

Guidelines on the management of massive blood loss

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The guideline group was selected to be representative of UK-based medical experts and included the authors of previous recommendations. Preparation of the guidelines included a review of key literature, including Cochrane Database and MEDLINE and consultation with representatives of relevant specialties. Recommendations are based on appraisal of the relevant literature and expert consensus.

The writing group produced the draft guideline, which was subsequently revised by consensus by members of the Transfusion Task Force of the British Committee for Standards in Haematology. The guideline was then reviewed by a sounding board of approximately 100 UK haematologists, the British Committee for Standards in Haematology (BCSH) and the British Society for Haematology Committee and comments incorporated where appropriate. Criteria used to quote levels and grades of evidence are as outlined in appendix 3 of the Procedure for Guidelines Commissioned by the BCSH (<http://www.bcsghguidelines.com/process1.asp#App3>).

The objective of this guideline is to provide healthcare professionals with clear guidance on the management of massive blood loss. They do not address the specific problems associated with major obstetric haemorrhage; these are being addressed by the Royal College of Obstetricians.

In all cases individual patient circumstances may dictate an alternative approach.

Guideline update

These guidelines summarise current opinions regarding the management of massive blood loss and update previous recommendations (Stainsby *et al*, 2000).

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Summary of key recommendations

These are presented as a template that can be modified to suit local circumstances, and then displayed in clinical areas. The left-hand column outlines the key steps or goals, the centre column adds procedural detail and the right-hand column provides additional advice and information (Table I).

1 Background

Major blood loss jeopardises the survival of patients in many clinical settings and is a challenge for haematological and blood transfusion services. Tensions may arise between those attempting to treat bleeding, those supplying blood and those providing laboratory services. Discord can waste scarce resources, or, worse, result in a bad outcome for the patient.

1.1 Definitions

Massive blood loss is arbitrarily defined as the loss of one blood volume within a 24 h period (Mollison *et al*, 1997), the normal adult blood volume being approximately 7% of ideal body weight in adults and 8–9% in children. Alternative definitions that may be more helpful in the acute situation include a 50% blood volume loss within 3 h or a rate of loss of 150 ml/min (Fakhry & Sheldon, 1994).

It is imperative to recognise major blood loss early and institute effective action promptly if shock and its consequences are to be prevented.

1.2 Therapeutic goals

These are;

- Maintenance of tissue perfusion and oxygenation by restoration of blood volume and haemoglobin
- Arrest of bleeding by
- treating any traumatic, surgical or obstetric source
- judicious use of blood component therapy to correct coagulopathy

A successful outcome requires prompt action and good communication between clinical specialties, diagnostic labor-

Table I. Summary of key recommendations

Goal	Procedure	Comments
Restore circulating volume	Insert wide bore peripheral or central cannulae Give pre-warmed crystalloid or colloid as needed Avoid hypotension or urine output <0.5 ml/kg/h	14 gauge Monitor central venous pressure Keep patient warm Concealed blood loss is often underestimated
Contact key personnel	Clinician in charge Consultant anaesthetist Blood transfusion Biomedical Scientist Haematologist	A named senior person must take responsibility for communication and documentation. Arrange Intensive Care Unit bed
Arrest bleeding	Early surgical or obstetric intervention Interventional radiology	
Request laboratory investigations	FBC, PT, APTT, Thrombin time, Fibrinogen (Clauss method); blood bank sample, biochemical profile, blood gases and pulse oximetry Ensure correct sample identification Repeat tests after blood component infusion	Results may be affected by colloid infusion Ensure correct patient identification May need to give components before results available
Maintain Hb >8 g/dl	Assess degree of urgency Employ blood salvage to minimise allogeneic blood use Give red cells Group O Rh D negative In extreme emergency Until ABO and Rh D groups known ABO group specific when blood group known Fully compatible blood Time permitting Use blood warmer and/or rapid infusion device if flow rate >50 ml/kg/h in adult	Collection of spilt blood can be set up in <10 min D positive is acceptable if patient is male or postmenopausal female Further serological crossmatch not required after 1 blood volume replacement Transfusion laboratory will complete crossmatch after issue
Maintain platelet count >75 × 10 ⁹ /l	Allow for delivery time from blood centre Anticipate platelet count <50 × 10 ⁹ /l. after 2 × blood volume replacement	Allows margin of safety to ensure platelet count >50 × 10 ⁹ /l Keep platelet count >100 × 10 ⁹ /l if multiple or CNS trauma or if platelet function abnormal
Maintain PT & APTT < 1.5 × mean control	Give FFP 12–15 ml/kg (1 l or four units for an adult) guided by tests Anticipate need for FFP after 1–1.5 × blood volume replacement Allow for 30 min thawing time	PT/APTT >1.5 × mean normal value correlates with increased microvascular bleeding Keep ionised Ca ²⁺ > 1.13 mmol/l
Maintain Fibrinogen > 1.0 g/l	If not corrected by FFP give cryoprecipitate (Two packs of pooled cryoprecipitate for an adult) Should be available on-site. Allow for 30 min thawing time	Cryoprecipitate rarely needed except in DIC
Avoid DIC	Treat underlying cause (shock, hypothermia, acidosis)	Although rare, mortality is high

FBC, full blood count; PT, prothrombin time; APTT, activated partial thromboplastin time; FFP, fresh frozen plasma; DIC, disseminated intravascular coagulation.

atories, hospital transfusion laboratory staff and the Blood Service. Blood component support takes time to organise and the supplying blood centre may be several hours away from the

hospital. Special transfusion requirements for specific indications, e.g. components suitable for neonates, irradiated components for patients at risk of transfusion-associated

graft-versus-host disease, (British Committee for Standards in Haematology Blood Transfusion Taskforce, 1996, 2004), should be taken into account if time permits, but it may be necessary to make a pragmatic decision regarding the relative risks of delaying transfusion or giving components that are not of the appropriate specification.

1.3 Communication

Early consultation with senior surgical, anaesthetic and haematology colleagues is essential and the importance of good communication and co-operation in this situation cannot be over-emphasised. Appropriate surgical expertise for the area of bleeding is vital; involvement of vascular surgeons, cardiothoracic surgeons or others with specific interests is crucial to success. Consideration should be given to early referral and if necessary transfer to such expertise. In the more difficult cases, confidence to pack visceral cavities, cross clamp and tie off major vessels may be required. Radiological embolisation or stenting has an established role.

An intensive care bed is likely to be required and early warning of this is advisable. A member of the clinical team should be nominated to act as co-ordinator responsible for overall organisation, liaison, communication and documentation. This is a critical role for a designated member of the permanent clinical staff. Accurate documentation of blood components given and the reason for transfusion is necessary in order to enable audit of outcome and satisfy the legal requirement for full traceability (Blood Safety and Quality Regulations, 2005).

The hospital transfusion laboratory must be informed of a massive transfusion situation at the earliest possible opportunity. This will provide an opportunity to check stock, reschedule non-urgent work and call in additional staff if required out of hours.

Clear local protocols for management of massive blood loss should be accessible in all relevant clinical and laboratory areas and understood by all involved staff. Regular 'drills' can improve awareness and confidence and ensure that the blood transfusion chain works efficiently. It is good practice to review such cases after the event, to assess what went well and what did not, and to update protocols accordingly.

1.4 Role of the Hospital Transfusion Committee (HTC)

The Hospital Transfusion Committee (HTC) has a central role in ensuring the optimum and safe use of blood components. The development of protocols for management of massive transfusion is an important part of its remit. The HTC also provides a forum in which a rapid communication cascade can be agreed and massive transfusion episodes critically reviewed. As part of contingency planning for national blood shortage situations, every hospital should have an Emergency Blood Management Plan in place that provides guidance on clinical priorities for the use of large volumes of blood components. In

exceptional circumstances it may be necessary to make a decision to cease energetic transfusion support when the chances of the patient's survival are low, determined on a case-by-case basis. The HTC should establish a mechanism for making such difficult decisions on an individual basis, taking into account such factors as co-morbidity, potential for control of bleeding, reversal of the underlying cause and competing demands for available blood components.

2 Volume resuscitation

The over-riding first requirement is maintenance of tissue perfusion and oxygenation, which is critical in preventing the development of hypovolaemic shock and consequent high mortality from multi-organ failure.

Restoration of circulating volume is initially achieved by rapid infusion of crystalloid or colloid through large bore (up to 14 gauge) peripheral cannulae (Donaldson *et al*, 1992). Alternatively, larger bore central access devices can be used dependent on local skills and availability. The use of albumin and non-albumin colloids *versus* crystalloids for volume replacement has been the subject of debate following controversial meta-analyses (Cochrane Injuries Group Albumin Reviewers, 1998; Schierhout & Roberts, 1998). A controlled trial of normal saline *versus* 4% human albumin for fluid resuscitation involving over 7000 patients in 16 intensive Care Units in Australia and New Zealand (Finfer *et al*, 2004) found no difference in outcome at 28 d and concluded that they were clinically equivalent (*Grade A recommendation, Level 1b evidence*).

Hypothermia increases the risk of end organ failure and coagulopathy (Iseron & Huestis, 1991; American College of Surgeons, 1997) and may be prevented by pre-warming of resuscitation fluids, patient warming devices such as warm air blankets and the use of temperature controlled blood warmers (*Grade C recommendation, Level IV evidence*).

3. Investigations

Blood samples should be sent to the laboratory at the earliest possible opportunity for blood grouping, antibody screening and compatibility testing, as well as for baseline haematology, coagulation screen (including fibrinogen estimation and thrombin time) and biochemistry investigations.

Accurate patient identification is of paramount importance in all aspects of healthcare, and particularly in emergency situations. Every patient must wear an identification wristband, and there must be a robust and tested system in place for identification of unknown unconscious patients, including subsequent merging of clinical and laboratory records.

When dealing with an evolving process it is important to check parameters frequently, (and after each therapeutic intervention) to monitor the need for and the efficacy of component therapy. Appropriate use of near-patient testing

devices, which can include thromboelastography (TEG), can offer rapid data to guide component therapy (Samama & Ozier, 2003) but requires expert interpretation.

Advice should be sought from a consultant with transfusion expertise regarding appropriate investigations, their interpretation and optimum corrective therapy.

4 Blood component therapy

Allogeneic blood from volunteer donors is a limited and valuable resource that must be used carefully, appropriately and safely. There is a lack of good evidence from randomised controlled trials to support recommendations for the use of blood components in massive transfusions, although the design of such trials in the emergency setting is problematic. The target platelet count and the efficacy of fresh frozen plasma are key areas where further research is needed.

All blood components supplied by the UK transfusion services are now leucodepleted as a precaution against transfusion transmission of variant Creutzfeldt-Jakob disease (vCJD), in addition to mandatory testing for viral markers (human immunodeficiency virus [HIV], hepatitis B virus [HBV], hepatitis C virus [HCV] and Human T lymphotropic virus type 1 [HTLV 1]).

Benefits of leucodepletion also include reduced non-haemolytic febrile transfusion reactions, reduced transmission of leucocyte-associated viruses, such as cytomegalovirus, reduced immunosuppressive effects of transfusion (Blajchman *et al*, 2001) and reduced cytokine-mediated organ damage. (Erber, 2002) Transfusion giving sets, which should be changed at least 12-hourly during red cell infusion and prior to platelet infusion, include a screen filter. Any additional filter is unnecessary and may impede blood flow. (McClelland, 2001)

4.1 Red cells

The function of red cells is oxygen delivery to tissues; they should not be used as a volume expander. In the UK, the blood services will routinely provide leucodepleted red cells in optimal additive solution, which has low viscosity (Hogman *et al*, 1983) and contains minimal plasma.

Red cells also contribute to haemostasis by their effect on platelet margination and function. The optimal haematocrit to prevent coagulopathy is unknown, but experimental evidence suggests that a relatively high haematocrit, possibly 0.35 l/l, may be required to sustain haemostasis in patients with massive blood loss (Reiss, 2000; Hardy *et al*, 2004).

Red cell transfusion is likely to be required when 30–40% blood volume is lost; over 40% blood volume loss is immediately life-threatening (American College of Surgeons, 1997). Blood loss may be underestimated particularly if concealed and in young fit people, such as in the obstetric setting. Blood replacement should be guided by clinical estimation of blood

loss in conjunction with the patient's response to volume replacement.

Haemoglobin and haematocrit levels should be measured frequently, but in the knowledge that the haemoglobin level is a poor indicator of blood loss in the acute situation. Red cells are rarely indicated when the haemoglobin concentration is >10 g/dl but almost always indicated when it is <6 g/dl. (British Committee for Standards in Haematology Blood Transfusion Task Force, 2001) (*Grade C recommendation, Level IV evidence*). Decisions on red cell transfusion at intermediate haemoglobin concentrations should be based on the patient's risk factors for complications of inadequate oxygenation, such as rate of blood loss, cardiorespiratory reserve, oxygen consumption and atherosclerotic disease. Measured physiological variables, such as heart rate, arterial pressure, pulmonary capillary wedge pressure and cardiac output may assist the decision-making process, but it should be emphasised that silent tissue or organ ischaemia may occur in the presence of stable vital signs.

The clinician who communicates with the transfusion laboratory should indicate the timescale within which blood is needed at the bedside, (i.e. immediately, within 20 min, within an hour) in order that the laboratory scientist knows how much time is available for ABO and D grouping and pre-transfusion testing. In an extreme situation where blood is required immediately and the patient's blood group is unknown, it may be necessary to issue Group O uncrossmatched red cells. Females of reproductive age (i.e. under 50 years) whose blood group is unknown must be given group O Rh D negative red cells in order to avoid sensitisation and the risk of haemolytic disease of the newborn in subsequent pregnancy. It is acceptable to give O Rh D positive cells to males and older females of unknown blood group (Schwab *et al*, 1986), as group O Rh D negative blood is a scarce resource.

ABO group-specific red cells should be given at the earliest possible opportunity. Blood group determination takes less than 10 min and so it should not be necessary to give large volumes of group O blood.

In a patient with known red cell antibodies, the risk of a haemolytic transfusion reaction will need to be assessed against the risk of withholding transfusion until compatible blood can be provided.

Red cells undergo changes during 4°C storage (the 'storage lesion') including increase of extracellular potassium, decrease in ATP and 2,3-diphosphoglycerate (2,3 DPG) content and lowering of pH. Although 2,3 DPG is undetectable after 14–21 d storage, regeneration has been demonstrated within 24 h of transfusion (Heaton *et al*, 1989) and studies have not shown any clinically significant impact on oxygen diffusion when older components are transfused (Ho *et al*, 2003).

Intraoperative blood salvage may be of great value in reducing requirements for allogeneic blood and can be

rapidly deployed in hospitals where it is in routine use. (Hughes *et al*, 2001).

Synthetic oxygen-carrying solutions (artificial blood) are theoretically an attractive alternative to allogeneic blood but there is no product currently available in the UK (Prowse, 1999; Hess, 2004).

4.2 Platelets

Expert consensus advises that the platelet count should not be allowed to fall below the critical level of $50 \times 10^9/l$ in the acutely bleeding patient (Contreras, 1998) (*Grade C recommendation, level IV evidence*), and this is endorsed by the BCSH guidelines for the use of platelet transfusions. (British Committee for Standards in Haematology Blood Transfusion Task Force, 2003) A platelet count of $50 \times 10^9/l$ may be anticipated when approximately two blood volumes have been replaced by fluid or red cell components (Hiippala *et al*, 1995) but there is marked individual variation. A platelet transfusion trigger of $75 \times 10^9/l$ in a patient with ongoing bleeding is therefore recommended here, so as to provide a margin of safety to ensure that the level does not fall below that critical for haemostasis. A higher target level of 100×10^9 platelets/l has been recommended for those with multiple high-velocity trauma or central nervous system injury. (Development Task Force of the College of American Pathologists, 1994; Horsey, 1997) (*Grade C recommendation, level IV evidence*). Empirical platelet transfusion may be required when platelet function is abnormal, as occurs after cardio-pulmonary by-pass, in patients with renal dysfunction or secondary to anti-platelet therapy.

In assessing the requirement for platelets, frequent measurements are necessary, and it may be necessary to request platelets from the blood centre at levels above the desired target in order to ensure their availability when needed. Platelets can be given via an unused blood giving set, although a platelet giving set reduces wastage because it has less dead space. Transfusion of platelets through a giving set previously used for red cells is not recommended.

4.3 Fresh frozen plasma (FFP) and cryoprecipitate

Coagulation factor deficiency is the primary cause of coagulopathy in massive transfusion because of dilution of coagulation factors following volume replacement with crystalloid or colloid and transfusion of red cell components. The level of fibrinogen falls first; the critical level of 1.0 g/l is likely to be reached after 150% blood volume loss, followed by the fall of other labile coagulation factors to 25% activity after 200% blood loss. (Hiippala, 1998; Reiss, 2000). Prolongation of the activated partial thromboplastin time (APTT) and prothrombin time (PT) to 1.5 times the mean normal value is correlated with an increased risk of clinical coagulopathy (Ciavarella *et al*, 1987).

It is essential that laboratory tests of coagulation are monitored frequently; these may need interpretation by a

haematologist. Fibrinogen should be estimated using the Clauss method, and not derived from the prothrombin time, as this is unreliable. Laboratories should have standard operating procedures in place to ensure that clinical staff are contacted appropriately. Experienced laboratory scientists should be empowered to issue blood components in the first instance using a locally agreed algorithm. It may be necessary to request components before results are available, depending on the rate of bleeding and the laboratory turnaround time. Although 'formula replacement' with fresh plasma is not recommended, it may be required in situations where rapid turnaround of coagulation tests cannot be guaranteed. Infusion of FFP should be considered after one blood volume is lost (Hiippala, 1998). The dose should be large enough to maintain coagulation factors well above the critical level, bearing in mind that the efficacy may be reduced because of rapid consumption (Development Task Force of the College of American Pathologists, 1994; Hiippala, 1998) (*Grade C recommendation, level IV evidence*).

It should be borne in mind that, although FFP is recommended (British Committee for Standards in Haematology Blood Transfusion Taskforce, 2004) and widely used in situations of massive blood loss, there is little evidence of its clinical efficacy from randomised trials. (Stanworth *et al*, 2004)

Fresh frozen plasma alone, if given in sufficient quantity, will correct fibrinogen and most coagulation factor deficiencies, but large volumes may be required. If fibrinogen levels remain critically low (<1.0g/l), cryoprecipitate therapy should be considered. (Development Task Force of the College of American Pathologists, 1994; Hiippala, 1998) (*Grade C recommendation, level IV evidence*). Standard FFP contains 2–5 mg fibrinogen per ml, National Blood Service Quality Monitoring data indicates that pooled cryoprecipitate contains approximately 1.8 g per pool (range 1.6–2.0), though the minimum specification is 0.7 g. Hence 1 l of FFP might be expected to provide 2–5 g fibrinogen, whilst an adult therapeutic dose (two pools) of cryoprecipitate provides 3.2–4 g fibrinogen in a volume of 150–200 ml. Cryoprecipitate also contains factor VIII, factor XIII and von Willebrand factor. It should be remembered that transfusion of cryoprecipitate exposes the patient to multiple donors. Virus-inactivated fibrinogen concentrate is used in some European countries but is not licensed in the UK.

Fresh frozen plasma, once thawed, may be stored at 4°C for up to 24 h (British Committee for Standards in Haematology Blood Transfusion Taskforce, 2004). It is therefore advisable for the laboratory to thaw a therapeutic dose of FFP as soon as they become aware of a massive transfusion situation, in order to minimise delay.

British Committee for Standards in Haematology Guidelines on Oral Anticoagulation recommend prothrombin complex concentrate as an alternative to FFP when major bleeding complicates anticoagulant overdose (British Committee for Standards in Haematology, 1998).

5 Use of pharmacological agents to reduce bleeding

5.1 Antifibrinolytic drugs

Antifibrinolytic drugs, such as tranexamic acid and aprotinin, have been used to reverse established fibrinolysis in the setting of massive blood transfusion. (Koh & Hunt, 2003) Systematic reviews concluded that there is insufficient evidence from randomised controlled trials of antifibrinolytic agents in trauma to either support or refute a clinically important treatment effect (Henry *et al*, 2001; Coats *et al*, 2004) and recent evidence is conflicting (Sedrakyan *et al*, 2006).

5.2 Recombinant factor VIIa (rVIIa)

This drug is licensed for use in haemophiliacs with inhibitors to treat active bleeding or as prophylaxis for surgery.

Its use has been described off license as a 'universal haemostatic agent' in various settings of massive blood transfusion and there are many encouraging anecdotal case reports of its successful use. The common theme of these reports is a sudden reduction in blood loss following administration of the drug, with subsequent patient survival from exceptionally high-risk situations. The drug is expensive, but may prove cost effective through transfusion reduction in this setting. The use of rVIIa as 'last ditch' treatment for patients with massive haemorrhage has been shown to be ineffective (Clark *et al*, 2004).

A recent systematic review concluded that the application of rVIIa in patients with severe bleeding is promising and relatively safe (1–2% incidence of thrombotic complications). Sound evidence from controlled trials is not available so far; forthcoming trials are likely to provide more substantiation for its use (Levi *et al*, 2005).

Until such evidence becomes available it is reasonable to consider the use of rVIIa in situations where there is blood loss of >300 ml/h, with no evidence of heparin or warfarin effect, where surgical control of bleeding is not possible and there has been adequate replacement of coagulation factors with FFP, cryoprecipitate and platelets and correction of acidosis. There should be a local protocol in place and the decision should be made at consultant level.

6 Disseminated intravascular coagulation (dic)

Disseminated intravascular coagulation is a feared complication in the acutely bleeding patient but is fortunately rare outside obstetric practice. The cardinal clinical sign of DIC is microvascular oozing, whilst microthrombi in small vessels can result in end-organ damage. A DIC-like syndrome can result from the activation of the coagulation cascade secondary to tissue trauma, resulting in excessive consumption of platelets and coagulation factors. Patients at particular risk are those with tissue damage due to prolonged hypoxia, hypovolaemia

or hypothermia, and those with massive head injury or extensive muscle damage (Hardy *et al*, 2004). This syndrome carries a high mortality, and is difficult to reverse. Prolongation of the PT and APTT in excess of that expected by dilution, together with significant thrombocytopenia and fibrinogen of <1.0 g/l are highly suggestive of a developing DIC-like state and hence frequent estimation of platelet count, fibrinogen (using Clauss method), PT and APTT is strongly recommended. Measurement of D-dimer may also be useful in providing an early warning. Laboratory evidence of a consumption coagulopathy should be sought before microvascular bleeding becomes evident, so that appropriate and aggressive action can be taken to address the underlying cause.

Treatment of the coagulation defect consists of platelets, FFP and cryoprecipitate given 'sooner rather than later' in sufficient dosage, but avoiding circulatory overload.

7 Risks of massive transfusion

The most frequently reported adverse event associated with blood transfusion is the giving of the wrong blood to the patient, which can at worst result in a fatal haemolytic reaction (Stainsby *et al*, 2005). Reports of such events to the Serious Hazards of Transfusion (SHOT) scheme suggest that the risk of error may be particularly high in an emergency situation. Protocols must be in place for the administration of blood and blood components (British Committee for Standards in Haematology Blood Transfusion Taskforce, 1999) and these must be adhered to whatever the degree of urgency.

Transfusion-related acute lung injury (TRALI) and other acute immunologically mediated reactions are uncommon, but occur 5–6 times more frequently following administration of platelets and FFP than red cells (Stainsby *et al*, 2005). The National Blood Service now produces FFP from male donors to reduce the risk of TRALI.

7.1 Metabolic consequences of massive transfusion

Complex metabolic changes may occur due to hypovolaemia, hypothermia and the infusion of large volumes of stored red cells and blood products, especially plasma. The commonest is ionised hypocalcaemia due to citrate toxicity (Dzik & Kirkley, 1988) This may occur as a result of large volume plasma infusion, particularly in the presence of abnormal liver function, where citrate metabolism is slowed. It should be corrected by intravenous infusion of calcium chloride (not gluconate as this requires liver metabolism to release ionised calcium). A dosage of 10 ml of 10% calcium chloride i.v. has been recommended (Spence & Mintz, 2005), alternatively 2.5 to 5.0 mmol calcium chloride can be given in divided doses over 10 min, when the assay should be repeated.

Reduced ionised calcium reduces myocardial contractility, causes vasodilation and exacerbates further bleeding and shock. Ionised calcium is the best measure of active element and most modern blood gas analysers perform this assay.

Hyperkalaemia may occur, due to the high extracellular potassium concentration in stored red cell units. This may be compounded by oliguria and the metabolic acidosis associated with shock. If >6 mmol/l it should be treated with glucose insulin regimens together with bicarbonate to correct acidosis. Early haemofiltration is likely to be required after the arrest of bleeding in the most severe cases.

8 Patient survival

Successful treatment of massive haemorrhage depends on prompt action, good communication and involvement of senior clinicians with the necessary expertise. Survival has improved due to better understanding of the associated physiological changes. This has resulted in more aggressive resuscitation, with effective blood component therapy guided by laboratory/near-patient testing, and effective warming techniques (Cinat *et al*, 1999). Patient age and co-morbidity, duration and degree of shock, and development of DIC influence the outcome. (Erber, 2002)

Suggested topics for audit

1. Initial resuscitation with crystalloids should be preceded by blood sampling for full blood count, coagulation screen, biochemistry, blood gases and blood grouping.
2. Documentation (using a designated checklist sheet and identified member of the resuscitation team) should consist of a minimum dataset that must record: type of blood component or replacement fluid, time given, amount (dosage), indication for replacement, effectiveness of the transfusion. Full traceability of blood components given.
3. Local protocols and algorithms must be available and displayed in high-risk units e.g. Accident and emergency, Intensive care units, Theatre and blood banks.
4. Regular practices of emergency management of massive transfusion should be held and learning points documented to inform protocol development.
5. Regular retrospective audit of management of massive transfusions – review by Transfusion Team and Hospital Transfusion Committee against the guidelines with learning points documented to inform protocol review.

Disclaimer

While the advice and information in these guidelines is believed to be true and accurate at the time of going to press, neither the authors, the British Society for Haematology nor the publishers accept any legal responsibility for the content of these guidelines.

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