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Longitudinal Changes in Nasal Airflow and Patient-Reported Outcomes Following Acoustic Nasal Therapy: A Pilot Study

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ABSTRACT

Background and Aims: Acoustic nasal therapy has emerged as a potential non-pharmacological adjunct for allergic rhinitis (AR) and chronic rhinosinusitis (CRS), but longitudinal evidence on its effects on nasal airflow and patient-reported outcomes remains limited. This early-phase study evaluated changes in objective nasal airflow and symptom burden following a 4-week acoustic nasal therapy intervention and a follow-up period after treatment withdrawal.

Methods: A prospective single-arm quasi-experimental longitudinal pilot study was conducted in 30 adults with clinician-diagnosed or symptom-consistent AR and/or CRS. Participants used a bilateral intranasal acoustic device for 10 min twice daily for 4 weeks. Outcomes were assessed at baseline, post-treatment, and follow-up after discontinuation. The primary longitudinal outcomes were peak nasal inspiratory flow (PNIF) and Sino-Nasal Outcome Test-22 (SNOT-22) scores. Secondary variables of subgroup, adherence, correlation, responder, and multivariable analyses were also explored.

Results: PNIF showed no significant change immediately post-intervention (-2.0 L/min, $p = 0.67$) but increased between post-treatment and follow-up ($+12.2$ L/min, $p = 0.036$); change from baseline to follow-up was not significant ($+10.1$ L/min, $p = 0.069$ after Holm adjustment). Symptom burden improved significantly during treatment (-15.43 points, $p < 0.0001$) but partially attenuated after discontinuation ($+11.97$ points, $p = 0.0003$), with no significant difference between baseline and follow-up (-3.47 points, $p = 0.242$). At post-treatment, 66.7% achieved the minimum clinically important difference, decreasing to 33.3% at follow-up. Objective airflow changes did not consistently align with symptom improvement.

Conclusion: This study provides early longitudinal evidence on acoustic nasal therapy in adults classified as having AR and/or CRS. Patient-reported symptoms improved during active treatment and attenuated after withdrawal, while changes in objective nasal airflow were less consistent. These hypothesis-generating findings support controlled studies to clarify efficacy, optimal dosing, and durability of response.

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Summary

- **What is known:** Acoustic nasal therapy is being explored as a non-pharmacological adjunct for allergic rhinitis and chronic rhinosinusitis, but longitudinal evidence on both objective airflow and symptom outcomes is limited.
- **What is new:** In this prospective longitudinal pilot study, SNOT-22 improved significantly during active treatment, while PNIF showed less consistent change and did not consistently parallel symptom improvement.
- **Clinical implications:** Acoustic nasal therapy may offer short-term symptomatic benefit, but objective airflow response and durability after treatment withdrawal remain uncertain, supporting the need for controlled trials.

1 | Introduction

Allergic rhinitis (AR) and chronic rhinosinusitis (CRS) are common upper airway conditions that impose a substantial symptom burden and impair daily functioning, often through persistent or fluctuating nasal obstruction, congestion, and reduced nasal airflow, which affect sleep, productivity, and overall well-being [1, 2]. Current management strategies primarily involve pharmacological therapies, including antihistamines and intranasal corticosteroids, and in more severe or refractory CRS cases, surgical intervention may be required to improve sinus drainage and symptom control [1, 3–5]. Despite these options, many patients continue to experience incomplete symptom relief or difficulty sustaining treatment benefits and side-effects [6], highlighting the need for adjunctive non-pharmacological approaches that may provide additional or complementary therapeutic effects.

In contemporary rhinology, there is a recognition that treatment response should be evaluated using both objective measures of nasal function and patient-reported outcomes, as these capture distinct yet clinically meaningful dimensions of disease burden and therapeutic benefit [7–9]. This has prompted interest in interventions that may improve nasal function while also addressing patients' subjective experience of symptoms.

Acoustic-based nasal interventions have emerged as a potential adjunct therapy for AR and CRS. Related interventions have been described in the literature using terms such as vibroacoustic nasal therapy, acoustic resonance therapy (ART), and acoustic vibration therapy [10–12]. For consistency, the intervention evaluated in the present study is referred to here as “acoustic nasal therapy.” Previous work investigating acoustic nasal therapy suggests potential symptomatic benefit and feasibility in selected populations [11–16]. However, the current evidence base is limited and heterogeneous, with some studies relying on cross-sectional comparisons or short-term assessments that do not adequately characterize the persistence or stability of treatment effects beyond the period of active use. This limitation is relevant for chronic conditions such as AR and CRS, where symptom severity varies over time and clinically meaningful benefit depends not only on

immediate improvement but also on the durability and consistency of response [17, 18].

Longitudinal studies that evaluate both objective nasal airflow and patient-reported outcomes are needed to better understand the temporal pattern of response to acoustic nasal therapy and its potential role as an adjunct strategy in AR and CRS management. This early-phase, hypothesis-generating study therefore evaluated whether acoustic nasal therapy was associated with changes in nasal airflow and patient-reported outcomes over time in adults classified as having AR and/or CRS, and whether these changes persisted after treatment withdrawal.

2 | Methods

2.1 | Study Design and Setting

A prospective single-arm quasi-experimental longitudinal pilot study was conducted to evaluate changes in nasal airflow and patient-reported outcomes following 4 weeks of acoustic nasal therapy. Participants were recruited in Aotearoa New Zealand between August and December 2025, and attended study visits at Auckland University of Technology clinical research facilities. Outcomes were assessed at baseline (V1), immediately post-treatment following a 4-week intervention period (V2), and at follow-up after discontinuation of device use (V3).

Ethical approval for this study was granted by the New Zealand Health and Disability Ethics Committee (HDEC; reference 2025 EXP 22987). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and its subsequent amendments. Written informed consent was obtained from all participants prior to enrollment and before any study-related procedures were undertaken. Participants were provided with detailed verbal and written information about the study objectives, procedures, potential risks, and their right to withdraw at any time without consequence. The study was also registered with the Australian New Zealand Clinical Trials Registry (ANZCTR; ACTRN12624001309572p).

2.2 | Sample Size

Practical considerations related to feasibility, device availability, and study duration supported recruitment of approximately 30 participants. Sample size justification was informed by prior nasal intervention studies reporting large within-participant effects on symptom-related outcomes (Cohen's $d \approx 1.0$ – 1.1) [16, 19]. An illustrative paired calculation using Cohen's $d = 1.04$ indicated that approximately 23 participants would provide 90% power to detect a pre-post difference at $\alpha = 0.05$. Recruitment of 30 participants allowed for potential attrition and additional variability. This calculation applied to the main within-participant comparison. Additional analyses for subgroup, interaction, correlation, or multivariable analyses were intended to be exploratory.

2.3 | Participants

Adults aged 18–80 years were eligible if they reported a prior clinician diagnosis of AR or CRS, or symptoms consistent with

AR or CRS on structured screening based on established clinical guidance [1, 4]. For participants who were uncertain of a prior diagnosis, a study classification of AR, CRS, or AR/CRS was assigned from the reported symptom pattern. Key exclusion criteria included fixed structural causes of nasal obstruction, active nasal pathology that could interfere with device use, recent severe epistaxis, pregnancy, known hypersensitivity to device materials, or medical conditions preventing safe participation (Supporting Information File S1). Eligibility was confirmed by an otolaryngologist or a trained study personnel member prior to enrollment.

2.4 | Study Procedures

Participants attended three in-person visits. At V1, written informed consent was obtained and demographic and clinical information were collected (Supporting Information File S1). An anterior nasal examination using Killian's or Thudicum's nasal specula was performed to identify fixed structural obstruction or active pathology that would preclude device use; this examination was used for eligibility rather than formal confirmation of AR or CRS. Baseline nasal airflow and symptom burden were then assessed. Participants used the Goodair Nosebuds (Goodair), a bilateral intranasal device delivering low-frequency acoustic stimulation synchronized with nasal inhalation (Figure 1), for approximately 10 min twice daily for 4 weeks. The intervention provides mechanical stimulation of the nasal passages during breathing and may facilitate retention of endogenously produced nasal nitric oxide (NO) within the upper airway [19].

At V2, conducted approximately 28 days after V1, nasal airflow and symptom measures were repeated, and participants completed a structured feedback questionnaire assessing device-use



FIGURE 1 | Placement of the acoustic nasal device during therapy. Demonstration of the Goodair Nosebuds acoustic nasal device positioned within the anterior nares during use. The device is inserted into both nostrils and delivers acoustic vibrations of 130 Hz during treatment sessions.

frequency and duration, tolerability, perceived effects, inter-current illness, and medication changes. Device-use frequency and duration were recalled at V2 using categorical response options. Treatment events were not prospectively timed or electronically logged. Participants were instructed to discontinue device use following V2 (Supporting Information File S1). V3 was conducted approximately 28 days after V2, when outcome measures and feedback were repeated.

2.5 | Outcome Measures

Peak nasal inspiratory flow (PNIF) was measured at each visit using a standard PNIF meter (VMedical). Participants performed three maximal inspiratory maneuvers through the nose, and the mean value was recorded [20]. Participants were instructed and supervised to ensure correct technique and maximal effort. Measurements were obtained during scheduled study visits under routine clinical conditions; timing was not standardized for time of day or nasal cycle phase. Symptom burden was assessed using the Sino-Nasal Outcome Test-22 (SNOT-22). Although originally validated in CRS, SNOT-22 has also been shown to be reliable and responsive for assessing quality of life and symptom control in persistent AR [7, 21]. Higher scores indicate greater symptom burden.

2.6 | Statistical Analysis

All analyzes were conducted in R (version 2025.09.2 + 418). Baseline characteristics were summarized descriptively using counts and percentages. The main longitudinal analyzes examined change in PNIF and total SNOT-22 across visits using linear mixed-effects models with visit as a fixed effect and participant as a random intercept [22]. Pairwise visit comparisons were obtained using estimated marginal means and adjusted using Holm's sequential Bonferroni correction, which controls the family-wise error rate while offering greater power than conventional Bonferroni correction [23]. All 30 participants had PNIF and SNOT-22 measurements at V1, V2, and V3; no imputation was required for these outcomes.

All additional analyzes were exploratory and hypothesis-generating. These included visit $\times$ diagnostic-classification interactions, SNOT-22 domain analyzes, associations with baseline medication categories, demographic and environmental multivariable models, adherence-response correlations, concordance between PNIF and SNOT-22, and perceived improvement. SNOT-22 responder analysis used a reduction of at least 9 points from baseline. This threshold is consistent with the established CRS literature and falls within the 6–11 point range reported in persistent AR [21, 24]. Exploratory analyzes were not used for confirmatory inference; nominal p values are reported unless an adjustment is stated. Statistical significance was defined as two-sided $p < 0.05$.

3 | Results

3.1 | Baseline Participant Characteristics

A total of 31 participants were recruited but one participant did not complete the study, resulting in a final analytical sample

size of 30 participants. All 30 participants completed V1, V2, and V3 and had complete PNIF and SNOT-22 measurements available at all three visits. No imputation was required for the main outcomes. Ethnicity was reported using the New Zealand standard classification and analyzed using a total-response approach, whereby participants reporting multiple ethnicities were counted in each relevant category. Seventeen participants were classified as AR, seven as CRS, and six as AR/CRS (Table 1).

3.2 | Participant-Reported Device Usability and Experience

Participant-reported usability, device use, and perceived experience were assessed at V2 and V3 (Supporting Information File S1). During active treatment, 70% reported daily use and 27% reported use on most days. Seventy percent reported using the device for around 10 min twice daily, while 23% reported longer use and the remainder shorter or variable use. Sixty percent described the device as somewhat or very comfortable, and 57% reported no side effects. At V2, 37% reported slight improvement, 37% moderate improvement, and 17% significant improvement. At V3, device use had stopped per protocol and several participants reported worsening symptoms. Medication use was not uniformly unchanged during follow-up: some participants reported reducing antihistamine or nasal spray use, whereas others reported additional antihistamine or intranasal spray use. These interval changes were self-reported rather than prospectively recorded as time-varying medication exposure.

3.3 | Nasal Airflow (PNIF) Across Study Visits

Mean PNIF values were similar at V1 and immediately after V2, with a higher mean observed at V3. Substantial inter-individual variability in PNIF measurements was evident at all time points. Examination of within-participant change scores showed considerable heterogeneity across study intervals, with both improvements and declines in PNIF observed (Figure 2, Supporting Information File S2). Changes between V2 and V1 were generally centered on zero. In contrast, larger positive changes were more frequently observed between V3–V2 and V3–V1.

Pairwise comparisons from the mixed-effects model showed no significant change in PNIF immediately following the intervention (V2–V1: -2.0 L/min, $p = 0.67$). PNIF increased between V2 and V3 (V3–V2: $+12.2$ L/min, $p = 0.036$), while the change from V1 to V3 did not reach statistical significance after multiplicity correction (V3–V1: $+10.1$ L/min, $p = 0.069$). An exploratory multivariable model examining 18 demographic and environmental/lifestyle predictors of V1–V3 PNIF change was not statistically significant ($F(18,11) = 0.95$, $p = 0.55$). Given the number of predictors relative to the sample size and the 11 residual degrees of freedom, this model was considered hypothesis-generating rather than evidence that these factors have no association with PNIF change. Exploratory models using baseline medication categories also did not identify statistically significant associations with PNIF change across the study intervals (V2–V1: $F(5,24) = 0.79$, $p = 0.564$; V3–V2: $F(5,24) = 1.39$, $p = 0.264$; V3–V1: $F(5,24) = 0.27$, $p = 0.924$).

TABLE 1 | Participant characteristics.

Group	Characteristic	N = 30 ¹
Demographic characteristics	Study classification	
	AR	17 (57%)
Demographic characteristics	AR/CRS	6 (20%)
	CRS	7 (23%)
Demographic characteristics	Age group	
	18–29	4 (13%)
Demographic characteristics	30–44	14 (47%)
	45–59	8 (27%)
Demographic characteristics	60–80	4 (13%)
	Gender	
Demographic characteristics	Female	18 (60%)
	Male	10 (33%)
Demographic characteristics	Non-binary	1 (3.3%)
	Prefer not to say	1 (3.3%)
Demographic characteristics	Ethnicity	
	Asian	11 (36.7%)
Demographic characteristics	Māori	2 (6.7%)
	New Zealand European	16 (53%)
Demographic characteristics	Other	2 (6.7%)
Demographic characteristics	Household size	
	1	4 (13%)
Demographic characteristics	2	9 (30%)
	3–4	11 (37%)
Demographic characteristics	5+	6 (20%)
	Environmental/lifestyle factors	
Demographic characteristics	Ex-smoker	8 (27%)
	Exposed to smoking	2 (6.7%)
Demographic characteristics	Exposed to vaping	3 (10%)
	Vape myself	2 (6.7%)
Demographic characteristics	Other exposure	1 (3.3%)
	Pets	19 (63%)
Demographic characteristics	Dusty environment	7 (23%)
	Medication-related factors	
Demographic characteristics	Antibiotics	2 (6.7%)
	Antihistamines	15 (50%)
Demographic characteristics	Intranasal corticosteroids	4 (13%)
	Decongestants	5 (17%)
Demographic characteristics	Probiotic use	4 (13%)
	No prednisone use	30 (100%)
Demographic characteristics	No listed medication	13 (43%)

¹n (%).

Exploratory interaction models did not identify statistically significant differences in PNIF trajectories between AR, AR/CRS, and CRS study-classification groups at V2 ($p = 0.86$ – 0.93) or V3 ($p = 0.40$ – 0.76). Baseline estimated PNIF values were similar across groups (AR: 125 L/min [95% CI 104–145];

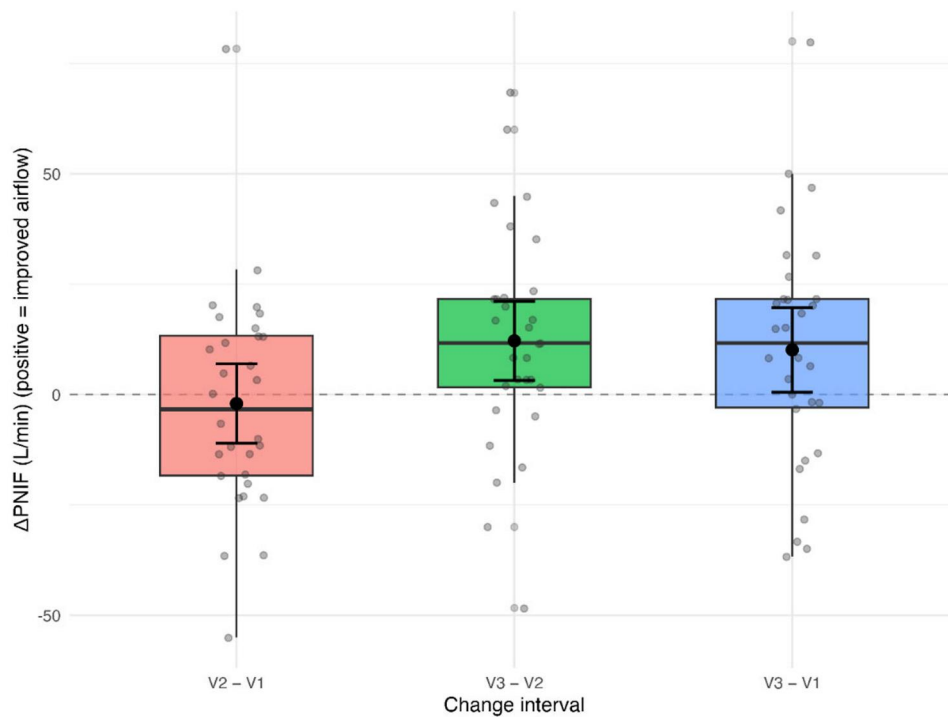


FIGURE 2 | Individual PNIF changes across study intervals.

AR/CRS: 139 L/min [105–174]; CRS: 136 L/min [104–168]), with no statistically significant pairwise differences at V1 (all $p \geq 0.05$). These subgroup analyses were not powered for between-group inference as no statistical differences were detected.

3.4 | Patient-Reported Symptom Severity Across Study Visits (SNOT-22)

Mean SNOT-22 scores decreased substantially from V1 to V2 and then increased toward baseline levels at V3 (Figure 3). Substantial inter-individual variability was observed at all visits.

Pairwise comparisons from the linear mixed model showed a significant reduction in SNOT-22 scores from baseline to post-treatment (V2–V1: -15.43 points, $p < 0.0001$), exceeding the 9-point responder threshold. No significant difference was observed between baseline and follow-up (V3–V1: -3.47 points, $p = 0.242$), while scores increased from post-treatment to follow-up (V3–V2: $+11.97$ points, $p = 0.0003$). Exploratory visit $\times$ study-classification models did not identify statistically significant interactions. Within-group contrasts showed V1–V2 improvement in the AR group ($p < 0.0001$) and CRS group ($p = 0.0107$), but not the AR/CRS group ($p = 1.00$). From V2 to V3, worsening was statistically significant in the AR group ($p = 0.0002$) but not in the AR/CRS or CRS groups ($p > 0.05$). V1–V3 comparisons were not statistically significant in any group ($p > 0.05$). These subgroup contrasts were exploratory and were not powered to establish differences between diagnostic categories.

Using a ≥ 9 -point reduction in SNOT-22 as the responder threshold, 20 participants (66.7%) met the criterion

immediately post-treatment and 10 (33.3%) met it at V3. When stratified by responder status, PNIF change distributions overlapped substantially (Supporting Information File S2). Non-responders sometimes showed equal or greater positive PNIF change than responders, particularly after treatment withdrawal. In an exploratory logistic regression using baseline medication categories, no medication category showed a statistically significant association with responder status ($p > 0.05$); this analysis was not powered for firm predictor inference.

3.5 | Domain-Specific Patterns in SNOT-22 Scores Across Study Visits

Mean scores across all five SNOT-22 domains (nasal, ear/facial, sleep, function, and emotion) decreased from V1 to V2, indicating broad-based symptom improvement during the intervention period (Figure 4). Statistically significant reductions were observed between V1 and V2 for all domains: nasal (mean change -4.73 points, $p = 0.0013$), ear/facial (-1.87 points, $p = 0.0004$), sleep (-2.33 points, $p = 0.0010$), function (-4.73 points, $p = 0.0013$), and emotion (-4.43 points, $p = 0.0007$).

At V3, domain scores were no longer significantly different from baseline for any domain (all $p > 0.05$) after device discontinuation. Comparisons between V3 and V2 demonstrated significant increases in symptom scores for the ear/facial ($p = 0.0223$), sleep ($p = 0.0143$), function ($p = 0.0013$), and emotion ($p = 0.0117$) domains, consistent with partial symptom recurrence after therapy cessation. The nasal domain showed a similar trend, although the V3–V2 comparison did not reach statistical significance ($p = 0.0816$).

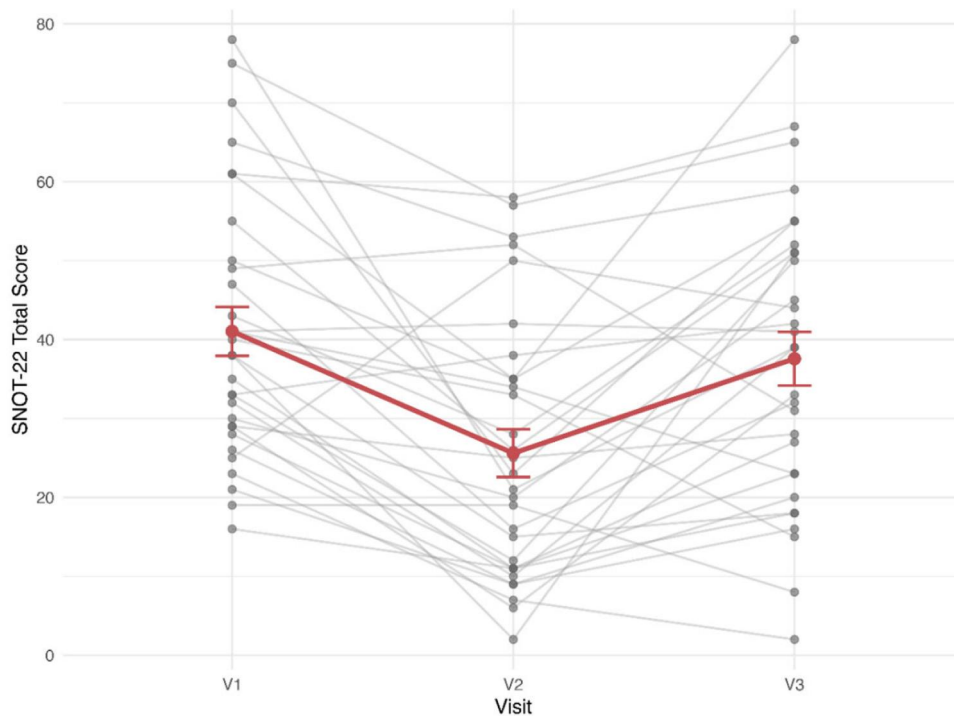


FIGURE 3 | SNOT-22 trajectories across visits.

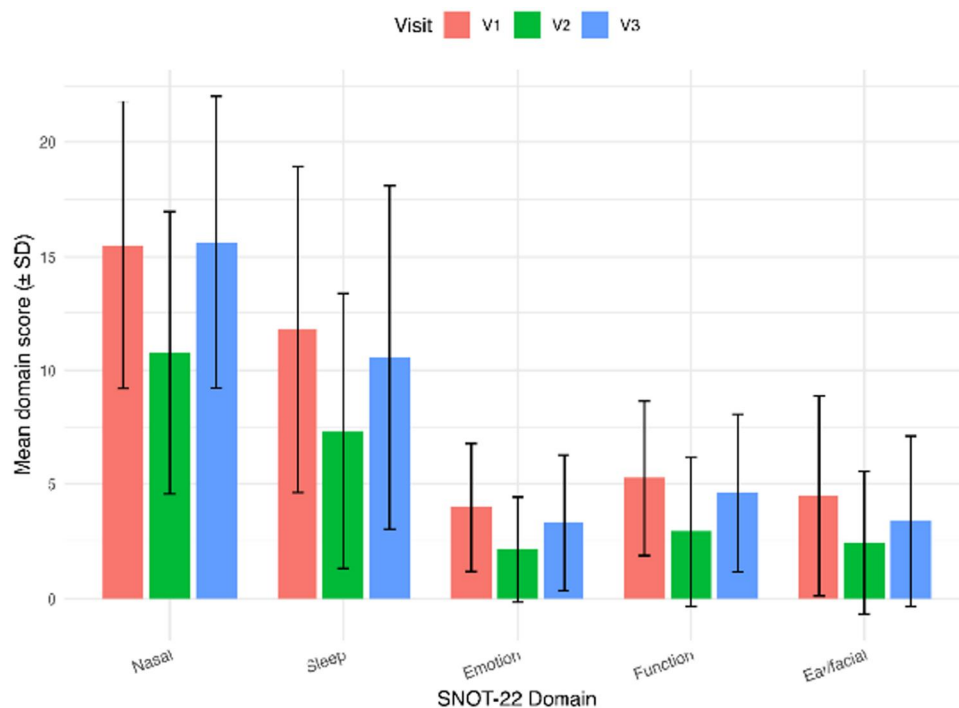


FIGURE 4 | SNOT-22 domain scores across study visits.

3.6 | Associations Between Participant-Reported Device Experience and Objective and Subjective Outcomes

3.6.1 | Adherence-Response Patterns

At V2, reported frequency of device use was not associated with PNIF change (Spearman $\rho = -0.03$, $p = 0.86$; Supporting Information File S2). Duration showed a modest inverse

association with PNIF change ($\rho = -0.41$, $p = 0.028$), with shorter reported duration associated with larger PNIF improvement. PNIF responses were variable across categories. One participant reported variable duration and was excluded from the ordinal duration correlation. Because device use was recalled in broad categories rather than measured prospectively, these analyses represent approximate exposure-response patterns rather than a measured dose-response relationship.

3.6.2 | Adherence and SNOT-22 Change

Reported device-use frequency and duration were not associated with SNOT-22 change from V1 to V2. Spearman correlations were weak and non-significant for frequency ($\rho = -0.09$, $p = 0.64$) and duration ($\rho = -0.12$, $p = 0.52$) (Figure 5). Symptom changes were widely distributed across the broad use categories, with no consistent exposure-response pattern.

3.6.3 | Alignment Between Perceived Improvement and SNOT-22 Change

At V2, greater perceived improvement on the feedback questionnaire was associated with a larger reduction in SNOT-22 score (Spearman $\rho = -0.55$, $p = 0.001$). This association reflects agreement between two patient-reported measures and was considered exploratory.

3.6.4 | Concordance Between Objective and Subjective Outcomes

While a subset of participants demonstrated concordant improvement in both nasal airflow and symptom burden, considerable inter-individual variability was observed (Supporting Information File S2). Mixed response patterns were frequent, with some participants showing improvements in objective

airflow without corresponding symptom improvement, and others reporting symptom improvement without measurable gains in airflow.

4 | Discussion

This study provides longitudinal evidence on acoustic nasal therapy in adults with AR and CRS, showing that short-term improvements in patient-reported symptoms were not consistently mirrored by changes in objective nasal airflow. The acoustic nasal device appeared feasible and acceptable, with most participants reporting daily or near-daily use during the active phase and infrequent side effects. SNOT-22 scores improved significantly during treatment, with 66.7% of participants achieving the minimal clinically important difference (MCID) threshold in existing literature [7, 24, 25], but this benefit attenuated after treatment withdrawal, and the difference between follow-up and baseline was not statistically significant.

The symptom improvements observed in this study are broadly consistent with previous studies of acoustic or oscillatory nasal interventions, which have reported short-term improvement in congestion-related outcomes. Acoustic vibration has been associated with improvement in subjective congestion after

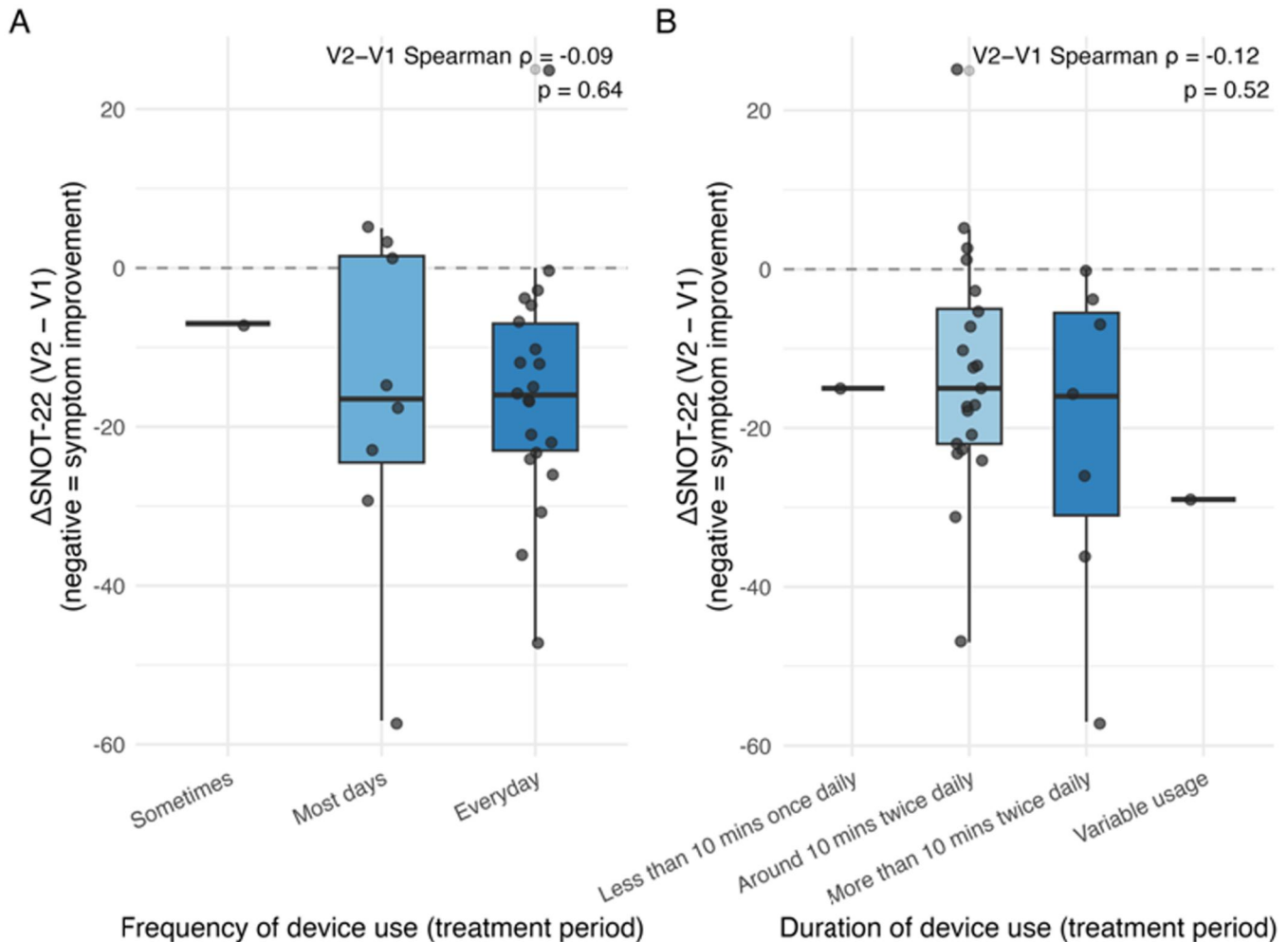


FIGURE 5 | Reported device use and change in SNOT-22 scores.

brief self-applied sessions [12], while another study reported improvement in both PNIF and SNOT-22 [16]. A randomized double-blind sham-controlled trial of acoustic resonance therapy in rhinitis also found greater improvement than sham in nasal congestion sub-scores and total nasal symptom score [14]. Differences between studies may partly reflect design and intervention differences; trials with sham or control groups can estimate treatment effects more directly than the single-arm design used here.

Objective airflow changes did not parallel symptom change. PNIF can be influenced by time of day, nasal cycle phase, short-term physiological variability, technique, and inspiratory effort [26–28]. Most measurements in this study were obtained within a 4- to 5-h period of the day, but time of day and nasal cycle phase were not formally standardized. Previous acoustic nasal therapy studies have generally used fixed treatment schedules rather than comparing alternative doses, so the optimal frequency and duration remain uncertain [14, 16].

The pattern in our study, with clear SNOT-22 improvement during active use followed by attenuation after cessation, is consistent with a therapy that may primarily modulate symptoms during the period of use rather than produce durable effects after withdrawal. The less consistent PNIF response, including the absence of an immediate post-intervention improvement, differs from some prior reports. For example, a study reported a significant improvement in PNIF at a 2-week follow-up, with an increase of 25.0 L/min (31% from baseline, $p < 0.001$) [16]. This discrepancy may reflect differences in device mechanism, such as acoustic effects alone compared with combined oscillatory expiratory pressure, differences in timing of outcome assessment, such as acute post-session effects compared with visit-based measurements, and population heterogeneity, including mixed AR and CRS phenotypes and the use of concomitant therapies. It also highlights the importance of including both PROMs and objective measures when evaluating nasal interventions, as recommended in rhinology outcome frameworks [1].

This study also observed weak alignment between objective airflow change and patient-reported improvement at the individual level, despite concordance between perceived improvement and SNOT-22 change at V2. This was not unexpected. The correlation between objectively measured nasal airflow and the subjective sensation of obstruction is frequently poor [29–32]. A potential explanation might be that humans largely sense airflow via mucosal cooling, mediated by trigeminal cool thermoreceptors (TRPM8), rather than “airflow volume” [33]. Consequently, interventions that alter heat transfer dynamics or the distribution of airflow across the nasal mucosa may change perceived nasal patency without producing a commensurate change in PNIF. This physiology is demonstrated clinically by menthol: individuals often report improved nasal patency after menthol inhalation despite little or no objective decongestant effect [34]. Conversely, in obstructive sleep apnea, nasal CPAP can provoke nasal symptoms and perceived congestion that contribute to mask removal, even when the airway is being pneumatically splinted; heated humidification modifies these upper-airway side effects, underscoring how temperature/humidity and mucosal conditioning influence subjective

experience [35]. A review also noted that perceived patency is associated with dynamic temperature changes during breathing and can be influenced by the nasal cycle and side-to-side airflow asymmetry [33]. This provides a coherent interpretive frame for our concordance plots showing mixed response patterns, including objective improvement without symptom improvement and symptom improvement without objective improvement, as well as the lack of a stable relationship between MCID response and PNIF change. It also suggests that future studies of acoustic interventions may benefit from incorporating measures of nasal heat exchange or mucosal temperature change alongside classic airflow metrics, as proposed earlier [36].

Exploratory adherence analyses did not show a conventional dose-response pattern. Reported frequency was not associated with PNIF change, while reported duration showed a modest inverse association; neither frequency nor duration was associated with SNOT-22 change. These findings should be interpreted cautiously. Use was recalled at V2 using broad categories such as every day, most days, or sometimes and approximate duration categories rather than prospectively timed treatment events. The categories therefore represent approximate self-reported exposure rather than measured treatment dose and are vulnerable to recall error and misclassification [37, 38]. The inverse duration association could reflect chance, confounding, or PNIF variability rather than a true dose effect. Future studies could use objective adherence tracking and prospectively defined dose strata.

From a pragmatic clinical perspective, the observed short-term improvement in SNOT-22 across multiple domains suggests that some individuals may experience broad symptomatic benefit while actively using the device. This is clinically relevant because contemporary CRS/rhinitis care emphasizes patient-centered symptom and quality-of-life outcomes, for which validated PROMs such as SNOT-22 are widely used [1]. However, the loss of benefit after discontinuation suggests that sustained symptom control may require ongoing use and/or combination with standard therapies (e.g., intranasal corticosteroids in inflammatory disease phenotypes), rather than expecting durable disease modification from short courses of acoustic nasal therapy alone. The divergence between PROM improvement and objective airflow underscores the need to avoid over-interpreting PNIF as a surrogate for symptom response. Another explanation for this divergence is that these measures operate over different time frames. PNIF reflects a single clinic-based measurement at a specific time point, whereas SNOT-22 captures symptom burden over the preceding 2 weeks, so discrepancies may partly reflect immediate versus longer-term experience of benefit. The field therefore recommends integrating objective and subjective outcomes, recognizing that they capture different constructs such as maximal airflow, perceived nasal patency and functional impact [39].

Several limitations temper causal inference. The single-arm quasi-experimental design did not include a sham control, so expectancy effects and temporal changes unrelated to treatment cannot be excluded. The small sample also limits subgroup and predictor analyses. In particular, the 18-predictor PNIF model had only 11 residual degrees of freedom, so its estimates are unstable and the absence of statistically significant predictors should not be interpreted as evidence of no association. Nonetheless, the analysis

provided preliminary information on the direction and relative magnitude of associations across demographic and environmental factors, helping to identify variables that may warrant more focused evaluation in larger studies. Diagnostic classification was pragmatic, where some participants reported a prior clinician diagnosis, whereas others were classified from symptom patterns without required allergy testing, nasal endoscopy, or computed tomography. Misclassification is therefore possible, and non-allergic rhinitis could not be excluded among participants classified as AR from symptoms alone [4]. The anterior nasal examination at enrollment was used to exclude fixed obstruction or active pathology relevant to device use, not to confirm AR or CRS. Interval medication use was also not fully stable and was recorded retrospectively rather than as a time-varying exposure, so concurrent treatment remains a possible confounder. Subgroup, medication, adherence, correlation, and multivariable findings should therefore be regarded as exploratory.

The follow-up structure also complicates interpretation of PNIF; improvement occurred after protocol-mandated discontinuation rather than immediately post-intervention, raising the possibility of delayed effects, regression to the mean, seasonal and environmental fluctuation, or measurement variability related to PNIF technique and the nasal cycle. However, the longitudinal design also provides advantages, as repeated measurements over time allow within-participant trajectories to be examined and reduce the influence of baseline heterogeneity [40, 41]. In our cohort, PNIF values varied substantially at baseline, ranging from 83.3 to 257 L/min, and similar variability was observed at subsequent visits (V2: 71.7–268 L/min; V3: 80–257 L/min). This degree of inter-individual variability illustrates how between-participant comparisons alone can obscure treatment-related changes, whereas repeated within-participant measurements enable the detection of temporal changes despite heterogeneous baseline airflow values.

Given the exploratory nature of this study, future trials could prioritize randomized, double-blind sham-controlled designs, as demonstrated in ART studies, together with more frequent and time-standardized assessment windows, including acute post-use measurements. Future work may also benefit from objective adherence monitoring and the inclusion of mechanistically informative outcomes beyond PNIF, such as rhinomanometry or acoustic rhinometry, measures of nasal heat exchange or mucosal temperature, and potentially nasal NO, given evidence that oscillatory airflow (e.g., humming) can markedly increase nasal NO and sinus ventilation [42]. These steps could clarify whether the benefits of the acoustic device arise predominantly from altered airway mechanics, altered mucosal conditioning/thermosensory signaling, or a combination of both, and would better define the patient phenotypes most likely to benefit.

5 | Conclusion

In this single-arm longitudinal pilot study of adults classified as having AR and/or CRS, acoustic nasal therapy was associated with substantial short-term improvement in patient-reported symptom burden during active use, with two-thirds of participants achieving a clinically meaningful reduction in SNOT-22 scores. Symptom improvement attenuated after device

discontinuation, while objective nasal airflow changes were modest and did not consistently align with symptom response. Exploratory subgroup and predictor analyses were inconclusive.

These results suggest that acoustic nasal therapy is feasible and well tolerated and may provide short-term symptomatic benefit. However, the mixed diagnostic cohort, symptom-based classification in some participants, concurrent medication use, self-reported device exposure, and absence of a sham-controlled comparison limit causal inference. Controlled, sham-randomized studies with larger samples, objective adherence measures, and mechanistic endpoints are needed to clarify efficacy, durability, and which patients are most likely to benefit.

Author Contributions

Jude Oluwapelumi Alao: investigation, data curation, methodology, formal analysis, visualization, writing – original draft, writing – review and editing, funding acquisition. **Jim Bartley:** supervision, project administration, validation, methodology, investigation, resources. **Chris Pulluwa:** supervision, project administration. **David White:** resources, writing – review and editing. **Kevin Lee:** supervision. **Kelvin E. M. Lau:** conceptualization, supervision, project administration, methodology, validation, funding acquisition, writing – review and editing, investigation.

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Ethics Statement

Ethical approval for this study was granted by the New Zealand Health and Disability Ethics Committee (HDEC; reference 2025 EXP 22987). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and its subsequent amendments. Written informed consent was obtained from all participants prior to enrollment and before any study-related procedures were undertaken. Participants were provided with detailed verbal and written information about the study objectives, procedures, potential risks, and their right to withdraw at any time without consequence. The study was also registered with the Australian New Zealand Clinical Trials Registry (ANZCTR; ACTRN12624001309572p).

Conflicts of Interest

DW is the Chief Scientific Officer and a shareholder of My Better Breathing Ltd, trading as Goodair (the device manufacturer), and had no role in the study design, data collection, data analysis, interpretation of results, or manuscript preparation. All other authors declare no competing interests.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request due to privacy and ethical restrictions.

Transparency Statement

The lead author affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Supporting File 1: hsr273303-sup-0001-Supplementary_File_1.docx.

Supporting File 2: hsr273303-sup-0002-Supplementary_File_2.docx.