A comparative study of systolic pressure variation and blood volume measurements.

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Introduction

Clinical assessment of circulating blood volume (BV) has been a challenge especially in patients with third spacing of fluids during shock states.

Studies suggest that systolic pressure variation (SPV), the difference between maximal and minimal systolic pressure values during one mechanical breath, is correlated with cardiac fluid responsiveness defined as an increase in the stroke volume index or cardiac index >10 to 15% (1). However, SPV is confounded by the patient's cardiac function, mechanics of breathing, and ventilator settings and may not reflect circulating BV.

Blood volume (BV) can be measured utilizing a radioactive-iodine labeled albumin technique. Blood volume analysis (BVA) provides information on intravascular circulating volume and its component volumes [plasma volume (PV) + red cell volume (RBCV)]. Assessment of intravascular blood volume in critically ill patients may be useful to guide clinicians in administering fluid therapy.

This study describes the relationship between SPV, a surrogate marker of cardiac fluid responsiveness and BVA, a measurement of intravascular volume.

Hypothesis

There is no relationship between SPV and circulating BV.

Methods

Simultaneous measurements of blood volume and arterial blood pressure were taken on critically-ill surgical patients. BVA was done after initial resuscitation on days 1, 2, 3, and 5-7 if the patients remained in the ICU.

Systolic Pressure Variation (SPV)

SPV is the difference between the maximum and the minimum systolic pressure (SP) over a single respiratory cycle and can be expressed in millimeters of mercury; SPV (mmHg) = SPmax – SPmin, or as a percent; SPV (%) = 100 x (SPmax – SPmin)/(SP max + SPmin)/2. We calculated the SPV percentage from paper tracings of arterial pressure. All data acquired during irregular heart rate and spontaneous respirations were excluded.
SPV > 10% indicates cardiac fluid responsiveness (2).

Figure 1 shows the arterial pressure fluctuations during mechanical ventilation. The systolic pressure is maximal during inspiration and declines during expiration.

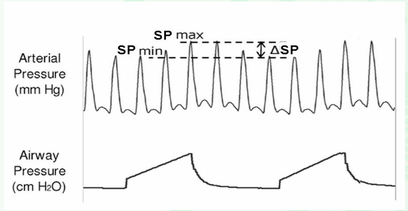

Fig. 1. Arterial pressure fluctuations during mechanical ventilation

Blood Volume Analysis (BVA)

Plasma volume (PV) was measured using the BVA-100 (Daxor, NY, NY). After obtaining a baseline sample of 5 mL of blood, 1 mL of I-131 labeled albumin was injected over 1 minute. After 12 minutes to allow complete mixing, 5 blood samples were collected at 6 minute intervals and extrapolated to time 0 to account for albumin extravasation from the intravascular space. Hematocrit (Hct) measurements = [RBCV/(RBCV + PV)] were obtained at time of BV measurements.

BV is equal to RBCV + PV. From BV and Hct results RBCV was calculated. BV results are reported as a percentage deviation from the patient's normal/ideal BV (Fig. 2). Predicted normal values were derived from a formula based on patient height, weight, and % deviation from ideal weight (3). For these critically ill patients with vascular volume expansion, hypovolemia was defined as any value less than 0% deviation from normal/ideal blood volume. Euvolemia was defined as 0-8% deviation from normal/ideal BV, and hypervolemia was defined as >8% deviation from normal/ideal BV (Table1) (4). BVA was done after initial resuscitation on days 1, 2, 3, and 5-7 if the patients remained in the ICU.

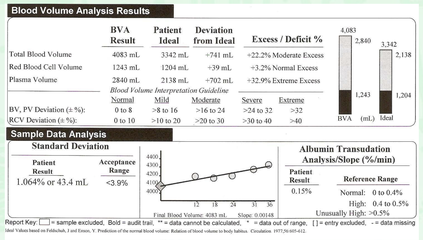

Fig. 2. An example of Blood Volume Analysis Results.

Table 1. A method of categorizing deviations from the ideal or normal blood volume.

	Whole Blood Volume	Red Cell Volume	Plasma Volume
Normal	±8%	±10%	±8%
Mild Deviation	±9-16%	±11-20%	±9-16%
Moderate Deviation	±17-24%	±21-30%	±17-24%
Severe Deviation	±25-32%	±31-40%	±25-32%
Extreme Deviation	>32%	>41%	>32%

Results

The patients (n=100) contributed 301 data points with the following demographics: age 62 ± 16 years, Male: Female 61:39, APACHE II = 24 ± 3.0. Diagnoses included: severe sepsis/septic shock (n=68), ARDS (n=37), cardiovascular collapse (n=21). Regression analysis showed no correlation between BV and SPV (R2=0.004, p=0.254). (See Fig. 3)

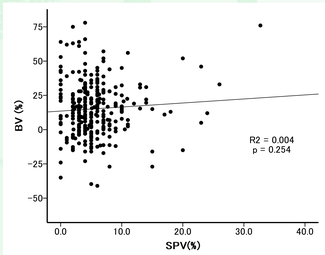

Fig. 3. A simple regression analysis between circulating BV(%) (percent deviation from normal/ideal blood volume) and SPV (%).

A cross table analysis of SPV versus BV is presented in Table 2. BV demonstrated euvolemic/hypervolemic state in 23 of 26 instances (88%) when SPV was >10%, namely false positive. BV demonstrated a hypovolemic state in 44 of 275 instances (16%) when SPV was ≤10%, i.e. false negative. When predicting hypovolemia, SPV>10% has a positive predictive value of 12% and negative predictive value of 84%, respectively.

Table 2. Cross table analysis of SPV versus BV. FN = false negative, TP = true positive, TN = true negative, FP = false positive.

		condition	
		Positive	Negative
		hypovolemia (BV < 0%)	Euvolemia / hypervolemia (BV 0 - 8%) / (BV > 8%)
Test outcome	Positive (SPV > 10%)	3 (TP)	23 (FP)
	Negative (SPV ≤ 10%)	44 (FN)	231 (TN)

Discussion

This poor relationship between SPV and BV may be due to following reasons. The magnitude of SPV may be confounded by the patient's heart rate, cardiac function, aortic properties, chest wall characteristics, lung compliance and ventilator settings. In addition, SPV may be affected by sepsis with decreasing systemic vascular resistance and impaired sympathetic nervous system. It is also important to consider the definition of cardiac fluid responsiveness. Whether the patient's heart responds to fluid therapy is dependent on the position on the Frank-Starling curve, but does not necessarily imply that the patient needed the fluid.

Conclusions

There was no relationship between SPV and circulating BV. Although SPV may reflect cardiac responsiveness to fluid, there was no relationship to intravascular blood volume. While SPV >10% indicated hypovolemia in 12% of the time, SPV ≤10% indicated adequate circulating blood volume in 84 % of the time.

While there is no ideal method of assessing intravascular volume status, clinicians must use caution when extrapolating surrogate markers such as hemodynamic parameters to determine intravascular blood volume status.

References

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