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Alzheimer's disease (AD) and normal pressure hydrocephalus (NPH) are neurological disorders characterized, in part, by impaired brain clearance mechanisms and altered cerebrospinal fluid (CSF) dynamics. In AD, accumulation of amyloid-β initiates a cascade of neurodegenerative processes, including glial activation, tau pathology, and synaptic dysfunction, ultimately leading to neuronal loss. As disease progression limits therapeutic reversibility, there is increasing interest in interventions targeting early pathophysiological mechanisms, particularly those related to glymphatic and CSF-mediated clearance.

Transcranial low-intensity focused ultrasound has emerged as a promising non-invasive neuromodulatory modality capable of influencing brain physiology without structural disruption. Preclinical studies demonstrate that ultrasound stimulation can enhance CSF movement, modulate glymphatic transport, and facilitate the clearance of pathological proteins. In addition to these indirect effects, accumulating evidence suggests that ultrasound may directly influence aggregated protein structures, contributing to reduced neurotoxicity.

Translational in vivo studies further support these findings, showing reductions in pathological burden and enhanced peripheral clearance signals following repeated stimulation.

Early clinical investigations indicate that low-intensity ultrasound neuromodulation is feasible and well-tolerated, with preliminary signals suggesting potential cognitive and functional benefits in patients with AD. In parallel, NPH represents a condition in which impaired CSF circulation plays a central role, and early clinical evidence suggests that ultrasound-based modulation may improve functional outcomes, particularly in gait performance.

Together, these findings highlight focused ultrasound as a promising therapeutic strategy targeting brain clearance systems across neurodegenerative and neurofluid disorders.