Visual Object Recognition

Neurobiology 230 – Harvard / GSAS 78454

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Dates: Mondays
Time: 3:30 – 5:30 PM
Location: Biolabs 1075
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

Holmes. British Journal of Ophthalmology, 1918
Riddoch, Brain 1917
Hemianopia and hemianopic blindsight

• Initial retinotopic mapping in primary visual cortex was derived from brain injuries sustained by First World War soldiers (Holmes, Riddoch)

• “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)
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)

Dean 1976
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-luminance

Form-from-motion
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
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

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

Drawing from memory
- Acuity, color recognition and motion perception are preserved
- Bilateral lesion of the anterior inferior temporal lobe

Warrington 1985
Example: category-specificity in object agnosia

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

![Examples of objects](image)

**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>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of items</td>
<td>Object recognition</td>
</tr>
<tr>
<td>Group I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Animals</td>
<td>46</td>
<td>0</td>
</tr>
<tr>
<td>Fruits and vegetables</td>
<td>24</td>
<td>0</td>
</tr>
<tr>
<td>Musical instruments</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Group II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furniture</td>
<td>14</td>
<td>64.3</td>
</tr>
<tr>
<td>Vehicles</td>
<td>10</td>
<td>70</td>
</tr>
<tr>
<td>Tools</td>
<td>12</td>
<td>58.3</td>
</tr>
<tr>
<td>Body parts</td>
<td>12</td>
<td>83.3</td>
</tr>
<tr>
<td>Kitchen utensils</td>
<td>14</td>
<td>57.1</td>
</tr>
<tr>
<td>Clothes</td>
<td>19</td>
<td>73.7</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)].

Magnie et al. 1998
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)

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

<table>
<thead>
<tr>
<th>Experiment Type</th>
<th>N</th>
<th>Identity Recognition (% correct)</th>
<th>Average familiarity rating (s.d. in parentheses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retrograde-family experiment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target</td>
<td>8</td>
<td>0</td>
<td>6.0 (0.0)</td>
</tr>
<tr>
<td>Nontarget</td>
<td>42</td>
<td>—</td>
<td>6.0 (0.0)</td>
</tr>
<tr>
<td>Retrograde-famous experiment</td>
<td></td>
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<td>42</td>
<td>—</td>
<td>6.0 (0.0)</td>
</tr>
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</table>

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

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
“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
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)
- Transgenics 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
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


