International Journal Dental and Medical Sciences Research
Volume 5, Issue 2, Mar - Apr 2023 pp 430-433 www.ijdmsrjournal.com ISSN: 2582-6018

Evaluation of Local Anesthetic in dentistry: a Literature Review

Dr Srishti Sudhir Salunke, Dr Anuj Shailendra Dadhich, Dr Seemit Shah, Dr Harish Saluja, Dr Srishti Sudhir Salunke
Pravara institute of medical sciences, Loni, Maharashtra.
Pravara institute of medical sciences, Loni, Maharashtra.
Pravara institute of medical sciences, Loni, Maharashtra.
Pravara institute of medical sciences, Loni, Maharashtra.
Pravara institute of medical sciences, Loni, Maharashtra

Date of Submission: 12-03-2023 Date of Acceptance: 22-03-2023

ABSTRACT
The objective of this review was to investigate the efficacy of dental local anesthetics, as it is well known among clinicians that local anesthesia may be challenging in some circumstances. Therefore, the focus of this review was on the efficacy of the products used in dental local anesthesia.
A profound anesthesia is important for the success of any surgical procedure. The failures in local anesthetic action can be attributed to various factors. These include Intravascular injection, unusual anatomy, bone density, accessory innervations, double or accessory mental foramen, cross innervation , inability to achieve anesthesia in presence of tissue inflammation, inactive anesthetic solutions, incorrect technique, and lack of patient co-operation.
KEYWORDS: Efficacy of LA, Local Anesthesia, Ropivacaine, Dentistry

I. INTRODUCTION

The change that took place in Western Europe between 1750 and 1850, created an atmosphere favorable to the discovery of anesthetics. Dentists were responsible for the discovery of anesthesia, given their close day-to-day contact with pain. Doctors focused more on infections than pain. [1]

It was two dentists, then, who first introduced, anesthesia: Horace Wells (1815–1848), with nitrous oxide in 1844, [2–4] and William Thomas Green Morton (1819–1868), with ether in 1846. [5]

EVOLUTION OF LOCAL ANESTHESIA BEFORE COCA LEAF

One of the first examples of pain control by man was in Egypt over 4,500 years ago around the year 2500 BC. Paintings of apparatuses used to compress peripheral nerves to numb limbs were found on the walls inside the ancient Egyptian tomb of Saqqara [1]

EVOLUTION OF LOCAL ANESTHESIA SINCE COCA LEAF

Coca leaves are taken from a shrub of the genus Erythroxylum, a member of the Erythroxylaceae family, so named by Patricio Browne because of the reddish hue of the wood of the main species. [6]

Of the various species in this genus, Erythroxylum coca contains the highest concentration

of the alkaloid known as cocaine in its leaves, up to 0.7–1.8% by weight.[7-8] The earliest cultivation and use of the coca leaf in the Bolivian and Andean region date back to 700 B.C.[6]

Alfred Bühler hypothesized that the Arhuaco, a tribe from the Negro River region, were the first to discover the properties of the drug and spread this knowledge to other neighboring peoples. [9]

For some writers, Florentine Amerigo Vespucci (1451–1512) was the first European to document the human use of the coca leaf. [8, 10]

Austrian naturalist Carl Von Scherzer (1821–1903) traveled around the world in the frigate Novara in 1857–1958; during his stay in Peru, he collected a sizeable sample of coca leaves, which he sent to German chemist Albert Niemann (1834–1861).[11,12]Niemann, in

The Friedrich Wohler Laboratory in Gottingen, in 1860, managed to isolate the active principle, which he named cocaine [12]. Dr. William Stewart Halsted (1852–1922) and his coworker Richard John Hall (18?–1897) read Noyes' report and immediately became interested in local anesthesia.[13] On December 6, 1884, Hall published a report on the first successful nerve block, which happened to be achieved in the context of dentistry: Dr. Nash of New York was able to block the infraorbital plexus with 8 minims (approximately 0.5 ml) of 4% cocaine hydrochloride ("hydrochloride of cocaine" in

International Journal Dental and Medical Sciences Research
Volume 5, Issue 2, Mar - Apr 2023 pp 430-433 www.ijdmsrjournal.com ISSN: 2582-6018

Hall's report) to obturate an upper incisor, whereas Dr. Halsted blocked the inferior dental nerve in a medical student using 9 minims of the same solution.[14] Halsted and his colleague Hall went on to develop nerve and regional blocking techniques, although it was François Franck who coined the term in 1892.[15]

As the undesirable effects of cocaine (toxicity, addiction, and others) gradually became known, new anesthetic drugs were sought to replace it. None of these attempts were successful, however, until November 27, 1904, when German chemist Alfred Einhorn (1856– 1917)75 patented 18 para-aminobenzoic derivatives that had been developed in the Meister Lucius and Brüning plants at Höchst, in Hesse, Germany. His compound number two was to bring about a radical change in the existing scenario.[16] Its name, novocaine, appeared for the first time in 1905 in an article published by Professor Heinrich Braun, in which he compared it to other promising local anesthetics such as stovaine and alypine. This analog was much safer and caused fewer side effects than cocaine. It also did not have the addictive properties of cocaine. However, surgeons and dentists soon realized that it caused vasodilation and easily spread systemically. It was then combined with epinephrine to cause vasoconstriction, which allowed the medication to remain locally.

Braun compared three new compounds: alypin; amylocaine (Stovaine); and procaine (Novocaine). Pain on injection and tissue irritation led Braun to suggest that alypin should not undergo clinical trial, and the local hyperaemia that followed amylocaine might have had the same result, but Braun[17] accepted tBraun gave procaine only qualified approval, perhaps recognising that it does not meet requirements [18] and [19]. Then, in 1909, Le Brocq[20] published a more detailed study of a larger number of drugs (including alypin), concluding that procaine was the 'most satisfactory'. Definitively, he found that amylocaine produced tissue necrosis after subcutaneous injection, this leading to its decline in spite of Bier's recommendation and Barker's uneventful use of it in studies of spinal anaesthesia.[21] However, one of procaine's deficiencies is its short duration of action, and subsequent developments looked to overcome that. Both cinchocaine and tetracaine were synthesised in the late 1920s, [22, 23] but a longer duration of action is closely related to greater systemic toxicity, thus limiting the major use of both to spinal anaesthesia he opinion of August Bier, the pioneer of spinal anaesthesia, that it was the best drug for that indication.

In 1943–1946, Nils Löfgren and Bengt Lundquist developed a xylidine derivative they called lidocaine, whose chemical composition is very different from novocaine but which is nonetheless safe and has a stronger effect and scant allergenic action. [24] Soon thereafter, amide-type anesthetic drugs began to be developed. In 1957, Bo afEkenstamet al. [25] synthesized mepivacaine and bupivacaine.Amideanesthetics possess an aromatic head which is linked to ahydrocarbon chain by an amide bond rather than an ester. This results in amide anesthetics being more stable and hence less prone to causing allergic reactions as compared to ester. As a butyl group homologue of meivacaine, bupivacaine was initially discarded as it was found to be four times more toxic. The discovery of mepivacaine's optically active isomers, and the extensive study of their decreased toxicology, led to the selection and development of a pure S-(-) enantiomer Ropivacaine in 1996; and in 1972, Adams et al. [26] developed etidocaine. The first article published on articaine [27] also appeared in 1972.Levobupivacaine, the S-(-) enantiomer of bupivacaine, was approved by FDA in 1999.

II. DISCUSSION

Based on this review, which covers the past 10 years, the findings suggest that articaine has been researched the most and that it also has the highest efficacy of the amides used in dental local anesthesia. The fact that articaine received so much attention is probably attributable to the fact that before the year 2000, articaine was not available in the USA, whereas in Europe it was already marketed in 1976.

Although, it was not within the scope of this review paper, nevertheless, the authors are aware of the dubious reputation of articaine with regard to post-operative paresthesia and the discussion about it being manufactured as a 4% solution instead of 2% like lidocaine for dental local anesthesia. [28-30]
Ropivacaine (Naropin) is a new aminoamide local anesthetic. It is the monohydrate of the hydrochloride salt of 1- propyl-2', 6'-pipecoloxylidide and is prepared as the pure S-enantiomer. It is one of a group of local anesthetic drugs, the pipecoloxylidides, which were first synthesized in 1957. [31]Ropivacaine causes reversible inhibition of sodium ion influx, and thereby blocks impulse conduction in nerve fibers. [32] This action is potentiated by dose-dependent inhibition of potassium channels. Ropivacaine is less lipophilic than bupivacaine and is less likely to

penetrate large myelinated motor fibers; therefore, it has a selective action on pain transmitting Aδ and C nerves rather than Aβ fibers, which are involved in motor function. [33]

III. CONCLUSION

Local anesthesia has helped a lot in dentistry, Anxiety, fear & apprehension should be recognized & managed before administration of a local anesthetic. Vasoconstrictors should be included in all local anesthetics unless specifically contraindicated. Many advances in local anesthesia therapeutics and armamentarium have become available to the dental practitioner in recent years. Ropivacaine is an effective long-acting local anesthetic and the first produced as a pure enantiomer. The sensory block provided by ropivacaine is similar to that produced by an equivalent dose of bupivacaine. Ropivacainemay be suitable for time-consuming oral procedures and/or when prolonged postoperative analgesia is desirable.

REFERENCES

[1]. Greene NM: A consideration of factors in the discovery of anesthesia and their effects on its development. ANESTHESIOLOGY 1971; 35:515–22.
[2]. Wells H: A History of the Discovery of the Application of Nitrous Oxide Gas, Ether and Other Vapors to Surgical Operations. Hartford, J Gaylord Wells, 1847, pp 5–14.
[3]. Menczer LF, Jacobsohn PH: Dr Horace Wells: The discoverer of general anesthesia. J Oral MaxillofacSurg 1992; 50:506–9.
[4]. Jacobsohn PH: Dentistry's answer to "the humiliating spectacle": Dr. Wells and his discovery. J Am Dent Assoc 1994; 125:1576–81.
[5]. Greene NM: Anesthesia and the development of surgery (1846–1896). AnesthAnalg 1979; 58:5–12.
[6]. Loza-Balsa G: Monografiasobre la coca. La Paz, Bolivia, EditaSociedadGeográfica de la Paz, 1992, ppix:x, xiv, xv, 3.
[7]. Cadwell J, Sever PJ: The biochemical pharmacology of abused drugs: I.Anfetamines, cocaine, and LSD. Clin Pharm Ther 1974; 16:625–38
[8]. Van Dyke C, Byck R: Cocaine. Sci Am 1982; 246:128–41
[9]. Bülher A: Die Kokabei den Indianernsüdamerikas. Ciba Z 1944;8:3338–51
[10]. Guerra F: The Pre-Columbian Mind. London, Seminar Press, 1971, pp 47,52, 126, 191
[11]. Bühler A: Zurerforschung des Kokagenusses. Ciba Z 1944; 8:3353–9
[12]. Niemann A: Uebereineneueorganische Base in den Cocablättern. Arch Pharm 1860; 153:129–55, 291–308
[13]. Olch PD, William S: Halsted and local anesthesia: Contributions and complications. ANESTHESIOLOGY 1975; 42:479–86
[14]. Hall RJ: Hydrochlorate of cocaine. N Y Med J 1884; 40:643–4
[15]. Matas R: Local and regional anesthesia: A retrospect and prospect: II. Am J Surg 1934; 25:362–79
[16]. Farbwerkevorm: Meister Lucius und Brüning in Höchst A.M. VerfahrenzurDarstellung von p-Aminobenzoësäurealkaminestern. Patentschrift n° 179 627 (klasse 12q. Gruppe 6). November 27, 1904
[17]. Braun H. U¨ bereinigeneueo¨rtlicheAnaesthetica (Stovain, Alypin, Novocain). Deutsche MedizinischeWochenschrift 1905; 31:1667–1671.
[18]. Arnott J. On cold as a means of producing insensibility. Lancet 1848; 2: 98–99
[19]. Niemann A. Uebereineneueorganische Base in den Cocabla¨ttern. Viertel Jahresschriftfu¨ rpractische Pharmacie 1855; 9:489–524.
[20]. Le Brocq CN. Report on local anaesthetics recommended as substitutes for cocaine. BMJ 1909; 1:783–785.
[21]. Barker AE. A report on clinical experience with spinal analgesia in 100 cases. BMJ 1907; 1:665–674.
[22]. Miescher K. Studienu¨berLokalana¨ sthetica. Helvetica ChimicaActa 1932; 15:163–190.
[23]. Fussga¨nger R, Schaumann O. U¨ bereinneuesLokalana¨sthetikum der Novokainreihe (Pantokain). Naunyn-SchmiederbergsArchivfu¨rexperimentelle Pathologie und Pharmakologie 1931; 160:53–65.
[24]. Löfgren N, Lundquist B: Studies on local anaesthetics: II. SvenksKemTidskr 1946; 58:206–17
[25]. Ekenstam B, Egner B, Pettersson G: Local anaesthetics: I. N-alkyl pyrrolidine and N-alkyl piperidine carboxylic acid amides. ActaChemScand 1957;11:1183–90.

[26]. Adams HJ, Kronberg GH, Takman BH: Local anesthetic activity and acute toxicity of (_) 2-(N-Ethylpropylamino)-2', 6'-butyroxylidide, a new long-acting agent. J Pharm Sci 1972; 61:1829–31
[27]. Winther JE, Nathalang B: Effectivity of a new local analgesic Hoe 40 045. Scand J Dent Res 1972; 80:272–8.
[28]. Piccinni C, Gissi DB, Gabusi A, Montebugnoli L, Poluzzi E. Paraesthesia after local anaesthetics: An analysis of reports to the fda adverse event reporting system. Basic ClinPharmacolToxicol 2015; 117: 52-6.
[29]. Hillerup S, Jensen RH, Ersboll BK. Trigeminal nerve injury associated with injection of local anesthetics: Needle lesion or neurotoxicity? J Am Dent Assoc 2011; 142: 531-9.
[30]. Garisto GA, Gaffen AS, Lawrence HP, Tenenbaum HC, Haas DA. Occurrence of paresthesia after dental local anesthetic administration in the united states. J Am Dent Assoc 2010; 141: 836-44.
[31]. AfEkenstam B, Egner B, Petersson G. Local anaesthetics: 1. N-alkyl pyrrolidine and N-alkyl piperidine carboxylic amides. ActaChemicaScandinavica 1957; 11: 1183–1190.
[32]. Hansen TG. Ropivacaine: A pharmacological review. Expert Rev Neurother 2004;4:781-91.
[33]. Kindler CH, Paul M, Zou H, Liu C, Winegar BD, Gray AT, et al. Amide local anaesthetics potently inhibit the human tandem pore domain background K+ channel TASK-2 (KCNK5).