Damage Control: Collective Review

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Advances in prehospital care and trauma resuscitation have enabled the early survival of many injured patients who previously had a high chance of dying at the accident scene or en route to the hospital. The change in the spectrum of injury severity, characterized by high-energy blunt trauma with multiple-organ injury and fractures, and the emergence of semiautomatic handguns with multiple penetrating wounds, present new challenges to all surgeons. In conventional trauma care, definitive control and repair of all injuries may be accomplished in the immediate postinjury setting; however, the physiologic derangements of the massive shock state caused by the aforementioned injury patterns often lead to a fully repaired but dead patient. In response to these catastrophic challenges, the concept of "damage control" as a treatment merely to control but not definitively repair injuries has arisen. This term was originally coined by the United States Navy, in reference to "the capacity of a ship to absorb damage and maintain mission integrity."[1] In the patient with multiple injuries who is exsanguinating, this has been paraphrased to indicate the sum total of the maneuvers necessary to ensure patient survival above all else.[2]

Definitive control of hemorrhage by pressure is not new to surgery. Pringle first enunciated the principles of compression and hepatic packing for control of portal venous hemorrhage in 1908.[3] This was modified by Halsted, who inserted rubber sheets between the liver and packs to protect hepatic parenchyma.[4] Military experience in World War II and Vietnam discouraged this practice.[5] As early as 1963, however, Shaftan et al. observed that to limit the mortality of liver injury, both faster and better resuscitation and better treatment of the wounds were necessary.[6] In 1979, Calne et al. described four cases in which massive exsanguinating hemorrhage from the liver was temporarily controlled with gauze packing, enabling safe transfer and definitive management at a more appropriate institution.[7]

In 1983, Stone and associates popularized the technique of truncation of laparotomy, establishment of intra-abdominal pack tamponade, and then completion of definitive surgical repair later, once coagulation had returned to an acceptable level. This proved to be lifesaving in previously nonsalvageable situations.[8] Damage control, abbreviated laparotomy specifically to salvage trauma patients with exsanguination, was described at several institutions almost simultaneously in the early 1990s.[9–11] Rotondo et al. found a remarkable salvage rate of over 70% in a limited number of patients treated with damage control for abdominal vascular injury and massive shock, hypothermia, and acidosis. Since then, damage control has gained widespread use throughout North America, Israel, and South Africa. Recently, a review by Rotondo and Zonies identified 961 damage control patients in the literature, with 50% mortality and 40% morbidity overall.[12] Subsequent reports have expanded this list to over 1,000 patients (Table 1).

Damage control as currently practiced has three separate components. The first is abbreviated resuscitative surgery for rapid control of hemorrhage and contamination. This is obtained as quickly as possible in the operating room, but traditional repairs are deferred in favor of rapid measures that control hemorrhage, restore flow where needed, and control or contain contamination. Intra-abdominal packing and temporary abdominal closure complete this truncated first and critical step (Part I). The patient is then moved to the intensive care unit, where Part II consists of ongoing core rewarming, correction of coagulopathy, fluid resuscitation and optimization of hemodynamic status, as well as reexamination to diagnose all injuries. When normal physiology has been restored, reexploration is undertaken for definitive management of injuries and abdominal closure (Part III).[9,13]

Increases in firearm violence present a new source of challenge to which damage control techniques are often applicable. In one study from the United States in an urban setting, firearm-related homicide increased by 123% over 5 years, and the number of victims who died at the scene rose from 5% to 34%. This was despite the designation of six trauma centers in that county, reflecting a shift toward high-velocity, high-caliber weaponry.[14] It has been apparent in our clinical practice for some time that inner city street weaponry and wounding patterns are changing, with multiple-shot injury patterns becoming much more common. These patients often present with multicavitary sites of exsanguination, and damage control techniques have assumed a prominent role in their initial management.

This article reviews the principles of damage control as a series of linked surgical maneuvers, designed to address the

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Table 1 Cumulative Review of Damage Control

Year	Author	N	Mortality	% Mortality	Morbidity	% Morbidity
1976	Lucas	3	0/3	0	—	—
1979	Calne	4	0/4	0	—	—
1981	Feliciano	10	1/10	10	6/9	67
1982	Svoboda	12	2/12	17	—	—
1983	Stone	17	6/17	35	11/11	100
1984	Carmona	17	2/17	12	5/15	33
1986	Baracco	36	6/36	17	4/36	11
1986	Ivatury	14	8/14	57	5/6	83
1986	Feliciano	66	38/66	58	9/49	19
1988	Cogbill	52	31/52	60	3/21	14
1990	Saifi	9	2/9	22	6/9	67
1990	Beal	49	19/49	39	7/30	23
1990	Aprahamian	20	4/20	20	9/16	56
1990	Cue	35	17/35	49	19/21	90
1992	Krige	22	6/22	27	12/16	75
1992	Burch	200	134/200	67	38/86	44
1992	Sharp	39	17/39	44	6/22	27
1992	Shen	6	3/6	50	—	—
1992	Talbert	11	4/11	36	3/7	43
1993	Carillo	14	2/14	14	9/12	75
1993	Rotondo	24	10/24	42	5/14	36
1993	Morris	107	64/107	60	22/43	51
1994	Hirshberg	124	72/124	58	—	—
1996	Garrison	70	47/70	67	6/38	16
1996	Richardson	21	8/21	38	—	—
1997	Ivatury	1	0/1	0	0/1	0
1997	Porter	1	0/1	0	0/1	0
1997	Shaftan	5	0/5	0	2/5	40
1997	Carillo	3	0/3	0	2/3	67
1998	Chang	6	0/6	0	6/6	100
1998	Demetriades	3	0/3	0	0/3	0
Total		1,001	503/1,001	50	193/480	40

Adapted with permission from Rotondo MF, Zonies DH: The damage control sequence and underlying logic. *Surg Clin North Am.* 1997;77:761–777.

physiologic abnormalities first, followed by a secondary resuscitative phase, and then the definitive surgical procedures themselves. Though damage control was traditionally described for massive abdominal trauma with vascular injury, recent applications in the chest and even to peripheral vascular injury have been reported.[15–17]

GENERAL CONSIDERATIONS AND SECONDARY RESUSCITATION

The goal of the damage control approach is to preserve the living patient. The triad of hypothermia, acidosis, and coagulopathy in the patient with multiple injuries is often lethal; Ferrara et al. reported 90% mortality in patients with these findings requiring massive transfusion.[18] Rewarming, replacement of coagulation factors, and fluid and blood resuscitation are critical to counter this state.[11] Damage control encompasses this algorithm, emphasizing rapid but definitive hemostasis, closing all hollow viscus injuries or performing only essential bowel resections, and delaying the more traditional or standard reconstruction until after the patient has been stabilized and all physiologic parameters have been corrected.

Primary Operation and Hemorrhage Control

The initial damage control laparotomy (Part I) includes five components: control of hemorrhage, exploration, control of contamination, definitive packing, and rapid abdominal closure.[19] An important distinction should be made between resuscitative and therapeutic packing. Resuscitative packing with manual compression of a bleeding site is often used as an initial short-term (minutes) measure to control or minimize blood loss while repairing other higher priority injuries or "catching up."[20] Therapeutic packing, in contrast, provides tamponade of bleeding when it is surgically unmanageable or a coagulopathy has developed. This is used to enable a longer period of resuscitation, to give the body time to correct the metabolic derangements mentioned above,[21] and to access other means of definitive vascular control such as therapeutic angiography.

The decision to truncate the procedure should be made early when, in the judgment of the surgeon, definitive repair is either likely to exceed the patient's physiologic reserve or is technically impossible.[22] The indications for damage control have recently been described in six general categories

Table 2 Indications for the Damage Control Approach

1. Inability to achieve hemostasis due to coagulopathy
2. Inaccessible major venous injury
3. Time-consuming procedure in a patient with suboptimal response to resuscitation
4. Management of extra-abdominal life-threatening injury
5. Reassessment of intra-abdominal contents
6. Inability to reapproximate abdominal fascia due to visceral edema

Modified with permission from Moore EE, Burch JM, Franciose RJ, et al: Staged physiologic restoration and damage control surgery. *World J Surg.* 1998;22:1184–1191.

(Table 2).[23] Critical variables include the surgeon's ability to control hemorrhage, the severity of the injuries, and the presence of other associated injuries. Packing as a therapeutic procedure should be implemented well before massive blood loss (10–15 units of packed red blood cells) has occurred.[24,25] Other variables that have been identified as significant include severe injury (Injury Severity Score >35), hypotension (shock in excess of 70 minutes), hypothermia (temperature <34°C), coagulopathy (prothrombin time >19 seconds or partial thromboplastin time >60 seconds), and acidosis (pH <7.2).[5,26,27] With these caveats, the need for packing as a planned therapeutic approach can often be anticipated preoperatively. Packing does not take the place of vessel ligation or clamping. Most vessels can be rapidly isolated, repaired, ligated, or shunted if necessary. Once definitive vascular control is obtained, packing of all raw or dissected surfaces is done. Occasionally, with injury to the liver, pelvis, or large muscle beds, packing must be done and prompt angiography performed to control these intraparenchymal or intramuscular vessels. Regardless of the bleeding source, expeditious vascular control is of paramount importance. Without it, exsanguination is ensured.

Hypothermia, Acidosis, and Coagulopathy: The Lethal Triad

Hypothermia

Thermal homeostasis depends on a balance between the factors governing heat loss—conduction, convection, evaporation, and radiation—and the body's ability to generate and maintain metabolic energy. Heat loss begins at the moment of traumatic insult, and is exacerbated by extenuating circumstances such as shock and low perfusion, prolonged exposure, immobility of the acutely injured patient, and extremes of age. In the absence of a preemptive treatment approach, this process continues in the emergency department, where the patient is unclothed and left fully exposed, with a resultant patient–room temperature gradient of 15°C. In this setting, measurable temperature loss occurs in up to 92% of patients.[28,29] Clinically significant hypothermia is considered present when the core temperature is less than 35°C,[30] and temperature less than 34°C has been linked with a need for early therapeutic packing.[27]

Hypothermia has been reported in 21% of all severely injured patients, and up to 46% of trauma victims requiring laparotomy leave the operating room hypothermic.[29,31] Clinically, hypothermic patients have significantly greater fluid, transfusion, vasopressor and inotropic requirements, resulting in higher incidences of organ dysfunction, mortality, and markedly prolonged intensive care unit stay.[31–35] Hypothermia itself may not be the cause of these conditions, but it reflects the magnitude of the original injury and the associated shock state.

Passive external rewarming techniques include patient shivering and simple covering of the patient to minimize convective heat loss. Active external rewarming techniques include fluid-circulating heating blankets, convective warm air blankets, and radiant warmers. Paradoxically, the initial response to these techniques may be adverse, as fluid shifts and changes in vascular tone decrease venous return, lower blood pressure, and diminish cardiac output. The return of cold, acidotic blood to the central circulation may initially lower core temperature, and has been associated with ventricular fibrillation during rewarming.[36,37]

Active core rewarming techniques include warmed airway gases, heated peritoneal or pleural lavage, warmed intravenous fluid infusion, and extracorporeal rewarming. Cold (i.e., room temperature) intravenous fluid administration has been invoked as the fastest way of accelerating hypothermia.[28] Countercurrent heat exchange mechanisms have enabled the rapid infusion of warmed banked blood products.[38]

Extracorporeal rewarming techniques may be limited by the need for anticoagulation. Continuous arteriovenous rewarming, however, can be accomplished with a heparin-bonded circuit without a pump that may eliminate this limitation. This process is driven by the patient's blood pressure; hence, its effectiveness is limited by hypotension. This technique can accomplish rewarming at a rate of 4° to 5°C per hour, which is far more efficient than the 1° to 2°C possible with other methods. Because warmed blood is sent directly to core organs, continuous arteriovenous rewarming rapidly increases core temperature by nonshivering thermogenesis, which may increase metabolic effectiveness.[36]

Acidosis

Acidosis associated with hypovolemic shock contributes to coagulopathic bleeding, which worsens the shock state. Correction requires not only control of hemorrhage but also optimization of oxygen delivery, through blood transfusion and pharmacologic augmentation of cardiac output. Although controversial, a variety of resuscitative end points have been proposed that go beyond conventional "vital sign" and urinary output parameters, including serum lactate levels,[28,39] base deficit,[40,41] mixed venous oxygen saturation,[42] and gastric mucosal pH.[43]

Coagulopathy

Dilution of coagulation factors and platelets by fluid resuscitation, decreased total and ionized calcium concentration, hypothermia, the severity of injury, shock, and metabolic acidosis may all contribute to the dysfunction of normal hemostatic mechanisms.[44] The clinical observation of coagulopathy is not always confirmed by laboratory investigation, suggesting that elements other than the concentration of clotting factors and the number and function of platelets may be involved in the development of hemostatic failure.[18,45–48]

Hypothermia in the critically ill patient leads to dysfunction of intrinsic and extrinsic coagulation cascades.[49] Inhibition of the enzymatic reactions of these pathways is demonstrated by prolongation of prothrombin and partial thromboplastin times in hypothermic conditions, even where coagulation factors are known to be normal. Coagulation testing is normally performed at 37°C rather than at the patient's core temperature, and may underestimate the degree of coagulopathy.[50,51]

Platelet dysfunction in hypothermia leads to prolonged bleeding time, from a reversible, temperature-dependent defect in thromboxane B_2 production.[52] Changes in enzyme kinetics affected by temperature may also delay the initiation and propagation of platelet aggregation, despite the adequate replacement of platelet number.[18,38] There is often poor correlation between platelet count and continued bleeding in patients who have received massive transfusion, and the presence of continued hemorrhage in this setting is an indication for platelet transfusion even with a "normal" platelet count.[45–47,53–56]

Increased fibrinolytic activity in hypothermic conditions has been attributed to the stimulation of intrinsic catecholamines. Imbalances in the production and degradation of fibrin may also lead to excessive bleeding.[57,58]

Thromboelastography is a simple test that can broadly determine coagulation abnormalities and give information about fibrinolytic activity and platelet function that is not available from routine coagulation screens.[59] Thromboelastography documents the interaction of platelets with the protein coagulation cascade from initial platelet-fibrin interaction, through platelet aggregation, clot strengthening, and fibrin cross-linking, to clot lysis, within 20 minutes. It is practical for use in the operating room, simplifies the diagnosis of coagulopathy, and may be an early predictor of the need for transfusion in patients with blunt injury.[56,59]

Reported results of the damage control approach to coagulopathic bleeding have been generally positive. After an initial report by Ivatury et al. in 1986, showing no difference in mortality compared with historical controls, Feliciano et al. have reported high survival rates utilizing damage control techniques.[11,60,61] Subsequent reports have described successful application both in a wider array of trauma patients (i.e., nonhepatic injuries) and in noninjured general surgery patients.[5,62,63]

Abdominal Compartment Syndrome and Unpacking the Abdomen

Increases in intra-abdominal pressure may adversely affect systemic circulation and organ perfusion, especially the kidneys, heart, and brain.[64] This compartment syndrome can result from abdominal trauma accompanied by visceral swelling, hematoma, or the use of abdominal packs.[10] Venous return is decreased by direct caval compression and pooling of pelvic and lower extremity blood. In addition, increased abdominal pressure, associated diaphragmatic elevation, and a relative increase in afterload all combine to diminish cardiac output.[65,66] Visceral blood flow to the liver, intestines, and kidneys may all be reduced, and renal dysfunction to the point of anuria may also arise from renal vein compression. Direct compression of the kidneys can elevate renal vascular resistance and worsen this process.[67] Respiratory dysfunction usually results from increased abdominal pressure with resultant decreases in thoracic volume and lung compliance. Ventilation-perfusion mismatching worsens oxygenation. Paradoxically, hypoxia in this setting may worsen with positive end-expiratory pressure.[65,68,69] More thorough discussion of the pathophysiology and diagnosis of this syndrome is beyond the scope of this review, but is well described in the surgical literature.[70]

Perihepatic packing as a damage control procedure can be a cause of clinically significant abdominal compartment syndrome, with compression of the suprarenal vena cava leading to renal dysfunction. In this setting, a patient should be returned to the operating room for evacuation of clots and, if possible, removal of some of the laparotomy pads to decrease intra-abdominal pressure and relieve caval compression.[61,65] Decompression leads to improvement in visceral perfusion, cardiac function, and pulmonary mechanics.[67,71]

In the absence of this complication, packs should be left in place until the patient's hemodynamic status has stabilized, acidosis has resolved, and coagulopathy has been corrected. In general, this should be accomplished within 24 to 48 hours of the initial packing. Earlier retrieval may be appropriate if gross contamination was initially present, as this may lessen the incidence of subsequent abscess formation.[21,72] At reoperation, a thorough search for missed injury should be undertaken. Repacking is rarely required, and formal abdominal closure should be performed if visceral edema has subsided enough to allow this without tension.

DAMAGE CONTROL: SPECIFIC CONSIDERATIONS

Initial Laparotomy

The incision of choice for rapid abdominal exploration in the trauma patient is midline, made in one motion from xiphoid process to pubic symphysis. In the presence of severe pelvic fracture, limiting the incision initially to above the umbilicus may be prudent. In our experience with patients with a previous midline incision, access to the abdomen alternatively can be gained via a bilateral subcostal approach,

to facilitate visualization and dissection of adhesions and bowel from the undersurface of the midline scar.

The initial maneuvers on entering the abdomen should be rapid but orderly. Blood and clot are quickly removed. Wide lateral retraction of the abdominal wall is performed to enable four-quadrant multiple laparotomy pad packing.

This initial four-quadrant packing allows gathering of information about the sites of bleeding, most commonly from retroperitoneal vascular structures in penetrating injury, or liver in blunt trauma. Massive pelvic bleeding is less common. At this point a large mechanical abdominal retractor is placed to provide maximal abdominal visualization and to free all hands on the surgical team. If bleeding seems controlled with the four-quadrant packing or if, during initial exploration, an injury was found and controlled with hands or clamps, this is an excellent time to allow the anesthesia team to "catch up" with volume, blood, and component replacement. If no compelling source of bleeding has been found, then a retroperitoneal vascular injury is a likely source, and retroperitoneal hematoma should be sought by more careful visual inspection. The small bowel is eviscerated and the retroperitoneal viscera are mobilized to bring the aorta, inferior vena cava, and their branches or tributaries into the surgical field. This is especially important in penetrating injury.

Further exploration follows pack removal, beginning most remotely from the suspected sites of injury, thus improving exposure and maximizing working room.[19] Control of hemorrhage is the primary goal, and is accomplished by repair or ligation of vessels.[73] Critical vessels, such as the superior mesenteric artery, renal artery, or common iliac artery, can be shunted for temporary restoration of flow.[74] Shunts can be simple sterile plastic tubes or specialized intraluminal vascular shunts, placed between heavy arterial ligatures. Balloon catheter tamponade of vascular and solid viscus injuries has been described, but in our experience is less applicable.[75–77] Temporary aortic occlusion may be necessary for completion of hemorrhagic control and temporary revascularization.[78] Thoracotomy for this purpose is highly morbid; therefore, most surgeons prefer high abdominal or diaphragmatic aortic occlusion or tamponade as a temporary measure to increase supradiaphragmatic blood flow.

Hollow viscus injury must be controlled so as to limit spillage, and may be accomplished nearly simultaneously, with clamps, staples, suturing, or resection without anastomosis. Occasionally, simply wrapping the injured bowel with pads or towels is adequate to contain gross spillage.

After temporary control of hemorrhage and contamination is obtained, the decision to proceed with definitive repair must be made in concert with the resuscitating anesthesia team. In the face of hypothermia, acidosis, prolonged shock, or coagulopathy, the procedure should be truncated and the patient taken to the intensive care unit for further resuscitation. The presence and status of extra-abdominal injuries must be taken into consideration when estimating the patient's physiologic reserve; the more injured patient with more severe pathophysiology *paradoxically* requires less to be done at this initial stage.

Laparotomy pads are the best available material for damage control packing, and should be placed to emphasize three principles: pressure stops bleeding, pressure vectors should recreate tissue planes, and tissue viability should be preserved. Dry fibrin sealants have promise as hemostatic adjuncts.[79] Some authors have described the use of plastic sheeting or omentum as an intervening layer between viscera and packing material to facilitate pack removal at reexploration, but we have not found these to be necessary.[19,61]

Abdominal closure of the packed abdomen is best accomplished by rapid skin closure only; leaving the fascia open limits the risk of abdominal compartment syndrome and preserves the fascial edges and length. The massive visceral edema associated with fluid resuscitation and reperfusion in these patients may make even skin closure impossible.[64,65] Use of a Silastic (Dow Corning, Midland, MI) sheet or sterilized 3-liter intravenous fluid bag sewn to skin has been described as a temporary prosthetic in this instance.[10,22,69,80] Some have described sewing to the fascial edges but, again, to preserve the fascia for later closure we use only the full-thickness skin. Our preference is never to touch or use the midline fascia for temporary closure. Preferentially, we use a modified vacuum-container sandwich approach by placing an Ioban-wrapped surgical towel (3M, St. Paul, MN) over the top of the abdominal contents with its edges tucked under the fascia.[81] Closed suction drains (Jackson-Pratt, Zimmer, Dover, OH) are placed above the towel and brought through the subcutaneous tissue and skin at the superior (cranial) margin of the wound. An Ioban sheet is placed over the towel and drains and secured to the surrounding skin of the chest, flanks, and suprapubic area, while the lateral abdominal walls are gently pushed toward the midline. The application of suction to the drains collects abdominal fluid percolating to the surface and applies a vacuum effect to the surface dressing, optimizing the seal. While dehiscence has been described as a problem with the damage control approach, we have had no such occurrence in our own institution using this approach.[19,65]

Reoperation

The goals of reoperation are definitive organ repair and complete fascial closure. The operation should be undertaken when the patient is completely resuscitated, with correction of hypothermia, acidosis, and coagulopathy. Early reoperation may be necessary if there is evidence of ongoing hemorrhage (more than 10 units of packed red blood cells in the early postoperative period), uncorrectable acidosis, or evolving abdominal compartment syndrome.[82]

Persistent massive visceral edema may limit abdominal closure at reoperation, but successful fascial closure has been described in over 85% of cases.[9] Restoration of enteric continuity is essential at reoperation, and enteral access tube

placement should be considered. While the safe use of transabdominal feeding tubes in an open abdomen has been described, this is not our preference. We have avoided transabdominal feeding tubes or colostomy in the patient whose abdomen cannot be closed because of our experience that any violation of the abdominal wall may complicate subsequent efforts at fascial closure or promote enteric fistulae. Creation of enteral stomas is limited to those cases in which there is no other reasonable option for restoration of continuity. We prefer to provide full nutritional support initially with total parenteral nutrition, and reserve enteral support until a nasoduodenal tube can be successfully placed in the completely resuscitated patient. Reapplication of a damage control dressing may be appropriate for 48 to 72 hours to allow for diuresis of edema fluid and subsequent repeat attempt at closure. If at this point the fascia cannot be closed but the skin can, a large ventral hernia is accepted with planned repair 9 to 12 months later. If skin and fascia cannot be closed, a staged abdominal wall reconstruction is performed, beginning with the tension-free application of an absorbable mesh fascial closure. Two to 3 weeks later there is sufficient granulation for application of a split-thickness skin graft. Over 6 to 12 months this skin graft separates from the underlying viscera and fascial closure can be accomplished, sometimes requiring local muscle flap rotation.[72,83]

Complications

Given the physiologic condition of the patient who requires the damage control approach, it is not surprising that the rate of complications and mortality is high.[5,8,9,10,11,13,21,22,27,60–62,72,80,82,84–86] Morbidity includes wound infection (5–100%), intra-abdominal abscess (0–83%), dehiscence (9–25%), bile leak (8–33%), enterocutaneous fistula (2–25%), and abdominal compartment syndrome (2–25%). Multisystem organ failure is described in 20% to 33% of patients, contributing significantly to the mortality rate of 12% to 67%.

Liver

In patients undergoing laparotomy for hepatic injury, approximately 30% of deaths have been ascribed to hemorrhage.[60] Techniques to control liver bleeding include direct ligation of bleeding vessels, hepatorrhaphy, cauterization, topical hemostatic agents, partial resection, and hepatic artery ligation. Catheter balloon tamponade[76,87] and angiographic embolization have also been described.[21,88,89]

Abdominal packing or tamponade is an adjunctive procedure to control refractory hemorrhage. The need for this approach has been reported in 4% to 5% of liver injuries.[61,90] To be successful, the packs must provide optimal hemostatic pressure and stop hemorrhage; large, uncontrolled intrahepatic arteries that are bleeding are not amenable to control by tamponade.

Spleen

The spleen is commonly injured in abdominal trauma and may present as a source of life-threatening exsanguination. The patient in extremis with splenic injury requires immediate splenectomy, and attempts at splenorrhaphy are ill-advised, unnecessary, and inappropriate.[19] Occasionally, the splenic fossa needs temporary packing to allow tamponade of small vessels until coagulopathy is reversed. Later, at reoperation, the packs are removed, and drains are placed at the surgeon's discretion.

Duodenum and Pancreas

Penetrating injuries to the duodenum and pancreas are often associated with major vascular injury in the patient in extremis. Immediate mortality is related to hemorrhage. The duodenum should be mobilized using the Kocher maneuver and areas of injury oversewn. The area should then be widely drained and packed. Definitive repair is undertaken at relaparotomy, and may include pyloric exclusion, tube duodenostomy, or other, more extensive procedures.[19,91]

Urologic Injuries

Severe renal injury in the exsanguinating patient is best dealt with by rapid nephrectomy if a contralateral normal kidney is palpable. A stable retroperitoneal hematoma in the face of other severe injuries should be packed rather than explored, to avoid more bleeding and certain nephrectomy. An expanding hematoma, however, suggests ongoing hemorrhage from an uncontrolled retroperitoneal vessel, and mandates exploration for direct control. In some cases, postoperative renal artery embolization for hemorrhage (ongoing or delayed presentation) may be more appropriate and safer than operative intervention.[92] Ureteral injuries are amenable to delayed reconstruction in the unstable patient both because ureteral bleeding is minimal and because temporary control of urinary extravasation can be accomplished. Temporary urinary diversion with externalized ureteral stents can be easily accomplished.[92,93]

Bladder injury in the unstable patient can also be temporarily managed with catheter drainage using a transurethral or suprapubic catheter. Definitive bladder repair can be accomplished at subsequent operation.[92]

Pelvic and Extremity Fractures

Few data are available to guide the management of the severely injured patient with devastating pelvic injury. External fixation can restore bony stability of the pelvis in open book pattern fractures, and effectively limits venous bleeding. Associated arterial bleeding is best dealt with by angiographic embolization. When emergent laparotomy extends into the field of expanding retroperitoneal hematoma, the best course of action is to pack the pelvis with unmarked gauze to achieve tamponade, abort the laparotomy, and proceed directly to angiography for embolization to control

Table 3 Abdominal Vessel Ligation and Expected Complications

Vessel	Complication	Recommendations
Celiac axis	None	
Splenic artery	None if short gastric vessels intact	
Common hepatic artery	None if portal vein intact; possible gallbladder ischemia	Cholecystectomy (may be done at second-look)
Superior mesenteric artery	Bowel ischemia	Second-look procedure
Superior mesenteric vein	Bowel ischemia	Second-look procedure
Portal vein	Bowel ischemia	Second-look procedure
Suprarenal inferior vena cava	Possible renal failure	Wrap and elevate legs; assess for compartment syndrome
Infrarenal inferior vena cava	Lower extremity edema	Wrap and elevate legs; assess for compartment syndrome
Left renal vein (proximal)	None	
Right renal vein	Renal ischemia	Nephrectomy
Common and external iliac artery	Lower extremity ischemia	Ipsilateral calf fasciotomies or extra-anatomic bypass
Common and external iliac vein	Lower extremity edema	Wrap and elevate legs
Internal iliac vein	None	

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hemorrhage.[94] In our experience, these cases are rare and highly mortal. At times, fascial closure has been necessary to limit the decompression of hematoma and assist with pelvic tamponade.

External fixation and temporary soft tissue coverage at open fracture sites supplants more conventional approaches to fractures in the patient in extremis. Distal perfusion in the extremity can be accomplished with temporary intraluminal shunting if arterial injury is present. Simple arterial ligation should be considered as an option, and may be lifesaving.[1,95] Fasciotomy should be performed liberally in the setting of ischemia. In the mangled extremity, very little time and blood should be spent debating the limb salvageability. Formal amputation gives way to temporary wound closure and reoperation after achieving physiologic stability.

Thoracic Injuries

Packing the thoracic cavity when severely injured has unique physiologic consequences, and is limited to the apices and cardiophrenic angles. The goal of abbreviated thoracotomy is to stop bleeding and restore a survivable physiologic state. Stapled, nonanatomic wedge resections of the lung can often rapidly achieve hemostasis and control of air leaks.[15]

Pulmonary tractotomy may be an effective way to control hemorrhage in penetrating lung injury.[96] The lung bridging the wound tract is opened between long clamps or with a linear stapler, allowing direct inspection, selective vessel ligation, and control of air leaks. This obviates the need for formal lung resection.

Tracheal injury can be treated with airway control at the site of injury. For proximal bronchial injuries, single-lung ventilation may be helpful, but can be quite difficult to achieve with active hemorrhage. Endotracheal tubes with an integrated balloon can be used. Extensive bronchial repairs are not feasible in the patient in extremis, and rapid resection of the affected lobe or lung is preferable. Esophageal injury is best treated by diversion and wide drainage.[15]

Vascular Injuries

Vascular injury poses two problems: how to control hemorrhage and still maintain flow to vital tissues. Simple repair and shunting are rapid techniques. More complex repair, such as end-to-end anastomosis and graft interposition, at times are too time consuming to use in the cold, coagulopathic, exsanguinating patient. Percutaneous vascular control using balloon tamponade through the wound site has been described.[16]

Vascular injury in the abdomen is especially troubling for the patient in extremis. Most arteries and veins can be ligated to save the patient's life (Table 3). Ligation of the aorta, vena cava, superior mesenteric artery, or common or external iliac artery, however, often precipitates significant ischemia, threatening end-organ damage or death. This maneuver should be reserved for the most desperate of situations. Immediate consideration must be given to lower extremity fasciotomies and second-look laparotomy to reassess visceral viability. An alternative to ligation may be the rapid placement of temporary arterial or venous shunts.[74,97,98]

There is little justification for losing time and blood performing limb vein reconstruction in the patient in extremis. Fasciotomy must accompany extremity arterial ligation to avoid compartment syndrome.[16]

Trauma System Applications

The damage control approach can be applied in any operating room by general surgeons. Therefore, the first maneuver of laparotomy or thoracotomy with control of bleeding and contamination, packing, and skin closure is ideal for

smaller hospitals where experience with these complex injuries may be limited, or the resources necessary for resuscitation may be unavailable. Once Part I is completed, the patient can be transferred to a regional trauma center or hospital with surgeons and intensivists experienced in the subsequent patient management and definitive repair of the injuries.

CONCLUSION

Damage control is of great value as a lifesaving maneuver in selected patients with exsanguinating trauma and intra-abdominal injuries. The technique can be applied in a variety of injury patterns, and is appropriate in patients developing acidosis, hypothermia, and coagulopathy from hypovolemia. The resuscitation is aimed at reestablishing normal physiology and correcting oxygen kinetics, coagulopathy, hypothermia, and acidosis. Signs of abdominal compartment syndrome must be responded to promptly. The relaparotomy and pack removal is the time to perform definitive repair and drainage, and to place feeding tubes. Abdominal closure with or without mesh must be done with no tension on the fascia and skin edges. On the basis of the results from several studies, up to 60% of patients survive this approach, yet the risks of intra-abdominal abscess and multisystem organ failure are high. Future study is needed to establish more firmly better criteria for utilizing this maneuver. Additional research is also needed to determine the best timing of relaparotomy, how to gauge adequacy of resuscitation, better methods of temporary wound closure, and how to limit intra-abdominal abscess formation and prevent or blunt multisystem organ insult and failure. The use of adjunctive procedures (angiography) and the extension of the damage control approach into disciplines of surgical specialty caring for concomitant injuries require further evaluation.

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