Efficacy of noninvasive pulse co-oximetry as compared to invasive laboratory-based hemoglobin measurement during spinal anesthesia

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Background: The Masimo Radical 7 (Masimo Corp., Irvine, CA, USA) pulse co-oximeter® noninvasively determines the hemoglobin concentration using the principle of transcutaneous spectrophotometry. We compared hemoglobin levels determined using this device (SpHb) with those determined using an invasive laboratory-based technique (tHb) during spinal anesthesia.

Methods: Thirty patients received spinal anesthesia with 0.5% hyperbaric bupivacaine. The pulse co-oximeter probe was mounted on the second toe, and arterial blood samples were obtained from a radial artery catheter. SpHb, tHb, and perfusion index (PI) values were recorded before and 20 and 40 min after intrathecal injection of bupivacaine.

Results: Before spinal anesthesia, the SpHb and tHb showed a significant difference of −2.86 ± 1.56 g/dl ($P < 0.005$), but no significant differences were found between tHb and SpHb at 20 and 40 min after spinal anesthesia (−0.16 ± 2.45 g/dl and 0.29 ± 2.68 g/dl). Additionally, PI was significantly increased at 20 and 40 min after spinal anesthesia compared to the pre-anesthetic value ($P < 0.001$).

Conclusions: The toe is not the monitoring site for pulse co-oximetry in adult patients, but the pulse co-oximetry on the toe appears to be appropriate as a noninvasive hemoglobin monitoring device after spinal anesthesia. **(Anesth Pain Med 2014; 9: 277-281)**

Key Words: Hemoglobin, Pulse co-oximetry, Spinal anesthesia.

INTRODUCTION

The monitoring of hemoglobin levels during surgery is essential for patients in whom bleeding is anticipated. However, there are several problems associated with monitoring: it is an invasive procedure; requires continuous monitoring, which is impossible; and transfusion plans are delayed until test results are obtained. The recently developed Masimo Radical 7 (Masimo Corp, Irvine, CA, USA) pulse co-oximeter®, which uses a multi-wavelength spectrophotometric method, enables immediate, noninvasive, and continuous measurement of hemoglobin by attachment of a sensor to the patient's finger, similar to that used currently for monitoring oxygen saturation [1]. This device has an in-built processor and filter, which has an algorithm to identify and quantify various types of hemoglobin, including oxyhemoglobin, carboxyhemoglobin, and reduced hemoglobin.

Several studies have compared the accuracy of hemoglobin levels measured by invasive laboratory-based techniques (tHb) to those measured using a pulse co-oximeter (SpHb) and have found that the accuracy of SpHb is equal to that of tHb [1-3]. However, because of the principle of the measurement technique, i.e., transcutaneous spectrophotometry, the hemoglobin of pulse co-oximetry can be affected by blood flow dysfunction, a change in sympathetic tone, and the use of vasopressor drugs [4]. Studies on the effect of contraction or relaxation of peripheral blood vessels on hemoglobin measured by pulse co-oximetry are rare. In the present study, we determined and compared the changes in SpHb measured noninvasively before and after spinal anesthesia.

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MATERIALS AND METHODS

From among patients scheduled for lower extremity surgery under spinal anesthesia at our hospital from July to December 2011, 30 adults of ASA class 1 or 2 (age range, 20–59 years) were selected as subjects. This research was approved by the institutional review board of our hospital, and patients provided informed consent after the purpose of the study was explained to them.

Patients with diabetes, hypertension, a history of heart disease, abnormalities in blood coagulation or platelets, lumbar pain, central or peripheral nervous system disorder, vascular disease, abnormalities in the Allen test, abnormalities in toe skin and toenails, or a heart rate of < 50 beats/min were excluded. In addition, to rule out measurement errors due to extreme blood loss, patients with a blood loss volume of 100 ml or more were also excluded.

After the patients arrived in the operating room, the heart rate, blood pressure, electrocardiogram, and temperature were monitored using the patient monitoring system (Hewlett Packard-6M1046; Bőblingen, Germany). Temperature was measured on the foot sole, opposite side of operative field. To measure peripheral oxygen saturation (SpO_2), SpHb, and perfusion index (PI), the pulse co-oximeter sensor was placed on the second toe, opposite side of operative field. PI is the pulsatile signal indexed against the non-pulsatile signal and is an indication of localized perfusion. After stable SpO_2 and SpHb values were measured, a 22 G catheter was inserted in the left radial artery to collect arterial blood. The arterial hemoglobin oxygen saturation (SaO_2) and tHb in the arterial blood were measured using a blood gas analyzer (Rapidpoint® 400; Siemens, Munich, Germany).

7 ml/kg lactated Ringer's solution was administered to all patients for 30 minutes before study in order prevent a sudden decrease in blood pressure due to vasodilation, and 1.5 ml/kg/h was administered until the end of the study. Spinal anesthesia was administered with the patient in the lateral decubitus position, using a 25 G Quincke spinal needle inserted in the L3-4 or L4-5 intervertebral space in the midline. After the cerebrospinal fluid was checked, 10–12 mg 0.5% hyperbaric bupivacaine (Marcaine®; Astra Zeneca, Sweden) was injected for 30 s, and the patient was moved to the supine position. After 15 min, the sensory block level was checked using a pinprick, and the heart rate, blood pressure, temperature, SpO_2, SpHb, PI, SaO_2, and tHb were measured 20 and 40 min after spinal anesthesia.

The sensor of the pulse co-oximeter was covered with a black shield to prevent optical interference. The blood gas analyzer was calibrated daily, and the pulse co-oximeter was calibrated automatically before each measurement.

All measurements were expressed as mean ± standard deviation, and SPSS version 14.0 (SPSS Inc, Chicago, USA) was used for analysis. Repeated-measures ANOVA was used to examine changes in blood pressure, heart rate, SpO_2, SaO_2, SpHb, tHb, PI, and temperature. The paired t-test was used to analyze the difference between SpO_2 and SaO_2, and between SpHb and tHb. Statistical significance was defined as $P < 0.05$.

RESULTS

Patient characteristics and level of spinal anesthesia block are summarized in Table 1. Blood pressure and heart rate were significantly decreased after spinal anesthesia ($P < 0.05$; Table 2).

After spinal anesthesia, the tHb and SaO_2 were not significantly different but SpHb was significantly increased and SpO_2 was significantly decreased at 20 and 40 min compared to the pre-anesthetic value ($P < 0.001$; Table 3). The PI and temperature were significantly increased after spinal anesthesia ($P < 0.05$; Table 3).

Before spinal anesthesia, the SpHb and tHb showed a significant difference of −2.86 ± 1.56 g/dl ($P < 0.005$), but no significant differences were found between tHb and SpHb at 20 and 40 min after spinal anesthesia (−0.16 ± 2.45 g/dl and 0.29 ± 2.68 g/dl). The proportion of patients with SpHb − tHb = 1.5 g/dl or less was 55.9% after spinal anesthesia, compared with the 13.3% it was before spinal anesthesia, and the proportion of patients with SpHb − tHb = 1.6 g/dl or more decreased to 44.1% after spinal anesthesia compared with the 86.7% it was before spinal anesthesia ($P < 0.005$; Table

Table 1. Demographic Data (n = 30)

Age (yr)	41.0 ± 14.0
Sex (M/F)	24/6
Height (cm)	167.4 ± 8.3
Weight (kg)	69.6 ± 11.4
ASA class (I/II)	22/8
Level of thoracic sensory block	9.9 ± 1.2

Values are mean ± SD or number of patients. ASA: American society of anesthesiologists.

Table 2. Changes of Blood Pressure and Heart Rate before, 20 and 40 Minute after Spinal Anesthesia

	Before	20 min	40 min	P value
SBP (mmHg)	131.6 ± 16.1	121.4 ± 17.2*	121.6 ± 15.5*	< 0.001
DBP (mmHg)	79.3 ± 8.0	73.5 ± 10.0*	72.2 ± 10.0*	< 0.001
MBP (mmHg)	97.0 ± 10.4	91.3 ± 10.7*	90.8 ± 10.8*	0.006
HR (beats/min)	67.1 ± 12.1	62.8 ± 12.2	61.3 ± 10.6*	0.006

Values are mean ± SD. SBP: systolic blood pressure, DBP: diastolic blood pressure, MBP: mean blood pressure, HR: heart rate. *P < 0.05 compared with values before spinal anesthesia.

Table 3. Changes of SpO_2, SaO_2, SpHb, tHb, Perfusion Index and Temperature before, 20 and 40 Minute after Spinal Anesthesia

	Before	20 min	40 min	P value
SpO_2 (%)	98.6 ± 1.3‡	97.0 ± 1.5*	96.6 ± 1.8*	< 0.001
SaO_2 (%)	96.4 ± 1.3	96.7 ± 1.2	96.4 ± 1.3	0.345
SpHb (g/dl)	10.8 ± 1.2§	13.2 ± 1.9*	13.7 ± 2.0*†	< 0.001
tHb (g/dl)	13.7 ± 1.4	13.4 ± 1.4	13.4 ± 1.5	0.132
PI	2.1 ± 1.2	7.8 ± 3.3*	8.2 ± 3.5*	< 0.001
Temperature (^{o}C)	28.9 ± 1.5	34.7 ± 0.6*	35.2 ± 0.5*†	< 0.001

Values are mean ± SD. SpO_2: pulse oxygen saturation, SaO_2: arterial oxygen saturation, SpHb: non-invasive Masimo hemoglobin, tHb: invasive laboratory hemoglobin, PI: perfusion index. *P < 0.05 compared with values before spinal anesthesia. †P < 0.05 compared with values 20 minute after spinal anesthesia. ‡P < 0.05 compared with SaO_2. §P < 0.05 compared with tHb.

Table 4. Groups Based on Magnitude of Differences between Non-invasive Masimo Hemoglobin (SpHb) and Invasive Laboratory Hemoglobin (tHb) before, 20 and 40 Minute after Spinal Anesthesia

	Before	20 min	40 min	Total of 20 min and 40 min values
≤ 1.5 g/dl	4	16	17	33
	13.3%	53.3%	58.6%	55.9%
1.6-3.0 g/dl	13	8	5	13
	43.3%	26.6%	17.2%	22.0%
> 3.0 g/dl	13	6	7	13
	43.3%	20.0%	24.1%	22.0%
Total	30	30	29	59
	100%	100%	100%	100%

4).

The SaO_2 level was significantly lower than the SpO_2 before spinal anesthesia (2.14 ± 0.79%, P < 0.005), but no significant difference at 20 and 40 min after spinal anesthesia (0.26 ± 1.30% and 0.22 ± 1.63%).

DISCUSSION

In this study, there was no statistically significant difference between the tHb value after spinal anesthesia, and that before spinal anesthesia, but SpHb showed a significant increase after anesthesia. And differences between SpHb and tHb were decreased after spinal anesthesia.

The pulse co-oximeter has been used for several years as a monitoring device to measure hemoglobin levels noninvasively and continuously, and generally, it has provided satisfactory support in the evaluation of acute bleeding and decisions regarding transfusion [2,3]. However, the accuracy of SpHb levels is debatable. By determining the bias (mean difference between SpHb and tHb) and precision (1 SD of the bias) of the 2 techniques, Macknet et al. [1], Miller et al. [2], and Berkow et al. [3] concluded that SpHb had an accuracy equal

to that of tHb (−0.15 ± 0.92 g/dl, 0.26 ± 1.79 g/dl, and −0.1 ± 1.0 g/dl). On the other hand, Gayat et al. [5] and Nguyen et al. [6] reported that SpHb lacks clinical accuracy (1.8 g/dl and −1.7 g/dl). Our study showed bias and precision values of −2.86 g/dl and 1.56 g/dl, respectively, before spinal anesthesia, and in 86.7% of the cases, the values before anesthesia were 1.6 g/dl or higher. Possible explanations for these results could be as follows: First, the manufacturer recommends the fingertip is the standard monitoring site for pulse co-oximeter. The hand or foot (sometimes toe) is often used in neonatal patients. Surgical patients, however, are subject to unpredictable changes in peripheral perfusion, particularly with a large degree of variability in body temperature. But we mounted it on the second toe, as we were determining the SpHb of the lower extremities after spinal anesthesia. Second, the average temperature of the patient's soles measured before spinal anesthesia was low, at 28.9 ± 1.5°C, and contraction of the peripheral blood vessels due to hypothermia could have decreased the amount of blood flow in this region, resulting in lower SpHb than tHb. Third, the bias of SpHb is dependent on the type of infusion fluid. Bergek et al. [7] reported that SpHb had decreased more than tHb at the end of the infusion of Ringer's acetate 20 ml/kg over 30 min. They hypothesized that fluid load might have disturbed this balance by expanding the vessels and also by diluting Hb differently in arterioles, capillaries, and veins. Faster disappearance of crystalloids than colloids from the bloodstream promotes tissue edema, which could diminish the relative strength of the pulsatile part of the signal by affecting the background noise. Finally, zero adjustment was performed on the equipment before the experiment with each patient, but machine error cannot be excluded.

When changes in the measured values according to spinal anesthesia were examined, no difference was found between tHb before and after spinal anesthesia, but SpHb was greater after anesthesia. This increase is presumed to result from the expansion of blood vessels in the lower extremities due to sympathetic nerve block after spinal anesthesia, which increases the amount of blood flow. Miller et al. [8] compared a group of 20 subjects with finger digital nerve block (DNB) to a group of 20 subjects without DNB and found high PI in the DNB group. In particular, when the PI exceeded 2.0, the accuracy of SpHb increased, and 83% of such cases showed a difference of 1.5 g/dl or less between SpHb and tHb. Miller et al. [8] attributed this result to an increase in blood flow due to DNB. In our study as well, PI increased significantly to 7.84 ± 3.38 at 20 min after spinal anesthesia and to 8.27 ± 3.58 at 40 min after spinal anesthesia, compared with the 2.13 ± 1.20 it was before spinal anesthesia ($P < 0.05$; Table 3). In addition, cases in which the difference between SpHb and tHb exceeded 1.6 g/dl decreased to 44.1% after spinal anesthesia, from 86.7% before spinal anesthesia. Miller et al. [8] demonstrated a similar result, wherein the percentage of cases showing a difference of 1.6 g/dl between SpHb and t H b values, was at 17% while using the DNB as compared to 29% while not using DNB. Possible explanations for this phenomenon include the variability of blood flow due to degree of the block and due to degree of dilatation of the blood vessels in the lower extremities.

There was a difference between SpO_2 and SaO_2 before spinal anesthesia, but after spinal anesthesia, the SaO_2 was not significant different and the SpO_2 decreased significantly, so although the significant difference between the two measurements disappeared. There is contradictory evidence regarding the changes in SpO_2 after spinal anesthesia. In previous studies, the SpO_2 of the upper extremities was found to decrease after spinal anesthesia or epidural anesthesia, but the SpO_2 of the lower extremities increased [9,10]. On the other hand, some studies have reported no changes in SaO_2 after spinal anesthesia but an increase in SpO_2 in the upper extremities [11]. In addition, Yang et al. [12] reported no change in SpO_2 in the upper extremities after spinal anesthesia but a decrease in the SpO_2 of the lower extremities, which is similar to our research results (Table 3). The precise mechanism for the decrease in SpO_2 in the lower extremities after spinal anesthesia is not known, but it is thought to result from the increased amount of blood flow in the skin over the lower extremities and the resulting decrease in arteriovenous shunting and congestion. Hynson et al. [13] and Schramm et al. [14] reported that changes in skin temperature affect oxygen saturation; when lower extremity temperature increases because of vasodilation caused by sympathetic nerve block, the diffusion of arterial pulsations into veins is quickened, which reduces the oxygen saturation of arterial blood.

The present study has certain limitations, the first of which is the limited number of patients examined. Second, if another pulse co-oximeter from the same company had been used on the upper extremities along with the lower extremities during spinal anesthesia, the measured values could be compared to better assess credibility. Third, the manufacturer recommends mounting the pulse co-oximeter sensor on the finger, but we mounted it on the second toe in order to measure the

hemoglobin levels in the lower extremities after spinal anesthesia. Hence, some errors may have ensued. Additional research is required to determine the accuracy of SpHb according to the location at which the pulse co-oximeter sensor is mounted.

In conclusion, although the SpHb and tHb showed a significant difference before spinal anesthesia, no significant differences were found between tHb and SpHb at 20 and 40 min after spinal anesthesia. On the basis of these results, the toe is not the monitoring site for pulse co-oximetry in adult patients, but the pulse co-oximetry on the toe appears to be appropriate as a noninvasive hemoglobin monitoring device after spinal anesthesia.

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