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• The reduction of the oxygen amount given to the cells may be primarily due to a decrease of DO 2 consequent to a low CO or Hb decrease. In septic shock, despite normal or high DO, the capillary blood flow alterations determine very low cellular oxygen concentrations. This is in large part due to the increased distance between perfused vessels and cells with peripheral shunt and reduced cellular O 2availability.

- Oxygen demand is not coupled with oxygen supply
- Shock is often associated with low blood pressure
- Hypotension is one of the most common clinical presentations of the shock states
- peripheral hypoperfusion, Tachycardia
- The main feature of shock condition is the decrease in oxygen utilization at cellular level

- However, shock may be also defined as a condition in which hypotension is associated with a variable degree of organ derangement (i.e., oliguria, mottled skin, confusion, dyspnea, etc
- $DO 2 = CO \times CaO2$
- $CaO 2 = (Hb \times SaO2 \times 1.34) + (PaO 2 \times 0.003)$
- $VO 2 = CO \times (CaO2 CvO 2)$
- $CvO 2 = (Hb \times SvO2 \times 1.34) + (PvO2 \times 0.003)$

- The difference between arterial and mixed venous saturation (SaO 2 SvO 2) is also defined as oxygen extraction rate (O 2 ER) and identifies the amount of oxygen extracted by peripheral tissues during each cardiac cycle.
- After critical O2ER has been reached, aerobic metabolism begins to be impaired, and a shift toward anaerobic metabolism.
- Regardless the cause and the feature of each type of shock, shock states may be all defined as conditions implying an altered oxygen utilization at cellular level.

Shock Classification

• SvO2 is another parameter which may be used to assess the existence of an imbalance between oxygen demand and supply, as well as the adequacy of CO. Normal value is about 65–70%. In low-CO shock, SvO2 values are typically decreased, while in distributive shock, they are increased.

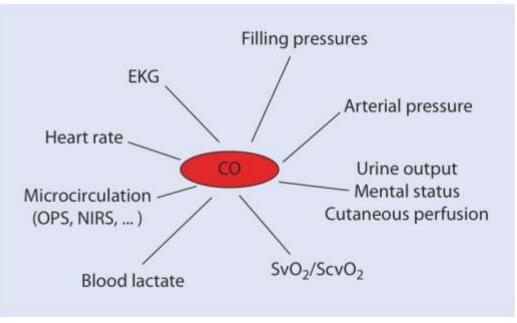
	Low cardiac output			High cardiac output
	Hypovolemic	Cardiogenic	Obstructive	Distributive
Filling pressure	Low	High	High	Low/normal
End- diastolic volumes	Low	High	Low (high in pulmonary embolism)	Low/Normal
SVR	High	High	High	Low
MAP	Normal/low (last phases)	High/normal/ low	Low	Low
SvO ₂	Low	Low	Low	High
Echocar- diography	Small cardiac chambers, preserved contractility	Dilated cardiac chambers, impaired contractility	In tamponade: pericardial effusion, small right and left ventricles In tension pneumothorax: small cardiac chambers In pulmonary embolism: small left ventricle, compressed by the dilated right ventricle	Normal cardiac chambers, preserved contractility (unless septic cardiomyopa- thy occurs)
Clinical signs	Cold and pale skin and	Cold extremi- ties, dyspnea,	Jugular vein distention, dyspnea, increased	Mottled skin, tachycardia,

Table 2.2 Diagnostic tools available to diagnose shock state				
Diagnostic tool	Advantages	Limits		
Clinical signs	Available bedside Easy to detect	Low specificity		
Lactate	Good marker of tissue hypoperfusion Available with ABG point of care Reliable prognostic value Trend over time has a prognostic value	Possibility of false positive Relatively slow normalization (hours)		
ScvO ₂ -SvO ₂	Available with ABG point of care Good marker of O ₂ debt in conditions of low DO ₂	Normal values do not guarantee adequate perfusion		
CO ₂ gap	Available with ABG point of care Correlates CO to metabolism	pH and temperature derangements may alter its interpretationABGs must be drawn exactly at the same time		
Respiratory quotient	Reliable marker of anaerobic CO ₂ production May predict response in terms of O ₂ consumption	CO ₂ content is complex to calculate bedside		
Echocar- diography	Available bedside Useful to identify different types of shock	It requires a skilled operator It does not give any functional information		

Cardiac Output as an Essential Component of DO2

■ Fig. 3.1 Some of the factors that can indicate the (in) adequacy of cardiac output. OPS orthogonal polarization spectral imaging, NIRS near-infrared spectroscopy, SvO₂ mixed venous oxygen saturation, ScvO₂ central venous oxygen saturation, EKG electrocardiogram

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Cardiac Output as an Essential Component of

• Fig. 3.2 The four determinants of cardiac output, using an analogy to the speed of a bicycle. (Reproduced from [6])

