Inferior Alveolar Nerve Impairment After Mandibular Sagittal Split Osteotomy: An Analysis of Spontaneous Recovery Patterns Observed in 60 Patients

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Sensory impairment after bilateral sagittal split osteotomy (BSSO) due to inferior alveolar nerve (IAN) lesions may be either temporary or permanent and either complete or partial. The aim of this prospective study is to evaluate, by means of objective sensory testing, IAN sensory disturbances development in patients who underwent BSSO.

IAN sensory disturbances development at the first week, fourth week, sixth month, and twelfth month of follow-up review in a group of 60 patients who underwent BSSO from January 1, 1998, to July 31, 1999, at the Maxillofacial Surgery Department of the "La Sapienza" University of Rome. The 60 patients were examined in the presurgical period; the IAN functionality regarding thermal sensibility, nociception, and two-point discrimination, was assessed at follow-up in 120 sides. In our study the highest rate of spontaneous recovery of the entire IAN functionality was observed at the sixth month. This finding witnesses how neuropraxia and axonotmesis give a spontaneous recovery that most frequently occurs within 6 months from surgery, independently from age and sex of the patient. The persistence of anesthesia over 12 months could be a sign of neurotmesis.

Key Words: Anesthesia, paraesthesia, hypoaesthesia, BSSO, IAN

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Introduction

Bilateral sagittal split osteotomy (BSSO) is a common surgical procedure performed in orthognatic surgery to obtain mandibular advancement or setback. Because of the vicinity of the inferior alveolar nerve, (hereafter abbreviated IAN) to the surgical site, sensory impairments in the IAN distribution may frequently follow BSSO. IAN sensitive component originates from the postero-medial branch of the Trigeminal Nerve and provides the sensitive innervation of oral and perioral regions.[1–2] Sensory impairment following BSSO due to IAN lesions may be either temporary or permanent and either complete or partial. The evolution of post-surgical impairments could depend on several variables such as the age of the patient, the direction or the amount of the mandibular movement, the surgical technique, the surgical manipulation, eventual direct injury to the nerve bundle due to surgery, as well as the type of fixation used.

Recovery patterns in IAN impairments should be evaluated by means of objective standard sensory testing modalities. Objective assessments of nerve functionality at follow-up review, together with the analysis of the surgical variables for each case, could lead to a better comprehension of the role played by surgical factors in determining a temporary/permanent, complete/partial IAN sensory deficit.

The aim of this prospective study is to evaluate, by means of objective sensory testing, IAN sensory disturbances development at the first week, fourth week, sixth month, and twelfth month of follow-up review in a group of 60 patients who underwent BSSO from January 1, 1998, to July 31, 1999, at the Maxillofacial Surgery Department of the "La Sapienza" University of Rome. To assess any relationship

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with IAN recovery pattern, the patients' age and sex and the surgical variables have been collected for each patient.

Materials and Methods

Sixty consecutive patients requiring orthognatic surgery consisting of BSSO alone or in combination with Le Fort I osteotomy were enrolled in our prospective study. The patients were 23 males and 37 females and their ages ranged from 17 to 40 years (mean age: 25.8). The 60 patients were examined in the presurgical period and the IAN functionality regarding thermal sensibility, nociception, and two-point discrimination was assessed at follow-up review in 120 sides. Moreover, the surgeons were asked to describe the grade surgical manipulation to the IAN, as suggested in international literature.[3]

To analyze the correlation of each of several variables with the IAN recovery pattern, data about our patients (the age during the surgical period and the sex) and about the surgical treatment (the direction and the amount of repositioning, the surgical technique, the kind of fixation used, and the post-surgical infections), were recorded for each case. Progenic syndrome was diagnosed in 35 patients and Prognatic syndrome was diagnosed in the remaining 25. The presurgical assessment revealed that the whole of the 60 patient group, who were healthy and neurological disease free, exhibited no sensory disturbances in the IAN region. To obtain an adequate dental alignment, an orthodontic presurgical treatment was performed in all the 60 patients aforementioned.

The surgical treatment consisted in BSSO in 5 cases and in a combination of both BSSO and Le Fort I osteotomy in the remaining 55. In 35 cases mandibular setback was performed while 25 cases had mandibular advancement. The mandibular setback ranged from 2 to 5 millimeters while the mandibular advancement ranged from 2 to 7 millimeters (2 cases); 20 cases underwent minor surgical procedures also during orthognatic surgery: 10 cases had also wisdom teeth extraction, 4 cases had genioplasty, 4 case septoplastic, 1 case had both septoplastic and wisdom teeth extraction, and 1 case had genioplasty and wisdom teeth extraction.

As all the 60 patients of our study had bilateral sagittal split of the mandible, 120 nerves were involved in the surgical repositioning of the mandible. Bilateral sagittal split osteotomies (BSSO) were performed in all the 60 cases of our study following general anesthesia induction via nasal intubation and infiltration with 2% lidocaine with 1:100,000 epinephrine into the surgical sites. To visualize the mandibular bone, reflection and retraction of the periosteum were accomplished following vestibular incision by means of curved retractors. The bone cuts were excavated on the medial aspect of the ramus just few millimeters above the entrance of the IAN into the mandibular canal, on the lateral aspect of the ramus few millimeters anteriorly to the mandibular angle, and on the crestal cortical bone connecting the two aforementioned osteotomies. All the bone cuts were obtained with medium and short burs. The splitting procedures were performed using a hammer and fine chisels first, then medium chisels. After the splitting procedure was completed, the integrity and the position of the IAN were assessed in all patients by visual check. After mobilization of the distal fragment, maxillomandibular fixation was performed and the correct occlusion was set. In all the aforementioned patients, the BSSOs were fixed with three bicortical screws at the angle of each side and the maxillomandibular fixations were removed. To avoid injuring the nerve bundle, the screws were positioned under visual check of the IAN position.

Eventual nerve damage was classified by surgeons into the following degrees, as shown in international literature[3]: Class I: Nerve encased within the bony canal of the mandible not visualized in the distal fragment; Class II: Nerve visualized and not injured or manipulated; Class III: Manipulated nerve; Class IV: Transected nerve.

Clinical neurosensory testing was performed presurgically and in the first and fourth week and in the sixth and twelfth month of follow-up review on both sides of patients. The testing methods included two-point discrimination, nociception, and temperature sensitivity in all 60 patients.

Two-point-discrimination was tested by a gauge with blunted points to avoid causing any pain. The gauge was applied on the chin's skin, beginning at the right side, with equal pressure on both points. Two-point discrimination was assessed for each patient until the patient could no longer discriminate correctly the two points. This distance was then recorded.

The same procedure was then repeated on the left side. A distance of 5 mm or less was judged as a value for a positive sensory response.

The 60 patients of our study were tested for nociceptive sensibilities by means of a sharp tool stimulating the chin's skin. The patients could choose among three grades of response: numb, dull, or light pain. Good nociceptive sensibility was judged by having adequate responses to a sharp stimulation.

Thermal sensibility was tested using ice cubes

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and warmed blunted metal tools. At the beginning, the ice cubes were placed on the lower lip of the patients, first on the right side and then on the left side. All patients were asked to report their sensations to cold. A few minutes later, the blunted metal tools were warmed up to a temperature of 38° centigrade. Then the warmed metal tools were used in the same way, first on the right side and then on the left one. The patients were asked to report their sensations to warm. The patients could express the grade of thermal sensibility about cold and warm using the following values: high grade, medium grade, low grade, no sensibility on lower lip.

In our analysis, hypo-aesthesia was given by an imperfect and incomplete response observed during testing modalities; paraesthesia was defined as a sensibility qualitative painless alteration, with numbness, itching, or tilting sensations. Anesthesia was determined by a complete loss of sensibility as recorded by testing. Full range of IAN functionality was judged by having adequate responses at the three tests.

RESULTS

One hundred eighteen of 120 nerves were assigned by the surgeons to class I and II; the remaining two nerves were classified in group III, as surgical manipulation was required to debride the nerve bundle from the lateral aspect of the ramus and to drive it gently toward the medial aspect of mandibular bony canal (patients #27 left side and #32 right side) (Table 1).

In all the 120 sides, the surgical technique was the same and consistent with what has been previously described; the type of fixation used consisted in three bicortical screws positioned at each mandibular angle, as previously mentioned.

Post-surgical infections were observed during the first days since surgical treatment in three patients (patients # 8, # 32, and # 49). Complete resolution of infections was obtained within 7 days from surgical treatment in all the three patients and it was given by careful medications and by an adequate antibiotic therapy. No other complications occurred.

At the first week of follow-up review (Table 1 and Fig 1), a full range of sensibility in the IAN distribution concerning tactile two-point discrimination, thermal sensibility, and nociception sensibility was observed in 21 sides, corresponding to 14 patients (17.5% of 120 sides).

Hypoaesthesia was reported in 35 sides (29% of 120 sides), on both sides in 10 patients and on one side in 15 patients (Table 1 and Fig 1). Hypoaesthesia involved tactile discrimination, temperature sensibility, and nociception in all the 35 sides. Paraesthesia was observed in 18 sides (15% of 120 sides); that is corresponding to 9 patients; as shown in Table 1 and Figure 1. At the first week of follow-up review, anesthesia (Table 1 and Figure 1) was found in 46 sides (38.3% of 120 sides), corresponding to 27 patients: 19 patients had anesthesia on both sides and 8 patients had it on one side only.

At the fourth week of follow-up review (Table 1 and Fig 1), a full range of IAN sensibility was found in 35 sides (29.1% of 120 sides), that is on both sides in seven patients. Hypoaesthesia was reported in 71 sides (59.1% of 120 sides), corresponding to 44 patients. In 29 sides it represented a residual impairment from previous hypoesthesia and in 42 sides it was an evolution of the previous condition of anesthesia. The thermal, tactile, and nociceptive sensibilities were diminished in all the hypoaesthetic sides, and the tactile discrimination resulted in the most seriously impaired. At the fourth week of follow-up review, the persistence of paresthesia was observed in 10 sides (8.3% of 120 sides), as shown in Table 1 and in Fig 1, corresponding to 9 patients. Anesthesia was still reported in four sides, corresponding to four patients (3.33% of 120 sides).

At the sixth month of follow-up review (Table 1 and Fig 1), a full range of sensibility was observed in 100 sides (83.3% of 120 sides), corresponding to 58 patients. Hypoaesthesia was observed in 14 sides (12.5% of 120 sides), corresponding to 13 patients. In three sides, hypoaesthesia represented the evolution of an anesthesia observed at the fourth week of follow-up review. Paraesthesia was observed at the sixth month of follow-up in five sides (4.16% of 120 sides) and in all the five sides the qualitative alteration of sensibility was less serious and harmful than before. The persistence of anesthesia was observed in one side at the sixth month follow-up review (Table 1 and Figure 1).

At the twelfth month of follow-up, the full range of sensibility in the IAN distribution was observed in 114 sides (95% of 120 sides), and 56 patients had a total and complete spontaneous IAN recovery on both sides with a total disappearance of tactile, thermal, or nociceptive impairments (Table 1 and Fig 1).

Hypoaesthesia was still present in four patients on six sides (5% of 120 sides). Two-point discrimination was still lightly involved in all the four sides, but thermal and nociceptive sensibilities had a correct and complete functionality. Paraesthesia and anesthesia were not present at the twelfth month of follow-up review on any of the 120 sides.

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Table 1. Evolution of IAN Impairments and its Relation to Entity and Direction of Surgical Repositioning, Sex, and Age of Patients

Pt	*Age*	*Sex*	*Mand Movement*	*1st week—sides*	*4th week—sides*	*6th month—sides*	*12th month—sides*
1	23	F	Adv 3 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
2	22	M	SB 3 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
3	34	F	Adv 3 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
4	19	F	SB 5 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
5	28	F	SB 4 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
6	21	M	Adv 3 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
7	32	F	Adv 3 mm	Full range (2)	Full range (2)	Full range (2)	Full range (2)
8	18	F	Adv 2 mm	Hypoesthesia (R)	Hypoesthesia (R)	Full range (2)	Full range (2)
9	20	M	SB 4 mm	Hypoesthesia (R)	Hypoesthesia (R)	Full range (2)	Full range (2)
10	22	M	SB 3 mm	Hypoesthesia (R)	Hypoesthesia (R)	Full range (2)	Full range (2)
11	40	F	SB 3 mm	Hypoesthesia (L)	Hypoesthesia (L)	Full range (2)	Full range (2)
12	21	F	SB 4 mm	Hypoesthesia (L)	Hypoesthesia (L)	Full range (2)	Full range (2)
13	31	F	SB 5 mm	Hypoesthesia (R)	Hypoesthesia (R)	Full range (2)	Full range (2)
14	25	M	SB 5 mm	Hypoesthesia (R)	Hypoesthesia (R)	Full range (2)	Full range (2)
15	23	F	Adv 4 mm	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)	Full range (2)
16	34	F	Adv 4 mm	Hypoesthesia (2)	Hypoesthesia (L)	Full range (2)	Full range (2)
17	17	M	SB 3 mm	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)	Full range (2)
18	19	F	Adv 3 mm	Hypoesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
19	21	F	SB 3 mm	Hypoesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
20	22	M	Adv 3 mm	Hypoesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
21	23	F	SB 3 mm	Hypoesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
22	21	F	SB 2 mm	Hypoesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
23	19	F	SB 3 mm	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)	Full range (2)
24	18	M	SB 3 mm	Hypoesthesia (2)	Hypoesthesia (L)	Full range (2)	Full range (2)
25	23	F	Adv 3 mm	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (R)	Full range (2)	Full range (2)
26	22	F	Adv 2 mm	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (2)	Full range (2)	Full range (2)
27	31	F	SB 3 mm	Anesthesia (L) + Hypoesthesia (R)	Hypoesthesia (2)	Hypoesthesia (L)	Hypoesthesia (L)
28	30	M	SB 3 mm	Anesthesia (L) + Hypoesthesia (R)	Hypoesthesia (2)	Hypoesthesia (L)	Full range (2)
29	20	F	SB 4 mm	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)
30	23	M	SB 2 mm	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)
31	22	F	SB 4 mm	Anesthesia (L) + Hypoesthesia (R)	Hypoesthesia (2)	Hypoesthesia (L)	Full range (2)
32	21	M	Adv 3 mm	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (2)	Hypoesthesia (R)	Hypoesthesia (R)
33	25	F	Adv 3 mm	Paresthesia (2)	Paresthesia (2)	Paresthesia (R)	Full range (2)
34	26	F	SB 4 mm	Paresthesia (2)	Paresthesia (R)	Full range (2)	Full range (2)
35	28	F	SB 4 mm	Paresthesia (2)	Paresthesia (L)	Full range (2)	Full range (2)
36	20	F	Adv 2 mm	Paresthesia (2)	Paresthesia (R)	Full range (2)	Full range (2)
37	35	M	SB 5 mm	Paresthesia (2)	Paresthesia (R)	Full range (2)	Full range (2)
38	29	F	Adv 3 mm	Paresthesia (2)	Paresthesia (L)	Paresthesia (L)	Full range (2)
39	19	F	SB 5 mm	Paresthesia (2)	Paresthesia (L)	Paresthesia (L)	Full range (2)
40	28	M	SB 4 mm	Paresthesia (2)	Paresthesia (R)	Paresthesia (R)	Full range (2)
41	26	F	SB 5 mm	Paresthesia (2)	Paresthesia (L)	Paresthesia (L)	Full range (2)
42	28	F	Adv 3 mm	Anesthesia (2)	Hypoesthesia (2)	Hypoesthesia (L)	Full range (2)
43	17	M	SB 5 mm	Anesthesia (2)	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)
44	19	F	Adv 2 mm	Anesthesia (2)	Hypoesthesia (2)	Hypoesthesia (R)	Full range (2)
45	21	F	SB 4 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)

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Table 1. Continued

Pt	Age	Sex	Mand Movement	1st week—sides	4th week—sides	6th month—sides	12th month—sides
46	22	M	SB 4 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
47	77	M	SB 4 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
48	26	M	SB 4 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
49	25	M	Adv 3 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
50	23	F	Adv 3 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
51	20	M	SB 2 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
52	21	F	Adv 4 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
53	21	M	SB 5 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
54	21	F	SB 5 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
55	25	M	Adv 3 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
56	26	F	Adv 3 mm	Anesthesia (2)	Hypoesthesia (2)	Full range (2)	Full range (2)
57	24	M	Adv 4 mm	Anesthesia (2)	Hypoesthesia (L) + Hypoesthesia (R)	Hypoesthesia (L)	Full range (2)
58	22	M	Adv 7 mm	Anesthesia (2)	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (2)	Hypoesthesia (2)
59	31	F	SB 5 mm	Anesthesia (2)	Anesthesia (R) + Hypoesthesia (L)	Hypoesthesia (R)	Full range (2)
60	19	F	Adv 4 mm	Anesthesia (2)	Anesthesia (L) + Hypoesthesia (R)	Anesthesia (L) + Hypoesthesia (R)	Hypoesthesia (2)

M = male, F = female; Adv = Mandibular Advancement; SB = Mandibular setback; (2) = Both sides; (R) = Right side; (L) = Left side.

Discussion and Conclusion

It is known that the IAN can be damaged after BSSO. The nerve fibers can be injured directly by surgical manipulation, osteotomy procedures, or mandibular bicortical screws positioning, as previously reported.[3–5] According to Nagakawa e Ueki[6] postsurgical neurosensory impairments can be indirectly caused by hypossia and edema due to the compression of the nerve bundle within the mandibular canal.

Direct or indirect injuries can cause neuroapraxia (defined as a temporary dysfunction, with a full spontaneous recovery), assonotmesis (defined as assonal degeneration with a slow and uncertain spontaneous recovery), and neurotmesis (defined as a total interruption of nerve fibers with absence of spontaneous recovery).[7,8,9]

As many studies suggest, the spontaneous recovery of the full sensibility could be influenced by the age or the sex of patient[10,11], and by numerous surgical parameters, such as the entity of the mandibular repositioning [3], movement direction [3], nerve manipulations, or direct surgical injuries [3–6, 10–15], the kind of fixation used [12] as well as the surgical technique.[3]

All BSSOs were performed with the same surgical technique and were carried out on all the 120 sides, through bicortical screws positioning. This makes not possible efforts to establish the correlation between different fixing techniques and sensory impairments.

In accordance with what Jacks and Zuniga, Nagakawa, Ueki et al, Fridrich and Holton, referred[3–5,10], the age of our patients at the time of surgery did not associate with slower or faster spontaneous recovery in our experience (Table 1).

The direction of the mandibular surgical reposi-

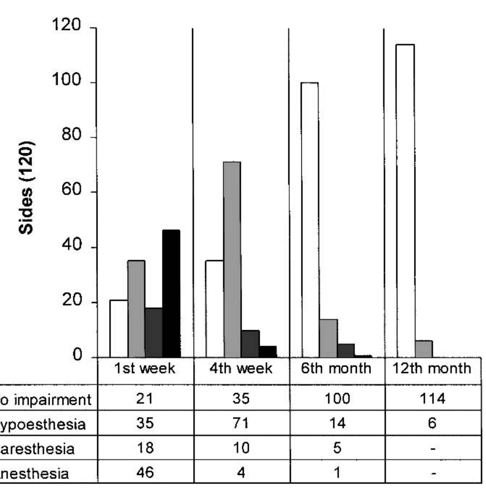

Fig 1 Evolution of spontaneous recovery of IAN impairments after 120 BSSO.

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tioning (advancement vs setback) showed only weak correlation with the evolution of post-surgical impairments in the IAN distribution (Table 1).

Our experience suggests that the entity of mandibular movement associated with advancement-related surgery could be judged as an important factor in determining different recovery pattern, since four sides with hypoaesthesia at the twelfth month of follow-up review underwent mandibular advancement longer than 5 mm.

Long advancement can be responsible for eventual nerve bundle stretching occurred during mandibular repositioning; this represents, together with the evidence of intraoperatory direct damage to the IAN, the clearest cause for nerve impairments lasting more than 12 months, as others reported.

Differently from nerve transections, however, the stretchings still permit an evolution of the nervous functional deficit, but at a slower rate, as our data suggested.

In our experience with 120 BSSOs, only in two cases a surgical manipulation of the nervous bundle was necessary. In both cases the nerve was found to be encased within the lateral aspect of the splitted ramus. Consequently tiny partial ostectomies above and below the nerve allowed to perform a gentle maneuver of holding the IAN away from the lateral splitted side and laying it into the bony canal at the medial wall of splitted ramus. In both the aforementioned cases a persistence of a low-grade hypoesthesia over 12 months was observed. Direct surgical manipulation seem to cause, as well as for the nerve stretching, a slower rate in the spontaneous recovery of the impairment. This seems to be confirmed by the finding of a faster rate in the IAN at the other side, that was not manipulated.

Our analysis revealed that thermal and nociceptive sensibilities show a faster spontaneous recovery than the epicritic and discriminative sensibilities. This seems to be due, as previous studies suggested[3], to a better spontaneous restoration of the mielinic and amielinic fibers with a small diameter, such as those conducting thermal and nociceptive sensibilities. Mielinic fibers with a bigger diameter, conducting discriminative and epicritic sensibilities, seem to have a slower recovery.

In our study, the highest rate of spontaneous recovery of the entire IAN functionality was observed at the sixth month. This finding witnesses how neuroapraxia and axonotmesis give a spontaneous recovery that most frequently occurs within six months from surgery, independently from age and sex of patient. The persistence of anesthesia over 12 months could be a sign of neurotmesis.

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