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MODERN TRAUMA RESUSCITATION (AW KIRKPATRICK, SECTION EDITOR)

Monitoring and End-Points of Trauma Resuscitation

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Abstract
Purpose of Review Monitoring end-points of trauma resuscitation and understanding principles of hemostatic resuscitation have become important in the management of severely injured patients. The purpose of this review is to discuss a selected number of parameters used to guide resuscitation of trauma patients.
Recent Findings In trauma, shock occurs because of inadequate tissue perfusion and oxygen delivery. The goal of resuscitation is to restore adequate tissue perfusion while avoiding over-resuscitation and associated complications. Understanding of the dynamic cardiopulmonary interactions provides the physician with a tool to evaluate pulse pressure variation and stroke volume variation. Measurements of mixed and central venous oxygen saturation give an indication of systemic oxygen extraction. Bedside echocardiography has dramatically improved the diagnostic evaluation of patients in shock. The metabolic markers of perfusion are indicators of micro-circulation resuscitation and include characterization of oxygen extraction, lactate production, and base deficit. Characterization of regional oxygen delivery is an evolving field with new technological developments.

Despite the numerous measures used to characterize end-points of trauma resuscitation, there is no clear single measure used to guide optimal resuscitation. The physician must provide careful interrogation of hemodynamic, metabolic, and hemostatic parameters and their respective trends over time to establish optimal therapy. The use of thromboelastography to guide hemostatic resuscitation has the potential to optimize individual patient resuscitation thereby reducing unnecessary blood product utilization.
Summary Careful monitoring of end-points of trauma resuscitation and understanding of hemostatic resuscitation are needed to guide resuscitation of critically ill patients and improve outcomes.

Keywords End-points of resuscitation · Shock · Hemodynamic end-points · Metabolic end-points · Hemostatic resuscitation

This article is part of the Topical Collection on *Modern Trauma Resuscitation*

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Introduction

Trauma is the leading cause of death and disability worldwide under the age of 40. Patients with significant traumatic injury are at risk of hemorrhagic shock [1]. Uncontrollable hemorrhage is responsible for 30 to 40% of trauma mortality and accounts for almost 50% of deaths occurring in the initial 24 h following traumatic injury [2]. As such, in trauma patients with active hemorrhage, early recognition of the injury pattern and degree of physiologic derangement is essential. This should be followed by prompt execution of targeted resuscitation (resuscitation goals specific to your patient based on physical examination, hemodynamics, and laboratory parameters) with definitive surgical management in order to improve patient outcomes. The purpose of this chapter is to provide an overview of hemodynamic monitoring, identify end-points of trauma resuscitation, and discuss the evolving role of thromboelastography in trauma for patient-specific hemostatic resuscitation.

Shock

In trauma, shock occurs because of inadequate tissue perfusion and oxygen delivery. This results in the failure to meet the metabolic demands of peripheral tissues leading to metabolic acidosis, inflammation, and coagulopathy. The degree of shock is represented on a spectrum of physiologic derangements ranging from compensated to decompensated shock. Traditional methods of evaluating shock are guided by early recognition of the injury pattern and bedside evaluation of blood pressure, heart rate, mental status, urine output, and skin perfusion. Regardless of the underlying shock etiology, resuscitation goals are aimed at reestablishing tissue perfusion. Adequacy of tissue oxygenation is dictated by the balance between oxygen delivery (DO_2) and oxygen consumption (VO_2). The delivery of oxygen is determined by:

$$\text{Oxygen Delivery } (DO_2) = \text{Oxygen Content } (CaO_2) \times \text{Cardiac Output}(CO)$$

where

CaO_2 $[(1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)]$

Hb Hemoglobin (Hbg) measures the amount of the hemoglobin molecule in a volume of blood. Normal range for men: 13.8 to 17.2 g/dL, women: 12.1 to 15.1 g/dL

SaO_2 the arterial O_2 saturation (%)

PaO_2 partial pressure of O_2 in arterial blood (mmHg)

The majority of severely injured trauma patients arriving at hospital will have either occult or compensated shock because the cardiovascular system is able to support perfusion but tissue hypoxia may continue. Further interrogation of a patient's hemodynamic and metabolic end-points may help characterize the degree of physiologic derangement and provide guidance for resuscitation. Patients in decompensated shock are physiologically deranged. Restoration of tissue oxygenation is achieved through therapeutic fluid administration, control of hemorrhage, and management of the coagulation system. There is no single optimal end-point of resuscitation, but patient-specific resuscitation goals should be used to evaluate the effectiveness of therapeutic interventions whenever possible. The collective interpretation of vital signs, physical examination, injury pattern, laboratory values, and other advanced hemodynamic parameters will enable the physician to target resuscitation to a specific patient (Fig. 1). Resuscitation efforts should continue until homeostasis has returned. This requires frequent ongoing evaluation of a patient. As technology advances, our ability to precisely evaluate injury patterns and resuscitation end-points will likely improve.

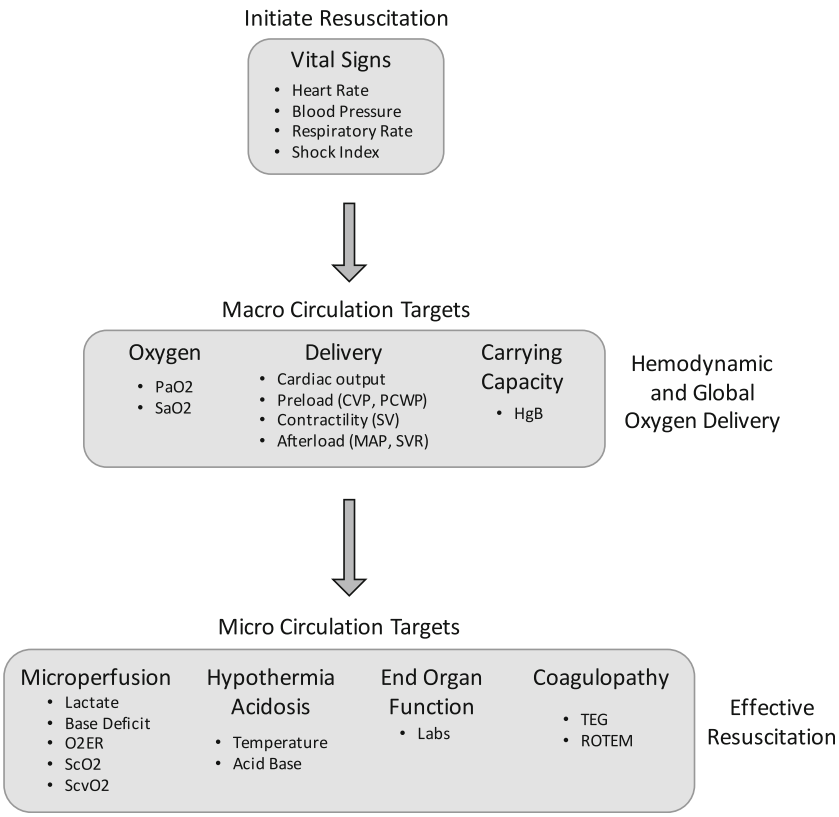

Fig. 1 Evaluation of end-points of resuscitation

Cardiopulmonary Interactions

There is growing recognition that static predictors of fluid responsiveness (heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP), pulmonary artery diastolic (PAD), pulmonary artery occlusion pressure (PAOP)) are unreliable. Understanding cardiopulmonary interactions provides the physiologic rationale for dynamic evaluation of intravascular volume status. These interactions form the basis of changes in intrathoracic pressure (ITP) and lung volume on venous return to the heart and the resultant left ventricular function. During periods of spontaneous ventilation, there is an increase in the negative ITP during inspiration resulting in an increase in venous return which increases right ventricular (RV) volume. The increased RV volume causes the intraventricular septum to shift left resulting in a decreased left ventricular (LV) preload and subsequent reduction in stroke volume and pulse pressure [3•].

The reverse occurs in patients under positive pressure ventilation. In this case, the LV stroke volume increases during inspiration. This is the result of LV preload increase under mechanical ventilation and concurrent LV afterload decrease. Under these conditions, the arterial pressure rises during inspiration and falls during expiration due to changes in ITP secondary to positive pressure ventilation [4••].

Certain maneuvers may be undertaken to accentuate the cardiopulmonary interactions to provide further assessment of a patient's intravascular volume status. A passive leg raise provides an acute volume expansion of 500–700 cm^3 in an adult patient. Evaluation of dynamic parameters provides a tool to determine further fluid responsiveness [5].

Pulse pressure variation (PPV) and stroke volume variation (SVV) are measures that reflect volume responsiveness and rely on the principles of cardiopulmonary interactions in patients who are mechanically ventilated [6•]. As a patient is mechanically ventilated, there is variability in cardiac output with the respiratory cycle. The greater the change in cardiac output in relation to respirations, the more likely the patient is to respond to a fluid bolus with a resulting increase in cardiac output [7].

Pulse Pressure Variation (PPV)

Pulse pressure variation is a dynamic variable used to characterize changes in pulse pressure width during the course of the respiratory cycle in patients who are mechanically ventilated [$PPV = (PP_{max} - PP_{min})/PP_{mean}$]. It can be evaluated in patients with arterial waveform monitoring and calculated using a standard bedside monitor.

The patient must be in normal sinus rhythm, in a controlled mode of mechanical ventilation with a tidal volume of at least 8 mL/kg without spontaneous respiratory efforts. Meta-analysis data suggests a PPV of >12% is associated with volume responsiveness with a sensitivity and specificity of 0.89 and 0.88, respectively [8]. Causes of increased systolic pressure variation in trauma patients include hypovolemia, tamponade, LV dysfunction, massive PE, pneumothorax, and raised intrathoracic or intra-abdominal pressure.

Stroke Volume Variation (SVV)

Stroke volume variation is the percentage change in SV over the respiratory cycle [$SVV\ (\%) = (SV_{max} - SV_{min})/SV_{mean}$]. Waveform analysis of the arterial catheter pulse pressure contour is used to calculate SV. This analysis technique is based on proprietary algorithms used in commercial devices such as the FloTrac™ and the PiCCOplus™. The FloTrac™ system calculates SV based on the contribution of pulse pressure evaluation of vascular resistance and compliance using pulse wave analysis. The PiCCOplus™ system evaluates SV by measuring the area under the arterial pressure wave and dividing this area by the aortic impedance [9]. A SVV threshold of 12% suggests fluid responsiveness [10]. This is an indication that stroke volume is sensitive to preload changes resulting from positive pressure ventilation. The utility of SVV is limited in patient with small tidal volumes, ARDS, high PEEP, arrhythmia, and patient on high doses of vasopressors.

Mixed and Central Venous Oxygen Saturation

Venous oxygen saturation monitoring provides a means of measuring global oxygen uptake and is a measure of the end result of O_2 consumption and delivery. It provides useful information to help guide resuscitative efforts and help augment cardiac output and oxygen delivery to peripheral tissues. Mixed venous oxygenation saturation (S_vO_2) and central venous oxygenation saturation ($S_{cv}O_2$) are representative of oxygen extraction by tissues. The terminology used is dependent on the sampling location of the venous blood. Sampling of venous blood prior to oxygen uptake in the pulmonary capillaries is our best way of evaluating venous oxygen saturation. This sampling requires the use of a pulmonary artery catheter and is referred to as mixed venous oxygen saturation (S_vO_2) monitoring. The S_vO_2 is measured intermittently by withdrawing a blood sample from the distal port of a PA catheter or continuously by using a fiber optic PA catheter via a spectrophotometer. The S_vO_2 is affected by the cardiac output, S_aO_2, Hb, and VO_2. Alternatively, a commonly used central venous catheter can be positioned such that the catheter tip is at the level of the right atrium. Sampling venous gases at this site is referred to as the central venous oxygen saturation. The drawback of this method results from incomplete mixing of blood from the inferior vena cava (lower body), superior vena cava (upper body), and the coronary sinus (heart). A normal oxygen extraction is 25–30% which corresponds to a $S_{cv}O_2$ >65%. An $S_{cv}O_2$ less than 65% is a marker of impaired tissue oxygenation. Patients with a $S_{cv}O_2$ greater than 80% suggests a high flow state seen in patients with microcirculatory

shunting, cytotoxic dysoxia, or left to right shunts [11]. $S_{cv}O_2$ is normally less (approximately 2–4%) than S_vO_2 because the majority of sampled blood is from the SVC where upper body oxygen extraction is higher. Patients with distributive shock from sepsis, targeting a $S_{cv}O_2$ >70% as a goal-directed therapeutic strategy, have a significant mortality reduction (16%) [12••]. Using S_vO_2 as an end-point during resuscitation is favorable as it provides a rapid, real-time, continuous method for evaluating tissue oxygenation [13–15]. This technique is typically used in the ICU rather than the trauma bay or the operating room. The approach is to evaluate S_vO_2 or $S_{cv}O_2$ before and after an intervention to assess the effectiveness of an intervention provided whether it is administration of fluids or the addition of vasopressors or inotropes.

Echocardiography

The introduction of transthoracic (TTE) and transesophageal (TEE) echocardiography in the management of critically ill trauma patients has dramatically improved the diagnostic evaluation of patients in shock. With modest amounts of training, a surgical intensivist can perform a goal-oriented echocardiogram to obtain qualitative evaluation of the RV and LV size and function, cardiac output, identification of pericardial tamponade, and assessment of fluid responsiveness.

A focused cardiac ultrasound examination can provide valuable information and help guide resuscitation of a critically injured patient. The examination is based on the scanning of three main anatomic areas (Fig. 2):

- Parasternal (long-axis and short-axis views)
- Apical (four-chamber view)
- Subcostal (four-chamber view)

Echocardiography is used to evaluate both static and dynamic parameters and provides a global picture of a patient's circulatory state. The following measurements are used for the evaluation of hemodynamic and volume status: IVC diameter, SVC distensibility index, IVC collapsibility index, LV end-diastolic area, and LV outflow track velocity time integral.

The use of echocardiography for the dynamic evaluation of fluid responsiveness is limited to patients on volume-controlled mode ventilation, the presence of a normal sinus rhythm, and the absence of RV dysfunction and increased intra-abdominal pressures. Echocardiographic features in favor of hypovolemia include hyperkinetic LV and RV activity, small LV end-systolic and end-diastolic areas, increased LV ejection fraction, dynamic LV obstruction, and small IVC diameter with respiratory variation.

Cardiac Tamponade Evaluation for pericardial tamponade is necessary in any trauma patient presenting with hemodynamic instability. This will dictate the need for urgent surgical intervention. Cardiac tamponade can be identified on bedside

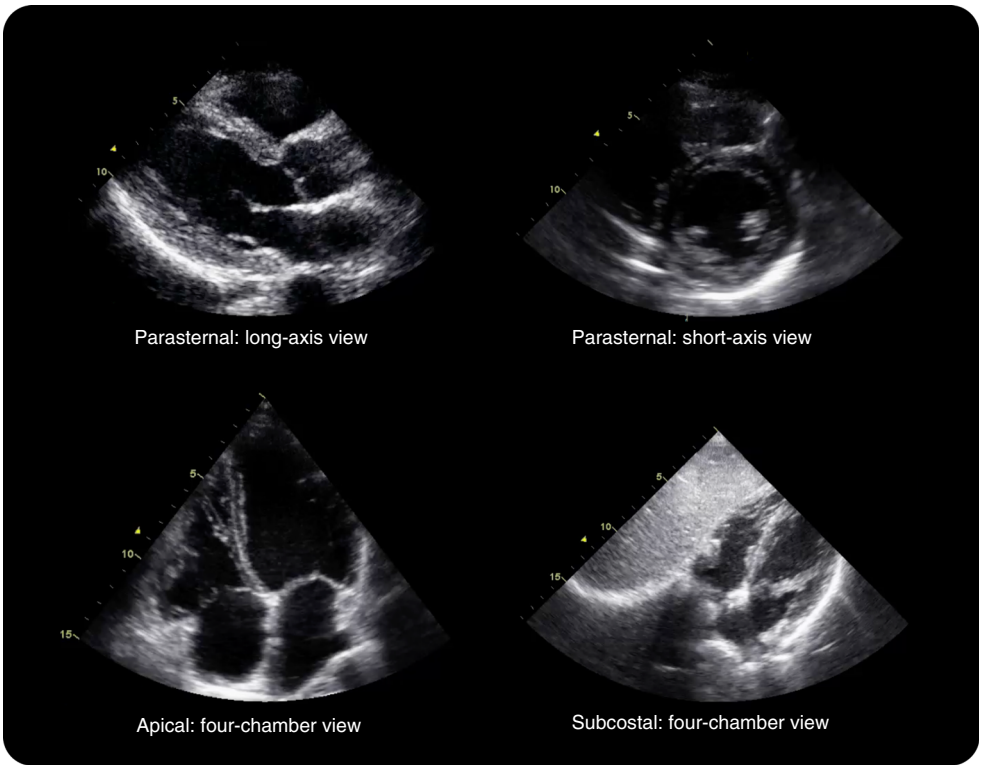

Fig. 2 ECHO—hemodynamic assessment views

echocardiography and is best seen on the apical four-chamber view.

Echocardiographic Measures of Fluid Responsiveness The dynamic evaluation of LV end-diastolic area (LVEDA) in response to a fluid bolus serves as a surrogate marker of cardiac output. Measurement of the LVEDA is done by tracing the endocardium in the parasternal short-axis view [16]. This allows a quantitative evaluation of the ventricular size. The absolute area is not a reliable indicator of fluid responsiveness [12••] but rather changes in LVEDA with fluid administration. A hyperdynamic left ventricle with an LVEDA in the PSAX view of less than 10 cm^2 is indicative of hypovolemia [17]. The variation of LVEDA with respiration has also been demonstrated to be predictive of fluid responsiveness (change >16%) [18]. In this setting, a fluid challenge of 500 cm^3 may benefit the hemodynamic function and should be evaluated with a follow-up echo within 60 min of the fluid bolus.

Dynamic Echocardiographic Parameters Stroke volume evaluation using echocardiography is an ideal method for predicting fluid responsiveness. This can be evaluated by measuring the velocity-time integral (VTI) of blood flow across the aortic valve multiplied by the aortic valve cross-sectional area. The VTI is identified using the apical five-chamber view and measured using Doppler flow across the aortic valve (Fig. 3). This technique combined with a passive leg raise is a useful indicator of volume responsiveness. A 12.5% change in VTI with volume expansion has been demonstrated to be 77% sensitive and 100% specific for detection of >15% in cardiac output [19].

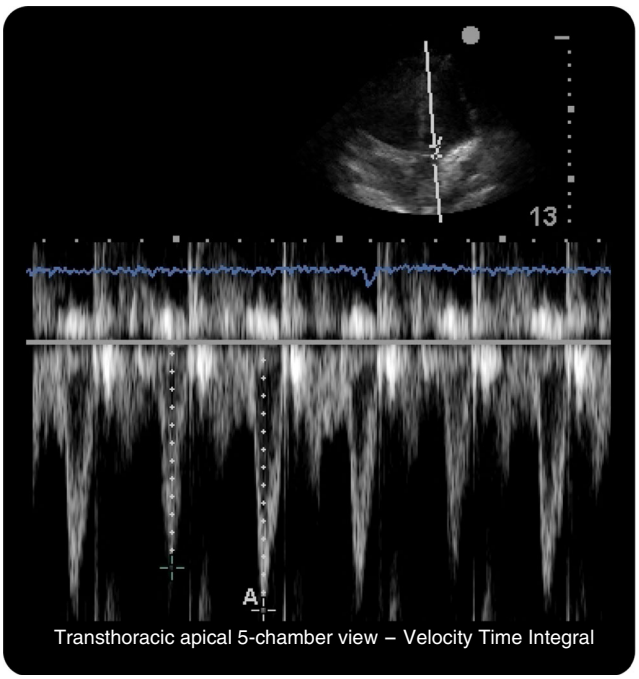

Fig. 3 ECHO—transthoracic apical 5-chamber view with velocity time integral

Respiratory Variation in Vena Cava Diameter Ultrasound examination of the vena cava in patients with mechanical ventilator support can provide important information about fluid responsiveness. This evaluation requires characterization of changes in the cross-sectional area of the vena cava during the respiratory cycle (Fig. 4). During mechanical ventilation, the IVC distends during inspiration and collapses during expiration. The opposite is true for the SVC where the SVC is collapsed during inspiration and distended during expiration. This observation is exaggerated in patients with relative hypovolemia. Inferior vena cava distensibility greater than 12 to 18% [20, 21•] and superior vena cava collapsibility greater than 36% is associated with fluid responsiveness [21•].

Metabolic End-Points of Resuscitation

Hemorrhagic shock in trauma patients results in regional hypoxia leading to anaerobic metabolism, lactic acid production, and eventually to organ failure and death. The metabolic markers of perfusion are indicators of micro-circulation resuscitation. These parameters include oxygen extraction ratio, lactate, pH, and base deficit (BD). These markers are well-established end-points of resuscitation which provide valuable information when evaluated over time. These measures are limited by the inability to process in real time [22]. The frequency of monitoring is dependent on the severity of shock and trending of each parameter. A patient with active hemorrhage with a rising lactate and BD may require an arterial blood gas every hour until improvement of metabolic parameters is seen. Measuring micro-circulation through regional end-points in resuscitation is an emerging field of study.

Oxygen Extraction Ratio

The oxygen extraction ratio is a measure of the oxygen uptake in the peripheral tissues. It is calculated as the ratio of oxygen consumption (VO_2) to oxygen delivery (DO_2) [$O_2ER = VO_2 / DO_2 = (SaO_2 - S_vO_2)/SaO_2$], where the global oxygen delivery (DO_2) is the total amount of oxygen delivered to the tissues per minute and the oxygen consumption (VO_2) is the total amount of oxygen removed from the blood per minute [23, 24]. A normal oxygen extraction ratio is in the range of 20–28%. An O_2ER >50% is a marker of tissue dysoxia.

Lactate

Lactate is the direct byproduct of systemic hypoperfusion leading to anaerobic metabolism. During periods of inadequate oxygen supply, the body reverts to anaerobic

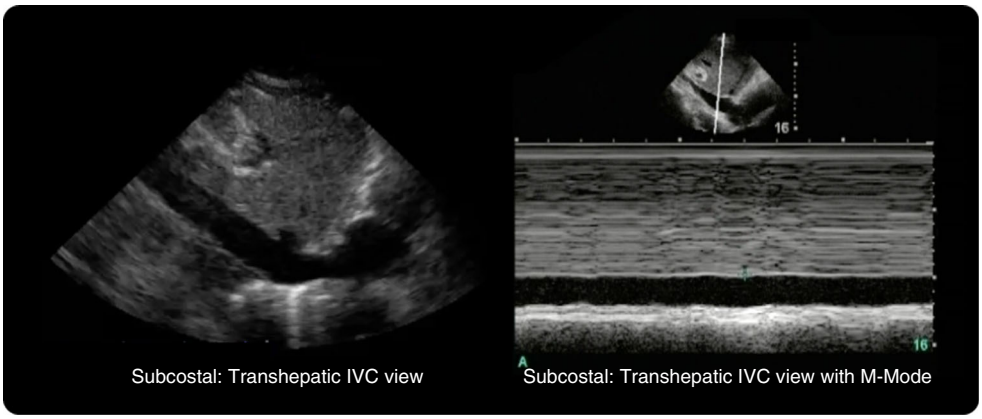

Fig. 4 ECHO—subcostal transhepatic IVC view with M-Mode tracing

metabolism where energy (ATP) is created through the breakdown of carbohydrates in the absence of oxygen. This is represented by the following equation:

$$\text{Pyruvate} + \text{NADH} + \text{H} \longleftrightarrow \text{Lactate} + \text{NAD}$$

Within the trauma literature, the initial serum lactate level correlates with clinical outcome [25]. A lactate level of 3.4 mmol/L or greater is predictive of in-hospital mortality [26–28]. The inability to clear serum lactate by 24 h prognosticates a poor outcome. Early clearance of lactate in response to resuscitation has been found to be a positive prognostic indicator [29]. Therefore, early resuscitative efforts should be targeted at rapid normalization of lactate levels [30]. There are several potential causes of increased serum lactate levels despite normal tissue perfusion. These causes include aerobic glycolysis, Beta-2 stimulation, cytokine stimulation, impairment of oxidative phosphorylation, inhibition of pyruvate dehydrogenase, and in patients with liver or kidney dysfunction.

Base Deficit

Base deficit (BD) provides a useful measure of tissue hypoperfusion and acidosis and is a predictive marker of mortality in trauma patients. The degree of acidosis is largely a reflection of increased lactate due to tissue hypoperfusion. The BD represents the pure metabolic component of a patient's acidosis and represents the amount of base needed to return plasma back to pH 7.4. An elevated BD during resuscitation is reflective of impaired oxygen utilization. The BD correlates with mortality, intra-abdominal injury, and transfusion requirements. Failure to correct the BD within the first 24 h of admission is a marker of worse outcome. A BD of less than −6 is correlated with poor patient outcomes and need for blood transfusions [31]. Caution must be used when interpreting the BD value in patients who have received high-volume resuscitation with normal saline with resulting hyperchloremia, the use of sodium, patients with alcohol or cocaine use, and diabetic patients. The BD provides a rapid indication of adequacy of resuscitation and is useful as a serial measure. The degree of acidosis in trauma influences coagulation and hemodynamics in patients [32]. Patients with pH below 7.10 are at increased risk of arrhythmias, pulmonary vasoconstriction, decreased cardiac output, decreased systemic vascular tone, and reduced responsiveness to catecholamines.

Regional End-Points

Many techniques to evaluate global tissue micro-circulation have been developed. Global indicators of oxygen delivery marking end-points of resuscitation include lactate level, BD, DO_2, VO_2, S_vO_2, and $S_{cv}O_2$. These parameters characterize overall tissue perfusion but provide little information on regional cellular oxygenation [33]. Consumption and delivery of oxygen will vary between different areas of the body; therefore, in an effort to optimize regional oxygen uptake, technologies have been developed for the evaluation of regional tissue micro-circulation. These technologies have the benefit of providing information regarding the regional update of oxygen in specific tissue beds. This is in contrast to $S_{cv}O_2$ which provides a systemic marker of oxygen uptake. These technologies include gastric tonometry, sublingual capnography, and near-infrared spectroscopy. Although these technologies are promising new developments, their widespread utilization remains limited.

Temperature

Trauma patients presenting with hypothermia are at risk of coagulopathy and worse outcomes. The degree of hypothermia at presentation is associated with higher mortality rates: >34 °C, 7%; 33–34 °C, 40%; 32–33 °C, 67%; and <32 °C, 100% [34]. Aggressive rewarming of hypothermic patients should begin in the pre-hospital setting and continue in hospital. As an adjunct to the primary survey, all clothing should be removed from the patient and replaced with preheated blankets or a forced air warming device. Patients with severe

hypothermia may require additional measures of rewarming including administration of warm intravenous fluid or blood products, intracavitary rewarming with warm saline, central venous or esophageal warming catheters, and lastly extracorporeal rewarming.

Hemostatic Resuscitation

Damage control resuscitation in trauma is an approach integrating principles of permissive hypotension, hemostatic resuscitation, and damage control surgery. The underpinning of permissive hypotension is to maintain a blood pressure high enough to allow perfusion but to avoid exsanguination by disrupting unstable clots and worsening bleeding. Permissive hypotension should be disregarded in patients with suspect traumatic brain injury as the goal is to maintain cerebral perfusion pressure in the setting of possible increased intracranial pressure and to ensure adequate oxygen delivery to the brain. The goal of hemostatic resuscitation is early identification and correction of hypothermia and acidosis, early use of blood products, and limiting crystalloid use. The use of blood products in resuscitation should target a ratio similar to whole blood. The aim of this strategy in combination with avoidance of crystalloids is to prevent a dilutional coagulopathy and complications of aggressive crystalloid fluid resuscitation. Hemodilution decreases the concentration of clotting factors and leads to coagulopathy. The excessive use of crystalloids will cause redistribution of fluid into the extravascular space. This leads to tissue edema, pulmonary edema, and abdominal compartment syndrome. Crystalloid resuscitation in excess will also lead to worsening acidosis. The use of blood products should target a "balanced" ratio resembling whole blood. The precise ratio of platelets, plasma, and pRBCs is still being debated. Blood products administered to patients should be warmed in order to avoid hypothermia. Calcium replacement may be required in patients who have received massive transfusion. Early transfer to the operating room for arrest of hemorrhage is essential if indicated, and the principles of damage control surgery may apply in patients with severe physiological derangements.

Coagulopathy

Exsanguination from traumatic injury is a major cause of death with nearly 25% of patients having a significant trauma-associated coagulopathy (TAC) at the time of presentation. The predominant clinical factors contributing to acute traumatic coagulopathy are the degree of tissue injury and tissue hypoperfusion resulting in the uncontrolled release of tissue factor from endothelial injury. Other factors include hemodilution, hypothermia, acidosis, systemic inflammation, and genetic predisposition. The resulting uncontrolled thrombin generation and consumption of clotting factors lead to uncontrolled hemorrhage from distortions in the coagulation cascade. Coagulopathy is further worsened by the combination of acidosis, hypothermia, and hypoperfusion.

Evaluation of a patient's coagulation system has traditionally been based on partial thromboplastin time (PTT) and prothrombin time or international normalized ratio (INR). The PTT and INR measure the intrinsic and extrinsic clotting pathway functions, respectively. These measures of hemostasis do not accurately reflect the true underlying coagulopathy of trauma as they only evaluate the plasma components of the coagulation cascade. These tests lack the ability to identify specific coagulation factor deficiencies and are often time consuming to process.

Over the past decade, our understanding of the pathophysiology of TAC has improved dramatically. Using the cell-based model of coagulation, the in vivo behavior of coagulation is described as three overlapping stages: initiation, amplification, and propagation. This model has the benefit of providing a more accurate functional representation of the coagulation system. To further characterize the dynamics of clot formation, viscoelastic coagulation assay (VCA) has become a useful tool in the evaluation of patients with massive hemorrhage and coagulopathy. Unlike PTT and INR, VCA provides a functional measure of the entire clotting cascade beginning with platelet aggregation. This evaluation method provides a dynamic characterization of the coagulation system through the evaluation of the time to initiation of clot, clot formation, clot stability, and fibrinolysis [35].

The viscoelastic properties of blood can be rapidly evaluated using a point-of-care tool referred to as thromboelastography (TEG). Originally described by Hartert in 1948 [36], thromboelastography is used in the characterization and evaluation of a patient's coagulation state (Table 1). This technique relies on a small sample (0.3 mL) of whole blood placed into a cup. A torsion wire is suspended within the cup which oscillates to simulate venous flow (Fig. 5). As a clot begins to form, the coupling of blood to the pin will exert a torque on the torsion wire. This torque is measures and the peaks of oscillations are recorded and used to create a TEG profile (Fig. 6). A series of measured and calculated values are generated. This evaluation of the viscoelastic behavior of blood can be used to identify the pattern of coagulopathy (Fig. 7). TEG values and plots can be used to identify specific abnormalities in coagulation enabling patient specific resuscitation strategies.

Based on these results, specific hemostatic abnormalities can be identified allowing resuscitation strategies to be individualized for the patient. This goal-directed approach allows targeted therapy for patient-specific coagulation abnormalities thereby reducing unnecessary exposure to blood products and their potential side effects. Hemostatic resuscitation strategies guided by thromboelastography using a balanced transfusion approach result in improved hemostasis.

Table 1 TEG values and interpretation

Parameter		Normal range	Description	Measures
Clotting time	*R*	5.0–10.0 min	• Represents the enzymatic reaction • Elongated R: • Coagulation factor deficiencies • Anticoagulant drugs (warfarin, heparin) • Short R: • Presence of hypercoagulability	Clotting factors (intrinsic pathway)
Clot kinetics	*K*	1.0–3.0 min	• Represents the speed of clot formation • Time from the end of R until the clot reaches 20 mm • Function of the intrinsic pathway, platelets, and fibrinogen • Short K: • Increased platelet activity	Fibrinogen, platelet number
	Alpha	53.0–72.0°	• The alpha angle is calculated by taking the tangent of the curve produced to reach the K value • Rate at which a solid clot is formed • High angle: • Higher platelet activity or blood fibrinogen • Low angle: • Anticoagulants are or platelet inhibitors are present	Fibrinogen, platelet number
Clot strength	MA	50.0–70.0 mm	• Maximum amplitude • Measure of the strength of the clot • High MA: • Higher quality of platelet, fibrinogen, and factor XIII. Relies on the interaction of fibrin and platelets • Low MA: • Insufficient platelet-fibrin clot formation	Platelet number and function
Clot stability	LY30	0–3%	• Measure of fibrinolysis • Time interval between MA and 0 amplitude in the TEG	Fibrinolysis

Dysregulation of the fibrinolytic system is an important contributor to trauma-induced coagulopathy and trauma-associated mortality. The likely source of this dysregulation is the presence of increased serum levels of thrombomodulin and tissue plasminogen activator [37]. By reducing excessive fibrinolysis, tranexamic acid has been demonstrated to decrease transfusion requirements and improve mortality in trauma patients [38••]. However, with the use of TEG, there is a growing body of literature suggesting anti-fibrinolytic treatments should be tailored to patient-specific needs. Current retrospective data sets suggest the characterization of fibrinolysis greater than 3% should be the trigger for anti-fibrinolytic therapy [39].

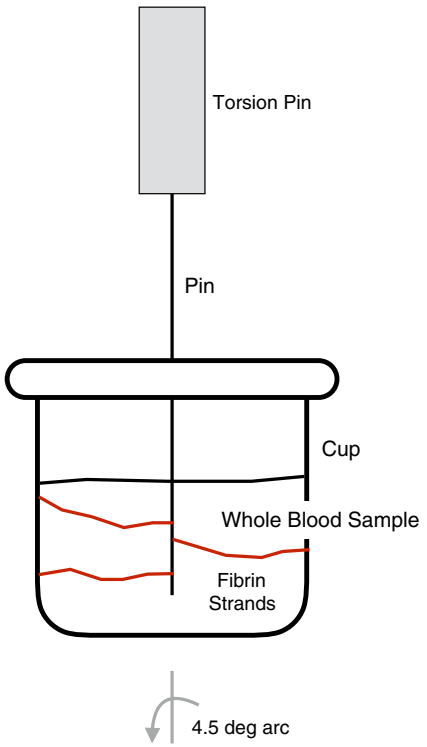

Fig. 5 TEG sample processing—whole blood sample (0.6 cm^3) is placed into a cup and oscillated within a rotational range of 4.5° with a torsion pin placed within the sample

Clinical Integration

The clinical integration of hemodynamic and metabolic endpoints of resuscitation can be a challenge as data points may provide conflicting information regarding the resuscitation status of a patient. Careful review of individual parameters and trends is needed to guide optimal therapy. Patient resuscitation should be divided into three phases: (1) trauma bay resuscitation, (2) intraoperative resuscitation, and (3) postoperative/ICU resuscitation. Early resuscitation efforts in the trauma bay should be guided by recognition of the injury pattern, clinical examination (including bedside ultrasound), and interrogation of bedside vital signs. Based on this early evaluation, decisions on blood product utilization and surgical interventions are made. Intraoperative resuscitation is guided by the amount of blood loss and interrogation of vital signs,

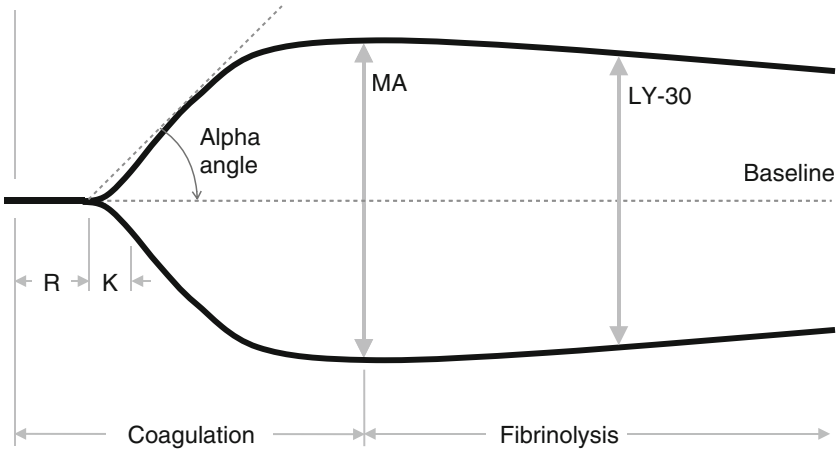

Fig. 6 TEG tracing—measured parameters described in Table 1

arterial waveform data, metabolic parameters including lactate and BD, and use of TEE. Hemostatic resuscitation is also initiated and guided by TEG. The final stage of early resuscitation occurs in the ICU where addition volume resuscitation is guided by vital signs, arterial waveform data, metabolic parameters including lactate and BD, TEG parameters, and use of TTE/TEE.

In patients requiring active resuscitation, repeat evaluation of hemodynamic and metabolic parameters is needed until clinical improvement. The frequency of these reassessments is dependent on the severity of a patient's metabolic derangement. A patient with actively uncontrolled surgical hemorrhage may require continuous real-time monitoring of resuscitation end-points where a patient monitored in the ICU with surgical control of hemorrhage may only require a single evaluation of hemodynamic and metabolic function. At present, there is no randomized control data to suggest one measure is better than another.

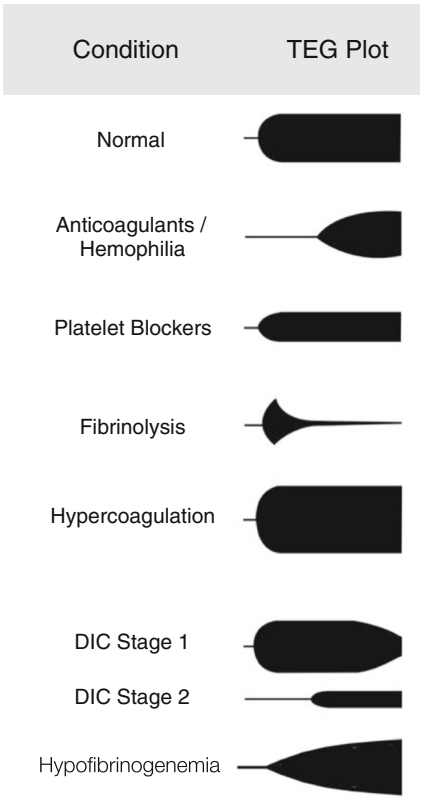

Fig. 7 Sample of TEG tracings

Conclusions

Early and directed resuscitation of trauma patients with the goal of reversing physiologic derangements and correction of coagulopathy is needed to improve patient survival. Resuscitation is guided by both macro- and micro-circulation targets. There are numerous end-points used to guide resuscitation, but none that are universal. The combination of physical examination, the use of hemodynamic parameters, and micro-circulation targets will help guide effective resuscitation. The use of damage control resuscitation strategies including rapid hemorrhage control, early administration of blood products, and minimization of crystalloid fluids combined with point-of-care assessment of coagulopathy has improved survival of traumatically injured patients.

Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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