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Comparing Electrical and Ultrasound Transcutaneous Auricular Vagus Nerve Stimulation on Associative Memory

Benjamin J. Griffiths, PhD[1]; Ziqi He, MSc[1]; Irem Ciftepinar, MSc[1]; Hyuk Choi, PhD[2]; Jae-Jun Song, MD, PhD[3]; Marcus Kaiser, PhD[4,5,6]; JeYoung Jung, PhD[1,5,6]

ABSTRACT

Objectives: Associative memory, the ability to bind and retrieve relationships between unrelated elements, is a cornerstone of human cognition and a primary target for neurorehabilitation. Vagus nerve stimulation (VNS) has emerged as a promising method to modulate the locus coeruleus-norepinephrine system and hippocampal-prefrontal circuits essential for memory. However, the comparative efficacy of noninvasive modalities such as electrical transcutaneous VNS (E-taVNS) and the emerging field of ultrasound (U-taVNS) remains poorly understood in the context of active recall.

Materials and Methods: In this study, participants performed a cross-modal video–word associative memory task before and after receiving either E-taVNS or U-taVNS in active and sham conditions. We investigated whether these modalities enhance cued recall accuracy and retrieval reaction time.

Results: Our results revealed that neither E-taVNS nor U-taVNS significantly improved recall accuracy. However, E-taVNS significantly accelerated response times specifically for correctly recalled items. These findings suggest that although taVNS may not increase the likelihood of recalling associative memories after a brief retention interval, electrical stimulation may enhance the efficiency with which we do so.

Conclusions: These findings suggest that E-taVNS is a viable tool for facilitating memory processes, although further research is required to optimize ultrasound parameters and validate mechanistic pathways through physiologic monitoring.

Keywords: Associative memory, cognitive enhancement, noninvasive neuromodulation, transcutaneous vagus nerve stimulation, ultrasound stimulation

INTRODUCTION

Memory enables us to encode, store, and retrieve past experiences and forms a core foundation of human cognition.[1] Among different memory systems, associative memory is the ability to bind and retrieve relationships between otherwise unrelated elements and is critical for daily learning, decision-making, and constructing coherent situational representations.[2,3] Extensive evidence suggests the hippocampus and broader medial temporal lobe (MTL) structures as central to associative binding, integrating multimodal information into unified memory traces.[4] Associative memory also depends strongly on prefrontal executive control, particularly during strategic encoding or selective retrieval.[5] Lifespan neuroimaging studies show that frontal gray matter volume predicts associative performance even more robustly than do MTL structures, highlighting the role of prefrontal–hippocampal interactions.[6] Because associative memory relies heavily on hippocampal integrity, it is particularly vulnerable to age-related

Address correspondence to: JeYoung Jung, PhD, School of Psychology, University of Nottingham, University Park, Nottingham NG7 2RD, UK. Email: jeyoung.jung@nottingham.ac.uk

[1] School of Psychology, University of Nottingham, Nottingham, UK;
[2] Department of Medical Sciences, Graduate School of Medicine, Korea University, Seoul, Republic of Korea;
[3] Department of Otorhinolaryngology-Head and Neck Surgery, Korea University Medical Center, Seoul, Republic of Korea;
[4] School of Medicine, University of Nottingham, Nottingham, UK;
[5] National Institute for Health and Care Research Biomedical Research Centre, University of Nottingham, Nottingham, UK; and
[6] Centre for Neurotechnology, Neuromodulation and Neurotherapeutics, University of Nottingham, Nottingham, UK

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decrease and is recognized as an early cognitive marker of Alzheimer's disease (AD).[7,8] Its dependence on neuromodulatory systems, especially norepinephrine (NE), further positions associative memory as a compelling target for interventions that modulate hippocampal–prefrontal networks.[9,10]

One promising intervention is vagus nerve stimulation (VNS). The vagus nerve is a key component of the parasympathetic nervous system and primarily comprises afferent fibers (~80%) projecting from the periphery to central neuromodulatory hubs, including the nucleus tractus solitarius, locus coeruleus (LC), and basal forebrain.[11,12] Through these pathways, vagal activation modulates neurotransmitter systems, most prominently NE and acetylcholine, linked to attention, emotional processing, and memory.[13] Although direct VNS has shown therapeutic efficacy for epilepsy and depression,[14–16] its invasive nature limits feasibility for cognitive research in healthy populations.

Growing recognition of the therapeutic potential of vagal modulation has stimulated increasing interest in noninvasive alternatives, most notably transcutaneous VNS (taVNS). Two principal modalities have emerged: electrical (e) VNS and more recently, ultrasound (u) VNS. eVNS is typically delivered auricularly at the cymba conchae of both ears using surface electrodes to stimulate afferent vagal fibers, thereby modulating LC–hippocampal and prefrontal circuits and providing a noninvasive route to influencing cognitive processes.[17,18] uVNS involves the external application of focused ultrasound pulses to the auricular branch of the vagus nerve, allowing deeper energy penetration and potentially more selective engagement of vagal fibers.[19]

A few behavioral studies have explored whether taVNS can enhance memory performance.[20] Early findings showed that a single session of taVNS improved face–name associative recall in older adults[21] and enhanced verbal order memory, but only for phonologically similar items.[22] These results align with broader evidence suggesting that eVNS can enhance memory.[23,24] Neuroimaging and neurophysiologic studies provide a mechanistic basis for these effects: eVNS modulates brainstem and limbic circuits, decreasing blood-oxygen-level-dependent signals in the LC, hippocampus, and parahippocampal gyrus, while increasing activation in the insula, anterior cingulate, inferior and superior frontal gyri, caudate, and putamen.[25,26] Recent work also shows that taVNS increases hippocampal connectivity with the prefrontal cortex and cingulate cortex in individuals with mild cognitive impairment and AD.[27] Combined with electroencephalogram, a study reported that taVNS enhanced P3b amplitudes,[28] and another study showed that taVNS attenuated alpha-oscillations power related to the LC-NE system.[29] In addition, eye-tracking studies have shown that taVNS modulates pupil diameter and task-evoked pupillary responses, which are widely considered a noninvasive proxy of LC activity and noradrenergic engagement in memory processes.[29,30] However, behavioral findings remain inconsistent: Some studies report no memory improvement in young adults,[31] and evidence for uVNS is largely preclinical,[32] with no human memory studies to date. Thus, despite strong theoretical and neurobiological rationale for LC-NE–driven modulation of hippocampal–prefrontal circuits, the reliability of taVNS-related associative memory enhancement remains unclear.

Here, we investigated the effects of both E-taVNS and U-taVNS on associative memory. Participants completed a video–word associative memory task before and after taVNS and before the experiment filled out questionnaires assessing anxiety, depression, and interoceptive perception. Each participant received either E-taVNS or U-taVNS in both active and sham conditions. We hypothesized that active E-taVNS and U-taVNS would enhance associative memory performance relative to both prestimulation and sham, reflecting our primary objective of testing within-subject active-vs-sham effects for each modality. Furthermore, we explored potential differences in efficacy between E-taVNS and U-taVNS.

METHODS AND MATERIALS

Participants

An a priori power analysis was conducted using G*Power 3.1.9.7[33] (Heinrich Heien, University Düsseldorf, Germany) for the *t*-test family (paired-samples design), reflecting the within-subject comparisons of interest (active vs sham and pre vs post stimulation). Assuming a moderate effect size (Cohen's $d = 0.65$), $\alpha = 0.05$, and power = 0.80, based on previous findings (Johnson and Steenbergen, 2022), the minimum required sample was 19 participants. The analysis was based on the primary within-subject contrasts (active vs sham; pre vs post), rather than comparisons of modalities. To ensure adequate statistical power and allow possible attrition, we aimed to recruit 30 participants per stimulation modality.

A total of 59 healthy adults (46 women, 13 men; mean = 23.6 years, SD = 2.88) participated in the study. Participants were randomly allocated to either the electrical stimulation group (E-taVNS; $n = 30$) or the ultrasound stimulation group (U-taVNS; $n = 29$).

All volunteers completed a taVNS safety questionnaire to confirm eligibility. Exclusion criteria included age >18 years; presence of any active implanted medical device (eg, pacemaker, cochlear implant); history of carotid atherosclerosis or cervical vagotomy; cardiovascular abnormalities (hypertension, hypotension, bradycardia, tachycardia); metallic implants in the head or neck; current or past neurologic or psychiatric disorders; fainting tendency; family history of epilepsy; current use of psychoactive medication (except hormonal contraception); and pregnancy.

All participants provided written informed consent for participation and publication of anonymized results. The study received ethical approval from the University of Nottingham Ethics Committee (F1619R) and adhered to the Declaration of Helsinki.

Transcutaneous Auricular VNS

taVNS was delivered using two modalities: electrical stimulation (Healaon Pro, Neurive Inc, Gimhae, Republic of Korea) and ultrasound stimulation (ZenBud, NeurGear Inc, Rochester, NY).

For E-taVNS, stimulation was applied bilaterally to the cymba conchae using conductive rubber electrodes. Parameters were set to 30 Hz at an intensity of level 5 (2.5 mA), and each stimulation block lasted 30 minutes. A continuous wind-like sound was played during both active and sham sessions to mask any device-related noise. For the E-taVNS sham condition, the device was positioned identically on the cymba conchae as in the active condition. To mimic the initial sensation of stimulation and maintain blinding, a brief stimulation period (~30 seconds at 2.5 mA) was delivered at the beginning of the session, after which stimulation was turned off (0 mA) for the remainder of the session.

For U-taVNS, stimulation was applied to the right cymba conchae. The ultrasound protocol used a center frequency of 5.3 MHz, a pulse repetition frequency of 41 Hz, a 50% duty cycle, a

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peak mechanical output of 0.081 W/cm^2, and an average pressure amplitude of 1.03 MPa. For the U-taVNS sham condition, a dedicated sham device was used that reproduced the same auditory buzzing sound as the active device but delivered no ultrasound output. This ensured that auditory cues associated with device operation were matched between active and sham sessions.

Participants were informed that two different stimulation protocols would be tested but were not told which session involved sham stimulation. At the end of the second session, participants were verbally asked whether they could distinguish between the two conditions. No participant reported being able to identify the active stimulation session, suggesting that the blinding procedure was effective.

Experimental Design and Procedures

Before the experiment, participants completed several questionnaires: the Beck Anxiety Inventory (BAI),[34] the Beck Depression Inventory (BDI),[35] and the Multidimensional Assessment of Interoceptive Awareness (MAIA-2).[36]

Each participant completed two sessions (active and sham), scheduled ≥five days apart, with stimulation order counterbalanced across participants. At the beginning of each session, participants completed an associative memory task comprising an encoding phase, a distraction task, and a retrieval phase (Fig. 1a). After the first completion of the task, participants received 30 minutes of taVNS. Immediately after stimulation, they completed the associative memory task again in the poststimulation phase. At the end of each session, participants completed a side-effects questionnaire assessing any mild adverse sensations (eg, headache, neck tension, nausea, tingling, or muscle contractions).

Associative Memory Task

We used the four dynamic videos and 369 words from a prior study.[37] Figure 1b illustrates the task procedure. The associative memory task had two phases: encoding and retrieval. During the encoding phase, each trial presented a dynamic video for 3000 milliseconds in the middle of the screen, followed by a word that was shown on the screen for 3000 milliseconds. In these 6000 milliseconds, participants were instructed to vividly associate the video with the word to memorize the video-word association. Next, they were asked to rate plausibility of the video-word association on a 1-to-4 scale (1 = very implausible, 4 = very plausible) using Z X C V keys. Between the encoding and retrieval phases, the distraction task was administered. In the number judgment task, a single number appeared at central fixation for 500 milliseconds, and participants were required to judge whether the number was odd or even through designated buttons for only 1500 milliseconds. This task lasted approximately two minutes. Stimuli were presented in randomized order. This task included 50 trials. During the retrieval phase, after each encoded word reappearing, participants had to select the associated video from the set of four videos stimuli, and then rated confidence (1 = not confident, 4 = very confident) using the Z X C V keys. There were strict time limits for retrieval phase; the participants had 4000 milliseconds to select one of the four videos, followed by 4000 milliseconds to report their confidence. The associative memory task had 48 trials, and

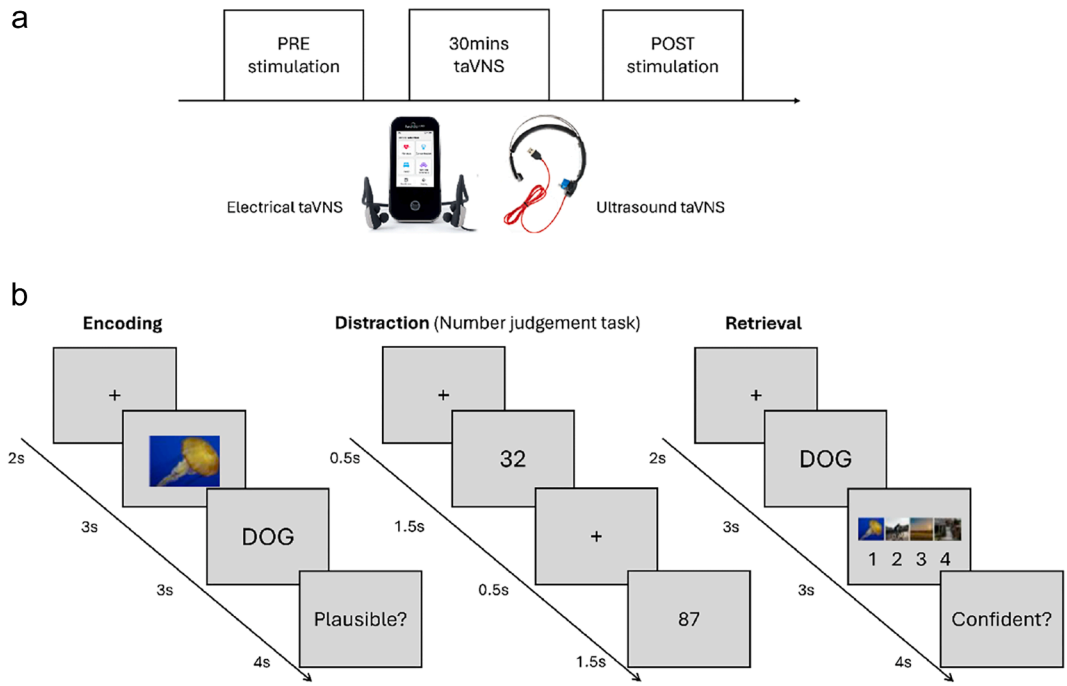


Figure 1. Experimental design and task procedure. a. Overview of the stimulation protocol. Participants completed a prestimulation session, followed by 30 minutes of taVNS, and a poststimulation session. Two stimulation modalities were used: E-taVNS and U-taVNS. b. Associative memory task. The task comprised encoding and retrieval phases separated by a distraction task. During encoding, a dynamic video was followed by a word, after which participants rated the plausibility of the video–word association on a four-point scale. Each trial began with a 2-second fixation. A number judgment distraction task (odd/even decision) was then administered. During retrieval, encoded words were presented, and participants selected the associated video from four options, followed by a confidence rating (four-point scale). The average delay between encoding and retrieval of a given item was approximately 7 minutes. [Color figure can be viewed at www.neuromodulationjournal.org]

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each trial started with 2000 milliseconds fixation. The average delay between encoding and retrieval of any given item was approximately 7 minutes, comprising the distractor task (~2 minutes) and the encoding or retrieval of other items (~5 minutes). The combination of this delay and intervening item load substantially exceeds the capacity and duration limits of working memory, making it unlikely that recall performance is driven by active maintenance.[38,39] Notably, however, given such memories can transform further[40–42] over the course of weeks, months, or years, our experiment cannot explain whether historic memories can benefit from taVNS. Stimulus items differed across sessions to avoid repetition or carry-over effects. The experiment was programmed and presented using PsychoPy (version 2024.2.4).

Statistical Analysis

Before conducting the central analyses, we cleaned the data using a series of exclusion criteria. First, trials when participants failed to choose a stimulus or provide a confidence rating were excluded (percentage of trials removed: 0.88%). Second, per-participant, reaction times for the memory responses that exceeded a ±2 SD threshold from the participant's mean reaction time were excluded (percentage of trials removed: 4.87%). Third, we removed participants with a memory performance <30%, reasoning that memory performance this low most probably reflects performing at chance level (25%). Notably, however, no participant fell below this threshold, meaning all participants were included in the final analyses.

Our central analyses investigated the impact of taVNS on the accuracy and speed of associative memory retrieval after a short delay, focusing specifically on the responses made when selecting the target stimulus from the line-up of videos. We conducted analyses separately for E-taVNS and U-taVNS. We isolated the effects of active taVNS using difference scores, computed by subtracting performance after sham stimulation from performance under active conditions for each participant at each timepoint. We then conducted a series of one-tailed *t*-tests to evaluate our hypotheses that taVNS improves memory performance and hastens memory responses. First, we used one-sample *t*-tests to evaluate whether pre- and/or poststimulation performance differed from zero (ie, performance after sham stimulation). We then directly contrasted pre- and poststimulation performance using paired-samples *t*-tests. For the reaction time analyses, we conducted a follow-up analysis to evaluate whether stimulation effects differed as a function of trial accuracy. We separated trials into correct and incorrect responses and repeated the planned contrasts previously described for correct and incorrect responses separately.

To assess adverse effects, χ^2 analyses were performed to compare the frequency of reported side effects in stimulation conditions (active vs sham). Statistical significance was defined as $p < 0.05$.

To assess how participant demographics and adverse effects affected any memory-related effects, we computed a "hastening index," which captured taVNS-driven changes in response times. This is formalized as:

$$Hastening\ Index = \left(active_{post} - active_{pre}\right) - \left(sham_{post} - sham_{pre}\right)$$

The hastening index was then correlated with participant age using Pearson's correlation, and contrasted in men and women, and in those who experienced skin irritation and those who did not using an independent samples *t*-test.

RESULTS

Demographic Results

To confirm baseline comparability of groups, independent-samples *t*-tests and χ^2 tests were performed on demographic and questionnaire scores (Table 1). No significant differences were observed between the E-taVNS and U-taVNS groups in sex distribution ($\chi^2 = 0.00$, $p = 1.000$) or age ($t = -0.28$, $p = 0.781$). Similarly, the groups did not differ in anxiety (BAI: $t = -0.47$, $p = 0.637$), depression (BDI: $t = 0.08$, $p = 0.939$), or interoceptive awareness as assessed by the MAIA ($t = -0.74$, $p = 0.463$).

Effects of taVNS on Associative Memory

Across conditions, participants on average were correct on 64.7% of trials (SD: 14.7%), with an average response time of 0.95 seconds (SD: 0.26 seconds). We found no significant differences in memory performance or reaction times between the U- and E-taVNS groups [accuracy: $t(57) = 1.359$, $p = 0.179$; RT: $t(57) = -1.698$, $p = 0.095$].

Neither E- nor U-taVNS had a significant impact on memory accuracy, with performance after active stimulation being no greater than sham stimulation [electrical: $t(29) = 0.694$, $p = 0.247$; ultrasound: $t(27) = -0.747$, $p = 0.769$; Fig. 2a] and no greater than performance before stimulation [electrical: $t(29) = 0.004$, $p = 0.498$; ultrasound: $t(27) = -0.133$, $p = 0.552$]. Bayesian analyses found anecdotal evidence for the null [electrical, active > sham: $BF_{01} = 2.01$; electrical, post > pre: $BF_{01} = 2.56$; ultrasound, active > sham: $BF_{01} = 1.87$; ultrasound, post > pre: $BF_{01} = 2.42$]. These results suggest that taVNS does not produce a significant enhancement in associative memory performance.

In contrast, however, we did observe a substantial hastening of memory responses after E-taVNS. Specifically, memory responses were faster after active E-taVNS than after sham stimulation [$t(29) = -2.999$, $p = 0.003$; Fig. 2b] and faster than responses before stimulation [$t(29) = -1.887$, $p = 0.035$]. This was a speeding of approximately 100 milliseconds (or 8.5% change) relative to sham. Although some may question whether the speeding of responses reflects a training effect, this idea is refuted by the observation that responses also were faster after active than after sham stimulation—a contrast in which both conditions incurred the same degree of training. The refutation of practice effects is reinforced by the absence of similar hastening effects after U-taVNS [active < sham: $t(27) = -0.391$, $p = 0.349$, $BF_{01} = 2.33$; post < pre:

Table 1. Participant Demographic and Characteristics.

Variable	E-taVNS	U-taVNS	Statistics (x^2/t)	*p*
n	30	29		
Sex (F/M)	23/7	23/6	0	1
Age (y)	23.57 ± 2.88	23.79 ± 3.31	−0.28	0.781
BAI	8.87 ± 6.66	9.66 ± 6.11	−0.47	0.637
BDI	7.00 ± 5.84	6.86 ± 7.68	0.08	0.939
MAIA	2.89 ± 0.63	3.00 ± 0.52	−0.74	0.463

F, female; M, male.

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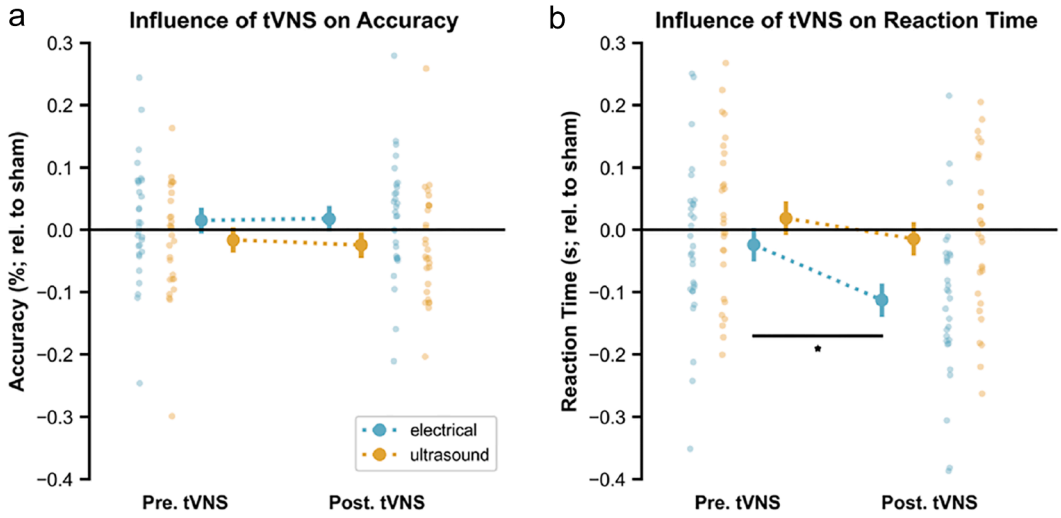


Figure 2. The influence of taVNS on behavioral performance during associative recall, relative to sham stimulation. a. The impact of E-taVNS (green) and U-taVNS (yellow) on recall accuracy. Large dots in the center of the plots represent the group, with accompanying errors describing SD from the mean. Smaller dots on the sides of the plot depict individual participant effects. b. The impact of E-taVNS (green) and U-taVNS (yellow), relative to sham stimulation, on response times. Plot details match panel a. *$p < 0.05$. [Color figure can be viewed at www.neuromodulationjournal.org]

$t(27) = -0.694$, $p = 0.247$, $BF_{01} = 2.00$]. Altogether, these results suggest that E-taVNS hastens memory-related responses.

One critical open question remains: Is the stimulation-induced hastening of responses something specific to successful retrieval, or is it a more general effect (eg, hastening sensorimotor processing)? To answer this, we split trials on the basis of retrieval success, reasoning that if taVNS aids memory, these effects should only be observable for successfully retrieved items. In contrast, if taVNS exerts a more general sensorimotor effect, it should be observable regardless of memory retrieval. When analyzing recalled items, we observed a significant hastening of reaction times for E-taVNS relative to sham and prestimulation measurements [active vs sham: $t(28) = -2.816$, $p = 0.004$; post < pre: $t(28) = -1.907$, $p = 0.033$; Fig. 3b]. For forgotten items, although there was a similar hastening for active to sham stimulation [$t(27) = -1.766$, $p = 0.044$], there was no hastening relative to prestimulation [$t(27) = -0.357$, $p = 0.362$]. Given that analysis of responses times between active and sham sessions, before stimulation, revealed a similar, trending hastening for forgotten items [$t(27) = -1.486$, $p = 0.074$], it would suggest that faster responses for forgotten items reflect a general hastening from sham to active sessions, rather than an effect that is specific to stimulation. Therefore, these results suggest that taVNS causes a hastening of response times that is tied to the success of recall and is not reflective of a more general cognitive/sensorimotor processes.

Aversive Effects Results

A χ^2 analysis was conducted to assess whether the incidence of aversive effects differed by stimulation modality (Table 2). Overall,

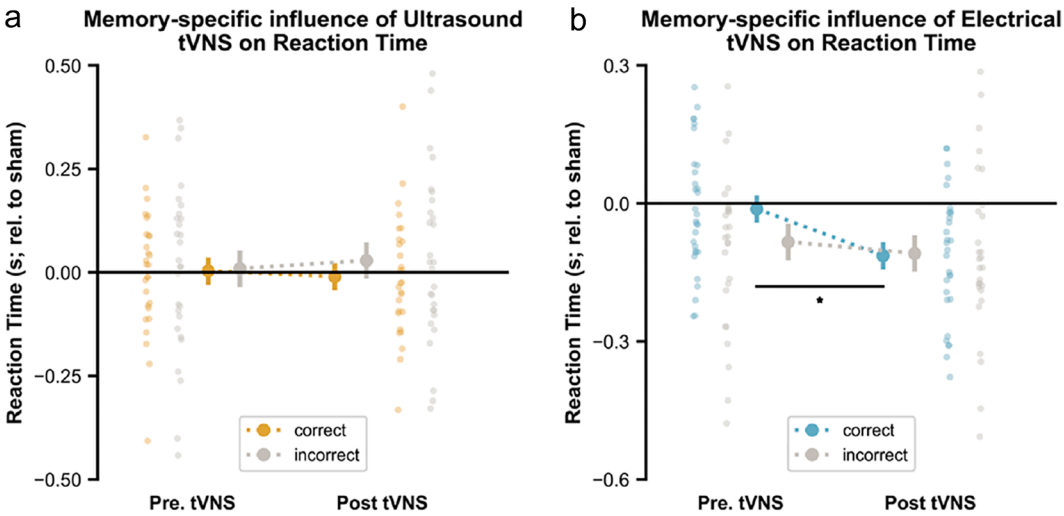


Figure 3. The influence of taVNS on reaction times as a function of recall success, relative to sham stimulation. a. The impact of U-taVNS on reaction time for correct (colored) and incorrect (gray) memory responses. Large dots in the center of the plots represent the group, with accompanying errors describing SD from the mean. Smaller dots on the sides of the plot depict individual participant effects. b. The impact of E-taVNS on response times as a function of recall success. Plot details match panel a. *$p < 0.05$. [Color figure can be viewed at www.neuromodulationjournal.org]

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Table 2. The Summary of Aversive Effects.

Aversive event	E-taVNS				U-taVNS				E-taVNS vs U-taVNS	
	Active	Sham	x^2	*p*	Active	Sham	x^2	*p*	x^2	*p*
Skin irritation	6	3	1.20	0.273	1	0	1.02	0.313	3.86	0.049*
Headache	1	2	0.35	0.552	0	0	-	-	0.98	0.321
Dizziness	0	2	2.08	0.150	1	0	1.02	0.313	1.05	0.305
Nausea or vomiting	0	1	1.02	0.313	0	0	-	-	-	-
Facial drooping	0	0	-	-	0	0	-	-	-	-
Hoarse voice	1	1	0.00	1.000	0	0	-	-	0.98	0.321
Pain or tingling in the throat or neck	1	2	0.35	0.552	0	0	-	-	0.98	0.321
Cough	1	0	1.02	0.313	0	0	-	-	0.98	0.321
Shortness of breath	0	0	-	-	0	0	-	-	0.98	0.321
Ear pain	5	3	1.48	0.224	3	3	0.00	1.000	0.5	0.478
Others	3	3	0.00	1.000	1	0	1.02	0.313	1	0.317

Values are presented as the number of participants reporting each adverse event. An em dash (—) indicates that statistical analysis was not performed because no events occurred.
E-taVNS, electrical transcutaneous auricular vagus nerve stimulation; U-taVNS, ultrasound transcutaneous auricular vagus nerve stimulation.
*$p < 0.05$.

no significant aversive effects were observed relative to sham in either modality. However, skin irritation was more frequently reported in the eVNS group than in the uVNS group, yielding a significant association between stimulation type and skin irritation ($\chi^2 = 3.86$, $p = 0.049$). This finding indicates that U-taVNS was associated with a lower incidence of skin irritation than was E-taVNS.

Potential Mediating Effects

Because the effects of taVNS can differ as a function of age and sex,[43,44] we evaluated whether participant demographics mediate the hastening effects we observed. To this end, we computed a hastening index that provided a single measure of the change in reaction time attributable to taVNS (Methods for derivation of the index) and correlated this with age in a Pearson correlation. We did not find a significant effect for either E-taVNS [$r = -0.081$, $p = 0.670$] or U-taVNS [$r = -0.131$, $p = 0.505$]. We then directly contrasted this hastening index in men and women in an independent samples *t*-test. For E-taVNS, we observed a trending effect whereby hastening was more pronounced in women than men [$t(28) = 1.923$, $p = 0.065$], mirroring the stronger physiologic responses to E-taVNS in women than men.[43] No sex effect was observed for U-taVNS [$t(28) = -1.052$, $p = 0.303$]. These results suggest there is a marginally larger hastening effect for women than for men but no change across age.

Given the significant differences in reported skin irritation between conditions, we investigated whether this could relate to the hastening effect. To this end, we conducted an independent samples *t*-test comparing those who reported skin irritation with those who did not. There was no significant difference in the hastening effect between those who reported skin irritation during active E-taVNS and those who did not [$t(28) = 1.634$, $p = 0.113$; notably, the direction of this *t*-value indicates that those who reported skin irritation showed a smaller hastening effect]. Similarly, there was no difference in hastening between those who reported skin irritation during sham E-taVNS and those who did not [$t(28) = 0.477$, $p = 0.637$]. We were unable to conduct an equivalent analysis for the U-taVNS cohort because only a single participant reported skin irritation. Altogether, this suggests that skin irritation did not affect the hastening effects previously reported.

DISCUSSION

There is growing interest in the therapeutic potential of taVNS in tackling memory issues across society. Here, we investigated its ability to enhance the cued recall of cross-modal associative memories, diverging from established approaches using recognition tests and unimodal memories. Contrary to recent studies of recognition memory, we did not find that taVNS enhanced cued recall accuracy. Intriguingly, however, we did observe a hastening of response times after taVNS that was specific to memories that were correctly recalled. This finding also contrasts with studies of recognition memory, which have often not observed taVNS-driven effects on reaction time. When combining our results with the existing literature, it suggests that taVNS exerts widespread and diverse effects over human memory.

Our results contribute to a growing, nuanced understanding of the impact of taVNS on the accuracy of longer-term (ie, beyond the timespan of working) memory. Several studies have shown that taVNS can enhance recognition performance for a variety of stimuli, including images,[45–47] faces,[21] and Kanji word pairs.[48] However, these effects are often conditional. For example, taVNS preferentially boosts recognition for negative emotional stimuli, with smaller effects for neutral stimuli.[45,46] Furthermore, the recognition-related benefits of taVNS are often observed for recollected stimuli but not those that are recognized through familiarity-based processes.[49] Our observation that taVNS does not enhance memory when participants must actively recall a paired associate suggests that the nature of the retrieval process also moderates the effectiveness of taVNS in modulating memory. Notably, our results align with the observation that taVNS fails to influence the free recall of verbal stimuli,[31] suggesting that the absence of memory effects is not attributable to particular stimuli or ways of administering a recall test.

Intriguingly, however, we did observe an effect of E-taVNS on reaction times when successfully recalling a memory. Several

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studies in other cognitive domains have indicated that taVNS can hasten response times, including auditory oddball detection[50] and conflict processing.[51] Although these results suggest that taVNS has a more general cognitive effect on response times, such effects cannot explain the reason we observe hastening of response times specifically for correctly recalled items. Instead, the hastening of responses times must be related to memory trace itself. Given we applied taVNS offline, before both the encoding and the retrieval stages, this could be related to how the memory is stored during encoding, facilitating access during subsequent retrieval, or could be due to a hastening of the memory search process during active recall. Future research could disentangle these ideas by exploring either whether our reaction time effects can continue to be observed after the effects of taVNS have washed out, which would favor an encoding-based account, or whether the effects are enhanced when taVNS is delivered immediately before/during retrieval, favoring a memory search account.

The neural mechanisms that underpin this memory-specific hastening of reaction times remain to be observed. A viable candidate is the formation and/or retrieval of associative links by the hippocampus. Indeed, a past study using the same paradigm used here has shown that successful recall is predicted by hippocampal engagement during the formation and retrieval of the associative links between stimuli.[37] Although hippocampal activity was not measured in the present study, taking this previous study in conjunction with the documented effects of taVNS on hippocampal function,[25–27] one could theorize that taVNS hastens the recall process by affecting hippocampal activity at critical junctures during either encoding or retrieval. An alternative candidate mechanism is alpha oscillations. taVNS has been shown to desynchronize alpha oscillations,[29] and such alpha desynchronization is known to facilitate memory formation and retrieval in the task we used here.[52,53] Future research that pairs taVNS with electrophysiology or neuroimaging may be key for delineating these theories.

The selective speeding of reaction times for correctly retrieved memories provides further insight into the mechanisms underlying taVNS effects. That this effect was specific to correctly recalled items (and absent for incorrect responses after accounting for pre-existing session differences) argues against a purely general account based on arousal, attention, or motor/decision processes, which would predict hastening regardless of recall success. Nevertheless, faster correct responses remain consistent with several memory-related mechanisms, including enhanced retrieval fluency, pattern completion, or memory access, among which the present data cannot definitively distinguish. This pattern also is consistent with recent pupillometry findings showing that taVNS modulates LC-linked arousal responses during cognitive tasks, supporting a role for the LC–NE system in enhancing retrieval efficiency rather than memory strength per se.[29,30]

We did not, however, observe any effects of U-taVNS on associative memory performance. This null result should be interpreted with caution owing to two key differences between our ultrasound and electrical protocols. First, although our E-taVNS was applied bilaterally, the ultrasound stimulation was unilateral. Given that most conventional protocols target the left ear to mitigate right-sided cardiac risks, it is possible that bilateral ultrasound application is necessary to reach the threshold for measurable cognitive effects. Second, the two modalities operate through distinct biophysical mechanisms. Although electrical stimulation triggers direct, current-induced depolarization, ultrasound uses mechanical pressure waves to deform neuronal membranes and gate mechanosensitive ion channels.[54] These fundamental differences suggest that ultrasound may require specific parameter optimization to effectively modulate the vagal afferent pathways involved in associative memory.

Several limitations warrant consideration when interpreting these results. First, although the sample size exceeded the a priori estimate based on effect sizes from previous E-taVNS studies, it may still have been underpowered to detect subtle effects. In particular, the power analysis was informed by E-taVNS literature, given there are no established studies evaluating the effects of U-taVNS on cognitive processing, especially memory. As such, the study may have been insufficiently powered to detect smaller or more variable effects for U-taVNS. Second, the retention interval in our associative memory task was brief (approximately 7 minutes), meaning that our findings speak specifically to short-delay cued recall rather than to longer-term memory consolidation. It remains unclear whether taVNS would similarly affect retrieval after longer delays (eg, weeks), after which the memory trace may have transformed.[40–42] Third, this study focused exclusively on short-term outcomes; consequently, it remains unclear whether these effects persist or accumulate through repeated sessions. Fourth, the lack of concurrent physiologic monitoring, such as heart rate variability or pupillometry biomarkers, precludes a direct validation of the underlying mechanistic pathways. Fifth, an important limitation concerns differences in stimulation protocols across modalities. E-taVNS was delivered bilaterally using established human protocols,[18,55] whereas U-taVNS was applied unilaterally using a single, device-constrained parameter set. The ultrasound protocol was informed by emerging neuromodulation principles, including the use of approximately 40-Hz stimulation linked to memory-related gamma activity[56,57] and parameters shown to induce neural activation in low-intensity focused ultrasound studies.[58] However, these parameters are not yet optimized for vagal stimulation in humans. Accordingly, this study does not constitute a fully controlled, parameter-matched comparison, and the absence of behavioral effects for U-taVNS should not be interpreted as evidence of ineffectiveness. Rather, differences in laterality, parameterization, and underlying mechanisms limit direct cross-modality conclusions and highlight the need for systematic optimization in future work. Last, stimulation intensity was not formally standardized across participants using an objective calibration procedure. As a result, interindividual variability in effective stimulation dose may have contributed to variability in behavioral responses and potentially reduced sensitivity to detect consistent effects. Future studies should consider incorporating individualized calibration procedures (eg, perceptual thresholds or physiologic markers) to better standardize stimulation intensity across participants.

In sum, although taVNS did not improve the accuracy of active recall, we find that electrical taVNS significantly hastens response times when correctly recalling memories after a short delay, suggesting that it may be a valuable tool for facilitating memory processes.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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Authorship Statements

Benjamin J. Griffiths and JeYoung Jung were responsible for the study conceptualization and writing the original draft of the manuscript. Benjamin J. Griffiths, JeYoung Jung, Marcus Kaiser, Hyuk Choi, and Jae-Jun Song were responsible for the study methods and for writing, reviewing, and editing the manuscript. Ziqi He, Irem Ciftepinar, and JeYoung Jung were responsible for the study investigation. All authors approved the final version of the manuscript.

Conflict of Interest

Hyuk Choi is a member of the Scientific Advisory Board of Neurive Inc (Gimhae, Republic of Korea). Jae-Jun Song is the chief executive officer of Neurive Inc. Marcus Kaiser is a member of the Scientific Advisory Board of NeurGear Inc (Rochester, NY). None of these companies had any involvement in the study design; data collection, analysis, or interpretation; manuscript preparation; or the decision to submit the work for publication. The remaining authors reported no conflict of interest.

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COMMENT

This study addresses a translational question in noninvasive VNS for memory improvement. A major strength of the work is its controlled design, including active and sham conditions and pre- and post-intervention assessments. The findings suggest that E-taVNS modulates retrieval speed for correct trials, whereas ultrasound-based stimulation did not produce measurable behavioral effects under the tested parameters. The manuscript also highlights a challenge for the field: Differences in stimulation parameters, laterality, and form factor can complicate direct modality comparisons. Overall, this work makes a useful contribution by refining expectations for behavioral effects of taVNS.

Gansheng Tan, MS
St. Louis, MO USA

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