Visual Neuropsychology

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Synonyms

Visual cognition; Visual neuroscience; Perceptual processing; Perceptual correlates; Vision science

Definition

Visual neuropsychology is the field of enquiry devoted to elucidating links between the anatomy and physiological functioning of visual cerebro-cortical structures and the ►visual perceptions and visually-mediated behaviors to which they give rise through experimentation in healthy and brain damaged humans.

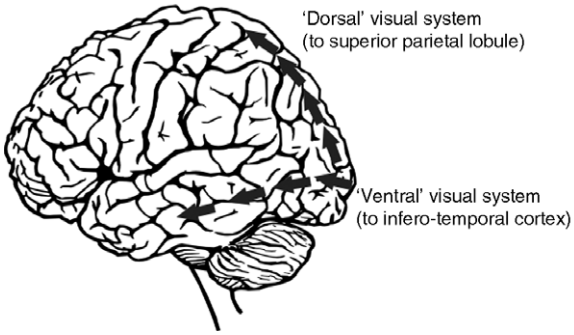
Characteristics

Visual neuropsychology is the sub-discipline of neuropsychology dedicated to understanding how visual information is processed by cerebro-cortical and subcortical mechanisms and how that information gives rise to perceptions and other behaviors. The discipline has grown in conjunction with knowledge of the anatomical connections and tuning properties of visual cortical neurons in ►occipital cortex and extra-occipital cortex. In particular, the discovery of streams of processing arising within the ►primary visual cortex (Brodmann's area 17; striate cortex; ►area V1) and extending into other cortical areas (see Fig. 1) (►Visual processing streams in primates; Extrastriate visual cortex), and the elucidation of the contribution of those processing streams to visual behaviors through scientific and clinical observations forms the basis of visual neuropsychology. Thus, visual neuropsychologists explore cortical functioning using psychophysical and neuroimagining techniques in healthy and brain-damaged humans to understand the nature of visually mediated behaviors.

Interestingly, while visual perceptions seem coherent and unitary, striking clinical dissociations illustrate that each stream can to some extent operate independently of the other (►Visual perception). Behavioral dissociations manifest as processing difficulties in one domain in the relative absence of difficulties in another, so suggesting independence of the two domains. An example of a single-dissociation within visual neuropsychology has been the observation of a patient who has impaired motion perception (►Motion blindness or ►akinetopsia) but almost normal form perception (see for example [2]). Double dissociations occur when two single dissociations with complementary deficit/ability profiles due to comparable size of brain damage are observed. Thus, while clinical participants are more heterogeneous in terms of their neural functioning than healthy participants, case studies illuminate sometimes unpredicted relationships between visual processes. Indeed neuropsychological case observations have often provided first clues on functional specialization within the human brain. It now seems clear, then, that visual perceptions and visually guided behaviors arise from simultaneous and largely parallel processing in both streams (►Visual perception).

Akinetopsia

Akinetopsia or motion blindness is defined as a selective impairment in perceiving the direction and speed of visual motion stimuli (►Visual motion processing), while other aspects of the visual scene such as color and form are perceived normally. The most clear and comprehensive demonstration of akinetopsia has been provided by Zihl and colleagues [2] who described a patient suffering from extensive and bilateral brain damage at the occipito-temporo-parietal junction. While the patient was unable to discriminate objects in motion the ability to perceive static form and color was normal. Interestingly, auditory motion perception and the perception of static and biological forms defined by visual motion stimuli are normal or much



Visual Neuropsychology. Figure 1 The so-called "where" pathway runs dorso-medially from visual area V1 through visual cortical ►areas V2, the ►V3/V3a complex, the ►superior temporal sulcus (►STS) or ►middle temporal sulcus (MT) to ►posterior parietal cortex (►PPC). It encodes primarily spatial- and timing-related information, and is also heavily implicated in visuo-motor processing. Damage along this pathway can result in a number of deficits, the most striking of which is ►spatial neglect, akinetopsia, or optic ataxia (see below). The second pathway runs from area V1 ventro-laterally through areas V2, V3, V4 and to the ►inferior temporal cortex (►IT). It processes information relating to the color and shape of objects within the visual array and for that reason commonly is referred to as the "what" pathway (see for example [1]). Damage along this pathway can result in a number of deficits; the best studied are ►visual agnosia and prosopagnosia (see below).

less impaired. Patients with severe motion blindness may also suffer from illusory visual motion perceptions suggesting that ►area MT±/V5 is also relevant for conscious motion perception.

Brain damage, from tumors, trauma, or stroke, may lead also to more complex forms of motion blindness such as a selective deficit in the detection and discrimination of forms defined by visual motion stimuli (►Form-from-motion (FfM) blindness). Double dissociations between motion blindness and FfM blindness also have been observed [3]. FfM blindness seems to arise due to damage to at least three distinct posterior processing areas: Severe FfM blindness without akinetopsia is due to damage in, usually, the right hemisphere ventral occipito-temporal cortex, whereas FfM blindness with akinetopsia is due to damage to either or both area MT+/V5 and the ►lateral occipital complex (►LOC), an area that is highly active during object perception and object recognition [4] (►Visual object representation).

Finally, the perception of ►biological motion (BM) (►Biological motion processing) stimuli may also be impaired by focal brain damage. BM blindness seems to be associated with parietal and temporal lobe damage. Patients with BM blindness often have normal visual motion perception but often suffer also FfM blindness. Neuroimaging work in healthy subjects has shown that a distributed network of brain areas is involved in BM perception including the superior temporal sulcus, the lateral occipital complex, MT+/V5, and the ►fusiform gyrus. A remarkable aspect of BM is that, along with motion direction and speed information, it conveys also a large range of socially relevant information. Accordingly, it seems that left frontal brain damage may impair the perception of personality traits and that damage to right ►somatosensory cortex may impair the perception of emotions as conveyed by BM stimuli.

Balint's Syndrome

Named after Hungarian physician Rudolf Balint and sometimes referred to as ►Balint-Holmes syndrome in honor of the later contributor Gordon Holmes, the syndrome is classically defined as an acquired, triple-symptom complex. The original patient described by Balint exhibited a range of symptoms including firstly ►optic ataxia or misreaching for objects under conditions of visual guidance. Behaviors associated with this symptom include, for example, lighting the middle rather than the end of one's cigarette, or misreaching for a door-handle. Based on Balint's observations that when assessed independently his patient's elementary visual and motor capabilities appeared intact, the deficit was defined as being specific to visuo-motor integration (►Visuomotor integration). The second symptom, termed psychic paralysis of gaze by Balint but now more commonly known as ►optic apraxia, relates to the occurrence of sticky visual fixations. Such ►spasms occur in spite of intact extraocular function (and thus unrestricted eye rotations), and manifest as an inability to voluntarily shift one's gaze from one object within the visual scene to another. The third and final symptom reported by Balint was spatial disorder of attention (►Visual attention), now more commonly referred to as ►simultanagnosia. In this case, a patient's ability to recognize individual objects is compromised when those objects are presented simultaneously within the visual scene.

In the case of Balint's original patient and those reported subsequently, attempts have been made to map the neuroanatomical underpinnings of the triple-symptom complex. Whilst some lack of consensus remains about the neural structures giving rise to the syndrome, most reports implicate involvement of bilateral posterior parietal cortex: post-mortem results of Balint's and others' patients, and more recent neuroimaging studies support the contention that damage to bilateral occipito-parietal cortex underlies the syndrome. Certainly that analysis is consistent with the role in visually guided behaviors of the so-called visual dorsal processing stream (which includes

occipito-parietal cortex). Indeed, convergent evidence related to visuo-temporal processing capabilities – attributed directly to dorsal stream processing - has recently been reported. As predicted by models of normal dorsal stream processing, such capabilities are disrupted in patients exhibiting ►Balint's syndrome.

The cortical damage on which Balint's syndrome onset appears to be based arises via a number of mechanisms. These include trauma through accident, stroke, and the development of cortical tumors. High levels of co-occurrence have also been observed in patients exhibiting posterior cortical atrophy (►Posterior cortical atrophy) and degenerative diseases such as Alzheimers (►Alzheimer's disease). Perhaps unsurprisingly those same diseases lead also to akinetopsia. Consistent with the aetiology of those diseases, incidence of the syndrome is observed almost exclusively in adults, although at least one case study involving a child has been reported [5]. The most effective treatment strategies are multi-contextual and involve first raising awareness of their functional deficits in sufferers, and then teaching them to anticipate conditions under which deficits manifest. That process usually depends upon patients being taught functionally adaptive and compensatory techniques.

Problems disambiguating Balint's syndrome from disorders such as visual ►neglect (see below), along with the scarcity of classical triple-symptom presentations has led to recent re-conceptualizations of the syndrome. That is, the symptoms may not be sufficiently autonomous to satisfy Benton's criteria for an independent syndrome. Rather, attention is now focused on understanding the three symptoms individually rather than considering them as a consistent constellation of signs.

Optic Ataxia

Considered the typical visuo-motor deficit, optic ataxia (OA) was first described by Rudolf Balint as one of a constellation of symptoms associated with what later came to be known as Balint's syndrome (see above). Classic definitions of the disorder focus on pronounced deficits reaching for objects under conditions of visual guidance. That is, patients are deficient in their capacity both to reach for and grasp objects represented in visual space despite preservation of visual, motor, and musculo-skeletal processing capacity. For example, and as originally described by Balint, patients can search with their knife outside a plate for food positioned in plain view on the plate. Based on its frequent occurrence in the absence of the other symptoms of Balint's syndrome, OA has emerged as an independent neuropsychological disorder.

More recent reviews of the disorder cite three key characteristics. The first is the pronounced deficit in the control of direction of arm and hand movements made under visual guidance, particularly for objects located in *peripheral* visual space. The second is the specifically *visuo*-motor aspect of the disorder: equivalent motor tasks performed under ►proprioceptive guidance, for example, can be performed without deficit. The third characteristic is the specificity of the deficit for *immediate* visuo-motor functions. Tasks involving delayed behavioral responses are performed with higher levels of accuracy than those performed "online".

Neural accounts of the disorder emphasize the key role played by the action-oriented visual dorsal processing stream: Patients typically exhibit symptoms after extensive damage to posterior parietal cortex. Specific sub-areas involved likely include the ►superior parietal lobule, ►intraparietal sulcus, and ►occipito-parietal sulcus in various combinations. OA manifests as a consequence of both bilateral and unilateral parietal damage. In the latter cases, hemispheric asymmetries are observed: damage to right cortex results in mis-reaching via both hands to objects presented in contralesional visual space (the so-called *field* effect), whilst damage to left cortex results in mis-reaching to either visual field, particularly in the case of the right hand (the so-called *hand* effect).

Prosopagnosia

Also known as *face blindness* or *facial agnosia*, ►prosopagnosia is a disorder in which sufferers are unable to recognize previously familiar human faces (►Face processing in different brain areas). This is the case in spite of preserved ability to recognize objects (►Visual object representation) more generally and to make fine visual discriminations.

Historically multiple examples of patients presenting with such patterns of impairment were reported in the eighteenth Century (for an historical overview see [6]). However the term prosopagnosia itself was not coined until 1947. At that time, German neurologist Joachim Bodamer published a report identifying the existence of a number of patients deficient in the ability to discriminate between familiar individuals on the basis of visual inspection of their facial features. For example, Bodamer described one of his patients as manifesting a severe deficit in ability to discriminate friends, family or himself on that basis, whilst the ability to perform the same task on the basis of cues such as individual speech or gait patterns was retained.

Neural correlates of prosopagnosia have been identified primarily on the basis of lesion analysis. Such analyses most commonly implicate bilateral damage to the inferomedial part of the temporo-occipital region,

specifically the fusiform gyrus and ►lingual gyrus. Less commonly unilateral, right-hemisphere damage to the same region gives rise to the disorder [7]. Based on those findings and in acknowledgement of its key role in vision-based face recognition, the right mid-fusiform gyrus is now commonly referred to as the ►fusiform face area or FFA [8].

Acquired prosopagnosia arises usually from stroke damage, brain injury, or from diseases such as Alzheimer's and Parkinson's (►Parkinson's disease). However, less common congenital presentations have also been reported. The patterns of cortical disruption on which the disorder is based also commonly give rise to additional vision-based deficits including bilateral upper visual field deficits, ►achromatopsia or left ►hemiachromatopsia and ►topographagnosia (for a review see [7]).

Neurocognitive accounts of the disorder vary. The co-occurrence of prosopagnosia with deficits in performing general object recognition (►Visual object agnosia) has been presented as evidence that the disorder is domain-general. That is, prosopagnosia represents one specific manifestation of more general object recognition deficits. That account is weakened by the existence of facial/object recognition double dissociations: Complementary to face-specific deficits of the type described by Bodamer and others, the existence of patients with deficits in object recognition but not in face-recognition have been reported. In an example reported by Moscovitch and colleagues [9] patient CK was shown to be poor at recognizing non-face objects and at reading, but not deficient in terms of his capacity to recognize faces.

Initial diagnosis of the disorder is commonly based upon self-report and a simple clinical test in which one familiar individual, dressed in a similar fashion to several others amongst whom he or she stands, must be identified on the basis solely of visual inspection. An inability to perform that task, combined with tests demonstrating the face-specificity of the deficit while excluding the existence of general intellectual impairment or poor visual processing, form the basis for confirmation of the initial diagnosis. Once diagnosed, treatment options are limited. Most commonly, the debilitating social and functional aspects of the disorder are addressed through training in the implementation of compensatory strategies. These include using alternative perceptual cues such as the gait, speech patterns, and isolated (rather than global) facial features to identify individuals, and cognitive strategies such as keeping lists of the individuals likely to be present at particular times of day and in particular locations.

Spatial Neglect

►Neglect (also known as spatial, unilateral or hemi-spatial neglect) is a syndrome characterized by the tendency to act as if half of the world does not exist. Typically it manifests across sensory domains (vision, touch, audition, even smell) despite normal processing, at least peripherally, in each. Neglect arises typically from unilateral damage to inferior parietal or superior temporal cortex, often as a result of an infarction of the right middle cerebral artery. Involvement of ►prefrontal cortex, along with ►thalamus and ►basal ganglia, has also been implicated. While the syndrome occasionally manifests as a function of damage to left hemisphere, it arises more commonly, and with more severe and enduring symptoms from damage to right hemisphere.

The behavioral correlates of neglect can be mapped across three conceptual, patient-referent spatial areas: personal, peri-personal (within reaching distance), and extra-personal space (within sensory processing distance). Examples of behaviors related to personal space include the application of make-up only to the ipsi-lesional side of one's face, and the dressing of only the ipsi-lesional side of one's body. Symptomatic behaviors in peri-personal space include inattention to contra-lesional text and pictures presented in books, and lack of awareness of the presence of food positioned contra-lesionally on one's plate. Finally, behaviors associated with extra-personal space include a tendency to ignore people and objects in the contra-lesional hemifield and a failure to navigate routes or spaces that require contra-lesional turns or deviations.

Contemporary theories of the cognitive correlates of neglect focus on disruption to processes mediating mechanisms of spatial attention (►Visual attention). Such disruptions can occur in the absence of patient awareness (►Anosognosia) and can temporarily be ameliorated by the deliberate re-focusing of attention to a target within the neglected space. Similarly, the severity of neglect is spatially heterogeneous, becoming more severe as a function of distance into contra-lesional space. Thus, neglect must be differentiated from functional deficits associated with sensory (e.g. ►Hemianopia) or motor impairment, neither of which is moderated by attentional strategies. Interestingly, and in a further contrast to sensory and motor deficits, neglect can manifest in tasks requiring recall (►memory) and ►mental imagery. Memory deficits particularly are interesting: Patients may be unable to describe, from memory, that part of a familiar scene corresponding to neglected space. However, if asked to adopt a different perspective, one that moves features that previously fell into neglected space into ipsi-lesional space, those features can be recalled.

Diagnostically the most reliable tools are simple pencil-and-paper tests. On drawing-from-memory or copying tasks neglect patients will omit details from the neglected side of the object to be recalled or copied. On line-bisection tasks requiring participants to mark the mid-point of a horizontal line neglect patients

typically indicate their perceived midpoint far to the right of the actual midpoint (and normal subjects on the same task typically mark a little to the left of the actual midpoint, so-called ►pseudo-neglect). Similarly, on line- and other types of cancellation tasks neglect patients will delete only targets in their non-neglected hemi-field.

Amelioration of neglect can be achieved by ►opto-kinetic stimulation and ►caloric stimulation, but improvements are temporary. Similarly transient improvements are associated with the application of vibration to the neck. The only reliable and medium-to-long-term treatment for neglect seems to be achieved by using ►prism adaptation [10]. In the absence of an effective treatment for neglect most patients undergo rehabilitative therapies that focus on the development of cognitive strategies to overcome inattention to objects within the neglected hemifield.

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