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 specific techniques in resuscitation, surgery and interventional radiography. This article summarizes the latest strategies in the field of trauma resuscitation.

Keywords Damage control; massive haemorrhage; resuscitation; trauma

Introduction

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

Rotando and colleagues[1] first used 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) describes a systematic approach to minimize haemorrhage, prevent coagulopathy and maximize tissue oxygenation, in order to optimize patient outcome in the major trauma patient. DCR incorporates the concepts of both DCS and damage control radiology (DCRad).

The concept of DCR was initially used in the military to describe an approach to managing severely injured trauma patients, and has evolved significantly in the last two decades. 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 focusing on normalizing physiology from the dual insults of injury and surgery, as opposed to providing immediate definitive repair.[2,3] DCRad incorporates diagnostic and interventional radiological solutions used to treat severely injured patients.[4]

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 trauma expertise, and provides a system for recognizing and intervening with life-threatening problems. The ATLS 'Airway, Breathing, Circulation, Disability, and Exposure' (ABCDE) mantra is familiar globally. It is likely this approach has saved many lives; however, with recent experience gained in sizeable military campaigns, and the advent of regional trauma networks, an approach that reaches beyond ATLS is now the expected standard of care in civilian practice.

DCR is a more recent evolution, enhancing major trauma resuscitation; moreover, as experience has driven innovation, DCS and DCR have evolved significantly.

Pathophysiology

Acute traumatic coagulopathy and trauma-induced coagulopathy

Haemorrhage remains the leading cause of death in both civilian and military trauma. 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 has emerged.

In 2003 Brohi and colleagues identified the existence of acute traumatic coagulopathy (ATC),[5] a complex multifactorial endogenous process occurring after severe injury in approximately a third of patients, which independently predicts death and prolonged ICU stay. Hypoperfusion and poor tissue oxygenation are thought to be key drivers, with onset commonly within 30 minutes of injury. Worsening coagulopathy may develop from enzyme and platelet dysfunction, driven by hypothermia, acidosis and serum dilution. These can be intensified during the resuscitation, particularly where high volume 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.[6] and a simplified 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% that of 37°C

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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 the bleeding. The military recognized that compressible haemorrhage from extremity wounds kills rapidly but can often be treated easily. They changed from the ABCDE approach of ATLS to <C>ABCDE, giving treatment priority to catastrophic haemorrhage (<C>). Field dressings, tourniquets and topical haemostatic agents are applied in a stepwise fashion. Severe internal bleeding must also be arrested in a timely manner, and hence early invasive interventions such as DCS and DCRad are an integral 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). Uncontrolled crystalloid infusion in trauma is associated with serious complications including acute lung injury, abdominal compartment syndrome, worsening coagulopathy and (in shocked burns patients) reduced end organ perfusion. Ley and colleagues[7] demonstrated that crystalloid infusion $\geq$1.5 L in trauma patients was an independent risk factor for mortality.

ATLS currently advocates initial administration of isotonic crystalloid up to 1000 ml (adult) or 20 ml/kg (paediatric); however, in the UK, it is now common practice to replace blood with blood products from the outset. 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), cryoprecipitate (Cryo) or fibrinogen concentrate. These are typically given in an empirical manner initially, according to local policy until a more goal directed approach can be used, usually utilizing thromboelastography (TEG or RoTEM).

Damage control resuscitation

The key tenets of DCR are controlling the bleeding (including DCS and DCRad), hypotensive or novel hybrid resuscitation, haemostatic resuscitation and massive transfusion.

Controlling the bleeding

Effective DCR starts at the point of injury. A range of interventions are utilized, from direct pressure, haemostatic agents

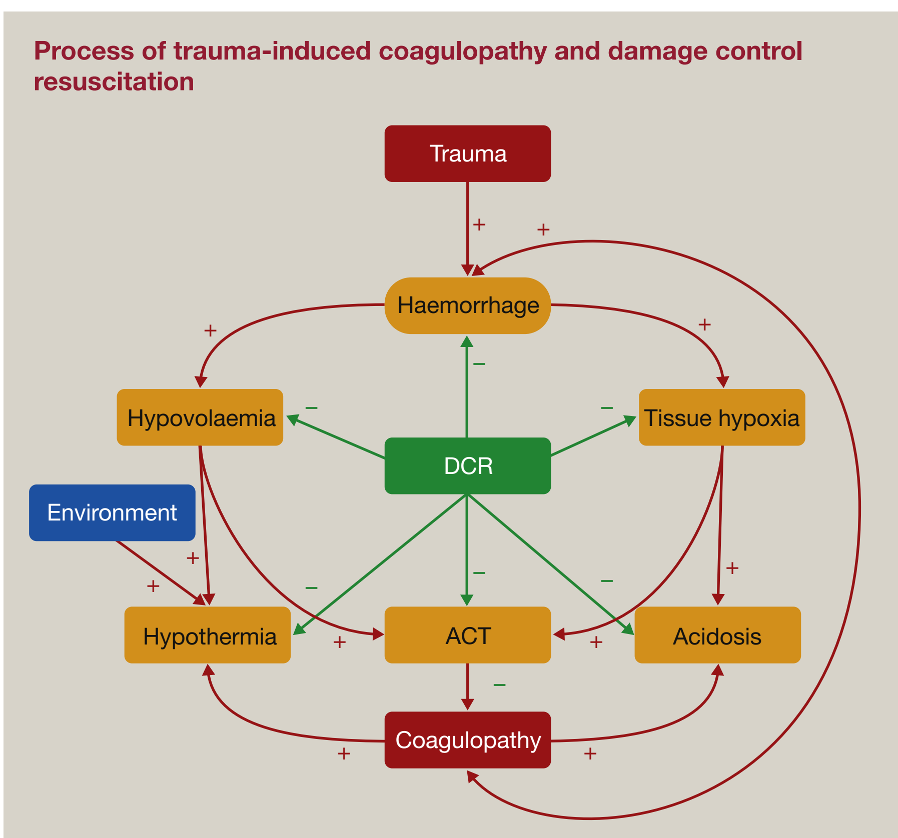

Figure 1

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