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Caring for Critically Ill Patients in the Emergency Department

Introduction

The practice of emergency medicine has evolved significantly since the first 24/7 emergency departments (EDs) opened in the 1950s and 1960s. In the past few decades, EDs have experienced an onslaught of increased patient volumes, increased demand for critical care services, increasing ED lengths of stay (LOS), and increased patient boarding.¹⁻³ These conditions fuel legitimate concern of an ED environment that is arguably becoming a threat to patient safety, specifically with regard to critically ill patients.^{3,4}

Emergency departments have an established record of providing a significant volume of critical care with respect to management, procedures, and resource utilization.^{1,5-9} Nelson et al reported that in a large, urban hospital, the ED provided 15% of all critical care in the hospital, both pediatric and adult. Nguyen and coworkers documented that the ED provided 467 days of critical care management each year for a heterogeneous cohort of critically ill patients.⁹ The authors also noted that the greatest clinical improvement occurred in the initial six hours while in the care of the ED. Most recently, a cross-sectional analysis from the National Hospital Ambulatory Medical Care Survey revealed that the volume of hours of care provided to ED patients ultimately admitted to an intensive care unit tripled (3.19 million to 9.9 million hours per year; $P < 0.01$) from 2001 to 2008.¹⁰

Although there is significant overlap between the critical care practiced in the ED and that practiced in an intensive care unit, significant differences still exist.¹¹ Emergency physicians (EPs) receive patients from the community who are undifferentiated, undiagnosed, unaccessed, and unresuscitated. Most patients arrive in the intensive care unit (ICU) having undergone an initial assessment, resuscitation, and stabilization, often in the ED. Additionally, there are now several time-sensitive interventions targeting critically ill patients across a spectrum of etiologies that EDs are charged with mastering and executing to improve patient outcome.^{12,13} It makes sense, therefore, that competency in the resuscitation of critical medical and trauma patients is one of the core competencies of the training of EPs according to the Accreditation Council for Graduate Medical Education (ACGME).¹¹ The objective of this report is to define the scope of the problem and describe strategies and management options to optimize the care of ICU patients cared for and boarded in the ED.

Recognition of Shock and Principles of Resuscitation

The basic principles of oxygen delivery (DO_2) and oxygen consumption (VO_2) are important concepts. Traditionally, shock has been defined as hypotension with evidence of hypoperfusion or end organ dysfunction. An alternative view is that shock should be defined as an imbalance between DO_2 and VO_2 . Conceptually, there are four vital components of oxygen transport:

Executive Summary

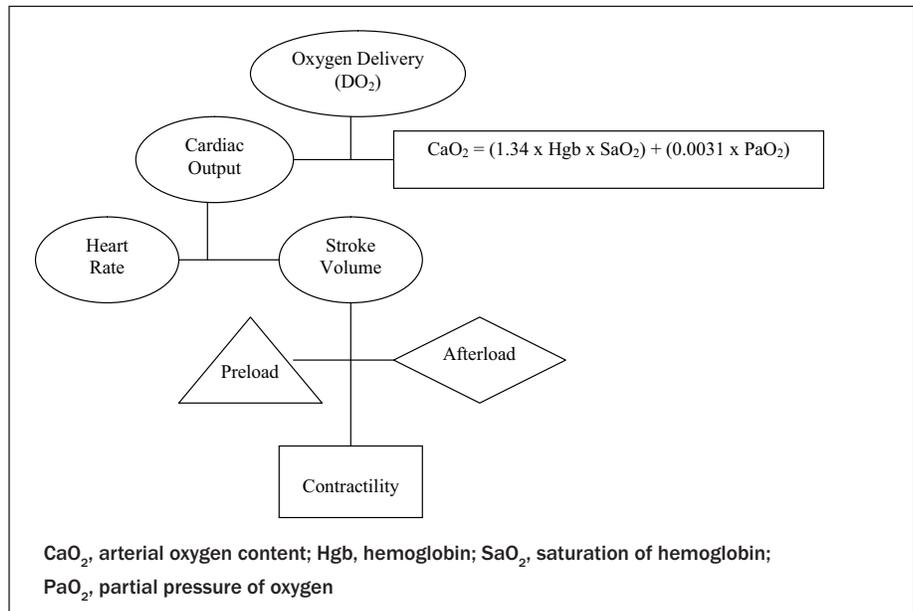
- Early hemodynamic monitoring or serum lactate can detect impaired perfusion with greater sensitivity than clinical assessment or vital signs.
- Oxygen demand can be reduced by lowering fever and providing mechanical ventilation.
- Alternative modes of mechanical ventilation can be used when standard volume-cycled techniques are ineffective.
- Consider sepsis in any unstable patient and administer broad-spectrum antibiotics early.

mechanical respiration to bring oxygen into contact with pulmonary vasculature, alveolar-capillary diffusion, bulk flow of oxygen primarily via an oxygen carrier (hemoglobin), and, finally, diffusion from the blood to mitochondria.¹⁴

Normally, the body's DO_2 is more than adequate to meet its basic metabolic demands. Oxygen delivery can be broken down into its primary components. (See Figure 1.) Essentially, any intervention performed or recommended to resuscitate a critically ill patient can fall into one of these boxes. When disease occurs, it can lead to either a decrease in DO_2 (i.e., myocardial infraction causing decrease in cardiac output) or a substantial increase in VO_2 (i.e., due to the onset of severe sepsis requiring increasing oxygen requirements), which results in a state of delivery-consumption mismatch. (See Figure 2.) As DO_2 falls below the critical threshold of VO_2 , the body shifts into anaerobic metabolism manifested by rising serum lactate levels and declining mixed venous oxygen saturation values.

Oxygen consumption is arguably the more complex side of this equation. Usually, resuscitation and supportive care will allow the body to heal and eventually normalize its VO_2 . The physician has a few interventions to reduce oxygen consumption. A fever will increase the basic metabolic rate and worsen the mismatch, so antipyretic medications to treat a fever are important. Additionally, for patients even in mild respiratory distress, increased work of breathing will cause a marked imbalance in the equation by consuming DO_2 . Therefore, the clinician should strongly consider

Figure 1: Flow Diagram of the Components of Oxygen Delivery



intubating patients with objective evidence of significant or worsening hypoperfusion even without respiratory distress.

Finally, no discussion about shock would be complete without a review of the various forms of shock. (See Table 1 and Table 2.) The most important component of this diagnosis is the history. In the ED, details about hemodynamics such as cardiac output (CO) and systemic vascular resistance (SVR) are not often available; therefore, a detailed exam and assessment coupled with an expedited workup is necessary. Key components of the history and workup include a primary and secondary survey (evidence of trauma, perfusion status, pulses, mental status, neurologic exam, and cardiopulmonary exam), serum lactate (to assess global perfusion), and electrocardiogram (ECG) (cardiogenic or toxic-metabolic causes). If

an etiology of shock is not readily apparent (i.e., massive gastrointestinal bleed or trauma), an expanded workup is recommended, which can be augmented by the use of specific hemodynamic monitoring techniques, including ultrasound.¹⁵ Still, a general rule with undifferentiated shock is to start with the ABCs (airway, breathing, circulation), initiation of an adequate fluid bolus, which is defined as at least 20-30 mL/kg for hypotensive patients, and expedited laboratory and radiographic workup as appropriate. The serum lactate level remains a reliable marker of hypoperfusion, and patients with mild elevations > 2.0 mmol/L have increased mortality and morbidity.^{16,17} A good rule of thumb for undifferentiated shock is to always consider sepsis. Sepsis is the most common form of shock, and in septic shock, antibiotic delay beyond the first hour

of presentation is associated with marked increases in in-hospital mortality.^{18,19}

Hemodynamic Optimization

Hemodynamic monitoring can be a challenge to implement in the ED. Classically, hemodynamics employed the use of a pulmonary artery catheter (PAC); however, its utility has been debated and its use has fallen out of favor due to lack of a clear demonstrable outcome benefit.²⁰ The PAC is not useful to most ED physicians, and hemodynamics can be assessed in various other ways.

Available hemodynamic monitoring devices have varying degrees of invasiveness, and there is little evidence to support one device over another.²¹ The purpose of any type of hemodynamic monitor is to ensure the “upstream” adequacy of DO_2 . This consists of the interplay among the arterial oxygen content, preload, afterload, and contractility.

While the modality of monitoring and which endpoint to use during resuscitation may not be crystal clear, there is definite benefit in targeting defined metabolic goals rather than vital signs. Donati et al utilized an oxygen extraction ratio (OER) target of < 27% in high-risk surgical patients, while Rivers et al targeted CVP and $ScvO_2$ in septic shock patients.^{12,22} Both studies reported outcome benefits related to using these targets as endpoints of resuscitation despite normalized vital signs. Moreover, Kern and Shoemaker’s meta-analysis of hemodynamic optimization showed that critically ill patients (> 20% mortality) fared better if goals were met before the onset of organ failure.²³

Estimates of Preload

The concept of preload optimization relates to the Frank-Starling law of the heart. (See Figure 3.) Cardiac output is a product of heart rate (HR) and stroke volume (SV). With all else being equal, an increase in SV or HR will lead to increases in cardiac output. Increases in preload will increase SV only up until the

Figure 2A: State of Consumption-Delivery Balance

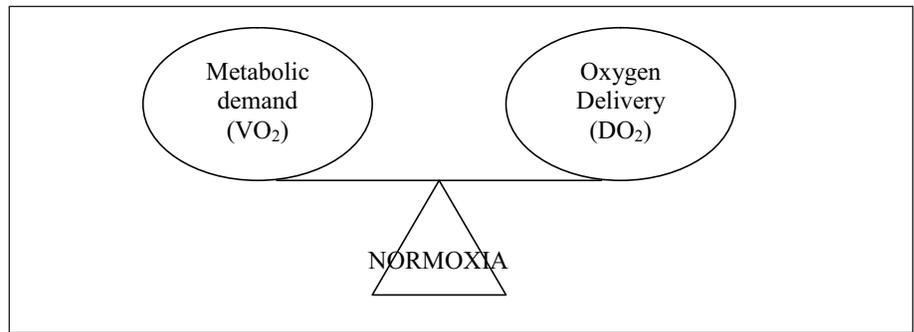


Figure 2B: Development of Shock and State of Consumption-Delivery Mismatch

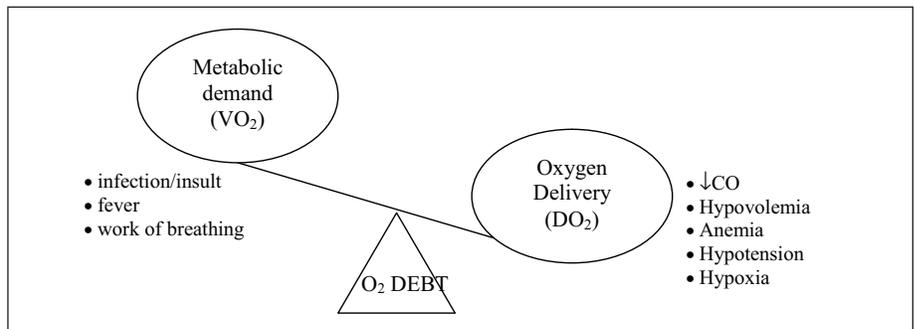
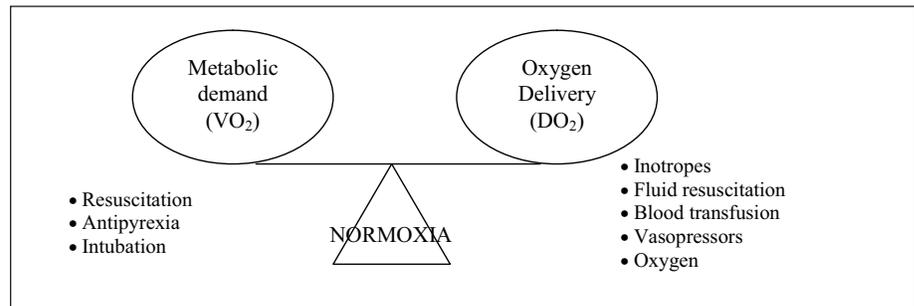


Figure 2C: Following Resuscitation Efforts, Return to a State of Consumption-Delivery Balance



Frank-Starling curve plateaus, and at higher levels of preload, may actually decrease SV as the curve arcs downward. The heart has a finite volume, and over-distention will impair contractility and SV, while HRs that are extremely high will hamper diastolic filling.

Static Values — Central Venous Pressure (CVP)

For the EP, perhaps the most widely used assessment of preload is the CVP. Most clinicians understand that a low CVP (< 8-10 mmHg) may represent intravascular volume depletion. As a surrogate for LV-EDV, a volume that cannot be directly measured, the CVP is criticized because it is subject to several

intrinsic (i.e., tricuspid regurgitation) as well as extrinsic physiologic conditions (i.e., PEEP) that can lead to measurement errors.²⁴ For example, a CVP of 12 mmHg in a young, healthy patient without cardiopulmonary disease who is not on a ventilator may indicate preload is optimized, whereas it may not in a patient who has an ejection fraction of 10% with ARDS on mechanical ventilation. The CVP remains a legitimate resuscitation target and is most accurate in patients without a history of cardiopulmonary pathology, but should be interpreted with appropriate regard to its limitations. A patient with a history of cardiomyopathy who presents in septic shock with an initial CVP of 18 mmHg

Table 1: Common Physical Exam and Hemodynamic Findings of Patients in Different Forms of Shock

	HR	Skin Exam	CO	CVP	SVR	Comments
Hypovolemic	↑	Variable	↓	↓	↑	Need to find source of volume loss
Cardiogenic	↓↑	Cool/ diaphoretic	↓	↑	↑	“Pump failure” or rhythm disturbance
Distributive						
Septic (early)	↑	Variable	↑	↓	↓	Hyperdynamic state
Septic (late)	↑	Cool	↓	↑↓	↑	Hypodynamic state
Neurogenic	↓	Warm	↓	↓	↓	Survey for co-existent trauma
Anaphylaxis	↑	Variable	↓	↓	↓	Can develop bradycardia prior to arrest
Obstructive	↑	Cool	↓	↑	↑	CXR or bedside ultrasound can lead to quick diagnosis
Dissociative	↑	Warm	↓	↑↓	↓	Variable presentations

HR, heart rate; CO, cardiac output; CVP, central venous pressure; SVR, systemic vascular resistance

Table 2: Examples of Etiologies and Shock Subtypes

<p>Hypovolemic</p> <ul style="list-style-type: none"> • Hemorrhage • Serum/plasma loss • Gastroenteritis • 3rd spacing 	<p>Cardiogenic</p> <ul style="list-style-type: none"> • Myocardial infarction (and complications) • Dysrhythmia • Congestive heart failure • Congenital heart disease
<p>Distributive</p> <ul style="list-style-type: none"> • Anaphylactic • Neurogenic • Septic 	<p>Obstructive</p> <ul style="list-style-type: none"> • Tension pneumothorax • Cardiac tamponade • Aortic dissection • Massive pulmonary embolism
<p>Dissociative</p> <ul style="list-style-type: none"> • Heat stroke • Cyanide • Carbon monoxide 	

is still most likely intravascularly depleted, requiring further volume expansion. In such cases, an alternate method of preload measurement might be useful.

Dynamic Measurements

Responsiveness to other modalities, whether it is a fluid bolus, a straight-leg raise test, or from normal respiratory physiology, can provide valuable information. Dynamic parameters such as pulse pressure variation (PPV), systolic blood pressure variation (SBPV), and stroke volume variation (SVV) vary in response to intervention, and the amplitude of the variance can

be a useful determinant of volume responsiveness. For example, a stroke volume (SV) or cardiac output (CO) increase by 10% after an IV fluid bolus or passive leg raise suggests a patient’s heart is operating on the steeper portion of the Frank-Starling curve and, therefore, the patient is fluid responsive. An SVV (normal < 10-15%) persistently > 20% suggests a preload-dependent state and, therefore, someone requiring fluid resuscitation.²⁵ Again, these parameters are far from perfect, but they appear to have sensitivities of 80-90%, with specificities of 85-95% with regard to predicting patients who will respond to fluid boluses.²⁶⁻²⁸

Continuous Cardiac Output

Several devices are commercially available to provide similar general measurements, including CO, systemic vascular resistance (SVR), indices of fluid status (such as thoracic fluid content [TFC]), and dynamic parameters such as SVV.²¹ The esophageal doppler monitor (EDM) provides information based on the flow in the descending thoracic aorta. Arterial line-based or noninvasive blood pressure finger-cuff devices use a pulse contour analysis algorithm. Some systems additionally utilize the concept of transpulmonary thermodilution to provide information about CO and extravascular lung water (EVLW). Some information may be obtained using Doppler ultrasound principles and Bernoulli’s equation. Thoracic bioimpedance and bioreactance both utilize the energy change of a low voltage current across the chest to determine cardiac output. While these devices have been compared and correlated to the PAC techniques, no modality has been shown to provide better clinical outcomes over another.²⁹ These devices all, for the most part, provide similar information regarding the hemodynamics of the patient beyond what the clinician could otherwise measure through examination, laboratory, or radiographic assessment. Ideally,

their use will improve the clinician's diagnostic ability or alter management with respect to fluid resuscitation, introduction of inotropes, or selection of vasopressor.³⁰

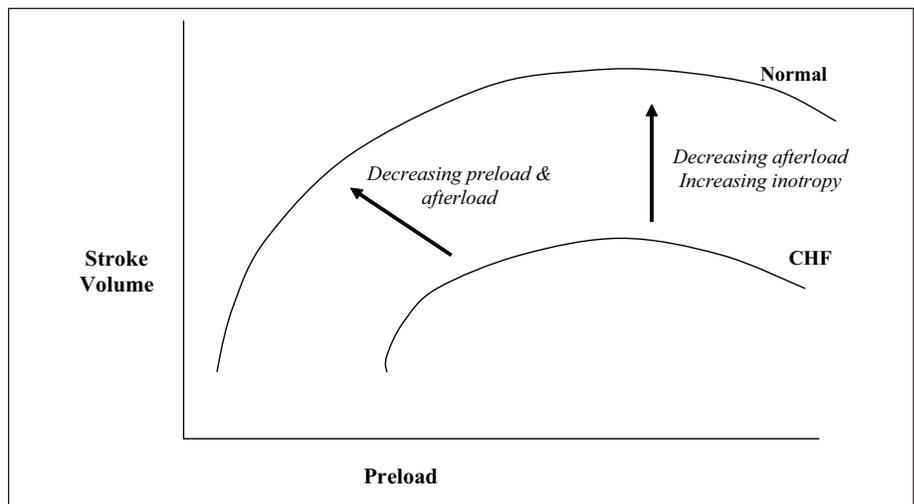
Estimates of Afterload

It has long been touted that oscillometric noninvasive blood pressure measurement can grossly under- or overestimate BP and can be even unmeasurable in those with marked hypotension/hypovolemia.^{31,32} Use of invasive arterial blood pressure (ABP) assessment is considered more accurate and can even be used to assess hemodynamics. Considering the risk of vasopressors, including peripheral ischemia, arrhythmia, or extravasations, it is prudent to limit the total vasopressor exposure by using the most accurate measurement possible. Femoral and radial sites are the common placements for ABP catheters, and the readings are essentially interchangeable.³³ Consensus recommendations for the management of vasopressor-dependent patients support the use of invasive ABP monitoring.³⁴

Proposed Endpoints

Published data support the use of objective parameters both to recognize those who may need resuscitation as well as to guide the care itself. Useful endpoints are ones that not only provide information for the clinician to risk stratify and predict severity of illness, morbidity, and mortality but also are reliable and easily obtainable. In a large review of the literature, Tisherman et al evaluated the use of many different modalities and no single modality showed clear superiority over another, although the authors did recommend using something in order to guide resuscitation.³⁵ The "something" may be a metabolic parameter such as lactate or base deficit or targeting a specific component of oxygen delivery. Most studies agree that earlier timing of the resuscitation is crucial to impact morbidity and mortality.^{12,23,36}

Figure 3: Frank-Starling Curve



Ultrasound

The use of bedside ultrasound has grown to broader uses. The Focused Assessment with Sonography for Trauma (FAST) and Extended-Fast (E-FAST) exams allow the clinician to identify injuries in the blunt trauma patient that may require intervention.^{37,38} In recent years, protocols have been suggested to include ultrasound in the rapid assessment of undifferentiated critically ill patients. The Undifferentiated Hypotensive Patient (UHP) protocol was described in 2001.

The Rapid Ultrasound in Shock (RUSH) exam takes a three-faceted approach to evaluate cardiac function ("pump"), intravascular volume ("tank"), and large vessels ("pipes").³⁹ To evaluate the "pump," the authors recommend assessing the contractility of the heart as well as size of chambers or presence of pericardial fluid or ventricular thrombus. To evaluate the "tank," they recommend assessing the jugular veins and vena cava, as well as for presence of pleural or peritoneal fluid and pneumothorax. To evaluate the "pipes," they recommend assessment of the aorta and for venous thrombosis in the proximal lower extremities.

In a similar but broader and more comprehensive fashion, the World Interactive Network Focused On Critical Ultrasound (WINFOCUS) group also outlines how ultrasound

Table 3: Core Emergency Ultrasound Applications

- Trauma
- Intrauterine pregnancy
- Abdominal aortic aneurysm
- Cardiac
- Urinary
- Biliary
- Deep venous thrombosis
- Soft tissue/
musculoskeletal
- Ocular
- Thoracic
- Procedure guidance

use may be incorporated into an "ABCDE" model of assessment and resuscitation to both evaluate and treat the critically ill patient. This "ABCDE" model allows the user to evaluate the patient as it relates to airway (neck, trachea, airway), breathing (lung, pleura, etc.), circulation (cardiac, vascular, soft tissue), disability (central nervous system, optic nerve), and exposure (exclude missed findings or other life-threatening lesions).⁴⁰

While there is no evidence thus far to say that any resuscitative ultrasound protocol in and of itself leads to improved outcomes, the utility of the individual components of the exam have sufficient utility that the American College of Emergency Physicians (ACEP) has incorporated them into its most recent ultrasound guidelines.⁴¹ (See Table 3.)

Table 4: Dosage and Receptor Activity of Commonly Used Vasopressor and Inotropic Agents

Agent	Dose Range	α 1	β 1	β 2	DA
Phenylephrine	0.5 to 9.0 mcg/kg/min	+++++	-		-
Norepinephrine	0.01 to 3.0 mcg/kg/min	+++++	+++	++	-
Epinephrine	0.01 to 0.10 mcg/kg/min	+++++	++++	+++	-
Dopamine	2.5 to 20.0 mcg/kg/min	+++	++++	++	+++++
Dobutamine	2.5 to 20.0 mcg/kg/min	+	+++++	+++	-
Isoproterenol	2.0 to 10.0 mcg/min	-	+++++	+++++	-

Vasoactive Medication

Choosing the appropriate vasopressor is an important decision in the care of the critically ill and is mainly based upon understanding of vasopressor receptor physiology (*see Table 4*) and the patient's hemodynamic status. In general, vasopressors can all lead to unwanted ischemia, arrhythmias, and other complications; therefore, physicians should aggressively titrate these medications only to maintain an acceptable MAP, generally accepted to be > 65 mmHg, as there is no evidence to support targeting higher MAPs than this.⁴²

There is little evidence to guide the selection of a vasopressor agent with respect to specific shock states. Overall, either dopamine or norepinephrine should remain the initial choice for septic shock or undifferentiated shock. A common clinical practice often applied is based on the patient's heart rate: if the HR > 130 bpm, then use norepinephrine, and if the HR < 130, start dopamine.

Despite this, in the United States dopamine remains the workhorse vasopressor in many facilities for a number of reasons. This is partly related to the fact that it is available in a stable premixed form immediately upon request. It is a physiologic precursor to norepinephrine, but also can act at dopaminergic receptor sites providing vasopressor support as well as inotropic support. Traditional teaching dictates that, at lower doses, dopaminergic activity predominates, and at doses approaching 20 mcg/kg/min, it is alpha-receptor activity with beta-receptor activity primarily in the moderate range dosing. "Renal

dose" dopamine as a protective agent has not been supported in subsequent trials and is no longer recommended.^{34,43}

A recent trial comparing dopamine with norepinephrine in a heterogeneous population of shock found more arrhythmic events among the patients treated with dopamine than among those treated with norepinephrine (24.1% vs. 12.4%, $P < 0.001$).⁴⁴ Although there was no overall survival benefit, a subgroup analysis showed that dopamine was associated with an increased rate of death at 28 days among patients with cardiogenic shock ($P = 0.03$).

These results suggest that patients in cardiogenic shock may fare better with agents that have less beta-receptor activity. Other than cases of bradycardia, where inotropic or chronotropic activity is desired, a pure alpha-antagonist such as neosynephrine or phenylephrine should be considered, as it can augment afterload without markedly increasing myocardial oxygen demand.

In anaphylactic shock, epinephrine is the consensus recommended first-line agent, although rigorous data are lacking. Neurogenic shock is due to cervical spine injury that leads to a paucity of sympathetic output. The patient will have warm extremities (vasodilation from lack of sympathetic tone, hence, "warm shock"), bradycardia, and hypotension. Dopamine, with its balanced receptor activity, is the best selection in patients with neurogenic shock. A pitfall in trauma resuscitation, however, is overlooking a coexisting hemorrhagic shock component that may be present.

Endpoints of Resuscitation

Traditional Endpoints of Resuscitation. Parameters such as urine output, mental status, and vital signs have long been used to demonstrate adequacy of resuscitation; however, this approach may be inadequate either due to intrinsic disease, delay in onset, and confounders of measurement.^{45,46} Up to 85% of severely injured trauma victims may still be in shock despite normal vital signs.^{47,48}

Tachycardia has not been shown to be a sensitive indicator of dehydration⁴⁹; likewise, neither hypotension nor the presence of orthostatic vital signs are adequately sensitive or specific enough to identify patients in septic shock.⁴² Even resuscitating patients to normal vital signs and urine output can still result in a patient with persistent hypoperfusion.^{26,50} This can be problematic in not only identifying the at-risk, or "cryptic" shock, patient, but also in making decisions as to when the patient is fully resuscitated. Cryptic shock refers to a patient with biochemical evidence of hypoperfusion (i.e., elevated lactic acid > 4.0 mmol/L) despite having vital signs within normal parameters.

The shock index (SI) is a simple parameter derived from traditional vital signs (heart rate (HR) / systolic blood pressure (SBP)). It has been reported to be a more sensitive indicator than traditional vital signs alone, and an SI of > 0.9 is indicative of potential critical illness.⁵¹ A recent review of data from a large trauma registry (1,166 patients) found that patients with SI > 0.9

had a mortality more than twice that of those with an SI < 0.9.⁵² Other recent studies in trauma (SI > 0.9)⁵³ and pneumonia (SI > 1.0)⁵⁴ patients corroborate that SI can be specific for identifying critical illness. Unfortunately, SI is not sensitive enough that a normal value (< 0.9) can reliably exclude significant morbidity or mortality.

Modern Endpoints of Resuscitation. Given the limitations of these traditional markers both on recognition of shock and as an endpoint of resuscitation, it is clear that better indices are needed. Markers or endpoints of resuscitation can be categorized in multiple ways. Pieracci differentiates between “upstream” and “downstream” markers of perfusion,⁵⁵ while Tisherman describes the need for two distinct categories: global (“whole body”) and regional (“single organ,” e.g., the brain).³⁵

Upstream markers measure adequacy of DO₂ and its components, which include cardiac output, arterial hemoglobin concentration, and hemoglobin saturation. Downstream markers, conversely, measure the balance between supply (DO₂) and demand (VO₂). In a normal physiologic state, delivery far exceeds consumption. However, in times of hypoperfusion, it is possible to become supply-dependent such that VO₂ exceeds DO₂, resulting in maximization of oxygen extraction ratio (OER), decreasing central venous oxygenation saturation (ScvO₂), and elevated lactate or base deficit.

For the EP, ScvO₂ is the most relevant of these markers. When mismatch occurs and oxygen debt develops, the body begins to increase the amount of oxygen it extracts (normally 20-25% of DO₂ by volume) via increasing the OER. As OER increases, the saturation of the blood returning to the central vessels, where the ScvO₂ is measured, falls. Thus, as DO₂/VO₂ mismatch and oxygen debt worsens, the OER goes up and ScvO₂ falls. When ScvO₂ (which is normally 70-75%) drops, it suggests that either the DO₂ is inadequate or that VO₂ has dramatically increased.

Serum Lactate Measurement

Serum lactate has been widely recommended as a marker for critical illness and a guide for resuscitation. Lactate is an important early biomarker for tissue hypoxia that can be used to monitor therapy in the early presentation of critical illness.^{16,56,57}

Shock states lead to oxygen debt, resulting in hypoxia at the intracellular level. Anaerobic glycolytic pathways for ATP production take over when mitochondrial oxidative phosphorylation fails due to hypoxia or a DO₂/VO₂ mismatch, yielding increased intracellular lactate. Prolonged tissue hypoxia results in lactate diffusion into the bloodstream in concentrations proportional to the duration and severity of the tissue oxygen debt. Both initial serum lactate elevations and persistent elevations (> 24 hours) are associated with increased mortality and ICU admissions.^{57,58}

Lactate is cleared by hepatic and renal metabolism, and decreasing serum levels indicate restoration of tissue oxygen delivery. Serial measures of lactate clearance over time are also predictors of organ failure and mortality.⁵⁷⁻⁶¹ A suggested goal of resuscitation is to resuscitate until the serum lactate is normal or < 2.0 mmol/L.⁵⁹

Noninvasive Positive Pressure Ventilation

Despite the fact that endotracheal intubation (ETI) is often necessary in critically ill patients, it is not necessarily a benign procedure. In appropriate cases, utilization of non-invasive positive pressure ventilation (NPPV) in lieu of ETI can avoid immediate and delayed complications of intubation, including trauma, aspiration, hypoxia, failed airway, and ventilator-associated pneumonia (VAP) or other complications. Each episode of VAP carries a mortality rate of up to 50% and an increase of inpatient cost of \$28,000 per patient.⁶⁰ Furthermore, NPPV allows the patients to communicate, speak, or eat when the mask is removed and mobilize their secretions more effectively.

There are many patients in whom

Table 5: Contraindications for the Use of NPPV

- Severe head, face, or neck trauma or deformity
- Cardiac or respiratory arrest or near-arrest
- Severe encephalopathy or GCS < 10
- Significant upper gastrointestinal bleeds or emesis
- Inability to handle secretions or maintain airway
- Upper airway stridor or obstruction

NPPV is contraindicated or should be used with caution. (*See Table 5.*) Delivery is via a tight-fitting mask that is either full-face or nasal. NPPV can provide the patient with end expiratory airway pressure (EPAP) alone or EPAP plus inspiratory positive airway pressure (IPAP). The former is generally referred to as continuous positive pressure (CPAP) and the latter as bilevel positive airway pressure (BiPAP).

The primary function of EPAP is to recruit alveoli and augment the total surface area for gas exchange to improve hypoxemia. This increases the functional residual capacity of the patient and mitigates work of breathing. Increases in IPAP increase pulmonary ventilation, giving additional support. For patients whose primary problem is hypoxia, more EPAP is needed. When patients have increasing levels of PaCO₂, more IPAP is needed to help improve alveolar ventilation.

There is no uniform method to initiate NPPV, and it often requires a trained respiratory therapist (RT) several minutes to work with and reassure an anxious patient to successfully initiate NPPV. The goals, however, are to improve the work of breathing (WOB) and maintain oxygen saturations > 90%. Patients, however, should quickly report a subjective improvement in dyspnea within a few minutes of initiation. The patient is always the best

Table 6: Recommended Initial Ventilator Settings for Various Patients

	FiO ₂	TV	RR	I:E	PEEP
General	1.0	6-8 mL/kg	12-14	1:2	5 cm H ₂ O
COPD/ Asthma	1.0	6-8 mL/kg	8-12	1:3	5-10 cm H ₂ O
Trauma	1.0	6-8 mL/kg	12-14	1:2	0-5 cm H ₂ O
ARDS	1.0	6-8 mL/kg	12-14	1:2	5-12 cm H ₂ O

barometer of improvement or deterioration in respiratory failure.

If one elects to use CPAP (EPAP only), the pressure can be set from 8-12 cm H₂O initially and increased as needed to improve oxygenation and decrease WOB. BiPAP can be initially set with settings (IPAP/EPAP) of 6/3 cm H₂O, but can be set at any level per the patient's respiratory requirements. The settings can be changed as needed to address hypoxia (EPAP) or hypoventilation (IPAP) by titrating by 2-3 cm H₂O at a time. Each change should be allowed 20-30 minutes to take full effect.

It is not uncommon for patients to remain anxious and uncomfortable during the use of NPPV. Mild sedation with small doses of a benzodiazepine and anxiolysis is appropriate. The most common complications are pressure sores related to the mask application (10%) and aspiration pneumonia (5%).^{62,63} Patients who require > 20 cm H₂O are at high risk for barotrauma and should be considered for ETI. Any patient with objective evidence of ischemia from cardiac (STEMI), neurologic (stroke), or shock (lactic acidosis or vasopressor-dependent), in addition to respiratory embarrassment, would benefit more from ETI instead of NPPV to minimize the VO₂ utilized by respiratory effort.

In a recent systematic review, Keenan et al reported that in acute hypoxemic respiratory failure, NPPV reduced the need for ETI by 23% (95% CI, 10-35%), reduced ICU length of stay by 2 days (95% CI, 1-3) and reduced ICU mortality by 17% (95% CI, 8-26%).⁶⁴

The Intubated Patient in the ED

Specific clinical indications for instituting mechanical ventilation are numerous, but sometimes may be distilled down to simple EP gestalt that, "This patient is not going to do very well on their own." Working knowledge of ventilator-patient interaction and an appreciation of the patient's underlying disease state relating to the need for mechanical ventilation (i.e., trauma vs. medical, etc.) is vital to appropriate selection of initial ventilator settings and ongoing monitoring of the patient's status.

Once the patient has been successfully intubated and tube placement confirmed, the physician will need to choose a ventilator strategy. This is primarily guided by host and disease parameters. (See Table 6.) The majority of patients receiving mechanical ventilation in the ED will be started on volume-cycled (VC) ventilation (as opposed to pressure-cycled [PC] ventilation), which simply means that a tidal volume is selected and the ventilator measures the pressures that are generated. With PC ventilation, which is utilized in patients with poorly compliant lungs often due to acute respiratory distress syndrome (ARDS), an airway pressure is adjusted to generate an appropriate tidal volume.

For patients spontaneously breathing, pressure support ventilation (PSV) can provide respiratory flow rate and inspiratory pressure in addition to supplemental oxygen. In PSV the only settings selected are the inspiratory pressure and FiO₂. The tidal volume is determined by the patient's lung compliance. PSV

is generally not the optimal option in critically ill patients. It is a good option for undifferentiated dyspneic patients with a pre-existing tracheostomy. For the remaining patients, either assist control (AC) or synchronized intermittent mandatory ventilation (SIMV) will be selected. In AC, the ventilator delivers a prescribed number of fully supported breaths either patient- or ventilator-triggered. The SIMV mode is better synchronized with the patient's respiratory cycle and allows the patient to spontaneously produce unsupported breaths if breathing above the ventilator. In a comatose patient without spontaneous respirations, both modes will have the exact effect. With patients who are spontaneously breathing, AC mode may lead to dangerous hyperventilation or ventilator dyssynchrony if the patient is not properly sedated.

The clinician can manipulate the minute ventilation (L/minute; respiratory rate and tidal volume), the pressure gradient (FiO₂), and the surface area for gas exchange with positive end-expiratory pressure (PEEP). The initial tidal volume should be 6-8 cc/kg and should be based on the patient's ideal body weight (IBW). The initial fraction of inspired oxygen (FiO₂) is generally set at 1.0, but this is not established convention and the clinician may initiate the FiO₂ at any appropriate level. The FiO₂ can be weaned by pulse oximetry to maintain adequate oxygenation > 93% and the PaO₂ on the ABG > 60 mmHg. Many pragmatic practitioners wean the FiO₂ by pulse oximetry to avoid unnecessary ABGs. Otherwise, adjustments should be made based on ABG analysis performed within 20-30 minutes of initiation or ventilator adjustment.

PEEP is used to prevent alveolar collapse and maintain functional residual volume (FRC). Unfortunately, PEEP can also lead to hypotension (due to increasing intrathoracic pressure and decreased venous return), increased intracranial pressure, and heterogeneous lung processes (that expose areas of the lung to different pressures due

to varying compliance throughout the parenchyma), and should be employed with caution and appreciation of these effects.

Proximal Airway Pressures (PAP). Physicians who place patients on mechanical ventilation should have an intimate understanding of proximal airway pressures and their management. Airway pressures should be reviewed as a vital sign for all patients on mechanical ventilation. The two airway pressure measurements important to monitor are peak (P_{peak}) and plateau (P_{plat}) airway pressures. These reflect resistance to flow in the airways, and lung and chest wall compliance, respectively. Normal airway pressures are less than 15-20 cm H_2O .

Peak airway pressures represent the highest pressure present in the proximal airways, which occurs during airflow. This can be determined on the ventilator monitor as an ongoing measurement. Anything that increases resistance (R_a) to airflow increases P_{peak} . Clinically, this is limited to three primary causes: bronchospasm, airway secretions, and the patient biting the endotracheal tube. Plateau airway pressures are a reflection of lung and chest wall compliance, the change in volume per a change in pressure ($C = \Delta V / \Delta P$). Increased P_{plat} suggests significant decrease in compliance. Conditions that lead to worsening compliance include pulmonary disorders (pneumonia, ARDS, pulmonary edema, and pneumothorax) and chest wall conditions (morbid obesity, kyphoscoliosis, thoracic surgery, and burns to the chest causing an eschar). At the very least, the workup for increased P_{plat} should include reassessment of the patient, chest X-ray, and an ABG. Troubleshooting an intubated patient should always include an assessment of PAP.

Advanced Settings

Pressure Cycled Ventilation. When a patient's plateau pressures begin to increase beyond 30 cm H_2O , it is important to consider changing ventilation strategy to avoid sequelae of barotraumas. As

previously mentioned, PC ventilation involves setting the airway pressure as the target parameter. The tidal volume that results from the setting requires close monitoring and adjustment upon initiation to ensure appropriate ventilation. As with VC ventilation, the ventilator needs to be selected for AC vs. SIMV, PEEP, FiO_2 and I:E ratios. Patients who require PC ventilation have significantly decreased lung compliance due to their disease process, and further deterioration in compliance will lead to hypoventilation unless the ventilator settings are modified in response to this. Choosing an alternative strategy should include discussions with an intensivist and a respiratory therapist.

BiLevel/APRV. Alternatives to PC in patients with increased plateau pressures include BiLevel Ventilation. High and low pressures are selected on the ventilator, as well as a time at each pressure; the low pressure is also referred to as the release phase. Low pressure times vary from 0.2-1.5 seconds, at the higher end for bronchospasm and at the lower end for restrictive lung diseases. The general I:E ratios are in the order of 8-9:1. The advantage of BiLevel (also known as BIPAP or Bilevel CPAP) and APRV is that they allow the patient to breath spontaneously through the cycle. Other methods of ventilation utilized on such patients, such as inverse ratio ventilation (IRV), generally require extended neuromuscular paralysis and heavy sedation.

Post-intubation Care. Adequate post-intubation care can avoid unnecessary in-hospital morbidity and mortality. The Institute for Healthcare Improvement (IHI) endorses a bundle that includes head-of-bed (HOB) elevated 30-45°, oral care, prophylaxis for both peptic ulcer disease and deep venous thrombosis, daily vacation from sedation, and regular assessment for extubation.⁶⁵⁻⁶⁷

Bonomo et al reported a concerning deficiency with respect to appropriate sedation and analgesia provided to 117 intubated

ED patients at a large tertiary care center. In patients who remained in the ED for at least 30 minutes after intubation, 39 patients received no anxiolysis (33%), 62 received no analgesia (53%), and 23 patients received neither anxiolytic nor analgesic (20%). Of 70 patients given a paralytic post-intubation, 67 received either no or inadequate anxiolysis or analgesia (96%).⁶⁸ There are no evidence-based recommendations. Appropriate sedation and analgesia should be provided for patients requiring mechanical ventilation. Additionally, it is recommended that patients be assessed for pain and anxiety and response to therapy in a timely manner.⁶⁹

Auto-PEEP (Auto-Positive-End-Expiratory Pressure). When a patient is unable to completely exhale due to airway resistance or insufficient expiratory time, residual volume of alveolar gas collects. This can occur with spontaneously breathing patients with severe bronchospasm or in patients on mechanical ventilation. This excess gas is referred to as auto-PEEP, also known as dynamic hyperinflation or intrinsic-PEEP (iPEEP). Auto-PEEP can be assessed at the ventilator with an expiratory hold maneuver. Increasing levels of auto-PEEP lead to dangerous levels of intra-thoracic pressure, decreasing venous return (and cardiac output), barotrauma, and patient distress. Factors that lead to suspicion of the development of auto-PEEP include unexplained patient agitation, tachypnea or tachycardia, inability to trigger the ventilator, or when inspiratory flow begins prior to completion of expiration on the ventilator display.

As auto-PEEP increases intrathoracic volume, the patients breathe with muscles stretched to a very inefficient state. Normally, it requires 2-3 cm H_2O of negative inspiratory force (NIF) to initiate a breath. When 10 cm H_2O of auto-PEEP develops, a patient now has to generate 12-13 cm H_2O of NIF to initiate a breath or trigger the ventilator. The auto-PEEP forces a patient to work 4-5 times harder with respiratory

muscles at a significant mechanical disadvantage.

To minimize auto-PEEP, the goal is essentially to minimize minute ventilation. Increasing the inspiration:expiration (I:E) ratio to 1:3 or 1:4 will maximize gas emptying. In patients with severe bronchospasm, a permissive hypercapnia strategy can be employed. In permissive hypercapnia, PCO₂ levels are allowed to rise to 75-80 mmHg with pH levels approximately 7.20-7.25, to mitigate the development of auto-PEEP or increasing airway pressures. Finally, the addition of extrinsic PEEP (ventilator-generated PEEP) to about 85% of measured auto-PEEP will reestablish the normal gradient of 2-3 cm H₂O typically required to initiate a breath. Patients requiring any of the above techniques also require deep sedation and often paralysis for comfort and to avoid ventilator dyssynchrony.

Cardiac Arrest

In-hospital cardiac arrests occur at a rate of 3-6/1000 admissions.¹³ Survival rates vary widely region to region (4% to 40%), but generally are associated with age, time to advanced life support, and presenting rhythm, among other factors.⁷⁰ Successful resuscitation of the cardiac arrest victim includes:

- Early CPR with emphasis on chest compressions;
- Rapid defibrillation;
- Effective advanced life support;
- Integrated post-cardiac arrest care.

In-hospital CPR is often described as being too slow, too shallow, or having ventilation rates far outside recommended guidelines.⁷¹ Excessive ventilation rates (> 10-12 breaths/minute) can increase intrathoracic pressures, causing a deleterious effect on coronary perfusion pressures and diminished rates of return of spontaneous circulation (ROSC).⁷² Suboptimal compression depths and not allowing for proper chest decompressions are also both associated with poorer rates of ROSC.⁷³ Minimizing off-chest time is essential, as several cycles are needed to

build up cardiac output after even brief interruptions in CPR.⁷⁴ Current guidelines for adult CPR recommend a chest compression rate of at least 100/minute at a depth of at least 5 cm, allowing full chest recoil between compressions and ventilation rates of no more than 8-10/minute.¹³ Pauses in CPR for any reason should be as brief as possible, ideally not to exceed 10 seconds.⁷³

The most important advancement in post-cardiac arrest care has been the introduction of induced hypothermia.^{75,76} Induced hypothermia can decrease the cerebral metabolic rate by approximately 6% per every 1°C, thus limiting oxygen utilization and potentially catastrophic neurologic injury. The current recommendation is to initiate mild therapeutic hypothermia (32-34°C) in all adult comatose survivors after a VF and pulseless ventricular tachycardia arrest.⁷⁷ The original trials did not include pulseless electric activity or asystolic patients, but the pathology of a global ischemic insult followed by a reperfusion injury is the same; therefore, induced hypothermia is recommended for these patients with a lower level of evidence. The goal is to reach target temperature within 4-8 hours of ROSC, remain at target induced hypothermia for 12-24 hours, followed by controlled rewarming. Appropriate monitoring of temperature, electrolytes, cardiac rhythm, and sedation levels are required for the duration of induced hypothermia and controlled rewarming, and generally requires an established multidisciplinary protocol for successful adoption.

Oxygen toxicity is another real threat to post-cardiac arrest survivors.⁷⁸ Excess oxygen can “feed the ischemia-reperfusion” fire in which reperfused oxygenated blood leads to lipid peroxidation and results in a chain reaction converting unsaturated lipids to lipid peroxides, cell death, and neurologic demise. The recommendation is to maintain oxygen saturations > 94% and to minimize FiO₂. Finally, a significant number of patients with ROSC are found to have significant

Table 7: Systemic Inflammatory Response Syndrome (SIRS) Criteria

- Temperature ≤ 36° C or ≥ 38° C
- Pulse ≥ 90 beats/minute
- Respiratory rate ≥ 20 breaths/minute
- PaCO₂ < 32 mmHg
- WBC ≤ 4,000 or ≥ 12,000 cells/mm³ or > 10% band forms

CAD amenable to intervention. Availability of coronary catheterization for appropriate patients is a valuable resource to improve patient outcomes.⁷⁹

Severe Sepsis and Septic Shock

In the United States alone, sepsis claims approximately 210,000 lives of an estimated 750,000 reported cases annually and its incidence is rising.^{80,81} Half of these patients are admitted to an ICU where sepsis remains the leading cause of death.^{80,82}

Management of severe sepsis and septic shock has evolved in the past several years. Despite these medical advances, sepsis mortality remains staggering.⁸³ In direct response to this, 11 international organizations spanning critical care, surgery, nursing, infectious disease, and emergency medicine united in October 2002. The campaign, now widely known as the Surviving Sepsis Campaign (SSC), has recently published revised guidelines³⁴ based on modified Delphi methodology.³⁴ These guidelines represent an exhaustive review of current literature and a summation of accrued understanding regarding sepsis.

The most important step in managing sepsis patients is identification. The presumptive diagnosis of sepsis requires the presence or suspicion of infection in addition to two systemic inflammatory response syndrome (SIRS) criteria.⁸² (See Table 7.) Serum lactate measurement serves three distinct purposes: as a marker

of disease severity; as a trigger for an aggressive resuscitation protocol; and as an endpoint of resuscitation. To encourage lactate measurements, the practice at some institutions is to draw a lactate on any patients getting blood cultures. This can be a reasonable surrogate for sepsis, as an order for blood cultures suggests: 1) there is a “suspicion of infection”; and 2) the patient is likely being admitted to the hospital.

The SSC guidelines recommend that sepsis patients with serum lactate > 4.0 mmol/L receive early goal-directed therapy (EGDT, see below).³⁴ We also encourage that all patients with lactate elevations, even mild, get resuscitated and repeat lactate measurements drawn to assure normalization of lactate levels to < 2 mmol/L or at least a 10% clearance.⁵⁸ A common clinical practice is the “rule of 2s”: after 2 liters (approximately 30 cc/kg in the average 70-kg person) or 2 hours (the half-life of lactate is 15-20 minutes), obtain the second lactate measurement.

The SSC’s recommendations include³⁴:

Initial Resuscitation

- Immediate resuscitation (EGDT) in patients with hypotension or serum lactate > 4 mmol/L; this should not be delayed until ICU arrival.

- For patients receiving EGDT, target the following resuscitation goals: CVP 8-12 mmHg, MAP \geq 65 mmHg, urine output \geq 0.5 mL/kg/hr, ScvO₂ \geq 70%. If ScvO₂ remains < 70%, consider further fluid resuscitation, transfuse packed red cells if required to hematocrit \geq 30%, or consider dobutamine infusion.

Diagnosis

- Obtain blood cultures (2 or more) before initiating antibiotics.
- Obtain radiography or sampling of any site of suspected infection.

Antibiotic Therapy

- Begin broad-spectrum IV antibiotics as early as possible, ideally within 1 hour of recognition of either severe sepsis or septic shock.

Source Control

- Identify the site of infection as soon as possible.

- Institute source control measures if possible (drainage, debridement).

- Remove infected intravascular devices as soon as possible.

Two recent studies have provided strong evidence illustrating that expeditious delivery of antibiotics is key to improving outcomes in sepsis patients.^{18,19} Kumar et al reported that for every 1 hour delay in antimicrobial administration over the initial 6 hours was associated with a decline in survival of 7.6%. In multivariate analysis, time to antibiotics was the strongest predictor of outcome. This evidence was corroborated by Gaieski et al, who showed that septic shock patients who received antibiotics within 1 hour had lower in-hospital mortality than patients who received antibiotics > 1 hour from triage (mortality 19.5 vs. 33.2%; odds ratio, 0.30 [95% confidence interval, 0.11-0.83]; *p* = .02).

EGDT is one of the most integral and controversial components of the SSC. The original trial by Rivers et al that included 263 patients with sepsis who had either an elevated lactate (> 4.0 mmol/L) or a systolic blood pressure < 90 mmHg (despite at least 20-30 cc/kg of crystalloid infusion) remains the only randomized controlled trial in support of EGDT.¹² The authors reported a 28-day mortality absolute risk reduction of 15.9% (*P* = 0.01). Fewer patients in the treatment group received mechanical ventilation (*P* = 0.02), vasopressor therapy (*P* = 0.02), and pulmonary artery catheterization (*P* = 0.01).

There are several corroborative studies evaluating the EGDT protocol solely or as a component of a larger sepsis intervention.^{12,84-91} Critics of the original protocol have highlighted the absence of corroborating evidence, the single-center nature of the study, and the lack of data to support any individual component of the trial.⁹²⁻⁹⁶ Ultimately, the protocol makes physiologic sense, as it addresses all of the components of oxygen delivery such as preload (central venous pressure), afterload (mean arterial pressure), arterial oxygen content

(hemoglobin), and contractility (ScvO₂).

In a recent multicenter, non-inferiority trial, Jones et al compared two alternate goal-directed protocols to determine the utility of lactate clearance targets vs. ScvO₂ as endpoints of resuscitation in severe sepsis and septic shock. Patients were resuscitated to either a lactate clearance of at least 10% from baseline or an ScvO₂ of 70% or greater. The data showed that lactate clearance was as good of a measure of short-term sepsis survival, if not better, than ScvO₂. The authors concluded that the “substitution of lactate measurements in peripheral venous blood as a safe and efficacious alternative to a computerized spectrophotometric catheter in the resuscitation of sepsis” (i.e., lactate clearance may be used as a resuscitation endpoint in lieu of ScvO₂).⁵⁹

Iatrogenic Infections

Hospital-acquired infections have caught the attention of professional societies, quality improvement organizations, patient-safety associations, health-insurance companies, and health care regulators at both the state and federal level. While the details vary from study to study, a common theme is that hospital-acquired infections are related to significant increases in health care costs, morbidity, and mortality. Therefore, one of the most effective ways that EPs can impact patient outcomes is in the prevention of hospital-acquired infections through: appropriate hand hygiene, maximal use of maximal sterile barriers, appropriate ventilator hygiene, and judicious use of catheter insertions.

It is estimated that up to 33% of all hospital-acquired infections are preventable through appropriate measures, and ED care for critically ill patients provides ample opportunity for prevention. Simple interventions such as the use of sterile barriers and regular hand hygiene are effective measures. Guidelines are available for the prevention of both intravascular and urinary catheter-related infections from the CDC. In a landmark

study, Pronovost et al reported that implementation of a simple checklist for central-line insertions decreased the rate of central line-associated bloodstream infections from 7.7 to 1.4 per 1000 catheter days in the first 18 months.⁹⁷ There are clearly significant incentives and opportunities for EPs to prevent hospital-acquired infections.

Conclusion

The success of each specialty and each location that provides care to the critically ill patient is highly dependent on the presenting status of the patient. As noted by Peter Safar, credited as the father of intensive care medicine, "The most sophisticated intensive care often becomes unnecessarily expensive terminal care when the pre-ICU system fails."⁹⁸ The ED will often provide the early initial care that maximizes the chance for full and complete recovery. The principles of early hemodynamic monitoring, ventilatory support, goal-directed resuscitation, and early antibiotic therapy are crucial to ED care of the critically ill patient.

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 23. The reliability of CVP monitoring to predict fluid responsiveness can be affected by:
 - A. high fever
 - B. patient age
 - C. anemia
 - D. presence of cardiomyopathy
 24. The Rapid Ultrasound in Shock exam involves:
 - A. ocular ultrasound to estimate intracranial pressure
 - B. assessing for cardiac function and chamber size
 - C. determining the presence of an intrauterine pregnancy
 - D. measuring the superior vena cava as a measure of fluid status
 25. For patients who are vasopressor-dependent:
 - A. The target MAP should be 85 mm Hg.
 - B. Oscillometric measurement is preferable over invasive measurements.
 - C. Radial artery ABP measurements are more accurate than femoral.
 - D. Renal-dose dopamine is protective in patients with shock.
 - E. Patients in cardiogenic shock should receive norepinephrine, not dopamine, as the initial agent of choice.
 26. The best marker to identify a critically ill patient is:
 - A. systolic blood pressure
 - B. heart rate
 - C. serum lactate
 - D. orthostatic vital signs
 27. Which of the following is true in states of hypoperfusion and shock?
 - A. DO_2 far exceeds VO_2 .
 - B. OER and serum lactate are very low.
 - C. ScVO_2 decreases below 70%.
 - D. Abnormal VO_2 is easily and quickly normalized with IV fluid.
 28. Which of the following patients is a good candidate for NPPV?
 - A. a 45-year-old man with agonal respirations and multiple severe facial injuries

Physician CME Questions

21. Which of the following is a component of oxygen delivery?
 - A. arterial oxygen content
 - B. pulse pressure
 - C. respiratory rate
 - D. venous oxygen saturation
22. Which of the following can improve oxygen consumption?
 - A. adding vasopressors to increase mean arterial blood pressure
 - B. using inotropes to augment myocardial contractility
 - C. anti-pyretics for fever
 - D. spontaneous respirations for a dyspneic patient in septic shock.

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

- B. a 90-year-old woman with severe upper airway stridor and confusion
 - C. an 18-year-old woman with progressive tongue and upper airway edema and developing anaphylactic shock
 - D. a 32-year-old man with peri-oral burns, hoarseness of voice, and severe acidosis following a house fire
 - E. an 83-year-old woman with hypoxia and respiratory acidosis (pH 7.25, PaO₂ 72, PaCO₂ 90) secondary to a CHF exacerbation.
29. Following 6 hours of resuscitation, which of the following targets satisfies the SSC guidelines regarding the initial resuscitation of septic shock patients?
- A. CVP 12 mm Hg, MAP 70 mm Hg, urine output of 1.0 cc/kg/hr, ScvO₂ 80%
 - B. CVP 3 mm Hg, MAP 85 mm Hg, urine output of 0.6 cc/kg/hr, ScvO₂ 80%
 - C. CVP 18 mm Hg, MAP 50 mm Hg, urine output of 0.2 cc/kg/hr, ScvO₂ 70%
 - D. CVP 26 mm Hg, MAP 100 mm Hg, urine output of 0.0 cc/kg/hr, ScvO₂ 50%
 - E. CVP 14 mm Hg, MAP 100 mm Hg, urine output of 1.0 cc/kg/hr, ScvO₂ 40%
30. Which of the following patients were managed best according to the most recent recommendations for CPR?
- A. CPR: 90 compressions/min at 3 cm depth, ventilation rate of 20
 - B. CPR: 100 compressions/min at 5 cm depth, ventilation rate of 12
 - C. CPR: 60 compressions/min at 5 cm depth, ventilation rate of 20
 - D. CPR: 120 compressions/min at 3 cm depth, ventilation rate of 30

CME Instructions

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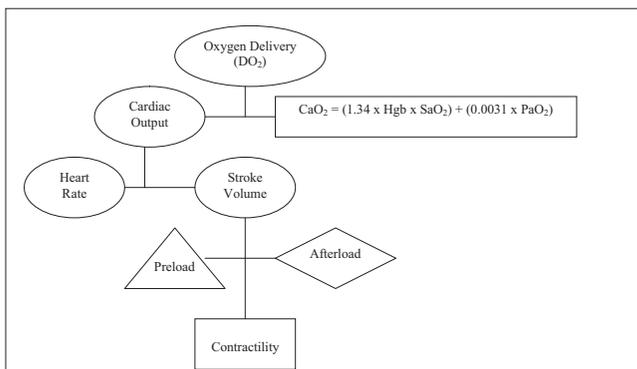
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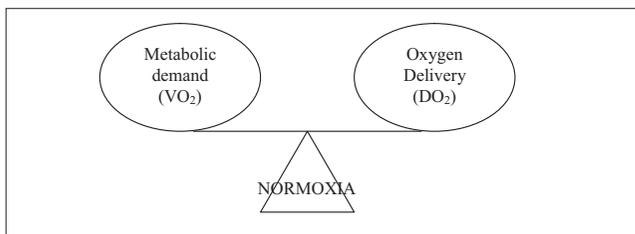
Caring for Critically Ill Patients in the ED

Flow Diagram of the Components of Oxygen Delivery

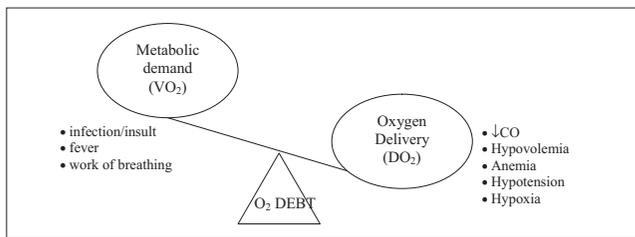


CaO₂, arterial oxygen content; Hgb, hemoglobin; SaO₂, saturation of hemoglobin; PaO₂, partial pressure of oxygen

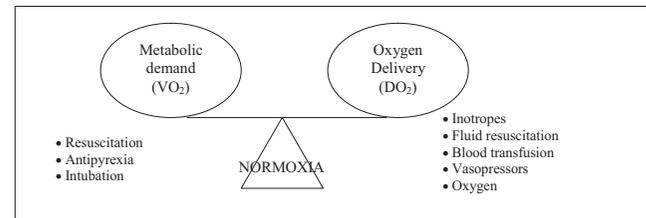
State of Consumption-Delivery Balance



Development of Shock and State of Consumption-Delivery Mismatch



Following Resuscitation Efforts, Return to a State of Consumption-Delivery Balance



Common Physical Exam and Hemodynamic Findings of Patients in Different Forms of Shock

	HR	Skin Exam	CO	CVP	SVR	Comments
Hypovolemic	↑	Variable	↓	↓	↑	Need to find source of volume loss
Cardiogenic	↓↑	Cool/diaphoretic	↓	↑	↑	"Pump failure" or rhythm disturbance
Distributive						
Septic (early)	↑	Variable	↑	↓	↓	Hyperdynamic state
Septic (late)	↑	Cool	↓	↑↓	↑	Hypodynamic state
Neurogenic	↓	Warm	↓	↓	↓	Survey for co-existent trauma
Anaphylaxis	↑	Variable	↓	↓	↓	Can develop bradycardia prior to arrest
Obstructive	↑	Cool	↓	↑	↑	CXR or bedside ultrasound can lead to quick diagnosis
Dissociative	↑	Warm	↓	↑↓	↓	Variable presentations

HR, heart rate; CO, cardiac output; CVP, central venous pressure; SVR, systemic vascular resistance

Recommended Initial Ventilator Settings for Various Patients

	FiO ₂	TV	RR	I:E	PEEP
General	1.0	6-8 mL/kg	12-14	1:2	5 cm H ₂ O
COPD/Asthma	1.0	6-8 mL/kg	8-12	1:3	5-10 cm H ₂ O
Trauma	1.0	6-8 mL/kg	12-14	1:2	0-5 cm H ₂ O
ARDS	1.0	6-8 mL/kg	12-14	1:2	5-12 cm H ₂ O

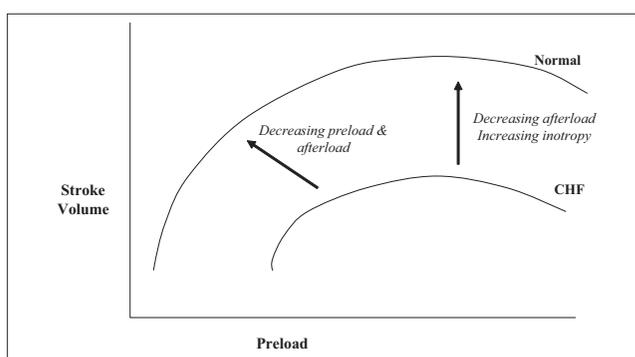
Dosage and Receptor Activity of Commonly Used Vasopressor and Inotropic Agents

Agent	Dose Range	α 1	β 1	β 2	DA
Phenylephrine	0.5 to 9.0 mcg/kg/min	+++++	-		-
Norepinephrine	0.01 to 3.0 mcg/kg/min	+++++	+++	++	-
Epinephrine	0.01 to 0.10 mcg/kg/min	+++++	++++	+++	-
Dopamine	2.5 to 20.0 mcg/kg/min	+++	++++	++	+++++
Dobutamine	2.5 to 20.0 mcg/kg/min	+	+++++	+++	-
Isoproterenol	2.0 to 10.0 mcg/min	-	+++++	+++++	-

Examples of Etiologies and Shock Subtypes

Hypovolemic <ul style="list-style-type: none"> Hemorrhage Serum/plasma loss Gastroenteritis 3rd spacing 	Cardiogenic <ul style="list-style-type: none"> Myocardial infarction (and complications) Dysrhythmia Congestive heart failure Congenital heart disease
Distributive <ul style="list-style-type: none"> Anaphylactic Neurogenic Septic 	Obstructive <ul style="list-style-type: none"> Tension pneumothorax Cardiac tamponade Aortic dissection Massive pulmonary embolism
Dissociative <ul style="list-style-type: none"> Heat stroke Cyanide Carbon monoxide 	

Frank-Starling Curve



Core Emergency Ultrasound Applications

- Trauma
- Intrauterine pregnancy
- Abdominal aortic aneurysm
- Cardiac
- Urinary
- Biliary
- Deep venous thrombosis
- Soft tissue/musculoskeletal
- Ocular
- Thoracic
- Procedure guidance

Contraindications for the Use of NPPV

- Severe head, face, or neck trauma or deformity
- Cardiac or respiratory arrest or near-arrest
- Severe encephalopathy or GCS < 10
- Significant upper gastrointestinal bleeds or emesis
- Inability to handle secretions or maintain airway
- Upper airway stridor or obstruction

Systemic Inflammatory Response Syndrome (SIRS) Criteria

- Temperature $\leq 36^{\circ}\text{C}$ or $\geq 38^{\circ}\text{C}$
- Pulse ≥ 90 beats/minute
- Respiratory rate ≥ 20 breaths/minute
- $\text{PaCO}_2 < 32$ mmHg
- WBC $\leq 4,000$ or $\geq 12,000$ cells/ mm^3 or $> 10\%$ band forms

Supplement to *Emergency Medicine Reports*, July 18, 2011: "Caring for Critically Ill Patients in the Emergency Department." Authors: **Robert Sherwin, MD, FACEP**, Assistant Professor, Department of Emergency Medicine, Wayne State University School of Medicine, Detroit, MI; **Audwin J. Garcia, MD**, Senior Staff Physician, Emergency Medicine and Critical Care Medicine, Henry Ford Health System, Assistant Clinical Professor, Wayne State University School of Medicine, Detroit, MI; **Meena Munshi, MD**, Emergency Medicine Resident, Wayne State University School of Medicine, Detroit Receiving Hospital, Detroit, MI.

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