ICU MANAGEMENT & PRACTICE

ICU Volume 7 - Issue 1 - Spring 2007 - Matrix Features

Management of Massive Operative Blood Loss

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Introduction

A massive blood loss is defined as the loss of 100 % of circulating blood volume within 24 hours, 50% of circulating blood within 3 hours, 150mL/min or 1.5mL/kg/min over at least 20 minutes (Erber 2002; Hiippala 1998; Stainsby et al. 2000). Therapeutic goals are the maintenance of: (1) normovolemia, (2) adequate tissue oxygenation, (3) sufficient coagulatory function, (4) normothermia, (5) electrolyte balance and (6) acid base balance (Pape et al. 2006a).

Normovolemia

When a loss of 30% of the circulating blood volume remains unresuscitated, hypovolemic shock is imminent (Peitzman et al. 1995). Therefore, any blood loss should be treated initially by the infusion of crystalloid and colloidal solutions, in order to provide normovolemia and nutritive tissue perfusion.

Tissue Oxygenation

Acellular fluid resuscitation implies the dilution of the cell mass remaining in the vasculature (hemodilution) with a corresponding dilutional anemia, or decrease of Hb-concentration and arterial O2-content (CaO2). Although hemodilution initially improves tissue perfusion (Messmer and Sunder- Plassmann 1974), progressive dilutional anemia requires the elevation of CaO2 to maintain adequate tissue oxygenation. The first step to achieve this goal is to elevate the inspiratory O2 fraction (FiO2) to 1.0 (hyperoxic ventilation, HV). Although frequently underestimated, the efficacy of HV to provide adequate tissue oxygenation even in critical normovolemic anemia has been demonstrated repeatedly (Meier et al. 2004; Pape et al. 2006b).

A definitive increase of CaO 2 will be achieved by the transfusion of pRBCs. Generally, pRBC transfusion management should aim to keep Hb-concentration above 6g/dL in otherwise healthy patients and above 8-10g/dL in patients with elevated cardiovascular risk (Weiskopf 1996). Although a healthy organism may tolerate Hb-concentrations beyond 6g/dL, Hb-levels lower than 6-8g/dL should be avoided, since the dynamic of blood loss is often difficult to anticipate.

The transfusion of allogeneic blood is still associated with risks for the recipient, such as “clerical error”, transmission of infectious diseases, immunomodulation or transfusion-related lung injury (TRALI) (Spahn and Casutt 2000). However, the concept of intraoperative cell salvage and autologous retransfusion has been demonstrated to be highly effective in reducing the need for allogenic blood transfusion (Dai et al. 2004).

Coagulation

During massive operative bleeding, coagulatory function is impaired by: (1) wash-out of platelets and clotting factors (dilutional coagulopathy), (2) hypothermia and acidosis and (3) disseminated intravascular coagulation (DIC, consumptive coagulopathy). After exchange of one circulating blood volume with acellular solution and RBCs, plasmatic coagulatory factors are diluted to 37% of the initial value. Fibrinogen is the first factor to decrease in this situation, so that the severity of dilutional coagulopathy can be estimated from fibrinogenconcentration, if other reasons for decreased fibrinogen (e.g. DIC) are excluded (Erber 2002). Fresh frozen plasma (FFP) represents a physiological composition of all coagulatory factors and plasma proteins. During massive bleeding, sufficient bolus-doses of FFP (i.e. 5-20mL/kg) are recommended to achieve adequate concentrations of coagulation factors (Hiippala 1998). When FFPtransfusion becomes insufficient to provide adequate levels of fibrinogen-concentration and factor activities, the additional substitution of fibrinogenconcentrates and/or prothrombin complex concentrates (PCC, FII, FVII, FX, FIX) may become necessary (Blauhut 1999).

The majority of massively bleeding patients becomes thrombocytopenic after the exchange of two circulating blood volumes (Hiippala 1998). Platelet transfusion should aim at PC>50000; for intracranial surgery, PC>100000 is recommended (Weiskopf 1996). Although presently only approved for the treatment of haemophiliac patients, recombinant activated factor VII (rVIIa) has been successfully applied in non-haemophiliac patients suffering otherwise untreatable bleeding (Gowers and Parr 2005; O'Connell et al. 2003). A “last-ditch-use” of rVIIa in profound dilutional coagulopathy, however, seems ineffective (Clark et al. 2004).

Hypothermia

The rapid replacement of massive blood losses frequently induces hypothermia, which impairs platelet function and plasmatic coagulation.

Moreover, hypothermia compromizes cardiovascular performance, O_2-transport (left-shift of Hb-dissociation- cure) and hepatic elimination of drugs. These unfavorable effects underline the necessity to keep the patient warm and to warm up all blood products and resuscitation fluids as effectively as possible (Sessler 1997).

Electrolyte and Acid-Base Balance

Electrolyte disorders resulting from massive transfusion consist of hyperkalemia resulting from hemolysis and increased extracellular potassium in pRBC-units, as well as transient hypocalcemia related to the high citrate-content in pRBCs and FFP. Depending on severity, these disorders may require pharmacological intervention. Acidic pH of stored pRBCs and microcirculatory disorders frequently result in acidemia, which may be corrected by adjusting ventilation buffering (Na-bicarbonate or TRISS-buffer).

Outcome After Massive Transfusion

Although survival rates after massive transfusion have increased in recent decades, mortality of massive blood loss still ranges between 40% and 70% (Vaslef et al. 2002). Nevertheless, the reduction of mortality is predominantly attributable to improved logistics of blood supply and development of modern critical care systems, both enabling a goal-directed management of massive blood loss (Mikhail 2004). For patient survival, the most crucial treatment priority is the prevention of hemorrhagic shock through provision of adequate tissue oxygenation at all times. Additionally, restoration of homeostasis (normothermia, reversal of base excess and acidosis) and aggressive correction of coagulopathy contribute to improved survival (Cinat et al. 1999). Therefore, the best practices discussed in this article may help improve a patient’s chance for survival following massive blood loss.

Published on : Thu, 15 Aug 2013