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Damage Control Surgery—The Intensivist's Role

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"Damage control" surgery has evolved during the past 20 years from an accepted surgical technique in the traumatized, moribund patient to an expanded role in critically ill, nontraumatized patients. Physicians caring for these patients *in extremis* have begun to recognize a pattern of severe physiologic derangement that prompts an abbreviated laparotomy after hemorrhage and contamination control. Emphasis then shifts from the operating theater to the intensive care unit, where the patient's physiologic deficits are corrected. Once these derangements have been resolved, the patient is taken back to the operating room for definitive, reconstructive surgical care. The purpose of this article is to review the concept of "damage control" in reference to the patient whose pathophysiologic depletion prompts the need for it. Resuscitation in the intensive care unit will be summarized, pitfalls will be identified, and treatment plans will be delineated. Complications such as abdominal compartment syndrome and difficult abdominal wall closures will also be discussed.

Although some form of "damage control" surgery has been practiced for more than 20 years [1,2], traditional surgical principles dictated a series of standard steps that included access, exposure, hemostasis, resection, and reconstruction [3]. Surgeons continued to use these principles to complete a surgical procedure, irrespective of the patient's physiologic condition, and found that despite the performance of a technically correct procedure, the patient often died. The realization, in modern surgical critical care, that survivorship takes precedence over morbidity represents a paradigm shift from the traditional, elective surgical mentality [3,4]. Consequently, the emphasis has turned toward the

intensive care unit (ICU) as the optimal location for resuscitation after initial evaluation and operation. The goal of this review is to describe the pathophysiology of the critically ill patient and to define the selection criteria for using damage control. In addition, the sequence of the damage control procedure is reviewed, treatment strategies are provided along with the end points of resuscitation used to guide the resuscitation effort, and, finally, complications and outcomes are discussed in the context of ICU management.

History

Packing of bleeding surfaces and early termination of a laparotomy date back to the early 20th century, when Pringle [5,6], Halsted [6], and Schroeder [7] individually reported on successful cases of hemorrhage arrest after packing an injured liver. Despite initial bleeding control with packing, recurrent hemorrhage was often evident. However, this technique remained the mainstay of care until the end of World War II, when with improved surgical technique, packing fell into disfavor. Madding [8], in 1955, downplayed packing as a primary surgical technique as reports of necrosis, sepsis, and hemorrhage began to appear in the literature [9]. By the early 1970s, the reintroduction of perihepatic packing was gaining acceptance at several centers, and limited success was noted in a highly selective group of patients [10,11]. Feliciano et al [2], in 1981, reported on 10 patients with exsanguinating hepatic hemorrhage. A 90% survival rate was noted, and surgeons began to reconsider perihepatic packing as a viable technique [2]. In 1983, Stone et al [1] suggested that the development of coagulopathy was contributing significantly to poor outcomes in these exsanguinating patients. They proposed a method to rapidly terminate the procedure, reverse the coagulopathy, and return the patient to the operating room (OR) at a later time for definitive care.

By the 1990s, the presentation of trauma patients was changing. There were more gunshot wounds

noted during this time. Multiple penetrations, previously only rarely seen, were now routinely being treated at the major urban trauma centers [12]. Because of this alarming trend, and necessity being the mother of invention, the trauma group at the University of Pennsylvania began codifying the fundamental concepts of "damage control" in victims of penetrating abdominal trauma. These concepts included the rapid termination of the procedure after control of hemorrhage, continuation of aggressive ICU resuscitation, and return to the operating room for definitive care [13]. In 1997, Rotondo and Zonies [4] reported on a 20-year cumulative review of the literature of the mortality and morbidity rates using damage control principles for hepatic injury. In their review of 495 patients, they found a mortality rate of 44% and a morbidity rate of 39%. When the literature review was extended to include injuries other than liver trauma, the mortality rate increased to 60% and the morbidity rate rose to 43%. When both reviews were combined, a cumulative mortality rate of 52% and a morbidity rate of 40% resulted. Despite the high complication and death rates, damage control principles continued to gain acceptance because of the recognition that this was an extremely ill subset of patients whose constellation of injuries were not thought to be survivable.

Pathophysiology of Exsanguination

Proper understanding of the pathophysiology of the bleeding patient *in extremis* is the foundation on which the damage control principles were founded. Kashuk et al [14] coined the term "bloody vicious cycle," which refers to the downward spiral of the patient's physiologic condition. A lethal triad of events including hypothermia, coagulopathy, and metabolic acidosis characterizes the cycle [4,14].

Hypothermia

Hypothermia causes cardiac dysrhythmias; reduces cardiac output; shifts the oxygenation hemoglobin saturation curve to the left, thus preventing the offloading of oxygen to the cells; and affects the clotting cascade [4]. Burch et al [15] attempted to quantify the amount of heat loss seen during a laparotomy for trauma. They found that even with warmed intravenous fluids, warmed anesthesia gases, and heated air convection blankets, the estimated temperature loss during the procedure was conservatively estimated at 4.6°C per hour. They proposed that the real benefit of the abbreviated laparotomy was to limit the amount of heat loss, thus allowing the temperature-sensitive clotting cascade to restore itself [15]. Jurkovich et al [16] reported that the mortality increased from 40% to 100% when a trauma patient's core temperature went from 34°C to less than 32°C. Hypothermia is thought to affect the clotting cascade by restricting the temperature-sensitive, enzyme-activated serine esterases that lead to platelet dysfunction, endothelial abnormalities, and alterations in the fibrinolytic system [17]. Conversely, Krishna et al [18] suggested that the failure to generate an adequate core temperature is partly the result, rather than the cause, of the general cellular metabolic failure.

Coagulopathy

Every aspect of coagulation is adversely affected in the hypothermic patient. Rohrer and Natale [19] found that standard coagulation assays calibrated at 37°C did not accurately reflect the actual clotting impairment of a hypothermic patient. With each degree Celsius of cooling, both the prothrombin time and the activated partial thromboplastin time were significantly elevated [19]. Platelet dysfunction, as evidenced by increased bleeding times, was noted during hypothermia in an animal model attributable to decreased plasma thromboxane levels [4]. This is thought to be caused by changes in function of the GIIIb–IIa receptor sites. Moreover, the known dilutional effect on platelet factor V and VIII coupled with the effect of hypothermia renders the clotting cascade dysfunctional [4].

Metabolic Acidosis

Acidosis results from hypoperfusion and subsequent oxygen debt to tissues resulting in a shift from aerobic to anaerobic metabolism at the cellular level and subsequent lactic acidosis. This "cellular hypoxia" is in contradistinction to "cellular dysoxia," which occurs in a relatively rich oxygen environment in mitochondria where microcirculatory shunting of oxygen at the cellular level results in insufficient oxygen delivery to sustain aerobic metabolism [20].

The use of lactate clearance as a marker of successful resuscitation is now widely accepted. Abramson et al [21] showed that if trauma patients

were able to clear their lactate within 24 hours, a 100% survival was noted. Likewise, if the lactate was not cleared within 24 hours, only 14% survived. Multiple studies have documented the prognostic value of blood lactate as an index of oxygen delivery, morbidity, and mortality in hemorrhagic shock. More than 13 studies in the past 5 years have demonstrated the value of lactate as a prognostic index of outcome in greater than 600 trauma patients [20,22].

Damage Control Sequence

The damage control sequence evolved in an effort to combat the lethal triad of hypothermia, coagulopathy, and metabolic acidosis and to abort the "bloody vicious cycle." By definition, the damage-controlled patient is at or near the point of physiologic exhaustion. To properly treat this small subset of patients, the critical care team and its supporting hospital must be prepared to urgently intervene on behalf of a dying patient in a carefully choreographed, preconceived manner. Communication is paramount between the resuscitation team and the initial receiving areas within the hospital, including the emergency department, the OR, and the ICU. In addition, it is of utmost importance that careful coordination occur with 4 additional hospital services: blood bank, laboratory, pharmacy, and invasive radiology [23]. The surgeon, as the team leader, serves as the cornerstone of the system. Supervision of the resuscitation usually requires a continued bedside surgical presence during the first critical hours after admission to the ICU and a minimum ratio of 2 nurses to 1 patient. The surgeon is also responsible for coordinating the subspecialty consultants, ensuring proper communication regarding the patient's progress, and delineating the evolving plan of further care.

Damage control consists of 3 distinct parts including initial abbreviated laparotomy, ICU resuscitation, and subsequent reoperation for definitive repair. Although the first phase of the damage control sequence takes place in the OR and consists of immediate laparotomy for control of hemorrhage and contamination, identifying viable candidates for the damage control procedure is the key to the successful application of the technique. Damage control selection can be thought of in terms of conditions, complexes, and critical factors (Table 1). Several conditions—including high-energy, blunt torso trauma; multiple torso penetrations; hemodynamic instability; and coagulopathy and/or hypo-

Table 1. Damage Control: Key Factors in Patient Selection

Conditions
  High-energy blunt torso trauma
  Multiple torso penetrations
  Hemodynamic instability
  Presenting coagulopathy and/or hypothermia
Complexes
  Major abdominal vascular injury with multiple
    visceral injuries
  Multicavitary exsanguination with concomitant
    visceral injuries
  Multiregional injury with competing priorities
Critical factors
  Severe metabolic acidosis (pH <7.3)
  Hypothermia (temperature <35°C)
  Resuscitation and operative time >90 minutes
  Coagulopathy as evidenced by development of
    nonmechanical bleeding
  Massive transfusion (>10 units packed red blood
    cells)

Adapted from Rotondo MF, Zonies DH. The damage control sequence and underlying logic. *Surg Clin North Am.* 1997;77:761.

thermia on admission—predispose the patient to physiologic exhaustion and the need for damage control. Patients who present with a complex major abdominal vascular injury with multiple visceral injuries, multicavitary exsanguination with visceral injuries, or multiregional injury also warrant close scrutiny for a damage control procedure. Emphasis should be placed on controlling hemorrhage within and across multiple cavities so that time spent on nonbleeding areas is minimized. Finally, there are several critical factors that call for damage control: (a) pH less than 7.30; (b) temperature less than 35°C; (c) combined resuscitation and procedural time exceeding 90 minutes; (d) nonmechanical bleeding; and (e) transfusion requirements surpassing 10 units of packed red blood cells (PRBCs), which should obligate the surgeon to perform an abbreviated laparotomy [4,24,25]. Others further recommended that if the base deficit is worse than –18 mmol/L in a patient less than 55 years old, damage control should be used. Similarly, a base deficit worse than –8 mmol/L in a patient greater than 55 years old or in any patient with a head injury should receive consideration for a damage control procedure. Likewise, a patient requiring celiotomy with a lactate greater than 5 mmol/L may also be a damage control candidate [26-28]. Morris et al [24] deemed patients unsalvageable if they were 70 years or older, had suffered a prehospital blunt cardiac arrest, or had a fatal head injury.

Early pattern recognition is encouraged such that the decision to apply damage control is made early

in the patient's course. This ability takes practiced discipline in this select, small subset of critically ill patients.

Damage Control Part I—The OR

Before the patient's arrival in the OR, the OR staff should have a room identified and ready for the patient presenting *in extremis*. All the resuscitation equipment should be available as well as the standard exploratory laparotomy trays. The room should be heated to 85°F on notification that an unstable patient is en route to the OR. In addition, a warming device (Arctic Sun, Medivance, Louisville, CO) should be preheated and made ready to be applied to the patient's posterior thorax.

The conduct of the operation proceeds with a generous midline vertical incision. Once the abdomen opening is completed, immediate hemorrhage control with packing, ligation, clamp application, or balloon catheter tamponade should commence. When bleeding is controlled, attention is then turned to contamination control. After a quick inspection of the entire bowel, contamination containment is achieved through the use of simple suture closure or clamping of the visceral perforations. No reconstruction efforts should be made during damage control part I.

On termination of the initial procedure, some form of abdominal wall closure should be undertaken. Available techniques for rapid abdominal wall closure include using multiple towel clips, using a running No. 2 nylon skin and subcutaneous tissue suture, suturing a sterilized plastic intravenous bag to the skin, or placing a vacuum pack dressing [3,4,9,23-25]. If closure of the skin is possible, our preference is to close the skin with a running large No. 2 nylon suture. The benefits of this closure include preservation of the fascia and the ease and relative speed at which the skin closure can be accomplished; this closure also allows for angiographic/radiographic procedures to be done without radio-opaque clamps obstructing the fluoroscopy. The risks of using this technique include the abdominal compartment not being able to expand with the potential for intra-abdominal hypertension and abdominal compartment syndrome development. If the skin cannot be reapproximated, we typically construct a vacuum-type dressing. Fluid management is a critical role for the intensivist during the patient's resuscitation with a vacuum dressing in place. This dressing can often evacuate several liters of fluid per day, and the fail-

ure to recognize this ongoing fluid loss may result in a underresuscitated patient.

Over the course of damage control part I, the intensivist must continually ask the following questions: Has all mechanical bleeding been controlled? Are the packs required? Is this heroic care futile? [29]. The decision making over the course of the first operation is critical to the overall success of the approach.

Interventional Radiology

If solid organ bleeding concerns persist, a detour to the angiography suite for embolization should be undertaken before transfer to the ICU. The move from the OR can be challenging and should be thought out in advance. Several members of the team will be required to transport the patient to the ICU/angiography suite because these patients will often have a ventilator, two Rapid Fluid Warmer devices (Level 1 Technologies, Marshfield, MA), monitors, intravenous and blood bags, and possibly vasoactive drips accompanying them [3]. The surgeon/intensivist should be in attendance during the patient transport to the angiography suite and direct continued efforts at resuscitation and rewarming. The angiography suite will function as the ICU during the embolization procedure. The angiographer should attempt to embolize any obvious bleeding sites in the liver or pelvis. The embolization should be as distal as possible in the artery because a more proximal embolization may result in an increased risk for tissue ischemia and lactic acidosis. Depending on the area embolized, significant muscle breakdown may occur resulting in rhabdomyolysis with subsequent renal failure if adequate resuscitation is not maintained in the ICU. The angiographer should leave the arterial sheath in place in anticipation of further embolization procedures.

Damage Control Part II—The ICU

Once the abdomen has been temporarily closed, the second phase of the damage control sequence begins—ICU resuscitation. The intensivist's role begins with the commencement of damage control part II.

The team's focus should turn to secondary resuscitation in an effort to rewarm the patient and correct the patient's acidosis and coagulopathy [30].

This resuscitation requires frequent assessment and exhausts nursing and physician resources.

Ventilation Techniques

The patient should be initially placed on a ventilator with inspired gas warmed to 40°C. The damage-controlled patient is at risk for the development of acute lung injury (ALI) and adult respiratory distress syndrome (ARDS). Unique factors that may predispose the damage-controlled patient to ALI and ARDS, in addition to the direct parenchymal lung injury and shock often associated with trauma patients, include the massive resuscitation volumes that this patient receives during the first day of resuscitation. The resuscitation volume can reach more than 2 L/h during this initial resuscitation. This large volume creates chest wall compliance issues and contributes to pulmonary edema. Abdominal packing and the development of intra-abdominal hypertension can elevate the diaphragm and contribute to increases in thoracic pressure and decreased compliance. The goal of our ventilatory strategy is to maintain oxygenation and ventilation but also to prevent volutrauma. This strategy has evolved during the last several years from the pressure-volume work group [31]. To combat ALI in our institution, the patient is placed on a pressure-regulated, volume control mode with an initial tidal volume of 6 to 8 cc/kg in an attempt to keep the peak inspiratory pressure (PIP) less than 40 torr as we attempt to maintain a low plateau pressure. The fraction of inspired oxygen (FiO_2) is initially set at one (100%) and weaned to keep the oxygenation saturation of the blood greater than or equal to 93%. This often requires the addition of positive end-expiratory pressure (PEEP). The PEEP is initially set at 5 cm H_2O and increased in 2 to 3 cm H_2O increments over the next several hours to decrease the FiO_2 to less than 60%. High levels of PEEP, often reaching into the high 20s, are frequently required to maintain oxygenation. When the PEEP levels are sufficient to impede venous return and effect preload, typically at the 15 cm H_2O level, pulmonary artery catheterization is undertaken to assess the left ventricular end diastolic volume and to optimize the patient's preload. If oxygenation remains a problem, the inspiration to expiration (I:E) ratio is reduced to aid in oxygenation, thus allowing a permissive hypercapnea to occur as long as the patient maintains a pH >7.2. If the I:E ratio is reversed, intravenous sedation, analgesics, and paralytics are used to control the patient's ventilatory efforts.

Typically, the patient is placed on morphine and lorazepam infusions postoperatively. If paralysis is needed, an intravenous vecuronium infusion is used to maintain 2 to 4 twitches in a train-of-4 when tested using a nerve stimulator. If despite these efforts, the PaO_2 to FiO_2 ratio (P/F ratio) remains less than 250, rotational therapy is used. The patient is placed on a rotokinetic bed and rotated in both directions in a 40° arc angle from supine. In addition to rotokinetic therapy, prone positioning has been effectively used to improve the ventilation/perfusion mismatch that is seen in the damage-controlled patient. The prone position is contraindicated in patients with an unstable pelvic fracture or spinal column injury but has been successfully used in the patient with an open abdomen. Should the patient fail to respond to these maneuvers, a salvage technique may be used in an effort to maintain oxygenation. At our institution we consider using oscillating ventilation.

The oscillating ventilator (HFOV 3100B, SensorMedics Corporation, Yorba Linda, CA) initial mean airway pressure (mPaw) is set at 5 cm H_2O pressure above the conventional ventilator's mPaw. An initial recruitment maneuver may be considered if severe hypoxia is present. This recruitment maneuver is done by applying 40 cm H_2O for 40 to 60 seconds. If oxygenation does not improve, the clinician should increase the mPaw in 3- to 5-cm increments every 30 minutes until maximum settings are reached. Next the power is set at 4.0 and should be adjusted to obtain a visible vibration from the patient's shoulders to the midthigh area ("chest wiggle"). The frequency is next established by setting the Hertz. The initial Hertz should be set at 5 to 6. If PcO_2 is noted to climb, the Hertz can be decreased by 1 every 30 minutes until a minimal level of 3 is obtained. Finally set the inspiratory time (IT) at 33%. This value may be increased to 50% if the patient cannot be ventilated. The intensivist should start the initial FiO_2 at 1.0 (100%) and as the oxygenation improves slowly decrease the FiO_2 to 0.4 to 0.6 as tolerated by the patient. If the patient still has a severe hypercapnea, the endotracheal tube cuff can be deflated to produce a "cuff leak." This should be done until an mPaw decrease of 5 cm H_2O is noted.

Resuscitation

The patient should have several large-bore intravenous lines, and central access is recommended before the development of the edema that is antici-

pated as a result of the high-volume fluid resuscitation. This kind of access is usually established using multiple 8.5F introducer catheters placed in either the internal jugular or subclavian positions. Often the patient arrives to the ICU with a femoral introducer. The goal is to remove the femoral introducer within 24 hours of its placement after additional access has been established in the ICU. The resuscitation is guided by reestablishment of end-organ perfusion as evidenced by adequate urinary output, restoration of vital signs, and clearance of lactic acidosis. Typically, lactic acid measurements are drawn on the patient's arrival to the ICU along with a complete blood count, a coagulation profile, and a chemistry profile, including magnesium, ionized calcium, and phosphorous. Lactic acid measurements are continued every 4 hours until 2 consecutive measurements are less than or equal to 2 [21]. If the lactic acid fails to clear, or worse, it rises, a high-volume fluid resuscitation is instituted with warmed lactated Ringer's solution. The patient's maintenance rate is increased to 250 cc/h above the vacuum pack dressing fluid removal rate. The patient typically receives intravenous fluid boluses of 1-liter increments in response to rising lactic acid measurements, a decreasing urinary output, a decreasing mixed venous oxygenation saturation (SvO_2), or pulmonary artery indices consistent with hypovolemia. If the lactic acid measurements continue to rise, further adjustments to the maintenance intravenous fluid rate are initiated. This resuscitation may be facilitated by the use of an oximetric or volumetric, flow-directed pulmonary artery catheter to maximize hemodynamics and to restore volume to a non-flow-dependent state of oxygen consumption [4]. Clearance of lactic acid on serial measurements indicates the progress of the resuscitation, and normalization of the patient's vital signs indicates that the patient is being successfully resuscitated. Moore et al [30] suggested achieving an oxygen delivery index (DO_2I) greater than 600 mL/min/m^2 and an oxygen consumption index (VO_2I) greater than 150 mL/min/m^2 within 12 hours of the patient's arrival to the ICU [29,30,32]. These numbers typically correspond to an SvO_2 measurement of 68% to 72%. If the oxygen delivery index goal is not being met with standard intravenous fluids, PRBC transfusion is begun with the plan to maintain the hemoglobin above the 10-g/dL level. With the advent of the newer volumetric pulmonary artery catheters, the right ventricular end-diastolic volume index (RVEDVI) has been shown to have a better correlation with left ventricular end-diastolic volume than pulmonary artery capillary pressures at the higher PEEP levels that these

patients require. The cardiac output computer attached to the volumetric pulmonary artery catheter calculates the RVEF. The area under the thermal decay curve, based on R-R interval measurements, is the patient's cardiac output. The residual temperature change between beats is used to derive the RVEF. RVEDVI equals the stroke volume index (SVI) divided by the right ventricle ejection fraction (RVEF). If hypoperfusion is evident, the RVEDVI should be pushed to exceed 120 mL/m^2, which might require 20 L/d or more of intravenous crystalloid solution to maintain [26]. To obtain these physiologic parameters, inotropic support should be considered. The addition of vasopressors should be started cautiously and only after proper volume loading has occurred with crystalloid solutions and blood. A careful tertiary survey should occur during damage control part II to evaluate the patient for missed injuries, particularly fractures, or other signs of a missed injury. However, the failure of the lactic acid to clear after ongoing fluid resuscitation should prompt the intensivist to consider a missed injury, ongoing hemorrhage, or the development of abdominal compartment syndrome (ACS).

Recent concerns about the use of pulmonary artery catheterization and the potential for complications with invasive monitoring techniques have led to an increased interest in the development of newer, less invasive techniques for monitoring cardiac index in a critically ill patient. The PiCCO catheter (Pulsion Medical, Munich, Germany) allows for monitoring of continuous cardiac output via arterial pulse contour analysis combined with transpulmonary thermodilution measurements. The manufacturer suggests placing the arterial catheter in a proximal artery, either the axillary or femoral artery, which may limit its usefulness as a noninvasive monitoring device. Studies on cardiac surgery patients have validated its accuracy in measuring cardiac output, although the cardiac output must be determined by another method to initially calibrate the PiCCO catheter. However, when combined with the transpulmonary thermodilution technique, the PiCCO catheter does allow the intensivist to estimate the patient's preload. The lithium dilution cardiac output monitor (LiDCO, LiDCO Ltd, London, UK) is another minimally invasive technology on the market for determining cardiac output. This technique requires the injection of a small amount of lithium chloride into the venous circulation. A withdrawal of a small blood sample is required to calculate the amount dilution of lithium chloride. Central venous catheterization is not required. The cardiac output measurements obtained via the LiDCO catheter appear to correlate well with other

measurements of cardiac output, but this device does not allow for assessment of the patient's pre-load status [33]. The majority of the studies evaluating these emerging technologies have been done on cardiac surgery patients. We know of no studies evaluating this technology in the damage-controlled patient. Additionally, because neither of these catheters assesses the SvO_2, the application of this technology to monitor just the continuous cardiac output in a critically ill patient has limited application in the damage-controlled patient [34].

Unplanned Reoperation

Reoperation is anticipated to occur during damage control as a planned procedure in a hemodynamically stable, fully rewarmed, and physiologically "recaptured" patient. This reoperation cannot occur until after the patient has been aggressively resuscitated to acceptable parameters in the ICU. Conversely, emergent reoperation as an unplanned event usually occurs in three types of clinical scenarios: (a) ongoing bleeding, (b) missed enteric injury resulting in systemic inflammatory response syndrome and shock, and (c) development of ACS. The aim at this juncture of the resuscitation is to control hemorrhage or contamination and, if necessary, decompress the peritoneal cavity [4,22,24,35].

Urgently returning to the OR is inherently difficult because of the physiologic instability of the patient and stresses on the transport system. Some bleeding is anticipated postoperatively. If the bleeding is noted to require in excess of 2 units of PBRCs per hour for 3 hours, or when it exceeds the surgeon's anticipated expectations, particularly in a warmed, noncoagulopathic patient, a trip back to the OR should be contemplated [29,35]. These types of transfusion requirements represent mechanical bleeding as a result of failure of damage control part I, in which the liver packing may have failed or a clotted vessel may have resumed bleeding. If solid organ bleeding is suspected, consideration should be given to returning the patient back to the angiography suite to evaluate the organ in question for angiographic embolization of a bleeding vessel.

A continued picture of distributive shock may represent a missed injury or a failed repair that has begun leaking enteric contents or a repair or an injury mechanism that has resulted in an ischemic organ. Reopening this patient at the bedside should be done with great trepidation, because most ICUs are not equipped to handle major bleeding at the bedside. If moving the patient is not an option, planning to have the necessary instruments, lighting, and suction is paramount to the operation's success. The presence of an anesthesiologist at the bedside may prove to be an invaluable addition to the ICU team. Another option would be to consider a reangiogram of the patient via the existing femoral sheath.

Resuscitation should continue during the transportation of the patient. A battery-equipped ventilator is a must to maintain the high PEEP requirements that these patients usually require. Taking the patient off the ventilator and attempting to manually bag the patient is a recipe for disaster. Because of the rapid loss of PEEP, desaturation can quickly occur, leading to a potentially unrecoverable, terminal event.

ACS has pathophysiologic changes noted in multiple organ systems. The cardiovascular system has hypovolemia, decreased cardiac output, and decreased venous return. The systemic vascular resistance is elevated, as is the pulmonary artery occlusion pressure and the central venous pressure. The nervous system has increased intracranial pressure and decreased cerebral perfusion pressure. The gastrointestinal system has decreases in celiac, superior, and inferior mesenteric artery blood flow. These result in bowel ischemia and dilation. The liver has decreased portal blood flow with a resultant decreased lactate clearance. The pulmonary system notes an increased intrathoracic pressure, with resulting increases in PIP, peak airway pressure, and shunt. The lung compliance decreases, as does the alveolar oxygen content. The kidneys have decreased renal blood flow, which results in a decreased urinary output and decreased glomerular filtration rate. Clinically, ACS is manifested by abdominal distention, an increased intra-abdominal pressure (bladder pressure) exceeding 25 mm Hg, an increased PIP greater than 45 cm H_2O, and oliguria. Additional signs include hypercarbia, hypoxemia refractory to increasing oxygen concentrations or increasing PEEP, refractory metabolic acidosis, and increased intracranial pressure [36]. Not all of these signs need to be present to diagnose ACS. Surveillance for intra-abdominal hypertension requires a high index of suspicion, because ACS can develop insidiously. Monitoring is simply accomplished by measuring bladder pressure every 4 hours using the technique described by Cheatham and Safcsak [37]. Bedside decompression should occur when bladder pressures are greater than 25 mm Hg and there are concomitant signs of ACS. Morris et al [24] described the phenomenon of

reperfusion syndrome that caused immediate cardiac arrest, on decompression, in several patients. The intensivist should be wary of this complication and plan to load the patient with several liters of crystalloid immediately before abdominal decompression because the sudden release of abdominal pressure may result in the loss of compression of the inferior vena cava resulting in unrecoverable hypotension. If a delay in the recognition of ACS has occurred, the administration of a liter of lactated Ringer's solution, augmented with 25 grams of mannitol and 1 ampule of sodium bicarbonate, before decompression is advocated. This regimen appears to prevent reperfusion acidosis from occurring as a result of the washout of the high concentrations of acids, potassium, and other anaerobic metabolites into the systemic circulation from the hypoperfused abdominal viscera and lower extremities [23,34].

If possible, the decompressing team should also include a respiratory therapist. The therapist's job is to monitor PIP during decompression and to provide immediate reduction in the amount of ventilatory support to the patient, thus preventing alveolar overinflation and barotrauma [38]. If possible, a gradual abdominal decompression over 1 or 2 minutes is preferred to a sudden "explosive" evisceration of the abdominal contents. Once the abdomen is open and the peritoneal serous fluid has been evacuated, a vacuum dressing should be applied. Serial bladder pressures should continue to be monitored, because ACS can rarely reoccur with the vacuum dressing in place.

Rewarming

Terminating the procedure and providing a temporary closure of the abdominal wall are the first steps in the active rewarming process. Successful rewarming aids the resuscitative process by allowing cofactors in the clotting process to work, thereby obtaining hemorrhage control and clearance of lactic acidosis. The rewarming process should be continued in the ICU. The ICU suite should be preheated to 85°F in anticipation of the patient's arrival. The patients should be removed from any wet linen and dried as soon as possible after arrival to the ICU. An aluminum foil cap should be placed on the patient's head if it has not been done already in the OR. The ventilator circuit should be warmed. An air-convection blanket should cover the patient and be set at 40°C. All transfusion lines should have a dedicated fluid-warming device attached to them [4,9,29].

The patient should be warmed to 37°C within 4 hours of arrival to the ICU. If the patient's core temperature does not respond and remains lower than 35°C, pleural lavage with warm saline via multiple chest tubes should be considered [13]. Should the temperature remain less than 33°C, continuous arteriovenous warming should be considered using a specially adapted Rapid Fluid Warmer device (Level 1 Technologies, Marshfield, MA) as described by Gentilello [39]. Arctic Sun® warming device pads can be maintained on the patient when transferred from the OR to the ICU. An additional warming device is maintained separately in the ICU for the purpose of rewarming the patient. These pads can be left in place for up 24 hours. The pads are connected to the warming device, and a temperature probe is inserted into the patient and connected to the device. The goal temperature should be set at 37°C.

Coagulopathy Correction

Damage control part II may require 24 to 48 hours to establish "normal" physiology. The practitioner can anticipate massive transfusion requirements. Burch et al [36] reported that during the first 24 hours, they averaged 9.8 units of PRBCs, 8.8 units of fresh frozen plasma (FFP), and 10.7 units of platelets per patient. Morris' group [24] reported similar numbers: they transfused an average of 9.5 units of PRBCs, 8 units of FFP, and 6.2 units of platelets during the first 24 hours of resuscitation. Mortality rates for these 2 groups were 70% and 66%, respectively. The 10-unit rule (10 units each of PRBCs, FFP, and platelets) should serve as a transfusion guide during the first 24 hours. However, blood products should be administered until the prothrombin time is less than 15 seconds and platelet counts are greater than 100 000/mm^3. Cryoprecipitate is administered when the fibrinogen level is less than 100 mg/dL and should be given every 4 hours until the fibrinogen level is greater than 100 mg/dL [29]. Recombinant factor VIIa (rFVIIa), a potent prohemostatic agent, has been used with increasing frequency in damage control part II for the treatment of coagulopathy associated with severe hemorrhage. Studies to date suggest that rFVIIa is relatively safe, with a low incidence of thrombotic complications. Although there is a paucity of data from prospective controlled trials, case series suggest that the use of rFVIIa should be considered for life-threatening, nonsurgical hemorrhage during damage control part II [40].

Correction of Acidosis

The patient's acidosis usually corrects itself once the patient is adequately rewarmed and resuscitated. The oxygen debt is repaid, and the patient reverts from anaerobic to aerobic metabolism. We seldom administer sodium bicarbonate during resuscitation unless the pH is less than 7.2, particularly if cardiotonic agents are being used, because those agents perform better in a less acidic environment.

Damage Control Part III—Definitive Operation

Planning for Definitive Operation

Once hemodynamic stability has been achieved and the patient is warm and not coagulopathic, the decision to return to the OR for the definitive operation can be made. This return usually occurs within the first 12 to 48 hours after a damage control laparotomy. The intensivist plays an important role in preparing the patient to return to the OR. The patient typically has undergone a massive fluid resuscitation, sometimes approaching 20 liters since the first operation. The intensivist should anticipate anasarca and labile fluid shifts resulting from the evacuation of fluids from the patient's open abdomen. Meeting the patient's ongoing fluid requirements is the paramount goal of the intensivist. The patient should have type and crossed blood available for use in the OR, and FFP should be thawed in anticipation of bleeding in the OR. Blood chemistry disturbances should be anticipated and corrected. Hypocalcemia, hypomagnesemia, and hypophosphotemia are found commonly in patients undergoing resuscitation after damage control surgery and may require multiple electrolyte infusions before the patient's return to the OR. A broad-spectrum antibiotic should be administered when the OR calls for the patient.

The patient will often require advanced ventilatory techniques to manage elevated PIPs. The patient may be on elevated PEEP levels that would make disconnecting the ventilator circuit hazardous for the patient. When the patient is on high ventilatory support, the intensivist should consider the use of a transport ventilator with battery support that would accompany the patient to the OR. Maximal effort should be made to avoid disconnecting the patient from the ventilator and maintaining the patient on the ICU ventilator settings.

Goals of this planned procedure consist of pack removal, full abdominal exploration with injury reassessment, and copious irrigation [4,23,24,26]. Should physiologic derangements recur during this typically 2- to 4-hour procedure, the surgeon should revert to the damage control part I mentality and repack the patient, again abbreviating the procedure and applying a temporary abdominal closure dressing. The most compelling reason to return to the OR is an ischemic limb or to reestablish intestinal continuity [36].

In anticipation of closing the patient, consideration should be given to providing the patient with an access for enteral nutrition. Options include a long nasojejunal tube, gastrostomy, or jejunostomy. If skin or fascial closure is possible at the conclusion of this procedure, a single radiograph of the abdomen is recommended to eliminate the possibility of any retained laparotomy sponges.

Physiologic Consequences of Abdominal Closure and Long-Term ICU Consideration

In the damage-controlled patient, a tension-free fascial closure may not be possible. This observation is supported by the recognition that, during the abdominal closure, an increase in PIP can occur. Increasing inspiratory pressure indicates that the patient is not yet suitable for formal abdominal wall closure. At this juncture, skin closure only may be attempted. The rationale is that it provides a more biologic closure, although the surgeon must accept the presence of a ventral hernia that will require reoperation in 3 to 9 months. Another technique requires the suturing of an absorbable mesh to the fascia. This technique provides some evisceration protection and provides a substrate for granulation tissue to form. Split thickness skin grafting can then be performed when a bed of granulation tissue can support a graft. Again the patient will be required to return to the OR for a ventral herniorrhaphy at a later date [4,26,36,39]. If neither of these techniques is appropriate for the patient in damage control part III, a vacuum-assisted dressing can be reapplied. Both homemade and commercially available devices are available. Either technique allows for abdominal content expansion, limits the possibility of abdominal compartment syndrome, and permits continued evacuation of third space fluid. As the patient recovers, and bowel and retroperitoneal edema subsides, the vacuum-assisted dressing can

be changed at the bedside, typically every 2 to 3 days until the patient's abdomen is ready for formal closure.

Covering the open abdomen is an important step in restoring the patient's nutritional and metabolic losses. Until the abdomen is closed or covered, the patient will require nutritional support in excess of standard daily requirements. If the patient's abdomen remains open or edematous, total parenteral nutrition is used as the primary nutritional source. Once the abdomen has been closed, or has had mesh placed, enteral feeds are usually begun 24 hours after the patient's return to the ICU. The patient should be off any vasopressors and fully resuscitated. Tube feedings are then initiated at a low rate, typically 20 cc/h, to prevent mucosal atrophy during the patient's early recovery from lack of enteral feeding. The rate of tube feed administration is increased over the next 2 days to the patient's calculated goal. The rate of this incremental increase is based on the level of intestinal edema, the presence of enteric anastomosis, and the amount of residual tube feedings present. Additional protein supplementation is also recommended at 1.5 g/kg/d. The use of metabolic cart technology or the establishment of a positive nitrogen balance can serve as a guide to replenishing the patient's nutritional deficit.

An untreated source of infection can result in the development of sepsis. The patient typically presents with a worsening hemodynamic profile associated with high cardiac output and low systemic vascular resistance index. The patient often has an increasing white blood cell count and a low-grade fever. Unexplained hyperglycemia may be an early warning sign of impending sepsis. A septic source should be sought out and eradicated. Computed tomography surveillance at postoperative day 7 is standard practice at our institution when the patient has an elevated white count or fever after damage control surgery in an effort to identify an intra-abdominal abscess. In addition, levels of pancreatic enzymes and liver function tests are drawn to rule out pancreatitis and cholecystitis as possible infectious sources.

Treatment of the systemic inflammatory response requires a multifaceted approach. Typically, the patient has an ongoing fluid requirement and the patient's preload is optimized with crystalloid. Should hypotension persist, vasopressors are used. Initially dopamine is used to improve the patient's hypotension. If dopamine fails to improve the patient's blood pressure, norepinephrine infusion is then added and the infusion is adjusted to maintain a mean arterial pressure greater than 70 mm Hg

while the dopamine is weaned off. Vasopressin infusion is added at 0.01 to 0.04 U/min to aid in vasoconstriction and replenishment of vasopressin levels [41,42]. Vasopressin is usually weaned off over the next 72 hours as hemodynamic stability is achieved. If vasopressor support does not improve the patient's hemodynamic status, a random cortisol level should be drawn. If the cortisol level is less than 15 μg/dL, consideration should be given to emergent administration of dexamethasone for correction of adrenal insufficiency [43] with continued cortisol replacement with hydrocortisone at 100 mg every 8 hours [44].

Intra-abdominal abscess is another entity that has been recognized with increasing frequency [36]. Small abscess cavities can be eradicated using computed tomography–guided localization and drain placement. If that technique is unsuccessful, reoperation may be required. A leaking viscous in a previously damage-controlled patient provides one of the most hostile environments faced by the surgeon. With careful exploration, missed injuries are sought out and suture lines reinspected. If uncontrolled intra-abdominal infection is encountered as a result of a leaking viscous, consideration must be given to diversion of the intestinal flow proximal to the leak. A precautionary note: the creation of a stoma near an already open abdomen is very difficult to manage. If split thickness skin grafting can be completed, ostomy appliances can be applied to those grafted areas. Enterocutaneous or, more aptly titled, "enteroatmospheric" fistulas present even a larger problem. Enteroatmospheric fistulas can arise after damage control surgery and vent into the atmosphere and onto the granulating wound. Standard causes of fistulas should be eliminated.

The damage-controlled patient is often on a ventilator in excess of a week and often exceeds a month on the ventilator. Should ARDS develop, we use a low tidal volume, pressure-regulated style of ventilatory management with additional PEEP added to maintain oxygenation while preventing barotrauma from elevated PIP. The intensivist must also maintain vigilance for the development of pneumonia. Empiric antibiotics should only be used based on the ICU-specific antibiotic resistance profile for a selected organism. Moreover, culture-proven pneumonia should be treated judiciously with the least expensive available agents based on organism sensitivity. Early tracheostomy is used at our institution, typically being done during damage control part III. Tracheostomy allows for better pulmonary physiotherapy and improves the ease of bronchoscopy for quantitative cultures obtained during bronchoalveolar lavage. We have found that

early tracheostomy also shortens the number of ventilator days and improves patient comfort.

Summary

The principles of damage control surgery to the critically ill patient have been delineated. The damage control sequence includes a staged approach consisting of an initial operation, ICU resuscitation, and planned reoperation. The intensivist provides near-constant bedside presence during the early stages of management of the damage-controlled patient to the ICU. A high index of suspicion for complications must be maintained during the patient's stay in the ICU. Early recognition and expeditious treatment of these complications delineate the difference between success and failure.

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