

Abnormalities in higher cortical visual processing

Abstract

Normal visual processing includes signal stream from the retina via the lateral geniculate nucleus to the striate cortex. Extra-striate visual cortex performs the processings involving color and motion perception. Thus, the striate occipital lesions cause deficits affected the visual field while as extrastriate lesions cause deficits related to the perception of color and motion. Extra-striate cortical regions include ventral occipitotemporal and dorsal occipitoparietal streams. Ventral stream lesions may produce defects such as agnosia, prosopagnosia, alexia, and achromatopsia while dorsal stream lesions can cause akinetopsia and Balint syndrome. Ophthalmologists have often difficulties in understanding the higher cortical visual deficits and, so they usually ignore them. The main purpose of this review article is to summarize the higher cortical and visual dysfunctions and to facilitate the understanding of these.

Keywords: abnormality, high cortical, visual, high order, extra-striate, processing, function, deficit, cognitive blindness, gnosis, recognition, graphia, writing, praxia, motion, lexia, reading, phasia, speech, nomia, aphasia naming ability

Volume 8 Issue 4 - 2018

Burak Turgut,¹ Feyza Çaliş Karanfil,¹ Fatoş Altun Turgut²

¹Department of Ophthalmology, Yüksek İhtisas University, Turkey

²Elazığ Training and Research Hospital, Turkey

Correspondence: Feyza Çaliş, Assistant Professor of Ophthalmology, Yüksek İhtisas University, Faculty of Medicine, Department of Ophthalmology, 06520, Ankara, Turkey, Tel +90 312 2803601, Fax: +90 122803605, Email feyzacalis@gmail.com

Received: May 23, 2018 | **Published:** July 11, 2018

Introduction

Sixty percent of the human brain is formed by visual pathways and high visual centers. Thirty visual centers work normally in concordance with systematic and constant communication with each other. Visual cortical areas include primary and secondary areas. Normal visual processing includes the signalization and neuronal processing initiated from the retina through the lateral geniculate body to the striate cortex.^{1,2} Visual cortical areas include Broadman-17 (primary visual cortex, area striata, visual area V1) area related to shape and size of the objects in striate cortex at lobus occipitalis; Broadman-18 (area parastriata, prestriate cortex, visual area V2) area related to the analysis of object motion in parastriate cortex at lobus occipitalis; Broadman-19 associated with visual area V3 in posterior parietal lobe related to visual integration and cortical color vision; V4 and V5 areas in superior temporal sulcus, related to motion perception (M cells) input, direction and depth perception; visual area V6 in parietal cortex associated with extra-personal perception.^{2,3} Lesions in visual areas 18 and 19 are associated with visual agnosia. The injury in left may be associated with pure alexia. Defects in the posterior parietal cortex cause optic ataxia while the damage to the medial supero-temporal cortex and medial temporal visual cortex results in loss of visual motion perception (akinetopsia) in different directions. The damage to the inferotemporal cortex causes visual agnosia. Damage to V4 results in loss of color vision, achromatopsia, while damage to V6 causes an inability to distinguish two-dimensional patterns.⁴⁻⁶

Secondary visual areas play a role in evaluating the impulses from the primary visual center and performing higher-level functions such as analyzing the shapes of objects, observing moving objects, distinguishing of different hues of the same colors.^{2,3} The primary visual cortex is located almost entirely on the medial surface of the occipital lobe; a portion of about 1cm extends around the posterior pole onto the lateral surface. The calcarine fissure travels from the parieto-occipital sulcus to the posterior pole, separating the visual cortex into an upper part (the cuneus gyrus) and a lower part (the lingual gyrus); most of the primary visual cortex is buried in the tissue within the calcarine fissure.^{2,3,7} Extra-striate visual cortex is responsible for processing regarding color and motion perception. Extra-striate cortical regions include ventral occipitotemporal and dorsal occipitoparietal streams. The striate occipital lesions cause

visual field deficits while as extra-striate lesions cause deficits related to the perception of color and motion.¹⁻³

Ventral stream

This stream travels below the calcarine fissure into the medial lower temporal lobe similar to the visual library and the ventral occipitotemporal stream is functioned in the object recognition and it carries the visual information that gives the answer to the question “what do we see?”. Cortical visual projections from primary visual cortex to the inferotemporal cortex is related to the identification of properties like shape and color. The ventral stream projects from V1 to the temporal lobe. The function of ventral stream is to perceive or identify or recognize an object or a pattern or a specific face and the coding of this information for storage and for use in cognitive processes such as imagining, planning, and recognition. Ventral information from V1 uses a way directly visual association areas V2 (included in the processing of simple properties such as orientation, color, and spatial frequency), V4 (included in the orientation, spatial frequency, color, and object shape recognition), and ultimately to the inferior temporal lobe. fMRI studies in normal people demonstrated signal increasing in the ventral stream while identification form, texture, color or recognizing objects and faces.¹⁻⁴ Ventral stream lesions may produce defects such as object agnosia, prosopagnosia, alexia and achromatopsia.^{4-6,8,9}

Dorsal stream

The stream of cortical visual projections from primary visual cortex to the superior posterior parietal cortex, concerned primarily with the visual control of action. The dorsal stream arrives into the occipitoparietal and temporo-parieto-occipital areas which locate superolaterally from the striate cortex. The dorsal stream projects from the primary visual cortex (V1) to the posterior parietal lobe, carry instant information about the objects location and play a role in the visual control of skilled movements diverted to this objects. The occipitoparietal stream provides the spatial processing and the answer to the question “where do we see?”. The ‘where’ stream is carried dorsally, to medial temporal and posterior parietal cortices, and carries information about an object’s location and motion, including one’s own body and its spatial relationship to an object (visual guidance and reaching movements). The dorsal stream lesions

can cause selective impairment of motion perception, akinetopsia, and Balint syndrome.^{2,6,10} Visual association cortex is a sophisticated area in which top-level processing is performed and in which is located in the temporal lobe. It provides the perception of the object which was seen.^{2,3}

Angular gyrus

It is located at parietal lobe and related to the writing and reading the objects name which was seen.^{2,3,11}

Corpus callosum

In the vision, our main sensory system, the corpus callosum serves to bind together the separate representations of the two halves of the visual field. One key role of the callosum is to combine these two partial cortical maps of the visual field into a single, coherent representation.^{2,12}

Inferotemporal cortex

It is located in inferior surface of the temporal lobe and particularly important for object recognition.^{2,3} Blindsight (cortical blindness) (CB) is a form of total or partial bilateral vision loss in a normal-appearing eye resulted from a damage in the visual center of primary visual cerebral cortex in the brain and a pathway which travels from the magnocellular cells in lateral geniculate body directly to the thick stripes of V2, providing input to the dorsal stream. Thus, actually, primary involved area in CB is V1, however, the extra-striate cortex is also often damaged. So, here, its definition will be appropriate for the understanding the higher visual cortical dysfunctions. The causes of acute and permanent CB are anoxia, traumatic brain injury, infections or metastatic tumors, or the operations for their removal, massive infarcts or hemorrhage located in the visual occipital cortex. However, the most common cause is stroke resulting from the posterior or middle cerebral arteries. Additionally, migraine, mild head trauma, brief episodes of hypoglycemia or hypotension, and benign occipital epilepsy may cause transient cortical blindness, especially in children. The main feature of cortical blindness is the loss of vision with preservation of the pupillary light reflex. The fundoscopic examination is normal. Patients with CB have often healthy anterior and posterior segment structures and optic discs and normal pupils, best-corrected vision is at or less than the level of counting fingers. The abilities of motion and light perception are usually preserved (Riddoch phenomenon), but static objects remain undetected. Vision loss is usually sudden. In some patients, cortical blindness may be present with Anton or Charles-Bonnet Syndrome.^{5,6,9,10}

Anosognosia refers to a patient's inability to recognize consciously the presence of somatic dysfunction indicative of a disease process. Patients with left hemiplegia caused by stroke may be entirely unaware of their deficit. Anosognosia generally happens predominantly, by non-dominant parietal cortex lesions and is accepted more a disorder of caution than perception.¹⁴ Alexia is the loss of pre-existing normal reading ability due to a brain disorder. The subject cannot read a whole word or all of the sentence though he/she recognizes individual letters. In alexia, there is a connection failure in the stimulations transmitted from visual cortex to the left angular gyrus or the left angular gyrus.¹⁵

Alexia without agraphia (agnosic alexia, pure alexia, alexia without agraphia, pure word blindness)

Pure alexia is a very rare abnormality. The patient can talk, understand and write. However, he/she cannot understand a written text or cannot copy a written word. Pure alexia occurs in the lesion of

the connections from both visual cortexes to dominant angular gyrus. The right hemianopia always associates to this abnormality. Although pure alexia usually occurs in the occlusion of the posterior cerebral artery, it can also exist in hemorrhage, arteriovenous malformation, herpetic encephalitis and multiple sclerosis. In this pathology, the subject has the inability to recognize the words visually. In the left occipital lobe lesions including splenium of corpus callosum, alexia is with contralateral hemianopsia but not agraphy.^{6,9,15}

Alexia with agraphia

Alexia is more common with agraphy. The person has no abilities of both reading and writing using both hands. The lesion is in the dominant angular gyrus and the abnormality usually occurs in the events of the parietal cortical branch of the middle cerebral artery. It is usually with nominal aphasia, acalculia, and hemianopsia. In the left angular gyrus lesions, alexia is with agraphy.¹⁵ Agraphia is the impairment of pre-existing normal writing ability due to a brain disorder. It is the common feature of all aphasia syndromes.^{6,9} Hemialexia is defined as the inability to read the words presented in one of the two visual hemifields, especially in the left visual field, in absence of hemianopia. It may occur after callosotomy (complete or partial involving only the splenium and represents a visual disconnection syndrome. The disconnection hypothesis for pure alexia requires two disconnections of the left angular gyrus, one for each hemifield. In left hemi-alexia, reading is impaired in the left hemifield only, because of isolated damage to the splenium or the callosal fibers elsewhere. Right hemi-alexia has been reported with a lesion of the left medial and ventral occipital lobe. Left hemiparalexia is a rare syndrome reported with splenial damage after surgery for arteriovenous malformations. Subjects make substitution and omission errors for the first letter of words, much like neglect dyslexia, but they do not have hemineglect and have left-sided lesions with right hemianopia rather than the converse.¹⁵

Optic ataxia is characterized by a deficit in the visual control of the direction of arm reaching to visual target or in the visually guided arm movements accompanied by defective eye-and orientation and grip formation. It occurs with the lesions in posterior parietal cortex (PPC), superior parietal lobule (SPL) and areas around the intraparietal sulcus (IPS). The inability cannot be explained by primary motor, somatosensory defects or the defects in visual field or acuity. The defect is at a more integrative sensorimotor level. The subjects with ocular ataxia can recognize the object but cannot reach and grasp the object in the contralesional visual field. It is a component of Balint's syndrome.¹¹⁻¹⁶ Alonia is inability to identify words.⁹ Prosopagnosia Inability to recall the name of a person presented visually. A selective deficit of naming faces. The person is able to recognize the famous person whose his photo is shown but not able to name him/her.^{17,18}

Visual agnosia

In this condition patients can not able to identify visually presented objects although elementary sensory functions are protected. For the diagnosis we have to evaluate the patients ability to name, describe uses for, and pantomime the use of visually presented objects. The main reason of this disease is the damage of the occipital cortex because of anoxia or severe infarction. The subject can recognize the localization and position of an object but cannot name or a copy draw it. Damage of the connecting pathways between occipital and parietal or temporal lobes of the brain causes visual agnosia. It is usually seen in the bilateral lesions of visual cortical areas of association (areas 18 and 19). The patient cannot recognize or name an object he or she

sees, even if it is enough to see it. The patient with visual agnosia can also name the object with touching which priorly he/she could not name with viewing (Table 1). The reason of the agnosia is usually the damage of the parietal and temporal lobes. Strokes, encephalitis or traumas may cause this damage. Prosopagnosia, pure alexia, and

topographagnosia are the selective agnosias. The parietal and temporal lobes allow you to understand the meaning of this information. Visual agnosia is usually associated with unilateral or bilateral homonymous hemianopsia, and it occurs occipital or parieto-occipital lesions.^{4,8,14,19}

Table 1 The common abnormalities of higher visual cortical processings and lesion regions

High cortical visual abnormality	Clinical ability/inability	Possible pathology region	Associated features
Alexia without agraphia (Agnosic alexia, pure alexia)	Writable but unreadable Focal lesions of the brain have resulted with reading disorder	Contralateral occipital lobe and corpus callosum	Ipsilateral homonym hemianopsia
Alexia and agraphia	Unwritable and unreadable Writing disorder related to focal or diffuse pathologies of the brain	Contralateral parietal lobe and angular gyrus	None
Cerebral diplopia (Polyopia)	Visual perception of more than one image of a single object	Right occipital lobe	None
Peripheral visual dyslexia	Reading inabilities due to visual inability	Cerebral Lesions	Homonymous visual field defects
Palinopsia (visual perseveration)	Visual image persists or recurs after the visual stimulus has been removed	Focal cerebral lesions involving in the right hemisphere	
Astereognosis	Perception of the position of objects in depth is another type of spatial computation	Bilateral occipitoparietal lesions	
Astereopsis (stereoblindness)	Impaired binocular depth perception	Occipitoparietal lobes	
Hemichromatopsia	Unability of color vision in hemi-field	Contralaterale occipital-temporal lob fusiform and lingual gyrus (V4)	Ipsilateral homonym upper quadransopia
Visual agnosia	Inability to identificate objects without visual impairment and demans	Bilateral occipital-temporal lob inferior long fasc.	Alexia without agraphia, prosopagnosia
Visual anosognosia (denial of loss of vision)	Not able to see but deny it	Parietal white matter injury leading to a disconnection syndrome	Which is associated with confabulation in the setting of obvious visual loss and cortical blindness
Anton Syndrome	Not able to see but deny it	Bilateral occipital lobe	Bilateral vision loss
Prosopagnosia (face blindness)	Inability to recognize familiar faces	Bilateral occipital-temporal lobe mid-fusiform gyrus	Alexia without agraphia, visual agnosia
Akinetopsia(motion blindness)	Inability to recognize motion	Bilateral lateral occipital-temporal lobe (V5)	

Table Continued

High cortical visual abnormality	Clinical ability/inability	Possible pathology region	Associated features
Neglecton	Inability to report, respond, or orient to external visual stimulation or mental images of objects and scenes that are positioned contralateral to the brain lesion	Contralateral inferior parietal lobule	Ipsilateral sensory and motor deficit
Simultanagnosia	Inability to perceive the field of vision as a whole	Bilateral parietal-occipital lobe	Bilateral inferior altitudinal visual field defects
Balint Syndrome	Simultanagnosia	Bilateral parietal-occipital lobe	Ocular apraxia, optical ataxia
Ocular apraxia	Inability to perform previously learned motor skills without any motor deficit, incoordination, sensory deficit, misunderstanding or carelessness	Bilateral parietal-occipital lobe	
Optical ataxia	An impaired visual control of the direction of arm reaching to a visual target,	In humans, optic ataxia is associated with lesions of the superior parietal lobule (SPL), which also affect visually guided saccades and other forms of eye-hand coordination. Bilateral parietal-occipital lob	Accompanied by defective hand orientation and grip formation. a component of Balint's syndrome
Optical aphasia	Inability to name visually presented objects	Probable functional disconnection between visual perception and language systems	Large left PCA infarction, right homonymous hemianopia
Topographagnosia	The inability to orient oneself in one's surroundings as a result of focal brain damage.	Right hemisphere lesions	
Alonia	Alonia is inability to identify words	Dominant occipital cortex and adjacent temporal and parietal cortex	Homonym hemianopia, visual object agnosia
Achromatopsia-Cerebral (Central) Dyschromatopsia	Cerebral dyschromatopsia is impaired color perception due to an acquired brain lesion. In achromatopsia, there is the complete absence of color perception.	Damage of V4 area	Subjects with achromatopsia complain that everything appears in shades of gray
Prosopagnomia	Inability to recall the name of a person presented visually. A selective deficit in naming faces		The person is able to recognize the famous person whose his photo is shown but is not able to name him/her
Charles-Bonnet Syndrome	Cortical blindness but patient is unaware of the blindness or deny it	Occipital cortex and other cortical regions are affected	
Visual hallucinations	Perception of a non-existent image by the patient	Irritation of the primary visual cortex or visual association cortices	

High cortical visual abnormality	Clinical ability/inability	Possible pathology region	Associated features
Peduncular hallucinosis	Vivid hallucinations	Lesions of the mesencephalon and thalamus	Vivid, colorful, and sometimes distorted images of animals and people
Visual allesthesia	The transposition of visual images from one-half of the visual field to the other	Right hemisphere lesions	
Visual Distortions	Seeing the images as deformed or skew/crooked	Brainstem lesions	Wallenberg syndrome, vertebral artery dissection, following surgery of the third ventricle
Hemialexia	Inability to read the words presented in one of the two visual hemifields	Lesions of the splenium, callosal fibers or medial and ventral occipital lobe	Subjects make substitution and omission errors for the first letter of words and have left-sided lesions with right hemianopia rather than the converse
Cortical blindness	Total or partial bilateral vision loss in a normal-appearing eye	Primary visual cerebral cortex	Sometimes present with Anton or Charles-Bonnet Syndrome

Apperceptive visual agnosia

A form of visual agnosia in which a person cannot reliably name, match, or discriminate visually presented objects, despite adequate elementary visual function (visual fields, acuity, and color vision). Apperceptive visual agnosia causes difficulty in assembling parts of an image into an understandable whole. “Apperceptive” form that is caused by impaired visual processing that results in the poor perception of the object. This condition may cause you to have difficulty in understanding the relationship between objects. You may, for instance, try to copy a picture of a circle and end up drawing a series of concentric scribbles. You can still use vision to navigate your environment and pick up objects without trouble. Apperceptive visual agnosia is usually caused by lesions of the parietal or temporal lobes on both sides of the brain. Patients with severe apperceptive agnosia usually have extensive and diffuse occipital lesions and tend to have residual field defects.^{8,14}

Associative visual agnosia

A form of visual agnosia in which a person cannot use the derived perceptual representation to access stored knowledge of the object’s functions and associations but able to copy and match the drawing even though unable to identify it. Associative visual agnosia is the inability to recall information associated with an object. This can include an object’s name, use, or origin. This form of agnosia does not prevent you from being able to draw a picture of an object. You may be unable to name the object in the drawing. You could recognize and use an object shown to you but may be unable to say what the name of the object is. The reason of the “associative” form is the lesions of the associative cortex and it causes inability to match the correctly

formed visual percepts with previously committed sensory data and identification.^{8,14,19}

Integrative visual agnosia

A form of visual agnosia in which one retains the ability to recognize elements of objects but unable to integrate these elements together into comprehensible percept.^{8,14,19}

Prosopagnosia (face blindness)

A form of visual agnosia in which a subject cannot recognize familiar faces or the faces of people which whom he/she already knew and cannot learn these, despite adequate elementary visual function (visual fields, acuity, and color vision). It occurs in bilateral lesions of inferior and medial visual association cortex. Prosopagnosia is caused by lesions in the fusiform face area in the brain which recognizes faces.^{20,21} Simultanagnosia is characterized by the inability or difficulty in perceiving more than a single object at a time caused by bilateral occipitoparietal defects. It presents bilateral inferior altitudinal visual field defects.^{22,23}

Ventral simultanagnosia

A decrease in the ability to identify multiple visual stimuli quickly, so identification continues in part-by-part mode.^{22,24}

Dorsal simultanagnosia

An inability to detect more than one object at a time, with difficulty shifting attention from one object to another.^{22,23,25} Balint’s syndrome is agnosic syndrome caused by large bilateral occipital and parietal lesions and composed of optic ataxia (incompetence in moving a

hand to an object looking at), oculomotor apraxia (difficulty in fixing eyes) and simultanagnosia (inability to perceive the field of vision as a whole). Subjects have a deficit in attention, in that they cannot attend to more than one or at most a few objects at a time. Thus they have trouble with visual search tasks maintaining attention over large regions of space, and cannot count the number of objects present. Simultanagnosia play a key role in this syndrome. Patients with this syndrome cannot recognize multiple objects at the same time. Balint Syndrome usually develops in vascular pathologies, sudden and severe hypotension, encephalopathies and hypoxia.^{26,27} Selective impairment of motion perception is a rare condition which caused by the injury of the areas of extrastriate visual cortex analogous to V5, that is, damage of the both lateral occipitotemporal areas. Patients may be unable to perceive the fast motion or motion in depth. Rapid objects seems to jump rather than move. Patients may have difficulties about recognizing the direction and speed of cars.^{28,29}

Optical aphasia

A condition in which a person cannot name a visually presented object, despite being able to indicate the identity of the object through gesture and sort the visual stimuli into categories. In optic aphasia, patients are unable to name visually presented objects but otherwise show relatively intact knowledge about objects and are, thereby, able to categorize and demonstrate their use through pantomime. Patients typically present with large left PCA territory infarction with right homonymous hemianopia. It has been suggested that there is a functional disconnection between visual perception and language systems.^{30,31} Topographagnosia (Topographical disorientation) is difficulty in managing the spatial layout of an environment and losing in familiar places or the inability to navigate or to orient in an environment oneself resulting from the focal brain damage. The patients are unable to find their way and fail to recognize landmarks and they lost internal map or sense of direction. Lesion is usually in dominant occipital cortex and adjacent temporal and parietal cortex, especially dorsal convexity of the right parietal lobe.³²

Hemiachromatopsia

It is impaired discrimination of colors in the contralateral hemifield. Hemiachromatopsia is loss of color limited to the contralateral hemifield. Typically it is asymptomatic until the defect is demonstrated. Hemiachromatopsia is usually associated with a homonymous superior quadrantanopia, and so the color defect is only demonstrable in the lower quadrant. Two rare cases with color defects limited to one quadrant have been described. It is not clear whether these were true chromatic defects or subtle relative scotomata, but the quadrant representation of the human V4 area on functional neuroimaging suggests that a quadrant dyschromatopsia is theoretically possible.^{33,34}

Central Cerebral dyschromatopsia (Achromatopsia)

This abnormality is impaired color perception due to an acquired brain lesion. In achromatopsia there is complete absence of color perception. Subjects with achromatopsia complain that everything appears in gray color or its hues, black or white. The lesion is in the V4 region of the brain. It's the inability to name colors despite being able to perceive them. Color anomia occurs when a lesion separates the V4 regions of the brain from the language areas. If dyschromatopsia is with superior quadrantanopia and prosopagnosia, it is possible that there are lesions in the inferior occipital lobes in both hemisphere.³⁴

Peripheral dyslexia

It is the reading inabilities due to visual inability, or/and the failure of ocular motility in cerebral lesions. Hemianopic dyslexia is a frequent and disabling functional impairment following the brain injury. In this form of peripheral dyslexia patients have acquired impairment in reading. Patients who had homonymous visual field defects have problems about reading in spite of having normal language functions.³⁵ Palinopsia (visual perseveration) is the abnormality in which a visual image persists or recurs after the visual stimulus has been removed. Palinopsia is an usual visual symptom which lasts typically for several days to a few weeks. It usually occurs often in focal cerebral lesions involving in the right hemisphere such as cerebral infarct, infection, stroke, trauma, arteriovenous malformation, tumor, parasite, migraine, seizure activity, drug use and as a result of the ictal phenomenon in association with the signs of the parietooccipital and occipital lobes in opposite side.³⁶⁻³⁸ Cerebral diplopia (Polyopia) is the visual perception of more than one image of a single object especially with one eye even after removal of an object from the visual field after fixation on a stimulus. Although it occurs in the lesions of right occipital lobe, its exact etiopathogenesis and lesion region is not known. The appearance of many of the same images while watching a single object is called palinopsia, which is a phenomenon distinct from polyopia.^{36,38}

Akinetopsia (motion blindness), a very rare higher visuospatial deficit, is the inability or difficulty in perceiving the motion. The human visual motion area (V5/MT) has been identified to lie in the lateral cortex at the junction of the occipital, parietal, and temporal lobes. In the literature there is only two cases who had akinetopsia because of bilateral damage have been reported.^{39,40} Astereognosis is perception of the position of objects in depth is another type of spatial computation. The most well-known process that is involved in depth perception is stereopsis. Two objects located at different distances from the observer have a different relationship to each other in the retinal image of the right versus the left eye. The power of this depth cue can be appreciated by trying to thread a needle with one eye closed and then with both eyes open. Loss of stereopsis can occur with bilateral occipitoparietal lesions, and can be demonstrated with common tests of stereovision used in eye clinics, such as the Titmus stereo fly test or Randot Stereo Test, which require the patient to wear polarized glasses and present slightly different images to each eye. Stereopsis is not the only cue to depth relationships, to which any one-eyed person or painter can attest. Relative size and saturation are strong pictorial cues to distance; moving one's head can give depth cues from optic flow patterns and motion parallax. It is not known whether the cerebral lesions that cause astereopsis also affect the ability of a patient to derive depth from these monocular cues.⁴¹ Astereopsis (stereoblindness), the impaired binocular depth perception, is one of the important clues to distance from the observer is the disparity between the retinal images of the object in the two eyes. It may occur as a developmental disorder or acquired disorder following a neurological disease affecting bilateral occipitoparietal lobes. However, unilateral lesions can cause milder deficits. Astereopsis can be diagnose with stereo tests including some cards with different polarized or colored glasses.⁴²

Visual neglect

Visual neglect refers to the failure of a patient to report, respond, or orient to external visual stimulation or mental images of objects and scenes that are positioned contralateral to the brain lesion, which

caused neglect (i.e. contralesional). Further, the failure is not due to a primary sensory or motor deficit such as hemianopia or paralysis. This entry describes neglect because of its effects on visual processing, with an emphasis on its assessment, types, spatial dimensions, causes, and treatment. It is important to note that what can be said about visual neglect can also be said about neglect in other sensory modalities. Almost every time left hemifield is affected by neglect. Because the left hemisphere observes the right hemifield, but the right hemisphere observes both hemifields. A left hemisphere lesion will still allow the right hemisphere to survey the entire visual field, hence neglect does not occur. By contrast, with a right hemisphere lesion, the left hemisphere monitors only the right hemisphere; therefore because of the lack of monitoring the left hemisphere, left-sided neglect occurs.⁴³

Visual hallucinations (visual gain) is a positive phenomenon defined as the perception of a non-existent image by the patient and it is a strong indicator of an organic disease such as the acute confusional state and Lewy body disease. However, some drugs such as atropine are the most common causes of visual hallucinations. These may be lights, lines, light flashes, or more complex images such as people, animals, figures, or landscapes. Visual hallucinations can occur due to psychophysiological (a disturbance of brain structure), psychobiochemical (a disturbance of neurotransmitters), and psychodynamic (an emergence of the unconscious into consciousness) causes. Irritating (e.g., seizure activity) the visual processing cortical areas brings out the first reason. Irritation of the primary visual cortex (Brodmann's area 17) causes simple elementary visual hallucinations, while irritation of the visual association cortices (Brodmann's areas 18 and 19) causes more complex visual hallucinations.^{44,45}

Charles Bonnet syndrome (CBS) includes nonthreatening hallucinations in patients who have no neurological and no psychological abnormalities. Patients develop vivid visual hallucinations in the absence of psychiatric illness but with significant visual impairment secondary to ocular disease, such as macular degeneration and diabetic retinopathy. Because of the fear of a mental illness being diagnosed, patients are often reluctant to discuss these hallucinations. The images tend to be complex (animals, people) and insight is usually retained. In the great majority of cases, a decline in visual acuity precedes the development of CBS. Patients may realize that the images are hallucinations, not real by the normal reality tests.⁴⁶ Peduncular hallucinosis describes vivid hallucinations first described by Lhermitte in 1922 and which are animated and spooky and often accompanied by gaze paresis in both vascular and infective lesions of the mesencephalon and thalamus. These tend to occur in the evenings and to disappear over weeks. The visions are usually reported to be vivid, colorful, and sometimes distorted images of animals and people. They are typically considered non-threatening by the patient.^{47,48}

Visual distortion

The subjects see the images as deformed or skew/crooked. Visual distortion includes micropsia, macropsia, metamorphopsia and rarely upside-down reversal of vision. In the later, images are seen in fully upside down position for a few seconds to 12 hours and it can occur in Wallenberg syndrome, vertebral basilar ischemia, vertebral artery dissection, following surgery of third ventricle and brainstem lesions.⁴⁹ Visual alliesthesia (optical alliesthesia) is the transposition of visual images from one-half of the visual field to the other in association with sustained neural activity in the contralateral parietal cortex. The objects on the healthy side seem on the defective side. Greek

allache: elsewhere and aisthesis: perception. This dysfunction is a rare condition experienced by patients with injury or tumors compressed to temporal or occipital cortex especially in right side or parieto-occipital arteriovenous malformation, occipital calcified cysticercus, occipital infarct, malformation, and Epilepsia partialis continua. The visual images may also rarely be transposed between the vertical visual hemifields instead of horizontal hemifields. Visual alliesthesia is often with palinopsia. Pathophysiology of visual alliesthesia still remains unknown. Visual alliesthesia usually results from right hemisphere lesions with left-sided neglect or extinction. Hallucinations from temporal and occipital lesions may be distinguish from visual alliesthesia with the association to the parietal localization.⁵⁰⁻⁵²

Anton's Syndrome The ability to recognize visually presented objects and words depends on the integrity not only of the visual pathways and primary visual area of the cerebral cortex (area 17 of Brodmann) but also of those cortical areas that lie just anterior to area 17 that is areas 18 and 19 of the occipital lobe and area 39—the angular gyrus of the dominant hemisphere (visual association areas). Bilateral infarction in the distal PCAs produces cortical blindness (blindness with preserved pupillary light reaction). The patient is often unaware of the blindness or may even deny it (Anton's syndrome). Tiny islands of vision may persist, and the patient may report that vision fluctuates as images are captured in the preserved portions. Rarely, only peripheral vision is lost and central vision is spared, resulting in 'gunbarrel' vision. Although cerebrovascular disease is the most common cause, surgery, particularly cardiac surgery and cerebral angiography are also major causes. Anton's syndrome describes the condition in which patients deny their blindness despite objective evidence of visual loss, and moreover confabulate to support their stance. This type of cortical blindness is a rare condition in which both occipital cortex and other cortical regions are affected and patients think that they have sight. Although the anterior visual tracts are intact, the visual association centres in the occipital cortex may be compromised. In this syndrome patients think, say and behave as they have a sight however they cannot. Attention to the possibility of the condition is, however, drawn when they walk into walls, fall over furniture and describe objects that are not present.⁵³⁻⁵⁵ Visual anosognosia, or denial of loss of vision, which is associated with confabulation in the setting of obvious visual loss and cortical blindness is known as Anton's syndrome. Bilateral occipital brain damage results in blindness; however, patients start to confabulate to fill in the missing sensory input. Although visual anosognosia is frequently believed to represent cortical phenomenon, it is probably more often caused by parietal white matter injury leading to a disconnection syndrome.⁵³⁻⁵⁵

Authorship contributions

Concept and Design: Burak Turgut, Feyza Çalış Karanfil, and Fatoş Altun Turgut; Data Collection, Processing, Analysis, Interpretation: Burak Turgut and Feyza Çalış Karanfil; Literature Search: Fatoş Altun Turgut; Writing: Burak Turgut and Feyza Çalış Karanfil.

Financial disclosure

The authors declared that they received no financial support for the research, authorship, and/or publication of this article.

Acknowledgements

None.

Conflict of interest

No conflict of interest was declared by the authors.

References

- Michael Titus AT, Revest P, Shortland P. *The Nervous System*. 2nd ed. London; Elsevier-Churchill-Livingstone; 2010:121–140.
- Remington LA, Visual Pathway. *Clinical Anatomy and Physiology of the Visual System*. 3rd ed. Butterworth-Heinemann; 2012:233–252.
- Jacobson S, Marcus EM. Neuroanatomy for the Neuroscientist. Higher Cortical Functions. In: Neuroanatomy for the Neuroscientist. Boston, MA: Springer; 2008:375–395.
- Greene JD. Apraxia, agnosias, and higher visual function abnormalities. *J Neurol Neurosurg Psychiatry*. 2005;76(Suppl 5):25–34.
- Barton JJ. Disorders of higher visual processing. *Handb Clin Neurol*. 2011;102:223–261.
- Barton JJ. Higher cortical visual deficits. *Continuum (Minneapolis)*. 2014;20(4 Neuro-ophthalmology):922–941.
- Kansu T. Beyin ve Görme. *Turk J Neurol*. 2004;10(2):85–91.
- Biran I, Coslett HB. Visual agnosia. *Curr Neurol Neurosci Rep*. 2003;3(6):508–512.
- Goodwin J. Disorders of higher cortical visual function. *Curr Neurol Neurosci Rep*. 2002;2(5):418–422.
- Melnick MD, Tadin D, Huxlin KR. Re-learning to see in cortical blindness. *Neuroscientist*. 2016;22(2):199–212.
- Andersen RA, Andersen KN, Hwang EJ, et al. Optic ataxia: from Balint's syndrome to the parietal reach region. *Neuron*. 2014;81(5):967–983.
- Seghier ML. The Angular Gyrus: Multiple Functions and Multiple Subdivisions. *Neuroscientist*. 2013;19(1):43–61.
- Pietrasanta M, Restani L, Caleo M. The Corpus Callosum and the Visual Cortex: Plasticity Is a Game for Two. *Neural Plast*. 2012;2012:10.
- Baugh LA, Desanhere L, Marotta JJ. Agnosia In: Koob G, Moal ML, Thompson R, editors. *Encyclopedia of Behavioral Neuroscience*, Academic Press; London; Elsevier; 2010;1:27–33.
- Benson DF, Geschwind N. The alexias. In: *Handbook of Clinical Neurology*. Vinken PJ, Bruyn GW, editors. North-Holland, Amsterdam, 1969;4:112–114.
- Mayer AB, Caminiti R. Optic ataxia as a result of the breakdown of the global tuning fields of parietal neurones. *Brain*. 2002;125(Pt 2):225–237.
- Semenza C, Sartori G, D'Andrea J. He can tell which master craftsman blew a Venetian vase, but he can not name the Pope: a patient with a selective difficulty in naming faces. *Neurosci Lett*. 2003;352(1):73–75.
- Carney R, Temple CM. Prosopagnosia a possible category-specific anomia for faces. *Cognitive Neuropsychology*. 1993;10(2):185–195.
- Baugh LA, Desanhere L, Marotta JJ. Agnosia In: Reference Module in Neuroscience and Biobehavioral Psychology. Elsevier. 2017.
- Tohgi H, Watanabe K, Takahashi H, et al. Prosopagnosia without topographagnosia and object agnosia associated with a lesion confined to the right occipitotemporal region. *J Neurol*. 1994;241(8):470–474.
- Corrow SL, Dalrymple KA, Barton JJ. Prosopagnosia: current perspectives. *Eye Brain*. 2016;8:165–175.
- Mazza V. Simultanagnosia and object individuation. *Cogn Neuropsychol*. 2017; 34(7-8):430–439.
- Thomas C, Kveraga K, Huberle E, et al. Enabling global processing in simultanagnosia by psychophysical biasing of visual pathways. *Brain*. 2012;135(Pt 5):1578–1585.
- Sakurai Y, Hamada K, Tsugawa N, et al. Ventral simultanagnosia and prosopagnosia for unfamiliar faces due to a right posterior superior temporal sulcus and angular gyrus lesion. *Neurocase*. 2016;22(1):122–1229.
- Jackson GM, Shepherd T, Mueller SC, et al. Dorsal simultanagnosia: An impairment of visual processing or visual awareness? *Cortex*. 2006;42(5):155–157.
- Rizzo M. Balint's syndrome and associated visuospatial disorders. *Baillieres Clin Neurol*. 1993;2(2):415–437.
- Robertson L, Treisman A, Friedman-Hill S, et al. The Interaction of Spatial and Object Pathways: Evidence from Balint's Syndrome. *J Cogn Neurosci*. 1997;9(3):295–317.
- Vaina LM, Makris N, Kennedy D, et al. The selective impairment of the perception of first order motion by unilateral cortical brain damage. *Vis Neurosci*. 1998;15(2):333–348.
- Newsome WT, Paré EB. A selective impairment of motion perception following lesions of the middle temporal visual area (MT). *J Neurosci*. 1988;8(6):2201–2211.
- Peña-Casanova J, Roig-Rovira T, Bermudez A, et al. Optic aphasia, optic apraxia and loss of dreaming. *Brain Lang*. 1985;26(1):63–71.
- Gil R, Pluchon C, Toullat G, et al. Visuo-verbal disconnection (optical aphasia) for objects, pictures, colors and faces with abstractive alexia. *Neuropsychologia*. 1985;23(3):333–349.
- Mendez MF, Cherrier MM. Agnosia for scenes in topographagnosia. *Neuropsychologia*. 2003;41(10):1387–1395.
- Short RA, Graff-Radford NR. Localization of hemiachromatopsia. *Neurocase*. 2001;7(4):331–337.
- Bartolomeo P, Bachoud-Lévi AC, Thiebaut de Schotten M. The anatomy of cerebral achromatopsia: a reappraisal and comparison of two case reports. *Cortex*. 2014;56:138–144.
- Bub D. Alexia and related reading disorders. *Neurol Clin*. 2003;21(2):549–568.
- Pomeranz HD, Lessel S. Palinopsia and polyopia in the absence of drugs or cerebral disease. *Neurology*. 2000;54(4):855–859.
- Stagno SJ, Gates TJ. Palinopsia: a review of the literature. *Behav Neurol*. 1991;4(2):67–74.
- Kataoka H, Ueno S. Cerebral polyopia and palinopsia in a patient with occipital lobe epilepsy. *Epilepsy Behav*. 2009;14(4):684–686.
- Cooper SA, Joshi AC, Seenan PJ, et al. Akinetopsia: acute presentation and evidence for persisting defects in motion vision. *J Neurol Neurosurg Psychiatry*. 2012;83(2):229–230.
- Zeki S. Cerebral akinetopsia (visual motor blindness). A review. *Brain*. 1991;114(Pt 2):811–824.
- Mauguière F, Desmedt JE, Courjon J. Astereognosis and dissociated loss of frontal or parietal components of somatosensory evoked potentials in hemispheric lesions. Detailed correlations with clinical signs and computerized tomographic scanning. *Brain*. 1983;106(pt 2):271–311.
- Miller LJ, Mittenberg W, Carey VM, et al. Astereopsis caused by traumatic brain injury. *Arch Clin Neuropsychol*. 1999;14(6):537–543.
- Mennemeier M. Visual Neglect: In: *Encyclopedia of Clinical Neuropsychology*. Kreutzer J, DeLuca J, Caplan B, editors.

- Science+Business Media, LLC; New York; Springer. 2011;2638–2642.
44. Teeple RC, Caplan JP, Stern TA. Visual Hallucinations: Differential Diagnosis and Treatment. *Prim Care Companion J Clin Psychiatry*. 2009;11(1):26–32.
 45. Manfred M, Andermann F. Complex visual hallucinations. Clinical and neurobiological insights. *Brain*. 1998;121(Pt 10):1819–1840.
 46. Schadlu AP, Schadlu R, Shepherd JB. Charles Bonnet syndrome: a review. *Curr Opin Ophthalmol*. 2009;20(3):219–222.
 47. Penney L, Galarneau D. Peduncular Hallucinosis: A Case Report. *Ochsner J*. 2014;14(3):450–452.
 48. Lhermitte J. Syndrome de la calotte du pedoncle cerebral. les troubles psycho-sensoriels dans les lesions du mesocephale. *Rev Neurol*. 1922;38:1359–1365.
 49. Piano ME, Bex PJ, Simmers AJ. Perceptual Visual Distortions in Adult Amblyopia and Their Relationship to Clinical Features. *Invest Optim Vis Sci*. 2015;56(9):5533–5542.
 50. Kasten E, Poppel DA. A mirror in the mind: a case of visual alliesthesia in homonymous hemianopia. *Neurocase*. 2006;12(2):98–106.
 51. Jacobs L. Visual allesthesia. *Neurology*. 1980;30(10):1059–1063.
 52. Reptsis A, Demirtzoglou I, Nikolakopoulos A, et al. Visual allesthesia in a patient with glioblastoma multiforme. *Open J Ophthalmol*. 2012;2:97–102.
 53. Zukić S, Sinanović O, Zonić L, et al. Anton's Syndrome due to Bilateral Ischemic Occipital Lobe Strokes. *Case Rep Neurol Med*. 2014:474952.
 54. Maddula M, Lutton S, Keegan B. Anton's syndrome due to cerebrovascular disease: a case report. *J Med Case Rep*. 2009;3:9028.
 55. Caselli RJ. Visual syndromes as the presenting feature of degenerative brain disease. *Semin Neurol*. 2000;20(1):139–144.