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Shock - A Review

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INTRODUCTION:

Shock is a life threatening condition characterized by poor tissue perfusion with impaired cellular metabolism, manifested in turn by serious physiological abnormalities.

- The term initial or primary shock is used for transient and usually a benign vasovagal attack resulting from sudden reduction of venous return to the heart caused by neurogenic vasodilatation and peripheral pooling of blood.
- Clinically patients of primary shock suffer from attack lasting few seconds or minutes and develop brief unconsciousness, weakness, sinking sensation, pale and clammy limbs.

TERMINOLOGIES:

CVP

- The **central venous pressure** (**CVP**) is the **pressure** measured in the **central** veins close to the heart. It indicates mean right atrial **pressure** and is frequently used as an estimate of right ventricular preload.
- The central venous pressure (CVP) measures the filling pressure of the right ventricular (RV); it gives an estimate of the intravascular volume status and is an interplay of the (1) circulating blood volume (2) venous tone and (3) right ventricular function.
- **Normal range** for **CVP** is 2-8 cm H20 or 2-6 mmHg. Measured by seldinger technique.
- The response of central venous pressure to a small fluid challenge (200ml of crystalloid) helps in distinguishing between cardiogenic and hypovolemic shock.

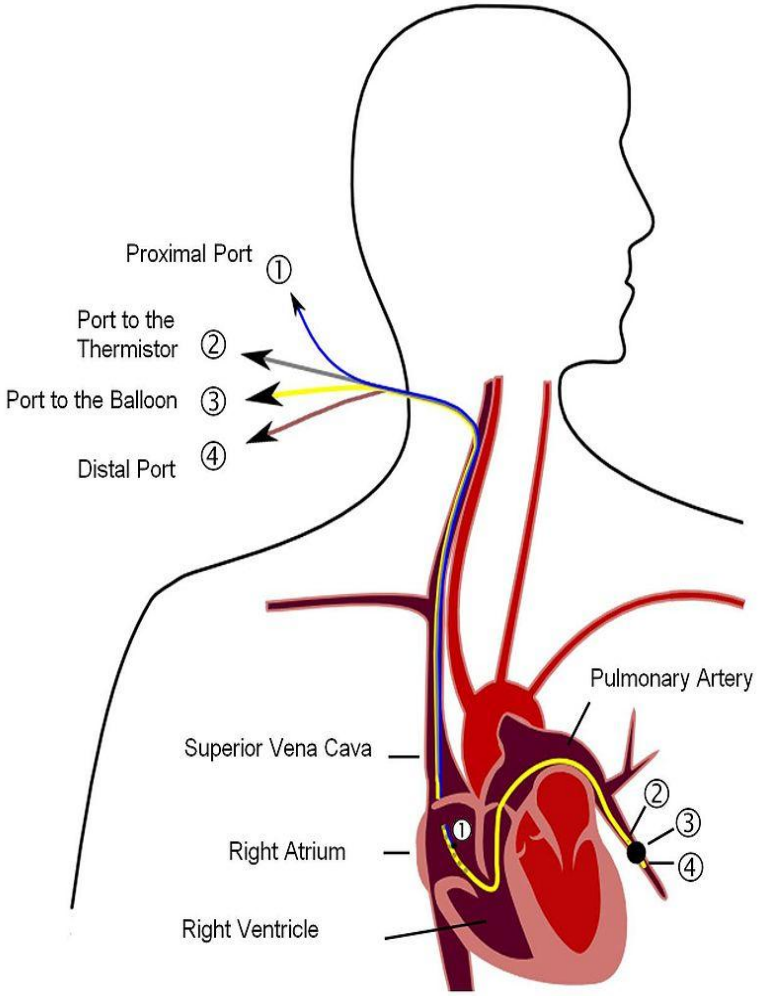
"Central Venous Catheter Physiology"

PCWP

- The pressure measured by wedging a pulmonary catheter with an inflated balloon into a small pulmonary arterial branch.
- Because of the large compliance of the pulmonary circulation, it provides an indirect measure of the left atrial pressure.
- For example, it is considered the gold standard for determining the cause of acute pulmonary edema. It has also been used to diagnose severity of left ventricular failure and mitral stenosis, given that elevated pulmonary capillary wedge pressure strongly suggests failure of left ventricular output.

Postgrad. med. J. (August 1969) 45, 506-511.

- Better indicator of both circulating blood volume and left ventricular function obtained by a pulmonary artery flotation ballon catheter.
- It is used to differentiate between left and right ventricular failure, pulmonary embolus, septic shock and ruptured mitral valve.
- It is used to measure cardiac output.



CLASSIFICATION AND ETIOLOGY OF SHOCK

1) Hypovolemic shock

- Acute haemorrhage
- Dehydration from vomitings, diarrhoea
- Burns
- Excessive use of diuretics
- Acute pancreatitis

2) CARDIOGENIC SHOCK

1) Deficient emptying

- Myocardial infarction
- Cardiomyopathies
- Rupture of the heart, ventricle, papillary muscle
- Cardiac arrhythmias

2) Deficient filling

- Cardiac tamponade from haemopericardium

3) Obstruction to the outflow

- Pulmonary embolism
- Ball valve thrombus
- Tension pneumothorax
- Dissecting aortic aneurysm

3) SEPTIC SHOCK

- Gram-negative septicaemia e.g. Infection from E.coli, Klebsiella, Pseudomonas
- Gram-positive septicaemia e.g. Infection with streptococci, pneumococci

4) OTHER TYPES

1) Traumatic shock

- Severe injuries
- Surgery with marked blood loss
- Obstetrical trauma

2) Neurogenic shock

- High cervical spinal cord injury
- Accidental high spinal anaesthesia
- Severe head injury

3) Hypoadrenal shock

- Administration of high doses of glucocorticoids
- Secondary adrenal insufficiency

PATHOGENESIS OF SHOCK

All forms of shock involve following derangements:-

a) Reduced effective circulating blood volume

b) Reduced supply of oxygen to the cells and tissues with resultant anoxia.

c) Inflammatory mediators and toxins released from shock induced cellular injury.

TYPES OF SHOCK

1) VASO-VAGAL SHOCK

- It is brought about by pooling of blood in larger vascular reservoirs and by dilatation of the arteriolar bed causing reduced venous return to the heart, low cardiac output and reflex bradycardia.
- The reduced cerebral perfusion causes cerebral hypoxia and unconsciousness but prostration and reflex vasoconstriction increases the venous return and cardiac output to restore cerebral perfusion and consciousness.

It must be remembered that if the patient is maintained in upright position as in a dental chair, it will cause permanent cerebral damage.

2) HYPOVOLEMIC SHOCK

It is due to loss of intravascular volume by haemorrhage, dehydration, vomiting and diarrhoea. It mostly occurs from the systemic venules and small veins which usually contain about 50% of total blood volume.

- Loss of blood causes decreased filling of the right heart.
- This causes decrease of filling of pulmonary vasculature.
- Decreased filling of the left atrium and ventricle.
- Drop in the arterial blood pressure.

COMPENSATORY MECHANISMS

The following compensatory mechanisms come into play:-

a) Adrenergic discharge

- It starts within **60 secs** after the blood loss.
- It causes vasoconstriction of the venules and small veins, increase heart rate and also constricts the vascular sphincters in the kidney, skin etc.
- This constriction displaces the blood into the right atrium and ventricle increasing the diastolic pressure in the right ventricle and also its stroke volume.
- This selective vasoconstriction leads to increase in filling in right heart and cardiac output.
- Diverting the blood to the heart and brain.

Hyperventilation

- This occurs in response to metabolic acidemia which develops shortly after haemorrhage.
- Spontaneous deep breathing sucks blood from extrathoracic sites to the heart and lungs.
- This leads to increase in the filing of left ventricle and also the stroke volume.
- Both adrenergic discharge and hyperventilation occur within one minute of blood loss.

Release of vasoactive hormones

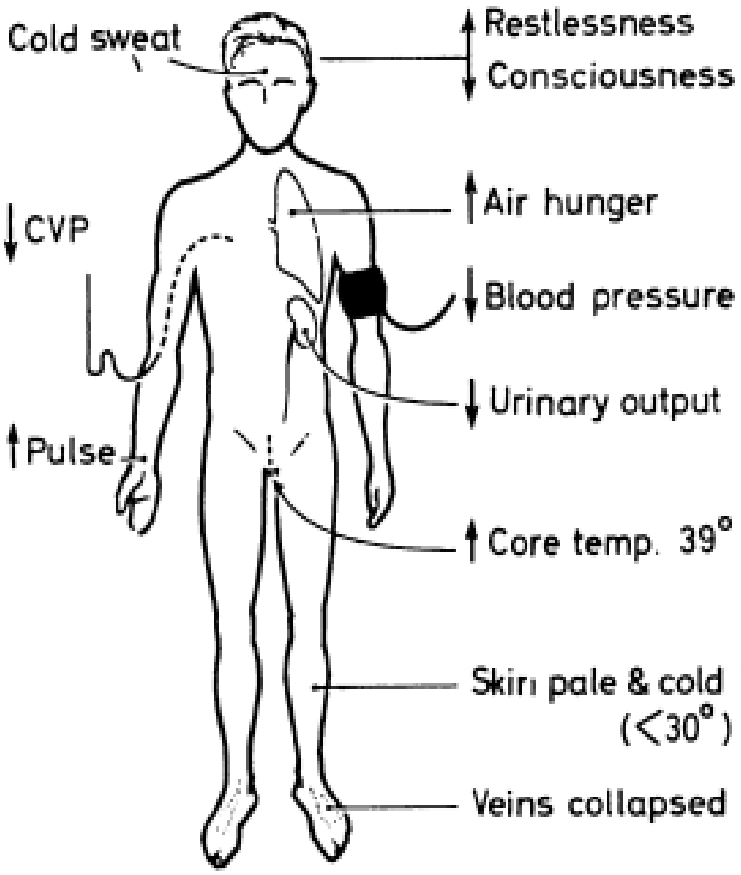
- Low perfusion of kidneys leads to release of hormone known as renin from juxta-glomerular apparatus.
- Renin releases angiotensin I from liver, which is converted to angiotensin II by the lungs.
- Angiotensin is a potent vasoconstrictorleading to constriction of vasculature of splachnic organs, kidneys and skin.

Another potent vasoactive hormone is epinephrine which is released from adrenal medulla as a conequence of discharge of adrenergic nervous system.

Collapse

Assumption of recumbent posture due to collapse automatically displaces blood from the lower part of the body to the heart and increases the cardiac output.

CLINICAL FEATURES OF SHOCK



CLINICAL FEATURES OF HYPOVOLEMIC SHOCK

1) MILD SHOCK

- Loss of less than 20% of blood volume leads to mild shock.
- The most sensitive clinical finding is due to adrenergic constriction of blood vessels in the skin.
- The result is the collapse of sub-cutaneous veins of the extremities, particularly the feet which become pale and cool.

- There is sweat in the forehead, head and feet due to adrenergic discharge.
- Urinary output, pulse rate and blood pressure at this stage are normal. The patient feels thirsty and cold.

2) MODERATE SHOCK

- It involves loss of 20-40% of blood volume. Alongwith the findings mentioned above, there will oliguria.
- This oliguria is because of adrenergic discharge alongwith the effects of circulating aldosterone and vasopressin.
- The pulse rate is increased but is less than 100 beats per minute.

3) SEVERE SHOCK

- Loss of blood volume more than 40% causes severe shock.
- There is pallor, low urinary output, rapid pulse and low blood pressure.

CLINICAL MONITORING

- Once the shock is diagnosed, constant monitoring of the patient is required to assess the degree of blood loss and hemodynamic impairment.

1) BLOOD PRESSURE

- The diastolic pressure is the main indicator of the degree of vasoconstriction. The systolic pressure indicates vasoconstriction with stroke volume and rigidity of vessels.
- The pulse pressure indicates the stroke volume and the cardiac output.

2) RESPIRATION

- Hyperventilation is a normal response of an early shock. If the patient is not hyperventilating, he is surely suffering from central nervous system or respiratory damage.
- Persistent hyperventilation is an ominous sign and indicates treatment of shock.

3) URINE

- Urine output is a good indication of severity of shock. Urine output is affected quite early even in moderate shock. It is also a good index of adequacy of replacement therapy.

4) CVP

- Measurement of Central Venous Pressure is quite important in assessing shock. In hypovolemic shock, the blood volume is decreased, so is the CVP, whereas in cardiogenic shock there is no depletion of blood volume and the CVP remains normal.

5) E.C.G.

- In severe shock electrocardiogram may show signs of myocardial ischemia with depression results are less accurate than the above method.

6) PULMONARY CAPILLARY WEDGE PRESSURE

- It is a better indicator of circulating blood volume and left ventricular function. If the catheter in a portion of the lung where inflation of the lung occludes the pulmonary capillaries the end of catheter estimates the pressure in the alveoli rather than pressure in left atrium.
- This catheter is used to differentiate between left and right ventricular failure, presence of pulmonary embolism and can also be used as a guide to therapy with fluids.

TREATMENT:

1) RESUSCITATION

This should began immediately as the patient shows the signs of hypovolemic shock. This starts with the establishment of a clear airway and maintaining adequate ventilation and oxygenation.

Lowering of head with support of the jaw to prevent airway obstruction and administration of oxygen are usually all that are needed.

Lowering of the head will improve venous return preventing stasis of blood in the muscles of lungs and preventing edema.

This also improves the cerebral circulation which is quite important at this stage.

Many patients in shock, particularly those who are suffering from traumatic or septic shock require intubation and positive-pressure ventilation.

- Positive pressure ventilation improves the patient's cardiovascular status. Abrupt increase in airway pressure expands the alveoli, displaces blood from pulmonary vasculature into left atrium and ventricle.
- Both the left ventricular output and systemic arterial pressure increase.

2) IMMEDIATE CONTROL OF BLEEDING

This can be achieved by raising the foot-end of the bed and by the compression bandage to tamponade external haemorrhage.

Operation may be required to stop such bleeding as soon as resuscitation has been achieved.

3) **EXTRACELLULAR FLUID REPLACEMENT**

Fluid replacement should be started following the control of bleeding immediately.

A non-sugar, non-protein crystalloid solution with a sodium concentration that of plasma is preferable in the initial stages of fluid replacement.

The solution can be Ringer's lactate, Ringer's acetate or normal saline.

- The solution is run at a speed of 45 minutes between 1000-2000 ml solution is given intravenously. It is often observed that the blood pressure will come back to normal and become stable after infusion of 1 or 2 litres of such solution.
- Resuscitation should always be started with crystalloid solution even if blood is available. If it is started with acidotic cold bank blood with potassium concentration, efficiency of myocardium is tremendously jeopardized.

- 3 litres of fluid given over 45 minutes should resuscitate any patient with arrested haemorrhage.
- The need for more fluid indicates continuation of bleeding and such haemorrhage should be controlled surgically.

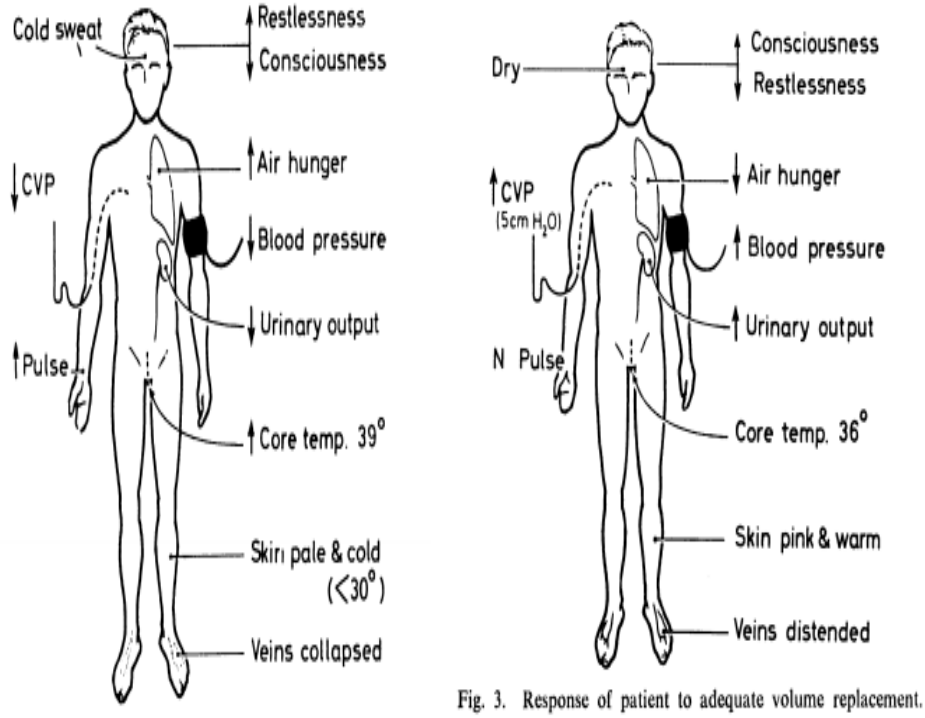


Fig. 3. Response of patient to adequate volume replacement.

4) **DRUGS**

Few drugs are used sometimes in different types of shock:-

1) Sedatives

These are used to alleviate pain and some amount of sedation is always required in any type of shock.

2) Chronotropic drugs

The patients in shock who have slow heart rate benefit from chronotropic drugs as these increase heart rate.

Atropine is the most widely used drug in this group, followed by isoproterenol.

These also act as vasodilators of systemic arterial and capillary sphincters.

3) Ionotropic drugs

These drugs improve the strength of cardiac muscle contraction.

Patients with cardiogenic and severe septic shock requirethis drug especially.

The most commonly used are dopamine and dobutamine.

These lower the myocardial contractility and increase the renal blood flow by dilating the renal vasculature.

4) Vasodilators

The most commonly used are nitroprusside and nitroglycerine as these are reversible and short acting.

These are used when systemic vascular resistance is too much raised.

5) β- blockers

Patients in cardiogenic shock with stiff myocardium and rapid heart rate are benefitted.

The drug increases the efficiency of ventricular contraction.

Propranol is the most widely used drug in this group.

6) Diuretics

These drugs reduce the vascular volume and decrease filling pressure.

Though oliguria is one of the main clinical manifestations of hypovlemic shock, yet diuretics will not correct the underlying cause but will further cause hypovolemia.

These are also not used in septic shock.

TRAUMATIC SHOCK

- It is primarily due to hypovolemia from bleeding external and internal wounds.
- The peculiarity of this shock is that traumatised tissues activate the coagulation system and release microthrombi into the circulation. These may occlude or constrict parts of pulmonary microvasculature to increase pulmonary vascular resistance. This increases right ventricular diastolic and right atrial pressures.
- Humoral products of these microthrombi induce a generalised increase in capillary permeability.
- This leads to loss of plasma into the interstitial tissues throughout the body.
- This depletes the vascular volume to a great extent.

CLINICAL FEATURES

The traumatic shock are almost similar to those of hypovolaemic shock. The two differentiating features are:-

1) Presence of peripheral and pulmonary oedema

2) Infusion of large volumes of fluid which may be adequate for pure hypovolaemic shock is usually inadequate for traumatic shock.

TREATMENT

1) Resuscitation

In this type mechanical ventilatory support is more needed.

2) Local treatment of trauma and control of bleeding

Same as the hypovolaemic shock. Surgical debridement of ischaemia and dead tissues and immobilisation of fractures may be required.

3) Fluid replacement

More fluids are required to bring back the patient to normalcy.

Role of anti-coagulation has a debatable role.

Increased coagulation consumes clotting factors of the blood leading to more bleeding.

Moreover obstruction of microvasculature with such microthrombi leads to ischemia.

Anti-coagulation with doses of heparin is large enough to fully anticoagulate the patient may reverse the condition.

CARDIOGENIC SHOCK

It occurs when more than 50 percent of the wall of the ventricle is damaged by infarction.

It is due to primary dysfunction of one ventricle over the other. Such dysfunction may be due to myocardial infarction, chronic congestive heart failure.

PATHOGENESIS

- Dysfunction of right ventricle leads to decreased ability of right heart to pump blood in adequate amounts to blood. Filling of left heart decreases.
- Left ventricular output decreases. Fluid overload leads to over-distension of the left ventricle, with pump failure.
- High filling pressure in right ventricle make fluid leakage of the pulmonary capillaries causing pulmonary oedema and hypoxia.
- Reduction in pumping efficiency of heart leads to excess sweating, vomiting, diarrhoea and diminishes cardiac output.

CLINICAL FEATURES

- In the beginning, cool and the urine output is low, gradually pulse becomes rapid and arterial blood becomes low.
- In cases of right ventricular dysfunction the neck veins become distended and liver may also become enlarged.
- In left ventricular dysfunction the patient has bronchials rales and a third heart sound is heart. Gradually heart becomes enlarged and right ventricle also fails.
- Distented neck is always.

TREATMENT

- ABC
- In case of right sided failure caused by massive pulmonary embolus, should be treated with large dose of heparin I.V.
- In case of left sided failure, morphine should be given.
- For fulminant pulmonary oedema, diuretics should be given.

NEUROGENIC SHOCK

- It is caused by traumatic or pharmacological blockage of sympathetic nervous system producing dilatation of resistance arterioles and capacitance veins, leading relative hypovolemia and hypotension.

PATHOPHYSIOLOGY

- Dilatation of systemic vasculature which lowers the systemic systemic arterial blood pressure
- Blood pools in systemic venules and small veins
- Right heart filling and stroke volume decreases.
- This decreases the pulmonary blood volume and left ventricular output decreases.

The discharge of adrenergic nervous system to the innervated parts of body and release of angiotensin and vasopressin are compensatory mechanism which fail to restore the cardiac output to normal though systemic arterial pressure responds in part.

CLINICAL FEATURES

The peculiar feature is that skin remains warm, and well perfused in contradistinction to the hypovolemic shock.

Urine output maybe normal but B.P. is low.

TREATMENT

- Elevation of legs
- Assumption of Trendelenburg position displaces blood from systemic venules and small veins into the right heart and thus increases cardiac output,
- Left ventricular emptying is quite efficient inspite of elevated legs as the systemic vascular resistance is low.
- Administration of fluid is important. This increases filling of right heart which in turn increases cardiac output.

It can be treated with vasoconstrictor drugs. Its prompt action saves the patient from sudden low B.P. and low cardiac output from imminent damage to the more important organs like brain, heart and kidneys

SEPTIC SHOCK

This type of shock is most frequently caused by gram-positive and gram-negative bacteria, though any agent is capable of causing shock (including viruses, parasites, fungi).

The importance of this shock is that it posses a high mortality rate of about 50%.

The common organisms which are concerned with septic shock

1) E.coli

2) Klebsiella aerobacter

3) Proteus

4) Pseudomonas

5) Bacteriods

CLINICAL FEATURES

Septic shock is often recognised initially by the development of chills and elevated temperature above 100˚ F.

EARLY WARM SHOCK

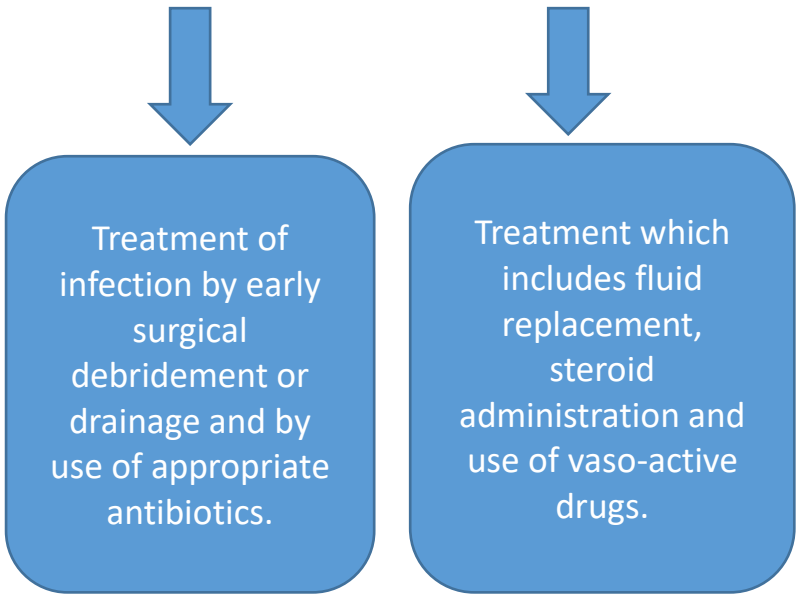
- There is cutaneous vasodilatation. The toxins from the infected tissues increase the body temperature. To bring this temperature down, the vasculature of the skin dilates.
- The cutaneous vasodilatation decreases the systemic vascular resistance. So the arterial blood pressure falls, but the cardiac output increases because the left ventricle has minimal resistance against it.
- Adrenergic discharge further increases cardiac output.
- At this stage the skin is warm, pink and well perfused. The pulse rate becomes high and systemic arterial pressure is low.

LATE COLD SHOCK

- There is increased vascular permeability due to liberation of toxic products into the centre circulation.
- This results in hypovolemia and right heart filling decreases.
- There is decrease in flow into pulmonary vasculature, so left heart filling decreases
- Hence cardiac output decreases.

TREATMENT

Treatment can be divided into two groups:-



STEROIDS

Steroids protect the body cell and its content from the effect of endotoxin. Larger doses of steroids are known to exert ionotropic effect on the heart and produce mild peripheral vasodilatation.

Short term, high dose steroid therapy is recommended in most cases that donot respond to the other methods of treatment.

An initial dose of 15 to 30 mg per kg body weight of methyl prednisolone or equivalent dose of dexamethasone is given I.V. for 5 to 10 minutes.

The same dose may be repeated within 4 hrs if the beneficial effects are not attained.

VASOACTIVE DRUGS

- The vasopressure drugs with prominent alpha adrenergic effects are of limited value in treatment of this type of shock.
- Vasodilator drugs such as phenoxybenzamine are more popular particularly when combined with fluid administration.
- Ionotropic agents eg iso-proterenol or dopamine is ideal when simple volume replacement and other measures have failed to restore adequate circulation.

1) Once a case has been diagnosed, the source of infection is made while treatment of shock is started with fluid replacement.

- Careful monitoring of central venous pressure, pulmonary capillary wedge pressure, urine output and venous blood gases.
- Debridement or drainage of infection should be performed under local or general anaesthesia as soon as possible after the initial stability of the patient.
- The use of specific antibiotics based on appropriate culture and senstivity results .
- Often a combination of antibiotics may be sarted. Cephaothin (6-8 gm/day I.V. in 4 to 6 divided doses).
- Gemtamycin (5mg/day), clindamycin are the antibiotics to be started in beginning.

2) Fluid replacement is of great importance to provide sufficient volume to vital organs.

3) Mechanical ventilation along with endotracheal intubation is needed in treating patients with late septic shock.

- Inadequate tissue oxygenation is a consistent feature of shock and attention to all components of oxygen transport system is essential.

BLOOD LOSS DURING PERIODONTAL FLAP SURGERY:

Post operative bleeding after oral and periodontal surgery is a common complication. The surgical procedure presents a challenge to the body's haemostatic mechanism.

Following surgical procedures, hemorrhage can range from a minor leakage or oozing at the site, to extensive or frank bleeding at surgical site.

The likelihood of this may be attributed to many factors, like the

- tissues of mouth and jaw are highly vascular
- infection
- intrinsic trauma
- presence of foreign bodies

Even after repeated instructions patients tend to play with the area of surgery with their tongue and dislodge the blood clot, which initiates secondary bleeding.

The tongue may also cause suction of blood by creating small negative pressures that cause secondary bleeding. Salivary enzymes may lyse the blood clot before it gets organized.

- Post operative bleeding may be present immediately (primary hemorrhage),within 24hrs or as delayed post operative bleeding (reactionary hemorrhage).
- It can be due to slippage of suture, dislodgement of clots, cessation of reflex vasospasm, normalization of blood pressure.
- Hemorrhage occurring after 7-14 days is secondary to trauma or surgery. The attributed cause is infection and sloughing of blood vessels. Signs and symptoms may include continuous flow, oozing or expectoration of blood or copious pink saliva. Bleeding may be accompanied by pain.
- Patients lost an average of 134 ml of blood, with a range of 16 to 592 ml, during periodontal flap procedures involving an average of 5 1/2 teeth in posterior sextant.

TREATMENT INCLUDES:-

- Reassurance, pressure pack, source of bleeding should be determined.
- If bleeding is due to residual granulation tissue or liver clot type then it should be removed by high speed suction or curettage.
- Bony bleeding can be controlled by crushing the bone with appropriate instrument.
- Soft tissue bleeding may be treated by clamping it with hemostat, if it still persists vessel ligation with sutures, laser coagulation or electro-cautery may be necessary.
- Additional haemostatic agents may also be used.
- Surgery has become commonplace in the treatment of the periodontal patient. A flap approach is commonly used to allow the exposure and correction of periodontal defects. Although such surgery may be routine, little attention has been directed to the extent of hemorrhage occuring during this type of procedure.
- A healthy adult may lose up to 1 litre of blood before developing hypotension, and many people frequently donate 500 ml to a blood bank without any apparent adverse consequences.
- However, considering the possibility of postoperative oozing and blood lost into the tissues during surgery, several investigators recommend that **blood losses greater than 500 ml should be replaced immediately with intravenous fluids or whole blood.**

- Balanced salt solutions do not involve as many hazards as blood transfusion and should be used for volume replacement when the loss of volume is less than 1 litre and the haemoglobin concentration is adequate.

The following procedures are recommended while operating in large areas of the mouth at one sitting or operating for long time periods:

- Preoperative and postoperative blood pressures with the patient sitting should be taken.
- A postoperative standing blood pressure should be recorded to assess possible orthostatic hypotension resulting from acute blood loss.
- Patients experiencing a drop in systolic blood pressure in the standing position of 20 mm Hg, or a drop in diastolic blood pressure of 10 mm Hg following surgery, should be treated with balanced salt solutions intravenously until the blood pressure returns to normal.
- A simple volumetric measurement of blood loss during periodontal flap surgery should be performed using an aspirator with a collection reservoir. The volume can be assessed by using a known volume of irrigating fluids and subtracting this from the total volume of fluid.
- Intravenous fluid replacement should be performed when the patient either

  (a) experiences orthostatic hypotension, or

  (b) loses 500 ml or more of blood.
- Postoperative management of all periodontal surgery patients should include instructions to drink 1 to 2 liters of fluid the day of surgery and 2 days following surgery to help prevent postoperative dehydration.
- The combination of possible postoperative blood loss with operative haemorrhage suggests that patients undergoing multiple quadrants of periodontal flap surgery on the same day may lose as much blood as patients undergoing a major surgical operation.

In dentistry there is a need to be more conscious of the possible sequelae and treatment of hypotension and dehydration resulting from surgical blood loss. This is especially true in periodontal surgery, as most patients are being treated on an outpatient basis.

DIFFERENTIAL DIAGNOSIS:

Identifying the underlying cause of undifferentiated shock is critical, as it can arise from a broad range of conditions that fall within the four primary categories of shock. In some cases, patients may present with a combination of shock types. Additionally, "pharmacological shock" should be considered in the differential diagnosis—this form of shock results from medication-induced vasodilation or myocardial depression, and may be triggered by drugs such as benzodiazepines, beta-blockers, calcium channel blockers, opioids, anticholinergics, or sildenafil.

PROGNOSIS:

Sepsis and septic shock are typically linked to significant long-term morbidity and mortality. Many survivors require admission to long-term acute care or post-acute rehabilitation facilities. Septic shock carries a mortality rate of approximately 40% to 50%. Cardiogenic shock remains

more severe, with mortality rates between 50% and 75%, though this reflects improvement compared to historical outcomes. In contrast, hypovolemic and obstructive shock usually have lower mortality rates and tend to respond more favorably to prompt and appropriate treatment.

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