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Effect of pulse pressure on the predictability of stroke volume variation for fluid responsiveness in patients with coronary disease☆

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Abstract

Purpose: We hypothesized that the predictability of stroke volume variation (SVV) on fluid responsiveness would be reduced in patients with coronary disease who have wide pulse pressure (PP).

Methods: Sixty-six patients undergoing coronary surgery were divided into 2 groups according to the PP measured 1 day before surgery: normal PP (n = 33, PP ≤ 60 mm Hg) or wide PP (n = 33, PP > 60 mm Hg). After applying mechanical ventilation, hemodynamic parameters (including SVV measured by the FloTrac/Vigileo system [Edwards Lifesciences, Irvine, Calif]) were recorded before and 10 minutes after fluid replacement in a closed-chest condition. Prediction of fluid responsiveness was tested by calculating the area under the receiver operating characteristic curves.

Results: Twenty-one patients in the normal PP and 16 patients in the wide PP condition were fluid responders. The area under the receiver operating characteristic curves of SVV to predict fluid responsiveness were 0.808 (P = .022) and 0.609 (P = .288) in the normal PP and wide PP patients, respectively. In the normal PP condition, a SVV value of 13% discriminated between fluid responders and nonresponders with a sensitivity of 76% and a specificity of 67%.

Conclusions: In contrast to patients with normal PP, SVV does not predict fluid responsiveness in patients with coronary disease who have wide PP.

1. Introduction

Adequate volume replacement is often considered the initial step to augment cardiac output, yet proper assessment of the intravascular volume status is clinically challenging. When deciding whether fluid loading will result in an increase in cardiac output (fluid responsiveness), previous studies have reported that dynamic indices derived from arterial pressure wave form, such as stroke volume variation

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(SVV) and pulse pressure variation (PPV), are more reliable than are central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP) [1–5].

Both SVV and PPV are based on heart-lung interaction under mechanical ventilation reflecting respiratory changes in stroke volume (SV), which is directly reflected by pulse pressure (PP) [6]. Pulse pressure, however, has been assumed to be mainly associated with not only SV but also arterial stiffness [7]. Thus, the primary relationship between SV and arterial pressure is significantly influenced by changes in arterial tone [8], whereas the validity of algorithms using arterial pressure by industry to tract actual SV in patients with altered arterial stiffness has not been addressed heretofore.

In patients with coronary artery disease, systemic involvement of atherosclerosis results in increased arterial stiffness. Wide PP (>60 mm Hg) can be viewed as an extreme form of increased arterial stiffness, which results from decreased vascular compliance of large conduit arteries [9]. Not surprisingly, wide PP has been shown to be a major determinant of cardiovascular events and mortality in patients with coronary disease [10,11]. Furthermore, the importance of maintaining proper cardiac output in patients with coronary disease who have concomitant wide PP has been emphasized because these patients are more prone to develop myocardial ischemia after episodes of hypotension and/or reduced cardiac output [12].

The aim of this study was to validate the usefulness of static and dynamic indices including SVV measured by the FloTrac/Vigileo system (Edwards Lifesciences, Irvine, Calif) to predict fluid responsiveness as assessed by changes in SV measured by pulmonary artery catheter (PAC) in patients with coronary disease with wide PP.

2. Methods

The current trial was approved by the Institutional Ethics Committee of Yonsei University Health System and has been registered at http://clinicaltrials.gov (NCT 01404455). Between October 2010 and August 2011, we enrolled 66 patients older than 20 years who were scheduled for elective coronary artery bypass grafting (CABG). Written informed consent was obtained from all participants according to the results of their noninvasive blood pressure measurements (UA-621; A & D Medical, Saitama, Japan) in the ward 1 day before surgery. Patients were excluded from the study if any of the following criteria were present: arrhythmia, reduced left ventricular function (ejection fraction <40%), valvular heart disease requiring concomitant surgical correction, pulmonary hypertension (mean pulmonary arterial pressure ≥30 mm Hg), peripheral arterial occlusive disease, pulmonary disease (asthma, chronic obstructive pulmonary disease, and lung resection), end-stage renal disease, and a body mass index (BMI) of 35 kg/m^2 or greater.

Preoperative characteristics including age, sex, BMI, medical history, cardiac medications, and left ventricular ejection fraction (LVEF) were collected. All patients received 0.05 mg/kg of morphine intramuscularly as a premedication 1 hour before surgery. Upon arrival at the preanesthetic care unit, patients were placed in a supine position, and arterial blood pressure was measured 5 times by an automatic oscillometric measuring device (IntelliVue M70 monitor; Philips Medical Systems, Boeblingen, Germany) using an adapted brachial cuff size. To assure blindness to study design, blood pressure was measured by an independent nurse. Pulse pressure was calculated by subtracting the diastolic pressure from the systolic pressure. The median of the 5 PP values was selected and compared with PP value in the ward 1 day before surgery. Pulse pressure measurements in the preanesthetic care unit were not different from those of the ward 1 day before surgery; thus, none of the patients were excluded after obtaining informed consent. Patients were divided into 2 groups according to the PP: normal PP (n = 33, PP ≤ 60 mm Hg) or wide PP (n = 33, PP > 60 mm Hg). Because PP greater than 60 mm Hg is a risk factor for mortality after myocardial infarction (MI) [10], we divided the patients according to PP values of 60 mm Hg.

2.1. Anesthesia and monitoring

Before induction of anesthesia, arterial cannulation was performed in the right radial artery with a 20-gauge catheter as a part of standard monitoring, and a PAC (Swan-GanzCCOmbo; Edwards Lifesciences LLC) was inserted through the right internal jugular vein and connected to an analysis system (Vigilance; Edwards Lifesciences LLC).

Anesthesia was induced with intravenous midazolam (0.03-0.07 mg/kg) and sufentanil (1.5-2.0 μg/kg) and maintained with sevoflurane (0.8%-1.5%) and continuous infusion of sufentanil (0.5–1.0 μg kg^{-1} h^{-1}). Neuromuscular blockade was achieved by administration of rocuronium (0.9 mg/kg) and maintained with a continuous infusion of vecuronium (1-2 μg kg^{-1} min^{-1}). The patients' lungs were ventilated with a tidal volume of 10 mL/kg and an *I*/*E* ratio of 1:2 at a rate of 8 to 12 breaths/min in 40% oxygen with air, and 5 cmH_2O of positive end expiratory pressure was applied throughout the study period. After starting mechanical ventilation, a FloTrac/Vigileo system (Edwards Lifesciences; version 3.02) was connected to an existing radial arterial cannula to obtain SVV values. All transducers were zeroed at the midaxillary level, and care was taken to ensure that the pressure waveform was not dampened during data collection.

A transesophageal echocardiography probe was inserted to intraoperatively assess global cardiac function. During the study period, norepinephrine infusion was initiated when mean arterial pressure (MAP) was less than 60 mm Hg and stopped when MAP was greater than 80 mm Hg.