

# Endodontics-Related Paresthesia of the Mental and Inferior Alveolar Nerves: An Updated Review

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## ABSTRACT

Paresthesia is a burning or prickling sensation or partial numbness resulting from neural injury. Paresthesia resulting from periapical pathosis or various stages of root canal treatment is of great importance in the field of endodontics. The purpose of this paper is to review paresthesia caused by periapical lesions, local anesthesia, cleaning, shaping and obturation.

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Sensory disturbances such as paresthesia, anesthesia, hypoesthesia and hyperesthesia may occur in the oral cavity. Paresthesia is defined as a burning or prickling sensation or partial numbness caused by neural injury. Patients have described it as warmth, cold, burning, aching, prickling, tingling, pins and needles, numbness and formication (itching in the absence of stimuli).<sup>1-3</sup>

In dentistry, paresthesia can be caused by systemic or local factors. Multiple sclerosis,<sup>4,5</sup> sarcoidosis,<sup>6</sup> metastasis,<sup>7</sup> viral and bacterial infections,<sup>8,9</sup> leukemia and lymphoma<sup>10</sup> are some of the systemic disorders that may cause orofacial paresthesia. Local factors include traumatic injuries, such as mandibular fractures, expanding compressive lesions (benign or malignant neoplasia and cysts), impacted teeth, local infections (osteomyelitis, periapical and peri-implant infections), iatrogenic lesions after tooth extractions, anesthetic injection, endodontic therapy (overfilling and apical surgery), implantology, orthodontic surgery and preprosthetic surgery. The purpose of this paper is to review the endodontics-related causes of paresthesia: periapical infections, overfilling and apical surgery.<sup>11</sup>

## Diagnosis of Paresthesia

Diagnosis of neural anesthesia or paresthesia requires an accurate patient history to determine the onset of the sensory alteration and its evolution. Examination of the affected area can be carried out using thermal, mechanical, electrical or chemical tests that elicit subjective responses.<sup>12</sup> A more objective test is based on electrophysiologic analysis of the nerve. Radiographic and neurophysiologic screening are also required.<sup>13</sup> Radiographic measures may also be helpful in determining the cause of paresthesia. In addition to periapical and panoramic radiographs, a computed tomography (CT) scan will be helpful in displaying small structures and revealing their spatial relations in 3 dimensions.<sup>14</sup>

In cases of nonpersistent episodes of nerve irritation, paresthesia should resolve within days or weeks as the cause is removed.

## Anatomical Considerations

Beyond the trigeminal ganglion, the fifth cranial nerve divides into 3 branches; the lateral branch is the largest and is the sensory root of the mandibular nerve. The mandibular

branch leaves the cranium through the oval foramen and runs into the infratemporal fossa. When it emerges from the cranium, it unites with the motor root to form the mandibular nerve—a mixed nerve. The mandibular nerve immediately divides into many sub-branches, including the buccal nerve, the masseteric nerve, the pterygoid nerves, the temporal nerves, auriculotemporal nerve, the lingual nerve and the inferior alveolar nerve (IAN).<sup>15</sup>

The IAN runs posteriorly and slightly to the lateral side of the lingual nerve, between the pterygoid muscles. It turns laterally and enters the mandibular canal through the mandibular foramen. The IAN, which is strictly sensory, then passes through the mandible to the premolar region, where it divides into the incisive branch, which innervates the anterior segment of the mandible, and the mental nerve, which leaves the mandible through the mental foramen and innervates the overlying tissues.<sup>16</sup> Within the mandible, the IAN has dental and interdental branches. The dental branches form the dental plexus and innervate the teeth; the interdental branches innervate the alveolar bone, the periodontium and the gingivae. The IAN's route through the mandible is located beneath the roots of the teeth close to the dental apices.<sup>17</sup>

The anatomy of the IAN is more complicated than previously thought; paralleling the alveolar artery, it divides into subcanals forming a sort of plexus. Moreover, the neurovascular bundle has been shown to be in contact or close to the lingual cortical plate between the mandibular and mental foramina.<sup>18</sup>

According to Littner and colleagues<sup>19</sup> the upper border of the mandibular canal is located 3.5 to 5.4 mm below the root apices of the first and second molars. The apices of the third molar are closest to the alveolar nerve. Denio and colleagues<sup>20</sup> revealed that the apex of the second molar is 3.7 mm from the upper border of the mandibular canal and that the mesial root apices of the first molars are farther from the alveolar canal by about 6.9 mm. Therefore, although the relation between the IAN and the molar root apices varies, these structures are sometimes very close, allowing pathologic periapical conditions to affect the nerve structures in the mandibular canal.

## Endodontics-Related Causes of Paresthesia

### Periapical Infections

Paresthesia due to periapical lesions may be caused by mechanical pressure and ischemia associated with the inflammatory process (edema), local pressure on the mental nerve resulting from accumulation of purulent exudate in the mandibular bone as well as the toxic metabolic products of bacteria or the inflammatory products

released with tissue damage. The expanding infectious process and the associated edema can cause pressure on the nerve fibres severe enough to induce the symptoms of paresthesia. A subsequent hematoma can also cause sufficient pressure on the nerve fibres to induce paresthesia.<sup>21</sup>

Mental nerve paresthesia (MNP) and IAN paresthesia (IANP) due to periapical pathosis have been reported. (IANP includes paresthesia of both mental and incisive branches of the IAN.) Giuliani and colleagues<sup>17</sup> reported a case of IANP caused by endodontic pathosis in a lower right second molar. Di Lenarda and colleagues<sup>12</sup> reported a case of MNP caused by a periapical infection of the right mandibular second premolar. In another case, MNP was reportedly caused by infection in an impacted tooth.<sup>21</sup> Morse<sup>21</sup> reported a case of mental nerve paresthesia caused by periapical infection of a mandibular canine

tooth. In this case, MNP had occurred even though the canine tooth and the periapical pathosis were not in direct contact with the neurovascular bundle, were

far from the anterior border of the mental nerve and only slight swelling was present. Ozkan and colleagues<sup>11</sup> reported MNP originating from apical pathosis of a mandibular canine tooth.

### Local Anesthesia

Anesthesia that persists for days, weeks or months following injection of local anesthetic solution signals a potential problem. Injection of local anesthetic solutions contaminated by alcohol or sterilizing solution may produce irritation resulting in edema and increased pressure, leading to paresthesia. Alcohol, especially, is neurolytic and can produce paresthesia lasting for months to years. Another factor to consider is trauma to the nerve sheath by the needle. In these cases, the patient reports the sensation of an electric shock. Hemorrhage into or around the neural sheath may increase pressure on the nerve and lead to paresthesia.<sup>22</sup>

### Cleaning and Shaping

According to Rowe,<sup>23</sup> the IAN can be directly damaged during root canal preparation because of overinstrumentation. Grossman<sup>24</sup> found that repair of mechanical damage to the IAN by scarring can cause immediate but temporary paresthesia. Furthermore, excessive preparation of the root canal often results in disruption of the apical constriction,<sup>25</sup> which in turn, is responsible for extrusion of irrigants, medicaments and obturation materials (gutta-percha and sealer) into the periapical region and subsequent chemical and mechanical nerve injury.<sup>26</sup>

Another potential cause of paresthesia is the irrigation solution, especially sodium hypochlorite (NaOCl), used during cleaning and shaping. Inadvertent injection

The anatomy of the inferior alveolar nerve is more complicated than previously thought.

of NaOCl beyond the apical foramen may occur in teeth with wide apical foramina or when the apical constriction has been destroyed during root canal preparation or by resorption. In addition, extreme pressure during irrigation or binding of the irrigation needle tip in the root canal leaving no room for the irrigant to leave the root canal coronally may result in large volumes of the irrigant contacting apical tissues. If this occurs, the excellent tissue-dissolving capability of NaOCl will lead to tissue necrosis.<sup>27</sup>

Becking<sup>28</sup> has presented 2 cases of NaOCl injection into the periapical soft tissues. In the first, NaOCl of unknown concentration was extruded through the apical foramen of a mandibular left second molar with a perforation at the cemento-enamel junction, resulting in progressive swelling of the left side of the face extending to the patient's neck. After 1 day, necrosis of the mucosa and anesthesia of the mental nerve were apparent. With antibiotic and analgesic therapy, pain and swelling diminished after 5 days, paresthesia of the nerve resolved after 10 days and healing of the mucosa occurred in 2 months. In the second case, apical overextrusion of NaOCl occurred during root canal preparation of a mandibular left second premolar resulting in severe pain, swelling and anesthesia of the mental nerve. Hulsmann and Hahn,<sup>27</sup> as well as Gernhardt and colleagues,<sup>29</sup> have questioned the use of NaOCl during root canal irrigation of mandibular molars and premolars.

Another possible inadvertent complication of root canal treatment of mandibular premolar and molar teeth with apical periodontitis is the extrusion of infected debris into the mandibular canal or mental foramen. All hand and rotary instrumentation techniques potentially extrude debris and bacteria beyond the root canal.<sup>30</sup> According to Morse,<sup>21</sup> this infected debris might breach the protective perineurium of the IAN and impair nerve conductivity.

### Root Canal Filling

Root canal irrigants, intracanal medicaments and filling materials should be limited to the root canal and not extend to periapical tissues or other neighbouring structures.<sup>31</sup> Root canal filling materials, including gutta-percha and sealers, can induce paresthesia via mechanical or chemical mechanisms.<sup>32</sup> Their spread beyond the apical foramen can result in clinical manifestations related to the toxicity of the product, although minor material extrusions are generally well tolerated by the periradicular tissues.<sup>33</sup> When the filling materials come into contact with nerve structures, the toxic effects can

manifest in the form of anesthesia, hypoesthesia, paresthesia or dysesthesia that may prove irreversible.<sup>34</sup>

Gutta-percha is the most common solid root canal filling material.<sup>35</sup> Gutta-percha cones generally contain 20% gutta-percha, 60%–70% zinc oxide, plasticizing agents (waxes and resins), barium sulfate, colouring agents and trace metals. Gutta-percha has a low level of toxicity, which makes it a material of choice in endodontic treatment.<sup>35</sup> Thus, the passage of a gutta-percha cone beyond the apex rarely causes paresthesia.<sup>36</sup> Indeed, the cone tends to deform without compressing the nerve or artery.

In addition, it is a relatively inert material that causes no lesions of chemical origin if it contacts the neurovascular bundle.<sup>32</sup> Gutta-percha has been evaluated in various cell culture systems and found to elicit no or low cytotoxicity, although cytotoxic effects varied among brands.<sup>37</sup> However, depending on the gutta-percha product used, in vivo studies in both soft and hard tissues resulted in reactions varying from none to chronic inflammation. The observed inflammation may have been induced by anti-oxidants and oxides associated with the gutta-percha compound.<sup>37</sup> Systemic reaction to gutta-percha products has not been reported.<sup>36</sup>

Another potential cause of paresthesia is root canal sealer. Paresthesia caused by sealers is mainly of chemical origin.<sup>37</sup> Zinc oxide and eugenol-based sealers are among the most commonly used in endodontic treatment. Toxicity is mainly due to eugenol,<sup>31,36</sup> whose neurotoxic effect and its involvement in paresthesia or anesthesia when in direct contact with the IAN has been demonstrated.<sup>38</sup> Ozeki<sup>39</sup> reported that a concentration of 200 ppm eugenol can anesthetize crayfish. Trowbridge and colleagues<sup>40</sup> showed that freshly mixed zinc-oxide eugenol inhibits intradental nerve activity.

Calcium hydroxide (Ca[OH]<sub>2</sub>)-based sealers are also common. Boiesen and Bodrin<sup>41</sup> found that they caused irreversible nerve damage in an experimental model, and this was confirmed in clinical studies.<sup>42</sup> Ahlgren and colleagues<sup>43</sup> reported a case in which extrusion of a large amount of a Ca(OH)<sub>2</sub>-based sealer into the periapical area and near the IAN caused paresthesia due to mechanical compression.

Regarding thermal nerve injury, Blanas and colleagues<sup>44</sup> reported a case of paresthesia caused by apical extrusion of gutta-percha following obturation of the root canal with a thermoplastic technique. This method of obturation, described by Schilder,<sup>45</sup> can, in fact, cause paresthesia if used after excessive root canal preparation.<sup>46</sup> Paresthesia results from overheating of the inferior

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alveolar artery or nerve.<sup>44</sup> The temperature for softening thermoplastic gutta-percha ranges from 53.5° to 57.5°C<sup>47</sup> but intracanal temperatures of 50–100°C have been reported.<sup>46</sup> Because temperature elevations of as little as 10°C can cause bone damage and necrosis<sup>48</sup> and nerve tissue is thought to be even more sensitive to thermal insult than bone,<sup>46</sup> the mechanism of nerve injury is understandable with overfill or overextension of thermoplastic gutta-percha into the inferior alveolar canal.

### Treatment Strategies

In cases of nonpersistent episodes of nerve irritation, paresthesia should resolve within days or weeks as the cause is removed. Infection-related paresthesia subsides after appropriate nonsurgical endodontic therapy.<sup>11,21</sup> Paresthesia related to local anesthesia resolves within approximately 8 weeks without treatment.<sup>22</sup>

Long-term or even permanent paresthesia can result in cases of nerve fibre laceration, prolonged pressure on the nerve or contact with toxic overfilled endodontic materials.<sup>10</sup> The immediate therapy should be based on removal of the cause (when possible) and control of inflammation, edema, hematoma or infection. Drugs for such therapy include antibiotics, nonsteroidal anti-inflammatory drugs and corticosteroids, proteolytic enzymes to disintegrate the coagulum and vitamin C, which has antioxidative action and reduces the effects of ischemia.

During the reparative phase (within 30 days of the damage), both pharmacologic and instrumental methods can be used. Drugs include topical steroids, cocarnitine, somatotrophic hormone, nerve growth factor, vitamins C and E (antioxidative), vasodilators (to reduce ischemia) and ozone, which improves the activity of red corpuscles and increases tissue oxygenation.<sup>11,49</sup> Instrumental therapy includes magnetotherapy, laser therapy and application of electrical fields. In a late phase, when repair is no longer possible, the pharmacologic approach is limited to the treatment of persistent neuralgia.<sup>49</sup>

Another approach to managing paresthesia is surgical intervention. Although studies of IAN repair are limited, some have reported acceptable patient response to surgical treatment. In one study, the overall postoperative satisfaction of more than 55% of patients receiving surgical repair for inferior alveolar and lingual nerve injuries was good to excellent.<sup>50</sup> Strauss and colleagues<sup>51</sup> found that 92.2% of patients who underwent IAN microsurgery had statistically significant neurosensory improvement.

According to Zuniga,<sup>52</sup> better treatment outcomes are achieved if mental nerve paresthesia is treated as early as possible. On the other hand, Gregg<sup>53</sup> stated that, because most IAN injuries are known to resolve spontaneously, there is no conclusive evidence that early intervention is better than delayed nonsurgical management (more than 3 months after injury).

### Prognosis

There seems to be a correlation between duration, origin, significance of the injury and prognosis of paresthesia. The longer the mechanical or chemical irritation persists, the more the nerve fibres degenerate and the greater the risk that the paresthesia will become permanent.<sup>32</sup> ♦

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### References

- Morgan GW. Proprioception, touch and vibratory sensation. In: Goetz CG, Pappert EJ, editors. *Textbook of clinical neurology*. Philadelphia: Saunders; 1999. p. 315-32.
- Lambrianidis T, Molyvdas J. Paresthesia of the inferior alveolar nerve caused by periodontal-endodontic pathosis. *Oral Surg Oral Med Oral Pathol*. 1987;63(1):90-2.
- Morse DR. Endodontic-related inferior alveolar nerve and mental foramen paresthesia. *Compend Contin Educ Dent*. 1997;18(10):963-8, 970-3, 976-8.
- Vicente A, Herrero C, Martin E, Valls J, Graus F, Mascaro JM. Trigeminal sensory-neuropathy in systemic sclerosis. *Clin Exp Dermatol*. 1991;16(5):403.
- Farrell DA, Medsger TA Jr. Trigeminal neuropathy in progressive systemic sclerosis. *Am J Med*. 1982;73(1):57-62.
- Bonnet F, Mercie P, Morlat P, Loiseau H, Lifermann F, Lacoste D. [Isolated involvement of the trigeminal nerve of sarcoidosis origin.] *Rev Neurol Paris*. 1997;155(1):59-61. French.
- Chin A, Liang TS, Borislow AJ. Initial presentation of hepatocellular carcinoma as a mandibular mass: case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998;86(4):457-60.
- Schwartz O, Kvorning SA. Tooth exfoliation, osteonecrosis of the jaw and neuralgia following herpes zoster of the trigeminal nerve. *Int J Oral Surg*. 1982;11(6):364-71.
- Barrett AP, Smith MW. Maxillary nerve involvement in bacterial endocarditis. *J Oral Maxillofac Surg*. 1985;43(10):816-7.
- Barrett AP. Selective anesthetics of the inferior alveolar nerve in leukemia and lymphoma. *J Oral Maxillofac Surg*. 1985;43(12):992-4.
- Ozkan BT, Celik S, Durmus E. Paresthesia of the mental nerve stem from periapical infection of mandibular canine tooth: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2008;105(5):e28-31.
- Di Lenarda R, Cadenaro M, Stacchi C. Paresthesia of the mental nerve induced by periapical infection: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;90(6):746-9.
- Zuniga J, LaBanc J. Advances in microsurgical nerve repair. *J Oral Maxillofac Surg*. 1993;51(1 Suppl 1):62-8.
- Gluskin AH. Mishaps and serious complications in endodontic obturation. *Endod Top*. 2005;12:52-70.
- Sicher H. The nerves of head and neck. The trigeminal nerve. In: Sicher H, editor. *Oral anatomy*. St Louis: Mosby; 1965. p. 369-86.
- Williams PL, Warwick R, Dyson M, Bannister LH. Trigeminal nerve. In: Davies DV, Coupland RE, editors. *Gray's anatomy: descriptive and applied*. 34th ed. London: Longman Group Ltd; 1967. p. 1089-92.
- Giuliani M, Lajolo C, Deli G, Silveri C. Inferior alveolar nerve paresthesia caused by endodontic pathosis: a case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2001;92(6):670-4.



18. Gowgiel JM. The position and course of the mandibular canal. *J Oral Implantol*. 1992;18(4):383-5.
19. Littner MM, Kaffe I, Tamse A, Dicapua P. Relationship between the apices of the lower molars and mandibular canal — a radiographic study. *Oral Surg Oral Med Oral Pathol*. 1986;62(5):595-602.
20. Denio D, Torabinejad M, Bakland LK. Anatomical relationship of the mandibular canal to its surrounding structures in mature mandibles. *J Endod*. 1992;18(4):161-5.
21. Morse DR. Infection-related mental and inferior alveolar nerve paresthesia: literature review and presentation of two cases. *J Endod*. 1997;23(7):457-60.
22. Malamed SF. Local complications. In: Malamed SF, editor. *Handbook of local anesthesia*. 4th ed. St Louis: Mosby; 1997. p. 248-9.
23. Rowe AH. Damage to the inferior dental nerve during or following endodontic treatment. *Br Dent J*. 1983;155(9):306-7.
24. Grossman LI. Paresthesia from N2 or N2 substitute. Report of a case. *Oral Surg Oral Med Oral Pathol*. 1978;45(1):114-5.
25. Gutiérrez JH, Brizuela C, Villota E. Human teeth with periapical pathosis after overinstrumentation and overfilling of the root canals: a scanning electron microscopic study. *Int Endod J*. 1999;32:40-8.
26. Scolozzi P, Lombardi T, Jaques B. Successful inferior alveolar nerve decompression for dysesthesia following endodontic treatment: report of 4 cases treated by mandibular sagittal osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2004;97(5):625-31.
27. Hulsmann M, Hahn W. Complications during root canal irrigation — literature review and case reports. *Int Endod J*. 2000;33(3):186-93.
28. Becking AG. Complications in the use of sodium hypochlorite during endodontic treatment. Report of three cases. *Oral Surg Oral Med Oral Pathol*. 1991;71(3):346-8.
29. Gernhardt CR, Eppendorf K, Kozlowski A, Brandt M. Toxicity of concentrated sodium hypochlorite used as an endodontic irrigant. *Int Endod J*. 2004;37(4):272-80.
30. Er K, Sümer Z, Akpınar KE. Apical extrusion of intracanal bacteria following use of two engine-driven instrumentation techniques. *Int Endod J*. 2005;38(12):871-6.
31. Poveda R, Bagán JV, Fernández JM, Sanchis JM. Mental nerve paresthesia associated with endodontic paste within the mandibular canal: report of a case. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2006;102(5):e46-49.
32. Tilotta-Yasukawa F, Millot S, El Haddioui A, Bravetti P, Gaudy JF. Labiomandibular paresthesia caused by endodontic treatment: an anatomical and clinical study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2006;102(4):e47-59.
33. Pertot W, Camps J, Remusat M, Proust J. In vivo comparison of the biocompatibility of two root canal sealers implanted into the mandibular bone of rabbits. *Oral Surg Oral Med Oral Pathol*. 1992;73(5):613-20.
34. Brodin P, Røed A, Aars H, Orstavik D. Neurotoxic effects of root filling materials on rat phrenic nerve in vitro. *J Dent Res*. 1982;61(8):1020-3.
35. Pascon EA, Spångberg LS. In vitro cytotoxicity of root canal filling materials: 1. Gutta-percha. *J Endod*. 1990;16(9):429-33.
36. Khabbaz MG, Papadopoulos PD. Deposition of calcified tissue around an overextended gutta-percha cone: case report. *Int Endod J*. 1999;32(3):232-5.
37. Dahl JE. Toxicity of endodontic filling materials. *Endod Top*. 2005;12:39-43.
38. Kozam G. The effect of eugenol on nerve transmission. *Oral Surg Oral Med Oral Pathol*. 1977;44(5):799-805.
39. Ozeki M. The effects of eugenol on the nerve and muscle in crayfish. *Comp Biochem Physiol C*. 1975;50(1):183-91.
40. Trowbridge H, Edwall L, Panopoulos P. Effect of zinc oxide-eugenol and calcium hydroxide on intradental nerve activity. *J Endod*. 1982;8(9):403-6.
41. Boiesen J, Bodrin P. Neurotoxic effect of two root canal sealers with calcium hydroxide on rat phrenic nerve in vitro. *Endod Dent Traumatol*. 1991;7(6):242-5.
42. Yatsuhashi T, Nakagawa K, Matsumoto M, Kasahara M, Igarashi T, Ichinohe T, et al. Inferior alveolar nerve paresthesia relieved by microscopic endodontic treatment. *Bull Tokyo Dent Coll*. 2003;44(4):209-12.
43. Ahlgren FK, Johannessen AC, Hellem S. Displaced calcium hydroxide paste causing inferior alveolar nerve paraesthesia: report of a case. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2003;96(6):734-7.
44. Blanas N, Kienle F, Sándor GK. Inferior alveolar nerve injury caused by thermoplastic gutta-percha overextension. *J Can Dent Assoc*. 2004;70(6):384-7.
45. Schilder H. Filling root canals in three dimensions. *Dent Clin North Am*. 1967;11:723-44.
46. Fanibunda K, Whitworth J, Steele J. The management of thermomechanically compacted gutta percha extrusion in the inferior dental canal. *Br Dent J*. 1998;184(7):330-2.
47. Cohen BD, Combe ED, Lilley JD. Effect of thermal placement techniques on some physical properties of gutta-percha. *Int Endod J*. 1992;25(6):292-6.
48. Eriksson AR, Albrektson T. Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit. *J Prosthet Dent*. 1983;50(1):101-7.
49. Franco A, Ferronato G. *Nervo mandibolare in odontostomatologia*. Padova, Italy, Frafın Sas; 1996.
50. Lam NP, Donoff RB, Kaban LB, Dodson TB. Patient satisfaction after trigeminal nerve repair. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2003;95(5):538-43.
51. Strauss ER, Ziccardi VB, Janal MN. Outcome assessment of inferior alveolar nerve microsurgery: a retrospective review. *J Oral Maxillofac Surg*. 2006;64(12):1767-70.
52. Zuniga JR. Surgical management of trigeminal neuropathic pain. *Atlas Oral Maxillofac Clin North Am*. 2001;9(2):59-75.
53. Gregg JM. Surgical management of inferior alveolar nerve injuries (Part II): The case for delayed management. *J Oral Maxillofac Surg*. 1995;53(11):1330-3.