Vietnam Journal of Community Medicine, Vol. 67, English version No. 1, 2026, 240-244

EFFECT OF MOVEMENT DIRECTION AND MAGNITUDE ON INFERIOR ALVEOLAR NERVE RECOVERY AFTER BILATERAL SAGITTAL SPLIT OSTEOTOMY: A 24-MONTH PROSPECTIVE COHORT STUDY

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Received: 12/05/2026
Revised: 20/05/2026; Accepted: 25/05/2026

ABSTRACT

Objective: To evaluate the effects of surgical movement direction (advancement vs. setback) and magnitude on inferior alveolar nerve (IAN) recovery following bilateral sagittal split osteotomy (BSSO) over 24 months.

Methods: A prospective cohort study enrolled 94 patients undergoing BSSO (47 advancements, 47 setbacks). IAN function was assessed at seven timepoints using the Visual Analog Scale (VAS) for subjective evaluation and the Zuniga classification for objective evaluation: Level A (two-point discrimination), Level B (static light touch), and Level C (pain/thermal thresholds). Statistical analyses included repeated-measures ANOVA (RM-ANOVA), Cochran's Q, and multivariate logistic regression.

Results: Neurosensory disturbance (NSD) peaked at one week (93.62% advancement; 95.74% setback). Level C fibers fully recovered by 12 months; Level A deficits persisted in 6.38% and 8.51% of patients at 24 months. Movement direction did not significantly affect NSD recovery ($p > 0.05$). Multivariate regression identified movement magnitude ≥5 mm (OR = 3.45; $p = 0.01$) and age ≥25 years (OR = 2.80; $p = 0.03$) as independent risk factors for persistent NSD at 6 months. VAS scores correlated strongly with Level A deficits ($\rho = 0.79–0.90$).

Conclusion: IAN recovery after BSSO is independent of movement direction but significantly delayed by older age and larger surgical movements. Level A sensory assessment (two-point discrimination) most accurately reflects patient-reported long-term paresthesia.

Keywords: Bilateral sagittal split osteotomy, inferior alveolar nerve, neurosensory disturbance, Zuniga classification.

1. INTRODUCTION

Bilateral sagittal split osteotomy (BSSO) is the most commonly performed orthognathic procedure, widely indicated for the correction of mandibular skeletal discrepancies including prognathism (Class III) and retrognathism (Class II). Despite its well-established aesthetic and functional outcomes, BSSO carries a significant risk of inferior alveolar nerve (IAN) injury, with early neurosensory disturbance (NSD) rates reaching 80–100% and long-term dysesthesia ranging from 0% to 85%, depending on the assessment method and follow-up interval [1].

IAN injuries in BSSO predominantly involve temporary conduction block (neuropraxia) or axonal disruption with preserved myelin sheath (axonotmesis), arising from dissection, soft-tissue retraction, and pressure exerted by fixation hardware [2, 3]. The anatomical relationship between the mandibular canal and the osteotomy line has also been identified as a contributing risk factor [4]. A persistently debated question is whether the direction of mandibular displacement (advancement vs. setback) influences the incidence and recovery trajectory of NSD; reported inconsistencies are largely attributed to heterogeneous measurement tools and short follow-up durations [1, 5].

The Zuniga and Essick classification system is recognized as the clinical gold standard for peripheral sensory nerve evaluation, enabling systematic stratification of dysfunction by nerve fiber subtype [6]. Clinical and electrophysiological evidence indicates that small unmyelinated fibers (Level C) recover within 3–6 months, whereas large myelinated fibers (Level A) may require 12–24 months [7]. Nevertheless, longitudinal application of the Zuniga classification to directly compare advancement and setback cohorts over extended follow-up remains limited.

Accordingly, this 24-month prospective cohort study was designed to evaluate the effects of surgical movement direction (advancement vs. setback) and magnitude on inferior alveolar nerve (IAN) recovery following bilateral sagittal split osteotomy (BSSO) over 24 months.

2. SUBJECTS AND METHODS

2.1. Study population, setting, and period

Ninety-four adult patients indicated for BSSO were enrolled at Van Hanh General Hospital and assigned to two groups: Advancement group (Mandibular Advancement, n = 47) and Setback group (Mandibular

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Setback, n = 47). All procedures were performed under general anesthesia by the same experienced maxillofacial surgical team.

- Inclusion criteria: Adults aged ≥18 years with stable general health (ASA I or II); diagnosed with mandibular skeletal discrepancy indicated for BSSO; complete imaging records and surgical plan; written informed consent and willingness to comply with follow-up schedules.

- Exclusion criteria: Pre-existing peripheral neuropathy or prior trigeminal nerve injury; intraoperative adverse events including bad split or nerve transection (neurotmesis).

2.2. Study methods

- Study design: Prospective cohort study with 24-month longitudinal follow-up. Sample size was calculated at α = 0.05, statistical power 1−β = 0.80, and effect size 0.44, requiring a minimum of 82 patients; 94 patients were enrolled to account for potential attrition.

- Neurosensory evaluation protocol: IAN function was assessed at seven timepoints: pre-operatively (T0) and post-operatively at 1 week (T1), 1 month (T2), 3 months (T3), 6 months (T4), 12 months (T5), and 24 months (T6). The examination area comprised the lower lip and chin bilaterally, divided into four zones. Subjective assessment used a 100-mm Visual Analog Scale (VAS; 0 = normal sensation, 100 = complete anesthesia). Objective assessment followed the Zuniga and Essick three-tier classification [6]: Level A (large myelinated A-alpha/beta fibers) assessed by Two-Point Discrimination (TPD) using a Boley gauge, normal threshold <15 mm; Level B (small A-beta fibers) assessed by Static Light Touch (SLT) using Semmes-Weinstein monofilaments; Level C (unmyelinated A-delta/C fibers) assessed by pinprick (27-gauge needle) and thermal discrimination (ethyl chloride vs. heated metal probe). Deficit was defined as abnormal response in ≥3 of 4 trials per zone.

- Bias control: Examining clinicians were blinded to surgical technique and movement magnitude. Intraclass Correlation Coefficients (ICC) >0.85 were required for both intra-rater and inter-rater reliability, confirmed on 20 randomly selected patients.

2.3. Study variables and indicators

- The study variables included age, sex, direction of mandibular movement, magnitude of movement, operative time, and indicators of inferior alveolar nerve sensory function.

- The direction of mandibular movement was classified into two groups: advancement and setback. The magnitude of movement was recorded in millimeters and categorized as <5 mm and ≥5 mm. Age was recorded in years and categorized as <25 years and ≥25 years.

- Inferior alveolar nerve sensory function was assessed at the following time points: before surgery and at 1 week, 1 month, 3 months, 6 months, 12 months, and 24 months after surgery. Subjective assessment was performed using a 100-mm Visual Analog Scale (VAS), in which 0 indicated normal sensation and 100 indicated complete sensory loss.

- Objective assessment was performed according to the Zuniga and Essick classification, including Level A for two-point discrimination, Level B for static light touch, and Level C for pain and thermal sensation. Neurosensory disturbance was defined as an abnormal response in the lower lip or chin region compared with the preoperative status.

- The primary outcome was persistent inferior alveolar nerve neurosensory disturbance after surgery, particularly at 6 months. The factors analyzed included direction of mandibular movement, magnitude of movement, age, sex, and operative time.

2.4. Statistical analysis

Data were analyzed using SPSS 26.0. Between-group comparisons used the Independent t-test (normally distributed continuous variables), Mann-Whitney U test (ordinal variables), and Chi-square test (categorical variables). Longitudinal within-group changes were assessed by RM-ANOVA (continuous), Friedman test, and Cochran's Q test (proportions). Multivariate logistic regression identified independent predictors of persistent NSD, expressed as Odds Ratios (OR) with 95% confidence intervals (95% CI). Statistical significance threshold was $p < 0.05$.

2.5. Ethical approval

The study received ethical approval from the Institutional Ethics Committee of Tra Vinh University (Ref: 185/GCN.ĐC-HĐĐ) and Van Hanh General Hospital (Ref: 01/2025/GCN-HĐĐ). All data were anonymized and used exclusively for research purposes.

3. RESULTS

Reliability analysis on 20 randomly selected patients yielded ICC values of 0.87–0.93 (Level A and C) and ICC = 0.88 (Level B), confirming excellent measurement consistency throughout the 24-month observation period.

Table 1. Baseline demographic and surgical characteristics (N = 94)

Clinical characteristic	Advancement group (n = 47)	Setback group (n = 47)	p-value	Clinical characteristic	Advancement group (n = 47)	Setback group (n = 47)	p-value
Age (years), mean ± SD	24.50 ± 4.20	25.10 ± 4.50	0.51[1]	Movement magnitude (mm), mean ± SD	6.20 ± 1.80	5.80 ± 2.10	0.32[1]
Age category: ≥25 years (%)	21 (44.68%)	24 (51.06%)	0.53[2]	Magnitude ≥5.00 mm (%)	34 (72.34%)	31 (65.96%)	0.50[2]
Sex, Female/Male (%)	28/19 (59.57/40.43%)	30/17 (63.83/36.17%)	0.67[2]	Operating time (min), mean ± SD	115.40 ± 18.20	122.30 ± 20.50	0.09[1]

[1]: Independent t-test; [2]: Chi-square test. Statistical significance p < 0.05.

The two groups were balanced at baseline, with no statistically significant differences in age, sex, movement magnitude, or operating time (p > 0.05) (Table 1).

Table 2. IAN neurosensory assessment and recovery by Zuniga classification over 24 months

Timepoint	Group	Subjective NSD (%)	Level A - TPD (mm ± SD; n abn.%)	Level B - SLT (g; n abn.%)	Level C n abnormal (%)
Pre-op (T0)	Adv.	0.00	8.10 ± 1.20; 0 (0.00%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
	Set.	0.00	7.90 ± 1.15; 0 (0.00%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
1 Week (T1)	Adv.	93.62	18.50 ± 4.20; 44 (93.62%)	0.021 ± 0.02; 42 (89.36%)	39 (82.98%)
	Set.	95.74	19.10 ± 4.50; 45 (95.74%)	0.023 ± 0.03; 43 (91.49%)	40 (85.11%)
1 Month (T2)	Adv.	74.47	14.20 ± 3.80; 35 (74.47%)	0.015 ± 0.01; 28 (59.57%)	21 (44.68%)
	Set.	78.72	15.00 ± 3.90; 37 (78.72%)	0.017 ± 0.02; 31 (65.96%)	23 (48.94%)
3 Months (T3)	Adv.	42.55	11.50 ± 2.50; 20 (42.55%)	0.010 ± 0.01; 12 (25.53%)	7 (14.89%)
	Set.	44.68	12.10 ± 2.70; 21 (44.68%)	0.011 ± 0.01; 14 (29.79%)	8 (17.02%)
6 Months (T4)	Adv.	19.15	9.80 ± 2.10; 9 (19.15%)	0.008 ± 0.00; 2 (4.25%)	1 (2.13%)
	Set.	21.28	10.20 ± 2.20; 10 (21.28%)	0.008 ± 0.00; 3 (6.38%)	2 (4.25%)
12 Months (T5)	Adv.	10.64	8.70 ± 1.80; 5 (10.64%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
	Set.	12.77	9.10 ± 1.90; 6 (12.77%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
24 Months (T6)	Adv.	6.38	8.30 ± 1.40; 3 (6.38%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
	Set.	8.51	8.50 ± 1.50; 4 (8.51%)	0.008 ± 0.00; 0 (0.00%)	0 (0.00%)
Time-effect test (p)		Cochran's Q p < 0.001	RM-ANOVA p < 0.001 Cochran's Q p < 0.001	Friedman p < 0.001 Cochran's Q p < 0.001	Cochran's Q p < 0.001

abn. = abnormal; Cochran's Q: proportions; RM-ANOVA: continuous variables; Friedman: non-parametric ordinal.

Table 2 demonstrates that subjective NSD peaked at one week post-surgery (93.62% advancement; 95.74% setback) and declined progressively. Recovery rates differed markedly by fiber subtype: Level C fibers recovered fastest, reaching 100% normality by 12 months in both groups. Level B fibers also achieved complete recovery by 12 months. Level A fibers showed the slowest recovery; at 24 months, 6.38% of advancement and 8.51% of setback patients remained abnormal on TPD. Cochran's Q, RM-ANOVA, and Friedman tests confirmed significant time-effects across all sensory parameters ($p < 0.001$).

Table 3. Persistent NSD analysis and subjective-objective correlations over 24 months

Factor analyzed	T1 (1 wk)	T2 (1 mo)	T3 (3 mo)	T4 (6 mo)	T5 (12 mo)	T6 (24 mo)
Univariate analysis of subjective NSD – Chi-square test (p-value)						
Movement direction (Advancement vs. Setback)	0.65	0.58	0.49	0.38	0.41	0.51
Movement magnitude (<5.00 mm vs. ≥5.00 mm)	0.01*	0.01*	0.02*	0.01*	0.08	0.15
Age (<25 vs. ≥25 years)	0.03*	0.04*	0.04*	0.03*	0.12	0.20
Subjective (VAS) vs. objective (Zuniga) correlation – Spearman's ρ						
Level A (TPD – Two-point discrimination)	0.82**	0.84**	0.86**	0.79**	0.88**	0.90**
Level B (SLT – Static light touch)	0.61*	0.64*	0.55*	0.46	N/A	N/A
Level C (Pinprick – Pain threshold)	0.45	0.35	N/A	N/A	N/A	N/A

** p < 0.05; ** p < 0.01. N/A: correlation not computed as Level B and C abnormality rates approached zero at later timepoints*

Univariate analysis (Table 3) showed that movement direction produced no significant difference in NSD rates at any timepoint ($p > 0.38$). Movement magnitude ≥5 mm and age ≥25 years were significantly associated with persistent NSD during the first 6 months ($p < 0.05$). Spearman correlations between VAS and Level A remained consistently high throughout 24 months ($\rho = 0.79–0.90$; $p < 0.01$), while Level B and C correlations were unavailable at later timepoints as abnormality rates approached zero.

Table 4. Multivariate logistic regression for independent predictors of persistent NSD at 6 months (N = 94)

Variable	OR	95% CI	p-value
Movement direction (Advancement as reference)	1.15	0.45 – 2.85	0.51
Movement magnitude ≥5.00 mm	3.45	1.25 – 9.50	0.01*
Age ≥25 years	2.80	1.10 – 7.15	0.03*

** p < 0.05 indicates statistical significance*

The multivariate logistic regression model (Table 4) confirmed that movement direction was not an independent predictor of persistent NSD (OR = 1.15; 95% CI: 0.45–2.85; p = 0.51). Two independent risk factors were identified: movement magnitude ≥5 mm (OR = 3.45; 95% CI: 1.25–9.50; p = 0.01) and age ≥25 years (OR = 2.80; 95% CI: 1.10–7.15; p = 0.03).

4. DISCUSSION

The central question of this study was whether the direction of mandibular displacement constitutes a determinant of persistent IAN neurosensory deficit after BSSO. Both univariate and multivariate analyses consistently demonstrated that movement direction carries no independent predictive value for NSD at 6 months (OR = 1.15; p = 0.51). This finding aligns with systematic reviews in the literature reporting no significant difference in IAN injury rates between advancement and setback procedures [2, 5].

The mechanistic equivalence between both surgical directions is explained by the shared nature of the resulting nerve injury. In advancement, the IAN sustains longitudinal tensile force, inducing ischemia of the vasa nervorum (intraneural microvasculature). In setback, the nerve tends to buckle and kink between the osteotomy segments, generating equivalent compressive force on the nerve sheath. Both mechanisms converge on the same injury phenotype, axonotmesis, characterized by disruption of the internal myelin architecture with preservation of the epineurium [2, 3]. This shared pathophysiology explains the absence of between-group differences in recovery speed and extent.

In contrast to movement direction, magnitude ≥5 mm was confirmed as a significant independent risk factor (OR = 3.45; p = 0.01). This is directionally consistent with findings by Ylikontiola et al., who demonstrated significantly increased NSD with larger mandibular displacements [8]. The 5-mm threshold identified here is lower than the commonly cited threshold of >7 mm, suggesting that even moderate-magnitude movements warrant meticulous technique with minimal IAN dissection and retraction [2, 8]. The recent report by Li et al. (2025) on the role of lingual split pattern in IAN recovery further reinforces the clinical importance of microsurgical nerve preservation techniques [9].

Age ≥25 years was also confirmed as a significant independent risk factor (OR = 2.80; p = 0.03). The relationship between advancing age and delayed NSD recovery after BSSO has been demonstrated by Van Sickels et al. in a large patient series [10], and corroborated by Bruckmoser et al. [5]. The biological basis encompasses age-related declines in neuroplasticity, peripheral perfusion, and Schwann cell remyelination capacity, all of which prolong the nerve regeneration timeline. The relatively low threshold of 25 years likely reflects the young demographic profile of this cohort, in which even modest age differences produced clinically meaningful differences in recovery trajectory.

The 24-month longitudinal Zuniga data (Table 2) reveal a physiologically coherent, fiber-dependent recovery pattern. Level C fibers (unmyelinated or thinly myelinated) recovered most rapidly, achieving full normality by 12 months, consistent with their relative resistance to ischemic injury and high regenerative velocity [7]. Level A fibers (large myelinated, governing TPD) require complex, protracted remyelination, resulting in 6.38–8.51% persistent abnormality at 24 months. The sustained high Spearman correlations between VAS and Level A deficits (ρ = 0.79–0.90; p < 0.01) throughout the observation period confirm that patient-reported long-term paresthesia principally reflects large-fiber dysfunction [6, 7]. When small fibers (Level C) have recovered but large fibers (Level A) remain impaired, loss of gate-control mechanisms causes ordinary tactile stimuli to be perceived as dysesthesia or allodynia. Clinically, this finding advocates for prioritizing TPD assessment (Level A) in long-term sensory follow-up after BSSO.

This study has several limitations. The relatively small sample size (n = 94) constrains further subgroup analyses. Evaluation relied exclusively on validated clinical tests without quantitative electrophysiological methods such as trigeminal somatosensory evoked potentials. Additionally, the non-randomized cohort design cannot fully exclude unmeasured confounders.

5. CONCLUSION

This 24-month prospective cohort study of 94 BSSO patients demonstrates that IAN neurosensory disturbance peaks at one week post-surgery and recovers progressively in a fiber-dependent pattern. Level C fibers recover completely by 12 months, while Level A fibers remain abnormal in 6.38–8.51% of patients at 24 months. Movement direction carries no independent prognostic value for NSD outcomes. Movement magnitude ≥5 mm and age ≥25 years are statistically significant independent risk factors that should be incorporated into preoperative counseling and postoperative monitoring protocols. Level A sensory assessment (TPD) is the most accurate objective correlate of patient-reported long-term paresthesia and should be prioritized in clinical sensory follow-up after BSSO.

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