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Do positional changes of the inferior alveolar canal after sagittal split mandibular osteotomy affect neurosensory recovery?

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Abstract. The purpose of this study was to assess the pre- and postoperative position and dimensions of the inferior alveolar canal (IAC) following sagittal split osteotomy (SSO) and identify any association with postoperative neurosensory deficit (NSD) at 1 year. This retrospective cohort study enrolled consecutive patients who had SSO performed to correct skeletal malocclusion. The pre- and postoperative cone beam computed tomography data were superimposed to visualize differences in IAC position and dimensions. Subjective and objective neurosensory tests were used to determine NSD in the inferior alveolar nerve distribution. A total of 20 subjects were included. The preoperative distance from the lateral cortex of the IAC to the inner aspect of the lateral cortex of the mandible was significantly greater in sides with NSD when compared to sides without NSD ($P = 0.01$). A significantly greater reduction in the postoperative distance measurement was seen in sides with NSD when compared to sides without NSD ($P = 0.01$). The magnitude of mandibular movement was significantly increased in sides with NSD ($P = 0.02$). The preoperative location of the IAC, as well as certain changes in the mediolateral and vertical positions as a result of SSO, are risk factors for postoperative NSD.

Key words: neurosensory deficit; sagittal split osteotomy; superimposition; cone beam computed tomography; inferior alveolar nerve.

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The sagittal split osteotomy (SSO), indicated for the surgical correction of dentofacial skeletal abnormalities involving the mandible, is one of the most commonly performed orthognathic procedures. Although patients undergoing SSO report a significant improvement in oral and general health-related quality of life and physiological function, the most commonly reported persistent complaint of patients following SSO is an unpleasant or altered sensation of the lower lip and chin caused by injury to the inferior alveolar nerve (IAN)[1].

The incidence of neurosensory deficit (NSD) of the lower lip and chin after SSO varies in the literature. The incidence of short-term neurosensory impairment has been reported to be between 63.3% and 83% in the early postoperative period, whereas long-term NSD has been reported to be 12.8–39% at 1 year following the surgery[2]. In general, altered sensation tends to be temporary and often recovers within 6 months following SSO[3]. However, up to 40% of patients undergoing SSO experience permanent NSD (>1 year) with an incidence of 22.6% at the 1-year follow-up[4].

The IAN is at significant risk during all phases of an SSO procedure[5]. Even if the nerve maintains its integrity during the surgery, any surgical manipulation of the nerve, its vascular supply, or the mesoneurial structures surrounding the nerve by compression or stretching may result in impaired IAN function. This manipulation is extremely variable, and rarely reported, but can be responsible for significant IAN dysfunction in the absence of a frank IAN transection injury[4,6,7]. Furthermore, the proximal and distal bone fragments may interfere with, or alter the course of the inferior alveolar canal (IAC) following SSO. The IAN may be damaged either directly from bony interferences, especially in cases of asymmetric mandibular repositioning, or via entrapment of the nerve between bony segments[7]. Following bony healing after SSO procedures with mandibular repositioning, the IAC path in the mandibular body and ramus changes from the preoperative position, but the relationship between these changes and the occurrence of IAN paresthesia is unknown.

Compression or crush injuries generally cannot be visualized by either magnetic resonance imaging (MRI) or computed tomography (CT), although magnetic resonance neurography (MRN) shows promise in the diagnosis of trigeminal nerve injury[7]. In one study, nerve damage was not appreciated on MRI or CT imaging in a case series after SSO surgery, even in the presence of neuropathic pain or a laceration or discontinuity of the nerve[8]. Therefore, since the IAN is not easily visualized, the borders of the IAC may be used to approximate the location of the IAN[9].

The proposed hypotheses regarding the causal factors of IAN injury and the risk factors related to SSO are highly debated. Also, the underlying reasons for cases of persistent NSD in the chin and/or lower lip are still poorly understood. The goal of the present study was to elucidate the relationship between the positional and dimensional changes of the IAC on cone beam computed tomography (CBCT) with NSD at 1 year postoperative following SSO. The primary outcome measure was neurosensory status (subjective and objective scores). The primary predictor variable was postoperative changes in IAC position. Secondary predictor variables included age, sex, skeletal deformity type, timing of mandibular third molar removal, and magnitude of the surgical movement.

Materials and methods

Study design

A retrospective cohort study design was implemented that included patients diagnosed with a skeletal class II or III dentofacial deformity, who subsequently underwent orthognathic surgery between June 2017 and June 2019 in the Department of Oral and Maxillofacial Surgery, University of Illinois at Chicago. Inclusion criteria were (1) follow-up period of at least 1 year, (2) presence of subjective (visual analog scale, VAS) and objective (Medical Research Council Scale, MRCS) neurosensory testing, (3) presence of preoperative and 1-year postoperative CBCT scans, and (4) availability of surgical planning records. Exclusion criteria were (1) presence of pre-surgical NSD of the IAN, (2) previous facial surgery or maxillofacial trauma, (3) significant mandibular asymmetry with a discrepancy in planned surgical setback of greater than 5 mm, (4) concomitant genioplasty at the time of SSO, (5) documented intraoperative iatrogenic IAN or mental nerve damage, and (6) lack of complete or clear medical records or CBCT imaging.

The primary outcome variable was neurosensory status (subjective VAS and objective MRCS scores). The primary predictor variable was postoperative changes in position of the IAC. Other covariates included age, sex, skeletal deformity type, timing of mandibular third molar removal, and the magnitude of surgical movements.

This study was approved by the University of Illinois Institutional Review Board (Protocol # 2019-0383).

Surgical procedure

Patients underwent bimaxillary surgical procedures, including a Le Fort I osteotomy and SSO, performed by one senior surgeon (MM). The SSO was performed using a modification of the Obwegeser–Dal Pont technique. A Lindemann bur was used under copious irrigation, followed by careful splitting of the mandible using a series of osteotomes. When the IAN was entrapped in the proximal segment after the split, it was gently released. However, when it was deemed that excessive trauma was anticipated to release the IAN, or the magnitude of the surgical movement was small (<3 mm), the IAN was left encased in the proximal segment. When the IAN was not visualized, no attempt was made to locate it since this might cause unnecessary iatrogenic IAN injury. When potential sources of IAN impingement were found on the medial surface of the proximal segment, they were removed prior to fixation. All fixation was performed using miniplate and monocortical screw osteosynthesis (KLS Martin, Jacksonville, FL, USA). Any observed trauma to the IAN was documented. All surgical procedures were planned using computer-assisted virtual surgical planning (VSP) with (3D Systems, Golden, CO, USA).

Clinical variables

The clinical variables, including subjective VAS and MRCS scores, age, sex, skeletal deformity type, magnitude of the surgical movements (linear distance between the proximal and distal segments on the VSP plan), and reported intraoperative IAN status, were obtained from the electronic medical records. Neurosensory testing was performed using a standardized form (MRCS classification for functional sensory recovery, Table 1[10]) by oral and maxillofacial surgery faculty and residents who routinely perform the neurosensory testing protocol. The MRCS classification uses common neurosensory tests, including two-point discrimination, brush-stroke directional discrimination, contact detection, and pain and temperature nociception to monitor functional neurosensory recovery[10]. A score of S3, S3+, or S4 on the MRCS is considered to indicate functional sensory recovery; such patients are classified as having no NSD or as being NSD-negative (NSD (−)). An MRCS score of S0, S1, S1+, S2, or S2+ is considered to indicate the presence of a NSD; such patients are classified as having a NSD or as being NSD-positive (NSD (+)).

Additionally, subject-perceived neurosensory recovery was recorded using a VAS. The patients marked their perceived neurosensory function on a 10-cm line

Table 1. Medical Research Council Scale for the recovery of sensitivity (adapted from Salomon et al.[10]).

Grade[a]	Recovery of sensitivity
S0	No recovery
S1	Recovery of deep cutaneous pain
S1+	Recovery of some superficial pain
S2	Return of some superficial pain and tactile sensation
S2+	S2 with over-response
S3[a]	Return of some superficial pain and tactile sensation without over-response; two-point discrimination >15 mm
S3+[a]	S3 with good stimulus localization; two-point discrimination = 7–15 mm
S4[a]	Complete recovery; S3+; two-point discrimination = 2–6 mm

[a] Grades S3, S3+, and S4 indicate functional sensory recovery and are considered neurosensory deficit-negative.

with five equidistant intervals classified as 1 to 5, such that ‘1’ is complete absence of sensation, ‘2’ is almost no sensation, ‘3’ is reduced sensation, ‘4’ is almost normal sensation, and ‘5’ is fully normal sensation.

In the interpretation of the objective and subject-perceived neurosensory scores, all patients were considered as NSD (+) when objective tests and/or subjective evaluation aligned with the following statements (Table 2): (1) if the subject-perceived neurosensory recovery VAS score was 1, 2, or 3, the result was considered NSD (+); (2) subjects with an MRSC grade of S0, S1, S1+, S2, or S2+ were considered NSD (+) even if the VAS score was 4 or 5.

CBCT measurements

CBCT data were acquired using an i-CAT CBCT machine (KaVo, Brea, CA, USA) with an extended field of view of 23 × 17 cm and a voxel size of 0.3 × 0.3 × 0.3 mm, at 18.54 mA and 120 kVp. Dolphin 3D software version 11.95 (Dolphin Imaging and Management Solutions, Chatsworth, CA, USA) was used for the registration and data analysis. The preoperative CBCT was oriented in a standardized fashion, with orbitale coincident with the coronal plane, and right porion and bilateral orbitale coincident on the same sagittal plane. Axially, the preoperative CBCT was aligned so that nasion and basion were on a plane perpendicular to the axial reference plane. Then, the preoperative and postoperative CBCT scans were superimposed on the unoperated symphyseal region using three-dimensional voxel-based superimposition to allow for accurate and precise measurement of the IAC position (Fig. 1).

Three specified points in the distal segment of the mandible were identified for the IAC measurements on both pre- and postoperative coronal and axial CBCT views: (I) the mid-mandibular first molar, (II) the mid-mandibular second molar, and (III) at 12 mm proximal to the distal surface of the mandibular second molar (Fig. 2).

Six measurements were performed at each point on the pre- and postoperative CBCT in the coronal view for each side, as follows (Fig. 3): (1) inferior distance (ID): the distance between the most inferior outer cortical point of the IAC and the inferior bony border of the mandible; (2) superior distance (SD): the distance between the most superior outer cortical point of the IAC and the superior bony border of the mandible; (3) medial dis-

Table 2. Interpretation of NSD-positive and NSD-negative outcomes.

	Group	
	NSD (+)	NSD (−)
Objective test results	MRSC grade of S0, S1, S1+, S2, or S2+	MRSC grade of S3, S3+, or S4
Subjective test results	VAS score 1, 2, or 3	VAS score 4 or 5

MRSC, Medical Research Council Scale; NSD, neurosensory deficit; NSD (+), with NSD; NSD (−), without NSD; VAS, visual analog scale.

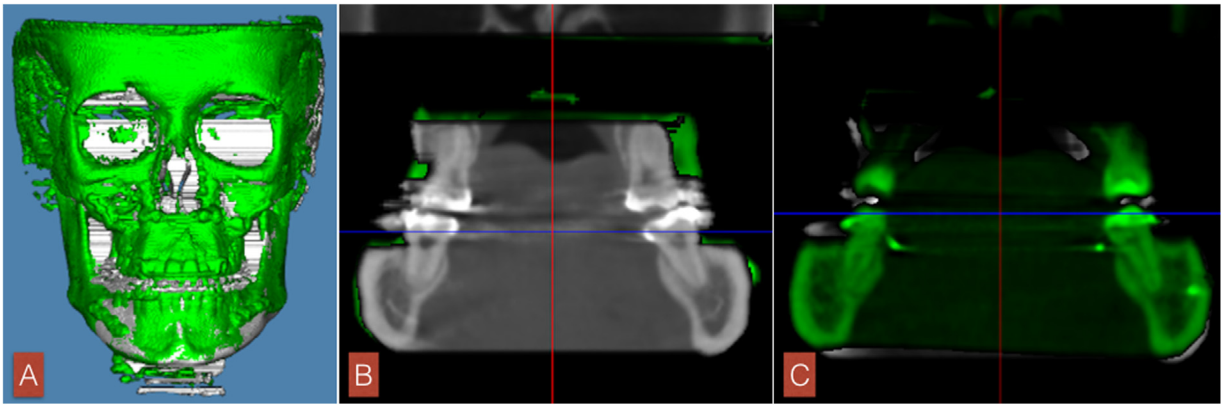

Fig. 1. (A) Superimposition of the preoperative and postoperative CBCT scans. (B) Preoperative image of the IAC on coronal view at a specified point. (C) Postoperative image of the IAC on coronal view at the same specified point. The gray color represents the preoperative image, whilst the green color represents the postoperative image. (CBCT, cone beam computed tomography; IAC, inferior alveolar canal.)

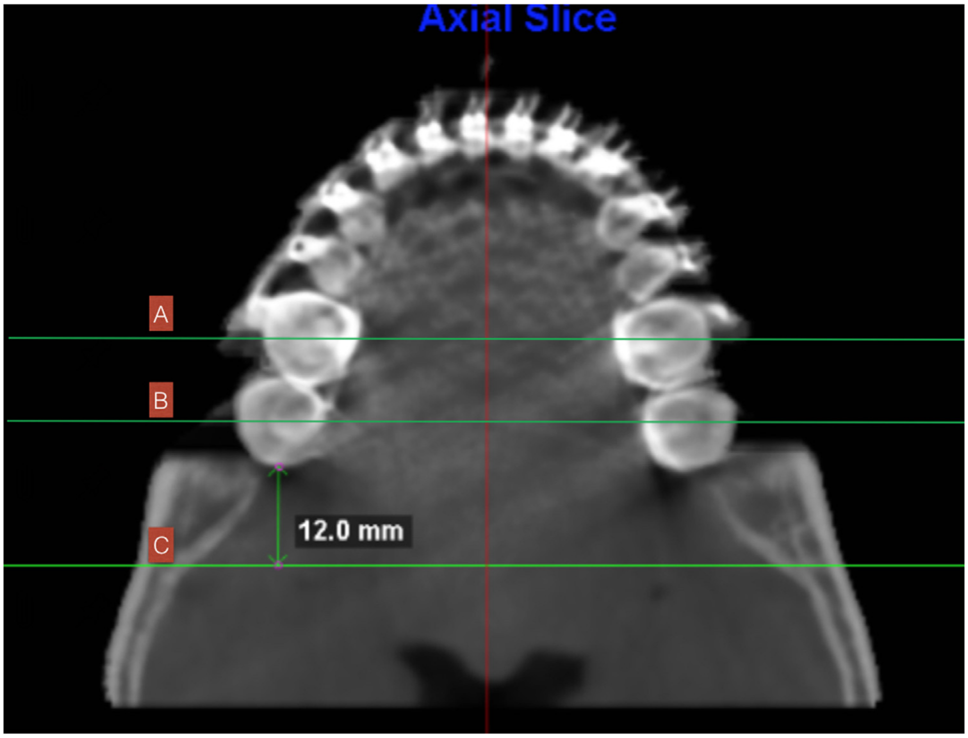

Fig. 2. (A) Position of the first specified point at the mid-mandibular first molar. (B) Position of the second specified point at the mid-mandibular second molar. (C) Position of the third specified point, 12 mm proximal to the distal surface of the mandibular second molar.

tance (MD): the distance between the most medial outer cortical point of the IAC and the medial bony border of the mandible; (4) lateral distance (**LD**): the distance between the most lateral outer cortical point of the IAC and the lateral bony border of the mandible; (5) canal height (CH): the distance between the most inferior and superior inner cortical bone points; (6) canal width (CW): the distance between the most medial and lateral inner cortical bone points.

In addition, the occurrence of iatrogenic screw intrusion into the IAC canal was evaluated on the postoperative CBCT and recorded.

Reliability

Two investigators, blinded to the sensory dysfunction, performed the CBCT measurements independently. To ensure reliability and validity, all distances were measured twice by the examiners at a 1-week interval. Intra-observer agreement was assessed with two-way mixed intraclass correlation using MedCalc 4.0 (MedCalc Software Ltd, Ostend, Belgium). Correlation between all pre- and postoperative dimensional measurements of the two examiners was also performed to ensure reliability and reproducibility.

Statistical analysis

The statistical analyses were performed using IBM SPSS Statistics for Windows, version 20.0 (IBM Corp., Armonk, NY, USA). All data were expressed as mean ± standard deviation values. Pre- and postoperative dimensions and the dimensional changes (Δ) of all landmarks in the NSD (+) and NSD (−) groups were examined for statistical significance based on the independent samples *t*-test. The χ^2 test or Fisher's exact test was used to assess the significance of differences among categorical variables, including sex, concomitant extraction of impacted third molars, and magnitude of jaw movement. A receiver operating characteristics (ROC) curve analysis was performed to determine the cut-off value for how much movement causes nerve injury. A *P*-value less than 0.05 was considered statistically significant.

Results

Twenty patients (11 female, nine male) with a mean age of 19.5 ± 4 years, ranging from 17 and 36 years, met the inclusion criteria (Table 3). Four subjects had class II skeletal deformities, while 16 had class III skeletal deformities. There were no cases of nerve transection or significant iatrogenic IAN injury documented, and IAN continuity was preserved in all 40 SSO sides (20 patients). None of the subjects had CBCT evidence of screw intrusion into the IAC on the postoperative CBCT. Due to the small number of patients who underwent mandibular advancement (4/20), the type of SSO could not be analyzed as a predictor variable.

The reliability analysis for the measurements performed by the two examiners on the CBCT scans revealed an intra-class correlation coefficient (ICC) of 0.944 (95% confidence interval (CI) 0.935–0.952), indicating satisfactory reliability for all of the measurements. These results allowed the average of the examiners' measurements to be used appropriately. Correlation between all pre- and postoperative measurements of the two examiners was also statistically significant ($r = 0.962$, 95% CI 0.973–0.948; $P < 0.001$).

NSD was diagnosed on 20 sides (50%) of all subjects. Altered sensation was found bilaterally in seven patients and unilaterally in six patients. There were 10 right-sided and 10 left-sided deficits. The altered sensation reported subjectively via the VAS was as follows: complete absence of sensation in 2.5% of the patients, almost no sensation in 20% of the patients, reduced sensation in 17.5% of

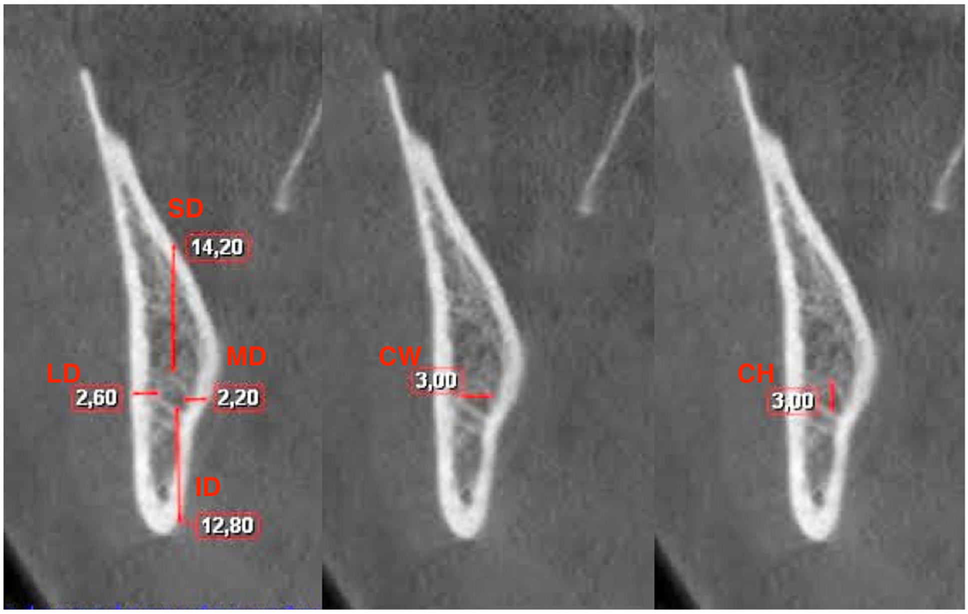

Fig. 3. Measurements of the inferior alveolar canal on coronal view: ID, inferior distance; SD, superior distance; LD, lateral distance; MD, medial distance; CH, canal height; CW, canal width.

the patients, almost normal sensation in 17.5% of the patients, and fully normal sensation in 42.5% of the patients. The patients who reported subjective evaluation VAS scores of 1, 2, or 3 had NSD (+) in all sides. There were no complaints of postoperative hyperesthesia or dysesthesia, or other signs or symptoms of neuropathic pain.

The relationships between NSD and demographic variables, concomitant extraction of impacted third molars, magnitude of jaw movement, and the differences between preoperative and postoperative measured distances (Δ) in the three designated regions of the IAC were evaluated on each side. The changes in all distances in the NSD (+) and NSD (−) groups are shown in Table 4. A significantly greater reduction in the postoperative lateral distance was seen on NSD (+) sides when

Table 3. Demographic variables, surgery type, third molar removal, neurosensory deficit status, and magnitude of the jaw movement (millimeters).

Patient	Sex	Age (years)	Surgery type	Third molar removal[a]	NSD		Magnitude of the jaw movement (mm)	
					Right	Left	Right	Left
1	F	19	Setback	Prior	–	–	5.135	3.385
2	M	21	Advancement	Prior	–	–	1.780	1.910
3	F	17	Setback	Prior	+	+	0.685	4.575
4	M	19	Setback	Same time	+	+	4.250	3.650
5	M	36	Advancement	Prior	+	–	5.170	2.400
6	F	18	Setback	Same time	+	+	2.640	3.425
7	F	17	Setback	Prior	+	+	5.600	3.680
8	F	17	Advancement	Same time	–	+	3.500	4.780
9	M	19	Advancement	Prior	–	–	3.230	3.140
10	F	18	Setback	Prior	+	–	0.970	2.105
11	F	17	Setback	Prior	+	–	5.116	4.213
12	M	19	Setback	Prior	–	–	3.985	0.955
13	F	17	Setback	Same time	–	+	1.410	1.055
14	M	18	Setback	Same time	+	+	7.565	7.655
15	M	18	Setback	Same time	–	+	4.330	6.780
16	M	20	Setback	Prior	–	–	2.500	2.460
17	F	18	Setback	Prior	+	+	3.285	1.155
18	M	25	Setback	Prior	–	–	1.740	2.810
19	F	19	Setback	Same time	–	–	2.600	5.615
20	F	18	Setback	Prior	+	+	5.740	8.815

F, female; M, male; NSD, neurosensory deficit.

[a] Prior to or at the same time as the surgery.

Table 4. Measurements of six different distances performed at three stable points on CBCT, changes in the measurements from preoperative to postoperative for both sides, and the change in measurements according to the presence or not of a NSD; mean ± standard deviation values. (For change (**Δ**), a negative value indicates an increase and a positive value indicates a decrease in the postoperative measurements).

Distance	Point	Measurements						Overall changes in measurements (both sides)	Changes in measurements according to the presence of NSD		
		RIGHT SIDE			LEFT SIDE						
		Preoperative (mm) (n:20)	Postoperative (mm) (n:20)	p	Preoperative (mm) (n:20)	Postoperative (mm) (n:20)	p	Δ (mm) (n:40)	Δ (mm) NSD (-) (n:20)	Δ (mm) NSD (+) (n:20)	P-value
Lateral Distance	I	6.80 ± 1.50	5.80 ± 1.80	0.001	6.50 ± 1.90	6.20 ± 1.60	0.0001	0.60 ± 1.20	0.46 ± 1.50	0.74 ± 0.96	NS
	II	6.95 ± 1.64	6.10 ± 1.50	0.003	6.90 ± 1.60	6 ± 1.50	0.002	0.90 ± 1.30	0.38 ± 0.96	1.40 ± 1.40	0.01
	III	3.90 ± 1.06	4.50 ± 1.20	0.03	4.10 ± 0.90	4.70 ± 0.60	NS	−0.60 ± 1	−0.74 ± 0.80	−0.55 ± 1.20	NS
Medial Distance	I	3.44 ± 1.30	3.80 ± 1.70	0.0001	3.30 ± 1.10	3.80 ± 1.70	0.0001	−0.42 ± 1.04	−0.22 ± 0.90	−0.60 ± 1.10	NS
	II	2.60 ± 0.70	3.80 ± 1.50	0.01	2.80 ± 1.40	3.30 ± 1.20	NS	−0.82 ± 1.60	−0.50 ± 1.80	−1.10 ± 1.30	NS
	III	3.30 ± 1.70	4.30 ± 2.50	0.04	2.90 ± 1.40	4 ± 1.90	NS	−1 ± 2.10	−0.90 ± 2	−1.10 ± 2.20	NS
Superior Distance	I	17 ±2.90	18 ± 3.20	0.001	17.90 ± 3.40	17.80 ± 3.80	0.0001	−0.40 ± 2.10	−0.83 ± 2.40	−0.02 ± 1.60	NS
	II	15.20 ± 3.70	14.20 ± 4.70	0.0001	15.50 ± 3.30	15 ± 3.90	0.0001	0.70 ± 2.60	0.83 ± 3	0.60 ± 2.20	NS
	III	17.70 ± 4	17.80 ± 6	NS	17.40 ± 4.20	16 ± 5.50	NS	0.60 ± 6	0.20 ± 5.80	1.10 ± 6	NS
Inferior distance	I	8.40 ± 3	7.70 ± 2.20	0.0001	7.30 ± 1.90	7.50 ± 1.60	0.0001	0.25 ± 1.50	0.44 ± 2.10	0.07 ± 0.80	NS
	II	6.70 ± 1.40	7.10 ± 2.20	NS	6.30 ± 1.40	6.70 ± 2.44	NS	−0.45 ± 2.50	−0.40 ± 0.80	−0.50 ± 3.30	NS
	III	10.20 ± 3	11.60 ± 4.50	0.003	10 ± 3.10	12.40 ± 4.80	0.003	−1.80 ± 3.60	−0.70 ± 3	−3 ± 3.80	0.04
Canal Width	I	2.50 ± 0.50	2.60 ± 0.60	NS	2.50 ± 0.50	2.60 ± 0.50	NS	−0.06 ± 0.60	−0.02 ± 0.60	−0.12 ± 0.60	NS
	II	2.40 ± 0.50	2.60 ± 0.60	NS	2.60 ± 0.90	2.70 ± 0.50	NS	−0.20 ± 0.80	−0.20 ± 0.60	−0.20 ± 1	NS
	III	2.50 ± 0.50	2.50 ± 0.50	NS	2.50 ± 0.50	2.60 ± 0.70	NS	−0.06 ± 0.60	−0.09 ± 0.60	−0.02 ± 0.70	NS
Canal Height	I	3.20 ± 0.90	3 ± 0.90	0.04	2.70 ± 0.50	3 ± 0.80	NS	−0.01 ± 0.90	0.02 ± 1.10	−0.03 ± 0.60	NS

Table 5. Comparison of mean mandibular movements in the NSD-positive and NSD-negative groups and the mean magnitude of mandibular movement for setback and advancement cases.

	NSD (+) ($n = 20$)	NSD (−) ($n = 20$)	*P*-value[a]	Setback cases ($n = 16$)	Advancement cases ($n = 4$)
Mean value of mandibular movement (mm)	4.31 ± 2.30	2.95 ± 1.22	0.02	3.74 ± 2.11	3.23 ± 1.24

NSD, neurosensory deficit.

[a] *P*-value for the comparison NSD (+) vs NSD (−).

compared with NSD (−) sides (*P*=0.01). A significant increase in the inferior distance at this same location was seen on NSD (+) sides when compared with NSD (−) sides (*P* = 0.04).

Pre- and postoperative measured distances on the right and left sides of the mandible and the changes in distance in the three designated regions are reported in Table 4. A significant reduction in the postoperative canal height was seen at the first and second specified points of the right side (P= 0.04 and P = 0.02, respectively), whereas a great increase in the postoperative canal height was seen at the third specified point of the left side (P = 0.0001) in the mandible. The changes in canal height and width from pre- to postoperative were minimal, and there was no statistically significant difference in the change in height or width between the NSD (+) and NSD (−) groups(Table 4).

There was no statistically significant difference in NSD according to sex (*P* = 0.056). Concomitant third molar extractions were performed on 14 sides in seven patients. Neurosensory impairment was found in nine sides (64.3%) of these patients, while neurosensory impairment was present in 11 sides of 13 patients (42.3%) who underwent third molar extractions prior to SSO (*P* = 0.18).

The mean magnitude of mandibular movement for all procedures was 3.63 ± 1.95 mm (range 0.97–8.8 mm). Setback cases had a mean magnitude of 3.74 ± 2.11 mm, whereas advancement cases had a mean magnitude of 3.23 ± 1.24 mm. The magnitude of the mandibular movement was found to be greater in the NSD (+) group (with a mean movement of 4.31 ± 2.30 mm) when compared to the NSD (−) group (with a mean movement 2.95 ± 1.22 mm), and this was statistically significant (*P* = 0.02) (Table 5). On ROC curve analysis, a cut-off value of 3.2 mm had 75% (95% CI 51–91%) sensitivity and 65% (95% CI 41–85%) specificity to predict positive nerve injury cases (Fig. 4).

Discussion

Sensory deficit following SSO is often temporary and IAN function generally recovers within 6–12 months, depending upon the extent of the damage[4]. Permanent neurosensory dysfunction lasting more than 1 year is likely to indicate an axonotmesis injury under Seddon's classification, which includes damage to axons and endoneurium, and less likely to the perineurium and the epineurium, although a witnessed or unwitnessed neurotmesis injury or transection of the nerve during surgery could also be responsible for prolonged or permanent paresthesia[11,12]. However, in this study, since none

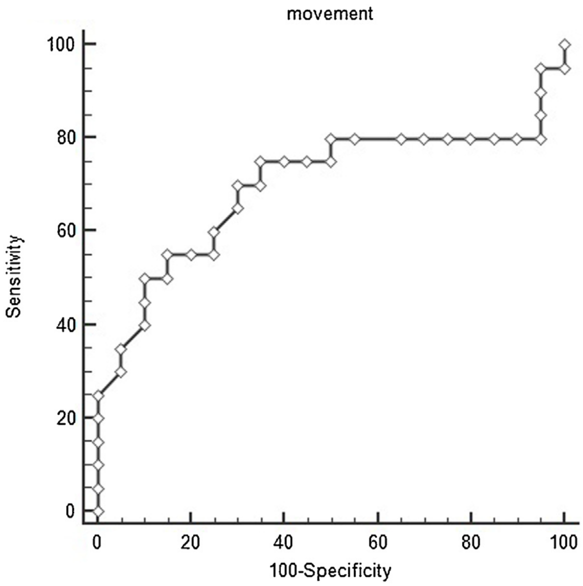

Fig. 4. Receiver operating characteristics curve generated for the magnitude of the movements, showing the ability to predict positive nerve injury cases.

of the nerves was reported to be transected during surgery, the prolonged NSD seen in half of the study subjects is likely to have been caused by crush injury, compression, or nerve traction during splitting or repositioning of the jaw.

Chronic nerve compression alters the normal anatomical and functional integrity of the nerve[13]. From a biological standpoint, extraneural fibrosis, one of the signs of chronic physical stress by nerve compression or tension, is considered to be a major contributing factor to restricting optimal functional recovery[14]. Even though scar formation occurs in the repair process following injury, excessive perineural fibrosis occurring in response to nerve injury impedes normal axonal regeneration[14,15], thus ultimately impairing the neural signal conduction to sensory end targets[12,14]. Moreover, it is known that during nerve regeneration, a period of growth of both axonal diameter and myelin sheath thickness is needed to ensure full functional neurosensory recovery[13].

In all CBCT measurements in the present study, a slight enlargement of the postoperative canal height and width was observed, similar to previously published studies[9,16]. Furthermore, chronic nerve compression, which may result in extrinsic scarring, could be a reason for the prolonged sensory deficit in the NSD (+) group. However, no significant results or conclusions in support of NSD positivity were derived for the canal height and width and thus, these observations weakly suggest that chronic nerve compression may be involved in the etiopathogenesis of persistent NSD. Based on the present results and previous ultrastructural studies, further investigation with an increased number of subjects is needed to better understand the postoperative dimensional changes of the IAC and their relationships with NSD.

Although it is still unclear which factors affect the incidence of lower lip and chin hypoesthesia after SSO, it is very important to know the preoperative location of the IAC in order to avoid direct damage to the nerve. Studies have shown that the prevalence of NSD increases on sides where the IAC comes into direct contact with, or is in close proximity to, the lateral cortical plate of the mandible[4,6,17,18]. It has been suggested that if the marrow space lateral to the canal is $\geq$1.0 mm, the incidence of IAN injury is low. Otherwise, performing the vertical osteotomy and splitting the mandible without contact with, and potential injury to, the IAN can be difficult when the distance is $<$1.0 mm. It has also been demonstrated that if the width of the marrow space between the IAC and lateral cortex is $<$0.8 mm, NSD is significantly more likely to be observed 1 year following the SSO surgery[6,18].

With regard to this issue, in the present study the average preoperative distances between the lateral cortical wall and the IAC at the first and second measured locations were $>$6 mm on both sides. Accordingly, the preoperative values which were higher for the prevention of osteotomy injury, seem to have the negligible effect on the prolonged neurosensory impairment.

The only significant postoperative alteration seen in the IAC in the NSD (+) group was a reduction in the lateral distance at the osteotomy site. The data from this study may indicate that intra-bony interferences between the lateral cortex and the neurovascular bundle were eliminated before repositioning of the fragments, and at this stage, the IAN may have been damaged by increased manipulation.

Regardless of the direction of the mandibular movement, it is advocated that increased manipulation of the IAN by a greater degree of fragment movement is a contributing factor to the persistence of NSD[19]. This is particularly important in cases of asymmetry correction where interferences may compress the IAN, or where the surgical reduction of those bony interferences may cause iatrogenic IAN injury. The findings of the present study support the idea that a greater magnitude of mandibular movement is a risk factor for persistent IAN neurosensory impairment in patients undergoing SSO.

It has been proposed that the risk of NSD is greater in mandibular advancement procedures, due to the increase in elongation of the IAN, as opposed to mandibular setbacks where there might be more redundancy of the nerve, and that a stretch-type injury would occur less commonly[18]. Some authors have reported that the incidence of NSD at 1 year after SSO is similar in both mandibular advancement and setback surgery and have claimed that stretching of the IAN likely does not affect the neurosensory recovery during the 1-year follow-up period[19], especially if it does not exceed the ratio of pathological elongation (approximately 30%)[20]. In the present study, even though maximum jaw movement in advancement cases was modest and did not exceed this threshold at 5.17 mm, persistent NSD was diagnosed in two subjects. Although a correlation analysis could not be performed for this parameter owing to the small sample size (4/20 patients), as reported previously[19,21], this alteration may be explained by medial dissection and retraction leading to increased postoperative swelling and manipulation of the neurovascular bundle, which were not investigated in the present study. These possibilities should be taken into account while instrumenting the medial side of the mandibular ramus, regardless of the direction or magnitude of the mandibular surgery. It must be acknowledged that there are many heterogeneities in how surgical movements are measured. In this study, measurements were made on the overlap or gap between the proximal and distal segments on the VSP report. This may not have matched with the actual measurement. Also, such measurements between the proximal and distal segments may vary significantly from other ways of measuring sagittal movements, such as displacement of different landmarks along a reference plane.

The distance between the mental foramen and mandibular foramen is decreased following setback procedures, so in these cases, IAN hypoesthesia may occur by compression of the nerve trunk at the mandibular foramen due to posterior shifting of the distal mandibular segment. As compared to advancement surgery, the IAN is more vulnerable to damage at the entrance into the mandible than at the exit point at the mental foramen[22]. It has been reported that full recovery occurred in 93.5% of setback cases compared to 100% of advancement cases at 6 months of follow-up[19]. The results of the present study are consistent with this finding, but larger and equivalent sample size studies are needed to compare the neurosensory recovery between advancement and setback procedures.

Objective and/or subjective methods are frequently used to predict NSD and monitor the recovery process after an SSO procedure. Some authors consider that subjective evaluations are more critical due to the fact that patient-reported outcomes and subjective recovery are imperative in selecting the appropriate treatment modality and providing appropriate informed consent[23]. On the other hand, some authors claim that both objective and subjective tests are required to adequately assess the incidence of postoperative IAN deficit[24]. In the present study, both subjective and objective tests were applied to the study sample, and a reasonable correlation was identified between the two types of outcome evaluation.

The occurrence of neurosensory impairment is thought to be influenced by other factors, including patient age and sex, concomitant third molar removal, and other IAC-related factors such as the presence of a bifid or trifid IAC, or close proximity to the buccal cortex and inferior border of the mandible[25]. As reported previously, patients of advanced age ($\geq$40 years) and female patients are at higher risk of neurosensory impairment following SSO, as well as the development of neuropathic pain following SSO[6,18,25,26]. It has been recommended that mandibular third molars be removed at least 6–12 months prior to surgery to prevent intraoperative complications, such as bad splits and IAN injury[27]. In contrast, some researchers have found that the removal of third molars prior to SSO can lead to an increased risk of NSD[28]. In this study, sex, age, and concomitant third molar extraction did not have an influence on the neurosensory outcomes, similar to other reports in the literature[11,25,27,29].

Accurate assessment of the morphological and structural changes of the IAN in patients with NSD is crucial in order to clarify the possible etiology of the injury and potential relevant correlations. Despite providing a valuable resource in the surgical verification after SSO[30], making a precise measurement of the IAN on a CBCT is challenging. Thus, quantitative measurement of the volume of the IAC with a highly sensitive technique may be more beneficial to determine the likelihood of injury to an area along the course of the IAN. These changes in the IAC may lead to better understanding of the reasons for prolonged NSD after SSO.

In conclusion, this study demonstrates that changes in the location of the IAC following SSO procedures correlate with postoperative NSD, and that these changes may provide additional information regarding the complex etiology of persistent paresthesia in some patients. The results showed that a moderate magnitude of mandibular movement via SSO, regardless of the direction, may lead to prolonged NSD. Furthermore, minimizing IAN manipulation and possible iatrogenic injury at any stage of the surgery may certainly be beneficial in reducing the incidence of NSD after mandibular SSO procedures.

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Competing interests

Dr Miloro is a Consultant for AxoGen, Inc., Alachua, FL, USA.

Ethical approval

This retrospective study was performed in accordance with guidelines outlined in the 1964 Declaration of Helsinki and was approved by the University of Illinois Institutional Review Board (Protocol # 2019-0383).

Patient consent

Not required.

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