SPECIAL SECTION

DISORDERS OF VISUAL ATTENTION AND THE POSTERIOR PARIETAL CORTEX

Parashkev Nachev[1] **and Masud Husain**[1,2]

([1]Division of Neuroscience and Psychological Medicine, Imperial College London, London, UK; [2]Institute of Cognitive Neuroscience, University College London, London, UK)

ABSTRACT

Traditionally, both the monkey and human posterior parietal cortex (PPC) have been considered to have a privileged role in spatial perception or action. Lesions to this region of the human brain, particularly of the right hemisphere, undoubtedly lead to spatially lateralised deficits such as visual extinction or neglect. However, although studies in monkeys have revealed much about the spatial functions of the parietal lobe, the monkey PPC may not be a good model system with which to understand fully the disorders of attention that follow damage to the human parietal cortex. Several lines of evidence, from functional imaging as well as investigations of patients with parietal damage, demonstrate that parts of the human inferior parietal lobe (IPL) have non-spatial functions. Here, we argue that it is important to distinguish spatially lateralised from spatial deficits. Both spatial and non-spatial impairments might, in principle, contribute to a spatially lateralised behavioural syndrome such as neglect. In this review, we discuss the evidence for such a proposal and suggest that a better understanding of human parietal syndromes may emerge from considering both the spatial and non-spatial functions of this region.

Key words: unilateral neglect, hemispatial neglect, inattention, spatial working memory, sustained attention

INTRODUCTION

The posterior parietal cortex (PPC) has traditionally been considered to have a special role in spatial functions. In monkeys, the PPC is viewed as the target of the "dorsal" visual stream of cortical pathways which originates in primary visual cortex and projects to the parietal lobe. This dorsal system is envisaged to have a critical role in visuospatial perception (Ungerleider and Mishkin, 1982) or visually-guided action (Milner and Goodale, 1995). Both electrophysiological recording studies from awake behaving monkeys (Colby and Goldberg, 1999; Andersen and Buneo, 2002) and investigations of the effects of lesions to the PPC (Faugier-Grimaud et al., 1985) leave little doubt that the representation of space – for perception or action – is an important function of this region.

In humans, too, lesions of the PPC have classically given rise to several different types of disorder that are classically associated with spatial deficits. For example, patients with inferior parietal lobe (IPL) damage (see Figure 1), particularly of the right hemisphere, often encounter difficulty in appreciating the spatial relationships of items in the visual scene (Paterson and Zangwill, 1944). When asked to copy complex drawings or models, they appear to perceive local details but cannot synthesize them correctly to produce a veridical copy, thereby demonstrating constructional apraxia. Other parietal patients, particularly those with superior parietal lobe (SPL) or intraparietal sulcus (IPS) involvement, demonstrate disorders of visually-guided movement, often misreaching to targets in the visual periphery, so-called optic ataxia (Perenin and Vighetto, 1988). More recently, functional imaging has also appeared to support the view that in humans, just as in monkeys, the PPC has a privileged role in visuospatial functions. Directing visual attention to points in space (without making eye movements) has consistently been associated with SPL and/or IPS activation

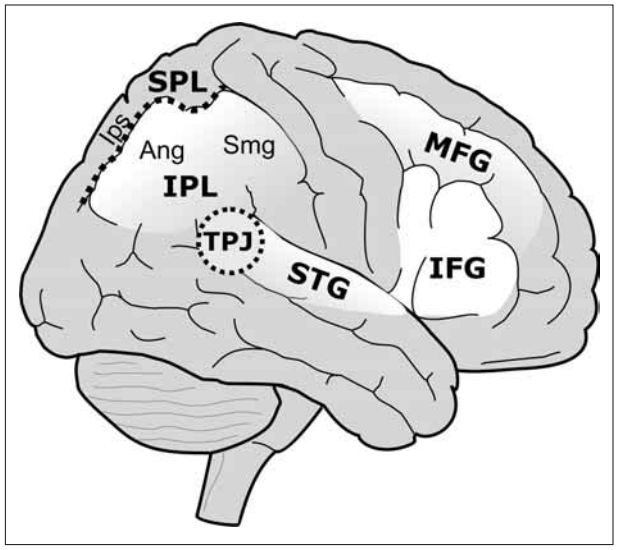

Fig. 1 – *Right hemisphere regions of the human brain. The inferior parietal lobe (IPL) consists of the angular gyrus (ANG) and the supramarginal gyrus (SMG) and a border zone with temporal lobe which is termed the temporo-parietal junction (TPJ). The superior parietal lobe (SPL) is separated from the IPL by the intraparietal sulcus (IPS). Also marked are the middle frontal gyrus (MFG) and the inferior frontal gyrus (IFG), as well as the superior temporal gyrus (STG) region implicated by Karnath et al. (2001) in neglect.*

(Corbetta and Shulman, 2002; Vandenberghe et al., 2001), while directing eye or limb movements to spatial targets activates similar or nearby dorsal parietal regions in the healthy human brain (Luna et al., 1998; Connolly et al., 2003).

Understandably, this large body of experimental data from studies in humans and monkeys has often been used as the framework with which to consider the disorders of "attention" associated with parietal damage in humans – neglect and extinction (Pouget and Driver, 2000). In this review, we consider the evidence for such a proposal and argue that spatial functions may not be sufficient to capture the role of the human PPC or explain the deficits that follow damage to this region.

Disorders of "Attention"

Perhaps the most devastating deficits of visual perception and action are seen in the thankfully rare cases of Balint's syndrome observed after bilateral posterior damage, including involvement of the IPL (Balint, 1907; Husain and Stein, 1988; Rafal, 2001). These patients suffer from profound visuospatial disorientation, bumping into objects in front of them, groping effortfully to reach items and demonstrating impairments in directing gaze accurately to visual targets (Holmes, 1918). But, in addition to these spatial deficits, they appear to suffer a non-spatial impairment, demonstrating a severe limit in the capacity to process visual information, regardless of the spatial location of objects. Indeed, they may report seeing only one item at a time, even when line drawings are overlapped in one spatial location – so-called simultanagnosia (Balint, 1907; Luria, 1959; Humphreys et al., 1994). Finally, patients with Balint's syndrome appear to have a severely constricted field of vision, neglecting items on both sides of space. Such a bias towards local details may be a prominent feature also of visual perception in patients with unilateral neglect who have damage only to the right hemisphere (Rafal, 1994; Robertson et al., 1988).

The neglect syndrome is one of the commonest of disorders following unilateral damage to the IPL, particularly of the right hemisphere, in humans (Vallar and Perani, 1986). This classical localisation has recently been contested by Karnath et al. (2001) who proposed that the syndrome was due instead to damage of the mid-superior temporal gyrus (STG; Karnath et al., 2001, 2004). However, the results of subsequent reports have demonstrated that lesions of the angular gyrus of the IPL (Mort et al., 2003), or parietal white matter (Doricchi and Tomaiuolo, 2003), are critically associated with the condition.

Patients with neglect often fail to be aware of objects in contralesional space (left side for right hemisphere patients), even when given unlimited time to explore their environment (Mesulam, 1999; Bisiach and Vallar, 2000; Heilman and Watson, 2001; Parton et al., 2004). Neglect may occur in up to two-thirds of right hemisphere stroke patients acutely (Bowen et al., 1999). If it persists it is associated with a poor prognosis for functional independence (Denes et al., 1982; Jehkonen et al., 2000; Cherney et al., 2001).

Importantly, neglect need not be associated with any primary deficit in sensation (such as an absolute loss of vision in a sector of the visual field) or movement (such as hemiparesis). Moreover, patients can be cued to direct their gaze towards items in their neglected field, although they soon revert to their ipsilesional bias (right side for right hemipshere patients) unless reminded not to do so. For these reasons, neglect has come to be considered a disorder of "attention", specifically of spatial attention, with patients demonstrating a pathological bias for ipsilesional items at the expense of contralesional ones (Driver and Vuilleumier, 2001).

Patients with extinction also appear to have a disorder of spatial attention, although their impairment is evident when only briefly presented stimuli are used. These patients may fail to be aware of a transient contralesional stimulus when there is a simultaneous ipsilesional one (Driver et al., 1997). Thus the contralesional object is said to be "extinguished" from awareness when there is a competing ipsilesional one. Like neglect, extinction may occur in the absence of pure sensory or motor loss. Interestingly, some patients show both extinction and neglect, and several investigators have argued that extinction-like phenomena play a key part in the neglect syndrome.

Both neglect and extinction are clearly characterised by spatially lateralised deficits, with patients being worse in detecting contralesional items compared to ipsilesional ones. Given the large body of data of electrophysiological demonstrating the association of monkey PPC with spatial functions, it would seem natural to consider that there are strong homologies between species. Moreover, it might seem to follow that the spatially lateralised attention deficits that occur in humans after damage to the PPC are explained by the loss of neurones performing spatial functions homologous to those in monkey PPC.

Monkey and Human Parietal Cortex are not the Same

But there are several reasons why we should question such an assumption and entertain, instead, the possibility that human parietal cortex may be different from monkey PPC. First, although unilateral lesions of the monkey PPC lead to extinction, they do not lead to severe neglect (Milner, 1997).