Damage Control Surgery In The Era Of Damage Control Resuscitation

M J Midwinter

Defence Professor of Surgery, Royal College of Surgeons of England and Honorary Professor, School of Medicine, University of Swansea

Introduction

Damage Control Surgery (DCS) is an operative strategy that sacrifices the completeness of the immediate surgical repair in order to address the physiological consequences of the combined trauma (double hit) of injury and surgery. In the past this has been very much focussed on abdominal trauma and the idea of performing an "abbreviated laparotomy". The concepts are however clearly applicable to injury beyond the abdomen [1-3].

Damage Control Resuscitation (DCR) is a more recent concept. It has variously been defined in the USA as *'a proactive early treatment strategy that addresses the 'lethal triad' (rapid reversal of acidosis, prevention of hypothermia and coagulopathy) on admission to a combat hospital'* [4] and in UK as *'systematic approach to major trauma combining the catastrophic bleeding, airway, breathing and circulation (<C>ABC) paradigm with a series of clinical techniques from point of wounding to definitive treatment in order to minimise blood loss, maximise tissue oxygenation and optimise outcome'* [5].

These two definitions express the DCR concept differently but consist of the same practical measures to achieve the aims of DCR. The UK definition extends the DCR principle forward to the point of wounding and is a more generalised statement. Central to both is early recognition and proactive management of the physiological consequences of the injury.

The principles of Damage Control Surgery (DCS) have been well described for over 20 years but have been slow to gain universal acceptance. However, it is now recognised that severely injured trauma patients, who are still alive at the point of medical intervention, are now more likely to die from the metabolic consequences of the injury rather than the completeness of the immediate surgical repair to their damaged organs. While there has been academic recognition of the importance of addressing resuscitation and surgical issues concurrently, it is only with the development of the concept of DCR and emerging technological useful clinical tools, that this has been consolidated into practice. It is now timely to reassess both DCR and DCS.

Pathophysiology

The central observation behind the philosophy of DCS is the adverse effects of the combined triad of hypothermia, acidosis and trauma-induced coagulopathy (TIC) (as a consequence of the hypothermia, acidosis, consumption and dilution of clotting factors) in the trauma patient [1,6,7]. Hypothermia leads to adrenergic stimulation with vasoconstriction, exacerbating any organ hypoperfusion that may be already present secondary to hypotension from the injury. This leads to worsening acidosis. This hypothermia and acidosis is then further exacerbated by aggressive fluid resuscitation, especially with normal saline (pH 5.5). The hypothermia and acidosis (combined with the consumption, dilution and failure to replace clotting factors) now promotes an established coagulopathy [8,9]. Even if major surgical bleeding is now physically controlled, the patient continues to bleed from all cut surfaces - intensifying the 'bloody vicious triad'. The degree of this coagulopathy is also known to be under-estimated by most standard laboratory tests of coagulation [10].

Hypothermia is directly correlated to injury severity and remains an independent risk factor for mortality, reaching 100% when core temperature is less than 32°C in patients undergoing a laparotomy [11,12]. Whilst the contribution of acidosis, hypothermia and the dilution / consumption of clotting factors to the development of trauma coagulopathy is important [13,14]; it has been recently recognised that in up to 30% of patients with major traumatic injuries there is an endogenous coagulopathy that is also present early. This response appears to be mediated through activation of the protein C pathway [14-17]. This acute traumatic coagulopathy (ATC) is induced by the combination of trauma, shock and tissue hypoperfusion. Patients who have evidence of ATC on admission to the emergency department are more likely to require massive blood transfusion, develop multi-organ failure and have up-to-fourfold chance of dying [15-17].

Damage Control Resuscitation

These new insights into the patho-physiology of trauma have influenced our military approach to resuscitation. In patients with injury and massive haemorrhage, massive transfusion protocols have been developed to counter the dilution and consumption of clotting factors and address the hypothermia and acidosis. This has been achieved primarily by transfusing plasma after certain number of units of packed red cells (PRBC), combined with administering fibrinogen and platelets to correct TIC.

The early presence of ATC before significant dilution or consumption of clotting factors has occurred mandates pro-active treatment. Recognition had been largely based on clinical indicators of injury severity and blood loss. However clinically based predictions perform with only about 80% sensitivity and specificity [18]. Standard laboratory tests for coagulopathy are not sensitive for detecting ATC and have a significant lag behind the clinical picture due to the time delay in their performance. Some newer protocols go with a 'one size fits all' approach. Liberal use of blood and plasma can be associated with adverse outcomes other than just those potential risks from exposure to diseases transmitted by blood products. A prospective multi-centre cohort study in trauma patients who survived their initial injury, showed that fresh frozen plasma (FFP) transfusion was independently associated with multi-organ failure (MOF) and acute respiratory distress syndrome (ARDS) [19].

Corresponding Author: Surg Capt MJ Midwinter BMedSci (Hons), DipAppStats, MD, FRCS(Eng) FRCS(Gen) RN. Defence Professor of Surgery, Royal Centre for Defence Medicine, Birmingham
Email: PS.MILSURG@rcdm.bham.ac.uk

A pilot study in combat casualties has indicated that allogeneic blood transfusion is associated with increased perioperative infection and impaired wound healing [20]. However, haemostatic resuscitation during surgery with a 1:1 ratio (or as close as possible) of FFP to PRBC in patients with TIC improves survival [15]. These studies emphasise the requirement to make a rapid diagnosis so appropriate treatment can be given to patients requiring haemostatic resuscitation and those patients not requiring such intervention are not exposed to the unnecessary risks.

Damage Control Surgery

A decision to adopt a damage control approach to surgical intervention in a trauma patient should be reached early, in order to avoid the vicious circle even being entered - rather than employing DCS as a measure of desperation once the consequences outlined above are established. Physiological measures to define when a DCS approach should be adopted have been suggested [21]:-

- **Injury severity score of more than 25,**
- **Systolic blood pressure less than 70 mmHg,**
- **Core temperature less than 34°C**
- **A pH of less than 7.1.**

Additional factors include lactate or base deficit, blood transfusion requirement, injury mechanism and anatomic complexes [22]. Rotondo & Zonies [23] have identified key factors in patient selection under the useful headings of Conditions, Complexes and Critical Factors (Box 1).

Conditions

High energy blunt trauma
Multiple torso penetration
Haemodynamic instability
Presenting coagulopathy and/or hypothermia

Complexes

Major abdominal vascular injury with multiple visceral injuries
Multifocal or multicavity exsanguinations with concomitant visceral injuries
Multiregional injury with competing priorities

Critical Factors

Severe metabolic acidosis (pH 7.30)
Hypothermia (temperature <35 C)
Resuscitation & operative time > 90 minutes
Coagulopathy as evidenced by the development of non-mechanical bleeding
Massive transfusion (>10 units of PRBC).

Box 1. Factors to consider for the Damage Control Surgical approach

DCS is only applicable to a minority of trauma patients and if used too liberally may be no better or even worse than immediate definitive surgery. However, too strict a definition as to when to adopt the approach particularly based on laboratory indicies, can mean that the adverse physiological consequences are already established. Experience and rapid surgical assessment are key to making a positive, informed decision to adopt a DCS strategy.

The Five Stages of DCS

1-Patient selection

This is coupled with decision-making and is described above. This should occur rapidly either immediately pre-operatively or within minutes of the start of surgery.

2-Intra-operative

Priorities are haemorrhage control, limiting contamination and temporary closure or cover. Haemorrhage control may be achieved by ligation, suture, tamponade (by packing or balloon), or shunting. Definitive vascular repair by grafting or anastomosis is not considered a DCS procedure. Contamination control is often achieved by tape closure of the ends of the injured hollow viscus. Anastomoses and stomas are not fashioned in DCS. Pre-emptive strategies to prevent compartment syndromes such as fasciotomies and laparostomy are employed.

Operating room time is limited to around one hour. Once haemorrhage control and contamination limitation are achieved, temporary closure or cover is established to allow the patient to be moved to critical care environment. Time in Stage 2 should as short as possible however, taking a patient with active surgical bleeding patient to the ITU is futile. Active warming is important and a theatre temperature of 26ºC is normal practice.

3-Critical Care Stage

In the critical care environment attempts continue at correcting the physiological consequences of the injury and its associated metabolic failure. Active rewarming measures with air-warming devices, fluid warmers or arteriovenous warming techniques continue. An overall warm ambient environment remains important [12]. Perfusion is restored to the body tissues. The acidosis usually corrects and the oxygen debt from the anaerobic metabolism is repaid. Coagulopathy is corrected by administration of fresh frozen plasma, cryoprecipitate and platelets as necessary [4,24]. Early return to the operating theatre is indicated if there is obvious ongoing surgical bleeding or a compartment syndrome develops.

4-Return to the operating theatre

This is dictated by improvement in the patient's physiological status. The following indices are often used to guide re-operation; base deficit greater than -4 mmol/l, lactate of less than 2.5 mmol/l, core temperature greater than 35°C and an international normalised ratio of less than 1.25.

Before the decision to return to the operating theatre is made, plans to assemble the appropriate surgical team must be put in place to ensure that the optimum repairs of the injuries are performed in the optimum surgical environment ('right patient, right time, right place, right team'). This may require more than one surgical specialty, but with a clearly identified leader to orchestrate the procedures and take a global view of the patient's condition. At this stage anastomoses are fashioned, stomas raised and vascular repairs performed.

5-Formal closure

This may not be possible at stage 4 as there may still be significant oedema or clinical risk of developing a compartment syndrome (abdominal or extremity). Therefore a planned further operative phase for closing or covering the site is made.

Developing an Integrated "DCR-DCS" Approach

With the developments of DCR, a change in approach is required to integrate the early resuscitation and surgical phases more closely. This is achievable by adopting real time, point of care technologies to aid rapid diagnosis of conditions such as ATC and to measure the effect of treatment. Thromboelastometry allows early diagnosis of coagulopathies and monitoring of therapy [10,25,26]. Other near patient tests of acid-base status and electrolyte abnormalities are also available together with other monitoring techniques such as near infra-red spectroscopy (NIRS) [27-29] to measure tissue oxygenation, allow near continuous physiological monitoring and tailoring therapeutic approaches to individual casualty responses rather than a 'one size fits all' protocol approach.

Individual tailoring of treatment options optimises the therapy for that specific casualty and minimises the potential risks of unnecessary interventions. It can also conserve resources. This "DCR-DCS" approach may lead to early correction of physiological status which in the classical DCS approach occurred in stage 3 (critical care). In time, this physiological optimisation may allow a longer DCS surgical window.

Summary

Addressing the physiological consequences of trauma, with proactive diagnostics and rapid treatment paradigms - aimed in particularly at coagulopathy, as part of a Damage Control Resuscitation (DCR) strategy from the outset of resuscitation will result in improved intra-operative physiology and survival. Early surgery is an inexorable and intimate part of that resuscitation process [4]. DCR and DCS need to occur concurrently from the outset. Many of these lessons have been already been (re-)learnt on the battlefield and have potential consequences for improvements in civilian trauma management[30] The earlier this surgery takes place the better. Any approach that minimizes any pre-theatre delay is important. The 'new' concept of direct patient admission to the operating theatre from point of wounding, bypassing the ED must now bear closer examination (Figure 1).

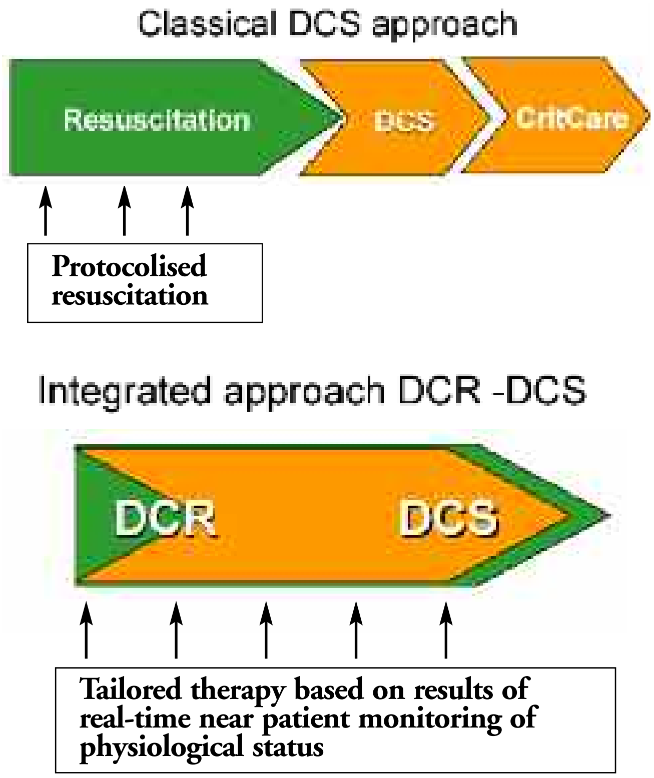

Figure 1 Schematic outlining the integration of DCS and DCR

Adopting this "DCR-DCS" approach has changed the potential surgical goals with improvement of the patient's physiological status. Recent reports of this in vascular surgery in a combat support hospital setting has allowed extended operating (median time 4.5 hours) with more definitive revascularisation to be undertaken [31]. The authors state *"combining these two concepts (DCR & DCS) may allow preservation of limbs that previously would have been amputated for fear of developing or exacerbating the lethal triad of coagulopathy, acidosis or hypothermia."* A further study has shown improved survival with early haemostatic resuscitation used intra-operatively in patients with TIC [15].

Conclusion

The development in understanding of pathophysiological consequences of injury and the development of DCR and potential techniques to aid early diagnosis and monitor effects of therapy has opened the possibility of optimising the patient's physiological status in stages 1 & 2 of the classical DCS sequence that had previously occurred in stage 3. This has led to the ability to individually tailor therapy and of extending the surgical options. In order to achieve the goal of optimising outcome DCR and DCS should be considered a single concept (DCR-DCS) and requires a fully integrated cross-disciplinary trauma team with joint training and understanding in this approach. This is the objective of the newly established Military Operational Surgical Training (MOST) course at the Royal College of Surgeons of England.

References

1. Loveland J, Boffard K. Damage control in the abdomen and beyond. Brit J Surg 2004; **91:** 1095-1101.
2. Rezende-Neto J, Marques A, Guedes LJ, Teixeira LC. Damage control principles applied to penetrating neck and mandibular injuries. J Trauma 2008; **64**(1142-1143).
3. Wall MJ, Soltero E. Damage control for thoracic injuries. Surg Clin N Am 1977; **77(4):** 863-877.
4. Holcomb J, Jenkins D, Rhee P et al. Damage Control Resuscitation: Directly Addressing the Early Coagulopathy of Trauma. J Trauma 2007; **62:** 307-310.
5. Hodgetts TJ, Mahoney PF, Kirkman E. Damage Control Resuscitation. Journal of the Royal Army Medical Corps; **153(4):** 299-300.
6. Rotondo M, Schwab CW, McGonigal MD et al. 'Damage Control': an approach for improved survival in exanguinating penetrating abdominal injury. J Trauma 1993; **35**(3): 375-383.
7. Burch JM, Oritz VB, Richardson RJ. Abbreviated Laparotomy and Planned Reoperation for Critically Injured Patients. Ann Surg 1992; **215:** 476-484.
8. Gregory JS, Flancbaum L, Townsend MC, Clouther CT, Jonasson O. Incidence and timing of hypothermia in trauma patients undergoing operations. The Journal of Trauma Injury, Infection and Critical Care 1991;**31**(6): 795- 800.
9. Steinemann S, Shackford SR, Davis JW. Implications of admission hypothermia in trauma patients. The Journal of Trauma Injury, Infection and Critical Care 1990;**30**(2): 200-202.
10. Kheirabadi B, Crissey J, Deguzman R, Holcomb J. In vivo bleeding time and in vitro thromboelastography measurements are better indicators of dilutional hypothermic coagulopathy than prothrombin time. J Trauma 2007; **62**(6): 1352-1361.
11. Jurkovich GJ, WB G, Luterman A, Curerri P. Hypothermia in trauma victims: an ominous predictor of survival. J Trauma 1987;**27:** 1019-1024.
12. Morris Jr J, Eddy V, Blinman T, Rutherford E, Sharp K. The staged celiotomy for trauma: issues in packing and reconstruction. . Ann Surg 1993; **217:** 576-586.
13. Martini WZ, Pusateri AE, Uscilowicz JM, Delgado AV, Holcomb JB. Independent contributions of hypothermia and acidosis to coagulopathy in swine. J Trauma 2005; **58**(5): 1002-1009.
14. Brohi K. Diagnosis and management of coagulopathy after major trauma. Brit J Surg 2009; **96**(9): 963-964.
15. Duchesne JC, Islam TM, Stuke L et al. Hemostatic resuscitation during surgery improves survival in patients with traumatic-induced coagulopathy. J Trauma 2009; **67**(1): 33-39.
16. Brohi K, Cohen M, Ganter MT, Matthay MA, Mackersie RC, Pittet JF. Acute traumatic coagulopathy: initiated by hypoperfusion: modulated through the protein C pathway? Ann Surg 2007; **245**(5): 812-818.
17. Brohi K, Cohen M, Davenport R. Acute coagulopathy of trauma: mechanism, identification and effect. Curr Opin Crit Care 2007; **13**(6): 680-685.
18. Nunez TC, Voskrenensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA. Early prediction of massive transfusion in trauma: simple as ABC (assessment of blood consumption)? J Trauma 2009; **66:** 346-352.
19. Watson GA, Sperry JL, Rosengart MR et al. Fresh frozen plasma is independently associated with a higher risk of multiple organ failure and acute respiratory distress syndrome. J Trauma 2009; **67**(2): 221-230.

20. Dunne JR, Hawksworth JS, Stojadinovic A et al. Perioperative blood transfusion in combat casualties: A pilot study. J Trauma 2009; **66:** S150-S156.
21. Moore EE, Burch JM, Franciose R, Offner P, Biffl W. Staged physiologic restoration and damage control surgery. World J Surg 1998; **22**(12): 1184-90.
22. Talbert S, Trooskin S, Scalea T, al. e. Packing and re-exploration for patients with non-hepatic injuries. The Journal of Trauma Injury, Infection and Critical Care 1992;**33:** 121-125.
23. Rotondo M, Zonies D. The damage control sequence and underlying logic. Surg Clin N Am 1997; **77:** 761-777.
24. Kirkman E, Watts S, Hodgetts T, Mahoney P, Rawlinson S, Midwinter M. A proactive approach to the coagulopathy of trauma: the rational and guidelines for treatment. J R Army Med Corps 2008; **153**(4): 302-306.
25. Rugeri L, Levrat A, David J et al. Diagnosis of early coagulation abnormalities in trauma patients by rotation thromboelastography. J Thromb Haemostasis 2006; **5:** 289-295.
26. Bilkovski R, Rivers E, Horst H. Targeted resuscitation strategies after injury. Curr Opin Crit Care 2004; **10**(6): 529-538.
27. Taylor JH, Mulier KE, Myers DE, Beilman GJ. Use of near-infrared spectroscopy in early determination of irreversible hemorrhagic shock. J Trauma 2005; **58**(6): 1119-1125.
28. Cohn SM, Nathens AB, Moore FA et al. Tissue oxygen saturation predicts the development of organ dysfunction during traumatic shock resuscitation. J Trauma 2007; **62**(1): 44-55.
29. Chaisson NF, Kirschner RA, Deyo DJ, Lopez JA, Prough DS, Kramer GC. Near-infared spectroscopy-guided closed-loop resuscitation of haemorrhage. J Trauma 2003; **54:** S183-S192.
30. Moor P, Rew D, Midwinter MJ, Doughty H. Transfusion for trauma: civilian lessons from the battlefield. Anaesthesia 2009; **64:** 469-472.
31. Fox CJ, Gillespie DL, Cox ED, Kragh JF Jr, Mehta SG, Salinas JBHJ. Damage control resuscitation for vascular surgery in a combat support hospital. J Trauma 2008; **65**(6): 1-9.