1 Introduction of Shock

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1.1 Introduction

1.1.1 Definition of Shock

Traditionally shock was defined as an arterial hypotension resulting from impaired cardiac output, blood loss, or decreased vascular resistance. With development of the technology and the increase in understanding shock physiology, cell-level definition has been introduced. In this respect, shock is a state of circulatory failure to deliver sufficient oxygen to meet the demands of the tissues, that is, the imbalance between oxygen delivery and oxygen consumption in the tissues, which results in cellular dysoxia. One recent consensus meeting defined shock as "a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells" [1].

1.1.2 Cellular Oxygen Delivery and Utilization

Oxygen is crucial for ATP production to maintain cellular metabolic function and homeostasis. Inadequate oxygen supplement cannot meet the oxygen demand and causes cellular injury.

In shock state, oxygen delivery (DO_2) is deceased and tissue oxygen consumption (VO_2) is increased. Imbalance between DO_2 and VO_2 is a key mechanism of the shock.

Restoration of tissue perfusion, prevention of cell damage, and maintenance of organ function are basic principles of shock management [1–6].

1.1.2.1 Tissue Oxygen Delivery

Tissue oxygen delivery is defined as a process to deliver arterial oxygenated blood to tissue. Arterial oxygen content (CaO_2) is determined by the amount of oxygen bound to hemoglobin (SaO_2) and dissolved oxygen in plasma.

Arterial oxygen content is described as follows:

$$CaO_2 = \frac{1.34 \times Hb \times SaO_2}{(\text{Hemoglobin} - \text{bound oxygen amount})} + \frac{0.0031 \times PaO_2}{(\text{Dissolved oxygen to plasma})}$$

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Oxygen delivery to tissue (DO_2) can be expressed as a product of arterial oxygen content and cardiac output (CO).

Therefore, the equation for DO_2 is as follows:

$$\begin{aligned} DO_2 &= CO \times CaO_2 \\ &= CO \times (1.34 \times Hb \times SaO_2 + 0.0031 \times PaO_2) \end{aligned}$$

The amount of oxygen dissolved in plasma is so small relative to oxygen bound to hemoglobin that the dissolved oxygen in plasma has a limited role in tissue oxygen delivery.

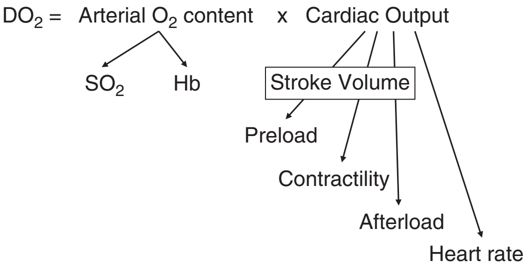


Fig. 1.1 Determinants of oxygen delivery. *DO_2* oxygen delivery, *SaO_2* oxygen saturation, *Hb* hemoglobin

Therefore, the equation for DO_2 can be simplified [7]:

$$DO_2 = CO \times (1.34 \times Hb \times SaO_2)$$

CO is the product of stroke volume (SV) and heart rate (HR).

SV is composed of three components: preload, myocardial contractility, and afterload.

Therefore, adequate CO, hemoglobin level, and oxygen saturation are essential (Fig. 1.1).

Tissue Oxygen Uptake

Tissue oxygen uptake means the amount of oxygen consumed by tissues and cannot be measured directly.

Instead, VO_2 is calculated from difference between the amount of oxygen supplement (DO_2) and amount of oxygen in returned venous blood (Fig. 1.2).

Venous oxygen content (CvO_2) can be expressed similarly to arterial oxygen content:

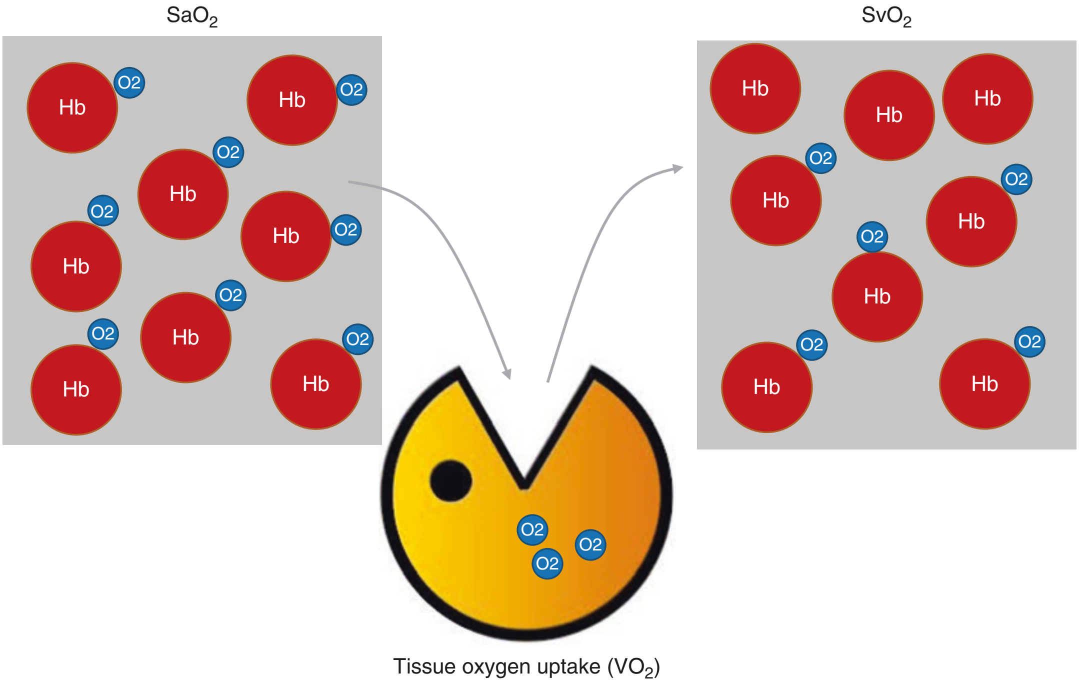


Fig. 1.2 Tissue oxygen uptake is calculated by difference between arterial oxygen saturation and venous oxygen saturation

$$CvO_2 = 1.34 \times Hb \times SvO_2$$
$$VO_2 = CO \times \left(CaO_2 - CvO_2\right)$$
$$= CO \times 1.34 \times Hb \times \left(SaO_2 - SvO_2\right)$$

SvO_2 means mixed venous oxygen saturation. It can be measured with pulmonary artery catheter. Because pulmonary artery catheterization is an invasive procedure, central venous oxygen saturation ($ScvO_2$) which can be drawn from central venous catheter can be used as a surrogate marker for SvO_2 [2]. However, substituting SvO_2 by $ScvO_2$ may be inappropriate because the difference between SvO_2 and $ScvO_2$ is variable in some critically ill patients [8, 9].

1.1.3 Epidemiology

The presence of the shock is usually risk factors of poor prognosis. According to a European multicenter trial, septic shock was the most common (62%) type of shock in the ICU, followed by cardiogenic (16%), hypovolemic (16%), distributive other than septic (4%), and obstructive shock (2%) [10].

1.2 Classification of Shock

Shock has been traditionally classified into four types: hypovolemic, cardiogenic, obstructive, and distributive shock (Table 1.1) [6, 11].

Hypovolemic shock occurs when circulating blood volume is decreased such as bleeding, dehydration, and gastrointestinal loss. Decreased circulating blood causes deceased preload, stroke volume, and cardiac output. Reduced cardiac output causes a compensatory increase in systemic vascular resistance.

Cardiogenic shock is caused by failure of cardiac pump function. Most common cause of cardiogenic shock is myocardial infarction. Other conditions including arrhythmia, cardiomyopathy, and valvular heart disease may decrease cardiac output.

Obstructive shock is caused by the anatomical or functional obstruction of cardiovascular flow system. It includes pulmonary embolism, pericardial tamponade, tension pneumothorax, and systemic arterial obstruction (large embolus, tumor metastasis, direct compression by adjacent tumor, aortic dissection, etc.).

Table 1.1 Type of shock

Type	Hemodynamic changes	Etiologies
Hypovolemic	Decreased preload Increased SVR Decreased CO	Hemorrhage, capillary leak, GI losses, burns
Cardiogenic	Increased preload Increased afterload Increased SVR Decreased CO	MI, dysrhythmia, heart failure, valvular disease
Obstructive	Decreased preload Increased SVR Decreased CO	PE, pericardial tamponade, tension pneumothorax, LV outlet obstruction
Distributive	Decreased preload Increased SVR Mixed CO	Septic shock, anaphylactic shock, neurogenic shock

CO cardiac output, *GI* gastrointestinal, *SVR* systemic vascular resistance, *MI* myocardial infarction, *PE* pulmonary embolism, *LV* left ventricle

Systemic vasodilation and secondary effective intravascular volume depletion result in distributive shock. Septic shock, the most common type of shock, is a kind of distributive shock. Neurogenic shock and anaphylaxis are also included in distributive shock [11, 12].

Several types of shock can coexist in a patient. For example, a patient with septic shock may be complicated by cardiogenic shock, which is caused by stress-induced cardiomyopathy.

1.3 Pathophysiology of Shock

Although there are various kinds of shock with many different clinical conditions, shock is a circulatory mismatch between tissue oxygen supply and tissue oxygen demand.

1.3.1 Vascular Response

For maintaining vital organ perfusion, several autonomic responses are activated.

Stimulation of carotid baroreceptor stretch reflex activates the sympathetic nervous system.

The activation of sympathetic nervous system increases heart rate and myocardial contractility and redistributes the blood flow from skin, skeletal muscles, kidney, and splanchnic organs to vital organs. Dominant autoregulatory control of blood flow spares cerebral and cardiac blood supply.

Release of vasoactive hormones increases the vascular tones. Antidiuretic hormone and activation of renin-angiotensin axis inhibit renal loss of sodium and water and help to maintain intravascular volume.

1.3.2 Microcirculatory Dysfunction

In normal condition, capillary perfusion is well maintained. In shock, however, reduced capillary density and perfusion are shown. Shock is also characterized by endothelial cell damage, glycocalyx alteration, activation of coagulation, microthrombi formation, and leukocytes and red blood cell alteration, which lead to microcirculatory dysfunction [5, 13].

1.3.3 Cellular Injury

Under the normal condition, 38 adenosine triphosphates (ATP) are produced via aerobic glycolysis and TCA cycle.

In shock, however, pyruvate cannot enter into the TCA cycle due to insufficient oxygen delivery (anaerobic glycolysis), which results in only two ATP production. In this process, pyruvate is converted into lactate in cell which is released into circulation (Fig. 1.3).

When cellular hypoperfusion persists, cellular energy stores are rapidly decreased due to inadequate ATP regeneration. After ATP depletion, energy-dependent cellular systems are impaired, cellular homeostasis is threatened, and the breakdown of ultrastructure occurs.

Inappropriate activation of systemic inflammation also causes cellular injures, which leads to multiple organ dysfunction (Fig. 1.4).

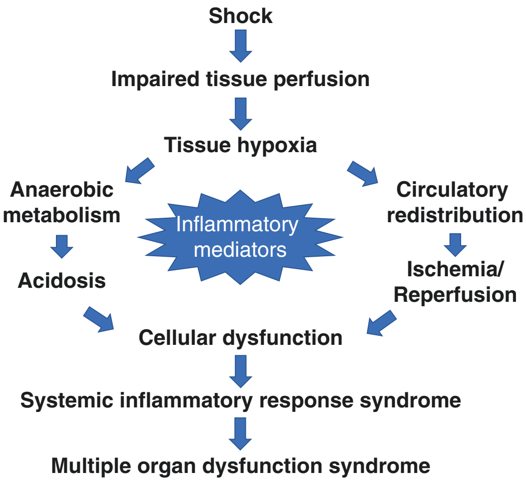
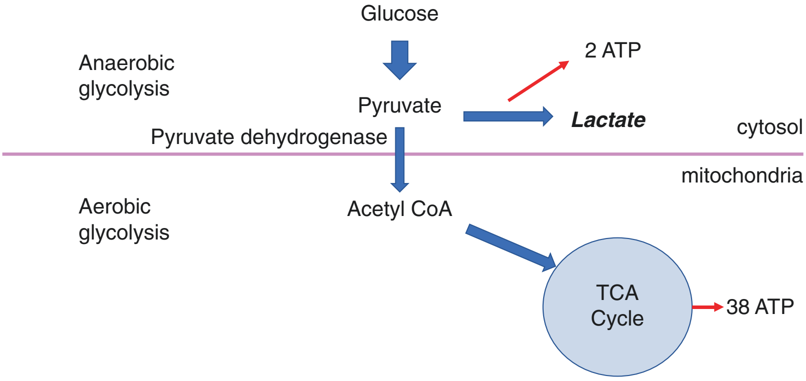


Fig. 1.4 Pathophysiology of shock

Fig. 1.3 Glycolysis pathway. Without oxygen, efficiency of ATP generation is markedly decreased. Lactate is a by-product of anaerobic glycolysis pathway. *ATP* adenosine triphosphate, *TCA* tricarboxylic acid



1.4 Diagnosis of Shock

Diagnosis of shock should be based on comprehensive considerations of clinical, hemodynamic, and biochemical features.

1.4.1 Clinical Features

Tissue hypoperfusion in shock state can cause various kinds of organ dysfunctions. A comprehensive and detailed clinical assessment for the early detection and acute management is required.

1.4.1.1 General Appearance

Shock is a life-threatening condition and stressful reactions such as anxiety, irritability, and agitation can be observed. Diaphoresis, pale skin, and mottled skin suggesting tissue hypoperfusion may be present. Capillary refill time more than 2 s can be used as a surrogate marker of tissue hypoperfusion.

1.4.1.2 Central Nerve System

Patients with shock often present with various symptoms of CNS dysfunction. Visual disturbance, dizziness, syncope, agitation, mental status, delirium, or seizure can be present. Decreased mentality or presence of delirium is associated with increased mortality [14, 15].

1.4.1.3 Respiratory System

Tachypnea is a component of the systemic inflammatory response, and common symptom of shock. Medullary hypoperfusion stimulates respiratory center and augments respiratory effort. Increased workload of breathing combined with persistent hypoperfusion to respiratory muscles eventually causes respiratory muscle fatigue and leads to early respiratory failure. ARDS can develop as a consequence of inflammatory responses induced by shock.

1.4.1.4 Kidney

Renal hypoperfusion and oliguria cause ischemic renal damage. The extent of acute kidney injury is variable in shock. There are a number of clinical tools for the assessment of acute kidney injury. Among them, RIFLE criteria and KIDIGO definition are most commonly used (Tables 1.2 and 1.3) [16, 17].

1.4.1.5 Gastrointestinal Tract

Bowel mucosa is injured by hypoperfusion, splanchnic vasoconstriction caused by the redistribution of blood, and inflammatory insult. Bowel injury causes the destruction of mucosal

Table 1.2 RIFLE criteria [16]

	GFR criteria	Urine output criteria
Risk	Increased serum creatinine × 1.5 or GFR decrease >25%	UO < 0.5 mL/kg/h × 6 h
Injury	Increased serum creatinine × 2 or GFR decrease >50%	UO < 0.5 mL/kg/h × 12 h
Failure	Increased serum creatinine × 3 or GFR decrease >70% or serum creatinine 4 mg/dL (acute rise 0.5 mg/dL)	UO < 0.3 mL/kg/h × 24 h or anuria × 12 h
Loss	Persistent AKI Complete loss of kidney function >4 weeks	
ESRD	End-stage kidney disease (>3 months)	

GFR glomerular filtration rate, *UO* urine output

Table 1.3 KIDIGO definition of AKI [17]

AKI is defined as any of the following: - Increase in SCr by ≥0.3 mg/dL within 48 h - Increase in SCr to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days - Urine volume <0.5 mL/kg/h for 6 h
Stage 1 - Increase in SCr by 1.5–1.9 times baseline - Increase in sSCr by ≥0.3 mg/dL - Urine output <0.5 mL/kg/h for 6–12 h Stage 2 - Increase in SCr by 2.0–2.9 times baseline OR - Urine output <0.5 mL/kg/h for ≥12 h Stage 3 - Increase in SCr by 3.0 times baseline - Increase in SCr to 4.0 mg/dL - Initiation of renal replacement therapy - In patients <18 years, decrease in eGFR to 35 mL/min/1.73 m^2 - Urine output <0.3 mL/kg/h for ≥24 h - Anuria for ≥12 h

AKI acute kidney injury, *SCr* serum creatinine, *eGFR* estimated glomerular filtration rate integrity, leading to bacterial translocation and inflammation-mediated injury [18].

1.4.1.6 Liver

Liver is vulnerable to hypoperfusion and tissue hypoxia. Increase in hepatic enzymes including transaminase and lactate dehydrogenase is common. The synthesis of coagulation factors is impaired by hepatic dysfunction.

1.4.1.7 Hematologic Disorder

Anemia can develop due to direct blood loss (e.g., hemorrhagic shock, acute gastric mucosal bleeding), myelosuppression, and hemolysis. Thrombocytopenia, coagulopathy, and disseminated intravascular coagulation (DIC) can develop. As mentioned above, hepatic injury can worsen the coagulation dysfunction.

1.4.1.8 Metabolic Disorder

Circulatory shock is a stressful event and sympathetic activity is stimulated in the early phase. An increase in release of catecholamine, cortisol, and glucagon and decrease in insulin release can be shown. As a result, hyperglycemia can be shown in the early phase of shock. In advanced stage of shock, hypoglycemia can be present due to glycogen depletion or failure of hepatic glucose synthesis.

Fatty acids are increased early in shock period. However, fatty acids are decreased in the late phase due to hypoperfusion to adipose tissue.

1.4.1.9 Clinical Scoring Systems

Several clinical scoring systems can be used for the assessment of circulatory shock for critically ill patients. Acute Physiology and Chronic Health Evaluation (APACHE) scores (II, III, IV), Simplified Acute Physiology Score (SAPS II), and Sequential Organ Failure Assessment (SOFA) score are commonly used and can be applied to the circulatory shock patients (Table 1.4) [19–23].

1.4.2 Hemodynamic Features

1.4.2.1 Blood Pressure and Heart Rate Monitoring

Blood Pressure

A decrease in cardiac output causes vasoconstriction, leading to decreased peripheral perfusion to maintain arterial pressure. However, preserved blood pressure due to vasoconstric-

Table 1.4 Sequential Organ Failure Assessment (SOFA) score

	0	1	2	3	4
Respiratory PaO_2/FiO_2 (mmHg)	>400	≤400	≤300	≤200 and mechanically ventilated	≤100 and mechanically ventilated
Coagulation Platelet (×10^3/μL)	>150	≤150	≤100	≤50	≤20
Liver Bilirubin (μmol/L)	1.2	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Cardiovascular Hypotension	No hypotension	MAP <70 mmHg	Dopamine <5 or dobutamine (any)	Dopamine >5, epinephrine ≤0.1, or norepinephrine ≤0.1	Dopamine >15, epinephrine >0.1, or norepinephrine >0.1
Central nerve system GCS scale	15	13–14	10–12	6–9	<6
Renal Creatinine (μmol/L) or urine output (mL/d)	<1.2	1.2–1.9	2.0–3.4	3.5–4.9 or <500	>5.0 or <200

Catecholamine doses = μg/kg/min

FiO_2 fraction of inspired oxygen, *MAP* mean arterial pressure, *GCS* Glasgow coma score

tion may be associated with inadequate tissue perfusion, such as decreased central venous oxygen saturation ($ScvO_2$) and increase in blood lactate. Although the presence of hypotension is essential in the diagnosis of septic shock, it is not necessary to define the other types of shock [1, 5, 6].

Indirect measurement of blood pressure is often inaccurate in severe shock status and insertion of arterial catheter should be considered. Mean arterial pressure (MAP) reflects cardiac output better than systolic or diastolic pressure, and is often used as the guidance of shock treatment. The radial artery is commonly used. Femoral, brachial, axillary, or dorsalis artery can be used [7, 24, 25].

Heart Rate

Heart rate is the vital component of the cardiac output. According to the ATLS classification, class II hemorrhage (estimated blood loss 15–30%) showed a tachycardia of >100 beats/min, but normal systolic blood pressure. It means that heart rate is a more sensitive indicator than blood pressure in the early phase of hemorrhage shock [26].

Shock Index

Shock index is HR/systolic BP ratio. It reflects better circulatory status than heart rate or blood pressure alone. Normal ratio is between 0.5 and 0.8. Increased shock index is related with poor outcomes of traumatic or septic shock [27, 28]. Shock index also has predictive value for cardiogenic shock [29, 30].

1.4.2.2 Central Venous Pressure (CVP)

CVP, a direct right atrial pressure, is an indicator of blood volume status. Low CVP (<4 mmHg) in critically ill patient indicates severe volume depletion such as dehydration or acute blood loss requiring volume resuscitation (Table 1.4). However, because CVP is affected by multiple factors including venous tone, intravascular volume, right ventricular contractility, or pulmonary hypertension, CVP-guided shock treatment is no longer recommended. CVP should be interpreted together with other hemodynamic parameters [25, 31].

1.4.2.3 Cardiac Output

Pulmonary Artery Catheter

Pulmonary artery catheter is a flow-directed catheter with balloon tip. It is inserted through the jugular, subclavian, or femoral vein and advanced to the right atrium, right ventricle, and pulmonary artery. It measures cardiac output with thermodilution method and has been the reference method for measuring cardiac output in shock states. However, no randomized trial showed benefit of pulmonary artery catheter placement in critically ill patients [32–37]. Because of its invasiveness, routine placement of pulmonary artery catheter is not recommended. However, pulmonary artery catheter can measure accurate right atrial pressure and pulmonary artery pressure; it may be particularly useful in cases of shock associated with the right-sided heart failure, pulmonary hypertension, and/or difficult ARDS (Tables 1.5 and 1.6) [24].

Table 1.5 Hemodynamic characteristics of the shock

	Preload			
	Pulmonary capillary wedge pressure	Central venous pressure	Cardiac output	Systemic vascular resistance
Hypovolemic	Decreased	Decreased	Decreased	Increased
Cardiogenic	Increased	Increased	Decreased	Increased
Obstructive	Decreased	Decreased	Decreased	Increased
Distributive				
Early	Decreased	Decreased	Increased	Decreased
Late	Increased	Increased	Decreased	Increased

Table 1.6 Hemodynamic monitoring of shock

Preload	Cardiac contractility	Afterload	Cardiac output	Cellular oxygenation
Pulmonary artery catheter CVP Echocardiography Transpulmonary thermodilution systems	Echocardiography Transpulmonary thermodilution systems	Transpulmonary thermodilution systems	Pulmonary artery catheter Transpulmonary thermodilution systems Bioimpedance	NIRS Videomicroscopy techniques

Transpulmonary Thermodilution

Although less invasive than pulmonary artery catheter, transpulmonary thermodilution method also requires the insertion of central venous catheter and arterial catheter for the measurement of cardiac output. This method has been shown to be equivalent in accuracy to invasive pulmonary artery thermodilution technique [24]. Cardiac output is intermittently measured via the thermodilution technique using cold saline infusion. Compared to pulmonary artery catheter, the difference is that cold saline is injected not into the right atrium but into a central vein and changes of the blood temperature are detected not in the pulmonary artery but in a systemic artery. Cardiac output measured by this technique has shown a good agreement with that using pulmonary artery catheter in critically ill patients [38].

Continuous cardiac output is measured by the arterial pulse contour analysis. Global end diastolic volume, intrathoracic blood volume, extravascular lung water volume, pulmonary blood volume, pulmonary vascular permeability index, global ejection fraction, contractility, and systemic vascular resistance can also be measured or calculated with this device. Currently commercially available devices are PiCCO and VolumeView/EV1000 system [29].

Transpulmonary Dye Dilution

In this method, lithium, instead of saline, is injected through vein (central or peripheral) and measures changes of the blood temperature in a peripheral artery using specialized sensor probe [39].

LiDCO system is a commercially available transpulmonary dye dilution device.

Ultrasound Flow Dilution (The Costatus System)

After cold saline infusion, this method measures cardiac output with ultrasound velocity and blood flow change instead of thermodilution. It requires a primed extracorporeal arteriovenous tube set (AV loop). Two ultrasound flow-dilution sensors are placed on the arterial and venous ends and provide ultrasound dilution curve through which cardiac output can be calculated [40].

Echocardiography

Echocardiography is an important diagnostic method for evaluation of cardiac status. Nowadays its use is increasing for the management of acute and critically ill patients using bedside sonographic devices [41].

Cardiac output can be measured using pulsed-wave Doppler velocity in the left ventricular outflow tract. Comprehensive sonographic approach can help differential diagnosis of shock. It can help rapidly recognize the physical status of patients, and select therapeutic options [42–44]. Moreover, repeated evaluations can be done easily and help evaluating response to the treatment and help.

Pulse Contour and Pulse Pressure Analysis

Several kinds of devices are developed to estimate cardiac output from an arterial pressure waveform signal. This method reflects changes of cardiac output well in stable patients. However, accuracy is not guaranteed if vascular tone change occurs, which is common in the shock state or when vasoactive drugs are used [45]. Several devices including FloTrac/Vigileo and LiDCOrapid/pulseCO are available.

Bioimpedance

Blood has a relatively low electrical resistance and intrathoracic blood volume change causes significant impedance changes of thoracic cavity. This method detects voltage changes using skin electrode and postulates blood volume changes during cardiac cycle and cardiac output. Any conditions which can affect intrathoracic fluid, such as pleural effusion or lung edema, influence the result of bioimpedance method. This is not a calibrated method and accuracy in measuring cardiac output is questionable [24].

1.4.2.4 Microcirculatory and Tissue Perfusion Monitoring

Near-Infrared Spectroscopy

Near-infrared spectroscopy (NIRS) is a noninvasive technique used for observing real-time changes in tissue oxygenation. Several studies showed prognostic ability of NIRS in septic shock [46–48].

Videomicroscopy Techniques

These handheld microscopic camera devices can visualize capillaries, venules, and even movement of erythrocyte. These methods can help evaluating microcirculatory status. Sublingual microcirculation is usually evaluated in humans. Vessel perfusion status, quality of capillary flow, and presence of non-perfused area are often evaluated [49].

Sidestream dark-field (SDF) or incident dark-field (IDF) technique is used. The orthogonal polarization spectral (OPS) imaging device has been replaced by newer devices based on SDF or IDF imaging [49].

1.4.2.5 Other Indirect Methods

Gastric Tonometry

Tissue hypoxia causes lactate production and metabolic acidosis. Gastrointestinal mucosa is vulnerable to hypoxic injury, easily influenced by remote organ injuries. Stomach can be easily assessed with nasogastric tube. Gastric tonometry measures gastric mucosal CO_2 and calculates gastric mucosal pH assuming that arterial bicarbonate and mucosal bicarbonate are equal. Tissue hypoperfusion results in reduction of gastric mucosal pH. However, this assumption is not correct and mucosal bicarbonate and pH are influenced by various conditions; results should be interpreted with caution [50].

1.5 Management of Shock

1.5.1 Initial Management

1.5.1.1 Airway and Breathing

Airway management is important in patients with shock. Early intubation should be considered in case of respiratory distress, hypoxemia, severe acidosis, and decreased mentality and when airway protection is threatened.

Increased work of breathing increases the oxygen consumption of the respiratory muscles. Decreased work of breathing with intubation and adequate sedation can help improve the tissue oxygen delivery.

Positive pressure ventilation can reduce preload and worsen the hypotension or cause cardiovascular collapse. Volume resuscitation and vasopressor support (if indicated) should be performed before positive ventilation.

1.5.1.2 Fluid Resuscitation

Fluid resuscitation should be started for restoring microvascular circulation when there is evidence of shock.

Initial fluid should be started with isotonic crystalloid. However endovascular permeability is increased in shock state; risk of acute edema with unwanted consequence is high when excessive fluid is infused. Careful monitoring of fluid responsiveness is required. Volume status, cardiac output, blood pressure, and tissue perfusion status should be evaluated repeatedly [6, 25].

1.5.1.3 Fluid Responsiveness

Although adequate volume restoration is a key to the treatment of the shock, excessive fluid resuscitation causes tissue edema, endothelial injury, and impairment of tissue perfusion. Volume overload is related with the poor prognosis of shock patients. Static parameters such as CVP or PAWP or global end diastolic volume is no longer useful, and they alone should not be used for predicting fluid responsiveness. Dynamic parameters such as pulse pressure variation (PPV), stroke volume variation (SVV), or velocity time integral (VTI) are better than static variables to predict fluid responsiveness (Table 1.7) [1, 51].

Pulse Pressure or Stroke Volume Variation

In case of volume depletion, the cardiac output is influenced by the change of the thoracic pressure. During inspiration period, the thoracic pressure rises and right ventricular and left ventricular preload decrease.

These parameters are usually checked during mechanical ventilation and adequate amount of tidal volume (≥7–8 mL/kg). In cases of spontaneous breathing, low tidal volume, or cardiac arrhythmia, pulse pressure or stroke volume variations cannot be assessed accurately. Changes more than 12% are considered as volume-sensitive status (sensitivity 79–84%, specificity 84%) [52].

Table 1.7 Methods for evaluating fluid responsiveness

Static parameter	Dynamic parameter
Central venous pressure Pulmonary capillary wedge pressure	Pulse pressure variation Stroke volume variation Inferior vena cava variation Response to passive leg raising Changes in cardiac output following passive leg raising

Static parameters no longer recommended for evaluation of fluid responsiveness

Passive Leg Raising

Passive leg raising causes movement of blood pooled in the lower extremity to the central circulation. Maximizing the response, the patient has semirecumbent position and change to leg-raising position (Fig. 1.5). During the procedure, direct measurement of cardiac output should be performed.

Positive fluid balance can be expected with 10% or more changes in cardiac output (sensitivity 88%, specificity 92%) [51, 52].

1.5.1.4 Vasopressor

Vasopressor should be started after adequate fluid resuscitation except anaphylactic shock (epinephrine should be injected first) or cardiac arrest. There is no universal optimal target blood pressure. In hemorrhagic shock, hypotensive resuscitation is recommended before definite bleeding control. However, blood pressure target in traumatic brain injury should be higher for maintaining cerebral perfusion pressure [1, 6, 25].

Most vasopressors improve the blood pressure by increasing the vascular resistance and can result in decrease in the capillary perfusion.

1.5.2 Restoring Tissue Perfusion

1.5.2.1 Lactate

Lactate is the product of tissue anaerobic metabolism. Increased blood level reflects the tissue hypoxia and hypoperfusion, and is particularly a useful tool to identify patients with septic shock. If the lactate level has not decreased by 10–20%

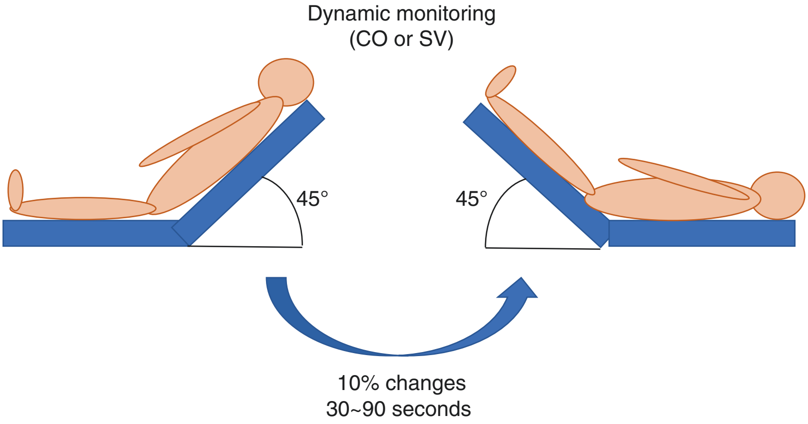


Fig. 1.5 Passive leg-raising test

within 2 h after resuscitation, additional interventions to improve tissue oxygenation should be implemented [1, 25].

1.5.2.2 Specific Treatment of Causes of Shock

Etiology of shock is various and accurate methods to maintain tissue perfusion can be different according to the etiology of shock. Causes of shock should be sought aggressively and etiology-specific treatment should be started promptly. These will be discussed in later parts of this book.

1.6 Summary

- Shock is an imbalance between tissue oxygen supplement and utilization, not just a state of low blood pressure.
- Fundamental of shock treatment is restoration of tissue oxygenation and tissue function.
- Close monitoring of perfusion status and supportive care for organ dysfunctions is important.
- Find specific etiologies of shock and treat them.

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