

Visual Object Recognition

Computational Models and Neurophysiological Mechanisms

Neurobiology 130/230. Harvard College/GSAS 78454

Web site: <http://tinyurl.com/visionclass>
→ Class notes, Class slides, Readings Assignments

Location: Biolabs 2062

Time: Mondays 03:30 – 05:30

Lectures:

Faculty: Gabriel Kreiman and invited guests

TA: Yuchen Xiao

Contact information:

Gabriel Kreiman	Yuchen Xiao
gabriel.kreiman@tch.harvard.edu	yxiao@g.harvard.edu
617-919-2530	

Office Hours: After Class. Mon 05:30-06:30 or by appointment

Visual Object Recognition

Computational Models and Neurophysiological Mechanisms

Neurobiology 230. Harvard College/GSAS 78454

Class 1. Introduction to pattern recognition [Kreiman]

Class 2. Visual input. Natural image statistics. The retina. [Kreiman]

Class 3. Lesion and neurological studies of visual deficits in animals and humans. [Kreiman]

Class 4. Psychophysics of visual object recognition [Jiye Kim]

October 9: University Holiday

Class 5. Introduction to the thalamus and primary visual cortex [Camille Gomez-Laberge]

Class 6. Adventures into *terra incognita*. Neurophysiology beyond V1 [Frederico Azevedo]

Class 7. First steps into inferior temporal cortex [Carlos Ponce]

Class 8. From the highest echelons of visual processing to cognition [Leyla Isik]

Class 9. Correlation and causality. Electrical stimulation in visual cortex [Kreiman].

Class 10. Theoretical neuroscience. Computational models of neurons and neural networks. [Kreiman]

Class 11. Computer vision. Towards artificial intelligence systems for cognition [Bill Lotter]

Class 12. Vision and Language. [Andrei Barbu]

Class 13. **[Extra class]** Towards understanding subjective visual perception. Visual consciousness. [Kreiman]

FINAL EXAM

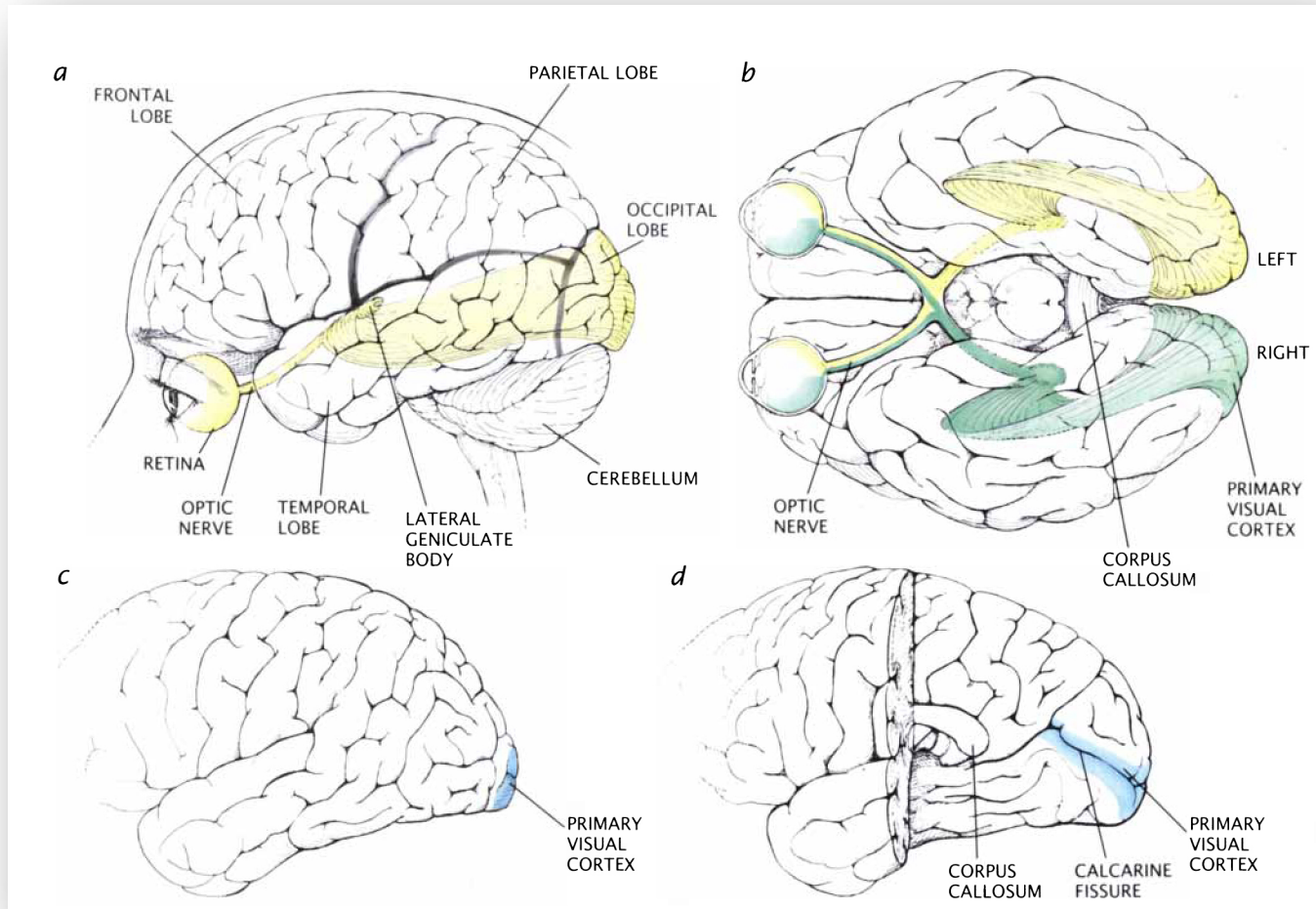
The discovery of visual cortex

- Initial retinotopic mapping in primary visual cortex was derived from brain injuries sustained by the Russia-Japanese War and First World War soldiers (Inouje, Holmes, Riddoch)



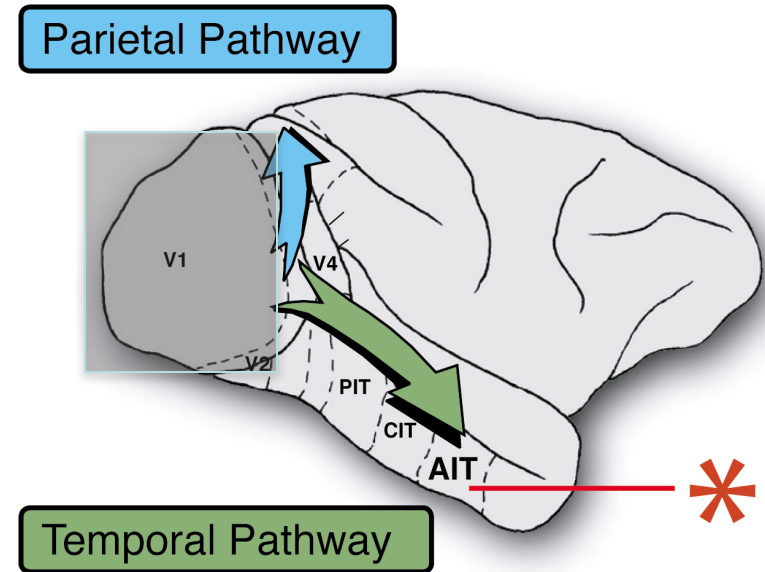
Glickstein, The discovery of the visual cortex. Scientific American 1988
Holmes, Disturbances of visual orientation. British Journal of Ophthalmology 1918.

Basic path of visual signals from the eyes to primary visual cortex

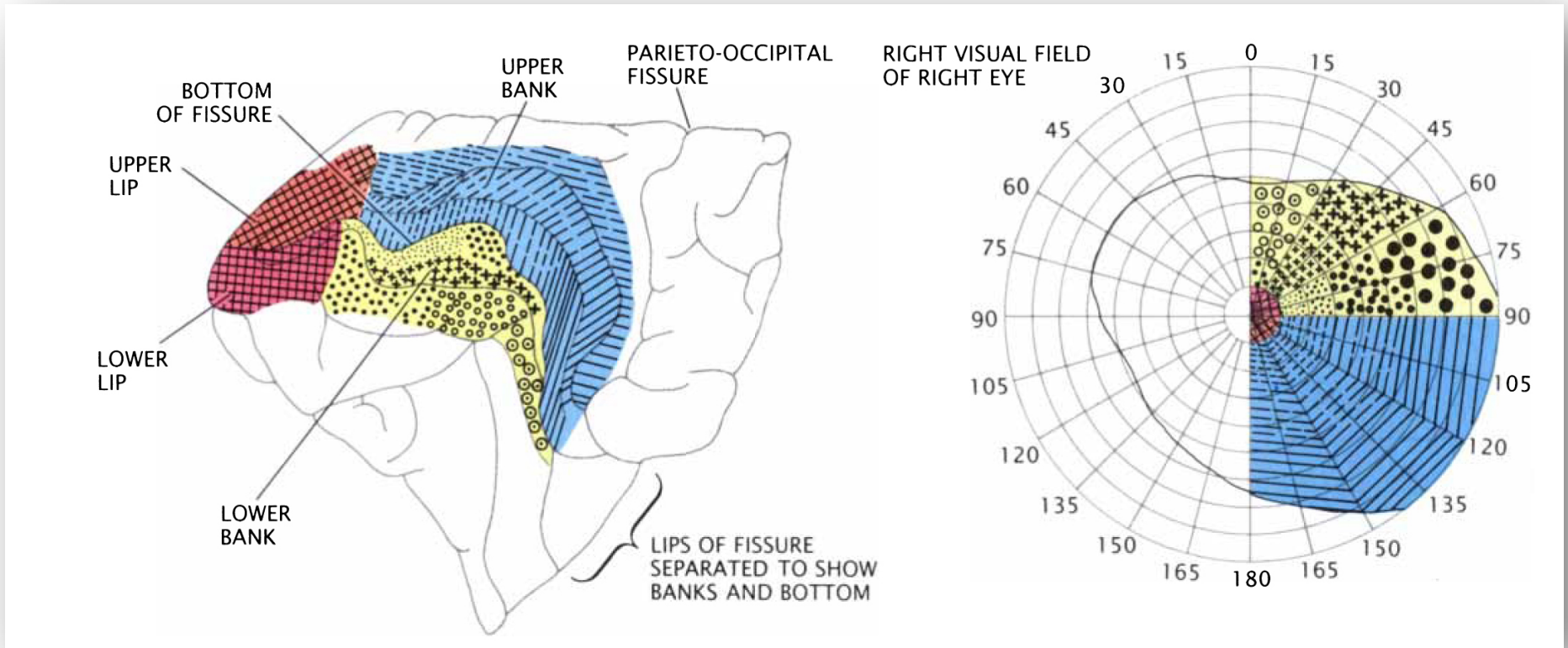


V1 lesions lead to topographically specific scotomas

- The involvement of primary visual cortex (V1) in visual processing was quite clear early on
- Vascular damage, tumors, trauma studies
- Visual field deficits contralateral to the lesion
- Shape and color discrimination are typically absent



How the visual field maps onto the visual cortex



Note the disproportionately large representation of the fovea

Blindsight

“Blindsight”: persistent visual function in the hemianopic field

- Some subjects detect presence/absence of light, some can even localize light.
- Some subjects can even discriminate orientation, color and direction of motion.
- In some cases, there may be intact islands within the blind field
- In some cases, LGN-extrastriate pathways can subserve visual function
- In some cases, subcortical pathways could be responsible

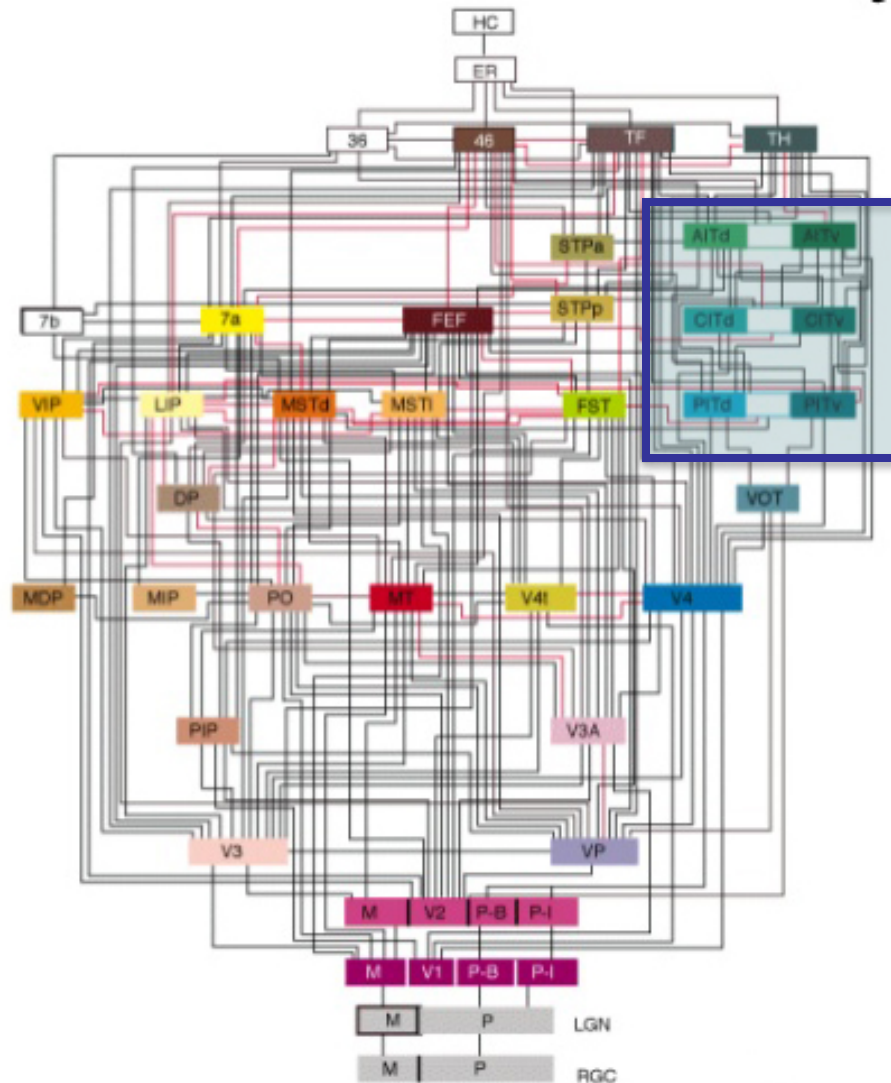
Is there any visual function beyond V1?

In human subjects there is no evidence that any area of the cortex other than the visual area 17 is important in the primary capacity to see patterns. . . . Whenever the question has been tested in animals the story has been the same. (Morgan and Stellar, 1950)

. . . visual habits are dependent upon the striate cortex and upon no other part of the cerebral cortex. (Lashley, 1950)

. . . image formation and recognition is all in area 17 and is entirely intrinsic. . . . the connections of area 17 are minimal. (Krieg, 1975)

Visual system circuitry (macaque monkeys)



Initial examinations of the temporal cortex

The Kluver-Bucy syndrome

Earliest reports: Brown and Schafer 1888

Kluver and Bucy. Preliminary analysis of the functions of the temporal lobes in monkeys.
Archives of Neurology and Psychiatry, (1939). **42**: 979-1000.

- Bilateral removal of temporal lobe in rhesus monkeys
- Original reports included both visual and non-visual areas
- Original reports: loss of visual discrimination, increased tameness, hypersexuality, altered eating habits

Refined by Mishkin 1954, Holmes and Gross 1984

Moral: Location, location, location. The specific details of the lesion matter.

Lesions in macaque monkey IT cortex

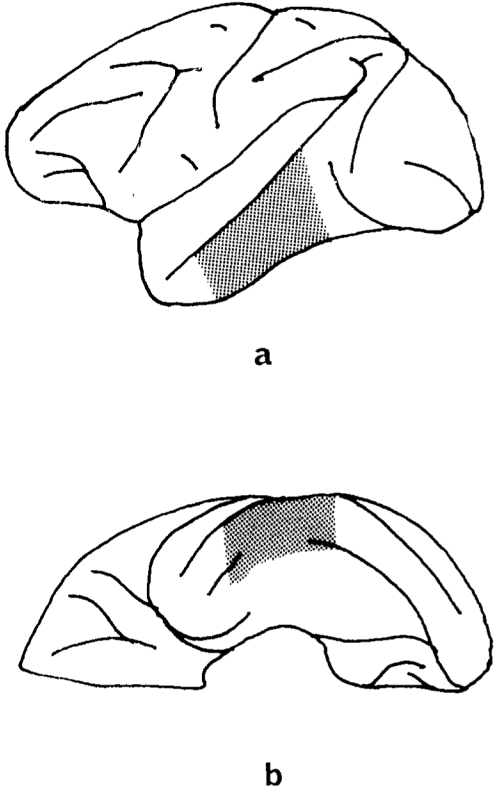


FIGURE 1. Classical inferotemporal lesion in *Macaca mulatta*: (a) lateral view and (b) ventral view.

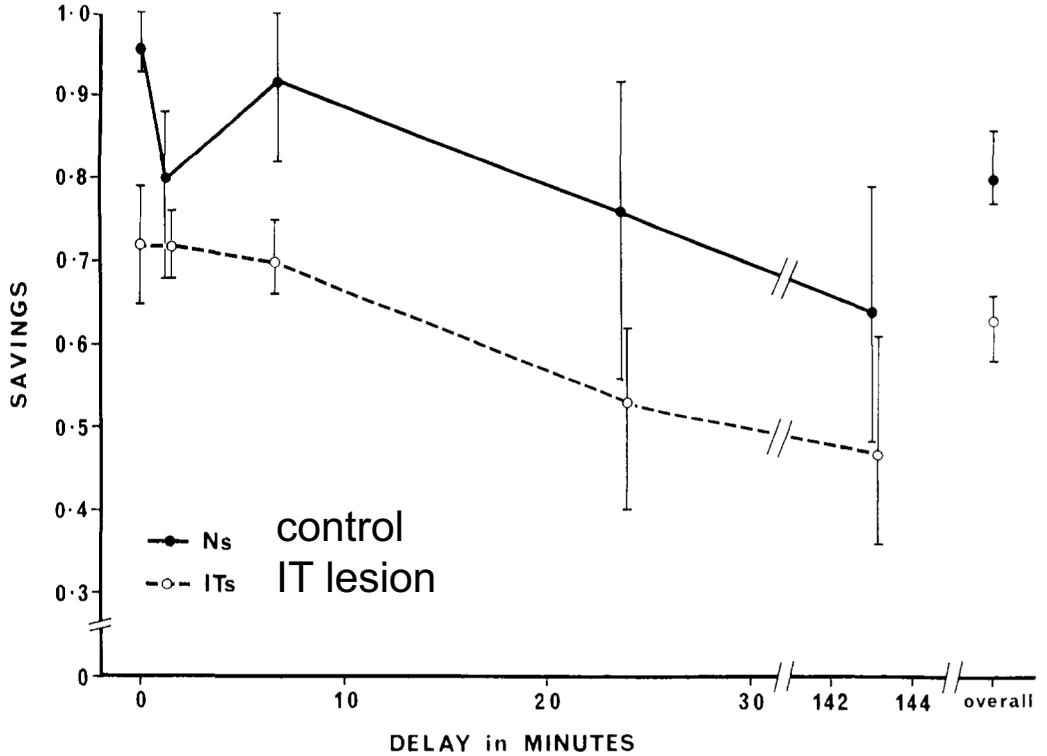


FIGURE 4. Group mean savings scores (and ranges) for object discriminations plotted against length of test-retest delay. (Points at extreme right are from all delays pooled together. Data from Weiskrantz & Mingay, Note 2).

L = errors in original learning
 R = errors on retest
 Savings = $(L-R)/(R+L)$

Lesions in macaque monkey IT cortex

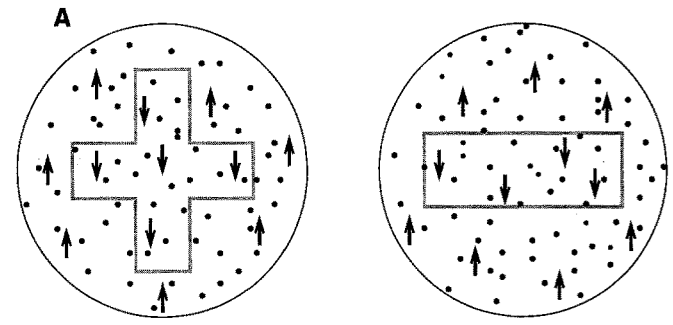
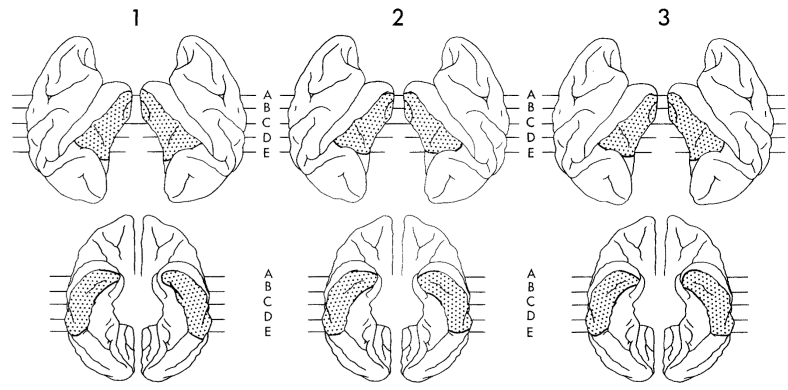
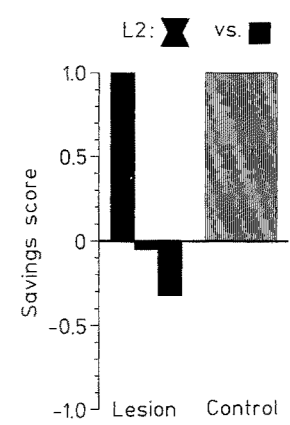
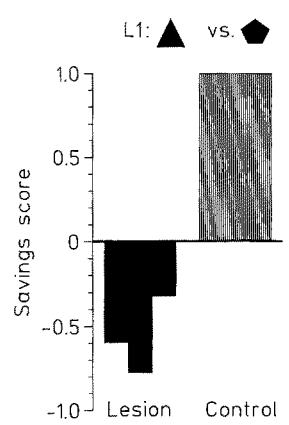
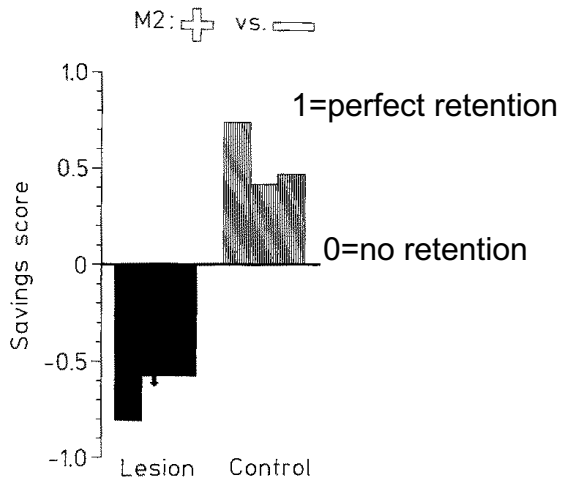
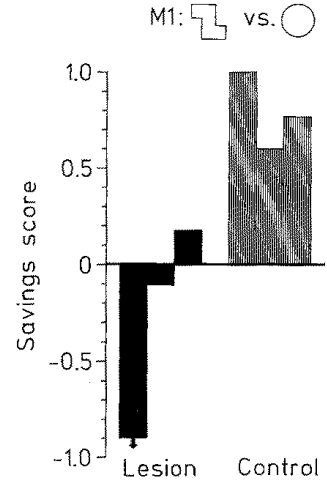


Fig. 3A-E. Surface-view reconstructions of the IT lesions in cases 1-3. Each reconstruction is displayed on standard lateral and ventral surface views of the cerebral hemisphere. A-E Indicate the anterior-posterior level for each section illustrated in Figs. 1 and 2

$$\text{savings} = \frac{(\text{time to threshold}_{\text{preop}} - \text{time to threshold}_{\text{postop}})}{(\text{time to threshold}_{\text{preop}} + \text{time to threshold}_{\text{postop}})}$$

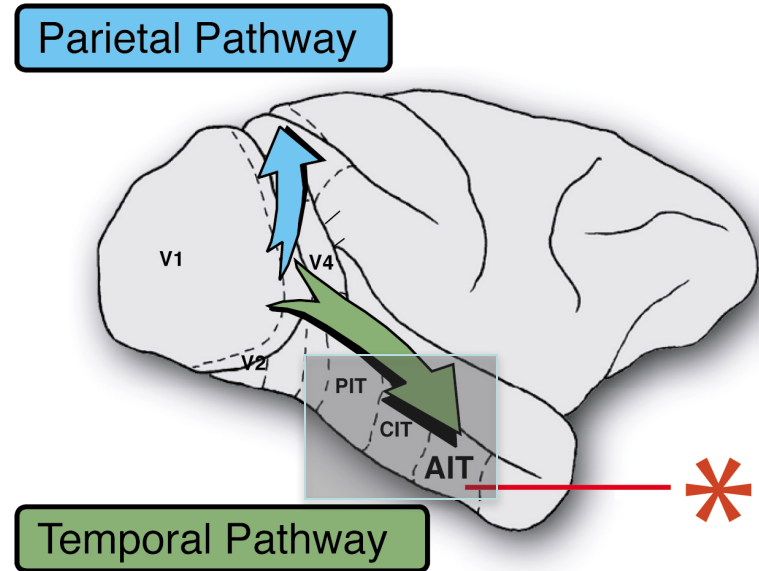


Form-from-motion

Form-from-luminance

Lesions in macaque monkey IT cortex

- Bilateral removal of IT cortex
- Impaired in learning visual discriminations
- Impaired in retaining discriminations learned before lesion
- Applies to objects, patterns, orientation, size, color
- Severity of the deficit typically correlated with task difficulty
- Defect is long-lasting
- Deficit appears to be restricted to vision and not touch, olfaction or audition
- No apparent visual acuity, orientation deficits, social deficits, none of the “psychic blindness” effects of Kluver-Bucy.



“Natural” lesions in the human brain

- Carbon monoxide poisoning
- Bullets and other weapons
- Viral infections
- Bumps
- Partial asphyxia (particularly during the first weeks of life)
- Tumors
- Hydrocephalus
- Stroke

Cortical visual deficits in humans – dorsal stream

- Akinetopsia – Specific inability to see motion
(Zeki 1991 Brain 114: 811-824)
- Hemineglect
(Bisiach & Luzzatti 1978; Farah et al. 1990)
- Simultanagnosia (Balint) – Inability to see more than one or two objects in a scene
- Optic ataxia (Balint) – Inability to make visually guided movement

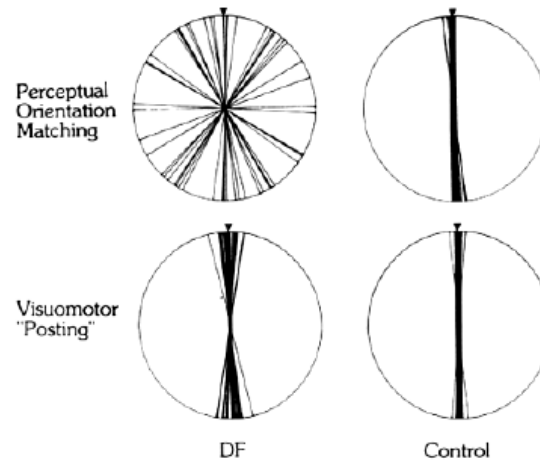
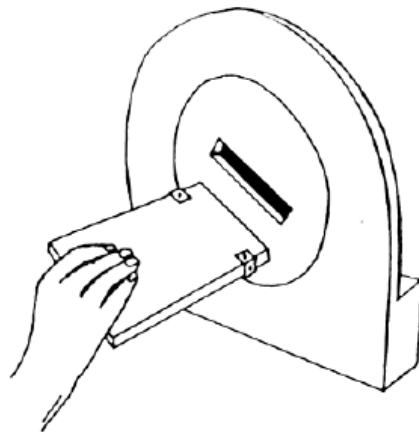
Vision for action can be dissociated from shape recognition

Subject with temporal lobe damage

Severely impaired shape recognition

Yet, appropriate reach response

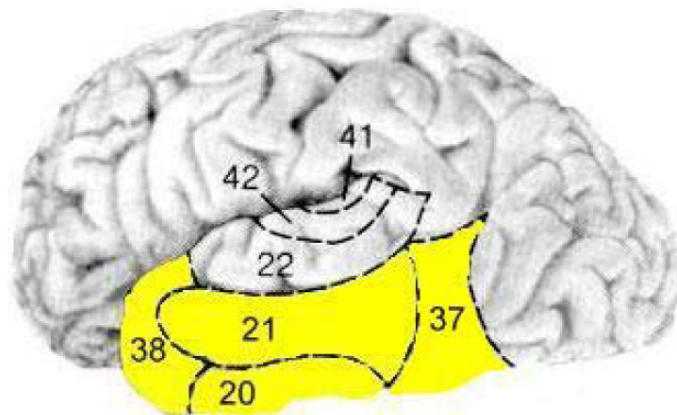
And correct behavioral performance in visuo-motor tasks



Goodale and Milner. Separate visual pathways for perception and action. Trends in Neurosciences. 1992 **15**:20-25

Cortical visual deficits in humans – ventral stream

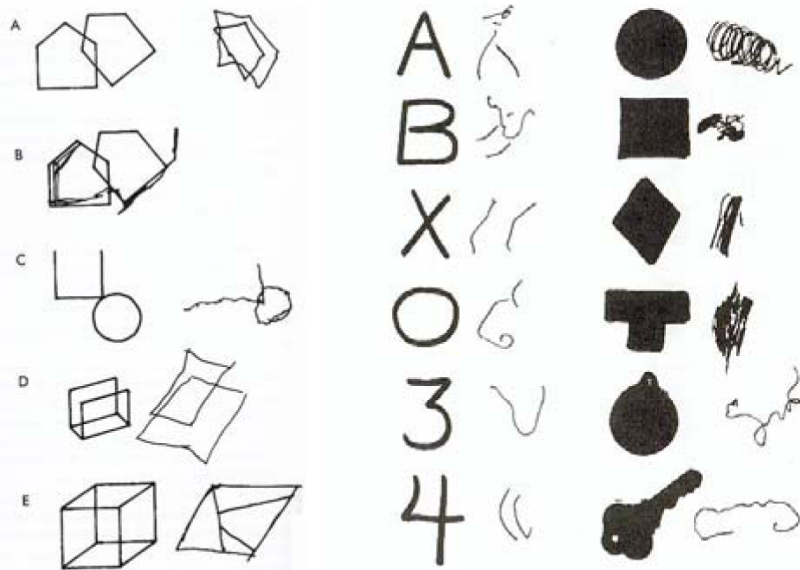
- Achromatopsia (Cortical color blindness) – Specific inability to recognize colors (Zeki 1990 Brain 113:1721-1777)
- Dutton (2003) describes a patient who showed “... no vision for anything that was not moving...” Eye (2003) 17, 289-304.
- Object agnosias
Warrington and Shallice. Brain (1984) 107:829-854



Areas typically affected
in object agnosias

Apperceptive visual agnosia

Copying shapes



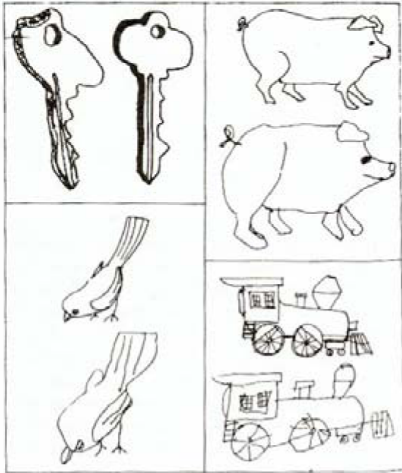
- Patient cannot name, copy or match simple shapes
- Acuity, color recognition and motion perception are preserved
- Bilateral damage to extrastriate visual areas

Matching shapes



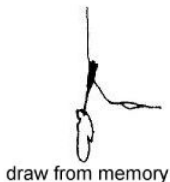
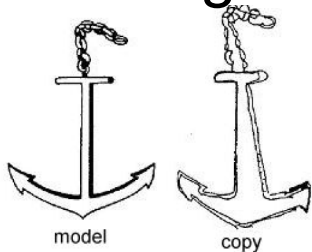
Associative visual agnosia

Copying from templates



- Subject can copy complex drawings, match complex shapes and use the objects correctly
- Subject cannot identify (name) those shapes
- Subject cannot draw from memory
- Acuity, color recognition and motion perception are preserved
- Bilateral lesion of the anterior inferior temporal lobe

Drawing from memory



Example: category-specificity in object agnosia

Table 4
Percentage of correct responses in object recognition and manipulation recall tests performed with real objects

Categories	Number of items	Object recognition	Manipulation gestures
Body parts	12	100	100
Common objects	25	60	96
Fruits and vegetables	15	0	0
Musical instruments	5	0	40

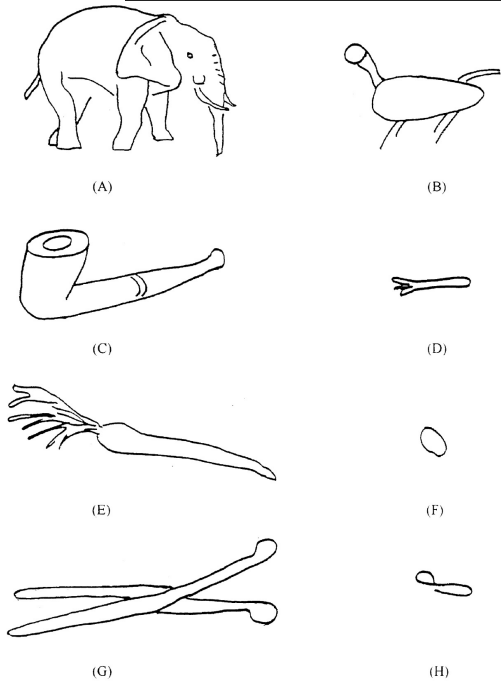


Table 5
Percentage of correct responses in object recognition and manipulation recall tests performed with pictured objects and percentage of correct responses in object verbal definitions

Categories	Pictures of objects			Name of objects
	Number of items	Object recognition	Manipulation gestures	Definition
Group I				
Animals	46	0	0	2.2
Fruits and vegetables	24	0	0	4.2
Musical instruments	9	0	0	0
Group II				
Furniture	14	64.3	71.4	71.4
Vehicles	10	70	80	80
Tools	12	58.3	58.3	91.7
Body parts	12	83.3	83.3	100
Kitchen utensils	14	57.1	57.1	85.7
Clothes	19	73.7	68.4	84.2

Fig. 1. Examples of J.M.C.'s drawings from a model and from memory on verbal command, respectively: (A) and (B) elephant; (C) and (D) pipe; (E) and (F) carrot; (G) and (H) scissors. The copying task was carried out using pictured objects [(A) and (C)] and real objects [(E) and (G)].

Prosopagnosia

Agnosia (Gr): “not knowing”
Prosopon (Gr): face

- Inability to recognize faces with unimpaired performance in other visual recognition tasks

- The most studied form of visual agnosia (e.g., Bodamer 1947, Landis et al. 1988, Damasio et al. 1982)

- Very rare

- Acquired prosopagnosia, typical after brain damage (c.f. “congenital prosopagnosia”)

- Typically caused by strokes of the right posterior cerebral artery

- Fusiform and lingual gyri

- Ongoing debates about the extent to which the deficit is specific for faces (e.g. Gauthier et al. 2000)

Table 1 Identity recognition and familiarity ratings for target and nontarget faces (patient E.H.)

	N	Identity recognition (% correct)	Average familiarity rating (s.d. in parentheses)
Retrograde-family experiment			
Target	8	0	6.0 (0.0)
Nontarget	42	—	6.0 (0.0)
Retrograde-famous experiment			
Target	8	0	6.0 (0.0)
Nontarget	42	—	6.0 (0.0)

Damasio et al 1990

Congenital prosopagnosia

- Deficits apparent from early childhood
- No clear neurological deficit
- Extremely rare
- Intact sensory functions
- Normal intelligence
- Able to detect face presence
- Subjects rely on voice, clothes, gait accessories.
- No comparison basis. Subjects may be unaware of their deficit!
- Failure to recognize even family members or self



There are several claims about object-specific agnosias that do not involve faces

Visual agnosias for objects, topography, body parts, animals, letters and numbers (e.g. Hecaen and Albert 1978)

“Inanimate” versus “animate” objects

“Manipulable” versus “Non-manipulable” objects

“Concrete” concepts versus “Abstract” concepts

In addition to the previous generic concerns about lesion studies:

Many of these deficits are not exclusively visual (sometimes subjects also show non-visual deficits)

What is a “living” object? Does the definition depend on movement (what about cars, what about flowers)? Does the definition depend on “Man-made” objects (what about a microscopic image of bacteria or yeast)?

Typically, studies are quite concerned about sub/supra-ordinate and other semantic distinctions, less so with basic visual properties such as contrast, size, etc.

Some general remarks about lesion studies (general)

- Distinction: local effects and “fibers of passage” effects
- It is essential to ask the right questions
 - e.g.1: For a long time, it was believed that there was nothing wrong with split-brain subjects after callosotomy
 - e.g.2: For a long time, many investigators believed that there was no visual function beyond V1
- Distinction: immediate effects and long-term effects. Beware of plasticity!
- Compensatory mechanisms.
 - There are two hemispheres. Effects due to unilateral lesions could be masked by activity in the other hemisphere
 - Other brain areas may play compensatory roles as well

Lesion studies in non-human animals

Tools to study the effects of removing or silencing a brain area

- Lesions
- Cooling
- Pharmacology
- Imaging combined with cell-specific ablation
- Gene knock-outs / knock-ins

General remarks about lesion studies (non-humans)

- It may be difficult to make anatomically-precise lesions
- Behavioral assessment may pose a challenge
- Subjective perception can be explored in non-human animal models but it is not easy

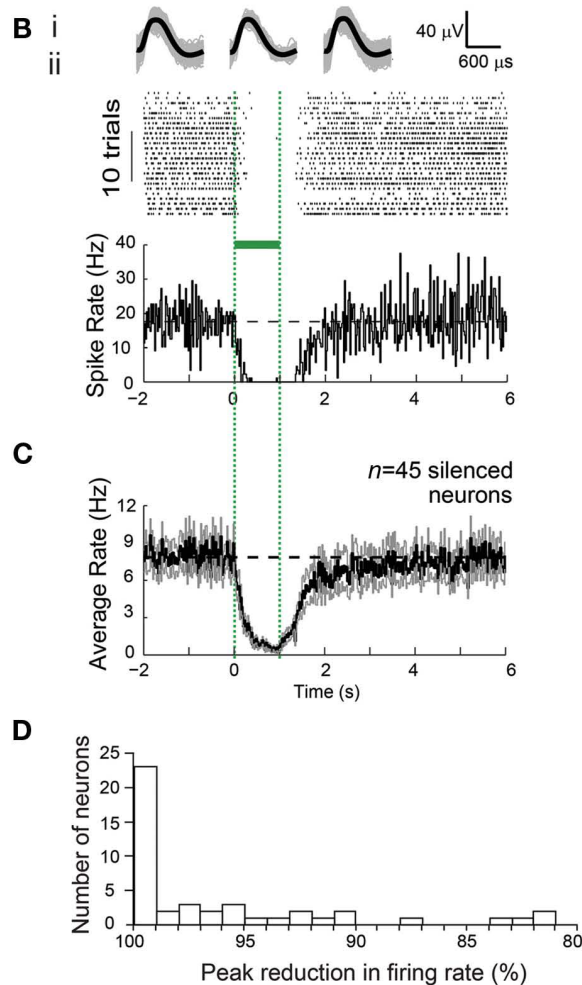
General remarks about lesion studies (humans)

- In general, human lesions are not well-delimited. Beware of multiple effects.
- In many studies, $n=1$.
- In studies where $n>1$, it may be hard to compare across subjects because of the differences in the extent of brain damage.
- In some studies, it may be difficult to localize the brain abnormality (e.g. autism)

Towards high-resolution lesion studies in non-human animals

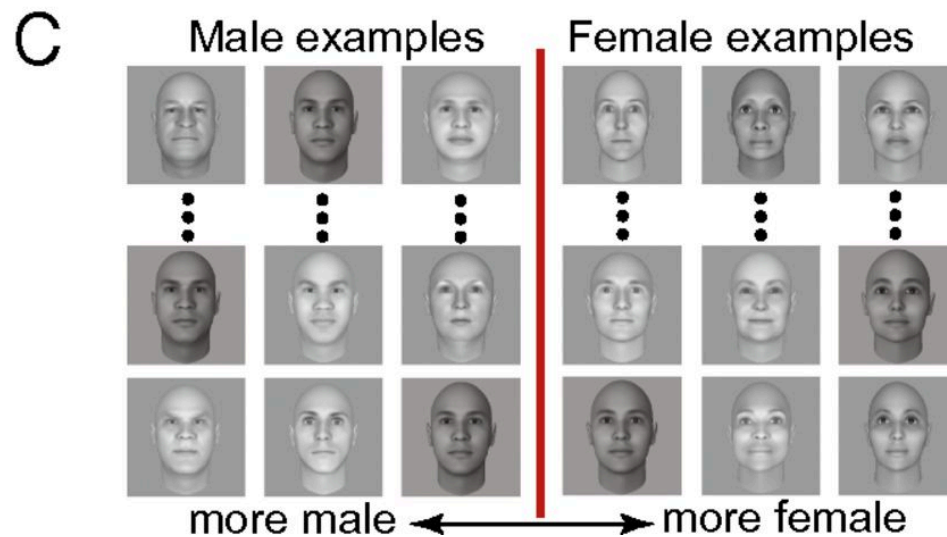
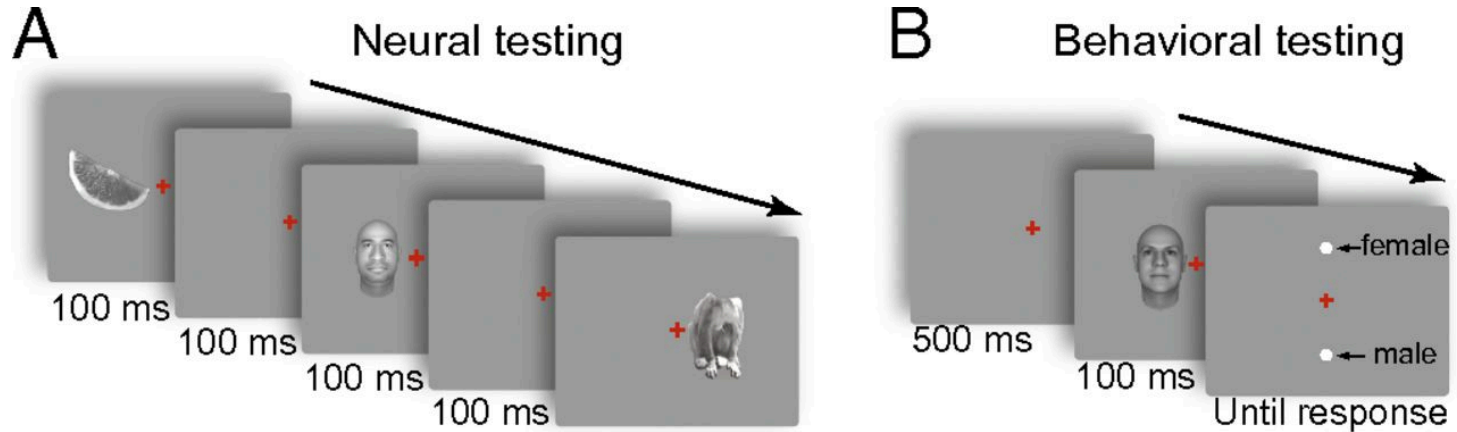
- Molecular biology can provide specificity in the study of neural circuits
- Promoters can direct gene expression to specific neuronal populations/layers/areas (e.g. Berman et al, PNAS 2002)
- Several molecules could be used to transiently inactivate neurons (e.g. Slimko et al, J. Neuroscience 2002)
- Trangenics for rodents, virus injection for monkeys (e.g. Lois et al Science 2002)
- Temporal control
- Reversibility

Towards high-resolution lesion studies in non-human animals

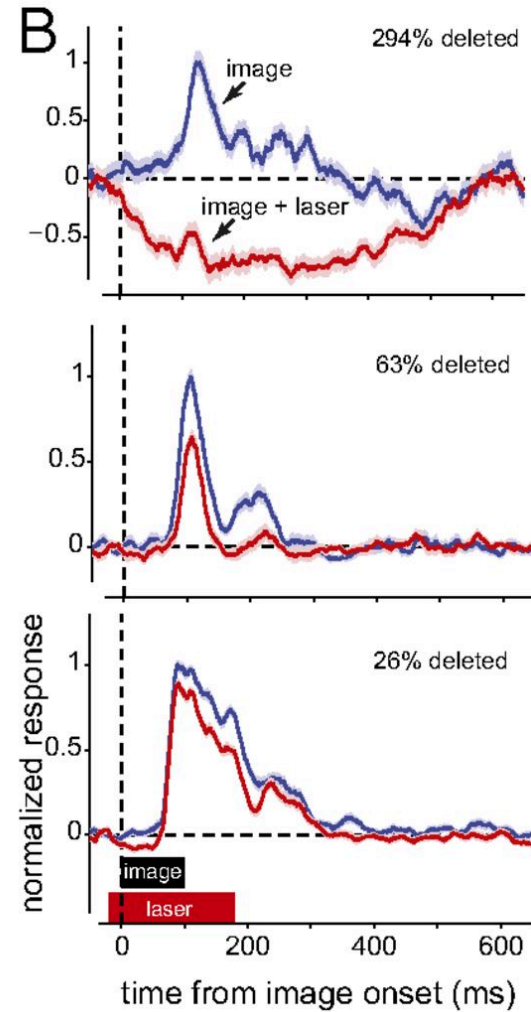
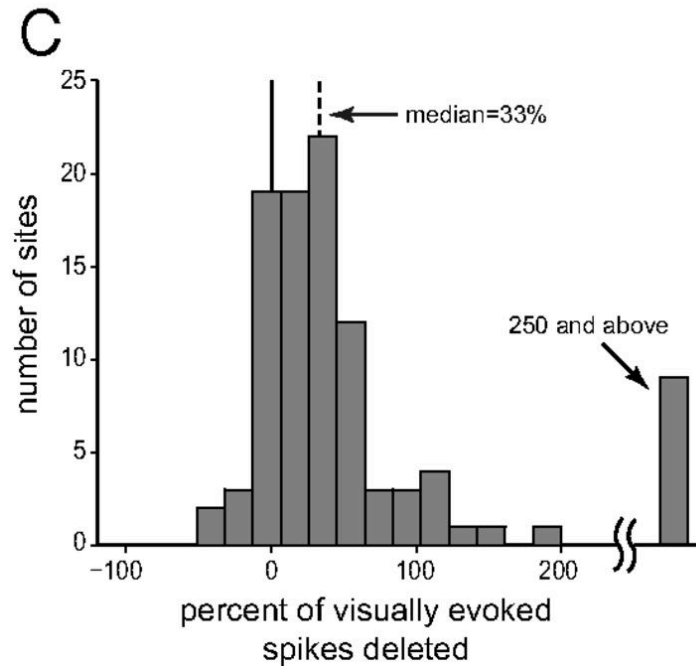


ArchT-mediated silencing of cortical neurons in the awake primate brain

High-resolution lesions in monkeys impair object recognition

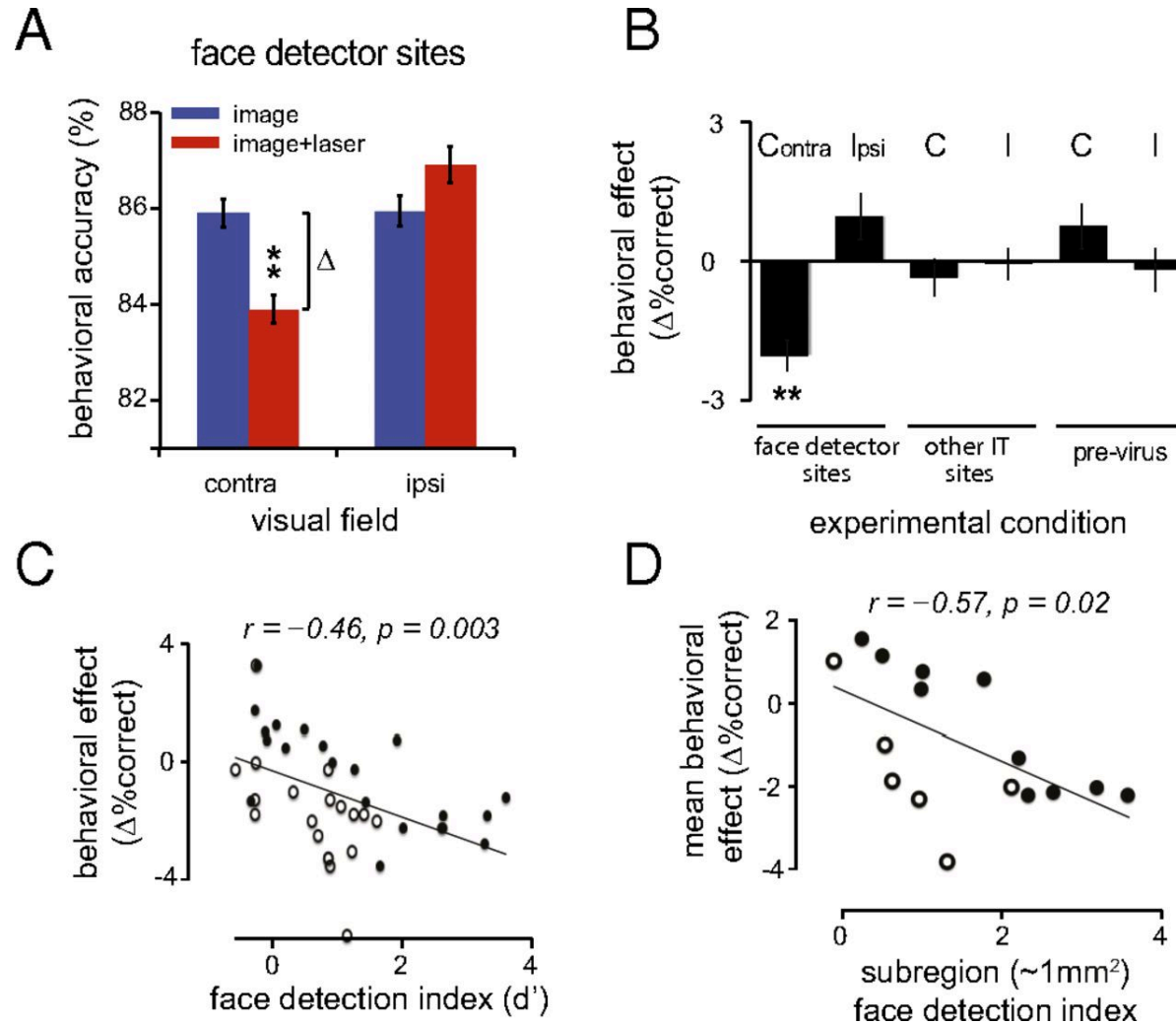


Neural effects of optogenetic perturbation of the IT cortex.

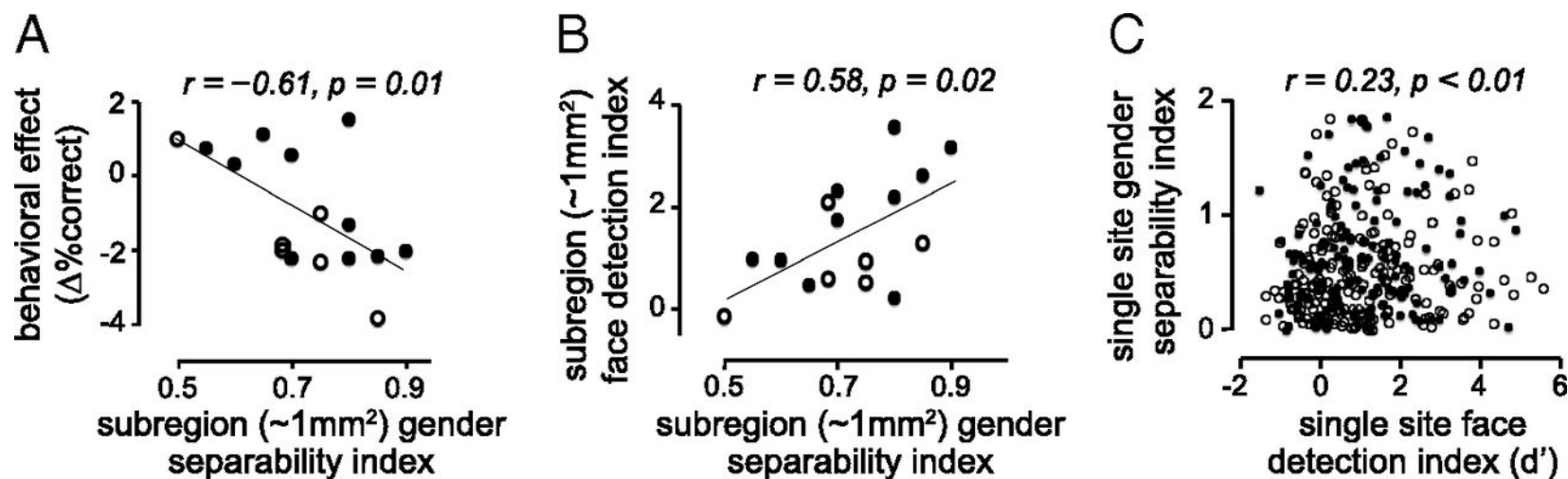


Arash Afraz et al. PNAS 2015;112:6730-6735

Behavioral effects of optogenetic suppression of local IT neural activity

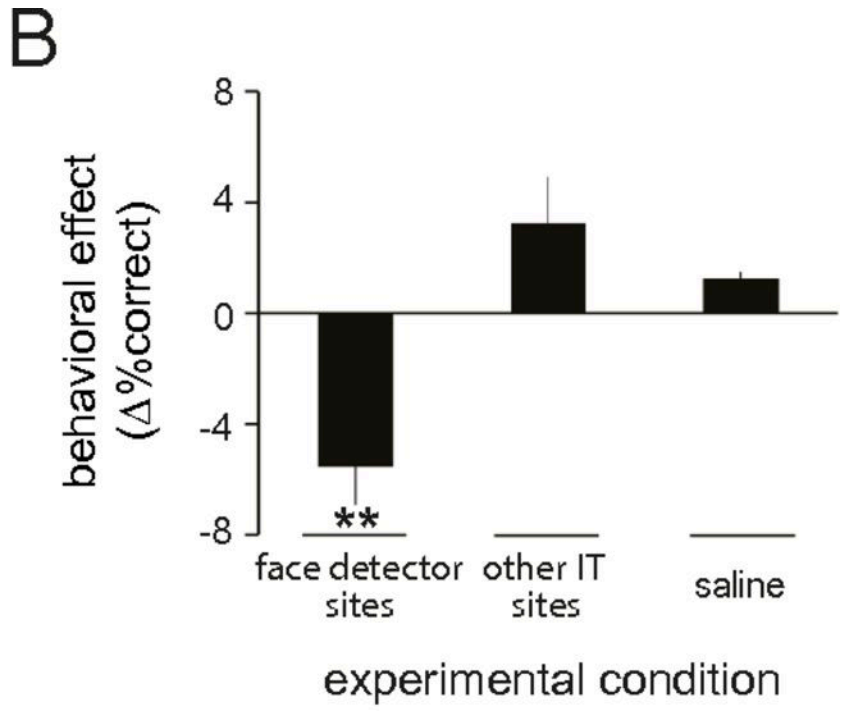
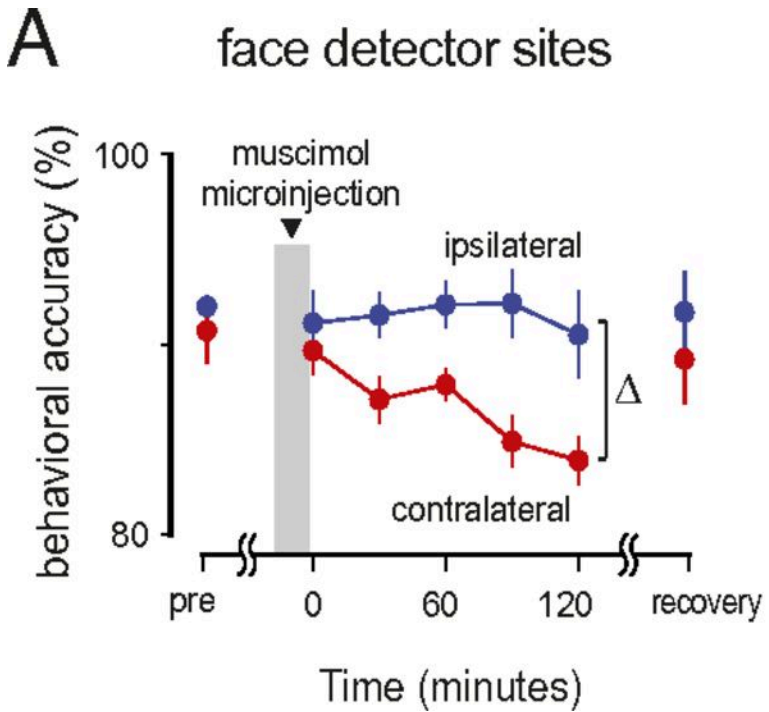


Explicit encoding of facial gender in CIT. (A) The relationship between explicit neural encoding of facial gender in various IT subregions and the effect of photosuppression of those subregions on face gender-discrimination behavior.



Arash Afraz et al. PNAS 2015;112:6730-6735

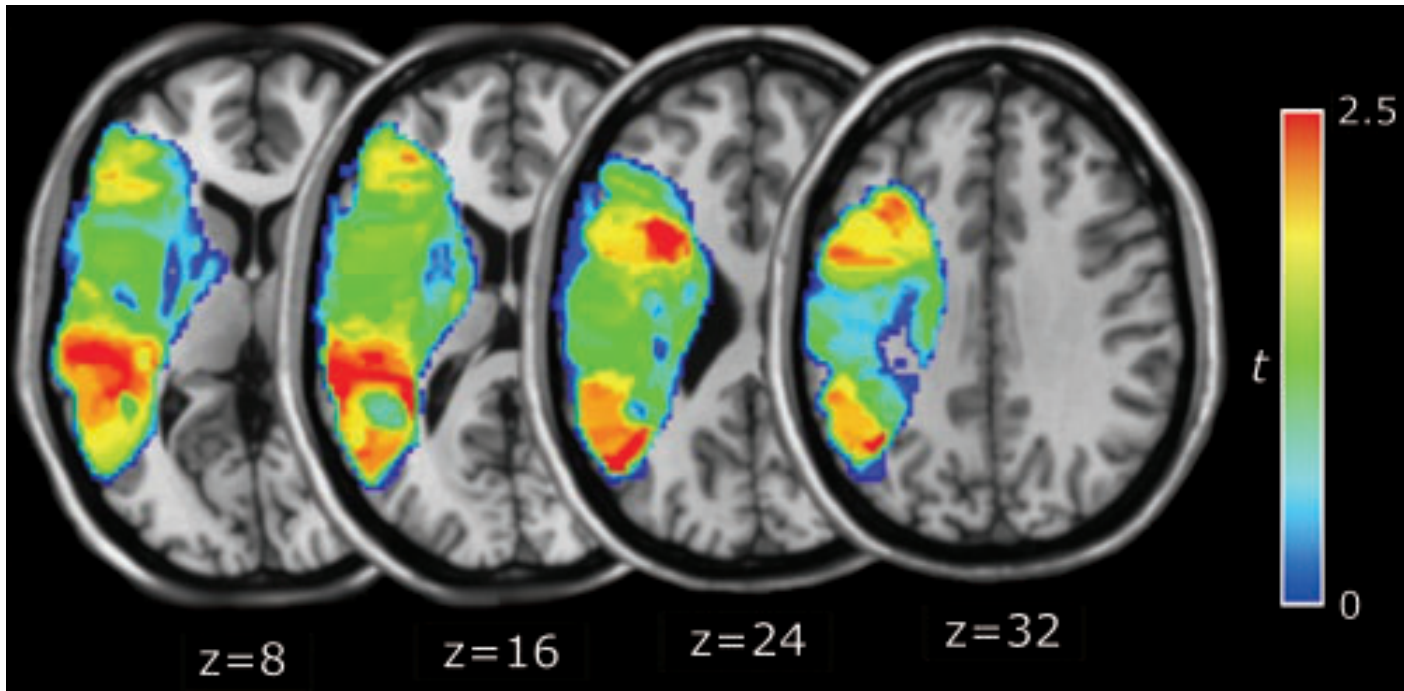
Behavioral effect of drug microinjection in IT cortex



Towards high resolution studies in humans

- Most of the molecular biology tools in the previous slide cannot be easily applied to humans
- High-resolution structural MR images could point to structural abnormalities at the sub-mm scale
- Novel MR-based imaging techniques can provide information about white matter and about coarse connectivity maps
- Needed: detailed anatomical comparisons across subjects (it is conceivable that many long discussions in the literature are based on different lesion patterns)
- Needed: controlled psychophysics studies

These approaches are seeing some use!



- This is not fMRI!
- Relationship between lesion location and action-perception deficits in 60 lesion patients

Saygin 2007

Cited works

- Afraz et al. PNAS (2015);112:6730-6735
- Behrmann, M., & Avidan, G. (2005). Congenital prosopagnosia: Face-blind from birth. *Trends in cognitive sciences*, 9(4), 180-187.
- Berman, B. P., Nibu, Y., Pfeiffer, B. D., Tomancak, P., Celniker, S. E., Levine, M., ... & Eisen, M. B. (2002). Exploiting transcription factor binding site clustering to identify cis-regulatory modules involved in pattern formation in the Drosophila genome. *Proceedings of the National Academy of Sciences*, 99(2), 757-762.
- Bisiach, E., & Luzzatti, C. (1978). Unilateral neglect of representational space. *Cortex*, 14(1), 129-133.
- Bodamer, J. (1947). Die prosop-agnosie. *Archiv für Psychiatrie und Nervenkrankheiten*, 179(1-2), 6-53.
- Brown, S., & Schafer, E. A. (1888). An investigation into the functions of the occipital and temporal lobes of the monkey's brain. *Philosophical Transactions of the Royal Society of London. B*, 303-327.
- Britten et al (1992). Effects of inferotemporal cortex lesions on form-from-motion discrimination in monkeys. *Experimental Brain Research*. 88:292-302.
- Damasio, A. R., Damasio, H., & Van Hoesen, G. W. (1982). Prosopagnosia Anatomic basis and behavioral mechanisms. *Neurology*, 32(4), 331-331.
- Damasio A, Tranel D, Damasio H (1990) Face agnosia and the neural substrates of memory. *Annual Review of Neuroscience* 13:89-109.
- Dean P (1976) Effects of inferotemporal lesions on the behavior of monkeys. *Psychological Bulletin* 83:41-71.
- Dutton, G. N. (2003). Cognitive vision, its disorders and differential diagnosis in adults and children: knowing where and what things are. *Eye*, 17(3), 289-304.
- Farah, M. J., Brunn, J. L., Wong, A. B., Wallace, M. A., & Carpenter, P. A. (1990). Frames of reference for allocating attention to space: Evidence from the neglect syndrome. *Neuropsychologia*, 28(4), 335-347.
- Farah, M. J. (1994). Perception and awareness after brain damage. *Current opinion in neurobiology*, 4(2), 252-255.
- Gauthier, I., Skudlarski, P., Gore, J. C., & Anderson, A. W. (2000). Expertise for cars and birds recruits brain areas involved in face recognition. *Nature neuroscience*, 3(2), 191-197.
- Gross CG (1994) How inferior temporal cortex became a visual area. *Cerebral cortex* 5:455-469.
- Goodale M, Milner A (1992) Separate visual pathways for perception and action. *Trends in Neurosciences* 15:20-25.
- Hahn et al (2011). A high-light sensitivity optical neural silencer: development and application to optogenetic control of non-human primate cortex. *Frontiers in Systems Neuroscience* 5:18.
- Holmes G (1918) Disturbances of vision by cerebral lesions. *British Journal of Ophthalmology* 2:353-384.
- Holmes, E. J., & Gross, C. G. (1984). Effects of inferior temporal lesions on discrimination of stimuli differing in orientation. *The Journal of Neuroscience*, 4(12), 3063-3068.
- Humphreys G, Riddoch M (1993) Object agnosias. *Bailliere's Clinical Neurology* 2:339-359.
- Klüver H, Bucy PC (1939) Preliminary analysis of the functions of the temporal lobes in monkeys. *Archives of Neurology and Psychiatry* 42:979-1000.
- Landis, T., Regard, M., Bliedle, A., & Kleihues, P. (1988). PROSOPAGNOSIA AND AGNOSIA FOR NONCANONICAL VIEWS AN AUTOPSED CASE. *Brain*, 111(6), 1287-1297.
- Lois, C., Hong, E. J., Pease, S., Brown, E. J., & Baltimore, D. (2002). Germline transmission and tissue-specific expression of transgenes delivered by lentiviral vectors. *Science*, 295(5556), 868-872.
- Magnié, M. N., Ferreira, C. T., Giusiano, B., & Poncet, M. (1998). Category specificity in object agnosia: Preservation of sensorimotor experiences related to objects. *Neuropsychologia*, 37(1), 67-74.
- Mishkin M, Pribram KH (1954) Visual discrimination performance following partial ablations of the temporal lobe. I. Ventral vs. lateral. *J Comp Physiol Psychol* 47:14-20.
- Riddoch G (1917) Dissociation of visual perceptions due to occipital injury with especial reference to appreciation of movement. *Brain* 40:15-57.
- Saygin, A. P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain*, 130(9), 2452-2461.
- Slimko, E. M., McKinney, S., Anderson, D. J., Davidson, N., & Lester, H. A. (2002). Selective electrical silencing of mammalian neurons in vitro by the use of invertebrate ligand-gated channels. *The Journal of neuroscience*, 22(17), 7373-7379.
- Sperry R (1982) Some effects of disconnecting the cerebral hemispheres. *Science* 217:1223-1226.
- Stoerig, P., & Cowey, A. (1997). Blindsight in man and monkey. *Brain*, 120(3), 535-559.
- Warrington, E. K., & Shallice, T. (1984). Category specific semantic impairments. *Brain*, 107(3), 829-853.
- Warrington, E. K. (1985). Agnosia: the impairment of object recognition. *Handbook of clinical neurology*, 45, 333-349.
- Weiskrantz L (1996) Blindsight revisited. *Curr Opin Neurobiol* 6:215-220.
- Zeki, S. (1990). A century of cerebral achromatopsia. *Brain*, 113(6), 1721-1777.
- Zeki, S. (1991). Cerebral akinetopsia (visual motion blindness) a review. *Brain*, 114(2), 811-824.

Bumping into things: the Pulfrich phenomenon

- Delayed retinal processing or delayed conduction from one eye
- Temporal mismatch of incoming visual information
- Inaccuracy in perception of moving targets in 3D space

Some manifestations:

Swerving inappropriately to avoid oncoming traffic or parked traffic

A need to swerve or duck when going through doorways

In crowds swerving and bumping into people

Difficulty with fine tasks

Causes:

Pupil dilatation

Demyelination

Ischemic optic neuropathy