where the FFA and IOG are found in normal subjects. Our study casts light on the important role of FFA and IOG in the network of areas involved in face recognition, and indicates limits of brain plasticity.

Hum Brain Mapp. 2002;16(3):176-82

Deficits in cortical visual function.

STASHEFF SF, BARTON JJ, BOSTON, MA.
Lesions of extrastriate cortex cause selective defects in visual function. Damage to portions of the "ventral stream" in medial and inferior occipitotemporal cortex lead to impaired perception of color or various specific visual object recognition defects, such as prospagnosia, the inability to recognize familiar faces, and alexia, the inability to read. The latter must be distinguished from a variety of other reading defects related to primary visual, attentional, linguistic, or ocular motor impairments. Damage to the "dorsal stream" in lateral occipito-temporo-

parietal regions impairs visuospatial capabilities, leading to akinetopsia (impaired motion perception) or Balint's syndrome, a loosely bound triad of simultanagnosia, optic ataxia, and ocular motor apraxia. Topographagnosia can occur with ventral or dorsal lesions for different reasons. Considerable evidence has accumulated showing that residual vision or even "blindsight," which is visual perception in the absence of awareness, can persist after lesion of striate cortex in some patients.
Ophthalmol Clin North Am 2001;14(1):217-42.

Visual syndromes as the presenting feature of degenerative brain disease.

CASELLI JR.
The symptoms of a degenerative brain disease are dictated by its topography. Visuo-spatial impairment may be a severe and early feature of degenerative dementia. Visual symptoms in such patients are broadly divisible into dorsal and ventral visual syndromes, which result from a degenerative focus in occipito-parietal and occipito-temporal visual association cortices, respectively. The dorsal visual syndrome includes asimultanagnosia and Balint's syndrome. The ventral visual syndrome includes alexia and visual agnosia (prosopagnosia). Less often, hemineglect or visual field defects result. When Alzheimer's disease and Creutzfeldt-Jakob disease present in this way there is a topographic shift of neurodegenerative changes to posteriorly situated cortices. Patients with corticobasal ganglionic degeneration often develop symptomatic involvement of contiguous sensorimotor cortices causing mixed perceptual-motor syndromes. Even in patients with more typical patterns of dementia, the degree of visuo-spatial impairment may hinder driving skills, and the issue of driving should be addressed early in the clinical course.
Semin Neurol 2000;20(1):139-44.

Upcoming International Meetings

15-18 October 2005
Annual Meeting of the American Academy of Ophthalmology (AAO)
(Neuro-ophthalmology Subspecialty Day: October 15, 2005)
Chicago, Illinois
CONTACT: Email: meetings@aao.org
Website: http://www.aao.org/aao/annual_meeting/

25 February - 3 March 2006
32nd North American Neuro-Ophthalmology Society (NANOS) Annual Meeting
Tucson, Arizona
CONTACT: Email: ekunsey@nanosweb.org
Website: http://www.nanosweb.org/meetings/nanos2006/

University of Toronto Department of Ophthalmology and Vision Sciences

Upcoming events

October 13, 2005	**World Sight Day**
October 15-18, 2005	**American Academy,** Chicago, Illinois
October 27, 2005	**VPP** – Dr. Emmett Cunningham, New York, N.Y. "Treatment Principles in Uveitis"
November 3, 2005	**Faculty Research Day**, Vaughan Estates
November 10, 2005	**VPP** – Dr. Emily Chew, Bethesda, Maryland Nutrition Supplement for AMD
November 17, 2005	**VPP** – Dr. Guillermo Rocha, Brandon, Manitoba New Trends in the Management of Keratoconus
November 24, 2005	**VPP** – Dr. Timothy Murphy, Boston, Massachusetts "Research Ethics"
December 2-3, 2005	**Walter Wright Day** The Old Mill, Toronto, Ontario "Eye Care: What Works? What Doesn't?" Contact: Jan Spencer (416) 978-1617
December 8, 2005	**VPP** – Dr. Jayne S. Weiss, Detroit, Michigan "The FDA Ophthalmic Devices Panel – The inside story"
January 28, 2006	**2006 Toronto Cataract Course**

Note: This year's (September 2005 to May 2006) VPP rounds will be held at:
The Hospital for Sick Children
555 University Avenue, Toronto
Main Auditorium, Elm Wing,
1st Floor, Room 1246, 5:30PM – 7:30PM.

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