Pulse Pressure Variation – A Clinical Predictor of Fluid Responsiveness

Application Note

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Today, many clinicians continue to measure a patient’s volume status using standards such as peripheral venous pressure (PVP) and central venous pressure (CVP), along with oxygen saturation, respiratory variables, and urine output. When these methods fail clinically, there is an increased need for other forms of hemodynamic monitoring – including cardiac output (CO), pulmonary arterial occlusion pressure (PAOP) or wedge pressure, pulmonary arterial pressure (PAP), mixed venous oxygen saturation ($SvO_2$), and stroke volume variation (SVV).1

Current clinical methods of assessing a patient’s fluid responsiveness

Over the last several years, the dynamic measurement of pulse pressure variation (PPV) has become the “gold standard” for measuring fluid responsiveness in patients. However, all forms of hemodynamic measurement – static or dynamic – are valuable tools for guiding clinicians in providing optimal care for critically ill patients.

Pulse Pressure Variation (PPV)

The heart and lungs, like other organs in the body, require a continuous supply of blood, oxygen, and nutrients. In healthy individuals, the coronary arteries are the vessels that carry oxygen-rich blood to the heart – specifically to the myocardium or heart muscle. This is because the cardiovascular and pulmonary system automatically increase oxygenation as needed during times of increased heart and respiratory rates. In critically ill patients these normal physiological mechanisms are often impaired, necessitating hemodynamic monitoring and therapies aimed at restoring adequate oxygen supply.2 A lack of cellular oxygenation in critically ill patients leads to irreparable damage of vital organs, which then can lead to multisystem failure and death. Since the heart is located between the lungs and trachea, it is affected by changes in intrathoracic pressure caused by breathing. If a patient breathes spontaneously, even while on assist control ventilation, they can change their intrathoracic pressure and cause exaggerated positive pressure breaths – which will change intrathoracic pressure. Positive pressure ventilation means that airway pressure is applied at the patient’s airway through an endotracheal or tracheostomy tube, allowing air to flow into the airway until the ventilator breath is terminated.1

PPV in transpulmonary blood flow reflects the left ventricular preload variations during three consecutive respiratory cycles. It is also the ability of the cardiac output to increase in response to a fluid challenge and is a dynamic means of assessing a patient’s responsiveness to volume. During inspiration in positive pressure ventilation, pulse pressure (PP) slightly increases. This early increase in PP is caused by an increase in left ventricular preload. The increase of transpulmonary pressure is the net distending pressure applied to the lung by contraction of the inspiratory muscles or by positive-pressure ventilation ($TPP = P_{alv} - P_{pl}$). This effect is especially important in patients with hypervolemia.1,2

When a patient’s heart rate slows after several beats, there will be a decrease in pulse pressure – reducing venous return and RV (right ventricular) preload. The Frank-Starling law states that the stroke volume of the heart increases in response to an increase in the volume of blood in the ventricles before contraction (the end diastolic volume), when all other factors remain constant. A decrease in LV afterload is caused by the increase in pleural pressure, which decreases the systolic LV wall stress and aortic impedance resulting in an increase in LV ejection. Moreover, LV contraction can be supported by the expanding lung. This effect is especially important during left ventricular failure.1

Document purpose

This document has been developed for application training and to help end-users understand the clinical benefits of the pulse pressure variation (PPV) measurement tool available in the Infinity® Acute Care System VG7. This Application Note is for informational purposes only. The user must always exercise his or her own professional judgement and follow applicable hospital protocols and practice guidelines when making treatment decisions.
Why is this important?

One of the most important things to remember is that an early definition of intravascular content is one of the most critical concepts in patient resuscitation. Any factor that decreases left ventricular preload can be associated with PPV amplification, such as hypovolemia, or decrease in the volume of blood plasma.¹

To clinically measure PPV with the IACS VG7 monitor, the patient must have consistent and demonstrable cardiopulmonary interactions. According to the College of Emergency Medicine Physicians, this means that the patient:
- Must be in normal sinus rhythm
- Must be intubated and mechanically ventilated, making no spontaneous respiratory efforts
- Must be ventilated with at least 8 – 10 mL/kg of tidal volume

There are many factors that affect PPV and there is no consensus as to what the exact measurement value range should be. However, many articles suggest that a PPV of between >10 – 15% is associated with volume responsiveness in both the operating room and intensive care unit (ICU) and with a PPV <10% the patient is unlikely to be preload responsive.¹ Therefore, it is recommended that you assess a patient’s PPV over a trended time period and in combination with other hemodynamic information, such as systolic pressure variation (SPV).

SPV is another form of dynamic measurement that allows clinicians to determine the difference between the minimum and maximum value of systolic blood pressure following a single positive pressure breath. In critically ill patients with hypovolemia the SPV is known to increase. With spontaneous breaths a patient’s systolic blood pressure fluctuates 5 mmHg – 10 mmHg. In the critically ill patient on positive pressure ventilation, the response is reverse of a patient who is spontaneously breathing. Remember that mechanical ventilation decreases preload and increases afterload of the right ventricles. The mean of the SPV values during three consecutive breaths is calculated. Systolic Pressure Variation (SPV) (Spmax – Spmin) >5 mmHg the patient is likely to be preload responsive and patients with an SPV <5 mmHg is unlikely to be responsive to fluids.³

How to use PPV in the Infinity® Acute Care System VG7

You can now trend PPV and other hemodynamic values over time using the analysis tools in the IACS VG7 in order to make well informed decisions about patient-centric care and help optimize patient outcomes. PPV calculation is based on the following formula:

\[ \text{PPV} = \frac{(\text{PPmax} - \text{PPmin})}{[(\text{PPmax} + \text{PPmin}) / 2] \times 100} \]

With IACS VG7, it is recommended that the user manually save PPmax and PPmin with the respiratory cycle.⁴

Respiratory and Ventilator Effects

During spontaneous inspiration, pressure waveforms fall. During ventilated inspiration on a positive pressure ventilator, pressure waveforms rise. The systolic pressure is maximal during inspiration and declines during expiration. Similarly, arterial pulse pressure is maximal during inspiration and minimal during expiration.²

Also, since pressure lines are zeroed to atmospheric pressure and therefore do not take intrapleural changes into account, it is necessary to negate the effects of intrapleural pressures when using PPV pressures. It is recommended that you record the value at the end of expiration (PPmin). Best practice on a ventilator is to identify end-expiration, and observe the pressure waveform for respiratory variation and artifact. Doing this will help you determine the end-expiration point, display the respiration or etCO₂ waveform, and observe it simultaneously with the arterial pressure waveform.³

Note: Pulse Pressure (PP) is defined as the difference between systolic and diastolic arterial blood pressure. The type of ventilator and ventilator mode can have different effects on the pressure waveforms. If you are having difficulty determining end-expiration, refer to a textbook on hemodynamic waveform analysis or follow policies of your unit.
PPV Specifications:

- Available on IACS VG 7 and higher
- Manual, dynamic calculation
- No individual alarms
- Available in Trends
- Available in Analysis Tool
- You can adjust the sweep speed, respiratory reference, gridlines, scales, and waveforms to be analyzed
- You can perform as many PPV calculation as you want, but the monitor will use the last 3 measurements in the average
- You can configure device connectivity waveforms as respiratory references
- The product does not associate PPV values with specific waveforms
- PPV is supported on the network, at the Infinity® CentralStation, and in PatientWatch® as discrete parameters (date and timestamp)

References:

Additional references: