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EVIDENCE-BASED CURRENT SURGICAL PRACTICE

Lessons from Trauma Care: Abdominal Compartment Syndrome and Damage Control Laparotomy in the Patient with Gastrointestinal Disease

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Introduction

Abdominal compartment syndrome (ACS) and damage control surgery (DCS) have long been thought of as features of the trauma patient.[1–3] In the last two decades, however, the pathology of ACS and the utility of damage control techniques have been increasingly recognized in general surgery.[4–7] Indeed the physiology of the ill emergency general surgery patient often mimics that of the hemorrhaging trauma victim.[5,8] Utilizing the lessons learned from the decades of trauma research and mapping them appropriately to general surgery should help improve outcomes and standardize care. This review will examine the diagnosis and management of ACS, the indications for DCS, and management of the open abdomen.

CME questions for this article available to SSAT members at http://ssat.com/jogscme/

Learning Objectives
- Be able to identify intra-abdominal hypertension and the Abdominal Compartment Syndrome
- Describe the management of ACS
- Understand the indications for DCS techniques and use of the open abdomen in emergency general surgery patients
- Describe DCS techniques
- Describe the options for temporary abdominal closure
- Discuss methods to facilitate primary fascial closure

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Abdominal Compartment Syndrome

Intra-abdominal Hypertension

Intra-abdominal pressure (IAP) is defined as the steady-state pressure concealed within the abdominal cavity. This value is obtained in the completely supine patient at end-expiration after ensuring that abdominal muscle contractions are absent, and the transducer is zeroed at the mid-axillary line. Normal IAP ranges from 0 to 15 mmHg depending on age and body habitus. The International Congress on Intra-Abdominal Hypertension and Abdominal Compartment Syndrome defined the normal value for critically ill patients between 5 and 7 mmHg with intra-abdominal hypertension (IAH) defined as sustained or repeated measurements greater than 12 mmHg. The degree of IAH is further stratified to help guide the urgency of monitoring and therapy (Table 1).[9]

Abdominal Compartment Syndrome

Increasing IAP has effects on both the peritoneal contents and the physiology of the entire patient. Within the abdomen, the

Table 1 Stratification of intra-abdominal hypertension

IAH grades	
Grade I	IAP 12–15 mmHg
Grade II	IAP 16–20 mmHg
Grade III	IAP 21–25 mmHg
Grade IV	IAP > 25 mmHg

increase in IAP leads to reductions in microcirculatory blood flow that eventually overwhelms the compensatory mechanisms of the affected organ system leading to organ dysfunction. Ischemia to the hollow viscera injures the mucosa causing edema, increased permeability, bacterial translocation, and eventually necrosis.[10] Compression of the renal micro- and macro-vasculature leads to oliguric acute kidney injury. These renal effects can manifest at a lower IAP than other organ systems; a drop in urine output may be the first sign of IAH-related organ dysfunction.[11,12] Outside the abdomen, IAH impairs venous return to the heart and increases afterload which results in a depressed cardiac output. The rise in abdominal pressure causes cephalad displacement of the diaphragm, causing increased the work of breathing, atelectasis, and a reduction in functional residual capacity. In ventilated patients, pulmonary dysfunction presents as an in increase in airway pressures, a reduction in compliance, and impaired oxygenation.[13] Historically, ACS was defined as a sustained IAP > 20 mmHg that was associated with new organ dysfunction or failure. However, organ dysfunction can begin to occur at any IAP, making a strict numerical diagnosis of ACS difficult. Therefore, ACS is best defined as IAH causing end organ derangements. For clinical purposes, ACS should be considered in any patient with IAH and new-onset shock, lactic acidosis, respiratory failure, renal insufficiency, or intracranial hypertension regardless of the IAP.[9,14]

Risk Factors for IAH/ACS

Although ACS in the trauma patient has been recognized for decades, diagnosis of the syndrome in general surgery patients has only recently become routine.[6,9,15] ACS is classified as either primary or secondary, based on the etiology. Primary ACS derives from pathology within the abdominopelvic region. Solid organ injury; traumatic injury to bowel or abdominal vasculature; rupture of abdominal vasculature; intestinal ischemia, ileus, obstruction, or perforation; postoperative hemorrhage; and pancreatitis are examples of primary causes. Secondary ACS results from extra-abdominal pathology, such as sepsis, burns, or hemorrhage, that leads to a significant inflammatory burden or resuscitation with large volumes of blood or crystalloid; the resulting retroperitoneal edema, bowel edema, and ascites lead to IAH.[9,16] In both primary and secondary ACS, exuberant volume resuscitation appears to be central to IAH and subsequent organ dysfunction. Rates of both primary and secondary ACS increase in patients receiving significant resuscitation volume, especially when accompanied by a significant inflammatory burden.[17–19] The amount of infused volume that put an individual at risk for ACS has different reported thresholds: > 10 L crystalloid or 10 units of red cells,[20] net positive fluid balance of > 5 L in 24 h,[17] and > 250 cc/kg of resuscitation fluid.[21] Regardless of the precise milliliters of infusion, the central theme correlates zealous, albeit at times necessary, volume resuscitation with ACS.

In addition to the direct etiologies noted above, additional risk factors for IAH and ACS have been identified. These factors function to increase the volume of the peritoneal contents, increase the volume of peritoneal fluid, or decrease the abdominal compliance. For example, liver dysfunction with the associated capillary leak and resultant ascites can increase the abdominal pressure. Similarly, obesity can predispose for IAH by increasing the visceral contents as well as causing extrinsic compression on the abdomen.[9] Mechanical ventilation particularly with positive end-expiratory pressure (PEEP) > 10cmH_2O may impair venous and lymphatic drainage from the abdomen and, hence, increase the risk for IAH.[22,23] Acidosis and hypothermia are both independent risk factors for ACS, presumably due to the relative increase in the patient's fluid requirements and resultant capillary leak.[9] Given that many, if not all of these factors can be present in critically ill patients, monitoring for IAH and ACS should be standard for all critically ill or injured patients admitted to the intensive care unit (ICU) with those factors to prevent delays in diagnosis and to facilitate management.[6,9]

Management of ACS

Rapid decompression of the abdomen is required to reverse the pathophysiology of ACS.[14] For most patients, decompression is performed via a midline laparotomy incision. This allows for evacuation of peritoneal fluid or blood along with evisceration of edematous bowel. The laparostomy can then be managed with standard open abdominal techniques and closed as soon as the patient will tolerate.[14] Percutaneous alternatives to surgical decompression are possible when large amounts of peritoneal fluid are the primary etiology for or a significant component of a patient's IAH. Bedside ultrasound-guided catheter drainage appears effective for management of patients who may not tolerate operative intervention. However, success of catheter drainage depended on volume removed and failure was evident within the first 4 h indicating that the surgeon must remain ready to transition to surgical management.[14,24,25]

Of note, there is a subset of patients who, despite an open abdomen and temporary abdominal closure, will continue to manifest IAH and possibly recurrent ACS.[8,26,27] This may be from a technical error in closure, for example, not allowing

enough redundancy in the vacuum dressing for ongoing edema and capillary leak. It is therefore important to continue to monitor IAP, especially in the face of ongoing organ failure.[16,26,27]

Outcomes

Despite ongoing advances in critical care, the morbidity and mortality of ACS remains significant. De Waele et al. reported a mortality rate of 36% at 28 days and 55% at 1 year with early decompression for ACS.[28] In a longitudinal study evaluating the impact of an evolving evidence-based algorithm for management of ACS, Cheatham et al. reported a 22% improvement in survival to discharge, as well as improvements in primary fascial closure and overall resource utilization.[29] Early and aggressive therapy appears to offer the best chance of survival for these critically ill patients.

Damage Control Surgery

DCS was originally conceived to help manage the exsanguinating trauma patient.[30] The bloody lethal triad of hypothermia, coagulopathy, and acidosis due to progressive hemorrhage would overwhelm resuscitative efforts as the surgeon sought complete and definitive repair of all of a patient's injuries. A shift in surgical approach to control major hemorrhage and contamination with an abbreviated initial surgical intervention in the physiologically deranged patient improved survival significantly.[2] Performing only the essential interventions enables one to return the patient to the ICU for resuscitation and to correct the patient's metabolic exhaustion, coagulopathy, and hypothermia.

In its original descriptions, DCS is divided in to five phases. First, identifying a patient who will benefit from an abbreviated laparotomy based on injury pattern, pathophysiology, or metabolic derangements. Second, obtaining sufficient control of hemorrhage and contamination to allow for resuscitation. Third, physiologic restoration in the intensive care unit including correction of acidosis, coagulopathy, hypothermia, and hypovolemia. Fourth, definitive repair of injuries including restoration of gastrointestinal (GI) continuity and durable vascular repair/reconstruction. Lastly, abdominal closure with either primary fascial closure or abdominal wall reapproximation.[1] Some authors have advocated adding an additional pre-operative stage to emphasize the need for early goal-directed resuscitation. This is important for the exsanguinating trauma patient but may be even more so for the patient who is ill with intra-abdominal sepsis. Ensuring the patient has the necessary preload, inotropic support and antibiotics can make the difference between survival to the operating room (OR) and survival through OR.[7,31,32]

Indications for DCS/Open Abdomen Management

Physiological Derangement/Coagulopathy

The same physiologic derangements of hypothermia, hypovolemia, acidosis, and coagulopathy that are evident in the critically injured trauma patient are often present in the acutely ill emergency general surgery patient.[5,8,33,34] Indeed, in some patients, underlying comorbidities may aggravate the response to abdominal sepsis or ischemia. It is therefore reasonable to consider the indications for DCS of the trauma patient in the critically ill general surgery patient. Commonly quoted patient parameters at which DCS should be considered are as follows: temperature lower than 35 °C, pH less than 7.2 or base deficit greater than 15 mmol/L (6 mmol/L if older than 55 years), and clinical or laboratory evidence of coagulopathy.[1,30,35] These values indicate impending metabolic exhaustion and the decision to abbreviate a procedure should be made as early as possible so as to limit iatrogenic progression. Additionally, the surgeon should consider DCS in patients requiring large volumes of blood product or crystalloid resuscitation.

Need for Second-Look/Serial Debridement

More clinically stable patients without the marked metabolic pathophysiology noted above may still benefit from a more abbreviated intervention and resuscitation. In acute mesenteric ischemia, the initial operation is limited to revascularization and resection of only the obviously nonviable bowel followed by a planned re-exploration in 24–48 h. This allows for marginal sections of bowel to declare themselves, any further areas of ischemia to demarcate, and can permit adjunctive endovascular reperfusion techniques.[36,37] A similar approach has been explored in patients presenting with complicated diverticulitis. Patients with Hinchey III or IV diverticulitis managed with a damage control approach (i.e., resection with colon left in discontinuity followed by repeat washout with anastomosis if appropriate) appear to have higher rates of primary anastomosis, lower rates of ostomy, and no significant difference in complications or length of stay when compared to standard single operation with definitive resection, washout, and ostomy (i.e., Hartmann's). Although studies are small and not randomized, the data is certainly promising.[38–40]

Historically, planned re-laparotomy for pancreatitis to facilitate ongoing debridement and septic source control was performed.[41–43] Modern series suggest a reduction in morbidity and mortality using a percutaneous or "step-up" minimally invasive strategy.[44,45] Therefore, damage control techniques in necrotizing pancreatitis should be reserved for patients meeting standard criteria for DCS and not to facilitate ongoing debridement.

Contamination

Management of severe peritoneal contamination and intra-abdominal sepsis using DCS/OA is similarly tempting. Multiple washouts would seem to improve clearance of septic foci and hopefully ameliorate the septic response and organ failure. Authors have described their success in using such a strategy.[6,46,47] However, both meta-analyses and a RCCT utilizing planned re-laparotomy versus on-demand laparotomy found no significant differences in rates of intra-abdominal abscesses, organ failure, or mortality. Planned re-laparotomy patients did see an increased number of returns to the OR, longer ICU and hospital LOS, and increased cost of care.[48,49] Theses studies utilized primary closure, rather than vacuum assisted closure, which limits their impact on current practices. More modern temporary closure methods, including augmentation with direct peritoneal resuscitation, may expand the utility of DCS for management of sepsis and peritonitis.[6,47,50] For now, in patients without other objective indications for DCS, attempts should be made to perform a single stage procedure, as source control can generally be obtained during the initial laparotomy.

Open Abdomen Techniques

Despite the variations in indication, the techniques or surgical management in damage control are uniform. The goal is to perform reliable, rapid but temporary procedures. To arrest hemorrhage, non-essential vascular structures are ligated, while critical structures are repaired, shunted, or packed for hemostasis. To limit enteric contamination, small injuries can be temporarily closed with a whip-stitch suture. Larger gastrointestinal injuries are resected using staplers, with an effort made to limit the extent of resection as much as possible. The bowel is left in discontinuity for later definitive reconstruction. Debridement of infected or necrotic tissues should be performed to limit progression of sepsis. Closed suction drains should be placed to control potential leaks and evacuate incompletely controlled septic sources.[4,5,7]

As the indications for DCS and the open abdomen have evolved, so have the techniques to manage the laparostomy. The ideal temporary abdominal closure (TAC) is rapidly applied and removed, provides good control and quantification of fluid losses, minimizes the potential loss of domain while allowing for ongoing edema, facilitates eventual fascial closure, and is cost effective. Skin-only closure using towel clamps, suture, or staples was the first method described for closure. This technique has largely been abandoned due to the inability to manage fluid losses and the persistent risk of ACS. Silo and patch techniques like the Bogota Bag or the Wittman patch involve suturing a prosthetic material between the skin or fascial edges for temporary closure. The clear plastic utilized for the Bogota Bag allows for continuous assessment of the viscera but is ineffective at controlling fluid effluent, traumatizes the skin edges, and still possesses risk for ACS. The Wittman Patch utilizes Velcro-like material to close the fascia. The edges are then progressively pulled to midline, facilitating delayed fascial closure. The issues with this technique, fascial trauma, poor fluid management, and time, are similar to the issues with the silo techniques. Both patch and silo methods are time consuming to place or revise, and require returns to the OR should any issues arise. Contemporary vacuum assisted closure methods appear to offer the best combination of efficacy and ease-of-use. For these systems, a fenestrated plastic drape is placed over the abdominal viscera to minimize adherence to the abdominal wall and allow drainage of intra-abdominal fluid. The suction system is then layered over the visceral covering between the edges of the abdominal wall and covered with another adhesive drape that is secured to the skin surrounding the incision. Suction is applied to remove the peritoneal fluid and provide traction to the wound edges. This technique can be performed with either "homemade" or commercially available systems (Fig. 1). The vacuum systems are relatively compliant allowing for significant changes in visceral edema without causing increases in IAP.[6,51,52]

After appropriate resuscitation in the ICU, the patient should be returned to the operating room for definitive intervention. Normalization of the patient's physiologic parameters can typically be accomplished in 12–24 h.[53] Early returns to the OR are associated with improved rates of fascial closure as well as lower rates of infectious complications.[5,6,53] Durations longer than 48 h between re-explorations should be avoided. Necessary definitive vascular repairs should be performed. Necrotic or devitalized tissues should be debrided. Gastrointestinal continuity should be restored, and any necessary stomas created. GI anastomoses or repairs should be protected within the pelvis or covered with healthy loops of bowel to help minimize the risk of fistula or leak. Abdominal closure and consideration of enteral access for feeding can begin once repairs are complete.[5,6,47]

Outcomes

Native tissue fascial closure is the goal for any open abdomen. Primary fascial closure rates for all methods of TAC vary between 11 and 100%, with 50–60% as standard for most series evaluating vacuum assisted closure.[51,52,54,55] Bioprosthetic fascial grafting or skin graft coverage of bowel are sometimes required.[51,52] However, a methodical vacuum-assisted sequential fascial closure appears to all but eliminate the need for planned hernia.[55–57] Component separation and tissue flap mobilization should generally be avoided in the acute setting due to increased risk of wound complications, and increased difficulty with later abdominal wall reconstruction.[5,47,53]

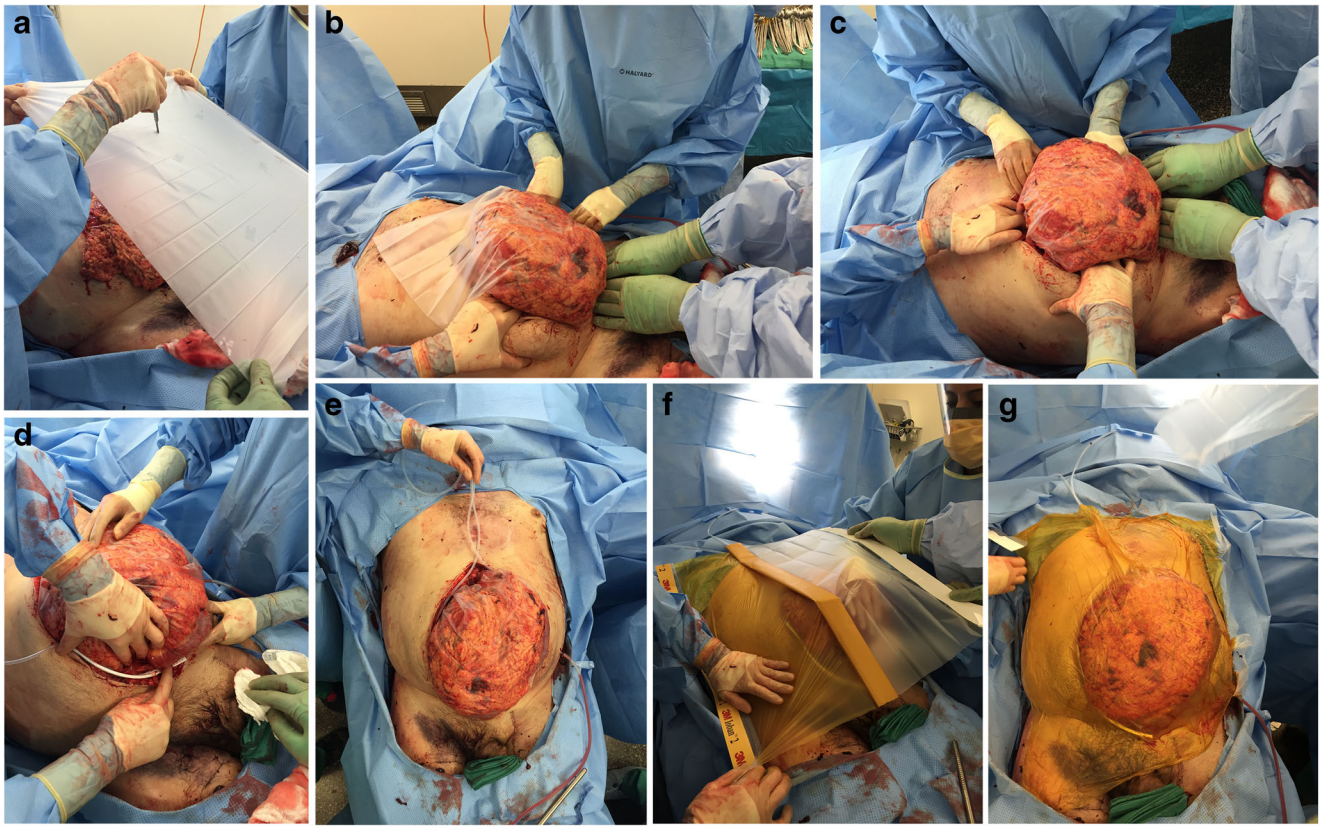

Fig. 1 The "1010 drape and ioban closure" of the abdomen. A 1010 steridrape is fenestrated using a 15 blade to make small apertures (**a**). The plastic drape is placed over the abdominal viscera and under the adjacent abdominal wall (**b**). To remove intra-abdominal fluid that accumulates, two Jackson Pratt drains are placed on either side of the viscera along the fascial edge (**c**); the tubing should run cephalad (**d**). An ioban adhesive drape is secured to the skin surrounding the incision, covering the viscera (**f**, **g**) and wall suction is applied to the drains

Although DCS addresses several of the lethal problems associated with the patient's primary pathology, the technique still has noted complications. Intra-abdominal infection and enteroatmospheric fistula (EAF) are the most morbid of these complications. Rates of recurrent or persistent infection or abscess range greatly but meta-analysis of non-trauma open abdomens found a rate of 12% when including both deep and superficial surgical site infections.[58] In general these infections can be managed similar to any SSI, with imaging-guided percutaneous drainage for the deeper collections and standard wound care for the superficial ones. This infectious rate of 12% matches the 12% rate for grossly contaminated wounds found in contemporary series.[59] EAF occurs in 12–20% of abdomens managed with DCS that remain open.[6] As with all fistulae, management of these fistula can be difficult. Isolation and adequate drainage of the fistula is important to minimize contamination and allow effective wound care. Ensuring adequate nutrition is key to resolve the fistula and heal the wound. Once the patient has recovered from the initial insult, definitive closure with either grafting for coverage or resection of the fistula can proceed.[6,51,53,60]

Patients requiring DCS have a high rate of morbidity. Mortality rates for non-trauma patients managed with DCS and OA range from 20 to 40%.[8,46,51,58] This high rate is likely due to the severity of the underlying illness. Although no randomized trials have been published evaluating the impact of DCS on non-trauma patients, a number of prospective and retrospective analyses have found significant improvements in mortality for critically ill acute care surgery patients when comparing DCS with primary closure.[33,34] Bleszynski et al., in a retrospective review of severity-matched abdominal sepsis, found a 16% decrease in in-hospital mortality when managed with DCS.[46] Girard et al. noted improvements in observed versus expected 30-day mortality between 9 and 41%, depending on scoring system utilized, for all acute care surgery patients managed with DCS.

Despite its potential benefits, if incorrectly applied, DCS may increase both morbidity and mortality. Two studies evaluating the use of DCS in the less severely injured trauma patients found significantly higher rates of wound complications, pulmonary complications, ICU and hospital length-of-stay, and mortality when compared to similar patients undergoing a single stage operation.[61,62] Presumably, similar data may be found in general surgery patients managed with DCS and suggest a regimented

application of these techniques is important to prevent patient harm.

Conclusion

Management of the abdominal compartment syndrome and appropriate use of damage control surgery are an important part of the surgeon's skillset. Advances in critical care and resuscitation mean that sicker patients can survive to the operating room. Skillful adaptation of the lessons learned in trauma can improve outcomes and minimize disability for any patient with GI disease.

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CME questions

1. Indications for damage control surgery include:

a) Temperature < 36°C

b) Blood loss > 2L

c) Arterial pH < 7.2

d) Lactate > 3

2. A 55 year old man with perforated diverticulitis is undergoing a laparotomy. Segmental resection of the sigmoid has been performed. The anesthesiologist informs you the patient is on levophed and vasopressin infusions and his ABG is 7.05/22/89/13/91%/-19. The next most appropriate step is:

a) Leave the bowel in discontinuity and close the abdomen with a temporary abdominal closure.

b) Mature an end colostomy and close the abdomen with a temporary abdominal closure.

c) Mature an end colostomy and close the fascia, leaving the skin open.

d) Perform a primary anastomosis and close the abdomen

e) Perform a primary anastomosis, mature a diverting loop ileostomy, and close the abdomen.

3. Intra-abdominal hypertension (IAP) is defined as:

a) IAP > 8

b) IAP > 12

c) IAP > 16

d) IAP > 20

4. Decompressive laparotomy should be performed for

a) Intra-abdominal hypertension

b) IAP > 20

c) Abdominal distension and shock

d) Intra-abdominal hypertension and end organ failure

5. Secondary abdominal compartment syndrome is due to

a) Bleeding from solid organ injuries

b) Postoperative hemorrhage

c) Swelling for perforated viscus

d) Resuscitation induced bowel edema and ascites

6. End organ derangements in abdominal compartment syndrome affect the

a) Renal system

b) Cardiovascular system

c) Pulmonary system

d) All

7. Specific end organ effects observed in abdominal compartment syndrome include

a) Decreased peak airway pressure

b) Increased preload

c) Decreased urine output

d) Decrease intracranial pressure

8. A reported risk factor for abdominal compartment syndrome is a net positive fluid balance of > 5L in 24 hours.

TRUE