SHOCK

The shock is a condition of generalized inadequate blood flow, inadequate tissue perfusion, and inadequate removal of cellular waste products. It can be defined in many ways.

The shock is a clinical condition characterized by cardiovascular collapse, inadequate peripheral circulation, tissue perfusion and oxygenation. It occurs whenever the cardiac output fails to maintain adequate circulation.

The shock is a condition in which patient has tachycardia and is pale, ashen in color and is sweating. It may also be characterized by hypotension, peripheral cyanosis, hyper-ventilation, clouding of conscious level and oliguria.

Tissue damage occurs due to hypoxia and lack of nutrients. Multiple organ function deterioration or failure occurs due to the disturbance of cellular function. All these changes lead to progressive shock and death of the patient.

ETIOLOGY AND TYPES OF SHOCK

The basic patho-physiologic derangement in shock is the imbalance between the supply and requirement of the oxygen at the cellular level. This imbalance leads to anoxic changes in the cellular metabolism due to insufficient tissue perfusion.

The anaerobic glycolysis leads to:

- Accumulation of excess lactic acid.
- Inadequate carbon dioxide removal.
- Accumulation of carbonic acid.

These changes lead to cellular and generalized metabolic acidosis.

Multiple and variable factors may affect individually or in combination and lead to following abnormalities and shock:

FAILURE OF PUMP

It is the basic cause of shock in following clinical conditions of inadequate cardiac output;

- Myocardial infarction.
- Toxic states of heart.
- Severe valvular dysfunction.
- Arrhythmias.

85% of the patients suffering from this problem usually have a fatal ending.

FAILURE OF VOLUME

Inadequate effective vascular volume is the basis of this type of shock. It is seen in following clinical conditions;

- Diminished blood volume (hemorrhage)
- Dehydration (Excessive sweating, Diarrhoea, Vomiting)
- Third space fluid loss (Pancreatitis)

FAILURE OF RESISTANCE

The shock is due to failure or decrease in the vascular tone and is seen in the following conditions;

- Septicaemia etc.
Various stages of shock include;

**COMPENSATED OR NON PROGRESSIVE SHOCK**
It is the type of shock when normal compensatory mechanisms reverse the shock and patient achieves full recovery without any exogenous help.

Following factors play a significant role in managing the shock by body's own compensatory mechanisms;

- Baro-receptor reflex.
- CNS ischemic stimulus.
- Reverse stress relaxation of circulatory system.
- Formation of angiotensin.
- Formation of ADH or vaso-pressin.
- Mechanisms such as absorption of fluid and electrolytes and maintenance of urinary output.

**PROGRESSIVE SHOCK**
It is the type, when the shock becomes worse and eventually results in death. There is decrease in coronary blood flow and decrease in cardiac output.

**IRREVERSIBLE SHOCK**
It is the end stage of progressive shock (shock of shocks). Different types of shock depending upon primary etiological factors are as following:

**HEMORRHAGIC SHOCK (ACUTE LOSS OF BLOOD)**
This is the type of shock which occurs following sudden and significant loss of blood.

Sudden loss of 35% to 45% of blood brings the cardiac output and blood pressure to zero.

Sudden loss of 33% of blood volume may cause death.

Smaller percentage of loss of blood volume and reduction in the cardiac output tends to lower the blood pressure and increase the heart rate.

Hemorrhagic shock encephalopathy is highly fatal and it is common in infants. It presents with following features;

- Convulsions, progressive coma, hyperpyrexia, shock, generalized bleeding tendency, abnormal hepatic and renal functions and metabolic acidosis.

Autopsy shows cerebral edema, petechial hemorrhages of gut, lungs and kidneys and generalized depletion of lymphocytes in lymphoid tissues.

Significant unfavourable signs of haemorrhagic shock are;

- Increase in pulmonary resistance.
- Decreased pulmonary blood flow.
- Ventilation, perfusion disturbances preceding onset of circulatory decompensation.
- Congestive process in lungs.
- Reduction of microvascular bed.
- Shock inhibits markedly the myelopoietic response.
- Severe and prolonged hemorrhagic shock leads to lack of microvascular blood flow.
Laser Doppler flowmetry (LDF) is a useful technique for repeated assessment of microvascular blood flow.

Resuscitation with Ringer’s lactate does not restore microvascular blood flow although central venous pressure may be double the normal value.

Decreased microvascular blood flow is the basis of multi-organ failure in prolonged shock.

Antishock clamp (External fixator) provides direct reduction and compression of pelvic fracture diastases about the sacroiliac joint.

It rapidly stabilizes the posterior pelvic ring in hypotensive and shocked patients. It is new way of control of haemorrhage in specific conditions.

SEPTICAEMIC SHOCK (OVERWHELMING INFECTION) TOXIC SHOCK SYNDROME (TSS).
This occurs due to overwhelming infections. Gas gangrene also causes shock like condition due to powerful exotoxin. Staphylococcal toxins can also cause shock.

Endotoxin, specially of coliform bacilli leads to generalized Schwartzman reaction leading to shock. It is also known as endotoxic shock. It is due to generalized vasodilatation as well as loss of fluid in the interstitial spaces.

BURN SHOCK (LOSS OF PLASMA)
This is produced in extensive burns and is very similar to the haemorrhagic shock in the early period.

Early clinical picture is very much misleading as patients with severe burns may not be desperately ill one hour after the burn accident. But 8-10 hours later they may be in irreversible shock due to massive loss of fluids.

It is true that severity of the shock is proportional to the percentage of area burnt.

In children, if the burnt area is more than 10%, intravenous fluid therapy should be provided and the patient should be hospitalized.

In adults same should be done if the burns are more than 15% of the total body surface area.

The fluid loss is about the volume of whole plasma in the patient with 50% burnt area. This leads to severe reduction in circulatory volume and hypovolemic shock if treated inadequately.

Later on (a week after burn accident) wounds may get infected and patient is likely to develop septicaemic shock.

HYPOVOLEMIC SHOCK (LOSS OF FLUID AND ELECTROLYTES)
This may be the result of excessive loss of fluids and inadequate replacement as in:
- Excessive sweating.
- Vomiting.
- Diarrhoea.

Infants are more vulnerable to this problem as they are unable to produce more concentrated urine. They cannot save more fluids. They have smaller fluid compartment. Their compensatory mechanisms are poor and under developed.
CARDIOGENIC SHOCK (MYOCARDIAL INFARCTION)
Myocardial infarction produces shock similar to the hypovolemic shock. The cardiac output is reduced due to pump failure. It leads to reduction in tissue perfusion.

Cellular hypoxia leads to metabolic acidosis which causes peripheral vasodilatation and pooling of the blood. This makes shock even worse.

Failing heart further deteriorates this situation because failure of the pump action deteriorates progressively.

PSYCHOGENIC SHOCK (SUDDEN FRIGHT AND PAIN)
This is the type of shock which follows sudden fright or severe pain. The patient may remain in shock and unconscious for few moments. Even death may occur in worst cases.

NEUROGENIC SHOCK
This type of shock occurs in patients with sudden loss of vasomotor tone. It leads to massive dilatation of veins. It leads to pooling of the blood into veins thus reducing the peripheral resistance, effective circulatory volume and adequate venous return.

The mean systemic filling pressure is reduced and patient suffers from neurogenic shock.

It is seen in patients after deep general anaesthesia when there is collapse of vasomotor center.

Spinal injuries and spinal anaesthesia may block the sympathetic outflow and thus cause vasomotor collapse.

Brain trauma or cerebral hypoxia also leads to vasomotor center depression and thus dilatation of peripheral veins which in turn result in neurogenic shock.

VASO-VAGAL SHOCK (EMOTIONAL FAINTING)
This condition is seen in emotional states when there is vascular dilatation due to sympathetic signals and strong activation of parasympathetic system causing bradycardia.

Both these features lead to decreased cardiac output, decreased venous return and decreased blood pressure.

This leads to the sudden pooling of blood into the limb muscle vessels and dilatation of the splanchic bed. There is loss of effective circulatory volume. It leads to hypotension and unconsciousness.

ANAPHYLACTIC SHOCK (ANTIGEN ANTIBODY REACTION)
Many drugs and substances may cause this type of shock. Penicillin is notorious for causing anaphylaxis.

This is caused by the combination of antigen with the immunoglobulin E (IgE) leading to release of large amount of histamine, histamine-like substances and slow-release-substance-anaphylaxis (S.R.S.A).

These lead to bronchospasm, laryngeal oedema, respiratory depression, hypotension and shock.

These substances also cause sudden;
• Generalized venous dilatation.
• Dilatation of arterioles.
• **Increased capillary permeability.**

These changes decrease the venous return drastically and suddenly. It leads to severe shock and death may occur very quickly.

**STATE OF SHOCK**
It is similar to end stage of irreversible shock. This condition frequently precedes death after different conditions regardless of the cause as electrocution, drowning and massive total body irradiation.

In this condition, there is depletion of high energy phosphates from the cells. ATP changes to ADP and AMP and then to adenosine which leaves the cell and is converted into uric acid.

Once depleted, these stores can never be replenished. It leads to death of cells, organs and the persons.

**PATHOLOGY OF SHOCK**
Most of the changes are due to inadequate perfusion and inadequate removal of cellular waste products of the tissues.

Various compensatory mechanisms modify these changes.

The initial changes are at the cellular level. Inadequate perfusion and hypoxia causing mitochondrial swelling or damage. This damage is seen maximally in the tissue at venous end of the capillaries.

These changes tend to depress almost all the cellular functions. The cellular anoxia is made worse by depressed circulation. There is anaerobic glycolysis leading to accumulation of lactic acid.

There is inadequate removal of carbon dioxide. There is excessive production of carbonic acid. It causes cellular acidosis and then acidemia.

All these changes depress cell metabolism and lower the body temperature. The damaged cells release enzymes which are toxic on their own or activate other blood constituents which are harmful to the body such as conversion of fibrinogen which causes disseminated intravascular coagulopathy (D.I.C).

The other substances increase capillary permeability and thus cause loss of fluids. This leads to reduction in effective vascular volume. It causes lowering of blood pressure.

The capillaries have no muscles in their walls and are controlled by pre and post-capillary sphincters.

The normal transit time of red cells through capillaries is about one second. Low circulatory volume increases this period and causes stagnation and anoxia.

Adequate flow through capillaries is extremely important while treating the shock. The venous constriction while arterioles are relaxed leads to the pooling of blood in the capillaries. This is seen in shock due to myocardial infarction and septicemia.

There is diminished carbohydrate metabolism and reduced energy
production during shock. This leads to metabolic acidosis due to under perfusion of cells.

In shock, the problem is not only reduced circulatory volume but reduced flow of the blood as well.

This is also due to clumping and aggregation of the red cells. It is called “SLUDGING” and this process can be reversed by infusing low molecular weight dextran and albumin.

**REVERSIBLE SHOCK**

This is the type of shock described according to the clinical presentation. This may be encountered in any kind of shock.

In patients with impending shock, the heart rate increases and tries to improve the cardiac output. Release of catecholamines and steroids in response to stress of injury or infection increases peripheral resistance leading to improvement in blood pressure.

Auto regulation mechanism shifts the blood to brain and heart from less vital organs like skin and muscles.

Blood pressure improves, pulse rate becomes normal and tissue perfusion shows improvement. This is why this stage is called reversible shock.

**IRREVERSIBLE OR PROGRESSIVE SHOCK**

It is the end stage of progressive shock. The body compensatory mechanisms fail or are inadequate and patient suffers from state of shock. It occurs when the blood pressure continues to lower down despite treatment. Once the critical level is reached, the shock becomes progressive (shock causes more shock and a vicious circle starts) until death. All methods of known therapy fail to reverse the shock.

The patient’s condition deteriorates and death occurs. This occurs due to continuous under-perfusion of vital organs leading to irreparable damage. This is usually due to inefficient treatment or with severe injuries or when patients have severe infection.

**CLINICAL FEATURES**  

**PALLOR**

The patient looks pale, ashen gray, lethargic and ill. The luster or shining look is lost.

**SWEATING**

There is sweating but it is different than the sweating during hot weather because patient is cold and ashen in colour and has earthy gray looks.

**PALPITATION**

This is an early sign when the heart rate is fast and patient is anxious. One should diagnose and treat shock adequately and actively at this stage and should not wait for other manifestations of shock to appear.

**RESTLESSNESS**

The patient complains of being unwell and is restless and irritable.

**AIR HUNGER**

The patient has hyperapnoea and air hunger. This is due to increased physiological dead space due to reduction in the alveolar perfusion and metabolic acidosis.
THIRST
The patient usually complains of severe thirst and continue asking for drinks. Patient has dry mouth and depressed salivary secretion.

This is due to hypovolaemia and reduced blood supply of the salivary glands leading to compensatory diversion of blood to the brain.

CONFUSION
The conscious level deteriorates in patients with shock. They are confused, disorientated and fail to concentrate.

All these changes of depressed cerebral functions are due to cerebral hypoxia.

HYPOTENSION
Hypotension develops later when body compensatory mechanisms have failed either temporarily or permanently. This is due to reduction in effective circulatory volume.

OTHER SYMPTOMS
- Rigors.
- Vomiting.
- Nausea.
- Diarrhoea.
- Fever.

These symptoms are seen in septic and other types of shock.

CYANOSIS
This is one of the later manifestations of the shock when there is stagnation and pooling of blood and marked reduction in oxygenation.

OLIGURIA OR ANURIA
The urinary output is depressed. This is due to the compensatory diversion of blood from the kidneys to the brain and the heart.

Later on, it may be due to continuous anoxia of the kidneys leading to acute cortical necrosis and acute renal failure.

Improvement in urinary output is a good indication of adequate treatment of shock.

TREATMENT OF SHOCK
The basic objective is restoration of adequate tissue perfusion as early as possible.

The treatment should be started on anticipation of shock before its features are even manifested. Unless the shock is treated actively and efficiently, patients may go into irreversible shock and may die.

RESTORATION OF BLOOD VOLUME
This is of primary importance because only after restoration of blood volume, the cardiac output, peripheral vaso-constriction and compensatory diversion of blood to brain and heart will reverse to normal.

Although these compensatory mechanisms keep the blood pressure as near to normal as possible, their prolonged presence leads to damage to many vital organs like kidneys.

Blood transfusion or other appropriate fluids should be given as fast as possible to replace the lost amount of blood volume which should be normalized quickly.

FLUID AND ELECTROLYTES
When the blood is not available or patient has lost more fluids and
electrolytes, these should be replaced at fast speed. The central venous pressure monitoring should be done to avoid the sudden circulatory overload.

The estimation of left atrial pressure or pulmonary wedge pressure is thought to be better indicator of left heart function than central venous pressure which is a reasonable indicator of right heart function.

MAINTENANCE OF RENAL FUNCTION
Renal function improves after adequate replacement of the fluids. If there has been delay in the treatment of shock, irreversible renal damage may lead to death of the patient.

Renal dialysis may help in reversing the acute tubular necrosis.

REVERSAL OF SLUDGING
Adequate fluids, low molecular weight dextran (Rheomacrodex) and albumin lead to reversal of aggregation of the red cells.

This helps in the improvement of capillary blood flow and improvement of tissue perfusion.

CORRECTION OF METABOLIC ACIDOSIS
The metabolic acidosis is reversed once adequate perfusion of the tissues and adequate oxygenation at cellular level is maintained. The accumulated lactic acid is metabolized to CO$_2$ and H$_2$O in the presence of oxygen.

The role of sodium bicarbonate is controversial in the management of acidosis due to shock. If at all it is to be given, it should be infused after correction of volume deficit.

CORRECTION OF PULMONARY VENTILATION
Improvement in the vascular volume and clearance of the airways helps in the improvement of pulmonary ventilation.

Inhalation of 30% oxygen is also helpful for better diffusion of gases.

DRUGS
In shocked patient, the drugs should be given by intravenous route and through larger veins otherwise there is delay in the absorption and desired effects are not achieved.

On the other hand repeated administration of the drugs and delayed absorption leads to undesirable accumulative toxic effects at the later stage.

Vasoactive drugs can be given to raise the blood pressure for a limited period but only after volume restoration as these drugs will further compromise tissue perfusion in presence of hypovolemia. These should be monitored very carefully.

Steroids have got very important role in the management of endotoxic shock. Larger bolus doses given intravenously are of great help. These should not be used repeatedly because their repeated use may mask the actual picture of the shock.

The vaso-dilator drugs can be used to reduce vaso constriction, (phenoxym benzamine 1 mg/kg intravenously over 24-48 hours) if the adequate fluid replacement fails to improve the tissue perfusion.
These should be used with great care and continuous monitoring of the central venous pressure is essential as vaso-dilatation may lead to further reduction of the blood pressure in already shocked patient.

**DIGITALIZATION**
It is required in patients in whom central venous pressure rises above 10-12 cm of water without restoration of blood pressure after adequate fluid replacement.

**GLUCOSE INSULIN INFUSION**
Intravenous infusion of glucose insulin drip helps in re-entry of potassium into the cells and improvement of the cellular perfusion.

**TEMPERATURE**
Hypothermia as well as hyperthermia are to be avoided in a patient of shock.

Room temperature should be comfortable and the fluid to be infused should be of the same temperature.

Higher temperature will lead to vaso-dilatation and reduction in effective vascular volume while hypothermia interferes with many body mechanisms including coagulation.

**ANTIBIOTICS**
The role of antibiotics is limited to septic shock.

The antibiotics should be used to cover all the infecting organisms. If the culture and sensitivity of the infecting organism is available, proper antibiotics should be started immediately, otherwise after collecting the specimen for culture, a combination of antibiotics covering all types of organism should be given.

Usually triple regimen of antibiotics is used to cover all possible bacteria both aerobes and anaerobes.

One should be careful in using antibiotics antagonizing each others actions.

**TREATMENT OF THE CAUSE OF THE SHOCK**
The injuries causing haemorrhagic shock or infected focus causing septicaemic shock should be properly treated.

If any complication has occurred due to injuries or infection, this should be treated as well.
REFERENCES


The author(s):
Muhammad Shuja Tahir
FRCS, FCPS
is Professor of Surgery at Independent Medical College Faisalabad.
editor@theprofesional.com

Abid Rashid, FCPS
is associate professor in department of Surgery at Independent Medical College Faisalabad.