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Contemporary hemodynamic monitoring, fluid responsiveness, volume optimization, and endpoints of resuscitation: an AAST critical care committee clinical consensus

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ABSTRACT

This article, on hemodynamic monitoring, fluid responsiveness, volume assessment, and endpoints of resuscitation, is part of a compendium of guidelines provided by the AAST (American Association for the Surgery of Trauma) critical care committee. The intention of these guidelines is to inform practitioners with practical clinical guidance. To do this effectively and contemporarily, expert consensus via the critical care committee was obtained. Strict guideline methodology such a GRADE (Grading of Recommendations Assessment, Development and Evaluation) was purposefully NOT used so as not to limit the possible clinical guidance. The critical care committee foresees this methodology as practically valuable to the bedside clinician.

INTRODUCTION

Intravascular volume optimization has been found to improve critical care outcomes. There is a delicate balance between hypovolemia (and hypoperfusion) and volume overload, which is equally associated with complications. Contemporary hemodynamic monitoring aims to offer providers with objective guidance as to their patient's actual volume status.

PURPOSE

Volume status assessment is measured via a compilation of "endpoints of resuscitation." Contemporarily, there is no perfect endpoint of resuscitation, each with their limitations, and thus current practice involves an assimilation of multiple endpoints of resuscitation into an overall assessment. The purpose of this guideline is to review the commonly used endpoints of resuscitation, providing an understanding of the mechanism of measure, accuracy, and potential limitations.

END POINTS OF RESUSCITATION

1. Heart rate, blood pressure, and urine output
2. Serum lactate
3. Central venous pressure (CVP)
4. Mixed venous oxygen saturation (SvO₂)
5. Pulmonary artery catheterization (PAC)
6. Blood flow variation secondary to respiratory variation

7. Echocardiography

DISCUSSION

Heart rate/blood pressure/urine output

Heart rate, blood pressure, and urine output are basic vital signs that can be obtained non-invasively. Variations in heart rate can be one of the earliest signs of hypovolemia and malperfusion. Blood pressure and urine output provide added qualification for suspected diagnoses but are delayed in their presentation. There are many limitations to using these endpoints of resuscitation in isolation. However, due to their ease of acquisition, they form the basis of basic patient assessment and should be supplemented by other elements to be discussed in the following sections.

Recommendation: Heart rate, blood pressure, and urine output should be monitored in all patients who are undergoing resuscitation. These endpoints must be used in context of the individual patient and their limitations considered thoroughly.

Serum lactate

Serum lactate is a biomarker of global tissue malperfusion. Lactic acidosis is the most common type of metabolic acidosis present in hospitalized patients. Assuming normal cellular respiration, abnormal lactate levels can be assumed to be based on abnormal oxygen delivery. When poor oxygen delivery is due to hypovolemia and/or low cardiac output, lactate can be used to guide resuscitative efforts.

Serum lactate is a component of many resuscitative algorithms including the Surviving Sepsis Campaign and the sepsis3 pathways as described by the Society of Critical Care Medicine.¹⁻³ The optimal frequency in which to follow serum lactate is unclear. Every 6 hours until normal has been well described but newer, point-of-care devices allow for a much higher frequency, the value of which is yet to be seen definitively in the literature. Initial serum lactate in the setting of septic shock and its rate of clearance are strongly predictive of survival.⁴⁻⁶

Recommendation: Serum lactate should be followed serially during a resuscitation until normalized. The frequency of these serial assessments should be no longer than every 6 hours but where appropriate, higher frequencies may be beneficial. Serum lactate should not be used in isolation.

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CENTRAL VENOUS PRESSURE

CVP approximates right atrial pressure and therefore RV end-diastolic volume. This can be further extrapolated to estimate left ventricular (LV) end-diastolic pressure and volume.⁷ CVP ultimately estimates the LV stroke volume, which is the closest approximation of the overall intravascular volume state, and for which the classic Starling principle applies.

While increasing stroke volume is the goal of any fluid challenge,⁸ the relationship between ventricular filling pressures and ventricular volume⁹ is not linear, due in part to diastolic dysfunction and altered ventricular compliance during critical illness. Additionally, many disease states such as pulmonary hypertension and congestive heart failure can elevate CVP regardless of the actual, underlying volume status. Thus, the use of CVP in isolation, especially as a static number, may yield inaccurate estimations of volume status.^{7 8 10 11} In one study, using CVP to guide fluid challenge in septic patients showed that a CVP <8 mm Hg predicted fluid responsiveness only 47% of the time, and the area under the receiver operating curve for predicting responsiveness was only 0.63.¹² Due to the lack of easy availability of other more accurate measures, CVP is still used by a significant proportion of critical care providers worldwide.^{13 14}

Recommendation: CVP is highly flawed and not recommended for use as a static value. It can be trended over time for improved efficacy. It should also not be used in isolation.

SvO₂ and ScvO₂

Two markers of oxygen utilization, the central venous oxygen saturation from the superior vena cava (ScvO₂), and the SvO₂ from the proximal pulmonary artery, have been used to guide fluid administration and resuscitation.¹⁵ Use of these markers for volume assessment is based on the concept that the oxygen content of mixed venous blood returning from the body is dependent on the amount of oxygen delivered to tissues on the arterial side (as measured by arterial oxygen content and CO), as expressed in the Fick equation (assuming tissue oxygen consumption remains constant). Since CO may be influenced by a fluid challenge (by raising stroke volume), the SvO₂ and ScvO₂ have been used as a surrogate parameter for volume responsiveness.

ScvO₂ is about 2%–5% less than SvO₂ in healthy persons¹⁶ due to contribution of more highly oxygenated venous return from the kidneys, but may be higher than SvO₂ during critical illness¹⁷ and measures only the venous saturation from the upper part of the body since it uses a blood sample from the superior vena cava. During the 1980s and 1990s when PAC use was more ubiquitous, several small series studies suggested venous saturation could be used as an indicator of the degree of blood loss,¹⁸ and a therapeutic target in the multiply injured.¹⁹ More recently, the change in ScvO₂ after volume expansion in 30 critically ill patients was noted to correlate with changes in cardiac index, with a change of 4% indicating a fluid responsive state.²⁰ Another small prospective study demonstrated that in responders to a fluid challenge, the response was marked by an increase in ScvO₂ that also correlated with changes in CI ($r=0.702$, $p<0.001$).²¹

Mixed venous oxygen has also been studied in surgical patients, suggesting goal directed therapy intraoperatively reduced organ dysfunction and hospital length of stay.²² Another surgical prospective randomized study showed that an ScvO₂ threshold of about 71% was useful in predicting complications.²³

Although broader studies on early goal-directed therapy have revealed controversial results,^{24–30} mixed venous oxygen saturation specifically was not directly assessed. More research

is needed in trauma and other non-sepsis surgical patients to assess SvO₂ and ScvO₂ in terms of definitively guiding fluid responsiveness.

Recommendation: A helpful adjunct and can be used similarly to serum lactate. Should not be used in isolation.

Pulmonary artery catheterization

PACs (AKA Swan-Ganz catheters) are an invasive central line placed through the right side of the heart. Pressure measurements along its length allows for direct measurement of CVP, pulmonary artery pressure, pulmonary capillary occlusion (wedge) pressure, and CO. PACs also allow for indirect measurement of systemic vascular resistance, stroke volume, and oxygen delivery among others. PACs can be used to differentiate among various shock states and mechanisms of pulmonary edema as well as optimizing cardiogenic shock in particular. PACs are not subject to error secondary to irregular heart rhythms or valvular disease.

Routine use of PACs has fallen out of favor based on a multitude of more contemporary studies suggesting common misinterpretation of PAC pressures and waveforms.^{31–35} This included significant inter-observer variability in interpretation that led to over or under-estimation of wedge pressure in particular. PACs are also associated with various complications related to central line placement, traversing the right heart, and the PAC position including pulmonary artery rupture.³⁶ With the advent of less-invasive hemodynamic monitoring, the use of PACs has further decreased.

Recommendation: Cautioned use as experience in today's practitioners is low, especially in determining the wedge pressure. Select increased efficacy in certain cardiac shock situations. Generally, not used as a first-line adjunct but more useful when other endpoints of resuscitation are conflicting.

Passive leg raise

Passive leg raising is a dynamic measurement used to assess if a patient will respond to a fluid bolus. With the passive leg raise, the bottom half of the bed is raised to a 45° angle above the patient's head, or the patient's legs are raised manually. The idea is that this maneuver will increase preload by transferring blood that has pooled in the patient's lower extremities to the chest. If the blood pressure rises within 60s, the patient will most likely respond favorably to a fluid bolus.³⁷ The usefulness of this technique is limited in patient with increased intra-abdominal pressure (due to ascites or compartment syndrome).

Recommendation: Helpful adjunct when an actual fluid bolus may be detrimental. Simple and non-invasive. Strong recommendation for use.

Stroke volume variation

Stroke volume variation (SVV) is based on the difference in intrathoracic pressure between inspiration and expiration in patients on positive pressure ventilation. Positive pressure ventilation reduces preload by increasing intrathoracic pressure. This may translate to a reduced stroke volume. The larger the difference in intrathoracic pressure between expiration and inspiration, the more likely the patient will respond favorably to a fluid bolus. Using an intra-arterial line tracing, the pulse pressure variation (PPV) can be calculated. Many modern monitors are able to calculate this value automatically. For values greater than 12%, the patient will most likely respond to a fluid bolus. Less than 8%, the patient is most likely euvolemic. Between 8% and 12%, fluid responsiveness may or may not be present. Using other clinical indications such as urine output or CVP could help

give insight as to whether fluids might help in this gray zone. Additionally, giving an intravascular fluid bolus and assessing for any change in the patient's PPV can also suggest further fluid responsiveness.^{38–40} There are several variations to PPV based on measured characteristics in the change in blood flow throughout the respiratory cycle, these include SVV and stroke volume index (SVI).

In order for PPV to be used effectively, the patient should have very regular respirations, such as a controlled ventilation mode (volume or pressure). If the patient is spontaneously breathing, the PPV will not be accurate. Also, the patient must have a sufficient tidal volume to force a display of change in stroke volume. Low stretch (lung protective) ventilator settings preclude adequate measure of SVV. Additionally, the patient must be in a sinus rhythm. Irregular rhythms, such as atrial fibrillation, will not give accurate arterial tracings because preload will change with each beat. RV failure will also not allow prediction of fluid responsiveness since adding fluid to the RV may not translate to an increase stroke volume in the LV.

PPV/SVV can be measured via various methods of blood pressure assessment. This includes conventional arterial blood pressure (invasively and non-invasively), pulse contour CO, bioimpedance, and esophageal Doppler. Bioimpedance estimates the water content and composition of the body using electrical current flow and resistance. Alternating electric current passes through the body via fluid (directly) and across cell membranes (indirectly). The volume of water in the body determines the width of the passage through which electricity flows. The passage width determines the flow of electric current, and is referred to as impedance. This, along with the body's total resistance, can then be used to estimate total body water using electrodes on the outside of the body. The procedure is non-invasive, and the results are instantaneous and reproducible. Bioimpedance is ideal for tracking total body water content over time especially when these values may be in constant flux, such as during a dialysis procedure. Esophageal Doppler is a small ultrasound probe inserted into the esophagus. The probe measures the velocity of the blood flow in the descending aorta. Stroke volume and CO along with SVV and SVI can be calculated with this method. These values can then be used to directly guide fluid management during surgery or at the bedside in a critical care setting. However, if the patient's esophagus is inaccessible (eg, trauma, presences of strictures, thrombocytopenia), this modality may not be optimal.

Recommendation: PPV/SVV is helpful in correlation with other endpoints of resuscitation. They should not be used in isolation. Limitations such as irregular heart rhythms or low tidal volumes must be recognized.

Echocardiography

Bedside "hemodynamic" ultrasonography (u/s) for assessment of fluid responsiveness and basic cardiac function in intensive care unit (ICU) setting is becoming an increasingly used adjunct to hemodynamic assessment. The definition of fluid responsiveness is the ability to increase CO in response to a fluid challenge. Global ventricular size, wall motion, and function suggest not only fluid responsiveness but also potential responsiveness to vaso-active medications as well. Thus, ultrasonography can facilitate hemodynamic optimization. Additionally, as an endpoint of resuscitation, echocardiography is advantageous in that it avoids the need for invasive lines and probes.

Hemodynamic ultrasonography can elucidate measures to improve CO in several ways:

1. Volume status optimisation by inferior vena cava diameter variation (IVCDV) throughout the respiratory cycle
Similar to the concept of PPV and SVV, IVCDV measure changes in IVC size and infers that if the size varies with respiration, the patient will be fluid responsive. In patients without spontaneous respirations, IVDV predicts fluid responsiveness (positive LR, 5.3; 95% CI, 1.1 to 27; pooled specificity, 85%).⁴¹ There is less sensitivity when breathing is more erratic, a similar limitation to other measures of fluid responsiveness based on respiratory variation.
2. Volume status optimisation by ventricular size and function
When small, LV size is predictive of fluid responsiveness. A hyperdynamic LV with an end-diastolic area of less than 10 cm² or papillary apposition (kissing ventricles) is strongly indicative of hypovolemia. Significant narrowing of the outflow tract during systole can be induced by under filling of the LV in patients, particularly when in a hyperdynamic state such as sepsis. Echocardiography can also measure SVV which is a good indicator of fluid responsiveness. Using an equation for flow volume and an apical five-chamber view, SVV of more than 12% accurately predicts fluid responsiveness with values over 14% having a very high positive predictive value and less than 10% a high negative predictive value.⁴²
3. Functional status/contractility optimisation
Echocardiographic assessment of LV ejection fraction is feasible and accurate. Despite adequate fluid resuscitation (filling pressures), myocardial contractility may be altered in various physiologic states such as sepsis. The use of vasopressors to improve LV contractility may therefore be beneficial in certain patient populations. The use of echocardiography to recognize poor contractility can guide resuscitative efforts.^{38 43}

Additionally, right ventricular (RV) failure secondary to elevated pressures within the pulmonary circulation may lead to inadequate LV filling. In the ICU, this may occur with chronic heart and lung disease or use of elevated positive end expiratory pressures to improve oxygenation. Diagnosis of RV dysfunction and/or failure can be achieved with echocardiography demonstrating RV dilation and dysfunction. If RV failure is due to elevated afterload, the use of a prostaglandin analog (eg, Esoprostenol Rx) a potent pulmonary vasodilator can be used. If RV failure is due to volume overload, diuresis ± an inotrope may be beneficial.

Recommendation: Bedside echocardiography is rapidly becoming a highly valued bedside measure but has an extended learning curve, both with image acquisition and interpretation. It also requires a rigorous learning curriculum.

A comment on mitigating over-resuscitation

As the ProMISE, ProCESS, and aRISE trials revealed, waiting to throttle-back a resuscitation until ALL endpoints of resuscitation are met will likely result in over-resuscitation and possibly hypervolemia.^{27 29 44} Almost all of the current endpoints of resuscitation only measure hypovolemia and give no assessment of the range including euvolemia and hypervolemia. The two measures that may be helpful in this respect are echocardiography and the chest X-ray.

Recommendation: It is imperative that endpoints are tracked real time and that as the endpoints approach a normal range, the resuscitation be titrated accordingly, so as not to overshoot euvolemia.

Summary

A synopsis of contemporary endpoints of resuscitation, their clinical uses and limitations

	Mechanism of assessment	Limitations	Ability to assess hypovolemia
HR/BP/UOP	Indirect measure	Many confounding factors	No
Serum lactate	Malperfusion causing anaerobic cellular respiration	Decreased clearance in liver disease	No
CVP	Estimate of right-sided filling pressure	Many things falsely elevate CVP including pulmonary HTN and CHF	No
SvO ₂	Measure of oxygen delivery	Systemic shunting may falsely elevate	No
PAC	Cardiac filling pressures and thermo-dilution	Invasive. Waveforms subject to interpretation	Possibly via elevated PA pressures
PPV/SVV	Cardiac output variation secondary to the respiratory cycle	Irregular HR; low tidal volumes; spontaneous respiration	No
Echocardiography	Direct measure of ventricular filling and cardiac function	Image acquisition and interpretation; learning curve	Possible via RV size and lack of change in caval size with respiration

BP, blood pressure; CHF, congestive heart failure; CVP, central venous pressure; HR, heart rate; HTN, hypertension; PPV, pulse pressure variation; RV, right ventricle; SvO₂, mixed venous oxygen saturation; SVV, stroke volume variation; UOP, urine output.

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