Critical Care Monitoring In The Emergency Department

Another four patients have been triaged and placed on the tracking board as the emergency department census continues to climb well beyond capacity. You’ve already notified the administrator on duty that patient transport to the floor will need to be expedited, but unfortunately the hospital has no available beds and you’ve been told that significant delays should be expected. Adding to an already difficult situation, you will be short staffed for another 2-3 hours. Unfortunately, this isn’t a new situation; it’s a typical Monday night. You take a deep breath, another drink of coffee, and get back to work.

Before you can see anyone new, you are called to the nursing triage desk to evaluate a patient. She is an elderly woman brought from home with increasing confusion, fever, and cough over the past several days. Lying on the EMS gurney, she is breathing quickly and is only barely responsive to your questions. Suspecting pneumonia, you place a peripheral IV and obtain basic labs and cultures. Her chest X-ray confirms your suspicion of pneumonia and she perks up with a little IV fluid and antibiotics but remains hypotensive at 90/50 mmHg. You administer an additional 500 cc of normal saline and place her on a dopamine drip through a peripheral IV.

Her blood pressure now looks great, so you admit her to the floor, pat yourself on the back for a job well done, and continue to see patients. The next morning you get an email from your medical director asking, “Do you remember the elderly patient with pneumonia you admitted yesterday morning . . . “

Providing optimal care of the critically ill patient can present a dilemma for the emergency physician. While the importance of the emergency department’s (ED) role in the early care of critical patients continues to be reinforced and redefined, an estimated 15% of a patient’s total hospital critical care is provided in the ED. The additional responsibilities of caring for complex patients and prolonged “boarding” of admitted patients place an increasing strain on already overcrowded facilities and often overextended emer-

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gancy staff. Therefore, an understanding of the utility of advanced monitoring devices is of absolute importance, not only to provide emergency physicians with the confidence that the best care possible is being supplied to their patients but also to optimize the efficiency of such care. While the addition of advanced monitoring technologies may seem to add more complexity and work, the sine qua non of good monitoring is the diagnostic simplification; good monitoring grants control of chaotic situations. This issue of Emergency Medicine Practice provides an overview of the current evidence regarding the benefit of respiratory, hemodynamic, and neurologic monitoring of the critically ill patient.

Critical Appraisal Of The Literature

Evidence regarding the clinical application of monitoring devices was initiated with a PubMed search of literature published between 1950 and 2007 using the keywords: capnometry, capnography, end-tidal CO₂, pulse oximetry, reflectance oximetry, arterial pressure monitoring, cardiac output monitoring, pulmonary artery catheter, partial carbon dioxide rebreathing technique, venous arterial CO₂ gradient, pulse contour analysis, PiCCO, LiDCO, pulseco, FlowTrac, transthoracic electrical bioimpedance, esophageal Doppler, central venous pressure, hemodynamic optimization, fluid responsiveness, cardiac filling pressure, systolic pressure variation, delta pulse pressure, stroke volume variation, pre-ejection period variation, central venous oxygenation, early goal directed therapy, surviving sepsis campaign, microcirculatory abnormalities in sepsis, sublingual capnometry, percutaneous carbon dioxide, and bispectral index. The results were reviewed and relevant citations from each study were searched manually. The results of the fluid and catheter treatment trial (FACTT) and the PAC-Man trial regarding the utility of pulmonary catheterization were also reviewed.


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Prehospital Care

Advanced monitoring of the critically ill in the prehospital setting is currently limited by logistical difficulties, cost, and training. Though few well designed studies have explored the benefit of monitoring during Emergency Medical Services transport, a slowly growing body of evidence suggests significant outcome benefit when monitoring devices are used. While using pulse oximetry is considered standard of care by EMS in many locales, the importance of comprehensive prehospital management in maximizing good outcomes is becoming increasingly recognized, and there is a growing need to equip advanced life support units with advanced monitoring technologies. In the following section, evidence regarding the utility of continuous assessment of end-tidal carbon dioxide (ETCO₂) in the prehospital environment to ensure proper endotracheal tube placement as well as the prevention of inadvertent hyperventilation in traumatic brain injury will be discussed.

Respiratory Monitoring

An EMS notification of prehospital cardiac arrest is received by your community emergency department. Per the paramedic crew, the patient is a 62-year-old man initially complaining of chest tightness who became unresponsive and apneic during transport approximately 3 minutes prior to arrival. The rhythm was noted initially to be ventricular fibrillation; after a single biphasic countershock his rhythm became organized but he remained pulseless. He was intubated en route and is now being wheeled into your critical care area receiving active chest compressions.

You glance at the EMS monitor and note a slow narrow complex rhythm; chest compressions are continued and his prehospital capnography tubing is connected to the ED monitor. The characteristic waveform reassures you of proper ET tube placement, and the ETCO₂ is 14 mmHg. You obtain central access and a round of ACLS drugs is administered. At the following rhythm check you note ventricular fibrillation; he receives another shock and chest compressions continue. You now note an ETCO₂ of 36 mmHg, a central pulse is appreciated, and his plethysmographic waveform becomes clearly defined. Hypothermia is induced and the patient is admitted to the
cardiac critical care unit.

Monitoring a patient’s respiratory status is crucial in all but the most benign of emergency department presentations. If the patient is critically ill, real-time monitoring is obligatory. Respiratory status can be divided into monitoring of the airway, ventilation, and oxygenation. While monitoring of the airway is often a clinical evaluation, technology can aid our assessment of the patient’s ventilatory and oxygenation status.

End-Tidal Carbon Dioxide (ETCO2)

Ventilation (the pulmonary exchange of carbon dioxide [CO2] and its subsequent expiration) is typically monitored in the ED by two modalities: colorimetric capnometry or continuous infrared spectroscopy. Colorimetric capnometers display a threshold concentration of carbon dioxide qualitatively or semiquantitatively by color change, providing the clinician with confirmation of endotracheal tube placement, see Table 1 on page 6. Capnometry by infrared absorbance spectroscopy, on the other hand, allows continuous quantitative assessment of carbon dioxide concentrations displayed by numerical value. Many capnometers also graphically depict the CO2 waveform as a function over time; this capability (known as capnography) provides the clinician with additional information regarding the patient’s ventilatory status, see Figure 1. Additionally, volumetric capnography plots expired CO2 concentration along with exhaled volume during the respiratory cycle, providing information regarding alveolar and anatomic dead space. This may allow for additional applications, including the bedside diagnosis or exclusion of pulmonary embolism.3,4

Correlation Of End-Tidal CO2 To Arterial PaCO2

End-tidal CO2 concentration (the concentration of CO2 at the end of exhalation) typically underestimates arterial CO2 concentration (PaCO2) in healthy individuals by 4-5 mmHg. The fact that ETCO2 does not equate to PaCO2 is a critical point; the discrepancy can primarily be explained by three factors:6

- The PaCO2-ETCO2 gradient is determined at the level of the alveoli by both venous admixture/shunt (increased by atelectasis, pulmonary edema, or pneumonia) and the alveolar dead space of the lungs (increased by pulmonary emboli, reduced cardiac output, cardiac arrest, or hypovolemia.)
- The gradient is further widened by the addition of the anatomic (functional) dead space of the conducting airways.
- As tidal volume decreases, the fraction of the lung ventilated but not perfused increases, widening the PaCO2-ETCO2 gradient. Likewise, obstructive pulmonary disease increases the gradient secondary to inadequate ventilation due to delayed emptying of alveoli. This proportion of wasted ventilation is represented by the dead space/tidal volume ratio (VD/VT).7

The determinants of the PaCO2-ETCO2 gradient are multi-factorial and the magnitude of their effect is often unpredictable. Therefore, while the correlation between PaCO2 and ETCO2 may be somewhat reliable in stable unintubated ED patients, it is typically considered unreliable in critically ill patients and may be assumed only with caution.8,9,10,11,12,13

It would be reasonable to hypothesize that ETCO2 levels would be useful if the gradient were calculated by an initial PaCO2 determination by blood draw. However, the stability of the PaCO2-ETCO2 gradient was found to occur in only 60-80% of patients, with changes in the opposite direction occurring unpredictably. These results were supported in a study of multi-trauma patients which found a 27% erroneous prediction of PaCO2 change by ETCO2.14 However, the PaCO2-ETCO2 gradient is almost entirely in the positive direction, and when a negative gradient exists it is typically very small. Therefore, while a low ETCO2 value may provide little information regarding a patient’s ventilatory status, a high ETCO2 value almost always correlates...
with an equal or higher PaCO₂ value. This concept may prove beneficial in the continuous monitoring of ventilatory status in patients in respiratory extremis secondary to status asthmaticus or congestive heart failure decompensation. Likewise, in situations in which targeted PaCO₂ values may be of value, such as in patients with evidence of increased intracranial pressure and acute brain herniation, a high ETCO₂ value may signal the necessity for adjustments in the patient’s mechanical ventilatory parameters or the need for arterial blood gas sampling.

ETCO₂ Monitoring Applications

**Verification Of Endotracheal Tube Placement:**

Arguably, the most useful application of continuous ETCO₂ monitoring is to allow real-time confirmation of adequate ventilation through capnographic waveform analysis. Continuous ETCO₂ monitoring is a valuable tool for preventing misplacement of the endotracheal tube (ETT), either through continuous verification of placement following intubation or for airway management during CPR or transport.

Stewart et al reported a 90% intubation success rate in 779 adult patients who were either in a coma or in cardiopulmonary arrest. They reported a 2% missed tracheal intubation rate with 14 esophageal intubations. In another study of adults, Pelucio et al reported a 6% esophageal intubation rate before their system implemented a protocol for detecting esophageal intubations. In a series of studies from San Diego, paramedics intubated successfully 84% of the time; 16% of these patients required a rescue device to secure the airway. These representative studies stress both the critical importance of CO₂ detection protocols in any pre-hospital system using endotracheal intubation and the imperative for these systems to have airway rescue devices available in case of a failed intubation. Auscultation over the chest does not detect up to 15% of esophageal intubations, while fogging in the ETT is reported in up to 85% of esophageal intubations. In a study from the Orlando, Florida EMS system, Katz and Falk reported that 28/107 (25%) patients who had a prehospital intubation arrived in the ED with an unrecognized, misplaced endotracheal tube, 18 in the esophagus and 9 above the vocal cords. However, when ambulances and aeromedical units were equipped with continuous ETCO₂ monitors in a follow up study, Silvestri et al reported that the incidence of unrecognized misplaced intubations was 0%, compared to the 23% incidence of misplaced ETT placement in those units where continuous ETCO₂ monitoring was not available. Likewise, Grmec et al studied 81 patients (58 with severe traumatic brain injury) who underwent prehospital intubation (by emergency physicians in Slovenia) and compared auscultation to capnometry with capnography for confirmation of proper endotracheal tube placement. Successful intubation was observed in 73 patients; however, 8 patients were intubated into the esophagus as shown by capnometry. Of those, 4 were incorrectly thought to be in the trachea based upon auscultation.

Confirmation of correct ETT placement is critical. Silvestri et al reported a 69% mortality associated with unrecognized misplaced endotracheal intubation and 100% mortality if the patient was apneic upon arrival to the emergency department.

Additionally, the use of continuous ETCO₂ in confirming prehospital intubation during cardiac arrest has also been shown to be more effective than colorimetric capnometry and auscultation. The presence of ETCO₂ greater than 5 mmHg at breath seven was found to be 100% sensitive and 100% specific for correct placement of the ETT, while qualitative capnometry was 100% specific but only 80% sensitive in cardiac arrest. Both methods were 100% sensitive and 100% specific in non-arrest intubations.

Continuous ETCO₂ monitoring beginning with laryngoscopy/intubation and continuing throughout surgery is considered standard of care by the American Society of Anesthesiologists as a protective measure to avoid unrecognized misplaced intubation. However, the American College of Emergency Physicians currently does not differentiate between the utility of qualitative, quantitative, or continuous ETCO₂ detectors for the verification or reconfirmation of endotracheal tube placement.

**Monitoring During Procedural Sedation:**

When performing procedural sedation, reliable monitoring of the patient’s ventilatory status is crucial. While clinical indicators like chest rise or the plethysmography-derived respiratory rate provided by ECG lead placement can be used, monitoring the capnographic waveform for hypopneic and bradypneic hypoventilatory patterns provides the clinician with a quick and more accurate indication of acute respiratory events. Burton et al reported that capnographic changes (defined as a change in ETCO₂ level greater than 10 mmHg or intrasedation ETCO₂ less than 30...
mmHg or greater than 50 mmHg) predicted respiratory events by up to 271 seconds. Often, nasal prongs with an additional port are used to provide continuous CO₂ sampling. If commercial devices are not available, a regular nasal cannula can be modified, see Figure 2. It is important to note that the CO₂ waveform will show ventilatory rate and duration, but that the vertical axis does not represent ventilatory depth (of course, it represents the amount of CO₂ in that ventilation).

**Traumatic Brain Injury (TBI):** Hyperventilation with hypocapnea may worsen outcome in brain injured patients. Therefore, monitoring of ETCO₂ is emerging as a fundamental component of traumatic brain injury management not only in the hospital but also in the prehospital arena. After TBI, there may be a period of prolonged hypoperfusion with cerebral blood flow (CBF) reduced by as much as two-thirds of normal. Hyperventilation can further decrease the CBF, potentially to the point of cerebral ischemia or by converting ischemic areas into infarction. Evidence from in-hospital studies indicates that prophylactic early hyperventilation can seriously compromise cerebral perfusion and worsen patient outcome. Inadvertent hyperventilation during prehospital transport is associated with increased mortality.

Several studies have demonstrated the incidence of induced hypocapnia during the field management of TBI patients. In a retrospective study from San Diego, 59 adult severe TBI patients who were unable to be intubated without rapid sequence intubation (RSI) were matched to 177 historical non-intubated controls. The study utilized ETCO₂ monitoring and found an association between hypocapnia and mortality and a statistically significant association between ventilatory rate and ETCO₂. Both the lowest and final ETCO₂ readings were associated with increased mortality versus matched controls. ETCO₂ monitoring was used in 144 patients to assess whether closer monitoring would result in a lower rate of inadvertent severe hyperventilation (defined as ETCO₂ less than 25) after RSI. Patients with ETCO₂ monitoring had a lower incidence of severe hyperventilation (5.6% vs. 13.4%; p = 0.035). Patients who were severely hyperventilated had a higher mortality rate (56% vs. 30%; p = 0.016). However, as previously discussed, the PaCO₂-ETCO₂ gradient in traumatically injured patients is unreliable, specifically in regards to low ETCO₂ concentrations. Therefore, the reliance on ETCO₂ as an indicator of hypocapnia must be further evaluated in the emergency setting before it is used to guide ventilatory management of traumatically brain injured patients in the ED.

**Prognosis Of Continued Cardiopulmonary Resuscitation:** As discussed previously, pulmonary CO₂ exchange is affected by multiple factors at the level of the alveoli. In contrast to this, at extremely low flow states ETCO₂ is determined almost entirely by pulmonary flow secondary to a logarithmic relationship between cardiac output and ETCO₂ which exists during cardiac arrest. This relationship between pulmonary flow and ETCO₂ during arrest makes capnometry an important prognostic marker during cardiopulmonary resuscitation (CPR) in
the ED. In multiple studies, an ETCO₂ level of 10 mmHg or less 20 minutes after the initiation of resuscitation in patients with pulseless electrical activity accurately predicted death in patients suffering prehospital arrest.²²,³³ These findings were also supported by a small prospective trial of both in- and out-of-hospital arrest³⁴ and were only minimally affected by variations of ventilatory rate.³⁵ However, the initial ETCO₂ had no correlation with outcome or survival,²² and very low initial ETCO₂ (less than 6 torr) has been associated with survival.³⁵ Accordingly, the 2005 ACLS guidelines state, “Intubated adults receiving CPR with ETCO₂ less than 10 have poor prognosis” and they consider ETCO₂ “unreliable immediately after starting CPR.”³⁶

ETCO₂ Key Points
- Capnography allows for the continuous verification of endotracheal tube placement which is essential in the unstable prehospital and ED environment in which patients are frequently moved; adequate sedation can occasionally be difficult and accidental extubation is an ongoing risk.
- Continuous capnography may be superior to qualitative CO₂ detectors in detecting correct ETT placement during cardiac arrest.
- An ETCO₂ less than 10 mmHg following 20 minutes of CPR is predictive of death and indicates that continued attempts at resuscitation are likely futile.
- A rapid increase in ETCO₂ concentration during CPR often represents the return of spontaneous circulation and can be a useful guide in determining timing of pulse checks.
- Continuous non-invasive ETCO₂ monitoring can be useful in the monitoring of patients with tenuous respiratory status, such as those with severe reactive airway disease or congestive heart failure.
- While a low ETCO₂ concentration is difficult to interpret, an ETCO₂ concentration greater than 40 will almost always indicate hypercapnia. The degree of this hypercapnia may be underrepresented by the ETCO₂.

Pulse Oximetry
Your asthmatic patient has become so exhausted that she is barely moving any air. She will not tolerate non-invasive ventilation and it’s obvious that she needs to be intubated. You hand the laryngoscope to a trusted fourth year resident. He proceeds to place the tube and states that he saw it pass through the cords. A few seconds later, the pulse oximeter reading drops to 40%. You shake your head and grab the laryngoscope. Your resident points out the yellow color change of the end-tidal CO₂ detector, but you now notice the pulse oximeter is reading 36%. You place the blade and are surprised to note that the tube is clearly between the cords. After another minute, the pulse oximeter is reading 100%.

Pulse oximeters are almost uniformly present in US emergency departments. Noninvasive pulse oximeters differentiate oxyhemoglobin from reduced hemoglobin utilizing differing absorption components at the two wavelengths 660 nm and 940 nm. The pulsatile AC component is then divided by the non-pulsatile DC component to determine the arterial pulse-added absorbance. This value corresponds to the pulse oximeter saturation estimate (SpO₂).³⁹

While typically considered accurate when compared to arterial concentrations of oxygen determined by arterial blood gas co-oximetry, there are several limitations and misconceptions regarding the accuracy of pulse oximetry which should be addressed when considering its reliability in the monitoring of the critically ill ED patient.

ETCO₂ Key Points

<table>
<thead>
<tr>
<th>Color of Capnometry</th>
<th>CO₂ Concentration (%)</th>
<th>Pulse Oximeter Saturation Estimate (SpO₂)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue</td>
<td>0–1</td>
<td>95–100%</td>
</tr>
<tr>
<td>Green</td>
<td>1–2</td>
<td>90–95%</td>
</tr>
<tr>
<td>Yellow</td>
<td>Greater than 3%</td>
<td>80–90%</td>
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</tbody>
</table>

Table 1. Semi-Quantitative Colorimetric Capnometry

The reliability of pulse oximetry is reduced in states of severe hypoxia. While pulse oximeters possess an accuracy of ± 2% to one standard deviation when in the range of 70-100% SaO₂ (oxygen saturation), their accuracy falls to ± 3% (SD) between 50-70% SaO₂ and are generally not considered accurate when determining O₂ saturations below 50%.³⁰

The accuracy of pulse oximetry has also been questioned in the heterogeneous population of the critically ill as a whole. The incidence of intraoperative pulse oximetry data failure (independently predicted by hypotension and hypothermia) was found to be 9% in a review of 9203 anesthesia records.⁴⁰
However, if an acceptable plethysmographic waveform can be detected, the reliability of pulse oximetry has been demonstrated over a wide range of clinical scenarios. When compared with invasive arterial measurements, the accuracy of pulse oximetry measured by finger probe was not reduced in states of poor peripheral perfusion (cardiac index less than 2.2) or low peripheral temperature (less than 28°C). Additionally, although pulse oximetry was previously considered inaccurate in the presence of hemoglobin levels less than 5 g/dL, a small study of patients with hematocrit of less than 20% provided evidence that SpO₂ was accurate at hemoglobin concentrations as low as 2.3 g/dL. These results were supported by a study which showed no significant effect of moderate anemia or acidemia on the accuracy of SpO₂.

While SpO₂ readings may eventually correlate with ABG determination of blood oxygenation, there still may be significant delay associated with the detection of hypoxemia in hypothermic, vasoconstricted, or low-flow states. In a study of healthy volunteers, a response time of 131 seconds and 215 seconds to hypoxic events was found during induced vasoconstriction or hypothermia, respectively. This delay was significantly reduced (22 seconds and 40 seconds) by the use of forehead reflectance oximetry probes. (However, forehead probes are associated with their own limitations, including distortion of findings by venous pulsation and the incidence of skin burn.)

Because of overlap in the absorbance of light at 660 nm and 990 nm, dyshemoglobinemias result in a distortion of true oxygen saturation when measured by traditional pulse oximeters. Based on animal studies, the presence of methemoglobinemia causes a significant overestimation of SpO₂ by pulse oximetry. With increasing concentration of methemoglobin, SpO₂ has been found to plateau at approximately 85%. Likewise, a carboxyhemoglobinemia level of 70% results in a SpO₂ of 90% while the actual measured SaO₂ is 30%. In response to this limitation, a new generation of pulse oximeters that can noninvasively differentiate oxyhemoglobin, deoxyhemoglobin, methemoglobin, and carboxyhemoglobin saturation have become available.

Ambient light has been shown to have no effect on the accuracy of oximetry readings in hemodynamically stable patients. Additionally, when studied in healthy volunteers the accuracy of pulse oximetry was decreased in the presence of dark fingernail polish (black, blue, or green) but was not felt to be clinically significant in mechanically ventilated patients. Dark skin pigmentation has also been suggested as a source of interference in the accuracy of pulse oximetry; however, this was not supported by an investigation of emergency department patients.

While motion artifact has presented a significant problem to oximeter design, recent advances in motion-resistant and read-thru-motion using different algorithms have improved accuracy and rely less on lengthening of signal averaging times which may lead to missing significant short-lived hypoxemic events.

**Pulse Oximetry Applications**

The presence of continuous pulse oximeters is considered necessary during all phases of definitive airway management. In a study of ED patients, those monitored by pulse oximetry were found to have significantly fewer episodes of hypoxemia during emergency intubation (27% vs. 15%), and the episodes were of shorter duration than non-monitored patients (1.4 minutes vs. 0.7 minutes). Additionally, a systematic review of perioperative pulse oximetry in more than 20,000 patients showed a reduced incidence of hypoxemia and increased incidence of both naloxone and oxygen administration in those patients monitored with oximetry. However, the usefulness of pulse oximetry in avoiding hypoxemia during airway management should not be misconstrued to suggest utility in verifying correct ET tube placement. In a series of pre-oxygenated patients, SpO₂ was unable to detect esophageal intubation at 30 seconds.

The availability of pulse oximetry has been shown to substantially reduce arterial blood gas sampling in the ED without an increase in adverse outcomes. Pulse oximetry can also be used to safely and quickly titrate the FiO₂ in mechanically ventilated patients from potentially toxic oxygen concentrations.

**Pulse Oximetry Key Points**

- Pulse oximetry remains accurate over a wide range of critical illness, including shock, hypothermia, and significant anemia.
- Although pulse oximetry is necessary during all phases of definitive airway management in order to reduce the length and frequency of hypoxic events, it lacks sensitivity in verifying
correct ETT placement.

- Pulse oximetry readings may actually be showing desaturations which occurred up to 1 minute in the past secondary to redistribution of oxygenated blood from central to peripheral circulation and prolonged signal averaging times. This leads to the common clinical scenario of a large drop in the pulse ox reading immediately following successful placement of the endotracheal tube. While sometimes terrifying, the drop often reflects the desaturation during the apneic period prior to the tube placement.

**Hemodynamic Monitoring**

A 65-year-old man, new to your emergency department, is brought in by EMS with a history of abdominal pain and vomiting for 2 days. He is obtunded, tachypneic, and hypotensive with a MAP of 55. His SpO₂ is 90% on 100% NRB and the decision is made to intubate. Central access is obtained, crystalloid is administered, and a point of care panel reveals a normal hematocrit but extremely low ionized calcium. His BP rebounds with an ampule of calcium chloride, and IV fluid is continued.

The patient’s family soon arrives and explains that he was well until yesterday when he began complaining of abdominal pain. He is an insulin-dependent diabetic and suffered a myocardial infarction 1 year prior. His ECG, chest XR, and cardiac enzymes are normal; however, his lipase is grossly elevated and his creatinine is 4.3. Severe acute pancreatitis is assumed to be the likely cause of the patient’s illness and the ICU is contacted. Unfortunately, you are told that there are no available beds in either the medical or surgical ICU and there may be a significant delay before a patient can be transferred out.

He quickly becomes hypotensive necessitating a norepinephrine drip and an intra-arterial line. You explain to the patient’s family that he is extremely ill, but you will do everything possible to give him the best chance to survive...

The assessment of hemodynamic status is essential in the care of the critically ill patient in order to ensure adequate organ perfusion and, ultimately, tissue oxygenation. Common monitoring modalities of three interrelated components of hemodynamic adequacy will be reviewed. The first is the basic determination of satisfactory organ perfusion pressure by the monitoring of mean arterial pressure (MAP) utilizing either an intra-arterial pressure transducer or automated blood pressure cuff. The second is the estimation of cardiac function and fluid responsiveness by monitoring cardiac output and filling pressures using a variety of non-invasive and invasive monitoring modalities. The third and final component of hemodynamic monitoring is the determination of overall adequacy of oxygen delivery, oxygen consumption, and tissue perfusion by measuring central venous oxygen saturation and regional tissue carbon dioxide and oxygen concentrations. The benefits, controversy, and application of various hemodynamic monitoring modalities will be discussed in this section.

**Arterial Pressure Monitoring**

Mean arterial pressure, determined by the product of total peripheral resistance and cardiac output, is of critical importance in the maintenance of organ perfusion. While local autoregulatory functions provide constant blood flow over a wide range of pressures, a MAP of 60 mmHg is considered the autoregulatory threshold below which blood flow becomes pressure dependent. Conversely, no increased blood flow occurs with mean arterial pressures greater than 65 mmHg. The principle of autoregulation is important because while a blood pressure greater than 65 mmHg does not ensure acceptable organ perfusion, a mean arterial pressure less than 65 mmHg almost always represents hemodynamic inadequacy.

The importance of blood pressure as either a reflection or a major determinant of overall function was recently demonstrated by a study which found that a single occurrence of hypotension (defined as systolic blood pressure less than 100 mmHg) conveyed a three-fold increased risk of in-hospital death in non-traumatic emergency patients. Sustained hypotension of greater than 60 minutes increased that risk an additional three-fold, and more significant hypotension (systolic blood pressure [SBP] less than 80 mmHg) increased the risk six-fold.

Monitoring of blood pressure can be accomplished by several means. It was previously taught that the presence of only a carotid pulse indicates a SBP between 60-70 mmHg, the presence of carotid and femoral pulses indicates a SBP of 70-80 mmHg, and the presence of a radial pulse indicates a minimum SBP of 80 mmHg. However, a study comparing physical findings with intra-arterial BP measurements revealed that these findings consistently overestimated or did not correlate at all with the patient’s actual BP.

In most ED patients, the blood pressure is moni-
tored non-invasively by automatic blood pressure cuffs. Non-invasive pressure measurements are usually provided by oscillometric pressure determination. This method is considered more accurate than auscultatory methods, but it typically overestimates diastolic pressures by 4-10 mmHg, underestimates systolic pressures by 0-16 mmHg, and underestimates MAP by 2.3-12.3 when compared to invasive arterial catheters. Intermittent non-invasive blood pressure monitors also suffer from delay in the monitoring of rapid or transitory changes in blood pressure which could have important implications for the critically ill patient. Invasive arterial pressure monitoring can be accomplished by arterial catheterization of the radial, brachial, axillary, dorsalis pedis, or femoral artery. The two most common sites of catheterization (the radial and femoral artery) have been found to be clinically interchangeable in the monitoring of the critically ill in all but the most vasoconstricted states. Many intensivists shy away from the use of the brachial artery site as thrombosis leaves the distal upper extremity with no blood flow.

In terms of complications, 19,617 radial artery cannulations were reviewed; temporary occlusion of the radial artery was found to be the most common complication, occurring in 19.7% of cannulation attempts. Temporary occlusion of the radial artery was defined in this review as any non-permanent occlusion of the artery which did not result in ischemic damage, limb loss, necrosis, or other serious long lasting effects. Occlusion was diagnosed by Doppler, palpation of pulses, or inaccurate transducer readings and lasted as long as 75 days. However, serious ischemic damage occurred in only 0.09%. Hematoma formation was the second most common complication, occurring in 14.40%. Other complications, such as pseudoaneurysm and sepsis, were rare. Likewise, 3899 femoral artery cannulations were reviewed and were found to be associated with a much lower incidence of temporary occlusion (1.18%) and hematoma formation (6.1%); however, permanent ischemic damage was more frequent than in radial artery procedures (0.18%). Overall, serious complications of radial, femoral, and axillary cannulation are considered rare. However, when possible, peripheral cannulation should preferably be performed on the non-dominant limb in the unlikely event of serious complication.

The utility of the Allen test in the prediction of hand ischemia following radial artery cannulation deserves mention. While the teaching of the Allen test continues to plague medical students and residents around the world, its usefulness has been seriously challenged. The first difficulty in the utility of the Allen test is the inability of clinicians to agree on what constitutes an abnormal test. In a review of 15 studies, abnormal criteria for return of normal hand perfusion after release of simultaneous occlusion of radial and ulnar arteries ranged from greater than 5 seconds to greater than 15 seconds. Additionally, inter-observer agreement of exam interpretation is also completely unreliable. Finally, positive results do not predict the occurrence of post cannulation hand ischemia. Therefore, the routine use of the Allen test should be considered misleading at best and will hopefully be de-emphasized in the future.

Arterial Pressure Monitoring Applications
Although not an absolute indication, invasive arterial monitoring is generally recommended when continuous infusions of vasopressors or vasodilators are being utilized to target a specific blood pressure. Any patient who requires frequent blood gas draws will appreciate the one stick of an arterial line rather than multiple sticks from intermittent draws. Other applications, such as cardiac output determination derived from pulse contour analysis and prediction of fluid responsiveness, will be reviewed later in this article.

Arterial Pressure Monitoring Key Points
- Non-invasive blood pressure determinations are accurate in stable patients, so long as a reasonable interval of sampling is chosen.
- Intra-arterial monitoring should be considered in patients in shock or on a continuous infusion of vasopressors.

Cardiac Output Monitoring
The monitoring and manipulation of cardiac output, either directly or indirectly, is of extreme importance in the care of the critically ill. Cardiac output is a function of multiple variables, each of which plays an essential role in the proper functioning of the organism as a whole. Therefore, knowledge of cardiac output provides a sense of the overall performance of the cardiovascular system. Additionally, measurements of cardiovascular functionality provide the capability to monitor its normalization or optimization, the utility of which will be discussed in this section.
While an in-depth discussion of cardiovascular physiology is beyond the scope of this text, a brief review is necessary. Cardiac output is the product of stroke volume and heart rate. Stroke volume, in turn, is dependent on several variables. The first of these variables is preload, its value reflected by end-diastolic volume of the ventricles and its effect on cardiac output as described by the Frank-Starling relationship. The second factor is afterload which is determined by systemic vascular resistance. The final factor is the intrinsic contractility of the myocardium. Each of these variables may be estimated, typically by indirect means, and manipulated in order to optimize cardiac function, see Figure 3.

**Figure 3. Hemodynamic Monitoring**

Simultaneous monitoring of the cardiovascular, pulmonary, and hematologic systems by various modalities provides a comprehensive approach to hemodynamic monitoring, allowing for the individual manipulation of each component in order to normalize or optimize hemodynamic function and ultimately maintain satisfactory oxygen delivery.

**Pulmonary Artery Catheter**

While pulmonary artery catheterization is not a typical ED procedure, it would be impossible to discuss the monitoring of cardiac output without an understanding of the pulmonary artery catheter (PAC) and the controversy regarding its use. Additionally, nearly all studies evaluating alternative cardiac output monitors utilize thermodilution measured by the PAC as their criterion standard. However, the imprecision of pulmonary artery thermodilution is well documented.

The pulmonary artery catheter, introduced by Harold Swan in 1970, is typically inserted through either the internal jugular clavian vein and guided by flow direction through the right atrium, then the right ventricle, and ultimately to the pulmonary artery. At this point, the PAC allows estimation of cardiac output by principles of thermodilution (injecting a solution in the right atrium and recording the resultant temperature change by a thermistor at the tip of the PAC resting in the pulmonary artery). Two modes are available: bolus (for intermittent evaluation) or continuous thermodilution (for continuous monitoring of cardiac output). Studies have typically demonstrated better reproducibility of measurements with continuous cardiac output estimation versus bolus methods.

Insertion of pulmonary artery catheters is an invasive procedure and, likewise, carries the risk of serious complications. The PAC-Man trial (a prospective trial investigating the benefit of routine PAC use) found that PAC insertion was associated with a 10% incidence of complications, consisting largely of hematoma, arterial puncture, and dysrhythmias (including cardiac arrest) but also including a small number of pneumothorax, hemothorax, and the necessity of guidewire retrieval. Another study demonstrated that the incidence of complications (including dysrhythmia [one fatal], subclavian vein thrombosis, endocardial damage, and infective endocarditis) were increased with prolonged catheter use. Additionally, the ESCAPE trial (Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheter Effectiveness) demonstrated a 5% complication rate, including infection, pulmonary hemorrhage/infarct, catheter knotting, and ventricular tachycardia. While overall complications of PAC insertion responsible for significant morbidity and mortality are considered rare, recent studies question its utility beyond less invasive methods such as central venous pressure and clinical exam.

**Carbon Dioxide Fick Principle**

The Fick principle, based on the conservation of mass, observes that the amount of gas release (typically $O_2$ or $CO_2$) which occurs in the pulmonary capillary bed is the product of alveolar blood flow and the difference in gas concentration in the arterial and venous circulation. This allows for the calculation of cardiac output (equivalent to alveolar blood flow) by measuring or estimating the rate of carbon dioxide elimination from the lungs ($VCO_2$) over the arteriovenous PaCO$_2$ gradient.
Utilizing a partial carbon dioxide rebreathing technique, the non-invasive cardiac output monitor (NICO) applies the CO2 Fick principle to estimate cardiac output in the intubated patient by indirectly estimating CO2 elimination, venous CO2, and arterial CO2 by changes in measured ETCO2 during the normal respiratory cycle and rebreathing. However, to accurately assess cardiac output by expired CO2, pulmonary capillary blood flow must also be measured. The amount of pulmonary shunting is estimated by SpO2 and FiO2, thus NICO provides a non-invasive determination of cardiac output by utilizing mainstream capnometry, a differential pressure pneumotachometer, and pulse oximetry.81

Unfortunately, as described previously, ETCO2 is an unreliable estimate of PaCO2 in unstable patients because of the variability of V/Q mismatch in the critically ill. This can be compensated for by estimating pulmonary capillary blood flow. However, several studies demonstrate that the calculated pulmonary shunt fraction by NICO differs considerably from traditionally calculated shunt fraction by blood gas analysis.82,83 In light of this, cardiac output (CO) calculated by NICO was typically considered accurate when limited to stable patients with normal pulmonary function or when monitoring trends.84 All prior studies problematically compared NICO to PAC thermodilution which is not universally considered the ‘gold standard’ of cardiac output determination.85 A recent study comparing NICO with continuous and bolus thermodilution as well as with the true ‘gold standard’ for intraoperative cardiac output determination (transit time flowmetry of the ascending aorta) revealed comparable estimation of CO, with a diversion in the immediate post cardiopulmonary bypass period, with thermodilution overestimating CO and NICO underestimating CO. This offers a reasonable explanation of the lack of agreement demonstrated in previous studies,86 and provides a potentially quick and simple method of determining CO in the ED.

In a similar application of the CO2 Fick principle, the arteriovenous carbon dioxide gradient can be used to estimate cardiac index by directly measuring the mixed venous CO2 and arterial PaCO2 gradient.87 However, the calculation of this gradient requires mixed venous CO2 samples from the pulmonary artery and, therefore, a PAC. Recently, a study demonstrated that the central venous CO2-arterial CO2 gradient also correlates well with cardiac index (CI = CO/body surface area) determined by both mixed CO2-arterial CO2 gradient and thermodilution, allowing for a potential minimally invasive method to determine CI using only a central venous catheter (CVC) in the superior vena cava.88

Pulse Contour Analysis
The contour of the arterial waveform represents the change in intravascular pressure over the cardiac cycle and reflects the relationship between vascular compliance and cardiac output. By utilizing information obtained by intra-arterial pressure transducers, several novel devices allow the continuous assessment of cardiac output by less invasive means in patients in sinus rhythm.

PiCCO™ calculates cardiac output from the change in diastolic/systolic pressure over time and an estimate of aortic impedance. However, a direct determination of aortic impedance is difficult if not impossible at the bedside, so the device must first be calibrated by calculating cardiac output using transpulmonary thermodilution to provide an accurate estimate of impedance. This requires a central venous line and a manufacturer supplied proximal arterial (femoral or axillary) catheter.84 An ice water bolus is injected through the central line and the resultant temperature change is recorded by the arterial catheter. This method has been found to correlate well with pulmonary artery thermodilution.89

After this calibration, continuous measures of cardiac output are determined by pulse contour analysis and have been found to correlate acceptably with thermodilution.90-92 Intrathoracic blood volume (ITBV) can also be calculated by transpulmonary thermodilution as an estimate of cardiac preload, the accuracy of which was recently validated.93 Recalibration is typically recommended every 8 hours or whenever a change in patient status occurs.90

LiDCO™ uses a slightly different algorithm than PiCCO™ and it calibrates using lithium bolus dilution in place of transpulmonary thermodilution. Unlike transpulmonary thermodilution, lithium dilution does not require central access; only peripheral intravenous and peripheral arterial catheterization are required so it is even less invasive. Cardiac output determined by lithium dilution correlates well with pulmonary artery thermodilution,94 as does the pulse contour algorithm used by the continuous monitoring device.95 Like PiCCO™, LiDCO™ requires recalibration every 8 hours to remain accurate.96

FloTrac™, a newly introduced cardiac output monitor, requires only a peripheral arterial catheter

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and does not need calibration. Cardiac output is calculated by the FloTrac™ device from pulsatility and an estimation of resistance/compliance based on age/gender/weight/height. There are currently limited data reflecting the reliability of FloTrac™, and studies are conflicting. In one study, comparable bias and precision were found when compared to continuous cardiac output thermodilution. However, another investigator concluded that there was unacceptable agreement with thermodilution, although this study was performed using the first generation FloTrac™ and interval improvements of the analytic algorithm have been incorporated since the study was completed.

Transthoracic-Electrical Bioimpedance
Transthoracic-electrical bioimpedance (TEB) monitors operate by measuring resistance in the thorax to high frequency, low magnitude current to estimate changes in total thoracic fluid content over the cardiac cycle which allows for the calculation of cardiac output. This is accomplished quickly and non-invasively with several skin electrodes (similar to those utilized by ECG) applied to the neck and thorax. Unfortunately, TEB is unable to accurately assess cardiac output in the presence of dysrhythmias (such as atrial fibrillation or atrial flutter) and provides no direct preload assessment.

In terms of reliability, a meta-analysis comparing 154 studies revealed good correlation with cardiac output estimated by thermodilution; however, in their analysis, accuracy was diminished in cardiac patients. A study of critically ill emergency department patients echoed the accuracy of TEB overall but again demonstrated somewhat reduced accuracy in patients with extensive pulmonary edema, pleural effusions, chest wall edema, or chest tubes parallel with the aorta. More recently, a study utilizing current generation TEB demonstrated good correlation with thermodilution in both hemodynamically stable and unstable cardiac surgery patients and was found to correlate well with CI measured by PAC in a large study of trauma patients with TEB initiated while in the ED.

Esophageal Doppler
Esophageal Doppler is another minimally invasive monitor of cardiac output which estimates stroke volume by recording the velocity of blood flow in the descending aorta by means of an ultrasound probe, roughly the size of a gastric tube, placed nasally or orally. In order to accurately assess stroke volume from flow, esophageal Doppler measurement makes several assumptions. The first is that there is stable cephalic-caudal blood flow; this flow may be inconsistent in the critically ill. The second assumption is the existence of a constant cross sectional area of the aorta. This aortic measurement is either estimated by calculations based on height/weight/age or directly measured by integrated real-time M-Mode echocardiographic capabilities of some esophageal Doppler models. The utilization of the esophageal Doppler is a learned skill, but a study suggests that no more than 12 placements are needed to become adept. The reliability of esophageal Doppler was discussed in a systematic review of 11 validation papers that revealed low bias but limited agreement in

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absolute CO values when compared to PAC thermodilution but high clinical agreement when following trends. However, the authors concluded that there were an inadequate number of studies reviewing the accuracy of the esophageal Doppler with integrated M-Mode aortic measurement capabilities. A recent study comparing esophageal Doppler with direct echocardiographic aortic measurement to PAC thermodilution and PiCCO™ revealed good agreement between all methods. Additionally, esophageal Doppler allows preload estimation with more accuracy than pulmonary artery perfusion pressure (PAOP) using corrected flow time (FTc) and contractility estimation from peak velocity (PV) of aortic blood flow.

Cardiac Output Monitoring Applications
Currently, all studies which have investigated the benefit of cardiac output monitoring in the critically ill utilize pulmonary artery catheterization in their experimental group. Therefore, it is necessary to briefly outline the controversy which surrounds the use of pulmonary artery catheterization in the routine management of patients in the intensive care unit. Although the validity of the routine utilization of the pulmonary artery catheter in the care of the critically ill has long been questioned, the SUPPORT investigators were the first to provide preliminary data that the use of PACs may in fact be counter productive. In a large retrospective study of 5735 critically ill patients, they found an increased 30 day mortality, increased hospital cost, and increased length of ICU stay associated with the use of PACs. The lack of benefit was later confirmed by several prospective trials, including the PAC-Man trial which demonstrated that the routine placement of PACs had no effect on morbidity or mortality. Additionally, the ESCAPE trial found no difference in mortality or length of hospital stay when PAC parameters were compared with clinical assessment in the management of severe CHF patients. However, none of the trials utilized clearly defined treatment endpoints or therapies based on the measurements obtained from the PAC, instead relying only on the discretion of the individual intensivist in determining their usage and interpretation.

Targeted Resuscitation: While routine use of pulmonary artery catheters is now considered unbene

ing to optimize cardiac output. Because CO monitoring historically required PAC placement in

the ICU, most non post-operative studies only investigated patients optimized late (more than 24 hours) in their hospital course. These studies were disappointing, demonstrating no difference in mortality or even increased mortality for supranormal target values. However, a meta-analysis of early hemodynamic optimization with PACs found supranormal target values (defined as CI greater than 4.5 L/min*m², pulmonary artery occlusion pressure less than 18 mmHg, oxygen delivery (DO₂) greater than 600 mL/min*m² and oxygen consumption (VO₂) greater than 170 mL/min*m²) were associated with decreased mortality in studies with high control group mortality (greater than 20%). In this systematic review, early hemodynamic optimization was defined as therapy occurring within the first 12 hours after surgery, less than 24 hours after trauma, before the onset of organ failure, or less than 4 hours after the diagnosis of sepsis. Additionally, therapy was only considered effective when it resulted in differences in DO₂ between protocol and control groups. Of note, one study initiating early optimization of severe trauma patients in the emergency department by utilizing transthoracic electrical bioimpedance and changing to PAC in the ICU found no difference in mortality. However, there was also no difference in oxygen delivery between control and protocol groups in those who died because optimal values could not be obtained. Ultimately, there was no difference in treatment between study groups; therefore, it is not surprising that there was no difference in outcome. This study has also been criticized for the low mortality rate of the control group (less than 15%).

Recognition Of Hemodynamic Instability: The identification of the hemodynamically unstable patient can be difficult, and reliance solely upon clinical indicators is considered inadequate. Blood pressure (MAP) has been demonstrated to be an unreliable predictor of blood flow and cardiac output, and while shock index (HR/systolic BP greater than 0.9) fares better, it’s not sensitive in identifying critical illness. Furthermore, a large portion of critically ill patients with significant global tissue hypoxia (identified by elevated lactate and decreased central venous oxygenation) will present with normal MAP, HR, and SI and, likewise, demonstrate no change in vital signs despite trends of systemic improvement. While the
usefulness of high serum lactate levels in identifying high-risk patients is well established, the requirement of serial blood draws (and possibly significant laboratory turn around time in the absence of departmental point of care measurement devices) makes for potentially significant delays in the identification of patients with declining hemodynamic status. However, the application of non-invasive or invasive hemodynamic monitors may improve the clinician’s ability in the early identification of the critically ill prior to overt decompensation. A recent study demonstrated that hemodynamic trends in CI, oxygen delivery and consumption, and tissue perfusion obtained by completely non-invasive monitoring modalities (TEB, SpO$_2$, and transcutaneous oxygen and carbon dioxide monitors) predicted non-survival in severe trauma patients. Additionally, when blunt and abdominal trauma patients were evaluated with the addition of a stochastic analysis/mathematical search and display program to calculate survival probabilities utilizing measurements from the same non-invasive modalities, a further increase in the ability to avoid misclassification of non-survivors and survivors was noted.

Cardiac Output Monitoring Key Points
- Minimally invasive cardiac output monitors allow for time efficiency in the ED setting and provide valuable information regarding the overall cardiovascular status of the patient.
- A declining cardiac index in the critically ill patient indicates a necessity for aggressive clinical re-evaluation and intervention. In the traumatically injured patient, for example, sources of potential ongoing blood loss must be investigated.
- Early hemodynamic optimization may be beneficial in the critically ill ED patient. Although further prospective trials need to be performed, maintaining CI greater than 4.5 L/min*m$^2$, oxygen delivery (DO$_2$) greater than 600 mL/min*m$^2$ and oxygen consumption (VO$_2$) greater than 170 mL/min*m$^2$ may provide additional survival benefits in conditions with high patient mortality.

Preload Monitoring

Your patient with severe acute pancreatitis has now received 6 liters of crystalloid but still lacks any output from his Foley catheter. You question how much fluid this patient will require before you begin overloading an already diseased heart and a patient with likely acute tubular necrosis. You connect the patient’s existing arterial line to a pulse contour cardiac output monitor and note that his cardiac index is low and his stroke volume varies considerably with his respiratory cycle indicating inadequate preload. You administer another 2 liters of IVF and observe as his cardiac index begins to rise…

While measures of cardiac output offer a composite of heart rate, preload, inotropy, and afterload, it is often necessary to determine whether the patient needs additional volume. Measures of preload have traditionally (and only somewhat successfully) been used to answer this question.

Pulmonary Artery Perfusion Pressure

By ‘wedging’ the distal balloon of the PAC into smaller branches of the pulmonary artery, the pulmonary artery occlusion pressure (PAOP) can be measured. The PAOP is an estimate of left ventricular end-diastolic pressure (LVEDP) in the absence of disturbances of the pulmonary vasculature, the left atrium, or the mitral valve and an estimate of left ventricular end-diastolic volume (LVEDV) in the absence of abnormal ventricular compliance. If no alterations present, LVEDV can then be used as an estimate of preload. A newer volumetric PAC allows the estimation of right ventricular end-diastolic volume (RVEDV) by thermodilution, which may be superior to PAOP in the assessment of preload. However, PACs are not routinely placed in the ED; given the complications mentioned in the previous section, this is not likely to change in the future.

Central Venous Pressure

Measured in the superior vena cava by the distal port of central venous catheter or the proximal port of a pulmonary artery catheter, central venous pressure (CVP) is a representation of right atrial pressure (RAP). RAP can provide an estimate of venous return and a very rough estimation of preload. The estimation is rough because it is also affected by right ventricular compliance, pericardial disease, valvular disease, and factors that affect intrathoracic pressure (such as positive pressure ventilation, positive end-expiratory pressure, and normal respiratory variation). Thus static CVP measurements rely on a complex interaction of variables and are typically considered poor indicators of preload and volume status, see Figure 4. While the normal CVP range is between 4-6 mmHg, significant hypervolemia or
Clinical Pathway: Sepsis Protocol

Patient identified with severe sepsis or septic shock.

Monitoring: ECG/continuous telemetry, SpO₂, MAP transduced by arterial catheter.
Minimum vascular access: one large bore peripheral IV, and quadruple lumen central venous catheter.
Consider definitive airway management.

FLUID RESPONSE

Administer rapid bolus of 500 cc IVF over 5-10 minutes.

Signs of hemodynamic improvement:
- falling heart rate
- rising MAP
- positive change in plethysmographic waveform
- increase in SpO₂
- increase in peripheral warmth
- increase in urine volume

No signs of hemodynamic improvement or
Adverse effects on pulmonary function:
- rising airway pressure
- fall in SpO₂
- frothing from ETT

INOTROPIC SUPPORT

Target MAP: 90-110
Target HR 90-120 in sinus rhythm
- norepinephrine infusion
- dopamine infusion (caution: AF and SVT are common)
- consider dobutamine
- consider digoxin after potassium repletion in absence of conduction disturbances

Continuous clinical assessment of cardiovascular function.

NOTE: Adapted with permission from an excerpt of dialogue on the Critical Care Medicine Listserv by Dr. Stephen Streat. These are not clinically validated practice guidelines. Instead, they represent an alternative, clinically based algorithm to supplement care of the critically ill septic patient.
hypovolemia may exist in patients within this range, and caution must be used in the interpretation of this value by itself.

In terms of practical aspects of CVP monitoring, while placement of catheters via the SVC or IJ is typically considered preferable, CVP measurements by femoral line are, on average, 0.5 mmHg lower and may be reliable as an alternative when line placement supradiaphragmatically is difficult or contraindicated. Additionally, saline administered through a multi-lumen CVC did not affect CVP when measured through the distal port, regardless of the rate of infusion. CVP can also be measured by external jugular vein examination, see Table 3.

Despite these limitations, CVP has an important role in the monitoring of critical patients. Because our patients are evaluated pre-resuscitation and often before the onset of volume overload and edema, the CVP may be more accurate in the ED population as compared to the ICU patient. However, because little literature exists apart from one well-performed randomized control trial discussed below, CVP’s role in the emergency population remains somewhat nebulous.

Preload Monitoring Applications
Preload monitoring is typically utilized to determine whether or not a patient would benefit from additional volume. However, while the primary application of preload monitoring is to assess fluid responsiveness, the utilization of static cardiac filling pressures like CVP and PAOP to predict volume response is problematic, as discussed further.

Fluid responsiveness: A discussion of fluid responsiveness must begin with its differentiation from preload. While preload reflects end-diastolic volume of the ventricles (typically estimated by filling pressures measured by invasive and minimally invasive methods) it provides no information regarding where on the Frank-Starling curve the heart is operating and, therefore, can be difficult to interpret. However, fluid responsiveness, while dependent on preload, describes the ability to increase cardiac output by infusing volume, see Figure 5. Predicting

TABLE 3 – Estimation Of CVP By External Jugular Vein Examination

1. Position the patient at a 30° – 45° angle.
2. Identify the external jugular vein (EJV). If not readily identifiable, increasing intrathoracic pressure by Valsalva maneuver or occluding the EJV at the base of the neck may help distend the vein.
3. Identify the apex of venous pulsations in the EJV by stripping. This is accomplished by placing one finger at the top of the venous column and spreading another finger along the vein’s course to its base. When the lower finger is released, the EJV will refill in a retrograde fashion and identification of the venous pulsation may be more clearly observed.
4. Measure the height of the EJV from the patient’s angle of Louis located at the 2nd intercostal cartilage on the sternum.
5. Add 5 centimeters of water to this measurement to account for the distance between the angle of Louis and the right atrium.
6. This value is the CVP in cm of water. Multiply by 0.75 to obtain CVP in mm Hg.
volume response is critically important in hemodynamically unstable patients, as fluid infusion in an already volume overloaded patient may result in volume overloaded patient may result in worsening of overall condition without any increase in cardiac output.

The method used to identify fluid responsiveness depends on whether the patient is breathing spontaneously or is deeply sedated/paralyzed and not triggering the mechanical ventilator. In both spontaneously breathing and mechanically ventilated patients, static cardiac filling pressures such as right atrial pressure (equivalent to CVP in the absence of vena-caval occlusive disease), PAOP, RVEDVI, and ITBVI are consistently unable to predict fluid responsiveness despite their ability to monitor preload.126-130

Dynamic measurements are much more promising in their ability to predict fluid response and rely on the physiologic relationship between respiratory variation and right ventricular function. A demonstration of this concept is provided by explaining the variation of blood pressure observed in intubated patients. A positive pressure (mechanical) breath decreases venous return which then decreases right ventricular filling and ejection fraction which in turn causes a decrease in left ventricular ejection fraction a few beats later during expiration and ultimately results in a decrease in systolic pressure. The magnitude of variation is exaggerated in volume depleted individuals and can be utilized to predict fluid response. In several studies, systolic pressure variation (SPV) has been found to correlate well with volume responsiveness in mechanically ventilated patients.127,131 Similarly, delta pulse pressure (DPP) greater than 13% (defined as the difference between the maximum pulse pressure during inspiration and the minimum pulse pressure during expiration) was found to be more sensitive than SPV in predicting fluid response,131 see Figure 6. Utilizing pulse contour analytic devices such as PiCCO™ to assess stroke volume variation (SVV) in cardiac surgery patients has also been shown to correlate well with volume responsiveness while re-demonstrating the limitations of static CVP measurements.129,132 Pre-ejection period variation (defined as respiratory variation of the time interval between the beginning of the R wave on the ECG and the upstroke of the radial artery pressure curve [PEPkt] or pulse plethysmographic waveform [PEPlet]) corresponds with fluid responsiveness in mechanically ventilated patients and was found to be as accurate as DPP and allows for completely non-invasive determination of fluid response with only pulse oximetry/plethysmography.133

In spontaneously breathing patients, the prediction of fluid response is more difficult. A single study found that a drop in right atrial pressure/ CVP greater than 1 mmHg during inspiration predicted fluid response in spontaneously breathing patients;126 these findings were not supported by a more recent study which also demonstrated no correlation with stroke volume variation and fluid response in spontaneously breathing patients.134

When used in conjunction with cardiac output monitoring, a fluid challenge may also be utilized to determine volume responsiveness. Volume is infused until CVP is increased by 2 mmHg; if cardiac output subsequently increases by 300 mL/min or more then the patient is likely operating on the ascending portion of the Frank-Starling curve and is considered fluid responsive.123 Relying on a similar concept but increasing central venous return by physical maneuvers instead of by infusing additional volume, a novel dynamic predictor of fluid response measuring change in aortic blood flow (ABF) using esophageal Doppler found that increased ABF greater than 10% during passive leg raise (45°) predicted volume response in both spontaneously breathing and mechanically ventilated patients. Additionally, this method may prove to be even more versatile as it is not affected by the presence of non-sinus rhythm, a limitation of other dynamic measurements.135

While the use of dynamic markers to predict fluid response seems promising, prospective trials that demonstrate their ability to affect outcome do not exist at this time. Interestingly, despite the well

![Figure 6. Delta Pulse Pressure](https://example.com/figure6.png)

Changes in pulse pressure over the respiratory cycle (delta PP) are calculated as the difference between Ppmax and Ppmin divided by the mean of the two values. A value of 13% or greater accurately predicted fluid responsiveness in mechanically ventilated patients without spontaneous breathing. (Used with permission of Chad Meyers, MD)131
documented limitations of static measurements in the prediction of fluid response, in a study that will be discussed at greater length later in this review, Rivers et al utilized a target CVP greater than 8 mmHg in spontaneously breathing patients or a CVP greater than 12 mmHg in mechanically ventilated patients with severe sepsis or septic shock and found a significant reduction in mortality when treatment was combined with other hemodynamic goals. However, the utility of CVP in the management of fluid resuscitation in septic patients does not seem to extend to the ICU. In a recent study, a CVP of 8 mmHg or less in spontaneously breathing patients or 12 mmHg or less in mechanically ventilated patients after 6 hours of resuscitation was not predictive of fluid response (defined as an increase in cardiac index of 15% or more). Additionally, no correlation was demonstrated when CVP measurements were combined with PAOP or a pre-volume challenge reduced stroke volume index. Thus, pre-resuscitation CVP values alone may have a positive predictive value of volume status and fluid response when less than 8 mmHg; however, this inference has not been evaluated objectively.

Preload Monitoring Key Points

- Dynamic markers of fluid responsiveness allow the clinician to appropriately administer volume in both the hemodynamically stable and unstable patient thereby avoiding volume overload and potentially worsening the patients overall clinical status.
- In a patient with a low cardiac output, markers indicating no fluid responsiveness should lead to the initiation of inotropic support to maintain adequate tissue perfusion.
- In the severely septic or septic shock patient, a CVP of 8 mmHg or less in the spontaneously breathing patient or 12 mmHg or less in the mechanically ventilated patient indicates the requirement for additional volume administration.

Tissue Perfusion Monitoring

Back to the patient suffering from severe acute pancreatitis: his mean arterial pressure is now stable and his cardiac index has normalized; however, his extremities remain mottled and cold and he has not yet produced any urinary output. You obtain a blood gas sample from his subclavian central venous catheter and note that his central venous oxygen saturation is 60%. You recheck for stroke volume variation on the cardiac output monitor but he now lacks any indication of fluid response. You begin a dobutamine infusion and an hour later, you re-sample his central venous blood, and his ScvO₂ is 71%. His toes are now warm, and 80 cc of urine is present in his Foley bag.

Months later, you hear from your ICU colleagues that the patient had a long complicated hospital course including numerous pancreatic necrosectomies. Against all odds, he eventually left the hospital for a rehabilitation facility a few weeks prior and is now doing well.

Perhaps the most important purpose to hemodynamic monitoring is to provide the answer to one question: “Are we getting oxygen to the tissues?” All of the markers we have discussed up until now answer this question only indirectly. Tissue oxygen balance is dependent on two variables: oxygen delivery (DO₂) and oxygen consumption (VO₂). Both variables are a function of cardiac output, oxygen carrying capacity, and oxygen saturation. If the body’s metabolic requirements for oxygen exceed oxygen delivery cellular dysoxia occurs, and if this dysoxia induces organ dysfunction then a state of shock exists. By varying the ratio of oxygen extraction, the quantity of oxygen delivered to the microcirculation is held constant over a wide range of hemodynamic states. However, a critical DO₂ value exists below which VO₂ becomes supply dependent; any further decreases in cardiac output, oxygen carrying capacity, or arterial oxygen content that lower delivery below this point will result in further decreases in VO₂ and the likelihood of organ dysfunction and shock increases, see Figure 7.

Figure 7. Critical Supply Dependent Oxygen Delivery
Central Venous and Mixed-Venous Oxygenation

Mixed-venous oxygenation (SvO₂) represents the oxygen saturation of blood in the pulmonary artery which consists of blood returning from the SVC, IVC, and coronary sinuses. Therefore, SvO₂ is an admixture of the venous return from the whole body. In contrast, central venous oxygenation (ScvO₂) represents the oxygen saturation of blood in the SVC alone. A normal SvO₂ in most individuals is 65-75%, but ScvO₂ is typically lower in healthy individuals and 5-10% higher in critically ill patients. Despite this, there is still good correlation in measurements as well as trends between the two values. ScvO₂ can be measured either continuously by specially designed central venous catheters utilizing fiberoptic reflectance spectroscopy or periodically by intermittent blood draws and blood gas analysis.

Central and mixed-venous oxygen saturation are important because they provide a global indicator of tissue perfusion and whole-body O₂ balance, which is difficult (if not impossible) to determine by clinical indicators. For instance, Rady et al demonstrated the presence of signs of global ischemia represented by decreased ScvO₂ and elevated lactate in absence of traditional clinical indicators of shock (such as elevated HR, decreased BP, or elevated shock index). Likewise, an ScvO₂ less than 65% predicted hemorrhage and was found to be more accurate than vital signs in traumatically injured patients. In another study, ScvO₂ was able to differentiate occult cardiogenic shock in patients with known EF less than 30% who were otherwise clinically indistinguishable.

Tissue Perfusion Monitoring Applications

Cardiac Arrest: Although not typically considered a primary indication for placement of a ScvO₂ catheter, an existing continuous ScvO₂ monitor may provide useful information in regards to the prognosis and adequacy of cardiopulmonary resuscitation. In one study, all patients with ScvO₂ greater than 70% demonstrated return of spontaneous circulation (ROSC), while no patient with a maximal ScvO₂ less than 30% attained ROSC. Additionally, adequate chest compressions may be reflected by an increase in ScvO₂ greater than 40%. ScvO₂ values between 40-72% indicate progressively increasing chances of ROSC. ScvO₂ values greater than 60% indicate that ROSC is likely and values greater than 72% indicate that ROSC has likely already occurred. In the post arrest patient, venous hyperoxia (ScvO₂ greater than 75%) was associated with non-survival in those with coincident lower range DO₂ values. Non-survivors had higher ScvO₂, higher lactate, and lower VO₂ at the same DO₂ as survivors. This derangement in VO₂ and oxygen extraction is hypothesized to be the result of microcirculatory abnormalities or large doses of epinephrine given during resuscitative measures.

Targeted Resuscitation: As previously discussed, the benefit of resuscitation strategies is likely a function of time at which protocol is started, and, not surprisingly, a targeted SvO₂ greater than 70% has not been shown to reduce mortality when applied to ICU patients if initiated late in these patients’ hospital course. However, in a landmark study investigating the effects of early hemodynamic optimization of severely septic patients in the first 6 hours of their hospital course, Rivers et al were able to demonstrate an overall reduction in mortality from 46.5% to 30% (AKR 16.5%), yielding a number needed to treat of 6. In their study, patients identified as suffering from septic shock or severe sepsis with SBP less than 90 mmHg despite fluid therapy or lactate greater than 4 mmol/L, respectively, were treated with liberal fluid therapy targeting CVP greater than 8 mmHg, and maintenance of MAP between 65 and 90 mmHg using vasopressors or vasodilators, see the Severe Sepsis Clinical Pathway insert. If patients continued to demonstrate global oxygen deficit (defined as decreased ScvO₂ less than 70%) then hemodynamic optimization of oxygen delivery was attempted by inotropic support and/or blood transfusion as well as the minimization of metabolic oxygen requirements by mechanical ventilation. The Early Goal Directed Therapy (EGDT) protocol has since been incorporated into the international Surviving Sepsis Campaign, and the findings have been supported by numerous studies demonstrating the feasibility and benefit of its implementation.

Regional Perfusion

Sublingual Capnometry

Unlike other shock states, microvascular flow patterns are deranged and heterogeneous in septic shock leading to regional dysoxia despite resuscitative measures aimed at restoring global oxygenation. This phenomenon, termed microcirculatory and mitochondrial distress syndrome (MMDS), has
been demonstrated by direct visualization of the microvasculature by orthogonal polarization spectral (OPS) imaging. Microvascular shunting in sepsis can lead to lower tissue PO2 values than venous PO2 or a PO2 gap that could result in apparent normal or high values of global oxygenation (such as SvO2/ScvO2) despite significant regional tissue dysoxia. These microvascular derangements have important implications and when compared to survivors of sepsis, microcirculatory alterations predicted non-survival in patients despite similar indicators of global perfusion (SvO2, lactate) in both groups.

Estimation of microcirculatory competency can be accomplished non-invasively by the measurement of CO2 in the tissue. This is based on the observation that CO2 accumulation in the tissue reflects low blood flow and decreased CO2 washout. Tissue CO2 can be measured by a variety of methods, including gastric tonometry, sublingual capnometry, or buccal capnometry. Sublingual capnometry non-invasively measures CO2 by sublingual optode and has largely replaced gastric tonometry due to its relative ease of insertion and greater reliability in certain patient populations. Sublingual CO2 difference (PslCO2–PaCO2) was found to correlate well with microvascular circulation determined by OPS, suggesting that the main determinant of PslCO2 was microcirculatory flow.

Sublingual Capnometry Applications
The minimal invasiveness of sublingual capnometry has the potential to create a useful triage tool for the identification and stratification of the critically ill patient in the ED. A PslCO2 greater than 70 mmHg was found to correlate with circulatory shock and decreased likelihood of hospital survival, with normal values ranging between 43 to 47 mmHg and averaging 45.2 ± 0.7 mmHg. In a more recent study, an initial PslCO2-PaCO2 difference greater than 25 mmHg had a PPV of 79% and a NPV of 73% of non-survival.

Additionally, by functioning as a surrogate marker of microcirculatory flow, PslCO2 could potentially be utilized to monitor therapies directed at microvascular recruitment. Dobutamine has been shown to have a positive effect on microvascular flow in sepsis separate from its effect as an inotrope as determined by both OPS and PslCO2. Other recruitment strategies, such as nitroglycerin infusion after adequate fluid loading, have also been found to have a positive effect on microvascular flow by OPS in sepsis but have not yet been correlated with PslCO2. Although these studies are promising, none have been evaluated for their benefit on mortality or morbidity.

Percutaneous O2 and CO2
Percutaneous O2/CO2 can be noninvasively monitored by heated probes placed on the skin. The monitor requires approximately 15 minutes to equilibrate and the probes must be moved every 4 hours to avoid skin burns. While PtcO2 correlates with PO2 in normal patients, it significantly overestimates PO2 in low flow states. This is reflected by the relationship between low PtcO2/FiO2 ratio and the incidence of shock. Similarly, PtcCO2 levels climb and diverge from PaCO2 in states of low cardiac output. In trauma patients, percutaneous measurement of O2/CO2 has been shown to be a predictor of mortality with PtcCO2 values of greater than 90 torr, PtcCO2 remains greater than 60 torr for 30 minutes or PtcO2 remains less than 50 torr for 60 minutes. In another trial studying the predictive value of percutaneous measurements in trauma patients, an increased PtcCO2 and decreased PtcO2/FiO2 ratio correlated well with non-survival.

Tissue Perfusion Key Points
- ScvO2 less than 70 mmHg or greater than 80 mmHg represents inadequate global perfusion and, therefore, oxygenation. During early supply dependent stage of resuscitation, these findings should prompt a diligent attempt to normalize oxygen balance by correcting anemia, optimizing cardiac index, and minimizing oxygen consumption.
- Sublingual capnometry is a non-invasive and quick method of determining regional perfusion. PslCO2 greater than 70 mmHg or a PslCO2-PaCO2 gap greater than 25 mmHg correlate with non-survival and severity of critical illness and signal the potential necessity for aggressive management and resuscitative measures.
- Percutaneous CO2 greater than 90 torr or remaining greater than 60 torr for 30 minutes and percutaneous O2 remaining less than 50 torr for 60 minutes are predictors of mortality in the critically ill trauma patient.
Because the management of the critically ill often necessitates aggressive control of the airway and subsequent deep sedation, clinical assessment of neurologic function is difficult if not impossible. Typically, complex monitoring devices such as electroencephalography (EEG) are beyond the scope of most emergency physician’s practice; however, the development of bispectral electroencephalographic monitors provides the non-specialist with a simplified tool to assess a patient’s cortical activity.

**Bispectral Electroencephalographic Monitors**

The bispectral index (BIS) is derived from bispectral EEG signal processing of bioelectric potentials obtained by forehead leads and displayed as a numerical value ranging from 0 (representing cortical silence) to 100 (representing full alertness). Its primary clinical use has been in the operating room to assess adequate sedation and minimize anesthetic usage. Recent studies have attempted to demonstrate benefit in other settings, such as the emergency department.165

**Bispectral Electroencephalographic Monitors Applications**

Determining the level of adequate sedation in mechanically ventilated patients in the ED is difficult, and the accuracy of various clinical scoring systems is considered inadequate.166 Therefore, a monitoring device with the ability to objectively determine sedation for our critically ill patients might be helpful. Although findings concerning the use of the BIS as a method of continuous assessment of adequate sedation in mechanically ventilated patients in the ICU were initially encouraging,167 recent studies in a more heterogeneous surgical ICU population and the emergency department have not shown any correlation between BIS and clinical markers of sedation.168,169 Despite these findings, a prospective observational study in the ED found good correlation between a consistent BIS less than 70 and lack of recall concerning intubation, suggesting the possibility of some utility of BIS in the prediction of amnesia and its dependence on adequate level of sedation in the ED.170

BIS may be useful in the initial assessment of traumatically brain injured patients with relatively normal clinical exams. While prior studies have shown that clinical indicators such as low Glasgow Coma Scale are associated with poor outcomes in TBI patients,171 they are typically inadequate when excluding injury or radiographic abnormalities in those patients with normal or near-normal physical exam.172 In a single study of closed head injury patients, an initial BIS greater than 95 excluded all patients with poor outcomes regardless of radiographic findings, while, conversely, all patients with poor outcomes had an initial BIS less than 95, including patients with normal physical and radiographic examination. In this study, poor outcome was defined as death, persistent vegetative state, or discharge to long term care facility for neurologic disabilities; these results need prospective validation before any clinical use.173

**BIS Monitoring Key Points**

- Bispectral index monitoring allows for a non-invasive assessment of cortical activity. While BIS usage intraoperatively has been shown to be accurate in predicting adequate sedation, no correlation between clinical markers of sedation and BIS have been found in mechanically ventilated patients in the ED.
- Although not prospectively validated, an initial BIS less than 95 may predict potential poor neurologic outcome in patients suffering from closed head injury.

**Controversies**

The availability of less invasive monitoring devices has already and will continue to change the landscape of critical care in the emergency department. However, the necessity of such devices and the potential requirements they mandate of emergency physicians is a source of considerable controversy. In a questionnaire distributed to academic emergency departments in July of 2004, a dismal 7% of departments implemented EGDT in the care of patients demonstrating signs of severe sepsis or septic shock. The lack of specialized monitoring devices was listed as the reasoning in 75% of institutions questioned.174

The benefit of the early hemodynamic optimization of the critically ill, a concept encapsulated by EGDT, applies regardless of the presence or absence of specialized monitors. While the use of continuous monitoring may be preferable, “low-tech” alternatives do exist when they are unavailable. For example, the measurement of central venous pressure is possible without the use of a pressure transducer. Using an infusion line on an existing central venous
catheter, the stopcock is held at the patient’s phlebo-
static axis (mid axillary line, 4th intercostal space in
the supine patient), a length of saline filled extension
tubing is attached, and the stopcock is opened to the
new tubing. At this point, a column of fluid exists
from the level of the right atrium to the meniscus in
the extension tubing and as the patient breathes, the
meniscus of the column will rise and fall. The height
of the column measured from the phlebotstatic axis to
the meniscus represents the central venous pressure
in centimeters of water. This number can be convert-
ed to mmHg by multiplying by 0.75. If this level of
invasiveness is still too much, the CVP can be esti-
mated by examination of the external jugular vein,
see Table 3 on page 16.

In the absence of continuous ScvO2 monitors,
intermittent samples of blood drawn from the central
venous catheter may be sent for blood gas analysis.
However, another simple bedside clinical sign can
also be of significant benefit in the identification of
perfusion abnormalities. In an eloquent study pub-
lished by Joly et al in 1969, the temperature of the

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**Risk Management Pearls**

1. **Relying on clinical indicators of hemodynamic insta-
bility to rule out critical illness.**

   While vital signs are valuable tools if abnormal, 50%
of critically ill patients may present with normal blood
pressure and heart rate. Indicators of global or region-
al perfusion (e.g., lactate, ScvO2, sublingual capnome-
try) or cardiac index are considered more reliable.

2. **Estimating blood pressure through physical exam.**

   Palpating various peripheral pulses for blood pressure
determination is both unreliable and misleading.
Utilize intermittent automated cuff pressure or contin-
uous intra-arterial monitoring to determine blood
pressure in the critically ill.

3. **Leaving the automated blood pressure cuff set to
record at 30 minute intervals.**

   Often, default recording times for blood pressure
measurement are set at lengthy time intervals. This
will invariably lead to significant delay in appropriate
intervention or missing transient episodes of hypoten-
sion, which may have significant implications in the
critically ill.

4. **Utilizing normal SpO2 as an indicator of correct
endotracheal tube placement.**

   While pulse oximetry is a valuable tool in preventing
hypoxic events, because of the shape of the oxyhemo-
globin dissociation curve, significant desaturation may
occur before changes in SpO2 are evident.

5. **Relying on ETCO2 as an estimate of PaCO2.**

   While ETCO2 may reflect PaCO2 in hemodynamically
stable healthy volunteers, its relationship is unpredi-
table and unreliable in patients who are unstable or
with abnormal pulmonary function. For example,
monitoring of the decompensated COPD patient for
hypercapnia by ETCO2 alone may lead to overconfi-
dence in clinical status and the overlooking of signifi-
cant clinical deterioration (i.e., an ETCO2 of 30 mmHg
may actually reflect a PaCO2 of 95 mmHg due to sig-
nificant pulmonary shunting).

6. **Utilizing CVP as an estimate of preload in the resus-
citated patient.**

   The utility of CVP as a measure of preload and fluid
responsiveness has never been validated in patients
after 6 hours of resuscitation. Reliance on CVP to
monitor volume status after this period may be mis-
leading.

7. **Being reassured by a very high ScvO2.**

   ScvO2 greater than 80% reflects decreased oxygen uti-
lization by the tissues. In septic shock, this can be a
sign of microcirculatory abnormalities and mitochon-
drial dysfunction.

8. **Being reassured by a normal SpO2 following smoke
inhalation.**

   The presence of carboxyhemoglobinemia following
smoke inhalation may mask significant hypoxia due to
the limitations of traditional pulse oximetry caused by
the overlap of light absorbance between oxyhemoglo-
bin and carboxyhemoglobin.

9. **Being reassured by a high non-invasive blood pres-
sure measurement in the initial resuscitation of
severe trauma.**

   Because of high endogenous catecholamine levels, the
initial cuff blood pressure measurement in the arm of
a patient with large amounts of blood loss may read a
high value even though the actual central arterial
pressure is low. The most accurate blood pressure
readings in these patients are obtained from a femoral
intra-arterial line. The readings between this catheter
and the cuff measurements will often be widely dis-
crepant.

10. **Relying on monitoring devices over clearly contra-
dictory clinical judgment.**

    Since all monitoring devices are sometimes inaccu-
rate, clinical gestalt still plays a large role in the evalua-
tion of a critically ill patient. If a patient looks much sicker
than a monitoring device indicates, continued vigil-
lance is often rewarded.
Unfortunately, the intense labor and time required, that role will also likely continue to expand. As the importance of the emergency physician's role in the care of the critically ill continues to be redistributed and you are left wondering if the patient's outcome could have been different. 

Several hours after your pneumonia patient from yesterday was transferred to the floor, she became increasingly short of breath and hypoxic requiring intubation. A subsequent arterial blood gas revealed a lactate of 7.8 mmol/L, and she continued to have no urine output. Liberal IV fluid was administered through a central venous catheter along with vasopressors. Despite the aggressive management of the floor team, she coded later that morning and subsequently died as a consequence of septic shock. When the case was presented at the hospital's M&M conference, a copy of the River's study on EGDT was distributed and you are left wondering if the patient's outcome could have been different.

Summary

As the importance of the emergency physician's role in the care of the critically ill continues to be redefined, that role will also likely continue to expand. Unfortunately, the intense labor and time required—ments of critical care will also undoubtedly further strain the staff of already overcrowded emergency departments. Thus, the development of minimally and completely non-invasive monitoring modalities is crucial, not only to guide treatment of the critically ill more effectively but also more efficiently and quickly. Additionally, the availability of advanced monitoring devices will potentially increase the diagnostic acumen of the emergency physician, minimizing the misidentification of the less severely ill and limiting the misappropriation of valuable resources. Ideally, by enhancing the ability of the emergency physician to initiate optimal care of the critically ill, effective monitoring will play an important role in the integration of our specialty in the continuum of critical care. A continuum which begins in the emergency department and inarguably has an indelible impact on the outcome of the most fragile patients.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, are included in bold type following the reference, where available.

12. AARC (American Association for Respiratory Care) clinical practice guidelines.
89. Sakka, S.G., et al. Is the placement of a pulmonary artery catheter still
88. Cuschieri, J., et al. Central venous-arterial carbon dioxide difference as
EBMedicine.net • July 2007 25 Emergency Medicine Practice©
92. Bein, B., et al. Comparison of esophageal Doppler echocardiographic,
91. Jaffe, M.B. Partial CO2 rebreathing cardiac output—operating princi-
90. Lefrant, J.Y., et al. Training is required to improve the reliability of
effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-
85. Cholley, B.P . and D. Payen. Noninvasive techniques for measurements
84. Chaney, J.C. and S. Derdak. Minimally invasive hemodynamic moni-
78. Rowley, K.M., et al. Right-sided infective endocarditis as a conse-
81. Jaffe, M.B. Partial CO2 rebreathing cardiac output—operating princi-
75. Le Tulzo, Y., et al. Reproducibility of thermodilution cardiac output
80. Jarden, P., et al. Training could improve the reliability of the noninvasive
eutrocardiac catheter: A prospective analysis in 219 patients. Crit Care
82. Bein, B., et al. Comparison of esophageal Doppler echocardiographic,
fick, and thermocardiographic cardiac output in critically ill patients. J
87. Simonsson, G., et al. Noninvasive monitoring of cardiac output using the
electrically derived CO2 rebreathing technique: a comparison with the traditional
86. Madan, A.K., et al. Shock index and the clinical assessment of
84. Slama, K., M. Ikrar, and T. Al-Bardan. Noninvasive assessment of cardiac output using a
83. Odenstedt, H., L. Sjöholm, P. Björk, and J. Lindblom. The effect of
82. Nilsen, S. and J. Bakkum. Comparison of transpulmonary thermocardiogram,
81. Jaffe, M.B. Partial CO2 rebreathing cardiac output—operating princi-
80. Lefrant, J.Y., et al. Training is required to improve the reliability of
t methods of measuring extracorporeal blood flow. J Cardiothorac
89. Sakka, S.G., et al. Is the placement of a pulmonary artery catheter still
88. Cuschieri, J., et al. Central venous-arterial carbon dioxide difference as
91. Jaffe, M.B. Partial CO2 rebreathing cardiac output—operating princi-
90. Lefrant, J.Y., et al. Training is required to improve the reliability of
effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-
85. Cholley, B.P . and D. Payen. Noninvasive techniques for measurements
84. Chaney, J.C. and S. Derdak. Minimally invasive hemodynamic monitor:
a review.
78. Rowley, K.M., et al. Right-sided infective endocarditis as a conse-
76. Meier, B., et al. Comparison of esophageal Doppler echocardiographic,
fick, and thermocardiographic cardiac output in critically ill patients. J
87. Simonsson, G., et al. Noninvasive monitoring of cardiac output using the
electrically derived CO2 rebreathing technique: a comparison with the traditional
86. Madan, A.K., et al. Shock index and the clinical assessment of
84. Slama, K., M. Ikrar, and T. Al-Bardan. Noninvasive assessment of cardiac output using a
1. End-tidal CO₂ monitoring has been shown to:
   a. verify correct tube placement after RSI and throughout intubation.
   b. predict adverse respiratory events in procedural sedation.
   c. guide pulse checks and demonstrate ROSC during CPR.
   d. all of the above

2. The reliability of pulse oximetry is affected by:
   a. severe anemia.
   b. poor peripheral perfusion.
   c. poor waveform.
   d. A and B

3. CVP in the setting of sepsis:
   a. is reliable only when obtained by internal jugular or subclavian central venous catheters.
   b. when decreased, may represent hypervolemia.
   c. when decreased, always represents hypovolemia.
   d. when decreased, can be utilized to predict fluid responsiveness in ICU patients.

4. Central venous oxygenation:
   a. if less than 70%, represents global perfusion abnormalities.
   b. if greater than 90%, represents adequate resuscitation of septic shock.
   c. if greater than 75%, represents good prognosis in patients with ROSC.
   d. A and B

5. In the critical care literature, which of the following has been associated with improved mortality?
   a. accurate determination of patients who are volume responsive
   b. late hemodynamic optimization of severely septic patients upon admission to the ICU
   c. increasing cardiac output to supranormal levels to increase oxygen delivery in certain high risk patients
   d. all of the above

6. Bispectral electroencephalographic monitoring:
   a. allows for determination of mechanically ventilated patients’ level of sedation in the operating room.
   b. allows for determination of mechanically ventilated patients’ level of sedation in the emergency room.
   c. allows for determination of mechanically ventilated patients’ level of sedation in the ICU.
   d. none of the above

7. Microvascular flow and regional perfusion abnormalities:
   a. correlate with markers of global perfusion such as central venous oxygen.
   b. are typically heterogenous in states of traumatic hemorrhagic shock.
   c. are typically homogenous in states of severe sepsis and septic shock.
   d. can be responsible for significant regional oxygen debt in the presence of elevated central venous oxygen levels.

8. Pulse oximetry has a vital role during intubation and airway management; it can be used to:
   a. confirm endotracheal tube placement.
   b. always obtain accurate arterial oxygenation monitoring immediately following placement of the tube.
   c. provide a reasonable approximation of PaO₂ during ventilation of an intubated patient.
   d. none of the above

9. The pulmonary artery catheter’s:
   a. benefits on patient mortality are clearly established in numerous studies.
   b. risks may outweigh its benefits.
   c. main use is to obtain accurate MAP measurements.
   d. should be abandoned for new, non-invasive output monitors which have clearly proven their utility.

10. The utilization of cardiac output measurements determined by pulmonary artery catheterization to guide therapy:
    a. has no benefit on patient morbidity.
    b. has been shown to benefit patients in decompensated congestive heart failure.
    c. is more reliable than CVP to guide fluid resuscitation.
    d. has demonstrated benefit in patient groups with high mortality when initiated early in their hospital course.

11. Mixed venous oxygen saturation (SvO₂):
    a. refers to a sample taken from the tip of a central venous catheter.
    b. requires the lab to mix a sample from a PAC with a sample from an arterial puncture.
    c. refers to a mixture of blood from the SVC, IVC, and coronary sinuses.
    d. is best obtained from the subdiaphragmatic femoral vein.

12. An ETCO₂ of less than 10 during arrest:
    a. predicts dismal outcome at any point during cardiac arrest.
    b. indicates that the respiratory therapist should decrease ventilation frequency.
    c. always indicates a poorly positioned ET tube.
    d. predicts no ROSC 20 minutes after PEA arrest.

13. In Early Goal Directed Therapy (EGDT):
    a. inotropes should always be started before volume loading to prevent edema.
    b. an endpoint is an ScvO₂ between 70 and 80.
    c. requires vasopressors regardless of blood pressure.
    d. resulted in a mortality reduction of only 2%.

14. Critically ill emergency department patients in shock may benefit from:
    a. intra-arterial blood pressure monitoring to measure accurate MAP.
    b. CVP monitoring to help guide pre-resuscitation volume optimization.
    c. continuous capnography to ensure continued endotracheal tube placement and early detection of hypercapnia.
    d. all of the above
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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of the literature; and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

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Critical Care Monitoring In The Emergency Department

Clinical Pathway: Severe Sepsis Protocol

- Septic patient with hypotension or lactate greater than 4
- Cultures and broad spectrum antibiotics
- 20 cc/kg isotonic fluid bolus
- Central venous access in internal jugular or subclavian using full sterile technique

Hyoxemia

- YES
  - Intubation and lung protective ventilation
  - Sedation and pain control using sedation scale

NO

- CVP*
  - Less than 10
  - 500 cc isotonic fluid bolus
  - Greater than 10

- MAP‡
  - Less than 65
  - Titrate norepinephrine
  - Decadron 4 mg IV Q6
  - Greater than 65

- ScvO₂
  - Less than 70
  - Transfuse PRBC
  - Greater than 70

HCT

- Less than 30
  - Titrate dobutamine
- Greater than 30

*If patient is hypotensive, start pressors before fluid loading completed, then titrate them off if possible
‡If MAP is greater than 90, start nitroglycerin infusion


This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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