Hypotension After Local Anesthetic Infiltration Into The Oral Submucosa During Oral And Maxillofacial Surgery

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Citation

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Abstract

We have noted hypotensive episodes in close association with submucosal infiltration of adrenaline contained in lidocaine. We investigated the effects of adrenaline contained in lidocaine on hemodynamics during oral and maxillofacial surgery under general anesthesia. Sixteen patients with an American Society of Anesthesiologists physical status of I scheduled to undergo sagittal splitting ramus osteotomy were included in this study. The patients comprised 6 men and 10 women with a mean age of 22.3 years and mean weight of 58.5 kg. We measured the patients' systolic arterial pressure (SAP), mean arterial pressure (MAP), diastolic arterial pressure (DAP), and pulse rate (PR). The patients exhibited a mean 30% decrease in SAP, mean 29% decrease in MAP, mean 33% decrease in DAP, and mean 13% increase in PR. All data were normalized to the values immediately before infiltration and expressed as a relative percentage. The average time from injection to minimum pressure was 88 seconds, and the mean duration of the trough was about 60 seconds. This study suggests that there is an approximately 90-second lag time in hemodynamic changes after local anesthetic injection into the oral submucosa. Local anesthetic infiltration with adrenaline contained in lidocaine elicits temporary but severe hypotension.

INTRODUCTION

Adrenaline contained in lidocaine is widely used in neurosurgery, otorhinolaryngological procedures, dental treatment, and oral and maxillofacial surgery to decrease surgical bleeding [1-3], lessen mucosal congestion, and maintain a clear field of view [4, 5]. In neurosurgery and otorhinolaryngological procedures, local infiltration with adrenaline-containing lidocaine solution elicits temporary but severe hypotension [2, 3, 5]. However, hemodynamic changes has not been known after local anesthetic injection into the oral submucosa for dentists. We here report hypotensive episodes in close association with submucosal infiltration of local anesthetics with adrenaline during general anesthesia.

METHODS

This observational study was approved by the Committee on Clinical Investigation for Human Research (IRB) at Iwate Medical University. Written informed consent was obtained from all patients.

We studied 16 patients with an American Society of Anesthesiologists physical status of I who were scheduled to undergo sagittal splitting ramus osteotomy. The patients comprised 6 men and 10 women with a mean age of 22 years and mean weight of 58.5 kg (Table 1). And we could measure 32 cases since the surgeon infiltrated the tissue around both the right and left side of the ascending ramus with a 1 hour time difference (right side: 16 cases, left side 16 cases). All patients underwent intravenous administration of atropine (0.05 mg/kg) and midazolam (0.5 mg/kg) 30 minutes before admittance to the operating room. Anesthesia was induced with a mixture of either thiopental (5 mg/kg) or propofol (2 mg/kg) with fentanyl (50-100 μg) and vecuronium bromide (0.1 mg/kg), then maintained with sevoflurane (1.0%–1.5%) in oxygen (40%). Remifentanyl (0.1-0.2 μg/kg/min) were administered after endotracheal intubation. A catheter was inserted into a dorsal artery of the foot after anesthetic induction to monitor the arterial blood pressure (ABP). The patient's hemodynamics and respiration were stable at this time. First, the surgeon infiltrated the tissue around the right side of the ascending ramus using 1% lidocaine (1.5 ± 0.4 mg) in combination with 1/100.000 adrenaline (1.5 ± 0.4 μg). Second, the surgeon infiltrated the tissue around the left side of the ascending ramus using same local anesthetic at almost 60 min after infiltrated the tissue

around right side of the ascending ramus. The local infiltration was applied at three to five points on the oral mucosal incision, and the infiltration time was controlled at 20 to 30 seconds using the same needle gauge. We measured the ABP via catheter, the systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), pulse rate (PR), and blood oxygen saturation (SpO2) in all patients. The SAP, DAP, MAP, PR and SpO2 were continuously monitored with life scope 8 (Nihon Kohden, Tokyo, Japan) and recorded using a PowerLab 4/25T data acquisition system (ADInstruments, Bella Vista, Australia). Each parameter was measured at immediately before injection, reaching minimum arterial blood pressure, 1min after injection, 3min after injection and 5min after injection.

Values are presented as mean ± standard deviation. Intragroup comparisons were made using one-way analysis of variance for repeated measurements followed by Dunnett's test for multiple comparisons. Differences were considered statistically significant at a P value of <0.05.

RESULTS

As shown in Figure 1, the changes in arterial blood pressure were recorded using the PowerLab data system. The change in arterial blood pressure exhibited a delay of 43 seconds and then rapidly decreased; the duration of the trough was about 60 seconds.

There were significant differences between value of the SAP, DAP, MAP, and PR immediately before injection (control) and that of at 90 seconds after injection. There was a 30% decrease in SAP (mean, 33.7 mmHg), 29% decrease in MAP (mean, 20.9 mmHg), 33% decrease in DAP (mean, 18.7 mmHg), and 13% increase in PR (mean, 9.1 bpm). All data were normalized to the values immediately before injection and expressed as a relative percentage (Fig. 2). SpO2 was stable at 99-100%.

The average time from infiltration of the local anesthetic to reaching minimum pressure was 88 seconds (88 ± 46 s) and the mean duration of the trough was about 60 seconds (60 ± 8 s) These results suggest that there was an approximately 90-second lag time in hemodynamic changes after local anesthetic injection into the oral submucosa. Local anesthetic infiltration with adrenaline contained in lidocaine solution elicited temporary but severe hypotension.

DISCUSSION

In this study, we found out two important clinical issues. Hypotensive episodes may be in close association with submucosal infiltration of local anesthetics with adrenaline during general anesthesia. The hypotension is temporary but severe.

First, hypotensive episodes may be in close association with submucosal infiltration of local anesthetics with adrenaline during general anesthesia. The hypotension occasionally occur after infiltration local anesthetic with adrenaline into oral submucosa has not well known for dentists. Hemodynamic changes including hypotension were caused by the absorption of adrenaline contained in lidocaine solution [5, 6]. The hemodynamic effects of adrenaline are dose-dependent, and different doses of adrenaline may activate different types of sympathetic receptors. An infusion rate of 1 to 2 g/min, although rarely used, should predominantly activate 2-receptors with resultant vascular and bronchial smooth muscle relaxation. A rate of 2 to 10 g/min should predominantly activate 1-receptors to increase the heart rate, contractility, and conduction and decrease the refractory period. Doses of >10 g/min cause marked -stimulation with generalized vasoconstriction [5, 7]. The major mechanism of the occurrence of hypotension is presumed to be activation of 2-receptors [3, 5, 7]. A decrease in MAP with an increase in heart rate was observed at 1.0, 1.5, and 2.0 minutes when 1% lidocaine with 40, 80, and 160 g of adrenaline was administered; additionally, the average increase to the highest heart rate was approximately 10 bpm after the beginning of local infiltration [5]. The cause of this increase of HR mainly involves a baroreceptor reflex that decreases the blood pressure and gently stimulates 2-receptors [5, 8, 9].

Second, the hypotension is temporary but severe. In this study, there was a 30% decrease in SAP, 29% decrease in MAP, 33% decrease in DAP and the mean duration of the through was about 60 seconds. The temporary but severe hypotension was observed within almost 1 min, and have never been continuous in all case. Therefore, when we observe the severe hypotension after local anesthetic infiltration into the oral submucosa, we may avoid to administer intravenously atropine sulfate or an adrenergic agonist, ephedrine hydrochloride et al., in haste.

In neurosurgery and otorhinolaryngological procedures, local infiltration with adrenaline-containing lidocaine solution elicits temporary but severe hypotension. When 1% lidocaine with 40, 80, and 160 g of adrenaline was administered to the scalp in neurosugery, the MAP

temporally decreased by >30%, the average time from the start of local infiltration to the lowest MAP was 102 seconds, and the hemodynamic changes in blood pressure and PR tended to become relatively stable by 5 minutes [5]. Neither the MAP nor the PR changed significantly when 1% lidocaine without adrenaline was administered [5]. In functional endoscopic sinus surgery, a low dosage of adrenaline may produce significant hemodynamic effects, particularly obvious hypotension combined with a slight increase in heart rate [5, 7].

Limitation in this study, we did not measure the hemodynamic changes when lidocaine without adrenaline was infiltrated into the oral submucosa. Though we did not demonstrate directly that hypotensive episodes in association with submucosal infiltration of local anesthetics with adrenaline, we emphasize that local infiltration with adrenaline-containing lidocaine solution elicits temporary but severe hypotension within almost 90s after infiltrated into oral submucosa.

In conclusion, hypotensive episodes may be in close association with submucosal infiltration of local anesthetics with adrenaline during general anesthesia and the hypotension was temporary but severe. Clinicians must consider the possibility of marked hemodynamic changes when adrenaline contained in lidocaine solution is infiltrated into the oral submucosa. We carefully observe without any treatment because the changes only last approximately 1 minute or so.

Table 1

Demographic data : Data are presented as mean ± standard deviation unless otherwise indicated.

Patients, n	16
(Case; both right and left side, n)	(32)
Age, years	22 ± 4
Weight, kg	58.5 ± 8.8
Sex, male:female	6:10
Lidocaine dose, mg/kg	Right, 1.5 ± 0.4; left, 1.5 ± 0.4
Adrenaline dose, μg/kg	Right, 1.5 ± 0.4; left, 1.5 ± 0.4
Type of surgery	Sagittal splitting ramus osteotomy

Figure 1

Changes in arterial blood pressure. The arterial blood pressure changes with a delay of 43 seconds, and the duration of the trough is about 60 seconds.

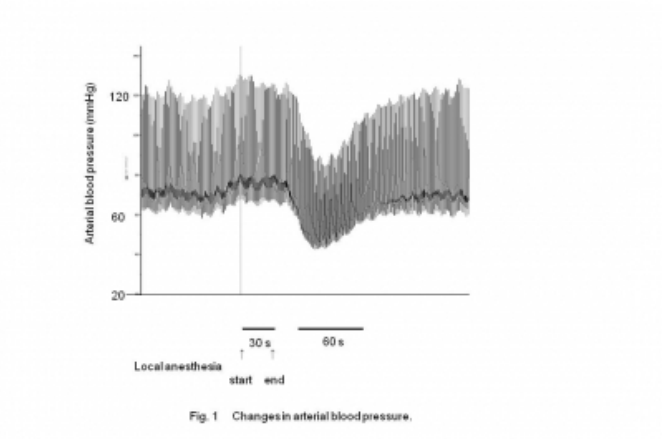

Fig. 1 Changes in arterial blood pressure.

Figure 2

Changes in arterial blood pressure and pulse rate. The values on the ordinate are as follows: control; immediately before injection, 30 seconds after injection, 60 seconds after injection, 90 seconds after injection, 120 seconds, 180 seconds and 300 seconds. Then the systolic, diastolic, and mean arterial pressure decrease to their minimum values, the pulse rate increases. There are significant differences in these parameters between before and 90 seconds after injection.

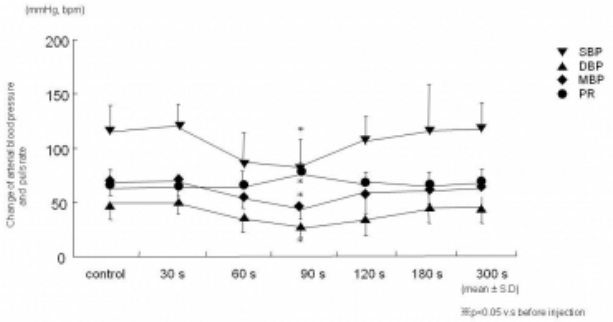

Fig. 2 Changes in arterial blood pressure and pulse rate.

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