Chapter 4
Principles and Philosophy of Damage Control Surgery

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This technique of initial abortion of laparotomy, establishment of intra-abdominal pack tamponade, and then completion of the surgical procedure once coagulation has returned to an acceptable level has proven to be lifesaving in previously non-salvageable situations.

– Harlan Stone, 1983 [1]

The term "damage control" originated within the US Navy and referred to doing the minimum amount necessary with limited resources in the face of a catastrophe to keep a vessel afloat until help arrived. This concept was then applied to trauma surgery in response to advancements in the understanding of hemorrhagic shock. Specifically, the potentiating effects of hypothermia, acidosis, and coagulopathy, referred to as the bloody viscous cycle [2], are now understood to eventuate in exsanguination that cannot be stopped by mechanical surgical interventions. The high mortality associated with the development of the bloody viscous cycle led surgeons to investigate alternatives to lengthy, complicated initial operations in exsanguinating trauma patients. From this experimentation arose the damage control approach, which addresses the problem of nonsurgical coagulopathy by

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performing an abbreviated initial operation, centered upon rapidly controlling immediately life-threatening injuries, followed by goal-directed resuscitation in the controlled setting of the intensive care unit (ICU), and after temporarily packing and closing body cavities to limit further blood, protein, and heat loss. Only after restoration of metabolic and coagulation integrity does the patient return to the operating room for definitive repair of injuries. Originally described in the setting of abdominal trauma, the damage control approach has now been applied to a variety of body regions, including thoracic, orthopedic, and neurosurgical; many of these specific areas are presented in further detail throughout this text. Most recently, the damage control approach has been extended beyond the realm of trauma into that of emergency general surgery [3]. The success of the damage control approach brought forth new challenges; the management of open, edematous body cavities, particularly the open abdomen, has become a necessary topic of discussion as a direct consequence of survival following damage control surgery. This chapter will review the indications for, technique of, and sequelae of damage control surgery.

4.1 What Is Damage Control Surgery?

Broadly defined, the damage control approach begins with an abbreviated initial surgery in the face of profound hemorrhagic shock, the goal of which is to preserve life. This philosophy is contingent upon an understanding of the negative impact that metabolic failure has on the ability of the trauma patient to tolerate further surgical insults. Specifically, hemorrhage, in conjunction with radiant heat loss from exposed body cavities, and compounded by additional tissue trauma from further

dissection, results in acidosis, hypothermia, and coagulopathy. These three basic factors constitute a "bloody viscous cycle," positively feeding back upon one another and eventuating in mortality from exsanguination. Any further operative maneuvers in this setting serve only to exacerbate the cycle, as mechanical bleeding is not the primary source of the problem. Accordingly, the damage control approach involves doing the least amount of operating to save the patient's life and temporarily close body cavities. Surgical hemorrhage is stopped via packing, ligation, or shunting, and gross contamination from the gastrointestinal tract is addressed by rapid over sewing or stapling. Anything more complicated than this, such as vascular anastomosis or bypass grafting, intestinal resection, anastomosis, or enteral feeding access, should not be undertaken during the initial operation. The damage control approach is thus predicated on the somewhat counterintuitive surgical principle of "less is more." Because the temptation in the face of bleeding and shock is to keep operating, adoption of the damage control approach requires both self-control, resisting the urge to expose the unstable trauma patient to additional unhelpful operative insults, and the perspective to address the overall clinical scenario rather than individual injuries. Definitive repair of injuries and fascial closure is forgone for hours to days until after a period of resuscitation in the ICU.

4.2 What Is the History of Damage Control Surgery?

The surgical approach that has recently been termed damage control has existed for over 100 years. Both Pringle [4] and Halstead [5] outlined the utility of packing for management of hepatic trauma in the early twentieth century. However, as

surgical techniques improved, packing for control of hepatic hemorrhage fell out of favor and was almost universally abandoned following World War II. One major concern at the time involved infection from retained packs, and surgeons such as Madding emphatically asserted that abdominal packs were to be removed prior to the end of the operation [6]. By the early 1970s, advances in transport of trauma patients increased the number of patients presenting to major trauma centers who were exsanguinating but salvageable. Accordingly, renewed interest in temporary packing in highly select patients arose. Reports of success began to surface in small groups of patients using hepatic packing specifically. In 1976, Lucas and Ledgerwood described a prospective 5-year evaluation of 637 patients treated for severe liver injury [7]. Packs were inserted in only three patients, all of whom survived. At the 1979 meeting of the Southwestern Surgical Congress, our group reported that over 80 % of deaths from liver trauma were due to uncontrollable, nonsurgical hemorrhage, strengthening the concept of post-injury coagulopathy and the merits of abbreviated laparotomy with packing [2]. Calne et al. [8] and Feliciano et al. [9] soon followed with case series of nearly 100 % survival following hepatic packing.

A landmark study in the evolution of the contemporary damage control sequence was performed by Stone et al. in 1983 [1]. Trauma patients who developed a major coagulopathy during laparotomy were managed either by completion of the procedure in detail or by abortion of the procedure, intra-abdominal packing, and return for definitive repair once coagulation status had normalized. Eleven of 17 (64.7 %) patients managed with abbreviated laparotomy survived, whereas only 1 of 14 (7.1 %) patients with definitive repair during the initial procedure lived. From these studies as well as their own experience, Rotondo and Schwab et al. popularized the term “damage control” within the discipline of trauma surgery [10]. Shortly after, Moore detailed the five classic stages in our current damage control sequence [11].

4.3 What Are the Stages of Damage Control Surgery?

Once initiated, the damage control sequence follows specific stages that begin and end at predetermined time points. Transition through stages is triggered by the overall hemodynamic and metabolic states of the patient.

Stage I, Patient Selection for Abbreviated Laparotomy: The initial step in the damage control sequence involves rapid identification of patients who will benefit from this approach. Although the decision to perform damage control surgery is often made intraoperatively, certain risk factors are recognized to increase the likelihood of damage control and thus may be used to anticipate this decision in the preoperative setting and prepare accordingly. These parameters are summarized in Table 4.1. In general, hemodynamic instability with a presumed thoracic, abdominal, pelvic, or extremity vascular injury should initiate preparation for a damage control approach. Patients who survive emergency department thoracotomy and are transported to the operative room are also included in this subset. Patients who require multiple emergency procedures (e.g., craniotomy and laparotomy) should also be considered for damage control.

Table 4.1 Cases in which damage control should be considered preoperatively

High-energy blunt torso trauma
Multiple torso penetrations
Hemodynamic instability
Presenting coagulopathy and/or hypothermia
Major abdominal vascular injury with multiple visceral injuries
Multifocal or multi-cavity exsanguination with visceral injuries
Multiregional injury with competing priorities

When the decision to pursue damage control surgery has been made preoperatively, minimal additional evaluation in the emergency department is necessary prior to transporting the patient to the operating room. Time spent in the emergency room should be limited to establishment of a definitive airway and intravenous access and evaluation for both pneumothorax/massive hemothorax (via physical exam with or without chest radiography) and pericardial hemorrhage (via focused examination of the abdomen for trauma).

When made intraoperatively, the decision to perform damage control surgery is based broadly upon the six variables outlined in Table 4.2. After initial control of major bleeding and gastrointestinal contamination, an overall assessment of the patient's metabolic and coagulation integrity is made by querying the following parameters: (1) hemodynamic status, (2) metabolic status, (3) temperature, (4) coagulation status, and (5) clinical assessment of nonsurgical bleeding. Hypotension from hemorrhagic shock is a clear indication to perform damage control. However, more subtle indicators of shock, such as increasing vasopressor requirements, hypocapnia, and metabolic acidosis, should all be evaluated. This task requires frequent and clear communication with the anesthesiology team. Patient temperature and coagulation status should be monitored frequently. Routine coagulation parameters, such as the activated partial thromboplastin time, prothrombin time, and international normalized ratio, are insensitive in detecting coagulopathy because they measure only the earliest stages of clot formation. Furthermore, results of these tests are typically not available immediately. For these reasons, we prefer point-of-care thromboelastography (TEG) [12]. Finally, the astute clinician will recognize the development of nonsurgical bleeding as evidenced by hemorrhage from raw surfaces, needle holes, and intravenous catheter sites. These findings signify the development of profound coagulopathy and mandate damage control surgery.

Table 4.2 Cases in which damage control should be considered intraoperatively

Indication	Example
Inability to achieve hemostasis secondary to a recalcitrant coagulopathy	Massive transfusion with disseminated intravascular coagulation
Inaccessible major venous injury	Retrohepatic vena caval disruption
Anticipated need for a time-consuming procedure	Pancreaticoduodenectomy
Demand for nonoperative control of extra-abdominal life-threatening injuries	Ruptured pelvic fracture hematoma requiring selective arterial embolization
Inability to approximate the abdominal incision due to extensive splanchnic reperfusion-induced visceral edema	Protracted shock with massive fluid administration
Desire to reassess abdominal contents	Extensive mesenteric venous injury

Although the technical aspects of the abridged laparotomy are dictated by the injury pattern, in general, the initial damage control operation is divided into three sequential steps: (1) control of hemorrhage, (2) control of gastrointestinal contamination, and (3) temporary closure. Hemorrhage may be controlled by a variety of maneuvers, including packing, ligation, and shunting. In general, venous bleeding may be controlled by pressure pack tamponade using laparotomy pads. The packs are then left in place for transport to the ICU and removal at reoperation. By contrast, arterial or portal venous bleeding requires control with suture repair, ligation, or shunting. Under the extreme conditions of damage control, ligation of almost any vessel is compatible with life. If hemorrhage control can only be accomplished by manual pressure (e.g., sponge stick tamponade of an inferior vena cava injury prior to obtaining proximal and distal control), the surgeon should do so in order to allow for crystalloid and blood product resuscitation prior to exposing the patient to

further hemorrhage. In extreme circumstances, control of hemorrhage may be possible only by cross-clamping of the abdominal aorta. Patients may be transported to the ICU with the clamp in place, provided that it is moved to an infrarenal location and that the cross-clamp time is monitored carefully, with flow being reestablished intermittently by transient release of the clamp. Control of gastrointestinal perforation is accomplished by rapid closure using either suture or staples. Although this phase of the operation should proceed rapidly, it should not be haphazard. Poorly placed sutures in tenuous areas such as the esophagus or duodenum may lead to catastrophic consequences subsequently provided that the patient survives, and the surgeon should take the extra few seconds to ensure proper suture placement through viable tissue and complete wound closure.

During the initial operation, efforts should be made to minimize both hypothermia and coagulopathy. Techniques to both prevent and reverse hypothermia include increasing the operating room temperature to >30 °C, infusing warmed fluids, covering body areas not in the operative field with warming devices, and using warmed irrigation fluid. Restoration of clotting function should be goal-directed using serial TEG tracings. When this method is not possible, blood component therapy should be replaced with a ratio of RBC/FFP/plts/cryoprecipitate of approximately 10:5:1:1 [13].

Stage II, Reassessment for Hemorrhage Control: An often underemphasized issue in the damage control literature involves the decision of when to transfer the patient from the operating room to the ICU. Although prompt transfer is both rational and cost-effective, premature departure with ongoing mechanical bleeding may lead to an inexorable bloody viscous cycle in the ICU. Finally, there are select cases in which packing may not be necessary and fascial closure is possible once coagulation integrity has been restored.

In cases of ongoing bleeding, a determination that the current amount of hemorrhage is “acceptable” as being nonsurgical

must be made. Although this decision may be guided by the development of obvious nonsurgical bleeding (e.g., from intravenous sites), it is the authors' contention that this decision is often made too late and the operation continued unnecessarily. One technique to aid in this determination is temporary (i.e., 20–30 min) abdominal closure with towel clips (Fig. 4.1), followed by reopening and assessment of the amount of residual intra-abdominal hemorrhage. During this time, collective (i.e., surgery, anesthesia, blood bank) efforts are focused on normalizing temperature, acid–base status, and coagulopathy and critically reevaluating patient salvageability. One important exception to this practice is an isolated pelvic fracture with arterial hemorrhage, which warrants immediate angiographic intervention.

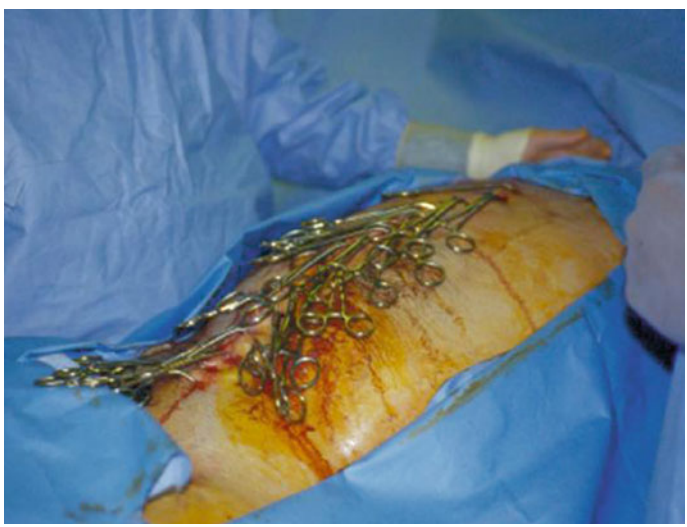

Fig. 4.1 Towel-clipped abdomen

After the brief period of towel clamp closure, the abdomen is examined for residual hemorrhage. Packs, except those successfully tamponading major hepatic venous injuries, are withdrawn sequentially to determine both efficacy and necessity (there are cases, although relatively infrequent, in which the packing can be completely removed and the fascia closed without incident). At this time, a search is undertaken for both residual mechanical bleeding and missed gastrointestinal injuries. In general, if more blood is present in the abdominal cavity than has been transfused during the period of towel clamping, surgical bleeding still exists and should be investigated. By contrast, bleeding from coagulopathy will only worsen if the operation is continued, and in this case, the patient should be closed temporarily and transported to the ICU, at which time damage control stage III begins. Although it is often uncomfortable to stop operating on a patient who is still bleeding, in the setting of profound shock and coagulopathy, this decision is usually lifesaving.

The final step in damage control stage II involves temporary wound closure. In the case of thoracic damage control via a lateral thoracotomy incision, the most rapid and simple technique involves stapled closure of skin only over tube thoracostomy drainage. In the case of a sternotomy, application of an adhesive, translucent dressing over the wound is sufficient. More options exist for laparotomy wound closure, ranging from towel clipping to achieve further tamponade to insertion of a translucent plastic dressing over the abdominal contents, over which drains may be placed and connected to a continuous suction to manage fluid efflux and monitor ongoing hemorrhage (Fig. 4.2). Disadvantages of the towel clip method include an increased likelihood of abdominal compartment syndrome (ACS) and inability to visualize the underlying bowel. Disadvantages of the latter method involve promotion of ongoing hemorrhage from the closed suction drains. None of these techniques has been proven superior to the others, and the overarching principle of rapid, controlled closure should not be overshadowed by the specifics of the dressing.

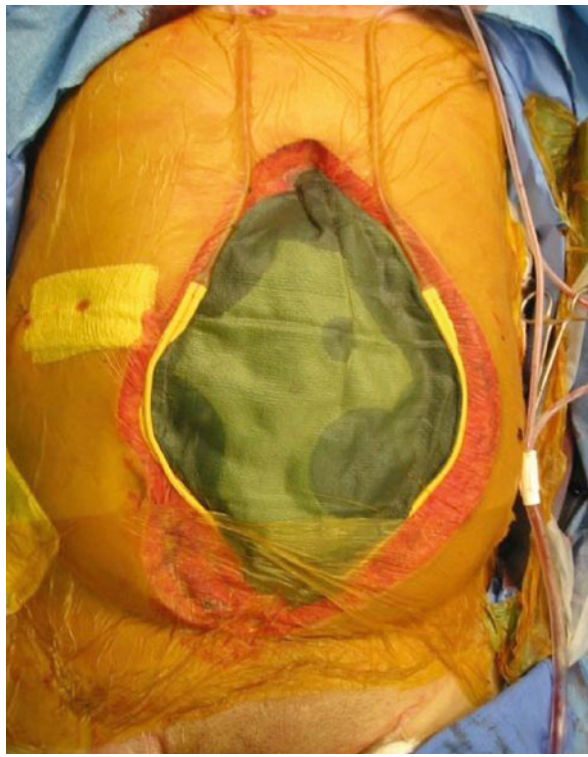

Fig. 4.2 Temporary abdominal closure with green towel, Jackson Pratt drains, and Betadine-impregnated adhesive dressing

Stage III, Physiologic Restoration in the ICU: The patient for whom a damage control approach has been selected usually arrives to the ICU in shock. As such, damage control stage III is centered on resuscitation. Hypothermia and coagulopathy are reversed aggressively. We favor goal-directed restoration of both enzymatic and platelet clotting function using serial TEG tracings. Although the optimal hemoglobin concentration during

resuscitation from hemorrhagic shock remains unknown, a concentration of 8–10 g/dL is reasonable, and the decision to transfuse pRBCs should be based primarily upon clinical parameters (e.g., hemodynamic instability) and estimated ongoing blood loss from open wounds and drains. Many endpoints of resuscitation, such as serum lactate concentration, base deficit, and venous hemoglobin oxygen saturation, have been debated and are likely equivalent in terms of monitoring progress. Regardless of which marker is chosen, resuscitation should be guided by serial determinations and stop when normalization has occurred. Furthermore, utilization of multiple markers should be employed, such that the overall clinical picture is given preference over any one laboratory value. This strategy minimizes the possibility of misinterpreting isolated values and either terminating resuscitation prematurely or continuing with unnecessary and potentially deleterious volume expansion. A worsening base deficit in the otherwise resuscitated patient is usually due to a metabolic acidosis from large volume infusion of chloride-rich fluids (e.g., normal saline). Calculation of both the anion gap and serum chloride concentration will aid in this determination; a non-anion gap, hyperchloremic metabolic acidosis is characteristic. A worsening or persistently elevated lactate concentration in the otherwise resuscitated patient may be due to impaired hepatic clearance. Determination of a lactate/pyruvate ratio will elucidate this cause.

During damage control stage III, all organ systems should be supported, and no attempts at either liberation from mechanical ventilation or institution of enteral nutrition should be made until the patient is fully resuscitated. However, once resuscitated, institution of enteral nutrition (provided that the gastrointestinal tract is in continuity) is associated with improved subsequent fascial closure rates, morbidity, and mortality in open abdomen patients without associated intestinal injury and equivalent outcomes in those patients with associated intestinal injury [14]. It is thus the authors' practice to institute enteral

nutrition in the resuscitated damage control patient with an open abdomen.

Once the patient is warmed, and both enzymatic and platelet coagulopathy and metabolic derangements have been corrected, consideration is given to progression to damage control stage IV. It is important to recognize that, in contrast to the initial operation of damage control stage I, the reoperation of damage control stage IV is non-emergent. As such, the operation should occur after ensuring availability of blood products, personnel, and equipment. Abundant data now exists documenting the safety of maintaining intracorporeal laparotomy pads, indwelling vascular shunts, and gastrointestinal discontinuity for hours to days. Furthermore, time should be taken during damage control stage III to conduct a thorough evaluation for associated injuries that may have been overlooked heretofore.

Two instances in which consideration should be given to earlier return to the operation room warrant discussion. The first involves concern for ongoing surgical bleeding. Although differentiation from diffuse coagulopathy is often difficult, Morris et al. proposed indications for emergent reoperation during damage control phase III: for blunt trauma, normothermia with a rate of hemorrhage of >2 units pRBC per hour and, for penetrating trauma, either hypothermia with a hemorrhage rate of >15 units pRBC per hour or normothermia with a hemorrhage rate of >2 units pRBC per hour [15]. The second instance in which reoperation should be considered earlier is when the viability of bowel is in question, as is the case following ligation of a major mesenteric vein such as the portal or superior mesenteric. Furthermore, any patient who remains acidotic following correction of temperature, anemia, and coagulation status should be reexplored early with the specific concern of intestinal necrosis.

Stage IV, Return to the Operating Room for Definitive Procedures: At planned reoperation, intra-abdominal packing is removed, definitive vascular and intestinal tract repair is

accomplished, a thorough exploration for missed injuries is undertaken, and fascial and skin closure may be performed provided that there is adequate laxity of the anterior abdominal wall tissues, and risk factors for the development of ACS are absent (discussed below). Packing should be removed carefully and after wetting to prevent dislodgement of formed clot. Persistent venous hemorrhage may necessitate repacking and a planned third operation.

Both options and techniques for definitive repair of specific injuries are discussed elsewhere in this text. Once definitive repairs have been achieved, and additional missed injuries have been excluded, a decision regarding fascial closure is made. In certain instances abdominal wall and bowel wall edema is so pronounced that fascial closure is obviously impossible. In the remainder of cases, consideration is given to both the anticipated volume of postoperative fluid resuscitation and the amount of physiologic derangement caused by fascial closure. One useful test involves temporarily re-approximating the fascia with towel clips and monitoring the patient's airway pressure. A steep rise in either peak or mean airway pressures signifies a high likelihood of ACS following fascial closure.

Patients in whom definitive fascial closure is achieved following damage control phase IV must be monitored aggressively for the development of ACS. Intra-abdominal hypertension leading to ACS is a particularly devastating complication of damage control surgery with a high associated morbidity and mortality [16]. The pathophysiology of ACS involves a progressive increase in abdominal pressure due to any combination of diminished abdominal wall compliance, increased intraluminal intestinal contents, increased intraperitoneal fluid, and increased tissue edema. Increases in abdominal pressure eventually become sufficient to impede venous return from both abdominal viscera (resulting in intestinal ischemia) and the inferior vena cava (causing decreased filling pressures and obstructive shock). Both impedance of urinary drainage and respiratory

embarrassment secondary to elevated airway pressures are also characteristic. Several risk factors for ACS are recognized; fascial closure during damage control stage IV has been identified repeatedly as a risk factor for the development of ACS. Both large volume fluid resuscitation and attempts to resuscitate to supranormal physiology have also been implicated [17–19].

Physical exam findings, such as elevated airway pressures, oliguria, and tube feeding intolerance, may aid in the diagnosis of abdominal hypertension, but are in and of themselves insensitive [20, 21], mandating measurement of intra-abdominal pressure. Several techniques have been described, including transduction of intragastric, intravesicular, and intraperitoneal pressure. Measurement of the intravesicular pressure is the current reference standard with several noteworthy technical considerations. Measurement should occur at the midaxillary line, at end expiration, in the absence of muscle contractions, and after instilling no more than 25 mL of sterile saline into the urinary bladder [16]. Pressure is expressed as mm Hg. Normal intra-abdominal pressure is <7 mmHg, increases >12 mmHg constitute abdominal hypertension, and a sustained pressure ≥20 mmHg in the presence of organ failure is diagnostic of ACS. Disease severity may also be expressed as the abdominal perfusion pressure, defined as the mean arterial pressure minus the intra-abdominal pressure. An abdominal perfusion pressure <50–60 mmHg is associated with poor outcomes among patients with intra-abdominal hypertension [22].

Medical therapy aimed at reducing abdominal pressure may be attempted for the hemodynamically stable patient in the absence of worsening organ failure. Paralysis, intestinal decompression, and diuresis are all effective means to decrease abdominal pressure. However, sustained or worsening intra-abdominal hypertension after a brief trial of nonoperative maneuvers mandates surgical decompression, as delay in definitive decompression worsens outcomes substantially [22]. Percutaneous catheter decompression may be considered when elevated abdominal

pressure is secondary to intraperitoneal fluid (e.g., ascites). Small case series suggest that this technique may be particularly useful among burn patients [23–25]. However, beyond this specific circumstance, surgical decompression via laparotomy remains the definitive treatment for ACS. Failure of improvement following surgical decompression should raise concern for either inadequate decompression or misdiagnosis. When timely and effective surgical decompression is achieved, the abdomen is usually amenable to closure within 7 days.

Stage V, Abdominal Wall Reconstruction: When fascial closure is not possible at the time of damage control phase IV, a variety of methods exist for temporary abdominal dressing, ranging from intravenous solution bags to vacuum-assisted closure devices. General management principles for the open abdomen in the acute setting include wound care, fluid and electrolyte balance, nutritional support, and attempts at sequential closure. Provided an adequate dressing is in place, patients with an open abdomen may be awoken, extubated, and participate in their care and medical decision making. Although evisceration around the abdominal dressing with abrupt increases in intra-abdominal pressure is possible (e.g., coughing fits), this risks is overshadowed by the potential complications of prolonged sedation, paralysis, and mechanical ventilation.

During the immediate postoperative days to weeks, our practice is to perform sequential washouts and partial primary closures approximately every 48 h with the goal of ultimate fascial closure during the index hospitalization. This technique involves sequential primary re-approximation of the midline fascia with interrupted suture bites over a vacuum-assisted closure sponge [26]. Because all damage control patients are markedly total body volume overloaded, aggressive diuresis is helpful to maximize the likelihood of fascial closure, provided that renal and cardiac function will tolerate it. As mentioned previously, enteral nutrition is not contraindicated in the patient with an

open abdomen and likely improves outcomes [14]. Using a standardized protocol that incorporates these principles, we are able to achieve fascial closure in nearly all damage control patients during the index hospitalization [26].

The incidence of complications associated with the acutely open abdomen raises sharply after a period of 1–2 weeks [27]. The two most devastating complications are entero-atmospheric fistulae and tertiary peritonitis. The term entero-atmospheric fistula refers to a communication between a hollow viscus and ambient air through the open abdominal incision (as opposed to through the skin in the case of an entero-*cutaneous* fistula). The risk of entero-atmospheric fistula increases linearly with time as a consequence of multiple dressing changes and prolonged exposure and manipulation of the vulnerable, edematous intestinal contents. Entero-atmospheric fistulae are particularly difficult to manage due to the lack of surrounding skin to which dressing appliances may be secured.

Tertiary peritonitis refers to persistent infection of the abdominal cavity despite multiple attempts at source control [28]. At the cellular level, tertiary peritonitis is characterized by failure of host peritoneal defense mechanisms, such that infection is encountered at each reoperation despite prior efforts to eradicate it. Although the peritoneal cavity is initially continuous, adhesions that form over days to weeks partition the space into multiple potential sites of abscess formation, including sub-diaphragmatic, inter-loop, and pelvic. As time elapses, these cavities become increasingly difficult to drain effectively, and the benefit of drainage is rapidly outweighed by the risk of bowel injury from repeated manipulation. As opposed to secondary peritonitis, the microbiology of tertiary peritonitis is characterized by a high prevalence of multidrug-resistant organisms such as enterococci, *Pseudomonas aeruginosa*, and *Candida* spp., making effective antimicrobial therapy challenging.

The best treatment of both entero-atmospheric fistulae and tertiary peritonitis is prevention. Accordingly, if fascial closure of the open abdomen is not possible after 1–2 weeks, we advocate temporarily skin closure over the abdominal contents, either with native skin (provided there is enough laxity) or autologous, split-thickness skin grafting from another site. Although this approach relegates the patient to a planned ventral hernia, the aforementioned complications of the open abdomen are minimized. Definitive fascial closure using more complex techniques, such as component separation and myocutaneous advancement flaps, should not be attempted in the acute setting, especially in the setting of peritonitis. Rather, development of the planes necessary for these operations should be reserved for the elective setting, at which time the risk of recurrence is much lower.

The time period following hospital discharge with a planned ventral hernia represents the final period in the damage control sequence [29]. Techniques for late closure are many and beyond the scope of this chapter. In the majority of cases, a combination of techniques, in conjunction with assistance from a plastic surgeon, can achieve definitive fascial closure with durable results and minimal morbidity.

Damage Control Ground Zero: Several authors have expanded the damage control concept to include the time between initial insult and operation, termed damage control ground zero. This phase highlights the importance of triage, emergency medical services scene time, rewarming in the trauma bay, early injury pattern recognition, and the early decision to initiate the damage control sequence. As mentioned previously, once the decision to initiate damage control has been made, minimal additional time should be spent in the emergency department prior to operation.

4.4 What Has Been the Impact of Damage Control Surgery on Patient Outcomes?

Since the initial description of the damage control technique, many groups have published data detailing improved outcomes using this approach. Although no randomized trials exist, several carefully matched case control series have concluded that mortality is decreased following adoption of damage control, provided that both appropriate indications and triggers for progression through the sequence exist [30, 31]. These improved outcomes are likely multifactorial in etiology, including broadened threshold to initiate damage control, earlier decision to terminate the initial operation, improved ICU resuscitation, and advancements in the techniques for achieving definite abdominal closure.

4.5 Summary

The damage control approach recognizes the notion that prolonged operation in the face of both shock and coagulopathy increases mortality. It is thus predicated on an initial, abbreviated, lifesaving laparotomy, followed by a period of resuscitation in the ICU, and finally by a planned reoperation at which definitive repair is accomplished. Multiple factors contribute to the decision to initiate the damage control sequence and can be broadly grouped into the physiologic variables of hemodynamics, temperature, metabolism of acid, and coagulopathy. A necessary sequelae of the damage control approach is the open abdomen. Although a variety of management options exist, of

paramount importance is achieving coverage of intestinal contents so as to minimize the development of fistulae and peritonitis. Adoption of the damage control approach has been associated with markedly improved outcomes for the sickest injured patients and should be part of the armamentarium of all traumatologists.

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