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Hemodynamic changes after infiltration of local anesthetic into the neck skin in radical neck dissection

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ABSTRACT

Though adrenaline combined with lidocaine is widely used for dental treatment and oral and maxillofacial surgery, marked hemodynamic changes occurred after infiltration of local anesthetic into oral mucosa or other regional portion. We observed hypotensive episodes during infiltration of local anesthetic into the neck skin during radical neck dissection. We wanted to establish whether this hypotension influenced cerebral perfusion. We investigated the effects of adrenaline combined with lidocaine on hemodynamics and brain blood circulation after local anesthetic infiltration. Sixteen patients were scheduled to undergo radical neck dissection. We measured systolic arterial pressure, mean arterial pressure, diastolic arterial pressure, pulse rate and the concentrations of cerebral oxyhemoglobin, deoxyhemoglobin, total hemoglobin and cytochrome oxidase. There was a mean 21.4% decrease in systolic arterial pressure, a mean 22.6% decrease in mean arterial pressure, a mean 22.8% decrease in diastolic arterial pressure, and a mean 4.8% increase in pulse rate. The average time from infiltration to minimum pressure was 132 s and the mean duration of the trough was ~1 min. There was a mean decrease of 1.5±1.3 nmol/L in oxy-hemoglobin, mean decrease of 0.7±0.4 nmol/L in deoxy-hemoglobin, mean decrease of 1.0±1.2 nmol/L in total-hemoglobin, and mean increase of 0.3±0.6 nmol/L in cytochrome oxidase. Local infiltration into the skin of the neck with adrenaline and lidocaine elicits temporary but moderate hypotension, which may have an effect on intracerebral oxygen environment.

KEYWORDS : Adrenaline, hypotension, neck skin, radical neck dissection

INTRODUCTION

Vasoconstrictor administered concomitantly with local anesthetic decreases blood loss, maintains clear visualization, slows the rate of absorption, reduces toxicity, and prolongs duration [1-3]. Adrenaline combined with lidocaine is widely used for dental treatment and oral and maxillofacial surgery. However, adrenaline has several side effects, including hypertension, hypotension, tachycardia, bradycardia and arrhythmia. Adrenaline administered during lidocaine infiltration of the scalp before craniotomy [3-5], in the nasal mucosa for functional endoscopic sinus surgery [6, 7], and oral mucosa for oral and maxillofacial surgery and dental treatment [8], induces marked hemodynamic changes, including decreased blood pressure during general anesthesia [1, 2, 8]. We observed hypotensive episodes during infiltration of local anesthetic into the neck skin during radical neck dissection. We wanted to establish whether this hypotension influenced cerebral perfusion.

Blood pressure changes, such as severe hypertension or hypotension, affect brain oxygen metabolism and blood circulation [10, 11]. Near-infrared spectroscopy (NIRS) is a non-invasive method that can be used to measure changes in the intracerebral oxygen environment [12, 13]. Intra-cerebral oxygenation is assessed by measuring the concentration of cerebral oxyhemoglobin (oxy-Hb), deoxyhemoglobin (deoxy-Hb), total hemoglobin (total-Hb) and cytochrome oxidase (cyt).

In this study, we investigated the effects of adrenaline-induced severe hypotension on brain oxygen metabolism and blood circulation to measure changes in oxy-Hb, deoxy-Hb, total-Hb and cyt when local anesthetic was infiltrated into the neck skin

Material and Methods

This observational study was approved by the Committee on Clinical Investigation for human research at Iwate medical University (approval number 01205).

We studied 16 patients with an American Society of Anesthesiologists physical status of I or II who were scheduled to undergo radical neck dissection. There were eight men and eight women with a mean age of 65.9 years and mean weight of 55.5 kg (Table 1). All patients underwent intravenous administration of atropine (0.05 mg/kg) and midazolam (0.5 mg/kg) 30 min 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 and vecuronium bromide (0.1 mg/kg), then maintained with sevoflurane (1.0–1.5%) in oxygen (40%). Fentanyl and remifentanil were administered after endotracheal intubation. A catheter was inserted into a dorsal artery of the foot after anesthetic induction. Arterial cannulation was performed to monitor the arterial blood pressure (ABP). The patients' hemodynamics and respiration were stable at this time. The surgeon infiltrated the skin around the right or left neck using 1% lidocaine (2.6 ± 0.9 mg/kg) in combination with 1/100,000 adrenaline (2.9 ± 0.9 μg/kg). Local infiltration was carried out at four or five points at the neck skin incision, and the infiltration time was controlled at 40–60 s using the same needle gauge.

We continuously monitored the systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), pulse rate (PR), and blood oxygen saturation (SpO_2) with Life Scope 8® (Nihon Kohden, Tokyo, Japan).

And we monitored changes in oxy-Hb, deoxy-Hb, total-Hb and cyt with a NIRO 500 near-infrared oxygen monitor® (Hamamatsu, Japan). The NIRO sensor was placed on the opposite sides of the forehead before starting the operation. The optodes of the NIRO were placed in an opaque optode holder supplied by the manufacturer and the holder was secured to the forehead with tape. This resulted in an optode separation of 4.8 cm. To ensure light shielding, the NIRO sensors were covered with crepe bandage wrapped loosely around the head. The NIRO measured the changes in parameters from a baseline that was set at zero at the start of measurement. Baseline measurements were made for 1 or 2 min.

All parameters were continuously recorded using a PowerLab 4/25T data acquisition system (ADInstruments, Bella Vista, Australia). Each parameter immediately before infiltration (control) was compared with that at reaching a minimum value of ABP, and 1 and 3 min after reaching a minimum value. Values are presented as mean ± standard deviation. Intra-group 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 $P<0.05$.

Results

As shown in Fig. 1, the changes in ABP in extra-oral regions were recorded using the Powerlab data system. When local anesthetic was infiltrated into the skin of the neck (Fig. 1a), ABP decreased rapidly and the duration of the trough was ~1 min. In other regions, for example, ABP decreased when local anesthetic was infiltrated into the skin of the abdomen (Fig. 1b).

There were significant differences between the values of SAP, DAP, MAP and PR before infiltration and those when ABP reached a minimum. There was a 21.4% (mean: 25.3 mmHg) decrease in SAP, 22.6% (mean: 16.9 mmHg) decrease in MAP, and 22.8% (mean: 12.4 mmHg) decrease in DAP, and a 4.8% (mean: 3.3 bpm) increase in PR when ABP reached a minimum. Data were normalized to the values before injection and expressed as a relative percentage (Fig. 2).

The average time from local anesthetic infiltration to the minimum ABP was 132±42 s and the mean duration of the trough was ~1 min (Fig. 3). Our results suggested that there was a time lag of ~132 s for changes in hemodynamics after infiltration of local anesthetic into the neck skin.

The changes in intracerebral oxygen environment are shown in Fig. 4. There were significant differences in oxy-Hb before infiltration (control) and at reaching a minimum ABP and at 3 min after reaching a minimum ABP. There was a 1.5±1.3 nmol/L decrease at reaching a minimum ABP, a 1.2±1.1 nmol/L decrease at 1 min after reaching a minimum ABP, and a 1.7±1.3 nmol/L increase at 3 min after reaching a minimum ABP. There were no significant changes in deoxy-Hb. There was a 0.7±0.4 nmol/L decrease at reaching a minimum ABP, a 0.1±0.6 nmol/L decrease at 1 min after reaching a minimum ABP, and a 0.0±1.1 nmol/L decrease at 3 min after reaching a minimum ABP. There was a significant difference in total-Hb before infiltration and at 1 min after reaching a minimum ABP. There was a 0.8±1.4 nmol/L decrease at reaching a minimum ABP, a 1.0±1.2 nmol/L decrease at 1 min after reaching a minimum ABP, and a 0.9±0.9 nmol/L increase at 3 min after reaching a minimum ABP. There were no significant changes in cyt. There was a 0.0±0.2 nmol/L increase at reaching a minimum ABP, a 0.3±0.6 nmol/L increase at 1 min after reaching a minimum ABP, and a 0.0±0.4 nmol/L decrease at 3 min after reaching a minimum ABP.

Table 1 Demographic data
Data are presented as mean±S.D

Number of patients	16
Age (years)	65.9±2.3
Weight (kg)	55.5±2.7
Sex (M:F)	8:8
Dose of lidocaine (mg/kg)	2.6±0.9
Dose of adrenaline (ug/kg)	2.6±0.9
Type of surgery	Radical neck dissection

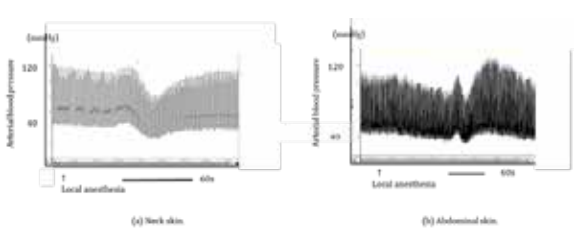

Fig. 1. Changes in arterial blood pressure in extra-oral regions. (a) Neck skin. (b)Abdominal skin. Arterial blood pressure changed with a delay of ~55 s (a) and ~108 s (b), and the duration of the trough was ~1 min (a and b).

Discussion

We established two important clinical issues. Local infiltration into the skin of the neck with adrenaline and lidocaine elicits temporary but moderate hypotension, which has an effect on intracerebral oxygen environment.

We report temporary but moderate hypotension induced by adrenaline infiltrated with lidocaine into the skin of the neck. Our literature survey using PubMed and Medline found no previous reports. However, previous studies have shown that adrenaline/lidocaine infiltration into the scalp before craniotomy, nasal mucosa for functional endoscopic

Time	Systolic arterial pressure (mmHg)	Mean arterial pressure (mmHg)	Diastolic arterial pressure (mmHg)	Pulse rate (bpm)
Before injection	116.9±15.2	74.6±8.6	54.3±5.1	55.9±3.5
Minimum	91.6±6.7	57.7±6.7	41.9±3.5	58.6±2.1
3 min after injection	134.6±20.3	76.4±8.2	50.9±3.1	60.1±4.2
5 min after injection	138.0±19.6	78.8±10.4	50.2±4.5	57.6±3.4

Table 2 Changes of arterial blood pressure and pulse rate and intra cerebral oxygen environment
(a): Changes of arterial blood pressure and pulse rate

Time	Δoxy-Hb	Δdeoxy-Hb	Δtotal-Hb	Δcyt
Before injection	0.0	0.0	0.0	0.0
Minimum	-1.5±1.3※	-0.7±0.4	-0.8±1.4	0.0±0.2
3 min after injection	-1.2±1.1	-0.1±0.6	-1.0±1.2※	0.3±0.6
5 min after injection	1.7±1.3※	0.0±1.1	0.9±0.9	0.0±0.4

(b): Changes of intra cerebral oxygen environment

sinus surgery, and oral mucosa for oral and maxillofacial surgery and dental treatment, induces marked hemodynamic changes, including decreased blood pressure during general anesthesia [1-3].

The hemodynamic effects of adrenaline are dose-dependent and different doses may activate different types of sympathetic receptors. A rate of 1–2 μg/min, although rarely used, should predominantly activate β_2-receptors with resulting vascular and bronchial smooth muscle relaxation. A rate of 2–10 μg/min should predominantly activate β_1-receptors to increase PR, contractility, and conduction and decrease the refractory period. Doses in excess of 10 μg/min cause marked α-adrenoceptor stimulation with generalized vasoconstriction [1, 3, 4]. Yang [5] reported that Mean arterial pressure (MAP) temporarily decreased by >30%, with the average time from initiation of local infiltration to the lowest MAP being 102 s, and the changes in blood pressure and PR tended to be relatively stable after 5 min, when 1% lidocaine with 40, 80, 160 μg adrenaline was applied. Neither MAP nor PR changed significantly when 1% lidocaine without adrenaline was applied.[4] The major mechanism for the occurrence of hypotension was presumed activation of β_2-receptors [1, 3, 5]. In our study, the average dose of adrenaline was 2.6 μg/kg (~144.3 μg), the average time to the lowest MAP was 132 s, and the average duration of the trough was ~1 min.

That decrease in MAP and increase in PR at 1, 1.5 and 2 minutes were also observed when 1% lidocaine with 40, 80, 160 μg adrenaline was applied. And so the increase to the highest PR was approximately 10 bpm after the beginning of local infiltration [4]. In our study, the average increase to the highest PR was ~3.3 bpm. The increase in PR was mainly due to baroreceptor-reflex-induced decrease in BP and gentle stimulation of β_2-receptors [4, 5].

In the present compared with previous studies, the decrease in rate of change of SAP, MAP and DAP, and increase in PR, were smaller and the average time to the lowest MAP was longer. We think this is because the increase in plasma adrenaline concentration is lower in neck skin

than in oral mucosa or scalp regions.

Blood pressure changes, such as moderate hypertension or hypotension, affect brain oxygen metabolism and blood circulation [10, 11]. We measured the changes in intracerebral oxygen environment with NIRS and assessed intracerebral oxygenation to measure oxy-Hb, deoxy-Hb, total-Hb and cyt. There was a mean decrease in oxy-Hb, mean increase in deoxy-Hb, mean decrease in total-Hb and mean increase in cyt. These results suggest cerebral ischemia. The decrease in oxy-Hb occurred as a result of decreased arterial blood flow into the brain; the increase in deoxy-Hb occurred as a result of the increase in oxygen extraction fraction; and the decrease in total-Hb occurred as a result of the decrease in cerebral blood volume [12, 13]. We conclude that, if we infiltrate adrenaline/lidocaine into the neck skin for head and neck surgery, temporal hypotension may increase the risk of or exacerbate cerebral ischemia. In general anesthesia, the effects of the observed changes in oxy-Hb detected by NIRS deserve comment. NIRS currently measures changes in oxy-Hb and total-Hb from an arbitrary baseline, and it is therefore not possible to calculate saturation changes directly. NIRS measures samples from arterial, venous and capillary compartments, but the relative contribution of each to the signal has not been defined. It is therefore not possible to determine whether the observed changes in saturation and blood volume occurred equally in all three compartments [12].

In the present study, the sample size was small (n=16) and this may not have been sufficient to determine statistical significance. However, we believe that the data provide accurate and reliable information on hemodynamic changes induced by adrenaline/lidocaine when infiltrated into the skin of the neck, and are clinically useful for management of anesthesia.

Conclusions

Local infiltration of adrenaline/lidocaine into the skin of the neck elicits temporary but severe hypotension, which affects intracerebral oxygen environment. Such hypotension may also increase the risk of or exacerbate cerebral ischemia. It is prudent to consider the possibility of marked hemodynamic changes when adrenaline/lidocaine is infiltrated into the neck skin.

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