FACTS & FICTIONS ABOUT LOCAL ANAESTHETIC FAILURES – A LITERATURE REVIEW & SUGGESTED PROTOCOL TO COUNTERACT

Review Article

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ABSTRACT

The goal of today's modern dental practise is painless procedure however local anaesthetic failure is an unavoidable aspect of dental practice. A number of factors contribute to this, which may be related to either the patient or the operator. Patient-dependent factors may be anatomical, pathological or psychological. This paper emphasise on the failure of inferior alveolar nerve block and modalities to overcome the failure.

KEYWORDS: Inferior alveolar nerve block, causes of failure, effect of operator's experience

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INTRODUCTION:

The inferior alveolar nerve (IANB) block is the most frequently used technique for mandibular treatment[1]. Difficulty experienced in obtaining satisfactoryanesthesia after (IANB), remains a common clinical problem resulting in highest percentile failure. Following are different factors contribute to anaesthetic failure.[2-5]

1. Operator dependent factors

A) Choice of solution

B) Choice of technique

2. Patient dependent factors

A) Anatomical

B) Pathological

C) Psychological

1. Operator dependent factors

A) Choice of solution

a) Type of anesthetic 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; however Mepivacaine, a more recent, anaesthetic agent, does not require the addition of a vasoconstrictor when used in dental anaesthesia. Cohen et al.[6] 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 was compared with 2% lidocaine (1:100 000 epinephrine) for IANB in healthy lower molars[7]. 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, articain-hydrochloride, is poorly documented. Despite the increasing choice of newly developed anaesthetic agents, the failure rate seems to be unchanged.

b) Concentration of anaesthetic agent

One of the earliest clinical investigations designed to establish the minimum effective concentration for dental use was performed with lidocaine[8]. 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[4] that 2% solution is necessary to induce anaesthesia with almost complete success. They suggested that their results are applicable to infiltration injections. Vreeland et al.[9] 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 analgesia.

c) 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 [10] demonstrated that anaesthesia is positively related

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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-Kohier&Fortsch[11], 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 1:50000 and 1:100 000 epinephrine are evaluated. Similarly, Handler & Albers [12] could not demonstrate a relationship between the concentrations of the vasoconstrictor in a 2% lidocaine solution and reliability of 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 [13]

d) Volume of anaesthetic solution

Franz & Perry [14] 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 [5]

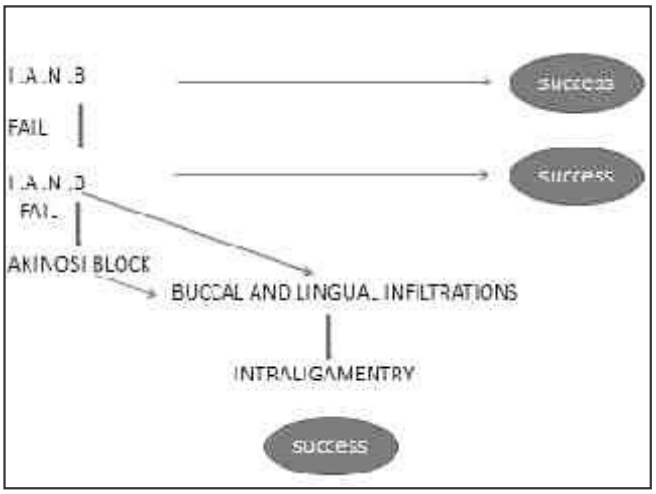
whole nerve it is necessary to apply the anaesthetic agent along a distance of no less than three intermodal lengths of the largest fibres. The longest intermodal spans in the human inferior dental nerve have been found to be 1.8 mm [15]. Thus, not less than 6 mm of nerve would need to be exposed to local anaesthetic

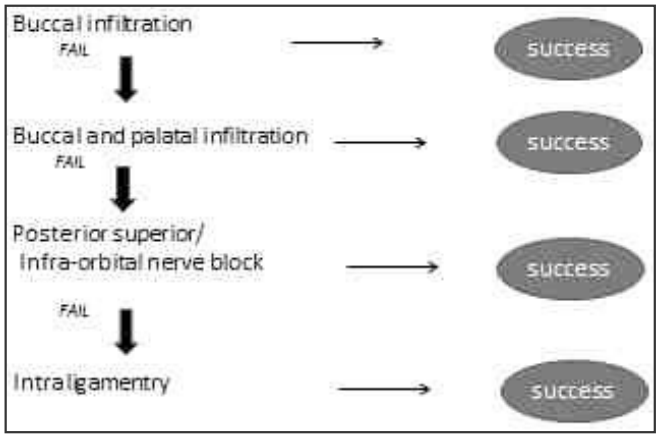
B) Choice of technique

The most likely defect in technique is faulty needle placement. Failure to aspirate before injection, which could lead to intravascular deposition of solution might also lead to failure of anaesthesia although this has never been proven. Success may be related to the speed at which the solution is deposited. It is easy to imagine the anaesthetic being directed away from a nerve trunk during forceful injection. There is evidence in the surgical literature that the success of some techniques is increased with slower injection speeds.[16] As far as conventional methods of local anaesthesia are concerned poor technique usually relates to mandibular anaesthesia, specifically failed inferior alveolar nerve block injections.The success rate for inferior alveolar block injections with lignocaine and adrenaline is more than 90%.[17-18] Practitioners who regularly fail with this method should reassess their technique. The best way to achieve success with the inferior alveolar nerve block is to use the direct technique where the dentist places the thumb intra-orally at the deepest concavity of the anterior ascending ramus and the index finger at the same height extra-orally on the posterior aspect of the ramus. The puncture point is half-way between the mid-point of the thumb nail and the pterygomandibular raphe and the needle is advanced through this point being delivered parallel to the occlusal plane from the premolar teeth of the opposite side. The properbony end point is reached between 15 and 25 mm of penetration. The common causes of failure are touching bone too soon on the anterior ascending ramus (rectified by swinging the syringe across the mandibular teeth on the same side, advancing [6]

1 cm and then returning to the original angle of approach) or injecting inferior to the mandibular foramen (countered by injecting at a higher level).

In most cases the dentist who experiences the odd failure rectifies the problem with a repeat injection, perhaps at a slightly higher level. An orthopantomogrammay help in locating the position of the mandibular foramen. In those cases where a second injection has not overcome the failure, an alternative approach to the inferior alveolar nerve should be considered.





2. Patient dependent factors

A) Anatomical

a) Accessory Innervations

Judging from clinical and anatomical studies,[19-20] the mylohyoid nerve is the accessory nerve most often cited as a cause for failure with mandibular anesthesia. Clark et al compared the inferior alveolar nerve block alone to a combination injection of the inferior alveolar nerve block plus the mylohyoid nerve block, which was aided by the use of a peripheral nerve stimulator. The investigators found that the mylohyoid injection did not significantly enhance pulpal anesthesia of the inferior alveolar nerve block. Therefore, the result of the study does not lend [7]

much credibility to the notion that the mylohyoid nerve is a major factor in failure with the inferior alveolar nerve block.

ACCESSORY NERVE SUPPLY TO THE TEETH-

TOOTH	MAIN SUPPLY	ACCESSORY SUPPLY	ACCESSORY SUPPLY COUNTERED BY
MAXILLARY	SUPERIOR ALVEOLAR NERVE	GRETER PALATINE	PALATAL BLOCK
MANDIBULAR	INFERIOR ALVEOLAR NERVE	LONG BUCCAL NERVE	LONG BUCCAL BLOCK
		LINGUAL NERVE	LINGUAL NERVE BLOCK
		MYLOHYOID NERVE	HIGH BLOCK
		AURICULO TEMPORAL NERVE	HIGH BLOCK
		UPPAR CERVICAL NERVES	BUCCAL AND LINGUAL INFILTRATION

b) Cross Innervation

Cross innervation from the contralateral inferior alveolar nerve has been implicated in failure to achieve anesthesia in anterior teeth after an inferior alveolar injection. Experimentally, cross innervation occurs in incisors, but plays a very small role in failure with the inferior alveolar nerve block.

c) Individual variations in the position of nerves and foramina

The foramina of importance in regional block anaesthesia in dentistry do not have a consistent location between patients. Many of the methods described above to surmount poor technique will overcome any problems resulting from anatomical variations. Available radiographs may be helpful in

anticipating this situation.

d) Morphological variations of mandible

Depending upon the shape of mandibular parabola position of the mandibular foramina varies significantly. If the mandible is v in shape as in vertical growers the foramina is posteriorly placed thus, sulcus mandibularis which is target area for inferior alveolar nerve block is located posteriorly so the direction and depth of needle insertion should be modified; direction of insertion should be changed to more posteriorly i.e. rather than inserting needle from opposite side premolar we have to insert it from opposite side molar and more depth than normal.

If mandible is 'U' shape as in horizontal growers the position of sulcus mandibularis is more anteriorly placed so direction of insertion should be changed to more anteriorly i.e. rather than inserting needle from opposite side premolar we have to insert it from opposite side canine-premolar region to a lesser depth than normal.

B) Pathological

a) Inflammation

It is apparent to all practitioners that area with inflammation can be difficult to anaesthetise. A number of suggestions have been proposed to explain this finding. The classic explanation for this is that the low tissue pH in areas of inflammation affects the activity of the local anaesthetic solution by decreasing the concentration of the un-ionised (lipophilic) fraction which diffuses through nerve sheaths. Similarly areas of inflammation have an increased blood supply due to vasodilatation and this might increase anaesthetic 'wash-out'. However, these answers do not explain the failure of regional block techniques where the solution may be deposited 4 or 5 cm from the area of inflammation. The most plausible explanation is that inflammation makes nerves hyperalgesic.[19] Minimal stimulation results in conduction. However, no tooth is resistant to local anaesthesia. The practitioner therefore has to decide on the maximum volume of local anaesthetic he is willing to inject for that patient and be prepared to use up to that maximum to anaesthetise that tooth. This may mean limiting treatment to only one tooth but if it takes the maximum safe dose — so be it. On no account should the predetermined safe maximum dose be exceeded. In healthy patients there is usually sufficient room for manoeuvre to administer a dose sufficient to halt conduction in the tooth without producing generalised central nervous system effects. The use of higher concentrations of local anaesthetic solutions (such as 5% lignocaine[20]), although effective, is not a viable option in practice. The answer is to inject more solution. This does not have to be at the same site, eg the combination of infiltration and regional block anaesthesia can be used in the maxilla (eg infiltration at the apex of an upper lateral incisor plus an infraorbital nerve block). This can be supplemented with intraligamentary or intra-osseous injections if required.

Anomalous anatomical variants and anatomical relations constitute the principal cause of inferior alveolar nerve anaesthesia failure. A double or bifid inferior alveolar nerve represents a [10] possible cause of failure in inferior alveolar nerve block.[21] In 0.4% of cases the inferior alveolar nerve presents two or eventhree trajectories through accessory foramina containingsmall sensory nerve fibres.Some patients, particularly those of advanced age, present an increased bone density in the mandibular teeth, thus leading to deficient anaesthesia when using periapical in-filtration techniques.[22] The mylohyoid nerve may possess a sensory component, thereby providing accessory innervation and causing inferior alveolar nerve block failure.[23] Contra lateral innervation of the anterior teeth can cause anesthetic failure in upper jaw and mandible.[24] Inflammation is also a cause of anesthetic failure, particularly in situations of pulpitis or apical periodontitis.[25]Anxious patients pose a challenge for dental treatment. Anxiety and fear can cause a patient to refer pain once anesthesia has been achieved. Early identification of this problem, a meticulous technique, and sedation can help in such situations. This problem can be resolved by discussing with the patient his or her fear of injections.[24-25] The orientation of the needle bevel (away or toward the mandibular ramus) for an inferior alveolar nerve block did not affect anesthetic success or failure.[26]

b) Factors precluding access

Factors which can preclude access include trismus (because of a number of causes) and anatomical changes because of trauma or surgery. Trismus is

the most likely factor in practice and this is often because of an infective cause. Buccal infiltrations in the maxilla are still possible with the mouthclosed. A way to anaesthetise the palatal tissuesin the patient with trismus is to inject while advancing a needle toward the palate through the mesial and distal gingival papillae from the buccal side.

C) Psychological

There are undoubtedly patients who do not do well with local anaesthesia but in whom the local anaesthetic appears to have been effective. This may be because of fear and apprehension. In such patients the use of sedative techniques can be helpful as successful anaesthesia is easier to achieve in the relaxed patient.[27] Benzodiazepines offer the added bonus of reducing local anaesthetic toxicity which is useful when multiple injections are being administered.

Conclusions :

Failed local anaesthesia is a feature of dental practice. Most practitioners will experience it less often than they achieve success. The answers offered above, based on an understanding of the reasons for failure, should help overcome most cases encountered in practice.

SUMMARY

CAUSE FOR FAILURE		METHOED TO COUNTER
Operator dependent	Choice of solution	As per surgical prerequisite
	Choice of technique	As per surgeons dexterity
Patient dependent	Anatomical	Comprehensive knowledge of applied surgical neuroanatomy
	Pathological	Counteract pH changes
	Psychological	Stress reduction protocol

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