44 Shock

44.1 Assessment of shock; hypovolaemia

Shock is any abnormality of the circulatory system that results in inadequate organ perfusion and altered energy metabolism. It is dangerous to depend on any particular blood pressure figures to make the diagnosis because people can be in shock and still have relatively normal BP and pulse rates (this is called "compensated shock").

The delivery of oxygen to the issues (and its subsequent utilization producing energy) is dependent on multiple factors: haemoglobin levels, haemoglobin oxygen saturation levels, systemic cardiac output (in turn dependent on blood volume, heart rate and heart contractility), regional blood flow (vasoconstriction) and the integrity of the cellular metabolic system (which can be affected by toxins and energy status). Disruption of any one of these can cause cellular-level shock (altered oxygen and energy metabolism).

You can classify shock as follows:

(a) Haemorrhagic (hypovolaemic) shock: from loss of circulating volume, by far the most common type in trauma & burn patients.

(b) Cardiogenic shock (44.5): arising from heart dysfunction such as cardiac contusion, or occluded venous inflow owing to cardiac tamponade or tension pneumo- or haemo-thorax (49.1). Myocardial infarction is uncommon in trauma, *but may be caused by reduced perfusion*.

(c) Neurogenic shock (44.5): *is not shock associated with head injury*. A spinal cord injury (often associated with vertebral fracture) may result in hypotension owing to loss of sympathetic vasomotor tone.

(d) **Septic** (distributive) shock (44.5): arising from septicaemia, rare in the initial stages of trauma but may occur within hours of injury, especially with abdominal injuries. In pregnant women, amniotic embolism causes a pattern of distributive shock.

(e) Anaphylactic shock is similar in its appearance.

HYPOVOLAEMIC SHOCK.

In hypovolaemic shock, the loss of circulating fluid volume causes a progressive vasoconstriction of the arteries and emptying of the venous capacitance vessels resulting in a diminished blood return to the heart. Tachycardia is the earliest sign (but may not be obvious in athletes and older patients on beta-blockers).

The resulting drop in cardiac output results in tissue hypoperfusion and that in turn, causes metabolic (lactic) acidosis.

MANAGEMENT

Administer oxygen by mask to all hypovolaemic patients and attempt to restore their circulating volume with isotonic fluids (Ringers lactate or 0.9N saline) and/or blood.

Here are some important guiding principles: (1) **Presume** shock in trauma to be hypovolaemic until you have proved it is<u>n't</u>.

(2) The most urgent need is to locate the source of bleeding & stop the bleeding and at the same time, restore the circulating volume and therefore venous return to adequate levels. Failure to stop ongoing bleeding leads to the lethal triad of acidosis, hypothermia and coagulopathy, which ultimately ends in death.

(3) Don't pour in blood or IV fluids if the systolic BP is >80mmHg till you have controlled the bleeding!

(4) **Don't diagnose 'shock' by looking for specific BP values:** recognize inadequate perfusion and the clinical signs of shock. *Any injured patient who is cool, has tachycardia, and poor peripheral perfusion is in shock until proven otherwise.* It is very possible to be in shock with a normal BP.

(5) In acute bleeding, the **Hb** and haematocrit values will *not* fall until you or the body system restore the circulatory blood volume; therefore initial values will only tell you about pre-existing anaemia but *not* tell how much blood has been lost. A patient with anaemia before injury will often need blood transfusion sooner.

Follow ABC: Check the airway. Ensure the patient is breathing normally Put up an IV line. Stop the bleeding!

N.B. Don't use the Trendelenburg (head-down) position because it is uncomfortable for the patient, causes cerebral congestion and impairs respiration by making diaphragm movements hard work.

Place 2 large-bore intravenous cannulae (14-18G for adults) in peripheral veins *or if you fail*, insert intra-osseous needles (tibia, femur, iliac crest, sternum or humerus), especially in children, if you have them.

Consider a cut-down (the saphenous vein anterior to the medial malleolus of the ankle is often a good option) or inserting a central venous infusion.

The femoral vein is perhaps the safest if you are not experienced. *Don't use the same femoral vein site again* because of the risk of infection and thrombosis. Inserting subclavian or internal jugular central lines carries a risk of pneumothorax, so place them on the side where you might have to insert a chest drain! The external jugular vein is an alternative.

Use ultrasound to help you guide the needle, if you can.

N.B. Central IV infusions don't allow flow as fast as peripheral lines because the length of the IV line increases the resistance and diminishes the flow rate (Poiseuille's law). They should be your last resort. Intra-osseous lines or short peripheral catheters are a better option.

Begin the 1st bolus infusion (1L for adults or 20mL/kg for children) of warmed isotonic crystalloid fluid (0.9% normal saline or Ringers lactate).

Check the glucose level and type and cross-match blood. Collect the patient's own blood, if it is feasible to do so for possible autotransfusion (44.4), while looking for sites for intravenous access.

DEGREE C	OF HAEMORRHAGIC SHOCK
----------	-----------------------

Degree		II	III
BP	<20%	<20-40%	<40%
Pulse	Normal	Vol ↓	Weak
Temperature	Normal	Cool	Cold
Colour	Pale	Pale	Mottled
Circulation	Slow	Slow	Sluggish
Thirst	Normal	Definite	Severe
Urine	Normal	Vol ↓	Oliguria
Mentation	Distressed	Apathetic	Comatose

Fig. 44-1 GRADING SHOCK. After Blackburn G, Field Surgery Pocket Book, Min Defence, HM Stationery Office, London. with kind permission.

HOW SHOCKED IS THE PATIENT?

Don't rely on one sign only: assess the overall picture. Check the BP, the pulse rate and its quality, the skin colour, the peripheral temperature, and the presence of sweating. Ask about thirst and assess the mental state.

Later, the amount of urine passed (ml/h) will be your best guide to the degree of shock (44-1).

How full are the peripheral veins?

Empty any convenient superficial vein by pressing it between 2 fingers. Remove your more peripheral finger, and see how fast the empty vein fills up. (It should do so immediately)

How fast is the capillary return in a nailbed?

Apply pressure on the nailbed. If it takes ≥2sec for the colour to return after releasing pressure, this is suggestive of shock.

Is there mottling of the skin? This is a sign of severe acidosis.

What is the interstitial fluid pressure?

Look for: (1) sunken eyes, (2) loss of skin elasticity, (3) lowered eyeball tension, and (4) in severe cases, the classic Hippocratic facies (sunken eyes and temples, pinched nose, and tense hard skin suggestive of impending death). These are late signs.

If the respiration is shallow and rapid (air hunger), there is severe shock.

If you have the means, and if you are sufficiently skilled, insert a central venous line and measure the CVP. This will be useful for monitoring treatment.

INSERTING A CVP LINE

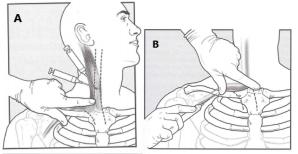


Fig. 44-2 INSERTING A CENTRAL VENOUS LINE. A, using the internal jugular route (direct puncture of the internal jugular vein is shown). B, using the subclavian route.

INSERTING A CVP LINE (GRADE 1.4)

Access may be difficult in a trauma patient, especially if the neck is injured. (In this case, femoral access is preferable, if you have the equipment). Place the patient in 15° of head down tilt. Ultrasound is very helpful as a guide. Identify the junction of the subclavian & internal jugular veins, which lies in a triangle formed by the 2 heads of *sternomastoid* & the medial $\frac{1}{3}$ of the clavicle.

Introduce the needle in the centre of this triangle at 30° to the frontal plane, directing it toward the midline, to a depth of 1-2cm (*but never >4cm*). When blood flows freely into the syringe, advance the guidewire (if you have one) or the long cannula.

N.B. It is useful to have an ECG monitor to check for disturbances of rhythm.

Once connected to a 1L bag, lower this to the floor to check if there is free flow back into the tubing. Fix the CVP line with a suture and get a chest radiograph to check the position of the CVP line, and to rule out a pneumothorax.

An alternative route (44-1A) is directly into the internal jugular vein through the mid-point of the *sternomastoid* advancing the needle at $30-45^{\circ}$ to the frontal plane, or inserting it at the lateral border of the *sternomastoid* $\frac{1}{3}$ of the distance from clavicle to mastoid process, and aim for the sternal notch, advancing it at 45° to the frontal plane & 45° to the border of the muscle.

DIFFICULTIES WITH CVP LINE INSERTION

The most dreaded complication is air embolism, which presents as a sudden drop in cardiac output. You may hear a continuous heart murmur with distended jugular veins.

Turn the patient on his side, administer 100% oxygen, and aspirate the air from the right atrium using the CVP line you have just inserted.

Other complications are:

- (1) Pneumo-, Hydro- or Chylo-thorax,
- (2) Subclavian artery injury, or AV fistula,
- (3) Embolization of catheter fragments,
- (4) Phrenic nerve or brachial plexus injury,
- (5) Cardiac dysrhythmias
- (6) Local infection or bacteraemia.

CAUTION!

(1) A falling blood pressure is a late sign of increasing shock.

(2) Don't administer vasopressor drugs (adrenaline or noradrenaline) before IV fluids.

ORTHOSTATIC HYPOTENSION TEST

A simple way to identify patients with impending haemorrhagic shock is to take the BP and radial pulse while the patient is lying flat), and then again when sitting up. If there is a sharp fall in BP and an increase in pulse rate, the blood volume is depleted. This test may be very useful if you suspect bleeding in a patient who has no other obvious sign.

CORRECTION OF HAEMORRAGIC SHOCK

Generally, administer 1L *warmed* crystalloid IV fast and another if the vital signs have not improved. (This takes a total of 15mins if you use a 14G cannula.) At that point, there is already \geq 40% drop in blood volume and when you have restored the fluid deficit, the haematocrit will have dropped by \geq 40%.

N.B. Colloids, starch, and hypertonic saline are expensive and have significant risks (such as fluid leak into the tissues and electrolyte disturbances. *They have no advantage over isotonic crystalloids* (0.9% saline or Ringer's lactate solution)

After you have infused 2L of fluid, transfuse whole blood (group-specific, if confirmed, or O-ve). Do this earlier if the patient was previously known to be anaemic.

For children, infuse boluses of 20mL/kg warmed isotonic crystalloid fluid at a time. This reflects 20% of their blood volume.

Children with normal haematocrit can tolerate 3 such boluses before blood is needed, but anaemic children need blood after the 2nd bolus.

N.B. Children who are severely malnourished don't tolerate large boluses of fluid well: infuse 10mL/kg of crystalloid and then carefully examine for signs of volume overload or capillary leakage (liver enlargement, pulmonary and peripheral oedema) before adding another bolus.

Estimate the amount of blood lost by the response to the amount of fluids infused (44-5). Those with Class I haemorrhage and some with Class II will respond rapidly with the fluid bolus and will then maintain their blood pressure. These are people who have lost 15 -20% of their circulating volume. They are called **'rapid responders'**.

There are intermediate groups, Class II & III, who will respond initially to boluses of fluid (often ≥2L) but then relatively rapidly become hypotensive and tachycardic again as they continue to bleed and the fluid equilibrates between the intravascular and interstitial compartments. They are called **'transient responders'**. They will need blood in addition to the crystalloid fluid and, very often, will need an operative procedure to control their bleeding.

Non-responders have Class IV haemorrhage: they fail to restore their blood pressure and drop their pulse with fluids. *They always need blood* and almost always need surgery to control ongoing bleeding. 70

CAUTION! Be aware of the '3 for 1' rule. Because of a continued equilibrium between the intravascular and the interstitial compartment, crystalloid infused into the vascular compartment will continue to shift into the interstitial space until it reaches an equilibrium.

Practically, this means you need 300mL of crystalloid to replace 100mL blood lost. The fluid that shifts can cause significant oedema, which is particularly a problem in the brain & lungs.

You must monitor the patient closely to avoid overand under- replacement. In some conditions, even more fluid will be needed to maintain the intravascular volume.

MANAGEMENT OF HYPOVOLAEMIC SHOCK

At this point, you should have finished your primary survey (41.1), taken blood for cross matching, and set up at least 1 (preferably 2) good intravenous infusions, controlled any external bleeding (49.1), added tranexamic acid 1g IV and administered oxygen.

HOW MUCH FLUID TO INFUSE AND HOW FAST?

If possible, replace the volume of blood you calculate is lost. In every class II shock (44-5), infuse 1L in 5mins. If there is class IV shock infuse 2-3L, but *if you don't arrest the bleeding or transfuse blood,* you will make the patient profoundly anaemic!

If the patient is fit and has no cardiac problem, you can transfuse at the most rapid convenient rate until the systolic BP reaches 90mm Hg. *If you push the BP up higher, bleeding may start again!*

If you cannot get venous access in a child <6yrs, try the intra-osseous route (44-3).

INTRA-OSSESOUS ACCESS

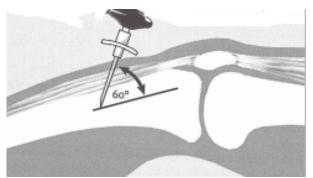


Fig. 44-3 INTRA-OSSEOUS ACCESS. Introduce the needle at a 60^o angle 3cm below the tibial tubercle through its flat antero-medial cortical surface with a twisting motion into the bone marrow. After Nicol, A, Steyn E. Handbook of Trauma for Southern Africa. OUP Cape Town 2004.

Place a pillow under the knee and flex it to 30°. Infiltrate LA, and then introduce the intra-osseous needle at right angles to the periosteum 3cm below the tibial tubercle. Then angle it to 60°, making sure you are inferior to the epiphyseal plate, and twist it into the bone marrow. Aspirate and flush the needle with saline; *check there is no swelling of subcutaneous tissues.* Connect the needle to a fast-flowing IV line and push the needle into the bone & fix it securely with tape.

SAPHENOUS VEIN CUT-DOWN

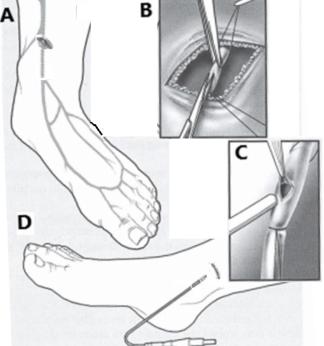


Fig. 44-4 SAPHENOUS VEIN CUT-DOWN. A, the incision 3-4cm above the medial malleolus. B, dissecting out the vein until 2-3cm is visible & passing 2 ligatures under the vein; C, opening the vein cleanly. D, close the incision and fix the cannula securely with tape and a bandage *After Nicol*, *A*, Steyn E. Handbook of Trauma for Southern Africa. OUP Cape Town 2004.

If you cannot access a suitable upper limb vein, try a saphenous vein cut-down. This is more reliable than trying to cannulate the vein blind. After infiltrating LA, make a 2cm transverse incision 3-4cm above the medial malleolus. Pass 2 ligatures under the vein and use the proximal one to lift it up. Tie the distal one. If you have small scissors, make a neat cut into the vein and dilate it with artery forceps & introduce the biggest cannula that will fit. Otherwise transfix the vein with a needle, and cut adjacent to this with a sharp knife to open the vein. Fix the cannula with both previous sutures, and close the incision. Tape the cannula in place and secure it with a bandage (and POP if necessary).

A QUANTITATIVE (A	DULT) FIGURE	S FOR SHOCK &	k ITS MANAGEMENT	
	Class I	Class II	Class III	Class IV
Blood loss	500-750 ml	750-1250ml	1250-2000ml	≥2000ml
(% blood volume)	<15%	15-25%	25-40%	≥40%
Pulse rate	< 100/min	> 100/min	> 120/min	>140/min
Blood pressure	normal	normal	decreased	decreased
Pulse pressure	normal or increased	decreased	decreased	decreased
Capillary refill time	<2 sec	>2 sec	<3 sec	<5 sec
Respiratory rate	14-20/min	20-30/min	30-40/min	>35/min
Respiratory effort	normal	accessory muscles	rib retraction alar flaring	grunting apnoeic attacks
Urine output	≥30 ml/h	20-30 ml/h	10-20 ml/h	negligible
Mental status	slightly anxious	mildly anxious	anxious & confused	confused or lethargic
Skin appearance	pale	grey	dusky	mottled
B QUANTITATIVE (C	,	-		
	Class I	Class II	Class III	Class IV
Blood loss	up to 300 ml	300-850ml	850-1500ml	≥1500ml
(% blood volume)	up to 15%	15-25%	25-40%	≥40%
Pulse rate	>100/min	> 120/min	> 140/min	<50/min
Blood pressure	normal	normal	decreased	collapsed
Pulse pressure	increased	decreased	much decreased	absent
Capillary refill time	>3 sec	>4 sec	<5 sec	none
Respiratory rate	20-25/min	30-35/min	40-45/min	10-20/min
Respiratory effort	accessory	rib retraction	grunting	feeble

Respiratory enort	accessory	TID TELIACLION	grunning	leepie
	muscles	alar flaring	apnoeic attacks	
Urine output	≥40 ml/h	20-40 ml/h	10-20 ml/h	anuria
Mental status	drowsy	confused, irritable	lethargic	coma
Skin appearance	pale, dusky	grey, cyanosed	mottled	mottled, cold

C QUANTITATIVE (TODDLER, 1-3YRS) FIGURES FOR SHOCK & ITS MANAGEMENT

	Class I	Class II	Class III	Class IV	
Blood loss	up to 250 ml	250-450ml	450-750ml	≥750ml	
(% blood volume)	up to 15%	15-25%	25-40%	≥40%	
Pulse rate	>100/min	> 120/min	> 140/min	<50/min	
Blood pressure	normal	normal	decreased	collapsed	
Pulse pressure	increased	decreased	much decreased	absent	
Capillary refill time	>3 sec	>4 sec	<5 sec	none	
Respiratory rate	20-30/min	30-40min	40-50/min	10-20/min	
Respiratory effort	accessory muscles	rib retraction alar flaring	grunting apnoeic attacks	feeble	
Urine output (ml/h)	≥40 ml/h	20-40 ml/h	10-20 ml/h	anuria	
Mental status	sleepy	inconsolable	seizures, asymmetric pupils	coma	
Skin appearance	pale, dusky	grey, cyanosed	mottled	mottled, cold	

72

D QUANTITATIVE (INFANT, <11R) FIGURES FOR SHOCK & ITS MANAGEMENT				
	Class I	Class II	Class III	Class IV
Blood loss	up to 50 ml	50-100ml	100-250ml	≥250ml
(% blood volume)	up to 15%	15-25%	25-40%	≥40%
Pulse rate	>100/min	> 140/min	> 170/min	<50/min
Blood pressure	normal	normal	decreased	collapsed
Pulse pressure	normal or increased	decreased	decreased	decreased
Capillary refill time	>3 sec	>4 sec	<5 sec	none
Respiratory rate	30-40/min	40-50/min	50-60/min	20-30/min
Respiratory effort	accessory muscles	rib retraction alar flaring	grunting apnoeic attacks	none
Urine output (ml/h)	≥30 ml/h	20-30 ml/h	10-20 ml/h	anuria
Mental status	sleepy	inconsolable	seizures, asymmetric pupils	coma, dilated pupils
Skin appearance	pale, dusky	grey, cyanosed	mottled	mottled, cold

E QUANTITATIVE (NEONATE <1 MONTH) FIGURES FOR SHOCK & ITS MANAGEMENT

	Class I	Class II	Class III	Class IV
Blood loss	up to 30 ml	30-50ml	50-80ml	≥80ml
(% blood volume)	up to 15%	15-25%	25-40%	≥40%
Pulse rate	>120/min	> 160/min	> 190/min	<50/min
Blood pressure	normal	normal	decreased	collapsed
Pulse pressure	normal or increased	decreased	decreased	decreased
Capillary refill time	>3 sec	>4 sec	<5 sec	none
Respiratory rate	40-60/min	50-70/min	60-80/min	30-40/min
Respiratory effort	accessory muscles	rib retraction alar flaring	grunting apnoeic attacks	none
Urine output	≥20 ml/h	10-20 ml/h	anuria	anuria
Mental status	sleepy	inconsolable	seizures, asymmetric pupils	coma, dilated pupils
Skin appearance	pale, dusky	grey, cyanosed	mottled	mottled, cold

Fig.44-5 AMOUNTS OF BLOOD LOSS & RESPONSE TO FLUID REPLACEMENT. A, adults. B, children. C, toddlers, D, infants, E, neonates. *Modified from Classification and Management of Haemorrhagic Shock, ACS ATLS Student Course Manual* 9th ed with kind permission.

N.B. (1) In non-adults, bolus replacements IV are better than infusions.

(2) Premature neonates have 90-100ml blood/kg (more than term neonates).

(3) Obese patients have lower blood volumes per weight (ml/kg).

(4) Measure systolic BP with a cuff 40% the circumference of the upper arm.

(5) In children, a normal systolic BP = $[2 \times age(yrs)] + 65$.

RESPONSE TO INITIAL FLUID REPLACEMENT (2I in adults; 20 ml/kg in children)					
	Rapid Response	Transient Response	No Response		
Vital Signs	Return to normal	Show transient improvement; ↑BP and ↓HR, then ↓BP and ↑HR	Remain abnormal		
Estimated blood loss (% blood volume)	Minimal (10 – 20%)	Moderate & ongoing (20 – 40%)	Severe (>40%)		
Need for more crystalloid	Low	High	High		
Need for blood	Low	Moderate to high	Immediate		
Blood preparation	Type & cross-match	Type-specific	Emergency blood transfusion		
Need for operative intervention to control ongoing bleeding	Possible	Likely	Almost certain		

Fig.44-6 RESPONSE TO INITIAL FLUID REPLACEMENT. Modified from Classification and Management of Haemorrhagic Shock, ACS ATLS Student Course Manual 9th ed with kind permission.

If the patient is old, hypertensive, or has cardiac disease, infuse repeated rapid boluses of 100 mL, watching the jugular venous pressure carefully between each infusion. Do this until there are signs that the cardiac output is normal. A change in the JVP or CVP is more important than its absolute value. Listen to the bases of the lungs for crepitations, indicating overload.

If there is a +ve gag reflex and no abdominal injury, you can resuscitate a patient with oral fluids: diluted cereal porridges based on local foodstuffs are ideal.

MONITORING THE URINARY OUTPUT:

It is best to attach a condom catheter (or more reliably, insert a urinary catheter) to measure the output. There is no point doing this if no-one is going to collect the urine & record the output!

If you suspect a urethral injury, insert a suprapubic catheter (55.1). *Don't try to pass a urinary catheter into a damaged urethra!*

Aim to get an output of ½mL/kg/h (1mL/kg/h for a child, 2mL/kg/h for an infant, and 3mL/kg/h for a neonate). This is the most useful indication that you have treated hypovolaemic shock adequately.

Examine the first urine from this catheter. Look at its colour, murkiness & especially for blood and, if possible, culture it.

If, later, no urine appears in the bag, make sure that the catheter is not kinked, and the system is open and properly functioning.

If the catheter only produces a little urine and some blood, suspect that there is a bladder or urethral injury (55.17).

Start a FLUID BALANCE CHART.

WHEN HAVE YOU INFUSED ENOUGH FLUID TO A SEVERELY SHOCKED PATIENT?

The skin should become warm, dry, and the mucosa, nailbeds and palms will become pink, instead of being cold, damp and white. The nail beds should fill up in <2secs and the nose tip becomes warm.

Don't overinfuse fluids, because if secondary bleeding occurs, the patient will have used up all his reserve! It is a good idea to aim at 80mm Hg systolic pressure; *perfusion may still be inadequate* as the patient's blood is now diluted.

Loss of consciousness will not occur from volume loss unless the BP drops <60mm Hg; it will take only 1-2h of severe hypotension to cause irreversible kidney damage.

MONITORING THE CVP:

Inserting a central venous line is a skill made much easier by ultrasound guidance. This will enable you to measure the central venous pressure (CVP).

If a patient is hypovolaemic, you can infuse fluid safely and rapidly until the CVP rises to 12cm H₂O. If it is >15cm, you are overinfusing fluid, or the heart is failing.

If the CVP rises, but the BP and peripheral circulation don't improve, you may have to use IV 5µg/min noradrenaline, *but this needs a pump and careful monitoring.*

METABOLIC ACIDOSIS

In shock, metabolic acidosis results from inefficient anaerobic metabolism of pyruvate to lactic acid. *This is a more reliable sign of shock than blood pressure.* It is best to use arterial blood gas measurements to get an accurate pH value. Correction of shock allows pH to normalize; you very rarely need to administer IV bicarbonate, even if the pH < 7.2.

CAUTION! Acidosis causes coagulation problems.

BLOOD TRANSFUSION

Warm the blood you transfuse. *Don't infuse it thawed (but still cold) straight from the fridge:* (it may cause ventricular fibrillation). A safe way to warm blood is to pass it through 2 IV drip sets connected together through a water bath at 37°C, measured with a thermometer.

Slowly infuse 5mL 10% calcium chloride, or 10mL 10% calcium gluconate IV for every 4 units of blood you transfuse. *N.B. Rapid infusion may produce vasodilation, bradycardia, cardiac arrhythmias, syncope, hypotension & cardiac arrest*

44.2 External bleeding

CONTROL OF ONGOING BLOOD LOSS IS YOUR HIGHEST PRIORITY!

You *must* determine where the bleeding is coming from. To find the source of the bleeding, remember the mnemonic:

'Blood on the floor and four more'

Blood on the floor:

100 mL of blood covers c. $1000cm^2$ (or $1ft^2$). 1L covers c. $1m^2$. The most likely sources are scalp injuries, large lacerations and broken bones.

'Four More' are the chest, abdomen, pelvis and long bones (44.3)

EXTERNAL BLEEDING (49.1)

It is difficult to judge the amount of external blood loss accurately but always remember that significant blood loss may have occurred at the scene of the injury. The history from the patient or from the first-responders will be of some help.

CONTROL OF EXTERNAL BLEEDING:

CAUTION: Don't let dramatic external bleeding draw your attention from Airway and Breathing as your first priorities.

Methods you can use are: (a) Elevation

If a limb is bleeding, raise it. This will usually control venous bleeding. If the wound is in the upper part of the body, sitting the patient up may help, but be careful that it does not cause fainting. **(b) Pressure**

Your gloved finger (or someone else's) is usually the best option to stop external bleeding. Adequate localized pressure stops almost all kinds of external bleeding. For arterial bleeding, this works best at certain sites (49.1)

ISRAELI BANDAGE

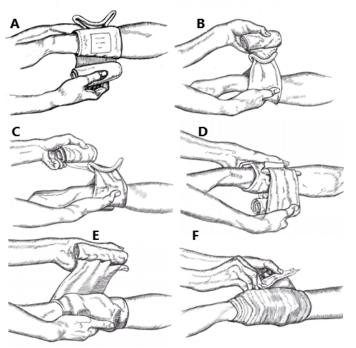


Fig. 44-7 CORRECTLY APPLYING AN ISRAELI BANDAGE. A, Place the bandage with the non-stick pad (opposite the label which states 'other side') on the wound, and wrap it round. B, insert the bandage into the clip (pressure bar). C, tighten the bandage. D, reverse direction of the bandaging, forcing the clip down against the pad. E, wrap the bandage round fully covering the pad. (You can twist the bandage for greater pressure, *but this might convert this into a tourniquet!*) F, Secure the system by hooking ends of the closure bar onto the elastic bandage. *After Bar-Natan, B. The Emergency Bandage 1995.*

N.B. The bandage comes in 3 different sizes: 10,15, 20cm wide.

75

Compression dressings, as traditionally used, are notoriously ineffective on non-extremity wounds. They often soak up & hide bleeding more than they stop it. They fail on trunk wounds because they don't apply focused pressure; the bandage often sits ineffectively on top of the skin rather being put circumferentially.

An elasticated bandage is essential, the most effective type, especially in combat situations, is the 'Israeli Bandage', This has a non-adhesive bandage pad sewn in, which prevents re-bleeding when you remove it. *Don't do anything more until you have waited for at least 5mins by the clock*, unless a torrent of blood pours from the dressing (which will only occur if you have applied the Israeli bandage incorrectly, or you have used inadequate pressure). If bleeding stops, be thankful and *don't meddle with the dressing*.

N.B. A tight dressing might act like a tourniquet, so note when it was applied, and remove it before too long!

(c) Packing

Use this to control deep inaccessible bleeding. Use broad strips of folded gauze (soaked in benzoin for its sealant effect, or iodine for antisepsis), and place these systematically in the wound, *not as a rolled up wodge*. If necessary, hold the pack in place with deep sutures. Here you can make good use of haemostatic agents, if they are available.

SUBFASCIAL PACKING with proximal compression can arrest severe bleeding from highenergy penetrating injuries and amputation wounds.

BALLOON TAMPONADE

Where haemorrhage is welling up from a deep knife wound or gunshot track, put a Foley urinary catheter (or sterilized condom) into the track as far as possible, and inflate the balloon, and then apply traction to it. Suture it in place if necessary, and close the skin hole, as a temporary measure.

N.B. You can also use this in the cervix or uterus for gynaecological haemorrhage (22-10).

(d) Tourniquet

Use a tourniquet in massive exsanguination but remember it causes ischaemia & the danger of reperfusion injury. Don't use it unless direct compression is ineffective. Take the victim to the operating theatre as soon as possible afterwards.

Tourniquets are useful in a mass casualty setting; it is better to lose a limb and save a life! They may also have a temporary role in an overstretched and understaffed emergency situation.

A FIELD TOURNIQUET

Improvised tourniquets don't usually work well. Replace an improvised or makeshift tourniquet by a commercial, or pneumatic one, *as soon as possible.*

N.B. Direct pressure on the brachial artery (in mid-humerus) or femoral artery (at the groin) may temporarily slow distal bleeding, but ties up one rescuer. If you are alone, put pressure on the mid-arm or groin with your knee as you kneel over the patient while getting ready to put on the tourniquet.

AN EFFECTIVE TOURNIQUET needs:

(1) Material: this is a band of some sort to wrap around the extremity. Good options are 3-4cm wide: tie, scarf, bandana, any fabric long enough to wrap around limb, nylon webbing or elastic bandage, and 10-20cm long.

N.B. Poor choices include: belts, cables and other narrow devices. Narrow belts require more pressure and can cause more damage, especially to nerves.

Don't try to tighten it with the buckle itself: this will not work. The material needs to be loose enough to slip a windlass under it to twist.

(2) Windlass: this is a rigid object used to twist the material (*hand tightening is ineffective*) so that the grip tightens. It should be sturdy and ≥15cm long. Good options are: a car jack handle, sturdy stick, or shortened broom handle.

(3) Securing mechanism for the windlass: this is something to keep the windlass from unwinding after you have tightened the tourniquet. It must secure the windlass to the band by slipping around the windlass. Suggested options are: another strip of cloth with or without a rubber band, hair band or ribbon, zip-tie, key ring, carabiner, plastic ring from an opened water bottle, etc.

THE COMBAT APPLICATION TOURNIQUET (CAT) is the most popular tourniquet for out-ofhospital use. It is made of a self-adhering band with a windlass strap, a rod and a clip. It costs c. US\$15. To use it properly, you should:

(1) first put direct pressure on the bleeding site for 5mins, if possible. If the wound is still bleeding, proceed to place the tourniquet,

(2) place the band 5-8cm proximal to the wound (or at the level of the lowest compressible tissue),

(3) pull the strap through the buckle and pull it snugly (44-8B); double it back, using the hook-and-loop to fasten it,

(4) turn the windlass in either direction (44-6C) until the bleeding stops and the distal pulses are no longer palpable. *Don't turn it >5 times!* (Each turn may deliver 100mm Hg pressure),

(5) hook the windlass under the clip (44-6D),

(6) close the clip and **record the time of application** on the limb or the tourniquet (44-6E), with a permanent marker, and

(7) check for absence of distal pulses.

You must always do the last 2 things!!

DIFFICULTIES WITH TOURNIQUETS

Wider compression straps at lower pressures produce less injury to nerves, blood vessels & tissue than narrow tourniquets (*e.g.* cables, ropes, narrow belts) at higher pressures.

Don't use more pressure than is necessary to stop arterial bleeding and obliterate distal pulses. Use a wide-enough band as the tourniquet.

If the tourniquet is not effective, use a 2^{nd} tourniquet proximal to the 1^{st} rather than tightening the 1^{st} one further.

If you still need the tourniquet on arrival to the hospital, replace it with a wider pneumatic tourniquet. Place it proximal to the most proximal bleeding site where tissue compression is possible: *not as "high and tight" as you can.*

SECURING A COMBAT TOURNIQUET

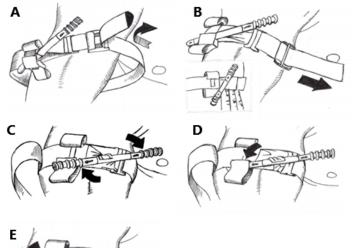


Fig. 44-8 SECURING THE COMBAT APPLICATION TOURNIQUET. A, pull the strap through the buckle, and B, pull it snugly so you can't pass 3 fingers under the strap. C, double the strap back through the clip. D, turn the windlass till distal pulses are no longer palpable, and hook the it under the clip. E, replace the band through the clip over the windlass & record the time of application. After Crown E, US Army Medical Materiel Agency Public Affairs, Fort Detrick, Maryland, USA.

N.B. Tourniquets can be life & limb savers when properly used but you MUST use them properly.

INDICATIONS FOR TOURNIQUET USE

(1) life-threatening or multiple bleeding wounds in a limb,

(2) a wound which is not accessible (*e.g.* patient is trapped in a vehicle),

(3) lack of time because of other severe problems,

(4) failure of simpler measures.

If you use a pneumatic cuff, put soft cotton to protect the skin surface. Pump the cuff slightly higher (not >80 mm Hg) than the patient's systolic blood pressure. Bleeding will stop if you have applied the tourniquet properly.

CAUTION! If it only impedes venous backflow (not enough pressure!) bleeding will increase.

CAUTION! Never use a tourniquet when simple pressure or a pressure dressing can control the bleeding. Multiple bleeding sites, major arterial bleeding, traumatic amputation and multiple fractures are likely to be indications for their use.

CAUTION! Never partially apply a tourniquet! Always apply with sufficient constriction to occlude arterial bleeding completely. If it does not occlude all bleeding, take it off after you have put another tourniquet more proximally.

CAUTION! Never forget you have applied a tourniquet. Always time the application & remove it as soon as safely possible.

CAUTION! Systolic blood pressure may increase with effective resuscitation and result in re-bleeding. You may need to re-apply the tourniquet.

CAUTION! You may need to perform a fasciotomy (49.8) later.

CAUTION! If you leave a tourniquet for $\geq 6h$, the limb involved is at great risk of ischemia and/or nerve damage and eventual amputation.

CAUTION! Don't loosen the tourniquet periodically! This worsens ischemia-reperfusion injury.

CAUTION! Don't leave a tourniquet >1½h on the arm or >2h on the leg. (30min & 45min respectively for thin adults & children.)

If the accident-to-definitive-care time is <2h, leave the tourniquet in place the entire time. However, under good conditions, remove it <2h if possible. Ischemic tissue damage begins around this time.

If the patient is no longer in shock, let down the tourniquet if:

(1) bleeding is controlled and you can observe the victim;

(2) a haemostatic dressing is effective;

(3) evacuation is prolonged >6h; or

(4) you can relocate the tourniquet more distally to preserve extremity length.

Don't let the tourniquet down before definitive care if (1) there is an amputation or known arterial injury, (2) the tourniquet has already been in place ≥6h, or (3) to let the tissues 'recover'. Intermittent release of a tourniquet increases the risk of myoglobinaemia, myoglobinuria & renal shut down!

Proceed quickly to vascular control or repair (49.1)

ALWAYS NOTE THE TIME WHEN YOU HAVE APPLIED THE TOURNIQUET!

Don't forget analgesics: a correctly applied tourniquet is always painful!

The scalp can bleed profusely. Stapling or rapidly closing the skin with a running haemostatic suture is useful. You may need to make a more definitive exploration later when the patient has stabilized. The same applies to the face.

Never try to use diathermy in the emergency situation: it causes further necrosis to the tissues.

Never use haemostatic (arterial) forceps blind: they can easily cause damage to nerves, veins and other issues.

If, however, you can see a bleeding vessel clearly, try to isolate it and clamp or ligate it.

N.B. Haemostatic agents are expensive, and don't take the place of a pressure dressing.

This means you must know how to dissect out vessels correctly, control them, and know whether to ligate them or attempt a shunt or repair (49.5). This may also mean an amputation at the appropriate level (60.1) or a fasciotomy (49.8).

44.3 Internal bleeding

The 'Four More' causes of bleeding:

BLEEDING IN THE CHEST

The chest can easily hold 3L blood or more. A patient can lose all his blood easily into the thorax.

Suspect a haemothorax if air entry is poor, the chest is dull to percussion, there is clinical evidence of rib fractures, or an ultrasound scan or chest radiograph suggests this (43.2).

Insert a large chest drain (Ch32-36) and if there are large volumes of blood to drain, a 2^{nd} drain.

If you are uncertain, put in a drain! Use the blood from chest drains for auto-transfusion (5.3). Simply attach a collecting bag (*e.g.* a urine bag) to the chest drain tube *via* a 2-way tap, and re-transfuse the blood.

BLEEDING IN THE ABDOMEN

The abdomen can easily hold 3L of blood or more. A patient can lose all his blood easily into the abdomen.

Since bleeding is not visible and you may well not know what the abdomen looked or felt like before the injury, you must always suspect abdominal bleeding in a shocked patient.

Abdominal distension and marks from the seat-belt or steering wheel are the most common signs of significant intra-abdominal bleeding in motor crashes.

A doughy consistency of the abdomen may indicate bleeding. Peritoneal irritation may suggest an associated visceral injury (which often causes some bleeding).

Remember that the liver and spleen are under the rib edge; obvious trauma to the lower chest and ribs may damage these structures, especially if there has been significant impact, *e.g.* against a steering wheel, a seat, a seat-belt, or the handlebars of a cycle.

However all these signs take some time to be obvious. *Always keep re-evaluating the patient!*

N.B. Unrecognized or missed intra-abdominal injuries remain amongst the leading causes of potentially preventable trauma deaths worldwide.

ULTRASOUND

The FAST (Focused Assessment with Sonography in Trauma) examination is a skill you can easily learn. Fluid shows up black on the scan. Use a lowfrequency (3.5MHz) curvilinear transducer. If some views are not good (particular those through the ribs), it is sometimes necessary to switch to a small cardiac probe to see between the ribs. There must be 200mL blood in the peritoneal cavity for this test to be positive.

However, if you only find such small amounts of fluid, this is *not* the cause of hypovolaemic shock; keep looking for another reason.

Repeat examinations at various intervals if you suspect ongoing bleeding.

N.B. Clotted blood will not show up as fluid, and blood in the retroperitoneal space is not readily visible. Also injury to solid or hollow organs is difficult to visualize.

Use 4 views (called acoustic windows):

(1) Place the probe in the sub-xiphoid area (below and to the right of the xiphoid process), looking toward the heart. Look for a fluid collection in **pericardium** outside the heart. If this view does not give good images, try a sagittal (longitudinal) parasternal view of the heart. (2) Place the probe over the posterior axillary line over the 10th rib. Orient the probe as before. Look for a fluid collection between spleen and kidney (**perisplenic** area).

(3) Place the probe in the right mid-axillary line (a **perihepatic** view of Morrison's pouch). Orient the probe to get a coronal view looking somewhat up). Look for a fluid collection between liver and kidney.

(4) Place the probe transversely over bladder (**pelvic**): look for a fluid collection outside the bladder. *Make sure to do this before there is a catheter in the bladder*. In females, fluid usually collects in the pouch of Douglas behind the uterus. This FAST examination is positive if you find free fluid in *any* of the 4 acoustic windows, negative if you see no fluid, and indeterminate if you cannot assess any of the acoustic windows. **If you are uncertain**, repeat the scan after 10mins.

If the FAST scan shows free fluid in the abdomen, take the patient to theatre. Do this urgently without delay! Even if the patient is haemodynamically stable, you should still explore the abdomen, rather than wait for shock to occur!

FAST SCAN

FAST view: Normal FAST view: Free Fluid

Fig. 44-9. FOCUSED ABDOMINAL SCAN: The four key windows for FAST and their schematic ultrasound views. 1, pericardial (subxiphoid). 2, perisplenic, 3, perihepatic. 4, pelvic. Hui A. www.ashleyhui.com/Toronto-notes- 2015 with kind permission

N.B. FAST does not readily show injuries to the pancreas, diaphragm, or an isolated perforated hollow viscus. Views may be restricted by the presence of excessive gas in the bowels, obesity, massive ascites, and surgical emphysema.

If the ultrasound scan is unhelpful or not available, and the patient is haemodynamically unstable, check for blood loss elsewhere (44.2) before proceeding to a diagnostic peritoneal lavage.

DIAGNOSTIC PERITONEAL LAVAGE (DPL) (GRADE 1.4)

This is also valuable in settings where ultrasound is not available N.B. This is not suitable for perforating abdominal injuries!

INDICATIONS

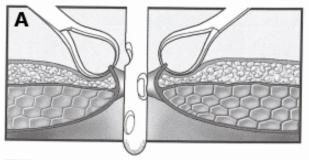
Patients with a spinal cord injury.

- Multiple injuries and unexplained shock, and you need to assess priorities.
- (3) Intoxicated patients or those with impaired conscious level with possible abdominal injury.
- (4) Patients with potential intra-abdominal injury
- who are having anaesthesia for another procedure.
- (5) Indeterminate FAST examination.

RELATIVE CONTRAINDICATIONS

- (1) morbid obesity,
- (2) 3rd trimester pregnancy,
- (3) previous lower abdominal surgery,
- (4) advanced cirrhosis
- (5) coagulopathy.

DIAGNOSTIC PERITONEAL LAVAGE (DPL)



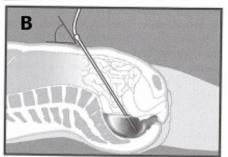


Fig. 44-10 DIAGNOSTIC PERITONEAL LAVAGE (DPL). A, Elevate the edges of the abdominal sheath, and make a small incision in the peritoneum in order to insert the peritoneal catheter under direct vision. B, Guide the catheter at 45^o into the pelvis. After Nicol, A, Steyn E. Handbook of Trauma for Southern Africa. OUP Cape Town 2004.

TECHNIQUE OF DPL

Check a pelvic radiograph to see whether the patient has a pelvic fracture before starting. A false-positive result can occur if you use an infraumbilical approach in a patient with a pelvic fracture, because you may enter the haematoma in the preperitoneal space anteriorly.

Insert a urinary catheter and a nasogastric tube to empty the bladder & stomach. Prepare a warm 1L bag of sterile IV 0.9% saline (you can use sterile water *in extremis*), attached to a collecting bag. Infiltrate LA down to the peritoneum & use a sterile technique. Make a 3-5cm sub-umbilical midline incision, extended to and through the *linea alba*. Place haemostats to control any skin bleeding. Make sure this control is perfect.

N.B. In the pregnant woman, place the incision above the umbilicus.

Open the peritoneum under direct vision and insert the peritoneal tubing (which can be a catheter or IV line) towards the pelvis. Place a purse string in the peritoneum to close the hole and avoid leakage. First use a large syringe and attempt to aspirate free intraperitoneal blood from the tube.

If you obtain ≥10mL of grossly bloody aspirate *before* you start lavage, the test is +ve. Proceed then to laparotomy in the operating theatre.

If you aspirate nothing or <10mL thinly bloodstained fluid, run 1L of warm fluid that you have prepared rapidly into the abdomen through the tubing, gently agitate the abdomen for 2mins (or tilt the patient up & down and side to side) and then lower the empty collecting bag onto the floor. Fluid will run out through the tube.

If you see blood-stained fluid, the test is +ve. You may need laboratory analysis to check the results, which are positive if the siphoned lavage fluid has:

- (1) >100 RBCs/mL,
- (2) >500 WBCs/µL,
- (3) amylase >20IU/L,
- (4) bile,
- (5) bacteria,
- (6) bowel content or vegetable matter,
- (7) urine.

In the meantime, try to read newspaper print through the IV tubing. If you cannot, the test is almost certain to be +ve.

If the DPL is taking time to set up and the patient is becoming shocked, abandon it and proceed to laparotomy!

COMPLICATIONS OF DPL include:
(1) bleeding from the incision
(2) bleeding from insertion of the tube
(3) infection in the wound or peritoneum
(4) injury to intra-abdominal viscera
These complications may increase the chance of false-positive studies.

DPL is very accurate, sensitive and specific, but apart from the risks described, it may be so sensitive, you may end up performing an unnecessary laparotomy. Only c. 30mL blood makes the DPL test positive. So if bleeding from the procedure contaminates the fluid, it can produce a false-positive result. Even so, a -ve result does not mean there cannot be an abdominal injury.

If peritoneal lavage fluid comes out of a chest drain or urinary catheter, perform a laparotomy!

RETROPERITONEAL & PELVIC BLEEDING **Bleeding in the retroperitoneum** is very difficult to detect by examination or ultrasound.

Abdominal pain, abdominal distension, the presence of an abdominal mass, severe back and lower quadrant pain and femoral neuropathy are all nonspecific signs and symptoms.

Ultrasound may pick up <50% of cases. Bruising in the flanks (Grey Turner's sign) or the umbilicus (Cullen's sign) are usually delayed for several hours to days.

PELVIC FRACTURES, especially open-book fractures, can cause life-threatening bleeding. Examine the pelvis only once as *repeat examinations will only increase bleeding*. Use a bed sheet as a binder to close the 'open book'. Place the sheet at the level of the hips (greater trochanters), *not higher*.

Apply and tighten the sheet carefully, at least until you get a radiograph. You may worsen bleeding from lateral compression fractures if you apply excessive force.

BLEEDING IN LONG BONES

Multiple fractures or fractures in the femur are the most likely source of severe blood loss from bones. The average adult blood loss in closed fractures is: (1) Radius & ulna: 150-250mL

(2) Humerus: 250-500mL

(3) Pelvis: 1½-3L

(4) Femur:1-2L

(5) Tibia and fibula: ¹/₂-1L

Appropriate splinting or traction is the best method of reducing further blood loss until more definitive treatment is possible. 80

You can therefore calculate the revealed total blood loss: *e.g.* if there is a haemothorax of 1.5L, and a fractured femoral diaphysis (1L), and you have transfused 5 units of blood, but the patient is still hypovolaemic, it is likely that there is another source of blood loss which you have missed. This is likely to be abdominal or pelvic.

N.B. Bleeding inside the head of an adult can never account for enough blood loss to cause hypovolaemic shock: you must look elsewhere. The situation is quite different in a small child, where the size of the head is proportionately much larger compared with the rest of the body.

THE CHEST RADIOGRAPH

We have deliberately left this till last, as the benefit from X-raying the chest in trauma is not very high for the effort and time incurred. Unless you can take a radiograph in the resuscitation area, *don't bother with it for the shocked patient*!

The ultrasound scan is more sensitive in detecting free liquid in the chest and abdomen.

These are the features to look for, which suggest abdominal injury may be present:-

(1) A positive FAST

(2) Fractures of the lower ribs

(3) Free air under the diaphragm (erect film)

(4) Foreign bodies (e.g. bullets)

(5) Fluid or air in the pleural spaces

(6) Abdominal viscera in the thorax.

(7) A 'ground glass' appearance between loops of bowel.

(8) Fractures of the pelvic bones

(9) A raised hemidiaphragm

(10) A dented or displaced gastric shadow

Many of these are subtle signs, and not very reliable, which you must add to your clinical impression.

44.4 Autotransfusion

Blood from clean traumatic injuries of the chest or abdomen, or from an ectopic gestation, is ideal for auto-transfusion; it can be life-saving. Also, it is warm and has all the necessary clotting factors and carries no risk of hepatitis or HIV, and it will be perfectly cross-matched.

Autotransfusion may be life-saving even more in the operating theatre scenario (5.3). Rapid blood loss of \geq 1L blood should make you start thinking of autotransfusion, especially if cross-matched blood is not available or in short supply.

You don't need sophisticated equipment. A simple collection bag system (44-11) is ideal. Re-infused blood recovers its clotting capacity within 24-72h.

Blood from the thorax is defibrinogenated and so does not clot, unless it is from the major vessels (when there isn't enough time for defibrinogenation), and so you can use it up to 72h after injury! One filtration is usually adequate.

N.B. If the blood has been lying in the thorax or abdomen for >12h, and the patient has compensated haemodynamically, IV crystalloids are probably needed rather than this blood autotransfused, although you can still use it for blood transfusion.

However, blood from the abdomen and the limbs needs filtering through 6-8 gauze layers (5-1).

Blood from the thorax does not need anticoagulant, but blood from the abdomen needs a half dose of citrate (in blood transfusion bags) or 1000 units of heparin per 500ml of blood, although this is not absolutely necessary if not available.

(a) Thorax

In extremis, the simplest method is to drain blood from the thorax into a sterile chest drain bottle containing 100mL normal saline. Then disconnect this, invert it, and connect it to an IV giving set. At the same time connect both chest drain bottles and repeat the process.

If you have a lesser degree of urgency, drain the blood from the thorax or abdomen through 6-8 layers of sterile gauze, into a sterile bottle, or better, an empty 1L bag. Then infuse this, preferably through a standard blood transfusion filter of 150-200µm.

(b) The abdomen

Ladling blood through a funnel (5-1) is timeconsuming and inefficient; it is better to use suction *at low pressure*, as the higher the pressure the greater the haemolysis. *Keep the sucker under the fluid surface*, so as not to suck up air and cause frothing and risk of more haemolysis & air embolism.

In extremis, if you have no alternative and the blood is contaminated with gastric juice, bile, faecal, or foreign matter, remove the gross contamination manually & filter out the particulate matter (because this is the cause of DIC). Then pass the blood through 2 filters, as before, and administer broad spectrum antibiotics IV simultaneously. Don't get glove powder mixed in with the blood!

Whilst there are risks of fever, clotting disorder, renal failure (from haemolysis), sepsis & ARDS, the advantages of autotransfusion when blood transfusion is necessary far outweigh its risks.

AUTOTRANSFUSION EQUIPMENT



Fig. 44-11 AUTOTRANSFUSION EQUIPMENT. A sterile 2L abdominal drainage (or even urine) bag can collect the blood through one port and re-infuse it through the other (drainage) port, using a blood giving set with a filter. After Kothari R, Pandey N, Sharma D. A simple device for whole blood autotransfusion in cases of hemoperitoneum and hemothorax. Asian J Surg 2019; 42: 586-587.

44.5 Septic & other kinds of shock

SEPTIC SHOCK

Septic shock is a life-threatening condition caused by a severe localised or system-wide infection causing organ failure that requires immediate medical attention.

This is a type of distributive shock, caused by loss of normal arterial vasomotor tone, which leads to an alteration in the afterload. As the systemic vascular resistance drops, the cardiac output must increase in compensation (in contrast to other types of shock).

Sepsis, anaphylaxis and amniotic embolism may all have similar physiologic presentations, despite differing aetiology.

Cytokines and bacterial toxins generally cause disruption of capillary cell integrity, resulting in a secondary leakage of fluid from the intravascular space into the tissues. This results in hypovolaemia and oedema. It can be difficult to differentiate between septic and hypovolemic shock.

Only patients in early sepsis will have a normal circulating volume, modest tachycardia, warm skin, nearly normal systolic pressures and a wide pulse pressure.

Septic shock in trauma patients is uncommon and usually occurs usually in patients with peritonitis who have a delayed presentation.

ADEQUATE FLUID RESUSCITATION

Infuse \geq 30mL/kg IV crystalloids (*i.e.* 1.5-3L) within 1h of making the diagnosis. Be aggressive with this; several patients will need much more fluid.

Aim for a urine output of ≥30mL/h. The normal 2:1 ratio between the interstitial fluid volume & the vascular compartments may increase to 10:1.

Fluid may leak not only into the tissues but also into the lungs, which may then require post-pressure ventilation.

Watch the Na⁺ & especially the K⁺ levels; if you can, correct them.

Don't try to obtain 'normal' BP values as long as the brain is being well perfused (normal conscious level) and urine output is adequate. A systolic pressure of 80mm Hg may still be adequate.

N.B. The *BP* may not respond well to vasopressors. Start noradrenaline to target a mean arterial pressure of 65mm Hg.

ANTIBIOTIC TREATMENT

Antibiotics and drainage of any source of infection is critical (6.1). Take blood cultures and culture pus from any possible source.

Cover Gram negative & positive organisms and anaerobes. Choose drugs depending on known antibiotic sensitivities at your hospital.

If you don't know the antibiogram at your hospital, try these suggestions. Use large doses of \geq 3 bactericidal antibiotics, if possible IV, as bolus injections, every 3h, & every 10 units transfused.

Choices for Gram-positives include:

(1) Chloramphenicol 1g (12.5mg/kg in children) qds IV, or streptomycin 500 mg qds.

(2) Ceftriaxone 2g (50-80mg/kg in children) od slowly IV. This covers most Gram-negatives except *Pseudomonas* and *Acinetobacter.*

Don't use it with Ringer's lactate as it calcifies in the veins.

Choices for gram-negatives include:

(1) Gentamicin 5-7mg/kg od slowly IV. (In renal failure increase the interval between the doses.)
(2) Amikacin 15mg/kg bd (up to 22.5mg/kg tds) slowly IV. Use 7.5mg/kg bd in children.
For anaerobes, use metronidazole 1g tds PR, or bd IV. For a child use 7.5mg/kg tds either PR or IV.

N.B. Avoid quinolones & glycopeptides.

Control the fever with tepid sponging and oral or rectal antipyretics. Support the hypermetabolism which occurs with oxygen by a mask.

PULMONARY OEDEMA

This is characterized by poor oxygen saturation, crackly lung sounds, and frothy sputum. Use large doses of furosemide (100-200 mg bd or tds) but *be warned that they may not be effective*.

If acute left ventricular failure develops despite vasopressors, try digoxin at 0.125-0.25mg PO/IV od; you may rarely need to increase these doses up to 0.375-0.5 mg/day.

For patients with impaired renal function or low lean body mass, use low doses. If a pulse deficit (difference in apex beat & pulse) develops, he has excess digoxin.

If cardiac output remains low despite high CVP,

add a dobutamine infusion, if you can, at 10-20µg/kg/min to any vasopressor in use. *Don't use dopamine in low doses.*

Steroids in sepsis are of doubtful value.

This is a last ditch effort: use hydrocortisone 50mg IV qds, a dosage designed to replace normal adrenal function.

ANAPHYLACTIC SHOCK

This is another type of distributive shock where vasomotor tone is affected.

Use adrenaline IM 0.01mg/kg (maximum 0.3 mg in a prepubescent child, and 0.5mg in a teenager/adult) into the *vastus lateralis* muscle of the thigh.

N.B. Many people with allergies have this medication (as an 'epipen') in a pocket or handbag. Remove the source of the antigen if known.

In addition:

(1) Use nebulized adrenaline for laryngospasm.

(2) Use nebulized albuterol or salbutamol for bronchospasm

(3) Use histamine-1 & 2 blockers together, *e.g.* diphenhydramine or hydroxyzine; plus ranitidine or cimetidine.

(4) Corticosteroids have no immediate effect! However, administer them early to prevent a potential late-phase reaction (biphasic anaphylaxis). Use prednisolone at 1mg/kg in divided doses and, in children, 0.5-1mg/kg per day. A tapering regimen is not necessary unless the patient has been taking steroids chronically.

AMNIOTIC FLUID EMBOLISM

Similar treatment as for septic shock is needed with large volumes of IV fluids as well as vasopressors.

Correcting coagulopathy may not be possible with your resources.

A Caesarean section is indicated at 23/40 gestation if the mother bleeds profusely without stopping, but these are heroic last ditch efforts.

FAT EMBOLISM

This is the dreaded complication of pelvic & closed long bone fractures, usually in young well-built adults, or sickle disease children (58.16).

It can also occur in pancreatitis, liver injury, during orthopaedic surgery, or liposuction, and in decompression injury. The victim becomes confused, without neurological signs, restless and hypoxic as fat globules pass to the lungs from long leg veins.

You may see tell-tale signs of petechiae on the chest & in the mouth & conjunctivae, and fat globules in the urine, sputum & veins on retinoscopy. There may also be 'cotton wool' exudates and retinal haemorrhages. Human albumin IV may help. Renal & respiratory function deteriorate; high-flow oxygen with ventilatory support is usually necessary, and the outcome is fatal in up to 50% of cases. *Don't use heparin!*

ARTERIAL GAS EMBOLISM

When a diver resurfaces too quickly from a longer period in the depths, the inert gas (nitrogen & helium) which has dissolved in the blood at high pressures below the water surface rapidly comes out of solution. It literally bubbles into the blood, and so blocks its flow.

This is also known as barotrauma or decompression sickness. It is more common the longer or deeper a diver has been, and the quicker he surfaces. Most recommendations are to surfaces at rates <10m/min.

The risk is greater in people with diabetes, cardiovascular or chronic airways disease, and especially those with a patent *foramen ovale* (which may be quite small, and undiagnosed)

Symptoms are classically pains in elbows, knees and ankles, known as 'the bends', but can be associated with central or spinal neurological deficit: nausea, dizziness, extreme fatigue, confusion, fits, or paralysis.

Treatment is to supply 100% oxygen through a rebreathing system for 12-24h.

CARDIOGENIC SHOCK

This refers to any intrinsic cardiac condition which diminishes cardiac output. In trauma, cardiac contusion (43.5), very rarely in snake bite (46.11) or, less rarely, an associated myocardial infarction or ventricular rupture will diminish cardiac contractility.

Any pre-existing condition may be the cause: dysrhythmias, cardiomyopathy, toxins and medications.

In severe deceleration impact, a cardiac valve replacement may rupture and so cause acute leftsided heart failure (if aortic) or right-sided (if mitral).

Try to get an ECG done, and cardiac ultrasound, if possible

Oxygen, fluid restriction and ionotropic agents may be indicated.

Obstruction to outflow. or diminution of inflow. can occur in cardiac tamponade (44.6), tension pneumothorax (43.2)and rarely tension haemothorax, as well as pulmonary embolism. Extended FAST ultrasound scan can make a quick diagnosis: immediate emergency treatment may be life-saving.

NEUROGENIC SHOCK

Remember that isolated intracranial injuries don't cause shock in adults.

Neurogenic shock occurs in patients with cervical and upper thoracic spinal cord injuries which cause a loss of sympathetic vasomotor tone.

As cardiac reflexes are also disrupted, tachycardia does not occur.

Neurogenic shock is, however, a diagnosis of exclusion.

Treat for hypovolaemia first! Only after you are sure that you have adequately restored the fluid volume, should you add vasopressors.

N.B. Differentiate this from so-called spinal shock, which is a temporary disruption of normal spinal cord function but not associated with hypotension. The speed of functional recovery is highly variable.

ELECTRIC SHOCK

A high current can stop the heart: remember it is amps that vamps and volts that jolts!

Lightning delivers c.50Kamp + 100Gv over 10-100msec at a temperature of 30,000°C. One metre away from where lightning has struck reduces the voltage by c.1Kv, but is obviously still significant. There may also be a side flash. Immediate results may include:

(1) Asystole

- (2) Apnoea
- (3) Blast lung injury

(4) Cerebral haemorrhage

If the victim survives, it is essential to administer external cardiac massage (44.9) and continue till there is a resumption of spontaneous cardiac activity. Don't give up till at least 30mins!

Look for signs of a pneumothorax (43.2) and insert a needle if you suspect one.

An ECG may show ventricular tachycardia, which may respond to 1mg adrenaline IV, atrial fibrillation or just prolonged QT intervals.

Later, check for pulmonary damage on a chest radiograph, evidence of other blunt trauma, neuropathy, hearing loss, cataract, burns, or limb compartment syndrome (49.8). The victim may suffer a prolonged ileus.

Similar sequelae may follow an electric current shock.

44.6 Cardiac tamponade

The pericardium can fill up with fluid, pus (9.3) or blood. This may be due to blunt, but more likely penetrating trauma. A stab wound to the anterior left chest (or upwards through the abdomen) must always suggest a heart injury. This is not always immediately fatal.

Tachycardia, muffled heart sounds and distended neck veins are the classic signs (Beck's triad), but sudden hypotension (especially in a young male stabbed in the chest, where personal violence is common) must ring alarm bells.

You may not have time to perform a rapid ultrasound scan to confirm the diagnosis; do this only in a haemodynamically stable patient!

If a victim arrives still alive with a stab wound of the heart, the only way he will survive is if you open his chest!

Bleeding into the pericardial cavity prevents the heart filling normally, which: (1) raises the jugular venous pressure, (2) makes the heart sounds faint, (3) causes pulsus paradoxus (a peripheral pulse stronger on expiration), and (4) hypotension.

The clinical signs depend on the speed at which fluid fills up the pericardial cavity. If this is relatively slow, owing to a puncture of the right atrium (whose pressure is <10mm Hg), the amount of fluid may still allow some cardiac output.

A chest radiograph shows a round, globular heart shadow (44-12), but this is not very specific, and a late sign. Ultrasound is much clearer (44-13-15).

PERICARDIAL TAMPONADE



Fig. 44-12 RADIOLOGICAL SIGN OF PERICARDIAL FLUID. A globular shaped heart needs a considerable amount of liquid to give this shape, so is a late sign.

ULTRASOUND: CARDIAC TAMPONADE

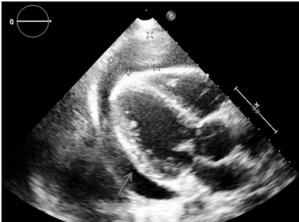


Fig. 44-13 SUBXYPHOID VIEW OF PERICARDIAL FLUID. There is a uniform blackness around the heart. Flocculation suggests clotted blood, or pus.

ULTRASOUND: PERICARDIAL FLUID

Pericardial Effusion RV LV LA Descending Aorta

Fig. 44-14 PARASTERNAL VIEW OF PERICARDIAL FLUID. The pericardial effusion is anterior to the descending aorta.

ULTRASOUND: PLEURAL FLUID

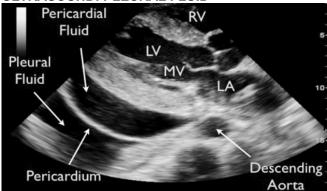


Fig. 44-15 PARASTERNAL VIEW OF PLEURAL FLUID. The pleural effusion is posterior to the descending aorta.

PERICARDIAL TAP (GRADE 2.3)

You might buy time by draining the pericardial cavity. *However, this is inadequate for a perforating cardiac injury.*

Insert a 16G 12cm needle from just under the xiphoid (if possible, with ultrasound guidance), aiming towards the left shoulder and leave a cannula *in situ* (9-4). Watch for an injury pattern from the ECG attached to the needle (or on a monitor). If you see this, withdraw and redirect the needle slightly.

Attach the cannula to a 3-way tap, and aspirate what you can but removal of even relatively small amounts (15-20mL or more) of blood may relieve the pressure on the heart. Leave the cannula in place. Prepare for a thoracotomy urgently.

EMERGENCY THORACOTOMY (GRADE 3.4)

This can, almost miraculously, transform a moribund patient into one who the next day is asking for his breakfast!

The aim is simply to close the (presumed) perforation in the right atrium (or much more rarely, in another heart chamber). Though this may sound daunting, a simple suture will suffice.

You obviously need to open the chest. Do this either through the sternum, or the left chest. A sternotomy needs either a Gigli wire, circular saw, or a Lebsche knife; you should use this only for precordial stab wounds. If you don't have the equipment, or the injury is more complex (particularly gunshot wounds), opt for a left thoracotomy. The difficulty with this is stretching the ribs apart.

(a) Median Sternotomy

You may (initially) need no anaesthesia! Ketamine is the ideal choice.

Make a midline incision from the sternal notch to just below the xiphoid. With your fingers, create a plane for 2-3cm under the sternum both inferiorly and superiorly (44-11).

Hook up the sternum at the lower end, and cut through it along its middle. Put a self-retaining retractor between the two halves of the sternum.

(b) Anterolateral left thoracotomy

Tilt the patient slightly to the right with a pillow under the right shoulder; make a bold incision in 3 strokes through the 4th or 5th intercostals space (below the nipple in a male and along the inframammary fold in the female) from the costochondral junction anteriorly to the mid-axillary line laterally.

OPENING A RETROSTERAL PLANE

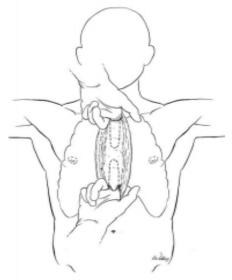


Fig. 44-16 OPENING A RETROSTERNAL PLANE using your fingers prior to a sternotomy. *After Hirschberg A, Mattox KL. Top Knife. Tfm Shrewsbury 2005 with kind permission.*

In the 1st stroke, incise the skin & subcutaneous tissue along the upper border of the lower rib, in the 2nd, divide the pectoralis fascia & *serratus anterior* posteriorly, and in the 3rd, get into the pleural space. Break down any adhesions with your fingers.

Then divide the intercostal muscles with strong scissors. Avoid *trapezius* & *pectoralis major*.

Insert a Finochietto retractor to spread the ribs apart. You may need an assistant to retract the lung laterally. You will now see the pericardium right in front of you. It may not be bulging but still hide a tamponade! N.B.

(1) Bright red blood pooling in the thorax is usually from the chest wall (43-22).

(2) Blue blood is from a pulmonary hilum injury.

(3) A mediastinal haematoma means a large vessel injury.

(4) Blood with air bubbles is from a lung injury.

Open the pericardium superiorly, avoiding the innominate vein & phrenic nerve, and expose the heart. Some blood will gush out; get your assistant to aspirate it. Put a finger over the hole in the heart to occlude it, or a Foley catheter (if it is bigger) and pull on this. Occasionally you can use a Satinsky vascular clamp for an atrial laceration.

If the situation suddenly becomes calm, the patient might even wake up at this stage, so use anaesthesia with ketamine, if you have not done this already!

Repair the heart (but *not* full thickness) with a figure-of-8 2/0 deep non-absorbable suture on a large round-bodied needle which, ideally, you have passed through a small pledget of pericardium; *avoid the coronary arteries!* (44-17) *Don't tie the sutures too tight,* or they will cut out. If the muscle is friable, use mattress sutures.

N.B. The right heart is a low pressure system with thin muscle, so be careful not to let your sutures cut out (tie gently & use pledgets). The left heart is a high pressure system with thick muscle and so is more resilient.

N.B. You may find moving in rhythm with the heart makes it easier to place the sutures.

Check if there is a perforation posteriorly by gently passing your hand under the heart. *Don't lift it up* as this will cause a dysrhythmia!

REPAIR OF A CARDIAC PEFORATION

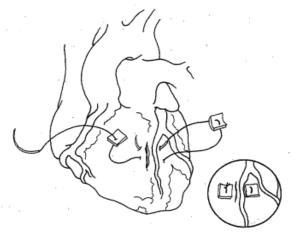


Fig. 44-17 REPAIRING A CARDIAC PERFORATION. Use pledgeted sutures, and avoid the coronary arteries (if they are in the way). After Szul AC, Davis LA (eds) Emergency War Surgery. Dept Defense 3rd US rev 2004.

85

If the heart has stopped, start cardiac compressions with both hands (*not the thumbs*) before attempting to close the perforation. Insert a soft pericardial drain & loosely approximate the pericardium with absorbable sutures. Insert a pleural chest drain, & close the pleura with absorbable sutures.

Close the sternum with wire c. 2cm from its edge, or round the whole bone in the intercostal spaces. Close discrete muscle layers with absorbable sutures, then close the skin.

N.B. Don't attempt to perform these procedures for blunt or gunshot cardiac injury. Remember to ligate the mammary arteries after a lateral thoracotomy!

44.7 Venous thrombosis prophylaxis

Pulmonary embolism (PE) is the scourge of surgery, because it can affect anyone. It can be suddenly fatal. However, certain types of surgery and certain patients are more prone to develop deep venous thrombosis (DVT), and therefore are more at risk of pulmonary embolism, than others.

In order to assess the risk of developing deep venous thrombosis, you can score various factors:

Risk Factor	0-1	2	3-4	5+
Point Score				
DVT	2%	10-20%	20-	40-
Incidence			40%	80%
Risk Level	Low	Moderate	High	Very
			-	High

The following score **5 points** if <1 month previous there was:

- (1) Major lower limb joint surgery
- (2) Hip, pelvis, or leg fracture
- (3) Cerebrovascular accident (stroke)
- (4) Multiple trauma
- (5) Spinal cord injury with paralysis

These score 3 points:

- (1) Age >75yrs
- (2) Previous or family history of DVT or PE
- (3) Past history of MI, CCF or COPD
- (4) Thrombocytosis

These score 2 points:

(1) Age >60, but <75yrs

(2) History of malignancy, current chemotherapy or radiotherapy

- (3) Surgery lasting, including tourniquet >45mins
- (4) Being confined to bed >72h
- (5) Having a leg cast <1month

These score 1 point:

(1) Age 41-60yrs Minor surgery Pregnancy within 1 month Varicose veins or leg swelling Inflammatory bowel disease Obesity (BMI >25kg/m²) Use of oral contraceptives, or hormone replacement therapy
Only 10% of those with DVT extend to PE.

Don't use prophylaxis when the score is <2.

Knee- or thigh-length graduated compression (elastic) stockings prevent DVT to some degree, but are not as effective as anticoagulants in moderate or high-risk cases.

(a) Moderate risk patients (score 2 points).

Use standard (unfractionated) heparin 5000 U SC 2h before surgery, and then bd for 7 days or till ambulant, as well as stockings.

(b) Higher risk patients (score >3 points) Use low molecular weight heparins, heparinoids or fondaparinux, which is a factor X inhibitor. These are expensive, but effective, so you should consider their use before committing a patient with high risk to major surgery (especially elective).

N.B. Heparin may cause thrombocytopenia (an immune reaction) *or hyperkalaemia* (by inhibiting aldosterone) *as well as haemorrhage.*

CAUTION! Don't use low-dose aspirin alone as a prophylactic agent for any patient group.

CAUTION! Warfarin is effective but has a longlasting effect and needs monitoring.

It can, however, be reversed by vitamin K. The goal is an INR target of 2.5 for DVT prophylaxis. Start it the night before surgery and continue it postoperatively during the discharge period. You won't usually reach INR target levels until postoperative day 3.

HEPARIN OVERDOSAGE

This is reversible with protamine sulphate, which itself is an anticoagulant. 1mg can neutralize c.100U of heparin activity. You must infuse it very slowly IV over 10mins to a maximum of 50mg.

Time Elapsed Since Heparin Dose	Dose of protamine (mg) to neutralize 100 units of heparin
<1/2 h	1-1.5mg protamine per
< 1/2 II	1-1.5mg protamine per 100 units of heparin
30-120 min	0.5-0.75mg protamine per 100
	units of heparin
>2 h	0.25-0.375mg protamine per
	100 units of heparin

Because heparin is rapidly cleared from the circulation, the amount of protamine needed decreases rapidly. Titrate the final dosage according to coagulation studies, or by the scheme above.

44.8 **Protecting the kidneys**

The kidney is similar to the brain in that the secondary insult often causes more damage than the initial damage. Rapid correction of shock causing hypoperfusion is the single most important thing you can do to spare the kidneys a long-term insult.

If you don't resuscitate a patient in severe hypovolaemic shock rapidly and adequately (with fluid and blood), immediate death occurs, or the cortices of the kidneys necrose and the kidneys fail. Post-traumatic renal failure is thus the major complication of hypovolaemic shock.

Although a period of acute hypovolaemia can injure the lungs, the heart or the liver, it is the effect on the kidneys that is so marked and so preventable.

The more severe the hypovolaemia and the longer it lasts, the more likely are the kidneys to shed their tubular cells and stop functioning. Days or weeks may elapse before they recover.

During that time, a patient can die from uraemia, potassium intoxication, or infection. Prevent these disasters by treating hypovolaemic shock quickly and adequately. *A patient may require a lot of blood and fluid!*

Acute renal failure may also complicate extensive burns, crush injuries, severe muscle wounds (especially if they are heavily infected), causing rhabdomyolysis (49.9) or transfusion & drug reactions, causing haemolysis.

If a patient passes no urine, check first that the catheter is neither kinked nor blocked, and that the urinary tract is not obstructed. If the amount passed is <20mL/hr (for a child, <0.5–1mL/kg/h), suspect that there is post-traumatic renal failure.

Before diagnosing this, consider these other possibilities:

(1) There is still hypotension due to hypovolaemia. If the BP is still <80mm Hg systolic, or the peripheral circulation is still severely constricted, the glomerular filtration rate will be low. Once you correct the hypovolaemia and restore the blood pressure, the urine output may increase, provided that hypotension has not lasted long enough to damage the kidneys. (2) There is an expected metabolic response to injury owing to increased ADH secretion. This may reduce the urine output for 8-36h.

Don't rely on this being the right diagnosis unless the condition is stable in other respects, the urine is chemically normal and its specific gravity is high. The practical consequence of making this diagnosis is that you should not infuse more fluid to increase the urine output if all other signs are satisfactory.

If you have excluded these 2 conditions, and there is <20mL/h of urine over 12h, this is probably acute post-traumatic renal failure.

Diagnose it early, before the blood urea starts to rise, if: (1) the urine specific gravity is <1.016 in the absence of glycosuria or albuminuria, or (2) there is pigment or protein in the urine, whatever its specific gravity.

If the patient recovers, there are then 2 phases to pass through:

(a) An oliguric phase during which the kidneys cannot correct for the water and electrolyte intake, and so these need to be restricted. During this phase, a danger is that excess potassium will enter the plasma from dead or dying tissues. Try to minimize this.

Unfortunately, you cannot diagnose the earlier phases of hyperkalaemia clinically: you have to use laboratory tests frequently. Don't use potassiumcontaining solutions such as Darrow's or Ringer's lactate in this phase.

This phase may be followed gradually or suddenly by the next phase, diuresis.

(b) A **diuretic phase** during which the kidneys may pass 6-9L/day of urine, regardless of the fluid intake. While this phase lasts, the danger is of loss of electrolytes and dehydration.

Replace the electrolytes and the water lost. It is difficult to know when to stop adding large volumes of fluid input. If you keep adding fluid, the patient will go on excreting fluid, so you won't know if they are needed or not!

TREATMENT IN THE OLIGURIC PHASE

(a) Correct hypovolaemia & electrolyte deficit: chart what has been lost and what you have administered. Correct the calculated water and electrolyte deficit before you start the period of fluid restriction. (b) Administer the measured output of water, plus an estimate of the insensible loss. Give it as water by mouth, or as 5% dextrose IV.

N.B. Don't use any electrolyte solutions, except those necessary to replenish losses, because he cannot excrete any excess.

The measured output is the total volume of urine, vomit, or watery diarrhoea. The insensible loss in a temperate climate is c.500mL (6-7 mL/kg), and may rise in hot climates, or in fever to 1000mL (12 mL/kg) or more.

CAUTION!

(1) Don't include blood, plasma, or plasma substitutes in these estimates.

(2) *Don't allow thirst to influence the intake volume*. Watch that the patient does not overhydrate.

(3) The dose of many antibiotics, especially gentamicin and other aminoglycosides, needs to be modified in the presence of renal failure.

(4) Don't use diuretics.

(c) Minimize hyperkalaemia.

(1) Remove all dead and dying tissue with a really thorough wound toilet.

(2) Avoid hypoxia. If he needs an anaesthetic, use ketamine or LA.

(3) *Don't add potassium in any form.* There is potassium in milk and fruit juice, soup and meat, Darrows and Ringer's lactate, and in many drugs.

(4) Minimize catabolism by providing a high energy, no protein diet. If there is no nausea, gastric suction, nor intestinal pathology, include ≥400g/day of glucose or lactose, or, failing these, sucrose, by mouth or by nasogastric tube.

This will provide 1,600 kcal/day. Add 20mL of 50% glucose with 10U of soluble insulin into a large vein, preferably the vena cava (by a central line), qds.

(d) Weigh the patient daily. There should be c.500g weight loss daily after the initial fluid replacement. If there is weight gain, this will be retaining fluid from being overhydrated.

TREATMENT IN THE DIURETIC PHASE

In every 24h during this phase, add 1500mL of maintenance fluid on top of the volume of urine output over the previous 24h.

Infuse 1L 0.9% saline plus 1L 5% dextrose and the balance as ½-strength Darrow's solution. This contains 17mM of potassium. The normal potassium requirements are c.35mmol/day. A total of 6-10L/day of fluid may be needed. Check the potassium level in the laboratory, if possible.

If the urine specific gravity is still very low after 4days, you are probably over-infusing. Start cautiously to reduce the fluid intake.

CAUTION! Don't start protein feeding until ≥1500mL/day of urine is passed, and the blood urea is <25mmol (2.5g/l). Starting it too early increases the danger of uraemic complications.

44.9 Cardiac massage & defibrillation

ASYSTOLE

When the heart stops and there is no rhythm, this is a sign of death. *Asystole is not a dysrthymia. It is not treatable by an electric shock.* You must reverse the causative events quickly for any hope of success.

First of all, you and your team must continue CPR while you determine whether the asystole is real.

Connect the patient to a monitor (if not already wired up), then:

(1) check the leads to make sure they are connected,

(2) change the gain on the monitor to make the ECG lead look larger on the screen, and

(3) use the selector knob to change the leads from position II (where it usually is) to another lead.

Check possible causes that you might be able to reverse.

N.B. This might well be too late, but go through them with this mnemonic: 6H4T:.

- (1) Hypovolaemia: infuse 0.9% saline.
- (2) Hypoxia: administer oxygen and ventilate.
- (3) **Hypoglycaemia**: check a finger-prick test immediately, and administer 200mL 10% dextrose IV if <2mmol/L.
- (4) **Hydrogen ion excess** (acidosis): *don't administer bicarbonate!*
- (5) Hypothermia: cover with warm blankets, use a heating fan, and warm IV fluids. Don't move the patient if the temperature is <28°C.</p>
- (6) Hypo- or hyperkalaemia: you may well not be able to check this quickly, but slow infusion of 10mL 10% calcium gluconate is the best option for reversing hyperkalaemia.
- (7) **Tension pneumothorax** (43.2): insert a needle into the 2nd intercostal space in the mid-clavicular line.
- (8) **Tamponade** (cardiac, 44.7): perform a pericardiac tap (9-4), or open the chest!
- (9) Toxins: a hard diagnosis to make unless you have evidence of an empty bottle (of alcohol or medicines) or a labelled syringe. You can only reverse a few overdosages. If the cause is opiates, use naloxone; if benzodiazepines use flumanezil; if β-blockers use adrenaline,
- (10) **Thrombosis**, pulmonary or coronary.

The success of cardiopulmonary resuscitation (CPR) is relatively poor, even in tertiary hospitals, but it is zero if nothing is done.

Most cardiac arrests in LMICs are not from heart attacks, they occur because an acute respiratory, septic or other condition has deteriorated to the point of no return.

This is why prevention is so necessary by good attention to the critical ill and the course of their illness; prevention beats the best CPR.

This is especially true in children (who make up 50% of the population in many countries), because their strong compensatory physiology means they are very often severely metabolically deranged when they hearts stop.

Respiratory problems are more common than cardiac events in children; the reverse is true in adults. Therefore concentrate on the AB of ABC.

There are many cultural and educational issues which may interfere with the timely recognition of impending death, preventing intervention. It is important to teach your team who is at high risk of arrest, and of the need for prevention.

For effective CPR, you need a motivated, trained team. You should gather any necessary equipment ahead of time and keep it at a central location on a 'crash cart'. Ideally, each main unit of the hospital (including the emergency department and the operating theatre) should have its own cart.

Ensure that your team knows how your AED (automated external defibrillator) or independent defibrillator and monitor works.

For the latter, know how to switch from unsynchronized defibrillator mode to synchronized cardioversion mode. Also, know how to turn the pacer on and change both heart rate and amperage to that you can capture the runaway heart. The time to learn these things is NOT during the middle of a cardiorespiratory arrest.

Assign 2 persons to be responsible for checking each day (and after each usage) that the supplies are all present or replaced and all electrical items are working (including rechargeable and disposable batteries).

EQUIPMENT IN ALL CARTS

(1) Basic airway equipment including different sizes of self-inflating bag valves and masks, oral and nasal airways, oxygen masks, nasal cannulas, & Magill forceps.

(2) Intravenous access equipment including venous catheters, IV tubing and IV fluid.

If intraosseous access is available, include a drill motor and/or needles.

(3) An automatic external defibrillator: keep this charged! (This might be the least necessary equipment)

Have fresh disposable batteries if any equipment needs them, and a check list of all supplies.

ADVANCED LIFE SUPPORT EQUIPMENT

Add these items to the cart and checklist:

(1) Adrenaline, amiodarone and lidocaine.

(2) Adenosine, diltiazem, metoprolol and atropine.

(3) Methylprednisolone and diphenhydramine.

(4) Laryngoscopes with various sizes of straight and curved blades (adult and paediatric), various sizes of ET tubes and perhaps laryngeal mask airways, or supraglottic airways.

(5) Nitro-glycerine spray or 0.4mg tablets

(6) Naloxone

BASIC LIFE SUPPORT

You should have a system in place to recognize and activate a 'crash call'

Teach team members how to recognize that a person is unresponsive, not breathing or breathing ineffectually (gasping), and know how to call for help, ideally by pressing a 'crash button'

(a) Check the airway

(b) Check the breathing (40.1)

(c) Check the pulse for ≥5secs but ≤10secs at the carotid or femoral in adults; in infants, at the brachial in the mid-upper arm.

If the pulse is absent, start CPR compressions at cycles of 30 compressions and 2 breaths. Hook up the monitor and run the checks. If there is a 2^{nd} rescuer, increase the 30:2 ratio to 15:2 in pre-pubertal children.

If there is bradycardia <60 beats/min in a child, continue compressions at 15:2 as before.

(d) Effective compression means pushing down on the lower half of the sternum in adults; *don't deviate off the midline*.

In infants stay high enough to avoid pushing on the xiphoid. *Push hard and push fast.* Maintain a rate of at least 120 beats/min.

	Neonate <1 month	Infant <1yr	Child <7yrs	Adult
Compression detail				
Place	Centre	of	sternum	lower ⅓ of sternum
Depth	¹ ∕₃ of	chest	diameter	5-6cm
Rate	100-120	beats	per	minute
Method	1 finger	1-2 fingers	palm of 1 hand	palms of both hands

Your compressions should be \geq 5cm in adults and $\frac{1}{3}$ the antero-posterior diameter in children & infants (c. 5cm & 4cm respectively). Be sure to allow the chest to recoil fully between compressions (to allow venous return).

Change rescuers every 2mins: compressing the chest is tiring if done right! Minimize any interruptions to compression (never stop for ≥10secs!)

(e) Blow in 2 breaths with the airway clear.

Tilt the head with the chin lift or the jaw thrust (especially if neck trauma is suspected).

N.B. You may need to put a pad under the thorax to avoid hyperflexion of the airway in small children (with their outsized occiput).

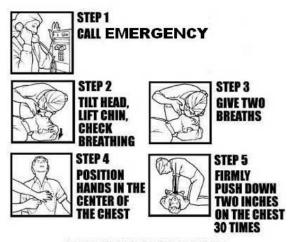
Use a one-way mask (this should be on your crash cart) to give the breaths. Each breath should be just enough to see the chest rise. Introduce the air in >1sec: *don't overinflate the chest*. Allow 1–1.5secs for exhalation before giving the 2^{nd} breath.

If the chest does not rise, return immediately to compression and prepare to suction the airway. Consider introducing an oropharyngeal airway or improve the position of the head before your next attempt to give 2 breaths.

Continue this cycle until help arrives. Stop to check pulses every five cycles (every 2mins). Stop if a spontaneous perfusing rhythm is present. **If you find none, continue CPR.**

N.B. Defibrillation is very rarely an option in the district hospital setting, and is not described here.

CARDIOPULMONARY RESUSCITATION (CPR)



CONTINUE WITH TWO BREATHS AND 30 PUMPS UNTIL HELP ARRIVES

Fig. 44-18 CARDIOPULMONARY RESUSCITATION. Follow this scheme if the victim is in public, after making sure you & he are in a safe place. *N.B. This applies to a person likely to have suffered a 'heart attack'*, the priority for a patient who is bleeding is to STOP THE BLEEDING!

ADVANCED LIFE SUPPORT

Pulseless electrical activity is the presence of cardiac arrest in the presence of an organized cardiac rhythm (of any type) which is *not* accompanied by a pulse. It can look like normal sinus rhythm or any one of a hundred variants. Treat it like asystole. Remember to look for reversible causes.

Only one drug is indicated: adrenaline. For adults, administer 1mg IV (or intra-osseous) every 4mins (*i.e.* between every other cycle of compressions). Use a 20mL saline flush after each dose and elevate the arm. For children, use 0.01mg/kg.

If you don't have venous access, squirt 2mg in 10mL saline down the endotracheal tube.

If you have no IV or intra-osseous access, you can spray 10 x the above dose of adrenaline down the tube.

If you see ventricular fibrillation or unstable ventricular tachycardia, only 3 drugs are indicated:

(1) Adrenaline, as above, or:

(2) Amiodarone 300mg bolus with a 2nd dose of 150mg if needed 4mins afterwards.

For children, use 5mg/kg and you can repeat this dose twice every 24h, or:

(3) Lidocaine 1-1.5mg/kg as a bolus, with a 2^{nd} dose in 5-10mins if refractory.

N.B. For children, use 1mg/kg loading dose and 20–50µg/kg/min infusion.

If you see a bradycardia <50 beats/min, determine whether there are signs of distress or hypoperfusion (mental confusion, ischaemic chest pain, hypotension, diminished capillary refill, heart failure, administer atropine 0.5mg IV as a bolus. Repeat this as necessary every 3–5mins up to a maximal dose of 3mg (6 doses).

In children, use 0.02mg/kg (minimum dose 0.1mg; maximum single dose 0.5mg). You can repeat this once. The maximum total dose for a child is 1mg; for an adolescent 3 mg (6 doses), as an adult.

Maintain the airway (assisting with bag valve mask if necessary, administering oxygen if hypoxic)

N.B. Don't start futile resuscitation if the pupils are already dilated and fixed.

WHEN SHOULD YOU STOP RESUSCITATION? There is no one right way or wrong way, but here is one possible conversation you might have with your team (with the family listening in or not). "Right, we are on the 5th cycle of CPR and good ventilation. We have run through the H's and T's and found nothing we could reverse. We have given 1mg of adrenaline at 4 & 8 mins. We have given amiodarone 300mg without effect. There is asystole, which we have checked. I think we have done all we can. You have all done a good job. Does anyone have any suggestions?"

After listening to comments, continue, "So, our next check is in 2mins. We know that almost no one lives or regains brain function after this long. If it shows asystole again, we will stop. I am sorry, but thank you very much for all your efforts."

This shows good communication, appreciation, compassion and a professional approach, to both the team and anyone else who is listening.

The only thing left to do is to document what transpired and to have a debriefing meeting later in the day.

COMMON PROBLEMS DURING CPR

(1) You can't achieve IV or intraosseous access. Remember some drugs can be absorbed from the lung *via* an endotracheal tube: lidocaine, adrenaline, atropine and naloxone.

(2) *Don't wait for others to start the CPR:* yell for help and start immediately!

(3) Well-done CPR requires practice. You must push deep enough, fast enough and not stop for >10secs once you start, no matter what.

(4) Most (>90%) who survive do so with completion of the 1st or 2nd cycles of compression; remember to look for reversible reasons within these 1st 4mins.

(5) Don't waste time trying to put in an endotracheal tube if you are ventilating well with a bag-valve mask

(6) Make sure the crash cart is regularly checked, drugs are replaced and batteries are charged or replaced.

(7) Make sure you know how to use your equipment: *the middle of a CPR is not the place to learn.*

(8) Assign roles during CPR who does what. Make sure orders are clear & repeated back to you, as the leader. *Don't forget to ask for ideas, but be polite if someone is doing something wrong.*

(9) Don't forget to keep the family informed.

(10) Do a debriefing after the event. Support those who have taken it hard. Figure out what was done well, and what not so well, so that, as a team, you can do better next time.

N.B. Limb Compartment Syndrome (49.8) *N.B.* Abdominal Compartment Syndrome (11.10)

