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Adapting the concepts of brain and cognitive reserve to post-stroke cognitive deficits: Implications for understanding neglect

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ABSTRACT

Advanced lesion mapping and connectivity analyses are currently the main tools used to understand the mechanisms underlying post-stroke cognitive deficits. However, the factors contributing to pre-stroke architecture of cognitive networks are often ignored, even though they reportedly play a decisive role in the manifestation of cognitive impairment in neurodegeneration. The present review on post-stroke cognitive deficits therefore adopts the concept of brain and cognitive reserve, which was originally developed to account for the individual differences in the course of aging and neurodegenerative diseases. By focusing on spatial neglect, a typical network disorder, it is discussed how individual susceptibility to stroke lesion might explain the reported discrepancies in lesion anatomy, non-spatial deficits and recovery courses. A detailed analysis of the literature reveals that premorbid brain (age, brain atrophy, previous strokes, leukoaraiosis, genetic factors, etc.) and cognitive reserve (IQ, life experience, education, occupation, premorbid cognitive impairment, etc.) greatly impact the brain's capacity for compensation. Furthermore, the interaction between pre-stroke brain/cognitive reserve and the degree of stroke-induced system impairment (e.g., hypoperfusion, lesion load) determines both the extent of neglect symptoms variability and the course of recovery. Premorbid brain/cognitive reserves should thus be considered to: (i) understand the mechanisms of post-stroke cognitive disorders and sufficiently explain their inter-individual variability; (ii) provide a prognosis for cognitive recovery and hence post-stroke dependency; (iii) identify individual targets for cognitive rehabilitation: in the case of reduced brain/cognitive reserve, neglect might occur even with a confined lesion, and non-spatial training of general attentional capacity should represent the main therapeutic target also for treatment of neglect; this might be true also for non-cognitive domains, e.g., motor deficit. This alternative view of how neglect and other cognitive deficits occur and recover promotes discussion about plasticity and recovery to a general rather than a single stroke-based domain, providing more efficiency in recovery research.

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1. Introduction

Stroke continues to be one of the main causes of disability in humans. While the neuroimaging era is associated with a plethora of new data, the technique itself has mainly been used to study isolated brain functions, or is often only applied in one imaging mode, thus restricting our understanding of the causes and consequences of a particular functional deficit. Furthermore, while stroke often leads to cognitive decline or even manifestation of dementia, knowledge about stroke recovery and cognitive reserve are poorly combined and integrated, hindering further development of rehabilitation approaches. As life expectancy increases, cognitive networks are more likely to be compromised before stroke; therefore, clinicians and researchers will be increasingly confronted with more stroke cases that are accompanied by cognitive decline. Until now, neurodegenerative diseases and post-stroke cognitive deficits have been researched and discussed in different frameworks, and while the underlying pathologies are likely to differ, the biological and cognitive mechanisms of brain adaptation to impairment might not. Using spatial neglect as an example, the present review aims to highlight the individual susceptibility to stroke damage by presenting post-stroke cognitive deficits and establish a concept for tailored rehabilitation approaches by discussing (i) the role of premorbid individual characteristics for emerging of post-stroke cognitive deficits, and (ii) the factors influencing neural compensation and hence recovery on the network level. Although this review focuses on the role of brain and cognitive reserve, it is likely one of many factors that contribute to the clinically observed heterogeneity in the recovery from post-stroke deficits.

2. Spatial neglect and its controversial anatomy

Spatial neglect is the common term for deficits in the ability to perceive relevant contralesional stimuli, which cannot be explained by a primary sensory disturbance (Brain, 1941; Critchley, 1949; Heilman, Watson, & Valenstein, 2003). It is evoked by a dysfunction of a large-scale attention network (Mesulam, 1990), rather than by structural damage of specific brain regions (Corbetta & Shulman, 2011). This makes neglect an appropriate model for understanding the emergence and recovery patterns of post-stroke cognitive deficits. Neglect occurs more often and more severely after right hemisphere stroke (Becker & Karnath, 2007; Stone, Halligan, & Greenwood, 1993), and can be observed in any sensory or motor modality, or in distinct spaces related to patient's body or object coordinate (e.g., in the peri- or personal space; ego- or allocentric), though the most clinically-relevant and researched form is visual neglect (Vallar & Bolognini, 2014). The variability of neglect symptoms across patients emphasizes the complexity of the system used to code spatial information, as well as the heterogeneous entity of neglect syndrome (Malhotra & Russell, 2015; Vallar & Bolognini, 2014). This is also reflected in several theories of neglect (Bartolomeo, 2014; Karnath, 2015; Vallar & Bolognini, 2014), which are not intended to be

discussed here, but which, however, hardly explain recovery process from neglect and its variability.

Data on the neuroanatomy of neglect are controversial (Malhotra & Russell, 2015). Structural lesions in the inferior parietal lobe (Mort et al., 2003; Vallar & Perani, 1986), the anterior cingulate (Leibovitch et al., 1998), the temporo-parietal junction or superior temporal cortex (Karnath, Ferber, & Himmelbach, 2001), or the inferior frontal cortex (Husain & Kennard, 1996) in the right hemisphere can each evoke neglect. Neglect is also considered to be evoked by damage to either the long fronto-parietal pathways (Bartolomeo, Thiebaut de Schotten, & Doricchi, 2007; Doricchi, Thiebaut de Schotten, Tomaiuolo, & Bartolomeo, 2008; Shinoura et al., 2009; Thiebaut de Schotten et al., 2011, 2014; Umarova et al., 2010; Urbanski et al., 2008) or the deep temporo-parietal white matter (Samuelsson, Jensen, Ekholm, Naver, & Blomstrand, 1997) connecting the parahippocampal and angular gyri (Bird et al., 2006). The controversies in anatomical correlates of spatial neglect have been linked to the heterogeneity of the behavioral symptoms. Distinct lesions might affect different domains of spatial cognition and can lead to a heterogeneous clinical profile (Azouvi et al., 2002; Verdon, Schwartz, Lovblad, Hauert, & Vuilleumier, 2010), while some clinical manifestations of neglect might not be detected due to insufficient testing (Bowen, McKenna, & Tallis, 1999; Saj, Verdon, Vocat, & Vuilleumier, 2012; Stone, Halligan, Marshall, & Greenwood, 1998). Another reason for the discrepancies in lesion anatomy might be the time point at which neglect is assessed: Since neglect might completely recover soon after stroke (Cassidy, Lewis, & Gray, 1998; Khurshid et al., 2012; Stone, Patel, Greenwood, & Halligan, 1992; Umarova et al., 2016), lesion anatomy during distinct stroke phases can differ significantly. The data, however, remain to be inconclusive even taking into account the heterogeneity of neglect. For example, egocentric neglect is reportedly associated with perisylvian lesions, while allocentric deficit is linked to more posterior lesions of the angular, middle temporal, and middle occipital gyri (Chechlacz, Rotshtein, & Humphreys, 2012). At the same time, hypoperfusion of temporal regions has been reported to lead to allo- and of parietal lesions to egocentric neglect (Hillis et al., 2005; Shirani et al., 2009).

Spatial extinction represents another type of spatial deficit, which is characterized by the failure to respond to a contralesional stimulus presented simultaneously with an ipsilesional one. Whether extinction is a 'mild' form of neglect or another kind of attentional deficit is still an open question. Some data have shown that these syndromes are dissociable (Hillis et al., 2006; Vallar, Rusconi, Bignamini, Geminiani, & Perani, 1994), also due to the distinct functional state of the attention network (Umarova et al., 2011). On the other hand, extinction has also been described as part of the neglect syndrome, especially when the lesions are clustered in the inferior parietal lobe (Posner et al., 1984; Vallar et al., 1994; Rees et al., 2000; Vuilleumier and Rafal, 2000). Anatomically, extinction can be caused by hypoperfusion and subsequent lesion to the right temporo-parietal junction and temporal cortex (Chechlacz et al., 2013; Karnath, Himmelbach, & Küker, 2003; Ticini, de Haan, Klose, Nagele, & Karnath, 2010), as well as to the occipital (Hillis et al., 2006) and inferior parietal cortex (Vossel et al., 2011). Though a few studies were able to

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