Direct measurement of physiologic systems is often impractical. To overcome these obstacles, indirect physiologic measures have been developed. Indirect physiologic measures such as heart rate, blood pressure, and many others are surrogates that are believed to accurately represent the function of a physiologic system.

Although a powerful tool, physiologic measurement has several potential limitations and errors. This can result in erroneous instrument data. For that reason, it is the responsibility of the clinician to question and interpret monitor output and to ultimately correctly assess validity of the measurement. This article reviews commonly used intraoperative monitoring techniques and discusses their potential limitations as they relate to hypovolemia and hemorrhagic shock.

Keywords: Hemodynamic monitoring, hypovolemia, measurements.

Hemodynamic Measurement in the Operating Room: A Review of Conventional Measures to Identify Hypovolemia

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Direct measurement of physiologic systems is often impractical. Placement of monitoring equipment in vital structures, such as the brain or heart, is often unrealistic due to the possibility of exposing the patient to potential trauma, infection, and nosocomial morbidity. To that end, indirect physiologic measures have been developed. These indirect measures are surrogates that are believed to accurately represent the function of a physiologic system.

The goal of hemodynamic monitoring in the operating room is to provide insight, or “a window,” into tissue perfusion. Perfusion is elusive and difficult to measure because it encompasses multidimensional variables such as heart rate, cardiac contractility, systemic vascular tone, and intravascular status. Changes to any of these variables will cause a cascade of compensatory mechanisms. One of the many clinical intraoperative challenges is to identify and treat physiologic derangements, like hemorrhagic shock, before pathology ensues. To that end, surrogate measures have been developed to measure organ perfusion. This article will review and evaluate common perioperative physiologic instruments that are currently being used to measure intravascular volume and perfusion during hemorrhagic shock.

Pathophysiology of Hemorrhagic Shock

Hemorrhagic shock is the second leading cause of death due to traumatic injury. Hemorrhagic shock, however, is not limited to traumatic injury, nor is it the only concern of those who care for trauma-affected patients. Uncontrolled bleeding occurs every day in perioperative care. Often, the only difference between uncontrolled hemorrhage from patients with trauma and those without trauma is the cause of the bleeding. Regardless of the cause, the goal of the anesthetist is to maintain and, in many circumstances, restore perfusion.

Massive injury and overt hemorrhage are easily identified and managed. Contrary to this, subacute injuries and blood volume depletion are less obvious and often go unnoticed. It is the undiagnosed, hypovolemic patient who presents the greatest challenge to the anesthetist. Essentially, this is because the symptoms can go unrecognized. Delayed resuscitation can increase perioperative morbidity and mortality.

Hemorrhagic or hypovolemic shock is a result of lost blood volume exceeding the body’s ability to compensate, which can ultimately lead to hypoperfusion and cellular hypoxia. The physiologic response to depleted blood volume is a complex and dynamic process. Although hypovolemia does not equate to shock, it does share a similar pathology and physiologic compensatory mechanisms. Hypovolemia has a very deliberate pathological progression that activates both a rapid and a slow response. Decreased intravascular volume causes baroreceptors in the great vessels, heart, and carotid bodies to become activated. This activation stimulates the afferent sympathetic nervous system and the vasomotor center of the medulla. Catecholamines released from neurons in the efferent sympathetic nervous system stimulate adrenergic receptors. These receptors trigger constriction of vascular smooth muscle and increasing systemic vascular resistance (SVR), cardiac inotropy, and chronotropy, with the goal of improving perfusion pressure.

As hypovolemia worsens and the glomerular filtration rate (GFR) is reduced, the juxtaglomerular cells of the af-
fert arterioles release renin. Renin begins a negative feedback loop, with the intent of reversing hypovolemia via the renin-angiotensinogen-aldosterone system (RAAS). The primary function of the RAAS is to produce angiotensin II. This potent vasoconstrictor acts directly on the vascular smooth muscle. In addition, angiotensin II indirectly acts by increasing the release of SNS neurotransmitter and reducing the metabolism of neurotransmitter yielding an increased SVR and improved perfusion pressure.

Aldosterone, antidiuretic hormone, and atrial natriuretic peptide (ANP) are released to increase fluid reabsorption. While the release of neuroendocrine hormone occurs with the sympathetic response, it is a time-dependent process, in many cases requiring more than 24 hours to increase volume.

The influence of hypovolemia at the level of the tissue is variable based on volume deficit, the patient’s pathology, and metabolic demand of the tissue. In the early stages, decreased intravascular volume causes blood to shunt away from “ischemia-tolerant” tissues. Although many patients can tolerate this for an extended time, if uncorrected, the shunting blood can lead to anaerobic respiration and the accumulation of metabolic byproducts such as lactate.

Assuming normal cardiac function, blood pressure can be viewed as a dynamic continuum between intravascular volume and systemic vascular resistance. In a nonseptic state, this relationship is inversely proportional. As intravascular volume decreases (ie, dehydration and hemorrhage), vascular tone increases (vasoconstriction) to support and maintain perfusion. It is the body’s ability to compensate during hypovolemic states that creates the greatest challenge to recognizing hemorrhage. Essentially, the body will appear euvolemic (near “normal” vital signs) until it is no longer able to compensate. At that point, however, the patient will already have been suffering substantial metabolic debt. Clinicians commonly fail to notice the subtle signs and symptoms of hypovolemia and hemorrhagic shock. One study estimates that as many as 50% of clinicians’ evaluations of intravascular volume are incorrect. The Figure illustrates the dynamic process of compensated hemorrhagic shock.

**Surrogate Measures**

Several surrogate measures are used in clinical practice to estimate the degree of hemorrhagic shock. These measures include a variety of techniques with the intent of determining intravascular pressure, blood volume, and ultimately perfusion. Typically, perfusion is evaluated by measurement of blood pressure. Tissue oxygenation is essentially dependent on adequate perfusion, as well as arterial oxygen tension. Unfortunately, normal blood pressure does not always equate to adequate perfusion. Although this relationship maintains homeostasis, it can be misdiagnosed, leading to severe morbidity and ultimately mortality.

**Specific Measures as They Relate to Hypovolemia**

- **Heart Rate.** Tachycardia is considered one of the classic symptoms to identify hypovolemia. Interestingly, heart rate, by itself, plays a small role in maintaining
blood pressure. Because of this, heart rate is not a sensitive indicator for identifying individuals who are hypovolemic and potentially in “shock.” This finding is consistent with the traditional teaching that tachycardia is a late symptom of hypovolemia. Table 1 lists the 4 stages of hemorrhagic shock. It has been estimated that changes in blood volume in excess of 15% to 30% may lead to changes in heart rate. The late and often irregular response of heart rate to changes in blood volume makes heart rate an ineffective measure. It is of little wonder that as many as 50% of clinician estimations of blood volume, based on routine clinical markers such as heart rate, are incorrect.

- Blood Pressure. Peripheral blood pressure (PBP) is a commonly used measure to assess intravascular volume and perfusion. Both peripheral arterial and oscillometric measurements are commonly used in the operating room. Unfortunately, the accuracy of these instruments, as they relate to aortic pressure, has not been well documented. The disparity between arterial and noninvasive pressure is alarming. The significance of this is relatively unknown. What is clear, is that the farther from the heart that blood pressure measurement is taken, the less accurate the reading. Causes for these discrepancies are numerous, including patient age, vascular distensibility, patient position, clinician error, and faulty instrument mathematical modeling.

Measurement of PBP is a poor early predictor for hypovolemia. Several researchers have demonstrated that patients remain normotensive despite large changes in blood volume. This inaccuracy can best be explained as follows: an outflow from the sympathetic nervous system causes vasoconstriction and redistribution of blood flow from the periphery, maintaining increased aortic pressures. Despite common belief, hypotension does not correlate with the onset of hypovolemia but is rather a delayed indication.

- Central venous pressure (CVP). The CVP is a routinely monitored hemodynamic variable that clinicians correlate with intravascular volume status. Like heart rate and blood pressure, research has demonstrated that CVP is an inaccurate predictor of intravascular volume. Cardiovascular compensatory mechanisms that are activated during hypovolemia can falsely elevate CVP by changing SVR and increasing venous return to the right side of the heart. Additionally, myocardial compliance and therapeutic interventions (mechanical ventilation) are common factors that affect intraoperative CVP measurement.

Despite evidence to the contrary, CVP continues to be used as a primary indicator for intravascular volume. Its use appears dogmatic. Although many clinicians do not rely on the actual measurement, they do consider CVP trends. Trending is regularly discussed in clinical practice. Essentially, trending measurements are thought to provide insight into volume status or cardiovascular function. The conventional wisdom is that increases or decreases in CVP translate to changes in volume status. Unfortunately, the literature in this area is poorly developed, and this practice is without basis. Trends in CVP are rarely consistent when resuscitating a patient, employing technical modes of ventilation, or changing surgical position. Researchers have noted that “trending” is highly provider dependent and has not been clearly articulated in the literature.

- Urine Output. Urine output has been clinically used as a surrogate marker for perfusion and fluid management. The premise for the use of urine output is that perfusion to the kidney will be reduced in hypovolemic states. Despite the regular use of urine output, scarce data exist to support its accuracy and/or influence on patient outcome. Urine output has not been shown to be predictive of perfusion or kidney function.

Factors that can influence the precision of urine output include renal calculi, extrarenal obstruction of urinary flow, abdominal insufflations, and patient position. Additionally, diuretics, alcohol, and changes in circadian rhythm can confound the clinical picture. Urine output is insensitive when identifying early hypoperfusion and hypovolemia, and is often normal despite changes in volume. Similar to other methods used to assess vascular status, physiologic compensation can maintain renal perfusion and urine output despite hypovolemia, hypoperfusion, and ischemia.

- Shock Index (SI). The SI is the ratio of heart rate divided by systolic blood pressure (SBP). This noninvasive measure uses existing data reported in the literature more than a decade ago. An SI greater than 0.7 is considered abnormal and highly correlates with hypovolemia. Repeatedly, the SI has been demonstrated to correlate

<table>
<thead>
<tr>
<th>Stage</th>
<th>Blood volume deficit</th>
<th>Mental status</th>
<th>Heart rate</th>
<th>Blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>≤ 15%</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>II</td>
<td>&lt; 30%</td>
<td>Anxious</td>
<td>Normal to mildly elevated</td>
<td>Normal</td>
</tr>
<tr>
<td>III</td>
<td>&lt; 40%</td>
<td>Anxious</td>
<td>Tachycardia</td>
<td>Hypotension</td>
</tr>
<tr>
<td>IV</td>
<td>≥ 40%</td>
<td>Change in mental status</td>
<td>Tachycardia</td>
<td>Severe hypotension</td>
</tr>
</tbody>
</table>

Table 1. Stages of Hemorrhagic Shock

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with serum lactate and with morbidity and mortality from hemorrhagic shock.31-34 It has been suggested that the SI is more sensitive to early acute hypovolemia than either heart rate or blood pressure alone.34

- **Transesophageal Echocardiography**. Transesophageal echocardiography (TEE) has been used in practice since the early 1990s. It provides clinicians with the ability to evaluate cardiovascular filling and global cardiac function. Two physiologic structures have been evaluated as markers to identify hypovolemia. They are the inferior vena cava and the ventricular orifice at end diastole.35,36 To this point, there has not been any comparison between TEE and blood volume analysis using validated standards such as the tracer dilution technique.

A TEE evaluation is clinician dependent, making its reliability questionable. Probe placement and patient position are only 2 of the variables that may influence TEE imaging and evaluation. Whereas research has demonstrated the usefulness of TEE in guiding resuscitation compared with other measures (pulmonary artery pressure and CVP), there have been no formal reports of TEE sensitivity and association with hypovolemia.37

- **Transesophageal Doppler**. Transesophageal Doppler measures acoustic characteristics of blood flow as it is ejected from the left ventricle and as it passes through the descending aorta. Several authors have demonstrated substantial reductions in morbidity and mortality when they used transesophageal Doppler as a method for goal-directed fluid therapy, yet its place in resuscitation has not been determined.34 Measured data are translated to cardiac output. The accuracy of transesophageal Doppler as it relates to cardiac output is well established.3,4 Although transesophageal Doppler is highly accurate and precise, like TEE, it is prone to error if not used properly.3 Most notably, precision is influenced by probe position. Small variations in probe position or rotation can render the measurement unreliable. Despite high technical requirements, transesophageal Doppler has been shown to be effective in identifying and managing intraoperative hypovolemia.3,4,38-40

- **Tracer Dye Analysis**. Dilutional techniques have been in practice for longer than a century. The tracer dye analysis method of evaluating intravascular volume involves injecting a known tracer substance into the intravascular space and measuring the diluted concentration from blood samples. The dilution technique is a direct measure of intravascular volume.41-44 It is the gold standard for accurately determining blood volume.41 Several products have recently been introduced into the marketplace with the intent of alleviating some of the limitations of tracer dye analysis. For instance, the LiDCO plus system (LiDCO Ltd, Cambridge, United Kingdom) is a blood volume analyzer that uses intravenous lithium to measure intrathoracic blood volume. Although still early in investigation, the data show promise that this system and similar technologies will bring rapid, accurate blood volume analysis to the operating room. One study demonstrated that use of this particular system reduced hospitalization by 12 days when used in patients who underwent major abdominal surgery.45

- **Arterial Pulse Pressure Variation**. Arterial pulse pressure variation (aPPV) has been evaluated for its use in

### Table 2. Summary of Common Hemodynamic Measures for Evaluating Hypovolemia

<table>
<thead>
<tr>
<th>Measure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Typically requires &gt;15% of blood volume for HR change to occur. Victims of traumatic injury often present with tachycardia due to pain and sympathetic nervous system outflow.</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Primarily augmented by SVR. Changes to blood pressure occur between 15% and 30% of blood volume loss. Normotension does not equate to perfusion.</td>
</tr>
<tr>
<td>CVP</td>
<td>Invasive. Pressure measurements are influenced by SVR, patient position, myocardial contractility, and intrathoracic and extrathoracic pressures.</td>
</tr>
<tr>
<td>Urine output</td>
<td>Poor indicator of renal perfusion and blood volume.</td>
</tr>
<tr>
<td>Shock index</td>
<td>SI = HR/SBP. Correlates with serum lactate and morbidity and mortality from data from large national trauma registry.</td>
</tr>
<tr>
<td>TEE</td>
<td>Diagnostic tool. Not a routine monitoring technique. Highly provider dependent. Anatomic evaluation of IVC and ventricular orifice may provide insight into intravascular volume.</td>
</tr>
<tr>
<td>TD</td>
<td>Has been successfully used for goal-directed fluid therapy, but its use has not been demonstrated for resuscitation after severe hemorrhage.</td>
</tr>
<tr>
<td>aPPV</td>
<td>Has not been studied in hemorrhagic shock. It is not clearly understood how vasoconstriction alters aPPV.</td>
</tr>
</tbody>
</table>
identifying hypovolemia. The aPPV is the difference between the maximum and minimum pulse pressure after 1 positive pressure ventilation.\(^4\)\(^6\) Its accuracy to identify hypovolemia remains debatable.\(^4\)\(^6\)-\(^4\)\(^8\) Variation of pulse pressure has been identified during vasoconstricted, hypovolemic states as well as in pharmacologically induced vasodilatation during euvolemia. The unresolved issue with regard to this measure is the definition of pulse variation.

Preliminary data suggest that aPPV may be predictive of fluid responsiveness in suspected hypovolemia, yet not in uncontrolled hypovolemia.\(^5\)\(^6\)\(^9\) The work by Solus-Biguenet and colleagues,\(^4\)\(^6\) like many others in the area, is useful as a starting point but does not yet address several issues that relate to precision. Outstanding issues to be addressed include: how do vascular compliance (ie, atherosclerosis), patient position, and modes of positive pressure ventilation influence changes in pulsation?

**Discussion**

The physiology of intravascular volume and perfusion makes hypovolemia and hemorrhagic shock a challenging clinical problem to manage. Alterations in blood volume produce reflexive vascular and cardiac changes that allow for patient physiology to appear near euvolemic. The clinician is left to decipher the data and decide whether the patient is “wet” or “dry.” Unfortunately, clinician subjectivity is inadequate. Even the most seasoned provider can overlook the subtle signs of hypovolemia and withhold treatment.\(^9\) The impact of missed treatment, at this point, is unknown.

Several monitoring instruments are available and used in clinical practice. Table 2 summarizes these measures and techniques. Many measures such as heart rate, blood pressure, urine output, and CVP are relatively simplistic and readily used. Other measuring devices, such as TEE, transesophageal Doppler, and tracer dye analysis are labor intensive and/or require a high degree of training. With the variety of options, the clinician must ultimately decide which device will provide the most reliable data in regard to accuracy, precision, sensitivity, and specificity. By pooling the strengths and weaknesses of each method or instrument and the data available, the clinician should be able to recognize and manage hypovolemia and hemorrhagic shock more rapidly and to the benefit of both the patient and the provider.

Based on the literature, several observations and recommendations can be made with regard to intraoperative hemodynamic monitoring systems. No monitoring technique is a reliable, early predictor of hypovolemia or hemorrhagic shock. The more technically advanced instruments and related measures, such as TEE, transesophageal Doppler, and tracer dye analysis, provide increased accuracy, yet are currently less practical for routine use and patient screening.

Individually, CVP, urine output, blood pressure, and heart rate are inadequate. These instruments and related measures evaluate only 1 aspect of intravascular volume, which limits the conclusions that can be drawn. In contrast, the SI provides more depth when evaluating intravascular volume. Because it uses existing data and is easily employed, the SI is an attractive adjunct for clinical problem solving.

**Summary**

Hypovolemia is a commonly misdiagnosed problem facing the anesthetist. Reduced intravascular volume causes redistribution of blood flow and ultimately may lead to end-organ ischemia. Intraoperative fluid therapy appears to positively influence patient outcome, yet the data are limited in anesthesia practice.

Hemodynamic monitors provide a window for clinicians to evaluate their patients. Currently, there is no ideal way to immediately identify and thus treat this type of shock. There is no perfect monitoring technique. Future research is promising, yet at this time, clinicians are tasked with managing these complexities with the tools that are available. For successful management of these patients, it is imperative that the anesthetist understands the strengths and, more importantly, the limitations of physiologic instruments when managing likely episodes of hypovolemic and hemorrhagic shock.

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