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THE PATHOGENESIS OF SPATIAL NEGLECT SYNDROME

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Unilateral spatial neglect (USN), also referred to as hemispathic neglect or spatial neglect syndrome, is a common and incapacitating neuropsychological disorder that occurs after unilateral brain injury, most frequently right-hemisphere stroke. It is characterised by the inability to pay attention to, report, or react to stimuli in the contralateral hemisphere. The prevalence in the acute post-stroke phase is generally between 29% and 38%; however, rates are much greater (up to 38–50%) following injury to the right hemisphere than following damage to the left.

The right hemisphere is in charge of paying attention to both the left and right visual fields, whereas the left hemisphere can only govern the right, which explains the more severe left-hemisphere neglect syndrome that occurs after right-hemisphere loss. Neglect is less noticeable when the right hemisphere has a compensatory role due to injury. Perceptual-attentive, motor-intentional, egocentric, allocentric, personal, peri-personal, and extra-personal forms are all included in this complex syndrome, even when core sensory and motor capabilities are preserved.

One of the most obvious disruptions between higher-order spatial perception and surviving primary sensory processing is anosognosia, which frequently coexists with it. Patients may neglect food on the left side of the plate while denying any impairment (anosognosia), come across objects on the ignored side, or leave the left side of their bodies nude. Even though injuries in the right posterior parietal region have typically been associated with USN syndrome, modern neuroscience shows that the attention, motor, and arousal systems are all severely compromised. Its acute impairment is only one aspect of its therapeutic value; it is also associated with increased carer burden, longer hospital stays, and slower rehabilitation.

The temporoparietal junction supplied by the right middle cerebral artery, the right inferior parietal lobe, or the posterior parietal cortex are all affected by the classic lesion. Neglect syndrome, however, can also result from injury to the frontal, superior temporal, subcortical (thalamus, basal ganglia), or white matter. In particular, injury to the right hemisphere releases the left hemisphere from reciprocal inhibition, resulting in an interhemispheric imbalance that causes a rightward attention bias and contralateral hypokinesia.

While the bilateral dorsal frontoparietal network (intrauterine sulcus, frontal visual fields) governs spatial mapping and target selection, the right ventral network (temporoparietal junction, inferior frontal gyrus) recognises significant events and reorients attention. As was previously mentioned, whereas left-hemisphere strokes never cause severe neglect syndrome, right-hemisphere strokes frequently impact both sides. Neglect syndrome after subcortical strokes is explained by damage to the superior longitudinal fasciculus, inferior occipitofrontal fasciculus, and other long-distance fibres, which results in separation of cortical nodes due to remote cortical hypoperfusion or diaschisis. Functional outcomes influence day-to-day activities and may have an impact on quality of life. The difficulty in recognising and characterising hemineglect symptoms can account for the variable incidence of hemineglect patients, which ranges from 8% to 90%.

A multimodal battery of bedside and environmental testing is used to make the diagnosis, even though treatment options include behavioural therapy (such as prism adaptation and visual scanning), noninvasive brain stimulation, novel neuro technologies, and interdisciplinary rehabilitation. The majority of patients recover on their own in three to six months, despite the fact that up to one-third of patients experience persistent impairment, which denotes a decreased degree of functional independence. This review focuses on the pathophysiology of USN syndrome and includes details on the damaged brain regions, symptoms, and treatments.