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Facial Nerve Injury Complications During Inferior Alveolar Nerve Block: A Review Article

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Abstract: ***The Inferior Alveolar Nerve Block (IANB) is the most utilized regional anesthetic technique in dentistry, but it carries a rare risk of transient Facial Nerve (CN VII) palsy, reported at approximately 0.3\%.This motor complication manifests as unilateral facial weakness, drooping of the corner of the mouth, and critical inability to close the ipsilateral eye (lagophthalmos). The primary etiology is a technical error: the inadvertent deposition of local anesthetic directly into the parotid gland capsule, a consequence of overly deep or posterior needle placement . While immediate palsy is a chemical conduction block, delayed presentation is hypothesized to stem from localized ischemic neuritis and secondary edema. Management is focused on immediate patient reassurance, mandatory ocular protection (lubricants and patching) to prevent corneal damage , and the use of systemic corticosteroids to mitigate inflammation . The prognosis is excellent, with complete functional recovery commonly observed within eight weeks. Prevention relies on rigorous adherence to injection protocol, including aspiration, slow injection, and strict depth control to prevent posterior needle penetration .***

Keywords: Inferior Alveolar Nerve Block (IANB),DentalAnesthesiaComplications,Facial Nerve Palsy,House-Brackmann Grading System (HBGS)

I. INTRODUCTION

The Inferior Alveolar Nerve Block (IANB) is the most widely utilized regional anesthetic technique in modern dental practice, essential for various surgical and restorative procedures involving mandibular teeth. Despite its efficacy, IANB carries a rare risk of transient Facial Nerve (CN VII) palsy, with a reported incidence of approximately 0.3%. Unlike sensory nerve injuries to the Inferior Alveolar or Lingual nerves—which can occur in up to 98% of cases in certain surgical settings—CN VII palsy is a motor complication that manifests as unilateral facial weakness, drooping of the mouth, and an inability to close the ipsilateral eye (lagophthalmos). The extreme rarity of this motor involvement suggests that its pathogenesis relies on a specific technical error: the unintended deposition of anesthetic into the parotid gland capsule, where the facial nerve is located.

II. MATERIALS AND METHODS

The diagnosis and assessment of IANB-induced facial nerve palsy rely on a detailed medical history and comprehensive clinical examination to exclude non-iatrogenic causes such as idiopathic Bell's palsy or Ramsay Hunt syndrome. Clinical evaluation is standardized using the House-Brackmann Facial Nerve Grading System (HBGS), which objectively monitors nerve function and recovery progress.

Grade	Dysfunction Level	Key clinical features
Grade I	Normal	Normal facial function in all areas
GradeII	Slight dysfunction	Slight weakness noticeable on close inspection;normal tone and symmetry at rest
Grade III	Moderate dysfunction	Obvious difference,but not dysfiguring; complete eye closure maintained with effort.
Grade IV	Moderate severe dysfunction	Obvious facial weakness; inability

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		to close eye completely without effort
Grade V	Severe dysfunction	Little to no ability to smile,frownor make expressions;only slight visible movement
Grade VI	Total paralaysis	No visible facial motion.

Management protocols involve a multidisciplinary approach focusing on acute protection, pharmacological intervention, and physical rehabilitation. Immediate care prioritizes ocular protection to prevent corneal damage (exposure keratitis) through the use of lubricants and eye patching. Systemic corticosteroids, such as a tapered regimen of prednisolone, are utilized to mitigate inflammation and nerve compression, particularly in delayed onset cases. Physical therapy exercises, including forehead, ocular, and lower facial muscle movements, are implemented to maintain muscle tone and prevent contracture during the recovery phase.

III. RESULTS

Prognosis for recovery from IANB-induced palsy is overwhelmingly favorable, with complete functional restoration commonly observed within eight weeks. Even in severe presentations initially classified as HBGS Grade VI (Total Paralysis), significant clinical improvement is typically achieved between the fifth and seventh months. Recovery is often slow and progressive; however, objective tracking via the HBGS frequently shows visible improvements by the four-week follow-up.

The distinction between immediate and delayed onset is clinically significant for results:

* Immediate Onset: Caused by a direct chemical conduction block (neurapraxia) within the parotid gland.
* Delayed Onset: Occurring approximately 24 hours post-injection, this is hypothesized to stem from a sympathetic reflex spasm of the vasa nervorum, leading to localized ischemic neuritis and secondary edema.

Regarding anesthetic choice, systematic reviews indicate no conclusive evidence that 4% articaine presents a greater risk of nerve damage than 2% lidocaine, suggesting that anatomical misplacement is the primary determinant of palsy.

IV. DISCUSSION

The primary mechanism for IANB-induced facial nerve palsy is the inadvertent deposition of local anesthetic directly into the parotid gland capsule. This occurs when a technical error in needle trajectory specifically overly posterior or excessively deep placement causes the needle tip to traverse the pterygomandibular space and penetrate the gland substance. The parotid gland occupies a position immediately posterior and lateral to the mandibular ramus, meaning it encases the extracranial segment of the facial nerve and its five terminal branches. The pathogenesis is categorized into two distinct temporal presentations:

Immediate Onset: Occurs within minutes due to a direct chemical conduction block (neurapraxia) of the facial nerve.

Delayed Onset: Manifests approximately 24 hours after administration. This is hypothesized to result from a sympathetic reflex spasm of the vasa nervorum, triggered by mechanical trauma or the anesthetic solution, leading to localized ischemic neuritis and secondary edema.

Regarding pharmacological factors, evidence suggests that the specific local anesthetic agent used does not significantly alter the risk profile. Systematic reviews indicate there is no conclusive evidence that 4% articaine carries a higher risk of nerve damage than 2% lidocaine. Instead, the primary determinant of this complication remains anatomical misplacement rather than inherent drug toxicity or the volume of the standard cartridge.

Clinical diagnosis relies on recognizing unilateral motor deficits, such as the inability to close the eye (lagophthalmos) and a drooping mouth corner, while the corneal reflex typically remains intact. To objectively track recovery, clinicians utilize the House-Brackmann Facial Nerve Grading System (HBGS), which scales dysfunction from Grade I (normal) to Grade VI (total paralysis).

Management protocols prioritize patient reassurance and the prevention of ocular damage. Because the eye cannot close, mandatory protection includes the use of artificial tears, ocular lubricants, and eye patching to prevent exposure keratitis and desiccation. Systemic corticosteroids are frequently employed to mitigate the inflammatory and ischemic components, particularly in delayed presentations. A common regimen involves a four-week taper of prednisolone starting at 60 mg daily. Furthermore, physical therapy exercises—such as frowning, puckering, and facial massage—are essential to maintain muscle tone and facilitate the return of complex movements.

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Ultimately, prevention is rooted in rigid adherence to injection technique. The most critical safety mechanism is ensuring the needle makes contact with the medial aspect of the mandibular ramus (bony resistance) at a depth of approximately 15 mm to 24 mm. If bony contact is not achieved, it indicates a posterior trajectory that risks parotid gland penetration.
Would you like me to draft a specific patient education handout based on these management protocols?

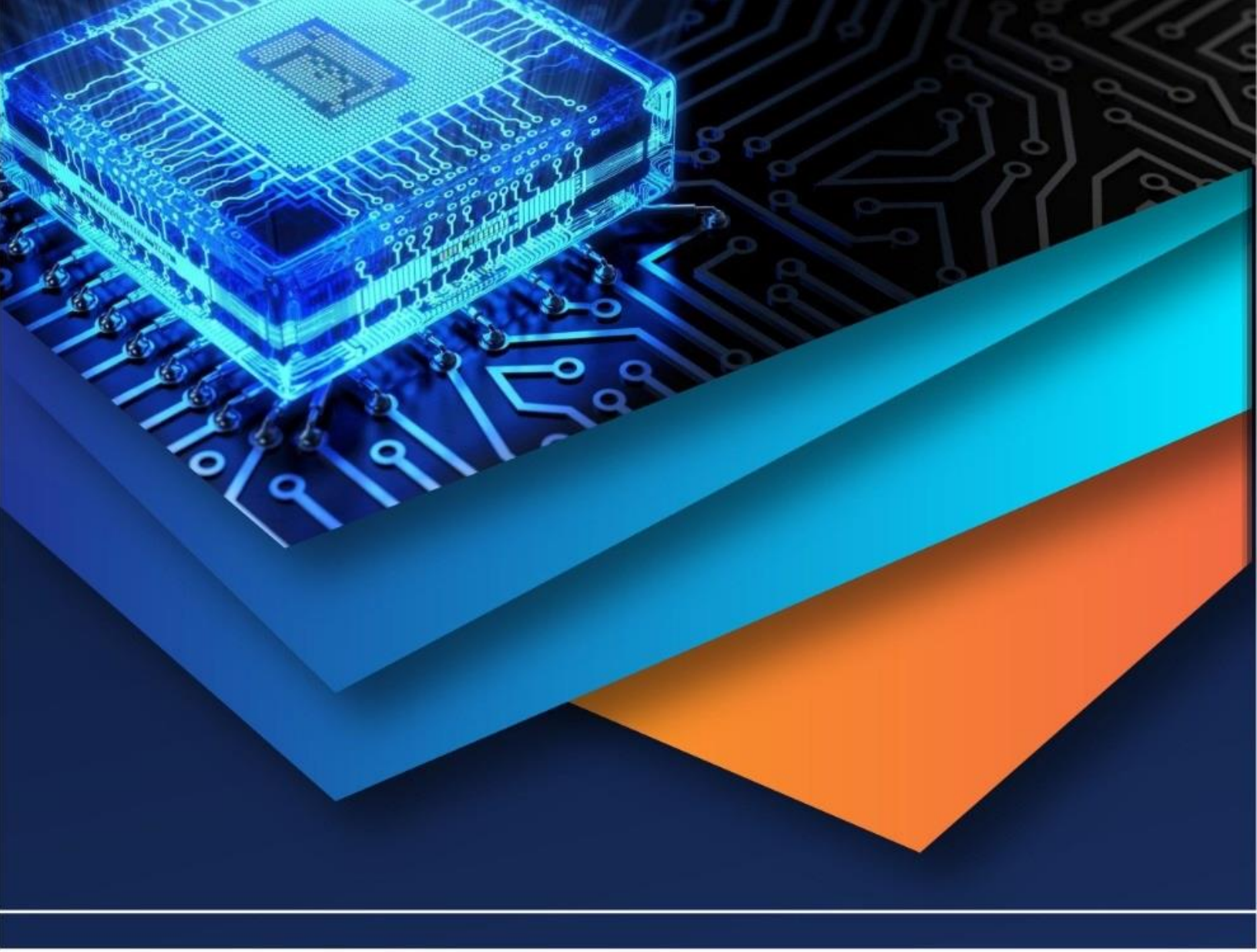
V. CONCLUSION

IANB-induced facial nerve palsy is a rare but significant iatrogenic event. Prevention is the single most effective strategy and relies exclusively on rigorous adherence to injection protocols, including mandatory aspiration, slow injection, and strict depth control to ensure the needle contacts bone (the medial aspect of the mandibular ramus). When complications occur, a combination of patient reassurance, mandatory ocular care, and corticosteroid therapy ensures high recovery rates. Future efforts should focus on defining standardized anatomical parameters and validating the ischemic neuritis hypothesis to further refine management protocols.

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