Neurophysiological Contributions to Dysphonia: The Role of Cortical Silent Periods in AdLD☆

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SUMMARY: Objective. Adductor laryngeal dystonia (AdLD) is a task-specific focal dystonia characterized by involuntary vocal fold hyperadduction and impaired voice quality. While reduced cortical inhibition is a hallmark of AdLD, its relationship to acoustic measures of voice dysfunction remains unclear. This study investigated whether intracortical inhibition, as measured by the cortical silent period (cSP) from the laryngeal motor cortex (LMC), is associated with cepstral peak prominence (CPP), a quantitative acoustic marker of voice harmonic periodicity.

Methods. Thirty-two participants (17 AdLD, 15 controls) underwent transcranial magnetic stimulation with fine-wire electromyography targeting the LMC to measure cSP. Acoustic recordings of sustained vowels and connected speech were used to derive CPP. Linear models were used to assess the relationship between cSP, CPP, and perceptual voice severity (Consensus auditory-perceptual evaluation of voice overall severity ratings).

Results. Participants with AdLD exhibited significantly shorter cSP durations and lower CPP values than controls ($P < 0.05$). Across all participants, longer cSP durations were significantly associated with higher CPP values ($\beta = 0.075$, $P = 0.03$), indicating that greater cortical inhibition is associated with better harmonic periodicity in the voice. However, cSP did not independently predict perceptual voice severity after adjusting for age and CPP.

Conclusion. These findings link impaired cortical inhibition in the LMC to less harmonic periodicity in AdLD, supporting a mechanistic role for disinhibition in dysphonic voice production. While cSP did not improve the prediction of perceptual severity, it may serve as a physiological correlate of acoustic dysphonia and a potential biomarker for future neuromodulatory interventions.

Keywords: Adductor laryngeal dystonia–Cortical silent period–Intracortical inhibition–Cepstral peak prominence– Transcranialmagnetic stimulation.

INTRODUCTION

Laryngeal dystonia (LD), or spasmodic dysphonia, is a rare, task-specific focal dystonia affecting speech. Adductor laryngeal dystonia (AdLD), the most common subtype, is characterized by involuntary contractions of the adductor muscles, leading to vocal fold hyperadduction and resulting in a strained or strangled voice, as well as intermittent voice breaks.[1–3] These symptoms impact work, leisure, and social participation, and are associated with lower quality of life.[4] Diagnosis is challenging and often delayed due to perceptual similarity to other vocal disorders and a lack of diagnostic biomarkers.[5–7]

The pathophysiology of AdLD is unknown. Previous work suggests that focal dystonia is a functional brain network disorder characterized, in part, by abnormalities in cortical inhibitory function.[8–13] The cortical silent period (cSP), a measure of gamma-aminobutyric acid type B ($GABA_b$) receptor-mediated inhibitory processes in the motor cortex,[14] has demonstrated shortened durations in AdLD in several muscle groups using transcranial magnetic stimulation (TMS), including the cortical representation of the larynx muscle and more distal muscles unrelated to voice production.[9–11]

Recent work from our group directly measured the cSP from the laryngeal motor cortex (LMC) using intramuscular electromyography (EMG) of the thyroarytenoid (TA) muscles, revealing impaired inhibition in the LMC in AdLD.[11] The LMC is a hub for learned, volitional vocal control and a key node in the speech motor network.[15] Intact motor control depends on balanced input from the basal ganglia-thalamo-cortical circuit, where direct and indirect pathways modulate cortical excitability. In focal dystonia, an imbalance in these pathways is thought to reduce cortical inhibition, leading to excessive and involuntary muscle activation (for review[16]). Abnormal cSPs originating from this region may provide critical insight into the cortical inhibition deficits associated

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with dystonic voice control. Moreover, shortened cSP duration has been reported in hand and facial muscles in AdLD, suggesting a widespread decrease in cortical inhibition beyond the symptomatic laryngeal area.[9,17,18]

Although the cSP may serve as a neurophysiological marker of cortical inhibition, its clinical utility remains uncertain. The cSP has not been reliably linked to functional voice outcomes, and no established clinical correlates have been identified in the context of vocal dysfunction. Moreover, reduced cSP duration is not specific to AdLD. It reflects a general feature of motor circuit dysfunction and has been observed in other neurological conditions.[19] As a result, the clinical relevance of the cSP as a diagnostic or severity marker has yet to be established. This gap highlights the need to connect neurophysiological measures with perceptually and acoustically validated indicators of dysphonia.

Cepstral peak prominence (CPP), an acoustic marker derived from the spectral properties of the speech signal, has emerged as a reliable, objective measure of voice quality that correlates well with perceptual measures of dysphonia severity,[20,21] and has been endorsed by the American Speech-Language-Hearing Association (ASHA)[22] as the preferred acoustic measure for estimating overall noise in the vocal signal and as a general indicator of dysphonia. In AdLD, CPP is reduced compared with control participants,[21] consistent with the presence of hyperadductive vocal fold behavior and irregular glottal pulse timing.[23,24] However, the relationship between CPP and underlying neurophysiological deficits has never been formally investigated.

This study investigates the relationship between cSP from the LMC and CPP in AdLD and healthy controls to assess how variability in intracortical inhibition relates to differences in voice quality. We hypothesize shorter cSP durations will correspond to lower CPP values. Secondly, we evaluated whether incorporating cSP improves the prediction of perceptual voice severity, as rated by the consensus auditory-perceptual evaluation of voice (CAPE-V) overall severity, beyond CPP alone. Establishing the relationship between cortical inhibitory markers, acoustic markers, and auditory-perceptual clinical voice severity could provide a more comprehensive understanding of the neurophysiological basis of AdLD, contribute to the development of biomarkers for disease severity or treatment response, and inform future treatments targeting cortical excitability and voice impairment, such as repetitive TMS.

METHODS

Study design

This cross-sectional study was conducted over two sessions, either on consecutive days or spaced no more than seven days apart. Magnetic Resonance Imaging (MRI) was completed in the first session and used for neuronavigation during TMS testing. Voice recordings and TMS data were collected during the second session. MRI acquisition details are provided in the Supplement.

Participants with a confirmed diagnosis of AdLD or healthy controls were recruited via multiple channels, including flyers posted in local voice clinics, internet advertisements, and direct referrals from the Massachusetts General Hospital Voice Center and collaborating otolaryngology practices. Diagnosis was based on a comprehensive evaluation by a board-certified otolaryngologist.[1] All AdLD participants had neurogenic dysphonia secondary to focal LD. Diagnosis was confirmed through a comprehensive evaluation that included: (1) review of medical history and symptomatology consistent with AdLD,[1] (2) laryngoscopic examination demonstrating normal vocal fold structure to rule out organic pathology (eg, masses, lesions, and structural abnormalities), and (3) characteristic voice breaks and strain during connected speech. Organic causes of dysphonia were excluded in all participants based on laryngoscopic findings.

The inclusion criteria required participants to be between 21 and 85 years of age. For AdLD participants, inclusion required stable AdLD symptoms. For those receiving botulinum toxin (BoTN) injections (n = 20), data collection was planned to coincide with the period when symptoms were at their most severe, typically approximately three months post injection, corresponding to the end of the therapeutic cycle when the effects of BoTN had waned. Participants self-reported that their symptoms had returned to their baseline severity prior to scheduling their study visit.

Exclusion criteria included other forms of dystonia, essential tremor, vocal fold pathology, vocal fold paralysis, other neurological conditions, history of laryngeal surgery or cancer, and diagnosis of psychiatric disorders (eg, bipolar disorder, substance use disorder). Individuals with other dystonias, essential tremor, or TMS[25] and MRI contraindications were excluded.[25] The full inclusion and exclusion criteria are listed in the Supplement.

All participants provided written informed consent. The study was approved by the Institutional Review Boards of Mass General Brigham and the University of Minnesota and conducted in accordance with the Declaration of Helsinki.

Phonatory measures

Voice recordings were made in clinic rooms during the study visit using a Tascam DR-05X handheld recorder, positioned approximately 60 cm from the speaker's mouth, digitized at a 44.1 kHz sampling rate and 16-bit resolution. Tasks included sustained vowels /i/ and /a/ (1–3 seconds each for most participants) and a sample of connected speech, in which participants were asked to speak for one minute about a topic of their choice. Suggested topics included their voice problem, voice functioning, work, and family.

Consensus auditory-perceptual evaluation of voice (CAPE-V)

Auditory-perceptual severity was assessed using the CAPE-V, a validated clinical tool that includes ratings of overall

severity, roughness, strain, and breathiness.[26] Two voice-specialized speech-language pathologists with experience treating patients with AdLD independently rated all recordings on all CAPE-V parameters. Ratings were performed using a CAPE-V interface implemented in MATLAB. Experts listened to recordings of sustained vowels and spontaneous speech and were allowed to listen to each sample more than once. Spontaneous speech was selected rather than standardized CAPE-V sentences because it better captures the natural variability and task-specific nature of AdLD symptoms, which are most prominent during connected speech production. Each expert re-rated 15% of samples to assess inter-rater reliability. The initial 2002 ASHA version of the CAPE-V form was used.[27]

A random 15% of files were re-rated by both raters to assess inter-rater reliability, which was excellent (Intraclass Correlation Coefficient [ICC 3,k] = 0.92). As a result, the two ratings were averaged to produce a single CAPE-V severity score for each participant, which was used in all subsequent analyses. Only CAPE-V overall severity scores were used as the perceptual outcome measure in this study.

Cepstral peak prominence

CPP, an acoustic measure of signal periodicity,[22] was calculated using Praat and a custom MATLAB script.[28,29] CPP refers to the smoothed version of cepstral peak prominence, which is the standard implementation in current acoustic analysis software. CPP was derived from a concatenated sound file that included at least 1 second of the sustained vowel /a/, at least 1 second of the sustained vowel /i/, and spontaneous connected speech. This approach also aligns with current recommendations that CPP from connected speech may be more ecologically valid and sensitive to dysphonia than sustained vowels alone.[20,30]

For the majority of participants (n = 21), approximately 50 seconds of connected speech were recorded (mean = 49.7 seconds, standard deviation [SD] = 21.9 seconds). For 11 participants, speech segments were incomplete, but as much usable speech material as possible was included. Vowel recordings were missing for one participant.

CPP was chosen because it does not rely on fundamental frequency tracking across multiple cycles of vibration or cycle-to-cycle variations in amplitude, making it robust to severely dysphonic voices with irregular periodicity, such as in AdLD.[20,30] CPP also correlates well with auditory-perceptual severity ratings in other voice disorders.[20,30]

Cortical silent period

TMS data were collected as previously reported.[11,31] The skin around the area of the laryngeal prominence was cleaned using alcohol wipes. A topical anesthetic cream (LMX 4% Lidocaine, Ferndale Laboratories, Inc., Ferndale, MI) was applied to the cleaned area. Then, a numbing agent (Xylocaine, 2% lidocaine HCL and epinephrine, 1:100,000, Professional Veterinary Laboratories, NB, Canada) was injected superficially into the skin to raise a wheal at the injection site. After skin preparation, paired hook-wire electrodes (Natus Neurology Inc., Middleton, WI) were percutaneously inserted into bilateral TA muscles by an experienced otolaryngologist following standard procedures for laryngeal EMG.[32] Electrode placement in the TA muscle was confirmed in real time using an EMG oscilloscope during phonation. Appropriate signal amplitude and pattern during voicing confirmed correct placement. If signals indicated improper placement, electrodes were repositioned or reinserted. Because the TA muscles are bilaterally innervated, EMG was recorded from both sides simultaneously.

TA EMG signals were recorded using fine-wire electromyographic preamplifiers (MA-416-003, Motion Lab Systems, Inc., Baton Rouge, LA) with a fixed gain of ×20 and a bandwidth of 15 Hz to 3.5 kHz. Signals were subsequently amplified by a main amplifier with a gain of ×2000 and a band-pass filter set between 20 Hz and 2000 Hz. The amplified signals were digitized at 6.4 kHz using a 24-bit analog-to-digital converter (Micro3 1401, Cambridge Electronic Design, UK). Data acquisition and real-time EMG monitoring were performed using custom software written in Signal (Cambridge Electronic Design, Cambridge, UK).

Single-pulse TMS was delivered using a 70-mm figure-of-8 coil connected to a Magstim 200 stimulator (The Magstim Company LTD, UK). The coil was placed tangentially to the scalp over the left and right hemisphere LMC with the handle pointing posteriorly. Coil positioning was guided by frameless stereotactic neuronavigation (Brainsight, Rogue Research Inc., Montreal, Canada) using each participant's individual T1-weighted MRI.

The TA motor hotspot was identified as the scalp location that reliably elicited the largest and most consistent motor-evoked potentials (MEPs) and visible cSPs during phonation. Once the hotspot was identified, the cSP threshold was determined as the lowest stimulation intensity (expressed as a percentage of maximum stimulator output) that elicited a visible cSP in at least three out of five consecutive trials. For data collection, participants were instructed to sustain an /i/ vowel at a comfortable pitch and volume for 2–3 seconds per trial. TMS pulses were delivered during sustained phonation, approximately 1 second after phonation onset. Stimulation was delivered at the cSP threshold, and thirty trials were recorded from the left and right hemispheres for all participants.

cSP duration was defined as the time interval between stimulus onset and return of EMG to prestimulus baseline levels. Processing was performed on averaged and rectified EMG signals across all 30 trials per hemisphere for each participant. First, EMG signals were full-wave rectified. Then, a 10-millisecond moving SD window was applied to generate an SD curve of the rectified signal. The prestimulus baseline contraction level was calculated as the mean value of the SD curve during a 100-millisecond window immediately preceding the TMS pulse. The onset of the cSP was defined as the time of stimulus delivery

(0 milliseconds). The offset of the cSP was identified as the first time point after the stimulus when the SD curve returned to and remained above the prestimulus baseline level for at least 10 consecutive milliseconds. Two examiners, blinded to participant group assignment, independently confirmed the cSP offset by visual inspection of the averaged EMG traces. The final offset value was determined by consensus between the two examiners if differences existed.

cSP durations were calculated separately for the left and right TA muscles and then averaged to yield a single bilateral cSP value per participant per hemisphere. When data from one TA were missing due to electrode malfunction or poor signal quality, the available side was used (n = 13). This approach increased reliability and statistical power, as prior work has shown minimal differences in cSP duration between sides.[31]

Trials contaminated by large TMS-induced electrical artifacts that obscured the EMG signal or trials in which participants failed to maintain consistent phonation were excluded from analysis. Although stimulation data were collected from both the left and right hemisphere LMC, only the left hemisphere stimulation data were included in the linear regression analyses, given the left hemisphere's established dominance in speech motor control.[15,33]

Statistical analysis

Data were screened for missing values and outliers. Descriptive statistics were calculated, and model assumptions (normality, linearity, and homoscedasticity) were assessed. Group differences in cSP were evaluated using a two-way analysis of variance (ANOVA) (group × hemisphere). A Welch *t*-test assessed CPP group differences.

To examine the association between cSP and CPP, a linear regression model was used with CPP as the dependent variable, and group, age, and cSP as predictors. Age was included as a covariate to account for potential age-related variation in both cSP and CPP.[34–38] We also tested for an interaction between cSP and group to evaluate whether the relationship between cSP and CPP differed between individuals with and without AdLD by fitting a second model with a cSP × group interaction term and comparing it to the main model using a likelihood ratio test (LRT). Sex was not included as a covariate in the primary model. While CPP values are known to differ by sex (with females typically showing lower values),[39] sex has not been consistently shown to affect cSP duration in prior studies.[40] Our approach prioritized examining the neurophysiological-acoustic relationship while accounting for potential confounders (age and group) that could affect both variables. We conducted sensitivity analyses, including sex as an additional covariate, to assess whether the observed relationship was robust to differences in sex distribution across groups.

In a secondary analysis, we also examined whether cSP contributes to perceptual severity ratings using a linear model. We compared a base model including CPP and age with an extended model that also included cSP, using LRTs to evaluate model improvement. Model fit was assessed using pseudo-R^2 and log-likelihood values.

Model assumptions were evaluated through standard diagnostic plots and statistical tests. Significance was set at $P < 0.05$. All statistical analyses were conducted using R (v2024.12.1 + 563).[41]

RESULTS

Study cohort

Of the total 53 participants with AdLD or who were healthy controls that were recruited, eight were excluded prior to data collection due to withdrawal (n = 2), study termination before participation (n = 2), and diagnosis of abductor LD (n = 4). The remaining 45 participants (23 AdLD, 22 Control [CTL]) were enrolled and completed study procedures.

Following data collection, three additional participants were excluded due to poor fine- wire EMG signal quality (1 AdLD, 2 CTL), resulting in 42 participants with usable cSP data (22 AdLD, 20 CTL). Of these 42, voice data were incomplete or unusable for 10 participants (5 AdLD, 5 CTL) due to corrupt audio files (n = 3), dampening sound quality (n = 1), or excessive environmental noise (n = 6). Descriptive statistics are presented in Table 1. Two mild and expected adverse events were reported following the study session: transient throat tightness and soreness during swallowing related to EMG electrode placement (n = 1), and mild headache (n = 1).

Cortical silent period

A two-way ANOVA revealed a significant main effect of Group, $F(1, 39) = 5.02$, $P = 0.03$, indicating shorter cSP durations in AdLD. No effects of Hemisphere $F(1, 39) = 0.06$, $P = 0.80$, nor a significant Group × Hemisphere interaction, $F(1, 39) = 0.0005$, $P = 0.98$, suggest that the reduction in cSP observed in AdLD was consistent across hemispheres. In AdLD, mean cSP durations were 48.08 milliseconds (SD = 12.41) and 47.76 milliseconds (SD = 13.22) in the left hemisphere and right hemisphere, respectively. In controls, cSP durations were 59.03 milliseconds (SD = 15.08) on the left and 57.78 milliseconds (SD = 16.59) on the right (Figure 1). Representative cSP responses during left cortical stimulation are shown in Figure 2.

Smoothed cepstral peak prominence

AdLD (M = 8.32, SD = 2.55 dB) demonstrated significantly lower CPP values compared with controls (M = 10.37, SD = 2.38 dB), $t(34.99) = -2.54$, $P = 0.02$, 95% Confidence Interval (CI) [–3.70, –0.41] dB, indicating that AdLD demonstrated less harmonic periodicity in the voice.

Relationship between CPP and cSP

Linear regression demonstrated that longer cSP durations were associated with higher CPP ($\beta = 0.075$, $P = 0.03$). The

TABLE 1.
Baseline Demographic and Clinical Characteristics (N = 32)

Group	AdLD (n = 17)	Control (n = 15)	*P* Value
Sex female (%)	13 (81.25)	4 (18.8)	0.02
Age (years)			0.11
Mean ± SD	59.53 ± 11.01	52.27 ± 14.35	
Median (range)	64 (38–75)	52 (29–70)	
Symptom duration (years)		N/A	N/A
Mean ± SD	16.81 ± 10.15		
Median (range)	17 (1–36)		
Voice handicap index total		N/A	N/A
Mean ± SD	61.24 ± 19.58		
Median (range)	62 (8–94)		
CAPE-V overall severity	49.3 ± 15.78	N/A	N/A
Mean ± SD	47.79		
Median (range)	(28.71–78.35)		
Left hemisphere RMT (%)			0.48
Mean ± SD	51.12 ± 10.5	53.67 ± 9.52	
Median (range)	50 (33–75)	54 (34–70)	
Right hemisphere RMT (%)			0.95
Mean ± SD	53.12 ± 10.28	53.33 ± 9.29	
Median (range)	50 (40–75)	54 (38–70)	

Demographic and other characteristics are summarized as median (range), N (%), or mean ± standard deviation.
Abbreviations: AdLD, adductor laryngeal dystonia; CAPE-V, consensus auditory-perceptual evaluation of voice; RMT, resting motor threshold; SD, standard deviation.

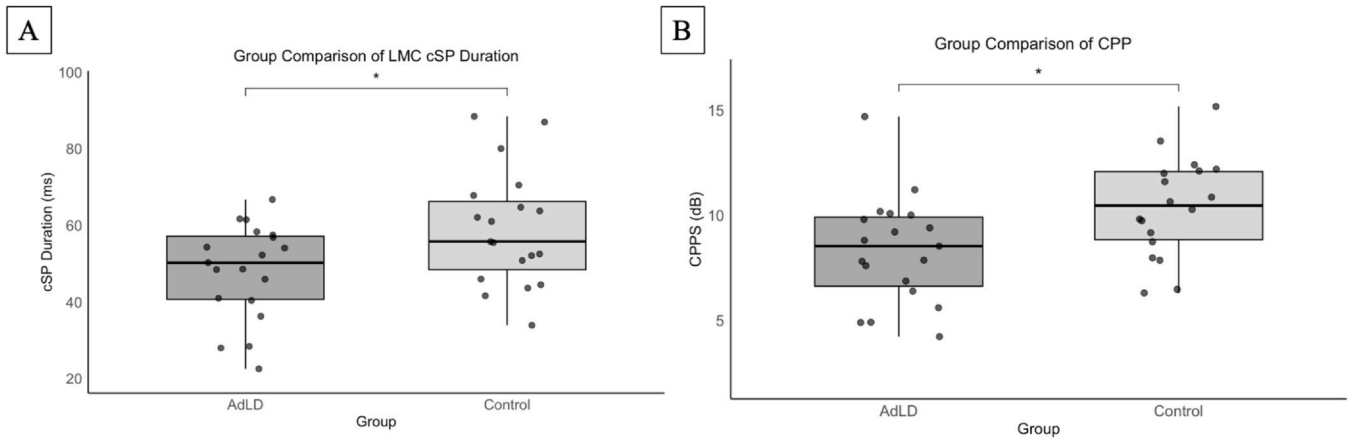

FIGURE 1. **(A)** Boxplot with individual data points showing cortical silent period (cSP) durations measured from the left hemisphere laryngeal motor cortex (LMC) in individuals with AdLD and healthy controls. The AdLD group showed significantly shorter cSP durations than controls, F (1,28) = 5.55, $P = 0.02$. **(B)** Boxplot with individual data points showing CPP in AdLD and healthy controls. CPP values were significantly lower in the AdLD group compared with controls, $t(34.99) = -2.54$, $P = 0.02$, 95% CI [–3.70, –0.41]. Together, these results demonstrate group-level reductions in both cortical inhibition and voice periodicity in AdLD. AdLD, adductor laryngeal dystonia; CI, confidence interval; cSP, cortical silent period; CPP, smoothed cepstral peak prominence; LMC, left hemisphere laryngeal motor cortex.

model was significant overall, $F(3, 28) = 4.82$, $P = 0.0079$, explaining 34% of variance in CPP ($R^2 = 0.34$, adjusted $R^2 = 0.27$) (Figure 3). Age showed a negative trend ($\beta = -0.069$, $P = 0.062$), indicating potential age-related decline in voice quality, though this was not significant. Group differences (AdLD vs control) were not significant after adjusting for cSP and age ($\beta = 0.91$, $P = 0.36$).

Additionally, a model including the cSP × Group interaction term was compared using an LRT. The interaction did not significantly improve model fit, $\chi^2(1) = 0.01$, $P = 0.92$, suggesting that cSP and acoustic voice quality do not differ between AdLD and controls. Model assumptions were evaluated and met.

Given the significant sex distribution difference between groups ($\chi^2 = 5.21$, $P = 0.02$), we conducted a sensitivity analysis including sex as an additional covariate. The relationship between cSP and CPP remained significant when controlling for sex, age, and group ($\beta = 0.073$, $P = 0.033$).

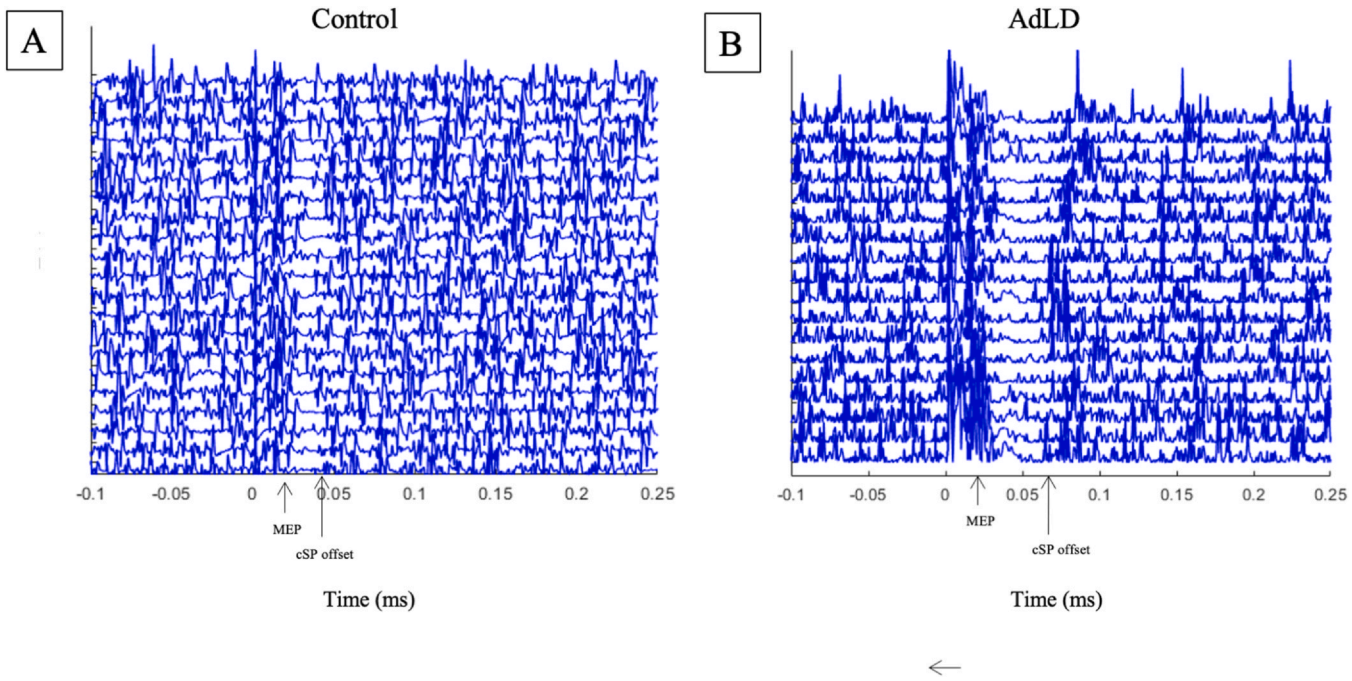

FIGURE 2. Representative raw EMG data of two participants demonstrating the cortical silent period (cSP) waterfall plot were collected. **(A)** In total, 30 individual traces of left TA responses to left cortical stimulation from a control participant; **(B)** 15 individual traces of left TA responses to left cortical stimulation from an AdLD participant. The stimuli were delivered at 0 milliseconds. AdLD, adductor laryngeal dystonia; EMG, electromyography; MEP, motor-evoked potential.

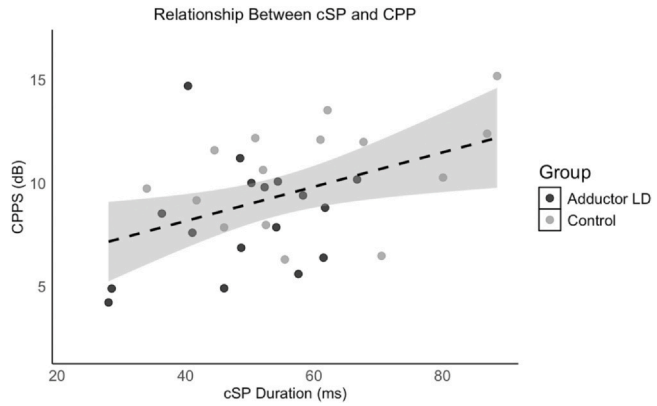

FIGURE 3. Scatterplot showing the relationship between left hemisphere LMC cSP duration and CPP across participants. The dashed black line represents a simple linear regression fit between cSP and CPP across all participants, with the gray shaded area showing the 95% confidence band. In a multiple regression model controlling for age and group, longer cSP durations were significantly associated with higher CPP values ($\beta = 0.073$, $P = 0.03$, $R^2 = 0.34$), indicating that greater intracortical inhibition was linked to more periodic, less dysphonic voice signals. AdLD, adductor laryngeal dystonia; cSP, cortical silent period; CPP, cepstral peak prominence; LMC, laryngeal motor cortex.

Sex showed a positive but nonsignificant effect ($\beta = 1.146$, $p = 0.279$), age showed a negative trend ($\beta = -0.067$, $P = 0.071$), and group differences remained nonsignificant ($\beta = 0.722$, $P = 0.482$). The overall model fit was comparable to the primary model ($R^2 = 0.36$, adjusted $R^2 = 0.27$ vs $R^2 = 0.34$, adjusted $R^2 = 0.27$ in the primary model; $F(4, 27) = 3.79$, $P = 0.015$), highlighting that sex distribution differences did not substantially affect the primary cSP-CPP relationship.

cSP and CPP as predictors of CAPE-V overall severity

A model including CPP and age significantly predicted CAPE-V overall severity, $F(2, 14) = 4.82$, $P = 0.026$, with an R^2 of 0.41 (adjusted $R^2 = 0.32$); however, CPP was not a significant individual predictor ($\beta = -1.03$, $P = 0.41$). Age was positively associated with severity ($\beta = 0.80$, $P = 0.017$), indicating that older age was linked to greater perceived severity.

An extended model including cSP yielded a slightly higher R^2 of 0.43 (adjusted $R^2 = 0.30$), $F(3, 13) = 3.25$, $P = 0.057$. However, this was not statistically significant ($F(1,13) = 0.47$, $P = 0.50$). Neither CPP ($\beta = -0.72$, $P = 0.59$) nor cSP ($\beta = -0.21$, $P = 0.50$) were significant predictors, while age remained significant ($\beta = 0.87$, $P = 0.017$). An LRT comparing the two models confirmed that adding cSP did not significantly improve model fit ($\chi^2(1) = 0.61$, $P = 0.44$). Therefore, cSP did not account for additional variance in CAPE-V overall severity scores beyond age and CPP. Model assumptions were evaluated and met.

DISCUSSION

This study is the first to investigate the relationship between cortical inhibition and an acoustic measure of voice quality in AdLD. Our results demonstrated that AdLD had

shorter cSP compared with controls, indicating abnormalities in intracortical inhibition as measured from the LMC, consistent with previous studies.[11] We also found that longer cSP durations were associated with higher CPP in our linear regression model, indicating that greater intracortical inhibition was linked to more harmonic periodicity in the voice signals. In other words, individuals with more inhibition in the LMC tended to have better voice quality, regardless of diagnosis. These neurophysiological findings have important potential implications for clinical practice and future therapeutic development.

Our findings suggest that variability in cortical inhibition may have functional consequences for acoustic voice quality and thus, laryngeal control. Impaired cortical inhibition in AdLD is linked to dysfunction in the basal ganglia-thalamo-cortical circuit, which normally regulates motor output through a balance of excitatory (direct pathway) and inhibitory (indirect pathway) influences on the motor cortex. Previous work has demonstrated an imbalance between the direct and indirect basal ganglia pathways, leading to excessive thalamo-cortical drive and reduced inhibitory modulation of the motor cortex.[42] In line with this model and with previous studies, we observed shorter cSP in the LMC among AdLD, suggesting reduced $GABA_B$-mediated inhibition in the cortical region responsible for volitional voice control. This imbalance manifests as reduced GABAergic inhibition at the motor cortex, which the cSP captures at the cortical level.

We further found that longer cSP duration was associated with higher CPP, indicating that greater intracortical inhibition in the LMC was associated with better voice quality. The association between cSP and CPP across both AdLD and control participants suggests that intracortical inhibitory mechanisms contribute to phonatory stability even in typical voice production. In this light, AdLD may reflect a pathological amplification of a more general neural mechanism underlying voice control.

AdLD is characterized by intermittent, involuntary overactivation of the TA muscles, leading to excessive vocal fold adduction that disrupts periodic vibration and produces the characteristic strained-strangled voice. Abnormal motor output from central circuits has long been implicated in the pathological laryngeal muscle activation observed in AdLD.[24] Consistent with this proposed mechanism, physiological studies demonstrated that AdLD is associated with excessive glottic resistance and elevated subglottic pressure during phonation, contributing to voice strain and instability.[23] Defining physiological features of AdLD (eg, increased glottic compression, vocal fold collision forces, and transient elevations in subglottic pressure) may reflect dysregulation in laryngeal motor control. In this context, reduced cortical inhibition may limit the brain's ability to regulate excessive motor outputs. The disruption in motor control could thus destabilize periodic vocal fold vibration, increase the amount of effort required to speak, and contribute to a strained and unstable voice. While reduced CPP has been reported in AdLD,[43] our findings suggest that impaired cortical inhibition may underlie this dysregulated laryngeal drive. This supports the idea that insufficient inhibitory control at the cortical level may contribute to dysphonia. If validated in larger studies, cSP measures could potentially serve as neurophysiological markers to guide treatment selection or monitor response to interventions targeting cortical excitability.

We also found that incorporating cSP did not improve the prediction of CAPE-V overall scores beyond CPP and age. This suggests that cSP did not explain additional variance in perceived severity of voice. These findings are consistent with previous work, which found that while lower motor thresholds (ie, higher cortical excitability) correlated with worse severity, the cSP, measured from nonlaryngeal muscles, was not associated with the overall severity scores of the CAPE-V.[9] Similarly, our study found no relationship between cSP and CAPE-V, despite using a more targeted measure of inhibition. One possible explanation is that dysphonia in AdLD may arise from both primary dystonic spasms and secondary compensatory or maladaptive hyperfunctional behaviors.[44] If CAPE-V ratings reflect a mixture of these mechanisms, the specific contribution of reduced cortical inhibition may be masked or diluted in perceptual measures. Similarly, CPP does not account for all aspects of dysphonia. For example, it may miss compensatory hyperfunction that contributes to the voice disorder but is not reflected in signal noise. Additionally, CPP and CAPE-V overall severity were not significantly associated in our sample. Although CPP and CAPE-V are strongly correlated in other voice disorders,[20,30] this relationship may be weaker in AdLD,[45] suggesting that acoustic and perceptual measures capture partially distinct aspects of the disorder. This discrepancy may reflect limited power, differences in speech stimuli, or inherent variability of perceptual ratings. However, it may also point to fundamental differences in what these measures assess. Objective acoustic measures primarily reflect harmonic periodicity and noise in the voice signal, while perceptual ratings incorporate broader auditory features, including effort, strain, and pitch variability. These data underscore the importance of using objective and perceptual measures as complementary approaches to capture the multifaceted nature of voice dysfunction in AdLD.

Limitations

The sex distribution differed significantly between groups, with our AdLD sample having proportionally more females than the control group, consistent with reported epidemiological trends in AdLD, though findings on prevalence sex differences are mixed.[6,46] CPP values are generally lower in females,[39] and thus sex-related variation likely contributes to the group-level CPP reduction observed here. However, sensitivity analyses revealed that the relationship between cSP and CPP remained significant when controlling for sex ($\beta = 0.073$, $P = 0.033$), indicating that the neurophysiological-acoustic relationship is independent of sex distribution differences between groups.

Nevertheless, the imbalance limits our ability to fully disentangle potential sex effects from disease effects on acoustic voice quality, and future studies with sex-matched samples would provide additional clarity.

The cross-sectional design of this study precludes causal inference. While we observed associations between cortical inhibition and voice quality, the directionality of these relationships remains unclear. We cannot determine whether reduced cortical inhibition drives voice dysfunction or whether chronic voice disorder leads to cortical changes. Additionally, our sample size may have limited our ability to detect smaller effects, particularly in the perceptual severity models. Our methodological choices regarding acoustic and perceptual measurements may also have constrained our findings. The lack of established normative cSP data specific to laryngeal muscles and voice disorders also limits our ability to define clinically meaningful thresholds or cutoff values for abnormal cortical inhibition. Additionally, we used the initial 2002 ASHA version of the CAPE-V form.[27] The subsequently published standardized protocol[26] introduced modifications, including symmetrically arranged markers that may offer improved sensitivity for detecting perceptual changes in future studies.

Lastly, our acoustic analysis focused exclusively on CPP. While this measure is endorsed by ASHA as the preferred acoustic marker for overall dysphonia[22] and is particularly robust to the aperiodic vocal signals in AdLD,[20,30] other acoustic parameters (eg, pitch strength, computed sharpness) may capture dimensions of vocal strain not reflected in CPP alone. Our perceptual analysis similarly focused on the overall severity ratings of CAPE-V from sustained vowels and spontaneous speech, rather than the full CAPE-V protocol. This decision was driven by data completeness across participants and psychometric considerations, as overall severity demonstrated superior inter-rater reliability (ICC = 0.92) compared with other perceptual parameters, such as strain (ICC = 0.77). However, this approach precluded examination of relationships between cortical inhibition and specific perceptual dimensions (ie, strain, roughness, or breathiness) that may be differentially sensitive to neurophysiological dysfunction. Future studies incorporating broader acoustic and perceptual measurement batteries may clarify which specific features of voice dysfunction are most closely linked to cortical disinhibition.

Future directions and clinical implications

Our findings have potential implications for clinical practice, patient management, and research, although significant validation is needed before clinical implementation. The link between reduced cortical inhibition and voice dysfunction suggests that cSP measures may potentially help stratify patients for different therapeutic approaches. For example, patients with more pronounced cortical disinhibition might be candidates for neuromodulatory interventions targeting cortical excitability, such as repetitive TMS, which has shown promise in other focal dystonias.[47–49] Conversely, patients with relatively preserved cortical inhibition might respond preferentially to other interventions such as standard BoTN therapy. However, significant barriers remain before cSP can be routinely implemented in clinical settings. The current methodology requires invasive laryngeal EMG, specialized equipment, and technical expertise, making it impractical for widespread clinical use. Exploring noninvasive proxies, such as cSP from more accessible muscles or alternative neurophysiological markers like cortical activity measured via electroencephalography, would be necessary to translate these findings into routine clinical practice. Further validation in larger, diverse cohorts and demonstration of clinical utility (eg, improved treatment outcomes, cost-effectiveness) would also be essential steps toward clinical integration.

Longitudinal studies tracking cSP and CPP are needed to determine whether cortical inhibition is a stable trait or fluctuates with symptom severity, and whether changes in cortical inhibition predict or follow changes in voice function. Such studies could explore whether baseline cSP values predict treatment response to BoTN injections or whether changes in cSP parallel changes in voice function following intervention. Incorporating cSP measures into clinical trials of novel therapeutics (eg, neuromodulation, pharmacological agents targeting GABAergic function, and intensive voice therapy) could provide mechanistic insights into treatment effects and help identify responders vs nonresponders. Integration of cSP measures with other neurophysiological and neuroimaging biomarkers would provide a comprehensive assessment of cortical and subcortical network dysfunction in AdLD. Combining TMS measures of cortical excitability (eg, motor threshold, intracortical facilitation, and MEP amplitude) with structural and functional MRI could identify relationships between cortical inhibition deficits and basal ganglia-thalamo-cortical circuit dysfunction. Advanced imaging techniques such as GABA-edited magnetic resonance spectroscopy could directly assess GABAergic neurotransmitter concentrations in the motor cortex and examine their relationships with TMS-derived measures of inhibition and acoustic voice outcomes. Additionally, comparative studies examining cSP across the spectrum of voice disorders would help establish the specificity of cortical inhibition deficits to AdLD, and interventional studies incorporating neurophysiological measures as secondary outcomes could evaluate whether treatments that improve voice function also normalize cortical inhibition.

Collectively, this work supports the hypothesis that cortical disinhibition may underlie the aperiodic, strained vocal quality characteristic of AdLD. While preliminary, these findings also point toward the possibility of integrating neurophysiological measures into future biomarker frameworks for dystonia. For example, cSP could complement acoustic and perceptual assessments in efforts to stratify severity, monitor treatment response, or differentiate AdLD from perceptually similar voice disorders.

Additional research is needed to validate this approach in larger and more diverse samples and to examine the responsiveness of these markers to interventions.

CONCLUSIONS

This study demonstrates that impaired cortical inhibition in the LMC, as measured by shortened cSPs, is associated with lower acoustic voice harmonic periodicity. While this suggests a broader role for cortical inhibition in shaping phonatory control, its disruption in AdLD appears to represent a pathological exaggeration of this mechanism. These findings provide preliminary support for a mechanistic link between intracortical motor regulation and the observable voice abnormalities in AdLD, underscoring the potential value of neurophysiological markers for understanding pathophysiology and informing clinical assessment.

CRediT Authorship Contribution Statement

Design: BPH, TJK. Data collection: BPH, JB, and TJK. Analysis: BPH, KLM, CES, and TJK. Writing, original draft: BPH. Revising and editing: BPH, KLM, CES, and TJK. Funding: TJK.

Declaration of Competing Interest

All other authors report no competing interests.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jvoice.2026.01.028.

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