Trauma resuscitation and the damage control approach

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Abstract

Trauma is a leading cause of morbidity and mortality worldwide. Developments in trauma care, from point of wounding to rehabilitation, have often been led and driven by military medical experiences in recent conflicts. Trauma mechanisms are manifold, affecting an omnifarious patient population indiscriminately, and potentially resulting in significant multisystem dysfunction or damage, sometimes permanently. The horizontal approach to trauma resuscitation, whereby a patient is assessed and treated by multiple specialists to prioritize management of life-threatening issues swiftly and concurrently, was exploited to good effect by clinicians at Camp Bastion in Afghanistan. This tactic is just one element of the dynamic and synchronous teamwork that this complex and challenging area of clinical practice demands. Similarly, the damage control approach deploys medical and surgical treatment strategies in parallel, balancing therapies in pursuit of physiological equipoise, aiming to reduce mortality and accepting the risk of morbidity. Damage control therapy embodies bold, yet nuanced, clinical care.

Keywords Catastrophic haemorrhage; coagulopathy; damage control; human factors; trauma

Damage control

Much of the current understanding of successful trauma care, as attested by the number of unexpected survivors, has come from recent UK military medical experiences in Iraq and Afghanistan. This corporate knowledge has been used to shape civilian approaches to trauma care. The damage control paradigm encompasses the concepts of damage control resuscitation (DCR) and damage control surgery (DCS). Together, these comprise processes and procedures that focus on temporary prioritization of physiological stabilization over definitive anatomical repair of the trauma patient, allowing only necessary surgical interventions to be performed (for control of haemorrhage and minimization of contamination) alongside concurrent, early, and aggressive correction of the components of the so-called lethal triad (coagulopathy, hypothermia and acidosis). Damage control interventional radiology (DCIR) is a useful adjunct to these endeavours. The aim is to establish sufficiently robust patient physiology in order for them to withstand further surgery, potentially involving multiple procedures, and to facilitate overall patient survival. This practice is not without challenge, both logistical and moral, as any reduction in expected mortality often coexists with increased morbidity and disability.

Exsanguination from catastrophic haemorrhage is the leading cause of preventable death in trauma.[1] The majority of external or extremity bleeding can be controlled initially by methods such as compression, elevation, splinting, and application of haemostatic agents, and it is feasible for these treatments to be initiated in the pre-hospital setting. Patients with suspected internal, non-compressible, haemorrhage principally require rapid transfer to hospital for surgical intervention. The specifics of pre-hospital management for these patients will be determined by the kit and skill-set of the clinicians in attendance, with many physician-led enhanced pre-hospital care teams in the UK now carrying blood products. The CRASH-2 (Clinical Randomization of an Antifibrinolytic in Significant Haemorrhage 2) trial showed that intravenous administration of tranexamic acid (TXA) is beneficial when given within 3 hours of injury occurring, and this antifibrinolytic agent is administered routinely to trauma patients by NHS paramedics.

Systematic assessment, taking into account the mechanism of injury as well as physical findings and patient observations, facilitates estimation of the degree of blood loss. In addition to obvious external blood loss, the mnemonic 'blood on the floor and four more' prompts consideration of potential internal sources of haemorrhage, secondary to intra-thoracic and intra-abdominal haemorrhage, and long bone and pelvic fractures. Where there is a requirement for surgical intervention, patient stability will determine the timeframe that is available for pre-operative investigations, including imaging, to aid clinical judgement and decision making. A judicious balance must be struck between medical analysis and therapeutic momentum.

Damage control strategies, whereby surgery and resuscitation are delivered in tandem, aim to achieve safe and timely progression of trauma haemorrhage treatment. Crudely put, in the context of catastrophic haemorrhage the priority of the former is to 'turn off the tap', while the goal of the latter is to replace what has been lost, and to address issues that have arisen secondary to that loss.

Trauma-induced coagulopathy

Tissue injury and shock cause a multidimensional disorder of haemostasis, termed trauma-induced coagulopathy (TIC). TIC involves disturbance to the vascular endothelium, immune system, and inflammatory processes.[2] It perpetuates haemorrhage at the molecular level, with issues caused at every juncture of the coagulation pathway, resulting in decreased clot formation and increased fibrinolysis. Despite significant advances having been made in investigating the pathophysiology of TIC, and in managing patients with this condition, there is still a lack of evidence regarding the precise nature of these mechanisms *in vivo*.

Trauma causes multi-system failure, to the potential detriment of all organs and tissues, of which blood could be considered to be one. White et al. regard the blood–endothelial unit as an organ system that connects all other organ systems, and describe traumatic blood failure, whereby injury and blood loss result in a cascade of events that culminate in blood dysfunction.[3] This issue centres largely on the impact of trauma on the vascular

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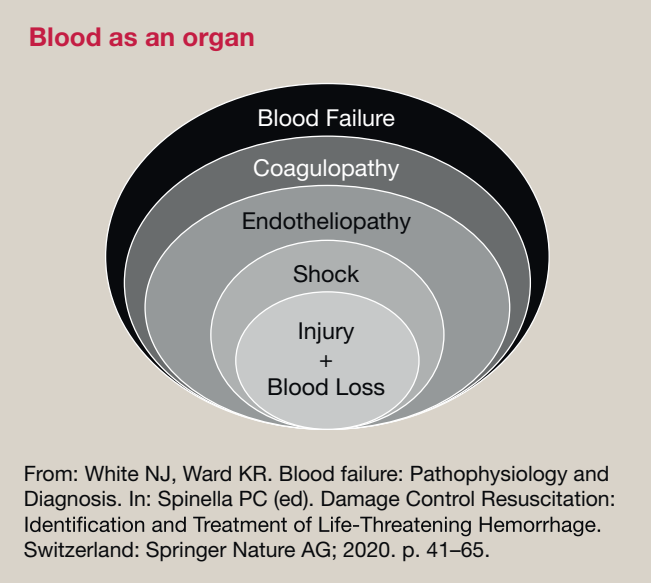
endothelium, which normally operates as a blood–organ barrier and is integral to haemostasis, with both procoagulant and anticoagulant functions. The endothelium is not only subject to direct tissue injury in trauma but is also susceptible to the sequelae of haemorrhage (hypoperfusion, hypoxia), and any subsequent restoration of blood flow (reperfusion injury). The resultant endotheliopathy, caused by endothelial cell and glycocalyx damage, is characterized by dysregulation of paracellular permeability, defective haemostasis, and uncontrolled inflammation.[3] The ramifications of traumatic haemorrhage on blood are therefore both quantitative and qualitative.

In terms of TIC, the problems are manifold, and the loss of clotting components secondary to catastrophic haemorrhage is just the tip of the iceberg. Physical depletion of coagulation factors is exacerbated by the consumptive coagulopathy that follows trauma, along with functional defects that appear to develop in clotting constituents that are produced subsequently, and by the impact of trauma-associated acidosis and hypothermia. The dilutional effect of TIC, owing to interstitial fluid shifting into the vascular space, may be aggravated further by an iatrogenic contribution to coagulopathy caused by inappropriate crystalloid fluid resuscitation. A combination of endothelial dysfunction and deranged physiology results, in what has been described as a downward spiral of blood failure, with a loss of haemostatic homeostasis (Figure 1).[4]

TIC comprises multiple cellular responses to tissue injury and shock. One of the chief mechanisms of TIC is thrombin-induced activation of protein C, which is a serine protease produced by the liver and which, when activated, has a regulatory role in anticoagulation, inflammation and cell death. Activated protein C (APC) inhibits activation of the coagulation cascade, by proteolytic degradation of factor Va and factor VIIIa. It also simultaneously stimulates fibrinolysis, by increasing plasmin levels via inactivation of plasminogen activator inhibitors, and by decreasing the formation of thrombin. In addition to APC, tissue plasminogen activator (tPA) activates fibrinolysis by catalysing the conversion of plasminogen to plasmin, which is the enzyme responsible for fibrin clot degradation. However, APC does have anti-inflammatory and cytoprotective properties that would potentially be of benefit to a trauma patient, and even its anticoagulant effect would be advantageous during the hypercoagulable phase of trauma, which occurs within a number of hours post-injury. It has been conjectured that harnessing the desirable characteristics of APC, by means of newly engineered forms, would enable inhibition of its harmful anticoagulant function, with concurrent exploitation of its cytoprotective function, in the immediate aftermath of trauma.[2]

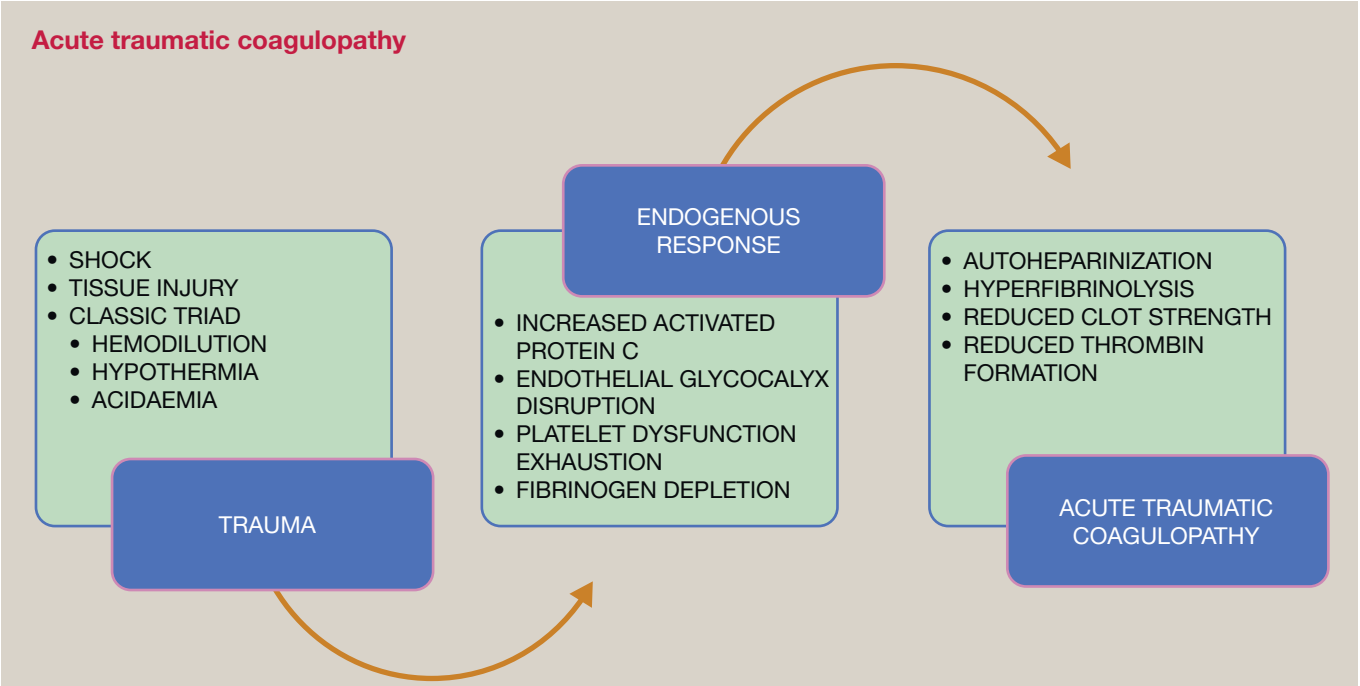


From: White NJ, Ward KR. Blood failure: Pathophysiology and Diagnosis. In: Spinella PC (ed). Damage Control Resuscitation: Identification and Treatment of Life-Threatening Hemorrhage. Switzerland: Springer Nature AG; 2020. p. 41–65.

Figure 1

The dysregulation of blood components and coagulation processes secondary to trauma is endemic, with both the quantity and quality of constituents contributing to the resultant blood failure.[3] Thus, thrombocytopenia and hypofibrinogenaemia are exacerbated by underperformance of the platelets and fibrinogen that are actually available. In addition to reducing the formation of stable clots, injury-induced alteration in the biology and function of such clotting components is associated with increased morbidity and mortality.[2] However, even replacement of platelets and fibrinogen, included as part of major haemorrhage treatment protocols, may not in itself be completely effective or straightforward. Studies have shown that platelet transfusion fails to reverse post-injury impairment in platelet aggregation, and goal-directed fibrinogen replacement still lacks evidence-based specificity.[2] Nevertheless, haemostatic resuscitation with blood products is a core element of DCR, transfusing a number of components in order to attempt to restore full blood function.

Many coagulopathic changes occur early after trauma and, even by the time damage control measures are undertaken, the coagulation abilities of the severely injured patient are already compromised (Figure 2). Hypothermia (temperature $<35°C$) and acidosis ($pH < 7.35$), along with coagulopathy, complete the lethal triad of trauma, and also worsen TIC. After a trauma insult, normal central thermoregulation may be impaired by a multitude of mechanisms, such as haemorrhagic shock, traumatic brain injury (TBI), and alcohol intoxication. Other contributing elements to hypothermia include trauma involving burn injury and lengthy environmental exposure, for example owing to prolonged patient extrication. Patient risk factors, such as extremes of age or certain comorbidities, also play a part. Administration of cold resuscitation fluids may aggravate the situation further. Hypothermia has been shown to result in haemoconcentration, leukopenia, thrombocytopenia, disordered fibrinolysis, and impaired coagulation enzyme and platelet function. Furthermore, hypothermia can exacerbate acidosis via the metabolic effects of shivering, respiratory depression, and hypoglycaemia. The principal cause of acidosis in trauma is poor tissue perfusion, resulting from blood loss and haemorrhage-induced and hypothermia-induced vasoconstriction, so that hypoxic cells shift to anaerobic metabolism and produce lactic acid. The situation may be exacerbated by the metabolic sequelae of hypothermia already mentioned, as well as hyperchloraemic acidosis if unbalanced crystalloid solutions are administered during resuscitation. Acidosis leads to a reduction in coagulation factor V, VIIa, and X activity, and inhibition of thrombin generation. The lethal triad demonstrates harmful synergy, whereby each of its elements both contribute to, and are intensified by, one another. Furthermore, there are myriad other multi-organ and multi-system complications that characterize the impact of the lethal triad in trauma.



From: Simmons JW, Powell MF. Acute traumatic coagulopathy: pathophysiology and resuscitation. *Br J Anaesth* 2016; 117: iii31–43.

Figure 2

Problems with coagulation in trauma are not limited to issues with impaired clot formation. Fibrinolytic activities at pathological extremes of the spectrum, whether excessive fibrinolysis or fibrinolysis shutdown, increase the risk of mortality.[2] Severe hypothermia can trigger a hypercoagulable state, secondary to activation of platelets in the splenic pool. Under certain circumstances, treatment with antifibrinolytic agents, such as TXA, may also contribute towards a pro-thrombotic state. Consequently, further issues may arise in the aftermath of TIC, when coagulopathic processes have ceased and the patient is more at risk of thromboembolic complications. In addition, downstream end-organ effects of widespread immune system activation pose further coagulatory and other physiological challenges for the trauma patient. Damage control treatment strategies provide counterpoise to the multisystem disorder they are used to address, while mirroring the complex cascade of trauma sequelae in terms of comprising intertwining elements, which in the case of damage control are multidisciplinary skills, both technical and non-technical.

Damage control resuscitation (DCR)

DCR is synonymous with the concept of balanced resuscitation, and consists of permissive hypotension and haemostatic resuscitation, with the aim of establishing survivable patient physiology before definitive surgical or radiological intervention and further aggressive resuscitation are undertaken. Permissive hypotension involves maintaining a blood pressure that is low enough to reduce active haemorrhage and clot disruption, prior to surgical haemostasis being achieved, but high enough to preserve end-organ perfusion. The use of permissive hypotension is a balancing act, with haemorrhage and under-perfusion the undesirable corollaries on either side that both result in significant physiological insult. The depth and duration of permissive hypotension must therefore be regulated. A target systolic blood pressure (SBP) threshold is raised for patients with suspected TBI, to maintain cerebral perfusion pressure. Despite a growing body of research, there is no hard evidence on which to base a specific SBP goal. It has been suggested that the currently accepted target SBP of 90 mmHg for non-TBI patients is too low, and that a target of 100 mmHg, with an upper limit of 110 mmHg, might be more appropriate.[5] A novel hybrid resuscitation strategy proposes limiting the duration of permissive hypotension to 60 minutes, to mitigate poor oxygen delivery and metabolic acidosis, before normotensive resuscitation is instigated.[6]

As noted previously, factor depletion and other alterations to coagulation occur independently of fluid resuscitation, although the latter may compound these issues if not undertaken appropriately. In trauma, NICE recommends titration of volume resuscitation to achieve a palpable central pulse (with a less restrictive approach for suspected TBI), until definitive haemorrhage control is accomplished, using blood products in preference to any other fluid.[7] Many pre-hospital services in the UK now carry red cells, replacement of which will improve the oxygen-carrying capacity of the blood, and other blood products, which can be transfused using portable blood-warming devices. Haemostatic resuscitation advocates early use of blood products[7,8] in ratios similar to whole blood: red blood cells, plasma and platelets administered 1:1:1. The aim of this balanced strategy is to address blood failure by restoring oxygen delivery, haemostatic function, and endothelial function,[4] as well as to avoid complications associated with crystalloids,

including dilution of red cell and coagulation factor concentrations, and exacerbation of acidosis, hypothermia, oedema, and immune system activation causing cellular injury. In view of the requirement to replace all blood components in trauma, the use of fresh whole blood has been mooted as a superior option to component therapy.[4] The Study of Whole Blood In Frontline Trauma (SWIFT) trial, currently ongoing, aims to determine whether pre-hospital transfusion of leukocyte-depleted whole blood is more effective than standard component therapy in reducing mortality or massive transfusion requirements at 24 hours post-injury. Nevertheless, transfusion of any blood product is not without risk, and must be undertaken judiciously and in response to clinical findings. In addition to monitoring haematological and coagulation markers and cardiovascular status, restoration of adequate perfusion can be assessed at the microcirculatory level by measuring serum lactate and base deficit.[9] Anaesthetic techniques can be utilized to good effect, as vasodilatation secondary to anaesthetic agents and opioids will facilitate maintenance of low systemic vascular resistance and high cardiac output, thereby maximizing oxygen delivery to the tissues.[5] Anaesthetic and critical care expertise is also used for intubation and ventilation to optimize oxygenation and mitigate acidosis, as well as siting of appropriate intravenous access and invasive monitoring and overall interpretation and manipulation of patient physiology.

DCR is about aiding blood recovery, using a holistic approach to replace the functionality of whole blood.[4] The volume and timing of product transfusion will initially be ratio based and protocol driven, but can become more nuanced and targeted based on patient physiological response and, more accurately, the use of laboratory evidence. TIC is multifaceted, so it is unsurprising that no single test alone completely elucidates the nature of the haematological dysfunction at play. Even the optimal combination of investigations required to measure the complex and interlinked pathways of blood failure, in terms of coagulation, vascular homeostasis and inflammation, is undetermined.[2] A European guideline on management of major bleeding and coagulopathy following trauma makes a number of recommendations, including using a combination of laboratory tests for early and repeated monitoring of haemostasis.[10] Point of care coagulation testing (POCCT), using a viscoelastic haemostatic assay (VHA), is particularly beneficial once bleeding has been controlled, for tailoring the resuscitation strategy in a timely manner. This mitigates transfusion risks, by limiting patient exposure to blood components through more focused selection and scheduling of resuscitation fluids, and also rationalizes blood product supplies in a resource-limited or mass casualty setting. In a dynamic DCR situation it is vital that there is a rapid and robust means of assessing ongoing blood product requirements, and the effectiveness of those that have already been given, thereby facilitating evidence-based transfusion. Some studies have concluded that early use of a VHA has been shown to identify patients requiring a massive transfusion, and others that use of POCCT has reduced the volume of blood products being transfused in high-risk surgeries.[11]

VHAs generate the continuous measurement and display of the viscoelastic properties of a whole blood sample, from the initial phase of fibrin formation and clot generation through to clot breakdown and fibrinolysis. VHAs comprise thromboelastography (TEG) and rotational thromboelastometry (ROTEM), which work slightly differently but are based on the same principles. Both feature a pin immersed in a cup of whole blood, and either the cup (TEG) or pin (ROTEM) rotates, so that fibrin strands from clot formation increase torque between the pin and the cup, and the changes in torque are detected by an electromechanical transducer with a processor that creates contemporaneous graphical and numerical output.[11] Using a whole blood sample allows analysis of the interaction between red cells, plasma clotting factors and platelets, during all the phases of coagulation (initiation, amplification, propagation, and lysis), resulting in a real-time visual display of clot evolution at the point of care. Although this is arguably superior to conventional laboratory coagulation testing, which monitors activated partial thromboplastin time (APTT), prothrombin time (PT), and the international normalized ratio (INR), there are some disadvantages to VHAs. Coagulation processes are being measured under artificial conditions in a cuvette, rather than in blood flowing within an endothelialized vessel, and rigorous quality assurance standards are more difficult to institute outside a laboratory. Furthermore, the use of VHAs requires training of non-laboratory staff, and there may be other additional costs involved. Several comparative studies demonstrate that clot formation results are not completely interchangeable between TEG and ROTEM. VHA results must be used to trigger, rather than target, treatment, as even with POCCT there will be some lag time between test turnaround and current patient physiology.[11]

Whichever combination of coagulation studies is used, there are four key components to treatment targeting coagulopathy in trauma — fresh frozen plasma (FFP) or factor concentrates, cryoprecipitate or fibrinogen concentrate, platelets, and TXA. There is a need for further evidence on many coagulation concentrates, and how they should best be utilized. As previously explained, transfusion ratios of red cells, FFP, and platelets are initially protocolized in DCR, then finessed as clinical and laboratory findings allow. Fibrinogen is the final crucial constituent in the clotting cascade and is converted by thrombin to fibrin, the strands of which enmesh red cells and platelets in order to form blood clots. Hypofibrinogenaemia is common in trauma, and is associated with massive haemorrhage, higher transfusion requirements, and increased mortality. Early fibrinogen replacement has been shown to increase survival, and current guidelines advise that fibrinogen levels should be maintained above 1.5 g/L.[12] Ionized calcium is also essential for fibrin polymerization, as well as platelet activity. The level of calcium, which will already be depleted in a patient who is bleeding, due to consumption as a co-factor in the clotting cascade, may fall further during massive transfusion, owing to binding with the citrate in stored red blood cell units. Hypocalcaemia should be treated in the coagulopathic trauma patient.

Fibrinolysis, which results in clot breakdown and is stimulated as part of TIC, is another potential target for addressing coagulopathy during DCR. The CRASH-2 trial demonstrated that intravenous administration of the antifibrinolytic agent TXA is beneficial when given within 3 hours of injury occurring. Similarly, the CRASH-3 (Clinical Randomization of an Antifibrinolytic in Significant Head Injury 3) trial showed that intravenous TXA treatment within 3 hours of injury reduces head injury-related death.

Hypothermia causes an array of complications in the bleeding trauma patient and should be addressed as part of DCR. Passive

rewarming, by covering the patient and increasing the ambient temperature, can be augmented significantly by means of active warming techniques, such as warm intravenous fluid administration, application of forced air warming heating blankets, and, in more severe cases, body cavity lavage.

DCR can expedite surgical control of bleeding, and improve patient outcome significantly, although it is no substitute for definitive haemostasis. Indeed, according to the damage control paradigm, DCS could be considered an element of DCR, and vice versa, as effective DCR may reduce the need for DCS if an improvement in their physiological condition renders a patient better able to tolerate definitive surgery.[13] Just as no single treatment is a panacea for the physical, physiological and functional consequences of trauma, so DCR is one link in the trauma chain of survival.

Damage control surgery (DCS)

DCR and DCS have been associated with improved outcomes for the severely injured trauma patient.[13] DCS limits the scope of the initial operation to control of haemorrhage and minimization of contamination, rather than definitive injury repair, so that there is opportunity for the full gamut of damage control strategies collectively to stabilize the patient, and increase the chances of survival. The effectiveness of DCR would be, at best, diminished without DCS, as both mechanical haemorrhage control and haemostatic resuscitation are necessary for DCR.[4]

One of the goals of damage control is to limit the time for which the patient is exposed to significant interventions, such as general anaesthesia, that will challenge further their already compromised physiology. An intentionally abbreviated laparotomy technique, in the context of trauma patients who developed intraoperative coagulopathy, was first described over 40 years ago. The effectiveness of DCS is also contingent on it being applied to the right patients, and promptly. It should be employed for those whose physiological vulnerability would compromise the success of more extensive initial surgical intervention, and outcome overall (estimated to be around 10% of major trauma patients), but not performed on patients who are able to withstand definitive management at an earlier stage, and whose risk of morbidity and mortality would potentially be increased by non-essential multiple procedures.[13] If not identified preoperatively, indications to change to DCS are largely based on physiological derangement, including major haemorrhage (>10 units red blood cells transfused), severe metabolic acidosis (pH < 7.3), lactate >5 mmol/L, hypothermia (temperature <35 C), coagulopathy (demonstrated on laboratory tests, or judged clinically), or operative time >90 minutes.[13] Meaningful recognition of the requirement for damage control, and its instigation, is dependent on robust communication and teamwork within the multidisciplinary team.

The DCS patient is placed in a so-called cruciform position on the operating table, with both arms out at right angles to the table, and is prepped from chin to mid-thighs, to maximize exposure in anticipation of the need for access to more than one body cavity. Similarly, the equipment available consists of laparotomy, vascular, and chest instruments. Cell salvage equipment should be organized, to facilitate autologous blood transfusion. A vertical midline laparotomy incision allows good access to the abdominopelvic region, and can also be extended to expose the chest and upper abdomen if required.[13] The first priority is haemorrhage control, involving removal of large clots and four quadrant abdominal packing, while injuries are located and assessed. Similar principles apply in cases of chest trauma, where DCS consists of an abbreviated thoracotomy. If aortic compression is needed to control significant arterial bleeding, a clamp should be applied as distally as possible and for as little time as possible, to reduce the risk of visceral ischaemia. Once catastrophic haemorrhage has been managed, packs can be removed sequentially so that damage control surgical procedures can be performed under more controlled conditions.[13]

Repair of vascular and visceral injuries is undertaken to preserve life and limb during DCS. Ligation or shunting are surgical options for the former, and interventional radiology may also be utilized for embolization or stent grafting when surgical techniques fail to halt bleeding. Depending on the organ and the injury, surgical management may utilize methods to control bleeding, such as suture repair, Foley catheter placement, resection, haemostatic agent application, or packing. Specific temporizing procedures, such as pulmonary hilum twists for the lungs or the Pringle manoeuvre for the liver, may be performed in order to prevent exsanguination while DCR is ongoing and surgical plans are made. The priority after haemorrhage control during a damage control laparotomy is limitation of contamination. Minimal bowel perforations may be sutured, but more extensive bowel injury will require resection and leaving the bowel in discontinuity. Undertaking anastomosis or stoma formation is not appropriate in the damage control setting, not least because bowel injury may continue to evolve and result in the requirement for further resection during subsequent operations. Bladder injuries may be sutured, or packed, and the bladder catheter-drained. Once vascular and visceral injuries have been managed sufficiently, packing and temporary abdominal closure is achieved, balancing the need to maintain application of pressure to tamponade bleeding while allowing some leeway for inevitable postoperative bowel oedema. As with temporary abdominal closure, temporary chest closure for a damage control thoracotomy allows surgery to be paused, so that patient physiology can be stabilized further in the critical care environment, prior to planned re-exploration and non-damage control surgery taking place. The timing of subsequent surgery can be decided by the multidisciplinary team, guided by patient physiology.

Significant trauma to a number of body regions will require DCS input from multiple surgical teams. Orthopaedic procedures, for example fixation of pelvic or long bone injuries, and neurosurgical procedures, such as a decompressive craniectomy, may need to be performed at this stage. Decisions regarding injury prioritization and sequencing of procedures can be complex and challenging and are best managed by using a multidisciplinary approach.

Damage control interventional radiology (DCIR)

Radiology, both diagnostic and therapeutic, is another important tool in the damage control setting. In this context, radiology is used to identify life-threatening injuries including bleeding sites, ascertain head or spinal injury, and triage patients to either the operating theatre for DCS or the angiography suite for endovascular haemorrhage control.[14] Judicious use of diagnostic radiology

is essential during the management of significantly injured patients. Knowing when, and how, to use this resource is a skill in itself. The mechanism of injury, anatomical injury extent and pattern, and patient physiology are used to determine the requirement for, and coverage of, any scan. It may be the case that a patient is perceived to be too unstable to tolerate time in the CT scanner, and that this should be bypassed in preference for heading straight to the operating theatre. However, aggressive DCR usually stabilizes the patient sufficiently for them to undergo a CT scan, which can provide very useful information to guide surgical prioritization and planning. Modern UK scanners use multidetector computed tomography (MDCT). MDCT has been assessed to be more comprehensive, sensitive, and specific than clinical examination, plain films, or ultrasound for injury identification.[14] It also takes less than 90 seconds to perform, and the information that it provides is likely to benefit most the unstable, severely injured patient, by directing treatment to any life-threatening injuries.[14] These should be identified by a non-contrast head scan to assess for intracranial haemorrhage, followed by a dual-phase contrast injection for the neck and trunk, with the same scan extended to the lower legs to provide angiographic imaging if there is lower limb injury.[14] However, scanning should take place as soon as is safely possible, with DCR ongoing. This timeline should, in theory, be compressed if the scanner is situated close to the Emergency Department trauma bay. While initial trauma team plans will be made based on a preliminary verbal radiology report that highlights any life-threatening findings, NICE recommends that in trauma there should be a provisional written radiology report within 60 minutes of the scan.[7]

In addition to diagnostic radiology, there may be a requirement for therapeutic radiology, DCIR, as a means of achieving haemostasis in the bleeding trauma patient. DCIR includes temporary balloon arterial occlusion, embolization to occlude arteries, and stent grafting to repair injured vessels.[14] DCIR and DCS are complementary interventions, with one, or both, used to control haemorrhage, depending on the site of bleeding, patient condition, and anticipated timeframes factored into the treatment plan. As with any intervention, DCIR is not without risk. Successful DCIR, like DCS, is dependent on effective DCR. This emphasizes further the importance of multidisciplinary collaboration in the damage control setting.

Damage control human factors

Human factors illuminate the relationship between humans and the systems in and with which they interact. They are encapsulated in ergonomics and non-technical skills, both of which, if executed well, can make it easier to work in the right way. Understanding human factors and their application is crucial for developing and improving performance in the workplace, including in the context of trauma care. Several frameworks exist for non-technical skills for a number of the professional groups that work together in trauma.

Teamwork is central to these non-technical skills, highlighting the multidisciplinary aspect of trauma care (Figure 3). Each team member has specific responsibilities, but also a common purpose. For example, during the initial phase of trauma treatment, the anaesthetist will determine the timing and conduct of a rapid

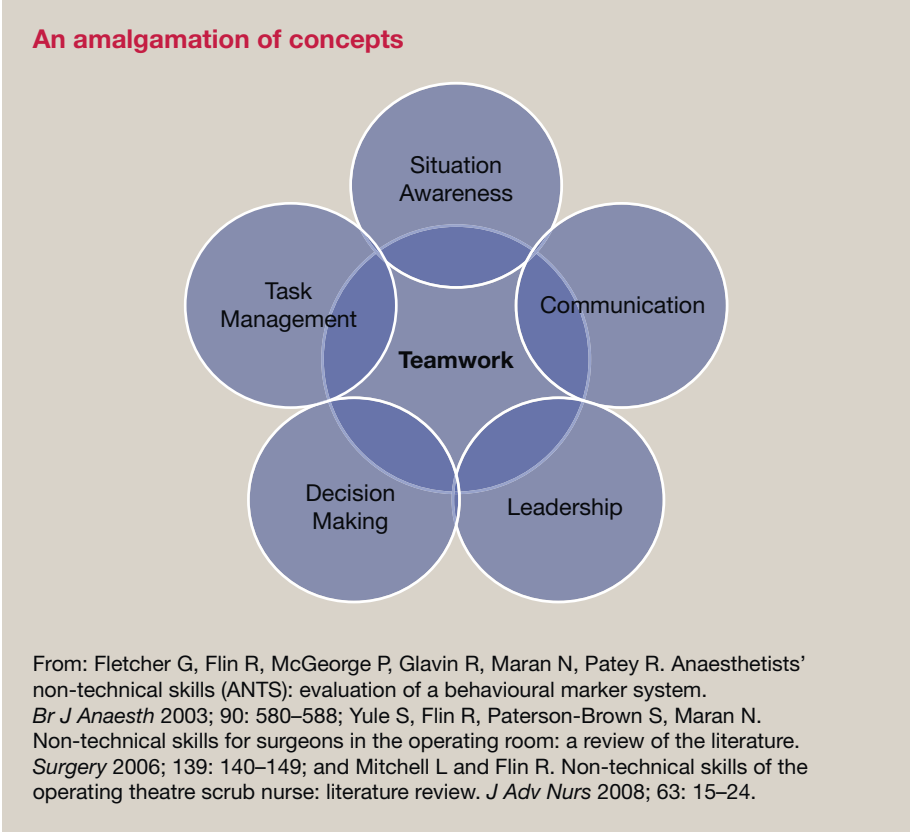


Figure 3

sequence induction, for establishment of a definitive airway and mechanical ventilation, as well as placement of large bore venous access for haemostatic resuscitation, and also institution of invasive monitoring. However, these interventions need to be performed with an appreciation of what else is happening at the time, and also to facilitate vital tasks being carried out by the rest of the team. Good leadership within the trauma team will encourage good followership, and there is a need to nurture expertise, but also flatten hierarchies, so that the most junior members of the team feel empowered to speak out when there is a problem. The leadership role in trauma is dynamic, passing between specialties depending on the location and management priorities at the time. Effective communication between members of the team should support this, and aid sound decision-making. Military trauma team nomenclature, some of which has emerged in the civilian setting, exemplifies expedient communication, and emphasizes clinical priorities in damage control circumstances. Examples include the 'snap brief' (a streamlined version of the WHO Surgical Safety Checklist) and 'STACK brief' (S – SBP, T – Temperature, A – Acidosis, C – Coagulopathy, K – Kit/Surgical plan), which can be used at appropriate time intervals intra-operatively, as part of a 'SITREP' (situation report) that allows anaesthetists and surgeons to apprise themselves of patient stability, and to plan further treatment accordingly. These tools support timely decision-making, such as agreeing an appropriate endpoint to the current procedure, based on operative time and patient condition.

Robust non-technical skills, just like damage control strategies, should be applied throughout the trauma patient's journey. The damage control paradigm is exhibited longitudinally, from the pre-hospital environment to the emergency department, then the operating theatre, followed by intensive care (with the potential for moves in either direction between some of those locations). It also exemplifies a horizontal treatment approach, whereby DCR and DCS are used simultaneously (with radiological input) to address patient physiology and anatomy in tandem. The technical skills of trauma clinicians should be complemented and reinforced by excellent non-technical skills, in order to address immediate issues, and anticipate likely challenges, contemporaneously. Equipoise is both fundamental to, and an endeavour of, the damage control approach.

Damage control future developments

Damage control strategies rely on the amalgamation and co-operation of a vast array of specialist processes and practitioners, each one of which has the capacity for development and improvement. Despite considerable research and progress, there is still very little evidence so far with regards to the specifics of damage control therapy. It is clear that effective technical and non-technical skills have an important part to play, and that interested and invested trauma clinicians and academics will continue to drive achievement and innovation in this field.

Summary

- Trauma, and exsanguination from catastrophic haemorrhage, remain significant causes of morbidity and mortality worldwide.
- Damage control strategies aim to prioritize patient stabilization, in order to facilitate ongoing management and reduce mortality.
- Every intervention is a balance of risks, and effective multidisciplinary team involvement throughout is paramount to the success of the damage control approach.
- Robust technical and non-technical skills, in combination with evidence-based practice and medical innovation, will enable the right decision to be made for the right patient at the right time.

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REFERENCES

1 Eastridge BJ, Holcomb JB, Shackelford S. Outcomes of traumatic hemorrhagic shock and the epidemiology of preventable death from injury. *Transfusion* 2019; **59:** 1423–8.

2 Kornblith LZ, Moore HB, Cohen MJ. Trauma-induced coagulopathy: the past, present, and future. *J Thromb Haemostasis* 2019; **17:** 852–62.

3 White NJ, Ward KR. Blood failure: pathophysiology and diagnosis. In: Spinella PC, ed. Damage control resuscitation: identification and treatment of life-threatening hemorrhage. Switzerland: Springer Nature AG, 2020; 41–65.

4 Cap AP, Gurney JM, Meledeo MA. Hemostatic resuscitation. In: Spinella PC, ed. Damage control resuscitation: identification and treatment of life-threatening hemorrhage. Switzerland: Springer Nature AG, 2020; 117–44.

5 Pang A, Chauhan R, Woolley T. Permissive hypotension. In: Spinella PC, ed. Damage control resuscitation: identification and treatment of life-threatening hemorrhage. Switzerland: Springer Nature AG, 2020; 101–15.

6 Dutton RP. Haemostatic resuscitation. *Br J Anaesth* 2012; **109:** i39–46.

7 National Institute for Health and Care Excellence guideline. Major trauma: assessment and initial management. 2016, https://www.nice.org.uk/guidance/ng39/resources/major-trauma-assessment-and-initial-management-pdf-1837400761285.

8 Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma* 2007; **62:** 307–10.

9 Simmons JW, Powell MF. Acute traumatic coagulopathy: patho-physiology and resuscitation. *Br J Anaesth* 2016; **117:** iii31–43.

10 Spahn DR, Bouillon B, Cerny V, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition. *Crit Care* 2019; **23:** 98.

11 Jones CP, Keene DD, Nordmann GR. Viscoelastic testing of coagulation. *Int Anesthesiol Clin* 2017; **55:** 96–108.

12 Klein AA, Arnold P, Bingham RM, et al. AAGBI guidelines: the use of blood components and their alternatives 2016. *Anaesthesia* 2016; **71:** 829–42.

13 Lamb CM, MacGoey P, Navarro AP, et al. Damage control surgery in the era of damage control resuscitation. *Br J Anaesth* 2014; **113:** 242–9.

14 Chakraverty S, Zealley I, Kessel D. Damage control radiology in the severely injured patient: what the anaesthetist needs to know. *Br J Anaesth* 2014; **113:** 250–7.

FURTHER READING

World Health Organization Fact Sheet. Injuries and violence. https://www.who.int/news-room/fact-sheets/detail/injuries-and-violence.

Practice points

- The damage control approach to trauma care is multifaceted and necessitates prompt initiation of complementary resuscitative and surgical clinical activities
- Trauma causes multi-system failure, including multiple cellular responses to tissue injury and shock such as TIC, further exacerbating physiological derangement
- DCR comprises permissive hypotension and haemostatic resuscitation and is a temporary strategy to achieve survivable patient physiology, whilst DCS targets initial control of haemorrhage and contamination
- Effective multidisciplinary management of the trauma patient facilitates comprehensive and concurrent damage control treatment, reducing both morbidity and mortality

