Edwards FloTrac Sensor & Edwards Vigileo Monitor

Understanding Stroke Volume Variation and Its Clinical Application
Topics

- System Configuration
- Pulsus Paradoxes
- Reversed Pulsus Paradoxus
- What is Stroke Volume Variation (SVV)?
- How is SVV Calculated?
- Sensitivity and Specificity
- Clinical Application of SVV
- Limitations of SVV
The Vigileo monitor by Edwards Lifesciences supports both the FloTrac sensor for continuous cardiac output and the Edwards PreSep central venous catheter for continuous central venous oximetry (ScvO2)
The Vigileo monitor continuously displays and updates Continuous Cardiac Output, Cardiac Index, Stroke Volume, Stroke Volume Index, Systemic Vascular Resistance*, Systemic Vascular Resistance Index*, and Stroke Volume Variation every 20 seconds when used with the FloTrac sensor. DO2 and DO2I are also available for manual calculation.** These parameters help guide the clinician in optimizing stroke volume through precision guided management of preload, afterload, and contractility.

Vascular tone = vessel compliance and resistance

The Vigileo monitor then helps identify the adequacy of cardiac output by monitoring central venous (ScvO2) or mixed venous (SvO2) oxygen saturation when used with Edwards venous oximetry technologies.

* These parameters require the CVP value to be slaved from bedside monitor for continuous monitoring. SVR/SVRI can also be assessed on the Derived Value Calculator for intermittent calculations using either slaved or manually entered MAP, CVP, and CO values.

**These parameters require the SpO2 and PaO2 values to be manually entered. If CO is being continuously monitored, the calculator will default to the existing CO value. Otherwise, the user may override the continuous value to manually enter CO.
The specially designed \textit{FloTrac} sensor provides the high fidelity arterial pressure signal required by the \textit{Vigileo} monitor to calculate the stroke volume.

The Vigileo monitor uses the patient's arterial pressure waveform to continuously measure cardiac output. With inputs of height, weight, age and gender, patient-specific vascular compliance is determined.

The \textit{FloTrac} sensor measures the variations of the arterial pressure which is proportional to stroke volume. Vascular compliance and changes in vascular resistance are internally compensated for.

Cardiac output is displayed on a continuous basis by multiplying the pulse rate and calculated stroke volume as determined from the pressure waveform.

The \textit{FloTrac} sensor is easily setup and calibrated at the bedside using the familiar skills used in pressure monitoring.
Stroke Volume Variation
In a normal individual who is breathing spontaneously, blood pressure decreases on inspiration. The range of normal peak decreases in systolic pressure have been reported between 5 – 10 mmHg.

The exaggeration of this phenomenon, called pulsus paradoxus, was initially reported by Adolf Kussmal in constrictive pericarditis and was described as a “pulse disappearing during inspiration and returning during expiration” despite the continued presence of the cardiac activity during both respiratory phases.


A phenomenon that is the reverse of the conventional pulsus paradoxus has been reported during positive pressure breathing.

The inspiratory increase in arterial blood pressure followed by a decrease on expiration has been called at different times:
- Reversed pulsus paradoxus
- Paradoxical pulsus paradoxus
- Respirator paradox
- Systolic pressure variation (SPV)
- Pulse pressure variation

At least 12 peer-reviewed English-language studies have demonstrated the usefulness of the respiratory variation in arterial pressure (or its surrogates) in answering a crucial clinical question; “Can we improve cardiac output and hence hemodynamics by giving fluid?”

Mechanical ventilation induces cyclic changes in vena cava blood flow, pulmonary artery blood flow, and aortic blood flow. At the bedside, respiratory changes in aortic blood flow are reflected by “swings” in blood pressure whose magnitude is highly dependent on volume status. (Michard)

The left ventricular stroke volume increases during inspiration because left ventricular preload increases while left ventricular afterload decreases. In contrast, the right ventricular stroke volume decreases during inspiration because right ventricular preload decreases while right ventricular afterload increases. Because of the long (approximately 2 sec) pulmonary transit time of blood, the inspiratory decrease in right ventricular filling and output only a few heartbeats later, i.e., usually during the expiratory period. (Michard)

In healthy patients undergoing neurosurgery, a SVV value of 9.5% was found to predict a positive (=5%) increase in stroke volume in response to only 100ml of plasma expander with a 79% sensitivity and 93% specificity. (Berkenstadt)

Currently, the majority of clinical literature supports the use of SVV in patients who are only mechanically ventilated.
The above equation is the conventional method by which SVV is calculated. The difference between the maximum and minimum stroke volumes within a given time are divided by the mean of the maximum and minimum SVs. The SVV is displayed as a percent value.
These two studies demonstrate that SVV has a high sensitivity and specificity in determining if a patient will respond (increasing stroke volume) when giving additional volume. This is referred to as “preload responsiveness”.

SVV values used to guide preload responsiveness have varied between 10 and 15%.

Using SVV allows us to answer the question “Can we use fluid to improve hemodynamics?”

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Using SVV allows us to answer the question “Can we use fluid to improve hemodynamics?”
The above case is an exploratory laparotomy with tumor removal. The FloTrac sensor was used in addition to traditional vital signs.

The patient experienced a sudden loss of blood during the procedure and was volume resuscitated (packed red blood cells and Normal Saline). SVV was used as a guide for starting and stopping volume resuscitation at the points shown by the arrows. The following slides demonstrate the patient’s response to resuscitation at the point highlighted by the second arrow above.
With a SVV of 19% and stroke volume of 45 ml/beat, the patient received one unit of packed red blood cells and 500ml Normal Saline.
After resuscitation the SVV decreased to between 9 - 6% with a Stroke Volume increase to approximately 60 ml/beat. One aberrant reading of 25% (at 2:32 p) was due to an arrhythmia.
As displayed in the numerical trend screen, the graphical trend screen also shows an increase in cardiac output in response to volume resuscitation.
What are the Limitations of SVV?

**Mechanical Ventilation**
- Currently, literature supports the use of SVV on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8cc/kg and fixed respiratory rates.

**Spontaneous Ventilation**
- Currently, literature does not support the use of SVV with patients who are spontaneously breathing.

**Arrhythmias**
- Arrhythmias can dramatically affect SVV. Thus, SVV’s utility as a guide for volume resuscitation is greatest in absence of arrhythmias.

Although a powerful tool managing your patients volume resuscitation, SVV has limitations.

**Mechanical Ventilation:**
Current literature supports the use of SVV on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8cc/kg and fixed respiratory rates.

**Spontaneous Ventilation:**
Currently, literature does not support the use of SVV with patients who are spontaneously breathing. Spontaneous breaths uses negative pressure ventilation with small, varying tidal volumes.

**Arrhythmias:**
Arrhythmias can dramatically affect SVV. Thus, SVVs utility as a guide for volume resuscitation is greatest in absence of arrhythmias.

**SVR:**
The effects of vasodilatation therapy on SVV should be considered before treatment with additional volume.
Positive End Expiratory Pressure (PEEP) and SVV

- PEEP may decrease cardiac output and offset the expected benefits of oxygen delivery

- Arterial waveform analysis is useful to predict and prevent the deleterious hemodynamic effects of PEEP in mechanical ventilation

The deleterious hemodynamic effects of PEEP are caused by an increase in pleural pressure (reducing right ventricular filling) and an increase in Transpulmonary pressure (increasing right ventricular afterload).

When cardiac output decreases with PEEP the arterial pressure variation increases. If PEEP does not affect cardiac output the arterial pressure variation is similarly unaffected by PEEP.

The arterial pressure waveform analysis is useful to predict and prevent the deleterious effects of PEEP in mechanical ventilation.

Summary

• SVV is easily measured and displayed at the bedside with the Vigileo monitor

• Studies reviewed by Michard demonstrate that dynamic indicators such as SVV and PPV are effective in determining the patient’s fluid responsiveness, while static indicators of preload (CVP, PAD, PAOP) are frequently unable to correctly answer an important question...

“Can we use fluid to improve hemodynamics?”

Edwards Vigileo monitor when used with the FloTrac sensor, easily measures and displays SVV at the bedside.

SVV is a very reliable indicator of the patient’s preload responsiveness with a high sensitivity and specificity

When used within its limitations, it is a powerful tool that can correctly answer that important question.....

“Can we use fluid to improve hemodynamics”


Edwards

Helping patients is our life’s work, and

life is now