

# [Methods of neuropsychological studies](https://assignbuster.com/methods-of-neuropsychological-studies/)

The term ‘ syndrome’ denotes the statistical co-occurrence of a cluster of symptoms. However, it is not imperative that all symptoms of a syndrome be present in a given patient. The statistical cluster is supposed to indicate an underlying cause of various symptoms. A major thrust of cognitive neuroscience is the clarification of structure – function of relationships in the human brain – understanding the relationship between the human brain structure and function is a major focus of cognitive neuroscience. The methods available to achieve this goal have undergone significant changes over the last 15 years; in particular, functional neuroimaging is rapidly replacing neuropsychological studies of people with brain lesions as the central method in this field, over the last several years, functional neuroimaging has risen in prominence relative to the lesion studies that formed the historical core of work in this field.

At the outset, it is important to bear in mind that regardless of the specific method used, inferences from impaired performances following brain damage always focus on the same conclusion; which is, an assumption regarding which structures are necessary to perform a given task. The study of cognitive and behavioural consequences of focal brain lesions has been an indispensable method for relating brain structure to function. Lesion studies rely on correlating damaged structure and abnormal function to determine crucial brain regions necessary for normal function. For this purpose it is necessary not only to detect lesions but also to accurately delineate their spatial extent. The lesion method was influential for our understanding of functions as diverse as memory, emotion, hemispheric specialization, language, vision and motor control. For example, recent neuropsychological research has refined our understanding of how emotions are processed, with damage to the amygdala resulting in difficulty in recognizing whether faces are expressing fear (Adolphs et al., 1995), and damage to the left insula and basal ganglia leading to a selective difficulty in identifying disgust (Calder et al., 2000). Work involving patients with brain damage has also shown that the posterior ventral cortex is involved in recognizing objects, and that the posterior dorsal regions are involved in integrating visual information with goal-directed motor responses e. g. grasping a door handle (Goodale & Milner, 1992).

Various types of disorders have been described by clinical neurologists and none has been more frequent and vivid than the syndrome of unilateral spatial neglect. Hemispatial neglect is a common disabling condition following unilateral brain damage, particularly of the right hemisphere. Although it can be caused by various different pathological conditions, it is most often observed after cerebral infarction or hemorrhage and affects up to two thirds of right hemisphere of stroke patients acutely (Stone et al., 1991; Bowen et al., 1999). Unilateral neglect is traditionally defined as a failure to report, respond to or orient towards stimuli in contralesional space (Driver & Mattingley, 1998; Halligan, Fink, Marshall, & Vallar, 2003; Heilman, Watson, & Valenstein, 1993). Perhaps a more appropriate description, especially for severe neglect patients, would be to suggest that the patient behaves as if one half – the contralesional side (the left side for patients with right brain damage) – of their world has simply ceased to exist, they attend instead to items towards the same side as their brain damage—their ipsilesional side. Their neglect may be so profound that they are unaware of large objects, or even people, in extrapersonal space. Neglect may also extend or be confined to personal space, with patients failing to acknowledge their own contralesional body parts in daily life (Bisiach et al., 1986; Zoccolotti & Judica, 1991; Beschin & Robertson, 1997).

The earliest descriptions of unilateral neglect that were able to localize the underlying lesion with any degree of certainty came from cases initially described by Paterson and Zangwill (1944). Identifying the neuroanatomical correlate of spatial neglect in humans is as challenging because human brain lesions vary tremendously in size and the neglect syndrome itself is multifaceted. While the Paterson and Zangwill (1944) case described a patient with a discrete lesion resulting from a penetrating head wound, the more common cause of neglect is a middle cerebral artery stroke causing widespread damage to the lateral cortical surface and underlying white matter that this artery subserves (Duvernoy, 1999).

Unilateral spatial neglect has been investigated in a systematic manner, by comparing performances of unselected groups of right and left brain-damaged patients, both of which were asked to perform tasks requiring an adequate exploration of space. However, the results of these studies have been somewhat varied, and there is still disagreement about both qualitative and incidence aspects of unilateral neglect in lateralized cerebral lesions. For example Battersby et al., (1956) found that lesions of the posterior areas of either hemisphere frequently produced unilateral neglect but Hecaen (1962), in his observation series of 59 patients with unilateral spatial neglect found only one case was suffering from a left hemispheric lesion, and emphasized the relationship between unilateral spatial neglect and lesions of the minor hemisphere. Also, Gainotti (1968) attempted to study the same problem by means of a battery of tests simple enough to be administered to all patients; his results showed that unilateral spatial neglect is not only significantly more frequent, but also definitely more severe in patients suffering from lesions of the right hemisphere.

Lesions of the right hemisphere are far more likely to lead to severe and enduring neglect than left hemisphere damage (Bowen et al., 1999; Stone et al., 1992), perhaps because of the specialization of the latter for language. Cortical damage involving the right inferior parietal lobe or nearby temporoparietal junction has classically been implicated in causing neglect (Vallar & Perani, 1986). It has become apparent, however, that the syndrome may also follow focal lesions of the inferior frontal lobe (Vallar, 2001; Husain & Kennard, 1997), although lesions confined to the frontal lobe may lead to a more transientneglect (Walker, 1998).

Recent studies making use of fMRI scans in neglect patients have suggested that the critical region of overlap in a series of neglect patients’ lesions is either in the superior temporal gyrus (Karnath et al., 2001; Karnath et al., 2004) or the temporoparietal junction (Mort et al., 2003).

Regarding localization of functions, research has demonstrated the variability (Kertesz, 1979) as well as the extent of the lesions that give rise to particular language disorders. Paul Broca (1861) suggested that lesions in the inferior frontal gyrus, now corresponding to Brodmann’s areas (BA) 44 and 45, were implicated in speech production disorders (Schiller, 1992). However, as advances in technology have made patients’ lesion information easier to obtain (e. g. CT and MRI scans), lesion–symptom relationships derived from the classical models of aphasia have proven to be less predictive than expected. In many instances, left frontal lesions do not result in Broca’s aphasia (Basso, Lecours, Moraschini, & Vanier, 1985; Willmes & Poeck, 1993). Moreover, fluency problems can be reliably associated to lesions outside of Broca’s area, including underlying white matter tracts and anterior insula (Bates et al., 2003; Damasio, 1992; Dronkers, 1996; Mohr et al., 1978). Conversely, lesions to Broca’s area can cause deficits in domains other than speech production, indeed even outside of language (Saygin, Wilson, Dronkers, & Bates, 2004).

Research has shown that a lesion restricted to Broca’s area gives rise to a transient impairment of language production and that the full complement of symptoms associated with Broca’s aphasia (articulation problem coupled with simplified sentence structure—the pattern known as “ agrammatism”) is the result of more extensive damage to the frontal cortex (Mohr et al., 1978). Evidence suggests, moreover, that the articulation problem present in Broca’s aphasia (“ apraxia of speech”) is associated with damage to a portion of the insula, a part of the cerebral cortex that is not visible from the brain’s surface because of the growth of other parts of the frontal lobe (Dronkers, 1996).

There are specialized mechanisms for the perception of speech, which consists of brief stimuli that change rapidly in wavelength composition. Isolating input to critical left temporal areas as a result of a left hemisphere lesion, or in some cases lesions in both hemispheres (the lesion on the right deprives the left hemisphere of transcallosal input), results in the disorder known as “ pure word deafness,” in which patients can hear but cannot understand speech; their native tongue, for example, sounds to them like a foreign language. Moreover, these patients have difficulty discriminating between speech sounds (between “ pa” and “ ba,” for example), although they have little or no difficulty producing speech and understanding written language (Saffarn et al., 1976). Virtually all aphasic patients suffer from verbal short-term limitations (as measured, for example, by asking them to repeat digit strings; their performance tends to be well below the normal span of about seven.). One particular group with left posterior parietal lesions (Shallice & vallar, 1990) suffers from short-term memory limitations but little else, and many of these patients have sentence comprehension deficits similar to those described in Broca’s aphasics (Saffaran & Martin. 1990).

It is interesting to note that Broca patients also have difficulty with certain grammaticality judgment tasks, in particular, those that entail linking particular word identities (and/or their meanings) to particular locations in the sentence. Thus, they proved to be insensitive to infractions involving reflexive sentences (e. g., “ The woman looked at himself in the mirror”) in which the gender of the pronoun conflicts with that of the noun to which it refers (Linebarger et al., 1983; Linebarger, 1995). This further suggests that the capacity to integrate the various types of information required for the understanding of sentences is limited in these patients. It may also be the case that frontal activation is critical for sentence production. One view of the sentence production deficit in Broca’s aphasics is that it reflects a timing problem in which lexical items are retrieved too slowly to integrate with sentence structure (Kolk, 1995). Lesions that affect anterior inferior regions of the left temporal lobe are known to result in semantic deficits in which patients have difficulty finding words and understanding them and often exhibit impairments with pictured materials as well (Howard & Patterson, 1992). Studying aphasia and its associated lesions in the late 19th century led to many insights about the neural organization of language functions and many of these insights have been confirmed and elaborated in more recent studies using advanced imaging to localize areas of dysfunctional brain tissue associated with particular language deficits or using functional imaging to identify areas of the brain that were activated during a particular language task in normal controls (Wise, 2003; Bookheimer, 2002) or in recovering aphasic Individuals (Price & Crinion, 2005).

Technological advances in recent years (e. g. functional imaging techniques) have allowed neuroscientists to measure and localize brain activity in healthy individuals. This has fueled the zeitgeist that the lesion method is an inferior and conceivably antiquated technique. Nonetheless, while the lesion method has notable weaknesses, it can be argued that it supplements the newer methods. Additionally, recent developments deals with many of the criticisms of the lesion method. Patients with brain lesions provide a unique window into brain function, and this methodology will fill an important niche in the growing resource of tools that constantly become available to neuroscientists and neuropsychologists for future research. Nevertheless, it is beneficial to consider whether new technologies can be used to optimize the lesion method. Whilst, some of the lesion method’s limitations are intrinsic to the technique, other weakness can be addressed by recent technical innovations. The lesion method has much to offer, despite its limitations; new techniques for imaging the brain and analyzing lesion data have the potential to improve the lesion method. Still, the strength and weaknesses of the lesion method and other imaging techniques such as fMRI are complementary, as some brain functions might be difficult to determine using the lesion method alone or functional neuroimaging alone, but can be successfully undertaken with a combination of these techniques (Price & Friston, 2002).