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FCA REFRESHER COURSE

Massive haemorrhage and transfusion

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Introduction

Definitions

Massive blood loss is defined as the loss of one blood volume within 24 hours. Normal blood volume is 70 ml/kg in adults (ideal body weight), 60 ml/kg in the elderly and 80–90 ml/kg in children. An alternative definition of massive blood loss is the loss of 50% of the blood volume within three hours or a rate of loss of greater than 150 ml per minute.[1]

In adults, several definitions of massive transfusion (MT) exist based on the volume of the blood products transfused and also the time frames over which these transfusions occur. The three most common definitions of MT in adult patients are (i) transfusion of ≥ 10 red blood cell (RBC) units, which approximates the total blood volume (TBV) of an average adult patient, within 24 hours, (ii) transfusion of > four RBC units in one hour with anticipation of continued need for blood product support, and (iii) replacement of > 50% of the TBV by blood products within three hours.[2]

Total blood volume estimation

Table I and II provide information on the TBV for adults and children.[2]

Table I: Total blood volume estimation in adults[2]

Patient	Fat	Thin	Normal	Muscular
Male	60	65	70	75
Female	55	60	65	70
TBV for adults based on Gilcher's rule of five for blood volume (in ml/kg body weight)				

Table II: Total blood volume estimation in children[2]

Patient	In ml/kg body weight
Neonate (0–4 kg)	85
Infant (5–9 kg)	85
Young child (10–24 kg)	75
Older child (25–49 kg)	70
Young adult (≥ 50 kg)	Use Gilcher's rule

Coagulation process

Haemostasis in massive haemorrhage involves four major processes. Primary haemostasis is the formation of a platelet plug involving adhesion, aggregation, as well as vasoconstriction. Secondary haemostasis is achieved through the activation of coagulation factors to generate thrombin. The third event is fibrin clot formation and stabilisation, and with the generation of a clot, the fourth process of thrombin inhibition and fibrinolysis is initiated.[3]

Haemostatic response to vascular injury consists of a series of interactions between the subendothelial matrix, platelets and coagulation proteins (Figure 1). Normally, the endothelial cells lining the inner wall of blood vessels prevent the subendothelial matrix and tissue factor from coming into direct contact with circulating platelets and coagulation proteins. Vascular injury disrupts the integrity of the endothelial lining, exposing the subendothelial matrix. The adherence of platelets to the subendothelial matrix leads to platelet activation and platelet plug formation. The platelet plug acts as a catalytic surface for the recruitment and activation of coagulation proteins, optimising the coagulation process.[4]

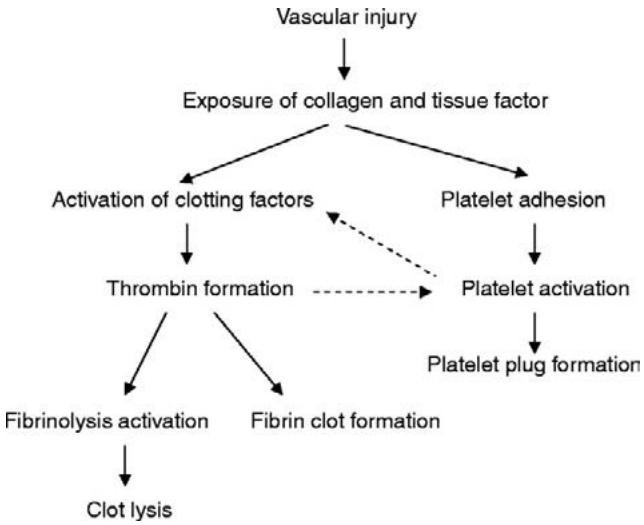

Figure 1: The haemostasis process[4]

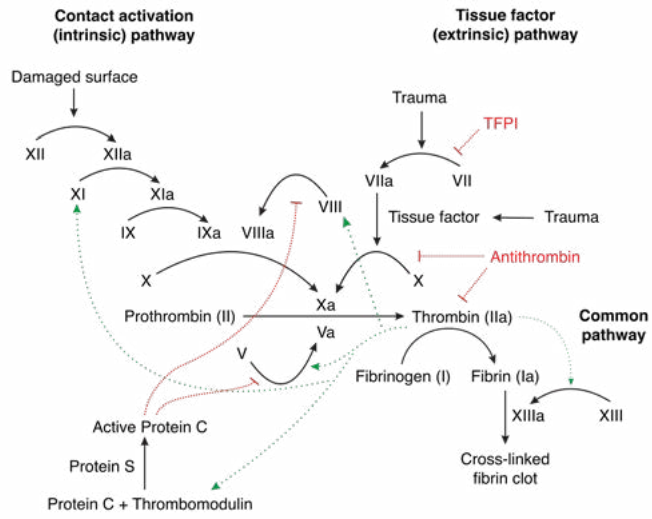

Figure 2: The coagulation process[5]

The coagulation process (Figure 2) is initiated by the binding of activated factor VII (which normally circulates in minute quantities) to the exposed tissue factor, which initiates coagulation by activating factors IX and X. Activated factor IX also activates factor X. Activated factor X, in turn, rapidly converts prothrombin to thrombin, generating small amounts of thrombin which are insufficient to convert fibrinogen to fibrin. The generation of thrombin is amplified by several feedback mechanisms. First, the generation of activated factor VII is increased by activation of factor VII bound to tissue factor by activated factors VII, IX and X.[4]

Second, the thrombin generated activates factors V and VIII, the cofactors which accelerate the activation of prothrombin and factor X, respectively. Thrombin increases the generation of activated factor IX by converting factor XI to an activated form. The generation of large amounts of activated factor X by activated factors IX and VIII ensures that sufficient amounts of thrombin are continuously generated to convert fibrinogen to fibrin, hence forming a clot. In the final step of coagulation, thrombin activates factor XIII to activated factor XIII, which then cross-links the soluble fibrin monomers to form a stable fibrin clot.[4]

In addition, thrombin activates the thrombin-activatable-fibrinolysis inhibitor which protects the clot from premature fibrinolysis. The haemostatic system is regulated by several anticoagulant proteins and inhibitors, as well as by the fibrinolytic process. When operating in balance, these interdependent processes ensure that the formed fibrin clot stops the bleeding, and subsequently, revascularisation occurs to maintain the blood flow.[4]

Effects of massive haemorrhage

Cardiovascular

The physiological response is an initial tachycardia with increased systemic vascular resistance (SVR) to maintain mean arterial pressure (MAP) despite a decreasing cardiac output. Once 20–30% blood volume is lost, there is loss of SVR resulting in a decreased MAP. Continued blood loss exceeding 40% leads to a pre-terminal phase of increased sympathetic drive with tachycardia and hypotension.[6]

Increased sympathetic tone diverts blood away from non-vital organs to maintain perfusion to vital organs, leading to hypoperfusion and inadequate oxygen delivery to the non-vital vascular beds and the microcirculation. Persistent, untreated hypoperfusion to the microcirculation will result in increased activation of the vascular endothelium, resulting in an exaggerated inflammatory response. The microcirculation 'unit' consists of an arteriole, a capillary bed and a venule and is particularly susceptible to hypoxic insults. The delivery of oxygen is dependent on blood flow and increases in pre-capillary vasomotor tone either by endogenous catecholamines or by prescribed vasopressors that will potentiate any hypoperfusion to the microcirculation and potentially worsen the inflammatory response.[6]

Haemorrhagic shock then results. The different grades of haemorrhagic shock are shown in Table III.

Early trauma-induced coagulopathy

The haemostatic defects in patients undergoing massive haemorrhage are dynamic and have multifactorial pathogenesis that relate to early trauma-induced coagulopathy (ETIC,

Table III: Grading of haemorrhagic shock[1]

	Grade of Shock			
Clinical variable	Class I	Class II	Class III	Class IV
Blood loss (ml)*	Up to 750	750–1 500	1 500–2 000	> 2 000
Blood loss (%)	Up to 15	15–30	30–40	> 40
Pulse rate (min^{-1})	< 100	100–120	120–140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (min^{-1})	14–20	20–30	30–40	> 35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Urine output (ml/hr)	> 30	20–30	5–20	Negligible
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

*For a 70 kg man

also termed acute coagulopathy of trauma), transfusion of blood products and infusion of crystalloids. Historically, ETIC was attributed to crystalloid and RBC transfusion without administration of platelets, plasma or both. However, subsequent studies in both adult and paediatric trauma patients demonstrated that ETIC was present in 24% and up to 56% of severely injured patients, usually within 30 min of injury, even before receiving RBC and fluid resuscitation.[2] The precise cause is difficult to identify and is likely to be multifactorial as demonstrated in Table IV.[4]

The presence of ETIC correlates with poor clinical outcomes independent of the severity of injury. ETIC is associated with systemic anticoagulation and hyperfibrinolysis. In brief, tissue injury from trauma or surgery releases tissue factor, locally and subsequently systematically, which activates coagulation pathways. This initiation results in massive consumptive coagulopathy leading to a consumptive disseminated intravascular coagulation-like syndrome. Furthermore, hypoperfusion from massive haemorrhaging leads to thrombomodulin expression on endothelial cells. Thrombin-thrombomodulin complex then activates protein C, which further limits coagulation by inhibiting activated factors V and VIII and enhancing fibrinolysis by depleting plasminogen activator inhibitor-1 (PAI-1) and accelerating plasmin formation. The diversion of thrombin from cleaving fibrinogen (for clot formation) to binding to thrombomodulin also reduces activation of thrombin-activatable fibrinolysis inhibitor (TAFI), which further leads to hyperfibrinolysis. The end result of these complex mechanisms is characterised by early coagulopathy due to systemic anticoagulation and hyperfibrinolysis.[2]

Anaemia itself contributes to further coagulopathy as erythrocytes are important providers of adenosine diphosphate, which assists in activating platelets. Erythrocytes also activate platelet cyclooxygenase and provide procoagulant phospholipids, which have a direct role in increasing thrombin generation. Furthermore, erythrocytes have a rheological effect by clustering in the centre of blood vessels. This results in an increased concentration of platelets at the vessel perimeter, where they can be exposed to endothelial trauma and thus activate and trigger coagulation. This process has a significant effect and has been shown to result in seven times the average concentration of platelets adjacent to the vessel walls. There is considerable evidence in animal, human volunteer and clinical models of a decrease in bleeding time (BT) in thrombocytopenia following a transfusion of RBCs.[3]

Hypothermia-induced coagulopathy

In trauma patients without pre-existing disease or massive head injury, the following conditions have been identified as significant risk factors for life-threatening coagulopathy: injury severity score > 25, systolic blood pressure < 70 mmHg, metabolic acidosis with pH < 7.10 and hypothermia with a body temperature < 34 °C. The interrelationship between hypothermia, metabolic acidosis and progressive coagulopathy is referred to as the 'lethal triad'; each factor exacerbates the others, leading to life-threatening bleeding or exsanguination (Figure 3). The causes of hypothermia are multifactorial and interdependent, including altered central thermoregulation, decreased heat production due to tissue hypoperfusion in haemorrhagic shock, exposure to low ambient temperature and infusion of inadequately warmed resuscitation fluids and blood components.[4]

Table IV: The major causes of coagulopathy in trauma patients[4]

- Blood loss
- Consumption of platelets and coagulation factor
- Increased fibrinolysis
- Dilution of coagulation factors and platelets
- Impaired functions of platelets and coagulation factors
- Hypothermia
- Coagulation-compromising effect of colloids
- Hypocalcaemia

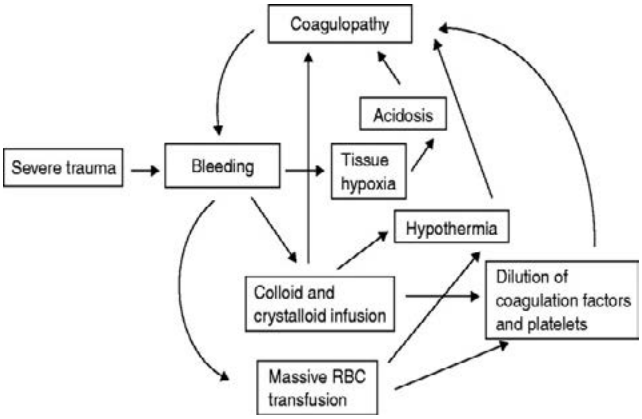

Figure 3: The interplay between metabolic acidosis, hypothermia and progressive coagulopathy in trauma[4]

The coagulation process consists of multiple enzymatic reactions, which are temperature-dependent and function optimally at 37 °C. The deleterious effect of hypothermia on coagulopathy in trauma patients has been well documented (Figures 4 & 5) and, when occurring in conjunction with metabolic acidosis, can result in a mortality rate as high as 90%. The effect of hypothermia on coagulopathy is difficult to identify by routine coagulation screening tests, such as prothrombin time (PT) and activated partial thromboplastin time (aPTT), because these tests are routinely carried out at 37 °C. Nevertheless, when PT and aPTT tests are carried out at low temperatures, as seen in hypothermic patients, both are significantly prolonged. In

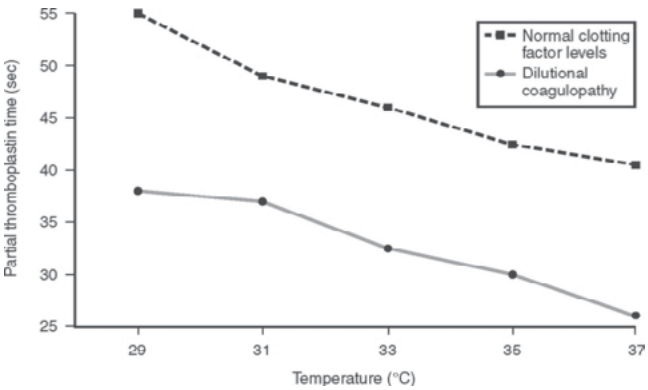

Figure 4: Hypothermia and activated partial thromboplastin time[7]

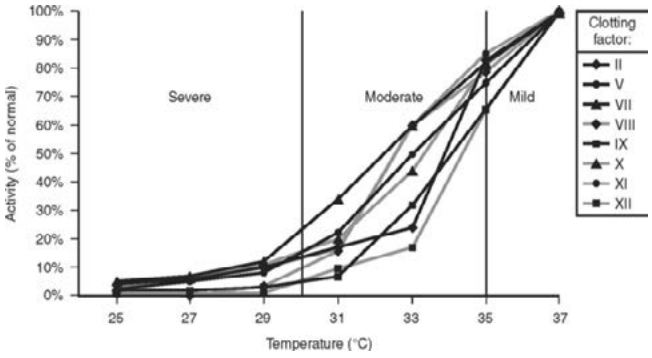

Figure 5: Hypothermia and clotting factors activity[8]

addition, both in vitro and in vivo studies have shown that hypothermia significantly impairs platelet function and the formation of a platelet plug and activates fibrinolysis.[4]

In summary, hypothermia impairs thrombin generation and the formation of platelet plugs and fibrin clots, and at the same time increases clot lysis, resulting in coagulopathy and uncontrollable bleeding.[4]

Inflammation

Tissue hypoperfusion induces an inflammatory response characteristic of ischaemia-reperfusion injury. A range of inflammatory mediators, cytokines, and oxidants are released, which may lead to secondary organ damage associated with multiple-organ failure (MOF) and death. The systemic inflammatory response syndrome (SIRS) invokes a simultaneous compensatory anti-inflammatory response (CARS), leading to a reprioritisation of cellular functions and suppression of adaptive immunity, the so-called 'genomic storm'. The extent and duration of the CARS and SIRS are related to the extent and duration of the initial inflammatory insult. Patients who recover their genomic expression in 2–3 days have uncomplicated recoveries, whereas those who do not have complicated recoveries. Rapid reversal of tissue hypoxia and restoration of blood flow to the microcirculation will lead to a reduction in SIRS, the associated CARS and potentially improved survival.[6]

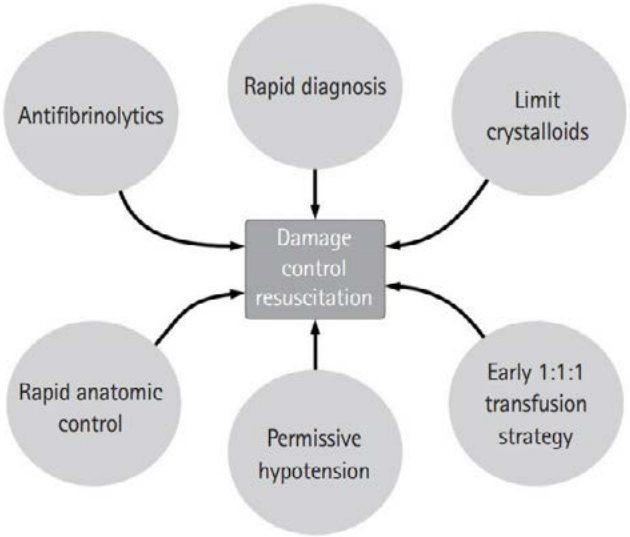

Figure 6: Tenets of damage control resuscitation[9]

Management of massive haemorrhage

Damage control resuscitation (DCR) emphasises novel resuscitation strategies that attempt to limit secondary blood loss and prevent the development of coagulopathy. These start at the time of patient presentation and continue through the entire phase of resuscitation. Use of this approach has been demonstrated to improve mortality, decrease healthcare costs and decrease length of stay. The tenets of DCR include hypotensive resuscitation techniques, early airway control, early use of blood and blood products and other haemostatic agents.[9]

Permissive hypotension

Hypotensive resuscitation is an integral part of DCR. Hypotensive resuscitation restricts the use of crystalloid fluids allowing the blood pressure to remain lower than normal, limiting secondary blood loss until initial haemostasis can be accomplished. While the optimal blood pressure target has yet to be defined, based on the currently available data, an SBP of 90–100 mmHg is likely safe for most patients. For patients with traumatic brain injury or spinal cord injuries, higher blood pressures are recommended and hypotensive resuscitation should not be used in these patients. According to the latest guidelines for the management of traumatic brain injury, an SBP >100 mmHg is recommended for patients aged 50–69, and an SBP > 110 mmHg is recommended for patients younger than 49 or older than 70 years.[9]

Rapid anatomical control

Damage control surgery minimises the amount of time and initial procedures done in the operating room to only critical interventions. This includes controlling major haemorrhage, containing contamination and applying temporary closure devices. The goal is to minimise time in the operating room, limit further bleeding and heat loss to allow time for the patient to be resuscitated in an intensive care unit to correct physiological and metabolic derangements before a planned return to the operating room.[9]

Limit crystalloids

The practice of crystalloid fluid resuscitation was supported by 1960s data which suggested that haemorrhage was associated with a loss of isotonic fluid in the extracellular space. This fluid deficit could be readily replaced with isotonic crystalloid. However, there is a growing body of literature highlighting the harms of crystalloid fluids in shock states, and, specifically, in major trauma. Crystalloid fluid promotes acidosis, dilutes coagulation factors, and disturbs inflammatory mediators.[9]

The harms of crystalloid resuscitation in trauma patients are well documented. A retrospective study by Ley et al. demonstrated that emergency department administration of greater than 1.5 L of crystalloid in trauma patients was associated with increased mortality.[10] In another retrospective study, Neal et al. demonstrated that a crystalloid (litre):packed RBC (unit) ratio of greater than 1.5:1 in patients who had received MT

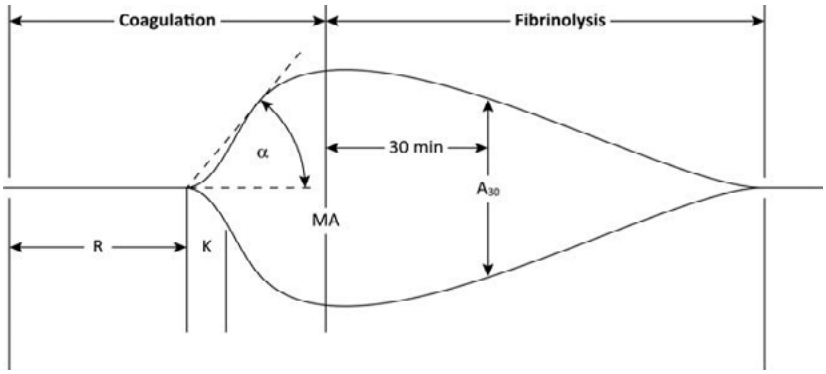

Figure 7: Thromboelastometry tracing[13]

was associated with a 70% higher risk of multiple organ failure (MOF) and a two times greater risk of acute respiratory distress syndrome as well as abdominal compartment syndrome.[11] More recently, the administration of prehospital plasma was studied in a large prospective multicentre cluster randomised trial which compared prehospital resuscitation with thawed plasma vs standard care. It demonstrated a significantly lower mortality at 30 days than the standard care group (23% vs 33%, $p = 0.03$).[9]

Use of antifibrinolytics

A large, randomised placebo-controlled trial of tranexamic acid in trauma patients with significant haemorrhage (CRASH-2) demonstrated significantly reduced all-cause mortality and death due to bleeding in the treatment arm. The authors concluded that consideration should therefore be given to routinely treating such patients with tranexamic acid (TXA), 10 mg/kg IV immediately and another dose over six hours, provided the first dose can be administered within the first three hours after injury.[12] The findings from the CRASH-2 trial were supported by the MATTERs (Military Application of Tranexamic Acid in Trauma Emergency Resuscitation) study – a retrospective observational study of soldiers resuscitated after combat injury in a surgical hospital in southern Afghanistan which compared TXA administration against control. There was a reduction in unadjusted mortality in the TXA group over the no TXA group (17.4% vs 23.3%, $p = 0.03$). The benefit from TXA was the greatest in patients who received MT (14.4% vs 28.1%, $p < 0.001$).[9]

Early massive transfusion strategies

In 2013, the PROMMTT study group performed a prospective, observational, multicentre major trauma transfusion study demonstrating that higher plasma:RBC and platelet:RBC ratios conferred survival benefits in the first six hours. Patients with ratios below 1:2 were three to four times more likely to die than patients with ratios greater than 1:1. In 2015, the PROPPR (pragmatic, randomised optimal platelet and plasma ratios) study group performed a large, multicentre, randomised control trial comparing ratios of plasma, platelets, and RBCs in a 1:1:1 versus 1:1:2 ratio. While there were no significant differences in mortality at 24 hours or 30 days, more patients in the 1:1:1 group

Table V: Thromboelastometry interpretation and management[2]

TEG parameter	Definition	Haemostatic phase	Aetiologies for abnormalities	Potential management
R	Time from the start of the test till the first sign of clot formation; it should take 4–8 minutes; it has an amplitude of 2 mm; it is dependent on clotting factors	Initiation of coagulation	Prolonged R: factor deficiencies or anticoagulants Shortened R: plasma hypercoagulability	Fresh frozen plasma (FFP) for prolonged R
K	Time from the start of clot formation to the time when the curve reaches amplitude of 20 mm; it should take 1–4 minutes; it is dependent on fibrinogen	Amplification of coagulation	Prolonged K: factor deficiencies, hypofibrinogenaemia, dysfibrinogenaemia, thrombocytopenia or platelet dysfunction.	Cryoprecipitate, FFP, platelets
α angle	Measures the speed at which fibrin build-up and cross-linking takes place, hence assessing the clot formation rate; it should be 47–74°; it is dependent on fibrinogen	Propagation of coagulation	Low α: factor deficiencies, hypofibrinogenaemia, dysfibrinogenaemia, thrombocytopenia or platelet dysfunction	Cryoprecipitate, FFP, platelets
MA	Represents the ultimate strength of the fibrin clot; it reaches an amplitude of 55–73 mm; it is dependent on platelets and fibrin	Propagation of coagulation	Low MA: factor XIII deficiency, hypofibrinogenaemia, dysfibrinogenaemia, thrombocytopenia or platelet dysfunction	Platelets, consider FFP or cryoprecipitate
A30/LY	Percentage decrease in amplitude at 30 minutes post MA; the decrease should be by 0–8%	Fibrinolysis	Increased LY: hyperfibrinolysis	Antifibrinolytic medication

achieved haemostasis and fewer died of exsanguination in the first 24 hours.[9]

Cryoprecipitate is a pooled human blood product which contains fibrinogen, factor VIII, factor XIII, vWF and fibronectin. It is included in many current MT protocols. Most patients in the PROMMTT trial did not receive cryoprecipitate, but in those who did, there was no in-hospital mortality benefit. However, in a retrospective review of the MATTERs II Study, cryoprecipitate was found to independently add to the survival benefit of tranexamic acid in major trauma.[9]

Besides the risk of transfusion reactions that occur with single-unit transfusions, patients with MT are at risk of other adverse events due to large transfusion volumes, such as hypocalcaemia and acidosis due to citrate and hypothermia due to cold storage. The patient should be monitored closely for these complications because they might contribute further to coagulopathy.[2] Since the administration of blood products comes with serious risks, it is imperative for the clinician to think about each blood product transfusion as soon as is reasonable during the resuscitation. The timing of this switch from protocol to targeted therapy depends on clinical circumstances, number of experienced personnel and information available to the clinician.[6]

Clotting tests should be performed to guide and monitor therapy, however, traditional laboratory tests take too long and their use in trauma has been challenged. Platelet counts are rapidly available as long as the laboratory systems in a particular hospital are set up to perform the analysis immediately. Point-of-care (POC) arterial blood gas monitoring is rapid and gives information about base deficit and haemoglobin, but no information on coagulation status. The use of thromboelastometry (TEG or ROTEM) has been increasing during surgery and trauma. It is more likely to give a dynamic interpretation of whole blood clotting and monitor clot initiation, strength, fibrinolysis and the relative contributions of functional fibrinogen and platelets.[6]

Cell salvage

Cell salvage represents a key strategy in reducing exposure to allogeneic blood transfusion and preserving donor blood. International guidelines are applicable to the South African context and recommend that cell salvage should be used when it will reduce the likelihood of blood transfusion or severe postoperative anaemia. It should ideally be available after-hours, necessitating appropriate staff training and availability of equipment. The use of cell salvage should be considered for high- or medium-risk surgery in non-obstetric adult patients where blood loss > 500 ml is likely and in major obstetric haemorrhage. Blood should be collected from the start of surgery, but the decision to process blood can be based on the volume collected to minimise the risk of opening expensive processing sets that are not used. In patients with malignancy or obstetric haemorrhage, a leucocyte filter must be used. Bacterial contamination of the surgical field remains a relative contraindication and should also be given through a leucocyte filter if transfused. Cell salvage may also be continued in the postoperative period.[14]

Table VI shows recommendations regarding the use of cell salvage. Each recommendation has been given a grade, using the following definitions set by the Australian National Health and Medical Research Council (NHMRC):

Grade A: Body of evidence can be trusted to guide practice.

Grade B: Body of evidence can be trusted to guide practice in most situations.

Grade C: Body of evidence provides some support for recommendation(s), but care should be taken in its application.

Grade D: Body of evidence is weak and recommendations must be applied with caution.[14]

Table VI: Use of cell salvage grading of recommendation[14]

	Consensus	Grade
The use of cell salvage should be considered for high- or medium-risk surgery in non-obstetric adult patients where blood loss of > 500 ml is likely	Yes	B
The use of cell salvage should be considered for obstetric major haemorrhage	Yes	B
In patients with malignancy or obstetric haemorrhage, a leucocyte filter must be used	Yes	C
Bacterial contamination of the surgical field remains a relative contraindication; use of a leucocyte filter is advised	Yes	C
Cell salvage may also be continued in the postoperative period	Yes	B

Complications of massive blood transfusion

Transfusion reactions

Allergic: Range from simple urticaria to anaphylaxis. Steroids and diphenhydramine might be given to patients with allergic transfusion.[2]

Haemolytic transfusion reaction: Acute and/or delayed. Might be reduced by giving group O RBCs and AB plasma for emergency release of blood products.[2]

Febrile non-haemolytic transfusion reaction: Diagnosis of exclusion.[2]

Immunological reactions

Transfusion-related acute lung injury (TRALI): This has been defined as the presence of hypoxaemia (PaO2:FiO2 ≤ 300 or oxygen saturation < 90% on room air), with bilateral infiltrates on chest radiograph and no evidence of circulatory overload.[15]

Transfusion-related immunomodulation (TRIM): Might be responsible for increased risk of bacterial infection.[2]

Transfusion-associated graft vs host disease (Ta-GVHD): Irradiation of cellular blood products in patients at risk (such

as neonates and immunosuppressed patients) can prevent Ta-GVHD.[2]

Post-transfusion purpura (PTP): Can be treated with IVIg infusion, steroids or plasma exchange.[2]

Metabolic complications

Hypocalcaemia: Due to citrate overload from rapid transfusion of blood products. Neonates and patients with pre-existing liver disease are at risk for hypocalcaemia. Monitor ionised calcium level and correct if necessary.[2]

Hypomagnesaemia: Due to a large volume of magnesium-poor fluid and citrate overload. Monitor ionised magnesium level and correct if necessary.[2]

Hyperkalaemia: Due to haemolysis of RBC from storage, irradiation or both. Neonates and patients with pre-existing cardiac and renal diseases are at risk for hyperkalaemia. Monitor potassium level and correct if necessary. Fresh RBCs (5–10 days old), irradiating < 24 h before transfusion or washing may decrease risk.[2]

Hypokalaemia: Due to re-entry into transfused RBCs, release of stress hormones or metabolic alkalosis. Monitor potassium level and correct if necessary.[2]

Metabolic alkalosis: Due to citrate overload. Monitor acid–base status.[2]

Acidosis: Due to hypoperfusion, liver dysfunction and citrate overload. Monitor acid–base status.[2]

Hypothermia: Due to infusion of cold fluid and blood products, opening of body cavities, decreased heat production and impaired thermal control. Neonates and infants are at increased risk. Measures to prevent further heat loss and rewarm the patient should be used.[2]

Other adverse events

Haemostatic defects: Result from complex mechanism (discussed in the pathophysiology section).[2]

Infection: Over the years, the risk of transfusion-transmitted infections caused by known pathogens, such as hepatitis B, hepatitis C and human immunodeficiency virus (HIV), has been significantly decreased. However, there remains a residual risk of infection caused by these pathogens. There is increasing concern over infection caused by emerging pathogens, such as the agent of variant Creutzfeldt–Jacob disease (vCJD), hepatitis G virus and West Nile virus.[4]

Transfusion-associated circulatory overload (TACO): This is more common than TRALI and is essentially cardiogenic pulmonary oedema.[15] Infants and patients with pre-existing cardiac disease are at increased risk. Oxygen and diuresis can be used.[2]

Air embolism: A rare but potentially fatal complication. Instructions and/or protocols on how to use rapid infuser must be followed.[2]

Conclusion

Table VII is a general summary by the South African Society of Anaesthesiologists on measures to be taken for patients expected to bleed significantly during surgery.

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Table VII: Patient blood management measures for patients expected to bleed significantly during surgery[14]

	Consensus	Grade
Preoperative		
Preoperative Hb should be measured, recorded and optimised as required	Yes	A
Elective surgery should be postponed in patients with untreated anaemia	Yes	A
Review and consider stopping antiplatelet and anticoagulant medication the recommended number of days before surgery	Yes	B
Consider a minimally invasive or laparoscopic surgical technique	Yes	B
POC testing should be available with appropriate training	Yes	C
Intraoperative		
Position patient carefully to optimise venous drainage	Yes	B
Use patient warming to maintain temperature > 36 °C	Yes	A
Consider cell salvage if blood loss > 500 ml anticipated	Yes	B
Consider giving TXA if blood loss > 500 ml anticipated	Yes	C
The dose of TXA should be 15 mg/kg	Yes	C
Apply restrictive transfusion threshold (Hb 7–8 g/dL) depending on patient characteristics and haemodynamics)	Yes	B
Consider use of topical haemostatic agents	Yes	C
Postoperative		
Maintain oxygen delivery, targeting oxygen saturation levels > 92%	Yes	A
Single unit blood transfusion policy, with subsequent re-assessment of Hb concentration and clinical need between RBC units	Yes	A
Consider postoperative drains for cell salvage	Yes	A

Hb – haemoglobin, POC – point-of-care, TXA – tranexamic acid, RBC – red blood cell

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