

Shock

Consequences of shock

Unresuscitatable shock

Patients who are in profound shock for a prolonged period of time become unresuscitatable and the ability of the body to compensate is lost;

- ✚ There is myocardial depression and loss of responsiveness to fluid or inotropic therapy;
- ✚ Peripherally there is loss of the ability to maintain systemic vascular resistance and no longer respond appropriately to vasopressor agents causing further hypotension;
- ✚ Death is the inevitable result.

This stage of shock is the combined result of the severity of the insult and delayed, inadequate or inappropriate resuscitation in the earlier stages of shock.

Multiple organ failure

This result from prolonged systemic ischaemia and reperfusion injury.

Multiple organ failure is defined as two or more failed organ systems (Table1).

Table 1

Effects of organ failure

Lung	Acute respiratory distress syndrome
Kidney	Acute renal insufficiency
Liver	Acute liver insufficiency
Clotting	Coagulopathy
Cardiac	Cardiovascular failure

Treatment

Resuscitation

Immediate resuscitation manoeuvres for patients presenting in shock are:

- 1) Ensure a patent airway
- 2) Adequate oxygenation and ventilation.
- 3) Cardiovascular resuscitation.

Conduct of resuscitation

If there is initial doubt about the cause of shock, it is safer to assume the cause is hypovolaemia and begin with fluid resuscitation, followed by an assessment of the response.

In all cases of shock, regardless of classification, hypovolaemia and inadequate preload must be addressed before other therapy is instituted.

Administration of inotropic or chronotropic agents to an empty heart will rapidly and permanently deplete the myocardium of oxygen stores and dramatically reduce diastolic filling and therefore coronary perfusion. Patients will enter the unresuscitatable stage of shock as the myocardium becomes progressively more ischaemic and unresponsive to resuscitative attempts.

First-line therapy, therefore, is intravenous access and administration of intravenous fluids. Access should be through short, wide-bore catheters that allow rapid infusion of fluids as necessary.

Type of fluids

Crystalloid solutions (normal saline, Hartmann's solution, Ringer's lactate) are preferred over colloids (albumin or commercially available products) because they are cheap and available.

Most importantly, the oxygen-carrying capacity of crystalloids and colloids is zero. If blood is being lost, the ideal replacement fluid is blood, although crystalloid therapy may be required while awaiting blood products.

Hypotonic solutions (e.g. dextrose) are poor volume expanders and should not be used in the treatment of shock unless the deficit is free water loss (e.g. diabetes insipidus) or patients are sodium overloaded (e.g. cirrhosis).

Dynamic fluid response

In total, 250–500 ml of fluid is rapidly given (over 5–10 min) and the cardiovascular responses in terms of heart rate, blood pressure and central venous pressure (CVP) are observed. Patients can be divided into 'responders', 'transient responders' and 'nonresponders'.

Responders show an improvement in their cardiovascular status, which is sustained. These patients are not actively losing fluid but require filling to a normal volume status.

Transient responders show an improvement but then revert to their previous state over the next 10–20 min. These patients either have moderate on-going fluid losses (either overt haemorrhage or further fluid shifts reducing intravascular volume).

Non-responders are severely volume depleted and are likely to have major on-going loss of intravascular volume, usually through persistent uncontrolled haemorrhage.

Vasopressor and inotropic support

Vasopressor or inotropic therapy is not indicated as first-line therapy in hypovolaemia. Vasopressor agents (phenylephrine, noradrenaline) are indicated in distributive shock states (sepsis, neurogenic shock), in which there is peripheral vasodilatation and a low systemic vascular resistance, leading to hypotension despite a high cardiac output.

In cardiogenic shock or when myocardial depression complicates a shock state (e.g. severe septic shock with low cardiac output), inotropic therapy (dobutamine) may be required to increase cardiac output.

Monitoring**Monitoring for patients in shock****Minimum**

- Electrocardiogram
- Pulse oximetry (O₂ saturation and pulse rate)
- Blood pressure
- Urine output

Additional modalities

- Central venous pressure
- Invasive blood pressure
- Cardiac output
- Base deficit and serum lactate

Cardiovascular

As a minimum, cardiovascular monitoring should include continuous heart rate [electrocardiogram (ECG)], oxygen saturation and pulse waveform and non-invasive blood pressure.

Central venous pressure

A fluid bolus (250–500 ml) is infused rapidly over 5–10 min. The normal CVP response is a rise of 2–5 cmH₂O, which gradually drifts back to the original level over 10–20 min. Patients with no change in their CVP are empty and require further fluid resuscitation.

Patients with a large, sustained rise in CVP have high preload and an element of cardiac insufficiency or volume overload.

Cardiac output

Measurement of cardiac output, systemic vascular resistance and preload can help distinguish the types of shock that are present (hypovolaemia, distributive, cardiogenic), especially when they coexist. The information provided guides fluid and vasopressor therapy by providing real-time monitoring of the cardiovascular response.

Systemic and organ perfusion

- The best measures of therapy remain the urine output; however, this is an hourly measure and does not give a minute-to-minute view of the shocked state.
- The level of consciousness is an important marker of cerebral perfusion, but brain perfusion is maintained until the very late stages of shock and, hence, is a poor marker of adequacy of resuscitation.

Base deficit and lactate

Lactic acid is generated by cells undergoing anaerobic respiration.

The degree of lactic acidosis, as measured by the serum lactate level and/or the base deficit, is a sensitive tool for both the diagnosis of shock and the monitoring of the response to therapy.

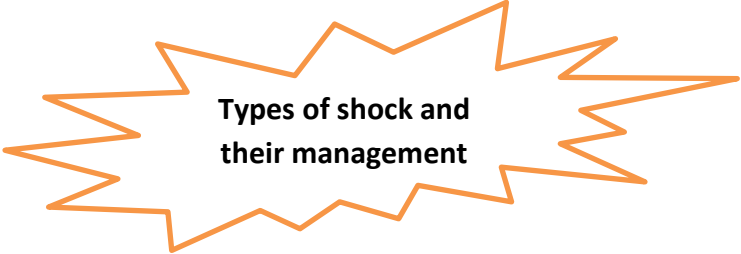
Mixed venous oxygen saturation

The percentage of saturation of oxygen returning to the heart from the body is a measure of the oxygen delivery and extraction by the tissues. Accurate measurement is via analysis of blood drawn from a long central line placed in the right atrium. Normal mixed venous oxygen saturation levels are 50–70%. Levels below 50% indicate inadequate oxygen delivery and increased oxygen extraction by the cells. This is consistent with hypovolaemic or cardiogenic shock. High mixed venous saturation levels (> 70%) are seen in sepsis. In sepsis there is disordered utilisation of oxygen at the cellular level and arteriovenous shunting of blood at the microvascular level. Thus, less oxygen is presented to the cells, cells cannot utilise what little oxygen is presented and venous blood has a higher oxygen concentration than normal.

Patients who are septic should, therefore, have mixed venous oxygen saturation levels of > 70%.

Note

Deal with the cause of the shock: e.g. stop the bleeding, drain the abscess, remove the source of the anaphylactic antigen, etc.



Types of shock and their management

Hypovolaemic shock

- **Treatment.**
 - Lie patient flat; high flow O₂; lift legs to auto-transfuse.
 - Give and repeat fluid infusion 500mL IV rapidly: you should see rise in BP. If no rapid improvement in BP look for other causes.
 - Take blood and send for FBC, U & E, clotting, and cross-match.
 - Take arterial blood gas: estimate Hb, K as well as blood gases.

Anaphylactic shock

- **Causes:** drug allergy, blood product reaction.
- **Clinical features:** history of sudden onset after administration of drug. Stridor or bronchospasm, angioedema, urticaria, pruritus, rash.
- **Treatment.**
 - Sit patient up; give high flow O₂; call anaesthetist if stridor.
 - If IV access: give 1mL of 1:10 000 adrenaline bolus; flush; then 100mg hydrocortisone bolus; flush; then 10mg chlorpheniramine IV.
 - Repeat again in 5-10 min if no improvement.
 - If no IV access: give 1mL 1:1000 adrenaline IM. Then secure IV access.
 - If wheezy give 5mL nebulized salbutamol.
 - IV fluid

Septic shock

- **Cause.** Overwhelming sepsis .
- **Clinical features.** May be the same as hypovolaemic shock or, if established, with circulatory collapse. Earlier in the evolution the patient may look septic pyrexial, flushed, bounding pulses.
- **Treatment.**
 - As for hypovolaemic shock.
 - Take blood cultures; then give IV antibiotics.

Cardiogenic shock

- **Rapidly reversible causes:** cardiac tamponade (trauma, post-cardiac surgery), arrhythmias, tension pneumothorax.
- **Clinical features.** History of recent surgery/trauma, chest pain, dyspnoea, palpitations.

Treatment.

According to the cause:

For heart failure give high flow O₂, 2.5mg morphine IV (anxiolytic, venodilator, analgesic, anti-arrhythmic) put the patient on cardiac monitors; request 12-lead ECG, treat arrhythmias.

For myocardial ischaemia give 0.1mg GTN, 300mg aspirin.

Tension pneumothorax and cardiac tamponade need surgical intervention.

Additional measures include

- Consider central venous and peripheral arterial monitoring.
- Send blood for arterial blood gases, FBC, U & E, clotting, troponin.
- Catheterize the patient.
- Request CXR look for pulmonary oedema.
- Treat fluid overload with diuretics: frusemide 40mg IV.
- Consider transthoracic echo to exclude pericardial effusion and valvular lesions, and to assess LV function.

No more at the time being; may be later on.....

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