Damage Control Resuscitation

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ABSTRACT Damage control resuscitation (DCR) is a strategy for resuscitating patients from hemorrhagic shock to rapidly restore homeostasis. Efforts are focused on blood product transfusion with whole blood or component therapy closely approximating whole blood, limited use of crystalloid to avoid dilutional coagulopathy, hypotensive resuscitation until bleeding control is achieved, empiric use of tranexamic acid, prevention of acidosis and hypothermia, and rapid definitive surgical control of bleeding. Patients receiving uncrossmatched Type O blood in the emergency department and later receiving cumulative transfusions of 10 or more red blood cell units in the initial 24-hour post-injury (massive transfusion) are widely recognized as being at increased risk of morbidity and mortality due to exsanguination. Ideally, these patients should be rapidly identified, however anticipating transfusion needs is challenging. Useful indicators of massive transfusion reviewed in this guideline include: systolic blood pressure <110 mmHg, heart rate > 105 bpm, hematocrit <32%, pH < 7.25, injury pattern (above-the-knee traumatic amputation especially if pelvic injury is present, multi-amputation, clinically obvious penetrating injury to chest or abdomen), >2 regions positive on Focused Assessment with Sonography for Trauma (FAST) scan, lactate concentration on admission >2.5, admission international normalized ratio ≥1.2–1.4, near infrared spectroscopy-derived StO2 < 75% (in practice, rarely available), BD > 6 meq/L. Unique aspects of out-of-hospital DCR (point of injury, en-route, and remote DCR) and in-hospital (Medical Treatment Facilities: Role 2b/Forward surgical teams – role 3/ combat support hospitals) are reviewed in this guideline, along with pediatric considerations.

BACKGROUND

Hemorrhage is the leading cause of preventable death on the battlefield.[1] damage control resuscitation (DCR) emerged as an extension of a principle used by trauma surgeons called damage control surgery (DCS), which limits surgical interventions to those which address life-threatening injuries and delays all other surgical care until metabolic and physiologic derangements have been treated.[2] Recognizing that this approach saved lives, DCR was developed to work synergistically with DCS and prioritize non-surgical interventions that may reduce morbidity and mortality from trauma and hemorrhage.[3] The major principle of DCR is to restore homeostasis and prevent or mitigate the development of tissue hypoxia and oxygen debt as well as coagulopathy.[4] This is accomplished through aggressive hemorrhage control and blood transfusion, which restores tissue oxygenation and not only avoids platelet and coagulation factor dilution but also replaces lost hemostatic potential.

Efforts are focused on blood product transfusion with products that provide the functionality of whole blood (WB, either WB or a mixture of components that includes red blood cells [RBCs], plasma, and platelets), limited use of crystalloids to avoid dilutional coagulopathy and other adjunctive measures used to mitigate hemorrhagic shock and acute traumatic coagulopathy, including:

- Relatively hypotensive resuscitation to avoid re-bleeding (target systolic blood pressure [SBP] 80–90 mmHg in adults);
- Compressive/hemostatic dressings and devices;
- Empiric use of tranexamic acid (TXA) which has been shown to reduce mortality in trauma, likely due to reduction in fibrinolysis;[5]
- Prevention of acidosis and hypothermia; and
- Expeditious delivery to definitive surgical control

Advanced Trauma Life Support (ATLS) guidelines, historically, advocated a linear resuscitation strategy beginning

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with an emphasis on crystalloid infusion, particularly during the pre-hospital phase, followed by the addition of RBCs, and finally plasma. Platelets were delayed until a low platelet count was documented and reserved either for severe thrombocytopenia or thrombocytopenia in the presence of active hemorrhage. As documented in retrospective reports from the civilian trauma literature, this approach resulted in excessive crystalloid use and was associated with a higher risk of dilutional coagulopathy, abdominal compartment syndrome, multiple organ failure, and death;[6] however, selection bias may have contributed to these findings. It should be noted that in recognition of these problems, the latest edition of the ATLS manual (9th ed.) suggests limiting the use of crystalloids to one liter during initial resuscitation and incorporating early use of blood products including plasma and platelets in patients at risk of massive transfusion (MT).[7]

During the conflicts in Iraq and Afghanistan, between 2003 and 2012, 14% of patients admitted to Role 3 Military Treatment Facilities (MTFs, combat support hospitals) received a transfusion of at least one blood product. Of these, 35% received a MT (MT; ≥10 units of RBCs and/or WB in 24 hours). The proportion of patients receiving a MT reached approximately 50% by 2011 in parallel with increasing injury severity scores, decreased crystalloid and colloid use, and increasing use of blood for resuscitation.[8] During this period, mortality fell as military clinicians became experts in the treatment of very severe multisystem trauma accompanied by massive hemorrhage. Civilian ATLS-based practice gave way to a hemostatic resuscitation approach designed to mimic WB functionality. There is now strong retrospective evidence in both civilian and military trauma populations that patients requiring MT benefit from a higher ratio of plasma and platelets to red cells (e.g., 1 unit plasma: 1 unit platelets: 1 unit of packed RBCs [PRBCs]). MT at a 1:1:1 ratio is associated with improved survival.[3,9–12] Recently, prospective randomized data from the pragmatic randomized optimal platelet and plasma ratios (PROPPR) trial revealed that mortality at 3 hours after injury due to exsanguination was lower in patients resuscitated with a 1:1:1 ratio compared with 1:1:2.[13] These were important findings given that the differences between resuscitation strategies were small – and probably best characterized by an early vs. late platelet approach. There was no difference in overall mortality at 24 hours or 30 days, likely due to the confounding effect of head injury. Balanced resuscitation was not associated with increased complication rates.[13,14] Although physicians continue to debate the lessons of the PROPPR trial and the relative benefits of specific blood component ratios, the practice of giving large amounts of crystalloid or RBCs alone in the initial resuscitation period is no longer the standard of care.

BLOOD PRODUCTS FOR DCR

Red Blood Cells

RBC units may be stored for up to 42 days under refrigeration when stored in additive solution (e.g., AS-5). In addition, "frozen" RBCs (fRBCs, stored frozen with glycerol cryoprotectant for up to 10 years at <−65°C, then thawed and rinsed in an automated process) are used interchangeably and successfully with standard RBC units when needed, although these units require at least an hour and a half to prepare. Transfusion of thawed fRBC units without removal of glycerol is absolutely contraindicated and is lethal to the recipient. Thawed and deglycerolized RBCs can be stored for 14 days with refrigeration.

Plasma

Plasma can be stored frozen and thawed "on demand" (FFP), or pre-thawed and stored refrigerated for up to 5 days (so-called "thawed plasma"). The delay in treatment imposed by slow thawing of FFP (up to 30 minutes or more) has necessitated the widespread maintenance of thawed plasma inventories for immediate, emergency use. This typically results in significant waste due to the 5-day post-thaw shelf life. Plasma can also be supplied as "liquid" (never frozen) plasma and stored for 26 days in citrate phosphate dextrose (CPD) anticoagulant solution or 40 days in citrate phosphate dextrose adenine (CPDA-1). Available data suggest that "thawed" and "liquid" plasma may be functionally interchangeable in most trauma patients. Note that no randomized trials have compared these products and that data regarding the hemostatic capacity of liquid plasma stored beyond 28 days are very limited.[15,16] Freeze-dried plasma (FDP) was used by U.S. Forces during World War II and has been in use by the French military since the 1940s. French military FDP is available to U.S. Special Operations Forces under an Investigational New Drug (IND) protocol.[17] FDP is considered functionally interchangeable with other plasma products for trauma resuscitation. FDP or spray-dried plasma may become more broadly available to U.S. Forces in the near future.[18] Although group AB plasma is classically considered to be the only universally compatible plasma, it is now widely recognized that A plasma can, in fact, be considered universal since group A individuals do not generally make high titer anti-B antibodies and B red cells express the B antigen at low density, thus making them much less susceptible to hemolysis than A red cells. The U.S. military, as well as many civilian trauma centers, routinely uses A plasma as universal emergency release plasma.[19–22]

Platelets

In contrast to red cells and plasma, platelets collected in theater by apheresis traditionally have been stored at room temperature (20–24°C), under constant agitation, for a maximum of 5 days with an extension to 7 days if shipped to another facility. These storage conditions are optimized to extend in vivo platelet circulation, but not hemostatic function, safety, or availability.[23] Platelets are vital for hemostasis and their early use in a balanced transfusion strategy is associated with increased survival in trauma.[10,13,24,25] Platelets stored

under refrigeration (1–6°C), or "Cold-Stored Platelets" (CSP), maintained without agitation for up to 3 days in plasma, are approved by the Food and Drug Administration (FDA) for treatment of bleeding patients. Refrigerated storage better preserves platelet hemostatic function and clearly reduces the risk of bacterial growth, the major hazard of transfusing room temperature-stored platelets.[26,27] CSP have been proven effective in clinical trials and used successfully in combat trauma patients in the U.S. Central Command (CENTCOM) area of operations.[23,28] CSP in platelet additive solution (CSP-PAS) retain function for at least 15 days.[29] CSP-PAS can be collected in theater and used interchangeably with other platelet products.[30]

HEMOSTATIC PRODUCTS FOR DCR

Empiric Use of TXA and Other Pharmaceutical Hemostatics

Hemostatic pharmaceutical adjuncts to limit blood loss are another subject of considerable investigation. TXA is the only therapy in this class that has been found to reduce mortality in a large randomized controlled trial. Strong evidence demonstrates a significant improvement in survival following the early use of TXA, but only when given with 3 hours of injury, after which mortality is higher.[5] Prospective and retrospective evidence from a decade ago suggested that rFVIIa used early in the resuscitation of patients with MT results in decreased blood transfusion, but not higher survival. The use of rFVIIa is no longer recommended in most trauma patients since it has not been shown to reduce mortality and may increase risk of adverse events.[31–34] Prothrombin complex concentrates are only indicated for patients requiring urgent warfarin reversal and have not been adequately studied in a broad trauma population. Prothrombin complex concentrates should not be used in trauma outside the context of a clinical trial as they may cause harm due to excessive thrombogenicity.[35] Fibrinogen concentrate has not been studied adequately in trauma patients either, but several factors suggest that it may be helpful. These include: (1) fibrinogen is the fundamental substrate of clot formation; (2) fibrinogen is rapidly consumed in trauma; and (3) cryoprecipitate, a less purified source of fibrinogen, has been shown to be an essential component of MT protocols for mitigating the dilutional coagulopathy caused by red cell additive solution and anticoagulant.[11,36–38]

Management Principles for DCR

In casualties at high risk of hemorrhagic shock, TXA reduces mortality if given within 3 hours of injury. TXA given >3 hours post-injury increases the risk of mortality. For eligible casualties (see section above titled "Recognition of Patients Requiring DCR"), one gram of I.V. TXA should be administered in 100 mL of normal saline over 10 minutes, followed by another 1 gram dose delivered over 8 hours; the first dose must be given within 3 hours of injury. Although lactated Ringer's solution is compatible with TXA, its use should be avoided in this setting since the mixing of calcium-containing lactated Ringer with blood products in chaotic resuscitation settings may cause clotting of blood products and thromboembolic phenomena. Normal saline and PlasmaLyte A are the only crystalloid solutions compatible with blood products. For eligible casualties (see section above titled *Recognition of Patients Requiring DCR*), 1 g of I. V. TXA should be administered in 100 mL of normal saline solution over 10 minutes, followed by another 1 g dose delivered over 8 hours.

Recognition of Patients Requiring DCR

Patients receiving uncrossmatched Type O blood in the emergency department (ED) or resuscitation area and later receiving cumulative transfusions of 10 or more RBC units in the initial 24-hour post-injury (MT) are widely recognized as being at increased risk of morbidity and mortality due to exsanguination. Ideally, these patients should be rapidly identified and hemostasis established at the earliest level of care possible in order to prevent or mitigate shock and coagulopathy. Due to diagnostic challenges, particularly in the case of truncal hemorrhage, anticipating the transfusional needs of these patients requires experience and the coordination of extensive resources, including development of MT protocols.

Robust pre-hospital data are lacking, but a number of factors predict the need for MT support in trauma.[39] In a patient with serious injuries, the presence of three of the four features below indicates a 70% predicted risk of MT and 85% risk if all four are present:

- Systolic blood pressure <110 mmHg
- Heart rate >105 bpm
- Hematocrit <32%
- pH < 7.25

Other risk factors associated with MT or at least need for aggressive resuscitation:[40–43]

- Injury pattern (above-the-knee traumatic amputation especially if pelvic injury is present, multi-amputation, clinically obvious penetrating injury to chest or abdomen)
- >2 regions positive on FAST scan
- Lactate concentration on admission >2.5
- Admission INR ≥1.2–1.4
- Near infrared spectroscopy-derived StO2<75% (in practice, rarely available)
- BD > 6 mEq/L

Recognition of clinical patterns associated with the need for MT is essential for effective triage. These include: uncontrolled truncal or junctional bleeding, uncontrolled major bleeding secondary to large soft tissue injuries, proximal, bilateral, or multiple amputations, a mangled extremity, clinical signs of coagulopathy (e.g., paucity of clots or petechial bleeding), or severe hypothermia. It is critical to communicate with the blood bank at the MTF when a potential MT patient has been identified.

Blood banks within theater have developed procedures for providing blood products in the appropriate proportion to support resuscitative efforts. Upon arrival to the ED, laboratory evaluation such as viscoelastic testing (Thromboelastography [TEG] or Rotational Thromboelastometry [ROTEM]) may also facilitate early identification of patients who will require MT, although this technology is not widely available in the deployed setting, particularly at Role 2 facilities.[44,45] It should be noted that many point-of-care coagulation tests that measure prothrombin time/international normalized ratio (PT/INR) have linear ranges only between INR 2.0–3.0 and are unreliable in clinical conditions characterized by loss of fibrinogen. These devices should not be relied upon to evaluate the coagulation function in trauma patients.[46,47] An example of MT protocol is presented in Table I.

POINT OF INJURY, EN-ROUTE, AND REMOTE DCR

Optimization of Fluids

Volume resuscitation, particularly crystalloid and colloid, should be used sparingly in the pre-hospital setting, given the potential for harm and the limited resources; blood products are preferred for hemorrhagic shock resuscitation.[3,48]

Casualties at low risk of developing shock should not receive IV fluids or adjunctive medications.

The order of priority for fluid administration should be:

– whole blood (Group O low titer preferred);
– blood components at a 1:1:1 ratio;
– RBCs plus plasma = 1:1 ratio;
– plasma with or without RBCs; and
– RBCs alone.[49]

Whole Blood

In deployed environments, the inability to supply blood components due to logistical constraints led to the use of WB collected onsite from "walking blood banks," especially to provide platelets for hemostatic resuscitation. It should be noted that blood products collected in the deployed setting (platelets or WB) are not prospectively tested for transfusion-transmitted diseases (TTDs). Recipients of these products must be tested at 3, 6, and 12 minutes post-transfusion to monitor for disease transmission. WB delivers all the components of blood in the correct ratio and is independently associated with improved survival.[50–52] Type-specific whole blood (TSWB), often referred to as fresh whole blood (FWB), is collected from donors in the deployed setting and must be an ABO match with the recipient. The availability of TSWB may be limited due the constrained pool of donors who must be tested for TTDs and blood group compatibility with recipients. In addition, the chaotic conditions of mass casualty scenarios complicate the matching of blood types between donors and recipients, increasing the risk of clerical errors causing hemolytic transfusion reactions. In order to improve the availability and safety of WB, low anti-A and anti-B titer (<1:256 by tube method) group O blood has been identified as a practical, effective universal blood product for resuscitation of exsanguinating hemorrhage.[17,49] Like all blood donors, "O low titer" donors should be tested for TTDs and undergo confirmatory typing and an antibody screen (type and screen) in addition to testing for anti-A and anti-B antibodies. Group O

TABLE I. Example of a MT Procedure at an USCENTCOM Level III Facility Considerations for Use with MT

A flexible procedure for use in the ED, operating room, and intensive care unit which can be initiated or ceased by the site-specific provider as dictated by the patient's needs when in that specific venue. It consists of batches as defined below, which vary in composition, but are directed toward approximating a 1:1:1:1 ratio of PRBC, FFP, platelets and cryoprecipitate (cryo). Note: one unit of apheresis platelets is approximately the equivalent of 6 units random donor platelets, therefore 1 u apheresis platelets should be given for every 6 units of PRBC to approximate 1:1:1 resuscitation.
Initiate MT procedure if patient has received 4 u PRBC/4 u FFP emergency release blood products • Pack One: 4 u PRBC, 4 u FFP, 1 u apheresis platelets, 1 10-unit bag cryo. Strongly consider the early use of TXA: Infuse 1 g of TXA in 100 mL of 0.9% NS over 10 minutes intravenously in a separate IV line from any containing blood and blood products. (More rapid injection has been reported to cause hypotension.). Hextend should be avoided as a carrier fluid. Infuse a second 1-g dose intravenously over 8 hours infused with 0.9% NS carrier. • Pack Two: 4 u PRBC and 4 u FFP • Pack Three: 4 u PRBC, 4 u FFP, 1 u apheresis platelets, 1 10-unit bag of cryo and ± rFVIIa (obtained from Pharmacy) • Pack Four: 4 u PRBC and 4 u FFP • Pack Five: 4 u PRBC, 4 u FFP, 1 u apheresis platelets, and 1 10-unit bag of cryo A reassessment of the progress of the resuscitation, hemostasis and the need to continue the MT Procedure should be conducted between the providers taking care of the patient at that time • Packs six and seven are identical to packs four and five • Packs eight and nine are identical to packs four and five
Definitions Emergency Release: Uncrossmatched 4 u PRBC (O+ or O- for males, O- for females) and 4 u AB or A FFP (NOTE: A FFP is not a universal donor but its use in MT patients when supplies of AB FFP are limited or absent may improve survival and help preserve resources with a low risk to the patient. The decision to use A FFP or to switch from AB FFP to A FFP in the same patient should be a decision based on the interaction of the medical/surgical staff in concert with laboratory staff. Once the patient's type has been identified, type-specific plasma should be given as soon as possible).

Low Titer Whole Blood (LTOWB) can be collected from pre-screened walking blood banks in the deployed setting or collected in Armed Services Blood Program donor centers and stored refrigerated for 21 days in CPD or 35 days in CPDA-1.[53] Available data suggest that Cold-Stored WB (CWB) will provide platelet hemostatic function during the first 2 weeks of storage. Function is moderately reduced during the remaining shelf life (21 days for CPD WB and 35 days for CPDA WB), but it should be noted that WB plasma hemostatic function is comparable to that of liquid plasma and that CWB remains a relatively hemostatic product (compared with RBCs alone) throughout its shelf life.[54–56] Patients receiving MT with CWB stored for more than 2 weeks may require additional support with platelet transfusions or FWB (consider a ratio of 3:1 of CWB: FWB as available). Similarly, CWB that has been leukoreduced and that contains fewer or effectively no platelets requires supplementation with platelet or FWB transfusion. Cold-stored LTOWB and TSWB have been used successfully and safely to treat trauma and other causes of massive hemorrhage, such as obstetric emergencies and bleeding in cardiac surgery, in leading U.S. civilian hospitals.[57–65] For guidance regarding use of fRBCs and FWB, see the Joint Trauma System (JTS) CPGs entitled Frozen and Deglycerolized Red Blood Cells and Whole Blood Transfusion, respectively.[66,67]

Albumin (5% or 25%) provides effective and more physiologic volume expansion than other colloids, but given alone contributes to hemodilution. Consideration should be given to supplementing albumin with fibrinogen concentrate and TXA, if available.

BLOOD PRODUCT TRANSFUSION

Cryoprecipitate is available in hospital settings and should be added to the component mix to create a 1:1:1:1 ratio of products in order to adequately supply fibrinogen and other clotting factors (Factors VIII, FXIII, and vWF).

When operationally necessary due to component shortages, WB from walking blood banks can be life-saving. For additional information, refer to the JTS CPG titled Whole Blood Transfusion.[67]

Hextend or Hespan use should be avoided as these products worsen coagulopathy.[68,69]

Hypertonic saline does not improve mortality in hemorrhagic shock and should only be used for patients with traumatic brain injury and evidence of raised intracranial pressure.[70]

A key element of fluid optimization is careful documentation of all fluids, interventions, and medications given in the pre-hospital phase – for more information, see the JTS CPG titled Battle and Non-Battle Injury Documentation: The Resuscitation Record.[71]

Calcium (consider one 10 ml ampule of 10% calcium chloride, or 30 ml of 10% calcium gluconate) should be given to patients in shock after approximately four units of citrated blood products transfused. Ideally, ionized calcium should be monitored.

Blood products should ideally be warmed with approved in-line blood heaters with the goal of transfusing products warmed to 37°C.

ADJUNCTIVE THERAPIES

Hypotensive Resuscitation

In casualties without central nervous system (CNS) injury, resuscitation prior to surgical control of bleeding focuses on maintaining a relatively lower target SBP (~90 mmHg) to reduce re-bleeding by minimizing intravascular hydrostatic pressure. While empirically attractive, this approach represents a significant logistical challenge due to the difficulties in obtaining frequent and high fidelity SBP monitoring between the time of injury and definitive care and due to the limited interventions possible in challenging environments. Acute changes in mental status or pulse quality have been used to detect impending hypotensive shock, but have not been adequately tested and can be misleading in the acute setting by the concomitant use of analgesics and sedatives such as ketamine. Hypotensive resuscitation should not be utilized for patients with CNS injury because of associated adverse outcomes in this population. In general, patients with CNS injury benefit from avoidance of even transient hypotension and hypoxia. For additional information, see the CPG titled Neurosurgery and Severe Head Injury.[72]

Compressive/Hemostatic Dressings and Devices

Prevent further hemorrhage with direct pressure, topical hemostatic dressings, and/or tourniquets, if possible, to minimize the risk of shock. REBOA can be highly effective if rapidly implemented by skilled providers.

Prevention of Acidosis and Hypothermia

Metabolic acidosis resulting from acute trauma is a consequence of inadequate tissue perfusion leading to lactic acid production and is best addressed with resuscitation with WB or equal ratio components. Crystalloid resuscitation will contribute to the acidosis and should be avoided. Hypothermia is multifactorial and strategies should address as many causes as are identified, including cold exposure, cold resuscitation fluids, significant blood loss, and shock. Heated fluids, fluid blankets, and ventilators may not be available, but wounds should be covered, "space blankets" (e.g., HPMK) used to cover the casualty, and shock avoided or treated. See JTS CPG Hypothermia Prevention for additional information.[68]

Expeditious Delivery to Definitive Surgical Control

Casualties may require care as described and emergency procedures for life-threatening conditions in the pre-hospital setting; however, these should be balanced against the need to expeditiously deliver the patient to definitive care. DCS at the forward surgical units should only focus on control of hemorrhage and contamination. Only absolutely necessary

procedures should be performed. In general, every effort should be made to deliver the critically injured casualty to the highest available level of care as rapidly as possible.

DCR AT MEDICAL TREATMENT FACILITIES

Although principles remain the same, DCR in medical facilities differs in that there are more resources available, including access to operative surgical control and some therapies such as TXA may have already been given in the pre-hospital phase. Resuscitation to physiologic endpoints such as lactate and StO2 should be considered since tissue hypoxia and oxygen debt are known drivers of coagulopathy. Reversal of tissue hypoxia should thus be a central tenant of hospital-based resuscitation.

ADJUNCTIVE THERAPIES

Hypotensive Resuscitation

As in the pre-hospital period, resuscitation of casualties without Central Nervous System (CNS) injury prior to definitive surgical control should maintain a lower target SBP (~90 mmHg) to reduce hemorrhage by minimizing intravascular hydrostatic pressure. Hypotensive resuscitation should not be utilized for patients with isolated CNS injury because of associated adverse outcomes in this population. For additional information, see the JTS CPG titled Neurosurgery and Severe Head Injury.[72]

Compressive/Hemostatic Dressings and Devices

Until definitive surgical control is established, prevent further hemorrhage with direct pressure, topical or intratruncal hemostatic dressings, and/or tourniquets to avoid the development of shock. In extremis, procedures such as resuscitative thoracotomy or REBOA are indicated. Use of these devices should occur as rapidly as hemorrhage is identified and should not delay transport to the operating room.

Prevention of Acidosis and Hypothermia

Metabolic acidosis resulting from acute trauma is a consequence of inadequate tissue perfusion leading to lactic acid production and is best addressed with resuscitation with WB or equal ratio components (1:1:1) in combination with early hemorrhage control. Crystalloid or colloid resuscitation will contribute to the acidosis (as well as dilutional coagulopathy) and should be avoided. Hypothermia is multifactorial and strategies should address as many causes as are identified, including cold exposure, cold resuscitation fluids, significant blood loss, and shock. Hypothermia occurs even in when ambient temperatures are elevated and medical personnel are uncomfortably warm, due to blood loss and hypoperfusion. Treatment should include urgent, active re-warming with all available means including heated fluids, fluid blankets, ventilators, warm environments, and rapid surgical care to minimize blood and heat loss.

PEDIATRIC CONSIDERATIONS

There are no prospective studies of transfusion resuscitation in pediatric trauma. Most major children's centers extrapolate from adult literature and are using similar DCR strategies in major hemorrhage. There are currently no data determining which patients may benefit from these strategies.

For children under a weight of 30 kg, transfusions of RBC units, FFP, or apheresis platelets should be given in "units" of 10–15 mL/kg. One unit of cryoprecipitate is typically administered for every 10 kg of body weight. Blood volume in children can be estimated at between 60 and 80 mL/kg. Bear in mind that a "trauma pack" containing 6 U RBCs + 6 U FFP + 1 U apheresis platelets will deliver between 3,000 and 4,000 mL of intravascular volume. A child of 30 kg may have a total blood volume of 1,800–2,400 mL. Over-resuscitation contributes to morbidity and mortality. It may be more convenient and safe to resuscitate children with WB since this product delivers full oxygen delivery and hemostatic functionality and may support more accurate volume dosing. For example, a typical unit of whole blood contains about 500–600 mL (depending on bag type and volume: 450 or 500 ml blood volume plus anticoagulant). For a severely injured, shocked child, a quarter to a half of a WB unit may provide adequate initial resuscitation, which can then be further titrated.

Although there are limited retrospective data demonstrating the benefit of TXA in pediatric trauma,[73] there are studies of TXA use in pediatric cardiac, orthopedic and cranial surgeries showing overall safety and decreased transfusion requirements.[74–77] There is no prospectively validated dosing available for pediatric trauma but loading doses of 10–100 mg/kg IV followed by 5–10 mg/kg/h infusion doses are commonly used in elective surgery. The UK Royal College of Pediatrics and Child Health has recommended a loading dose of 15 mg/kg (up to 1 gm) followed by 2 mg/kg/h over 8 hours (or up to 1 gm over 8 hours). This regimen reflects standard adult dosing in trauma.[69]

Viscoelastic clot testing (e.g., TEG or ROTEM) can be utilized to direct transfusion requirements as in adults utilizing the same thresholds discussed in the full JTS CPG. Viscoelastic testing should not be used to withhold TXA during initial resuscitation of bleeding trauma patients.[68,78]

CONCLUSION

The DCR approach to the initial management of a critically injured casualty requires a significant expenditure of resources and the coordination of a diverse group of health care providers. This is frequently performed in a clinical scenario of multiple casualties and limited resources. It is incumbent upon the clinical leaders at each level of care to be fully versed on available resources and to employ them judiciously and appropriately. Patients requiring MT should be resuscitated using DCR principles and should undergo early DCS.

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