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# Unexpected Temporary Mental Nerve Deficit after Extraction of Mandibular First Molar: Case Report

**Abstract:** Temporary neurologic deficit affecting the distribution of the left mental nerve was reported in a warfarinized, diabetic patient who had a history of previous CVA with right-sided body weakness. The neurologic deficit was reported following routine extraction of a left mandibular first molar. Recovery of sensation began six weeks after the procedure. Possible mechanisms and preventive measures are discussed.

**Clinical Relevance:** The reader should understand that mental paraesthesia can be an unexpected outcome in the most routine of mandibular extractions.

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Neurologic deficit affecting the mental nerve following a routine extraction of a mandibular first molar is an unexpected outcome. There are several possible causes for isolated mental neuropathy, but the most common by far is as a result of iatrogenic dental treatment.<sup>1</sup> This paper documents an occurrence of anaesthesia related

to the distribution of the left mental nerve following administration of local anaesthetic for the extraction of the lower left six.

## Case report

A 40-year-old woman was referred to the Oral and Maxillofacial Surgery Department of the Royal Dental Hospital of Melbourne for the extraction of a carious broken down lower left six. She was initially seen in the emergency department where she complained of having broken a tooth in quadrant 3, which was now irritating her tongue.

Her past medical history included CVA and Type 2 diabetes mellitus. Her medication included warfarin, insulin, omeprazole, artovastatin, spirinolactone and iron and vitamin B tablets. Her extra-oral examination was unremarkable, with no lymphadenopathy noted. She had no history of facial numbness.



**Figure 1.** Periapical radiograph from emergency department before temporization.

On examination, the tooth was not tender to percussion. Fracture of the crown of the lower left six was noted with only the mesio-lingual cusps still standing. The remainder of the tooth was broken down below the gingival margin. A periapical radiograph was taken (Figure 1). On review of the orthopantomogram, the roots of the lower left six were not in

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**Figure 2.** Periapical radiograph of socket of lower left six two weeks post extraction.



**Figure 3.** Area of extra-oral anaesthesia two weeks post extraction.



**Figure 4.** Six weeks post extraction. Red – nil response to cold sensation (CO<sub>2</sub> stick). Green – nil response to touch and two point discrimination.



**Figure 5.** Nine weeks post extraction. Red – nil cold sensation (CO<sub>2</sub> stick).

proximity to the inferior alveolar nerve (IAN) or mental foramen. A periapical radiolucency of approximately one millimetre diameter was associated with the mesial and distal roots.

The patient's INR on the day of extraction was measured as 3.2. The extra-oral examination was unremarkable, with no evidence of altered sensation.

A left inferior alveolar nerve block and long buccal block was performed after negative blood aspiration with 2% lignocaine 1:80,000 adrenaline with 4.4mls for the IAN and 2.2 mls for the buccal. The administration of anaesthesia was unremarkable. The remainder of the lower left six crown was then sectioned coronally without the formal raising of a buccal flap. The buccal tissue was protected with a Minnesota retractor and the roots divided and elevated out separately. Haemostasis was achieved following four 3.0 plain gut interrupted sutures and oxidized cellulose (Surgicel®, Johnson and Johnson Pty Ltd, North Ryde, 2113 NSW) was placed in the socket. Post-operative instructions were given and the patient was discharged.

She returned for routine post-operative review two weeks later complaining of numbness of the lip and chin on the left side. A mildly tender slight swelling was noted in the left submandibular region. No nodes were palpable. An extra-oral area of numbness over the left lower lip and chin region with no ability to discriminate between two points and no response to touch and pressure were noted. On intra-oral examination there was food packing in the socket of the lower left six, the area was tender and the gingivae inflamed. A new periapical radiograph was taken (Figure 2), which was unremarkable. The socket was irrigated with saline, and the patient was given a syringe to aid cleaning of the socket and oral hygiene instructions. A minor soft tissue infection was diagnosed and Amoxicillin 500 mg tds for five days was prescribed and the patient given a review appointment for one week.

The patient returned three weeks post extraction for review. She

reported no change in the area of anaesthesia. The area of extra-oral anaesthesia was mapped out with a green pen (Figure 3). There was no response to fine touch, sharp, dull sensation, two point location, brush stroke or cold. Intra-oral anaesthesia on the mucosa labial to the lower left one, two, three and four was noted. However, the tongue, lingual mucosa, and buccal mucosa in the molar region of quadrant three had normal sensation. All the teeth in quadrant three were CO<sub>2</sub> positive with normal responses. This indicated possible sparing of the incisive branch of the mental nerve. A review appointment for three weeks was made.

Six weeks post-operative the extra-oral area of complete anaesthesia was mapped again with a pen. It was found to have diminished in size. An area of negative cold response, but positive to light touch, dull pressure and two point discrimination was noted and mapped in red (Figure 4).

Further review at nine weeks post extraction revealed almost complete resolution of the anaesthetic area. She had a 7/10 touch and pressure sensation response in all the previously affected area and only a small area of nil cold response remained (Figure 5).

## Discussion

Review of the literature documents that most forms of isolated mental neuropathy are dental in origin.<sup>1,2</sup> Included in this category are surgical manoeuvres near the branches of the IAN, as well as local anaesthesia. Other important causes have been recorded. These include infection<sup>3-9</sup> damage to an accessory or variant positioned mental nerve,<sup>10</sup> prolonged pressure in the mental region or lip,<sup>11</sup> toxicity from dental materials,<sup>12</sup> neoplasm<sup>1,13,14,15</sup> and sickle cell crisis.<sup>16</sup>

Omeprazole-related paraesthesia has been reported. The risk is believed to be equal to 0.1% and resolves on cessation of the medication. Artovastatin has been shown on clinical trial evidence to produce paraesthesia and peripheral neuropathy, but this is rare.<sup>17</sup> Paraesthesia related to the

patient's medication can be ruled out in this case as the patient had taken her medication for several years without experiencing altered sensation.

Nerve damage can be related to mechanical, chemical and thermal factors. The CO<sub>2</sub> positive tests of the pulps of the lower left premolar, canine and incisors, and the sparing of buccal sensation suggest that only a portion of the mental nerve fibres were affected.

The administration of a dental injection itself can cause nerve injury.<sup>2</sup> The exact mechanism is unknown, but several have been suggested.

#### **Direct trauma from the injection needle**

The lingual nerve is involved in 70% of cases. When the patient's mouth is held open, the lingual nerve is held taut within the interpterygoid fascia and cannot be deflected away from the needle. The long bevelled dental needle is prone to become barbed after multiple injections and/or bone contact. The barbs may cause perineurium rupture, endoneurium herniation and transection of multiple fibres or fascicles. The injury most commonly occurs on needle withdrawal. Any number of fascicles may be damaged causing transient paresis. Healing occurs within two weeks in 81% of patients.<sup>18</sup>

#### **Trauma to intraneural blood vessels by dental needle**

Trauma to intraneural blood vessels may cause an intraneural haematoma. This results in constrictive epineuritis and localized neurotoxicity. Recovery occurs over several weeks with release of pressure and subsequent remyelination. The speed of recovery depends on the severity of the injury to the blood vessels and pressure from the haematoma.

#### **Neurotoxicity of local anaesthetic**

At higher concentrations, lignocaine has been shown to cause neurotoxic damage following both perineural and intrafascicular injection.<sup>19–21</sup> The local anaesthetics prilocaine and articaine cause more nerve injuries per use than lignocaine.

The causes are postulated to be chemical trauma, or ischaemia and reperfusion injury.

Mental nerve injury accounts for less than one third of nerve injuries after injection.<sup>2</sup> In most cases, the entire distribution of the affected nerve seems to be involved rather than a small number of fascicles. Spontaneous complete recovery from altered sensation occurs within eight weeks in 85–94% of cases. Patients with paraesthesia lasting longer than eight weeks have less chance of full recovery.

An 'electric shock' sensation is believed to occur when a dental needle contacts a nerve trunk. In our patient the anaesthetic lignocaine was administered in three separate injections using the same needle (two passes for the inferior alveolar nerve and one for the long buccal). The injection process was unremarkable. Multiple passages of the dental needle into the space near the inferior alveolar nerve could plausibly have caused damage to some of the fascicles. This could account for the mental nerve paraesthesia. Warfarin anticoagulation with an INR of 3.2 could possibly have contributed to bleeding and haematoma formation in the area around the IAN post injection.

The patient's recovery period, nine weeks, falls within the most common described period for recovery for injection-related inferior alveolar nerve damage.

Infection-related paraesthesia is a possible reason for the sensory deficit observed in this case. Sensory deficit as a result of infection is usually related to mechanical pressure and ischaemia associated with the inflammatory process. Another cause of paraesthesia could be the toxic metabolic products of bacteria and inflammatory products released following tissue damage.<sup>3,4,5,6,8,9</sup> The symptoms usually resolve after management of the infection. As a diabetic, the patient falls within a group more susceptible to infection. In this case, after a five day course of antibiotics and apparent local resolution of inflammation, the neurologic deficit persisted.

In the current case there

was no radiographic evidence that the mental foramen was any further distal than its usual anatomic position inferior and mesial to the apex of the second premolar. A variation in the path of the nerve or an accessory branch is possible. However, no flap was raised during the extraction, nor was any buccal bone removed. The removal of loose interseptal bone and gingival suturing have not been previously associated with mental nerve damage. The literature reports accessory branches of the mental nerve have been found exiting the mandible superior and distal to the mental foramen.<sup>9</sup> Surgicel® has been shown to block axonal conduction when placed in close proximity to nerve tissue, but its effects diminished after two to four weeks.<sup>22,23</sup> Surgicel® was placed in the socket of the lower left six immediately post extraction, but radiographs show the socket to be some distance away from both the inferior dental canal and mental foramen.

With regard to management of patients sustaining iatrogenic nerve injury in the oral and maxillofacial region at our institution, the protocol involves a follow-up at two weeks, four weeks and eight weeks post injury.<sup>24</sup> At each appointment, the depth of paraesthesia is determined by history and examination of the remaining sensation with two point discrimination, dull pressure and fine touch. Sensation is assessed on a visual analogue scale from 0 to 10. Figure 0 is measured as no sensation at all and 10 is normal sensation. Paraesthesia is not numerically assessed as this tends to be confusing.

At the third follow-up appointment (ie eight weeks), depending on the level of sensation and amount of resolution, the patient is given a detailed explanation about treatment options and offered a referral for possible surgical nerve exploration. It has been recommended that exploration and treatment of the damaged nerve should be performed within three months of injury. Should the patient elect conservative treatment, follow-up appointments continue at monthly intervals up to six months post injury, then in two-monthly intervals up to twelve months after the injury. Follow-

up will usually conclude once total resolution of the condition occurs or if it plateaus such that, after discussion with the patient, further review is not required.

## Conclusion

The cause of temporary paraesthesia in this patient is unknown, although the most likely cause of neurologic deficit in this case is trauma from the dental injection and/or local infection. However, her sensory deficit may have also been caused by pressure on an accessory branch of the mental nerve during extraction.

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