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University of Illinois to Hananeh Esmailbeigi for potential commercialization. The other authors declare that they have no competing interests.

Tongue Agility Measures Reveal Distinct Movement Signatures in Dysarthria and Apraxia of Speech

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Abstract

Background

Precise and rapid tongue movements are central to speech production, yet clinical assessment of motor speech disorders relies largely on perceptual and acoustic measures, and lack objective, quantitative evaluations. We addressed this gap by isolating and quantifying tongue agility as an objective measure of oromotor performance in motor speech impairment.

Methods

In this preliminary cross-sectional study, we evaluated tongue movements in 9 individuals with dysarthria and 10 with apraxia of speech, comparing their performance with data from 16 neurotypical controls. Tongue movements were captured using our standalone wireless intraoral *Tongue-Trackpad* during a self-directed exploration task, *free exploration,* with real-time visual feedback, from which movement probability distributions were generated.

Results

Quantitative analysis revealed individualized movement signatures and distinct group-level patterns in dysarthria and apraxia of speech. Compared with the neurotypical control patterns, individuals with apraxia of speech exhibited centrally concentrated movement surrounded by broad, continuous bilateral regions of reduced movement, whereas individuals with dysarthria showed localized, right-lateralized reductions. The area of reduced movement was 7.21 times greater in apraxia of speech than in dysarthria. In dysarthria, selected movement measures were significantly associated with diadochokinetic rate, linking quantified tongue movement measurements to speech motor performance. We further introduced the composite

Tongue Agility Score, integrating spatial and velocity movement range and tongue contact area as a multidimensional measure of overall performance relative to the neurotypical controls. The score revealed significant group-level differences among neurotypical controls, dysarthria, and apraxia of speech. Together, individualized maps characterize where tongue movement deviates from neurotypical patterns, while the Tongue Agility Score quantifies the magnitude of overall deviation.

Conclusions

The results demonstrated the feasibility of objectively characterizing tongue movement impairment and revealing group-level patterns in dysarthria and apraxia of speech using a brief, portable assessment. By quantifying tongue agility as a component of oromotor function, this pilot study provides a foundation for a clinically accessible individualized assessment, with potential applications in longitudinal monitoring and intervention planning.

Keywords: Precision assessment; Intraoral wearables; Tongue motor control; Oromotor impairment; Precision rehabilitation; Neurological injury

Background

The complex and coordinated movements of the tongue are essential for vital functions such as speech, breathing, chewing, and swallowing. Neurological injuries, including stroke and traumatic brain injury (TBI), can disrupt tongue motor control and contribute to clinically significant impairments in oromotor function [1,2]. These impairments may include muscle spasticity, abnormal muscle coupling, reduced strength, and irregular or poorly coordinated movements [2–4]. Although tongue movement occurs within a coordinated oromotor system involving the lips, jaw, pharynx, and soft palate, direct characterization of *tongue agility*, the

ability to execute rapid and precise movements, may provide insight into the nature and severity of oromotor impairment.

Dysarthria and apraxia of speech are motor speech disorders associated with neurological injury. Dysarthria is associated with weakness and impaired coordination and control of musculature, whereas apraxia of speech is characterized by challenges in motor planning and programming [5–8]. Clinical assessment of these disorders relies largely on perceptual and acoustic evaluations. Common assessments include diadochokinetic (DDK) rate, which measures accuracy and speed of sequential syllable production and is interpreted as an indicator of articulatory strength, speed, and coordination [9–12]. DDK testing is widely used as it is rapid to administer, can be performed by speech-language pathologists (SLPs) without external evaluators, and is applicable across disorders [13,14]. Additional disorder-specific assessments include the Frenchay Dysarthria Assessment, the Assessment of Intelligibility of Dysarthric Speech , and the Apraxia of Speech Rating Scale [15–17]. Although these assessments are clinically valuable, their interpretation may be influenced by examiner expertise, perceptual judgment, scoring requirements, and variability in administration [18–21].

These limitations become particularly important when characterizing motor speech disorders, such as dysarthria and apraxia of speech, that share overlapping perceptual and behavioral features [22]. A recent review of diagnostic practices across 198 apraxia of speech studies further emphasized the absence of an objective diagnostic reference standard and the continued reliance on perceptual features, clinical judgment, and expert consensus [22]. Recent research in progressive neurological disorders has further emphasized the need for objective, quantitative measures to complement auditory-perceptual motor speech assessments [23]. Characterization of motor speech impairment is clinically important because it may inform

intervention planning, prognosis, and interpretation of treatment response [24–27]. Impairment profiles may also change during recovery following neurological injury [28–30]. Together, these challenges highlight the potential value of quantitative measures that complement established clinical assessment. In this study, we investigated whether quantifying tongue movement during a non-speech exploration task could provide such an objective measure. Although isolated tongue movement is not itself a functional task, it may expose underlying motor constraints that contribute to functional impairment.

The neural control of tongue movement has been investigated at the cortical level through invasive techniques neural implants and noninvasive methods, including electroencephalography, positron emission tomography, and transcranial magnetic stimulation [31–38]. Techniques such as videofluoroscopy, ultrasound, sonography, electropalatography, and electromagnetic articulography have enabled the visualization of tongue movement [39–47]. In addition, magnetic resonance imaging has been used to evaluate tongue movement and neural activity in individuals with motor speech disorders, primarily for research purposes [48,49]. However, the broader adoption of these approaches for routine quantitative tongue assessment has been limited by equipment size, lack of portability, technical complexity, and the need for specialized expertise. The Iowa Oral Performance Instrument offers an objective measurement of tongue pressure against the palate but does not directly assess movement [50,51]. Consequently, there remains a need for a clinically accessible method capable of objectively quantifying tongue agility and characterizing individualized movement impairment during brief clinical assessments.

Our group has developed a method to address this gap by isolating and quantifying tongue agility. In prior work, we introduced the *Tongue-Trackpad*, a standalone wireless intraoral device positioned along the palate that captures tongue-contact locations and provides

real-time visual feedback of the movement [52,53]. When used during self-directed voluntary exploration task, *free exploration*, the platform revealed individualized movement patterns in neurotypical individuals [52]. We also showed that real-time visual feedback during free exploration encouraged individuals to express a broader range of their movement capabilities [52]. Free exploration paradigms have similarly been used in studies of upper-limb movement to characterize individualized movement patterns and reveal motor impairments following stroke [54–59]. We further applied this approach to precision rehabilitation and demonstrated that individualized visual feedback interventions significantly improved tongue agility over seven one-hour intervention sessions in individuals with dysarthria [53]. Together, these findings support visually guided free exploration as a framework for eliciting and quantifying individualized movement capabilities.

In the present study, we apply this framework to individuals with dysarthria and apraxia of speech and evaluate isolated tongue movement without constraining performance to functional speech or swallowing tasks. We hypothesized that quantitative analysis of self-directed exploratory tongue movements would reveal individualized movement signatures, identify differences between dysarthria and apraxia of speech, and provide objective measures of tongue movement performance. We introduce the *Tongue Agility Score*, a composite measure integrating spatial and velocity movement range and tongue contact area to quantify overall tongue-movement performance relative to the neurotypical reference. Rather than proposing a standalone diagnostic method, this pilot study evaluates tongue movement as one measurable component of the broader oromotor system. It represents an initial step toward a potentially clinically accessible, objective method for individualized characterization of tongue agility during brief assessments, with the potential to contribute to the multidimensional characterization of motor speech impairment.

Methods

Participants

Nineteen participants (mean age: 49.3 ± 17.4 years; 8 females), at least 3 months post-injury, were recruited for this study (Table 1). Nine had a clinical diagnosis of dysarthria and ten of apraxia of speech, as documented in their medical records. Given the preliminary nature of the study and availability of eligible participants, no formal sample-size calculation was performed. An SLP measured participants' DDK rate. Participants provided informed consent in accordance with Northwestern University's Institutional Review Board (IRB) (protocol number: STU00214010) and received no compensation for their participation. The inclusion criteria were: 1) age 18 - 80 years; 2) clinical diagnosis of dysarthria or apraxia of speech without decisional impairments or oral health concerns; 3) ability to complete the research tasks; and 4) no known sensitivity to the device materials. Participants were primarily recruited through the Shirley Ryan AbilityLab (SRALab) institutional database, where individuals had enlisted and consented to be contacted for eligible studies. Additionally, SLPs from SRALab outpatient and day rehabilitation recommended individuals who had consented to be contacted.

Control data were obtained from 16 neurotypical participants (mean age: 25.7 ± 5.6 years; 6 females) who had taken part in an earlier study [52]. These participants provided informed consent in accordance with the University of Illinois Chicago IRB (protocol number: 2017-0550).

Participants with Motor Speech Disorders

Participant ID	Age	Sex	Motor Speech Disorder	DDK Rate	Neurological Injury	Stroke Type	Months Post-Injury
D1	59	F	Dysarthria	0	Stroke	Bilateral	33
D2	32	M	Dysarthria	0	TBI[b]	-	57
D3	73	M	Dysarthria	4	Stroke	Pontine Infarction	13
D4	52	M	Dysarthria	8	Stroke	ICH	45
D5	32	F	Dysarthria	10	TBI	-	173
D6	42	M	Dysarthria	17	Stroke		16
D7	31	F	Dysarthria	21	TBI	-	135
D8	34	F	Dysarthria	23	Stroke	Left CVA Ischemic and Hemorrhagic	20
D9	24	M	Dysarthria	29	TBI	-	42
A1	23	M	Apraxia of Speech	2	TBI	-	3
A2	49	M	Apraxia of Speech	4	Stroke	-	11
A3	71	M	Apraxia of Speech	4	Stroke	Left MCA	14
A4	78	F	Apraxia of Speech	5	Stroke	Ischemic Stroke	24
A5	46	M	Apraxia of Speech	6	Stroke	Ischemic Stroke	18
A6	64	M	Apraxia of Speech	8	Stroke	Left MCA	124
A7	57	M	Apraxia of Speech	9	Stroke	Left MCA	21
A8	64	F	Apraxia of Speech	9	Stroke	-	20
A9	67	F	Apraxia of Speech	15	Stroke		11
A10	39	F	Apraxia of Speech	22	Stroke	Left MCA	10

Neurotypical Control Group

Number of Participants	Age (mean ± std)	Sex
16	25.7 ± 5.6 years	6 F and 10 M

Table 1. Participant characteristics. Demographic and clinical characteristics of participants with motor speech disorders, including age, sex, motor speech disorder, diadochokinetic (DDK) rate, neurological injury, stroke type, and months post-injury. Participants are ordered by increasing DDK rate within each disorder group. Participant IDs indicate the motor speech disorder (D: dysarthria; A: apraxia of speech) and participant number. The neurotypical control group sample size, mean age, and sex distribution are also shown. CVA, cerebrovascular accident; MCA, middle cerebral artery; TBI, traumatic brain injury; ICH, intracerebral hemorrhage.

Protocol

Participants with motor speech disorders completed one session using the intraoral Tongue-Trackpad. The device was developed by our team and described previously (Fig. 1A) [52]. The device captured the center of tongue-tip contact (Fig. 1B) and provided participants with real-time visual feedback via a custom graphical user interface in Hypertext Markup Language (HTML) (Fig. 1C). Tongue-tip position appeared as a 1-mm black dot within the sensing area, with preceding dots showing movement history. Participants received 1 minute to familiarize themselves with the device and feedback. Participants then performed a self-directed exploration task, *free exploration*, in which they were instructed to continuously explore the sensing area with the tongue tip, aiming to maintain constant contact and cover as much of the sensing area as possible at a comfortable speed. They completed seven 30-second blocks (3.5 minutes), separated by 30-second rests. Tongue-tip coordinates were recorded. Neurotypical controls completed ten 30-second blocks (5 minutes) [52]. These durations were based on time-to-characterization analyses showing sufficient characterization after 5 minutes of free exploration in neurotypical participants and 3.5 minutes in participants with neurological injury [52,53].

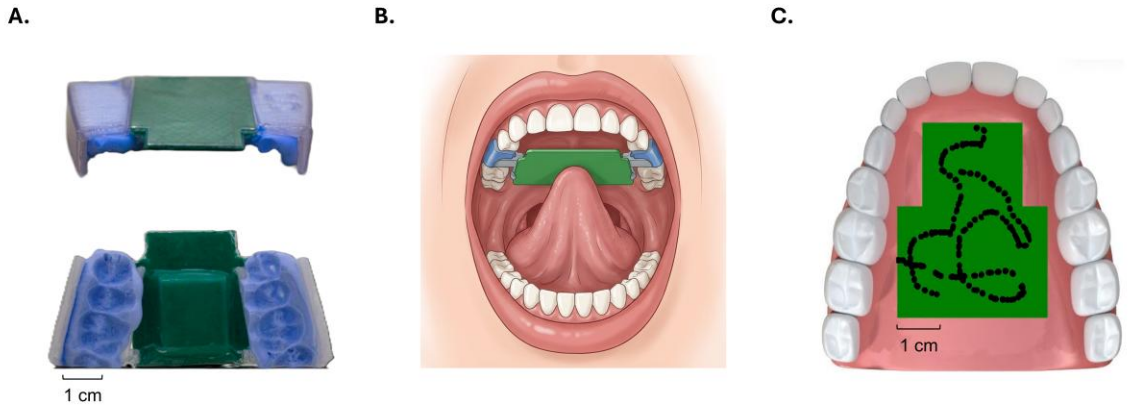


Fig. 1. Tongue-Trackpad device and graphical user interface. (A) Top and back views of the Tongue-Trackpad device. The top illustration shows the green capacitive sensing area that detects tongue-tip contact. The back view shows the region of the device positioned against the palate and secured to the teeth using individualized dental impression material. (B) Schematic illustrating placement of the device against the palate and contact between the tongue tip and the sensing area during the free exploration task. (C) Custom graphical user interface providing real-time visual feedback of the center of tongue-tip contact, represented by a 1-mm-diameter black dot. During the free exploration task, each detected tongue-tip contact location is displayed as a black dot, with the cumulative contact history preserved, allowing users to visualize the movement trajectory.

Analysis

Tongue contact coordinates were post-processed in MATLAB R2025b (The MathWorks, Inc.) using our prior methodology [52]. Data were resampled at 180 Hz, and two-dimensional spatial probability distributions were generated using a 20 x 20-bin histogram, of which 352 bins fell within the sensing area and were retained for analysis. Each bin was normalized by the maximum possible number of contact points, calculated as the total free exploration time (seconds) multiplied by the sampling frequency. To assess the range of

movement, spatial coverage area was calculated as the percentage of bins with values exceeding the fifth percentile of all bin values.

We introduced a uniformity score to quantify how evenly contacts were distributed across the sensing area. The score was derived from the Gini index (range: 0 – 1) and expressed as a percentage, with higher scores indicating a more uniform distribution.

Participant-specific movement patterns were assessed by comparing probability distributions between participant pairs using the coefficient of determination (CoD), *inter-participant similarity value*. For distributions p and q, containing n bins, CoD was defined as:

$$\mathrm{CoD} = 1 - \frac{\sum_{i=1}^{n}(q_i - p_i)^2}{\sum_{i=1}^{n}(q_i - \bar{q})^2}, \quad (1)$$

In this formula p is the predictor distribution, q is the response distribution, and $\bar{q}$ is the mean of q. Because CoD is asymmetric and can be negative for highly dissimilar distributions, it was calculated in both directions for each pair, yielding an asymmetric 19 × 19 similarity matrix (Fig. 3A). Diagonal elements represented *self-similarity values*, calculated by averaging CoD across all pairwise combinations of each participant's 30-second exploration blocks.

Comparison to the capability reference

The neurotypical tongue movement capability reference established in our prior work was used to assess deviations in movement tendencies from neurotypical (Fig. 2A) [52]**.** Two-dimensional differences were calculated by subtracting the neurotypical reference from each participant's probability distribution, following prior approach [52]**.** Bin-wise comparisons were performed against the values from the 16 neurotypical controls. Normality of the neurotypical values was assessed at each bin using the Lilliefors test. Normally distributed bins

were evaluated using a two-tailed z-test ($\alpha = 0.05$), whereas non-normally distributed bins were evaluated against a distribution generated by shuffling each participant's difference histogram 1,000 times, with thresholds at the 2.5th and 97.5th percentiles. Significantly negative and positive bins were classified as *deficit* and *excess bins*, indicating reduced and increased contact relative to neurotypicals, respectively. Deficit and excess areas were quantified as the percentage of the 352 bins.

Group-level movement maps

We compared group-level probability distributions with the neurotypical capability reference to characterize patterns of movement impairment and differences between disorders. For each bin, probability values from participants in each group were compared with the 16 neurotypical values using a Wilcoxon rank-sum test. All bin-wise comparison *P*-values were corrected for multiple comparisons using the Benjamini–Hochberg false discovery rate (FDR). Bins were significant when the FDR-adjusted P-value was below 0.05.

Rank-biserial correlation was calculated at each bin to quantify magnitude and direction of the group difference. Significant bins with negative and positive rank-biserial correlations were classified as deficit and excess bins, respectively, indicating lower or higher tongue-contact probability than neurotypicals. Group-level maps were generated to visualize the spatial distribution of these bins, with color intensity and sign representing magnitude and direction of rank-biserial correlations (Fig. 5). Deficit and excess areas were calculated as percentages of total bins. The same analysis was repeated in stroke survivors. Spatial laterality was quantified using a laterality index:

$$Laterality\ Index\ (\%) = \frac{Bin\ Count_{Right-side} - Bin\ Count_{Left-side}}{Bin\ Count_{Right-side} + Bin\ Count_{Left-side}} * 100\ , \quad (2)$$

Right- and left-side bin counts, $Bin\ Count_{\text{Right-side}}$ and $Bin\ Count_{\text{Left-side}}$, represent the numbers of deficit or excess bins in each half of the map. A laterality index of 0% indicates symmetry; positive and negative values indicate right- and left-sided laterality, respectively, with larger absolute values indicating greater asymmetry.

Velocity and tongue contact area analysis

Secondary metrics were calculated to further characterize movement performance. Velocity was derived by differentiating the spatial data. Mediolateral and anteroposterior velocity probability distributions were represented using 20 × 20 histograms, spanning -16 to 16 $\frac{cm}{s}$ along each axis, following our methodology [53]. Velocity coverage area was calculated as the percentage of 400 bins within each participant's 95th-percentile velocity contour. Deficit and excess areas were determined by bin-wise comparison with the neurotypical velocity reference and expressed as percentages of the 400 bins.

Movement directionality, defined as the orientation of the principal movement axis, was quantified using principal component analysis (PCA) of each participant's two-dimensional velocity data within the 95th-percentile contour. The first principal component (PC1), representing the axis of greatest variability, was expressed relative to the anteroposterior axis from −90° to +90°, with 0° indicating an anteroposterior and ±90° indicating mediolateral orientations. Because PC1 is axial not directional, orientations separated by 180° are equivalent; hence angles were doubled to obtain directional data for circular analysis. Mean orientation, dispersion, and within-group directional consistency were assessed using the Watson–Williams, Brown–Forsythe, and Rayleigh tests, respectively; reported mean orientations were transformed back to the original ±90° scale.

For each participant, median tongue contact area during free exploration was calculated after excluding values beyond 1.5 interquartile ranges from the first or third quartile; medians were used because the data were non-normally distributed (Shapiro–Wilk test).

Tongue Agility Score

A composite score was developed to quantify tongue agility relative to neurotypical controls. The score integrated spatial and velocity coverage, and tongue contact area, as these metrics capture complementary dimensions of movement performance. Each metric was z-score normalized using the mean (μ) and standard deviation (σ) of the neurotypicals. The dimensionless composite score summed spatial and velocity coverage z-scores and subtracted contact area z-score, as greater coverage and smaller contact area reflect neurotypical behavior more closely. By construction, the mean neurotypical score is zero, and lower scores indicate lower tongue agility.

$$Tongue\ Agility\ Score = \frac{A_{spatial\ coverage} - \mu_{NT\ spatial\ coverage}}{\sigma_{NT\ spatial\ coverage}} + \frac{A_{velocity\ coverage} - \mu_{NT\ velocity\ coverage}}{\sigma_{NT\ velocity\ coverage}} - \frac{A_{contact} - \mu_{NT\ contact}}{\sigma_{NT\ contact}}, \quad (3)$$

Where $A_{spatial\ coverage}$ and $A_{velocity\ coverage}$ represent the participant's spatial and velocity coverage areas, respectively, converted from percentages to proportions (range: 0 – 1) by dividing each value by 100. $A_{contact}$ represents the tongue contact (cm^2). For each metric, μ_{NT} and σ_{NT} denote the mean and standard deviation of the neurotypical (NT) group. For ease of interpretation, the composite defined in Equation 3 was uniformly rescaled by the smallest absolute coefficient to obtain the reported Tongue Agility Score in Equation 4. All scores reported here use this rescaled formulation.

Sensitivity analysis assessed ranking robustness to the three metrics equal weighting. Metric weights were varied from 0 to 1 in 0.1 increments while summing to 1, yielding 66 combinations. For each combination, participants were ranked according to scores. Rankings for each combination were compared to equal-weight ranking using Spearman's rank correlation coefficient (ρ).

Statistical Analysis

We assessed normality using the Shapiro–Wilk test. Three-group comparisons used one-way analysis of variance (ANOVA) with Tukey–Kramer post hoc tests when all groups were normal; otherwise, Kruskal–Wallis with Dunn's Holm–Bonferroni-corrected comparisons were used. Two-group comparisons used Welch's *t*-test when both groups were normal and Wilcoxon rank-sum otherwise. Associations between each metric and DDK rate were assessed using Pearson's correlation coefficient when both variables were normally distributed and Spearman's rank correlation coefficient (ρ) otherwise. For Pearson correlations, the coefficient of determination (R^2) was reported. Significance was defined as $P < 0.05$. No data were missing for any variable included in the analyses. To limit analytic bias, analyses were independently repeated by at least two team members.

Results

Quantitative tongue movement analysis revealed individualized movement signatures. Group-level movement maps identified distinct spatial patterns of movement in the two disorders. The apraxia of speech group-level movement map was characterized by centrally concentrated movement surrounded by a broad, continuous bilateral deficit area, 7.21 times greater than the localized, right-lateralized deficit area in dysarthria. In the dysarthria group, several spatial measures were significantly associated with DDK rate. Integration of spatial and

velocity coverage areas, as well as tongue contact area into the Tongue Agility Score yielded a composite measure of movement performance, revealing significant group-level differences.

Disorder Groups Exhibited Reduced Spatial Agility Measures of Movement Range and Uniformity

Participants exhibited individualized movement patterns during self-directed free exploration task (Fig. 2A). Compared with neurotypicals, participants with motor speech disorders showed reduced coverage and movement uniformity (Fig. 2B, C).

We observed significant group differences in spatial tongue agility measures of coverage area (one-way ANOVA, $F(2, 32) = 64.22$, $P < 0.001$) and uniformity score (Kruskal–Wallis, $H(2) = 25.39$, $P < 0.001$). The dysarthria group achieved a mean coverage area of 76.33 ± 10.94% (mean ± SD), corresponding to a 12.97% reduction relative to neurotypicals (87.71 ± 3.75%; Tukey–Kramer, $q(3, 32) = 4.31$, $P = 0.012$) (Fig. 2B). The dysarthria group also exhibited a median uniformity score of 32.81% (IQR: 23.30 to 45.40%), representing a 33.82% reduction relative to controls, whose median uniformity score was 49.58% (IQR: 46.87 to 53.01%; Dunn's test, Holm–Bonferroni-adjusted, $z = -2.49$, $P = 0.026$) (Fig. 2C). A similar pattern was observed in the apraxia of speech group, which exhibited lower spatial coverage area and uniformity score than neurotypicals ($q(3, 32) = 15.93$, $P < 0.001$; $z = -4.99$, $P < 0.001$; respectively). Compared with the dysarthria group, the apraxia of speech group demonstrated a 38.40% reduction in coverage area (mean ± SD: 47.02 ± 12.48%; $q(3, 32) = 10.07$, $P < 0.001$) and a 57.94% reduction in movement uniformity score (median: 13.80%; IQR: 8.58 to 17.03%; $z = -2.12$, $P = 0.034$). These findings indicate that movements were more spatially restricted and less uniformly distributed in participants with apraxia of speech than in those with dysarthria.

Both spatial metrics showed significant associations with DDK rate in the dysarthria group. Greater coverage area and higher uniformity scores were associated with higher DDK rates (Pearson $r(7) = 0.82$, $R^2 = 0.67$, $P = 0.007$; $r(7) = 0.85$, $R^2 = 0.72$, $P = 0.004$; respectively) (Fig. 2B, C).

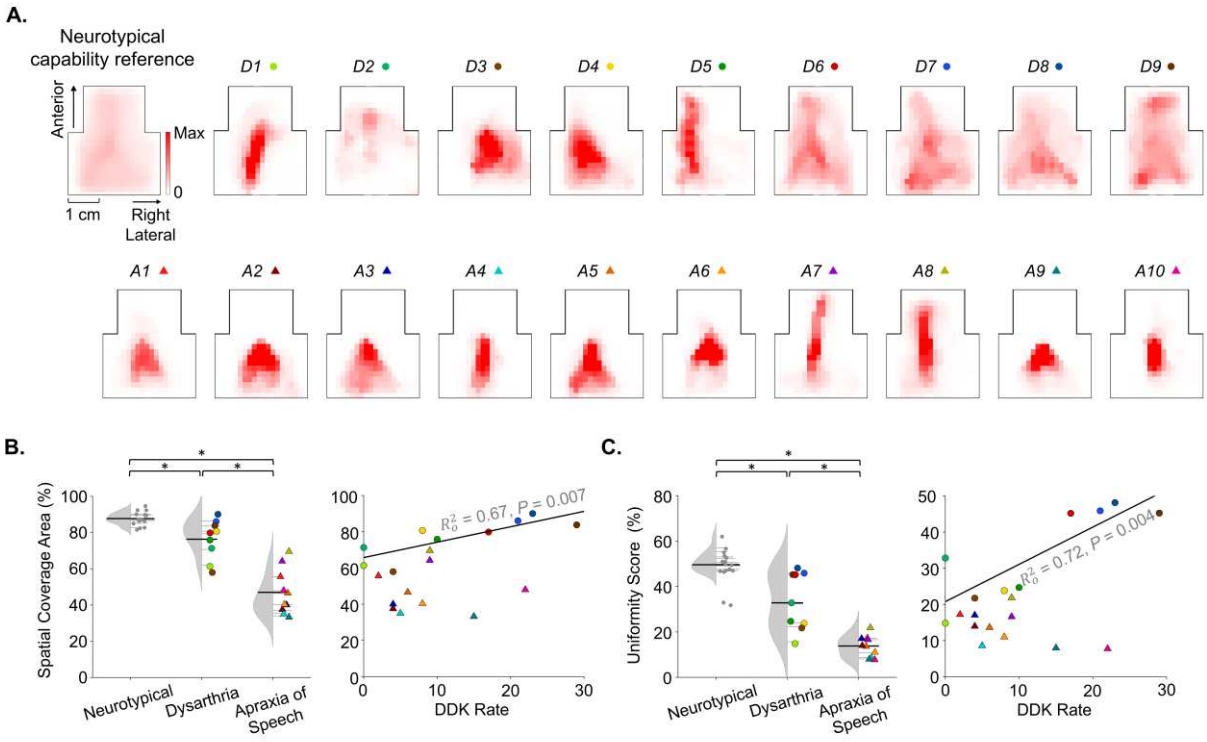


Fig. 2. Tongue agility measures of spatial movement range and uniformity. (A) Neurotypical capability reference (top left) and spatial probability distributions of participants with dysarthria (top row) and apraxia of speech (bottom row). The color bar ranges from white (zero probability) to dark red (maximum probability across all participants). Participant-specific colors and symbols, circles (●) for dysarthria and triangles (▲) for apraxia of speech, are used consistently throughout the manuscript. (B) Violin plots showing the percentage of the sensing area covered by neurotypical, dysarthria, and apraxia of speech groups (left). Violins represent the data distribution; the thick dark line indicates group mean, and colors identify individual participants. Significant differences are indicated by '∗' ($P < 0.05$). Spatial

coverage area for all participants is plotted against the DDK rate (right). The linear regression fit for the dysarthria group is shown as a black line, with corresponding R^2 and *P*-value displayed. (C) Violin plots showing uniformity scores for the neurotypical, dysarthria, and apraxia of speech groups (left). Violins represent the data distribution; the thick dark line indicates group median, and colors identify individual participants. Significant differences are indicated by '*' ($P < 0.05$). Uniformity scores for all participants are plotted against the DDK rate (right). The linear regression fit for the dysarthria group is shown as a black line, with corresponding R^2 and *P*-value displayed. The results indicate that both disorder groups exhibited lower spatial coverage areas and uniformity scores than the neurotypical controls (dysarthria: 12.97%, $P = 0.012$, and 33.82%, $P = 0.026$, respectively; apraxia of speech: 46.39%, $P < 0.001$, and 72.17%, $P < 0.001$, respectively). Higher coverage area and uniformity scores were associated with higher DDK rates in the dysarthria group ($R^2 = 0.67$, $P = 0.007$; $R^2 = 0.72$, $P = 0.004$; respectively).

Tongue Movement Patterns Were More Similar Within the Same Disorder Group Than Between Disorder Groups

Tongue movement patterns were more similar among participants within the same disorder group than between disorder groups (Fig. 3). The inter-participant similarity values within the same disorder comparisons, dysarthria–dysarthria and apraxia of speech–apraxia of speech, had a median value of 0.17 (IQR: -0.91 to 0.40). In contrast, the inter-participant similarity values for between-disorders comparisons, dysarthria–apraxia of speech and apraxia of speech– dysarthria, had a median of -0.07 (IQR: -1.21 to 0.21). Distributions within the same group had significantly higher inter-participant similarity values than between disorders (Wilcoxon rank-sum, $z = 3.52$, $P < 0.001$; Fig. 3B), indicating greater similarity among

movement patterns within the same disorder group and suggesting the presence of movement signatures that differed across the disorders.

In addition, inter-participant similarity values were significantly higher within the apraxia of speech group (median: 0.32; IQR: -0.46 to 0.52) than within the dysarthria group (median: -0.16; IQR: -1.73 to 0.24; $z = 4.80$, $P < 0.001$). This finding indicates greater similarity in spatial movement patterns in apraxia of speech group, whereas the dysarthria group exhibited greater inter-participant variability in movement patterns.

In contrast, self-similarity values did not differ significantly between participants with dysarthria (mean ± SD: 0.12 ± 0.44) and participants with apraxia of speech (mean ± SD: 0.08 ± 0.44; Welch's t-test, $t(16.8) = 0.18$, $P = 0.856$), indicating comparable within-participant consistency of movement patterns across repeated exploration blocks.

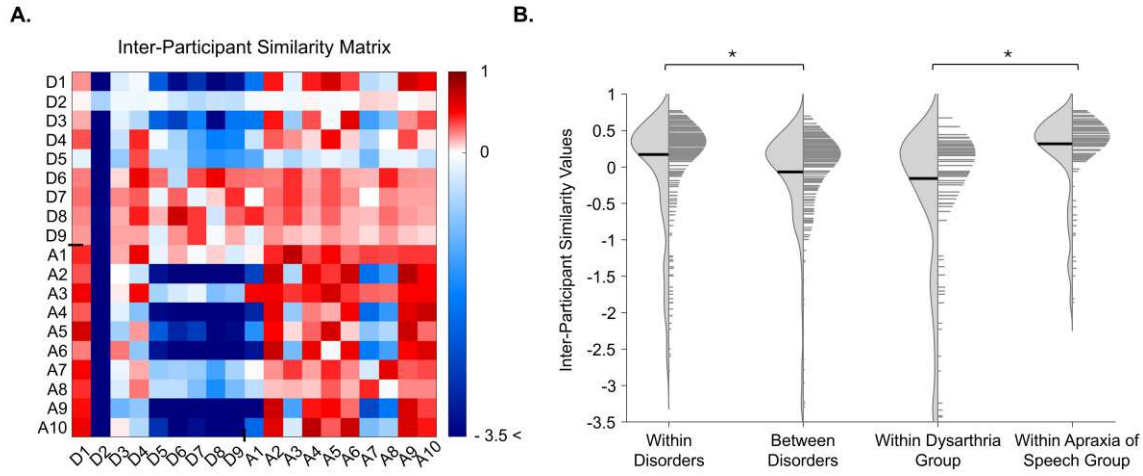


Fig. 3. Inter-participant movement similarity. (A) Spatial probability distribution similarity matrix. The 19 × 19 matrix displays the coefficient of determination (CoD), referred to as the inter-participant similarity value, for each participant pair. Rows represent participants whose distributions were treated as predictors, and columns represent participants whose distributions were treated as responses. Diagonal elements represent self-similarity values calculated from

repeated exploration blocks. Higher similarity values are shown in darker shades of red, with the maximum similarity value set to 1, whereas lower values are shown in darker shades of blue. (B) Violin plots showing inter-participant similarity values within the same disorder comparisons (dysarthria–dysarthria and apraxia of speech–apraxia of speech) versus between disorder comparisons (dysarthria–apraxia of speech and apraxia of speech–dysarthria), as well as comparisons within each disorder group separately. The left half of each violin represents the data distribution; the right half shows the corresponding histogram, and the thick black line indicates the median. Outliers, defined as values more than 1.5 interquartile ranges below the first quartile or above the third quartile, were omitted from the visualization but retained in the statistical analyses. Significant differences are indicated by '∗' ($P < 0.05$). The results indicate that participants' movement distributions were more similar within the same disorder group than between the two disorder groups ($P < 0.001$). The apraxia of speech group also revealed higher similarity values than dysarthria group ($P < 0.001$), indicating greater similarity in spatial movement patterns among participants with apraxia of speech. Self-similarity did not differ between the two disorder groups ($P = 0.856$).

Movement Deficit Patterns Were Individualized and Associated with DDK Rate

Contrasting each participant's spatial probability distribution with the neurotypical capability reference identified regions of significantly under- and over-expressed tongue contact, defined as deficit and excess areas, respectively (Fig. 4A). Deficit areas differed significantly among the three groups (Kruskal–Wallis, $H(2) = 24.20$, $P < 0.001$). In the dysarthria group, deficit area was significantly associated with DDK rate (Fig. 4B).

The dysarthria group exhibited a median deficit area of 9.94% (IQR: 0.78 to 14.99%), 35.50 times the median of neurotypical controls (median: 0.28%; IQR: 0 to 0.71%; $z = 2.16$, $P = 0.038$) (Fig. 4B). The apraxia of speech group showed an even larger median deficit area of 20.45% (IQR: 19.03 to 23.30%), 2.06 times that of the dysarthria group ($z = 2.35$, $P = 0.038$) and 73.04 times the median of neurotypical controls ($z = 4.91$, $P < 0.001$). In contrast, excess area did not differ significantly among the groups (one-way ANOVA, $F(2, 32) = 1.53$, $P = 0.233$), averaging 7.48 ± 4.72% (mean ± SD) in the dysarthria group and 6.19 ± 1.61% in the apraxia of speech group. In the dysarthria group, larger deficit areas were associated with lower DDK rates (Pearson $r(7) = -0.94$, $R^2 = 0.88$, $P < 0.001$) (Fig. 4B).

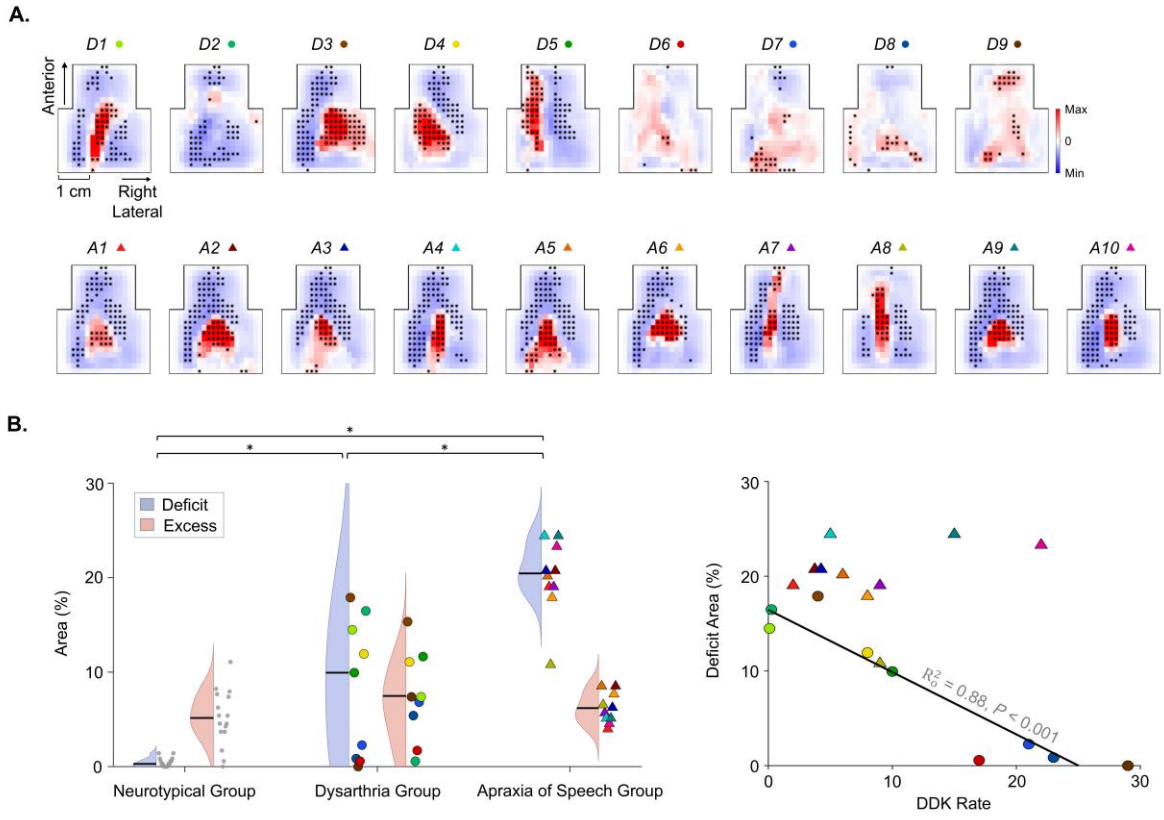


Fig. 4. Individualized tongue movement areas of under- and over-expressed movement relative to the neurotypical capability reference. (A) Individualized contrast maps relative to the neurotypical capability reference for participants with dysarthria (top row) and apraxia of speech (bottom row). The color bar ranges from dark blue (negative differences) to dark red

(positive differences). Bins exhibiting significantly under-expressed tongue contact (deficit) or over-expressed tongue contact (excess) relative to neurotypical controls are marked with '∗' ($P < 0.05$). Participant-specific colors and symbols, circles (●) for dysarthria and triangles (▲) for apraxia of speech, are used consistently throughout the manuscript. (B) Violin plots showing the percentage of deficit (blue) and excess (red) areas for the neurotypical, dysarthria, and apraxia of speech groups (left). Violins represent the data distributions, and the thick dark line indicates the group median for deficit area and the group mean for excess area. Colors identify individual participants. For visualization, deficit-area values more than 1.5 interquartile ranges below the first quartile or above the third quartile were omitted, whereas excess-area values more than 1.5 standard deviations from the group mean were omitted; all values were retained in the statistical analyses. Significant differences are marked with '∗' ($P < 0.05$). Individual participants' deficit areas are plotted against DDK rate (right). The linear regression fit for the dysarthria group is shown as a black line, with the corresponding R^2 and *P*-value displayed. The results indicate that deficit area was significantly greater in the apraxia of speech group than in the dysarthria group ($P = 0.038$) and was significantly greater in both disorder groups than in neurotypical controls (dysarthria: $P = 0.038$; apraxia of speech: $P < 0.001$). Within the dysarthria group, higher DDK rates were associated with smaller deficit areas ($R^2 = 0.88$, $P < 0.001$).

Spatial Movement Patterns Differed Between Dysarthria and Apraxia of Speech

Group-level analysis revealed distinct spatial organizations of tongue movement impairment in dysarthria and apraxia of speech (Fig. 5). In the dysarthria group-level map, deficit area accounted for 10.80% of the sensing area (FDR-adjusted $P < 0.05$; r_{rb} range = -1.00 to -0.68) (Fig. 5). In contrast, deficit areas in the apraxia of speech group-level map

encompassed 77.84% of the sensing area, 7.21 times the area of dysarthria (FDR-adjusted $P <$ 0.05; r_{rb}range = -1.00 to -0.38). Deficit areas in the dysarthria group-level map were predominantly right-lateralized, with a laterality index of 68.42%, whereas the apraxia of speech group-level map exhibited a broader, continuous bilateral deficit pattern, with a laterality index of 5.11%. The apraxia of speech group-level map also exhibited a left-lateralized excess area encompassing 5.40% of the sensing area (FDR-adjusted $P < 0.05$; r_{rb} range = 0.51 to 1.00; laterality index = -57.89%).

These differences in spatial organization were preserved when the analysis was restricted to stroke survivors within each group (Fig. 5, insets). Despite the predominance of unilateral stroke lesions, the broad bilateral deficit pattern remained evident in the apraxia of speech group-level map. These results suggest that the observed differences in spatial movement patterns were not explained solely by lesion laterality.

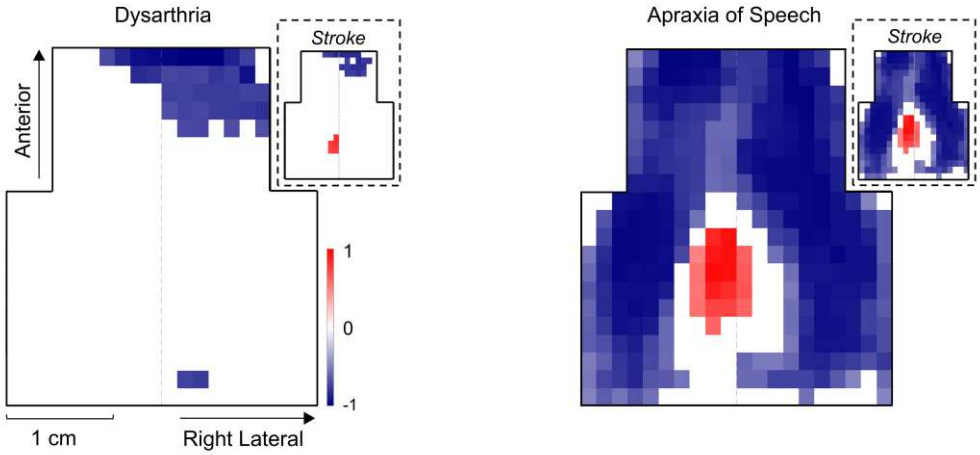


Fig. 5. Group-level movement maps in dysarthria and apraxia of speech. Group-level movement maps are shown for dysarthria (left) and apraxia of speech (right), with inset maps derived from analyses restricted to stroke survivors. Color intensity indicates the magnitude of the rank-biserial correlation values for statistically significant bins (FDR-adjusted $P < 0.05$). Blue bins represent deficit bins, defined as significantly under-expressed tongue contact probability relative to neurotypical controls, whereas red bins represent excess bins, defined as

significantly over-expressed tongue contact relative to neurotypical controls. The maps revealed distinct spatial organizations of tongue movement impairment, with dysarthria exhibiting a right-lateralized deficit pattern covering 10.80% of the sensing area, whereas apraxia of speech exhibited a bilateral deficit pattern covering 77.84% of the sensing area, 7.21 times greater than that observed in dysarthria, together with a left-lateralized excess region covering 5.40% of the sensing area.

Velocity and Contact-Area Metrics Revealed Additional Group Differences in Tongue Agility

To further characterize tongue agility, movements were evaluated in the velocity domain. Participants exhibited individualized velocity patterns, as reflected in velocity probability distributions and contrast maps (Fig. 6A). Several velocity measures differed among groups (Fig. 6B-D).

Velocity coverage area differed significantly across groups (Kruskal–Wallis, $H(2) = 7.06$, $P = 0.029$; Fig. 6B). The dysarthria group had a median velocity coverage area of 25.09% (IQR: 10.51 to 26.13%), which did not differ from that of neurotypical controls (median: 31.02%; IQR: 14.52 to 48.10%; $z = -1.56$, $P = 0.240$). In contrast, the apraxia of speech group had a median velocity coverage area of 11.07% (IQR: 7.68 to 22.35%), representing a 64.31% reduction relative to neurotypical controls ($z = -2.58$, $P = 0.030$).

Both disorder groups exhibited significantly larger velocity deficit areas than neurotypical controls (Kruskal–Wallis, $H(2) = 13.45$, $P = 0.001$; dysarthria: median = 9.00%; IQR: 2.75 to 16.06%; $z = 2.30$, $P = 0.042$; apraxia of speech: median = 14.12%; IQR: 12.75 to 15.75%; $z = 3.51$, $P = 0.001$) (Fig. 6C). However, velocity deficit area did not differ between the two disorder groups ($z = 0.99$, $P = 0.324$). Velocity excess area also differed significantly

among groups (one-way ANOVA, $F(2, 32) = 4.11$, $P = 0.026$). Post hoc comparisons indicated that the dysarthria group did not differ from neurotypical controls (mean ± SD: 4.50 ± 3.88%; $q(3, 32) = 1.07$, $P = 0.730$), whereas the apraxia of speech group exhibited a lower velocity excess area than controls (1.62 ± 0.99%; $q(3, 32) = 4.03$, $P = 0.020$). In the dysarthria group, greater velocity excess area was associated with higher DDK rate (Pearson $r(7) = 0.73$, $R^2 = 0.53$, $P = 0.025$). No significant associations with DDK rate were observed for the other velocity metrics.

Mean movement directionality did not differ significantly among groups (Watson–Williams test, $F(2, 32) = 0.54$, P = 0.586); however, directionality dispersion differed (Brown–Forsythe test, $F(2, 32) = 5.07$, $P = 0.012$) (Fig. 6D). This difference was driven by greater dispersion in the dysarthria group than in the apraxia of speech group ($F(1, 17) = 8.74$, Holm–Bonferroni-adjusted $P = 0.027$). Mean directionality was +17.39 ° in the dysarthria group, +8.98 ° in the apraxia of speech group, and +3.08 ° in neurotypical controls. Consistent with the dispersion analysis, the dysarthria group did not exhibit significant within-group directional consistency (Rayleigh test: $R = 0.275$, $Z = 0.68$, $n = 9$, $P = 0.519$), indicating greater inter-participant variability in movement directionality and the absence of a preferred group level orientation. In contrast, directional consistency was observed in the apraxia of speech group ($R = 0.985$, $Z = 9.71$, $n = 10$, $P < 0.001$) and neurotypical controls ($R = 0.765$, $Z = 9.36$, $n = 16$, $P < 0.001$).

To further characterize tongue agility, tongue contact area was evaluated during the free exploration task. Contact area differed significantly among groups (one-way ANOVA, $F(2, 32) = 37.92$, $P < 0.001$) (Fig. 6E). The dysarthria group had a mean contact area of 1.48 ± 0.62 cm^2 (mean ± SD), which was comparable to neurotypical controls (mean ± SD: 1.19 ± 0.22 cm^2; $q(3, 32) = 1.82$, $P = 0.414$). In contrast, the apraxia of speech group exhibited a mean

contact area of 3.07 ± 0.81 cm², 2.58 times the mean of neurotypical controls ($q(3, 32) = 11.98$, $P < 0.001$) and 2.07 times that of the dysarthria group ($q(3, 32) = 8.87$, $P < 0.001$). DDK rate was not significantly correlated with contact area in either group.

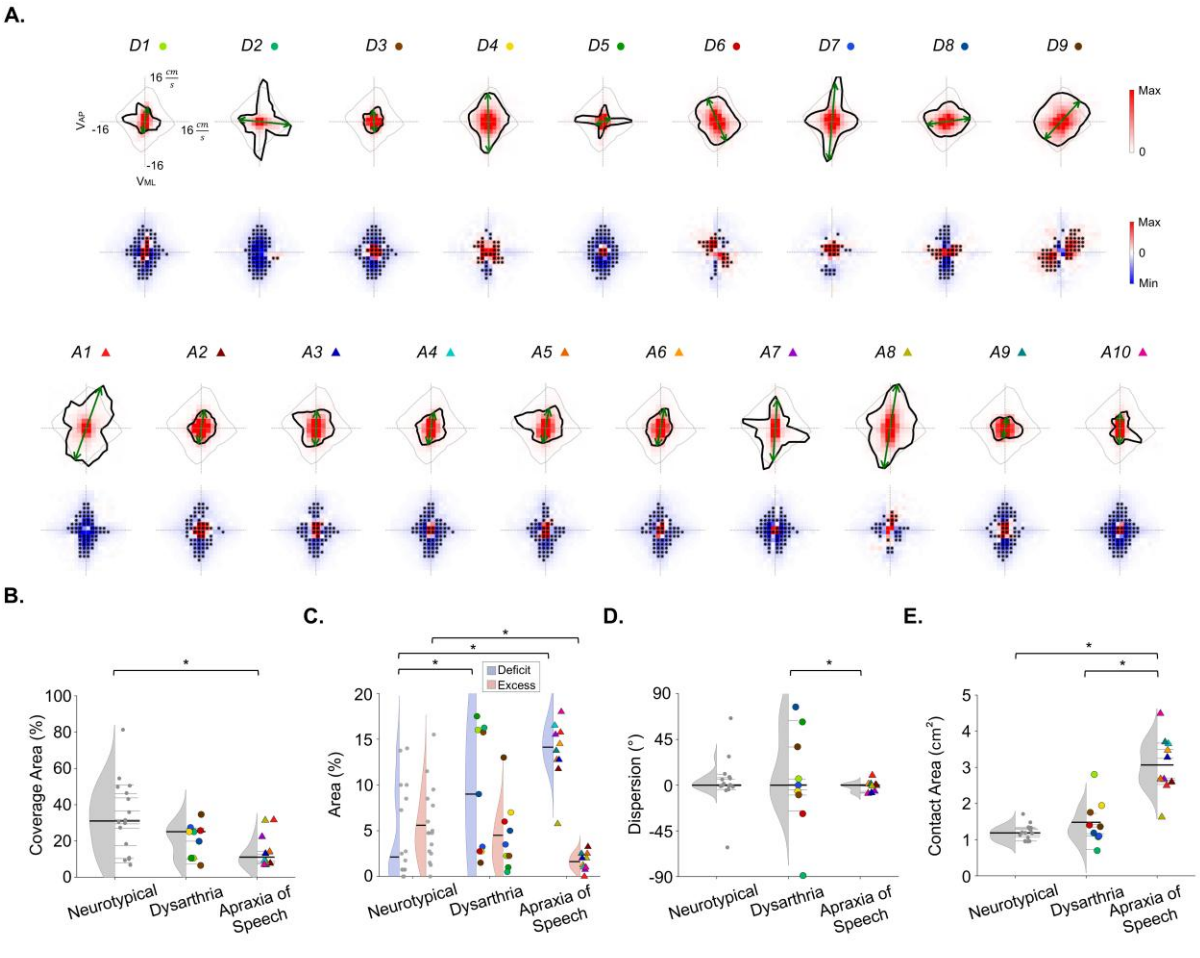


Fig. 6. Tongue agility measures in the velocity-domain and tongue contact area. (A) Participant-specific velocity probability distributions in the anteroposterior (AP) and mediolateral (ML) directions for participants with dysarthria (●) and apraxia of speech (▲). The 95th-percentile velocity contour is shown in bold black, neurotypical capability reference contour in gray, and movement directionality in green. Color bar ranges from white (zero probability), to dark red (maximum probability across all participants). Contrast maps beneath each participant's probability distribution show deviations from the neurotypical capability reference, ranging from dark blue (negative) to dark red (positive). Asterisks (*) mark bins

with significantly lower (deficit) or higher (excess) velocity probability relative to neurotypical controls ($P < 0.05$). Violin plots show (B) velocity coverage area (bins percentage within the 95th-percentile velocity contour); (C) velocity-deficit (blue) and velocity-excess (red) areas; (D) directionality dispersion (°) (each participant's deviation from the group median direction); and (E) median tongue contact area (cm²) for each group. Thick black lines within the violins indicate group median (B–D) and mean (E). Colors identify individual participants. Asterisks '∗' indicate significant differences ($P < 0.05$). The results demonstrate individualized velocity movement patterns, with the apraxia of speech group exhibiting a median velocity coverage area 64.31% lower than that of neurotypical controls ($P = 0.030$). Both disorder groups exhibited a significantly greater velocity deficit area than neurotypical controls (dysarthria: $P = 0.042$, apraxia of speech: $P = 0.001$), while the apraxia of speech group exhibited a significantly smaller velocity excess area than controls ($P = 0.020$). The central tendency of movement directionality did not differ among groups ($P = 0.586$); however, movement directionality dispersion was significantly greater in the dysarthria than in the apraxia of speech group ($P = 0.027$). Additionally, the apraxia of speech group exhibited a significantly greater tongue contact area than both the neurotypical controls and the dysarthria group (both $P < 0.001$).

Tongue Agility Scores Differed Across Motor Speech Disorders and Neurotypical Groups

The Tongue Agility Score, integrating spatial coverage area, velocity coverage area, and tongue contact area, provided a composite measure of movement performance relative to neurotypical controls:

548 $$Tongue\ Agility\ Score\ = 5.83\ A_{spatial\ coverage} + 1.07\ A_{velocity\ coverage}\ - A_{contact} - 4.29$$

549 , (4)

550 DDK rates did not differ significantly between the dysarthria and apraxia of speech
551 group (12.4 ± 10.5 vs. 8.4 ± 6.0 syllables, respectively, Welch's t-test, $P = 0.330$), Tongue
552 Agility Scores differed significantly among the three groups (one-way ANOVA, $F(2, 32) =$
553 59.82, $P < 0.001$) (Fig. 7B). By design, scores in the neurotypical control group were centered
554 at zero (SD = 0.41). The dysarthria group had a significantly lower mean score of -1.10 ± 1.16
555 than the neurotypical control group ($q(3, 32) = 3.65$, $P = 0.038$). The apraxia of speech group
556 exhibited the lowest mean score, -4.45 ± 1.49, which was significantly lower than that of
557 neurotypical controls ($q(3, 32) = 15.31$, $P < 0.001$) and the dysarthria group ($q(3, 32) = 10.12$,
558 $P < 0.001$).

559 Sensitivity analysis across metric-weight combinations indicated that participant
560 rankings were robust to the selection of metric weights. Rankings across weight combinations
561 were highly correlated with those obtained using the equal-weight formulation in Eq. 3 (median
562 Spearman's $\rho = 0.975$), and the relative ordering of the three groups was preserved across
563 weight combinations.

564 In the dysarthria group, higher Tongue Agility Scores were associated with higher DDK
565 rates (Pearson $r(7) = 0.70$, $R^2 = 0.49$, $P = 0.037$), whereas no correlation was observed in the
566 apraxia of speech group ($r(8) = -0.34$, $R^2 = 0.12$, $P = 0.334$).

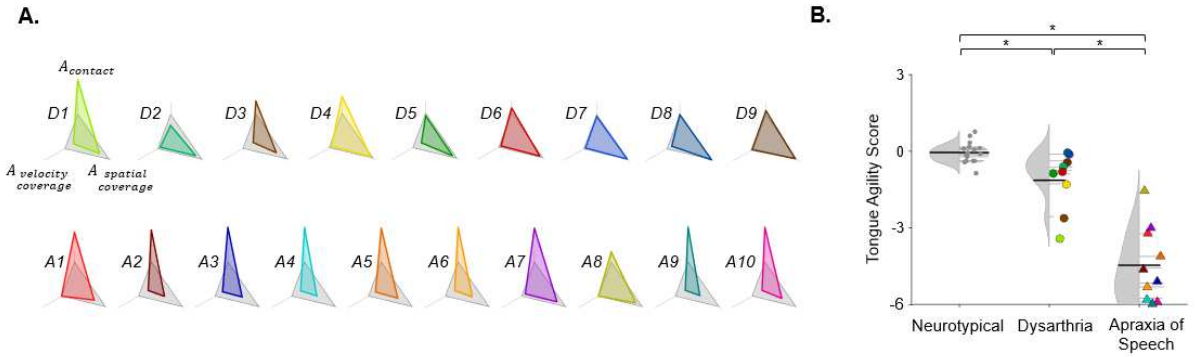


Fig. 7. Composite Tongue Agility Score. (A) Individual tongue agility metric profiles for participants with dysarthria (top row) and apraxia of speech (bottom row). Each plot displays the three components of the Tongue Agility Score: spatial coverage area, velocity coverage area, and tongue contact area. Each axis is scaled to the maximum value observed in the neurotypical group. The gray-shaded triangle indicates the neurotypical control group mean for each metric. Participant-specific colors are used consistently throughout the manuscript. (B) Violin plot showing Tongue Agility Scores for the neurotypical, dysarthria, and apraxia of speech groups. The thick black line within the violin indicates the group mean, and colors identify individual participants. Significant differences are marked with '$*$' ($P < 0.05$). Tongue Agility Scores were significantly lower in both motor speech disorder groups than in neurotypical controls (dysarthria: $P = 0.038$; apraxia of speech: $P < 0.001$) and were significantly lower in the apraxia of speech group than in the dysarthria group ($P < 0.001$).

Discussion

In this preliminary study, our findings supported the hypothesis that quantitative analysis of self-directed exploratory tongue movement can characterize individual movement signatures, identify group-level differences in movement characteristics, and provide objective measures of tongue-movement performance.

Individualized spatial and velocity probability distributions captured participant-specific signatures, while spatial group-level movement maps identified distinct organizations of movement in dysarthria and apraxia of speech. Integrating spatial coverage area, velocity coverage area, and tongue contact area into the Tongue Agility Score provided a composite measure of overall tongue motor performance relative to neurotypical controls. Together, these complementary outputs characterize where movement is limited and how far overall performance deviates from neurotypicals. Despite the limited sample size, the significant individualized and group-level differences in movement patterns support the feasibility of this framework as an objective, multidimensional approach for characterizing individualized performance.

Both groups exhibited more restricted and less uniform movement than neurotypical controls, with greater reductions and more centrally concentrated movement in apraxia of speech. In contrast, dysarthria showed greater inter-participant variability. Spatial coverage area and uniformity score were associated with DDK rate in dysarthria with lower rates corresponding to more restricted and less uniform movement. Although these associations do not establish causality, they support the potential clinical relevance of these metrics as quantitative measures of movement performance.

Movement patterns were more similar within each disorder group than across groups, with greater inter-participant similarity observed in apraxia of speech. In contrast, comparable self-similarity values in each group indicated within-participant movement consistency. The greater inter-participant variability observed in dysarthria may reflect heterogeneity in the underlying neuromotor impairments and residual function. In contrast, the greater similarity and spatially constrained movement patterns observed in apraxia of speech may be consistent with disruption in planning and organization of voluntary movements. Although these

interpretations remain preliminary, the distinct movement organizations observed across the groups may reflect differences in the underlying motor-control mechanisms associated with the disorders.

Individualized contrast maps revealed participant-specific deviations from neurotypical capability reference. Both disorder groups exhibited significantly enlarged deficit areas indicating spatial evidence of restricted movement. Importantly, the disorders differed in the spatial organization of deficits, with apraxia of speech depicting centrally concentrated movement surrounded by broad bilateral deficits. Overall excess area did not differ among groups, indicating that impairment was characterized primarily by reduced expression of movement within regions rather than increased movement in specific regions. Deficit area was associated with DDK rate in dysarthria, suggesting that greater movement restriction corresponded to lower DDK performance. Unlike DDK rate, which reflects speech-token production rate, deficit mapping pinpointed where movement limitations occurred. This spatial information may enable more detailed characterization of individualized impairment and reveal differences in movement patterns among individuals with comparable DDK rates.

A notable finding was the distinct group-level spatial organization of movement impairment in dysarthria and apraxia of speech. These patterns revealed differences in how deficit areas were organized and provided information beyond global measures of function. It is important to note that these are behaviorally defined disorders, whereas stroke and TBI are neurological injuries that can manifest distinctly. Hence, we repeated the analysis in stroke survivors to examine whether the observed patterns could be explained by injury type or lesion laterality. The broad bilateral deficit pattern in apraxia of speech persisted despite predominantly unilateral stroke lesions. The persistence of these patterns suggests that the observed differences were not explained solely by injury type differences and persisted despite

differences in lesion laterality. Larger cohorts with detailed lesion characterization are needed to determine the neuroanatomical basis of these patterns.

An informative observation emerged for one participant initially classified as having apraxia of speech whose spatial movement distribution exhibited characteristics more similar to those observed in the dysarthria group. This finding prompted further review of the clinical classification, and subsequent clinical evaluation resulted in a diagnosis of dysarthria. Following clinical re-evaluation, the participant was reclassified before statistical analysis; accordingly, the final group sizes of nine participants with dysarthria and ten with apraxia of speech reflect the corrected diagnoses, and all analyses reported here were performed using these final classifications. Although a single case cannot establish diagnostic validity, it illustrates how spatial movement maps may reveal patterns that warrant closer clinical examination. The intuitive visualization of individualized movement distributions may therefore facilitate data interpretation and could ultimately serve as an adjunct to clinical assessment, pending validation in larger, independently diagnosed cohorts.

One advantage of the free exploration task is that it allows individualized movement strategies to emerge as it is not restricted to predetermined trajectories; thus, participant-specific patterns reflect available movement range and control. Although such variability was expected, disorder-associated differences still emerged, suggesting that free exploration captures both individual movement characteristics and shared disorder-related features. Deficit and excess regions in some neurotypical participants may also reflect the individual's variability as well as the visual feedback design, in which repeated contacts did not increase feedback intensity. Whether progressively darkening the display with repeated contact promotes more uniform exploration remains to be determined.

Although the free exploration task did not explicitly constrain exploration velocity, distinct velocity patterns emerged, revealing dynamic aspects of movement beyond spatial exploration. Reduced velocity coverage, particularly in apraxia of speech, and greater velocity deficit areas in both disorder groups suggest that impairment affects not only where the tongue moves but also the range of velocities it can generate. Directionality analysis further showed that dispersion differed across disorders, with dysarthria characterized by greater inter-participant variability and apraxia of speech by more consistent movement directions. Mean movement direction did not differ among groups, indicating that the primary difference was variability rather than a systematic shift in preferred direction. Thus, velocity-domain analysis provided complementary information beyond spatial measures and revealed additional differences between disorders.

Tongue contact area provided a complementary measure of interaction with the sensing area. As participants received no visual feedback on contact area, these differences emerged independently of explicit task design. Contact area was comparable between the dysarthria and neurotypicals groups but substantially greater in the apraxia of speech group, suggesting that this metric captures aspects of impairment not reflected by spatial or velocity measures.

Spatial, velocity, and tongue contact measures captured complementary aspects of motor performance, supporting their integration into the Tongue Agility Score as a multidimensional measure of overall tongue movement relative to neurotypical performance. With equal weighting and standardization to neurotypical controls, control scores were centered at zero, whereas disorder groups showed negative mean scores, reflecting reduced movement performance. Score distributions differed significantly among groups, suggesting that integrating complementary movement features may provide a more comprehensive representation of group differences than individual metrics alone. Additionally, participant

rankings were robust to changes in metric weights, indicating that the score reflected underlying performance rather than the specific weighting scheme.

The value of this multidimensional approach was further evident when compared with DDK rate. Although DDK rate did not differ between disorder groups, tongue agility measures revealed significant group-level differences across multiple metrics. Higher Tongue Agility Scores were associated with higher DDK rates in the dysarthria group; however, the two measures captured distinct aspects of motor performance. DDK primarily reflects functional speech-token production rate, whereas the Tongue Agility Score integrates spatial, velocity, and contact characteristics of movement. This distinction was evident in participant pairs D1–D2, A2–A3, and A7–A8, who exhibited comparable DDK rates but distinct agility metrics (Fig. 7A) and distribution maps (Figs. 2A and 4A). These findings suggest that conventional quantitative measures may overlook differences in motor performance and highlight the value of multidimensional assessment for revealing features obscured by a single functional measure.

Importantly, the Tongue Agility Score is intended as a composite measure of movement performance relative to neurotypical controls rather than a diagnostic classifier. Because the score is derived from scalar measures, it does not preserve the spatial organization of movement captured by individualized contrast maps. The two outputs are therefore complementary, with the Tongue Agility Score summarizing overall performance, whereas individualized contrast maps identify where movement deficits occur. Together, they provide a more comprehensive quantitative characterization of tongue motor performance.

The methodology introduced here could provide a quantitative baseline measure of tongue agility. Our previous work in individuals with dysarthria demonstrated improvements in tongue agility metrics following a targeted visual feedback intervention [53,60].

Accordingly, repeated assessments could quantify changes in motor performance and monitor response to therapy.

A limitation of this study is that the neurotypical group was significantly younger (mean ± SD: 25.7 ± 5.6 years) than both the dysarthria (42.1 ± 16.0 years, $P = 0.015$) and apraxia of speech groups (55.8 ± 16.6 years, $P < 0.001$), whereas age did not differ significantly between the two disorder groups ($P = 0.086$). Thus, some differences relative to neurotypical controls may partly reflect age-related changes in motor performance rather than neurological impairment alone. These findings should therefore be interpreted cautiously and validated in larger, more diverse, age-matched cohorts to better isolate disorder-related effects from aging. However, because both disorder groups were compared with the same neurotypical cohort, age differences are less likely to account for the distinct patterns observed between dysarthria and apraxia of speech.

Conclusions

This study demonstrates the feasibility of quantifying individualized tongue movement characteristics and identifying group-level differences among individuals with dysarthria, individuals with apraxia of speech, and neurotypical controls. These findings support the use of probability-based movement analysis and multidimensional metrics to objectively characterize tongue motor performance. With further validation in larger, more diverse cohorts, this framework may serve as an adjunct to clinical assessment, longitudinal monitoring, and targeted rehabilitation, while supporting future investigations of the neural mechanisms underlying tongue motor control.

List of abbreviations

ANOVA: Analysis of variance

AP: Anteroposterior

CCTS: Center for Clinical and Translational Science

CoD: Coefficient of determination

DDK: Diadochokinetic

FDR: False discovery rate

HTML: Hypertext Markup Language

IQR: Interquartile range

IRB: Institutional Review Board

ML: Mediolateral

PC1: First principal component

PCA: Principal component analysis

SD: Standard deviation

SLP: Speech-language pathologist

SRALab: Shirley Ryan AbilityLab

TBI: Traumatic brain injury

Declarations

Ethics approval and consent to participate

Participants with motor speech disorders were recruited under a protocol approved by the Northwestern University Institutional Review Board (protocol number: STU00214010), and all provided informed consent before participation. Neurotypical control data were obtained from a previous study approved by the University of Illinois Chicago Institutional Review Board (protocol number: 2017-0550), in which all participants provided informed consent.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analysed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Competing interests

Hananeh Esmailbeigi is an inventor of the Tongue-Trackpad device used in this study. The technologies underlying the device were released by the University of Illinois to Hananeh Esmailbeigi for potential commercialization. The other authors declare that they have no competing interests.

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Authors' contributions

A.Z. contributed to data collection and analysis, prepared the figures, and contributed to manuscript writing. V.B. contributed to study design, development of the data acquisition system and algorithms, data collection and analysis, and manuscript writing. L.D.S. contributed to data collection, analysis, initial figure preparation and writing. K.C. contributed to study design, development of the data acquisition system and algorithms, data collection and analysis, and helped with manuscript writing. E.B. contributed to study design, provided clinical expertise, directed participant recruitment, and participated in data collection and analysis, and writing. L.C. provided clinical expertise, contributed to data analysis, and writing. J.P. contributed to study design, data analysis, and writing. H.E. led the study design, development of the data acquisition system and algorithms, recruitment, manuscript writing,

and supervised data analysis and interpretation. All authors reviewed and approved the final manuscript and agreed to be accountable for all aspects of the work.

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