The Comparison of Stroke Volume Variation and Arterial Pressure Based Cardiac Output with Standard Hemodynamic Measurements during Cardiac Surgery

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Citation

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Abstract

BACKGROUND: Hemodynamic monitoring and fluid status assessment are essential for cardiovascular care in the surgical patient. Knowing when a patient's hemodynamic instability comes from hypovolemia or other causes is imperative in providing safe patient care. Commonly used methods of determining fluid status such as central venous pressure (CVP) and pulmonary artery pressure (PAP) do not accurately reflect the left ventricular end diastolic area and volume values, even when trends are followed after the administration of a fluid challenge during hemodynamic instability. The purpose of this study was to compare the stroke volume variation (SVV) and arterial pressure based cardiac output (APCO) with the current accepted methods on cardiac output and preload status.METHODS: Prospective, observational study in 100 cardiac surgery patients. Continuous cardiac output (CCO), APCO, SVV, PCWP, CVP and ventricular end diastolic area and volume were measured. The APCO were compared with CCO, the cardiac output values of continuous thermodilutional method. The central APCO was compared with peripheral APCO. SVV values were compared with LVEDA, LVEDV, CVP, PAP, and other hemodynamic values.RESULTS: The correlations between SVV and LVEDA and LVEDV were R^2=0.7027 and R^2=0.7924 (p<.0001) respectively. The correlation between APCO and CCO was R^2=0.8309 (p<.0001). The correlation between central APCO and peripheral APCO was R^2=0.9155 (p<.0001).CONCLUSIONS: SVV is a good indicator of the cardiac preload. It is superior to static indicators of cardiac preload and has a higher correlation with LVEDA and LVEDV. It can therefore be used to guide intraoperaitve fluid therapy. APCO measured from peripheral artery has a high correlation with the central CO and conventional method for CO measurement, therefore, it can accurately reflect cardiac output.

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INTRODUCTION

Hemodynamic monitoring plays a central role in cardiovascular diagnosis and treatment. Adequate volume replacement to achieve optimal cardiac performance is a primary goal of hemodynamic management in patients undergoing cardiac and non-cardiac surgery. However, in only half of the patients deemed to need volume replacement, does the cardiac output (CO) increase after a fluid challenge and the rest of the half does not. Physicians would therefore need a reliable criteria to distinguish theose two patient populations to avoid any deleterious consequence of fluid overload.

It has been demonstrated that neither the standard preload indices such as central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), heart rate or blood pressure nor their trends in response to fluid challenge reflected left ventricular preload or its trends in patients receiving a fluid challenge for hemodynamic instability. Therefore, they are not capable of predicting cardiac response to fluid therapy [1,2]. As an alternative to these static variables, assessment of stroke volume variation (SVV) has been used as a dynamic index to guide fluid therapy in patients receiving mechanical ventilation. Cardiac preload is highly susceptible to changes in intrathoracic pressure induced by mechanical ventilation. As the stroke volume (SV) varies, the changes of systolic arterial pressure and pulse pressure variation (PPV) can be observed. The SVV/PPV are more pronounced during hypovolemia and the variation decreases if intravascular volume is restored, and it has shown to reliably predict changes in CO [3]. This study

was designed to validate the arterial pressure based CO (APCO) and SVV by 1. comparing SVV with the "gold standard" of cardiac preload, the transesophageal echocardiography (TEE) measurement of the left ventricular end-diastolic area (LVEDA) and left ventricular end-diastolic volume (LVEDV). 2. Comparing SVV with other standard preload parameters generated from the pulmonary arterial catheter (PAC) and other hemodynamic parameters. 3. Comparing APCO to continuous thermodilutional cardiac output (CCO) from PAC. In this study, we also tested: 4. Whether the CO obtained from peripheral (radial artery) reflects the central (aortic root) CO.

METHODS

This study was approved by the Institutional Review Board. A total of 100 scheduled to undergo open-heart surgery in a single-university setting were included in this observational study (Table 1). All the patients were mechanically ventilated with the tidal volume 8 ml/kg since the tidal volume can significantly affect the SVV value [4, 5]. Patients were excluded if they refused and/or if they presented with atrial fibrillation (AF), severe arrhythmias, a permanent pacemaker, and the need for mechanical cardiac support. The Vigileo system (Edwards Lifesciences, LLC, Irvine, CA) using a FloTrac (Edwards Lifesciences, LLC, Irvine, CA) arterial pressure sensor was used in all the patients to measure SVV and APCO.

Figure 1

Table 1. Patients Demographic Data

Total patients	n =100
Gender:	
Male:	67
Female:	33
Age (years):	61±12
Height (cm):	172±10
Weight (kg):	84±21
CPB time (minutes):	188±54
Cross-clamping time (minutes):	146±42

CPB: cardiopulmonary bypass

The standard anesthesia monitors were used in the operating room which included: continuous electrocardiograms, pulse oximetry, end-tidal carbon dioxide and noninvasive blood pressure (Bp) monitoring, all patients received a 20-G radial arterial catheter (Arrow International, LLC, Reading, PA) on the side with a more prominent pulse. All patients received a right internal jugular vein introducer (Arrow International, LLC, Reading, PA) and PAC (Edwards Lifesciences, LLC, Irvine, CA) and the PAC was connected to the Vigilance monitor (Edwards Lifesciences, LLC, Irvine, CA). CVP, PAP, CCO were measured throughout the surgery. After induction, general anesthesia was maintained with oxygen, sevoflurane, muscle relaxants, and narcotics. TEE (Agilent SONOS 5500, Philips Medical Systems, Andover, MA) was routinely used in all cardiac surgery patients to monitor the cardiac volume status (LVEDA, LVEDV), anatomy, and ventricular function. The SVV, APCO, CCO, HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), PAP and CVP were recorded during surgery and at the same time the LVDEV and LVEDA measured with TEE. LVEDV in 40/100 patients was measured by tracing the LV area along the endocardial border in the mid-esophageal LV 2 chamber view at the end of diastole. The Agilent SONOS 5500 system (Agilent Technologies, Andover, MA) calculated the LV volume using the modified Simpson rule. LVEDA in 80/100 patients was measured in the transgastric LV mid-papillary short axis view by tracing the endocardium border.

For the comparison between SVV with LVEDA and SVV with LVEDV, the patients with poor LV function (EF< 35%) were excluded. 80/100 patients were in the group of comparing SVV with LVEDA. There were 40 out of the 80 patients were in the group of comparing SVV with LVEDV. Half of the data were collected before bypass and the other half of the data were collected after bypass. Half of the data were collected before bypass and the other half of the data were collected after bypass. All the patients, regardless of the LV size and function, were included in the comparison between the CCO and APCO. 40/100 patients who received central arterial pressure monitoring via anterograde cardioplegia catherters were used to compare whether the CO measured at the radial artery reflects the central CO measured at the aortic root.

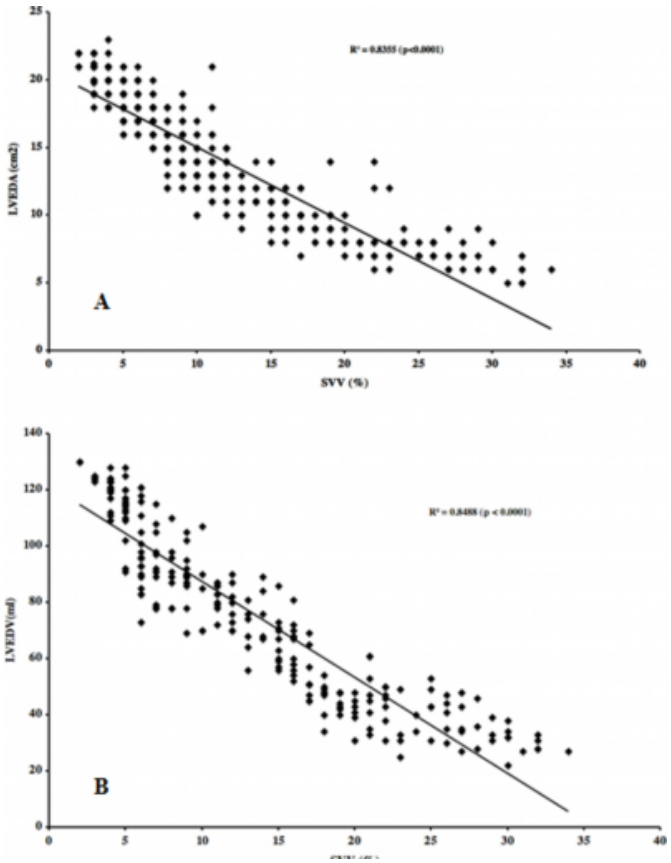
Bland-Altman analysis, Pearson's correlation and ANOVA were used for statistical analysis. A p value less than 0.05 was considered to be statistically significant. Data were presented as Mean±SD.

RESULTS

Comparing SVV with LVDEA and LVEDV: As presented in Figure 1A, 480 data points were collected and the SVV had significant correlation with LVEDA with the R^2=0.7027 (P<0.0001). In Figure 1B, there were 240 data points collected and the SVV had a high correlation with LVEDV (R^2=0.7924, P<0.0001).

Figure 2

Figure 1: A: presented the results of the correlation between the SVV and LVEDA. N = 480. B: presented the results of the correlation between the SVV and LVEDV. SVV: stroke volume variation; LVEDA: left ventricular end diastolic area. N = 240 and P < 0.05 is considered to be statistically significant.

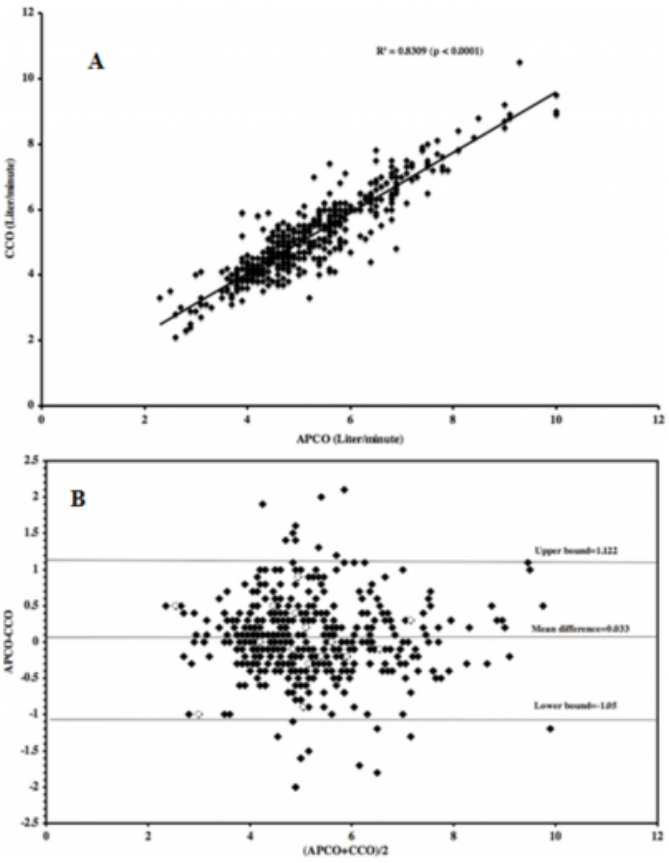


Comparing SVV with other hemodynamic parameters: Our data are consistent with the others reports in the literature that standard hemodynamic parameters such as CVP, PAP, HR, Bp do not reflect the preload and intravascular volume status. SVV compared with CVP, PAP, HR and Bp showed no correlations between SVV and each of the hemodynamic parameters (Table 2). Those standard hemodynamic parameters also had no correlation with the LVEDA except there were week correlations with systolic PAP (r^2 = 0.4140, p < 0.05), diastolic PAP (r^2 = 0.3118, p < 0.05) and mean PAP (r^2 = 0.2134, p < 0.05) (Table 2).

APCO and CCO: Our data demonstrated that the CO derived from using arterial contour analysis has a high correlation with the CO measured via continuous thermodilution method in 480 measurements (r^2=0.8309, p<0.0001) (Figure 2A). The Bland-Altman analysis demonstrated the limits agreement for APCO vs. CCO were -1.05 to +1.122 L/min with a mean difference of 0.033 L/min) (Figure 2B).

Figure 3

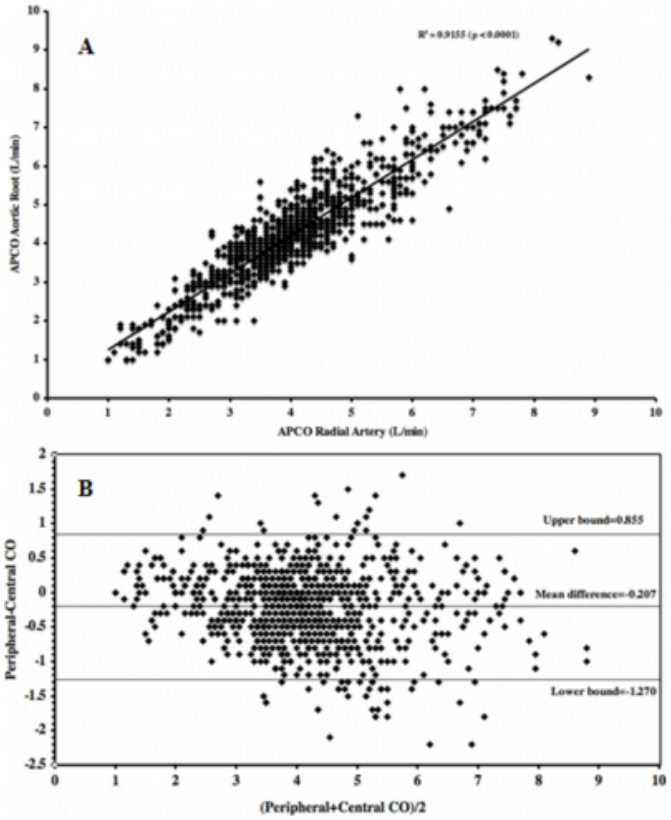
Figure 2: A: presented the results of the correlation between the APCO and CCO. B: shown the results of Bland and Altman plot for the comparison of APCO and CCO values. APCO: arterial pressure based cardiac output; CCO: continuous cardiac output. N = 480 and P < 0.05 is considered to be statistically significant.



Central vs. Peripheral APCO: The APCOs were measured from radial artery and aortic root at the same time from the same patients. A total of 1044 measurements were performed. Our data demonstrated that the CO obtained from the peripheral radial artery can accurately represent the central CO obtained from the aortic root. They have a high correlation with R^2=0.9155 (p<0.0001) (Figure 3A). The limits of agreement for central CO vs. peripheral CO were -1.270 to 0.855 L/min with a mean difference of -0.207 L/min (Figure 3B).

Figure 4

Figure 3: A: presented the results of the correlation between the central cardiac output measured at the aortic root and peripheral cardiac output measured at the radial artery. B: shown the results of Bland and Altman plot for the comparison of central cardiac output and peripheral cardiac output values. APCO: arterial pressure based cardiac output. N = 1044 and P < 0.05 is considered to be statistically significant.



DISCUSSION

Our data show that the SVV has a high correlation with LVEDA and LVEDV measured by TEE for LV preload assessment. Our study also demonstrated that CO by pulse contour analysis using Vigileo system has a high agreement and correlation with the standard transpulmonary thermodilution method for CO assessment. We have further demonstrated that the APCO measured in the peripheral artery had a high limit of agreement and correlation with the APCO measured in the central artery.

The assessment of a patient's CO typically involves the placement of a catheter in the pulmonary artery and performing thermodilution measurement. Although this method is accurate under most clinical conditions and has been well accepted by most clinicians, it is invasive and associated with many complications. Most importantly, the measurement is obtained in the right heart and may not reflect events in the left heart. Bouchard et al compared right and left ventricular stroke work index with echocardiography-derived indices of left ventricular performance in cardiac surgery patients. Correlation between right and left ventricular stroke work index changes was poor. Thus, there is a significant discrepancy and limited relation between the preload, estimated with classical variables, and the left ventricular performance [3]. The APCO is a CO monitoring system based on the analysis of the systemic arterial pressure wave that does not require pulmonary artery catheterization and most importantly it provides measurement of the left heart. Our data suggests that APCO can adequately reflect the CO and correlates well with the CO measured with transpulmonary thermodilution method. The APCO measured from radial artery not only has a high correlation with the CCO but also accurately represents the central CO measured at the aortic root. Our results are in consistent with the recently published studies using Vigileo/FloTrac system for CO/CI measurement. The authors have demonstrated a good agreement in CO/CI with that measured by continuous and intermittent thermodilution techniques using PAC [6, 7, 8]. Although there were some differences between the APCO and CCO, this difference may not invalidate the use of APCO. Since the CCO measurements are obtained in the right heart, the observed discrepancy could be the result of the differences between the right and left heart. Furthermore, the CO measured at the distal radial artery could well represent the CO at the end organ level.

The use of arterial pulse wave to determine the patient's hemodynamic status has intrigued both scientists and clinicians for decades. But only 1% of physicians in a survey consider the "swings" in blood pressure during respiration as part of their decision-making process regarding volume expansion [9]. Only recently has this method become available for commercial use in the equipment to measure the hemodynamics. These devices include the LiDCO system (LiDCO Limited, UK), which requires calibration using lithium dilution, the PiCCO system (Pulsion Medical System, Munich, Germany), requiring transpulmonary thermdilution, the Finapres Modelflow system (Finapres Medical Systems, The Netherlands), with which calibration with another means of CO measurement is advisable to achieve acceptable accuracy, and the most recent FloTrac /Edwards Vigileo system (Edwards Lifesciences, LLC, USA), instead of recalibrating every few hours, it calculates

stroke volume through continuous self-calibration and provides continuous CO, and SVV measurements from the arterial pressure wave by using the information of arterial pulsatility, resistance, compliance, age gender, height, weight and waveform characteristics [10, 11]. The advantage is that it allows the physicians to provide an un-interrupted patient care during surgery.

Morgan et al. first reported that the mechanical ventilation induces cyclic changes in vena cava blood flow, pulmonary blood flow and aortic blood flow. During the inspiratory period, the vena cava blood flow decreases first, followed by a decrease in pulmonary artery flow and then in aortic blood flow. The decrease in vena cava blood flow has been related both to an increase in right atrial pressure and to the compression of the vena cava due to the inspiratory increase in pleural pressure during mechanical ventilation [12]. According to the Frank-Starling mechanism, the inspiratory decrease in right ventricular preload results in a decrease in right ventricular output and pulmonary artery blood flow that finally leads to a decrease in left ventricular filling and output [4].

The left ventricular end-diastolic area assessed by echocardiography has been considered a “gold standard” to reflect the left ventricular preload. It is considered a better indicator of LV preload than the PCWP and is very sensitive to changes in blood volume [13, 14, 15]. Study found a significant relation between the left ventricular diastolic area and the magnitude of systolic pressure variation in aortic surgery patients [15]. The LV mid short axis view has been extensively used in intra-operative monitoring of the LV preload and patient volume status [13, 14, 15]. In this study, we demonstrated that the SVV change had a very good correlation with the LVEDA and LVEDV in non-dilated left ventricles with normal cardiac function. On the other hand, the LVEDA and LVEDV are significantly increased in the patients with poor LV function and dilated left ventricles and therefore could not been adequately used to reflect the patient’s volume status. In this case the SVV could be a better indicator for patient’s circulatory volumes as it also demonstrated by Reuter et al [16]. This observation has been previously shown to have a good correlation with the left ventricular end-diastolic area changes measured by echocardiography [15].

Although SVV has not been widely used to guide everyday practice in volume therapy, there are several publications that demonstrated its usefulness in fluid management and resuscitation in the critical care and the cardiac surgery settings. Hofer et al reported the use of SVV and PPV for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting (OPCABG). The authors concluded that SVV and PPV are closely related. SVV and PPV assessment showed comparable good performance in predicting fluid responsiveness in patients with preserved left ventricular function undergoing OPCABG [17]. In a different study the authors investigated whether the degree of PPV can predict an increase CO in response to volume challenge in postoperative patients who have undergone coronary artery bypass grafting. The authors concluded that PPV can be used to predict whether or not volume expansion will increase CO in postoperative CABG patients and PPV is superior to CVP and PCWP [18]. Several other studies were conducted to test the use of dynamic parameters (SVV/PPV) in patients with unstable hemodynamic requiring fluid resuscitation. Their conclusions are that the dynamic parameters should be used preferentially to static parameters to guide fluid resuscitation in ICU patients [19, 20]. In agreement with above conclusions, our results also demonstrated that there was no significant correlation between the SVV and static parameters CVP and PCWP. There was also no significant correlation between the LVEDA/LVEDV and CVP and PCWP.

While the usefulness of using SVV and APCO to detect preload sensitivity is indisputable, a number of limitations must be remembered. The dynamic induces cannot be used in spontaneously breathing patients and/or patients with severe arrhythmias. Even if the detection of fluid responsiveness is found to be of use in the decision-making process regarding volume expansion, two important points must be kept in mind. First, since both ventricles of a healthy subject operate on the steep portion of the preload/SV relationship, volume responsiveness is a physiological phenomenon related to a normal preload reserve. This may not be true in subjects with exhausted preload reserve. Therefore, detecting volume responsiveness must not automatically lead to a decision of infusing fluid. Second, it is reasonable to postulate that volume loading should be more beneficial in a hypotensive patient with low CO and volume responsiveness than in a hypotensive patient with a high CO and less degree of volume responsiveness for whom administration of vasopressors should be more logical. This emphasizes the great interest in new commercially available devices that monitor and display both CO and SVV/PVV together from beat-to-beat analysis of arterial pressure waveform.

There are some specific limitations in this study. This is an observational study not an outcome study. When we designed the study, our goal was to compare this new method to the "standard methods" in our clinical practice. It would be necessary to design an outcome study to test whether this method can improve the clinical outcome. So the implications are only limited to the clinical situations the conclusions were drown.

From this study, we concluded that the APCO not only adequately reflect the CCO but also represents the central left heart CO. The dynamic index, SVV, is superior to static indicators (CVP, PAWP) in monitoring and predicting LV preload and circulatory volume. The SVV has a high correlation with LVEDA and LVEDV measured by TEE. When used with awareness of their limitations, SVV and APCO are sensitive tools that can be used to guide the appropriate management of the patient's LV preload to achieve optimal CO.

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