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# Assessing Fluid Responsiveness by the Systolic Pressure Variation in Mechanically Ventilated Patients

## *Systolic Pressure Variation as a Guide to Fluid Therapy in Patients with Sepsis-induced Hypotension*

ACHIEVING optimal preload conditions is one of the most important therapeutic goals when circulatory failure is either present or imminent. To date, estimation of the preload status is performed mainly by the measurement of cardiac filling pressures or the left ventricular end-diastolic area using transesophageal echocardiography. In this issue of ANESTHESIOLOGY, Tavernier *et al.*[1] report that, in patients with sepsis who require mechanical ventilation, the systolic pressure variation (SPV) predicts the response to volume loading better than the pulmonary artery occlusion pressure or the left ventricular end-diastolic area.

The SPV, the difference between the maximal and minimal values of the systolic blood pressure (SBP) during a mechanical breath,[2] has been shown to be a sensitive indicator of preload experimentally and clinically.[2–5] In normotensive anesthetized patients, the SPV, which is approximately 8–10 mmHg, is normally composed of two different segments:[2] (1) The delta down (dDown), a decrease of approximately 5–6 mmHg[3,4] in the SBP relative to the SBP during a short apnea, reflects the normally occurring transient inspiratory decrease in venous return. (2) The delta up (dUp), an early inspiratory increase in the SBP of approximately 2–4 mmHg, reflects an augmentation of the stroke output mainly caused by increased left-sided preload. In the presence of congestive heart failure the dUp may be also caused by the afterload-reducing effect of the increased intrathoracic pressure.

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During hypovolemia, the dDown may increase to 20 mmHg, being responsible for nearly all of the SPV.[2,4] As fluids are being administered, the dDown decreases,[1–5] whereas during hypervolemia or congestive heart failure, or both, it practically disappears. Therefore, in the presence of circulatory failure, the dDown can readily differentiate between inadequate preload and cardiac dysfunction. Moreover, the uniqueness of the dDown is its being a dynamic parameter of volume-responsiveness. A steep left ventricular function curve (volume-responsiveness) is associated with a large dDown, whereas a flat curve (nonresponsiveness) is associated with a small dDown. It is therefore that Tavernier *et al.*[1] and others[3,5] found the dDown to reflect the response to volume loading better than the static parameters of preload, such as the pulmonary artery occlusion pressure and the left ventricular end-diastolic area.

Although it is common clinical practice to interpret a high SPV value as a sign of hypovolemia, mere eyeballing of the arterial pressure curve can be misleading. This is true especially during congestive heart failure, when the SPV is composed mainly of a dUp, denoting stroke output augmentation. The introduction of a short apnea for the accurate determination of the dDown and dUp components is somewhat cumbersome clinically.

The SPV cannot be interpreted in the presence of irregular arrhythmias, although nodal rhythm may increase it because of the reduction of effective preload. In the presence of hypotension, the SPV and dDown should be expressed as percentages of the SBP value. The SPV is clinically meaningful only during controlled mechanical ventilation because spontaneous breathing has opposite cardiovascular effects and its variability precludes accurate SPV quantification. The SPV and dDown are directly affected by the magnitude of the tidal volume and can be greatly exaggerated in the presence of air-trapping or reduced chest-wall compliance. Decreased lung compliance should theoretically decrease SPV, although the effects of increased airway pressure and reduced airway transmission



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may cancel each other. Other factors that may theoretically influence the SPV are the arterial elastance and the presence of vasoactive drugs. However, Tavernier *et al.*[1] showed for the first time that the SPV is a useful parameter for a wide range of systemic vascular resistances and drug therapy.

Using the tidal volume as a challenge of the cardiovascular system enables the clinician to easily measure dynamic parameters that reflect volume status and predict the response to volume load. Such true linkage of ventilator and monitor should be automated in the future, possibly contributing to a reduced use of more invasive or expensive, or both, monitoring techniques.

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# *Blood Volume Measurement*

## *The Next Intraoperative Monitor?*

THIS month's issue of ANESTHESIOLOGY features two independent investigations of a new method for measuring circulating blood volume (CBV) at the bedside. In Haruna *et al.*[1] and Iijima *et al.*[2] the new noninvasive method of pulse dye densitometry is postulated as a more practical alternative to traditional measurements of blood volume. This method is "semi-noninvasive" because it necessitates the intravenous injection of indocyanine green dye for each CBV measurement. Indocyanine green dye is rapidly distributed to the circulating compartment and then eliminated by the liver within approximately 20 min. The pulse dye densitometer measures circulating dye concentration *versus* time using two-wavelength light absorption, similar to pulse oximetry. (One of the coauthors is the original inventor of the pulse oximeter: Takuo Aoyagi) The dye elimination curve is back-extrapolated to the "first-pass" time, and the blood volume is calculated as the total dye dose divided by the initial concentration.

In both studies, the new method is compared with two "gold-standard" methods, one of which involves injection of radioactive iodine-labeled albumin. The goal is to compare simultaneous measurements of CBV using the new and old methods and decide whether the new dye method can replace the old gold standard. This is the format of a typical methods comparison study. Whenever we read a methods comparison study, we should

This Editorial View accompanies the following articles: Haruna M, Kumon N, Yahagi N, Watanabe Y, Ishida Y, Kobayashi N, Aoyagi T: Blood volume measurement at the bed-side using ICG pulse spectrophotometry. ANESTHESIOLOGY 1998; 89:1322–8; and Iijima T, Iwao Y, Sankawa H: Circulating blood volume measured by pulse dye-densitometry: Comparison with $^{131}$I-HSA analysis. ANESTHESIOLOGY 1998; 89:1329–35.

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