BMJ Open

Integrated acoustic and respiratory biomarkers of dysarthria in acquired brain injury: protocol for a cross-sectional study

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To cite: Argoubi A, Díez García R, Hernández Moreda B, *et al*. Integrated acoustic and respiratory biomarkers of dysarthria in acquired brain injury: protocol for a cross-sectional study. *BMJ Open* 2026;**16**:e115669. doi:10.1136/bmjopen-2025-115669

► Prepublication history and additional supplemental material for this paper are available online. To view these files, please visit the journal online (https://doi.org/10.1136/bmjopen-2025-115669).

Received 19 December 2025
Accepted 07 April 2026

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ABSTRACT

Introduction Dysarthria secondary to acquired brain injury (ABI) is a motor speech disorder characterised by impaired coordination of the respiratory, phonatory and articulatory subsystems, with a negative impact on communication and quality of life. Previous studies have largely examined these components separately, which limits understanding of their functional interactions. An integrated approach combining respiratory and acoustic–articulatory measures may improve the characterisation of dysarthria and support the identification of objective markers of severity. The aim of this study is to jointly analyse respiratory function and speech acoustic parameters in adults with ABI and to examine their association with the presence and severity of dysarthria.

Methods and analysis A cross-sectional observational study will be conducted in adults with a confirmed diagnosis of ABI, both with and without dysarthria, with an estimated sample size of 97–101 participants. The protocol will include collection of sociodemographic and clinical data, cognitive assessment using the Mini-Mental State Examination, swallowing assessment using the Eating Assessment Tool-10, acoustic and aerodynamic analyses following standardised procedures, respiratory function assessment using spirometry and perceptual evaluation of dysarthria using the Frenchay Dysarthria Assessment-2. Between-group comparisons and multivariate analyses will be performed to examine associations between respiratory, acoustic, aerodynamic and dysarthria severity measures.

Ethics and dissemination The study has been approved by the Ethics Committee for Research with Medicines of the Salamanca Health Area (CEIm PI 2025 04 1897-TD) and will be conducted in accordance with the Declaration of Helsinki. Written informed consent must be provided by all participants prior to inclusion. Findings will be disseminated at scientific conferences and in peer-reviewed journals. Anonymised data will be made available following publication of the main results, in compliance with data protection regulations.

Trial registration number NCT07435155.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study adopts an integrated phonorespiratory approach, combining acoustic, aerodynamic and respiratory measures to characterise dysarthria in people with acquired brain injury (ABI), addressing a significant gap in literature.
⇒ The integration of objective instrumental biomarkers with a structured perceptual assessment, the Frenchay Dysarthria Assessment-2 (FDA-2), allows for multidimensional characterisation with potential clinical applicability in neurorehabilitation.
⇒ The explicit identification of exposure variables, outcome, confounding factors and possible effect modifiers reinforces the internal validity of the study.
⇒ Convenience sampling may introduce selection bias and limit the generalisation of findings to other populations with ABI.
⇒ The FDA-2 has not been fully validated in Spanish-speaking populations, which should be considered when interpreting perceived severity.

INTRODUCTION

Dysarthria is one of the most common and disabling sequelae of acquired brain injury (ABI), yet its pathophysiology remains incompletely understood. Most research to date has examined voice and respiratory function separately, treating them as isolated systems and focusing independently on acoustic, aerodynamic, intelligibility or respiratory measures. This fragmented approach hinders understanding of how phono-respiratory alterations interact to affect speech production and limits interpretation of dysarthria as an integrated phenomenon. Consequently, there is a conceptual gap that impedes the development of comprehensive explanatory models and underscores the need for studies analysing both systems together to elucidate the biomechanics of dysarthria in ABI.

In this context, dysarthria is recognised as a speech disorder caused by the alteration of the cerebral neuromotor circuits responsible for coordinating the movements involved in speech production. This alteration

compromises the sensorimotor integration necessary to control breathing, phonation, velopharyngeal closure, and tongue and jaw movements, causing speech modifications that vary according to the subsystems affected.[1]

In epidemiological terms, strokes, traumatic brain injuries (TBI) and cranial nerve paralysis are the most common causes of dysarthria.[2] In patients with stroke, between 20 and 42% develop a non-progressive form in later stages,[3] while in severe TBI the prevalence reaches approximately 30%, with symptoms persisting for months.[4] Clinically, these alterations manifest themselves through reduced and concentrated articulatory space, limited tongue movements, greater mandibular and labial restriction, slower articulation and uncoordinated gestures.[5 6]

Traditionally, perceptual speech assessment has significant limitations: subjectivity, poor reproducibility and low sensitivity to detect subtle changes, which reduces its usefulness in clinical monitoring and research.[7] These limitations prompted the development of instrumental methods to objectively assess speech motor performance. Among these, acoustic analysis has been shown to provide robust quantitative measures that complement perceptual assessment and allow for more accurate characterisation of motor disorders.[7 8] This approach has led to multiparametric models that integrate segmental and suprasegmental domains, offering new perspectives on the relationship between neuromotor systems, articulatory execution and communicative function.[9]

However, the emphasis placed on the acoustic component has overshadowed the role of respiratory function, despite it being the biomechanical substrate that supports speech production. Respiratory alterations characteristic of ABI, such as decreased inspiratory and expiratory muscle strength, reduced forced vital capacity (FVC) or restrictive ventilatory patterns, condition the availability of air for phonation and directly modify the stability, intensity and duration of the sounds emitted.[10–12] From a biomechanical standpoint, expiratory airflow constitutes the driving aerodynamic force underlying vocal fold vibration, and insufficient or unstable expiratory control compromises subglottic pressure modulation, reducing vibratory regularity and producing measurable acoustic instability. In this framework, respiratory dysfunction operates not as a parallel deficit but as a primary determinant of acoustic and phonatory control in ABI.[10–12] Intervention evidence supports this interaction: improvements in FVC, forced expiratory volume in 1 s (FEV1), maximum voluntary ventilation (MVV) and maximum respiratory strength translate into functional changes in speech production, underscoring the central role of breathing in dysarthria rehabilitation.[13]

These findings suggest that acoustic parameters alone do not fully capture the complexity of motor speech disorders in ABI.[14 15] Integration of acoustic and respiratory measures is required to identify sensitive and clinically relevant biomarkers for differential diagnosis, therapeutic monitoring and prediction of functional outcomes. This integrated approach represents a crucial improvement in understanding dysarthria as a phono-respiratory disorder rather than a collection of isolated impairments.

Therefore, this study aims to identify objective biomarkers by analysing respiratory and acoustic speech parameters in patients with ABI, with the goal of optimising clinical assessment and informing evidence-based on neurorehabilitation interventions.

METHODS AND ANALYSIS

Study design

An observational, cross-sectional study is proposed to analyse the relationship between acoustic, respiratory and perceptual speech parameters in adults with ABI. This design will allow the identification of phonorespiratory patterns and examination of associations between variables. This study will be conducted at the facilities of the Faculty of Nursing and Physiotherapy, University of Salamanca, Spain. The laboratories are equipped for comprehensive and controlled respiratory and acoustic speech assessments.

Study population

The target population comprises adults with a confirmed diagnosis of ABI. The sample will be obtained through non-probabilistic convenience sampling, including individuals who meet the eligibility criteria and provide informed consent.

The sample size has been established considering as the main contrast the comparison of the mean vowel space area (VSA) between groups with and without dysarthria secondary to ABI. According to the reference data described by Mou *et al.*,[6] speakers without dysarthria have a mean VSA of 0.096 kHz², while patients with varying degrees of severity show values between 0.079 and 0.075 kHz², representing an approximate reduction of 18–22% in the dysarthric population. Based on this relative difference and considering the expected clinical-functional heterogeneity in the sample, a moderate effect size ($d = 0.60$) was assumed for the comparison between groups. With a bilateral significance level of $\alpha = 0.05$ and a statistical power of 80%, the calculation for two independent means indicated the need to include 44 participants per group. Sample size calculations were performed using G*Power Software (V.3.1) (sample size calculations software package provided by the G*Power Team, Germany, downloaded from http://www.gpower.hhu.de/en.html). To compensate for possible data losses or invalid records, an additional 10–15% was added, estimating a minimum total sample size of between 97 and 101 participants.

Participants will be recruited over a period of approximately 15 months through healthcare centres and organisations that care for people with ABI. The initial classification of subjects into the ‘with dysarthria’ and ‘without dysarthria’ groups will be based on medical records and reports prepared by neurologists or rehabilitation physicians. In addition, the Frenchay Dysarthria

Assessment-2 (FDA-2)[16] will be administered on an exploratory basis to support the diagnostic characterisation and description of the severity of dysarthria.

Inclusion and exclusion criteria

Adults aged 18 years or older with a confirmed diagnosis of ABI (stroke, TBI or brain tumour), with or without dysarthria diagnosed according to medical criteria, will be eligible for inclusion. To ensure comprehension of instructions and accurate performance of experimental tasks, a minimum score of 24 on the Mini-Mental State Examination (MMSE)[17] will be required. Written informed consent must be provided by all participants prior to inclusion. Exclusion criteria include a history of chronic respiratory diseases (such as chronic obstructive pulmonary disease or asthma), hearing disorders, additional neurological or neurodegenerative conditions, pre-existing dysphonia or vocal disorders prior to the ABI episode and persistent pharyngeal symptoms such as secretions, mucus or foreign body sensation.

Outcome measures

The outcomes included in the study are organised into five functional domains: sociodemographic and clinical characteristics, cognitive function, swallowing function, respiratory function and acoustic characteristics of speech. In accordance with methodological recommendations for observational studies, each variable is prespecified according to its analytical role as an exposure, outcome, confounder or potential effect modifier. Detailed operational definitions, instruments and supporting references for all study outcomes are provided in online supplemental file table S1).

This analytical framework will permit a rigorous assessment of interactions across respiratory, phonatory and articulatory subsystems, and the investigation of associations between phonorespiratory profiles and dysarthria presence and severity.

In comparative analyses between groups, the main exposure will be the state of dysarthria, categorised as presence/absence of dysarthria and, complementarily, the severity of dysarthria determined using the FDA-2.[16] The main outcomes will include acoustic and articulatory parameters of speech, namely median fundamental frequency (F_0), jitter, shimmer, noise to harmonics ratio (NHR), harmonics to noise ratio (HNR), VSA and formant centralisation ratio (FCR), together with aerodynamic voice measures, including maximum phonation time (MPT), maximum emission time (MET) and the s/z ratio.[18–26] In addition, respiratory function will be assessed using standard spirometry parameters, including vital capacity (VC), FVC, MVV, FEV_1 and the FEV_1/FVC ratio.[27]

For association analyses, respiratory function parameters will be considered as primary exposures, while the outcomes will be speech acoustic and articulatory parameters (especially VSA and FCR) and the clinical severity of dysarthria (FDA-2 global score). In specific models, acoustic and respiratory parameters will be included as predictors of dysarthria severity, treated as an outcome variable.

Sociodemographic and clinical variables will be identified as potential confounding factors: age, sex, educational level, aetiology of brain damage (stroke, TBI, tumour), time elapsed since the injury event and relevant medical history. Additionally, global cognitive function, assessed using the MMSE, and the presence of dysphagia symptoms, quantified using the Eating Assessment Tool-10 (EAT-10),[28] will be considered as confounding factors. These variables will be incorporated into the multivariate models based on their clinical relevance and their bivariate association with the exposure and outcome variables.

Finally, several possible effect modifiers will be defined a priori. We will explore whether the aetiology of ABI and the severity of dysarthria (FDA-2 categories) modify the association between respiratory parameters and acoustic measures, as well as the relationship between these and the severity of dysarthria. To this end, interaction terms will be included in the regression models, and, in the event of statistical significance or clinical relevance, stratified analyses will be presented.

Enrolment and study completion

Participant enrolment will start in 2026 following approval of the clinical trial application. The recruitment and experimental phase is expected to last 15 months, with study procedures completed thereafter. Full study completion, including data analysis and reporting, is anticipated later in 2027.

Experimental procedures

The assessment protocol will be structured into three consecutive sessions, each lasting approximately 60 minutes, and will be conducted in a controlled environment to ensure standardisation of measurements.

In the first session, relevant sociodemographic and clinical data will be recorded. Overall cognitive function will then be assessed using the MMSE,[17] followed by administration of the EAT-10[28] to identify subjective dysphagia symptoms. Aerodynamic and acoustic voice assessments will subsequently be conducted in accordance with protocols validated in the literature[23 29 30] to obtain standardised measures of phonatory efficiency and articulatory stability.

The second session will focus on respiratory function assessment by spirometry, using standardised manoeuvres to obtain parameters of ventilatory capacity and respiratory flow. Recording quality will be ensured by following international recommendations for pulmonary function testing.

The third session will involve administration of the FDA-2, a 28-item instrument covering different speech subsystems.[16] The assessment provides a structured evaluation of oral reflexes, breathing, phonation, resonance, movement and strength of the lips, tongue and soft palate, as well as speech intelligibility.[16 31] Speech

intelligibility is assessed using standardised word and phrase cards, allowing estimation of the degree of speech comprehension. The FDA-2 yields ordinal scores for each item and an overall functional profile of speech motor performance. It is one of the most widely used clinical tools for assessing dysarthria in both clinical and research contexts, enabling perceptual characterisation of dysarthria patterns and severity. This assessment complements the instrumental measures obtained in previous sessions and allows for a multidimensional characterisation of each participant's phonorespiratory profile.[16 31]

Assessment procedures

Aerodynamic and acoustic assessment

The aerodynamic assessment will be conducted in accordance with standardised clinical protocols designed to evaluate respiratory–phonatory coordination and glottal efficiency.[23 29] Participants will be seated upright, with the torso aligned and feet flat on the floor, to promote stable breathing and controlled phonation.[30] Sustained emissions of the vowel /a/ at a comfortable pitch and intensity will be recorded, along with sustained productions of /s/ and /a/ to determine the s/z ratio, an indicator of respiratory control and glottal closure.[30] Each task will be performed three times consecutively, with rest intervals to prevent fatigue and the mean of the three measurements will be used as the representative value.[30]

Following the aerodynamic evaluation, acoustic analysis will be performed under the same controlled conditions. Voice recordings will be made in a soundproof room (noise level < 35 dB; reverberation time < 1 s) using a Tonor TC30 cardioid condenser microphone with a flat frequency response (60 Hz – 20 kHz) connected to a ZOOM H1n digital recorder. Signals will be captured in uncompressed waveform audio file format at 44.1 kHz and 16-bit depth, with manual gain control to avoid saturation. The microphone will be positioned 20 cm from the corner of the mouth at a 45° angle to the axis of emission, according to clinical recording standards.[23 29]

Participants will produce sustained phonations of the vowels /a, e, i, o, u/ at a comfortable pitch and intensity, as well as additional emissions of /a/ in high, low, strong and weak phonatory conditions to evaluate vocal stability across variations in pitch and intensity. Sustained phonation of /a/ will serve as a reference signal for extraction of jitter and shimmer, quantifying short-term instability of the vocal source.[23 30]

Acoustic processing will be performed using Praat: Doing Phonetics by Computer (V.6.4.48; Phonetic Sciences, University of Amsterdam)[32] to extract F_0, jitter, shimmer, HNR, NHR, VSA and FCR. VSA will be calculated from the first and second formant frequencies (F1–F2) of the corner vowels (/i/, /a/, /u/) representing the articulatory workspace, while FCR, derived from the mean formant values of (/i/, /a/, /u/), will quantify vowel centralisation, an acoustic correlate of articulatory precision and vocal tract stability.[25] All recordings will be visually inspected spectrographically to ensure a high signal-to-noise ratio and absence of artefacts.

VSA will be obtained by calculating the area of the triangle formed by the F1–F2 coordinates of the vowels /i/, /a/ and /u/ using the following equation[26]: VSA = $1/2 \cdot |F1i \cdot (F2a–F2u) + F1a \cdot (F2u–F2i) + F1u\ (F2i–F2a)|$.

The FCR will be calculated using the following equation, based on the relationship between formants that change with vowel centralisation[25]: FCR = (F2u + F2a + F1i + F1u)/(F2i + F1a).

Respiratory assessment

Respiratory function will be assessed using a Datospir Touch Spirometer, previously calibrated with a 3 L syringe in accordance with the calibration and reproducibility standards of the European Respiratory Society (ERS) and the American Thoracic Society (ATS).[33] Measurements will follow the Datospir Touch spirometer protocol to ensure standardised manoeuvres and reproducible records.

Participants will be seated upright, wearing a nose clip. Single-use antibacterial filters and mouthpieces will be employed to ensure hygiene. Standardised verbal instructions and visual feedback will be provided throughout each manoeuvre to minimise intra-individual variability and enhance performance consistency.[33] A rest interval of at least 60 seconds will be provided between manoeuvres to minimise fatigue, and participants will be offered a cup of water for hydration, in accordance with ERS/ATS reproducibility recommendations.[33]

Three respiratory manoeuvres will be performed: VC, FVC and MVV. In the VC manoeuvre, participants will take several calm breaths, followed by a maximum inhalation and a complete, effortless exhalation, allowing estimation of the maximal volume of air that can be exhaled following a maximal inspiration. The FVC manoeuvre will record the volume and flow rate of a forced exhalation after full inspiration, generating flow–volume loops from which FVC, FEV_1 and the FEV_1/FVC ratio will be extracted as indicators of ventilatory efficiency and airway patency.[28 33] The MVV test will assess respiratory muscle endurance and ventilatory reserve through rapid, deep breathing for 15 s.[27 33]

Each manoeuvre will be repeated three times, with the best acceptable and reproducible curve retained for analysis according to ERS/ATS repeatability criteria.[33] Quality control will include automated verification by the W20s-Datospir software, which ensures compliance with start, end, and plateau criteria, as well as visual inspection of the flow–volume curves to confirm technical acceptability and absence of artefacts.[27 33]

The W20s-Datospir software will process the spirometric data to extract primary variables (flow (L/s), volume (L) and time (s)) and calculate secondary indices, including mean inspiratory and expiratory flow and peak expiratory flow.

Approaches to mitigate potential biases

Several potential biases inherent to the observational design and characteristics of the ABI population have been identified.

Selection bias may arise, as patients with better functionality, milder dysarthria or higher cognitive status may be more likely to participate, potentially compromising sample representativeness. Recruitment from multiple centres could also lead to imbalances in ABI etiologies (stroke, TBI or tumour), affecting comparability between subgroups. To mitigate these biases, consecutive recruitment will be implemented across collaborating centres, broad inclusion criteria will be applied and reasons for non-participation will be documented whenever possible. Baseline characteristics (age, sex) will be compared between participants and non-participants, and efforts will be made to balance groups according to key variables.

Measurement bias is another consideration. Acoustic and respiratory parameters are sensitive to variations in assessor technique, participant task performance, fatigue and environmental conditions. These sources of variability can affect reliability, particularly for variables such as VSA, F_0 or respiratory flows, which require instrumental and behavioural consistency. To minimise this bias, all acoustic, aerodynamic and respiratory assessments will be conducted following standardised, previously piloted protocols, using equipment calibrated daily in controlled environmental conditions. Assessments will be performed by trained and experienced evaluators, with repeated measurements for critical variables and rest periods included to avoid fatigue effects.

Confounding bias may result from factors that simultaneously influence speech production, respiratory function and dysarthria severity. Relevant confounders include age, sex, aetiology of brain damage, time since injury, education level, cognitive function (MMSE), presence of dysphagia (EAT-10) and respiratory or neuromuscular comorbidities. Failure to account for these variables could distort associations between acoustic, respiratory and clinical parameters. To control confounding, these sociodemographic and clinical variables will be systematically collected and included in multivariate statistical models. Sensitivity analyses will also be performed, excluding participants with significant respiratory comorbidities to assess the robustness of the observed associations.

The combination of these strategies is expected to substantially reduce the influence of potential biases, enhancing the internal validity of the study and supporting more accurate interpretation of associations between respiratory, acoustic and clinical measures.

Statistical analysis

Statistical analyses will be conducted using IBM SPSS Statistics (V.28 or higher) and, when required for advanced models or graphical representation, R (V.4.0 or higher) or Python. A two-sided significance level of $\alpha = 0.05$ will be applied.

Data integrity will be checked by verifying ranges, impossible values and duplicates. Reasons for invalid records will be documented (eg, unacceptable spirometric manoeuvres, recordings with insufficient signal-to-noise ratio). In accordance with the protocol, all acoustic, aerodynamic and respiratory measurements will be obtained in three consecutive attempts. Acceptability and reproducibility criteria will be applied for each variable; when all three attempts meet these criteria, the best technically acceptable record will be selected following international recommendations. The number of valid attempts will be recorded for quality control purposes.

Continuous variables will be summarised using mean and SD or median and IQR, depending on their distribution. Categorical variables will be described using frequencies and percentages. Normality will be assessed using the Shapiro-Wilk test and graphical inspection. Outliers will be identified using graphical methods and robust criteria (eg, 1.5×IQR), and their treatment will be justified (eg, error verification, analysis with and without outliers).

For comparisons between participants with and without dysarthria, the main exposure will be the presence or absence of dysarthria, with severity (FDA-2) considered complementarily. Acoustic parameters (F_0, jitter, shimmer, HNR/NHR, VSA, FCR), aerodynamic parameters (MPT, MET, s/z ratio) and respiratory parameters (VC, FVC, FEV_1, FEV_1/FVC, MVV) will be compared using Student's t-test or Mann-Whitney U test, as appropriate. Categorical variables will be compared using χ^2 or Fisher's exact test. Effect sizes (Cohen's d or r, and/or mean differences with 95% CI) will be reported to facilitate clinical interpretation.

Associations between respiratory function and acoustic–articulatory measures (integrated biomarkers) will be examined using bivariate correlations between respiratory parameters (eg, FVC, FEV_1, MVV, FEV_1/FVC ratio) and acoustic–articulatory parameters (especially VSA, FCR, jitter, shimmer, HNR) using Pearson or Spearman correlation coefficients, depending on distribution. Associations between aerodynamic measures (MPT, MET, s/z ratio) and acoustic parameters will also be explored as an approximation of phono-respiratory coordination.

Multivariate models will be constructed to predict the presence of dysarthria using binary logistic regression with acoustic and respiratory biomarkers as predictors. To model dysarthria severity (FDA-2), linear regression will be applied if the total score is approximately continuous; otherwise, ordinal logistic regression will be used. All models will be adjusted for predefined confounders: age, sex, educational level, ABI aetiology (stroke/TBI/tumour), time since injury, and, where appropriate, MMSE and EAT-10 scores. Covariate selection will follow a mixed criterion of clinical relevance and bivariate association with exposure and outcome. Model assumptions (linearity in the logit for continuous predictors in logistic regression, normality and homoscedasticity of residuals in linear models) and collinearity (variance inflation factor

and tolerance) will be evaluated. ORs, standardised betas, 95% CIs and model fit metrics [eg, adjusted R^2, area under the curve, calibration] will be reported.

Potential effect modification by aetiology or dysarthria severity (FDA-2 categories) will be assessed using interaction terms (eg, FVC×aetiology; MVV×severity). Where statistically or clinically relevant interactions are detected, stratified analyses will be performed.

The proportion and pattern of missing data per variable will be quantified (completely random vs non-random). If missing data are minimal, complete case analysis will be applied; if substantial, multiple imputation will be considered according to a plausible mechanism, and results will be compared with complete case analysis. Sensitivity analyses will also be conducted excluding participants with significant respiratory comorbidities not initially detected or with technically suboptimal records, to assess the robustness of the findings.

Data management

The principal investigator or sub-investigator will provide participants with a detailed written explanation of the study and obtain their informed consent prior to participation. A case report form (CRF) has been specifically designed for data collection. All data collected will be analysed on a secure, offline computer to ensure the confidentiality of participants' personal information. Additionally, the data will be stored on password-protected digital versatile discs (DVD-Rs). These materials will be kept in a locked cabinet within the *NeuroUsal* research team facilities at the Department of Nursing and Physiotherapy, University of Salamanca, Spain, under the supervision of the team members, for a period of 5 years following study completion. After this retention period, all DVD-Rs will be securely destroyed and consent forms shredded.

Data monitoring

On-site monitoring will be conducted by a designated individual appointed by the principal investigator in accordance with the study protocol. The monitoring officer declares no conflicts of interest. All adverse events will be documented on the CRF, including detailed information regarding the nature, onset and resolution, severity, treatment, and outcome of each event, along with an assessment of causality and seriousness. Follow-up investigations will be performed when deemed necessary to ensure participant safety.

Patient and public involvement

None.

ETHICS AND DISSEMINATION

The study has been approved by the Drug Research Ethics Committee of the Salamanca Health Area, Spain (CEIm PI 2025 04 1897-TD), and will be conducted in accordance with the principles of the Declaration of Helsinki and current regulations on biomedical research. All participants will provide written informed consent prior to inclusion, and the confidentiality of their data will be ensured through coding and secure storage, accessible only to the research team.

Results will be disseminated via institutional repositories, national and international scientific conferences and peer-reviewed publications. The dissemination strategy will include a primary article reporting overall study results, as well as derivative publications addressing specific analyses of speech and voice deficits in patients with dysarthria secondary to ABI.

The anonymised data generated during the study will be made available following publication of the main results. The dataset will include acoustic, aerodynamic and respiratory variables, along with basic clinical information, excluding any data that could directly or indirectly identify participants. Data will be deposited in an institutional or thematic repository with controlled access and shared for scientific purposes on justified request, in accordance with data protection regulations and the conditions established by the ethics committee.

DISCUSSION

The findings are expected to provide practical guidance for clinical assessment and monitoring of dysarthria, supporting the work of speech and language therapists, physiotherapists and other rehabilitation professionals, and contributing to a better understanding of the interaction between respiratory function and speech production in this population.

Patients are expected to present an altered acoustic profile, including reduced VSA, elevated FCR, increased jitter and shimmer and decreased HNR, alongside a reduced respiratory pattern (VC, FVC, FEV_1, MVV, MPT and MET). The integration of these dimensions may reveal consistent associations defining differentiated phonorespiratory profiles. These measures are also anticipated to act as objective biomarkers of perceived dysarthria severity, as reflected in FDA-2 scores.

Dysarthria secondary to ABI represents a clinical and scientific challenge of considerable complexity due to its multisystemic nature and variability of manifestations. Simultaneous involvement of neuromotor circuits coordinating breathing, phonation and articulation generates heterogeneous phonorespiratory profiles that cannot be adequately explained by one-dimensional approaches. Despite this understanding, much of the literature has examined acoustic and respiratory components separately, limiting insights into the integrated biomechanics of altered speech. This highlights the need for multimodal methodological approaches capable of identifying functional patterns that more accurately reflect the complexity of motor speech disorders.

Integration of acoustic and respiratory parameters allows more comprehensive characterisation of individual phonorespiratory profiles. Previous studies have demonstrated that various acoustic parameters are sensitive

biomarkers capable of discriminating between healthy speakers and individuals with dysarthria. In particular, VSA and FCR have been shown to identify articulatory alterations following stroke, indicating reductions in vocal stability and precision associated with decreased speech intelligibility.[34 35] These results align with larger studies, such as Kim *et al*, who identified eight key acoustic parameters in a sample of 107 patients with dysarthria secondary to ABI, including the slope of the F2 formant, articulation rate, duration of voiceless intervals, spectral moments of fricatives, VSA and F_0 and intensity ranges, which classified dysarthria subtypes more accurately than traditional perceptual assessments.[15] Taken together, these findings support the value of acoustic analysis as a sensitive instrumental tool for defining clinically meaningful articulatory profiles.

Complementary evidence highlights the decisive role of respiratory function in speech production. Previous studies have documented significant reductions in respiratory force, FVC and FEV_1 in people post-stroke, forming a restrictive pattern that limits the air support available during phonation.[10 13 34] Although some research has explored the relationship between respiratory parameters and acoustic alterations in populations with Parkinson's disease,[36] the systematic integration of spirometry and acoustic analysis in the study of dysarthria following ABI has been rarely addressed. This methodological gap limits understanding of how respiratory deficits affect articulatory stability, prosodic modulation and phonatory efficiency—critical elements for interpreting the complexity of motor speech disorders.

The originality of this study lies in its integrated approach. Combining acoustic, respiratory and perceptual measures via the FDA-2 provides a robust framework for exploring correlations between respiratory efficiency, articulatory precision and perceptual characteristics of speech. This multimodal strategy has the potential to identify objective biomarkers of both severity and articulatory profile, supporting refinement of diagnostic processes and monitoring of clinical evolution. Furthermore, integration of these domains enables development of predictive models to guide individualised rehabilitation interventions, accounting for the marked heterogeneity characteristic of dysarthria in ABI. Such models could contribute to defining precise clinical subgroups, optimising therapeutic objectives and enhancing the effectiveness of evidence-based interventions.

Contributors AMM-N contributed to the conceptualisation of the study, methodological design, statistical analysis planning, manuscript drafting, scientific supervision and overall project coordination, and was responsible for funding acquisition as principal investigator and group leader. AA contributed to the conceptualisation of the study, development of the methodology, manuscript drafting and coordination of project activities. BHM and RDG contributed to the study design and manuscript drafting. All authors contributed to the critical revision of the manuscript, approved the final version for publication and agreed to be accountable for all aspects of the work. AMM-N is the guarantor of the study and the principal investigator, and accepts full responsibility for the integrity of the data and the accuracy of the analysis.

Funding This study will be funded by the Professional College of Physiotherapy of Castile and León, Spain, as part of the 12th call for applications for the 'Miguel Ángel Galán' innovation and research grants for 2026 (INV2026-64).

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting or dissemination plans of this research.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

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