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
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Office Hours: After Class. Mon 05:30-06:30 or by appointment
Class 1. Introduction to pattern recognition [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 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 (Inouye, Holmes, Riddoch)

Basic path of visual signals from the eyes to primary visual cortex
V1 lesions lead to topographically specific scotomatas

- 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

Holmes. British Journal of Ophthalmology, 1918
Riddoch, Brain 1917
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)

As cited in Gross 1994. Cerebral Cortex 5: 455-469
Visual system circuitry (macaque monkeys)

Felleman and Van Essen. Cerebral Cortex 1991
Initial examinations of the temporal cortex
The Kluver-Bucy syndrome

Earliest reports: Brown and Schafer 1888


- 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

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

Britten et al. *Experimental Brain Research* 1992

Form-from-motion

Form-from-luminance

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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.

Dean 1976; Holmes and Gross 1984; Mishkin and Pribram 1954
“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

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

Areas typically affected in object agnosias
Apperceptive visual agnosia

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

Copying shapes

Matching shapes

Warrington 1985
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

Warrington 1985
Example: category-specificity in object agnosia

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

<table>
<thead>
<tr>
<th>Categories</th>
<th>Number of items</th>
<th>Object recognition</th>
<th>Manipulation gestures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body parts</td>
<td>12</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Common objects</td>
<td>25</td>
<td>60</td>
<td>96</td>
</tr>
<tr>
<td>Fruits and vegetables</td>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Musical instruments</td>
<td>5</td>
<td>0</td>
<td>40</td>
</tr>
</tbody>
</table>

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

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

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

• 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)
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

Behrmann and Avidan, Trends in Cognitive Science 2005
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

Hahn et al Frontiers in Systems Neuroscience 2011
High-resolution lesions in monkeys impair object recognition

A

Neural testing

B

Behavioral testing

C

Male examples

Female examples

more male

more female

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

A

**face detector sites**

<table>
<thead>
<tr>
<th>visual field</th>
<th>contra</th>
<th>ipsi</th>
</tr>
</thead>
<tbody>
<tr>
<td>image</td>
<td>85</td>
<td>87</td>
</tr>
<tr>
<td>image+laser</td>
<td>88</td>
<td>85</td>
</tr>
</tbody>
</table>

B

**behavioral effect (Δ%correct)**

<table>
<thead>
<tr>
<th>experimental condition</th>
<th>Contra</th>
<th>Ipsi</th>
<th>C</th>
<th>I</th>
<th>C</th>
<th>I</th>
</tr>
</thead>
<tbody>
<tr>
<td>face detector sites</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>other IT sites</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre-virus</td>
<td></td>
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</tr>
</tbody>
</table>

C

$r = -0.46$, $p = 0.003$

D

$r = -0.57$, $p = 0.02$

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

\[ r = -0.61, p = 0.01 \]

\[ r = 0.58, p = 0.02 \]

\[ r = 0.23, p < 0.01 \]
Behavioral effect of drug microinjection in IT cortex

[A graph showing the behavioral accuracy of face detector sites over time after muscimol microinjection.]

[B A bar graph contrasting the behavioral effect (Δ% correct) across experimental conditions: face detector sites, other IT sites, and saline.]

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