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REVIEW ARTICLE

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STUDY ON LINGUAL NERVE INJURY AFTER SURGICAL REMOVAL OF MANDIBULAR THIRD MOLAR AND HOW TO AVOID IT -A REVIEW ARTICLE

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ABSTRACT

Impacted mandibular 3rd molar is most common in younger adults and the patient seeks treatment whenever there is pain, swelling or any other discomfort such as sensory disturbances. Although the rate of complication is low, the efforts to limit intraoperative or postoperative complications may have a great impact in terms of enhancing patient outcome. Mandibular Impacted third molar are in close proximity to the inferior alveolar nerve, lingualnerve, mylohyoid nerve and buccal nerves. Each of these nerves is at risk of damage during surgical removal of mandibular third molar, but the most troublesome complications result from inferior alveolar or lingual nerve injuries. The majority of these injuries results in transient sensory disturbance but, in some cases, permanent paraesthesia (abnormal sensation), hypoaesthesia (reduced sensation) or, even worse, some form of dysaesthesia (unpleasant abnormal sensation) can occur. This paper reviews the lingual nerve injury after surgical removal of mandibular third molar and how to prevent from lingual nerve injury.

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INTRODUCTION

Impacted teeth can be defined as those teeth whose normal eruption is prevented by adjacent tooth, overlying bone or soft tissue, malpositioning and lack of space in the arch, or other impediments. Impacted mandibular 3rd molar is most common in younger adults and the patient seeks treatment whenever there is pain, swelling or any other discomfort such as sensory disturbances. Although the rate of complication is low, the efforts to limit intraoperative or postoperative complications may have a great impact in terms of enhancing patient outcome. Mandibular Impacted third molar are in close proximity to the inferior alveolar nerve, lingualnerve, mylohyoid nerve and buccal nerves. Each of these nerves is at risk of damage during surgical removal of mandibular third molar, but the most troublesome complications result from inferior alveolar or lingual nerve injuries. The majority of these injuries results in transient sensory disturbance but, in some cases, permanent paraesthesia (abnormal sensation), hypoaesthesia (reduced sensation) or, even worse, some form of dysaesthesia (unpleasant abnormal sensation) can occur. These sensory disturbances can be troublesome causing problems with mastication, speech and may adversely affect the patient's quality of life. They also constitute the most frequent causes of complaints and litigation. It is therefore imperative that patients sustaining nerve injuries are managed correctly, and includes correct diagnosis of type of injury, treatment of appropriate cases and monitoring recovery. The incidence of lingual nerve injury may occur due to surgeon's inexperience, procedure methodology, certain specific factors such as raising and retracting a lingual mucoperiosteal flap with a Howarth periosteal elevator[(1)].

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LINGUAL NERVE

ANATOMY OF LINGUAL NERVE: The lingual nerve branches from the third division of the trigeminal nerve after it exits the foramen ovale. It carries with it taste fibers from the chorda tympani that supply the anterior two thirds of the tongue. The lingual nerve may be flat, oval or round and varies in size from 1.53 mm to 4.5 mm. The nerve is either monofascicular or oligofascicular in structure at the pterygomandibular space, making it susceptible to injury by injection in this area. It runs deep to the lateral pterygoid muscle parallel to the inferior alveolar nerve, lying anterior and medial to it. It then runs between the internal and medial pterygoid muscles and passes obliquely over the superior pharyngeal constrictor and styloglossus muscles before approaching the side of the tongue. The nerve courses submucosally in contact with the periosteum, covering the lingual or medial wall of the third molar socket. It crosses the Wharton duct and then loops back to cross it again. It may run below and behind the tooth before swerving superficially across the surface of the mylohyoid muscle. The lingual nerve morphologically differs from the inferior alveolar nerve. At the usual site of injury (adjacent to the lower third molar) the nerve is covered with only a thin layer of soft tissue and mucosa, rather than a bony canal. Consequently, if sectioned, the cut nerve ends retract apart and, if the adjacent soft tissue is also distorted, the nerve ends may become misaligned and constricted by scar tissue. Regeneration of axons across a gap will be less successful than if the nerve ends remain in apposition. In addition, the presence of a range of functionally distinct nerve fibre types in this nerve e.g. thermosensitive, gustatory, mechanosensitive, vasomotor and secretomotor may make successful regeneration of the axons back to the correct receptor/effector and location less likely.

INJURY TO LINGUAL NERVE: Robinson in 1971reported frequency of lingual nerve injuries during third molar removal, with 0.2–22% of patients reporting sensory disturbances in the early post-operative period and 0–2% a permanent disturbance[2]. There are several explanations for the wide range in the incidence of lingual nerve injury. First, the variation may reflect differences in the time interval between the tooth removal and the assessment of the sensory impairment; early assessments will report many transient sensory changes that recover completely and rapidly, and which would be missed if assessment takes place after a longer recovery period. Secondly, the incidence of nerve injury may depend upon whether the sensory deficit was established objectively by the clinician or subjectively by patient assessment. Finally, it may reflect differing surgical techniques; Mason and Blackbum[3,4] have stated that the raising and retraction of a lingual mucoperiosteal flap is associated with an increased frequency of lingual nerve damage. Two recent studies and a systematic review have concluded that raising and retracting a lingual periosteal flap is not necessary and can be best avoided[5,6,7]. Blackbum[3,4] in 1989 stated that Lingual nerve damage is particularly associated with deeply impacted teeth ,particularly if distal bone removal is required. The results of studies comparing the incidence of lingual nerve injury during surgery utilizing bone removal with burs or chisels are unclear [3,4]. A recently published study by Renton and McGurk stated that factors reflecting the surgical skill (i.e. lingual plate perforation) and the difficulty of the extraction were the strongest predictors of permanent and temperory lingual nerve injury.[8]

PREDISPOSING FACTORS

- NERVE LOCATION- The Close proximity of the lingualnerve to the cortex of the mandible may cause entrapment [9]. Variability in the lingual nerve anatomy associated with location of the third molar provides for higher risk of injury. The nerve may lie above the lingual plate or even on the alveolar crest [10,11,12]. It is emphasized that the clinician cannot use only the lingual plate as a reference to ensure lingual nerve protection, as often the nerve is in the soft tissue above the bone.
- HARMONAL CHANGES- Most orofacial trigeminal dysesthesia occurs in women usually in their 40s[13,14]. Sex-based differences have been seen in many pain disorders. The role of sex hormones and perpetuation of central sensitization is obviously important [15]. Coyle et al in 1996 stated that female rats that had been ovariectomized, there was a greater chance those with estrogen were more likely to develop allodynia after injury than those without estrogen.[16].Further understanding of central pain inhibition may help explain these phenomena. In a recent report by Pogra and Thamby, more females are affected than males by neuropathic pain.
- GENETICS-There is no strong evidence of genetic predisposition for nerve injury but the recovery and resultant pain may have a genetic base.

CLASSIFICATION OF NERVE INJURY: The consequences and recovery following nerve damage depends upon the severity of the injury and they are classified based on the classifications of nerve injury proposed by Seddon [19] and Sunderland. In 1943, Seddon classified nerve injury as:

(1) **Neuropraxia—**conduction blockage resulting from mild trauma, without axonal damage, and with resolution of sensory deficit within days to months;
(2) **Axonotmesis—**more severe injury, with preservation of the nerve sheath but afferent fiber degeneration, and incomplete sensory recovery; neuroma formation may occur, and the typical clinical presentation involves severe dysesthesia;
(3) **Neurotmesis—**most severe injury, with nerve severance and anesthesia in the nerve distribution, and no sensory recovery (especially if the nerve course is in soft tissue; if the nerve course is in bone, regeneration may occur) [17].

In 1951, Sunderland classified nerve injury based on the degree of tissue injury[18]. First-degree injury, there are 3 types, is similar to Seddon's neuropraxia.

Type 1 results from mild traction of nerve, nerve trunk manipulation, or mild compression and is thought to reflect transient ischemia. If blood flow is restored, nerve function usually returns to normal; with more prolonged ischemia and permanent injury may occur.

Type 2 injuries result from more prominent traction or compression that produces intrafascicular edema, decreased blood flow, and a conduction block. Recovery is variable.

Type 3 injuries result from severe nerve traction or compression causing segmental mechanical disruption of the myelin sheaths and demyelinization. Recovery is delayed and sensory loss may be permanent [19]. Second-, third-, and fourth-

degree injuries correspond with Seddon's classification of axonotmesis. The afferent or efferent fibers are damaged, but endoneurium, perineurium, and epineurium remain intact. There may be necessory for Surgical decompression, and recovery requires axonal regeneration. Third-degree injury occurs when the intrafascicular tissue components (axons and endoneurium) are damaged. Surgical reconstruction may be needed if there is poor clinical recovery. Fourth-degree injury implies fascicular disruption: all components are damaged and only epineurium remains intact. The prognosis is poor, and surgical reconstruction may be indicated. Fifth-degree injury implies nerve transsection. Surgical approximation may be required. MacKinnon and Dellon, in 1988, added sixth-degree injury to describe a variation wherein a combination of Sunderland's 5 degrees of injury coexist within the same nerve trunk [(20)]. If there is complete numbness that improves over time, this is indicative of first- or second-degree injury per the Sunderland classification. The presence of complete numbness initially does not indicate nerve severity.

SEQUELAE OF NERVE INJURY: Temporary block of nerve conduction (1st degree injury) is usually accompanied by some local thinning of the axons and segmental demyelination but the changes are reversible [(21)].Recovery occurs within a few days of surgical trauma but in case of segmental demyelination, recovery is slightly slower[(22)]. The wallerian degeneration occurs distal to the site of injury and extends centrally for few millimetres and this degeneration consists of disintegration of both the axon and myelin and phagocytosis by the macrophages and schwann cells and the proliferation of later within the endoneurial sheath forming bungner [(23,24)]. Central to the site of injury, chromatolytic changes occurs in the rest of the neuron which it may or may not recover. Regeneration of the axons that survive occurs after an initial delay, which includes a recovery period and the time needed for growth of the axon. There is then a delay during the regenerating fibres across the injured zone. The initial delay prior to regeneration, and the success of axons in crossing the injury site, depends upon the nature of the injury. Crush injuries will recover more rapidly than section injuries because the endoneurial sheaths usually remain intact and so the regenerating axons are guided back to the appropriate receptor type and at the appropriate location[(25)]. In contrast, after nerve section injuries, the regenerating axons enter the endoneurial sheaths of the distal stump, apparently at random, and may be guided to an inappropriate receptor at a new location. This may give rise to abnormalities in location and the sensation perceived in response to a particular stimulus. Recovery from crush injuries may take several months and may not be complete sometimes. Recovery from nerve section injuries will progress for at least a year and will never be fully complete.

METHODS USED TO MONITOR NERVE RECOVERY: Most cases of nerve damage during wisdom tooth removal are not identified at the time of third molar removal but in the post-operative period. It is essential to assess residual function to make a judgement if the nerve is likely to recover spontaneously or if the surgical intervention is require. If there is only partial sensory loss, it suggests a first degree injury. If there is a complete anaesthesia following injury, it is impossible to determine if it is caused by crushed or section injury. However, careful monitoring of sensory recovery over a three month period should distinguish between these different types of injury the most sensitive indicator of a sensory abnormality is the patient's own subjective report, as minor sensory disturbances may not be detected by testing.

Simple Sensory Testing [(26)]: In this test, the patient should be seated comfortably in a quiet room. Test should be undertaken with the patient eyes closed and the detection of a stimulus is indicated to the examiner by the patient raising a finger. The results of each test are then compared with the normal (uninjured) side. Ideally, the first sensory tests should be undertaken within two weeks of the injury in order to monitor changes. The equipment needed to perform some of these tests is not readily available and must be constructed by individual clinicians.

Light Touch Sensation [(26)]: Light touch is most commonly tested by gently applying a wisp of cotton wool to the skin or mucosa. However, it is difficult to apply this stimulus in a reproducible manner and the use of a cotton wool wisp on moist oral mucosa is difficult.

Greater consistency and reproducibility can be obtained using Von Frey hairs and the method of making these instruments with a standard force of 20 mN (2g) has been described.[27] Stimuli is applied randomly and the area of an aesthesia can be detected by moving outwards in small steps until the stimulus is felt.

Pin prick sensation [(26)]: Testing pin prick threshold is often performed using a dental probe or needle, but again reproducibility is poor. A simple device, described by Sunderland, overcomes these difficulty. A pin is attached to a spring, which allows the pressure of up to 150 mN (15 g). Initially, stimuli of up to 150 mN can be applied randomly over the test areas to determine whether any sensation is perceived or not.

Thus, the areas of an aesthesia can be detected. If sensation is present within the affected area on the injured side, then the pinprick sensation threshold is determined. For this test the pin is applied at steadily increasing pressures and the patient asked to indicate the point where the sensation becomes sharp rather than dull. The pin prick sensation threshold is noted for a series of randomly chosen areas on both the injured and the uninjured side.

Two Point Discrimination [(26)]: This test can quickly be performed with the pairs of blunt probes with different separations (2–20 mm). They are mounted around a disc .The probes are applied at a series of fixed sites chosen on the lips or tongue, depending on which has been damaged. The probes are drawn a few millimeter across the surface, at a constant pressure, and the patient is asked whether one or two points are felt. The minimum separation, that is consistently reported as two points, is termed the two point discrimination threshold value. This threshold varies in different regions of the mouth (2–4 mm on the tongue and lip, 8–10 mm on the skin over the lower border of the chin).

Taste Stimulation [(26)]: Lingual nerve injury will result in taste loss from the ipsilateral anterior segment of the tongue. Taste testing may not be undertaken routinely but it is easy and simple to perform. Cotton wool pledgets are soaked in 1M sodium chloride, 1M sucrose, 0.4M acetic acid or 0.1M quinine and are drawn 1–2 cm across the lateral border of the tongue and the patient asked to indicate whether they taste salt,

sweet, sour, bitter or no taste, before replacing the tongue in the mouth. Stimuli should be applied in random order, to each side of the tongue, and rinsing with tap water between tests is permitted.

TREATMENT STRATEGIES

NONSURGICAL

Corticosteroids –Immediately after injury, there may be reason to consider high-dose corticosteroids to reduce immune inflammatory reaction. It is common for neurosurgeons to prescribe corticosteroids following intracranial surgery. The use of an NMDA antagonist may also be beneficial. The therapy for trigeminal dysesthesia is aimed at reducing peripheral nociceptive inputs and simultaneously enhancing central nervous system pain inhibitory.

Topical Applications: The use of topical therapies are still under research. There is evidence that capsaicin applied regularly will result in desensitization and relief of pain relief. The recommended dosage is 5 times per day for 5 days, then 3 times per day for 3 weeks. If the patient cannot withstand the burning produced by the application, the addition of topical local anesthetic, either 4% lidocaine or EMLA, is useful. Clonidine can be applied to the hyperalgesic region by placing the subcutaneous delivery patch where it is most tender. Alternatively, the use of 4%gel can be compounded and delivered over a larger area. For local intraoral application, a neurosensory stent has been created. After an oral impression, an acrylic stent is manufactured to cover the painful site[(28)]. Topical clonazepam (0.5 to 1.0 mg 3 times per day) has been effective at reducing burning oral pain. Patients were instructed to suck on a tablet for 3 minutes (and then discard it) 3 times per day for at least 10 days [(27)]. Lidocaine infusion (200 mg over 1 hour) may be used therapeutically in various forms of neuropathic pain [(29,30)].

TRICYCLIC ANTIDEPRESSANTS: It is well documented that tricyclic antidepressants are effective in many pain problems. Solberg and Graff-Radford have studied the response of amitriptyline in traumatic neuralgia. It is noted that the effective range is 10 to 150 mg per day usually taken in a single dose at bed time. Many antidepressants may be used [(31).]

MEMBRANE STABILIZERS: These medications include the antiepileptic agents, lidocaine derivatives, and some muscle relaxants. They have been used in intermittent, sharp, electric like pains. The newer generation of medications that affect γ -aminobutyric acid appears to be effective in continuous as well as intermittent pain. These include gabapentin, topiramate, and zonisamide [(28).]

SURGICAL: The microsurgical techniques for nerve repair have been used for many years. When it comes to lingual nerve, there is littlest and ardized manner in assessing outcome, and the numbers studied are very limited. Repair may entail decompression, direct suture, or grafting. Micro vascular decompression is very effective in compression neuropathies. It is also suggested that there is an increase in fungiform papillae and pores over time. Taste is usually compensated for over time, and no known treatment is helpful [(29).]

LINGUAL FLAP AND NERVE PROTECTION: Use of instruments to protect the lingual nerve during lower third molar extraction is still debated. The use of a lingual flap retractor can provide efficient and greater operating safety, it is also thought to cause lingual nerve injury sometimes [(32)]. Although there are different types of lingual retractors such as Howarth, Ward, Meade, Hovell, Walters, and Rowe, there is no scientific database showing the efficiency of one instrument over another. However, a large and smooth rather than narrow and pointed active retractor surface has been suggested to be less traumatic during flap retraction. Some authors claimed that retractor placement can cause temporary nerve damage but also could decrease the incidence of permanent nerve injury as they ensure better access and visibility of the surgical area which ensures the safer use of the instruments [(33)]. Pichler and Beirne [(7)] stated that lingual retraction might cause temporary nerve damage, but it neither protects nor harms the lingual nerve permanently. Recent studies did not highlight any significant differences in permanent lingual nerve damage between the simple buccal approach and the combined approach (buccal approach plus lingual flap retraction) but they stated that the later was significantly associated with an increased risk of temporary damage. Avoiding lingual flap detachment during the surgical procedure can decrease the incidence of lingual nerve damage [(33)]. Lingual nerve protection through the lingual flap should be restricted to selected cases in which the presence of more unfavorable surgical variables predicts a high risk of nerve injury.

TOOTH SECTIONING AND OSTECTOMY IN LOWER THIRD MOLAR SURGERY: Tooth sectioning is considered as a significant risk factor for lingual nerve injury during lower third molar surgery[(34)]. But the studies related to it did not show any statistically relevant differences between the incidence of lingual nerve damage in surgeries involving tooth sectioning and that of surgeries in which it was not performed. During tooth sectioning, the lingual nerve can be involved by the use of surgical bur during bone guttering. Predisposing condition.s of lower third molar bone impaction includes pre-existing bone fenestrations and iatrogenic intraoperative perforation of the alveolar wall at the level of the lingual nerve. In such cases, subtotal lower third molar sectioning can be done followed by complete separation of fragments using an elevator or another hand instrument [(35)]. There by tooth sectioning can avoid lingual nerve injury by decreasing the extent of the ostectomy or even helps avoid it in some cases. But the literature lacked clear and comprehensive information on the number of cases in which ostectomy was actually performed and the incidence of nerve damage in cases in which it was or not performed. Blackburn and Batainch[(1)] claimed that ostectomy should be carried out only when a total view of the periradicular bone surface is possible and they also stated that ostectomy should not be blindly performed, especially at the lower third molar removal of distal and distolingual sites. The need for the removal of bone distal and distolingual to lower third molar should be done based on preoperative planning as they aid in limited retraction of lingual tissue thereby providing improved visibility of the surgical field and also helps in protecting the lingual nerve from the rotary instruments[(36)].

CONCLUSION

This review article emphasis that there is no significant differences between buccal approach and buccal approach combined with lingual flap retraction in lower third molar surgery in permanent nerve damage but the later was found to

be strongly associated with temporary nerve damage. The lingual split technique was statistically associated with an increased risk of temporary nerve damage when compared with the buccal approach with lingual flap retraction and only buccal approach, but did not show any differences in lingual nerve damage compared with the buccal approach with lingual flap retraction. Therefore, it seems preferable to avoid lingual flap elevation, except in selected cases in which the presence of more unfavorable surgical variables predicts a high risk of injury to lingual nerve. There seemed to be no statistically relevant difference between the incidence of lingual nerve damage in surgeries in which tooth sectioning was performed and those in which it was not. Removal of peri-radicular bone tissue, especially at the lingual or distolingual sites, was strongly associated with lingual nerve damage. Moreover, most of the studies done were observational and very limited sample was compared with the rareness of lingual nerve damage, so long-term randomized large-scale prospective studies are necessary to identify risk factors for lingual nerve injury.

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