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Edited by Peter J. Davis and Etsuro K. New York, Mosby Year Book, The resuscitative effort of this classic text was carried out in the 5th edition by Peter J. Davis and Etsuro Motoyama, who received the baton from Robert M. Representing one of only two comprehensive multi-authored texts on the subject of pediatric anesthesia, this text, in my opinion, had become the text preferred by most pediatric anesthesiologists. The arduous efforts by Motoyana and Davis have, once again, payed off in the 6th edition, providing an important resource to all anesthesiologists who practice pediatric anesthesia. The text maintains a handsome academic physical appearance. However, the blue background on the faceplate and side of the text peels rather easily, in a manner similar to a teflon frying pan. I was surprised several times by the referencing of very recent works. The referencing is thorough, and I am particularly fond of the inclusion in the text of both the author and date for each reference. The organization of the text continues to be in the same four parts as in previous editions. The chapter on cardiovascular physiology has been skillfully rewritten and expanded by Maureen Strafford in a manner that reflects her training in pediatric cardiology and her extensive experience in cardiac anesthesiology. The chapter on pharmacology remains one of my favorites, and has been improved through contributions by Jerrold Lerman. The similarity is easily overlooked, because I can think of nobody better to write a chapter on pediatric pain than Berde. A cursory chapter on fluid and blood therapy has been disbanded, but one can still find most of this information in other sections of the new edition. The chapter on hypotensive anesthesia has appropriately been integrated into the chapter on blood conservation. The third section of the text deals with anesthesia for particular medical and surgical subgroups. The chapters in this section are uniformly strong. There is considerable repetition, but this is understandable, because these chapters are clearly meant to stand alone. Of note is one apparent error, which pertained to an unusually low dose of morphine recommended in the chapter on anesthesia for dentistry. Because there is a new chapter on the history of pediatric anesthesia written by Robert Smith, the title of the section might need to be changed. I would prefer not to think of history as a problem. The remainder of the chapters in this section are titled as in the previous edition, with some revisions and lots of updates. The chapter on systemic disorders is well written and very useful, but does have a few errors in transcription i. Such errors were rare occurrences in this text. Lastly, this volume continues to include an appendix on syndromes and their anesthetic implications. This appendix is a real bonus, which has great value in the clinical setting. Many practitioners, myself included, find that, even with years of pediatric experience, it is nearly impossible to keep the syndromes straight. In summary, this comprehensive textbook is outstanding and is well worth the price. It can be recommended to anesthesia practitioners at all levels who wish to improve their skills in pediatric anesthesia, or who simply need a strong reference text.

Chapter 2 : Yearbook Of Anesthesiology calendrierdelascience.com: Sehgal Raminder: Books

The Year Book of Anesthesia Stephen C. Ronald MD. Anesthesia & Analgesia: May - Volume 72 - Issue 5 - ppg BOOK REVIEW: PDF Only.

Dent Res J Isfahan. Panchkula Haryana , India Address for correspondence: This article has been cited by other articles in PMC. Abstract Local anesthesia forms the major part of pain-control techniques in dentistry. The prevention and elimination of pain during dental treatment has benefited patients, their doctors and dental hygienists, enabling the dental profession to make tremendous therapeutic advances that would otherwise have been impossible. Introduced in the late s, the amide local anesthetics represent the most used drugs in dentistry. Local anesthetics also represent the safest and most effective drugs in all of medicine for the prevention and management of pain. They are also accompanied by various adverse effects which should be well known and be able to be controlled by the clinician. The article reviews the types of agents used as local anesthetics and their effects on the human body. Pain and dentistry are often synonymous in the minds of patients, especially those with poor dentition due to multiple extractions, periodontal disease requiring surgery or symptomatic teeth requiring endodontic therapy. Dental practitioners, before the procedure, identify a good anesthetic as one that allows them to focus solely on operative procedures without distractions from pain-induced patient movements. Research has shown that the fear of pain associated with dentistry is closely associated with intraoral administration of local anesthetics, which is the most common method for blocking pain during dental procedures. This is considered aversive due to the pain associated with the injection and the perceived threat of needle puncture prior to the injection. History of local anesthesia The history of local anesthesia started in , when cocaine was isolated by Niemann. In , the opthalmologist Koller was the first, who used cocaine for topical anesthesia in ophthalmological surgery. In , regional anesthesia in the oral cavity was first performed by the surgeon Halsted, when he removed a wisdom tooth without pain. However, a number of adverse effects were observed with the clinical use of cocaine. Thus, other local anesthetic agents had to be developed. In , Einhorn reported the synthesis of procaine, which was the first ester-type local anesthetic agent. Procaine was the most commonly used local anesthetic for more than four decades. Lidocaine was marketed in and is currently the most commonly used local anesthetic in dentistry worldwide, though other amide local anesthetics were introduced into clinical use such as; mepivacaine , prilocaine and bupivacaine In , articaine was synthesized by the chemist Muschaweck and was approved in as a local anesthetic in Germany. The use of reversible local anesthetic chemical agents is the most common method to achieve pain control in dental practice. Table 1 Open in a separate window Pharmacology Local anesthesia is induced when propagation of action potentials is prevented, such that sensation cannot be transmitted from the source of stimulation, such as tooth or the periodontium, to the brain. Local anesthetics work by blocking the entry of sodium ions into their channels, thereby preventing the transient increase in permeability of the nerve membrane to sodium that is required for an action potential to occur. Structurally, local anesthetics have specific fundamental features in common. These include a lipophilic group, joined by an amide or ester linkage to a carbon chain which, in turn, is joined to a hydrophilic group. Local anesthetics are classified by these amide or ester linkages. Blood levels The blood level of local anesthetic agent following injection is a function of both rate of absorption from the site of injection and uptake into the systemic circulation, and its removal through distribution from the vascular compartment into tissue compartments, and elimination via metabolic and excretory pathways. If toxic levels are reached or exceeded, local anesthetics may cause toxic signs and symptoms, which are mainly referable to the central nervous system and to the cardiac vascular system. Pharmacokinetic parameters, including maximum serum levels, time of maximum serum levels and elimination half time, are important to estimate the risk of systemic intoxication following injection and to recommend maximal dose in single and repeated injection. The absorption and subsequent blood level of local anesthetic agents are related to the total dose of drug administered. For most agents, there is a linear relationship between the amount of drug administered and the resultant peak of anesthetic blood level. The peak of anesthetic blood level does not appear to be related to either the concentration or volume of the local

anesthetic solution employed. Failure of anesthesia Failure of local anesthetics to achieve profound analgesia may be related to inaccurate anatomic placement of local anesthetic solution, use of inadequate amount of anesthetic solution, allowing insufficient time for the solution to diffuse to be effective, injection of solution into inflamed or infected tissues, and finally use of an outdated or improperly stored anesthetic solution. It is recommended that a local anesthetic not be injected into an infected tissue because of the risk of spreading the infection and the increased probability of achieving less than effective anesthetic results owing to the low pH within the infected tissue maintaining the ionized, non lipid-soluble state to the anesthetic. Adverse reactions Complications of local anesthetic administration include both local effects and systemic effects. A patient may be allergic to other compounds in the anesthetic cartridge. For example, methylparabens are preservatives necessary for multidose vials and were present in dental cartridges in the past. They are no longer included as dental cartridges are single-use items. Allergy to para-aminobenzoic acid would rule out use of esters and methylparabens. It may be best to avoid a vasoconstrictor if there is a true documented allergy to sulfites, as metabisulfite is added as an antioxidant whenever vasoconstrictor is present. Toxic complications as a result of an overdose of local anesthetic solution, resulting in dangerously high concentrations in the brain, are usually produced only by rapid injection directly into a blood vessel. As the anesthetic solutions of the amide type e. A normal volume of anesthetic will become potentially toxic in such people. The final route for elimination of the metabolized anesthetic solution is excretion in the urine and so any patient with impaired renal function will also be unable to eliminate these products and be predisposed to toxic accumulation. Psychogenic reactions Anxiety-induced events are by far the most common adverse reaction associated with local anesthetics in dentistry. These may manifest in numerous ways, the most common of which is syncope. In addition, they may present with a wide variety of symptoms, including hyperventilation, nausea, vomiting and alterations in heart rate or blood pressure. Psychogenic reactions are often misdiagnosed as allergic reactions and may also mimic them, with signs such as urticaria, edema and bronchospasm. Methemoglobinemia This uncommon adverse reaction is associated most notably with prilocaine but may also occur with articaine or the topical anesthetic benzocaine. Cyanosis becomes apparent when methemoglobin levels are low, but symptoms of nausea, sedation, seizures and even may result in coma when levels are very high. Facial nerve palsy The most common neurological complication following an inferior alveolar nerve block is a facial nerve palsy. Generalized weakness of the ipsilateral side of the face, inability to close the eyelids, obliteration of the nasolabial fold, drooping of the corner of the mouth and deviation of the mouth to the unaffected side. Facial nerve palsy following inferior alveolar nerve block may appear immediately or be delayed. The immediate transient palsy generally recovers within 3 h of administration of the local anesthetic. It is probably due to anesthesia of the facial nerve trunk as a result of an abnormal nerve anatomy such as passage of the nerve along the deep surface of the parotid gland. Alternatively, it may be caused by a congenital abnormality such as the gland failing to envelop the nerve and its divisions, thus increasing its chances of direct exposure to local anesthetic solution. Delayed-onset facial palsy occurs after several hours and in some cases many days after the administration of the anesthetic. Some hypotheses have been put forward to explain this: The anesthetic solution or its breakdown products stimulate the sympathetic plexus associated with the external carotid artery. From the external carotid artery, fibers of this plexus continue in association with the stylomastoid artery as it passes into the parotid gland. The stimulation of the stylomastoid sympathetic plexus causes a delayed reflex spasm of the vasa nervorum of the facial nerve, leading to ischaemic neuritis and secondary edema. The origin of these sympathetic fibers is in the superior cervical ganglion which gives rise to lateral, medial and anterior branches. Of these, it is the anterior branches that pass onto the common and external carotid arteries to form plexuses that accompany the blood vessels. Most of these reactions are transient and resolve within 8 weeks, but they may become permanent. Articaine and prilocaine were reported as more likely than other anesthetics to be associated with paresthesia, a difference that was statistically significant when their distribution of use was taken into account. Direct inferior nerve trauma feels like an electric shock, sometimes causing the patient to suddenly jerk their head. The practitioner should cease the injection immediately if this occurs. Significant bleeding may produce swelling, act as an irritant to the tissues, and causes pain and trismus. Theoretically, the localized collection of blood becomes an ideal culture

medium for bacteria, although infection of a hematoma is unusual. Total body hemiparesis Inadvertent intravascular injection of local anesthetic with subsequent retrograde internal movement in branches of the internal carotid artery has been suggested as a mechanism for a reported case of total body hemiparesis 15 min following inferior dental nerve block. This comprised ptosis, occipital and neck stiffness, anesthesia of the right side of the face with dysphasia, and led to complete aphasia and a right hemiparesis. The effects lasted for approximately 45 min and were attributed to excess pressure created during the administration of the injection leading to a retrograde flow into the internal carotid artery. Previous recommendations, now known to be wrong, precluded the use of specific local anesthetics in these patients. Today it is well accepted that all local anesthetics are safe for patients who are susceptible to malignant hyperthermia. Adverse effects of epinephrine Depending on the dose, sympathomimetic amines can evoke a variety of systemic reactions. The major systemic effects of injected sympathicomimetic amines involve the cardiovascular system. Cardiovascular responses of epinephrine often include tachycardia, mild hypertension, and occasionally premature ventricular contractions. The majority of adverse reactions are mild and short of duration. This arose due to penetration of the local anesthetic through the lateral pharyngeal and prevertebral spaces, causing blockade of the stellate ganglion. The features of the syndrome include: Flushing of the face on the same side, ptosis of the eyelids, vasodilatation of the conjunctiva; pupillary constriction and occasionally a rash over the neck, face, shoulder and arm of the ipsilateral side. Paralysis of combinations of cranial nerves Use of techniques such as Gow-Gates may result in local anesthetic, which is deposited in a superior position, gaining access to the cavernous sinus following inadvertent intravenous injection. Careful aspiration and direction of the needle to an area with fewer large-bore blood vessels such as the lateral aspect of the condyle are recommended to avoid this complication. The suggested explanation implies that the venous systems within the mandibular region provide access for the anesthetic to the middle ear and that this, due to the added vasoconstrictor, results in localized vasospasm of the cochlear division of the internal auditory artery, leading to dysfunction of the cochlear nerve. Therefore, it is recommended that the maximum safe dose be halved in such patients. Whilst performing these primary measures the practitioner should summon help and contact the nearest casualty department. Placing the patient supine and administering oxygen will allow monitoring of vital signs pulse, respiration and blood pressure. Convulsions, which occur in some cases, may need to be treated with a slow over 2 min intravenous infusion of 10 mg diazepam. The rectal route is an alternative when intravenous access is difficult. Endocarditis risk Injections such as the intraligamentary injection can force bacteria into the systemic circulation and cause bacterial endocarditis. Local anesthetics during pregnancy All local anesthetics cross the placenta to some degree. Although this is a highly unlikely effect at the low dose of felypressin used in local anesthetics, it is best avoided during pregnancy. Lidocaine with epinephrine is commonly used for pregnant dental patients. Although high-dose vasoconstrictors used to manage significant hypotension may be a concern for pregnant patients, the doses of epinephrine used in local anesthetic formulations for dentistry are so low that they are unlikely to significantly affect uterine blood flow. The benefits of epinephrine or levonordefrin at the concentrations found in dental anesthetic cartridges justify their use. Local anesthetics in children Available data suggest that the adverse reactions in pediatric patients are commonly caused by inadequate dosage reduction. For very obese children, the maximum dose should be calculated on the basis of lean body weight or ideal weight, not the true body weight.

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Research and development of blocking anesthesia harkens back to , when pressure-point anesthesia was used to compress nerves and vasculature to numb parts of the body. Cocaine was introduced years later as the first local anesthetic for surgical procedures for glaucoma, followed in by procaine ie, Novocain , which changed the way dentistry is performed. Demonstrating a more rapid onset, lidocaine remains safe and efficacious among the many local anesthetics available today. The efficacy and safety of local anesthetics are dependent upon their molecular structure. How these molecules interact with the bodyâ€”which vary among local anestheticsâ€”impacts their potency and duration of anesthetic effects. Therefore, it behooves clinicians to understand the basics of local anesthetics, their componentry, and their mechanism of action in order to deliver them appropriately and effectively. Basic Principles of Local Anesthetics Local anesthetics are either esters or amides based on the aromatic ring in their molecular structure Table 1. Other portions of their molecules include an amine group and an intermediate linkage. The amine group, usually a secondary or tertiary amine, is associated with water solubility and carrying the molecule through the system, but is not necessary for anesthetic activity. Compounds lacking the amine portion are insoluble in water and useful only topically. The intermediate linkage, which connects to aromatic residue via an ester or amide linkage, determines the route of metabolism, allergic potential, and how the compound performs clinically. The mechanism of action of local anesthetics involves blocking the sensation of pain by interfering with the propagation of nerve impulses along peripheral nerve fibers. Factors influencing local anesthetic effectiveness include its pKa, lipid solubility, and protein binding. All local anesthetics are weak bases with a pKa range of 7. The pKa ie, the free base or uncharged state that readily penetrates connective tissue and membranes is responsible for the onset of anesthesia and how available in the system it will be Table 2. Local anesthetics that demonstrate a lower lipid solubility are typically provided by manufacturers in a higher concentration Table 3. Protein binding affects duration of anesthetic effects. The more tightly bound the local anesthetic is to the sodium channel and blocking it, the longer the nerve will be blocked and the longer the duration of action Table 4. Local anesthetic selection will, therefore, be dependent upon multiple factors, including type of clinical procedure performed and level of anesthetic required and for how long. The elimination half-life of local anesthetics ie, time required for metabolic breakdown and total elimination will also be significant Table 6 , particularly in the context of managing local anesthesia overdose. Signs of moderate-to-severe overdose include seizures in conjunction with central nervous system depression and depressed vital signs ie, heart rate, blood pressure, respiration Figure 1. When a patient exhibits symptoms of over-dose, treatment should be stopped, and the patient reassured and given oxygen via a controlled airway. Vital signs should be monitored, and someone should remain with the patient at all times. The patient should be closely monitored for the time necessary for the local anesthetic to distribute and be metabolized ie, elimination half-life based on the number of cartridges used; this, therefore, makes understanding the elimination half-life significant. It is always advisable to remember that children, the elderly, and medically complex patients are more susceptible to local anesthetic overdose. It is also important to know the maximum doses of both local anesthetics Table 7 and vasoconstrictors Table 8.

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