 . 2021 Jan 27;71(2):8795. doi: 10.1111/idj.12615Over the past century, there is perhaps no greater contribution to the practice of clinical dentistry than the development and application of local anaesthesia. What were once considered painful procedures have now been made routine by the deposition and action of local anaesthetics. This article will serve as a review of basic pharmacological principles of local anaesthesia, subsequent sequelae that can arise from their use, considerations when using local anaesthetics, and recent advances in the delivery of local anaesthesia.Key words: Local anaesthetics, Pharmacology, Adverse reaction, Drug interaction, Mechanism of actionAn average dentist will administer over 1,500 cartridges of dental local anaesthetic a year.1 As such, anyone administering this drug should be intimately familiar with what the drug does to the body, as well as what the body does to that drug. This article will serve as a review of the pharmacokinetics and pharmacodynamics of local anaesthetics, possible consequences and adverse events from their use, and emerging technologies pertaining to the use of local anaesthetics.Modern local anaesthetics are typically differentiated based on their chemical structure, specifically the linkage (an amide versus an ester linkage) between common elements of the compound. The majority of commonly used dental local anaesthetics fall into the amide category (lidocaine, mepivacaine, bupivacaine, prilocaine), though there are some ester-type local anaesthetics that also contain an additional ester linkage (articaine). While both types of local anaesthetics have the same mechanism of action, they differ slightly in their metabolism as described below. It is rare in dentistry that ester-type anaesthetics are used for local anaesthesia purposes, though these types of anaesthetics are used more commonly for topicalisation prior to injection to reduce discomfort associated with mucosal needle puncture.Local anaesthetics all act in the same manner they bind to cellular sodium channels and inhibit the influx of sodium into the cell which prevents cell depolarisation and subsequent transmission of the previously propagating action potential.2 This is beneficial in that the action potential of a painful stimulus, such as drilling into the dentin of a tooth, can be stopped from reaching the higher processing centres of the brain and otherwise painful procedures can be completed with relative patient comfort.The onset of local anaesthesia is contingent on two factors: the lipid solubility and the pKa of the local anaesthetic. The more lipid-soluble a local anaesthetic is, the greater its potency.3 For the local anaesthetic base to be stable in solution, it is formulated as a hydrochloride salt.3 At that time, the molecules exist in a water-soluble state and thus are unable to penetrate the neuron.3 Therefore the time for onset of local anaesthesia is directly related to the proportion of molecules that convert to the lipid-soluble structure when exposed to physiologic pH (7.4). This proportion is determined by the ionisation constant (pKa) for the anaesthetic and is calculated using the Henderson-Hasselbalch equation.3 This implies that the higher the pKa for a local anaesthetic, the fewer molecules are available in their lipid-soluble form and thus the further is the delay in the onset of action.2,3 This is why it is harder to anaesthetise a patient with an infection, as the environment pH is much lower (around pH of 5.2) and this favours the water-soluble state.For instance, bupivacaine is the most lipid-soluble local anaesthetic so a lower percent of drug dissolved in solution is required to cause nerve blockade compared to a less lipid-soluble local anaesthetic like mepivacaine. Thus, for a rapid onset of action, the lower the pKa of a local anaesthetic, the more ionised drug is present in normal tissue and the faster the onset of blockade.The duration of action of a local anaesthetic is contingent on two factors: the protein binding and redistribution of the local anaesthetic. Protein binding of the local anaesthetic is an inherent drug characteristic the more protein-bound a drug is, the longer the duration of action.4 Duration of action on dental pulp and soft tissues is contingent almost completely on diffusion away from the site of action of the local anaesthetic. If an area is more vascular, the faster the drug will be absorbed into systemic circulation and away from the target tissue.Amide-type local anaesthetics are biotransformed into water-soluble metabolites in the liver by hepatic microsomal enzymes and subsequently excreted by the kidney.5 Articaine is primarily metabolised via its ester linkage by plasma esterases in the blood.6,7Knowing that the duration of action of a drug is determined by its protein binding and redistribution, cartridges of local anaesthetic can be modified to have additional components present in the cartridge in order to increase the duration of action. Specifically, a vasoconstrictor such as epinephrine or levonordefrin can be added so that when the solution is deposited at the site of action, the surrounding vascular beds will vasoconstrict, the drug will be more slowly absorbed into the circulating bloodstream, and the duration of action of the local anaesthetic will be increased.8 Local anaesthetics with vasoconstrictors should be used carefully for patients with pre-existing hypertension or cardiac irritability as their presence in the body may further increase blood pressure or cause cardiac dysrhythmias.In addition to the local anaesthetic, the distilled water in which it is dissolved, and potentially a vasoconstrictor, there is the possibility of an oxidant included in the cartridge. Local anaesthetics are generally stable and are resistant to degradation, but vasoconstrictors are much more susceptible to degradation from the presence of molecular oxygen, light, elevated temperature, heavy metals, and an increase in pH.9 As such, antioxidants such as metabisulfite are added to the local anaesthetic cartridge to reduce the rate of deterioration of the vasoconstrictor.Previously, methylparaben (an anti-fungal) was included in local anaesthetic cartridges as it was originally included in the formulation of multi-dose vials of local anaesthetics used for medical purposes. When single-use cartridges were introduced in the dental setting, methylparaben was included until it was appreciated that it was no longer necessary to ensure an additional infection control measure, and it was then phased out of dental cartridges in North America with the last amount of methylparaben included in 1984.10 It should be noted that there are still countries where formulations of local anaesthetic exist with the addition of methylparaben.11While local anaesthetics have the ability to produce anaesthesia of intended nerves and anatomic areas, they are not exempt from Paracelsus law, Only the dose makes the poison. That is, they too can be toxic in higher doses. When systemic concentrations of circulating local anaesthetic become high enough, there can be unintended and severe consequences which are neurologic and cardiac in nature. Inhibitory neurons in the nervous system are generally those first affected, which when blocked will produce excitatory symptoms such as visual and auditory disturbances, seizures, and muscle twitching.12 As plasma concentrations continue to rise, depressive clinical manifestations begin to appear such as decreased level of consciousness possibly leading to coma and respiratory arrest. Following increased plasma concentration leading to adverse neurological events, cardiac conditions can arise from heightened concentration of the drug. Local anaesthetics will again act to block sodium channels, but this time in areas of the heart required for propagation of cardiac conduction. A variety of sequelae can manifest from tachyarrhythmias to bradyarrhythmias, up to the point that plasma levels of the drug will inhibit cardiac function altogether and cause an arrest.13The best means of avoiding local anaesthetic systemic toxicity is awareness of the patient's weight, the maximum per kilogram (or absolute) dose of the local anaesthetic (see Table 1), and careful calculation so as to avoid systemic concentrations of the drug that could disrupt regular cell membrane function. Included in the Table is a list of the most commonly used local anaesthetics in dentistry as well as their associated maximums that can be administered to patients on a per kilogram basis. In order to determine the maximum dose for a patient, one must simply multiply the patient's weight by the per kilogram maximum specific to the local anaesthetic being used by the dentist.Recommended Canadian maximum doses of local anaesthetics1,13DrugMaximumArticaine WITH vasoconstrictor7 mg/kg (up to 500 mg)5 mg/kg in childrenBupivacaine WITH vasoconstrictor2 mg/kg (up to 200 mg)Lidocaine WITH vasoconstrictor7 mg/kg (up to 500 mg)Mepivacaine WITH vasoconstrictor6.6 mg/kg (up to 400 mg)Prilocaine WITH vasoconstrictor8 mg/kg (up to 500 mg)Mepivacaine WITHOUT vasoconstrictor6.6 mg/kg (up to 400 mg)Prilocaine WITHOUT vasoconstrictor8 mg/kg (up to 500 mg) In order to determine how much local anaesthetic is in a cartridge (see the example in Table 2), the percent solution of the local anaesthetic expressed in mg/mL must be multiplied by the amount of solution in the cartridge. Of note, percentage of local anaesthetic is the number of grams per 100 mL, or mg per mL. For example, a 2% solution represents 20 mg/mL, and a 4% solution represents 40 mg/mL. A typical dental local anaesthetic cartridge in North America contains approximately 1.8 mL, while many countries use 2.2 mL cartridges.Example calculation of amount of local anaesthetic in a dental anaesthetic cartridgeInformationCalculation and amount2% solution has a concentration of 20 mg/mL2% solution = 20 mg/mLThe cartridge has 1.8 mL of solution20 mg/mL×1.8 mL=36 mg4% solution has a concentration of 40 mg/mL4% solution = 40 mg/mLThe cartridge has 1.8 mL of solution40 mg/mL×1.8 mL=72 mgAgain, the amount of local anaesthetic being deposited should be less than the per kilogram maximum specific to the local anaesthetic being used and less than the absolute maximum associated with that local anaesthetic (Table 1).Despite the best efforts of clinicians, drug errors occur when the patient receives too large a dose of local anaesthetic, or an intravascular administrationof local anaesthetic occurs, and the patient demonstrates an abnormal reaction of systemic toxicity despite no pre-existing medical condition,14 or perhaps the patient had an unknown medical condition that predisposed them to local anaesthetic systemic toxicity.15 As such, astute clinicians should be ready to recognise and treat the symptoms of local anaesthetic systemic toxicity (tinnitus, metallic taste, circumoral numbness, altered mental status, slurred speech, hypotension, bradycardia, seizures, ventricular arrhythmias, and cardiac arrest). The management of local anaesthetic systemic toxicity includes (but is not limited to):Activating emergency medical services when in an ambulatory location in order to be able to transport and monitor the patient in a tertiary care facilityEnsuring adequate oxygenation (may include administering supplemental oxygen and/or manual ventilation of the patient)Provided that intravenous access is established and the provider has been trained to administer intravenous rescue medications:a.Administering of intravenous Intralipid 20% (1.5 mL/kg for a child or 100 mL bolus for an adult over 65 kg and a subsequent infusion of 0.25 mL/kg/min or more if hypotension persists) to treat the cardiac aspects of local anaesthetic systemic toxicityb.Treating seizures, if present (titration of intravenous midazolam beginning with 100 mcg/kg for a child or 5 mg for an adult)c.Treating bradycardia and/or hypotension with an intravenous vasopressord.Monitoring for ventricular fibrillation or ventricular tachycardias and treating as appropriate.Anticipating acidosis, hypercarbia, hyperkalemia, and treating as necessary.True documented allergy to amide-type local anaesthetics is exceedingly rare. It is estimated to have a possible incidence of allergy from 0.1% to 1%,17 recent data suggest a possible increase in the incidence of this allergy (specifically to lidocaine).18 When an allergy to local anaesthetic is suspected, there exist several complex means of investigating whether the symptoms present are a true anaphylactic reaction. The patient should be referred to an allergist or immunologist in order to determine if indeed an allergy exists and, if so, to determine the allergenic components of the local anaesthetic cartridge. It is now understood that an allergist-administered intradermal administration test, followed by subsequent provocation challenges (if necessary), can demonstrate adequate predictive value in order to rule out possible local anaesthetic allergy.18While some patients may note allergy-like symptoms from a dental injection, it is likely that these symptoms appear from either a psychogenic reaction, which can mimic allergy and even anaphylaxis, or it could be that the patient does have an allergy to one of the contents in the local anaesthesia cartridge other than the local anaesthesia itself. Historically, local anaesthetic multi-dose vials were re-used between patients and so an agent, such as methylparaben a bacteriostatic, anti-fungal agent was also present in these vials. It was not uncommon for patients to experience allergic reactions from these types of agents. Today's commonly used dental local anaesthetics with vasoconstrictors contain the preservative sodium metabisulfite, which some case reports have suggested as a possible allergen.18,19 It should be noted that ester-type local anaesthetics, such as benzocaine which is used in many formulations of topical anaesthetic, is one of the more allergenic agents found in a dental office after latex, non-steroidal anti-inflammatory drugs, and penicillin-type antibiotics. Dental practitioners should pay particular attention for the signs and symptoms of allergy or anaphylaxis after the application of any ester-type topical anaesthetic.19 Benzocaine applied as a topical anaesthetic may cause aphthous ulcers in some patients (a possible sign of sensitivity or even allergy); should this occur, topical lidocaine would be more appropriate for future appointments.With the above in mind, while noting that allergy to a local anaesthetic or a component in the cartridge is exceedingly rare, such an event is not altogether impossible and, as such, the prudent practitioner should therefore be prepared to recognise and treat the signs and symptoms of an allergic reaction. A conscious patient experiencing an anaphylactic reaction would generally have a constellation of symptoms involving the dermatologic (rashes, hives, urticaria, erythema, mottling), respiratory (wheezing, dyspnea from airway angioedema), and gastrointestinal systems (cramping, vomiting, diarrhoea). A patient who is in a state of sedation may have some of these clinical features masked and may instead present with cardiovascular collapse (significant hypotension or asystole) or respiratory compromise (hypoxia, dyspnea, tachypnea, coughing/bucking, bronchospasm).20 The management of anaphylaxis includes (but is not limited to):Activating emergency medical services when in an ambulatory location in order to be able to transport and monitor the patient in a tertiary care facilityDiscontinuing or removing the anaphylaxis-inciting agentEnsuring airway patency (considering intubation) and administering 100% oxygenAdministering epinephrine (0.3 mg for > 30 kg body weight; 0.15 mg for up to 30 kg, IM in lateral thigh as an initial dose of epinephrine).These additional steps may be carried out provided the medications are available and the provider has been trained in their administration:Administering an H1 and H2 blocker (respectively: diphenhydramine 1 mg/kg IM to a maximum of 50 mg and ranitidine 1 mg/kg IM to a maximum of 50 mg)Administering a corticosteroid like hydrocortisone (2 mg/kg IM to a maximum of 100 mg)Provided that intravenous access is established and the provider has been trained to administer intravenous rescue medications:a.Administering a bolus push of intravenous fluid (20 mL/kg to a maximum of 1 L, or more if necessary).Pre-syncope and syncope (vasovagal reactions) are among the most common medical emergencies that occur in dental offices. Patients anxieties about the deposition of local anaesthesia, or any other dental procedure for that matter, may manifest clinically with variations in heart rate and blood pressure, pallor, nausea, vomiting, and dyspnea.21 Care should always be taken to probe a patient's level of anxiety in the dental office so as to be able to implement pharmacologic or non pharmacologic means that will make patients comfortable for the duration of care in the office setting.Another common issue for patients after having received local anaesthesia for a dental procedure is the associated soft tissue anaesthesia that persists and subsequent trauma to those anaesthetised tissues from lack of sensory feedback.22 Care must be taken to warn patients about this time of vulnerability to those tissues at risk. Additionally, cotton rolls or gauze can be used as a shield to prevent patients from gnawing chewing on these structures. If possible, the use of a local anaesthetic without epinephrine will reduce the amount of time that this soft tissue anaesthesia persists. Additionally, a practitioner may decide that the use of phentolamine mesylate would be appropriate, to be administered in order to cause local vasodilation in the area where a vasoconstrictor was previous administered, thus accelerating the rate of redistribution of the local anaesthetic, accelerating the offset of the drug, and subsequently accelerating the reversal of collateral soft tissue anaesthesia. This medication is further discussed later in the paper.Trismus, a reduction in the range of mandibular motion, can occur after a dental injection. It is often caused by the needle passing through a muscle of mastication which in turn causes spasticity to the muscle.23 It can also be caused by the accumulation of a haematoma (see below) impeding excursive movements that permit full opening. Analgesics and a soft diet are mainstay therapies in the acute phase of trismus following a dental injection, with a gradual return to function and physiotherapy if necessary.In order to administer local anaesthesia, a loaded syringe with an attached needle is inserted into the desired location of deposition. New needles have an associated bevel that are sharp in order to comfortably puncture oral mucosa, but that same quality reducing initial discomfort of mucosal puncture and travel through connective tissue can lead to puncture of surrounding vascular structures. When a dentist has reached their intended endpoint where local anaesthesia is to be deposited, it is recommended that, at minimum, one aspiration manoeuvre is performed. This act (via either pressing an aspiration ring or withdrawing from a cartridge that has a harpoon inserted into the cartridge's rubber stopper) introduces negative pressure into the cartridge and serves the purpose of aspirating whatever matter is at the tip of the needle. If a needle tip is located inside a vascular structure, the negative pressure into the cartridge will draw visible blood into the cartridge and alert the dentist of the needle's tip inside a blood vessel.Unfortunately, there are times when an aspiration manoeuvre is carried out while the needle tip is indeed in a blood vessel but there is a false negative aspiration (that is, no blood is visualised in the cartridge). This false negative event could be due to either the bevel of the needle being positioned in such a way that the action of aspiration draws the wall of the vascular structure to the bevel instead of blood, or because the gauge of needle was too small. In either case, if the contents of the cartridge are deposited directly into the bloodstream, patients can experience immediate symptoms from both the local anaesthetic and vasoconstrictor.24 While these symptoms are often related to the blood flow from vessels adjacent to the block being performed, they may present as palpitations, headaches, visual disturbances, and vertigo. While patient reassurance that these symptoms will resolve is first-line management of an intravascular injection, the dental practitioner would be prudent to continue to monitor level of consciousness and vital signs until the local anaesthetic has redistributed away from the site of injection and the patient has returned to their baseline state. Anecdotally, just one cartridge of lidocaine can and in cases has produced seizures, illustrating how the speed attending intravascular injection may be the decisive factor in the severity of local anaesthetic systemic toxicity.As the needle is passing through connective tissue, it is not uncommon for the tip to puncture a vascular structure. Occasionally, blood will extravasate from this breach of endothelium into the surrounding extravascular area and accumulate locally. This can be associated with facial swelling, soreness, and trismus from the expansion of the potential space where the bleeding from the insult occurred.25 If a haematoma is suspected, a dentist should immediately apply pressure to the area in order to aid in haemostasis of the punctured vessel and to attempt to reduce the amount of extravasation in the area. The patient should only be dismissed once the dentist is confident that the bleeding has ceased.There have been case reports published for over 50 years about various ocular complications arising from the administration of inferior alveolar blocks. There may be one or more symptoms including, but not limited to, amaurosis, diplopia, total ophthalmoplegia, mydriasis, ptosis, and blanching of the periorbital skin.26 The common belief of the cause of this phenomenon is the variable anatomy of the maxillary artery in which there is a subsequent intravascular injection which could carry the local anaesthetic in a retrograde manner from the middle meningeal artery through the foramen spinosum and back to the lacrimal and optic arteries resulting in anaesthetisation of cranial nerves 3, 4 and 6. Generally supportive measures and tincture of time suffice to resolve this complication, but a differential diagnosis should be formed and referral to the appropriate sub-specialist if necessary.There are extremely rare cases in the literature and closed-claim analysis where patients who had completed nerve blocks subsequently experienced permanent paraesthesias of associated nerves when there was no surgical procedure involved.27 Given the rarity of such events, the study of non-surgical paraesthesias is very difficult and almost exclusively retrospective in nature. With that being said, speculation exists that a combination of trauma to the nerve from the needle and higher percentages of local anaesthetics like articaine and prilocaine are the most likely causes of such paraesthesias from direct neurotoxicity to nerve trunks.28 Additionally, the number of respective nerve fascicles and the ratio of nerve fascicles affected may correlate to the severity of paraesthesia (e.g. higher incidence of lingual nerve paraesthesia and a lower number of nerve fascicles being affected for the lingual nerve).29 However, this topic does remain controversial as there has been other literature published noting that the hypothesis may not be the case.30, 31, 32 In any and all cases of deposition of local anaesthetic, the dental practitioner must weigh the pros and cons of choice of drug and route of administration prior to the injection.Exposure to some local anaesthetics (mainly the ester-type local anaesthetics and principally prilocaine) can precipitate a change in the iron atom in the haemoglobin, specifically from a ferrous state to a ferric state to create a molecule called methaemoglobin. This is of concern as the ferric haemoglobin molecule has a much greater affinity for oxygen, so much so that the oxygen will not dissociate from the haemoglobin and therefore not be available for tissue and organ usage. If an exposure causes enough haemoglobin to become methaemoglobin, the patient may experience signs and symptoms of hypoxemia such as cyanosis and shortness of breath. If this condition is suspected, supplemental oxygen should be administered (despite an ineffective oxygen-carrying capacity from the methaemoglobinemia) and emergency medical services contacted. Definitive treatment for this emergency is the intravenous administration of methylene blue.Table 3 provides example calculations for maximum dose for local anaesthesia for various patients.Example calculations of maximum dose of local anaesthetic for various patientsInformationCalculation and maximum20 kg patient (e.g. a 5-year-old)2% lidocaine with 1:100,000 epinephrine in a 1.8 mL cartridge20 kg7 mg/kg maximum=140 mg maximum140 mg maximum/36 mg per cartridge=3.8 cartridges of 2% lidocaine with 1:100,000 epinephrine20 kg patient (e.g. a 5-year-old)4% articaine with 1:100,000 epinephrine in a 1.8 mL cartridge20 kg7 mg/kg maximum=140 mg maximum140 mg maximum/72 mg per cartridge=1.9 cartridges of 4% articaine with 1:100,000 epinephrine60 kg patient (e.g. a 40-year-old)2% lidocaine with 1:100,000 epinephrine in a 1.8 mL cartridge60 kg7 mg/kg maximum=420 mg maximum420 mg maximum/36 mg per cartridge=11.6 cartridges of 2% lidocaine with 1:100,000 epinephrine100 kg7 mg/kg maximum=700 mg maximum2% lidocaine with 1:100,000 epinephrine in a 1.8 mL cartridgeBUT reported maximum is 500 mg500 mg maximum/36 mg per cartridge=13.8 cartridges of 2% lidocaine with 1:100,000 epinephrineUnless the patient is on a concomitant local anaesthesia infusion for an ailment (a potential risk for local anaesthetic systemic toxicity), there are no significant drug interactions with non-epinephrine-containing local anaesthetics. Drug interactions stemming from the contents of a local anaesthetic cartridge are almost exclusively from the included vasoconstrictor. The most notable interactions are noted in Table 4 below. As such, epinephrine should be use with great caution when concomitant use of one of the drugs is present, and levonordefrin should be avoided altogether when the patient is taking a tricyclic antidepressant.Drugs known to cause interactions with vasoconstrictors and potential associated effectsDrugEffectBeta blockers (drugs that end in -olol)Ex. metoprolol, propranolol, labetalol, bisoprolol, atenololBeta-blockers block beta-adrenergic receptors and can produce unopposed alpha-adrenergic receptor agonism with corresponding hypertension when epinephrine is present.Volatile anaesthetics (drugs that end in -ane)Ex. halothane, sevoflurane, isoflurane, desfluraneVolatile anaesthetics sensitise the myocardium to catecholamines cardiac arrhythmias can be induced with the injection of exogenous epinephrine.Amphetamines (names vary)Ex. cocaine, methamphetamineAmphetamines increase blood pressure and can cause cardiac arrhythmias by themselves with the potential for adverse event synergism from epinephrine.Tricyclic antidepressants (names vary)Ex. amitriptyline, imipramine, trimipramine, nortriptyline, protriptyline, desipramineTricyclic antidepressants increase the systemic circulation of catecholamines and can lead to systemic hypertension when supplementary epinephrine is present.While supraperiosteal injections are generally sufficient to achieve pulpal anaesthesia for maxillary dentition, these types of injections are significantly less successful for mandibular teeth due to the thickness of the bone cortex. As such, deposition of local anaesthetic adjacent to the inferior alveolar nerve (IAN) must be carried out via one of several approaches, all of which have varying rates of success,33,34 and none of which are able to accomplish nerve blockage 100% of the time.35 This can be attributed to various hard and soft tissue characteristics creating uncertainty about the position of the needle tip relative to the IAN, fascial linings acting as a potential barrier to the diffusion of local anaesthetic solution, and the sphenomandibular ligament possibly impeding diffusion of local anaesthetic to the IAN (likely because the needle tip was too medial to the mandibular foramen).36 Accessory innervation from the mylohyoid nerve, the long buccal nerve, the greater auricular nerve, and even a bifid IAN can also carry additional sensory fibres to mandibular dentition.31 Given the above information, all injections should be performed with prior examination of the patients anatomical features to permit minor alterations to technique as necessary in order to maximise chances of success of a nerve blockade.As carious lesions increase in size and proximity to pulpal tissue, various biologic markers are produced and subsequent inflammatory mediators are recruited to the site.37 This inflammation creates a localised area of inflammatory acidosis where the lowered pH value inhibits the action of local anaesthetics due to the altered interaction with components of the liposomes.38 Additionally, various isoforms of tetrodo-toxin-resistant sodium channels (i.e. sodium channels on which lidocaine has a reduced antinociceptive effect)39 are recruited in the inflammatory state.40 This combination of factors can make reliable and profound anaesthesia very difficult to achieve, and practitioners should be prepared to administer adjunctive techniques such as intra-osseous or periodontal ligament injections in order to provide a comfortable experience for their patients.41At this time, only lidocaine and prilocaine have an FDA foetal risk classification indicating no risk of teratogenic effects based on the results of human and animal studies. Other commonly used local anaesthetics (bupivacaine, articaine, mepivacaine) have an FDA foetal risk classification indicating that teratogenic risk cannot be eliminated on the basis of human and animal studies.42 The first trimester of pregnancy poses the highest threat for teratogenicity and so only emergent dental work should be completed during this trimester. It is currently believed that the second trimester poses the lowest risk of foetal harm and local anaesthesia use should in theory be safe.43 While it is possible to complete elective dental treatment during the third trimester of pregnancy, there is a higher risk of aortocaval compression and increased conduction blockade. If local anaesthesia is to be administered in the third trimester, lower doses should be used.Current demographic data show that the North American population is aging, and projections suggest that the percentage of older people will continue to increase.44 In those of advanced age, the pharmacokinetics and pharmacodynamics of many drugs are altered.44 No significant differences in the response of the elderly to local anaesthetics are expected. However, as aging is accompanied by decreased liver and kidney function, doses below the maximum are recommended.44Also, geriatric patients commonly have cardiovascular disease and, thus, the dose of epinephrine contained in anaesthetics should be limited to a maximum of 0.04 mg.44 Even without a history of overt cardiovascular disease, it is prudent to minimise the use of epinephrine in elderly patients simply because of the expected effect of aging on the heart. Monitoring blood pressure and heart rate is advised when considering multiple administrations of epinephrine-containing local anaesthetic.Children are at higher risk for soft tissue injury due to a relative lack of awareness after local anaesthetic administration. Children are at a higher risk for local anaesthetic systemic toxicity because they weigh significantly less than an adult patient so their absolute threshold for local anaesthesia deposition is much lower than that of adults.45 Practitioners should also be wary of patient and personal safety when delivering local anaesthetic to a pre-cooperative or anxious child as needle-stick injury may be more likely given a mobile target for local anaesthesia deposition.It is currently understood that patients within therapeutic international normalised ratio (INR) ranges can receive local anaesthetic nerve blocks without cessation of the anticoagulant beforehand. Even if a haematoma does occur, local haemostatic measures are generally sufficient to produce haemostasis.There are now several electronic devices on the market that aid in the delivery of local anaesthesia, specifically with digital controls that can be manipulated to aid in aspiration and continuous delivery of local anaesthetic solution.46 Many microprocessor-aided local anaesthesia devices will monitor the counterpressure exerted by the tissues into which the local anaesthetic is being injected and vary the rate of deposition of injectate accordingly.47 In addition to assuming a less threatening appearance than a traditional syringe and needle armamentarium, these computer-controlled devices will ensure both appropriate aspiration and duration of delivery of the local anaesthesia which may reduce injection pain.Most local anaesthetic cartridges deposited worldwide contain epinephrine, so for patients at higher risk of traumatic injury to soft tissues or simply patients who wish to have their blockade reversed more quickly, phentolamine mesylate is a vasodilator that when deposited in a similar location to the original epinephrine-containing local anaesthetic solution can overwhelm the previous vasoconstriction and aid in the redistribution (and the clinical offset) of the local anaesthetic.48 Recent studies suggest that it may have particular use in children in providing a more rapid recovery of lip sensation which may decrease the incidence of soft tissue trauma associated with local anaesthetic delivery in this age group.49It is believed that an increased pH of a solution being deposited could decrease the amount of discomfort associated with injections and increase the speed of onset of nerve blockade. Current studies show that the above claims above are increasingly likely to be true and dental manufacturing companies are creating devices that alkanise, that is increase the pH of, local anaesthetic solutions prior to dental injection.50 It should be noted that a recent meta-analysis shows that buffered local anaesthetics have 2.23 times greater likelihood of achieving profound anaesthesia in pulpally involved teeth.51A combination of local anaesthetics (tetracaine) and nasal decongestants (oxymetazoline) is being used to anaesthetise maxillary anterior teeth. This combination of drugs may demonstrate less successful pulpal anaesthesia and more adverse events compared to traditionally deposited local anaesthetics.52In an attempt to increase the duration of local anaesthetics, a formulation of bupivacaine has been produced where the local anaesthetic molecule is loaded in multivesicular liposomes. This slow-release formulation of drug is able to delay the release of local anaesthetic and therefore extend the duration of pain relief for the patient for up to 72 hours, compared to unaltered bupivacaine traditionally providing up to 8 hours of analgesia. It has been demonstrated to be suitable for local infiltration leading to increased duration of action and subsequent sparing of other analgesic medications (such as opioids).53 The safety profile is currently being established and appears not to differ from that of bupivacaine with no additional incidence of adverse events being noted. Some trials have noted no difference in reducing the duration of analgesia of necrotic teeth from that of traditional bupivacaine.54 That being said, additional trials with significant power are needed before its use can be recommended.Following surgical procedures, clinicians must determine the most appropriate means of controlling any post-operative pain associated with the procedure. This can be accomplished by a variety of localised or systemic means (some of which have been noted previously), one of which is an emerging method of patient-controlled localised deposition of local anaesthetic at the site of injury or surgery. There are many examples of a patient-controlled local anaesthetic infusion pump such as the ON-Q pain pump being used for general medical surgery,55, 56, 57 but there is still much research to be carried out about these infiltrating catheters and their potential benefit in treating the head and neck region,58 and possible intraoral applications.In order to negate mandibular anatomical differences in varied patient populations, the use of ultrasonography to visualise and direct the blockage of the IAN may prove worthwhile. Previous studies have either used Doppler ultrasound (i.e. indirect assessment) of the IAN position for local anaesthetic deposition59 or injected coloured dye on cadavers to assess proximity of injectate deposition to the IAN.60 There are currently ongoing studies using B-mode ultrasound (i.e. direct assessment) to directly visualise the IAN while using intraoral ultrasound to guide intraoral inferior alveolar blocks on patients with subsequent objective pulpal anaesthesia testing.This paper provides a review of the pharmacology, techniques, and advances of local anaesthesia use in dentistry and should serve as a baseline for understanding that general dental practitioners possess for safe treatment of patients. Clinicians are encouraged to continue to expand both their didactic knowledge and practical clinical skills through advanced reading, discussion with colleagues, continuing education courses and treatment of patients.None.None.1.Gaffen AS, Haas DA. 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This activity reviews the indications, contraindications, anatomical considerations, equipment, techniques, and complications of local anesthesia in dentistry and highlights the dental team's role in the safe and efficient delivery of local anesthesia.Objectives:Identify the indications and contraindications of local anesthesia in dentistry.Review the anatomical course of the nerves supplying the oral cavity.Explain the different local anesthetic techniques utilized in dentistry.Outline the potential complications of using local anesthesia in dentistry.Access free multiple choice questions on this topic.Pain is an unpleasant sensory and psychological experience resulting from actual or potential tissue damage and is commonly associated with dental treatment.[1]Local anesthesia is a safe and effective way of managing pain. Anesthetic agents work by reversibly binding to sodium channels, preventing the entry of sodium into the cells and thereby inhibiting the propagation of nerve impulses. Consequently, nociceptive impulses associated with painful stimuli do not reach the brain, and the patient does not perceive pain.[2][3]The maxillary and mandibular branches of the trigeminal nerve provide sensory innervation to the soft and hard tissues of the oral cavity.[4][5]The anesthetic solution must be injected adjacent to the nerve supplying the area where dental treatment will be performed. A thorough understanding of these trigeminal nerve branches and associated anatomical landmarks is essential.[2]Local anesthesia in dentistry can be given as either infiltration or block anesthesia. Generally, infiltration anesthesia is commonly used in the maxilla, whereas block anesthesia is frequently used in the mandible.[6]In addition, there are supplemental local anesthesia techniques that can be utilized when infiltration and block anesthesia have failed to achieve profound anesthesia. Supplemental techniques include intraligamentary, intraosseous, intrapulpal, and interseptal anesthesia.[7]This paper will explore the anatomical course of the nerves supplying the oral cavity and the different techniques utilized to anesthetize these nerves prior to delivering dental treatment. The equipment required for dental local anesthesia, including common anesthetic agents and their applications, will be considered. Furthermore, the prevention and management of complications associated with local anesthesia will be discussed.Infraorbital NerveThe infraorbital nerve originates from the maxillary division of the trigeminal nerve as it enters the inferior orbital fissure after passing through the pterygopalatine fossa. The nerve travels along the infraorbital groove and infraorbital canal in the orbital floor before entering the face via the infraorbital foramen.[4]The infraorbital nerve delivers sensation to the maxillary teeth via the superior alveolar nerve branches prior to entering the infraorbital foramen. It divides into palpebral, nasal, and superior labial terminal branches on the face. These terminal branches provide sensation to the skin of the lower eyelid, nose, cheek, and upper lip.[4]Superior Alveolar NervesThe posterior superior alveolar (PSA) nerve emerges before the maxillary nerve enters the infraorbital groove. It descends along the maxillary tuberosity, where it innervates the gingiva and buccal mucosa of the molar region. It then enters the posterior alveolar canal and provides sensory fibers to the maxillary sinus mucosa, the maxilla, maxillary molar teeth, and periodontium.[4]The middle superior alveolar (MSA) nerve branches off within the infraorbital canal and runs down along the maxillary sinus lateral wall. This nerve innervates the maxillary premolar teeth and periodontium. In some individuals, the MSA nerve is absent, and the premolar teeth are supplied by the PSA nerve instead.[4]The anterior superior alveolar (ASA) nerve also originates within the infraorbital canal and descends along the anterior wall of the maxillary sinus. This nerve innervates the canine and incisor teeth and their periodontium.[4]Palatine NervesThe palatine nerves originate from the pterygopalatine ganglion, which is supplied by the maxillary division of the trigeminal nerve[4].The greater palatine nerve runs through the greater palatine canal and enters the greater palatine foramen, usually located medial to the third maxillary molar, to innervate the hard palate and palatal gingiva.[4]The lesser palatine nerve descends through the lesser palatine foramen, which is posteromedial to the greater palatine foramen, to provide sensation to the soft palate, uvula, and tonsils.[4]The nasopalatine nerve passes across the roof of the nasal cavity, running along the nasal septum. The nerve then emerges through the incisive foramen of the hard palate to innervate the anterior hard palate and anterior palatal gingiva. The nasopalatine nerve also provides sensory fibers to the maxillary incisors in some patients.[4]Buccal NerveThe buccal nerve, also known as the long buccal nerve, stems from the anterior trunk of the mandibular division of the trigeminal nerve.[5]It descends along the lateral pterygoid muscle and perforates the temporalis tendon, where it provides sensory branches to the posterior buccal mucosa and the buccal gingiva of the second and third mandibular molars. The nerve runs across the buccinator and provides sensory innervation to the skin over the anterior buccinator and the mandibular vestibule. The buccal mucosa innervation extends superiorly up to the region opposite the maxillary molars and anteriorly to the lateral aspect of the lips.[8]Lingual NerveThe lingual nerve stems from the posterior trunk of the mandibular division of the trigeminal nerve. It descends between the tensor veli palatini, lateral pterygoid, and medial pterygoid muscles to enter the pterygomandibular space.[5]The lingual nerve passes anterior and medial to the inferior alveolar nerve (IAN).[9]It runs to the body of the mandible, passing medial to the third molar and above the mylohyoid line, generally 2 or 3 mm below the alveolar crest, where it is only covered by gingival mucoperiosteum. The nerve then runs across the extrinsic tongue muscles, mylohyoid muscle, sublingual gland, and submandibular glandthe nerve curves around the submandibular duct before dividing into its terminal branches.The lingual nerve provides somatic sensation to the anterior two-thirds of the tongue, the floor of the mouth, and lingual gingiva. The lingual nerve also carries fibers from the chorda tympani branch of the facial nerve to provide secretomotor supply to the submandibular and sublingual glands and to carry taste sensation from the anterior two-thirds of the tongue.[5]Inferior Alveolar Nerve (IAN)The IAN stems from the posterior trunk of the mandibular division of the trigeminal nerve.[5]It runs deep to the lateral pterygoid muscle and gives off a mylohyoid nerve branch before descending into the pterygomandibular space and entering the mandibular foramen. The nerve runs within the mandibular canal below the teeth apices, giving off branches innervating the teeth via their apical foramina and interdental branches to innervate the periodontium.At the mental foramen, located below the premolar apices, the IAN divides into incisive and mental nerves. The incisive nerve continues within the mandibular canal to supply the teeth and buccal gingiva of the first premolar, canine, and incisors. The incisive nerve sometimes extends beyond the midline to provide additional sensation to the contralateral anterior teeth. The mental nerve exits via the mental foramen to supply the skin and labial mucosa of the lower lip and chin.[5]The mylohyoid nerve originates from the IAN approximately 13.4 to 14.7 mm above the mandibular foramen.[10]It pierces the sphenomandibular ligament and runs along the mylohyoid groove of the lingual surface of the mandible. It provides motor supply to the mylohyoid and anterior belly of digastric muscles.[5]Despite its primarily motor nature, in some cases, the mylohyoid nerve can provide accessory sensory innervation to the mandibular teeth, associated soft tissues, and submental skin. This can be due to anastomosis with the lingual nerve and is an anatomical variation of the mylohyoid nerve that can account for the failure to fully anesthetize mandibular teeth and soft tissues.[10]Pterygomandibular SpaceThe pterygomandibular space contains the inferior alveolar neurovascular bundle, the lingual nerve, and the sphenomandibular ligament.[9]It is bound by the lateral pterygoid muscle superiorly, the medial pterygoid muscle inferiorly and medially, the mandibular ramus laterally, the parotid gland posteriorly, and the pterygomandibular raphe anteriorly. The pterygomandibular raphe is formed by the junction of the buccinator and superior constrictor muscles.[9][11]The space is mainly made up of loose areolar connective tissue.[9]The aim of an IAN block (IANB) is to deliver the anesthetic solution to the vicinity of the IAN in the pterygomandibular space.[11]The inferior alveolar neurovascular bundle is located in the sulcus colli, a bony depression on the medial aspect of the ramus. The mandibular foramen is positioned at the inferior aspect of the sulcus colli, inferior to the midline of the ramus, and the level of the mandibular occlusal plane.[11]A crest of thick bone, known as the crista endocoronoidea, forms the anterior border of the sulcus colli, and it should be contacted during IANB administration. Contacting this bony protrusion helps ensure that the anesthetic solution is delivered adjacent to the IAN, without mechanical injury to the nerve and superior to the lingula.[9]The lingula is a bony projection anterior to the mandibular foramen. It is important to deliver the IANB above the lingula because the sphenomandibular ligament attaches to the lingula, and this fibrous ligament can prevent the effective diffusion of the anesthetic solution.[9][11]Local anaesthesia is essential for pain management in dentistry. Dental treatments can be associated with mechanical, thermal, or chemical stimuli, eliciting a painful response. Such dental treatments may include oral surgery, periodontal, endodontic, prosthetic, and restorative treatments. Local anesthesia is used to provide temporary sensory loss to allow for the delivery of dental treatment.[11]A known allergy to a local anesthetic agent or an ingredient of the anesthetic solution contraindicates its use. Allergy is the only absolute contraindication to local anesthesia. However, some anesthetic agents or techniques should be avoided or used with caution in certain individuals.Furthermore,toxicity and potential interactions need to be considered. Toxicity can result from exceeding the maximum recommended anesthetic dose or from the concomitant use of the anesthetic agent by the patient. Drug interactions with local anesthetics are rare. However, interactions of vasoconstrictors with beta-blockers, tricyclic antidepressants, amphetamines, and volatile anesthetics leading to hypertension and cardiac arrhythmias have been reported.[3]Cautionis Necessary for the Following CircumstancesHepatic or Renal ImpairmentLower anesthetic doses should be considered for patients with hepatic or renal impairment, as reduced liver or kidney function can affect the metabolism of anesthetic agents. Lidocaine and mepivacaine are almost entirely metabolized in the liver, with a small percentage being excreted unchanged in the urine. Prilocaine is metabolized by kidneys, lungs, and liver. Whereas for articaine, only 10-15% of the drug is metabolized by the liver, and the remaining drug is converted to articainic acid, a non-toxic and inactive form, in the blood.[6]Cardiovascular DiseaseAdrenaline is commonly added to local anesthetic agents, and caution is recommended for patients with cardiovascular disease. A maximum dose of adrenaline of 0.04 mg is advised for patients with cardiovascular disease, compared to 0.2 mg for a healthy adult.[12]However, there is limited evidence on the effects of the adrenaline found in local anesthesia on patients with cardiovascular disease. Using a low dose of adrenaline does not significantly affect blood pressure or heart rate. Adverse effects can be associated with intravascular injection, injection of a highly vascular area, or exceeding the recommended dose.[13]Furthermore, intraligamental and intraosseous injection with adrenaline-containing anesthetics is not recommended for patients with cardiovascular disease as the drug rapidly enters the circulation with these techniques.[7]Elderly PatientsCaution is essential when delivering local anesthesia to the elderly, and lower doses should be considered due to reduced kidney and liver function. Cardiovascular disease is also more common in the elderly, and limiting the use of adrenaline should be considered.[3]PregnancyLidocaine can be relatively safe, and there is no clear evidence that local anesthesia increases the risk of complications for the mother or fetus. However, any local anesthesia administered can cross the placenta and enter the fetus. Therefore, the operator should weigh the benefits and risks of local anesthesia, considering the health of the mother and fetus, the latest evidence regarding local anesthesia, and the risk of delaying treatment. It is generally recommended that elective dental treatment be deferred in pregnancys first and third trimesters.[3][14]Some clinicians avoid using prilocaine with felypressin in pregnancy as there is a theoretical risk of labor induction with felypressin and fetal methemoglobinemia with prilocaine. Nonetheless, these complications are rare at the low doses utilized in dentistry.[15]Patients with Bleeding Disorders or Taking AnticoagulantsLocal infiltration techniques are preferred over block anesthesia for patients with inherited bleeding disorders. This is because of the risk of intramuscular hematoma with potential airway compromise associated with IAN or PSA nerve blocks in these patients.[16]Although some practitioners may be concerned with block anesthesia in anticoagulated patients, studies suggest that mandibular blocks can be used safely for anticoagulated patients.[17]Personal protective equipmentDental syringeA 25, 27, or 30 gauge, short or long dental needle, depending on the anesthetic techniqueLocal anesthetic solutionTopical anesthesia can be consideredLocal Anesthetic AgentsLocal anesthetics are divided into two classes: amides and esters. Amide anesthetics are common in dentistry, including lidocaine, prilocaine, mepivacaine, and bupivacaine. Articaine is an amide anesthetic with an ester linkage. Ester anesthetics are less frequently utilized in dentistry, but drugs such as benzocaine may be used for topical anesthesia.[3]The gold standard and most widely used local anesthetic in dentistry is lidocaine due to its safety and effectiveness. Adrenaline is added to lidocaine to counteract its vasodilating properties and delay drug absorption, which prolongs the duration of anesthesia and reduces the toxicity risk.[6]Articaine with adrenaline, while not as frequently utilized as lidocaine, is heavily relied upon as a result of its superior solubility. Articaine has a high lipid solubility due to its thiophene ring and can diffuse across the maxillary and mandibular bone more readily than other anesthetics. Articaine buccal infiltrations are particularly useful in the posterior mandible as, unlike other agents, articaine can penetrate the dense cortical bone to anesthetize the IAN. It must be noted that articaine is not recommended for IANB due to a greater risk of nerve damage.[6]Comparatively, prilocaine and mepivacaine have weak vasodilation properties and can be used without adrenaline. Both are short-acting anesthetics and good options for children, the elderly, and patients with contraindications to adrenaline.[6]Children are more prone to soft tissue injury by inadvertently biting soft tissues when anesthetized; therefore, a short-acting anesthetic can be advantageous.[3]Mepivacaine is the anesthetic of choice for patients with cardiovascular disease. Furthermore, studies suggest that mepivacaine can be more successful than other agents in anesthetizing teeth with irreversible pulpitis [6]. Bupivacaine is a long-acting anesthetic with effects lasting up to eight hours, meaning it is less commonly used in dentistry, where prolonged anesthesia is not often required.[6]Local anesthesia is generally administered by dentists, dental hygienists, or dental therapists with the support of a dental assistant. The dental assistant ensures that the necessary equipment and materials are available. The operator or assistant can assemble the dental syringe. The operator administers the local anesthesia and appropriately disposes of the sharp instruments. The dental assistant is then responsible for sterilizing or disposing of the remaining equipment. Either the operator or the assistant can give the patient post-operative instructions concerning the local anesthesia following the procedure.The patients medical history, including any medical conditions, medications, and allergies, should be reviewed as this can affect the choice of the anesthetic agent. The patients weight should be known to avoid exceeding the safe dose of anesthetic solution. The dental syringe is assembled by the operator or dental assistant, confirming the anesthetic agent to be utilized and its expiry date. Under good lighting, local anesthesia is administered with the patient in the dental chair in a supine or semi-supine position. Anatomical landmarks are observed before administering the local anesthesia. Furthermore, the application of topical anesthesia or freezing can be considered before injection to reduce discomfort.[6]Infiltration AnesthesiaBuccal InfiltrationThe needle is inserted 2or 3 mm into the buccal sulcus adjacent to the tooth to be treated. The solution diffuses across the periosteum and alveolar bone to anesthetize the nerves supplying sensation to the tooth, periodontium, and buccal gingiva.[16]Infiltration anesthesia is commonly reserved for the maxilla because the maxillas porous structure allows the anesthetic solution to easily penetrate the bone.[6]However, the introduction of articaine has facilitated mandibular buccal infiltrations. Articaine has a high lipid solubility and can be used for buccal infiltrations in the posterior mandible as an alternative to or to supplement an IANB. Success rates of 84 to 94% have been reported for the ability of articaine buccal infiltrations to anesthetize the mandibular molars.[11][16]In addition, studies have suggested that palatal anesthesiacan be achieved following a maxillary buccal infiltration with articaine, negating the need for a separate palatal infiltration.[18]Palatal InfiltrationA palatal infiltration can be administered to anesthetize the nasopalatine or greater palatine nerve endings, thus providing anesthesia to the palatal gingiva. This injection is often described as painful due to the separation of the tightly-bound mucoperiosteum from the underlying hard palate bone. Methods to reduce discomfort may include topical anesthesia, cooling, applying pressure with a mirror handle, or slightly retracting the needle before injection.[1]Intrapapillary InfiltrationIntrapapillary (also known as transpapillary) infiltration can sometimes be used to avoid the need for a palatal infiltration. Following a buccal infiltration, the needle is inserted across the buccal interdental papilla and advanced above the alveolar bone to reach the palatal papilla. This technique anesthetizes the palatal interdental papilla and palatal free gingiva. Intrapapillary infiltration is commonly used for primary teeth.[1]Maxillary BlocksThe posterior superior alveolar block is used to anesthetize the maxillary molars, excluding the mesiobuccal root of the first molar. It also anesthetizes their periodontium and the adjacent buccal soft tissues. The needle is inserted 15 mm into the buccal vestibule distal to the malar at 45 degrees to the occlusal plane, and 1 ml of anesthetic solution is injected.[2]The middle superior alveolar block anesthetizes the maxillary premolars, the mesiobuccal root of the first molar, their periodontium, and adjacent buccal soft tissues. The needle is inserted 5 mm into the buccal vestibule adjacent to the maxillary second premolar, and 1 ml of anesthetic solution is given.[2]The anterior superior alveolar block anesthetizes the maxillary incisor and canine teeth, periodontium, and buccal soft tissues. The needle is inserted 5 mm into the buccal vestibule of the maxillary canine, and 1 ml of anesthetic solution is given.[2]The infraorbital block serves to anesthetize the ipsilateral maxillary teeth, periodontium, buccal soft tissues, maxillary tuberosity, and the skin of the lower eyelid, nose, cheek, and upper lip.[4]The needle is parallel to the second premolar and inserted into the mucosa above this tooth. The operators fingers of the non-dominant hand are placed over the infraorbital rim, and the needle is inserted until palpated in the vicinity of the infraorbital foramen. Alternatively, an extraoral approach can be used by inserting the needle through the skin and muscle in the mid-pupillary line after locating the inferior border of the infraorbital rim.[19]The greater palatine block anesthetizes the ipsilateral hard palate posterior to the canine tooth. The needle is inserted at the entrance of the greater palatine foramen to a depth of less than 5 mm until the bone is contacted and 0.5 ml of anesthetic solution is injected. The greater palatine foramen is a palpable depression or soft area on the hard palate, usually at the third maxillary molar level or 5 mm anterior to the hard and soft palate junction and halfway between the gingiva and palate midline.[2][4]The nasopalatine block anesthetizes the palatal premaxilla bilaterally. In some patients, it further provides some anesthesia to the maxillary incisors. Following buccal and intrapapillary infiltrations, the needle is inserted into the blanched incisive papilla to a depth of less than 5 mm until the bone is contacted and the tip contacts the lateral soft tissue of the incisive papilla. 0.25 ml of anesthetic solution is usually sufficient.[2]Mandibular BlocksInferior Alveolar Nerve Block (IANB)The IANB anesthetizes the ipsilateral mandibular teeth, periosteum, lower lip, chin, and the buccal soft tissues from the premolars to the midline.[2]The lingual nerve is generally anesthetized simultaneously, providing anesthesia to the ipsilateral lingual soft tissues, tongue, and floor of the mouth. For this technique, it is essential that the patient fully opens their mouth; otherwise, the IAN relaxes away from the lingula tip, and anesthesia may not be achieved.[11]The dental syringe is positioned above the contralateral premolars. A long needle is inserted 1 to 1.5 cm superior to the mandibular occlusal plane into the pterygotemporal depression to reach the pterygomandibular space.[2][9][11]The pterygotemporal depression is located between the pterygomandibular raphe and the coronoid notch of the mandibular ramus. The coronoid notch is the most concave point on the anterior ramus, and it can be palpated with the non-dominant hand before the injection. The needle is inserted 20 to 25 mm until the bone of the crista endocoronoidea is contacted.[11]The needle is then withdrawn 1 to 2 mm, aspiration is performed to prevent intravascular injection, and 1.5 ml of anesthetic solution is delivered. The needle is then withdrawn halfway, and the remaining solution is given to anesthetize the lingual nerve. Nonetheless, an IANB usually anesthetizes the lingual nerve even if this last step is omitted.[2]Anesthesia onset is generally three to five minutes.[11]Gow-Gates TechniqueThe Gow-Gates technique blocks the mandibular nerve adjacent to its division into auriculotemporal, inferior alveolar, mylohyoid, lingual, and buccal nerves [2]. Therefore anesthetizing all these nerves and providing anesthesia to the ipsilateral mandibular hard and soft tissues, anterior two-thirds of the tongue, the floor of the mouth, buccal mucosa, and the skin of the zygoma and temple.[12]The patient should open their mouth fully to allow the condyle to rotate and translate forwards. The operator palpates the condyle and retracts the cheek with their non-dominant hand. The syringe is placed at the level of the contralateral mandibular canine, and a long needle is inserted at the level of the upper second molar and just below the lateral pterygoid muscle insertion.[2]The needle is advanced 25 mm to contact the bone of the condylar neck.[11]The needle is then withdrawn 1 mm and the solution injected following aspiration.[2]The patient should be asked to maintain their mouth open for 20 seconds following the injection.[11]For experienced clinicians, the Gow-Gates approach is associated with a higher success rate and a lower risk of positive aspiration than the conventional IANB.[2][11]The higher success rate may be attributed to anesthetizing nerves that provide accessory innervation, such as the mylohyoid nerve. However, it has a slower onset of anesthesia of 5 to 25 minutes, and, in inexperienced clinicians, it can be associated with higher rates of complications and failure.[11]Vazirani-Akinosi TechniqueThe Vazirani-Akinosi technique, also known as the closed-mouth block, is helpful for patients with trismus or when the landmarks used for a conventional IANB are not clearly defined. This technique anesthetizes the inferior alveolar, mylohyoid, lingual, and buccal nerves in 5 to 7 minutes.[11]The patients mouth remains closed with the muscles of mastication relaxed, and the operator palpates the coronoid process before the injection. A long needle parallel to the maxillary occlusal plane is inserted between the coronoid process and the maxillary tuberosity at the level of the maxillary posterior teeth mucogingival junction.[11]The bevel of the needle should point away from the ramus to ensure deflection occurs towards the ramus. The needle is advanced to a depth of half the anteroposterior width of the ramus. For most adults, this depth is 25 mm from the maxillary tuberosity, and the hub of the needle sits above the mesial aspect of the upper second molar.[2]The anesthetic solution is injected following aspiration. In this technique, the bone should not be contacted; bone contact means that the needle was inserted too laterally.[11]Mental and Incisive BlocksAlthough the mental and incisive nerves are anesthetized by an IANB, mental and incisive blocks are useful when bilateral anesthesia is desired on or anterior to the mandibular premolars.[2]A short needle is inserted in the buccal sulcus next to the mental foramen, usually located between the premolar apices. The needle is inserted 5to 6 mm with the bevel facing the bone, and the anesthetic solution is administered following aspiration. Gentle pressure and massaging at the injection site for two minutes allow the anesthetic solution to enter the mental foramen to anesthetize the incisive nerve. The mental and incisive blocks provide anesthesia to the premolar, canine, incisor teeth and their periodontium, buccal soft tissues, the lower lip, and the chin. However, anesthesia of the lingual tissues is not provided.[2]Buccal Nerve BlockThe buccal nerve block is administered when anesthesia of the buccal mucosa or the buccal gingiva of the mandibular molars is required. The needle is advanced 1to 3 mm into the buccal vestibule distal to the second or third molar until the bone is contacted. The point of insertion is medial to the coronoid notch. A small amount of anesthetic solution, usually 0.25 ml, tends to suffice.[2]Modified IANB TechniquesTable 1 outlines examples of modified IANB techniques described in the literature. Supplemental Anesthesia TechniquesSupplemental techniques can be useful when conventional block and infiltration methods have failed to provide adequate anesthesia.Intraligamentary AnesthesiaA conventional dental syringe with a short needle or a specialized intraligamental syringe can be used.[7]The needle is inserted into the gingival sulcus at 30 degrees to the tooths long axis to reach the periodontal ligament (PDL) space, and it is advanced as far apically as possible.[2][7]The injection is given slowly mesially and then repeated distally.[2]The anesthetic solution diffuses from the PDL to the adjacent cancellous bone under the high pressure of the injection. Anesthesia onset is rapid, usually within 30 seconds. However, the duration of anesthesia is short, generally lasting 10 to 45 minutes.[7][11]Reported complications for intraligamentary injections include cartridge fracture due to high pressure; damage to unerupted teeth; damage to the PDL; tooth extrusion; discomfort; and bacteriemia with a potential risk of endocarditis for high-risk patients. The risks of cartridge fracture and discomfort can be mitigated by injecting slowly.[7]Generally, 0.2 ml of the solution is administered over 20 seconds.[2]The intraligamentary technique is commonly utilized following a failed IANB and appears to be more successful for exodontia than endodontic treatment.[7][11]Intraligamentary injection should be avoided in the infected or inflamed periodontium. It should also be avoided in primary teeth as enamel hypomineralisation or hypoplasia of the developing permanent teeth has been described.[11]Intraosseous AnesthesiaSpecialized intraosseous injection devices are used to drill a small hole through the cortical plate to reach the cancellous bone, and the anesthetic solution is injected slowly through this hole.[2]A small amount of anesthesia is given via local infiltration to the adjacent gingiva prior to perforation to reduce discomfort.[11]The perforation is made through the attached gingiva at a point 2 mm apical to the junction of two imaginary lines. These lines run horizontally across the buccal gingival margins and vertically to bisect the interdental papilla distal to the tooth to be anesthetized.[7]Anesthesia onset is rapid, often immediate, and anesthesia usually lasts 15 to 60 minutes, depending on the use of a vasoconstrictor.[11]Intraosseous injection should be avoided near the mental foramen and at sites of periapical infection or periodontal disease.[2][11]Intrapulpal AnesthesiaA short needle is inserted into the pulp chamber or root canal to a point where there is a tight fit, and 0.2 to 0.3 ml of anesthetic solution is delivered under pressure.[2][7]Anesthesia is achieved due to the pressure rather than the anesthetic solution, and similar results have been observed when using saline solution instead of an anesthetic agent.[11]Injecting all canals in multirooted teeth has been recommended. In addition, an alternative technique described is allowing the pulp chamber to bathe in the anesthetic solution for 30 seconds.[7]Intraseptal AnesthesiaA short needle is inserted at 90 degrees into the interdental papilla, 2 mm apical to the gingival margin. A small amount of anesthetic solution is administered when the needle enters the gingiva. The needle is then advanced until the bone is contacted and pushed a further 1 to 2 mm into the interdental septum by applying gentle pressure. Then, 0.2 to 0.4 ml of anesthetic solution is injected over at least 20 seconds. Intraseptal injection should be avoided at sites of infection.[2]Allergic ReactionsAllergy to amide local anesthetics is very rare, and an incidence of 0.1 to 1% has been reported. Some patients may have an allergic reaction to the sodium metabisulfite preservative found in many local anesthetics. Allergy to ester anesthetics, such as topical benzocaine, is more common. Allergic reactions can range from aphthous ulceration to anaphylaxis. Clinicians must be able to identify and manage such reactions.[3]Failure to Achieve AnesthesiaCauses for anesthetic failure include anatomical variations, inadequate technique, patient anxiety, and local infection or inflammation.[9]Anesthetic failure is more commonly associated with the conventional IANB method. The reported success rate of IANB is 80 to 85%. However, lower success rates ranging from 19 to 70% have been described for mandibular molars with irreversible pulpitis.[11]Accessory innervation is a common cause of anesthetic failure and should be suspected when an IANB provides anesthesia for the lower lip but not the dentition.[11]The mylohyoid nerve can provide accessory innervation to mandibular teeth, and an IANB generally fails to anesthetize the mylohyoid nerve as this nerve branches off superior to the point of injection. Furthermore, the sphenomandibular ligament provides a physical barrier to the diffusion of the anesthetic solution to the mylohyoid nerve, and the mylohyoid nerve sometimes travels within the bone.The Gow-Gates, Vazirani-Akinosi, or supplemental techniques can help address the mylohyoid accessory innervation. These techniques can also help overcome failure resulting from bifid mandibular canals, a variation that has been described in 0.35 to 1% of the population.[9]In addition, the buccal and great auricular nerves can provide accessory innervation to mandibular teeth via the retromolar foramen when present.[11]Injecting a small amount of anesthetic into the retromolar area can help anesthetize any accessory nerves entering the retromolar foramina. Furthermore, contralateral incisive nerve innervation is common, and when profound anesthesia in anterior teeth is not achieved, a contralateral incisive nerve block may be required.[5]There is also a link between skeletal pattern and IANB failure, with a greater risk of IANB failure in patients with retrognathic mandibles. One study found a failure rate of 14.5% for patients with retrognathic mandibles compared to 7.3 % for patients with class I skeletal patterns. This can be explained by the higher position of the mandibular foramen and a smaller mouth opening in retrognathic mandibles, both due to short condyles. Injecting the IANB at a higher point than usual, or using alternative techniques such as Gow-Gates and Vazirani-Akinosi methods, have been proposed to improve anesthetic success in retrognathic mandibles.[20]Furthermore, infection lowers the pH of the environment, and this acidity can affect the efficacy of local anesthesia. It is recommended that the anesthetic solution is injected as far as possible from the area of infection.[5]The pH of the anesthetic solution also plays a role. Local anesthesia is generally acidic, with the commonly used lidocaine with adrenaline having a pH of 3.5. Studies have shown that increasing the pH of local anesthetic, for example, by buffering with sodium bicarbonate, can shorten the speed of anesthesia onset and reduce patient discomfort during injection.[10]HematomaOccasionally, blood can extravasate and accumulate to form a hematoma when the needle punctures a blood vessel. A hematoma can cause soreness, facial swelling, and trismus. If a hematoma is suspected, the clinician should apply pressure to help stop the bleeding and ensure hemostasis has been achieved before the patients departure.[3]In addition, there are reported cases of patients where a maxillary artery puncture has led to a hematoma in the infratemporal fossa resulting in pressure in the IAN and lingual nerve, with sensory disturbances such as hyperalgesia, allodynia, and facial pain as a consequence [5]. Due to the higher needle insertion point, there is a greater risk of a maxillary artery or pterygoid plexus injury with the Gow-Gates and Vazirani-Akinosi techniques.[10]Intravascular InjectionIntravascular injection of local anesthesia can lead to palpations, visual disturbances, headaches, and vertigo. The patient should be monitored and reassured that the symptoms should be transient and self-limiting. An intravascular injection can be prevented by aspirating before injection. The negative pressure of aspiration withdraws blood into the cartridge if the needle tip is inside a blood vessel,warning the operator not to inject the anesthetic solution at this location.[3]The conventional IANB technique has the highest risk of positive aspiration.[2]The inferior alveolar vessels run posterior to the IAN and can be exposed to trauma from the needle during IANB. In an IANB, the needle must be retracted 1to 2 mm before aspiration as the inferior alveolar vein may be pinned to the bone, resulting in a false negative result of aspiration.[11]MethemoglobinemiaExceeding the maximum dose of ester anesthetics or prilocaine can lead to methemoglobinemia. Methemoglobinemia is when the iron in hemoglobin is changed from a ferrous to a ferric state, creating methemoglobin. The ferric hemoglobin has a high affinity for oxygen, preventing oxygen from dissociating and depriving tissues and organs of oxygen. This can lead to hypoxemia, with symptoms such as shortness of breath and cyanosis. It is a medical emergency, oxygen must be administered, and emergency services contacted. This condition is treated with intravenous methylene blue.[3]Needle FractureNeedle fracture is a rare complication of local anesthesia with modern needles. The most common cause of breakage is sudden movement. This usually involves patient movement, but cases of dentist movement, change in needle direction, and sudden swallowing have been reported. Patients may develop symptoms such as pain, trismus, swelling, and dysphagia. A few patients may remain asymptomatic.[21]Needle breakage is most likely with mandibular nerve blocks and if a needle with a small diameter is used. Therefore, a 27 gauge or less needle should be used for mandibular blocks. A long needle must be used so that at least 5 mm of the needle is outside the mucosa to allow retrieval in the case of breakage. The mandible should be held firmly, and the patient should be advised against sudden movements. Furthermore, forceful or repeated pre-bending of the needle should be avoided, and the needle should be changed if repeated injections are required.If a needle breaks, the broken fragment should be removed as soon as possible. Broken needle fragments following a mandibular block most commonly sit within the pterygomandibular region. However, migration to the external auditory canal, superficial post-auricular region, and base of the skull has been reported.[21]Nerve Injury and Altered SensationNerve injury may result from direct injury by the needle, damage to intraneural blood supply leading to a hematoma compressing the nerve, or neurotoxicity of the anesthetic solution. Neurotoxicity is believed to be greater for articaine and prilocaine than lidocaine.[22]Nerve injury can lead to temporary or permanent loss of sensation. Following an IANB, the reported incidence of temporary IAN and lingual nerve impaired sensation ranges from 0.15 to 0.54%, while permanent sensory loss ranges from 0.0001 to 0.01%.[23]The nerve at most significant risk of injury during an IANB is the lingual nerve, as it sits close to the mucosa without the bony protection of the lingula or sulcus colli bone.[9][22]Nerve injury with maxillary infiltration is very rare.[22]When a patient presents with sensory loss following local anesthesia, careful examination of the distribution of sensory loss should be performed. The patient is followed up regularly until resolution, and referral to a specialist should be considered.[22]Ocular ComplicationsOcular symptoms have been described as complications following intravascular injection into the maxillary artery. Retrograde flow of the anesthetic solution to the lacrimal and optic arteries can lead to anesthesia of the oculomotor, trochlear, and abducens nerves. Symptoms may include diplopia, ptosis, ophthalmoplegia, amaurosis, mydriasis, transient vision loss, and periorbital skin blanching. These are generally self-resolving.[3][5]Psychogenic ReactionsPatient anxiety regarding the administration of anesthesia or the dental treatment itself can lead to increased heart rate, changes in blood pressure, dyspnea, pallor, nausea, vomiting, and syncope.[3]Soft Tissue TraumaPatients should be warned that soft tissues such as lips, cheeks, and tongue can still be numb following dental treatment, and care must be taken until the anesthesia fully wears off. There is a risk of patients biting these soft tissues due to poor sensory feedback, leading to soft tissue trauma.[3]Systemic ToxicityToxicity can occur if the maximum safe dose of local anesthesia is exceeded and can lead to neurological and cardiac adverse effects. Excitatory neurological symptoms manifest initially. These may include visual disturbances, sensory disturbances, and seizures. This is followed by depressive symptoms, such as decreased consciousness which could lead to coma and respiratory arrest. Cardiac manifestations can range from tachyarrhythmia to bradyarrhythmia to cardiac arrest. Dental practitioners must be able to recognize and manage symptoms of toxicity.[3]Toxicity can be avoided by not exceeding the maximum anesthetic dose based on the patients weight. Table 2 outlines the maximum dosage for common anesthetic solutions used in dentistry. The percentage of anesthetic solution signifies the grams per 100 ml. For example, a 2% solution has 2 grams in 100 ml, therefore 20 mg/ml.To calculate the amount of anesthetic agent in a cartridge, the mg/ml figure is multiplied by the total solution in the cartridge, usually 1.8 ml or 2.2 ml. Table 3 illustrates an example of the calculation.[3]Toxicity is an emergency and, therefore, emergency medical service must be contacted. Management involves supplemental oxygen and intravenous medication, including Intralipid, midazolam if the patient has seizures, and vasopressor if the patient has hypotension or bradycardia. The patient should be monitored for ventricular tachycardia, ventricular fibrillation, acidosis, hyperkalemia, and hypercarbia, and these should be treated accordingly.[3]Table 2: Maximum dosage for anesthetic solutions commonly used in dentistry. Table 3: Example of maximum dose calculation. Transient Facial Nerve PalsyTransient facial palsy is a rare complication and can be immediate or delayed. Immediate palsy can occur due to direct anesthesia of the facial nerve after injection into the parotid gland when an IANB is given too posteriorly. Facial weakness occurs within minutes of the injection and resolves within 3 hours.[24]Delayed palsy occurs several hours or days after the local anesthetic administration, and recovery can take 24 hours to many months. The mechanism is more complicated, and postulated theories include: the anesthetic agent or mechanical injury from the needle leading to a sympathetic vascular reflex and ischemic neuritis; mechanical trauma leading to the activation of a latent herpes simplex or varicella-zoster virus, and these viral infections leading to neural sheath inflammation and facial palsy; nerve damage due to breakdown products of the local anesthetic; prolonged mouth opening stretching the facial nerve; and intravascular injection. It is important to protect the eye with an eye patch when a patient develops transient facial palsy. Artificial tears and sunglasses are also recommended.[24]TrismusTrismus can result from muscle spasticity if the needle pierces through a muscle of mastication or from a hematoma preventing mandible movements. Trismus is usually managed conservatively with a soft diet, analgesia, and physiotherapy.[3]Profound anesthesia is a prerequisite for most dental treatments as it allows the patient to be comfortable and the clinician to deliver treatment.[6][22]Furthermore, given that dental anxiety frequently stems from painful experiences, effective pain management through local anesthesia can be key in preventing and mitigating dental anxiety.[6]The selection of local anesthetic agents and delivery techniques can influence the efficacy of anesthesia.[6]Subsequently, dental practitioners must have a robust knowledge of the local anesthetic agents and techniques utilized in dentistry, including their indications and contraindications.Although local anesthesia is rarely associated with serious adverse effects, clinicians must additionally understand the potential complications to ensure patient safety. This must also extend to understanding how best to manage or prevent these complications.Furthermore, understanding the nerve supply to the dentition and surrounding tissues is essential to ensure that the anesthetic solution is deposited near the nerves supplying the area of interest. In addition, an awareness of possible anatomical variations allows the clinician to understand why anesthesia might fail and what methods can be utilized to overcome failure.Failure to achieve anesthesia can delay treatment and cause the patient to lose confidence in the clinician and dentistry. Clinicians must understand why anesthesia can fail and be able to utilize alternative techniques where conventional methods have failed.All members of the dental team have a responsibility to ensure patient safety. The operator and dental assistant should ensure that the anesthetic solution has not expired and the maximum dose is not exceeded.The dental team members must be aware of the risk of toxicity and understand how to prevent, identify, and manage toxicity. All team members must also be able to recognize and address any medical emergencies that may occur during or after the administration of local anesthesia. Dental assistants can also play a key role in patient care, for example, by providing reassurance to anxious patients. All team members must work together to deliver patient-centered care to ensure good patient outcomes.Review QuestionsTrigeminal Nerve Divisions. This illustration shows the distribution of the 3 trigeminal nerve divisions. The ophthalmic division gives rise to the lacrimal nerve and supplies the superior third of the face and skull. The maxillary divisionsupplies (more...)Facial and Trigeminal Nerve Interactions in Lacrimal Gland Function. This illustration shows the facial and trigeminal branches contributing to lacrimal gland function and includes the maxillary, great superficial petrosal, facial, deep petrosal, and (more...)Trigeminal Nerve. The image shows the mandibular division of the trigeminal nerve from the midline. The small figure provides an enlarged view of the otic ganglion. Henry Vandyke Carter, Public Domain, via Wikimedia Commons Mandibular foramen Image courtesy O.Chaigasame Maxillary blocks Contributed and Modified from: Henry Vandyke Carter, Public domain, via Wikimedia Commons 1.Sruthi MA, Ramakrishnan M. Transpapillary Injection Technique as a Substitute for Palatal Infiltration: A Split-mouth Randomized Clinical Trial. Int J Clin Pediatr Dent. 2021 Sep-Oct;14(5):640-643. [PMC free article: PMC8645621] [PubMed: 34934275]2.Reed KL, Malamed SF, Fonner AM. Local anesthesia part 2: technical considerations. Anesth Prog. 2012 Fall;59(3):127-36; quiz 137. 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[PMC free article: PMC3309298] [PubMed: 22428971]Disclosure: Marie Mathison declares no relevant financial relationships with ineligible companies.Disclosure: Tom Pepper declares no relevant financial relationships with ineligible companies.

Most commonly used local anesthetic in dentistry. Types of local anesthesia in dentistry. Types of local anesthesia. Different types of local anesthesia in dentistry. Names of local anesthesia in dentistry. What is local anesthesia in dentistry.