

## Original Article

# Acoustic Voice Changes Induced by Transcutaneous Auricular Vagus Nerve Stimulation

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Seong Hee Choi<sup>1,2,3</sup>,  
Woo Sung Choi<sup>1,3</sup>,  
Geun Young Han<sup>1,3</sup>

<sup>1</sup>Department of Audiology & Speech-Language Pathology, Gyeongsan, Republic of Korea

<sup>2</sup>Institute of Biomimetic Sensory Control, Gyeongsan, Republic of Korea

<sup>3</sup>Catholic Hearing Voice Speech Center, Daegu Catholic University, Gyeongsan, Republic of Korea

**Purpose** Transcutaneous auricular vagus nerve stimulation (taVNS) is being increasingly applied in both clinical and experimental context; however, its effects on vocal function remain poorly understood. This study investigated the short-term effects of bilateral taVNS on voice production in healthy adults using acoustic perturbation, cepstral, spectral, and electroglottographic measures.

**Methods** A total of 39 healthy adults (8 males and 31 females; aged 21-36 years) without voice or neurological disorders were randomly allocated to either an active taVNS group (n = 20) or a sham group (n = 19) in a double-blind design. Participants received a single 30-minute session of bilateral taVNS (25 Hz, 200  $\mu$ s, 0.5 mA below pain threshold) or sham stimulation. Voice measures were obtained immediately before and after stimulation during sustained vowel phonation and connected speech. Pre- to post-stimulation changes were compared between groups, and subjective symptoms were monitored.

**Results** No consistent effects of taVNS were observed across acoustic, electroglottographic, or cepstral-spectral measures. Fundamental frequency, perturbation indices, vocal fold contact measures, and overall cepstral-spectral metrics remained stable following stimulation in both groups. A significant change was observed only in the low-to-high spectral ratio during sustained phonation in the taVNS group, with no corresponding effects observed during connected speech.

**Conclusions** Under the tested stimulation parameters, short-term bilateral taVNS did not produce robust or systematic changes in objective measures of voice production in healthy adults. These findings suggest that acute taVNS has limited immediate influence on vocal fold function and overall voice quality.

**Keywords** transcutaneous auricular vagus nerve stimulation (taVNS), voice acoustics, healthy adults, voice safety, cepstral measures, perturbation measures

## INTRODUCTION

The vagus nerve (VN; cranial nerve X) is a major parasympathetic pathway mediating bidirectional communication between the brainstem and visceral organs, including the heart, lungs, and gastrointestinal tract. Composed predominantly of afferent fibers that transmit sensory information to central autonomic centers and efferent fibers that convey motor signals to target organs, the VN plays

a critical role in autonomic regulation, neuroimmune modulation, and visceral sensory integration, thereby maintaining physiological homeostasis and facilitating brain-body interactions [1-4].

Vagus nerve stimulation (VNS) is a well-established neuromodulation technique used in epilepsy [5,6], depression [7], stroke [8,9], and other neurological or psychiatric disorders [10,11].

Conventional invasive VNS requires surgical im-

### Corresponding to

Seong Hee Choi  
Department of Audiology & Speech-Language Pathology, Research Institute of Biomimetic Sensory Control, and Catholic Hearing Voice Speech Center, Daegu Catholic University, 13-13, Hayang-ro, Hayang-eup, Gyeongsan, 38430, Republic of Korea  
TEL. +82-53-850-2542, FAX. +82-53-359-6780, E-mail. shgrace@cu.ac.kr

plantation of electrodes around the cervical vagus nerve, enabling direct modulation of central autonomic and neuromodulatory circuits; however, injury to the efferent branches of the vagus nerve can lead to adverse effects, including hoarseness, dyspnea, dysphagia, vocal fold paresis, and transient voice weakness, likely resulting from stimulation of the recurrent laryngeal nerve [12-14]. To overcome these limitations, noninvasive approaches such as transcutaneous vagus nerve stimulation (tVNS) have been developed.

In particular, transcutaneous auricular vagus nerve stimulation (taVNS) delivers electrical stimulation to the auricular branch of the VN via electrodes placed on the concha or tragus, activating the ipsilateral nucleus tractus solitarius and associated brainstem and forebrain regions [15,16]. Among auricular stimulation sites, the cymba conchae has been shown to elicit the strongest activation in vagus nerve-related brain regions, including the nucleus tractus solitarius and locus coeruleus [17].

taVNS protocols typically employ stimulation frequencies of 20-25 Hz, pulse widths of 100-500  $\mu$ s, and individualized current intensities set to approximately 80% of the sensory threshold or 0.5 mA below pain threshold [18,19], which are well-tolerated and minimize discomfort. These parameters are considered sufficient to engage vagal afferent pathways while reducing the risk of adverse effects, especially since taVNS avoids direct stimulation of laryngeal motor fibers.

Despite growing clinical and experimental use, empirical evidence regarding the effects of taVNS on voice production remains limited. Whereas invasive VNS has been associated with measurable voice changes, no studies examining taVNS generally report voice changes in acoustic voice outcomes. Furthermore, systematic investigations incorporating acoustic perturbation and cepstral, spectral, and electroglottographic measures in healthy adults are lacking, highlighting the need for comprehensive studies to evaluate the potential impact of taVNS on vocal function. In this study, we assessed these objective meas-

ures to determine whether short-term bilateral taVNS influences vocal function in healthy adults.

## METHODS

### Participants

The participants in this study were 39 healthy young adults (8 males and 31 females), aged 21-36 years, recruited from the Daegu and Gyeongbuk regions. The mean age of the participants was 23.52 years (SD = 2.68).

The inclusion criteria for the study were as follows:

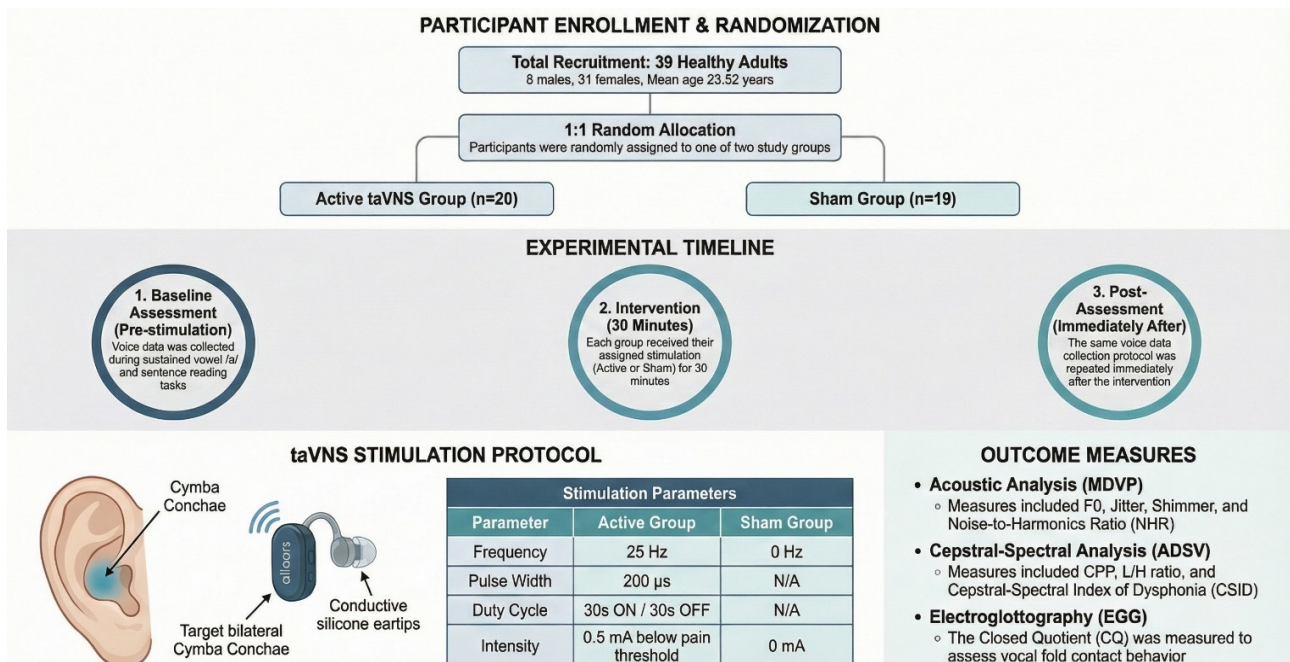
1. No history of upper respiratory infections or respiratory diseases in the month prior to the study.
  2. No current complaints related to voice, neurological issues, or ear (otologic) problems, and no history of these disorders within the previous three months.
  3. A Grade (G) score of 0 on the GRBAS scale, as assessed by a certified speech-language pathologist with over 10 years of clinical experience in voice disorders.
  4. Normal or corrected-to-normal vision and hearing.
  5. No prior diagnosis of speech or language disorders.
  6. No history of cardiovascular disease.
  7. Not currently pregnant.
  8. No facial or ear pain, and no recent history of ear trauma.
- Independent sample t-tests showed no statistically significant difference in age, sex, or acoustical test results between the taVNS group and the sham group except shimmer (all p-values > 0.05). The demographic characteristics of the participants are summarized in Table 1.

### Study design

A randomized, double-blind, sham-controlled design was employed. Participants were randomly assigned to either an active taVNS group (N = 20) or a sham stimulation group (N = 19)(Figure 1).

**Table 1.** Demographic Characteristics of Participants in the taVNS and Sham Groups

Variable	taVNS group (N=20)	Sham group (N=19)	statistics	p-value
Age	21.89±1.41	22.10 ± 3.54	1.464	.160
Sex, N (%)				.622
Male	4 (20%)	4 (21%)	.178	.163
Female	16 (80%)	15 (79 %)		
GRBAS				
Grade=0, N(%)	20(100%)	19(100%)		-
F0 (Hz)	197.7±46.8	198.2±54.7	-.264	.794
jitter (%)	1.21±.71	.82±.52	2.019	.061
shimmer (%)	3.52±1.56	2.53±.92	2.145	.046*
NHR	0.11 ± .029	0.11 ±.030	.536	.599
CQ (%)	46.50±2.54	44.48 ± 2.60	.256	.801
CPP_V (dB)	11.41±2.14	11.37 ±1.62	.266	.793
CPP_C (dB)	6.80±.95	7.40 ± .59	-1.486	.155
L/H ratio_V (dB)	31.46 ±4.12	32.15 ±5.73	-.431	.669
L/H ratio_C (dB)	32.02 ± 3.05	33.03 ± 2.88	-1.055	.298
CSID_V	10.86 ± 8.91	10.73 ± 6.80	-.047	.963
CSID_C	36.32 ± 8.70	35.63 ± 11.85	.394	.698

\**p* < .05**Figure 1.** Research study flowchart and outcome.

## Stimulation protocol

Stimulation was delivered using a wireless transcutaneous auricular vagus nerve stimulation (taVNS) de-

vice (allears; TODOC, Seoul, Republic of Korea), targeting the cymba conchae (Figure 1). The device consisted of an ear hook and ear tip fabricated from conductive silicone materials. Electrical stimulation was administered

at a frequency of 25 Hz [20-23], with a pulse width of 200  $\mu$ s [19,24]. An intermittent stimulation paradigm was employed, consisting of 30 s of stimulation (ON) followed by 30 s of rest (OFF), repeated continuously for a total session duration of 30 minutes.

The stimulation intensity was individually calibrated for each participant. The pain threshold was first determined, and the stimulation current was then set 0.5 mA below this threshold to ensure tolerability and minimize discomfort [19,25,26].

Participants assigned to the sham group underwent an identical procedure, including bilateral electrode placement on the cymba conchae and a 30-minute wearing duration. Bilateral stimulation of the cymba conchae was applied in the active condition, whereas the sham condition involved electrode placement without electrical current (0 mA) [19,27].

## Voice assessment

Voice assessments were conducted immediately before stimulation and 30 minutes after vagus nerve stimulation. Participants were instructed to sustain the vowel /a/ and read a standardized Korean passage titled "*Kaeul*" at their habitual pitch and loudness. For the sustained vowel phonation, each participant produced the vowel /a/ for approximately 5 seconds. To ensure signal stability, a 3-second segment was selected for analysis by excluding the initial and final 1-second intervals, which corresponded to phonation onset and offset. For the connected speech analysis, the second sentence of the "*Kaeul*" passage was selected to ensure natural and continuous speech production.

Acoustic analysis of sustained vowel phonation was performed using the Multi-Dimensional Voice Program (MDVP; Computerized Speech Lab, KayPENTAX Inc., Lincoln Park, NJ, USA) to extract perturbation measures, including fundamental frequency (F0), jitter (%), shimmer (%), and noise-to-harmonics ratio (NHR). Cepstral-spectral

measures were obtained using the Analysis of Dysphonia in Speech and Voice (ADSV) system, including cepstral peak prominence (CPP), low-to-high spectral ratio (L/H ratio), and the cepstral-spectral index of dysphonia (CSID).

For the connected speech analysis, silent intervals exceeding 200 ms, which could influence acoustic measurements, were manually removed before analysis. Cepstral-spectral measures (CPP, L/H ratio, and CSID) were subsequently extracted from the edited speech samples.

All recordings were conducted in a sound-treated booth with ambient noise levels below 35 dB SPL. Vocal signals were captured using a Shure SM48 dynamic microphone (Shure Inc., Chicago, IL, USA) positioned 10 cm from the participant's mouth at a 45° angle. The signals were digitized using the Computerized Speech Lab (CSL; KayPENTAX Inc., Lincoln Park, NJ, USA) at a sampling rate of 44.1 kHz with 16-bit quantization.

Electroglottographic recordings were obtained during the sustained /a/ phonation to derive the closed quotient (CQ) as an index of vocal fold contact behavior.

## Statistical analysis

Statistical analyses were conducted using IBM SPSS Statistics (Version 30.0; IBM Corp., Armonk, NY, USA). Baseline homogeneity between the taVNS and sham groups was assessed using independent-samples *t*-tests on pre-intervention values for each acoustic variable.

To examine the effects of stimulation, a two-way mixed-design analysis of variance (ANOVA) was performed for each outcome measure, with group (taVNS vs. sham) as the between-subjects factor and time (pre- vs. post-intervention) as the within-subjects factor. When a significant group  $\times$  time interaction was observed, post hoc paired-samples *t*-tests were conducted to evaluate within-group changes across time points.

All statistical tests were two-tailed, and the level of statistical significance was set at  $p < .05$ .

## RESULTS

### Perturbation measures

Table 2 presents the means and standard deviations of fundamental frequency (F0), jitter, shimmer, noise-to-harmonics ratio (NHR), CQ, CPP\_V, CPP, C, L/H ratio\_V, L/H ratio\_C., CSID\_V, and CSID\_C for the taVNS and sham groups before and after stimulation. Overall, no significant differences were observed between groups for F0 or perturbation measures following stimulation. For F0, the group  $\times$  time interaction was not significant ( $F(1, 37) = 0.149, p = .702, \text{partial } \eta^2 = .004$ ). Similarly, neither the main effect of time ( $F(1, 37) = 0.158, p =$

$.693, \text{partial } \eta^2 = .004$ ) nor the main effect of group ( $F(1, 37) = 0.008, p = .931, \text{partial } \eta^2 = .000$ ) showed significant results.

For jitter, the group  $\times$  time interaction was also not significant ( $F(1, 37) = 0.339, p = .564, \text{partial } \eta^2 = .009$ ), and no significant main effect of time was observed ( $F(1, 37) = 1.259, p = .269, \text{partial } \eta^2 = .033$ ). Although a significant main effect of group was initially detected ( $F(1, 37) = 5.023, p = .023, \text{partial } \eta^2 = .132$ ), this effect did not remain after adjusting for baseline values. A one-way ANCOVA with pre-treatment jitter as a covariate revealed no significant post-treatment difference between groups ( $F(1, 36) = 1.888, p = .178$ ), indicating that baseline performance accounted for the observed group effect.

**Table 2.** Means and Standard Deviations of Acoustic Perturbation, CQ, and Cepstral & Spectral Measures before and after taVNS and Sham Stimulation

Measure	Group	Pre-stimulation (Mean $\pm$ SD)	Post-stimulation (Mean $\pm$ SD)	Group $\times$ Time F (p)
F0 (Hz)	taVNS	197.7 $\pm$ 46.8	194.50 $\pm$ 50.3	0.149 (.702)
	Sham	198.2 $\pm$ 54.7	197.66 $\pm$ 47.1	
Jitter (%)	taVNS	1.21 $\pm$ 0.71	1.36 $\pm$ 0.82	0.339 (.564)
	Sham	0.82 $\pm$ 0.52	0.87 $\pm$ 0.37	
Shimmer (%)	taVNS	3.52 $\pm$ 1.56	3.24 $\pm$ 1.32	4.814 (.035*)
	Sham	2.53 $\pm$ 0.92	3.03 $\pm$ 0.96	
NHR	taVNS	0.11 $\pm$ 0.29	0.12 $\pm$ 0.03	0.257 (.615)
	Sham	0.11 $\pm$ 0.30	0.12 $\pm$ 0.02	
CQ (%)	taVNS	46.50 $\pm$ 2.54	46.28 $\pm$ 3.13	1.407 (.243)
	Sham	44.48 $\pm$ 2.60	45.64 $\pm$ 4.15	
CPP_V (dB)	taVNS	11.41 $\pm$ 2.14	11.46 $\pm$ 1.88	0.544 (.465)
	Sham	11.37 $\pm$ 1.62	11.64 $\pm$ 1.87	
L/H ratio_V (dB)	taVNS	31.46 $\pm$ 4.12	30.32 $\pm$ 4.16	6.994 (.012*)
	Sham	32.15 $\pm$ 5.73	32.55 $\pm$ 6.47	
CSID_V	taVNS	10.86 $\pm$ 8.91	12.14 $\pm$ 10.55	0.084 (.774)
	Sham	10.73 $\pm$ 6.80	13.25 $\pm$ 16.93	
CPP_C (dB)	taVNS	6.80 $\pm$ 0.95	6.78 $\pm$ 0.67	0.161 (.691)
	Sham	7.40 $\pm$ 0.59	7.46 $\pm$ 0.75	
L/Hratio_C (dB)	taVNS	32.02 $\pm$ 0.66	31.39 $\pm$ 0.76	1.893 (.177)
	Sham	33.03 $\pm$ 0.68	33.06 $\pm$ 0.78	
CSID_C	taVNS	36.32 $\pm$ 8.70	37.22 $\pm$ 3.50	3.359 (.075)
	Sham	35.63 $\pm$ 11.85	34.94 $\pm$ 8.02	

Values are presented as mean  $\pm$  standard deviation.

Group  $\times$  time effects were tested using two-way mixed-design ANOVA.

F0 = fundamental frequency; NHR = noise-to-harmonics ratio; CPP\_V: CPP measured in the /a/ vowel, CPP\_C: CPP measured during connected speech, L/Hratio\_V; L/Hratio measured in the /a/ vowel, L/Hratio\_C: L/Hratio measured during connected speech, taVNS = transcutaneous auricular vagus nerve stimulation. \* $p < .05$

For percent shimmer, a significant group  $\times$  time interaction was found ( $F(1, 37) = 4.814, p = .035$ ). However, post hoc paired-samples  $t$ -tests revealed no significant pre- to post-treatment changes within either group. The sham group exhibited a marginal, non-significant increase in shimmer ( $t(18) = -2.044, p = .056$ ), whereas the taVNS group exhibited a slight decrease following stimulation, which was also not statistically significant ( $t(19) = 1.091, p = .289$ ). Thus, despite the significant group  $\times$  time interaction, within-group analyses did not demonstrate reliable treatment-related effects.

For NHR, neither the main effect of time ( $F(1, 37) = 0.542, p = .466, \text{partial } \eta^2 = .014$ ) nor the main effect of group ( $F(1, 37) = 0.021, p = .886, \text{partial } \eta^2 = .001$ ) was significant. The group  $\times$  time interaction was also non-significant ( $F(1, 37) = 0.257, p = .615, \text{partial } \eta^2 = .007$ ).

### Electroglottographic measures

For closed quotient (CQ) during sustained /a/ vowel phonation, no significant group  $\times$  time interaction was observed ( $F(1, 37) = 1.407, p = .243, \text{partial } \eta^2 = .037$ ). Neither the main effect of time ( $F(1, 37) = 0.699, p = .419, \text{partial } \eta^2 = .018$ ) nor the main effect of group ( $F(1, 37) = 2.564, p = .118, \text{partial } \eta^2 = .065$ ) was statistically significant. These results suggest that the vocal fold contact behavior remained consistent over time and across different groups.

### Cepstral and spectral measures

In the analysis of cepstral peak prominence (CPP) during sustained /a/ vowel phonation, no significant group  $\times$  time interaction was detected ( $F(1, 37) = 0.544, p = .465, \text{partial } \eta^2 = .014$ ). Additionally, neither the main effect of group ( $F(1, 37) = 0.015, p = .903, \text{partial } \eta^2 = .000$ ) nor the main effect of time ( $F(1, 37) = 1.001, p = .324, \text{partial } \eta^2 = .026$ ) was significant. During sentence read-

ing, CPP revealed no significant main effect of time ( $F(1, 37) = 0.025, p = .875, \text{partial } \eta^2 = .001$ ) nor a group  $\times$  time interaction ( $F(1, 37) = 0.161, p = .691, \text{partial } \eta^2 = .004$ ). Although a significant main effect of group was observed ( $F(1, 37) = 8.936, p = .005, \text{partial } \eta^2 = .195$ ), this difference did not persist after adjusting for baseline CPP using ANCOVA ( $F(1, 36) = 2.822, p = .102, \text{partial } \eta^2 = .073$ ). This indicates that the initial group effect was likely due to pre-existing variability rather than treatment-related changes.

For the low-to-high spectral ratio (L/H ratio) during sustained vowel phonation, the group  $\times$  time interaction reached significance ( $F(1, 37) = 6.994, p = .012, \text{partial } \eta^2 = .159$ ). The post hoc paired-samples  $t$ -tests demonstrated significant pre- to post-treatment changes in taVNS group ( $t(19) = 3.687, p = .002$ ), while no change was observed in sham group ( $t(18) = -.579, p = .570$ ). In contrast, the L/H ratio in connected speech, the L/H ratio exhibited no significant main effects for time ( $F(1, 37) = .955, p = .335, \text{partial } \eta^2 = .025$ ) or group ( $F(1, 37) = 1.152, p = .290, \text{partial } \eta^2 = .030$ ), and no significant group  $\times$  time interaction ( $F(1, 37) = 1.893, p = .177, \text{partial } \eta^2 = .049$ ).

Regarding the Cepstral-Spectral Index of Dysphonia (CSID) during sustained vowel phonation, neither the main effect of time ( $F(1, 37) = 0.791, p = .380, \text{partial } \eta^2 = .021$ ) nor the main effect of group ( $F(1, 37) = 0.027, p = .870, \text{partial } \eta^2 = .001$ ) was significant. The group  $\times$  time interaction was also non-significant ( $F(1, 37) = 0.084, p = .774, \text{partial } \eta^2 = .002$ ). In connected speech, there were again no significant main effects for time ( $F(1, 37) = 0.023, p = .880, \text{partial } \eta^2 = .001$ ) or group ( $F(1, 37) = 1.940, p = .172, \text{partial } \eta^2 = .050$ ). The group  $\times$  time interaction likewise did not reach statistical significance either ( $F(1, 37) = 3.359, p = .075, \text{partial } \eta^2 = .083$ ).

Taken together, these results indicate that most perturbation, electroglottographic, and cepstral-spectral measures showed no reliable changes over time or differences

between the taVNS and sham groups, with the exception of the task-specific reduction in the low-to-high spectral ratio observed during sustained /a/ phonation in the taVNS group.

## Discussion and conclusions

The present study investigated the short-term effects of bilateral transcutaneous auricular vagus nerve stimulation (taVNS) on acoustic, electroglottographic, and cepstral-spectral voice measures in healthy adults. Overall, the findings indicate that, under the parameters tested, taVNS, did not produce widespread changes in vocal function; however, task-specific alterations were observed in spectral energy distribution, as evidenced by a reduction in the low-to-high (L/H) spectral ratio during sustained /a/ vowel phonation. Across perturbation measures, including jitter and noise-to-harmonics ratio (NHR), no consistent group  $\times$  time interactions were identified. Similarly, fundamental frequency (F0) remained stable following stimulation. These results suggest that short-term taVNS does not substantially affect vocal fold vibratory stability or cycle-to-cycle variability in healthy speakers. Given that perturbation measures are particularly sensitive to pathological voice conditions rather than subtle physiological modulation, the absence of significant changes may reflect the robustness of normal phonatory control in healthy individuals. Electroglottographic analysis further demonstrated that closed quotient (CQ) during sustained vowel phonation did not change significantly over time or between groups. This finding indicates that taVNS did not alter vocal fold contact patterns or the overall balance between the open and closed phases of the glottal cycle. From a clinical perspective, the stability of CQ suggests that taVNS does not compromise glottal closure or induce phonatory inefficiency in healthy adults.

Cepstral measures, including cepstral peak prominence (CPP) and the Cepstral-Spectral Index of Dysphonia

(CSID), also remained largely unchanged following stimulation. Although a significant main effect of group was initially observed for CPP during connected speech, this effect was no longer significant after controlling for baseline values, indicating that pre-existing interindividual variability rather than stimulation-related effects accounted for the difference. CPPS is also considered a robust acoustic metric for quantifying dysphonic voice characteristics in sustained phonation as well as connected speech [28,29]. The cepstral/spectral index of dysphonia (CSID) is a composite metric calculated via multiple linear regression of cepstral peak prominence (CPP), the low-to-high (L/H) ratio, and the standard deviation of the L/H ratio [30]. These findings collectively suggest that taVNS does not exert a measurable influence on overall voice quality or dysphonia-related indices in healthy speakers.

In contrast, the most notable finding of this study was a significant reduction in the L/H ratio following taVNS, observed exclusively during sustained /a/ vowel phonation. A lower L/H ratio indicates a relative increase in high-frequency spectral energy, as this measure reflects the distribution of spectral energy between frequencies below 4000 Hz and those above 4000 Hz. Previous studies have associated increased high-frequency energy with greater spectral turbulence or breathiness in the voice signal [31]. Although reduced L/H ratios are commonly reported in pathological or aging voices, often in the context of glottal insufficiency, such interpretations should be approached with caution in the present study, as all participants exhibited normal voice function. Rather than reflecting a degradation in voice quality, the observed decrease in L/H ratio may represent subtle modulation of phonatory aerodynamics or laryngeal muscle regulation associated with vagal nerve stimulation. The vagus nerve plays a central role in autonomic regulation [1,2], and taVNS has been shown to influence parasympathetic activity. It is therefore plausible that taVNS induced minor adjustments in respiratory-phonatory coordination or airflow

regulation, leading to a relative increase in high-frequency spectral components without affecting vocal fold contact or vibratory stability.

Clinically, these findings suggest that taVNS can be applied without adverse effects on core vocal parameters in healthy adults, supporting the safety of taVNS in healthy adults and fulfill a necessary prerequisite for conducting subsequent clinical trials.

Moreover, the selective sensitivity of the L/H ratio to taVNS highlights the potential utility of spectral measures as early indicators of neuromodulatory effects on voice production. This may have implications for future research exploring taVNS as an adjunctive intervention for voice disorders associated with autonomic imbalance, excessive laryngeal tension, or aging-related changes.

Long-term follow-up studies of VNS in patients with epilepsy have demonstrated its effectiveness in reducing seizure frequency. However, the most frequently reported adverse effects included coughing, pitch breaks, and mild intermittent shortness of breath, observed in approximately one-third of patients. Videolaryngoscopic assessments revealed secondary supraglottic muscle tension and hyperfunction, accompanied by reduced mobility of the ipsilateral vocal fold, although no cases of aspiration were noted. These findings indicate that laryngeal side effects—particularly hoarseness and altered vocal fold motion—are among the most commonly reported consequences of VNS implantation [32]. Similarly, Shaffer et al. (2022) reported significant subjective differences in vocal quality and videolaryngoscopic findings between VNS-implanted patients and age- and sex-matched controls. During VNS activation, the most frequently observed changes included increased vocal fold tension, supraglottic muscular hyperfunction, and reduced vocal fold mobility. Notably, two of ten patients exhibited immobile left vocal folds even in the absence of active stimulation. Although maximum phonation time was generally reduced in the study group, this decrease did not reach statistical significance. Furthermore, abnormal laryngeal

electromyographic findings were observed in six of ten patients, including large-amplitude multiphasic motor unit potentials and reduced recruitment [33].

Electromyographic investigation also reported that six of the 13 participants exhibited significant vocal fold mobility impairments at two weeks postoperatively. Preoperative laryngeal electromyography identified notable abnormalities in five participants, all of whom showed persistent left vocal fold paresis at three months following surgery. The authors concluded that perioperative vocal fold paresis occurs in approximately 50% of patients [34].

Unlike invasive VNS, which directly stimulates motor fibers causing vocal fold paresis or hoarseness, taVNS targets the afferent auricular branch. The fact that CSID (an overall index of dysphonia) did not change despite the L/H ratio shift confirms that taVNS does not induce voice quality degradation. Instead, the increase in high-frequency energy may reflect the activation of the brainstem's autonomic and sensorimotor circuits, which subtly influence the laryngeal system without the "collateral damage" seen in invasive procedures.

This finding is clinically significant for researchers and practitioners. It demonstrates that taVNS is not purely 'neutral' regarding the voice; it can induce measurable changes in spectral distribution.

Building on these findings, the present results suggest that taVNS exerts a modulatory—rather than disruptive—effect on vocal function. Unlike invasive VNS, which can compromise phonatory control through unintended activation of efferent motor fibers, taVNS selectively engages afferent pathways of the auricular branch of the vagus nerve. The absence of significant changes in CSID, despite a clear shift in the low-to-high (L/H) spectral energy ratio, indicates that taVNS does not deteriorate perceived voice quality or induce dysphonia. Instead, it appears to subtly reshape the acoustic structure of the voice while preserving overall vocal stability. The task-specific nature of the L/H ratio change, observed only during sustained vowel phonation but not during connected speech,

may be attributable to differences in physiological demands and acoustic stability across speech tasks. Sustained vowel production provides a relatively steady-state phonatory condition with minimal articulatory and prosodic variability, making it more sensitive to subtle changes in laryngeal or aerodynamic control. In contrast, connected speech involves dynamic modulation of pitch, intensity, articulation, and respiratory patterns, which may obscure or compensate for small stimulation-related effects on spectral energy distribution. Consequently, any minor taVNS-induced modulation of phonatory aerodynamics or airflow regulation may be detectable under controlled vowel conditions but diluted within the greater variability inherent to connected speech. Additionally, cepstral-spectral measures obtained from connected speech reflect an integration of supralaryngeal articulation and linguistic prosody, which may reduce their specificity to laryngeal-level changes. Previous studies have demonstrated that sustained vowel tasks are particularly sensitive to alterations in glottal configuration and airflow, whereas connected speech measures tend to capture more global aspects of voice quality. Thus, the absence of significant changes in the L/H ratio during connected speech suggests that taVNS-related effects, if present, are subtle and task dependent, rather than indicative of generalized alterations in functional voice production. Importantly, the lack of concurrent changes in perturbation measures, closed quotient, or cepstral indices further supports the interpretation that the observed vowel-specific L/H ratio change reflects a limited and context-dependent modulation rather than a clinically meaningful deterioration of voice quality. These findings underscore the importance of task selection when evaluating neuromodulatory effects on voice and suggest that sustained phonation may be more sensitive than connected speech for detecting subtle autonomic or neuromotor influences.

The observed increase in high-frequency energy may reflect enhanced central regulation of laryngeal function mediated by brainstem nuclei, such as the nucleus trac-

tus solitarius, and their downstream connections to autonomic and sensorimotor networks. These circuits are known to influence respiratory-laryngeal coordination and fine motor control of phonation. Notably, such neuromodulatory effects appear to occur without the collateral damage commonly associated with invasive neuromodulation techniques, highlighting the favorable safety profile of taVNS with respect to vocal function.

From a clinical and translational perspective, these findings challenge the notion that taVNS is acoustically neutral. Instead, taVNS can induce measurable and systematic changes in spectral energy distribution, suggesting a capacity to fine-tune vocal output. Future studies should investigate whether these spectral changes translate into perceptual or functional benefits in patient populations and whether they can be optimized through parameter-specific taVNS protocols. Longitudinal designs and multimodal assessments combining acoustic, perceptual, and neurophysiological measures will be essential to clarify the mechanisms underlying taVNS-induced vocal modulation and to establish its therapeutic relevance in voice and neurorehabilitation research.

Several limitations should be acknowledged. First, the study examined only the immediate effects of a single taVNS session; cumulative or long-term effects remain unknown. Second, the sample consisted exclusively of healthy adults, limiting the generalizability of the findings to clinical populations. Finally, perceptual voice ratings and aerodynamic measures were not included, which may have provided complementary insights into the functional significance of the observed spectral changes.

In conclusion, short-term bilateral taVNS did not produce significant changes in perturbation, electroglottographic, or cepstral indices of voice quality in healthy adults, but it did induce selective changes in spectral energy distribution as reflected by reduced L/H ratios. These results suggest that taVNS exerts subtle, non-disruptive effects on voice production and support further investigation into its potential role in voice therapy and neuro-

modulation-based interventions.

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## REFERENCES

1. Yuan H, Silberstein SD. Vagus nerve and vagus nerve stimulation, a comprehensive review: Part I. Headache. 2016;56(1):71-8.
2. Yuan H, Silberstein SD. Vagus nerve and vagus nerve stimulation, a comprehensive review: Part II. Headache. 2016;56(2):259-66.
3. Berthoud HR, Neuhuber WL. Functional and chemical anatomy of the afferent vagal system. *Auton Neurosci*. 2000;85(1-3):1-17.
4. Austelle CW, Cox SS, Wills KE, Badran BW. Vagus nerve stimulation (VNS): recent advances and future directions. *Clin Auton Res*. 2024;34(6):529-47.
5. Vagus Nerve Stimulation Study Group. A randomized controlled trial of chronic vagus nerve stimulation for treatment of medically intractable seizures. *Neurology*. 1995;45(2):224-30.
6. Uthman BM, Wilder BJ, Penry JK, Dean C, Ramsay RE, Reid SA, Hammond EJ, Tarver WB, Wernicke JF. Treatment of epilepsy by stimulation of the vagus nerve. *Neurology*. 1993;43(7):1338-45.
7. Rush AJ, Marangell LB, Sackeim HA, George MS, Brannan SK, Davis SM, Howland R, Kling MA, Rittberg BR, Burke WJ, Rapaport MH, Zajecka J, Nierenberg AA, Husain MM, Ginsberg D, Cooke RG. Vagus nerve stimulation for treatment-resistant depression: a randomized, controlled acute phase trial. *Biol Psychiatry*. 2005;58(5): 347-54.
8. Zhang C, Wang JX, Sun, FH, Xie YJ, Ou X, Yang SB. The effect of VNS on the rehabilitation of stroke: a meta-analysis of randomized controlled studies. *J Clinical Neuroscience*. 2020;81:421-25.
9. Dawson J, Liu CY, Francisco GE, Cramer SC, Wolf SL, Dixit A, Alexander J, Ali R, Brown BL, Feng W, DeMark L, Hochberg LR, Kautz SA, Majid A, O'Dell MW, Pierce D, Prudente CN, Redgrave J, Turner DL, Engineer ND, Kimberley TJ. Vagus nerve stimulation paired with rehabilitation for upper limb motor function after ischaemic stroke (VNS-REHAB): a randomised, blinded, pivotal, device trial. *Lancet*. 2021;397(10234):1545-53.
10. Vargas-Caballero M, Warming H, Walker R, Holmes C, Cruickshank G, Patel B. Vagus nerve stimulation as a potential therapy in early Alzheimer's disease: a review. *Front Hum Neurosci*. 2022;16:866434.
11. Merrill CA, Jonsson MA, Minthon L, Ejnell H, Silander HC, Blennow K, Karlsson M, Nordlund A, Rolstad S, Warkentin S, Ben-Menachem E, Sjögren MJC. Vagus nerve stimulation in patients with Alzheimer's disease: additional follow-up results of a pilot study through 1 year. *J Clin Psychiatry*. 2006;67(8):1171-8.
12. Ben-Menachem E. Vagus nerve stimulation, side effects, and long-term safety. *J Clin Neurophysiol*. 2001;18(5): 415-8.
13. Ben-Menachem E. Vagus-nerve stimulation for the treatment of epilepsy. *Lancet Neurol*. 2002;1(8):477-82.
14. Handforth A, DeGiorgio CM, Schachter SC, Uthman BM, Naritoku DK, Tecoma ES, Henry TR, Collins SD, Vaughn BV, Gilmartin RC, Labar DR, Morris 3<sup>rd</sup> GL, Salinsky MC, Osorio I, Ristanovic RK, Labiner DM, Jones JC, Murphy JV, Ney GC, Wheless JW. Vagus nerve stimulation therapy for partial-onset seizures: a randomized active-control trial. *Neurology*. 1998;51(1):48-55.
15. Ben-Menachem E, Revesz D, Simon BJ, Silberstein S. Surgically implanted and non-invasive vagus nerve stimulation: a review of efficacy, safety and tolerability. *Eur J Neurol*. 2015;22(9):1260-8.
16. van Leusden JWR, Sellaro R, Colzato LS. Transcutaneous vagal nerve stimulation (tvNS): a new neuromodulation tool in healthy humans? *Front Psychol*. 2015;6:102.
17. Badran BW, Brown JC, Dowdle LT, Mithoefer OJ, LaBate NT, Coatsworth J, George MS. *Tragus or cymba conchae?* Investigating the anatomical foundation of transcutaneous auricular vagus nerve stimulation (taVNS). *Brain Stimul*. 2018;11(4):947-8.
18. Yap JYY, Keatch C, Lambert E, Woods W, Stoddart PR, Kameneva T. Critical Review of Transcutaneous Vagus Nerve Stimulation: Challenges for Translation to Clinical Practice. *Frontiers in Neuroscience*. 2020;14:284.
19. Kang YM, Choi SH, Lee K, Choi CC. Effects of transcutaneous auricular vagus nerve stimulation (taVNS) on verbal working memory, word fluency, and verbal episodic memory in healthy young adults. *Commun Sci Disord*. 2025;30(2):322-34.
20. Horinouchi T, Nezu T, Saita K, Date S, Kurumadani H, Maruyama H, Kirimoto H. Transcutaneous auricular vagus nerve stimulation enhances short-latency afferent inhibition via central cholinergic system activation. *Sci Rep*. 2024;14(1):11224.
21. Lakshmi SS, Srinivasan V, Suganthirababu P, Kumar P, Dhanusia S, Kumaresan A, Vishnuram S. Effect of vagal nerve stimulation on cognitive impairment among sub-

- jects with anterior cerebral artery syndrome: a pilot study. *Indian J Physiother Occup Ther.* 2024;18(5):151-6.
22. Zhang H, Guo Z, Qu Y, Zhao Y, Yang Y, Du J, Yang C. Cognitive function and brain activation before and after transcutaneous cervical vagus nerve stimulation in healthy adults: a concurrent tcVNS-fMRI study. *Front Psychol.* 2022;13:1003411.
  23. Baig SS, Falidas K, Laud PJ, Snowdon N, Farooq MU, Ali A, Majid A, Redgrave JN. Transcutaneous auricular vagus nerve stimulation with upper limb repetitive task practice may improve sensory recovery in chronic stroke. *J Stroke Cerebrovasc Dis.* 2019;28(12):104348.
  24. Thakkar VJ, Richardson ZA, Dang A, Centanni TM. The effect of non-invasive vagus nerve stimulation on memory recall in reading: a pilot study. *Behav Brain Res.* 2023; 438:114164.
  25. Chang JL, Coggins AN, Saul M, Paget-Blanc A, Straka M, Wright J, Datta-Chaudhuri T, Zanos S, Volpe BT. Transcutaneous auricular vagus nerve stimulation (tAVNS) delivered during upper limb interactive robotic training demonstrates novel antagonist control for reaching movements following stroke. *Front Neurosci.* 2021; 15:767302.
  26. Mertens A, Gadeyne S, Lescrauwaet E, Carrette E, Meurs A, De Herdt V, Dewaele F, Raedt R, Miatton M, Boon P, Vonck K. The potential of invasive and non-invasive vagus nerve stimulation to improve verbal memory performance in epilepsy patients. *Sci Rep.* 2022;12(1):1984.
  27. Kaniusas E, Kampusch S, Tittgemeyer M, Panetsos F, Gines RF, Papa M, Kiss A, Podesser B, Cassara AM, Tanghe E, Samoudi AM, Tarnaud T, Joseph W, Marozas V, Lukosevicius A, Ištuk N, Šarolić A, Lechner S, Klonowski W, Varoneckas G, Széles JC. Current directions in the auricular vagus nerve stimulation I-a physiological perspective. *Front Neurosci.* 2019;13:854.
  28. Maryn Y, Weenink D. Objective dysphonia measures in the program PRAAT: smoothed cepstral peak prominence and acoustic voice quality index. *J Voice.* 2015; 29(1):35-43.
  29. Watts CR, Awan SN, Maryn Y. A comparison of cepstral peak prominence measures from two acoustic analysis programs. *J Voice.* 2017;31(3):387.e1-387.e10.
  30. Awan SN, Roy N, Zhang D, Cohen SM. Validation of the cepstral spectral index of dysphonia (CSID) as a screening tool for voice disorders: development of clinical cutoff scores. *J Voice.* 2016;30(2):130-44.
  31. Awan SN, Roy N, Jette ME. Quantifying dysphonia severity using a spectral/cepstral-based acoustic index: comparisons with auditory-perceptual judgements from the CAPE-V. *Clin Linguist Phon.* 2010;24(9):742-58.
  32. Omari AI, Alzoubi FQ, Alsalem MM, Aburahma SK, Mardini DT, Castellanos PF. The vagal nerve stimulation outcome, and laryngeal effect: Otolaryngologists roles and perspective. *Am J Otolaryngol.* 2017;38(4):408-13.
  33. Shaffer MJ, Jackson CE, Szabo CA, Simpson CB. Vagal nerve stimulation: clinical and electrophysiological effects on vocal fold function. *Ann Otol Rhinol Laryngol.* 2005;114(1 Pt 1):7-14.
  34. Saibene AM, Zambrelli E, Pipolo C, Maccari A, Felisati G, Felisati E, Furia F, Vignoli A, Canevini MP, Alfonsi E. The role of laryngeal electromyography in vagus nerve stimulation-related vocal fold dysmotility. *Eur Arch Otorhinolaryngol.* 2017;274(3):1585-9.