LEC.8 & 9

Shock

The answer commonly provided to the question ‘what is shock?’ is often vague ‘patient with a low blood pressure’, followed by some threshold blood pressure below which ‘shock’ is said to be occurring.

The reason for the uncertainty about what should be a simple concept relates to the fact that the term ‘shock’ is used in a multitude of contexts (e.g., septic, hemorrhagic, distributive, hypovolemic, cytotoxic, cardiogenic, anaphylactic, toxic, spinal, neurogenic, cervical and, even electrical shock). However, the unifying feature of shock, irrespective of the initiating disease or clinical features, is a **failure to deliver and/or utilize adequate amounts of oxygen.**

CIRCULATORY PHYSIOLOGY

OXYGEN DELIVERY: Each day the heart pumps 8000 liters of blood deliver O_2 and nutrients to an estimated 100 trillion cells. This circulatory supply of O_2 and nutrients is tightly regulated by the cardiovascular system.

Oxygen delivery (DO_2)

Is the product of cardiac output (CO) and the oxygen content of arterial blood. Assuming adequate arterial oxygen content, CO is the main determinant of DO_2.

OXYGEN CONTENT: **CaO_2**.

Content can be measured directly or calculated by the oxygen content equation: $CaO_2 = Hb\ (gm/dl) \times 1.34\ ml\ O_2/gm\ Hb \times SaO_2 + PaO_2 \times (0.\ 003\ ml\ O_2/mm\ Hg/dl)$.

TYPES OF SHOCKS

Clinically, the impairment of circulatory supply of O2 to the cells is commonly classified according to which component of the circulation is primarily disturbed – that is,

- **Hypovolemic Shock** (inadequate preload).
- **Cardiogenic Shock** ('pump' failure).
- **Obstructive Shock** (obstructed pump outflow).
- **Distributive or Vasodilatory Shock** (altered vascular capacitance).

HYPOVOLAEMIC SHOCK

Total blood volume is **(55-60)** 70 mL/kg and comprises blood cells and serum.
Hypovolemic shock occurs when **acute blood loss** or **excessive fluid losses** (e.g., gastrointestinal, urinary tract, burns) lead to decreased circulating blood volume .
Loss of circulatory volume will reduce preload and SV. About 10% of circulating volume loss can be restored by the movement of interstitial fluid into the circulation.
Blood loss beyond this invokes cardiovascular compensatory mechanisms in order to restore preload and maintain CO and systemic blood pressure.

These mechanisms include:

- **Increasing venous tone**: vasoconstrictions is an early compensatory response to hypovolemic shock. The venous system holds about 80% of blood volume and acts as a blood reservoir. The sympathetic nervous system controls venous tone and capacitance of the venous system.

- **Increasing arteriolar tone**: sympathetic stimulation of arteriolar resistance vessels increases perfusion pressure to the organs. However, this does not necessarily equate with increased blood flow. The extent of change of arteriolar tone varies between organs in order to ensure adequate blood flow to the vital organs.

- **Increasing HR**: to compensate for the reduction in SV, HR is increased in an attempt to maintain CO.

- **Increasing contractility**: the heart will contract more vigorously in order to increase SV and maintain CO.

Causes of Hypovolemic Shock

- **Blood loss**
  - Vascular injury leg. trauma, surgery
  - Gastrointestinal bleeding (e.g. peptic ulcer, diverticular,
  - Anglo-dysplasia, varicas)
  - Obstetric bleeding (e.g. placenta preivia, post-partum
  - hemorrhage
  - Intra-abdominal hemorrhage e.g. splenic laceration, liver Injury
  - Retroperitoneal hg. aortic aneurysm, femoral artery bleeding, pelvic fracture ectopic rupture,
  - Long-bone fracture
  - Pulmonary hemorrhage, heamothorax

- **Fluid loss**
  - Vomiting
  - Diarrhea
  - Ileostomy losses
  - Sweating
  - Polyuria leg. Glycosuria, diabetes insipidus
  - **Burns**
  - Pancreatitis
  - Ascites
  - Inadequate fluid intake

American College of Surgeons Classes of Acute Hemorrhage

Factors	I	II	III	IV
Blood loss	<15% (<750ml)	15-30% (750-1500ml)	30-40% (1500-2000ml)	>40% (>2000ml)
Pulse	>100	>100	>120	>140
B.P.	Normal	Normal	↓	↓↓
Pulse pressure	N or ↓	↓	↓↓	↓↓
Capillary refill	<2s	2-3s	3-4s	>5s
Resp. rate	14-20	20-30	30-40	>40
Urine output ml/hr	30 or more	20-30	5-10	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious & confused	Confused Lethargic

Priorities in the management of hypovolemic shock are:

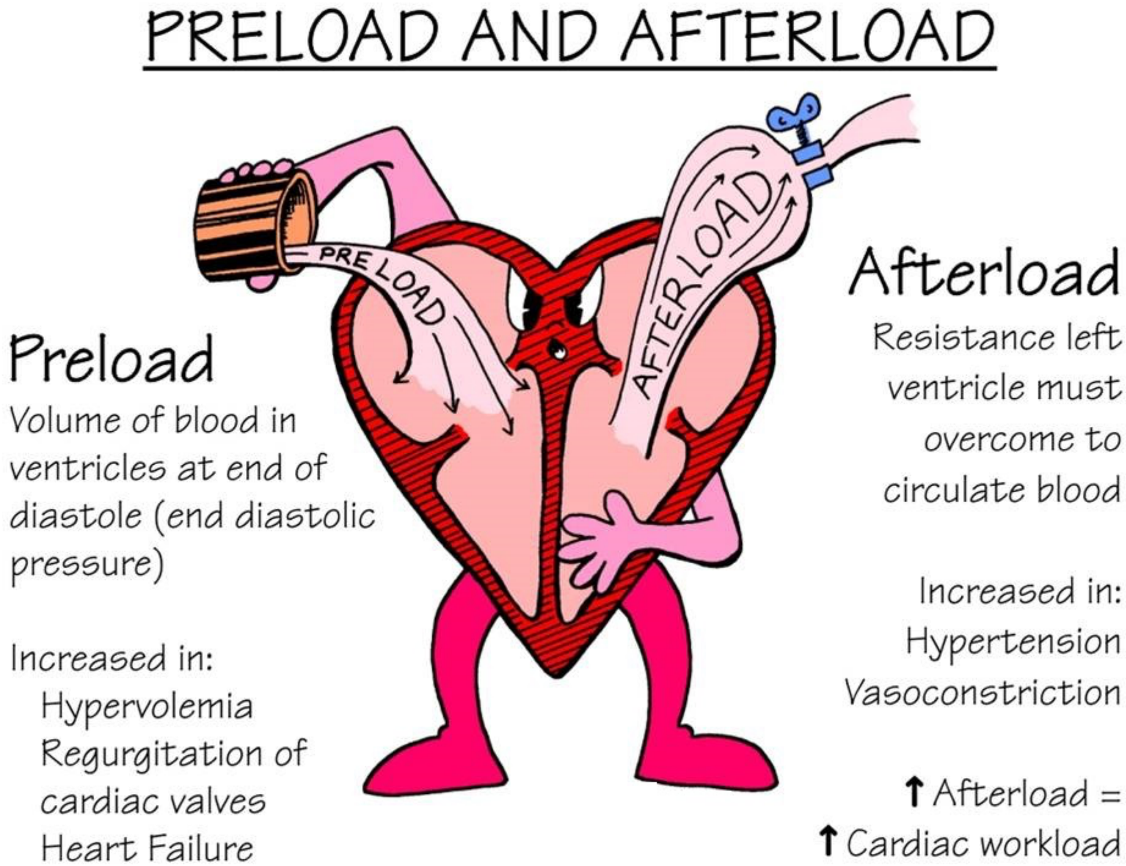
1) controlling the source of blood and/or volume loss, and
2) restoring the circulating volume

CARDIOGENIC SHOCK

The heart is central to the circulatory supply of O2 and if the pump fails then there are few compensatory mechanisms available. Hence, cardiogenic shock has a very high in-hospital mortality rate ranging from 45–100%, depending on the etiology.

Myocardial ischemia is the most common cause of cardiogenic shock, but other etiologies must be considered.

Treatment priorities in cardiogenic shock involve urgent correction of the underlying acute cardiac disease and consideration of afterload reduction while ensuring adequate organ perfusion.



Causes of cardiogenic shock

- Myocardial Ischemia
- Acute valve dysfunction (e.g. chordae rupture, prosthetic valve thrombus)
- Myocarditis
- Contusion
- Septal /ventricular rupture
- Drugs leg. Ca channel blocker overdose, beta blocker overdose
- Brady arrhythmias (e.g. complete heart block)
- Tachyarrhythmia's (e.g. atrial or ventricular tachycardia)

OBSTRUCTIVE SHOCK

Mechanical obstruction to the flow of blood through the cardiac chambers will lead to reduced cardiac output.

The limitation of flow may be due to obstruction within the heart (e.g., valve thrombosis, myxoma) or extrinsic compression (e.g., tension pneumothorax, cardiac tamponade).
Treatment is directed at urgent removal of the obstruction (e.g., drainage of pericardial effusion, lysis of thromboembolism).

DISTRIBUTIVE SHOCK

Blood distribution around the vascular network is controlled by **vascular auto regulation, the autonomic nervous system and hormones**.

Distributive shock results from the failure of these mechanisms, leading to inappropriate distribution of blood (Box 15.4).

Unlike other forms of shock, CO may initially be increased as the heart endeavors to compensate for maldistribution of blood.

Management priorities are to identify and treat the precipitating cause and to improve organ perfusion with resuscitation fluids and vasoactive drugs.

Causes of distributive shock

- **Septic shock**
- Toxic shock
- **Anaphylactic shock**
- Neurogenic shock
- Adrenal/thyroid Insufficiency
- Toxicity (e.g. drugs)
- As a component of multi-organ dysfunction syndrome

SEPSIS and SEPTIC SHOCK

Systemic inflammatory response syndrome (SIRS):
is a syndrome of two or more of the general variables shown in Box 1. It does not mean the patient is septic. Thus, sepsis can be defined as, ‘SIRS with evidence of infection.

- **Sepsis** is infection with systemic manifestations (Box 1).
- **Severe sepsis** is when sepsis induces significant organ dysfunction or tissue hypo perfusion (Box 2).
- **Septic shock** is when there is induced hypotension that persists despite adequate fluid resuscitation.

Box 1

Systemic manifestations associated with sepsis

General variables

- Core temperature >38.3°C or <36°C
- Heart rate >90 bpm
- Tachypnoea (may not feel respiratory distress but a rate >30 pm)
- Significant oedema or positive fluid balance (>20 ml/kg over 24 hours)
- Hyperglycaemia-plasma glucose >7.7 mmol l^{-1}. Diabetics are higher risk

Inflammatory variables

- Leucocytosis (WBC count >12,000 μl^{-1})
- Leukopenia (WBC count <4000 μl^{-1})
- Plasma C-reactive protein: 2 SD above the normal value
- Plasma procalcitonin: 2 SD above the normal value (not routine in all hospitals)

Box 2

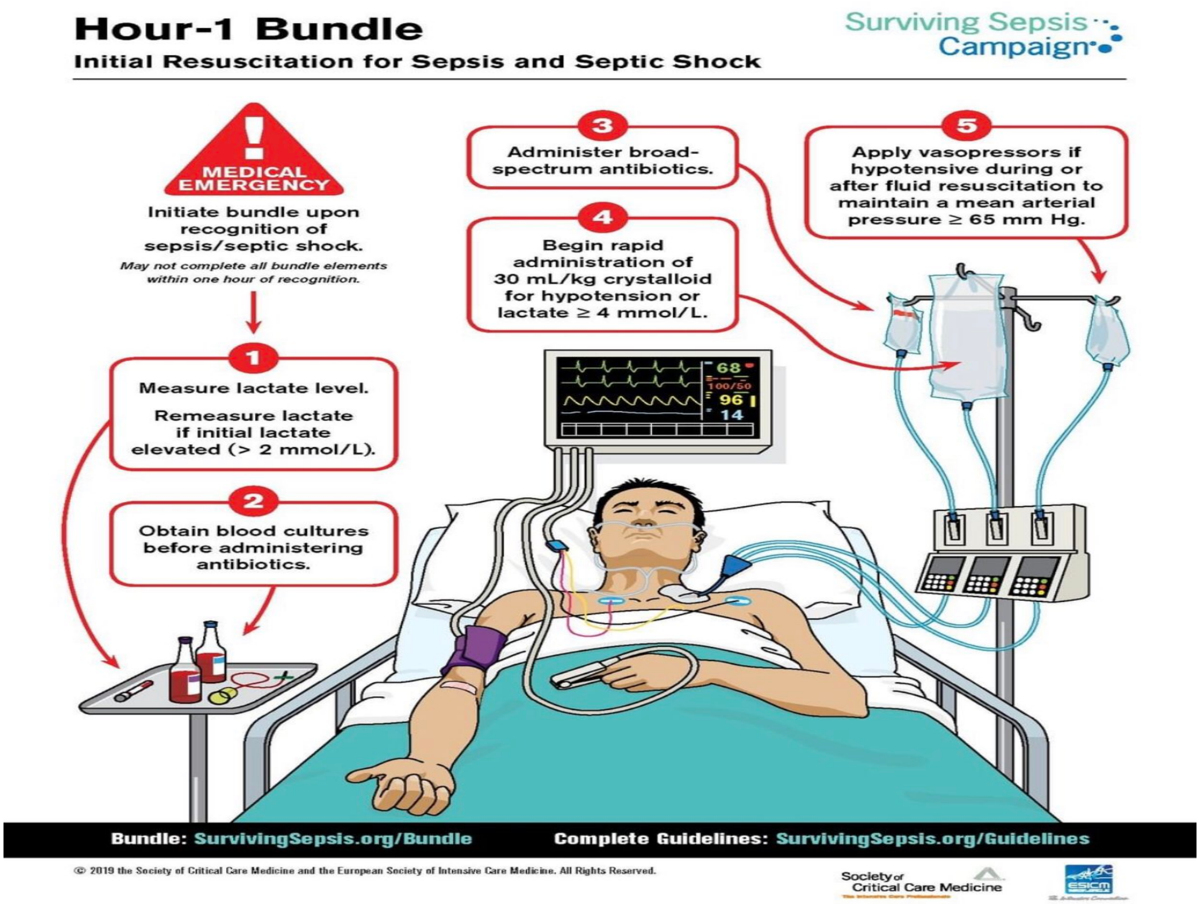
Signs of organ dysfunction associated with severe sepsis

- Sepsis-induced hypotension
- Lactate greater than 4 mmol l^{-1}
- Urine output <0.5 ml/kg/hr for >2 hours, despite fluid resuscitation
- ALI with PaO_2/FiO_2 <250 in the absence of pneumonia as infection source
- ALI with PaO_2/FiO_2 <200 in the presence of pneumonia as infection source
- Creatinine >176 mmol l^{-1}
- Bilirubin >34 mmol l^{-1}
- Platelet count <100,000 μl^{-1}
- Coagulopathy INR >1.5

ALI, acute lung injury; INR, international normalized ratio.

Hemodynamic Variables
- Arterial Hypotension: SBP < 90 mmHg; MAP <65 mmHg

Organ Dysfunction Variables
- Arterial hypoxaemia : SaO_2 <93% on air or (PaO_2/Fi O_2 <300)
- Acute oliguria: urine output <0.5 ml/Kg/hr or <45 ml in 2 hours, despite fluid resuscitation.
- Creatinine increase: >44 μmol/l in 24 hours
- Coagulation abnormalities: INR >1.5 or APTT >60 seconds.
- Ileus (absent bowel sounds)
- Thrombocytopenia: platelet count ,100000 μl^{-1}.
- Hyperbilirubinemia: plasma total bilirubin >34 μmol/l
- Hyperlactatemia >4 mmol/l
- Decreased capillary refill



Hour-1 Bundle
Initial Resuscitation for Sepsis and Septic Shock
Surviving Sepsis Campaign
MEDICAL EMERGENCY
Initiate bundle upon recognition of sepsis/septic shock.
May not complete all bundle elements within one hour of recognition.
1
Measure lactate level.
Remeasure lactate if initial lactate elevated (> 2 mmol/L).
2
Obtain blood cultures before administering antibiotics.
3
Administer broad-spectrum antibiotics.
4
Begin rapid administration of 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L.
5
Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥ 65 mm Hg.
68
100/50
96
14
Bundle: SurvivingSepsis.org/Bundle
Complete Guidelines: SurvivingSepsis.org/Guidelines
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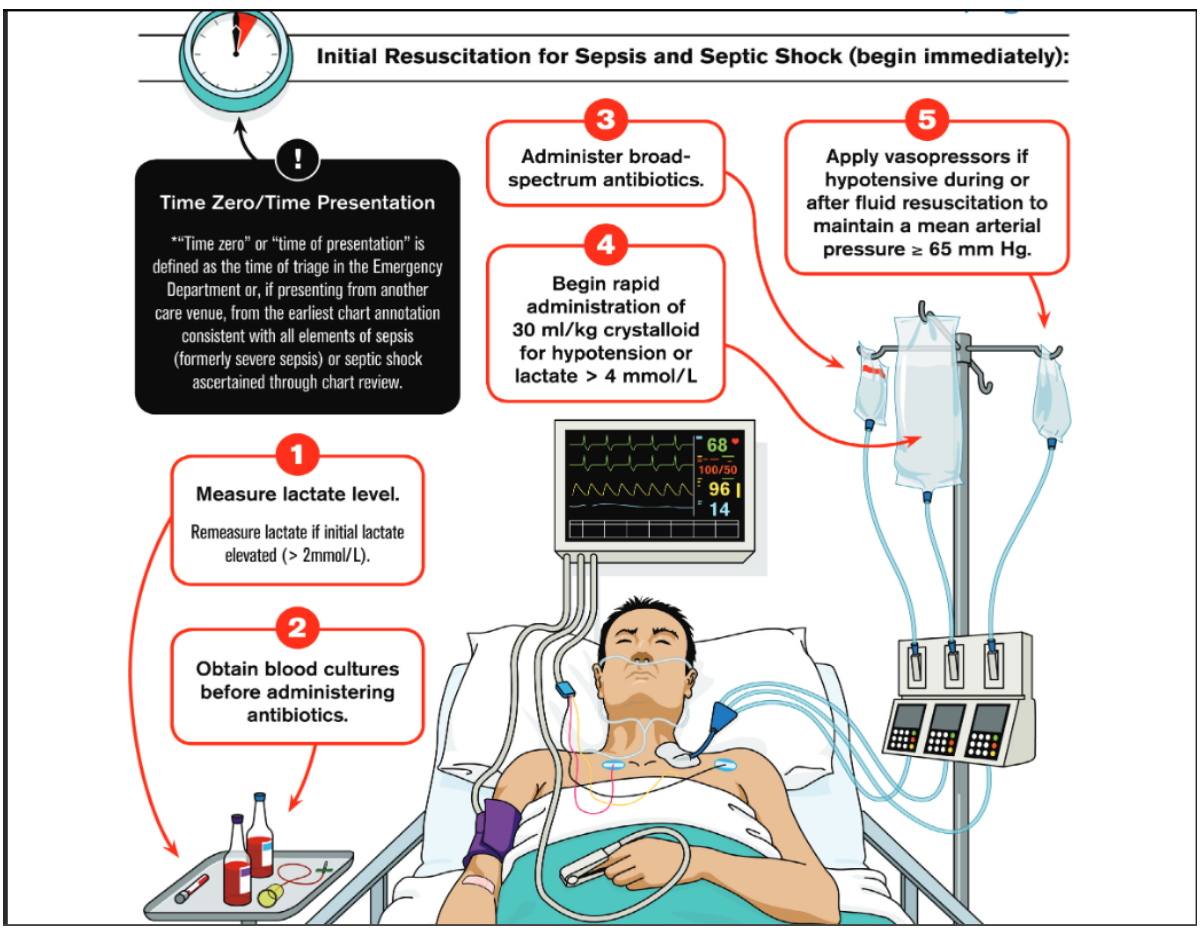

Initial Resuscitation for Sepsis and Septic Shock (begin immediately):
Time Zero/Time Presentation
*"Time zero" or "time of presentation" is defined as the time of triage in the Emergency Department or, if presenting from another care venue, from the earliest chart annotation consistent with all elements of sepsis (formerly severe sepsis) or septic shock ascertained through chart review.
1
Measure lactate level.
Remeasure lactate if initial lactate elevated (> 2mmol/L).
2
Obtain blood cultures before administering antibiotics.
3
Administer broad-spectrum antibiotics.
4
Begin rapid administration of 30 ml/kg crystalloid for hypotension or lactate > 4 mmol/L
5
Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥ 65 mm Hg.
68
100/50
96
14

CLINICAL SIGNS

The clinical features of shock relate to a critically inadequate circulation and insufficient O2 delivery and/or utilization. However, these features are non-specific and will depend on a number of factors including:

- Process leading to shock.
- Severity of precipitating disease or injury
- Physiological reserve of the patient
- Effects of medications.

The compensatory mechanisms to shock and subsequent clinical manifestations are affected by advancing age, cardiovascular disease, autonomic disease and medications.

For example, patients on beta antagonists will not mount the same tachycardic response to fluid loss and patients with pre-existing cardiac disease are less capable of circulatory compensation and so develop features of shock earlier.

Due to the non-specific and varied clinical signs of shock, repeated assessment with frequent monitoring of vital signs is essential.

American College of Surgeons Classes of Acute Hemorrhage

Factors	I	II	III	IV
Blood loss	<15% (<750ml)	15-30% (750-1500ml)	30-40% (1500-2000ml)	>40% (>2000ml)
Pulse	>100	>100	>120	>140
B.P.	Normal	Normal	↓	↓↓
Pulse pressure	N or ↓	↓	↓↓	↓↓
Capillary refill	<2s	2-3s	3-4s	>5s
Resp. rate	14-20	20-30	30-40	>40
Urine output ml/hr	30 or more	20-30	5-10	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious & confused	Confused Lethargic

Hypotension is a sentinel feature of shock and signifies circulatory failure. However, hypotension develops late as systemic blood pressure is initially maintained by compensatory mechanisms (i.e., vasoconstriction, tachycardia, increased myocardial contractility). A decline in mean arterial pressure (MAP) below the lower limit of auto regulation results in reduced perfusion to the vital organs. In a healthy adult, tissue perfusion is typically impaired with a MAP of ≤50 mmHg (6.65 kPa).

In contrast, elderly patients with pre-existing hypertension or vascular disease generally require a higher MAP to ensure adequate regional blood flow.

If systemic blood pressure cannot be accurately determined by the usual auscultatory techniques, then the patient is likely to be markedly hypotensive. Establish the presence of a central (carotid, femoral) or peripheral (radial, posterior tibia) pulse, followed by palpation of the systolic blood pressure using a blood pressure cuff.

Tachycardia is an early compensatory sign of shock. However, in some condition's Bradycardia is the cause of shock (e.g., complete heart block, increased vagal tone in cervical shock, unopposed vagal tone in neurogenic shock).

Tachypnea steadily increases with worsening shock but falls in the pre-terminal phase of shock.

Oliguria is secondary to reduced glomerular filtration and increased filtrate reabsorption. In shock states, the rate of urine production is a useful guide to adequacy of the circulation.

Altered mental status is a common feature of shock as cerebral function is very sensitive to altered O2 delivery.

During shock, mental state progressively changes from anxiety, agitation, confusion and delirium, toward drowsiness and coma. Impaired peripheral perfusion provides a clinically useful clue regarding the likely mechanism of shock.

Cool, clammy peripheries with pale or mottled skin are suggestive of hypovolemic or cardiogenic shock, whereas warm peripheries are suggestive of distributive shock.

INVESTIGATION FOR SHOCK (as clinically Indicated)

Bedside

- Hemoglobin,
- Arterial blood gas.
- Lactate
- ECG
- Ultrasound (e.g. FAST scan, AAA scan),
- Echocardiogram

Laboratory

- Full blood count,
- Coagulation studies,
- D-dimer Electrolytes,
- Creatinine,
- urea, liver function tests Cardiac enzymes, serum lipase

Cultures

- urine,
- blood, sputum,
- pus

Toxicology assays

Radiology

- Chest, abdominal X-ray
- Trauma series radiology chest, pelvis, spinal CT
- Angiography (e.g. coronary, visceral, pulmonary)

MONITORING

All causes of shock have improved outcomes when managed in an environment that closely monitors clinical signs and physiological parameters (i.e., an ICU).

Clinical monitoring involves frequent assessment of **heart rate, blood pressure, respiratory rate, conscious state, urine output, peripheral perfusion and temperature**.

An Arterial Cannula provides beat-to-beat measurement of systemic pressure and is particularly useful for measuring blood pressure when clinical techniques become difficult and unreliable.

Arterial cannula also allows ready sampling for blood gas and lactate measurement.

A Central Venous Cannula allows measurement of central venous pressure (CVP), which is often used as an estimate of venous volume, and hence preload.

However, CVP bears a variable relationship to venous volume, as it is dependent on location of the catheter to the right atrium, intrathoracic pressures, venous compliance, position of the patient and tricuspid valve competence. Thus, CVP is a guide to the pressure status of the venous system rather than a measure of intravascular volume and preload. CVP correlates poorly with fluid response to shock.

Echocardiographic Assessment of end-diastolic ventricle volume may better a predictor of preload than invasive pressure measurement, but the technique is operator- and patient-dependent.

MANAGEMENT

Resuscitation of shock is a medical emergency. The aim of therapy is to restore systemic DO2 rapidly and effectively and improve tissue perfusion.

History, examination, and investigation must occur concurrently with resuscitation. The usual resuscitation principles of airway, breathing, circulation apply.

The principles of management of shock are:

1. **Supply O2**
2. **Vascular access**
3. **Volume resuscitation**
4. **Vasoactive agents**

5. Manage precipitating illness or injury.

6. Monitoring.

Supply oxygen

Ensure any causes of hypoxia are urgently corrected including providing adequate FiO2, ensuring ventilation is adequate and that any reversible cause of pulmonary shunt is corrected (e.g., pleural collection, bronchus obstruction).

Vascular access

Insertion of intravenous wide bore cannula and C.V line is essential for administration of fluids and medications.

Fluid resuscitation

is usually the first therapeutic strategy in the management of shock, particularly in hypovolemic or distributive shock.

However, it is important to note that not all patients will respond to fluid loading with a significant increase in CO. If the heart is working on the terminal (flat) portion of the Frank–Starling curve, increased preload may not result in a significant increase in SV. Nevertheless, even patients with cardiogenic shock may benefit from a judicious fluid challenge and dynamic assessment of volume responsiveness (i.e., the ability to increase CO with fluid loading or straight leg raise) is preferred to static measurements of volume state (e.g., central venous pressure).

A. Trendelenburg

A quick method to increase venous return is to tilt the patient's pelvis above horizontal (i.e., head down). This will 'auto-transfuse' blood from leg and pelvic veins and increases venous filling pressures and MAP. Increases in CO are minimal if venous capacitance remains high and the extra blood volume is accommodated.

B. Crystalloids

Crystalloid solutions comprise electrolytes (with or without dextrose) and water. These fluids cross semipermeable membranes easily and are rapidly distributed through the intravascular and extravascular spaces. 0.9% saline is commonly used for initial volume replacement. 0.9% saline is slightly hyperosmolar (300 mOsm/L) and hyperchloremic (150 mEq/L) relative to plasma.

When large volumes are used for resuscitation, hyperchloremia can contribute to bicarbonate loss and a normal anion gap metabolic acidosis. Lactated Ringer's solution (Hartmann's) is isotonic and contains lactate (29 mEq/L) and electrolytes in a ratio similar to plasma. However, the calcium in Hartmann's (4 mEq/L) is incompatible with certain drugs and lactate levels may rise if hepatic function is markedly impaired or a lot of fluid is administered

C. Colloids

These solutions remain in the circulation for longer, have a smaller volume of distribution and hence are more effective at increasing intravascular volume than the same volume of crystalloid. There are a number of colloid solutions available, which differ based on their type and concentration of colloidal molecules. Albumin, Starch Solutions and Blood products

Vasoactive agents

When fluid administration alone fails to restore adequate oxygen delivery and organ perfusion, vasoactive agents should be initiated. In extreme shock, it may be necessary to commence fluid resuscitation and vasoactive therapy concurrently.

Vasoactive agents are commonly referred to as 'inotropes' as many of them increase cardiac contractility (i.e., inotropy). However, many agents have their primary effect on vascular tone rather than directly altering contractility.

The choice of agent will depend on which aspect of the cardiovascular physiology is deranged and the goals of therapy.

For cardiogenic shock, medications may be required to increase contractility (e.g., dobutamine, milrinone,), reduce afterload, maintain adequate systemic and coronary perfusion pressure, increase diastolic relaxation and increase or decrease heart rate.

In distributive shock, medications that produce venoconstriction and increased systemic pressures are required. There is little role for cardiovascular medications in hypovolemic shock.

The choice of which catecholamine to use in shock (i.e., norepinephrine (noradrenaline) vs epinephrine (adrenaline) vs dopamine) has been the subject of considerable debate and opinion.
Different catecholamines exhibit different pharmacological properties (e.g., $\beta1$- vs $\beta2$ adrenergic receptor stimulation

Manage precipitating illness or injury.

As the circulation is being resuscitated, the cause of the circulatory disturbance needs to be identified and corrected. Unless this occurs, shock will continue to worsen and death will ensue. Time to definitive treatment of the cause of shock is related to survival. This has been clearly illustrated in cardiogenic shock (time to reperfusion), hemorrhagic shock (time to hemorrhage control) and septic shock (time to appropriate antibiotics).

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