Int. J. Oral Maxillofac. Surg. 2010; xxx: xxx–xxx
doi:10.1016/j.ijom.2010.11.016, available online at http://www.sciencedirect.com

International Journal of Oral & Maxillofacial Surgery

Clinical Paper
Trauma

Evaluation of sensitivity of teeth after mandibular fractures

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D. Brajdić, M. Virag, V. Uglešić, N. Aljinović-Ratković, I. Zajc, D. Macan: Evaluation of sensitivity of teeth after mandibular fractures. Int. J. Oral Maxillofac. Surg. 2010; xxx: xxx–xxx.

Abstract. The sensitivity of teeth anterior to a fracture between the mental and mandibular foramina has been tested and followed up until reinnervation or 3 years has passed. This study assessed the reinnervation period, the number of denervated teeth, and their clinical importance. Fifty patients and 459 teeth were examined. Two hundred and seventy-three teeth were affected and had potentially impaired innervation. Tests after injury showed non-responsive teeth in 81% of affected teeth. Six weeks after injury, 19% of teeth were reinnervated; by 1 year after injury, 92% of initially non-responsive teeth were reinnervated. Most teeth (34%) were reinnervated from 6 weeks to 3 months. All 23/186 initially non-responsive, unaffected, contralateral corresponding teeth were reinnervated within 6 weeks. A year after injury, 95% of incisors, 91% of canines, 94% of premolars, and 82% of molars were reinnervated. Three years after injury, 8% of teeth remain denervated. During the second and third years, no reinnervation occurred, but clinical signs of pulp devitalisation of denervated teeth occurred in 18% or 1% of the initially non-responsive affected teeth. The results revealed the stability of pulp 1 year after injury. Denervated teeth should not be treated if no clinical or radiological signs of devitalisation exist.

Key words: mandibular fracture; tooth sensitivity; tooth vitality; dental pulp.

Accepted for publication 22 November 2010

Mandibular nerve injury is a common complication of mandibular fractures between the mental and mandibular foramina. As a consequence of nerve injury, disturbances of the skin and mucous membrane, and teeth sensitivity occur[16]. Patients experience these disturbances at different intensities, but the condition gradually improves with time. Although it is known from clinical experience that teeth anterior to a fracture line can demonstrate disturbed sensitivity, the problem has not been addressed sufficiently in the literature[27].

Tooth sensitivity testing methods are based on pain, so it is impossible to differentiate vitality (a function of pulp vascularisation) from sensitivity (a function of innervation)[1,4]. Tissue blood perfusion of the oral area can be detected using laser Doppler flowmetry (LDF)[12,14] and pulse oximetry[8]. The use of these physiometric tests for detecting tooth vitality is a valuable resource[15]. Calil et al.[8] concluded that further studies are required to assess their effectiveness and validity in determining pulp vitality in traumatized teeth. If the injury causes an interruption in pulp vascularisation, the result will be pulp tissue death (including the nerve); if only a nerve injury occurs, the vitality of the pulp will not be impaired. Some injuries damage the nerve without influencing the survival of the pulp. Terminal and electrical stimuli only assess the sensitivity of the pulp, so they are not indicated for the direct evaluation of vitality. A tooth that does not change colour and lacks necrotized pulp is vascularized; the innervation is thus of secondary importance. It is known from clinical experience that teeth anterior to the fracture line primarily demonstrate temporary disturbed sensitivity.

The incidence and natural history of post-traumatic sensory disturbances in the distribution of the inferior alveolar nerve (IAN) are insufficiently documented in the literature. This problem has been recognized[16,25] and published studies

0901-5027/000001+05 $36.00/0

include fractures and osteotomies that may or may not involve the mandibular canal in relation to specific methods and periods of fracture reduction[5]. Only a few studies evaluate IAN disturbances by examining tooth sensitivity[33].

In tooth vitality investigations, the value of different stimuli in the detection of tooth vitality is debated. Some studies report electric stimuli to be 100% precise, although they cannot distinguish the quality of vitality[35]. Other reports consider such stimuli to be unreliable[21]. Others prefer vitality tests that measure electric amplitude without power[28], whereas some prefer a thermal vitality meter[13]. It can be concluded that thermal and electric 'vitality' tests, history, and clinical and radiological findings should be secondary methods for detecting pulp status. Such status depends on many things, including age, general status, tooth size, past injuries, and pathological pulp changes[9].

The aims of this investigation were to evaluate IAN disturbances by assessing tooth sensitivity after mandibular fracture with the use of an electric tester and to determine the number of denervated teeth and the time period in which normalisation of tooth sensitivity or devitalisation occurred.

Material and methods

This prospective study used a sample derived from the population of patients with mandibular fractures treated at the Department of Oral and Maxillofacial Surgery in Zagreb between 2006 and 2009. Inclusion criteria were: the presence of a minimally displaced (<3 mm) mandibular fracture between the mental and mandibular foramina because these fractures place the IAN at direct risk of injury[16]; treatment with closed reduction and maxillomandibular fixation with elastics because late deleterious effects on the teeth and periodontal tissues from interdental wiring are uncommon 1 year after the removal of interdental wiring[45]; preoperative and postoperative panoramic radiographs as routine imaging, although it is possible to diagnose the interruption of IAN continuity with magnetic resonance imaging (MRI)[22]; and patients who accepted more follow-up examinations and pulp testing. The investigation included 50 patients with fractures between the mental and mandibular foramina. Anterior to the fracture line, these patients had affected teeth that initially seemed to be avital but actually were not, and they had potentially impaired innervation. The authors assumed that the lack of responsiveness to electric pulp testing was due to inferior dental nerve injury because there was no evidence of direct tooth trauma. Complete documentation was obtained for the patients and complete follow-up was carried out until reinnervation or 3 years had elapsed (in patients in whom reinnervation of all teeth did not occur). Patients with parasymphyseal fractures, teeth involved in the fracture line, carious teeth, teeth with prosthetic restorations, previously devitalized teeth, and teeth injured in the fractures were excluded from the study.

An electric vitality tester was used for sensitivity testing (Digitest model No. D626D, Parkell). It consisted of an instrument casing with a battery. The tester contained a digital electric stimulus slide ranging from 0 to 64, with electrodes patched for examining tooth surface sensitivity and a connection cable applied to the patient's lip. Teeth were dried and isolated with cotton, electrodes were moistened, and the lowest intensity stimulus that caused a reaction was marked as the level of sensitivity. In this investigation, initially sensitive teeth were noted as vital from this time onward, regardless of the presence of a later reaction. The eventual change in the level of sensitivity was not analysed. Teeth that did not react even at the highest level of electric stimulus were considered to be denervated.

Teeth were considered vital if they did not have any clear signs of avitality (e.g. colour change, pathological mobility, radiological periapical transparency, root resorption, or other clinical indicators and process symptoms). Teeth were selected for placement in the avital group based on clinical signs, not because of a negative electric test.

The sensitivity of all potentially endangered teeth was examined on admission (prior to therapy). Electric pulp testing was carried out on the contralateral, corresponding, unaffected teeth for control purposes. The sensitivity of all initially non-responsive teeth was examined 6 weeks, then 3, 4, 6 and 12 months after jaw fracture treatment. The teeth for which sensitivity was not verified (even 12 months after therapy) underwent an additional 2 years of testing as long as they did not show clear clinical signs of avitality.

Results

The sensitivity of 459 teeth was tested. Of these, 273 (60%) were affected anterior to the mandibular fracture between the mental and mandibular foramina and thus had potentially impaired innervation. Of the 459 teeth, 186 (41%) were unaffected, contralateral, corresponding teeth for control purposes. 222/273 (81%) of the affected and 23/186 of the unaffected, contralateral, corresponding teeth were initially non-responsive.

Table 1. Number of reinnervated affected initially non-responsive teeth in relation to the time period after the injury.

Time (months)	No	%
>1.5	42	18.9
>3	117	52.7
>4	156	70.3
>6	188	84.7
>12	205	92.3
>36	205	92.3

The number of reinnervated teeth increased with time. Six weeks after the injury, 19% were reinnervated. Roughly 85% of teeth were reinnervated after 6 months, and 92% of teeth were reinnervated 1 year after the injury. No reinnervation occurred later than 1 year following the injury (Table 1).

When the reinnervation of teeth in a determined time period was analysed, most of the teeth were reinnervated in the period 6 weeks to 3 months after the injury (34%). Fewer teeth were reinnervated in the period 7–12 months after the injury (8%), and no teeth were reinnervated in the period 1–3 years after the injury.

The results were analysed for groups of teeth, because of the small number of samples for particular teeth. A year after injury, 95% of incisors, 91% of canines, 94% of premolars, and 81% of molars were reinnervated (Table 2).

Most medial incisors (31%) were reinnervated by 6 weeks after injury or between 6 weeks and 3 months (31%). Only 7% of medial incisors were reinnervated between months 7–12. Most lateral incisors (46%), canines (36%), second premolars (31%), first molars (33%), and second molars (36%) were reinnervated in the period between 6 weeks and 3 months. The same number of first premolars (26%) was reinnervated during the period from 6 weeks to 3 months and the period from months 5–6. Most wisdom teeth were reinnervated during months 5–6. None of the second molars was reinnervated until 6 weeks after injury, but the same number was reinnervated during months 5 and 6 and from 7 to 12 months (1/11).

Three years after the injury, 17 of 222 teeth (8%) remained denervated. The most frequently denervated teeth were molars (19%), while the least frequently denervated teeth were the incisors (5%). Canines (4/45) and first premolars (3/35)

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Table 2. Reinnervation of affected initially non-responsive teeth groups in determined time period.

	Groups of teeth									
	Incisors (86)		Canines (45)		Premolars (64)		Molars (27)		Total (222)	
Time (months)	No	%	No	%	No	%	No	%	No	%
>1.5	21	24.4	10	22.2	8	12.5	3	11.1	42	18.9
1.5–3	33	38.4	16	35.5	18	28.1	8	29.6	75	33.8
>4	10	11.6	9	20.0	16	25.0	4	14.8	39	17.5
5–6	11	12.8	3	6.7	13	20.3	5	18.5	32	14.4
7–12	7	8.1	3	6.7	5	7.8	2	7.4	17	7.7
13–36	0	0	0	0	0	0	0	0	0	0
Total	82	95.2	41	91.1	60	93.8	22	81	205	92.3

were numerically the most frequently denervated teeth; as a percentage, however, third molars (29%) were the most frequently denervated teeth.

From 1 to 3 years after injury, 14/17 (82%) non-responsive teeth were denervated. This represents 6% of the initially non-responsive teeth (14/222). From 1 to 3 years after injury, 3/17 (18%) of denervated teeth were devitalized. Only three of 222 (1%) of the initially non-responsive teeth remained devitalized 3 years after the injury. The teeth that were devitalized included one lateral incisor from the 44 initially denervated lateral incisors (2%), one canine from the 45 initially denervated canines (2%), and one wisdom tooth from the 35 initially denervated wisdom teeth (3%).

Discussion

In this investigation, the number of reinnervated teeth increased with time after the injury. One year after injury, 92% of teeth were reinnervated. Only Ferdousi & Macgregor[11] analysed the vitality changes of teeth after mandibular fractures in addition to other changes after IAN damage. In his investigation, all teeth responded in vitality tests 6 months after injury.

A question remains regarding which part of the tooth reacts to the stimulus. In 1967, Mumford[29] suggested that periodontal tissue reacts to the stimulus. Although the weakest electric stimulus can produce a periodontal tissue reaction and produce false information regarding tooth sensitivity and/or vitality, Närhi[30] concluded that the stimulus necessary for a periodontal tissue reaction is much higher and that the monopolar vitality meter is safe when used with careful handling. Tooth innervation is much less important than vascularisation and the pulp integrity depends on the blood supply[46].

Although parasymphyseal fractures were excluded from this study, affected teeth with potentially impaired sensitivity are all from the fracture line between the mental and mandibular foramina to the midline of the mandible. Thus a question remains regarding crossover sensation from the contralateral side. The most interesting time periods regarding reinnervation were suggested by Machida[26] and Robinson[36]. Machida[26] concluded that reinnervation starts 4 weeks after cutting of the nerve and that revascularisation starts 5 days after arteria alveolaris inferior binding. In cats, Robinson[36] found that pulp reinnervation starts 3–9 weeks after IAN cutting due to the ipsilateral mylohyoid, ipsilateral and contralateral lingual, and contralateral alveolar nerves that enter the pulp. Except for the ipsilateral lingual nerve, these do not normally innervate the pulp. Mucosal and skin reinnervation crossing the midline has not been demonstrated elsewhere[18]. If the original innervation is allowed to regenerate after such a collateral reinnervation has been formed, the collateral innervation is not withdrawn[37]. When the injury is extended to mimic some aspects of reconstructive jaw surgery, the sources of reinnervation of tooth pulp are the recovering ipsilateral IAN, the contralateral IAN, and the mental and lingual nerves on both sides[39]. Robinson[40] also studied 21 adult patients with unilateral IAN or mental nerve lesions. They were divided into three groups on the basis of the type of nerve injury. Among other tests, he carried out tooth pulp sensation using a monopolar electric pulp tester. The vitality of all of the lower teeth on the side of the injury, which had not been crowned or root-filled, was recorded. When nerve compression occurred, tooth pulp sensation appeared to return to normal by 1 day to 4 months post-injury. When nerve section occurred, tooth pulp sensation was normal by 3, 6 and 11 months post-injury. Fifteen months post-injury one patient reported having had two restorations on the side of the injury, painlessly without local anaesthesia, despite the teeth responding normally to electric pulp testing. After nerve section and regeneration block (resected mandible and reconstructed defect), tooth pulp sensation returned in one patient in the ipsilateral incisors, canine and both premolars between 6 and 9 months post-injury. The results of pulp testing suggested the development of a collateral reinnervation of the teeth. Robinson said that the false localisation of stimuli on the side of injury to a position near to the midline on the contralateral side would be consistent with the development of a collateral reinnervation across the midline. His previous research demonstrated collateral reinnervation after IAN injuries in the cat, after tooth reimplantation and after segmental osteotomy. Trigeminal nerve fibres are able to sprout across the midline into the pulps of denervated contralateral teeth[38]. It is initiated by a peripheral stimulus, probably a trophic factor in the denervated tissue[39]. A similar pattern of recovery has been described after IAN injuries[11,41]. Owen et al.[32] suggested that nerve growth factor plays an important role in collateral reinnervation from high-threshold sensory nerves.

Studies of denervated or poorly innervated teeth have identified contributions of the nerve fibres to tooth repair[6]. The incidence of tooth necrosis increases after injury when nerve fibres are missing[3]. The location and the size of the injury[7] and the rate of infection[20] determine the extent to which the injury of denervated teeth leads to irreversible pulpitis.

In this investigation, the greatest number of teeth remaining denervated 3 years post fracture, were canines (4/45), but, based on percentages, wisdom teeth exhibited the highest frequency (2/7, 29%). The most frequently denervated group of teeth was the molars (19%). The reason for this may be that these teeth have few roots therefore their potential for damage is higher. The canine area is at higher risk for fracture and damage because canines have longer roots[23]. The situation is similar for devitalized

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teeth. One incisor, canine, and third molar became avital; however, when percentages are considered wisdom teeth (14%) were the most frequent, owing to the small sample size in this investigation.

No teeth became reinnervated later than 1 year after the injury, and only three of 222 initially non-responsive teeth (1%) were devitalized during the 1–3 year period after the injury. Therefore, it can be concluded that the pulp is vitally stable 1 year after the injury.

It is known from clinical experience that denervated teeth after segmental osteotomies regain their innervation and vascular supply[10]. This is supported by experimental micro-angiographic studies showing that, with few exceptions, the pulp remains following experimental dentofacial surgery. Blood flow is present in the teeth at all times after posterior segmental osteotomy, but there is a risk of ischaemia[24].

Patients with mandibular fractures are difficult to study because of the non-elective nature of the fracture and the high incidence of accompanying injuries. The type of nerve injury varies due to fracture type and displacement. In most studies, these fractures are treated with miniplates, monocortical screws, and intermaxillary fixation[34]. The incidence of IAN injury after mandibular fracture ranges from 46%[34] to 81%[16] preoperatively, and 77%[42] to 91%[19] postoperatively; a 1 year follow-up shows an incidence of 0% to 45%[19].

The present results can be compared with those from recent investigations of sensitivity alterations after mandibular osteotomies and distraction osteogenesis[2]. The incidence of IAN disturbances ranged from 10% to 94%, depending on the testing method used[44]. The most pronounced nerve damage recovery occurred during the first 3 months[43], and most patients declared their sensation to be 'normal' 1 year after the operation[47]. Recovery of IAN neurosensory function after a bilateral sagittal split osteotomy varies from 2 to 30 months, depending on the surgical technique, patient's age, fixation method, and perioperative position of the nerve[31]. The highest rates of recovery after third molar surgery and iatrogenic injury to the IAN were observed during the 6 months after injury. The IAN cannot retract on transaction in a bony canal, and the canal wall may act as a conduit for sprouting axons[17]. Considering the present results in relation to these investigations, it is obvious that the reinnervation of teeth and the recovery from IAN sensory disturbance starts between 6 weeks and 2 months after injury and can proceed for 2.5 to 3 years.

In conclusion, the current study clearly shows that denervation occurred in four-fifths of the affected teeth and that 1 year after injury, 92% of the initially non-responsive, affected teeth were reinnervated. The pulp is vitally stable 1 year after the injury, and denervated teeth should not be treated if neither clinical nor radiological signs of devitalisation are present.

Competing interests

None declared.

Funding

This work has been supported by Croatian Ministry of Science, Education and Sports Grant No. 065-1080057-0429.

Ethical approval

Not required.

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