

Editorial Note on Neuromodulatory Traumatic Brain Injury

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Editorial Note

Neuromodulation, as defined by the International Neuromodulation Society, is the "alteration of nerve activity by delivering a targeted stimulus to modulate the abnormal behavior of the neural pathway caused by the disease process." The "alteration of nerve activity" indicates that stimuli of varying intensities both below and above the firing threshold of a neural target can mediate neuromodulation. Sub-threshold stimulation can lead to membrane depolarization, but the depolarization magnitude does not exceed the critical level in order to initiate an action potential in an excitable cell. Neurostimulation, on the other hand, initiates an action potential at or above the firing threshold that may mediate activity or signal transmission between neurons, within and between neural circuits, triggering downstream results. It is easy to see that, using neuromodulation, the initiation of an action potential plays a critical role in promoting recovery after TBI. When considering the broad body of work, stimuli of different strengths are required to include meta-plasticity mechanisms that facilitate relearning and restoration of skills. It is also apparent that sub threshold stimuli are equally important for the recovery and recovery of TBI.

Rehabilitation of TBI can be too restrictive, because the definition states that the "abnormal neural pathway" is the stimulation target. Restricting neuromodulation to the abnormal pathway is contradictory to an increasing awareness of how neuroplasticity facilitates rehabilitation after acquired brain injury. Specifically, evidence indicates that network-level reorganization allows more intact neural pathways to compensate for less functional pathways following acquired brain injury. For instance, correct lateralized network reorganization is correlated with language

recovery after stroke. Additionally, An increasing number of advanced imaging studies suggest that TBI recovery is also supported by neuronal changes occurring within and between functional and dysfunctional pathways. For instance, improvements in network communications (i.e. firing synchrony) and structural neural integrity (e.g. white fiber tracts) of more and less intact pathways are linked to functional skills recovery. Neuromodulatory treatments for TBI should not be limited to the targeting of abnormal pathways when considering this data, combined with the awareness that neuromodulation can enable complex control of neuronal circuits. Neuromodulation has the ability to reconfigure neural networks into various functional circuits for TBI recovery Improving the functioning and structural integrity of viable networks, engaging inactive networks, and likely generating new neural ties.

For TBI recovery, the INS concept is also problematic, as it does not reflect the very limited evidence regarding the protection of neuromodulation of any neural pathways after TBI. This relates to long-standing evidence that people with TBI are at an increased risk for seizures without neuromodulation. For TMS, there are empirically validated safety recommendations, but very few studies documenting safety for TBI remain, even for TMS. In view of the limited data, this particular issue requires a safety study on the provision of repetitive TMS (rTMS), alone and in conjunction with pharmacological neurostimulants To individuals that remain after TBI in states of disordered consciousness. As it offers an overview of rTMS safety evidence for mild TBI, a scoping analysis is also included in section. Although these studies resolve awareness gaps, insufficient TBI-specific safety evidence continues to exist. The safety of neuromodulation of any neuropathway following TBI is, therefore, largely unknown.

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