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ISHEMIK INSULTDAN KEYINGI KOGNITIV BUZILISHLARDA TRANSKUTAN VAGUS NERV STIMULYATSIYASI: NEYROPLASTIKLIK, BDNF, EEG BIOMARKERLARI VA PRECIZION REABILITATSIYA MODELI

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ANNOTATSIYA

Ishemik insultdan keyingi kognitiv buzilishlar uzoq muddatli funksional mustaqillik, ijtimoiy moslashuv va hayot sifatiga sezilarli ta’sir ko‘rsatadi. Transkutan aurikulyar vagus nerv stimulyatsiyasi (taVNS) noinvaziv neyromodulyatsiya usuli bo‘lib, vagal afferentlar orqali miya ustuni yadrolari, noradrenergik va xolinergik tizimlar, neyroplastiklik, yallig‘lanish nazorati va neyrotrofik mexanizmlarga ta’sir ko‘rsatishi mumkin. Ushbu maqolada kechki tiklanish davridagi postinsult kognitiv buzilishlarda taVNSning zamonaviy dalillari tahlil qilinadi; MoCA va Trail Making Test kabi neyropsixologik ko‘rsatkichlar, EEG dinamikasi, qon zardobidagi BDNF va funksional mustaqillik o‘rtasidagi bog‘liqlik alohida ko‘rib chiqiladi. Mavjud klinik dalillar istiqbolli bo‘lsada, kognitiv natijalar bo‘yicha katta randomizatsiyalangan tadqiqotlar hali cheklangan. Shu sababli taVNS samaradorligini klinik, neyropsixologik, neyrofiziologik va neyrotrofik markerlarni birlashtirgan multimodal modelda baholash taklif etiladi.

Kalit so‘zlar: ishemik insult, postinsult kognitiv buzilish, transkutan aurikulyar vagus nerv stimulyatsiyasi, taVNS, BDNF, EEG, MoCA, Trail Making Test, neyroplastiklik, neyroreabilitatsiya.

Давлатбаев Алишер Фахраддин угли
Мажидова Ёкутхон Набиевна

ТРАНСКУТАННАЯ СТИМУЛЯЦИЯ БЛУЖДАЮЩЕГО НЕРВА ПРИ ПОСТИНСУЛЬТНЫХ КОГНИТИВНЫХ НАРУШЕНИЯХ: НЕЙРОПЛАСТИЧНОСТЬ, BDNF, ЭЭГ-БИОМАРКЕРЫ И МОДЕЛЬ ПРЕЦИЗИОННОЙ РЕАБИЛИТАЦИИ

АННОТАЦИЯ

Постинсультные когнитивные нарушения существенно ограничивают долгосрочную функциональную независимость, социальную адаптацию и качество жизни пациентов после ишемического инсульта. Транскутанная аурикулярная стимуляция блуждающего нерва (taVNS) представляет собой неинвазивную нейромодуляционную технологию, способную через вагальные афференты воздействовать на стволовые ядра, норадренергические и холинергические системы, механизмы нейропластичности, нейровоспаления и нейротрофической поддержки. В статье проанализированы современные данные о применении taVNS при постинсультных когнитивных нарушениях в позднем восстановительном периоде. Особое внимание уделено интеграции нейропсихологических показателей (MoCA, Trail Making Test), ЭЭГ, сывороточного BDNF, неврологического дефицита и функциональной независимости. Прямые данные по когнитивным исходам остаются ограниченными, поэтому предлагается мультимодальная стратегия оценки терапевтического ответа.

Ключевые слова: ишемический инсульт, постинсультные когнитивные нарушения, транскутанная аурикулярная стимуляция блуждающего нерва, taVNS, BDNF, ЭЭГ, MoCA, нейропластичность, нейрореабилитация.

Davlatbayev Alisher Faxraddin o‘g‘li
Majidova Yoqutxon Nabiyevna

TRANSCUTANEOUS VAGUS NERVE STIMULATION IN POST-STROKE COGNITIVE IMPAIRMENT: NEUROPLASTICITY, BDNF, EEG BIOMARKERS, AND A PRECISION REHABILITATION FRAMEWORK

ANNOTATION

Post-stroke cognitive impairment (PSCI) is a major determinant of long-term disability after ischemic stroke and may compromise independence even when motor recovery is relatively favorable. Transcutaneous auricular vagus nerve stimulation (taVNS) is an emerging non-invasive neuromodulation technique that activates vagal afferents projecting to the nucleus tractus

solitarius and, through downstream noradrenergic and cholinergic systems, may facilitate experience-dependent plasticity, modulate neuroinflammation, and influence neurotrophic signaling. This narrative review examines the rationale for taVNS in PSCI with a specific focus on the late recovery period, 6-12 months after ischemic stroke. The review integrates direct cognitive evidence with the broader stroke-rehabilitation literature and proposes a multimodal response model combining cognitive outcomes (Montreal Cognitive Assessment and Trail Making Test), neurological deficit, functional independence, electroencephalographic dynamics, and serum brain-derived neurotrophic factor (BDNF). A randomized protocol specifically targeting PSCI has established the feasibility of an 8-week taVNS paradigm, while a 2024 DTI case report described improvement in global cognition and executive performance together with white-matter remodeling. More recent stroke trials and meta-analyses strengthen the biological plausibility of taVNS by demonstrating functional benefit, neurophysiological effects, and treatment-related changes in plasticity biomarkers including BDNF. Nevertheless, direct high-quality evidence for cognitive recovery after stroke remains limited, and stimulation parameters, patient selection, timing, and response biomarkers are not yet standardized. A precision-rehabilitation strategy based on convergent clinical, neuropsychological, EEG, and neurotrophic markers may therefore be a rational next step for clinical research.

Keywords: ischemic stroke, post-stroke cognitive impairment, transcutaneous auricular vagus nerve stimulation, taVNS, BDNF, electroencephalography, MoCA, Trail Making Test, neuroplasticity, neurorehabilitation.

I. Introduction.

Ischemic stroke remains a major cause of death and long-term neurological disability worldwide. Survival after stroke has improved, but the clinical problem has shifted increasingly toward the quality of recovery. Cognitive dysfunction is especially important because it influences communication, medication adherence, rehabilitation engagement, instrumental activities of daily living, social reintegration, and the capacity to return to work. Post-stroke cognitive impairment is not a single syndrome: depending on lesion characteristics and premorbid reserve, patients may exhibit abnormalities in attention, processing speed, executive control, memory, language, visuospatial functions, or combinations of these domains [1,2].

The late recovery period deserves specific attention. During the first months after stroke, spontaneous biological recovery and intensive rehabilitation may produce rapid gains. By 6-12 months, however, the trajectory becomes more heterogeneous. Some patients continue to improve, whereas others plateau with persistent cognitive inefficiency that is not adequately addressed by conventional motor-focused rehabilitation. The dissertation rationale on which this article is based therefore emphasizes patients 6-12 months after ischemic stroke and proposes simultaneous assessment of cognition, neurological deficit, functional independence, electroencephalography (EEG), and serum brain-derived neurotrophic factor (BDNF). This multidimensional design is clinically attractive because it treats cognitive recovery as a systems-level process rather than an isolated psychometric score.

Vagus nerve stimulation (VNS) has emerged as one of the most compelling neuromodulatory approaches in stroke rehabilitation. Invasive VNS paired with task-specific upper-limb rehabilitation has already shown efficacy in chronic ischemic stroke, providing proof of principle that timed vagal activation can enhance rehabilitation-induced plasticity [3]. Transcutaneous approaches are particularly attractive for cognitive rehabilitation because they avoid surgery, can be repeated over weeks, and may be suitable for home-based or outpatient programs. The auricular branch of the vagus nerve can be stimulated non-invasively, engaging brainstem and forebrain neuromodulatory circuits that are relevant to attention, learning, memory, arousal, and plasticity [4,5].

The central scientific question is no longer whether vagal stimulation has biological effects, but which patients are most likely to benefit, which stimulation parameters should be used, how cognitive response should be measured, and which biomarkers can verify that the intended neuroplastic process has occurred. This review addresses those questions by combining the evidence on PSCI with mechanistic and rehabilitation studies of VNS. Particular emphasis is placed on a precision framework integrating MoCA, Trail Making Test (TMT), EEG, serum BDNF, NIHSS, and Barthel Index outcomes.

II. Literature search strategy

A focused narrative literature search was conducted using PubMed-indexed sources through September 2026. Search terms included combinations of "post-stroke cognitive impairment", "ischemic stroke", "transcutaneous auricular vagus nerve stimulation", "taVNS", "vagus nerve stimulation", "cognition", "BDNF", "EEG", "neuroplasticity", and "stroke rehabilitation". Priority was given to randomized clinical trials, trial protocols, systematic reviews, meta-analyses, mechanistic reviews, and recent human studies that included cognitive, electrophysiological, functional, or neurotrophic outcomes.

Because direct randomized evidence for taVNS specifically in PSCI is still sparse, the review distinguishes three evidence layers: (1) direct PSCI evidence, (2) clinical stroke-rehabilitation evidence demonstrating feasibility, safety, and functional effects of VNS or taVNS, and (3) mechanistic evidence relevant to cognitive recovery, including neuromodulatory, neuroinflammatory, and BDNF-related pathways. This distinction is important to avoid extrapolating motor-rehabilitation findings as if they were already proven cognitive treatment effects.

III. Post-stroke cognitive impairment as a neurorehabilitation target

Cognitive impairment after stroke reflects both focal lesion effects and distributed network disruption. Strategic infarcts involving frontal-subcortical circuits, thalamus, hippocampal connections, temporoparietal association cortex, or dominant-hemisphere language networks may produce characteristic profiles, but even small lesions can cause disproportionate dysfunction when they disconnect high-value network hubs. Executive slowing and attentional inefficiency are especially common after vascular brain injury and can reduce the apparent effectiveness of rehabilitation because patients learn new motor strategies more slowly, fatigue earlier, and show reduced transfer from supervised therapy to daily life.

Cognitive status should therefore be treated as a rehabilitation outcome rather than a background comorbidity. The Montreal Cognitive Assessment (MoCA) is useful as a global screen because it samples executive, attentional, visuospatial, language, memory, and orientation domains and is generally more sensitive than the Mini-Mental State Examination for mild vascular cognitive deficits. However, a global score can conceal the mechanism of change. Trail Making Test part A provides information about visual scanning and processing speed, whereas part B adds set-shifting and executive

control. The relationship between TMT-B and TMT-A is clinically informative because a patient can improve in basic speed without recovering higher-order flexibility.

The late recovery period is particularly appropriate for mechanistic research because the most rapid spontaneous recovery has diminished, yet the brain remains plastic and responsive to training. A treatment-induced improvement at 6-12 months is therefore less likely to be explained solely by early spontaneous resolution. At the same time, the late period brings new confounders: depressive symptoms, fatigue, sleep disturbance, recurrent vascular events, medication effects, and reduced physical activity can all influence cognitive scores. A rigorous taVNS study should account for these factors rather than interpret every change as a direct neuromodulatory effect.

IV. Why the vagus nerve is a plausible gateway to cognitive plasticity

The vagus nerve provides a unique route by which peripheral stimulation can influence widespread central networks. Auricular vagal afferents project to the nucleus tractus solitarius, which is functionally connected to the locus coeruleus and other neuromodulatory nuclei. Through these pathways, VNS can influence noradrenergic and cholinergic transmission, both of which are fundamental to attention, salience detection, memory encoding, and experience-dependent synaptic change [4,5].

The timing principle is central to VNS-assisted rehabilitation. Vagal activation is not conceptualized simply as generalized electrical stimulation. Instead, brief or repeated stimulation is thought to create a neurochemical state in which behaviorally relevant network activity is more likely to be reinforced. The pivotal VNS-REHAB trial in chronic ischemic stroke demonstrated that implanted VNS paired with rehabilitation improved upper-limb impairment more than sham stimulation paired with the same therapy [3]. Although the outcome was motor rather than cognitive, the trial established a clinically important concept: vagal neuromodulation can amplify task-linked recovery even years after stroke.

For cognition, the same principle suggests pairing taVNS with structured cognitive engagement rather than delivering stimulation in isolation. Attention tasks, memory exercises, executive training, reading, language practice, and everyday goal-directed activities may provide the behavioral substrate upon which vagally facilitated plasticity acts. The precise timing and dosing required for cognitive rehabilitation remain uncertain, which is one reason parameter standardization is a major research priority.

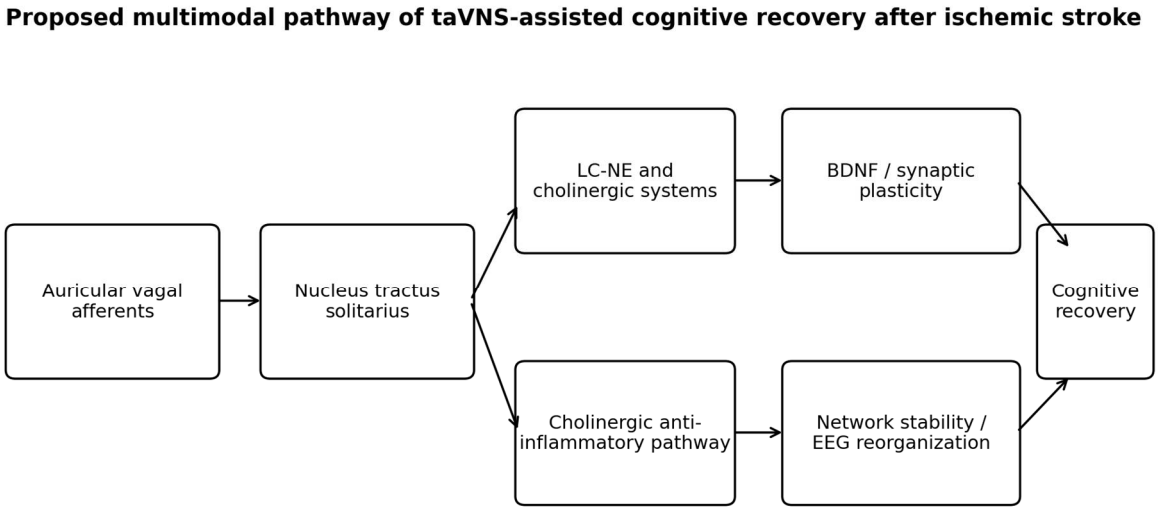


Figure 1. Proposed mechanistic pathway linking taVNS to cognitive recovery after ischemic stroke

Figure 1 illustrates a mechanistic framework rather than a proven causal sequence. The strongest human evidence supports engagement of neuromodulatory systems and clinically measurable rehabilitation effects; BDNF, EEG normalization, and cognitive recovery should be tested as linked but distinct outcome domains.

V. Direct clinical evidence in post-stroke cognitive impairment

Direct evidence for taVNS in PSCI is promising but still limited. Li and colleagues published a randomized controlled trial protocol designed to enroll 88 participants with post-stroke cognitive impairment. The intervention was planned for 8 weeks, 5 days per week, with two 30-minute sessions per day, comparing taVNS with sham stimulation. Cognitive outcomes were designated as primary endpoints, while activities of daily living, quality of life, and serum oxidative-stress markers were included as secondary outcomes [6]. The protocol is important because it demonstrates that PSCI is being treated as an independent therapeutic target rather than a secondary endpoint of motor rehabilitation.

A 2024 case report by Chen et al. provides direct human evidence of potential cognitive and structural effects in long-term PSCI. A 71-year-old man with chronic cognitive impairment after stroke received home-based taVNS twice daily for 30 minutes, five days per week, for 8 weeks. Global cognition improved on MoCA, performance on the Shape Trails Test became faster, and diffusion tensor imaging suggested white-matter remodeling in bilateral dorsolateral prefrontal regions [7]. Because the report involved a single patient, causality cannot be established, but the convergence of behavioral and imaging change is scientifically valuable and supports the use of multimodal outcomes in future trials.

More broadly, VNS has shown cognitive effects across neurological and non-neurological populations. A 2024 systematic review summarized effects on attention, executive functions, learning, and memory, while a 2026 meta-analysis across more than 1,500 participants reported an overall cognitive effect and relatively stronger effects for executive control and cognitive flexibility/learning [8,9]. These findings cannot be directly transferred to PSCI, but they strengthen the biological plausibility that vagal stimulation can influence cognition when the appropriate neural systems and behavioral tasks are engaged.

VI. Bdnf as a neurotrophic response marker

BDNF is a logical biomarker for a study of neuromodulation and cognitive recovery because it participates in synaptic plasticity, dendritic remodeling, learning, and memory. Experimental stroke research has repeatedly linked auricular VNS to neuroplastic processes that include increased BDNF expression. A systematic review and meta-analysis of animal stroke models found that auricular VNS increased neuroplasticity-related markers, including BDNF protein and RNA expression, while improving neurological outcomes and reducing infarct-related damage [10].

The translational gap, however, must be handled carefully. Brain BDNF signaling cannot be inferred directly from a single serum measurement, and peripheral BDNF is influenced by age, physical activity, platelet biology, medications, sleep, metabolic status, and inflammatory conditions. Therefore, serum BDNF should not be treated as a stand-alone surrogate of cortical plasticity. Its value is greater when interpreted longitudinally and in parallel with cognitive, functional, and neurophysiological changes.

Human stroke data are now beginning to support this approach. In a randomized comparative study of chronic stroke patients, trans-auricular VNS added to rehabilitation was associated with improvement in serum BDNF and IL-6 together with gains in gross manual dexterity [11]. Although the primary clinical outcome was motor rather than cognitive, the study demonstrates that neurotrophic biomarkers can be integrated into a taVNS trial and can show treatment-related dynamics. For PSCI, the key question is whether BDNF change correlates with improvement in MoCA and executive-attentional performance rather than merely changing in parallel with general rehabilitation.

VII. Eeg as a functional biomarker of network recovery

EEG offers a complementary window into post-stroke recovery because it measures brain-network dynamics directly and repeatedly at relatively low cost. Ischemic injury commonly produces focal or diffuse slowing, altered hemispheric symmetry, reduced alpha organization, and changes in functional connectivity. These abnormalities can persist beyond the acute stage and may reflect the integrity of thalamocortical and corticocortical systems that support attention and cognition.

A taVNS study should define EEG outcomes prospectively rather than simply describing the tracing as "improved". Potential quantitative measures include relative delta and theta power, alpha power, alpha/delta ratio, hemispheric asymmetry, spectral entropy, and connectivity metrics when technically feasible. A biologically coherent response pattern might include reduction of excessive slow-wave activity, improved alpha organization, or normalization of asymmetry, but the exact marker should be selected in advance and analyzed consistently.

The importance of EEG is not that it proves a mechanism by itself, but that it can connect clinical improvement to measurable changes in brain function. If a patient demonstrates better MoCA and TMT performance without EEG change, the treatment may still be clinically useful. If cognitive improvement is accompanied by reproducible EEG normalization and rising BDNF, however, the convergence strengthens the interpretation that a broader plasticity process has occurred. This "clinical-neuropsychological-neurophysiological-neurotrophic" chain is one of the most valuable conceptual strengths of the proposed research model.

VIII. What the broader stroke vns literature adds

The stroke literature outside cognition provides several lessons for PSCI research. First, VNS effects are highly dependent on treatment context. The 2021 VNS-REHAB trial enrolled 108 participants with chronic ischemic stroke and moderate-to-severe upper-limb impairment. Participants received identical rehabilitation, but active VNS produced greater improvement in upper-limb impairment and a higher rate of clinically meaningful response than sham stimulation [3]. This shows that neuromodulation can add value beyond rehabilitation alone.

Second, the evidence base is moving from proof-of-concept toward parameter optimization. Recent reviews emphasize that stimulation modality, frequency, intensity, treatment duration, timing relative to rehabilitation, and patient phenotype are potential sources of heterogeneity [4,5,12]. A 2026 meta-analysis of ten randomized taVNS trials involving 512 stroke patients found improvements in motor function, mental-health outcomes, activities of daily living, and neurophysiological indicators, and identified stimulation frequency, intensity, and intervention duration as important modulators [13]. These outcomes are not equivalent to cognition, but they show why a cognitive trial should record stimulation dose with the same precision as a drug study records dose and duration.

Third, non-invasive VNS has practical advantages but should not be assumed to reproduce every effect of implanted VNS. Auricular stimulation depends on electrode site, current intensity, waveform, individual anatomy, skin impedance, and adherence. Sham design is challenging because participants may perceive active stimulation. For a future PSCI trial, blinding procedures, tolerability reporting, adherence tracking, and predefined parameter reporting are therefore essential.

Table 1

Selected evidence relevant to tavns and post-stroke cognitive rehabilitation

Study	Population / design	Intervention	Key outcome	Relevance to PSCI
Li et al., 2022 [6]	88 planned PSCI participants; randomized protocol	taVNS vs sham; 8 weeks	Cognition primary; ADL/QoL and serum markers secondary	Direct PSCI trial framework

Chen et al., 2024 [7]	71-year-old with long-term PSCI; case report	Home taVNS; 8 weeks	MoCA improved; Shape Trails faster; DTI white-matter changes	Direct cognitive + imaging signal
Dawson et al., 2021 [3]	108 chronic ischemic stroke patients; pivotal RCT	Implanted VNS paired with rehabilitation	Greater upper-limb recovery than sham	Proof of task-paired VNS neurorehabilitation
Moustafa et al., 2026 [11]	78 chronic stroke patients; randomized comparative trial	Trans-auricular VNS + rehabilitation vs sham	BDNF and IL-6 improved; motor dexterity improved	Supports BDNF as human response biomarker
Wan et al., 2026 [13]	10 RCTs, 512 stroke patients; meta-analysis	taVNS-based rehabilitation	Benefits in motor, mental health, ADL, neurophysiology	Shows parameter-dependent clinical effect
Liu & Li, 2026 [9]	53 studies; >1,500 participants; cognitive meta-analysis	taVNS across clinical/nonclinical samples	Overall cognitive benefit; stronger executive/learning effects	Supports cognitive plausibility; not stroke-specific

Table 1 shows that the strongest direct PSCI evidence remains limited, while the broader VNS literature provides increasingly robust support for neurorehabilitation and biomarker-oriented study designs.

IX. A precision assessment model for late-recovery psci

A clinically useful taVNS study should avoid a single-outcome design. A patient may improve in one cognitive domain without a large change in global MoCA score; functional independence may improve because of better attention even if neurological deficit is stable; EEG may normalize before cognition changes; BDNF may rise without clinically meaningful benefit. The most informative design therefore integrates several domains and tests whether they move together.

The proposed framework begins with clinical phenotyping. Stroke date, vascular territory, lesion side, lesion volume when available, recurrent stroke history, vascular risk factors, education, premorbid cognitive reserve, depression/anxiety, sleep, medication exposure, and physical activity should be documented. Neurological status is quantified using NIHSS, while Barthel Index captures functional independence. These measures do not replace cognitive testing but help explain why two patients with similar MoCA scores may have very different rehabilitation needs.

The cognitive core should include MoCA and TMT-A/B as proposed in the dissertation rationale. If resources permit, adding a verbal learning measure and semantic or phonemic fluency would increase domain specificity. For longitudinal interpretation, the same testing conditions should be used at baseline and follow-up, preferably at similar times of day and with standardized management of fatigue. Practice effects must be considered, especially when tests are repeated at short intervals.

EEG and serum BDNF add mechanistic depth. The key analytical question is not simply whether each variable changes, but whether changes correlate. For example, does a greater increase in BDNF predict a larger improvement in TMT-B? Does reduction in slow-wave activity correlate with MoCA executive/attention items? Do these associations remain after adjustment for age, lesion characteristics, baseline cognition, depression, and rehabilitation dose? This is where a descriptive clinical study becomes a mechanistically informative one.

Table 2

Proposed multimodal outcome framework for tavns in late-recovery psci

Domain	Core measure	Interpretive question	Potential response signal
Global cognition	MoCA	Does overall multidomain cognition improve?	Increase beyond expected retest effect
Attention / executive function	TMT-A and TMT-B	Is processing speed or set shifting preferentially affected?	Shorter completion time; fewer errors
Neurological deficit	NIHSS	Does cognitive change occur independently of major neurological change?	Stable or improved NIHSS
Functional independence	Barthel Index	Does cognitive improvement translate into daily function?	Higher independence score
Neurophysiology	Quantitative EEG	Is cognitive recovery accompanied by network normalization?	Less excessive slow activity; improved alpha organization/asymmetry
Neurotrophic biology	Serum BDNF	Is there a systemic marker associated with plasticity response?	Longitudinal increase correlated with cognitive gain

Affective state	Validated depression/anxiety scale	Could mood explain cognitive change?	Adjusted analysis separates affective and cognitive effects

X. Patient selection and response stratification

Patient selection may be as important as the stimulation itself. PSCI is heterogeneous, and a uniform taVNS protocol applied to all stroke survivors may dilute treatment effects. Potentially favorable candidates may include medically stable patients in the late recovery period who have persistent but testable cognitive impairment, preserved ability to engage in rehabilitation, and no major contraindication to electrical stimulation. Severe aphasia, profound dementia, uncontrolled epilepsy, unstable cardiac conditions, or inability to cooperate with testing may limit interpretability and should be addressed explicitly in the protocol.

Response stratification should ideally distinguish cognitive responders from non-responders using a prespecified clinically meaningful criterion rather than post hoc impressions. The threshold may be based on change in MoCA, improvement in executive-attentional testing, or a composite response that incorporates both cognitive and functional change. Multivariable analysis can then evaluate whether baseline BDNF, EEG slowing, lesion features, age, stroke-to-treatment interval, or baseline cognitive severity independently predict response.

The value of such a model is practical. Instead of concluding that taVNS "works" or "does not work" for PSCI as a whole, the study can identify a phenotype in which treatment is more likely to be useful. This is the logic of precision rehabilitation: matching a neuromodulatory intervention to the biological and clinical characteristics of the patient.

XI. Stimulation parameters, safety, and implementation

Parameter heterogeneity is one of the main obstacles to interpretation across taVNS studies. Published protocols differ in stimulation site, frequency, pulse width, current intensity, session duration, number of sessions, and whether stimulation is delivered during or separate from rehabilitation. The PSCI protocol by Li et al. used an intensive 8-week schedule with twice-daily 30-minute sessions [6], whereas other stroke studies use shorter courses or pair stimulation directly with task practice. Recent reviews consistently identify parameter optimization as an unresolved problem [5,12,13].

For clinical research, the stimulation site should be anatomically specified, electrode geometry documented, current titrated within a predefined range, and adverse sensations recorded. Adherence should be measured rather than assumed. Non-invasive taVNS is generally considered well tolerated, with transient local discomfort or tingling among the more common issues in the literature, but safety reporting should still be systematic. Patients with implanted electronic devices, unstable arrhythmias, or other relevant risks require protocol-specific screening.

Implementation also depends on rehabilitation pairing. If taVNS is intended to facilitate cognitive plasticity, stimulation should be integrated with a structured cognitive task or standardized rehabilitation session whenever feasible. This creates a testable hypothesis: taVNS is an amplifier of task-relevant plasticity rather than a passive treatment administered independently of learning.

XII. Discussion

The current evidence positions taVNS at an intriguing transition point in post-stroke neurorehabilitation. The biological rationale is strong, the clinical stroke literature increasingly supports VNS-assisted recovery, and direct PSCI studies have begun to emerge. At the same time, the evidence is not yet sufficient to present taVNS as an established treatment for post-stroke cognitive impairment. The difference between promise and proof is especially important in a field where motor-recovery data can easily be overgeneralized to cognition.

Two observations support further focused research. First, the 2024 PSCI case report demonstrated parallel cognitive and DTI changes after an 8-week home taVNS program [7]. Second, human stroke trials now show that taVNS can influence measurable biological markers such as BDNF in addition to clinical function [11]. These strands of evidence converge with mechanistic work showing that VNS engages noradrenergic, cholinergic, inflammatory, vascular, and neuroplastic pathways [4,5,10,14]. Together they provide a coherent rationale for testing whether cognitive improvement is accompanied by EEG and BDNF changes in a defined late-recovery PSCI cohort.

The proposed 6-12 month window is scientifically valuable because it reduces the dominance of early spontaneous recovery while preserving a plausible capacity for training-induced plasticity. It also reflects a clinically underserved group: patients who are medically stable yet remain unable to resume previous cognitive roles. For these individuals, a gain in attention or executive control may have more impact on independence than a small change in motor impairment.

The main methodological challenge will be separating specific cognitive effects from general rehabilitation effects. Both active and comparison groups should receive equivalent standard rehabilitation, and outcome assessors should be blinded when feasible. Baseline cognitive severity, lesion characteristics, affective state, and rehabilitation dose should be measured because they may confound response. Statistical analysis should emphasize between-group change, effect size, confidence intervals, and responder analysis rather than relying only on within-group p values.

A second challenge is biomarker interpretation. Serum BDNF and EEG are attractive because they are scalable and repeatable, but neither is specific to taVNS. Their greatest scientific value lies in convergence. A pattern of cognitive improvement, functional gain, EEG reorganization, and BDNF increase would provide stronger mechanistic support than any single endpoint. Conversely, discordant results could identify subtypes of response and generate new hypotheses.

XIII. Limitations of the current evidence

The direct PSCI literature remains small, and much of the clinical confidence surrounding VNS in stroke comes from motor-rehabilitation studies. Sample sizes in cognitive reports are limited, stimulation protocols vary, and sham conditions are not standardized. Cognitive tests are vulnerable to practice effects, fatigue, education, language, and mood. BDNF is influenced by many systemic factors, while EEG outcomes can differ depending on montage, preprocessing, artifact rejection, and analytic method.

Recent meta-analyses demonstrate overall benefit of taVNS in stroke rehabilitation and cognition more broadly, but pooled results combine heterogeneous populations and outcomes [9,13]. These findings justify research but do not eliminate the need for diagnosis-specific trials. Future PSCI studies should pre-register primary outcomes, define responder criteria, report stimulation

parameters in full, and include sufficiently long follow-up to determine whether gains persist after stimulation ends.

XIV. Conclusion

Transcutaneous auricular vagus nerve stimulation is a biologically plausible and increasingly evidence-supported neuromodulatory strategy for stroke rehabilitation. For post-stroke cognitive impairment, however, the field remains at an earlier stage than motor recovery. The most persuasive current model is not to view taVNS as a stand-alone cognitive enhancer, but as a potential facilitator of rehabilitation-dependent plasticity.

In patients 6-12 months after ischemic stroke, a precision-rehabilitation framework that combines MoCA and TMT with NIHSS, Barthel Index, quantitative EEG, and serum BDNF can address three questions simultaneously: whether cognition improves, whether improvement translates into function, and whether clinical change is accompanied by neurophysiological or neurotrophic evidence of plasticity. This multimodal approach directly addresses the major gaps in the current literature and may help define which patients are most likely to respond.

The next generation of studies should therefore prioritize rigorous patient selection, sham-controlled design when feasible, standardized stimulation parameters, longitudinal biomarkers, and clinically meaningful responder analysis. If cognitive, EEG, and BDNF outcomes converge, taVNS could become an important component of individualized neurorehabilitation for persistent post-stroke cognitive impairment.

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