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Functional reorganization of the motor cortex following ischemic stroke: Insights from longitudinal neurophysiology studies.

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Introduction

Ischemic stroke, a leading cause of adult disability, often results in substantial motor impairments due to disruption of neural pathways involved in voluntary movement. The primary motor cortex (M1), along with associated sensorimotor regions, frequently suffers direct or indirect damage following stroke, leading to compromised motor control on the contralateral side of the body. However, the brain's remarkable capacity for plasticity allows for varying degrees of functional recovery. In particular, longitudinal neurophysiology studies employing tools such as functional magnetic resonance imaging (fMRI), transcranial magnetic stimulation (TMS), and electroencephalography (EEG) have shed light on how motor cortical networks reorganize themselves to compensate for stroke-induced damage. These studies reveal that functional reorganization is not static but evolves across acute, subacute, and chronic phases of recovery, and this dynamic process is influenced by lesion location, severity, and the intensity and timing of rehabilitation [1].

In the early phase after an ischemic stroke, functional imaging studies have demonstrated a shift in cortical activation patterns. Patients often show increased activity in the contralesional hemisphere, particularly in the homotopic motor cortex and supplementary motor areas. This bilateral cortical recruitment appears to be a compensatory mechanism, facilitating early motor function recovery while the damaged hemisphere undergoes reorganization. However, this increased contralesional activation is not always beneficial in the long term. Some studies suggest that excessive reliance on the contralesional motor cortex may interfere with the reestablishment of lateralized function in the affected hemisphere. Longitudinal data from TMS studies have shown that interhemispheric inhibition from the contralesional to ipsilesional M1 increases during early recovery but tends to normalize as function improves. These findings emphasize that the interplay between hemispheres is dynamic and may serve both adaptive and maladaptive roles depending on the stage of recovery [2].

As patients progress into the subacute phase of recovery, typically between one week and three months post-stroke, a shift toward more localized activation in the ipsilesional hemisphere is often observed. Functional recovery is associated with the re-engagement of spared motor networks within the damaged hemisphere. EEG studies reveal increased synchronization in the alpha and beta frequency bands over the ipsilesional sensorimotor cortex, indicating improved cortical excitability and coordination. Concurrently, resting-state fMRI studies show a gradual restoration of connectivity within the motor network, particularly between M1, premotor cortex, and supplementary motor areas. Structural plasticity also plays a role, as diffusion tensor imaging (DTI) has documented changes in white matter integrity along the corticospinal tract. These neurophysiological markers correlate with clinical improvements, reinforcing the idea that the brain is actively remodeling itself to regain lost function [3].

In the chronic phase of stroke recovery, which extends beyond six months, the nature of cortical reorganization becomes more stable. Patients with significant recovery often exhibit normalized activation patterns that closely resemble those of healthy individuals, with predominant activity in the ipsilesional motor cortex during movement tasks. However, patients with persistent deficits may continue to show aberrant activation patterns, such as diffuse bilateral activation or heightened contralesional engagement. TMS studies in chronic stroke survivors demonstrate variability in cortical excitability, with some patients exhibiting hypoexcitability in the affected M1 and others showing signs of cortical hyperexcitability, potentially reflecting compensatory recruitment. These inter-individual differences underscore the heterogeneity of stroke recovery and highlight the importance of personalized neurorehabilitation approaches. Importantly, studies have shown that even in the chronic phase, the motor cortex retains some degree of plasticity, which can be harnessed through targeted interventions such as constraint-induced movement therapy (CIMT) or non-invasive brain stimulation techniques [4].

Neurophysiological insights have not only deepened our understanding of motor cortex reorganization but also informed the development of novel therapeutic strategies. Techniques like repetitive TMS (rTMS) and transcranial direct current stimulation (tDCS) are being explored to modulate cortical excitability and promote functional recovery. These interventions aim to either enhance excitability in the ipsilesional M1 or reduce inhibitory signals from the contralesional hemisphere. Combined with motor training, they have shown promise in improving motor outcomes, particularly when applied during critical windows of heightened plasticity. Additionally, neurofeedback protocols using real-time fMRI or EEG allow patients to consciously modulate their brain activity, potentially accelerating the recovery process. Longitudinal neurophysiology studies continue to play a pivotal role in evaluating the efficacy of such interventions, providing biomarkers that track the evolving patterns of cortical reorganization in response to therapy. These findings collectively support a paradigm in which rehabilitation is dynamically tailored to the brain's plastic capacity at different stages of recovery [5].

Conclusion

The functional reorganization of the motor cortex following ischemic stroke is a complex, dynamic process characterized by shifting patterns of activation, connectivity, and excitability across time. Longitudinal neurophysiology studies have been instrumental in revealing how the brain adapts to injury, highlighting both compensatory mechanisms and maladaptive patterns that can influence recovery. These insights have led to the development of targeted rehabilitation strategies that aim to guide cortical reorganization toward more effective outcomes. Understanding the temporal evolution of motor cortex plasticity and its relationship with functional recovery is essential for optimizing therapeutic windows and personalizing interventions. Continued research in this field holds great promise for enhancing post-stroke rehabilitation and improving the quality of life for millions of stroke survivors worldwide.

References

1. Bajo M, Varodayan FP, Madamba SG, et al. IL-1 interacts with ethanol effects on GABAergic transmission in the mouse central amygdala. Front. Pharmacol. 2015;6:49.
2. Chiappalone M, Casagrande S, Tedesco M, et al. Opposite changes in glutamatergic and GABAergic transmission underlie the diffuse hyperexcitability of synapsin I–deficient cortical networks. Cereb Cortex. 2009;19(6):1422-39.
3. Liu P, Zhang X, He X, et al. Spinal GABAergic neurons are under feed-forward inhibitory control driven by A δ and C fibers in Gad2 td-Tomato mice. Mol Pain. 2021;17:174.

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4. Sun YG, Wu CS, Renger JJ, et al. GABAergic synaptic transmission triggers action potentials in thalamic reticular nucleus neurons. J Neurosci. 2012;32(23):7782-90.
5. Xie R, Manis PB. GABAergic and glycinergic inhibitory synaptic transmission in the ventral cochlear nucleus studied in VGAT channelrhodopsin-2 mice. Front. Neural Circuits.2014;8:84.

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