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IMPACT OF SKIN INCISION ON THE PLETH VARIABILITY INDEX

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ABSTRACT. Objective. The pleth variability index (PVI), which is calculated from respiratory variations in the perfusion index (PI), reportedly predicts fluid responsiveness. However, vasomotor tone fluctuations induced by nociceptive stimuli change the PI and may reduce the accuracy of PVI. The aim of this study was to confirm the effects of surgical stimuli on PVI. **Methods.** Twenty-four patients were examined after the induction of general anesthesia. Heart rate (HR), mean arterial blood pressure (MBP), PI, PVI, stroke volume variation (SVV), and cardiac index (CI) were recorded before and after the skin incision. PI and PVI were calculated using a Radical 7 pulse oximeter, and SVV and CI were calculated using the FloTrac/Vigileo system. **Results.** After the skin incision, the PI decreased significantly from 5.3 (4.0–6.2%) to 3.6% (1.8–4.7%), whereas the PVI increased significantly from 9.5 (7.0–12.0%) to 13.5% (9.0–16.0%). A significant negative correlation was observed between the changes in PI and PVI before and after the skin incision. The skin incision did not affect the HR, CI, or SVV but increased the MBP. **Conclusion.** This study showed a significant increase in the PVI and a negative correlation between the changes in PVI and PI before and after the skin incision. The PVI can be calculated from the variations in the PI caused not by mechanical ventilation, but rather by fluctuations in vasomotor tone. When using the PVI as an indicator for fluid responsiveness, it is crucial to pay attention to fluctuations in vasomotor tone induced by nociceptive stimuli.

KEY WORDS. pleth variability index, perfusion index, skin incision.

INTRODUCTION

Dynamic indicators that rely on respiratory variations in arterial pulse pressure [1, 2] and in the pulse contour stroke volume (SV) [3, 4] in mechanically ventilated patients have been established as sensitive predictors of fluid responsiveness, compared with conventional static indicators such as central venous pressure or capillary wedge pressure. The plethysmographic signal displayed during pulse oximetry resembles the peripheral arterial pressure waveform, and the respiratory variations in the pulse oximetry photoplethysmographic (POP) waveform amplitude have recently been proposed as an alternative to invasive dynamic indicators [5–7]. In addition, the pleth variability index (PVI; Masimo Corp., Irvine, CA, USA), which is calculated from respiratory variations in the perfusion index (PI) in mechanically ventilated patients, has also been reported to be an equally accurate predictor

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of fluid responsiveness, similar to other dynamic indicators [8, 9]. The main advantage of PVI is that it provides clinicians with a numerical value that can be obtained noninvasively, automatically, and continuously.

Various stimuli, such as surgical stress [10, 11], depth of anesthesia [12, 13], pharmacological influences [14], and cold stress [15], have been confirmed to alter vasomotor tone and to cause changes in the POP waveform amplitude. The POP signal is divided into two components: a direct current (DC) and an alternating current (AC) component. The DC component is the nonpulsatile component, whereas the AC component is the pulsatile component [11, 16]. The DC component is filtered out, and typically only the AC component is displayed as the POP waveform in most commercial pulse oximeters, while the PI is indicated as the ratio of the AC component to the DC component in the Masimo Radical 7 pulse oximeter [8, 9]. Since the PI has been reported to reflect changes in peripheral perfusion [17] and to be sensitive to vasomotor tone [18–20], various stimuli that cause sympathetic vasoconstriction might decrease the PI, as well as the POP waveform amplitude; in addition, the variation in the PI induced by not only the heart–lung interaction during mechanical ventilation, but also the changes in vasomotor tone might reduce the accuracy of PVI as a predictor of fluid responsiveness.

The aim of this study was to confirm whether surgical stimuli have an impact on the PVI in patients under general anesthesia.

METHODS

Patients and anesthesia

This study was approved by the Human Ethics Committee of Kagoshima University Hospital. All the subjects provided their written informed consent to participate in the study. The subjects were 24 ASA I or II patients who were scheduled for elective surgery under general anesthesia with endotracheal intubation. The operations consisted of abdominal and/or thoracic surgery in 20 patients, face or neck surgery in two patients, and lower-extremity surgery in two patients. The 13 men and 11 women had a median age (interquartile range) of 57 (53–68) years. Their median body weight was 58 (54–64) kg, and their median body height was 160 (154–166) cm. Patients with cardiac arrhythmia, an intracardiac shunt, valvular heart disease, reduced left ventricular function (preoperative ejection fraction $<50\%$), or peripheral vascular disease were excluded. After the placement of the standard monitors, including an electrocardiogram, pulse oximetry, end-tidal carbon dioxide, and noninvasive blood pressure monitors, anesthesia was induced with propofol (1 mg/kg), remifentanil (0.3 μg/kg/min), and vecuronium (0.1 mg/kg) and maintained with remifentanil (0.1–0.5 μg/kg/min) and sevoflurane (1.5%). Following endotracheal intubation, mechanical ventilation was performed with an oxygen concentration of 50%, a tidal volume of 8–10 mL/kg, and a respiratory rate of 10–12 breaths/min to obtain normocapnia (end-tidal carbon dioxide range of 35–40 mmHg). None of the ventilator settings were changed during the study. Vecuronium (2 mg) was administered every 30 min to eliminate the spontaneous respiratory effort. In addition to the standard monitors, a 20-G catheter was inserted into the left or right radial artery for continuous monitoring of the radial arterial pressure, and a pulse oximeter probe (LNOP® Adt; Masimo Corp., Irvine, CA, USA) was attached to the index finger or the third finger of the same hand and shielded to prevent outside light from interfering with the signal. The pulse oximeter probe was connected to a Masimo Radical 7 monitor with PVI software (version 7.3.1.1). In 12 patients, the radial artery catheter was also connected to the FloTrac™/Vigileo™ system (Edwards Lifesciences, Irvine, CA, USA, version 1.74) to allow the measurement of SV, CO, and stroke volume variation (SVV). The intravenous infusion of bicarbonate Ringer's solution at a rate of 4 mL/kg/h was continued throughout the study.

Calculation of PVI

The PVI was calculated based on the dynamic changes in PI that occurred during a complete respiratory cycle. The Radical 7 automatically and continuously calculated the PVI from the changes in PI over a time interval sufficient to include one or more complete respiratory cycles using the following formula: $\mathrm{PVI} = ([\mathrm{PI}_{max} - \mathrm{PI}_{min}]/\mathrm{PI}_{max}) \times 100$. PI was expressed as the ratio of the AC component divided by the DC component ($\mathrm{PI} = [\mathrm{AC}/\mathrm{DC}] \times 100$).

Calculation of SVV

The SVV was calculated from the percent changes in the SV during a respiratory cycle. The FloTrac™/Vigileo™ system automatically and continuously calculated the SVV using the following formula: $\mathrm{SVV}\ (\%) = (\mathrm{SV}_{max} - \mathrm{SV}_{min})/\mathrm{SV}_{mean}$, where SV_{max}, SV_{min}, and SV_{mean} were determined during a time window of 20 s. The system calculates SV based on arterial pulsatility (standard deviation of the pulse pressure over a 20-s interval), vascular resistance, and arterial compliance data. The proprietary algorithm is described in detail elsewhere [21]. CO was calculated using the following formula: CO = heart rate (HR) × SV.

Study protocol

Monitoring was begun in all the subjects after at least a 5-min period of hemodynamic stability without volume expansion. The remifentanil dosage was increased to 0.3–0.5 μg/kg/min at 5 min before the skin incision, and the dose was adjusted by the attending anesthesiologist according to the changes in the blood pressure and HR caused by the stimuli associated with surgery. The PVI, PI, SVV, cardiac index (CI), mean radial artery pressure (MBP), and HR were recorded just before the skin incision and again at 1 and 5 min after the skin incision. Data recorded before the skin incision was measured at least 15 min after the tracheal intubation in all the subjects.

Statistical analysis

The data were expressed as the median values and the interquartile range (25–75%). The Friedman test was used to identify significant changes in all the variables during the study period; if a significant change was detected, pairwise post-hoc comparisons were performed using the Wilcoxon signed-rank test with a Bonferroni correction ($P \leq \alpha/n$) to avoid numerous spurious positive results, since the alpha value needed to be lowered to account for the number of comparisons being performed. Therefore, *P* values on the Friedman test and the Wilcoxon signed-rank test that were less than 0.05 and 0.0167, respectively, were considered statistically significant. Correlations between the changes in the PVI and the PI were analyzed using the Spearman rank correlation coefficients and a simple linear regression analysis, and *P* values of less than 0.05 were considered statistically significant.

RESULTS

Data obtained before and 1 and 5 min after the skin incision are shown in Table 1 and Figure 1. Significant decreases in PI from 5.3% (4.0–6.2%) before to 3.6% (1.8–4.7%) at 1 min after and 3.3% (2.2–4.5%) at 5 min after the skin incision and significant increases in the PVI from 9.5% (7.0–12.0%) before to 13.5% (9.0–16.0%) at 1 min after and 16.5% (12.0–20.5%) at 5 min after the skin incision were observed. The MBP increased significantly from 65.5 (60.0–74.0 mmHg) to 70.0 mmHg (63.5–80.0 mmHg) at 1 min after the skin incision; however, no significant changes in the HR, CI, or SVV were observed before and after the skin incision.

The correlation between the changes in the PI and PVI values observed before the skin incision and at 1 min after the skin incision is shown in Figure 2. The changes in the PI and PVI were calculated as the ratio of the PI value and the PVI value after the skin incision to their respective values before the skin incision. A significant negative correlation ($P < 0.0001$, $r^2 = 0.747$) was observed between the changes in the PI and PVI values observed before and 1 min after the skin incision, and a weak but significant negative correlation ($P = 0.0014$, $r^2 = 0.376$) was observed between the changes in the PI and PVI values before and 5 min after the skin incision. In addition, a poor negative correlation ($P = 0.026$, $r^2 = 0.206$) was observed between the changes in the PI and MBP values observed before and 1 min after the skin incision. The change in the MBP was calculated in the same way as the changes in the PI and PVI.

DISCUSSION

The results of this study showed that increases in the PVI were accompanied by decreases in the PI after skin

Table 1. Data obtained before and after skin incision

	Before skin incision	1 min after skin incision	5 min after skin incision	Friedman test *P* value
MBP (mmHg) n = 24	65.5 (60.0–74.0)	70.0 (63.5–80.0)★	71.0 (65.0–80.3)	<0.01
HR (beats/min) n = 24	68.0 (63.8–75.3)	67.0 (62.0–77.5)	66.0 (63.0–75.0)	NS
CI (L/min/m^2) n = 12	2.5 (2.3–2.8)	2.3 (2.3–3.4)	2.4 (2.3–2.9)	NS
SVV (%) n = 12	9.0 (7.5–11.5)	9.0 (6.0–10.0)	8.0 (6.0–9.5)	NS
PI (%) n = 24	5.3 (4.0–6.2)	3.6 (1.8–4.7)★	3.3 (2.2–4.5)★	<0.01
PVI (%) n = 24	9.5 (7.0–12.0)	13.5 (9.0–16.0)★	16.5 (12.0–20.5)★	<0.01

Data are shown as the medians (interquartile range) and were evaluated using the Friedman test ($P < 0.05$).*MBP* mean radial artery blood pressure, *HR* heart rate, *CI* cardiac index, *SVV* stroke volume variation, *PI* perfusion index, *PVI* pleth variability index. ★ Wilcoxon signed rank test with Bonferroni correction, $P < 0.0167$ versus before skin incision was regarded as significant.

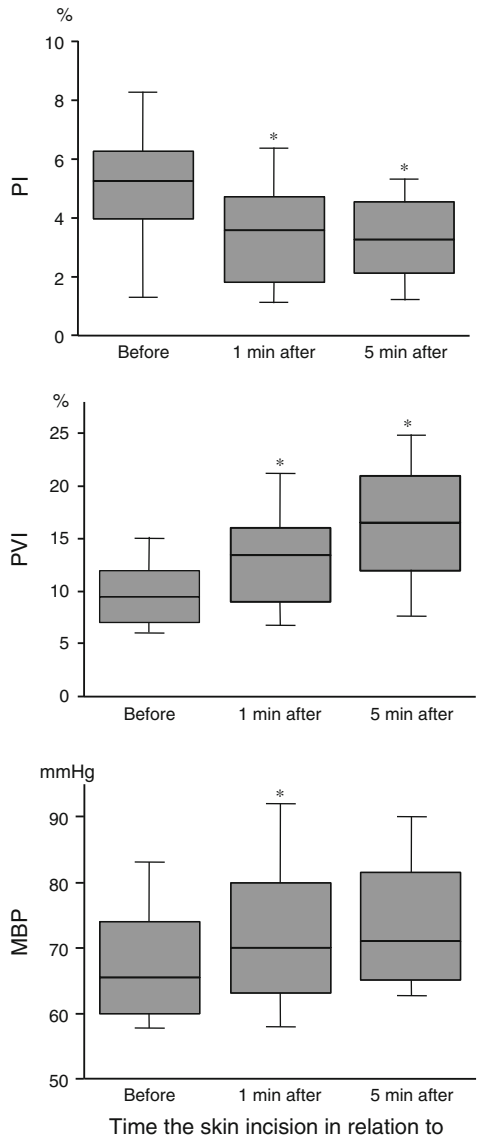

*Fig. 1. Changes in the PI, PVI, and MBP before and after skin incision. The PVI, PI, and MBP were recorded just before the skin incision (before), 1 min after the skin incision (1 min), and 5 min after the skin incision (5 min). The middle line of each box represents the median value, the edges of the box represent the inter-quartile range, and the error bar represents the 10th and 90th percentiles. *$P < 0.0167$, compared with the values obtained before the skin incision using the Wilcoxon signed rank test with Bonferroni correction. PI perfusion index, PVI pleth variability index, MBP mean radial artery blood pressure.*

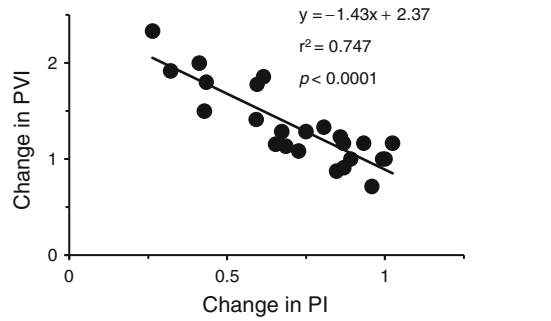

Fig. 2. Correlation between the changes in the PI and the PVI before and at 1 min after skin incision. The changes in the PI and the PVI are represented as the ratio of the PI and the PVI value observed after skin incision to the value observed before skin incision, respectively. PI perfusion index, PVI pleth variability index.

incision and that a significant negative correlation was observed between the changes in the PVI and the PI. Since the skin incisions did not cause bleeding leading to a reduction in the venous return and hypovolemia, the dynamic indicators of fluid responsiveness that are based on the heart–lung interaction should remain constant. In this study, however, the PVI, which was based on the respiratory variations in the PI, was dramatically elevated after the skin incision. The stability of the SVV and CI obtained from the FloTrac™/Vigileo™ system and the heart rate and the absence of a reduction in the MBP in response to the skin incision support the stability of the circulating blood volume. The reason for the elevation in the PVI after the skin incision, despite the absence of a circulating blood volume loss, might not be an increase in the respiratory variation in the PI during the respiratory cycle (i.e., in the respiratory variation in the stroke volume based on the heart–lung interaction), but rather the rapid reduction in the PI that was induced by sympathetic vasoconstriction in response to the nociceptive stimulus of the skin incision, because the PVI is calculated from the difference between the two extreme PI values recorded during a time interval of at least one complete respiratory cycle.

The PI was calculated as the ratio of the AC component to the DC component of the POP signal. The DC component, which is mostly related to the light absorption as a result of tissue, venous blood, and the diastolic volume of the arterial blood, exhibited a slow variation caused by changes in the venous blood volume. On the other hand, the AC component, which is related to the light absorption arising from the pulsating volume of arterial blood, represents the beat-to-beat variation in the POP signal [11, 16]. The POP waveform amplitude (i.e., the AC component) depends not only on the systemic intravascular pulse pressure, but also on the distensibility of the vascular wall, and hence on the sympathetic tone [14, 22]. Therefore, various stimuli, such as surgical stress [10, 11], alpha-adrenergic agents [14], and cold stress [15], cause sympathetic vasoconstriction to reduce the AC component. In addition, sympathetic vasoconstriction also reduces the PI as well as the POP wave amplitude. Judging from a report by Graybeal et al. [23], the

nociceptive stimulus arising from exposure to cold mainly reduces the AC component, leading to a reduction in the PI. In this study, therefore, sympathetic vasoconstriction in response to the skin incision reduced the PI, leading to a dramatic elevation in the PVI. Both the concomitant increase in the MBP and the stability of the SVV after the skin incision support this explanation; however, the stability of the CI seems to be inconsistent with sympathetic vasoconstriction, since sympathetic vasoconstriction of the veins, especially the splanchnic blood vessels, increases the venous return, leading to an increase in the SV and the CI. In this study, the elevation in systemic vascular resistance induced by sympathetic vasoconstriction of the arteries in response to the skin incision might have cancelled the increases in the SV and the CI induced by venoconstriction. If the skin incision increased the venous return, the dramatic elevation in the PVI after the skin incision would have betrayed the inability of the PVI to evaluate fluid responsiveness, since an increase in venous return reduces the PVI. The FloTrac™/Vigileo™ system, which calculates the SV based on an arterial pulse contour analysis, is influenced by factors that affect the arterial blood pressure and the arterial pressure waveform independently of the SV. The SVV might be less affected than the PVI by changes in the vasomotor tone [24]; however, the SV and the SVV measured using the FloTrac™/Vigileo™ system are unreliable among patients undergoing rapid changes in blood pressure as a result of rapid changes in vascular resistance [25]. Judging from the slight elevation in arterial blood pressure in response to the skin incision, the CI and the SVV appeared to be minimally affected by the skin incision. In this study, the fact that a significant negative correlation was observed between the reduction in the PI and the elevation in the PVI after the skin incision strongly supports the explanation that not the increase in respiratory variation in the PI, but rather the sharp reduction in the PI induced by the skin incision caused the dramatic elevation in the PVI. According to data submitted by the Masimo Corporation, the measurement interval for calculating the PVI in our version was more than 60 s, leading to the stronger negative correlation between the changes in the PI and the PVI values observed before and at 1 min after the skin incision, rather than at 5 min after the incision.

Several clinical studies on fluid challenges have focused on dynamic indicators derived from the POP signal to predict fluid responsiveness accurately, compared with other dynamic indicators derived from the arterial pressure waveform, but the results of these studies have been controversial [5–9, 26, 27]. The studies that showed the usefulness of the PVI or respiratory variation in the POP waveform amplitude for evaluating fluid responsiveness were performed over the course of a short experimentation period, in the absence of a nociceptive stimulus, and under general anesthesia or deep sedation (i.e., under conditions in which the vasomotor tone was constant) [5–9]. In the studies by Canneson et al., measurements of the PVI [8, 9] or the respiratory variation in the POP waveform amplitude [7] for evaluating fluid responsiveness were performed before and after a 10-min fluid challenge in patients under general anesthesia before the start of surgery. On the other hand, the studies in which the respiratory variation in the POP waveform amplitude was shown to be a poor predictor of fluid responsiveness were conducted during hepatic surgery [26] or over the course of a long experimentation period that permitted various stimuli to change the vasomotor tone [27]. Based on these results, if the vasomotor tone remains constant during the interval used to calculate the PVI, in other words, if the PVI is calculated under conditions in which the changes in the PI depend only on the changes in the stroke volume induced by mechanical ventilation, the PVI can predict fluid responsiveness accurately. However, other factors besides mechanical ventilation easily affect the PI during clinical practice, since the walls of the cutaneous vessels in the fingers are richly innervated by alpha adrenoceptors and the changes in the POP waveform amplitude of the fingers are more sensitive to sympathetic vasoconstriction than they are in other regions [15, 28]. In the present study, therefore, the skin incision was thought to cause the rapid reduction in the finger PI, resulting in the dramatic elevation in the PVI. Monitoring PI and the PVI at the fingers may be useful not for predicting fluid responsiveness, but rather for indicating the balance between the intensity of the surgical stimuli and the depth of anesthesia. The Surgical Pleth Index, which is calculated based on the POP waveform amplitude and the heart beat intervals, has been proposed as a means to assess the balance between noxious stimulation and the anti-nociceptive effects of anesthesia [29]. To use the PVI as an indicator of fluid responsiveness in a clinical setting, a sufficient depth of anesthesia is needed to maintain a constant vasomotor tone against various stimuli. Furthermore, the fluctuation in the PI during the previous few minutes should also be examined to exclude the possibility of changes in the vasomotor tone. On the other hand, to improve the technical accuracy of the PVI for assessing fluid responsiveness, the measurement interval for calculating the PVI should be shortened to reduce the impact of changes in vasomotor tone on the PI. If possible, adjusting the interval used to calculate the PVI to an interval equivalent to a single respiratory cycle would be a much better way of analyzing the relative changes in the PI induced by mechanical ventilation. The PI trend over several minutes displayed on the pulse oximeter is useful for confirming whether the PVI is calculated from the variation in the PI induced by mechanical ventilation

Table 2. Clinical or technical points for improving the accuracy of the PVI for assessing fluid responsiveness

Clinical
Maintain the depth of anesthesia so as to maintain a constant vasomotor tone
Pay attention to fluctuations in the PI during the previous few minutes
Choose sites of measurement that are insensitive to sympathetic vasoconstriction
Technical
Shorten and adjust the measurement interval for calculating the PVI so that it is equivalent to a single respiratory cycle
Display the PVI trend over several minutes on the pulse oximeter

PI perfusion index, *PVI* pleth variability index.

or by changes in vasomotor tone. A list of clinical or technical points for improving the accuracy of the PVI for assessing fluid responsiveness is shown in Table 2.

Our study had several limitations. The skin incision was thought to have caused sympathetic vasoconstriction, reducing the PI substantially and thereby dramatically elevating the PVI. In this study, however, the hemodynamic parameters before and after the skin incision were not evaluated using gold-standard methods: cardiac output was not measured using the thermodilution method, and the PVI was not compared with the respiratory variation in the arterial pulse pressure, which is considered to be the most sensitive predictor of fluid responsiveness [2]. Therefore, the affect of the skin incision on the circulating blood volume was unclear. However, we do not believe that a skin incision without bleeding would reduce the circulating blood volume. In addition, there was no evidence that the skin incision changed the vasomotor tone; rather, the changes in the vasomotor tone were only inferred based on the changes in the PI. Since nothing other than the skin incision occurred at the start of surgery, there is no doubt that the PI depends on the vasomotor tone. The PVI was recorded at the finger, which is more sensitive to sympathetic vasoconstriction than other regions, and respiratory variations in the POP waveform amplitude depend on the site of measurement. For example, the POP waveform amplitude recorded from the ear is less affected by sympathetic vasoconstriction and is more affected by positive pressure ventilation [30]. Therefore, the PVI recorded at the ear may be useful for assessing fluid status and guiding fluid therapy.

In conclusion, the results of this study showed a significant elevation in the PVI and a negative correlation between the changes in the PVI and the PI, despite the absence of changes in the circulating blood volume, in response to the skin incision. Under conditions in which the PI changes substantially in response to the nociceptive stimulus produced by a skin incision, the PVI should not be calculated from the respiratory variations in the PI, but rather from the variation in the PI induced by the fluctuation in vasomotor tone. When the PVI is used as an indicator for fluid responsiveness, it is crucial to pay attention to fluctuations in vasomotor tone induced by exposure to nociceptive stimuli.

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