Changes of cardiac output measured with Vigileo-flo Trac devise after local anesthetic infiltration into the oral mucosa

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A B S T R A C T

Background: Though we observed hypotensive episodes during infiltration of local anesthetic into oral mucosa, this hemodynamic changes has not been known for dentists.

Aims: We investigated the effects of adrenaline-induced hypotension on hemodynamics to measure changes in cardiac output with Vigileo-flo Trac devise when local anesthetic was infiltrated into the oral submucosa.

Materials and Methods: Ten patients (three men, seven women; mean age, 22 years; mean weight, 56.4 kg with an American Society of Anesthesiologists physical status of I who were scheduled for sagittal split ramus osteotomy were included in the present study. We measured systolic arterial pressure, diastolic arterial pressure, mean arterial pressure, pulse rate, blood oxygen saturation, Cardiac output, Cardiac index, Stroke volume, Stroke volume index and Stroke volume variation.

Results: At arterial blood pressure reached its minimum, SAP decreased by mean 25.3 mmHg, MAP decreased by mean 16.8 mmHg, DAP decreased by mean 16.8 mmHg and puls rate increased by mean 8 beat/min. There was a slight increase in cardiac output, cardiac index, stroke volume, stroke volume index and stroke volume variation but not a significant difference in five parameters.

Conclusion: It was thought that hypotension induced by adrenaline may not affect the cardiac output. However, it is prudent to consider the possibility of hemodynamic changes when infiltrating the oral submucosa with lidocaine containing adrenaline.
Introduction

Adrenaline contained in lidocaine is widely used in neurosurgery, otorhinolaryngological procedures, dental treatment, and oral and maxillofacial surgery to decrease surgical bleeding, lessen mucosal congestion, and maintain a clear field of view\textsuperscript{1-3}. However, adrenaline has several side effects, including hypertension, hypotension, tachycardia, bradycardia and arrhythmia. In oral and maxillofacial surgery and dental treatment, adrenaline contained in lidocaine induces marked hemodynamic changes, including decreased blood pressure during general anesthesia\textsuperscript{1,2,4,5}. We observed hypotensive episodes during infiltration of local anesthetic into oral mucosa during sagittal split ramus osteotomy. But these hemodynamic changes has not been known after local anesthetic injection into the oral submucosa for dentists, therefore, we wanted to establish whether this hypotension influenced hemodynamics.

In this study, we investigated the effects of adrenaline-induced hypotension on hemodynamics to measure changes in cardiac output with Vigileo-flo Trac devise when local anesthetic was infiltrated into the oral submucosa.

Material and Method

This observational study was approved by the Committee on Clinical Investigation for Human Research at Iwate Medical University.

Ten patients (three men, seven women; mean age, 22 years; mean weight, 56.4 kg with an American Society of Anesthesiologists physical status of I who were scheduled for sagittal split ramus osteotomy were included in the present study (Table 1). All patients received intravenous administration of atropine (0.05 mg/kg) and midazolam (0.5 mg/kg) 30 min before transfer to the operating room. Anesthesia was induced with propofol (2 mg/kg) with fentanyl and rocuronium bromide (0.08 mg/kg), and was maintained with sevoflurane (1.0–1.5%) in oxygen (40%). Fentanyl and remifentanil were administered after endotracheal intubation. A catheter was inserted into a radial artery after anesthetic induction. Arterial cannulation was performed to monitor arterial blood pressure (ABP). The patient’s hemodynamics and respiration were confirmed to be stable. The surgeon infiltrated the oral mucosa around the right or left ramus with 1% lidocaine (1.6 ± 0.4 mg/kg) combined with 1/100,000 adrenaline (1.6 ± 0.4 μg/kg). Local infiltration was performed at three or four points along the oral mucosa incision, and the infiltration time was controlled at 30 to 40 seconds with the same needle gauge.

We measured systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), pulse rate (PR), and blood oxygen saturation (SpO\textsubscript{2}) with a Life Scope 8® (Nihon Kohden, Tokyo, Japan). We monitored changes in Cardiac output (CO), Cardiac index (CI), Stroke volume (SV), Stroke volume index (SVI) and Stroke volume variation (SVV) with Vigileo-flo Trac devise (Edwards Lifesciences Corporation, Irvine, California, USA). Vigileo-flo Trac devise was connected to a Life Scope 8® (Nihon Kohden, Tokyo, Japan). The Vigileo-flo Trac devise measured changes in parameters at every 20 seconds for 5 minutes. All parameters were continuously recorded with a PowerLab 4/25T data acquisition system (ADInstruments, Bella Vista, Australia). The value for each parameter immediately before infiltration (control) was compared with the value at arterial blood pressure reached its minimum, 1 min after arterial blood pressure reached its minimum and 3 min after arterial blood pressure reached its minimum.
Values are presented as mean ± standard deviation (S.D). Intragroup comparisons were made with one-way analysis of variance for repeated measurements followed by Dunnett’s test for multiple comparisons. Differences were considered statistically significant at \( P < 0.05 \).

**Results**

At minimum ABP, SAP decreased by 26.8% (mean, 25.3 mmHg), MAP decreased by 28.7% (mean, 16.8 mmHg), DAP decreased by 29.4% (mean, 16.8 mmHg). Data were normalized to the values before infiltration and expressed as a relative percentage. There were significant differences between the values of SAP, MAP and DAP before infiltration versus those when ABP reached its minimum (Table 2). The average time from local anesthetic infiltration to minimum ABP was 98.9 ± 5.0 s and the mean duration of the trough was approximately 1 min, suggesting that there was a lag time of approximately 100 s for changes in hemodynamics after infiltration of local anesthetic into the oral submucosa.

The changes in parameters with Vigileo Flo-trac device were shown in Table 2. There was a 5.2 ± 0.4 L/min increase in CO arterial blood pressure reached its minimum, followed by a decrease to 4.9 ± 0.4 L/min. There was a 2.8 ± 0.2 L/min/m² increase in CI arterial blood pressure reached its minimum, followed by a decrease to 2.6 ± 0.2 L/min/m². There was a 75.1 ± 6.3 ml/beat increase in SV arterial blood pressure reached its minimum, followed by a decrease to 71.2 ± 5.3 ml/beat. There was a 40.3 ± 3.4 ml/beat/m² increase in SVI arterial blood pressure reached its minimum, followed by a decrease to 38.3 ± 2.7 ml/beat/m². There was a 12.2 ± 2.5% increase in SVV arterial blood pressure reached its minimum, followed by a decrease to 8.5 ± 0.8%. There was not a significant difference in five parameters with Vigileo-flo Track device.

**Discussion**

Blood pressure changes, such as severe hypertension or hypotension, affect blood circulation. Local infiltration of the oral submucosa with lidocaine containing adrenaline causes temporary but moderate or severe hypotension. Studies have found that infiltration of lidocaine containing adrenaline into the scalp before craniotomy, into the nasal mucosa for functional endoscopic sinus surgery, and into the oral mucosa for oral and maxillofacial surgery and dental treatments induces marked hemodynamic changes, including decreased blood pressure during general anesthesia \(^1\), \(^2\), \(^4\)-\(^6\). The hemodynamic effects of adrenaline are dose-dependent and different dose adrenaline may active different types of sympathetic receptors. “A rate of 1 to 2 μg / min, through rarely used, should predominantly activate \( \beta_2 \)-receptors with resulting vascular and bronchial smooth muscle relaxation. A rate of 2 to 10 μg / min should predominantly activate \( \beta_1 \)-receptors to increase heart rate, contractility, and conduction and decrease the refractory period. Dose in excess of 10 μg / min cause marked \( \alpha \)-stimulation with generalized vasoconstriction”\(^7\), \(^8\). And the major mechanism for the occurrence of the hypotension was presumed activation of \( \beta_2 \)-receptors\(^4\), \(^7\), \(^8\). We thought that the absorption of adrenaline is different, the blood levels of adrenaline are low which mainly excite \( \beta_2 \)-receptors and \( \beta_2 \)-receptor-induced vasodilation in muscle beds would occur suddenly\(^7\)-\(^9\).

We evaluated changes in the hemodynamics by using Vigileo-flo Trac to measure CO, CI, SV, SVI and SVV. We found that there was a slight increase in CO, CI, SV, SVI and SVV and not a significant
difference in five parameters. Generally, when hypotension occurs, CO increases with the increase in heart rate as compensatory action. To compensate for this vasodilation, the cardiovascular system adjusts by increasing cardiac output. Cardiac output is raised by increasing both the heart rate and the stroke volume. It is known that the increase in the stroke volume is achieved by better emptying of the blood from the left ventricle by augmentation of the ejection fraction. This increased ejection results from an increase in the strength and velocity of ventricular contraction from increased adrenergic activity to the heart and a decrease in peripheral resistance from the vasodilation.

SVV has been developed for new algorithm by pulse contour analysis from standard peripheral (typically radial) artery line. SVV is shown to be a reliable predict of fluid responsiveness. The baseline SVV was correlated to the fluid responsiveness, as changes in cardiac output or stroke volume, and was able to predict fluid responsiveness across a wide spectrum of clinical settings. SVV can be employed as a reliable predictor of fluid responsiveness in patients with controlled mechanical ventilation. At arterial blood pressure reached its minimum we did not administer the fluid bolus, it was thought that SVV slightly increased since the stroke volume increased as compensatory action. In this study, changes in CO, CI, SV, SVI and SVV increased slightly within normal rage and there were not significant differences in five parameters. It was thought that hypotension induced by adrenaline may not affect the CO.

Conclusion

We evaluated changes in the hemodynamics by using Vigileo-flo Trac to measure CO, CI, SV, SVI and SVV when local infiltration of lidocaine containing adrenaline into the oral submucosa causes temporary but moderate or severe hypotension. There was a slight increase in CO, CI, SV, SVI and SVV but not a significant difference in five parameters. However, it is prudent to consider the possibility of hemodynamic changes when infiltrating the oral submucosa with lidocaine containing adrenaline.

Acknowledgements

None.

Conflict of interest

None.

Funding source

None.

References


Table 1. Demographic data

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<thead>
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<th>Number of patients</th>
<th>10</th>
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<td>(Case; both right and left side, n)</td>
<td>(20)</td>
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<tr>
<td>Age (yr)</td>
<td>22±5</td>
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<tr>
<td>Weight (kg)</td>
<td>56.4±3.8</td>
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<tr>
<td>Sex (M:F)</td>
<td>3:7</td>
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<tr>
<td>Dose of lidocaine (mg/kg)</td>
<td>1.6±0.4</td>
</tr>
<tr>
<td>Dose of adrenaline (ug/kg)</td>
<td>1.6±0.4</td>
</tr>
<tr>
<td>Type of surgery</td>
<td>Sagital Splitting Ramus Osteotomy</td>
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</table>

Data are presented as mean±S.D.

Table 2. Hemodynamic variables

<table>
<thead>
<tr>
<th></th>
<th>Immediately before injection</th>
<th>Blood pressure reached its minimum</th>
<th>1 min after blood pressure reached its minimum</th>
<th>3 min after blood pressure reached its minimum</th>
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<tr>
<td>Maximum arterial blood pressure (mmHg)</td>
<td>90.9±2.7</td>
<td>65.6±6.2*</td>
<td>84.8±5.6</td>
<td>89.2±5.6</td>
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<td>Mean arterial blood pressure (mmHg)</td>
<td>59.4±2.6</td>
<td>42.6±3.8*</td>
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<td>55.0±3.2</td>
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<tr>
<td>Minimum arterial blood pressure (mmHg)</td>
<td>47.0±3.0</td>
<td>30.2±3.3*</td>
<td>40.2±3.6</td>
<td>41.4±3.0</td>
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<td>Pulse rate (beat /min)</td>
<td>58.1±5.0</td>
<td>68.2±5.2</td>
<td>61.6±3.1</td>
<td>60.1±4.8</td>
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<td>Cardiac output (l/min)</td>
<td>4.2±0.3</td>
<td>5.2±0.4</td>
<td>5.1±0.4</td>
<td>4.9±0.4</td>
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<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.2±0.2</td>
<td>2.8±0.2</td>
<td>2.7±0.2</td>
<td>2.6±0.2</td>
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<tr>
<td>Stroke volume (ml)</td>
<td>64.8±4.8</td>
<td>75.1±6.3</td>
<td>74.1±5.4</td>
<td>71.2±5.3</td>
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<tr>
<td>Stroke volume index (ml/m²)</td>
<td>36.2±2.5</td>
<td>40.3±3.4</td>
<td>39.8±2.9</td>
<td>38.3±2.7</td>
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<td>Stroke volume Variation (%)</td>
<td>9.3±0.7</td>
<td>12.2±2.5</td>
<td>8.7±0.9</td>
<td>8.5±0.8</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD.
* < 0.05 vs Immediately before injection