General Reading List


Lectures 8 and 9 (Parallel visual pathways)


Additional Reading:


Schneider, GE (1967). Two visual systems: brain mechanisms for localization and
discrimination are dissociated by tectal and cortical lesions. Science, 163, 895-902.


**Lecture 10 (Blindsight)**


Perenin, MT and Jeannerod M (1978) Neuropsychologia, 16, 697-708. (Main Library)

**Lecture 11 (Balint's Syndrome)**

Harvey, M. (1995) 'Psychic paralysis of gaze, optic ataxia, spatial disorder of


Additional Reading:


Lectures 12 and 13 (Agnosia)


Additional Reading:

Adler, A. (1944) Archives of Neurology and Psychiatry, 51, 243-259


**Lectures 16 to 19 (Neglect)**


List of terms to help with revision (this is out of the top of my head, no guarantee provided)

**Agnosia:** patients fail to recognise objects that they consciously detect, distinction between apperceptive (D.F.) and associative

**Allaesthesia:** a contralesional stimulus is reported as ipsilesional

**Anosognosia:** denial of illness

**Balint’s syndrome** includes hemineglect, psychic paralysis of gaze, optic ataxia

**Blindsight:** ability to perform certain tasks (pointing, grasping) with having awareness of this (damage in V1)

**Constructional apraxia:** an impairment in combinatorial or organising activity in which details must be clearly perceived and in which the relationship among the component parts must be apprehended (might be due to parietal lesions)

**Directional Hypokinesia:** deficit to respond with either limb (or head or eyes) to contralesional side (Heilman)

**Dorso lateral geniculate nucleus (dLGN):** main subcortical relay station of visual info (Biggest pathway in primates and humans, part of basal ganglia), do not confuse with ventral LGN (audition)

**Efron test:** subject has to judge whether pairs of rectangles are the same or different (All shapes are matched for surface area so that differences in brightness cannot be used as a cue)

**Extinction:** failure to respond to contralesional stimulus when bilateral stimuli are present (sensory) or to move contralesional limb when bilateral movements are required (motor)

**Hemiakinesia:** reluctance to make a movement or delay in initiating a movement (contralesional side) in absence of clinical evidence for weakness

**Hemianopia:** visual field deficit
**Hemifield**: visual field in relation to retinal coordinates

**Hemineglect**: failure to respond or act in parts of space (might be due to inferior parietal lesions, also frontal and subcortical), also called visuo-spatial neglect, unilateral neglect; visual hemi-inattention, spatial agnosia

**Hemiplegia**: paralysis of one half of the body

**Hemispace**: visual area in relation to subject’s head/trunk

**IT**: inferior temporal lobe, endpoint of the ventral stream

**LIP**: ‘visuomotor’ area in the dorsal stream

**M cells** (or P alpha or type A) (with large cell bodies)

**MT** (or V5 when Zeki talks): ‘movement area in the dorsal stream’

**Optic chiasm**: subcortical relay station where visual pathways cross before they reach the dLGN

**Optic ataxia**: inability to shape hand appropriately to target demands (might be due to superior parietal lesions)

**P cells** (or P beta or type B) (medium to small cell bodies) are two types of ganglion cells in the primate retina, the dLGN and visual cortex

**Perimetry/Perimeter**: procedure/instrument to measure size of the intact visual field

**PO**: area in the dorsal stream, indicated in localising stimuli in periphery

**PPC**: posterior parietal cortex, endpoint of the dorsal stream (contains both superior and inferior parietal lobe)

**Psychic paralysis of gaze**: inability to perceive more than one object at a time

**Pulvinar**: part of basal ganglia
Superior Colliculi: subcortical relay station of visual information (biggest pathway in non-primates)

Topographical Disorientation: problems with route finding, spatial maps (might be due to medial temporal lesions)

V1, primary visual cortex or area 17 or striate cortex (all synonyms)

V3a: area in dorsal stream containing orientation selective cells

V4: ‘colour’ area in the ventral stream

**Learning Outcomes of the course**

*(Knowledge)*

1. To outline the major theories of visual processing in the brain and examine some of their strengths and weaknesses.
2. To present a new theory of parallel processing and contrast it with the existing ones.
3. To relate higher visual disorders seen in the clinic to these major theories:

Higher visual disorders fascinating:

- Blindsight
- Agnosia
- Optic ataxia
- Neglect

**Learning Outcomes of the course (Key Skills)**
Professional (Understanding and working with special populations)
Information finding (articles on reading list and novel searches)
Literacy (essay)
Analytic skills (critical evaluation of theories, methods)
Public communication (present issues in tutorial, participation in lectures)
WHAT SHOULD YOU KNOW ALREADY?

Terms of relationship: rostral - caudal, dorsal - ventral, lateral medial; sagittal, coronal, horizontal plane

Visual pathways and types of hemianopia

Thalamus (Pulvinar and LGN)

Superior colliculi

Anatomy of the cortical lobes, especially temporal and parietal

Retinotectal pathway: from the retina to the superior colliculi to extrastriate cortex
Retinogeniculate pathway: from retina to LGN to primary visual cortex (striate cortex or V1)

Visual areas after V1:
Overview Lecture 8:

1) Definition of Perception

2) Input pathways of the visual system:

3) Discuss the main visual systems hypotheses:

   a) Schneider (1969): subcortical
   b) Ungerleider and Mishkin (1982): cortical
   c) Livingstone and Hubel (1988: cortical and subcortical)
Function of vision:

- object recognition which in turn allows learning about the structure of our environment (representation of the world)
- allows us to perform skilled actions such as catching a ball, grasping an object, walking through a crowded room

I will argue that these two functions of vision depend on independent systems

Definition of Perception:

- Perception refers to a process which allows one to assign meaning and significance to external objects and events.
- Perception provides the foundation for the cognitive life of an individual, allowing to construct long-term memories and models of the environment.
Input pathways into the visual system

Two largest pathways: retinotectal and retinogeniculate. In mammals the retinogeniculate pathway is by far the most prominent.
Neurones in the dLGN project to cerebral cortex, with almost all the fibres terminating in V1 (alias striate cortex, alias primary visual cortex).
The retinotectal pathway runs from the retina via the superior colliculi via the thalamus (pulvinar) to different extrastriate areas.

Other pathways:
- ventral LGN (audition)
- Pretectum (papillary light reflexes)
- Nucleus of the optic tract (automatic reactions)
- Accessory optic tract (optic flow, posture and locomotion)
- Suprachiasmatic nucleus (day- night cycle)
The main visual systems hypotheses:

Schneider (1969): subcortical
Studied hamsters: argued that the retinal projection to the superior colliculus enables organisms to localise a stimulus in visual space, while the geniculostriate system allows them to identify that stimulus.

Lesioned the SC in the Syrian hamster and found a poor orienting response to stimuli displayed overhead. Hamsters were not impaired in a visual discrimination task where they had to push one of two doors with different patterns. They correctly identified the pattern.

Lesions of the visual cortex produced the opposite effect: grossly impaired visual discrimination but intact responses to overhead stimuli.

Schneider concluded that SC lesioned hamsters can detect visual stimuli (what) but have trouble localising stimuli in space (where).
Ungerleider and Mishkin (1982) cortical:

Claimed that the appreciation of an objects’ qualities (object identification) and of its spatial location (where is it) depends on the processing of information in the inferior temporal (IT) and posterior parietal cortex (PPC) respectively.

Same distinction as Schneider between what and where but this time the dichotomy is entirely cortical: dorsal vs ventral stream processing.

Main evidence:
Found that monkeys with IT lesions are impaired in a discrimination task but not in a landmark task (choose the food well that is closer to the object), while monkeys with PP lesions were impaired on using a spatial landmark but not on discriminating between two objects.

Problem: response modality not taken into account. Model focuses on the animals decision about the stimulus array and neglects the animals visuomotor behaviour.
Livingstone and Hubel (1988):

claim that anatomical pathways are separate all the way from the retina via LGN via V1 to IT and PPC respectively.

Quote evidence from both anatomical and physiological studies
Two types of ganglion cells in the primate retina
M cells (or P alpha or type A) (with large cell bodies)
P cells (or P beta or type B) (medium to small cell bodies).

At the level of the dLGN the P cells project to the 4 parvocellular layers
and the M cells to the two magnocellular layers.

Physiological differences:
1. Chromatic sensitivity:
90% per parvo cells are colour sensitive
10% of parvo and all magno cells are insensitive to colour (broad band)
2. Acuity, spatial frequency sensitivity
magno cells have 2-3 times larger receptive fields than parvo cells (high acuity and spatial resolution)
3. Speed and Duration
Magno: fast but transient (movement)
Parvo: slow but sustained
4. Contrast sensitivity:
parvo: peak sensitivity for high contrasts (cells respond slowly but saturate at high contrasts)
magno: peak sensitivity for low contrast stimuli (cells respond fast but level off at 10-15% contrast)
At the level of V1
magnocellular axons terminate in layer 4C alpha
parvocellular axons in 4C beta.
Both then project to others layers in V1:
4c alpha projecting to 4B
4c beta projecting to layers 2 and 3.

More recently it was found by Wong-Riley that layers 2 and 3 form a regular pattern of blobs and interblobs
Seem to be three streams of processing in V1:
from 4c alpha to 4b
from 4c beta to the blobs
4c beta to the interblobs.

Physiological differences:
- Cells in the blobs are mainly colour sensitive, prefer
low spatial frequencies, not orientation selective, nor
movement selective.
- Cells in interblobs little colour sensitivity (though more
than magno cells) but high spatial frequencies (for
acuity) along with orientation sensitivity, could be
responsible for high resolution form perception.
Although most input from parvo-cells does not seem
explicitly colour-coded.
- Cells in 4b selective for motion (from magno) but also
orientation and no colour.

At the level of V2
some projections from 4B(magno) to MT
projections run from 4B (movement) to the thick stripes,
from the blobs (colour) to the thin stripes
from the interblobs (form) to the pale stripes.

Physiological differences:
- cells in the thick stripes motion and orientation selective and coding for depth, no colour
- thin stripes colour coded but not orientation or direction selective
- pale stripes orientation selective and end-stopped (respond better to short edges or lines) seem to encode shape, no colour, direction sensitivity

**Higher visual areas**
MT receives input from the thick stripes (magno) and again cells respond to motion and stereoscopic depth
V4 receives input from the thin (colour) stripes and pale stripes (shape) also project to V4. Cells sensitive to colour.

Livingstone and Hubel argue that the parvo and magno channels remain segregated well beyond the primary visual cortex.

Propose that the ventral and dorsal stream projections identified by Ungerleider and Mishkin might represent a continuation of the magno and parvo systems: the parvo channel remains independent from the eye to IT and plays an essential role in object identification magno channel, again running independently from the eye to PP is critical for the localisation of objects.

"SEGREGATION OF FUNCTIONS BEGUN AT THE EARLIEST LEVELS IS PERPETUATED AT THE HIGHEST LEVELS SO FAR STUDIED. INDEED, THE SEGREGATION SEEMS TO BE MORE AND MORE PRONOUNCED AT EACH SUCCESSIVE LEVEL, SO THAT SUBDIVISIONS
THAT ARE INTERDIGITATED IN VISUAL AREAS 1 AND 2 BECOME SEGREGATED INTO ENTIRELY SEPARATE AREAS AT STILL HIGHER LEVELS”.

Livingstone & Hubel, 1988

What and where can be traced back to the subdivisions of the dLGN.
Evidence for the model:

Rationale: If the magno and parvo system differ in speed, colour selectivity, acuity, and contrast sensitivity, then visual tasks requiring form, colour, movement and stereo vision should differ in their speed, colour selectivity, acuity and contrast sensitivity.

Examples

1. Observers can detect brightest changes at much faster rates than colour changes (the magno system is fast but colour blind).

2. Movement detection is disturbed when subjects track isoluminant gratings (isoluminant gratings vary in wavelength but not in brightness) e.g. Patrick Cavanaugh (the magno system is colour blind).

3. Motion perception is impaired at high spatial frequencies (the magno system is better at lower spatial frequencies).

4. Depth perception with isoluminant stimuli is impaired (stereo is handled by the magno system which is colour blind).

5. Depth from motion (either motion of the viewer or motion of a point-light stimulus) is lost at isoluminance.

6. Depth from perspective is impaired at isoluminance.
Lecture 9:

1) Problems with Livingstone and Hubel’s Model

2) Extrageniculate inputs (projections from the pulvinar to where?)

3) Milner and Goodale’s theory of parallel visual processing (1992)
Summary of the last lecture

Input pathways to the visual system:
A) retina, dLGN, V1 and beyond
B) retina, SC, pulvinar and beyond.

Three theories on ‘two visual systems’

*Schneider (1969):*
geniculostriate pathway responsible for visual discrimination, SC responsible for orienting, localisation

*Ungerleider and Mishkin (1982):*
Dorsal stream (from V1 via MT/MST to PP) responsible for spatial perception (where)
Ventral stream (from V1 via V4 to IT) responsible for visual discrimination/object perception (what)

*Livingstone and Hubel (1988):*
parallel processing all the way from the retina to higher cortical areas:

M cells - 2 magno layers of dLGN - V1: to 4Calpha to 4B - V2 thick stripes and MT - PP

P cells - 4 parvo layers of dLGN - V1: to 4C beta to layers 2 and 3 (blobs and interblobs) - V2: blobs to thin stripes, interblobs to pale stripes both to V4 and then IT.
Problems with Livingstone and Hubel’s model:

1. Is all of the information in higher extrastriate cortical regions coming from V1?
No, Cells in area MT still respond when V1 is lesioned (must be another pathway)

2. Are the magno/parvo channels really that separate in V1 and V2?
No, magno input into the blobs and interblobs

3. Does MT (dorsal stream) get only magnocellular input?
NO, magnocellular lesions of LGN do not eliminate all responsiveness in MT (although they do in a fair bit of it)

4. Does V4 (ventral stream) get only parvocellular input??
V4 neurons equally affected by inactivation of magno or parvo layers in dLGN (magno should make no difference), obvious that magno goes to blobs and interblobs.

Conclusion: both dorsal and ventral stream appear to receive magno and parvo input, although most of the dorsal stream appears to be magno.
**Extrageniculate input to the cortex**

Main source is the pulvinar:

*receives* input directly from the retina and SC

*sends* projections to the dorsal and ventral streams

( medial pulvinar to dorsal stream, lateral pulvinar to ventral stream)

**Functions of these two routes:**

*SC - pulvinar - dorsal stream* implicated in motion:

- neurons in MT (dorsal stream) still active for motion when V1 lesioned
- however, become inactive when SC lesioned as well

*SC - pulvinar - ventral stream* projections not understood

- cells in V2, V3, V4 and IT no longer respond when V1 is lesioned
- seems to be no benefit from pulvinar route

Conclusions: ventral stream seems to rely on intact geniculostriate pathway, dorsal stream seems to receive other input as well.
Functional and anatomical properties of the two streams

Anatomical properties:
Distinction between dorsal and ventral stream has been repeatedly confirmed and intermediate regions have been identified.
Striking modularity of the dorsal stream (separate streams and lots of interconnections), not the case for the ventral stream (mainly one line of projections)

Functional properties:
Ungerleider and Mishkin (1982):
distinction in terms of input processing (what and where)
one system for spatial vision (dorsal stream, stimulus location)
the other for object vision (ventral stream, size, shape orientation and colour processing).

Livingstone and Hubel also focus entirely on the processing of incoming information.

<table>
<thead>
<tr>
<th>New approach by Milner and Goodale (1992)</th>
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<tbody>
<tr>
<td>Dorsal stream for visuomotor control (how)</td>
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<tr>
<td>Ventral stream for visual perception (what)</td>
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- Emphasis put on the output characteristics of the two systems (how a stimulus is processed depends on what it is used for):
- The visual inputs and transformations required for action are quite different from those leading to object identification:

**Reaching and grasping:**
- coding with respect to the viewer, i.e. motion and location of the object have to be specified in viewer-centred coordinates.
- Coordinate system used (retina, head or body) dependent on the effector system (eyes, hand or both).
- object’s structure and shape has to be coded with respect to the hand and fingers grasping it.
- fast computations since the relative position of goal and observer changes quickly and has to be updated quickly.

**Object identification:**
- constancies across different views, independent from the observer.
- Objects structure and shape coded independently from observer
- long term storage of the identities of objects.
Milner and Goodale claim:

The different transformations required for action vs perception has driven the evolution of the two streams of visual processing.

Cells in the dorsal stream perform computations necessary for visually guided actions. Networks in the ventral stream form perceptual and cognitive representations of the enduring characteristics of objects and their relations.

Division cuts right across the distinction between object and spatial vision. (what needed for action, where needed for identification)
Evidence for the theory (what vs how)

1) Taira et al. (1990) Processing of form in monkey dorsal stream

Response properties in area LIP in the monkey while performing different visuomotor tasks

1. four-fingered pull lever
2. push button
3. open pull knob (four fingers)
4. pull knob in groove--finger & thumb

some units visually responsive, some respond during hand movements

some respond to specific visual stimuli and the movements appropriate for those stimuli fairly independent of the target's location in space. Cells are sensitive to object parameters such as size and orientation but not location (very against U and M)
2) **Multiple dorsal routes from V1 to PPC (support idea of visuomotor processing in the dorsal stream):**

a. The most prominent route passes through MT which has many motion directive cells. This route could be responsible for ocular pursuit and locomotion and posture.

b. route through V3A with many orientation selective cells. Route could be responsible for visual information to guide grasping.

c. route through PO least well known, seems to specialised for dealing with stimuli located in periphery. (Could be responsible for providing visual information to guide saccades or movements towards peripheral stimuli.)

3) **Specific projections from the PPC to the frontal cortex**

a. From LIP to the frontal eye fields, responsible for eye movements)

b. From VIP and 7a to the inferior premotor cortex (implicated in control of arm movements).

4) **Descending projections from dorsal stream to SC** (eye-movements)

5) **Projections from the dorsal stream to the pontine nuclei** who in turn are closely linked to cerebellum (motor control).

No such projections exist from ventral stream.

**Conclusion:**
Seems to be that the PPC provides visual information to a number of motor-related structures both in frontal cortex, basal ganglia and brainstem.
Information processing closely linked to visuomotor behaviour (eye-movements, grasping, reaching)
Functional properties of the ventral stream
(from V1 via V2, ventral part of V3, V4 and TEO to IT)

1) more detailed coding of visual features the further along one records from V1 though to V4 and IT. In IT cells can be very specific. Seem to respond best to complex visual stimuli such as hands, faces.

2) Cells sensitive to similar stimulus features are clustered together in columns.

3) Specific colour sensitive cells in IT.

4) IT primarily dependent on the geniculostriate system (from dLGN to V1) and V4 has both magno and parvo input.

5) many cells in IT have large receptive fields. (allows generalisation across views)

6) cells respond in anaesthetised monkeys, suggests that ventral stream cells are not involved in on-line control of behaviour (here and now). (cells fire independently of own limb movement; in sharp contrast to parietal cortex cells fire specifically for monkey’s own arm movements (again action vs perception).

7) Ventral stream no projections to motor areas but instead projections to amygdala (limbic system)

Conclusion:
Properties in cells in IT consistent with object recognition and enduring characteristics (object centred).
Basis for recognition memory and long-term representation of the visual world.
Milner and Goodale (1992):

**Dorsal stream (HOW):**

- viewer centred coding
- transient (fast but not long lasting)
- cell units related to movement
- on line

**Ventral stream (WHAT):**

object-centred coding
perceptual constancies (over time and space)
colour and object perception
off line
Lecture 10 (Blindsight)

1) Evidence for Blindsight
2) Evidence against Blindsight
3) Explanation of Blindsight
4) Limits of the Explanations of Blindsight
Can Milner and Goodale’s model explain blindsight:

Prediction:
damage to V1 should result in loss of the visual perception of the world (ventral stream function) but should still allow certain actions (dorsal stream and this streams does not rely entirely on V1 input)

Patient DB studied by Weiskrantz (1974):
damage to V1 (from removing a tumour) resulted in a hemianopia and thus complete blindness in that area

- D.B. insisted on not seeing anything in his blind field but was able to make reasonably good eye movements towards lights flashed into that field.
- Performed even better on a pointing task where he was asked to point his finger to the light.

Weiskrantz named this phenomenon blindsight (accurate performance despite lack of awareness)

Soon confirmed by Jeannerod and Perenin (1975 for six more patients) and others
Major critique of blindsight concept by Campion and Latto (1983)

Patients not completely blind. The intact parts of their visual fields can aide performance: Light from the stimulus could have scattered into the good region and the patient might guess the appropriate location from this.

Inadequate fixation would allow the stimulus to fall into the good field and thus be located.

There could be nonvisual cues such as sound or heat or experimenter effects. The subjects is desperate for a cue and the examiner might unconsciously cue.

Areas of preserved vision in the hemianopic field could be responsible for the accurate response (cortex actually intact)
Replies to the critique:

Weiskrantz demonstrated that D.B. is unable to point accurately to stimuli that fall on the blind spot.

- Should be able to perform just as well at blind spot since the light would scatter from there as well, inaccurate fixation would enable pointing, the other cues should help.

Jeannerod and Perenin (1978) tested patients with severed optic chiasm as controls for cortically blind patients:
They should have no residual vision (Blindsight) in the hemianopic field (bitemporal) since the actual optic nerve fibres are interrupted.

Showed no evidence for blindsight although again they should have been able to use scattered light information.
What mediates blindsight

Milner and Goodale (1995):
Damage to V1 and consequently impaired input to the ventral stream is responsible for the lack of report. The patients do not see anything (perception).

Visual input still available through tectopulvinar route: the dorsal stream networks continue to participate in the control of spatially organised eye and arm movements (this processing does not lead to conscious perception, action).

Blindsight collection of visuomotor responses elicited by the SC’s and the dorsal stream.

Certain input\output systems can be more effectively used than others. (Looking vs Pointing)

More evidence for action related processing in Blindsight:
Perenin and Rossetti (1993):
Patients reach out and grasp objects reasonably accurately in the blind field. Can obviously use orientation and size information to guide a motor action.
Anatomical basis for blindsight (Milner and Goodale, 1995):

Major input route via the SC route. (SC alone unlikely since there is no indication of orientation coding in the SC).

Tectopulvinar inputs reach the cortex as early as V2 but cells in V2 and V3 lose their visual responsiveness when V1 is lesioned.

Area V3A responds when V1 is lesioned.

Cells in V3A code for orientation just as well as cells in V1. Likely that this area responsible for the various visuomotor behaviours shown.
Problems with Milner and Goodale’s explanation:

1) **Blindsight can be demonstrated in patients with only one hemisphere:**
Not only is there no striate cortex but no extrastriate cortex and thus no dLGN and pulvinar (projections degenerate when area of cortical projections are lesioned)

*How does blindsight work in these patients?*
Either a puzzle how patients with hemispherectomy show blindsight or certain degree of cortical reorganisation (likely since operation in childhood).
Maybe SC’s of lesioned hemisphere linked to cortex in the intact hemisphere through intercollicular commissure and thalamus.

2) **Cowey and Stoerig (1987, 1992) show colour discrimination in the blind field.**
Obviously colour processing after V1 lesions.

Problem for Goodale and Milner interpretation of blindsight as visuomotor responses (i.e. SC and dorsal stream processing):
No evidence for colour processing in SC and subsequent route but

*a very small number of cells (only a few thousand) project directly from the parvo cellular layers of the dLGN to V2 and V4. They survive lesions of V1.*
Very likely that these cells provide the necessary information to subserve colour discrimination in blindsight.
Not clear what function this pathway has in the normal brain.
Conclusion:

Blindsight paradoxical if vision regarded as a unitary process. If one accepts separate mechanisms for visual experience and others for action the paradox disappears. It would mean that in such patients retinal stimulation does not lead to the experience of sight but instead enables access to visuomotor control mechanisms.
Lecture 11

1) Balint’s patient (1909)
   - hemispatial neglect
   - psychic paralysis of gaze
   - optic ataxia

2) Balint vs Holmes
   - what vs how
   - what vs where

3) Specific impairments of patients with optic ataxia
Balint’s Patient (studied by Balint over 3 years):

- bilateral brain infarcts
- normal visual fields
- *hemispatial neglect* obvious when tested for acuity and reading
  - *psychic paralysis of gaze* (only sees one object at a time): also impairs reading

- *Optic Ataxia*:

  - not primary motor disturbance nor disturbance of proprioception
    - only movements that required visual control were faulty
    - only right hand affected

post mortem showed that patient had bilateral lesions in the posterior parts of both parietal lobes
Holmes (1918, 1919):

- published two papers on patients whose symptoms very similar to those of Balint’s patient.

- stressed the spatial rather than the visuomotor problem

- both studies frequently cited unlike Balint’s
- fit with Ungerleider and Mishkin (1982)
Problems with Ungerleider and Mishkin’s model in relation to optic ataxia:

Assumption one

_a deficit in space perception should be either in contralateral space or in case of a bilateral lesion in both hemispaces (but independent of hand)._  

Balint’s patient impaired in either hemispace but only for his right hand (Balint’s patient had a bilateral lesion).

but

optic ataxia also after unilateral lesion: only contralesional field affected or sometimes only quadrant if lesion is small (agrees with U and M)

but

not only space is important but effector system: even if both hands show an impairment, eye movements towards the target can be accurate (Ratcliff and Davies-Jones, 1972)

Conclusion: misreaching cannot be a loss of the sensory representation of space but must be a deficit linked to specific effector systems. Both hands or both hemifields can be impaired in a number of combinations although the lesion tends to be in the same region (generally the superior part of the parietal lobe (Balint slide). Must be that the visuomotor network underlying reaching can be impaired at different stages from in the transformation from visual to motor...
coordinates.
Assumption two:

*Optic ataxics should fail to localise a stimulus but should be able to use size and orientation cues to guide their processing of the stimulus*

Jeannerod (1988):

in prehension transport and grasp components are temporally coupled: the hand opens in anticipation over time, opens wide and then homes in.

Perenin and Vighetto (1988):

10 optic ataxia patients impaired in both the transport and grasp component of a movement: made errors in hand rotation as they reached toward an oriented slot.

but

could localise dots (align them with a number on the screen). Some degree of spatial localisation still there.

Jakobson et al (1991) showed that their optic ataxia patient only showed a very weak relationship between object size and grip scaling.

Conclusion:

Obvious that lesions to the parietal cortex produce more than just spatial location deficits. They cause deficits in the sensitivity of a grasp regarding an objects size, shape and orientation. The lesions also damage specific effector systems (hand, space, difference between hand and eyes etc) rather than spatial location *per se.*
Lecture 12 (Agnosia I)

1) Definition of agnosia

2) Famous cases

3) Patient DF

4) Residual processing capacities of DF

5) Pathways mediating residual processing
**Definition of Agnosia**

-A profound deficit to visually recognise objects  
- patients fail to recognise objects which they consciously detect.  
- problems in the visual domain only  
- very rare; requires intact V1  
- usually bilateral damage in the occipito-temporal area, i.e. the ventral stream which is responsible for object processing is cut off from the input of V1.

**Historical account:**

Lissauer (1890) [two types of agnosia]

- Apperceptive agnosia makes patients unable to perceive a coherent percept of the structure of an object.  

\[\textit{a disruption at very early stages of perceptual processing}\]

- A patient suffering from associative agnosia can perceive an object okay but is still unable to recognise it.

\[\textit{a disruption at a higher cognitive level where percepts would normally be associated with stored semantic information}.\]
Visual form agnosia

- severest form of apperceptive agnosia
- only a few patients ever reported
  all have a particular pathological history
suffered from an anoxic episode (usually suffered from carbon monoxide poisoning)

CASES:
Adler (1944) : Patient H.C.)
Benson and Greenberg (1969): Mr S.
Campion and Latto (1985): Patient RC
Milner and Goodale (1992): Patient DF

Tissue most vulnerable to anoxia are the watershed areas
(Areas lying in the border regions between the territories of the different arterial systems)

MRI’s available of H.C. and D.F. indicate the existence of dense lesions in the occipital cortex bilaterally
Mr S (extensively tested by Efron)

- severe object and pattern recognition deficits,
- unable to recognise faces, identify line drawings or even simple shapes
  better with real objects (more cues such as colour, reflectance, texture)
  normal performance when haptic information available

Efron argued that Mr S’s disorder was one of visual contour, i.e. shape discrimination.

Devised the Efron test:

the subject has to judge whether pairs of rectangles are the same or different (All shapes are matched for surface area so that differences in brightness cannot be used as a cue)

*Mr S was unable to distinguish between any of the rectangles apart from the 9:1 ratio (even then only 90% correct).*

Now a standard test for visual form agnosia. Does not require shape perception (square vs oblong) but can be done by comparing orientation or size information. Implies that visual form agnosics are impaired on these simple transformations as well.
Patient DF (Milner et al (1991))

- suffered from carbon monoxide poisoning while having a shower.

- severe recognition disorder cannot be explained by low-level visual deficits.

severely impaired on the Efron Task, cannot recognise line drawings and cannot copy them

- cannot recognise letters of digits but can print them from dictation, can also draw from memory.

- better at recognising real objects but again uses colour, texture and brightness cues

- unable to recognise faces but recognises people by their voice, brightly coloured hair etc.

- again normal recognition in the tactile and auditory domain.
Milner et al. (1991) Slot experiment

DF confronted with a disc in which a slot was cut which could be oriented at different angles

Asked to do two separate tasks: 
*Demonstrate the orientation of the slot* (unable to do so at all)

*Asked to insert her hand into the slot* (she was perfect)

In the *matching* task the subjects were reporting their perception of the slot.

To *post* the hand is a natural *goal-directed movement* which requires the rotation of the hand in order to be successful. It can be performed independently of a conscious perception of the orientation of the hand or the slot.

*Special visuomotor system responsible that is independent of object recognition.*

Same dissociation observed with regard to size and shape: can grasp Efron shapes but cannot indicate their width.

*Findings strongly suggest that DF has a fully functioning visuomotor system that uses orientation and size information although her perceptual system does not.*
What mediates these residual functions?

Very likely that size and orientation calibration comes from the dorsal stream since her ventral stream (or at least the access to her ventral stream) is damaged. The input to dorsal stream could come either from V1 or the tectopulvinar route.

Know that \(V1\) is spared in DF, simple sensory perception intact. Likely that human equivalents of \(V2\), \(V3\) and \(V4\) are disrupted.

Likely that neither the magno (4B) nor the \textit{Parvo Interblob channel} (Layers 2 and 3) can send information to the ventral stream via \textit{pale} stripes. If this region is no longer able to convey information to the ventral stream, the ventral stream would be deprived of both magno and parvo derived information on form.

Seems that DF’s vision can no longer rely on the Interblob channel. The magnochannel through V2 and V3 seems also impaired since she has trouble with low spatial frequencies.

However, even a complete lesion in V2 and V3 and V4 could still leave visuomotor abilities intact since the \textit{dorsal stream} could still receive input from \textit{V1 direct} (to \(MT\), \(V3A\) and \(PO\)) and there is also the tectothalamic pathway (\(Sc\), pulvinar). These are routes on how the dorsal stream could receive input to allow the visuomotor behaviour.
How come she can still use colour and texture?
If access to ventral stream is blocked she should be unable to do this?

Unlikely that this is mediated by the dorsal stream, no evidence for colour coding there just yet.
Seems to be mediated by the ventral stream. Seems likely that the **blob channel has intact connections through V2 to V4 and IT**. Seems to be colour only, not size or shape, but possibly surface characteristics of objects (texture).
Lecture 13 (Agnosia II)

1) Limitations of visuomotor processing in DF

2) Can the visuomotor system aid object recognition?

3) Can Ungerleider and Mishkin’s Model explain DF?
Limitations of visuomotor processing in DF (1)

accurate processing of orientation and size

Processing of shape:
  impaired when asked to post a T-shape into a slot
  on half the trials she was correct on the other she was incorrect.
  Suggests that she can take account of only one of the two segments at once.

POSSIBLE PROBLEMS WITH SHAPE PROCESSING?

Experiment 1:
  presented with a series of everyday objects and asked her to pick them up and demonstrate their use.

Is the object grasped appropriately as well as accurately?

  approach was fine, anticipatory opening of the hand but

  when the object was rotated inappropriate grasp points and far more tactile exploration than the controls (needs non-visual information)

Conclusion: her grasp is not guided by functional knowledge of the object. She seems to use visual information of axis orientation and size to guide her
grasp but then uses tactile feedback to demonstrate the objects function.

Obvious that functional object knowledge is not accessible via the dorsal stream but suggests that it comes from the ventral stream and from there projected to the dorsal stream.

**Second experiment:**

*Can she combine size and orientation information to guide her actions?*

Efron shapes presented at different orientations and asked to pick them up accurately (requires orientation of the hand adjustment of the grip size at the same time).

DF adjusts both her grip aperture and the orientation of her hand well in advance of target contact.

*No problem in combining orientation and size information*

**Experiment 3:**

*Can she process shape to guide her actions?*

Asked to reach out for a cross that was presented at different orientations. (no principal axis she can use to guide her orientation)
She does not rotate her hand in anticipation to the rotation of the cross
Does not spread her fingers to engage with the shape of the cross.
Conclusion: Her visuomotor abilities seem to be limited in so far as she is not able to integrate more than one orientation in exercising her motor control. It seems that her general good grasping performance is due to her identifying the principal axis and combine this with size processing.

Seems unlikely that DF’s visuomotor system can deal accurately with different visual contour orientations to combine them into a shape. DF as such does not give evidence that the dorsal stream can handle form.
Can the abilities of her visuomotor system aid her object recognition?

Yes, to some extent:

Asked to stamp shapes (orientation accurate)
When asked not to stamp directly onto the stimulus but instead on a separate sheet (perceptual task), surprisingly she was good.

But:

moved towards the stimulus with her stamp and then changed in mid-flight to stamp the blank paper. (only apparent through careful video analysis)

Obviously used the shaping of her movement and transferred that to the blank paper giving her the right orientation.

She monitors her visuomotor performance to enhance her perceptual judgements.
How does Ungerleider and Mishkin’s model explain DF’s residual processing abilities?
She should be able to localise objects in space but not much else.
Should not be able manipulate objects without knowledge of the objects identity.

BUT

She not only knows where an object is but also how to approach it.

Obvious that this is not done by the ventral stream since even though she can reach for objects there is no object recognition and no functional knowledge of the object.

Milner and Goodale argue that the distinction is better explained in terms of **What** for ventral and **How** for dorsal.

**How** requires a **Where** since otherwise one could not get there in the first place but it also involves an approach that is guided by the intrinsic properties of the object and an on-line handling of that object.

Comparison of DF to optic ataxia patients? Very obvious dissociation.
Optic ataxics cannot orient their hand appropriate,
DF no problem
They do not show anticipatory opening of the hand to grasp an object, DF perfect
They can recognise object, DF cannot
They can orient their hand to match, DF cannot
Milner and Goodale’s assumptions for the two streams: What vs How

Ventral stream characteristics (perception):
- object-centred coding (independent of observer)
- perceptual constancies (how does this look like over different views, spatial positions)
- colour, object perception
- "off-line functions" (access to memory of objects and object relations)

Dorsal stream characteristics (action)
- viewer-centred coding (where are things in relation to the person)
- transient (quickly picks up even subtle changes)
- many units related to movement (action)
- "on-line functions" (where is now, and how does it change)
Lecture 16

Spatial Attention and Visual Neglect

1. Description of the neglect syndrome

2. Historical Overview:
   a) Issue of laterality
   b) Locus of lesions in neglect
   c) Task differences

3. Standard neglect tests today

4. Recovery from neglect
Components of the Neglect Syndrome:

**Hemispatial Neglect:** failure to *act* in the contralesional hemispace.

**Hemi-inattention:** failure to *respond to/explore* stimuli presented in contralesional hemispace.

**Extinction:** failure to respond to contralesional stimulus when bilateral stimuli are present (sensory) or to move contralesional limb when bilateral movements are required (motor).

**Allaesthesia:** a contralesional stimulus is reported as ipsilesional.

**Hemiakinesia:** reluctance to make a movement or delay in initiating a movement (contralesional side) in absence of clinical evidence for weakness.

**Directional Hypokinesia:** deficit to respond with either limb (or head or eyes) to contralesional side (Heilman).

**Anosognosia:** denial of illness.

**Synonyms for neglect:**
visuo-spatial neglect; hemispatial neglect
unilateral neglect; Visual hemi-inattention
spatial agnosia
Issue of Laterality:

Brain (1941) first to give a detailed description of neglect:
- three patients with large right parietal lesions in which he noticed a disorder of route finding: 'agnosia for the left half of space'
- restricted to patients with minor (i.e.) right hemisphere lesions
- different from hemianopia, hemiplegia
- possible that absence in left hemisphere lesioned patients is due to masking (i.e. aphasia)

Since then debate in the literature: Does neglect occur more frequently with right-than left hemisphere lesions, and if so, what significance does that have?

Oliver Zangwill's group: left sided neglect present in 14 out of 21 right hemisphere lesioned patients but only in one out of eight left hemisphere lesions but
Battersby and colleagues (1956): tested 85 patients and found no difference as to the side of lesion: found the syndrome in 29 per cent of patients with non-dominant (right) lesions and 9 per cent of patients with dominant (left) lesions but 29 per cent of left hemisphere patients had to be excluded because of aphasia so they judged 62 per cent of the left hemisphere cases and 59 per cent of the right hemisphere cases to be without neglect

Gainotti (1968) designed a battery of tests simple enough to include aphasics: found that neglect is not only more frequent but also more severe after right hemisphere damage
Localisation of Lesions in Neglect:

Zangwill's group: right and left hemisphere lesioned group differed in incidence of visual field deficit and papilloedema suggesting that the right hemisphere lesioned patients had larger lesions

general problem:
no CT, MRI scans, right hemisphere lesions tend to be silent, only noticed when fairly large

Locus of lesion:
-not only parietal
Vallar and Perani (1986): CT study on 110 right hemisphere lesioned patients
Cortical neglect more frequent with retrorolandic than frontal lesions
- inferior parietal lobule most frequently involved
Subcortical neglect more frequent when grey nuclei (thalamus and basal ganglia) rather than white matter damaged
Task Differences:

Problem: different investigators use different tasks to assess neglect (i.e. drawing, copying, reading)

Gainotti (1972): Right hemisphere lesioned patients more impaired than left h. patients when task required attention to be focused on small portions of space but no difference between groups when task required exploration of large displays

Consensus of laterality debate: some people still argue but majority thinks that neglect is more frequent and more severe after right h. damage especially after more than three days post onset
Standard Neglect Tests Today:

In Britain: Behavioural Inattention Test (BIT); Wilson, Cockburn and Halligan (1987)

- 80 patients with right or left hemisphere damage compared to 50 controls on:

Line Bisection (Schenkenberg, 1980)
Line Crossing (Albert, 1973)
Letter cancellation
Star cancellation
Figure and shape copying
Representational drawing

gives a cut-off score for each test and overall
Recovery from Neglect:

**Clinical picture:**

in severe cases very strong ipsilesional eye and head deviation

changes to neglect symptoms (i.e. failure to dress, shave etc)

changes to inattention (failure to respond)

Inattention can fade into extinction

all signs may disappear unless specifically tested for, for example:

**Goodale et al (1990):**

tested 10 right h. damaged (recovered from neglect) on a visuomotor bisection task: patients showed rightward errors
Lecture 17

Spatial Attention and Visual Neglect

Theories of Neglect (from 1940 to recent thinking):

1. Sensory hypothesis (Battersby et al.)
2. 
3. Representational approach (Bisiach et al.)
4. 
3. Directional Hypokinesia (Heilman et al.)
4. 
5. Attentional approach (Posner et al.)
6. 
5. Spatial misperception (Milner et al.)
Brain (1941) and Zangwill group: neglect independent of sensory and general intellectual disturbances

but

1. Sensory hypothesis: Bay (1950) and Battersby (1956): a combination of sensory defects and intellectual deterioration can account for the disorder

but

2. Representational Approach: Bisiach (1978): demonstrated neglect in tasks without sensory inflow (i.e. imagination)
- RHD patients asked to face Milan cathedral and describe scene, asked to face away from cathedral and describe scene
patients omitted items on the left depending on the perspective taken
-failure to exploit otherwise intact representation
3. Directional Hypokinesia (Heilman, 1979):
  a unilateral brain lesion hypoarouses the hemisphere and causes a selective loss of orienting responses in the hemispace contralateral to the lesion
  - the left hemisphere controls orienting in right space only
  - the right hemisphere controls orienting in left and right space
therefore: left hemisphere lesion can be compensated, right hemisphere lesions cannot

Experimental confirmation: RH patients initiate responses into left space more slowly than LH patients responses into right space (same for magnitude of errors)
Posner's paradigm:
   +  *  +
cues indicate to be attended locations:
80 per cent valid
20 per cent invalid

Normal Subjects:
benefit from valid trials because already engaged in
target
but cost of invalid trials as attention needs to be
disengaged and moved and reengaged on new
target

Patients with parietal lesions:
benefit from valid cues like controls (in both left
and right space)
also same cost when invalid cue in left space and
they have to reengage to right space
but
high cost when invalid cue in right space and they
have to reengage to left (sometimes failed to detect
stimulus completely)
-problem to reallocate attention once it has been
directed to the right
5. Spatial misperception (Milner et al., 1992, 1993)

Is it possible that neglect patients who fail to attend to left hemispace misperceive it as relatively shrunk compared to right hemispace?
- Impossible to tell with bisection task
- but Landmark task can distinguish between perceptual neglect and directional hypokinesia

**Landmark Task**: centrally prebisected line, patient is asked to indicate to which end the central mark is closer to
- 7/8 neglect patients point to the left (perceptual neglect)
- 1/8 points to the right (directional hypokinesia)
Other evidence for these two types of neglect:

**Bisiach *et al.*, 1990: pulley device on line bisection**
Congruent condition: a rightward movement moves pointer to the right (and VV)
Incongruent condition: a rightward movement moves pointer to the left (and VV)
Results (incongruent condition): some patients move pointer leftwards to set transection to the right (perceptual neglect), others move pointer rightwards and transect to the left (directional hypokinesia)

**Tegner and Levander (1991): line cancellation by looking in mirror**
all patients neglect left space in normal view
Mirror view:
4/18 cancel lines in right hemispace only (directional hypokinesia)
10/18 patients cancel lines in left hemispace only (perceptual neglect)
4/18 cancel only central lines (combination of the two deficits?)
Dissociation between two types with regard to line length (Harvey et al., 1994)

Patients with perceptual neglect make smaller and smaller errors the shorter the line becomes (line bisection), patients with DH make large errors throughout
Lecture 18

Neglect and the two visual systems

1. Revision of Theories
2. Object-centered neglect (Driver & Halligan)
3. Blindsight? (Marshall & Halligan but see also Bisiach)
4. Does neglect result from damage to the dorsal stream?
   (Evidence for and against)
2. **Object-centred neglect** *(Driver and Halligan, 1991)*

- single case study
- patient neglects the left side of objects even when presented in right hemispace
- even neglects left side of stimuli when objects rotated and left part of stimulus is on right side of the display
(interpreted as impaired spatial representation of objects)
but maybe viewer centred rather than object centred representational deficit (Rizzolatti)

- single case study
- patient presented with drawing of a burning and non-burning house, she claimed houses are the same
- asked where she would prefer to live: pointed to the non-burning house

replicated by Bisiach and Rusconi (1990)

- four patients, various stimuli
results inconsistent
- some patients prefer broken glass
- some unflowered pot
- some the burning house
4. **Is hemispatial neglect a result of dorsal stream damage:**

**Ungerleider and Mishkin (1982):**

Claimed that the appreciation of an objects’ qualities (object identification) and of its spatial location (where is it) depends on the processing of information in the inferior temporal (IT) and posterior parietal cortex (PPC) respectively. Same distinction as Schneider between what and where but this time the dichotomy is entirely cortical: dorsal vs ventral stream processing.

**Main evidence:**
Found that monkeys with IT lesions are impaired in a discrimination task but not in a landmark task (choose the food well that is closer to the object), while monkeys with PP lesions were impaired on using a spatial landmark but not on discriminating between two objects.

Problem: response modality not taken into account. Model focuses on the animals decision about the stimulus array and neglects the animals visuomotor behaviour.
evidence for it

monkey evidence showed that the PPC is implicated in spatial attention (Mohler and Wurtz)

thus

reasonable assumption that neglect in humans following damage to the posterior brain areas must be result of damage to the end-points of the dorsal stream

and

a spatial imbalance of attention is present in most cases of neglect.
in most patients the lesions include parts of the parietal lobe.

Nonetheless there is serious doubt that neglect results form dorsal stream lesions.
Evidence against it:

1. Proved very difficult to mimic neglect properly in monkeys. Monkeys do not show the attentional failure, the perceptual failure, object centered neglect (Milner, 1987).

2. What is the human equivalent of PPC?

   - monkey evidence associates visual guidance of reaching, grasping and eye-movements with the dorsal stream. They are impaired by PPC lesions.
   - in humans equivalent deficits are associated with lesions of the superior parietal areas: optic ataxia, psychic paralysis of gaze
hint that in humans the *superior parietal areas* are the equivalent of monkeys dorsal stream mechanisms

Human neglect most commonly associated with the inferior parietal lobe and parietotemporal lobe.

3. Object centered neglect:
Argues against neglect as a deficit within the coordinates of egocentric space (space with relation to the person acting) since object based neglect cannot be independent of object identity processing.

Possible that inferior parietal and temporo-parietal regions have evolved in the human to deal with this form of abstract spatial processing and that they rely heavily on the input from the ventral stream

Possibility that impairment of abstract spatial processing on one side leads to an incorrect computation of stimulus size
Lecture 19: Are all neglect symptoms independent of dorsal stream processing:

Extinction

Directional Hypokinesia

Attention and Awareness
**Extinction within neglect syndrome problematic:**

1. Double dissociation between neglect and extinction (it can occur without neglect (De Renzi, 1984, Barbieri and De Renzi, 89) and neglect can occur without extinction (cannot be a mild form).

2. Extinction found to an equal extent after right and left hemisphere lesions while neglect is generally more frequent after right h. lesions.

3. Evidence for an anatomical distinction:
   superior parietal lobe lesions produce optic ataxia and extinction (Perenin and Vighetto, 1988) but not neglect.

*Conclude that visual extinction may follow damage to the dorsal stream in both humans and monkeys but that neglect does not.*

Extinction is primarily a failure to orient. This function is easily carried out by the dorsal stream or by the SC’s. It should be closely linked to visuomotor control mechanisms. Should be more obvious when patients are asked to point than through verbal report (Bisiach (1989).
Directional hypokinesia

Most investigators now distinguish between premotor and perceptual forms of neglect.

Most studies have found that premotor neglect is related to frontal and subcortical damage while perceptual damage results from inferior parietal damage.

Directional hypokinesia can be demonstrated in monkeys and even rats. Seems to be related to damage to motor or premotor areas in the brain.

Quite different from the neglect which is a selective failure of perceptual awareness of incoming stimuli.
Attention (Milner and Goodale’s hypotheses)

In the dorsal stream attention is closely linked to the particular effector systems. There are specific attentional mechanisms for eye-movements, arm movements etc.

In the ventral stream attention is linked to a particular stimulus (filtering of irrelevant stimuli) rather than to a selective response.

Only the attentional mechanisms associated with the ventral stream allow conscious awareness of objects and events in the world.

The dorsal stream although involved in selective attention does not give rise to visual awareness.
Evidence for these assumptions

1. **Attentional modularity in the dorsal stream:**

Goldberg and Wurtz studies:

- a typical cell in the superficial layers of SC would respond to a visual stimulus flashed into its receptive field but they also showed that the cell response was greatly enhanced when the stimulus was relevant to the animal.
- Similar attentional effects were also found in the frontal eye-fields, PPC and pulvinar but not in the primary visual cortex.

In a covert attention ask the monkey was asked to fixate and respond to the stimulus by pressing a key. It was found that **no enhancement** was found in the SC or frontal eye field neurons but in PPC and the pulvinar.

This was interpreted at the time the SC and frontal eye fields may be specifically associated with gaze shift (saccades) while the PPC mediates attention independent of gaze.

As been derived from this that PPC mediated attention generally (some sort of superordinate attentional process)

but

the monkey was pressing a key in response.

Same study can be taken as evidence for selective visual attention in relation to reaching and saccadic behaviour.
2. *Attention and the ventral stream:*

Attentional modulation in discrimination tasks (Moran and Desimone, 1985):

- cells which are selective for shape and colour show larger response magnitudes when the monkey is attending to the stimulus.
- seems to be independent of the type of response that is shown.

Attention is stimulus driven rather than response driven. It facilitates the perceptual analysis of objects in the visual array.

Conclusion: in the ventral stream an object is segregated and intensified to filter it out from irrelevant objects, in the dorsal stream an object is segregated and intensified to facilitate actions towards that object.
3. Evidence for linking ventral but not dorsal stream to awareness

*Evidence from blindsight patients:*

Blindsight patients do not have conscious percept of the stimulus but can nevertheless point.

Argued that this residual ability is performed by the dorsal stream through input from the pulvinar.