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Complexity vs. unity in unilateral spatial neglect

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ABSTRACT

Unilateral spatial neglect constitutes a heterogeneous syndrome characterized by two main entangled components: a contralesional bias of spatial attention orientation; and impaired building and/or exploration of mental representations of space. These two components are present in different subtypes of unilateral spatial neglect (visual, auditory, somatosensory, motor, allocentric, egocentric, personal, representational and productive manifestations). Detailed anatomical and clinical analyses of these conditions and their underlying disorders show the complexity of spatial cognitive deficits and the difficulty of proposing just one explanation. This complexity is in contrast, however, to the widely acknowledged effectiveness of rehabilitation of the various symptoms and subtypes of unilateral spatial neglect, exemplified in the case of prism adaptation. These common effects are reflections of the unity of the physiotherapeutic mechanisms behind the higher brain functions related to multisensory integration and spatial representations, whereas the paradoxical aspects of unilateral spatial neglect emphasize the need for a greater understanding of spatial cognitive disorders.

Unilateral spatial neglect is an intriguing spatial cognitive deficit characterized by a behavioral bias directed towards the side of the lesion. Unlike behavioral biases of vestibular or cerebellar origin, or caused by optic ataxia, this bias is associated with a lack of awareness of the contralateral space, thereby involving the absence of sensory information-processing on the neglected side, a reduction of movements performed towards that side and hyperattention of the ispsilesional side (Fig. 1). The first component refers to a contralesional bias of spatial attention orientation, while the second one corresponds to impaired building and/or exploration of mental representations of space [1–3]. These two components constitute the core symptoms of unilateral spatial neglect. They can be objectified by the mere observation of the patient's default tonic posture, with ocular and cephalic deviations towards the lesion side, and/or by simple paper-and-pencil tests revealing deviations to the right (for example, bisecting horizontal lines to the right of the objective

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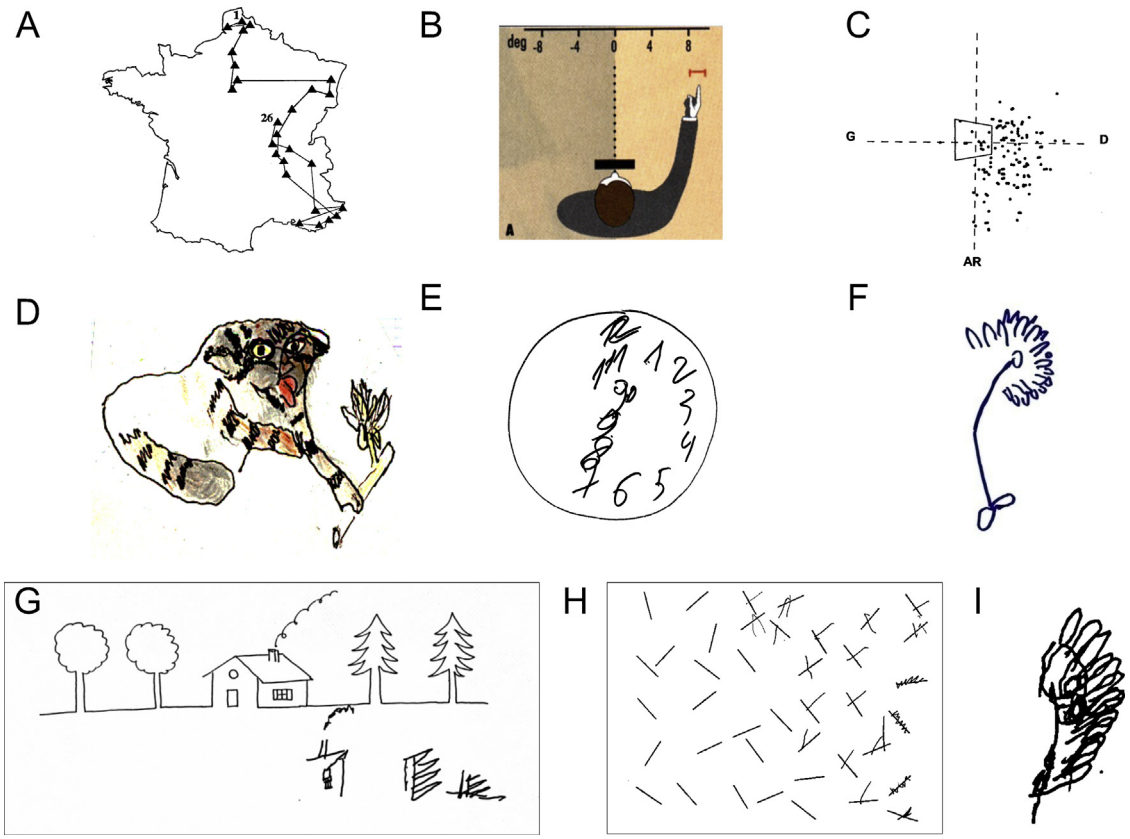

Fig. 1 – Deficits of unilateral spatial neglect include: ipsilesional bias, which can be evidenced by (A) a mental imagery task (representational neglect), (B) a shift in straight-ahead pointing movements in the dark and (C) displaced weight-bearing, leading to postural asymmetry; allocentric neglect, which can be revealed by (D–F) making drawings from memory and (G) by copying; egocentric neglect, which is also shown by (G) drawing and copying and (H) by cancellation task; and ispilesional graphic perseverations, as shown by (H) cancellation tasks and (I) making drawings from memory.

midline). These two components are present in many clinical subtypes:

- visual neglect;
- auditory neglect;
- extrapersonal neglect;
- personal neglect;
- egocentric neglect;
- allocentric neglect;
- representational neglect [4–8].

However, unilateral spatial neglect is more frequent, severe and long-lasting following damage to the right hemisphere in right-handed subjects because of the predominance of the right hemisphere in spatial attention and spatial representations [9]. Nevertheless, the semiological and anatomical complexity of neglect contrasts with the global efficacy of therapeutic interventions.

1. Anatomo-clinical pictures and their underlying deficits

Visual neglect is the most frequently described subtype of neglect (Table 1), and its manifestations reflect the two components of the syndrome: the default attention orientation towards the ispilesional side; and an incapacity to detect or respond to stimuli presented to the contralesional visual field [2]. Visual neglect is distinct from hemianopia, although the deficit can mimic pseudohemianopia [9]. Anatomical lesion correlates of visual neglect are the temporoparietal junction (TPJ), including the inferior parietal lobule (IPL; Brodmann area [BA] 39 or supramarginal gyrus [SMG] and BA40 or angular gyrus [AG]) [10–14], and superior temporal gyrus (STG) [15,16], as well as the premotor cortex (BA6, BA8 and BA44) [5,17], basal ganglia and thalamus [17,18]. Chronic visual neglect is caused by a disconnect in the intrahemispheric white-matter pathways linking parietal areas to frontal ones involved in spatial selective attention [19]: the second and third branches of the superior longitudinal fasciculus (SLF II and III); the arcuate fasciculus (AF) [20–24]; the inferior longitudinal fasciculus (ILF) [20]; and the inferior occipito-frontal fasciculus (IFOF) [25]. Furthermore, damage to the interhemispheric connections, especially in the posterior part of the corpus callosum (forceps major), also contributes to chronicity [26–29].

The two components of unilateral neglect syndrome are also present in auditory neglect, defined as an inattention to sounds or verbal stimuli stemming from the hemispace contralateral to the lesion. Patients with auditory neglect