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Spatial cognition in Parkinson's disease and neurodegenerative dementias

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Abstract Spatial awareness is dependent on the cortical areas of the temporal–parietal junction and on a larger parietal–frontal network communicating via the superior occipito–frontal fasciculus. The hippocampal formation may enhance the spatial information processed within short term memory with cognitive information through the neocortical–hippocampal loop of the newly acquired spatial information. Several aspects of spatial cognitive performance have been found to be significantly and positively correlated with striatal DA (D2 and to a lesser extent D1) receptor availability, where sub-clinical variations in striatal DA (D2) receptors may have measurable behavioural consequences. Visual speed of processing and attention are impaired in early phases of Parkinson's disease (PD), where visual attention impairments may underlie some of the cognitive deficits observed in this disease. Visual attentional impairment in PD may involve dopaminergic and cholinergic mechanisms at cortical and subcortical levels. Functional neuroimaging in PD shows hypoperfusion in the occipital and parietal regions, even in patients without dementia. The degenerative process in PD begins in the brainstem and ascends to involve the anteromedial temporal mesocortex and high-order sensory association and prefrontal areas. The resulting decline in visual function affects basic sensory functions, visual perception, and cognition and is present in mild to moderate PD, owing to degeneration of multiple neuronal systems at retinal, subcortical, and cortical levels. Visual dysfunction contributes to parkinsonian disability through its influences on cognition and locomotion. Alzheimer's disease (AD) presents with progressive memory loss and cognitive impairments. More than one-third of patients with AD have disabling visuospatial disorientation that interferes with safe driving and independent living. Hippocampal involvement in patients with early stages of AD could impair landmark orientation by undermining the role of hippocampal place neurons in maintaining cognitive maps. This would force patients with AD to rely on path integration that may be maintained by parietal cortical integration of self movement cues. Involvement of parietal cortex in the later stages of AD could impair path integration, leaving patients with AD without a viable orientation mechanism and causing spatial disorientation. Both age and AD affect spatial orientation. Elderly nondemented subjects with isolated spatial deficits may have a spatial variant of mild cognitive impairment (MCI). Such monosymptomatic syndromes may identify patients who are at greater risk for developing AD and who may be good candidates for focused monitoring and possible treatment. The lack of a relationship to memory impairment suggests that this is not the critical factor in spatial disorientation and is consistent with the relationship between impaired visual motion processing and ambulatory and vehicular navigation in AD, possibly reflecting posterior parietal cortical dysfunction in integrating multisensory cues about selfmovement. In dementia with Lewy bodies (DLB) spatial function is disproportionately severe early in the course of the disease compared to AD. The distinctive patterns of neuropsychological impairment in these diseases probably represent a different distribution of pathological changes. The neuropathological substrate of AD affects preferentially the entorhinal cortex and the neocortical association areas, which explains the dysfunction with encoding and storing information; in DLB subcortical nucleus together with frontal, temporal, and parietal lobes are mostly affected by neuronal loss and Lewy body deposition, justifying the predominantly attentional, executive, and visuospatial dysfunction found in this disease.

Keywords Spatial cognition · Alzheimer's disease · Parkinson's disease · Dementia with Lewy bodies.

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