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ORIGINAL ARTICLE

Assessment of cardiac preload status by pulse pressure variation in patients after anesthesia induction: comparison with central venous pressure and initial distribution volume of glucose

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Abstract
Purpose Recognition of intraoperative hypovolemia is important for fluid management. Previous studies demonstrated functional preload parameter pulse pressure variation (PPV) could predict volume changes in response to fluid loading and loss. In this study, we examined the correlation between PPV and other two cardiac preload indicators, central venous pressure (CVP) or initial distribution volume of glucose (IDVG), in patients after anesthesia induction.
Methods In 30 patients undergoing scheduled craniotomy surgery, we compared measurement of PPV (%) using the Ohmeda monitor method to simultaneously measure CVP and IDVG after anesthesia induction through correlation analysis and receiver operating characteristic (ROC) curves.
Results Pulse pressure variation has negative linear correlation with IDVG ($r = -0.65$, $P < 0.01$). IDVG values ($n = 13$) when PPV $\geq 11\%$ showed a significant difference compared with those ($n = 17$) when PPV $< 11\%$ ($P < 0.001$). The ROC curve showed the best cutoff value of IDVG is 122 ml/kg, equivalent to the threshold of PPV (11%) for predicting fluid responsiveness. However, there is no significant correlation between CVP in normal ranges (4–9 mmHg) and PPV ($r = -0.12$, $P > 0.05$).
Conclusion As an indicator of cardiac preload, PPV has a negative linear correlation with IDVG in patients after anesthesia induction. It does not correlate well with CVP in the normal range. Our results imply that an individual PPV, not CVP, is equivalent to IDVG in assessing volume status after induction.

Keywords Hypovolemia · Pulse pressure variation · Initial distribution volume of glucose · Central venous pressure

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Introduction

Accurate assessment of cardiac preload status is important to avoid hypovolemia or fluid overload and to optimize intraoperative fluid therapy. However, it seems difficult to know the exact intravascular volume because measurement of absolute blood volume, which is based on the indicator dilution principle, is not readily available in the clinical setting. The measurement of central venous pressure (CVP) is the most commonly used and clinically accepted method for assessing the volume status or cardiac preload [1]. The initial distribution volume of glucose (IDVG), representing central extracellular fluid (ECF) volume, is another tool for assessment of cardiac preload. Some studies indicated IDVG had closer correlation with cardiac output (CO) than plasma volume determined by the indocyanine green dilution method [2], and this method has been successfully used for prediction of postoperative hypovolemia [3, 4].

The functional hemodynamic parameters now are regarded as better predictors in terms of fluid responsiveness compared to those static indices, including CVP in patients under mechanical ventilation [5, 6]. Recent evidence is mounting that pulse pressure variation (PPV) or stroke volume variation (SVV) is a sensitive and reliable indicator of intravascular volume or preload status [7, 8]. However, the relationship between PPV with CVP or

IDVG as preload indicators has not been studied so for. We therefore examined whether PPV obtained from the Datex method can correlate with two indicators of cardiac preload, CVP and IDVG, in patients after anesthesia induction.

Methods

Subjects

After the approval of the institutional ethics committee for human studies and personal informed consent were obtained, 30 American Society of Anesthesiologists (ASA) grade I and II patients undergoing elective craniotomy were included in the study. Those patients with diabetes mellitus, cardiopulmonary dysfunction, liver or renal dysfunction, significant arrhythmia, or extensive peripheral arterial occlusive disease were excluded.

Sample size determination

To achieve a power above 90% for investigation of the correlation between PPV and CVP, or PPV and IDVG, we aimed for analysis of at least 24 patients, which criterion was reached after inclusion of 30 patients in this study.

Anesthesia induction and monitoring

Surgery was preceded by an 8-h fasting period. Upon arrival in the operating room, noninvasive arterial blood pressure, electrocardiogram, and pulse oximetry were monitored in all patients (Datex Ohmeda S/5, Helsinki, Finland).

Anesthesia was induced with intravenous (IV) midazolam (0.04 mg/kg), target control infusion (TCI) propofol (plasma concentration, 4.0 μg/ml), and fentanyl (3 μg/kg). Succinylcholine (2 mg/kg) IV was used to facilitate tracheal intubation. All participants' lungs were ventilated in volume-controlled mode (Drager Julian, Philips Healthcare, The Netherlands) with a tidal volume of 8–10 ml/kg at a frequency of 10 bpm and zero end-expiratory pressure. Anesthesia was maintained with propofol TCI (3–4 μg/ml) in O_2 and continuous infusions of vecuronium (1 μg/kg/h). The ventilator settings were unchanged during the study. Patients whose peak airway pressures exceeded 40 cm H_2O were excluded from the study [9]. All patients received a crystalloid solution (Plasmalyte A; Baxter International, Deerfield, IL, USA) with limited infusion rate (1 ml/kg/h) before and during the induction period. A phenylephrine bolus (40–80 μg) was administrated for increasing blood pressure if systolic blood pressure was less than 90 mmHg. All variables obtained during an unstable hemodynamic state and immediately after an IV bolus of vasoactive agent or anesthetics were excluded.

Invasive hemodynamic monitoring was initiated after the induction of anesthesia. Invasive arterial blood pressure monitoring was established via a 20 G catheter inserted in the radial artery. A double-lumen central venous catheter (CV-17702; Arrow International, Reading, PA, USA) was inserted in the right internal jugular vein. All monitoring transducers were positioned and zeroed at the midaxillary level. A fast flush test was performed. If the test revealed unacceptable pressure recording as defined elsewhere, the data registration was excluded. Heart rate, mean arterial pressure (MAP), and CVP were continuously monitored and recorded during the study period.

Pulse pressure variation measurement

After induction and hemodynamic stabilization, the measurement of PPV was instituted, using simple tools on the Datex Ohmeda S/5 as described in our previous study [10]. Briefly, the "PA" and "wedge pressure" scales to record arterial and pulse pressure were changed as follows. Label the systemic arterial blood pressure curve as "pulmonary arterial pressure" and change the scale accordingly. In the wedge pressure menu, the screen will freeze and a horizontal line will appear. It can now be freely moved to the uppermost point of the systolic pressure curve, and then down to the lowest systolic pressure. The corresponding diastolic pressures and pulse pressures are also recorded. PPV (%) were then calculated using the following formulas:

$$\mathrm{PPV}(\%) = 200 \times (\mathrm{PP}_{\max} - \mathrm{PP}_{\min})/(\mathrm{PP}_{\max} + \mathrm{PP}_{\min})$$

where $\mathrm{PP}_{\max}$ and $\mathrm{PP}_{\min}$ are the maximal and minimal values within one respiratory cycle. PPV (%) was calculated in triplicate over three consecutive respiratory cycles. The mean value of the three determinations was used for analysis.

Initial distribution volume of glucose measurement

Immediately after the PPV value was obtained, 10 ml 50% glucose (5 g) was injected through the internal jugular vein within 30 s to measure initial distribution volume of glucose (IDVG). Arterial blood samples were drawn to allow plasma glucose concentrations to be measured immediately before and at 1, 3, 5, and 60 min post infusion. Plasma glucose concentrations and electrolytes were measured via a combined blood gas and glucose analyzer (ABL 800 FLEX; Radiometer Medical, Denmark) immediately after blood sampling. According to the previously described method [11], IDVG was calculated using a one-compartment model from the increased plasma glucose

concentrations between 3 and 7 min. In this study, values at 3 min post injection for approximated IDVG determination were used [12]. Approximated IDVG is calculated as follows:

$$\mathrm{IDVG}(L) = 24.4 \times \exp(-0.03 \times \Delta\mathrm{gl}) + 2.7$$

where exp is the exponential function and Δgl = incremental glucose level at 3 min after intravenous glucose injection. The IDVG (ml/kg) is presented based on the basal body weight before anesthesia.

Statistic analysis

Numerical data are expressed as mean ± standard deviation (SD). Comparison analysis was performed using Student's *t* test, and correlation analysis was achieved using a Pearson test or Spearman's rho test when necessary. The correlation between IDVG (ml/kg) and PPV (%) was determined using a least squares regression technique to find the line of best fit. To calculate the comparable threshold values of IDVG or CVP, PPV = 11% as threshold of hypovolemia is selected. The receiver operating characteristic (ROC) curves are used to determine the most discriminating threshold using the following equation: $(1 - \text{specificity})^2 + (1 - \text{sensibility})^2$. Statistical analysis was performed with SPSS 15.0 statistics software (SPSS, Chicago, IL, USA). The level of statistical significance was $P < 0.05$.

Results

Thirty neurosurgical patients undergoing craniotomies were enrolled. None of the patients was excluded from the study. The demographic characteristics and preoperative diagnosis of participants are shown in Table 1. The CVP values in all patients after anesthesia induction were in the normal range (4–9 mmHg). Their heart rate, MAP, and CVP remained virtually unchanged before and 5 min after 5-g glucose injection through the internal jugular vein (Table 1, $P > 0.05$).

Arterial blood glucose levels before and 1, 3, and 5 min after 5-g glucose injection were 5.39, 9.72, 8.61, and 8.18 mmol/l, respectively (Fig. 1). Blood glucose levels 60 min (5.31 mmol/l) after 5-g glucose injection had returned to preinjection levels (Fig. 1, $P > 0.05$).

The scatter plot figures demonstrated the correlation among PPV, IDVG, and CVP. A significant negative correlation was found between PPV (Datex derived parameter) and IDVG (3 min post injection of glucose): correlation coefficient (r) = −0.65 (Fig. 2); $P < 0.01$; 95% confidence interval, −0.76 to −0.61. However, there is no correlation between PPV and CVP ($r = -0.12$, $P > 0.05$).

The ROC curve was drawn (Fig. 3), and the best cutoff value of IDVG was 122 ml/kg (sensitivity = 82.4%; specificity = 100%) if 11% is the threshold of the PPV value for evaluating whether preload volume is inadequate. The comparison of IDVG values between patients with PPV ≥ 11% and those with PPV < 11% showed a significant difference, although not for CVP values (Table 2).

Discussion

Insufficient intravascular volume can lead to cardiac preload decrease and therefore to tissue hypoperfusion and cellular oxygenation impairment [13]. The ability to recognize hypovolemia or reduced cardiac preload continues to be a challenge because of blood loss, alteration of vasomotor tone, and capillary leak during surgery [14]. Administering fluids is the first line of therapy in hypovolemia hypotension to augment and improve patient hemodynamics. In this study, we found that IDVG has a negative linear correlation with PPV (%) ($r = -0.65$, $P < 0.001$), and can be a discriminator of hypovolemia, but CVP, representing right ventricular filling pressure, does not. The best cutoff value of IDVG equivalent to the threshold of PPV (11%) for predicting hypovolemia is 122 ml/kg.

Prolonged preoperative fasting does not result in reduction of volume status in surgical patients [15]. For post-induction patients, decrease in intrathoracic blood volume (ITBV) may be the result of the vasodilatory effects of general anesthesia or the increase of intrathoracic pressure by mechanical ventilation. Brock et al. [16] demonstrated that pre-induction ITBV of 23.3 ± 1.8 ml/kg changed to 19.3 ± 1.6 ml/kg after induction of anesthesia. It is possible that central extracellular fluid shifts from the central to peripheral compartments during the anesthesia induction, which presents as relative hypovolemia without any apparent volume loss.

CVP is one of the clinically common hemodynamic indices. Its accuracy to reflect cardiac preload is critically dependent on the presence of normal cardiopulmonary conditions. Basically, volume status usually can be detected by trend tracking of CVP but not a single-point CVP value, which may result in delay in identification of smaller volume depletion or volume overload in either critically ill patients [17, 18] or normal subjects [19]; this can be explained from the standpoint of the limited reliability of cardiac filling pressures to estimate intravascular volume status. For example, there is a poor relationship between CVP and right ventricular end-diastolic volume [20]. Interestingly, none of the 30 patients would be diagnosed as hypovolemic because their CVP values were greater than 4 mmHg. Actually, normal CVP value does not mean

Table 1 Patient demographics and monitoring parameters after anesthesia induction

Characteristics/parameters	Value
Number of patients	30
Age (years)	45.1 ± 15.6
Gender (male/female)	11/19
Height (cm)	163.5 ± 8.1
Weight (kg)	60.3 ± 10.8
ASA physical status (I/II)	24/6
Diagnosis of intracranial lesion	
Meningioma	20 (67.0%)
Glioma	5 (17.0%)
Neurinoma	3 (10.0%)
Hemangioblastoma	2 (6.0%)
HR (bpm)	
Pre-injection	75.3 ± 12.5
Post-injection	73.5 ± 15.7
MAP (mmHg)	
Pre-injection	58.5 ± 13.9
Post-injection	60.2 ± 9.4
CVP (mmHg)	
Pre-injection	7.0 ± 1.4
Post-injection	7.0 ± 1.5

Values are presented as mean ± standard deviation or as number of patients (percentage)

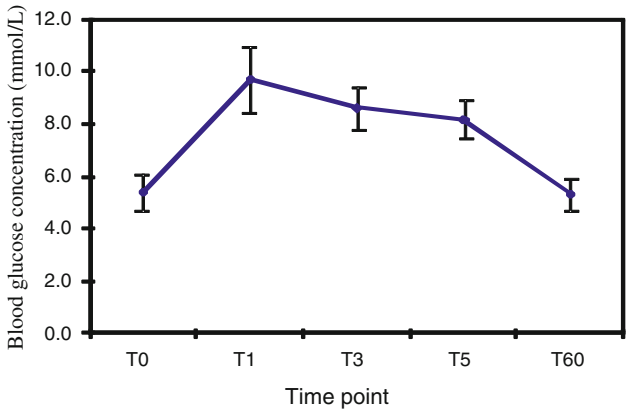

Fig. 1 Blood glucose concentrations before (T_0) and 1, 3, 5, or 60 min (T_1, T_3, T_5, T_{60}) after intravenous 5 g glucose injection in patients ($n = 30$)

normal intravascular volume status. Pestel et al. [7] indicated CVP value did not differ from its baseline even with withdrawal of 30% estimated blood volume in an experimental hypovolemic pig model. However, the difference in pulse pressure (%) correlates well with graded blood loss and seems to be a sensitive indicator of hypovolemia. Our failure to demonstrate a relationship between CVP in normal range and PPV raises the question of statistical

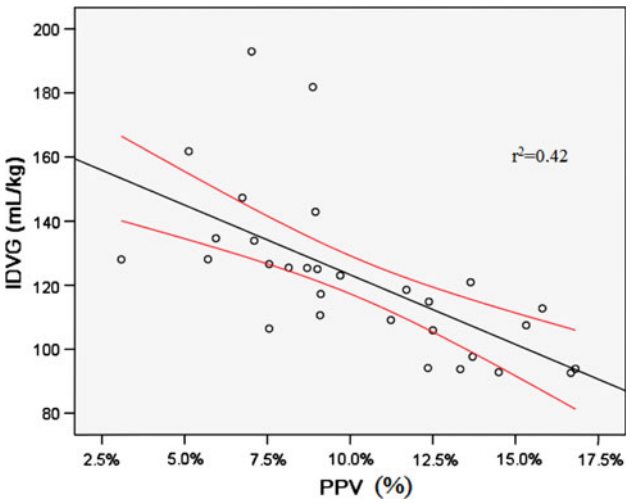

Fig. 2 Relationship between the initial distribution volume of glucose (*IDVG*) using approximated method and PPV (%) obtained from Detax method in 30 patients after anesthesia induction. *Straight line* line of best fit showing a significant negative linear correlation between PPV (%) and IDVG [correlation coefficient (r) = −0.65, $P < 0.001$]; *Curved lines* 95% confidence interval for the predicted IDVG for any given PPV (%)

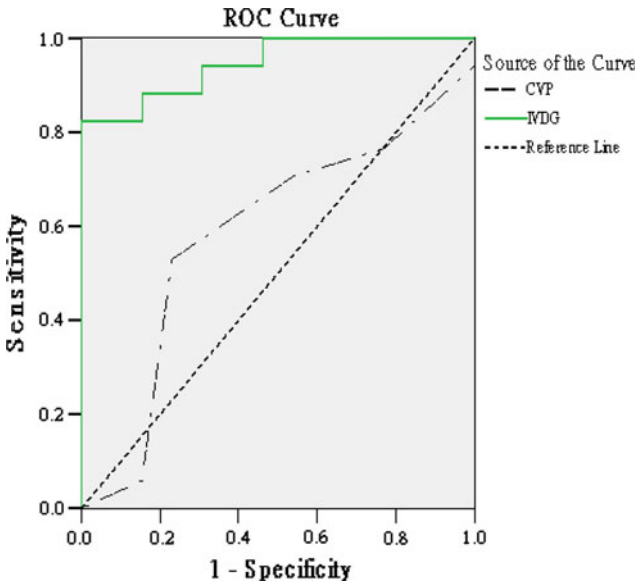

Fig. 3 Receiver operating characteristic (*ROC*) analysis for initial distribution volume of glucose (*IDVG*) and central venous pressure (*CVP*). The area under the IDVG (ml/kg) ROC curve was 0.946 ($P < 0.001$; 95% confidence interval, 0.871–1.021), and the area under the CVP (mmHg) ROC curve was 0.584 ($P = 0.439$; 95% confidence interval, 0.369–0.798)

power. Post hoc power calculations indicate that our sample size of 30 provides 99% power to detect correlation at $r^2 = 0.01$ ($P = 0.594$) between CVP and PPV. Independent statistical analysis would also indicate if there was a relationship between CVP and PPV (%) within a wider population; this relationship would be small.

Table 2 Comparison of CVP and IDVG values between PPV ≥ 11% and PPV < 11%

Preload indicators	PPV ≥ 11% ($n = 13$)	PPV < 11% ($n = 17$)	P value
CVP (mmHg)	6.7 ± 1.4	7.0 ± 1.5	0.562
IDVG (ml/kg)	104.2 ± 10.5*	135.9 ± 23.4	<0.001

* $P < 0.001$, compared with group PPV < 11%

PPV pulse pressure variation, *CVP* central venous pressure, *IDVG* initial distribution volume of glucose

PPV is a functional preload parameter defined as the maximal pulse pressure less the minimum pulse pressure divided by the average of these two pressures [21]. It also can be used for assessment of cardiac preload similar to analogous variable SVV [22, 23] or systolic pressure variation (SPV) [24, 25]. Calculation of PPV has repeatedly been shown to be a reliable predictor of fluid responsiveness or of the position on the preload–stroke volume relationship (Frank–Starling curve) in mechanically ventilated patients [26]. Therefore, PPV is believed to be a validated indicator of hypovolemia [27]. However, measurement of PPV requires expensive instruments (for example, PiCCO system) [5, 6] or specific software systems [28], which limits routine intraoperative application of this method. A bedside PPV measurement via clinically commonly used multiparameter monitor has been described [29] and used easily and continuously for intraoperative fluid therapy [30]. Other sources [9] and our studies [10] have demonstrated that PPV derived from the Detax method could reliably predict fluid responsiveness during graded fluid loss or loading. Therefore, it appears that the PPV value may change with intravascular fluid loss or gain, which implies the magnitude of PPV is linked to the degree of hypovolemia in mechanically ventilated patients. This PPV monitoring has advantages over other monitoring techniques because it is simple and is not associated with additional costs or complications beyond arterial cannulation.

Blood volume analysis provides information on intravascular circulating volume. IDVG is a measure of the central extracellular compartmental volume that is independent of the plasma glucose values present before glucose injection or infusion of insulin and/or vasoactive drugs [11, 31]. In contrast to circulating blood volume, IDVG in fact plays a key role in determining central blood volume regardless of redistribution of blood volume between the central and peripheral compartments. Recent findings have showed IDVG to be highly correlated with either cardiac output [32] or intrathoracic blood volume (ITBV) [33] in clinical and experimental studies. Although not widely accepted, IDVG has been reported to predict postoperative hypovolemia hypotension in patients undergoing abdominal aortic surgery [4] and esophagectomy [3, 34], but not cardiac surgery [35]. This disparity in results may come from a different understanding of IDVG but is not attributable to methodological flaws of IDVG determination [36]. In our study, the maximum IDVG was 193 ml/kg, whereas the minimum was 92.6 ml/kg. Hence, high consistency was apparent within the normal clinical range, not only at low blood pressures. It is reported that the threshold value of PPV, which enables fluid responsiveness to be predicted, ranges from 9% to 17% [37]. If we take PPV = 11% as obtained from our previous result [10] to calculate the comparable IDVG value, the threshold of IDVG to predict post-induction hypovolemia is 122 ml/kg, which is consistent with the value (110–130 ml/kg) reported in a previous study [3].

There are some limitations in our study. The first and most important is that we did not measure circulating blood volume using the radioactive-labeled iodine technique as a "gold standard" [38]. This technique is not available in our hospital, so hypovolemia is difficult to define. In this study, the comparable value of IDVG can be calculated when we using the thresholds of PPV (11%), a validated predictor of fluid responsiveness, although an absolute PPV value to predict hypovolemia has not been established. Second, we did not manipulate intravascular volume to observe whether this linear relationship remains after fluid load or loss. Because of possible hyperglycemia in neurosurgical patients and the reproducibility of IDVG, the measurement would be of concern. van Tulder et al. [39] found that IDVG seems inadequate to assess individual response to a fluid challenge, although the bias of IDVG measurements was only 0.08 ± 0.32 l at a 30-min interval without fluid infusion in another study [40].

In conclusion, PPV has a negative linear correlation with IDVG in patients after anesthesia induction. It does not correlate well with CVP in the normal range. Thus, our results imply that an individual PPV, not CVP, is equivalent to IDVG in assessing volume status after induction.

Conflict of interest None.

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