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Comparison of cardiovascular responses after injection of lidocaine with either clonidine or adrenaline: a two-year comparative analysis

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Abstract

Our aim was to evaluate the efficacy of clonidine with lidocaine as a local anaesthetic agent for inferior alveolar mandibular nerve blocks for dental extraction. We studied 200 patients who required extraction of mandibular teeth and divided them into two groups of 100 each, the first of which was given lidocaine and adrenaline (12.5 μg/ml) and the second lidocaine and clonidine (15 μg/ml). Cardiovascular vascular variables (blood pressure, heart rate, and mean arterial pressure) were assessed before, during, and after extraction, and postoperative pain was measured on a visual analogue scale. There was a significant reduction in systolic blood pressure ($p = 0.0001$) and heart rate ($p = 0.000$) after injection of clonidine. However, they both increased after injections of lidocaine plus adrenaline, and there was a significant reduction in pain at four hours postoperatively with clonidine ($p = 0.000$). Our results showed that anaesthesia with lidocaine and clonidine decreases systolic blood pressure and heart rate 10 minutes after injection for extraction of lower mandibular teeth. We suggest that patients who have local anaesthetic with lidocaine and clonidine are at minimal cardiovascular risk and there is no difference in the onset of anaesthesia.

Keywords: lidocaine; adrenaline; clonidine; inferior alveloar nerve block; extraction; α-2 receptor

Introduction

Lidocaine hydrochloride is the most widely used anaesthetic in dentistry. It is a potent vasodilator, to which adrenaline bitartrate is added to counter vasodilatation, help to achieve haemostasis, and prolong the duration of anaesthesia. Adrenaline is also widely used in dental anaesthetic solutions to produce localised vasoconstriction and to increase the duration of action of the local anaesthetics.

Concentrations ranging from 1:50,000 (20 μg/ml) to 1:200,000 (5 μg/ml) are available in commercially produced dental cartridges.[1] It has been hypothesised that the amount of lidocaine absorbed after local dental anaesthesia is less than the amount produced endogenously in response to pain that results from inadequate anaesthesia or anxiety associated with the procedure.[2] However, there are limitations to the use of solutions that contain adrenaline, particularly in patients with compromised cardiovascular function, hyperthyroidism, or thyroid storm. Other viable options have therefore been sought to retain its vascular effects while simultaneously providing haemodynamic stability, and it has been suggested that lidocaine combined with clonidine could

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Table 1
Inclusion and exclusion criteria.

Inclusion criteria	Exclusion criteria
1. All patients between 18–45 years of age	1. Pregnant women,
2. Normal healthy patients or patients with no coexisting conditions such as hypertension, diabetes mellitus, asthma, or bleeding disorders	2. Known hypertension, children, and patients with localised infection in the oral cavity
3. Patients with no known allergy to either drug	3. Nursing mothers
4. Presence of dental caries with apical periodontitis, periapical abscess, or chronic pulpitis, which could not be restored	4. Apprehensive patients
5. Patients who refused conservative management	5. Patients with impacted or submerged teeth
6. Simple, uncomplicated extractions	6. Teeth after root canal treatment
	7. Patients who were taking opioids 48 hours preoperatively
	8. Patients known to be sensitive or allergic to either drug
	9. Patients with infection in the orofacial area or who had had dental treatment the previous day
	10. Previous history of dental extraction

be a useful and safe alternative to lidocaine with adrenaline for intraoral anaesthesia.

Clonidine is a α2-adrenoceptor agonist that is being increasingly used together with local anaesthetics for spinal or epidural analgesia. It also produces a minor degree of nerve conduction blockade (C-fibres) at high concentrations, and sympatholysis and reduced blood pressure by its actions at the periphery, brainstem, and spinal cord, which is opposed by direct vasoconstriction from α2-adrenergic agonists in the periphery. Clonidine does not produce respiratory depression as opioids do, but there have been a few sporadic reports.[3]

Blood pressure and heart rate should be monitored for at least two hours after a bolus injection of clonidine, and pulse oximetry should be considered if large boluses (>300 μg) are given.[4] Epidural clonidine is an approved analgesic. However, α2-agonists have been given by various routes for long-term and short-term perioperative pain control.[5]

We designed the present study to compare the cardiovascular response (blood pressure and heart rate) after injection of clonidine with lidocaine, and adrenaline with lidocaine, before, during, and after the extraction of mandibular teeth. We also compared the intensity of pain after the injections using a visual analogue scale (VAS) four hours postoperatively.

Patients and Methods

We designed a prospective, randomised, controlled, parallel-group study with the approval of the Department of Oral and Maxillofacial Surgery over the period of two years from September 2013-September 2015. The study protocol was approved by the Institutional Ethics Committee, and followed the Helsinki guidelines. The procedures were explained to the patients verbally and in writing, and informed consent was taken before enrolment. The inclusion and exclusion criteria are shown in Table 1. Patients were allocated into two groups of 100 patients each using the slot method of randomisation. One group was given 2% lidocaine with 1/80 000 adrenaline (12.5 μg/ml) and the second group 2% lidocaine with clonidine (15 μg/ml). The latter was freshly prepared during the procedure as the shelf-life of clonidine is 6-8 hours. To prepare the lidocaine plus clonidine (15 μg/ml), 2% lidocaine 9 ml was mixed with clonidine 150 μg/ml x 1 ml in a 10 ml syringe, and the solution was transferred to a 2 ml syringe so that each mm of solution contained clonidine 15 μg/ml.

Extraction was done under aseptic conditions. Local anaesthetic 2 ml was used in both groups and patients who required more than 2 ml were withdrawn from the study. Haemodynamic variables and possible cardiovascular side effects induced by clonidine and adrenaline were continuously monitored by electrocardiography. Systolic and diastolic blood pressure, arterial pressure, and heart rate were recorded before (preoperative baseline values) and during the injection of local anaesthesia, five minutes after injection of the anaesthetic, during the operation, and 10 minutes after extraction. All extractions were done by the same surgeon. The onset of anaesthesia was calculated by a pinprick test, and the intensity of anaesthesia during extraction by a VAS after the completion of the treatment. Patients were shown a 10 cm VAS and asked to indicate no pain, just noticeable, weak, moderate, severe, or excruciating pain. All patients were given diclofenac sodium 50 mg postoperatively, with instructions to take it only after four hours. Patients were followed up by telephone for the first four hours postoperatively to assess the immediate analgesic potency of clonidine.

Statistical analysis

The data were analysed using SPPS (version 11.5, SPSS Inc, Chicago, IL, USA). An unpaired Student's *t* test was used to compare the significance of differences between the means of the two groups, and probabilities of less than 0.05 were accepted as significant.

Results

We studied 200 patients of both sexes, mean (range) age 31 (18-45) years who required uncomplicated extractions. Fig. 1

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