Hemorrhage

Introduction

- 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.
- Hemorrhage could be revealed or concealed:
  Revealed hemorrhage is obvious external hemorrhage, such as an open wound.
  Concealed hemorrhage is contained within the body cavity and must be suspected, investigated and controlled. In trauma, hemorrhage may be concealed within the chest, abdomen, pelvis or in the limbs.
- **Causes of hemorrhage**:
  - Trauma (e.g. penetrating wounds).
  - High pressure inside the blood vessel (e.g. hypertension).
  - Erosion of blood vessels (e.g. as in malignancy)

Types of hemorrhage

1. According to source of hemorrhage

✓ **Arterial hemorrhage**

- Bright red in color (oxygenated)
- Spurting as jet which rises and falls in time with pulse

✓ **Venous hemorrhage**

- Dark red in color
- Steady flow
- Copious with rapid blood loss when large veins are opened as femoral or internal jugular veins.
- Bleeding from pulmonary veins is bright red (oxygenated)

✓ **Capillary ooze**

2. According to time of 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 a clot by resuscitation.
  - Normalization of blood pressure and vasodilatation.
  - 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.

3. According to site of hemorrhage

- ✓ **External hemorrhage:** which is visible and revealed
- ✓ **Internal hemorrhage** : which is invisible and concealed, this type may become revealed in
  - Hematemesis (bloody vomiting) or malena (blood with stool) as in bleeding from the GIT.
  - Hematurea (blood in urine) as in bleeding from kidneys or bladder.
  - Vaginal bleeding from uterus in pregnancy as in threatened abortion.

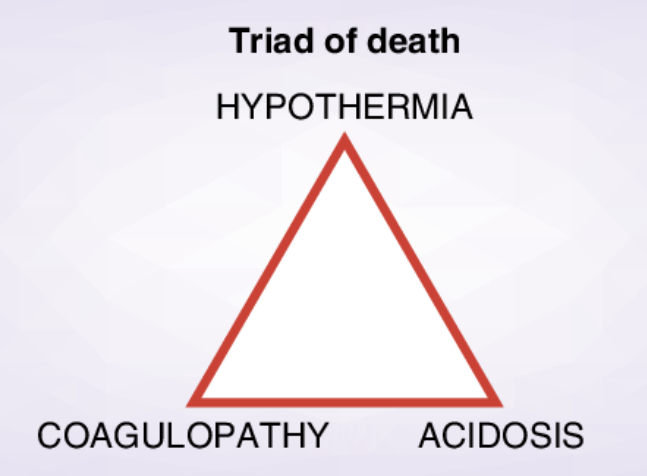
Pathophysiology

Hemorrhage leads to a state of hypovolemic shock. The combination of prolonged excessive bleeding and hypovolemic shock leads to the development of an endogenous **coagulopathy** called acute traumatic coagulopathy (ATC). Ongoing bleeding with fluid resuscitation leads to a dilution of coagulation factors which worsens the coagulopathy.

The **acidosis** induced by the hypoperfused state leads to decreased function of the coagulation resulting in coagulopathy and further hemorrhage. Many crystalloid fluids are themselves acidic (e.g. normal saline has a pH of 6.7).

Underperfused 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. 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.
These three factors result in a downward spiral leading to physiological exhaustion and death.



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.

Degree and classification

The amount of hemorrhage can be classified into classes 1–4 based on the estimated blood loss:

- **CL I: <15%**
- **CL II: 15–30%**
- **CL III: 30–40%**
- **CL IV: >40%**

The hemoglobin level is a poor indicator of the degree of hemorrhage because it represents a concentration and not an absolute amount. In the early stages of rapid haemorrhage, the haemoglobin 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, 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.

Management

Hemorrhage control must be achieved rapidly to prevent the patient entering the triad of coagulopathy–acidosis– hypothermia and physiological exhaustion.

❖ Identify hemorrhage

External hemorrhage may be obvious, but the diagnosis of concealed hemorrhage may be more difficult. Any shock should be assumed to be hypovolaemic until proven otherwise and, similarly, hypovolemia should be assumed to be due to hemorrhage until this has been excluded.
Diagnostic peritoneal lavage and FAST (Focused Assessment Sonography in Trauma) are useful procedures to reveal the concealed hemorrhage

❖ Immediate resuscitative manoeuvres

- Direct pressure should be placed over the site of external hemorrhage or perform ligation if necessary.
- Some procedures need to be performed in the operating room to arrest the bleeding (deep vessels ligation or the use of surgical diathermy for cauterization).
- Intravenous access should be instituted and blood drawn for cross-matching.

❖ Restore the blood volume

This is final step and done by blood transfusion and warm I.V. fluids to act as volume expanders.

This is the End of the Lecture – Good Luck