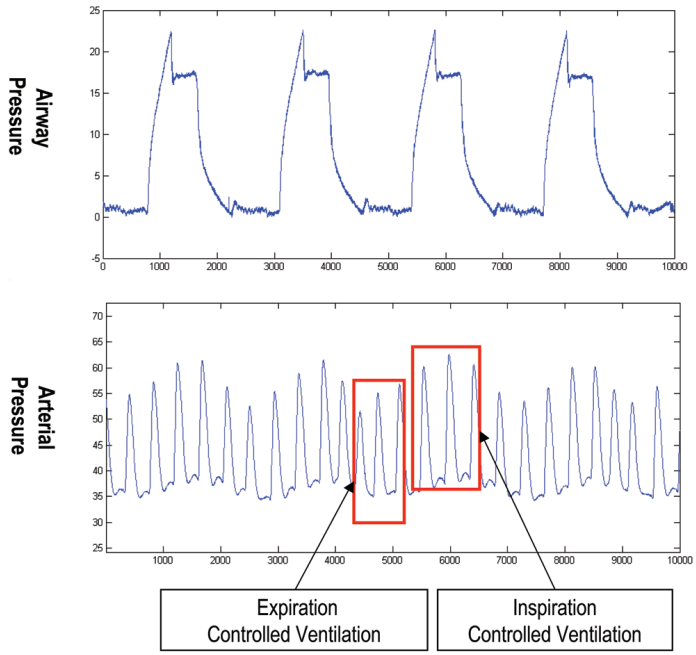
Stroke Volume Variation
"Can We Use Fluid to Improve Hemodynamics?"

Introduction

In the quest to achieve optimal oxygen delivery (DO_2), clinicians are often forced to use imprecise, non-specific information to guide their therapy. Traditional hemodynamic monitoring parameters (HR, MAP, CVP, and PAOP) are often insensitive and sometimes misleading in the assessment of circulating blood volume. However, the appropriateness of their interventions is often crucial to avoid the deleterious effects of over-, under-, or inappropriate resuscitation. Volume is one of the first therapeutic interventions selected when optimizing DO_2. Often times the choice to intervene using fluid is accompanied by the difficult questions, "Can using fluid improve hemodynamics?" and, "Is it the appropriate intervention?" Stroke volume variation (SVV) as available on the FloTrac system may help answer these questions.



What Causes Stroke Volume Variation?

Stroke volume variation is a naturally occurring phenomenon in which the arterial pulse pressure falls during inspiration and rises during expiration due to changes in intra-thoracic pressure secondary to negative pressure ventilation (spontaneously breathing). Variations over 10mmHg have been referred to as pulsus paradoxus. The normal range of variation in spontaneously breathing patients has been reported between 5-10mmHg.

Reverse pulsus paradoxus is the same phenomenon with controlled mechanical ventilation, however, in reverse. Arterial pressure rises during inspiration and falls during expiration due to changes in intra-thoracic pressure secondary to positive pressure ventilation. In addition to reverse pulsus paradoxus, it has also been referred to as paradoxical pulsus, respiratory paradox, systolic pressure variation and pulse pressure variation. Traditionally SVV is calculated by taking the SV_{max} - SV_{min} / SV mean over a respiratory cycle or other period of time.

SVV and Assessing Fluid Response

SVV and its comparable measurement, pulse pressure variation (PPV), are not indicators of actual preload but of relative preload responsiveness. SVV has been shown to have a very high sensitivity and specificity when compared to traditional indicators of volume status (HR, MAP, CVP, PAD, PAOP), and their ability to determine fluid responsiveness. The following table of studies demonstrates SVV sensitivity and specificity in predicting fluid responsiveness against a specified infused volume and defined criteria for a fluid responder.

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Study	Patients	Volume	Tidal Volume ml/Kg	Parameters Tested (Artery)	R^2	Def. of Responder	Sensitivity	Specificity
Michard[2]	Sepsis	500 ml	8 to 12	ΔPP (R or F)	0.85	ΔCO≥15%	94	96
Berkenstadt, et al.[1]	Neuro Surgery	100 ml	10	ΔSVV	0.53	ΔSV≥5%	79	93
Reuter, et al.[3]	Cardiac	10 x BMI	10	ΔSVV	0.64	ΔSV≥5%	79	85

How Can I Use SVV?

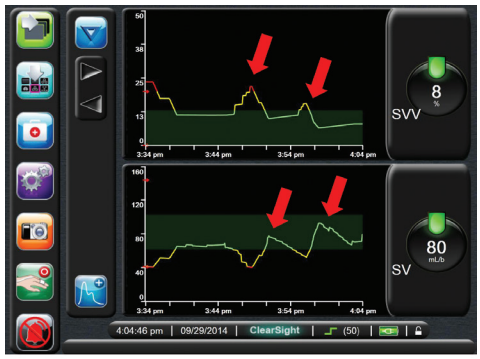
Normal SVV values are less than 10-15% on controlled mechanical ventilation. The figures to the right demonstrate using SVV as a guide for volume resuscitation with a goal SVV of <13%. SVV increased to 19% with a stroke volume (SV) of 43 ml/beat, blood and saline were given to obtain a SVV of 6% and a SV of 58 ml/beat.

What are the Limitations of SVV?

- **Small Tidal Volume and Spontaneous Breathing**
  Currently, literature supports the use of SVV only on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8cc/kg, fixed respiratory rates and no spontaneous breaths.

- **Open Chest Conditions**
  Currently, literature does not support the use of SVV in patients who have an open chest.

- **Sustained Arrhythmias**
  Edwards APCO technology allows for the filtering of aberrant beats caused by arrhythmias and the continued use of SVV to determine preload responsiveness. A yellow heart icon indicates that there are too many arrhythmias (i.e. A-Fib) to filter and SVV cannot be used to determine preload responsiveness.



2 Units of PRBC followed by 1 liter NaCl

- **Effects of Therapies on SVV**
  Increasing levels of positive end expiratory pressure (PEEP) may cause an increase in SVV, the effects of which may be corrected by additional volume resuscitation if warranted.

- **Vascular Tone**
  Vasoactive medications can affect SVV. Vasopressors may decrease SVV and Vasodilators may increase SVV.

Summary

When used within its limitations SVV is a sensitive tool that can be used to guide the appropriate management of the patient's preload to achieve optimal DO_2 and answer the question "Can we use fluid to improve hemodynamics?"

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References
1. Berkenstadt H, et al. Stroke volume variation as a predictor of fluid responsiveness in patients undergoing brain surgery. *Anesth Analg* 2001;92:984-989.
2. Michard F. Changes in arterial pressure during mechanical ventilation. *Anesthesiology* 2005; 103:419-428.
3. Reuter DA, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. *Crit. Care Med* 2003; 31:1300-404.

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