

The beginning of the end for damage control surgery

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Damage control surgery has been practised for millennia. Exsanguinating battlefield victims with extremity injuries have undergone rapid amputation throughout history. Pringle and Halsted both described 'packing' liver injuries in the early 20th century. In the modern era, Lucas and Ledgerwood¹ described perihepatic packing in three of 637 patients with liver injuries treated at Detroit General Hospital in 1976 and Feliciano *et al.*² reported a 90 per cent survival rate in ten patients with severe liver injuries treated at the Ben Taub General Hospital in 1981. Damage control surgery has become widespread. It is now performed with much greater frequency, raising the question of whether it is being overutilized.

The philosophy of damage control dictates abbreviation of operations before the development of physiological exhaustion. The goal is to interrupt the vicious cycle or lethal triad of hypothermia, acidosis and coagulopathy by resuscitation, coagulation factor replacement and rewarming in the intensive care unit, followed by a return to the operating room for definitive therapy. Unfortunately, resuscitation practices and the failure to achieve early haemostasis have contributed significantly to the incidence of this 'bloody' vicious cycle, further promulgating the damage control philosophy. Newer strategies that focus on improved haemorrhage control, limitation of crystalloids for resuscitation and rapid goal-directed correction of coagulopathy should reverse this trend toward increased use of damage control.

Early treatment of injured patients has traditionally focused on aggressive resuscitation with high chloride-containing crystalloid solutions. Resuscitation with crystalloid exacerbates each element of the lethal triad. Prehospital and early in-hospital resuscitation has been shown to increase morbidity and mortality in patients with penetrating torso trauma³. Crystalloid resuscitation results in increased blood pressure that displaces clots in patients with uncontrolled haemorrhage. The absence of clotting activity in both crystalloid solutions and packed red blood cells contributes to dilutional coagulopathy. High chloride content in crystalloid solutions exacerbates the acidosis of shock, and prehospital fluid maintained at room temperature contributes to hypothermia. Aggressive fluid resuscitation has been associated with secondary abdominal compartment syndrome necessitating a damage control approach even in the absence of abdominal injury⁴.

Novel strategies are designed to prevent the lethal triad. Recent versions of the Advanced Trauma Life Support course focus on stopping the bleeding. This can be accomplished in a number of ways including a resurgence of tourniquet use and new haemostatic dressings instead of simple gauze. Those involved in military trauma are being trained to withhold resuscitation unless a casualty has either a lowered mental state or absent pulse, and to give only enough fluid to restore these abnormalities. The fluid of choice is 6 per cent hetastarch in a balanced salt solution that contains lactate. This has been

shown not to exacerbate coagulopathy by thromboelastography even with substantial volumes and has been shown to reduce blood loss in patients undergoing major surgery compared with 6 per cent hetastarch in normal saline⁵.

Originating from experience with military casualties, haemostatic resuscitation has emerged as a strategy to prevent the lethal triad in patients who are predicted to require massive transfusion. This strategy dictates transfusion of packed red blood cells, plasma, platelets and cryoprecipitate in a 1:1:1:1 ratio while minimizing the use of crystalloid. It is designed to avoid the dilutional coagulopathy associated with traditional crystalloid and packed red blood cell resuscitation. Multiple retrospective studies from both military and civilian sources have shown an association between haemostatic resuscitation and reduced mortality^{6,7}. While this high ratio approach to transfusion appears superior to traditional resuscitation, it is not optimal in correcting coagulopathy. Transfusion of 1 unit of packed red blood cells, 1 unit of fresh frozen plasma, 1 unit of platelets and 1 unit of cryoprecipitate yields a haematocrit of 29 per cent, a platelet count of 87 000/mm³, 65 per cent coagulation activity and 750 mg fibrinogen. A single unit of fresh whole blood yields a haematocrit between 38 and 44 per cent, a platelet count between 150 000/mm³ and 400 000/mm³, 100 per cent clotting factor activity and 1500 mg fibrinogen⁸. The use of whole blood in the military setting was associated with improved survival

compared with component therapy in a retrospective analysis⁹.

The use of procoagulant concentrates to avoid or correct coagulopathy is increasing. These agents are rapidly available and correct coagulopathy without large volume transfusions. In prospective randomized trials, recombinant factor VIIa reduced the need for allogenic transfusions and massive transfusions, and reduced the incidence of adult respiratory distress syndrome in patients suffering from blunt trauma^{10,11}. Prothrombin complex concentrate (PCC) is derived from human plasma and contains variable amounts of factors II, VII, IX and X. Three thousand units of PCC will replace 40–80 per cent of normal factor activity. Fibrinogen concentrate is also available as a lyophilized powder derived from human plasma. Reconstituted fibrinogen concentrate has a concentration similar to that of cryoprecipitate (1 g/50 ml). In a study of 131 severely injured patients who required 5 units or more of packed red blood cells, fibrinogen concentrate and PCC were given as first-line therapies for coagulopathy based on thromboelastography. Transfusion of fresh frozen plasma and cryoprecipitate was avoided in the vast majority of patients and outcomes were better than predicted¹².

The advent of new technologies and strategies for the management of severely injured patients is already impacting on the damage control paradigm. A focus on rapid control of haemorrhage followed by directed resuscitation with agents that prevent or correct coagulopathy, together with avoidance of over-resuscitation with crystalloid, will reduce the need for damage control. In this new paradigm, patients with bleeding will

be brought to the operating room where the bleeding can be rapidly stopped. Coagulopathy will be corrected with concentrates and directed component therapy as necessary, crystalloid therapy will be minimized and definitive therapy completed at the first operation in patients who are warm, well perfused and without coagulopathy.

Disclosure

The author declares no conflict of interest.

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