2nd Edition

Manual of Local Anesthesia in Dentistry

AP Chitre

JAYPEE

Manual of
Local Anesthesia
in DENTISTRY

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Second Edition

AP Chitre MDS

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Manual of Local Anesthesia in Dentistry

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Dedicated to
my parents

Prof. C. Bhasker Rao MDS FDSRCPS
Principal, SDM College of Sciences and Hospital
Director, Craniofacial Surgery and Research Center
Vice President, Dental Council of India

Foreword to the First Edition

It gives me great pleasure to write a foreword on the text *Manual of Local Anesthesia in Dentistry*. It is a common practice all over the globe to use dentist's name to quieten a child. This was largely due to the painful experiences one often underwent during dental treatment in the past. The advances in the knowledge of pain mechanisms, thorough understanding of pharmacology has resulted in the development of various strategies for pain control particularly in dentistry. Local anesthesia still remains the most favoured choice of pain control method. It is surprising that in a country like ours, with nearly 200 Dental Schools does not possess a single book on local anesthesia written by any experienced teacher or a clinician of Indian origin.

Professor AP Chitre, an experienced senior Maxillofacial Surgeon in the Country has undertaken the task of putting down his experiences and scripted this manual of local anesthesia. It appears, to be a comprehensive book covering all the aspects related to the subject and I am sure, not only the students but all the dentists will find it useful in day to day practice.

I have no hesitation to recommend this book for both undergraduate students and practicing dental professionals. I congratulate Professor Chitre for this wonderful work, which will benefit the dental profession.

Prof. C. Bhasker Rao

Preface to the Second Edition

It gives me great pleasure and sense of satisfaction to present to you the second edition of my book, *Manual of Local Anesthesia in Dentistry*. This has been possible due to the good response given by the students and teaching colleagues. I thank them all.

The new edition has been updated by adding seven new chapters, keeping in mind the present scenario and the curricula of all Indian universities. The new chapters are on Theories of Pain Perception, Methods of Pain Control, Medical Evaluation, Infection Control, Preanesthetic Medications, Newer Injection Techniques, and Management of Dental Clinic Waste.

Some of the old chapters have been revised and upgraded. The notable additions are the newer local anesthetic agents. The maximum recommended doses have been dealt with in details. A lot of new diagrams related with Local Anesthetic Agents, Armamentarium, Injection Techniques have also been added. The chapter on HIV and Guidelines for Occupational Post-Exposure Prophylaxis (PEP) has been totally revised, which includes the latest guidelines given by National AIDS Control Organization (NACO), Ministry of Health and Family Welfare, Government of India.

I gratefully acknowledge the unstinted support and work put in by Dr MI Parkar, without whose efforts this edition would not have been so completed. His son Sa'ad Parkar who with his expert knowledge of computers helped in preparing the manuscripts and diagrams. I remain grateful to Dr Jyotsna Meshri, for guiding and helping to update the chapter on Systemic Complications of Anesthesia and their Management, Dr Amrita Haldar, for volunteering to be the charming model for injection techniques, and Professor (Dr) Seema Muzumdar, for her critical assessment of the First edition and the review. I also thank the publishers, Jaypee Brothers Medical Publishers (P) Ltd, for their support.

I am indebted to Padma Bhushan Brigadier Dr Anil Kohli, President Dental Council of India for releasing the First edition.

I hope that the new edition fulfils the requirements of students and the teaching staff, as an attempt is made to cover all the aspects of local anesthesia in dentistry.

AP Chitre

Preface to the First Edition

Dental treatment has been associated with pain from time immemorial. In fact, patients are scared to sit in the Dental Chair and will prefer to suffer until it becomes worse and unbearable. Dental pain is unique and worst compared to all other pains in the body. In Greek Mythology there is reference to the punishment given to Saint Appolonia who was branded as a witch. It was decreed that her teeth should be removed without anaesthesia only one at a time. Later, however, it was proved that she was not a witch and was granted sainthood. Performance of painless dental procedures is highly appreciated by the patients and is easily possible with the help of available local anaesthetic solutions and techniques. However, sound knowledge of surgical anatomy comprising of osseous landmarks, position of exit foramina of the 5th cranial nerve, attachment of muscles, innervation of soft and hard tissues is essential. Correct position of the patient and operator while performing the procedure gives successful results.

Detailed, relevant and meticulous history taking will forewarn the operator and will avoid a mishap. Adequate information about the drug used as local anaesthetic agent with relevance to proper dosage, contraindications and possible side reactions will help the Dental Surgeon to be well prepared for the eventualities. Last but not the least gentle tissue handling will avoid many untoward reactions and unpleasant situations.

Sound local anaesthesia is epitome of successful dental practice hence the need for this treatise for Dental students and practitioners.

AP Chitre

Acknowledgments

The completion of this book is the effort of teamwork. Notable to mention would be my two colleagues: Dr MI Parkar, Assistant Professor in Oral and Maxillofacial Surgery, at YMT's Dental College and Hospital, Kharghar, Navi Mumbai, who prepared and corrected the manuscripts after the basic format was prepared. He also collected the various references and prepared the bibliography for every chapter. Dr Raj Shekhar Halli, Associate Professor in Oral and Maxillofacial Surgery, at KLES's Institute of Dental Sciences, Belgaum, Karnataka, who prepared the various diagrams on computers showing the basic anatomy for various injection techniques. The photographer and the model who spent their time patiently. Last but not the least the Publishers, Jaypee Brothers Medical Publishers (P) Ltd, and their representative, Mr CS Gawde of Mumbai Branch, who approached me for writing the book and their subsequent cooperation.

I am deeply indebted to Professor C Bhasker Rao, Dean, SDM Dental College and Hospital, Dharwad, Karnataka; and Vice-President, Dental Council of India, for writing the foreword.

I thank all the concerned people who have made the publication of this book a reality.

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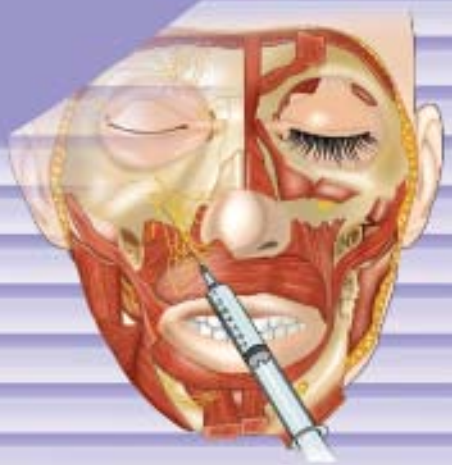


Human Race and Pain Relief

- History of Anesthesia

Chapter 1

History of Anesthesia

EARLY HISTORY OF ANESTHESIA

Human beings have suffered from painful injuries and diseases from time immemorial. Since then, he has constantly strived hard to find some method of relieving himself from pain.

Approximately 500,000 years ago, the early man, or the subman, Pithecanthropus erectus, struggled with nature, wild beasts, and his fellow submen for existence. Approximately, around 50,000 years ago, the Neanderthal man, appeared on the scene. He first lived alone like his ancestors. Later he joined with other individuals to form groups for mutual help in hunting and in battling with other groups for their women (Wells, 1921). These early men discovered that bruises and sprains felt better when the injured part was held in a cold stream or lake. Other painful wounds felt better when exposed to the sun's heat, and as a result, the radiant heat from fire and warm stones was probably used. If the injuries were severe, early man either suffered and died, or suffered and got well without help.

Approximately 25 to 40,000 years ago, the first true man developed. In each village, there evolved one person who developed greater skills in the treatment of injuries and diseases, the Cro-Magnon medicine man (Haggard, 1934). This medicine man frightened away the evil spirits who were blamed for the unfortunate victim's plight, treated his suffering parts by building smoky fires in which his various "therapeutic agents" were burnt, at the same time moaning mysterious incantations. The patient, flat on his back where the heavy smoke was the thickest, became semi-asphyxiated. This might be termed the first form of "inhalation anesthesia."

St. Appolonia was persecuted because of her christian faith, and in punishment for her refusal to renounce her religion, her teeth were knocked out, one at a time (Bruck, 1915). This is to indicate the severity of pain experienced while teeth are removed without anesthesia, and hence the severity of punishment. Of course, later, she was pardoned and granted sainthood.

Later the narcotic properties of certain plants were discovered. The benumbing or intoxication effect caused by juices of some plants were also utilized. Alcohol in an overdose, had been employed quite often, as an

agent to produce a state of unconsciousness in which pain of surgery was relieved. Hippocrates, about 450 BC, mentioned in his works that he produced perfect narcosis by having the patient inhale the vapor of bangue.

Galen, the Greek physician and philosopher, about 165 AD, used his anesthetic agent for extraction of teeth, used the application of "pirethrin" root and strong vinegar, from the action of which, the remaining teeth were preserved by covering them with a layer of wax. The Peruvian Indians chewed cocoa leaves and allowed the saliva laden with extract to drop on the injured part. A modified technique was practised by natives in Africa. The patient chewed the bark of enklovidi tree, swallowing the saliva to deaden pain.

The Egyptians practised compression and many surgeons from time to time used this practice of long-continued "Mechanical Compression" to produce numbness in the part to be operated upon. Hugh of Luca, in about 1250 AD, who was the teacher of Theodoric, used the "spongia somnifera" or sleeping ball to prevent pain.

During the 14th, 15th, and 16th centuries, it was the custom of some of the jailors to give criminals about to undergo torture, a compound of narcotics, which deaden their pain. Guy de Chauliac and Brunus (1350 AD) are the only surgeons who have used narcotics, mostly opium, and "sleeping sponge" to relieve pain during surgical procedures.

Dominique Jean Larry, Surgeon-in-chief, Napolean's Army, at the battle of Eylan, noticed when operating on wounded soldiers who were half-frozen from the intense cold that they felt very little pain. From this discovery arose "Refrigeration anesthesia". In 1786, Frederick Anton Mesmer used what he believed to be " magnetic emanations" to produce state of insensibility to surgical pain (Hollander, 1932).

The first truly modern experimental work was done in 1799, when Sir Humphrey Davy, published an account of his researches and experiments with various vapors and gases. Davy observed that the pain caused by an erupting "wisdom tooth" was relieved when he inhaled nitrous oxide (Davy, 1799). Henry Hill Hickman, who follows Davy in the history of anesthesia, seemed to have had a strong realization of clinical potentialities of gases as anesthetics. In 1824, he had performed many surgical operations on animals, which he had rendered unconscious with carbon dioxide (Hickman, 1930) (Table 1.1).

DISCOVERY OF INHALATION ANESTHESIA

The word "anesthesia" is derived from the Greek language. The words "an", means without; and "aisthetos" means sensation. The word was coined by Oliver Wendell Holmes in 1846. He, at the time of discovery of ether, wrote to Morton, and said that the state should be called "anesthesia".

Table 1.1: Historical events at a glance in chronological order

Year	*Pioneer*	*Agent*
1799	Sir Humphrey Davy	Nitrous oxide
1823	Henry Hill Hickman	Carbonic acid gas
1842	Crawford C Long	Ether inhalation
1844	Horace Wells	Nitrous oxide—first discovered, demonstrated, and proclaimed surgical anesthesia
1844	Gardener D Colton	Nitrous oxide (laughing gas)
1846	James Y Simpson	Chloroform
1846	WTG Morton	Manufactured ether— Gave first public demonstration of ether as an anesthetic
1851	Charles G Pravaz	Hypodermic syringe
1853	Alexander Wood	Hypodermic needle
1858	JB Francis	Electrogalvanic anesthesia
1861-62	M Fournier	Refrigeration anesthesia
1862	Schraff	Cocaine
1872	Castle AC	Reintroduced compression anesthesia
1884	William Halsted	Cocaine—Mandibular block
1890	Edwards C Briggs	Pressure anesthesia
1894	Carlson	Ethyl chloride spray
1896	Thiesing	Ethyl chloride spray
1896	Anon	Reintroduction of cataphoresis (electrogalvanic anesthesia)
1900	Legrand	Chemical method for hemostasis (mixture of gelatin with cocaine solution)
1904	Fourneau	Stovaine
1904	Hoffman	Elypin
1905	Alfred Einhorn	Procaine (Novocaine)
1905	Braun H	Introduced procaine into medical practice
1906	J Shepley Part	First paper on novocaine in local anesthesia in English language
1917	Harvey C Cook	Gave the idea of using local anesthetic solutions and drugs in cartridges
1933	Leo Winter	Ischemic agent—Cobefrin (Corbasil)
1936	Goldberg and Whitmore	Research on PABA esters (Monocaine)
1936	Mendel Nevin	Introduced into dental practice
1943	Nils Lofgren	Lidocaine
1952	Clinton and Laskowski	Propoxycaine
1953	Lofgren and Tegner	Prilocaine
1957	AF Ekenstam	Mepivacaine
1957	AF Ekenstam	Bupivacaine
1969	H Rusching et al	Articaine
1971	Takman	Etidocaine

The adjective will be anesthetic. Thus, he said, "state of anesthesia" or "the anesthetic state".

Crawford W Long, a physician, removed a tumor from the neck while the patient was under the effect of sulphuric ether, in 1842 (Long, 1849). A year subsequent to the discovery of ether, as an anesthetic, Sir James Y Simpson, an English Surgeon, demonstrated successfully the anesthetic properties of chloroform. This new anesthetic soon became popular in Europe and supplemented ether for a number of years.

Horace Wells, who was a practicing Dental Surgeon, closely observed the sufferings caused to patients while extracting teeth. He had been looking for some agent to alleviate suffering during dental surgical work. Horace Wells gave a considerable thought to the subject of pain relief during extraction of teeth. In an attempt to constantly enlarge his knowledge, he attended a lecture on chemical phenomena by Gardener, D Colton, a travelling chemist on December 10, 1844. Colton manufactured nitrous oxide, known as "laughing gas". Colton invited spectators from the audience to inhale the "laughing gas fumes". Wells, in the audience, observed that no signs of pain exhibited when the volunteers under the influence of the gas stumbled around the stage. Soon the idea of inhalation anesthesia was crystallized in the mind of Wells.

After the lecture, Wells talked to Colton and persuaded him to bring a bag of gas to his office next day. Wells had an aching tooth and intended to have his tooth removed painlessly by inhaling sufficient nitrous oxide. Wells, had the courage of his convictions, and persuaded Colton, despite objection from Colton, because of fear of fatal outcome, inhaled a bag of gas until he lost consciousness. Then his associate and former pupil, John Riggs, extracted the aching wisdom tooth painlessly. Colton administered the laughing gas to Wells. On regaining consciousness, Wells explained "a new era in tooth pulling." Thus anesthesia was born on 11th December, 1844. Later, Wells arranged a demonstration through a former pupil and partner, William TG Morton. Dr Morton was actually a dental student with Wells in Boston who later became associated with him in practice. Unfortunately, for some reason, the demonstration was not successful. Later, Wells, continued to use nitrous oxide in his practice and taught others to use it.

Morton received the idea of inhalation anesthesia from the demonstration given by Wells. He performed some experimental work with ether; and continued to use ether at the suggestion of Charles T Jackson, physician and chemist of Boston, in place of nitrous oxide. Finally, on 30 Sept, 1846, he extracted a firmly rooted bicuspid tooth in a patient, under the influence of a disguised ether compound. The patient became unconscious almost immediately. He recovered in a minute and knew nothing of what had been done for him.

Nitrous oxide, at first was discredited, however, proved to be a boon to humanity later. It stimulated further experiments with other agents and that led to the final discovery of other anesthetic agents.

Morton gave a public demonstration of practicability of anesthesia in the Massachusetts General Hospital in Boston, on Oct 16, 1846. Amongst the several prominent surgeons and physicians present was the Surgeon-in-Charge, John C Warren, to whom credit is due for giving Wells and Morton the opportunity to demonstrate publicly their anesthetic agents. The operation involved was the removal of a tumor from the left side of the jaw bone of a young man, performed by Warren. The exhibition of the anesthetic and the operation was a complete success. The patient after recovery from the slumber exclaimed, "I have felt no pain". Warren deeply impressed, turned to the audience and said, "Gentlemen, you have witnessed a miracle, this is no humbug" (Rice, 1858).

The American Dental Association (ADA), at its 4th annual meeting, in 1864, adopted a resolution wherein the credit and honor of introduction of anesthesia in USA, was given to Horace Wells of Hartford, (then deceased) (ADA 1864). This resolution was reaffirmed in 1872 (ADA 1872). The American Medical Association in 1870, in its 21st annual meeting, approved the following resolution, "the honor of the discovery of practical anesthesia is due to the late Dr Horace Wells, of Connecticut".

Charles G Pravaz, a French physician, in 1851, discovered hypodermic syringe. Alexander Wood, a physician from Edinburgh, in 1853, discovered hollow hypodermic needle for subcutaneous injections to relieve neuralgic pains. Both Pravaz and Wood and their contemporaries used the syringe and the needle for injection of morphine, opium and other drugs to alleviate pain and for surgical analgesia.

SUBSEQUENT ADVANCES

In 1856, JB Francis, discovered new use for electricity, the alleviation of pain during extraction of teeth. In 1858, JD White, a member of the Franklin Institute's Scientific Committee reported of having used electrogalvanic anesthesia for extraction of teeth; as first used by Francis, a Dental Surgeon. In 1859, he published several case reports of satisfactory use of electrogalvanic anesthesia for tooth extraction.

From 1850-1890, chloroform was used for dental, as well as major surgical operations, as a local anesthetic. In the mouth, cotton wool saturated with chloroform or ether was held to the buccal and lingual tissues with a small tray like device (Richardson, 1860). Refrigeration for local anesthesia was advocated by Fournier (Fournier, 1861-2), who exposed the part to be operated upon to spray of acetic acid and chloroform. He called this process "chloracetisation".

Narcotic spray was used for local anesthesia in 1867. However, the pain during and after the production of local anesthesia was greater than that due to extraction of tooth. Cases of marked sloughing of tissue were also observed.

Claude Bernard, In 1869, injected morphine as a preliminary narcotic before administration of ether or chloroform, the two general anesthetic agents which became popular at that time.

Castle, in 1872, claimed to have extracted teeth painlessly by "obtunding or benumbing the extremities of temporal nerves", for a period of 32 years. He used ice to the temples or had his "assistant press for one minute with persistent firmness into the fossa or hollow behind the ridge of temporal bone that forms the external bone circle orbit of the eye (Castle, 1872). This was reintroduction of so-called compression anesthesia practised centuries ago by the Egyptians.

Local anodynes had been in use to reduce the pain of drilling carious teeth.

Subsequently, a new anesthetic destined to be of major significance—cocaine was discovered. The local anesthetic effect of cocaine hydrochloride was discovered by Schraff in 1862, when he noted the local analgesic properties of this substance when it was placed on the tongue. Carl Koller, later, in 1884, discovered the local anesthetic properties of cocaine, and instilled the agent into the eye of frog and guinea pig.

In 1884, William S Halsted and Hall RJ utilized this knowledge to block inferior alveolar nerve to remove mandibular teeth. They showed that the injection of nerve trunk with a 4% cocaine solution in any part of its course is followed by loss of sensation in its entire peripheral distribution. Halsted made the injection and Hall was the patient. The first mandibular injection was made at Bellevue Hospital, in New York City, in November, 1884, exactly forty years after nitrous oxide was used for the same purpose on Horace Wells by John M Riggs. Thus, Halsted was the first to have introduced nerve block injections. The nerve he first "blocked" was the inferior alveolar nerve (mandibular nerve). In 1922, the Maryland State Dental Association honoured Dr Halsted with a gold medal for his original researches and discoveries in local and neuroregional anesthesia. Later, Halsted transferred his activities to John Hopkins Hospital.

In 1894, Carlson, and in 1896, Thiesing, both Dental Surgeons, when producing local anesthesia by spraying ethyl chloride on the gums, observed that several patients became unconscious (Thiesing, 1896). This observation prompted Thiesing to make experiments to employ ethyl chloride as a general anesthetic. In 1896, it passed from dentist's chair to operation theatre in the hospitals.

In 1890, "Pressure Anesthesia" was introduced into dentistry, by Edwards C Briggs (Briggs, 1891). Many high pressure obtunding syringes

charged with 4% cocaine, were advocated for desensitising teeth for cavity preparation and pulpal anesthesia.

Cataphoresis, a method of anesthesia, which was discarded 40 years earlier, was reintroduced. The technique was to saturate a piece of cotton with cocaine and the cotton containing electrode is applied to the part to be influenced. A weak current being turned on in the meantime. The part was supposed to become "obtunded" or "benumbed" (Anon, 1896). In some cases the electrode was connected to forceps.

In 1900, Legrand (Legrand, 1900) used a mixture of gelatin with his cocaine solution. Gelatin acted as a hemostatic. For the first time the chemical method was employed for achieving hemostasis. The other methods used earlier were application of physical aids such as (i) cold; for its vasoconstriction action and (ii) pressure; in the form of a tourniquet, around an extremity, effectively limited circulation; thereby definitely retarding absorption of cocaine. However, a great advancement took place when the action of epinephrine was demonstrated by Elsberg, Barber and Braun, when mixed with solution of cocaine.

In 1897, eucaine was used in place of cocaine. In 1901, George B Haycock, used betaeucaine, produced his "Bucaine compound" in tablet form. It was claimed to be superior to cocaine. It was non-toxic; could be sterilized by boiling and solutions could be stored indefinitely.

The intraosseous injections were introduced in England, using procaine hydrochloride as the anesthetic agent (Masselink, 1910). A high-pressure syringe, such as Gunthorpe's, was necessary for this procedure.

The research and development continued for a better local anesthetic agent; as cocaine had toxic and irritating properties, which resulted in extensive sloughing of tissues in many cases. A search was made to find synthetic substitute to cocaine, which would possess the anesthetic properties of cocaine, but without its disadvantages. The contributors were Liebermann, Willstaetter, and particularly Einhorn. It was found that the anesthetic property of cocaine depended upon the esterification of a basic alcohol with benzoic acid. Einhorn stated the definite principle that all esters of aromatic acids produced a greater or lesser degree of local anesthesia.

The first attempt to prepare cocaine synthetically was made and resulted in the eucaines, which were synthetically prepared by Merling. Two types, α and β eutacaine; were introduced. Eucaines were subsequently discarded on account of their irritation action, increased toxicity, and less intense anesthetic action. A more simple class of synthetic local anesthetic, stovaine, was prepared by Fourneau in 1904. Another drug, Elypin, which was prepared by Hoffmann, is chemically closely related to stovaine. These substances, because of their toxicity and irritating action on the tissues were not suitable for local anesthesia.

Novocaine (Procaine hydrochloride) (Procaine is the American name for Novocaine), one of the most popular substitutes, was discovered in Germany, by Alfred Einhorn in 1905. However, it was introduced in the practice of medicine by Braun in 1905 (Braun, 1905). The first paper in English on the use of novocaine in local anesthesia was that of Shepley Part (Part, 1906), in 1906. Since then, procaine rapidly replaced cocaine as a local anesthetic. Initially, the drug was supplied commercially in powder or tablet form. The dental practitioner mixed his solution as per his needs. That was a cumbersome, time-consuming and inaccurate method. The idea of using anesthetic solutions and drugs in cartridges, was first developed by Harvey, S Cook, a physician in 1940.

In 1933, Cobefrin, an ischemic agent, for use in local anesthetic solutions, was introduced in the dental profession by Leo Winter (Winter, 1933). Corbasil, the original name for Cobefrin, which is the American name, was first prepared by W Gruettefien, and was first reported in the application for German patent in 1911. Tiffeneau (Tiffeneau, 1915) published the first pharmacological report on Cobefrin. However, the most complete study was carried out much earlier by Schaumann, but the results were reported in 1930. The first clinical tests with Corbasil, or Cobefrin, alone and in combination with Novocaine and other local anesthetics, were carried out by Schaumann in 1933.

Monocaine was introduced to dental profession by American chemists, Goldberg and Whitmore, who performed a research on PABA esters of monoalkylamino alcohols. Monocaine hydrochloride was introduced at the Dental Clinic of the Ocean Hill Memorial Hospital in 1936, by Mendel Nevin (Goldberg and Whitmore, 1937).

Later, in the middle of 20th century a number of newer local anesthetic agents were introduced which are popular and commonly used today (Table 1.2). Lidocaine was prepared by Nils Lofgren in 1943 but was introduced in 1948. Propoxycaine was prepared by Clinton and Laskowski in 1952. Mepivacaine was prepared by Ekenstam in 1957 but was introduced into dentistry in 1960 as a 2% solution containing the synthetic vasopressor levonordefrin; and in 1961 as a 3% solution without a vasoconstrictor. Prilocaine was prepared by Lofgren and Tegner in 1953 but was reported in 1960. Articaine was prepared by Rusching et al in 1969 but was introduced in 1978 in the Netherlands, in 1980 in Austria and Spain, and in 1983 in Canada. Bupivacaine was prepared by Ekenstam in 1957. Etidocaine was prepared by Takman in 1971.

As Nevin has pointed out that it is a matter of great pride to the dental profession that Horace Wells first discovered, demonstrated, and proclaimed the blessings of surgical anaesthesia and William TG Morton two years later successfully introduced ether anesthesia. While Nevin and

Table 1.2: Timewise development of various local anesthetic agents

Year	*Esters*	*Amides*	*Discoverer*
1905	Procaine		Alfred Einhorn
1943		Lidocaine	Nils Lofgren
1948			Applied in clinical practice
1952	Propoxycaine		Clinton and Laskowsky
1953		Prilocaine	Prepared by Lofgren and Tegner
1956-57		Mepivacaine	Prepared by AF Ekenstam
1960			Introduced in dentistry
1957		Bupivacaine	AF Ekenstam
1969		Articaine	H Rusching et al
1971		Etidocaine	Takman

Nevin have pointed out that it is paradoxical that two dental practitioners, Horace Wells and William Morton discovered inhalation anesthesia, which is mainly used by physicians; while a physician Koller, to whom belongs the honour of discovery of anesthetic properties of cocaine and another physician, Halsted, who was the first one to introduce block anesthesia, which is universally used by dental practitioners.

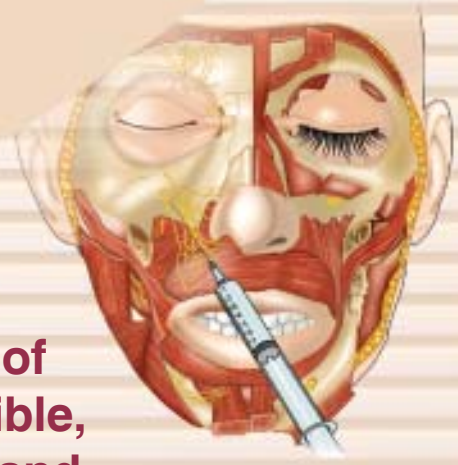
BIBLIOGRAPHY

1. American Dental Association. Transactions of the 11th Annual Meeting, White Sulphur Springs 1872.
2. Anon. Suggestions on the use of cataphoresis. A Practical Treatise. Baltimore: The Chloride of Silver Dry Cell Battery Co 1896.
3. Archer WH. A manual of dental anesthesia: An illustrated guide for students and practitioners. 2nd ed. WB Saunders Company 1958.
4. Braun H. Ueber einige neue ortliche Anesthetica (Stovain, Alypin, Novocain), Deutsche med. Wchnschr, (Oct. 19) 1905;31:1667.
5. Briggs EC. Removal of the pulp by the use of cocaine. Internat Dent J (May) 1891;12:296.
6. Bruck W. Kulturgeschechte Der Zahnheilkunde. In Einzeldarstellungen-part II. Das Martyrium Der Heiligen Appolonia, Verlag Von Hermann Neusser in Berlin 1915.
7. Castle AC. Painless extraction of teeth. Am J Dent Sc third series, (monthly summary), (April) 1872;5:569.
8. Davy, Sir Humphrey. Researches, Chemical and Philosophical 1799.
9. Fournier M. Local anaesthesia "Chloracetization." Dental Cosmos 3(7):1861-62.
10. Goldberg SG, Whitmore WF. Preparation of para-aminobenzoic acid esters of monoalkylamino alcohol. J Am Soc 1937;59:228.
11. Haggard HW. The Doctor in History. New Haven: Yale University Press 1934.
12. Hickman Henry Hill. Henry Hill Hickman Centenary Exhibition, 1830-1930, at the Wellcome Historical Museum, London, The Wellcome Fd. 1930.
13. Hollander B. Hypnosis and anaesthesia. Brit J of Anaesth (April) 1932;9:3.

14. Howe GL, Whitehead FIH. Local anesthesia in dentistry. 3rd ed. A Dental Practitioner Handbook Series 14. Wright. Mumbai: Published in India by Varghese Publishing House 1997.
15. Koller Carl. Historical notes on the beginning of local anesthesia. JAMA (May 26) 1928;90(1742).
16. Legrand. Solution Anesthetique-Hemostatique (Formula). Am J Dent Sc third series, (April) 1900;33:576.
17. Long CW. An account of the first use of sulphuric ether by inhalation anaesthetic in surgical operation. South Med & Surg J (Dec) 1849;5:12.
18. Masselink BH. The advent of painless dentistry. Dental Cosmos (Aug) 1910;52:868.
19. Nevin, Mendel, Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY. Dental Items of Interest Publishing Company Inc 1954.
20. Nevin, Mendel, Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY. Dental Items of Interest Publishing Company Inc 1948.
21. Part JS. Lancet Nov. 24 1906.
22. Raymond EH. Hydrochlorate of cocaine as a local anaesthetic in dental practice. Dental Cosmos 1885; 27:208.
23. Rice NP. Trials of a Public Benefactor, as Illustrated in the discovery of Etherization, New York: Pudney and Russell 1858.
24. Richardson BW. New method of producing local anesthesia (communication from the Lancet). Dental Cosmos (Sept) 1860;2:129.
25. Thiesing Dr Aethychlorid und Wilson's Local Anaestheticum, Deutsche Monatsch. f. Zahnh 1896; 140.
26. Tiffeneau. Compt. rend. Soc. de biol 1915;78:36.
27. Wells HG. The Outline of History 3rd ed. New York: The Macmillan Company 1921.
28. Winter Leo. An experimental and clinical investigations of a new ischemic agent for use in local anaesthetic solutions. Dent Outlook (Dec)1933;20:12.

Section

2



Applied Anatomy of Maxilla and Mandible, Neurophysiology and Pharmacology

- Osteology of Maxilla and Mandible
- Neuroanatomy
- Fundamentals of Nerve Impulse Generation and Transmission
- Theories of Pain Perception
- Mode of Action of Local Anesthetic Agents
- Local Anesthetic Agents
- Vasoconstrictors
- Local Anesthetic Cartridges and Vials

Chapter 2

Osteology of Maxilla and Mandible

MAXILLA

The maxilla consists of (a) central body, and (b) four processes.

a. *Central body:* It is hollowed out by the maxillary sinus.
b. *Processes:* Comprise of the following (1) frontal, (2) zygomatic, (3) palatine, and (4) alveolar processes.

Body of Maxilla

The body of the maxilla can be described as a three-sided pyramid with its base facing the nasal cavity. It lies at an almost horizontal axis, the apex being elongated into the zygomatic process.

Sides

The three sides of the pyramid are as follows:

i. *Superior or orbital surface:* It forms the greater part of the orbital floor.
ii. *Anterolateral or malar surface:* It forms part of the skeleton of the cheek and face.
iii. *Posterolateral or infratemporal surface:* It is the surface towards the infratemporal fossa.

Base

It is rimmed on its inferior edge by the alveolar process housing the tooth sockets.

Processes of Maxilla

Frontal Process

It ascends from the anteromedial corner of the body, and serves as the connection with the frontal bone.

Zygomatic Process

It forms the lateral corner of the body and connects with the zygomatic bone.

Palatine Process

It is a horizontal process that arises from the lower edge of the medial surface of the body; and along with the process of the other maxilla, forms the major anterior part of the skeleton of the hard palate.

Alveolar Process

It is a curved process, extends downward; and carries the sockets for the maxillary teeth.

Surfaces of Body of Maxilla

Medial or Nasal Surface

Maxillary sinus: The medial or nasal surface of the body of maxilla contains the large irregular maxillary hiatus in its posterior part, which leads to the maxillary sinus. Behind the opening, the bone is roughened for its junction with the vertical plate of the palatine bone.
Pterygopalatine groove: It is a shallow sulcus that begins above the middle of the posterior border, and descends obliquely downward and forward. It terminates at the angle formed by the posterior borders of the palatine process and the medial surface of the body. The upper edge of the nasal surface contains, in its posterior part one or two shallow depressions that complete and close some ethmoid cells.
Lacrimal sulcus: It is a deep vertical groove further forward that flattens out inferiorly. This sulcus is bordered in front by the continuation of the posterior border of the frontal process and in the back by a prominent bony spine that projects from the anterior edge of the maxillary opening. The remainder of the anterior half of the nasal surface of the body of maxilla is slightly concaved and terminates anteriorly at the sharp border of the piriform or anterior nasal aperture.
Conchal crest: It is a horizontal rough crest in front of the lacrimal sulcus and at its lower end. It serves for the attachment of the inferior nasal concha.

Orbital Surface

It is triangular and slightly sloping laterally and anteriorly. Its medial edge is sharp and joins, in its anterior part, the lacrimal bone and in its posterior part, the *lamina papyracea* of the ethmoid bone. At its posterior corner the palatine bone completes the orbital floor with its triangular orbital process.

Infraorbital rim: The anterior border of the orbital surface is smooth in its medial half. This part is thickened and forms a portion of the infraorbital

rim. The lateral part of the anterior border of the orbital surface is rough and continues laterally onto the triangular sutural surface of the zygomatic process. This entire area serves as the junction with the zygomatic bone. Posteriorly, the orbital surface is separated from the infratemporal surface by a blunt border that forms the inferior boundary of the inferior orbital fissure.

Infraorbital sulcus: This is a groove that courses from approximately the middle of the blunt border, anteriorly on the floor of the orbit. This groove contains the infraorbital nerve and vessels. The lateral edge of the sulcus is rather sharp and bends tonguelike over the lateral part of the sulcus. Further forward this thin bony process completely covers the infraorbital sulcus and transforms it into the infraorbital canal. Anteriorly, the bony roof of the canal known as the orbital plate thickens considerably. Thus, the canal descends in its most anterior part and turns slightly inward at the same time. The axes of the two infraorbital canals converge downward and, if extended, would cross each other at a point 1-2 cm in front of the upper central incisors.

Frequently, a suture line can be seen extending from the infraorbital foramen upward to the infraorbital margin. This is the line at which the orbital plate joins the body of maxilla.

A characteristic and rare anomaly of the infraorbital canal is mentioned. The infraorbital canal and foramen are sometimes shifted laterally and the canal follows a laterally convex arch through the base of the zygomatic process, which may be of clinical importance in anesthesia of the infraorbital nerve.

Anterior or Anterolateral or Malar Surface

The anterolateral surface of the body of maxilla forms the skeleton of the anterior part of the cheek and is, therefore, called the malar surface. It is bounded posteriorly by a bony crest that begins at the tip of the zygomatic process and continues in an arc, concave laterally and inferiorly, in the direction of the socket of the first molar, and disappears at the base of the alveolar process. This bony crest is the zygomaticoalveolar crest or jugal ridge, or the buttress of the zygoma.

Medially, the malar surface extends to the edge of the border of the pyriform aperture; in the midline the bone projects at the lower border of the aperture as a sharp spine that forms, with the corresponding projection of the contralateral bone, the anterior nasal spine.

Canine fossa: The lateral part of the anterior surface that continues onto the zygomatic process is concave. The concavity is variably deep and contains near its upper and inner corner the opening of the infraorbital canal, the infraorbital foramen. This foramen is found exactly below the

boundary between the smooth and roughened part of the anterior border of the orbital surface that is below the most medial point of the zygomaticomaxillary surface. The border of the infraorbital foramen is sharp in its upper and lateral circumference and blunt below and medially because of the oblique course of the infraorbital canal.

Posterior or Posterolateral or Infratemporal Surface

It is part of the anterior wall of the infratemporal fossa. This surface is convex in its greater medial part, the lateral part continues into the posterior concave surface of the zygomatic process. The posterior convexity of the body of maxilla is called the maxillary tuberosity or tuber. The posterior superior alveolar nerves enter the bone in the center of this surface through two or three fine openings, the posterior superior alveolar foramina. They lead into a narrow canal that runs downward and forward in the thin wall of the maxillary sinus.

The infraorbital canal itself juts into the maxillary sinus at the border between its roof and anterior wall. The wall between the infraorbital canal and the maxillary sinus can be deficient in persons with a large maxillary sinus.

a. *Zygomatic process:* The zygomatic process of the maxilla is the elongated apex of the pyramidal body. Its triangular superior surface is inclined laterally and is irregularly rough. It serves as the suture with the zygomatic bone. The anterior surface of the zygomatic process is an extension of the anterolateral surface of the body of maxilla, the posterior surface is concave and continues into the convex infratemporal surface of the body of the maxilla.
b. *Frontal process:* It is a bony process which lies in an almost sagittal plane.
   i. Its anterior border takes part in forming the upper border of the piriform aperture and then extends upward in a straight edge, to which the nasal bone is joined.
   ii. The posterior border of the frontal process arises approximately at the anteromedial corner of the orbital surface, it is in contact with the lacrimal bone.
   iii. The upper border of the frontal process is thickened and abuts the frontal bone.

Anterior lacrimal crest: It is a blunt vertical crest, parallel to and close to the posterior border, runs up the lateral surface of the frontal process. Behind this crest the frontal process forms part of the lacrimal groove. In front of the lacrimal crest the lateral surface of the frontal process is smooth and fairly flat.

Ethmoid crest: It is a fine horizontal ridge that crosses the inner surface of the frontal process, near its lower end, to which the middle concha of the ethmoid bone is attached.

Lower Surface

The alveolar process arises from the lower surface of the body of the maxilla. It consists of two roughly parallel plates of bone that unite behind the last tooth to form a small rough prominence, the alveolar tubercle, which often contains a single large narrow space. The lateral or external alveolar plate continues upward into the anterolateral and posterolateral surfaces of the body of maxilla, the internal alveolar plate continues into the palatine process and behind the posterior end of the latter into the nasal surface of the body of maxilla. The deep furrow between the two alveolar plates is divided by radial bony plates into the sockets of the individual teeth. These interalveolar or interdental septa connect the outer and inner alveolar plates. The sockets for multirooted teeth are, in turn, divided by intra-alveolar or inter-radicular septa. In the socket for the first premolar the intra-alveolar septum is parallel to the alveolar plates. In the sockets of the molars a sagittal septum divides the socket of the palatal root from those of the buccal roots, which themselves are separated by a secondary frontal septum.

Surface of Palatine Process

The palatine process arises as a horizontal plate from the body of the maxilla at the boundary between the body and the alveolar process. The palatine process is, in anteroposterior direction, shorter than the body of the maxilla and terminates posteriorly with a rough beveled border, to which the horizontal plates of the palatine bone are joined in the transverse palatine suture, and thus the skeleton of the hard palate.

At the corner, between the posterior edge of the palatine process and the medial wall of the maxilla, the bone is notched where the pterygopalatine sulcus ends. The palatine bone closes this notch to form the greater palatine foramen. From this notch the palatine groove extends anteriorly at the border between the palatine and alveolar processes. The groove houses the greater palatine nerve and vessels.

The border between the palatine and alveolar processes is sharp only in its posterior part, where the two processes join almost at right angles. Anteriorly, the angle between these processes is not so sharply defined, and in the region of the canine and incisors the inner plate of the alveolar process continues in a smooth curve into the palatine process.

The lower, or oral, surface of the palatine process is rough and irregular. The nasal surface is smooth and transversely concave. Along the midline the bone is elevated to the sharp nasal crest, which serves as the attachment of the nasal septum.

The incisive canal marks the boundary between the two constituent parts of the maxillary bone that unite early in embryonic life, the pre-

maxilla and the maxilla proper. From the nasal opening of the incisive canal a remnant of the line of fusion between the pre-maxillary and maxillary palatine processes can sometimes be traced, more often, however, the remnant of the incisive suture is observed at the oral surface of the palatine process. Here it begins at the incisive foramen and extends towards the mesial border of the canine sockets.

PALATINE BONE

The palatine bone supplements the maxilla and provides the link between the maxilla and the sphenoid bone.

It consists of a horizontal plate and a vertical plate joined at right angles. The horizontal plate, with its smooth superior or nasal surface and its rough inferior or oral surface forms the posterior part of the hard palate. Its anterior beveled border overlaps the palatine process of the maxilla in the transverse palatine suture. The medial border connects with the palatine bone of the other side, in the median or interpalatine suture, which is a continuation of the intermaxillary suture. At its nasal surface, the medial border is elevated to a sharp crest that continues with that of the other side, the nasal crest of the maxillary bones. The posterior border of the horizontal plate is concave and terminates medially in a sharp spine that forms, in conjunction with that of the contralateral bone, the posterior nasal spine. In front of its posterior border, the oral surface of the horizontal plate is often elevated as a sharp transverse ridge, the palatine crest. The lateral border, between the horizontal and vertical plates, is connected to the medial surface of the maxilla at the level between the body and the alveolar process. This border is cut near its anterior end by a notch that completes its cut near its anterior end by a notch that completes a similar notch in the maxilla to form the greater palatine foramen.

The vertical plate of the palatine bone is almost twice as high as it is wide. Its upper border is divided, by the deep sphenopalatine notch into two processes: (a) a larger anterior orbital process and (b) a smaller posterior sphenoid process. The greater part of the vertical lamella is in contact with the inner surface of the body of maxilla posterior to the opening of the maxillary sinus. A tongue like maxillary process of the vertical lamella, arising in the inferior part of its anterior border, closes posteroinferior part of the maxillary hiatus, thus narrowing it considerably. On the outer surface of the vertical plate a sulcus begins below the sphenopalatine notch and ends at the notch of the greater palatine foramen. This sulcus, the pterygopalatine groove, fits to the pterygopalatine groove of the maxilla and thus completes the walls of the pterygopalatine canal. Above this sulcus and around the palatine notch the lateral surface of the vertical plate of the palatine bone is smooth and forms the inner wall of pterygopalatine fossa.

The sphenoid process behind the sphenopalatine notch joins the sphenoid body. The orbital process fits into the posterosuperior corners of the maxillary body, where its orbital, infratemporal and nasal surfaces meet. It completes the orbital surface of the maxilla at this posterior corner. The orbital process is often hollowed out by a shallow fovea that closes a posteroinferior ethmoid cell. Behind this fovea this orbital process is connected to the anterior surface of the body of sphenoid. Since both the orbital and sphenoid processes are joined to the sphenoid bone above, the notch between these processes is closed by the body of the sphenoid to form the sphenopalatine foramen, a communication between pterygopalatine fossa and nasal cavity.

The medial or nasal surface of the vertical plate is generally smooth. At the border between the inferior third and middle third it is traversed by a sharp horizontal ridge, the conchal crest which serves as the attachment for the inferior concha. Near its base the orbital process is crossed by another horizontal rough line, the ethmoid crest, to which the posterior end of the middle nasal concha of the ethmoid bone is joined.

Pyramidal process: It is a short stout process that arises from the junction of vertical and horizontal plates at the posterolateral corner of the palatine bones. It serves as the firm anchorage of the maxillopalatine complex to the lower end of the pterygoid process of the sphenoid bone. The pyramidal process fills the pterygoid plates, which themselves fit into grooves at the posterior surface of the pyramidal process. The lower surface of pyramidal process continues as the oral surface of the horizontal palatine plate at the posterolateral corner just behind the greater palatine foramen.

Lesser palatine foramina: These are one or two sometimes even three smaller openings, medially and behind this sharp crest behind the greater palatine foramen, at which narrow canals open that branch from the pterygopalatine canal and perforate the pyramidal process.

MANDIBLE

The mandible consists of two parts: (a) a horseshoe-shaped body, the two halves of the body meet in the midline; and is continuous upward and backward on either side with (b) the two mandibular rami.

a. *Body:* It is thick and has two borders: (i) the rounded lower border and (ii) the upper or the alveolar border (margin) that carries the alveolar process. It extends backward from the chin at the midline symphysis to the anterior limit of the ramus.
b. *Ramus:* It is for the most part a thin quadrilateral plate. It extends backward from the groove for the facial artery (antegonial notch) to include the region called the mandibular angle. It reaches upward to end in two processes, (i) the anterior muscular coronoid process and (ii) the posterior articular condylar process.

Parts

Mental Protuberance or Bony Chin

It is a triangular prominence projecting from the anterior surface of the body in and adjacent to the midline. The base of this triangle coincides with the lower border of the body and is extended on either side as a small tubercle, the mental tubercle. A depression, the mental fossa, lies laterally, on either side of the triangular chin. Two or three tiny openings through which small blood vessels pass are sometimes present in this fossa. Also a small raised oval area for the origin of the mentalis muscle can be found in the fossa.

Mental Foramen

It is through this foramen, the mental nerve and blood vessels pass. In the horizontal direction, it is located on the lateral surface of the body, below and between the roots of the first and second premolars (sometimes below the apex of the second premolar). In the vertical dimension, the foramen lies halfway between the lower border of the mandible and the alveolar margin. Sometimes, especially in younger individuals, the foramen lies closer to the lower border. In the elderly or in the edentulous mandible, the foramen lies close to the alveolar border. The opening of the foramen faces outward, upward, and backward, consequently, the foramen is sharp only at its anteroinferior circumference, whereas its posterosuperior margin slants in gradually from the outer surface of the mandibular body. In edentulous mandible, the foramen comes to lie closer to the alveolar margin.

Superior View

a. *Body:* The alveolar process stems out from the upper border of the body and has a sharper curvature than does the bulk of the body itself. Thus, the body continues posterolaterally whereas the alveolar process turns inward toward the sagittal plane. Because of this, the posterior end of the alveolar process projects strongly inward from the arch of the body.
b. *Ramus:* It continues along the plane of the mandibular body, and is therefore situated well lateral to the plane of the alveolar process in the entire molar region. Thus, the anterior border of the ramus continues along the body lateral to the alveolar process as a blunt ridge, the oblique line or the external oblique ridge, running downward and forward to disappear at about the level of the first molar.

Body

Digastric Fossae

On the posterior part of the lower surface of the chin, a shallow oval roughened depression lies on each side of the midline. These are meant for the origins of the anterior bellies of digastric muscles.

Mental Spines/Genial Tubercles

The mandibular symphysis is elevated in more or less sharply defined projections slightly above the lower border on its inner surface. These may be fused or distinctly divided into right and left; and superior and inferior knobs, with small, roughened lateral hollows. The superior and inferior tubercles and adjacent hollow serve for the origins of the genioglossus and the geniohyoid muscles respectively.

Mylohyoid Line/Mylohyoid Ridge

From the region of the third molar a rough and slightly irregular crest extends diagonally downward and forward on the inner surface of the mandibular body. It is usually, most prominent in its superior and posterior parts. If well developed, it reaches the lower border of the mandible in the region of the chin, passing between digastric fossa and mental spines. The mylohyoid muscle takes its origin from this crest. Since this muscle forms the floor of the oral cavity, the bone above the line is part of the wall of the oral cavity, whereas, the bone below this line, forms the lateral wall of the submandibular space and therefore is accessible from the neck.

Submandibular Fossa

It is the slightly concave area below the mylohyoid line. It is so termed, because of its relation to the submandibular salivary gland.

Sublingual Fossa

It is a shallow depression above the anterior part of the mylohyoid line. It is in relation to the sublingual salivary gland.

Ramus

Mandibular Angle

In the region of the mandibular angle the bone is irregularly rough on the outer as well as on the inner surface. Depressions alternate with more or less pronounced ridges that frequently extend to the border, ending in small knoblike elevations. The irregularities are caused by the two muscles attached to the mandibular angle, the masseter muscle on the lateral side and the medial pterygoid muscle on the medial side.

The upper end of the ramus is divided into the posterior condylar and the anterior coronoid processes by the semilunar, sigmoid or mandibular notch.

Condylar Process

It carries the mandibular condyle or mandibular head, an irregulary cylindroid structure, the axis of which extends medially and posteriorly from laterally and anteriorly. The axes of the two condyles form an obtuse angle of 150° to 160° which is open to the front. The connection of the mandibular head with the mandibular ramus is slightly constricted and is known as mandibular neck. Above the neck the condyle itself is slightly bent anteriorly so that the articulating surface faces upward and forward. The sharp border of the mandibular notch continuing backward and upward meets the lateral pole of the condyle.

Pterygoid fovea: It is a depression found medial to this crest and on the anterior surface of the mandibular neck, to which most of the fibers of the lateral pterygoid muscle are attached.

Coronoid Process

It is a thin, triangular plate either sharply pointed or ending in a backwardly curved hook. Its posterior border is concave. Its anterior border is convex above then becomes concave below, the concavity coronoid notch being an important landmark for palpation in pterygomandibular block anesthesia.

The coronoid process as it runs downwards divides into two bony ridges (little above halfway). The lateral border continues up to first molar which is the anterior border of ramus of mandible, and is also known as external oblique ridge. The coronoid notch is the deepest portion on the anterior border. The other ridge runs downwards and medially to join the posterior part of the alveolar process in the region of the last standing molar tooth. This ridge is known as internal oblique ridge; and the inverted triangular space thus created is known as retromolar fossa.

Mandibular Foramen

It is a wide opening almost exactly in the center of the medial surface of the mandibular ramus, approximately 6-10 mm above the occlusal plane of mandibular molars, from where the mandibular canal starts. At its anterior circumference, a bony process, the mandibular lingula is found. At its posteroinferior circumference a narrow, sharply demarcated groove, the mylohyoid groove, commences and runs in a straight line downward and forward. It ends below the posterior end of the mylohyoid line and sometimes is closed to form a canal for some part of its length. It houses the mylohyoid nerve, which supplies the mylohyoid muscle.

At the tip of the coronoid process a crest begins that runs straight down, traversing the coronoid process and then continuing on the medial surface of the ramus, becoming more prominent in its downward course. Behind the last molar it bends into an almost horizontal plane and widens to a rough triangular field, the retromolar triangle. The prominent medial and lateral borders of the triangle continue into the buccal and lingual alveolar crests of the last molar. The vertical crest on the medial surface of the ramus serves as the attachment of the deep tendon of the temporal muscle and therefore is best designated as the temporal crest. Between it and the anterior border of the ramus is a depression the retromolar fossa, which is variable in its width and depth. The retromolar fossa continues between the alveolar process and the oblique line.

Ridge or Crest of the Mandibular Ramus

Another ridge begins on the inner surface of the mandibular ramus at the inner pole of the mandibular condyle, crosses the mandibular neck in a forward and downward course and continues in the region above and in front of the mandibular foramen, where it fuses with the elevation of the temporal crest. This ridge is of variable height and in contrast to the temporal crest, smooth and blunt. Because of its relation to the neck of the mandible and because it is here that this ridge is most prominent, it may be designated as the ridge or crest of the mandibular ramus. It transmits the forces of the mastication from the base of alveolar process in a trajectory to the mandibular head and from there to the base of the skull. The trajectory consisting of strengthened and parallel trabeculae of the spongy bone, bulges toward the inner surface of the mandibular ramus, thus causing the elevation described as the ridge of the mandibular neck. The compact bone itself along this crest is also thickened behind and below the ridge. The bone is depressed to a shallow groove, the groove of the mandibular neck, which is bounded posteriorly and inferiorly by a more or less prominent fairly sharp ridge to which the posterior fibers of the sphenomandibular ligament are attached. The groove itself bends into the mandibular foramen. The groove does not contain the inferior alveolar nerve.

Mandibular canal

It houses the inferior alveolar nerve and blood vessels, begins at the mandibular foramen, curves downward and forward, and turns into a horizontal course below the roots of the molars. In the region of the premolars, the mandibular canal splits into two canals of unequal width, the narrow incisive canal continues the course of the mandibular canal toward the midline; and the wider branch the mental canal turns laterally, superiorly and posteriorly to open through the mental foramen.

Alveolar Process

It consists of two compact bony plates, the external and internal alveolar or cortical plates. These two plates are joined to each other by the radial interdental and in the molar region by the inter-radicular septa, thus forming the sockets for the teeth much in the same manner as in the upper jaw. The outer alveolar plate is free distally to the level of the second molar. In the region of the second and especially the third molar, however, the bony substance of the oblique line is super-imposed on the outer alveolar plate because of the divergence of the mandibular bony and the alveolar process. This relation between alveolar process and the ramus gives the impression that the outer alveolar plate is of a considerable thickness in the distal part of the molar region.

BIBLIOGRAPHY

1. Atkinson ME, White FH. Principles of Anatomy and Oral Anatomy for Dental Students. Churchill Livingstone 1992.
2. Decker GAG, du Plessis DJ. Lee McGregor's Synopsis of Surgical Anatomy. 12th ed. Mumbai India: KM Varghese Co 1986.
3. DuBrul EL. Sicher and DuBrul's Oral Anatomy 8th ed. Ishiyaku EuroAmerica Inc. USA. ITBS Publishers and Distributors 2000.
4. Last RJ. Anatomy: Regional and Applied. 8th ed. Churchill Livingstone 1990.
5. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi, India: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
6. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. Delhi, India: CBS Publishers and Distributors 1990.
7. Sinnatamby CS. Last's Anatomy: Regional and Applied. 10th ed. International ed. Churchill Livingstone 2000.

THE TRIGEMINAL NERVE (V)

It is the largest cranial nerve, composed of a large sensory root and a small motor root. The name is derived from "trigemina", which means triplets.

Functions

The trigeminal nerve supplies sensation to meninges, skin of anterior part of the head, nasal and oral cavities and the teeth. It provides motor innervation to the muscles of mastication derived from the first branchial arch, and other muscles in the region.

Attachment

It is attached to the lateral aspect of pons, near middle cerebellar peduncle. It has two nuclei. (a) Motor nucleus, and (b) Sensory nucleus.

a. *Motor nucleus:* It is situated in upper pons. It is branchial for muscles of 1st pharyngeal arch. It lies deep beneath the floor of the 4th ventricle.
b. *Sensory nucleus:* It has three parts: (i) Mesencephalic nucleus, (ii) Pontine nucleus, and (iii) Spinal nucleus (pars oralis, pars interpolaris, and pars caudalis).

Divisions

The three divisions of trigeminal nerve register on spinal nucleus upside-down (i.e. mandibular division in upper medulla, maxillary division in closed medulla and ophthalmic division in upper cervical cord).

Course

It emerges from pons by a large sensory root and a small motor root. The two roots pass forward in posterior cranial fossa to trigeminal ganglion over the apex of petrous temporal bone, in the middle cranial fossa.

Together, the two roots pass below tentorium cerebelli to the mouth of trigeminal cave. The dural sheath containing the two roots passes forwards, separating the two layers of duramater that make the floor of middle cranial

fossa, just lateral to where the same two layers separate apart to enclose cavernous sinus. The sensory root then expands into large, flat, crescentic trigeminal ganglion; the motor root remains separate. The dural sheath obliterates subarachnoid space by fusing with piamater halfway along the ganglion; this is the anterior extremity of Meckel's cave.

As the roots run forward beneath dural floor of middle cranial fossa, they carry with them a loose sleeve of dura and arachnoid mater which lines trigeminal (Meckel's) cave. The dura and arachnoid fuse with epineurium of trigeminal ganglion in its anterior half so that only the posterior half, the sensory roots and motor nerve are bathed in CSF.

Types of Fibers

The sensory fibers are present in all three divisions of trigeminal nerve, only the mandibular division contains motor fibers.

Trigeminal Ganglion (Gasserian Ganglion/Semilunar Ganglion)

The trigeminal (sensory) ganglion is located in a small depression on the anterior surface of petrous part of temporal bone, known as the trigeminal cave or Meckel's cave. There are two ganglia; one innervating each side of the face. The ganglia are flat and crescent shaped, their convexities facing anteriorly and downward; and they measure 1 × 2 cm. It contains the cell bodies of all primary sensory neurons in all three divisions of trigeminal nerve, except those neurons carrying proprioceptive impulses.

It is partially surrounded by CSF in an extension of subarachnoid space below superior petrosal sinus. The anterior half of trigeminal ganglion and its three divisions, (V_1, V_2, and V_3), lie anterior to trigeminal cave. The upper part of trigeminal ganglion with V_1 and V_2 nerves lie in the lateral wall of cavernous sinus. The lower part of trigeminal ganglion and V3 nerve lie between the two layers of duramater lateral to cavernous sinus.

Roots

Motor Root

It arises separately from sensory root; originating in the main nucleus with pons and medulla oblongata, slightly cranial and medial to sensory root.

Its fibers, forming a small nerve root, leave pons superior to the site of entry of sensory fibers, travel anteriorly along with the sensory root, but separately, to the region of trigeminal ganglion, but come to lie inferior to sensory fibers in trigeminal ganglion. At the ganglion, the motor root passes in a lateral and inferior direction under the ganglion toward foramen ovale,

through which it leaves middle cranial fossa, along with the sensory root, the madibular nerve. In the foramen ovale, the motor root lies medial to the sensory root. Just after leaving the skull, the motor root unites with sensory root of mandibular division to form a single trunk.

The motor fibers of trigeminal nerve supply the following muscles:

1. Muscles of mastication: Masseter, temporalis, medial pterygoid and lateral pterygoid.
2. Other muscles in the region: Mylohyoid, anterior belly of digastric, tensor tympani and tensor veli palatini.

Sensory Root

Sensory root fibres of trigeminal nerve comprise of the central processes of ganglion cells located in trigeminal ganglion.

Divisions (Figs 3.1 to 3.3)

It divides into three branches. Sensory root fibers enter the concave portion of each crescent, and the three sensory divisions of trigeminal nerve exit from the convexity.

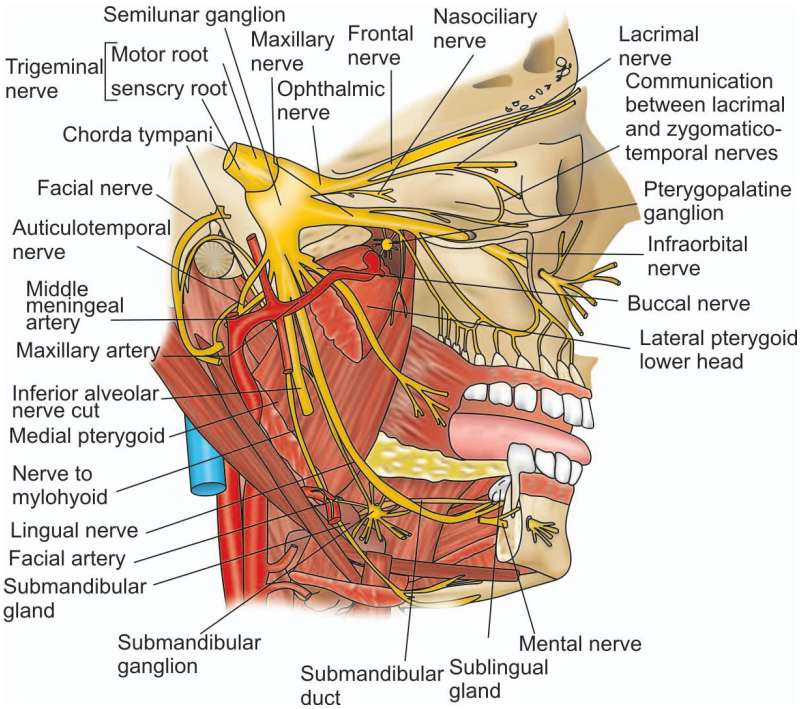


Fig. 3.1A: Trigeminal nerve and its branches

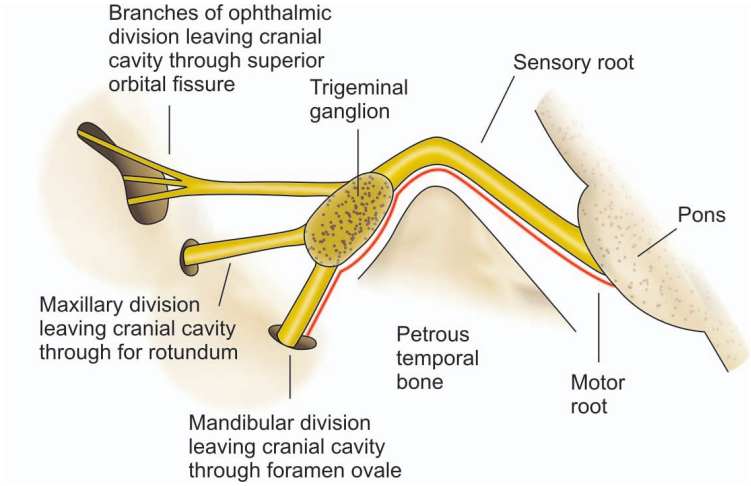


Fig. 3.1B: Roots and divisions of trigeminal nerve

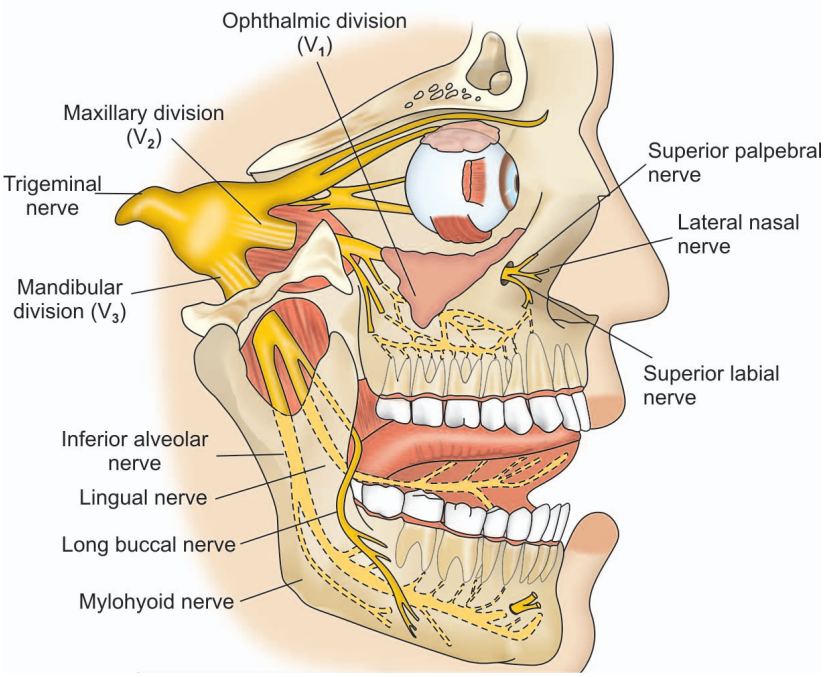


Fig. 3.2: Trigeminal nerve and its branches in relation to maxilla, mandible and teeth (diagrammatic)

Ophthalmic division (V_1): It travels anteriorly in lateral wall of cavernous sinus to medial part of supraorbital fissure, through which it exits the skull into the orbit. The area of its distribution is cornea, conjunctiva, upper eyelid, forehead, nose and anterior half of the scalp.

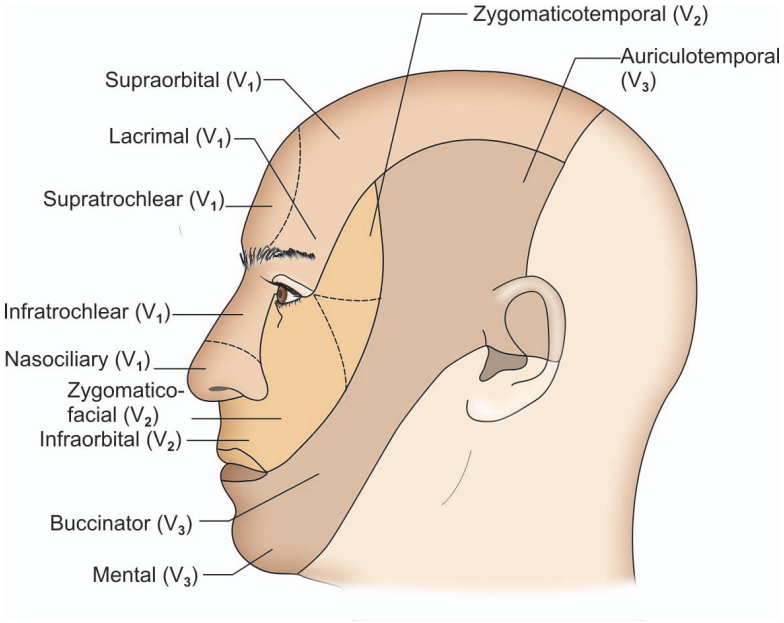


Fig. 3.3: Cutaneous sensory branches of the head and face regions

Maxillary division (V_2): It travels anteriorly and downward to exit the cranium through foramen rotundum into upper portion of pterygopalatine fossa. It crosses infraorbital fissure, the floor of the orbit, and emerges through the infraorbital foramen.

It supplies sensory innervation to the skin of the lower eyelid, skin of the cheek, skin of the lateral aspect of the nose, skin and mucosa of upper lip, maxillary teeth, and the osseous maxilla. It also supplies the mucosal surfaces of the uvula, hard palate, nasopharynx and inferior part of the nasal cavity.

Mandibular division (V_3): It is a mixed nerve. It travels almost directly downwards to exit the skull, along with the motor root, through foramen ovale. These two roots then intermingle, forming one nerve trunk, that enters infratemporal fossa. The sensory distribution is to the skin over the mandible, skin of the auricle, the anterior part of the external auditory meatus, buccal surface of the cheek, lower lip, buccal and lingual gingivae, mandibular teeth, floor of the mouth, submandibular and sublingual salivary glands, and the anterior two-thirds of the tongue. The motor component supplies the muscles of mastication and other muscles in the region, such as mylohyoid, anterior belly of digastric, tensor tympani and tensor veli palatini.

THE OPHTHALMIC NERVE (V_1)

It is the nerve of frontonasal process. It is the first and the smallest of the three divisions of trigeminal nerve; and is a sensory nerve.

Functions

The ophthalmic nerve transmits sensory innervation from eyeballs, conjunctiva, skin of the upper face and anterior scalp, the lining of upper part of nasal cavity and paranasal air sinuses, and the meninges of anterior cranial fossa. Its branches also convey parasympathetic fibers to the ciliary and iris muscles for accommodation and pupillary constriction, and to the lacrimal gland.

Nerve Fibers (Central Connexons)

a. *Somatic sensory fibers:* Sensory nuclei of trigeminal nerve are present in all branches of ophthalmic nerve. The axons pass centrally to trigeminal ganglion where cell bodies are situated. The central axonal processes pass to pons and trigeminal sensory nuclei.
b. *Parasympathetic fibers:*
   i. Edinger-Westphal nucleus to muscles of ciliary body and iris.
   ii. Superior Salivatory Nucleus to lacrimal gland (Table 3.1).

Origin and Course

It originates from the upper part of trigeminal ganglion in the middle cranial fossa. It passes anteriorly, through lateral wall of cavernous sinus, below trochlear (CN IV) nerve. Here it picks up sympathetic fibers from cavernous plexus, these are for dilator pupillae muscle. At the anterior end of cavernous sinus, it gives off meningeal branches. Finally, just before it enters the supraorbital fissure, it divides into three terminal branches and is distributed to cornea, conjunctiva, upper eyelid, forehead, nose, and anterior half of scalp.

Branches (Fig. 3.4)

1. *Branches in the cranium: Meningeal nerves (or tentorial nerves):* Which provide sensation to all supratentorial duramater except that in the bony floor of middle cranial fossa.
2. *Communicating branches:* To oculomotor, trochlear, and abducens cranial nerves.
3. *Terminal branches:* (a) Lacrimal, (b) Frontal, and (c) Nasociliary nerves. All these branches pass through supraorbital fissure into the orbit.
   a. *Lacrimal nerve:* It is the smallest of the three branches, and supplies lacrimal gland; and a small area of adjacent skin and conjunctiva.

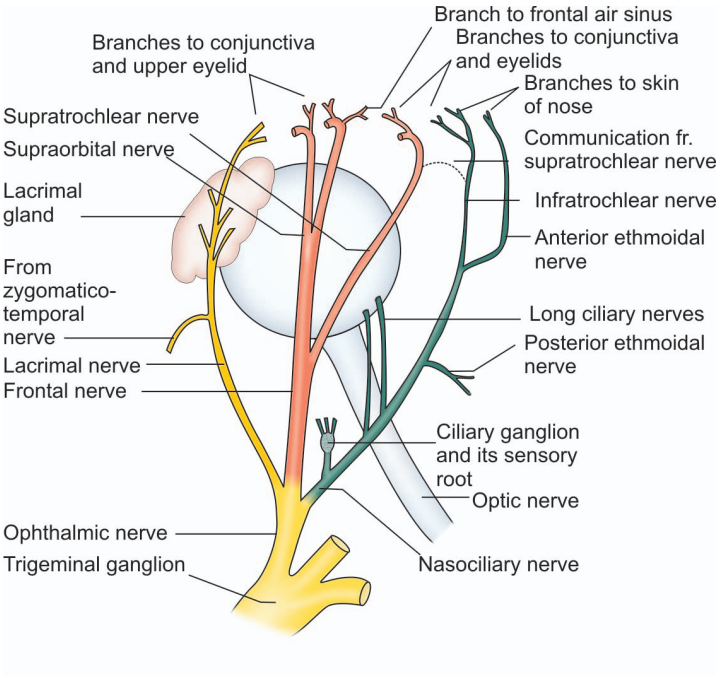


Fig. 3.4: Distribution of ophthalmic (V_1) nerve

It passes through supraorbital fissure, lateral to, and outside common tendinous ring, then it proceeds along the upper part of lateral wall of the orbit. Here it receives, postganglionic parasympathetic secretomotor fibers from pterygopalatine ganglion, which enter orbit with zygomatic nerve for distribution to lacrimal gland. It is sensory to a small area of skin at the lateral end of upper eyelid and to both palpebral and ocular surfaces of corresponding conjunctiva.

b. *Frontal branch:* It is the largest of the three branches; it appears to be the direct continuation of ophthalmic division.

It leaves cavernous sinus to traverse supraorbital fissure, just lateral to the common tendinous ring, bunched together between lacrimal and trochlear nerves. It passes forwards in contact with the periosteum above levator palpebrae superioris, just below the frontal bone. It supplies frontal sinus and skin of forehead and scalp. Just behind superior orbital margin, it divides into two branches: (i) Supratrochlear, and (ii) Supraorbital nerves.

i. Supratrochlear: It is the smaller and medial branch. It supplies skin of the medial aspect of upper eyelid and conjunctiva, and a narrow strip of skin of forehead alongside midline. It scarcely extends into scalp, where the two supraorbital nerves meet each other.

ii. Supraorbital: It is the larger and the lateral branch. It leaves the orbit through the supraorbital notch or foramen (with supraorbital branch of ophthalmic artery), and turns upwards. It supplies frontal sinus, upper eyelid (skin and both surfaces of conjunctiva), skin on the forehead, except the central strip, and the frontal scalp as far posteriorly as the vertex (as far back as the parietal bone, and to the lambdoid suture).

NB: The nerves and vessels in scalp lie superficial to fronto-occipital aponeurosis.

c. *Nasociliary nerve:* It passes through the supraorbital fissure, medially, within the common tendinous ring, and then travels along the medial border of the roof of the orbit. It supplies meninges, whole eyeball, some of the mucous membrane of the upper part of nasal cavity, ethmoidal air cells, and skin of the external nose, and ends in the skin at the root of the nose.

Branches

The branches are divided into those arising (a) in the orbit, (b) in the nasal cavity, and (c) on the face.

1. *Sensory root of ciliary ganglion:* This passes through ciliary ganglion and via the short ciliary nerves, is sensory to whole eyeball including cornea (not conjunctiva).
2. *Branches in the orbit:*
   a. Long ciliary nerves, and (b) Short ciliary nerves.
   a. *Long ciliary nerves:* It is the long and the sensory root which arises from nasociliary nerve. Two branches of nasociliary nerves enter sclera independently. They carry sympathetic fibers picked up by ophthalmic nerve in the cavernous sinus, to dilator pupillae muscle. It leaves cavernous sinus and enters the tendinous ring between the two divisions of oculomotor (CN III) nerve. It passes straight forwards into the cone of the muscles above optic (CN II) nerve and divides into its terminal branches. These are (i) infratrochlear, (ii) anterior ethmoidal, and (iii) posterior ethmoidal nerves. These nerves leave the cone of the muscles above medial rectus and below superior oblique muscles. These are the only nerves to traverse the cone of the muscles, and all three pass through the same intermuscular slit.

   These nerves pass to the eyeball to innervate ocular structures including cornea.

   i. *Infratrochlear nerve:* It passes forwards just below the trochlea of superior oblique tendon, supplying skin and conjunctiva at the medial end of upper lid and ends on skin over bridge of the nose (nasal bone).

ii. *Anterior ethmoidal nerve:* It gives sensory fibers to the meninges of anterior cranial fossa; and enters nasal cavity. It supplies, along with posterior ethmoidal nerve, the upper part of nasal cavity and sphenoid and ethmoidal air cells.

It passes into anterior ethmoidal foramen below frontal bone. It runs obliquely forwards in the roof of the middle and anterior ethmoidal air cells, and supplies both. It passes onto cribriform plate, between two layers of duramater. It descends through nasal slit alongside the front of crista galli into roof of the nose. It straddles the nose, supplying anterior superior quadrant of lateral wall and anterosuperior half of the nasal septum. It then continues towards the bridge of the nose and becomes superficial at the junction of nasal bone and cartilage of nasal bridge, under the name of external nasal nerve, and supplies skin over external nasal cartilages, down to the tip of the nose.

iii. *Posterior ethmoidal nerve:* It enters posterior ethmoidal foramen and supplies posterior ethmoidal air cells, and adjacent sphenoidal sinus. It does not reach nasal cavity.

b. *Short ciliary nerves:* These nerves contain preganglionic parasympathetic fibers from Edinger-Westphal Nucleus and oculomotor nerve (CN III) passing to Ciliary Ganglion (synapse). Long and short ciliary nerves also contain sympathetic fibers.

THE MAXILLARY NERVE (V_2)

It is the nerve of maxillary process that differentiates from 1st pharyngeal arch. It is the second division of the trigeminal nerve, is purely sensory, and is intermediate in size between ophthalmic and mandibular nerves.

Functions

The maxillary nerve transmits sensory fibers from the skin of face between the lower eyelid and the mouth, from the nasal cavity and sinuses, from the maxillary teeth and also from the maxilla.

Origin, Course and Branches (Figs 3.5 to 3.9)

It originates from the middle part of trigeminal ganglion in the middle cranial fossa. At its origin from the pons, it contains only sensory fibers. Some of its branches receive postganglionic parasympathetic fibers from pterygopalatine ganglion which pass to the lacrimal, nasal and palatine glands, and others convey taste (visceral sensory) fibers from the palate to the Nucleus of the Solitary Tract (Nucleus of Tractus Solitarius).

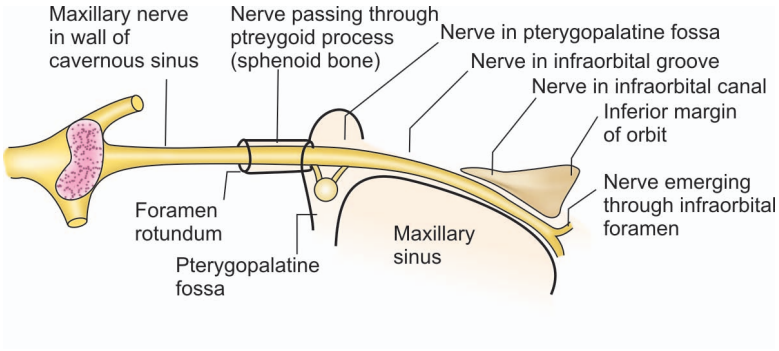


Fig. 3.5: Course of maxillary (V_2) nerve

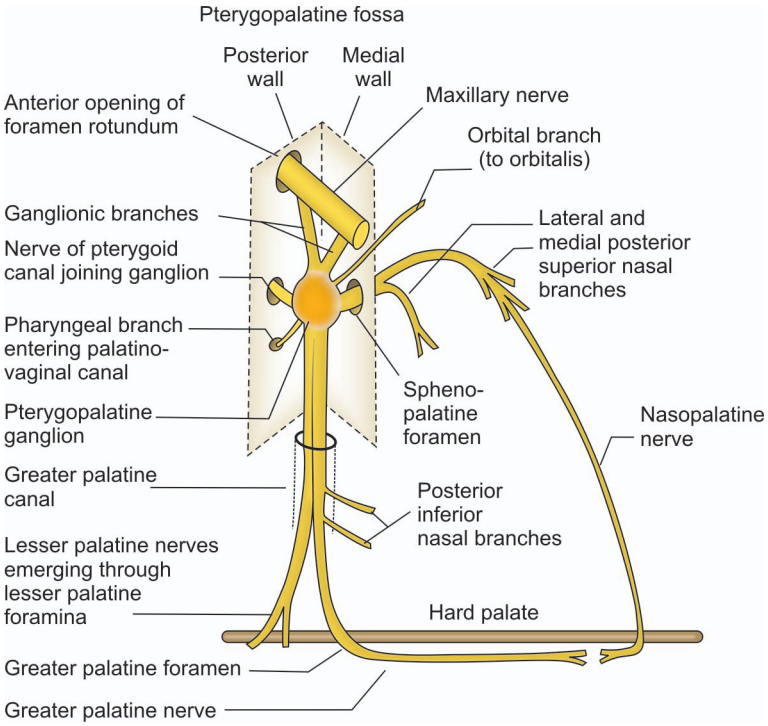


Fig. 3.6A: Branches of pterygopalatine ganglion (PPG)

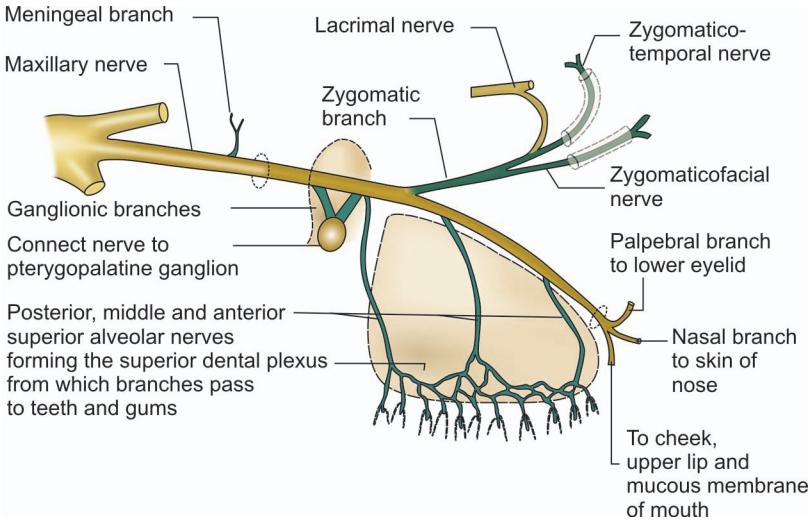


Fig. 3.6B: Direct branches arising from maxillary (V_2) nerve (including its infraorbital continuation)

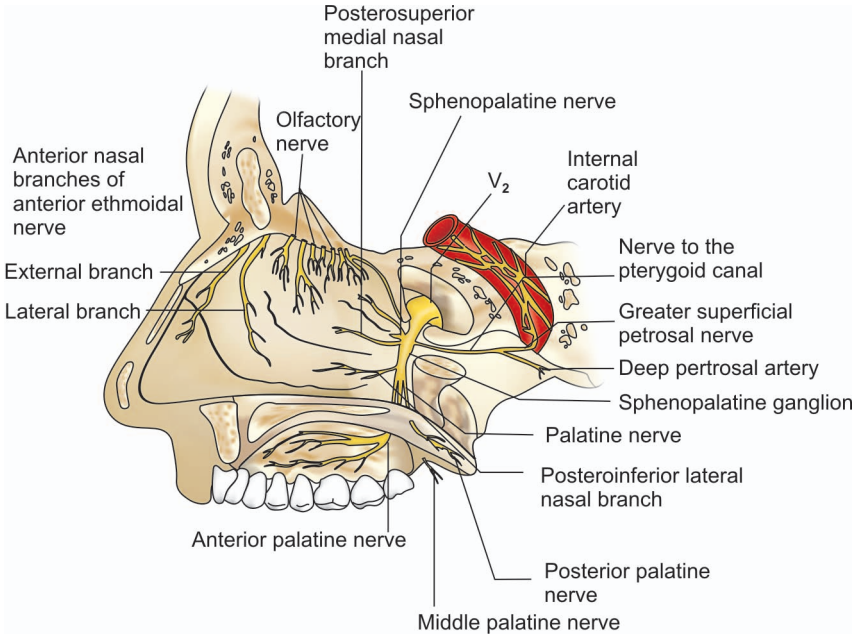


Fig. 3.7: Innervation of the right lateral wall of the nose

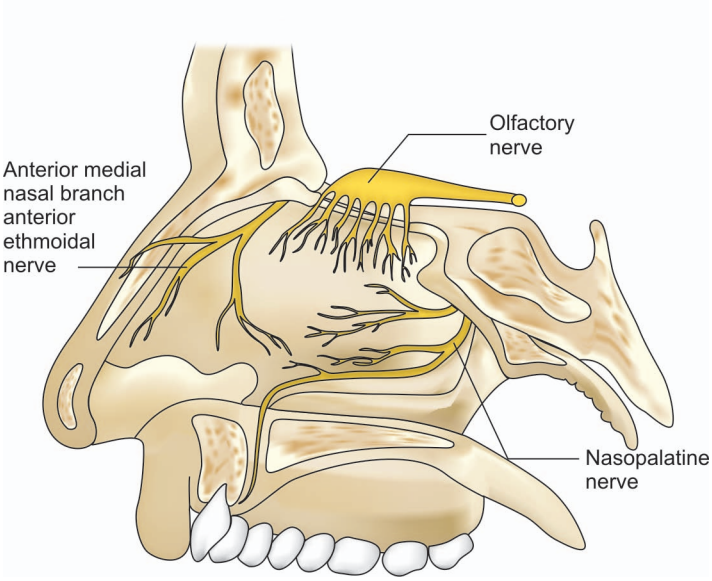


Fig. 3.8: Innervation of the nasal septum

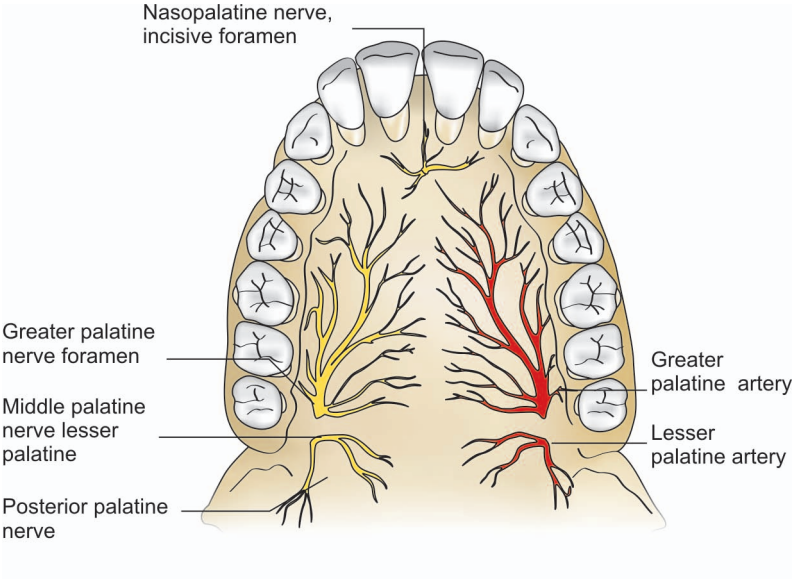


Fig. 3.9: Innervation and blood supply of the palate

It gives off meningeal branch in the middle cranial fossa; which is sensory. The maxillary nerve runs forwards through lower part of lateral wall of cavernous sinus below ophthalmic nerve. The lateral wall here fuses with the endosteal layer of duramater at the lateral margin of foramen rotundum; so the maxillary nerve is directed through the foramen rotundum into the uppermost part of pterygopalatine fossa, between pterygoid plates of sphenoid bone and the palatine bone.

It has a short course below the roof of pterygopalatine fossa to infraorbital fissure. As it crosses pterygopalatine fossa, it gives off its main branches; to pterygopalatine ganglion, posterior superior alveolar nerves and zygomatic branches. It then angles laterally in a groove on the posterior surface of maxilla, entering the orbit through infraorbital fissure. Within the orbit, it occupies the infraorbital groove, and becomes infraorbital nerve, which courses anteriorly into the infraorbital canal. Then the nerve emerges on the anterior surface of face through infraorbital foramen, where it divides into its terminal branches supplying skin of the lower eyelid, skin of the middle portion of face (cheek), skin of the lateral aspect of the nose, and skin and mucosa of upper lip, maxillary teeth and maxilla. It also supplies mucosal surfaces of uvula, hard palate, nasopharynx, and inferior part of nasal cavity.

Innervation

It provides sensory supply to the following structures:

1. Skin of:
    i. Middle portion of face,
    ii. Lower eyelid,
    iii. Side of the nose, and
    iv. Upper lip.
2. Mucous membrane of:
    i. Nasopharynx,
    ii. Maxillary sinus,
    iii. Soft palate,
    iv. Tonsils, and
    v. Upper lip.
3. Hard palate.
4. Pulps of maxillary teeth and buccal periodontal tissues.

Pterygopalatine Ganglion (PPG) or Meckel's Ganglion

This ganglion is connected with the maxillary division of Vth cranial nerve and is the parasympathetic relay station between the Superior Salivatory Nucleus in the pons and the lacrimal gland and mucous and serous glands of the palate, nose and paranasal sinuses.

It gives off branches in four regions:
A. Within the cranium,
B. In the pterygopalatine fossa,
C. In the infraorbital canal, and
D. On the face.

Branches within the Cranium

Middle meningeal nerve: Immediately after separating from trigeminal ganglion, the maxillary nerve gives off a small branch. It travels along with middle meningeal artery. It provides sensory innervation to the duramater of anterior half of middle cranial fossa.

Branches in the Pterygopalatine Fossa

It gives off following branches:

i. Ganglionic branches/Pterygopalatine nerves
ii. Zygomatic nerves
iii. Posterior Superior Alveolar nerves

Ganglionic Branches/Pterygopalatine Nerves

These are two short nerves that suspend the pterygopalatine ganglion. These nerves pass through the ganglion into its branches. They also serve as a communication between pterygopalatine ganglion and maxillary nerve. These branches mingle with post-ganglionic parasympathetic secretomotor fibers of greater petrosal nerve and sympathetic fibers of deep petrosal nerve (Table 3.1).

By way of pterygopalatine ganglion, the maxillary nerve has the following branches, all of which carry sensory, secretomotor and

Table 3.1: Parasympathetic ganglia associated with trigeminal nerve

S. No.	*Division*		*Ganglia*	*Source of pre-ganglionic fibers*	*Innervation*
1.	V_1	Ciliary	Oculomotor nerve		(i) Muscles of accommodation (ciliary muscle) (ii) Sphincter pupillae
2.	V_2	Pterygopalatine	Facial nerve: Greater petrosal nerve		Lacrimal gland
3.	V_3	Submandibular	Facial nerve: Chorda Tympani nerve		(i) Submandibular salivary gland (ii) Sublingual salivary gland
		Otic	Glossopharyngeal nerve	Lesser petrosal nerve	Parotid salivary gland

sympathetic fibers. The branches of pterygopalatine nerves, which are divided into four groups, and supply four areas: Orbit, nose, palate and pharynx; and hence are: (i) orbital, (ii) nasal, (iii) palatine, and (iv) pharyngeal branches.

1. *Orbital branches:* These are the ascending branches which supply periosteum of orbit.
2. *Nasal branches:* These are the internal branches which are distributed to the nasal cavity, and supply mucous membranes of superior and middle conchae, the lining of posterior ethmoidal sinus, and posterior portion of nasal septum. These convey post-ganglionic parasympathetic fibers from pterygopalatine ganglion to nasal glands.
   a. *Nasopalatine nerve (Long sphenopalatine nerve) (Table 3.2):* It is a significant branch. It enters sphenopalatine foramen, crosses the roof of nasal cavity and slopes downward and forward, where it lies between mucous membrane and periosteum of nasal septum (It passes over roof of nasal cavity to reach septum). There it supplies its posterior and inferior half.

      The nasopalatine nerve continues downwards reaching the floor of nasal cavity and giving off its branches to the anterior part of nasal septum and the floor of nose. It then enters incisive canal, through which it passes into oral cavity via the incisive foramen, located in the midline of hard palate, about 1 cm posterior to maxillary central incisors. The right and the left nasopalatine nerves emerge together through this foramen and provide sensory innervation to palatal mucosa in the region of premaxilla (central incisors, lateral incisors and canines).
   b. *Posterior superior lateral nasal nerves (Short sphenopalatine nerves):* These branches enter sphenopalatine foramen and supply posterior superior quadrant of lateral wall of the nose.
3. *Palatine branches:* These are the descending branches. These are: (a) Greater palatine nerve (earlier known as anterior palatine nerve), (b) Lesser palatine nerves (earlier known as middle and posterior palatine nerves) (Table 3.2).
   a. *Greater palatine nerve:* It provides sensory supply to the hard palate. It descends down through the greater palatine (pterygopalatine) canal of maxilla. It emerges on hard palate through greater palatine foramen. It is usually located about 1 cm towards the palatal midline, just distal to second molar. Multiple branches supply the posteroinferior quadrant of the lateral wall of the nose and adjacent floor of the nose, others supply maxillary sinus nearby.

      The nerve crosses anteriorly, after emerging from greater palatine foramen between mucoperiosteum and osseous hard palate supplying sensory innervation to palatal soft tissue and bone, up to

canine/first premolar, where it communicates with terminal fibers of nasopalatine nerves. It also provides sensory innervation to some parts of soft palate. It supplies all the hard palate except that in the incisor area.

b. *Lesser palatine nerves:* These are sensory nerves to soft palate. They descend through lesser palatine foramina in the palatine bone and pass backwards to supply mucous membrane on both the surfaces of soft palate.

4. *Pharyngeal branches:* These are distributed to the upper part of pharynx and the posterior part of the roof of the nasal cavity. These nerves provide sensory innervation to nasopharynx. It is a small nerve, leaving posterior part of pterygopalatine ganglion, passes backwards, through the pharyngeal canal and supplies mucous membrane of nasopharynx, posterior to Eustachian (auditory) tube; down to the level of Passavant's muscle.

Zygomatic Nerve

It is a terminal branch of maxillary nerve. It comes out of pterygopalatine fossa and travels anteriorly entering the orbit through infraorbital fissure and runs along the lower part of lateral wall of orbit. The zygomatic nerve enters zygomatic bone and divides into two branches. Some authors state that it divides into its branches in the infraorbital fissure.

It provides sensory innervation to the skin over zygomatic region. These nerves also convey postganglionic parasympathetic fibers from pterygopalatine ganglion via the lacrimal nerve to the lacrimal gland.

a. *Zygomaticotemporal nerve:* It perforates the temporal surface of zygomatic bone, pierces temporalis fascia and supplies skin above the zygomatic arch; the skin of the side of the forehead or the "hairless" skin of the temple.

Just before leaving orbit, zygomatic nerve sends a branch that communicates with lacrimal branch of ophthalmic nerve. It carries secretomotor fibers from pterygopalatine ganglion for the lacrimal gland. These branches leave zygomatic nerve and go up the lateral wall of orbit to join lacrimal nerve.

The postganglionic parasympathetic fibers to the lacrimal gland pass through the zygomaticotemporal branch of the maxillary nerve and the lacrimal branch of the ophthalmic nerve.

b. *Zygomaticofacial nerve:* It perforates the facial surface of zygomatic bone and supplies the skin over the zygomatic bone (skin over the prominence of cheek).

Superior Dental Plexus

Nerve Loops

1. *Outer nerve loop:* The loop comprises of the following nerves: Posterior Superior Alveolar nerve, Middle Superior Alveolar nerve, and Anterior Superior Alveolar nerve.

   It supplies pulps of the teeth; through fine nerve filaments that actually pass through the apical foramina into the pulp chambers. It also supplies the buccal alveolar plate in the region of bicuspids and molars; the labial plate in the region of anterior teeth and the periosteum and mucous membrane covering the labial/buccal alveolar plate.

   The innervation of the roots of all teeth, bone and periodontal structures are derived from terminal branches of larger nerves. These plexuses are composed of small nerve fibers or filaments from the three superior alveolar nerves; Anterior, Middle and Posterior Superior Alveolar nerves. These nerves before entering apical foramina of teeth, form a network termed as "Superior Dental Plexus", from which fine filaments are given off and pass through the apical foramina.

   The Middle Superior Alveolar nerve anastomoses with Posterior Superior Alveolar nerve, forming a small enlargement, the "Ganglion of Valentine". The Middle Superior Alveolar nerve also anastomoses with the Anterior Superior Alveolar nerve and forms a similar enlargement, the "Ganglion of Bochdalek".
2. *Inner nerve loop:* The nerves forming the inner nerve loop are the Nasopalatine and the Greater Palatine nerves.

   It has no connection with the pulps of the teeth. It supplies the palatal alveolar plates of maxillary arch and the overlying soft tissues, the periosteum and the mucous membrane.

   Hence, in cases of pulp extirpation, grinding of teeth, preparation of cavities, and for any surgical procedure on maxillary teeth which do not involve palatal alveolar plate and the mucous membrane, the outer nerve loop only needs to be anesthetised. While in situations, such as extraction of teeth, removal of cysts and any similar surgical procedure, which would involve the palatal plate, the inner nerve loop, as well as the outer nerve loop, should be anesthetised to achieve complete anesthesia.

   The innervation of maxilla considerably differs from that of mandible. The inferior alveolar nerve supplies not only the mandibular teeth but also the outer and inner cortical plates.

Inner Nerve Loop and the Pterygopalatine Ganglion (PPG)

The inner nerve loop is derived from pterygopalatine ganglion, which is located in pterygopalatine fossa just below maxillary division. Every

ganglion has incorporated in it the three types of nerve cells. The sensory source of pterygopalatine ganglion comprises of two short branches from maxillary division.

The vidian nerve, which joins the ganglion at its posterior portion, is formed by greater palatine nerve, a motor nerve and deep petrosal nerve, a branch from the sympathetic plexus on internal carotid artery.

Superior Alveolar Nerves

These nerves are 2-3 in number, descend from the main trunk of the maxillary nerve in the pterygopalatine fissure just before the mandibular division enters inferior alveolar canal. They emerge through the pterygomaxillary fissure. They pass downward to the pterygopalatine fissure and reach the posterior surface of maxilla (or infratemporal surface of maxilla). When more than two, one branch remains external to bone, continuing downwards on the posterior surface of maxilla, to provide sensory innervation to buccal gingiva in maxillary molar region; and adjacent facial mucosal surfaces.

The other branch enters maxilla (along with a branch of internal maxillary artery), through the posterolateral wall of maxillary sinus, and provide sensory innervation to mucous membrane of sinus. Continuing downwards, this second branch provides sensory innervation to alveolar bone, buccal periodontal ligament and pulpal tissues of maxillary molars, with the exception of mesiobuccal root of first molar.

Branches in the Infraorbital Canal

There are two versions:

a. In the infraorbital groove, it gives off middle superior alveolar nerve and in the infraorbital canal, it gives off anterior superior alveolar nerve.
b. In the infraorbital canal, the infraorbital nerve gives off two branches:
   i. Middle superior alveolar nerve, and
   ii. Anterior superior alveolar nerve.

   While in the infraorbital groove and canal the maxillary division is known as infraorbital nerve. The nerve passes forwards along the floor of orbit, sinks into a groove, then enters the canal, and emerges on the face through infraorbital foramen. It supplies multiple small branches through the orbital plate of maxilla to the roof of maxillary sinus. Infraorbital nerve has many communications with local branches of facial nerve (Cranial Nerve VII). These are the proprioceptive supply of nearby facial muscles.

   i. *Middle superior alveolar nerve; Site of origin:* It is anywhere in the infraorbital canal, from the posterior part in the canal to anterior portion. The nerve runs down, supplying adjacent lining of maxillary

sinus, pulps of premolars and mesiobuccal root of first molar, buccal periodontal tissues, buccal soft tissues and alveolar bone in the premolar region.

ii. *Anterior superior alveolar nerve:* It is a relatively larger branch. It is given off from the infraorbital nerve in the infraorbital canal, approximately 6-10 mm prior to its exit from the infraorbital foramen. It descends first, laterally and then inferiorly to the infraorbital canal. Within the wall of maxillary sinus, it provides innervation to central and lateral incisors and canines, as well as sensory innervation to adjacent lining of maxillary sinus, buccal periodontal tissues, buccal bone and buccal gingivae of these teeth. It reaches anteroinferior quadrant of lateral wall of the nose (including nasolacrimal duct) and adjacent floor of nose. It ends on nasal septum.

In patients where middle superior alveolar nerve is absent, the anterior superior alveolar nerve provides sensory innervation to premolars and occasionally the mesiobuccal root of first molar.

Three types of nerves emerge from the superior dental plexus, which are as follows: (i) Dental nerves, (ii) Interdental nerves, and (iii) Inter-radicular branches.

These are accompanied along its pathway by corresponding artery.

i. *Dental nerves:* Enter a tooth through apical foramen dividing into many branches within the pulp.

ii. *Interdental nerves:* Also known as perforating branches, travel through entire height of interdental septum providing sensory innervation to periodontal ligaments of adjacent teeth to alveolar bone. They emerge at the height of the crest of interalveolar septum and enter the gingiva to innervate the interdental papilla and buccal gingiva.

iii. *Inter-radicular nerves:* These nerves travel the entire height of inter-radicular septum, providing sensory innervation to periodontal ligaments of tooth. They terminate in the periodontal ligament at the furcation of the roots.

Branches on the Face

Infraorbital nerve emerges on the face through infraorbital foramen, lies between levator labii superioris and levator anguli oris, and divides into its terminal branches:

i. *Inferior palpebral nerve:* It supplies skin of lower eyelid with sensory innervation to both surfaces of conjunctiva.

ii. *External or lateral nasal nerve:* It provides sensory innervation to the skin over the lateral aspect of nose.

iii. *Superior labial nerve:* It provides sensory innervation to the skin and mucous membrane of whole of upper lip (and sometimes also the adjacent gingivae from midline to second premolar teeth).

THE MANDIBULAR NERVE (V_3)

It is the nerve of first branchial (mandibular) arch. It is the largest of the three divisions of trigeminal nerve. It is a mixed nerve and consists of two roots; a large sensory root and a small motor root; the latter, representing the entire motor component of the trigeminal nerve.

The first arch gives rise to; a precursor of mandible, the Meckel's cartilage; and the spine of sphenoid, sphenomandibular ligament, malleus, incus, and muscles of mastication (primary and secondary).

Functions

a. *Sensory:* It transmits sensory fibers from (i) the skin over the mandible, side of the cheek and temple, (ii) the mucosa of the oral cavity and its contents, (iii) external ear, tympanic membrane, (iv) temporomandibular joint, and (v) it also supplies the meninges of cranial vault.
b. *Motor:* It is motor to the muscles derived from 1st branchial arch; namely, temporalis, masseter, medial pterygoid and lateral pterygoid (primary muscles of mastication), mylohyoid, anterior belly of digastric (secondary muscles of mastication), and tensor tympani and tensor palati.
c. *Secretomotor:* Some of its distal branches also convey parasympathetic secretomotor fibers to salivary glands, and taste fibers from anterior portion of tongue.

Distribution (Figs 3.10 to 3.16)

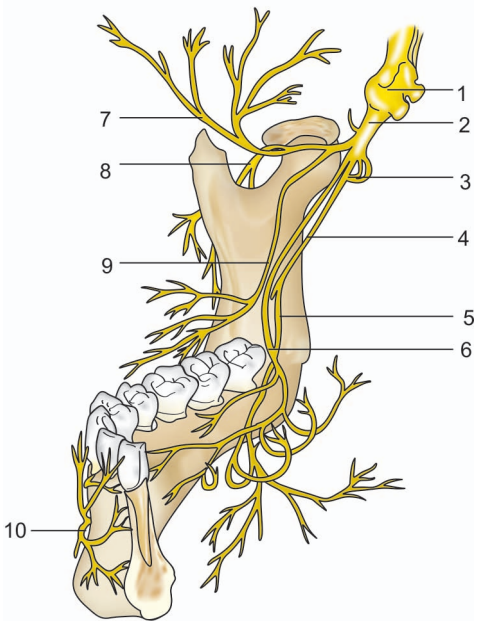


Fig. 3.10: Trigeminal nerve (1) and the branches of its mandibular subdivision (V_3) (2), Auriculotemporal nerve (3), Inferior alveolar nerve (4), Mylohyoid nerve (5), Lingual nerve (6), Deep temporal nerve (7), Masseteric nerve (8), Long buccal nerve (9), and Mental nerve (10).

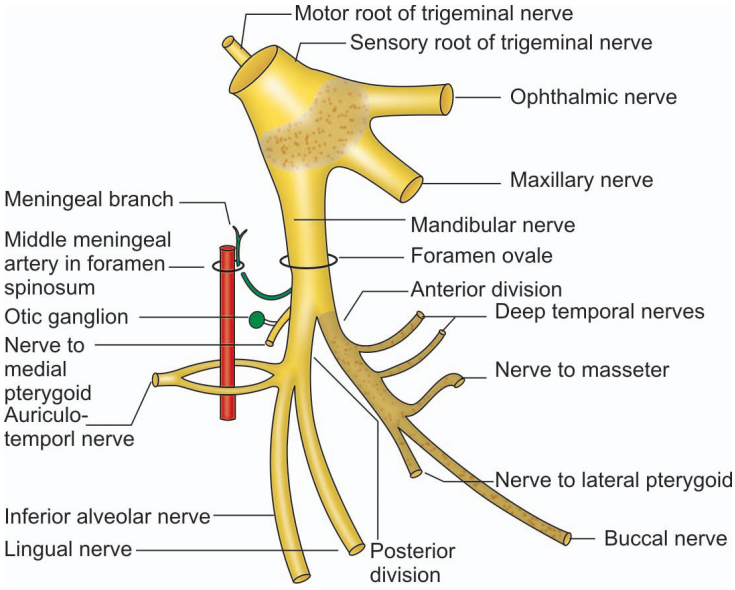


Fig. 3.11: Branches of mandibular nerve

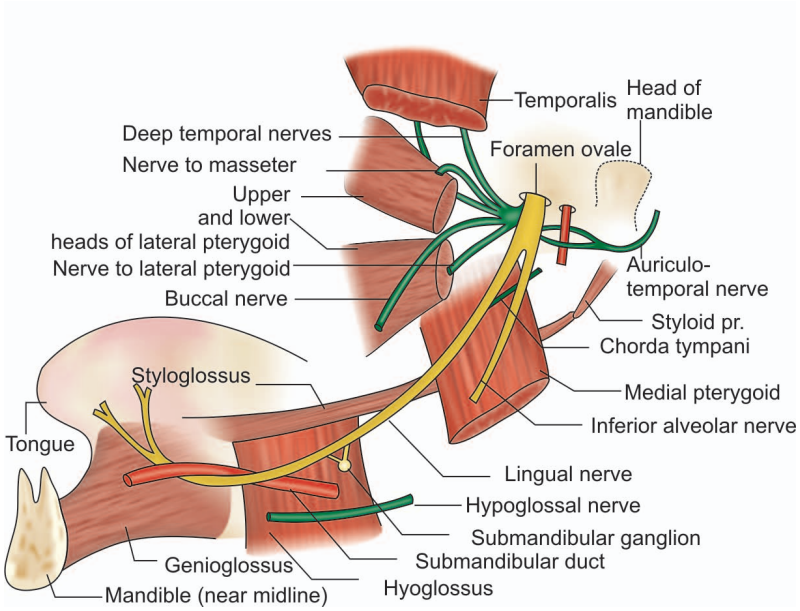


Fig. 3.12: Course and some relations of lingual nerve and other branches of mandibular nerve

Sensory to skin over the mandible, skin of auricle, external part of external auditory meatus, buccal surface of cheek, lower lip, gingivae, lower teeth, floor of mouth and tongue. The motor root supplies muscles of mastication.

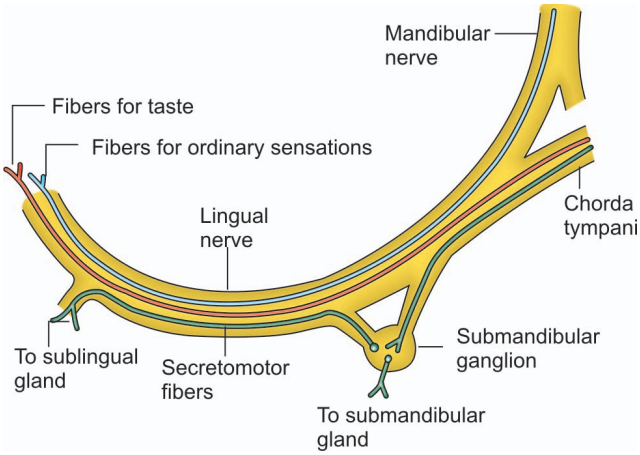


Fig. 3.13: Course of the three different types of fibers carried by the lingual nerve

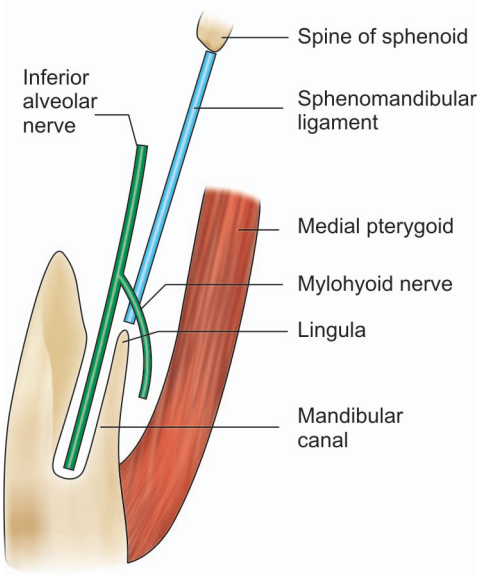


Fig. 3.14: Coronal section through ramus of the mandible to show how the inferior alveolar nerve enters the mandibular canal

Origin, course and branches:

1. *Sensory root:* It originates at the inferior angle of trigeminal ganglion in middle cranial fossa.
2. *Motor root:* It arises in motor cells located in pons and medulla oblongata.

The two roots emerge from the cranium, separately, through foramen ovale, the small motor root lying medial to sensory root. They unite just

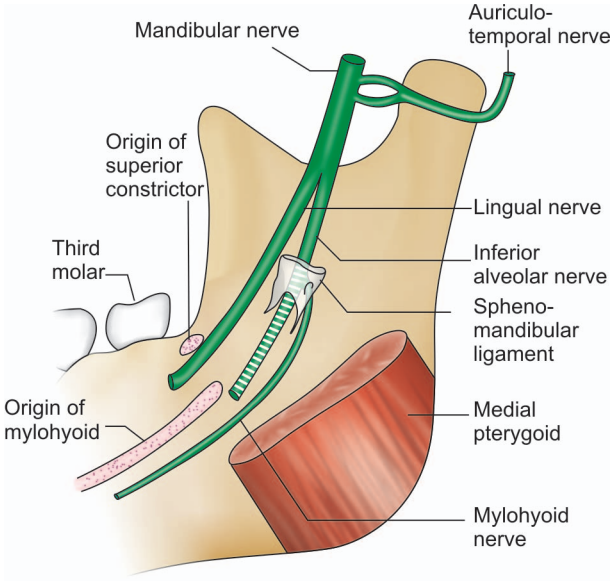


Fig. 3.15: Lingual, inferior alveolar and auriculotemporal nerves viewed from the medial side

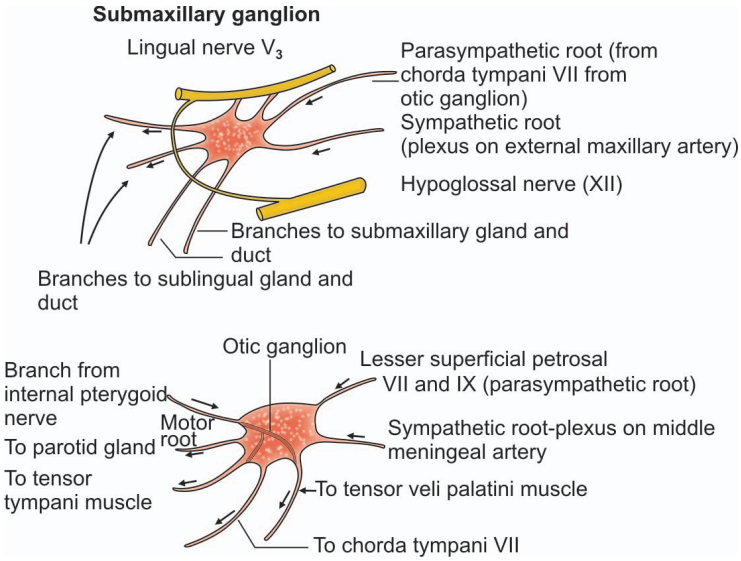


Fig. 3.16: Connexons of submandibular and otic ganglia

outside or at the foramen ovale, and form the main trunk of V_3. The main trunk remains undivided only for a short distance of 2-4 mm, and then it divides into its major branches:

Table 3.2: Old and New terms

	Old terms	*New terms*
Nerve and vessels	Long sphenopalatine nerve and vessels	Nasopalatine nerve and vessels
	Short sphenopalatine nerve and vessels	Posterior Superior Lateral Nasal nerve and vessels
	Anterior palatine nerve and vessels	Greater palatine nerve and vessels
	a. Middle palatine nerve and vessels	Lesser palatine nerves and vessels
	b. Posterior palatine nerve and vessels	
Nerve	Buccinator/Buccal nerve	Long buccal nerve
Ganglia	Sphenopalatine ganglion	Pterygopalatine ganglion
	Submaxillary ganglion	Submandibular ganglion
Vessels	External maxillary artery	Facial artery
	Anterior facial vein	Facial vein
	Posterior facial vein	Retromandibular vein
Muscles	a. Muscles of facial expression	
	Quadratus labii superioris	Levator labii superioris
	Caninus	Levator anguli oris
	Triangularis	Depressor anguli oris
	Square muscle of the lip	Depressor labii inferioris
	b. Muscles of mastication	
	External pterygoid muscle	Lateral pterygoid muscle
	Internal pterygoid muscle	Medial pterygoid muscle

i. *Small anterior trunk:* Its branches are all motor except one.
ii. *Large posterior trunk:* Its branches are all sensory except one.

The mixed nerve, emerges from foramen ovale, into infratemporal fossa between upper head of lateral pterygoid muscle and tensor palati; which lies on the side wall of nasopharynx.

Branches

It gives off branches in three areas:
A. Branches from the Undivided Trunk
B. Branches from the Anterior Trunk
C. Branches from the Posterior Trunk

Branches from the Undivided Trunk

These are two branches: (a) Meningeal branch, and (b) Nerve to medial pterygoid muscle.

i. *Meningeal branch (nervous spinosus):* It is the nerve of first pharyngeal pouch. It passes upwards and re-enters the cranium through foramen spinosum along with middle meningeal artery.

Here it supplies: (i) The cartilaginous part of eustachian tube. (ii) In the middle cranial fossa; it supplies—dura mater in the posterior half; (iii) Then it passes between squamous and petrous parts of temporal bone to supply mastoid air cells.

ii. *Nerve to medial pterygoid:* It enters the deep surface of the muscle. It has a branch that passes close to otic ganglion and supplies the two tensor muscles; tensor tympani and tensor veli palatini.

Branches from the Anterior Trunk

The anterior trunk is significantly smaller than the posterior trunk. The branches provide: (i) motor innervation to the muscles of mastication, and (ii) sensory innervation to mucous membrane of cheek and buccal gingivae of mandibular molars.

Motor Branches

While under the lateral pterygoid muscle, the nerve gives off several branches providing motor innervation to respective muscles.

i. *Deep temporal nerves:* These nerves pass above the upper head of lateral pterygoid, turn above infratemporal crest, and sink into deep surface of temporalis. (These branches are further divided into anterior and posterior deep temporal nerves).

ii. *Nerve to masseter:* Likewise passes above the upper head of lateral pterygoid, proceeds laterally behind temporalis and through mandibular notch to sink into masseter. It gives off a branch to temporomandibular joint.

iii. *Nerves to lateral pterygoid:* One nerve to each head of the muscle.

Sensory Branch

The anterior trunk runs forwards under lateral pterygoid muscle for a short distance and then reaches the external surface of that muscle by either passing between its two heads, or less frequently, winding over its upper border. From this point it is known as long buccal nerve.

Long Buccal Nerve (Buccal/Buccinator nerve)

It is the only sensory branch of anterior trunk and the only nerve to pass between the two heads of lateral pterygoid muscle. It passes down, deep to temporalis, on lower head of lateral pterygoid; and emerges under the anterior border of masseter muscle, continuing in an anterolateral direction.

At the level of occlusal plane of mandibular third or second molar, it crosses in front of ramus of mandible, and enters the cheek through buccinator muscle.

Cutaneous Branch

Long Buccal Nerve gives off a cutaneous branch: A sensory branch to a small area of skin over the soft cheek immediately below zygomatic bone. After piercing buccinator, it supplies the mucous membrane adherent to deep surface of muscle; passes into the retromolar triangle; and ends by supplying sensory innervation to buccal gingivae of mandibular molars and adjoining muco-buccal fold. It carries secretomotor fibers to the molar and buccal salivary glands from otic ganglion.

Branches from the Posterior Trunk

It is primarily sensory, with a small motor component. It descends for a short distance, downward and medially to lateral pterygoid muscle, at which point it branches into:

(1) Auriculotemporal, (2) Lingual, and (3) Inferior alveolar nerves.

Auriculotemporal Nerve

It arises immediately beneath foramen ovale, by two roots that pass back around middle meningeal artery. It traverses upper part of parotid gland; between temporomandibular joint and external auditory meatus, crosses posterior portion of zygomatic arch; and ascends close to superficial temporal artery.

It supplies the temporomandibular joint, parotid fascia, skin of the temple, most of the skin of external auditory meatus and tympanic membrane. For a short distance between foramen ovale and parotid gland, it carries parasympathetic fibers originating in inferior salivatory nucleus through otic ganglion to parotid gland.

Branches: It gives off a number of branches, all of which are sensory. These include:

i. Communication with facial nerve: It provides sensory fibers to the skin over the area of innervation of the following motor branches of facial nerve: Zygomatic, buccal, and mandibular branches.
ii. Communication with otic ganglion: It curves around the neck of mandible, and communicates with otic ganglion, providing sensory, secretory and vasomotor fibers to parotid gland.
iii. Anterior auricular branches: These branches supply the skin over the helix and tragus of the ear.
iv. Branches to external auditory meatus: These branches innervate skin over the meatus and tympanic membrane.
v. Articular branches: These branches pass backward deep to neck of mandible and supply posterior portion of temporomandibular joint.
vi. Superficial temporal branches: These branches run upwards over the root of zygomatic process of temporal bone, behind superficial temporal vessels; and supply hairy skin over temporal region/scalp.

Lingual Nerve

It is the second branch of posterior trunk of V_3. It passes downwards medial to lateral pterygoid muscle; and, as it descends, lies between the ramus and the medial pterygoid muscle in pterygomandibular space. It runs anterior and medial to inferior alveolar nerve, whose path it parallels. It then continues downwards and forwards, deep to pterygomandibular raphe and below the attachment of superior constrictor of pharynx, to reach the side of the base of tongue, slightly below and behind and medial to mandibular third molars. There, it lies below mucous membrane in the lateral lingual sulcus.

It then proceeds anteriorly in the floor of the mouth, winding around submandibular (Wharton's) duct; passing first lateral, then beneath, then medial to the duct across muscles of tongue looping downwards and medial to submandibular duct, to deep surface of sublingual gland, where it breaks up into its terminal branches.

Chorda Tympani

Lingual nerve is joined by chorda tympani (a branch of facial), about 2 cm below the base of skull, deep to lower border of lateral pterygoid muscle. It carries visceral sensory and secretomotor/parasympathetic fibers. The parasympathetic fibers originate in Superior Salivatory Nucleus and relay in the submandibular ganglion, to the sublingual and submandibular salivary glands. The visceral sensory fibers pass to the facial nerve (see Table 3.1).

Lingual nerve provides sensory innervation to:

i. Gingivae on the lingual side of mandible, right from the last molar to the central incisor up to the midline,
ii. Mucous membrane of floor of mouth,
iii. Sublingual and submandibular salivary glands,
iv. Mucous membrane of the anterior 2/3rds of the tongue for both general sensation, as well as special sensation of taste (gustation), from anterior 2/3rds of the tongue, and
v. It carries parasympathetic secretomotor fibers to submandibular ganglion.

Inferior Alveolar Nerve

It is the largest branch of mandibular nerve. It descends medial or deep to lower head of lateral pterygoid muscle; and posterolateral to lingual nerve to the region between sphenomandibular ligament and medial surface of ramus of mandible; where it enters mandibular canal at the level of mandibular foramen. Throughout its path, it is accompanied by inferior

alveolar artery (a branch of internal maxillary artery) and inferior alveolar vein. The artery lies just anterior to the nerve. In the mandibular canal, the three structures together are referred to as "Inferior Alveolar Neurovascular Bundle".

It supplies the following structures:

(i) Inferior portion of the ramus of the mandible (ii) Entire body of the mandible (iii) Pulps of the mandibular incisors, canines, premolars, and molars.

The nerve, artery and vein travel anteriorly in mandibular canal, as far forward as mental foramen, which is located at a point below and between the roots of the premolars, where the nerve divides into its terminal branches, (i) mental nerve, and (ii) incisive nerve.

i. *Mental nerve:* It emerges from the mandibular canal through the mental foramen in the form of a major bulk and divides into three branches that innervate (i) skin of chin, (ii) skin and mucous membrane of lower lip, and (iii) buccal mucosa from the incisor to the premolars. It carries a few secretomotor fibers from chorda tympani to labial minor salivary glands.
ii. *Incisive nerve:* It is the smaller terminal branch and the continuation of inferior alveolar nerve, within the substance of the body of the mandible, anterior to the mental foramen. It supplies the pulps of anterior teeth, central and lateral incisors, and canine, and sometimes the first bicuspid, supporting alveolar bone, periodontal ligament, and the overlying soft tissues anterior to the mental foramen.

It is commonly found that the mandibular central incisor has a dual nerve supply, from the incisive nerve on its own side and from the terminal twigs of the incisive nerve of the opposite side.

Mylohyoid Nerve

It branches off from inferior alveolar nerve prior to its entry into mandibular canal. It runs downwards and forwards in mylohyoid groove on medial surface of ramus and body of mandible to reach mylohyoid muscle.

It is a mixed nerve, being motor to two of the suprahyoid muscles; mylohyoid and anterior belly of digastric muscles. It is thought to contain sensory fibers for the skin on inferior and anterior surface of mental protuberance.

Rarely, it provides sensory innervation to mandibular incisors, mandibular molars, and very rarely, mesial root of mandibular first molar.

BIBLIOGRAPHY

1. Atkinson ME, White FH. Principles of Anatomy and Oral Anatomy for Dental Students. Churchill Livingstone 1992.
2. Decker GAG, du Plessis DJ. Lee McGregor's Synopsis of Surgical Anatomy. 12th ed. Mumbai (India): KM Varghese Co 1986.
3. DuBrul EL. Sicher and DuBrul's Oral Anatomy. 8th ed. Ishiyaku EuroAmerica Inc. USA: ITBS Publishers and Distributors 2000.
4. Last RJ. Anatomy: Regional and Applied. 8th ed. Churchill Livingstone, 1990.
5. Monkhouse WS. Cranial Nerves: Functional Anatomy. Study Notes from the Royal College of Surgeons of Ireland, England 1995.
6. Nevin Mendel, Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1954.
7. Nevin Mendel, Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1948.
8. Sinnatamby CS. Last's Anatomy: Regional and Applied. 10th ed. International ed. Churchill Livingstone 2000.

4 Fundamentals of Nerve Impulse Generation and Transmission

ACTIONS OF LOCAL ANESTHETIC AGENTS

The local anesthetic agents act in the following ways. They prevent both (i) the generation, and (ii) the conduction of nerve impulses.

ANATOMY OF A NERVE CELL OR A NEURON

It is the structural unit of nervous system. It transmits messages between central nervous system and all parts of body.

Types

There are two basic types of neurons. The basic structure of these neurons differs significantly.

(1) Sensory (afferent) neuron, and (2) Motor (efferent) neuron.

Sensory Neuron

It transmits sensation of pain. It consists of 3 major portions (Fitzgerald, 1992):

Dendritic Zone

A neuron is composed of arborisations of free nerve endings, and is the most distal segment of sensory neuron. These free nerve endings respond to stimulations produced in the tissues in which they lie, provoking an impulse that is transmitted centrally along the axon.

Axon

It is a thin cable-like structure; that may be quite long (the giant axon has been measured to be 100-200 cm). At its medial or central end there is an arborisation similar to that seen in dendritic zone.

It is a single nerve fiber, which is in the form of a long cylinder of neural cytoplasm (axoplasm), encased in a thin sheath, the nerve membrane, or axolemma.

Cell Body or Soma

In a sensory neuron, the cell body is located at a distance from the axon, or the main pathway of impulse transmission in the nerve, or their cell bodies are interposed between axon and dendrites. The cell body of sensory neuron, is therefore, not involved in the process of impulse transmission. The primary function of cell body is to provide metabolic support to the entire neuron.

Axoplasm

It is the neural cytoplasm and is separated from extracellular fluids by a continuous nerve membrane, known as axolemma.

Motor Neuron

The motor neurons are structurally different from sensory neurons. These nerve cells conduct impulses from central nervous system peripherally. The motor neurons are an integral component of impulse transmission system but also provide metabolic support to the cell.

Nerve Cell Membrane

In some nerves, this membrane is covered by an insulating lipid-rich layer of myelin. The configuration of a biological nerve membrane is as follows:

a. *Thickness:* The thickness is 70-80 Å (1 angstrom unit is 1/10,000 of a micrometer).
b. *Structure:* It consists of two layers of lipid molecules (bilipid layer of phospholipids) and associated proteins, lipids, and carbohydrates. The lipids are oriented with their hydrophilic (polar) ends facing the outer surface; and the hydrophobic (non-polar) ends projecting towards the middle of membrane.

Proteins

Proteins are considered as primary organizational elements of nerve membranes (Singer and Nicholson, 1972). Proteins are classified as (a) transport proteins (channels, carriers, or pumps), and (b) receptor sites.

Channel Proteins

These are continuous pores through the nerve membranes. Under proper circumstances, these channels allow some ions (Na^+, K^+, and Ca^{++}) to flow passively; whereas other channels are "gated", permitting ion flow only when the gate is "open." (Noback et al, 1991).

The nerve membrane lies at the interface between extracellular fluid (ECF) and axoplasm. It separates highly diverse ionic concentrations within

the axon from the outside. The resting nerve membrane has electric resistance about 50 times greater than that of intracellular fluid (ICF) and extracellular fluid (ECF), thus preventing passage of Na^+, K^+, and Cl^- ions down their concentration gradients.

When a nerve impulse passes, the electrical conductivity of the nerve membrane increases approximately 100-fold. This increase in the conductivity permits the passage of Na^+ and K^+ ions along their concentration gradients through the nerve membrane. It is the movement of these ions that provides immediate source of energy for impulse conduction along the nerve.

Current Concepts

Changes Occurring at the Nerve Membrane

The nerve excitability and conduction are both attributed to the changes developing within the nerve membrane. The cell body and axoplasm are not essential for nerve conduction. They are important, however, as the metabolic support of membrane is probably derived from axoplasm.

Channel Specificity

Recent evidence indicates that channel specificity exists, in that sodium channels differ from potassium channels (Hille, 1975). The gates on sodium channels are located near external surface of nerve membrane, whereas those on potassium channels, are located near the internal surface of nerve membrane.

Types of Nerve Fibers

Myelinated Nerve Fibers

These fibers are enclosed in spirally wrapped layers of lipoprotein myelin sheaths, which are actually a specialized form of Schwann cell. Each myelinated nerve fiber is enclosed in its myelin sheath. The outermost layer of myelin consists of Schwann cell cytoplasm and its nucleus. There are constrictions located at regular intervals (approximately every 0.5-3 mm) along the myelinated nerve fiber. These are called as "Nodes of Ranvier". They form a gap between the two adjoining Schwann cells and their myelin spirals (Denson and Mazio, 1991). At these nodes nerve membrane is exposed directly to extracellular compartment (ECC). The insulating properties of myelin sheath enable a myelinated nerve to conduct impulses at a much faster rate than an unmyelinated nerve of equal size.

Unmyelinated Nerve Fibers

These fibers are also surrounded by Schwann cell sheath. The groups of unmyelinated nerve fibers share the same sheath.

PHYSIOLOGY OF PERIPHERAL NERVES

Function

The function of a nerve is to carry messages from one part of body to another. These messages are in the form of electrical action potentials and are called "impulses". Impulses are initiated by chemical, thermal, mechanical, or electrical stimuli.

Electrophysiology of Nerve Conduction

Nerve conduction is the self-propagated passage of an electric current or an impulse along the nerve fibers. The conduction of an impulse by a nerve depends upon the electric potential that exists across the nerve membrane. The electrical events that occur within a nerve during conduction of an impulse are described in stepwise manner in the following way, as described by Hodgkin and Huxley (1954).

Action Potentials

These are transient membrane depolarizations that result from a brief increase in permeability of membrane to sodium ion, and usually also from a delayed increase in permeability to potassium ion (Heavner, 1991).

Resting State

Phase 1

The unstimulated nerves or the nerves at rest, are said to possess an electric potential across the nerve membrane and is known as resting potential (RP).

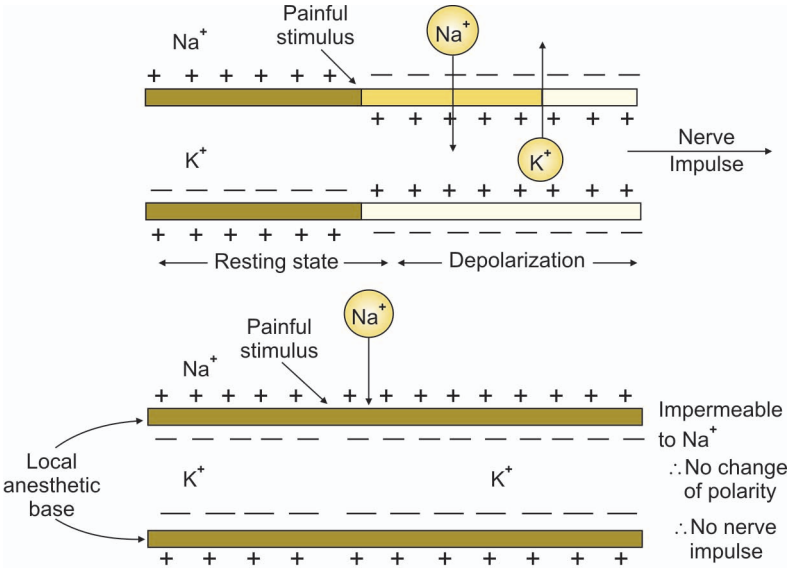
This electrical potential is negative and is usually –70 to –90 mV. It indicates that the inside of nerve membrane becomes 70 to 90 mV more negative than the outside (Figs 4.1 and 4.2).

This difference in the concentration of ions on either side of the nerve membrane creates a potential electrical difference (Table 4.1). This potential difference results in the inside of the cells to be negative and the outside positive. This is achieved by:

i. Active diffusion of ions through the membrane, and
ii. Passive diffusion; diffusion of ions across the nerve membrane because of a difference in the electrical gradient.

Table 4.1: Showing the concentrations of intracellular and extracellular ions

Ions	*Intracellular mEq/L*	*Extracellular mEq/L*	*Approximate Ratio*
Sodium (Na^+)	5-10	140	1:14
Potassium (K^+)	110-170	3-5	27:1
Chloride (Cl^-)	5-10	110	1:11



Figs 4.1A and B: Mode of action of local anesthetic agents (A) The diagram shows that the nerve at the resting state and getting depolarized after application of painful stimulus which creates a nerve impulse. (B) When the nerve membrane is stabilized by the local anesthetic agent, depolarization is prevented and no impulse is conducted (Adapted from Local Anesthesia in Dentistry by Howe GL and Whitehead FIH, Dental Practitioner Handbook No. 14, 3rd ed. 1997)

The table shows the differing concentrations of ions found within nerve cells and in extracellular fluids. Significant differences exist for ions between their intracellular and extracellular concentrations. These ionic gradients differ because the nerve membrane exhibits selective permeability.

At rest, there are greater number of anions (–) inside the cell membrane and an equal number of cations (+) outside the nerve membrane. Thus K^+ ions are concentrated inside, while Na^+ and Cl^- are concentrated outside the membrane.

The resting potential of the nerve is due to the relative permeability of cell membrane to K^+ ions and its relative impermeability to Na^+ ions. The nerve membrane is freely permeable to K^+ ions, which remains within the axoplasm.

K^+ ions are positively charged and are retained by electrostatic mechanism to the negatively charged nerve membrane. Chloride ions remain outside as a result of opposing electrostatic mechanism. The maintenance of resting potential is mainly due to an active mechanism known as "sodium pump". It moves Na^+ from an area of lesser concentration inside to an area of greater concentration outside. This pump controls the concentration of Na^+ ions on both sides of the membrane and maintains it in the polarized state.

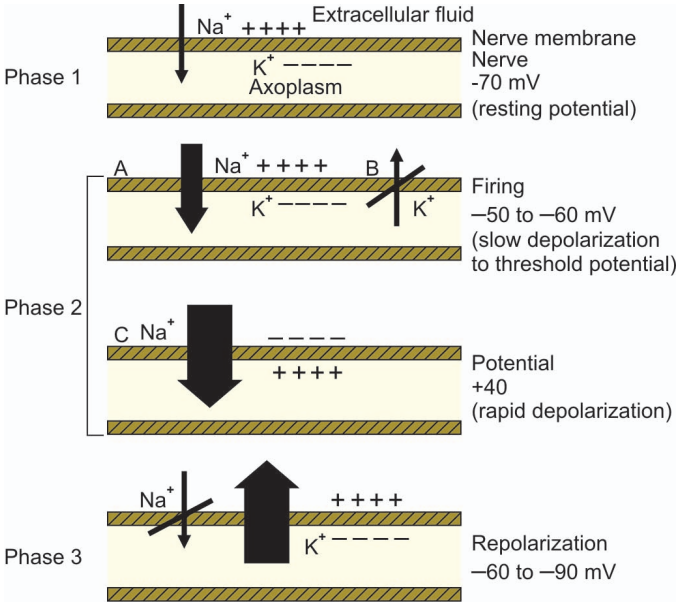


Fig. 4.2: Phase 1: Nerve membrane at rest showing resting potential. Phase 2: A and B show slow depolarization to threshold potential. C. rapid depolarization. Phase 3: Repolarization (Adapted from Handbook of Local Anesthesia, by Stanley Malamed, 4th ed. 2001)

Phase 2

Depolarization (Hodgkin and Huxley, 1954)

When a stimulus of sufficient intensity is applied to the nerve, it gets excited and the following events occur in sequence.

i. *Slow depolarization:* This is the initial phase wherein the electrical potential inside the nerve becomes slightly less negative (Fig. 4.2, Phase 2A).
ii. *Threshold potential or firing threshold or rapid depolarization:* The electrical potential which is becoming less negative, reaches a critical level, it results in an extremely rapid phase of depolarization (Fig. 4.2, Phase 2B).
iii. *Reversal of electrical potential:* With the phase of rapid depolarization, there is a reversal of electrical potential across the nerve membrane. The interior of the nerve is now electrically positive and the exterior is negative. There is an electrical potential of +40 mV on the interior of the nerve cell (Fig. 4.2, Phase 2C).

The interior of nerve is now electrically positive in relation to exterior.

Phase 3

Repolarization

It occurs at the end of the various phases of depolarization.

The electrical potential gradually becomes more negative inside the nerve cell relative to outside until the original resting potential of –70 mV is restored (Fig. 4.2).

Duration of the Complete Cycle

The entire process (phases 2 and 3) takes 1 msec; depolarization phase (phase 2) takes 0.3 msec, while repolarization phase (phase 3) takes 0.7 msec.

ELECTROCHEMISTRY

The sequence of events that occur across the nerve membrane depend on the following factors:

1. Concentration of electrolytes in axoplasm (interior of cell); and extracellular fluids (ECF); and
2. Permeability of nerve membrane to sodium and potassium ions.

Resting State

In resting state, the nerve membrane is:

i. Slightly permeable to sodium ions.
ii. Freely permeable to potassium ions.
iii. Freely permeable to chloride ions.

Potassium ions remain within axoplasm despite its ability to diffuse freely through nerve membrane; and despite its concentration gradient (passive diffusion usually occurs from a region of greater concentration to a region of lesser concentration), because negative charge of nerve membrane restraints the positively charged ions by electrostatic attraction.

Chloride ions remain outside the nerve membrane instead of moving along its concentration gradient into nerve cell because the opposing, nearly equal, electrostatic influence (electrostatic gradient: From inside to outside) forces outward migration. The net result is no diffusion of chloride ions through the membrane.

Sodium ions migrate inwardly because both the concentration gradient (greater outside) and electrostatic gradient (positive ion attracted by negative intracellular potential) favor such migration. Only the fact that resting nerve membrane is relatively impermeable to sodium prevents a massive influx of this ion.

Membrane Excitation

Depolarization

Excitation of a nerve segment leads to an increase in permeability of cell membrane to sodium ions. This is accomplished by transient widening of transmembrane ion channels sufficient to permit hydrated sodium ions.

The rapid influx of sodium ions to interior of nerve cell causes a depolarization of nerve membrane from its resting level to its firing threshold of approximately –50 to –60 mV (Fig. 4.2, Phases 2A and B) (Noback and Demarest, 1981).

The firing threshold is actually the magnitude of decrease in negative transmembrane potential that is required to initiate an action potential (impulse).

A decrease in negative transmembrane potential of 15 mV (i.e. from –70 to –55 mV) is required to reach firing threshold (FT); a voltage difference less than 15 mV will initiate an impulse.

In a normal nerve, firing threshold remains constant. Exposure of a nerve to a local anesthetic raises its firing threshold. Elevating firing threshold means more sodium must pass through membrane to decrease negative transmembrane potential to a level when depolarization occurs.

When firing threshold is reached, permeability of membrane to sodium increases dramatically, and sodium ions rapidly enter axoplasm. At the end of depolarization (the peak of action potential) the electrical potential of nerve is actually reversed as an electrical potential of + 40 mV exists (Fig. 4.2, Phase 2C). The entire depolarization process requires approximately 0.3 msec.

The initiation of the changes occur as a result of displacement of calcium ions from their phospholipid-binding site. Then the marked increase in the diffusion of Na^+ into the cell is followed by the passage of K^+ out of the cell. This action is said to abolish the resting potential and depolarize the membrane. It has been said that the term "depolarized" is inaccurate; as there is actually a reversal of polarity, the outside becomes negative relative to inside, resulting in a potential (reversal potential) of about twice that of the resting potential. The alteration in the permeability of the nerve membrane is believed to be due to the result of liberation of a transmitter, acetylcholine, at the site of the stimulation.

Repolarization

The action potential is terminated when membrane repolarizes. This is caused by the inactivation of increased permeability to Na^+, while the increased permeability to K^+ is restored. This helps in restoring the original electrochemical equilibrium and the resting potential. In many cells,

permeability to potassium ions also increases, resulting in efflux of potassium ions, leading to more rapid membrane repolarization and return to its resting potential.

The movement of sodium ions into the cell during depolarization and subsequent movement of potassium ions out of cell during repolarization are passive (non-energy requiring) actions, since each ion moves along its concentration gradient (e.g. from an area of high concentration to an area of low concentration).

Following the return of membrane potential to its original level (–70 mV), a slight excess of Na^+ exists within the nerve cell, and a slight excess of K^+ exists extracellularly. A period of metabolic activity then begins. Active transfer of Na^+ ions out of cell occurs via the sodium-pump. And expenditure of energy is needed to move sodium ions out of nerve cell against their concentration gradient. This energy comes from oxidative metabolism of adenosine triphosphate (ATP). The same pumping mechanism is thought to be responsible for the active transport of K^+ ions into the cell against their concentration gradient. The entire process of repolarization requires 0.7 msec.

Absolute Refractory Period (ARP)

Immediately, after a stimulus has initiated an action potential, a nerve, is unable, for a time, to respond to another stimulus, regardless of its strength. This is termed absolute refractory period, and it lasts for about the duration of main part of action potential.

Relative Refractory Period (RRP)

The ARP is followed by a relative refractory period, during which a new impulse can be initiated but only by a stronger than normal stimulus. The RRP continues to decrease until the normal level of excitability returns, at which time the nerve is said to be repolarized.

During depolarization the major portion of ionic sodium channels are found in their open (O) state, thus permitting rapid influx of sodium ions. This is followed by a slower decline into a state of inactivation (I) of the channels to non-conducting state.

Inactivation temporarily converts the channels to a state from which they cannot open in response to depolarization (ARP). This inactivated state is slowly converted back, so the majority of channels are found in their closed (C) resting form when the membrane is repolarized (–70 mV).

Upon depolarization the channels change configuration, first to open ion-conducting (O) state and then to an inactive non-conducting (I) state. Although both (C) and (I) states correspond to non-conducting channels, they differ in that depolarization can recruit channels to conducting (O) state from (C) but not from (I) states (sodium channel transition stages) (Keynes, 1979).

MEMBRANE CHANNELS

Discrete aqueous pores through the excitable nerve membrane are called sodium (or ion) channels, which are molecular structures that mediate its permeability.

A channel is a lipoglycoprotein firmly situated in the membrane. It consists of an aqueous pore spanning the membrane, that is narrow enough at least at one point to discriminate between sodium and other ions (sodium ions pass through 12 times more easily than potassium ions).

The channel also includes a portion that changes configuration in response to changes in membrane potential, thereby gating the passage of ions through the pore (C, O and I states described above). The presence of these channels helps explain membrane permeability or impermeability to certain ions. Sodium channels have an internal diameter of approximately 0.3 × 0.5 nm (Cattarall, 1988).

A sodium ion is "thinner" than either a potassium ion or a chloride ion, and should therefore diffuse freely down its concentration gradient through membrane channels into nerve cell. This does not occur, however, because all these ions attract water molecules and thus become hydrated. Hydrated sodium ions have a radius of 3.4 Å, which is approximately 50% greater than 2.2 Å radius of potassium and chloride ions.

Sodium ions are therefore too large to pass through the narrow channels when a nerve is at rest. Potassium and chloride ions can pass through these channels.

During depolarization sodium ions readily pass through the nerve membrane because configurational changes that develop within a membrane produce a transient widening of these transmembrane channels to a size adequate to allow the unhindered passage of sodium ions down their concentration gradient into axoplasm (transformation from the C to O configuration). This concept can be visualized at the opening of a gate during depolarization that is partially occluding the channel in resting membrane.

Channel specificity: Recent evidence indicates that channel specificity exists, in that sodium channels differ from potassium channels (Hille, 1975). The gates on sodium channels are located near external surface of nerve membrane, whereas those on potassium channels, are located near internal surface of nerve membrane.

Impulse Propagation

Following initiation of an action potential by a stimulus, the impulse moves along the surface of axon. Energy for impulse propagation is derived from nerve membrane in the following manner: The stimulus disrupts the resting equilibrium of nerve membrane, the TMP—is reversed momentarily; the

interior of cell changing from negative to positive, the exterior changing from positive to negative. This new electrical equilibrium in this segment of nerve produces local currents that begin flowing between depolarized segment and the adjacent resting area. These local current flow from positive to negative, extending for several mm along the nerve membrane.

As a result of this current flow, the interior of adjacent area becomes less negative and exterior less positive. Transmembrane potential decreases, approaching firing threshold for depolarization. When transmembrane potential is reduced by 15 mV from resting potential, firing threshold is reached and complete depolarization occurs.

The newly developed segment sets up local currents in adjacent resting membrane, and the entire process starts a new.

Conditions in segment that has just depolarized return to normal following absolute refractory period (ARP) and relative refractory period (RRP). Because of this the wave of depolarization can spread in only one direction. Backward (retrograde) movement is prevented by unexcitable, refractory segment.

Impulse Spread

The propagated impulse travels along the nerve membrane towards central nervous system. The spread of this impulse differs depending on whether or not a nerve is myelinated.

Unmyelinated Nerves

An unmyelinated nerve is basically a long cylinder with high electrical resistance cell membrane, surrounding a low resistance conducting core of axoplasm, all of which is bathed in low resistance extracellular fluid.

The high resistance cell membrane and low resistance intracellular and extracellular media produce a rapid decrease in density of current within a short distance of depolarized segment. In areas, immediately adjacent to this depolarized segment, local current flow may be adequate to initiate depolarization in resting membrane. Farther away, it will prove to be inadequate to achieve firing threshold.

The spread of an impulse in unmyelinated nerve fiber, is therefore, characterized by a relatively slow forward-creeping process. The conduction rate in unmyelinated C fibers is 1.2 m/sec as compared to 14.8 to 120 m/sec in myelinated A –α and A –δ fibers (Ritchie, 1984).

Myelinated Nerves

Impulse spread within myelinated nerves differs from that in unmyelinated nerves, because of the layer of insulting material separating intracellular and extracellular charges. The farther apart the charges, the smaller will

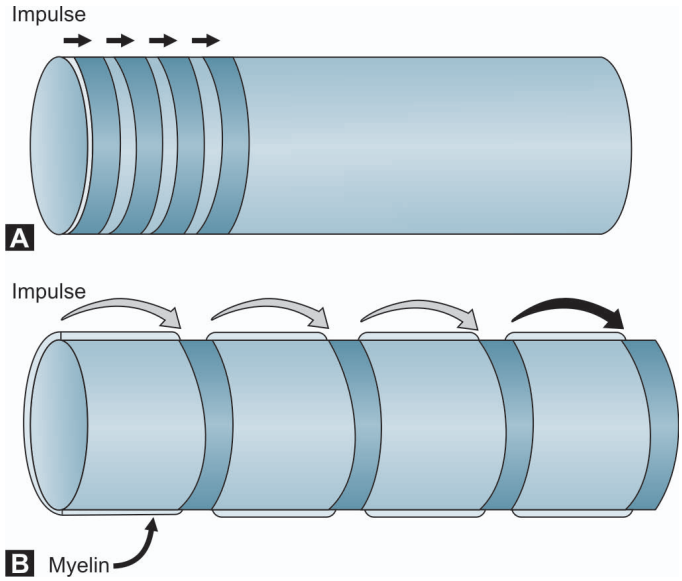
be the current required to charge the membrane. Local currents can thus travel much farther in a myelinated nerve than in an unmyelinated nerve before becoming incapable of depolarizing the nerve membrane ahead of it.

Impulse conduction in myelinated nerves occurs by means of current leaps from node to node, a process termed as "saltatory conduction" ("saltare" latin verb "to leap"). This form of impulse conduction proves to be much faster and more energy efficient than that which is employed in unmyelinated nerves.

The thickness of myelin sheaths increases with increasing diameter of axon. The distance between adjacent nodes of Ranvier increases with greater axonal diameter. Because of these two factors, saltatory conduction is more rapid in a thicker axon (Figs 4.3A and B).

Saltatory conduction usually progresses from one node to the next in a stepwise manner. However, it can be demonstrated that the current flow at the next node still exceeds that necessary to reach firing threshold of nodal membrane.

If conduction of an impulse is blocked at one node, the local current will skip over that node and prove adequate to raise membrane potential at the next node to its firing threshold and produce depolarization. A



Figs 4.3A and B: Comparison of impulse propagation in the two types of the nerves. (A) Unmyelinated nerve: The impulse moves in forward direction by depolarization of short adjoining segments of the nerve membrane. (B) Myelinated nerve. The impulse leaps in forward direction from one node of Ranvier to another node. This is known as saltatory conduction. It is to be noted that the myelinated nerve, the impulse is far ahead of that of the unmyelinated nerve (Adapted from Handbook of Local Anesthesia, by Stanley Malamed, 4th edn. 2001)

minimum of perhaps 8-10 mm of a nerve must be covered by the anesthetic solution to ensure thorough blockade (Franz and Perry, 1974).

Tachyphylaxis

It is defined as an increasing tolerance to a drug that is administered repeatedly. It can be easily developed, if nerve function is allowed to return prior to reinjection.

Etiology

It is difficult to give an explanation. However, it is brought about through some or all of the factors such as edema, localized hemorrhage, clot formation, transudation, hypernatremia, and decreased pH of the tissues.

Action of Norepinephrine

Successively repeated doses of these drugs will prove to be less effective than those given previously because of the depletion of norepinephrine stores. This phenomenon is not seen with drugs that act directly on adrenergic receptors.

FACTORS AFFECTING NERVE CONDUCTION

Diffusion of Solution

Local anesthetic agents are alkaloid bases, which are combined with acids, usually hydrochloric acid, to form water-soluble salts. All local anesthetic salts are formed by a combination of a weak base and a strong acid. The purpose of using salts is to render the salts stable and soluble in water. Water solubility is necessary for their diffusion through the interstitial fluids to the nerve fiber.

The salts of local anesthetics in the form of solution, exist as both uncharged molecules, also called the free base (RN), and positively charged molecules, called the cation (RNH^+), in a state of equilibrium with each other; which is represented as:

$$RNH^+ \leftarrow \rightarrow RN + H^+$$

The uncharged base and the charged ionic form, both are involved in the process of nerve penetration and conduction block. The uncharged lipid-stable form is essential for diffusion through the nerve sheath.

Dissociation Constant (pKa)

Local anesthetic agents with high pKa have poor anesthetic quality as the agents have insufficient number of free-base molecules. This is because they have few molecules present as the free base (lipid-soluble form) at normal tissue pH (7.3-7.4).

Injection of Local Anesthetic Agents Close to the Nerve Membrane

For maximum action, the anesthetic agent, should be injected sufficiently close to the target nerve, so that adequate concentration is available for diffusion into the nerve.

pH of the Tissue

The presence of a low pH, as in areas of infection (pH of pus is 5.5 to 5.6); may interfere with achieving adequate anesthesia by preventing deprotonization and liberation of the free base.

Lipid Solubility and Protein Binding

Higher the lipid solubility and percentage of protein binding, more rapid and long lasting are the effects. The addition of a butyl group to procaine results in the formation of tetracaine, an agent having 16 times the anesthetic activity and 4 times the duration of the parent compound.

Type of Nerve

This is an important factor in the development of adequate anesthesia. The myelinated nerves usually require a greater concentration of local anesthetic solution. These nerves also require more time for blocking them. The non-myelinated nerve-fibers require lesser concentration and lesser time. The former are protected by an insulating barrier of myelin and can be reached only at the nodes of Ranvier, which interrupt myelin sheath at every 1-2 mm.

Size of Nerve

The diameter of the nerve also plays an important part. The larger the diameter of nerve fiber, the greater is the concentration required to prevent impulse conduction.

The local anesthetic agents produce loss of function in the following order: Pain, temperature, touch, proprioception and skeletal muscle tone. The return of sensation is in the reverse of the preceding order. Since pain is the only modality of sensation in the tooth, all sensations are adequately eliminated when pain fibers are anesthetised. Analgesia is produced as a result of blockage of afferent transmission, whereas relaxation of skeletal muscles and inhibition of autonomic innervated structures cause blockade from afferent transmission.

Presence of a Vasoconstrictor

Local anesthetic solutions containing a vasoconstrictor are less readily absorbed than those without a vasoconstrictor. The solution which are rapidly absorbed into cardiovascular system, there is less likelihood of developing an adequate block anesthesia.

Injection into a Vascular Area

A local anesthetic solution injected into a highly vascular area is rapidly absorbed in cardiovascular system. The rapid absorption reduces the effective concentration in the vicinity of the nerve and causes a rapid termination of analgesia.

Presence of Infection

Infection reduces alkalinity of tissues which retards the deprotonization of local anesthetic agents. This prevents liberation of free alkaloid base, which is necessary for the development of effective analgesia.

EFFECTIVENESS OF LOCAL ANESTHETIC AGENTS

The effectiveness of local anesthetic agents depends also on the following:

1. The chemical nature of the drug used.
2. The concentration of the drug used.
3. The volume of solution injected.
4. The rate of diffusion of both the anesthetic salt and the free base.
5. Addition of vasoconstrictor. It influences the time during which the free base remains in contact with the nerve.

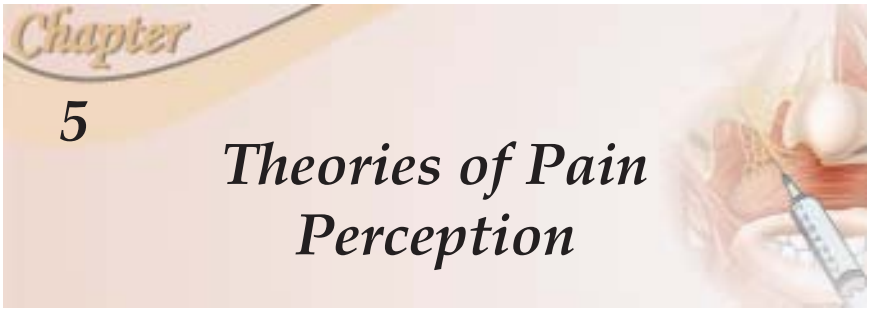
SEQUENCE OF MECHANISM OF ACTION

The following sequence of the mechanism of the action of local anesthetics was proposed by Covino and Vassallo (1976):

1. Displacement of calcium ions from the sodium channel receptor site.
2. Binding of the local anesthetic molecule to this receptor sites.
3. Blockade of the sodium channel.
4. Decrease in the sodium permeability.
5. Depression of the rate of the electric depolarization.
6. Failure to achieve the threshold potential.
7. Lack of development of propagated action potentials.
8. Conduction blockade.

BIBLIOGRAPHY

1. Cattarall WA. Structure and function of voltage-sensitive ion channels. Science 1988;242:50-60.
2. Covino BG, Vassallo HG. Local anesthetics: Mechanisms of action and clinical use. New York: Grune and Stratton 1976.
3. Denson DD, Maziot JX. Physiology, pharmacology and toxicity of local anesthetics: Adult and pediatric considerations. In: Raj PP (ed): Clinical Practice of Regional Anesthesia. New York: Churchill Livingstone 1991.
4. Fitzgerald MJT. Neuroanatomy: Basic and Clinical, London, Bailliere Tindall 1992.
5. Franz DN, Perry RS. Mechanisms for different block among single myelinated and non-myelinated axons by procaine. J Physiol 1974;235:193-210.
6. Heavner JE. Molecular actions of local anesthetics. In: Raj PP (ed): Clinical Practice of Regional Anesthesia. New York: Churchill Livingstone 1991.
7. Hille B. Ionic selectivity, saturation, and block in sodium channels: A four-barrier model. J Gen Physiol 1975;66:535-60.
8. Hodgkin AL, Huxley AF. A quantitative description of membrane current and its application to conduction and excitation in nerve. J Physiol 1954;117: 500-44.
9. Keynes RD. Ion channels in the nerve-cell membrane. Sci Am 1979;240(3): 126-35.
10. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
11. Noback CR, Demarest RJ. The human nervous system: Basic principles of neurobiology. 3rd ed. New York: McGraw-Hill 1981;44-45.
12. Noback CR, Strominger NL, Demarest RJ. The human nervous system: Introduction and Review, 4th ed. Philadelphia, Lea and Febiger 1991.
13. Richard Bennett C. Monheim's local anesthesia and pain control in dental practice. 7th ed. Delhi: CBS Publishers and Distributors 1990.
14. Ritchie JM. Physiological basis for conduction in myelinated nerve fibers. In: Morell P (ed): Myelin, 2nd ed. New York: Plenum Press 1984;117-45.
15. Singer SJ, Nicholson GL. The fluid mosaic model of the structure of cell membranes. Science 1972;175:720-31.



There are various theories put forward to understand the nature of pain. No single pain theory is currently adequate to explain all aspects of pain phenomenon. Each approach still leaves a great many unknown aspects. The theories have focussed on the neurophysiological structures related to pain.

THEORIES OF PAIN PERCEPTION

There are following theories:

1. Specificity Theory
2. Pattern Theory
3. Gate-control Theory.

Specificity Theory

It's a pain system based on a specific set of peripheral nerve fibers that are nociceptive in function. There are sets of free nerve endings at the periphery; A -δ and C fibers. These are associated with two qualities of pain:

i. Short — latency pricking pain and
ii. Long — latency burning pain, respectively.

Pricking pain impulses enter the dorsal spinal cord where they synapse and ascend via the anterolateral system to thalamic centers and from there to somatic sensory areas of the cerebral cortex.

Burning pain impulses follow a similar course into the anterolateral system but are projected to different thalamic, hypothalamic cortical areas. These latter projections seem to account for the affective, autonomic reaction to pain impulses. The nature, location, and interactions of higher pain centers are still not clearly understood.

Pattern Theory

This theory opposes that pain has its own set of specialized receptors. It proposes that pain perception is based on stimulus intensity and central summation (Goldscheider, 1894).

Crue and Corregal (1975), the recent advocates of this theory argue that there is no need to speak of pain as a primary sensory modality. Therefore, there are no pain endings, pain fibers, or pain neurons in the peripheral nervous system. There is no such thing as a pain stimulus, only stimuli that are painful. Pain is a result of the summation of a spatial and temporal pattern of input. Melzack and Wall (1965, 1970) and Melzack (1973) after thorough evaluation reject both the two theories.

Specificity in viewed as making unwarranted physiologic assumptions and do not adequately account for the various aspects of pain phenomena. Pattern theory is described as going against physiological facts.

Melzack and Wall (1965, 1970) reject specificity but accept specialization.

Specialization can be found at receptor sites such as A-δ and C fibers that respond to particular types and ranges of physical energy. However, specialization is not specificity.

Specificity implies responding to one and only one kind of stimulus. The specific receptors, calling these receptors pain fibers implies a direct connection from the receptor to a brain center where pain would always be perceived. This is merely a physiological assumption.

There are a small number of fibers that respond only to intense stimulation. However, this does not mean they are pain fibers that always produce pain when stimulated. There are many things happening at various levels of energy stimulation.

Besides activation of specific fibers, changes occur in the total number of neurons responding as well as in their temporal and spatial relationships. The Patten theory, appears to contradict physiological evidence.

Gate-control Theory

This theory of pain contains elements of both specificity and pattern theories.

It accounts for psychological influences on pain perception as well as certain clinical findings such as spread of pain and persistence of pain after tissue healing.

The concept of this theory is that it proposes a dorsal spinal gating mechanism in the substantia gelatinosa (SG); which modulates the sensory input by the balance of activity of small diameter (A-δ & C) and large diameter (A-B) fibers.

Activity of large fibers closes the gate and prevents synapatic transmission to centrally projecting T (transmission) cells, while small diameter fibers open the gate and facilitate T-cell activity once a critical level is reached.

Small fiber activity is believed to be responsible for prolongation of pain and spread to other parts of the body. A central control trigger can

also influence the gate. Thus cognitive processes can either open or close the gate.

The exact mechanism involved in Gate-control theory is still not clear. Mendell and Wall (1964) and Hillman and Wall (1969) spoke of pre- and post-synaptic effects.

A signal electrical stimulus delivered to small fibers produces a burst of nerve impulses followed by repeatitive discharges in the spinal cord. Successive electrical stimuli produce a burst followed by discharges of increasing duration after each stimulation. Successive electrical stimulation of large fibers produces a burst of impulses followed by period of silence after each pulse.

These studies form much of the mechanical basis of Gate-control theory.

Gate-control theory emphasizes the role of psychological variables and how they affect the reaction to pain.

Successful pain control often involves changing the motivational component while the sensory component remains intact, especially in chronic pain.

DUAL NATURE OF PAIN

All theories of pain, ascribe to the two aspects or the dual nature of pain. The two aspects are as follows: (1) Pain Perception, and (2) Pain Reaction: The variations of pain reaction raise or lower the pain threshold.

Pain Perception

It is the physioanatomical process, whereby an impulse is generated, subsequent to application of an adequate stimulus, and is transmitted to Central Nervous System.

This aspect of pain is quite similar in all healthy individuals; and varies little from day to day, The intensity of stimulus and the duration for which it must be applied are uniform, as is the rate of conduction of impulse for particular types of nerves.

Pain Reaction

This aspect of pain is psychophysiological process. It represents the person's overt manifestations of the unpleasant process of perception. This aspect of pain, depends on complex neuroanatomical and psychological factors involving the cortex, limbic system, hypothalamus and thalamus. These complex factors determine how an individual would react to the unpleasant experience. These factors are analogous to the events triggered and occurring with the system of action as described in Gate-Control theory.

Pain reaction, unlike pain perception varies markedly from individual to individual; and from day to day, in the same individual. The threshold of pain reaction is usually interpreted as being inversely proportionate to pain reaction. A person who is hyporeactive is considered to have a high pain threshold, while a person who has a low pain threshold hyperreacts in response to noxious stimulus.

If pain is to be controlled, both the aspects of the nature of pain have to be considered. The use of only regional analgesia for the control of dental/operative pain in apprehensive patients may be inadequate. These individual, because of anxiety and apprehension, may misinterpret non-noxious stimuli subconsciously.

In many instances, the control of pain perception by the use of local anesthetics must be coupled with the use of various analgesic and psychoactive drugs for control of pain reaction.

Various Factors

There are various factors which have a definitive bearing on an individual's threshold of pain reaction. These are as follows:

Emotional States

The pain threshold of individuals depends to a great degree on their attitude towards the operator, the surroundings and the procedure.

Fatigue

Patients who have taken good rest or who had good sleep the night before the day of procedure have higher threshold to pain reaction.

Age

Older individuals tend to have a higher threshold of pain reaction than younger individuals. However, in cases of extreme of age or senility, pain perception may get affected.

Racial and Nationality Characteristics

Some authors have reported that racial characteristics do reflect in the threshold to pain reaction. Amongst the nationality group, in some reports, Latin Americans and Southern Europeans, are more emotional and therefore, have low threshold to pain reaction than North Americans or Northern Europeans.

Sex

It is a general belief, that men have a higher threshold for pain reaction than women.

Fear and Apprehension

In most instances, the threshold of pain reaction is lower with the increase in fear and apprehension. These patients overreact to the original stimulus; and magnify the pain out of proportion.

Such patients should be treated very carefully. It is necessary for the operator to gain patient's confidence. In addition, psychological and pharmacological methods should be employed to control fear and apprehension in the dental office.

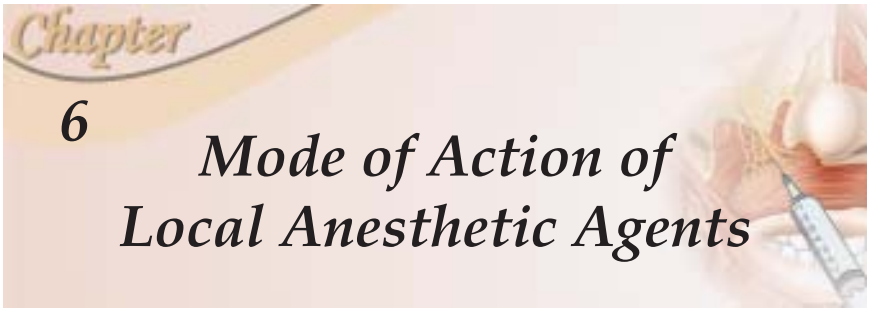
Alternative Methods

The effective alternatives and supplements to pharmacological and surgical methods in control of pain are as follows:

(1) Hypnosis, (2) Anxiety reduction, (3) Desensitization, (4) Attention (5) Distraction, and (6) Other behavioral approaches.

BIBLIOGRAPHY

1. Crue BL Jr, and Carregal EJA. Pain begins in the dorsal horn—with a proposed classification of the primary senses. In Crue BL Jr., ed. Pain: Research and Treatment, New York, Academic Press, Inc.1975.
2. Goldscheider, A. Uber den schmerz in physiologischer und klinescher kinsicht, Berlin, A. Hirschwald 1894.
3. Hillman P, and Wall PD. Inhibitory and Excitatory factors influencing the receptive fields of lamina Spinal Cord cells. Exp Brain Res 1969;9:284.
4. Laskin DM. Oral and Maxillofacial Surgery Vol I. CV Mosby Co. AITBS Publishers India 1992.
5. Mendell, LM, and Wall PD. Presynaptic hyperpolarization: A role for fine afferent fibres. J Physiol 1964;172:274.
6. Melzack R, and Wall PD. Pain mechanisms: A new theory, Science 1965;150:971.
7. Melzack R, and Wall PD. Psychophysiology of Pain. Int Anesthesiol Clin 1970;8:3.
8. Melzack R. The puzzle of pain. New York. Basic Books, Inc, Publishers 1973.
9. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice.7th ed.CBS Publishers and Distributors 1990.



In order to understand the mode of action of local anesthetic agents, it is essential to understand the process of nerve conduction. This has been described in the Chapter on "Fundamentals of Nerve Impulse Generation and Transmission".

MECHANISM OF ACTION

The theories explaining the mechanism of action of local anesthetic agents are:

1. Acetylcholine Theory
2. Calcium Displacement Theory
3. Surface-Charge (Repulsion) Theory
4. Membrane Expansion Theory
5. Specific Receptor Theory

While describing the theories we must first know the proposition of the theory; and then study the evidence available in the light of present scientific studies reported in the literature.

Acetylcholine Theory

Acetylcholine is involved in nerve conduction. Acetylcholine is a neurotransmitter at the nerve synapses. However, there is no evidence that acetylcholine is involved in neural transmission along the body of neuron.

Calcium Displacement Theory

Local anesthesia was produced by displacement of calcium from some membrane sites that controlled permeability to sodium. Studies have shown that variation in concentrations of calcium did not affect local anesthetic potency.

Surface Charge Theory or Repulsion Theory

Local anesthetic agents act by binding to nerve membrane; and changing the electrical potential at the membrane surface. Local anesthetic agents

made electrical potential at the membrane surface more positive, thus reducing the excitability of nerve by increasing threshold potential.

Current evidence showed that:

i. Resting potential of nerve membrane; remains unaltered by local anesthetic agents, i.e. they do not become hyperpolarized.
ii. Conventional local anesthetic agents act within the channels in the nerve membrane rather than at membrane surface.

Membrane Expansion Theory

Local anesthetic molecules diffuse to hydrophobic regions of excitable membrane, expanding some critical regions in nerve membrane; and thus preventing an increase in permeability to sodium ions. This theory explains local anesthetic activity of benzocaine which does not exist in cationic form, yet exhibits potent topical anesthetic activity.

Specific Receptor Theory

It is the most favored theory today. Local anesthetic agents act by binding to specific receptors on sodium channels in the nerve membrane. Biochemical and electrophysiological studies have shown that specific receptor sites exist in sodium channels either, on: (i) External surface or (ii) Internal surface of nerve membrane.

Once local anesthetic agents gained access to receptors, permeability to sodium ions is reduced or eliminated; and nerve conduction is interrupted.

On the basis of the ability of local anesthetic agents to react with specific receptor sites in sodium channels; there are four sites in sodium channels; at which drugs can alter nerve conduction as shown in Table 6.1 (Covino and Vassallo, 1976).

Table 6.1: Classification of local anesthetic agents on the basis of site and mode of action

Class	*Definitions*	*Chemical substances*
A	Local anesthetic agents acting on receptor-sites on external surface of nerve membrane	Biotoxins (e.g. tetrodotoxin and saxitonin)
B	Local anesthetic agents acting on receptor-sites on internal surface of nerve membrane	Quarternary ammonium analogues of lidocaine and scorpion venom
C	Local anesthetic agents acting on receptor-independent mechanisms	Benzocaine
D	Local anesthetic agents acting by combination of receptor-dependent and receptor-independent mechanisms	Most clinically used local anesthetic agents, e.g. lidocaine, mepivacaine, and prilocaine

BIBLIOGRAPHY

1. Covino BG, and Vassallo HG. Local anesthetics: mechanisms of action and clinical use. New York 1976. Grune and Stratton.
2. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
3. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. New Delhi: CBS Publishers and Distributors 1990.
4. Roberts DH, Sowray JH. Local Analgesia in Dentistry. 3rd ed. Bristol: Wright 1987.

PROPERTIES OF AN IDEAL LOCAL ANESTHETIC AGENT

The properties that are desirable in a local anesthetic solution are as follows:

1. It should be non-irritating and produce no local reaction to the tissues to which it is applied.
2. It should not cause any permanent change in the nerve structure.
3. It should cause minimal systemic toxicity.
4. It must be effective when injected into the tissues and should have sufficient penetrating properties to be effective as a topical anesthetic, when applied topically to the mucous membrane.
5. It should have a short time of onset, if possible.
6. The duration of action must be long enough to allow completion of procedure.

Bennett (1974) has added some properties which are as follows:

1. It should have enough potency to give complete anesthesia without the use of harmful concentrated solutions.
2. It should be relatively free from producing allergic reactions.
3. It should be stable in solution and readily undergo biotransformation in the body.
4. It should either be sterile or be capable of being sterilized by heat without deterioration.

CLASSIFICATIONS

The local anesthetic agents can be classified in various ways.

On the Basis of Occurrence in Nature

1. Naturally occurring, e.g. cocaine
2. Synthetic compounds
   a. Nitrogenous compounds
      i. Derivatives of para-aminobenzoic acid (PABA)
         o Freely soluble, e.g. procaine
         o Poorly soluble, e.g. benzocaine

ii. Derivatives of acetanilide, e.g. lignocaine (lidocaine, xylocaine)
iii. Derivatives of quinolone, cinchocaine (nupercaine)
iv. Derivatives of acridine, e.g. bucricaine (centbucridine, centoblock)

b. Non-nitrogenous compounds, e.g. benzyl alcohol, and propanediol

3. Miscellaneous drugs with local anesthetic action, e.g. clove oil, phenol, chlorpromazine, certain antihistaminics such as diphenhydramine.

On the Basis of Chemical Structure

Esters

These can be further classified as:

i. Esters of benzoic acid, e.g. cocaine, benzocaine (ethylaminobenzoate), and butacaine.
ii. Esters of para-aminobenzoic acid, e.g. procaine, chloroprocaine, and propoxycaine.

Amides

For example, articaine, bupivacaine, lidocaine, mepivacaine, and prilocaine.

On the Basis of Duration of Action

a. *Short-acting:* Articaine, lidocaine, mepivacaine, prilocaine, etc.
b. *Long-acting:* Bupivacaine, etidocaine, bucricaine, etc.

PROPERTIES OF SPECIFIC LOCAL ANESTHETIC AGENTS

Common properties of local anesthetic agents are as follows:

1. All are synthetic compounds.
2. All the agents contain amino group.
3. All the agents form salts with strong acids. The synthetic compounds used as injectable local anesthetic agents are weakly basic in nature; and poorly soluble in water. They all form salts with strong acids. When combined with hydrochloric acid, form salts, which are soluble in water; and acid in reaction.
4. The alkali increases concentration of unionised free base. The unionised free base is soluble in lipids. The chemical characteristics are so balanced that they have both lipophilic and hydrophilic properties. If hydrophilic group predominates: The ability to diffuse into lipid-rich nerves is diminished. If lipophilic group predominates: It is of less clinical value as an injectable local anesthetic, since it is insoluble in water; and unable to diffuse through interstitial tissues.
5. The salts are acid in reaction and relatively stable.

6. The agents either get hydrolysed by plasma cholinesterase or undergo biotransformation in liver. The amides undergo biotransformation in liver. The ester type of local anesthetic agents undergo hydrolysis in plasma (plasma cholinesterase).
7. Their actions are reversible.
8. The agents are compatible with epinephrine or allied drugs.
9. The agents are compatible with metal salts of mercury, silver and so forth.
10. The agents affect nerve conduction in a similar manner.
11. The agents are capable of producing toxic systemic effects when a sufficiently high plasma concentration is reached.
12. The agents have little or no irritating effect on tissues in anesthetic concentrations.
13. Potency: Depends solely upon its chemical structure.
14. Duration of anesthesia: Depends upon: (i) molecular configuration, and (ii) presence of a vasoconstrictor.
15. Toxicity: The first symptom of toxicity of all synthetic local anesthetic agents, usually manifested by signs and symptoms of CNS stimulation. This may vary from mild restlessness to severe convulsions. The stimulating phase is usually followed by depression, which, if severe enough, may cause death.
16. All local anesthetic agents cause inhibition of contractility of myocardium.

Cocaine

It is an alkaloid obtained from the leaves of cocoa tree (erythroxylon cocoa). It is the methylbenzoyl ester of ecgonine which is chemically related to atropine. It is seldom used in dentistry, may be precluded in patients with severe renal dysfunction, or who are undergoing renal dialysis. It is almost completely excreted unaltered by the kidney.

Cocaine is a drug of dependence, and is a major drug of abuse. The concentration used to produce local anesthesia is poisonous to many structures like leukocytes and tissue cells.

Procaine (Novocaine)

It is a diethyl aminoethyl ester of para-aminobenzoic acid (PABA). It was first synthesized by Einhorn in 1905. It is non-irritant and as effective as cocaine as a local anesthetic agent. It is much less toxic and does not produce drug dependence. It is now seldom used in dentistry because of availability of more potent local anesthetic agents.

Procaine can reduce the effectiveness of sulfonamides; because excessive amounts of PABA, which is a metabolite of procaine, can reverse the action of sulfonamides. Excessive amounts of this entity reverses the inhibition and decreases effectiveness of the antibiotic. Procaine is compatible in solution with all vasoconstrictors.

Pharmacology

Procaine had been the standard of comparison for potency and toxicity with other local anesthetic agents for over 50 years. It has been assigned a potency and toxicity of 1.

The vasodilation caused is more profound than all other local anesthetic agents. The effect is very brief if used without a vasoconstrictor. A major nerve block with procaine may have a duration of 5 minutes; however, the duration is extended to 1 hour if a suitable vasoconstrictor is used.

Procaine is readily absorbed when injected into the tissues. The ability to diffuse through interstitial tissues is poor. Hence, a good injection technique; particularly an accurate placement of needle; is essential to produce profound anesthesia.

Hydrolysis

Procaine is hydrolysed to PABA and diethyl aminoethanol in the plasma. It is a reaction that is catalysed by an enzyme, plasma cholinesterase, present in plasma and liver.

Availability in Dentistry

Procaine is used alone in dentistry as 4% solution; and in combination as 2% solution. It is available as 4% solution. It is marketed as 2% solution in a combination of 0.4% propoxycaine and 1:30,000 levarterenol or 1:20,000 levonordefrin.

Onset and Duration of Action

The onset of action takes 3-5 minutes and the duration of pulpal anesthesia is 30 minutes.

Maximum Recommended Dose

It is 15-20 mg/kg body weight not to exceed 1000 mg.

Toxic Reactions

Central Nervous System (CNS)

It is capable of producing both stimulation and depression.

Cardiovascular System (CVS)

The effect on CV system depends largely on the amount of drug used.

a. *In small amounts:* Procaine has no effect on CV system except dilation of microcirculation in the area of injection.
b. *In high doses:* Procaine produces sudden and profound CV collapse with marked and generalized vasodilatation accompanied by severe bradycardia or asystole.

Respiratory System

i. Mild doses have minimal direct effects.
ii. Higher doses may cause relaxation of bronchioles.
iii. Large toxic doses may severely depress respiration as a result of depression of CN system. In most instances of toxic overdose, of a local anesthetic agent, respiratory arrest occurs before cardiac arrest.

TOPICAL ANESTHETIC AGENTS

(Figs 7.1 to 7.16)

Benzocaine (Ethyl p-aminobenzoate) (Hurricaine)

i. It is the most popular topical anesthetic agent; and is an ingredient in several medical preparations.
ii. It is an ester of aminobenzoic acid (ABA).
iii. It does not contain the basic nitrogen group, because of which it does not form soluble anesthetic salts. Hence, it is poorly soluble in water and not fit for injection purposes.
iv. It is an irritant to the tissues and can produce toxic symptoms if absorbed into cardiovascular system in sufficient quantities.

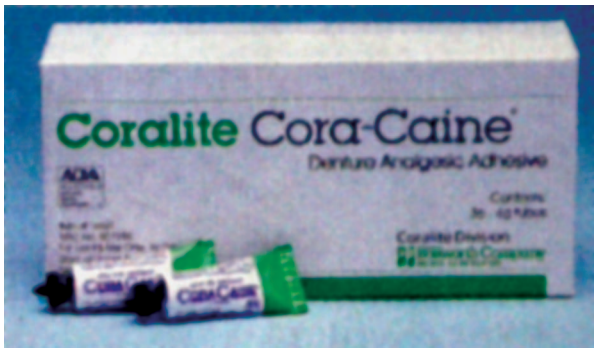


Fig. 7.1: Topical gels: Cora-caine
• For relief of pain and comfort from new or immediate dentures
• Ideal for including in new denture patient's initial take-home packet

Fig. 7.2: Topical gels: Gingicaine—20% Benzocaine
• Quick onset, • Eases patient discomfort and anxiety, • Different flavours

Fig. 7.3: Topical gels: Hurricaine—20% Benzocaine • Fast onset of 20 seconds

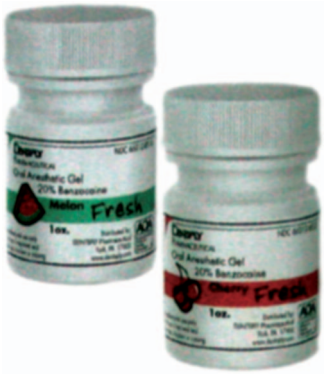


Fig. 7.4: Topical gels: Oral anesthetic gel—20% Benzocaine water soluble with polyethylene glycol base
• Short duration: 12-15 minutes, • Easy to use,
• Virtually no systemic absorption

Fig. 7.5: Topical gels: Handicaine Stix—20% Benzocaine
Each premeasured unit contains 0.40 g of 20% Benzocaine gel
• Ideal for use with a set-up tray

Fig. 7.6: Topical gels: Topical anesthetic—20% Benzocaine
- Fast-acting with no systemic absorption

Fig. 7.7: Topical gels: Lidocaine ointment 5%

Fig. 7.8: Topical gels: Lidocaine viscous 2%

Fig. 7.9: Topical spray: Cetacaine rapid onset within 30 seconds, lasts up to 30 minutes.
- The only treatment formula topical anesthetic with three active ingredients
- Controls pain, eases discomfort, and suppresses the gag reflex, thus providing patient cooperation
- J4 autoclavable cannula

Fig. 7.10: Topical spray: Hurricaine:
- 20% Benzocaine
- Suppresses gag reflex
- Fast onset of 20 seconds
- Spray kit contains: 2 oz spray can and 200 disposable extension tubes

Fig. 7.11: Topical spray: Pharmaethyl topical spray
- Pleasant taste
- Used in local applications in the mucous membrane or the oral cavity for:

i. Lancing of abscesses,
ii. Extraction of mobile deciduous and permanent teeth with gross gum recession and periodontal disease,
iii. Pulp vitality testing,
iv. Temporary relief of TM joint by external spray on skin surface around TM joint area, and
v. Suppression of gag reflex; facilitates deep scaling and curettage procedure

Fig.7.12: Topical spray: Topex metered spray
- Fast-acting with no systemic absorption
- Built-in safety feature delivers 50 mg / spray with no chance of overspray
- More than 1000 sprays per can
- Great for gaggers, deep scaling and X-rays
- Disposable tips included with each can

Fig. 7.13: Topical liquids: Caine tips
- Oral pain reliever
- 20% Benzocaine solution Ideal for:
  i. Dental procedures: Prior to local anesthetic injection
  ii. Periodontal curettage and deep scaling
  iii. Impression taking and radiography
  iv. Pain relief: Pain due to ulcers, wounds, and other minor mouth irritations

Fig. 7.14: Topical liquids: Cetacaine
- Unique for, fast-acting, long-lasting, triple-active ingredient treatment formula that can be applied directly to pain site
- Onset time of 30 seconds
- Effective for up to 30 minutes

Fig. 7.15: Topical liquids: Hurricaine: 20% Benzocaine
- Fast onset of 20 seconds
- For minor mouth irritations, stomatitis, and Canker sores,
- Deep scaling / periodontal treatment

Fig. 7.16: Topical patches: Dentipatch—Lidocaine transoral delivery system
- It is effective in reducing needle pain and pain associated with soft tissue procedures. Dentipatch is approximately 3 cm long x 1 cm wide and 2 cm thick and contains 46.1 mg of lidocaine. It is applied directly to the oral mucosa. Each unit consists of a broadhesive matrix acting as a reservoir to hold and deliver a steady, focussed release of lidocaine which provides a duration of anesthesia adequate to cover most dental procedures with a single application.

v. The low solubility in water, and slow absorption from the area of topical application prolongs the duration of anesthesia and reduces its toxicity.

Cinchocaine

It is a potent but a toxic local anesthetic agent. It is not indicated for use for infiltration or block anesthesia. It is used locally in the form of ointment.

Water-soluble Topical Anesthetic Agents

Lidocaine Hydrochloride

i. It is water-soluble.
ii. Availability: It is available in 2%, 5%, 10% and 15% concentration.
iii. Penetration: It has a better tissue penetration than lidocaine base because of its solubility in water.
iv. Lidocaine 2% is useful in patients who tend to gag during dental procedures.
v. Maximum recommended dose is 200 mg. When used with the help of cotton pliers or packs, the maximum recommended dose is 1-5 ml (40-200 mg).

INJECTABLE LOCAL ANESTHETIC AGENTS

Lignocaine (lidocaine, xylocaine, octocaine, dentocaine)

It is the most commonly used local anesthetic agent in dentistry.

Chemistry

i. It is an amide; and an acetanilide (xylidine) derivative.
ii. It was first synthesised by Nils Lofgren in 1943; and was marketed in 1948.
iii. It is the first non-ester type of local anesthetic agent to be used in dentistry.
iv. It is a white crystalline powder; with a melting point of 69°C; and used as hydrochloride salt.
v. The lidocaine base is slightly water-soluble; but hydrochloride salt is readily water soluble.
vi. It is stable, as it can be stored for a long time at room temperature.
vii. It withstands boiling and autoclaving.
viii. It is compatible with all types of vasoconstrictors.
ix. The pH of plain solution is 5 to 5.5; while that of the solution containing a vasoconstrictor is 3 to 3.5.

Pharmacology

Diffusion: It rapidly diffuses through interstitial tissues, into lipid rich nerve, giving a rapid onset of anesthesia.
Dissociation constant (pKa) = 7.85. It favours deprotonisation and produces more available unionised free base for action on the nerve membrane and for production of conduction block.

Biotransformation

Lidocaine undergoes biotransformation in liver.

a. *Metabolism:* It is metabolised in liver by microsomal fixed-function oxidases.
   Lidocaine and other amide-type local anesthetic agents are not affected by enzyme plasma cholinesterase. Hence, these are the agents of choice in patients with abnormal or insufficient amounts of this enzyme. Use of ester-type of local anesthetic agents should be avoided or greatly reduced in these patients. Since mechanism for their degradation is deficient, excessive amounts could accumulate in blood stream leading to toxic manifestations.
b. *Excretion:* Lidocaine and its breakdown products are excreted to some extent in urine, by kidney; < 10% unchanged, > 80% by various other mechanisms.

Potency: 2 times as potent as procaine. Today, it is taken as the standard for comparison of various other local anesthetic agents.

Toxicity: 2 times as toxic as procaine.

Action on blood vessels: The action on blood vessels is less than that of procaine but more than those of mepivacaine and prilocaine.

Time of onset of action: Rapid (2-3 minutes).

Duration of action: It depends upon:

i. Type of injection: Nerve block has longer duration than infiltration.
ii. Amount of vasoconstrictor used in the solution.

Effective dental concentration: 2%

Anesthetic half-life: 1.6 hours

Topical anesthetic action: It has topical anesthetic effect. It forms an excellent surface anesthetic. It is used in the following forms topically:

- 2% in the form of jelly
- 5% in the form of ointment
- 10% and 15% in the form of spray (aerosol).

Maximum Recommended Dose (MRD)

Lidocaine is the most commonly used local anesthetic agent, hence it is necessary to consider the instructions of the manufacturers, and the recommendations of American Dental Association are worth following.

Instructions of the Manufacturer

To understand the concept of Maximum Recommended Doses, we need to understand the concentration of local anesthetic agents. Consider the example of lignocaine which is the most commonly used local anesthetic agent. It is used in the concentration of 2%. It simply means 2 g of solute is contained in 100 ml of solution, which means 2000 mg of the solute are present in 100 ml of solution. Hence, it means 20 mg of the solute is contained in 1 ml of solution. In other words 1 ml of 2% lignocaine solution contains 20 mg of the local anesthetic agent (lignocaine).

The instructions for the local anesthetic agents with or without a vasoconstrictor, are as follows:

(a) Local anesthetic agents with a vasoconstrictor:

As per the manufacturer, the recommended dose of lidocaine with a vasoconstrictor, such as epinephrine, is 7.0 mg/kg BW, but not to exceed 500 mg.

A 2% lidocaine solution contains 2 g/100 ml or 2000 mg/100 ml or 20 mg/ml of local anesthetic solution. In India, a standard cartridge contains 2 ml of local anesthetic solution. Hence, the recommended dose (500 mg) of the agent will be contained in ($500 \times \frac{1}{20}$ =) 25 ml. or 12½ cartridges of the local anesthetic solution.

(b) Local anesthetic agents without a vasoconstrictor:

Similarly, as per the manufacturer, the recommended dose of lidocaine, without a vasoconstrictor is 4.4 mg/kg BW, but not to exceed 300 mg. A lesser dose is recommended because of faster absorption of the local anesthetic agent. This dose will be contained in 15 ml or 7½ cartridges of the local anesthetic solution.

Recommendations of the American Dental Association (ADA)

However, the dosage regimen as suggested by the Council on Dental Therapeutics of the American Dental Association and the USP Convention, the Maximum Recommended dose for lidocaine with or without a vasoconstrictor is 4.4 mg/kg body weight.

The Maximum Recommended Doses are mentioned here as shown in Table 7.1, for individuals with their body weights ranging from 10 to 70 kg.

Table 7.1: The maximum recommended doses for various body wieghts

Body weight (kg)	Dose (Body weight x 4.4) = mg	MRD (ml)	MRD (Cartridges) (approx)
10	44	2.2	1.1
20	88	4.4	2.2
30	132	6.6	3.3
40	176	8.8	4.4
50	220	11	5.5
60	264	13.2	6.6
70	300	15.4	7.7

The dental cartridges contain 2 ml of local anesthetic solution in India, 1.8 ml in USA and France, and 2.2 ml in UK and Australia.

Other local anesthetic agents used in dentistry are in various concentrations. The various concentrations and the contents of the solution are shown in Table 7.2.

Availability in Dentistry

i. Dental cartridges: 2% lidocaine with epinephrine 1:80, 000 in India.
ii. Vials:
   a. 2% lidocaine, without epinephrine (Lidocaine Plain)
   b. 2% lidocaine with epinephrine 1:80,000, 1:100,000, and 1:200,000.

Toxicity

The deleterious effects of toxic doses on various systems are as follows:

Central Nervous System

Toxic doses of local anesthetic agents first produce stimulation, followed by depression. The manifestations of stimulation vary from mild restlessness to severe convulsions. The phase lasts for a brief period. The depression is manifested as drowsiness to loss of consciousness.

Lidocaine administered intravenously is capable of producing a degree of analgesia and even general anesthesia.

Table 7.2: Various local anesthetic agents, along with their concentrations and contents

Percentage	Example	Amount of local anesthetic agent/ml	Amount of local anesthetic agent/cartridge
0.25%	Bupivacaine	2.5 mg/ml	5
0.5%	Bupivacaine	5 mg/ml	10
1%	Lignocaine	10 mg/ml	20
2%	Lignocaine	20 mg/ml	40
3%	Mepivacaine	30 mg/ml	60
4%	Prilocaine	40 mg/ml	80

Cardiovascular System

The effects on cardiovascular system vary with the dose used.

a. *Moderately large doses:* It produces overall inhibition on the contractility of heart muscle, in the form of:
   i. A decrease in the electrical excitability of myocardium.
   ii. A decrease in the force of contraction (negative inotropic effect).
   iii. A decrease in the rate of electrical impulse conduction (negative chronotropic effect).
b. *Large doses:* In large doses such as, 50-100 mg (1.5 mg/kg BW), it is given IV during GA and surgery to correct ventricular arrhythmias that occur during surgical procedures. It is one of the most popular anti-arrhythmic agents in medicine.

Vasculature

Vasodilatation is produced by direct relaxing effect on smooth muscle of vessel walls. In toxic doses, this action contributes to hypotension and cardiovascular collapse.

Respiratory System

a. In small doses it causes mild bronchodilatation.
b. In large doses it causes respiratory arrest (apnea) (the most common cause of death related to overdose of local anesthetic agent). In majority of cases, respiratory arrest precedes cardiac arrest. However, artificial ventilation and basic life support (BLS) measures will prevent serious sequelae.

Mepivacaine

It is used in the concentration of 3%. As per the manufacturer, MRD is 6.6 mg/kg BW not to exceed 400 mg for an adult patient. The MRD as per Malamed is 4.4 mg/kg BW not to exceed 300 mg, as shown in Table 7.3 (Tables 7.6 and 7.7).

Table 7.3: Mepivacaine HCl without a vasoconstrictor

Weight in kg	MRD 6.6 mg/kg		MRD 4.4 mg/kg	
	mg	ml	mg	ml
10	66	2.2	44	1.4
20	132	4.4	88	2.9
30	198	6.6	132	4.4
40	264	8.8	176	5.5
50	330	11.0	220	7.3
60	396	13.2	264	8.8
70	462	15.4	308	10.2

Bupivacaine

It is an amide, with a butyl group replacing ethyl group at the hydrophilic end. It is a long-acting local anesthetic agent (Tables 7.6 and 7.7).

A clinical comparative study was carried out by Chitre and Parkar (1981), whereby xylocaine (lignocaine) and marcaine (bupivacaine) were used in a group of patients who underwent minor oral surgical procedures. The parameters compared were rapidity of onset, depth and duration of anesthesia, and their side effects. The duration of anesthesia was definitely prolonged in marcaine group.

Maximum Recommended Dose

MRD is 1.3 mg/kg BW not to exceed 90 mg (Jakobs 2003). It is used in the concentrations of 0.25% and 0.5%, as shown in Table 7.4.

Prilocaine

It is used in the concentration of 4%. The MRD is 6.0 mg/kg BW to a maximum of 400 mg, as shown in Table 7.5 (Derrikson and Granberg, 1985). (Tables 7.6 and 7.7).

Table 7.4: Concentration = 0.5%

Body weight (kg)	*MR dose (Body weight × 4.4) = mg*	*MRD (ml)*
10	13	2.6
20	26	5.2
30	39	7.8
40	52	10.4
50	65	13.0
60	78	15.6
70	91	18.2

Table: 7.5. Concentration = 4%

Body weight (kg)	*MR dose (Body weight × 4.4) = mg*	*MRD (ml)*
10	60	1.5
20	120	3.0
30	180	4.5
40	240	6.0
50	300	7.5
60	78	9.0
70	91	10.2

Table 7.6: Showing the comparison of various other local anesthetic agents

Properties	*Mepivacaine*	*Bupivacaine*	*Prilocaine*
Trade names	Scandonest, Polocaine, Carbocaine	Marcaine, Sensorcaine	Citanest, Citanest forte
Chemical structure	Amide, with N-methyl group at hydrophilic end	Amide, with a butyl group replacing ethyl group at hydrophilic end	Amide, a derivative of toluidine
Onset	1½ - 2 min	6-10 min	2-4 min
Duration of action	2-3 hr	6-8 hr	2-3 hr
Potency	Similar to lignocaine	4 times as potent as lignocaine	Similar to lignocaine
Toxicity	Similar to lignocaine	Similar to lignocaine	Lesser than lignocaine
Metabolism	By hepatic fixed-function oxidases	By hepatic amidases	By hepatic amidases
Excretion	Kidney	Kidney	Kidney
pH			
i. Plain solution	4.5	4.5-6.0	4.5
ii. Solution with vasoconstrictor	3.0-3.5	3.0-4.5	3.0-4.0
Effective dental concentration	2% with vasoconstrictor and 3% without vasoconstrictor	0.25% and 0.5%	3% and 4%
Anesthetic half-life	1.9 hours	2.7 hours	1.6 hours
Maximum recommended dose	6.6 mg/kg BW, not to exceed 400 mg	1.3 mg/kg BW, not to exceed 90 mg	6 mg/kg BW; not to exceed 400 mg
Contraindications			i. Patients with congenital or idiopathic methemoglobulinemia ii. Patients taking acetaminophen or phenacetin
Others uses		i. Spinal anesthesia, ii. Epidural anesthesia	

SELECTION OF LOCAL ANESTHETIC AGENTS

There are several anesthetic agents available for use in dentistry. Each agent has got its own indications. The agents are selected on the basis of the needs of a particular patient. The following factors are to be considered for the selection of a particular agent (Tables 7.6 and 7.7).

Table 7.7: Showing comparative anesthetic properties of local anesthetic agents

Local anesthetic agents (with vasoconstrictor)	Onset of anesthesia		Duration of anesthesia	
	Regional	Infiltration	Regional	infiltration
Lignocaine	5 min	3 min	3 hr	2 hr
Prilocaine	3 min	4 min	3 hr	2 hr
Bupivacaine	5 min	2-5 min	5-6 hr	5 hr
Procaine	7 min	4 min	2.5 hr	2 hr

1. Duration of action
2. Need for control of postoperative pain
3. Physical and mental status of the patient
4. Concomitant medications.

Duration of Action

It is an important aspect to be considered. An approximate duration for completing of the surgical procedure for which anesthesia is required, should be taken into account.

The working time should be adequate. On the basis of duration of action, various local anesthetic agents may be arbitrarily grouped as follows:

1. *Ultra-short acting agents:* Where the duration of action is less than 30 minutes:
   i. Procaine without a vasoconstrictor
   ii. 2-chloroprocaine (1.2% or 3%) without a vasoconstrictor
   iii. 2% lidocaine without a vasoconstrictor
   iv. 4% prilocaine without a vasoconstrictor for infiltration.
2. *Short-acting agents:* Where the duration of action is 45 to 75 minutes.
   i. 2% lidocaine with 1:100 000 epinephrine
   ii. 2% mepivacaine with 1:200 000 levonordefrine
   iii. 4% prilocaine when used for nerve block
   iv. 2% procaine, 0.4% propoxycaine with a vasoconstrictor.
3. *Medium-acting agents:* Where the duration of action is 90 to 150 minutes:
   i. 4% prilocaine with 1:200,000 epinephrine
   ii. 2% lidocaine and 2% mepivacaine with a vasoconstrictor (pulpal anesthesia).
4. Long-acting agents: Where the duration of action is 180 minutes or longer.
   i. 0.5% bupivacaine with 1:200,000 epinephrine
   ii. 0.5% or 1.5% etidocaine with 1:2,00,000 epinephrine.

Need for Control of Postoperative Pain

Most of the oral surgical procedures result in varying amount of post-operative pain. The local anesthetic agent serves as an additional medication that sometimes eliminates the need for postoperative analgesics. The local anesthetic agents that have shown to produce longer duration of analgesia even after the other sensations have returned are bupivacaine and etidocaine.

Physical, Medical and Mental Status of the Patient

Any co-existing medical conditions, such as hypertension or diabetes mellitus should be considered. A patient with a history of allergy to a specific local anesthetic agent, or of any of the other components used along with the local anesthetic agents should be considered. Patients with a history of malignant hyperpyrexia, the use of amide derivatives is contraindicated.

The mental status of the patients has to be evaluated. Small children and mentally challenged adults are sometimes fascinated with the numbness and the tingling sensation produced by the local anesthetic agents. These patients land up into traumatising their lip, tongue or cheeks intentionally or inadvertently. Hence for these reasons, the use of long acting local anesthetic agents is contraindicated in such patients.

Concomitant Medications

The use of vasoconstrictors is contraindicated in patients who are taking monoamino-oxidase (MAO) inhibitors, tricyclic (TCA) antidepressants, etc.

NEWER LOCAL ANESTHETIC AGENTS

There are four new local anesthetic agents which are now available in the market with improved efficacy and safety. (1) Articaine, (2) Etidocaine, (3) Ropivacaine, and (4) Centbucridine.

Etidocaine (Duranest)

It is classified as an amide. It was prepared by Takman in 1971 and was approved by US FDA in 1976. It is considered as a long-acting local anesthetic agent as bupivacaine.

Chemical Structure

It is a chemical analogue of lidocaine and mepivacaine. The chemical structure is identical to lidocaine except for a propyl (3 carbon) substitution and ethyl (2 carbon) addition.

Effective Dental Concentration: 1.5%

Onset and Duration of Action

Etidocaine is chemically related to lidocaine, however, its clinical indications are identical to those of bupivacaine. Etidocaine has slightly rapid onset of action of about 3 minutes, whereas bupivacane has an onset of 6-10 minutes (Dunsky and Moore, 1984, and Giovannitti and Bennett, 1983).

The depth of mandibular anesthesia provided by etidocaine with epinephrine appears to be equivalent to conventional agents. While the depth of anesthesia following maxillary infiltrations may be somewhat less (Moore and Dunsky, 1983).

In another study the duration and depth were variable following infiltration anesthesia, whereas the duration of pulpal anesthesia following a nerve block was considerably longer, ranging from 90 to 180 minutes (Moore, 1990). Another study, showed that etidocaine provides similar duration of soft tissue anesthesia following inferior alveolar nerve block (2-3 times that of lidocaine).

Indications

1. Both etidocaine and bupivacaine provide adequate surgical anesthesia, hence they are also useful for management of postoperative pain.
2. Also used in the management of chronic pain either as symptomatic, diagnostic, or definitive therapy.

Centbucridine (Bucricaine, Centoblock)

Chemical Structure

It is an acridine derivative, and also a quinolone derivative.

Potency

It is used in the concentration of 0.5%. Its potency is 5-8 times that of lidocaine; with an equally rapid onset of action, and equivalent duration of action (Gupta et al 1982, and Vacharajani et al, 1983).

Advantages

1. It has a longer duration of action than lignocaine.
2. It has some amount of inherent vasopressor activity. It therefore, does not require addition of a vasopressor for infiltration anesthesia.
3. Its CNS and CVS toxicity is less than that of lignocaine.
4. It can be used in patients with a history of allergy to lignocaine, because of its different chemical structure.

Availability

It is available as a solution as 5 mg/ml in the form of 30 ml vials.

Adverse Effects

It does not affect the CNS or CVS adversely, unless when administered in very large doses (Gupta et al, 1982).

Indications/Uses

(1) Subarachnoid and extra-dural anesthesia (Suri et al, 1982), (2) IV regional anesthesia (Suri et al, 1983), (3) Intraocular surgery (Beri et al, 1997), and (4) Dental extraction (Vacharajani et al, 1983).

Ropivacaine (Naropin)

Approved by FDA.

Chemical Structure

Structurally, it is similar to mepivacaine and bupivacaine (Sisk, 1992). Like etidocaine and bupivacaine, ropivacaine is considered to be a long-acting local anesthetic agent, belonging to amide group.

Indications

Currently indicated for (i) epidural obstetric anesthesia, (ii) surgical anesthesia, and (iii) management of post-surgical pain (Montvale, 1998). Its use is particularly advisable in patients in whom the use of vasoconstrictors is contraindicated or should be kept to a minimum.

Advantages

The reported advantages are:

i. Vasoconstrictive effect at small dosages; and
ii. A significantly smaller depression of cardiac conductivity than bupivacaine.
iii. Less affinity for cardiac Na-channels than bupivacaine.

Duration of Action

The duration of action is similar to bupivacaine and etidocaine. Ernberg and Kopp (2002) performed a study with ropivacaine in 30 subjects. The concentration of ropivacaine used was 0.75%. The pulpal anesthesia achieved following inferior alveolar nerve block within 10 minutes which lasted for 2-6 hours; while mandibular soft tissue anesthesia lasted for 5-9 hours.

Adverse Drug Reactions

The elimination half-life of ropivacaine is 25.9 minutes, which is considerably shorter than that of other amides (Arthur and Covino, 1988).

Ropivacaine has a greater margin of safety between convulsive and lethal doses than doses of bupivacaine and also a lower dysrhythmogenic potential than bupivacaine (Arthur et al, 1998). Ropivacaine has demonstrated decreased cardiotoxicity relative to bupivacaine, but its clinical duration of action is approximately 20% shorter (Brown et al, 1990 and Moller and Covino, 1992).

Availability

Currently available in ampoules and vials containing 2, 5, 7.5, and 10 mg/ml (0.2 to 1%) solutions.

Articaine (Ultracaine and Septocaine)

It was prepared by H Rusching et al in 1969. In USA, it is used in the formulation of 4% articaine HCl with 1:100 000 epinephrine bitartarate in 1.7 ml glass cartridges. In Canada, it is marketed under the brand names of Ultracaine and Septanest; and in USA as Septocaine. US FDA has approved its use in USA, in the year, 2000.

Chemical Structure

It is classified as an amide. It is the only amide type of local anesthetic agent containing a thiophene (sulphur-containing) ring. In addition it also contains an ester group.

Biotransformation

The biotransformation occurs in two ways:

i. In the plasma (hydrolysis) by plasma cholinesterase, and
ii. In the liver by hepatic microsomal enzymes.

Once it is absorbed from the injection site into the systemic circulation, it is rapidly inactivated by hydrolysis of the ester side chain to articainic acid, and therefore, has an extremely short plasma half-life (27 minutes)(Oertel et al, 1997).

Onset and Duration of Action

The time of onset, duration of action, and depth of anesthesia is similar to 2% lidocaine with 1:100,000 epinephrine (Malamed et al, 2000).

A 4% articaine solution with epinephrine is reported to have an onset of 1.5-3.0 minutes for maxillary infiltrations, and slightly longer for inferior

alveolar nerve blocks. While the duration of soft tissue anesthesia ranges from 2-3 hours for maxillary infiltration anesthesia and 3-4 hours for mandibular block anesthesia.

Effective Dental Concentration: 4% with 1:100 000 or 1:200 000 Epinephrine.

Indications

It is indicated in cases of extended minor oral surgical procedures, or for long appointments for cosmetic dentistry, full mouth restoration, full mouth periodontal surgery, or multiple implant placements.

Adverse Effects

Articaine does not have a greater allergenecity than other available local anesthetic agents, probably because the ester metabolite is not the allergen PABA. Reports of toxic reactions following the use of articaine for dental anesthesia are extremely rare.

The rapid inactivation of articaine by plasma esterases may explain the apparent lack of overdose reactions reported following its administration, even though, it is marketed as a 4% solution. Articaine and prilocaine have been associated with a slightly higher incidence of mandibular and lingual paresthesia (Haas and Lennon, 1995).

BIBLIOGRAPHY

1. Arthur GR and Covino BG. Whats new in local anesthetics? Anesthesiol Clin North Am 1988;6:357-70.
2. Arthur GR, Feldman HS, and Covino BG. Comparative pharmacokinetics of bupivacaine and ropivacaine, a new amide local anesthetic. Anesth Analog 1988;67:1053-58.
3. Beri S, Biswas NR, Shende D, et al. Injectable centbucridine and lidocaine hydrochloride for intraocular surgery, Ophthalmol Surg Lasers 1997;28: 12:1027-29.
4. Brown DL, Carpenter RL, and Thompson GE. Comparison of 0.5% ropivacaine and 0.5% bupivacaine for epidural anesthesia in patients undergoing lower-extremity surgery. Anesthesiology 1990;72:633-36.
5. Chitre AP, and Parkar MI. A clinical comparative study of anesthetic effects of marcaine and xylocaine in minor oral surgery. Dent Dialog 1 and 2, 1981;11-15.
6. Council on Dental therapeutics of the American Dental Association: Accepted Dental Therapeutics, 40th ed. Chicago: American Dental Association 1984.
7. Derrikson E, Granberg, PO. Studies on the renal excretion of Citanest and Xylocaine. Acta Anesth Scand Suppl 1985;16;79-85.
8. Donaldson D, James-Perdok L, et al. A comparison of Ultracaine DS (Articaine HCl) and Citanest forte (Prilocaine HCl) in maxillary infiltration and mandibular nerve block. J Can Dent Assoc 1987;53(1)38-42.

9. Dunsky JL and Moore PA. Long-acting local anesthetics: A comparison of bupivacaine and etidocaine in endodontics. J Endodont 1984;10:457-60.
10. Ernberg M, and Kopp S. Ropivacaine for dental anesthesia: A dose finding study. J Oral Maxillofac Surg 60:9:9004-1010, discussion 2002;1010-11.
11. Giovannitti, JA and Bennett CR. The effectiveness of 1.5% etidocaine HCl with epinephrine 1:200 000 and 2% lidocaine HCl with epinephrine 1:100 000 in oral surgery: A clinical comparison. JADA 1983;107:616-18.
12. Gupta PP, Tangri AN, Saxena RC, and Dhawan BN. Clinical pharmacology studies on 4-N-butylamino-1,2,3,4,-tetrahydroacrydine hydrochloride (Centbucridine): A new local anaesthetic agent, Indian J Exp Biol 1982;20:344-46.
13. Haas DA, Lennon D. A 21 year retrospective study of reports of paresthesia following local anesthetic administration. J Can Dent Assoc. 1995;61(4)319-30.
14. Hawkins JM and Moore PA. Local Anesthesia: Advances in agents and techniques. Dent Clin N Am 2002;46:719-32.
15. Hersh EV. Local Anesthetics in Oral and Maxillofacial Surgery. In Oral and Maxillofacial Surgery by Fonseca RJ. Vol I: Anesthesia / Dentoalveolar Surgery /Office Management. 58-78 Saunders 2000.
16. Howe GL. Minor Oral Surgery. 3rd ed. Mumbai: Butterworth-Heinemann Ltd. Varghese Publishing House, Reprint 1985.
17. Jakobs W: Actual aspect of dental anesthesia in Germany, presented at the 10th International Dental Congress on Modern Pain Control, IFDAS, Edinburgh, Scotland, UK, June 2003.
18. Kodak Dental Products: Prescribing information, Rochester, NY, Eastman Kodak Company Dental Products, 1993.
19. Laskin DM. Oral and Maxillofacial Surgery Vol I and II CV Mosby Co., India: AITBS Publishers 1992.
20. Malamed SF, Gagnon S, Leblanc D. Efficacy of Articaine: A new Amide Local Anesthetic. JADA 2000;31(5)635-42.
21. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
22. Malamed SF. Handbook of Local Anesthesia. 5th ed. Mosby 2004.
23. Marcaine Hydrochloride: Drug prescribing information, NY, Winthrop Pharmaceuticals 1990.
24. Moller RA, and Covino BG. Effects of progesterone on the cardiac electrophysiologic alterations produced by ropivacaine and bupivacaine. Anesthesiology 1992;77:735-41.
25. Montvale, N J. Naropin (Ropivacaine HCl injection). In Physicians' Desk Reference. 52nd ed. 1998;561-66. Medical Economics Company.
26. Moore PA, and Dunsky JL. Bupivacaine anesthesia: A clinical trial for endodontic therapy. Oral Surg 1983;55:176-9.
27. Moore PA. Long-acting local anesthetics: A review of clinical efficacy in dentistry, Compendium 1990;11:12, 24-26, 28-30.
28. Oertel R, Rahn R, and Kirch W. Clinical Pharmacokinetics of Articaine, Clin Pharmacokinet 1997;33(6)41725.
29. Peterson LJ, Ellis E III, Hupp JR, Tucker MR. Contemporary Oral and Maxillofacial Surgery. 2nd ed. Harcourt Brace and C. Asia. Pvt. Ltd. 1997.
30. Reizs, Haggmark S, Johansson G, et al. Cardiotoxicity of ropivacaine: A new amide local anesthetic, Acta Anesthesiol Scand 1989;33:93-98.

31. Richard Bennett C. Monheim's Local Anaesthesia and Pain Control in Dental Practice. 7th ed. Delhi: CBS Publishers and Distributors 1990.
32. Robin Woods. A Guide to the Use of Drugs in Dentistry, 12th ed. Sydney: Australian Dental Association Inc. 1996.
33. Satoskar RS, Bhandarkar SD, Ainapure SS. Satoskar, Kale, and Bhandarkar's Pharmacology and Pharmacotherapeutics. Revised 16th ed. Mumbai: Popular Prakashan 1999.
34. Sisk AL. Long-acting local anesthetics in dentistry. Anesth Prog 1992;39:53-60.
35. Suri YV, Patnaik GK, Nayak BC, et al. Evaluation of centbucridine for Intravenous regional Anaesthesia, Ind J Med Res 1983;77:722-27.
36. Suri YV, Singhal AP, Phadke VK, et al. Double blind study on centbucridine for subarachnoid and extradural anaesthesia, Ind J Med Res 1982;76:875-81.
37. Suri YV, Singhal AP, Phadke VK, et al. Double blind study on centbucridine for subarachnoid and extradural anaesthesia, Ind J Med Res 1982;76:875-81.
38. Suri YV, Patnaik GK, Nayak BC, et al. Evaluation of centbucridine for Intravenous regional Anaesthesia, Ind J Med Res 1983;77:722-27.
39. Vacharajani, GN, Parikh N, Paul T, and Satoskar RS. A comparative study of Centbucridine and lidocaine in dental extraction, Int J Clin Pharmacol Res 1983;3:251-55.

Vasoconstrictors form an integral and important component of most of the local anesthetic agents used in dentistry.

DEFINITION

Vasoconstrictors are the chemical agents or adjuncts added to local anesthetic solutions (a) to oppose vasodilatation caused by these agents and (b) to achieve hemostasis.

ACTIONS

The addition of a vasoconstrictor to a local anesthetic agent causes constriction of blood vessels and thereby controls tissue perfusion. The net effects caused by addition of vasoconstrictors to local anesthetic agents are:

1. It decreases the blood flow to the site of injection, because of vasoconstriction.
2. It decreases the rate of absorption of local anesthetic agent into cardiovascular system.
3. It lowers the plasma level of local anesthetic agent (Cannall et al, 1975) and Wildsmith et al, 1977), thereby, decreasing the risk of systemic toxicity of local anesthetic agent.
4. Higher volumes of local anesthetic agent remain in and around the nerve for longer periods; thereby increasing the duration of action of most local anesthetic agents (Brown, 1968).
5. It decreases bleeding at the site of injection because of decreased perfusion. This is useful when increased bleeding is expected during a surgical procedure (Carpenter et al, 1989; and Myers and Heckman, 1989).

CLASSIFICATIONS

These drugs, the sympathomimetic amines can be classified, on the basis of mode of action, into the following three categories. All vasoconstrictors used in conjunction with local anesthetic agents are direct acting agents.

1. *Direct acting drugs:* These drugs stimulate or exert their action directly on the adrenergic receptors. For example, epinephrine, norepinephrine, dopamine, levonordefrine and isoproterenol, etc.
2. *Indirect acting drugs:* These drugs act by releasing norepinephrine from the adrenergic nerve terminals (from their intraneuronal storage sites), e.g. tyramine, amphetamine, methamphetamine, hydroxyamphetamine.
3. *Mixed acting drugs:* These drugs have both direct and indirect actions. For example, metaraminol and ephedrine.

ADRENERGIC RECEPTORS

The concept of adrenergic receptors, was proposed by Ahlquist in 1948 and is well accepted today (Ahlquist, 1948).

Types

Ahlquist found 2 types of adrenergic receptors and termed as: (1) receptors, and (2) receptors, based on their action on smooth muscles.

α Receptors

Activation of receptors results in vasoconstriction.

β Receptors

β receptors are subcategorised further into β1, β2 and β3 receptors.

1. β1 receptors are found in the heart and small intestine; and are responsible for: (i) cardiac stimulation (increase in the HR and increase in the strength of contraction) and (ii) renin release.
2. β2 receptors are found in bronchi, vascular beds, and uterus; and are responsible for:
    i. Relaxation of bronchial muscles resulting in bronchodilatation
    ii. Relaxation of muscles in the walls of blood vessels, resulting in vasodilatation, and
    iii. Relaxation of uterus (Lands et al, 1967).
3. β3 receptors are found in brown and white adipose tissue; and are responsible for lipolysis.

DILUTION OF VASOCONSTRICTORS

The dilution of vasoconstrictors is commonly referred to as a ratio (e.g. 1 to 1000; and is written as 1:1000). The maximum doses of vasoconstrictors are presented as mg, and hence the following interpretations will help in conversion (Table 8.1).

- 1:1000 means 1 g or 1000 mg of solute (drug) contained in 1000 ml of solution.

- Therefore, a 1:1000 dilution contains 1000 mg in 1000 ml or 1.0 mg/ml of solution.

Vasoconstrictors used in local anesthetic solutions in dentistry are more diluted than 1:1000. Hence, a solution of a dilution 1:10,000 will contain 0.1 mg/ml of solution and 1:100,000 dilution will contain 0.01 mg/ml of solution.

Table 8.1: It shows the dilution and the amount of vasoconstrictor present

Dilution	*mg/ml*
1:1000	1.0
1:10 000	0.1
1:50 000	0.02
1:80 000	0.0125
1:100 000	0.01
1:200 000	0.005

PHARMACOLOGY OF SPECIFIC AGENTS

Epinephrine remains the most commonly used and the most effective vasoconstrictor used in medicine and dentistry. The other vasoconstrictors used are norepinephrine, phenylephrine, levonordefrine, and octapressin.

As a hydrochloride salt, all vasoconstrictors are stable. Vasoconstrictors are relatively unstable in solutions, and to ensure a reasonable shelf-life, it is necessary to include a preservative in local anesthetic solutions.

All vasoconstrictors used in dentistry can be prepared synthetically, however, epinephrine and norepinephrine are naturally occurring agents in human body.

Biosynthesis of epinephrine and norepinephrine occurs in chromaffin cells of adrenal medulla and the sympathetic postganglionic nerve fibers. Adrenal medulla contains about 2-4 mg of epinephrine and norepinephrine per gram of tissue. Out of this total amount (2-4 mg) only about 20-30% is norepinephrine. In other extramedullary catecholamine-secreting tissues, norepinephrine predominates.

EPINEPHRINE

Epinephrine remains the most effective and the most commonly used vasoconstrictor in medicine and dentistry.

Proprietary Name

Adrenaline.

Chemical Properties

i. In the form of an acid (hydrochloride) salt, vasoconstrictors are stable, and highly soluble in water.
ii. Slightly acidic solutions are relatively stable if they are protected from air.
iii. Deterioration through oxidation: It is hastened by the presence of metal ions.
Vasoconstrictors are relatively unstable in solutions, and to ensure a reasonable shelf-life, it is necessary to include a preservative or antioxidant in local anesthetic solutions. The most commonly used preservative added to dental cartridges containing a vasoconstrictor is sodium bisulfite. It competes with vasoconstrictor drug for available O_2 in the cartridge. In the presence of O_2, sodium bisulfite, which is more active, gets readily oxidised to sodium bisulfate, utilizing the available O_2, thus prevents the oxidation or deterioration of sympathomimetic amines such as the vasoconstrictors.
iv. The shelf-life of local anesthetic solution containing a vasoconstrictor is slightly shorter than that of a solution without a vasoconstrictor. Thus, the vasoconstrictors remain relatively unaffected.

Source: It is secreted primarily by adrenal medulla. It is available as a synthetic and is also obtained from the adrenal medulla of animals.
Mode of action: It acts directly on both α and β adrenergic receptors; β effects predominate.

It must be borne in mind that the endogenous release of adrenaline, caused by the stress of going to a Dental Surgeon, sight of dental instruments, and the apprehension of pain caused by the needle-sticks produces effects similar to those produced by excessive exogenous administration of adrenaline.

Maximum Recommended Doses (MRD) of Epinephrine

This drug is potent; and can produce undesirable results (a) if used in large volumes; or (b) if inadvertently injected intravascularly. Hence, these drugs should be used with due consideration to their benefits and risks.

It is recommended that the least concentrated solution that produces effective pain control should be used. Lidocaine is available with three dilutions of epinephrine (1:80,000, 1:100,000 and 1:200,000), in India and many other countries. In USA and Canada, it is available in dilutions of 1:50,000 and 1:100,000. The duration of effective pulpal and soft tissue anesthesia is equivalent with all these forms.

The New York Heart Association, in 1954, recommended that the maximum dose of epinephrine should be limited to 0.2 mg per appointment. The American Heart Association has recommended restriction of

epinephrine in local anesthetics when administered to patients with ischemic heart diseases.

The maximum doses as recommended by New York Heart Association, and as suggested by Bennette (1983) and Malamed (1997) are as follows:

A. Normal healthy adult patients (0.2 mg per appointment)

10 ml of a 1: 50,000 dilution (5 cartridges)
16 ml of a 1: 80,000 dilution (8 cartridges)
20 ml of a 1: 100,000 dilution (10 cartridges)
40 ml of a 1: 200,000 dilution (20 cartridges)

B. Patients with clinically significant cardiovascular disease (0.04 mg per appointment) (approximately 1/5th of the dose for normal patients)

2 ml of a 1: 50,000 dilution (1 cartridge)
3.2 ml of a 1: 80,000 dilution (1.6 cartridges)
4 ml of a 1: 100,000 dilution (2 cartridges)
8 ml of a 1: 200,000 dilution (4 cartridges)

In India, the cartridges available contain 2 ml of local anaesthetic solution. While in USA and France, the cartridges contain 1.8 ml of the solution and in UK and Australia, the cartridges contain 2.2 ml of the solution.

The most commonly used vasoconstrictor is epinephrine (adrenaline). The Maximum Recommended Doses (MRD) of epinephrine will be considered in two categories, which are as follows:

Normal Healthy Adult Individuals

The Maximum Recommended Doses (MRD) is 0.2 mg/appointment, as per the guidelines of American Heart Association and American Dental Association. The vasoconstrictors used are in a very small amount. This is achieved by dilution of the vasoconstrictors. The dilutions and the amount contained in that dilution is considered here.

A solution of 1:1000 dilution contains 1 g in 1000 ml, or 1000 mg in 1000 ml of the solution. Hence, a dilution of 1:1000 contains 1 mg/ml of the vasoconstrictor. Similarly, a solution of 1:80 000 dilution contains 1 g in 80 000 ml, or 1000 mg in 80 000 ml of the solution. Hence, a dilution of 1:80 000 contains 0.0125 mg/ml of the vasoconstrictor.

Similarly, a solution of 1:100 000 dilution contains 1 g in 100 000 ml, or 1000 mg in 100 000 ml of the solution. Hence, a dilution of 1:100 000 contains 0.01 mg/ml of the vasoconstrictor. Similarly, a solution of 1:200 000 dilution contains 1 g in 200 000 ml, or 1000 mg in 200 000 ml of the solution. Hence, a dilution of 1:200 000 contains 0.005 mg/ml of the vasoconstrictor.

The dilution and the corresponding amount of vasoconstrictor content are mentioned in Table 8.1.

Medically Compromised Individuals

The Maximum Recommended Doses (MRD) is 0.04 mg/appointment, (1/5th of the dose for normal healthy adult individuals, as per the guidelines of American Heart Association and American Dental Association).

For Hemostasis

Epinephrine is used in local anesthetic solution to minimise bleeding by depositing at the site of surgery via infiltration anesthesia during various surgical procedures. A local anesthetic solution containing 1:50,000 dilution of epinephrine is more effective in minimising bleeding than more dilute solutions such as 1:100,000 or 1:200,000 (Buckley, et al 1984).

Local anesthetic solution containing 1:50,000 epinephrine, when used in small volumes which are required for hemostasis do not increase patient's risk, the use of 1:100,000 dilution of epinephrine should always be considered, especially, in patients with history of sensitivity to catecholamines. This also holds true for patients with clinically significant cardiovascular disease and also elderly.

Systemic Actions

Myocardium

It stimulates β1 receptors of myocardium. The resultant events are as follows:

i. A positive inotropic effect (increases force of contraction), and
ii. A positive chronotropic effect (increases rate of contraction).

The resultant effects are that the cardiac output and heart rate are increased.

Pacemaker Cells

It stimulates β1 receptors and increases the irritability of pacemaker cells, leading to a greater incidence of dysrhythmias.

Coronary Arteries

It produces dilatation of coronary arteries, resulting in an increased coronary artery blood flow.

Blood Pressure

a. Systolic BP is increased, and
b. Diastolic BP:
   i. It is decreased in small doses; because of greater sensitivity to epinephrine of β2 receptors than α receptors present in blood vessels.
   ii. It is increased in larger doses; because of constriction of blood vessels produced by stimulation of receptors.

Cardiovascular System

The overall actions of epinephrine on the heart and cardiovascular system are the result of direct stimulation. If the stimulation of the heart persists for a long time, there will subsequently, be overall decrease in cardiac efficiency. These actions are as follows:

i. Increased stroke volume
ii. Increased heart rate
iii. Increased cardiac output
iv. Increased strength of contraction
v. Increased systolic and diastolic pressures
vi. Increased myocardial oxygen consumption.

Blood Vessels

The primary action of epinephrine is on microcirculation (small arterioles and precapillary sphincters). The blood vessels which supply skin, mucous membranes and kidneys contain primarily α receptors. Epinephrine produces constriction of these blood vessels. The blood vessels supplying skeletal muscles contain both α and β2 receptors, with β2 receptors predominating. Epinephrine in small doses produces dilatation of these vessels as a result of β2 effect. β2 receptors are more sensitive to epinephrine than α receptors. Large doses of epinephrine produce vasoconstriction because of stimulation of α receptors.

Rebound Phenomenon

Epinephrine is frequently used clinically as a vasoconstrictor for achieving hemostasis during surgical procedures. The injection of epinephrine directly into the tissues at surgical sites leads to high tissue concentration. It's α effect predominates. The stimulation of α receptor leads to vasoconstriction resulting in hemostasis.

However, subsequently, the biotransformation, reuptake and redistribution ultimately results in low tissue levels of epinephrine, when α receptors are stimulated, and β2 action predominates. The action on blood vessels changes; resulting in vasodilatation. This rebound activity is manifested as some bleeding at the end of, or a few hours after the surgical procedure.

Respiratory System

It has a α2 effect on the smooth muscle of the bronchioles, and hence is a potent dilator. It is a drug of choice in cases of bronchospasm as in bronchial asthma.

Central Nervous System

In therapeutic doses, epinephrine does not stimulate central nervous system. It stimulates central nervous system only if excessive doses are administered.

Metabolism

The actions of epinephrine are terminated by one of the following methods:

i. Primarily, by its reuptake by adrenergic nerves.
ii. Inactivated by the enzymes catechol-O-aminotransferase (COMT), and monoamine oxidase (MAO), both of which are present in liver.
iii. Very small amounts are excreted unchanged in urine.

Overdose

In small amounts commonly used in dentistry, only the arterioles in the immediate area of injection are affected by vasoconstrictors.

The factors that may produce toxic manifestations are: (i) large volumes, (ii) high concentration of vasoconstrictors; and (iii) inadvertent vascular injection of comparatively small amounts of vasoconstrictors. The manifestations can be in the form of tachycardia, hypertension, palpitation, headache, tremors, pallor, and in rare cases, ventricular fibrillation.

These side effects result from chemical interaction between catecholamine or vasoconstrictor molecules and the physiological receptor-site.

The systemic effects of vasoconstrictors usually last as long as the blood levels of the drug remain elevated.

a. *Small doses:* The initial clinical features of epinephrine overdose are related to stimulation of CN system, which include: Fear and anxiety, tension, restlessness, headache, tremor, weakness, dizziness, pallor, respiratory difficulty, and palpitation. This is known as "epinephrine reaction" (de Jong, 1994).
b. *Large doses:* The manifestations include: Cardiac dysrhythmias, dramatic increase in both systolic and diastolic BP, which may lead to cerebral hemorrhage, ventricular fibrillation is possible but rare, anginal episodes may be seen in patients with coronary insufficiency.

Clinical Applications in Dentistry

It is used along with a local anesthetic agent, as a vasoconstrictor:

(i) for achieving hemostasis, (ii) to decrease the absorption of local anesthetic agent into cardiovascular system, and (iii) to prolong duration of action.

Availability in Dentistry

In India, epinephrine is available in the following dilutions: 1:80,000, 1:100,000 and 1:200,000 for use in dentistry.

Norepinephrine (Nor-adrenaline)

It is not commonly used in dentistry. Refer to Table 8.2 for more details.

Felypressin

It is available as a vasoconstrictor, in combination with prilocaine, in dental local anesthetic cartridges, in many countries (Citanest forte).
Proprietory name: Octapressin
Chemical structure: It is a non-sympathomimetic amine; and a synthetic analogue of vasopressin (antidiuretic hormone).
Mode of action: It acts by directly stimulating vascular smooth muscle. It has little direct effect on the heart or on adrenergic nerve transmission. Its actions are more pronounced on venous rather than on arteriolar microcirculation (Altura, 1965).

Systemic actions:

i. *Myocardium:* No direct effects.
ii. *Coronary arteries:* Doses greater than therapeutic doses, may impair blood flow through coronary arteries.
iii. *Vasculature:* Doses greater than therapeutic doses, cause constriction of cutaneous blood vessels producing facial pallor.
iv. *Uterus:* It has both antidiuretic and oxytocic actions; hence the latter action contraindicates its use in pregnant patients.
v. *Maximum doses:* Patients with clinically significant cardiovascular impairment: Maximum Recommended Dose: 0.27 IU (international units) (that is 9 ml of 0.03 IU/ml or 41/2 cartridges).

Table 8.2: Showing comparison of epinephrine and norepinephrine

	Epinephrine	***Norepinephrine***
Receptor activity	Powerful stimulant of α and β receptors With higher doses α effects predominates, whereas lower doses primarily produce β receptor activity	Stimulates both α and β receptors; but α effect predominates
Blood pressure (BP)	Lesser effect	Greater increase in BP than epinephrine
Central nervous system	Greater effect of stimulation of central nervous system in large doses	Does not simulate central nervous system in therapeutic doses
Cardiovascular system	Greater effect of stimulation of cardiovascular system	
Bronchi	Dilatation	Little or no effect
Heart rate (HR)	Increase in HR is of greater degree	Increase in HR is of lesser degree

vi. *Side effects and overdose:* Studies have shown a wide margin of safety with felypressin (Klingenstrom et al, 1967).
vii. *Clinical applications:* Used as vasoconstrictor with local anesthetic to decrease their absorption and increase their duration of action.
viii. *Availability:* It is used in dentistry in a dilution of 0.03 IU (international units)/ml with 3% prilocaine.
ix. *Disadvantage:* It acts primarily on venous circulation, hence felypressin is not as effective as conventional vasoconstrictors in providing hemostasis during surgical procedures (McClymont and Crowther, 1988).
x. *Indications:*
   a. It may be safely used in patients with medical problems which contraindicate the administration of epinephrine (Anderson and Reagan 1993), which include: (i) mild to moderate cardiovascular diseases, including hypertension, and (ii) other non-cardiovascular diseases; such as hyperthyroidism.
   b. It can be used in patients who are on antidepressant drugs such as tri- or tetracyclic acid antidepressants or monoamine-oxidase inhibitors.
xi. *Contraindications:*
   a. Local anesthetic containing felypressin is not recommended for use when hemostasis is required because of their predominant effect on venous rather than arteriolar circulation (Newcomb and Wait, 1972).
   b. Pregnancy: It is not recommended for use during pregnancy because of its oxytocic action.

SELECTION OF A VASOCONSTRICTOR

The selection of an appropriate vasoconstrictor is based on following factors:
1. Length of the surgical or dental procedure
2. Requirement for hemostasis during the surgical procedure
3. Requirement for postoperative pain control
4. Medical or physical status of the patient and concurrent medications taken (Table 8.3).

Table 8.3: It indicates the various dilutions available in India and the MRD (in terms of ml) for normal healthy adult individuals and medically compromised individuals

Dilutions	*Normal adult healthy individuals (0.2 mg/appointment) (ml)*	*Medically compromised individuals (0.04 mg / appointment) (1/5th of normal dose) (ml)*
1 : 80 000	16	3.2
1 : 100 000	20	4
1 : 200 000	40	8

Length of the Surgical or Dental Procedure

The duration of pulpal and hard tissue anesthesia with 2% lidocaine only lasts for about 10 min. The addition of epinephrine of 1:50,000 or 1:100,000 dilution prolongs the duration to about 60 min (Newcomb and Waite, 1972; and Epstein, 1969).

Hence, for any oral surgical or dental restorative procedure requiring 40-50 minutes, it is difficult to achieve consistent pulpal anesthesia without inclusion of a vasoconstrictor.

Requirement of Hemostasis

Some of the vasoconstrictors are effective in minimising blood loss during surgical procedures. However, most of vasoconstrictors also produce vasodilatation as a result of decline in the tissue levels of epinephrine, which is known as rebound phenomenon.

While using epinephrine as a vasoconstrictor for hemostasis, we must consider that epinephrine is both α and β agonist. It possesses both α and β actions; and produces vasoconstriction through it's effect. When used in a dilution of 1:50,000 and even 1:100,000 (lesser degree), it produces a definite β effect, once α-induced asoconstriction has ceased.

However, felypressin stimulates venous circulation more than the arteriolar circulation, and therefore, is of minimal value in achieving hemostasis (Altura et al, 1965).

Vasoconstrictors affect microcirculation. Their principal actions are on arterioles and precapillary sphincters. Vasoconstrictors used for achieving hemostasis must be deposited into the area of surgery. Administration of a nerve block which is usually at a distance from the site of surgery, with a local anesthetic agent with a vasoconstrictor will not produce hemostasis in the area of surgery, however, the anesthesia may be profound. It requires only small volumes of local anesthetic solution with a vasoconstrictor to achieve hemostasis.

Requirement for Postoperative Pain Control

Profound pain control of adequate duration by a local anesthetic agent with a vasopressor is used. Plain local anesthetic agent produces pulpal anesthesia of shorter duration that a local anesthetic agent with a vasopressor and is likely to produce stress response.

Medical or Physical Status of the Patient and Medications Concurrently Taken

The benefits and risks of including a vasoconstrictor in a local anesthetic solution in patients who are medically compromised, must be weighed against benefits and risks of using plain local anesthetic solution (Goulet et al, 1992).

CONTRAINDICATIONS

In cases of significant cardiac and non-cardiac diseases, it is essential to:

i. Determine the degree of severity of underlying medical problems.
ii. Determine whether or not vasoconstrictor can be safely included or exclude from the local anesthetic solution.
iii. Obtain a medical consultation and the necessary information from the treating physician regarding the existing medical problem.

Once the medical status of the patient is improved or corrected, dental or surgical procedures requiring administration of local anesthetic agents with vasoconstrictors are indicated.

The groups where inclusion of vasoconstrictor is contraindicated are given below.

1. Patients with significant cardiovascular disease such as (a) ischemic heart disease, (b) hypertension, and (c) cerebral strokes.
2. Patients with certain uncontrolled non-cardiovascular diseases, such as: thyrotoxicosis or hyperthyroid states, and diabetes mellitus.
3. Patients receiving non-specific beta-blockers, monoamine-oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs) and phenothiazines.
4. Patients with sulfite sensitivity.
5. Patients who are undergoing general anesthesia with halogenated agents
6. Pregnancy.

1. Patients with significant cardiovascular disease such as (a) ischemic heart disease, (b) hypertension, and (c) cerebral strokes.

   Epinephrine and other vasoconstrictors can be used in moderate amounts in patients with mild to moderate cardiovascular disease. Felypressin has minimal cardiothoracic stimulatory actions and is nondysrhythmogenic; it is recommended for patients with significant cardiovascular disease.

   Patients with severe cardiovascular diseases are at too great a risk for elective dental therapy, such as:

   a. Ischemic heart disease
      i. Patients with history of acute myocardial infarction within last six months.
      ii. Patients with history of acute anginal episodes on daily basis or where signs and symptoms are increasing in severity (angina pectoris) (pre-infarction stage or unstable angina).
      iii. Patients with cardiac dysrhythmias despite appropriate antiarrhythmic drug therapy (Goulet et al, 1992).
      iv. Post-coronary artery bypass surgery, less than six months.
   b. Hypertension: Patients with systolic BP greater than 200 mm of Hg and diastolic BP greater than 110 mm of Hg should not receive any dental care unless BP is corrected.
   c. Cerebral strokes: Patients with history of less than six months after cerebrovascular accident.

Epinephrine and other vasoconstrictors can be used in moderate amount in patients with mild to moderate cardiovascular disease. Felypressin has minimal cardiovascular stimulatory actions and is nondysrhythmogenic; it is recommended for significant cardiovascular risk patients.

2. Patients with certain uncontrolled non-cardiovascular diseases, such as: thyrotoxicosis or hyperthyroid states and diabetes mellitus. Medical treatment of the conditions and a consent from the treating physician is necessary to use vasoconstrictor in such conditions.
   a. Thyrotoxicosis or hyperthyroid states: Epinephrine is contraindicated in patients with clinical evidence of hyperthyroid states (Goulet et al, 1992). The signs and symptoms include: Exophthalmos, tremors, hyperhydrosis, increased basal metabolic rate, irritability and nervousness, increased body temperature, inability to tolerate heat, increased HR, and increased BP.
   b. Diabetes mellitus: Diabetes is not only a metabolic, and endocrinal disease, but also a vascular one. Microcirculatory changes will result in impaired blood flow to the tissues. The inclusion of a vasoconstrictor in local anesthetic solution may further compromise the inadequate blood supply, and result in local ischemia and tissue sloughing.
3. Patients receiving non-specific beta-blockers, monoamine-oxidase inhibitors (MAOI), tricyclic and tetracyclic antidepressants and phenothiazines for psychiatric ailments.
   a. Patients taking monoamine-oxidase inhibitors (MAOI) are not at an increased risk provided the doses used are within the recommended range (Goulet et al, 1992; and Verrill, 1975).
   b. Patients receiving tricyclic and tetracyclic antidepressants: Such patients are at a risk of developing dysrhythmias with administration of epinephrine. Hence, its dose should be minimal. The administration of levonordefrin or norepinephrine in patients receiving tricyclic antidepressants is absolutely contraindicated. Large doses may induce severe (exaggerated) response. Felypressin has minimal cardiothoracic stimulatory actions and is nondysrhythmogenic, and hence can be used.

   In all the abovementioned conditions felypressin can safely be used.
4. Patients with sulfite sensitivity: Local anesthetic solutions contain an antioxidant, which retards oxidation of the vasoconstrictor. The most commonly used antioxidant in dental cartridges is sodium bisulfite.
5. Patients who are undergoing general anesthesia with halogenated agents: Epinephrine should not be used as a vasoconstrictor during GA when a patient is receiving an inhalational halogenated anesthetic agents

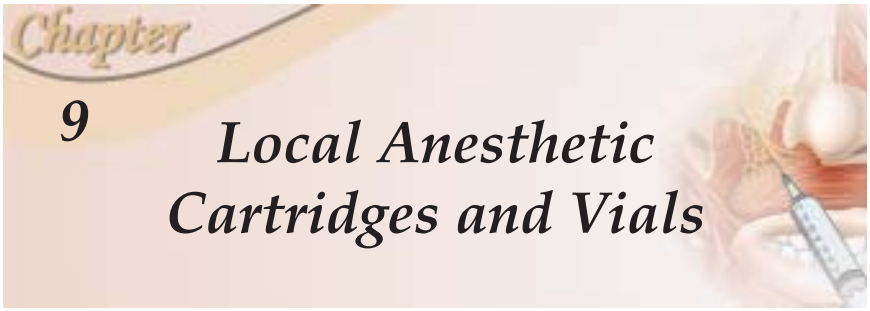
such as halothane, methoxyflurane, and ethrane. These vasoconstrictors cause cardiac dysrhythmias. In such situations, felypressin is recommended.

6. Pregnancy: Felypressin is not indicated in pregnant patients because of its potential oxytocic actions.

BIBLIOGRAPHY

1. Ahlquist RP. A study of adrenotropic receptors. Am J Physiol 1948;153:586-600.
2. Altura BM, Hershey SG, and Zweifach BW. Effects of a synthetic analogue of vasopressin on vascular smooth muscle. Proc Soc Exp Biol Med 1965;119:258-61.
3. Anderson LD, and Reagan SE. Local anaethetics and vasoconstrictors in patients with compromised cardiovascular systems. Gen Dent 1993;41(2):161-64.
4. Brown G. The influence of adrenaline, noradrenaline as vasoconstrictors on the efficacy of lidocaine. J Oral Ther Pharmacol 1968;4:398-405.
5. Buckley JA, Ciancio SG, and McMullen JA. Efficacy of epinephrine concentration in local anaesthesia during periodontal surgery. J Periodontol 1984;55:653-57.
6. Cannall H, Walters H, Beckett AH, and Saunders A. Circulating blood levels of lignocaine after peri-oral injections. Br Dent J 1975;138:87-93.
7. Carpenter RL, Jopacz DJ, and Mackey DC. Accuracy of Doppler capillary flow measurement for predicting blood loss from skin incision in pig. Anesth Analg 1989;68:308-11.
8. Cowan A. Further clinical evaluation of prilocaine (citanest), with and without epinephrine. Oral Surg Oral Med Oral Pathol 1968;26:304-11.
9. de Jong RH. Uptake, distribution, and elimination. In de Jong RH: Local anaesthetics, St Louis, Mosby-Year Book 1994.
10. Epstein S. Clinical study of prilocaine with varying concentrations of epinephrine JADA 1969;78:85-90.
11. Goulet JP, Perusse R, and Tercotte JY. Contraindications to Vasoconstrictors in Dentistry: Part I. Cardiovascular diseases. Oral Surg Oral Med Oral Pathol 1992;74:(5):679-86.
12. Goulet JP, Perusse R, and Tercotte JY. Contraindications to Vasoconstrictors in Dentistry: Part II. Hyperthyroidism, diabetes, sulfite sensitivity, corticodependent asthma, and pheochromocytoma. Oral Surg Oral Med Oral Pathol 1992;74:(5):687-91.
13. Goulet JP, Perusse R, and Tercotte JY. Contraindications to Vasoconstrictors in Dentistry: Part III. Pharmacologic Interactions. Oral Surg Oral Med Oral Pathol 1992;74:(5):692-97.
14. Howe GL. Minor Oral Surgery. 3rd ed. Butterworth-Heinemann Ltd., Mumbai: Varghese Publishing House, Reprint 1985.
15. Kaplan EL, editor: Cardiovascular Disease in Dental Practice. Dallas, American Heart Association 1986.
16. Klingenstrom P, Nylen B, and Westermark L. A clinical comparison between adrenaline and octapressin as a vasoconstrictor in local anaesthesia. Acta Anaesthesiol Scand 1967;11:35.

17. Lands AM, Arnold A, McAuliff JP, et al. Differentiation of receptor systems activated by sympathomimetic amines. Nature 1967;214:597-98.
18. Laskin DM. Oral and Maxillofacial Surgery Vol I and II C. V. Mosby Co. AITBS Publishers India 1992.
19. Malamed SF. Handbook of Local Anaesthesia.4th ed. Harcourt India Private Limited, Mosby 1997.
20. McClymont LG, and Crowther JA. Local anaesthetic with vasoconstrictor combination in septal surgery. J Laryngol Otol 1988;102:793-95.
21. Myers RR, and Heckman HM. Effects of local anaesthesia on nerve blood flow: Studies using lidocaine with or without epinephrine. Anesthesiology 1989;71:757-62.
22. Newcomb GM, and Wait IM. The effectiveness of local analgesic preparations in reducing haemorrhage during periodontal surgery. J Dent 1972;1:37-42.
23. Peterson LJ, Ellis E III, Hupp JR, and Tucker MR. Contemporary Oral and Maxillofacial Surgery. 2nd ed. Harcourt Brace and C. Asia. Pvt. Ltd. 1997.
24. Richard Bennett C. Monheim's Local Anaesthesia and Pain Control in Dental Practice. 7th ed. Delhi: CBS Publishers and Distributors 1990.
25. Robin Woods. A Guide to the Use of Drugs in Dentistry. 12th ed. Sydney: Australian Dental Association Inc 1996.
26. Satoskar RS, Bhandarkar SD, and Ainapure SS. Satoskar, Kale, and Bhandarkar's Pharmacology and Pharmacotherapeutics. Revised 16th ed. Mumbai: Popular Prakashan 1999.
27. Use of epinephrine in connection with procaine in dental procedures: Report of a Special Committee of the New York Heart Association, Inc, on the use of epinephrine in connection with procaine in dental procedures. J Am Dent Assoc 1950;50:108.
28. Verrill PJ. Adverse reactions to local anaesthetics and vasoconstrictor drugs. Practitioner 1975;214:382-87.
29. Wildsmith JAW, Tucker GT, Cooper S, et al. Plasma concentrations of local anaesthetics after interscalene brachial plexus block. Br J Anaesth 1977;49:461.



It contains primarily the local anesthetic drug, and also the other ingredients, which are as follows:

1. Local anesthetic drug
2. Vasopressor/vasoconstrictor drug
3. Preservative for vasopressor
4. Sodium chloride (NaCl) or Ringer's solution
5. Distilled water
6. General preservatives.

LOCAL ANESTHETIC DRUG

It provides pain control during dental therapy. It interrupts propagation of impulse preventing it from reaching brain.

Drugs are listed by their percentage (%) concentration. The number of mg of an agent contained in the cartridge can be calculated by multiplying the percentage (%) concentration (e.g. 2% = 20 mg/ml) by 2 (the number of ml in a cartridge). Thus, a cartridge containing 2 ml of 2% local anesthetic solution contains 40 mg of local anesthetic agent.

The local anesthetic solution is quite stable, capable of being autoclaved, heated or boiled without deterioration. However, other contents of cartridge, such as vasopressor and cartridge seal are more labile. These are broken down easily (Tables 9.1 and 9.2).

VASOPRESSOR/VASOCONSTRICTOR DRUG

It is added in various concentrations, to some dental cartridges to increase safety and prolong duration of action of local anesthetic agents. It also helps in controlling bleeding. The pH of dental cartridge containing local anesthetic agent with a vasoconstrictor is lower (more acidic) than that without a vasoconstrictor (pH of 3.3-4.0 v/s 5.5-6.0). Because of this pH difference plain anesthetics have somewhat more rapid or clinical action and are more comfortable (less burning on injection).

Table 9.1: Showing the typical constituents of a local anesthetic solution with adrenaline in the ratio of 1:80,000 in the form of a cartridge and a multidose vial in India

Constituents	*Amount*
Lignocaine hydrochloride (2%)	24.64 mg
Adrenaline bitartarate (1:80,000)	0.0125 mg
Sodium bisulfite	
Thymol	0.4 mg
Chlorbutol	5.0 mg
Ringer's solution	0.29 ml
Water for injection	To make 1 ml

Table 9.2: Showing the typical constituents of a local anesthetic solution with adrenaline in the ratio of 1:200,000 in the form of a multidose vial in India

Constituents	*Amount*
Lignocaine hydrochloride (2%)	21.30 mg
Adrenaline bitartarate (1:200 000)	0.005 mg
Sodium meta-bisulphite	0.5 mg
Methylparaben	1.0 mg
Sodium chloride	6.0 mg
Water for injection	To make 1 ml

PRESERVATIVE FOR VASOPRESSOR

Local anesthetic solutions containing vasoconstrictors also contain a specific agent, an antioxidant, that acts as a preservative for vasoconstrictors. The most frequently used antioxidant is sodium-bisulfite or sodium metabisulfite. It prevents biodegradation of vasopressor by O_2 which might be present in the cartridge either introduced during manufacture, or which got diffused through semipermeable membrane or the rubber diaphragm after filling at the time of storage of the cartridge. Sodium-bisulfite reacts with O_2 before O_2 can destroy vasopressor. Sodium-bisulfite is oxidised to sodium-bisulphate, a chemical with even lower pH (acidic), than before oxidation.

Clinical Relevance

It lies in the fact that increased burning (discomfort) is experienced on injection of an "older" cartridge with a vasopressor than with a fresher cartridge.

Once the cartridge container is opened, it should be used within a reasonable time. Local anesthetic solutions without vasoconstrictors have a shelf-life of about 48 months. Local anesthetic solutions containing vasoconstrictors have their shelf-life reduced to 18 and 12 months for epinephrine and phenylephrine; and norepinephrine and levonordefrine, respectively. This is so because of the instability of the vasoconstrictors. Hence fresh solutions produce better analgesia and cause less irritation to the tissues.

Allergy to bisulfites must be considered in pre-anesthetic medical evaluation.

SODIUM CHLORIDE (NaCl) OR RINGER'S SOLUTION

It is added to the contents of dental cartridge to make the solution isotonic with the tissues of the body. Hypertonic solution produces tissue edema, paresthesia, sometime lasting for several months following drug administration.

DISTILLED WATER

It is used as a diluent to provide the volume of solution in the dental cartridge.

GENERAL PRESERVATIVES

A number of chemicals are used as general preservatives. These are added to increase the shelf-life; and include: (i) Methylparaben, (ii) Thymol, and (iii) Chlorbutol.

i. Methylparaben: It is a bacteristatic and fungistatic agent. It has been excluded from cartridges in USA, from 1st January 1984, following reports of allergy. However, it is still used in multidose vials in some countries including India.
ii. Thymol: It is antiseptic, fungistatic, and antihelminthic.
iii. Chlorbutol.

BIBLIOGRAPHY

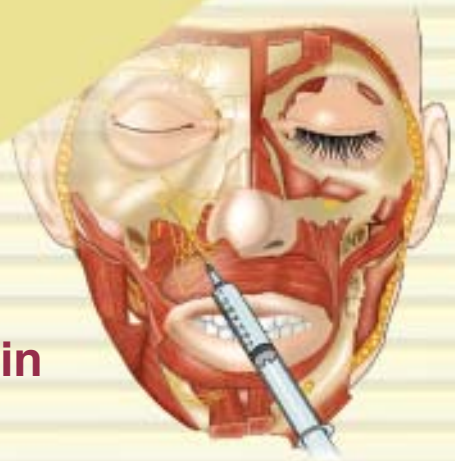
1. Aldous JA. Needle deflection; a factor in the administration of local anaesthetic. J Am Dent Assoc 1977;77:602-04.
2. Council on Dental Material and Devices: New American National Standard Institute/ American Dental Association Specification no. 34 for dental aspirating syringes. J Am Dent Assoc 1978;97:236-38.
3. Council on Dental Material, Instruments, and Equipment: Addendum to American National Standard Institute/American Dental Association Specification no. 34 for dental aspirating syringes. J Am Dent Assoc 1982;104:69-70.

4. Figge FHJ, Scherer RP. Anatomical studies on jet penetration of human skin for subcutaneous medications without the use of needles. Anat Rec 1947;97:335 (abstract).
5. Jeske AH, Boshart BF. Deflection of conventional versus non-deflecting dental needles in vitro. Anaesth Prog 1985;32:62-64.
6. Malamed SF. Handbook of Local Anaesthesia. Noida: Mosby. Harcourt India Pvt Ltd., Reprinted ed. 2001.
7. Margetis PM, Quarantillo EP, Lindgerg RB. Jet injection local anaesthesia in dentistry: A report of 66 cases. US Armed Forces Med J 1958;9:625-34.
8. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. Delhi: CBS Publishers and Distributors.
9. Robison SF, Mayhew RB, Cowan RD, Hawley RJ. Comparative study of deflection characteristics and fragility of 25-, 27-, and 30-gauge short dental needles. J Am Dent Assoc 1984;109:920-24.

The names of the drugs are mentioned here for the benefit of the readers. The author and the publisher do not have any financial relationship with the companies manufacturing those drugs.

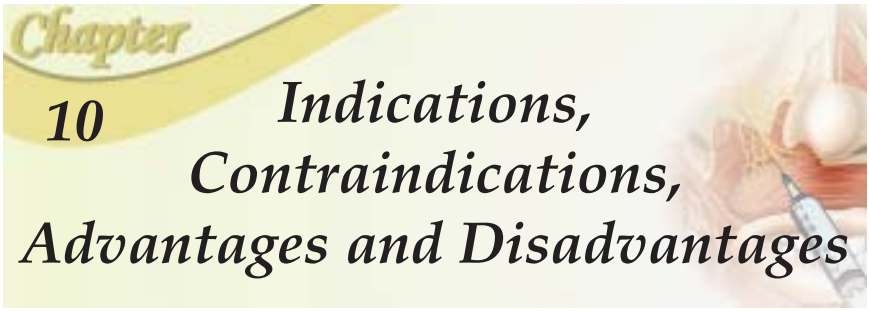
Section

3



Local Anesthesia in Dentistry

- Indications, Contraindications, Advantages and Disadvantages
- Methods of Pain Control



Chapter 10 Indications, Contraindications, Advantages and Disadvantages

There are two methods for obtaining anesthesia in dentistry, local and general.

1. *Local anesthesia:* It is a method, whereby a certain operative area is made insensitive to pain, without loss of consciousness. The sensory or the efferent nerves are blocked at the periphery or at any point between operative field and the center in the brain.
2. *General anesthesia:* It is a method, whereby a certain operative area is rendered insensitive to pain, with loss of consciousness, by blocking brain function.

DEFINITIONS

The word "anesthesia" is derived from Greek language. The words "an" means without; and "aisthetos" means sensation. The word was coined by Dr Oliver Wendell Holmes in 1846.

"Anesthesia" means loss of all forms of sensation including pain, touch, temperature and pressure perception and may be accompanied by impairment of motor function.

Another term "analgesia" is used to denote loss of sensation unaccompanied by loss of other forms of sensation. "Regional analgesia" refers to loss of pain sensation over a specific part of the body without loss of consciousness. "Regional anesthesia" denotes loss of pain sensation as well as interruption of all other forms of sensations including temperature, pressure, and motor function over a specific area of the body. As a general rule, a larger dose of the agent is required to obtain anesthesia than to obtain analgesia. When only a part of the body is involved then the terms "local analgesia" and "local anesthesia" are used.

"Local anesthesia" is defined as loss of sensation in a circumscribed area of the body caused by, (i) a depression of excitation in nerve endings, or (ii) an inhibition of conduction process in the peripheral nerves; without loss of consciousness.

The terms "regional analgesia" and "regional anesthesia" are often used interchangeably and indiscriminately, despite the difference in the area of drug action.

INDICATIONS

Whenever it is desirable to keep the patient in the state of consciousness while insensibility to pain is produced in the teeth and the supporting structures. In general, local anesthesia is used to render the teeth and supporting structures insensitive to painful procedures. Specifically, it is used for the following:

Oral Surgery

1. To make needle insertion painless
2. Extraction of teeth and fractured roots
3. Odontectomy
4. Treatment of alveolalgia
5. Alveolectomy
6. Apicoectomy
7. Incision and drainage of localised abscesses
8. Removal of cysts, residual infection areas, hypertrophic tissues and neoplastic growths, ranula and salivary calculi
9. In the treatment of tic douloureux by producing prolonged anesthesia with a combination of a local anesthetic agent and alcohol injection, for blocking the involved nerve
10. A therapeutic test to localise the source of vague pain about the face.

Conservative Dentistry

The following operative and restorative procedures:

i. Cavity preparation
ii. Crown and bridge abutment preparation
iii. Pulpotomy or pulpectomy.

Periodontology

i. Surgical treatment of periodontal diseases
ii. Deep scaling and prophylaxis treatment
iii. Mucogingival surgical procedures.

Prosthodontics

Giving denture patients relief from sore spots which are painful even though dentures have been relieved.

Orthodontia

Separation of teeth.

Radiology

To prevent gagging and retching caused by the contact of film with palatal tissues and posterior part of oral cavity. These tissues or the areas are anesthetised before placing the film. In theses cases usually surface anesthesia is used.

CONTRAINDICATIONS

These can be divided into two groups:

(a) Absolute contraindications, and (b) Relative contraindications

Absolute Contraindications

History of Allergy to Local Anesthetic Agents

Local anesthetic agents belonging to the same chemical group should not be used. However, local anesthetic agents in the different chemical group can be used. In case, a patient gives history of allergy to an amide local anesthetic agent, an ester local anesthetic agent can be used.

Documented Allergy to Other Constituents of the Solution Such as Bisulfites and Preservatives

History of allergy to any of the constituents of the local anesthetic solution. Bisulfites, in the form of Sodium-bisulfite and sodium-metabisulfite are used as anti-oxidants or as preservatives to the vasoconstrictor in the local anesthetic solutions. Other general preservatives present in the local anesthetic solution include thymol, methylparaben, and chlorbutol. The solution containing the constituent should be avoided. An alternate solution should be used, if possible.

Relative Contraindications

1. *Fear and apprehension:* Where the patient is uncooperative or refuses for regional analgesia.
2. *Presence of acute inflammation or suppurative infection at the site of insertion of the needle:* There are increased chances of dissemination of infection with the passage of the needle from the abscess area to the deeper tissues.
3. *Infants or small children:* These patients lack reasoning and understanding.
4. *Mentally retarded patients:* These patients are unable to cooperate.
5. *Restricted mouth opening:* When the patient cannot open his mouth sufficiently, in situations, such as (i) trismus, or (ii) partial or complete ankylosis of temporomandibular joint.
6. *Patients with significant medical disease:* (a) cardiovascular disease, (b) hepatic dysfunction, (c) renal dysfunction, and (d) clinical hyperthyroidism.

a. *Patients with significant cardiovascular disease:* All local anesthetic solutions containing high concentrations of vasoconstrictor, such as epinephrine, as in gingival retraction cords, should be avoided. Local anesthetic agents containing higher dilution of epinephrine, such as 1:100 000 or 1:200 000, or 3% mepivacaine or 4% prilocaine can be used (for nerve blocks).
b. *Patients with significant hepatic dysfunction:* All local anesthetic agents belonging to amide group undergo biotransformation in liver (fixed function oxidases). These agents are best avoided, if not, should be used judiciously.
c. *Patients with significant renal dysfunction:* All amides and esters should be avoided, however, these can be used judiciously.
d. *Patients with clinical hyperthyroidism:* High concentrations of vasoconstrictor, as in epinephrine gingival retraction cords, should be avoided. Local anesthetic agents containing higher dilutions of epinephrine such as 1:100 000 or 1:200 000, or 3% mepivacaine or 4% prilocaine (nerve blocks) can be used.

7. *Major surgical procedures*
8. *Presence of certain anomalies or developmental defects:* These situations make regional analgesia difficult or impossible.
9. *Presence of congenital methemoglobinemia:* It can be idiopathic or congenital.

a. Articaine and prilocaine when used in large doses, can produce methemoglobinemia (Bellamy et al, 1992). These two drugs should be avoided in patients with congenital methemoglobinemia, and other clinical syndromes with reduced O_2 carrying capacity of blood; because, there is increased risk of producing clinically significant methemoglobinemia. These agents may be administered, if absolutely necessary; however, their dose should be minimised. The maximum recommended dose of prilocaine is 8 mg/kg body weight.
Methemoglobinemia is less likely to occur at lower doses. Alternatively, local anesthetic agents belonging to other groups both amides or esters can be used.
b. Benzocaine, a topical local anesthetic agent, when used in very large doses can also induce methemoglobinemia (Guertler and Pearce, 1994; and Rodriguez et al, 1994).

10. *Presence of atypical plasma cholinesterase:* Cholinesterase, is an enzyme, present in plasma, and is required for biotransformation of all esters by causing hydrolysis.
This condition is a relative contraindication to the use of ester type of local anesthetic agents. These agents undergo biotransformation (hydrolysis) in the blood by the enzyme plasma cholinesterase, which is produced in the liver. These agents may be used, if necessary, however,

the dose is minimised. In such situations, it is preferable to use an amide type of local anesthetic agent. Amides do not present any risk of high blood levels, because these agents undergo biotransformation in the liver.

ADVANTAGES

1. Patient remains awake and cooperative.
2. Little distortion of normal physiology; therefore can be used in poor risk patients.
3. Low incidence of morbidity.
4. Patient can leave hospital unescorted.
5. Additional trained personnel not required.
6. Technique not difficult to master.
7. Percentage of failure is small.
8. No additional expense to the patient.
9. Patient need not miss the previous meal. In fact, should have one. Patients should not come on empty stomach.

DISADVANTAGES

No true disadvantage to the use of regional analgesia; when the patient is mentally prepared and when there are no contraindications. In every instance, when satisfactory anesthesia can be achieved and the patient is cooperative, regional analgesia is the method of choice.

BIBLIOGRAPHY

1. Archer WH. A Manual of Dental Anesthesia: An illustrated guide for students and Practitioners. 2nd ed. WB Saunders Company 1958.
2. Bellamy MC, Hopkins PM, Hallsall PJ, Ellis FR. A study into the incidence of methaemoglobinemia after "3-in-1" block with prilocaine. Anaesthesia 1992;47(12):1084-85.
3. Guertler AT, Pearce WA. A prospective evaluation of benzocaine-associated methaemoglobinemia in human beings. Ann Emerg Med 1994;24(4):626-30.
4. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
5. Prilocaine-induced methaemoglobinemia-Wisconsin, 1993, MMWR Morb Mortal Wkly Rep 1994;43(35):655-57.
6. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. New Delhi: CBS Publishers and Distributors 1990.
7. Rodriguez LF, Smolik LM, Zbehlik AJ. Benzocaine-induced methaemoglobinemia: A report of a severe reaction and review of the literature. Ann Pharmacother 1994;28(5):643-49.

There are various methods which are used for pain control, and these are as follows:

a. Methods affecting pain perception:
   i. Removal of the cause
   ii. Blockage of the pathway of painful impulses
b. Methods affecting both pain perception and pain reaction
   i. Raising the threshold of pain
c. Methods affecting pain reaction
   i. Prevention of pain reaction by causing cortical depression
   ii. Use of psychosomatic methods

The first two methods affect pain perception, the last two affect pain reaction, and the third may affect both aspects of pain.

METHODS

Methods Affecting Pain Perception

Removal of the Cause

This method of pain control clearly affects pain perception. The removal of the cause would, of course, be the desirable method of controlling pain. If this could be achieved, the environmental change in tissue would be eliminated; as a result, free nerve endings would not be excited and no impulses would be initiated. It is necessary that any removal of cause leave no permanent change in the tissues, as this condition would then create the impulse, even though original causative factors are eliminated. The removal of the cause would include controlling the infection with the help of antibiotics, by performing surgery, or by immobilization of the fractured fragments, etc.

Blockage of the Pathway of Painful Impulses

This method of pain control is possible by interfering with pain perception. It is the most widely used method in dental practice for controlling pain. By this method a suitable drug, possessing local analgesic properties, is

injected into the tissues in proximity to the nerve or nerves involved. The local anesthetic solution prevents depolarization of the nerve fibers at the area of absorption, thus preventing those particular fibers from conducting any impulses in the central direction beyond that point. As long as the solution is present in the nerve in sufficient concentration to prevent depolarization, the block will be in effect. The various techniques by which the nerve pathways can be blocked are by injection of a local anesthetic agent, or alcohol, ether, or by employing Leriche's method. The theories and mode of action of local anesthetic agents are discussed in detail in Chapters 4 and 6.

Methods Affecting Both Pain Perception and Pain Reaction

Raising the Threshold of Pain

Presently, the dental practitioner has become more aware of and has shown a greater interest for this method of pain control. This method of raising the pain threshold depends on the pharmacological action of drugs possessing analgesic properties; such as analgesic and anti-inflammatory agents.

These drugs raise the pain threshold centrally and therefore interfere with pain reaction. In this method of pain control, the cause of the original stimulus may still be present. The neuroanatomical pathways are intact and able to conduct impulses. In other words, pain perception is unaffected, but pain reaction is decreased and thus the pain reaction threshold is raised. However, it should be understood that the pain threshold can be raised to limited degrees only, depending on the drugs used. It is physiologically impossible to eliminate pain of the most severe degree by raising the pain threshold alone. In other words, the presence of more noxious stimuli creating severe pain necessitates blocking the pathway of the impulse or completely depressing pain reaction by a different method altogether, such as the use of a general anesthetic agent.

There are various drugs possessing analgesic properties in varying degrees, and some are more effective in raising the pain threshold than others. Certain drugs such aspirin (acetylsalicylic acid) are effective only in the relief of mild discomfort. On the other hand, the narcotics, although not pure analgesics (because they also possess hypnotic properties), are effective against more severe pain because they are able to raise the pain threshold to a greater degree.

All the drugs used to raise the pain threshold have optimal doses. Any increase in the dosage beyond this limit does not further increase the analgesic effective of the drug, without producing undesirable side effects. For example, 10 grains (650 mg) of aspirin may be the effective maximum

dosage; increasing the dosage cannot further raise the pain threshold. Morphine also has a maximal effective dose; any increase beyond this dosage, may decrease pain reaction by producing sleep or severe depression of central nervous system, rather than reducing the pain threshold alone.

As described earlier, certain agents have the ability to change the patient's mind through actions on cortical and subcortical centers. Fear, anxiety and apprehension are reduced, and augmentation of the descending control mechanism occurs because of this action. By modulating painful input at first synaptic levels, the perception of pain is reduced.

Methods Affecting Pain Reaction

Prevention of Pain Reaction by Causing Cortical Depression

The eliminating of pain by cortical depression is by means of the general anesthetic agents, and narcotic agents. By this method, the anesthetic agent prevents any conscious reaction to a painful stimulus, by depression of the central nervous system. In those cases in which the cerebral cortex is depressed only to the point that the inhibitions are suppressed, the patient may become hyper-reactive to a painful stimulus. Therefore any stimulation should be avoided in these instances.

Use of Psychosomatic Methods

Very often the psychosomatic approach to elimination or control of pain is neglected in dental practice. By this method so much can be gained with so little ill effects on the patient. This method affects both pain perception and pain reaction and depends for its effectiveness on putting the patient in the proper frame of mind. It is amazing what can be accomplished without the use of drugs when the patient's faith and confidence are gained.

One of the most important factors in this approach is honesty and sincerity towards the patient. This factor necessitates keeping the patient well informed about the procedure and what might be expected out of the procedure. The patient should be made to understand by a kind, considerate approach the extent of the discomfort that may be expected. Also, patients should be assured that any unpleasant sensory experience can be adequately controlled by the knowledge and methods available, and that these would be used in case of any discomfort that would arise. Patients like to believe that their comfort is of prime consideration to the dental practitioner. Once they feel secure, they will tend to tolerate unpleasant sensations to a higher degree. In this manner, pain reaction is depressed and the pain threshold is inversely raised. The patient's central control mechanism aids in the control of pain perception when non-pharmacological methods are used to alter the patient's frame of mind.

THERAPEUTIC PROCEDURES

There are various therapeutic procedures which are used in the management of pain:

1. Elimination of local pathological process
2. Physiotherapy and supportive treatment
3. Decompression of the nerve
4. Neurorrhaphy
5. Injection of the nerve with local anesthetic agents such as lidocaine
6. Injection of nerve with alcohol
7. Extracranial nerve section and nerve avulsions
8. Intracranial sections of posterior sensory root.

Elimination of Local Pathological Process

Facial pain is often associated with dental pathological processes. Hence, it is mandatory to find out and treat them. The presence of any condition that may be the source of pain should be eliminated by the most suitable or efficient method.

Teeth with chronic pulpitis should be treated by pulp extirpation or extraction of the tooth.

Periapical and periodontal abscesses require extraction of teeth. Unerupted/impacted teeth require surgical removal. Various diseases of bone and diseases of maxillary sinus require surgical intervention.

Physiotherapy

The use of heat for the relief has stood the test of time. The treatment is indicated, in case, if after removal or treatment of the cause, the pain persists.

Hot, moist towels, heated sandbags and modern electrical pads frequently give comfort but should be used as accessory treatment; after elimination of the cause of pain.

Decompression of Nerve

This is a procedure which consists of removing anything that impinges on the nerve and causes pain or numbness. This is indicated in cases of obstruction of the nerve canal by sclerotic bone.

Neurorrhaphy

Nerve regeneration in bony canals frequently occurs without surgical interference, since the canal acts as a guide to growth and union according to DeBats and Philips (1944).

Tinel's sign, is generally used as an indication of the start of nerve regeneration. It is elicited by percussion over the divided nerve, which results in a tingling sensation, in the part supplied by the peripheral section. Pin and needle sensation, too, are early signs of nerve uniting.

Injection of the Nerve with Local Anesthetic Agents Such As Lidocaine

The nerve, may be injected with lidocaine, in cases of neuralgia, which is not of the symptomatic type and in which no aetiological factor can be found. This method, which is designed to break the pain cycle, is known in France, as the method of Leriche. This method was also advocated by Arlotta (1951). The injection of lidocaine is recommended for atypical neuralgia in any part of the face.

Operative Procedure

An injection of 2-4% novocaine is made into the nerve trunk. Some patients get considerable relief after one injection. However, the injection can be repeated as often as 6-7 times. There may be recurrence of the pain but generally the patient gets relief for several months. The injections may be repeated if the pain recurs.

The Inferior Alveolar Nerve may be injected at the mandibular foramen, the Mental Nerve at the mental foramen, and the Infraorbital Nerve at the infraorbital foramen.

Complications

The complications as reported by Arlotta involve transitory edema at the site of injection, trismus of medial pterygoid muscle, and occasionally elevation of the temperature.

Injection of the Nerve with Alcohol

Indications

The injection of alcohol is recommended for the following conditions:

i. Atypical neuralgia, the cause of which cannot be found.
ii. Tic douloureux, if section or avulsion of the nerve is contraindicated because of the general health of the patient.
iii. Intractable pain in cases of incurable disease especially cancer of the mouth.

It is a good practice, first to make a test with an anesthetic solution, to find out if the pain is relieved. The patient also gets an opportunity to experience the feeling of numbness associated with this method of

treatment. If the patient finds the numbness worst than the pain, the symptoms may not be severe enough to be treated by alcohol injection, or nerve avulsion. In case of a tic douloureux, the patient will be glad to accept the anesthesia associated with pain relief.

Anesthesia

The injections are generally made without the aid of GA, since the reaction of the patient gives important information regarding the insertion of the needle. When the end of the nerve supplying the part is pricked, pain is elicited. A narcotic analgesic may be given to reduce the sensibility of the patient.

Operative Procedure

The injections are made either with a Leur-Lok syringe, with a needle of adequate configuration, or can be executed in the same manner as for local anesthesia.

Extraoral injections into the second and the third divisions of trigeminal nerve at the foramen in the base of the skull through which they emerge and injections into the Gasserian Ganglion are generally considered the most successful methods.

Some workers favor injection into smaller nerve branches, especially those which can be reached through foramina.

Gasserian Ganglion Injection

The injection of Gasserian Ganglion is generally used:

i. When the attempts with injection of the main branches is rendered unsuccessful, or
ii. When the peripheral nerve avulsion has failed to give relief.

Recurrence is not as common as in the case of injection of the trigeminal nerve. Ullik (1951), in his report of 104 injections had recurrence in 13.6% of the patients.

The injection is made under neurologic control, which gives precise information regarding the position of the needle. This is important since a needle passing through the foramen ovale, does not necessarily enter the ganglion. If the needle is in the ganglion, the injection will at once eliminate skin sensitivity. In case, anesthesia does not occur, Pichler (1948) recommended insertion of a second and even a third needle.

Peripheral Alcohol Injections

These injections are made into the peripheral nerve, so as to destroy the nerve fibers. Since the nerve fibers have a great power of regeneration, the

result of alcohol injections is not as long-lasting, as that of the injection of the ganglion.

The infraorbital and the mental foramina are excellent places for injection of the involved nerves, respectively. The infraorbital injection may be made from either an extraoral or an intraoral approach. In some cases, especially, after recurrence, the nerve may be exposed by means of an intraoral incision, and the needle can be inserted into it under direct vision. The sphenomaxillary ganglion and the posterior part of the infraorbital nerve may be reached through the palatine canal by means of an angulated needle.

The mental injection is made intraorally. Its location does not lend itself readily to a deep insertion of the needle. The amount of solution used is 0.5 to 1 ml. When the needle strikes the nerve, a sharp pain is felt in the lip or in the area supplied by it. Injections at the mandibular foramen are not recommended but injections of the entire second division at the foramen rotundum and the third division at the foramen ovale may be made in cases where the peripheral injections fail to give complete relief.

For all injections, 95% alcohol may be used, or the following solution:

Procaine/monocaine	2%
Chloroform	5%
Absolute alcohol	70%
Ringer's solution	23%

Generally, slight force is required to introduce the solution. An amount of 0.5 to 0.75 ml is injected. If the injection is successful, the anesthesia lasts for 6 months to 1½ years. At this time another injection may be necessary.

Complications

The complications include the following:

i. Temporary palsy of external rectus muscle following the injection of second division,
ii. Facial paralysis following injection of the third division, and
iii. Ulceration of the mouth following injection of second division.

Disadvantages

The main disadvantage of this method is that:

i. It must be repeated almost annually,
ii. The injection is dreaded buy the patient to the extent that he is reluctant to undergo another.

For these reasons, surgery is generally more satisfactory since section and avulsion are performed under anesthesia and in most cases give permanent relief.

Extracranial Nerve Section and Nerve Avulsion

These methods are more effective and more lasting than injection with alcohol. They give satisfactory results only if the pain originates in the peripheral branch. If the pain is of central origin, located in Gasserian Ganglion, or beyond, this method is of no avail.

One may ascertain, by means of a local monocaine or procaine block, of the nerve to be removed, whether its removal will give the desired relief; if not, division posterior to the ganglion is indicated.

Indications

The sectioning of the nerve or excision of a small portion of the nerve, is generally done when the nerve is outside the bone, while nerves within a bony canal are avulsed to prevent reunion of the nerve.

The indications are as following:

i. Injured nerves in cases of persistent paresthesia or atypical neuralgia.
ii. Severed nerves which have produced amputation neuromas that cause pain and tenderness, and when a neurorrhaphy cannot be performed.
iii. Trigeminal neuralgia or tic douloureux.

Details of Operative Procedure

General anesthesia is preferred. The procedure of choice is avulsion rather than peripheral sectioning, since it gives a greater assurance that regeneration will not occur.

The operations are performed for the following individual nerves/ branches:

(1) Inferior Alveolar Nerve, (2) Infraorbital Nerve, and (3) Lingual Nerve.

Intracranial Section of the Posterior Sensory Root

Division of the sensory root of the fifth cranial nerve posterior to the ganglion (retrogasserian neurectomy) generally gives permanent relief from pain even in the most severe cases of tic douloureux. The temporal approach just above the zygoma, is generally used, as recommended by Frazier (1925). This section is a differential one; the fibers which originate in the ophthalmic division are left intact, preserving the corneal reflex, and either the second or third division may be cut depending on the location of the pain.

Munro (1940) stated that Dandy's approach, which consists of cutting the nerve in the posterior and with it the ninth nerve as well, is of advantage in malignancies as it affects the nerves of the throat as well as the face.

Walter Dandy was the first to describe pulsatile, vascular compression (at Root Entry Zone or REZ) as a cause of TN. However, we now know that vascular compression can be arterial, venous or both, and though often at REZ, may be further away along the course of the nerve up to Gasserian Ganglion (GG).

Peter Jannetta pioneered the thoughts and conclusively proved in a large series of patients whom he explored and showed convincingly the pathological cause to be compression of trigeminal nerve by an abnormal loop of normal anatomical vessel(s). In such cases, decompression is done through intracranial approach.

Complications

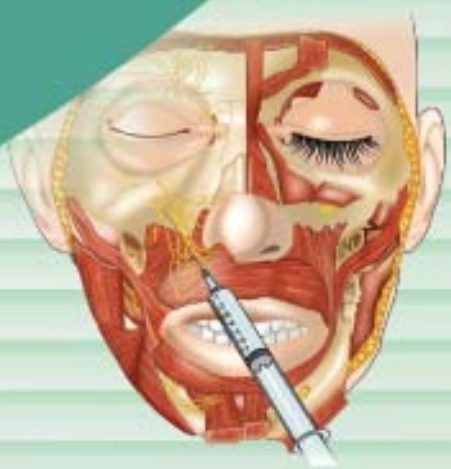
One of the complications of the sectioning of sensory root is that:

i. The petrosal nerve is cut, which may cause dryness of the eye, favoring the formation of an ulcer of the cornea.
ii. Partial facial paralysis.
iii. Injury to the III and VI Cranial Nerves may occur.

BIBLIOGRAPHY

1. Arlotta AA. Le traitment des nevralgies du trijumeau par les injections de novocaine, Rev. Belge de stomatol 1951;48:217.
2. DeBats ML and Philips WH: Nerve Injury Incident to Dental Surgery J Oral Surg 1944;2:13.
3. Frazier CH. Subtotal Resection of Sensory Root for Relief of Major Trigeminal Neuralgia. Arch. Neurol. and Psychiat 1925;13:378.
4. Laskin DM. Oral and Maxillofacial Surgery Vol I & II CV Mosby Co. AITBS Publishers India 1992.
5. Munro D: Neurosurgery, New England J. Med. 1940;222:398.
6. Pichler H and Trauner R. Mund Und Kiefurchirurgie, Wien, 1948, Urban and Schwarzenberg,11,1.
7. Richard Bennett C. Monheim's Local Anesthesia And Pain Control in Dental Practice.7th ed. CBS Publishers and Distributors1990.
8. Thoma KH. Oral Surgery: Vol II. CV Mosby Co 1953.
9. Ullik R. Die Injecktionsbehandlung der Trigeminusneuralgie, Oestr. Ztschr. F. Stomatol 1951;48:246.

Section 4



Armamentarium

- **Syringes, Cartridges and Needles**

Chapter 12

Syringes, Cartridges and Needles

ESSENTIAL COMPONENTS

Examination Instruments

The instruments required for initial examination are shown in Figure 12.1.

The essential components of armamentarium for local anesthesia are as follows:

a. Syringe
b. Needle
c. Local anesthetic solution in the form of a cartridge or a multidose vial

Syringe

It is an instrument or a vehicle whereby the local anesthetic solution is delivered through the needle into the tissues of the patient.

Types

The various types of syringes used in dentistry are as follows (Figs 12.2 to 12.15):

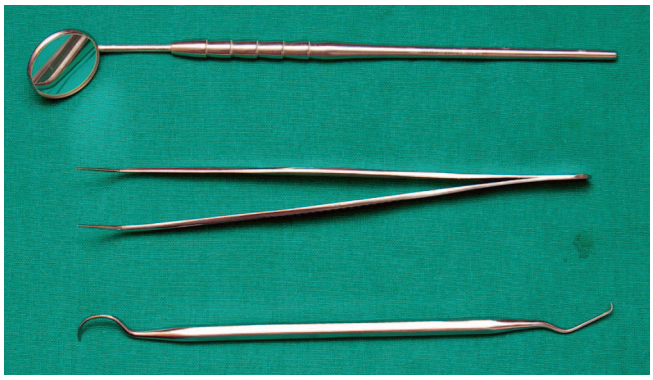


Fig. 12.1: Mouth mirror, tweezer and probe

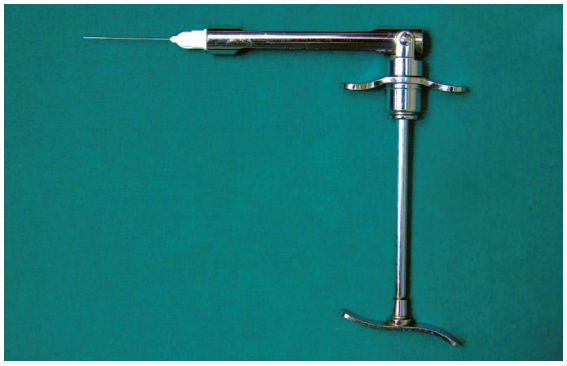


Fig. 12.2: Non-aspirating, breech loading cartridge syringe

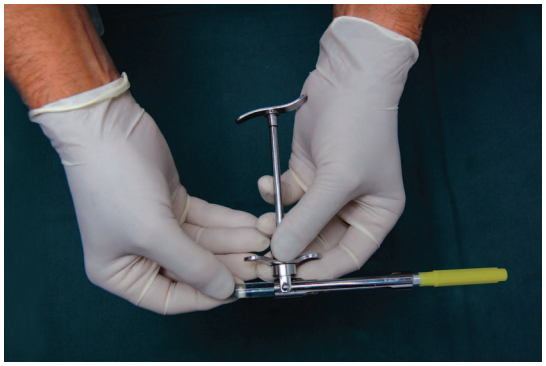


Fig. 12.3: Loading of non-aspirating, breech loading cartridge syringe

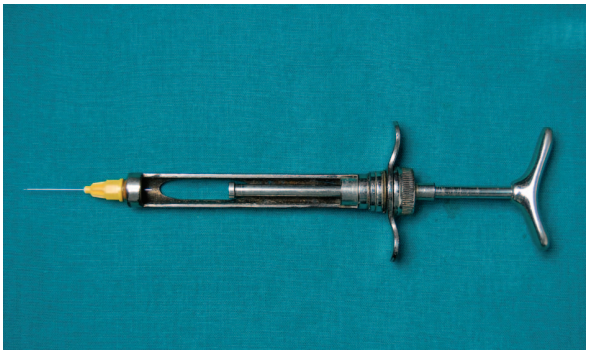


Fig.12.4: Non-aspirating side loading cartridge syringe

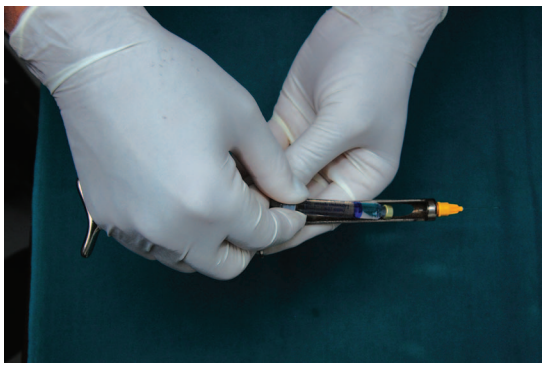


Fig. 12.5: Shows loading of the cartridge syringe

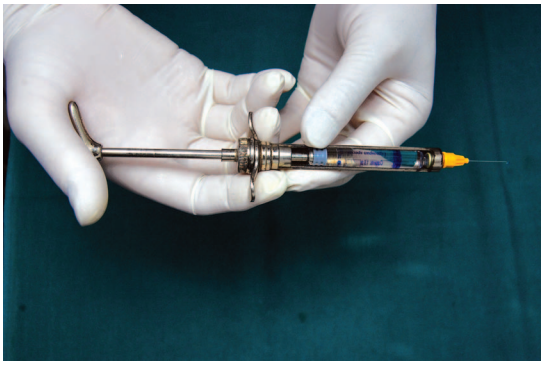


Fig.12.6: Shows loaded side-loading cartridge syringe

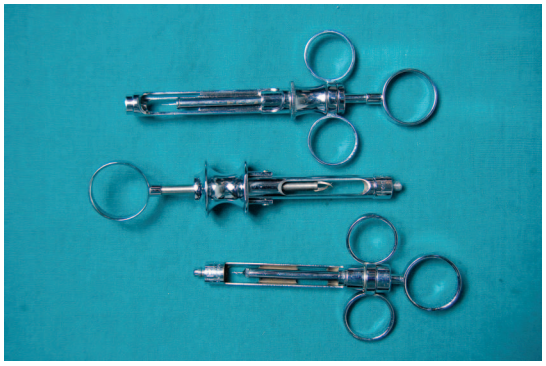


Fig. 12.7: Various types of aspirating cartridge syringes showing: A diamond, a fish-hook, and a harpoon; as seen from above downwards

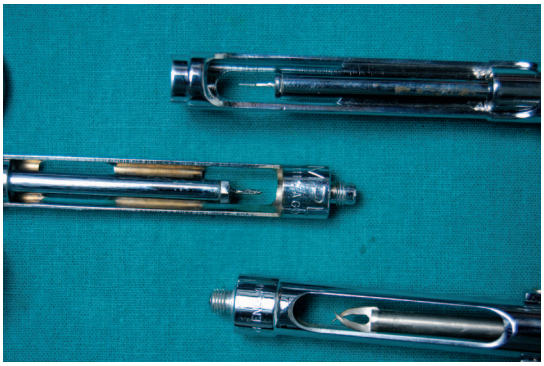


Fig. 12.8: The various types of harpoon heads with a closer view; showing a diamond, a harpoon and a fish-hook heads

Fig. 12.9: Showing the rubber diaphragm of the cartridge engaging the needle

Fig. 12.10: Showing the diamond-shaped harpoon in proximity with the rubber plunger of the cartridge

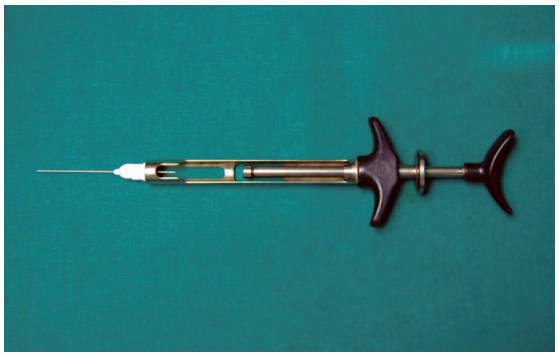


Fig. 12.11:
Self-aspirating syringe

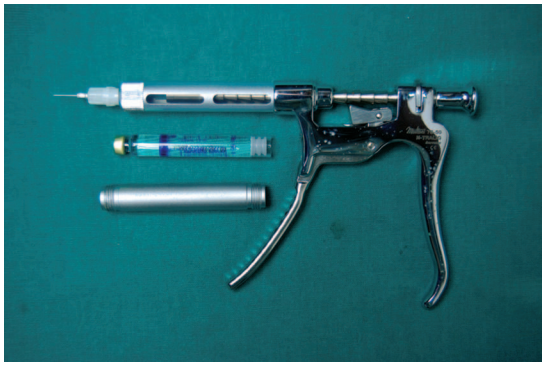


Fig. 12.12A: Specialized PDL syringe showing the syringe, barrel, cartridge and ultrafine needle. It injects 0.2 ml at a time

Fig. 12.12B: Showing the loading of the cartridge into the barrel of the specialized PDL syringe

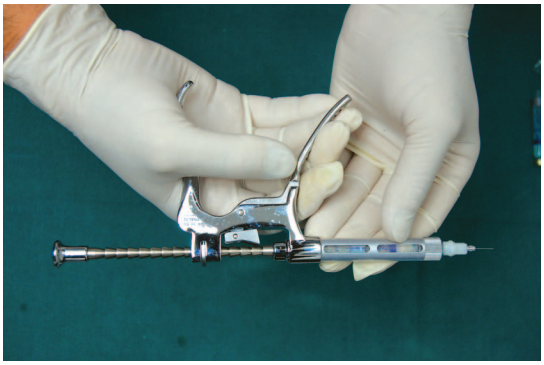


Fig. 12.12C: Mounting the barrel on the PDL syringe

Fig. 12.12D: Paroject intraligamental syringe

- Used for performing Periodontal Ligament (PDL) injections
- Compact and extremely durable
- Pen-style activator
- Injects only 0.06 ml of local anesthetic solution with each click of the activator level

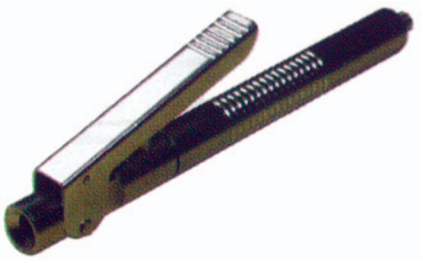


Fig. 12.13: Luer-Lock Syringes

- Plastic Luer-Lock design
- Non-reflective polypropylene barrel to reduce glare and eye fatigue

Fig. 12.14: Safety plus: self-contained, sterile injection system designed to help protect against needle-stick injuries

- It consists of a disposable barrel with attached needle and autoclavable handle and offers the user a safe method for providing single or multiple local anesthetic injections
- Easily snaps together
- Has adjustable needle sheath and auto-aspiration
- Can be sterilized in a steam or chemical autoclave or dry heat sterilizer
- Stainless steel needle is triple-beveled and silicone treated

Fig. 12.15: X-tip anesthesia delivery system

It has the following features:

- Funnel-shaped guide sleeve. It eliminates the problem of finding the hole
- Leak-proof fit of ultra-short 27-gauge needle included minimizes leakage of anesthetic solution
- Painless
- High success rate
- Profound anesthesia

I. Non-disposable (Reusable) syringes:
   1. Breech-loading, cartridge type syringe. These syringes are available in the following forms: (a) Metallic, or plastic, (b) Aspirating, or non-aspirating, and (c) Self-aspirating types.
   2. Side loading, aspirating and non-aspirating syringes.
   3. Pressure syringe.
   4. Jet injector.
   5. Luer-Lock syringes.

II. Disposable or plastic syringes.

III. Safety syringes.

Requirements of an Ideal Syringe

The requirements of an ideal syringe include the following:

1. It should be durable and be able to withstand repeated sterilization without deterioration, and it should allow repeated use.
2. The disposable syringe should be easily sterilizable and be packaged.
3. It should accept a wide variety of cartridges and needles of different manufacture.
4. It should not be expensive.
5. It should be light-weight, and easy to handle with one hand.
6. The aspirating type should have effective aspiration, and should be so designed that the blood may be easily seen in the cartridge.

Non-disposable (Reusable) Syringes

Breech-loading Cartridge Type, Aspirating Syringe

Breech-loading implies that cartridge is inserted into the syringe from one end or from the top of the barrel of the syringe.

Aspirating syringe has a device such as a tip or a harpoon, attached to the piston; which penetrates the thick rubber silicone stopper at the other end of the cartridge. When negative pressure is applied to the thumb ring by the operator, blood will enter the lumen of the needle and is seen in the

cartridge; only if the tip of the needle lies within a blood vessel, and if the needle gauge is adequate. When a positive pressure is applied to the thumb ring, it forces the local anesthetic solution into the lumen of the needle, and in turn, into patient's tissues around the area of the tip of the needle.

Breech-loading Cartridge Type, Plastic Aspirating Syringe

Recent advances in the field of plastic industry has led to the production of this plastic syringe, which is both autoclavable and chemically sterilizable.

Breech-loading Cartridge Type, Self-aspirating Syringe

Aspiration test is advisable, prior to administration of local anesthetic drugs. These syringes rely on the property of elasticity of rubber diaphragm in the cartridge to obtain the required negative pressure for aspiration. The self-aspirating syringes allow multiple aspirations to be performed easily throughout the period of deposition of local anesthetic solution.

Non-disposable Syringe (Metallic and Plastic) (Aspirating)

a. Advantages:
   i. Aspiration is possible with one hand
   ii. Autoclavable
   iii. Long-lasting with proper maintenance
   iv. Lower cost in the long run.
b. Disadvantages:
   i. Metallic syringes are heavier than plastic disposable syringes
   ii. Possibility of infection with improper care
   iii. Deterioration of plastic with repeated autoclaving.

Pressure Syringes

Used for intraligamentary injection, and also for pulpal anesthesia of one isolated tooth in mandibular arch.
Two types: (1) 1st generation: Pistol-grip type, and of larger size (Figs 12.12A to C) and (2) 2nd generation, of pen-grip type (Fig. 12.12D).

Jet Injector

It was introduced by Figge and Scherer in 1947, in the form of a jet or needleless injection. Margetis et al, (1958) for the first time, reported the use of jet injections in dentistry.

- *Principle:* Liquids forced through very small openings called jets, at very high pressure can penetrate skin or mucous membrane.
- *Calibration:* The syringe is calibrated to deliver 0.05 to 2.0 ml of solution at 2000 psi.
- *Primary use:* Topical anesthesia prior to insertion of a needle.
- *Secondary use:* May be used to obtain mucosal anesthesia of the palate. Regional nerve blocks or paraperiosteal injections are still required for complete anesthesia.

a. Advantages:
   i. Does not require use of a needle. Hence, recommended for needle-phobics and especially for young children.
   ii. Deposits very small volumes of local anesthetic solution with calculated amount of force.
   iii. Used in topical anesthesia.
   iv. Complete intraligamentary anesthesia can be achieved for individual tooth.
b. Disadvantages:
   i. Inadequate for pulpal or regional anesthesia.
   ii. Some patients are disturbed by the "jolt" of jet injection, and it upsets them.
   iii. Expensive.

Disposable/Plastic Syringes

Disposable/Plastic syringes are available in variety of sizes and with an assortment of needle gauges. Most often used for IM or IV drug administration, but may also be used for intraoral injections.

a. Advantages:
   i. Disposable; meant for single use.
   ii. Sterile until opened.
   iii. Light-weight, better tactile sensation.
   iv. Decreased chances of transmitting infections.
b. Disadvantages:
   i. Does not accept prefilled cartridges.
   ii. Difficult aspiration with one hand, requiring two hands
   iii. Increased cost in the long run.

Safety Syringes

These syringes minimise risk of accidental needle-stick injuries.

a. Advantages:
   i. Disposable; meant for single use.
   ii. Sterile until opened.
   iii. Light-weight, better tactile sensation.
   iv. Decreased chances of transmitting infections.
b. Disadvantages: More expensive than reusable syringes.

Needles

The needles permit the local anesthetic solution to travel from the dental cartridge into the soft tissues surrounding the tip of the needle. In India, the needles used for regional analgesia in dental surgery, range from 24, 25, 27 and 30-gauges; and from 25 mm (1") to 38-40 mm (1½ or 1⅝") in length.

Types

Needles in dental practice are made up of stainless steel, platinum, and iridium-platinum or ruthenium alloys. The stainless steel needle is most commonly used and is highly recommended. Needles currently available are usually pre-sterilized and disposable (Figs 12.16 to 12.18).

Advantages

The advantages of this needle are as follows:

i. *Rigidity:* It is rigid and hence can be easily guided into the tissues.
ii. *Sharpness:* It maintains the sharpness of the point.
iii. *Cost:* It is not expensive and therefore can be discarded after using for each patient.
iv. *Breakage:* It rarely occurs, if these are handled properly.
v. *Availability:* It is available in variety of lengths and gauges.
vi. *Sterilization:* It withstands boiling and autoclaving without corrosion and becoming weak.

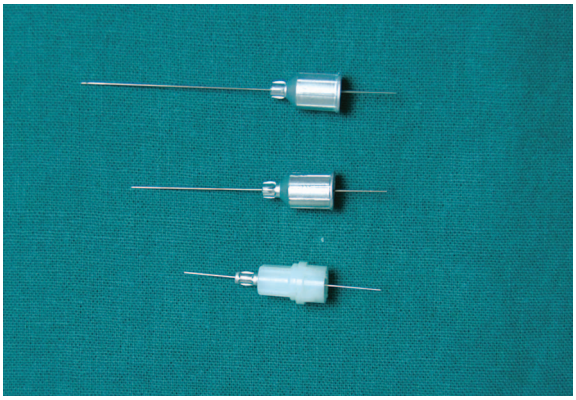


Fig. 12.16A: Disposable needles: Long (38-40 mm), short (25 mm), and extrashort and ultrafine

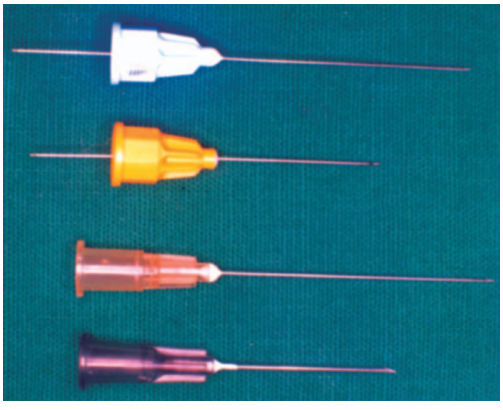


Fig. 12.16B: Disposable needles:
i. Long needle for cartridge syringe (27 G, 0.4 × 35 mm),
ii. Short needle for cartridge syringe (27 G, 0.4 × 23 mm,
iii. Long needle for disposable hypodermic syringe (26 G, 0.45 × 38 mm),
iv. Short needle for disposable hypodermic syringe (21 G, 0.8 × 25 mm)

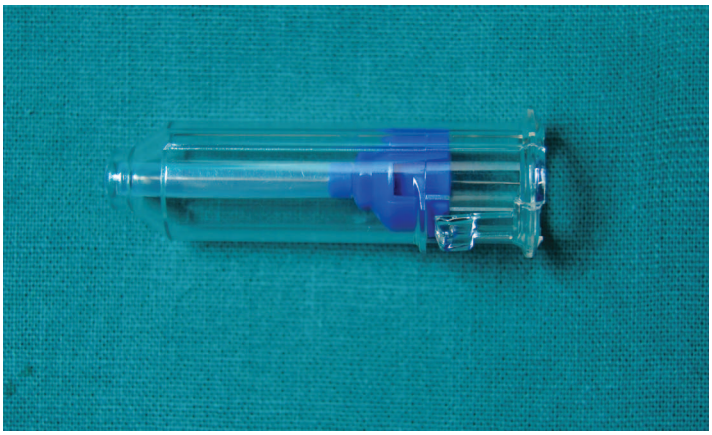


Fig. 12.17: Safety cover for the needle

Parts

The needles used for administration of local anesthetic solutions have the following components; (a) bevel, (b) shaft or shank, (c) hub, (d) syringe adaptor, and (e) cartridge-penetrating end.

a. *Bevel:* It defines the point or the tip of the needle. The bevels, as described by manufacturers are: (i) long, (ii) medium, and (iii) short.

   The recommended bevel is 12° and it influences the degree of deflection. The greater the angle of the bevel with the long axis of the needle; the greater will be the deflection as needle is passed through the soft tissues (Aldous, 1977; Jeske and Boshart, 1985; and Robison et al, 1984). Bennett advised that the short-beveled needle is superior to the long tapering bevel for regional analgesia, such as block anesthesia as it is less likely to be deflected from its intended path during insertion. Needles with point centered on the long axis, deflect less than beveled-point needles whose point is eccentric.

b. *Shank/Shaft:* The length of shank is measured from the hub to the point of the bevel.

c. *Hub:* It is a plastic or metal piece through which the needle is attached to the syringe.

   The interior surface of plastic syringe adaptor is not prethreaded. Therefore, to attach a plastic hubbed needle to a syringe, the needle must be pushed onto syringe while being screwed on. Metallic-hubbed needles are usually prethreaded.

d. *Syringe adaptor:* It is adapted to the needle adaptor end of the syringe.

e. *Cartridge-penetrating end:* It is placed into the needle adaptor of syringe and perforates the rubber diaphragm of glass cartridge. Its tip rests within the cartridge.

Selection of Needles

When needles are selected for use in various injection techniques the following two factors are considered:

Gauge

It refers to the internal diameter; or the diameter of the lumen of the shank of the needle. The gauge of the needles is indicated by number, with higher gauge number denoting the smaller diameter of the shaft. These are represented as follows: The greater the internal diameter of the needle, the smaller is the number (Table 12.1).

Table 12.1: Showing relation of the gauge and the diameter of the needles

Gauge	Diameter in mm
20	0.81
21	0.72
22	0.64
23	0.57
24	0.51
25	0.45
27	0.40
30	0.30

Disposable single use needles are of 24, 25, 27, and 30-gauge. 25-gauge needles have less deflection and greater accuracy leading to increased success of anesthesia. Also aspiration of blood is easier and more reliable through a larger lumen.

Important Features

The important features of these needles are as follows:

i. *Bevel indicator:* Needles are imprinted with a red dot that indicates the position of the bevel of the needle when in the soft tissues, or the gingival line.
ii. *Siliconized cannula:* The cannula of the needle is siliconized to ensure smooth insertion and almost painless penetration.
iii. *Pre-threaded plastic hub:* The plastic hub is meant for use with standard dental cartridges. The hub offers easy assembly and final disposal.
iv. *Triple-beveled tip:* The tip penetrates the diaphragm effortlessly.

Examples: Accuject, Monoject, Septoject

Bennett advised the use of 25 or 27-gauge needle for deeper penetration of the tissues. The advantages of this gauge are as follows:

i. It is rigid enough to be guided to the target area without deflection. Less deflection offers greater accuracy leading to increased success of anesthesia.

ii. The finer gauge permits easier penetration of the tissues and the discomfort to the patient.
iii. It is less likely to penetrate smaller blood vessels.
iv. Aspiration is possible with 25-gauge.
v. Breakage is less likely to occur with 25-gauge.

Length

Dental needles are available in two lengths: (i) Short–25 mm or 1"; and (ii) Long–38-40 mm or 1 ½". Short needles are recommended for paraperiosteal injections in entire maxilla; and anterior mandible. Long needles are preferred for all injection techniques requiring penetration of significant thickness of soft tissues. For example, Pterygomandibular Block, Vazirani-Akinosi Mandibular Nerve Block, Gow-Gate's Mandibular Nerve Block.

Advantages:

Larger gauge needles have distinct advantages over smaller gauge needles:
1. *Less deflection:* It occurs as needle passes through tissues. This leads to greater accuracy and higher success rates, especially in those techniques in which there is significant soft tissue depth, as in Pterygomandibular Block, Gow-Gate's Mandibular Nerve Block, Akinosi Mandibular Nerve Block and Infraorbital Nerve Blocks.
2. *Needle breakage:* It is not common with disposable needles, and is less likely to occur with larger gauge needles.
3. *Aspiration:* It is easier and more reliable through larger lumen.

Topical Local Anesthetic Agents

Prior to the administration of injectable local anesthetic solutions, it is desirable to use the local anesthetic spray or ointment to minimize the discomfort due to injection. The topical agents are available in the form of spray and ointment of various local anesthetic agents in different strengths (Fig. 12.18).

Fig. 12.18: Topical local anesthetic agents: Lidocaine spray and ointment

MUTIDOSE VIALS

Local anesthetic solutions are available in the form of (a) multidose vials and (b) cartridges. Local anesthetic solutions are available in multidose vials of 30 ml in different potencies of local anesthetic agents, i.e. 0.5%, 1%, 2%, 3%, and 4%, etc. In India, the local anesthetic solutions are further available of different dilutions of vasoconstrictor, i.e. 1:80 000, 1:100 000 and 1:200 000 (Figs 12.19 to 12.22).

DENTAL CARTRIDGE

It is a glass tube sealed at one end by a rubber stopper (plunger); while the other end is sealed by an aluminium cap over the rubber diaphragm. The rubber stopper is forced into the tube by the plunger of the cartridge syringe, while the rubber diaphragm is punctured by the cartridge end of the needle. It is available in the form of a presterilised glass cylinder.

Capacity

The capacity of cartridges in India is 2 ml, while that in USA and France is 1.8 ml, and that in UK and Australia is 2.2 ml.

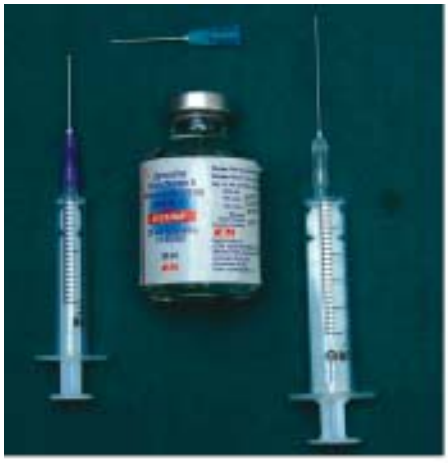


Fig. 12.19: Injectable local anesthetic agent in a multidose vial, along with 2 ml and 5 ml disposable non-leurlock hypodermic syringes with short and long disposable needles. The additional needle is for aspirating the solution from the multidose vial

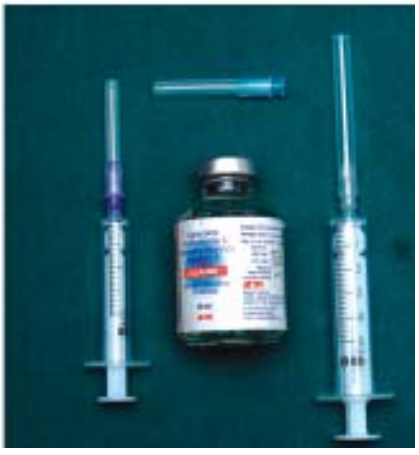


Fig. 12. 20: Injectable local anesthetic agent in a multidose vial along with 2 ml and 5 ml disposable non-leurlock hypodermic syringes with short and long disposable needles (Needles are shown without their sheaths)

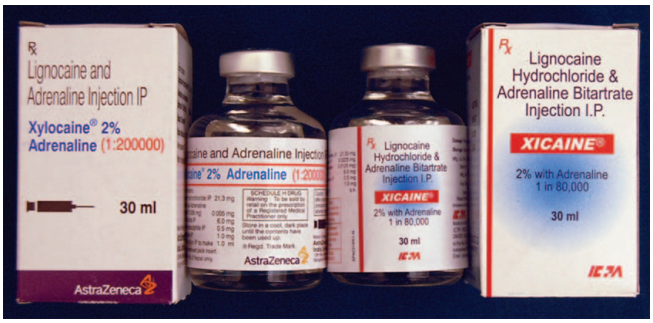


Fig. 12.21: Local anesthetic agents in the form of multidose vials containing 2% lignocaine with 1:80 000 and 1:200 000 adrenaline

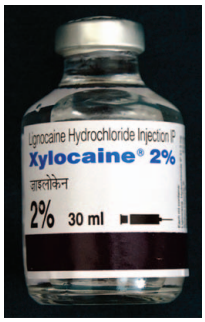


Fig. 12.22: Local anesthetic agent in the form of multi-dose vial containing 2% lignocaine without adrenaline

Care and Handling

Availability (Figs 12.23 to 12.30):

The cartridges are available in various forms of packages:

i. Vacuum sealed,
ii. Blister-packing: The pack remains clean and uncontaminated.

Glass dental cartridges should not be autoclaved. The seal on the cartridge cannot withstand extremes of temperature of autoclaving; and the heat labile vasoconstrictors will be destroyed.

Storage

The local anesthetic cartridge should be stored as aseptically as possible. These should be stored dry in their original container, and covered with a lid all the time; or in another suitable sterile container, that is kept covered all the times, preferably at room temperature (70°F), and in dark place. Local anesthetic dental cartridges should not be left exposed to direct sunlight, because some contents may undergo accelerated deterioration. Cartridge warmers are not necessary. Local anesthetic agents in dental cartridge, maintained at room temperature do not cause any discomfort on injection into the tissues, nor do patients complain of solutions being too cold. On the contrary, local anesthetic solutions at 80°F or above have a much greater incidence of being too hot or burning on injection.

There is no need to prepare a cartridge before use. If necessary apply an alcohol wipe moistened with undiluted 91% isopropyl alcohol or 70% ethyl alcohol to the rubber diaphragm.

Cartridge should not be permitted to soak either in alcohol or other cold-sterilising solutions, because the permeable rubber plunger will allow diffusion of these solutions into dental cartridge. This leads to

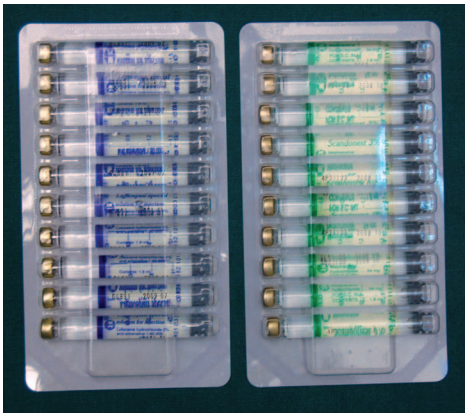
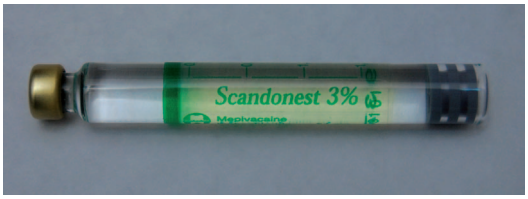


Fig. 12.23: Local anesthetic cartridges in blister packing containing 2% lignocaine with 1:80000 adrenaline (blue colored) and 3% mepivacaine without adrenaline (green colored).

Fig. 12.24: Local anesthetic cartridge containing 3% mepivacaine without adrenaline



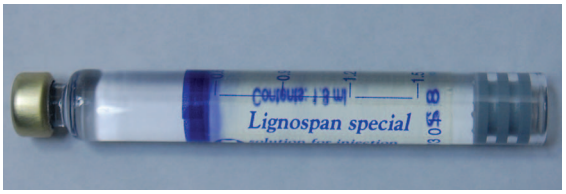
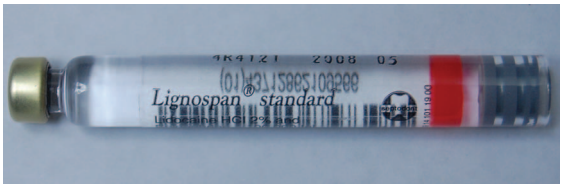


Fig. 12.25: Local anesthetic cartridge containing 2% lignocaine with 1:80000 adrenaline

Fig. 12.26: Local anesthetic cartridge containing 2% lignocaine without adrenaline



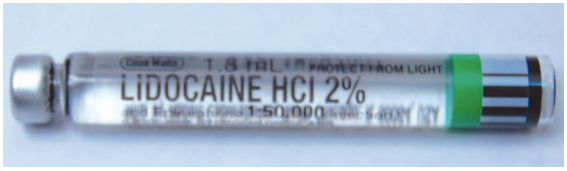
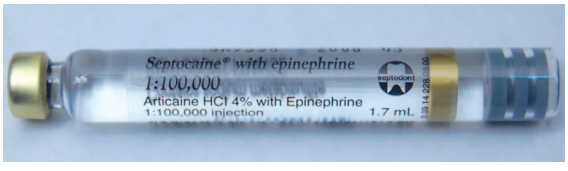


Fig. 12.27: Local anesthetic cartridge containing 2% lignocaine with 1:50 000 adrenaline

Fig. 12.28: Local anesthetic cartridge containing 4% articaine with 1:100 000 epinephrine



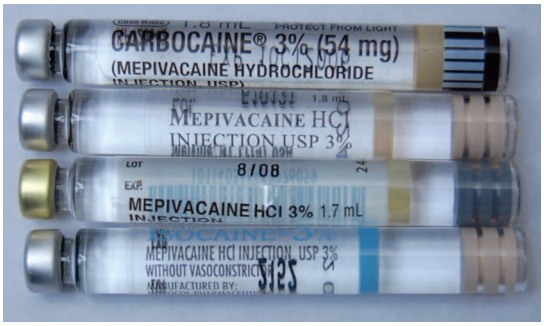


Fig. 12.29: Various local anesthetic cartridges containing 3% mepivacaine without adrenaline

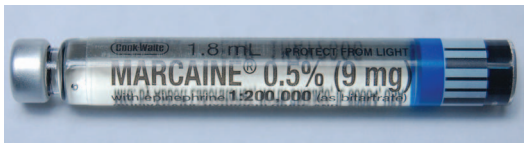


Fig. 12.30: Local anesthetic cartridge containing 0.5% bupivacaine with 1:200 000 epinephrine

contamination of the local anesthetic solution resulting in post-injection pain, edema and trismus.

Components

The prefilled dental cartridge consists of four parts: (i) cylindrical glass tube, (ii) rubber stopper (plunger), (iii) aluminium cap, and (iv) rubber diaphragm.

1. Cylindrical Glass Tube

A small bubble of approximately 1-2 mm is frequently seen in the cartridge. It is composed of nitrogen gas, which is bubbled into the local anesthetic solution during its manufacture to prevent oxygen from being trapped in the cartridge and potentially destroying the vasopressor or vasoconstrictor.

2. Rubber Stopper

It is located at the end of cartridge that receives the harpoon of syringe. The harpoon is embedded in plunger by gentle finger pressure on thumb ring of syringe. The rubber plunger occupies little more than 0.2 ml of volume of entire cartridge. In a normal intact dental cartridge, the rubber plunger is slightly indented from lip of the glass cylinder. Cartridges that contain plungers, which are equal with or extruded beyond the glass of the cylinder should not be used.

3. Aluminium Cap

It is located at the opposite end of cartridge from rubber plunger. It fits snugly around the neck of glass cartridge holding thin rubber diaphragm in position. It is silver colored in all cartridges.

Fig. 12.31: Cartridge dispenser and warmer

4. Rubber Diaphragm

It is a permeable membrane through which the cartridge end of needle penetrates. The permeability of diaphragm allows solution in which dental cartridge is stored to diffuse into cartridge, contaminating the local anesthetic solution.

ADDITIONAL ARMAMENTARIUM

1. Topical antiseptic: Such as 5% Betadine, Povidone-iodine (Win-Medicare) (Wockhardt).
2. Topical anesthetic: Such as 2% Lidocaine jelly, or 5% Benzocaine ointment (Mucopain).
3. Applicator sticks.
4. Cotton/gauze.
5. Hemostat: To remove needle from the soft tissue in the event of needle breakage.
6. Small stainless steel bowls.
7. Cartridge dispenser and warmer (Fig.12.31).

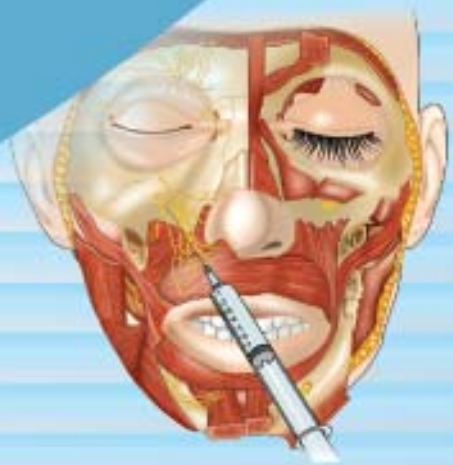
BIBLIOGRAPHY

1. Albers DD, Ellinger RF. Histologic effects of high-pressure intraligamental injections on the periodontal ligament, Quint Int 1988;19:361-63.
2. Fuhs QM, Walker WA, Gouigh RW, Schindler WB, Hartman KS. The periodontal ligament injection: Histological effects on the periodontium in dogs, J Endodont 1983;9:411-15.
3. Galili D, Kaufman E, Garfunkel AA, Michaeli Y. Intraligamentary anesthesia: A histological study, Int J Oral Surg 1984;12:511-16.
4. Hochman, MN, Friedman MJ. In vitro study of needle deflection: A linear insertion technique versus a bi-directional rotation insertion technique. Quint Int 2000;31:737-43.

5. Hochman, MN, Friedman MJ. In vitro study of needle force penetration comparing a standard linear insertion to the new bi-directional rotation insertion technique. Quint Int 2001;32:789-96.
6. Margetis PM, Quarantillo EP, Lindberg RB. Jet injection local anesthesia in dentistry: A case report of 66 cases, US Armed Forces Med J 1958;9:625-34.
7. Meechan JG. A comparison of three different automatic aspirating dental cartridge syringes, J Dent 1988;16:40-43.
8. Peterson JK. Efficacy of a self-aspirating syringe. Int J Oral Maxillofac Surg 1987;16:241-44.

Section

5



Practice of Local Anesthesia

- Medical Evaluation
- Infection Control
- Preanesthetic Medications
- Basic Techniques of Local Anesthesia
- Injection Techniques for Maxillary Nerve and its Branches
- Injection Techniques for Mandibular Nerve and its Branches
- Newer Injection Techniques
- Management of Dental Clinic Waste

Prior to commencement of any dental procedure or administration of any drugs, particularly, local anesthetic agents, the dental practitioner should determine whether the patient can tolerate the procedure or the drug safely or not. Specific modifications in the treatment and its implementation are necessary to decrease the risk associated with the procedure or the drug. This is important, as the local anesthetic agents, like many other drugs, exert actions on many systems of the body.

Local anesthetic agents undergo biotransformation in liver (amides) or blood (esters). Hence, the functional status of these systems should be determined before the administration of the drug. Further, the local anesthetic agents are excreted through kidneys. Hence, status of kidneys should also be evaluated.

DETAILED MEDICAL HISTORY

Medical Evaluation

A brief review of medical evaluation of the patient is advised, which is as follows:

The dental practitioner should get detailed information about the current status of medical history at the first visit and record it. Medicolegally, it is of great importance.

i. History of major illness or been under treatment in the past. These questions give you information about patient's general health.
ii. The drugs the patient is taking presently, and medications taken during the past 2 years.
iii. Is the patient taking any antiplatelet or anticoagulant medicines?
iv. The medical status should be updated regularly in the records.

History of Hospitalization, if Any

Even maternity history should be taken in case of female patients.

Fear and Anxiety or Nervousness

The dental practitioner can determine whether any additional steps such as sedation, psychosedation, stress reduction protocol or anxiety reduction protocol are required to aid in achieving pain control.

History of Allergy to Drugs

a. Drugs such as Penicillin, Sulpha group of drugs, Aspirin or Codein.
b. The incidence of true, documented, and reproducible allergy to amide local anesthetic agents is virtually rare (Sindel and deShazo, 1991; and Haas, 2002). Determine the name of the drug(s) the patient is allergic to, and the exact nature of adverse reaction that developed.
c. There could be history of allergy to local anesthetic agents, which could be due to any of the constituents of the local anesthetic solution. There are two types of preservatives used in a local anesthetic solution:
   i. Preservative to vasopressor such as sodium bi-sulfite or Na meta-bi-sulfite, or
   ii. General preservatives, such as methylparaben, chlorbutol, etc.

In such cases, the solutions containing these constituents should be avoided.

History of Excessive Bleeding that Required Special Treatment

Patients with the following bleeding disorders are at risk of bleeding:
1. Coagulation disorders
2. Bleeding disorders
3. Patients taking antiplatelet or anticoagulant drugs.

The insertion of an injection needle in the deeper soft tissues of the oral cavity which are highly vascular in nature, the patient can be at risk of severe bleeding which could be difficult to manage.

In such cases, deeper injections techniques which have a greater incidence of positive aspiration should be avoided such as: (i) Pterygomandibular block, (ii) Posterior Superior Alveolar Nerve Block (Tuberosity block), (iii) Maxillary Nerve Block (High tuberosity block), (iv) Gow-Gates Mandibular Nerve Block, and (v) Akinosi Technique, etc.

The techniques such as supraperiosteal, and periodontal ligament injection, etc. should be employed.

PAST DENTAL HISTORY

If there is a history of past bad experience in the dental office, it may require use of additional procedure such as psychosedation.

Interactions between local anesthetic agents and other drugs are less; however, these reactions can occur. It is important to include them in the medical history chart.

MAJOR ILLNESSES

1. Cardiovascular diseases
2. Cerebrovascular accident / stroke / Transient ischemic attack
3. Renal diseases such as chronic renal failure (CRF)
4. Endocrinological diseases such as diabetes mellitus and thyroid dysfunction
5. Respiratory diseases such as bronchial asthma
6. Neurological disorders such as epilepsy
7. Patients with liver diseases HAV, HBV, HCV, infective hepatitis
8. Patients on psychiatric medications
9. Hematological disorders such as anemia and hemophilia
10. Pregnancy
11. AIDS.

Cardiovascular Diseases

The patients might be having the following cardiovascular diseases, which demand modification of treatment strategy:

a. Ischemic heart disease / Coronary artery disease,
b. Congestive cardiac failure,
c. Hypertension,
d. Congenital heart disease (CHD),
e. Rheumatic heart disease (RHD) / Valvular heart diseases / Prosthetic heart valves,
f. Cardiac dysrhythmias,
g. Implanted heart devices: Such as—
   i. Pacemakers and ii. Stents
h. Coronary artery bypass grafting (CABG).

(a) Ischemic Heart Disease (IHD) / Coronary Artery Disease (CAD)

These are (i) Angina pectoris and (ii) Acute myocardial infarction

Angina Pectoris

Angina pectoris is defined as an attack of transient chest pain resulting from acute myocardial infarction, which is usually relieved by rest or administration of a vasodilator.

There are 2 types of Angina pectoris:

i. Stable angina or angina on exertion. Factors which increase myocardial O_2 requirement such as anxiety, stress, or inadequate pain control may precipitate an anginal episode.
ii. Unstable angina (pre-infarction stage) has a high-risk (Gottlieb and Flaherty, 1991).

Acute Myocardial Infarction (Heart attack)

Patients with a history of a recent attack (within or < 6 months) or repeated myocardial infarction, have increased risk during dental care or even after the administration of local anesthetic drugs. Such patients should not receive any elective dental care within 6 months of an acute myocardial infarction. There is a risk of occurring of re-infarction (Tarhan and Giuliani, 1974; Weinblatt et al, 1968). After this period of recuperation, most cases of myocardial infarction are treatable with appropriate therapy modification in consultation with the treating cardiologist.

(b) Congestive Cardiac Failure (CCF)

The degree of heart failure has to be assessed. CCF patients who are unable to complete normal functions without disability; or who have disability (fatigue, shortness of breath) at rest are likely to demonstrate some degree of decreased liver perfusion, leading to an increase in the half-life of amide local anesthetic agent (Thompson et al, 1973).

Patients with cardiac failure are less tolerant to stress; having a decreased functional reserve. Hence, anxiety must be dealt with adequately. Psychosedation is appropriate, however, inhalation sadation is preferred. Patients are not comfortable in supine position, because of orthopnea. Patients are more comfortable in upright position.

(c) Hypertension

The 7th Report of Guidelines given by The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure are recommended for reading and management of a hypertensive patient (Chobanian et al, 2003) (The JNC 7 Report-2003), as shown in Table 13.1.

Table 13.1. Classification of Blood Pressure based on Joint National Committee Report

Classification	SBP mm Hg	DBP mm Hg
Normal	< 120	< 80
Pre-hypertension	120-139	80-89
Stage I hypertension	140-159	90-99
Stage II hypertension	> 160	> 100
Hypertensive crisis	> 220	> 120

Any patient beyond the age of 45-50 years, should be asked to get his / her BP checked. If he/she happens to be known hypertensive; then local anesthetic agent injection should be given according to the advice of the treating physician.

Patients with mild to moderate elevations in Systolic BP and Diastolic BP are acceptable risks for dental care; including use of local anesthetic agents with vasoconstrictors having higher dilution, or avoided, if recommended by the treating cardiologist or the physician.

All hypertensive patients should follow compliance. They should make it a point to take morning dose of medications. Their BP should be checked at each appointment and be managed on the basis of the most recent reading.

(d) Congenital Heart Disease, (e) Rheumatic Heart Disease (RHD) / Valvular Heart Diseases / Prosthetic Heart Valves, (f) Cardiac Dysrhythmias, etc. (Gottlieb and Flaherty, 1991)

Patients who fall into these categories require antibiotic prophylaxis. Patients with mitral valve prolapse are also included. The current antibiotic prophylaxis regimen guidelines as advised by the American Heart Association are recommended for reading and are mentioned in the Guidelines for Antibiotic Prophylaxis (Dajani et al, 1997).

Artificial Heart Valve

Patients with a prosthetic heart valve require antibiotic prophylaxis before any dental care. Consultation with the patient's treating cardiologist is required (Dajani et al, 1997). These patients are usually taking anti-coagulant drugs. Due consideration should be given to stop these drugs a few days prior to surgical procedure in consultation with the patients treating cardiologist.

The administration of local anesthetic agent does not, by itself, require antibiotic prophylaxis. The one exception to this is the periodontal ligament injection (Quilici, 1990). The nature of the subsequent dental treatment would determine whether or not antibiotic prophylaxis is recommended.

Guidelines for Antibiotic Prophylaxis

The American Heart Association (AHA) and the American Dental Association (ADA), recently modified their recommended protocols for antibiotic prophylaxis against bacterial endocarditis (Dajani et al, 1997) (Table 13.2 to 13.4).

Table 13.2. Cardiac conditions associated with endocarditis

Endocarditis Prophylaxis Recommended

High-risk category

- Prosthetic cardiac valves, including bioprosthetic and homograft valves
- Previous bacterial endocarditis
- Complex cyanotic Congenital Heart Disease (CHD) (single ventricle states, transposition of the great arteries, tetralogy of Fallot)
- Surgically constructed systemic pulmonary shunts or conduits

Moderate risk category

- Most other congenital cardiac malformations (other than mentioned above and below)
- Acquired valvular dysfunction, e.g. Rheumatic Heart Disease (RHD)
- Hypertrophic cardiomyopathy
- Mitral valve prolapse (MVP) with valvular regurgitation or thickened leafltes

Endocarditis prophylaxis not recommended negligible risk category

- Isolated secundum atrial septal defect (ASD)
- Surgical repair of ASD, ventricular septal defect (VSD), or Patent Ductus Arteriosus (PDA)
- Previous coronary artery bypass graft surgery
- Mitral Valve Prolapse (MVP) without valvular regurgitation
- Physiologic, functional or innocent heart murmurs
- Previous Kawasaki disease without valvular dysfunction
- Previous rheumatic fever without valvular dysfunction
- Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators

Table 13.3. Dental procedures and endocarditis prophylaxis (Dajani et al, 1997)

A. Endocarditis prophylaxis recommended

- Dental extractions
- Periodontal procedures including surgery, scaling and root planing, probing and recall maintenance
- Dental implant placement and reimplantation of avulsed teeth
- Endodontic (root canal) instrumentation or surgery only beyond the apex
- Subgingival placement of antibiotic fibers or strips
- Initial placement of orthodontic bands but not brackets
- Intraligamentary local anesthetic injections
- Prophylactic cleaning of teeth or implants where bleeding is anticipated

B. Endocarditis prophylaxis not recommended

- Restorative dentistry (operative and prosthodontic) with or without retraction cord
- Local anesthetic injections (non-intraligamentary)
- Intracanal endodontic treatment; post-placement and buildup
- Placement of rubber dams
- Postoperative suture removal
- Placement of removable prosthodontic or orthodontic appliances
- Taking of oral impressions
- Fluoride treatments
- Taking of oral radiographs
- Orthodontic appliance adjustment
- Shedding of primary teeth

Table 13.4. Prophylactic regimens for dental, and oral procedures

Situation	Antibiotic	Regimen
Standard general prophylaxis	Amoxycillin	Adults, 2.0 g; Children, 50 mg/kg orally one hour before procedure
Unable to take oral medications	Ampicillin	Adults, 2.0 g IM/IV; Children, 50 mg/kg IM/IV within 30 minutes before procedure
Allergic to penicillin	Clindamycin, or	Adults, 600 mg; Children, 20 mg/kg orally one hour before procedure
	Cephalexin, or Cefadroxil, or Azithromycin or	Adults, 2.0 g; Children, 50 mg/kg orally one hour before procedure
	Clarithromycin	Adults, 500 mg; Children, 15 mg/kg orally one hour before procedure
Allergic to penicillin and unable to take oral medications	Clindamycin, or	Adults, 600 mg; Children, 20 mg/kg IV one hour before procedure
	Cefazolin	Adults, 1.0 g; Children 25 mg/kg IM/IV within 30 minutes before procedure

(g) Implanted Heart Devices (Pacemaker and Defibrillators)

Heart Pacemakers

Significant disturbance with the cardiac rhythm may necessitate the insertion of a pacemaker. Most of the currently used pacemakers are of the demand type (functioning only when needed), and these patients usually do not require antibiotic prophylaxis. Local anesthetic agents with vasoconstrictors may be safely given to these patients.

Defibrillators

Patients with significant atrial and ventricular dysrhythmias receive implanted defibrillators. These devices can sense the onset of potentially life-threatening irregularities in the rhythm of heart and deliver either a synchronized shock, to defibrillate the heart. These are of the size of a deck of playing cards, and are implanted under the skin on the left side of the chest. Leads are placed onto the heart to monitor its rhythm and deliver a shock, if needed. These devices are not located within the heart, hence, there is no need for antibiotic prophylaxis. However, it is advisable to take consultation with patient's cardiologist.

(h) Coronary Artery Bypass Grafting (CABG)

Patients who have undergone Coronary Artery Bypass Grafting; the nature of surgical procedure, the degree of any current disability and whether there is a need for antibiotic prophylaxis, should be determined. In most cases, patients with these conditions may safely receive dental care, including administration of local anesthetic agent with vasoconstrictors with little or no increase in risk. Physician's consultation is mandatory.

Cerebrovascular Accident/Stroke/Transient Ischemic Attack

This condition requires modification of treatment to decrease the risk. The guidelines are as follows: (i) monitor BP, (ii) use minimum effective dose of local anesthetic agent, and (iii) avoid intravascular administration of vasoconstrictors. These patients are considered as ASA II or III risk within 6 months or more after the acute attack.

Renal Diseases (Chronic Renal Failure) (CRF)

A small percentage (<10%) of local anesthetic agent is excreted unchanged by the kidneys in the urine. Patients with Chronic Renal Failure, theoretically, achieve high levels of local anesthetic agent in the blood, thereby increasing the risk of local anesthetic overdose. However, practically, the minimum doses of local anesthetic agent used, do not pose any increase in the risk, however, adrenaline is contraindicated.

Endocrinological Diseases

Diabetes Mellitus and Thyroid Dysfunctions

Diabetes Mellitus

Patients with diabetes mellitus are referred to their treating physicians, and the blood sugar is brought under control. Subsequently the surgical procedure is carried our under antibiotic cover.

Thyroid Dysfunctions

The signs and symptoms of hyperthyroidism include: Sensitivity to heat, easy sweating, tachycardia, palpitation, weight loss, increased body temperature, tremor of extremities, increased nervousness, etc. Patients with hyperthyroidism are sensitive to catecholamines, and may demonstrate an exaggerated response to vasopressors included in local anesthetic solution. These reactions can be prevented or minimized by adherence to the guidelines by using minimum concentration or higher dilution of vasoconstrictors, or avoid them in case of history of above symptoms.

Patients with corrected or medication-controlled hyperthyroid or hypothyroid conditions are termed as euthyroid and these conditions respond normally to catecholamines. Local anesthetic agents containing vasoconstrictors can be used, only if approved by the treating physician.

Respiratory Diseases

Patients with respiratory diseases such as bronchial asthma, are treated with stress reduction protocol. They should use the inhaler on the day of surgery, and also keep with them during the dental or surgical procedure. The medications for bronchodilatation should be kept handy, in case of an emergency. If the patient has taken corticosteroids earlier, it is necessary to consult the treating physician.

Neurological Disorders

Epilepsy/Seizures

The various causes of seizures in the dental office, are as follows:

(i) Stress, (ii) Hypoglycemia, and (iii) Hyperventilation

Stress: It may provoke seizures even in patients with well-controlled epilepsy.

Stress reduction protocol should be employed to minimize the risk of seizure development during treatment.

Severe overdose reaction due to local anesthetic agents manifest clinically as generalized tonic-clonic convulsions. However, the administration of local anesthetic agents is not contraindicated in seizure-prone individuals. Indeed, carefully administered IV local anesthetic agents may be used as anticonvulsants in a patient during grand-mal seizure (DeToledo, 2000).

Patients with Liver Diseases

HAV, HBV, HCV, and Infective Hepatitis

There is an increased risk of infection (HAV, HBV, and HCV), via blood, or saliva and an increased risk of liver dysfunction (HAV, HBV, liver disease, infective hepatitis. Thorough evaluation of the disease should be carried out to assess the degree of risk to both the practitioner and to the patient, before commencing the treatment. In significant liver dysfunction, the half-life of amide local anesthetic agents may be significantly prolonged, thereby increasing the risk of overdose. The local anesthetic agents are detoxified in liver, and hence, should only be used if approved by the treating physician.

Patients with Psychiatric Ailments and on Medications

Patients under psychiatric care, for Neurosis, Psychosis, and Depression, usually take psychiatric drugs. These drugs alter the behavior patterns. There are 2 types of psychotic drugs frequently used: (1) TC Antidepressants, and (2) MAO Inhibitors. These pose a minimal risk to the administration of local anesthetic agents with a vasoconstrictor; provided a minimum effective dose is used. In the recent past, MAO Inhibitors were

considered to be relative contraindications to the administration of vasoconstrictors. However, recent information has demonstrated the otherwise (Yagiela, 1999, and Naftalin and Yagiela, 2002). Citanest with Octapressin (Prilocaine with Felypressin) can be used in these cases.

Hematological Disorders

Anemia

There are various forms of anemia, which are as follows:

(1) Methemoglobinemia, (2) Iron deficiency anemia, and (3) Sickel cell anemia.

Methaemoglobinemia, which can be congenital, or acquired is a relative contraindication to the use of prilocaine, (Bardoczky, Wathieu and Dhollander, 1990; and Wilburn-Goo and Lloyd, 1991). Other forms of anemia do not pose any problems for administration of local anesthetic agents with or without vasoconstrictors.

Deficiencies such as (i) Hemophilia, (ii) Coagulopathies, (iii) Neoplastic (leukemia), (iv) Thrombocytic purpura, and (v) Thalassemia.

In all these disorders, the deficiencies are corrected with the help of the Hematologist, and with the necessary precautions and local hemostatic measures, surgical intervention can be undertaken.

Pregnancy

It is a relative contraindication for elective dental care, especially, in the first and third trimesters. Consultation with patient's obstetrician is necessary prior to oral surgical procedures involving use of local anesthesia, particularly, if there is history of any problems with the previous pregnancies, or if the patient is a primigravida.

Local anesthetic agents and vasoconstrictors are not teratogens and may be administered to pregnant patients during any trimester. However, it is important to be conservative in administering any drug to a pregnant patient, and it should be done in consultation with the obstetrician. Local anesthetic agents containing felypressin, such as Citanest forte (Prilocaine and Octapressin), should be avoided. Felypressin has got oxytocic properties. Lidocaine can be safely used, while Bupivacaine and Mepivacaine should be avoided.

AIDS

In a patient who is a known case of AIDS, the following tests are recommended to assess the general condition of the patient: Complete Blood Count including ESR, Mantoux test, liver function tests, such as serum proteins, CD4 and CD8 cell counts, routine urine examination, and

chest radiograph. CD cell counts tests decide whether the patient requires Antiretroviral therapy (ART) or not. The treating physician's consent is also taken.

The patient with AIDS is to be managed with the principles of "Universal Precautions". All barrier techniques should be employed. Face mask, head cap and double gloves should be donned. Today, special HIV kit is available for single patient use. The instruments used are sterilized by chemical sterilization by immersing in 1% sodium hypochlorite for 8-10 hours followed by double autoclaving.

BIBLIOGRAPHY

1. Bardoczky, GI, Wathieu M, and Dhollander A. Prilocaine-induced methaemoglobinaemia evidenced by pulse oximetry. Acta Anaesthesiol Scand 1990;34:162-64.
2. Chobanian AV, Bakris GL, Black HR, et al. National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute; National High Blood Pressure Education Programme Co-ordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. JAMA 2003;42:1206-52. (Complete version) (JNC-7).
3. Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. JAMA 1997;277(22):1794-1801.
4. DeToledo JC. Lidocaine and seizures. Therap Drug Monit 2000;22:320-22.
5. Gottlieb, SO, and Flaherty JT. Medical therapy of unstable angina pectoris, Cardiol Clin 1991;9:19-98.
6. Haas DA. An update on local anesthetics in dentistry. J Can Dent Assoc. 2002;68:446-551.
7. Naftalin LW, and Yagiela JA. Vasoconstrictors: Indications and precautions. Dent Clin N Amer 2002;46:433-46.
8. Quilici DL. Contraindications in the use of the periodontal ligament injection. Compendium 1990;11:26-100.
9. Sindel, LJ, and deShazo RD. Accidents resulting from local anesthetics. True or false allergy ? Clin Rev Allergy 1991;9:379-95.
10. Tarhan S, and Giuliani ER. General Anesthesia and myocardial Infarction, Am Heart J 1974;87:137.
11. Thompson P, Melmon K, Richardson J, et al. Lidocaine pharmacokinetics in advanced heart failure, liver diseases, and renal failure in humans. Ann Intern Med 1973;78:499.
12. Weinblatt E, Shapiro S, and Frank CW, et al. Prognosis of men after first myocardial infarction: Mortality and first recurrence in relation to selected parameters. Am J Public Health 1968;58:1329.
13. Wilburn-Goo D, and Lloyd LM. When patients become cyanotic: Acquired methaemoglobinaemia. JADA 1991;130:26-31.
14. Yagiela JA. Adverse drug interactions in dental practice: interactions associated with vasoconstrictors, Part V of a series. JADA 1999;130:501-709.

Chapter

14 Infection Control

ASEPSIS AND STERILIZATION

Asepsis and its role in the prevention of infection was put forward nearly two centuries ago. The general principles were laid down by Hungarian obstetrician, Ignaz Semmelweiss in Europe and Oliver W Holmes in USA. These principles were accepted after Joseph Lister's studies on prevention of wound infection. Subsequent development occurred with the introduction of steam sterilization. Later, there was addition by sterile gowns, gloves, drapes and surgical mask, etc.

DEFINITION OF VARIOUS TERMINOLOGIES

Cleaning: It is a process by which visible contamination is removed. However, it does not necessarily destroy micro-organisms. It is a necessary prerequisite for effective disinfection or sterilization.

Asepsis: It is the avoidance of pathogenic organisms.

Aseptic technique: It aims at excluding all micro-organisms. Surgical technique is aseptic, when sterile instruments and clothing and "no touch technique" is employed. The term describes methods which prevent contamination of wounds and other sites, by ensuring that only sterile objects and fluids come into contact with them.

Antisepsis: It is the procedure or application of an antiseptic solution, or an agent which inhibits the growth of micro-organisms, while remaining in contact with them, but does not necessarily imply sterility. The examples are scrubbing up and preparation of operative sites.

Antiseptic: It is a chemical, applied to living tissues, such as skin or mucous membrane to reduce the number of micro-organisms, by inhibition of their activity or by destruction.

Disinfection: It is a process which reduces the number of viable micro-organisms to an acceptable level, but may not inactivate some viruses and bacterial spores.

Disinfectant: It is a chemical substance, which causes disinfection. It is used on non-vital objects to kill surface vegetative pathogenic organisms, but not necessarily spore forms or viruses.

Sterilization: It is the process of destruction or removal of all microbial forms; including bacteria, spores, fungi, viruses. The term is applied to instruments and not to skin or mucous membrane.

METHODS OF STERILIZATION

The most commonly used methods are as follows (1) Heat: (a) by steam or moist heat, at a raised atmospheric pressure, in an autoclave, (b) by dry heat/hot air, at normal atmospheric pressure, in a dry oven. (2) Ethylene oxide. (3) Irradiation.

Heat (Figs 14.1 to 14.8)

Moist/Steam Heat: Autoclave

Steam is a very efficient means of sterilization, because of its:

i. High penetrating capacity, and
ii. It gives up a large amount of heat (latent heat) to the surface with which it comes into contact, and on which it condenses as water. Steam under pressure is the most practical and effective method of sterilization.

Advantages

i. The results are consistently good, and
ii. The instruments can be wrapped prior to sterilization.

Fig. 14.1: Dental clinic sterilizer

Fig. 14.2: Dry heat sterilizer

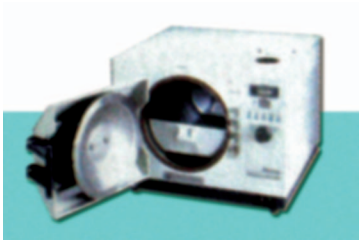


Fig. 14.3: Chemical sterilizer

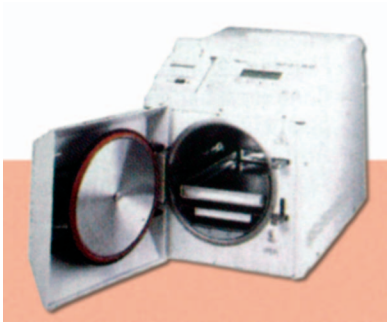


Fig. 14.4: Steam sterilizer

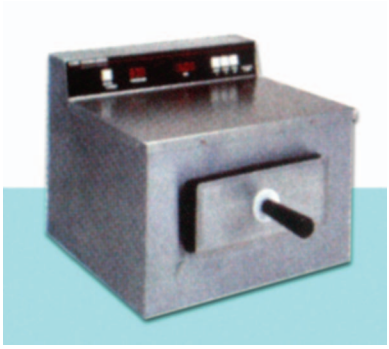


Fig. 14.5: Rapid heat transfer sterilizer

Fig. 14.6: Fully automatic autoclave

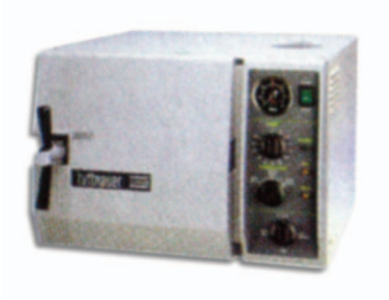


Fig. 14.7: Manual autoclave

Fig. 14.8: Valueklave 1730

Disadvantages

The main disadvantages are: (i) blunting and corrosion of sharp instruments, and (ii) damage to certain rubber goods.

Factors Required for Effective Autoclaving

There are three major factors required for effective autoclaving: (i) pressure, (ii) temperature and (iii) time:

i. *Pressure:* It is expressed in terms of psi (pounds per square inch) or kpa (kilopascals; kp = 0.145 psi).
ii. *Temperature:* To achieve required pressure, the temperature must be reached and maintained at 121°C (250°F).
iii. *Time:* Wrapped loads require a minimum of 20 minutes after reaching full temperature and pressure.

Materials Sterilized by this Method

A wide variety of materials can be sterilized by this method: Dressing packs, surgical instruments, and pharmaceutical products. Liquids can be sterilized by autoclave, provided that the contents do not get inactivated by the high temperatures.

The instruments are placed in sterilization bags, or in self-sealing or dual peel sterilization pouches, and then placed in the autoclaves (Figs 14.9 to 14.14).

The superiority of steam over hot air as a method of sterilization is due to the following:

i. Moist heat acts by denaturation and coagulation of enzymes and proteins; whereas dry heat acts by destructive oxidation of cell constituents.
ii. Condensation of a large amount of latent heat to the surface.
iii. Steam penetrates better than hot air.

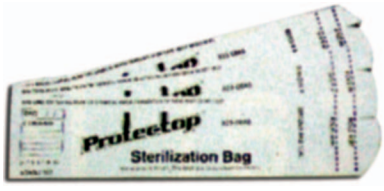


Fig. 14.9: Sterilization bags
- With sterilization indicators
- For use in steam chemiclave and dry heat

Fig. 14.10: Sterilization Bags—steam only paper

Fig. 14.11: Sterilization pouches

Features:

i. Internal process indicator to verify sterilization process
ii. New ¼" overlap for easy opening of sealed pouches

Fig. 14.12: Self-sealing sterilization pouches

Features:

i. Constructed with durable materials and thick seals to help prevent instrument and cassette "poke-through"
ii. Unique design optimizes the cassette fit, reducing bulky storage while simplifying packaging time
iii. Built-in external and internal quality control indicators relay accurate time and temperature verification
iv. The internal indicator strip can be attached to a patients chart for sterilization tracking

Fig. 14.13: Dual peel gas/steam sterilization pouches

Features:

- Combination paper/plastic peel-open pouches are designed for quick and easy presentation of sterile items
- The pouches can be used in gas or steam sterilizers
- Chemical process indicators on every pouch identify the sterilization process used
- One can view items within the pouch through the strong, clear plastic side
- Dual-peel film is tinted blue
- The other side is made of tough, moisture-resistant, surgical-grade paper which provides high porosity for fast sterilization while maintaining an effective bacterial barrier
- These self-seal pouches have a pressure-sensitive adhesive closure

Fig. 14.14: Sterilization pouches

Features:

- Easy peel-and-seal adhesive strip
- Exclusive internal processing indicator
- Autoclavable/chemiclavable
- Can be used for burs, implants, impression trays, bur blocks, hand instruments, surgical instruments, orthodontic instruments

Tests for Efficiency for Heat Sterilization

1. *Thermocouple:* This is a thermometric testing, and a reliable gauge of efficiency. One recording is taken from a thermocouple placed inside a test pack of towels and a second one from one in the chamber drain. Comparison between the two recordings gives a good guide regarding the speed at which the steam penetrates the load.
2. *Brown's test:* These are ampoules that contain a chemical indicator; which changes it's color; from red through amber to green at a specific temperature.
3. *Autoclave tape:* This is a tape printed with sensitive ink that undergoes a color change at a specific temperature (Bowie-Dick test). Two pieces of strips are stuck onto a piece of square paper and placed in the middle of the test pack. With the application of temperature of 134 degrees for 3.5 minutes, there is a uniform development of bars throughout the length of the strips. This shows that the steam has passed freely and rapidly to the center of the load.
4. *Spores of a non-pathogenic organism:* The organism used is an aerobic spore bearing organism, Bacillus steorothermophilus. Its spores are killed at 121°C after 15 minutes. The samples are put in autoclave, and subsequently, made to culture them.

Dry Heat Sterilization

It is an alternative method of sterilization of instruments, particularly, the sharp instruments. The basic action is dehydration and oxidation of micro-organisms. Dry heat has less penetration, and is less effective than moist heat.

Advantage

The advantage is that, the instruments do not rust if they are dried prior to their placement in the sterilizer.

Disadvantage

The disadvantage is that the process is time-consuming. It is achieved by two methods:

Hot Air Oven

It is used to sterilize items which do not get damaged by high temperatures, such as laboratory glassware, glass syringes and instruments. Hot air is a poor conductor of heat and has poor penetrating capacity. High temperature damages fabrics and melts rubber, hence should not be sterilized by this method.

Temperature and Time

This method of sterilization completely depends upon a combination of time and temperature. The following of temperatures and times are used (Table 14.1).

Table 14.1: Relation of temperature and time

Temperature		Time (minutes)
°C	°F	
160	320	120/60
170	340	60
150	300	150
140	205	180

Advantage

The only advantage of this method is the maintenance of sharp edges of cutting instruments, such as chisels. Hence sharp instruments are preferably sterilized by exposure to dry heat. Autoclaving will reduce their sharpness and lead to rusting.

Disadvantage

Carbon steel instruments can loose their hardness because of dry heat.

Ethylene Oxide Gas Sterilization

Ethylene oxide, a gas at temperatures above 108°C, is a highly penetrative, non-corrosive agent with a cidal action against bacteria, spores and viruses. It destroys micro-organisms by alkylation; and causes denaturation of nucleic acids of micro-organisms. It is highly toxic, irritant, mutagenic and potentially carcinogenic, and should not be used where heat sterilization of an object is possible. It is highly flammable, but when mixed with carbon dioxide this danger is minimized. It is ideal for electric equipment, flexible-fiber endoscopes and photographic equipment.

Advantages

The advantages are:

i. It penetrates extremely well, even going through plastics
ii. It can be used at a low temperature
iii. It leaves no residue
iv. It is a deodorizer
v. It is comparatively non-toxic
vi. Many heat sensitive articles, e.g. plastic, rubber, can be sterilized.

Disadvantages

The disadvantages include:

i. High cost of the equipment
ii. Toxicity of the gas, and the need for venting it
iii. Explosive and inflammable
iv. Longer period of aeration.

Boiling Water

Water maintains and conducts heat extremely well. Boiling water produces a temperature of 100°C at normal atmospheric pressure. It requires 10 minutes exposure to this temperature, to kill many bacteria and some viruses; (including HIV and HBV). However, prolonged time of 24 hours is required to kill bacterial spores, and even this prolonged time will not kill many viruses. Hence, boiling water is not recommended for sterilization of tissue penetrating instruments. Cutting instruments should not be sterilized by boiling as they loose their sharpness.

DISINFECTION

Disinfection (high level disinfection) is the term used for destruction of all pathogenic organisms, such as, vegetative forms of bacteria, mycobacteria, fungi and viruses but not spores, from inanimate surfaces, such as walls, furnishings, and equipment; and antisepsis is the term applicable to living tissues such as skin and mucous membrane.

Methods of disinfection include: (i) Cleaning, (ii) Heat, (iii) Boiling water, and (iv) Chemical disinfection.

i. **Disinfection by Cleaning:** Cleaning with a detergent is an excellent mode of disinfection. It removes almost all pathogens including bacterial spores.
ii. **Disinfection by Heat:** Heat is a simple and reliable disinfectant for almost anything except living tissues. Mechanical cleaning with hot water provides an excellent quality of disinfection for a wide variety of purposes.
iii. **Disinfection by Boiling Water:** Water maintains and conducts heat extremely well. Boiling water produces a temperature of 100°C at normal atmospheric pressure. It requires 10 minutes exposure to this temperature, to kill many bacteria and some viruses (including HIV and HBV). However, prolonged time of 24 hours is required to kill bacterial spores, and even this prolonged time well not kill many viruses. Hence, boiling water is not recommended for sterilization of tissue penetrating instruments. Cutting instruments should not be sterilized by boiling as they loose their sharpness. If the requirements as mentioned earlier, are not fulfilled, then it is not sterilization, but it is disinfection.
iv. **Disinfection by Chemical Agents:** They are used to disinfect the skin of a patient prior to surgery, and to disinfect the hands of the operator. No available chemical solution will sterilize instruments immersed in it. Secondly, there is a risk of producing tissue damage if residual solution is carried over into the wound while it is being used. The chemicals used are: Aldehydes, Diguanides, Phenolics and Halogen derivatives.

Aldehydes

i. *Formaldehyde:* It is a hazardous substance, flammable and irritant to the eye, skin and respiratory tract. It is used at up to 50°C and has limited sporicidal activity. It is used for large heat-sensitive equipment such as ventilators and suction pumps excluding rubber and some plastics.
ii. *Gluteraldehyde:* It is toxic, irritant and allergenic, and a high level disinfectant. It is applicable where heat cannot be used. It is frequently used for heat sensitive material. It is active against most vegetative bacteria (including *M. tuberculosis*), and some viruses (including HIV and HBV), fungi and bacterial spores. A solution of 2% gluteraldehyde (Cidex), requires immersion of 20 minutes for disinfection; and 6-10 hours for sterilization.

Diguanides

Chlorhexidine is active against a number of bacteria; including *Staphylococcus aureus* and some Gram-negative bacteria, but not spores, fungi and viruses. It can be prepared in alcohol or with cetrimide. It gets inactivated in the presence of soap, pus, plastics, etc. It is mainly used for cleaning skin and mucous membrane; 0.5% chlorhexidine in 70% alcohol; or chlorhexidine with cetrimide (Cetavlon or Savlon), or a 4% solution with detergent (Hibiscrub) as a preoperative scrub.

Halogens

Hypochlorites: They are active against bacteria, spores, fungi, and viruses, including Hepatitis B virus. They are readily inactivated by blood, pus and dilution. Sodium hypochlorite solution (household bleach) 1:10, freshly prepared solution, is an effective disinfectant.

DISINFECTANTS

A disinfectant may be classified, on the basis of their properties, into the following: Bactericidal, sporicidal, viricidal and fungicidal. They are usually used for decontamination of inanimate objects. The increase in their efficiency is related to increased toxicity.

Certain limitations have to be considered prior to embarking on their use: (i) Inactivation, (ii) Concentration, (iii) Stability, and (iv) Adequate contact.

Alcohols

Ethanol and isopropyl alcohols are frequently used as antiseptics. Alcohols possess some antibacterial activity, against some Gram-positive and negative bacteria, and especially against *M. tuberculosis*. Alcohols act by

denaturing proteins. They are not effective against spores and viruses. To have maximum effectiveness, the alcohol must have a 10-minute contact with the organisms. Solutions of 70% alcohol are more effective than higher concentrations, as the presence of water speeds up the process of protein denaturation. The alcohols do not function as disinfectants when instruments, handpieces, or other equipment are simply wiped with them, since they evaporate quickly. Instruments made of carbon steel should not be soaked in alcoholic solutions, as they are corrosive to carbon steel. Rubber articles absorb alcohol, and prolonged soaking can cause a reaction when the article subsequently comes in contact with living tissue.

Aqueous Quarternary Ammonium Compounds

The spectrum of their antibiotic activity is similar to alcohols, being limited largely to Gram-positive organisms and some Gram-negative organisms. It is not effective against spores, viruses and *M. tuberculosis*. It denatures intracellular protein. The effectiveness of these compounds depends on strength, activity and duration of contact.

Phenolic Compounds

The phenolic compounds are toxic to living tissues. These compounds, in high concentration, are protoplasmic poison, and act by precipitating the proteins and destroy the cell wall. Their spectrum of activity includes lipophilic viruses, fungi and bacteria but not spores. These compounds are used for disinfection of inanimate objects such as walls, floors and furniture. They may cause damage to some plastics, and they do not corrode certain metals, such as brass, aluminium, and carbon steel.

Aldehyde Compounds

i. Ageous solution of formaldehyde (formalin) and
ii. Glutaraldehyde (cidex) is effective disinfectants.

Formaldehyde

It is not popular because of its odor and because 18-30 hours of contact is necessary for cidal action.

Glutaraldehyde

Stonehill et al (1963) reported that glutaraldehyde kills vegetative bacteria, spores, fungi and virus by alkylation on a 10 hours contact. It is also toxic and irritating and, hence, not used on certain surfaces such as furniture, walls and floors. It can be safely used on metal instruments (for less than 24 hours), rubber, plastics and porcelain. It is activated by addition of sodium bicarbonate, but in its activated form it remains potent only for 14 days.

Antiseptics

An antiseptic is a chemical disinfectant (usually bacteristatic in the concentration it is used) that can be diluted sufficiently to be safe for application to living tissues (intact skin, mucous membranes, and wounds) while still retaining its antimicrobial property. They are less toxic than the disinfectants or the agents used for sterilization. Notable antiseptic agents include: Alcohols, aqueous quarternary ammonium compounds, hexachlorophene, and iodophor compounds.

Alcohols

They are frequently used for skin antisepsis prior to needle puncture. They are good organic solvents. The alcohols must have a prolonged contact with the organisms to have an antibacterial effect. This contact is prevented due to its rapid evaporation.

Ethanol (Ethyl alcohol) is employed in the concentration of 70% as a skin antiseptic. It has poor activity against bacterial spores, fungi, and viruses. Isopropyl alcohol is an inflammable secondary alcohol with an unpleasant burning taste. It is about twice as toxic as ethyl alcohol. It is slightly more potent a germicide than ethyl alcohol; and has a marked degreasing action. It is used in the concentration of 60-70% v/v, for disinfection of skin. The alcohols do not have reliable sporicidal, virucidal, or fungicidal action; hence, they are not useful for sterilizing surgical instruments.

Hexachlorophene Compounds

These are used for surgical scrubs and preoperative preparation of the surgical site. They are effective against Gram-positive organisms; less effective against Gram-negative organisms and fungi; and are not effective against viruses, spores, and *M. tuberculosis*. Skin cleansing with an agent causes reduction in surface bacteria.

Iodophor Compounds

Iodophor compounds are the most effective antiseptics. Iodine is complexed with organic surface-active agents, such as, polyvinyl-pyrrolidone (Betadine, Isodine). Their activity is dependent on the release of iodine from the complex. These compounds are effective against most bacteria, spores, viruses, and fungi. These are the most commonly used surface disinfectants along with hypochlorite. Concentrated solutions have less free iodine. Iodine is released as the solution is diluted. An appropriate dilution is 1: 2-3 parts of iodophor and distilled water, respectively.

Advantages

The advantages are: (i) Low toxicity, (ii) Prolonged residual effect, (iii) inexpensive, and (iv) Odorless. These compounds build up on the skin after successive scrubs, and that this provides long-lasting antibacterial activity.

Hand Disinfection

There are some proprietary preparations available for preoperative washing of hands of surgeons and assistants which have a bactericidal effect and which do not cause excessive drying of skin. The preparations available are:

i. Hibiscrub and Phisomed—contains 4% chlorhexidine gluconate.
ii. Betadine—contains 7.5% povidone-iodine.
iii. 3% PCMX.
iv. Soap containing disinfectants like hexachlorophene.
v. 70% Hibisol (2.5% chlorhexidine in 70% alcohol) lotion may be applied as an extra precaution.

Washing must be continued for 5 minutes in running water. This is followed by drying of hands and forearms.

Gloving

Hand Gloves

They help to protect the operator from infection by bacteria and viruses from patient's blood. Gloving is essential to protect both the surgeon and the patient from blood-borne viruses and to prevent wound becoming contaminated with the surgeons skin flora.

There are two types of gloves:

i. *Latex gloves:* It is a clear and the most common type of glove.
ii. *Brown milled rubber gloves:* These are thinner than latex gloves; and provide a better tactile sensation. However, they are more fragile and require more frequent changes during the operation. The "hand to glove", and "glove to glove" technique of donning the gloves should be employed. Double gloving affords extra protection, but at the expense of reduced sensitivity and dexterity, and possible discomfort.

Operating Room

A protocol should be followed which would comprise of general cleanliness of the ceiling, walls and floors of the clinic. The walls and the ceiling should be regularly painted, and periodic pest control should also be undertaken. The walls and the ceiling should be regularly disinfected by

fumigation, especially those clinics in which oral surgical and implantogical procedures are carried out. The air-conditioning system should be regularly maintained. General leakages and water drainage system are properly maintained and should be periodically checked for growth of fungus.

BIBLIOGRAPHY

1. Accepted Dental Therapeutcs. Chicago - American Dental Association 1977.
2. Crawford JR, Oldenburg TR. Practical Methods of Office Sterilization and Disinfection. J Oral Med 1967;22:133.
3. Kelsey JC. Sterilization and Disinfection: Objectives and Perspectives. Ann R Coll Surg Engl 1969;44:214.
4. Laskin DM. Oral and Maxillofacial Surgery Vol I and II. CV Mosby Co. AITBS Publishers India 1992.
5. McLundie AC. Sterilization in General Dental Practice. BDJ. 1968;124:214.
6. Peterson LJ, Ellis EIII, Hupp JR, Tucker MR. Contemporary Oral and Maxillofacial Surgery. 2nd ed. Harcourt Brace and Central Asia. Pvt Ltd 1997.
7. Rubbo S, and Gardner JF. A review of sterilization and disinfection. London-Lloyd-Luke Medical Books Ltd. 1965.
8. Stonehill AA, Krop S, and Borick PM. Buffered glutaraldehyde: A new chemical sterilizing solution. Am J Hosp Pharm 1963;20:458.

Preanesthetic Medications

Due considerations should be given to presurgical medications in patients who are undergoing extractions, minor oral surgical or periodontal surgical procedure, or restorative procedure, under local anesthesia.

Patients who are taking antibiotics should take probiotics, to counteract the side effects of antibiotics, and should be continued even after antibiotics are stopped. Patients who are taking analgesics, anti-inflammatory and antibiotics drugs for a pre-existing condition should take it in the immediate preoperative period and continue taking it for a few days postoperatively. Patients should use an antiseptic mouthwash such as chlorhexidine or betadine, a day prior to surgical procedure, after every meal; and on the day of surgical procedure.

Patients who become tense and sleepless, should take Tablet Alprazolam (such as Restyl) (0.25, 0.50 and 1.0 mg), the night before the procedure, to ensure good sleep. Patients with excessive salivation or other secretions are advised to take atropine (injection), or probanthine (tablet), or glycopyrrolate (both tablet and injection) preoperatively.

All medically compromised patients should continue with the medications they are taking, such as antihypertensive, anti-diabetic, and antithyroid agents, intraoperatively and postoperatively. Those patients taking anti-platelet and anti-coagulant drugs should stop the medications 3-5 days before and 2 days after the surgical procedure, in consultation with the treating cardiologist.

Patients who are taking anti-epileptic drugs and those taking psychiatric mediations should take the medications on the day of surgery.

METHODS OF LOCAL ANESTHESIA

There are several methods of achieving pain control with local anesthetic agents. The various types of techniques used for deposition of these agents, in dentistry are as follows: (1) Surface or topical anesthesia, (2) Infiltration anesthesia, (3) Field block, and (4) Nerve block or conduction anesthesia.

The selection of the type of anesthesia depends upon the area and the type of surgery. In general, infiltration anesthesia is adequate for a small isolated area, field block is indicated when two or more teeth are being treated, while a nerve block is indicated for dental or surgical procedure in a quadrant of a jaw.

Surface or Topical Anesthesia

By this method small terminal nerves in the surface area of the intact mucosa or the skin up to the depth of about 2 mm are anesthetised by application of a local anesthetic agent directly to the area.

Nerves Anesthetised

Superficial nerve endings.

Indications

i. Prior to the infiltration injection techniques or nerve blocks for making the insertion of the needle painless
ii. Prior to carrying out incision and drainage of abscesses
iii. Prior to removal of sutures.

Forms

Spray

I. The active ingradient is a suitable local anesthetic agent, such as 10% or 15% lignocaine hydrochloride in water base. The agent is expelled in small quantities from an aerosol container.

- *Advantage:* Rapidity of onset. The onset of time of anesthesia is approximately 1 minute and the duration of anesthesia is approximately 10 minutes.
- *Disadvantage:* When used as a spray, the solution is spread over more extensive area than desired.
- *Method of application:* It is used as a spray on the area in which the needle penetration is proposed to be made; or it can also be sprayed on a small cotton pellet or roll and then placed on the proposed site of injection for about one minute.

II. *Ethyl chloride spray:* It produces anesthesia by refrigeration. When sprayed onto either mucous membrane or skin, it gets volatilised rapidly, and produces rapid anesthesia.
- *Method of application:* The spray is directed over a limited area until "snow" appears.
- *Care:* Undue inhalation of vapours by the patient should be avoided.
- *Use:* Occasionally used to produce surface anesthesia prior to taking an incision for fluctuating abscesses.

Ointment

It is used for similar purposes as spray. The active ingredient is a suitable local anesthetic agent, such as 5% lignocaine hydrochloride.
- *Time of onset:* 3-4 minutes.
- *Application:* Ointments are used for application over tender and inflamed gingivae prior to deep scaling.

Emulsion

The active ingredient is a suitable local anesthetic agent, such as 2% lignocaine hydrochloride.
- *Indications:*
  i. When full mouth impressions are to be taken in patients who are prone to retching.
  ii. Relief of postoperative pain or tenderness following mucogingival surgical procedures such as gingivectomy.
- *Method of application:* One teaspoonful of the emulsion is swished around in the mouth and oropharynx for 1-2 minutes; and later is spat out immediately prior to taking impressions.

Jet Injection

- *Method:* It is a technique by which a small amount of local anesthetic solution is expelled as a jet into submucosa without the use of a hypodermic needle.

Specialised syringes are used for this technique. These techniques depend upon discharge of a small quantity of local anesthetic solution from a reservoir. It produces a fine jet of solution which penetrates the mucosa through a small puncture wound and produces surface anesthesia. The hypodermic needle is then inserted painlessly through the same wound. This technique is particularly useful prior to palatal injections.

Infiltration Anesthesia or Local Infiltration

This method is also known as terminal or peripheral anesthesia, as the induction of anesthesia is by the action of anesthetic agents on the terminal nerve fibers.

Maxilla and Mandible

Maxilla

The maxilla has thin labial/buccal cortical plate; and moreover shows areas of porosity, and the compact bone presents numerous foramina which aid in absorption of local anesthetic solution. These factors, therefore, make the maxilla more favorable for infiltration anesthesia techniques.

Mandible

The bone is generally dense and has thicker cortical plates than maxilla, particularly in posterior region, more so in the region of external oblique ridge. Only the anterior part of mandible presents sufficient porosity, which is favorable for infiltration techniques.

Nerves and Areas Anesthetised

Small peripheral terminal branches or free nerve endings in a certain area of oral surgical procedure are anesthetised by deposition of local anesthetic solution in the area. Solution is injected beneath the mucous membrane, or along the periosteum, or beneath the skin.

Examples

Administration of local anesthetic solution in submucosa for taking an incision, or into an interdental papilla prior to root planing.

Advantages

1. Easy and simple injection
2. Very high success rate, and
3. Good control of bleeding.

Disadvantages

The action is limited to a small area; hence considerable amount of solution has to be injected with multiple penetrations when large field is to be anesthetised.

Indications

This method is used when only the mucous membrane and the underlying connective tissues are to be anesthetised.

Contraindications

Presence of acute inflammation or infection at the site of injection.

Applications

Infiltration anesthesia helps in anesthetising (1) teeth as it affects dental nerves before they enter apical foramina; and (2) periodontal tissues.

Other Applications

Infiltration anesthesia is often used in conjunction with general anesthesia to reduce bleeding at the site of surgery; when vasoconstrictor is added to local anesthetic solution.

Technique

- *Needle:* The recommended gauge is 25, 27 or 30; and the recommended length is 1" or 25 mm.
- *Bevel of the needle:* The bevel should be facing the bone.
- *Point of insertion:* It is in the middle of the area to be operated.
- *Depth of penetration:* It is beneath the mucous membrane into the connective tissue.

This technique may require more than one needle insertions depending upon the extent of area to be anesthetised.

Care should be taken to avoid injury to the tissues in the following ways:

1. Avoid injecting the solution too rapidly.
2. Avoid injecting too large a volume of the local anesthetic solution.
3. Avoid injecting too superficially.

These situations will result in injury to the tissues in the form of pain at the time of injection, or persistent post-injection pain or sloughing of the overlying soft tissues.

Signs and Symptoms

Objective: Lack of demonstration of pain with instrumentation.

Types of Infiltration Anesthesia

There are various types of infiltration anesthesia depending upon the site of deposition of the local anesthetic solution. The solution can be deposited below the mucosa or in the submucosal layers, or in the subcutaneous connective tissue, just above the periosteum, beneath the periosteum, in the periodontal ligament, in the cancellous bone, in the interdental septum or in the pulpal tissues of the tooth. On the basis of deposition, these techniques can be categorised in the following ways:

1. Submucosal or subcutaneous anesthesia (Fig. 16.1)
2. Paraperiosteal or supraperiosteal anesthesia (Figs 16.2 and 16.3)
3. Subperiosteal anesthesia (Figs 16.4 and 16.5)
4. Intraligamentary (Periodontal ligament) anesthesia (Fig. 16.6)
5. Intrapulpal anesthesia (Fig. 16.7)
6. Intraosseous anesthesia (Fig. 16.8)
7. Intraseptal anesthesia
8. Palatal infiltration (Figs 16.9 and 16.10)

Infiltration Techniques

Submucosal Injection

- *Technique (Fig. 16.1):* The local anesthetic solution is deposited in the immediate submucosal tissue layers. The solution diffuses through the interstitial tissues and reaches the terminal fibers of the nerve in the area of deposition of the local anesthetic solution.
- *Procedure:* The needle is inserted beneath the mucosal layers. Care should be exercised to avoid injecting too superficially. Excessive amounts injected superficially may lead to sloughing of the overlying tissues. Usually 0.25-0.5 ml of the local anesthetic solution is deposited.

Paraperiosteal or Supraperiosteal Injection

It is commonly called the local infiltration and is the most frequently used local anesthetic technique. The paraperiosteal injection is commonly used injection technique for obtaining anesthesia in the region of all maxillary teeth and mandibular anterior teeth because of thin cortical plates and abundant cancellous bone.

- *Site of insertion:* The needle is inserted through the mucosa, and the solution is deposited in close proximity to the periosteum or along the periosteum, in the vicinity of the apex of the tooth to be treated, as close to the bone as possible. This facilitates diffusion through the periosteum and penetration through the haversian canals of the cortical bone. These canals are numerous; near apices of teeth near the surfaces.

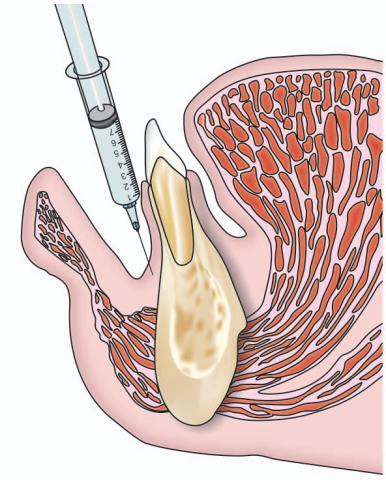


Fig. 16.1: Infiltration—submucosal injection—
the position of the point of the needle is in the submucosal layers

Dr Mendel Nevin has pointed out that the term "supraperiosteal" is not appropriate in its literal sense. It does not indicate where the injection is to be made; whether it is in the skin, the fascia, the muscle or the subcutaneous connective tissues. It can be anywhere between the surface of mucosa and the outer side of periosteum. Usually, in this technique, the solution is injected either into deeper submucosal layers, or just above and outside the periosteum. Hence the term "paraperiosteal", which means "along the periosteum", is more appropriate, and is used in preference over supraperiosteal because the solution is always deposited besides or along the periosteum.

- *Technique:* By this method the local anesthetic solution is deposited just above or besides the periosteum. It does not always produce satisfactory anesthesia due to distension of tissues due to superficial injection. In this method, the local anesthetic solution is not carried quickly from the area of deposition of the local anesthetic solution, through the alveolar bone; so that sufficient amount of local anesthetic solution does not diffuse through cortical layer into cancellous bone, which is necessary to produce satisfactory anesthesia. This is in particular, in cases of extirpation of pulp or preparation of a cavity. In these situations, it is necessary for the alveolar bone to be anesthetised but the local anesthetic solution should diffuse through apical foramen into the pulp chamber.
- *Dr Nevin's technique:* All maxillary incisors can be anesthetised by making the initial puncture over canine on each side and passing the needle horizontally towards central incisors infiltrating the apices of individual teeth. This is an excellent method of anesthesia for alveolectomy or gingivectomy in this region. Needless to say that palatal anesthesia is required.

- *Advantages:* Only two punctures are made labially. Similarly, if it is desired to anesthetise all mandibular incisors, only one labial puncture is made. The needle insertion takes place well down the vestibular mucosa of mandibular labial frenum; and is directed towards one canine fossa. About 1 ml of solution is deposited. The needle is then withdrawn to a point permitting its course to be directed to other canine fossa. Deposit 1ml of local anesthetic solution. Massage the solution to enhance absorption through numerous foramina.
- *Other common names:* Local infiltration, Supraperiosteal injection.
- *Nerves anesthetised:* Large terminal branches of superior dental plexus.
- *Areas anesthetised:* The region innervated by the large terminal branches such as the pulps of the maxillary teeth, labial/buccal periodontium, supporting alveolar bone, and labial/buccal mucoperiosteum; that includes labial/buccal periosteum, overlying connective tissue, and mucous membrane.
- *Indications:* This method is used for procedures in the entire maxilla and anterior mandible. In these areas, the cortical plates are thin, and there is abundant cancellous bone. The local anesthetic solution penetrates bone through Haversian canals. These canals are numerous near the apices of teeth near the surfaces.
  1. Pulpal anesthesia when treatment is limited to one or two teeth in maxilla, and anterior mandible.
  2. Soft tissue anesthesia for surgical procedures in a circumscribed area.
  3. Children and young adults. In children, this technique can be used in the posterior mandible to anesthetise deciduous molars as the cortical bone is thin in this region.
- *Contraindications:*
  1. Presence of acute inflammation or infection in the area of injection.
  2. Presence of dense bone covering the apices of teeth, as in maxillary first molar, because of overlying buttress of zygoma.
- *Advantages:*
  1. High success rate
  2. Technically easy injection
  3. Usually atraumatic
- *Disadvantages:*
  The technique is not recommended for large areas because of: (i) need for multiple penetrations, (ii) the necessity to administer larger volumes of anesthetic solution, and (iii) satisfactory anesthesia cannot be always produced.
- *Technique (Figs 16.2 and 16.3):*
  - *Needle:* A 25 or 27 gauge short needle is recommended.
  - *Point of insertion:* It is at the height of mucobuccal fold in the vicinity of the tooth to be anesthetised.

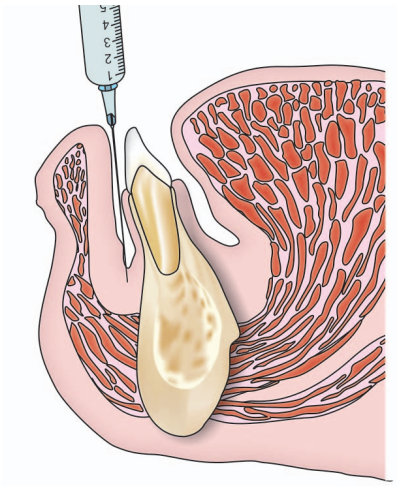


Fig. 16.2: Infiltration—paraperiosteal/supraperiosteal injection—the position of the point of the needle is above the periosteum

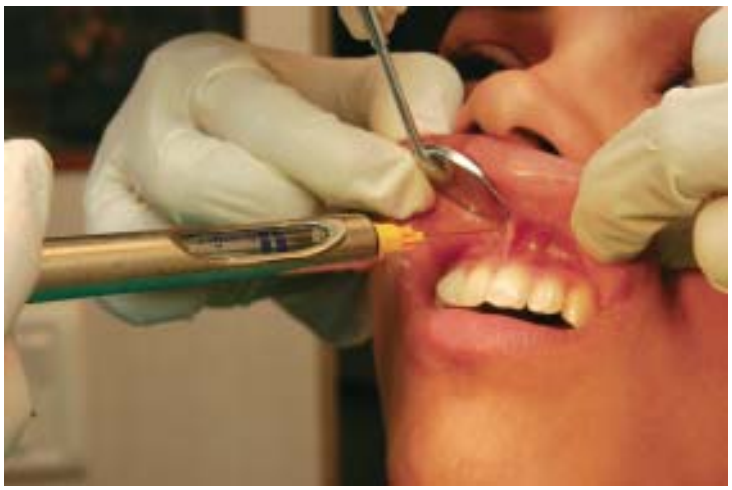


Fig. 16.3: Infiltration—paraperiosteal/supraperiosteal injection in anterior maxilla—the position of the point of the needle is at an angle of 45° to the long axis of the tooth

- *Target area:* The apical region or above the apex of the tooth to be anesthetised.
- *Depth of insertion:* Few millimeters.
- *Bevel:* The position of the bevel of the needle should be facing the bone.
- *Landmarks:*
  - Mucobuccal fold in the region of the tooth to be anesthetised.
  - Crown of the tooth.
  - Root contour of the tooth.

- *Procedure:*
  - *Position of the patient:* The occlusal plane of maxillary teeth should be at an angle of 45° to the floor.

- *Position of the operator:*
  - i. For maxillary injections, for the right side, the operator stands by the side of the patient; and for the left side, the operator stands in front of the patient.
  - ii. For mandibular injections, the operator stands by the side of the patient for the left side; and in front of the patient for the right side.
- Preparation of the tissues at the site of injection with an antiseptic.
- Application of topical anesthetic at the site of injection.
- Retract the lip/cheek, pulling the tissues taut.
- Take a preloaded syringe. Initially, hold it at an angle of 45° to the long axis of the tooth to be anesthetised, with the bevel of the needle facing the bone. Insert the needle at the height of mucobuccal fold, or a few millimeters away from the labial cortex.
- Aspirate, if negative, deposit approximately 0.5 ml of the solution slowly over 20 seconds.
- Depth of insertion: Few millimeters.
- Withdraw the syringe slowly.
- Cover the needle.
- Wait for 2-3 minutes, check for the signs and symptoms of anesthesia, and start the procedure.

- *Signs and symptoms:*
  1. *Subjective:* Feeling of numbness in the area of distribution of the nerve anesthetised.
  2. *Objective:* Absence of pain with instrumentation and during the procedure.

Subperiosteal Injection

In this method, the local anesthetic solution is injected beneath the periosteum. Subperiosteal injection has superiority over supraperiosteal injection. It confines the solution below periosteum; obviates permeating the pulp. The solution is under pressure enabling it to penetrate the cancellous bone, periodontal membrane and finally diffuses through apical foramen into the pulp.

- *Technique (Figs. 16.4 and 16.5).*
- *Needle:* Recommended length and gauge are 1" and 25 respectively.

The needle is inserted midway between gingival margin and the approximate apex of the tooth; and at right angle to the buccal alveolar plate, in order to penetrate mucous membrane, gingival tissue and periosteum.

The needle is then placed at an angle of 45° to the alveolar plate, bevel facing the bone and then it is advanced towards the apex of the tooth, beneath the periosteum. As the needle progresses, about 0.3-0.5 ml of local

Fig. 16.4: Infiltration—subperiosteal injection in anterior maxilla—the position of the point of the needle is at an angle of 90° to the long axis of the tooth and the alveolar bone as seen from the side

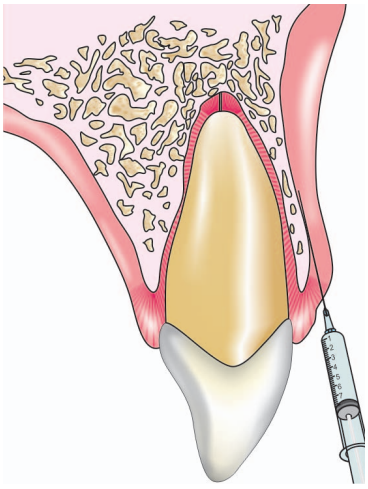


Fig. 16.5: Infiltration—subperiosteal injection— the position of the point of the needle is underneath the periosteum at an angle of 45° to the long axis of the tooth

anesthetic solution is injected slowly. The periosteum will force the solution through the cortical plate and into the cancellous bone.

The same procedure is repeated lingually. The amount of solution deposited is 0.5 ml. The diffusion of solution through the lingual alveolar plate is faster because of presence of numerous foramina. The length of the needle inserted is between 5 and 7 mm.

- *Periosteum:* There is an aspect, associated with the method of subperiosteal injection, which needs discussion.

  One theory advanced was that a subperiosteal injection may produce persistent or prolonged pain due to "tearing" of the periosteum from

bone. Contrary to general belief, the periosteum is not closely attached to bone in the same way a glove covers a hand.

According to Gray, the periosteum in the young bones, is thick and is vascular and is less closely connected with the body of the bone from which it is separated by a layer of soft tissues containing osteoblasts. Later in life, the periosteum becomes thinner and less vascular and the osteoblasts are converted into a layer of epithelial cells. Only at the ends of bones, the periosteum is closely adherent. This is quite well known that there is comparatively more resistance for raising mucoperiosteal flap at gingival margins; and once detached at this point, the separation of the rest of the mucoperiosteum is comparatively easy. On the other hand, the periosteum and gingival tissues are merged together and cannot be separated from one another.

If deposition of a small amount of the solution tears the periosteum, then lifting of mucoperiosteal flap should be considered to be more injurious. However, it is well known that the procedure of raising a flap causes little pain.

- *Advantages:*
  1. It is more appropriate, more specific and definite in region.
  2. There is no great trauma, contrary to belief.
  3. It is safe and much more effective than supraperiosteal injection.
  4. Less solution is required to produce the desired results. Total amount of solution sufficient to produce satisfactory and profound anesthesia is 0.3-0.5 ml.
  5. The onset of action is rapid. The depth of anesthesia for extraction is achieved immediately, however, for conservative restorative procedures such as preparation of cavities and crowns, and extirpation of pulps, it is advisable to wait for five minutes to allow the solution to reach pulp chamber and anesthetise the neural component.
  6. This method greatly reduces the incidence of intravascular administration.
  7. Reduces needle punctures.
- *Disadvantages:* There is theoretical damage to the periosteum. No greater trauma is created by injecting local anesthetic solution beneath the periosteum.

Supplementary Injections

Intraligamentary, intrapulpal, intraosseous, and intraseptal injection techniques: These are the other methods of producing anesthesia. These are satisfactory when executed properly. Each has its place of application in dentistry. Sometimes, these will produce anesthesia where all other methods fail. The abovementioned techniques numbered: 4, 5, 6 and 7 are not advised for beginners. These techniques give good results in experienced hands.

Intraligamentary (Periodontal or Peridental) Injection

As the name suggests, the local anesthetic solution is deposited into the periodontal ligament or membrane. This injection technique is claimed to be safe, provided the point of insertion of the needle is thoroughly cleaned and strict aseptic precautions are undertaken. The local anesthetic solution is carried from the alveolar bone and through the apical foramen into the pulp chamber.

- *Indications:* It is a very efficient method of producing anesthesia especially for cavity preparation, crown preparation, pulp extirpation, etc.
- *Advantages:*
  i. Rapid onset of action.
  ii. Specific analgesia to isolated teeth. Single mandibular tooth can be anesthetised without performing a pterygomandibular block. This avoids numbness of the lip and tongue. There is less likelihood of inadvertently traumatising these structures in the immediate post-injection phase.
  iii. Useful adjunct to conventional local anesthesia; and in experienced hands for minor surgical procedures.
- *Disadvantages:*
  i. Post-injection discomfort due to temporary extrusion.
  ii. Apparent increase in the incidence of "dry socket".
- *Technique (Fig. 16.6):*
  Needle: 25-gauge is recommended.

The local anesthetic solution is injected along periodontal membrane of maxillary and mandibular teeth, using small amounts of local anesthetic solution, usually 0.2 ml, delivered via a specifically designed system, which comprises of high pressure syringes and ultrafine needles. The high pressure forces the solution rather than causing diffusion, through the periodontal ligament to the nerves in that area. This technique can also be carried out by the conventional syringes, however, care should be exercised to avoid shattering of the glass cartridges.

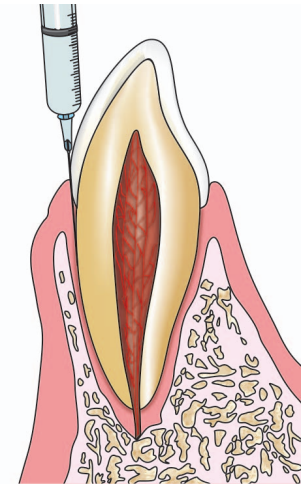


Fig. 16.6: Infiltration—intraligamentary injection—the position of the point of the needle is in the periodontal space

The needle is inserted into the gingival sulcus and into the periodontal ligament. This technique can anesthetise only single individual tooth. The single rooted tooth should be injected on the mesial and the

distal sides; or buccal and lingual sides; while multirooted teeth are injected over each root. The amount of local anesthetic solution injected is 0.1-0.2 ml.

Some studies have shown that application of excessive pressure and the injection of excessive amounts can cause avulsion of teeth.

- *Procedure:*
  i. Strict aseptic precautions.
  ii. The needle is inserted on the mesial side of the tooth to be treated, beneath the free margin of gingivae, or into the circular ligaments.
  iii. A few drops of local anesthetic solution are injected for superficial anesthesia. Solution is deposited in tissues to render further insertion of the needle painless.
  iv. The needle is then forced into periodontal ligament, directing it parallel to the long axis of the tooth.
  v. Usually 0.2-0.4 ml of anesthetic solution is sufficient to achieve profound anesthesia (1 ml contains 15.419193 drops).

Hold the syringe close to needle and reach into the membrane. Needle is held at an angle of 15° to the long axis of the root and inserted until interdental septum is reached at a point midway labiolingually or buccolingually, according to the location of tooth.

Using the septum as a guide from this point, deflect the needle towards the desired tooth to be anesthetised, then force the needle into mucous membrane. Solution is deposited with pressure, sufficient to rupture tissue and cause leakage of solution. If anesthesia has not taken place after 2-3 minutes, the flap on the other side of the tooth may be punctured and proceeded as above.

Intrapulpal Anesthesia

This technique is indicated for obtaining anesthesia for procedures which require direct instrumentation of the pulpal tissue. First, put a cotton ball soaked in local anesthetic solution in the cavity, wait for a minute; and then a 25 or 27-gauge needle is inserted directly into the pulp chamber. The needle should be held firmly or wedged into the pulp chamber or the root canal. Initially, slight discomfort is felt by the patient which subsequently gets subsided. Sometimes the needle is bent to get a proper angle for good approach (Fig. 16.7).

Intraosseous Injection Technique

In this method, the local anesthetic solution is deposited directly into the cancellous bone adjacent to the tooth to be anesthetised, between the two cortical plates of bone of maxilla and mandible. Intraosseous injection is usually an adjunct, and is used when conventional methods have been tried and failed.

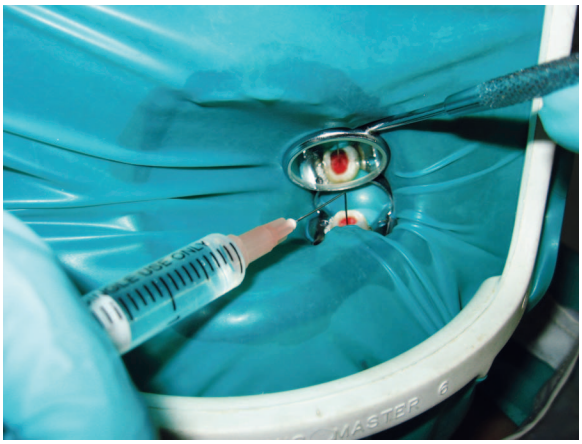


Fig. 16.7: Infiltration—intrapulpal injection—the point of the needle is in the pulp chamber (*Courtesy:* Dr Vibha Hegde)

Sometimes, it is impossible to obtain profound anesthesia of teeth and jaws by ordinary methods, not at least of sufficient intensity to allow extirpation of pulp, and preparation of cavities, etc. This may be due to one of the following reasons: (i) abnormal location of foramen and (ii) unusual density of external cortical plate. These difficulties are overcome by intraosseous technique.

- *Advantage:* It produces profound single tooth anesthesia.
- *Disadvantage:* Specialised equipment and technique is needed.
- *Technique (Fig. 16.8):*
  - The soft tissues overlying the apex of the tooth are first anesthetised with paraperiosteal injections. This injection should be made either mesial or distal to the tooth to be anesthetised, and slightly above the roots, in order to avoid injury to the teeth.

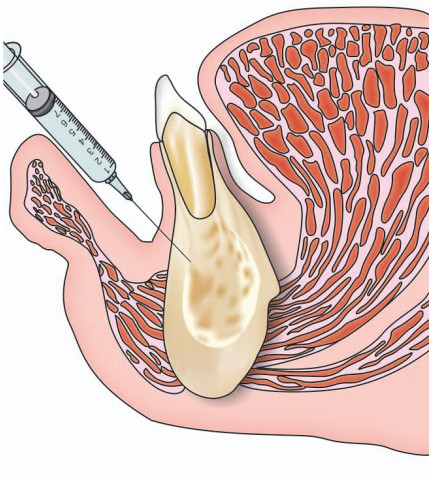


Fig. 16.8: Infiltration—intraosseous injection—the position of the point of the needle is in the cancellous bone through the perforation in the cortex

- An incision is made in the mucosa and the periosteum.
- A small opening or perforation is made in the outer cortical layer of bone with the help of SS White HP-8 round bur. The drill is similar to 25-gauge needle. The local anesthetic solution is placed through outer cortical plate into cancellous bone with the help of a needle, which is inserted through the perforation made in the bone. That allows the local anesthetic solution to reach the nerve immediately. The needle with a blunt point is used. The needle should not be forced against resistance at any time. The needle used should be of such a gauge (usually 23 or 25-gauge), that it fits snugly into the opening made in the bone to avoid possible leakage around the needle, while the local anesthetic solution is being injected.
- Here usually enough anesthesia is present to permit drilling through outer cortical plate painlessly. The drilling can be done either on the mesial or the distal side of the tooth to be treated.

- *Procedure:*
  - Preliminary infiltration: In order to prevent trauma, a preliminary injection of a few drops of infiltration is made at the selected site before making a perforation.
  - A small incision is made in the mucosa. The mucoperiosteum is elevated and then the buccal alveolar plate is perforated.
  - The outer cortical plate is perforated at the point with a round bur numbered in a straight handpiece.
  - The drill is directed almost at an angle of 45° to the long axis of the teeth directing it slightly palatally or lingually. Drill the external plate until it reaches cancellous bone. Stop drilling and withdraw the drill, until a drop of blood oozes out. The drill should not enter more than 2 or 3 mm.
  - The needle is inserted into the opening created; and approximately 0.5-1 ml of solution is slowly injected under pressure.

Anesthesia by intraosseous method will not be of very long duration, possibly between 10 and 20 minutes. If the operation is not completed, inject more solution and anesthesia will be re-established immediately.

- *Precautions to be taken:*
  1. Deposition of too much solution rapidly may produce signs and symptoms of toxic reactions, as the solution is rapidly absorbed in the cardiovascular system from cancellous bone.
  2. If for any reason, it is necessary to operate on the tooth at a later date, it is easy to find the drill hole, and inject a small amount of solution.
  3. Never attempt to anesthetise more than one tooth on each side of the drill opening. This requires too much of local anesthetic solution which may produce toxic symptoms.

The technique for intraosseous injection is essential in upper jaw, especially in anterior and bicuspid region. The beginners should get acquainted with upper anterior region before attempting lower teeth.

To obtain intraosseous anesthesia in the mandible, make drill opening in the retromolar triangle with a drill in straight handpiece.

Intraseptal Anesthesia

It is considered as a variation of intraosseous anesthesia. A needle is forced gently into the porous interseptal bone on either side of the tooth to be anesthetised. The local anesthetic solution is then forced under pressure into the cancellous bone. The solution is taken up by the pericementum and the apical nerves. The superficial mucous membrane should be anesthetised before the needle is inserted into the interseptal bone. This technique is more effective in children and young adults.

The interdental septum is of cancellous bone and the solution quickly diffuses through the alveolar bone.

- *Indication:* In older patients, there is a great degree of recession of gingivae, wherein the intraligamentary anesthesia is not quite effective.
- *Technique (1):* The injection is given in the septum of two adjoining teeth, in between the two cortical plates.
- *Procedure:* The syringe with the needle is inserted into the interdental septum, exerting sufficient pressure on the dense outer layer of bone to reach the deeper cancellous structures.
- *Technique (2):* Some operators advocate drilling the outer cortical plate similar to that for intraosseous injection. Then the needle is inserted into the opening made and then local anesthetic solution is deposited. The few drops of local anesthetic solution are injected slowly under pressure.

Local Infiltration of the Palate

The anesthesia of the hard palate is necessary for dental procedures involving manipulation of palatal soft and hard tissues. The palatal injections are one of the most painful injections. It is advisable to inform the patient prior to injection about the pain during the injection. This helps in preparing the patient psychologically.

- *Precautions to be taken:*
  i. Deposition of excessive amount of local anesthetic solution causes blanching of overlying soft tissues and results in necrosis in the area.
  ii. Use of highly concentrated vasoconstrictors in local anesthetic agents can lead to ischemic necrosis and sloughing of the soft tissues.

- *Measures to reduce discomfort:* The various measures, which reduce the discomfort to the patient during palatal anesthesia, are as follows:
  1. Provide adequate topical anesthesia at the site of injection.
  2. Use pressure anesthesia at the site before and during needle insertion and the deposition of the solution.
  3. Maintain control over the needle.
  4. Deposit the local anesthetic solution slowly.
- *Nerves anesthetised:* Terminal branches of greater palatine and nasopalatine nerves.
- *Areas anesthetised:* Soft tissues and bony hard palate in the vicinity of the injection.
- *Indications:*
  i. Anesthesia in a small area of injection
  ii. Hemostasis in the area of surgery
- *Contraindications:*
  i. Presence of acute inflammation or infection at the site of surgery.
  ii. Provides a small area of anesthesia.
- *Advantages:*
  i. It provides good hemostasis if vasoconstrictor is used along with the local anesthetic agent.
  ii. As it involves a small area of anesthesia, it gives minimum discomfort to the patient.
- *Disadvantage:* It is a potentially painful injection.
- *Technique (Figs 16.9 and 16.10)*
  - *Needle:* 27 or 30-gauge needle is usually recommended, however, 25-gauge needle can also be used.
  - *Point of insertion:* In the mucoperiosteum on a line 1 cm from the gingival margin, or midway between the gingival margin and the median palatine raphe.
  - *Target area:* Mucogingival tissues in the area of injection.
  - *Anatomical landmarks:* Mucogingival tissues in the vicinity of the area to be injected.

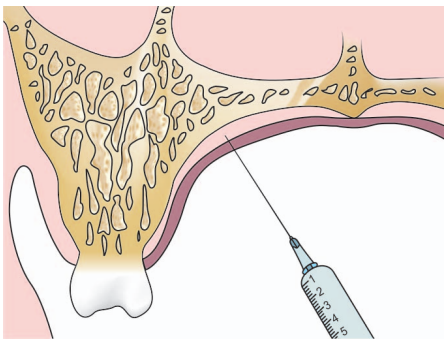


Fig. 16.9: Infiltration—palatal anesthesia—paraperiosteal / supraperiosteal injection

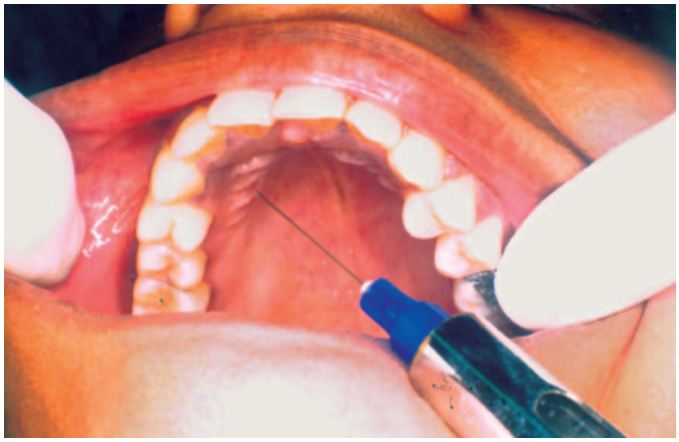


Fig. 16.10: Infiltration—palatal anesthesia paraperiosteal / supraperiosteal injection as seen in the mouth

- *Path of insertion:* The needle is inserted into the tissues from the opposite side at an angle of 45° to the bony surface of the palate.
- *Bevel:* It should be facing the palatal soft tissues and bone.

- *Procedure:*
  - *Position of the operator:* For the right-sided injections, the operator stands in front of the patient; and for the left-sided injections the operator stands by the side of the patient.
  - *Position of the patient:* The occlusal plane of the maxillary teeth is at an angle of 45° to the floor. The patient is asked to keep his mouth wide open and the neck extended. The head may be turned to the right or to the left for right-and left-sided injections respectively, to increase visibility.
  - *Preparation of the tissues:* The tissues are prepared with antiseptic and topical anesthetic solutions.
  - Take a preloaded syringe, and insert the needle at the point of insertion from the opposite side at an angle of 45° to the bony surface. Penetrate the mucoperiosteum with the needle to touch the bone gently. Withdraw the needle by 1 mm and deposit about 0.25-0.5 ml of the solution in the vicinity of the area to be anesthetised. Withdraw the needle slowly. Cover the needle and keep it aside.
  - Wait for a few minutes.
  - Start with the surgical or the dental procedure.
- *Signs and symptoms:*
  1. Numbness in the area of deposition of the local anesthetic solution.
  2. Lack of demonstration of pain with instrumentation.
  3. Absence of pain during the procedure.

- *Complications:* Sloughing and ischemic necrosis: It is seen when a local anesthetic agent with highly concentrated vasoconstrictors are used; and in excessive amount. Hence do not inject more than 0.5 ml at any one injection site; or stop as soon as blanching of the tissues occurs.

Field Block

The local anesthetic technique commonly used, and, referred to in dentistry as local infiltration, is in reality a field block, as the anesthetic solution is deposited at or above the apex of the tooth to be treated. This method is the most preferred method for all the teeth except the lower posterior teeth.

Nerves Anesthetised

Terminal nerve branches in the vicinity of the area to be anesthetised.

Areas anesthetised

The areas anesthetised by the field block will be larger and circumscribed. These areas include the pulps of the teeth and the tissues distal to the site of injection, which comprises of the supporting alveolar bone, buccal periodontium and overlying soft tissues.

Difference between Field Block and Nerve Block

The difference in the field block and the nerve block is basically the extent of anesthesia achieved or the nerve terminal anesthetised. Field block is more circumscribed, involving tissues in and around one or more teeth. The nerve blocks involve a larger area, as they involve a larger terminal nerve, along the course of the nerve, such as seen following pterygomandibular block.

Indications

1. All maxillary teeth
2. Mandibular anterior teeth.

Contraindications

1. Presence of acute inflammation or infection at the site of injection.
2. Mandibular posterior teeth. The alveolar bone in the region of mandibular bicuspids and molars is usually thick and dense to permit diffusion of the local anesthetic solution.

Technique

The local anesthetic solution is deposited near the larger terminal nerve branches. Technically speaking, all the injections given above the apices of all maxillary teeth to achieve anesthesia should be considered as field blocks. The examples are maxillary injections administered at or above the apices of the teeth.

Nerve Block or Conduction Anesthesia

By this method, a nerve trunk is blocked at some point between the periphery and the brain; thereby depriving the area of sensation distal to the point where the nerve is blocked. It does not interfere with brain centres, and does not cause loss of consciousness of the patient.

The local anesthetic agent is deposited close to a main nerve trunk, usually at a distance from the site of surgical procedure. It provides a larger anesthetic field as the entire area of distribution of the nerve is anesthetised.

Methods

There are (1) Intraoral, and (2) Extraoral nerve blocks.

1. Nerve blocks for maxillary subdivision and its branches:
   a. *Intraoral nerve blocks:* (i) Infraorbital nerve block, (ii) Posterior superior alveolar nerve block, (iii) Greater palatine nerve block, (iv) Nasopalatine nerve block, and (v) Maxillary nerve block.
   b. *Extraoral nerve blocks:* (i) Infraorbital Nerve Block, and (ii) Maxillary Nerve Block.
2. Nerve blocks for mandibular subdivision and its branches:
   a. *Intraoral nerve blocks:* (i) Pterygomandibular block, Direct and indirect techniques, (ii) Lingual nerve block, (iii) Long buccal nerve block, (iv) Mental nerve block, (v) Gow-Gates nerve block, and (vi) Vazirani-Akinosi nerve Block.
   b. *Extraoral nerve blocks:* Mandibular Nerve Block.

Indications

1. Extensive maxillary and mandibular oral and periodontal surgical procedures,
2. Restorative dental procedures; and
3. Extensive maxillofacial soft and hard tissue procedures.

Contraindications

Presence of acute inflammation or infection at the site of injection.

Advantages

1. Avoids multiple penetration of the needle.
2. Avoids deposition of large volume of local anesthetic agent.

Disadvantages

1. Larger area than required is anesthetised.
2. Additional local infiltration is required if hemostasis is required at the site of surgery.

BIBLIOGRAPHY

1. Archer WH. A Manual of Dental Anesthesia. An Illustrated Guide for Students and Practitioners. 2nd ed. WB Saunders Company 1958.
2. Howe GL, Whitehead FIH. Local Anesthesia in Dentistry. A Dental Practitioner Handbook. 3rd ed. Varghese Publishing House 1997.
3. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
4. Nevin, Mendel, Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1954.
5. Nevin, Mendel, Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1948.
6. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. New Delhi: CBS Publishers and Distributors 1990.

Chapter

17 Injection Techniques for Maxillary Nerve and its Branches

There are various techniques used to achieve adequate anesthesia of the teeth and soft and hard tissues in the maxilla.

A. Infiltration anesthesia
   1. Submucosal injection
   2. Paraperiosteal/Supraperiosteal injection (Figs 17.1 to 17.3)
   3. Subperiosteal injection (Fig. 17.4)
   4. Intraligamentary/Periodontal ligament injection
   5. Intrapulpal anesthesia
   6. Intraosseous injection
   7. Intraseptal injection
   8. Infiltration of the palate (Figs 17.5 and 17.6)

B. Nerve blocks
   a. *Intraoral nerve blocks:* (i) Infraorbital nerve block, (ii) Posterior superior alveolar nerve block, (iii) Nasopalatine nerve block, (iv) Greater palatine nerve block, and (v) Maxillary nerve block.

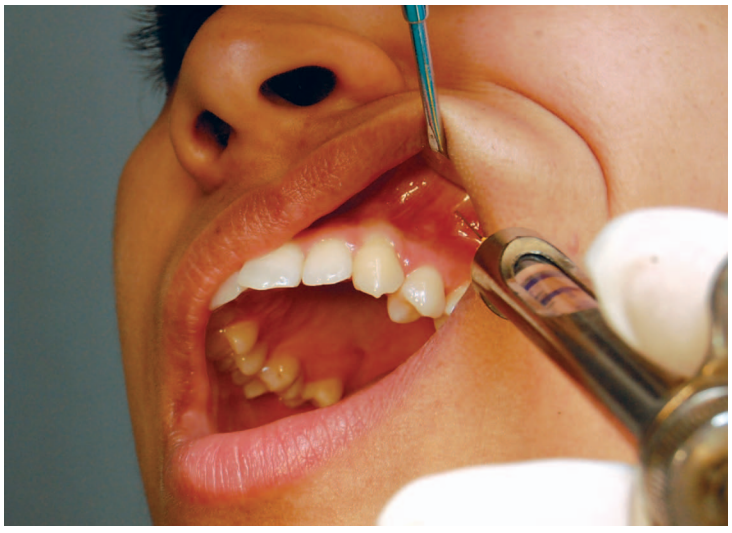


Fig. 17.1: Infiltration—Supraperiosteal injection in the premolar region of the maxilla. The position of the point of the needle is at an angle of 45° to the long axis of the tooth as seen from the side

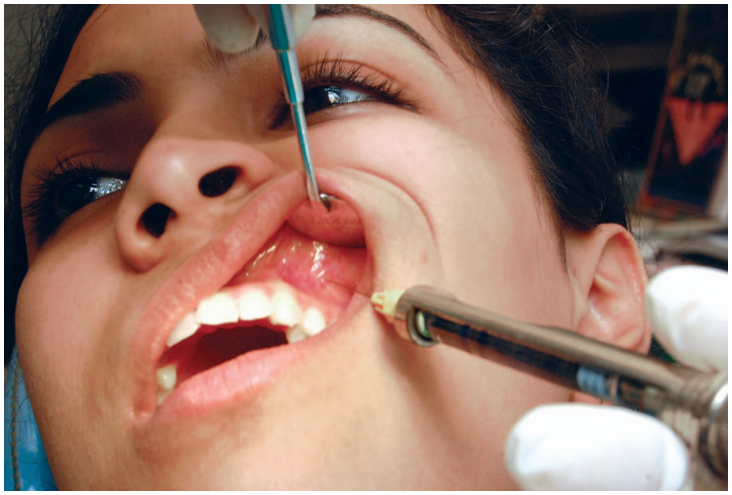


Fig. 17.2: Infiltration—Supraperiosteal injection in the premolar region of the maxilla. The position of the point of the needle is at an angle of 45° to the long axis of the left premolar tooth as seen from the front

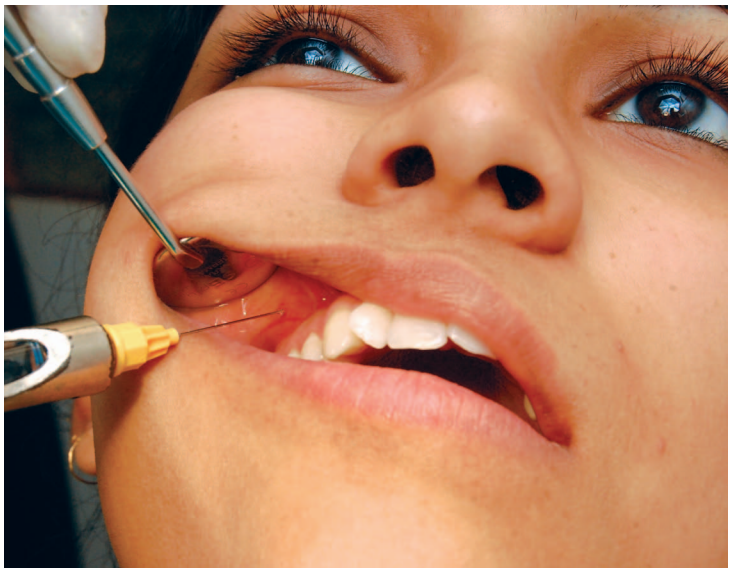


Fig. 17.3: Infiltration—Supraperiosteal injection in the molar region of the maxilla. The position of the point of the needle is at an angle of 45° to the long axis of the right molar tooth

b. *Extraoral nerve blocks:* (i) Infraorbital nerve block, and (ii) Maxillary nerve block.

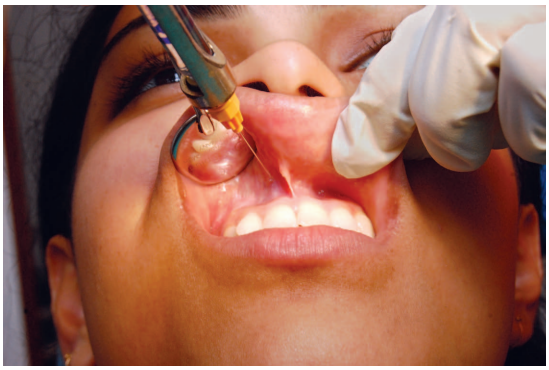


Fig. 17.4: Infiltration—Subperiosteal injection in anterior maxilla. The position of the point of the needle is at an angle of 90° to the long axis of the tooth and the alveolar bone as seen from the front

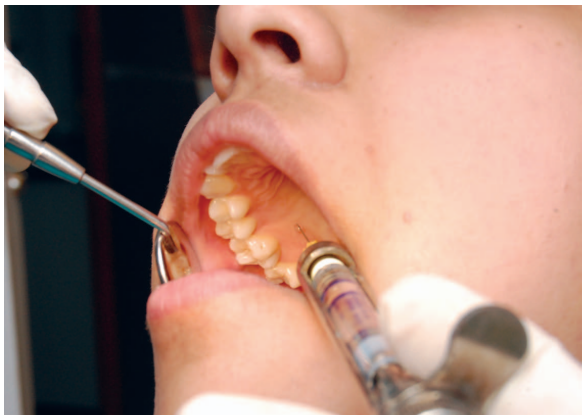


Fig. 17.5: Infiltration—Palatal anesthesia: Paraperiosteal/Supraperiosteal injection as seen on the right side in the molar region

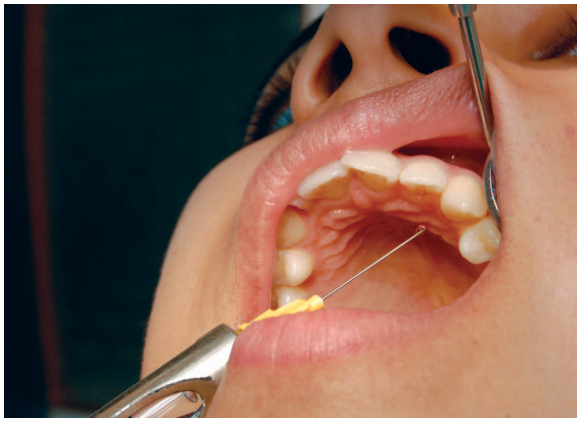


Fig. 17.6: Infiltration—Palatal anesthesia: Paraperiosteal/Supraperiosteal injection as seen on the left side in the anterior (canine) region

INFILTRATION TECHNIQUES

These techniques are described in Chapter 16.

NERVE BLOCKS

A. Intraoral blocks
B. Extraoral blocks.

Intraoral Nerve Blocks

Infraorbital Nerve Block

There are two approaches to execute the infraorbital nerve block; the bicuspid and the central incisior. The nerves and the areas anesthetised, indications, contraindications and advantages are same for both the approaches. However, the disadvantages are different, as discussed on page 189.

A good knowledge of anatomical landmarks and adherence to injection protocol are essential for this procedure.

Other Names

Anterior superior alveolar nerve block.

Nerves Anesthetized

1. Anterior superior alveolar nerve.
2. Middle superior alveolar nerve.
3. Infraorbital nerve-along with its terminal branches on the face: Inferior palpebral, lateral nasal, and superior labial nerves.

Areas Anesthetized

The following structures, on the side of injection, are anesthetized:
1. Pulps of maxillary central and lateral incisors, and canine.
2. Pulps of maxillary premolars and mesiobuccal root of first molar.
3. Supporting alveolar bone and the labial or buccal periodontium of these teeth.
4. Overlying labial or buccal mucoperiosteum in the region of incisors, canine and premolars.
5. Skin of lower eyelid and both surfaces of conjunctiva, skin of lateral aspect of the nose, and skin and mucosa of upper lip.

Indications

1. Oral and periodontal surgical procedures in the soft and hard tissues involving more than two maxillary teeth, such as apicoectomies, alveolectomies of maxillary anterior regions, impacted canines, and cysts.
2. Restorative and endodontic procedures involving more than two maxillary teeth.

3. Presence of acute inflammation or infection at the site of injection.
4. Presence of dense cortical bone that makes any infiltration technique ineffective.

Contraindications

1. Discrete treatment areas (one or two teeth only).
2. When hemostasis in the area of surgery is desirable. In such situations, an additional local infiltration into the area is indicated.

Advantages

1. The techniques are comparatively simple, easy and safe.
2. The techniques minimize the volume of solution to be injected and the number of needle punctures to be made in order to achieve the desired anesthesia.
3. The incisor approach lessens possibility of inadvertently entering the orbit.
4. It permits deeper penetration into the infraorbital canal, since the direction of the needle is parallel to the direction of the canal.

Disadvantages

- *Bicuspid approach:*
  1. Psychological: Fear of injury to the patient's eye
  2. Anatomical: Difficulty in defining landmarks.
- *Incisor approach:*
  3. There are higher chances of injuring the infraorbital neurovascular bundle with deeper penetration into the infraorbital canal.

Anatomical Landmarks

- *Bicuspid approach:* All the following structures on the ipsilateral side, such as (1) infraorbital margin, (2) infraorbital depression, (3) infraorbital foramen, (4) first bicuspid, (5) mucobuccal fold in the region of this tooth, (6) pupil of the ipsilateral eye in the forward gaze, (7) angle of the mouth, and (7) mental foramen.
- *Incisor approach:* Other additional landmarks: (i) central incisor and canine on the ipsilateral side, and (ii) mucobuccal fold in the region of canine.

Approximating Structures with the Tip of the Needle in the Final Position

The structures in the vicinity of the tip of the needle in the final position when the infraorbital nerve comes out of infraorbital foramen are: (i) the infraorbital head of quadratus labii superioris muscle is above, and (ii) the origin of levator anguli oris (caninus) muscle is below.

Approaches

- *Bicuspid approach:* This technique is comparatively easy and is recommended for the beginners. The bicuspid approach is simple and causes minimal complications. The needle passes through the mucosa and areolar tissue and during insertion should pass beneath and lateral to the facial artery and facial vein.
- *Technique (Figs 17.7 to 17.10):*
  - o *Position of the patient:* The patient is placed comfortably in the chair so that the maxillary occlusal plane is at an angle of 45° to the floor.

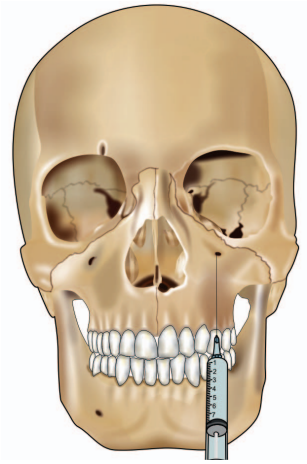


Fig. 17.7: Infraorbital nerve block—Bicuspid approach. The position of the needle is in the vicinity of infraorbital foramen

  - o *Position of the operator:* The operator stands on the right side of patient for right-sided block; and stands in front of the patient for the left-sided block.
  - o *Preparation of the tissues:* The tissues at the site of injection are prepared with an antiseptic.
  - o *Needle:* Long and 25-gauge needle is recommended.
  - o *Bevel:* The bevel is positioned in such a way that it is facing the bone.

Fig. 17.8: Infraorbital nerve block—Bicuspid approach. The position of the needle is for bicuspid approach for the right side

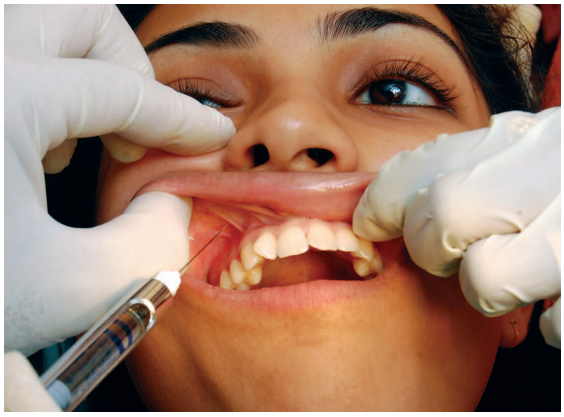


Fig. 17.9: Infraorbital nerve block—Bicuspid approach. The position of the needle is for bicuspid approach for the right side (closer view)

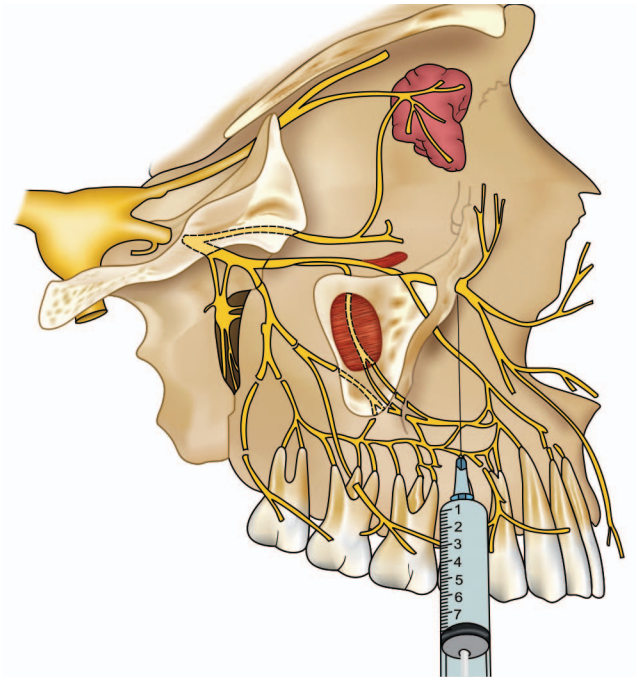


Fig. 17.10: Infraorbital nerve block—Bicuspid approach. The position of the point of the needle is in the vicinity of infraorbital foramen in relation to the infraorbital nerve

- *Depth of penetration:* 3/4th of an inch of the needle penetrates the soft tissues.
- *Area of insertion:* At the height of mucobuccal fold, or 4-5 mm away from the buccal cortex of maxilla in the region of first bicuspid.
- *Target area:* Infraorbital nerve as it comes out of infraorbital foramen.

- *Procedure*
  - o *Palpation of the anatomical landmarks:* Locate the infraorbital margin. Move your finger downward from the margin, applying gentle pressure to the tissues. As the finger continues inferiorly, a concavity will be felt. This is the infraorbital depression. The deepest part of the depression is the infraorbital foramen.
  - o Maintain your finger on the foramen or mark the skin at the site.
  - o Retract the lip, pulling the tissues in the mucobuccal fold taut, thus increasing the visibility.
  - o Take a preloaded syringe, and insert the needle into the height of the mucobuccal fold over the first bicuspid with the bevel facing the bone.
  - o Orient the syringe towards the infraorbital foramen.
  - o The needle should be held parallel to the long axis of the tooth as it is advanced to avoid premature contact with the bone, initially.
  - o Advance the needle until bone is gently contacted.
  - o Care should be taken to protect the eye with thumb/finger to limit the passage of the needle towards the eye.
- *Central incisor approach:* The needle passes through mucosa and areolar tissue and beneath the levator labii superioris (angular head of the quadratus labii superioris) muscle. It then passes anterior to the origin of levator anguli oris (caninus) muscle and beneath the facial artery and facial vein.
- *Technique (Figs 17.11 to 17.13):*

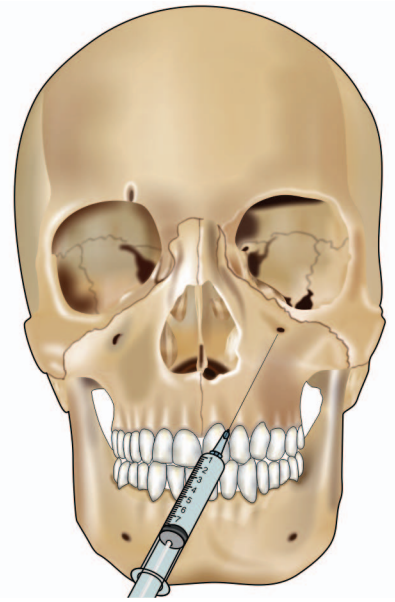


Fig.17.11: Infraorbital nerve block—Central incisor approach. The position of the point of the needle is in the vicinity of infraorbital foramen

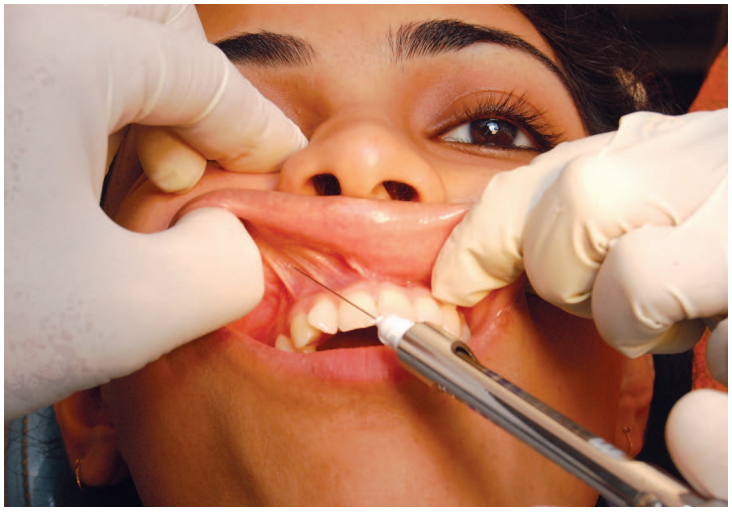


Fig. 17.12: Infraorbital nerve block—Central incisor approach.
The position of the needle is for incisor approach as seen for the right side

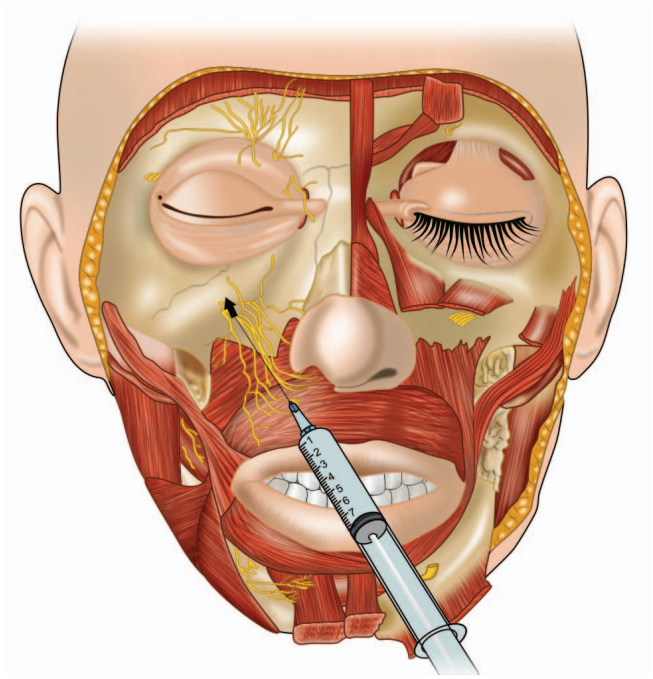


Fig. 17.13: Infraorbital nerve block—Central incisor approach. The position of the point of the needle is in the vicinity of infraorbital foramen in relation to the infraorbital nerve

- There are certain steps which are common to both the approaches, such as position of the patient, position of the operator, preparation of the tissues, configuration of the needle, and palpation of the anatomical landmarks; and are mentioned with the bicuspid approach.
- *Area of insertion:* In the central incisor approach, the direction of the needle is such that it bisects the crown of the ipsilateral central incisor from the mesioincisal angle to the distogingival angle. The area of insertion is at the height of mucobuccal fold, or 4-5 mm away from the labial cortex of maxilla in the region of ipsilateral canine. The needle is inserted about 5 mm from the mucobuccal fold in the region of ipsilateral canine.
- *Target area:* Infraorbital nerve, as it comes out of infraorbital foramen, between levator labii superioris muscle above and levator anguli oris muscle below.

- *Procedure:*
  - *Palpation of the anatomical landmarks:* This is done in the same way as for the bicuspid approach.
  - The needle is guided into the position by the thumb pressing over the infraorbital depression and marking the location of the infraorbital foramen).
  - In either approach, the needle should not penetrate more than 3/4th of an inch. Approximately, 1 ml of solution is slowly deposited in this area and the thumb is held in position until the injection is completed.
  - The surgeon will be able to feel the anesthetic solution, as it is deposited beneath the finger on the foramen, if the needle tip is in the correct position.
  - Maintain firm pressure with the finger over the injection site both during and for at least one minute after the injection.
  - Massage the tissue postero-superiorly so that the solution can easily diffuse through into the infraorbital foramen.
  - Wait for 3-5 minutes after completion of the injection before commencing the dental procedure.
- *Signs and symptoms:*
  a. *Subjective:* Tingling and numbness of the lower eyelid, side of the nose and upper lip.
  b. *Objective:*
     1. Comparing the sensation produced with tapping of anesthetised and adjacent unanesthetised teeth with an instrument.
     2. No pain during oral surgical or periodontal surgical procedures or dental therapy.

- *Complications*
  1. *Hematoma:* It may rarely develop.
  2. *Paresis of face:* It occurs when the injection is given superficially, when the needle lies in the vicinity of muscles of facial expression or the nerves innervating them. The effects disappear as the local anesthetic effect wears off.
  3. *Failure to obtain anesthesia:*
     a. Poor injection technique:
        i. Needle contacting bone below the infraorbital foramen. To correct, withdraw the needle a little, keeping the tip of the needle inside the soft tissues, redirect upwards towards the infraorbital foramen.
        ii. Needle deviation medial or lateral to the infraorbital foramen. To correct, withdraw the needle a little, keeping the tip of the needle inside the soft tissues, redirect towards the infraorbital foramen.
     b. Intravascular administration: Deposition of the local anesthetic solution into a vessel.

Posterior Superior Alveolar Nerve Block

Factors to be Considered

The factors to be considered prior to giving a posterior superior alveolar nerve block are as follows:

1. *Nerve supply of first molar:* The posterior superior alveolar nerve block is effective for the anesthesia of maxillary third, second and first molar (Adatia 1987). However, the mesiobuccal root of the maxillary first molar is not consistently innervated by the posterior superior alveolar nerve. Therefore, a second injection, usually a paraperiosteal, is indicated following the Posterior Superior Alveolar Nerve Block, when effective anesthesia of the first molar does not develop.
2. *Risk of hematoma formation:* In the target area, the needle lies in close proximity with the pterygoid plexus of veins. In order to reduce the incidence of hematoma, the following measures are recommended:
   i. Use short needles (1" or 25 mm in length). With this length overinsertion is avoided.
   ii. Always aspirate before deposition of the solution to avoid inadvertent intravascular injection.
   iii. Use 25-gauge needle, as it facilitates aspiration.
   iv. The needle should be oriented at an angle of 45° to the maxilla, in posterior, superior and medial direction.
3. *Patient's skull size:* This gives an idea of the depth of soft tissue penetration.

Other Names

(i) Tuberosity block, (ii) Zygomatic block.

Nerves Anesthetised

Posterior superior alveolar nerves and its branches.

Areas Anesthetised

1. Pulps of maxillary third, second and first molar (except the mesiobuccal root).
2. Adjoining alveolar bone of these teeth, buccal periodontium, and buccal mucoperiosteum.
3. Adjacent lining of maxillary sinus.

Indications

1. Oral surgical or periodontal surgical procedures in the area of maxillary molars.
2. Restorative procedures involving two or more maxillary molars.
3. When paraperiosteal injection is contraindicated as in the presence of acute inflammation or infection.
4. When paraperiosteal injection has failed.

Contraindication

When the risk of hemorrhage is high, as in a case of hemophilic. In such cases, a paraperiosteal or intraligamentary injection is recommended.

Advantages

(1) Atraumatic, (2) High success rate, (3) Minimizes the number of penetrations required, and (4) Minimizes the total volume of anesthetic solution injected.

Disadvantages

(1) Risk of hematoma, (2) Technique is somewhat arbitrary, as there are few bony landmarks during insertion, and (3) Second injection is required for anesthetising the first molar.

Technique (Figs 17.14 to 17.16)

- *Needle:* A 25-gauge, short needle of 25 mm in length is recommended.
- *Bevel:* The position of the bevel of the needle should be facing the bone.
- *Point of insertion:* It is at the height of mucobuccal fold in the region of the distal surface of maxillary second molar.
- *Depth of insertion:* It is approximately 16 mm.
- *Target area:* The posterior superior alveolar nerve as it enters the posterior or the infratemporal surface of maxilla. This nerve is located posterosuperior and medial to maxillary tuberosity.

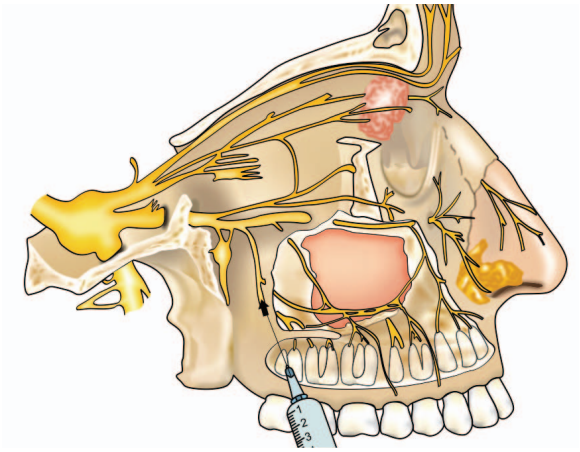


Fig. 17.14: Posterior superior alveolar nerve block. The position of the point of the needle is in relation to the posterior superior and lateral surface of maxilla in relation to the posterior superior alveolar nerve as seen in the mouth

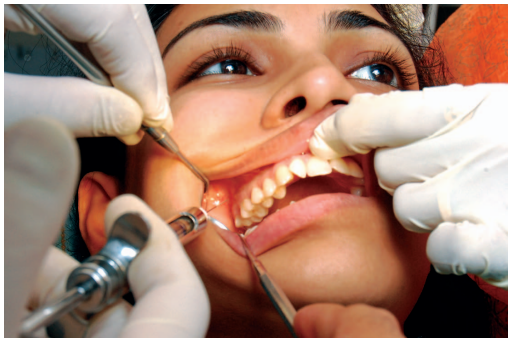


Fig. 17.15: Posterior superior alveolar nerve block. The position of the point of the needle is at the height of the mucobuccal fold opposite the distal surface of the maxillary second molar

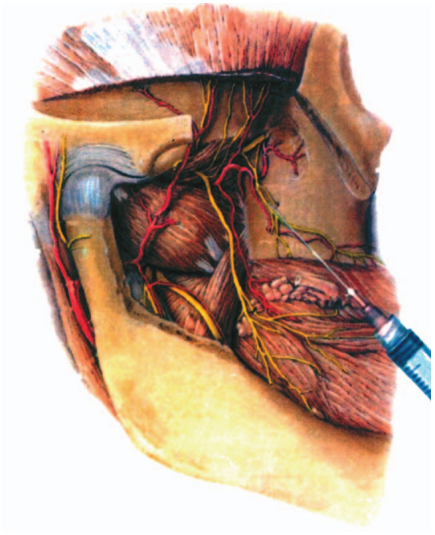


Fig. 17.16: Posterior superior alveolar nerve block. The direction of the needle is for posterior superior alveolar nerve block

- *Anatomical landmarks:*
  - Mucobuccal fold in the region of maxillary second molar
  - Maxillary tuberosity
  - Zygomatic process of maxilla or the buttress of zygoma
  - Infratemporal surface of maxilla
  - Anterior border and coronoid process of the ramus of the mandible.

Procedure

- *Position of the patient:*
  The patient is placed in semi-supine position with the occlusal plane of maxillary teeth at an angle of 45° to the floor.
- *Position of the operator:*
  i. For right-sided injection the operator stands by the side of the patient.
  ii. For left-sided injection the operator stands in front of the patient.
- *Preparation of the tissues:* The site of injection is prepared with the application of an antiseptic, followed by application of a topical anesthetic.
- Partially open the patient's mouth, pulling the mandible to the side of injection and maxillary occlusal plane at an angle of 45° to the floor.
- Retract the cheek, pulling the tissues taut.
- Palpation of the landmarks.

Technique I

- Place the index finger in the mucobuccal fold in the region of bicuspids and move it in the posterior direction till the prominence of the buttress of the zygoma is reached, which is approximately located above the first molar. At this point the index finger is rotated so that the fingernail is facing the attached gingiva, but the finger tip is still in contact with the prominence of the buttress. Pass the finger over the prominence and it will dip superiorly in the sulcus posterior to the buttress.
- Retract the finger laterally to expose the depth of the sulcus posterior and superior to the buttress. Adjust the finger so that it is in a plane at right angle to the occlusal plane of the maxillary teeth and at an angle of 45° to the patient's sagittal plane, posterior to the buttress of the zygoma.
- The point of the needle in this position should be located in the depth of the sulcus, above the roots of the third molar, and anterior to the maxillary tuberosity close to the lateral surface of the maxilla.
- The needle of the preloaded syringe is inserted into the tissue in a line parallel to the index finger and bisecting the fingernail with the bevel of the needle facing the bone.

Technique II

- Take a preloaded syringe. Insert the needle at the height of the mucobuccal fold, in the region of maxillary second molar.
- Advance the needle slowly superiorly, posteriorly, and medially, in one movement.
  - o *Superiorly:* At an angle of 45° to the occlusal plane.
  - o *Medially:* At an angle of 45° to the sagittal plane.
  - o *Posteriorly:* At an angle of 45° to the coronal plane.
- In an adult of normal size, penetration to a depth of 16 mm will place the needle tip in the target area, in the immediate vicinity of the foramina through which the posterior superior alveolar nerves enter the posterior surface of maxilla.
- Aspirate, if negative, deposit approximately 0.5-1.0 ml of local anesthetic solution slowly.
- Withdraw the syringe slowly.
- Cover the needle with its sheath, and keep it in a safe place.
- Wait for 3-5 minutes and start the procedure.

Signs and Symptoms of Anesthesia

1. *Subjective:* It is difficult to determine the extent of anesthesia subjectively. Feeling of numbness in the area of distribution of posterior superior alveolar nerve.
2. *Objective:* Absence of pain with instrumentation, and during the procedure.

Failure to Achieve Anesthesia

1. Poor injection technique:
   i. Needle too lateral: To correct, withdraw a little and redirect the needle tip medially.
   ii. Needle too low: To correct, withdraw a little and redirect the needle tip superiorly.
   iii. Needle too far posterior: To correct, withdraw the needle and redirect it anteriorly.
2. Intravascular administration: Deposition of the local anesthetic solution in pterygoid plexus of veins, and in posterior superior alveolar artery.

Complications

i. *Hematoma:* It is due to insertion of the needle too far posteriorly into the pterygoid plexus of veins. It is recommended that we use short needles to minimise the risk of puncturing the pterygoid plexus of veins and posterior superior alveolar artery. This can be seen immediately with the appearance of bluish swelling at the puncture point or copious bleeding on withdrawal of the needle.

ii. *Mandibular anesthesia:* The mandibular division of trigeminal nerve is located lateral and posterior to posterior superior alveolar nerves. Deposition of local anesthetic agent lateral to the desired location can produce varying degrees of mandibular anesthesia. In such situations patients will complain of anesthesia in the lip and tongue.

Nasopalatine Nerve Block

It is a potentially painful injection.

Other Common Names

Incisive nerve block, sphenopalatine nerve block.

Nerves Anesthetised

Nasopalatine nerves bilaterally. These nerves emerge from incisive foramen beneath the incisive papilla 1 cm behind maxillary central incisors in the midline.

Areas Anesthetised

Anterior portion of the hard palate (palatal mucosa) from the mesial of the right canine/first premolar to the mesial of the left canine/first premolar.

Indications

1. Oral surgical or periodontal surgical procedures involving palatal soft and hard tissues.
2. When anesthesia of palatal soft tissues is required for any restorative procedure on more than two teeth.

Contraindications

1. Presence of acute inflammation or infection at the site of injection.
2. Whenever there are smaller areas of dental or surgical procedures (one or two teeth).

Advantages

1. It minimises multiple needle penetrations and reduces the volume of local anesthetic solution to be deposited.
2. It minimises patient discomfort from multiple needle penetrations.

Disadvantages

1. There is no hemostasis except in the immediate area of injection. For achieving hemostasis in the areas away from deposition of the solution additional infiltration has to be given in the area of surgery.
2. It is potentially the most painful intraoral injection, if the preliminary preparatory injections are not employed.

Anatomical Landmarks

- Maxillary central incisor teeth
- Incisive papilla in the midline of the palate
- Incisive foramen.

Technique (Figs 17.17 to 17.23)

- *Needle:* The recommended gauge is 25 or 27, and the recommended length is 1" or 25 mm.
- *Area of penetration:* The palatal mucosa or the halo surrounding the incisive papilla.
- *Target area:* The nasopalatine nerve as it comes out of incisive foramen, beneath the incisive papilla.
- *Path of insertion:* Making an angle of 45° to the incisive papilla, approaching from the side.
- *Bevel:* It is facing the palatal soft tissues or facing the palatal bone.

Procedure

The nasopalatine nerve block is an extremely painful injection and hence a preparatory injection is necessary. It is recommended that the needle should not penetrate incisive papilla directly.

Preparatory Injections

There are two methods of giving preparatory injections. These make the entrance into papilla less painful.

a. *Labial approach (Figs 17.17 and 17.18):* The preparatory injection is made by inserting the needle into the labial intraseptal tissues in between the maxillary central incisors. The needle is inserted at a right angle to the labial cortical plate and passed into the tissues until resistance is felt. Then 0.25 ml of local anesthetic solution is deposited.

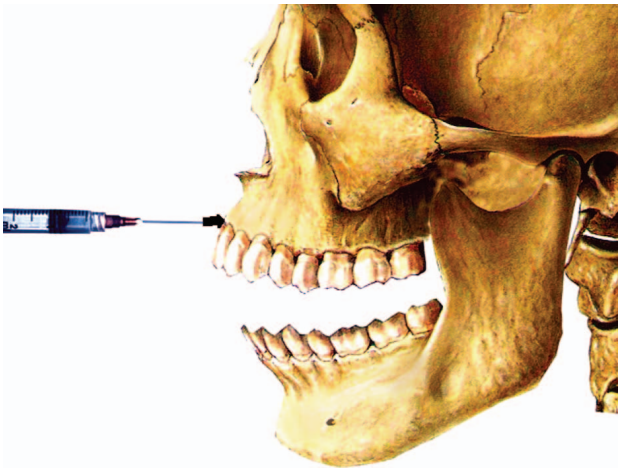


Fig. 17.17: Nasopalatine nerve block. Labial approach. The position of the needle is in the interdental papilla between the two maxillary central incisors in the midline on the labial side

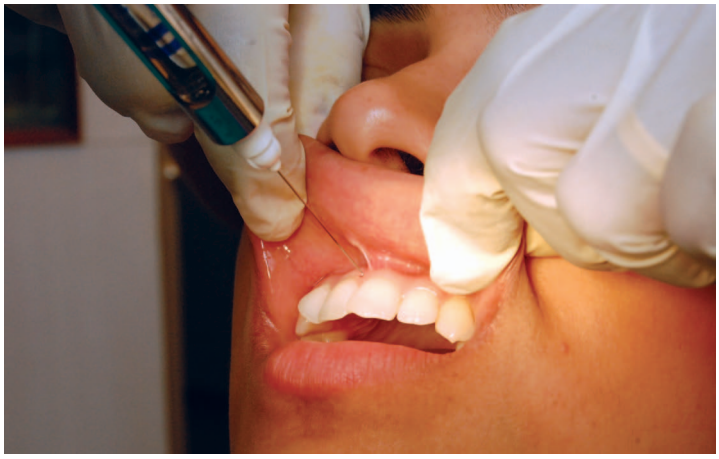


Fig. 17.18: Nasopalatine nerve block. Labial approach. The position of the needle is in the interdental papilla between the two maxillary central incisors

b. *Palatal approach (Figs 17.19 to 17.23):* The tip of the needle should be placed in the halo or the depression surrounding incisive papilla and a small amount or a few drops of local anesthetic solution is injected until papilla blanches.

In both the palatal and labial approaches, it is advisable to start injecting slowly as soon as the needle enters the mucosa. The palatal preparatory injection is preferred as the second injection needle prick is avoided.

After the preparatory injections are over, the needle is then withdrawn and reinserted slowly into the crest of the papilla. The needle is advanced slowly into the incisive foramen to an extent of about 0.5 cm into the canal, and about 0.25 to 0.5 ml of local anesthetic solution is injected.

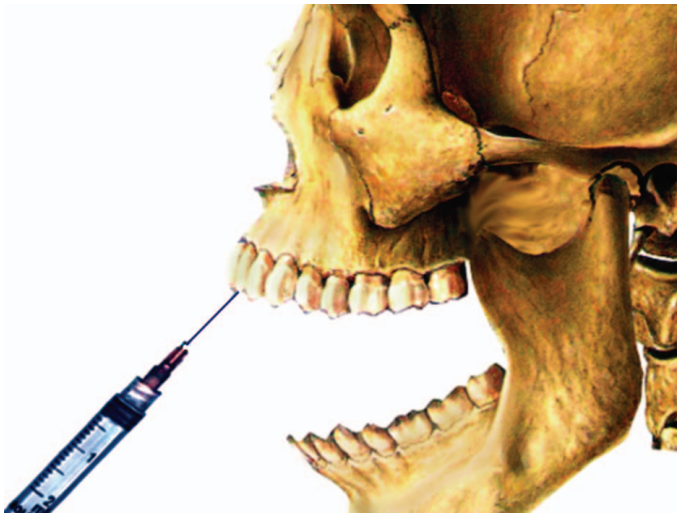


Fig. 17.19: Nasopalatine nerve block. Palatal approach. The position of the needle is for nasopalatine nerve block

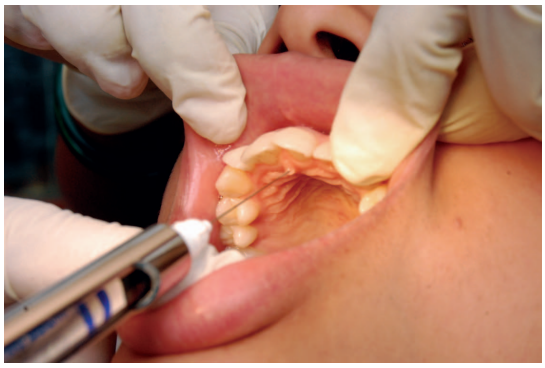


Fig. 17.20: Nasopalatine nerve block. Palatal approach. The point of the needle is in the incisive papilla as seen in the mouth.

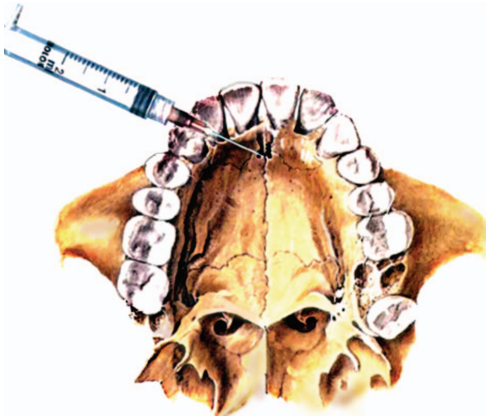


Fig. 17.21: Nasopalatine nerve block. Palatal approach. The position of the point of the needle is in the incisive foramen

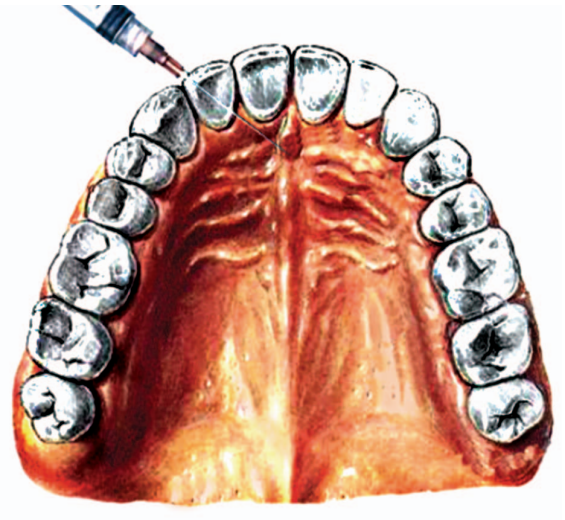


Fig. 17.22: Nasopalatine nerve block. Palatal approach. The position of the point of the needle is in the incisive papilla

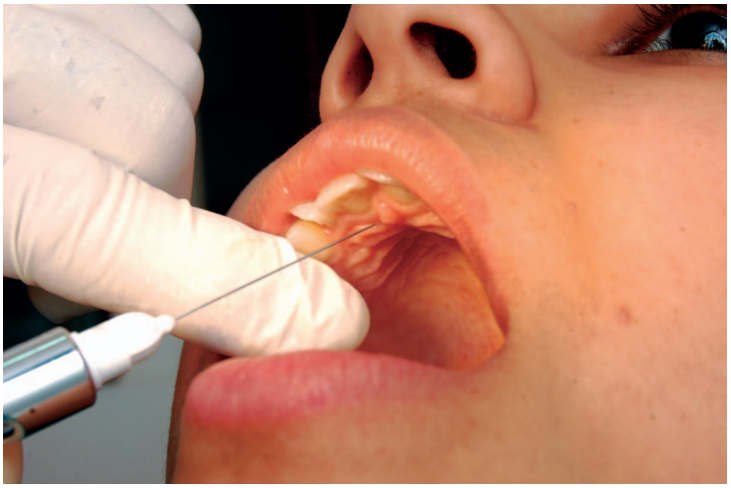


Fig. 17.23: Nasopalatine nerve block. Palatal approach.
The point of the needle is in the halo around the incisive papilla

Signs and Symptoms

1. Numbness in the anterior portion of the palate.
2. No pain during surgical procedures or dental therapy.

Complications

1. Necrosis of soft tissues is possible, if highly concentrated vasoconstrictor solution is used for hemostasis repeatedly.
2. The local anesthetic solution may "squirt" back out of the needle puncture site either during administration or after needle withdrawal, because of the density of soft tissues.

Greater Palatine Nerve Block

Other Common Names

Anterior palatine nerve block.

Nerves Anesthetised

Greater palatine nerve (anterior palatine nerve).

Areas Anesthetised

The posterior part of the hard palate and its overlying soft tissues, anteriorly as far as the canine/first premolar and medially upto the midline or the median palatine raphe.

Indications

1. For pain control during oral surgical or periodontal surgical procedures involving the palatal soft and hard tissues.
2. When palatal soft tissue anesthesia is required for restorative therapy on more than two teeth.

Contraindications

1. Presence of acute inflammation or infection at the site of injection.
2. Smaller areas of surgical procedures or restorative therapy (one or two teeth).

Advantages

1. It minimises the volume of solution to be deposited and the number of needle penetrations.
2. The technique is simple and easy.
3. Success rate is very high.

Disadvantages

1. It is a potentially painful injection technique.
2. No hemostasis. Hemostasis occurs only in the immediate area of injection. Additional infiltration will have to be given in the area of surgery for achieving hemostasis.

Anatomical Landmarks

- Greater palatine foramen
- Maxillary second and third molars
- Palatal gingival margin of second and third maxillary molars
- Median palatine raphe
- An area, approximately at a distance of 1 cm from the palatal gingival margin towards the median palatine raphe.

Technique (Figs 17.24 to 17.26)

- *Needle:* A needle of 25 or 27-gauge and 25 mm in length is recommended.
- *Point of insertion:* It is in the palatal soft tissues slightly anterior to the greater palatine foramen.

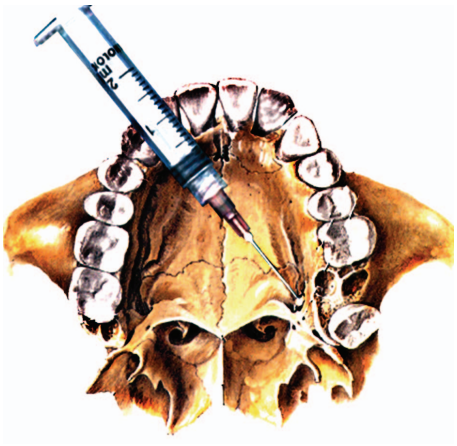


Fig. 17.24: Greater palatine nerve block. The position of the point of the needle is in relation to the greater palatine foramen

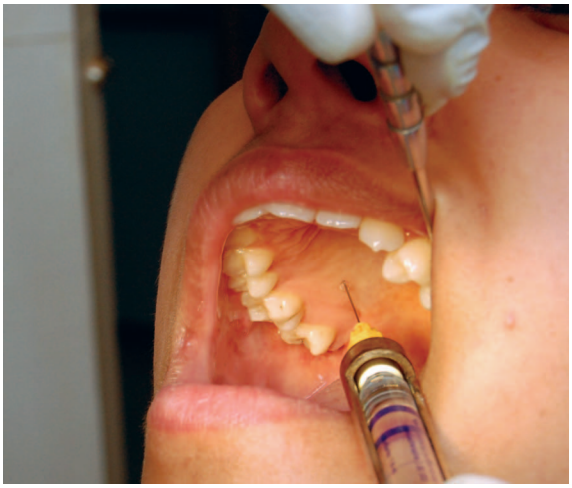


Fig. 17.25: Greater palatine nerve block. The position of the needle is for greater palatine nerve block at the apex of the palatal root of the maxillary second molar

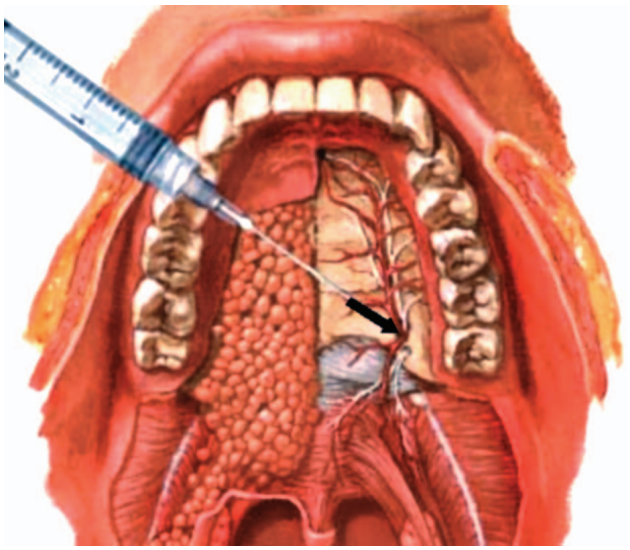


Fig. 17.26: Greater palatine nerve block. The position of the point of the needle is in relation to the greater palatine neurovascular bundle

- *Target area:* The greater palatine nerve as it comes out from the greater palatine foramen, and passes anteriorly between the palatal mucoperiosteum and the bone of the hard palate.
- *Bevel of the needle:* It is facing the palatal soft tissues.
- *Location of anatomical landmarks:* Locate the greater palatine foramen with a cotton swab which is most frequently located distal to the maxillary

second molar about 1 cm from the palatal gingival margin towards the midline.

- *Path of insertion:* The greater palatine foramen is approached from the opposite side at right angle to the curvature of the palatal bone.

Procedure

- The needle is inserted slowly until the palatal bone is contacted.
- Aspirate, to avoid inadvertent intravascular injection.
- Deposit 0.25-0.5 ml of local anesthetic solution very slowly.
- Withdraw the needle slowly and cover it with its sheath.
- The nerve may be blocked at any point along its anterior course after emergence from the foramen.

Signs and Symptoms

1. Numbness in the posterior portion of the palate.
2. No pain during dental surgical procedure.

Complications

1. *Ischemia and necrosis of soft tissues:* When highly concentrated vasoconstrictor is used for hemostasis, or if excessive amount of local anesthetic solution is used.
2. *Discomfort:* It can cause discomfort to the patient if the soft palate becomes anesthetised.
3. *Hematoma:* It is rare, as the palatal mucoperiosteum is firmly adherent to the bone of the hard palate.
4. *Failure to obtain anesthesia:*
   - o Poor injection technique: If the local anesthetic solution is deposited too far anterior or too far posterior to the greater palatine foramen.
   - o In the area of the maxillary first premolar there is overlapping of fibers from the nasopalatine nerve.

Nerve Blocks for Maxillary Nerve

A. Intraoral Maxillary Nerve Block, and
B. Extraoral Maxillary Nerve Block.

These blocks are used for achieving anesthesia of half of the maxilla.

Indications

i. Extensive oral and periodontal surgical procedures.
ii. Restorative procedures involving a quadrant of maxilla.

Intraoral Nerve Blocks

Approaches

There are two approaches:

(1) High tuberosity approach, and (2) Greater palatine canal approach.

Both the approaches are considered to be technically difficult. These should be attempted only if definitively indicated; and by experienced hands.

Major Difficulties

1. The difficulty encountered with greater palatine canal approach is in locating the canal and negotiating it completely.
2. The difficulty encountered with the high tuberosity approach is the higher incidence of hematoma. This occurs as a result of damage to the pterygoid plexus of veins with the needle.

Other Names

Second division (VII) Nerve Block

Nerves Anesthetised

Maxillary division of trigeminal nerve, and its branches.

Areas Anesthetised

The block anesthetises all the following structures on the ipsilateral side of the block.

1. The pulps of all maxillary teeth, and the buccal periodontal tissues, supporting alveolar bone, and the overlying soft tissues on the side of injection.
2. The bone of the hard palate, and part of the soft palate, maxillary sinus, and the lateral wall of nasal cavity.
3. Skin of the lower eyelid, side of the nose, cheek and the upper lip.

Indications

1. Control of pain during extensive oral surgical and periodontal surgical procedures, and restorative dental procedures to be carried out in the ipsilateral maxilla.
2. Presence of inflammation or infection at the site of injection that contraindicates the use of other regional block techniques, such as infraorbital nerve block, or posterior superior alveolar nerve block techniques.
3. Diagnostic and therapeutic procedures for trigeminal neuralgias involving the second division of trigeminal nerve.

Contraindications

1. When the administrator is inexperienced.
2. Child patients.
3. Uncooperative patients.
4. Presence of acute inflammation or infection at the site of injection.
5. Increased possibility of hemorrhage, especially in a hemophilic.

Advantages

1. The high tuberosity approach is less painful.
2. Success rate is high.
3. It minimises the number of needle penetrations.
4. It minimises the total volume of local anesthetic solution injected.

Disadvantages

1. Increased risk of hematoma, especially with high tuberosity approach.
2. The high tuberosity approach is a little arbitrary, as there is absence of bony landmarks.
3. Lack of hemostasis: There is no hemostasis at the site of surgery. It requires deposition of additional amount of local anesthetic solution at the site of surgery.
4. The greater palatine approach is painful.

Technique

i. *High tuberosity approach:*
   - o *Needle:* The recommended gauge of the needle is 25, and the length is 1 1/2 of an inch or 38-40 mm.
   - o *Bevel of the needle:* It should be facing the bone.
   - o *Point of insertion:* It is at the height of mucobuccal fold above the distal aspect of maxillary second molar tooth, as for posterior superior alveolar nerve block technique.
   - o *Depth of insertion:* 1 ¼ of an inch
   - o *Target area:* It is the maxillary nerve as it passes through the pterygopalatine fossa. It is superior and medial to the target area of posterior superior alveolar nerve block.
   - o *Anatomical landmarks:*
     - — Maxillary second molar tooth
     - — Height of mucobuccal fold above the distal aspect of the crown of maxillary second molar tooth
     - — Maxillary tuberosity
     - — Zygomatic process of maxilla or buttress of the zygoma.

- *Procedure*
  - o *Marking the length of the needle:* Mark the length of the needle to be inserted in the soft tissues (about 30 mm).

- *Position of the patient:* Supine or semisupine, the latter is preferable.
- *Position of the operator:* For the right-sided block, the operator stands on the side of the patient; and for the left-sided block, the operator stands in front of the patient.
- Request the patient to keep the mouth partially open.
- *Preparation of the tissues:* Prepare the tissues in the region of the height of mucobuccal fold above the distal aspect of maxillary second molar tooth, by application of topical antiseptic and topical anesthetic agents.
- Retract the cheek to increase visibility of the area.
- Take a preloaded syringe, and place it in the soft tissues at the height of mucobuccal fold above the distal aspect of maxillary second molar tooth.
- Advance the needle slowly in a superior, medial, and posterior direction as previously described for posterior superior alveolar nerve block, to a depth of 30 mm. At this depth the tip of the needle lies in pterygopalatine fossa in proximity to the maxillary division of trigeminal nerve.
- Aspirate and deposit about 1-1.5 ml of local anesthetic solution slowly.
- Withdraw the needle slowly.
- Wait for 3-5 minutes and commence with the oral surgical or dental procedure.

ii. *Greater palatine canal approach:*

- *Location of greater palatine foramen:* It is at the junction of horizontal and vertical processes of hard palate in the area of distal surface of the crown of maxillary second molar.
- *Needle:* The recommended gauge of the needle is 25; and the recommended length is 1½" or 38-40 mm.
- *Bevel of the needle:* It should be facing the palatal soft tissues.
- *Point of insertion:* The palatal soft tissues directly over the greater palatine foramen.
- *Palpation of the area of insertion:* Palpate the area with a slight depression directly over the greater palatine foramen with index finger. Needle is inserted into palatal mucosa in a posterolateral direction at a level of distal half of maxillary first molar.
- *Target area:* It is the maxillary nerve as it passes through the pterygopalatine fossa. The needle passes through the greater palatine canal to reach pterygopalatine fossa.
- *Anatomical landmarks:*
  - Greater palatine foramen (junction of maxillary alveolar process and palatine bone)
  - Maxillary second molar tooth
  - Palatal gingival margin in the area of this tooth
  - Median palatine raphe.

- *Procedure:*
  - o *Length:* Mark the length of the needle (30-35 mm).
  - o *Position of the patient:* The patient is positioned in such a way that the occlusal plane of maxillary teeth is at an angle of 45° to the floor, when the mouth is fully opened.
  - o *Position of the operator:* For the right-sided block, the operator stands in front of the patient, and for the left-sided block, the operator stands by the side of the patient.
  - o *Mouth:* Request the patient to keep the mouth wide open and the neck extended.
  - o *Location of the foramen:* Locate the greater palatine foramen at the distal aspect of maxillary second molar tooth.
  - o *Preparation of the tissues:* Prepare the tissues directly over the foramen with application of topical antiseptic and topical anesthetic agent.
  - o Take a preloaded syringe, and direct it into the mouth from the opposite side with the needle at an angle of 45° to the palatal bone, posteriorly, and enter the greater palatine foramen.
  - o *Bevel:* Orient the bevel of the needle against the soft tissues over the foramen.
  - o Penetrate the needle into the mucosa. Deposit a small volume of local anesthetic solution.
  - o Advance the needle slowly into the greater palatine canal to a depth of 30-35 mm. Do not attempt to force the needle against resistance. If resistance is felt, withdraw the needle slightly and change the angle slightly and advance it further into the canal. Always keep a few mm of needle outside the tissues.
  - o Aspirate and deposit about 1 ml of local anesthetic solution slowly. This will anesthetise greater palatine nerve almost immediately, and maxillary nerve later, allowing for relatively painless probing of greater palatine foramen.
  - o Withdraw the needle slowly, and keep it safe.
  - o Wait for 3-5 minutes and commence with the oral surgical or the dental procedure.

Signs and Symptoms

1. Numbness of lower eyelid, side of the nose, and upper lip.
2. Numbness in the teeth, buccal and palatal soft tissues on the side of injection.
3. Absence of pain during the procedure.

Precautions

1. *Overinsertion of the needle:* It is less likely with greater palatine canal approach and more likely with high tuberosity approach. It can be prevented by strict adherence to the protocol.

2. *Resistance to insertion of the needle:* It is found in the greater palatine canal approach. Never try to advance the needle against resistance.

Failures of Anesthesia

1. *Partial anesthesia:* It is due to underpenetration of the needle. To correct, withdraw the needle a little, and readvance the needle into the greater palatine canal up to a proper depth and deposit the local anesthetic solution.
2. Inability to negotiate the greater palatine canal. In the presence of obstruction in the canal, it is advisable to try with tuberosity approach. The greater palatine canal approach can be successful if the needle has penetrated at least 2/3rd of its length into the canal.

Complications

1. *Hematoma:* It occurs due to the injury to the maxillary artery, and injury to the pterygoid plexus of veins, via the tuberosity approach.
2. *Penetration of orbit:* It is very rare. It may occur in patients with small-sized skulls.
3. *Penetration of the nasal cavity:* The needle may penetrate the thin medial wall of the pterygopalatine fossa and thus the needle enters the nasal cavity.

Extraoral Nerve Blocks

Indications

There are various occasions, when an operator has to resort to extraoral injections. In these situations, the opening of the mouth is either very painful or impossible. These are as follows:

1. Wounds sustained due to accidents.
2. Swellings of head and neck, etc.
3. Presence of trismus due to various reasons.

Extraoral Injections

i. Are not difficult than intraoral injections
ii. The technique can be mastered easily
iii. Have easier accessibility
iv. Have easier achievement of asepsis
v. Larger areas can be anesthetised.

Infraorbital Nerve Block

The nerves and the areas anesthetised are the same as that for the intraoral infraorbital nerve injection technique.

Indications

1. When the anterior and middle posterior superior alveolar nerves are to be anesthetised; and the intraoral approach is not possible either because of presence of infection or trauma or any other reason.
2. When attempts to achieve anesthesia by the intraoral methods have been ineffective.

Anatomical Landmarks

(1) Infraorbital margin, (2) Infraorbital depression, (3) Infraorbital foramen, and (4) Pupil of the ipsilateral eye.

Technique (Figs 17.27 and 17.28)

- *Aseptic precautions:* The procedure should be carried out under strict aseptic conditions.
- *Preparation of skin:* The skin is prepared with an antiseptic.
- *Location of the infraorbital foramen:* With the help of the anatomical landmarks the foramen is located.
- *Anesthesia of the skin and the subcutaneous tissue:* It is achieved by deposition of a few drops of local anesthetic agent below the skin.
- Needle: Long or short 25-gauge needle is used.

Procedure

- It is introduced through the marked anesthetised area into infraorbital canal. The needle is inserted at an angle of about 45° through the skin medially and inferiorly to the foramen to compensate for the thickness of overlying tissues.

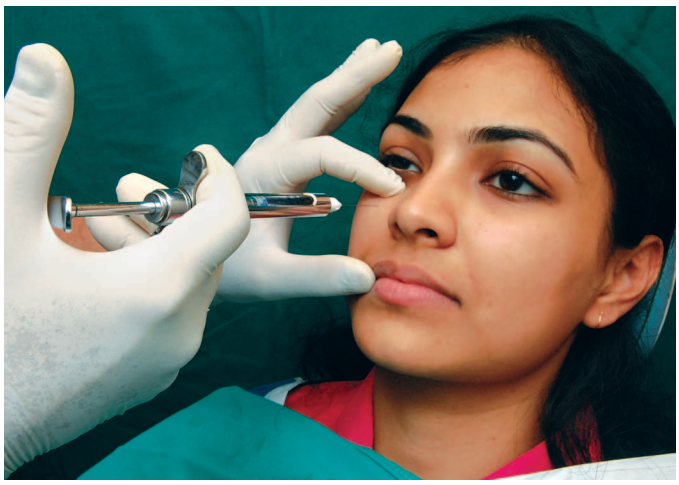


Fig. 17.27: Extraoral infraorbital nerve block technique with cartridge syringe for the right side as seen from the side

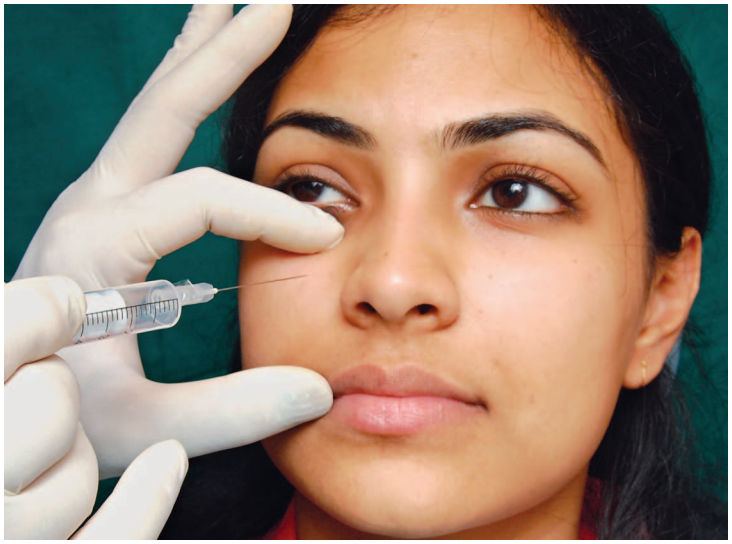


Fig. 17.28: Extraoral infraorbital nerve block technique with a hypodermic syringe for the right side

- With a slight probing action with the tip of the needle, the opening of the foramen is located. It is directed slightly upward and laterally to facilitate its entry into the foramen. Once found, needle is slowly advanced into the canal, to a depth not to exceed 1/8th, keeping in mind their orientation. The foramen and the canal, normally open downwards, forwards and medially. Incisors and canine are most easily anesthetised, as solution is injected close to anterior superior alveolar nerves.
- Carefully aspirate, and slowly deposit 1 ml of local anesthetic solution.
- Subsequently, withdraw the needle slowly, wait for about 10 minutes, and begin with the procedure.

Advantages

It is more precise; since anesthesia does not depend on diffusion of solution into the canal.

Relations

a. When the infraorbital nerve block by means of extraoral approach is being performed, the needle passes through the following structures: Skin, subcutaneous tissue, and quadratus labii superioris muscle.
b. When the needle is in final position for this injection the important structures in the vicinity of the tip of the needle are: Facial artery and vein. Since these vessels are tortuous, they may lie on either side of the needle. When the tip of the needle is in the canal, it is very close to the infraorbital nerve and vessels.

Infrequent Occurrence

There is spasm of internal maxillary artery, resulting in blanching of face over the area of distribution of lateral nasal, inferior palpebral, and superior labial arteries. This condition does not cause any discomfort to the patient, and it passes away in short time.

Care Should be Taken

(1) To advance slowly, (2) To aspirate as needle is advanced, (3) To ensure that needle remains in the confines of the canal, and (4) Aspirate and deposit 1-2 ml of the local anesthetic solution.

Signs and Symptoms

a. *Subjective:* Tingling and numbness of the lower eyelid, side of the nose and upper lip.
b. *Objective:* (1) Demonstration of absence of pain with instrumentation, and (2) No pain during the surgical procedure or the dental therapy.

Maxillary Nerve Block

Nerves Anesthetised

Maxillary nerve and all of its branches peripheral to the site of injection.

Areas Anesthetised

Anterior temporal and zygomatic regions, lower eyelid, side of the nose, upper lip, maxillary teeth, maxillary alveolar bone and overlying structures, hard palate, and part of soft palate, tonsils, part of the pharynx, nasal septum and floor of the nose and mucosa of the posterolateral part of the lateral wall of the nose and turbinate bones.

Anatomical Landmarks

- Midpoint of zygomatic arch
- Zygomatic notch
- Coronoid process of the ramus of the mandible
- Lateral pterygoid plate.

Indications

1. Where anesthesia of the entire distribution of the maxillary nerve is required for extensive surgery.
2. When it is desirable to block all the subdivisions of the maxillary nerve with only one needle insertion; and with a minimum of anesthetic solution.
3. When local infection, trauma, or other conditions make nerve blocks of the more terminal branches difficult or impossible.

4. For diagnostic or therapeutic purposes, such as tics or neuralgias of the maxillary divisions of the fifth cranial nerve.

Technique (Figs 17.29 and 17.30)

- *Asepsis:* This procedure should be carried out under strict aseptic conditions. These include preparation of the hands of the operator, including scrubbing and gloving; and surgical preparation of the field of surgery.
- *Palpation of the landmarks:* The midpoint of the zygomatic arch is located and the depression in its inferior surface is marked. The coronoid process of the ramus of the mandible is located by opening and closing the lower jaw.
- *Needle:* With a 25-gauge needle, a skin wheal is raised just below this mark in the depression, which the operator identifies by having the patient open and close the jaw.
- *Mark the needle:* Using a 4" (8.8 cm), 22-gauge needle attached to a leuerlock type of syringe, the operator measures 4.5 cm and marks with a rubber marker.
- *Insertion of the needle:* The needle is inserted through the skin wheal, perpendicular to the skin surface and to the median sagittal plane. Inject a few drops of local anesthetic solution as the needle penetrates deeper into the tissues, until the needle point gently contacts the lateral pterygoid plate. The needle should never be inserted beyond the depth of the marker.
- The needle is withdrawn, with only the point left in the tissues, and redirected in a slight forward and upward direction until the needle is inserted to the depth of the marker.

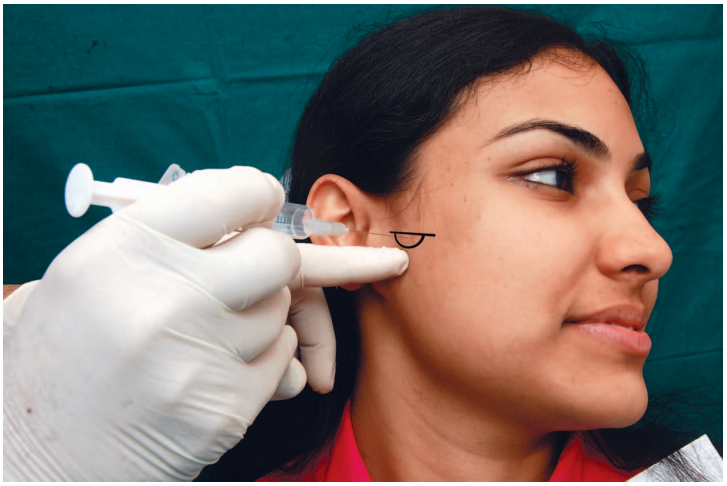


Fig. 17.29: Extraoral maxillary nerve block technique with markings as seen from the side

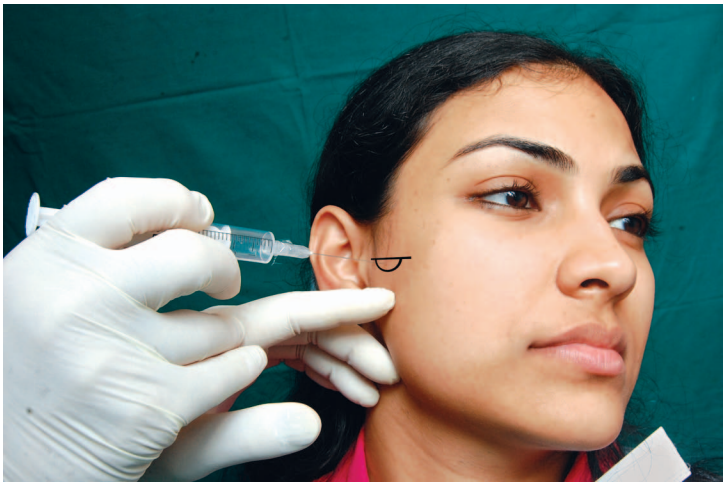


Fig. 17.30: Extraoral maxillary nerve block technique with markings as seen from the front

- After careful aspiration, 1-2 ml of local anesthetic solution is slowly injected. Care should be exercised to aspirate after each 0.5 ml of the solution is injected.

Relations

a. During the injection by means of an extraoral approach the needle passes through the following structures: Skin, subcutaneous tissue, masseter muscle, sigmoid notch, and lateral pterygoid muscle.
b. When the needle is in contact with the lateral pterygoid plate; the following structures are in its vicinity:
   i. Superiorly, the base of the skull.
   ii. Internal maxillary artery that crosses inferiorly and curves up anteriorly, entering the lower part of pterygomaxillary fissure.
   iii. Temporal vessels from the internal maxillary artery, which may lie on either side of the artery.
   iv. Superficially, transverse facial artery.
   v. Posteriorly, the mandibular nerve which passes through foramen ovale; and posterior to that, middle meningeal artery, which passes through foramen spinosum.
   vi. Anteriorly, the pterygomaxillary fissure, through which the needle may pass into pterygopalatine fossa.

Signs and Symptoms

1. *Subjective symptoms:* Tingling and numbness of upper lip, side of the nose, lower eyelid, and in some instances anesthesia of soft palate and pharynx, with gagging sensation.
2. *Objective symptoms:* Absence of pain sensation with instrumentation.

BIBLIOGRAPHY

1. Adatia AK. Effect of cytotoxic chemotherapy on dental development. J R Soc Med 1987;80:784-85.
2. Archer WH. A Manual of Dental Anesthesia: An Illustrated Guide for Students and Practitioners. 2nd ed. WB Saunders Company 1958.
3. Howe GL, and Whitehead FIH. Local Anesthesia in Dentistry. A Dental Practitioner Handbook. 3rd ed. Varghese Publishing House 1997.
4. Loetscher CA, Melton DC, and Walton RE. Injection regimen for anesthesia of maxillary first molar. J Am Dent Assoc 1988;117:337-40.
5. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
6. Nevin, Mendel, and Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1954.
7. Nevin, Mendel, and Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY: Dental Items of Interest Publishing Company, Inc 1948.
8. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. Delhi: CBS Publishers and Distributors 1990.
9. Roberts DH, and Sowray JH. Local Analgesia in Dentistry. 3rd ed. Wright, Bristol 1987.
10. Robin Woods: A Guide to the Use of Drugs in Dentistry. 12th ed.Sydney: Australian Dental Association Inc 1996.

Chapter 18 Injection Techniques for Mandibular Nerve and its Branches

INFILTRATION TECHNIQUES

The mandibular anterior teeth are anesthetised with infiltration techniques as illustrated in Basic Injection Techniques (Chapter 16) (Figs 18.1 to 18.4)

NERVE BLOCKS

(A) Intraoral, and (B) Extraoral nerve blocks.

Intraoral Nerve Blocks

Pterygomandibular Block

Other Common Names

Inferior alveolar nerve block, mandibular nerve block.

Nerves Anesthetised

a. Inferior alveolar nerve, along with its terminal branches such as incisive nerve and mental nerve

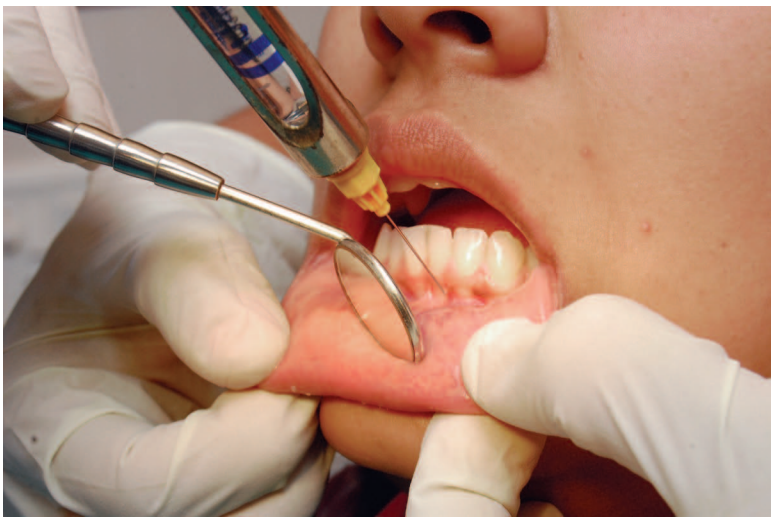


Fig. 18.1: Infiltration—Supraperiosteal injection in anterior mandible. The needle is at an angle of 45° to the long axis of the tooth

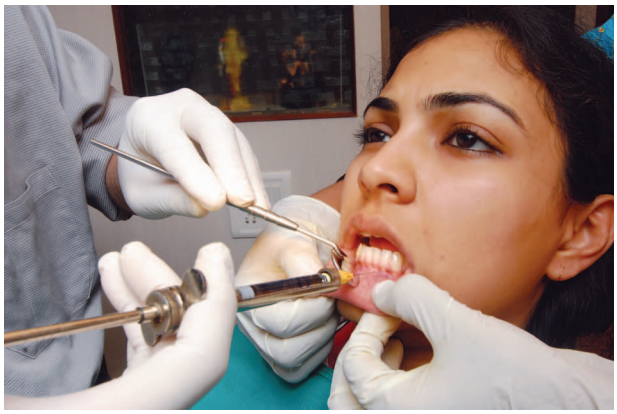


Fig. 18.2: Infiltration—Subperiosteal injection in anterior mandible. The needle is perpendicular to the long axis of the tooth and the alveolar bone as seen from the side

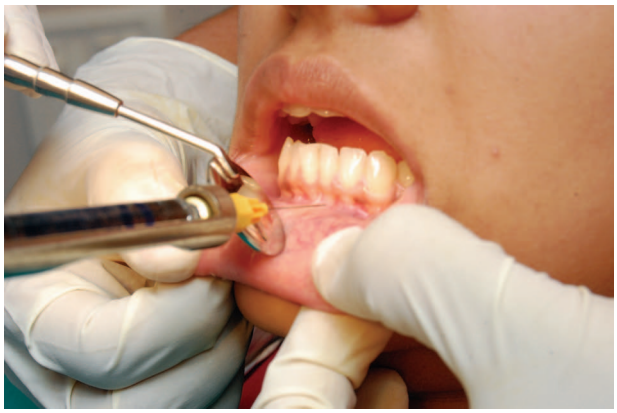


Fig. 18.3: Infiltration—Subperiosteal injection in anterior mandible. The needle is perpendicular to the long axis of the tooth and the alveolar bone as seen from the side (closer view)

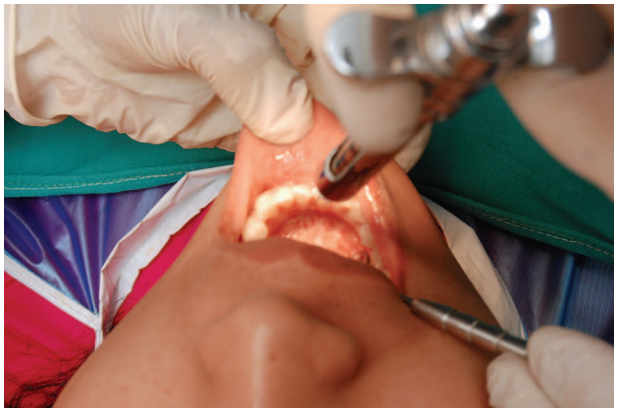


Fig. 18.4: Infiltration—Paraperiosteal injection in anterior mandible on the lingual side for anesthetizing the terminal branches of the lingual nerve as seen from above

b. Lingual nerve
c. Long buccal nerve.

Areas Anesthetised

a. Inferior alveolar nerve
    i. Pulps of all mandibular teeth from the last molar up to the central incisor in the midline
    ii. Body of the mandible
    iii. Inferior portion of the ramus of the mandible
    iv. Buccal mucoperiosteum, in the region of mandibular anteriors, anterior to mandibular second premolar or anterior to the mental foramen
    v. Skin of the chin, skin of lower lip, and mucosa of lower lip.
b. Lingual nerve
    i. Mucosa of anterior 2/3rd of the tongue, for both the general sensation as well as the special sensation (gustation; sensation of taste)
    ii. Mucosa of floor of the oral cavity
    iii. Lingual mucoperiosteum from the last molar tooth up to the central incisor in the midline
    iv. Sublingual and submandibular salivary gland.
c. Long buccal nerve
    i. Buccal mucoperiosteum in the region of mandibular molars or buccal mucoperiosteum posterior to mental foramen
    ii. Adjacent part of vestibular mucosa
    iii. Adjacent part of buccal mucosa
    iv. Mucosa of retromolar fossa.

Indications

1. Surgical procedures in the region of mandibular teeth in one quadrant.
2. When buccal soft tissue anesthesia in the region posterior to mandibular second premolar is required.
3. When lingual soft tissue anesthesia is required.
4. Restorative procedures in mandibular second premolar and molars.

Contraindications

1. Presence of acute inflammation or infection in the area of injection.
2. Patients who might bite either the lip or the tongue such as young children or mentally handicapped adults.

Anatomical Landmarks

- Mucobuccal fold in the region of premolars and molars
- External oblique ridge
- Anterior border of ramus of the mandible

- Coronoid process
- Coronoid notch
- Retromolar triangle or fossa
- Internal oblique ridge
- Pterygomandibular raphe
- Pterygomandibular space
- Sulcus mandibularis
- Occlusal plane of mandibular molars
- Contralateral premolars
- Buccal pad of fat.

The most important bony landmark in the opinion of the author is the internal oblique ridge. The needle has to be on the medial side of the ridge to approach sulcus mandibularis or the inferior alveolar nerve; and on the lateral side for the long buccal nerve.

Approximating Structures When Needle is in the Final Position

a. Superior to the following:
   - o Inferior alveolar vessels
   - o Inferior alveolar nerve
   - o Insertion of the medial pterygoid muscle
   - o Mylohyoid vessels
   - o Mylohyoid nerve

b. Anterior to the deeper lobe of the parotid gland
c. Medial to the medial surface of ramus of the mandible
d. Lateral to the following:
   - o Lingual nerve
   - o Medial pterygoid muscle
   - o Sphenomandibular ligament.

Techniques

There are two techniques practised from time immemorial (Halstead).

1. *Direct technique:* In this technique the inferior alveolar nerve is anesthetised first, hence it is known as "direct technique".
2. *Indirect technique:* In this technique the inferior alveolar is anesthetised in the third position, hence it is known as "indirect technique" or "three-positional block technique".

It is strongly recommended that the beginners should learn and master the indirect technique first; as it is easier, step by step and the chances of injury to inferior alveolar nerve are minimized. Further, in direct block technique, since the inferior alveolar nerve is anesthetised first, it is experienced that if the point of the needle is very close to or touching the nerve, the patient tends to jump because of shock-like pain and closes his mouth. This creates psychological fear for the later part of the injection

technique. However, in case of indirect block technique, having deposited local anesthetic solution, for the long buccal nerve as well as for lingual nerve, the subsequent penetration of the needle in the third position for inferior alveolar nerve is rendered painless.

Direct technique (Figs 18.5 to 18.7)

The nerves anesthetised are as follows:
1st position: The direction is from the opposite side—for inferior alveolar nerve.
2nd position: The direction is from the same side or opposite side—for lingual nerve.
3rd position: The direction is from the opposite side—to inject between the external and internal oblique ridges—for long buccal nerve with separate injection

- *Needle:* A 25-gauge long needle is recommended.
- *Target area:* The target area is the inferior alveolar nerve as it passes downward through sulcus mandibularis towards the mandibular foramen before it enters the foramen.
- *Position of the patient:* Semi-supine with mouth open; and the occlusal plane of the mandibular molars is parallel to the floor.
- *Position of the operator:* For the right inferior alveolar nerve block, the operator stands in front of the patient; and for the left inferior alveolar nerve block, the operator stands slightly behind and by the side of the patient.
- *Height of injection*
  a. Place the index finger or thumb of the left hand on the external oblique ridge or the anterior border of the ramus of the mandible.

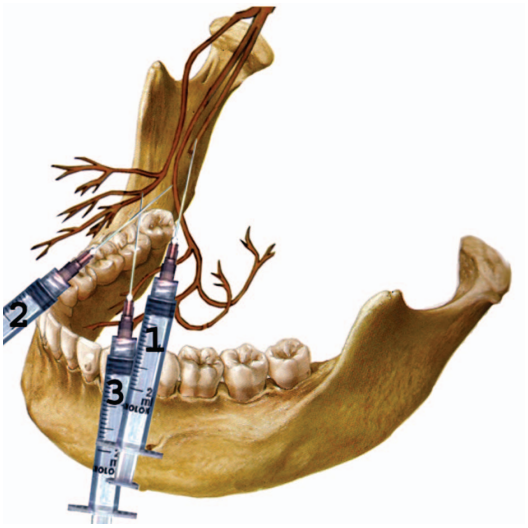


Fig. 18.5: Pterygomandibular nerve block. Direct technique. The three positions of the needle are shown: 1st position for inferior alveolar nerve, 2nd position for lingual nerve, and the 3rd position for long buccal nerve

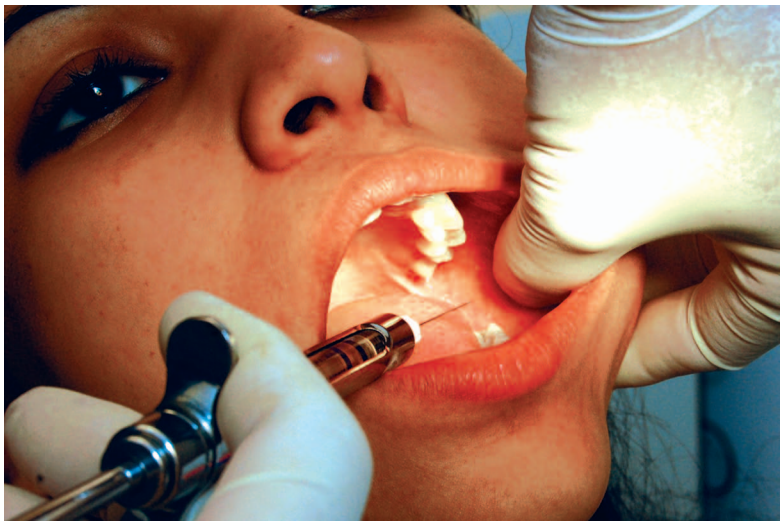


Fig. 18.6: Pterygomandibular block. Direct technique. The needle is in the first position for anesthetizing inferior alveolar nerve for the left side going from the opposite side

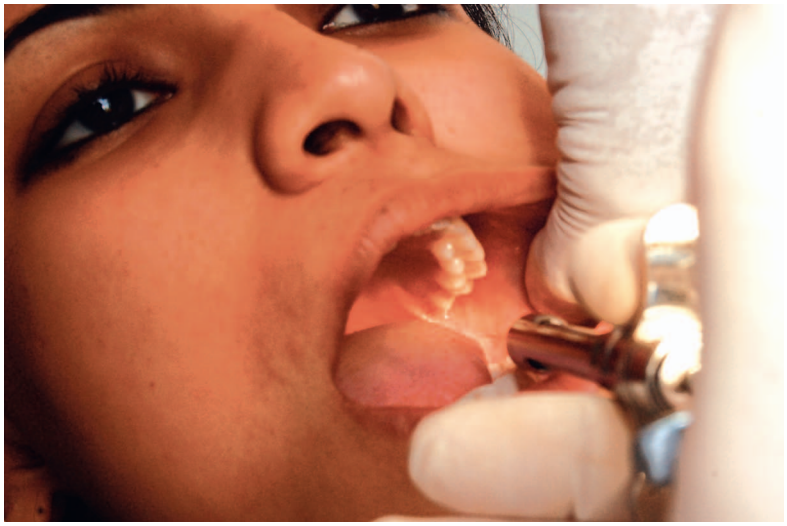


Fig. 18.7: Pterygomandibular block. Direct technique. The needle is in the second position for anesthetizing lingual nerve of the left side going from the same side

b. When the finger contacts the anterior border of the ramus, it is moved up and down until the greatest depth of the anterior border of the ramus is identified. This area is called the coronoid notch.
c. An imaginary horizontal line extends from the coronoid notch to the pterygomandibular raphe and determines the height of injection and is parallel to and 6-10 mm above the occlusal plane of mandibular molars.

- The palpating finger is then moved lingually across the retromolar triangle and onto the internal oblique ridge.
- The finger, still in line with the coronoid notch and in contact with the internal oblique ridge, is moved to the buccal side, taking with it the buccal pad of fat. This gives better exposure to the internal oblique ridge, the pterygomandibular raphe and the pterygotemporal depression.
- When palpating the intraoral landmarks with the thumb, the operator may place the index finger extraorally behind the ramus of the mandible, thus holding the mandible between the thumb and the index finger. In this manner the anteroposterior width of the ramus may be assessed.
- The depth of the needle penetration can be determined by estimating anteroposterior width of the mandibular ramus when the needle tip has been advanced half the distance between the palpating thumb and the index finger.
- The syringe and the needle is then inserted at the previously described height of insertion from the opposite mandibular premolars, at a level bisecting the finger and penetrating the tissues of the pterygomandibular space. The flaring nature of the ramus of the mandible should be kept in mind (see Fig. 18. 13).
- During insertion, the patient is asked to keep the mouth wide open. The needle is penetrated into the tissues until gently contacting bone on the medial surface of the ramus of the mandible.
- The needle is then withdrawn about 1 mm, aspiration done, to avoid intravascular administration of the local anesthetic solution, and 0.8-1.0 ml of the solution is deposited slowly.
- The needle is then withdrawn slowly and when about one-half of its inserted depth has been withdrawn, 0.5 ml of the solution is injected in this area to anesthetise the lingual nerve.
- The long buccal nerve is anesthetised with a separate insertion; and is described in the indirect technique.
- Wait for 3-5 minutes and commence with the surgical or the dental procedure.

Signs and Symptoms

1. Tingling and numbness of the lower lip on the side of injection indicates anesthesia of the mental nerve, a terminal branch of inferior alveolar nerve.
2. Tingling or numbness of one-half of the tongue on the side of injection indicates anesthesia of the lingual nerve.
3. Absence of pain during the surgical procedure or the dental therapy.

Failure of Anesthesia

1. Deposition of solution below the level of the mandibular foramen.
2. Deposition of solution too far anteriorly on the ramus.
3. Accessory innervation to the mandibular teeth, such as, cervical accessory nerve (cutaneous coli), buccal nerve, and mylohyoid nerve.
4. Anatomical aberration, such as a bifid inferior alveolar nerve, with second mandibular foramen located more inferiorly, on the medial aspect of ramus of the mandible.
5. *Cross innervation:* Incomplete anesthesia of central incisors due to innervation from the contralateral inferior alveolar nerve or mylohoid nerve.

Complications

(i) Hematoma, (ii) Trismus, (iii) Transient facial paresis.

Indirect Technique (Figs 18.8 to 18.12)

This technique of anesthetising the branches of mandibular nerve is also known as "three-positional nerve block technique".
1st position: The direction is from the opposite side—to inject between the external and internal oblique ridges—for long buccal nerve.
2nd position: The direction is from the same side—for lingual nerve.
3rd position: The direction is from the opposite side—for inferior alveolar nerve.

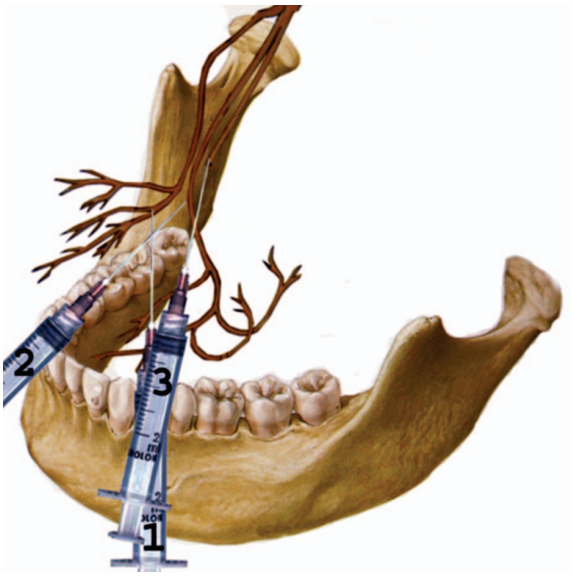


Fig. 18.8: Pterygomandibular block. Indirect technique. The three positions of the needle are shown: 1st position for long buccal nerve, 2nd position for lingual nerve, and the 3rd position for inferior alveolar nerve

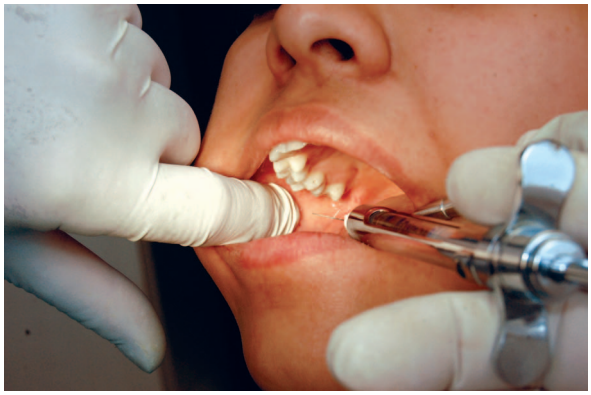


Fig. 18.9: Pterygomandibular block. Indirect technique. The needle is in the first position for anesthetizing long buccal nerve of the right side going from the opposite side

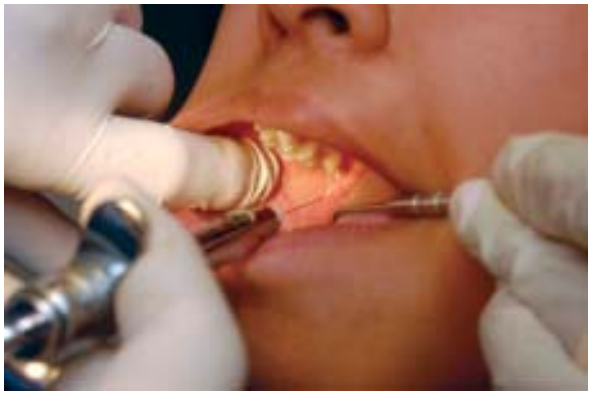


Fig. 18.10: Pterygomandibular block. Indirect technique. The needle is in the second position for anesthetizing lingual nerve of the right side going from the same side (side view)

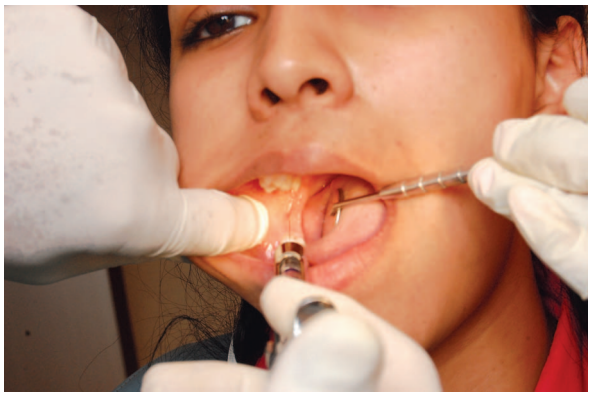


Fig. 18.11: Pterygomandibular block. Indirect technique. The needle is in the second position for anesthetizing lingual nerve on the right side as seen from the front

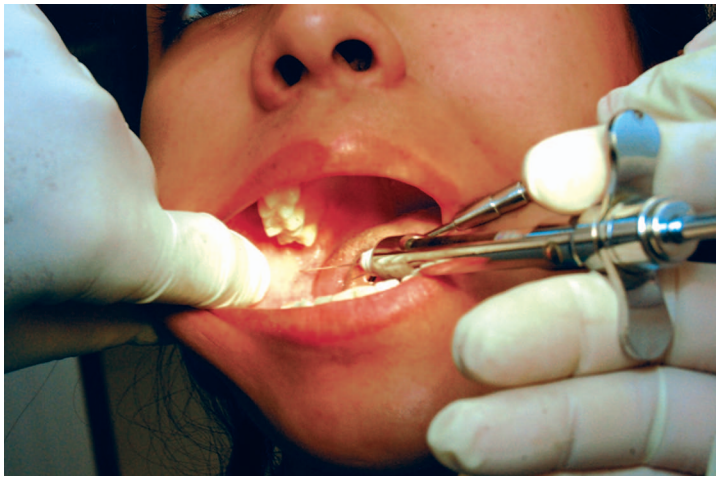


Fig. 18.12: Pterygomandibular block. Indirect technique. The needle is in the third position for anesthetizing inferior alveolar nerve of the right side going from the opposite side

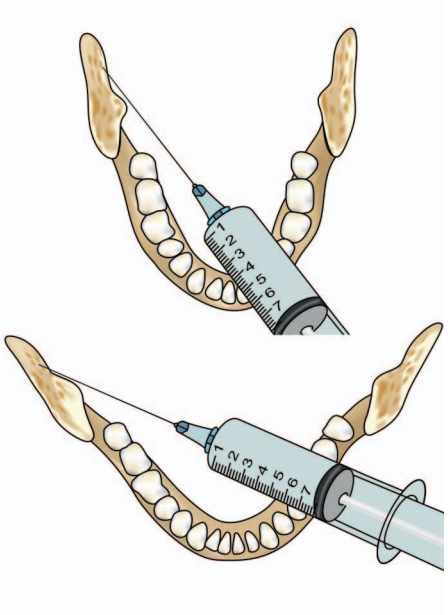


Fig. 18.13: Diagram showing flaring of the ramus dictating the position of the barrel of the syringe

1. Place the index finger in the mucobuccal fold in the region of mandibular premolars and molars.
2. Move the index finger posteriorly, until it is deflected upwards by the external oblique ridge and the ascending part of the ramus until the coronoid process is reached.

3. Keeping the ball of index finger in contact with the anterior border of the coronoid process, move the finger downward until the deepest part of the ascending ramus is located. This is known as "coronoid notch".
4. Keep the bulbous portion of the index finger in contact with the external oblique ridge. At the coronoid notch, rotate the finger so that the fingernail is turned towards the sagittal plane.
5. Maintain pressure against the coronoid notch, slide the index finger lingually. First a depression will be felt which is the retromolar triangle; and next another ridge, the internal oblique ridge.
6. Keep the distal phalanx of the index finger in contact with both the external and internal oblique ridges, and move the finger lingually for about ½" and then buccally, carrying under the distal phalanx the buccal pad of fat back to the cheek, away from the internal oblique ridge, which is partially covered.
7. When the tip of the finger rests over the internal oblique ridge, ask the patient to open the mouth as wide as possible.
8. Take a preloaded syringe with the local anesthetic solution, mounted with a 1⅝" needle, in a pen grasp, with the barrel over the contralateral bicuspid area, insert the needle in the mucous membrane at the center of the index finger nail. Keep the syringe parallel to the occlusal plane of the mandibular teeth or the mandibular ridge. Again, the flaring nature of the ramus of the mandible should be borne in mind.
9. The tissues should be penetrated in such a way that 1/4th or 1/3rd of the needle should remain outside the soft tissues. In majority of patients this will result in the tip of the needle resting directly over the sulcus mandibularis. In patients with exceptionally large or small skeleton, the width of the ramus can be measured by placing the thumb over the coronoid notch on the anterior border of the ramus and the index finger on the posterior border of the ramus outside the mouth.
10. The patient is asked to keep his mouth open wide until injection is completed. The operator must not attempt to contact bone. The operator's left index finger is held in position. Deposit local anesthetic solution slowly and watch the patient for abnormal reaction. At least 2 minutes should be taken to deposit 2 ml of solution for all the three positions (Rate of deposition: Ideal rate: 1 ml/min. Recommended rate: 1.8 ml/min).
11. Withdraw the needle slowly until approximately ¼" of the needle is in the tissues.
12. Deposit 0.5 ml of local anesthetic solution, to anesthetise the lingual nerve. Many times the long buccal nerve is also anesthetised at the same point.

13. *Long buccal nerve injection:* Hold the syringe with a 25-gauge and 1" needle, ready to inject at an angle of 45° to the body of the mandible keeping the bevel of the needle facing the bone. The tissue in the mucobuccal fold is entered just distal to the most posterior tooth or the area to be subjected to surgery. About 0.25-0.5 ml of local anesthetic solution is deposited. The anesthesia is obtained within 2-3 minutes.
14. Wait for subjective symptoms. These are:
    a. A feeling of warmth or tingling sensation in the lip, which starts at the corner of the mouth and spreads until it reaches the midline of the lip. The tingling changes into a gradually increased feeling of profound numbness; the lip may also feel swollen.
    b. The tip and side of the tongue tingle and then become numb. For profound anesthesia, the operator must wait for 5 to 10 minutes.

Long Buccal Nerve Block

The long buccal nerve is usually anesthetised as a part of pterygomandibular block in indirect technique.

Other Common Names

Buccinator nerve block, buccal nerve block.

Nerves Anesthetised

Long buccal branch of the mandibular nerve.

Areas Anesthetised

Mucoperiosteum buccal to the mandibular molar teeth, vestibular mucosa, adjacent part of buccal mucosa, and mucosa of the retromolar fossa.

Indications

When anesthesia of buccal soft tissues in the mandibular molar region is required for oral or periodontal surgical procedures.

Contraindications

Presence of acute inflammation or infection in the area of injection.

Advantages

i. High success rate
ii. Technically easy

Disadvantage

It has a potential for pain if the needle contacts periosteum during injection.

Anatomical Landmarks

- Ascending ramus of the mandible
- External oblique ridge

- Retromolar triangle
- Internal oblique ridge
- Last molar tooth.

Technique

- *Area of insertion:* It is the area of mucous membrane distal and buccal to the most distal tooth or the last molar tooth.
- *Target area:* The long buccal nerve as it crosses the anterior border of the ramus.
- *Needle:* A 1 inch 25-gauge needle is inserted into the mucosa just distal and buccal to the last molar tooth between the external and internal oblique ridges, and 0.25 to 0.5 ml of solution is deposited in this area.

Alternative Techniques

1. Insert the needle and deposit the solution directly into the retromolar triangle.
2. Insert the needle in the mucoperiosteum just buccal to the last molar tooth.

Signs and Symptoms

i. The patient rarely experiences any subjective symptoms.
ii. Lack of demonstration of pain with instrumentation in the anesthetised area.

Complications

Hematoma.

Mental Nerve Block and Incisive Nerve Block

Nerve Anesthetised

The terminal branches of inferior alveolar nerve: (i) Mental nerve, and (ii) Incisive nerve.

Areas Anesthetised

1. Labial mucous membrane anterior to the mental foramen, usually from the first premolar up to the midline.
2. Skin of the lower lip and chin.
3. Pulpal nerve fibers of the first premolars, canines and incisors.
4. Periodontium and the supporting alveolar bone of these teeth.

Indications

1. Dental restorative procedures requiring pulpal anesthesia of multiple mandibular anterior teeth.

2. When inferior alveolar nerve block is not indicated, e.g. when six or eight anterior teeth are treated, the incisive nerve block is recommended in place of bilateral inferior alveolar nerve blocks.
3. When buccal soft tissue anesthesia is required for procedures in the mandible anterior to the mental foramen, such as: (i) Soft tissue biopsies, and (ii) Suturing of soft tissues.

Contraindications

Presence of acute inflammation or infection in the area of injection.

Advantages

i. High success rate.
ii. Technically easy.
iii. Usually entirely atraumatic.
iv. Produces pulpal anesthesia, as well as soft and hard tissue anesthesia without lingual anesthesia. It is useful instead of bilateral inferior alveolar nerve blocks.

Disadvantages

1. It does not produce lingual anesthesia
2. Partial anesthesia may develop at the midline because of the overlap of the nerve fibers from those of the opposite side.

Anatomical Landmarks

Mandibular bicuspids; since the mental foramen usually lies below the apex of the second bicuspid or below and between the apices of first and second bicuspids.

Technique (Figs 18.14 and 18.15)

- The positions of the apices of the bicuspid teeth should be estimated.
- A 1 inch, 25, gauge needle is inserted into the mucobuccal fold after the cheek has been pulled laterally.
- The tissue is penetrated until the periosteum of the mandible is gently contacted slightly anterior to the apex of the second bicuspid.
- About 0.5 to 1.0 ml of local anesthetic solution is deposited in the area.

Signs and Symptoms

i. Tingling or numbness of the lower lip.
ii. Lack of pain during the surgical or dental restorative procedure.

Failure of Anesthesia

1. Inadequate volume of anesthetic solution in the mental foramen, with subsequent lack of pulpal anesthesia.
2. Inadequate diffusion of the solution into the mental foramen. To correct this, apply firm pressure over the injection site for 2 minutes, in order to force anesthetic solution into the mental foramen.

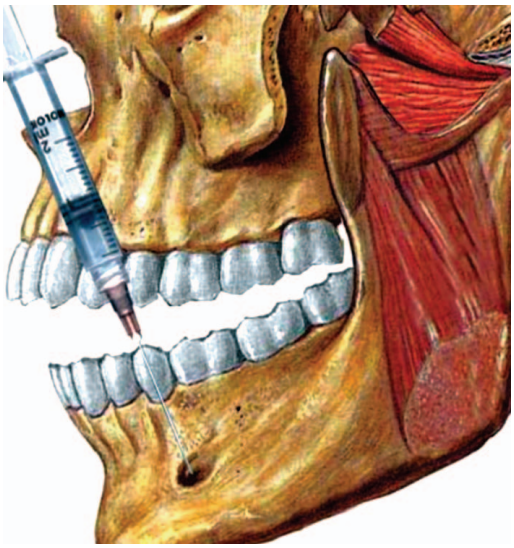


Fig. 18.14: Mental nerve block technique. The position of the needle is in the vicinity of mental nerve

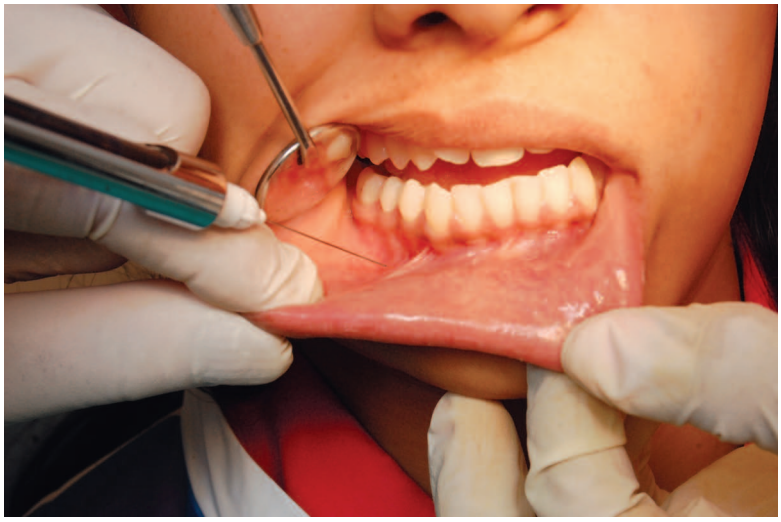


Fig. 18.15: Mental nerve block technique. The position of the needle is in between the apices of the bicuspids on the right side as seen from front

Complications

Complications are rare, with rare occurrence of hematoma.

NERVE BLOCKS FOR THE MANDIBULAR NERVE

a. Intraoral nerve blocks, and
b. Extraoral nerve blocks.

Intraoral Nerve Blocks

Gow-Gates' Mandibular Nerve Block

Nerves Anesthetised

The entire mandibular branch of trigeminal nerve is anesthetised, which includes the following: (i) inferior alveolar nerve along with its terminal branches; mental and incisive nerves, (ii) lingual, (iii) mylohyoid, (iv) auriculotemporal, and (v) long buccal nerves.

Areas Anesthetised

1. All mandibular teeth up to the midline on the side of injection
2. Buccal mucoperiosteum on the side of injection
3. Mucosa of the anterior 2/3rds of the tongue and floor of the mouth
4. Lingual mucoperiosteum from the last standing molar tooth up to the central incisor in the midline
5. Body of the mandible, and inferior portion of the ramus, etc.
6. Skin over the zygoma, posterior portion of the cheek and temporal regions, etc.

Indications

1. Surgical procedures on mandibular body and the ramus.
2. When buccal soft tissue anesthesia from the third molar up to the midline is required.
3. Surgical procedures in the tongue and the floor of the mouth.
4. When conventional inferior alveolar nerve blocks are unsuccessful.
5. Restorative procedures on multiple teeth.

Contraindications

1. Presence of infection or acute inflammation in the area of injection.
2. Patients who might bite either their lip or the tongue, such as young children and mentally challenged adults.

Anatomical Landmarks

a. Extraoral landmarks:
   - o External ear
   - o Intertragic notch of the ear
   - o Corner of the mouth.

b. Intraoral landmarks:
   - o Anterior border of the ramus of the mandible
   - o Tendon of temporalis muscle
   - o Mesiopalatal cusp of maxillary second molar.

Technique (Figs 18.16 to 18.20).

Target area: Lateral side of the condylar neck, just below the insertion of the lateral pterygoid muscle.

Procedure

- *Position of the patient:* The patient is placed in semi-supine position.
- *Position of the operator:* The operator stands in front of the patient for right-sided block; and by the side of the patient for left-sided block.
- *Identification of the landmarks:*
  - The operator visualises the landmarks and an imaginary line is drawn from the corner of the mouth to the intertragic notch of the ear.
  - The anterior border of the ramus and the coronoid process is palpated with the help of the thumb of the left hand. This helps in retraction of tissues and determination of the site of nerve penetration.
- *Configuration of the needle:* The recommended gauge and length of the needle are 25 and 40 mm respectively.

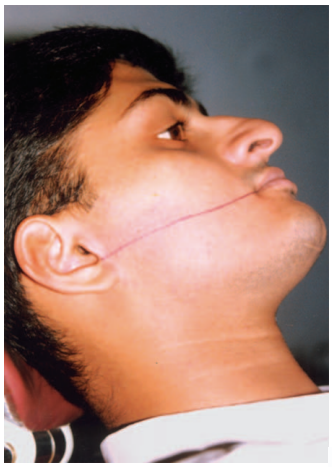


Fig. 18.16: Gow-Gates mandibular nerve block technique: Extraoral landmarks showing a line drawn from the lower border of intertragic notch to the corner of the mouth as in Gow-Gates technique

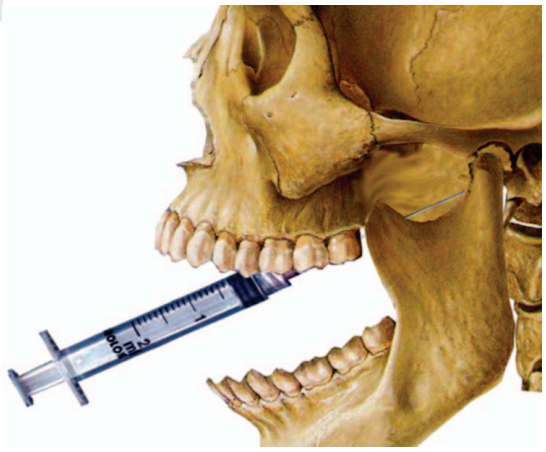


Fig. 18.17: Gow-Gates Mandibular nerve block technique: The position of the point of the needle is anteromedial to the condyle as seen from the side

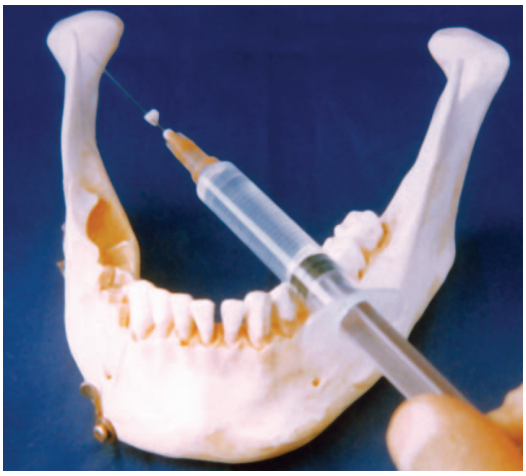


Fig. 18.18: Gow-Gates mandibular nerve block technique: Diagram showing Gow-Gates technique as demonstrated on mandible as seen from front

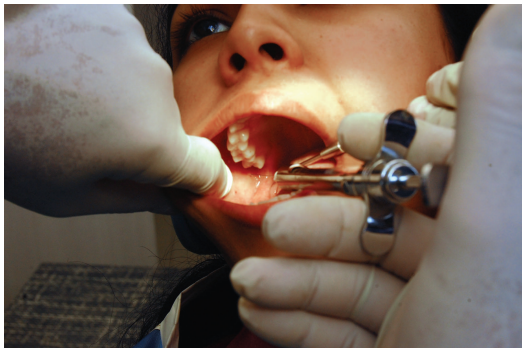


Fig. 18.19: Gow-Gates mandibular nerve block technique for the right side. The view shows the point of insertion of the needle and the retromolar area

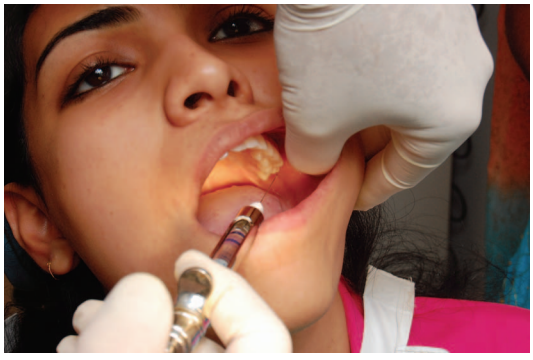


Fig. 18.20: Gow-Gates mandibular nerve block technique for the left side. The view shows the point of insertion of the needle, the retromolar and the tuberosity areas. The view of the retromolar area showing height and position of the puncture point for Gow-Gates technique in the oral cavity

The patient is advised to keep mouth widely open and to remain in that position until the injection is completed. This position moves the condyle anteriorly, thus facilitating the injection.

The operator takes a preloaded syringe and aligns the barrel of the syringe with the plane extending from the corner of the mouth to the intertragic notch directing the syringe from the corner of the mouth on the opposite side.

The needle is gently inserted into the mucous membrane just distal to the last maxillary molar tooth present, at the height of the mesiopalatal cusp of maxillary second molar. When the third molar is present, the site of penetration is distal to the third molar lateral to pterygotemporal depression and medial to the tendon of temporalis muscle.

- *Site and height of penetration: Depth of penetration:* The needle is advanced slowly until bone is contacted at the neck of the condyle. The average depth of soft tissue penetration is 25 mm.
- If bone contact is not established, the needle should be withdrawn slightly and redirected until the bone contact is made.
- If aspiration is negative, then 3 ml of local anesthetic solution is deposited slowly over 60-90 seconds.
- Withdraw the syringe and keep the needle covered.
- Ask the patient to keep the mouth open for 2-3 minutes to allow adequate diffusion of local anesthetic solution, and bathing of the nerve trunk with the solution.
- The onset of anesthesia with this technique is somewhat slower, requiring 5-7 minutes.

Signs and Symptoms

1. Numbness or tingling sensation of the lower lip
2. Numbness or tingling sensation of the tongue
3. No pain felt during surgical procedure.

Complications

1. Hematoma
2. Trismus
3. Temporary paralysis of cranial nerves II, IV and VI.
4. Failure of anesthesia:
   a. Too little volume of local anesthetic solution is deposited.
   b. Anatomical difficulties.

Akinosi (Closed Mouth) Mandibular Nerve Block

It was described by Joseph Akinosi in 1977.

Nerves Anesthetised

The entire mandibular branch of trigeminal nerve, comprising of inferior alveolar nerve along with its terminal branches; mental and incisive nerves, lingual, and mylohyoid nerves are anesthetised, except the long buccal nerve.

Areas Anesthetised

- All mandibular teeth on the side of injection up to the midline.
- Body of the mandible and inferior portion of the ramus.
- Buccal mucoperiosteum and mucous membrane in front of the mental foramen.
- Mucous membrane of the anterior 2/3rd of the tongue and floor of the oral cavity.
- Lingual soft tissues and periosteum.

Indications

1. Limited mandibular opening.
2. Multiple procedures on mandibular teeth.
3. Inability to visualise the landmarks for inferior alveolar nerve block.

Contraindications

1. Presence of acute inflammation or infection in the area of injection.
2. Patients who might bite their lip or tongue, such as young children and mentally challenged adults.
3. Inability to visualize or gain access to the lingual aspect of the ramus.

Advantages

1. Relatively atraumatic.
2. Patient need not be able to open his mouth.
3. Fewer postoperative complications (i.e. trismus).
4. Lower aspiration rate than with inferior alveolar nerve block.
5. Provides successful anesthesia where a bifid inferior alveolar nerve and bifid mandibular canals are present.

Disadvantages

1. Difficult to visualise the path of the needle and the depth of insertion.
2. No bony contact, so the depth of penetration is somewhat arbitrary.
3. Potentially painful if the needle is too close to periosteum.

Anatomical Landmarks

1. Occlusal plane of teeth in occlusion.
2. Mucogingival junction of maxillary molar teeth.
3. Anterior border of ramus of the mandible.
4. Maxillary tuberosity.

Technique (Figs 18.21 and 18.22)

- *Needle:* The recommended length is 1½" or 38-40 mm, and the gauge is 25.
- *Bevel:* The position of the bevel of the needle in the closed mouth mandibular block is very significant. It must be facing away from the bone of mandibular ramus and towards the midline.
- *Height of injection:* With Akinosi's technique it is below that of Gow-Gates' technique but above that of inferior alveolar nerve block.
- *Target area:* The soft tissues on the medial border of ramus of the mandible in the region of inferior alveolar nerve as it travels towards the mandibular foramen, lingual nerve, and mylohyoid nerves and vessels.

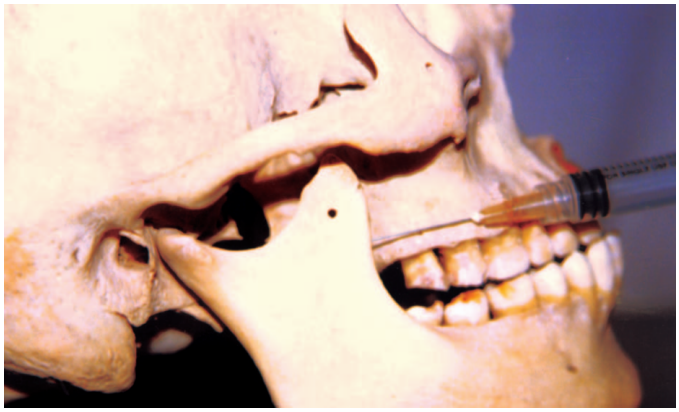


Fig. 18.21: Akinosi technique demonstrated on the mandible—The photograph shows the syringe and the needle are parallel to the occlusal plane of the maxillary teeth at the level of maxillary mucogingival junction

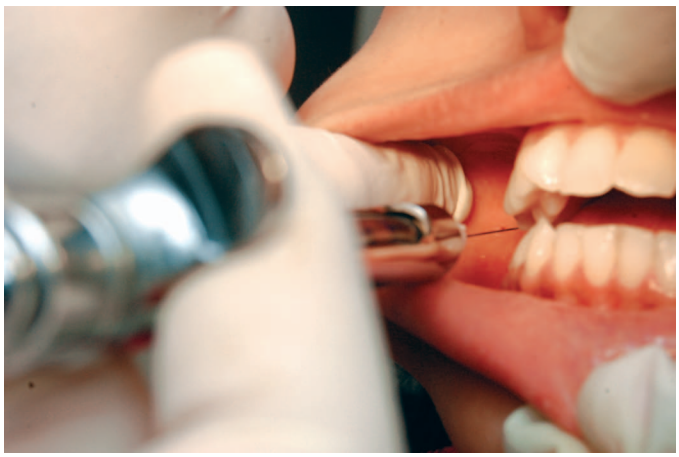


Fig. 18.22: Akinosi technique demonstrated clinically in the oral cavity. The mouth opening is inadequate

Procedure

- *Position of the patient:* The patient is seated in semireclining position with head, neck and shoulder adequately supported.
- *Position of the operator:* The operator stands in front of the patient for both right-sided as well as left-sided block.
- *Preparation of the tissues:* The site of penetration is prepared by topical application of antiseptic and anesthetic solutions.
- The patient is asked to bring teeth in occlusion. This aids in relaxation of cheek musculature and helps in good visualization of the landmarks. The operator retracts the patient's lips and cheek exposing the maxillary and the mandibular teeth on the ipsilateral side.
- The preloaded syringe with the recommended needle is taken and the barrel of the syringe is aligned parallel to the occlusal and sagittal plane but positioned at the level of the mucogingival junction of the maxillary molars.
- The needle penetrates the mucosa in the embrasure just medial to the ramus lateral to maxillary tuberosity and is inserted approximately 1½" or 25-30 mm. The tip of the needle lies in the target area in the midportion of pterygomandibular space, close to the branches of mandibular nerve.
- Following negative aspiration, about 2 ml of local anesthetic solution is slowly deposited approximately 1 minute.
- Motor nerves paralysis will develop as quickly or more quickly than sensory anesthesia. The patient with trismus will begin to notice increased ability to open the jaws shortly after the deposition of local anesthetic solution.
- Anesthesia of the lips and tongue will be noticed in 40-90 seconds and the surgical procedures can be usually started within 5 minutes

Signs and Symptoms

Same as of Gow-Gates technique (Table 18.1).

Failure of Anesthesia

1. Failure to appreciate the flaring nature of the ramus which deflects the needle more medially if, internal oblique ridge is not negotiated by keeping the syringe nearly at an angle of 90° (perpendicular) to the medial surface of ascending ramus. This can be easily achieved by retracting the angle of the mouth posteriorly with the barrel of the syringe.
2. Point of needle insertion is too low.
3. Underinsertion or overinsertion of the needle as no bone is contacted in this technique, the depth of soft tissue penetration is somewhat arbitrary. Akinosi recommended a penetration depth of 25 mm in the average sized adult measuring from the maxillary tuberosity.

Complications

1. Hematoma, rarely.
2. Trismus, rarely.
3. Transient facial nerve paresis due to overinsertion of the needle and deposition of the solution into the body of the parotid gland, near the posterior border of the ramus of the mandible.

Table 18.1: Comparison of the various injection techniques

	Conventional pterygomandibular technique	***Gow-Gates' technique***	***Akinosi technique***
Nerves anesthetised	Inferior alveolar, lingual, and long buccal nerves	Mandibular nerve	Mandibular nerve
Onset of anesthesia	Rapid: Usually 2-3 minutes	Slow	Slow
Depth of anesthesia	Adequate	Excellent	Adequate
Amount of solution required	2 ml	3 ml	2 ml
Incidence of inadvertent intravascular administration (Positive aspiration)	High; hence need for aspiration	Less	Less
Need for additional injection	For long buccal nerve in direct technique	Single injection Only one injection is required to anesthetise all the three nerves	Single injection Additional injection may be required for long buccal nerve
Complications	Trismus, and facial paresis may occur	Decreased incidence of trismus	Less incidence of trismus
Lack of achieving complete anesthesia	Sometimes encountered in mandibular teeth	Rarely encountered	Lower frequency of successful anesthesia due to absence of bony landmarks
Advantages			Can be used in closed mouth
Pain at the time of injection			Less painful

Extraoral Techniques for Anesthesia

Anesthetic Technique for Mandibular Nerve

Nerves Anesthetised

Mandibular nerve and its subdivisions; composed of branches from the anterior trunk and branches from the posterior trunk.

Areas Anesthetised

The entire region innervated by mandibular nerve and its subdivisions.

Temporal region, auricle of the ear, external auditory meatus, temporomandibular joint, salivary glands, anterior 2/3rd of the tongue, floor of the mouth, mandibular teeth, gingivae, buccal mucosa, lower portion of the face (except the angle of the jaw).

Indications

1. Presence of acute inflammation or infection at the site of injection for the subdivisions of mandibular nerve.
2. Presence of trauma that would contraindicate or make it difficult or impossible to anesthetise the subdivisions of mandibular nerve.
3. Whenever there is need to anesthetise the entire mandibular nerve and its subdivisions, with one single penetration and minimum of local anesthetic solution for extensive surgical procedures.
4. For diagnostic and therapeutic purposes.

Anatomical Landmarks

These are common to those for extraoral maxillary nerve block; and are as follows:

- Midpoint of zygomatic arch.
- Coronoid process of the ramus of the mandible; and prominence of the lateral pole of the condyle; which is located by having the patient open and close his mouth.
- Lateral pterygoid plate.

Technique (Figs 18.23)

The technique for mandibular nerve block is essentially the same as that for maxillary nerve block. The difference is that the marker is placed on the needle at a distance of 5 cm.

The needle contacts the lateral pterygoid plate, then it is withdrawn exactly in the same way as in the maxillary nerve block; however, when it is reinserted, the needle is directed upward and slightly posteriorly; in order for the needle to pass posterior to lateral pterygoid plate. The needle should not be introduced to a depth greater than measured 5 cm.

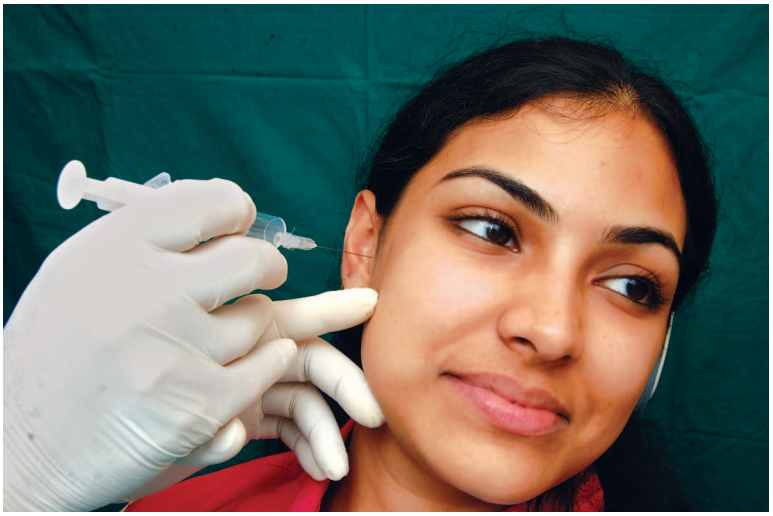


Fig. 18.23: Extraoral mandibular nerve block technique as seen from the front

Structures

The structures through which the needle passes and the structures adjacent to the needle when it is in contact with the lateral pterygoid plate are:

a. Structures through which the needle passes: Skin, subcutaneous tissue, masseter muscle, sigmoid notch, lateral pterygoid muscle.
b. Structures in the vicinity of the needle when the needle is in contact with lateral pterygoid plate.
   - o *Superiorly:* Base of the skull.
   - o Internal maxillary artery; as it crosses inferiorly and curves upwards anterior to it, entering the lower part of pterygomaxillary fissure.
   - o Temporal vessels for internal maxillary artery that may lie on either side of it.
   - o *Superficially:* The transverse facial artery which may lie above or below it.
   - o *Posteriorly:* Foramen ovale and posterior to it foramen spinosum.
   - o *Anteriorly:* Pterygomaxillary fissure through which the needle may pass into pterygopalatine fossa.

Signs and Symptoms

a. *Subjective:* Tingling sensation and numbness of lower lip and anterior 2/3rd of the tongue.
b. *Objective:*
   - i. Demonstration of difference in feeling of lower teeth while opening and closing the jaws.
   - ii. Lack of demonstration of pain with instrumentation.
   - iii. Absence of pain during surgical procedure.

Complications

1. Failure of anesthesia, and
2. Trismus.

BIBLIOGRAPHY

1. Akinosi JO. A new approach to the mandibular nerve block. BJ Oral Surg 1977;15:83-87.
2. Archer WH. A Manual of Dental Anesthesia: An illustrated guide for students and Practitioners. 2nd ed. WB Saunders Company 1958.
3. Gow-Gates GAE. Mandibular conduction anesthesia: A new technique using extraoral landmarks. Oral Surg 1973;36:321-28.
4. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
5. Nevin, Mendel, Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY: Dental Items of Interest Publishing Company, Inc 1954.
6. Nevin, Mendel, Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY: Dental Items of Interest Publishing Company, Inc 1948.
7. Patil SG, AP Chitre. Comparative evaluation and clinical assessment of mandibular nerve anesthesia by conventional technique, Gow-Gates technique and Akinosi technique; a dissertation, at KLES's Institute of Dental Sciences, Belgaum, Karnataka, under Rajiv Gandhi University of Health Sciences, Bangalore, Karnataka, in 1999.
8. Richard Bennett C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed. New Delhi: CBS Publishers and Distributors 1990.
9. Roberts DH, Sowray JH. Local Analgesia in Dentistry. 3rd ed. Wright, Bristol 1987.

ELECTRONIC DENTAL ANESTHESIA (EDA)

Mechanism of Action of EDA

The mechanism by which EDA acts for prevention of acute pain during any surgical or dental procedure, is adequately explained by the Gate-Control theory, proposed by Melzack and Wall in 1965.

EDA is used at a higher frequency (> 120 Hz). At this frequency, EDA causes the patient to experience a special sensation. This involves stimulation of larger diameter nerves (A fibers), which transmit the sensations of touch, pressure, and temperature.

If the patient can maintain a minimum "threshold" intensity of A fiber stimulation; then the pain impulse produced by the dental or surgical instruments, such as high-speed drill, blade, or curette that is transmitted to central nervous system more slowly along the smaller A-δ and C fibers, will come on a "closed gate". Hence, it will be unable to reach the brain, where it is translated into physical pain. Thus, the large-fiber input is said to inhibit central transmission of the overall effects of small-fiber input. The pain impulse failing to reach the brain, the pain sensation does not occur.

The blood levels of serotonin and endorphins are elevated during high frequency stimulation. These hormones probably play a secondary, but an important role, in providing acute pain control during most types of dental procedures.

Indications

Main Indication: Needle Phobia (Fear of Injection)

Patients who are fearful of needles, as injections are required in all procedures of dentistry.

Other Indications

i. *Ineffective local Anesthesia:* In cases, where local Anesthesia fails to act, due to any reason.

ii. *History of allergy to local Anesthetic agents:* In cases of true or documented allergy to local Anesthetic agents, these agents cannot be administered.

Contraindications

1. Cardiac pacemakers
2. Neurological disorders
   a. Post-cerebrovascular accident (CVA) (stroke)
   b. History of transient ischemic attacks
   c. History of epilepsy
3. Pregnancy
4. *Lack of maturity:* Inability to understand the concept of pain control
   a. Very young patient
   b. Very old patients with senile dementia
   c. Language communication difficulties.

Dental Procedures

The various dental procedures wherein EDA can be successful are:

1. Chronic pain in cases of TMJ, or Myofascial Pain Dysfunction (MFPD) syndrome
2. Administration of local anesthesia
3. Non-surgical periodontal procedures
4. Restorative dentistry
5. Fixed prosthodontic procedures
6. Endodontics.

Uses in Dentistry

Most Important Use

The most important use of this technique in dentistry, is that EDA is an effective method of minimizing patient discomfort during the injection of local Anesthetic agents.

However, proper application of topical anesthesic agent, and following the basic principles of injection technique such as slow injection of local anesthetic solution, minimizes patient's perception to a great extent.

Other Uses: Post-surgical Pain

EDA can also be used after surgical procedures. It can minimize post-surgical pain after surgical procedures.

Application

The use of EDA at a low frequency setting for 30-60 minutes at the completion of surgery provides the comfortable postoperative recovery from local anesthesia.

Advantages

The advantages of using EDA over Local anesthesia achieved following injectable local anesthetic agents are as follows:

1. No need for further needle pricks
2. No need for injection of drugs
3. Patient is in control of anesthesia
4. No residual anesthetic effect at the end of the procedure
5. Residual analgesic effect remains for several hours.

Disadvantages

1. Cost of the equipment
2. Training of the personnel
3. Intraoral electrodes: The intraoral electrodes offer a weak link in the entire system. However, a greater depth of anesthesia is obtained with intraoral electrodes. Also, in some units, extraoral electrodes are available.

TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS)

It is a treatment modality that has considerable utility in the management of pain. It is most often used in the management of chronic pain.

Mechanism of Action

It is used at a low-frequency of 2 Hz (hertz or cycles per second). TENS produces considerable changes in the blood levels of l-tryptophan, serotonin, and β-endorphins. l-tryptophan, a precursor of serotonin, is present in the blood in decreasing amounts as the duration of TENS increases. By contrast, serotonin levels in the blood increase with time (Silverstone 1989).

Serotonin possesses analgesic actions, elevating the threshold of pain reaction. At the same time, levels of β-endorphins and eukaphelins in the cerebral circulation also increase. β-endorphins and eukaphelins are potent analgesics produced by the body in response to certain types of stimulation (Hughes, Smith, and Kosterlitz, 1975).

Uses

Use in Chronic Pain

The elevated blood levels of these chemical agents are not achieved for a period of about 10 minutes after the start of TENS or EDA stimulation. Because of this mechanism, TENS may aid in the management of chronic pain.

Residual Effect

The blood levels of serotonin and β-endorphins remain elevated for several hours after the termination of TENS therapy. Because of these elevated blood levels patients have residual analgesic action in the immediate post-treatment period. Hence, the opioid-agonist analgesics which are usually prescribed for post-treatment pain, are not necessary when TENS or EDA has been used during or after treatment.

TENS in Dentistry

TENS has been used with great success in the management of chronic pain of dental origin such as TMJ problems and Myofascial Pain Dysfunction (MFPD) syndrome for many years. Meizels, (1987), Clark et al (1987), and Geissler and McPhee (1986), have reported the ease and efficacy of using this technique for TMJ and MFPD syndromes. EDA has been successfully used (low-frequency extra-oral stimulation of the area) in the management of limited mandibular opening secondary to TMJ problems.

Clark and associates (Clark et al, 1987), Christensen (1987), and others have reported significant success rates (increased range of motion and decreased discomfort) in their cases.

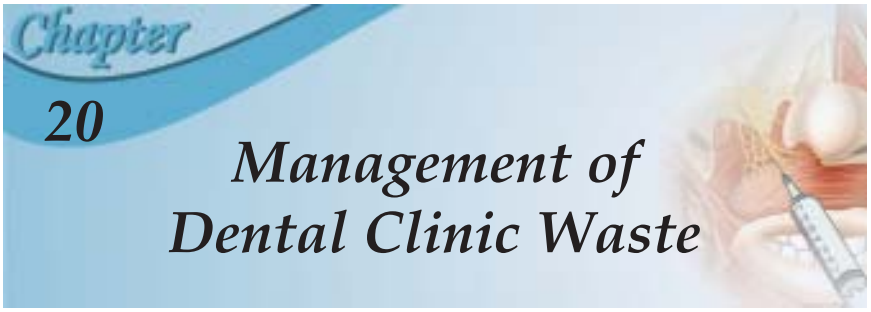
TENS in Sports Medicine

The application of a low-frequency electric current to an area, recently injured, can be of benefit in two ways:

1. *Hastens recovery process:* There is an increase in tissue perfusion produced by capillary and arteriolar dilatation. In addition, by stimulating, it results in contraction of skeletal muscles. It helps in decreasing edema, and clearing the tissue-breakdown products from the area of injury. In this way, TENS speeds recovery process.
2. *Possesses analgesic activity:* Low-frequency stimulation for longer than 10 minutes produces elevated blood levels of serotonin and endorphins. These increased levels for several hours after termination of TENS, helps in breaking the pain cycle. This analgesic action is helpful during the recovery from the injury.

BIBLIOGRAPHY

1. Christensen GJ. Electronic anesthesia: Research and thoughts. J Calif Dent Assoc 1987;15: 46-48.
2. Clark MS, et al. An evaluation of the clinical analgesia / anesthesia efficacy on acute pain using the high frequency neural modulation in various dental settings. Oral Surg 1987;63:501-05.
3. Geissler PR, and McPhee PM. Electrostimulation in the treatment of pain in the mandibular dysfunction syndrome. J Dent 1986;14:62-64.
4. Hughes J, Smith TW, and Kosterlitz HW. Identification of two related pentapeptides from the brain with potent opiate agonist activity. Nature 1975;258:577-80.
5. Malamed SF. Handbook of local anesthesia. 5th ed. Mosby (Elsevier) 2004.
6. Meizels P. H-wave and TMJ treatment. J Calif Dent Assoc 1987;15:42-44.
7. Melzack R and Wall PD. Pain mechanism: A new theory. Science 1965;150:971-79.
8. Silverstone, L. Electronic dental anesthesia. Dent Pract 1989;27:4-6.



Management of Dental Clinic Waste

WASTE DISPOSAL IN A HEALTH CARE SETTING

WHO Classification

WHO classification of waste is as follows:

i. General non-hazardous,
ii. Sharps,
iii. Chemical and pharmaceutical,
iv. Infectious, and
v. Other hazardous medical wastes.

Segregation

Segregation of waste allows sorting out of different categories of waste and placing them in different containers or bags. The containers or the bags should be labeled, bearing the international biohazard symbol. Segregation of different wastes should be done on the basis of classification; and as per the guidelines of the local Municipal Corporation, or as instructed by the local authority. All the waste, after segregation, must be stored in color-coded containers. The following is the color coding prescribed by Biomedical Waste (Management and Handling) Rules, 1998; Annexure 1. (Tables 20.1 to 20.3)

The Government of India has promulgated the Biomedical Waste (Management and Handling) Rules 1998. They are applicable to all persons who generate, collect, receive, store, transport, treat, dispose or handle biomedical wastes. This includes hospitals, nursing homes, clinics (medical and dental), dispensaries, veterinary institutions, animal houses, pathological laboratories and blood banks.

Table 20.1: The color-coding to be employed in waste disposal

Color-coding	*Type of container*	*Waste category*
Yellow	Plastic bag	*Human anatomical waste:* Human tissues, organs, body parts, teeth, etc. *Animal waste:* Animal tissues, organs, body parts used for research *Liquid:* Wastes generated from cleaning and disinfecting activities
Red	Disinfected container/ Plastic bag	*Solid:* Dressings, soiled plastercasts, gloves, gauze, cotton, dressings, linen, gloves, used impression material, components of intraoral periapical films, infected or pathological oral tissue *Solid:* Tubings, catheters, etc.
Blue/White Translucent	Plastic bag/Puncture-proof container (Hypochlorite 1%)	*Sharps:* Needles, syringes, blades, glass, wires, etc.
Black (Non-biological)	Plastic bag	Discarded medicines and chemicals used in disinfection (dry and wet)

Table 20. 2: Classification of waste

Category	*Type of waste*
Category 1	Human anatomical waste (human tissues, organs, body parts, teeth)
Category 2	Animal waste (animal tissues, organs, body parts, carcasses, bleeding parts, blood and experimental animals used in research)
Category 3	Microbiology and biotechnology waste (waste from lab culture, specimens from microorganisms, vaccines, cell cultures, toxins, dishes, devices used to transfer cultures)
Category 4	Waste Sharps (needles, syringes, scalpels, blades, glass)
Category 5	Discarded medicines and cytotoxic drugs (outdated, contaminated, discarded drugs)
Category 6	Soiled waste: Contaminated with blood and body fluids including gauze, cotton, dressings, plaster casts, linen, gloves, used impression material, components of intraoral periapical films, infected or pathological oral tissue
Category 7	Solid waste (tubes, catheters, IV sets)
Category 8	Liquid waste (waste generated from laboratory and washing, cleaning, disinfection)
Category 9	Incineration ash
Category 10	Chemical waste

Table 20.3: Color coding of the containers for waste management

Color-coding	*Type of container*	*Waste category*
Yellow	Plastic bags	Human and animal wastes (teeth), Microbial and Biological wastes and Soiled wastes (Cat 1,2,3 and 6)
Red	Disinfected container/Plastic bags	Microbiological and Biological wastes, Soiled wastes, Solid wastes (Cat 3,6 and 7)
Blue/ White/ Transparent	Plastic bag, puncture proof container	Waste sharps and Solid waste (Cat 4 and 7)
Black	Plastic bag	Discarded medicines, Cytotoxic drugs, Incineration ash and Chemical waste (Cat 5,9 and 10)
Green	Plastic container	General waste such as office waste, food waste and garden waste

Disposal of Dental Clinic Waste (Figs 20.1 to 20.8)

Fig. 20.1: Biohazard bags
Red plastic

Fig. 20.2: Handi-Hopper
- Small plastic waste receptacle for disposal of contaminated waste, mounted at chairside

Fig. 20.3: Plastic refuse liners

Fig. 20.4: Isolyser SMS sharps management system

Fig. 20.5: Sharpsafety

Fig. 20.6: Sharps collector

Fig. 20.7: Sharps container

Fig. 20.8: Needle and syringe cutter and burner: An extremely versatile apparatus for incinerating the injecting needles and to cut the syringe, used in medical, dental surgery and surgical clinic. Health units, any where needles represent a likely risk of inflection. The units in housed in an elegant casing and is a highly functional. Very efficient in operation.
Specification: Voltage: 230 VAC at 50 Hz, Weight: 1.2 kg, Dimension: 180 × 163 × 132 mm

i. *Non-infective waste:* It is to be collected in thick polythene bags and discarded like household waste.
ii. *Sharp infected dental clinic waste:* Needles and syringes should be disinfected and destroyed mechanically before disposal. The needles are cut and burnt and collected in a special container, and subsequently transferred in a puncture resistant container, or plastic containers containing 1% sodium hypochlorite, which can be closed or sealed (Fig. 20.8).
iii. *Infected dental clinic waste:* The infected waste should be collected in containers and then disposed off as per the guidelines of the local Municipal Corporation.

Section

6

Complications of Local Anesthesia and their Management

- Local Complications
- Systemic Complications of Anesthesia and their Management
- National AIDS Control Organization (NACO)

The complications arising from the use of local anesthetic agents are classified as in Table 21.1:

Table 21.1: Complications of local anesthesia

A. Complications arising from drugs or chemicals used for local anesthesia
   1. Soft tissue injury
   2. Sloughing of tissues (Tissue ischemia and necrosis)

B. Complications arising from injection techniques
   1. Breakage of anesthetic cartridge
   2. Breakage of needle
   3. Needle-stick injuries
   4. Hematoma
   5. Failure to obtain local anesthesia

C. Complications arising from both
   1. Pain on injection
   2. Burning on injection
   3. Infection
   4. Trismus
   5. Edema
   6. Mucosal blanching
   7. Persistent paresthesia or anesthesia
   8. Persistent or prolonged pain
   9. Post-injection herpetic lesions or post-anesthetic intraoral lesions
   10. Bizarre neurological complications
      a. Facial nerve paresis or paralysis
      b. Visual disturbances
         i. Diplopia, or double vision
         ii. Amaurosis or temporary blindness
         iii. Permanent blindness.

COMPLICATIONS ARISING FROM THE DRUGS OR CHEMICALS USED FOR LOCAL ANESTHESIA

Soft Tissue Injury

Causes

It is seen in the form of self-inflicted trauma to lips, tongue and cheek. It is common in children and mentally retarded adults.

Prevention

1. Use minimum effective dose of local anesthetic agent
2. Warn the patient's guardians or parents against biting of lips and tongue; and also against eating and drinking hot fluids; while the effect of anesthesia is still present.

Management

1. Symptomatic: It comprises of analgesics, and if necessary antibiotics.
2. Topical application of petroleum jelly, or local anesthetic or antibiotic cream.

Sloughing of Tissues (Tissue Ischemia and Necrosis)

Various Forms

i. Epithelial desquamation or ulceration.
ii. Sterile abscess.
iii. Tissue necrosis or sloughing.

Causes

1. Predisposition: Commonly seen in hard palate, as in the region of distribution of nasopalatine and greater palatine nerves, because the mucoperiosteum is firmly attached to the bone. It occurs at the site of injection. Necrosis leads to painful ulceration and sloughing.
2. Deposition of excessive volume of local anesthetic agent with a vasoconstrictor.
3. Rapid deposition of the local anesthetic solution with undue pressure.
4. Application of topical local anesthetic agent for prolonged period (epithelial desquamation).
5. Use of high concentration of vasoconstrictors (usually epinephrine), resulting in tissue ischemia and necrosis (Guinta, 1975). Sterile abscess occurs secondary to prolonged ischemia, resulting from epinephrine.

Prevention

1. Avoid using excessive amounts of local anesthetic agent (minimal effective dose).
2. Avoid using vasoconstrictors of high concentration.
3. Avoid rapid deposition and with excessive pressure. Excessive volume may have reaction secondary to excessive pressure.
4. Warn the patient against application of hot items; while the tissues are still anesthetised.

Management

Symptomatic: The management depends upon the extent of injury; and consists of analgesics, topical anesthetics, and bland diet, etc. It usually resolves in 1-2 weeks. An established abscess may require incision and drainage.

COMPLICATIONS ARISING FROM INJECTION TECHNIQUES

Breakage of Anesthetic Cartridge

Causes

It occurs when there is resistance to flow of local anesthetic solution into the tissues.

It occurs due to following reasons:

i. Blockage of the needle.
ii. Too rapid injection; especially during administration of palatal injection. It is due to the palatal mucoperiosteum which is firmly bound down to the underlying bone.
iii. Point of the needle in close contact with bone.

Management

In case, the glass of the cartridge breaks while injection, care should be taken to ensure complete removal of all broken pieces from the mouth; so as to avoid risk of ingestion and injury to the soft tissues of the oral cavity.

Breakage of Needle

The incidences of needle breakage have been reported in the literature. With the introduction of sterile, stainless steel disposable needles, the incidence of needle breakage within the tissues has become considerably less.

Causes

a. *Primary cause:* Sudden unexpected movements by the patient, as the needle penetrates muscle or contacts periosteum. It is basically because of sudden pain on injection.
b. *Secondary or other causes:*
   i. Size/diameter of the needle. Breakage is common in smaller (larger gauge, e.g. 27 G) needles. Smaller needles are more likely to break than larger needles. Gauge of the needle is inversely proportional to size or the diameter of the needle; e.g. 25 G is less in diameter than 22 G. It is a mistake to think that a needle, of a slightly lesser gauge

will produce trauma or that trauma is proportional to gauge of a needle. The sharpness of the point is of more importance than the actual gauge of the needle. It is not the thickness of needle that creates trauma, but rather the blunt point and the barbs.

ii. Previously bent needles. These are weak; and hence more likely to break.
iii. Redirection of needles once inserted inside the tissues.
iv. Needles with defect in the manufacture (Poor quality needles).
v. Forcing needles against resistance.
vi. Needle engaging the periosteum while giving nerve blocks, especially, the indirect pterygomandibular block technique with the bevel facing the bone (Nevin).

Prevention

The following principles should be followed to prevent the possibility of breakage of a needle.

1. Inform the patient about the technique and the procedure. Avoid a sudden unexpected needle insertion. An informed patient is always a better patient, and is much more co-operative. The patient should be warned not to move, as needle touches the mucous membrane.

   Injection of a small amount of LA solution in the tissues, slowly and carefully, in advance of needle, tends to lessen the pain associated with the insertion and injection.
2. Selection of proper gauge of needles.
   a. A needle of a very fine gauge (27 or 30) should not be used for nerve blocks. For nerve blocks, such as pterygomandibular block, posterior superior alveolar nerve block, maxillary nerve block, mandibular nerve block, use 25-gauge needles.

      For nerve blocks, especially mandibular injections, it is strongly recommended that 25-gauge needles should be used.
   b. For infiltration anesthesia: 25, or 27, or 30 G, and 1″ needles may be used. The use of fine gauge needles should be restricted to superficial injections.
3. Use presterilized disposable needles. Do not use resterilisable needles. These become dull and the shaft becomes weak.
4. The entire length of the needle should not be inserted into the tissues. Insertion of the needles must be up to a few mm away from the hub. In the past, when reusable needles were used; the junction of the shaft with the hub formed the weakest point of needle, and therefore, was the site where breakage usually occurred. The needle should be long enough (adequate length) so that a sufficient portion will be allowed to project beyond tissues when the point of the needle reaches

its final destination. This helps the operator, in case of breakage of the needle, to remove the broken needle by holding the portion of the needle outside the soft tissues. Therefore, in nerve blocks, 1½" (38-40 mm) needles should be used.

The needle should not be embedded into the tissues up to the hub. It is vulnerable to breakage in this position. The average insertion/penetration in nerve blocks seldom exceeds one inch (25 mm).

5. Do not redirect the needle once completely embedded inside the tissues. Excessive lateral force on the needle is an important factor in breakage. Always withdraw the needle almost completely, to the submucosal layers and then redirect it.
6. Use needles of good quality, and of a good manufacturer.
7. Gentle manipulation: At no time should there be any resistance to progress of needle during injection. If resistance is encountered, the technique of insertion is modified accordingly. Never force needles against resistance.
8. Do not permit the needle to engage the periosteum while giving a nerve block. There is always a danger of engaging the periosteum in mandibular injection, especially, if indirect approach is used. The needle may penetrate periosteum at the internal oblique ridge and when the syringe is swung to opposite bicuspids, the needle thus bound to bone may bend and break. This is the most frequent cause of needle breakage in mandibular injection.
9. Stabilization of the jaw: Mandible should always be supported by fingers or thumb at the inferior border of mandible during the procedure of injection.
10. Needle point should be inserted in the shortest and the most direct line to mandibular foramen. In other words, needle should always be kept straight during injection procedure.
11. Thorough knowledge of anatomy of the region involved. Do not attempt injections if you are not well aware of the anatomy of the area of the technique employed. Familiarise yourself with all the necessary anatomical landmarks of the area.
12. Avoid multiple penetrations with the same needle.

Management

Information and Reassurance

1. Remain calm and do not panic.
2. Inform the patient of the situation. Make an attempt to allay patient's fear and apprehension.

Localization of the Broken Needle

Various radiographs are taken in two different angles to localize the position of the broken needle, such as PA view, lateral view, or OPG, etc. Use of pilot needle/localizers or a radiopaque localizer or another needle can be used to facilitate localization or to pinpoint the site of the retained or the broken fragment. Once this relationship between localizing needle and the broken fragment is established radiographically, dissection is carried out to retrieve the broken needle under local or general anesthesia.

a. *In case, the needle has broken and is not completely buried in the soft tissues:* If the needle is visible, and is outside the soft tissues, hold it with a hemostat and remove it. When giving injection, it is wise to have a pair of mosquito forceps, hemostat, or curved Spencer Well's forceps at hand so that if the needle breaks and the broken end is visible in the tissues, then without shifting the gaze, or the steadying finger, the operator can pickup one of the above mentioned instruments, and grasp the broken end of the needle to remove it slowly. When the patient moves or swallows then the broken fragment may shift deeper into the tissues and set out of sight.
b. *In case, the needle has broken and is completely buried in the soft tissues:* If the needle is not visible, inform the patient of the situation and give assurance. The patient should be advised not to move the jaw. The patient should be referred to Oral and Maxillofacial Surgeon for its removal.

   Usually the needle breaks when the tip of the needle is in contact with the bone. In case, if the needle end is buried, then use the curved hemostat, open it and press the convex tip of the hemostat above and below the bleeding point present where the needle is broken, there is a good chance that the broken end will emerge out of soft tissues, grasp with another hemostat and remove.

Needle-stick Injuries

Definition

Accidental injuries occurring to dental staff caused by sharp instruments such as needles, blades, scalpels, explorers, root canal instruments, and wires, etc.

These injuries are not usually serious, unless, the instruments used were contaminated by blood from patients with conditions such as Hepatitis B virus (HBV) infection, Hepatitis C virus (HCV) infection, AIDS or AIDS Related Complex (ARC).

Causes

Careless technique.

Prevention

The accessories which help in the prevention of such injuries are available and are recommended for use, are shown in Figures 21.1 to 21.4.

1. Careful handling of sharp instruments, needles and wires.
2. Prophylactic vaccination of HBV infection taken and maintained.

The accessories for prevention of needle-stick injuries, are shown in the following diagram, which are available and recommended for use.

Management

If the injury involves a patient with HBV or HCV infection the following management protocols should be followed. The management depends upon immune status of the Health Care Worker (HCW).

i. HCW who never took vaccination should receive Hepatitis B immunoglobulins (HBIG) within 48 hours of exposure, and a course of HBV vaccination should be started as soon as possible.

ii. HCWs who have been vaccinated, the management depends upon the antibody titre. If the titre is more than 100 mu/ml within the previous year, no further treatment is required. If antibody titre was not done in the previous year, or if the titre is low; then a booster dose of the vaccine is taken, followed by testing of the antibody titre. Those HCW who fail to respond to the vaccine should be given protection by giving Hepatitis B immunoglobulins (HBIG).

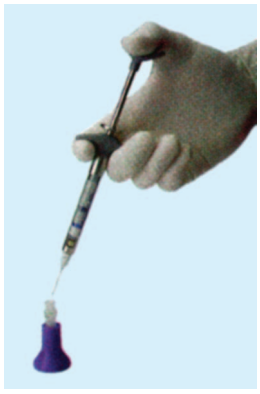


Fig. 21.1: Needle recappers—Aim safe needle guard
- Helps prevent needle-stick injuries
- Reusable and autoclavable up to 134° C
- Heavy duty rubber

Fig. 21.2: Jenker needle-stick protector
- Solid stainless steel receptacle for holding the needle sheath for safe recapping

Fig. 21.3: Needle capper—one handed uncapping and recapping
- Secures withy adhesive strips (included)
- Syringe is held ready for use in a convenient position, and after use, syringe is replaced for recapping
- Made with microban protection
- Available in two sizes
  i. standard—for x-short and short needles
  ii. long—for long needles

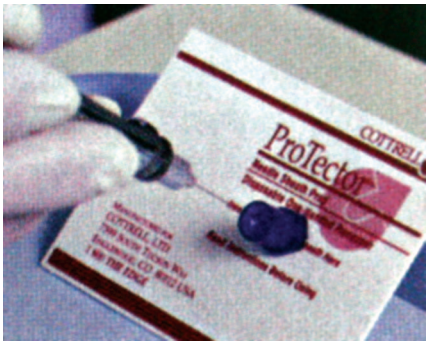


Fig. 21.4: Protector needle sheath prop
- A disposable device, that securely holds a needle-cap in a ready position
- Used to safely uncap and recap needles using one-handed technique

Presently, there are no guidelines for the prophylaxis for HCV infection. The management, however, consists of monitoring liver functions and testing for anti-HCV antibodies. Interferon α is being used in such cases.

If the injury involves a patient with HIV infection or AIDS or AIDS related complex, then the post-exposure prophylaxis (PEP) for HIV, as recommended by National AIDS Control Organization (NACO), should be followed. See Government related guidelines for HIV in Chapter 23.

Hematoma

Definition

Effusion of blood into extravascular spaces. Blood effusion continues until extravascular pressure exceeds intravascular pressure or until clotting occurs. Certain regions have a greater incidence of hematoma. The incidence is more in posterior superior alveolar nerve block and pterygomandibular block. The possible complications include pain, trismus, swelling and discoloration of the region. The density of the tissues surrounding the injured vessel is an important factor.

Causes

Nicking of blood vessels (artery and vein; usually an artery) during injection of local anesthetic solution.

Prevention

1. Good knowledge of anatomy.
2. Use short needle for posterior superior alveolar nerve block. It decreases the risk of hematoma.
3. Adherence to the protocol for the injection technique.
4. Minimise the number of needle penetrations.
5. Never use needle as a probe in the tissues.

Management

Explanation of the condition and assurance to be given to the patient. The swelling and discoloration usually resolve within 7 to 14 days.

Immediate Treatment

In the immediate phase the hematoma manifests as swelling and discoloration. Hence, the following guidelines will be helpful:

i. *Application of direct pressure:* If the area is accessible, application of mechanical pressure at the site of bleeding for a few minutes will be helpful. In most instances, except for posterior superior alveolar nerve block, the blood vessels lie between skin and bone, on which pressure should be applied for at least 2 to 3 minutes. This effectively stops bleeding.
ii. *External application of ice:* For a few hours will also control further formation of hematoma.

Delayed Treatment

Advise the patient of possible pain and limitation of movement. Pain is to be treated by analgesics. Treat the trismus with anti-inflammatory drugs and muscle relaxants.

Do not apply heat to the area in the early phase as it produces vasodilatation and may lead to increase in the size of hematoma. External ice application is advised for at least one day, as it causes vasoconstriction. Later, heat application is indicated for vasodilatation to absorb the hematoma.

Avoid dental treatment until there is resolution of symptoms.

Failure to Obtain Local Anesthesia

Causes

Operator-dependent

i. Selection of local anesthetic agent (type and dose; too small a dose).
ii. Use of a local anesthetic solution which has crossed its date of expiry as recommended by the manufacturer.
iii. Improper injection technique:
- *Wrong technique:* Inaccurate placement of solution (deposition of solution far away from the nerve).
- Not waiting long enough for anesthesia to act; before commencing the surgery.

iv. Injection of wrong solution.
v. Intravascular administration.
vi. Intramuscular administration.

Patient-dependent

i. Anatomical:
- Barriers to diffusion
- Anatomical aberrations
- Additional innervation

ii. Psychological:
Fear and apprehension
- Unco-operative patient, inadequate opening of the mouth, movement by the patient

iii. Pathological:
- Presence of infection
  a. It is widely believed that there is alteration in the pH of tissues in the presence of infection; which leads to acidic environment. The local anesthetic solutions are not effective as these agents are alkaloids, and are not dissociated in an active state
  b. There is an increased vascularity of acutely inflamed tissues, which may also be a factor
- Presence of trismus.

Prevention

1. Good knowledge of anatomy.
2. Good injection technique:
   i. Accurate placement of solution.
   ii. Deposition of adequate dose.
   iii. Wait for sufficiently long enough for anesthesia to act; and then commence surgery. Check for subjective and objective signs and symptoms.
   iv. Consider using aspiration technique.

Management

1. Repeat the injection.
2. Consider giving additional injections such as intraligamentary, intraosseous or intrapulpal injections; unless there is presence of infection surrounding the tooth.
3. Consider anesthetising additional nerves.
4. Avoid injection of a wrong solution.

   Prevention

   To avoid injection of a wrong solution, the following measures are recommended:

   Multidose Vials

   All the local anesthetic solutions and other injectable drugs in the similar containers, should be adequately labeled; or the other injectable drugs should not be kept on the same trolley, or in the vicinity of local anesthetic agents.

   Cartridges

   Encourage use of cartridges. Now, with the introduction of cartridges universally, the injection of solutions other than local anesthetic agents is avoided. There should be identifying marks on individual cartridges for easy identification by the dental practitioner.

Management

Antibiotics, local dressings, oral hygiene instructions, anti-inflammatory/analgesics, and diet restrictions.

Accidental IV Administration

The most toxic reactions are because of the direct result of an accidental IV injection, which introduces local anesthetic solution into blood stream and carries it directly to the heart and cerebral centres. Toxicity of local anesthetic solutions is markedly increased once it gets into blood stream. An intra-arterial injection would be 4 times as toxic as a subcutaneous injection, while an IV injection increases toxicity to 16 times that of subcutaneous injection.

The nerves are almost always accompanied by vessels; and arteries and veins leaving the foramen may be entered by the point of needle. Unlike long bevel of surgical needle, dental needles are bevelled at 45° angle to minimise the possibility of entering a vessel.

The tough muscular coat of arteries offers additional protection and the vessels tend to "bounce" away from the needle. Veins have more flabby structure; and hence encourage penetration; and consequently, our difficulties are usually associated with accidental IV injections.

Mandibular Injections

The two most common possibilities of accidental IV injection involving mandibular injections are inferior alveolar vein at sulcus mandibularis and retromandibular vein in the substance of parotid gland, behind the posterior border of ramus of the mandible.

Additional Innervation

Cutaneous colli nerve (cervical cutaneous nerve): (A branch of 3rd cervical nerve): It enters a small foramen on the lingual aspect of ramus and gives innervation to mandibular teeth.

It arises from 3rd cervical nerve. It appears at the posterior border of sternocleidomastoid muscle, crosses to midline of the neck, where it spreads out in a fan-like manner to innervate the region from sternum to chin. This is a sensory nerve, and it is possible that several twigs combine and enter the mandible on the lingual surface opposite the apex of second bicuspid and innervate posterior teeth. An injection on the lingual surface of the mandible opposite bicuspids should anesthetise this nerve. The foramen on the lingual aspect of mandible through which cervical cutaneous nerve passes is not always present.

In case of failure in obtaining operative anesthesia after a mandibular injection, a supplemental injection can be given to block cervical cutaneous nerve. This is done by inserting the needle lingually between two bicuspid teeth, at the reflection of mucous membrane and directing it posteriorly. About half of the needle is inserted and about 0.5 ml of solution is injected.

COMPLICATIONS ARISING FROM BOTH

Pain on Injection

This increases patient's anxiety; and may lead to a sudden unexpected movement by the patient and increases the risk of needle breakage.

Causes

1. Careless injection technique.
2. Dull needles: Needles become dull due to multiple injections.
3. Rapid deposition of local anesthetic solution.
4. Needles with barbs: There is pain while withdrawal of the needle from the tissues.
5. Temperature: Extremes of temperature such as warm or hot or very cold (refrigerated) local anesthetic solution.

Prevention

Every effort should be taken to administer local anesthetic solution as painlessly as possible.

1. Proper injection technique. The basic principles of injection should be followed. The lip or the cheek is properly reflected so that the tissues are tensed, the sharp point of the needle is placed at right angle to the mucosa, and the puncture is effected. The insertion of the needle should be slow and as atraumatic as possible. Multiple penetrations or insertion in the same area should be avoided.
2. Use sharp needles. Use good quality disposable needles supplied by a reputable manufacturer.
3. Use sterile local anesthetic solutions.
4. Apply topical local anesthetic agent prior to injection to the site of injection.
5. Inject local anesthetic solution slowly.
6. Temperature of local anesthetic solution should be the same as room temperature, and hence, the local anesthetic solutions should be stored at room temperature.

Management

Not required. However, steps should be taken to avoid pain associated with injection of local anesthetic agent.

Burning on Injection

Causes

1. Altered pH of the solution
   a. Presence of vasoconstrictor: The pH of local anesthetic solution without the vasoconstrictor is approximately 5 to 5.5; and that with a vasoconstrictor is approximately 3 to 3.5. It is more acidic.
   b. Old solution: The end result of oxidation of Na-bisulfite is Na-bisulfate which is more acidic.
2. Non-isotonic local anesthetic solution.
3. Other causes:
   i. Rapidity of injection: Especially in adherent tissues and confined areas, such as palatal mucoperiosteum.
   ii. Contamination of local anesthetic solution, especially when cartridges are stored in alcohol or other cold sterilizing solutions; leads to diffusion of these solutions into the cartridge.
   iii. High temperature of local anesthetic solution. Solutions warmed to body temperature are usually considered to be "too hot" by the patients.
   iv. Deposition of excessive amount of local anesthetic solutions.

Prevention

1. Slow injection. Ideal rate is 1 ml/min; while recommended rate is 1.8 ml/min.
2. Cartridges should be stored in a suitable container at room temperature without alcohol or any other cold sterilizing solutions.
3. The excess of cold sterilising solution should be removed by dipping the cartridge in sterile water or normal saline.
4. Use recently manufactured cartridges, as far as possible, to circumvent the problem of increased acidic medium of the solution, because of oxidation of vasoconstrictor.

Management

The tissue irritation caused due to the high pH of local anesthetic agent, usually, does not require any treatment, as most instances are transient, and do not cause tissue damage. It disappears as the action of local anesthetic agent takes place. Usually, there is no residual pain or burning after the action of local anesthetic agent subsides.

With rapid injection, or injection of a contaminated or a warm solution, there is a greater chance of tissue damage, which subsequently may lead to other complications such as trismus, edema, and paresthesia. In such situations, management of the specific individual problem is required.

Persistent irritation or burning during or after the injection warrants an investigation of the local anesthetic solution. The matter should be brought to the notice of the concerned manufacturer.

Infection

The incidence of injection-related infection has become less following introduction of pre-sterilised disposable needles and cartridges.

Causes

1. Contamination of the needles. Needles touching mucous membrane other than the area of insertion of the needle result in contamination of the needle. It is the major cause of post-injection infection.
2. Contamination of the local anesthetic solution. It is also rare, as the solutions are pre-sterilized.

   Contamination of needles or solutions may cause a low-grade infection, if placed in the deeper tissues, which may lead to trismus. Low-grade infection is not recognized immediately. The patient usually complains of pain and dysfunction in the immediate post-injection phase.

3. Improper injection technique: It includes the following:
   a. Improper handling of local anesthetic equipment (storage of the cartridges).
   b. Improper preparation of the site.
   c. Inadequate washing of operator's hands.
   d. Needle passing through an area of infection. It may disseminate infection.
4. Local anesthetic solution deposited under pressure; as in intraligamentary injection. It is claimed to deposit bacteria, in healthy tissues and thus spreading the infections.

Prevention

Take measures for complete asepsis, as far as possible, which are as follows:
1. Preparation of the site prior to needle penetration: Apply antiseptic, dry the area, and then apply topical anesthetic agent.
2. Careful handling of the needles. Avoid contamination of needles through contact with non-sterile surfaces.
3. Avoid multiple penetrations with the same needle.
4. Use pre-sterilized disposable needles.
5. Proper cleansing of operator's hands.
6. Avoid passing the needles through infected areas.
7. Proper handling of dental cartridges:
   i. Store cartridges aseptically, as far as possible, in a container covered with a lid all the time. Once the container is opened, cartridges should be stored dry in their original container or in another suitable sterile container that is kept covered at all times.
   ii. Avoid contamination of plunger and the diaphragm prior to their use. The diaphragm-end of cartridge should be wiped with a sterile disposable sponge soaked with an antiseptic prior to its insertion into the syringe and fixing of the needle.
   iii. Use cartridges available in blister packing.
   iv. Use cartridges only once (one patient). An attempt to use a portion for one patient and the remaining for another patient increases the possibility of cross-infection.

Management

The management is symptomatic; and it consists of the following:
   i. Analgesics
   ii. Antibiotics
   iii. Physiotherapy
   iv. Heat therapy
   v. Anti-inflammatory drugs

vi. Muscle relaxants, and
vii. Incision and drainage, if necessary.

Trismus

It is a fairly common complication of regional anesthesia, particularly while giving pterygomandibular block.

Causes

Primary Cause

i. Trauma to muscles (medial pterygoid, temporalis, and masseter), blood vessels in infratemporal and pterygomandibular fossae during insertion of the needle. It is the most commonest cause.

Secondary Causes

The following factors cause varying degree of trismus.

i. Injection of local anesthetic solutions containing irritating solutions, such as, alcohol or other cold-sterilizing solutions, which diffuse into the muscle tissue, producing irritation of the muscle leading to trismus.
ii. Local anesthetic solutions have mild myotoxic properties on skeletal muscles. Injection of local anesthetic solutions intramuscularly, or extramuscularly leads to progressive necrosis of the exposed muscle fibers (Benoit et al, 1980, Hinton et al, 1986, and Jastak et al, 1995).
iii. Hematoma (large volume of extravasated blood) leads to irritation of muscles fibers.
iv. Low grade infection following injection (Kitay et al, 1991).
v. Deposition of excessive amounts of local anesthetic solutions into a particular area. This occurs following multiple failed pterygomandibular blocks, and which lead to distension of the tissues and may result in post-injection trismus.

Prevention

1. Use sharp, sterile and disposable needles. This avoids the trauma of injection and prevents subsequent low grade infection.
2. Proper handling of local anesthetic cartridges.
3. Proper handling of needles. Avoid contamination of needles.
4. Avoid multiple penetrations into the same area. It increases the incidence of post-injection trismus.
5. Use minimum effective volumes of local anesthetic solutions. Avoid deposition of excessive amounts of local anesthetic solutions into a restricted area. It produces distension of tissues. This may lead to post-injection trismus.

6. Good knowledge of anatomy of the region and fascial spaces.
7. Follow proper injection technique.
8. Use aseptic technique. Topical application of antiseptics.
9. Use atraumatic technique.

Management

It depends on the cause of trismus, and consists of the following:

i. Heat therapy
ii. Warm saline rinses
iii. Analgesics
iv. Anti-inflammatory
v. Muscle relaxants (Himel et al, 1988, and Kouyoumdjian et al, 1988)
vi. Physiotherapy, and, if necessary
vii. Antibiotics.

i. Heat therapy: It includes application of heat, in various ways to the affected area, three to four times a day. For example, application of moist compresses for 15 to 20 minutes/hour for several days until the symptoms are relieved.

ii. Warm saline rinses: These are made by adding a teaspoonful of salt to a glass of water. It is held in the mouth on the involved side for a few minutes and is spat out.

iii. Analgesics.

iv. Anti-inflammatory drugs: The analgesics used belong to the group of non-steroidal anti-inflammatory agents (NSAIDs) such a aspirin, ibuprofen, codein (30-60 mg q6h), etc. which have both analgesic as well as anti-inflammatory actions.

v. Muscle relaxants: The commonly used muscle relaxants are Chlorzoxazone (250 mg in 2 to 3 divided doses, in combination with NSAIDs, and Diazepam (5-10 mg bid), or any other benzodiazepines. Meprobamate 1.2 g in 3 to 4 divided doses can also be used.

vi. Physiotherapy: It includes mouth opening exercises, as well as, lateral excursions (side to side movements), for 5 to 10 minutes every 3 to 4 hours.

vii. Antibiotics: If the cause of the trismus is hematoma or low grade infection, suitable antibiotics should be prescribed.

If the condition is due to trauma, mild physiotherapy, analgesics and muscle relaxants are advocated. If hemorrhage or low grade infection is the cause, then, antibiotics, anti-inflammatory drugs, and warm saline mouthwashes are indicated.

Avoid further dental treatment in the involved region until symptoms resolve, and the patient is comfortable. If for any reason, the dental care has to be continued, an alternate method or technique for achieving local

anesthesia may be employed. The Akinosi mandibular nerve block provides relief from the motor dysfunction and allows the patient to open the mouth and permits administration of an appropriate additional injection, if required.

Complete resolution of post-injection trismus takes approximately 6 weeks, with a range of 4 to 20 weeks (Hinton et al, 1986). If the above measures fail; refer the patient to Oral and Maxillofacial Surgeon.

Edema

Edema of tongue, pharynx and larynx may develop into potentially life-threatening situations.

Causes

1. Trauma during injection
2. Infection
3. Allergy
4. Hematoma
5. Injection of irritating solutions such as cold-sterilising solutions.

Each factor should be considered with regard to its prevention and management.

Prevention

1. Preoperative assessment: Complete medical evaluation of the patient, particularly history of allergy to any drug.
2. Careful handling of local anesthesia armamentarium.
3. Atraumatic anesthetic technique.

Management

Find out the cause.

1. In cases of traumatic injection and introduction of irritating solutions, the edema is minimal and resolves in a few days, and therapy is sometimes not required.
2. Analgesics for pain.
3. In case of infection, start suitable antibiotics.
4. If allergy: Administer antihistaminics orally and/or IM; sometimes it can be life-threatening. Consultation with an allergic specialist is mandatory. If breathing is compromised because of edema the following steps are taken:
   i. Patient is placed in supine position. In case of tongue or oro-pharyngeal region, right or left lateral position is taken.

ii. Institute Basic Life Support (BLS); Airway, Breathing, and Circulation (ABC).
iii. Emergency Medical Services (EMS) are summoned.
iv. Administer O_2.
v. Administer epinephrine: 0.3 mg (adults), 0.15 mg (child) IM/IV every 5 minutes until respiratory distress resolves.
vi. Administer antihistaminics.
vii. Administer corticosteroids.

5. Refer to oral and maxillofacial surgeon.
6. Life-threatening situations may require cricothyroidotomy. Transfer the patient to a general hospital with an ICU facility in your vicinity.

Mucosal Blanching

It is caused by the spasm of the artery accompanying the nerve at the point of injection.

Causes

1. Use of excessive amount of vasoconstrictor in the local anesthetic solutions.
2. Deposition of excessive volume of local anesthetic solution in firm or tight tissue.

Prevention

1. Good knowledge of anatomy.
2. Adherence to the anesthetic protocol.
3. Use of aspiration technique; and avoid intra-arterial administration of local anesthetic agents.

Management

Usually a transient phenomenon; and no treatment is necessary.

Persistent Anesthesia or Paresthesia (Nerve Injuries)

Nerve injuries could be (a) Post-injection, or (b) Postoperative.

It is the prolonged loss of sensation (hypoesthesia or anesthesia) of the part of soft tissues of face, (including tongue and lip), for days, weeks, or months, following injection of local anesthetic agent or removal of tooth/root. Paresthesia is altered sensation, and may be with partial anesthesia.

It is a disturbing, and sometimes unpreventable complication of local anesthetic administration. The most common sites are lips and tongue in their descending order of frequency. Paresthesia is more common with

prilocaine, followed by other injectable local anesthetic agents (Haas and Lennon, 1995). When lingual nerve is involved, sense of taste may also be affected *(chorda tympani)*.

Persistent paresthesia can lead to self-inflicted injury. Biting, or thermal or chemical insults can occur without the patient's awareness. The condition is more frequent as a result of operative procedure than injection itself.

The sensory nerves most frequently traumatised are inferior alveolar nerve, lingual nerve, and mental nerves in lower jaw; and infraorbital nerve in upper jaw.

Prolonged anesthesia of lip, tongue or nose and cheek follows injury to these nerves; the part affected depending upon which nerve is injured. The duration of anesthesia depends on severity and extent of nerve injury.

Causes

1. Injection of local anesthetic solution contaminated with cold-sterilising solution, such as alcohol, near a nerve. It produces irritation, resulting in edema and increased pressure in the region of the nerve, leading to paresthesia. Alcohol is neurolytic; and may cause paresthesia lasting for months and years.
2. Injection of a wrong solution. It may result from injection of a solution other than a local anesthetic solution, such as alcohol or other cold-sterilising solutions. Only in severe cases the damage is permanent. In the absence of a known cause for prolonged anesthesia, this factor should always be considered, if cold-sterilizing solutions are used, at any time.
3. Trauma to a nerve: It is usually due to lack of knowledge of anatomy, and faulty technique. It may lead to following situations:
   i. Trauma to a nerve or the nerve sheath: Initially the patient gets a feeling of an "electric shock" to the area innervated by the nerve. Insertion of the needle into a foramen also increases likelihood of nerve injury.
   ii. Complete severing of the nerve fibers/trunk with a needle is extremely rare. The damage from the needle is hyperalgesia and not anesthesia.
4. Hemorrhage into or around the nerve sheath caused by mild trauma of the needle.

The resultant hemorrhage is reabsorbed very slowly as a result of poor circulation in this area. This prolonged pressure may lead to degeneration of some of the nerve fibers and may lead to transient decreased sensation.

Prevention

1. Strict adherence to injection protocol.
2. Careful injection technique.
3. Proper handling of local anesthetic cartridges.
4. Good knowledge of anatomy.

Management

Most paresthesias resolve within 8 weeks without treatment (Nickel, 1990). Rare, only if the damage to the nerve is severe, then only the paresthesia may be permanent (Haas and Lennon, 1995).

1. Reassurance to the patient.
2. Administer neurotropic vitamins (Vitamins B_1, B_6 and B_{12}), parenteral and oral; or vitamin B_{12}, oral and parenteral. Presently, the use of methylcobalamine, which is an active form of vitamin B_{12}, which has got better absorption than other forms like cyanocobalamine, (also hydroxyl-, adenosyl-), and hence it is preferable to administer, in case of nerve injuries.
3. If dental treatment is to be continued, avoid readministration of the local anesthetic agent into the same region of traumatised nerve. Use alternate local anesthetic technique, if necessary and if possible.
4. Refer the patient to oral and maxillofacial surgeon, if necessary.

Persistent or Prolonged Pain

Causes

1. Poor injection technique. Subperiosteal injection of local anesthetic agent, and tearing of the periosteum by the tip of the needle.
2. Needle tip with barbs.
3. Ischemic necrosis (use of vasoconstrictors: Excess amount, higher concentration).
4. Multiple penetrations.

Prevention

1. Good injection technique: Avoid subperiosteal injection, and tearing of periosteum. Infiltration anesthesia should be given paraperiosteally or supraperiosteally, but not subperiosteally.
2. Avoid needles with barbs.
3. Use vasoconstrictors with higher dilution.
4. Avoid multiple penetrations.

Management

It is symptomatic, and comprises of analgesics.

Post-injection Herpetic Lesions or Post-anesthetic Intraoral Lesions

Patients' reporting of development of ulcerations around the site of injection a few days after intraoral injection of local anesthetic agent. Patient complains of intense pain.

Causes

Recurrent Aphthous Stomatitis (RAS): It is a frequent manifestation, developing in gingival tissues (movable part, i.e. not attached to the bone) such as buccal vestibule.

Herpes Simplex/Herpes Labialis: It is related to reactivation of dormant Herpes Simplex Virus (HSV) particles by the trauma of injection. It is usually seen in patients with history of recurrent herpes labialis, particularly, in the terminal area of distribution of trigeminal nerve (inferior alveolar nerve, or superior labial branch of infraorbital nerve), in a previously anesthetised nerve. HSV can develop intraorally, although it is commonly observed extraorally. It is manifested as small bumps on tissues attached to underlying bone; such as soft tissues of the hard palate. Trauma to the tissues by a needle, local anesthetic agent, cotton swab, or any other instrument, may activate the latent form of the disease process.

Prevention

1. Pre-anesthetic assessment: History of recurrent herpetic infections.
2. Delay surgical intervention in the active stage.

In susceptible patients, intraoral lesions cannot be prevented from developing. Intraoral Herpes Simplex, may be prevented, or its manifestations may be minimized, if treated in its prodromal phase (Prodrome: Mild burning or itching sensation at the site where the virus is present, e.g. lip.

Management

Explanation and assurance are integral parts of the management. The management, otherwise is symptomatic and includes: (i) Analgesics, (ii) Topical anesthetics, e.g. viscous lidocaine, applied topically to affected/painful areas, and (iii) Antiviral agents, (acyclovir) applied QID over the affected area. It minimizes the acute phase.

Bizarre Neurological Symptoms

It is seen in the form of (a) Facial nerve paresis, and (b) Visual disturbances.

Facial Nerve Paresis

Paresis of some of the muscles of facial expression which are supplied by some of the terminal branches of facial nerve, when the solution is deposited in their vicinity.

a. *Directly:* In the vicinity of terminal branches of facial nerve as (i) in infraorbital nerve block, or (ii) while giving a paraperiosteal injection for maxillary canine.

b. *Indirectly:* Into the deep lobe of parotid gland as in the pterygomandibular block.

It is a transient phenomenon and lasts for a few hours; depending upon: The agent used, its volume injected, and the proximity to the branches of facial nerve. There is usually minimal or no sensory loss.

Manifestations

i. *Face:* It results in unilateral loss of motor function of the muscles of facial expression. The face appears to be lopsided; there is drooping of lips, etc.
ii. *Eyes:* It manifests in the form of inability to voluntarily close the ipsilateral eye. The protective lid reflex of the eye is abolished. Winking or blinking becomes impossible. Cornea, however, retains its innervations. Thus, if irritated, the corneal reflex is intact, and tears will lubricate the eye.
iii. There will be no anesthesia in the area of injection.

Causes

1. Injection of local anesthetic solution in the capsule of, or the deep lobe of parotid gland, during a pterygomandibular block.
2. Injection, superficially, into the muscles of facial expression, or in the vicinity of the nerves innervating them, as in infraorbital nerve block.

Prevention

1. Good knowledge of anatomy.
2. Adherence to the standard protocol for local anesthesia technique, particularly, pterygomandibular block and infraorbital nerve block. In the pterygomandibular block, if the needle is advanced posteriorly and bone is not contacted, the needle should be withdrawn almost completely from the soft tissues; the barrel of syringe is shifted posteriorly, the needle is then advanced more anteriorly, until it contacts the bone.

Management

It does not require any treatment. The effect is transient and lasts as long as the effect of anesthesia.

1. Explanation of the situation, that it is transitory phenomenon; (as it lasts for a few hours), and reassurance given to the patient.
2. Eye dressing to be given over the affected eye. Until muscle tone returns, or ask the patient to close the eye manually; close the eyelids periodically to keep cornea lubricated.
3. Contact lenses, if any, should be removed, until muscular movements return.

Visual Disturbances

These complications are rare and difficult to explain. These complications are usually seen as unilateral or bilateral disturbances of vision; and are seen in the following forms:

a. Squint.
b. Diplopia or Double vision.
c. Transient amaurosis (blindness without a demonstrable lesion in the eye).
d. Permanent blindness.

The probable explanations of the associated phenomena of these situations, are as follows:

i. It is possible that vascular spasm.
ii. The accidental intra-arterial injection is most likely cause. In such situations, an unusual vascular distribution can be assumed to be the likely situation.
iii. It may be due to inadvertent anesthesia of the nerves in the region.

Diagnosis

These complications have to be diagnosed on the basis of clinical manifestations.

Prevention

i. Sound knowledge of anatomy and the landmarks for the injection technique.
ii. Strict adherence to the injection protocol.

Management

The best method is to prevent these complications.
Reassurance: The patient is assured that a normal region will be restored within 30 minutes.

Diplopia or Double Vision

The resultant disturbance in the vision will return back to normal as the solution gets diffused, usually within about 3 hours.
Example: The injection which may cause diplopia is infraorbital nerve block.

Explanations

i. The most likely explanation given is that some of the maxillary injections may result in local anesthetic solution infiltrating into the orbit to anesthetise the extrinsic ocular muscles of the eye.
ii. An accidental intra-arterial injection of local anesthetic solution in patients with uncommon vascular patterns. In these situations, it is presumed that the orbit is supplied either wholly or partly by middle meningeal artery, a branch of internal maxillary artery.

Management

No treatment is necessary; other than reassurance. The vision usually returns to normal at the end of the anesthetic effect.

Transient Squints and Double Vision

These complications have occurred following posterior superior alveolar nerve block and maxillary nerve block injections. These complications are due to paralysis of extrinsic muscles. The most likely explanation is that the local anesthetic solution gets diffused into the orbit from the pterygopalatine ganglion and infratemporal fossa via the infraorbital fissure.

By these routes, the local anesthetic solution, may affect the oculomotor, trochlear, and abducens nerves, which innervate the muscles attached to the eyeball.

Management

These complications pass off within 2 to 3 hours.

BIBLIOGRAPHY

1. Archer WH. A Manual of Dental Anesthesia: An illustrated guide for students and Practitioners. 2nd ed. WB Saunders Company 1958.
2. Benoit PW, Yagiela JA, Fort NF. Pharmacologic correlation between local anesthetic-induced myotoxicity and disturbances of intracellular calcium distribution. Toxic Appl Pharmacol 1980;52:187-98.
3. Haas DA, Lennon D. A 21-year retrospective study of reports of paresthesia following local anesthetic administration. J Can Dent Assoc 1995;61(4):319-20,323-26,329-30.
4. Himel VT, Mohamad S, Luebke RG. Case report: Relief of limited jaw opening due to muscle spasm. LDA J 1988;47:6-7.
5. Hinton RJ, Dechow PC, Carlson DS. Recovery of jaw muscle function following injection of a myotonic agent (Lidocaine-epinephrine). Oral Surg Oral Med Oral Pathol 1986;59:247-51.
6. Howe GL, Whitehead FIH. Local Anesthesia in Dentistry, 3rd ed, 1990 Indian ed. 1997. A Dental Practitioner Handbook Series 14 (Wright) Mumbai: Varghese Publishing House.
7. Howe GL. Minor Oral Surgery. 3rd ed. Butterworth-Heinemann Ltd. Reprint. Mumbai: Varghese Publishing House 1985.
8. Jastak JT, Yagiela JA, Donaldson D. Complication and side effects. In: Jastak JT, Yagiela JA, Donaldson D (Editors): Local Anesthesia of the Oral Cavity. Philadelphia, WB Saunders 1995.
9. Killey HC, Seward GR, Kay LW. An Outline of Oral Surgery Part II. Dental Practitioner Handbook (Wright) Mumbai: Varghese Publishing House 1992.
10. Kitay D, Ferraro N, Sonis ST. Lateral pharyngeal abscess as a consequence of regional anesthesia. J Am Dent Assoc 1991;122(7):56-59.
11. Kouyoumdjian JH, Chalin VA, Nimmo A. Limited mandibular movement: Causes and treatment. J Prosthet Dent 1988;59(3):330-33.

12. Laskin DM. Oral and Maxillofacial Surgery Vol I and II C. V. Mosby Co. AITBS Publishers, India 1992.
13. Malamed SF. Handbook of Local Anesthesia. 4th ed. New Delhi: Harcourt India Private Limited (Mosby) Indian Reprint 2001.
14. Nevin Mendel, Nevin HR. Problems in Dental Local Anesthesia. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1954.
15. Nevin Mendel, Puterbaugh PG. Conduction, Infiltration and General Anesthesia in Dentistry. Brooklyn, NY: Dental Items of Interest Publishing Company Inc 1948.
16. Peterson LJ, Ellis E III, Hupp JR, Tucker MR. Contemporary Oral and Maxillofacial Surgery. 2nd ed. Harcourt Brace and C. Asia. Pvt. Ltd 1997.
17. Richard Bennett C. Monheim's local anesthesia and pain control in dental practice. 7th ed. New Delhi: CBS Publishers and Distributors 1990.
18. Roberts DH, Sowray JH. Local Analgesia in Dentistry. 3rd ed. Wright, Bristol 1987.
19. Seward GR, Harris M, McGowan DA. Killey and Kay's Outline of Oral Surgery. Part I. Dental Practitioner Handbook (Wright). Mumbai: Varghese Publishing House 1992.

The systemic complications that occur due to the local anesthetic agents fall into following categories:

1. Vasodepressor syncope
2. Adverse drug reactions: Such as (a) allergic reactions, (b) anaphylactic reactions, (c) toxic reactions (overdose), and (d) idiosyncratic reactions.

VASODEPRESSOR SYNCOPE (VASOVAGAL ATTACK)

It is the most common systemic complication that occurs with local anesthesia in dental office.

Definition

It refers to a sudden, transient loss of consciousness usually secondary to cerebral ischemia. The cerebral ischemia is secondary to vasodilatation, or an increase in peripheral vascular bed, with a corresponding drop in blood pressure.

Fainting is not always associated with loss of consciousness. Loss of consciousness is an extreme manifestation of cerebral ischemia sufficient to interfere with cortical function.

Synonyms

Vasodepressor syncope, vasovagal syncope, atrial bradycardia, benign faint, neurogenic syncope, psychogenic syncope, simple faint, and swoon. Vasodepressor syncope is the most descriptive and accurate of these terms.

Predisposing Factors

The factors which can precipitate vasodepressor syncope may be divided into two groups:

1. Psychogenic factors, and
2. Non-psychogenic factors.

Psychogenic Factors

1. Fright and anxiety
2. Emotional stress
3. Receipt of unwelcome news
4. Pain of sudden and unexpected nature
5. Sight of blood or of surgical or other dental instruments, such as local anesthetic syringe, injection needle, etc.

In dental surgery set-up, the psychogenic factors are the most common precipitating factors. These factors lead to fight-or-flight response; and in the absence of muscular activity, manifest as loss of consciousness, termed as vasodepressor syncope.

Non-psychogenic Factors

i. Sitting in upright position or standing for prolonged period. It leads to pooling of blood in periphery, thereby decreasing cerebral blood flow.
ii. Hunger or starvation. There is a decrease in generalised and cerebral blood glucose level.
iii. Exhaustion.
iv. Poor physical condition.
v. Hot, humid and crowded environment
The above mentioned conditions (iii), (iv) and (v) contribute to a situation resulting in syncope.
vi. Injection of a local anesthetic agent with or without a vasoconstrictor into an artery.

Prevention of Vasodepressor Syncope

The prevention is directed at elimination of predisposing factors.

1. Dental office: It should not be hot, humid or crowded. The dental offices, as far as possible, should be air-conditioned.
2. Explanation to the patient, of what is to be done. Prewarned is prearmed.
3. Hunger: Patients should be asked to take a light meal, or snacks; prior to dental appointment to minimise the risk of hypoglycemia.
4. Preoperative assessment:
   i. Patient should be evaluated for presence of anxiety.
   ii. Dental care should be modified to minimise or eliminate it.
   iii. Medical history questionnaire: In medically compromised patients, serious considerations should be given to the medical condition and the drugs being taken for the condition. Treatment modification should be considered in the treatment planning.

5. Proper positioning of the patient: This complication can be prevented, in many instances, by placing the patient in supine or semi-supine or semireclining position with legs slightly elevated (10°) and back elevated (30-45°), at the time of administration of local anesthetic agent. This position keeps blood available to general circulation and avoids cerebral hypoxia. Otherwise blood pools in splanchnic region and legs. This position greatly reduces the incidence of vasodepressor syncope.
6. Use of sedation for relief of anxiety, if necessary; in the form of the following:
   i. Stress reduction protocol,
   ii. Psychosedation, and
   iii. Sedation techniques.
7. Use of topical local anesthetic agent.
8. Keeping the needle out of sight of the patient.
9. Proper injection technique.
10. Observe the patient: Never leave the patient unobserved and unattended at the end of administration of local anesthetic agent.

In most instances, it is possible to detect a change in patients' appearance; such as pallor. The patient may complain of feeling different or strange.

Clinical Manifestations

The clinical manifestations develop rapidly. However the actual loss of consciousness occurs after a period of time. The clinical features of vasodepressor syncope may be grouped into three definite phases: (i) Presyncope, (ii) Syncope, and (iii) Postsyncope (the recovery period).

Presyncope

a. *Early stage:* Patients developing syncope usually have several minutes of warning symptoms before the loss of consciousness (Martin et al, 1984), which include: Feeling of warmth, loss of color, pale or ashen-grey skin tone, heavy perspiration, restlessness, complaints of feeling faint, nausea, tachycardia, and blood pressure approximately at base line.
b. *Late stage:* Pupillary dilatation, hyperpnea, coldness in hands and feet, bradycardia, hypertension, dizziness, visual disturbances, and loss of consciousness (Reutz et al, 1967; Wright and McIntosh, 1971, and Glick and YU, 1963).

Syncope

Irregular breathing, dilatation of pupils, convulsions, bradycardia, low blood pressure, weakness, thready pulse, and loss of consciousness.

Postsyncope (Recovery)

In the postsyncopal phase the patient may exhibit pallor, nausea, weakness and sweating, which may last for a few minutes to many hours. Occasionally, symptoms persist for 24 hours (Friedberg, 1971). There may be a short period of mental confusion and disorientation. The heart rate and blood pressure gradually return to normal. Patient has a tendency to faint again if propped up in sitting position or if allowed to stand too soon, for a period of few hours.

Management

The right time to treat this complication is in its early phase, before the patient has lost consciousness. The management can be divided according to the stages:

Presyncope

As soon as the signs and symptoms of presyncope are noted, the following steps are to be taken:

1. Stop the surgical procedure.
2. Placement of the patient in a head low or supine position with legs slightly elevated.
3. Inhalation of aromatic spirit of ammonia. It stimulates respiratory system, and elevates blood pressure (Fig. 22.1).
4. Administration of O_2 with full-face mask or nasalhood.
5. If patient is conscious, he should be instructed to take deep breaths. This maneuver assists in venous return and provides adequate oxygenation.
6. Monitoring of heart rate and blood pressure.
7. Determination of the cause of syncope.
8. Administration of glucose powder (50 g) in a glass of water.

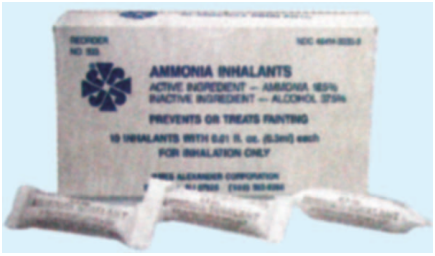


Fig. 22.1: Emergency drugs: Ammonia inhalants for inhalation only
- To prevent / treat fainting
- Contents: 35% isopropyl alcohol and 15% ammonia

9. Administration of 20 to 30% of glucose saline intravenously, if such facilities are available.
10. The patient should be reassured and re-evaluated.
11. Surgical procedure may be carried out, if the doctor and the patient feel it is appropriate. If there is any doubt, postponement of the procedure is recommended.

Syncope

The management remains similar to that for all unconscious patients. A summary of the various steps to be taken is presented here:

1. Assess the level of consciousness.
2. Call for medical assistance.
3. Position the patient in a head low or supine position with legs slightly elevated.
4. Administer Basic Life Support (BLS). Assess the Airway, Breathing and Circulation (ABC).
5. Administer O_2.
6. Monitor vital signs.
7. Provide definitive treatment.
8. Wait for the patient to recover.

Postsyncope

Subsequent to recovery from a period of unconsciousness, the following measures are to be taken:

i. Patient should not be subjected to an additional dental care/procedure for the rest of that day. Also, the patient should not be given additional local anesthetic injection, especially with adrenaline. The body requires at least 24 hours to return to its presyncopal state.
ii. The doctor should determine the precipitating factors, for example; fear, starvation, etc. These factors should be considered for future treatment planning.
iii. Arrangements to be made to escort the patient home with a family member or a friend.

ADVERSE DRUG REACTIONS

Local anesthetic agents are relatively safe and free of side effects provided they are administered in an appropriate dosage and in an appropriate anatomical location. The ester group of drugs are more toxic than the amide group.

The adverse drug reactions include: (1) allergic reactions, (2) anaphylactic reactions, (3) toxic reactions (overdose), and (4) idiosyncratic reactions.

The causes, signs and symptoms, prevention of these reactions are mentioned. The management procedures are common to all the reactions. These include the following:

1. Stop the dental procedure.
2. Place the patient in supine position (legs slightly elevated).
3. Call medical assistance.
4. Institute the preliminary medical care.
5. Keep the airway patent.
6. Administer O_2.
7. Monitor the vital signs.
8. Transfer the patient to a general hospital in the vicinity having ICU facility.

Allergic Reactions

Causes

The primary cause of allergic reactions is a specific antigen-antibody reaction, where the patient has been previously sensitised to a particular drug or a chemical agent.

Classifications of Allergic Reactions (Tables 22.1 and 22.2)

Table 22.1: Gell and Comb's classification of immunopathological reactions

Type of reaction	*Description*	*Antibody*	*Clinical reactions*
I	Anaphylactic	IgE	Anaphylaxis, urticaria
II	Cytotoxic	IgG, IgM	Hemolytic anemia, transfusion reactions, nephritis
III	Immune complex disease	Soluble immune complexes	Serum sickness, drug fever, Glomerulonephritis
IV	Delayed or mediated	None known	Contact dermatitis
V	Idiopathic		Macular eruptions, eosinophilia, S-J syndrome, exfoliative dermatitis

Table 22.2: Classification according to the time of onset

Type	*Onset*	*Clinical reactions*
Immediate	0-1 hours	Anaphylaxis, hypotension, laryngeal edema, urticaria angioedema, wheezing
Accelerated	1-72 hours	Laryngeal edema, urticaria, angioedema, wheezing
Late	> 72 hours	Skin rash, nephritis, serum sickness, drug fever, S-J syndrome, exfoliative dermatitis, hemolytic anemia

Signs and Symptoms

Allergic manifestations include skin rash, urticaria, pruritis, edema, erythema and wheezing.

Prevention

Proper pre-anesthetic evaluation: It comprises of:

i. Proper personal history
ii. Interrogation into past dental history, particularly history of allergy to local anesthetic agents, or history of allergy to any other drug.

True allergy to local anesthetic agents can be in the following forms:

Type I: Immediate hypersensitivity reactions mediated by IgE antibodies, or
Type IV: Delayed hypersensitivity reactions mediated by sensitised lymphocytes.

Management

1. Antihistamines such as diphenhydramine (Benadryl) 20-50 mg; or chlorpheniramine maleate (Avil) (10-20 mg).
2. Epinephrine 0.5 ml of 1:1000 IM (Fig. 22.2).
3. Administer O_2, if necessary.

There are a number of approaches to treat true allergy to local anesthetic agents:

Substitution

The local anesthetic agent can be substituted with another type of agent. If the reaction was in response to ester-type of local anesthetic agents, such as procaine, then an amide type of a local anesthetic agent, such as lidocaine or bupivacaine, can be substituted. If reaction was in response to an amide, several alternatives are available:

Fig. 22.2: Emergency drugs: Epipen epinephrine auto injector

- It is used for allergic emergencies. It is designed to provide fast and reliable self-administered first-aid for potentially fatal anaphylactic reactions in sensitive individuals. The indications include:

i. all types of allergic emergencies including drug reactions; also
ii. insect bites and stings, (iii) food reactions or (iv) exercise-induced anaphylaxis.

No prior filling, assembly, or preparation is necessary. It may be used directly through clothing.

Availability: It is available in two strengths:

i. Epipen (yellow label): 0.3 mg epinephrine 1:1000, and
ii. Epipen Jr. (white label):0.15 mg epinephrine 1:2000

i. *Skin testing:* Specific local anesthetic agent is to be used. These local anesthetic agents use methylparaben, a para-aminobenzoic acid (PABA) derivative, as a preservative. It can be the allergen. The ester-type of local anesthetic agents are also PABA derivative.

In the Indian market, the lingocaine solution is available in dental cartridges and multidose vials with 1:80,000 adrenaline and does not contain methylparaben as a preservative. Hence, it is recommended.

ii. *Use of cardiac lidocaine:* If there is a history of allergy to methylparaben; then cardiac lidocaine (which does not contain methylparaben) can be used.

iii. *Use of an antihistaminic:* If there is a history of allergy to local anesthetic agent, a 10% solution of diphenhydramine hydrochloride (Benadryl 20-30 mg) which has (i) local anesthetic properties, and (ii) antihistaminic properties (Bass, 1970) can be used.

Anaphylactic Reaction / Anaphylaxis

It is a life-threatening or the most devastating allergic reaction of a drug. It is sudden, often unexpected, and many times proves fatal, causing death within a few minutes. It is an IgE mediated mast cell activation. It may develop following administration of an antigen by any route; but most likely with parenteral route.

Signs and Symptoms (Table 22.3)

The signs and symptoms are highly variable. These are rapidly occurring and progressive. There are four major clinical syndromes recognised.

1. Skin reactions
2. Smooth muscles spasm (Gastrointestinal tract, genitourinary tract and respiratory tract)
3. Respiratory distress
4. Cardiovascular collapse; and loss of consciousness

The clinical signs of allergy; such as urticaria, erythema, pruritis or wheezing occur before patient collapses.

Table 22.3: Signs and symptoms

Cutaneous	*Gastrointestinal*	*Cardiovascular*	*Respiratory*
Flushing	Nausea	Lightheadedness	Laryngeal edema
Erythema	Vomiting	Fainting	Hoarseness of voice
Pruritis	Crampy abdominal	Syncope	Dysphonia
Urticaria	pain	Tachycardia	Dyspnea
Angioedema	Bloody diarrhea	Hypotension	Cough
		Dysrhythmias	Wheezing
		Coagulopathies	Cyanosis
			Bronchospasm

Management

Initial Therapy

The following steps should be taken rapidly:

1. Stop the triggering agent/drug.
2. Administer O_2 (100%).
3. Position the patient supine with legs slightly elevated.
4. Give Basic Life Support (BLS) (ABC of Resuscitation).
5. Call for medical assistance.
6. Administer epinephrine IM-IV (0.3-0.5 mg of 1:1000 for adults) immediately, or subcutaneous (SC) or intravenous (IV). Epinephrine is the mainstay of the initial pharmacotherapy.
7. Monitor vital signs.

Secondary Therapy

1. Administration of antihistaminic. If the patient shows signs of allergy, give antihistaminics such as IV/IM diphenhydramine 1 mg/kg (maximum 50 mg).
2. If the patient continues to deteriorate, administer glucocorticoids, such as:
   a. IV hydrocortisone sodium succinate 100 mg, or
   b. IV dexamethasone 8 mg.
3. CPR, if necessary.
4. Transfer the patient to a nearby hospital with ICU facilities in an ambulance, as soon as possible.

Differential Diagnosis of Anaphylaxis

1. Vasovagal reaction
2. Drug overdose
3. Aspiration
4. Bronchospasm
5. Myocardial infarction
6. Cerebrovascular accidents/stroke
7. Pulmonary edema
8. Pulmonary embolism.

Toxic Reactions (Overdose)

The toxic reactions are those clinical signs and symptoms that manifest as a result of administration of an excess amount of the drug, which produces elevated levels in the blood. Higher level of the drug in the bloodstream adversely affects CN system, CV system and respiratory system. The blood levels necessary to produce toxic effects may differ for a particular drug from individual to individual; and from day to day in the same individual.

The majority of toxic reactions occur because of the following:

1. Overdose
2. Inappropriate site of injection
3. Susceptibility to normal dosages.

Mechanism

To reach toxic blood levels to adversely affect the organ systems, a sufficient concentration of a particular local anesthetic agent must be absorbed into blood at a rate greater than at which it undergoes biotransformation, redistribution or elimination. With an inadvertent intravascular injection, the build up of the drug in the bloodstream is very rapid so that the biotransformation and elimination do not keep pace with it.

There are two types of drugs that can exhibit toxic reactions in dental practice. These drug reactions fall into two categories: (1) Local anesthetic overdose, and (2) vasoconstrictor overdose.

Local Anesthetic Overdose

The commonly used local anesthetic agent is 2% lidocaine. A 2% solution contains 2g /100 ml or 2000 mg / 100 ml or 20 mg / ml of local anesthetic agent. In India, a standard cartridge contains 2 ml of local anesthetic solution.

The recommended dose of local anesthetic agent with a vasoconstrictor is 7 mg/kg BW and not to exceed 500 mg. This dose will be contained in 25 ml or 12 and ½ cartridges) of the local anesthetic solution.

The recommended dose of local anesthetic agent without a vasoconstrictor is 4.4 mg / kg BW and not to exceed 300 mg. This dose will be contained in 15 ml or 7 and ½ cartridges) of the local anesthetic solution.

Causes of High Blood Level of Local Anesthetic Agent

The overdose resulting in high concentrations in brain; is usually, produced by:

1. *Intravascular administration:* Rapid injection directly into a blood vessel. To avoid such accidents, routine aspiration prior to deposition of local anesthetic solutions is essential.
2. *Impaired hepatic function:* The amide type of local anesthetic agents are metabolised in liver. Even a normal volume of local anesthetic agent may become potentially toxic in such patients, as because of hepatic dysfunction, the liver does not metabolise the local anesthetic agent resulting in its accumulation.
3. *Impaired renal function:* The final route of elimination of metabolised local anesthetic agent is excretion in urine. Patients with renal problems will be unable to eliminate these products; and will be predisposed to toxic accumulation of the agents.

Causes of High Blood Levels

- High blood levels are achieved in one or more of the following ways:
  i. Too large a dose of local anesthetic agent.
  ii. Rapid absorption of the drug.
  iii. Intravascular administration.
  iv. Slow biotransformation.
  v. Slow redistribution or elimination of the drug.
- Blood levels are necessary to create toxic overdose depend upon:
  i. General/physical condition of the patient at the time of injection.
  ii. Rapidity of injection.
  iii. Route of administration (e.g. inadvertent intravascular administration).
  iv. Amount of drug used.
  v. Age of the patient.

Systems Involved

The body systems mainly affected by toxic levels of circulating local anesthetic agent are (a) Central nervous system, (b) Cardiovascular system, and (c) Respiratory system.

Effects on Central Nervous System

The effects of build-up of local anesthetic agent or its breakdown products may occur in two distinct phases:

1. Initial CN system stimulation, followed by
2. A marked cerebral depression.

CNS contains two types of neurons:

1. Facilitatory neurons: These are the nerves that mediate function.
2. Inhibitory neurons: These are the nerves that modify function.
   a. *Non-toxic levels:* At non-toxic levels, the local anesthetic agent has no effect on CN system.
   b. *Mild toxic levels:* As mild toxic levels approach; the conduction of inhibitory neurons is blocked, resulting in unmodified action of facilitatory neurons (i.e. convulsion-like movements).
   c. *High toxic levels:* As the toxic levels increase, the facilitatory neurons are blocked, thus resulting in cessation of function. Certain amide type of local anesthetic agents such as lidocaine, affect primarily facilitatory neurons; hence depression is seen rather than excitation (de Jong, Robles, and Lorbin, 1969).

Stimulation

It is noted by symptoms ranging from increased anxiety, restlessness, hallucinations; to increased depth and rate of respiration, gagging, vomiting

and even the risk of severe cortical stimulation resulting in tremors and convulsions.

Depression

With medullary depression, these symptoms will fade away as there will be a lapse into unconsciousness, a drop in BP and a marked reduction in respiratory rate. Death would result from respiratory failure.

Clinical Manifestations of Local Anesthetic Overdose

a. Low to moderate overdose levels
   Signs: Confusion, apprehension, excitation, slurred speech, muscular twitching of face and extremities, nystagmus, and increased blood pressure, heart rate, respiratory rate.
   Symptoms: Headache, dizziness, blurred vision, unable to focus, ringing in ears, numbness of tongue and perioral tissues, drowsiness, disorientation, and loss of consciousness.
b. Moderate to high blood levels
   Signs and symptoms: Generalised tonic-clonic seizures, followed by: Generalised CNS depression, decreased blood pressure, heart rate, and respiratory rate.

Prevention

The best method to avoid toxic reactions is as follows:

i. Use smallest possible volume and lowest effective concentration of the drug.
ii. Limit dosages to reasonable quantities (use minimum effective dose).
iii. Local anesthetic solution should be injected slowly.
iv. Avoid intravascular administration. Aspiration should be performed prior to injection through the use of aspiration syringes.
v. Employ vasoconstrictor with local anesthetic agents, if not contraindicated.

Management of Severe Local Anesthetic Overdose

1. Stop the dental procedure
2. Position the patient supine with legs elevated
3. Seek medical assistance
4. Give reassurance to the patient
5. Institute Basic life Support (BLS), as indicated
6. Administer O_2
7. Monitor vital signs
8. Administer IV anticonvulsants (for prolonged seizures)
9. Allow patient to recover and then discharge
10. Consider additional management
11. If the patient fails to recover, then transfer the patient to a general hospital having ICU facility.

Additional Management Considerations

a. Convulsions: These are treated with IV infusion of 10 mg diazepam.
b. Rectal route: It is an alternative route when IV access is difficult.
c. Patient should be monitored for respiratory depression.
d. Cerebral depression is managed by:
    i. Elevation of foot part of dental chair,
    ii. Administration of O_2,
    iii. Respiratory, and
    iv. Circulatory support.
e. If supportive measures fail, and BP fails to respond, further hospital-based treatment may be required.
f. Psychomotor reactions: It usually due to fear of injection. This fear leads to a reflex dilatation of splanchnic blood vessels concomitant with cerebral anemia and hypoxia.

On recognition of these signs, the patient should be placed in supine position. Tight clothing should be loosened. If recovery is not seen forthcoming then a more serious medical problem should be considered.

Effects on Cardiovascular System

1. *Non-toxic levels:* No appreciable effects.
2. *Mild toxic levels:*
    i. Decrease in BP owing to decrease in peripheral resistance (Blood Pressure = Total Peripheral Resistance × Cardiac Output).
    ii. Decrease in myocardial contractility.
3. *Moderate toxic levels:* There is decreased myocardial contractility; since local anesthetic agents affect nerve conduction in the heart. This results in decreased HR and that will result in decrease in cardiac output (Cardiac Output = Heart Rate × Stroke Volume). This further reduces blood pressure.
4. *High toxic levels:* The local anesthetic agent can inhibit or completely block the intrinsic cardiac neuronal conduction system, resulting in cardiac standstill, (e.g. lidocaine therapy for arrhythmia).

At high toxic levels, the depression of intracardiac nerve conduction can result in (i) A-V dissociation, (ii) ventricular rhythm, (iii) ventricular fibrillation; and ultimately, (iv) cardiac standstill.

Effects on Respiratory System

The respiratory system is affected by CN system depression; which can result in respiratory arrest. Therefore, at toxic levels, both cardiac and respiratory arrest may be seen. All persons using these agents: (i) must have resuscitation equipment available, and (ii) must be trained in cardiopulmonary resuscitation (CPR).

Vasoconstrictor (Epinephrine) Overdose

Clinical Manifestations of Epinephrine Overdose

a. Small overdoses:
   Signs: Elevated blood pressure and heart rate.
   Symptoms: The initial clinical features of epinephrine overdose are related to stimulation of CN system, which include: Fear and anxiety, tension, restlessness, headache, tremor, perspiration, weakness, dizziness, pallor, palpitation and respiratory difficulty. This is known as "epinephrine reaction" (de Jong 1994).
b. Large overdoses: The manifestations include: Cardiac dysrhythmias, dramatic increase in both systolic and diastolic BP, which may lead to cerebral hemorrhage, ventricular fibrillation is possible but rare, anginal episodes may be seen in patients with coronary insufficiency.

Management

1. Stop the dental procedure
2. Position the patient upright
3. Give reassurance to the patient
4. Institute Basic Life Support (BLS), as indicated
5. Monitor vital signs
6. Summon medical assistance
7. Administer O_2
8. Wait for recovery
9. Discharge the patient.

Idiosyncratic Reactions

The condition is difficult to define; and as such, some individuals do not accept the existence of such a condition. Any reaction to a local anesthetic agent or any other drug, that cannot be classified as allergic or toxic reaction is often labelled as idiosyncrasy. Hence, when the signs and symptoms are strange or bizarre which cannot be associated with allergy or toxicity they are classified as idiosyncratic type of reactions. There could be presence of emotional factor.

Prevention

1. Preanesthetic evaluation is very important.
2. Precautions to be taken to protect the patient from injuring himself as a result of convulsive seizures, loss of consciousness, or similar reactions.
3. Psychotherapy may be employed, wherever indicated. It may reduce the role of interplay of emotional factors.
4. Proper and adequate premedication would be beneficial to the patients with emotional interplay.

Management

It depends upon signs and symptoms presented:
1. Position of the patient with legs slightly elevated.
2. Airway should be maintained.
3. Adequate oxygenation should be instituted.
4. Evaluate the circulation.
5. Administer parenteral fluids, if necessary.

EMERGENCY DRUGS

The role of a Dental Practitioner in the management of a medical emergency begins with prevention of such situations. This comprises of the following:

i. Thorough medical evaluation,
ii. Knowledge of the drugs presently taking, and
iii. Training in Basic Life Support (BLS), or Cardiopulmonary Resuscitation (CPR).

This helps the Dental Practitioner in providing skills to manage most of the medical emergencies, in terms of assessment, and if necessary, institution of Airway, Breathing and Circulation (ABC of CPR).

iv. Administration of emergency drugs.

The following is a list of drugs to remain available for anesthetic emergencies, with their dosages, routes of administration and their indications (Tables 22.4 and 22.5 and see Figs 22.1 and 22.2).

Table 22.4: List of Emergency Drugs

S. No	Drugs	Dosages	Routes of administration	Indications
1.	O_2	Sufficient quantity	100% inhalation	Almost any medical emergency, such as i. Cardiovascular system, and ii. Respiratory system
2.	Pentobarbitol (Nembutal)	As indicated	IM/IV	Toxic overdose
3.	Diphenhydramine (Benadryl)	20-30 mg	IV/IM	Allergic reactions
4.	Chlorpheniramine maleate	10-20 mg	IV/IM	Allergic reactions
5.	Epinephrine (Adrenaline)	0.3-0.5 mg 1 mg	IM IV	i. Anaphylactic reactions ii. Asthma iii. Cardiac arrest
6.	Glyceryl trinitrate	0.3-0.4 mg	Sublingual	*Angina pectoris*
7.	Isoproterenol hydrochloride	0.5% (1:200)	5-15 deep inhalations via nebulizer	Bronchial spasm
8.	Aminophylline	3.57 grain	IV	Asthmatic attacks
9.	Sodium bicarbonate		IV	Metabolic acidosis

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10.	Dextrose	50%	IV infusion	Severe hypoglycemia
11.	i. Decadron, or	4-12 mg	IV	i. Acute adrenal insufficiency
	ii. Hydrocortisone	100 mg	IV	ii. Allergic reactions iii. Anaphylactic reactions
12.	Mephentermine sulphate (Wyamine sulphate)	0.5-1 ml (7.5-15 mg)	IM/IV	Hypotension
13.	Atropine sulphate	0.5 mg	IV	Bradycardia
14.	Midazolam or	2 mg stat	IV	Convulsions
15.	Valium; Calmpose	5-10 mg	IV	Convulsions
16.	Thiopentone	25 mg 1 ml/kg		Convulsions

Table. 22.5. Essential emergency drugs

S. No.	*Indications*	*Drugs*	*Initial adult dose*
1	Almost any medical emergency	O_2	100% inhalations
2	(a) Anaphylaxis	Epinephrine (1:1000)	a. 0.1mg IV or 0.3-0.5 mg IM
	(b) Asthma unresponsive to albuterol		b. 0.1mg IV or 0.3-0.5 mg IM
	(c) Cardiac arrest		c. 1.0 mg IV
3	*Angina pectoris*	Nitroglycerine	0.3-0.4 mg sublingually
4	Allergic reactions	Antihistaminic (Diphenhydramine,	25-50 mg IV/IM
		or Chlorpheniramine Maleate)	10-20 mg IV/IM
5.	Asthmatic bronchospasm	Albuterol inhalar	2 sprays (180-200 µg) inhalations

Dosages

Pediatric doses (approximate, not to exceed the adult doses listed above) Epinephrine 0.01 mg/kg, Diphenhydramine 1.0 mg/kg, Albuterol: 1 spray (90 - 100 µg). Adult dose is 1.2 mg PO. 1 -2 hours before the procedure. *Preoperative medications:* Antihistamines: Atropine: 0.6 mg, or Glycopyrrolate 0.2 mg IM one hour before surgery. Tab. Alprazolam (0.25 mg, 0.5 mg) in the night before the surgery (Table 22.6).

Table. 22.6. Supplementary emergency drugs

S. No.	*Indications*	*Drugs*	*Initial adult dose*
1	Hypoglycemia in unconscious patient	Glucagon	1 mg IM
2	Clinically significant bradycardia	Atropine	0.6 mg IV or IM
3	Clinically significant hypotension	Ephedrine	5 mg IV or 10-25 IM
4	(i) Adrenal insufficiency (ii) Recurrent anaphylaxis	Hydrocortisone	100 mg IM or IV

EQUIPMENT USED FOR TREATMENT OF COMPLICATIONS

The complications related to the use of local anesthetic agents do occur. Hence, it is necessary that certain basic armamentarium should always be readily available in the dental office. This includes (Figs 22.3 to 22.17):

Emergency Tray

The tray contains syringes, needles, ampoules and vials of emergency drugs. The tray should be checked frequently and the drugs or the part of the equipment that is used should be replaced immediately.

a. *Emergency drugs:* The expiry dates of the drugs should be charted out and are replaced as and when required.
b. *Hemostats:* These are useful in case of breakage of the needles. The protruding end of the needle is grasped with the hemostat and is taken out.
c. *Oral and nasopharyngeal airways:* These airways are available in various sizes and help to keep the airway patent and protect the tongue.

Oxygen

Oxygen along with the necessary equipment for its administration, should preferably be available in the dental office.

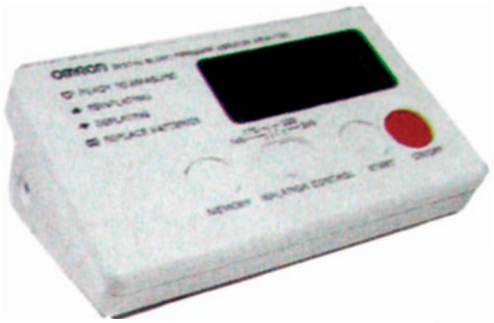

Fig. 22.3: Blood pressure monitors: Automatic inflation blood pressure monitor with pulse:
- Self-inflating and deflating D-ring contour oscillometric cuff
- LCD panel

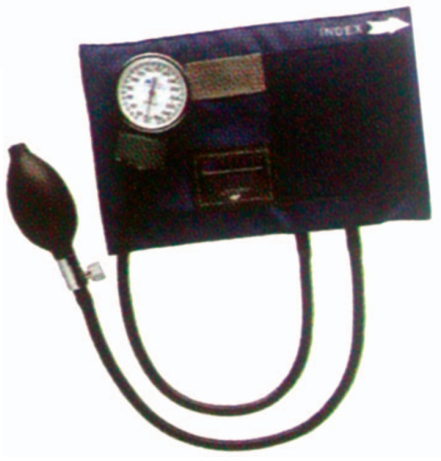

Fig. 22.4: Blood pressure monitors: Caliber aneroid sphygmomanometer

Fig. 22.5: Blood pressure monitors: Digital blood pressure monitor with pulse

Fig. 22.6: Stethoscope: Classic II stethoscope

Fig. 22.7: Pulse oximeter: Allied performs fast and accurate oximetry in a heartbeat

- Allied's a hand-held pulse oximeter
- Allied's light weight, high performance pulse oximeter provides reliable SpO2, and pulse rate measurement. It determines SpO2 and pulse rate by passing two wavelengths of light, one red, and one infrared through body tissues to a photodetector. The pulse oximeter processes these signals to identify the pulse rate and calculate O2 saturation.
- Built-in memory
- Spot check readings are accurate for up to 99 patients

Fig. 22.8: Pulse oximeter: Palco

- A portable, hand-held unit designed to non-invasively monitor arterial blood gas (O2) saturation
- 3-digit LED display shows percent O2 saturation
- 3-digit LED display shows pulse rate in beats/minute
- A red LED flashes in synchrony with the pulse
- Complete with sensor of your choice

Fig. 22.9: Emergency resuscitator: MADA

- Adult: Features pliable thin wall construction for exceptional long compliance sensitivity

Fig. 22.10: Emergency resuscitator

Features:

- One-way valve - provides rescuer protection
- Two-face masks - allows good patient fit
- Oxygen inlets - allows the use of supplementary O2 when available
- Head strap - keeps unit in place
- Quality clear vinyl
- Single use - allows observation of patient's condition

Fig. 22.11: Single oxygen unit resuscitator contents:
- 1 no. 1303Me Mada Cylinder III (210 liters)
- 1 no. 1308A fixed flow regulator (6 LPM AVG)
- 1 no. 1429 manual resuscitator, mask and tube in carrying case

Fig. 22.12:
Ambu's bag: Ambu spur bag—Disposable

Fig. 22.13. Airways: Brook airway professional tube
- Reusable
- For mouth-to-mouth artificial respiration
- With non-return valve

Fig. 22.14:
EPR armamentarium: CPR mask with 3M filter

Fig. 22.15:
CPR armamentarium: CPR microshield
- Physical barrier for mouth-to-mouth resuscitation

Figs 22.3 to 22.15: Emergency equipment

Fig. 22.16: Dental emergency kit

- 131/4"x93/4"x21/4"
- Designed specifically for dental needs, this kit conveniently provides pre-filled syringes for rapid action.

Features:

Color-coded Emergency Drug Kit with Drug Manufacturer's recommendations, with optional Automatic Drug Refill service available

Contents: 2 Ampoules epinephrine, 1 Pre-filled epinephrine,1:1000 Syringe, 3 Ammonia Inhalants, 2 Packs Aspirin, 2 Vials Diphenhydramine, 1 Nitrolingual Pump Spray, 1 tube Glucose 15, 1 Albuterol Inhaler, 2 3 ml Disposable Safety Syringes, 1 CPR Pocket Mask, 1 Airway, and 1 16" Latex-free Tourniquet

Fig. 22.17: Emergent-EZ kit: kit is designed specifically for medical and dental needs. It is an economical kit, providing the drugs in low-cost, easy to fill ampoules. It is color-coded with drug manufacturers' recommendations.

Contents:

2 Ampoules epinephrine, 1 Albuterol Inhaler, 3 Ammonia Inhalants, 2 Amyl-Nitrate Inhalants, 2 Ampoules Atropine, 1 Bottle Nitrostat, 1 Mix-o-vial Solu-Cortef, 1 Ampoule Talwin, 1 Ampoule Tigan, 2 vials Midazolam. 2 Ampoules Ephedrine, 2 Vials Diphehydramine, 1 tube Glucose, 2 3 ml Disposable Safety Syringes, 1 16" Latex-free Tourniquet. 1 CPR Mask, and 1 Plastic Airway

Figs 22.16 and 22.17: First Aid kits

Manual Resuscitators

Such as Ambu bag or Resussi folding bag, should be available in the dental office. These are inexpensive and efficient means of producing artificial ventilation of the lungs.

Blood Pressure Recording Devices

Such as sphygmomanometer, and stethoscope.

Suction Apparatus

Source of suction, or suction catheters or suction tubing with mastoid or tonsillar tip or Yankauer suction.

Other Apparatus

Thermometer, injection swabs, adhesive tape, dressing scissors, and IV infusion sets, etc.

Medical Emergencies in the Dental Office			
S. No.	***Emergencies***	***Specific diagnostic signs***	***Management***
1.	Vasodepressor syncope/ Syncope / Fainting / Vasovagal attack	Pallor, sweating, cold and clammy skin, giddiness, feeble pulse, turning of the eyes, visual disturbances, increased respiratory depth, bradycardia, hypovolemia, semi-consciousness or unconsciousness	**Step I** Stop the dental procedure **Step II** • Position of patient: Put the patient in Head-low position (with legs slightly elevated) • Splash/sprinkle cold water on the face • Ask patient to take deep breath • Loosen tight clothing **Step III** • Inhalation of aromatic vaporable poured on ammonium bicarbonate, or smelling salt; or • Oral aromatic spirit of ammonia; 2 to 4 ml in an ounce of water, or • Cold or wet towel applied on the forehead **Step IV** • Maintain airway • Administer O_2 • Call for medical assistance • Monitor vital signs **Step V** If the condition does not improve: • Check pulse and blood pressure • Check the level of consciousness **Step VI** • Inj. Atropine 0.6 mg/ml IV • Inj. Mephentine 10-30 mg. IM by IV drip in NS, or • RL solution, or IV 6-12 mg

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S. No.	Emergencies	Specific diagnostic signs	Management
			Step VII • Have the patient escorted home • Plan anxiety control measures during future dental care • Consult patient's physician prior to future dental treatment
2.	Hypertension	Headache, restlessness increased pulse rate, shortness of breath, palpitations	• Tab. Nifedipine 5-10 mg given sublingually or sprayed on the nasal mucosa with syringe subcutaneously after puncturing, or • Tab. Inderal 40 mg stat. orally, or • Tab. Aten 25-50 mg, or • Tab. Captopril 2.5-5 mg sublingually • Nitroglycerine patch; applied on the skin of forehead • Administer O_2 • Call for medical assistance
3.	Hypotension	Pallor, giddiness, sweating, lower pulse rate (thready) Severe Hypotension?	• IV Fluids: Normal saline • Inj. Mephentine 10-30 mg IM, or by IV drip in NS or Direct IV 6 mg • Inj. Ephedrine 30 mg/ml IM, 5mg IV • Inj. Efcorline 100 mg. IM, or • Inj. Decadron 8 mg. IM / IV (4 mg/ml) • Call for medical assistance • Ephedrine 5 mg IV, dose is increased in increments, or 10-25 mg IM
4.	Angina Pectoris	Crushing pain in chest (left side precordium or substernum), radiating to left shoulder, left hand and sometimes even to jaw.	• Tab. Nitroglycerine (Angised) 0.5 mg given sublingually; or • Tab. Isosorbide Dinitrate (Sorbitrate) 20 mg given sublingually followed by 3 times /day • Administer O_2 100% inhalations • Call for medical assistance

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S. No.	Emergencies	Specific diagnostic signs	Management
			• Nitroderm ointment: apply ½" over praecordium or forehead— 0.4 mg metered aerosol • Consult patient's physician for further dental treatment
5.	Hypoglycemic coma (known diabetic)	a. Anxiousness, sweating, hunger, headache, diplopia, convulsions, palpitations, unconsciousness (even in an unknown diabetic going into coma, give same treatment irrespective of whether he is hyperglycemic or hypoglycemic)	**Step I** Stop the dental treatment **Step II** a. If conscious: • Give Glucose powder (4 sugar lumps) in a glassful of water; orally or fruit juices • Monitor vital signs b. If unconscious: • Administer Dextrose / Glucose; 50 ml of 50% solution IV • Monitor vital signs • Call for medical assistance • Administer O_2 100% inhalations • Prepare for transfer to a nearby hospital with an ICU facility **Step III** • Consult patient's physician for prior to further dental treatment
		b. Hypoglycemia in unconscious patient	• Administer Glucagon 1mg / ml IM
6.	Toxic reactions: Drug-overdose a. Local anesthetic agent	a. *Stimulation:* Mild restlessness to severe convulsions b. *Depression:* Confusion, respiratory failure, cardiac arrest (drowsiness to loss of consciousness)	• Position of patient: Put the patient in Head-low position (with legs slightly elevated) • Administer O_2 100% inhalations • Keep airway patent • Call for medical assistance

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S. No.	Emergencies	Specific diagnostic signs	Management
			a. Stimulation: • Inj. Diazepam 5-10 mg/ml, or • Inj. Midazolam 1-2 mg IV b. *Depression:* Inj. Nikethamide (coramine)
7.	Toxic reactions (drug-overdose) (b) Vasoconstrictor agent	Increased heart rate Increased BP	**Step I** • Place the patient in semireclined position • Call for medical assistance • Keep airway patent • Administer O_2 100% inhalations • Tab. Nifedipine 5 mg sublingually • Nitroglycerine patch **Step II** • Tachycardia—carotid massage—pressure on the left carotid sinus • Bradycardia—atropine 0.6 mg IV
8.	Acute anaphylactic shock / Anaphylactic reaction/Anaphylaxis	Urticaria, angioneurotic edema, dyspnea, wheezing, asthma, vomiting, cramps, hypotensive shock, pallor, decrease in BP, loss of consciousness, death, if treatment is delayed or inappropriate	**Step I** • Stop administration of all drugs **Step II** • Position: Put the patient in Head-low position • Call for medical assistance **Step III** • Inj. Adrenaline 1:1000. 0.5-1 ml IM. Repeat after 15 minutes, if necessary, until patient responds. • Inj. Efcorline 100 mg IM, or • Inj. Decadron 8 mg IM (4 mg/ml)

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S. No.	Emergencies	Specific diagnostic signs	Management
			• Administer O_2, if necessary • Initiate CPR (Basic Life Support) • Monitor vital signs • Maintain airway • Inj. Diphenhydramine 25-50 mg IM or IV, or • Inj. Chlorpheniramine 10-20 mg (10 mg / ml) (Inj. Avil 2 ml IM-22.75 mg/ml) **Step IV** • Prepare for transfer to a nearby hospital with an ICU facility
9.	Tetany Ca-deficiency/ Parathyroid crisis	Carpopedal spasm (turning of feet and hands towards midline of body)	• Inj. Ca - Gluconate 10 ml of 10% IV very slowly • Call for Medical assistance
10.	Epileptic fits (Convulsions)	Tonic and clonic muscle spasm; convulsions, frothing of saliva from mouth, clenching of teeth (trismus).	**Step I** • Position: Put the patient in prone position or lateral position • Keep the airway clear **Step II** • Inj. Diazepam 5-10 mg IM, or Inj. Midazolam • Inj. Gardenal 50-100 mg IM, or • Inj. Epsoline 200 mg • Inj. Mephentine 10-30 mg IM • Administer O_2 100% inhalations • Inj. Epsoline 200 mg IV/IM • Call for medical assistance • Consult patient's physician for further dental treatment

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S. No.	Emergencies	Specific diagnostic signs	Management
11.	Acute adrenal insufficiency (Adrenal crisis)	Pallor, rapid weak pulse, feeling of weakness, cold and clammy skin, hypotension, loss of consciousness	a. H/O drugs taken during past two years: Give similar dose or double the dose at the time of procedure. b. H/O abrupt stopping of the drugs: Give similar dose at the time of procedure, with the physician's consent. c. H/O gradual tapering off: Give similar dose. **Step I** • Terminate all dental treatment **Step II** • Position: Place the patient in supine position with legs elevated above the level of head • Call for medical assistance • Inj. Hydrocortisone 100-200 mg IM/IV • Administer O_2 • Monitor vital signs • Prepare for transfer to a nearby hospital with an ICU facility
12.	Bronchial asthma (Acute asthmatic attack)	Difficulty in breathing, shortness of breath, wheezing, restlessness	**Step I** • Stop the dental procedure **Step II** • Position: Put the patient in fully sitting posture **Step III** • Administer bronchodilator by spray Bronchodilator Inhaler: Nebulizer (salbutamol) (asthalin) 100-200 mg, i. Albuterol—1-2 dose / spray (90-100 mcg of dose / spray or 180-200 mcg of dose / spray)

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S. No.	Emergencies	Specific diagnostic signs	Management
			ii. Metaproterenol (Alupent); repeat, if necessary iii. Terbutaline (Bricaryl) • Administer O_2 • Monitor vital signs **Step IV** If signs and symptoms not relieved • Inj. Deriphylline 2 ml IM, or • Inj. Aminophylline 250 mg IV slowly (diluted in 20 ml of NS) • Inj. Adrenaline 0.5-1 ml of 1:1000 IM • Inj. Betnesol 1 ml IM, or • Inj. Cortisone 100 mg IV, or equivalent • Prepare for transfer to a nearby hospital with an ICU facility • Consult patients physician for further dental treatment
13.	Hyperventilation syndrome	a. Neurologic: Dizziness, tingling or numbness of fingers, toes and lips b. Respiratory: Increased rate and depth of breath, shortness of breath c. Cardiac: Palpitations and tachycardia d. Muculoskeletal: Myalgia, muscle spasm e. Psychological: Extreme anxiety	**Step I** Stop the dental procedure **Step II** • Position: Put the patient in fully upright position • Reassurance: Attempt to verbally calm the patient **Step III** • Have the patient breathe CO_2-enriched air such as in and out of a paper bag. • Guided breathing: The patient may be guided to briefly breath, hold after each exhalation, thus slowing down the Respiratory Rate

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S. No.	Emergencies	Specific diagnostic signs	Management
			Step IV If symptoms persist or worsen: • Inj. Diazepam 10 mg IM, or titrate slowly IV until anxiety is relieved, or • Inj. Midazolam 0.5 mg IM, or titrate slowly IV, 1 mg until anxiety is relieved. • Monitor vital signs **Step V** • All future dental surgery procedures may be performed using Anxiety Reduction Protocol (ARP)
14.	Hypoventilation	Anxiety	• Administer O_2 through Ambu bag with Reservoir bag
15.	Respiratory distress	Difficulty in breathing	• Administer O_2 (100%) through Ambu bag • Inj. Coramine 2 ml IM • Inj. Doxapram 2 ml IM • Call for medical assistance • Consult patients physician for further dental treatment • Place the patient in an upright position, to sit forward with legs over the side of the chair, and hands on the knees. • If the patient has been given sedation prior to dental treatment, maintenance of airway is essential; this can be achieved by pulling the tongue out.

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S. No.	Emergencies	Specific diagnostic signs	Management
16.	Cardiac arrest	Sudden pallor, respiratory distress, sudden loss of consciousness, absence of arterial pulse; absence of heart beat and unrecordable BP	• Put the patient on a firm flat surface; if possible, on the floor. • Clear the airway. • Establish an IV line. • Administer O_2 (100%) through Ambu bag • Perform CPR • External cardiac massage; and defibrillator • Inj. Adrenaline I ml of 1:1000 (intracardiac) • Repeat external cardiac massage. • Once sinus rhythm is achieved; Give Inj. Decadron 8 ml IV • If severe asystole / bradycardia; Give Inj. Atropine 0.6 mg IV • Inj. Lidocaine 1 mg / kg • Inj. Sodium bicarbonate (1 mcg / kg)
17.	Bleeding	Gingival bleeding, post-extraction hemorrhage	a. Local measures: i. Mechanical pressure ii. Local adrenaline pack (1:1000) iii. Hemostatic agents: tincture of feri-perchlor, gelfoam, surgicel, feracrylum (supraheal, uniheal) bone wax, etc. iv. Electrocautery v. Suturing b. Systemic measures: i. Oral • Tab. Clauden 125 mg. 1-2 tabs. 3-4 / day • Tab. Styptobion 1-2 tab. 3 / day • Tab. Styptocid 125 mg 1-2 tab. 2-3 / day • Cap. CVP (citrous bioflavonoid compound) 100 mg and Vit C 100 mg. • Cap. CVP forte CVP 300 mg and Vit C 300 mg. • Tab. Ethamsylate 500 mg TID

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S. No.	Emergencies	Specific diagnostic signs	Management
			• Tab. Hexalan. Amicar 1 tab. 3 / day (EACA) • Tab. Vit K 10 mg TID • Tab. Tranexamic acid 250 and 500 mg 2-3 times/day ii. Parenteral • Inj. Haemocid 1g 3 / day IM/IV • Inj. Clauden 2.5 to 5 ml at site IV/IM • Inj. Styptobion 2-4 ml daily IV • Inj. Amicar 20 mg IV. • Inj. Styptocid 2 ml before the procedure; followed by 2 ml 6 hourly IM • Inj. Bothropace 2 ml every 4-6 hourly IM • Inj. Stadren 2 ml stat; repeat after 30 min. • Inj. Ethamsylate 250-500 mg during and after surgery; followed by 250-500 mg 6-12 hourly IM • Inj. Vit. K 10 mg/ml IM followed by 3 tabs/day • Inj. Feracrylum (supraheal, uniheal), 1% undiluted solution to be applied directly or poured over the bleeding surface. • Inj. Tranexamic acid 500 mg in 5 ml
18.	Severe dental pain		• Inj. Dynapar 75 mg / ml IM, or • Inj. Voveran 50-75 mg IM (25 mg/ml)(75 mg/3 ml), or • Inj. Tramazac IM (50 mg/ml), or • Inj. Ketorolac 3 ml IM, or • Inj. Fortwin 30 mg 1 ml IM

Paediatric Doses: Epinephrine 0.01 mg / kg (1:1000)

The names of the drugs mentioned here are for the benefit of the readers. The author and the publisher do not have any financial relationship with the companies manufacturing those drugs.

BIBLIOGRAPHY

1. Cawson RA and Odell EW. Emergencies in Dental Practice, in Essentials of Oral Pathology and Oral Medicine. 6th ed. Churchill and Livingstone 1998.
2. Chitre AP. Emergencies in Dental Practice. JIDA 1982-83.
3. CIMS: Jan- Apr 2008.
4. Covino BG. "Toxicity of local anesthetics". In: Stoelting RK, Barash PG, Gallagher TJ (Eds): "Advances in Anesthesia" 1986;3:37-65.
5. de Jong RH. Vasoconstrictors. In de Jong RH, editor: Local anesthetics, St Louis 1994;158-60.
6. Fridrich KL, Kirk G. "Dental Complications". In: Longnecker DE, Tinker JH, Morgan GE Jr (Eds): "Principles and Practice of Anesthesiology". 2nd ed. 2451-60.
7. Friedberg CK. Syncope: Pathological physiology, differential diagnosis and treatment, Mod Concepts Cardiovasc Dos 1971;40:55.
8. Glick G, Yu PN. Hemodynamic changes during spontaneous vasovagal reactions. Am J Med 1963;34:42.
9. Holzman RS, Hirshman CA. "Anaphylactic Reactions and Anesthesia". In: Longnecker DE, Tinker JH, Morgan GE Jr (Eds): "Principles and Practice of Anesthesiology". 2nd ed. 2385-2408.
10. Howe GL. Minor Oral Surgery. 3rd ed. Butterworth-Heinemann Ltd. Reprint. Mumbai: Varghese Publishing House 1985.
11. Killey HC, Seward GR, Kay LW. An Outline of Oral Surgery Part II, Dental Practitioner Handbook, (Wright) Bombay: Varghese Publishing House 1992.
12. Laskin DM. Oral and Maxillofacial Surgery Vol I and II. CV Mosby Co. AITBS Publishers India 1992.
13. Malamed SF. Handbook of Local Anesthesia. 4th ed. Mosby 2001.
14. Malamed SF. Medical Emergencies in the Dental Office. 4th ed. Mosby Year book 1993.
15. Martin GJ, Adams SL, Martin HG, others. Prospective evaluation of syncope. Ann Emerg Med 1984;13:499.
16. Michael Ferrante F. "Pharmacology of Local Anesthetics". In: Longnecker DE, Tinker JH, Morgan GE Jr (Eds): "Principles and Practice of Anesthesiology". 2nd ed 1330-60.
17. Miller RD. "Management of Anesthetic Emergencies". In: "Atlas of Anesthesia-Principles of Anesthetic Technique and Anesthetic Emergencies. 1997;vol IV;11.14 -11.15.
18. Peterson LJ, Ellis EIII, Hupp JR, Tucker MR. Contemporary Oral and Maxillofacial Surgery. 2nd ed. Harcourt Brace and Central Asia. Pvt. Ltd1997.
19. Reutz PP, Johnson SA, Callahan R. Fainting: A review of its mechanism and a study in blood donors. Medicine 1967;46:363.
20. Richard Bennett, C. Monheim's Local Anesthesia and Pain Control in Dental Practice. 7th ed, CBS Publishers and Distributors, Delhi, India 1990.
21. Roberts DH, Sowray JH. Local Analgesia in Dentistry. 3rd ed. Wright, Bristol 1987.
22. Satoskar, RS, Bhandarkar, SD, and Ainapure, SS. In Satoskar, Kale, and Bhandarkar's Pharmacology and Pharmacotherapeutics, Revised 16th ed. Popular Prakashan, Mumbai, India.
23. Scully C and Cawson RA. Management of Emergencies in the Dental Surgery, in Medical Problems in Dentistry, 2nd ed. Wright, Bristol 1987.

24. Scully C. The prevention and management of emergencies. BDJ Launchpad. 1997;18-22.
25. Seward GR, Harris M, McGowan DA. Killey and Kay's Outline of Oral Surgery. Part I Dental Practitioner Handbook (Wright). Mumbai: Varghese Publishing House 1992.
26. Weiss ME, Hirshman CA. "Allergic drug reactions-pathophysiology and management"-"A Practice of Anesthesia", Wylie and Churchill-Davidson's, Sixth ed 1995;516-43.
27. Wright KE, McIntosh HD. Syncope: A review of pathophysiological mechanisms. Prog Cardiovasc Dis 1971;13:58.

Chapter 23

National AIDS Control Organization (NACO)

Guidelines for Occupational Post-Exposure Prophylaxis (PEP)

INTRODUCTION

The incidence of HIV/AIDS infected individuals is alarmingly rising globally. Though there are other major etiological factors, the Dental Surgeon and Dental Health Care Workers (DHCWs) are constantly exposed to the probably infected patients' blood and saliva. In spite of taking necessary precautions like wearing the gloves, the members of the dental team are at a danger of inflicting a needle-stick injury inadvertently and expose themselves to AIDS during their professional careers.

With this scenario it is essential for the dental professionals to be aware of not only the precautions but how to take care of themselves and their colleagues in case of a mishap. Hence, it is essential to know the protocol in case of such an eventuality, as recommended by National AIDS Control Organization (NACO).

It is earnestly urged that the dental personnel should take utmost care in handling the syringes and needles, and at the same time, should not take such injuries lightly and ignore them or at the same time get unnecessarily panicky. The Post-Exposure Prophylaxis (PEP) guidelines for occupational exposure in the dental office should be properly understood and followed.

REVISED CLASSIFICATION OF HIV-ASSOCIATED ORAL LESIONS (WHO, 1990)

Group I—Lesions strongly associated with HIV infections:

1. Candidiasis: Erythematous, hyperplastic, and pseudomembranous,
2. Hairy leukoplakia (EBV),
3. HIV-gingivitis,
4. Necrotising ulcerative gingivitis,
5. HIV-periodontitis,
6. Kaposi's sarcoma, and
7. Non-Hodgkin's lymphoma.

Group II—Lesions less commonly associated with HIV infection:

1. Atypical ulceration (oropharyngeal).
2. Idiopathic thrombocytopenic purpura.
3. Salivary gland diseases:
   a. Dry mouth due to decreased salivary flow rate and
   b. Unilateral or bilateral swelling of major salivary glands.
4. Viral infections (other than EBV):
   i. Cytomegalovirus
   ii. Herpes simplex virus
   iii. Human papilloma virus (warty like lesions):
      a. Condyloma acuminatum,
      b. Focal epithelial hyperplasia, and
      c. Verruca vulgaris
   iv. Varicella-zoster virus:
      i. Herpes zoster virus, and
      ii. Varicella.

Group III—Lesions possibly associated with HIV infection:

1. Bacterial infections excluding gingivitis/periodontitis:
   a. Actinomycosis,
   b. *Enterobacter cloacae,*
   c. *Escherichia coli,*
   d. *Klebsiella pneumoniae,*
   e. *Mycobacterium avium intracellulare,* and
   f. Tuberculosis
2. Cat-scratch disease
3. Exacerbation of apical periodontitis
4. Fungal infection other than candidiasis: Cryptococcosis, Geotrichosis, Histoplasmosis, and Mucormycosis.
5. Melanotic hyperpigmentation
6. Neurologic disturbances: Facial palsy, and trigeminal neuralgia
7. Osteomyelitis
8. Sinusitis
9. Submandibular cellulitis
10. Squamous cell carcinoma and
11. Toxic cell epidermolysis.

Manifestations

The WHO report on AIDS (1990), describes the various signs and symptoms of HIV disease/AIDS, as follows:

1. Earlier signs of HIV infection are as follows:
   i. Unexplained weight loss of more than 10% of body weight.
   ii. Unexplained fever lasting more than one month.

iii. Unexplained chronic diarrhea.
iv. Shingles (caused by herpes zoster virus).
v. Oral thrush (infection by a fungus, *C albicans*).
vi. Oral hairy leukoplakia.
vii. Persistent generalized lymphadenopathy (PGL). Persistent increase (more than three months) in the size of the lymph nodes in several sites of the body; such as neck, axilla and groins.

2. Later signs of HIV infection (AIDS) are as follows:

Approximately 30-40% of seropositive patients will develop AIDS within seven years after infection. The manifestations appear when the immune system is severely damaged. AIDS is responsible for two main categories of disease: (a) Opportunistic infections, and (b) Certain tumors.

a. *Opportunistic infections:* The clinical signs vary according to the organism responsible and the organs affected. The principal organs affected are the lungs, gastrointestinal tract, brain and skin. The following is a list of the main signs of opportunistic infections more specifically diagnostic of AIDS.
   i. *Lungs:* Cough, shortness of breath, and chest pain.
   ii. *Gastrointestinal tract:* Difficulty in swallowing, nausea, vomiting, abdominal pain, severe weight loss (malabsorption), and chronic severe diarrhea.
   iii. *Brain:* (a) Headache, impaired mental function, fits, peripheral and central paralysis, incoordination and coma (b) Visual defects.
   iv. *Skin/Mucocutaneous:* (a) Perioral and oral ulceration, and (b) Genital and perianal ulceration.

b. *Certain tumors:* (i) Kaposi's sarcoma and (ii) Lymphomas.
   i. *Kaposi's sarcoma:* Generalised Kaposi's sarcoma is frequently associated with AIDS. It affects about 15% of patients with AIDS in Africa. It presents in the form of purple or red/brown cutaneous plaques or nodules. These lesions are found not only in the skin, but also found in the mucosal lining, such as in the mouth and in the lungs, and gut. A characteristic clinical appearance is of one or more erythematous or violaceous macules or swellings with or without ulceration. It is predominantly seen in the palate or in the gingivae.
   ii. *Lymphomas:* The risk of lymphoma (tumors of the lymph nodes, skin, gut and brain) is about 100 times greater in patients with AIDS than in normal subjects. The commonly seen tumor is Non-Hodgkin's lymphoma. It presents in the form of a firm, elastic, reddish or purplish swelling, with or without ulceration. The most common sites are gingiva and palate.

If universal precautions are undertaken, the infection control procedures will not change, due to patient's positive history of HBV or HIV. Referral to patient's physician for serological confirmation may be considered, if the results of such tests will help the dental practitioner in the management of the patient's overall dental needs.

Prevention of Occupational Exposure

Standard precautions (universal precautions) and safe practices:

- Wash hand after patient contact, removing gloves.
- Wash hands immediately if hands contaminated with body fluids.
- Wear gloves when contamination of hands with body substances anticipated.
- Protective eyewear and masks should be worn when splashing with body substance is anticipated.
- All health care workers should take precautions to prevent injuries during procedures and when cleaning or during disposal of needles and other sharp instruments.
- Needle should not be recapped.
- Needles should not be purposely bent or broken by hand.
- Needles are not removed from disposable syringe nor manipulated by hand.
- After use disposable syringes and needles, scalpel blades and other sharp items should be placed in a puncture resistant container.
- Health care workers who have exudative lesions or dermatitis should refrain from direct patient care and from handling equipment.
- All needle-stick injuries should be reported to infection control officer.
- Handle and dispose off sharps safely.
- Clean and disinfect blood/body substances spills with appropriate agents.
- Adhere to disinfection and sterilization standards.
- Regard all waste soiled with blood/body substance as contaminated and dispose off according to relevant standards.
- Vaccinate all clinical and laboratory workers against Hepatitis B.

Use of Protective Barriers (Table 23.1)

Protective barriers reduce the risk of exposure of the Dental Health Care Workers (DHCWs) skin or mucous membrane to potentially infective materials. Protective barriers include gloves, gowns, masks, protective eyewear.

Table 23.1. Selection of protective barriers

Type of exposure	*Examples*	*Protective barriers*
Low-risk		
Contact with skin with no visible blood	Examination of oral cavity Intraoral Dental charting	Gloves helpful but not essential
Medium-risk		
• Probable contact with blood • Splash unlikely	Use of Airotor, Scaling and Polishing, Root canal treatment, fillings (class II), crown preparation, local anesthetic injection, extraction of teeth, minor oral surgery, spills of blood	Gloves, gowns and aprons may be necessary
High-risk		
• Probable contact with blood, splashing, uncontrolled bleeding	Major surgical procedures, particularly in oral surgery and periodontal surgery;	Gloves, waterproof Gown or apron EyewearMask

The use of double gloves for DHCWs is not recommended. Heavy duty rubber gloves should be worn for cleaning instruments, handling soiled linen or when dealing with spills.

NACO GUIDELINES FOR POST-EXPOSURE PROPHYLAXIS (PEP)

OCCUPATIONAL EXPOSURE

Definition

Occupational exposure refers to exposure to potential blood-borne infections (HIV, HBV and HCV) that may occur in health care settings during performance of job duties. Post-exposure prophylaxis (PEP) refers to comprehensive medical management to minimise the risk of infection among Health Care Personnel (HCP) following potential exposure to blood-borne pathogens (HIV, HBV, HCV). This includes counseling, risk assessment, relevant laboratory investigations based on informed consent of the source and exposed person, first aid and depending on the risk assessment, the provision of short-term (four weeks) of antiretroviral drugs, with follow-up and support.

Who is at Risk?

All Health Care Personnel, including emergency care providers, laboratory personnel, autopsy personnel, hospital employees, interns and medical students, nursing staff and students, physicians, surgeons, dental surgeons, hygienists, and dental assistants, labor and delivery room personnel, laboratory technicians, health facility sanitary staff and clinical waste handlers and health care professionals at all levels. Also at risk are public safety workers, including law enforcement personnel, prison staff, fire-fighters, workers in needle exchange program and workers in HIV programs.

What is the Risk?

Health Care Personnel are at risk of blood-borne infection transmission through exposure of a percutaneous injury (e.g. needle-stick or cut with a sharp instrument), contact with the mucous membranes of the eye or mouth of an infected person, contact with non-intact skin (particularly when the exposed skin is chapped, abraded, or afflicted with dermatitis or contact with blood or other potentially infectious body fluids.

The average risk of acquiring HIV infection from different types of occupational exposure is low compared to risk of infection with HBV or HCV. In terms of occupational exposure the important routes are needle stick exposure (0.3% risk for HIV, 9-30% for HBV and 1-10% for HCV) and mucous membrane exposure (0.09% for HIV).

What is Infectious and What is Not?

Exposure to blood, semen, vaginal secretions, cerebrospinal fluid, synovial, pleural, peritoneal, pericardial fluid, amniotic fluid and other body fluids contaminated with visible blood can lead to infection. Exposure to tears, sweat, saliva, urine and faeces is non-infectious unless these secretions contain visible blood.

Step 1: First Aid in Management of Exposure

For Skin—if the Skin is Broken After a Needle-stick or Sharp Instrument

- Immediately wash the wound and surrounding skin with water and soap, and rinse. Do not scrub.
- Do not use antiseptics or skin washes (bleach, chlorine, alcohol, betadine).

Table 23.2: Don'ts
• Do not panic • Do not put pricked finger in mouth • Do not squeeze wound to bleed it • Do not use bleach, chlorine, alcohol, betadine, iodine or any antiseptic or detergent

After a Splash of Blood or Body Fluids on Unbroken Skin

- Wash the area immediately
- Do not use antiseptics.

For the Eye

- Irrigate exposed eye immediately with water or normal saline. Sit in a chair, tilt head back and ask a colleague to gently pour water or normal saline over the eye.
- If wearing contact lens, leave them in place while irrigating, as they form a barrier over the eye and will help protect it. Once the eye is cleaned, remove the contact lens and clean them in the normal manner. This will make them safe to wear again.
- Do not use soap or disinfectant on the eye.

For Mouth

- Spit fluid out immediately
- Rinse the mouth thoroughly, using water or saline and spit again. Repeat this process several times
- Do not use soap or disinfectant in the mouth
- Consult the designated physician of the institution for management of the exposure immediately.

Step 2: Establish Eligibility for PEP

The HIV sero-conversion rate of 0.3% after an AEB (for percutaneous exposure) is an average rate. The risk of infection transmission is proportional to the amount of HIV transmitted, which depends on the nature of exposure and the status of the source patient. A baseline rapid HIV testing of exposed and source person must be done for PEP. However, initiation of PEP should not be delayed while waiting for the results of HIV testing of the source of exposure. Informed consent should be obtained before testing of the source as per national HIV testing guidelines.

First PEP Dose Within 72 Hours

A designated person/trained doctor must assess the risk of HIV and HBV and HCV transmission following an AEB. This evaluation must be quick so as to start treatment without any delay, ideally within two hours but certainly within 72 hours; PEP is not effective when given more than 72 hours after exposure. The first dose of PEP should be administered within the first 72 hours of exposure. If the risk is insignificant, PEP could be discontinued, if already commenced.

Assessing Risk of Transmission

Exposure is defined under three categories based on the amount of blood/fluid involved and the entry port (Table 23.3). These categories are intended to help in assessing the severity of the exposure but may not cover all possibilities.

Table 23.3: Categories of exposure

Category	*Definition and example*
Mild exposure	mucous membrane/non-intact skin with small volumes, e.g. a superficial wound (erosion of the epidermis) with a plain or low calibre needle, or contact with the eyes or mucous membranes, subcutaneous injections following small-bore needles.
Moderate exposure	mucous membrane/non-intact skin with large volumes or percutaneous superficial exposure with solid needle, e.g. a cut or needle stick injury penetrating gloves.
Severe exposure	percutaneous with large volume, e.g. an accident with a high calibre needle (>18 G) visibly contaminated with blood; a deep wound (hemorrhagic wound and/or very painful); transmission of a significant volume of blood; an accident with material that has previously been used intravenously or intra-arterially.

The wearing of gloves during any of these accidents constitutes a protective factor.

Note: In case of an AEB with material such as discarded sharps/needles, contaminated for over 48 hours, the risk of infection is negligible for HIV, but still remains significant for HBV. HBV survives longer than HIV outside the body.

Assess Exposed Individual

The exposed individual should have confidential counseling and assessment by an experienced physician. Exposed individuals who are known or discovered to be HIV positive should not receive PEP. They should be offered counseling and information on prevention of transmission and referred to clinical and laboratory assessment to determine eligibility for Antiretroviral therapy (ART). Besides the medical assessment, counseling exposed HCP is essential to allay fear and start PEP.

Step 3: Counseling for PEP

Exposed persons (clients) should receive appropriate information about what PEP is about and the risk and benefits of PEP in order to provide informed consent for taking PEP. It should be clear that PEP is not mandatory.

Psychological Support

Many people feel anxious after exposure. Every exposed person needs to be informed about the risks, and the measures that can be taken. This will help to relieve part of the anxiety. Some clients may require further specialised psychological support.

Document Exposure

Documentation of exposure is essential. Special leave from work should be considered initially for a period of two weeks. Subsequently, it can be extended based on the assessment of the exposed person's mental state, side effects and requirements.

Practical Application in the Clinical Settings

- For prophylactic treatment the exposed person must sign consent form.
- Informed consent also means that if the exposed person has been advised PEP, but refuses to start it, this needs to be recorded. This document should be kept by the designated officer for PEP.
- An information sheet covering the PEP and the biological follow-up after any AEB must be given to the person under treatment. However, this sheet cannot replace verbal explanations.

Step 4: Prescribe PEP

Deciding on PEP Regimen

There are two types of regimens:

- Basic regimen: 2-drug combination
- Expanded regimen: 3-drug combination.

The decision to initiate the type of regimen depends on the type of exposure and HIV serostatus of the source person (Table 23.4).

Table 23.4: HIV PEP Evaluation

Exposure	*Status of Source*		
	HIV+ and asymptomatic	*HIV+ and clinically symptomatic*	*HIV status unknown*
Mild	Consider 2-drug PEP	Start 2-drug PEP	Usually no PEP or consider 2-drug PEP
Moderate	Start 2-drug PEP	Start 3-drug PEP	Usually no PEP or consider 2-drug PEP
Severe	Start 3-drug PEP	Start 3-drug PEP	Usually no PEP or consider 2-drug PEP

- In the case of a high-risk exposure from a source patient who has been exposed to or is taking antiretroviral medications, consult an expert to choose the PEP regimen, as the risk of drug resistance is high. Refer/consult expert physician. Start 2-drug regimen first.

Seek expert opinion in case of:

- Delay in reporting exposure (> 72 hours)
- Unknown source
- Known or suspected pregnancy, but initiate PEP
- Breastfeeding mothers, but initiate PEP
- Source patient is on Antiretroviral therapy (ART)
- Major toxicity of PEP regimen.

Step 5: HIV Chemoprophylaxis

Because post-exposure prophylaxis (PEP) has its greatest effect if begun within two hours of exposure, it is essential to act immediately. The prophylaxis needs to be continued for four weeks. Exposure must be immediately reported to designated authority and therapy administered. Never delay start of therapy due to debate over regimen. Begin with basic 2-drug regimen, and once expert advice is obtained, change as required (Table 23.5).

Table 23.5: Dosage of drugs for PEP

Medication	*2-drug regimen*	*3-drug regimen*
Zidovudine (AZT)	300 mg twice a day	300 mg twice a day
Stavudine (d4T)	30 mg twice a day	30 mg twice a day
Lamivudine (3TC)	150 mg twice a day	150 mg twice a day
Protease Inhibitors		**1st choice** Lopinavir/ritonavir (LPV/r) 400/100 mg twice a day or 800/200 mg once daily with meals **2nd choice** Nelfinavir (NLF)

Contd...

Contd...

	1250 mg twice a day or 750 mg three times a day with empty stomach **3rd choice** Indinavir (IND) 800 mg every 8 hours and drink 8-10 glasses (1.5 liters) of water daily

Note: If protease inhibitor is not available and the 3rd drug is indicated, one can consider using Efavirenz (EFV 600 mg once daily).

Monitoring should be instituted for side effects of this drug, e.g. CNS toxicity such as nightmares, insomnia, etc.

- Fixed Dose Combination (FDC) are preferred, if available. Ritonavir requires refrigeration.

Table 23.6: PEP regimens to be prescribed by Health Centres

	Preferred	***Alternative***
2-drug regimen (basic PEP regimen)	Zidovudine (AZT) + Lamivudine (3TC)	Stavudine (d4T) + Lamivudine (3TC)
3-drug regimen (consult expert opinion for starting 3 drug, e.g. LPV/r, NLF or IND regimen)		
Not recommended	ddI + d4T combination NNRTI such as Nevirapine should not be used in PEP	

More information on alternative schedules is available in the latest update USPHS guidelines issued 30 September 2005. (http://www. cdc.gov/mmwr/preview/mmwrhtml/rr5409a1.htm)

Selection of PEP Regimen When the Source of Patient is on Antiretroviral Therapy (ART)

The physician should consider the comparative risk represented by the exposure taking in view exposure source's history of and response to antiretroviral therapy based on clinical response, CD4 cell counts, viral load measurements (if available), and current disease stage (WHO clinical staging and history). If the source person's virus is known or suspected to be resistant to one or more drugs considered for the PEP regimen, exposed person needs to be given alternate PEP drug regimen, and referred for expert opinion (Table 23.6).

Changes in the PEP regimen can be made after PEP has been started. Re-evaluation of the exposed person should be considered within 72 hours post-exposure, especially as additional information about the exposure or source person becomes available.

Antiretroviral (ARV) Drugs during Pregnancy

Data regarding the potential effects of antiretroviral drugs on the developing fetus or neonate are limited. There is a clear contraindication for Efavirenz (first 3 months of pregnancy) and Indinavir (prenatal).

For a female Health Care Professional (HCP) considering PEP, a pregnancy test is recommended in case of a doubt. Pregnant HCP are recommended to begin the basic 2-drug regimen, and if a third drug is needed, Nelfinavir is the drug of choice.

Side Effects and adherence to PEP

Studies have indicated more side effects, most commonly nausea and fatigue, among HCP taking PEP than PLHAs taking ART. These side effects occur mainly at the beginning of the treatment and include nausea, diarrhea, muscular pain and headache. The person taking the treatment should be informed that these may occur and should be dissuaded from stopping the treatment as most side-effects are mild and transient, though possibly uncomfortable. Anemia and/or leucopenia and/or thrombocytopenia may occur during the month of treatment.

Adherence information and psychological support are essential. More than 95% adherence is important in order to maximise the efficacy of the medication in PEP. Side effects can be reduced through medications. A complete blood count and liver function tests (transaminases) may be performed at the beginning of treatment (as baseline) and after 4 weeks.

Step 6: Follow-up of an Exposed Person

Whether or not post-exposure prophylaxis is started, a follow-up is needed to monitor for possible infections and to provide psychological support.

Clinical Follow-up

In the weeks following an AEB, the exposed person must be monitored for the eventual appearance of signs indicating an HIV seroconversion: Acute fever, generalized lymphadenopathy, cutaneous eruption, pharyngitis, non-specific flu symptoms and ulcers of the mouth or genital area. These symptoms appear in 50-70% of individuals with an HIV primary (acute) infection and almost always within 3 to 6 weeks after exposure. When a primary (acute) infection is suspected, referral to an ART center or for expert opinion should be arranged rapidly.

An exposed person should be advised to use precautions (e.g., avoid blood or tissue donations, breastfeeding, unprotected sexual relations or pregnancy) to prevent secondary transmission, especially during the first 6-12 weeks following exposure. Condom use is essential.

Drug adherence and side effect counseling should be provided and reinforced at every follow-up visit. Psychological support and mental health counseling is often required.

Laboratory follow-up

Exposed persons should have post-PEP HIV tests. HIV-test at 3 months and again at 6 months is recommended. If the test at 6 months is negative, no further testing is recommended.

BIBLIOGRAPHY

1. Antiretroviral Therapy Guidelines for HIV-infected Adults and Adolescents including Post-exposure Prophylaxis, May 2007, NACO, Ministry of Health and Family Welfare, Government of India.

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