

## BATLS

# Battlefield Advanced Trauma Life Support (BATLS)

## CHAPTER 5 SHOCK

### AIM

0501. On successfully completing this topic you will be able to:

- Define shock.
- Identify clinical shock syndromes.
- Understand the difference between compressible and non-compressible haemorrhage.
- Relate the casualty's symptoms and signs to the underlying shock syndrome.
- Discuss the principles of treatment of hypovolaemic shock.
- Demonstrate techniques of fluid replacement.

### Pathophysiology

0502. Shock is the general response of the body to inadequate tissue perfusion and oxygenation. This simple statement encompasses a complex pathophysiological process. If progressive and uncorrected, this process will lead to cell death, organ failure and the death of the casualty.

**Shock is inadequate tissue perfusion.**

### Types of Shock

0503. Most cases will be caused by hypovolaemia, that is, a reduction of circulating volume due to haemorrhage or fluid loss in burns.

0504. Cardiogenic shock and neurogenic shock are both examples of hypoperfusion, when failure to maintain circulating volume is not due to blood loss. In cardiogenic shock the heart fails to pump blood around the body adequately. In neurogenic shock due to a spinal injury, blood vessels dilate causing pooling of blood and making the circulating blood volume inadequate. A similar situation arises in anaphylactic shock due to infection as a late complication of trauma. Both these mechanisms are related to the release of vasodilatory mediators.

#### TYPES OF SHOCK

- Hypovolaemic
- Cardiogenic
- Neurogenic
- Anaphylactic
- Septic

0505. *The treatment of shock is directed towards restoring cellular and organ perfusion with adequately oxygenated blood.* It bears repeating that inadequately treated tissue hypoperfusion causes cell damage and organ failure. Death inevitably follows.

### The Pathological Mechanisms - Early

0506. Fluids in the body lie within cells (intracellular fluid), between the cells (intercellular fluid), and within the blood vessels. The intracellular and intercellular fluids form the *extravascular* compartment; fluid within the blood vessels forms the *intravascular* compartment.

0507. Loss of circulating fluid causes decreased venous return (preload) with subsequent decreased stretch of the muscle in the right and left ventricles of the heart. As a result of this, cardiac output is reduced resulting in hypotension and hypoperfusion (Starling's Law). The body's response to loss of tissue fluid or blood is directed at maintaining circulating volume. The principle corrective mechanisms involved in this process are:

- Fluid shifts from tissues into blood vessels, that is, from the extravascular to the intravascular compartment.
- The heart rate rises (tachycardia) due to increased sympathetic nervous system outflow and reduced vagus nerve inhibition.
- Constriction of blood vessels (vasoconstriction) in the splanchnic bed and limb peripheries, (the cold, pale extremities of shock).
- Fluid retention due to reduced urine output.

### The Pathological Mechanisms - Late

0508. At the cellular level, hypoxic cells initially compensate by shifting to anaerobic metabolism. This results in the formation of lactic acid and the subsequent development of metabolic acidosis. If untreated, cells swell and burst producing marked tissue oedema and loss of function. These events compound the effects of hypovolaemia. Replacement of circulating volume is essential, as is adequate tissue oxygenation, in order to prevent further deterioration of this process of cell death.

## Hypovolaemic Shock

0509. In the battlefield situation, hypovolaemic shock due to trauma or burns is by far the most common cause of the shock syndrome. It is also the most amenable to prompt management. Haemorrhage is the acute loss of circulating blood. In adults, 7% of body weight is circulating blood (approximately five litres in a 70 kg adult or 70 ml/kg of body weight). In children, circulating volume is calculated to be 8-9% of body weight (90 ml/kg of body weight).

0510. Blood loss in trauma may be into five sites ('blood on the floor and four more'):

- External ('on the floor').
- Chest.
- Abdomen.
- Pelvis and retroperitoneum.
- Around long bone fractures (especially the Femur).

**Note:** The presence of significant amount of blood in the chest will be identified during **Breathing** in the primary survey. Identification of other sites of bleeding is an essential element of **Circulation**.

**You must be highly suspicious in all cases of blunt abdominal injuries; these can result in massive, concealed blood loss.**

0511. Major soft tissue injuries and fractures compromise circulating volume in two ways:

- Blood lost at the site of the injury.
- Oedema. Soft tissue injuries result in obligatory oedema, the magnitude of which is related to the severity of the injury. Since plasma and extracellular fluid are in continuity, loss of extracellular fluid will inevitably affect circulating volume. Approximately 25% of post-trauma oedema will be derived from plasma.

0512. Some idea of blood volumes lost from different injuries can be seen from the following:

- |                               |             |
|-------------------------------|-------------|
| • Closed femoral fracture     | 1.5 litres  |
| • Fractured pelvis            | 3 litres    |
| • Fractured ribs              | 150 ml each |
| • One blood-filled hemithorax | 2 litres    |

0513. The following may represent a loss of 500 ml:

- A closed tibial fracture
- An open wound the size of an adult hand.
- A clot the size of an adult fist.

0514. The elderly tolerate shock less well than the fit, young adult, and the very young in whom shock may not be clinically apparent until blood loss becomes quite severe.

## Rapid Assessment of the Cardiovascular System

0515. *Mental state.* If the casualty is conscious and talking sensibly, he is not only breathing through an open airway, he is perfusing his cerebral cortex with sufficient oxygenated blood (50% of the normal cardiac output). Increasing hypovolaemia and subsequent cerebral hypoxia cause alterations in the level of consciousness. These alterations begin with anxiety and if untreated, proceed through confusion and aggressiveness to eventual unresponsiveness and death.

0516. *Colour.* Hypovolaemic casualties become pale, cold, sweaty and cyanosed.

0517. *Pulse.* The presence of a palpable radial pulse implies that the systolic blood pressure is at least 90 mmHg. Absent radial pulses, but a palpable femoral pulse, imply a systolic blood pressure between 80 and 90 mmHg; a palpable carotid pulse, in the absence of other pulses, indicates that the systolic pressure is at least 70 mmHg.

**Radial pulse indicates blood pressure >90 mmHg.**

**Femoral pulse indicates blood pressure ≥80 mmHg.**

**Carotid pulse indicates blood pressure ≥70 mmHg.**

0518. *Capillary refill.* This test is performed by compressing a fingernail for five seconds. The test is normal if colour returns within two seconds of releasing compression (the time taken to say the words 'capillary refill'). Capillary refill is not effective as a measure of circulatory adequacy if the casualty is hypothermic or if it is dark!

**Normal capillary refill takes two seconds.**

0519. *Blood pressure.* This should be recorded during the primary survey and observations continued thereafter to ensure that the trend is towards normotension.

## Classification of Circulating Volume Lost (See Table 5.1)

0520. *Class I.* Loss of less than 15% of circulating volume (up to 750 ml in a 70 kg adult). This is fully compensated by the diversion of blood from the splanchnic pool. There are no abnormal symptoms and signs other than minimal tachycardia.

0521. *Class II.* Loss of 15 - 30% of circulating volume (750 - 1500 ml in a 70 kg adult) requires peripheral vasoconstriction to maintain systolic blood pressure. The pulse pressure is narrowed because of raised diastolic blood pressure; this is a valuable indicator of Class II.

0522. *Class III.* Loss of 30-40% of circulating volume (1500-2000 ml in a 70 kg adult) causes a measurable fall in systolic blood pressure because peripheral

vasoconstriction fails to compensate for the increasing loss. *This manifests itself as the classical symptoms and signs of shock.*

0523. *Class IV.* Loss of more than 40% of circulating volume (over 2000 ml in a 70 kg adult) is immediately life-threatening. Effective and aggressive treatment must be initiated quickly. Loss of more than 50% circulating volume results in loss of consciousness.

**This classification is the ultimate tennis match for survival:**

- Class I**            0-15
- Class II**          15-30
- Class III**        30-40
- Class IV**        40-game, set and exit tournament for good!

Table 5.1 Vital signs

Class	I Up to 750 ml <15% lost	II 750-1500 ml 15-30% lost	III 1500-2000 ml 30-40% lost	IV >2000 ml >40% lost
Heart rate	<100/min	100-120/min	120-140/min	>140/min
Systolic BP	Normal	Normal	Decreased	Decreased/ unrecordable
Pulse pressure	Normal	Narrowed	Narrowed	Very narrow/absent
Capillary refill	Normal	Prolonged	Prolonged	Prolonged/absent
Respiratory rate	14-20/min	20-30/min	>30/min	>35/min
Urine output	>30ml/hr	20-30 ml/hr	5-20 ml/hr	Negligible
Cerebral function	Normal/ slightly anxious	Anxious/ frightened/ hostile	Anxious/ confused	Confused/ unresponsive

### Initial Assessment and Management

0524. Obvious signs of shock are easy to recognise *but they do not usually appear until over 30% of circulating volume is lost.* The earliest signs are of peripheral vasoconstriction and tachycardia followed by narrowing of the pulse pressure. You must assume that any injured casualty with cold peripheries and a rapid heart rate is in shock, until proved otherwise. Remember, a pulse of 80 in a young, fit athletic soldier, whose normal resting pulse is 50, may represent a significant loss of circulating volume.

### Principles of Management of Hypovolaemic Shock

0525. The principles of management are:

- To save life.
- To prevent deterioration.
- To promote recovery.

0526. Diagnosis of hypovolaemic shock must be promptly followed by appropriate treatment, directed at restoring effective tissue perfusion. Restoration of adequate circulating volume is not a substitute for definitive treatment (surgery). Remember: *circulation with haemorrhage control*; attempts should be made to treat, where possible, the cause of the shock; for example, application of pressure dressings and splinting of fractures. **Stop the bleeding!**

0527. Shock is defined as inadequate tissue perfusion. It is now accepted that the appropriate end point for the initial resuscitation of the shocked casualty is the achievement of a blood pressure sufficient to maintain tissue perfusion. This is generally accepted to be a systolic blood pressure of 90 mmHg, that is, a palpable radial pulse. Evidence suggests that a rapid return to normal blood pressure is associated with effects such as the displacement of blood clot and a dilution of clotting factors. Both of these effects may cause rebleeding and adversely affect outcome. The foregoing brings into question the traditional approach of giving *all* shocked casualties a standard intravenous fluid challenge of two or more litres in an uncontrolled way. This is especially the case when the haemorrhage is non-compressible (see paragraph 0530 and table 5.2).

### Examination

0528. Physical examination is directed at the assessment of the **Airway, Breathing and Circulation**. Baseline recordings of vital signs (see Table 5.1) taken at this stage are important for subsequent decisions regarding treatment. Additionally, a rapid neurological survey (AVPU) will give important clues about cerebral perfusion. A more detailed secondary survey may offer information on the cause of the shock and on other conditions contributing to shock.

### Resuscitation

0529. After establishing a clear airway (and protecting the cervical spine when appropriate) you should deliver oxygen, when available, at a high flow rate (10-15 litres per minute), through a bag-valve-mask reservoir system. After correcting any life-threatening breathing difficulties you must turn your attention to *stopping obvious haemorrhage*. This can be achieved by direct or indirect pressure, by wound packing and judicious and correct use of a tourniquet. You can minimise haemorrhage from limb fracture sites by reducing and immobilizing the fracture.

### Haemorrhage

0530. When resuscitating the shocked casualty, you should consider haemorrhage to be of two types:

- Compressible haemorrhage.
- Non-compressible haemorrhage.

0531. Compressible haemorrhage is controllable by direct pressure, limb elevation, the application of a tourniquet or by splintage; All of which can be carried out by you.

0532. Non compressible haemorrhage is bleeding into a body cavity (chest, abdomen, pelvis or retroperitoneum) which can only be controlled by urgent surgery. This *cannot* be

carried out by you.  
 0533. In the case of pelvic fractures, although surgery may be needed, some control of bleeding may be achieved by splinting the pelvis using some form of pelvic splint as part of the resuscitation process.  
 0534. An algorithm for the management of shock is given at Table 5-2, this can be summarised as follows:

**Fluid Resuscitation**

Compressible haemorrhage	not shocked	No fluids
Compressible haemorrhage	shocked	IV fluids
Non compressible haemorrhage	evacuation available	No fluids
Non compressible haemorrhage	evacuation delayed	IV fluids

**Replacement of Lost Volume**

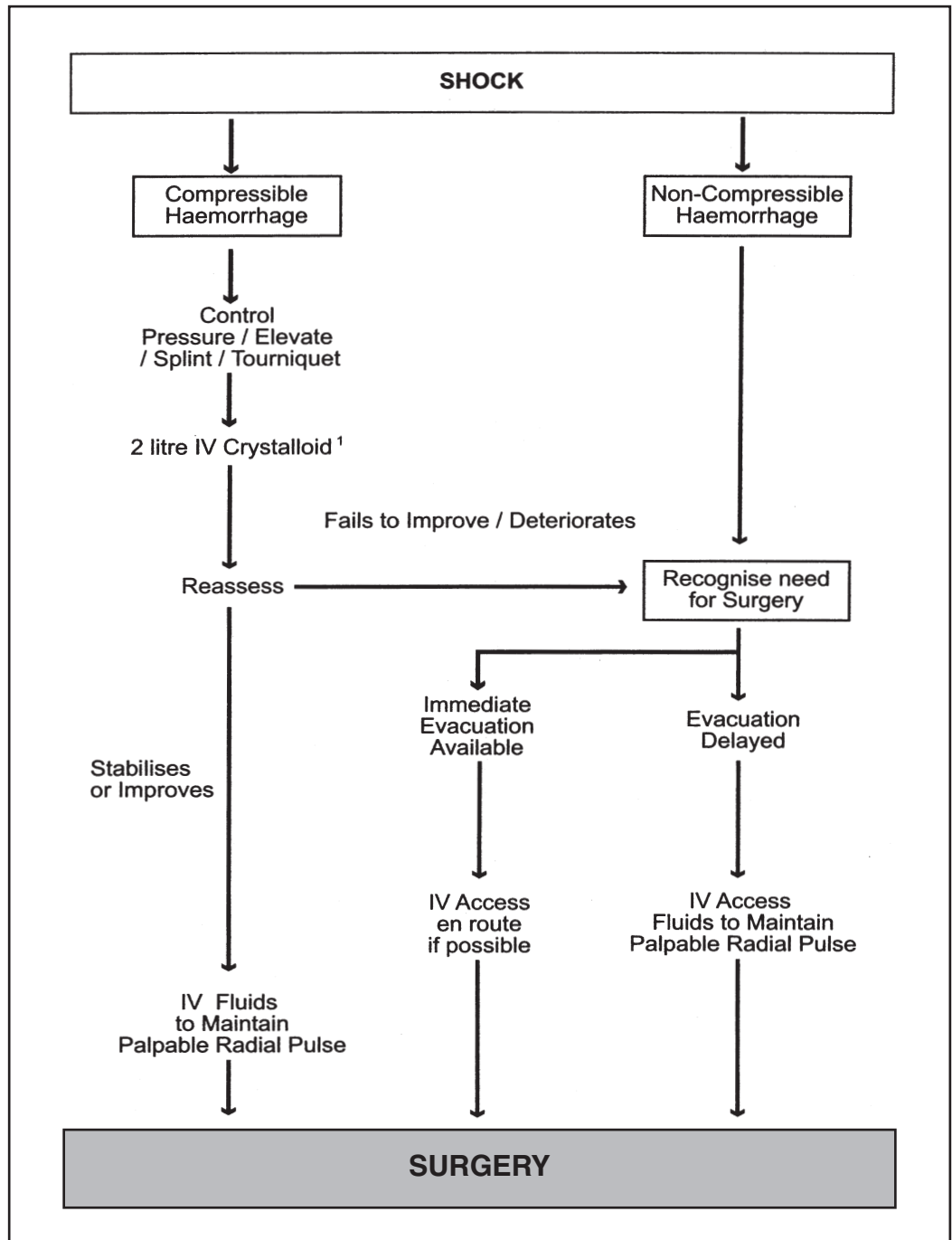
0535. Intravenous access is best achieved by inserting as large a cannula as possible into each antecubital fossa. If peripheral cannulation cannot be achieved consider:

- Femoral vein cannulation
- Intravenous cutdown

0536. Under no circumstances should attempts to obtain intravenous access delay casualty transfer to definitive care unless journey times are going to be prolonged.

0537. The choice of fluids is between crystalloid solutions and synthetic colloids; blood will be available at role 2 and 3 medical units.

Table 5.2 Management of shock



<sup>1</sup> Never give cold fluids by rapid intravenous infusion. Ingenuity may be required to keep crystalloids and colloids warm. For example, never leave fluids in vehicles overnight in a cold environment. If necessary take them to bed with you! Carry packs of fluid under your smock close to your body, this will keep them warm, ready for immediate use. Blood taken straight from a refrigerator should be administered through a blood warmer.

**Gaining intravenous access must not delay transfer to definitive care.**

### Crystalloids

0538. Crystalloids are physiological solutions which remain only temporarily in the circulation (about 30 minutes) before passing into the intercellular space. They are useful for the immediate replacement of lost volume, especially when evacuation times are short and definitive medical care is nearby. Initially, two litres of crystalloid (*Hartmann's Solution/Ringer's Lactate*) should be infused using wide-bore cannulae.

0539. The advantages of crystalloids are:

- They are inexpensive, plentiful and have a long shelf life.
- They have no allergenicity.
- They do not cause coagulation problems.
- There is no risk of transmitted infection.

0540. The disadvantages are:

- Three volumes are required for each volume of blood lost (the 3:1 rule).
- An overload may cause pulmonary and cerebral oedema.

### Colloids

0541. Colloids are either *natural* (derived from blood products) for example plasma, or *synthetic* (derived from starches and gelatins) for example, polygeline (*Haemaccel*) which is a gelatin suspended in physiological solution, or *Gelofusine*.

0542. The advantages of colloids are:

- They are inexpensive, plentiful and have a long shelf life.
- They replace lost volume on a one-to-one basis.
- They remain in the circulation for long periods.
- There is no risk of transmitted infection.

0543. The disadvantages are:

- Occasionally (1:5000), they cause allergic reactions.
- When cold, they either become viscous or form a jelly.

### Treatment regimen

0544. The response to resuscitation by intravenous fluids, and the need for further intravenous fluids and/or surgery, can be considered under four headings:

- *Type I response*. The pulse rate falls below 100, the systolic blood pressure rises above 100 and the pulse pressure widens; these signs remain stable. No further fluid challenge is required.
- *Type II response*. An initial fall of the pulse rate below 100, a rise of systolic blood pressure above 100 and widening of the pulse pressure, then a regression to abnormal levels of these vital signs. This means that either the fluid has been redistributed from the intravascular

compartment to the extravascular compartment, or blood loss continues. Give a further intravenous challenge of two units of colloid or whole blood if available. If the vital signs return to acceptable levels, the response was due to redistribution of fluid; if vital signs remain abnormal then this is a Type III response.

- *Type III response*. Continue intravenous colloid or whole blood at flow rates sufficient to sustain resuscitation (a palpable radial pulse). This casualty needs urgent surgery within the hour.
- *Type IV response*. No response to rapid intravenous infusion of crystalloid, colloid and/or blood. This casualty needs immediate damage control surgery (*to 'turn off the tap'*) if he is to survive.

0545. The above is a simple guide on how a casualty may respond to fluid resuscitation. More important is the question: has the casualty got non-compressible haemorrhage and can he be evacuated now? If the answer is 'yes' - **do it!**

0546. To resuscitate children, the initial bolus dose of crystalloid is 20 ml/kg of body weight. Further boluses will depend on the child's response. (see Supplement No 1).

### Supplementary Treatments and Supportive Measures

0547. Protect casualties from the environment as hypothermia exacerbates shock. Administer oxygen at the highest possible percentage whenever it is available. Blood is indicated for casualties who have sustained a Class III or Class IV haemorrhage. Whenever possible use type specific blood although, in an emergency, uncrossmatched whole blood can be life-saving.

0548. Painful stimuli exacerbate shock. Use analgesia in responsive casualties; remember fracture stabilization and immobilization will minimise haemorrhage at the fracture site in addition to alleviating pain.

0549. Gastric dilation may occur despite the presence of a nasogastric tube. To avoid the risk of aspiration in unconscious casualties, the airway must be protected by a cuffed endotracheal tube, together with intermittent aspiration of the nasogastric tube.

### Monitoring

0550. Once stabilized, the casualty must be continually monitored and reassessed to prevent deterioration and to ensure that all diagnoses have been made. Legible and accurate records are essential, noting the date and time of each intervention and observation. The variables that must be monitored are:

- Pulse (rate, rhythm and pressure).
- Capillary refill time.
- Respiration (rate, expansion and symmetry).

- Blood pressure
- Neurological state (AVPU).

0551. An additional guide to the response to resuscitation or casualty deterioration can be gained from:

- Pulse oximetry.
- Urine output (ideal: adults 50 ml/hr - children 1-2 ml/kg/hr).
- Blood gas analysis.

## Management Problems

### Continuing haemorrhage

0552. You must consider all potential sources of blood loss. Concealed haemorrhage is life-threatening and must be in the forefront of your mind in all hypovolaemic casualties who respond poorly or do not respond to treatment - Response types III and IV. Urgent surgery is required. You must also consider the possibility of dilution of clotting factors when large volumes of fluids have been infused. Remember that stored blood contains fewer clotting factors than fresh blood and fresh frozen plasma.

### Fluid overload

0553. Fluid overload is unlikely to occur in severely injured, previously fit young men. Fluid replacement should be titrated against haemodynamic effects, especially when estimates of loss can be calculated from the mechanism of injury and the haemorrhage is compressible. If fluid overload does occur and pulmonary oedema is detected, the infusion should be slowed to maintain intravascular access and you should consider the use of intravenous diuretics and intravenous morphine.

### Acid/base imbalance

0554. Initial respiratory alkalosis is due to tachypnoea. Metabolic acidosis may develop with severe or long-standing shock as a result of inadequate tissue perfusion and subsequent anaerobic metabolism. When arterial blood gas measurement is available and indicates the presence of metabolic acidosis, it should be treated with increasing intravenous fluids.

### Other Types of Shock

0555. In the battlefield situation, most shocked casualties will have hypovolaemia. The differential diagnosis should also include cardiogenic, neurogenic, anaphylactic and septic shock. Clues can be gained from the history, careful secondary survey, selected additional tests and the response to treatment:

- *Cardiogenic shock.* Myocardial dysfunction may occur following cardiac tamponade, myocardial contusion, air embolus, pulmonary embolus, tension pneumothorax or myocardial infarction.

Ideally, all casualties with blunt thoracic injury should have constant ECG monitoring. Measuring cardiac enzymes will not alter the acute management of myocardial infarction and are poor indicators of myocardial contusion.

- *Neurogenic shock.* Damage to some parts of the brain stem or high thoracic/cervical spinal cord, produces hypotension due to interruption of the sympathetic chain, with subsequent loss of vessel tone. Sympathetic denervation also removes the cardiac response to hypotension, that is, tachycardia. The vagus is unopposed resulting in bradycardia which may worsen if the vagus nerve is stimulated, for example, by passing an endotracheal tube or nasogastric tube. The casualty with neurogenic shock demonstrates hypotension without tachycardia. The immediate treatment of symptomatic bradycardia in neurogenic shock is atropine 0.5 - 1 mg intravenously.
- *Anaphylactic shock.* You should suspect this uncommon mechanism of shock in any casualty who has recently received medication or who has been exposed to other allergens, especially when the history is not known. Signs of anaphylactic shock include peripheral vasodilatation, oedema, bronchospasm and urticaria. Attention to the airway is essential. The definitive treatment is adrenaline 1 mg as 1 ml of 1:1000 solution intramuscularly or, in life-threatening cases, 1 mg as 10 ml of 1:10000 solution intravenously *slowly*.
- *Septic shock.* Septic (toxic) shock may occur if evacuation is delayed for many hours. It is most likely to occur in casualties with penetrating abdominal injuries and in whom the peritoneal cavity has been contaminated by intestinal contents. The mechanism of shock is one of vasodilatation caused by bacterial toxins. If there has been no haemorrhage (or if haemorrhage has been adequately corrected) the casualty, although hypotensive, will have a tachycardia, warm pink skin and a wide pulse pressure (a full bounding pulse).

## Summary

Hypovolaemia is the cause of shock in most battle casualties. A high index of suspicion is essential during assessment of the casualty. Management requires immediate control of haemorrhage either by direct compression, splintage, the application of a tourniquet or where necessary, by urgent surgery.

## Skills Station 4 Peripheral Intravenous Cannulation

### Aim

The aim of this skills station is to give you the opportunity to practise and demonstrate the technique of peripheral intravenous cannulation.

### Equipment

Model arm or IV practice pads.  
IV giving sets.  
14 gauge cannulae.  
Hartmann's Solution.  
Haemaccel.  
Micropore tape.  
Adhesive tape 3 inch.  
Alcohol sterets.  
Blood sample bottles.  
Venous tourniquet.  
Surgical gloves.

### Skills Procedures

- Run the intravenous solution through the giving set.
- Identify the vein to be cannulated (first choice is the antecubital fossa).
- Check there are no fractures proximal to the intended cannulation site.
- Apply a venous tourniquet proximal to the intended cannulation site.
- Prepare the skin with an alcohol steret.
- Insert the cannula into the vein; withdraw the trochar and feed the cannula further into the vein when blood is seen in the flash chamber.
- Draw 15 ml of blood for crossmatch, full blood count and haematocrit.
- Connect the giving set and commence flow at the required rate.
- Secure the cannula with Micropore tape.
- Cover the cannula site with adhesive tape.
- Secure the giving set tubing.
- If the casualty is going to be moved or evacuated ensure the taping of the cannula and giving set is robust enough to survive this; consider applying a POP backslab.

## Skills Station 5 Peripheral Venous Cutdown / Femoral Access

### Aim

The aim of this skills station is to give you the opportunity to practise and demonstrate the technique of peripheral venous cutdown.

### Equipment

Animal model or IV practice pads.  
IV giving sets.  
14 gauge cannulae.  
Hartmann's Solution.  
Micropore tape.  
Alcohol sterets.  
Sutures (3-0).

Ties (3-0).  
Scalpels (22 balde).  
Small haemostat forceps.  
Gauze swabs (4" x 4").  
Venous tourniquet.  
Surgical gloves.  
Scissors.

### Anatomical Considerations

- The primary site for cutdown is over the long saphenous vein above the ankle at a point approximately 2 cm anterior and 2 cm superior to the medial malleolus - but not if there is significant injury proximal to this site. (See Fig 5.1).
- The site of second choice is the median basilic vein, located 2.5 cm lateral to the medial epicondyle of the humerus in the antecubital fossa.

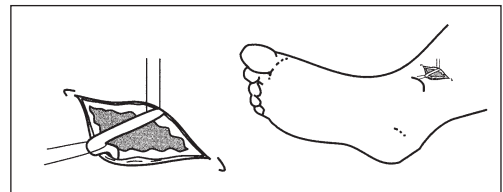


Fig 5.1 Cut down technique - long saphenous vein (right ankle).

### Skills Procedures

- Run the intravenous solution through the giving set.
- Apply a venous tourniquet proximal to the intended cannulation site.
- Prepare the skin with an alcohol steret.
- Infiltrate the area with local anaesthetic.
- Make a full-thickness transverse incision through the skin.
- By blunt dissection, identify and display the vein.
- Free the vein from its bed and elevate a 2 cm length.
- Ligate the distal end, leaving the suture in place for traction.
- Pass a tie around the proximal end of the vein.
- Make a small transverse venotomy and gently dilate the opening with the tip of a closed haemostat.
- Introduced the plastic cannula (without trochar) through the venotomy and secure it in place by tying the proximal ligature.
- Attach the giving set and commence flow at the required rate.
- If possible, close the incision, otherwise apply a sterile dressing and secure the giving set tubing in place.

### Complications

Haemorrhage or haematoma.  
Perforation of the posterior wall of the vein.  
Nerve transection.  
Phlebitis.  
Venous thrombosis.

## Femoral access

The femoral vein lies medial to the femoral artery (see Fig 5.2). This anatomy can best be remembered by use of the mnemonic **NAVY** - **N**erve, **A**rtery, **V**ein, **Y**-front.

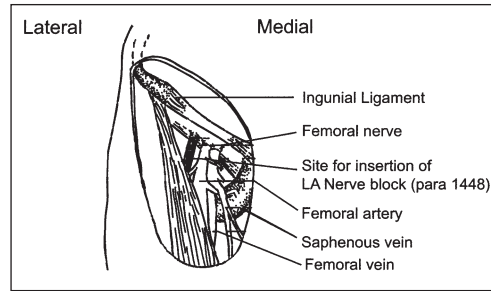


Fig 5.2 Anatomy of the femoral region.

## Skills procedures

- Run the intravenous solution through the giving set.
- Place a 10 ml syringe onto a brown venflon.

- Prepare the skin with an alcohol steret and ensure that suitable fastening materials are available.
- Identify the femoral artery and place the middle and index finger of the left hand on the pulsation.
- Insert the cannula medial to the two fingers advancing towards the head with the needle at 45° to the skin.
- Apply continuous moderate suction to the syringe by gently withdrawing the syringe plunger as you advance.
- When the vein is punctured blood will enter the syringe rapidly. Stop advancing the cannula, pause then gently advance the cannula into the vein while simultaneously withdrawing the needle.
- Suture or tape the cannula into place.
- Attach the giving set and commence flow at the required rate.
- Apply an appropriate dressing.