Trauma resuscitation and the damage control approach

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Abstract

Haemorrhage remains the biggest killer of major trauma patients. One-third of trauma patients are coagulopathic on admission, which is exacerbated further by other factors. Failure to address this results in poor outcomes. Damage control resuscitation is current best practice for bleeding trauma patients, and encompasses damage control surgery and damage control radiography. This review provides a summary of the latest concepts in the rapidly evolving field of trauma resuscitation management.

Keywords Damage control; massive haemorrhage; resuscitation; trauma

Introduction

Damage control (DC) was first termed to describe measures taken by a ship's crew to reduce damage that immediately threatened the integrity of the hull, and enabled return to port for definitive repairs. DC has been traced to the British Royal Navy as early as the 1600s.

Rotando and colleagues[1] were credited as the first to use the term DC in the medical literature to describe improved survival in exsanguinating, penetrating, abdominal trauma using damage control surgery (DCS), comprising haemorrhage control, peritoneal decontamination and packing and rapid closure rather than definitive laparotomy, although similar strategies had previously been documented.

Resuscitation aims to restore physiological normality to the acutely unwell and, may incorporate various techniques, including surgical and radiological intervention.

Damage control resuscitation (DCR) encompasses techniques to restore physiological balance to the major trauma patient, and describes a systematic approach to minimize haemorrhage, prevent coagulopathy and maximize tissue oxygenation to optimize patient outcome. DCR incorporates the concepts of both DCS and Damage Control Radiology (DCRad).

The concept of DCR was initially used in military situations to describe approaches to the management of the severely injured trauma patients and has evolved significantly in the last two decades.[2] Early resuscitation now employs a horizontal team approach, where rapid restoration of physiology has primacy over definitive surgical repair ('operating on physiology, not anatomy'). This philosophy has increasingly been adopted in the civilian environment.

DCS describes the specific, systematic surgical approaches focussing on normalizing physiology from the dual insults of injury and surgery, as opposed to providing immediate definitive repair.[3,4] DCRad incorporates diagnostic and interventional radiological solutions used to treat severely injured patients.[5]

Recent history of trauma care

Advances in trauma care commonly occur during warfare, where high numbers of seriously injured soldiers are treated, although a landmark change was the introduction of the Advanced Trauma Life Support® (ATLS) programme in 1978. ATLS was originally targeted at doctors with little expertise in trauma and provides a structured system for recognizing life-threatening problems and instigating appropriate interventions. The ATLS 'Airway, Breathing, Circulation, Disability, and Exposure' (ABCDE) mantra is familiar the world over. Whilst it is likely this approach has saved many lives over the years, with the advent of regional trauma networks and experience gained from large recent military campaigns, an approach that reaches beyond ATLS is now required in civilian practice.

DCR is a more recent evolution enhancing resuscitation of major trauma. Over the last decade, DCS and DCR have evolved significantly as increasing experience has driven clinical innovation.

Pathophysiology

Acute traumatic coagulopathy and trauma-induced coagulopathy

Haemorrhage remains the leading cause of death in both civilian and military trauma and considerable research has improved our understanding and treatment of massive haemorrhage. Initially, the 'bloody vicious triad' of acidosis, hypothermia and coagulopathy with increased consumption of platelets and clotting factors together with dilution by crystalloids was thought to produce trauma-induced coagulopathy (TC). Subsequently, a much more complicated picture is now emerging.

In 2003 Brohi and colleagues identified the existence of acute traumatic coagulopathy (ATC)[6] ATC is a complex and multifactorial endogenous process occurring after severe injury in approximately a third of patients and independently predicts death and prolonged ICU stay. Hypoperfusion and poor tissue oxygenation are thought to be the main drivers and the onset of ATC is fast, commonly within 30 minutes of injury. Worsening coagulopathy may develop from enzyme and platelet dysfunction, produced by hypothermia, acidosis and serum dilution, which can be intensified during the resuscitative phase particularly if a large amount of crystalloid is given. ATC is exacerbated by these factors, and collectively these processes constitute TC. Our currently incomplete understanding of haemostasis was summarized recently by Cohen et al.[7] and an overview is displayed in Figure 1.

Hypothermia

Hypothermia has many systemic effects including reduced respiratory function and cardiac output. Enzyme kinetics slow down; below 33°C coagulation efficacy is approximately 50%

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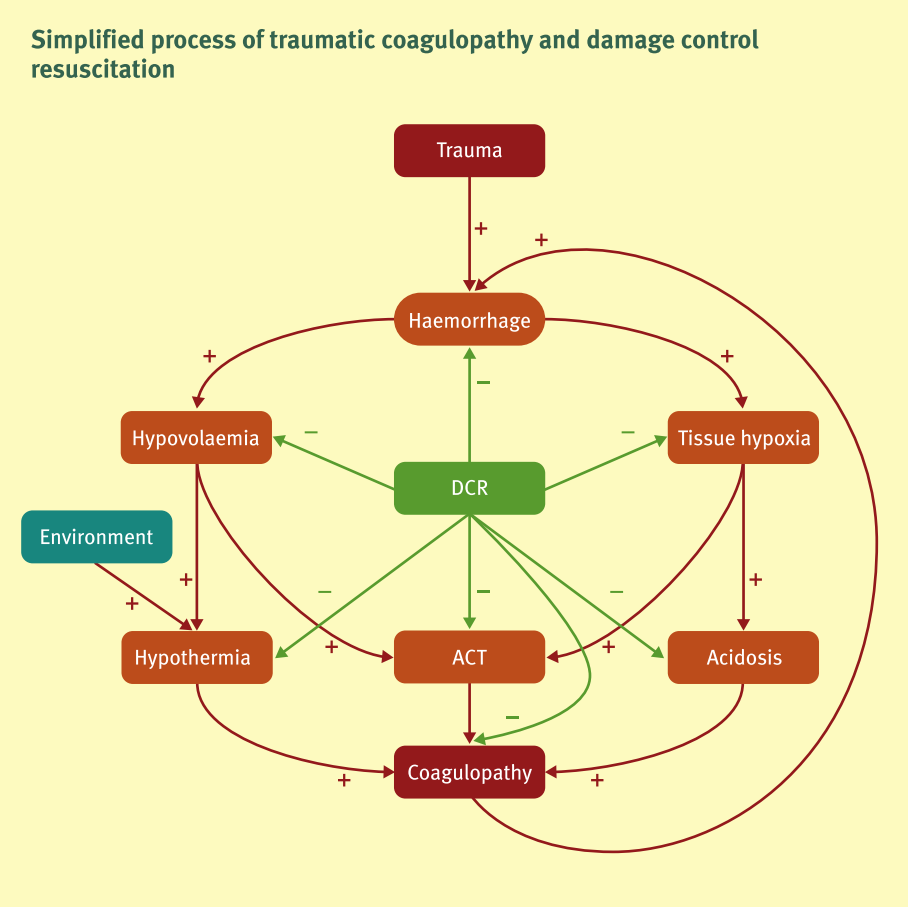

Figure 1

that of 37°C despite normal levels of clotting factors. Hypothermia inhibits the coagulation cascade, increases fibrinolysis and reduces platelet number and function due to morphological changes which decrease platelet aggregation, alter platelet surface molecule expression, and increase platelet sequestration in to the liver and spleen.

Acidosis

pH changes effect coagulation by reducing enzymatic conversion of coagulation factors into their active forms, particularly thrombin generation, and by alteration of platelet activity through decreasing platelet count and modification of calcium ion binding site morphology.

As our understanding of the pathophysiology of severe injury and TC has evolved so our treatment strategies have also adapted.

Changing paradigms

ABC becomes <C>ABC

The most important treatment for haemorrhage is to stop it. Recognition that compressible haemorrhage from extremity wounds kills rapidly (but can be treated with minimal training and equipment) resulted in changing the dogma of ABCDE to <C>ABCDE, where <C> denotes Catastrophic Haemorrhage.[2] This utilizes field dressings, tourniquets and topical haemostatic agents in a stepwise fashion. It follows that severe internal bleeding must also be arrested in a timely manner, and hence early invasive interventions such as DCS and DCRad are an essential part of DCR.

Fluid resuscitation

Fluid resuscitation in the 1970s focussed on initial high-volume replacement with crystalloid, followed by packed red blood cells (PRBC). ATLS has long advocated this strategy, and despite increasing evidence of harm, still advocates replacing each 1 ml of blood loss with 3 ml of crystalloid, up to 2 litres (adult) or 20 ml/kg (paediatric) of crystalloid. Uncontrolled crystalloid infusion in trauma patients has been termed the 'vicious salt water cycle' due to its association with serious complications such as acute lung injury, abdominal compartment syndrome, worsening coagulopathy, and (in shocked burns patients) reduced end organ perfusion. Ley and colleagues[8] demonstrated that crystalloid infusion of 1.5 litres or more in trauma patients was an independent risk factor for mortality.

Mature trauma systems now replace blood with blood products from the outset, although the exact ratio and quantity of products is still under investigation. Some trauma systems (mainly military) utilize whole blood transfusions for resuscitation, although most advanced civilian trauma systems are still reliant on PRBC, fresh frozen plasma (FFP), platelets (Plt) and cryoprecipitate (Cryo). These are typically given in an empirical manner initially, according to local policy.