4 The Open Abdomen: Balancing Pathophysiologic Benefits and Risks in the Era of Improved Resuscitation Practices

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Key Points

- Physiological derangements after damage control (DC) laparotomy in trauma, general, and vascular surgery patients are largely related to the lethal triad and perfusion/systemic inflammatory disturbances induced by acute bowel injury and intra-abdominal hypertension (IAH) (i.e., the "acute intestinal distress syndrome").
- Both acute bowel injury and IAH are made worse by large-volume crystalloid fluid resuscitation.
- There are several proposed physiologic benefits of the open abdomen (OA). These include that it prevents the onset of (and/or interrupts) the lethal triad and ACS in patients who received conventional trauma resuscitation.
- The survival benefit of DC laparotomy has recently been questioned because of the introduction of improved resuscitation practices (such as DC

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F. Coccolini et al. (eds.), *Open Abdomen,* Hot Topics in Acute Care Surgery and Trauma, https://doi.org/10.1007/978-3-319-48072-5_4

resuscitation and the avoidance of large-volume crystalloid fluid resuscitation) and the risks associated with open abdominal management, including progressive abdominal visceral edema, loss of abdominal domain, massive ventral hernias, enteric leaks, and enteroatmospheric fistulae.
- As equipoise begins to dwindle regarding the effectiveness and safety of liberal use of damage control laparotomy in the modern era of improved resuscitation practices, randomized evidence is increasingly required to elucidate the situations in which the associated benefits of open abdominal management outweigh the risks.

4.1 Introduction

Classically, there are three broad categories of reasons why surgeons leave the abdominal cavity open: anatomical, physiological, and logistical [1]. The specific indications underlying these categories were recently systematically reviewed and then "appropriateness rated" in two international expert consensus studies and a larger survey of surgeons practicing in the USA, Canada, Australia, and New Zealand [2–5]. Appropriate anatomical indications identified in these studies included several abdominal injury patterns and an inability to close the abdominal fascia because of visceral edema [2–5]. Appropriate physiological reasons included the finding of hypothermia, acidosis, and/or coagulopathy or development of signs of intra-abdominal hypertension (IAH) and/or abdominal compartment syndrome (ACS) during attempted abdominal fascial closure [2–5]. Finally, appropriate logistical reasons involved a planned relaparotomy to remove packs, reassess bowel viability (e.g., superior mesenteric artery/vein injuries), or reestablish gastrointestinal (GI) continuity [2–5].

Open abdominal management has long been reported to be associated with a number of physiological benefits among critically ill trauma, general, and vascular surgery patients [6]. In critically injured and massively bleeding patients, one of the conventional grounding principles underlying damage control (DC) surgery is that abbreviating the procedure by conducting "rapid conservative operative techniques" and leaving the abdomen open may lead to improved patient outcomes [7]. Abbreviated surgery theoretically helps prevent further bloodshed and avoids the onset of (and/or interrupts) the "vicious cycle" of hypothermia, acidosis, and coagulopathy [3, 8, 9]. Largely through this mechanism, it has been postulated since the late 1980s that DC surgery may prevent death from "physiological exhaustion" [3, 8, 9]. Thus, use of the open abdomen (OA) (defined as "non-closure of the abdominal fascia") is a necessary component of trauma DC laparotomy [10]. Further, among those with or at risk of significant abdominal visceral edema and/or IAH (e.g., typically patients who have received a large volume of intravenous crystalloid fluid resuscitation), leaving the abdomen open after operation may prevent ACS [10, 11].

Decompressive laparotomy and open abdominal management are also the standard treatment for ACS [10–12]. Finally, recent evidence suggests that use of temporary abdominal closure (TAC) techniques that employ constant negative pressure to the open abdomen (active negative pressure peritoneal therapy [13]) may reduce peritoneal inflammation and the systemic inflammatory response syndrome (SIRS) induced by acute bowel injury and thereby prevent multiorgan dysfunction syndrome (MODS) [13, 14].

However, open abdominal management has also been associated with a high rate of energy loss and a substantial incidence of potentially severe complications (intra-abdominal sepsis, enteric leaks, enteroatmospheric fistula formation, and massive ventral hernias) [3, 15–17]. The management of these complications often requires a number of hospital readmissions and subsequent surgical procedures [3, 15–17]. Further, some have questioned whether the physiologic benefits of DC surgery will continue to be realized in the modern era of improved resuscitation practices where large-volume crystalloid fluid resuscitation is avoided [7, 18]. Thus, decisions regarding whether open abdominal management is indicated or not should be based on modern evidence or until such evidence becomes available and the opinions of experts and the currently practicing surgical community [2–5]. In this chapter, we review the pathophysiology of the OA, including the balance between its potential pathophysiologic benefits and risks. Specifically, we describe the pathogenesis of the lethal triad, acute bowel injury, abdominal visceral edema formation, IAH/ACS, SIRS, and MODS relevant to patients with an OA. Within the context of these adverse pathophysiologic changes, we also review the proposed physiologic benefits of the OA. We end with a description of the present knowledge regarding the pathophysiology of progressive loss of abdominal domain, massive ventral hernias, enteric leaks, enteroatmospheric fistulae formation, and accelerated energy expenditure in this patient population.

4.2 The Changing Playing Field

Discussion of the use of the OA in trauma, general, and vascular surgery needs to be considered within the context of recent changes in resuscitation that have likely altered the "playing field" concerning hemorrhage resuscitation. Historically, use of the OA was considered essential to avoid early death in patients with dramatic abdominal visceral edema who underwent delayed hemorrhage control after receiving a large volume of crystalloid fluid resuscitation [7, 19–22]. With fundamental changes in resuscitation practices that focus on avoidance of crystalloid fluids and use of DC resuscitation principles, there have been reports of a dramatic reduction in the extent of abdominal visceral edema, severe IAH, and ACS after emergent laparotomy [7, 23]. This changing IAH/ACS epidemiology has been coupled with reports of concerns that DC surgery and open abdominal management may be overused in the era of DC resuscitation [7]. Thus, principles that were once deemed dogma, such as the importance of judging whether a seriously injured patient would

receive an OA even before beginning an operation, are less relevant or even irrelevant in modern surgical practices [7, 24]. However, as experts and practicing surgeons have reported that there are likely still benefits of DCS in select clinical circumstances, its use should continue for these indications until randomized evidence becomes available [2–5].

4.3 The OA for Trauma Versus the OA for Sepsis

Intensive care unit (ICU) patients with an OA typically constitute a mix of injured and infected patients. Previous studies addressing management issues related to the OA have typically combined these cohorts. However, it is becoming increasingly apparent that injury and infection are significantly different pathophysiologic insults [25]. Those with infection may be disproportionately affected by persistent inflammation and its associated obligatory edema [25]. In support of this, in a recent study by Loftus et al., failure to achieve primary fascial closure (PFC) after trauma was associated with persistence of the lethal triad beyond 48 h post-injury, whereas failure to achieve PFC after infection was most related to the operative course within 48 h of the index laparotomy [25].

4.4 Pathogenesis of the Lethal Triad, Abdominal Visceral Edema, IAH/ACS, SIRS, and MODS

4.4.1 The Lethal Triad

As the original use of the OA was largely driven by the need to address the significant fatality associated with physiological exhaustion [7], any discussion of the topic requires an understanding of the lethal triad. Exsanguination, or a blood loss exceeding 40% of total body blood volume with ongoing bleeding, is often associated with development of a lethal triad (or "vicious cycle") of hypothermia (core body temperature < 34 °C), acidemia (pH < 7.2), and clinical (absence of visible clots during operation) or laboratory [e.g., international normalized ratio and/or partial thromboplastin time > 1.5 times normal] coagulopathy [2, 3, 7, 26–28]. Importantly, all three components of the lethal triad may be precipitated or exacerbated by the administration of large volumes of crystalloid fluids.

Historically, hypothermia occurred in 57–66% of severely injured patients treated with standard, crystalloid-based resuscitation practices [29, 30]. This complication typically occurred after profound shock had been resuscitated with infusion of unheated crystalloids and blood products [31]. Its occurrence was associated with a 4–41 times higher risk of intraoperative mortality [26, 29, 30, 32]. The incidence of hypothermia increases with higher injury severity scores and worsening degrees of shock. The association between hypothermia and mortality has been

reported to be stronger among those with core temperatures <33°C or who were difficult to rewarm than those who remained warm or could be timely rewarmed [29, 32]. Although it would be predicted that the incidence of hypothermia would decline as resuscitation practices abandon infusion of large volumes of crystalloid fluids, even more recent series have reported development of severe hypothermia in injured patients [33].

Metabolic acidosis often coexists with hypothermia and coagulopathy and results predominantly from anaerobic metabolism and production of lactate secondary to inadequate peripheral tissue perfusion [26, 34]. Coagulopathy was originally thought to be caused by hypothermia, metabolic acidosis, administration of a large volume of crystalloid fluids, and other factors [26, 35, 36]. However, in 2003, Brohi et al. reported that 33% of 1008 severely injured patients presenting to hospital already had prolonged clotting times that were independent of the amount of crystalloid fluids administered [7, 37]. This "acute traumatic coagulopathy" (also sometimes referred to as the "acute coagulopathy of trauma") appears to occur "before significant fluid administration, that may be attributable to the injury itself" [37]. As hypothermia and metabolic acidosis worsen coagulopathy, and ongoing bleeding secondary to coagulopathy worsens hypothermia and metabolic acidosis, the initiation of the vicious cycle often leads to progressive, diffuse, difficult to control hemorrhage and ultimately death if actions are not taken to prevent or interrupt it [3, 8, 9].

4.4.2 Abdominal Visceral Edema, IAH/ACS, SIRS, and MODS

Surgeons have long hypothesized that inflammation and abdominal visceral edema were linked, especially among patients receiving traditional (crystalloid-based) resuscitation [7]. In trauma patients receiving large-volume fluid resuscitation, the abdominal viscera can sequester liters of crystalloid fluid and has been reported to grow to more than twice the volume of the abdominal cavity after emergent trauma laparotomy [38–40]. With aggressive crystalloid resuscitation, IAH/ACS may be induced early, especially if hemorrhage is not expeditiously controlled and if hyperresuscitative strategies are used [19–21]. Thus, it is possible that many (if not most) cases of early ACS were closely linked to crystalloid-focused resuscitation strategies in the past [41, 42]. However, despite the decline in use of crystalloid fluids and associated increase in use of blood product in recent years, overt cases of ACS continue to be reported.

Abdominal visceral edema and IAH/ACS may result from a two "hit" pathophysiologic process named the "acute intestinal distress syndrome" in 2008 by Malbrain and De Laet [11, 43–45]. In the first "hit," resuscitation of patients with hypovolemic or septic shock produces an ischemia–reperfusion injury of the bowel [11, 38, 39, 46]. This "acute bowel injury" results in upregulated transcription, translation, and release of a number of pro-inflammatory mediators [e.g., tumor necrosis factor-alpha, interleukin (IL)-1, and IL-6) into the

peritoneal cavity [11, 38, 44]. In patients with peritonitis, peritoneal inflammatory mediator concentrations have been reported to be 10–1000 times higher than levels in blood [47].

These mediators promote neutrophil priming and increase intestinal wall and mesenteric capillary permeability, which results in translocation of bacteria across the intestinal wall, release of bacterial endotoxin, and extravasation of fluid into the bowel wall, its supporting mesenteries, and the peritoneal cavity [11, 38, 39, 48–54]. Further, the pro-inflammatory mediators produced during the acute intestinal distress syndrome may also adversely affect distant organs [47]. Mediators absorbed across the bowel wall/peritoneum, via intra-abdominal portal or systemic veins, or mesenteric lymphatics in the bowel wall, the cisterna chyli, and the thoracic duct have been reported to induce SIRS and MODS. As the gut represents the largest surface area in contact with the external environment (and constitutes a reservoir of over 100 trillion bacteria), this process may serve as the "motor" that drives MODS during critical illness [55].

In the second "hit" of the acute intestinal distress syndrome, abdominal visceral edema (and intra-abdominal packing, hematomata, and/or ascites, if present), along with a likely concurrent reduction in abdominal wall compliance among the critically ill/injured, produces IAH [11, 38, 39, 56, 57]. IAH compresses intra-abdominal lymphatics, decreasing lymph flow out of the abdominal cavity [38, 39]. It also decreases bowel wall arterial inflow and venous outflow, resulting in a progressive (mucosa-to-serosa) intestinal wall necrosis and a further increase in bowel wall permeability, elevated bacterial translocation/systemic endotoxin absorption, and heightened release of pro-inflammatory mediators [11, 38, 39, 58]. This results in a further increase in intestinal wall permeability, abdominal visceral edema, and intra-abdominal pressure (IAP), especially when patients continue to receive massive crystalloid fluid resuscitation [11, 38]. In doing so, these events initiate a self-perpetuating process of progressive acute bowel injury, abdominal visceral edema, and IAH (the acute intestinal distress syndrome), ultimately culminating in the ACS [11, 38].

Ironically, the effects of IAH on the gut are similar to those of prolonged hypoperfusion, and, therefore, these two issues are compounding. Even after resuscitation and normalization of hemodynamics, gut vasoconstriction persists and is further exacerbated by IAH. Prolonged gut hypoperfusion can precipitate a severe inflammatory response due to mobilization of damage-associated molecular patterns (e.g., high mobility group box 1, heat shock proteins, s100 proteins, nucleic acids, and hyaluronan), pro-inflammatory cytokines, and other mediators [59]. Thus, the OA may have a profound effect on reducing the progression of severe injury/infection with shock to MODS through ameliorating injury to the gut. However, it remains unknown if the benefit of the OA in this setting results from correcting IAH or draining inflammatory ascites. There is a complex relationship between pressure, ischemia, and inflammation within the peritoneal cavity. Independently, the damaged gut seems to act as a continued source of inflammation propagating SIRS and potentiating MODS [60–62].

4.5 Proposed Pathophysiologic Benefits of the OA

Use of the OA allows laparotomy to be quickly terminated, which in theory limits surgical time, tissue injury, blood and temperature loss, and fluid and blood product requirements. Although this is widely believed to assist in avoiding and/or interrupting the lethal triad in patients with the most severe injuries or signs of impending physiological exhaustion, limited studies exist to support this. Further, as many severely injured patients likely present with acute traumatic coagulopathy, DC surgery is likely incapable of preventing coagulopathy in the majority of the critically injured [7]. Finally, as the development of the lethal triad in earlier studies of DC surgery and open abdominal management were related to the use of standard, crystalloid-based resuscitation, and the lethal triad may be prevented with the use of hemostatic resuscitation strategies, studies are urgently required to determine the role of DC surgery and open abdominal management in the context of changing resuscitation practices [7].

A now long-proposed benefit of the use of the OA is that it prevents against development of the ACS after DC laparotomy [7, 63]. However, as outlined by Balogh and colleagues, cases of overt ACS requiring decompressive laparotomy are becoming increasingly rare in many practice settings as this syndrome has been largely "eliminated" by "strategic research and focused preventions," especially the avoidance of over-resuscitation with crystalloid fluids [64]. Thus, this indication may become increasingly less important in modern day surgical practice and the current era of improved fluid resuscitation strategies. When needed, decompressive laparotomy has been reported to result in nearly immediate and sometimes substantial improvements in organ dysfunction in patients with the ACS. Despite this, careful (but retrospective) study has revealed that although IAP is consistently lower after decompression, mortality remains considerable, and recuperation of organ dysfunction after decompressive laparotomy for ACS is variable [65].

Finally, although limited clinical evidence yet exists, studies have suggested that active negative pressure peritoneal therapy TAC techniques may remove peritoneal cytokines, reduce systemic inflammation, and improve outcomes in trauma and emergency general surgery patients with an OA. These types of TACs include noncommercial (e.g., the Barker's vacuum pack) and commercial devices. A study allocated animals with a fecal blood clot model of intra-abdominal sepsis to negative pressure peritoneal therapy [the Kinetic Concepts Inc. (KCI) vacuum-assisted closure device, KCI, San Antonio, TX, U.S.A.] versus passive drainage of the peritoneal cavity and observed reduced levels of systemic pro-inflammatory cytokines and improved intestine, lung, kidney, and liver histopathology [14, 52]. A multicenter prospective cohort study reported in 2013 that use of the ABThera Open Abdomen Negative Pressure Therapy Device (KCI) after abbreviated laparotomy in patients with intra-abdominal injury or sepsis was associated with improved adjusted survival among when compared to the Barker's vacuum pack (Fig. 4.1) [14, 66]. This concept was subsequently further examined by a single-center randomized controlled trial (RCT) that allocated patients who underwent DC laparotomy for abdominal trauma or intra-abdominal sepsis to the ABThera versus Barker's vacuum pack technique [13, 14]. Interestingly, this study observed no significant

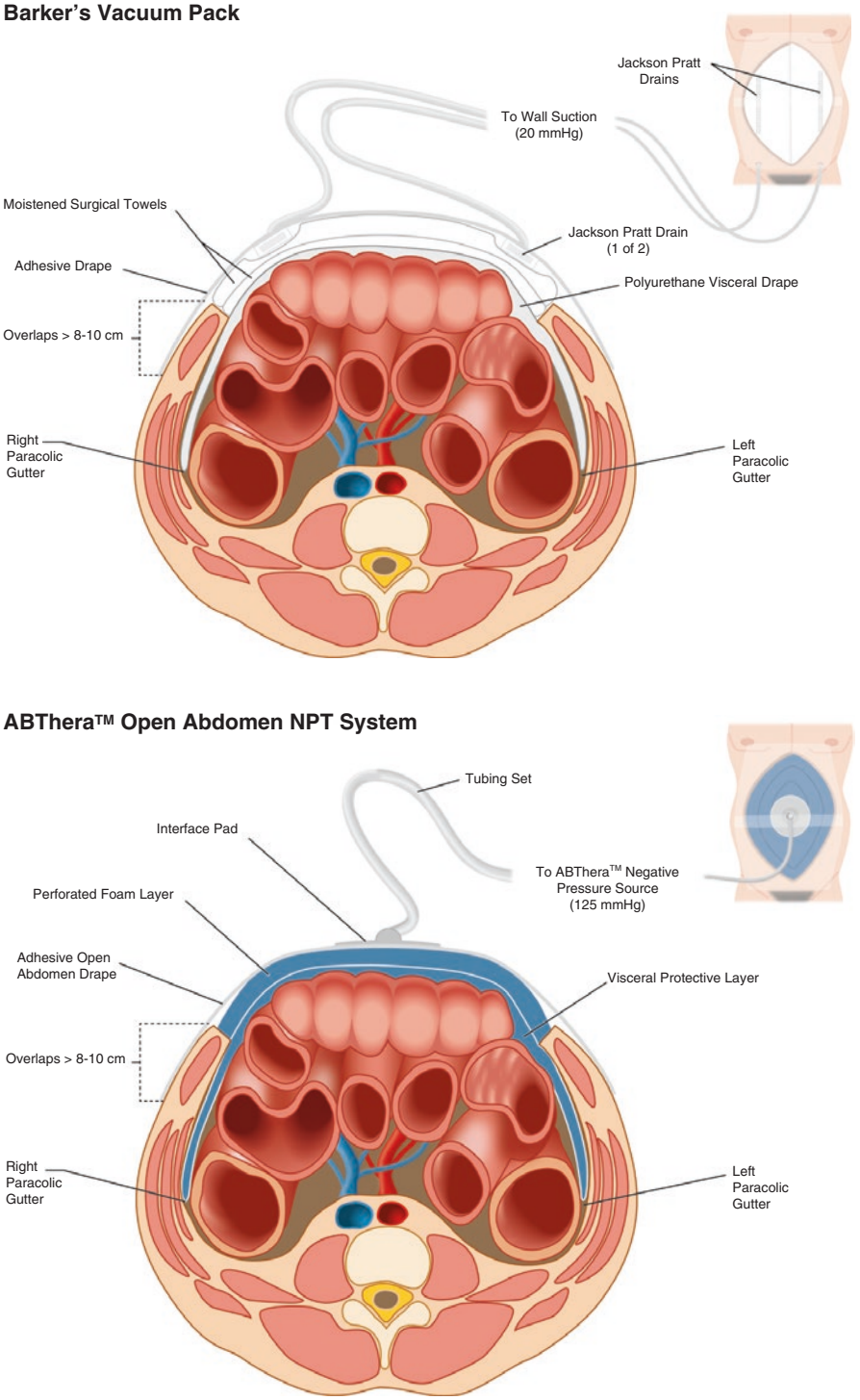

Fig. 4.1 Schematic of the Barker's vacuum pack (*left*) and ABThera Open Abdomen Negative Pressure Therapy System (*right*). Illustration reproduced from reference [13]

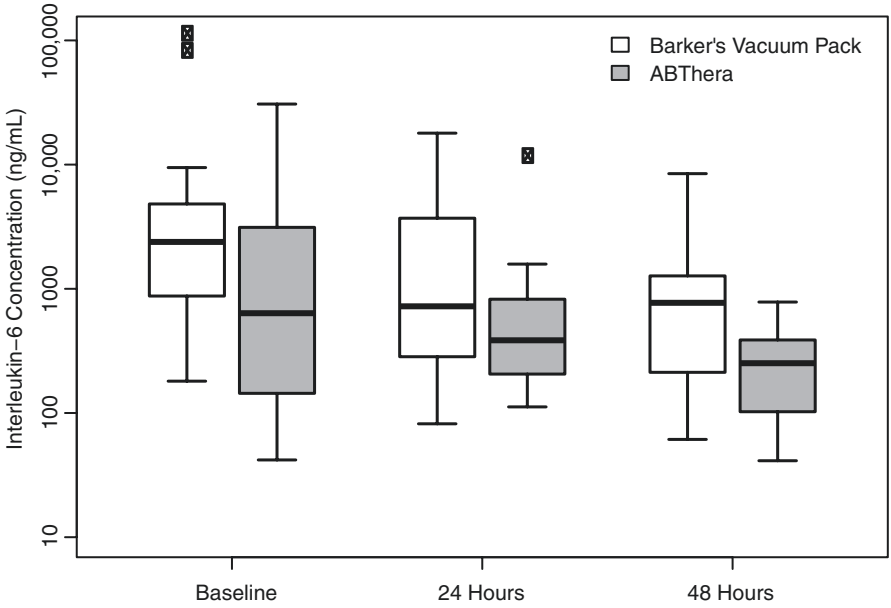

Fig. 4.2 Plasma interleukin-6 (IL-6) levels in a randomized controlled trial that allocated adults who underwent abbreviated laparotomy for intra-abdominal injury or sepsis to the ABThera Open Abdomen Negative Pressure Therapy System or the Barker's vacuum pack. Figures reproduced from reference [14]

difference in the plasma concentration of IL-6 and five other pro-inflammatory cytokines at baseline versus 24- or 48-h between groups (Fig. 4.2) [14]. However, the intention to treat analysis revealed a significantly improved 90-day survival in the ABThera versus Barker's vacuum pack group (hazard ratio = 0.32 for mortality) (Fig. 4.3) [14]. As the findings of improved survival could be the result of residual confounding or type I error, the authors suggested that further work was required to explain the potential mechanisms of improved outcomes and confirm the findings before they are used to inform surgical practice [14].

4.6 Pathophysiology of the OA and Its Associated Complications

There are several potential complications of open abdominal management. The proposed pathophysiology of some of the most significant of these complications is discussed below.

4.6.1 Progressive Abdominal Visceral Edema, Loss of Domain, and Massive Ventral Hernias

Patients who have suffered an acute bowel injury are likely particularly susceptible to fluid shifts during the early postoperative period after the index laparotomy for

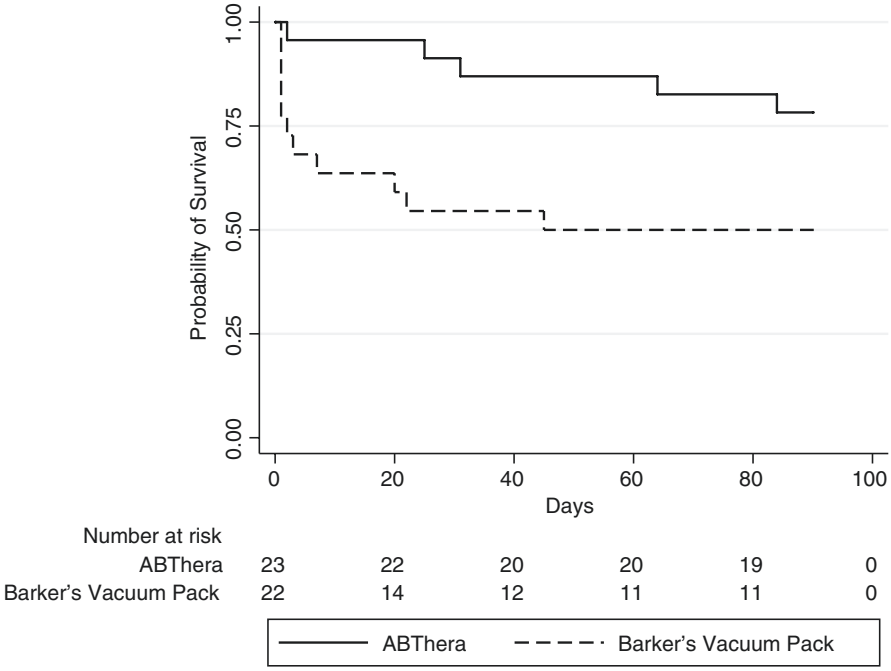

Fig. 4.3 Survival probability in a randomized controlled trial that allocated adults who underwent abbreviated laparotomy for intra-abdominal injury or sepsis to the ABThera Open Abdomen Negative Pressure Therapy System or the Barker's vacuum pack. Figures reproduced from reference [14]

open abdominal management [67]. Historically, they were also likely to receive a high volume and/or rapid infusion of crystalloid fluids during this period [67]. As fluid follows the path of least resistance, having an open abdominal cavity during this time may decrease resistance to flow and likely facilitates a rise in abdominal visceral edema and distention of abdominal contents that might not otherwise have occurred in a closed abdominal cavity [67]. This may result in a progressive increase in abdominal visceral edema (and an associated progressive decrease in the probability of primary fascial closure) after the index laparotomy [67]. In support of this argument, studies have suggested that if the abdomen is not closed early, the incidence of PFC declines significantly. In a prospective, multicenter cohort study, Pommerening et al. reported that after postoperative hour 24, each subsequent hour delay in returning to the operating room was independently associated with a 1% decrease in the odds of PFC [67].

If the abdomen is not closed within 10–14 days, a dense network of inflammatory and granulation tissue develops between bowel loops, the greater omentum, and the parietal peritoneum on the undersurface of the abdominal wall [7, 68]. This tissue forms a firmly adherent superficial layer over the entire viscera and acts to progressively obliterate the intraperitoneal space, creating a "frozen abdomen" [7, 68].

This process, when combined with the gradual, obligatory lateral retraction of the abdominal fascial edges and shortening of the abdominal wall muscles (which occurs because the lateral forces evoked by the oblique and transverse muscles overcome the medial forces evoked by the ipsilateral rectus muscles once the linea alba has been divided), both contribute to a progressive loss of intra-abdominal domain [7]. In the absence of surgical or TAC techniques that apply constant medial tension to the midline abdominal fascia, abdominal visceral edema and progressive lateral fascial retraction result in loss of domain and ultimately a massive ventral hernia.

4.6.2 Enteroatmospheric Fistulae and Enteric Leaks

With increased swelling, manipulation, trauma, desiccation, and infection of the bowel, enteroatmospheric fistulae or enteric leaks may form either superficial or deep within the abdominal cavity [7]. As outlined by Björck and colleagues, an enteroatmospheric fistula represents an enteric fistula within the middle of the open abdomen (with defining characteristics including the absence of a fistula tract, the lack of well-vascularized surrounding tissue, and spillage of enteric content directly into the peritoneal cavity) [10, 69]. In contrast, an enteric leak is characterized by spillage of enteric contents into the abdomen without established enteric fistula development [10, 69].

The pathophysiology underlying the formation of enteroatmospheric fistulae is inadequately understood. In a retrospective cohort study of 517 trauma patients with an open abdomen after DC laparotomy conducted across 14 trauma centers in the USA, Bradley et al. reported that independent predictors of the development of enteroatmospheric/enterocutaneous fistulae or intra-abdominal sepsis included large bowel resection, a total fluid intake at 48 h >5 L, and an increasing number of abdominal re-explorations [70]. Thus, crystalloid fluids, bowel manipulation, and local visceral trauma during repeated laparotomies may contribute to development of edematous and friable bowel and increase risk of enteroatmospheric fistulae and enteric leaks in patients with an OA [70–72]. Finally, although it remains unknown if this relationship exists in humans, the length of peritoneal air exposure has also been reported to be associated with a proportional increase in damage of the gastrointestinal tract in rats [73].

4.6.3 Energy Expenditure

The OA is associated with increased fluid and protein loss, which can produce nutritional insufficiency and a catabolic state [74]. A prospective cohort study by Cheatham et al. reported that nutritional calculations that fail to account for protein/nitrogen loss may underestimate actual nitrogen balance by an average of 3.5 g/24 h [75]. These authors suggested that in the absence of actual direct measurement of losses, an estimate of 2 g of nitrogen per liter of abdominal fluid should be included

in any nutritional calculations. However, with advanced TAC dressings, this concern may be less important. A recent RCT utilizing active negative pressure peritoneal therapy reported less fluid drainage compared to a less efficient TAC, with hypotheses being suggested that NPPT might actually reduce inflammation and thus edema generation [14].

As early enteral nutrition (EN) improves wound healing, decreases catabolism, preserves gastrointestinal integrity, and reduces septic complications and lengths of stay [75, 76], patients with an OA should be fed early. Despite this, there has been a reluctance to start feeds in this population, likely due to perceived concerns regarding the potential to exacerbate a preexisting ileus, visceral swelling, or clinical inattention to the benefits of EN. However, administering EN has been shown to be feasible in patients with an OA [74] and may even increase visceral blood flow [77], which has been speculated to mitigate bowel edema and facilitate PFC. Further, anything that preserves the integrity of the gut mucosal barrier and modulates inflammatory mediator generation presumably mitigates bowel swelling and therefore acts to improve the rate of PFC.

Conclusion

Surgeons leave the abdomen open for anatomical, physiological, and logistical reasons. Physiological derangements after damage control laparotomy are largely related to perfusion/systemic inflammatory disturbances induced by acute bowel injury and IAH. Importantly, both of these derangements are made worse by large-volume crystalloid fluid resuscitation. Potential physiologic benefits of the OA have long been hypothesized to include prevention or improvement of the adverse effects of ACS. The OA also prevents the more subtle consequences of IAH, facilitates use of active negative pressure peritoneal therapy, allows for delayed reconstructive options when abdominal domain has been lost, and permits planned abdominal re-exploration to remove sponges and reestablish intestinal continuity. However, the survival benefit of DC laparotomy has recently been questioned because of the introduction of improved resuscitation practices (such as DC resuscitation and the avoidance of large-volume crystalloid fluid resuscitation) and the risks associated with open abdominal management, including massive ventral hernias, enteric leaks, and enteroatmospheric fistulae. As equipoise begins to dwindle regarding the effectiveness and safety of liberal use of damage control laparotomy in the modern era of improved resuscitation practices, randomized evidence is increasingly required to elucidate the situations in which the associated benefits of open abdominal management outweigh the risks.

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