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Brief Report

Rewiring Recovery by Vagus Nerve Stimulation as a Durable Therapy for Post-Stroke Upper Limb Impairment

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ABSTRACT

Stroke remains a leading cause of long-term disability worldwide, with persistent Upper Extremity (UE) impairment affecting a significant proportion of survivors and posing a major challenge to functional recovery and quality of life. Conventional rehabilitation approaches, including physical and occupational therapy, Constraint-Induced Movement Therapy (CIMT), and neuromodulatory adjuncts, offer modest gains, particularly when initiated early. However, these benefits often plateau in the chronic phase, leaving many patients with enduring deficits. The VNS-REHAB trial marks a pivotal advancement in post-stroke rehabilitation by pairing Vagus Nerve Stimulation (VNS) with task-specific motor training. Unlike traditional uses of VNS in epilepsy and depression where stimulation is continuous or intermittent, this approach delivers VNS in a time-locked manner during rehabilitation, enhancing neuroplasticity and motor relearning. Clinical outcomes from this randomized controlled trial demonstrate not only significant improvements in motor function but also durable, patient-relevant gains persisting for at least one year. The success of this intervention reflects a broader evolution of VNS as a neuromodulatory tool, with expanding applications in epilepsy, treatment-resistant depression, migraine, inflammatory disorders, and cognitive dysfunction. Mechanistically, VNS engages central monoaminergic pathways, modulates cortical excitability, reduces inflammation, and amplifies experience-dependent plasticity, positioning it as a potent plasticity-enhancing adjunct in neurorehabilitation. While surgical implantation and patient selection pose challenges, the findings redefine the scope of recovery in chronic stroke, offering renewed hope for meaningful functional restoration long after injury.

Keywords: Vagus Nerve Stimulation, Stroke Rehabilitation, Upper Extremity Impairment, Neuroplasticity, Neuromodulation.

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INTRODUCTION

Stroke is one of the leading causes of long-term disability worldwide, and for many survivors, persistent Upper Extremity (UE) impairment severely limits daily function and quality of life.[1-3] The incidence rate of persistent UE impairment following a stroke is notably high, affecting a significant proportion of stroke survivors. Research indicates that approximately 50-70% of individuals experience UE motor deficits immediately after a stroke, and up to 40-50% continue to exhibit some degree of impairment six months later.[1,4] Despite initial recovery in many patients, persistent deficits in arm and hand function remain common, limiting independence and quality of life. Factors such as stroke severity, lesion location, and early rehabilitation engagement influence long-term outcomes.[1,4] These high incidence rates highlight the critical need for targeted interventions to improve UE recovery and functional use in daily activities.

Current treatment options for UE impairment after stroke include physical and occupational therapy, Constraint-Induced Movement Therapy (CIMT), robotic-assisted therapy, mirror therapy, Neuromuscular Electrical Stimulation (NMES), and pharmacologic or neuromodulation approaches.[5-9] Conventional therapy remains the cornerstone of rehabilitation, focusing on repetitive, task-specific training to improve motor control and function. CIMT, which promotes use of the affected limb by restricting the unaffected one, has shown strong evidence for improving motor outcomes in selected patients.[8] Robotic-assisted devices offer intensive, consistent movement practice and have demonstrated modest benefits, particularly in proximal UE function.[9] Mirror therapy and NMES are cost-effective adjuncts that enhance motor recovery, especially in early stages.[6,7] More recent approaches, such as Transcranial Magnetic Stimulation (TMS) and transcranial Direct Current Stimulation (tDCS), show promise in enhancing neuroplasticity when combined with physical therapy, though evidence remains mixed.[10,11] Overall,

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while many treatments offer moderate improvements, especially when started early and tailored to patient needs, full functional recovery remains challenging, and no single intervention is universally effective.[12,13] While intensive task-specific rehabilitation has shown benefits, those gains often plateau, particularly in the chronic phase post-stroke. In this landscape of unmet need, a recent study provides a compelling and timely look at the long-term promise of Vagus Nerve Stimulation (VNS) as a transformative adjunct to stroke rehabilitation.[14] Their findings confirm what the field has long hoped: that pairing VNS with motor rehabilitation can produce not only clinically meaningful improvements but also durable ones that last at least a year.[14]

The VNS-REHAB trial, was a multicentre, randomized, controlled study involving 108 participants with chronic ischemic stroke and moderate-to-severe UE impairment.[14] The treatment protocol involved 18 clinic-based rehabilitation sessions and three months of home-based exercises, with either active or sham VNS. Later, participants in the control group crossed over to receive active VNS, followed by a year of home-based, self-initiated VNS paired with rehabilitation tasks. At the 12-month follow-up, patients demonstrated sustained improvements in motor impairment, functional activity, and patient-reported outcomes, including measures of arm use, quality of movement, and quality of life. The statistical robustness of these outcomes is matched by their clinical relevance. Improvements on the Fugl-Meyer Assessment and Wolf Motor Function Test suggest tangible functional recovery.[14] Even more persuasive are the patient-centred outcomes: better scores on the Stroke Impact Scale and quality-of-life indices indicate that these gains matter in everyday life. In an era where many therapies show promising short-term results but fail to hold up over time, this study stands out for demonstrating lasting efficacy. This is particularly important because chronic stroke patients often face diminished expectations for further recovery expectations that this study directly challenges.

Comparing this to broader clinical applications of VNS, we see an evolving and increasingly sophisticated tool in neuromodulation. VNS has evolved into a versatile neuromodulatory intervention with applications across a range of neurological, psychiatric, and emerging medical conditions (Figure 1).[15,16] Initially developed and approved by the U.S. Food and Drug Administration (FDA) in the 1990s for the treatment of drug-resistant epilepsy, VNS has demonstrated significant efficacy in reducing seizure frequency, with long-term studies showing a sustained response in approximately 40-50% of patients.[10,17] The success in epilepsy led to its extension into psychiatry, with FDA approval for Treatment-Resistant Depression (TRD) in 2005.[18-20] In TRD, VNS has shown clinical benefit in mood stabilization and long-term remission rates, particularly in patients unresponsive to multiple pharmacologic interventions, although the onset of therapeutic effects may take several months.[18-20] Beyond its established indications, VNS is being actively investigated for a variety of other clinical applications. In chronic migraine, early trials have shown that non-invasive VNS can reduce headache frequency and severity, leading to CE marking in Europe and investigational use in the United States.[15,21] Inflammatory disorders such as rheumatoid arthritis and Inflammatory Bowel Disease (IBD) have also emerged as potential targets, based on the role of the Vagus nerve in modulating systemic inflammation through the cholinergic anti-inflammatory pathway.[22] Preliminary studies have demonstrated reductions in pro-inflammatory cytokines and symptomatic improvement, although larger randomized controlled trials are ongoing. Cognitive disorders, including Alzheimer's disease, represent another promising frontier.[23,24] While still in early stages, VNS has shown potential to enhance memory and attention, possibly by modulating neurotransmitter systems and promoting neural plasticity.[25] These findings suggest a role for VNS in cognitive rehabilitation and age-related cognitive decline, though more rigorous evidence is required.

VNS exerts its therapeutic effects through a complex network of neuroanatomical pathways and biochemical processes that influence both central and peripheral systems (Figure 1).[26,27] The Vagus nerve, the tenth cranial nerve, consists predominantly of afferent fibers (approximately 80%) that transmit sensory information from the body to the brain, and efferent fibers (around 20%) that regulate autonomic output.[28,29] By delivering electrical stimulation to the cervical Vagus nerve, VNS initiates a cascade of neural activity that impacts several brain regions and physiological systems. These mechanisms are responsible for the observed clinical benefits of VNS in conditions such as epilepsy, depression, stroke recovery, inflammatory diseases, and cognitive dysfunction. One of the primary mechanisms by which VNS exerts its effects is through activation of brainstem nuclei, particularly the Nucleus Tractus Solitarius (NTS), which receives vagal afferents.[26,27] The NTS serves as a relay to other critical brain structures, including the Locus Coeruleus (LC) and Dorsal Raphe Nucleus (DRN), which are major sources of norepinephrine and serotonin, respectively.[30,31] These monoaminergic systems project widely to cortical and subcortical areas, modulating brain regions involved in mood, arousal, attention, and seizure activity. This widespread neuromodulatory influence explains the efficacy of VNS in both neurological conditions like epilepsy and psychiatric disorders such as treatment-resistant depression. In epilepsy, VNS reduces seizure frequency by modulating cortical excitability and desynchronizing hyperactive neural circuits.[32,33] The therapy is believed to enhance inhibitory neurotransmission (GABAergic activity) while reducing excitatory glutamatergic signalling.[34] Functional imaging studies also show increased blood flow in seizure-related regions, including the thalamus and insula.[35] Over time, these effects contribute to long-term plasticity that elevates seizure thresholds, making VNS a valuable adjunct in managing refractory epilepsy. For treatment-resistant depression, VNS enhances the availability of serotonin and norepinephrine in limbic and prefrontal regions implicated in emotional

regulation.[36,37] It alters activity in the subgenual cingulate cortex, amygdala, and prefrontal cortex areas commonly dysregulated in depression. Additionally, VNS promotes synaptic plasticity and may stimulate hippocampal neurogenesis, mechanisms associated with sustained antidepressant effects.[38] It also improves the connectivity and regulation of large-scale brain networks, such as the default mode and salience networks, thereby contributing to mood stabilization and emotional resilience. In the context of stroke rehabilitation, VNS has demonstrated a unique mechanism of action.[14] When stimulation is paired with voluntary motor activity, as in the VNS-REHAB trial, it enhances activity-dependent plasticity by reinforcing the specific neural circuits engaged during movement. This timing-dependent stimulation promotes cortical reorganization and improves motor learning by facilitating long-term potentiation and the expression of genes associated with neural repair, such as Brain-Derived Neurotrophic Factor (BDNF).[14] This targeted neuromodulation approach has been shown to significantly improve upper extremity function in stroke survivors. VNS also exerts potent anti-inflammatory effects through the cholinergic anti-inflammatory pathway.[39] Activation of efferent vagal fibers modulates immune responses by reducing the release of pro-inflammatory cytokines like Tumour Necrosis Factor-alpha (TNF-α) via stimulation of α7 nicotinic acetylcholine receptors on macrophages.[40] This mechanism underlies the potential of VNS in treating chronic inflammatory conditions such as rheumatoid arthritis and inflammatory bowel disease and has even shown benefit in early-stage clinical trials. Emerging evidence suggests that VNS may also enhance cognitive performance and offer neuroprotective effects. By increasing cholinergic and noradrenergic transmission, VNS improves attention and working memory.[36,41] It also appears to support synaptic plasticity and reduce oxidative stress and inflammation in the brain, indicating potential utility in neurodegenerative diseases such as Alzheimer's and in recovery from traumatic brain injury. Although these applications are still under investigation, they highlight the growing scope of VNS in cognitive and neurological health.

Despite its promise, VNS for stroke recovery is not without limitations. The device requires surgical implantation, which carries risk, cost, and logistical complexity. While newer forms of non-invasive VNS are being explored, they remain unproven in rigorous clinical trials. Furthermore, patient selection will be crucial: not all stroke survivors will benefit equally, and factors such as lesion location, cognitive capacity, and engagement in therapy likely influence outcomes.[42,43] Nonetheless, the findings from this study should reshape how we think about long-term stroke rehabilitation. The notion that recovery "ends" in the chronic phase is increasingly outdated. With the right tools like VNS combined with focused, high-quality rehabilitation, even patient's years post-stroke can experience real, meaningful improvements. More than a neuromodulatory trick, VNS appears to act as a plasticity amplifier, making the brain more responsive to effort, repetition, and intention. In summary, VNS paired with rehabilitation represents a potent, FDA-approved intervention

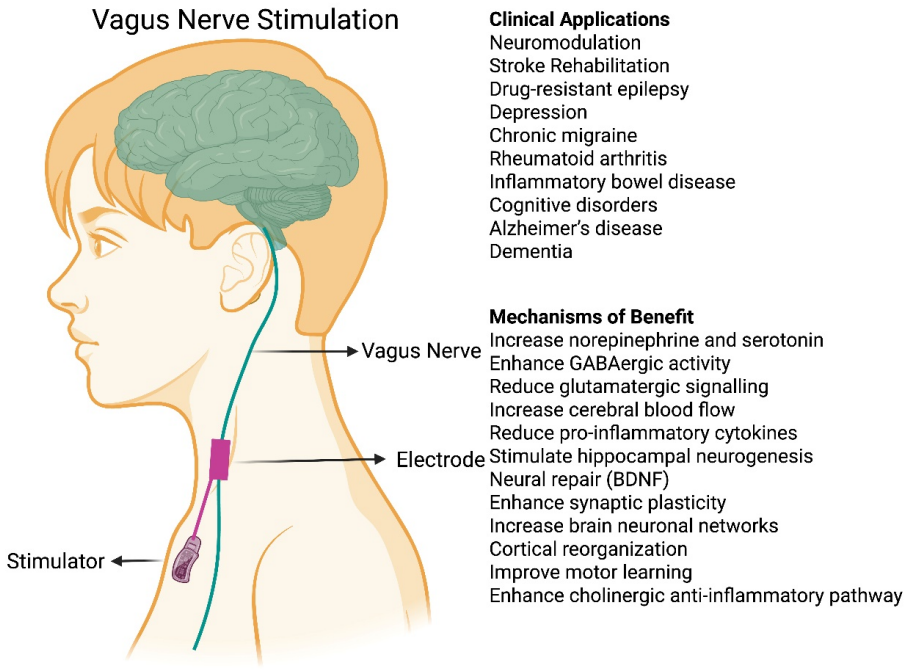

Figure 1: Vagus nerve stimulation. Vagus nerve stimulation involves the surgical implantation of a small device under the skin in the chest. A wire from this device is routed under the skin to the neck, where it is attached to the left Vagus nerve. Once in place, the device is programmed to send regular electrical impulses to the nerve, helping to regulate brain activity. After implantation, the stimulation settings can be adjusted externally by a healthcare provider. This procedure is primarily used to treat a verity of clinical condition where the clinical benefits are active by a diverse set of mechanisms.

with enduring benefit for chronic stroke survivors. Kimberley and colleagues have not only demonstrated clinical efficacy but also redefined what is possible in post-stroke recovery.[14] As neuromodulation technologies continue to evolve, VNS may well serve as a blueprint for future therapies that seek to harness the brain's latent potential long after injury. Rather than merely compensating for lost function, we are beginning to uncover new ways to restore it.

CONCLUSION

Upper extremity impairment remains one of the most debilitating and persistent consequences of stroke, significantly reducing independence and quality of life for millions of survivors. Despite a broad array of conventional and emerging rehabilitation strategies, many individuals particularly those in the chronic phase post-stroke experience limited functional recovery. The recent advances in neuromodulatory therapies, and in particular the integration of VNS with task-specific rehabilitation, represent a promising shift in this therapeutic landscape. The VNS-REHAB trial provides compelling evidence that activity-dependent VNS can enhance neuroplasticity and motor relearning, resulting in clinically meaningful and sustained improvements in upper limb function. Moreover, the success of VNS in stroke rehabilitation highlights its broader potential as a plasticity amplifier across diverse neurological and systemic conditions. Its expanding clinical applications from epilepsy and depression to inflammatory and cognitive disorders demonstrate a versatile mechanism of action grounded in robust neurobiological pathways. While challenges remain, including surgical access, cost, and optimal patient selection, the durability and relevance of the functional gains observed with VNS mark a paradigm shift in long-term stroke care. These findings challenge the outdated notion that recovery plateaus in the chronic phase, offering new therapeutic possibilities for restoring lost function rather than merely compensating for it. Going forward, VNS may serve as both a catalyst and a blueprint for the next generation of neuromodulation-based neurorehabilitation strategies.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

BDNF: Brain-derived neurotrophic factor; **CIMT:** Constraint-induced movement therapy; **DRN:** Dorsal raphe nucleus; **FDA:** U.S. Food and Drug Administration; **IBD:** Inflammatory bowel disease; **LC:** Locus coeruleus; **NMES:** Neuromuscular electrical stimulation; **NTS:** Nucleus tractus solitarius; **tDCS:** Transcranial direct current stimulation; **TMS:** Transcranial magnetic stimulation; **TNF-α:** Tumour necrosis factor-alpha; **TRD:** Treatment-resistant depression; **UE:** Upper extremity; **VNS:** Vagus nerve stimulation.

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