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Review article

Failure of inferior alveolar nerve block in endodontics

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Abstract – Analgesia is essential for successful completion of modern dental procedures. Standard inferior alveolar nerve block (IANB) is the primary method used to achieve mandibular analgesia. Difficulty experienced in obtaining satisfactory analgesia after IANB, especially of an acutely inflamed mandibular molar, remains a common clinical problem. Even when a proper technique is employed, clinical studies show that IANB fails in approximately 30% to 45% of cases. The reasons for failure are not fully understood. Anatomical considerations and abnormal physiological responses in the presence of inflammation as explanations for IANB failure are discussed in this paper.

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Key words: endodontics; inferior alveolar nerve block; local anaesthetics; mandibular nerve block

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Weinstein et al. (1) reported in a survey of dental patients that about one of seven patients experiences pain during treatment. Kaufman et al. (2) in a survey of 93 general practitioners found that 90% of practitioners have had some anaesthetic difficulties during restorative work. Anaesthetic failure occurs in 13% of injections overall, with the greatest number of failures (88%) occurring with the inferior alveolar nerve block (IANB). Interestingly, as pointed out by Kaufman et al. (2), the rate of failure for individual dentists ranges from 0% to 48.6%. The results of these surveys generally support the accepted fact that it is more difficult to achieve analgesia after an IANB than after an infiltration method. In clinical studies, overall failure rates of IANB for healthy lower molars have ranged from 15% to 35% (3–5). The results are assessed according to the method described by Dobbs & De Vier (6); failure is attributed to the cases where analgesia is not achieved (Grade A). Clinical studies using maximum output from the electric pulp tester as a criterion show even higher IANB failure rates. Vreeland et al. (7), evaluating the anaesthetic efficacy of IANB with different volumes and different concentrations of lidocaine on healthy lower human molars, reported 37%–47% failure. In a study by Childers et al. (8), including 40 subjects, anaesthetic failure was observed in 37% for the first molar and in 27% for the second molar.

Failure to achieve analgesia after IANB in mandibular molar teeth which clinically manifest pulpitis has been evaluated in only a few clinical studies. Cohen et al. (9) reported anaesthetic failure in 45% of cases. In a study by Dreven et al. (10), anaesthesia of teeth which clinically manifested pulpitis could not be attained in 27% of the cases, despite IANB supplemented by periodontal ligament injection.

Survey studies, in contrast to clinical studies, show lower rate and higher variability of failure. The higher success rate reported in survey studies may be due to the subjective interpretation of some patients' responses, i.e., anaesthetic failure may have been interpreted as nonanaesthetic related discomfort. In addition, lack of blind evaluation in these studies may have accounted for the variability.

Reasons for inadequate analgesia after IANB

Berns & Sadove (11), using radiopaque dyes and radiographs of needle placement, determined that the closer the anaesthetic solution is deposited to the in-

ferior alveolar nerve, the more successful is the nerve block. However, they found that 25% of accurately placed needles resulted in ineffective pulpal analgesia. This is supported by the report that no association was found between years of experience and failure percentage (2).

Anxiety and fear

The traditional conceptualisation of pain as a strictly neurophysiologic event triggered by current or impending tissue damage seems to be inadequate. There are many dental and nondental reports on the relationship between anxiety and other forms of arousal, and pain. Bronzo & Powers (12) found that anxiety introduced experimentally induces pain. Schumacher & Velden (13) showed that subjects under high anxiety conditions are less able to discriminate levels of weak pain stimuli. A positive relationship between anxiety and pain is present during dental treatment. It was shown that anxiety directly lowers the pain threshold (14).

Anxiety and fear may cause a patient to complain of pain even when anaesthesia is apparent. Psychological factors, such as expectation and anticipation, communication and control, personality, culture training and suggestion influence pain perception.

Nevertheless, approaching the anxious patient with a stress protocol may still fail. In these cases, anxiety and fear may be superposed by other factors involved in IANB failure.

Accessory innervation

Accessory innervation has long been given as a reason for inadequate dental anaesthesia (15, 16). The mandibular hard and soft tissues are supplied by a plexus of nerves. The main nerve in this plexus is the mylohyoid nerve, but lingual, buccal, inferior dental nerves, cutaneus coli, and the superior and inferior laryngeal branches of the vagus nerve also occasionally innervate the teeth (15–17). This plexus, with its many communications, may allow sensation even if the primary inferior alveolar nerve is blocked. A block of other nerves in this plexus may be required to render the mandibular teeth insensitive.

The mylohyoid nerve has been implicated as providing accessory innervation to mandibular molars. The probability that the mylohyoid nerve innervates the mandibular teeth varies from 10%–20% (16). Injection beneath the mylohyoid muscle has no effect on the vitalometric values of the lateral incisors, canines and premolars (16). Anaesthesia of the first molar was achieved in 21% of the subjects. It is possible that anaesthesia of the first molar is not attained through the real innervation from the mylohyoid nerve because a direct infiltration effect to the first molar can occur.

Gow-Gates (17) has introduced an alternative technique for a mandibular nerve block using a more lateral approach at a higher level than the conventional IANB. The injection would supposedly have anaesthetised the nerve closer to the site of its exit from the cranium and would block any nerves which branched at a higher level after leaving the foramen ovale, resulting in profound mandibular analgesia. The branched nerves would not be anaesthetised by the more inferior approach of the conventional technique. Using this technique, success rates of 92%–100% have been reported, compared to the standard technique achieving success rates of 65%–86% (3–5). However, Montagnese et al. (18), in a comparative study of the conventional technique *vs* the Gow-Gates injection, found no difference in analgesic effect between the two methods.

This success rate exceeds the incidence of accessory or cross-innervation suggested in the literature (16). Therefore, accessory innervation of mandibular molars can only partly explain the failure rate of IANB.

Anaesthetic solution

Type of anaesthetic agent

Lidocaine is the most frequently studied local anaesthetic, but with the increasing choice of newly developed anaesthetics, there is much to be learned about which anaesthetic is the most effective. For dental use, lidocaine is always combined with a vasoconstrictor. Mepivacaine, a more recent, anaesthetic agent, does not require the addition of a vasoconstrictor when used in dental anaesthesia. Cohen et al. (9) showed that 3% mepivacaine is as effective as 2% lidocaine with 1:100 000 epinephrine in achieving pulpal analgesia with the IANB. Considering the possible systemic effect of a vasoconstrictor and the acclaimed effectiveness of mepivacaine, there are many circumstances in which mepivacaine might be preferable (9).

Recently, Hinkley et al. (19) compared 4% prilocaine (1:200 000 epinephrine) and 2% mepivacaine (1:20 000 levonordefrine) to 2% lidocaine (1:100 000 epinephrine) for IANB. Using a pulp tester to determine anaesthesia, they found no significant differences in the effect of the three solutions. Anesthesia obtained with 4% prilocaine and 3% mepivacaine was compared with 2% lidocaine (1:100 000 epinephrine) for IANB in healthy lower molars (20). Anaesthetic success occurred in 43% to 63% of the molars. No statistically significant differences in onset, success, or failure were found among the solutions. The effectiveness of the latest local anaesthetic agent, articainhydrochloride, is poorly documented.

Despite the increasing choice of newly developed anaesthetic agents, the failure rate seems to be unchanged.

Concentration of anaesthetic agent

One of the earliest clinical investigations designed to establish the minimum effective concentration for dental use was performed with lidocaine (21). Swedish workers electrically stimulated healthy maxillary incisors, selected as free from caries or restorations, before and after the infiltration of various concentrations of lidocaine solutions. In this way, they identified that a 2% solution is necessary to induce anaesthesia with almost complete success. They suggested that their results are applicable to infiltration injections. Vreeland et al. (7) showed no significant difference in failure rate when lidocaine is doubled in concentration (2%–4%). This study does not support a higher concentration of lidocaine for achieving pulpal analgesia.

Concentration of vasoconstrictor in anaesthetic solution

The degree of anaesthesia obtained with different concentrations of vasoconstrictors in anaesthetic solutions has been tested in several studies. Fink (22) demonstrated that pulpal anaesthesia is positively related to epinephrine dose. He used an infraorbital nerve block model in rats and an epinephrine concentration varying from 1:50 000 to 1:400 000. In humans, Knoll-Kohler & Förtsch (23), reported success of anaesthesia proportional to the epinephrine concentration in a concentration range of 1:100 000 to 1:200 000.

On the other hand, the results of this study failed to show a dose-dependent effect of epinephrine on anaesthesia when lidocaine with 1:50 000 and 1:100 000 epinephrine are evaluated. Similarly, Handler & Albers (24) could not demonstrate a relationship between the concentration of the vasoconstrictor in a 2% lidocaine solution and reliability of pulpal anaesthesia. It is suggested that solutions of 2% lidocaine with different doses of epinephrine (1:50 000; 1:80 000; 1:100 000) can be considered equivalent in IANB of 50 min duration (25).

Volume of anaesthetic solution

Franz & Perry (26) observed that small myelinated axons of cat saphenus nerve are blocked more quickly than large myelinated axons. They indicated that differential rates of blocking among myelinated axons by local anaesthetics (procaine) are attributable to differences in the critical length of axons that must be exposed to blocking concentration rather than to differences in minimal concentrations necessary to block axons of different sizes. To induce blockade of a whole nerve it is necessary to apply the anaesthetic agent along a distance of no less than three internodal lengths of the largest fibres. The longest internodal spans in the human inferior dental nerve have been found to be 1.8 mm (27). Thus, not less than 6 mm of nerve would need to be exposed to local anaesthetic solution to induce an absolute block. This could account for some failures of analgesia when small volumes are administered.

When isolated nerve studies are carried out in the laboratory, it has been shown that increasing the volume of a dilute anaesthetic fluid increased the efficiency. However, once the effective volume is attained no further benefit can be achieved. The volume of a 2% lidocaine solution with adrenaline necessary to induce a satisfactory inferior dental block has been said to be 2.0 ml (28). Clinical studies support 1.0 ml as the effective volume below which consistent success cannot be expected (27). This correlates well with the measurement of the internodal lengths.

Central core theory

The central core theory states that the nerve fibres toward the centre of the nerve innervate the furthest targets and are the last to be anaesthetised (29). In some cases the anaesthetic solution may not completely diffuse into the nerve to produce an adequate nerve block in all teeth (29). In addition, exposure to radioactively labelled extracellular fluid indicators has induced sparser and more uneven labelling around densely packed myelinated nerve fibres than around adjacent connective tissue. An irregular distribution of extracellular markers throughout the endoneurium is also evident in immature mouse nerve exposed to protein tracers for 24 hours (30). Therefore, a nonuniform distribution of local anaesthetic molecules around all axons within the early minutes of exposure would appear likely. As suggested by Vreeland et al. (7) the central core theory may explain why onset of analgesia is faster in molars than in anterior teeth. However, it cannot provide an explanation for the failure of IANB.

Inflammation-related conditions in the nerve trunk

For the endodontist, anaesthesia of symptomatic teeth has been reported as more challenging than that of asymptomatic teeth (31). Acute or exacerbating pulpitis has been suggested as the reason for this difference (32).

Inflammation modifies the activity of peripheral sensory nerves. It has been shown in rats that the response threshold is lowered (primary hyperalgesia) in the presence of inflammation (33). This is said to be due to an alteration in the threshold of the sensory receptors (34). If the peripheral sensitivity somehow alters the mechanism of impulse generation in the sensory nerves, then this may modify the efficiency of an anaesthetic agent (29).

Inflammation takes place in the pulp chamber. How can it affect the nerve cell membrane some 200 mm distal to where the anaesthetic is given, to such

an extent that it permits impulse conduction across an anaesthetised portion of the fibre (31)?

Najjar (32) demonstrated general changes along the inflamed nerve in rabbits, distant from the inflammatory site, which he suggested could explain failure to achieve anaesthesia in the presence of inflammation. Wallace et al. (31) postulated that nerves in inflamed tissue have altered resting potentials and excitability thresholds and that these changes are not restricted to the inflamed pulp itself, but affect the entire neuron cell membrane in every involved fibre. The nature of these changes is such that the reduction in ion flow and in action potential created by local anaesthetic agent is not sufficient to prevent impulse transmission since the lowered excitability threshold allows transmission, even under conditions of anaesthesia.

Kimberly & Byers (35) reported that neuropeptides such as CGRP are elevated in trunk axons of trigeminal nerves that innervate inflamed tissue. They suggested that those fibers might have an altered capacity for anaesthesia due to chemical changes extending throughout the affected nerve fibers. This suggestion is supported by studies showing rapid alterations in the levels of neuropeptide mRNAs in dorsal root ganglia and spinal cord neurons within hours of onset of inflammation (36).

Conclusions

Analgesia is essential for successful completion of modern dental procedures. Standard inferior alveolar nerve block is the primary method used to achieve mandibular analgesia. Difficulty experienced in obtaining satisfactory analgesia after IANB, especially of an acutely inflamed mandibular molar, remains a common clinical problem. Even when proper techniques are used, clinical studies show that IANB fails in approximately 30% to 45% of healthy molars. The reasons for failure are not fully understood, but may be summarized as follows:

1. Anxiety directly lowers the pain threshold. Psychological factors, such as personality, expectation and anticipation, influence pain perception.
2. The failure rate of IANB exceeds the incidence of accessory innervation of the mandibular molars.
3. There seems to be no significant difference in IANB failure rates among anaesthetic agents currently used in endodontics. Increasing the concentration or volume of an anaesthetic solution above the standard does not decrease the failure rate of IANB.
4. Neurons and their axons innervating an inflamed tissue have altered resting potentials and excitability thresholds. Chemical changes extending throughout the affected nerve fibres may alter their capacity to be anaesthetised.

All the above-mentioned reasons can only partly explain the failure rate of IANB. Further research, especially in the field of inflammation-related changes in the nerve trunk and development of new anaesthetic agents, is needed to improve the effectiveness of IANB

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