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Neuroplasticity in Speech and Language Recovery Following Stroke or Traumatic Brain Injury

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Abstract

Background: Stroke and traumatic brain injury (TBI) frequently lead to acquired communication disorders (e.g., aphasia, dysarthria, cognitive-communication deficits). Recovery of speech and language functions in these populations depends on neuroplasticity—the brain's ability to reorganise neural networks in response to injury and therapeutic intervention.

Objective: To review mechanisms of neuroplasticity underlying speech and language recovery after stroke and TBI, summarise current evidence from neuroimaging and behavioural studies, discuss clinical implications for speech-language therapy and audiological rehabilitation, and propose future research directions.

Methods: A narrative review of literature published 2018–2025 was conducted via PubMed, Scopus, and ScienceDirect using keywords 'neuroplasticity', 'aphasia recovery', 'TBI language', 'speech therapy', 'stroke', 'traumatic brain injury', 'brain reorganisation'. Studies focusing on neural mechanisms, therapy-induced plasticity, and rehabilitation outcomes were included.

Results: Evidence identifies three major neuroplastic processes: (1) perilesional reorganisation within the dominant hemisphere, (2) recruitment of contralesional (non-dominant hemisphere) homologous regions and domain-general networks, and (3) restoration and remodelling of network connectivity (white matter tracts, functional networks). Intensive behavioural therapy (e.g., constraint-induced language therapy, melodic intonation therapy), neuromodulatory techniques (e.g., transcranial magnetic stimulation, tDCS), and auditory/motor-based interventions enhance plasticity and improve outcomes.

Conclusions: Neuroplasticity is fundamental to speech and language recovery after stroke or TBI. Therapeutic strategies that are intensive, targeted, and multimodal can optimise cortical and network-level reorganisation.

INTRODUCTION

Acquired brain injuries—such as ischemic or hemorrhagic stroke and traumatic brain injury—are among the leading causes of speech and language impairment in adults. Aphasia occurs in approximately 30-40% of stroke survivors and communication disorders are common after moderate-to-severe TBI.

Recovery is underpinned by neuroplasticity—the brain's capacity to reorganise synaptic connections, cortical activation patterns, and network connectivity in response to injury and therapeutic input. Understanding these mechanisms offers critical insights into how speech-language therapy and audiological rehabilitation can be optimised.

Methods

A narrative review was conducted. Databases (PubMed, Scopus, ScienceDirect) were searched for peer-reviewed articles published 2018–2025. Search terms included: 'neuroplasticity AND aphasia', 'TBI AND language recovery', 'stroke AND speech therapy AND neuroimaging', 'neuromodulation AND aphasia', 'white matter tracts AND language recovery'. Inclusion criteria: (1) adult participants with acquired brain injury (stroke or TBI); (2) studies investigating neural mechanisms (e.g., fMRI, DTI, EEG) or therapy outcomes; (3) English language; (4) speech-language or audiological outcome reported.

Results

Mechanisms of neuroplasticity include perilesional reorganisation, contralesional recruitment, and network-level connectivity changes. Therapy-induced plasticity is achieved via behavioural interventions, neuromodulation (TMS/tDCS), and auditory-music-based therapies. Factors influencing recovery include lesion size, age, therapy intensity, and comorbidities such as diabetes.

Discussion

For audiologists and speech-language pathologists, the neuroplasticity evidence emphasises key clinical take-aways: intensive and early therapy drives plasticity and better outcomes. Adjunct neuromodulation and music-based therapy enhance recovery. Network-based approaches, combining neuroimaging, cognitive assessment, and tele-rehabilitation, are promising directions for future practice.

Conclusion

Neuroplasticity underlies recovery of speech and language after stroke and TBI. Clinicians should adopt intensive, targeted, multimodal therapy strategies, informed by network and brain-health measures, to optimise recovery.

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