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Subjective Otologic and Vestibular Symptom Changes During 12 Months of Neuromuscular Orthotic Therapy in Patients with Temporomandibular Disorders: An Exploratory Longitudinal Study with Mandibular Kinesiography

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Abstract

Background/Objectives: Otologic and vestibular complaints are frequent in patients with temporomandibular disorders (TMDs), but evidence that TMD-directed treatment improves dizziness or aural fullness remains limited. This exploratory study evaluated 12-month changes in subjective otologic/vestibular symptoms and mandibular kinematics during neuromuscular orthotic therapy. **Methods:** Thirty-five patients with TMD underwent clinical assessment and K7 three-dimensional mandibular kinesiography at baseline (T0) and 12 months (T1); 20 asymptomatic subjects provided cross-sectional kinematic reference data. Audiologic and bedside otoneurological assessment screened for clinically evident primary auditory and vestibular disorders. Kinesiography was recorded on natural dentition at T0 and with the orthotic in situ at T1. Raw XYZ trajectories were analyzed with a reproducible post hoc signal-processing and segmentation pipeline. **Results:** Vertigo/dizziness improved or resolved in 10/10 patients (100%), aural fullness in 7/8 (87.5%), and tinnitus in 22/24 (91.7%). Across 35 paired recordings, vertical excursion decreased from 4.20 ± 3.16 to 3.14 ± 2.90 mm ($p = 0.084$), 3D path length from 16.84 ± 14.60 to 11.94 ± 11.74 mm ($p = 0.078$), and mean 3D speed from 5.25 ± 2.88 to 4.42 ± 3.42 mm/s ($p = 0.065$). A post hoc sensitivity analysis of 27 higher-confidence segmentations showed significant reductions in vertical excursion ($p = 0.049$), path length ($p = 0.049$), and mean speed ($p = 0.030$). **Conclusions**: Orthotic therapy was temporally associated with marked improvement in subjective otologic/vestibular symptoms and directional kinematic changes. The uncontrolled design, categorical symptom outcomes, non-age-matched controls, and different T0/T1 recording conditions preclude causal claims.

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1. Introduction

Temporomandibular disorders (TMDs) comprise a heterogeneous group of musculoskeletal conditions affecting the temporomandibular joints, masticatory muscles, and associated structures. Although pain, joint sounds, and altered mandibular function remain the principal clinical manifestations, patients with TMD frequently report symptoms outside the stomatognathic system, including tinnitus, otalgia, aural fullness, subjective hearing changes, dizziness, and vertigo [1–4]. A systematic review and meta-analysis by Porto De Toledo et al. reported high prevalences of ear fullness, tinnitus, and vertigo among adults with TMD, albeit with substantial between-study heterogeneity [2]. More recently, a 2026 systematic review and meta-analysis confirmed that dizziness is frequently reported in TMD populations while emphasizing the observational nature of the available evidence and the unresolved direction of causality [4].

The association between TMD and tinnitus has received the greatest attention. Somatosensory tinnitus describes tinnitus whose perceptual characteristics may be influenced by somatic input from the cervical or temporomandibular region; consensus diagnostic criteria have been proposed to improve its clinical identification [5,6]. Experimental and translational evidence supports interaction between somatosensory and auditory pathways, including modulation of dorsal cochlear nucleus activity by trigeminal and cervical inputs [7,8]. Clinical studies likewise show frequent coexistence of TMD and tinnitus, although coexistence does not establish a causal relationship in an individual patient [9].

Evidence is less mature for dizziness, subjective vertigo, and aural fullness. A 2024 systematic review of randomized controlled trials evaluating rehabilitation for otologic symptoms associated with TMD identified only five eligible trials and found evidence limited to secondary otalgia and tinnitus; no randomized trials evaluating ear fullness, dizziness, or vertigo were identified [10]. Observational and interventional studies have nevertheless reported improvement in otologic complaints following TMD-oriented management [11,12]. In somatosensory tinnitus, a randomized controlled trial demonstrated that multidisciplinary orofacial treatment can reduce tinnitus burden in selected patients [13]. These findings support further prospective investigation of vestibular-type complaints in well-characterized TMD cohorts, while maintaining a strict distinction between patient-reported symptom improvement and recovery of vestibular function.

Mandibular kinesiography provides a non-invasive method for describing three-dimensional jaw movement. Swallowing requires coordinated mandibular, lingual, suprahyoid, and masticatory activity and can therefore be used as a functional motor task, but it is not a direct test of auditory or vestibular physiology [14]. Movement characteristics can be described by geometric and velocity-based measures such as excursion, path length, mean and peak speed, spectral arc length (SPARC), and dimensionless jerk. Because smoothness measures are sensitive to filtering, sampling frequency, differentiation, movement duration, and event segmentation, their computational definitions must be explicit [15,16].

The present study was designed as a 12-month exploratory longitudinal investigation of an independent cohort of patients with TMD and associated otologic or vestibular complaints treated with a neuromuscular mandibular orthotic. The primary clinical objective was to describe changes in subjective vertigo/dizziness, tinnitus, and

aural fullness. A secondary objective was to characterize changes in mandibular kinematics, recorded during orthotic use, during a spontaneous swallowing-related motor task using reanalysis of the raw K7 trajectories. Healthy asymptomatic subjects were used only as a cross-sectional kinematic reference. We hypothesized that treatment would be accompanied by symptomatic improvement and by a directional reduction in excessive or complex mandibular movement.

2. Materials and Methods

2.1. Study Design and Participants

This longitudinal observational study included 35 patients with TMD and extra-stomatognathic symptoms and a separate group of 20 asymptomatic healthy subjects used as a cross-sectional kinematic reference. Patients were recruited consecutively between January 2022 and January 2025 from the Audiology and Odontostomatology Units of Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico and the Istituto Stomatologico Italiano, Milan, Italy. The clinical cohort included 19 women and 16 men, with a mean age of 47.8 ± 18.7 years (range, 15–84 years). The reference group included 13 women and 7 men, with a mean age of 24.7 ± 1.3 years (range, 23–27 years). Because the groups were not age-matched, between-group comparisons were considered descriptive and exploratory. No formal a priori sample-size calculation was performed; the sample consisted of all consecutive eligible patients fulfilling the criteria during the predefined recruitment period.

TMD evaluation was performed according to the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), Axis I [1]. Patients diagnosed with TMD and with at least one of the following symptoms, dizziness, vertigo, somatosensory tinnitus or aural fullness, were included. Patients with somatosensory tinnitus were classified according to published clinical criteria [5]. Exclusion criteria comprised tinnitus attributable to primary hearing loss, Ménière disease, middle-ear or intracranial pathology, clinically evident peripheral vestibular disease, previous whiplash injury or cervical surgery, neuromuscular or degenerative disease, traumatic temporomandibular joint injury, autoimmune joint disease, previous TMD treatment, pregnancy, active infection, neoplasia, and previous cervical rehabilitation.

Surface electromyography of the masticatory muscles was used clinically during the neuromuscular assessment and orthotic construction. However, the present study addresses mandibular movement only; EMG (electromyography) outcomes were not included in the current statistical analysis.

2.2. Audiologic, Otoneurological, and Symptom Assessment

All enrolled participants underwent a standardized audiologic and otoneurological evaluation to identify clinically evident primary auditory or vestibular disorders. The audiologic work-up included pure-tone audiometry in a sound-attenuated booth, with air-conduction thresholds assessed from 250 to 8000 Hz and bone-conduction thresholds assessed according to the ISO 8253-1 guidelines [17]. Tympanometry and acoustic reflex testing were performed; participants included in the TMD cohort showed Type A tympanograms and preserved stapedial reflexes.

A bedside neuro-otological examination included ocular pursuit, saccades, assessment for gaze-evoked nystagmus, positional and positioning maneuvers (including Dix–Hallpike and supine roll testing under Frenzel goggles), and bedside balance assessment with Romberg and Fukuda tests. This clinical screening was intended to exclude overt peripheral or central vestibular deficits; it did not constitute a quantitative longitudinal vestibular-function endpoint.

The clinical dataset recorded the presence of tinnitus, vertigo/dizziness, aural fullness (blocked-ear sensation), temporomandibular joint sounds, temporomandibular pain, cervical pain, and clenching/parafunction at T0. Symptoms were not mutually exclusive. At T1, clinical evolution was encoded categorically as resolved, improved, unchanged, worsened, or missing. Follow-up response for each symptom was conditioned on the presence of that symptom at baseline. Tinnitus Handicap Inventory (THI) scores were present in the working dataset but contained no usable paired T0/T1 values and were therefore not analyzed. THI > 40 was an exclusion criterion. Symptom improvement was based on patient self-report using a predefined scale. Dizziness-related disability was evaluated using the short-form Dizziness Handicap Inventory (DHIsf) developed by Tesio et al. [18]. The questionnaire comprises 13 dichotomous items, scored 1 for "yes" and 0 for "no", yielding a total score ranging from 0 to 13, with higher scores indicating greater self-perceived disability. These items explore three areas: eye and head movements, full-body activities, and mood alterations. No corresponding DHIsf assessment was available at T1; therefore, change in dizziness-related disability could not be quantified using a validated scale. At the 12-month visit, symptom evolution was categorized as resolved, improved, unchanged, worsened, or missing on the basis of patient report

For subjective vertigo/dizziness, aural fullness, and tinnitus, the proportion reporting improvement or resolution was calculated with exact binomial 95% confidence intervals. These proportions describe within-cohort symptom evolution and were not treated as estimates of treatment efficacy.

2.3. Neuromuscular Orthotic Intervention

The intervention consisted of a custom-made mandibular acrylic-resin repositioning orthotic constructed after ultra-low-frequency transcutaneous electrical nerve stimulation (ULF-TENS) and K7-assisted identification of a repeatable mandibular rest and closure trajectory. The clinical aim was to maintain the selected mandibular position during the treatment period rather than to provide a conventional protective bite guard.

ULF-TENS was delivered with a Myomonitor-type device using synchronous bilateral stimulation at approximately 40 pulses/min (0.66–0.67 Hz), a pulse duration of 500 μs, and an amplitude titrated from 0 mA up to the individual sensory/motor threshold, with a device range of approximately 0–25 mA. Electrodes were positioned bilaterally in the preauricular/coronoid-notch region according to the clinical protocol, with a common electrode posteriorly. Stimulation was applied for approximately 45–60 min while mandibular rest and closure trajectory were monitored. These stimulation parameters are consistent with previously described protocols used in TMD research [19].

Patients were instructed to wear the orthotic for approximately 20–22 h/day, removing it for meals, and were reassessed at 3-month intervals for clinical review and appliance adjustment. Objective electronic adherence monitoring was not available. The K7 system was used during follow-up to verify the mandibular position selected for the orthotic. At T0, the swallowing-related kinesiographic recording was acquired on natural dentition. At T1, 12 months later, the corresponding recording was performed with the orthotic in situ. This difference in recording condition is relevant to interpretation of longitudinal kinematic changes.

2.4. Kinesiographic Acquisition

Mandibular movement was recorded with the K7/EMG platform (version 15.0; Myotronics/Normed Inc., Tukwila, WA, USA). The system tracks displacement of a lightweight magnet positioned in the lower labial vestibular region relative to a head-

mounted sensor array and exports time-series data along the vertical (Z), anteroposterior (AP/X), and lateral (Y) axes together with an instrument-derived velocity channel.

Scan 20 recordings were used. Participants were instructed to relax and maintain the mandible at rest while movement was monitored. They were allowed to swallow spontaneously when the need arose and, after the swallowing event, were asked to close the mouth until dental/orthotic contact. The raw files contained five columns: Time, Vertical, AP, Lateral, and Velocity. Most recordings were sampled at approximately 240 Hz; a small number of baseline recordings used a higher sampling frequency and were standardized during computational preprocessing.

Although the beginning and end of the swallowing-related movement can be visually identified on the graphical Scan 20 trace, the exported raw text files do not contain a dedicated digital event marker. To avoid relying on manual caliper measurements of the plotted trace, the present reanalysis used an algorithmic event-segmentation procedure applied directly to the exported coordinates.

2.5. Post Hoc Raw-Signal Processing and Event Segmentation

Raw XYZ trajectories were processed in Python 3.13.5 using NumPy 2.3.5 and SciPy 1.17.0. All trajectories were linearly resampled to 240 Hz. Coordinates were low-pass filtered using a fourth-order zero-phase Butterworth filter with a 6 Hz cut-off frequency. Three-dimensional speed was reconstructed as the Euclidean norm of the first numerical derivative of the filtered XYZ coordinates.

The first 1.5 s of each trace were used to estimate resting motor activity. An adaptive detection threshold was defined as the maximum of 1.5 mm/s, the baseline median plus five median absolute deviations, and the 97.5th percentile of baseline speed. Candidate activity was searched after 1.2 s from recording onset and within the first 75% of the trace. Active components separated by no more than 0.55 s could be merged, with a maximum merged event duration of 4.0 s. A candidate segment qualified as a motor event when duration was at least 0.20 s, integrated movement/path was at least 0.5 mm, three-dimensional excursion was at least 0.35 mm, and peak speed was at least 2.0 mm/s.

Event segmentation was assigned a computational confidence category. Segments were classified as low confidence when duration exceeded 4.0 s, 3D excursion was <0.35 mm, or path length was <0.5 mm; as moderate confidence when duration exceeded 3.5 s or 3D excursion was <0.7 mm; and as high confidence otherwise. The complete 35-patient paired analysis was considered the principal kinematic analysis. A post hoc sensitivity analysis excluded pairs in which either T0 or T1 segmentation was classified as low confidence, leaving 27 paired observations.

The instrument-derived Velocity channel was retained for quality control but was not used to define the primary kinematic endpoints, because several near-stationary coordinate traces showed non-zero baseline values in that exported channel. Coordinate-derived 3D speed was therefore used consistently for event detection and speed-based outcomes.

2.6. Kinematic Outcomes

The segmented motor event was characterized using event duration (s); vertical, anteroposterior and lateral excursion (mm); three-dimensional excursion norm (mm); three-dimensional path length (mm); mean and peak three-dimensional speed (mm/s); SPARC; and log10-transformed dimensionless jerk. Path length was defined as cumulative Euclidean point-to-point displacement in three-dimensional space. SPARC and dimensionless jerk were included as exploratory smoothness descriptors and interpreted as method-dependent indices rather than interchangeable measures of a single construct [15,16].

2.7. Statistical Analysis

Continuous variables are presented as mean ± standard deviation and were also examined using medians and interquartile ranges in the analytic dataset. Because of the sample size and non-normal distribution of several kinematic measures, T0–T1 comparisons were performed with the Wilcoxon signed-rank test. Paired rank-biserial correlation was used as the effect-size measure in the sensitivity analysis. Independent comparisons between patients and healthy reference subjects were performed with the Mann–Whitney U test. Exact Clopper–Pearson binomial confidence intervals were calculated for clinical response proportions.

All tests were two-sided and $p < 0.05$ was considered statistically significant. No multiplicity correction was applied because the kinematic analysis was exploratory and no single primary kinematic endpoint had been prospectively specified. Consequently, isolated p values close to 0.05 were interpreted cautiously and in conjunction with direction, effect size, and consistency across related outcomes. Comparisons with healthy controls were considered descriptive because of the marked age imbalance.

2.8. Ethics and Reporting

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee Area 2, Milan, Italy, of Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico (protocol No. 653; approval date: 11 September 2018). Written informed consent was obtained from all participants or their legal guardians, as applicable. Data used for analysis were anonymized and stored under coded identifiers. Reporting was prepared with reference to the STROBE recommendations for observational studies [20].

3. Results

3.1. Study Population and Baseline Symptom Profile

All 35 patients had paired T0 and T1 K7 recordings available for longitudinal analysis. The patient cohort was older and considerably more age-heterogeneous than the asymptomatic reference group. As shown in Table 1 otologic and vestibular symptoms frequently overlapped with musculoskeletal TMD manifestations. Tinnitus was present in 24/35 patients (68.6%), subjective vertigo/dizziness in 10/35 (28.6%), and aural fullness in 8/35 (22.9%). Temporomandibular joint sounds were present in 30/35 (85.7%), temporomandibular pain in 18/35 (51.4%), cervical pain in 9/35 (25.7%), and clenching/parafunction in 14/35 (40.0%).

Table 1. Participant characteristics and baseline symptom prevalence. Values are mean ± SD (range) or n (%). The healthy group was used only as a cross-sectional kinematic reference.

Characteristics	TMD Patients (n = 35)	Healthy Subjects (n = 20)
Age, years	47.8 ± 18.7 (15–84)	24.7 ± 1.3 (23–27)
Sex, female/male	19/16	13/7
Tinnitus	24 (68.6%)	Asymptomatic by design
vertigo/dizziness	10 (28.6%)	Asymptomatic by design
Aural fullness	8 (22.9%)	Asymptomatic by design
TMJ sounds	30 (85.7%)	—
TMJ pain	18 (51.4%)	—
Cervical pain	9 (25.7%)	—
Clenching/parafunction	14 (40.0%)	—

3.2. Changes in Subjective Otologic and Vestibular Symptoms

Marked symptomatic improvement was recorded at the 12-month follow-up (Table 2). In particular, at baseline 10 patients reported vertigo/dizziness. The mean DHIsf total score was 4.2/13. Across the thirteen items, the total numbers of affirmative answers were 20 for full-body activities, 19 for eye/head movements, and 3 for mood-related items. These data provide a cross-sectional description of baseline self-reported burden; they cannot quantify change between T0 and T1. All 10 patients with vertigo/dizziness at baseline improved: four reported complete resolution and six reported improvement without complete resolution. The corresponding improvement/resolution proportion was 100% (10/10; exact 95% CI, 69.2–100%).

Among eight patients with aural fullness at baseline, one reported resolution, six improvement, and one no change, corresponding to 87.5% improvement/resolution (7/8; 95% CI, 47.3–99.7%).

Among 24 patients with tinnitus at baseline, 22 were coded as improved at T1 and two remained unchanged. The observed improvement proportion was 91.7% (22/24; 95% CI, 73.0–99.0%). Because no validated paired tinnitus-severity score was available, these findings describe categorical clinical change rather than a quantified change in tinnitus burden.

Table 2. Twelve-month clinical evolution among patients symptomatic at baseline. * Conservative denominator includes cases with missing T1 outcome. Symptoms were not mutually exclusive; outcomes were categorical and patient-reported.

Symptoms at T0	Baseline n	Resolved at T1	Improved at T1	Unchanged	Missing	Improved/Resolved
Subjective vertigo/dizziness	10	4	6	0	0	10/10 (100%)
Aural fullness	8	1	6	1	0	7/8 (87.5%)
Tinnitus	24	0	22	2	0	22/24 (91.7%)
TMJ Sounds	30	10	19	1	0	29/30 (96.7%)
TMJ Pain	18	9	9	0	0	18/18 (100%)
Cervical pain	9	0	7	2	0	7/9 (77.8%)
Clenching/parafunction	14	0	9	2	3	9/14 (64.3%) *

3.3. Main Longitudinal Kinematic Analysis

Across all 35 paired recordings, several kinematic variables changed consistently in the direction of reduced movement magnitude or speed, although none crossed the conventional $p < 0.05$ threshold (Table 3). Event duration decreased from 2.91 ± 1.43 s at T0 to 2.33 ± 1.47 s at T1 ($p = 0.097$). Vertical excursion decreased from 4.20 ± 3.16 to 3.14 ± 2.90 mm ($p = 0.084$), three-dimensional excursion from 5.19 ± 3.53 to 4.05 ± 3.46 mm ($p = 0.104$), and three-dimensional path length from 16.84 ± 14.60 to 11.94 ± 11.74 mm ($p = 0.078$). Mean three-dimensional speed decreased from 5.25 ± 2.88 to 4.42 ± 3.42 mm/s ($p = 0.065$).

The log10 dimensionless jerk decreased numerically from 7.93 ± 1.58 to 7.26 ± 1.81 ($p = 0.087$), whereas SPARC changed from −2.68 ± 0.68 to −2.46 ± 0.71 ($p = 0.184$). Taken together, the principal kinematic analysis indicated a coherent directional pattern but did not provide confirmatory statistical evidence for any individual endpoint.

Table 3. Primary T0–T1 kinematic analysis in all 35 paired recordings. Wilcoxon signed-rank test. No correction for multiple testing was applied; all kinematic outcomes were exploratory.

Kinematic Variable	T0 Mean ± SD	T1 Mean ± SD	*p* Value
Event duration (s)	2.91 ± 1.43	2.33 ± 1.47	0.097

Vertical excursion (mm)	4.20 ± 3.16	3.14 ± 2.90	0.084
3D excursion norm (mm)	5.19 ± 3.53	4.05 ± 3.46	0.104
3D path length (mm)	16.84 ± 14.60	11.94 ± 11.74	0.078
Mean 3D speed (mm/s)	5.25 ± 2.88	4.42 ± 3.42	0.065
Peak 3D speed (mm/s)	28.74 ± 22.17	24.19 ± 25.47	0.140
SPARC	−2.68 ± 0.68	−2.46 ± 0.71	0.184
log10 dimensionless jerk	7.93 ± 1.58	7.26 ± 1.81	0.087

3.4. Post Hoc Sensitivity Analysis of Higher-Confidence Segmentation

When the analysis was restricted to the 27 pairs in which both T0 and T1 motor-event segmentations were classified as high or moderate confidence, three changes reached $p < 0.05$ (Table 4). Vertical excursion decreased from 3.93 ± 3.07 to 2.73 ± 2.92 mm ($p = 0.049$; paired rank-biserial correlation, −0.434), three-dimensional path length decreased from 12.93 ± 10.13 to 9.18 ± 9.69 mm ($p = 0.049$; rank-biserial correlation, −0.434), and mean three-dimensional speed decreased from 4.90 ± 2.76 to 4.15 ± 3.70 mm/s ($p = 0.030$; rank-biserial correlation, −0.476). Three-dimensional excursion showed a similar directional change ($p = 0.058$).

Because segmentation confidence defined this subset post hoc and multiple correlated endpoints were tested without multiplicity correction, these findings are exploratory and cannot be considered confirmatory evidence of a treatment-related change.

Table 4. Post hoc sensitivity analysis restricted to 27 higher-confidence paired segmentations. Negative rank-biserial values indicate lower values at T1 for the first four variables. Analysis is post hoc and exploratory.

Kinematic Variable	T0 Mean ± SD	T1 Mean ± SD	*p* Value	Paired Rank Biserial
Vertical excursion (mm)	3.93 ± 3.07	2.73 ± 2.92	0.049	−0.434
3D excursion norm (mm)	4.78 ± 3.22	3.48 ± 3.39	0.058	−0.418
3D path length (mm)	12.93 ± 10.13	9.18 ± 9.69	0.049	−0.434
Mean 3D speed (mm/s)	4.90 ± 2.76	4.15 ± 3.70	0.030	−0.476
SPARC	−2.62 ± 0.73	−2.29 ± 0.68	0.081	+0.386

3.5. Exploratory Comparison with Healthy Reference Subjects

At baseline, patients showed larger and faster mandibular movement than the healthy reference group for most variables. Mean three-dimensional path length was 16.84 mm in patients at T0 versus 5.64 mm in controls ($p < 0.001$), mean three-dimensional speed was 5.25 versus 2.73 mm/s ($p < 0.001$), and vertical excursion was 4.20 versus 1.89 mm ($p < 0.001$). At T1, many patient–control differences were attenuated and no longer statistically significant. Three-dimensional path length yielded $p = 0.106$, vertical excursion $p = 0.323$, event duration $p = 0.270$, SPARC $p = 0.290$, and dimensionless jerk $p = 0.238$. Mean three-dimensional speed remained higher in patients than controls (4.42 vs. 2.73 mm/s; $p = 0.042$) (Table 5).

This pattern is compatible with a shift in several kinematic variables toward the healthy reference distribution. It does not demonstrate statistical equivalence or normalization, particularly because controls were substantially younger and because the T1 recordings were obtained with the orthotic in situ.

Table 5. Exploratory patient–control comparison for selected kinematic variables. Mann–Whitney U tests. Comparisons are exploratory; controls were not age-matched, and absence of a significant difference is not evidence of equivalence.

Variable	Control Mean	T0 vs. Control p	T1 vs. Control p
Event duration (s)	1.88	0.005	0.270
Vertical excursion (mm)	1.89	<0.001	0.323
AP excursion (mm)	1.18	<0.001	0.055
3D excursion norm (mm)	2.33	<0.001	0.113
3D path length (mm)	5.64	<0.001	0.106
Mean 3D speed (mm/s)	2.73	<0.001	0.042
Peak 3D speed (mm/s)	11.43	<0.001	0.062
SPARC	−2.27	0.032	0.290
log10 dimensionless jerk	6.79	0.005	0.238

4. Discussion

This exploratory longitudinal study produced two principal observations. First, subjective otologic and vestibular complaints improved markedly during the 12 months of neuromuscular orthotic therapy. All patients with baseline subjective vertigo/dizziness reported improvement or resolution, while improvement was also frequent for aural fullness and tinnitus. Second, reanalysis of the raw mandibular trajectories showed a coherent directional reduction in several measures of movement magnitude, trajectory length, speed, and jerk. These kinematic changes did not reach conventional statistical significance in the complete 35-patient paired analysis, but selected measures reached $p < 0.05$ in a post hoc sensitivity analysis restricted to higher-confidence event segmentations.

The data should be interpreted as temporal associations rather than evidence of a causal chain. The study does not demonstrate recovery of vestibular function, because no instrumented or quantitative vestibular-function endpoint was followed longitudinally. It also does not demonstrate that the kinematic changes mediated the symptomatic response. In addition, the T0 and T1 kinesiographic recordings were acquired under different occlusal conditions, natural dentition at T0 and with the orthotic in situ at T1, so the observed kinematic shift cannot be separated into long-term neuromotor adaptation versus the immediate mechanical effect of the appliance during recording.

The frequency of otologic complaints in the present cohort is consistent with the broader TMD literature. Porto De Toledo et al. reported substantial prevalences of ear fullness, tinnitus, and vertigo among adults with TMD [2]. A recent systematic review and meta-analysis likewise found dizziness and vestibular symptoms to be common in TMD populations while emphasizing that the available evidence is largely observational and cannot establish causation [4].

The finding for subjective vertigo/dizziness is clinically notable: 10 of 10 symptomatic patients reported improvement or resolution. However, the denominator was small and the exact confidence interval was correspondingly wide. Baseline bedside neuro-otological screening reduced the likelihood of an overt peripheral or central vestibular disorder, but no quantitative longitudinal vestibular tests were available, and although dizziness-related disability was assessed using the DHIsf at baseline, paired follow-up data were unavailable.

A proprioceptive or somatosensory contribution, potentially involving altered cervical or stomatognathic afferent input, may represent one possible explanation for subjective unsteadiness in patients with TMD. Nevertheless, this hypothesis cannot be confirmed, and concomitant vestibular involvement cannot be definitively excluded.

This distinction is especially important given the current interventional evidence. Ferrillo et al. identified randomized evidence for rehabilitative treatment of secondary otalgia and tinnitus in patients with TMD but found no randomized controlled trials evaluating dizziness, vertigo, or aural fullness [10]. Therefore, the present prospective symptom observations address a clinically under-investigated domain, but they remain

hypothesis-generating. A controlled study using standardized dizziness instruments and objective vestibular testing is required before vestibular improvement can be attributed to orthotic therapy.

Tinnitus improved in 22 of 24 patients who reported it at baseline. This response pattern is compatible with the concept that selected tinnitus presentations may have a somatosensory component related to the temporomandibular region. Consensus work has emphasized jaw/neck modulation, temporal relationships between tinnitus and musculoskeletal symptoms, and related clinical features as supportive rather than individually definitive criteria for somatosensory tinnitus [5]. Experimental evidence of somatosensory–auditory convergence within the dorsal cochlear nucleus provides a plausible neurophysiological substrate [7,8].

Clinical studies also suggest that treatment of the temporomandibular or broader orofacial system may reduce tinnitus burden in selected patients. A randomized controlled trial of multidisciplinary orofacial treatment demonstrated benefit in temporomandibular somatosensory tinnitus [13], and systematic reviews continue to support an association between TMD and tinnitus while recognizing heterogeneity and methodological limitations [9,10]. Previous work also documented frequent coexistence of somatosensory tinnitus and TMD in a separate cohort [21]

Aural fullness improved or resolved in seven of eight baseline cases. TMD has been proposed as one possible contributor to otherwise unexplained aural fullness [12], and recent neurotologic work emphasizes a broad differential diagnosis in patients whose standard otologic evaluation is normal [22]. The present finding should therefore not be generalized to patients with primary middle-ear, inner-ear, neurologic, or migraine-related disease. The standardized audiologic and bedside otoneurological screening used here strengthens phenotyping, but does not eliminate all alternative mechanisms.

The kinematic component provides a description of mandibular motor behavior, but it is secondary to the clinical symptom findings and requires cautious interpretation because baseline movements were measured on natural dentition, whereas follow-up movements were recorded with the orthotic in situ. Accordingly, the observed differences may reflect immediate mechanical guidance, altered vertical dimension or contact conditions, longer-term adaptation, or a combination of these factors. In the complete paired dataset, vertical excursion, three-dimensional path length, mean speed, and dimensionless jerk all decreased numerically, with several p values between 0.05 and 0.10. The higher-confidence sensitivity analysis strengthened this directional pattern and identified reductions in vertical excursion, path length, and mean speed with moderate paired rank-biserial effect sizes.

At the same time, the sensitivity analysis was post hoc and involved multiple correlated outcomes. More importantly, the orthotic was present during the T1 recording but not during T0. Consequently, a smaller excursion or shorter path at T1 may reflect both adaptation accrued during 12 months of therapy and immediate mechanical guidance or constraint from the appliance. The present design cannot partition these components. A future protocol should acquire T1 recordings both with and without the orthotic, ideally in randomized order, to distinguish treatment-state mechanics from persistent motor adaptation.

The reanalysis also illustrates why operational definitions are essential when terms such as “smoothness” or “jitter” are used. Smoothness is not a single universal variable; estimates vary with filtering, sampling, numerical differentiation, movement amplitude, duration, and segmentation [15,16]. The current analysis therefore used explicitly defined geometric and derivative-based measures. Earlier working values labeled “velocity jitter” and “Z-axis instability” were not retained because their computational derivation was not sufficiently documented for independent reproduction from the raw trajectories.

Several mechanisms could plausibly connect TMD-related somatosensory dysfunction with auditory symptoms. Trigeminal and cervical inputs can modulate central auditory processing, and maladaptive interactions within the dorsal cochlear nucleus are well described in somatosensory tinnitus [7,8]. Mechanical relationships between the temporomandibular region and the middle ear have also been discussed, although such anatomical considerations do not establish symptom causality. Extending a somatosensory brainstem model to dizziness or vertigo is biologically plausible but substantially less well demonstrated than for tinnitus.

The present data do not directly measure trigeminal excitability, central sensory integration, cochlear activity, vestibulo-ocular reflexes, otolith function, or postural control. Therefore, reductions in mandibular path length or speed should not be labeled as proof of "motor engram reprogramming," "removal of a perpetuating factor," or restoration of vestibular physiology. The experimentally supported observation is narrower: patient-reported otologic/vestibular symptoms improved during treatment, while the recorded swallowing-related mandibular movement pattern also changed over time under a different occlusal recording condition.

The main implication is not that an occlusal orthotic should be considered a primary treatment for vestibular disease. Rather, TMD assessment may be clinically relevant in patients with tinnitus, aural fullness, or dizziness when standard otologic evaluation does not identify a sufficient explanation and temporomandibular or masticatory signs coexist. This consideration is most established for somatosensory tinnitus, where multidisciplinary assessment and orofacial treatment already have a growing evidence base [5,9,10,13].

For dental clinicians, the results reinforce the need for precise symptom phenotyping and collaboration with audiology/ENT services. Improvement in a self-reported vestibular complaint should not be equated with resolution of vestibular pathology. Future studies should prospectively combine DC/TMD characterization with validated tinnitus and dizziness questionnaires, instrumented vestibular testing, standardized orthotic adherence data, and prespecified kinematic endpoints.

Neuroanatomical and physiological evidence provides a plausible basis for interactions between trigeminal somatosensory input and auditory processing at both subcortical and cortical levels. Somatosensory afferents originating from trigeminal structures, together with projections from brainstem somatosensory nuclei, reach the cochlear nuclei, where they can modulate neuronal firing and responses to acoustic stimulation. These interactions have been implicated in the pathophysiology of somatosensory tinnitus [23,24]. Multisensory convergence is also documented at cortical levels: somatosensory inputs can influence auditory cortical processing, including activity in the caudal belt regions, while vestibular signals are integrated with proprioceptive and other sensory information within a distributed cortical network that includes the parieto-insular vestibular cortex and somatosensory regions [25,26]. These pathways provide a potential neurobiological framework in which altered trigeminal or mandibular sensory input could interact with auditory and vestibular processing. However, the present study did not directly assess these neural pathways, and the observed symptom changes cannot establish that such interactions were responsible for the reported clinical outcomes.

A strength of this study is the availability of paired 12-month raw kinesiographic recordings for all 35 patients and the use of a transparent coordinate-based processing pipeline rather than reliance on undocumented summary indices. The clinical cohort underwent audiologic and bedside otoneurological screening, and the healthy reference group provided context for the direction of kinematic differences. The study also addresses dizziness and aural fullness, outcomes for which randomized TMD-treatment evidence is notably sparse.

The limitations are substantial. First, there was no untreated or sham-treated TMD control group; treatment-specific effects cannot be separated from natural history, regression to the mean, expectancy effects, behavioral changes, or concurrent care. Second, symptom outcomes were categorical and patient-reported, without validated scales or usable paired THI values. Third, although baseline clinical vestibular screening was performed, no quantitative instrumented vestibular outcome was measured longitudinally. Fourth, healthy reference subjects were considerably younger than patients and were unsuitable for causal between-group inference, thus precluding conclusions. Fifth, the swallowing-related event was not digitally marked in the exported raw files; segmentation was developed post hoc, and the sensitivity analysis was also post hoc. Sixth, multiple kinematic outcomes were assessed without multiplicity correction. Seventh, T0 and T1 kinematics were acquired under different occlusal conditions, with the orthotic present at T1 only; this is a major source of measurement-condition confounding. Eighth, orthotic adherence was prescribed but not objectively monitored. Finally, symptom-specific subgroup sizes were small, particularly for dizziness and aural fullness.

These limitations define the appropriate role of study. The findings support a prospective clinical signal and a parallel exploratory kinematic signal, but not a definitive treatment-effect estimate or mechanistic proof. They provide a basis for a controlled prospective trial designed specifically around audiologic and vestibular outcomes.

5. Conclusions

In this 12-month exploratory cohort of patients with TMD and associated otologic or vestibular complaints, neuromuscular mandibular orthotic therapy was followed by frequent self-reported improvement in subjective vertigo/dizziness, tinnitus, and aural fullness. Raw K7 reanalysis also showed a directional shift toward smaller mandibular excursions, shorter three-dimensional trajectories, and lower movement speed; selected differences reached statistical significance only in a post hoc higher-confidence segmentation analysis. Many baseline kinematic differences from younger healthy reference subjects were attenuated at follow-up. These observations suggest an association between symptom evolution and changes in the treated mandibular motor state, but they do not establish recovery of vestibular function, a causal effect of the orthotic, or a specific trigemino-vestibular mechanism. Future controlled studies should include age-matched comparison groups, standardized tinnitus and dizziness instruments, quantitative vestibular testing, objective adherence monitoring, prespecified kinematic endpoints, and T1 recordings both with and without the orthotic.

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Abbreviations

The following abbreviations are used in this manuscript:

TMD	Temporomandibular disorder
TMJ	Temporomandibular joint
EMG	Electromyography
SPARC	Spectral Arc Length
DC/TMD	Diagnostic Criteria for Temporomandibular Disorders
THI	Tinnitus Handicap Inventory
DHIsf	Dizziness Handicap Inventory
ULF-TENS	Ultra-low-frequency Transcutaneous Electrical Nerve Stimulation
ENT	Ear, Nose and Throat

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