DAMAGE CONTROL

Introduction

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Care of patients with massive injuries and profoundly deranged physiology represents the core of medical practice in trauma. Early definitive care of isolated injuries leads to more rapid patient recovery and better overall outcomes. In the past decade, however, there has been a growing recognition that the benefit of definitive repairs must be weighed against the physiologic risk to the patient of prolonged operative time and increased surgical blood loss. This understanding is encapsulated in the concept of damage control—doing just enough surgery to prevent early loss of life or limb while reserving more definitive procedures for a time when the patient is warmer and better resuscitated.

In this issue of *TraumaCare* are three looks at damage control, as it is thought of today. The contribution of Dr. Mohr and colleagues is a more detailed look at the indications for damage control, with specific recommendations for when and to which patients it should be applied. From South Africa, the contribution of Dr. Moeng and colleagues offers a history of the concept, and a practical guide to damage control in abdominal, thoracic, vascular, orthopaedic, and neurosurgical procedures. The third and final selection, Dr. Dutton's description of "Damage Control Anesthesia," is an overview of the nonsurgical care that should accompany damage control, including guidelines for fluid resuscitation, management of blood composition, and sedation and analgesia. Taken together, these three articles provide an excellent snapshot of the current "cutting edge" of trauma care today, and we hope they will be of use to practitioners around the world.

Guidelines for the Institution of Damage Control in Trauma Patients

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Learning Objectives: 1) To review the epidemiology and metabolic consequences of exsanguinaton in trauma. 2) To familiarize the reader with the evolution of the strategies for damage control. 3) To learn the technical aspects of current damage control strategies. 4) To review the algorithm for the management of exsanguination and understand when to activate and use such a protocol.

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"Hesitance.....must yield to decision;
tardiness to promptness;
timidity to boldness.
The patient is saved or lost in a moment."

—Samuel D. Gross

Abstract

The speed by which the exsanguinating trauma patient moves from the prehospital arena to the emergency department, operating room, and intensive care unit is important to survival. In this article we describe the current guidelines for the institution of damage control in trauma patients. Certain conditions and complexes of injuries require damage control. In this study we describe validated indicators that can be utilized both preoperatively and intraoperatively to improve outcomes. Emphasis is placed on the current indications for damage control as defined by key studies. Awareness of these guidelines can improve outcomes after major intraabdominal injuries and hemorrhage and also assist in the management of one of the well-known sequelae of damage control, the posttraumatic open abdomen.

Severe traumatic injury is a public health care problem, with injuries accounting for 12% of the global mortality.[1] Continued improvement in the survival of severely injured trauma patients is a paramount goal. Bailout/damage control surgery following trauma has developed as a major advance in surgical practice in the last 20 years. The principles of damage control surgery defied the traditional

surgical teaching of definitive operative intervention and were slow to be adopted. Currently, techniques developed by trauma surgeons known as damage control surgery have been successfully utilized to manage traumatic thoracic, abdominal, extremity, and peripheral vascular injuries. In addition, damage control surgery has been extrapolated for use in general, vascular, cardiac, urologic, and orthopaedic surgery.

Stone et al[2] were the first to describe the "bailout" approach of staged surgical procedures for severely injured patients. This approach emerged after their observation that early death following trauma was associated with severe metabolic and physiologic derangements following severe exsanguinating injuries. Profound shock along with major blood loss initiates the cycle of hypothermia, acidosis, and coagulopathy.[2-9] During the 1980s, hypothermia, acidosis, and coagulopathy were described as the "trauma triangle of death" or the "bloody vicious cycle." A fourth component was later described by Asensio and colleagues,[3,10-12] who added dysrhythmia, which usually heralds the patient's death. Coagulopathy, acidosis, and hypothermia make the prolonged and definitive operative management of trauma patients dangerous. The management technique, now described as "damage control" by Rotondo et al,[5] involves a multiphase approach, in which reoperation occurs after correction of physiologic abnormalities.

Metabolic Consequences of the "Lethal Triad" or Bloody Vicious Cycle

Hypothermia is a consequence of severe exsanguinating injury and subsequent resuscitative efforts. Severe hemorrhage leads to tissue hypoperfusion and diminished oxygen delivery, which leads to reduced heat generation. Clinically significant hypothermia is important if the body temperature drops to <36˚C for more than 4 hours. Hypothermia can lead to cardiac arrythmias, decreased cardiac output, increased systemic vascular resistance, and left shift of the oxygen-hemoglobin dissociation curve. It can also induce coagulopathy by inhibition of the coagulation cascade.[5,13-16] Low temperature also impairs the host's immunologic function. Hypothermia is aggravated by heat loss from either environmental factors or surgical interventions. The multidisciplinary team caring for trauma patients must make every effort to prevent heat loss and help to correct hypothermia.

Clinical coagulopathy occurs because of hypothermia, platelet and coagulation factor dysfunction, which occurs at low temperatures, activation of the fibrinolytic system, and hemodilution following massive resuscitation. Platelet dysfunction is secondary to the imbalance between thromboxane and prostacyclin that occurs in a hypothermic state. Hypothermia and hemodilution produce an additive effect on coagulopathy. After replacement of one blood volume (5,000 mL or 15 units of packed red blood cells [RBCs]) only 30%-40% of platelets remain in circulation.[14] The prothrombin time (PT), partial prothrombin time (PTT), fibrinogen levels, and lactate levels are not predictive of the severe coagulopathic state.

Anaerobic metabolism starts when the shock stage of hypoperfusion is prolonged, leading to metabolic acidosis caused by the production of lactate. Acidosis decreases myocardial contractility and cardiac output.[6] Acidosis also worsens from multiple transfusions, the use of vasopressors, aortic cross-clamping, and impaired myocardial performance. It is clear that a complex relationship exists between acidosis, hypothermia, and coagulopathy and that each factor compounds the other, leading to a high mortality rate once this cycle ensues and cannot be interrupted.

Evolution of Bailout/Damage Control

The original work by Stone et al[2] in 1983 described intraoperative clinical coagulopathy as an indication for "bailout." In this study, 17 patients underwent the bailout procedure, which included an initial laparotomy, followed by packing in patients with an observed clinical coagulopathy, then completion of the surgical procedure once the coagulopathy was improved. This resulted in 11 survivors, with a mortality rate of 35%. Rotondo et al[5] first described the multiphase approach to the management of exsanguinating patients sustaining abdominal injury, but did not define any objective parameters during the intraoperative phase of damage control. They reported a survival of 77% in a very small subgroup of patients with major vascular injury and two or more physical injuries. Burch et al[6] proposed a model based on core temperature ≤32˚C, pH ≤7.09, and RBC transfusion >22 units that could predict 48-hour survival; they also described the "Lethal Triad." Sharp and LoCicero,[17] in a study based on 39 patients, defined a temperature ≤33˚C, pH ≤7.18, PT of ≥16 seconds, PTT of ≥50 seconds, and RBCs tranfused ≥10 units as objective parameters to indicate the need for early packing.

Morris et al[8] described 107 patients who underwent staged laparotomy and abdominal packing. They proposed proceeding with damage control early in the course of operation based on patient's temperature <35˚C, a base deficit of >14, and the presence of coagulopathic bleeding. Similarly, Moore[7] described a progressive coagulopathy as the most compelling reason for staged laparotomy. A severe coagulopathic state was described as a PT and PTT >2 times normal, massive and rapid blood transfusion exceeding 10 units in 4 hours, and persistent shock, defined as oxygen consumption <110 mL/min/m^2, lactic acid level >5 mmol/L, pH <7.2, base deficit >14, and core hypothermia <34˚C. Cosgriff et al[9] subsequently postulated that the ability to predict the onset of coagulopathy would have significant implications with regard to instituting damage control. Their predictive model for life-threatening coagulopathy included a systolic blood pressure <70 mm Hg, temperature <34˚C, pH <7.10, and Injury Severity Score (ISS) ≥25.

No single model has been able to accurately predict the timing for institution of damage control.[2,5-9,18-21] A pH <7.1 or a core temperature of <33˚C may indicate that the "bloody vicious cycle" is too far advanced and cannot be interrupted. Similarly, it is difficult to obtain intraoperative results for PT, PTT, fibrinogen, and lactate levels at all hospitals or to place a Swan-Ganz catheter in the operating room.

In an attempt to institute the development of intraoperative guidelines for damage control/bailout, Asensio et al[4] first retrospectively evaluated 548 patients over 6 years who had been admitted to a very large urban trauma center with the diagnosis of exsanguination. Inclusion criteria were an intraoperative blood loss of at least ≥2000 mL, a minimum transfusion requirement of ≥1500 mL RBCs during the initial resuscitation, and the diagnosis of exsanguination. Data collected included demographics, prehospital and admission vital signs, and physiologic predictors of outcome; Revised Trauma Score (RTS), Glasgow Coma Scale (GCS), Injury Severity Score (ISS), volume of resuscitative fluids, need for thoracotomy in the emergency department (EDT), volume of fluids in the operating room, need for thoracotomy in the operating room (ORT), and intraoperative complications. In this patient population the Revised Trauma Score was 4.38 and the mean ISS was 32, denoting a physiologically compromised and severely injured patient population. There were 180 patients who underwent EDT with aortic

cross-clamping, open cardiopulmonary resuscitation; 99 (55%) succumbed in the emergency department. In addition to the 81 patients who survived EDT, 117 required ORT, for a total of 198 EDT and ORT, of which 56 (28%) survived to leave the operating room and the hospital.[4]

In this series, mean admission pH was 7.15, mean temperature was 34.3˚C in the operating room, and these patients received an average of 14,165 mL of crystalloid, blood, and blood products.[4] Overall, 449 patients survived to arrive in the operating room with some signs of life, and 281 patients died; 37% of these patients survived damage control. On the basis of these findings, objective intraoperative parameters were developed to predict outcome and provide guidelines on when to institute damage control. These parameters included operating room temperature of ≤34˚C, pH ≤7.2, serum bicarbonate ≤15 mEq/L, transfusion volume of RBC ≥4000 mL (12-14 units of RBCs), total blood replacement (including RBCs and whole blood) of ≥5000 mL, or total intraoperative fluid replacement, including crystalloid, blood, and blood products, ≥12,000 mL (Figure 1).[4]

Table 1. Physiologic Guidelines That Predict the Need for Damage Control

- Hypothermia ≤34˚C
- Acidosis pH ≤7.2
- Serum bicarbonate ≤15 mEq/L
- Transfusion of ≥4,000 mL blood
- Transfusion of ≥5,000 mL blood and blood products
- Intraoperative volume replacement ≥12,000 mL
- Clinical evidence of intraoperative coagulopathy

One of the natural sequelae in patients surviving damage control is an open abdomen. Asensio et al[22] prospectively validated their guidelines (see Table 1) in a series of 139 patients who underwent damage control and had posttraumatic open abdomen. This study consisted of two groups of patients: the first group included 86 patients studied retrospectively prior to the institution of these guidelines; the second group consisted of 53 patients studied prospectively after the institution of these guidelines. These groups were comparable by all parameters. Although there was no difference in the mortality rate between the two groups (24% for each), there were statistically significant differences in the number of intraoperative transfusions, less hypothermia and bowel edema, less postoperative infections and gastrointestinal complications, as well as shorter intensive care unit and hospital lengths of stay for the prospective group managed with these guidelines. Another significant finding in this study was that 93% of patients were able to undergo definitive abdominal closure in their hospital stay as compared with the historic control of 22%.

Awareness of potential triggers to initiate damage control is vital. A study of 68 patients who underwent damage control surgery found that the inability to correct pH >7.21 and PTT >78.7 seconds was predictive of 100% mortality.[23] Delayed recognition of the need for damage control as well as poor communication with the anesthesia and nursing team are deleterious to the care of the multiply injured patient. The authors concluded that the institution of these guidelines reduced the incidence of posttraumatic open abdomen.

Patient Selection

Not all trauma patients require damage control measures. In addition to the physiologic guidelines for the institution of damage control (Table 1), certain conditions and complexes of injuries assessed both preoperatively and intraoperatively require damage control (Figure 1). Multiple mass casualties and the need for EDT predict the need for damage control.[4] In the multiply injured trauma patient sustaining major abdominal injury, the need to evaluate for other extraabdominal injuries in a timely fashion may also indicate damage control.[18] Garrison et al,[18] in a retrospective study, found that the preoperative duration of hypotension (systolic blood pressure <90 mm Hg) was significantly different in those patients that exsanguinated as compared with survivors (45 vs. 85 minutes). Therefore, in addition other factors such as the preoperative assessment of hypothermia and coagulopathy, a period of sustained hypotension >60 minutes would predict the need for damage control.[19,21] Intraoperatively, certain complexes of injuries also predict the need for this technique. These injuries include major abdominal vascular, complex hepatic, major thoracic vascular injuries, and the need for intraoperative thoracotomy.[22]

Patients with exsanguination are perhaps the best candidates to undergo damage control. Asensio et al[22] have described an algorithm for the management of exsanguination that involves three phases (Figure 2). First, the patient is classified as exsanguinating; second, resuscitation as per Advanced Trauma Life Support Protocols is begun (Figure 2). In the third phase there is rapid transport to the operating room (exsanguination from penetrating injuries is a dramatic, ill-defined entity that requires leadership, prompt thinking, aggressive surgical intervention, and a well-thought out plan).[22] Rapid institution and damage control can lead to effective management of exsanguination and improve survival.

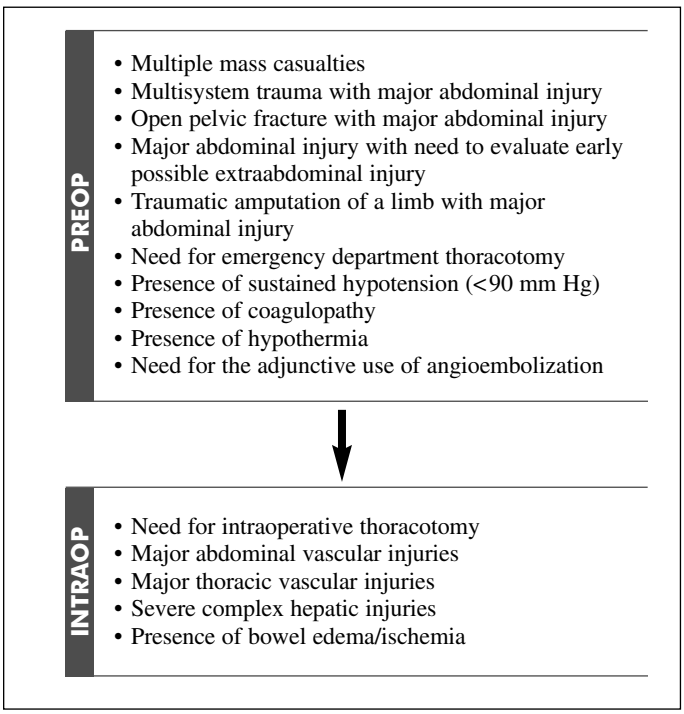

Figure 1. Preoperative and intraoperative states that suggest the need for damage control. Preop, preoperative; intraop, intraoperative.

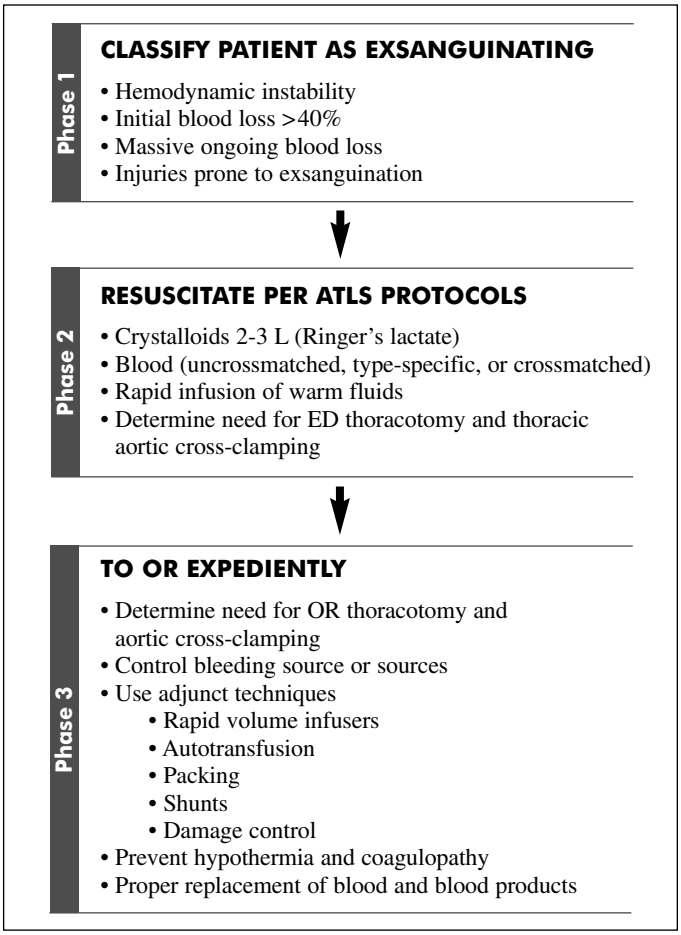

Figure 2. Algorithm for the management of exsanguinations. ED, emergency department; OR, operating room. (Courtesy of J. A. Asensio, MD)

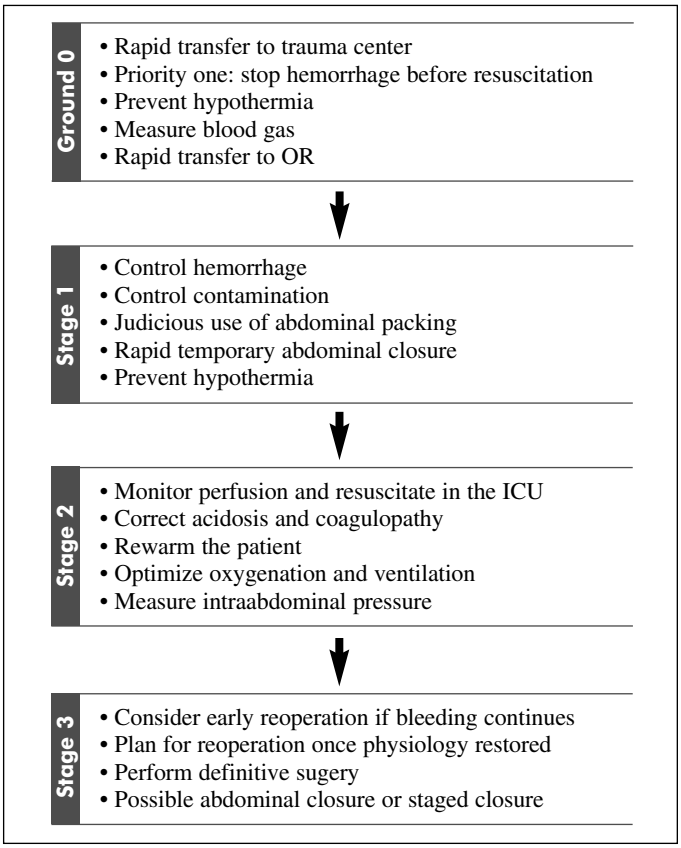

Figure 3. Four stages of damage control. OR, operating room; ICU, intensive care unit. (Courtesy of J. A. Asensio, MD)

Technical Aspects of Damage Control

The most important goal of early institution of damage control is patient survival. A four-stage damage control approach has been defined recently by Johnson et al[16] in a small study that included 24 patients who underwent damage control and were retrospectively compared with patients who underwent damage control a decade earlier (Figure 3). The "ground zero" stage includes the prehospital phase as well as early resuscitation in the emergency department.[16] This ground zero phase includes short paramedic scene times, identification of injury patterns in the emergency department that require damage control, as well as rewarming maneuvers that begin in the trauma bay.[16]

Damage control, according to Asensio et al,[4] implies immediate control of life-threatening hemorrhage, control of gastrointestinal contamination with rapid resections or closures, the use of intraluminal shunts, and judicious abdominal packing with temporary abdominal wall closures.[4,10-12] Specifically, for chest injuries one should repair cardiovascular injuries, perform stapled pulmonary tractotomy,[24] pack if needed, place chest tubes, and close the skin.[4] For abdominal injuries, damage control can involve control of major hemorrhage, hepatic packing, pancreatic drainage, temporary hollow viscus closures, rapid stapled resections, splenectomy, nephrectomy, vascular pedicle clamping in situ, and the use of intraabdominal vascular shunts.[4] Frequently, these patients experience abdominal compartment syndrome. Therefore, the posttraumatic open abdomen with temporary abdominal wall closure is used as an extension of damage control.

The second stage begins in the intensive care unit, where the trauma surgery team tries to correct the metabolic disorders. Rewarming the patient is a high priority because coagulopathy and acidosis can be corrected and maintained only after the body temperature returns to normal. Further inspections are then made to identify injuries that may not have been detected in the initial survey. Twenty-four to 72 hours may be needed to correct metabolic derangements.

The last stage of damage control involves the timing of reoperation when definitive procedures are performed. Reoperation is considered early if major blood losses continue. Usually, there is a window of 36-48 hours after the initial injury, between the correction of the metabolic disorder and the onset of the systemic inflammatory response syndrome and/or multiple organ failure. In this phase, definitive procedures are undertaken. Thorough reexploration is made for any additional injuries, and restoration of gastrointestinal continuity and vascular repair are done. Provisional feeding access may be placed, which is followed by washout of the abdominal cavity and an attempt at definitive closure. The patient then returns to the intensive care unit for further care.

Conclusions

The exsanguinating trauma patient who requires massive transfusion incurs the greatest risk for the multifactorial interactions between acidosis, hypothermia, and coagulopathy. There continues to be an ongoing challenge to identify better predictors of outcome, improved means of resuscitation, greater understanding of physiologic derangements, and better timing to institute damage control. There also remains a need to better understand the cellular

and subcellular mechanisms triggered by profound shock, exsanguination, acidosis, hypothermia, and coagulopathy. Delays in the decision to perform damage control contribute to a higher morbidity and mortality. Therefore, damage control is a vital part of the management of the multiply injured patient and should be performed before metabolic exhaustion.

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Damage Control: Beyond the Limits of the Abdominal Cavity. A Review

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Learning Objectives: 1) To understand the concept of damage control surgery. 2) To review the pathophysiology after major trauma. 3) To understand the indications for damage control surgery. 4) To understand the decision-making, procedures used, and timing involved in planning damage control procedures.

Abstract

The end point of any operation is the restoration of disrupted anatomy, and often technically impressive repairs accomplished after hours of surgery do not restore the physiology. Damage control is a concept in which the initial surgery becomes part of the resuscitation process rather than part of the curative process. The surgery is aimed at limitation of further physiologic insults, bleeding, and contamination. Once this limitation has been achieved, the patient's ongoing surgery is abbreviated and resuscitation continues in the intensive care unit. Only when the patient has become physiologically stable is the final therapeutic surgery embarked on. This process serves to limit the physiologic exposure to an unstable environment, allowing better resuscitation and outcome in the critically ill patient.

The concept of damage control was born out of the need to care for hemodynamically unstable patients who have sustained multiple high-energy injuries (including both blunt and penetrating trauma). This is not a modern concept, but its application represents a new paradigm in surgery. Damage control itself produces a whole new set of challenges, complications, and disease syndromes not previously encountered.

The end point of any operation is the restoration of disrupted anatomy and physiology. Technically impressive repairs (usually performed "after hours"), however, often expose the patient to a

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