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Update article

Attention and spatial cognition: Neural and anatomical substrates of visual neglect

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ABSTRACT

Unilateral spatial neglect (USN) is a neurological disorder often observed following damage to the right cerebral hemisphere. Patients with USN are no longer able to take into account stimuli presented on the left side of space. In this article, we will discuss the neuroanatomical correlates that underlie visuospatial attention and can cause USN, an area of growing research interest in the past 20 years. This syndrome has often been related to cortical damage, notably in the inferior parietal lobule. Other data have also implicated lesions in the inferior frontal gyrus or the superior temporal gyrus. In this article, we will highlight the relevance of viewing USN as a disconnection syndrome of interconnected cerebral areas, as opposed to a focal cortical syndrome. We will review data that provide evidence of intrahemispheric disconnection, in particular within the right hemisphere's frontoparietal networks connected by the superior longitudinal fasciculus. Recent findings suggest that interhemispheric disconnection could also contribute to the manifestations of USN. Most importantly, interhemispheric disconnection might be a predictive factor for the chronicity of this disorder. This hypothesis implies that the left hemisphere by itself is not able to compensate for the patients' deficits. Recovery requires the ability to exchange information between the two hemispheres, particularly in the posterior parietal and occipital regions.

It is now widely accepted that visuospatial functions are not distributed symmetrically between the hemispheres. This was initially observed in brain-damaged patients in whom spatial deficits were most often associated with damage to the right hemisphere as opposed to the left hemisphere 1941 [1]. In 1962, Hécaen [2] investigated the occurrence of spatial deficits in a large group of patients with unilateral post-Rolandic lesions. Spatial neglect, along with the inability to orient themselves on a map, loss of topographic memory, constructional apraxia and also dressing apraxia were found more often in patients with right hemisphere lesions. Since then, advances in brain imaging methods, in particular functional MRI, diffusion MRI, and transcranial and intracerebral magnetic stimulation have refined our understanding of anatomo-clinical relationships. In this article, we will review the neuroanatomical correlates of visuospatial attention and spatial neglect.

1. Anatomical and functional models of visuospatial attention

Neuroimaging data in healthy subjects have inspired an anatomo-functional model of visuospatial attention [3]. The model is composed of a dorsal attentional network (DAN), made up of the intraparietal sulcus, superior parietal lobule, precuneus, and frontal eye field, which shows increased BOLD signal when subjects voluntarily direct their attention towards a visual target. A ventral attentional network (VAN), including the temporoparietal junction and the middle and inferior frontal gyri, has an increased BOLD signal when the subject attempts to process a target in an unexpected location. These regions underpin non-spatial attentional processes such as waking, and also the reorientation of attention towards new or important unexpected events [4]. In healthy subjects, the two networks interact continuously. Importantly, the DAN is represented bilaterally on the two hemispheres, while the VAN is lateralised to the right hemisphere.

These two frontoparietal networks are anatomically supported by white matter tracts that form the superior longitudinal fasciculus (SLF). Three SLF branches have been identified in the monkey [5]. Recently, the use of tractography (reconstruction of

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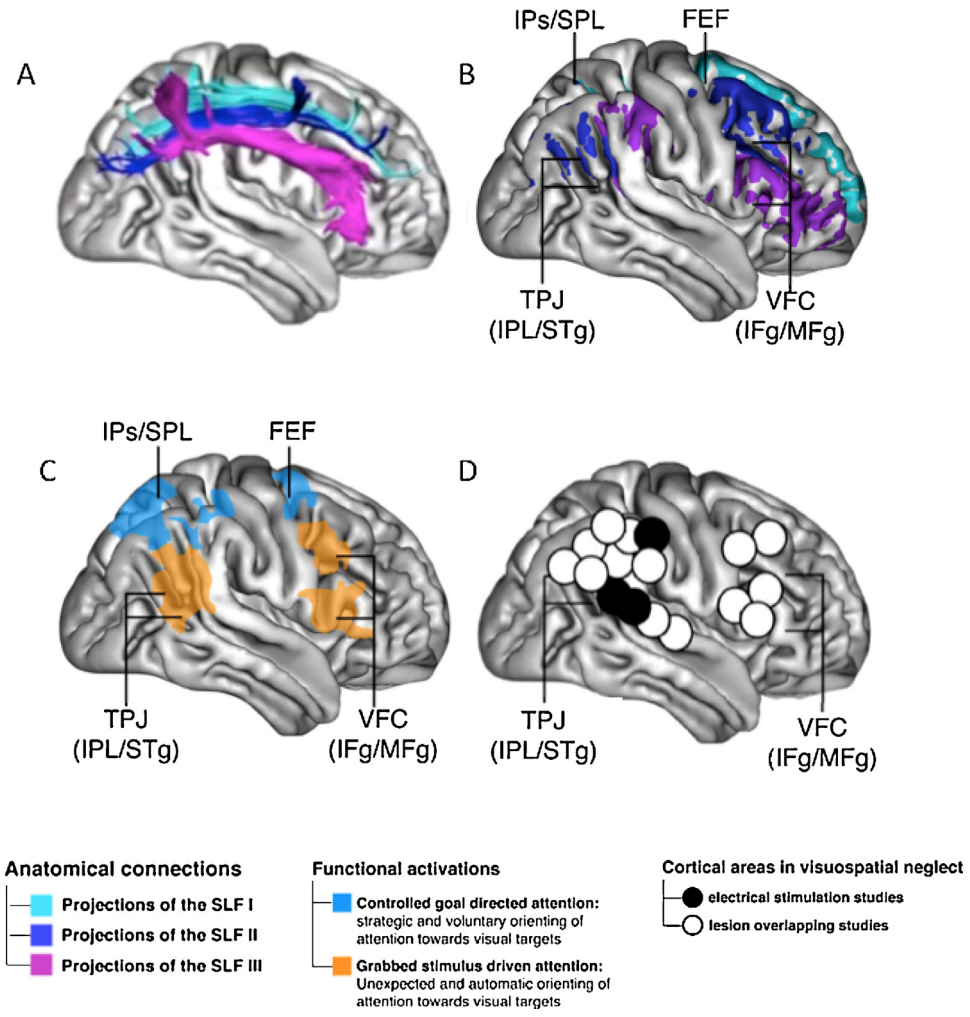

Fig. 1. Visualisation of the frontoparietal network supporting visuospatial attention processes: (A) The three branches of the superior longitudinal fasciculus as identified by diffusion-based tractography; (B) Its cortical projections as identified by tractography; (C) Cortical areas activated in functional imagery during orienting of visuospatial attention [3]; (D) Cortical areas related to the emergence of visuospatial neglect; (E) IPs: intraparietal sulcus; SPL: superior parietal lobule, FEF: frontal eye field, TPJ: temporoparietal junction, IPL: inferior parietal lobule, STg: superior temporal gyrus, VCF: ventral frontal cortex, IFg: inferior frontal gyrus, MFg: middle frontal gyrus. Adaptation of figure originally published in Nature Neuroscience 14(10);1245–6:2011 "A lateralised brain network for visuospatial attention", by M. Thiebaut de Schotten, F. Dell'Acqua, S.J. Forkel, A. Simmons, F. Vergani, D.G. Murphy, M. Catani. © 2011, with permission from Nature Publishing Group.

white matter tracts through diffusion imaging) has identified a similar architecture in humans [6] (Fig. 1A). The most dorsal branch (SLF I) originates at the precuneus and the superior parietal lobule (Brodmann areas, BA 5 and 7) and projects towards the superior frontal and anterior cingulate gyri (BA 8, 9, 32). The intermediate branch (SLF II) originates at the anterior intraparietal sulcus and the angular gyrus (BA 39 and 40), and joins the posterior portions of the superior and middle frontal gyri (BA 8 and 9). The most ventral branch (SLF III) originates at the temporoparietal junction (BA 40) and ends at the inferior frontal gyrus (BA 44, 45 and 47).

The cortical projections of these three branches overlap with nodes of the VAN and DAN [3] (Fig. 1B and C). The SLF I connects brain regions within the DAN. The SLF II connects parietal regions of the VAN with the DAN's prefrontal regions, allowing these two networks to communicate. The SLF III connects regions within the VAN network. In most subjects, these fibres are arranged in an asymmetric anatomical gradient, consistent with the functional asymmetry demonstrated in Corbetta and Shulman's model [3]: the SLF III is larger on the right than the left, the SLF I is symmetric, and the SLF II tends to be larger in the right hemisphere [6].

2. Unilateral spatial neglect: from a focal cortical syndrome to a disconnection syndrome

Unilateral spatial neglect (USN) is defined as "a failure to report, respond, or orient to stimuli that are presented contralateral to a brain lesion when this failure is not due to elementary sensory or motor disorders" [7]. Signs of neglect can occasionally develop following a left hemispheric lesion, but are far more common, severe and long-lasting after a right hemispheric lesion, causing patients' inability to consider events that occur in the left side of body or external space (contralateral to lesion). It is now generally accepted that 85% of patients with right hemispheric lesions have signs of neglect in the subacute phase, with moderate to severe signs in 36.2% of cases [8].

2.1. Lesion sites

Ischaemic or haemorrhagic strokes in the right perisylvian regions appear to be the main trigger for the development of neglect. Nevertheless, neglect can also occur following a stroke in the territories of the anterior cerebral artery [9] or of the posterior

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