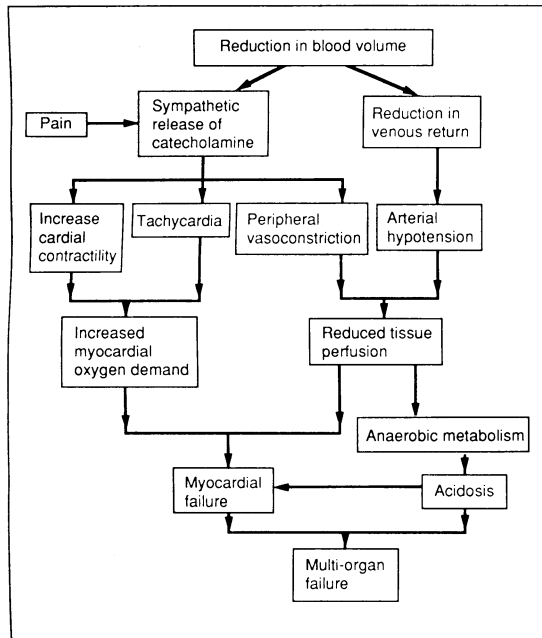


MANAGEMENT OF HYPOVOLAEMIC SHOCK

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Pathophysiology of hypovolaemic shock.

Classification of hypovolaemic shock according to blood loss

	Class I	Class II	Class III	Class IV
Blood loss:				
Percentage	<15	15-30	30-40	>40
Volume (ml)	750	800-1500	1500-2000	>2000
Blood pressure:				
Systolic	Unchanged	Normal	Reduced	Very low
Diastolic	Unchanged	Raised	Reduced	Very low or unrecordable
Pulse (beats/min)	Slight tachycardia	100-120	120 (Thready)	>120 (Very thready)
Capillary refill	Normal	Slow (>2s)	Slow (>2s)	Undetectable
Respiratory rate	Normal	Normal	Tachypnoea (>20/min)	Tachypnoea (>20/min)
Urinary flow rate (ml/h)	>30	20-30	10-20	0-10
Extremities	Colour normal	Pale	Pale	Pale and cold
Complexion	Normal	Pale	Pale	Ashen
Mental state	Alert	Anxious or aggressive	Anxious, aggressive, or drowsy	Drowsy, confused, or unconscious

Symptoms of hypovolaemia according to blood loss

Blood loss (ml)	Class	Symptoms
<750	I	None
750-1500	II	Cardiovascular signs due to catecholamine release: thirst, weakness, tachypnoea
1500-2000	III	Systolic pressure falls
>2000	IV	Systolic pressure becomes unreadable

Hypovolaemic shock is a clinical state in which tissue perfusion is rendered relatively inadequate by loss of blood or plasma after injury to the vascular tree.

A reduction in blood volume produces a fall in systolic pressure, which triggers a sympathetic catecholamine response that results in peripheral vasoconstriction, a rise in pulse rate, and a reduction in pulse pressure. The tachycardia and increased cardiac contractility lead to an increased myocardial oxygen requirement.

Blood flow to the skin and peripheral tissues is reduced in an effort to preserve reasonable perfusion of vital organs such as the brain, heart, liver, and kidneys. If there is continuing blood loss inadequate tissue perfusion results in anaerobic metabolism, acidosis, and reduction in the performance of the vital organs. Further myocardial depression accelerates this process, and pain stimuli add to the sympathetic outburst.

The following are early symptoms and signs of hypovolaemic shock. They reflect the underlying pathophysiology.

- Hypotension (due to hypovolaemia, perhaps followed by myocardial insufficiency)
- Skin pallor (vasoconstriction due to catecholamine release)
- Tachycardia (due to catecholamine release)
- Confusion, aggression, drowsiness, and coma (due to cerebral hypoxia and acidosis)
- Tachypnoea (due to hypoxia and acidosis)
- General weakness (due to hypoxia and acidosis)
- Thirst (due to hypovolaemia)
- Reduced urine output (due to reduced perfusion).

In most cases the signs and symptoms can be related to the amount of blood loss, which can be classified in four broad groups (classes I-IV).

In previously healthy young adults systolic pressure is often preserved despite quite appreciable blood loss (1.5-2.0 litres) owing to the effective response to sympathetic stimulation. Eventually, however, there is a precipitous fall as the myocardium suddenly fails because of hypoxia and acidosis. Conversely, patients with coronary arterial disease may become hypotensive because of myocardial insufficiency after only modest blood losses of up to 500 ml.

Patients receiving certain drugs (for example, β blockers) may not be able to produce an appropriate sympathetic response and may also become hypotensive after modest blood loss. Also, it must be taken into account that intravascular loss is accompanied by additional fluid depletion of the interstitial space, which amounts to about 25% of the overt blood loss.

Resuscitation of patients with trauma

- (1) Adequate pulmonary oxygenation
- (2) Control of haemorrhage
- (3) Replacement of lost volume
- (4) Monitoring the effects of (1), (2), and (3)
- (5) Support of myocardial contractility
- (6) Relief of pain

Generally, losses of up to 750 ml (class I) (15% of the circulating blood volume) do not generate any pronounced signs or symptoms. Further haemorrhage, amounting to 1.5 litres (class II), produces cardiovascular signs of catecholamine release, thirst, weakness, and tachypnoea. Systolic pressure continues to fall as blood loss mounts to 2 litres (class III) and often becomes unreadable after 2.5-3.0 litres (class IV) have been lost.

The objective of the management of hypovolaemic shock is to maintain tissue oxygenation and restore it to normal values. This entails applying the basic principles of resuscitation of patients with trauma. Resuscitation is followed by definitive treatment (including surgery).

Pulmonary oxygenation

Ventilate patients with hypovolaemic shock
Use 100% oxygen for patients with severe shock

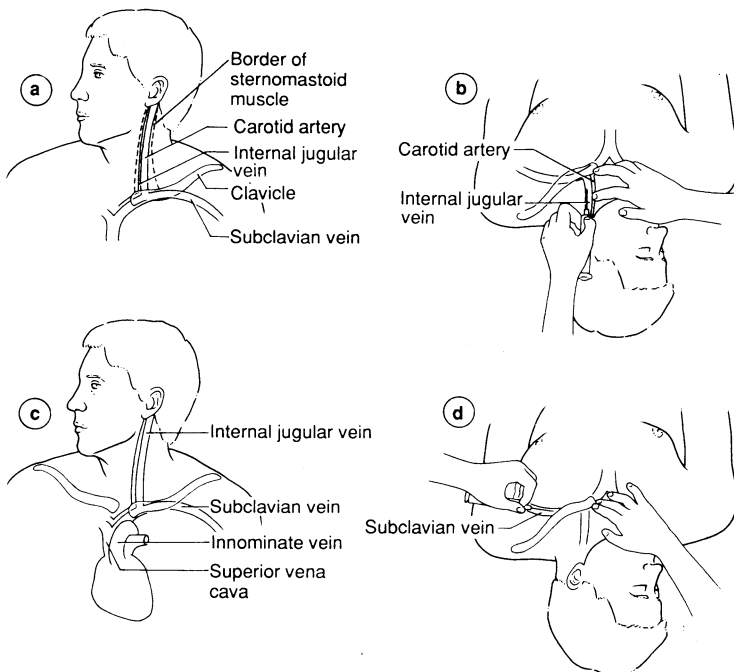
To ensure optimal pulmonary oxygenation patients with hypovolaemic shock should have a clear airway and be adequately ventilated with oxygen at a high inspired concentration. Unconscious patients with severe shock should be intubated and ventilated with 100% oxygen, and care should be taken to exclude impairment of ventilation due to pneumothorax, haemothorax, or diaphragmatic elevation caused by gastric distention.

Control of haemorrhage

For peripheral haemorrhage:
"Parts in the air, press on the hole"

Peripheral haemorrhage should be controlled by elevation of the injured part and by placing a firm pad and bandage over the wound. Tourniquets are rarely advised though may be essential and are relatively harmless in patients who are going to undergo amputation. Probing in the wound to search for ruptured vessels is not recommended outside the operating room.

Replacement of loss



Cannulation of the jugular vein ((a) and (b)) and of the subclavian vein ((c) and (d)).

Losses of blood should be replaced intravenously in response to clinical signs and symptoms and in all patients estimated to have lost more than 750 ml.

Intravenous cannulation

The site of haemorrhage should be considered carefully when cannulation is undertaken. There is little point in setting up an infusion in an injured limb or in the femoral vein in a patient with pelvic or abdominal injuries. With this proviso the peripheral veins of the arm, if accessible, are traditionally preferred for cannulation. There is much to be said, however, for more central access through a short cannula in the subclavian or internal jugular vessels, which are less subject to constriction and feed almost directly into the right atrium. Preference for one or other site is individual according to experience and expertise. In patients with injuries of the neck and arms the femoral vein is preferable. Venous cut down techniques are time consuming, rarely necessary, and usually less effective than direct cannulation of a central vein.

The diameter of the cannula in a cannula over needle system should be not less than 14 gauge. Cannulas of 10 gauge can be easily inserted into the internal jugular or subclavian veins. Long lines from the antecubital fossa are not suitable for rapid transfusion as the flow rate is inversely related to the length of the catheter.

Intravenous fluid replacement in haemorrhagic shock

Class I (haemorrhage 750 ml (15%))	2.5 l Ringer-lactate solution or 1.0 l polygelatin
Class II (haemorrhage 800 -1500 ml (15-30%))	1.0 l polygelatin plus 1.5 l Ringer-lactate solution
Class III (haemorrhage 1500-2000 ml (30- 40%))	1.0 l Ringer-lactate solution plus 0.5 l polygelatin plus 1.0-1.5 l whole blood or 1.0-1.5 l equal volumes of concentrated red cells and polygelatin
Class IV (haemorrhage 2000 ml (48%))	1.0 l Ringer-lactate solution plus 1.0 l polygelatin plus 2.0 l whole blood or 2.0 l equal volumes concentrated red cells and polygelatin or hetastarch



Intravenous replacement fluids.



Intravenous infusion in a military surgery.

Disadvantages of blood transfusion in hypovolaemic shock

- Time is required to group and cross match individual units of blood
- Blood has a high viscosity and the microcirculation in shock may be improved by a reduction in packed cell volume
- Blood stored for more than a few days has a high potassium ion concentration and the platelets and white cells fragment rapidly, losing normal function
- Blood and certain blood products may be infected. (The risk of acquiring HIV infection from transfused blood has been virtually eliminated in the United Kingdom)

Choice of intravenous fluid

Intravenous fluids should be given to restore an adequate circulating blood volume. Normal electrolyte and coagulation constituents and colloid osmotic pressure and a packed cell volume above 30% are necessary to ensure adequate oxygen carrying capacity. The choice of intravenous fluids in clinical practice lies among crystalloid, colloid, and albumin solutions; blood in the form of whole blood or packed red cells; and a judicious mix of all of these.

Colloid solutions replace intravascular loss and restore haemodynamic values towards normal. They do not replace interstitial loss. Crystalloid solutions replace both interstitial and intravascular loss, but large volumes are required to restore normal haemodynamics. In practice, a combination of a crystalloid solution and a colloid solution should be given to patients with blood loss of more than 1 litre.

Colloid solutions are generally iso-oncotic and may be used to replace lost volumes of blood on a 1:1 basis, restoring haemodynamic variables to normal values. Polygelatins are cheap and effective blood volume expanders. They have a long shelf life of six years, a half life in vivo of six to eight hours, and a low index of causing anaphylactic reactions. Haemaccel has a similar electrolyte content to plasma whereas Gelofusine contains very little potassium. Both are suitable for replacing blood losses of up to 1 litre and in patients with more extensive haemorrhage when used in combination with blood transfusion to maintain a packed cell volume of 30%.

Hetastarch (6% in isotonic saline) is an effective blood substitute in patients with mild and moderate blood loss. It is more expensive than the polygelatins but has a much longer half life (12 to 14 hours) in the circulation. Care must be taken, therefore, to avoid circulation overload when blood is transfused at a later stage to restore the packed cell volume. The incidence of anaphylactic reactions is low.

Crystalloid solutions—Ringer-lactate solution may be used in patients with mild class I haemorrhage of up to 15% of blood volume. Replacement volumes should be three to four times the estimated loss as the electrolyte solution is distributed throughout the extracellular (intravascular and interstitial) space. The volume should be increased to compensate for urine loss. Vascular support with isotonic electrolyte solutions is short lived.

Blood—Whole blood or packed red cells are required in patients with moderate and major blood loss to maintain a packed cell volume of 30%. It is not desirable to strive for higher values in the early stages of volume resuscitation as a modest reduction in packed cell volume allows improvement in the microcirculation, especially in the presence of arteriolar vasoconstriction.

Though whole blood is the ideal replacement in patients with major haemorrhage, limitations of supply may dictate that concentrated red cells are used, diluted to normal values of packed cell volume by concurrent transfusion of polygelatin or hetastarch.

Trauma and obstetric centres should retain a small number of relatively fresh units of O negative blood for immediate transfusion in cases of severe, life threatening haemorrhage.

Blood transfused rapidly should be warmed before infusion to maximise flow rates and to minimise the risk of cardiac arrhythmia and core hypothermia. Blood filters have not been proved to be of value.



Aspiration of "clean" blood from the cavity.

Autologous blood—In patients with severe thoracic or abdominal injuries "clean" blood may be aspirated from the cavity, anticoagulated, and returned to the patient through an intravenous cannula using a "cell saver" system. Autologous blood is valuable in patients with major vascular injuries of the thorax and abdomen and in those with a ruptured liver or spleen, but clearly the procedure cannot be applied in patients with abdominal trauma who have a ruptured bowel or in those with thoracic trauma who have oesophageal or lung damage. The transfusion of autologous blood has several advantages, particularly if blood of a patient's blood group is in short supply. The blood is the patient's own, free of infection, warm, and immediately available.

Coagulation problems

Coagulation problems occur in patients with massive blood loss because of dilution with blood substitutes and the fact that coagulation factors deteriorate rapidly in stored blood. Moreover, tissue destruction releases various products that inhibit the normal coagulation process. The clotting process should be monitored by regular screening and deficiencies treated definitively rather than by infusion of valuable fresh frozen plasma and platelets on an arbitrary basis.

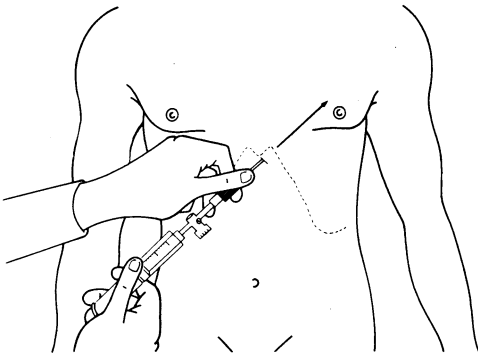
Monitoring progress and treatment

Variables to monitor in hypovolaemic shock

- Pulse rate
- Arterial pressure
- Pulse pressure
- Central venous pressure
- Urinary output
- Changes in the electrocardiogram
- Temperature
- Peripheral oxygen saturation
- Mental state

Requirements for blood volume replacement should be based on all of factors in the box, particularly pulse rate, arterial pulse and central venous pressures, peripheral oxygen saturation, and urine flow rates. Transfusion should be continued to produce an adequate arterial pressure, a urine flow of 50 ml/h, and a central venous pressure that responds to a rapid infusion of 200 ml by a sustained rise of more than 3 cm H₂O over the previous value.

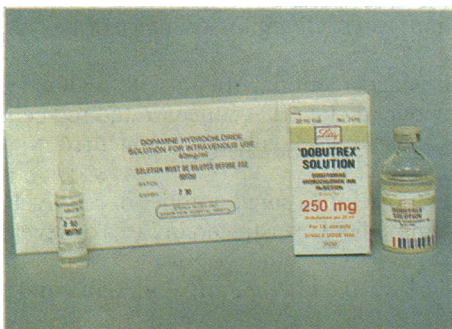
If these variables improve and the improvement is maintained then clearly the blood loss is under control. Failure to maintain the improved values indicates continuing loss and requires further transfusion and early surgery. If the patient does not respond satisfactorily to transfusions the rate of loss is exceeding the fastest possible rate of intravenous replacement. This is usually associated with major thoracic, abdominal, or pelvic injuries. In such instances the patient must be taken to the operating room for immediate thoracotomy or laparotomy and bleeding controlled with clamps or packs, or both, while the anaesthetist "catches up" with the transfusion requirements. Salvage of autologous blood may be appropriate.



Needle pericardiocentesis.

A rising central venous pressure associated with a low arterial pressure, tachycardia, and a reduced urine output indicates tension pneumothorax, cardiac tamponade, or cardiac failure. Cardiac tamponade is treated by thoracotomy, sometimes preceded by relief needle paracentesis. In patients requiring inotropic support because of myocardial failure measurement of pulmonary wedge pressure with a Swan-Ganz catheter may be helpful in comparing individual ventricular load and performance. In most patients with previously normal hearts, however, the central venous pressure and the pulmonary artery wedge pressure follow each other closely and the extra expense of this invasive technique is unjustified.

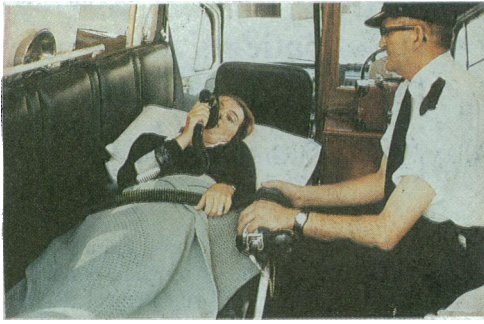
Cardiac contractility and renal output



Inotropic drugs.

A patient with a previously impaired myocardium may need inotropic support with dopamine and dobutamine. Such support is not a substitute for adequate volume replacement but is used to enhance myocardial contraction if required. Rates of dopamine infusion should be confined to "renal" doses (up to 5 µg/kg/h) that enhance urine output. Higher doses cause vasoconstriction and tachycardia, which results in an increase in myocardial oxygen demand that may not be achievable because of inadequate myocardial blood flow. Dobutamine should then be added to improve myocardial performance.

Pain relief



Patient receiving Entonox on the way to hospital.

Pain relief must be given not only for its compassionate value but also for its essential beneficial influence on the pathophysiology of hypovolaemic shock in reducing catecholamine secretion. Giving a mixture of 50% nitrous oxide and 50% oxygen (Entonox) is of value before the patient reaches hospital, and this should be supplemented with increments of intravenous morphine 5 mg, nalbuphine 10 mg, or ketamine 25-50 mg with diazepam 5-10 mg or midazolam 5 mg until analgesia is achieved.

Conclusion

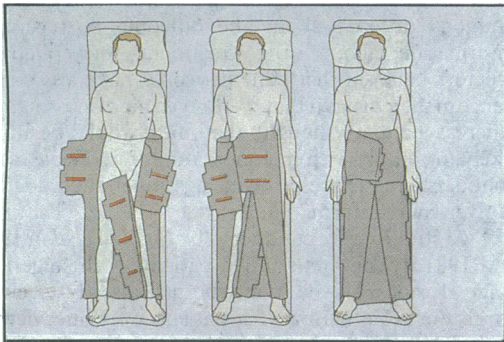
The photograph showing aspiration of blood was reproduced by kind permission of Solco Basle. The line drawings were prepared by the department of education and medical illustration services, St Bartholomew's Hospital.

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Many patients will die of hypovolaemic shock despite the fact that the principles of management and treatment should be well known and understood. Too often, however, in retrospect the treatment offered was too little, too late, allowing a malignant circle of pathophysiological changes to be irreversibly established. Early aggressive treatment offers the best results.

Pneumatic counter pressure suit



Pneumatic counter pressure suit.

Pneumatic counter pressure suits (medical anti-shock trousers) have been used extensively, particularly in the United States, to control haemorrhage from the legs, pelvis, and abdomen and spinal shock. The suit consists of inflatable sections for each leg and the abdomen and is radiotranslucent with access for urinary catheterisation and digital rectal examination.

Application of the suit produces:

- Autotransfusion of 0.5-1.0 litres of homologous blood
- Reduction in haemorrhage from tissues beneath the suit
- Reduction in the total functioning volume of the vascular compartment, permitting relatively improved perfusion of the heart, brain, and arms (thereby assisting with intravenous cannulation)
- Splinting of limb and pelvic fractures with consequent reduction in blood loss and pain.

- Do not apply the abdominal section in pregnant women or patients with abdominal injury with protruding viscera
- Do not deflate the suit until at least two wide bore intravenous cannulas are safely in situ and an adequate supply of blood and blood substitutes are available
- In patients with abdominal injuries or ruptured aortic aneurysm the suit should not be deflated until the patient is in the operating room and the surgical team ready to control haemorrhage
- Do not leave the suit inflated for more than one to two hours as ischaemic anaerobic metabolism may lead to profound general metabolic acidosis
- Take care not to overtransfuse patients with poor left ventricular function with the suit inflated because pulmonary oedema may occur.

The suit should be applied in patients with symptoms of hypovolaemia and a systolic blood pressure <90 mm Hg. Suit inflation pressures of 40-50 mm Hg (5.5-6.5 kPa) should be used initially, increasing to 80 mm Hg (10.5 kPa) if the systolic pressure does not improve. The time of application should be noted.