Original Article

Accuracy of pulse oximeter perfusion index in thoracic epidural anesthesia under basal general anesthesia

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Received May 8, 2014; Accepted May 23, 2014; Epub July 15, 2014; Published July 30, 2014

Abstract: Objective: To observe the change of PVI after thoracic epidural block on the basis of general anesthesia. Methods: In 26 patients undergoing elective upper abdominal operations, changes of SVI, PVI, SVV, PPV and CVP were monitored immediately before and 10 minutes after T8-9 thoracic epidural anesthesia on the basis of general anesthesia. The definition was that patients with ΔSVI greater than 10% belonged to response group to epidural block. Results: Before epidural block, the PVI, SVV and PPV baseline values in patients of response group were significantly higher than those in patients of non-response group. PVI, SVV and PPV after epidural block were significantly higher than immediately before epidural block (P < 0.001). PVI, SVV and PPV baseline values immediately before epidural block were positively correlated with ΔSVI; the correlation coefficients were 0.70, 0.71 and 0.63, respectively, P ≤ 0.001. The optimal critical values for PVI, SVV and PPV to predict response to T8-9 gap epidural block under general anesthesia were 16% (sensitivity 80%, specificity 92%), 13% (sensitivity 90%, specificity 62%) and 12% (sensitivity 90%, specificity 77%), respectively. Conclusion: PVI can be used as a noninvasive indictor to monitor volume change after thoracic epidural block on the basis of general anesthesia.

Keywords: Pleth variability index, pulse pressure variation, stroke volume variation, combined general, thoracic epidural anesthesia

Introduction

The epidural block combined with general anesthesia can effectively reduce the dose of general anesthetics, and can provide good intraoperative and postoperative analgesia. Thoracic epidural block combined with general anesthesia in major abdominal operations can increase tissue oxygen supply, and reduce postoperative mortality and postoperative complications [1, 2]. However, the epidural block can further aggravate the relative hypovolemia caused by peripheral vascular expansion which general anesthesia leads to, and the incidence of hypotension is relatively high [3]. High volume pre-filling before anesthesia is commonly clinically used to avoid the hypotension, but currently no ideal volume monitoring indictor is used to guide an anesthetist to conduct volume treatment.

Dynamic change of pulse pressure variation (PPV) and stroke volume variation (SVV) under mechanical ventilation can predict the blood volume change of a patient [4-6]. However, these indicators for evaluating volume dynamic changes are invasive and require the special transducer, thus they are not conducive to widely clinical use. Variations of the pulse oximeter plethysmography (ΔPOP) in the respiratory cycle can predict the patient’s hypovolemia, but this change cannot be continuously monitored [7]. Pleth variability index (PVI) is a non-invasive continuous monitoring indicator evolving from the ΔPOP. It has been reported that PVI is a kind of new predictive indicator of volume status and can effectively monitor the dynamic change of blood volume [8-11]. Currently there is no clinical research for PVI to predict volume change under thoracic epidural block combined with general anesthesia. According to changes of CVP, SVV, PPV and PVI before and after epidural block on the basis of general anesthesia, this study wanted to explore clinical application value of PVI under thoracic epidural block combined with general anesthesia.
Table 1. Baseline patient characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>R Group (n = 10)</th>
<th>NR Group (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female</td>
<td>9/1</td>
<td>10/3</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>47.6 ± 9.3</td>
<td>50.5 ± 8.5</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62.27 ± 9.76</td>
<td>61.0 ± 8.92</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.1 ± 4.9</td>
<td>25.1 ± 5.3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167 ± 10.8</td>
<td>166 ± 11.1</td>
</tr>
</tbody>
</table>

Footnotes: Data are expressed as mean ± SD or as frequency distributions (n).

Table 2. Hemodynamic changes at sample points T1 and T2

<table>
<thead>
<tr>
<th>T1</th>
<th>T2</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>66.4 ± 12.5</td>
<td>64.3 ± 12.6</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>88.7 ± 7.7</td>
<td>79.4 ± 9.0</td>
</tr>
<tr>
<td>PVI (%)</td>
<td>10.4 ± 4.0</td>
<td>15.7 ± 5.9</td>
</tr>
<tr>
<td>SVV (%)</td>
<td>9.4 ± 3.2</td>
<td>14.1 ± 4.3</td>
</tr>
<tr>
<td>PPV (%)</td>
<td>8.9 ± 2.8</td>
<td>13.0 ± 3.6</td>
</tr>
<tr>
<td>SVI (ml/each time)</td>
<td>36.9 ± 15.2</td>
<td>33.9 ± 14.1</td>
</tr>
<tr>
<td>PI (%)</td>
<td>3.4 ± 2.3</td>
<td>3.1 ± 2.5</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>10.3 ± 3.5</td>
<td>10.0 ± 2.6</td>
</tr>
</tbody>
</table>

Footnotes: Data are presented as mean ± SD. HR, heart rate; MAP, mean arterial pressure; PVI, pleth variability index; SVV, stroke volume variation; PPV, pulse pressure variation; SVI, stroke volume index; PI, perfusion index; CVP, central venous pressure.

Methods

This study had been approved by the hospital ethics committee and the informed consents of the patients had been obtained. We randomly selected 26 patients undergoing elective upper abdominal operations, who were 20~60 years old, had ASA grade I~II, could belong to either gender, and had body mass index (BMI) 18~30 kg/m². They had no preoperative cardiovascular or respiratory system diseases, and had no contraindications of spinal block, either.

Preoperative routine liquid fasting and fasting were performed, and preoperative medicine was not used. After a patient was transferred to the operation room, oxygen was provided to the patient via the nasal catheter at 2 L/min, and a multi-function monitor (DateX-Ohmeda, USA) was used to continuously monitor the electrocardiogram (ECG), pulse oxygen saturation (SpO2) and non-invasive blood pressure. The forearm peripheral vein (20G indwelling needle) was used to maintain infusion of sodium lactate Ringer’s solution at 2 ml/hour·kg. The patient was in right side-lying position, epidural puncture was performed at the gap between T8 and T9, and the epidural catheter was indwelled. 4 ml of 1% lidocaine as test dose was injected into the catheter, and needle prick was used to measure anesthesia block plane. After segment sensory difference appeared at the block plane and there were no clinical characteristics of subarachnoid block, under local anesthesia the radial artery puncture and right jugular vein puncture were performed. The FloTrac sensor was connected to the FloTrac/Vigileo monitor and the Datex ohmeda multi-function monitor. The Fixed FloTrac sensor was located in the right atrium plane level of the patient, and the Vigileo was used to monitor stroke volume index (SVI) and SVV. The Datex ohmeda multi-function monitor was used to measure heart rate (HR), arterial systolic pressure (SP), arterial diastolic pressure (DP), mean arterial pressure (MAP) and central venous pressure (CVP); pulse pressure variation (PPV) was obtained via calculation [11]. The Masimo Radica-7 monitor (Masimo Corp., Irvine, CA, USA) was connected to the middle finger which was contralateral to the punctured artery; the probe was wrapped and fixed for avoiding light so as to minimize heat dissipation and optical interference, and was used to continuously monitor perfusion index (PI) and PVI.

Midazolam 30 μg/kg, fentanyl 3 μg/kg, etomidate 0.3 mg/kg and cis-atracurium amine 0.15 mg/kg were used to induce anesthesia. After tracheal intubation, the tube was connected to the ventilator, and volume control type intermittent positive pressure ventilation was performed. Tidal volume (Vₜ) was set at 8~10 ml/kg, respiratory rate was set at 12 beats/min, and maintaining end-tidal carbon dioxide partial pressure (PₑCO₂) was at 35~40 mmHg. Anesthesia was maintained by the end-tidal sevoflurane concentration of 1.2 to 1.3%, and the inspired oxygen concentration was 0.5. Five minutes after general anesthesia was performed and blood circulation became stable, 8 ml 2% lidocaine was injected into of the spinal canal. When hypotension emerged during anesthesia (drop of SP ≥ 25% of baseline or SP value < 80 mmHg), 5 mg~10 mg ephedrine was diluted and then slowly intravenously injected and blood volume was supplemented and corrected; at last, all patients belonging to this kind were excluded from statistics.
HR, MAP, CVP, SVI, SVV, PI, PVI and PPV were recorded immediately (T1) before and 10 minutes (T2) after epidural administration. Greater than 10% drop of SVI without hypotension during anesthesia was defined as response to T8-9 gap epidural block (group R: responders); smaller than 10% drop was defined as nonresponse to T8-9 gap epidural block (group NR) [12].

MedCalc13.0 software was used to perform statistical analysis. Paired t test was used to compare hemodynamic indicators immediately before and after epidural administration. Mann-Whitney U test was used to compare hemodynamic indicators between group R (responders) and group NR after epidural block. \( P < 0.05 \) was considered statistically significant.

\[ \Delta \text{SVI} \] was percentage of drop of SVI 10 minutes (T2) after epidural block compared with SVI immediately (T1) before epidural block. Linear regression analysis was used to analyze the correlation between \( \Delta \text{SVI} \) and PVI, SVV, PPV, CVP immediately (T1) before epidural block. Drop of SVI greater than 10% was defined as positive indicator; the receiver operator characteristic (ROC) curve was used to calculate the areas under PVI, SVV, PPV and CVP curves, the sensitivity of the optimal critical value, specificity, and 95% confidence interval. We evaluated the feasibility of the issue that PVI predicted PVI, SVV and PPV baseline values in group R before T8-9 gap epidural block on the basis of general anesthesia were significantly higher than in group NR. MAP, PVI, SVV, PPV and SVI after epidural block (T2) significantly changed compared with those before epidural block (T1); there were no statistically differences between PI, HR, CVP after epidural block (T2) and those before epidural block (T1) (Table 2, Figure 1).

After epidural block on the basis of general anesthesia, patients who had \( \Delta \text{SVI} \) drop greater than 10% and had no hypotension was a responder to epidural block. The results displayed that the areas under ROC curve of PVI, SVV, PPV and CVP were 0.82, 0.86, 0.86 and 0.58, respectively. There were no differences among areas under ROC curve of PVI, SVV, PPV, and they all could effectively predict volume response after epidural anesthesia on the basis of general anesthesia. The optimal criti-
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![Graphs showing regression analysis](image)

**Figure 2.** Linear regression analysis between the baseline (T1) values of (A) pleth variability index, (B) stroke volume variation, (C) pulse pressure variation and (D) central venous pressure and the percentage value of changes in stroke volume index following epidural anesthesia. The solid lines represent the regression line. PVI, pleth variability index; ΔSVI, changes in stroke volume index; SVV, stroke volume variation; PPV, pulse pressure variation; CVP, central venous pressure.

...cal values for PVI, SVV and PPV to predict response to T8-9 gap epidural block under general anesthesia were 16% (sensitivity 80%, specificity 92%), 13% (sensitivity 90%, specificity 62%) and 12% (sensitivity 90%, specificity 77%), respectively. Area under CVP curve (0.58) was smaller than area under PVI curve, area under SVV curve and area under PPV curve; the optimal critical value was 8mmHg (sensitivity 40%, specificity 85%) (Figure 3 and Table 3).

**Discussion**

This study has showed that PVI, SVV and PPV can predict change of blood volume under thoracic epidural block combined with general anesthesia. After thoracic epidural block, there is no significant correlation between CVP and stroke volume index change, thus it cannot effectively predict the dynamic change of blood volume after epidural block. Studies have shown that during a high-risk operation, fluid optimization treatment to achieve minimum ΔPP (pulse pressure) can effectively reduce operative complications and costs [13]. Thus, it has an extremely important clinical value to effectively monitor volume dynamic change after thoracic epidural block under general anesthesia so as to guide fluid optimization treatment.

Respiratory variations in the pulse oximeter plethysmography (ΔPOP) under mechanical ventilation can reflect the change of ventricular preload well [14], and PVI is the variability of pulse perfusion index (PI) calculated on the basis [12]. In addition, intrathoracic pressure change during positive pressure ventilation can cause change of SV during a certain period by impacting returned blood volume. SVV is variability of SV calculated by the formula SVV (%) = (SV max - SV min)/SV mean. Similarly, arterial pressure (SP, DP) measured by radial artery puncture can be used to calculate PPV.
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Patients in group R. Epidural anesthesia might cause different cardiovascular responses due to different basic blood volumes of patients, suggesting that the patients in group NR had relatively abundant preoperative blood volume thus vascular dilatation caused by epidural block did not significantly lower blood pressure. However, due to the preoperative fasting, the primary diseases and other reasons, most patients were in a low-volume status before operation, thus epidural anesthesia could cause serious cardiovascular responses [17, 18]. We also found that 5 minutes after the start of epidural anesthesia the patients’ MAP and SV decreased significantly and reached the peak 10 minutes after the start of epidural anesthesia; therefore, we chose to monitor various indicators 10 minutes after the start of epidural anesthesia; we found that 10 minutes (T2) after the start of epidural anesthesia, PVI, SVV and PPV were significantly higher than before block (T1) and CVP did not significantly

Numerous studies have demonstrated that in the case of mechanical ventilation, dynamical hemodynamic parameters, i.e., PVI, SVV and PPV, which depend on the interaction between intrathoracic pressure and returned blood volume, are good predictors of the volume response [15]; there is significant correlation among PVI, SVV and PPV [16]. Our study has not only confirmed this correlation, but also has found good correlation between them and volume change after thoracic epidural block under light general anesthesia.

What we have studied is blood volume effect of epidural anesthesia on the basis of general anesthesia. Epidural block changes sympathetic tone of cardiovascular autonomic nervous system, and autonomic nervous system regulation works only within a certain range, therefore, this study performed T8–9 gap epidural injection of 8ml 2% lidocaine to cause epidural nerve block and to block thoracic sympathetic nerves, leading to thoracic and abdominal vascular dilatation. As a result, relative blood volume of a patient changes significantly. For example, 3 cases was excluded from statistics because after T8-9 gap epidural block their great blood pressure drop, which was caused by relative hypovolemia, reached set treatment standards and after blood volume expansion and ephedrine treatment their conditions became stable. 23 patients met the designed criteria. Drop of SVI greater or smaller than 10% was defined as response or non-response to T8-9 gap epidural block [12]. Statistics found that 13 patients were unresponsive to epidural block, and before block their PVI was 12.2% ± 5.0%, SVV was 11.8% ± 3.4% and PPV was 11.1% ± 3.2%. These indicators were significantly lower than those of the patients in group R. Epidural anesthesia might cause different cardiovascular responses due to different basic blood volumes of patients, suggesting that the patients in group NR had relatively abundant preoperative blood volume thus vascular dilatation caused by epidural block did not significantly lower blood pressure. However, due to the preoperative fasting, the primary diseases and other reasons, most patients were in a low-volume status before operation, thus epidural anesthesia could cause serious cardiovascular responses [17, 18]. We also found that 5 minutes after the start of epidural anesthesia the patients’ MAP and SV decreased significantly and reached the peak 10 minutes after the start of epidural anesthesia; therefore, we chose to monitor various indicators 10 minutes after the start of epidural anesthesia; we found that 10 minutes (T2) after the start of epidural anesthesia, PVI, SVV and PPV were significantly higher than before block (T1) and CVP did not significantly
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Table 3. Area under the ROC curves and cut-off values

<table>
<thead>
<tr>
<th></th>
<th>AUC</th>
<th>Standard error</th>
<th>Lower limit (95% CI)</th>
<th>Upper limit (95% CI)</th>
<th>P</th>
<th>Cut-off</th>
<th>Sensitivity (%) (95% CI)</th>
<th>Specificity (%) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVI</td>
<td>0.82</td>
<td>0.104</td>
<td>0.60</td>
<td>0.95</td>
<td>&lt; 0.001</td>
<td>16%</td>
<td>80 (44-98)</td>
<td>92 (64-100)</td>
</tr>
<tr>
<td>SVV</td>
<td>0.85</td>
<td>0.077</td>
<td>0.65</td>
<td>0.97</td>
<td>&lt; 0.001</td>
<td>13%</td>
<td>90 (56-100)</td>
<td>62 (32-86)</td>
</tr>
<tr>
<td>PPV</td>
<td>0.86</td>
<td>0.079</td>
<td>0.65</td>
<td>0.97</td>
<td>&lt; 0.001</td>
<td>12%</td>
<td>90 (56-100)</td>
<td>77 (46-95)</td>
</tr>
<tr>
<td>CVP</td>
<td>0.58</td>
<td>0.13</td>
<td>0.36</td>
<td>0.78</td>
<td>= 0.555</td>
<td>8 mmHg</td>
<td>40 (12-74)</td>
<td>85 (55-98)</td>
</tr>
</tbody>
</table>

Footnotes: Areas under the receiver operating characteristic (ROC) curves and cut-off values to discriminate between epidural anesthesia responders and nonresponders for pleth variability index (PVI), stroke volume variation (SVV), pulse pressure variation (PPV) and central venous pressure (CVP). AUC, area under the receiver operating characteristic curve; CI, confidence interval.

change. Under ROC curve, area of CVP (0.58) was significantly less than area of PVI (0.82), area of SVV (0.86) and area of PPV (0.86), confirming that PVI, SVV and PPV can effectively predict hypovolemia after thoracic epidural block; the optimal cut-off values were 16%, 13% and 12%, respectively. Change of CVP is affected by vascular volume, vascular tone, cardiac function, intrathoracic pressure and other factors, and as opposed to dynamic cardiac preload indicators, CVP cannot effectively predict hypovolemia caused by epidural block [19]. These have also showed that dynamic cardiac preload indicators more effectively reflect the change of blood volume after thoracic epidural block.

SVV is a fixed correction constant under the assumption that blood flow of the radial artery is 1% of cardiac output; in the case of normal perfusion, SVV is closely related to blood volume, but in the case of low or high perfusion, SVV cannot accurately reflect a patient’s blood volume. PPV is a dynamic blood volume monitoring indicator, which is defined as the variability of the difference between systolic and diastolic blood pressures in a respiratory cycle during mechanical ventilation. PPV is significantly superior to central venous pressure and other traditional indicators in predicting blood volume response, and its shortcoming is that a monitor cannot visually display it, causing the negative influence of its clinical application [20, 21]. PVI is calculated on the basis of perfusion index (PI). Vascular contraction affects PI, thus further affects PVI [22]. Some studies also think that scratch pain stimulation can cause PVI rise [23], therefore, when we use PVI to predict hypovolemia after epidural block on the basis of general anesthesia, should exclude other factors affecting PVI.

Thoracic epidural anesthesia combined with general anesthesia is widely used for major upper abdominal operations, and significantly impacts the blood circulation, causing relative hypovolemia. PVI is a more objective indicator for monitoring volume, and there is good correlation between it and SVV, PPV in blood volume monitoring; in clinical anesthesia monitoring, PVI can continuously non-invasively effectively predict hypovolemia after epidural block. Meanwhile, like other indicators, PVI should be used to perform comprehensive judgment combined with clinical manifestations. The study results have showed that PVI can be used as a non-invasive indicator of blood volume change after thoracic epidural anesthesia on the basis of general anesthesia, and can be used to guide us to optimize volume treatment for reducing operative complications.

Acknowledgements

This work was sponsored by the Science and Technology Support Program from the Science and Technology Commission of Shanghai Municipality, China (124119a3400).

Disclosure of conflict of interest

None.

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Reference

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