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Damage control surgery

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Damage control surgery is defined as rapid termination of an operation after control of life-threatening bleeding and contamination followed by correction of physiologic abnormalities and definitive management. This modern strategy involves a staged approach to multiply injured patients designed to avoid or correct the lethal triad of hypothermia, acidosis, and coagulopathy before definitive management of injuries. It is applicable to a wide variety of disciplines. During the first stage of damage control, hemorrhage is stopped, and contamination is controlled using the simplest and most rapid means available. Temporary wound closure methods are employed. The second stage is characterized by correction of physiologic abnormalities in the ICU. Patients are warmed and resuscitated, and coagulation defects are corrected. In the final phase of damage control, definitive operative management is completed in a stable patient.

Historical perspective

Traditional surgical dogma dictates that an operation should be completed definitively regardless of the physiologic condition of the patient. This means that complex reconstructions may be performed in severely compromised patients, resulting ultimately in death. Strategies designed to avoid this inevitable outcome are not new to surgery. Battlefield victims with exsanguinating extremity injuries have undergone rapid amputation for thousands of years. Pringle described compression of liver injuries with packs and digital compression of the portal triad to stop massive hemorrhage from the liver in 1908 [1]. A modification of this technique was described by Halsted, who placed rubber sheets between the liver and packs to protect the hepatic parenchyma [2]. These techniques fell out of favor and rarely were used during World War II and the Vietnam Conflict [3].

Reports of perihepatic packing as a damage control technique reappeared in the 1970s and 1980s. Lucas and Ledgerwood reported the use of temporary perihepatic

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packs in 3 of 637 liver injuries treated at Detroit General Hospital in 1976 [4]. Feliciano reported a 90% survival rate in 10 patients with severe liver injuries who were packed in 1981 [5]. The modern concept of abbreviated laparotomy first was described by Stone in 1983 [6]. In this report, the group from Emory described immediate cessation of the operation when coagulopathy was noted. Abdominal hemorrhage was controlled by tamponade; bowel injuries were resected, and the ends of the bowel were oversewn. Noncritical vessels and injured ureters were ligated, and biliopancreatic injuries were drained. Following correction of the coagulopathy, these patients were returned to the operating room for definitive repairs. Using these techniques, 11 of 17 patients survived, as compared with 1 of 14 patients treated using traditional strategies.

The term "damage control" was popularized by Rotundo in the 1990s [7]. The strategy has gained immense popularity around the world since that time, and it has become the standard of care for severely injured patients. Damage control principles can be applied to all disciplines of trauma care. A recent review by Shapiro et al identified over 1000 trauma patients who were treated using these modern techniques [8].

The lethal triad

The philosophy of damage control is to abbreviate surgical interventions before the development of irreversible physiologic endpoints. Uncontrolled hemorrhage and iatrogenic interventions ultimately result in the development of hypothermia, coagulopathy, and acidosis. Each of these life-threatening abnormalities exacerbates the others, contributing to a spiraling cycle that rapidly results in death unless hemorrhage is stopped, and the abnormalities reversed. This bloody vicious cycle is depicted in Fig. 1.

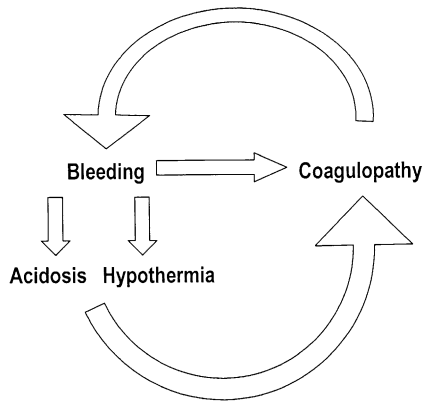

Fig. 1. The bloody vicious cycle.

Hypothermia

The definition of hypothermia in humans is a core temperature less than 35°C. Etiologies of hypothermia in trauma patients are numerous. The greatest potential for heat loss occurs secondary to fluid resuscitation [9]. Heat loss is proportional to the mass of fluid used to resuscitate the patient and the temperature gradient from the patient to the fluid. The equation for heat loss is: $Q = mc(T_2 - T_1)$, where Q equals heat in kilojoules, m equals mass in kilograms, and c equals the specific heat of water, which is 4.19 kJ/kg/°C. The heat lost when a single liter of room temperature fluid is given is to a patient with a temperature of 37°C is shown:

$$Q = (1\ \text{kg})(4.19\ \text{kJ/kg/°C})(37°\text{C} - 25°\text{C})$$

$$Q = 50.3\ \text{kJ}$$

As a comparison, Table 1 shows the approximate rate of heat transfer with available rewarming methods. Massive resuscitations seen with patients undergoing damage control can result in a tremendous amount of heat loss.

Advanced trauma life support principles dictate that all trauma patients should be exposed. This results in further heat loss because of convection and radiation. Patients with wet clothing suffer evaporative losses. Operative therapies require large incisions producing significant evaporative heat loss from peritoneal and pleural surfaces. Evaporative heat loss in the operating room is proportional to the size of the incision and the length of the procedure. Furthermore, blood loss results in decreased oxygen consumption, resulting in decreased heat production by the body.

Gregory et al studied the incidence of hypothermia in 100 consecutive trauma patients undergoing operation and found that the incidence at completion of the operation was 57% [10]. Hypothermia in trauma patients has been associated with a poor outcome. Mortality increases significantly in trauma patients with a core temperature less than 34°C and approaches 100% in trauma patients with a core temperature less than 32°C [11,12]. Hypothermia affects all body systems. The

Table 1
Approximate rate of heat transfer with available rewarming methods

Rewarming technique	Heat transfer (kJ/h)
Airway rewarming	33.5–50.3
Overhead radiant warmer	71.2
Heating blankets	83.8
Convective warmers	62.8–108.9
Body cavity lavage	150.8
Continuous arteriovenous rewarming	385.5–582.4
Cardiopulmonary bypass	2974.9

From Gentilello LM. Practical approaches to hypothermia. In: Maull KI, Cleveland HC, Feliciano DV, Rice CL, Trunkey DD, Wolferth CC, editors. Advances in Trauma and critical care, Volume 9. St. Louis: Mosby; 1994, p. 39–79; with permission.

detrimental effects include decreased heart rate and cardiac output, increased systemic vascular resistance, arrhythmias, decreased glomerular filtration rate, impaired sodium absorption, and central nervous system depression.

Hypothermia also results in coagulopathy because of effects on the coagulation cascade, platelets, and increased fibrinolytic activity [13]. Coagulation is dependent on a series of temperature-dependent reactions. Reed et al studied the effect of hypothermia on the prothrombin time (PT), thrombin time, and partial thromboplastin time (PTT) and found that these assays are prolonged significantly below a temperature of 35°C (Fig. 2) [14]. This effect is underestimated in the clinical scenario, because all clotting assays are performed at 37°C. In addition to the effects on coagulation reactions, hypothermia also causes platelet sequestration in the portal circulation and platelet dysfunction as measured by the bleeding time [15,16].

Acidosis

Metabolic acidosis in trauma patients occurs primarily as a result of lactate production from anaerobic metabolism. Hemorrhage results in decreased oxygen delivery secondary to decreased cardiac output and anemia. The failure to normalize either an abnormal lactate concentration or base deficit by 48 hours after injury has been correlated with mortalities ranging from 86% to 100% [17,18]. Resuscitation with fluids rich in chloride also has been associated with acidosis in trauma patients. This occurs most prominently with normal saline resuscitation but also may occur with lactated Ringer's resuscitation. The presence of hyperchloremic acidosis does not correlate with mortality in surgical ICU patients [19].

The detrimental effects of acidosis include depressed myocardial contractility, diminished inotropic response to catecholamines, and ventricular arrhythmias [20,21]. Acidosis also results in increased intracranial pressure, which may exacerbate outcomes in patients with head injuries. In addition to these systemic effects, acidosis also contributes to coagulopathy. Acidosis has been shown to independently result in prolongation of the PTT and decreased factor V activity [22]. Acidosis also may cause disseminated intravascular coagulation and a consumptive coagulopathy [23].

Coagulopathy

The etiologies, effects, and treatment of coagulopathy in trauma patients are discussed by Dr. DeLoughery elsewhere in this issue. In addition to hypothermia and acidosis, dilution contributes to coagulopathy. Primary trauma resuscitation fluids include crystalloids, colloids, and packed red blood cells. These fluids are devoid of coagulation factors. Tissue factor exposure secondary to trauma results in activation of the coagulation cascade and consumption of coagulation factors.

The combined effects of dilution and hypothermia on coagulation have been studied by Gubler et al [24]. Blood taken from trauma victims at Harborview Medical Center was diluted with phosphate buffered solution until the PT and PTT

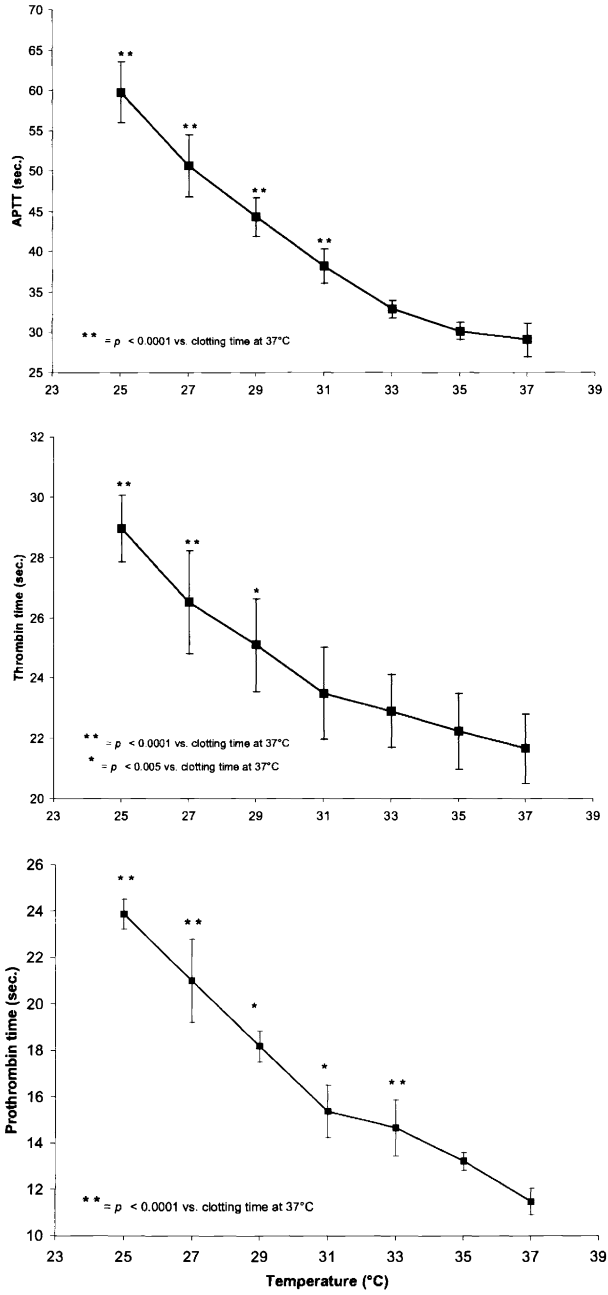

Fig. 2. The effect of temperature on activated partial thromboplastin time, thrombin time, and prothrombin time. (*From* Reed RL, Bracey AW, Hudson JD, Miller TA, Fischer RP. Hypothermia and blood coagulation: dissociation between enzyme activity and clotting factor levels. Circ Shock 1990;32:141–52; with permission from Wiley–Liss, a subsidiary of John Wiley & Sons.)

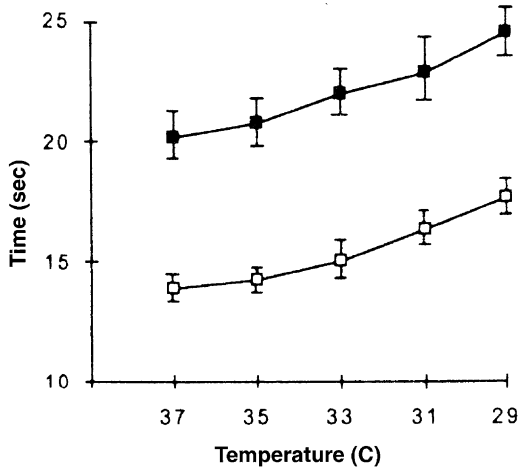

Fig. 3. The effect of hypothermia and dilution on prothrombin time. (*Filled squares*) diluted specimens, (*open squares*) nondiluted specimens. (*From* Gubler KD, Gentilello LM, Hassantash SA, Maier RV. The impact of hypothermia on dilutional coagulopathy. J Trauma 1994;36(6):847–51; with permission.)

were approximately 1.5 times baseline. Coagulation assays then were run on diluted and nondiluted specimens at temperatures ranging from 29°C to 37°C. The effects of dilution and hypothermia were found to be additive (Fig. 3). Fluid resuscitation before hemorrhage control also raises intravascular hydrostatic pressure, potentially causing displacement of established clots and increased bleeding. Immediate resuscitation of trauma victims with penetrating torso injuries has been shown to result in a higher mortality than delaying resuscitation until operative therapy is initiated [25].

The damage control procedure

Initial management

The initial management of trauma patients is based on principles described in the Advanced Trauma Life Support course. A systematic evaluation of the patient is performed focusing on treating life-threatening injuries. Patients with life-threatening injuries who require operative intervention are transported to the operating room rapidly. Efforts designed to avoid the lethal triad are implemented throughout this process. The operating room temperature is elevated, and resuscitation fluids are either warmed or given through the Level I rewarmer, which can deliver fluids at a maximum temperature of 41°C. The ventilator circuit is warmed, and a Bair Hugger (Augustine Medical, Eden Prairie, Minnesota) is placed on exposed areas. Because of the need for massive resuscitation and monitoring, central venous access is indicated. In massively hemorrhaging patients, early consider-

ation is given to replacement of coagulation factors with fresh frozen plasma, platelets, and cryoprecipitate.

Selection of patients who will benefit from damage control techniques is based on the constellation of injuries and the physiologic status of the patient. Patients with severe injuries that are technically difficult to repair or require extensive surgical procedures and patients with exsanguinating blood loss are the best candidates. Attempts have been made to define physiologic criteria for the initiation of damage control based on transfusion rate, hypothermia, coagulopathy, and acidosis, but this has not been standardized [26]. Ultimately, the surgeon must make the decision that definitive repair will result in an irreversible physiologic insult and likely death.

Stage 1—the damage control operation

Abdomen

Most damage control procedures are performed in the abdomen [27]. The specific technique used is dependent on the nature of the injury. The most common abdominal injuries that induce a damage control approach are liver injuries and abdominal vascular injuries [26,27]. The primary method of hemorrhage control for complex liver injuries is packing. Packing is performed using laparotomy pads placed with a goal to compress the source of hemorrhage. Retrohepatic vena caval injuries are treated by anterior packing of the liver, which compresses the vena cava. Other liver injuries frequently require anterior and posterior packing to compress the hepatic parenchyma. The goal is to tamponade bleeding while maintaining organ perfusion. Plastic drapes may be placed between the hepatic parenchyma and the packs to avoid displacement of clots when the packs are removed.

Several options exist for the abbreviated treatment of abdominal vascular injuries. Some vascular injuries may be treated successfully with packing. Ongoing bleeding following packing requires a more aggressive approach. Many abdominal vascular injuries can be treated safely by simple ligation [28–34]. Simple ligation is not tolerated in patients with aortic or proximal superior mesenteric artery injuries, and it is not feasible in patients with retrohepatic vena cava injuries.

Temporary intraluminal shunts are relatively easy to place, and they maintain end organ perfusion, allowing early termination of laparotomy. These shunts are secured in place, using silk sutures, rubber vessel loops, or umbilical tapes. The size of the shunt is determined by the size of the injured vessel. The largest shunt that easily fits within the vessel should be used. Argyle carotid shunts and Javid shunts have been used for this purpose [35,36]. Chest tubes may be used when larger conduits are required, as in the case of an aortic or inferior vena cava injury.

The largest series of temporary intravascular shunts was reported by Granchi et al from Ben Taub General Hospital, Houston, Texas [35]. In this series, 19 patients underwent shunt placement as a damage control technique. One of these shunts was placed in the iliac artery; the remaining shunts were placed in

extremity vessels. Shunts were left in place from 47 minutes to 4 days, and heparinization was not required to maintain patency. Argyle carotid shunts were used in all patients. Reports of prolonged use of temporary shunts for superior mesenteric artery and portal vein injuries also exist [36].

Another potentially lifesaving technique for exsanguinating patients with abdominal vascular injuries involves the use of inflatable balloon catheters. Indications for using inflatable balloon catheters include persistent hemorrhage from inaccessible locations or difficulty in controlling the injured vessel. Thirty milliliter Foley balloon catheters and number 3 to number 8 Fogarty balloon catheters have been used for this purpose [37,38].

Intravascular balloon catheters may either be placed directly into the entrance wound of the missile or knife track, directly into the defect in the injured vessel, or into the proximal part of an injured artery. They may be placed prograde or retrograde through an arteriotomy or venotomy. The balloon is advanced serially and inflated until hemorrhage control is achieved. The balloon is inflated with either saline or radiologic dye, depending on the need for visualizing the balloon postoperatively. This technique may permit immediate repair of the injured vessel or provide time to correct physiologic abnormalities before definitive repair.

Patients with penetrating abdominal trauma frequently suffer multiple injuries. In addition to solid organ and vascular injuries, hollow viscus and bilio-pancreatic injuries may occur. Hollow viscus injuries are treated by resection of affected areas using stapling devices. Reanastomosis is postponed until the patient is stabilized and returned to the operating room for definitive operation. The majority of bilio-pancreatic injuries can be temporized with closed suction drainage [39].

Potentially injured urologic structures include the kidney, ureters, and bladder. Patients with extensive kidney injuries that are reaching their physiologic limits frequently are best treated with rapid nephrectomy, especially if they do not respond to packing. Options for the management of patients with ureteral injuries who are undergoing damage control procedures include ligation and exteriorization [40]. Ligation of a transected ureter will result in obstruction of the nephric unit, which may be treated by temporary nephrostomy if definitive laparotomy is delayed for a prolonged period. Another option for the management of ureteral injuries is placement of a percutaneous ureterostomy. A tube is placed in the open end of the proximal ureter and brought out through the skin. This option avoids ureteral obstruction and the need for a nephrostomy. Most bladder injuries can be rapidly sewn closed with a single layer running suture or temporized using closed suction drainage.

After controlling hemorrhage and contamination, a decision must be made concerning the temporary management of the abdominal wound. The goals of temporary closure include containment of the abdominal viscera, control of abdominal secretions, maintenance of pressure on tamponaded areas, and optimizing the likelihood of ultimate abdominal closure. Formal closure of the abdominal fascia after damage control laparotomy has been associated with an increased risk of abdominal compartment syndrome (ACS), adult respiratory distress syndrome (ARDS), and multiple organ failure (MOF) [41].

Multiple options for the temporary closure of the abdomen exist. Simple options include closure of the skin alone using towel clips or a running suture [26]. This technique does not expand the abdominal volume significantly and still may result in the ACS. The Bogota bag is a sterilized 3 L urologic bag that is sewn to the skin [42,43]. This is an inexpensive method of containing the viscera and abdominal secretions, but it allows the abdominal fascia to retract, potentially lessening the likelihood of successful closure later.

Vacuum-assisted techniques for the temporary management of the abdomen have been described [44–46]. A nonadherent plastic drape with small perforations is placed over the bowel. A towel is placed over the drape, followed by two closed suction drains. An adherent drape then is placed over the skin surrounding the wound, creating an airtight seal. A vacuum is achieved by placing the drains on suction. In addition to containment of the viscera, this technique maintains some tension on the abdominal fascia and allows accurate quantification of abdominal fluid output and maintenance of a relatively sterile environment.

The final option for temporary closure of the abdomen involves sewing a prosthesis to the abdominal fascia. Choices for the prosthesis include absorbable mesh, nonabsorbable mesh, or Silastic [47–49]. The theoretical advantage of these techniques is that they maintain constant tension on the fascia, increasing the likelihood of definitive closure. A relatively new technique involves the use of synthetic Velcro [50]. Velcro is sewn to either side of the abdomen and closed (Fig. 4). The use of Velcro allows the surgeon to adjust abdominal tension frequently and at the bedside. Randomized controlled trials comparing methods for temporary abdominal closure do not exist, so that the method chosen tends to be institution-specific.

Chest

Trauma patients with possible thoracic injuries who are in extremis should undergo emergency department thoracotomy. This procedure permits rapid access to the thoracic cavity. If present, cardiac tamponade is treated by opening the pericardium. Cardiac injuries may be temporized by digital pressure or the careful use of a Foley catheter to tamponade bleeding. Additional interventions that can be performed include clamping the pulmonary hilum or twisting the lung along its hilar axis to stop exsanguinating bleeding from the pulmonary parenchyma. The right atrium can be cannulated with a Foley catheter for massive resuscitation. Packing sometimes may be effective at stopping chest wall bleeding. The descending aorta also may be cross-clamped to reduce blood flow to distal injuries and to increase brain perfusion (Fig. 5). Emergency department thoracotomy rarely results in a successful outcome in blunt trauma victims or in patients with extrathoracic penetrating injuries [51,52].

Some penetrating pulmonary parenchymal injuries are amenable to damage control techniques. Deep through-and-through lung injuries that do not involve hilar vessels or the main airways traditionally have been treated with formal lung resections to include lobectomy or pneumonectomy. These lesions can be treated using a technique known as pulmonary tractotomy [53]. A linear cutter stapler is

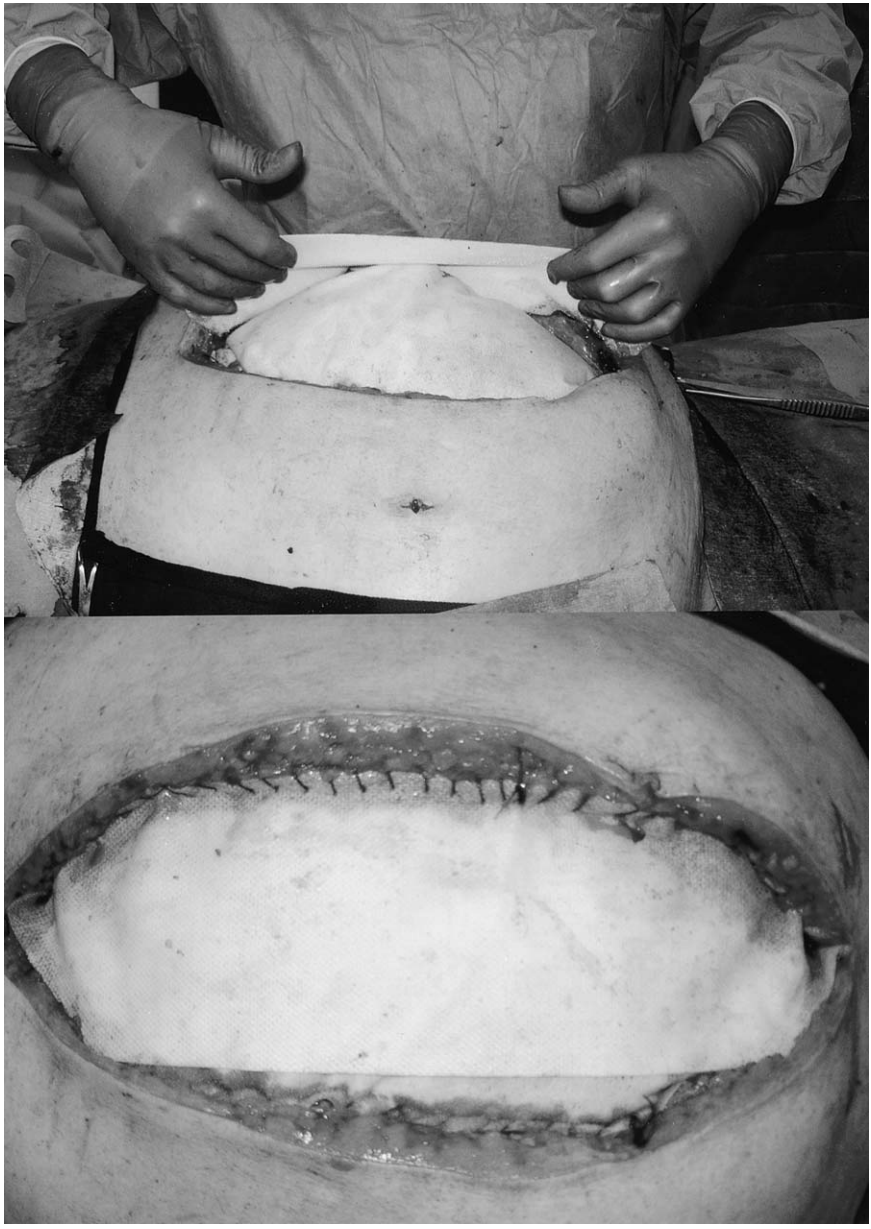

Fig. 4. An example of the use of a Velcro patch to close the abdomen while maintaining some tension on the fascia. The patch can be adjusted frequently, theoretically enhancing the likelihood of successful closure of the fascia.

Fig. 5. A picture of an emergency department thoracotomy. The clamp is on the descending aorta, and there is a Foley catheter in the right atrium for fluid resuscitation.

placed within the injury tract and fired. This exposes the base of the tract, permitting bleeding points and air leaks to be ligated selectively with sutures. Patients undergoing tractotomy have been shown to have a significantly lower mortality than patients undergoing formal pulmonary resections [54].

Stage 2—resuscitation

Hypothermia

Following the damage control procedure, patients are returned to the ICU for correction of their physiologic abnormalities. Evidence of the lethal triad is frequently most apparent immediately following the operation, and aggressive measures are indicated. Initial operating room conditions should be recreated in the ICU. The room and airway circuit should be warmed, and a Bair Hugger should be applied to the patient. All fluids and blood products should be warmed, and fluid boluses should be given through the level I rewarmer.

Continuous arteriovenous rewarming is a relatively new technology that permits rapid rewarming of hypothermic patients without requiring cardiopulmonary bypass or heparinization. This technique originally was described by Gentilello, and it involves the placement of 8.5 F femoral arterial and venous catheters to create an arteriovenous fistula that diverts part of the cardiac output through a heat

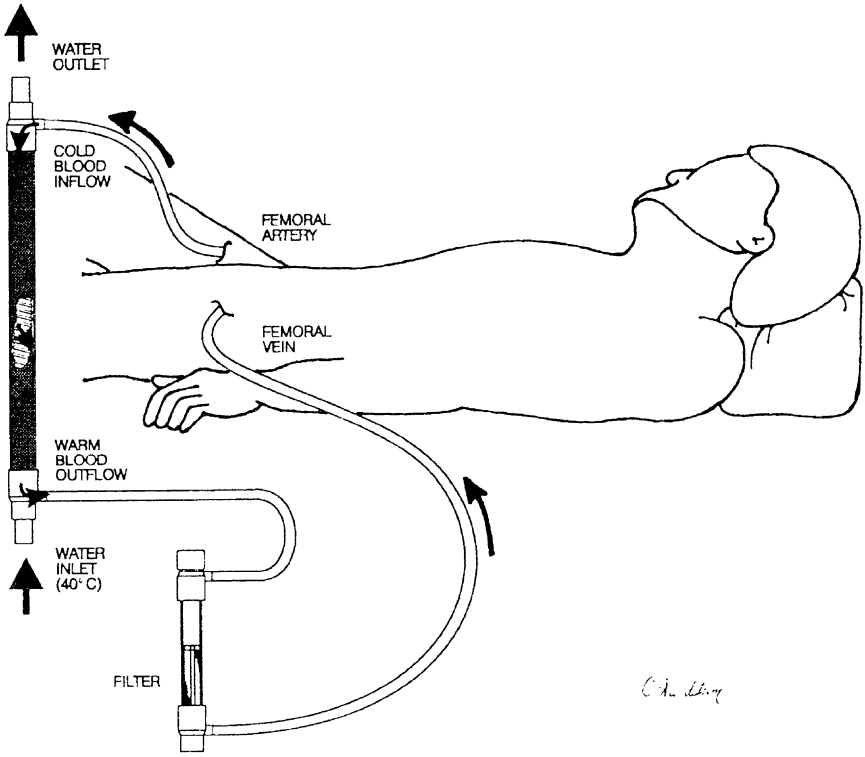

Fig. 6. Continuous arteriovenous rewarming. (*From* Gentilello LM, Jurkovich GJ, Stark MS, Hassantash SA, O'Keefe GE. Continuous arteriovenous rewarming: rapid reversal of hypothermia in critically ill patients. J Trauma 1992;32(3):316–27; with permission.)

exchanger [55,56]. Blood flows out of the femoral arterial catheter into tubing attached to the level I rewarmer. The blood is warmed and flows back into the body through the femoral venous catheter (Fig. 6). The effectiveness of this technique is dependent on the patient's cardiac output. Continuous arteriovenous rewarming has been shown to significantly reduce time to normothermia, resuscitation requirements, and early mortality in hypothermic trauma patients who are critically injured [57].

Acidosis

Treatment of acidosis in damage control patients requires optimization of oxygen delivery. The determinants of oxygen delivery are cardiac output, hemoglobin, and the oxygen saturation of arterial blood. Hemorrhage results in decreased preload, cardiac output, and anemia. For these reasons, blood is an ideal resuscitation fluid.

Central venous monitoring is generally adequate for young healthy patients during the resuscitation phase. Elderly patients, who may suffer from comorbidities, and patients with cardiopulmonary injuries are more likely to benefit from pulmonary artery monitoring. These patients may have underlying cardiac disease that contributes to inadequate oxygen delivery, and they may require inotropic support.

Persistent acidosis in adequately resuscitated trauma patients is almost always secondary to hyperchloremic acidosis [19]. Inaccurate interpretation of hyperchloremic acidosis may result in excessive resuscitation with fluid or blood and delay in definitive operation. Hyperchloremic acidosis and lactic acidosis usually can be discriminated by determination of the anion gap. Hyperchloremic acidosis results in a narrowed anion gap, while lactic acidosis results in a widened anion gap. Borderline cases can be differentiated by lactate measurement. Hyperchloremic acidosis rapidly resolves when resuscitation fluids are changed to sodium acetate, which contains no chloride. The benefit of this intervention has not been proven in randomized prospective trials.

Coagulopathy

Correction of hypothermia and acidosis significantly contribute to normalization of coagulation. Damage control patients require aggressive blood product replacement with fresh frozen plasma, platelets, and cryoprecipitate. Recombinant factor VIIa (rFVIIa) also has been shown to correct coagulopathic defects rapidly in animal models of trauma and small case series of severely injured trauma patients [58–61]. An extensive discussion of the management of coagulopathy in trauma patients by Dr. DeLoughery is included elsewhere in this issue.

Stage 3—definitive operation

Following reversal of the lethal triad, the patient is returned to the operating room for definitive treatment. The exact timing of reoperation has not been standardized. Patients who are returned to the operating room in less than 72 hours have been shown to have reduced morbidity and mortality, compared with patients who return later [62]. Premature return to the operating room may result in rebleeding and the need for additional operations.

During the definitive procedure, a complete exploration is performed to ensure that no injuries were missed. Packs are removed, and bleeding sites are identified and treated when possible. Small bowel continuity is restored, and patients with large bowel injuries are treated with exteriorization or repair depending on the extent of their injuries and their physiologic state. Consideration is given to closing the abdominal fascia, but this may not be possible because of diffuse edema secondary to injury and resuscitation. In the series by Hirshberg et al, 80% of patients required only a single reoperation [27]. Additional operations were required in patients who had an unplanned second operation for bleeding or unexpected complications and in patients who could not be closed successfully.

Results of damage control

Complications

Complications following damage control procedures may be related to the site of original injury or to the systemic complications of injury, hemorrhage, massive resuscitation, and infection. Examples of local complications include abscess, fistula formation, intestinal necrosis, and pancreatic pseudocyst. Systemic complications include ARDS and MOF. Massive resuscitation associated with ischemia and reperfusion also results in acute visceral swelling and decreased abdominal compliance, potentially causing ACS.

Abdominal compartment syndrome

Abdominal compartment syndrome is defined as end-organ dysfunction secondary to intra-abdominal hypertension. Patients present with a tensely distended abdomen, elevated peak airway pressures, inadequate ventilation, inadequate oxygenation, and oliguria. These processes are reversed with decompression of the abdomen. The physiologic effects of intra-abdominal hypertension are widespread, resulting from several factors, including diminished venous return, abdominal vein compression, upward displacement of the diaphragm, and increased intrathoracic pressure.

Hemodynamic effects include decreased cardiac output, hypotension, increased central venous pressure, increased pulmonary capillary wedge pressure, and increased systemic vascular resistance. Pulmonary effects include decreased compliance, decreased oxygenation, and hypercarbia. Decreased urine output has been shown to be related primarily to renal vein compression, although decreased cardiac output also may play a role [63]. Decreased venous return also results in increased intracranial pressure and decreased cerebral perfusion pressure, potentially exacerbating outcomes in head injury patients [64,65].

Although the diagnosis of ACS is made primarily on a clinical basis, the indirect measurement of intra-abdominal pressure adds important information. Bladder pressure has been shown to correlate closely with intra-abdominal pressure [66,67] and to correlate with onset of organ dysfunction [68]. Clinical evidence of organ dysfunction occurs in some patients when the bladder pressure exceeds 16 mm Hg, and nearly all aspects of ACS are manifested in patients with bladder pressures greater than 35 mm Hg.

Abdominal compartment syndrome has been reported to occur in approximately 30% of patients undergoing damage control celiotomy [41,69]. This incidence is reduced significantly when closure of the fascia is avoided before definitive operation. ACS also has been described in trauma patients without abdominal injuries [70,71]. This process is referred to as secondary ACS. These patients typically present in shock and receive massive resuscitations. In the study by Balogh et al, the mean 24-hour resuscitation requirement of patients with secondary ACS was 38 L of crystalloid and 26 units of packed red blood cells.

Table 2
Mortality following damage control surgery

Author	n	Year	Population	% Mortality
Stone et al [6]	17	1983	Abdomen	35
Feliciano et al [73]	66	1986	Liver	67
Burch et al [26]	200	1992	Abdomen	67
Rotundo et al [7]	24	1993	Abdomen	45
Morris et al [69]	107	1993	Abdomen	26
Hirshberg et al [27]	124	1994	Abdomen and chest	58
Abikhaled et al [62]	35	1997	Abdomen	20
Aoki et al [75]	68	2000	Abdomen	66
Offner et al [41]	52	2001	Abdomen	33
Johnson et al [73]	21	2001	Abdomen	10
Total	714			51

Outcome

Patients who undergo damage control procedures are at high risk for ARDS, MOF, and death. The independent risk factors for ARDS in trauma patients include the presence of sepsis, transfusion of more than 15 units of packed red blood cells in 24 hours, pulmonary contusion, and long bone fractures [72]. These are common events in damage control patients. The reported incidence of ARDS or MOF ranges between 14% and 53%, depending on the series [6,26,41,73].

Several studies have shown improved outcomes since the widespread institution of damage control techniques [6,7,74]. These studies primarily use historical and unmatched controls for comparison. Mortality following damage control procedures is shown in Table 2. Initial pH upon return to the ICU after the damage control procedure and the worst PTT from hospital admission to ICU admission have been shown to correlate highly with mortality [75]. In a study by Aoki et al, all patients who returned to the ICU with a pH less than or equal to 7.2 died, whereas 88% of patients who returned with a pH of greater than 7.33 lived. All patients whose pH was between 7.2 and 7.33 and whose PTT was greater than or equal to 78.7 seconds died. Eighty-two percent of patients survived if their pH was between 7.2 and 7.33 and their PTT was greater than 78.7 seconds.

Summary

Damage control is a staged approach to severely injured patients predicated on treatment priorities. Initially, life-threatening injuries are addressed expediently, and procedures are truncated. Normal physiology is restored in the ICU, and patients subsequently are returned to the operating room for definitive management. This strategy breaks the bloody vicious cycle and results in improved outcomes. Novel technologies like CAVR and rFVIIa contribute to the effectiveness of damage control.

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