Pitfalls of Hemodynamic Monitoring in Patients with Trauma

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INTRODUCTION

Resuscitation of the patient with trauma with ongoing blood loss has historically been geared toward maintaining normal or higher than normal blood pressure. Investigations of the role of hypotensive resuscitation have been conducted with mixed results.

KEYWORDS

- Trauma • Hemodynamic monitoring • Hemorrhagic shock
- Blood pressure measurement • Dynamic response • Natural frequency
- End-tidal carbon dioxide • Assessment of cardiac output

KEY POINTS

- Hypotensive resuscitation imposes additional burdens on the anesthesiologist to maintain adequate blood pressure while also avoiding blood pressure overshoots or sustained hypertension.
- Accuracy and interpretation of blood pressure monitoring in trauma depend on complex interactions between patient physiology and factors intrinsic to measurement devices.
- Oscillometry overestimates systolic blood pressure in hypotensive patients.
- The impact of hemorrhagic shock on reflection wave behavior may limit the reliability or confound interpretation of both noninvasive and invasive blood pressure measurements.
- Adequate dynamic response is required for proper recording of an arterial waveform, and dynamic response requirements increase with tachycardia.
- The only way to determine dynamic response is to conduct a flush test.
- End-tidal CO2 is correlated with cardiac output; however, this correlation is affected following cardiac or respiratory arrest.

INTRODUCTION

Resuscitation of the patient with trauma with ongoing blood loss has historically been geared toward maintaining normal or higher than normal blood pressure. Investigations of the role of hypotensive resuscitation have been conducted with mixed results.
A review of 52 animal studies of mortality during induced hemorrhage found that hypotensive resuscitation reduced the risk of death. However, in human studies, decreased blood loss and transfusion requirements have only been confirmed in penetrating injury. Other studies are currently in progress and may add evidence to support a paradigm shift in the management of blood pressure in the patient with trauma. It may no longer be sufficient to maintain blood pressure at an adequate level, and added vigilance may be needed to prevent sustained overshoots in blood pressure. It is therefore ironic that the monitoring strategies meant to inform the anesthesiologist of a patient’s hemodynamic status are limited in the context of hypotension and the deranged physiology associated with hemorrhagic shock.

This article discusses the complex interplay between alterations in the physiology of a patient with trauma, the monitoring devices engineered to measure these alterations, and interpretation of these data by the anesthesiologist. Focus is placed on the early stages of caring for the patient with trauma before surgical hemostasis is obtained, when hemodynamic assessment must be made quickly and accurately to maintain sufficient cardiac output and blood pressure without exceeding levels that may worsen outcome. Hemodynamic monitoring and resuscitation end points of patients in the period after surgical hemostasis have been reviewed elsewhere and are only mentioned here. For example, monitoring strategies based on arterial pressure waveform variation, venous oxygen saturation ($SvO_2$) and central venous oxygen saturation ($ScvO_2$), and laboratory parameters such as base excess and lactate have been, or are increasingly becoming, a part of the trauma anesthesiologist’s armamentarium, but it is often not feasible to implement these strategies during the initial, frenetic stages of care. The 3 bread-and-butter monitors heavily relied on during the initial assessment and resuscitation of the unstable patient with trauma are arguably the end-tidal pressure of CO$_2$ (PETCO$_2$), noninvasive blood pressure (NIBP), and invasive arterial blood pressure (IABP). The underpinnings and limitations of these monitors in the patient with trauma are discussed in reverse order starting with invasive blood pressure, acknowledging that the other two may be the only hemodynamic monitors available before an arterial catheter can be inserted. Wherever possible, figures are included to illustrate important points from the text.

**Invasive Blood Pressure Monitoring**

When using IABP to guide the management of patients with trauma, it is incumbent on the physician to discern true signal (the physiologic waveform) from artifact (a distorted waveform). The physiologic waveform should be processed with high fidelity to obtain an accurate recording. Several characteristics of the patient with trauma and of the recording device can lead to the generation of distorted (or what appears to be distorted) waveform morphology. Patients with trauma are likely to exhibit tachycardia, hypotension, decreased cardiac output, and high (or occasionally low) systemic vascular resistance (SVR). These derangements in patient physiology can exceed the processing capability of the catheter-tube-transducer (CTT) system used to invasively measure blood pressure. Factors that affect waveform morphology can lead to the display of inaccurate blood pressure values and potentially confound clinical interpretation. Two of the main factors are pulse wave reflection within arterial vasculature and the dynamic response characteristics inherent to the CTT system.

To understand why these factors are important, it is useful to recall that any physiologic waveform is decomposable into several sine waves of varying amplitude, frequency, and phase (Fig. 1). The reverse process is also true, and sign waves can be selected and summed to generate a simulated waveform. It takes just 3 sine waves to give a persuasive approximation of the typical arterial waveform. Of the 3 sine waves
Fig. 1. Generation of simulated arterial waveform from sine waves. Summation of 3 sine waves (A), 1 fundamental (sine wave 1) with frequency equal to pulse rate and 2 harmonics (sine waves 2 and 3) produces a good approximation of an arterial waveform at a pulse rate of 150 beats per minute (B). Sine waves of higher frequency relative to pulse rate are required to produce detailed features in the waveform such as the dicrotic notch and steep systolic upstroke; change in pressure (dP)/change in time (dt) (arrows). The simulation was performed using custom scripts written in MATLAB, and sine wave frequencies and amplitudes from Szockik and colleagues (2000). (Data from Szockik JF, Barker SJ, Tremper KK. Fundamental principles of monitoring instrumentation. In: Miller RD, editor. Anesthesia. 5th edition. Philadelphia: Churchill Livingstone; 2000. p. 1053.)

(see Fig. 1A), the first has the lowest frequency, which corresponds with the heart rate. The second and third sine waves have successively higher frequencies and lower amplitudes, which give the simulated waveform finer features such as the dicrotic notch. The second and third sine waves are required to simulate the systolic upstroke (see Fig. 1B).
Steeper upstrokes require the summation of sine waves of even higher frequencies. The CTT system must be able to process these higher frequencies to avoid waveform distortion (discussed later). The concept of waveform summation is also applicable to the morphology of the physiologic arterial waveform, which is determined by the overlap of pulse waves generated by the heart and waves reflected from the arterioles.

**Pulse wave reflection**

Reflection waves are not abnormal, but are important contributors to the arterial waveform in most animals. When blood is ejected from the heart, pulse pressure waves generated in the ascending aorta propagate toward the periphery at a velocity that is faster than the flow wave. On reaching distal arteriolar beds, the incident pulse wave is reflected, generating a retrograde pulse wave. The incident and reflected pulse waves overlap (summate) to form a new composite waveform (Fig. 2). However,

![Fig. 2](image)

**Fig. 2.** Demonstration of the effect of pulse wave reflection on arterial waveform morphology. The simulated arterial waveform from Fig. 1 is shown with its reflection (A). Summation of the incident and reflected wave produces a composite arterial waveform with systolic and diastolic waves (B). When pulse wave velocity is increased, as in hypertension, the reflected wave overlaps sooner (C) yielding a characteristic late systolic peak (arrow) and diminished diastolic wave. Hypotension is associated with slower pulse wave velocities, which leads to overlap of reflected waves later in the cardiac cycle (D), and large diastolic waves are seen (arrow). These simulations were performed using custom scripts written in MATLAB.
in the vasculature, the final arterial waveform is the summation of reflected waves from all arteriolar beds, with reflected waves sometimes being rereflected several times. Wave reflection is important in maintaining normal blood pressure and organ perfusion. Three factors influence the effect of wave reflection on the final arterial waveform: distance to reflection points (arteriolar beds), pulse wave velocity, and degree of vasocostriction. The typical distance a pulse wave has to travel before it is reflected depends on the location of the arteriolar bed. For example, the distance from the heart to the brachial bed is about 0.5 m. In a normal adult, pulse waves travel about 10 m/s, with the velocity varying directly with the arterial blood pressure. Reflected waves typically have amplitudes of 80% of the originating waveform amplitude. In young patients, diastolic blood pressure (DBP) is increased by reflected waves overlapping during diastole, and this helps maintain coronary perfusion. However, in elderly and hypertensive patients, pulse wave velocity is increased because of blood pressure and decreased arterial distensibility. This increased velocity allows the reflected wave to arrive sooner and overlap with the incident wave, leading to increased systolic blood pressure (SBP), decreased DBP, and the formation of a characteristic late systolic peak in the arterial waveform (see Fig. 2C, arrow).

Pulse wave reflection is particularly important in explaining the arterial waveform in the setting of hemorrhagic shock, which is associated with hypotension, tachycardia, and increased arteriolar vasoconstriction. At low blood pressures, pulse wave velocity can be significantly decreased, whereas the duration of systole is decreased in tachycardia. Vasoconstriction is a common physiologic response to shock and is often manipulated with pharmacotherapy by the anesthesiologist. Conditions that decrease SVR, such as sepsis or pharmacologic vasodilation, minimize the degree of wave reflection. However, as noted earlier, increased vasoconstriction in response to shock or administration of peripheral vasoconstrictors maximizes wave reflection. The net effect of hypotension, tachycardia, and vasoconstriction contributes to the generation of a composite arterial waveform with a characteristic large diastolic wave (see arrow in Fig. 2D and waveform in Fig. 3B), and, at times, a slowed systolic upstroke.

Central aortic pressure may be overestimated by up to 20% if SBP is measured with a peripheral catheter. This is because, in addition to the factors already discussed, waveform morphology depends strongly on the location of measurement, as shown by the observed differences in waveform appearance at the dorsalis pedis relative to the radial artery catheter site. These differences are attributed to overlap of incident, reflected, and rereflected waves from various arteriolar beds, and to the contribution of reflected waves to systolic pressure occurring earlier as measurements are recorded further from the heart. Therefore, many factors contribute to the morphology of the arterial waveform (Table 1), and the anesthesiologist must be careful to consider these factors when interpreting information obtained from arterial CTT systems. In particular, SBP overestimation should be avoided by considering SBP in conjunction with mean arterial pressure (MAP), because MAP is not significantly affected by reflection waves and measurement site.

The impact of trauma pathophysiology on the contribution of reflected waves to the arterial waveform can lead to incorrect identification of waveform distortion. Contrary to popular opinion, it is not possible to determine whether a recorded arterial waveform is overdamped or underdamped simply by casually observing waveform morphology (see Fig. 3). The only way to determine whether a recorded waveform is distorted is to assess the dynamic response of the CTT system. This assessment can be accomplished at the bedside by performing a flush test, or in simulations by introducing a square wave and analyzing the resulting oscillating waves.
Fig. 3. Two arterial waveform recordings typically seen in trauma. The first waveform could be incorrectly considered to be underdamped (A), whereas the second might be considered to be overdamped (B). However, the dynamic response characteristics underlying each recording were satisfactory.

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<th>Table 1</th>
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Abbreviations: n/a, not applicable; O, overestimated; U, underestimated; V, variable effect.
<sup>a</sup> Possible unreliability caused by group-specific differences in amplitude envelope.
<sup>b</sup> The algorithm used in oscillometry to calculate blood pressures is affected in part by arterial waveform morphology and pulse rate.
Dynamic response of the IABP monitoring system

CTT systems have been shown to behave like underdamped second-order systems. As such, the dynamic response of the CTT can be characterized in terms of its natural frequency and damping coefficient. This dynamic response depends on the mass of fluid in the tubing, the compliance of the tubing, and friction to fluid movement in the tubing. Clinical CTT systems are designed with short, thin, noncompliant tubing with the goal of obtaining an optimal dynamic response. The anesthesiologist can determine these values for a particular CTT system by performing a flush test. The resulting oscillating waveform is then analyzed to obtain the natural frequency of oscillation and the amplitude ratio of the first 2 oscillation peaks (for details see Mark [1998]). Next, a plot of natural frequency versus damping coefficient (Fig. 4) is referenced to determine whether the dynamic response is adequate to allow proper processing of the physiologic waveform. If the dynamic response is not adequate, the measured waveform may be distorted. The fallacy in attempting to determine dynamic response by casual observation of the waveform was shown by Gardner (1981) on analysis of waveforms from 37 patients. He showed that it is possible to have a waveform that appears to be underdamped when its dynamic response is determined to be overdamped, and vice versa.11

Introduction of a bubble into the tubing or the use of a resonance overshoot eliminator (ROSE) damping device (Spectramed, Oxnard, CA) are strategies commonly used to correct an underdamped CTT system. However, introduction of an air bubble increases damping and simultaneously decreases natural frequency, which may do little to improve, or may even worsen, the dynamic response of an underdamped system (see a in Fig. 4). Furthermore, an optimal dynamic response can inadvertently be made suboptimal (see b in Fig. 4), increasing the likelihood that the SBP measurement will be inaccurate. The ROSE damping device has been shown to increase the

Fig. 4. How natural frequency, damping coefficient, and patient physiology interact to define optimum dynamic response requirement. As either heart rate or the slope of the systolic upstroke (dP/dt) increases, the natural frequency necessary to allow proper processing of the arterial waveform increases and the range of damping coefficient associated with optimum dynamic response decreases (compare vertical arrow bars). Presence of a bubble increases damping coefficient and decreases natural frequency (a and b).
damping coefficient while having little effect on natural frequency, and therefore may improve measurement accuracy of an underdamped system. However, resonance may still occur at high heart rates (a common occurrence in patients with trauma) and with steep systolic upstroke.\textsuperscript{15}

In the case in which dynamic response is suboptimal because of low natural frequency, the recorded waveform may become distorted because of resonance and appear to be underdamped. A recorded waveform appears to resonate when the frequencies of sine waves that compose the physiologic waveform approach the natural frequency of the CTT system. The effect is increased in an underdamped system. A simple demonstration helps to explain the interaction between the natural frequency of the CTT system and the sine waves that make up the arterial waveform. \textbf{Fig. 5} shows the effect of resonance on the simulated arterial waveform constructed from 3 sine waves from \textbf{Fig. 1}. The frequencies of these sine waves increase with heart rate. For example, at a heart rate of 60 beats per minute (bpm), sine waves 1, 2, and 3, have frequencies of 1, 2, and 4 Hz, respectively. These frequencies are increased to 2.5, 5.0, and 10 Hz, respectively, when the heart rate is 150 bpm. At the higher heart rate, sine waves 2 and 3 resonate in a CTT system with natural frequencies of 5 (see \textbf{Fig. 5A}) and 10 Hz (see \textbf{Fig. 5B}), respectively. The effect of resonance is to distort the recorded waveform such that SBP is overestimated by 10 to 30 mm Hg and the diastolic pressure is underestimated, whereas the effect on MAP is not significant.\textsuperscript{11}

The dynamic response plot published by Gardner\textsuperscript{11} (1981) (see \textbf{Fig. 4}) was derived from analysis of only 2 physiologic waveforms. One waveform was thought to represent the general patient population and had a heart rate of 94 bpm. The other waveform (118 bpm) was chosen because it required the greatest dynamic response attributed to its steep systolic upstroke. Although there are no experiments that address this in the literature, it is feasible that physiologic waveforms obtained from patients with trauma in hemorrhagic shock with heart rates of 150 bpm or more and increased ventricular contractility may exceed the capability of the CTT system. Future research may help to determine whether this is the case and, if so, consideration for the design of CTT systems with natural frequencies more relevant to the trauma setting may be warranted. This possibility is especially germane when considering the potential role of hypotensive resuscitation in the management of patients with trauma. In the meantime, anesthesiologists should rely more on monitoring MAP than SBP, because MAP measurements are the least affected by the dynamic response characteristics of the CTT system.

Current CTT systems are claimed to have natural frequency between 10 and 20 Hz.\textsuperscript{5} At our institution, we have taken steps to optimize the dynamic response of our CTT systems. We now perform flush tests on the CTT system when we use invasive monitoring. We have found that the natural frequency is usually between 10 and 20 Hz, as advertised (\textbf{Fig. 6}). When extension tubing is used (eg, dorsalis pedis site), the natural frequency is reduced about 50\% (on occasion, we have observed natural frequencies of less than 5 Hz). Based on our observations, we now instruct residents not to use extension tubing, but instead to place the CTT transducer close to the catheter site. However, in considering that the pulse waveform of unstable patients with trauma may demand higher dynamic response than is advertised on out-of-the-box CTT systems, we have changed our practice further by removing any unnecessary tubing between the transducer and the catheter insertion site. By performing this modification, the natural frequency is usually doubled (see \textbf{Fig. 6}). We are currently investigating whether these modifications improve the fidelity of recorded waveforms in the setting of hemorrhagic shock.
NIBP monitoring is likely to be relied on to manage the patient with acute trauma in prehospital and emergency room settings. In the operating room, NIBP can be valuable in assessing hemodynamic status and effects of fluid and pharmacologic treatments until invasive monitoring is established. NIBP is easy to implement as long as an extremity is available for cuff placement. It is common for a patient with trauma to sustain injury in both upper extremities and, in such cases, cuff placement at the thigh or ankle provides MAP values that correlate well with values obtained at the

**Fig. 5.** The effect of resonance on arterial waveform morphology. Resonance occurs when a harmonic frequency of the physiologic waveform approaches the natural frequency of a particular CTT system. Two examples are shown using the first (A) and second (B) harmonics from Fig. 1. In both cases, amplification of the harmonic wave is observed in the recorded waveform, and SBPs and DBPs are overestimated and underestimated, respectively.

**NIBP Monitoring**

NIBP monitoring is likely to be relied on to manage the patient with acute trauma in prehospital and emergency room settings. In the operating room, NIBP can be valuable in assessing hemodynamic status and effects of fluid and pharmacologic treatments until invasive monitoring is established. NIBP is easy to implement as long as an extremity is available for cuff placement. It is common for a patient with trauma to sustain injury in both upper extremities and, in such cases, cuff placement at the thigh or ankle provides MAP values that correlate well with values obtained at the
arm, although measurements with thigh cuff placement take the longest to complete. NIBP has been reported as an effective means of tracking blood pressure changes and hypotension in unstable patients. However, it is usually desirable to establish an arterial line as soon as other procedures have been performed, such as securing the airway and obtaining large-bore intravenous access. Limitations to the use of NIBP include placing an inappropriately sized cuff, presence of dysrhythmias, and patient movement or movement associated with the surgical procedure (including surgeons). However, more relevant for the patient with trauma is the limitation of obtaining accurate blood pressure values during hypotension.

Most intraoperative NIBP measurements are performed with automated devices, which use the principle of oscillometry. In this technique, the amplitude of oscillations in an air-filled occlusive cuff is measured at cuff pressures that start at more than SBP and extend to less than DBP. A plot of oscillation amplitude versus cuff pressure generates the oscillation amplitude envelope (Fig. 7). The maximum amplitude algorithm (MAA) is used by most devices to estimate MAP, which is considered to be the pressure in the cuff at maximum oscillation amplitude. The SBP and DBP are calculated based on the shape of the amplitude envelope. However, the algorithm used is proprietary, not standardized, and varies with manufacturer. Clinical studies have shown that NIBP correlates well with IABP for pressure values in the normal range, whereas it has been shown to be less accurate at extremes of blood pressure. When compared with sphygmomanometry, oscillometric SBP values between 90 and 110 mm Hg...
overestimate SBP (119 ± 6 mm Hg vs 103 ± 4 mm Hg). The overestimation is more significant for SBP values less than 90 mm Hg (106 ± 6 mm Hg vs 80 ± 2 mm Hg).\(^\text{21}\) In contrast, oscillometric MAP has been shown to be consistent with IABP MAP for values less than 65 mm Hg,\(^\text{16}\) which may seem appropriate because MAP is considered to be a directly measured parameter in oscillometry.

Although it is commonly assumed that the MAP as measured by the MAA algorithm is more accurate than SBP and DBP, all 3 parameters have been shown in simulation studies to be influenced by other factors independent of MAA, such as pulse pressure, arterial pressure/volume relationship, and pulse wave morphology and heart rate.\(^\text{22–25}\) In addition, errors in estimates of SBP, DBP, and MAP have been shown in simulation studies to depend on interactions between the arterial waveform amplitude, waveform morphology, and arterial wall mechanical characteristics.\(^\text{23}\) Furthermore, there may be twice as much error associated with MAP than with SBP and DBP measurements.\(^\text{26}\) Another simulation study suggested that there is no direct relationship between the MAP estimate obtained with MAA and the actual MAP.\(^\text{22}\)

There is no standardization of the oscillometric algorithm used by manufacturers, and most validation studies have been conducted in healthy patients with hypertensive and hypotensive conditions being absent or underrepresented. These facts may add an additional layer of error to oscillometric measurements in certain patient populations. Group-specific differences between oscillometry and the auscultatory method have been reported in pregnant women,\(^\text{27,28}\) diabetics,\(^\text{29}\) and patients with decreased arterial elasticity.\(^\text{30}\) For example, hypertensive patients have been observed to display an oscillation amplitude envelope with an ambiguous maximum amplitude\(^\text{31}\) (see Fig. 7) leading to significant errors in estimated MAP. Although the effect of hypotension on the oscillation amplitude envelope has not been investigated in the setting of hemorrhagic shock, patient physiology and device factors may explain the unreliability of oscillometric measurements in unstable patients with trauma.

Given the many factors (see Table 1) that contribute to the unreliability of oscillometric monitoring, it is sobering to acknowledge that patients being managed with a hypotensive resuscitation strategy are sometimes monitored with these devices. Future
investigations on hypotensive resuscitation should indicate the methods used for blood pressure measurement to allow clinical interpretation of study results.

**PETCO₂ as a Measure of Cardiac Output**

Initial hemodynamic assessment and monitoring of patients with acute trauma can be difficult because of the frenetic and at times chaotic operating room environment in these cases. The act of placing monitors on the patient can be challenging, especially because many of the standard monitors are either inaccurate or do not function during low-flow circulatory and hypotensive states. Initial intraoperative hemodynamic assessment of the patient needs to be attained in a timely and efficient manner. A simple way to assess whether a patient has cardiac output on arrival to the operating room is to ventilate the patient and observe the end-tidal CO₂ level (PETCO₂).⁴ Although this can be accomplished with a colorimetric CO₂ detector (eg, in the emergency room or bay), capnography provides numerical values for PETCO₂ and is easily established on attaching the patient to the ventilator. However, special considerations must be taken into account when using this approach in the patient with trauma to avoid misdiagnosis or missed detection of severe hemodynamic instability.

The relationship between PETCO₂ and cardiac output has been investigated in animals³²–³⁶ and humans.³⁷–³⁹ Cardiac output and PETCO₂ have been shown to correlate during low-flow circulatory states including cardiogenic, septic, and hemorrhagic shock.³² The relationship between PETCO₂ and cardiac output has been shown to be a logarithmic curve.³⁴ A study in patients being weaned from cardiopulmonary bypass reported that when PETCO₂ was more than 30 mm Hg, the cardiac output was greater than 4 L/min. However, at PETCO₂ levels of more than 34 mm Hg, further increases in cardiac output did not correlate with comparable changes in PETCO₂.³⁷

The correlation between PETCO₂ and cardiac output in low-flow states can be explained based on the pathophysiology of shock. As cardiac output to the lungs decreases, less CO₂ is available for transfer into alveoli, there is an increase in the West zone 1, which contributes to dead space–like ventilation (ie, VQ mismatch), and absolute dead space secondary to pulmonary vessel collapse can occur.

An additional explanation can be obtained by considering 2 fundamental expressions from respiratory physiology that describe the relationship between alveolar ventilation (VA), CO₂ elimination (VCO₂), and partial pressure of alveolar CO₂ (PACO₂), (VA = VCO₂/PACO₂ × K), and the relationship between CO₂ elimination, cardiac output, and arterial and venous CO₂ content (CvCO₂) or Fick principle (VCO₂ = Q × (Caco₂ – CvCO₂)).⁴⁰ After assuming normal lung function and that PETCO₂, PA CO₂, and partial pressure of arterial CO₂ (Paco₂) are equal, Maslow and colleagues (2001) rearranged these expressions to obtain the following:

\[
\text{PETCO}_2 = \frac{AQ_p P\bar{V}co_2}{V_A + AQ_p}
\]

where Qₚ is pulmonary blood flow (ie, cardiac output), P\text{\textbar}co₂ is partial pressure of mixed venous CO₂, and A is CO₂ solubility in blood. A limitation of this equation is the assumption that lungs are homogeneous; however, in the setting of shock, VQ mismatch and absolute dead space are likely to be present. Nonetheless, the equation is useful in showing that PETCO₂ is affected by several interdependent factors.

During periods of apnea or hypoventilation, P\text{\textbar}co₂ (as well as Paco₂) increases because of decreased or absent pulmonary elimination of CO₂.⁴⁰ Reestablishment of ventilation in this circumstance reveals a PETCO₂ that is normal or greater than
normal even if cardiac output is unchanged. In contrast, in cardiac arrest or low cardiac output states $P_{\text{v}/\text{co}_2}$ increases because less CO$_2$ is delivered to the lungs. In this situation, $P_{\text{aco}_2}$ does not increase as much as $P_{\text{v}/\text{co}_2}$ and may decrease,$^{12}$ whereas the $P_{\text{v}/\text{co}_2}-P_{\text{aco}_2}$ difference increases.

Restoration of cardiac output after cardiac arrest or low-flow state results in increased PET$_{\text{CO}_2}$ level. However, in this circumstance, increased PET$_{\text{CO}_2}$ does not necessarily reflect adequate cardiac output. Experimental evidence in support of this phenomenon was shown in a study in which hemorrhagic shock was induced in sheep while mechanical ventilation rate was held constant.$^{12}$ Arterial and venous CO$_2$ were measured during baseline, hemorrhagic shock, and postresuscitation periods. During the shock period, cardiac output decreased (from 2.3 to 0.6 L/min), resulting in significant increase in $P_{\text{v}/\text{co}_2}$, modest decrease in $P_{\text{aco}_2}$, and decrease in PET$_{\text{CO}_2}$. The decrease in PET$_{\text{CO}_2}$ was predominantly caused by the decreased cardiac output. After resuscitation and return of normal cardiac output, $P_{\text{aco}_2}$ increased to more than baseline levels and the $P_{\text{v}/\text{co}_2}-P_{\text{aco}_2}$ difference decreased. Although PET$_{\text{CO}_2}$ was not measured in the postresuscitation period, it is possible to infer that it would have been increased to more than the baseline, assuming that the $P_{\text{aco}_2}$-PET$_{\text{CO}_2}$ difference remained the same between baseline and postresuscitation periods.

Applying this analysis to patients with trauma, there is the prospect that PET$_{\text{CO}_2}$ can reach near-normal levels with small increases in cardiac output after a period of shock or cardiac arrest. These relationships have not yet been quantified experimentally in patients with trauma. Nonetheless, it is prudent to consider prehospital events (ie, cardiac or respiratory arrest) that increase $P_{\text{v}/\text{co}_2}$ when using PET$_{\text{CO}_2}$ to assess hemodynamic status.

**SUMMARY**

The ultimate goal of resuscitation in the setting of uncontrolled hemorrhagic shock is to ensure adequate organ perfusion until surgical hemostasis is obtained. Tissue perfusion in this setting is usually indirectly assessed by monitoring blood pressure with either NIBP or IABP and cardiac output by monitoring PET$_{\text{CO}_2}$ level. A general theme of this article is that factors common in the trauma setting and characteristics of current monitoring devices can often interfere with accurate assessment of hemodynamic status. Overestimation of SBP as can occur with oscillometry and IABP is arguably one of the most important monitoring pitfalls to be avoided. More accurate assessment can be accomplished by considering SBP in conjunction with MAP measurements. However, oscillometric MAP has potential limitations in trauma, especially considering that the amplitude envelope is affected by factors such as arterial waveform shape and heart rate. Research and development need to be applied toward improving algorithms such that oscillometry is more reliable in patient subpopulations including trauma. However, the current lack of standardization is not aligned with this goal. It may be easier to improve the reliability of IABP monitoring, because improving the dynamic response of the CTT system can ensure that the arterial waveform is properly processed and not distorted. However, the extent to which the natural frequency should be increased to provide adequate dynamic response in the setting of trauma is unknown. We are currently conducting studies to address this question.

The unreliability of blood pressure monitoring is likely to be a significant confounding variable in studies investigating the role of hypotensive resuscitation. The most reliably measured blood pressure parameter is currently MAP, and the most reliable method
for MAP measurement is invasively with a CTT system independent of dynamic response (see Table 1). The reliability of oscillometric MAP measurements is unclear, because most evidence suggesting its unreliability comes from theoretic and simulation studies. Nonetheless, it is prudent to control for the methods used to monitor MAP in future studies.

REFERENCES


