Management of Shock

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Shock is defined as acute circulatory failure resulting in inadequate organ perfusion and cellular hypoxia. It includes several subgroups with distinct aetiologies and impaired cardiovascular function. The ultimate end result of shock due to any cause is cellular hypoxia and deranged cell function.

Cardiovascular physiology: It is important to learn the basic cardiovascular physiology in order to understand the pathophysiology of various types of shock. This relationship between cardiac output, blood pressure and systemic vascular resistance can be explained on the basis of Ohm's Law from physics.

Ohm’s Law: \( I = \frac{\Delta V}{R} \) in which current (I) equals the voltage difference (\( \Delta V \)) divided by the resistance (R). This can be considered to cardiovascular physiology where \( I = \) cardiac output, \( \Delta V = \) blood pressure and \( R = \) systemic vascular resistance. Cardiac output, the amount of blood pumped by left ventricle in each minute is determined by the product of stroke volume and heart rate. Stroke volume, the amount of blood pumped in each cardiac cycle is determined by preload, myocardial contractility and afterload.

Preload: It denotes the volume of venous return to the heart which is determined by the venous capacitance, volume status and the pressure difference between mean systemic venous pressure and right atrial pressure. Right atrial pressure is directly affected by intra-thoracic pressure. The volume of venous blood returned to the heart determines the stretch of myocardial muscle fibre.

Myocardial contractility: Frank-Starling law states that the force of contraction of the cardiac muscle is proportional to its initial length. In heart, within physiological limits, an increase in end diastolic produces a more forceful contraction and an increase in stroke volume (SV). If the venous return is reduced then the stroke volume and hence the cardiac output will be reduced.

Afterload: It is the resistance to the forward flow or it is the systemic vascular resistance. For a given stroke volume, increase in afterload results in decreased cardiac output and increased stroke work.
Shock can be classified in to different types depending on the cause of shock.

- Hypovolaemic shock
- Cardiogenic shock
- Obstructive shock
- Anaphylactic shock
- Neurogenic shock
- Septic shock

**Hypovolaemic shock**

In hypovolaemic shock, circulating blood volume is reduced and venous return to the right atrium falls and therefore stroke volume and cardiac output are reduced. Hypovolaemia may result due to blood loss or fluid loss. Blood loss may revealed as external bleeding or may be concealed (eg: retroperitoneal bleeding, bleeding in to the pelvic cavity in pelvic fracture). Fluid loss can be exogenous as it occurs in burns, diarrhoea and diuresis or endogenous where fluid is lost in to the body cavities (eg: intestinal obstruction, peritonitis). Hypovolaemia can also result due to iatrogenic factors such as poor fluid prescription, inappropriate use of diuretics, mechanical bowel preparation and prolonged fasting before surgery.

Compensatory response to blood loss and hypovolaemia involves immediate neural mechanism and delayed humoral mechanism. The immediate neural mechanism involves the baroreceptors in the carotid sinus. Decreased rate of firing from these receptors causes reduced inhibition of vasomotor and cardiac centres in medulla causing increased sympathetic activity which results in systemic vasoconstriction and rise in the diastolic blood pressure. In most cases, tachycardia is the earliest sign of hypovolaemic shock.
The humoral mechanism involves activation of renin-angiotensin system. Renin is produced by juxtaglomerular cells [JG cells](modified cells of afferent arteriole) and also by lacis cells located between the afferent and efferent arterioles. Decrease in pressure at the level of JG cells results in renin release. Renin converts angiotensigen in to angiotensin I, which is then converted into angiotensin II by ACE. Angiotensin II causes vasoconstriction, acts on adrenal cortex to increase the secretion of aldosterone, also increases the synthesis of angiotensinogen. Increased secretion of ADH from posterior pituitary results in sodium and water retention. The other hormones involved in the pathophysiology of shock include adrenocorticotropic hormone, beta-endorphin, endogenous opioid peptides and cytokines.

Compensatory mechanisms tend to preserve venous return and maintain cardiac output during the early stage of shock. At cellular level due to lack of oxygen anaerobic metabolism is initiated which results in lactic acid production and metabolic acidosis. If shock is prolonged cellular integrity is lost and mitochondrial damage soon follows. Lysosomes rupture and release enzymes that digest intracellular structural elements, as a result cellular oedema occurs.

**Haemorrhagic shock (Hypovolaemic shock due to blood loss):** This type of shock is commonly seen in trauma patients and postoperative patients. Haematocrit is unreliable for estimating the degree of acute blood loss. Low haematocrit following acute injury suggests preexisting anaemia or a massive blood loss. Haematocrit concentration falls once the intravascular volume is restored using intravenous fluids and subsequent trend of haematocrit may be useful in assessing blood loss. Haemorrhagic shock can be classified into four classes, based on the volume of blood loss.

- **Class 1 shock** is where patient has lost up to 15% of blood volume; this is equivalent to donating a unit of blood. Patient is unlikely to have any clinical signs at this stage. Replacement of primary fluid loss is only that is required at this stage.

- **Class 2 shock** is where a patient has lost 15 to 30% of blood volume. In a 70 kg man this would represent about 750-1500ml of blood loss. At this stage clinical features such as anxiety, tachycardia, and narrow pulse pressure may be seen. Urine output is slightly reduced but compensatory mechanisms may maintain a normal blood pressure.

- **Class 3 shock** (when 30-40% of blood volume is lost) manifests with classic signs of inadequate perfusion which includes tachycardia, tachypnoea, significant changes in mental status and fall in systolic blood pressure. Blood transfusion is always required with this degree of shock.

- **Class 4 shock** is where a patient has lost more than 40% of blood volume which can be life threatening if not managed immediately. This degree of blood loss will result in significant hypotension negligible urine output and altered level of consciousness. Patient appears pale and skin is cold. Immediate blood transfusion and surgical intervention to control the bleeding is essential.
Recognition and management

The diagnosis and management should go simultaneously in haemorrhagic shock. The basic management principle is to control the bleeding and replace the volume loss.

Initial approach involves airway, breathing and circulation. High concentration oxygen should be administered. A compromised airway and breathing requires definitive airway management and ventilatory support. After managing airway and breathing careful evaluation of patient’s circulatory system is essential for identifying early signs of shock. Tachycardia is the earliest sign. In adult heart rate greater than 100 is considered as tachycardia where as in children it varies with the age. Heart rate greater than 160 in infant, 140 in preschool child, 120 in school age child are considered as tachycardia. Blood pressure may remain normal at initial stages of shock due to vasoconstriction and raised diastolic blood pressure. Monitoring should include blood pressure, pulse, capillary refill, urine out put, level of consciousness, acid-base status, and central venous pressure.

Practical aspects in managing hypovolaemic shock

- Vascular access is best done using two large caliber (size14 G in adults) peripheral intravenous cannulae. The rate of flow of fluid through i.v. cannula is determined Hagen – Poiseuille’s law which states that the rate of flow is directly proportional to the fourth power of the radius, and inversely proportional to the length of the cannula. Hence short wide bore cannula is preferred for rapid transfusion.

- In circumstances where peripheral access is not available or difficult, central venous access (femoral, jugular or subclavian) should be considered.

- In children less than 6 years old, intraosseous route should be considered.

- Soon after gaining i.v. access, blood samples should be collected for type & cross match the blood, full blood count, glucose level, urea and electrolytes. Warm crystalloid solution should be used as initial resuscitation fluid. An initial bolus of 1-2 Litres in adults and 20ml/kg in children should be given rapidly. Response to fluid resuscitation and adequate perfusion of end organs should be assessed by monitoring level of consciousness, urine output and peripheral perfusion.

- If no response after initial fluid resuscitation and for ongoing blood loss, blood transfusion should be considered.

- Senior help should be requested at an early stage.

- In haemorrhagic shock immediate surgical intervention may be required to control haemorrhage.

Central venous pressure and fluid resuscitation

Central venous pressure represents the pressure in the right atrium (normal pressure =3-7 mm Hg). One should remember that CVP is an indirect estimate of left
ventricular end diastolic volume. It is the relationship between the left ventricular end
diastolic volume and stroke volume is more important in determining the cardiac
function. In some patients initial CVP may be high even with hypovolaemia as CVP is
also affected by intra-thoracic pressure. A trend of CVP readings are useful in guiding
fluid resuscitation. Three types responses may be seen for fluid resuscitation.

1. Rapid response: After initial fluid bolus, patient responds rapidly and remains
haemodynamically normal, CVP remains within normal range.

2. Transient response: Patient responds initially with the fluid resuscitation and then
deteriorates. This may be due to ongoing fluid or blood loss and inadequate
resuscitation.

3. No response: This may be due to ongoing severe blood loss and patient requires
surgical immediate surgical intervention to stop the bleeding.

A high CVP may suggest underlying cardiac failure secondary to myocardial injury or
cardiac tamponade. It may be also due to hypervolaemia due to overtransfusion.

**Fluid Challenge:** Fluid challenge involves rapidly transfusing a small amount fluid
(about 250-500 ml in adults) and assessing the response. Fluid challenges may have
to be repeated until the CVP shows a sustained rise and/or the other cardiovascular
parameters return towards normal. Blood transfusion will be required for severe blood
loss.

**Blood transfusion:** The decision to transfuse blood depends on patient’s response and
estimated blood loss. The main aim of blood transfusion is to restore haemoglobin and
oxygen carrying capacity. The type of blood used depends on the urgency of
transfusion. In patients with exsanguinating haemorrhage group O negative blood
should be used. Blood bank can issue group specific blood in 10 to 20 minutes. Fully
cross matched blood is preferred but it can take 45 to 60 minutes to obtain fully cross
matched blood. For stable patients requiring transfusion, cross matched blood should
be used. Autologous transfusion (collecting blood from the bleeding site eg: thoracostomy tube) should be used wherever possible.

Hypothermia should be prevented by warming the blood and intravenous fluids.
Coagulopathy should be managed by replacing appropriate blood products as dictated
by the clotting tests such as prothrombin time, activated partial thromboplastin time,
platelet count and fibrinogen.

**Septic shock**

In septic shock, the presence of sever infection triggers a massive inflammatory
response with systemic activation of leucocytes and release of a variety of potentially
damaging mediators. Common sources of sepsis include abdomen, chest, wounds,
urinary tract and intravascular line. These mediators result in profound vasodilatation,
increased capillary permeability and myocardial depression. Several mediators such as
nitric oxide, bradykinin, histamine, prostaglandins and cytokines (interleukin-1,
tumour necrosis factor and interleukin-6) are involved in the initiation of sepsis.
Haemodynamic changes include severe vasodilatation myocardial depression and
intravascular pooling of blood. Microvascular changes include increased capillary permeability, microembolisation and arteriovenous shunting. There may be primary disturbance of cellular metabolism and cells are unable to use oxygen and as a result oxygen extraction is impaired. Patient will be hypotensive with warm peripheries.

Management of septic shock includes rapid resuscitation to restore oxygenation, appropriate supportive measures, diagnosis and eradication of source of sepsis and judicious use of antibiotics. Aggressive fluid resuscitation and inotropic support is required at early stages to optimise oxygen delivery.

**Inotropic therapy:** Dobutamine has beta adrenergic effects, it increases heart rate, myocardial contractility and cardiac output. It reduces the systemic vascular resistance and has variable effect on blood pressure. Epinephrine has beta adrenergic effects at lower doses resulting in increased cardiac output and alpha adrenergic effects at higher doses results in increased systemic vascular resistance. Norepinephrine increases systemic vascular resistance and increases blood pressure. Low dose steroid therapy has shown benefits in some patients where adrenocortical deficiency can be demonstrated. Intravenous immunoglobins have been used in recent research and are shown to reduce the mortality.

**Anaphylactic shock**

It is due to anaphylactic reaction which is mediated by immunoglobulin E (IgE) antibodies. Activation of mast cells causes release of histamine and serotonin.

Clinical features of anaphylactic shock

*Cutaneous:* Flushing, erythema, urticarial rashes and swelling

*Respiratory:* Bronchospasm, oedema of the glottis and tongue result in airway obstruction and stridor.

*GIT:* Abdominal pain, diarrhoea, nausea or vomiting.

*CVS:* Hypotension, tachycardia and cardiovascular collapse.

Management of anaphylactic shock

- Stop the administration of drug
- Call for help
- ABC approach

Airway: 100% oxygen or high concentration oxygen should be administered. An definitive airway such as orotracheal tube may be required if airway oedema or if laryngeal oedema is suspected.

Circulation: Venous access should be secured using wide bore i.v. cannula and a liter of crystalloid should be transfused rapidly.

Drugs: Adrenaline 100mcgs (1 ml of 1: 10,000 epinephrine) should be given intravenously. If i.v. route is not available then 0.5 mg (0.5 ml of 1:1000 epinephrine) should be given intramuscularly.

- Secondary management includes bronchodilators, antihistamines and steroids.

**Cardiogenic shock**
In cardiogenic shock cardiac output falls due to the pathology in the heart itself and is defined as cardiac index less than 1.8 L/minute/m$^2$. (Cardiac index is cardiac output per meter of body surface area)

Causes of cardiogenic shock include
- Myocardial infarction, myocarditis
- Arrhythmias
- Cardiac tamponade
- Tension pneumothorax
- Acute aortic incompetence
- Left ventricular aneurysm

Management includes supportive measures, oxygen therapy, judicious use of fluids with careful monitoring of central venous pressure or pulmonary wedge pressure. Other monitoring should include continuous ECG, 12 lead ECG, urine output, urea and electrolytes and blood gases. Patient should be preferably managed in coronary care unit. Morphine 5 -10 mg i.v helps to relieve pain and anxiety associated with myocardial infarction. Inotropic support, vasodilators and mechanical circulatory support may be needed. Further management depends on the specific cause of cardiogenic shock.

**Special considerations in the diagnosis and management of shock**

**Age:** Elderly patients' exhibit reduced sympathetic activity; hence they cannot compensate well for hypovolaemia. Due to atherosclerosis many vital organs are very sensitive to reduced blood flow. They also have reduced respiratory reserve which limits the ability to meet the increased oxygen demand. Reduced renal function reduces the ability to preserve volume.

**Pregnancy:** It is a hypervolaemic state; hence the clinical signs may not be manifested till a large volume of blood has been lost. Hypovolaemic shock can result in decreased fetal perfusion.

**Drug history:** Beta blockers and calcium channel blockers can obtund the compensatory responses to hypovolaemia. Any drug that has significant effect on myocardial contractility, heart rate and peripheral vascular tone can alter the response to shock.

**Pacemaker:** Patients on pacemaker with fixed heart rate unable to respond to hypovolaemia.

**Further reading**


Advanced Trauma Life support for Doctors; Student course manual, 6th edition, 1997.