Damage control – trauma care in the first hour and beyond: a clinical review of relevant developments in the field of trauma care

AE Sharrock¹, M Midwinter²

¹Defence Postgraduate Medical Deanery, UK

²Royal Centre for Defence Medicine, UK

ABSTRACT

INTRODUCTION Trauma provision in the UK is a topic of interest. Regional trauma networks and centres are evolving and research is blossoming, but what bearing does all this have on the care that is delivered to the individual patient? This article aims to provide an overview of key research concepts in the field of trauma care, to guide the clinician in decision making in the management of major trauma.

METHODS The Ovid MEDLINE®, EMBASE™ and PubMed databases were used to search for relevant articles on haemorrhage control, damage control resuscitation and its exceptions, massive transfusion protocols, prevention and correction of coagulopathy, acidosis and hypothermia, and damage-control surgery.

FINDINGS A wealth of research is available and a broad range has been reviewed to summarise significant developments in trauma care. Research has been categorised into disciplines and it is hoped that by considering each, a tailored management plan for the individual trauma patient will evolve, potentially improving patient outcome.

KEYWORDS

Military – Damage control resuscitation – Haemorrhage – Rehabilitation – Coagulopathy

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CORRESPONDENCE TO

Anna Sharrock, Vascular Surgery Department, Salisbury District Hospital, Odstock Road, Salisbury, Wiltshire, SP2 8BJ
T: +44 (0)1722 336262; E: sharrock@doctors.org.uk

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Introduction

The 2007 *Trauma, Who Cares?* report by the National Confidential Enquiry into Patient Outcome and Death highlighted trauma as a significant problem.¹ As the leading cause of mortality in the first four decades of life, with two seriously disabled survivors for each death,¹ each fatality and injury costs the UK economy £750,000 and £50,000 respectively.² Trauma has become a ‘hot topic’, with interest fuelled by recent military engagements, alongside academic and political investment. The responsibility for the delivery of a good standard of care falls on the shoulders of every clinician,³ and trauma care is audited.² This summary of trauma developments aims to help translate academia into clinical practice. Specific areas of cover are haemorrhage control, damage control resuscitation, massive transfusion protocols, coagulopathy, acidosis and hypothermia, and damage-control surgery.

Methods

Hand and Medical Subject Headings searches were performed using Ovid MEDLINE®, EMBASE™ and PubMed da-

tases for articles on haemorrhage control, damage control resuscitation and its exceptions, massive transfusion protocols, prevention and correction of coagulopathy, acidosis and hypothermia, damage-control surgery, and enteric, splenic, hepatic and vascular torso injuries. Articles were included and discussed if they were directly relevant to the topic or suggested areas of new interest or development.

Haemorrhage diagnosis

Control of catastrophic haemorrhage is the priority in trauma, as exemplified by the emphasis placed on it in the modified <C>ABC algorithm,⁴ and is germane to prevention of immediate exsanguination, and emerging interacting sequelae of coagulopathy, hypothermia and acidosis (the ‘lethal triad’).⁵ Evidence of haemorrhage can be extracted using the MIST (Mechanism, Injuries sustained, vital Signs and Treatment given) history format, clinical examination and, to a lesser extent, from vital signs, which have been shown to be unhelpful in isolation,^{6,7} but suggestive in combination. Blood gas monitoring for acidosis can be used to identify occult hypotension early and improves survival by

guiding aggressive resuscitation.⁸ A useful adjunct is a viscoelastic haemostatic assay (VHA) such as rotation thromboelastometry or thromboelastography. These techniques use whole blood placed in a cup with a pin. This is oscillated to assess viscoelastic properties, and provide a real time image of clot formation and lysis.⁹ Reviews of these techniques have concluded that VHA appears to be advantageous over conventional coagulation assays in identifying coagulopathy resultant from significant haemorrhagic shock and that goal-directed therapy should be used until surgical haemostasis is achieved.^{9,10}

Damage control resuscitation

Damage control resuscitation incorporates permissive hypotension, correction and prevention of coagulopathy, and blood product use over other fluids.¹¹ Permissive hypotension has superseded the traditional approach of early liberal isotonic crystalloid resuscitation. A randomised controlled trial by Bickell *et al* showed the latter to have a detrimental effect on survival compared with delayed fluid resuscitation, probably by driving ongoing haemorrhage, thrombus disruption and dilution of blood constituents.¹² To reproduce these results, fluids should be titrated to maintain a systolic blood pressure of 90mmHg by administering 250ml aliquots of fluid.¹³

The concept of hypotensive resuscitation was considered by Duchesne *et al*, who concluded that until definitive control of bleeding is achieved, blood pressure should only be increased by a limited amount.¹⁴ Hypotensive resuscitation avoids the problems of excess fluid while improving the survival rate compared with no fluids¹⁵ and is advocated by the National Institute for Health and Clinical Excellence guidelines.¹⁶ Holcomb *et al* described the ideal resuscitation fluid as whole blood, replacing losses like for like.¹¹ However, O negative packed red cells are a suitable alternative when used as part of haemostatic resuscitation. This requires the replacement of clotting factors and platelets to prevent and treat coagulopathy.

Exceptions to permissive hypotension

A variety of exceptions to permissive hypotension can be managed by traditional normotensive resuscitation (Table 1).

An interesting group of patients considered an exception includes those exposed to prolonged evacuation times, classically in the hostile military setting. Traditionally, these patients were managed with normotensive resuscitation but an animal study by Kirkman *et al* in 2011 showed a survival advantage in this group subjected to blast when managed with 'novel hybrid resuscitation'.¹⁷ Here, the resuscitation goal is switched from hypotensive to normotensive after one hour of resuscitation, allowing clot formation as well as reversal of tissue hypoperfusion and acidosis. Normal saline was shown to be the fluid of choice in this experimental animal model, conferring a survival advantage over hypertonic saline dextran. Of note, these findings apply to haemorrhage with blast only and cannot be extrapolated

Table 1 Exemptions to permissive hypertension

Exception patient group	Reason for exception	Suggested alternative
Elderly / beta-blocked / hypertensive patients	Potentially unable to maintain an adequate cardiac output	Normotensive resuscitation
Head injuries	Cerebral autoregulation potentially disabled	Normotensive resuscitation
Pregnancy	Placental blood flow may become too low to sustain fetus	Normotensive resuscitation (consideration of emergency Caesarean section)
Prolonged evacuation times	Established acidosis, coagulopathy	Normotensive resuscitation / novel hybrid resuscitation

to haemorrhage alone. Such a strategy has been shown to mitigate coagulopathy and diminish inflammatory cytokine response.¹⁸

Massive transfusion and protocols

Catastrophic haemorrhage requires rapid massive transfusion of blood and blood products. Massive transfusion is defined in the literature as 10 or more red blood cell units in 24 hours (although the usefulness of this is debated), and it is associated with a high risk of mortality owing to coagulopathy, acidosis and hypothermia,¹⁹ alongside the standard complications of blood transfusion. Borgman *et al* reviewed 246 patients retrospectively¹⁹ and reproducibly²⁰ identified that a 1:1 ratio of fresh frozen plasma (FFP) to packed red blood cells (pRBC) was independently associated with improved survival compared with higher ratios of red blood cell units. The optimal ratio of FFP to pRBC conferring survival advantage is debated.²¹ Massive transfusion protocols are also associated with immunosuppression, worsened by aging blood, causing sepsis, organ failure and mortality,²² which may be neutralised by leucodilution.²³

To offset some of these complications, standardised massive transfusion protocols have been developed. Cotton *et al* showed a significant reduction in trauma related organ failures, sepsis and mortality compared with non-protocolised massive transfusions,²⁴ possibly because fewer blood products and crystalloids were used overall, and coagulopathy was managed aggressively. A firm consensus on product ratios has yet to be ascertained but high ratios of FFP to pRBC can be recommended.

Prevention and correction of coagulopathy

Acute traumatic coagulopathy (ATC) is related to injury severity and shock. It occurs early after injury, may be defined as a prothrombin time ratio of >1.2, and is associated with both increased mortality and transfusion requirements.²⁵ Unlike ATC, trauma-induced coagulopathy (TIC) is multifactorial

rial in aetiology and is not completely understood. Although ITC has been likened to disseminated intravascular coagulation,²⁶ individual cases vary and are not associated consistently with hyperfibrinolysis.²⁷ Management includes replacement of coagulation factors alongside pRBC units in the form of a massive transfusion protocol. Once initiated, this should ideally be monitored using a VHA and tailored to the patient using adjuncts. These may include tranexamic acid, recombinant factor VIIa (rFVIIa), and fibrinogen replacement.

CRASH-2 was a randomised controlled trial showing that early administration of tranexamic acid reduces all-cause mortality and risk of death due to bleeding in trauma patients.²⁸ A retrospective study by Morrison *et al* has reinforced this concept with 896 combat casualties, demonstrating the effect of tranexamic acid to be greater in more severely injured patients and those requiring massive transfusions.²⁹ In this study, only 7 patients needed to be treated to confer a mortality benefit, compared with 67 in CRASH-2.²⁸ Antifibrinolytic and anti-inflammatory effects are considered responsible pathways, and despite uncharacterised potential risks of venous thrombosis and embolus, the overall benefit of tranexamic acid seems to outweigh the risks, especially in those severely injured.

Holcomb *et al* advocated rFVIIa¹¹ but subsequent studies have shown that although rFVIIa reduces blood product usage, it probably does not improve overall survival.^{30–32} Early termination of the randomised controlled CONTROL trial³³ was prompted by a futility analysis owing to lower-than-projected mortality rates. Having primarily set out to compare 30-day mortality, this study of rFVIIa as an adjunct to direct haemostasis in major trauma identified that rFVIIa reduced blood product use significantly, without increasing adverse or thrombotic events. The better than projected mortality rates in this study were attributed to patient selection and strict management protocols designed to reduce variability in patient care. Fewer than 50% of the included patients required a massive transfusion, which emphasises the requirement to trial rFVIIa in more seriously injured patients before firm conclusions can be drawn.

Overall, to maintain the status quo of coagulation, haemostatic resuscitation with the appropriate use of adjuncts guided by VHA is recommended. Careful monitoring and correction of acidosis, haematocrit, hypothermia and hypocalcaemia³⁵ should be considered. The patient is vulnerable to coagulopathy long after the initial traumatic injury due to potential occult haemorrhage and ongoing surgery potentially causing blood loss.

Prevention and correction of acidosis

Hypoperfusion and poor oxygenation accounts for acidosis in trauma, alongside lactate from anaerobic metabolism. It is worsened by crystalloids. A pH of 7.1 or a base excess of -12.5mmol/l or less significantly impairs haemostasis as most coagulation proteases function at best between pH 8 and 8.5,^{35,36} factor VIIa being very sensitive to pH shifts.³⁷ Reversal of acidosis relies on improving tissue oxygen delivery by improving perfusion and oxygenation through blood volume, pressure and products, and rewarming. Blood transfu-

sion may paradoxically make acidosis worse as aged blood is acidic, hyperkalaemic and has an altered 2,3-diphosphoglycerate. This effect can be remedied by pH buffering, rewarming and ongoing resuscitation.

Prevention and correction of hypothermia

Hypothermia is common in trauma patients owing to the nature of the injury, exposure (often worsened in the emergency department and theatre) and altered thermoregulation. Hypothermia correlates directly with prognosis, and effects coagulation by reducing platelet adhesion and activation, and promoting sequestration.¹⁴ Temperatures below 34°C significantly reduce enzyme activity while causing microvascular thrombosis,³⁵ resulting in a mixed coagulopathy. Exposure should be minimised, the department warmed, Bair Hugger® (Arizant Healthcare, Eden Prairie, MN, US) therapy/blankets used and fluids warmed. Failing this, invasive rewarming may be indicated.

Damage-control surgery

The objectives of managing the trauma patient are primarily survival of the patient and secondly to maximise the patient's physical, psychological and social functional recovery as quickly as possible, and to do no further harm. These must be considered when undertaking damage-control surgery as a facet of damage control resuscitation. The aim is to manage life and limb-threatening limb injury expediently, and to avoid preventable complications by doing the minimum to stop haemorrhage quickly and control contamination. Its nature may require initiation in the emergency department and much improvisation with definitive surgery taking place at a later date.

Almost a third (32%) of laparotomies for intestinal injury performed at UK military forward hospitals between 2003 and 2008 were damage control procedures.³⁸ Of the remaining definitive laparotomies, 27% required unplanned relook procedures. Although numbers were low in this study, the implication is that damage-control surgery may be appropriate for a greater proportion of intestinal injury candidates.

Blunt torso trauma

Any abdominal trauma coupled with haemodynamic instability or diffuse abdominal tenderness is a strong indication for an emergency laparotomy.^{39–41} Selective non-operative management (NOM) for blunt trauma may be appropriate⁴² in a haemodynamically stable patient as up to 67% of exploratory laparotomies in this patient group are negative.⁴³ Computed tomography (CT) is the imaging of choice to identify splenic and hepatic injury, and should be performed with oral and intravenous contrast to exclude hollow viscus involvement.⁴⁴

Penetrating torso trauma

Up to 53% of laparotomies for stab wounds⁴⁰ and 47% of those for gunshot wounds⁴⁵ are negative. In response

to an argument for NOM of penetrating injuries, the Eastern Association for the Surgery of Trauma (EAST) concluded that a routine laparotomy is not indicated in haemodynamically stable abdominal stab wound patients in the absence of peritonitis or diffuse abdominal tenderness, or in those with tangential gunshot injuries and no peritoneal signs.^{41,46} Instead, triple contrast abdominopelvic CT alongside serial examinations for peritonitis is suggested, with the majority of patients suitable for discharge at 24 hours of observation providing there is minimal to no abdominal tenderness. However, further investigation or laparotomy is indicated in the presence of an unreliable clinical examination. Recommendations in these guidelines were drawn from 95 articles producing evidence albeit in the understandable absence of randomised controlled trials.

A recent article explored trauma surgeons' practice, demonstrating that those who had trauma fellowships and were from high-volume centres in countries such as South Africa and America were far more likely to practise selective NOM of penetrating trauma.⁴⁷ Despite low numbers and the theoretical risk of selection bias, this study showed a disparity in the execution of selective NOM in penetrating trauma. The concept has yet to graduate from theory to practice in some centres despite the 2009 EAST guidelines.⁴¹

Enteric injuries

CT is advocated and has been shown to consistently have above 90% sensitivity and specificity for peritoneal and retroperitoneal injury.⁴¹ Although reports vary, evisceration is considered an indication for surgery.⁴¹ Indeed, one study demonstrated a 100% and 76% positive laparotomy rate for intestinal and omental evisceration respectively.⁴⁸

Small bowel or non-destructive colon injuries (destruction of less than 50% of the colon circumference)⁴⁹ may be dealt with appropriately by primary repair at damage control laparotomy. A deteriorating patient and/or the presence of destructive or multiple injuries commands the use of a linear stapler to interrupt the bowel continuity expediently and control contamination,^{50,51} leaving repair/anastomosis or exteriorisation of the bowel to a relaparotomy at 48–72 hours.⁴⁹

A retrospective analysis by Burlew *et al* of post-injury open abdomen patients concluded that all should be considered for repair as mortality was significantly higher in the stoma (37%) than in the anastomosis (8%) group.⁵¹ Interestingly, this group noted reproducibly that a 3% leak rate in small bowel increased to 45% in left-sided colonic injuries, which worsened to 48% in those with an open abdomen beyond day 5 following the injury. Ott *et al* concurred and demonstrated high transfusion requirements to be an independent risk factor for leak.⁵² Ordoñez *et al* noted that severe acidosis, bowel wall oedema and persistent intra-abdominal sepsis should preclude delayed anastomosis.⁴⁹ In such high-risk patients with left-sided destructive injuries with an open abdomen beyond day five, exteriorisation of the bowel should be a serious consideration.

Splenic injuries

The majority (80%) of patients with blunt splenic injury can be managed non-operatively and need only be monitored as inpatients for 3–5 days.^{53,54} Should NOM fail, 60% will do so within 24 hours and 95% within 3 days.⁵³ Of the 60% failures in this study by Peitzman *et al*, 40% were due to misinterpretation of CT and ongoing haemodynamic instability, highlighting the importance of correct patient selection.⁵⁹ CT evidence of a traumatic pseudoaneurysm, active contrast extravasation or a grade 4 injury (which has less than 50% success of NOM)⁵⁵ or a grade 3 splenic injury with a large haemoperitoneum⁵⁵ suggest high risk of failure. Importantly, splenic injury grade is not a contraindication to NOM,⁴⁴ and mortality is unchanged in isolated splenic injuries managed operatively and in those with failed NOM,⁵³ suggesting that closely monitored patients may undergo a trial of NOM.

Sabe *et al* described splenic artery embolisation (SAE) as an adjunct to NOM that improves its success and splenic salvage while reducing mortality.⁵⁵ This must be considered in the light that groups were compared between consecutive years so other factors and developments besides increased use of SAE may have influenced the findings. However, SAE carries a 5% abdominal complication (pseudoaneurysm and abscess formation) risk,⁴² which compares favourably with the 36% risk of abdominal complications following a laparotomy. SAE is not without risk but it is a useful adjunct to NOM if deployed judiciously so as to not delay a potentially life-saving laparotomy or to not unnecessarily expose patients to associated complications. The 180-day risk of re-admission for NOM of these patients is 1.4%, the majority occurring within the first eight days,⁵⁶ illustrating the importance of close follow-up.

Hepatic injuries

NOM of haemodynamically stable patients with a CT proven liver injury has a success rate of approximately 90%^{57–59} and has reduced mortality in recent years.⁶⁰ Predictors of NOM failure include high-grade liver injuries and pooling of contrast⁴⁵ although a study by Velmahos *et al* revealed that only 2% of NOM failures were related to liver injury, reflecting a multiply injured patient group.⁵⁸ Parks *et al* identified 1.9 days as the average stay for successful NOM across all injury grades.⁵⁹ They concluded that haemodynamic status, physical examination and serial haemoglobin measurements can determine discharge safely as failures for haemorrhage occurred early and other failures developed in multiply injured inpatients. NOM is, however, not risk free. Indeed, hepatic complications run at 14% and a significant difference between grades has been identified reproducibly (5% in grade 3, 22% in grade 4, 52% in grade 5).⁶¹

Operative intervention is required in high-grade liver injuries with haemodynamic instability and mortality is high.⁵⁷ Packing with gauze to realign the anatomical shape is often adequate for minor bleeding associated with grade 1 and 2 liver injuries.⁶² Mobilisation may be required, and packs should be removed between 36 and 72 hours as secondary haemorrhage may occur with earlier removal.⁶³

Manual compression and the Pringle manoeuvre (performed safely for up to 60 minutes in normal livers)⁶⁴ in major haemorrhage alongside hepatotomy (finger fracture) and direct suture is the next logical step. This has a lower morbidity than hepatorrhaphy (deep parenchymal suturing),⁶⁵ which is useful in difficult areas but risks tissue infarction.

In penetrating trauma, a tractotomy of peripheral tracts, and tamponade of central tracts with a Foley catheter and Penrose drain⁶⁶ or Sengstaken–Blakemore tube⁶⁵ have been advocated. Tractotomies to central tracts risk ongoing haemorrhage and worsening bile leak, and should therefore be avoided.⁶⁶ Non-segmental resectional debridement and selective hepatic artery ligation in the absence of a major venous injury is an option of final resort.⁶² Where possible, NOM should be trialled as patients with high-grade liver injuries who are managed operatively have been shown to have worse outcomes than those with successful NOM.⁵⁷

Hepatic angiography and hepatic angiography embolisation (HAE) have improved the success of NOM and are indicated in CT proven contrast extravasation.⁶⁷ HAE is also used in selective embolisation of deep hepatic parenchymal bleeding in haemodynamically unstable patients with a grade 3 and above liver injury after a damage control laparotomy.^{68,69} Liver-related complications of HAE (bile leaks, abscesses, and liver and gallbladder necrosis) run high (50–70%)^{67,69} but selective HAE may reduce the latter.⁷⁰ HAE use has increased in trauma and it has been suggested that surgery may become an adjunct to HAE in level 1 trauma centres in those patients with borderline haemodynamic instability.⁷⁰ This concept should be deployed cautiously; vigilant patient monitoring and early exploratory surgery for failing patients in this situation is paramount.

Vascular injuries

Up to 25% of patients admitted to hospital with abdominal trauma and the majority of those who die have vascular injuries.⁷¹ Haemodynamically unstable non-responders should raise suspicions of vascular injury. It has been advocated by Tyburski *et al* that patients with a systolic blood pressure persistently below 70mmHg undertake a prelaparotomy thoracotomy for proximal aortic control.⁷¹ In their study, 51 patients underwent a prelaparotomy thoracotomy; 29 responded and, of those, 11 survived. Interestingly, all those who did not respond to aortic cross clamping by an increase in systolic blood pressure to at least 90mmHg died and these patients were considered irretrievable.

Avaro *et al* used aortic balloon occlusion at the supraceliac aorta in haemorrhaging pigs.⁷² They concluded that this potentially less invasive alternative to surgical proximal aortic control could be placed without radiological guidance and could be left in situ for up to 40 minutes without significant visceral or cardiac pathology developing. However, an earlier study questioned its safety, suggesting that renal and neurological complications were more frequent, and mortality was higher than in the control group.⁷³ Overall, aortic balloon occlusion is an interesting alternative to surgical proximal haemorrhage control but its role is not yet fully developed and its safety not fully established.

Vascular injuries in the abdomen may lie in the central retroperitoneum, lateral retroperitoneum, pelvis or retrohepatic and porta hepatis area. It is advocated that central haematomas should be explored, as should lateral haematomas in penetrating injuries and, if expanding, in blunt injuries⁷⁴ while non-expanding haematomas in blunt trauma may be managed expectantly.⁷⁵

Surgical management involves proximal and distal control, exposure and identification of the defect, then primary repair, shunt or interposition graft.⁵⁰ Where visceral spillage occurs and the risk of contamination of the repair is high, endovascular intervention has been performed successfully as an adjunct to surgery.^{76–78} Endovascular techniques have also been deployed swiftly in instances of secondary haemorrhage after repair in a contaminated, hostile abdomen⁷⁹ or in more stable lesions such as a pseudoaneurysm detected later. These case reports highlight the importance of a prompt accessible endovascular service. It must be considered that delay may be fatal for the patient.

Inferior vena cava (IVC) injuries are fortunately rare as approximately 66% of patients reaching hospital do not survive them, with figures worse for blunt and retrohepatic injuries.⁸⁰ Simple non-expanding lateral retroperitoneal haematomas in blunt injuries may be managed conservatively.⁵⁰ When damage-control surgery is necessary, such haematomas may be left unexplored. In the case of large or expanding retroperitoneal haematomas and haemodynamic instability, intervention may be necessary.

Novel surgical strategies have been described to repair the IVC but, ultimately, this involves adequate access, opening the haematoma, proximal and distal control, and primary (lateral) repair, temporisation or ligation of the IVC.⁵⁰ Blood loss may be immense, and case reports of endovascular balloon occlusion of the IVC,⁸¹ atriocaval shunting,⁸² and cardiopulmonary bypass and hypothermic circulatory arrest⁸³ describe successful methods with low numbers to reduce blood loss at the time of surgery. Endovascular therapy may have a role to play, and case reports exist of IVC endograft placement for surgical haemorrhage⁸⁴ and fenestrated endografting for juxtahepatic IVC injury via the right femoral vein.⁸⁰ Although long-term durability of such grafts is unknown, such techniques present an interesting alternative to technically difficult high-risk surgery.

Conclusions

A number of key concepts have evolved in trauma care. These have been addressed and salient research has been summarised to produce an overview. This is by no means exhaustive but it is hoped that this simplification will help clinicians without experience who are faced with managing trauma. This review has attempted to apply the significant developments to the individual patient's care by considering resuscitative and surgical management where research is ongoing and changes are evolving. It is, however, hoped that this article will guide decision making through the practical application of theory, and promote further interest in trauma care and its teaching.

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