Towards a Greater Understanding of the Visual and Auditory Systems: From Basic Anatomy to the Higher Cortical Dysfunctions

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Overview of Lecture

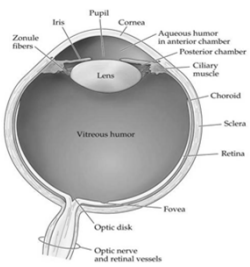
- Visual System
- Anatomy of Retina
- Optic nerves, subcortical nuclei
- Parvocellular/ Magnocellular pathways
- Cytoarchitecture of visual cortex
- Ventral & dorsal processing streams
- Primary visual cortex disorders
  - Field defects, achromatopsia, cortical blindness
- Associative visual disorders
  - Visual agnosias, Balint's syndrome
- Auditory System
- Anatomy of ear
- Cochlea
- Brainstem auditory nuclei
- Brainstem auditory tracts
- Thalamus-medical geniculate body
- Organization of primary auditory cortex – Heschl's gyri
- Primary auditory cortex disorders
  - Tinnitus, sound localization disorder
- Associative auditory disorders
  - Auditory agnosias, amusia

Visual System

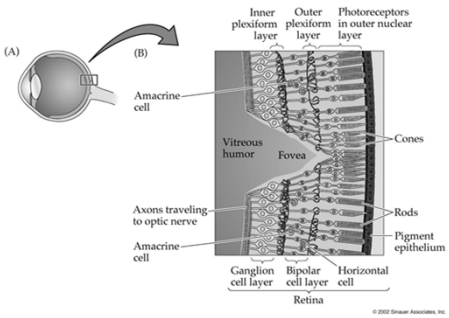
From Basic Anatomy to Higher Cortical Dysfunction

Most figures from: Blumenfeld, H. (2010). *Neuroanatomy through clinical cases.* 2nd ed. Sunderland, MA: Sinaurer.

Anatomy of Eye

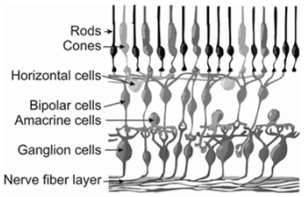


Retina



Retina

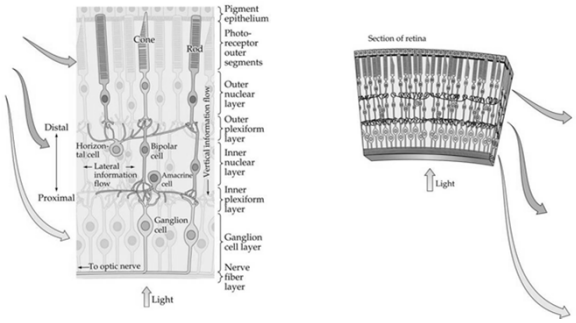
- Contains millions of cells
- About ½ millimeter thick
- Like a 3-layered cake
  - 3 layers of cell bodies
  - 2 filling layers where synapses occur
- # of cones = 6,400,000
- # of rods = ~120,000,000



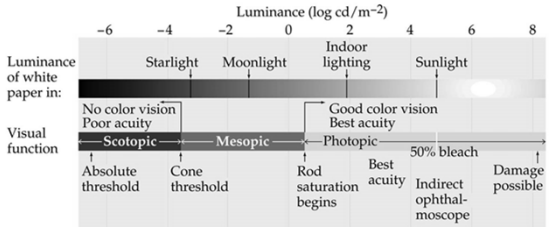
Photoreceptors = Rods & Cones

- Rods
  - More numerous than cones by a ratio ~20:1
  - Do not detect colors
  - Poor spatial resolution
  - Poor temporal resolution
  - Main function for low-level lighting conditions; far more sensitive than cones.
- Cones
  - Less numerous than rods
  - More represented in fovea where acuity is highest
  - Detect colors
  - High spatial resolution
  - High temporal resolution

Retina

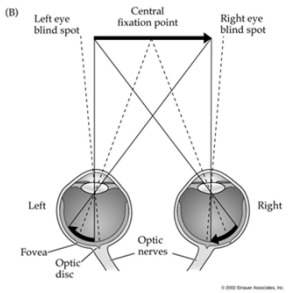


Rods & Cones – Specialized light sensitivity

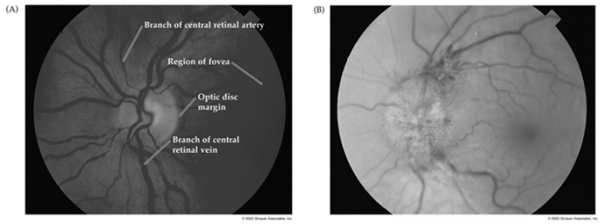


Fovea / Optic Disc

- Central fixation point for each eye falls onto fovea
- Fovea = region of highest visual acuity
- **Fovea** = ~200,000 cones; represented by ~1/2 fibers in optic nerve; ~1/2 cells in primary visual cortex; not fully developed until 4 years of age.
- **Macula** = oval region ~ 3 by 5 millimeters surrounding fovea relatively high acuity.
- Medial (nasal) to fovea is **optic disc** – axons leaving retina to form optic nerve.

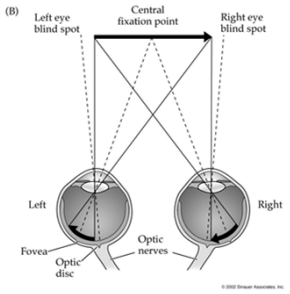


Papilledema

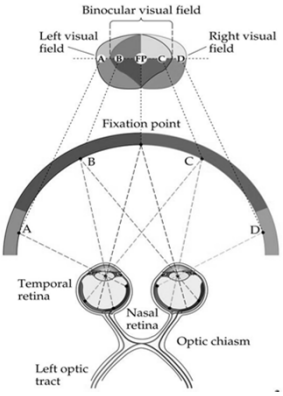


Blumenfeld, 2010

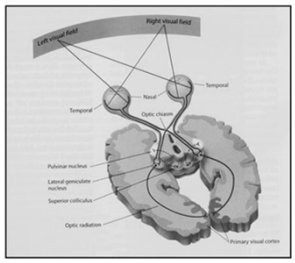
Images on Retina



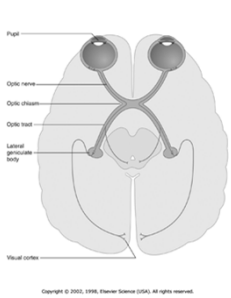
- Image on retina is inverted and reversed:
- Upper visual space projects onto lower retina
- Right visual space projects to left hemiretina of each eye

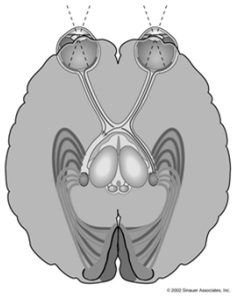


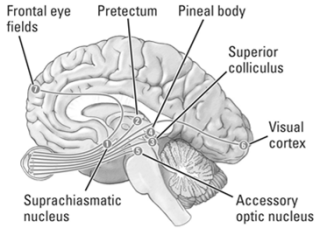
Lateralization



Optic nerve (CN II)

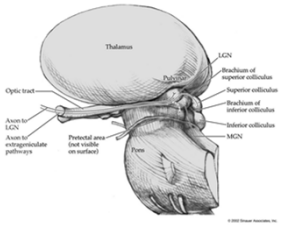


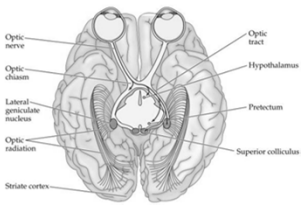




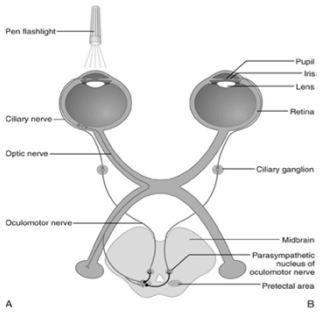
1. Superchiasmatic nucleus – daily rhythms (sleep, feeding).
2. Pretectum – changes in pupil size in response to light.
3. Pineal gland – long-term circadian rhythms.
4. Superior colliculus – head orienting.
5. Accessory optic nucleus – Eye movement to compensate for head movement (vestibulo-ocular reflex).
6. Visual cortex – Pattern perception, depth perception, color vision.
7. Frontal eye fields – voluntary eye movements.

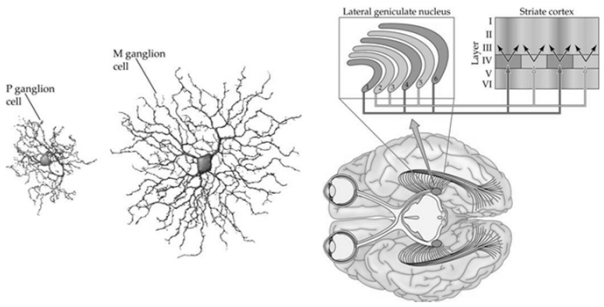
Major Visual Pathways



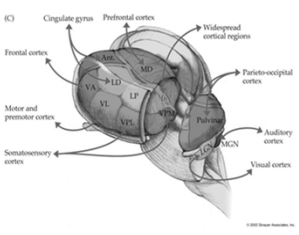


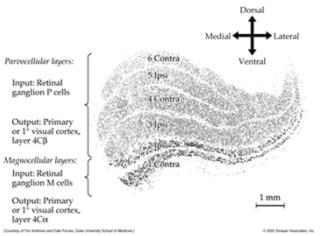
Testing Pupillary & Consensual Reflexes

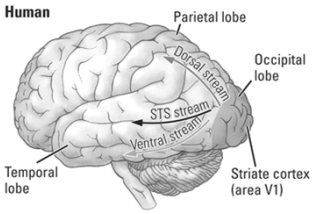




Thalamus – LGN Layers







- Dorsal stream = continuation of magnocellular division
- Ventral stream = continuation of the parvocellular division

Relative position of optic radiations through internal capsule (posterior limb)

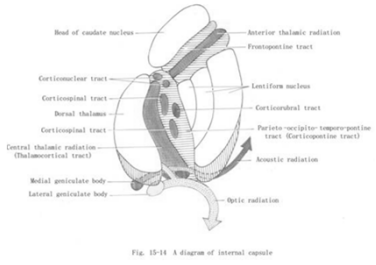
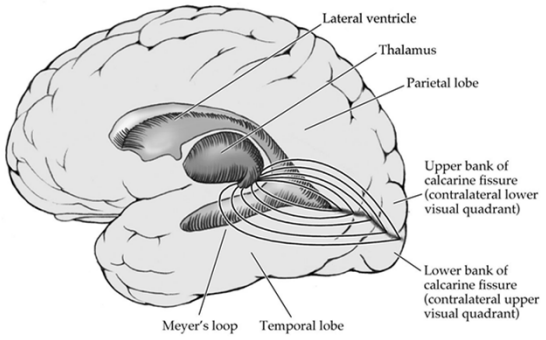
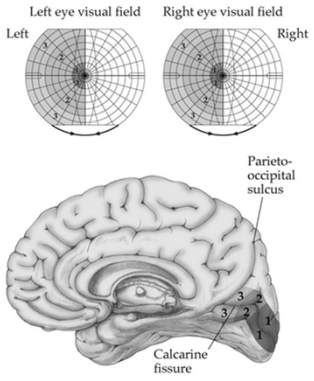
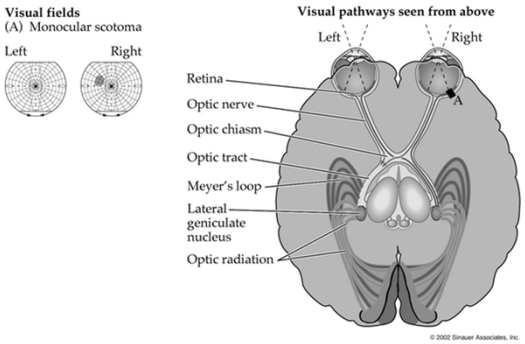


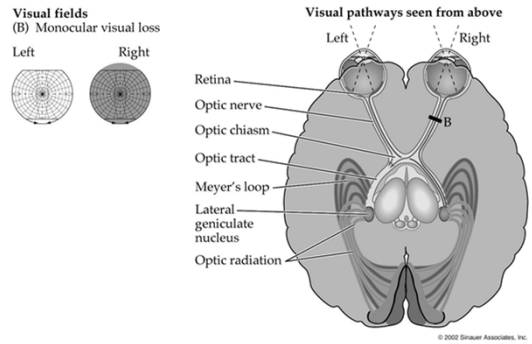
Fig. 15-14 A diagram of internal capsule

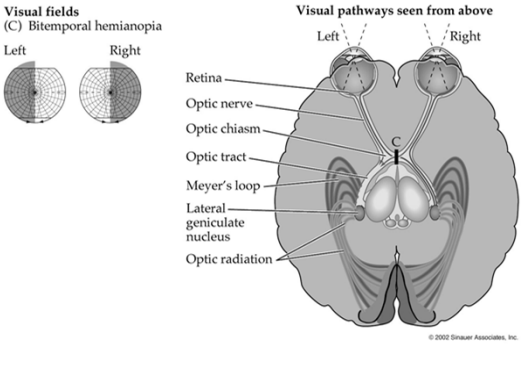


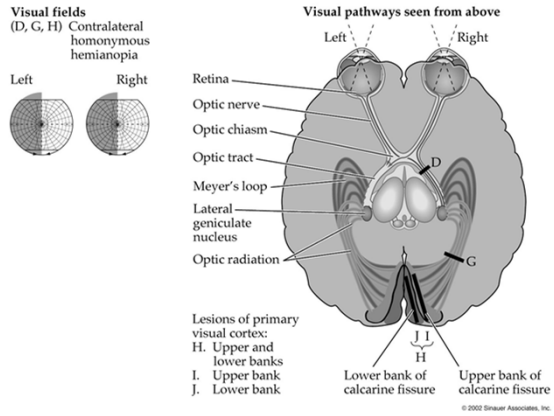


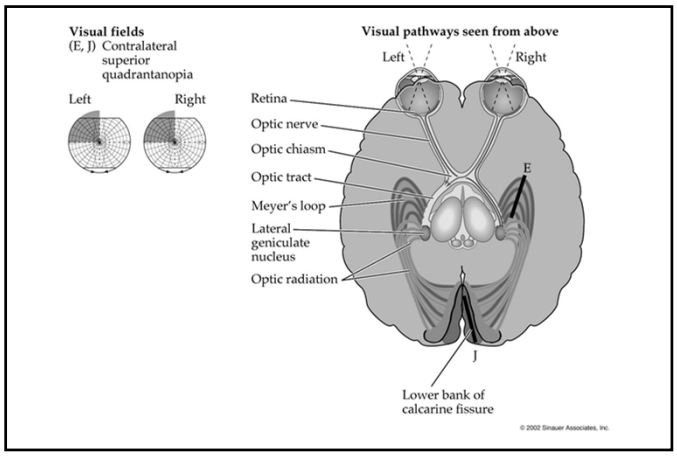
Disorders of Visual Pathways

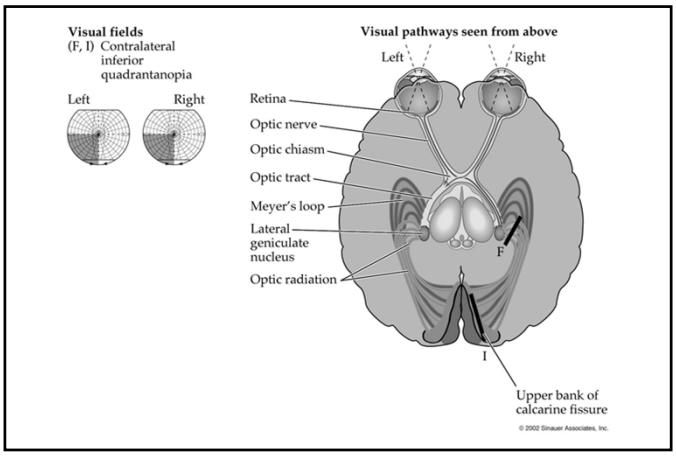


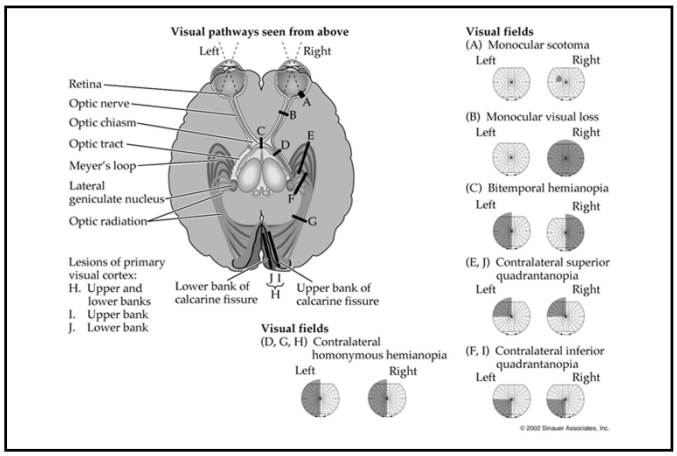




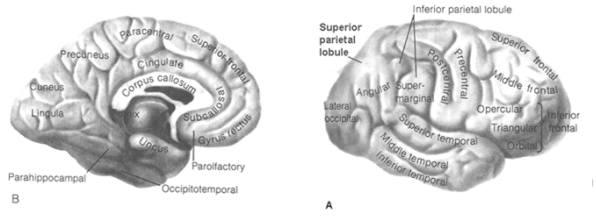


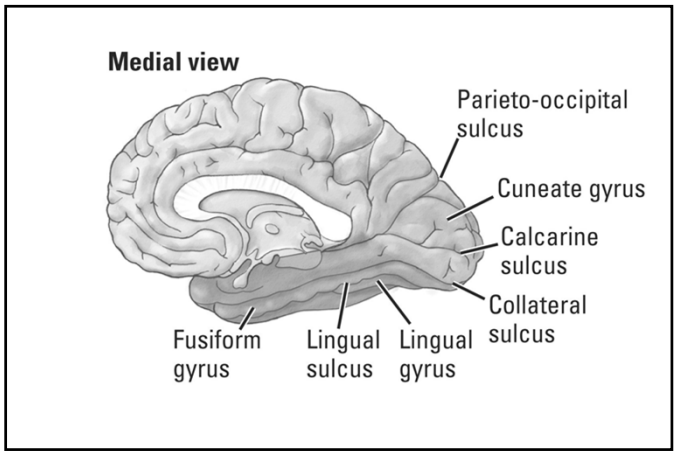


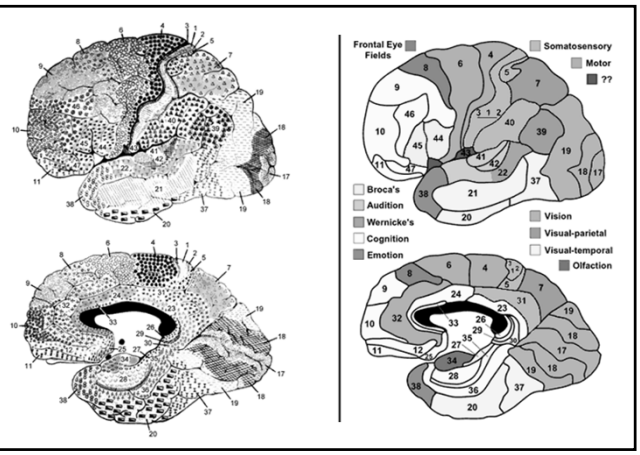


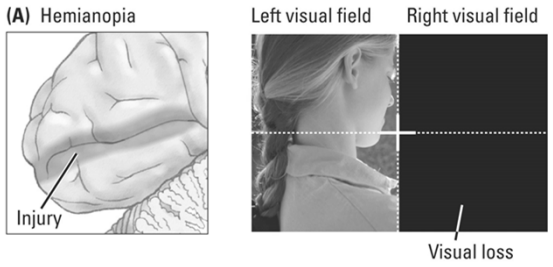


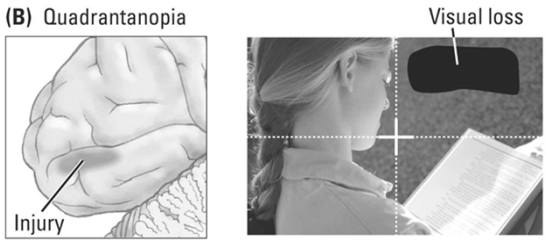
Occipital Lobe Gyri

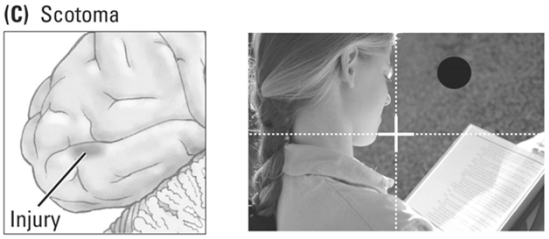


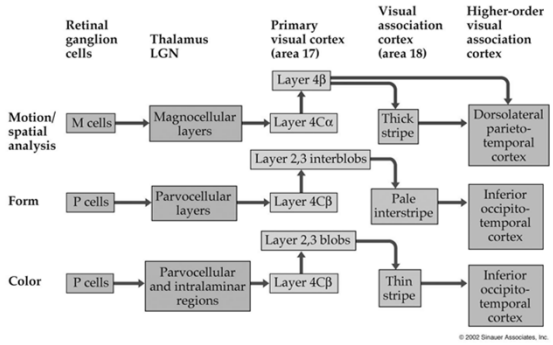




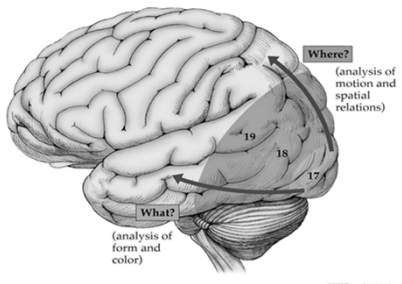




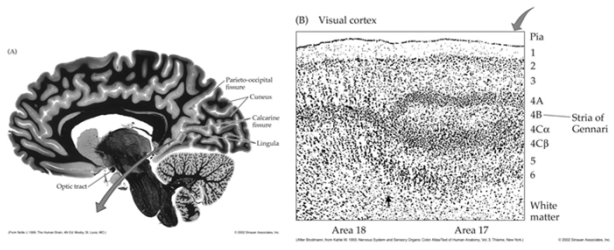


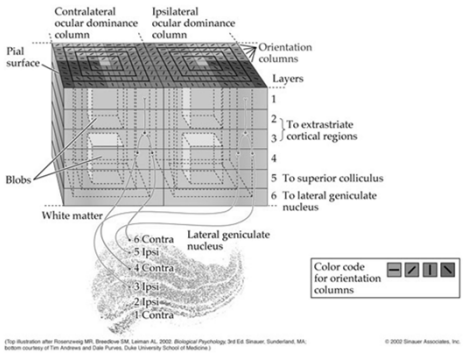


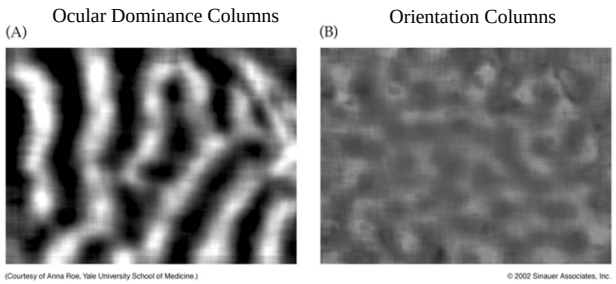
Ventral & Dorsal Visual Pathways



Cytoarchitecture of Visual Cortex

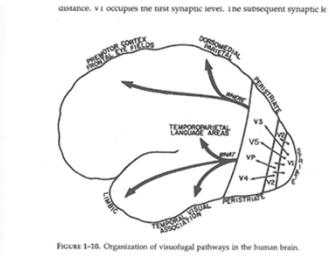






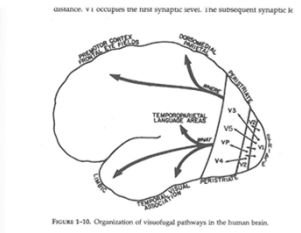
Subdivisions of Occipital Cortex

- At least 6 different occipital regions:
- V1, V2, V3, V3A, V4 & V5
- V1 = area 17
- V2 = ~area 18
- V3 & V3A = ~ area 19
- V4 = ~ area 19 on ventral surface
- V5 (a.k.a., MT) area 19 into area 37

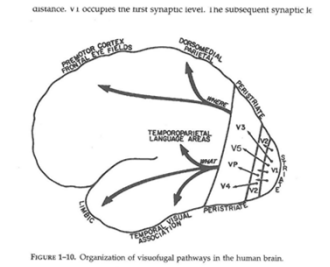


Connections of Visual Cortex

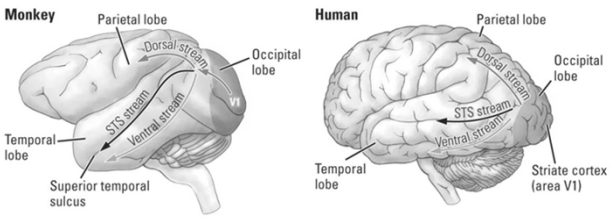
- LGB of thalamus to V1 & V2 via geniculocalcarine tract
- V1 primary projection area for vision
- V1 projects to all other occipital regions
- V2 second level in hierarchy – more elaborate visual processing
- V2 also projects to all other occipital regions.

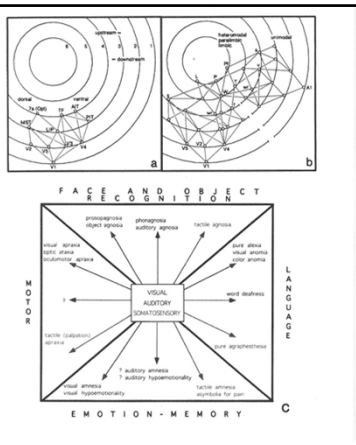


- After V2, 4 distinct parallel pathways emerge for further processing in V3-V5 & VP in parietal & temporal lobes.
- These converge into 3 pathways in monkey
  - 1 to parietal lobe
  - 1 through superior temporal sulcus
  - 1 inferior temporal pathway

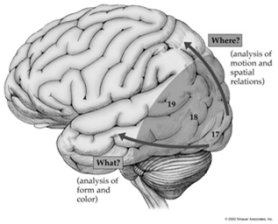


3 Processing Streams in Monkey & Man





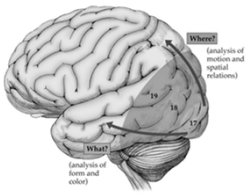
Dorsal visual processing stream

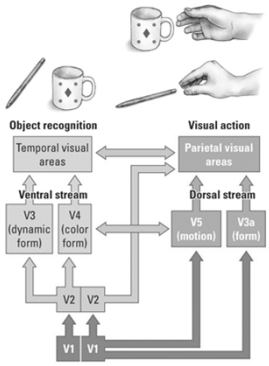


- "action stream" – "where"
- Motor movements under visual guidance
- Used to adjust limb movements to move through spatial environment
- Superior longitudinal fasciculus – from secondary visual processing areas to parietal lobe
- Mediates visual-spatial functions
- Mediates "vision for action"
- Visual processing required to direct specific movements

Ventral visual processing stream

- "perceptual stream" – "what"
- Used to form & identify visual percepts
- Visual recognition
- Mediates object perception (e.g., color, form, texture) & recognition
- Distinguish among individual members of a class (e.g., tools, foods, body parts, cars, etc.)
- Temporal lobe visual areas





Disorders of Visual Processing

Some Elemental Disturbances

TABLE 11.1 Some Terms to Describe Visual Disturbances

TERM	DEFINITION
Scotoma	A circumscribed region of visual loss
Homonymous defect	A visual field defect in the same region for both eyes
Refractive error	Indistinct vision improved by corrective lenses
Photopsias	Bright, unformed flashes, streaks, or balls of light
Phosphenes	Photopsias produced by retinal shear or optic nerve disease
Entopic phenomena	Seeing structures in one's own eye
Illusions	Distortion or misinterpretation of visual perception
Hallucination	Perception of something that is not present

Effects of occipital lobe lesions

- V1 = cortical “blindness”
- V2 = visual perceptive disorders
- V3 = defect of form perception
- V4 = defect of color vision; achromatopsia
- V5 = defect of motion detection

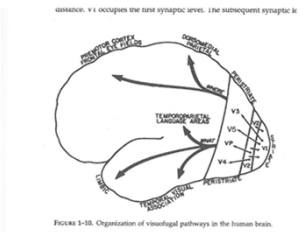


FIGURE 1–10. Organization of visuofugal pathways in the human brain.

Lesions of visual cortex

- In theory, V3 lesions should affect form perception
- But since V4 also processes form
- Large lesion(s) of both V3 and V4 would be needed to completely eliminate form perception.
- Damage to V4 = defect of color vision (achromatopsia); able to see only gray
- Also, loss of color cognition
- Can’t recall colors or even imagine colors
- Colors gone from dreams

Lesions of visual cortex

- Damage to V5 = inability to perceive objects in motion (akinetopsia)
- Objects at rest are seen, but once they move, they disappear.

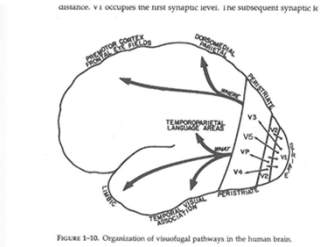
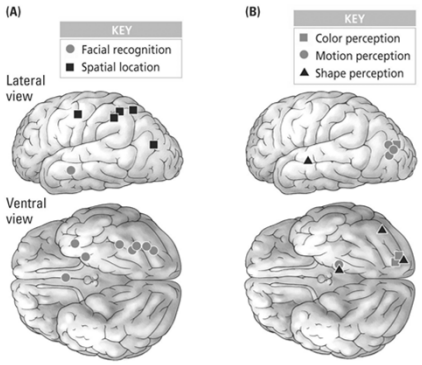
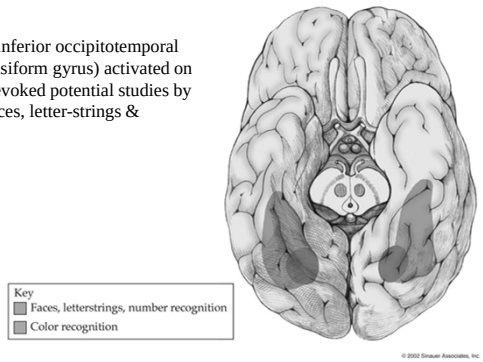


FIGURE 1–10. Organization of visuofugal pathways in the human brain.



Areas of inferior occipitotemporal cortex (fusiform gyrus) activated on fMRI & evoked potential studies by colors, faces, letter-strings & numbers.



Achromatopsia

- Loss of color vision
- Preserved form, movement depth, & other visual percepts
- Maybe both fields, hemi-, or quadrant field
- Full-field achromatopsia associated with visual agnosia
- Lesions involve V4 (middle 1/3rd of lingual gyrus)
- Affected field sees shades of gray
- May be unable to imagine or dream in color as well
- Lesion = PCA infarct (embolic)

Stereopsis

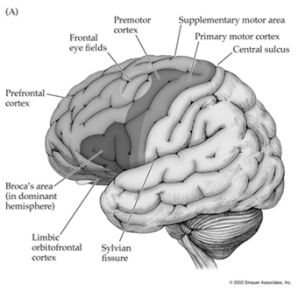
- *"Ability to discriminate depth from binocular visual input"*
- Physical separation of eyes causes 2-D projections of 3-D objects to occupy slightly different positions on L & R retinas.
- This disparity is the source of stereopsis.
- Each visual cortex has both monocular & binocular cells
- Neurons related to stereopsis are located in secondary association cortices
- Animals deprived of binocular stimuli at critical age develop primarily monocular cortex
  - E.g., strabismus or amblyopia

Cerebral akinetopsia

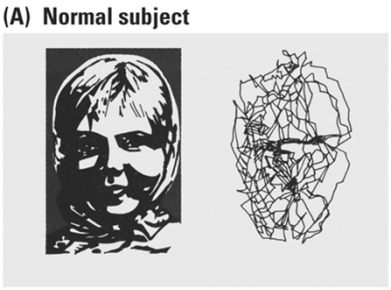
- *"an acquired defect of motion processing"* due to cerebral lesions
- Lack of motion cues may cause defective smooth pursuit movements & inability to perceive motion-defined objects
- Bilateral lesions around the P-O-T junction in V5 (MT)
- Patient L.M. (Zihl, 1983) could see movement in slowly moving targets
- But targets moving faster than 10-14° per second seemed to materialize at successive positions with no movement in between.
- Area MT contains neurons sensitive to direction, speed, orientation, & binocular disparity.

Frontal Lobe Visual Areas

- All pathways reach the frontal lobe (ILF, SLF, and the arcuate fasciculus)
- Area 8, Premotor, "Frontal Eye Fields"
- Directed, volitional visual scanning of selected portions of objects
- Voluntary gaze under executive control
- More scanning in left visual field



Frontal eye fields scanning example



Effects of Bilateral Occipital Lobe Disease

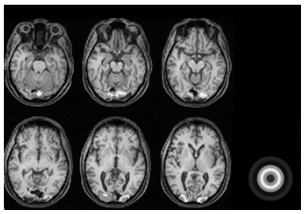
- Cortical Blindness
- Anton's Syndrome
- Achromatopsia
- Visual Agnosia
- Prosopagnosia
- Balint's Syndrome
  - Simultanagnosia
  - Optic ataxia
  - Ocular apraxia
- Pure alexia
- Stereopsis
- Cerebral Akinetopsia
- Bonnet's Syndrome

Cortical Blindness

- After bilateral V1 damage = person acts as if blind; are not aware of visual input.
- Testing shows they act on visual information; "**blindsight**"
- E.g., points to light flash locations, detect motion, discriminate orientation of lines
  (Case D.B. – Weiskrantz)
- Some visual info gets to higher levels despite V1 damage
- Via LGB to V2 and/or via
- Superior colliculus to pulvinar (thalamus via tecto-pulvinar tract) to cortex

Cortical Blindness

- Visual capabilities depend upon individual patient and lesion extent – often transient.
- Often a loss of words to describe any conscious perception or
- Patient describes salient stimuli as patterns of wavy lines or grids.



Anton's Syndrome

- Denial of (anosognosia for) cortical blindness,
- Patients deny any visual impairment even though they can make out very little.
- Loss of awareness of defect
- Often transient
- stimulation of the retinas and superior colliculi
- provide the parietal cortices with signals indicative of the usual situation of looking and seeing,
- provides the basis for subsequent misinterpretation that characterizes Anton's syndrome and blindsight.

Associative Visual Agnosias

- Disorder of object recognition
- Inability to recognize object despite accurate perception of object
- Disconnection between the visual percept (image) and the stored representation of the item
- Ventral stream lesions in both hemispheres
- Can demonstrate accurate perception by copying a drawing of the object
- Damage is further "upstream" in the ventral (inferior longitudinal fasciculus) pathway from occipital to temporal lobes
- Bilateral PCA or watershed infarcts (cardiopulmonary arrest, near drowning, carbon monoxide poisoning)

Prosopagnosia

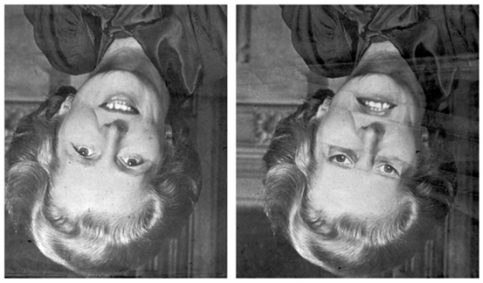
- An inability to recognize previously known (familiar) faces and to learn to recognize new faces
- Deficit not limited to faces
- Also difficulties identifying sub-members of any class of objects
- E.g., different makes of cars, species of birds
- Recognition is preserved via other sensory channels
- E.g., others recognized by their voice, body movements, attire.
- Generic recognition is intact; they know a face is a face or a car is car.
- Lesions = bilateral fusiform gyri

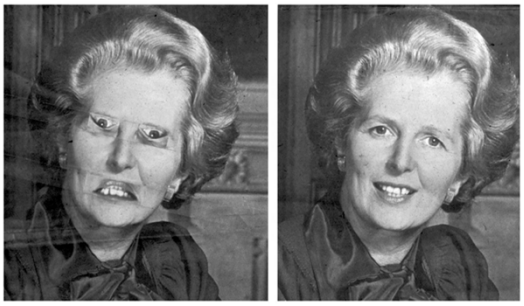
Prosopagnosia

- Prosopagnosics can non-consciously recognize faces; shown by intact SCRs.
- Suggests some part of the physiological process of face recognition is intact
- But results of this process are unavailable to consciousness
- This SCR response to familiarity is abolished by
- Bilateral damage to ventromedial prefrontal cortices
- Demonstrates a double dissociation between overt and covert face recognition

Face Perception

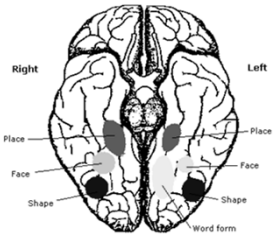
- Monkeys have neurons in temporal lobes specifically tuned to:
  - Facial identity
  - And others to facial expression
- Humans are particularly sensitive to the configuration of upright faces.
- Demonstrated by the *Margaret Thatcher illusion* (Haxby, 1999) presented by Kolb & Whishaw
- Haxby showed inverted faces processed by same cortical regions as other visual stimuli
- Whereas upright faces are processed in a separate face-perception system





Face perception brain regions

- Kanwisher (1997) identified a "fusiform face area" (FFA) - BA37 - using fMRI
- This region responds preferentially to faces
- Although it may also process some information about other objects



Balaint's Syndrome

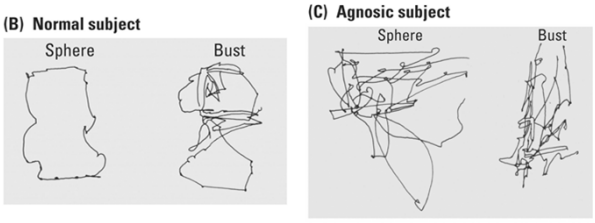
- *Simultanagnosia*
- *Optic ataxia*
- *Ocular apraxia*

- *Optic ataxia* = poor visual guidance of movement; defective "vision for action"; impairment of target pointing

- *Ocular apraxia* = defective voluntary eye movements; inability to shift gaze at will; "psychic paralysis of gaze"

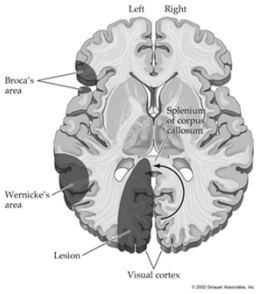
- *Simultanagnosia*
  - Inability to perceive multiple objects simultaneously or at one time
  - Can see basic shapes
  - Unable to see 2 objects in picture, only one at a time.
  - Often act as if they were blind
  - They are overwhelmed by the task of seeing the world

Ocular apraxia in Balint's



Pure alexia

- "alexia without agraphia"
- "wordblindness"
- Disorder of visual pattern recognition
- Can see sentences, words & letters, but cannot read
- Is a disconnection visual agnosia
- lesion(s) disconnect both visual cortices from language-related areas



Charles Bonnet syndrome

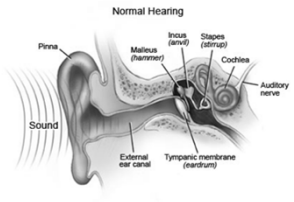
- "visual release hallucinations"
- Hallucinations in the visually-impaired elderly (reduced visual acuity)
- Often continuous hallucinations for years
- Begin with elementary hallucinations, but evolve into complex ones
- Often animated, vibrantly colored scenes involving entire visual field ("Lilliputian")
- Patients reject reality of hallucinations
- May be brief (days) or chronic (years)
- No proven treatment

Auditory System

From Basic Anatomy to Higher Cortical Dysfunction

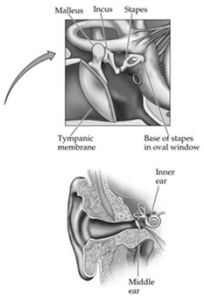
Most figures from: Blumenfeld, H. (2010). *Neuroanatomy through clinical cases*. 2nd ed. Sunderland, MA: Sinaurer.

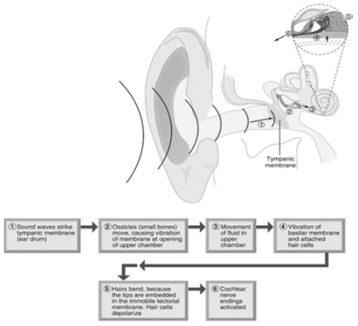
Cochlea



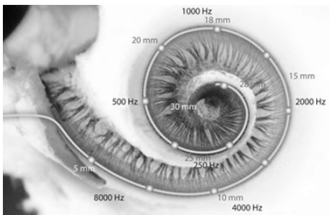
- Sound stimulates tympanic membrane
- Oval window vibrates ossicles
- Which vibrates round window
- Ossicles act as mechanical amplifiers; increase sound waves 22 times greater

Middle ear - ossicles

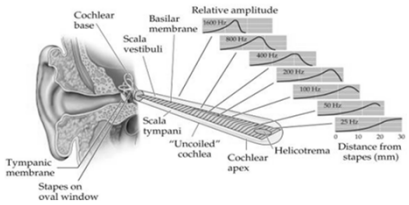




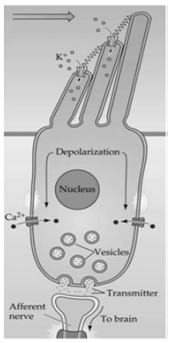
Organ of Corti within Cochlea



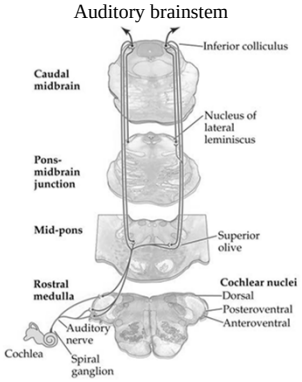
Uncoiled cochlea



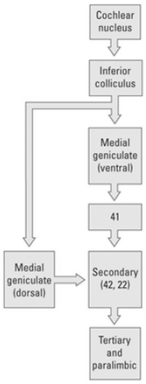
Depolarizing hair cell

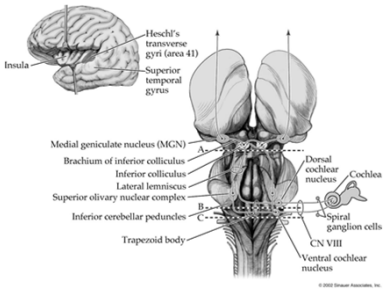


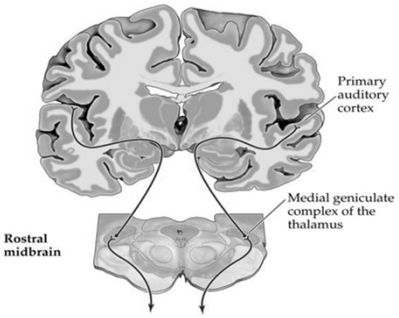
Auditory brainstem



Auditory Pathways

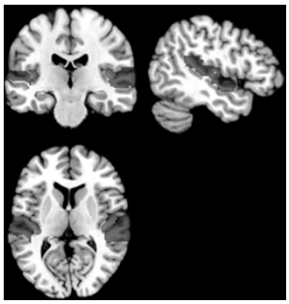


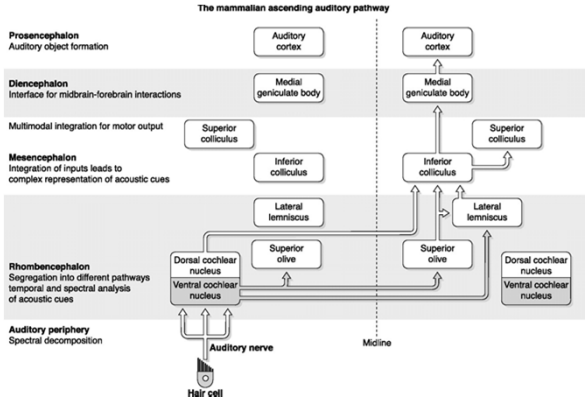




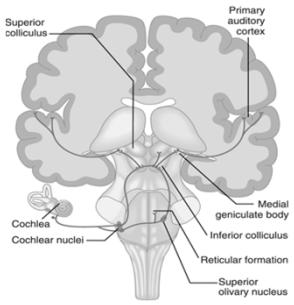
Primary Auditory Cortex

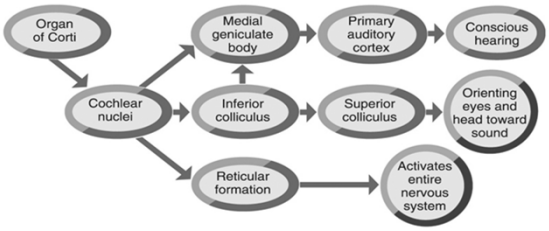
- Receives information from cochlea of both ears
- Has multiple tonotopic maps
- Ultimate goals:
  - Perceive sound objects
  - Locate sound
  - Make movements in relation to sound
- Conscious discrimination of loudness, pitch & timbre of sounds



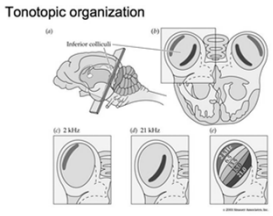


Auditory system



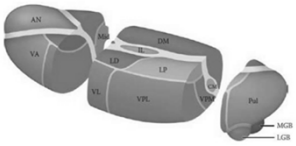


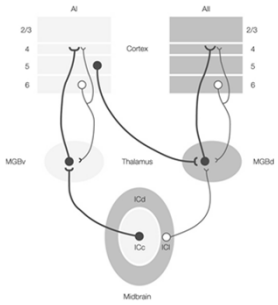
Tonotopic organization



- Just as visual system has retinotopic, point-to-point organization, auditory system has tonotopically organized.
- Different points in cochlear nuclei, inferior colliculus, medial geniculate nucleus & cortex respond maximally to different frequencies

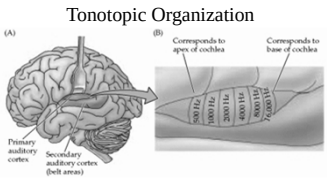
Auditory Pathways



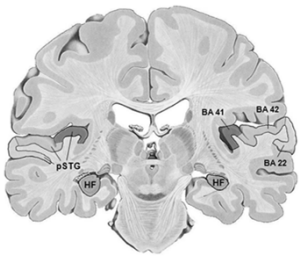


Cortical auditory representation

- Approximately 80% of cochlear nerve fibers cross in the trapezoid body.
- There are both ipsilateral & contralateral inputs to cortex
- So there is bilateral representation of each cochlear nucleus in both hemispheres

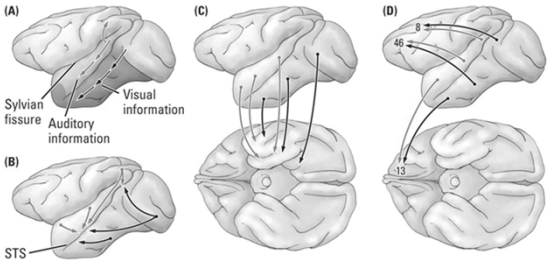


Transverse gyri of Heschl

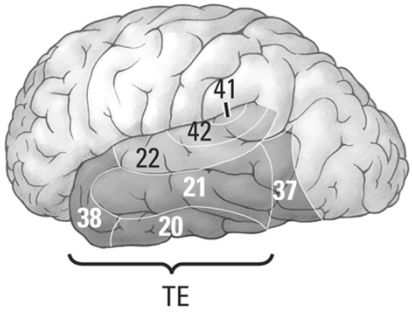


- Primary auditory cortex
  - BA 41, 42
- Secondary auditory association cortex
  - BA 22

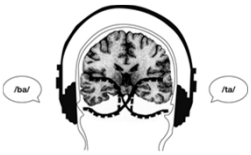
Temporal Lobe – auditory vs. visual areas

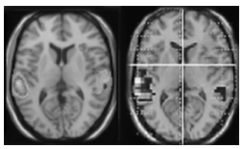


(B) Auditory and visual areas



Hemispheric asymmetry in auditory system





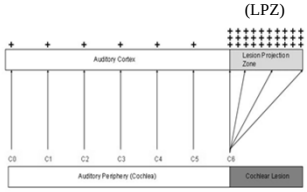
- Contralateral auditory extinction via dichotic listening
- Crossed auditory connections are more numerous & faster than ipsilateral projections
- Kimura (1964, 1967) – left hemisphere specialized for processing language-related sounds and
- Right hemisphere processes music-related sounds

Disorders of Auditory Processing

- Tinnitus
- Auditory hallucinations/illusions
- Sound localization defects
- Cortical deafness
- Auditory agnosia for speech (pure word deafness)
- Nonverbal sound agnosia
- Amusia
- Phonagnosia

Subjective Tinnitus

- Cochlear damage causes altered tonotopic organization in cortex.
- Neurons in the LPZ undergo 2 plastic changes:
  - Increase in spontaneous firing rate
  - Increase of the frequency representation of neurons that border the damaged region
- Loss of central inhibition of damaged regions and
- Cortical plasticity of neighboring regions of cortex that are still active.



Auditory hallucinations

- Most common symptom in schizophrenia - ~65% of cases.
- Usually fully formed verbal passages that appear to be coming from an external source
- Content usually hostile or paranoid
- Dierks et al. (1999) monitored paranoid schizophrenic patients in fMRI found
- Verbal hallucinations activated the following areas:
  - Primary auditory cortex
  - Broca's area
  - Speech zones of the posterior temporal cortex in left hemisphere
  - Activation of some limbic areas

Dierks, et al. Activation of Heschl's gyrus during auditory hallucinations. *Neuron* 22:615-621, 1999.

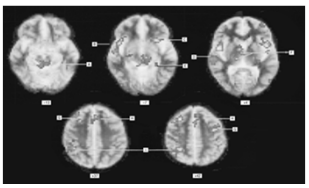
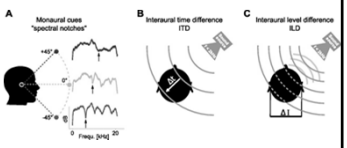


Figure Legend:

Group brain activation during random sampling of hallucinations. Five transverse sections through the brain, at different levels relative to the intercommissural plane (in millimeters). The right side of the brain is shown on the left side of each section. The colored areas are regions that were activated during auditory hallucinations, with the foci of maximal significance shown in yellow. The main activations (P<.001) were in the right inferior colliculus (A), the right and left insula (B and C), the left parahippocampal gyrus (E), the right superior temporal gyrus (D), and the right thalamus (F). Activation was also evident in the middle frontal (G) and anterior cingulate gyri (H), and in the right inferior and superior parietal lobule (I).

Sound localization defects

- Determining location of sound requires use of binaural hearing.
- There are no "spatial maps" in the cochlea.
- Sound from 2 ears converges onto neurons in auditory brainstem & cortex creating binaural representation.
- Neurons use the *interaural time difference* of sounds arriving from each ear to create a spatial sound map



This "textbook" explanation has been challenged recently.

Cortical deafness

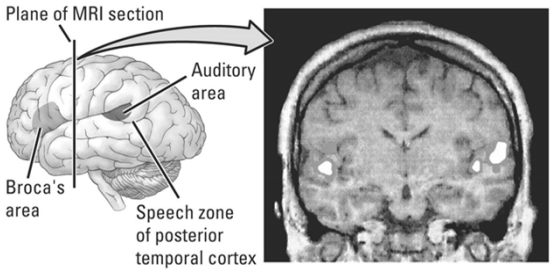
- Bilateral destruction of primary auditory cortex does not cause "deafness."
- Patients may feel deaf, but have normal auditory evoked potentials & audiometric sensitivity may be OK.
- Causes variety of cortical auditory disorders
- These lesions will cause deficits in:
  - localization of sounds
  - temporal auditory analysis
  - difficulties understanding speech
- Analogous to "blindsight"
- Many intact auditory pathways to auditory association cortices

Pure word deafness

- "auditory agnosia for speech"
- Lichteim (1885) – "inability to understand spoken words as an isolated deficit
- unaccompanied by disturbance of spontaneous speech or
- by severe disturbance in writing or understanding the printed word."
- "apperceptive agnosia"
- Hearing normal
- Not aphasic
- Comprehension of nonverbal sounds spared
- Patients complain speech sounds "like a foreign language" or "muffled."

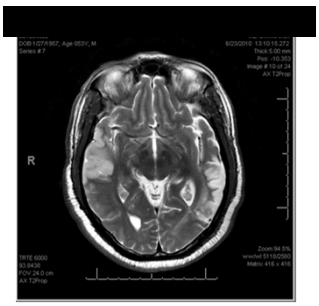
Pure word deafness

- Comprehension improved with context (knowing the topic of conversation) or when they can lip-read
- Preserved ability to comprehend paralinguistic aspects of speech (gesture, facial expression)
- Slowing presentation rate of words sometimes facilitates comprehension
- Lesion(s) either:
  - Bilateral, symmetric cortical-subcortical lesions involving anterior-superior temporal gyrus with some sparing of Heschl's gyrus
- OR
  - Unilateral lesion located subcortically in left temporal lobe destroying ipsilateral auditory radiation as well as callosal fibers from contralateral auditory region



Case of pure word deafness

- 53y.o., right-handed white male
- h/o HTN, DM, coronary artery disease, myocardial infarction, multiple cardioembolic CVAs
- June 2008, left, mid-temporoparietal CVA
- July 2010, right STG & MTG CVA



Case of pure word deafness

- Observations:
  - Speech = fluent
  - Prosody = normal
  - Articulation = normal
  - Paraphasic errors = none
  - Circumlocutions = none
  - BDAE Praxis
    - Buccofacial = 5/5, normal
    - Limb = 10/10, normal
  - Finger localization = 38/40, normal
- MAE Visual Naming – 39th %ile
- MAE Token Test – 2nd %ile (36)
- MAE Aural Comp. - <1st %ile (10)
- MAE Sentence Rep. - < 1st %ile (5)
- Phoneme Discrim. – 42nd %ile
- Word Fluency (COWA) -11th %ile
- MAE Reading Comp. – 17th %ile
- Writing to dictation – 4th %ile
- Sound Recognition - 34th %ile

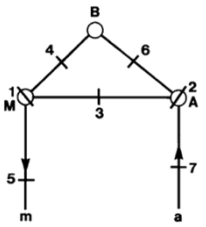
Case of pure word deafness

- Judgment of Line Orientation - 56 %ile
- RMT-Words – 82%ile
- RMT-Faces – 10th %ile
- Grooved Pegboard
  - Dominant (R) – 75th %ile
  - Nondominant (L) – 39th %ile
- Impairments of aural comprehension, sentence repetition & writing to dictation (spontaneous writing is normal)
- Without any evidence of clear aphasia, alexia, agraphia, or apraxia.
- Reading comprehension is normal.
- Memory, spatial judgment, fine motor dexterity = normal.

Case of pure word deafness

- Patient can read, write & speak normally
- Core difficulty is in comprehending spoken language.
- Is an auditory agnosia with selective impairment in speech-sound recognition
- Without impaired recognition of non-speech sounds or aphasia.
- Is the inability of auditory input to gain access to the phonological lexicon.
- Slightly less debilitating than Wernicke's aphasia & prognosis for eventual outcome is better.
- Use visual cues (lip-reading, facial expression) to aid comprehension; speak slowly; write important messages (reading is intact).

Wernicke-Lichtheim model

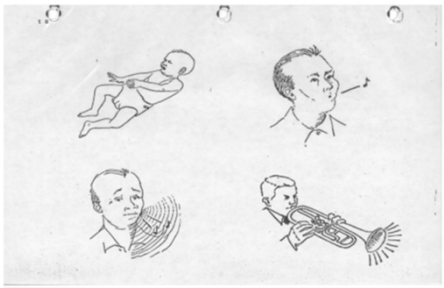


1. Broca's aphasia
2. Wernicke's aphasia
3. Conduction aphasia
4. Transcortical motor aphasia
5. Dyspraxia
6. Transcortical sensory aphasia
7. Pure word deafness

Nonverbal sound agnosia

- "auditory agnosia for non-speech sounds"
- Rarer than pure word deafness
- Isolated deficit of ability to recognize meaningful environmental sounds (e.g., telephone ringing)
- Variety of single case infarct locations – right STG & MTG, right P-T-O junction, right thalamus & inferior parietal lobe
- The following are normal:
  - Understanding language
  - Normal hearing (pure tone audiometry)
  - Normal brainstem auditory evoked potentials (BAERs)
  - Sound localization
- The following are abnormal:
  - Cannot identify common sounds
  - Delayed & attenuated cortical AEPs worse on the right
  - Left ear extinction on dichotic listening
  - Deficits of pitch discrimination and other aspects of amusia

Sound Recognition Test Example



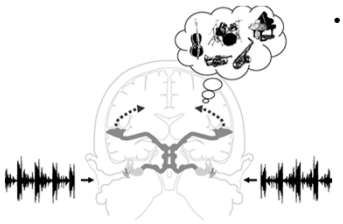
Amusia

- Refers to a range of distinct disorders
  - Vocal amusia
  - Loss of skilled instrumental ability (instrumental amusia)
  - Loss of ability to read or write music (musical alexia or agraphia)
  - Impaired recognition of music **(receptive amusia)**
  - Disorders of rhythm
- Receptive amusia
  - Refers to inability to appreciate various characteristics of heard music
- Characteristics may be affected to different degrees including
  - Pitch
  - Timbre
  - Loudness / intensity
  - Harmony
  - Rhythm

Amusia

- Primary auditory cortex of right temporal lobe (STG) vital for
  - Pitch, harmony, & timbre discrimination
  - Melody recognition, production, learning, and retention
- Zatorre (2002) – left temporal lobe plays major role in rhythm decoding – temporal grouping for rhythms
- Left hemisphere is specialized for processing speed of stimuli
- Right hemisphere is specialized for distinguishing frequency (pitch) differences.
- Cerebral organization of musical ability depends on training, experience & skill.

Musical Perception



- Dichotic listening studies show
  - Right hemisphere more important for processing musical & nonlinguistic sound patterns (prosody)
  - Left hemisphere is of major importance in processing of sequential, temporally organized material of any kind (including musical series).

Sensory (receptive) Amusia

- Not well defined –no subtypes based upon music subcomponents.
- Amusia is common in most cases of auditory sound agnosia
- Also common in cases of aphasia & pure word deafness
- But can also occur independently of these conditions
- Case reports of patients with
  - normal perception of speech & environmental sounds, but
  - Impaired perception of tunes (melodies), prosody, and voice.
- Some comprehensive tests that assess multiple musical subskills
  - Seashore Measures of Musical Talent – not used much now
  - Schuppert (2000) – German tests
  - Tonometric.com – (Mandell) simple online tests of pitch & rhythm

Phonagnosia

- Inability to recognize the identity of familiar voices
- despite preserved recognition of spoken words & environmental sounds.
- Phonagnosia is the auditory analogue of prosopagnosia in the visual modality.
- Hypothetically, represents a disconnection between unimodal auditory association cortex
- And transmodal temporoparietal cortex in the
- Right hemisphere.
- An fMRI study of familiar vs. unfamiliar voices showed activation in posterior cingulate region including retrosplenial cortex (Shah, 2001).

Auditory processing disorders – Lesion summary

- Tinnitus - cochlea
- Auditory hallucinations/illusions – undetermined CNS
- Sound localization defects – either left or right primary auditory cortex
- Cortical deafness – bilateral primary auditory cortex
- Amusia – right temporal lobe sparing primary auditory cortex
- Auditory agnosia for speech (pure word deafness) – either left or bilateral anterior STG partially sparing Heschl's gyrus
- Nonverbal sound agnosia – right temporal lobe (STG, MTG) sparing primary cortex, ? Right inferior parietal lobule
- Phonagnosia – deep white matter underlying right temporal lobe, ? Retrosplenial area

Table 15.1 Summary of major symptoms of temporal-lobe damage

Symptoms	Most probable lesion site	Basic reference
Disturbance of auditory sensation	Areas 41, 42, 22	Vignolo, 1969 Hécaen and Albert, 1978
Disturbance of selection of visual and auditory input	Areas TE, superior temporal sulcus	Sparks et al., 1970 Dorff et al., 1965
Disorders of visual perception	Areas TE, superior temporal sulcus, amygdala	Milner, 1968 Meier and French, 1968
Disorders of auditory perception	Areas 41, 42, 22	Samson and Zatorre, 1988 Swisher and Hirsch, 1972
Disorders of music perception	Superior temporal gyrus	Zatorre et al., 2002
Impaired organization and categorization of material	Areas TE, superior temporal sulcus	Wilkins and Moscovitch, 1978 Read, 1981
Poor contextual use	Area TE	Milner, 1958
Disturbance of language comprehension	Area 22 left	Hécaen and Albert, 1978
Poor long-term memory	Areas TE, TF, TH, 28	Milner, 1970
Changes in personality and affect	Areas TE, plus amygdala	Blumer and Benson, 1975 Pincus and Tucker, 1974
Changes in sexual activity	Amygdala, plus?	Blumer and Walker, 1975

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