

Some of the tradeoffs will always be present—for example, the tradeoff between increasing statistical power with more specific stimulus assumptions (using structured artificial stimuli, such as dots, bars, gratings) and losing statistical power with increased stimulus generality (noise stimuli in reverse correlation) occurs in every sensory modality and method. Characterizations using natural scenes as stimuli appear to strike an appropriate balance by providing a wide array of ecologically relevant data that efficiently probes the space of neural responses relevant to the animal. Even simple cells in V1 are quite complex and require mathematical descriptions that are more involved than traditional models, and better characterizations are necessary for a more thorough understanding.

Computational objectives, such as compression, sparse, and independent coding, offer succinct, high-level explanations of why these neurons have particular responses to stimuli. Of course, neural response characterizations and computational explanations are often idealizations. The neural code is not simply a set of isolated neurons firing at particular rates, but rather a complex combination of neurons firing at precise times in a population. Although many new methods of characterization may be quite sophisticated, they are strictly limited by the amount of data available; this limits model complexity, temporal and spatial extent of receptive fields, as well as temporal and population resolution. Also, attention and feedback modulate the neural code to selectively process the massive amount of information previously mentioned. The effects of attention make any narrow interpretation of sensory coding give way to a high-level, multimodal, and at times psychological understanding.

Making sense of the neural code is not a simple process. A wide variety of new methods and models have been introduced in recent years to address the apparent complexity of neural activity. The introduction of ideas regarding ecology and efficiency provide the opportunity to reduce this apparent complexity. They introduce a unifying framework that integrates many disparate aspects of neural coding. Neural response properties are linked to ecological goals through the study of statistical properties of the environment. This new framework allows researchers to address both “what” neurons respond to, and

more importantly “why” they respond that way. In this way, physiological, computational, and ecological results all become part of a more coherent picture of methods in neural representation and coding.

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*See also* Attention; Physiological; Auditory Processing; Central; Computational Approaches; Cortical Organization; Information Theory; Modularity; Multimodal Interactions: Neural Basis; Neural Recording; Psychophysical Approach; Reverse Correlation; Visual Processing: Extrastriate Cortex; Visual Processing: Primary Visual Cortex; Visual Scene Statistics

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## NEUROPSYCHOLOGY OF PERCEPTION

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The world surrounds us with an immense variety of sounds, colors, textures, and odors. In everyday life we need to receive and interpret this information to help us plan and adopt appropriate behaviors. Perception is the first phase of this interaction with the world. It progresses from initial encoding of data in a number of specialized sensory end organs for the different modalities (vision, hearing, taste, touch, and smell) through various stages of processing in subcortical and cortical structures in the brain, stages that elaborate,

focus, interpret, and ultimately integrate this information to provide us with what we regard as our experience of the world.

Given the complexities of perceptual processing and the wide range of neural structures involved, it is not surprising to find that a large number of human diseases frequently impair perception. How these disorders—particularly those involving the brain—impact perception and what this can tell us about how perception works is the research domain of perceptual neuropsychology.

Neuropsychology is the scientific discipline that investigates the structure and function of the brain by assessing the effect of brain lesions in relationship to specific psychological processes and behaviors. This entry describes the basic procedures and goals of neuropsychology, neuropsychological studies of visual perception, and neuropsychology of other perceptual modalities.

The neuropsychological approach consists of assessing cognitive functions (i.e., memory, attention, perception, language, reasoning, etc.) by relying on tests specifically designed to assess each of those cognitive functions selectively. Neuropsychological tests also provide an accurate assessment of a selective cognitive function according to different materials and sensory modalities. In the context of perception, for instance, a neuropsychological assessment involves the evaluation of perceptual skills in visual, auditory, or tactile modalities, confined to different types of information such as faces, objects, sounds, colors, or more complex stimuli. At each test, the patient's performance is compared to the performance of a group of healthy individuals matched for age and education, providing an accurate estimate of the degree to which a selective function is impaired in a brain-damaged patient.

For many decades, the neuropsychological approach was the only method available to study where functions were localized in the human brain, and such studies were often performed in tandem with studies using more precise recordings or artificial lesions in animals, particularly monkeys. In the modern era, functional neuroimaging techniques have added powerful new tools to the study of the relation between structure and function in the human brain. However, such approaches also have their limitations, and the neuropsychological approach still provides us

with information that cannot be obtained by other methods.

The two main approaches used in neuropsychology are the case study and the group study. Both have their advantages and disadvantages. *Single-case studies* with well-controlled experiments can provide important information about cognitive operations, but conclusions about the anatomic bases of the deficits in single cases is difficult. Natural lesions tend to be large, affecting a number of brain regions, limiting deductions about which region is the critical one. *Group studies* can show which lesions are associated with a specific functional deficit and which are not. On the other hand, group studies can suffer from the fact that any collection of patients is heterogeneous in many respects, regarding for example age, premorbid functioning, and lesion size and location. Careful selection, matching, and exclusion criteria can help, but heterogeneity can never be eliminated totally. Also, the assumption that all patients with a certain syndrome have the same functional deficit is not always correct, and group studies may inadvertently blur this distinction, leading to false conclusions about the anatomic correlates of function. To address some of the limitations previously described, neuropsychologists make use of an experimental technique named *dissociation*, which helps to identify the neural substrates of a selective cognitive function more reliably. In a *single dissociation*, the neuropsychologist is able to demonstrate that a specific brain lesion in region A is affecting the cognitive function X but not the cognitive function Y, which demonstrates that the two functions are independent and rely on different brain regions (a typical single dissociation is the inability to name an object when the patient can only see it, whereas he or she is able to name the same object by touching it). A *single dissociation* is strengthened when the neuropsychologist is able to demonstrate that a brain lesion in region A is affecting the cognitive function X but not Y and that a brain lesion in region B is impairing the cognitive function Y but not X. This is called a *double dissociation* and allows researchers to make conclusions about the independency of different neural substrates related to different cognitive functions (a typical example is a patient with a selective lesion in region A showing an impairment in visual memory but not

auditory memory, and a patient with a selective lesion in region B showing an impairment in auditory memory but not in visual memory).

### Goals of Neuropsychology

There are several reasons for using a neuropsychological approach to study any cognitive process. One of the earliest goals of neuropsychology was to correlate deficits in the mental processes of patients with the anatomic location of their lesions, the method of *anatomic-clinical correlation*. Using this logic, experimenters hope to gain insights into the neural structures that perform specific cognitive operations. If a lesion of a specific brain region results in a deficit in a certain behavior or percept, then the logical inference is that an important function that is required for that behavior or percept is performed by that cerebral region.

A second goal that has gained increased profile with the emergence of cognitive psychology as a field is to use psychological deficits as a *probe of the functional architecture of the mind* of how (rather than where) our brains process information. Many types of cognitive models have been developed to explicitly describe theories and mechanisms about the way our minds handle information. Common to many of these is the fractionation of a larger function into smaller modules that perform more limited and specific processes in a series of steps. In this context, a module is defined as a single component that is critical for the appropriate functioning of a more general cognitive skill. An example is the breakdown of face recognition into basic level encoding, perception of expression, perception of the facial elements that reveal identity, the matching of this identity information to memory stores of faces, and the accessing of semantic information about the person's name, biographical data, and so on. Which modules exist, the degree to which they exist independent of each other in the brain, and how information flows interactively between them are issues and proposals that can be constructed into a cognitive model, a functional architecture of how certain information is processed. Neuropsychological data can be a powerful means of testing whether these concepts are correct: If a patient's lesion selectively impairs a particular function and spares all other functions,

this provides strong evidence for the existence of a module for that function. This linkage of deficits to functional architecture has similarities to the linkage of deficits to structural damage in the anatomo-clinical correlative method, but its aims are different and do not depend upon the presence of a focal or visible brain lesion.

Probing the structural correlates and the functional architectural implications of deficits are probably the chief scientific aims of neuropsychology. However, as befitting a discipline that sits at the interface between medicine and psychology, there are also goals of the discipline that are focused more on the disease process than on the psychological theories. Thus, a third goal is to *understand how various neurological or psychiatric disorders impact the cognition of the patient* suffering from the problem. This may be done by unraveling some of the basic mechanisms of diseases that are currently poorly understood. For example, studies showing that face perception is abnormal in at least some autistic patients have led some researchers to hypothesize that, given the importance of faces in our interactions with other people, the social dysfunction in this disorder may stem in part from derangements in perceptual mechanisms. On a more pragmatic level, this can also be done with the purpose of contributing to the diagnosis of a disorder or evaluating the risk of its development in the future. The patterns of memory, attentional, linguistic, and visuospatial impairments on a well-constructed neuropsychological battery may help clarify which of several types of dementia is present in an early stage in an elderly person with recent behavioral changes, or which patients with minimal cognitive impairment are at greater risk of going on to develop Alzheimer's disease over the next five years, for example.

A fourth and also clinically oriented goal that is gaining in interest is the potential role of neuropsychology in the *treatment and rehabilitation of neurological disorders*. Whether behavioral training paradigms have the ability to change the course of a patient's deficit remains a source of conjecture for many psychological deficits. At present, there are many such attempts being undertaken in the field of hemineglect (failure to process stimuli on the left side of space), and the impact of face-learning protocols are being evaluated experimentally in the treatment of

prosopagnosia (the inability to recognize familiar faces) and autism. Even when the treatments being offered are not behavioral in origin, evaluations of their efficacy often are based in neuropsychological assessments.

The goals and the interpretation of data provided by neuropsychology are not without their own complexities and limitations. As with all disciplines, neuropsychology carries a set of assumptions about how the brain works. Whether these assumptions are valid for all cognitive operations can be questioned, and certainly all of them carry their own degrees of subtlety: few things are black and white. For instance, the ability to draw inferences about the functional and structural basis of human cognition from neuropsychological data requires that the mechanisms that the brain uses to process information do not radically change after brain damage. If a certain function X depends upon region A, then a lesion in A should cause a deficit M that reflects the loss of function X. However, if region B can learn to perform function X through some form of post-lesion plasticity (with either the same or a different type of computational mechanism than originally used by region A), then deficit M may not be apparent. In this case, the erroneous conclusion will be made that region A does not make an important contribution to the performance of X in the normal human brain.

Of course, there is evidence that the adult brain has some degree of plasticity and that things do change in the damaged brain. Cortical maps are reorganized after damage to peripheral structures, and even the opposite hemisphere appears to provide some compensation of loss of a lateralized function. These changes are somewhat limited, though, and it is generally assumed that these are minor quantitative variations rather than radically different qualitative rearrangements and shifts in computational mechanisms. Nevertheless, it is conceivable that for certain complex high-level perceptual processes, alternative routes and mechanisms to the same end might exist. In such a scenario, deficits may only emerge after a critical amount of damage has accumulated to disable not only the regions that perform the key computations in the normal brain, but also regions that might have been able to provide alternative compensatory routes or mechanisms for that function.

The complexity that this violation of *pathological constancy* imparts to the interpretation of neuropsychological data is evident.

### Neuropsychological Studies of Visual Perception

More is known about vision than any other perceptual modality: Hence, it is worth surveying what neuropsychological studies have contributed to our knowledge of visual function. The following is a short list of some of the more well-known neuropsychological syndromes of visual perception.

For over a hundred years, beginning with studies of soldiers in the Russo-Japanese War and World War I, it has been established that lesions of the occipital lobe lead to *cortical blindness*, with the location of the lesion corresponding to the area of the visual field lost (central versus peripheral, upper versus lower). Neurophysiological and neuroimaging studies have confirmed that the primary visual cortex (V1) is located in the banks of the calcarine sulcus, and that this serves as the primary cortical terminal of afferent visual information transmitted from the eyes through the optic radiations. From a neuropsychological perspective, visual loss from V1 damage conforms to many assumptions, including pathological constancy. The highly consistent relationship between the site of the lesion and the size and location of the blind area in the visual field on the opposite side of the lesion is well known and not an active area of research. More interesting is the question of what visual functions remain possible within these regions of blindness. Despite the denial of vision, some cortically blind patients can locate objects by pointing or eye movements, can sense the motion of objects, and can perform a number of other tasks at rates better than chance. This phenomenon is referred to as *blindsight*, and its investigation seeks to refine our notions of what visual operations are carried out independent of the processing in V1.

Beyond V1, an important anatomic concept that has grown from a convergence, first between neurophysiology and neuropsychology, and later from functional neuroimaging as well, is that of two visual processing streams, a dorsal one involving occipitoparietal cortical regions and a ventral one involving occipitotemporal regions, neural

pathways connecting occipital and parietal regions and occipital and temporal regions respectively. These visual processing streams were initially conceptualized as a *dorsal "where" stream* for information about the spatial location of objects, and a *ventral "what" stream* for information about the identity of these objects. More recent alternative formulations suggest that the dorsal stream is involved in preparation for action and the ventral stream in object recognition. The debate about these formulations continues to be fueled by data from neuropsychological studies of patients with dorsal or ventral lesions.

Occipitotemporal lesions result in a variety of perceptual syndromes. Severe bilateral lesions may cause a *general visual agnosia*, in which the patient is unable to recognize objects (e.g., the man who mistook his wife for a hat). One of the aims of current neuropsychological research is to understand the types of functional disturbance that can lead to this severe deficit. Time-honored distinctions contrast an *apperceptive* with an *associative* form of agnosia. In the apperceptive form, although the patient can see, the perceptual information is so degraded that it is impossible to recognize what the object is. In the associative form, the perception information is accurate, but the knowledge about the perceptual properties of objects is impaired or inaccessible, rendering the perceptual input useless. Neuropsychological research has also established a possible third form, an *integrative* agnosia, in which perception of parts and features of objects is accurate, but the ability to integrate the parts into a coherent whole object is impaired, impeding recognition of the entire item.

Selective agnosias also exist. Some patients suffer from prosopagnosia. These patients do not have problems recognizing objects at a coarse level (they can tell faces from hats), but more sophisticated distinctions between different faces (and in some patients, between different hats or other objects) are very difficult for them. Again, research has established that there is not one type of prosopagnosia, but several variants, from damage to different anatomic structures of a face-processing network and to different functional components of a face-processing strategy. *Pure alexia*, in at least some patients, can also represent a selective agnosia, in which the ability to read words is lost, even though the patient has sufficient residual linguistic

function to write, and can visually recognize other objects well. In other cases, pure alexia may represent a disconnection syndrome, due to damage transferring information from the visual cortex to linguistic processing centers in the left hemisphere.

*Topographagnosia*, the inability to find one's way in familiar surroundings, is another type of selective agnosia. It illustrates the complexities of functional modularity, because subjects navigating through space can use a variety of strategies. Different types of topographagnosia can result from damage to different orienting strategies. Some forms may be due to loss of the ability to recognize places and landmarks, and therefore a type of object recognition deficit: Not surprisingly, this type of topographagnosia is associated with damage to the occipitotemporal cortex. Other forms represent the inability to form a mental map of the environment, following hippocampal damage, or problems with utilizing a sequence of directions.

*Achromatopsia*, the loss of the ability to discriminate one color from another, is another deficit linked to occipitotemporal damage.

Occipitoparietal lesions do not impair object recognition, but result in a variety of syndromes involving spatial processing or the direction of attention into space. Though rare, defects in motion processing (*cerebral akinetopsia*) have been described. Bilateral occipitoparietal lesions are classically associated with *Bálint's syndrome*, which includes three separate features that are not always present in each patient. First is *simultagnosia*, the inability to process more than one object in the environment, which likely represents a failure of mechanisms of attention. Second is *ocular motor apraxia*, in which the patient has difficulty initiating and targeting eye movements to spatial locations accurately. Third is *optic ataxia*, in which the patient's ability to reach for objects is impaired. Both ocular motor apraxia and optic ataxia represent difficulties in deriving spatial coordinates of items in the environment for the guidance of the subject's own motor responses.

Unilateral occipitoparietal lesions are usually less dramatic. Hemineglect is the classic deficit, usually from a right-sided lesion, in which a subject fails to notice stimuli on the left side of space. *Dressing apraxia* may represent an impairment of

the ability to orient one's body to the complex and dynamic three-dimensional properties of one's own clothing.

### Neuropsychology of Other Perceptual Modalities

After vision, the next most studied perceptual modality in neuropsychology is audition. Just as there is a primary visual area, there is also a primary auditory cortical area, in the temporal lobe, and damage to this region causes *cortical deafness*. This disorder is rare, presumably because auditory information from each ear is distributed to both hemispheres, and damage to both temporal lobes is a far more infrequent occurrence than bilateral occipital lesions that cause cortical blindness. There are also blindsight-like issues with cortical deafness, regarding what sorts of auditory abilities might still be present. Beyond primary auditory areas, damage to the auditory association cortex may lead to *auditory agnosia*, in which subjects can still perceive frequency, intensity, and duration of sounds, but cannot recognize sounds, speech, or music. More selective auditory agnosias include *word-deafness*, in which subjects are unable to comprehend speech, and *amusia*, in which subjects cannot recognize music, most likely through loss of the ability to process melody. Neuropsychological studies have gone further to suggest that there may be selective deficits in recognizing musical contour versus musical intervals in amusia.

In the somatosensory modality (touch), there are also deficits linked to damage to the primary sensory cortex in the precentral gyrus. As well, there are patients with *tactile agnosia*, who have intact perception of shape and texture, who cannot recognize objects by touch. This too has been fractionated into an apperceptive (or integrative) form and an associative form.

For the modalities of taste (gustatory system) and smell (olfactory system), there is much less information. It has long been known that damage to the orbitofrontal cortex or anterior and medial temporal lobe can impair the sense of smell, but further classification of central olfactory disorders has not yet been attempted. The primary gustatory cortex is located in the insula and adjacent inner operculum, which project to the orbitofrontal cortex, allowing for an integration of smell and taste. There is even

less neuropsychological data on cerebral disorders of taste than there is for smell.

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*See also* Agnosia: Visual; Attention: Disorders; Brain Imaging; Consciousness: Disorders; Vision: Developmental Disorders

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## NONVERIDICAL PERCEPTION

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A person's entire life experience—everyone, everything, every experience he or she has ever known—exists to that person only as a function of his or her brain's activity. As such, it does not necessarily reproduce the physical reality of the world with high fidelity. *Nonveridical perception* is the sensory or cognitive discrepancy between the subjective perception and the physical world. Of course, many experiences in daily life reflect the physical stimuli that fall into one's eyes, ears, skin, nose, and tongue. Otherwise, action or navigation in the physical world would be impossible. But the same neural machinery that interprets veridical sensory inputs is also responsible for one's dreams, imaginings, and failings of memory. Thus, the real and the illusory or misperceived have the same physical basis in a person's brain.