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A COMPARATIVE STUDY OF TWO LOCAL ANAESTHETIC AGENTS- BUPIVACAINE AND LIGNOCAINE IN DENTISTRY

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ABSTRACT

The contribution of local anaesthetics to dentistry is vast; as almost all the branches in the dental and medical field require them. They are available inmany forms in the current era painless treatment is the at most requirement and the local anesthetics have undergone various advances to provide this painless treatment. This article discusses about the role of two local anesthetic agents- lignocaine and bupivacaine in dentistry.

KEY WORDS: **Lignocaine, Bupivacaine, Third molar surgery, Dentistry, Treatment**

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INTRODUCTION

Pain is an unpleasant sensation which humans try to avoid distinctly. Management of pain was of utmost interest in the field of medicine from ancient times. Today there are potent local anesthetic solutions available, which make satisfactory pain control possible during surgical procedures. Lignocaine is one of the most popular local anesthetics. It was the first amide type of local anesthetic synthesized by Nils Lofgren in 1948[1]. It continues to be one of the most popular local anesthetics in current use. This drug when used in conjunction with epinephrine requires about four hours for the disappearance from sites of administration. Bupivacaine, a synthetic drug, first prepared by A.F.Ekenstam in 1957 is marketed as sensorcaine. It is a long acting, amide local anesthetic. It is used in an ideal dental concentration of 0.5%, but available in different concentrations. The duration of action is two to three times longer than that of Lignocaine. In contrast to currently available local anesthetics, a single injection of bupivacaine provides satisfactory anesthesia that lasts for several hours, and in addition there is a comparatively long period of analgesia that persists after the sensation has returned. Additionally not only these agents are more effective than centrally acting narcotic analgesics in this situation, but also the side effects of centrally acting agents are avoided.

Lignocaine

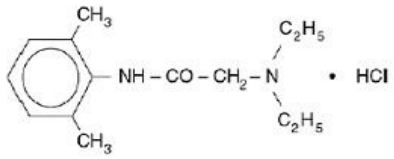

Figure 1
Lignocaine hydrochloride chemical Structure

Lignocaine diffuses readily through the interstitial tissues and into the lipid rich nerve fibres giving a rapid onset of anesthesia. It has a pka of 7.85, hence possess a rapid onset of action on the nerve membrane for production of conduction block, since approximately 35% of the substance exist in the base form at a tissue pH of 7.4. It has a rapid onset of action about two to three minutes. The duration of effectiveness is believed to be determined by the extent of binding to proteins which are immersed in lipids of the membrane. The greater the binding affinity to nerve proteins, the longer the anesthetic activity persists. In the amide series the plasma protein binding of lignocaine is about 65% to 75%.[2]

Bupivacaine

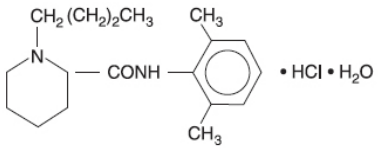

Figure 1
Bupivacaine hydrochloride chemical Structure

Bupivacaine has a Pka of 8.1, hence these substances exist primarily in the cationic form (80 -95%) at a tissue pH of 7.4 and hence is relatively slower in onset compared to Lignocaine [2]. It has an intermediate onset of action of about 2 to 5 minutes[3]. It is approximately four times more potent and four times less toxic as mepivacaine and Lignocaine. The plasma protein binding of bupivacaine is similar to etidocaine and is 90% protein bound.[2]

Application in dentistry

Lignocaine and bupivacaine have found its use in surface anesthesia, infiltration anesthesia, field block, nerve or conduction block. Lignocaine is used for regional and spinal anesthesia, where it is injected into the subarachnoid space, but bupivacaine is not used in regional anesthesia and spinal anesthesia because of excessive high plasma levels.[4] They are used in all branches of dentistry from extractions, minor oral surgical procedures, endodontics, periodontics either by conduction nerve block or infiltration anesthesia.

Recent advancement

Alkalinisation of lignocaine with sodium bicarbonate for nerve block during injection, is for rapid onset of anesthesia[5]. Modi,Rastogi et al (2009) [6] clearly indicates that buprenorphine added to the local anesthetic injected in performing various intraoral nerve blocks does provide prolonged postoperative analgesia and markedly decreases the need for pain medication in the early and late postoperative periods, at least up to 48 hours. Continuous administration of 60 mL of 0.5% bupivacaine HCl at 1 mL/ h with a pain pump and epidural catheter can be used as a transition treatment for patients with side effects from high-dose antiepileptic drugs and for patients awaiting neurosurgery or individuals who refuse cranial surgery.[7]

DISCUSSION

The action of local anaesthetics is dependent on their anaesthetic potency therefore highly lipid soluble local anaesthetics are very potent. The duration of action of local anaesthetics is directly proportional to protein binding properties. Bupivacaine is a local anaesthetic with a high potency and a long duration of action compared to Lignocaine which is of intermediate potency and duration of action. A study by Laskin et al (1977)[4] reported several advantages of Bupivacaine over Lignocaine. They showed that bupivacaine has a much greater potency; therefore a smaller concentration could be used with the same results. It has a lower toxicity at equally effective concentrations; therefore a larger dose can be used safely. For infiltration anesthesia where the nerve sheaths are not thick, the rates of onset of either Lignocaine or bupivacaine do not appear significantly different but studies have reported a longer time of onset of paresthesia for bupivacaine[8,9,10] . They have discussed a rapid rate for bupivacaine compared to Lignocaine, which was same with our study. It has been reported that there is a longer time of recovery of lip numbness with bupivacaine.[11, 8, 4, 3, 25] Bupivacaine is more than 2 times longer acting than Lignocaine with respect to duration of anesthesia.The most severe pain usually occurs in the first six to twelve hours. In view of this there appears to be a definite place for the use of long acting local anaesthetics. The studies have reported bupivacaine to be longer acting with respect to onset of post operative pain[8, 4, 12, 13, 14]. The free base form of the local anaesthetic agent is more lipid-soluble, and so diffuses quickly into the membrane of the nerve. The cytoplasm was acidified by the membrane-permeating carbon dioxide leading to the intracellular “trapping” of the cationic form of the local anaesthetic agent[15]. The margin of safety for nerve conduction and may have a direct action on the binding of the local anaesthetic to the sodium channel. The addition of sodium bicarbonate to solutions of lignocaine reduced the duration of onset of anesthesia[16, 17, 18], and was effective in reducing pain during the injection.[19] There was no sign of systemic toxicity noted after administration of the local anesthetic agents in previous literature. The serum levels of bupivacaine reached 0.44-1.5

mg/ ml after bolus dose of 20 ml of 0.25% bupivacaine[20]. This is far below the toxic level of 2-4 mg/ ml. Bupivacaine significantly reduced the postoperative pain experience only at the 8-hour period. No difference in analgesic requirements or cardiovascular responses was observed with the two local anesthetics. The mean blood concentrations of both agents were considerably lower than their respective toxic threshold concentrations.[21]. The study reported that there was no change in blood pressure with the administration of adrenaline-containing local anesthetics[22, 23, 24]. The decrease in heart rate between 15 and 30 minutes was statistically significant for both bupivacaine and Lignocaine. This reduction in heart rate appeared to be the result of the decrease in endogenous catecholamine associated with the termination of the surgical procedure.[21] Thus 0.5 % bupivacaine can be used safely and effectively in oral surgery especially in procedures of longer duration as it provides a much longer duration of action than 2% Lignocaine with 1:80,000 epinephrine as measured by recovery from lip numbness and onset of postoperative pain. In comparison with 0.5% bupivacaine, 4% articaine (both with 1:200,000 epinephrine) provided a shorter time of onset and comparable haemostasis and postoperative pain control with a shorter duration of soft tissue anesthesia in lower third molar removal[25]. Hence, the application of this anaesthetic agent to specific procedure is desirable than lignocaine. Bupivacaine was also used with opiods which increases the effect of analgesia by 3 times and has good use of minor oral surgical procedures.[6] Hence, the addition of buprenorphine to bupivacaine for intraoral nerve blocks in patients undergoing same-day surgery may be a way to provide postoperative analgesia for outpatients.

CONCLUSION

The review showed that, bupivacaine is found to have a longer time of anesthesia compared to Lignocaine. The literature indicates that the main difference between the two local anesthetics is the duration of anesthetic action which is longer with bupivacaine 0.5% as compared to Lignocaine 2% with 1:80,000 epinephrine and also the total period of painlessness was longer with bupivacaine than by Lignocaine. Though bupivacaine 0.5% has a longer duration of action than Lignocaine 2% with 1:80,0000 epinephrine , it is less commonly used in dentistry. Its slower onset of anesthesia is also found to be unfavorable by most operators, thus Lignocaine can be considered preferred local anesthetic in patients undergoing dental surgery.

REFERENCES

1. Nils Löfgren (1948). Studies on local anesthetics: Xylocaine: a new synthetic drug (Inaugural dissertation: Stockholm University). Stockholm, Sweden: Ivar Heggstroms
2. Covino, B.G and Giddon, D.B. (1981). Pharmacology of local anesthetic agents. J.Dent.Res. 60: 8:1454-59
3. Chapman P.J (1987). Post operative pain control for outpatient oral surgery. Int.J.Oral Maxillofac Surg. 16(3): 319-204
4. Laskin J.L, Wallace W.R, DeLeo B: (1977). Use of bupivacaine hydrochloride in oral surgery.A clinical study. J, oral surg. 35:25-29
5. Vinay Mohan Kashyap, Desai R, Reddy PB, Menon S (2011). Effect of alkalinisation of lignocaine for intraoral nerve block on pain during injection, and speed of onset of anaesthesia. British Journal of Oral and Maxillofacial Surgery 49 e72–e75
6. Mancy Modi,Sanjay Rastogi, Ashish Kumar,. (2009). Buprenorphine With Bupivacaine for Intraoral Nerve Blocks to Provide Postoperative Analgesia in Outpatients After Minor Oral Surgery. J Oral Maxillofac Surg 67:2571-2576.
7. Guhan Dergin, Gokhan Gocmen, B. Cem Sener, (2012). Treatment of trigeminal neuralgia with bupivacaine HCL using a

temporary epidural catheter and pain pump: Preliminary study. Journal of Cranio-Maxillo-Facial Surgery 40 124e128

8. Nespeca J.A (1976). Clinical trials with bupicaine in oral surg. Oral Surg.: 42:301-307
9. Pricco.D.F. (1977). An Evaluation of bupivacaine for regional nerve block in oral surgery. J.Oral Surg. 35:126-129
10. Maimon W.N and Schuller D.E, (1984). lidocaine vs bupvacaine in facial plaistic surgery. Arch.Otolaryngolo.110:525-528
11. Lund P.C , Cwik J.C, Vallesteros F (1970). Bupivacaine- A new long acting local anesthetic agent.A preliminary clinical and laboratory report: Anesth analg 49:1:103-114
12. Moore P.A and Dunsky J.L (1983). Bupivacaine anesthetia – A clinical trial for endodontic therapy. Oral Surg:176-179
13. Linden E.T, Abrams .H , Matheny J, Kaplan AL, Kopczyk RA, Jasper SJ Jr (1985). A Comparison of postoperative pain experience following periodontal surgery using two anesthetic agents. J.Periodontal.57:10:637-642
14. Danielsson K., Evers .H , Åke Nordenram (1986). Long acting local anesthetics in oral surgery. Int.J. Oral Maxillofac.Surg.15:119-126.
15. Masters J.E. (1998). Randomised control trial of pH buffered lignocaine withadrenaline in outpatient operations. Br J Plast Surg. 51:385–7
16. Wong K, Strichartz G.R, Raymond S.A. (1993). On the mechanisms of potentiation of local anesthetics by bicarbonate buffer: drug structure-activity studies on isolated peripheral nerve. Anesth Analg. 76: 131–43.
17. Gros O. Uber die narkotica und Lokalanasthetica. *Archiv fur experimentelle Pathologie und Pharmakologie* 1910; 63:80–106
18. Ritchie JM, Ritchie B, Greengard P. (1965). The effect of the nerve sheath on the action of local anesthetics. J Pharmacol Exp Ther 150:160–4.
19. Milner Q.J, Guard B.C, Allen J.G. (2000). Alkalinization of amide local anaesthetics by addition of 1% sodium bicarbonate solution. Eur J Anaesthesiol; 17:38–42.
20. Symreng T, Gomez MN, Johnson B, Rossi NP, Chiang CK (1989). Intrapleural bupivacaine: technical considerations and intraoperative use. J Cardiothorac Anesth 3: 139e141,.
21. Gary E: Bouloux, and Arumugam Punnia-Moorthy, (1999). Bupivacaine Versus Lidocaine for Third Molar Surgery: A Double-Blind, Randomized, Crossover Study. J Oral Maxillofac Surg 57:510-514
22. Meecham J, Thomson CW, Blair GS, Rawlins MD: (1991).The biochemical and haemodynamic effects of adrenaline in lignocaine local anaesthetic solutions in patients having third molar surgery under general anaesthesia. Br J Oral Maxillofac Surg 29:263,
23. Salonen M, Forsell H, Scheinin M (1988). Local anaesthesia with lidocaine and adrenaline: Effects on plasma catecholamines, heart rate and blood pressure. Int J Oral Maxillofac Surg. 17(6):392-4.
24. Vernale C (1960). Cardiovascular responses to local dental anaesthesia with epinephrine in normotensive and hypotensive subjects. Oral Surg Oral Med Oral Path.13:942,
25. Leonardo V. L. Gregorio, a Fernando P. M. (2008). A comparison of the clinical anesthetic efficacy of 4% articaine and 0.5% bupivacaine (both with 1:200,000 epinephrine) for lower third molar removal. Oral Surg Oral Med Oral Pathol Oral Radiol Endod;106:19-28