Frequency and Evolution of Lingual Nerve Lesions Following Lower Third Molar Extraction

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Purpose: To calculate the frequency of lingual nerve (LN) damage caused by lower third molar extraction and describe the evolution of LN sensitivity as well as the prognosis of LN damage based on preoperative data.

Patients and Methods: A retrospective study of 4,995 lower third molar extractions performed in 3,513 outpatients of the Department of Oral and Maxillofacial Surgery (University of Barcelona, Spain) between January 1998 and September 2001.

Results: Twenty-four extractions (0.5%) resulted in LN impairment. All involved ostectomy, with tooth sectioning in 20 cases. Cox regression analysis showed no risk factors for the persistence of LN injury during lower third molar extraction. The sensitivity recovery rate was greater in the first 3 months and then gradually decreased.

Conclusion: LN impairment usually recovers, the recovery rate being faster in the first months. LN damage is generally associated with ostectomy and tooth sectioning.
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J Oral Maxillofac Surg 64:402-407, 2006

Damage to the lingual nerve (LN) is a rare complication of lower third molar extractions. There is great variation in the published incidence[1] because of the use of different surgical techniques in removing lower third molars.

Local anesthesia and even general anesthesia can cause damage to the LN, which seems to be more exposed to this risk than the inferior alveolar nerve (IAN) (probably because of its more superficial location).[2,3] However, there is clear evidence that the most important etiologic factor is the manipulation of the lingual flap[4] or cortical bone because of the anatomic proximity between the lower third molar and the LN.

There are many reports on the risk factors of LN impairment[5-9] and its surgical repair.[10-14] However, permanent damage to the LN is uncommon, and there is little information in the dental literature addressing the risk factors associated with permanent rather than transient LN injury. There are also few detailed data on the spontaneous recovery rate. Survival analysis could be a useful tool for identifying the latter.

The principal aim of this study was to calculate the frequency of LN injury following lower third molar extraction in outpatients, with a description of the timing of sensory recovery, based on survival analysis. In addition, an evaluation was made of the influence of preoperative variables on recovery, based on multivariate techniques.

Patients and Methods

All lower third molar extractions in outpatients in the Department of Oral and Maxillofacial Surgery

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(University of Barcelona, Spain) between January 1998 and September 2001 were included. There were no exclusion criteria. A total of 3,513 outpatients were subjected to 4,995 lower third molar extractions. Of these patients, 1,482 were operated on bilaterally, spacing the operations by at least 3 weeks. Extraction was performed under local anesthesia, usually using articaine in 4% solution with epinephrine 1:100,000 (Ultracain; Hoechst, Barcelona, Spain). The surgical field and all surgical materials were sterile. The surgical technique used was similar to that described by Leonard.[15] The surgeon raised a buccal mucoperiosteal flap, which was protected by a Minnesota retractor. Lingual flap retraction was then carried out using an Obwegeser or a Freer periosteal elevator only when considered necessary by the surgeon. Sterile low-speed (20,000 RPM) handpieces and sterile saline solution were used for ostectomy and tooth sectioning when necessary. The wound was closed with 3-0 silk or catgut suture. Antibiotic and nonsteroidal anti-inflammatory medication was prescribed (usually amoxicillin 750 mg, orally, 3 times daily for 4 or 7 days [Clamoxyl 750; GlaxoSmithKline, Madrid, Spain], and sodium diclofenac 50 mg, orally, 3 times daily for 4 or 7 days [Diclofenaco Llorens 50 mg; Llorens, Barcelona, Spain]), with 0.12% chlorhexidine digluconate rinses twice a day for 15 days (Clorhexidina Lacer; Lacer, Barcelona, Spain).

Sutures were removed after 7 days, and the patients were questioned about tongue sensitivity. In cases presenting sensory impairment in the distribution of the LN, the affected area was mapped and two-point discrimination, pin prick, and light touch were assessed. The patient was questioned about tingling sensation in the distribution of the LN. Thermal sensation was not evaluated. The lesion was classified as either dysesthesia (painful sensation triggered by nonnoxious stimuli), hypoesthesia (diminished sensation), or anesthesia (absence of sensation), and was monitored at least after 15 days and after 1, 3, and 6 months. The patient was posteriorly followed-up every 6 months. No patients were subjected to nerve repair surgery.

The following data were collected by the same researcher from the clinical records of the patients with LN impairment: age, gender, operated site, Nolla's stage of the lower third molar,[16] position according to the Pell and Gregory[17] and Winter[18] classifications, difficulty rating (Table 1), degree of inclusion (erupted, mucosal retention, or bone retention), time to recovery or the last follow-up visit if recovery did not take place, and surgical technique (ostectomy or tooth sectioning). Three patients had removed the panoramic radiograph from their clinical records, and

Table 1. DIFFICULTY RATING BASED ON THE WINTER AND PELL AND GREGORY CLASSIFICATIONS

	Difficulty Score*
Angle	
Mesioangular	1
Horizontal/transverse	2
Vertical	3
Distoangular	4
Depth	
Level A	1
Level B	2
Level C	3
Space	
Class I	1
Class II	2
Class III	3

*For example, a vertical third molar presenting level A and Class III would have a difficulty rating of 7 (3+1+3).

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therefore radiologic information such as Nolla's stage and Pell and Gregory and Winter classifications (and therefore difficulty rating) could not be gathered.

Patients with incomplete follow-up (<350 days) were considered dropouts. The differences in the above variables between dropouts and complete follow-up cases were assessed by the U-Mann-Whitney and Pearson's chi-square or Fisher exact tests whenever possible. The level of significance was set at .05.

Statistical analysis and modeling was performed using the Statistical Package for the Social Sciences (SPSS version 9.0; SPSS, Chicago, IL). A Kaplan-Meier survival analysis was performed to calculate the recovery rate over time. The life tables and recovery rate were calculated with the actuarial method.

A Cox regression model was developed, using a stepwise backward procedure, based on the change in likelihood ratio. The dependent variable was the time (in days) elapsed until subjective recovery of the LN injury and normalization of the LN tests (objective recovery) or the last follow-up visit (if recovery did not take place), and the covariates were age, gender, Nolla's stage, the degree of inclusion, the Pell and Gregory ABC and 123 classifications, the Winter classification, the combined difficulty rating, ostectomy, and tooth sectioning. Another model was developed using a stepwise forward procedure based on the change in likelihood ratio. The criteria for inclusion and exclusion of a variable in the model were $P < .05$ and $P > .10$, respectively.

Table 2. PELL AND GREGORY CLASSIFICATION OF THIRD MOLARS

	I	II	III	Total
A	3	6	2	11
B	0	4	4	8
C	1	0	1	2
Total	4	10	7	21

NOTE. Three cases could not be classified because the panoramic radiograph was not available.

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Results

Twenty-three patients (17 women, 6 men) showed impaired LN function when the sutures were removed. Impairment was bilateral in 1 case. The proportion of extractions resulting in LN damage was 0.5% (95% confidence interval [CI], 0.3%–0.7%). One lesion was classified as total anesthesia (this patient was seen only at suture removal, 7 days after surgery, and no additional visits were performed), 2 as dysesthesia (both totally recovered after 60 and 245 days, respectively) and 21 as hypoesthesia. Lesions were followed-up for a median of 91.5 days (interquartile range [IQR], 138.8 days). Four injuries were lost before observing recovery, 1 after 357 days (the lesion in this case being considered permanent), 2 after 63 days, and 1 after 7 days. All patients, except the patient with total anesthesia of the LN, had some degree of tingling sensation.

Seventeen patients did not receive any treatment, while 5 received oral vitamin B complex once a day (usually Nervobion [Merck, Mollet del Vallès, Spain]) for a median of 7.0 days (IQR, 39.8 days), and 3 underwent low-power laser sessions (LASER 305-IRL; Eymara, Barcelona, Spain) weekly, for 7 to 15 weeks. Individuals that were not anxious about recovery were not treated, while those who were apprehensive were given 1 of these treatments according to the patient's preferences and ability to attend low-power laser sessions.

Patient age showed a non-normal distribution, with a median of 25.8 years (IQR, 12.3 years), and a range of 16.8 to 42.8 years. Nolla's development stage likewise showed a non-normal distribution, with a median of 10 (IQR, 1), and a range of 7 to 10.

Fourteen patients with LN damage had been subjected to left lower molar extraction, while 10 had undergone right lower molar extraction. The Pell and Gregory classification[17] is shown in Table 2. Five lower third molars were in a mesioangular position, while 5 were vertical, 8 were horizontal, and 3 were distoangular.

Seven mandibular third molars had been operated on by first-year fellows of the Master of Oral Surgery and Implantology, 10 by second-year fellows, 6 by third-year fellows, and 1 by an experienced surgeon. One lower third molar was erupted, 13 presented mucosal impaction, and 7 were intraosseous. Three lower third molars required ostectomy, and 20 both ostectomy and tooth sectioning. In 1 case (which was lost to follow-up after suture removal), the surgical technique could not be retrieved from the clinical records. The difficulty rating presented a normal distribution, with a mean of 6.0 and a standard deviation of 1.5.

The 3 non-recovered patients whose injuries were not considered permanent (dropouts) did not differ from the rest of the series in terms of age, Nolla's stage, difficulty rating, or duration of follow-up (U-Mann-Whitney test, $P > .05$). There were no differences between followed-up patients and dropouts in terms of gender, operated side, Pell and Gregory and Winter classifications, surgeon experience, surgical technique, depth of inclusion, and the treatment received (chi-square or Fisher exact test, $P > .05$).

Survival and Cox Regression were performed with the pooled sample, without making subgroups of hypoesthesia, dysesthesia, or anesthesia. The reasons were that the patient with anesthesia did not contribute to the analysis because it was an early censored time, and exclusion of the 2 cases with dysesthesia did not change either the survival analysis or the Cox regression.
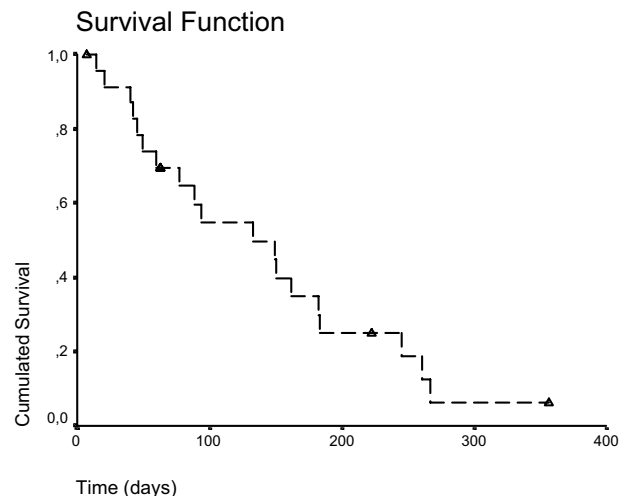
FIGURE 1. Survival function of LN injuries calculated with the Kaplan-Meier method. The horizontal axis represents days after surgery, while the vertical axis reflects the proportion of LN impairment. The discontinuous line represents the cumulative proportion of impaired LNs at a given moment in time. Triangles are censored times (ie, the maximal follow-up time of patients who were lost before observing recovery).

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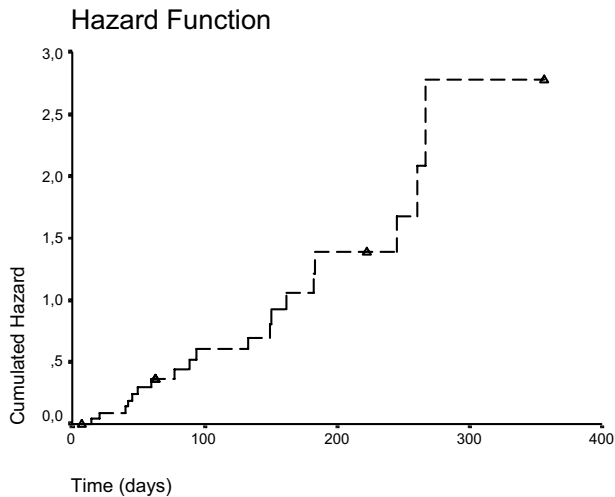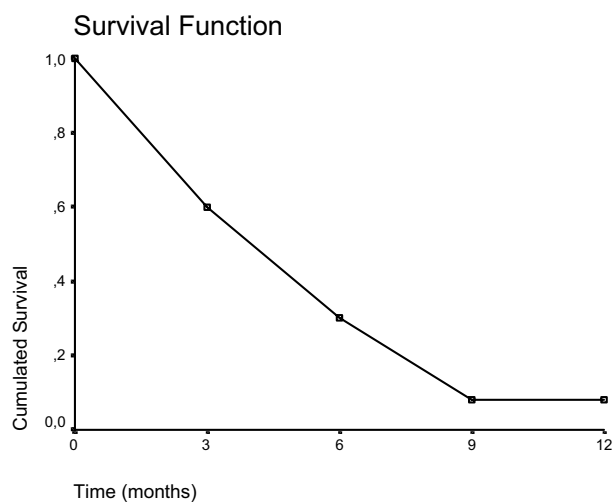
Hazard Function

FIGURE 2. Hazard function of LN impairments calculated with the Kaplan-Meier method. The horizontal axis represents days after surgery, and the vertical axis the cumulative risk of LN recovery. The discontinuous line represents the cumulative hazard of recovery at a given moment in time. Triangles are censored times (ie, the maximal follow-up time of patients who were lost before observing recovery).

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Density Function

FIGURE 4. Density function calculated with the actuarial method. The horizontal axis represents months after surgery, and the vertical axis the recovery rate. The latter seems to be very high in the first three months (0.13% per month), but then gradually decreases.

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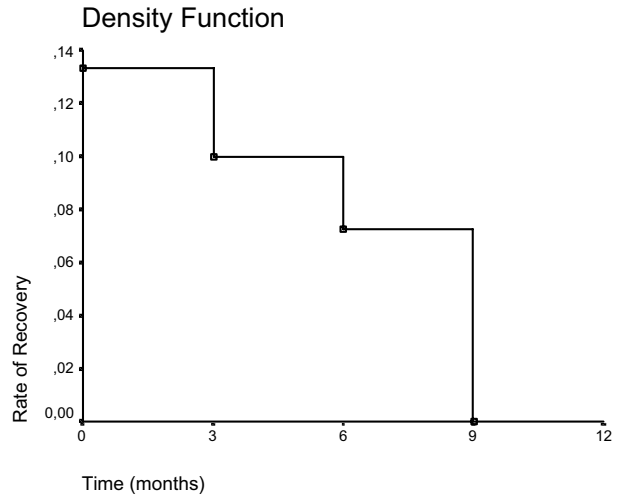
The Kaplan-Meier analysis of cumulative survival is represented in Figure 1. The hazard function is shown in Figure 2. Figures 3 and 4, in turn, show the survival function and density function calculated with the actuarial method. The life table is presented in Table 3.

Cox regression did not include any of the selected preoperative variables. Prognosis could not be related to patient age, gender, position, depth of the molar, surgical technique, or difficulty.

Discussion

There is considerable variation in the dental literature regarding the figures of LN damage after lower third molar extraction.[4,19] In a previously published prospective study on LN injury after lower third molar extraction,[6] the observed frequency was significantly higher (95% CI, 1.2%–2.8% vs 0.3%–0.7% in the present retrospective study). This difference cannot be attributed to underdetection because a retrospective study of IAN damage carried out in parallel with the present study by the same investigators[20] found no differences in the detection of nerve damage with respect to a previous prospective investigation conducted in the same institution.[21] The difference seems to be caused by a change in surgical technique: in 1998 separation of the lingual flap was a common procedure; however, the information that LN damage rate is clearly associated with the insertion of a periosteal elevator in the lingual side of the wound[6,22] probably changed the habits of both fellows and surgeons. One further consideration is that the low frequency of LN damage made it difficult to obtain a sizeable sample of patients with LN damage secondary to lower third molar extraction, thus limiting the possibility of establishing good multivariate models.

All LN impairments were associated with ostectomy, and more than 80% with tooth sectioning. This latter association has been related to the etiology of

Survival Function

FIGURE 3. Survival function calculated with the actuarial method. The horizontal axis represents months after surgery, and the vertical axis the proportion of LN impairments. Squares represent the proportion of LN impairments for a given interval.

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Table 3. LIFE TABLE OF LINGUAL NERVE RECOVERY

	Patients Exposed	Proportion Surviving	Cumulative Proportion of Survival (SE)	Probability Density (SE)	Hazard Rate (SE)
0–3 mos	22.5	0.60	0.60 (0.10)	0.13 (0.03)	0.17 (0.05)
4–6 mos	12	0.50	0.30 (0.10)	0.10 (0.03)	0.22 (0.09)
7–9 mos	5.5	0.27	0.08 (0.06)	0.07 (0.03)	0.38 (0.02)
10–12 mos	0.5	1.00	0.08 (0.06)	0.00 (0.00)	0.00 (0.00)

NOTE. Life table calculated with the actuarial method based on a timeframe of 12 months, subdivided into 3-month intervals. The column "patients exposed" represents the mean number of exposed patients at a given interval. "Proportion surviving" indicates the proportion of patients with lingual nerve impairment that continue to suffer impairment at the end of the time interval. "Cumulative proportion of survival" is the cumulative representation of this same proportion. "Probability density" is the rate of recovery in an interval (percentage of recoveries per month), while "hazard rate" is the rate of recovery for patients who have not recovered at the beginning of an interval (percentage of recoveries per month).

Abbreviation: SE, standard error.

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LN damage in a previous prospective study,[6] and also in a multivariate analysis.[23] One explanation could be that tooth sectioning, if complicated, requires raising a lingual flap. Another aspect is that it increases the risk of damaging cortical bone on the lingual side of the wound. However, we could not find any association between any preoperative or operative data and the prognosis of LN damage.

Local anesthesia also places the LN at risk, although the frequency of LN damage subsequent to anesthesia seems to be very low.[2] This is supported by the fact that LN injuries were observed in patients undergoing complicated extractions and separation of the lingual flap (ie, they were probably caused by the surgical technique). Local anesthesia causes damage to the LN more frequently than to the IAN.[24] While the diameter of the 2 nerves at the site of injection is similar,[25,26] the LN seems to be more exposed to the needle and has fewer fascicles,[27] which could explain why it is more frequently harmed by local anesthesia.

Anatomic factors, such as the proximity of the LN to the alveolar crest distal to the second permanent molar, could be clear risk factors of LN damage. Around 15% of all LN are over or level with the bone ridge at the site of the mandibular third molar.[25,28] This could explain why such an unlikely complication was found bilaterally in 1 patient. However, the course of the LN is not predictable, and can only be detected preoperatively by magnetic resonance imaging,[29] which is not reasonable, taking into account that the rate of LN damage is very low.

Patient age does not seem to have any effect on either the etiology[6] or evolution of LN injuries. It does not seem to be a prognostic factor. Although Renton and McGurk[23] found older age increased the risk of LN damage, this observation could be due to other well-established risk factors.

In the present study most injuries of the LN were transient and mild. Almost half of all LN impairments persisted for less than 3 months, and could be classified as neurapraxias or Sunderland type I or II injuries.[30,31] IAN injuries are generally more severe,[20] and the intraosseous course of the latter nerve makes it more vulnerable to compression, which could explain the difficulties of recovery, particularly in elderly patients.[21]

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