CHAPTER 49

Abdominal Compartment Syndrome

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OBJECTIVES

This chapter will:

1. Define intraabdominal hypertension, abdominal compartment syndrome, and abdominal perfusion pressure.
2. Identify key indicators of increased risk for intraabdominal hypertension and abdominal compartment syndrome.
3. Review appropriate monitoring techniques for intra-abdominal hypertension and abdominal compartment syndrome.
4. Discuss therapeutic interventions for intraabdominal hypertension and abdominal compartment syndrome.

In 1993 Rotondo et al. introduced the term "damage control" to describe a therapeutic strategy to manage injuries accompanied by near-exsanguinating hemorrhage.[1] Along with the concept of staged laparotomy for managing these life-threatening intraabdominal injuries came recognition of the consequences of increased intraabdominal pressure. In the trauma patient, intraabdominal hypertension (IAH) principally reflected ongoing hemorrhage or solid organ and visceral wall edema. Accordingly, a plethora of techniques arose to manage the sequelae of the increased intraperitoneal pressure. These organ system–devastating consequences were termed the *abdominal compartment syndrome* (ACS).[2] It is likely that recognition of the ACS represents one of

the most important life-saving concepts introduced into trauma care in the past 3 decades.

Recently, critical care has focused aggressively on managing sepsis with a significant focus on deploying new definitions as well as mitigating inpatient readmission for infection-related morbidity.[3] The previous cornerstone of resuscitation therapy, protocol-driven early goal-directed therapy (EGDT), has been challenged by three pivotal trials demonstrating no benefit to protocolized resuscitation.[4–6] However, there remains benefit to rapidly resuscitating patients presenting with sepsis or septic shock to support systemic and microvascular flow. Protocolized or not, resuscitation from septic shock remains principally crystalloid based in sharp contradistinction to hemostatic resuscitation for those presenting in hemorrhagic shock after injury.[7,8] Reduction in crystalloid resuscitation after injury has significantly mitigated against complications of excess resuscitation, such as pulmonary edema, visceral edema, and rapid accumulation of ascites.[9] Such changes have not been realized as broadly in those with septic shock, in whom visceral edema and tense ascites still may be incited during resuscitation in patients whose initial presentation did not include intraabdominal pathology. This clinical condition has been termed *secondary abdominal compartment syndrome* and most often occurs in patients without surgical disease.[10]

Other overlaps with trauma care continue, including most notably a "damage control" approach to emergency general surgery.[11] This approach often is used in the management of general surgery catastrophes, including intestinal ischemia, gastrointestinal perforation with feculent peritonitis, and intestinal obstruction with visceral distension and wall edema. The patients requiring surgical intervention for these conditions are no different from their medical counterparts just described except that they suffer from intraabdominal pathology. IAH and ACS may develop in patients in whom the peritoneal space initially is closed before vigorous resuscitation, or in those with an open peritoneal space managed with a temporary abdominal wall closure leading to an unplanned relaparotomy. An important point is that despite the different pathophysiologic processes leading to IAH and ACS, the therapeutic goals and interventions remain identical and are universally applicable. The pathomechanisms for developing ACS and its consequences are not universally understood across different medical specialties, leading to disparate rates of recognition and therapy.[12]

INTRAABDOMINAL HYPERTENSION

A normal intraabdominal pressure (IAP) is accepted to be less than 5 to 7 mm Hg by consensus arrived at by the World Society of Abdominal Compartment Syndrome (WSACS).[13] Some variation in this baseline occurs with conditions that increase the pressure exerted on the peritoneal space and its contents, including clinically severe obesity.[14] The upper limit of nonpathologic IAP generally is accepted to be 12 mm Hg by the WSACS, and sustained increases above 12 mm Hg constitute *intraabdominal hypertension,* four grades of which are recognized, as outlined in Table 49.1. IAH may be classified further by duration of increased pressure as chronic (clinically severe obesity, chronic ambulatory peritoneal dialysis [CAPD]), acute (postoperative ongoing hemorrhage, visceral and bowel wall edema—i.e., primary ACS), subacute (ascites from massive volume resuscitation—i.e., secondary ACS), and hyperacute (ruptured acute aortic aneurysm, massive postoperative hemorrhage, resuscitation from extraabdominal injury).[13,15] Because of the individual variation in baseline pressure and the possibility that IAH may be an individually contextually sensitive measurement, a more clinically useful measure may be the abdominal perfusion pressure (APP).[16]

The APP is calculated in a fashion similar to that for intracranial pressure and represents the difference between the inflow pressure, which is the mean arterial pressure (MAP), and the pressure limiting egress, which is IAP. Thus APP = MAP – IAP. A normal abdominal perfusion pressure is defined as 60 mm Hg. Thus a MAP of 65 mm Hg minus an IAP of 10 mm Hg gives an APP of 55 mm Hg. Although an IAP of 10 mm Hg does not constitute the ACS, a low APP should engender inquiry into the adequacy of the MAP to support bulk and microvascular flow. At present, few data support titration of therapy to APP in the absence of IAH. As a practical matter, clinicians commonly use the transition from grade II to grade III IAH (IAP greater than 20 mm Hg) and certainly the presence of the ACS as the trigger for initiating therapy.

TABLE 49.1

Grading of Intraabdominal Hypertension

GRADE	INTRAABDOMINAL PRESSURE
I	12–15 mm Hg
II	16–20 mm Hg
III	21–25 mm Hg
IV	>25 mm Hg

BOX 49.1

Common Signs and Symptoms of Abdominal Compartment Syndrome Associated With Organ Dysfunction or Failure

- Hypotension
- Oliguria
- Increased peak airway pressures (volume-cycled ventilation)
- Decreased resultant tidal volumes (pressure-cycled ventilation)
- Decreased release volumes (airway pressure–release ventilation)
- Hypoxia and decreased CO_2 clearance
- Lactic acidosis
- Increased core to peripheral temperature gradient
- Disordered mentation

ABDOMINAL COMPARTMENT SYNDROME

The diagnosis of ACS is based on a sustained IAP greater than 20 mm Hg or an APP less than 60 mm Hg that occurs in association with an attributable new-onset organ dysfunction or failure.[13] The commonly identified constellation of signs and symptoms outlined in Box 49.1 represents the typical manifestations of organ dysfunction and failure that constitute the ACS. Regardless of cause, the increased IAP diminishes venous return and distorts cardiac performance. The decreased cardiac output is further embarrassed by limited diaphragmatic excursions resulting from cephalad visceral displacement. Reduced thoracic cage space increases the reflected endobronchial pressures, increasing

intrathoracic and intrapleural pressures still further, thereby further limiting venous return and decreasing the transpulmonary pressure gradient in those spontaneously respiring. In addition, pulmonary artery pressures increase, limiting right ventricular ejection fraction on the basis of a relative increase in afterload in part from compressed pulmonary parenchyma and in part as a result of hypoxic pulmonary vasoconstriction; hypoxia commonly ensues with decreased cardiac performance.[17]

As a result, cerebral oxygen delivery falls and mentation is compromised. Indeed, intractable intracranial hypertension has been related directly to and resolved by treating the ACS.[18] Other important clinical correlates include oliguria secondary to renal vein compression and diminished renal blood flow from inadequate cardiac output. At the bedside, the triad of oliguria, hypotension, and increased peak airway pressures (on volume-cycled ventilation) or resultant decreased tidal volumes (on pressure-cycled ventilation) or decreased release volumes (on airway pressure–release ventilation) should enjoin the clinician to consider a diagnosis of ACS in at-risk patients (see later and Box 49.2). An increased IAP in isolation or in combination with a decreased APP then will establish the diagnosis and should prompt urgent surgical consultation for therapy. In one multicenter, prospective study of 265 patients admitted to a critical care unit, IAH was present in 32% (85 patients); of these, 4% (11) were hospitalized with ACS, and 68% of the overall series (140) had a normal IAP.[19] IAH on admission was associated with multiple organ dysfunction and failure. Moreover, IAH during the ICU stay served as an independent outcome predictor, but mean IAP at admission failed to surface as a marker of mortality and instead identified an at-risk population for morbidity.

At-Risk Patient Populations

As noted earlier, at-risk patient populations fall into three discrete categories (Box 49.2):

1. Trauma patients who have undergone a damage control laparotomy or thoracotomy for near-exsanguinating hemorrhage
2. Medical patients suffering from septic shock who have undergone large volume crystalloid or colloid resuscitation
3. General surgery patients requiring large volume resuscitation for an intraabdominal catastrophe

Any of these patient populations may receive large volume crystalloid resuscitation directed at the intravascular space, but they also require substantial resuscitation directed at the interstitium to manage preexisting deficits.[20] Coagulopathy is not an uncommon accompaniment to massive volume loading, and trauma patients in particular may be managed with a massive transfusion protocol as well. However, patients suffering from esophageal variceal hemorrhage, spontaneous retroperitoneal hemorrhage, or massive upper or lower gastrointestinal hemorrhage (non–portal hypertension-associated) may be managed similarly. Thus IAH and ACS may develop within hours of initiating resuscitation. Some data suggest that volume resuscitation with colloids instead of crystalloids may minimize risk of IAH and ACS,[9] but the current US standard remains crystalloid infusion for plasma volume expansion; colloid resuscitation more commonly is used in the European Union, the United Kingdom, and Australia. Certain operative maneuvers or postoperative interventions also increase the risk of IAH and ACS (Box 49.3). Nonetheless, at-risk patients should be monitored routinely for development of these entities to achieve early detection and prevention or therapy as appropriate.

BOX 49.2

Risk Factors for Intraabdominal Hypertension and Abdominal Compartment Syndrome

- Massive-volume resuscitation (>10 L crystalloid or 5 L colloid over 24 hours)
- Massive-volume transfusion protocol (>10 units packed RBCs over 24 hours)
- Management with an open body cavity (chest or abdomen)
- Core hypothermia (temperature <33°C)
- Coagulopathy requiring blood component therapy (aPTT >2× normal; INR >1.5)
- Septic shock regardless of cause
- Critical illness in the setting of cirrhosis or other form of liver failure accompanied by ascites
- Mechanical ventilation
- PEEP at >10 cm H_2O pressure (extrinsic or intrinsic)
- Major thermal injury

aPTT, Activated partial thromboplastin time; *INR,* international normalized ratio; *PEEP,* positive end-expiratory pressure; *RBCs,* red blood cells.

Monitoring Techniques

The most widely used monitoring technique to assess intraperitoneal pressure is measurement of intravesical pressure (bladder pressure). This technique is safe, reproducible, and readily accomplished by the bedside nurse using simple instrumentation that is routinely available in the critical care unit. A sample protocol for bladder pressure monitoring is presented in Box 49.4. Fig. 49.1 shows a diagram of an assembled bladder pressure monitoring apparatus. Some controversy exists regarding how much volume to infuse into the empty bladder to obtain the most accurate measurement. Currently, the WSACS suggests using no more than 25 mL of sterile 0.9% NSS to eliminate coaptation of the bladder walls around the measuring catheter. Greater than 50 mL increases the risk of overestimation of IAP.[21] It is important to use a consistent volume to reduce variability in measurement among clinicians. In addition, IAP should be recorded at end expiration, with the patient supine and the transducer secured at the phlebostatic axis, to obtain the most accurate reading.[22] Ideally, significant muscular activity should be absent; a temporary increase in sedation may

BOX 49.3

Common Operative or Postoperative Interventions With Increased Risk for Intraabdominal Hypertension and Abdominal Compartment Syndrome

- Repair of giant ventral hernia with a tight abdominal closure
- Postoperative abdominal binder
- Ileus
- Peritonitis of any cause
- Preoperative deliberate pneumoperitoneum for giant ventral hernia management
- Colonic gaseous distention (colonoscopy, Ogilvie syndrome)
- Postoperative hemorrhage
- Open body cavity with or without cavitary packing for hemorrhage control

BOX 49.4

Bladder Pressure Monitoring Guideline

Purpose

Measuring bladder pressure has been demonstrated to correlate well with intraabdominal pressure (IAP).

Progressive increases in IAP have been shown to lead to the abdominal compartment syndrome (ACS). ACS results in decreased perfusion to the kidneys and the gut while decreasing cardiac output and impeding respiratory excursion. ACS confers increased mortality and morbidity risk to patients with increased IAP. Recognition of a developing ACS allows the opportunity for early intervention before onset of complications from ACS.

Patients Covered by Guideline

All SICU patients at risk for intraabdominal hypertension

Risk Identifiers for Increased IAP

- Damage control laparotomy
- Intraabdominal procedure *in conjunction with*
  - Large-volume resuscitation (>10 L crystalloid equivalent) *or*
  - Coagulopathy requiring correction with the massive transfusion protocol *or*
  - Large-volume blood component therapy (packed RBCs > 10 units *or* FFP > 8 units)
- Septic shock
- Open body cavity
- Core hypothermia
- Cirrhosis or liver failure with ascites
- Mechanical ventilation with PEEP at >10 cm H_2O pressure (intrinsic or extrinsic)
- Physician discretion

Definitions

1. Intraabdominal hypertension: IAP >12 mm Hg
2. Abdominal compartment syndrome: a clinical syndrome resulting from increased IAP manifested as increased peak airway pressure, oliguria, metabolic acidosis, decreased cardiac performance (invasively or noninvasively measured), decreased abdominal perfusion pressure, and decreased mentation. ACS commonly is associated with IAP >20 mm Hg but may occur at lower pressures, as well as with specific patient characteristics.
3. Abdominal perfusion pressure (APP): APP = mean arterial pressure (MAP) – IAPNormal APP: ≥60 mm Hg

Guideline

1. On admission to the SICU, patients will be evaluated by the bedside nurse and the physician team for risk identifiers for increased IAP.
2. Patients who are identified as at risk will be monitored by bladder pressure measurements according to the following schedule:
   a. On arrival at the SICU
   b. Every 2 hours for the first 8 hours
   c. Every 4 hours for the next 8 hours
   d. Every 8 hours for the next 24 hours
3. The critical care team will be notified of all bladder pressure measurements >12 mm Hg and abdominal perfusion pressures <60 mm Hg.
4. These values will be recorded in the ICU flowsheet.

FFP, Fresh frozen plasma; *PEEP,* positive end-expiratory pressure; *RBCs,* red blood cells; *SICU,* surgical intensive care unit.

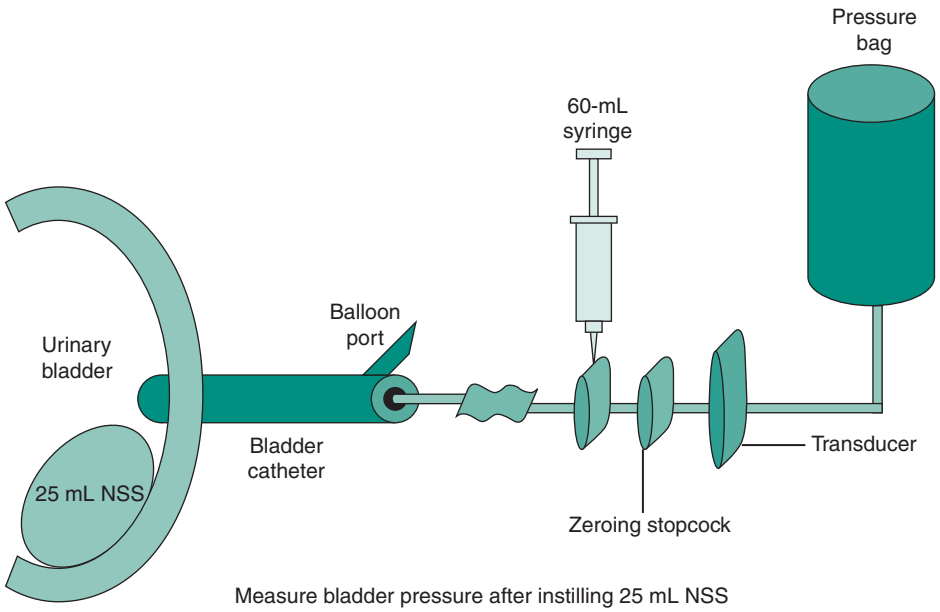

FIGURE 49.1 A standard bladder pressure monitoring set-up.

be needed to achieve optimal conditions. Neuromuscular blockade generally is not needed or recommended to obtain satisfactory measurements.

A variety of other locations and techniques have been proposed to record intraperitoneal pressure, including but not limited to the inferior vena cava and stomach and use of a continuous pressure monitor for the peritoneal space.[23,24] Of importance, none of the cited methods demonstrates the durability and ease of intravesical monitoring. Complete monitoring kits that do not require breaking the Foley catheter circuit are commercially available and may ease nursing measurement of IAP but are without durable data regarding decreases in catheter-associated urinary tract infection (CAUTI) rates.

Biomarkers

The National Institute of Health and European Medicines Agency has established categorical definitions for biomarkers to aid in marking the course of disease (type 0), reflecting the course of therapy (type 1), or serving as a surrogate endpoint (type 2).[25,26] There is no biomarker for IAH, and therefore clinicians must use surrogate markers such as elevated peak airway pressures, hypotension, and changes in urine flow. Because oliguria is one of the early signs that may be associated with IAH or ACS, early detection of renal dysfunction may be a useful marker of risk progression in the at-risk population, or may serve as a trigger for therapy depending on the clinical circumstance. Perhaps the most well studied is neutrophil gelatinase-associated lipocalin (NGAL), a 25-kD protein derived from neutrophils and covalently bound to gelatinase.[27] Plasma and urinary NGAL increase with increasing RIFLE (risk, injury, failure, loss, end-stage disease) score, and outperform serum creatinine for the detection of acute kidney injury (AKI). Importantly, multiple conditions unassociated with AKI do not result in increased plasma or urinary NGAL concentration. This marker is unique compared with others such as γ-GGT, kidney injury molecule-1 (KIM-1), cystatin C, or IL-18.

Urinary excretion depends on whether the marker is preformed or must be generated and the resultant plasma concentration, filtration rate, reabsorption rate, and the secretion rate. It is influenced by renal injury as well as the less common hyperfunctioning kidney. Filtered markers have the longest time course and preformed ones the shortest, perhaps allowing for detection failure resulting from rapid disappearance.[28] γ-GGT is preformed and with renal stress is released and rapidly cleared. KIM-1, IL-18, and NGAL are induced and upregulated with renal stress. Cystatin C and NGAL are filtered and resorbed. Unlike the aforementioned markers, only NGAL is filtered, reabsorbed, and secreted, making for more rapid increases in concentration and therefore more rapid detection. Supportingly, in a small group of living renal donors, those donating by an open approach were compared with those donating via a laparoscopic approach with an insufflation pressure of 12 mm Hg (grade 1 IAH). In the laparoscopic group, markers of inflammation such as C-reactive protein as well as NGAL were increased.[29] There are trials after cardiac surgery investigating NGAL as well as other biomarkers, but none specifically address IAH or ACS after injury or emergency general surgery nor offer a potentially important opportunity for investigation.[30,31]

Therapeutic Interventions

The standard of care for IAH leading to the ACS is decompressive laparotomy, which follows the same principles as those for managing an extremity compartment syndrome or a thoracic compartment syndrome.[32] Although reoperative therapy has been accepted widely in the trauma community, some reticence remains in the medical community to readily embrace surgical therapy for patients without abdominal disease. Similarly, the nontrauma surgical community less readily welcomes relaparotomy and open abdominal management than do their trauma counterparts. Instead, a variety of nonsurgical remedies have been explored as surgical alternatives. None of the alternatives has been subjected to prospective, randomized controlled trial analysis to substantiate its efficacy compared with the gold standard of decompressive laparotomy.

Nonsurgical Interventions

Proposed alternative modalities include neuromuscular blockade, percutaneous catheter fluid decompression (ascites management only), gastrointestinal content reduction, volume reduction strategies (diuretic therapy, continuous renal replacement therapy), and laparoscopic fasciotomy or component separation of parts (to eliminate or reduce fascial constraints while preserving skin integrity).[33] Although attractive, percutaneous drainage may not be well applied to compartment syndrome from blood because hemorrhage control is required and is not addressed by a drainage catheter. Moreover, catheter-based drainage is subject to catheter dysfunction from kinking, malposition, or obstruction, which may allow recurrent IAH and ACS at a time when the clinical picture is falsely benign owing to decreased drainage from catheter dysfunction. Neuromuscular blockade is thought to reduce the measured pressure by eliminating muscular resistance of the abdominal and chest walls. Although use of this strategy may result in reduction in the measured value, in clinical practice, little physiologic benefit is realized for those with the ACS, because the mean IAP change rarely exceeds 5 mm Hg; therefore this technique may be most useful in managing those with IAH before an established ACS.

Reduction in luminal gastrointestinal contents may be accomplished by means of nasogastric suctioning, rectal lavage, or prokinetic therapy. These maneuvers are useful in the initial management of small bowel obstruction, colonic distention after colonoscopy, and Ogilvie syndrome but have little to no therapeutic effectiveness with other causes of IAH or ACS. Volume reduction strategies may be intuitively attractive but must be applied after the patient's resuscitation has been completed. Attempts at volume reduction earlier than that may be fraught with hypoperfusion, unintentionally reestablishing the pathophysiologic process abrogated by the initial volume reduction therapy. Because many affected patients suffer from organ dysfunction, commonly acute kidney injury with oliguria despite appropriate effective circulating volume restoration, renal replacement therapy, especially ultrafiltration, may provide the sole means of volume reduction. Several retrospective reviews have investigated the success of this modality to repair IAH, with reports of limited success at best. Therefore this therapy cannot be recommended as end therapy for IAH or ACS but may be a useful adjunct, especially since IAP closely correlated with extracellular water.[34]

Surgical Interventions

Minimally invasive approaches are successful in managing a host of clinical conditions spanning cardiac bypass to organ procurement to appendicitis. Their application to ACS appears to be reasonably logical for secondary ACS. Clearly, these approaches are not appropriate for the trauma patient requiring damage control laparotomy, nor for relaparotomy in that patient population, because an intact anterior abdominal wall is absent. The general surgery patient in whom IAH or ACS develops in the postoperative period, as well as the medical patient with extraabdominal disease, may be served by these techniques. The most promising of these appears to be use of a laparoscopic but subcutaneous approach to performing multiple fascial releases without violating the peritoneal space; this approach has not been adopted widely.[35,36] An important point is that the driving forces that generate ascites will not abate with decompression of the ascites. Moreover, any passage that connects the peritoneal

space with the skin will provide a ready conduit for flow of ascitic fluid. Thus maintaining an intact peritoneum or anterior abdominal wall (albeit an expanded one) will reduce the risk of uncontrolled volume loss and inoculation of ascitic fluid, leading to infection and peritonitis.

Decompressive laparotomy simply enlarges the available space for the solid organs and viscera while evacuating fluid, blood, or clot from the peritoneal space. The peritoneal envelope is not reestablished in any way other than with temporary abdominal wall closure. In one animal model, IAH and the ACS have been demonstrated to create venous hypertension and to diminish mesenteric lymph flow as a mechanism of gut edema formation. Thus early decompression provides one means of limiting further hollow viscus wall edema.[37] The temporary closure uses an impermeable barrier that covers a suction system positioned over some protective cover placed on top of the intestines (Figs. 49.2 and 49.3). A proprietary system, the vacuum-assisted closure (VAC) device (Kinetic Concepts Incorporated, Langford Locks, UK), is available commercially, including one with deep extensions to help limit adhesions between viscera and the posterior aspect of the anterior abdominal wall (Ab-Thera device), in addition to "home-grown" versions using Jackson-Pratt drains and wall suction. The value of these systems is that fluid losses may be quantified, heat and evaporative losses minimized, and patient and bed soilage from fluid drainage controlled, leading to improved skin integrity. These devices are changed every 48 to 72 hours (or earlier as needed); peritoneal lavage and debridement, as appropriate, are common supportive measures. The procedures may be performed at the bedside or in the operating room with equal safety and efficacy and may be driven by lack of OR availability, hemodynamic instability, or advanced ventilation needs.[38]

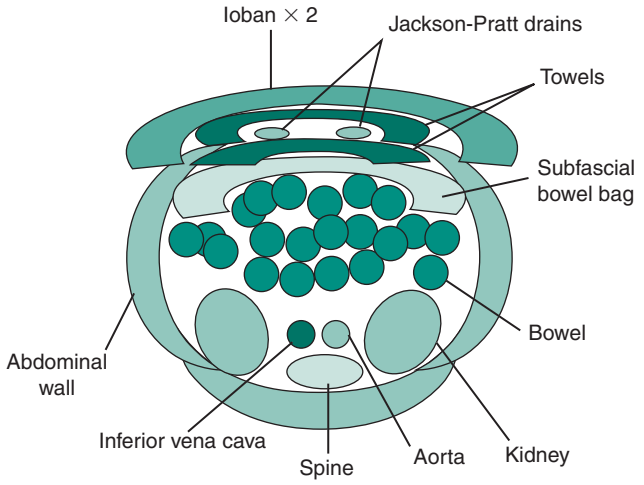

FIGURE 49.2 One method of securing a temporary abdominal wall closure using an impermeable bowel bag, sterile towels, two no. 10 flat Jackson-Pratt drains, and two sheets of Ioban secured to the skin.

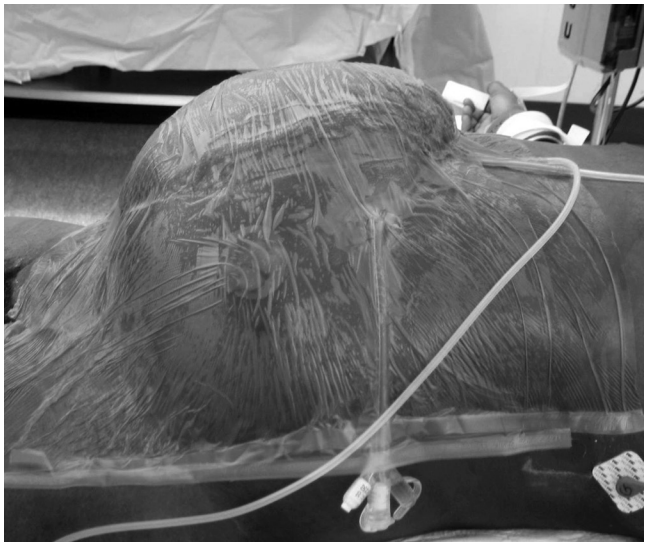

FIGURE 49.3 A patient with a temporary abdominal wall closure.

MITIGATION STRATEGIES

Several authors have investigated interventions to reduce the likelihood of developing IAH and ACS after resuscitation. Each of the strategies focuses on volume limitation in some protocol-driven fashion. After injury, multiple investigators have demonstrated reductions in IAH and ACS by reducing or elimination crystalloid resuscitation in favor of a blood component therapy–driven hemostatic approach. Others have evaluated creating an ED or pre-ICU "cap" for fluid resuscitation. This approach in particular seems appropriate for emergency general surgery and nonsurgical patients. By stopping the infusion of fluid after 2L in the ED and 4L before ICU admission, the authors were able to achieve a 75% reduction in the need for an open abdomen approach, and no change in mortality, multisystem organ failure, or ICU length of stay; the mean reduction in crystalloid volume was 800 mL.[39] Moreover, in their 81 consecutive patients, the authors were able to shift their IAH distribution to grade 1 (47) and 2 (20) as opposed to 3 (5) and 4 (2); no IAH was detected in 26. Recall that the abdominal wall is relatively elastic but does have a threshold past, in which further small increases in volume translate into much larger increases in pressure, akin to the physiology known for cardiac tamponade.[40] Abdominal wall compliance and elastance are related by the following formula:

Elastance = 1/abdominal compliance where compliance = ΔIAVolume/ΔIAPressure

Therefore small changes in fluid intake that lead to excessive increases in pressure significantly decrease elastance. In the aforementioned study the mean difference in volume was less than 1 L, suggesting that prescriptive control of fluid administration may have a vast impact on outcomes.

FUTURE DIRECTIONS

Relatively unexplored in the context of IAH or the ACS is the impact on the microbiome. Microbiome alteration has been well explored in the setting of *Clostridium difficile* colitis, anastomotic failure, and a host of other disease processes, including clinically severe obesity and autoimmune gastrointestinal illness. A recent human study explored the impact of anastomotic injury on specific intestinal and perianastomotic tissue microbiota. This study found that substantial increases in specific bacteria were coupled with a predominant profile of virulence-associated pathways, the

majority of which appeared focused on tissue degradation.[41] It remains unclear whether increases in IAH trigger changes in microbiome diversity of function, or whether reductions promote salutary effects on an existing microbiome with regard to diversity of function. However, it has been elucidated that acidifying the microenvironment of an electrosynthetic microbiome (one that generates complex carbon-carbon molecules using the intake of CO_2 and energy derived from electricity) results in an increase in proton generation, a potentially maladaptive response.[42] Increases in IAP are associated with lactic acidosis and are perhaps analogous to changes in the experimental microenvironment detailed above. Similarly, because the gut lumen is rich in flora, the impact of reduced perfusion and its impact on neutrophil trafficking and microbial trafficking are implicated in bacteremia and surgical site infection as well as the outcome of sepsis.[43] In fact, bacteria such as *Salmonella* species may "hide" within neutrophils and escape destruction via specific virulence mechanisms.[44,45] Therefore the microbiome offers a fruitful area of investigation as it interacts with changes in intraabdominal pressure and perfusion.

Key Points

1. Routine screening and monitoring for intra-abdominal hypertension and abdominal compartment syndrome are indicated in patients at risk for the development of these conditions.
2. A standardized monitoring protocol is used to ensure accuracy and reproducibility.
3. Abdominal compartment syndrome may occur in a given patient at a lower intraabdominal pressure than that associated with clinical characteristics of the syndrome in another.
4. Intraabdominal hypertension and abdominal compartment syndrome may develop in patients without intraabdominal disease.
5. Organ salvage depends on prompt abrogation of abdominal compartment syndrome, so early surgical consultation and aggressive surgical therapy are imperative once the syndrome is established.
6. Decompressive laparotomy remains the gold standard for treatment of intraabdominal hypertension and abdominal compartment syndrome.

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