

REGULAR REVIEW

Damage Control Surgery - Concepts and Practice

DMG Bowley, P Barker, KD Boffard

Introduction

The traditional surgical approaches to resuscitation and trauma have been based on tried and tested methods of managing penetrating injury with exploration, control of bleeding and contamination and attempts to achieve definitive repair of damaged structures. The 'damage control' approach was born out of a need to meet the challenge of the changing scope and severity of urban violence in America and elsewhere over the last decade and a half. Increased use of semi-automatic weapons, often with altered ammunition and fired with higher muzzle velocity, has led to increased numbers of patients presenting with exsanguination and critical physiological instability (1). Surgeons have been forced to reconsider their approach to the critically injured patient and to revisit their preconceptions. It has become necessary to stand back and look from a new perspective - described as an ability "to think outside the box" (2).

Historical perspective

The concept of 'damage control' surgery in the management of trauma victims is that only the minimum is done to stop bleeding and limit or contain contamination before the wound or cavity is packed and/or temporarily closed. The patient is then taken to Intensive Care and attempts are made to 'normalise' the patient physiologically before returning to the operating theatre for definitive surgery. This breaks the lethal cycle of hypothermia, acidosis and coagulopathy.

Despite this apparently novel approach, temporising manoeuvres in the treatment of critically injured patients are not new. In 1908, Pringle introduced a technique to control bleeding from hepatic trauma by using gauze pads secured by sutures (3). This approach was described in detail and subsequently modified by Halstead in 1913 (4). Packing for control of hepatic haemorrhage fell out of favour and by the end of World War II the technique was almost universally condemned due to problems with re-bleeding on pack removal and late sepsis (5). In 1955, Madding once again used packs to control bleeding but emphasised that the packs should be removed by the end of the operation (6). In 1976, Lucas and Ledgerwood described a

prospective series of 637 patients treated for severe liver injury, three were considered for insertion of perihepatic packs and all three survived (7). In 1978 AJ Walt summed up the growing confidence with packing when he said, "I have no wish to revivify the idea of the pack as a desirable standard practice. On the other hand, the judicious surgeon who chooses this method should in no way fear the whispered loss of his surgical manhood" (8). Calne, in 1979, reviewed 4 patients treated in this manner, all returned to theatre for definitive surgery and all survived (9). Following this trend, in 1981, Feliciano described a series where nine out of ten patients survived when intra-abdominal packing was used to control hepatic haemorrhage (5). Stone went further, and in 1983, described a stepwise approach using intra-abdominal packing and rapid termination of laparotomy with suggestions for temporising manoeuvres for other injured organs, such as the intestines and urinary tract. A 65% survival rate in 17 patients was reported with this approach (10). In 1992, Burch reported 200 patients treated with abbreviated laparotomy and planned re-operation with 33% survival (11). The following year, Charles Schwab in Philadelphia coined the phrase "damage control" and detailed a standardized approach with a 58% survival rate (12). Damage control has now firmly entered the surgical vocabulary and the underlying concepts have found application not only in the abdomen, but also for injury to the chest and pelvis, for urological, vascular and extremity injury and even in obstetric practice to treat spontaneous hepatic rupture during childbirth (13).

Physiological rationale for damage control

Trauma has been described as a 'disease of bleeding' (14). Recognition of acute blood loss after injury, and restoration of homeostasis is the cornerstone of the initial care of the badly injured patient. Ongoing haemorrhage leads to the onset of a cycle of three inter-related variables, metabolic acidosis, profound hypothermia and a clinically obvious coagulopathy (figure 1). Each of these factors reinforces the others and death of an exsanguinating patient is due to the consequences of what Kashuk and Moore have described as "this bloody

Maj DMG Bowley
FRCS RAMC
Specialist Registrar in
General Surgery
Dept of General
Surgery
Derriford Hospital
Plymouth PL6 8DH

E-mail:
doug.bowley@virgin.net

The Revd Prof P Barker
MS FRCS FICS
Surgeon Commander
Royal Navy
Defence Medical
Services Professor of
Clinical Surgery
Royal Hospital Haslar
Gosport Hants
PO12 2AA

Prof KD Boffard FRCS
FRCS(Ed) FACS
Professor of Trauma
Surgery
Johannesburg Hospital
Trauma Unit
Post net Suit 235
Private bag x2600
Loughton
Johannesburg
South Africa

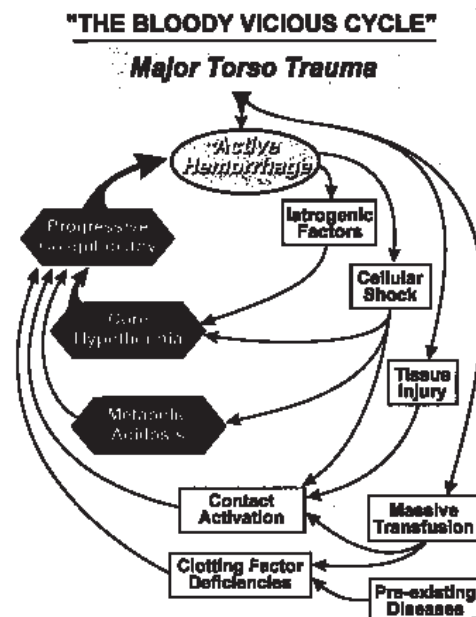


Fig 1. Pathogenesis of the bloody vicious cycle after severe injury is multifactorial, but progressive core hypothermia and persistent metabolic acidosis are pivotal. Reprinted from *Am J Surg.* 1996; 172(5): 405-10 Staged laparotomy for the hypothermia, acidosis and coagulopathy syndrome. Moore EE. Copyright (1996) with permission from Excerpta Media Inc.

vicious cycle" (15).

The thermoneutral zone (28°) is defined as the ambient temperature at which the basal level of thermogenesis is sufficient to offset ongoing heat loss, and sympathetic tone is neutral (16). When ambient temperature is below the thermoneutral zone and tissue oxygen consumption is limited by shock, heat production cannot offset loss and hypothermia occurs despite maximal sympathetic drive. Transfusion of fluids, exposure of the patient in a cold resuscitation room and surgical opening of body cavities compounds the problem. Furthermore, decreased thermogenesis is often complicated by anaesthetic agents and paralyzing drugs that can decrease heat production by up to 33% (16). Core body temperature below 35° significantly affects the ability of the blood to clot (17) and cold-mediated platelet dysfunction can result in coagulopathic bleeding despite a normal count. Laboratory measurements for standard laboratory values are obtained in specimens warmed to 37°, hence clotting times may be normal in the face of a significant coagulopathy due to hypothermia (18).

Bernabei found hypothermia to be an independent risk factor, predictive of increased blood loss during laparotomy for trauma (19). Jurkovich, measured the lowest recorded core temperature in a group of injured patients and stratified them according to Injury Severity Score, blood and fluid requirements and presence or absence of shock. Patients who became hypothermic (<35°) had significantly higher mortality than patients with similar injuries who remained warm (20). In this study,

mortality was 100% if the temperature fell below 32°, even in mildly injured patients. Aggressive re-warming has been shown to be associated with significant reduction in mortality, blood loss, fluid requirement, organ failure and length of intensive care unit stay (21).

The Damage Control Sequence

a) Patient selection

Most patients with penetrating abdominal injury require traditional management, with abbreviated laparotomy being necessary in only a few. During a 3 year period at the University Hospital in Cali, Columbia, 2437 laparotomies were performed for trauma. Of these, 117 (4.8%) required damage control (22). Damage control should be limited to those few patients who are critically unstable, with associated multi-visceral injury and exsanguination.

It may be possible to identify, or 'pre-select' patients who are likely to need the damage control approach before theatre, based on mechanism of injury, method of presentation and initial physiological compromise. High-energy blunt trauma or penetrating injury associated with abnormal vital signs should alert the physician to the possibility of severe or ongoing bleeding. Arterial blood gases can be helpful on presentation, with a base excess > -6 suggesting significant hypovolaemia. Blood lactate >2.5mg/L, is also a useful marker, if readily available.

The decision to abbreviate a laparotomy should ideally be made within the first few minutes of the operation, if not decided on before (23). The onset of diffuse bleeding in a cold patient implies the physiological limit of the patient has already been passed. Carrillo demonstrated that a core temperature of 34°, a pH of <7.25 and coagulopathy are associated with high mortality (24). In a retrospective series of 56 laparotomies for major trauma which were terminated abruptly, evidence of a diffuse coagulopathy was present in 37 and not in 19. Of the 37 coagulopathic patients, 16 died; whereas only 3 died out of 19 when coagulopathy was absent (25).

b) Initial laparotomy

A full midline incision is made, after prepping the patient to anticipate the need for thoracotomy, sternotomy or vascular access as necessary. Haemoperitoneum is evacuated and the abdomen packed in four quadrants. Control of haemorrhage is achieved either by direct means using sutures, ligatures, clips or electrocautery or by indirect means with use of packs or balloon tamponade for deep hepatic wounds (26). Packing is the fastest and best way to control bleeding from a disrupted liver (22). Even retrohepatic caval injury may be managed by packing alone (27,28).

There is a definite 'learning curve' to the use of packing in trauma surgery. The key principle is that packing should re-approximate disrupted tissue planes (29), thus the injured hepatic lobe must be mobilised and compressed by packs above and below. The two major pitfalls in packing are overpacking and underpacking. Overpacking will result in raised intra-abdominal pressure and haemodynamic compromise due to compression of the inferior vena cava, effectively producing an abdominal compartment syndrome. Underpacking is ineffective placement of packs, which fail to stop the bleeding. Packing must be effective in controlling bleeding at time of laparotomy as hope that time, intrinsic coagulation or tamponade will stop bleeding is futile. Packing may also be used for control of non-hepatic bleeding, but the technique will not control arterial bleeders, which must be controlled by other methods.

Hollow viscus injuries are controlled by tying with tapes, simple running sutures or via the use of stapling devices. Resection may be quickly achieved, but reconstruction is deferred, the closed-off loops of bowel being returned into the abdominal cavity. Vascular injuries may be treated by ligation or by placement of temporary vascular shunts (30). Angiographic embolization may complement other methods to achieve haemostasis (31).



Fig 2. Temporary closure of the abdominal wall using a "Bogota bag". A urology irrigation bag has been used in this instance.

Biliary, pancreatic and urological injuries should be controlled by external tube drainage with stenting when possible (32).

Temporary closure of the abdominal wall may be achieved by a variety of means, the simplest using skin clips or a running suture of the abdominal skin only. In the presence of severe visceral oedema, a plastic intravenous bag may be sutured to the skin edge of the abdominal wound (the so-called Bogota Bag, see figure 2). In Johannesburg, an even quicker method is preferred; an abdominal pack is opened out

and laid on the adherent surface of a sheet of Opsite™. This combination is tucked under the rectus sheath and 2 suction drains placed over it. The entire wound is then occluded with a large Opsite™ sheet and the suction drains are attached to continuous, low-pressure wall suction. This technique is known as the 'Opsite™ sandwich' (figure 3). Use of the Opsite™ sandwich is quick and reduces the likelihood of abdominal compartment syndrome; it eliminates any tissue damage to the skin or fascial sheath and prevents soiling of the bed from the sometimes considerable amount of fluid leaking from the abdominal wound.

c) Physiological restoration

On completion of the initial laparotomy, efforts are focused on restoring the patient's deranged physiology. The goal of critical care for the victim of major trauma is adequate oxygen delivery at a cellular level and the priorities are optimisation of haemodynamic status, re-warming and restoration of normal coagulation parameters. Cellular dysfunction leading to organ failure may be caused directly by inadequate oxygen delivery or via the effects of neurohumeral mediators released after an episode of shock or critical injury. Due to the profound existing deficit and exaggerated requirements due to the systemic inflammatory response syndrome, patients with 'normal' values for oxygen delivery may demonstrate signs of hypoperfusion (33). Enhancement of oxygen delivery is possible through manipulation of three therapeutic options: (1), augmenting cardiac output with volume infusion; (2), enhancing cardiac output with inotropic agents; (3), improving oxygen-carrying capacity with blood transfusion. Placement of a pulmonary artery catheter will allow measurement of the oxygen transport parameters.

The priority for the respiratory management of the critically wounded patient is to provide adequate oxygenation and adequate removal of CO₂ by means of mechanical ventilation. An initial setting for the ventilator might be; FiO₂ of 100%, PEEP of 5cm, tidal volume of 6mL/kg, rate of 12 breaths per minute and synchronised intermittent mandatory ventilation mode (SIMV) (34). Volume-cycled respirators deliver oxygen at whatever pressure is necessary to deliver the pre-determined volume. As interstitial oedema, collapsed alveoli and retained secretions cause reduced compliance and progressive acute lung injury develops, higher and higher pressures are required to achieve set tidal volumes. Pressure-controlled ventilation may be required to minimise barotrauma to the lung (35). Worsening airway pressures should also alert the clinician to the



Fig 3. Temporary closure of the abdominal wall using an “Opsite™ sandwich”. The edges of the wound have been reinforced with hydrocolloid dressing.

possibility of intra-abdominal hypertension and a developing abdominal compartment syndrome.

Blood, fresh frozen plasma and platelets are given to enhance oxygen delivery and correct coagulopathy. Laboratory tests of coagulation and haematocrit may lag behind real-time blood-loss and empirical transfusion is recommended (36). Rewarming should be actively pursued with external forced air devices and warming of all intravenous fluids and ventilator circuits. Ambient temperature should be raised into the thermoneutral zone (28–30°C) if possible.

d) Return to theatre

The timing of planned re-operation after damage control is controversial, while normothermia and a normal coagulation profile have been considered the minimal prerequisites for safe re-operation (37), prolonged delay to achieve homeostatic perfection may lead to second-hit phenomena as sepsis, ARDS and organ failure develop. However, re-operation has been delayed for between 48 and 96 hours in some series (24). Timing of re-operation will be dictated by the pattern of injury; for example, an injury that required placement of a vascular shunt will call for earlier definitive repair than stapled-off bowel. The usual range of time between damage control laparotomy and planned return to theatre is wide, varying between 6–90 hours, the majority being returned within 48 hours. It is clear that duration of packing does impact on morbidity, in a study of 35 patients treated with abdominal packing who survived to re-operation Abikhaled found patients packed for a total of 72 hours or less had lower abscess, sepsis and mortality rates than those packed for more than 72 hours. The differences in abscess rate and mortality were statistically significant (38). When the patient is returned to the

operating room for removal of packs and completion of definitive surgical procedures, a thorough search must be made for missed injuries. Injuries missed at initial operation have been shown to be a potent cause of morbidity and mortality and have been described as “the nemesis of the trauma surgeon” (39). Scalea reported experiences with missed injury and felt that intra-operative complications of hypotension, coagulopathy and hypothermia contributed to missing injuries in 75% of cases. In the remaining 25%, injuries were missed due to inadequate exploration or too low an index of suspicion in the presence of multiple injuries (39). The full missile trajectory must be traced, as a discontinuous trajectory, or an odd number of gastrointestinal perforations are classic markers of missed injury (40). Packs are sequentially removed and repairs inspected, vascular reconstruction may then be undertaken and urological and gastrointestinal continuity restored. If possible, ‘critical’ packs should be left undisturbed until the last moment as unpacking may lead to recurrent bleeding and repacking, the so-called “pack and peek syndrome” (27).

Trauma patients have increased nutritional needs and early enteral feeding has been shown to reduce postoperative septic morbidity after trauma. Following a number of randomised trials, a meta-analysis by Moore, in 1992, demonstrated a twofold decrease in infectious complications in patients treated with early enteral nutrition compared to total parenteral nutrition (41). It may be possible to manipulate a fine-bore feeding tube beyond the pylorus during relook surgery, but if not, then a feeding jejunostomy should be inserted depending on the pattern of injuries and the integrity of the intestinal tract. Intestinal anastomoses are not a contraindication to enteral feeding (42, 43).

When definitive surgery has been completed, it may not be possible to close the abdominal wall. Definitive closure may have to wait until visceral oedema has completely subsided. Occasionally, a return to theatre may be necessary due to severe ongoing blood loss or deterioration of the patient due to the abdominal compartment syndrome. Often, these two indications co-exist in the same patient. In the face of a severe abdominal compartment syndrome, temporary improvement may be achieved by decompressing the abdomen in the intensive care unit, but re-exploration in theatre and a search for active surgical bleeding is usually necessary.

Abdominal Compartment Syndrome

After damage control laparotomy, intra-

abdominal pressure (IAP) will rise due to a combination of the presence of abdominal packs, accumulation of blood and clot, visceral oedema, and closure of a swollen, non-compliant abdominal wall. The level at which raised IAP becomes clinically significant is unclear, however, Morris has labelled the presence of a tensely distended abdomen, elevated IAP, impaired ventilation due to high inspiratory pressures, hypercarbia and oliguria as the Abdominal Compartment Syndrome (ACS) (29). The lethality of ACS is well documented with mortality rates ranging between 42 and 68%, despite decompression and a mortality rate of 100% has been shown in patients with ACS who are not decompressed (44).

Historical perspective

The morbid results of raised IAP have been noted for almost 100 years; in 1911 Emerson noted small animals died of respiratory failure if their IAP was raised beyond 27–46 mmHg (45). ACS was first described by Richardson and Trinkle in a canine model in 1976 (46), who increased abdominal pressure while measuring the abdominal intra-caval pressure. When the IAP was 10mm Hg venous return and cardiac output decreased, as the IAP rose beyond 25 mm Hg, the airway pressure increased. In the early 1980's, Richard and Kron separately reported raised IAP, usually due to post-operative haemorrhage which led to oliguric or anuric renal failure and which responded to abdominal decompression (47, 48). Since then, there have been many clinical papers detailing the expanding spectrum of complications caused by intra-abdominal hypertension.

Cardiovascular effects

The fundamental abnormality in ACS is a reduction in cardiac output. The cause of this decrease is diminished venous return due to vena caval compression, increased peripheral resistance, or both (45).

Renal effects

Renal failure caused by ACS is multifactorial. Inadequate perfusion due to reduced cardiac output, direct compression of the renal parenchyma, obstruction of the renal venous outflow and ureteric compression have all been implicated. However, normalization of cardiac output and ureteric stenting without decompression of the abdomen will not return renal blood flow or glomerular filtration rate to normal (49).

Respiratory effects

Increasing IAP impairs ventilation by limiting diaphragmatic excursion; this causes decreased pulmonary compliance, an increase

in airways pressure, reduced tidal volumes and retention of carbon dioxide (50).

Visceral perfusion

In addition to a reduction in renal blood flow (51), and thereby glomerular filtration rate, raised IAP also results in reductions in tissue oxygenation in the bowel (52), liver (53) and abdominal wall (54) due to reduced perfusion.

Intra-cranial effects

Following observation that elevated IAP during laparoscopy caused a significant elevation in intra-cranial pressure (55); there have been reports of patients with multisystem trauma and raised intra-cranial pressure refractory to medical intervention. These patients have shown a reduction in intra-cranial pressure and an improvement in cerebral perfusion on decompression of the abdomen (56).

Effects on monitoring systems

Intra-abdominal hypertension also interferes with haemodynamic monitoring, observed central venous and wedge pressures may be spuriously high despite low intravascular volume (57).

Adverse effects of decompression

Sudden and severe hypotension and death may occur during or immediately after surgical decompression of ACS (29). The mechanism is thought to be due to an ischaemia-reperfusion phenomenon. This highlights the dangers of decompressing the abdomen when there is clear clinical evidence of organ dysfunction. If decompression of an established ACS is planned, then abrupt and severe haemodynamic changes must be anticipated. Volume pre-loading and infusion of mannitol and sodium bicarbonate immediately before and during the decompression are recommended (58).

Monitoring IAP

IAP is easily monitored by recording intra-vesical pressure in the intensive care unit. With the patient flat in bed, a central venous manometer system is connected to the urinary catheter. If available, on-line pressure transducers can be used. Up to one hundred mls of water is instilled into the empty bladder and the pressure recorded relative to the pubic symphysis. A rough guide for assessment of the IAP can be obtained by elevation of the catheter tubing and measurement of the column of urine. With use of routine monitoring of IAP and gastric tonometry, Ivatury has shown that splanchnic hypoperfusion and gut mucosal ischaemia commences long before ACS is clinically evident (59). Although the critical

intra-abdominal pressure is unclear, a level of 20–25 mmHg warrants careful monitoring and prompt decompression if it continues to rise (60). Abdominal Compartment Syndrome may also occur in the absence of abdominal injury. Maxwell has reported a series of patients with documented ACS after exsanguinating haemorrhage with injuries remote from the abdominal cavity. The underlying mechanism is proposed to be gut ischaemia caused by haemorrhagic shock, followed by re-perfusion injury that results in progressive ascites, visceral and retro-peritoneal oedema (44).

Military considerations

Hypovolaemia remains the commonest cause of death amongst those killed in action during military conflicts (61). The principle of damage control, with a short life-saving surgical encounter followed by stabilization and transfer for definitive surgery has great appeal for field surgeons. The scenarios where damage control may be most use to the military are perhaps those where surgical facilities are suddenly exposed to mass casualties, or where a front-line surgeon lacks the skills or equipment for complex reconstruction. It is an axiom of military surgery to utilize resources to salvage the greatest number of patients, however, damage control procedures may consume huge resources, many series reporting transfusion of more than 20 units of blood products within the first day (45). Techniques such as intra-operative blood salvage may be helpful in these circumstances, but triage protocols must be rigorously enforced by the surgical team in an austere environment. This will ensure scarce and valuable resources are not “wasted” on unsalvageable patients.

Summary

The results of prolonged and extensive procedures in the critically injured are poor, even in experienced hands. The operating theatre is a hostile and physiologically unfavourable environment for the severely injured patient. Laparotomy for major trauma involves dissipation of heat and massive blood loss requiring replacement. The result is a vicious cycle of hypothermia, acidosis and coagulopathy leading to death from an irreversible physiological insult (62). The damage control concept places surgery as an integral part of the resuscitative process, rather than an end in itself, and recognises that outcomes after major trauma are determined by the physiological limits of the patient, rather than by efforts of anatomical restoration by the surgeon. All those involved in the care of wounded patients should be familiar with this concept and its surgical and logistical implications.

References

- McGonigal MD, Cole J, Schwab CW, Kauder DR, Rotondo MF, Angood PB. Urban firearm deaths: a five year perspective. *J Trauma*. 1993;**35**(4):532-537.
- Mattox KL. Introduction, background and future projections of damage control surgery. *Surg Clin North Am*. 1997;**77**(4):753-759.
- Pringle J. Notes on the arrest of hepatic haemorrhage due to trauma. *Ann Surg*. 1908; **48**:541.
- Halstead W. The employment of fine silk in preference to catgut and the advantage of transfixing tissues and vessels in controlling haemorrhage. *JAMA*. 1913;**60**:1119.
- Feliciano D, Mattox KL, Jordan G. Intra abdominal packing for control of hepatic haemorrhage: a reappraisal. *J Trauma*. 1981;**21**(4):285-290.
- Madding G. Injuries of the liver. *Arch Surg*. 1955;**70**:748.
- Lucas C, Ledgerwood A. A prospective evaluation of haemostatic techniques for liver injuries. *J Trauma*. 1976;**16**(2):85-88.
- Walt AJ. The myth of hepatic trauma - or Babel revisited. *Am J Surg*. 1978;**135**:12-18.
- Calne R, McMaster P, Pentlow B. The treatment of major liver trauma by primary packing with transfer of the patient for definitive treatment. *Br J Surg*. 1979;**66**(5):338-339.
- Stone H, Strom P, Mullins R. Management of the major coagulopathy with onset during laparotomy. *Ann Surg*. 1983;**197**(5):532-535.
- Burch J, Ortiz V, Richardson R. Abbreviated laparotomy and planned re-operation for critically injured patients. *Ann Surg*. 1992;**215**(5):476-484.
- Schwab CW. Violence; America's uncivil war. Presidential address, Sixth Scientific Assembly of the Eastern Association for the Surgery of Trauma. *J Trauma*. 1993;**35**(5):657-665.
- Moise KJ Jr, Belford MA. Damage control for the obstetric patient. *Surg Clin North Am*. 1997;**77**(4):835-852.
- Scalea TM. Trauma, a disease of bleeding. In: Massive Transfusion and Control of Haemorrhage in the Trauma Patient. International Trauma Anaesthesia and Critical Care Society Seminar. 1998;**1**:3-5.
- Kashuk JL, Moore EE, Millikan JS, Moore JB. Major abdominal vascular trauma: a unified approach. *J Trauma* 1982;**22**(8):672-679.
- Gentilello LM. Advances in the management of hypothermia. *Surg Clin North Am*. 1995;**75**(2):243-256.
- Rohrer MJ, Natale AM. Effect of hypothermia on the coagulation cascade. *Crit Care Med*. 1992;**20**(10):1402-1405.
- Reed RL II, Johnston TD, Hudson JD, et al. The disparity between hypothermic coagulopathy and clotting studies. *J Trauma*. 1992;**33**(3):465-470.
- Bernabei AF, Levison MA, Bender JS. The effects of hypothermia and injury severity on blood loss during trauma laparotomy. *J Trauma*. 1992;**33**(6):835-839.
- Jurkovich GJ, Greiser WB, Lutenman A, et al. Hypothermia in trauma victims: an ominous predictor of survival. *J Trauma*. 1987;**27**(9):1019-1024.
- Gentilello LM, Cobean R, Offner PJ, et al. Continuous arteriovenous re-warming; rapid reversal of hypothermia in critically ill patients. *J Trauma*. 1992;**32**(2):316-327.
- Ferrada R, Birolini D. New concepts in the management of patients with penetrating abdominal wounds. *Surg Clin North Am*. 1999;**79**(6):1331-1356.
- Hirschberg A, Walden R. Damage control for abdominal trauma. *Surg Clin North Am*. 1997;**77**(4):813-820.
- Carrillo C, Fogler RJ, Shaftan GW. Delayed gastrointestinal reconstruction following massive abdominal trauma. *J Trauma*. 1993;**34**(20):233-235.

25. Hirshberg A, Wall MJ, Mattox KL. Planned re-operation for trauma: a 2 year experience with 124 consecutive patients. *J Trauma*. 1994;37(3):365-369.
26. Poggetti RS, Moore EE, Moore FA. Balloon tamponade for bilobar transfixing hepatic gunshot wounds. *J Trauma*. 1992;33(5):694-697.
27. Beal SL. Fatal hepatic haemorrhage: an unresolved problem in the management of complex liver injury. *J Trauma*. 1990;30(2):163-169.
28. Cue JI, Cryer HG, Miller FB, Richardson JD, Polk HC Jr. Packing and planned re-exploration for hepatic and retroperitoneal haemorrhage: critical refinements of a useful technique. *J Trauma*. 1990;30(8):1001-1013.
29. Morris JA, Eddy VA, Rutherford EJ. The trauma celiotomy: the evolving concepts of damage control. *Curr Probl Surg*. 1996;33(8):611-700.
30. Reilly PM, Rotondo MF, Carpenter JP, Shaw SA, Schwab CW. Temporary vascular continuity during damage control: intraluminal shunting for proximal superior mesenteric artery injury. *J Trauma*. 1995;39(4):75-80.
31. Velmahos GC, Demetriades D, Chahwan S, Gomez H, Hanks SE, Murray JA, Asensio JA, Berne TV. Angiographic embolization for arrest of bleeding after penetrating trauma to the abdomen. *Am J Surg*. 1999;178(5):367-73.
32. Moore EE, Burch JM, Franciose RJ, Offner PJ, Biffi WL. Staged physiologic restoration and damage control surgery. *World J Surg*. 1998;22(12):1184-91.
33. Cornwell EE IIIrd, Kennedy F, Rodriguez J. The critical care of the severely injured patient - Part I. *Surg Clin North Am*. 1996;76(4):959-969.
34. Belzberg HH, Cornwell EE IIIrd, Berne TV. The critical care of the severely injured patient - Part II. *Surg Clin North Am*. 1996;76(4):971-983.
35. Martin RR, Byrne M. Postoperative care and complications of damage control surgery. *Surg Clin North Am*. 1997;77(4):929-942.
36. Hirshberg A, Mattox KL. Planned re-operation for severe trauma. *Ann Surg* 1995;222(1):3-8.
37. Hirshberg A, Stein M, Adar R. Re-operation, planned and unplanned in Damage Control Surgery. *Surg Clin North Am*. 1997;77(4):897-907.
38. Abikhaleh JA, Granchi TS, Wall MJ, Hirshberg A, Mattox KL. Prolonged packing for trauma is associated both increased morbidity and mortality. *Am Surg*. 1997;63(12):1109-1112.
39. Scalea TM, Phillips TF, Goldstein AS *et al*. Injuries missed at operation: nemesis of the trauma surgeon. *J Trauma*. 1988;28(7):962-967.
40. Hirshberg A, Wall MJ Jr, Allen MK. Causes and patterns of missed injury in trauma. *Am J Surg*. 1994;168(4):299-303.
41. Moore FA, Feliciano DV, Andrassy RJ, *et al*. Early enteral feeding compared with parenteral reduces postoperative septic complications: the result of a meta analysis. *Ann Surg*. 1992;216(2):172-83.
42. Kiyana T, Efram DT, Tantry U, Barbul A. Effect of nutritional route on colonic anastomotic healing in the rat. *J Gastrointest Surg*. 1999;3(4):441-446.
43. Reissmann P, Teoh TA, Cohen SM, Weiss EG, Noguera JS, Wexner SD. Is early oral feeding safe after elective colorectal surgery? A prospective randomised trial. *Ann Surg*. 1995;221(1):73-77.
44. Maxwell RA, Fabian TC, Croce MA, Davis KA. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe haemorrhagic shock. *J Trauma*. 1999;47(6):995-999.
45. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. *Surg Clin North Am*. 1996;76(4):833-842.
46. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. *J Surg Res*. 1976;20(5):401-404.
47. Richards WO, Scovill W, Shin B, Reed W. Acute renal failure associated with increased intra-abdominal pressure. *Ann Surg*. 1983;197(2):183-187.
48. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg*. 1984;199(1):28-30.
49. Harman PK, Kron IL, McLachlan HD, Freeland AE, Nolan SP. Elevated intra-abdominal pressure and renal function. *Ann Surg*. 1982;196(5):594-597.
50. Meldrum DR, Moore FA, Moore EE, Haenel KB, Cosgriff N, Burch JM. Cardiopulmonary hazards of perihepatic packing for major liver injuries. *Am J Surg*. 1995;170(6):537-542.
51. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary and renal effects of massively increased intra-abdominal pressure in critically ill patients. *Crit Care Med*. 1989;17(2):118-121.
52. Bongard FB, Ryan M, Dubecz S. Adverse consequences of increased intra-abdominal pressure on bowel tissue oxygen. *J Trauma*. 1995;39(3):519-525.
53. Diebel LN, Wilson RF, Dulchavsky S. Effect of increased intra-abdominal pressure on hepatic arterial portal venous and hepatic microcirculatory blood flow. *J Trauma*. 1992;33(2):279-283.
54. Diebel L, Saxe J, Dulchavsky SA. Effect of increased intra-abdominal pressure on abdominal wall blood flow. *Am Surg*. 1992;58(9):573-576.
55. Josephs LG, Este-McDonald JR, Birkett DH. Diagnostic laparoscopy increases intra-cranial pressure. *J Trauma*. 1994;36(6):815-819.
56. Bloomfield GL, Dalton JM, Sugarman HJ, *et al*. Treatment of increasing intra-cranial pressure secondary to the acute abdominal compartment syndrome in a patient with combined abdominal and head trauma. *J Trauma*. 1995;39(6):1168-1170.
57. Ridings PC, Bloomfield GL, Blocher CR. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. *J Trauma*. 1995;39(6):1071-1075.
58. Eddy V, Nuinn C, Morris JA. Abdominal Compartment Syndrome, the Nashville experience. *Surg Clin North Am*. 1997;77(4):801-812.
59. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. *J Trauma* 1998;44(6):1016-1023.
60. Ivatury RR, Diebel L, Porter JM, Simon RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. *Surg Clin North Am*. 1997;77(4):783-800.
61. Bellamy RF. The causes of death in conventional land warfare: implications for combat casualty care research. *Mil Med*. 1984;149(2):55-62.
62. Hirshberg A, Mattox KL. Damage control in trauma surgery. *Br J Surg*. 1993;80(12):1501-1502.
63. Moore EE. Staged laparotomy for the hypothermia, acidosis and coagulopathy syndrome. *Am J Surg*. 1996;172(5):405-10.