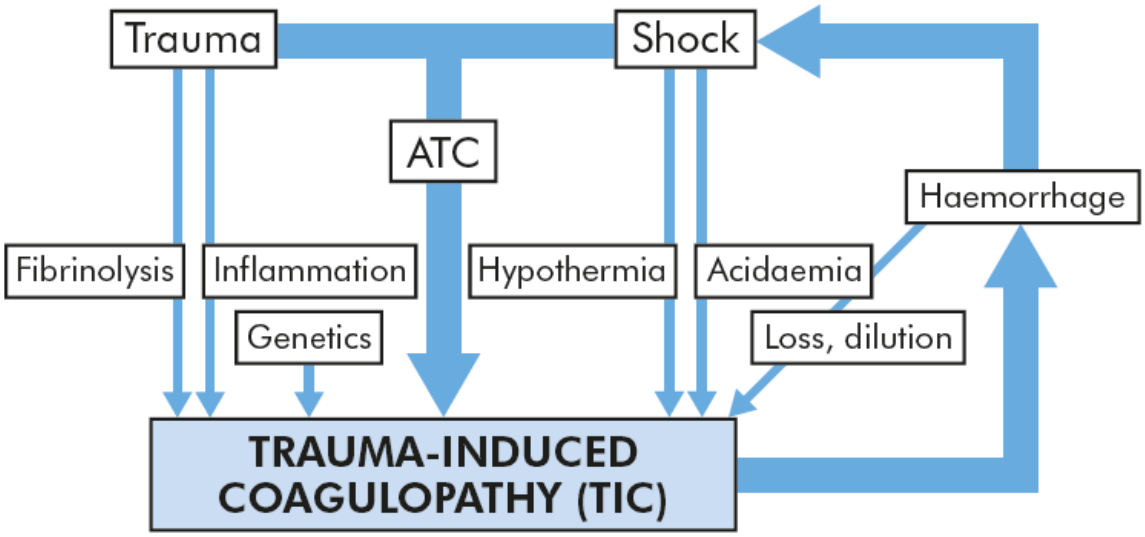
HAEMORRHAGE

Hemorrhage must be recognized and managed aggressively to reduce the severity and duration of shock and avoid death and/ or multiple organ failure.
Hemorrhage is treated by arresting the bleeding – not by fluid resuscitation or blood transfusion.
Although necessary as supportive measures to maintain organ perfusion, attempting to resuscitate patients who have ongoing hemorrhage will lead to physiological exhaustion (coagulopathy, acidosis and hypothermia) and subsequently death.

Pathophysiology

Hemorrhage leads to a state of hypovolemic shock. The combination of tissue trauma and hypovolemic shock leads to the development of an endogenous coagulopathy called acute traumatic coagulopathy (ATC). Up to 25 per cent of trauma



patients develop ATC within minutes of injury and it is associated with a four-fold increase in mortality. It is likely that ATC exists whenever there is the combination of shock and tissue trauma (e.g. major surgery).

Ongoing bleeding with fluid and red blood cell resuscitation leads to a dilution of coagulation factors which worsens the coagulopathy.
In addition, the acidosis induced by the hypo-perfused state leads to decreased function of the coagulation proteases, resulting in coagulopathy and further hemorrhage. The reduced tissue perfusion includes reduced blood supply to muscle beds.
Under-perfused muscle is unable to generate heat and hypothermia ensues. Coagulation functions poorly at low temperatures and there is further hemorrhage, further hypoperfusion and worsening acidosis and hypothermia. These three factors result in a downward spiral leading to physiological exhaustion and death.
Medical therapy has a tendency to worsen this effect. Intravenous blood and fluids are cold and exacerbate hypothermia.
Further heat is lost by opening body cavities during surgery. Surgery usually leads to further bleeding and many crystalloid fluids are themselves acidic (e.g. normal saline has a pH of 6.7).
Every effort must therefore be made to rapidly identify and stop hemorrhage, and to avoid (preferably) or limit physiological exhaustion from coagulopathy, acidosis and hypothermia

Definitions

Revealed and concealed hemorrhage

Hemorrhage may be revealed or concealed.
Revealed hemorrhage is obvious external hemorrhage, such as exsanguination from an open arterial wound or from massive hematemesis from a duodenal ulcer.
Concealed hemorrhage is contained within the body cavity and must be suspected, actively investigated and controlled.
In trauma, hemorrhage may be concealed within the chest, abdomen, pelvis, retroperitoneum or in the limbs with contained vascular injury or associated with long-bone fractures.
Examples of non-traumatic concealed hemorrhage include occult gastrointestinal bleeding or ruptured aortic aneurysm.

Primary, reactionary and secondary hemorrhage

Primary hemorrhage is hemorrhage occurring immediately due to an injury (or surgery).
Reactionary hemorrhage is delayed hemorrhage (within 24 hours) and is usually due to dislodgement of clot by resuscitation, normalization of blood pressure and vasodilatation.
Reactionary hemorrhage may also be due to technical failure, such as slippage of a ligature.

Secondary hemorrhage is due to sloughing of the wall of a vessel. It usually occurs 7–14 days after injury and is precipitated by factors such as infection, pressure necrosis (such as from a drain) or malignancy.

Surgical and non-surgical hemorrhage

Surgical hemorrhage is due to a direct injury and is amenable to surgical control (or other techniques such as angioembolization).

Non-surgical hemorrhage is the general ooze from all raw surfaces due to coagulopathy and cannot be stopped by surgical means (except packing).

Treatment requires correction of the coagulation abnormalities.

Degree and classification

The adult human has approximately 5 liters of blood (70 mL/ kg children and adults, 80 mL/kg neonates). Estimation of the amount of blood that has been lost is difficult, inaccurate and usually underestimates the actual value.

External hemorrhage is obvious, but it may be difficult to estimate the actual volume lost. In the operating room, blood collected in suction apparatus can be measured and swabs soaked in blood weighed.

The hemoglobin level is a poor indicator of the degree of hemorrhage as it represents a concentration and not an absolute amount. In the early stages of rapid hemorrhage, the hemoglobin concentration is unchanged (as whole blood is lost). Later, as fluid shifts from the intracellular and interstitial spaces into the vascular compartment, the hemoglobin and hematocrit levels will fall.

Treatment should therefore be based upon the degree of hypovolemic shock according to vital signs, preload assessment, base deficit and, most importantly, the dynamic response to fluid therapy. Patients who are ‘non-responders’ or ‘transient responders’ are still bleeding and must have the site of hemorrhage identified and controlled.

Table 2.4 Traditional classification of haemorrhagic shock.

	Class			
	1	2	3	4
Blood volume lost as percentage of total	<15%	15–30%	30–40%	>40%

Management

Identify hemorrhage

External hemorrhage may be obvious, but the diagnosis of concealed hemorrhage may be more difficult. Any shock should be assumed to be hypovolemic until proved otherwise, and similarly, hypovolemia should be assumed to be due to hemorrhage until this has been excluded.

Immediate resuscitative maneuvers

Direct pressure should be placed over the site of external hemorrhage. Airway and breathing should be assessed and controlled as necessary. Large-bore intravenous access should be instituted and blood drawn for cross-matching (see Cross-matching below). Emergency blood should be requested if the degree of shock and ongoing hemorrhage warrants this.

Identify the site of hemorrhage

Once hemorrhage has been considered, the site of hemorrhage must be rapidly identified. Note this is not to definitively identify the exact location, but rather to define the next step in hemorrhage control (operation, angio-embolization, endoscopic control).

Clues may be in the history (previous episodes, known aneurysm, non-steroidal therapy for gastrointestinal (GI) bleeding) or examination (nature of blood – fresh, melaena; abdominal tenderness, etc.). For shocked trauma patients, the external signs of injury may suggest internal hemorrhage, but hemorrhage into a body cavity (thorax, abdomen) must be excluded with rapid investigations (chest and pelvis x-ray, abdominal ultrasound or diagnostic peritoneal aspiration).

Investigations for blood loss must be appropriate to the patient's physiological condition. Rapid bedside tests are more appropriate for profound shock and severe hemorrhage than investigations such as computed tomography (CT) which take time.

Hemorrhage control

The bleeding, shocked patient must be moved rapidly to a place of hemorrhage control. This will usually be in the operating room but may be the angiography or endoscopy suites. These patients require surgical and anesthetic support and full monitoring and equipment must be available.

Hemorrhage control must be achieved rapidly so as to prevent the patient entering the triad of coagulopathy–acidosis– hypothermia and physiological exhaustion. There should be no unnecessary investigations or procedures prior to hemorrhage control to minimize the duration and severity of shock. This includes prolonged attempts to volume resuscitate the patient prior to surgery, which will result in further hypothermia and clotting factor dilution until the bleeding is stopped.

Attention should be paid to correction of coagulopathy with blood component therapy to aid surgical hemorrhage control.
Surgical intervention may need to be limited to the minimum necessary to stop bleeding and control sepsis. More definitive repairs can be delayed until the patient is hemodynamically stable and physiologically capable of sustaining the procedure. This concept of tailoring the operation to match the patient's physiology and staged procedures to prevent physiological exhaustion is called 'damage control surgery' – a term borrowed from the military which ensures continued functioning of a damaged ship above conducting complete repairs which would prevent rapid return to battle.
Once hemorrhage is controlled, patients should be aggressively resuscitated, warmed and coagulopathy corrected. Attention should be paid to fluid responsiveness and the end points of resuscitation to ensure that patients are fully resuscitated and to reduce the incidence and severity of organ failure.

Damage control surgery

_ Arrest hemorrhage
_ Control sepsis
_ Protect from further injury
_ Nothing else

Damage control resuscitation

These concepts have been combined into a new standard for the management of trauma patients with active hemorrhage called damage control resuscitation (DCR). The four central strategies of DCR are:

1 Anticipate and treat acute traumatic coagulopathy
2 Permissive hypotension until hemorrhage control
3 Limit crystalloid and colloid infusion to avoid dilutional coagulopathy
4 Damage control surgery to control hemorrhage and preserve physiology.

Damage control resuscitation strategies have been shown to reduce mortality and morbidity in patients with severe trauma and may be applicable in other forms of acute hemorrhage.