Shock and Circulatory Failure

Please try to read this before you come along on the 27th of September. It will help you to get more out of the day and hopefully you will find it interesting too!

Introduction

“Shock is a state of inadequate organ perfusion”

The prime function of the circulation is to provide oxygen and substrates to the tissues. Failure to do this results in the clinical condition “Shock” The classical textbook definition of shock expands on the above definition as “acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalised cellular hypoxia”. Shock does not mean hypotension, although the two often go together.

Try to think of shock not in absolute terms of blood pressure but as a state of inadequate organ perfusion. This may be the kidney (poor urine output), the heart (tachycardia), the lungs (shortness of breath), the skin (pale, clammy skin) or most easily diagnosed, the brain (confusion, aggression, anxiety).

This handout includes sections on basic cardiovascular physiology and pathophysiology followed by more detail on shock caused by specific pathologies, ending finally with an overview on the treatment of shock.

Basic Physiology Revision

Blood Pressure = Cardiac Output x Systemic Vascular Resistance (BP = CO x SVR)
Cardiac output = Stroke Volume x Heart Rate (CO = SV x HR)

(remember, cardiac output is the volume of blood pumped from the heart in one minute approx 5 litres/min)

Shock and circulatory compromise can be best diagnosed from the appearance of the patient at end of the bed, by counting the respiratory rate and by palpation of a patient’s peripheries. Circulatory failure with ensuing shock is one of the biggest challenges faced by acute care doctors but is often misunderstood and poorly diagnosed.
Basic Pathophysiology

“Shock” has multiple aetiologies and although the common result is inadequate tissue perfusion it is useful to consider the different causes grouped into categories based on their effect on basic cardiovascular physiology.

1. Fall in Flow

- Haemorrhage
- Diarrhoea
- Burns
- PE
- Tension Pneumothorax
- Tamponade
- Myocardial Infarction
- Cardiac Failure

Voluntary loss, fall in filling and fall in contractility all produce a fall in stroke volume which results in a drop in cardiac output (Flow). To try to maintain the cardiac output the heart rate increases. When CO falls BP will fall, the body compensates by activation of the sympathetic nervous system via baroreceptor receptors. This results in release of catecholamines increasing cardiac contractility, Heart rate and SVR. \( BP = CO \times SVR. \)

Clinically this results in cool peripheries, with a rapid, thready pulse. Shock secondary to a fall in cardiac output is usefully divided into hypovolaemic, obstructive and cardiogenic depending on the pathology. History taking, clinical examination and investigations will help to identify the actual aetiology.

2. Fall in Resistance

- Sepsis
- Anaphylaxis
- Neurological Injury
- Liver Failure

Vasodilation results in a drop in Systemic Vascular Resistance. In order to maintain blood pressure cardiac output increases resulting in the classical clinical presentation seen most commonly with sepsis: warm peripheries with a rapid, bounding pulse. This is sometimes called “high output” shock.
Specific types of shock

This section describes in more detail 5 types of shock this time categorised simply by their underlying aetiology.

1. Haemorrhagic shock
2. Cardiogenic shock
3. Septic shock
4. Neurogenic shock
5. Anaphylactic shock

Haemorrhagic Shock.

This results from blood loss! With causes such as trauma, oesophageal varices, epistaxis, placental abruption and aortic aneurysm rupture, it is a feature of many hospital specialties. The volume of blood lost is not always easily measured. It may be concealed by being swallowed in the case of epistaxis, or left at the roadside in a road traffic accident.

The American College of Surgeons developed a scheme used to estimate the volume of acute blood loss by relating it to changes in physiological parameters:

Classification of Haemorrhagic Shock

**Class I haemorrhage:** 0 - 15% blood loss, that is 0 - 750 mls in a 70kg adult patient

This amount of haemorrhage, although perceived by medical and nursing staff to be large, actually does not impinge very much on a patient’s physiology. Mild anxiety and pallor are occasionally observed but no other vital sign changes are present. This amount of haemorrhage is typically similar to that donated by blood donors.

**Class II haemorrhage:** 15 - 30% blood loss (750 - 1500 mls).

Signs and symptoms include tachycardia, anxiety, pallor and clamminess. Systolic blood pressure may be maintained, however, diastolic blood pressure is noted to rise i.e. the *pulse pressure* will decrease. The normal expected physiology of a pulse rate of about 70 and a blood pressure of 120/60 is now changed to a pulse rate of over 100 and a blood pressure typically of something in the order of 120/100. This subtle change in blood pressure is a useful sign that a substantial haemorrhage has occurred and should be acted on with speed. Blood transfusion may be necessary.

**Class III haemorrhage:** 30 - 40% blood loss (1500 - 2000 mls).

This is a major life threatening haemorrhage with significant associated mortality if not treated almost immediately. The physiological signs are obvious, with a marked tachycardia, anxiety and reduced consciousness, cool peripheries and a drop in the systolic blood pressure. Blood transfusion will almost certainly be necessary.

**Class IV haemorrhage:** >40% haemorrhage

Obviously this is major life threatening haemorrhage which will result in the death of the patient (50% mortality within 15 minutes) unless suitable action is taken. Blood transfusion will be necessary.
The principles of management of haemorrhagic shock in all patients are identical.

**Airway**
- with high flow O2

**Breathing**
- excluding tension pneumothorax

**Circulation**
- 2 Large iv cannulae
- FBC, U&E, Glc, **X-match**
- Fluid bolus 250ml over 2 minutes

The cannulae should be as big as possible preferably 16G (grey) or bigger, and are often most easily sited in the antecubital fossae.

As soon as a patient is identified as being in shock or at risk of developing shock secondary to haemorrhage, senior help should be called. A patient with major haemorrhage is sometimes likened to a bath full of water with the plug pulled out. Fluid resuscitation will maintain the volume in the bath but the most effective thing to do is put the plug back in i.e. stop the bleeding. It is vital therefore that the appropriate specialist team (e.g. surgeons, gastroenterologists) is called and as early as possible.

Fluid resuscitation should begin as soon as possible. Intravenous fluids come in 3 broad categories:

1) Crystalloids  e.g.0.9% NaCl (normal saline), Hartman’s Solution, 5% Dextrose
2) Colloids     e.g.Gelofusine
3) Blood Products e.g. Concentrated Red Cells, Human Albumin Solution, Fresh Frozen Plasma.

All of these solutions may be used in resuscitation with the exception of 5% Dextrose. Excessive use of 0.9% NaCl is associated with the development of metabolic acidosis, therefore many clinicians now prefer to use Hartman’s solution when using crystalloid.

Crystalloid solutions are retained in the intravascular compartment for a shorter period of time in comparison with colloids hence the need to use proportionately more crystalloid to replace blood loss. With crystalloid the ratio is 3:1 whereas with colloid it is closer to 1:1. It is more important, however, to assess the response of the patient to you fluid boluses than to rigidly adhere to these ratios.

A patient with class III or IV haemorrhagic shock will almost certainly need a blood transfusion. In extreme cases unmatched O–ve type blood may need to be used. It is immediately available in certain acute care areas. Group specific blood can be available within about 20-30 minutes. A full cross match may take an hour. Most hospitals now have a major haemorrhage protocol which, if activated, puts all personnel involved in caring for a bleeding patient on standby e.g. porters, haematologists, lab technicians, ICU doctors.

**Cardiogenic Shock**

This condition results from two common medical problems:

1) Major myocardial infarction
2) Severe left ventricular failure commonly due to ischaemic heart disease.

This type of shock is due to failure of the heart as an effective pump. This is therefore failure of flow i.e. reduced cardiac output as a consequence of reduced stroke volume. The reduction in stroke volume is a consequence of dysfunctional contractility.

Cardiogenic shock is difficult to treat but initial management starts, as usual, with **ABC**. Ensuring maximal oxygen supply to the patient is of prime importance.
Cardiogenic shock can be difficult to differentiate from advanced septic shock.

Efforts to make the pump work harder with, for example, inotropic drugs may actually worsen the problem by increasing myocardial demand at a time when either an acute myocardial infarction is occurring or left ventricular failure is advanced. In either case inotropic support will often do more harm than good.

If possible the patient should be sat as upright as their BP allows, with high flow oxygen. IV access should be gained an ECG recorded, and a chest X-ray requested. The normal algorithm of airway management followed by assessment of breathing can reveal useful signs. If the airway is compromised due to decreased conscious level, anaesthetic/ICU assistance should be sought. A patient with cardiogenic shock who requires intubation is at very high risk of dying. Inexperienced use of intubation drugs or airway manipulation with a laryngoscope could result in the death of the patient.

Cardiogenic shock is probably the most difficult type of shock to treat and senior and specialist help should be sought at an early stage. Depending on the aetiology interventions such as intra-aortic balloon pumps and non-invasive ventilation may be indicated. These interventions along with the use of inotropes are the remit of critical care doctors and cardiologists.

**Septic shock**

Septic shock is the end result of the septic process. It has a mortality of between 30 and 50%.

To aid communication the following definitions were developed by consensus between the Society of Critical Care Medicine and the American College of Chest Physicians in 1991:

1. Systemic Inflammatory Response Syndrome
2. Severe Systemic Inflammatory Response Syndrome
3. Sepsis
4. Septic Shock
5. Multiple Organ Failure
Systemic Inflammatory Response Syndrome (SIRS) is the result of an insult to the body either as a result of infection, pancreatitis, major thermal injury, multiple trauma, prolonged under-resuscitation, generalised ischaemia (especially of the bowel) and abuse of noxious substances. All of these triggers may lead to severe inflammation with consequent cytokine release. This is the start of the systemic inflammatory response syndrome and it is characterised by any 2 or more of the following:

i) a temperature of greater than 38°C or less than 36°C  
ii) a heart rate of greater than 90  
iii) a respiratory rate of greater than 20  
iv) a white blood cell count less than 4 or greater than 12 x10⁹ / litre or the presence of greater than 10% immature neutrophils (left shift).

Initially a systemic inflammatory response may be coped with fairly well by the body, and indeed most post-operative patients will show SIRS, but in the disease processes outlined above, progression to severe SIRS can occur. This is defined as the above factors plus evidence of organ failure. Organ failure under these circumstances can mean a poor urine output indicating early renal failure, confusion indicating brain failure or deterioration in blood gases or cardiovascular stability indicating respiratory and cardiovascular failure respectively.

SIRS with bacteraemia or viraemia or fungaemia = Sepsis.  
SIRS with bacteraemia or viraemia or fungaemia + Organ Failure = Severe Sepsis.  
SIRS with bacteraemia or viraemia or fungaemia + refractory hypotension = Septic Shock

Effective intervention at this stage with oxygen, fluids and antimicrobials can save lives. Recognition and appropriate treatment of severe sepsis is fundamental to improving the outcome from intensive care generally.

SIRS is created by cellular malfunction as a result of cytokine release secondary to a major insult to the body as outlined above. Septic shock occurs in response to the presence of bacterial / viral / fungus products within the circulation. Cytokines bind to the endothelium stimulating the release of excess nitric oxide (NO) via the induction of the enzyme inducible nitric oxide synthase (iNOS). This production of excess nitric oxide causes widespread vasodilatation of the vascular tree, which leads to the hypotension and hyperdynamic circulation of septic shock.

Treatment obviously follows the ABC algorithm:

Airway with high flow O2  
Breathing O2, check ABGs, may require ventilator support  
Circulation Large iv cannulae  
FBC, U&E, LFTs, CRP, Glucose, coag, lactate  
Establish Diagnosis Cultures, ECG, CXR

A patent airway with good oxygenation and the ability to remove CO2 is vital. Adequate breathing enables the body to compensate for the metabolic acidosis which may occur with SIRS. Vasodilation results in a relative decrease in effective circulating volume hence the need for administration of intravenous fluids.

Where infection is suspected it is absolutely vital to treat it with antibiotics. Cultures should be taken from every suspected site of sepsis and antibiotic therapy tailored to the causative organism. Early involvement of surgeons in the treatment of surgical sepsis is vital. A patient with a site of
infection that it is not excised or drained (e.g. appendicitis) is at an infinitely higher risk of death. Antibiotics alone are not enough.

The treatment for septic shock therefore is to treat the underlying septic process with antibiotics +/- surgery. Ensuring an adequate circulating volume to try and compensate for the unusual level of vasodilatation caused by excess nitric oxide within the microcirculation is also vital. The patient’s own sympathetic activity is often not sufficient to overcome the effects of vasodilatation and administration of exogenous catecholamines such as adrenaline or noradrenaline may be necessary. The characteristic clinical picture of a hyperdynamic circulation with high cardiac output and vasodilatation may ultimately lead to peripheral shutdown and circulatory failure simply due to the overwhelming nature of the septic shock. It is often difficult to diagnose septic shock when it presents late as the peripheries will be shut down and the cardiac output may not be high, it can thus mimic cardiogenic shock in its worst phase.

It is known that relatively cheap interventions, such as O2, fluids and antibiotics, when given at an early stage, can save lives.

**Anaphylactic shock**

Anaphylactic shock is a rapidly developing syndrome characterised by severe cardiovascular respiratory and mucosal symptoms that have been initiated by an immunological trigger such as an environmental or iatrogenic allergen such as bee sting or an antibiotic. The resulting antigen-antibody complex causes tissue mast cells and basophils to release large quantities of vasogenic substances including histamine, serotonin and bradykinin. These toxic substances cause an increase in capillary permeability, widespread vasodilatation and a consequent drop in blood pressure. There may also be mucosal oedema, which may threaten airway patency, bronchospasm and impaired myocardial contractility.

Senior and anaesthetic help should be sought at an early stage. Treatment as ever starts with **ABC**. Airway management may be complex and if there is any indication of compromise anaesthetic help will be required. Treatment is with of oxygen, intravenous fluids to support the circulation and administration of adrenaline to combat hypotension and bronchospasm. This is most easily done by administering 1mg of adrenaline intramuscularly.

**Neurogenic shock**

Neurogenic shock is caused by blockage of the sympathetic outflow to the intrathoracic sympathetic chain. This usually occurs due to a traumatic lesion of the cervical spine and thus is rare in medical patients. Blood pressure is low and associated with vasodilatation and **bradycardia**. The bradycardia marks this kind of shock out as unusual. Priapism may occur in the male patient. Treatment consists of rapid opinion from a neurosurgeon, ABC’s and administration of a fluid challenge. Vasopressors such as noradrenaline may be needed but should only be given under direct supervision of an intensivist.

**Treatment of Shock**

The treatment of the shocked patient begins in exactly the same way as for any critically unwell patient…**ABC**.
Airway

The airway is opened by means of head tilt / jaw thrust. **100% oxygen is the rule.** Remember simple airway adjuncts such as Guedel airway. Intubation of the shocked patient using anaesthetic drugs is a technique for the very experienced only. The majority of sedative agents will cause vasodilatation and some degree of myocardial depression, which will cause mean arterial pressure to “crash” on induction. For this reason, beware of the use of sedative drugs in the shocked patient. **Call for anaesthetic assistance in cases of doubt.** This also means that opiate analgesics should also be used judiciously, with re-assessment of ABC after each dose.

Breathing

Remember always to exclude tension pneumothorax by feeling for the position of the trachea, percussing and auscultating the chest. A deviated trachea (away from the affected lung) associated with hyperresonance and absent breath sounds should be treated immediately with chest decompression. However, this is not covered in detail in this sepsis day and you should ask about this during your surgical or A&E attachments. A patient with tension pneumothorax will present with distressing shortness of breath accompanied with hypotension and tachycardia. It is life threatening and must be treated immediately.

Circulation

*The Fluid Challenge*

A fluid bolus (250ml of Hartman’s or NaCl 0.9% over two minutes) will be both diagnostic and therapeutic in the treatment and differentiation of shock. It will help in hypovolaemic, septic and anaphylactic shock and will only rarely be dangerous in cardiogenic shock. SO DO IT! However, after each fluid bolus of 250ml, remember to go back and recheck the A,B,C’s. Remember that cannula gauge matters and that a 16 gauge cannula should be used as the minimum size.

Airway with high flow O2
Breathing
Circulation 2 iv cannulae
FBC, U&E, Glc, lactate +/- X-match
Establish Diagnosis ECG, CXR, +/- Cultures

Vasoactive Drugs

Inotropic drugs such as adrenaline, dobutamine, noradrenaline are for specialist intensive care doctors ONLY! Do not even think about using them until you are a cardiologist or an intensive care doctor!

Conclusions

Understanding shock is vital when treating the seriously ill. Knowledge of the presenting pathologies and appearances of the shocked patient can better direct treatment.

You (with a small group of your colleagues) will be presented with a simulated patient with a type of shock to resuscitate on the study day. This is not intended to intimidate you but will be of invaluable use to you when you qualify. This will also help you to understand why recognition and treatment of sepsis and infection are so important.

**Experienced doctors will help you through the scenario… humiliation will not happen!**

Looking forward to seeing you on the day!