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National Program on Technology Enhanced Learning (NPTEL)

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Course Title:

Basic Cognitive Processes

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Lecture 37: Disorders for Perception & Attention

Why study disorders?

- A well functioning system does little to tell you about “how” it is functioning?
- Only when it is broken, or needs to be repaired, one comes to know the various components & their functionality.
- The idea behind studying disorders of cognitive functions is that it will help us better understand normal function and find better ways to help patients cope with impairments.

Synaesthesia

- Have you ever wondered on questions like?
 - What color is number 7?
 - What does Monday taste like? Or
 - What is it like to touch blue?

- While some of you will find these questions difficult to grasp, a very small minority of you might completely understand these questions. Those small minority are referred to as *synaesthete*, i.e. persons having synaesthesia.
- When people with synaesthesia are presented with a stimulus, they tend to consistently and automatically experience another sensory event; which could either be in the same modality (e.g. vision) or in another modality (e.g. vision & gustation/olfaction etc.).

- the commonly reported experiences include experiencing a colour on seeing or hearing a letter, number or word.
- Similar examples could be lining colours with faces, days or weeks but also things like feeling sounds, seeing musical notes, seeing tastes and tasting colours (Baron – Cohen et al., 1996).
- Synaesthetes also report that inducers which follow a reliable sequence (e.g. letters of alphabet etc.) are experienced in a specific spatial arrangement.

- Early incidence of synaesthesia was reported in a famous paper by Baron – Cohen et al. (1987), who examined a synaesthete called EP; who called herself as ‘an artist who has experienced the life – long condition of hearing words and sounds in colour’.
- Baron – Cohen tested & established the replicability of EP’s synaesthetic experiences (100% vs. 17% by non – synaesthete).
- While for many synaesthetes, the colour induced by the first letter determines the apparent colour of the word; EP’s synaesthetic experience was mix of colours induced by each letter of the word (on pseudo words)

- *Nature of Synaesthesia*

- Mattingley et al. (2001) reported accuracy levels of 80 – 100% in a group of 15 synaesthetes compared with 30 – 35 percent in 15 non – synaesthete controls in reporting synaesthetic experience.
- Moreover, the reports were more consistent over a 3 month period of test – retest interval as compared to that of non synaesthetes after a delay of just one month.
- Synaesthesia is usually a uni – directional process; a letter may give rise to the perception of red but no vice versa.

- For most synaesthetes, simply imagining the inducer can be enough to produce the synaesthetic response.
- Although, synaesthetic experiences are limited to fairly low – level percepts such as colour or spatial location rather than the appearance of a face or an object (Grossenbacher & Lovelace, 2001).
- Also, some synaesthetic experiences have been traced to developmental experiences. For e.g. Witthoft & Winawer (2013) linked consistent letter – color associations in 11 synaesthetes to a specific Fisher – Price colored letter set, which participants recall seeing in their childhood.

- Carpenter (2001) report that most synaesthetes regard their condition as a good thing, though one of them was identified as having problems.
- Steven & Blakemore (2004) reported the details of six synaesthetes who experienced seeing colors on hearing or thinking about letters or numbers, despite being blind for many years.
- Steven & Blakemore (2004) comment that this suggests that synaesthesia, 'persists for very long periods with little or no natural experience in the referred modality' (2004:855).

- *Incidence & Familiarity*

- Baron – Cohen et al. (1996) estimated the incidence of synaesthesia at about 1 in 2,000, with about 80% being female.
- Ward (2013) cites prevalence rates from 0.2 per cent of the population for taste – shape synaesthetes, 1.4 for letter/number-color, & up to 20 percent for those who image sequences into spatial arrays; suggesting that an approximate equal number of men and women have synaesthetic experiences.

Experimental Studies of Synaesthesia

- Several researchers (e.g. Mills et al., 1999; Mattingley et al., 2001) have used versions of the Stroop test (Stroop, 1935) to investigate synaesthesia.
 - Typically, a synaesthete is asked to identify the actual colour of a series of stimuli, some of which are inducers and some not.
 - When the inducer color matches the synaesthetic colour, responses are significantly faster than on neutral, non-inducer, trials.
 - When an inducer has a different colour, it interferes with performance and responses are significantly slower than neutral trials.

- These findings suggest that synaesthesia cannot be 'switched off'; and hence are termed mandatory or automatic.
- Mattingley et al., (2001) modified the Strop task to investigate whether conscious processing of an inducer is necessary for a synaesthetic response.
 - Inducers were presented very briefly followed by a visual mask. It is known that such brief masked presentations are registered in the brain at some level because they can influence responses to subsequent related material.

- However, this technique prevents conscious awareness, and neither the synaesthetes nor the control participants were able to report what they had seen.
- Under these conditions, no synaesthetic interference was found.
- Hence, it was concluded that conscious awareness is therefore necessary for synaesthetic responses to occur.

- Such a finding is also supported by studies that have used visual search paradigms.
 - When asked to search for a red F amongst blue letters; synaesthetes did not have any added advantage as a blue F does not pop out because of their experience of the letter as red.
 - Suggesting that conscious attention to the inducer is necessary before the synaesthetic experience to occur.

- *Brain Imaging Studies of Synaesthesia*

- Functional brain – imaging techniques such as fMRI, allow researchers to examine changes in the measures of brain function as volunteers perform different task. Areas that are involved predominantly during any of these tasks receive more oxygenated blood, which can be tracked & hence give a spatial map of brain function..
- EEG: records minute changes in voltage detectable from electrodes harmlessly resting on the scalp. The measures are very sensitive to ms by ms changes but give only a rough idea about the spatial locations of neural activity.

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- **Transcranial magnetic stimulation** as a method allows some inference about the functional involvement of a brain area in process.
 - An electromagnetic coil near the scalp is used to induce an electrical pulse in the underlying cortical region, which can cause excitation, or with repeated stimulation, a period of under – excitability, resembling a temporary lesion.

- Nunn et al., (2002), used fMRI to report that synaesthetes showed increased activity in V4, region of the brain linked with colour perception, when listening to inducer compared with non – inducer words.
 - This signal was not found in non – synaesthetic control participants who also had been asked to associated colours & words.
 - Consistent findings were reported by Hubbard & Ramachandran (2005).
- These findings showed that synaesthetic colour experience was represented in exactly the same system as that based on ‘real’ colour experience.
- Though some other studies have also failed to detect similar activity & there are suggestions that different forms of synaesthesia may be associated with different patterns.

- *Last words:*
- Competing theories have been proposed to explain the occurrence of synaesthesia.
 - One of them proposed maintains that all of us were synaesthetes during early stages of childhood but have lost those connections during the course of development.
 - Ramachandran & Hubbard (2002) note that brain areas involved in colour perception (V4) are immediately adjacent to the areas active during letter reading.

- Grossenbacher & Lovelace (2001) put forward a 'disinhibited – feedback' theory suggesting that connections between different sensory pathways exist in 'normal' brains but that the activity of these pathways is usually inhibited to prevent unadaptive cross – talk between sensory modalities.
 - Interesting evidence has been reported supporting the disinhibition hypothesis; as Neufeld et al., (2001) found no difference between 14 auditory – visual synaesthetes and non – synaesthete fMRI participants in terms of functional connectivity between auditory & visual areas but greater connectivity between sensory areas and the IPS in the synaesthetic group; suggesting that it may be links via this integration area that are suppressed in non – synaesthetes.

Blindsight

- Poppel et al., (1973) studied a group of ex – servicemen who had suffered visual field deficits as a result of gunshot wounds to the striate cortex, & asked the participants to make judgments about the correct location of a flash of light presented to their ‘blind side’.
- As the servicemen, could not see the flashes, the light was paired with the sound of a buzzer, and on hearing the buzzer the servicemen were asked to move their eyes in the direction of the light source.
- While the servicemen found this a difficult task, all of them were able to direct their gaze towards the light which they could not see.

- Weiskrantz et al., (1974) described a patient DB who seemed to demonstrate the same remarkable ability; i.e. he could report details of objects appearing in the blind areas of his visual field despite having no conscious experience of seeing them. Weiskrantz coined the term 'blindsight' to describe this phenomenon.
- In a series of experiments, Weiskrantz & colleagues were able to systematically investigate the perceptual abilities preserved in the blind areas of DB's visual field.
 - DB was able to detect the presence of an object, & indicate its location in space by pointing. He could discriminate between moving & stationary objects; horizontal & vertical lines & the letter X from the letter O. However, he could not distinguish between X & a triangle.

- *Blindsight*: Cowey (2004) summarized the arguments put forward by sceptics such as Campion et al., (1983).
 - **The stray light hypothesis**: Campion et al. favored the stray light explanation of blind sight, which suggested that the blindsight patients responded to light reflected from the object onto the functioning areas of the visual field.
 - they described a patient who reported that he was using such a strategy to distinguish between vertical & horizontal bars presented to the blind areas of his visual field.
 - The patient claimed that he could see a faint glow in the preserved areas of his visual field and used this cue to undertake the task.

- Champion et al., demonstrated that such a strategy could lead to the accurate localization of a light in a 'blind' area of the visual field of normal subjects whose vision had been masked.
- However, some of these explanations did not hold up, as they could not explain DB's ability to distinguish letters such as X and O or two different spatial frequency gratings with the same average brightness.
- In addition DB could locate objects even against a bright background, whereas Champion et al.'s normal subjects could only locate a light source against a low level of background illumination.

- **Spared islands of residual vision**
 - Wessinger et al., (1997) suggested that blindsight was attributable to small areas or islands in the scotoma i.e. areas of blindness in the visual field, within which vision is spared & that blindsight may be mediated by what is left of the primary visual pathway rather than other secondary pathways.
 - Kentridge et al., (1997) tested the suggestion while looking for scattered regions of spared vision in a patient using a procedure which ensured no effects of eye movements.
 - Kentridge et al., noted that blindsight did not extend across the whole area of the scotoma, but was evident in some areas even after eye - movements had been eliminated.

- However, these lead to the conclusion that even though there may be some spared islands within the scotoma, these areas cannot account for all blindsight.
- Furthermore, Cowey (2004) reported results from an fMRI study wherein fMRI scanning of several blindsight patients has shown 'not a shred of evidence' of any sparing of the striate cortex in the area of the scotoma.

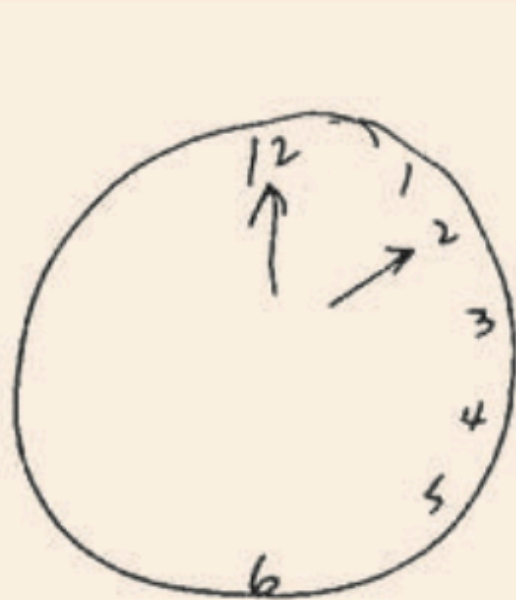
- *Final Words:*

- One of the possible explanations or implications of blindsight that have been put forward is that we have two separate visual systems, one primitive non – striate system and a more advanced striate system.
- The primitive non – striate system might be sensitive to movement, speed, & other potentially important characteristics of a stimulus without giving rise to conscious perception.

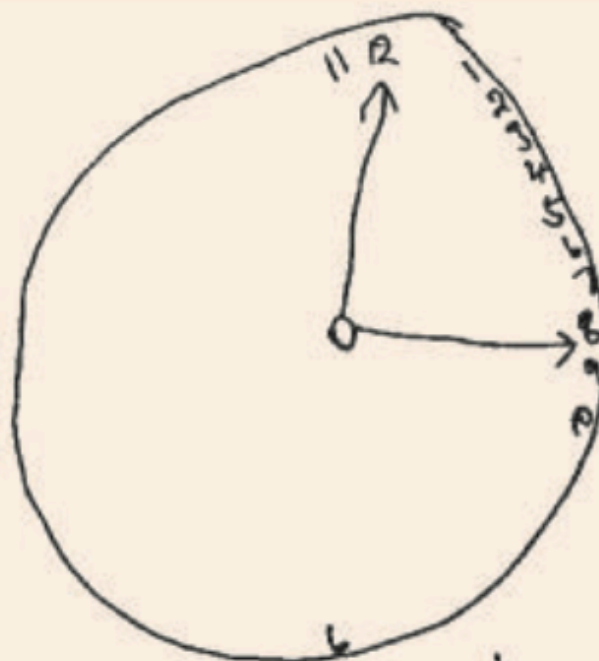
- Another similar explanation was that the striate & non – striate systems have evolved for different roles for e.g. the striate system has evolved to allow the identification of an object whereas the non – striate system has evolved to allow the localisation of the object in space.
- It is possible that during blindsight only the striate system is damaged whereas the non – striate one remains active.

Unilateral Spatial Neglect

- In unilateral spatial neglect – patients fail to respond to stimuli which they can see.
- A patient can have normal vision yet fail to react to objects or events to one side of the space – hence the term unilateral. The condition has also been referred to as *hemi – attention, contralateral neglect, & spatial neglect*.



2 o'clock



the clock is set at 8 o'clock

Image: Groome D. et al. (2013). An Introduction to Cognitive Psychology: Processes and Disorders. *Psychology Press*. 3rd Ed. (Fig. 4.3; pp. 114)

- The main cause of unilateral spatial neglect is stroke, an interruption to the brain's blood supply.
- Up to 84 percent of patients with damage to the right hemisphere of the brain from stroke will show evidence of ignoring information on their left visual field. Similarly upto 64 percent of patients with left hemisphere damage could show the opposite pattern.
- For some reason, left neglect following right – hemisphere lesion is markedly more severe & persistent than it's right neglect/left hemisphere equivalent.

- Patients with USN may fail to notice an object in 'clear view' on the left , ignore people approaching from the left, eat food only from the right side of the plate, or wash & dress only the right side of their own body.
- USN impact upon many activities of daily living, can exclude people from rehab and is associated with longer hospital stays and dependence on other.

- A disorder of attention?
 - USN is a failure of or difficulty in responding to information on one side of space that cannot be explained by basic sensory loss.
 - The obvious difference between USN & visual field loss is that the former can exert an influence across modalities, while the latter is restricted to vision.
 - USN has been reported in audition, tactile exploration, touch & body sensation (Vallar et al., 1993; McIntosh et al., 2002), imagery (Bisiach & Luzzatti, 1978) & even smell (Bellas et al., 1988).
 - While visual field losses are strictly retinotopic; USN varies in different spatial frameworks, i.e. it may occur for objects to the left side of the body or for objects on the left side of something regardless of where this is in relation to the person.

- USN may occur for the left side of each object within a scene (Driver & Halligan, 1991).
- Marshall & Halligan (1993) describe how patients kept on drawing only the left side of plants when asked to copy pictures.
- Is USN a disorder of attention?
 - Patients have been shown to attend to the left if cued to do so or if particularly salient events draw attention there.
 - Also, if a stimulus appears in face of competition with a rival on a good side, it is inevitably ignored.
 - There seems to be a gradient between left to right & not a clear boundary.

- Also, case studies by Bisiach & Luzzatti (1978) – patients were asked to describe the Piazza del Duomo in Milan from memory – they could describe the landmarks on their right; but when they were asked to describe the same landmark from the other end, the previously ignored landmarks could come back to attention and by Marshall & Halligan (1988) demonstrate that USN certainly seems to be more of a disorder of attention than of visual perception.

- Rehabilitation in USN

- Encouraging patients to look towards & be aware of the left has not produced the desirable outcomes, though movement of the left arm/hand has been found to generally enhance the visual awareness of the left side.

- Explaining USN:
 - USN occurs at remarkably high frequency following stroke & has also been reported following damage to a wide variety of brain areas including the parietal, temporal & frontal lobes and even subcortical areas (Mort et al., 2003).
 - This has led to the idea that normal spatial attention may reflect a dynamic & easily disrupted competitive balance between the widely distributed networks in the hemispheres (Mesulam, 1999).
 - In this view the left hemisphere is pushing attention into the right space & the right hemisphere is pushing attention back to the left.
 - So, in USN we are not just seeing the effect of the lesioned hemisphere but its exaggeration due to the suppressing effects of the undamaged rival hemisphere (Kinsbourne, 1977).

Visual Agnosia

- Agnosia – a Greek word meaning ‘non – knowledge’.
 - Patients with visual agnosia are not blind and the sensory processes are usually intact. However, the condition refers to an impairment in the ability to visually recognise objects.
 - They can move around & navigate spatially; can also recognise objects through other sensory modalities, but they just cannot recognise objects visually.

- Apperceptive & Associative Agnosia:
 - Someone suffering from apperceptive agnosia can be thought to have normal visual acuity with an inability to draw an object, to say whether two similar objects were the same or different or even to describe the component parts of an object.
 - Someone suffering from an associative agnosia would be able to draw an object, to match similar objects and be able to describe the component parts but they would be unable to recognise the objects they had just seen or drawn.

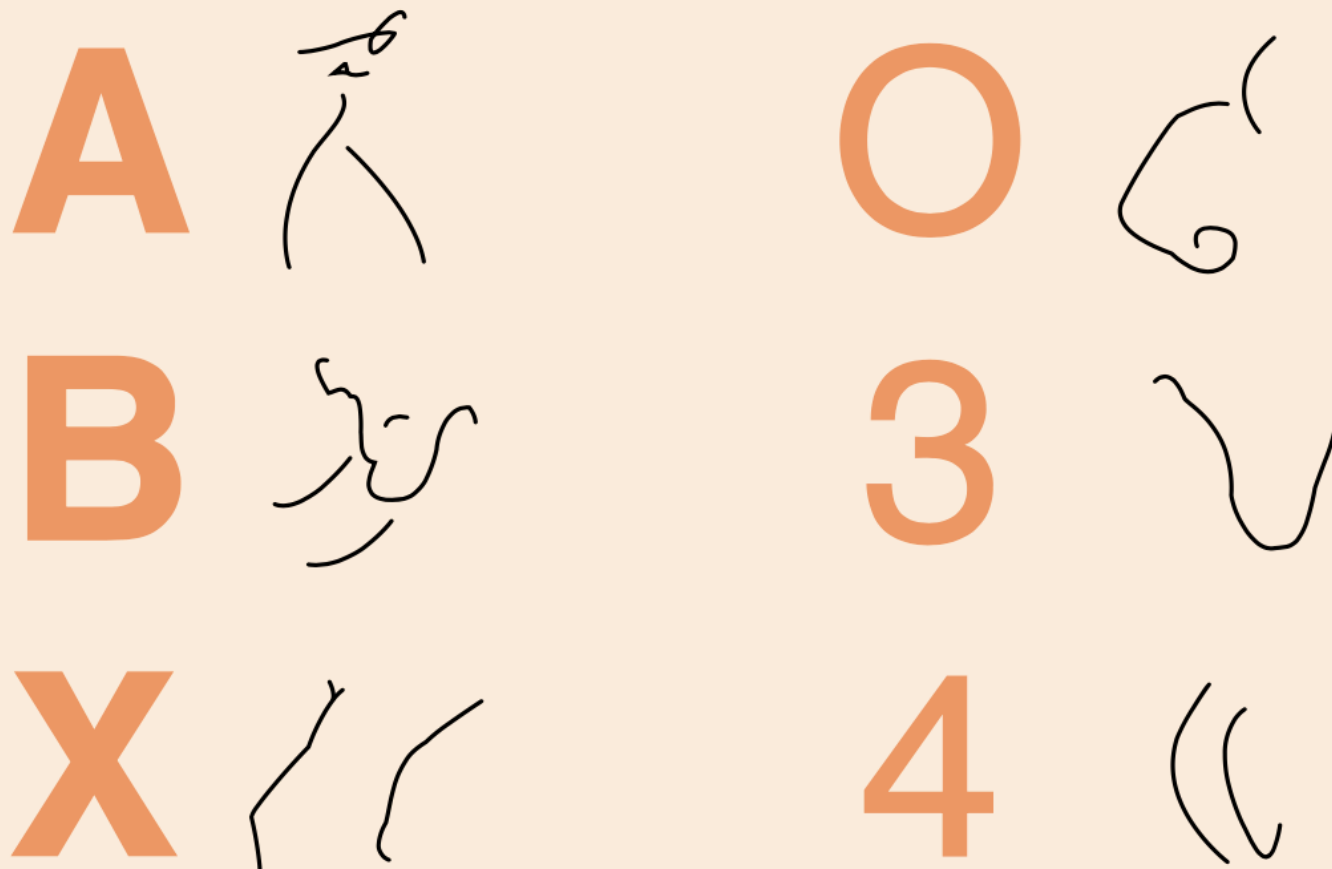


Figure 4.4 The attempts of a patient with apperceptive agnosia to copy six simple figures.

Source: Farah (1990), reproduced by permission of MIT Press.

- Lissauer (1890) proposed that these two stages were serial & hierarchical.
 - So, in the apperceptive stage the elements or components of the object are established & in then in the associative stage these elements are integrated into a representation of the whole object which enables recognition & identification.
 - This means that the patient with pure apperceptive agnosia have na intact store of knowledge about objects, but as they are unable to distinguish the shape of objects they are unable to identify the objects visually.
 - but patients with pure associative agnosia are able to perceive the objects but are often unable to identify them.
 - This principle was found useful in differentiating the patients with pure apperceptive or associative agnosia; those who could not copy had the former while the other kind had the latter.

- Form & Integrative Agnosia

- Form agnosia is used for patients who are unable to discriminate between objects and are unable to copy line – drawings of objects (Farah, 2004).
- Integrative Agnosia is the accepted term for associative agnoisa as it reflects the processing difficulties experienced by the patients. So it is used to refer to patients who can perceive the individual shapes and elements of objects but are unable to integrate these into a representation of the whole object.
 - Integrative agnosia has been reported with incidents of stroke, brain trauma & AD.

- Life with Visual Agnosia

- Patients (like HJA, Humphreys & Riddoch, 1987) with visual form agnosia experience a very confused & distorted visual world in which almost nothing seems familiar and even basic forms are indistinguishable from each other.

- Almost like looking at the world through a very powerful microscope; wherein despite seeing the details of the objects around it is hard to put together a mental picture of the overall structure.
 - Goodale & Milner (2004) describe such a patient Dee, who has severe visual form agnosia as a result of CO poisoning. Despite being unable to recognise objects or their shape, she uses the surface characteristics like texture & colour to help recognize objects.

- Animate vs. Inanimate Objects
 - A patient SA could identify animate objects much better than inanimate object while another HJA showed the reverse pattern.
 - Similarly, JBR could name drawings of many non – living objects (such as a spade or hairbrush), but he could not name the drawings of living things (such as dog or fly) or musical instruments (such as a trumpet) (Warrington & Shallice, 1984).

- Farah & McClelland (1991) argue that there is a difference between identifying living versus non – living things may in fact be an artefact.
 - In most cases where there is impaired knowledge of perceptual attributes with intact knowledge of functional properties, there is also an impairment for inanimate objects.
 - Because perceptual attributes (colour, size, shape etc.) are crucial for identifying animate objects; the identification of inanimate objects is crucially linked to functional attributes in semantic memory (Riddoch & Humphreys, 1987).

Prosopagnosia

- Is a form of agnosia that relates to faces.
 - The term prosopagnosia was first used by Bodamer (1947), who examined 3 patients, who, he believed showed a face – specific deficit, as they were unable to recognise non-face objects normally.
- People with prosopagnosia cannot recognise familiar faces of family members, friends & even themselves in the mirror, though their visual or sensory processes are intact.

- However, individual with PA are often able to use other cues such as voice to recognise familiar people.
 - Meaning that identity & semantic information has not been lost.
- Bruce & Young (1986) used evidence gained from studying individuals with face processing deficits and from studies of normal individuals to propose a model of face processing.
 - The model suggests that the recognition of identity, expression & facial speech analysis are independent processes, & subsequent evidence from brain damaged patients largely supports this view.

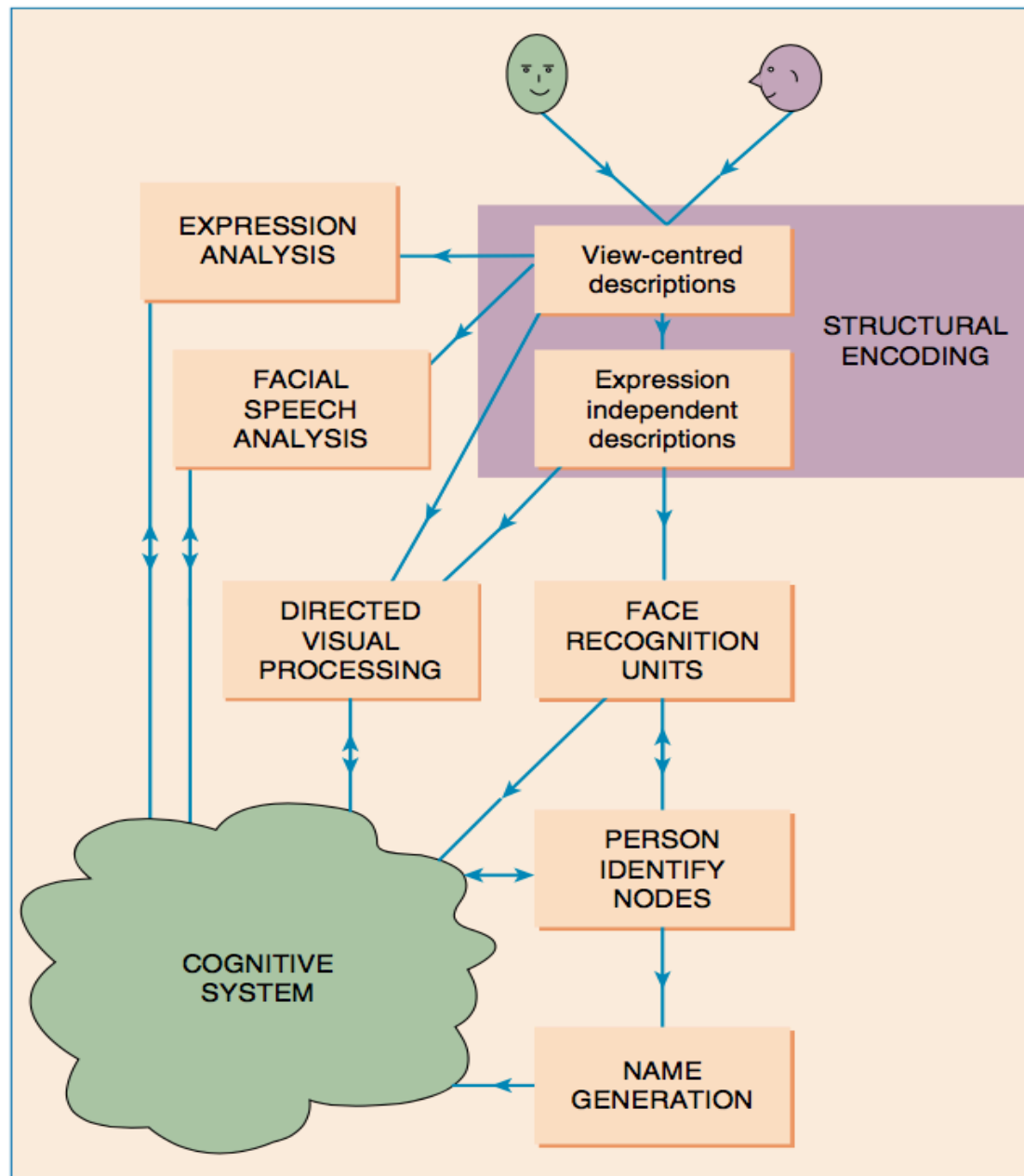


Figure 4.8 Bruce and Young's model of face processing showing independent pathways for face recognition, expression analysis and speech analysis.

Source: Bruce and Young (1986), reproduced by permission of the British Psychological Society.

Image: Groome et al. (2013). *An Introduction to Cognitive Psychology: Processes and Disorders*. Psychology Press. (Fig. 4.8; pp. 127).

- Given, that face recognition is one of the most demanding and sophisticated tasks that our visual system undertakes, it is unsurprising that an impairment in this ability can be acquired through brain damage.
 - However, brain damage rarely leads to complete destruction of the ability to recognise faces & there is a lot of variation in the severity of impairment, the associated deficits and types of face processing skills as well as in the location of and type of lesions that result in acquired prosopagnosia (Barton, 2008).

- Living with prosopagnosia
 - Data from imaging studies has also demonstrated that most cases of prosopagnosia have damage in the fusiform and lingual gyri, although there are also cases of damage in more anterior temporal areas (e.g. Gainotti et al., 2003).
 - The FA has been shown to be a key structure in face & object processing, & numerous studies have shown that the fusiform gyrus contains an area dedicated to face processing called the *fusiform face area* (FFA) (e.g. Haxby et al., 1994).
 - There is variability however, in the location of the FFA across individuals, & this may help to explain why prosopagnosia sometimes seems to occur with damage only in on hemisphere (Bruce & Young, 2012).

- Such variation has led researchers to think that there is no single area in the brain that is responsible for processing faces – rather a distributed neural network made of many bilateral regions is involved (Haxby et al., 2000).
- Barton (2008) reviewed the data from 10 patients & concluded that most severe impairments were found in patients who had bilateral occipito – temporal lesions involving the fusiform gyri.
- Also, he concluded that the right fusiform gyrus was involved in configural processing of faces & that memory for faces was more severely disrupted when these bilateral lesions included the right anterior temporal lobe damage.

- Prosopagnosia – face specific disorder
 - Riddoch et al., (2008) studied a patient FB & found that she was unable to identify faces of famous people that she was previously familiar with; but could make age, gender & expression judgments. Also, she could learn object names fast & could also respond to parts of faces ; but not whole faces. It can be safely concluded that she demonstrates a relatively pure form of prosopagnosia.
 - McNeil & Warrington (1993) describe the case of WJ, who took up farming after becoming prosopagnosic and remarkably showed evidence of being able to recognize his sheep; despite remaining prosopagnosic to human faces.

- Assal et al., (1984) described a farmer who was initially unable to recognise either humans or cows but after 6 months recovered the ability to recognise human faces but not cows.
- But while, there are such dissociations in the literature, Duchaine & Garrido (2008) argue that dissociations are not enough to provide support for face – specificity in prosopagnosia.
 - They state, ‘a network of areas is involved in face processing & their interactions remain poorly understood’.

References

- Groome D. et al. (2013). An Introduction to Cognitive Psychology: Processes and Disorders. *Psychology Press*. 3rd Ed.