

VISUAL AGNOSIA — AN UPDATE ON DISORDERS OF VISUAL RECOGNITION

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Abstract

Normally, we can recognise objects around us at a glance. However, selective brain damage can cause visual agnosia. Patients with this disorder are unable to recognise familiar objects, despite normal visual acuity. Although they can see well enough to accurately describe parts of the object, they cannot recognise what the object is or what it may be used for. What is more puzzling is that, when patients are allowed to hold the object or hear its characteristic sound, they can often identify it immediately. The study of this disorder has led to considerable progress in our understanding of the various ways visual processing can break down. A review is presented of the different types of visual agnosia which can occur, and implications for normal visual object recognition.

Key words: Visual object recognition, apperceptive agnosia, associative agnosia.

INTRODUCTION

Visual object recognition appears to be a natural and effortless achievement. Normally, we can "apprehend the meaning of objects, our prior associations with them, and their uses, from vision" with no difficulty at all.¹ However, damage to the posterior regions of the brain can cause visual agnosia. Visual agnosia is a recognition disorder that is not due to a primary sensory impairment, language deficit or intellectual deterioration.² Essentially this refers to patients who can still *see* objects but are unable to recognise what they are. When patients are allowed to feel the object or hear its characteristic sound they can usually identify it immediately.

Visual agnosia has been surrounded by much scepticism and controversy. For many years investigators argued that visual agnosia did not exist. For example, Bender and Feldman³ claimed that all reported cases of visual agnosia

could be explained by visual field defects and/or mental deterioration. However, current research indicates that visual agnosia not only exists, but is a complex disorder caused by impairment to different stages of visual object recognition.¹

Although significant advances have occurred in our understanding of how visual recognition breaks down, there is often confusion in its clinical assessment and diagnosis. Patients with visual recognition disturbances typically present with vague visual problems following injury to the brain. For example, patients may complain that "everything is slightly out of focus" or simply "I can't see". These patients are referred to the orthoptist and the ophthalmologist for visual assessment. To establish the diagnosis of visual agnosia it is critical to rule out the existence of any sensory impairment. Therefore the orthoptist has a role in the assessment of visual agnosia and should be aware of why and how this disorder occurs.

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The aim of this paper is to review two potential explanations of visual agnosia: the traditional model and the more recent multistage model. This review will focus on how the underlying processes of normal visual object recognition can breakdown to cause visual agnosia.

TRADITIONAL MODEL OF VISUAL AGNOSIA

The earliest account of visual agnosia was proposed by Lissauer in 1890.⁴ He proposed that two different stages are necessary for object recognition: apperceptive and associative. The apperceptive stage involves deriving conscious awareness of the sensory impression of an object. The associative stage involves establishing the meaning of the object by linking it to previous recollections and experiences.

(1) *Apperceptive agnosia*. Patients with a defect in the apperceptive stage are unable to recognise differences that distinguish two similar objects. They cannot name, match or copy pictures of objects or simple shapes. However, they can identify colours and appreciate changes in light intensity, as well as detect direction of movement.

(2) *Associative agnosia*. Patients with a defect in the associative stage are able to perceive objects reasonably well as they can match and copy objects correctly. However, they are then unable to recognise any of these objects. These patients are considered to have normal perception "that has somehow been stripped of its meaning".⁵

MULTISTAGE MODEL OF VISUAL AGNOSIA

Although Lissauer's model of visual agnosia has remained in use for over a hundred years, it is now regarded as inadequate. The increasing number of reported case studies indicates that the clinical presentation of visual agnosia varies considerably from one patient to another.⁶ Therefore it is argued that Lissauer's two stage model conceals the diversity of patients' symptoms and the complexity of underlying processes involved in visual object recognition.

According to Humphreys and Riddoch's model^{1,7,8} there are 5 different stages or levels

of visual object recognition plus a final 'matching' process:

1. The first stage involves extracting form information, including global object shape and local object features.
2. The next stage utilises the above information to obtain a retinotopic object description, that is, a description coded according to the image on the retina. This allows the geometric features of the object to be "binded" together into a perceptual whole.
3. After integrating all of the above information, a non-retinotopic object description is abstracted. In other words, orientation is assigned to the whole object so that it can be recognised from different viewpoints.
4. The fourth stage is concerned with accessing or using form knowledge, that is, stored information about the typical structure of objects. Essentially, this means the persons memory of an object's shape is matched against the object description obtained from Stage 3.
5. The next stage involves accessing semantic knowledge, that is, a person's knowledge or stored information about the meaning and function of objects.
6. Finally, a 'matching' process occurs between the object description (derived from stages 1, 2 and 3) and the persons memory of that object's shape and function (derived from stages 4 and 5).

According to this multistage model of visual object recognition, visual agnosia can be fractured into various subtypes. Each type of visual agnosia can be explained by an impairment to a particular stage of visual object recognition.

(1) *Impaired shape processing*. This refers to patients who have difficulty recognising objects as well as copying or matching simple shapes. Yet, like patients with associative agnosia, they have intact perception of colour, light and movement. Impaired shape processing is usually caused by multiple lesions in the occipital cortex due to carbon monoxide poisoning.⁹ These lesions are thought to produce undetected minute blindspots scattered throughout the patient's visual fields.

(2) *Impaired integration processes or loss of stereoscopic vision.* These patients have intact form information as they can match and copy simple objects correctly. However, they have problems with tasks requiring integrated form information. For example, patients can see the detail of objects clearly but do not recognise the object as a whole. This may be due to an impairment in integrating 2-dimensional shape information or due to the loss of depth perception. It typically occurs following bilateral damage in the posterior regions of the brain.

(3) *Impaired transformation processes.* Patients with this disorder can perform simple matching tasks and identify objects seen from a conventional view, but cannot recognise objects seen from unusual angles. For example, they can easily recognise a bucket from the more conventional side view, but not when it is viewed from directly above. Impaired transformation processes tend to be associated with damage to the parietal lobe.

(4) *Impaired object form knowledge.* Such patients can match and copy objects with no difficulty at all, but nevertheless have problems distinguishing between familiar and novel objects as well as drawing from memory. This occurs because patients can no longer remember what previously familiar objects look like.

(5) *Impaired semantic knowledge.* These patients have no difficulty with visual processing up to this point; they can match and copy objects correctly as well as recognise objects seen from unusual angles. However, they are unable to perform tasks which require classification on the basis of functional characteristics. For example, patients' cannot match semantically related objects such as a light bulb and a candle. This occurs because patients cannot remember the function of the object or any previous associations with the object. Such an impairment tends to occur after bilateral temporal lobe damage.

(6) *Impaired access to semantics.* These patients, like associative agnosics, perform normally on all tests of visual matching and copying as well as on verbal tests of object knowledge. Nevertheless, they still have difficulty recognising objects, especially when surrounded by other

structurally and semantically similar objects. For example, patients cannot recognise a horse if it is surrounded by other animals such as a cow or a pig. Problems recalling knowledge about an object's shape and meaning tends to be associated with posterior left hemisphere lesions.

DISCUSSION

This review indicates that disruption to the various stages of visual object recognition can result in many different types of visual agnosia. Clearly the hierarchical nature of normal visual object recognition is reflected in the clinical presentation of this disorder. In its severest form, patients may present as legally blind as they are unable to match, copy or recognise even simple objects. In its mildest form, patients may present as essentially 'normal' on most matching and copying tasks but nevertheless have problems recognising objects in specific situations.

The possibility of impaired shape processing is raised if the patient cannot recognise or match letters, shapes and pictures, demonstrates normal visual acuity using objective measures such as the Catford Drum and Stycar Balls and also has intact colour, light and movement perception. These patients experience significant problems with daily functioning. They cannot recognise even simple objects by vision yet can by other modalities such as touch and sound. The possibility of impaired integration processes is raised if patients have poor stereopsis and no other sensory defect. Such an impairment will cause difficulty with daily functioning as these patients typically adopt a slow feature-by-feature analysis of their environment since they cannot perceive objects as a whole.

An impairment to any of the other stages of visual object recognition is unlikely to be detected during the orthoptic assessment. These higher levels of visual processing are investigated by the neuropsychologist using a range of specific tests. They may involve identifying conventional and unusual photographs of objects (transformation processes), distinguishing between novel and familiar objects (accessing form knowledge), classifying objects according to its functional use (semantic knowledge) and distinguishing between

similar objects (access to semantics). The possibility of disruption to the higher stages of visual object recognition is raised if patients present with complaints of visual problems despite normal visual acuity and intact elementary sensations, as well as a history of occipital, parietal or temporal lobe damage, usually due to a stroke, tumour or head injury. Many of these patients function reasonably well and only experience problems recognising objects in particular situations.

CONCLUSION

The term 'visual agnosia' is a convenient shorthand expression that is used to describe patients who despite normal visual acuity, cannot recognise objects by vision yet can by other modalities such as touch and sound. What is often overlooked is that this term actually refers to a heterogeneous group of patients. It is now evident that different types of visual agnosia can occur when different stages of visual object recognition break down. There is no doubt that orthoptists are able to identify patients with an impairment to the early stages of visual recognition concerned with shape processing and integration processes. However, orthoptists should also be aware of the other types of

visual agnosia and indicate when higher levels of visual object recognition need further investigation.

References

1. Humphreys GW and Riddoch MJ. To see but not to see: A case study of visual agnosia. London: Lawrence Erlbaum Assoc, 1987a.
2. Bauer RM and Rubens AB. Agnosia. In Heilman KM and Valenstein E (eds). *Clinical Neuropsychology*. New York: Oxford University Press, 1985.
3. Bender MB and Feldman M. The so-called 'visual agnosias'. *Brain*, 1972; 95: 173-186.
4. Lissauer H. A case of visual agnosia with a contribution to theory, 1890. In Shallice T and Jackson M. *Agnosia*. *Cog Neuropsy*, 1988; 5: 153-192.
5. Benson DF. Disorders of visual gnosis. In: Brown JW (ed). *Neuropsychology of visual perception*. Hillsdale, NJ: Lawrence Erlbaum Associates, 1989.
6. Farah MJ. *Visual agnosia. Disorders of visual object recognition and what they tell us about normal vision*. London: MIT Press, 1991.
7. Humphreys GE and Riddochs MJ. The fractionation of visual agnosia. In: Humphreys GE and Riddochs MJ (eds). *Visual object processing: A cognitive neuropsychological approach*. London: Lawrence Erlbaum Associates, 1987b.
8. Riddoch MJ and Humphreys GW. Visual agnosia: anatomical and functional accounts. In: Kennard C and Rose FC (ed). *Physiological aspects of clinical neurophthalmology*. Year book medica publishers, 1988.
9. Campion J. Apperceptive agnosia: frameworks models and paradigms. In: Humphreys GW and Riddoch MJ (eds). *Visual object processing: A cognitive neuropsychological approach*. London: Lawrence Erlbaum Associates, 1987b.
10. Benson DF and Greenberg JP. Visual form agnosia. *Arch Neurol*, 1969; 20: 82-89.