

THE CENTRAL VISUAL PATHWAYS

REQUIRED READINGS: Blumenfeld, Chapter 11, pages 463 – 466 & 470 – 478, the Power Point Presentation and notes

LEARNING OBJECTIVES: after studying this chapter, students should be able to:

1. Describe the concept of visual fields
2. Trace the central visual pathways from the retina to the visual cortex.
3. Recognize the visual deficits produced by damage to the visual fibers at different locations along the visual pathways
4. Recognize the deficits produced by lesions of the striate and extra striate cortical areas

OVERVIEW

Information about objects in our visual fields their movement, shape, and color is captured by the photoreceptors and processed initially by the retinal cells. The axons of retinal ganglion cells form the optic nerves that take visual information to different diencephalic and midbrain structures, with most of the projections terminating in the lateral geniculate nucleus of the thalamus. Further processing of visual information occurs at this thalamic relay nucleus, which in turn projects to the primary visual cortex in the occipital lobe. From there, visual inputs are sent to other cortical areas from where the final visual perception emerges.

This chapter will be dedicated to the central visual projections and the processing of visual information by the thalamus and the cerebral cortex. We will also analyze the deficits produced by damage to the visual fibers along their trajectory.

Objective 1 – The visual fields

Slide 3 - The concept of visual fields can be understood as monocular visual field or binocular visual field. Each eye has its own monocular field of vision that is combined with the field from the other eye to produce the binocular visual field.

OBJ. # 1

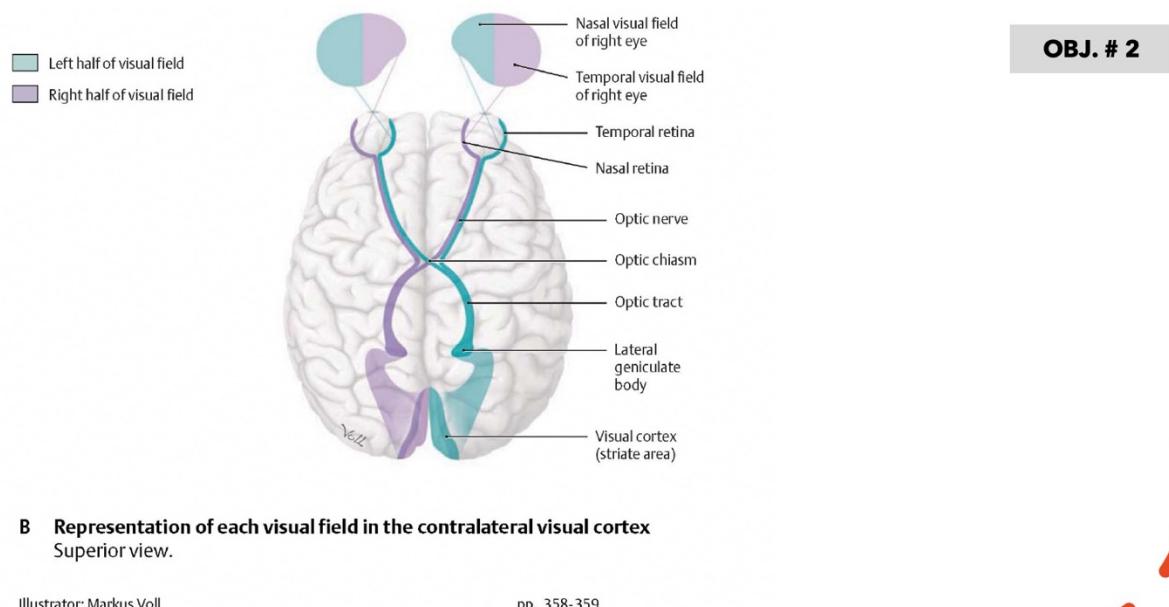
The Visual Fields

Binocular visual field is defined as the space we see with both eyes when the eyes are in primary position

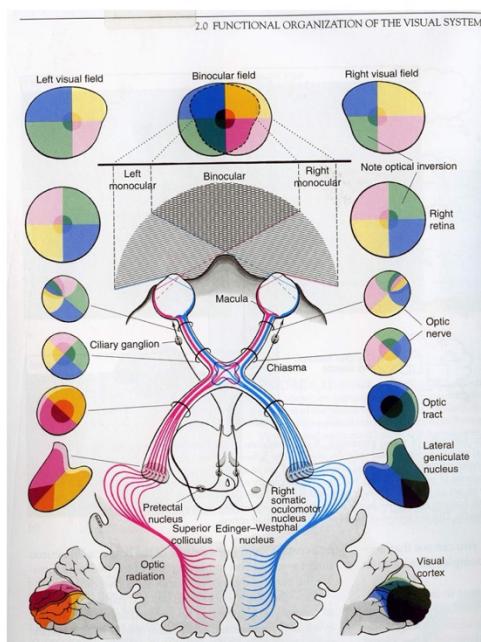


Objective 2 – The central visual pathways

Slide 4 - This diagram shows the entire visual pathway from the retina to the cerebral cortex. Axons from ganglion cells located on the nasal retinal areas from each side cross at the optic chiasm and join the contralateral temporal axons to form the optic tracts at each side of the brain. The optic tracts embrace the brainstem and reach the thalamus posteriorly to synapse with neurons in the lateral geniculate nucleus (LGN). Eighty percent of the fibers (axons) in the optic tracts terminate in this thalamic nucleus. After processing the visual information, the LGN neurons send it to the primary visual cortex or V1 in the superior and inferior banks of the calcarine sulcus in the occipital lobe.



Slide 5 – Another diagram showing the visual pathways with some more details.

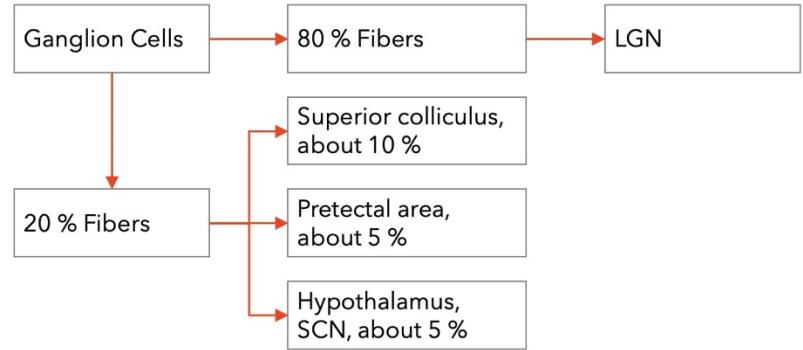


OBJ. # 2

Slide 6 - Not all retinal fibers terminate in the LGN. About 20% of the fibers terminate in other places: the superior colliculi and the pretectal areas, important for visual reflexes, and the fibers to the suprachiasmatic (SCN) nucleus of the hypothalamus, important for establishing circadian rhythms. The SCN is considered our internal body clock.

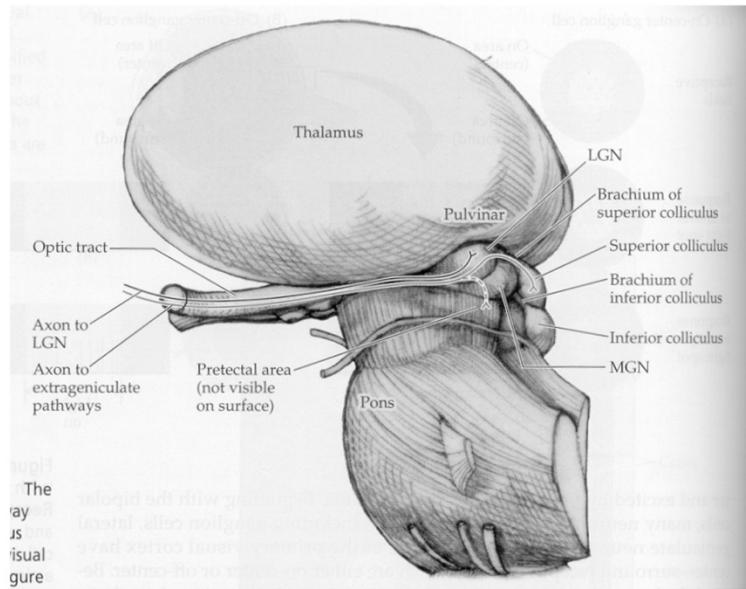
Retinal Output

OBJ. # 2



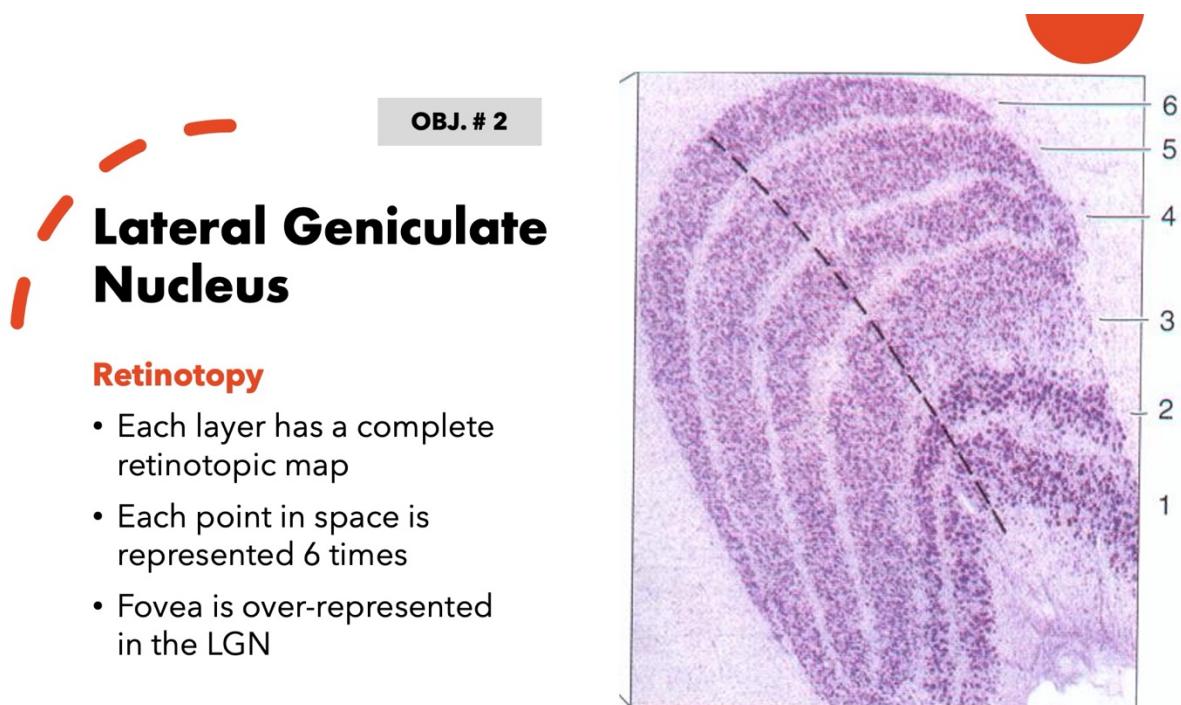
Slide 7 – This is a lateral view of the brainstem. As we move from the retina to the thalamus, we follow the optic tracts as they embrace the brainstem and terminate in the LGN and superior colliculus/ pretectal area.

OBJ. # 2

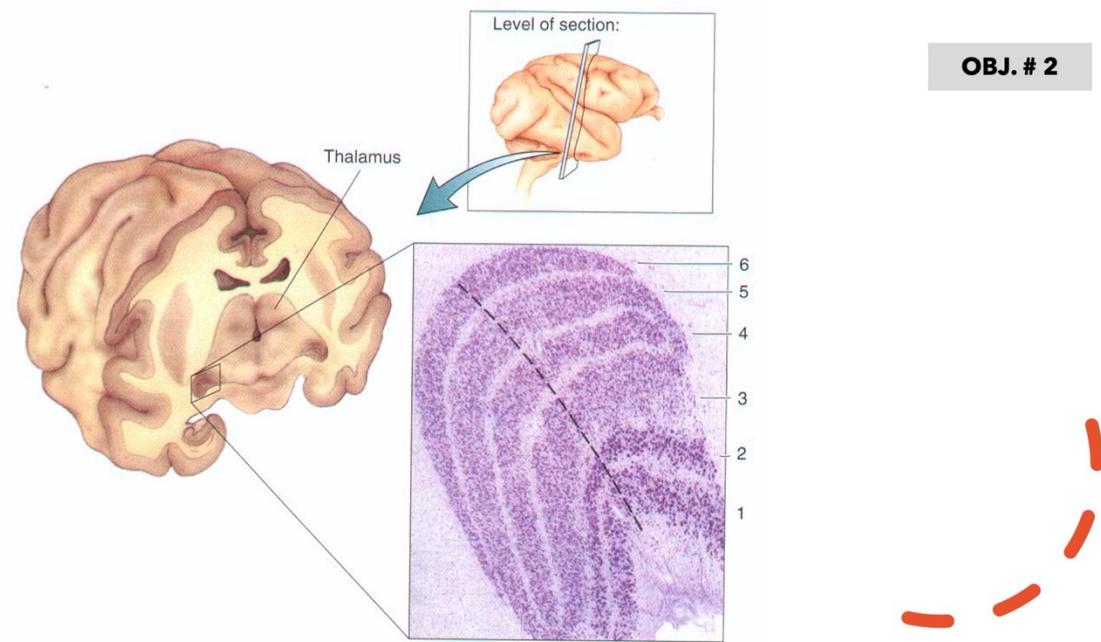


Trajectory Of The Visual Pathway

Slide 8 - Here is a histological section of the LGN showing the internal organization into 6 layers of neurons. Fibers in the optic tracts terminate in the ipsilateral LGN forming an organized retinotopic map. LGN layers 1 and 2 are called magnocellular layers and the rest are parvocellular layers. Magnocellular or parasol ganglion cell axons synapse exclusively with neurons in the magnocellular layers, while parvocellular or midget ganglion cell axons synapse exclusively with neurons in parvocellular layers 3 – 6. By this arrangement the input from the fovea where parvocellular or midget ganglion cells are more abundant is hugely overrepresented in the LGN. Also, information coming from the same point in space but viewed by both eyes terminate in different LGN layers: Layers 1, 4 and 6 receive information from the nasal fibers of the contralateral eye and layers 2, 3 and 5 receive information from the temporal fibers of the ipsilateral eye. In this way each point in space is represented 6 times in the LGN. If we could insert a probe through these layers at any location, we would see that each layer at that precise location processes visual information coming from the same point in space but viewed by different type of cells and by a different eye.

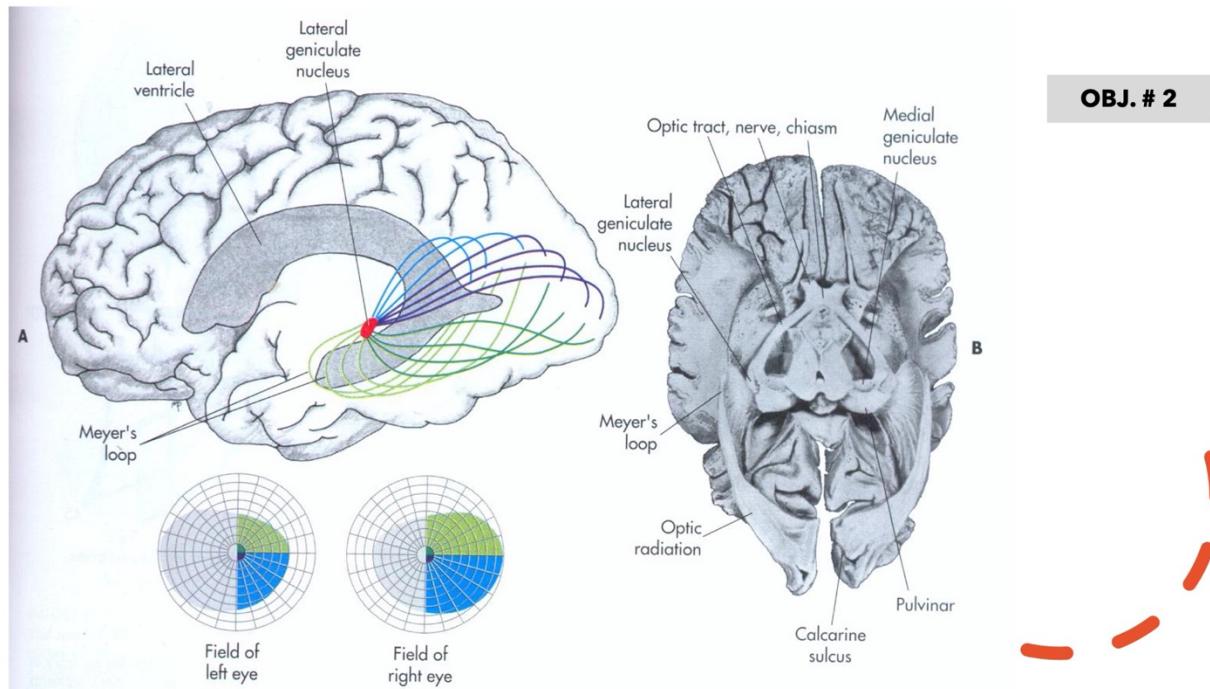


Slide 9 - This slide is to show the location of the LGN in a coronal section through the brain.



Slide 10 - This slide shows the pathway taken by the visual fibers from the LGN to the cortex in the occipital lobe.

The geniculocalcarine or geniculostriate tracts also called the optic radiations exit the LGN and divide into 2 pathways, the superior or direct pathway goes through the parietal lobe to reach the occipital cortex and terminate on neurons located in the gray matter of the superior bank of the calcarine sulcus. The inferior or indirect pathway makes a wide arc over the temporal horn of the lateral ventricle, then proceeds posteriorly to terminate on neurons located in the inferior bank of the calcarine sulcus. These inferior or indirect fibers are called the Meyer's loop. The so called direct fibers that terminate in the superior calcarine sulcus represent the superior part of the retina or the inferior part of the homologous visual fields for both eyes. The Meyer's loop fibers represent the inferior retina or the superior part of the homologous visual fields for both eyes. Homologous visual field is the way we refer to the visual field that is viewed by both eyes at the same time.



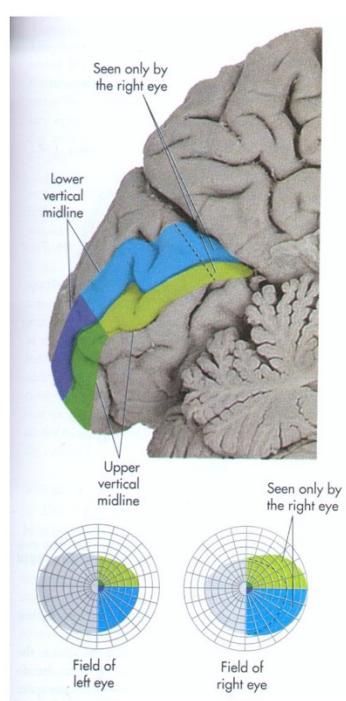
Slide 11 - The geniculocortical fibers or optic radiations terminate in the primary visual areas ipsilaterally and they represent the contralateral visual field. They synapse with cortical neurons forming a retinotopic map where each point in space is represented in the cortex.

The retinotopic map is such that the representation of the fovea takes the entire posterior half of both superior and inferior banks of the calcarine sulcus on each side. We see that there is here again an over representation of the fovea, it takes just 5 degrees of arc in the retina, but half of the space in the primary visual cortex! This can be explained if we remember that there were more ganglion cells in the central areas of the retina than in the periphery and that the central retinal fibers have an over representation in the different layers of the LGN.

OBJ. # 2

The occipital lobe - the primary visual cortex: the calcarine Sulcus

The most posterior half of the calcarine sulcus is the representation of the fovea



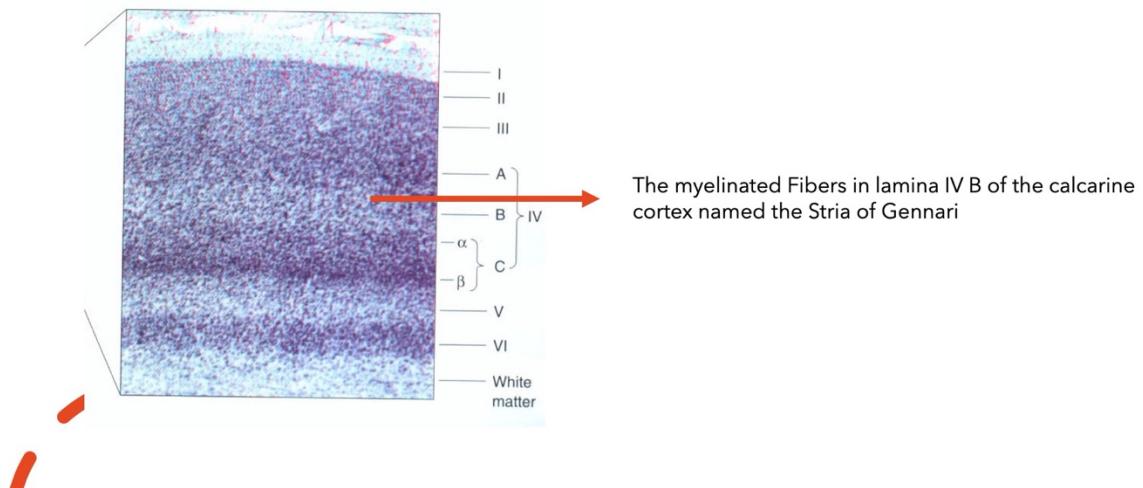
Slide 12 – Visual fibers terminate in various cortical layers with most of them going to layer IV of the primary visual cortices.

The primary visual cortex is also called V1 (visual I), Brodmann's area 17 or striate cortex. The name striate cortex comes from an unusual stripe of myelinated axons running parallel to the cortical surface at the level of layer IV named the Stria of Gennari.

Here is a histological section of the 6 layers of the primary visual cortex showing layer IV where the myelinated axons name the Stria of Gennari are located. Their presence gives the primary visual cortex the name of striate cortex.

The Layers Of The Primary Visual Cortex Or Striate Cortex

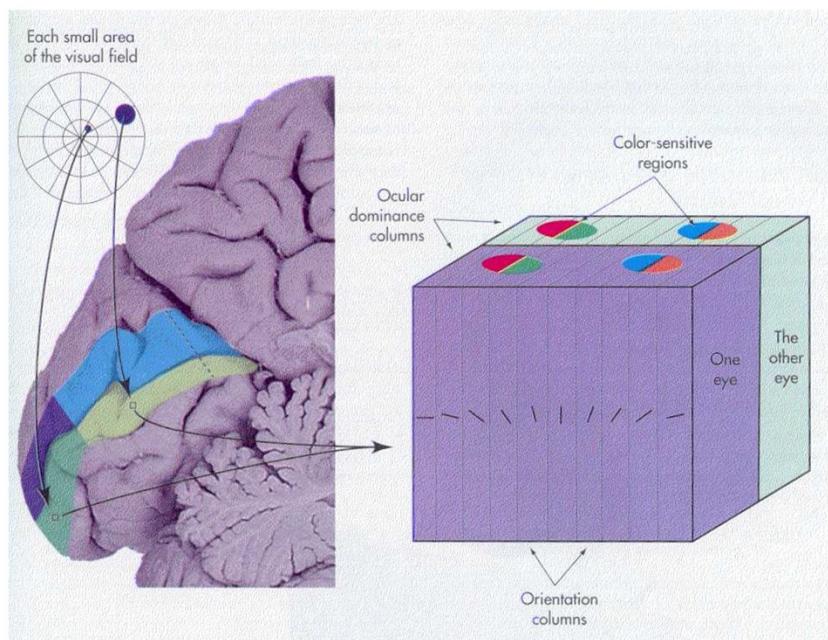
OBJ. # 2



Slide 13 - This image shows the columnar organization of the visual cortex.

Information coming from each eye is processed in different cortical columns. There is a complete segregation of information coming with the optic radiations about the ipsilateral and contralateral eye.

The cerebral cortex processes information, and not just visual but all types of information using a columnar organization. For the visual cortex, an ocular dominance column is a vertical cortical column, starting at the pial layer and ending at the border with the white matter, in which all neurons located in the 6 layers process the same information coming from one point in the visual field, viewed from the perspective of one eye. The next vertical column processes the same information but coming from the contralateral eye. Each one of these ocular dominance columns contains neurons that process information related to movement and direction of movement in the visual field, other neurons process color and others can communicate laterally with the next column and see the binocular space. Finally, each column has cells with complex receptive fields that allow them to have orientation selectivity, forming orientation sub columns. Each one of these sub columns respond better to a stimulus at a particular optimal orientation covering all the possible degrees of angle in space.



Slide 14 - In each ocular dominance column, visual information is processed using 3 different separate channels or pathways: there are neurons specialized in detecting motion, other neurons specialized in detecting fine details and shape of objects, and other neurons are specialized in the analysis of color. These 3 pathways of information are then transferred to higher visual areas where neurons have more complex visual fields and can extract more elements from the information received from the primary visual cortex.

Visual Information Is Processed Through 3 Different Channels

OBJ. # 2

Specialized in object motion information

Specialized in fine special information about object shape

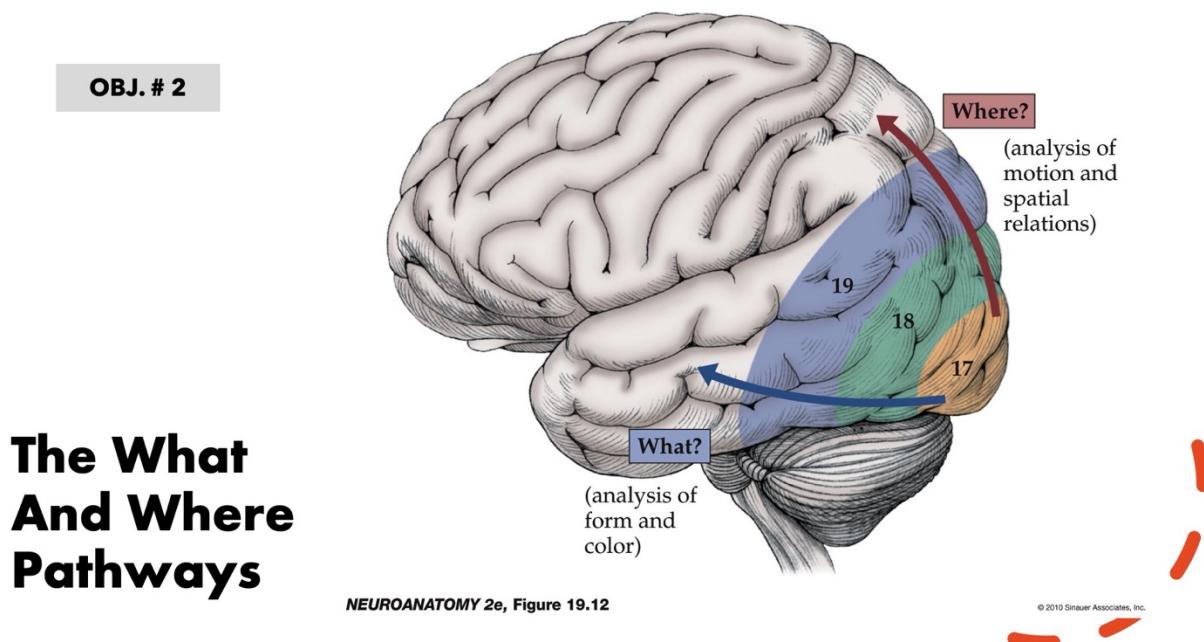
Specialized in the analysis of object color



Slide 15 - This slide from Blumenfeld's textbook shows the lateral surface of the cerebral hemispheres with the occipital lobe divided in 3 different areas. V1 is represented here at the most posterior part of the occipital lobe. V1 sends information to higher visual areas called visual association areas, Brodmann's 18 and 19 also named V2 and V3 that comprise the rest of the occipital lobe. These areas are also known as the extrastriate visual cortex.

Outside the occipital lobe, visual information projects to the parietal and temporal lobes using 2 pathways: a ventral pathway leading to the temporal lobe important for the perception of "what" objects are, and a dorsal pathway leading to the parietal lobe important for the perception of "where" objects are in space, mostly in relationship with us, so the visual system can guide actions toward those objects. The ventral pathway is important for processing information about color, shape, and identity of visual objects. There is plenty of cross talk between the two pathways with the parietal and temporal lobes sending projections to some common areas in the prefrontal cortex.

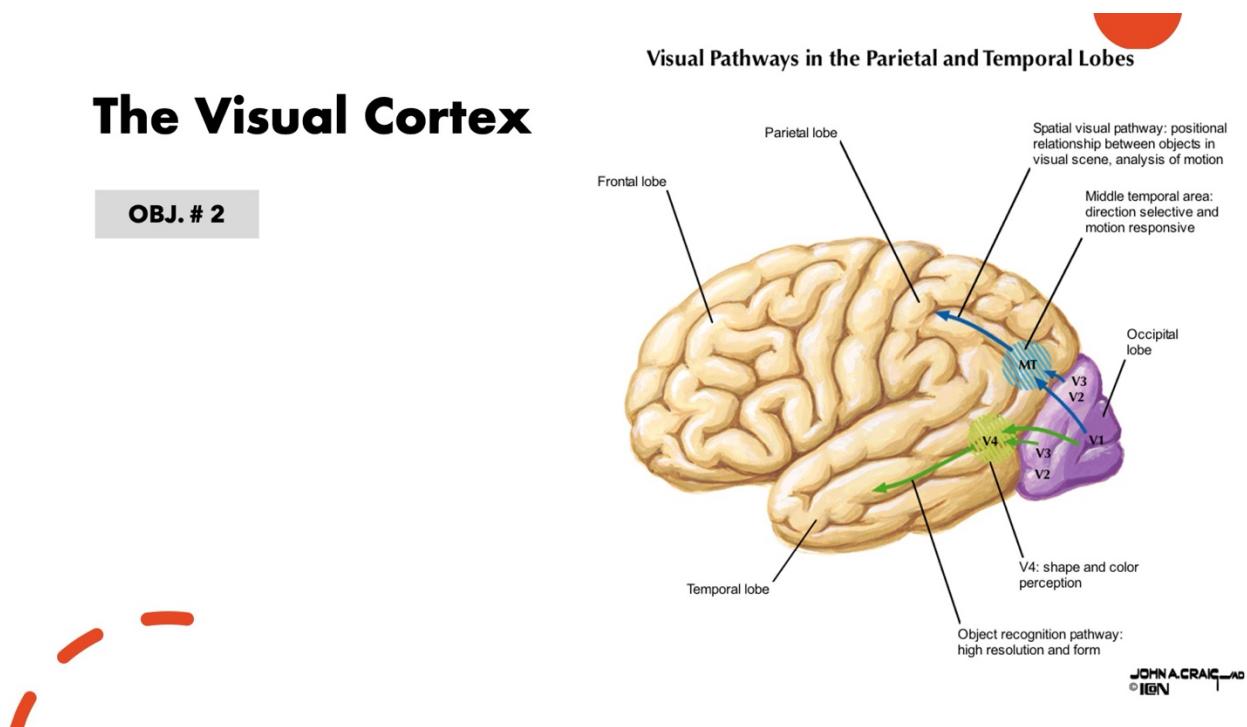
To give you an example of how these areas work imagine that you are reaching for an object in your visual field. To pick it up, you need the precise location of the object together with its size and orientation. All these elements are provided by the "where" pathway. At the same time your visual system is looking for a certain shape and color of the object in your visual space using the "what" pathway.



Slide 16 – In this diagram from Netter's Atlas we see how the ventral pathway reaches area V4 known to be the first area especially important for the perception of color. Neurons in area V4 are also sensitive to curvature or lines that meet at a specific angle. This information goes then to the object recognition area of the ventral temporal cortex.

The dorsal pathway reaches first area MT or middle temporal area, which is important for motion perception. Almost all the neurons there are motion sensitive and some of them respond to patterns of motion and can calculate what the overall direction of an object might be. When this area is damaged motion perception is severely altered.

Notice that this is only a diagram and the actual locations of MT and V4 are not accurate, see slide 17 for a more accurate location of these areas.



Slide 17 - Using neuroimaging studies in humans, several cortical areas with precise functions have been localized. The ventral or “what” pathway leads to 3 important areas for face, object, and place recognition in the cerebral cortex.

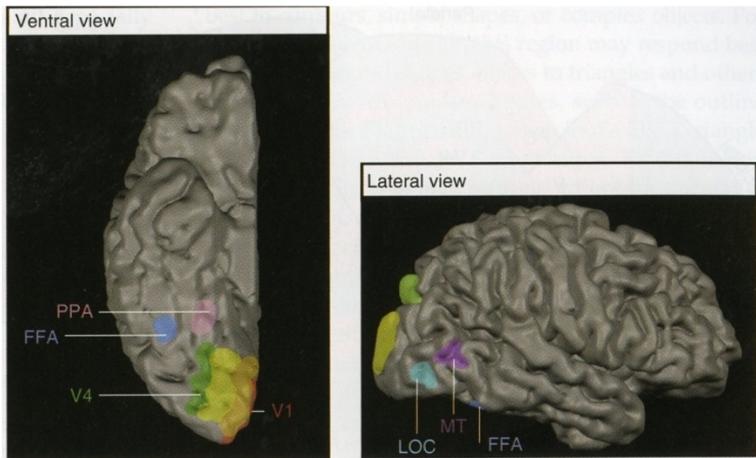
The LOC or lateral occipital complex area located on the lateral surface of the occipital lobe, just posterior to area MT, seems to recognize and respond to the shapes of objects.

The fusiform face area (FFA) which is part of the fusiform gyrus on the ventral surface of the temporal lobe is the place for face recognition. Neurons in this area respond best to faces than to any other object that is presented to them. In experiments with direct recording of these neurons in monkeys, researchers have concluded that these neurons were very good on telling apart different identities of faces.

The parahippocampal place area (PPA) is another cortical area where neurons respond best to houses, landmarks, and indoor / outdoor scenes but respond very little to faces, objects, animals etc.

OBJ. # 2

Object And Face Recognition Areas



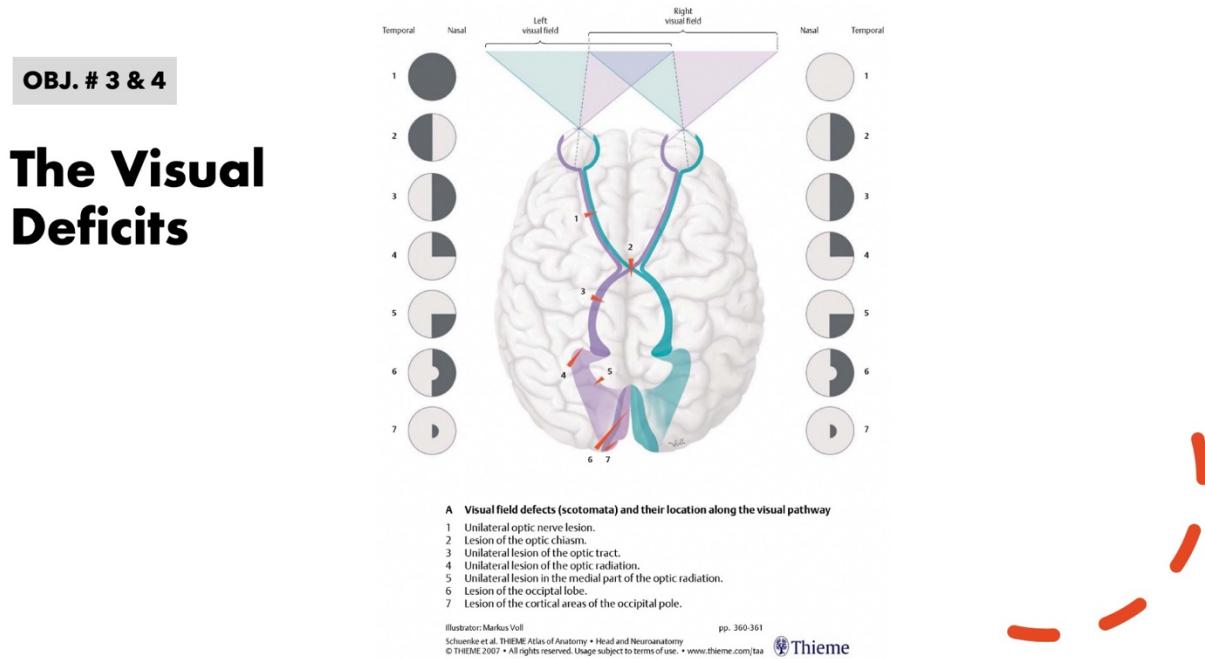
LOC area
Lateral occipital complex area

FFA
Fusiform face area

PPA
Parahippocampal place area

Objectives 3 & 4 – The visual deficits

Slide 18 - The visual deficits diagram

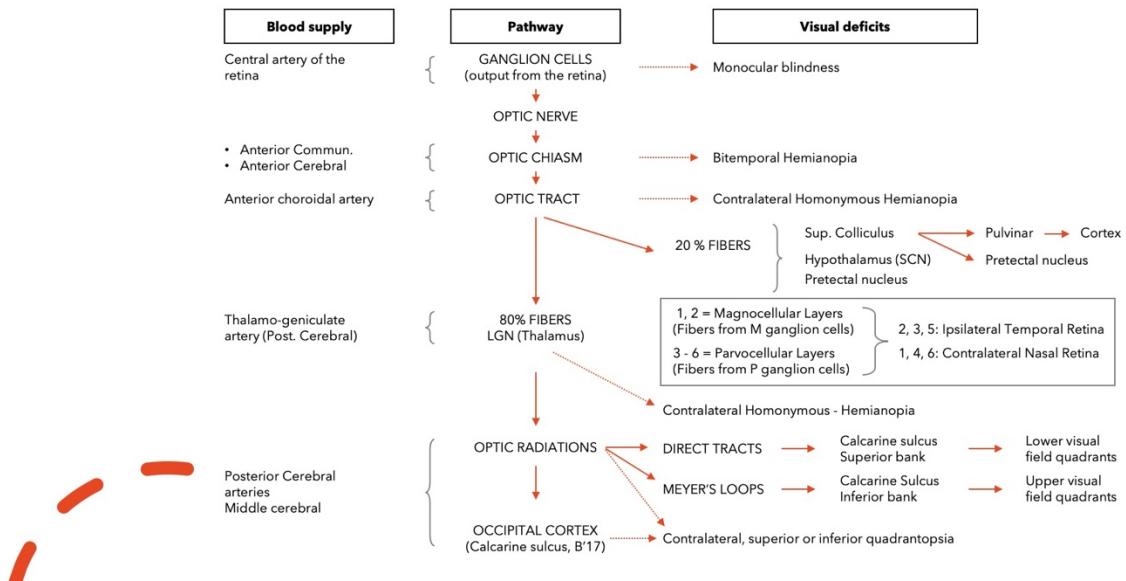


Slide 19 - This is a very busy diagram that summarizes the visual pathways. In the center of the diagram is the pathway, from the ganglion cell to the occipital cortex. On the left hand side, you can see the arteries providing blood supply to the different visual structures and pathways. On the right-hand side is a summary of the deficits produced by lesions of the visual pathways at different levels.



Visual Pathways

OBJ. # 3 & 4



Slide 20 - The typical deficits produced by lesions of the visual fibers. Remember that deficits are named by the visual field that is lost and not by the structure that has been damaged.

Visual Deficits

OBJ. # 3 & 4

Lesion to the optic nerve → Monocular blindness

Lesion to the central part of the optic chiasm → Bitemporal hemianopsia

Lesions to the optic tracts → Contralateral homonymous hemianopsia

Lesions to the optic radiations → Homonymous hemianopsia of a quadrant
↓
Contralateral superior or Inferior quadrantanopsia



Slide 21 – Damage to the visual cortex produces loss of visual perception of the contralateral visual field. In some of these patients, the ability to perform certain visual tasks could return after a period of recovery. Consider the case of a patient that claims to be blind on one side however can indicate the direction of movement of a light or objects in his blind visual field. This is called blind sight and for this phenomenon to be present it is important that the extrastriate pathways are intact. The LGN not only sends visual information to the primary visual cortex but also projects to several extrastriate areas mostly of the dorsal visual pathway (parietal lobe) bypassing V1. Neuroimaging studies have shown that unperceived stimuli presented to the blind side produces fMRI responses from the area MT, color-sensitive area V4 and regions involved in object recognition. These signals are however not sufficient to support visual awareness.

Cognitive Deficits Produced By Damage To Visual Cortical Areas

OBJ. # 3 & 4

Damage to the primary visual cortex results in loss of vision on the contralateral visual field. In some cases the macula is spared due to collateral blood supply to the occipital pole from the MCA

Some patients show what is known as **blindsight**. These patients report complete loss of visual perception however they can perform some visual tasks such as indicate the direction of movement of an object in their blind visual field



Slide 22 – When there is damage to the ventral cortical visual pathways, patients experience cognitive deficits. Damage to the ventral area V4 and other nearby areas processing color information produces cortical color blindness or achromatopsia. Patients report their experience as seen the world in shades of gray.

Visual agnosia is the deficit produced by lesions of the ventral object area. Patient with this deficit have difficulties recognizing objects by seeing them. They can still recognize objects by other attributes such as smell, touch, or hearing. The word “agnosia” comes from the Greek for lack of knowledge. Patients with visual agnosia can detect visually presented objects but they cannot perceive shape and cannot recognize or name the object. They have difficulty copying simple shapes such as circles or squares. Some of these patients can still perform certain visual tasks apparently using the visual input to the dorsal pathway to the parietal lobe. It is apparent that the dorsal pathway is not only responsible for where the objects are but also how actions can be performed towards a particular object, and the visual processing in this pathway is unconscious, so the patient is not aware of being using it to perform a task.

Other patients with visual agnosia have difficulty with object recognition even if there is apparent intact perception of them. The patients can describe the objects, they can copy them, mostly if given enough time but they cannot name the object. They can only recognize objects by smell or touch, so using other properties of the object outside visual recognition. This disorder is produced by damage to the ventral temporal cortex. In most cases patients with visual agnosia have difficulty recognizing both faces and objects. With lesions of the fusiform face area patients are still able to recognize objects but they cannot recognize faces. This deficit is called **prosopagnosia**. The deficit could be severe, with patients not recognizing family members or friends until they hear their voices or smell their perfume etc. Some patients cannot even recognize themselves in the mirror or photographs. Prosopagnosia occurs mostly with right sided lesions of the ventral pathway or bilateral lesions in the lateral occipital cortex, inferior temporal cortex and fusiform gyrus.

OBJ. # 3 & 4



Cognitive Deficits Produced By Damage To Visual Cortical Areas

Damage to the ventral pathway

- **Cortical color blindness or achromatopsia** - Produced by damage to area V4 and/or other color processing areas on the ventral temporal lobe
- **Visual agnosia** - Produced by lesions of the ventral pathway, object area - LOC

Slide 23 – Damage to the dorsal cortical visual pathway also produces cognitive deficits. Lesion to the posterior parietal and occipitoparietal cortex produce visual neglect. Patients with visual neglect completely ignore, do not respond to objects presented to the contralateral visual field. They can ignore also touch or sound coming from the contralateral side.

Lesions of the area MT and surrounding areas produce motion blindness. Unilateral lesions, usually due to stroke, lead to moderate deficits in the ability of the patient to perceive motion and there is considerable recovery over time. However, with bilateral MT damage the patients perceive the world as a series of snapshots. Crossing the street is dangerous for these patients because they cannot appreciate the distance cars really are from them or how fast they are approaching.

OBJ. # 3 & 4

Visual neglect - Produced by damage to the posterior parietal cortex

Motion Blindness - Produced by lesion of the area MT and surrounding areas



Cognitive Deficits Produced By Damage To Visual Cortical Areas

Damage to the dorsal pathway

Slide 24 – “Bilateral lesions of the parieto-occipital junction areas produce a more profound deficit called Balint’s syndrome. It is basically a deficit of spatial attention. In this uncommon syndrome, the appreciation of a coherent and detailed visual world is disrupted and the patient perceives only disconnected individual parts of the scene: visual simultanagnosia. Balint was a Hungarian neurologist the first to recognize this constellation of symptoms and signs. The defect is noted when the patient describes a complex scene in a disjointed way, single objects being pointed out, others missed entirely, the relationships and context of parts of the picture remaining unappreciated. The entire syndrome consists of (1) a disorder of visual attention mainly to the periphery of the visual field, in which the totality of a scene is not perceived despite preservation of vision for individual elements (visual simultanagnosia); (2) difficulty in grasping or touching an object under visual guidance, as though hand and eye were not coordinated (called by Balint optic ataxia); and (3) an inability to project gaze voluntarily into the peripheral field and to scan it despite the fact that eye movements are full (termed psychic paralysis of fixation of gaze by Balint, incorrectly called optic apraxia).

An essential feature of the Balint syndrome appears to be a failure to properly direct oculomotor function in the exploration of space. This is apparent when the patient is unable to turn his eyes to fixate an object in the right or left visual field or to consistently follow a moving object. The pattern in which the patient scans a picture is haphazard and fails to encompass all entire areas. Normal individuals accomplish visual scanning in a fairly uniform manner beginning paracentrally and moving clockwise, then to the corners.

Optic ataxia is detected when the patient reaches for an object, either spontaneously or in response to verbal command. To reach the object, the patient engages in a tactile search with the palm and fingers, presumably using somatosensory cues to compensate for a lack of visual information. The disorder may involve one or both hands and give the erroneous impression that the patient is blind. In contrast, movements that do not require visual guidance, such as those directed to the body or movements of the body itself, are performed naturally. The presence of visual inattention is tested by asking the patient to carry out tasks such as looking at a series of objects or connecting a series of dots by lines; often only one of a series of objects can be found, even though the visual fields seem to be full.

In almost all reported cases of the Balint syndrome, the lesions have been bilateral, mainly in the vascular border zones (areas 19 and 7) of the parietooccipital regions, although instances of optic ataxia alone have been described within a single visual field contralateral to a right or left parietooccipital lesion, and visual simultanagnosia has had variable localization.”

From: Adams and Victor’s, Principles of Neurology, Chapter 22, 10th edition, AccessMedicine

Cognitive Deficits Produced By Damage To Visual Cortical Areas

OBJ. # 3 & 4

Damage to the dorsal pathway

Balint's syndrome - Patients have difficulty to scan a complex visual scene or identify moving objects. They are able to perceive small regions of the visual field at a time. Patients have optic ataxia, ocular apraxia, and simultanagnosia

OPTIC ATAXIA

Inability to reach for an object in space under visual guidance or point to a target

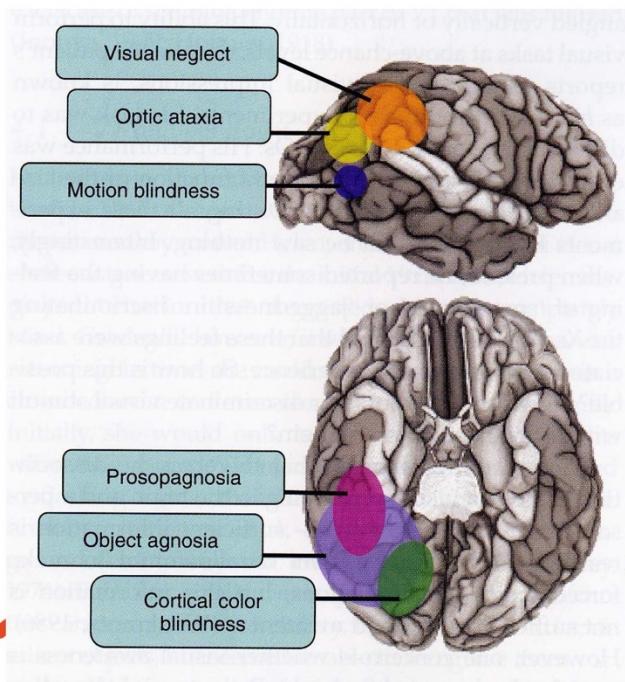
OCULAR APRAXIA

Difficulty in directing the gaze towards an object through saccades

SIMULTANAGNOSIA

Inability to perceive more than one object in the visual field simultaneously

Slide 24 - A diagram of the main visual areas for specific cognitive functions



Dorsal and ventral visual areas and cognitive visual deficits

OBJ. # 3 & 4