Damage control surgery in the abdomen: An approach for the management of severe injured patients

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Abstract  Damage control is well established as a potentially life-saving procedure in a few selected critically injured patients. In these patients the ‘lethal triad’ of hypothermia, acidosis, and coagulopathy is presented as a vicious cycle that often cannot be interrupted and which marks the limit of the patient’s ability to cope with the physiological consequences of injury. The principles of damage control have led to improved survival and to stopped bleeding until the physiologic derangement has been restored and the patient could undergo a prolong operation for definitive repair. Although morbidity is remaining high, it is acceptable if it comes in exchange for improved survival. There are five critical decision-making stages of damage control: I, patient selection and decision to perform damage control; II, operation and intraoperative reassessment of laparotomy; III, resuscitation in the intensive care unit; IV, definitive procedures after returning to the operating room; and V, abdominal wall reconstruction. The purpose of this article is to review the physiology of the components of the ‘lethal triad’, the indication and principles of abdominal damage control of trauma patients, the reoperation time, and the pathophysiology of abdominal compartment syndrome.

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Introduction

Damage control is well recognized as a surgical strategy that sacrifices the completeness of the immediate repair in order adequately to address the combined physiological impact of trauma and surgery. This term is derived from the US Navy and describes the capacity of a ship to absorb damage and maintain mission integrity.

Damage control surgery can be defined as a series of operations which are performed in order to accomplish definitive repair of abdominal injuries in accordance with the patient’s physiologic tolerance. Trauma surgeons focus more on the physiological reserve of patient rather than the anatomy of the lesions. Surgical techniques are focused on hemorrhage and contamination control to stop bleeding and control intestinal, biliary, or urinary leak into the abdominal cavity. Obviously, patients selection is crucial because patients with relatively simple abdominal injuries should not undergo unnecessary procedures. Optimal results are also achieved by the early identification of patients who require damage control. The clinical manifestations of hemorrhagic instability, hypotension, tachycardia, tachypnea, and altered mental status are indications for the potential need of damage control.

Another challenge for the surgeon in abdominal damage control is the complexity of injury and the high rate of patients’ mortality. The damage control approach is also associated with complications such as the most notably intra-abdominal hypertension or the abdominal compartment syndrome (ACS).

Currently, damage control is one of the most topical areas in trauma management. Not only do principles of damage control apply to the abdomen, but for many others body regions. This study reviewed the physiology of the components of the ‘lethal triad’, the damage control principles and indications, the time of reoperation, as well as the pathophysiology of ACS in trauma patients. The English-language literature about damage control surgery in the abdomen was identified using Medline, and additional cited works were not detected in the initial search obtained. Articles reporting on prospective and retrospective comparisons and case reports were included.

Historical retrospective

While there are many different components to damage control, abdominal packing has historically been the foundation principle of damage control and was first reported in the early 20th century by Pringle. His technique was modified by Halstead, who in 1913 recommended placing non-adhesive rubber sheets between the packs and the liver. This technique was used until the Second World War but then fell out of favor perhaps it was used only when nothing else worked.

In 1955, Madding wrote that temporary packs may be effective for checking bleeding and twenty years later, Lucas and Lederwood reported on three of their patients with major liver injuries, who were packed and survived as a part of a large series of evaluation of over 600 patients with liver injuries. These good results from liver packing were supported by Feliciano in 1981; while in 1983, Stone et al. described a stepwise operative management including initial abandonment of laparotomy, intra-abdominal packing, correction of coagulopathy and reoperation for definitive surgical repair. In 1993, Rotondo et al. introduced the term ‘damage control’ and detailed a standardized three-phase approach.

Since this date there has been a rise in the number of publications dealing with concepts of damage control surgery.

Physiology and pathophysiology of trauma patients

Trauma patients are often admitted to hospital with hypotension, hypothermia or both. The most important indications for a staged operative procedure are progressive coagulation and metabolic acidosis. Disturbances in the patients’ molecular, cellular, and hemodynamic equilibrium are associated with the triad of acidosis, hypothermia, and coagulopathy. Hypothermia is common in trauma patients; it results from environmental exposure, impaired thermoregulation in the intoxicated or neurologically impaired patient, resuscitation with unwarmed fluids, and accelerated heat loss. It is associated with sympathetic a-adrenergic overdrive, peripheral vasoconstriction, end-organ hypoperfusion, conversion from aerobic to anaerobic metabolism and metabolic acidosis predisposing to disorder of the coagulation cascade in cases of aggressive fluid resuscitation with normal saline. In trauma patients, a temperature less than 32 °C is associated with 100% mortality, and any decrease in temperature below 35 °C is a poor prognostic sign. The major negative effect of hypothermia in the trauma patient is coagulopathy. Hypothermia may also cause cardiac dysfunction with ventricular dysrhythmias and decreased cardiac output, poorly tolerated in a patient with diminished perfusion and oxygen delivery. Hirshberg et al. using a computer simulation of hypothermia during trauma laparotomy reported that in the operating room the critically injured patient’s heat balance is dominated by increased heat loss from the exposed peritoneal surface; while a closed abdominal cavity is associated with a decreased heat loss despite of bleeding. Finally, he supported that damage control laparotomy should be limited to 60–90 min.

The incidence of early coagulation abnormalities after trauma is high and they are independent predictors of mortality. An initial normal prothrombin time (PT) increases the adjusted odds of dying by 35% and an initial abnormal activated partial thromboplastin time (aPTT) increases the adjusted odds of dying by 326%. Dilution of coagulation factors and hypothermia are the most frequent preventable causes of coagulopathy and it is very important to keep the patient warm and avoid over-resuscitation. The decreased temperature results in cold hemoglobin which cannot release oxygen in tissues as readily as normothermic hemoglobin. The decreased temperature also results in a decrease in the rate of the cascade reaction and a decrease in the production of clotting factors. On the other hand, massive transfusion results in dilution of the clotting factors and thrombocytopenia.
The shift from aerobic to anaerobic metabolism which results in metabolic acidosis is caused by the increased production of lactic acid. The acidosis contributes to coagulopathic bleeding resulting in a vicious cycle of continued blood loss, coagulopathy, hypovolemia, and worsening acidosis. The acidosis also puts a large burden on the respiratory system. The degree of acidosis is an accurate sign of acidosis requires control of hemorrhage, optimization of oxygen delivery, and continued ventilatory support.

Indications and principles of damage control surgery

There are five critical decision-making stages of damage control surgery. The controversial aspects of each of these stages are addressed in the sequence encountered in the critically injured patient (Table 1).

Stage I: Patient selection and decision to perform damage control

This emphasizes to early recognition of the potential need for damage control surgery. The principles of this stage include rapid transport to hospital and in the hospital’s resuscitation room emphasis is placed on early decision making to facilitate hemorrhage control. The surgeon’s purpose in the preoperative phase is to think early surgery in the bleeding patient. Patient selection is crucial as patients with relatively simple abdominal injuries should not undergo unnecessary procedures. Hemodynamic instability manifested by hypotension, tachycardia and tachypnea, coagulopathy (PT >19 s or aPTT >60 s), and/or hypothermia are important indications for the damage control approach.4,5

Stage II: Operation and intraoperative reassessment of laparotomy

The second stage is consisted of control of hemorrhage and contamination with rapid techniques of intra-abdominal packing, following by temporary abdominal closure. The abdomen is initially packed in all four quadrants. Initial control of hemorrhage using this technique should take less than 5 min. After control of hemorrhage comes control of contamination. The intestine is inspected from the liga-ment of Treitz to the rectum, and contamination is controlled initially with non-crushing bowel clamps, simple suture, or umbilical tapes. Once the entire bowel has been inspected, gastrointestinal staplers may be used to resect devitalized areas. The packs are then removed, beginning in the quadrant farthest away from the greatest amount of hemorrhage. If ongoing non-surgical bleeding continues, the packs are replaced and may be left in the abdomen. Surgical bleeding is controlled with suture. Vascular injuries may be treated with vascular shunts, ligation, or extra-anatomic bypass rather than with definitive repair.42,43 Ureter injuries may also be treated with temporary shunting during damage control.44 Before closure, the entire gastrointestinal tract is rapidly inspected one more time for further injuries. No attempt is made to restore gastrointestinal continuity.

Stage III: Physiologic restoration in the intensive care unit (ICU)

The patients, after the initial operation, control of hemorrhage, and contamination are transferred to ICU. Priorities in the ICU focus on restoring the global physiologic status of the patient. The patients’ management consists of rewarmin, correction of coagulopathy, reversal of acidosis, minimized crystalloid transfusion, and decision of blood transfuse.45,46 Endpoints include a systemic lactate concentration of less than 2.5 mmol/l, base deficit greater than –4 mmol/l, core temperature greater than 35 °C, hemoglobin level >10 g/dl and hematocrit >30%.10

Stage IV: Return to operating room for definitive procedures

This stage consists of removal of packing, definitive repair of the abdominal injuries, and closure when the patient has been fully resuscitated, is warm, and the coagulopathy has been restored; usually 24–48 h after the initial intervention.47 The patients are returned to operating room for clotted blood, and fluid collections, debridement of dead tissue, reconstruction of digestive tract injuries, and jejun-al feeding access with gastric decompression. Occasionally, recurrent bleeding necessitates repacking.

Stage V: Abdominal wall closure/reconstruction

Management of the open abdomen has become a challenge for surgeons performing damage control surgery. Abdominal closure is performed when edema has resolved enough to allow closure without tension. However, mobilization of such interstitial fluid may not be complete and temporary closure may still be necessary. Definitive closure should be achieved as soon as possible. Preferred wall closure techniques include towel clip closure and running nylon suture when the wound edges can be approximated; otherwise Bogota bag, zippers, vacuum-assisted closure (VAC), placement of mesh (absorbable and non-absorbable) or polytetrafluoroethylene patches, and sandwich (Vaspac) techniques should be used or even strategies using native tissue (skin-only closure, component separation technique).48–50

The use of any sutured prosthetic material has led to abdominal wall hernias and intestinal fistulas.51 The significant inflammatory response to prosthesis and sutures in combination with the trauma to the fascia produced by the initial suturing may delay resolution of inflammation and edema and may make later definitive closure of the fascia more difficult and less secure.51 Miller et al.52 used a wide variety of techniques for temporary abdominal coverage and a variety of synthetic patches sutured to the fascial edges and reported that the delayed primary fascial closure before 8 days was associated with the best outcomes. Guy et al.53 reported their excellent results with human acellular dermal matrix (AlloDerm; Life-Cell, Branchburg, NJ) as a one-staged fascial closure with bipedicle flaps in nine patients with ACS.
The vacuum-pack technique is a simple and quick technique that maintains a sterile environment with a controlled egress of fluid that is easily quantifiable. In an attempt to avoid loss of abdominal domain, a polyurethane sponge (V.A.C.; KCI International, San Antonio, TX) has substituted for the surgical towel. Many authors have reported that closure rate was 88%–100% and the closure time was 4–21 days in patients who underwent VAC technique or modifications of it. Recently, the Wittmann Patch (Starsurgical, Inc, Burlington, WI) has shown promise in achieving abdominal wall closure with the avoidance of both the large hernias and the fistulas.

Timing for reoperation is governed by the indications for damage control; the spectrum of injuries; as well as the physiologic response of the patient in the ICU. The optimal time of planned reoperation varies from 12 to 96 h, but is usually between 24 and 48 h after the initial operation. At a minimum, the planned reoperation should be performed

<table>
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<th>Table 1</th>
<th>Stages of damage control surgery</th>
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<td>Stage I:</td>
<td>Patient selection and decision to perform damage control</td>
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| Clinical and laboratory indications for the implementation of damage control surgery | - Penetrating trauma or complex and major vascular injuries
- Hemodynamic instability
- Systolic BP < 70 mmHg
- Tachycardia, dysrhythmias
- Weak or non-palpable carotid pulses
- Compromised ventilation
- Coagulopathy (PT > 19 s, aPTT > 60 s)
- Hypothermia < 34 °C
- Acidosis: pH < 7.20
- Inability to control bleeding or transfusion > 10 units of PRBC
- Operative time > 90 min
- Associated life-threatening extra-abdominal injury |
| Stage II: | Operation and intraoperative reassessment of laparotomy with temporary abdominal wall closure |
| Stop bleeding and prevent contamination | - Control of hemorrhage
- Ligation, suturing or temporal shunting of vascular injuries
- Packing of liver injuries
- Splenectomy in the presence of splenic injury
- Control and prevention of contamination
- Suturing or stapling visceral injuries
- Resection of non-viable bowel
- Draining of pancreatic injuries
- Shunting of exteriorisation of ureteric injuries |
| Stage III: | Physiologic restoration in the intensive care unit (ICU) |
| Correction of hypothermia, acidosis and coagulopathy | - Reverse hypovolemia-resuscitation with fluid and blood products
- Rewarming
- Reverse coagulopathy using FFP, PLT, whole blood, cryoprecipitate, vitamin K, consider use of rVIIa
- Reduce complications (ACS, ARDS, DVT, peptic ulceration, infection, pulmonary embolism) |
| Stage IV: | Return to operating room for definitive procedures |
| Definitive repair and second look laparotomy for missed injuries | - Look for missed injuries
- Debridement of dead tissue
- Reconstruction of digestive tract injuries
- Removal of shunts and definitive repair of vascular injuries
- Removal of packing
- Jejunal feeding access (optional) |
| Stage V: | Abdominal wall closure/reconstruction |
| | - When possible |

PRBC: packed red blood cells; FFP: fresh frozen plasma; PLT: platelets; DVT: deep venous thrombosis; rVIIa: recombinant activated factor VII; ARDS: acute respiratory distress syndrome.
when the patient is normothermic (>36 °C) and effective coagulation has been restored. Additionally, restoration of oxygen transport should be achieved. Improvement of base deficit, clearance of serum lactate, and improvement in other physiologic parameters serve as markers of completed resuscitation. 39, 41, 59, 60 A lactate level below the 4 mmol/l is presently one of the best indicators of return of tissue perfusion, in conjunction with a base excess greater than −4 mmol/l and a normalized coagulation profile. These endpoints can usually be attained within 36 h. 47 It is important to perform only procedures that the patient can tolerate.

Ongoing uncontrolled intra-abdominal bleeding and/or the development of ACS require an unplanned reoperation. 47, 61 In general, reoperation is indicated in the normothermic patient who bleeds more than two units of packed cells/hour. 47 Alternatively, selective embolization for major hepatic or pelvic bleeding in the angiography suite can be life saving. If the unplanned reoperation is being performed for ACS secondary to bleeding, re-exploration should be done in the operating room. ACS without ongoing bleeding may be addressed by decompressing the abdomen in the ICU and using one of the temporary closures. The mortality rate associated with urgent reoperations following a damage control operation is 70%, 8 being much higher than the mortality seen in patients who undergo reoperation after a definitive laparotomy. 62 Generally, the decision should always be made by the surgeon who performed the initial procedure.

During a planned reoperation, definitive procedures should be performed before pack removal, as the latter may induce bleeding requiring repacking, and so prevent completion of the intended operation. Such procedures include restoration of gastrointestinal continuity, colostomy formation, solid organ debridement and placement of enteral feeding tubes.

**Abdominal compartment syndrome (ACS)**

This syndrome is defined as increased pressure within the abdominal cavity. Patients managed by damage control laparotomy are at high risk of intra-abdominal hypertension and ACS. 63 It is reported to occur in as many as 14% of patients who undergo laparotomy and who are found to have serious intestinal or hollow organ injury. 64 ACS may still occur because of increasing visceral swelling, expanding hematoma, and the use of abdominal packs. 65, 66 In patients with severe abdominal injuries, the predisposing factors to increase the intra-abdominal pressure are shown in Table 2.

### Table 2  Factors predisposing to increased intra-abdominal pressure in damage control surgery

<table>
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<tr>
<td>Severe abdominal injuries</td>
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<td>Spillage of intestinal content</td>
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<tr>
<td>Primary fascial closure under tension</td>
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<tr>
<td>Intra-abdominal packing for coagulation</td>
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<tr>
<td>Massive transfusion with bowel edema and distension</td>
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<tr>
<td>Failure to control the bleeding which results in increased acidosis and coagulopathy</td>
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Optimal outcome from ACS requires prevention and/or early recognition. 67 ACS may be recognized by the presence of a tensely distended abdomen, elevated peak airway pressures, inadequate ventilation, and hypoxia. However, these findings are relatively non-specific and can also be associated with other pathologies than intra-abdominal hypertension and ACS. 61 The acute expansion of the intra-abdominal contents affect the cardiovascular, renal, and pulmonary systems. 61 A pressure greater than 30 mmHg is associated with oliguria due to a decreased renal blood flow associated with increased renal venous pressure and an increased in renal vascular resistance. 68 ACS is also associated with a 30–40% decrease in cardiac output related to decreased venous return and an increase in systemic vascular resistance. 69 An intra-abdominal pressure of 25 mm Hg can also cause elevation in intracranial pressure by increasing central venous pressure. 70 ACS has profound effects on pulmonary function, with progressive hypoxemia and CO₂ retention, or a requirement for very high peak airway pressures to maintain adequate tidal volume.

Intra-abdominal pressure can be monitored and measured by direct and indirect methods. For direct measurement, a catheter placed in the peritoneal cavity is attached to a saline manometer or a pressure transducer. For indirect measurement, two major techniques are used, namely intragastric and intravascular monitoring, 71 although the former may be less reliable than the latter. A pressure, greater than 30 mmHg, requires emergency re-operation and decompression.

### Conclusions

Damage control surgery is a vital part of the management of a seriously injured patient. It has been shown to reduce mortality rates significantly when applied to injuries that were previously not survivable; in a select subgroup of patients. 72 It is also most effective when the decision to pursue this course is made early, before the patient’s physiology becomes completely exhausted. The central principle of damage control surgery is that patients are more likely to die from the ‘lethal triad’ of hypothermia, coagulopathy and metabolic acidosis than from a failure to complete operative repairs. The goals of the abbreviated laparotomy are to identify injuries, control hemorrhage, and control contamination. Efforts should not be made to restore bowel continuity or do definitive procedures, but should focus on getting the patient to the ICU for resuscitation. Reoperation is performed for ongoing bleeding, abdominal compartment syndrome, or definitive reconstruction when the patient is adequately resuscitated. With an organized approach, damage control can lead to improved patient survival.

### Conflict of interest

None.

### Funding

None.

### Ethical approval

Not required.