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Variation of left ventricular outflow tract velocity and global end-diastolic volume index reliably predict fluid responsiveness in cardiac surgery patients☆

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Abstract

Purpose: The ability of the global end-diastolic volume index (GEDVI) and respiratory variations in left ventricular outflow tract velocity (ΔVTI_{LVOT}) for prediction of fluid responsiveness is still under debate. The aim of the present study was to challenge the predictive power of GEDVI and ΔVTI_{LVOT} compared with pulse pressure variation (PPV) and stroke volume variation (SVV) in a large patient population.

Material and Methods: Ninety-two patients were studied before coronary artery surgery. Each patient was monitored with central venous pressure (CVP), the PiCCO system (Pulsion Medical Systems, Munich, Germany), and transesophageal echocardiography. *Responders* were defined as those who increased their stroke volume index by greater than 15% (ΔSVI_{TPTD} >15%) during passive leg raising.

Results: Central venous pressure showed no significant correlation with ΔSVI_{TPTD} ($r = -0.06$, $P = .58$), in contrast to PPV ($r = 0.71$, $P < .0001$), SVV ($r = 0.61$, $P < .0001$), GEDVI ($r = -0.54$, $P < .0001$), and ΔVTI_{LVOT} ($r = 0.54$, $P < .0001$). The best area under the receiver operating characteristic curve (AUC) predicting ΔSVI_{TPTD} greater than 15% was found for PPV (AUC, 0.82; $P < .0001$) and SVV (AUC, 0.77; $P < .0001$), followed by ΔVTI_{LVOT} (AUC, 0.74; $P < .0001$) and GEDVI (AUC, 0.71; $P = .0006$), whereas CVP was not able to predict fluid responsiveness (AUC, 0.58; $P = .18$).

Conclusions: In contrast to CVP, GEDVI and ΔVTI_{LVOT} reliably predicted fluid responsiveness under closed-chest conditions. Pulse pressure variation and SVV showed the highest accuracy.

1. Introduction

Several studies have shown that an individually titrated and appropriate fluid administration is a prerequisite for an adequate cardiac index (CI) and organ perfusion [1]. Goal-directed fluid therapy may reduce morbidity after major

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surgery and the length of stay on the intensive care unit (ICU) [2] by avoiding inappropriate fluid replacement or unnecessary application of vasopressors.

In the past, fluid therapy was commonly directed by static pressure–derived variables such as central venous pressure (CVP) or pulmonary artery occlusion pressure; however, numerous studies demonstrated static variables to be poor predictors of the response to a volume challenge [3]. More recently, dynamic ventilation-induced variables such as pulse pressure variation (PPV) and stroke volume variation (SVV) have achieved considerable interest regarding fluid therapy; and their accuracy and limitations have been demonstrated repeatedly in various patient populations [4-7]. In this context, the global end-diastolic volume index (GEDVI) as a static volumetric variable has been shown in small studies to accurately reflect preload and to be less susceptible to confounding variables such as intraabdominal hypertension; but its feasibility to predict fluid responsiveness is still under debate [8-12]. Furthermore, several studies showed good correlation between fluid responsiveness and ventilation-induced cyclic changes in aortic blood flow velocity (ΔVTI_{Ao}) or the velocity time integral obtained in the left ventricular outflow tract (ΔVTI_{LVOT}) [13-15]. The ability of ΔVTI_{LVOT} to predict the hemodynamic response to volume expansion, however, has never been challenged in a large-scale trial.

The aim of the present study was to investigate the ability of ΔVTI_{LVOT} and GEDVI to predict a percentage change greater than 15% in stroke volume index by transpulmonary thermodilution (ΔSVI_{TPTD}), compared with PPV and SVV, in a large patient population undergoing cardiac surgery. We hypothesized that GEDVI and ΔVTI_{LVOT} were both able to reliably predict fluid responsiveness.

2. Material and methods

The study was approved by the institutional review board (Christian-Albrechts University, Kiel, Germany), and all patients gave informed consent for participation in the study. Ninety-two patients undergoing elective coronary artery bypass grafting were studied before surgery after induction of general anesthesia. Patients older than 18 years and with a left ventricular ejection fraction of at least 0.5 fulfilled the inclusion criteria. Exclusion criteria were as follows: emergency procedures, hemodynamic instability requiring pharmacologic support, ongoing arrhythmia, intracardiac shunts, severe aortic or mitral stenosis or insufficiency, aortic aneurysm greater than 4 cm, and the use of an artificial left ventricular assist device or intraaortic balloon pump.

2.1. Instrumentation and protocol

All patients received premedication with midazolam 7.5 mg per os. After induction of anesthesia with sufentanil (0.5 μg/kg) and propofol (1.5 mg/kg), orotracheal intubation was facilitated with rocuronium (0.6 mg/kg). Anesthesia was maintained with sufentanil (1 μg/[kg h]) and propofol (3 mg/[kg h]), and patients were ventilated with an oxygen/air mixture and in volume-controlled mode using a tidal volume of 8 mL/kg related to the ideal body weight. Positive end-expiratory pressure was set at 5 cm H_2O. Continuous monitoring was performed including electrocardiogram, radial arterial pressure catheter, and a central venous catheter in the right or left internal jugular vein. Subsequently, a transpulmonary thermodilution catheter was placed in the femoral artery and was connected to a PiCCOplus monitor (PiCCOplus, Version 6.0; Pulsion Medical Systems, Munich, Germany). In addition, capnography, urine output, temperature (blood, bladder, and nasopharyngeal), airway pressure, and pulse oximetry were recorded.

Percentage changes in pulse pressure and stroke volume during the respiratory cycle are reflected by PPV and SVV and can be derived by the following equations:

The PPV was calculated as follows [16]:

$$PPV\% = (PP_{max} - PP_{min}) / [(PP_{max} + PP_{min}) / 2] \cdot 100(\%),$$

where PP_{max} and PP_{min} are the maximal and the minimal values of pulse pressure.

The SVV was computed as follows [17]:

$$SVV\% = (SV_{max} - SV_{min}) / [(SV_{max} + SV_{min}) / 2] \cdot 100(\%),$$

where SV_{max} and SV_{min} are the maximal and the minimal values of stroke volume.

The SVI and GEDVI were obtained by transpulmonary thermodilution. Measurements were performed by injecting 15 mL ice-cold saline (≤8°C) through the central venous line. Injections were repeated at least 3 times and randomly assigned to the respiratory cycle. All thermodilution curves were analyzed; and in case of a difference of CI of at least 15% with respect to the preceding measurement, the values obtained were discarded and calibration was repeated.

The GEDVI was calculated as follows [18]:

$$GEDVI\ mL/m^2 = CI \cdot (\text{mean transit time} - \text{downslope time}),$$

where GEDVI is the sum of the right- and left-heart end-diastolic volumes, derived by the product of CI and the difference between mean transit time and downslope time of the transpulmonary thermodilution curve. Transpulmonary thermodilution parameters were automatically indexed to body surface area.

Using transesophageal echocardiography (TEE), ventilation-induced variation of velocity time integral (ΔVTI_{LVOT}) was quantified in the left ventricular outflow tract by pulsed-wave Doppler.

ΔVTI_{LVOT} was calculated as follows [19]:

$$\Delta VTI_{LVOT}\% = (VTI_{LVOT\ max} - VTI_{LVOT\ min}) / [(VTI_{LVOT\ max} + VTI_{LVOT\ min}) / 2] \cdot 100(\%),$$

where $VTI_{LVOT\ max}$ and $VTI_{LVOT\ min}$ are the maximal and the minimal values of the velocity time integral.