Epidural anesthesia affects pulse oximeter readings and response time

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Abstract: We investigated the effects of epidural anesthesia on pulse oximeter readings (Spo₂) and response time because this type of anesthesia causes significant changes in microcirculation at measurement sites. Twenty patients were divided into lumbar epidural (L-EPI; n = 10) and the cervical epidural (C-EPI; n = 10) groups. Spo₂ and skin blood flow (SBF) were measured at the finger and toe simultaneously by pulse oximeter and laser Doppler flowmeter, respectively. Data were collected before and after epidural anesthesia for 1 min and the response time was calculated by the difference between the finger and toe using the breath-holding method. Epidural anesthesia increased SBF in the blocked area and decreased it in the nonblocked area in both groups (P < 0.01, respectively). In the L-EPI group, Spo₂ was increased at the finger (P < 0.05) and decreased at the toe (P < 0.05). In the C-EPI group, Spo₂ at both the finger and toe was decreased by the anesthesia. ΔSpo_2 (Spo₂ at the finger minus Spo₂ at the toe) was increased in the L-EPI group (P < 0.05) and decreased in the C-EPI group (P < 0.01). The difference in the response time became larger in the C-EPI group and smaller or opposite in the L-EPI group after anesthesia. The difference in response time and SBF were significantly correlated (r = 0.71; P < 0.05). These results indicated that epidural anesthesia lowered Spo₂ and shortened the response time through vasodilation in the blocked area and caused the opposite reactions in the nonblocked area through compensatory vasoconstriction.

Key words: pulse oximeter, epidural anesthesia, skin blood flow

Introduction

The pulse oximeter is a widely used type of oximetry monitor during anesthesia and other clinical procedures, because it provides a simple means to estimate

arterial oxygen saturation (Sao₂). However, pulse oximeter readings (Spo₂) are affected by many factors which cause the readings to differ from Sao_2 [1–3]. Among the factors, the decrease in blood flow at the measurement site due to vasoconstriction has been the focus of research because the detection of pulsatile flow is essential to distinguish arterialized blood from other nonpulsatile flow components to calculate Spo_2 [4,5]. In addition, low blood flow was reported to prolong the response time of the pulse oximeter, that is, the delay in recognition of changes in systemic Sao₂ [6]. During anesthesia, the peripheral vascular tone changes over a wide range, especially under regional anesthesia. Despite the routine use of the pulse oximeter during anesthesia, to our knowledge, no other reports have mentioned the influence of the anesthesia on pulse oximeter readings. The present study was designed to investigate the influence of epidural anesthesia on Spo₂ and the response time through simultaneous measurement at the finger and toe in relation to the skin blood flow measured by a laser Doppler flowmeter.

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Methods and materials

After obtaining approval for the study protocol from the Sapporo Medical University Committee on Human Research and informed consent from the patients, twenty ASA class 1 or 2 patients were assigned to the cervical epidural group (C-EPI; n = 10) and the lumbar epidural group (L-EPI; n = 10) depending on the region of surgery. An epidural catheter was inserted via the L1-2 or L2-3 interspace for the L-EPI group and via the C7-T1 interspace for the C-EPI group. Spo₂ at the second finger and at the second toe were measured simultaneously using two pulse oximeters (Nellcor N200, California, USA) with clip-type probes for adults. Skin blood flow at the first finger and the first toe were measured with two laser Doppler flowmeters

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were applied to the finger and toe alternatively to prevent mechanical bias. After completing the control measurement, 10 to 12 ml of 1.5% epinephrine-free lidocaine was injected through the epidural catheter, taking care to avoid accidental intravenous or intrathecal injection by checking vital signs. All the data collected from the pulse oximeters and the flowmeters were transferred to a PC9801 personal computer (NEC, Tokyo, Japan) once every second through an analogdigital converter (Canopus, Kobe, Japan). Before and 20 min after lidocaine injection, data were collected for 1 min when the patients' respiration was stable, and then averaged. Patients were also requested to hold their breath for as long as possible to desaturate arterial blood before and after the epidural anesthesia. The analgesic level was checked by the pin-prick method after completing measurement. The temperature of the operating room was maintained at 23°-25°C throughout the measurement. None of the patients required any procedure or drug to maintain blood pressure (e.g., ephedrine) except for an increase of infusion rate from 6 to 10 ml/kg/h. Statistical analysis was performed by Student's t-test for parameters and the chi-square method for patients' characteristics. P values of less than 0.05 were considered significant.

Results

There were no significant differences among the patients' characteristics except for the sex ratio in both groups (Table 1), because most cervical epidural anesthesia was used for mastectomy. During the control period, skin blood flow at the toe was low in both groups. In L-EPI group, skin blood flow was signifi-

Table 1. Characteristics of patients

∆SpO₂ (%)

2



Fig. 1. Changes in ΔSpo_2 induced by epidural anesthesia. ΔSpo_2 was calculated to offset the systemic changes in Sao₂ by subtracting Spo₂ at the toe from Spo₂ at the finger. *Closed circles* and *open circles* represent Mean ± SEM in the L-EPI and C-EPI groups, respectively. *PRE*, pre-epidural; *POST*, post-epidural. ***P* < 0.05 *vs* PRE; **P* < 0.05, ***P* < 0.01 *vs* L-EPI group

cantly increased at the toe and declined at the finger after the anesthesia. In C-EPI group, changes in skin blood flow at both the finger and the toe were the opposite of those seen in L-EPI group (Table 2).

During the control measurement, Spo_2 at the toe was statistically higher than at the finger in both groups. After the anesthesia, Spo_2 at the finger in L-EPI group was significantly increased, accompanied by a decrease in Spo_2 at the toe. In the C-EPI group, in contrast, Spo_2 decreased significantly at the finger with a tendency to decrease at the toe. ΔSpo_2 was calculated to offset the

Group	n	Age (years)	Sex (F/M)	Body weight (kg)	Analgesic level
L-EPI	10	40.0 ± 3.6	8/2	53.3 ± 2.4	$\begin{array}{c} T_{9,2} \sim L_{1.6} \\ C_{4,3} \sim T_{6,4} \end{array}$
C-EPI	10	50.2 ± 1.8	9/1	50.2 ± 1.8	

Mean \pm SEM.

L-EPI, lumbar epidural group; C-EPI, cervical epidural group.

Table 2. Effects of epidural anesthesia on Spo₂ readings and skin blood flow

Group		$\mathrm{Spo}_{\mathrm{2f}}$	Spo_{2t}	$\mathbf{SBF}_{\mathrm{f}}$	\mathbf{SBF}_{t}
L-EPI	pre	96.3 ± 1.0	$98.7 \pm 0.5*$	24.4 ± 4.9	6.4 ± 2.3**
	post	$97.9 \pm 0.8^{*}$	$97.2 \pm 0.5^{*}$	$17.2 \pm 4.8^{\text{##}}$	27.6 ± 2.5****
C-EPI	pre	95.7 ± 0.9	$97.1 \pm 0.9^{**}$	24.4 ± 3.2	8.4 ± 2.2**
	post	93.7 ± 0.9#	$96.2 \pm 1.1^{**}$	32.7 ± 3.2 ^{##}	$6.8 \pm 1.4^{**#}$

Mean ± SEM.

L-EPI, lumbar epidural group; C-EPI, cervical epidural group; pre, preoperatively; post, postoperatively.

* $P < 0.05 \text{ vs Spo}_{21}$ or SBF₆, ** $P < 0.01 \text{ vs Spo}_{21}$ or SBF₆; * P < 0.05 vs pre; ** P < 0.01 vs pre.



Fig. 2. Changes in the pulse oximeter response time caused by epidural anesthesia. The response time evaluated as the difference between the time at which a change in Spo_2 caused by holding breath appeared at two separate sites. Note that the response time difference of some patients exceeded 60 s before or after the epidural anesthesia



Fig. 3. Relationship between the skin blood flow (SBF) difference and the response time difference (16 points from 8 patients). The ordinate represents the difference in SBF calculated by subtracting SBF at the toe from SBF at the finger. The abscissa is the difference in response time between the toe and the finger calculated in the same manner as the Spo₂ difference

decrease at the toe. ΔSpo_2 was calculated to offset the systemic changes in Sao₂ by subtracting Spo₂ at the toe from that at the finger. As a result, ΔSpo_2 was opposite in the two groups; there was an increase from $-1.9 \pm 0.6\%$ to $0.7 \pm 0.6\%$ in L-EPI group and a decrease from $-0.9 \pm 0.3\%$ to $-2.5 \pm 0.4\%$ in the C-EPI group (Fig. 1).

Eight of the 20 patients (4 from each group) could hold their breath until the pulse oximeter readings changed. During control measurement, the changes were detected earlier at the finger than at the toe. After epidural anesthesia, the response time difference between the finger and the toe was greater in the C-EPI group and became smaller or opposite in the L-EPI group (Fig. 2). The difference in response time between the two sites was significantly correlated to the difference in skin blood flow (Fig. 3; P < 0.05).

Discussion

The skin blood flow in the present study was measured with a laser Doppler flowmeter, using a method newly developed for the clinical evaluation of microcirculation. The measurement principle is based on the detection of the Doppler shift of a laser beam from moving erythrocytes in vessels. The product of the Doppler shift multiplied by the volume of moving erythrocytes is equal to the blood flow in the hemisphere. The laser beam reaches a depth of 1 mm, which includes the subcutaneous arteriovenous anastomosis (AVA).

The results of the present study demonstrated that Spo₂ and the response time were affected by skin blood flow at the measurement sites. The finger and the toe are well known to have many AVAs, which are innervated by sympathetic nerves [7]. The amount of blood passing through AVAs was increased four times by pharmacological denervation [7]. Because high pressure arterial flow is shunted through AVAs into more compliant low pressure veins, the pulsatile component created in the veins should be greatly amplified [8]. Therefore, venous or venous-arterial mixed blood is also detected by a pulse oximeter as pulsatile component in blocked areas, resulting in a lower Spo₂. In the L-EPI group, skin blood flow decreased after epidural anesthesia probably due to compensatory vasoconstriction. A further decrease in shunt flow through AVAs at the finger and the subsequent reduction of venous blood pulsation may explain the increase in Spo₂ at the finger in this group. In the C-EPI group, the Spo_2 at both the finger and the toe were decreased by anesthesia, probably due to respiratory muscle relaxation. However, Δ Spo₂, which was calculated to offset the systemic changes in Sao₂, revealed that the decrease in Spo₂ at the finger was greater than that at the toe (Fig. 1). These results suggest that Spo₂ is increased by vasoconstriction and decreased by vasodilation. Increased sympathetic tone in patients in the operating room may enhance the effects of epidural anesthesia on Spo₂.

Another possible reason for the difference in Spo_2 may be a bias derived from the mechanical characteristics of the pulse oximeter. Pulse oximeter readings are adjusted by the manufacturer according to an empirical formula obtained from healthy volunteers [6]. Thus, Spo₂ does not necessarily reflect the actual Sao₂ under nonphysiological conditions, such as severe hypoxia. However, the measurement in this study was performed in normoxic condition, except for breath holding, and Spo₂ was shown to be within a physiological range. Although epidural anesthesia-induced changes in skin blood flow do not seem to be far from the fluctuation during the control period, changes in the signal quality of the pulse oximeter caused by increases or decreases

in skin blood flow, which were not evaluated in the

present study, might also contribute to changes in Spo₂. The response time of the pulse oximeter was demonstrated to be affected by epidural anesthesia based on the difference between the time of appearance of the change in Spo₂ at two different sites induced by breath hold as a means of avoiding invasive Sao₂ monitoring. It is clinically important that the difference between the two sites exceeded 60 s in some patients before or after anesthesia, which means that the recognition of the systemic changes in Sao₂ should be further delayed. The dependency of the response time of the pulse oximeter on local blood flow was previously reported [9]. Our findings, however, demonstrated that a clinically significant delay was observed in patients undergoing elective surgery under conditions like hypothermia, hypovolemia, or cardiogenic shock [10]. Epidural anesthesia could shorten the response time in the blocked area but cause a clinically significant delay in nonblocked areas due to compensatory vasoconstriction.

In conclusion, the present study showed that Spo_2 differed at the finger and toe due to skin blood flow differences. The response time of the pulse oximeter to

systemic Sao_2 changes depended on the skin blood flow at the measurement sites. Epidural anesthesia lowered Spo_2 and shortened the response time through vasodilation in the blocked area and caused an opposite reaction in the nonblocked area through compensatory vasoconstriction.

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