Journal Club

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Novel Hypotheses from a Neuropsychological Case Study: Is the Visual Ventral Cortex Critical for Both Category-Generic and Category-Specific Form Perception?

Johanna C. Goll

Dementia Research Centre, Institute of Neurology, University College London, National Hospital for Neurology and Neurosurgery, London WC1N 3BG, United Kingdom

Review of Karnath et al.

In 1991, Milner et al. described a neurological patient, D.F., who was able to calibrate precise movements toward objects but unable to perceive their visual forms. Despite being able to discriminate basic visual properties (e.g., color, intensity), she was impaired in perceiving geometric shape information and, as a consequence, also in object apperception (perceiving the structure of three-dimensional objects). This deficit is often termed visual form agnosia, to distinguish it from cases of impaired object apperception with intact shape perception (Milner et al., 1991). The observation of D.F. inspired the hugely influential “dual stream” model of visual cognition, which postulates independence between processing for perception in a “ventral” pathway and processing for action in a “dorsal” pathway. The dual stream model is also supported by further neurological phenomena: in blindsight, the ventral stream is damaged at V1, causing a lack of conscious visual perception, yet patients

show residual abilities for visually guided action; in optic ataxia, the dorsal stream is damaged at the posterior parietal cortex, impairing visually guided action, yet visual perception is spared.

The focus of D.F.'s cortical damage in the lateral occipital complex (LOC) of the ventral stream, bilaterally (see James et al., 2003), has led to the inference that the LOC is necessary for visual form perception. Furthermore, this conclusion is consistent with a wealth of more recent functional imaging studies of visual form perception in healthy subjects (Grill-Spector et al., 2001). However, Karnath et al. (2009) present an additional neurological case, patient J.S., with a behavioral profile similar to that of D.F. but with an intact LOC and damage to the nearby ventromedial occipitotemporal area bilaterally. This damaged area overlaps substantially with the visual ventral cortex (VVC), a region commonly associated with the apperception of particular categories of visual objects such as faces, places, and words (Haxby et al., 2001). In contrast, the LOC is implicated in visual form analysis at a category-independent stage of processing (Grill-Spector et al., 2001). Despite the known involvement of the VVC in object apperception, it is conceivable that it also interacts with processes of form perception when, for example, a category-specific object such as face is viewed from an unusual angle. More-

over, visual form processing and object apperception are likely to overlap both cognitively and anatomically within the ventral stream, and it is possible that the VVC also makes a direct contribution to visual form processing. Thus, the new evidence of Karnath et al. (2009) calls into question the widely accepted view that the LOC is critical for visual form perception and, instead, suggests that the necessary substrate may lie within the VVC.

Karnath et al. (2009) provide two possible explanations for the divergence of patient J.S. from patient D.F. First, they suggest that the association of D.F.'s visual form agnosia with her site of maximal damage, the LOC, may be misleading because she additionally shows diffuse damage throughout her brain. In contrast, the evidence of J.S., who shows relatively focalized damage, may be more reliable. However, in a functional imaging study of D.F., James et al. (2003) compared the processing of intact visual objects to matched scrambled stimuli, and found the peak difference of activation between controls and D.F. within the LOC. This evidence makes it particularly difficult to suggest that D.F.'s destroyed LOC is not a critical determinant of her visual form agnosia. In response, Karnath et al. (2009) also provide a second explanation for their data, which rests more comfortably with the literature: both the LOC and the VVC may be necessary for visual form perception.

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Correspondence should be addressed to Johanna C. Goll, Dementia Research Centre, Institute of Neurology, University College London, Box 16, National Hospital for Neurology and Neurosurgery, London WC1N 3BG, UK. E-mail: goll@drc.ion.ucl.ac.uk.

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While this claim will require corroborating evidence, it may eventually lead to a revision of visual perceptual theory.

Whichever explanation of their data is preferred, the association between the VVC and visual form perception presented by Karnath et al. (2009) is strong because it is derived from a neuropsychological investigation, i.e., the study of a patient with an isolated cognitive disability and focal structural brain damage. By reporting these features in J.S., the authors illuminate a brain region without which visual form perception cannot occur, i.e., one that is absolutely necessary (Price and Friston, 2002). In contrast, it is widely accepted that functional imaging studies of healthy subjects are unable to differentiate between necessary and superfluous brain areas. For example, in healthy subjects, two or more regions may be active during the performance of a particular task, even though only one is necessary (Price and Friston, 2002). Alternatively, although healthy subjects may use a single area for a particular task, they may possess a second area that would perform equally well; this second area would only become visible in studies of patients who have endured damage to the first region (Price and Friston, 2002). The lack of a one-to-one mapping between areas of functional activation in healthy subjects and underlying cognitive processes highlights the importance of patient studies, like Karnath et al. (2009), to cognitive neuroscience.

Nonetheless, the case of J.S. alone cannot justify changes to models of visual perception because it cannot determine the precise nature of the VVC's necessary contribution to form processing. For example, one possibility is that deficits are directly caused by an absent VVC and the consequent obliteration of necessary computations. Alternately, deficits might result not from the absent VVC directly, but from absent or deranged information flow in the VVC's connections with other regions. Diaschisis could follow, in which the absent VVC area causes deranged computations in other structurally intact regions (e.g., LOC). Third, deficits could be independent of the VVC, instead caused by deranged processing in other regions that have sustained damage invisible to magnetic resonance imaging (MRI) (see Price and Friston, 2002, for discussion of all three scenarios).

An analogous theoretical problem was faced by Rossion et al. (2003) in accounting for a patient (P.S.) with a highly selective prosopagnosia. Structural damage in P.S. spared the region most fre-

quently regarded as necessary for face processing, the right fusiform gyrus (FFG), centering instead upon the nearby right inferior occipital gyrus (IOG). During face detection, functional MRI (fMRI) showed that the FFG was functionally preserved, indicating a critical role for the IOG in P.S.'s prosopagnosia (Rossion et al., 2003). However, a subsequent experiment showed abnormal FFG function during face discrimination (Schiltz et al., 2006). Given normal FFG function during detection, functional abnormalities during discrimination probably result from task-specific faulty inputs from other regions (e.g., IOG). Together, these results suggest that both the IOG and its interactions with the FFG are necessary for successful face processing. In J.S., similar fMRI tasks might establish the precise role of the VVC in visual form perception.

Thus, neuropsychological studies must rise to the challenge of defining the functional contribution that lesions make to cognitive deficits. In addition, they must also face several familiar methodological problems. For example, single cases are subject to the idiosyncrasies of the patients studied, and significant cognitive biases may emerge both before brain damage (due to individual differences) and afterward (due to functional reorganization). Additionally, the "transparency assumption," essential to the methodology, is vulnerable to violation. This states that the function of the damaged brain plus the function of interest is equal to the function of a healthy brain, but is invalidated if there is a complex nonadditive relationship between these factors. While such problems in neuropsychology are real and apparent, studies of healthy subjects suffer from analogous and equally worrisome difficulties. For example, healthy subjects show equivalent idiosyncrasies and can employ a range of cognitive strategies to carry out an identical task. Additionally, experiments involving any kind of subject may violate the "subtractivity assumption," which states that cognition in a baseline condition plus cognition in an experimental condition is equal to cognition in the task of interest. This assumption is logically equivalent to the transparency assumption and is invalidated just as easily. While many problems are shared by different approaches, there is one weakness unique to neuropsychology: the scarcity and chance occurrence of patients with lesions in theoretically important brain areas, which makes the task of evidencing necessary substrates for all cognitive

models extremely difficult. Nonetheless, such rarity cannot detract from the strength of neurological cases already described, and instead underlines the value of cases like J.S.

Unlike patient studies, healthy subject studies cannot determine the necessary brain regions for particular tasks. However, they can do better than merely indicating peripheral involvement. For example, studies showing enhanced activation following learning can causally implicate brain regions in a task. After training subjects to perceive a set of visual objects at brief exposures, Grill-Spector et al. (2000) found increased activity in the LOC, which additionally correlated with behavioral performance. However, despite the strength of this evidence, it is possible that results reflect increased levels of arousal for trained stimuli rather than enhanced visual form perception. Therefore, regardless of the useful causal inferences that can be made from longitudinal healthy subject studies, cognitive neuroscience remains reliant upon neuropsychological evidence to determine necessary substrates.

In summary, the neuropsychological method has enabled Karnath et al. (2009) to strongly suggest that the VVC is necessary for visual form perception, although further evidence will be required to clarify and develop this claim. In contrast, most previous studies of the VVC focus on its role in processing particular categories of visual objects such as faces, places, and words. Therefore, the suggestion that this area also plays a critical role in category-generic form processing sparks brand new questions about VVC functionality. For example, how distributed is processing of visual form and what specific contributions do the VVC and the LOC make? In general, to what extent is the VVC involved in category-specific and category-generic visual cognition, and are these processes structurally and/or functionally distinct? A distributed model of visual form processing would echo previous suggestions of distributed object representation across the VVC (Haxby et al., 2001). Like D.F. in the 1990s, the contemporary case of J.S. should stimulate novel hypotheses, which can be tested within a range of methodologies and may ultimately lead to the refinement of cognitive models of visual form perception.

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