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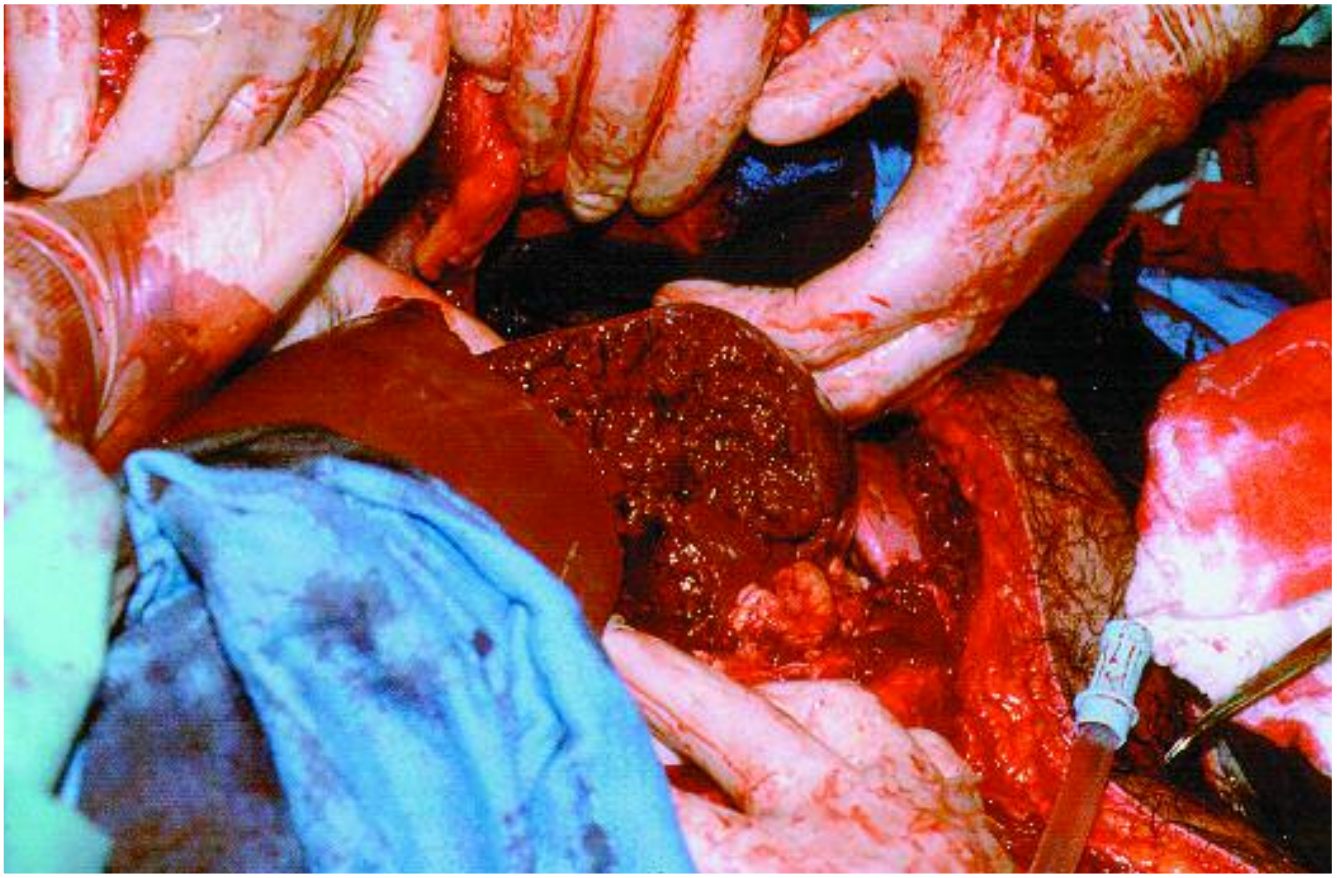

Surgical exploration of a grade V liver injury. Photo courtesy of Richard Dutton, MD.

Damage Control

- Guidelines for the Institution of Damage Control in Trauma Patients
- Damage Control: Beyond the Limits of the Abdominal Cavity. A Review
- Damage Control Anesthesia

SOCÍETÉ INTERNATIONALE D ANESTHÉSIE - REANIMATION EN TRAUMATOLOGIE
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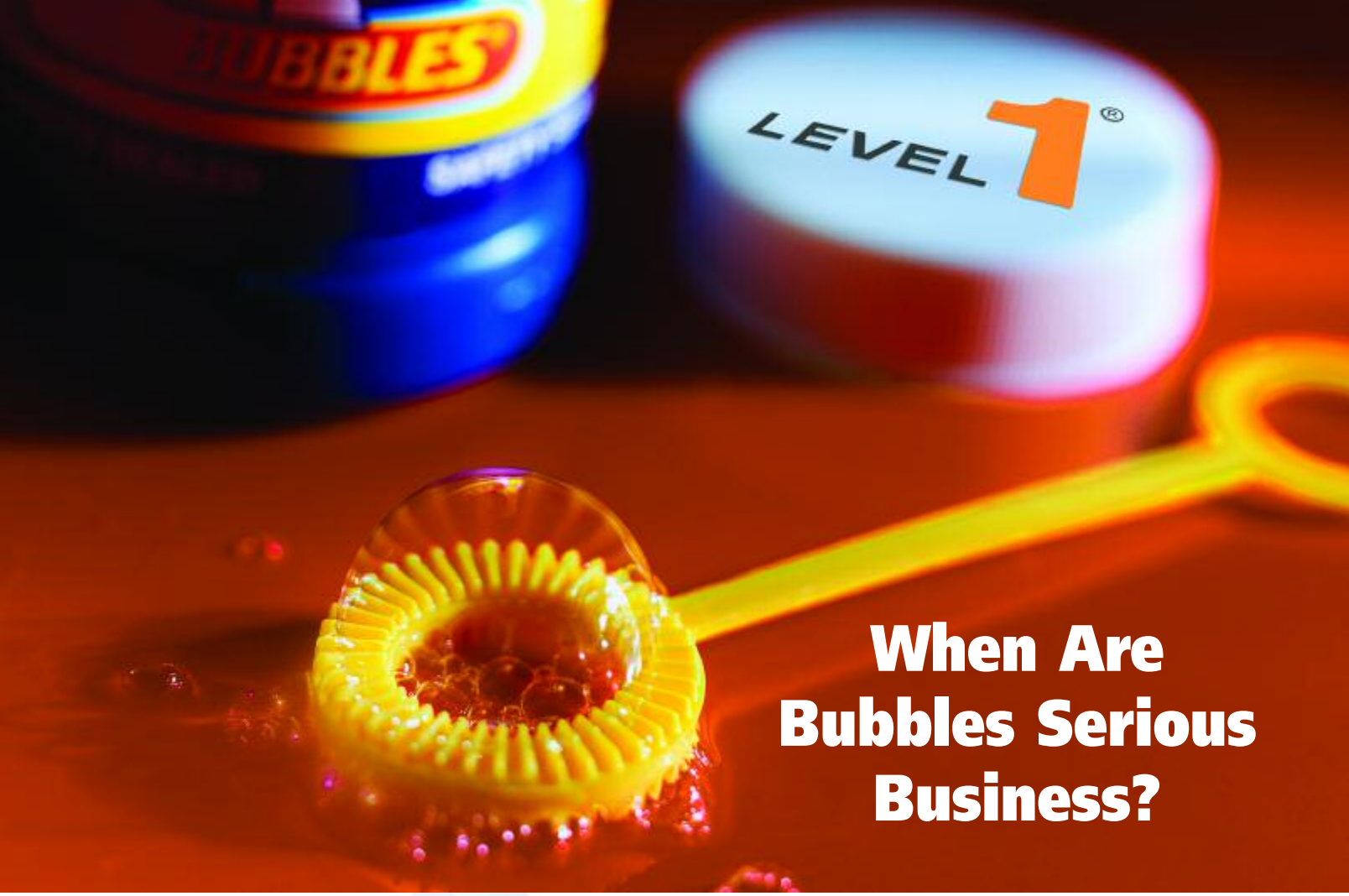

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PRESIDENT'S MESSAGE

"Damage Control": Anesthesiology Challenges in the United States

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Greetings and welcome to the Fall 2005 issue of *TraumaCare* with a focus on damage control surgery. I would like to thank our guest editor, Dr. Richard P. Dutton, Chief of Trauma Anesthesiology at the R Adams Cowley Shock Trauma Center, and his assembled panel for their contributions. The advent of damage control surgery signaled a dramatic change in the surgical approach to the critically injured trauma patient. This relatively recent strategy of resorting to the operating room to treat only immediately life-threatening injuries in the severely coagulopathic, hypothermic, and/or metabolically deranged patient has been accepted to yield improved survival rates. Damage control surgery decreases the likelihood of the dreaded "successful surgery, but the patient died" scenario and instead offers the chance to win the war despite losing a battle.

As I read the assembled collection of articles contained herein, I am struck by how, under a different rubric, anesthesiologists in the United States have been practicing "damage control" anesthesiology, continually struggling to preserve anesthesiology and quality patient care in the face of a continually expanding array of competing medical, political, and financial interests, yet seemingly unable to seize opportunities to perform definitive action. For example, in my personal experience as president of a state society of anesthesiologists, my colleagues and I continually deal with external forces hostile to anesthesiology through meetings, discussions, and negotiations. A significant portion of time is spent with state and federal governmental officials, fighting to stave off changes detrimental to anesthesiology and to patient care. Even modestly sized state societies utilize the assistance of a lobbyist, often their largest single expense. Recent concerns include both financial and regulatory matters. West Virginia anesthesiologists recently repelled an attempted retroactive, arbitrary reduction of anesthesia reimbursement by more than 25% for state programs, yet still were forced to accept an average reduction of 8%. The newest challenge is a proposed change in state regulations requiring a staff anesthesiologist to be physically present for the entirety of the case when an anesthesiology resident or nurse anesthetist student is involved in the care of the patient. This contrasts to the current nationwide supervision standard allowing supervision of more than one room; being present for induction, emergence, and all critical portions of the case while being immediately available throughout. These significant negative economic consequences would likely result in the closure of anesthesia training programs.

On a larger scale, the past decade presented even more challenges to anesthesiology in the United States, including expansion of nonphysician anesthesia providers, increasing regulatory environment, diminishing financial resources, and diminishing of scope of practice. Anesthesiology in the United States is still trying to recover from big hits that occurred in the 1990s. Early in that decade, the federal government, via the Centers for Medicare and Medicaid Services (CMS), arbitrarily decreased reimbursement for anesthesiology resident medical care in contrast to other medical specialties. This selective reduction in anesthesiology reimbursement decreased academic department revenues by up to 50%. The American Society of Anesthesiologists and its members have been lobbying for anesthesiology resident services to be reimbursed in a fashion consistent with other medical specialties ever since. Now, after more than 10 years of effort, it appears that our work may pay off and CMS may revisit this issue.

The 1990s also witnessed a dramatic proposal to restructure the United States health care system. Although in the end no significant changes occurred, concerns at that time regarding the possible detrimental financial effects nonetheless had a radical impact on anesthesiology. Departments and groups exerted fiscal restraint in the face of the unknown, and graduating residents found less-desirable job opportunities. Most groups offered decreased salaries and benefits, while many elected not to make any hires at all. Furthering the tension, a *Wall Street Journal* article inaccurately described anesthesia residents as unable to obtain jobs on completion of their training, counting among the "unemployed" those entering fellowships, the military, or practicing as locums tenems. These increasing anxieties, along with a projected oversupply of medical specialists and a major drive by medical school deans (overwhelmingly primary care physicians) for medical students to enter primary care fields devastated anesthesiology resident recruitment in the mid-to-late 1990s. Anesthesiology resident match numbers fell to less than 25% of what they were in the early 1990s. Within a short time, a severe shortage of anesthesiologists in the United States resulted, impacting anesthesiology to this day and demonstrating the folly of such reactionary thinking.

Training programs dramatically altered their behavior. At the peak of the manpower shortage, even some of the most prestigious programs gave substantial financial inducements to residents to enter their programs, largely so there would be bodies in operating rooms to "squeeze bags." Given the continued shortfall of residents to cover the rooms in teaching hospitals, hospitals markedly increased the presence of certified registered nurse anesthetists and anesthesiology assistants, with many teaching hospitals remaining reliant on nonphysician anesthesia providers.

Residency programs made their programs more attractive in other ways as well. Changes such as expansion of educational programs were viewed positively by some, yet considered "spoon-feeding" by others. Lessened work hours and reduced schedules produced a generation of anesthesiology residents often working fewer hours per week than their faculty, and certainly less than when their faculty were residents. Many programs promoted a "kinder and gentler" system in which the residents did not take in-house call on weekends, but instead calls were covered by in-house faculty. As programs and hospitals scrambled to fill the void, an unfortunate result is a generation of anesthesiologists who found anesthesiology residency programs pulling out all stops in their efforts to recruit them, and subsequently, departments and groups recruiting them with lavish packages on completion of their training. Recent anesthesiology graduates often are unaware that the primary source of anesthesiologists' income increase of more than 50% over the past 5 years is the result of hospital and university subsidies to keep

operating rooms open and remain competitive in their markets. Currently, anesthesia departments in level I trauma centers routinely receive subsidies in the millions of dollars. Academic departments, in general, today receive on average over one hundred thousand dollars subsidy per faculty member to recruit and retain faculty. Few residents and recent graduates anticipate that as the anesthesiology shortage eventually abates, such subsidies will almost assuredly disappear as market forces come to bear.

This environment has contributed to unrealistic expectations, potentially making new graduates less well equipped for entry into the postgraduate world of medicine than their immediate predecessors. An increasing strain has been noted between recent anesthesiology graduates and their established brethren. The American Society of Anesthesiologists' 2005 Practice Management meeting acknowledged such difficulties by providing a breakout session devoted to generational issues.

Perhaps more importantly, however, during these disruptions, anesthesiology saw an accelerated reduction in scope of practice in the United States in comparison with our international anesthesiology colleagues, particularly in regard to trauma care. The relatively new acute care specialties of emergency medicine and critical care medicine were originally outgrowths of anesthesiology. In many parts of the world these practice areas remain fully integrated within the anesthesiology's purview. In contrast, department leaders in the United States often appeared grateful to unload some of their commitments in the face of the dramatic anesthesiologist shortage. Regrettably, apparently well-intentioned leaders, in many cases, now appear myopic in retrospect.

I have the pleasure of frequent discussions and communication with international colleagues along with hosting international anesthesiologists. They are uniformly shocked and amazed by the reduced scope of anesthesiology in the United States. They are further shocked to find anesthesiologists in the United States constantly striving for respect from medical colleagues. They find the notion that nonanesthesiologists could replace anesthesiologists inconceivable, given the breadth of acute care medicine anesthesiologists practice elsewhere. While recognizing that economics have played a significant role in shaping anesthesia's current position in medicine in the United States, both in the evolution of relatively new fields such as emergency medicine and the reduction of anesthesia involvement in critical care medicine, the future of anesthesiology will be ensured by making the United States' anesthesiologists as indispensable as anesthesiologists elsewhere.

Again using trauma care as an example, anesthesiologists typically are the leaders of the trauma care teams for a large portion of the world. Anesthesiologists not only care for trauma patients in the operating room, but are also first responders at the out-of-hospital scenes of trauma. They coordinate trauma care from the receiving bay in the emergency department through the critical care unit. In this role, not only are anesthesiologists indispensable, but also are uniformly held in higher regard by their medical colleagues. Anesthesiologists in the United States once led medical advances in trauma care. They have now largely deserted or allowed themselves to be marginalized in many aspects of trauma care, perhaps in part as a result of anesthesia resources being stretched so thin by the ongoing shortage of anesthesiologists. The question is whether, when this shortage is resolved, as it likely will be with anesthesiology match numbers in the United States now approaching record numbers, will anesthesiology ever regain what is has given up? Emergency medicine physicians provide prehospital and hospital-receiving care, even supplanting anesthesiologists in many centers as emergency airway management experts. Readers may recall receiving a mass mailing from an emergency medicine physician inviting anesthesiologists to attend his difficult airway management seminars, heretofore anesthesiology's calling card.

Another area of acute care medicine originally led by anesthesiologists, yet now with continually diminishing anesthesia involvement in the United States, is critical care medicine. Despite a recent thrust to staff critical care units with full-time intensivists primarily from economic pressures exerted on health care systems by groups such as the Leapfrog Group, a consortium of more than 100 private and public sector health care purchasers finding health care costs to be reduced in hospitals with full-time intensivists, critical care continues its trend of nonanesthesiologist intensivists staffing most of the critical care units. Although perceived as financially attractive for some medical fields, anesthesiologists in the United States can at this moment generate more revenue per hour providing anesthesia care compared with critical care services. Those anesthesia departments still successful in critical care tend to be those with substantial commitments from hospitals or universities to do so. Additionally, while the American College of Graduate Medical Education's (ACGME) Anesthesiology Residency Review Committee (ARRC) recently recommended increasing the critical care months required in residency from 2 months to 6 months, and leaders in the American Society of Anesthesiologists have been leading the charge for a renewed emphasis in critical care medicine, it appears that largely by political and financial reasons, including academic chairs' desires to not lose anesthesia hands on providers to the critical care unit, that such efforts have been largely abated. This is unfortunate not just for anesthesiology but also for our patients. Anesthesia intensivists' unique background as perioperative physicians, daily adeptly managing ventilators along with the rapidly changing physiologic conditions of exceptionally ill and traumatized patients, offer an unparalleled skill set to care for the critically ill and injured.

Events of the past 15 years should serve as a wake-up call to all anesthesiologists. Rather than practicing "damage control" in the political and regulatory worlds, it must be the goal of all anesthesiologists to ensure the vitality of anesthesiology. Strong, visible leadership is necessary. We must provide mentorship and be role models for those now entering medicine and anesthesia. We cannot count solely on our elected or designated leaders. Leadership and mentoring are most effective when individually demonstrated daily. We must commit to lead by example and proactively advocate for anesthesiology and for our patients. The challenge begins today. We must learn from our mistakes, else we are doomed to repeat them. Such effort will be rewarded not just by a change of mind of those in anesthesiology, but also by a change of heart, and will thus renew anesthesiology's vigor and position in medicine in the United States.

The President's Message is included in each issue of TraumaCare *and is a separate entity unto itself. The message reflects the thoughts and concerns of the President of International TraumaCare and, although it may be related to the subject of the issue, does not necessarily directly relate to the content of the issue. The President may often use this forum to express thoughts that are of a more political nature and are thus not necessarily connected with the scientific/medical content.*

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.

None of the authors have any conflicts of interest to disclose.

"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.

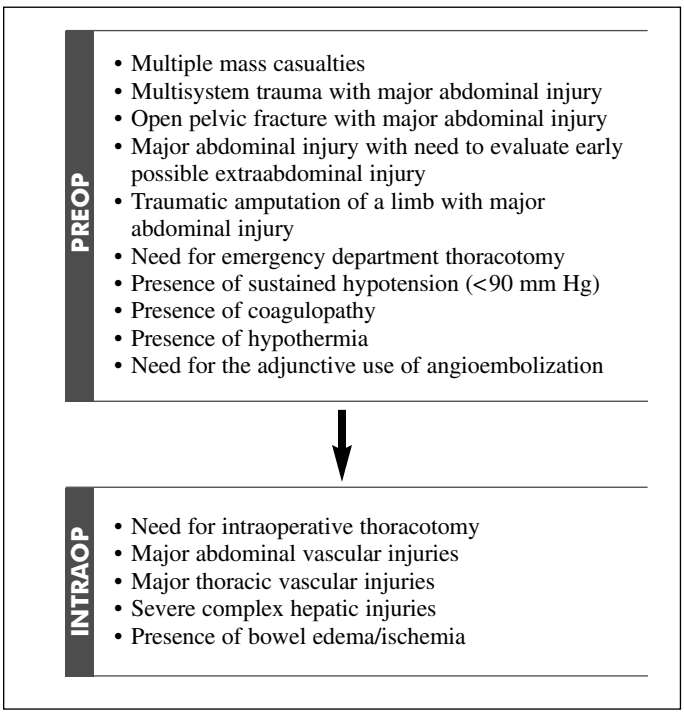

Figure 1. Preoperative and intraoperative states that suggest the need for damage control. Preop, preoperative; intraop, intraoperative.

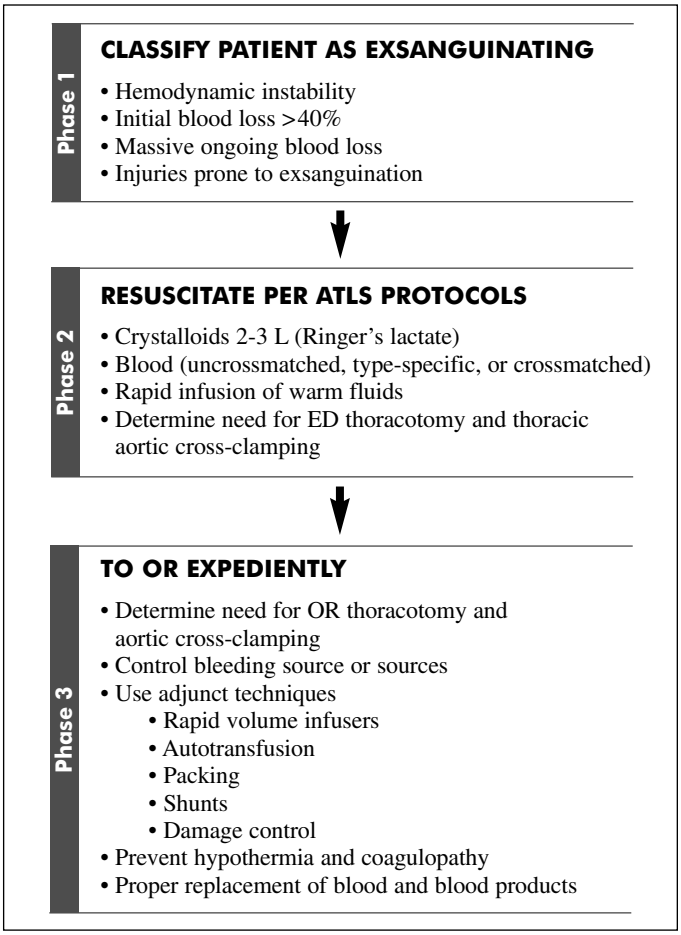

Figure 2. Algorithm for the management of exsanguinations. ED, emergency department; OR, operating room. (Courtesy of J. A. Asensio, MD)

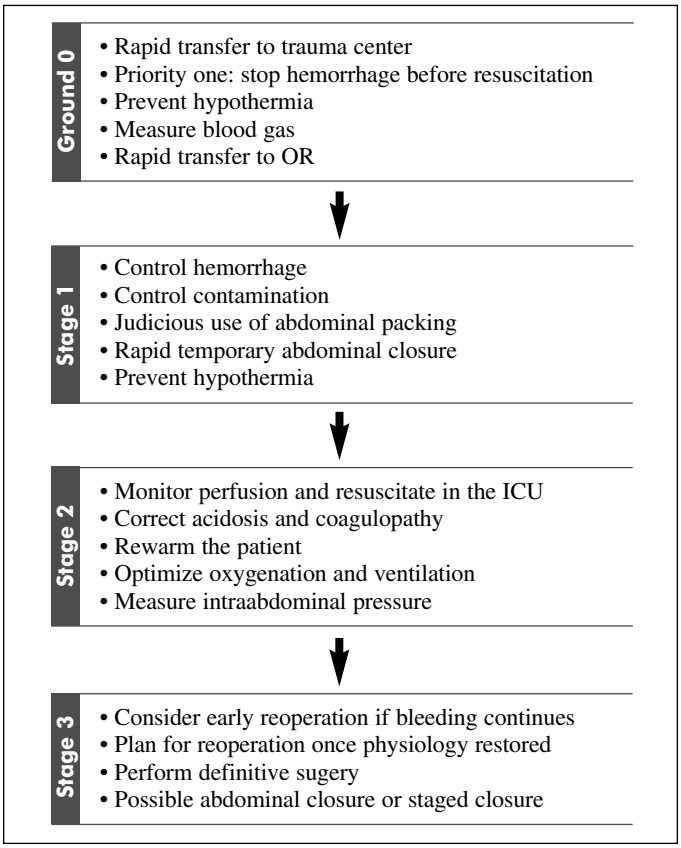

Figure 3. Four stages of damage control. OR, operating room; ICU, intensive care unit. (Courtesy of J. A. Asensio, MD)

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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harsh theatre environment that may worsen the physiologic insult. This creates a challenge to balance the metabolic insult of the initial operation against the then concept of early total care.

Damage control surgery (sometimes known as "damage limitation surgery" or "abbreviated laparotomy") is best defined as creating a suitable anatomical environment yet preventing the patient from progressing to an unsalvageable metabolic state. In many "good surgical" situations, patients are more likely to die from their "metabolic failure" than from failure to complete organ repairs.

Damage control is "a strategy that sacrifices the completeness of immediate repair so as to adequately address the combined physiological impact of trauma and emergency surgery."[1]

The term *damage control* originates from the United States Navy, with reference to "the capacity of a ship to absorb damage and maintain mission integrity."[2] This allowed for rapid assessment of the damage, thereafter instituting the best manner of sufficient temporary repair to facilitate expedient return to a controlled environment in port. This analogy of preventing a ship from sinking is even more relevant when one considers the anatomical and physiological damage inflicted on trauma patients.

In the early 19th century, Schroeder[3] and Halsted[4] discussed abbreviated laparotomy, describing planned reexploration for hepatic trauma. At this stage, packing was described with absorbable and nonabsorbable materials, which were sutured in place. The main complications were related to bleeding during removal of these materials. Pringle[5] subsequently described packing with occlusion of the porta hepatis to control liver bleeding. This continued into World War II, but was abandoned thereafter because of poor results related to complications associated with bleeding, necrosis, and sepsis, when Carmona et al[6] reported that perihepatic liver packing with planned reoperation was a "valuable adjunct…without incurring increased morbidity or mortality." This was confirmed when Feliciano et al[7] and others[6-11] reintroduced packing for control of hepatic hemorrhage in 1978.

In a prospective study, Stone et al[12] demonstrated a decreased mortality of 35% in the packed group, compared with 93% in the conventionally treated group. Seventeen patients were managed with damage control-type procedures, compared with 14 patients treated conventionally during the preceding 3 years. The majority of deaths were a direct result of uncontrolled hemorrhage.

Ivatury et al[9] did not demonstrate a decrease in mortality from hemorrhage, although sepsis remained an issue. A subset of the patients from this study gained from this approach of packing for tamponade. These were patients who required multiple transfusions and who were hypothermic and acidotic. This technique allowed time for transfer to a tertiary facility or abandonment of the procedure, resuscitation and correction of physiology, and controlled reoperation.[7,9,10]

The concept has evolved to include nonhepatic strategies, including thoracic injuries, vascular injuries,[13] complex soft tissue and orthopaedic injuries, and even neck injuries. As shown in Table 1, damage control is traditionally applicable as three or five phases,[14-27] although nowadays it is generally regarded as being divided into five phases.[16]

Pathophysiology

Elerding et al[28] emphasized the importance of preventing hypothermia as a prelude to coagulopathy in severe hepatic injury requiring massive transfusion. Trauma patients often present with hypotension, hypothermia, or both. Severity of initial injury, the harsh prehospital environment, and delay in transport can worsen the hypothermia. Hypothermia, bleeding, and metabolic acidosis further cause progressive coagulation defects, and these are the most important indicators for damage control procedures.

Hemorrhage leads to hypothermia. Impaired tissue perfusion further leads to decreased ability to generate heat at a cellular level. With hypothermia there is a sympathetic α-adrenergic overdrive, peripheral vasoconstriction, and more severe end organ hypoperfusion, resulting in conversion from aerobic to anaerobic metabolism and an ensuing metabolic lactic acidosis.[29,30]

Hypothermia is further associated with decreased myocardial contractility, increased systemic resistance, increased chances of cardiac arrhythmias, and left shift in the oxygen dissociation curve.[30] It further causes abnormalities in both active and slow-phase coagulation cascades. There is also a decrease in the production of thromboxane B_2,[31] which results in inhibition of platelet aggregation. Even with replacement of platelets, they may remain dysfunctional in hypothermia.

The coagulopathy has two causes. The dilutional component is invariably secondary to aggressive fluid resuscitation, and the second, a "consumptive coagulopathy," results from the system's activation as a normal physiologic response. The need to resuscitate appropriately with early replacement of red blood cells and clotting factors cannot be overemphasized. Active measures at warming the patient will limit further insult on the coagulation cascade.

The shock state causes anaerobic metabolism that results in metabolic acidosis. The increased adrenergic response further worsens cellular acidosis by direct effects in the cell. Aortic cross-clamping and vasopressors also contribute to metabolic acidosis. Acidosis causes a decrease in the patient's response to endogenous and exogenous catecholamines. This is the result of uncoupling of ß-adrenergic receptors.

Hirshberg and Mattox[1] described the operating room as "a physiologically unfavorable environment for the severely injured patient (involving) dissipation of body heat and blood loss, massive replacement (and the) result is hypothermia, coagulopathy and metabolic acidosis." The patient remains cold, becomes acidotic, and bleeds. This "bloody vicious cycle," the triad of hypothermia, acidosis, and coagulopathy is well described[7,9,10,13,32] and is seen as the trigger in events leading up to the requirement for damage control. Hypothermia is an independent risk factor,[33] but with a direct correlation to injury severity.[34] Mortalities of 100% in trauma patients undergoing laparotomy[33, 35] have been reported, with core temperatures of <32ºC.

Table 1. Staged Approach to Damage Control

Three-Stage Approach		Five-Stage Approach	
Stage 1	Patient selection and damage control surgery	Stage 1	Patient selection
		Stage 2	Damage control surgery
Stage 2	Restoration of physiology in ICU	Stage 3	Restoration of the physiology in ICU
Stage 3	Definitive operation with abdominal closure	Stage 4	Definitive surgery
		Stage 5	Abdominal closure

Indications

Traditionally, all methods of surgical control were attempted before progressing to packing as a last desperate measure.[7,10] In this situation, the decision to institute damage control is invariably made too late.[8,12] Although no definitive management plan existed, in 1986 Nichols et al[36] emphasized the importance of achieving appropriate control of surgical bleeding before closure. Cue et al,[32] reviewing packing and staged repair of both hepatic and retroperitoneal injuries, concluded that the procedure was more effective if instituted early, and that patients packed early required an average of six units of packed cells less than those packed after onset of coagulopathy. If effected early enough and adequately, packing will permit the return of the patient to the intensive care unit (ICU) for correction of acidosis, hypothermia, and coagulation abnormalities.[8] Consequently, there has been a search for early criteria to direct this decision and begin damage control in the resuscitation area.[6,32] Burch et al[13] were the first to use temperature and pH as indicators of coagulopathy; base excess and coagulation profile were subsequently added as independent indicators.[14] Length of procedure at this stage averaged 2.9 hours.

Other factors were recognized as predictive, not only for coagulopathy but for damage control itself.[15, 17-20] In 1998, Moore and colleagues[16] summarized the risk indicators of coagulopathy. Table 2 lists these development predictors for coagulopathy. At the time, the significance of a raised arterial lactate as a measure of anaerobic metabolism at tissue level, a readily available investigation, had not been realized. Only in 1992 did Talbert et al[14] report its use, although as an end point of resuscitation rather than as an indicator for abbreviated surgery. Systemic lactate provides a good indicator of the patient's physiologic insult, and therefore the awareness of the need for early damage control procedures. The inability to clear lactate in 48 hours is associated with increased mortality.[37]

Damage control is indicated only in a highly select group of patients,[18] and if the previously mentioned indicators are not adhered to, results will be no better than if the patient had undergone a definitive procedure in the first place. Morbidity may, in fact, increase, should inappropriate damage control procedures be instituted too late.

Table 2. Predictive Indicators for Coagulopathy

- Injury severity score >25
- Systolic blood pressure <70 mm Hg
- Temperature <34º C
- pH <7.10

Timing of Reoperation

Previous literature has described time to reoperation for periods between 8 hours and 10 days,[7, 12,16] generally at the surgeon's discretion. Formal thresholds have now evolved, principally the reversal of hypothermia, acidosis, and the correction of coagulopathy. A lactate level of 4 mmol/L is presently one of the best indicators of the return of tissue perfusion, in conjunction with a base deficit better than –4, and a normalized coagulation profile.

Differentiation between "planned" and "emergency" relook procedures is important. Although the guidelines noted here are appropriate to "planned" procedures, ongoing, uncontrolled surgical bleeding, the development of abdominal compartment syndrome (ACS), or both, may mandate an unscheduled revisit to the abdominal cavity. This may mean emergency return to the operating room, or urgent decompression in the ICU. Other situations may include limb ischemia after arterial ligation or stenting, or closed-loop obstruction following emergency bowel resection. Ideally, however, planned relook, although at the discretion of the surgeon, should be as soon as possible after physiologic parameters have been restored. The planned relook has a significantly lower mortality rate; Hirschberg et al[19] showed that 8 of 52 patients undergoing planned relook died, whereas 13 of 21 patients undergoing emergency relook died.

Complications

The most common condition directly associated with damage control is intraabdominal hypertension. This is easily measured with an indwelling urinary catheter[38] in a supine patient, zeroed at the midaxillary line. It should be expressed in millimeters of mercury. The intraabdominal hypertension can be divided into four grades,[39] as indicated in Table 3.

Table 3. Intraabdominal Hypertension

Grade	Intraabdominal Pressure Measurement (mm Hg)
0 (Normal)	<12
I	12–15
II	16–20
III	21–25
IV	>25

Abdominal compartment syndrome is not graded, but considered as an "all or nothing" phenomenon. Abdominal compartment syndrome, however, is defined as an intraabdominal pressure >20 mm Hg and /or abdominal perfusion pressure of <60 mm Hg in association with new-onset single or multiple organ failure. Better surveillance will lead to earlier detection and optimize outcome.

There is an associated increase in intraabdominal abscess formation with packing.[6-10,12,14] The use of antibiotic cover should be considered routinely in these cases. Care should be given to the number of packs used to avoid missed swabs, and it is good clinical practice to obtain radiographs of the abdomen in the operating room to ensure that there are no retained swabs. This is particularly relevant when one considers the urgency of these patients and the involvement of multiple personnel. Some septic complications were directly associated with selective hepatic artery ligation.[32]

Managing patients in this fashion also exposes them to complications of an open abdomen. The timing of closure, the best-available technique, and the patients' condition will determine the end results.

The Five-Stage Approach As Performed Today

The principles of the five-stage approach discussed in the next sections are as described by Moore (1998).[16]

The Damage Control Laparotomy

Stage 1: Decision to Perform Damage Control. For greatest benefit from this procedure, the decision to proceed to damage control surgery needs to be made as early as possible, before the physiologic derangement has reached the point of no return. This must be during initial planning of the operation or as soon as the operation is started and the injuries have been reassessed. Hirshberg and Mattox[15] emphasize the concept of index of suspicion based on "injury pattern recognition" rather than adherence to physiologic end points, although the two should be used in conjunction with each other, and the index of suspicion immediately confirmed intraoperatively (Table 4).

Table 4. Index of Suspicion for Damage Control

- Inability to achieve hemostasis because of coagulopathy
- Time-consuming procedure in appropriate patient (usually >90 min)
- Inaccessible major venous injury
- Associated life-threatening injury in second anatomical location
- Planned reassessment of abdominal contents
- Inability to approximate sheath because of visceral edema or ACS

Patients who will require operation time in excess of 90 minutes or those requiring multiple interventions should be recognized at this stage, and no attempt at definitive closure should be made.

The indications as described here, in conjunction with a systemic lactate of >5 mmol/L, together with an appropriate injury pattern, should prompt aggressive institution of damage control.

Stage 2: The Operation. During this stage, one aims at quick assessment of the injuries, definitive control of bleeding, control of contamination, and temporary abdominal wall closure. This is done simultaneously with ongoing anesthetic resuscitation. Coordination is essential to optimize outcome. The goals are shown in Table 5.

Table 5. Goals of the Damage Control Operation

- Initial control of hemorrhage (resuscitative packing)
- Exploration of the abdomen
- Control of contamination
- Therapeutic packing
- Rapid abdominal closure

The abdomen is explored initially, and all blood present is scooped or sucked out. Autotransfusion should be considered. Bleeding is controlled by vessel control (clamping) or initial packing. Packing is not a substitute for the direct control of large bleeding vessels. Aortic cross-clamping or pressure control may make assessment easier. Hemorrhage can then be assessed and dealt with by rapid repair, ligation, or use of stents for temporary control. Balloon catheter tamponade may be relevant for some injuries.

The Pringle maneuver and even total liver isolation may help in assessing complex liver injuries. Appropriate control of active hepatic bleeding and packing is useful in these cases.[19] A fine line exists between sufficient packing to achieve tamponade and increasing the risk of ACS.

Surgery when dealing with renal and splenic injuries, especially in the face of expanding hematomas, active bleeding, and coagulopathy, has become more aggressive, with nephrectomy or splenectomy being preferred to conservation in the unstable patient with bleeding from these organs. Ureteric injury is either stented, exteriorized, or even ligated in certain instances.

Intestinal spillage is controlled by stapling bowel ends without definitive repair, occlusion with umbilical tape, suture, or towel tag ligation. Proper end-to-end anastamosis and maturation of a stoma is delayed until reoperation. Running suture can be used temporarily in some injuries. Gastric contents must be adequately drained.

Moore et al[16] recommend that up to 30 minutes can be spent continuing active resuscitation, rewarming, and correcting the coagulopathy. The patient's condition should be reassessed for possible reopening and control of active bleeding before transfer to ICU. This is the time to apply damage control to other anatomical sites. It may also be feasible to consider primary closure.

The abdomen needs to be covered to minimize further heat dissipation and to make packing effective. Preferred wall closure techniques include running nylon skin suture or towel clip closure in situations where the wound edges can be approximated. The sheath should be left strictly alone at this stage.

Towel clip closure has the benefit of rapid application, that is, 1 to 4 minutes.[13] If applied at 1-cm intervals, there is less chance of visceral herniation, and easy abdominal wall decompression is possible. The sheath is not damaged with this closure technique[7,10,13,14]; however, towel clips interfere with the radiologic visualization of the abdomen. A running nylon suture for skin closure, therefore, may be an easier alternative under these circumstances.

Packing and aggressive fluid resuscitation may make skin closure difficult. Bogota bag, silo formation, and the Opsite (Smith and Nephew, Andover, Massachusetts) sandwich techniques are alternatives. The Bogota bag makes use of an opened intravenous fluid or urologic irrigation bag sutured to skin edge with a continuous suture. It offers the advantage of allowing inspection of the abdominal contents through the bag. The Opsite sandwich technique requires no suturing. A sheet of Opsite or Steridrape (3M Corporation, St. Paul, Minnesota) is placed on an abdominal swab. Holes may be cut in the drape to assist drainage. The swab is placed into the open abdomen with the drape in contact with bowel. The swab edges are tucked sufficiently far under the wound edges so that visceral herniation does not take place. Suction drains are placed on the swab, and a second drape is then used to close off the wound.

Mesh can also be used as a temporary closure, but thought must be given to the expense involved and time required for suturing the mesh in place.

Stage 3: Restoration of Physiology in the ICU. Patients are then transferred to the ICU for further resuscitation, reversal of acidosis, correction of coagulopathy, and rewarming. With a core temperature <35˚C, normal coagulation will not occur, despite aggressive component replacement.[35] It is essential to prevent and treat hypothermia. Rewarming is continued throughout patient care. Active anatomical cavity rewarming may be initiated in theatre. In ICU, passive warming techniques including warm air blankets, warmed fluids, and humidified ventilator gases are routinely used. External heaters should also be considered. Other aggressive techniques include continuous arteriovenous rewarming.[40]

Resuscitation should be aimed at restoring the oxygen-carrying capacity, volume, and reestablishing a normal coagulation profile. This requires early transfusion of red blood cells, fresh-frozen

plasma, and minimizing overuse of crystalloids. At this stage, laboratory results of hemoglobin and clotting factors will act as a guide. Hypothermia may lead to erroneous clotting profile results because these tests are designed for optimal results at 37˚C. Platelets must be kept above 100,000/mm^3 in the presence of active bleeding. Cryoprecipitate should be given if the fibrinogen level is expected to be <1 g/L.[30] Vitamin K and calcium gluconate should also be considered. Recombinant activated factor VIIa (rFVIIa) may also have a role in resuscitation,[41, 42] which can be maximized by improved patient monitoring. Although this can be achieved with invasive methods such as pulmonary artery wedge pressure monitoring, which will allow for assessment, for example, of cardiac output and oxygen extraction calculation, other less-invasive techniques are preferable in trauma. Transesophageal ultrasound probes, which measure variations in the descending thoracic aorta between systole and diastole, can be used to derive hemodynamic variables that can guide resuscitation. This device is positional-dependent and may require constant readjustment during patient position change. Other devices include NiCO, which uses CO_2 measurements to estimate cardiac output.

These devices will assist in goal-directed resuscitation and help in reaching the end points of resuscitation. These include a systemic lactate <2.5 mmol/L, base deficit better than −4, a core temperature >35˚C, an international normalized ratio less than 1.25 times normal, and a urine output of >1 mL/kg.

The challenge in this period is in recognizing patients with ongoing bleeding that is not the result of a coagulopathic state. This requires clinical acumen aimed at assessment of failure to improve the hemodynamic status and increasing evidence of bleeding from the drains or intraabdominal bleeding. This may occur despite improving the core temperature or even the coagulation. Failure to recognize this will lead to reentry into the vicious cycle.

ACS should also be monitored during this period, as previously discussed. Renal failure occurs when intraabdominal (intravesical) pressures exceed 20 to 25 mm Hg[38, 43] and decompression is urgent.

Patients at this stage are likely to have respiratory complications from the direct chest trauma, the systemic effects of major trauma, and complications of multiple transfusions. Protective lung ventilation should be used early.

Stage 4: Relook or Definitive Surgery. Timing of relook surgery is generally at the surgeon's discretion, although other factors include the reversal of the triad, the availability of resources, and the initial indication for the damage control procedure. Reports have varied from 8 hours to 10 days.[7,12,16] As soon as the end points of resuscitation (as described here) are met, the patient can be taken for a planned relook. Earlier consideration should be given to patients with vascular injuries that were stented, or those with multiple ligated bowel loops.

Relook on demand will be based on the evidence of overt bleeding, or ongoing occult hypotension despite appropriate measures, and ACS. These patients should be carefully assessed before their condition worsens. The procedure is best done in theatre with an appropriate team and good lighting, but some patients' conditions may mandate that this will be performed in the ICU.

Definitive procedures should be performed prior to pack removal because this may induce bleeding, necessitating repacking, preventing completion of the intended operation. Proper exploration is essential to detect missed injuries.[19]

Once surgery is completed, a decision for another "directed relook" may be made. This will determine if a temporary closure is still relevant.

Stage 5: Abdominal Wall Closure. The abdominal wall can be closed as soon as all the definitive operations are completed, packs are removed, and bowel edema has resolved. This can be achieved by a standard continuous abdominal closure in layers. This is the first time that the sheath should be sutured. However, mobilization of this interstitial fluid may not be complete at this stage and temporary closure may still be necessary. Some cases may still require fluid restriction. Albumin and mannitol may be used to hasten this process, but this is not common practice.

Use of abdominal synthetic mesh for temporary closure may be appropriate; this can be followed with skin graft once granulation tissue is adequate. A suction dressing can increase granulation tissue formation and result in earlier grafting. The patient may require later abdominal wall reconstruction. Abdominal wall separation techniques may also be considered at that stage. When these delayed abdominal wall closure methods are used, patients should be monitored for associated complications, which include formation of enterocutaneous fistulae.

Extremity Injury: Vascular Injuries

The use of shunts has been described regularly,[19,36,43-49] although it was only in 1994, after Scalea et al[50] published a case report describing staged procedures for exsanguinating lower limb injury, that the concept of using damage control in extremity injury was formally introduced.

The principles are similar to those described previously, and a similar operative approach is applied.

Stage 1: Indication for Damage Control in the Limbs. This will include patients with the previously mentioned physiologic insults and/or with multiple life-threatening injuries. Mass casualty situations and patients who require transfer to tertiary institutions may also benefit from this approach. Isolated severe complex limb injuries requiring multiple teams may benefit from shunting as well.

Stage 2: The Operation. A higher index of suspicion is required, erring on the side of more aggressive surgery with shunt insertion, ligation, or packing. In making this choice it is important to remember the concept of life over limb, in which ligation with ultimate limb loss may be the only option. Barros D'Sa[51-53] cites warm ischemia time as the critical predisposing factor leading to necrosis and renal failure. Shunting, with immediate restoration of flow, avoids this.

The operative technique is ultimately unchanged, although delayed, and principles of vascular repair are maintained. The only variation is the insertion of the shunt, which serves to buy time, allowing adequate resuscitation and maintaining adequate distal perfusion. It must be inserted with care to avoid intimal separation on either proximal or distal limb. The shunt can be secured with clamps, or "vascular keepers," using umbilical tape, ligatures, or a Ramel tourniquet. In case of associated fractured limbs, the shunt should be well secured in a way that it will not dislodge during orthopaedic manipulation. The postoperative challenge is constant clinical assessment for patency on the shunt. On return to the operating room, one is operating on a stable patient, without pressure of time, and with additional expertise available.

Choice of a shunt should be determined by the size of the vessel that requires shunting. Other options available include the Javid, Brenner, and Sundt shunts, although any plastic or silastic tubing is suitable. The use of large-bore intravenous tubing has been described.[26]

The use of concurrent anticoagulation remains controversial,[47,54] keeping in mind the appropriate patient might already be coagulopathic. In the absence of contraindications, the use of anticoagulation should be considered. If the shunt is technically correctly placed in animal models, shunt potency can be maintained for at least 24 hours without anticoagulation.[55]

Fasciotomy should be considered mandatory when dealing with combined arterial and venous injuries. When the vein is ligated in the setting of the patient in extremis, fasciotomy at the initial operation is

mandatory to prevent subsequent compartment syndrome and its contribution to further ischemia of the limb. Postoperative bleeding should be managed appropriately with dressing.

Stage 3: Restoration of the Physiology. This remains identical, concentrating on restoration of physiologic parameters prior to return to the operating room for definitive repair. A controversial issue is the length of time that a shunt can be left in situ without further compromising the limb. Essentially, as long as distal perfusion is regularly monitored, there is no urgency to return for definitive repair, although reoperation is recommended as soon as acidosis, hypothermia, and coagulopathy have been corrected. In clinical circumstances, shunts have been left in situ for periods of up to 17 hours with good results.[47]

Stage 4: Relook and Final Vascular Repair. Standard surgical techniques of proximal and distal control, debridement of damaged segment, and insertion of appropriate conduit should be maintained. Conduit should not be left exposed.

Stage 5: Wound Cover. Wound closure is less challenging, with primary closure often feasible. However, split-thickness skin grafts and occasionally tissue transfer techniques are necessary. Associated soft tissue injury will determine the appropriate cover.

Extremity Injury: Orthopaedic Injury

Initial fear of early manipulation of fractured long bones was subsequently replaced by the concept of "early total care" in the 1980s. This change was encouraged by growing knowledge about fat embolism and the advantages of early bone fixation. However, on further analysis, patients with an Injury Severity Score of >18 did not show benefit from this approach, and an increasing understanding of the physiologic insult of prolonged theatre time during definitive orthopaedic management and success with damage control in the abdomen led to the new era of damage control orthopaedics.[56]

Patients with associated severe head injury, significant pulmonary contusions, or severe associated injuries could be compromised by intramedullary reaming of long bones. Studies have shown that reaming is associated with an increase in polymorphonuclear leucocytes, intramedullary pressure, blood loss, and interleukin 6 levels,[56,57] all of which play a part in adding insult to the systemic effects of trauma.

Controversy still exists about the effects of unreamed nails that can be approached intraarticularly in a retrograde fashion.[58] The future may lie in nails that can simultaneously irrigate and apply suction to the intramedullary space to reduce the pressure.

Stage 1: Patient Selection. This stage is aimed at recognizing the patient with multiple fractures, or fractures with associated severe injuries to the chest, head, and abdomen. Included in this group will be those with deranged metabolic status. Early recognition is essential!

Stage 2: The Emergency Operation. The plan in the initial management of these patients is to offer a stabilization that is quick and minimizes further insult to the metabolic state. The femur can be managed with external fixation. With proximal fractures, skeletal or skin traction may be appropriate. Other fractures can also undergo external fixation or immobilization. Associated soft tissue injury will require debridement and control of bleeding.

Unstable fracture of the pelvis should be recognized from the beginning. A temporary measure, such as a sheath around the pelvis, C-clamp, or external fixation in theatre may help to stabilize a patient. Care must be taken during removal of the sheath to avoid patient deterioration. Traction may be considered in vertical shear injuries. Where appropriate and the patient's condition allows it, an angiogram can be used. Very rarely, laparotomy with packing or ligation of internal vessels may be relevant.

Stage 4: Definitive Fixation. Further wound debridement will be carried out as necessary. The external fixation can be converted to an intramedullary nail once the condition has stabilized. This can be delayed to as long as 10 to 14 days after the initial injury.

Stage 5: Wound Cover. Wound management may require myocutaneous or myofacial flaps with skin graft for definitive cover.

Thoracic Injury

The triad of hypothermia, acidosis, and coagulopathy are the best predictors of the need for damage control in the thorax.[59, 60] A high index of suspicion should be maintained, as for other injuries.

Stage 1: Patient Selection. There are many situations in the chest in which damage control is, in fact, the definitive surgery. However, there are situations, like elsewhere, where control of hemorrhage in a temporizing fashion supersedes all therapeutic surgery. Injury pattern recognition is crucial.

Stage 2: Emergency Operation. Clinical presentation of these patients may even require emergency room thoracotomy. This is more relevant in penetrating than blunt trauma,[61] and will allow for cross-clamping of the aorta, internal massage, control of thoracic bleeding, and temporary control of cardiac injuries. If staples or temporary suture or balloon catheter are used to control cardiac lesions, these will be converted in theatre as part of ongoing operation.

In theatre, thoracic structures are not easily controlled with temporary maneuvers. Rapid and definitive control of hemorrhage and air leaks is required, using abbreviated techniques rather than staged procedures. The heart and great vessels require definitive repair, although packing of the pleural cavity remains an option should diffuse nonsurgical bleeding develop. Packing is easier at the apex of the lung or the deep posterior area. Mediastinal packing may be associated with cardiovascular compromise.

Traditionally, major pulmonary parenchymal and bronchial injuries were treated with lengthy, anatomical resections during which the patient often became coagulopathic with profound shock and, ultimately, demise. Alternatively, patients who presented in extremis were subjected to pneumonectomy, with mortality rates approaching 100%, or formal lobectomy, with a 55% mortality.[62]

A double-lumen endotracheal tube is ideal, allowing differential ventilation of each lung, and is protective in that bleeding from one side may not spill across to the other. Unfortunately, however, the instability of these patients does not usually allow the use of a double-lumen tube. They may not even have a cleared spinal injury, excluding optimal positioning for some. This may make the operation technically challenging.

To achieve hemostasis, minimize air leak, and avoid air embolism, simpler techniques like pulmonary tractotomy and nonanatomical pulmonary wedge resection may be used.[59, 61] Tractotomy with a lineal stapler will allow for better control of vessels in the parenchyma, which may be sutured appropriately. When simple pulmonorrhaphy is used, the endotracheal tube should be checked to observe those patients with internal bleeding who may compromise the normal bronchus on the opposite side.

In cases with severe bleeding, visualization can be improved by hilar control. This can be achieved with noncrushing clamps or a Foley's catheter across the hilum. Some authors have also twisted the hilum through 360º to achieve control, and reassessed it later, once the condition had stabilized.[63]

Esophageal injuries may require drainage at this stage if repair cannot be achieved within a short period of time.

Chest wall closure may be temporarily achieved with a mass closure technique. This is quick and will control bleeding from the muscle edge. Alternatively, methods similar to those used in the abdomen can be used.

Stages 4 and 5: Definitive Operation and Chest Wall Cover. Definitive repair will be achieved if not accomplished earlier. Debridement of wall and final closure will be achieved at this stage. Myofascial flaps may be necessary in large wall defects.

Head Injuries

The most critical part of head injury management is the prevention of secondary brain injury.[64,65] This includes optimizing the mean arterial pressure, hemoglobin level, and oxygenation, at both pulmonary and cerebral level. Intracranial lesions should be dealt with after any drainable lesions are managed appropriately. Care should be aimed at minimizing intracranial pressure using supportive and medical therapy.

Unfortunately, some of these patients in extremis may have not had a computerized tomographic scan assessment of the head injury. In the absence of localizing neurologic symptoms, attention will be given to stabilizing the general condition. Optimizing the general condition of the patient is essential in optimizing outcome of head injury. Patients with severe head injury with evidence of terminal signs may require review of further management in consultation with neurosurgeons.

Craniectomy may be considered to alleviate cerebral swelling, even after drainage of hematomas. Dural cover may be expanded with temporalis fascia or even synthetic dura.[66]

Intracranial pressure monitoring can aid in the management of severe head injury. Coagulation status must be optimized before insertion, to avoid bleeding complications. If indicated, these can be inserted in casualty, theatre, or even in the ICU.

Spinal Injuries

Improvement in resuscitation has increased awareness of associated spinal injuries. In-line immobilization of the spine is critical. Optimizing general condition will also improve outcome of reversible spinal injuries. Once the condition has settled, further examination of the spine and appropriate fixation can be achieved.[67]

In the absence of other priorities, the initial management includes the use of external traction, such as halo traction, and fixation of easily accessible spine (e.g., posterior segments). The more complex repairs are reserved for later.[68] Debridement of open wounds and antibiotic cover in contaminated spinal injuries form part of initial management. Acute bleeding points should be sought out, and angiography may be used as well.

Eye Injuries

Damage control has been applied in eye management.[69] Wounds should be debrided, major globe lacerations sutured, and the retinas reattached. Most other injuries can be repaired subsequently when the condition improves. Choroidal hemorrhage is better allowed to settle initially, before reconstructive work is done. If operated early, it may be complicated by severe bleeding.

The Pediatric Patient

Technology in radiology has improved in the past years. The surface area in children allows for its easier use. Care should be given to minimizing unnecessary radiation exposure when ultrasound investigation can suffice. Pediatric surgery has been at the forefront of conservative management in blunt injury.

Immediate exploration is indicated only if profound hemodynamic instability, continued hemorrhage, hollow viscus perforation, and major pancreatic ductal disruption exist. The majority of these types of cases are treated definitively at the initial operation and rarely require temporizing measures and relook operations.

Although often primarily repaired, packing the liver with or without subsequent embolization may be beneficial. Asensio et al,[70] in a relatively small study, showed that grade 4 and 5 liver injuries treated with packing and subsequent embolization had survival rates of 92% and 78%, respectively.

A general surgeon may have to deal with the pediatric patient, especially with respect to perihepatic packing and vascular management, including stenting.[71-74] This may allow for stabilization of a patient and subsequent transfer to a tertiary institution for definitive care.

The role of damage control in pediatric injury needs to be defined further.

Conclusion

Damage control is an operative technique that has been used for almost a century. It has evolved from being performed almost at random, to having reasonably clear indications and defined end points for each stage.

Damage control is gaining momentum in use in areas other than the abdomen and the chest. These include orthopaedic-related, eye, and spinal injury. The most important issue is that other members of the team understand the rationale and participate more constructively in this team approach to patient care.

In a select subgroup of patients, the procedure has been proven to reduce mortality significantly when related to injuries that were previously not survivable.[75]

Level I evidence is still lacking. Given the proven benefits of this concept, it seems unnecessary to perform such a study. We still believe that studies should be dedicated to better definition of indications and toward improving the technical aspects of the procedure.

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Damage Control Anesthesia

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Learning Objectives: 1) To understand the essential components of damage control anesthesia. 2) To learn the resuscitation goals related to damage control surgery.

Abstract

The role of the anesthesiologist in the damage control approach to the patient is of utmost importance and can have a profound impact on the patient's ultimate outcome. The anesthesiologist is vital in overseeing the process of fluid resuscitation to optimize hemostasis and long-term survival. Another critical role of the anesthesiologist is prevention of a second hit caused by recurrent shock. It is essential for any anesthesiologist who cares for unstable trauma patients to know and understand the concepts of damage control anesthesia. This article presents these concepts in detail.

The Evolution of Damage Control

"Damage control" is one of the buzzwords in modern trauma care, a bandwagon that everyone has boarded. Yet what does it really mean? Put most simply, damage control is a plan of care for the badly injured patient. In the 1970s, thinking was focused on "the Golden Hour," a fictional construct intended to emphasize the importance of rapid diagnosis, surgery, and resuscitation.[1] This was a laudable goal, and necessary for the times. This led in the 1980s to a "fix everything now" mentality. Sometimes this had advantages: early fracture fixation improved patient mobilization and reduced the incidence of pulmonary complications[2]; early diagnosis and treatment of aortic injuries doubtless saved lives.[3] In other cases, though, prolonged surgical procedures may have done more harm than good. With the crack cocaine epidemic of the late 1980s came the need for a new paradigm.

Damage control was invoked as a means to avoid "operating the patient to death," a way of limiting prolonged surgical procedures that increased blood loss, and with it the potential for hypothermia, coagulopathy, and acidosis. Instead, only those procedures that were necessary for hemostasis were done, while others, such as anastamosis of discontinuous bowel segments or definitive closure of the abdomen, were delayed until the patient was more stable. Damage control was a technique confined to the abdomen, and known primarily by trauma surgeons.

In the new millennium, the concept of damage control has expanded. There is now a realization that prolonged early surgery may be deleterious to the patient even in the absence of hypoperfusion or coagulopathy. The "second hit" produced may become the straw that breaks the camel's back, leading to fatal exacerbation of traumatic brain injury (TBI), to the systemic inflammatory response syndrome, to the development of acute lung injury, or to early sepsis. This realization in turn has broadened the thinking about damage control, so that it is now applied to orthopaedic procedures (external fixation vs. intramedullary nailing of femur fractures),[4] intrathoracic surgery (delayed repair of stable aortic injuries), and even neurosurgery (craniotomy without replacement of the bone flap, even in the absence of massive edema). The previous articles in this issue of *TraumaCare* have described damage control in some detail as it is seen today by experienced and innovative trauma surgeons.

To date there has been nothing written about the anesthesia component of damage control, and little public discussion of what the anesthesiologist can do to facilitate the overall goals of the trauma team. To some degree, this reflects the lack of specialized trauma anesthesiologists in the United States; in addition, it stems from a failure to recognize the critical importance of anesthetic management in the early care of the trauma patient. Consider Table 1, a simple list of early management goals for the severely injured patient. When one thinks about how many of these variables are under the control of the anesthesiologist it becomes obvious that anesthetic management may be as critical as surgical management in achieving the best possible patient outcome.

Table 1. Goals for Damage Control in the Severely Injured Patient

- Stable airway and oxygenation
- Hemostasis—control of life-threatening hemorrhage
  - Exploratory laparotomy or thoracotomy
  - Rapid, wide exposure
  - Excision over repair of "expendable" organs
  - Focus on hemostatic procedures only
    - -Vessel ligation or repair (avoid grafting if possible)
    - -Packing for diffuse bleeding
  - Temporary closure
  - Angiographic embolization in selected cases
- Effective analgesia and sedation
- Appropriate blood composition:
  - Oxygen-carrying capacity (red blood cells)
  - Clotting potential (platelets, clotting factors)
  - Chemistry (especially calcium, glucose, potassium, chloride)
- Stabilization/reversal of tissue acidosis
- Normothermia

Table 2. The Essentials of Damage Control Anesthesia

- Airway and ventilator management
  - Rapid sequence intubation
  - Titration of ventilation
- Control of bleeding
  - Deliberate hypotensive resuscitation
  - Maintenance of blood composition
- Preservation of homeostasis
  - Normothermia
  - Restored and sustained end-organ perfusion
- Analgesia and sedation

The author has a financial interest in one of the products listed in this article.

The Goals of Damage Control Anesthesia

Table 2 is a modification of Table 1, focused on the components of damage control that are in the hands of the anesthesiologist. Some activities, such as airway control and provision of adequate analgesia, will be obvious to any practitioner. Other activities, such as control of hemostasis and avoidance of the second hit, require a deeper understanding of the trauma patient and the ways in which the anesthesiologist contributes to outcome. The remainder of this article will illustrate these points in detail.

Airway and Ventilator Management

The hemodynamically unstable patient is likely to be in pain, is likely to require one or more surgical procedures, and has a very high probability of clinical deterioration. For all of these reasons, early definitive airway management is strongly recommended.[5] Higher levels of oxygenation can be assured, the airway can be protected against aspiration, and adequate levels of analgesia and sedation can be provided without fear of hypoventilation or apnea. Intubation should occur early in the diagnostic phase, prior to sending the patient for a computerized tomographic (CT) scan, with the equipment and personnel on hand to do the job correctly.

Because the damage control approach emphasizes speed in diagnosis and therapy, the approach to intubation should be as swift and certain as possible. Figure 1 is the emergency airway management algorithm used at the Shock Trauma Center in Baltimore. This approach depends on the presence and participation of a senior anesthesiologist and experienced trauma surgeon, and focuses on achieving the best possible conditions on the first attempt at intubation.[6] Members of the trauma team are delegated to provide manual in-line cervical stabilization and cricoid pressure. Oxygen is provided throughout the procedure by bag-valve-mask assisted ventilation. A small, titrated amount of a sedative agent (thiopental or etomidate) is administered, immediately followed by a generous dose of succinylcholine (1.5 mg/kg). Intubation is attempted two or three times, facilitated by a gum-elastic bougie (intubating stylet) on the second attempt, and personally performed by the most experienced provider present on the last attempt. If successful endotracheal passage of the tube cannot be documented by the presence of exhaled carbon dioxide, the team moves rapidly to placement of a laryngeal mask airway (LMA). Clinical deterioration of the patient or inability to establish ventilation with the LMA is an indication for emergent cricothyroidotomy. Successful LMA placement may be followed by either cricothyroidotomy or formal tracheostomy, depending on the patient's hemodynamic status.

Because the patient was unstable even before the start of airway management, there is little role for awake intubation techniques or fiberoptic bronchoscopy, and no thought given to waking the patient if attempts at intubation are unsuccessful. Despite the simplicity of this approach and the chaotic environment in which it is usually applied, an experienced team can achieve excellent results. Quality management data from the Shock Trauma Center over the past decade has shown a consistently low need for surgical airway access, on the order of 0.1% of all emergency intubations.

In addition to skill at rapid sequence intubation, the anesthesiologist can contribute important insights to the ventilatory management of hemorrhaging patients. Already noted was the need to limit the dose of sedative medication in unstable patients because of the potential for hemodynamic collapse associated with a sudden drop in serum catecholamine levels. Similarly, initial tidal volumes and ventilating pressure should be kept as low as necessary to maintain oxygen saturation because positive intrathoracic pressure

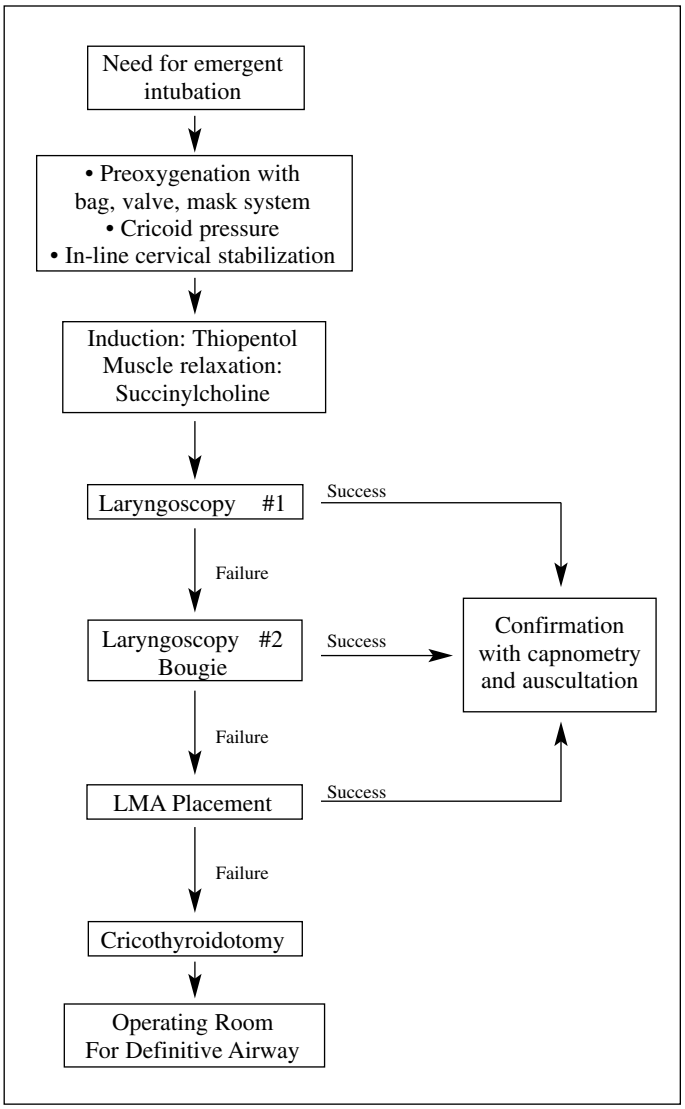

Figure 1. The emergency airway management algorithm currently in use at the Shock Trauma Center (January 2006). This algorithm is reviewed and updated each year to include new drugs (such as etomidate) or techniques (such as the LMA). Adapted from Dutton and McCunn.[6]

will decrease venous return to the chest, decrease right atrial filling, and reduce cardiac output. We commonly employ initial tidal volume settings of 5 to 6 mL/kg, with positive end-expiratory pressure (PEEP) of 5 cm, and a rate of 8 to 10 breaths per minute. Higher levels of PEEP, and even the conversion to pressure-controlled modes of ventilation, may well be necessary later in the patient's course to treat acute lung injury associated with trauma and massive resuscitation.[7] In the early phase, however, the greater risk is ongoing hemorrhage and associated hemodynamic instability.

Control of Bleeding

Most of the techniques for hemostasis described in this issue of *TraumaCare* are the province of the surgeon or angiographer. In clinical practice there is scant attention paid to the influence of fluid resuscitation on the rate of hemorrhage and the incidence of

rebleeding. It is true, however, that the kind and quantity of fluids administered will have an impact on the patient's clinical course. Animal models of uncontrolled hemorrhagic shock using standardized injuries and surgery have shown as much as a 60% difference in outcome based on the fluid resuscitation regimen.[8] In the clinical arena it is likely that the more this process is knowledgeably and consistently managed by a single individual—ideally the anesthesiologist—the better the patient will do.

Skillful resuscitation of the unstable patient requires adequate intravenous (IV) access, frequent diagnostic feedback, and experienced management. Placement of at least two large-bore (16 gauge or greater) IVs is a priority in the early management of the bleeding patient, usually accompanied by sampling of blood for laboratory assays (complete blood count, electrolytes, coagulation studies, lactate, and toxicology) and for blood bank cross-matching.[5] Failure to find peripheral IV access is an indication for central venous access, utilizing a pulmonary-artery introducer sheath or special trauma access line. At least one access site should be above the diaphragm in any patient with the possibility of abdominal or pelvic bleeding.

Once access is established, all IV fluids and blood products should be administered in defined doses, just as any other anesthetic drug, titrated to the patient's vital signs, the clinical scenario, and the available laboratory data. The anesthesiologist should be concerned with both the volume of fluids infused and the resulting composition of the patient's blood.

Fluid volume should be titrated by administration of 200- to 500-mL boluses to maintain cardiac output and blood pressure at a low, but sustainable, level.[9] Deliberate hypotensive resuscitation has been shown to enhance hemostasis in numerous animal studies, and was supported by two large prospective clinical trials.[10,11] Fluid restriction facilitates hemorrhage control in numerous ways: lower blood pressure enhances regional vasoconstriction and facilitates clot formation and stabilization. Controlled volume administration reduces the development of hypothermia and limits dilution of red cell mass, platelets, and clotting factors. Weighed against this is the potential for worsening hypoperfusion, with a risk for increased acidosis and organ system injury. The anesthesiologist must steer a careful course between the Scylla of shock and the Charybdis of rebleeding, understanding that deliberate hypotension in the face of ongoing hemorrhage is an inherently unstable situation (Figure 2). This is why fluids should be administered in small boluses, with close attention to the rate of surgical bleeding, to the vital signs, and to frequent laboratory studies.

Maintenance of blood composition is at least as important to achieving hemostasis as careful control of the vital signs. Logistical restraints in the trauma system (i.e., the speed with which blood products can be prepared and delivered to the bedside; the turnaround time for laboratory assays) make it likely that any trauma patient who is bleeding heavily will experience at least transient derangements in blood composition. Use of crystalloid solutions in prehospital and early emergency department care will lead to a rapid drop in hematocrit. Aggressive replacement of red blood cells (RBCs) will lead in turn to deficiency of clotting factors and platelets, especially as these are consumed at the site of vascular injury (and reconsumed when rebleeding occurs). Many institutions maintain massive transfusion protocols that recommend the administration of defined ratios of RBCs, plasma, and platelets. In reality, even the use of 1 unit of RBC matched to 1 unit of plasma and 1 unit of platelets will maintain only a barely acceptable blood composition, as illustrated in Figure 3. Fresh whole blood has obvious advantages as a resuscitative fluid for the actively bleeding patient. Because it is viable after collection for less time than is usually needed for viral screening, whole blood is available in only a few of the world's trauma systems (e.g., Israel, the U.S. military).

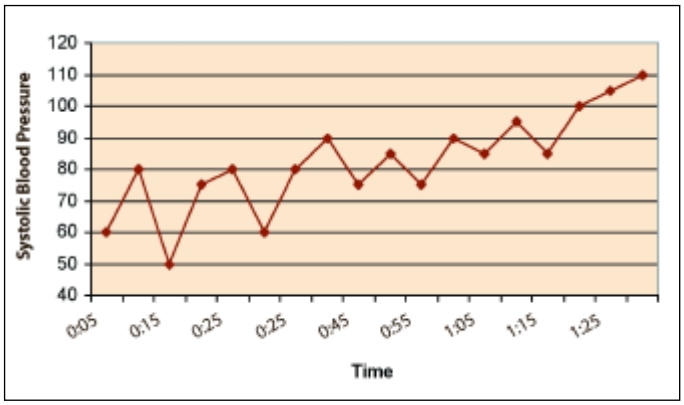

Figure 2. Typical variations in blood pressure seen during damage control surgery. Ongoing hemorrhage, fluid therapy, and administration of anesthetic drugs cause swings in blood pressure. Stability is achieved when bleeding is controlled, a deep anesthetic level is attained, and the patient is euvolemic.

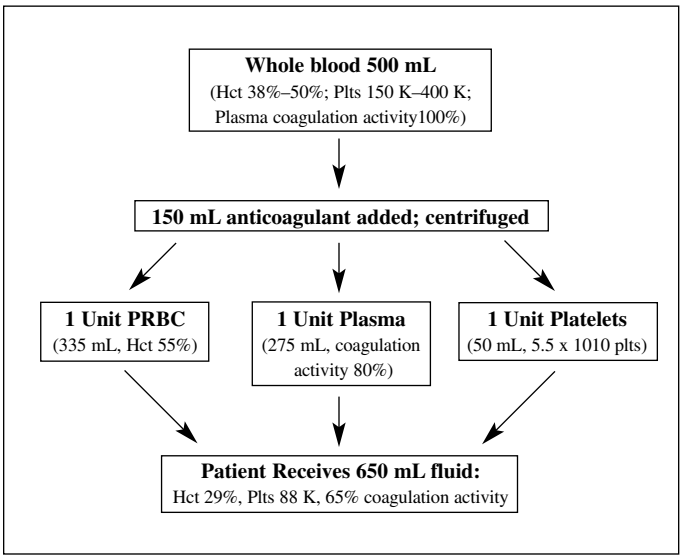

Figure 3. Final administered concentrations of red cells, clotting factors, and platelets achieved when a unit of whole blood is donated and fractionated, then given as separate components during damage control anesthesia. Hct, hematocrit; Plts, platelets; PRBC, packed red blood cells.

Recommendations for fluid resuscitation during damage control are listed in Table 3. Sustained hypotension is desirable unless contraindicated by patient history, by injury to the brain or spinal cord, or by laboratory evidence of increasing acidosis. Stabilization of blood pressure without recourse to ongoing fluid administration is the best clinical sign of successful hemostasis (see Figure 2). Blood products should be administered as early as possible, with uncross-matched type-O RBC recommended at the time of admission in patients who are obviously unstable.[12] Plasma and platelets should follow as soon as they are available from the blood bank. In the patient with significant ongoing blood loss, ratios of blood products similar to whole blood will be required to reach simultaneously acceptable levels of hematocrit, clotting factor concentration, and platelet count. Crystalloid administration should be reduced or eliminated all together once blood products are available. Frequent laboratory assay (every 1 hour or even more often) is indicated in the patient who is actively hemorrhaging.

Table 3. Resuscitation Goals *During* Damage Control Surgery*

- Systolic blood pressure 90 mm Hg
- Heart rate <120 beats per minute
- Pulse oximeter functioning, SaO_2 >95%
- Urine output present
- $PaCO_2$ <50 torr
- pH >7.25
- Hematocrit >25%
- Lactate stable or decreasing
- Ionized calcium >1.0
- International normalized ratio <1.6
- Platelets >50,000
- Normothermia
- Deep anesthesia

*Lower blood pressure may be tolerated as long as acidosis is not worsening.

Close attention is required to maintain blood composition and chemical equilibrium. Serum ionized calcium levels will fall rapidly during massive transfusion, caused by both acidemia and the chelating effects of sodium citrate administered with banked blood products. Calcium replacement will be necessary in any patient receiving more than 4 to 6 units of RBC in an hour.[13] Increase in blood pressure in response to calcium injection is an empiric indication of its value, and calcium administration should be considered while awaiting laboratory confirmation in any bleeding patient with hypotension who is unresponsive to bolus fluid administration. Other electrolytes will seldom become deranged during ongoing resuscitation with isotonic crystalloids and blood products. Although bicarbonate administration may have a transient pressor effect in the patient with extreme acidosis, it is unlikely that this therapy offers any benefit over further fluid administration and improvement of tissue perfusion. Similarly, derangements in sodium, potassium, or chloride levels indicate either a profound failure of resuscitation (such as disproportionate use of normal saline or hemolysis of mismatched RBC) or a state of shock so deep that survival is unlikely.

A final role of the anesthesiologist in achieving hemostasis is the decision to administer systemic procoagulant agents. Recent experience with the use of recombinant activated human clotting factor VII (FVIIa) has shown promise in reversing traumatic coagulopathy and improving long-term outcomes from hemorrhagic shock.[14,15] Although there is substantial enthusiasm in the trauma community for the "off-label" use of this agent, a definitive understanding of the potential risks (e.g., inappropriate thrombosis) and benefits (e.g., earlier hemostasis) of this approach will depend on the outcome of the multicenter international trials now underway. At the Shock Trauma Center we consider the use of FVIIa when severe hemorrhagic shock persists beyond the administration of the first 6 to 8 units of RBC and plasma, or when an underlying coagulopathy, such as warfarin use, is complicating the clinical picture.

Preservation of Homeostasis

The anesthesiologist's responsibility to the critically injured patient does not end when bleeding is controlled. Resuscitation must be completed, and the patient's physiology returned as close to normal as possible, or as close to the optimum for recovery as can be arranged.

Table 4. Resuscitation Goals *After* Damage Control Surgery*

- Systolic blood pressure >100 mm Hg
- Heart rate <90 beats per minute
- Pulse oximeter functioning, SaO_2 >97%
- Urine output >0.5 mL/kg/hr
- $PaCO_2$ <40 torr
- pH >7.35
- Hematocrit >20%
- Lactate normal
- Ionized calcium >1.0
- International normalized ratio <2
- Platelets >50,000
- Normothermia
- Cardiac output normal or high
- Light sedation (comfortable, able to initiate spontaneous ventilation)

* Normal lactate is the best marker for adequacy of resuscitation. A lower hematocrit can be tolerated in a patient who is not actively bleeding.

End points for resuscitation are listed in Table 4. Once bleeding is definitively controlled, fluid administration should be continued until the patient is demonstrably euvolemic. Simple normalization of vital signs should not be equated with restoration of tissue perfusion. The phenomenon of occult hypoperfusion is common in young trauma patients; vasoconstriction to compensate for inadequate fluid volume may produce a normal blood pressure even in the presence of ongoing organ system ischemia.[16] Resolution of metabolic acidosis on arterial blood gas analysis and normalization of serum lactate indicate a systemic return to aerobic metabolism and recovery from hemorrhagic shock. Multiple studies have shown that the faster lactate clears, the lower the patient's subsequent incidence of multiple organ system failure and death.[17,18] Other means to confirm recovery from shock include maximization of cardiac output in response to fluid administration, normalization of gastric or sublingual tissue acidosis (measured by minimally invasive tonometry or capnometry),[19] and tolerance of a normal level of anesthesia and analgesia.

Although deliberate hypothermia has been suggested as a treatment for hemorrhagic shock and has demonstrated good results in animal trials, this therapy is not yet recommended for trauma patients.[20] Hypothermia potentiates coagulopathy and increases the incidence of infection and sepsis. Further, the process of warming from hypothermia may impose additional cardiac stress on patients with limited reserve. Until hypothermia management protocols that avoid these risks are developed and validated, the clinical practitioner is better served by keeping the patient warm throughout the resuscitation. Fluid boluses should be administered through a warming system, a forced hot-air surface warmer should be used whenever possible, and the operating room (OR) environment should be kept warmer than usual.

Homeostasis is especially important to the brain-injured patient because outcomes are significantly worse following similar levels of TBI when a second hit is allowed to occur.[21] Retrospective data suggest that any single episode of hypotension or hypoxia increases the mortality from TBI by fourfold, and the occurrence of both in the same patient leads to a tenfold greater chance of dying. The effect appears to be mediated by increased inflammation in the vicinity of the brain tissue injured in the original trauma; this phenomenon is probably a representative model for effects throughout the body. Future

resuscitation research is closely focused on this point, and active manipulation of the postshock inflammatory cascade is likely to be part of the anesthesiologist's available resources a decade from now.

Analgesia and Sedation

The final task of the anesthesiologist in the damage control scenario is also the most obvious: the provision of pain relief and unconsciousness for the patient. Despite the fact that this is the most basic of goals for the anesthesiologist, there is often a reluctance to medicate the patient with ongoing hemorrhagic shock because of hemodynamic instability. It is true that any analgesic or sedative medication is likely to lower the unstable patient's blood pressure; in fact, an unexpected decline in blood pressure may help to suggest a decrease in blood volume. Hypotension is caused both by the direct vasodilatory and negative inotropic effects of the medication itself (as with propofol, midazolam, or isoflurane) and the indirect decrease in serum catecholamines that accompanies analgesia and sedation (as with fentanyl, etomidate, or ketamine).

Exacerbation of hypotension should not be a contraindication to anesthesia, however, but only a sign that it should be used with caution. Hemorrhage volumes and the duration of bleeding are known to be worse in the vasoconstricted subject. Vasoconstriction also reduces end-organ perfusion and contributes to exaggerated up-and-down swings in blood pressure. For these reasons, vasopressor agents have long been avoided in hypotensive trauma patients, with the thought that fluid administration was a more physiologic therapy. Although the concept has not been systematically studied in humans, it is logical to extend this thinking to the active use of anesthetic agents to move from a vasoconstricted state to a vasodilated one. It is known, for example, that similar blood loss is much better tolerated in the anesthetized human or animal than in one who is awake and alert.[22]

For these reasons, it is our customary practice to achieve a deep and stable level of anesthesia as early as possible in the care of the unstable trauma patient. We will begin loading the patient with fentanyl early in the resuscitation, using small doses at first and responding to drops in blood pressure (below our desired hypotensive target) with boluses of fluid. Our goal is to achieve a "cardiac anesthetic": 50 to 100 mcg/kg over the first few hours. Because fentanyl lacks any direct effect on the cardiovascular system, we expect the patient to sustain a low, stable blood pressure once hemorrhage is resolved and intravascular volume restored. We are not intending to awaken and extubate the patient at the end of resuscitation, and have little concern with inducing a long-lasting anesthetic. Indeed, the deeply narcotized patient is easier to manage through periods of early transport outside the OR to CT scan, angiography, magnetic resonance imaging, and the intensive care unit. Deep anesthesia also makes it easier to assess the patient's fluid volume over the remainder of the resuscitation because hypovolemia will cause an immediate decrease in blood pressure in the patient in whom catecholamine release has been blocked. Our anecdotal experience with this technique has been good, and we are presently exploring methods for a controlled prospective trial.

Conclusion

Conduct of anesthesia and fluid resuscitation is integral to the damage control approach to the patient with ongoing traumatic hemorrhage, and the actions of the anesthesiologist can have a profound impact on the patient's ultimate outcome. In addition to facilitating rapid transport to diagnostic studies and the OR, the anesthesiologist is best positioned to oversee the process of fluid resuscitation, adjusting the kind and quantity of fluid administered to optimize hemostasis and long-term survival. The anesthesiologist is also critical to avoidance of a second hit caused by recurrent shock, and may further benefit the patient by the careful, reasoned administration of analgesics and sedatives. Understanding the concepts of damage control anesthesia is important to any anesthesiologist who cares for unstable trauma patients.

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BOOK REVIEW

Going International A Course Catalogue of International Courses

Compiled by Dr. Gerhard Polak

This is the second review of the compendious catalogue edited by Dr. Gerhard Polak, who started this project in the 1990s in an attempt to facilitate specialized training and education for care providers intending to participate in international missions. In the ensuing years, this catalogue has been expanded to detail 2,500 high-quality courses around the globe.

Dr. Polak has covered every possible aspect of health care and humanitarian aid in his catalogue. From hands-on courses for first responders to academic lectures on tropical diseases, from psychological preparation and debriefing of first responders to advanced administrative subjects, quality assurance and management and organization of preventive health care, there are educational possibilities listed for every person involved in public health.

The catalogue is organized in two sections, "part A" listing courses and "part B" providing names, contact information, and website information on organizations of international collaboration and education.

The courses are arranged in chapters with topics such as "management and quality assurance," "humanitarian assistance and complex emergencies," seven such chapters in all. Every listing is accompanied not only with obvious data such as dates and location, but also with a standardized list of information including the course language, price, goals and objectives, target audience, faculty, and credit hours. Every chapter is preceded by a carefully written introduction, which by themselves make the catalogue worth reading. A thoughtfully arranged index greatly facilitates finding a desired course.

As stated previously, this catalogue is a fantastic resource for anyone intending to work in foreign health care systems, especially if the goal is a humanitarian mission.

Dr. Polak has been a key facilitator for such missions originating in Austria and has witnessed the sometimes dramatic and lasting problems befalling health workers who are not properly prepared. With this catalogue, he has masterfully accomplished his goal of providing every interested health worker, whether administrator, politician, or hands-on first responder, with an optimally compiled list of educational opportunities—many of them only a mouse click away.

Niels Chapman, MD
Department of Anesthesiology
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Albuquerque, NM, USA

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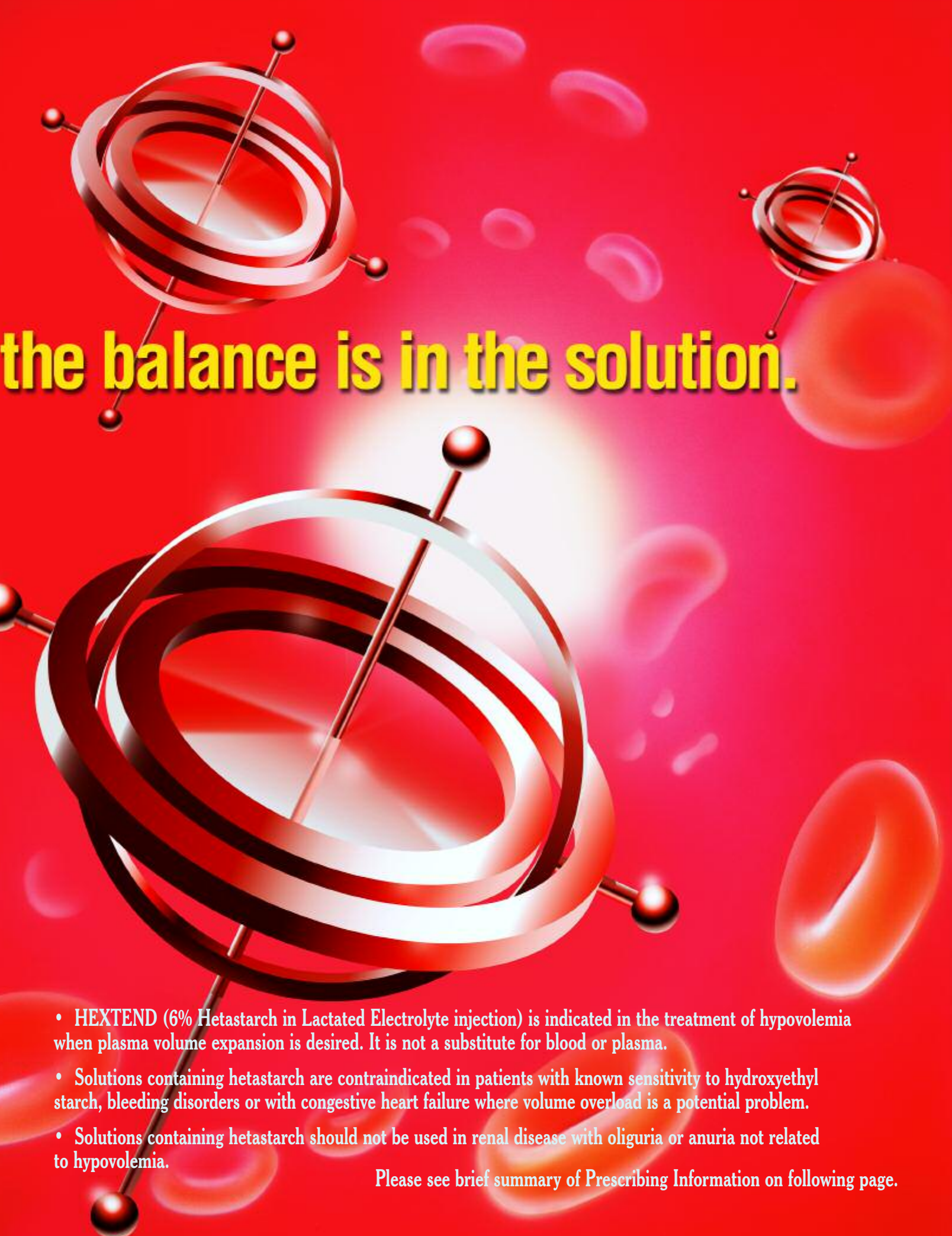

- HEXTEND (6% Hetastarch in Lactated Electrolyte injection) is indicated in the treatment of hypovolemia when plasma volume expansion is desired. It is not a substitute for blood or plasma.
- Solutions containing hetastarch are contraindicated in patients with known sensitivity to hydroxyethyl starch, bleeding disorders or with congestive heart failure where volume overload is a potential problem.
- Solutions containing hetastarch should not be used in renal disease with oliguria or anuria not related to hypovolemia.

Please see brief summary of Prescribing Information on following page.

BRIEF SUMMARY

HEXTEND®

6% Hetastarch in Lactated Electrolyte Injection

Flexible Plastic Container

INDICATIONS AND USAGE

HEXTEND (6% Hetastarch in Lactated Electrolyte Injection) is indicated in the treatment of hypovolemia when plasma volume expansion is desired. It is not a substitute for blood or plasma.

CONTRAINDICATIONS

Solutions containing hetastarch are contraindicated in patients with known hypersensitivity to hydroxyethyl starch or with bleeding disorders or with congestive heart failure where volume overload is a potential problem. Solutions containing hetastarch should not be used in renal disease with oliguria or anuria not related to hypovolemia.

Solutions containing lactate are NOT FOR USE IN THE TREATMENT OF LACTIC ACIDOSIS.

WARNINGS

Solutions containing calcium should not be administered simultaneously with blood through the same administration set because of the likelihood of coagulation.

Life threatening anaphylactic/anaphylactoid reactions have been rarely reported with solutions containing hetastarch; death has occurred, but a causal relationship has not been established. Patients who develop severe anaphylactic/anaphylactoid reactions may need continued supportive care until symptoms have resolved.

Hypersensitivity reactions can occur even after solutions containing hetastarch have been discontinued.

Solutions which contain potassium should be used with great care, if at all, in patients with hyperkalemia and severe renal failure and in situations in which potassium retention is present.

Solutions containing sodium ions should be used with great care, if at all, in patients with congestive heart failure and severe renal insufficiency and in clinical states in which edema with sodium retention occurs.

In patients with diminished renal function, administration of solutions containing sodium or potassium ions may result in sodium or potassium retention.

Solutions containing lactate ions should be used with great care in patients with metabolic or respiratory alkalosis. The administration of lactate ions should be performed with great care when dealing with conditions in which an increased level or an impaired utilization of these ions occurs, such as severe hepatic insufficiency.

DO NOT USE IN LEUKAPHERESIS.

Usage in Plasma Volume Expansion

Large volumes of isotonic solutions containing 6% hetastarch (HEXTEND or Hetastarch Injection) may transiently alter the coagulation mechanism due to hemodilution and a mild direct inhibitory action on Factor VIII. Hemodilution by isotonic solutions containing 6% hetastarch may also result in a 24 hour decline of total protein, albumin, and fibrinogen levels and in transient prolongation of prothrombin, activated partial thromboplastin, clotting, and bleeding times.

Hematocrit may be decreased and plasma proteins diluted excessively by administration of large volumes of isotonic solutions containing 6% hetastarch. Administration of packed red cells, platelets, and fresh frozen plasma should be considered if excessive dilution occurs.

In randomized, controlled, comparative studies of Hetastarch Injection (n = 92) and Albumin (n = 85) in surgical patients, no patient in either treatment group had a bleeding complication and no significant difference was found in the amount of blood loss between the treatment groups.

HEXTEND has not been adequately evaluated to establish its safety in situations other than treatment of hypovolemia in elective surgery. In some cases, the use of isotonic solutions containing 6% hetastarch has been associated with coagulation abnormalities in conjunction with an acquired, reversible von Willebrand's-like syndrome and/or Factor VIII deficiency when used over a period of days. Replacement therapy should be considered if a severe Factor VIII or von Willebrand deficiency is identified. If a coagulopathy develops, it may take several days to resolve. Certain conditions may affect the safe use of isotonic solutions containing 6% hetastarch on a chronic basis. For example, in patients with subarachnoid hemorrhage where an isotonic solution containing 6% hetastarch is used repeatedly over a period of days for the prevention of cerebral vasospasm, significant clinical bleeding may occur. Intracranial bleeding resulting in death has been reported with the use of Hetastarch Injection.

PRECAUTIONS

General

The possibility of circulatory overload should be kept in mind. Caution should be used when the risk of pulmonary edema and/or congestive heart failure is increased. Special care should be exercised in patients who have impaired renal clearance since this is the principal way in which hetastarch is eliminated and in clinical states in which edema with sodium retention occurs.

Indirect bilirubin levels of 8.3 mg/L (normal 0.0-7.0 mg/L) have been reported in 2 out of 20 normal subjects who received multiple infusions of Hetastarch Injection. Total bilirubin was within normal limits at all times; indirect bilirubin returned to normal by 96 hours following the final infusion. The significance, if any, of these elevations is not known; however, caution should be observed before administering isotonic solutions containing 6% hetastarch to patients with a history of liver disease.

If a hypersensitivity effect occurs, administration of the drug should be discontinued and appropriate treatment and supportive measures should be undertaken (see ***WARNINGS***).

Caution should be used when administering solutions containing hetastarch to patients allergic to corn because such patients can also be allergic to hetastarch.

Clinical evaluation and periodic laboratory determinations are necessary to monitor changes in fluid balance, electrolyte concentrations, acid-base balance, and coagulation parameters during prolonged parenteral therapy or whenever the condition of the patient warrants such evaluation.

Solutions containing dextrose should be used with caution in patients with known subclinical or overt diabetes mellitus.

Caution must be exercised in the administration of parenteral fluids, especially those containing sodium ions, to patients receiving corticosteroids or corticotropin.

Potassium containing solutions should be used with caution in the presence of cardiac disease, particularly in digitalized patients or in the presence of renal disease.

Solutions containing lactate ions should be used with caution as excess administration may result in metabolic alkalosis.

Elevated serum amylase levels may be observed temporarily following administration of solutions containing hetastarch although no association with pancreatitis has been demonstrated. Serum amylase levels cannot be used to assess or to evaluate for pancreatitis for 3-5 days after administration of solutions containing hetastarch. Elevated serum amylase levels persist for longer periods of time in patients with renal impairment. Solutions containing hetastarch have not been shown to increase serum lipase.

One report suggests that in the presence of renal glomerular damage, larger molecules of hetastarch can leak into the urine and elevate the specific gravity. The elevation of specific gravity can obscure the diagnosis of renal failure.

Hetastarch is not eliminated by hemodialysis. The utility of other extracorporeal elimination techniques has not been evaluated.

If administration is by pressure infusion, all air should be withdrawn or expelled from the bag through the medication port prior to infusion.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term studies of animals have not been performed to evaluate the carcinogenic potential of hetastarch.

Teratogenic Effects: Pregnancy Category C.

Hetastarch Injection has been shown to have an embryocidal effect on New Zealand rabbits when given intravenously over the entire organogenesis period in a daily dose 1/2 times the maximum recommended therapeutic human dose (1500 mL) and on BD rats when given intraperitoneally, from the 16th to the 21st day of pregnancy, in a daily dose 2.3 times the maximum recommended therapeutic human dose. When Hetastarch Injection was administered to New Zealand rabbits, BD rats, and Swiss mice with intravenous daily doses of 2 times, 1/3 times, and 1 times the maximum recommended therapeutic human dose, respectively, over several days during the period of gestation, no evidence of teratogenicity was evident. There are no adequate and well controlled studies in pregnant women. HEXTEND should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether hetastarch is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when HEXTEND is administered to a nursing woman.

Pediatric Use

The safety and effectiveness of HEXTEND in pediatric patients have not been established. Adequate, well-controlled clinical trials to establish the safety and effectiveness of HEXTEND in pediatric patients have not been conducted. However, in one small double-blind study, 47 infants, children, and adolescents (ages 1 year to 15.5 years) scheduled for repair of congenital heart disease with moderate hypothermia were randomized to receive either Hetastarch Injection or Albumin as a postoperative volume expander during the first 24 hours after surgery. Thirty-eight children required colloid replacement therapy, of which 20 children received Hetastarch Injection. No differences were found in the coagulation parameters or in the amount of replacement fluids required in the children receiving 20 mL/kg or less of either colloid replacement therapy. In children who received greater than 20 mL/kg of Hetastarch Injection, an increase in prothrombin time was demonstrated ($p = 0.006$). There were no neonates included in this study.

Geriatric Use

Of the total number of patients in clinical trials of HEXTEND (n=119), 30% were 65 or older while 12% were 70 or older. Other reported experience with Hetastarch Injection has not identified differences in responses between elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

This drug is known to be substantially excreted by the kidney, and the risk of toxic reactions to this drug may be greater in patients with impaired renal function. Because elderly patients are more likely to have decreased renal function, care should be taken in dose selection, and it may be useful to monitor renal function.

ADVERSE REACTIONS

In clinical trials comparing the plasma volume expanding properties of HEXTEND (n=60) with those of Hetastarch Injection (n=59), there were no significant differences in the number of adverse or serious adverse events between the two groups.

Reported adverse reactions with isotonic solutions containing 6% hetastarch include:

General

Hypersensitivity (see ***WARNINGS***).

Death, life-threatening anaphylactic/anaphylactoid reactions, cardiac arrest, ventricular fibrillation, severe hypotension, non-cardiac pulmonary edema, laryngeal edema, bronchospasm, angioedema, wheezing, restlessness, tachypnea, stridor, fever, chest pain, bradycardia, tachycardia, shortness of breath, chills, urticaria, pruritus, facial and periorbital edema, coughing, sneezing, flushing, erythema multiforme, and rash.

Cardiovascular

Circulatory overload, congestive heart failure, and pulmonary edema (see ***PRECAUTIONS***).

Hematologic

Intracranial bleeding, bleeding and/or anemia due to hemodilution (see ***WARNINGS***) and/or Factor VIII deficiency, acquired von Willebrand's-like syndrome, and coagulopathy including rare cases of disseminated intravascular coagulopathy and hemolysis. With extensive clinical use of Hetastarch Injection, rare cases of disseminated intravascular coagulopathy and hemolysis have been observed.

Metabolic

Metabolic acidosis.

Other

Vomiting, peripheral edema of the lower extremities, submaxillary and parotid glandular enlargement, mild influenza-like symptoms, headaches, and muscle pains. Hydroxyethyl starch-associated pruritus has been reported in some patients with deposits of hydroxyethyl starch in peripheral nerves.

Caution: Federal (USA) law prohibits dispensing without prescription.

 Reference 58-0851-R2-Rev. September, 1999 Printed in USA

Manufactured and Distributed by: Abbott Laboratories, North Chicago, IL 60064, USA
Under license from BioTime, Inc., Berkeley, CA 94710, USA

*For infections due to susceptible strains of indicated organisms.

Careful inquiry should be made concerning previous hypersensitivity reaction, as serious and occasionally fatal anaphylactic reactions have been reported in patients receiving therapy with penicillins. ZOSYN is contraindicated in patients with a history of these reactions to any of the penicillins, cephalosporins, or ß-lactamase inhibitors.

While ZOSYN possesses the characteristic low toxicity of the penicillin group of antibiotics, periodic assessment of organ system functions, including renal, hepatic, and hematopoietic, during prolonged therapy is advisable.

During clinical trials, pseudomembranous colitis has been rarely reported (<1%).

The most commonly reported adverse events in clinical trials, irrespective of relationship to therapy, included diarrhea (11.3%), headache (7.7%), constipation (7.7%), nausea (6.9%), and insomnia (6.6%).

Please see adjacent brief summary of Prescribing Information.

ZOSYN® IV

(piperacillin sodium/tazobactam sodium)

EMPIRIC THERAPY FOR SERIOUS INFECTIONS

ZOSYN® (Piperacillin and Tazobactam for Injection) **Brief Summary**

See package insert for full prescribing information.

CONTRAINDICATIONS ZOSYN is contraindicated in patients with a history of allergic reactions to any of the penicillins, cephalosporins, or β-lactamase inhibitors.

WARNINGS SERIOUS AND OCCASIONALLY FATAL HYPERSENSITIVITY (ANAPHYLACTIC/ANAPHYLACTOID) REACTIONS (INCLUDING SHOCK) HAVE BEEN REPORTED IN PATIENTS RECEIVING THERAPY WITH PENICILLINS INCLUDING ZOSYN. THESE REACTIONS ARE MORE LIKELY TO OCCUR IN INDIVIDUALS WITH A HISTORY OF PENICILLIN HYPERSENSITIVITY OR A HISTORY OF SENSITIVITY TO MULTIPLE ALLERGENS. THERE HAVE BEEN REPORTS OF INDIVIDUALS WITH A HISTORY OF PENICILLIN HYPERSENSITIVITY WHO HAVE EXPERIENCED SEVERE REACTIONS WHEN TREATED WITH CEPHALOSPORINS. BEFORE INITIATING THERAPY WITH ZOSYN, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO PENICILLINS, CEPHALOSPORINS, OR OTHER ALLERGENS. IF AN ALLERGIC REACTION OCCURS, ZOSYN SHOULD BE DISCONTINUED AND APPROPRIATE THERAPY INSTITUTED. **SERIOUS ANAPHYLACTIC/ANAPHYLACTOID REACTIONS (INCLUDING SHOCK) REQUIRE IMMEDIATE EMERGENCY TREATMENT WITH EPINEPHRINE. OXYGEN, INTRAVENOUS STEROIDS, AND AIRWAY MANAGEMENT, INCLUDING INTUBATION, SHOULD ALSO BE ADMINISTERED AS INDICATED.**

Pseudomembranous colitis has been reported with nearly all antibacterial agents, including ZOSYN, and may range in severity from mild to life-threatening. Consider this diagnosis in patients who present with diarrhea after antibacterial agent administration. Treatment with antibacterial agents alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of "antibiotic-associated colitis."

After the diagnosis of pseudomembranous colitis has been established, initiate therapeutic measures. Mild cases usually respond to drug discontinuation alone. In moderate to severe cases, fluids and electrolytes, protein supplementation, and treatment with an antibacterial drug clinically effective against *Clostridium difficile* colitis may be necessary.

PRECAUTIONS General: Bleeding manifestations have occurred in some patients receiving β-lactam antibiotics, including piperacillin. These reactions have sometimes been associated with coagulation test abnormalities such as clotting time, platelet aggregation, and prothrombin time and are more likely to occur in renal failure patients. If bleeding manifestations occur, discontinue ZOSYN and institute appropriate therapy.

The possibility of the emergence of resistant organisms that might cause superinfections should be kept in mind. If this occurs, appropriate measures should be taken.

As with other penicillins, patients may experience neuromuscular excitability or convulsions if higher than recommended doses are given intravenously (particularly in the presence of renal failure).

ZOSYN is a monosodium salt of piperacillin and a monosodium salt of tazobactam, containing 2.35 mEq (54 mg) of Na+ per gram of piperacillin; consider this when treating patients requiring restricted salt intake. Perform periodic electrolyte determinations in patients with low potassium reserves; the possibility of hypokalemia should be kept in mind with patients who have potentially low potassium reserves and who are receiving cytotoxic therapy or diuretics.

As with other semisynthetic penicillins, piperacillin has been associated with an increased incidence of fever and rash in cystic fibrosis patients.

In patients with renal insufficiency or in hemodialysis patients, the intravenous dose should be adjusted to the degree of renal function impairment. (See Full Prescribing Information—**DOSAGE AND ADMINISTRATION**.)

Laboratory Tests: Perform periodic assessment of hematopoietic function, especially with prolonged therapy, ie, ≥21 days. (See **ADVERSE REACTIONS—Adverse Laboratory Events.**)

Drug Interactions: *Aminoglycosides*—The mixing of ZOSYN with an aminoglycoside in vitro can result in substantial inactivation of the aminoglycoside. (See Full Prescribing Information—**DOSAGE AND ADMINISTRATION—Compatible Intravenous Diluent Solutions**.)

When ZOSYN was co-administered with tobramycin, the area under the curve, renal clearance, and urinary recovery of tobramycin were decreased by 11%, 32%, and 38%, respectively. Pharmacokinetic alterations of tobramycin when administered with ZOSYN may be due to in vivo and in vitro inactivation of tobramycin in the presence of piperacillin/tazobactam. The inactivation of aminoglycosides in the presence of penicillin-class drugs has been recognized. It has been postulated that microbiologically inactive penicillin-aminoglycoside complexes of unknown toxicity form. In patients with severe renal dysfunction (ie, chronic hemodialysis patients), tobramycin pharmacokinetics are significantly altered when administered with piperacillin. The alteration of tobramycin pharmacokinetics and the potential toxicity of the penicillin-aminoglycoside complexes in patients with mild to moderate renal dysfunction who are administered an aminoglycoside with ZOSYN are unknown.

Probenecid—Probenecid administered with ZOSYN prolongs the half-life of piperacillin by 21% and of tazobactam by 71%.

Vancomycin—No pharmacokinetic interactions with ZOSYN have been noted.

Heparin—Coagulation parameters should be tested more frequently and monitored regularly during simultaneous administration of high doses of heparin, oral anticoagulants, or other drugs that may affect the blood coagulation system or the thrombocyte function.

Vecuronium—Piperacillin used with vecuronium has been implicated in the prolongation of the neuromuscular blockade of vecuronium. ZOSYN could produce the same phenomenon if given with vecuronium. Due to their similar mechanism of action, the neuromuscular blockade produced by any of the non-depolarizing muscle relaxants could be prolonged in the presence of piperacillin. (See package insert for vecuronium bromide.)

Methotrexate—Piperacillin may reduce the excretion of methotrexate; therefore, serum levels of methotrexate should be monitored in patients to avoid drug toxicity.

Drug/Laboratory Test Interactions: As with other penicillins, ZOSYN may result in a false-positive reaction for glucose in the urine using a copper-reduction method (CLINITEST®§). Glucose tests based on enzymatic glucose oxidase reactions (such as DIASTIX®§ or TES-TAPE®§) are recommended.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long term carcinogenicity studies in animals have not been conducted with piperacillin/tazobactam, piperacillin, or tazobactam. Piperacillin/tazobactam was negative in the following mutagenicity tests/assays up to the concentrations noted: microbial mutagenicity assay (14.84/1.86 μg/plate), unscheduled DNA synthesis (UDS) test (5689/711 μg/mL), mammalian point mutation (Chinese hamster ovary cell HPRT) assay (8000/1000 μg/mL), and a mammalian cell (BALB/c-3T3) transformation assay (8/1 μg/mL). In vivo, piperacillin/tazobactam did not induce chromosomal aberrations in rats dosed I.V. with 1500/187.5 mg/kg; this dose is similar to the maximum recommended human daily (MRHD) dose on a body-surface-area basis (BSA) (mg/m^2).

Piperacillin was negative in the following mutagenicity tests/assays up to the concentrations noted: microbial mutagenicity assays (50 μg/plate), UDS test (10,000 μg/mL), and a cell (BALB/c-3T3) transformation assay (3000 μg/mL). There was no DNA damage in bacteria (Rec assay) exposed to piperacillin at concentrations up to 200 μg/disk. In a mammalian point mutation (mouse lymphoma cells) assay, piperacillin was positive at concentrations ≥2500 μg/mL. In vivo, piperacillin did not induce chromosomal aberrations in mice at I.V. doses up to 2000 mg/kg/day or rats at I.V. doses up to 1500 mg/kg/day. These doses are half (mice) or similar to (rats) the MRHD dose based on BSA (mg/m^2). In another in vivo test, there was no dominant lethal effect when piperacillin was administered to rats at I.V. doses up to 2000 mg/kg/day, which is similar to the MRHD dose based on BSA (mg/m^2). When mice were administered piperacillin at I.V. doses up to 2000 mg/kg/day, which is half the MRHD dose based on BSA (mg/m^2), urine from these animals was not mutagenic when tested in a microbial mutagenicity assay. Bacteria injected into the peritoneal cavity of mice administered piperacillin at I.V. doses up to 2000 mg/kg/day did not show increased mutation frequencies.

Tazobactam was negative in the following mutagenicity assays up to the concentrations noted: microbial mutagenicity assays (333 μg/plate), UDS test (2000 μg/mL), mammalian point mutation (Chinese hamster ovary cell HPRT) (5000 μg/mL), a cell (BALB/c-3T3) transformation assay (900 μg/mL). In another mammalian point mutation (mouse lymphoma cells) assay, tazobactam was positive at concentrations ≥3000 μg/mL. In an in vitro cytogenetics (Chinese hamster lung cells) assay, tazobactam was negative at concentrations up to 3000 μg/mL. In vivo, tazobactam did not induce chromosomal aberrations in rats at I.V. doses up to 5000 mg/kg, which is 23 times the MRHD dose based on BSA (mg/m^2).

Pregnancy: *Teratogenic effects—Pregnancy Category B:* Piperacillin/tazobactam: Reproduction studies in rats have revealed no evidence of impaired fertility due to piperacillin/tazobactam administered up to a dose which is similar to the MRHD dose based on BSA (mg/m^2).

Teratology studies in mice and rats have revealed no evidence of harm to the fetus due to piperacillin/tazobactam administered up to a dose which is 1 to 2 times and 2 to 3 times the human dose of piperacillin and tazobactam, respectively, based on BSA (mg/m^2). Piperacillin and tazobactam cross the placenta in humans.

Piperacillin: Reproduction and teratology studies in mice and rats have revealed no evidence of impaired fertility or fetal harm due to piperacillin administered up to a dose which is half (mice) or similar to (rats) the MRHD dose based on BSA (mg/m^2).

Tazobactam: Reproduction studies in rats have revealed no evidence of impaired fertility due to tazobactam administered at doses up to 3 times the MRHD dose based on BSA (mg/m^2).

Teratology studies in mice and rats have revealed no evidence of fetal harm due to tazobactam administered at doses up to 6 and 14 times, respectively, the human dose based on BSA (mg/m^2). In rats, tazobactam crosses the placenta. Concentrations in the fetus are less than or equal to 10% of those found in maternal plasma. There are no adequate and well-controlled studies with the piperacillin/tazobactam combination or with piperacillin or tazobactam alone in pregnant women. Use this drug during pregnancy only if clearly needed.

Nursing Mothers: Piperacillin is excreted in low concentrations in human milk; tazobactam concentrations in human milk have not been studied. Exercise caution when ZOSYN is administered to a nursing woman.

Pediatric Use: Safety and efficacy in pediatric patients have not been established.

Geriatric Use: Patients over 65 years are **not** at an increased risk of developing adverse effects solely because of age. However, dosage should be adjusted in the presence of renal insufficiency. The geriatric population may respond with a blunted natriuresis to salt loading. This may be clinically important with regard to such diseases as congestive heart failure. Because elderly patients are more likely to have decreased renal function, care should be taken in dose selection, and it may be useful to monitor renal function. (See Full Prescribing Information—**DOSAGE AND ADMINISTRATION.**)

ADVERSE REACTIONS During the initial clinical investigations, 2621 patients worldwide were treated with ZOSYN in phase 3 trials. In the key North American clinical trials (n=830 patients), 90% of the adverse events reported were mild to moderate in severity and transient in nature. However, in 3.2% of the patients treated worldwide, ZOSYN was discontinued because of adverse events primarily involving the skin (1.3%), including rash and pruritus; the gastrointestinal system (0.9%), including diarrhea, nausea, and vomiting; and allergic reactions (0.5%).

Adverse local reactions that were reported, irrespective of relationship to ZOSYN therapy, were phlebitis (1.3%), injection site reaction (0.5%), pain (0.2%), inflammation (0.2%), thrombophlebitis (0.2%), and edema (0.1%).

Adverse Clinical Events: Based on patients from the North American trials (n=1063), the events with the highest incidence in patients, irrespective of relationship to ZOSYN therapy, were diarrhea (11.3%); headache (7.7%); constipation (7.7%); nausea (6.9%); insomnia (6.6%); rash (4.2%), including maculopapular, bullous, urticarial, and eczematoid; vomiting (3.3%); dyspepsia (3.3%); pruritus (3.1%); stool changes (2.4%); fever (2.4%); agitation (2.1%); pain (1.7%); moniliasis (1.6%); hypertension (1.6%); dizziness (1.4%); abdominal pain (1.3%); chest pain (1.3%); edema (1.2%); anxiety (1.2%); rhinitis (1.2%); and dyspnea (1.1%).

Additional adverse systemic clinical events reported in 1.0% or less of the patients in the initial North American trials are listed below within each body system. *Autonomic nervous system*—hypotension, ileus, syncope. *Body as a whole*—rigors, back pain, malaise. *Cardiovascular*—tachycardia, including supraventricular and ventricular; bradycardia; arrhythmia, including atrial fibrillation, ventricular fibrillation, cardiac arrest, cardiac failure, circulatory failure, myocardial infarction. *Central nervous system*—tremor, convulsions, vertigo. *Gastrointestinal*—melena, flatulence, hemorrhage, gastritis, hiccough, ulcerative stomatitis. Pseudomembranous colitis was reported in one patient during the clinical trials. The onset of pseudomembranous colitis symptoms may occur during or after antibacterial treatment. (See **Warnings**.) *Hearing and Vestibular System*—tinnitus. *Hypersensitivity*—anaphylaxis. *Metabolic and Nutritional*—symptomatic hypoglycemia, thirst. *Musculoskeletal*—myalgia, arthralgia. *Platelets, Bleeding, Clotting*—mesenteric embolism, purpura, epistaxis, pulmonary embolism (See **Precautions, General**). *Psychiatric*—confusion, hallucination, depression. *Reproductive, Female*—leukorrhea, vaginitis. *Respiratory*—pharyngitis, pulmonary edema, bronchospasm, coughing. *Skin and Appendages*—genital pruritus, diaphoresis. *Special senses*—taste perversion. *Urinary*—retention, dysuria, oliguria, hematuria, incontinence. *Vision*—photophobia. *Vascular (extracardiac)*—flushing.

In a completed study of nosocomial lower respiratory tract infections, 222 patients were treated with ZOSYN in a dosing regimen of 4.5 g every 6 hours in combination with an aminoglycoside and 215 patients were treated with imipenem/cilastatin (500 mg/500 mg q6h) in combination with an aminoglycoside. Twenty-five (25, 11.0%) patients in the piperacillin/tazobactam group and 14 (6.5%) in the imipenem/cilastatin group ($p > 0.05$) discontinued treatment due to an adverse event. Adverse events that occurred in more than 1% of patients and were considered by the investigator to be drug-related were: diarrhea (17.6%), fever (2.7%), vomiting (2.7%), urinary tract infection (2.7%), rash (2.3%), abdominal pain (1.8%), generalized edema (1.8%), moniliasis (1.8%), nausea (1.8%), oral moniliasis (1.8%), BUN increased (1.8%), creatinine increased (1.8%), peripheral edema (1.8%), abdomen enlarged (1.4%), headache (1.4%), constipation (1.4%), liver function tests abnormal (1.4%), thrombocythemia (1.4%), excoriations (1.4%), and sweating (1.4%).

Drug-related adverse events reported in 1% or less of patients in the nosocomial pneumonia study of ZOSYN with an aminoglycoside were: acidosis, acute kidney failure, agitation, alkaline phosphatase increased, anemia, asthenia, atrial fibrillation, chest pain, CNS depression, colitis, confusion, convulsion, cough increased, thrombocytopenia, dehydration, depression, diplopia, drug level decreased, dry mouth, dyspepsia, dysphagia, dyspnea, dysuria, eosinophilia, fungal dermatitis, gastritis, glossitis, grand mal convulsion, hematuria, hyperglycemia, hypernatremia, hypertension, hypertonia, hyperventilation, hypochromic anemia, hypoglycemia, hypokalemia, hyponatremia, hypophosphatemia, hypoxia, ileus, injection site edema, injection site pain, injection site reaction, kidney function abnormal, leukocytosis, leukopenia, local reaction to procedure, melena, pain, prothrombin decreased, pruritus, respiratory disorder, SGOT increased, SGPT increased, sinus bradycardia, somnolence, stomatitis, stupor, tremor, tachycardia, ventricular extrasystoles, and ventricular tachycardia.

In a previous nosocomial pneumonia study conducted with a dosing regimen of 3.375 g given every 4 hours with an aminoglycoside, the following adverse events, irrespective of drug relationship, were observed: diarrhea (20%); constipation (8.4%); agitation (7.1%); nausea (5.8%); headache (4.5%); insomnia (4.5%); oral thrush (3.9%); erythematous rash (3.9%); anxiety (3.2%); fever (3.2%); pain (3.2%); pruritus (3.2%); hiccough (2.6%); vomiting (2.6%); dyspepsia (1.9%); edema (1.9%); fluid overload (1.9%); stool changes (1.9%); anorexia (1.3%); cardiac arrest (1.3%); confusion (1.3%); diaphoresis (1.3%); duodenal ulcer (1.3%); flatulence (1.3%); hypertension (1.3%); hypotension (1.3%); inflammation at injection site (1.3%); pleural effusion (1.3%); pneumothorax (1.3%); rash, not otherwise specified (1.3%); supraventricular tachycardia (1.3%); thrombophlebitis (1.3%); and urinary incontinence (1.3%).

Adverse events irrespective of drug relationship observed in 1% or less of patients in the above study with ZOSYN and an aminoglycoside included: aggressive reaction (combative), angina, asthenia, atelectasis, balanoposthitis, cerebrovascular accident, chest pain, conjunctivitis, deafness, dyspnea, earache, ecchymosis, fecal incontinence, gastric ulcer, gout, hemoptysis, hypoxia, pancreatitis, perineal irritation/pain, urinary tract infection with trichomonas, vitamin B12 deficiency anemia, xerosis, and yeast in urine.

Additional adverse events reported from worldwide marketing experience with ZOSYN, where causal relationship to ZOSYN is uncertain: *Gastrointestinal:* hepatitis, cholestatic jaundice. *Hematologic:* hemolytic anemia, anemia, thrombocytosis, agranulocytosis, pancytopenia. *Immune:* hypersensitivity reactions, anaphylactic/anaphylactoid reactions (including shock). *Infections:* candidal superinfections. *Renal:* interstitial nephritis, renal failure. *Skin and Appendages:* erythema multiforme and Stevens-Johnson syndrome; toxic epidermal necrolysis.

Adverse Laboratory Events (Seen During Clinical Trials): Of the studies reported, including that of nosocomial lower respiratory tract infections in which a higher dose of ZOSYN was used in combination with an aminoglycoside, changes in laboratory parameters, without regard to drug relationship, include: *Hematologic:* decreases in hemoglobin and hematocrit, thrombocytopenia, increases in platelet count, eosinophilia, leukopenia, neutropenia. The leukopenia/neutropenia appears to be reversible and most frequently associated with prolonged administration, ie, ≥21 days of therapy. These patients were withdrawn from therapy; some had accompanying systemic symptoms (eg, fever, rigors, chills). *Coagulation:* positive direct Coombs' test, prolonged prothrombin time, prolonged partial thromboplastin time. *Hepatic:* transient elevations of AST (SGOT), ALT (SGPT), alkaline phosphatase, bilirubin. *Renal:* increases in serum creatinine, blood urea nitrogen. *Urinalysis:* proteinuria, hematuria, pyuria.

Additional laboratory events include abnormalities in electrolytes (ie, increases and decreases in sodium, potassium, and calcium), hyperglycemia, decreases in total protein or albumin, blood glucose decreased, gamma-glutamyltransferase increased, hypokalemia, and bleeding time prolonged.

The following adverse reaction has also been reported for PIPRACIL® (sterile piperacillin sodium): *Skeletal:* prolonged muscle relaxation. (See **PRECAUTIONS—Drug Interactions**.)

Piperacillin therapy has been associated with an increased incidence of fever and rash in cystic fibrosis patients.

OVERDOSAGE There have been post-marketing reports of overdose with piperacillin/tazobactam. The majority of those events experienced including nausea, vomiting, and diarrhea have also been reported with the usual recommended dosages. Patients may experience neuromuscular excitability or convulsions if higher than recommended doses are given intravenously (particularly in the presence of renal failure). Treatment should be supportive and symptomatic according to the patient's clinical presentation. Excessive serum concentrations of either piperacillin or tazobactam may be reduced by hemodialysis. (See Full Prescribing Information—**CLINICAL PHARMACOLOGY**.)

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This Brief Summary is based on ZOSYN direction circular CI 7876-1 (Revised April 2003).

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This publication/activity is designed to provide trauma care professionals interested in the treatment of critically ill trauma patients with a regular overview and critical analysis of the most current, clinically useful information available, covering strategies and advances in the diagnosis of traumatic injuries and the treatment of trauma patients. Controversies, advantages, and disadvantages of diagnosis and treatment plans are emphasized. There are no prerequisites for participation in this activity.

After reading each issue, participants should have a working familiarity with the most significant information and perspectives presented and apply what they have learned promptly in clinical practice. Specific learning objectives are listed at the opening of each article.

CME QUESTIONS

This issue of *TraumaCare* can be used to earn 10 CME credit hours.

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CME QUESTIONS

1. Clinically significant hypothermia induces any of the following except:
   a. Cardiac arrhythmias
   b. Decreased cardiac output
   c. Right shift of the oxygen-hemoglobin dissociation curve
   d. Inhibition of the coagulation cascade

2. In the original work by Stone et al, the mortality rate associated with using coagulopathy as an indication for the institution of damage control was:
   a. 25% b. 35%
   c. 50% d. 70%

3. The best candidates for damage control surgery are patients who are exsanguinating.
   a. True b. False

4. According to Asensio, the first step in the algorithm for the management of the exsanguinating patient is to:
   a. Determine the presence of an exsanguinating injury.
   b. Resuscitate the patient as per Advanced Trauma Life Support protocol.
   c. Rapidly transport to the operating room.
   d. Perform an emergency department thoracotomy to stop a possible subdiaphragmatic bleeding point.

5. Rewarming the patient is a high priority because:
   a. It decreases the time for closing the abdominal wall in case the patient undergoes damage control.
   b. Coagulopathy and acidosis can be corrected and maintained only after the body temperature returns to normal.
   c. It reduces heat generation.
   d. It reduces the need for transfusion.

6. Which physiologic indication predicts the need for damage control?
   a. Serum bicarbonate ≤22 mEq/L
   b. Hypothermia <35°C.
   c. Intraoperative volume replacement ≥12,000 mL.
   d. Acidosis ≤7.3

7. "Damage control" is a term adopted from U.S. military language.
   a. True b. False

8. The indications for a damage control procedure include all the following except:
   a. pH 7.1
   b. Temperature <32°C
   c. Hemoglobin <7.0 g/dL.
   d. Mean arterial pressure of 50 mm Hg
   e. Severe coagulopathy

9. Abdominal compartment syndrome is an acute condition in which increased intraabdominal pressure causes organ dysfunction as a result of intraabdominal hypertension greater than:
   a. 10 mm Hg
   b. 15 mm Hg
   c. 20 mm Hg
   d. 25 mm Hg
   e. 30 mm Hg

10. The triad of hypothermia, acidosis, and coagulopathy is referred to as the "bloody vicious cycle."
    a. True b. False

11. The five stages of damage control include all the following except:
    a. Obtaining hemostasis
    b. Obtaining control of contamination
    c. Warming the patient
    d. Resuscitating the patient in the ICU
    e. Decompressing raised intraabdominal pressure

12. The initial closure of the abdominal wound is best achieved by:
    a. Sandwich closure using abdominal drapes
    b Suture of the sheath
    c Leaving the abdominal wound open with a dressing
    d Skin graft
    e. Subcuticular closure of skin

13. Coagulopathy associated with damage control surgery should be treated first with:
    a. Cryoprecipitate
    b. Aggressive rewarming and correction of acidosis
    c. Recombinant factor VIIa
    d. Heparin
    e. Fresh blood

14. Intraabdominal hypertension is an uncommon condition associated with damage control.
    a. True b. False

15. "Shock" may be characterized by which of the following?
    a. Increased core body temperature
    b. Decreased blood pressure following hemorrhage
    c. Bradycardia <60 bpm
    d. Metabolic alkalosis

16. Which of the following is an appropriate physiologic target for fluid resuscitation during uncontrolled hemorrhagic shock?
    a. Blood pressure of 120/80
    b. Base deficit = –2
    c. Hematocrit >25%
    d. Temperature of 33°C

17. During massive transfusion therapy (>10 units of red blood cells in a 4-hour period), which of the following electrolyte abnormalities will require active intervention?
    a. Hypocalcemia
    b. Mild hyperkalemia
    c. Hypochloremia
    d. Hypomagnesemia

18. Adequate resuscitation from hemorrhagic shock requires definitive control of bleeding and which of the following?
    a. Clearance to normal of elevated serum lactate
    b. Anticoagulation to a prothrombin time >18 seconds
    c. Blood pressure and heart rate elevated to >150% of normal
    d. Urine output >5 mL/kg/hr

19. Which of the following therapies will improve end-organ tissue perfusion in the patient with uncontrolled bleeding and severe hemorrhagic shock (SBP <60)?
    a. Isotonic crystalloid, 1 liter
    b. 2 units of uncrossmatched type–O blood
    c. 1 mg epinephrine
    d. 500 mg sodium bicarbonate

20. Deliberate hypothermia, which has been suggested as a treatment for hemorrhagic shock and has demonstrated good results in animal trials, is recommended as a treatment option for trauma patients.
    a. True b. False

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References:
1) BMJ Volume 320, 18 March 2000
2) To Err Is Human: Building a Safer Health System/Linda T. Kohn, Janet M. Corrigan, and Molla S. Donaldson, Editors., © 2000 by the National Academy of Sciences.

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