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A Simple Physiologic Algorithm for Managing Hemodynamics Using Stroke Volume and Stroke Volume Variation: Physiologic Optimization Program

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Abstract

Intravascular volume status and volume responsiveness continue to be important questions for the management of critically ill or injured patients. Goal-directed hemodynamic therapy has been shown to be of benefit to patients with severe sepsis and septic shock, acute lung injury and adult respiratory distress syndrome, and for surgical patients in the operating room. Static measures of fluid status, central venous pressure (CVP), and pulmonary artery occlusion pressure (PAOP) are not useful in predicting volume responsiveness. Stroke volume variation and pulse pressure variation related to changes in stroke volume during positive pressure ventilation predict fluid responsiveness and represent an evolving practice for volume management in the intensive care unit (ICU) or operating room. Adoption of dynamic parameters for volume management has been inconsistent. This manuscript reviews some of the basic physiology regarding the use of stroke volume variation to predict fluid responsiveness in the ICU and operating room. A management algorithm using this physiology is proposed for the critically ill or injured in various settings.

Keywords

stroke volume, stroke volume variation, hemodynamics, fluid responsiveness, challenges

Introduction

Volume status remains an important question in critical care medicine. Traditional methods of assessing volume status based on clinical data are unreliable, especially for intensive care unit (ICU) patients. Static pressure measurements using either the central venous pressure (CVP) or the pulmonary artery occlusion pressure (PAOP) have poor sensitivity and specificity, specifically as measures of volume responsiveness.[1] Advanced hemodynamic monitoring with pulmonary artery catheterization has declined in the ICU for a variety of reasons, lack of patient benefit being the most important.[2-8] Corrected flow time (FTc) can be obtained with esophageal Doppler. Biventricular filling and ejection fraction can be obtained with either transthoracic or transesophageal echocardiography. Both techniques are useful in answering questions about volume status but have limitations especially for continuous assessment.[9-14] Dynamic parameters of fluid responsiveness—stroke volume variation (SVV); pulse pressure variation (PPV); and systolic pressure variation (SPV)—can be obtained from analysis of the arterial waveform. These data have been shown to be more reliable in predicting volume responsiveness than the more traditional static parameters of CVP and PAOP.[15-18] These techniques are particularly useful in the ICU as the majority of patients have indwelling arterial catheters for both continuous blood pressure measurement and blood sampling.

Differences in systolic pressure and pulse pressure are generated by changes in stroke volume (SV) during the respiratory cycle.[19] The amount of variation correlates well with volume responsiveness.[16,20] Multiple clinical examples are well known to bedside practitioners in the ICU using basic physiologic concepts. Pulsus paradoxus and the dramatic changes in blood pressure that are sometimes seen during positive pressure ventilation, especially with large title volumes and high levels of positive end-expiratory pressure (PEEP), are extreme examples of this well-described physiologic phenomena.[21,22]

Basic Physiology of Positive Pressure Ventilation on SV

Figure 1 shows the impact of positive pressure ventilation on SV as reflected by the arterial waveform and describes the

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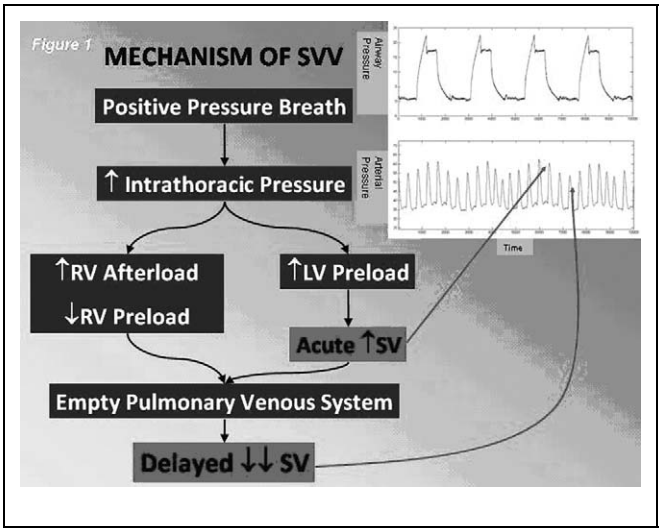

Figure 1.

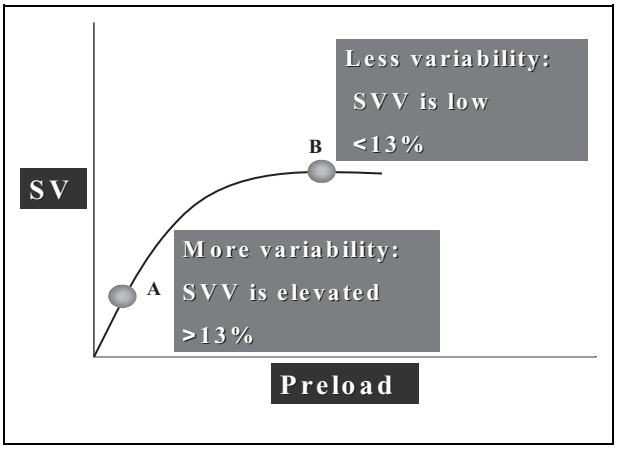

Figure 2. SVV is a dynamic variable that predicts volume responsiveness and individualizes the Starling relationship.
A: volume responsive SVV > 13%
B: non-volume responsive SVV < 13%

mechanism of SVV. The change in SV induced by phasic changes in preload and afterload on both the right and left ventricles are reflected in the arterial pressure waveform and blood pressure.

During a positive pressure breath, there is an increase in intrathoracic pressure as the ventilator cycles. This has the effect of decreasing right ventricular preload while simultaneously increasing right ventricular afterload. The right ventricle is now relatively underfilled as a result of the increase in pleural pressure. This will then show up several cardiac cycles later as left ventricular (LV) output and the SV per beat that ultimately generates the arterial waveform.[23,24] During the positive pressure breath, LV preload is enhanced by a squeezing of blood from the pulmonary capillaries and veins into the left side of the heart. Simultaneously, there is a decrease in LV afterload.[25] This results in an acute increase in LV SV coincident with the inspiratory phase of positive pressure ventilation (Figure 1). The positive pressure breath leaves an empty pulmonary venous system and right heart, causing a decrease in SV and subsequently blood pressure during expiration, as this diminished SV moves from the right to the left side of the heart and is pumped out to the arterial system. This is essentially the opposite physiology of pulsus paradoxus but well known to ICU practitioners treating mechanically ventilated patients. Interestingly, the degree of variability in SV is correlated with the degree of volume responsiveness.[1,16,18,20] It has been shown in several elegant studies that this correlation is linear.[16,20] Coupling the variability with the output of the heart per beat (SV) provides good understanding of both volume responsiveness and volume status under a variety of clinical conditions. Using basic physiologic principles provides foundation for the algorithm that follows.

Background

Stroke volume is the amount of blood in mL ejected from the ventricle with each cardiac contraction. Stroke volume variation is the variation in SV during the respiratory cycle calculated as: (SV max – SV min)/SV mean. Pulse pressure variation and SPV are calculated in a similar fashion. Stroke volume variation represents the dynamic change on right and LV performance by the influence of changing pleural pressure during positive pressure ventilation on ventricular filling and ejection. Stroke volume variation allows greater precision when uszing the Starling relationship to optimize cardiac performance by localizing an individual's position on their unique Starling curve. High variation in SV during the respiratory cycle corresponds to the steep part of the Starling curve, and these patients should respond to preload augmentation. Small variations in SV during positive pressure ventilation usually place the patient on the flat part of the curve where improvement in cardiac performance is not expected with further preload expansion (Figures 2 and 3).[16,18,26,27] Exceptions to these general rules will be described later in this manuscript.

The relationship of the arterial pulse waveform to SV can be thought of in terms of a hydraulic system of pipes (the vasculature) where the fluid (blood) is propelled by a pump (the heart). It is easy to then conceptualize that the characteristics of any waveform in the vascular system under static conditions (that does not exist in humans) is entirely determined by the amount of volume entering the circuit (SV) with each ejection of the pump. The human cardiovascular system is dynamic and consideration of vascular compliance, tone, capacitance, wave dampening, and reflection further complicate this simple analogy, but the basic concept will facilitate the understanding of waveform analysis as it is presently used to estimate SV.[28,29]

Estimation of PPV and SPV can be done by making simple measurements at the bedside observing the arterial pressure tracing on the bedside monitor. Detailed analysis of the pressure tracing allows accuracy and discrimination. Commercial systems are now available that display the variation on the bedside monitor (Phillips IntelliVue MP90).[30] Other commercial products that provide more advanced hemodynamics including cardiac output, SV, and SVV are available: PiCCO,[31-33]

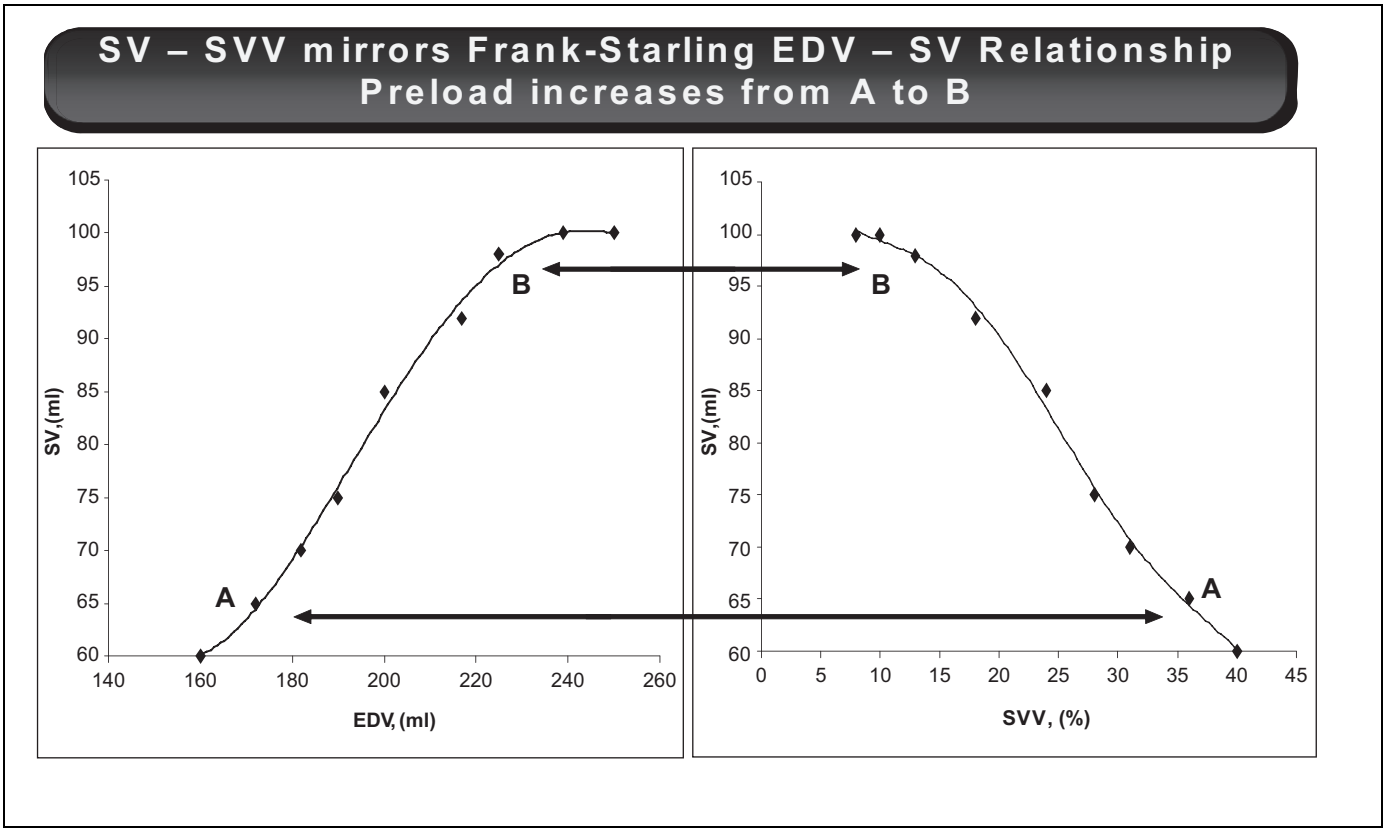

Figure 3. Simultaneously developed Frank-Starling curve with the corresponding SV-SVV relationship in an individual patient undergoing volume resuscitation. The SV-SVV relationship mirrors the Frank-Starling curve: as preload increased, cardiac performance reaches a plateau, similarly declining SVV is associated with improved cardiac performance. SVV-SV pairs can provide information about cardiac performance similar to a Frank-Starling curve. EDV = end diastolic volume.

LiDCO,[34-36] and the Edwards FloTrac Vigileo System.[37,38]. The FloTrac System also has the advantage of not requiring calibration or any other additional invasive technology beyond an arterial catheter.

A Simple Physiologic Algorithm Using Stroke Index and SVV

Physiologic optimization of cardiac performance is a critically important aspect of hemodynamic support. A physiologic algorithm has been developed using the concept of SVV as a predictor of volume responsiveness and indexed SV as the cardiac performance measure. Figure 4 shows the physiologic optimization program as it is currently being used in critically ill patients.

The ability of SVV to predict fluid responsiveness is not binary but more typically linear. Multiple studies have used a value of 10% to 15% for PPV or SVV to discriminate between responder and nonresponders.[1,12,16,20,26] For this algorithm, we simply chose a number (13%) within this range of values. Patients with lower values of SVV often do respond to a fluid challenge, although usually to a lesser and often clinically insignificant degree.[1,16,20] Critical questions around volume status initiate use of this algorithm in the clinical setting.[39,26] A daily challenge in the ICU is the approach to hypotension and/or oliguria. Basic concepts for use of this algorithm will now be described.

Volume Responsive Patients: SVV > 13%

An elevated SVV suggests volume responsiveness. Stroke volume variation greater than 13% provides discrimination between responsive and nonresponsive patients to a fluid challenge. Dynamic physiologic changes are best observed with a significant rapid volume challenge. Begin with a minimum of 1 L normal saline over 10 to 15 minutes. Colloid including blood or larger or lesser amounts of crystalloid may be used depending on the clinical scenario. Because of the influence of heart rate on cardiac output, the preference is to use stroke index (SI) data as the measure of cardiac performance. Stroke volume variation and SI can be obtained on a continuous basis and provide ongoing hemodynamic assessment of the preferred physiologic approach.

Nonvolume Responsive Patients: SVV < 13%

In patients with SVV less than 13% (nonvolume responsive patients), SI is evaluated. The clinical setting becomes even

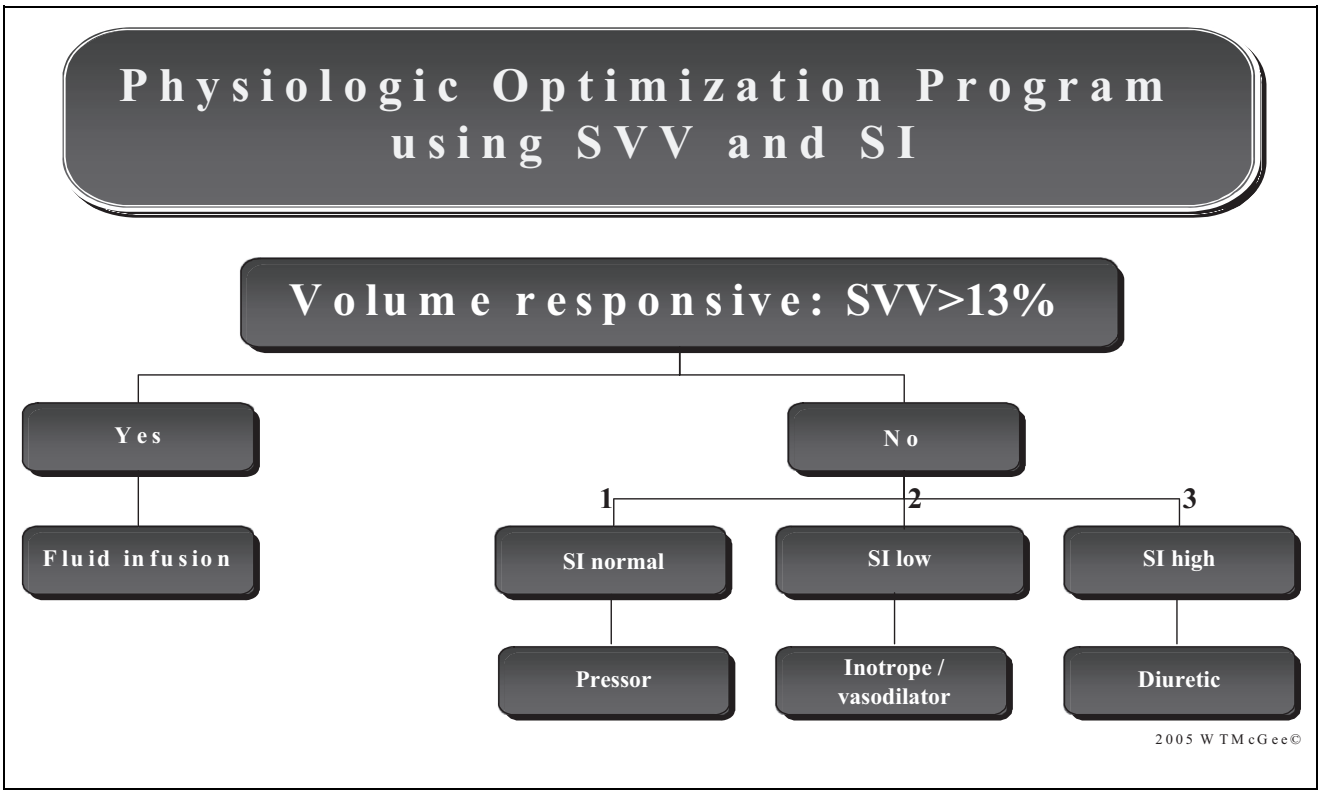

Figure 4. Pathways 1,2,3 utilize the stroke index(SI) along with SVV and are described in detail in the text.

more important in this circumstance to aid application of the most appropriate intervention based on assessment of the underlying pathophysiology. It is worth emphasizing that the algorithm is an adjunct to clinical assessment and not the other way around (Figure 5).

Pathway 1

When SI is normal or increased as typically occurs in a hyperdynamic patient with severe sepsis or septic shock, a vasopressor may be indicated. Often these patients maintain a good urinary output despite low blood pressure and an agent that increases vascular tone is indicated (Figure 5). For example, a well-resuscitated patient with septic shock remains hypotensive despite 8 L of crystalloid infusion within the first 4 hours of ICU admission. Stroke index is 50 mL per bt/m^2, and SV variation is 7%. Further fluid therapy is stopped and a vasopressor is titrated to a mean arterial pressure goal of 65.

Pathway 2

Patients in whom there is no further response to volume and clinically have pulmonary congestion may benefit from inotropic or vasodilator therapy. A tolerable systemic pressure will influence which specific agent is chosen. Most patients receiving inotropic or vasodilator therapy via this algorithm have a clinical diagnosis of congestive heart failure and "wet" lungs on evaluation. For those patients who fall into this category

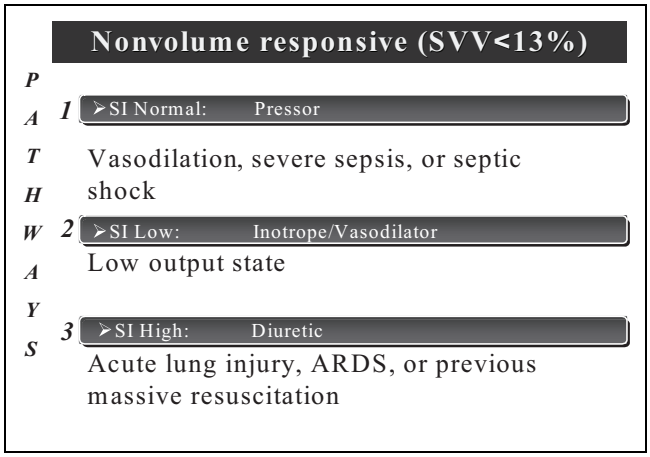

Figure 5. For non-volume responsive patients, the clinical impression along with the stroke index directs therapy.

with relatively clear lungs and/or a reasonably low A-a gradient, subsequent fluid boluses may be appropriate. If there is no response in cardiac performance, then this approach should be abandoned and the patient simply placed on an inotrope or vasodilator. Echocardiographic assessment of LV function, especially for patients with suspected diastolic dysfunction, will further refine hemodynamic management, in this subset of patients (Figure 5). For example, a patient with a history of congestive heart failure and known systolic dysfunction is admitted with shortness of breath, pulmonary edema, and

hypotension. Stroke index is 20 mL per bt/m^2 with simultaneous SVV of 6%. A 500-mL bolus does not appreciably change these data. The patient is then placed on dobutamine, with improvement in stroke index to 31 mL per bt/m^2 without change in SVV. Blood pressure is also improved with inotropic therapy.

Pathway 3

If the SI is high, especially for those patients who have acute lung injury, diuretic therapy used fairly aggressively is directed at keeping the lungs dry.[40,41] There is a clear advantage to keeping patients with acute lung injury (ALI)/acute respiratory distress syndrome (ARDS) dry rather than wet, and the finding of an elevated SI provides reassurance for the use of diuretic therapy. Although not validated through an experiment, a focus on flow data (SI) will better reflect organ perfusion than evaluating the static pressure parameters, CVP <9 or PAOP <13, used in the Fluid and Catheter Treatment Trial (FACTT).[41] Without lung injury, the value of diuretics is less clear, but volume should be minimized, and diuretics may be useful, especially in patients with prior massive resuscitation (Figure 5). For example, a patient with ARDS secondary to severe pancreatitis has been massively volume resuscitated during their initial ICU course. Fluid balance is 20 L after 4 days and oxygenation is poor. Stroke index is 52 mL per bt/m^2 with a SVV of 4%. Diuretics are given until SI drops to 40 mL per bt/m^2 with a consequent improvement in oxygenation and lung compliance.

Variations on the Basic Physiologic Algorithm

In the operating room, application of this approach has been useful to optimize fluid management.[42] The operating room provides ideal conditions for the use of this algorithm as the patients are paralyzed and mechanically ventilated typically with no acute lung disease and using larger title volumes.[1] These are the experimental conditions under which SVV as a predictor of volume responsiveness has the most validity.[43] In the operating room, the algorithm can be significantly simplified to Figure 6. Titrated therapy with volume and vasopressors is used allowing optimization of hemodynamic management without over or under resuscitation. No mortality or renal failure was observed. Physiologic endpoints provide reassurance of appropriate vasopressor use and volume loading.[42,44] The targeted volume management (TVM) approach using FTc and SV with the CardioQ monitor in the operating room is similar, and has been associated with improved outcomes.[44,45]

Acute Lung Injury or ARDS

The majority of patients with ALI/ARDS are mechanically ventilated and have an arterial catheter. The FACTT trial emphasizes the importance of volume management in this population.[41] Stroke index provides a physiologic goal that allows confident use of diuretics, while assured of adequate flow. In the FACTT trial, although there was no mortality benefit, a 2-day difference in ICU and hospital length of stay for those

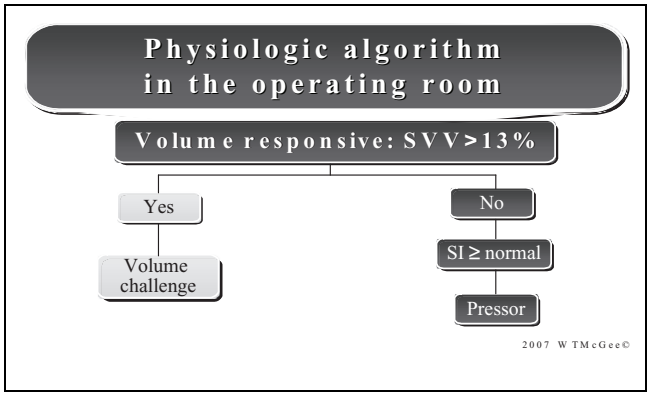

Figure 6. In the operating room, volume is infused, withheld, or the patient is temporarily placed on pressors (when indicated) which are typically weaned by post-op recovery. Diuretics are not used and inotropes only rarely.

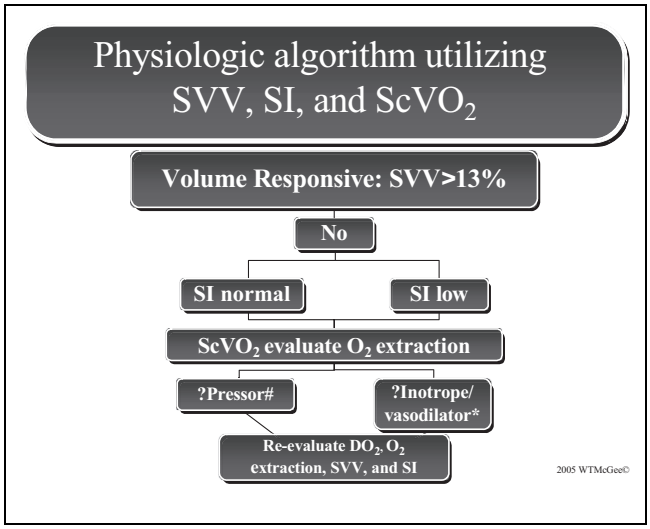

Figure 7. * - If O_2 extraction is high, an inotrope may be required to provide perfusion support.
- As individual organ perfusion may also depend on blood pressure, a MAP target > 60-65 may require a vasopressor even when O_2 extraction is normal.

patients kept "dry" (conservative fluid management) was shown. Reduction in length of stay was accomplished with titrated volume management using Furosemide targeting[41] CVP <9 or PAOP <13.

Identification of Adequate O_2 Delivery

Perhaps the most challenging patients identified in the ICU through advanced hemodynamic monitoring using this approach is the group whose SVV is less than 13%, but otherwise have either normal or low SI. The clinician must decide whether DO_2 (oxygen delivery) is adequate on an individual basis. Coupling the flow parameters, SVV and SI, with O_2 extraction using central venous oxygen saturation ($ScvO_2$) aids determination of the adequacy of DO_2 (Figure 7). Using O_2

extraction allows determination of whether a particular level of DO_2 is adequate for an individual patient under present conditions. Specifically, if O_2 extraction is low or normal (<33%), the argument that DO_2 should be increased is difficult.[6,7,46-51] Alternatively, when O_2 extraction is high (40% or greater), vigorous attempts at increasing DO_2 should be employed, simply on a physiologic basis. This is the basic resuscitation paradigm. It is clear that as O_2 extraction increases, physiologic reserve is compromised, lactic acidosis ensues, and mortality results.[52-58] Using O_2 extraction as an endpoint further refines hemodynamic care of this severely ill group of patients.

For those patients in whom these values do not provide an answer (O_2 extraction between 33% and 40%), best clinical management remains unclear. Complete assessment of DO_2 should be determined as a first step. For the majority of patients, using clinical assessment of physiology as it relates to individuals, frequent reassessment along with consideration of multiple other clinical parameters would typically lead to a watchful waiting approach or more aggressive resuscitation (Figure 7).

In applying these algorithms at the bedside in the ICU or operating room, the use of sound physiologic principles guides management in a group of patients in whom advanced hemodynamic monitoring can be easily and safely obtained.

Important Values

Central venous oxygen saturation allows individualized assessment of the balance between O_2 delivery and O_2 consumption. High O_2 extraction is represented by a low value of $ScVO_2$ and can often be corrected with improvement in oxygen delivery.[57] Central venous oxygen saturation is not identical to SvO_2; the former is superior vena cava oxygen saturation, and the latter is mixed venous oxygen saturation; however, in general, they provide similar data about the adequacy of perfusion.[57]

The Limitations of SVV

All of these systems have limitations, which is beyond the scope of this manuscript. In those systems that rely on interpretation of the arterial waveform, the quality of the waveform is critical. Severe aortic insufficiency or the presence of an intraaortic balloon pump would not allow acquisition of reliable data. During irregular rhythms, especially atrial fibrillation, SV changes on a beat-to-beat basis. Stroke volume variation is increased and, under these conditions, will not predict fluid responsiveness. It is still possible to look at trends in SV and cardiac output, but this limits use of the algorithm.

Delta Pleural Pressure

The concept of SVV is tightly coupled to the respiratory cycle and the change in pleural pressure during a mechanical breath. In Figure 1 (which shows airway pressure simultaneously displayed with blood pressure), there is a fairly significant change in pleural pressure during inspiration. As the pleural pressure changes are limited either by smaller tidal volume or poor lung compliance, the effect on venous return and subsequently SV is diminished.[43,59-63] In the extreme example for patients being oscillated, SVV has been seen either at or near zero. Despite very low SVV, these patients may be significantly fluid responsive. There are several ways to assess for fluid responsiveness with this situation at the bedside. This introduces the concept of providing a challenge to the patient and then observing their response. One challenge is simply to increase tidal volume.

Recruitable SV

Recruitable SV can be defined as fluid responsiveness and improvement in SV in patients who otherwise are not expected to be volume responsive (SVV <13%) by the use of challenges. The concept of recruitable SV can be elucidated by challenges. In the situation of a patient being ventilated with low tidal volume, the cardiopulmonary system can simply be challenged by increasing the tidal volume. In a graded fashion, the tidal volume may be increased by >100%. Start with a 50% increase in the tidal volume and evaluate the impact of the increased tidal volume on SVV. For those patients whose variation is low and who otherwise require volume, this increase in tidal volume may precipitate a significant degree of SVV. The maximum permissible increase in tidal volume must be determined by observation at the bedside of both airway pressures and hemodynamics. As most of these monitoring systems have a fairly rapid response time, it is possible to obtain this information while standing at the bedside and manipulating the ventilator.

Another challenge that has use in the ICU is passive leg raising. This technique has been described elsewhere[64] and involves raising the legs to at least 45° for 30 seconds to no more than several minutes. In a significant percentage of volume responsive critically ill patients with shock, this maneuver will predict fluid responsiveness with a high degree of sensitivity and specificity.[65,66] If SV improves significantly with this maneuver, the patient will also respond to a volume challenge.[65,66] The dynamic parameters of PPV and SVV will also predict volume responsiveness under these conditions, but the change in SV is the more important outcome parameter.[65,66] In those patients without a response to leg raising, improvement in SV with a fluid challenge is not anticipated. This improvement in cardiac performance will often not be reflected in blood pressure due to changes in vascular tone caused by raising the legs, making it particularly important to focus on SV, when employing this reversible volume challenge. In those patients in whom a volume challenge may be deleterious, that is, lung injury, renal failure, or clinical volume overload, this reversible volume challenge is safe.

Other Considerations

This algorithm has been found to have the most use in mechanically ventilated patients.[42,60,61] These concepts can be applied in nonventilated, spontaneously breathing patients as long as the underlying physiology and principles are considered. Most

published literature does not support this.[60,62,67] However, a good understanding of the underlying physiology does allow application in specific circumstances. If there is a small pleural pressure change related to patient effort or lung physiology, the impact on venous return is expected to be small and the use of SVV as a predictor of fluid responsiveness is limited.[60,63] Conversely, spontaneously breathing patients with a high SVV and low SV would be expected to be volume responsive. There are reasons that both SVV and SV could be increased. Kussmal respirations or a patient with status asthmaticus and large pleural pressure swings but with excellent underlying cardiac physiology and good volume status are some examples. As the cardiac performance measure that is important is the SV, further volume resuscitation may not be indicated despite the increased SVV.

Summary

In summary, applying physiology at the bedside using analysis of SVV and SV allows better discrimination of hemodynamic function and fluid responsiveness than simply looking at blood pressure, CVP, PAOP, or one or the other of these parameters by itself. These data typically supplement clinical impression and are not intended to replace clinical evaluation. It is important to at least have a conceptual physiologic framework on how to use this information so that its limitations can be fully understood. Some of these limitations can be circumvented by the use of challenges as described above. The use of dynamic parameters of fluid responsiveness along with flow data at the bedside in the critically ill has been a welcome addition to our evaluation of these patients. Evaluation of perfusion using O_2 extraction provides a fairly complete description of hemodynamic physiology that further refines clinical care.

There are several major caveats that must be considered when using this data to assess perfusion adequacy. Central venous oxygen saturation represents the venous effluent of the top half of the body with the only major organ influence being the brain. Despite this, there has been reasonable correlation with this value and SvO_2 in a variety of clinical circumstances.[55] O_2 extraction as measured by the saturation of central venous blood includes only perfused tissues. Finally, organ-specific perfusion cannot be assessed when using a global parameter such as $ScvO_2$. Furthermore, the failure of cellular energetics and the ability of mitochondria to use oxygen may provide false reassurance that cellular oxygenation is adequate.[68,69] Despite these limitations, there is no data that suggests that improving global O_2 delivery when global measurements of O_2 extraction are adequate improves patient outcome.[7,8,47,48]

Conclusion

Analysis of the arterial waveform and sampling of central venous blood through a central line can provide advanced hemodynamic monitoring using fairly typical ICU interventions, namely a central venous catheter and an arterial line without the need for more invasive pulmonary artery catheterization. The Physiologic Optimization Program employs basic physiology to optimize cardiac performance from these data.

Declaration of Conflicting Interests

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