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Review Article

The open abdomen in trauma, acute care, and vascular and endovascular surgery: comprehensive, expert, narrative review

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Abstract

Background: The open abdomen is an innovation that greatly improved surgical understanding of damage control, temporary abdominal closure, staged abdominal reconstruction, viscera and enteric fistula care, and abdominal wall reconstruction. This article provides an evidence-informed, expert, comprehensive narrative review of the open abdomen in trauma, acute care, and vascular and endovascular surgery.

Methods: A group of 12 international trauma, acute care, and vascular and endovascular surgery experts were invited to review current literature and important concepts surrounding the open abdomen.

Results: The open abdomen may be classified using validated systems developed by a working group in 2009 and modified by the World Society of the Abdominal Compartment Syndrome—The Abdominal Compartment Society in 2013. It may be indicated in major trauma, intra-abdominal sepsis, vascular surgical emergencies, and severe acute pancreatitis; to facilitate second look laparotomy or avoid or treat abdominal compartment syndrome; and when the abdominal wall cannot be safely closed. Temporary abdominal closure and staged abdominal reconstruction methods include a mesh/sheet, transabdominal wall dynamic fascial traction, negative pressure wound therapy, and hybrid negative pressure wound therapy and dynamic fascial traction. This last method likely has the highest primary fascial closure rates. Direct peritoneal resuscitation is currently an experimental strategy developed to improve primary fascial closure rates and reduce complications in those with an open abdomen. Primary fascial closure rates may be improved by early return to the operating room; limiting use of crystalloid fluids during the surgical interval; and preventing and/or treating intra-abdominal hypertension, enteric fistulae, and intra-abdominal collections after surgery. The majority of failures of primary fascial closure and enteroatmospheric fistula formation may be prevented using effective temporary abdominal closure techniques, providing appropriate resuscitation fluids and nutritional support, and closing the abdomen as early as possible.

Conclusion: Subsequent stages of the innovation of the open abdomen will likely involve the design and conduct of prospective studies to evaluate appropriate indications for its use and effectiveness and safety of the above components of open abdomen management.

Introduction

An open abdomen (OA) describes a situation where all layers of the abdominal wall are left open. This may be indicated for damage control (DC), intra-abdominal sepsis, or vascular surgical emergencies; to facilitate second look laparotomy (for example when bowel viability is questionable or packs are left in the abdomen) or avoid or treat abdominal compartment syndrome (ACS); and when the abdominal wall cannot be safely closed. Some have even suggested it may have a role in improving source control of intra-abdominal sepsis. The abdominal wall may not be able to be safely closed when attempted closure induces severe intra-abdominal hypertension (IAH)/ACS or following burst abdomen or excision of an abdominal wall

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infection or tumour. Closing the fascia and leaving the skin open, or closing the skin but not the fascia, is not considered an OA.

In this narrative review, 12 international trauma, acute care, and vascular and endovascular surgery experts were invited to review current literature and important concepts surrounding the OA. The purpose was to provide an evidence-informed, comprehensive review of the OA in trauma, acute care, and vascular and endovascular surgery to guide surgeons, other clinicians, and researchers who may utilize or study this technique. Experts were invited based on clinical expertise and because they had published extensively on the OA and/or participated in OA clinical practice guidance documents. The following are reviewed: the history and classification of the OA; indications for and use of the OA in trauma, intra-abdominal sepsis, vascular surgical emergencies, and severe acute pancreatitis (SAP); temporary abdominal closure (TAC) and staged abdominal reconstruction (STAR) techniques; direct peritoneal resuscitation (DPR); the relationship between perioperative resuscitation strategies, risk of IAH/ACS, and the need for the OA; enteroatmospheric fistulas (EAFs) and the hostile OA; and abdominal wall reconstruction post-OA management.

History of the innovation of the open abdomen

There are three separate eras in surgical history that contribute to the current practice of the OA. First, in 1897, McCosh described use of the OA in eight patients with generalized peritonitis[1]. He applied traction to open laparotomy wound edges with two to three silkworm-gut sutures and covered the intestines with gauze allowing egress of peritoneal secretions. During World War II, Ogilvie subsequently used the OA to treat abdominal injuries and recommended using it for peritonitis[2]. The method was seldom again described until 1979 when Steinberg suggested leaving the abdomen open to drain in cases of generalized peritonitis[3].

The second era of development occurred during the innovation of DC surgery[4]. Although therapeutic perihepatic packing (that is prolonged intra- and postoperative packing for control of hepatic haemorrhage[5]) for severe liver injury had already been reportedly used by Pringle in 1908[6], the concept of DC laparotomy was first formally introduced by Stone *et al.* in 1983[7]. After abbreviated laparotomy for control of exsanguinating haemorrhage and gross contamination, Stone *et al.* therapeutically packed the abdominal cavity and then left it open before reoperation for definitive repair of all injuries 24–72 hours later after an interval of ongoing resuscitation in the intensive care unit (ICU). During the interim interval, the viscera were protected with a TAC. This operative process was later named 'damage control' by Rotondo and Schwab in 1993[8].

The third era began after surgeons recognized the deleterious effects that raised intra-abdominal pressure (IAP) had on organ systems. Although known since the mid-19th century, the clinical significance of IAH/ACS was first fully recognized after the increasing use of laparoscopy. Fietsam *et al.* then reported the first case series of 'intra-abdominal compartment syndrome' (increased peak inspiratory and central venous pressures, oliguric renal failure, and massive abdominal distention) after open repair of ruptured abdominal aortic aneurysms (rAAAs) and massive fluid resuscitation[4,9]. The initial description of treating ACS by opening the abdomen and leaving it open was published in 1984[10]. Subsequently, the World Society of the Abdominal Compartment Syndrome (WSACS)—The Abdominal Compartment Society published IAH/ACS definitions in 2006 and clinical practice guidelines on diagnosis and management of IAH/ACS in 2007; these were then updated by the Society in 2013[11].

Table 1 Classification of the open abdomen

2009 Classification system		Amended 2016 classification system	
1A	Clean OA without adherence between bowel and abdominal wall or fixity (lateralization of the abdominal wall)	1A	Clean, no fixation
1B	Contaminated OA without adherence/fixity	1B	Contaminated, no fixation
		1C	Enteric leak, no fixation
2A	Clean OA developing adherence/fixity	2A	Clean, developing fixation
2B	Contaminated OA developing adherence/fixity	2B	Contaminated, developing fixation
		2C	Enteric leak, developing fixation
3	OA complicated by fistula formation	3A	Clean, frozen abdomen
		3B	Contaminated, frozen abdomen
4	Frozen OA with adherent/fixed bowel, unable to close surgically, with or without fistula	4	Established enteroatmospheric fistula, frozen abdomen

OA, open abdomen. Enteric leak describes the situation where there is spillage of enteric contents into the abdomen without having an established enteric fistula development.

Classification of the open abdomen

The first OA classification system was developed during two consensus conferences and published by Björck *et al.* on behalf of a working group in 2009[12]. Classes 1 through 3 of the system were subsequently expanded during development of the updated 2013 WSACS—The Abdominal Compartment Society clinical practice guidelines[11], and later published as an independent article in 2016[13]. The classification systems from 2009 and 2016 are presented in *Table 1*. The aims of these systems are to: describe the patient clinical course and aid clinicians in objectifying progress or lack thereof; stratify patients into risk groups; educate clinicians training to perform OA management; and facilitate OA cohort comparisons between studies. The WSACS 2013 classification system has been reported to be valid and reliable in a study of 111 patients treated with a combination of vacuum-assisted wound closure and mesh-mediated fascial traction (VAWCM)[14]. In this study, the most complex grade and deteriorating grade, grade C and grade 4 OAs, were associated with the worst patient outcomes[14].

The open abdomen in trauma

Although DC laparotomy is considered by many to be life-saving when used among the most critically injured patients (often defined as those who are approaching 'physiological exhaustion'), it has potentially severe complications, including complex ventral hernias and EAFs[15]. Therefore, it should only be used when

appropriately indicated. In 2013, Roberts *et al.* and the Indications for Trauma DC Surgery International Study Group conducted a series of studies to determine the clinical situations in which the expected survival benefit of conducting DC laparotomy was expected to exceed the risk of complications[5,16–21]. They first conducted a scoping review and identified 1107 indications for DC surgery reported in the literature[19]. They then used qualitative research methods to reduce these 1107 indications into 123 unique indications for DC surgery, of which 101 (82.1 per cent) were subsequently rated to be appropriate for use in practice by an international panel of trauma surgery experts[21]. Roberts *et al.* then surveyed 366 surgeons who treated injured patients in level 1–3 trauma centres across four different countries (the USA, Canada, Australia and New Zealand) to determine their opinions on the appropriateness of the indications rated in the expert appropriateness rating study[18]. Respondents rated 15 (78.9 per cent) preoperative and 23 (95.8 per cent) intraoperative indications to be appropriate for use in practice[18]. The indications for DC laparotomy rated to be appropriate by both experts and practicing surgeons are listed in *Table 2*[18,21]. Although a subsequent systematic review by this research group found that few of these indications had evidence supporting that they were valid and/or reliable[17], a cohort study conducted in the USA observed that they accurately predicted use of DC laparotomy in practice[16]. Therefore, the indications listed in *Table 2* may be used to guide choice of operative profile (that is DC or definitive) during trauma laparotomy and trauma centre quality improvement practices[15,21].

Table 2 Published indications for damage control laparotomy in trauma patients rated to be appropriate by experts and practicing surgeons

Indication
Abdominal trauma requiring laparotomy
AND at least one of the following: 1. Persistent systolic blood pressure <90 mmHg or a successfully resuscitated cardiac arrest during transport to hospital 2. Persistent systolic blood pressure <90 mmHg in the preoperative setting or during operation 3. Preoperative core body temperature <34°C, arterial pH <7.2, or INR/PT >1.5 times normal (with or without a concomitant PTT >1.5 times normal) 4. Core body temperature <34°C and arterial pH <7.2 at the beginning of operation 5. Persistent core body temperature <34°C or persistent arterial pH <7.2 during operation 6. INR/PT and PTT >1.5 times normal or a clinically observed coagulopathy during operation 7. Core body temperature <34°C, arterial pH <7.2, and laboratory confirmed (INR/PT and/or PTT >1.5 times normal) or clinically observed coagulopathy in the preoperative setting, at the beginning of operation, or during the conduct of operation 8. Estimated blood loss >4 l in the operating room OR 9. >10 U PRBCs were administered to the patient in the pre- or pre- and intraoperative settings
An expanding and difficult to access pelvic haematoma
A juxtahepatic venous injury
An abdominal vascular injury and at least one major associated abdominal solid or hollow organ injury
Devascularization or destruction of the pancreas, duodenum, or pancreatoduodenal complex with involvement of the ampulla/proximal pancreatic duct and/or distal CBD

CBD, common bile duct; INR, international normalized ratio; PRBC, packed red blood cells; PT, prothrombin time; PTT, partial thromboplastin time.

The open abdomen in intra-abdominal sepsis

Secondary peritonitis may result from disruption of the gastrointestinal (GI) tract and severe complicated intra-abdominal sepsis (defined as uncontrolled or unconfined purulent, feculent, or enteric peritoneal spillage in the setting of intra-abdominal sepsis)[22]. Severe complicated intra-abdominal sepsis is associated with an increased risk of multiple organ failure, morbidity and mortality rates[23]. This results from life-threatening organ dysfunction caused by a dysregulated host response to infection[24].

Intra-abdominal sepsis is particularly challenging to manage as the pathology originates within a semi-rigid container within which the primary disease and subsequent therapies frequently, if not always, result in IAH[11]. This container comprises the bulk of the human microbiome within the GI tract. The primary disease combined with IAH and vasomotor changes quickly induces a pathological gut microflora or dysbiome that has adverse consequences for the host[25]. Animal studies have reported that IAH decreases the abundance and diversity of the gut microbiota and increases the relative amounts of pathogenic lipopolysaccharide-secreting Proteobacteria[26,27]. Further, dysbiotic faeces from previously IAH-injured rats exacerbates the injury[27].

The OA is sometimes utilized in patients with severe complicated intra-abdominal sepsis. This is because some have suggested that in the most critically ill patients it may reduce operating times, facilitate reoperation, allow for early identification and increased drainage of residual infection, avoid IAH/ACS, and permit potentially safer, delayed GI anastomoses[22,28]. An area of active research is therefore whether it may also permit better source control and removal of inflammatory mediator-rich peritoneal fluid[22,28]. The World Society of Emergency Surgery (WSES) suggests that despite high-quality evidence, the technique (OA) might be an important option in the treatment of severe peritonitis and severe sepsis/septic shock under the following circumstances: abbreviated laparotomy due to severe physiological derangement, the need for a deferred intestinal anastomosis, a planned second look for intestinal ischaemia, persistent source of peritonitis (failure of source control), or extensive visceral oedema with concern for the development of ACS[29,30].

Cornerstones of therapy for severe complicated intra-abdominal sepsis include early detection, initiation of antibiotic and haemodynamic resuscitation, and definitive control of the initiating cause. Failure of adequate source control is an independent predictor of mortality rate[31]. However, it remains unclear if source control relates to control of macroscopic contamination, biomediators and/or biomarkers, or resolution of deranged septic physiology[32]. In those with severe intra-abdominal sepsis, the abdominal cavity acts as a reservoir for proinflammatory cytokines[33]. Disrupting inflammatory flow to the systemic circulation or better removing inflammatory ascites ameliorates inflammation and multiple organ failure in animals[34].

Kirkpatrick *et al.* previously conducted an RCT that suggested use of a negative pressure wound therapy (NPWT) TAC technique that may afford potentially more efficient peritoneal drainage (The ABThera™ Open Abdomen Negative Pressure Therapy System (Acelity, San Antonia, TX, USA)) than another (the Barker's vacuum pack (see below)) may reduce the mortality rate[35,36]. However, this RCT was unable to demonstrate that the

ABThera™ significantly improved peritoneal fluid drainage at 24 or 48 hours after DC laparotomy; further, peritoneal drainage was significantly less with this device than the Barker's vacuum pack at 48 hours. They also found no significant between-group differences in peritoneal and systematic proinflammatory mediator concentrations that may have mediated the observed improvement in mortality rate[35].

An international RCT, the Closed or Open after Laparotomy for Severe Complicated Intra-Abdominal Sepsis (COOL) trial, recently began randomly allocating patients with severe complicated intra-abdominal sepsis to use of the OA and an NPWT TAC method or fascial closure at the end of the index emergency laparotomy (clinicaltrials.gov registration number: NCT03163095 ((http://www.clinicaltrials.gov))[37,38]. If this active negative pressure peritoneal therapy (that is application of NPWT to the OA) improves outcomes after severe complicated intra-abdominal sepsis, this may be a surgical strategy with global utility.

The open abdomen in vascular surgical emergencies

The OA may be used after endovascular or open repair of ruptured abdominal aortic aneurysms (rAAAs) or laparotomy for mesenteric ischaemia. For patients with mesenteric ischaemia, use of the OA may improve perfusion of the remaining bowel and facilitates re-look laparotomy[39]. DC resuscitation, which includes expedient operation to rapidly control haemorrhage, permissive hypotension, avoidance of excessive crystalloid administration, and administration of plasma, platelets, and packed red blood cells (PRBCs) in a 1:1:1 haemostatic ratio is often utilized in patients with rAAAs[40–43]. These interventions are thought by some to prevent or ameliorate the lethal triad (hypothermia, acidosis, coagulopathy) and visceral oedema, IAH, and ACS in vascular surgery and trauma patients[40–43].

The estimated incidence of ACS after open repair of rAAAs in one cohort study was 6.8 per cent[44] while that after ruptured endovascular aortic repair (rEVAR) was 9 per cent (95 per cent c.i. 8 to 11 per cent)) in a systematic review and meta-analysis of 44 studies[45]. Although it has been suggested that type II endoleaks (collateral retrograde flow into the residual aortic sac from aortic branches) from lumbar arteries or the inferior mesenteric artery into the ruptured aortic aneurysm sac may contribute to the development of ACS after rEVAR, this could not be verified in a large study that included a core lab study of all imaging[46]. However, it is well established that those who develop ACS after rEVAR do so earlier after completion of the operation than those who undergo open repair of an rAAA[47].

The development of ACS after rAAA repair has been associated with a mortality rate ranging from 17 to 83 per cent[44,45,47,48]. Further, those who require laparotomy during rEVAR for rAAA have an adjusted odds of mortality rate nearly six-fold higher than those that do not require laparotomy[49]. The duration of IAH before decompressive laparotomy has also been associated with an increased adjusted odds of postoperative renal replacement therapy (a complication which has repeatedly been reported to be a principal predictor of poor outcomes after rAAA)[47]. In patients undergoing rEVAR for rAAA, preoperative systolic blood pressure <70 mmHg, need for proximal aortic occlusion, and transfusion of >5 units of PRBCs have been reported to increase the risk of ACS[46]. Other risk factors likely include prolonged shock, preoperative cardiac arrest, and severe hypothermia (temperature <33°C) and acidosis (pH <7.2)[50,51].

In patients who develop overt ACS (defined as severe IAH in a critically ill/injured patient and new cardiorespiratory and/or renal failure[52]) after or during endovascular repair of rAAA, emergent decompressive laparotomy is indicated[11]. In patients with equivocal signs of ACS, a scoring system has been developed to help guide surgeons on when to perform intraoperative decompressive laparotomy after rEVAR[53]. A cohort study by Smidfelt *et al.* did not find a survival advantage or difference in major complications in patients whose abdomen was prophylactically left open instead of primarily closed after open repair of rAAA[54].

It remains unclear which TAC technique after decompressive laparotomy for rAAA is most effective and safe. A systematic review of seven non-randomized studies reported a primary fascial closure (PFC) rate ranging from 79 to 100 per cent and an aortic graft infection risk of 0 per cent after use of the Barker's vacuum pack, commercial NPWT, and VAWCM[55]. However, the number of patients included in these studies ranged from four to 30 and only 60–100 per cent had an rAAA[55]. A subsequent multicentre, retrospective cohort study conducted by Acosta *et al.* reported a PFC rate of 91.8 per cent after a median of 11 days among patients treated with VAWCM after aortic repair because their abdomen could not be closed at primary or secondary operation[56]. Sixty-one per cent of these patients had an rAAA, 3.7 per cent developed an aortic graft infection within 6 months, and the 1-year mortality rate was 28.6 per cent[56]. It is important to recognize that NPWT devices may drain substantial amounts of fluid containing albumin, which may need to be compensated for. Before applying a TAC to a patient post-rAAA, surgeons should consider therapeutic intra-abdominal packing and cover surgical aortic grafts with retroperitoneal tissue, the peritoneum, and/or the omentum to decrease the risk of graft infection during repeated laparotomies.

The open abdomen in severe acute pancreatitis

SAP occurs in 10 per cent of patients with acute pancreatitis and has an associated mortality rate of 18–49 per cent[57]. The relationship between SAP and ACS is well established and ACS is described by some authors as the most lethal complication of SAP[58]. Diagnosis of ACS in patients with SAP is difficult because symptoms and signs can resemble those of the systemic inflammatory response syndrome (SIRS), acute respiratory distress syndrome, infected pancreatic necrosis, and multiorgan dysfunction syndrome (MODS). Therefore, the most recent WSACS—The Abdominal Compartment Society guidelines recommend measuring IAP in all patients with SAP at admission and then at regular intervals (for example 4–6 h)[11].

The degree to which IAH contributes to the progression of MODS in patients with SAP is unknown. Although IAP correlates with Acute Physiology and Chronic Health Evaluation-II scores (APACHE-II) after SAP, a causal relationship between ACS and MODS has not been established. The development of MODS in patients with SAP is multifactorial and SIRS plays an important role in the pathogenesis. Activation of circulating neutrophils and monocytes in patients with SAP is an early event and often precedes ICU admission[59]. In experimental models, IAH has been shown to induce the release of proinflammatory cytokines into circulation and contribute to liver, intestine, pancreas, and lung injury[60].

The ideal treatment for SAP-related ACS is its prevention, which is likely best done by limiting excessive fluid administration (and especially excessive crystalloid fluid administration). The treatment of IAH/ACS after SAP should follow that suggested by the WSACS—The Abdominal Compartment Society[11]. Surgical

decompression is necessary and recommended when conservative methods are insufficient to manage IAH/ACS. A recent systematic review demonstrated that this intervention considerably reduces IAP and may possibly be associated with a lower mortality rate and improved respiratory and renal function[58].

However, it remains unknown when decompressive laparotomy should be performed and by what method (midline laparotomy, bilateral subcostal laparotomy, or subcutaneous linea alba fasciotomy), despite midline laparotomy being most common[61]. The OA in patients with SAP is associated with a significant morbidity rate, a high rate of reoperation on, enteric fistula formation, and a low rate of PFC.

Importantly, visceral or mesenteric ischaemia has been reported to be found among 34–62 per cent of patients after decompressive laparotomy for pancreatitis-related ACS[62,63]. This complication is difficult to diagnose in these patients without laparotomy and is associated with an increased mortality rate[62,63]. Therefore, decompressive laparotomy may be the preferred management of pancreatitis-related ACS when conservative management fails[62,63]. Unfortunately, the optimal IAP threshold requiring surgical decompression and timing for intervention is unclear. Recent evidence demonstrates that patients with an IAP >25 mmHg in the first 4 days after disease onset may potentially be candidates for surgical decompression[64].

Given the morbidity rate of the OA in patients with SAP, non-invasive means of lowering IAP are an attractive alternative and include percutaneous catheter drainage among those with intraperitoneal collections that would be appropriate to drain[65]. A severity-of-illness-matched cohort study compared percutaneous catheter drainage with decompressive laparotomy and reported that catheter drainage was similarly effective at reducing IAP and may have avoided need for open surgical decompression in 81 per cent of the 31 treated patients[65]. However, percutaneous catheter drainage will not assist in diagnosing intestinal ischaemia and it remains unknown at what time percutaneous drainage should ideally be performed in patients with SAP-related IAH and which placement technique should be used.

Temporary abdominal closure and staged abdominal reconstruction methods

When the abdomen is left open, the viscera need to be covered and the abdomen temporarily 'closed' to prevent visceral desiccation and fluid, protein, and temperature losses[66,67]. TAC methods include the Bogotá bag (*Fig. 1*), Wittmann patch[68], and non-commercial (that is the Barker's vacuum pack)[69] (*Fig. 2*) and commercial NPWT systems (for example the ABThera™ Open Abdomen Negative Pressure Therapy System (Acelity, San Antonia, TX, USA) and The Renasys AB Abdominal dressing kit (Smith & Nephew, Memphis, TN, USA)) (*Fig. 3*). Further, if the abdomen cannot be closed after patients undergo their first re-look laparotomy, a STAR technique may be used during which the abdomen is progressively closed during a series of relaparotomies[67]. These usually include NPWT combined with some form of dynamic fascial traction. Important principles of TAC and STAR include preventing adhesion formation between the intestines and abdominal wall (through use of a visceral protective sheet placed between the viscera and parietal peritoneum and tucked deep into the pericolic gutters) and lateralization of the abdominal wall away from the midline (by placing constant or progressive, dynamic tension on the midline fascia)[67]. The Eastern Association for the Surgery of Trauma (EAST) clinical practice guideline conditionally recommended use of

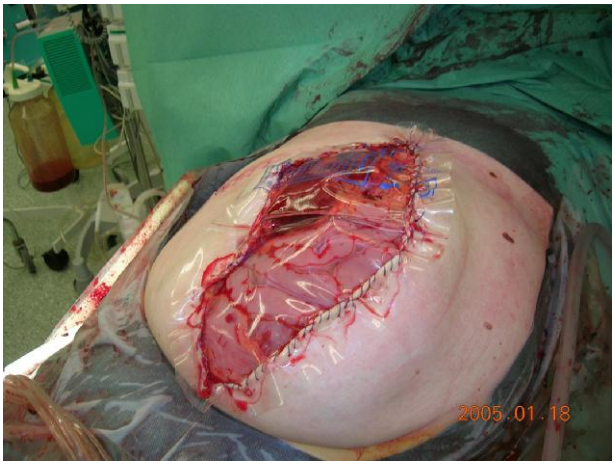

Fig. 1 Bogotá bag

fascial traction over routine care in patients with an OA to improve PFC[70]. This can be achieved by progressive fascial suture closure; the Wittmann patch; the ABdominal Reapproximation Anchor (ABRA) device (Canica Design Inc. Almonte, Ontario, Canada); or mesh-mediated fascial traction as in VAWCM (*Fig. 4*)[67,71]. *Table 3* provides a detailed description of TAC and STAR techniques.

NPWT is recommended as a preferred TAC method for patients with an OA by a number of international societies (including the WSACS—The Open Abdomen Society, WSES, and European Hernia Society (EHS)) and several evidence syntheses[11,30,83–86]. The WSES, EHS, and EAST also recommend the use of dynamic fascial closure in combination with NPWT. However, these recommendations are based largely on low-certainty evidence. A 2022 Cochrane systematic review of RCTs reported that based on sparse available trial data (only two RCTs, including one comparing NPWT to the Bogotá bag and another comparing it to an alternate NPWT system), the authors were uncertain whether NPWT had any benefit over alternate methods of TAC in terms of PFC, fistula formation, or mortality rate in non-trauma patients[72]. Kirkpatrick *et al.* previously conducted an RCT that reported use of the ABThera™ instead of Barker's vacuum pack may reduce the mortality rate in trauma and non-trauma patients[35]; however, results of this RCT have not been replicated. Finally, systematic reviews and meta-analyses of largely uncontrolled cohort studies have reported that NPWT, the Wittmann patch, and VAWCM in particular, are associated with high rates of PFC and low rates of mortality and enteric fistula formation (see *Table 3* for a summary of some of the results of these meta-analyses)[62,73–82]. However, results of these systematic reviews are often difficult to interpret because of their pooling of studies with and without control groups and the varying methodology used by the included primary studies[67]. Further, many of these studies mix young trauma victims treated with an OA for only 24–48 h with more complex patients who frequently had intra-abdominal sepsis and required weeks or even months of OA management.

Direct peritoneal resuscitation

In the 1990s, surgeons became increasingly aware of the association between aggressive fluid resuscitation, IAH/ACS, and outcomes. This led the group in Louisville, Kentucky, to experiment with alternate strategies to improve outcomes in those with an OA. Initial work by Zakaria *et al.* using

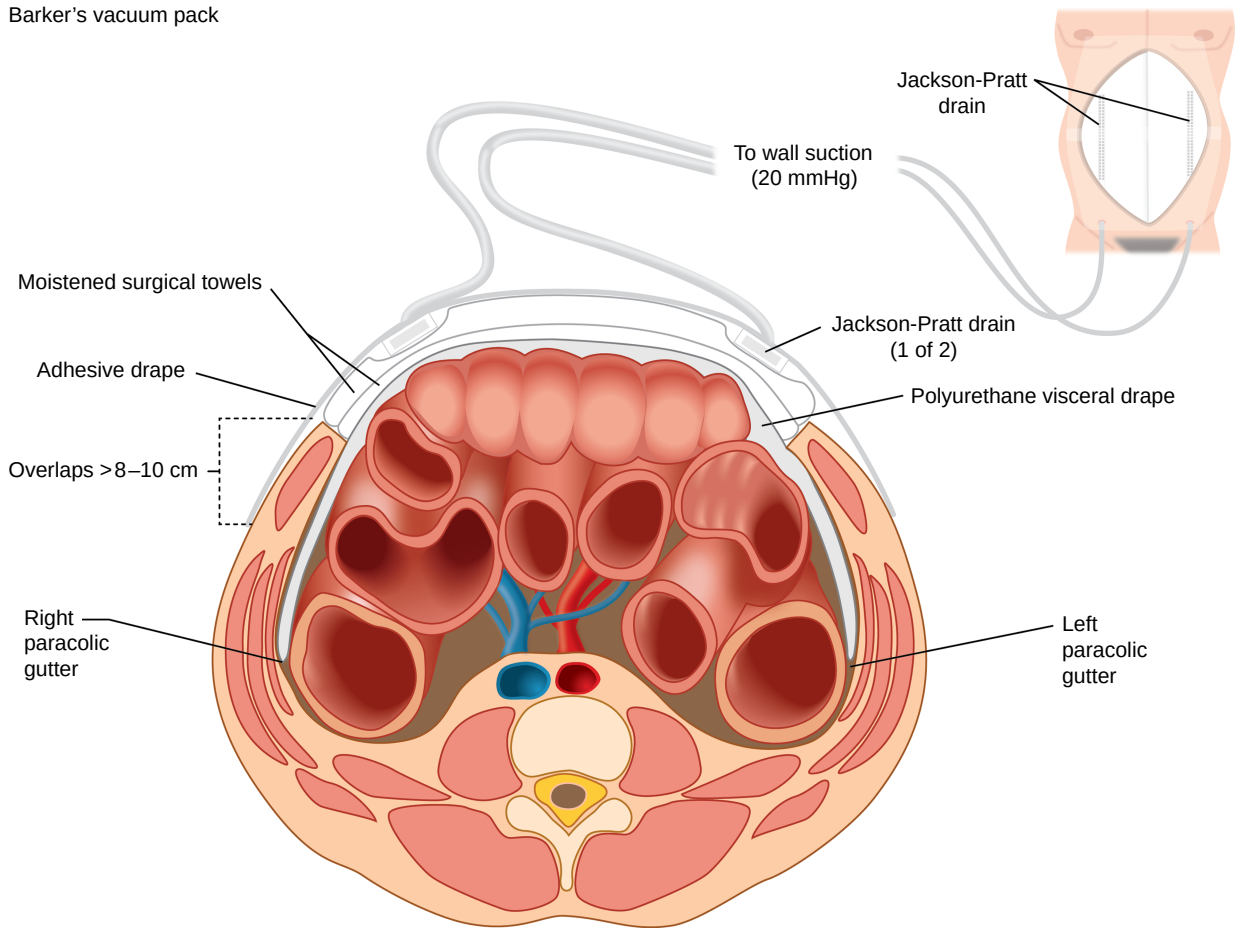

Fig. 2 Barker's vacuum pack

Figure reproduced from Roberts *et al.*[36]

commercially available peritoneal dialysis 4.25 per cent glucose-based solution (Delflex; Fresenius USA, Ogden, UT, USA) reported that this solution increased intestinal blood flow from a baseline of 568 (s.d. 31) nl/second to 1049 (s.d. 46) nl/second[87]. Garrisson *et al.* subsequently reported that DPR prevented haemorrhagic shock/resuscitation-induced organ ischaemia and hypoperfusion[88]. Enhancement of organ blood flow by DPR is attributed to the vasoactive nature of the dialysis solution driven predominantly by its osmolarity[88]. DPR may cause visceral arteriole dilatation, augment hepatic and visceral blood flow, and reduce organ oedema and serum levels of proinflammatory cytokines following haemorrhagic shock.

There are two commonly reported approaches to performing DPR (*Table 4* and *Fig. 5*). Although there is limited evidence to guide the optimal infusion, infusion volumes, and dwell time, it is important to monitor the patient's blood glucose as patients may absorb it from the peritoneal cavity. In the first technique, 300 ml/hour of 2.5 per cent glucose-based peritoneal dialysis solution is instilled via a Robinson catheter placed in the left upper quadrant at the root of the small bowel mesentery and fluid is removed via NPWT until abdominal closure is achieved. In the second technique, 300 ml/hour of 2.5 per cent glucose-based peritoneal dialysis solution is instilled via a superior Sensatrac pad of an ABThera™ with 30 minutes of soak time. This fluid is then removed in 1 hour with a target pressure of 100 mmHg until definitive abdominal closure. One of the authors uses 3-hourly cycles when performing the above technique.

Smith *et al.*, who pioneered the technique, suggested that DPR could assist in achieving earlier and increased PFC rates and reduced complications[89]. In 2016, in an RCT of DPR in 103 patients undergoing DC surgery (52 of which were allocated to DPR and 51 to a control resuscitation group), time to PFC was reduced in the DPR compared with the control group (4.1 (s.d. 2.2) days *versus* 5.9 (s.d. 3.5) days; $P \leq 0.002$)[90]. PFC was also higher in the DPR group (83 per cent *versus* 67 per cent; $P = 0.05$)[90]. Edwards *et al.* recently reported that DPR had no impact on abdominal infectious complications; further, if DPR is not initiated at the index operation, time to closure may be significantly prolonged[91]. In a recent systematic review of 20 articles on DPR, the authors reported that DPR had beneficial local and possibly systemic effects on perfusion, hypoxia, acidosis, and inflammation, and may potentially be associated with improved outcomes and reduced complications[92]. However, many of the studies in this systematic review were preclinical and therefore at present the technique remains experimental and an active area of investigation.

Perioperative resuscitation strategies, intra-abdominal hypertension/abdominal compartment syndrome, and need for the open abdomen

Use of trauma resuscitation strategies that utilized excessive crystalloid fluids likely contributed to an epidemic of IAH/ACS and the OA in the 1980s to 2000s[4,93]. The crystalloid fluids

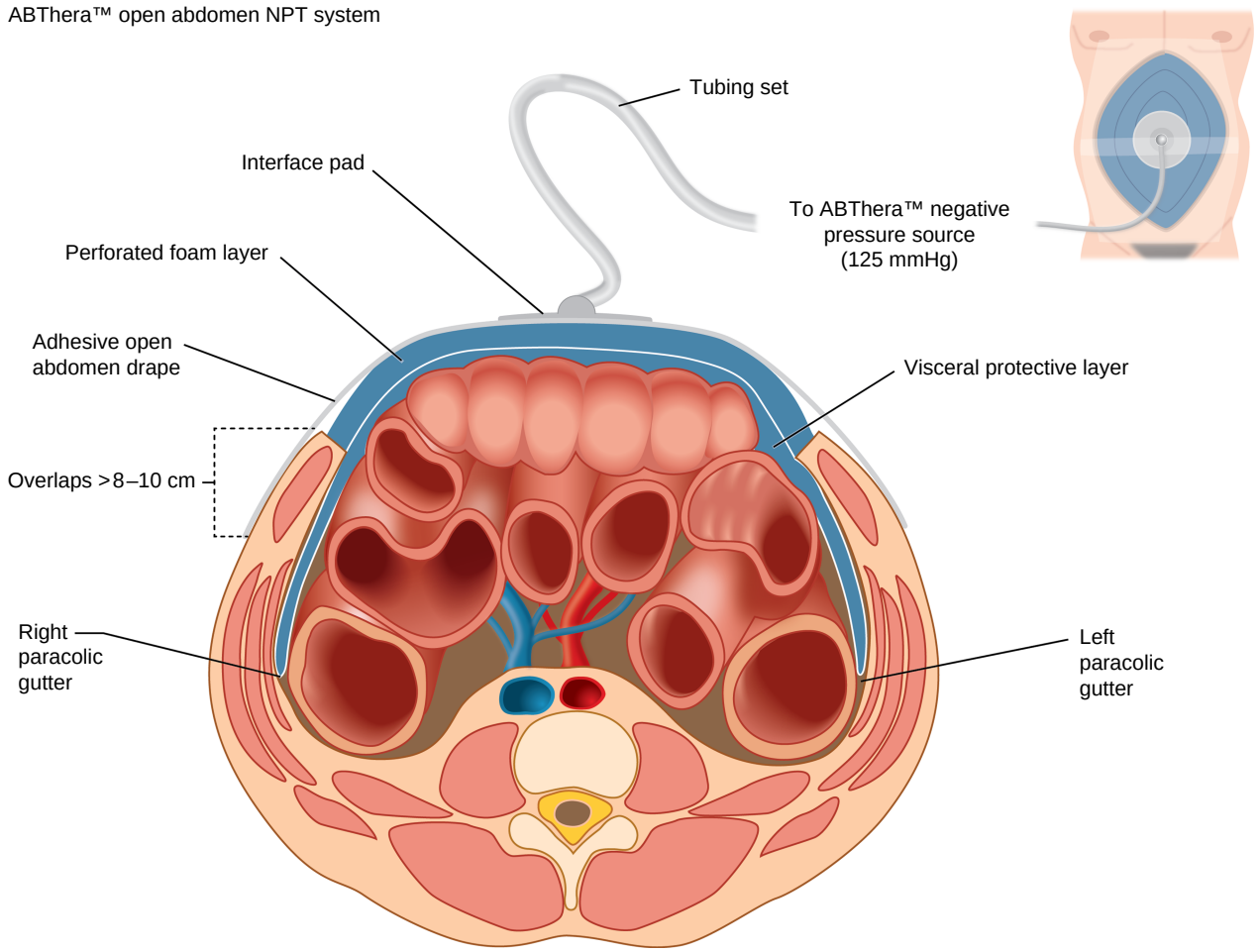

Fig. 3 ABThera™ Open Abdomen Negative Pressure Therapy System
Figure reproduced from Roberts *et al.*[36]

provided to these patients in the Emergency Department and operating room in combination with the practice of 'supranormal resuscitation of trauma patients' in the ICU (to optimize oxygen delivery and prevent occult hypoperfusion) led to midgut oedema, elevated IAP, and markedly increased risk of primary ('refers to a condition associated with an injury or disease in the abdominopelvic region') and secondary ('refers to conditions that do not originate in the abdominopelvic region') IAH/ACS[4,11,93]. These events increased the incidence of and need for decompressive laparotomy and the OA postinjury[4,11,93].

As outlined by Balogh *et al.*, increasing use of DC resuscitation (which consists of expedient operation to rapidly control haemorrhage, avoidance of excessive crystalloid administration, and administration of plasma, platelets, and PRBCs in a 1:1:1 haemostatic ratio) essentially eliminated postinjury primary ACS and minimized the prevalence of secondary ACS[93]. It also likely changed the indications for use of trauma DC laparotomy and increased the probability of earlier PFC among those with an OA postinjury. In support of this, in a survey of 366 surgeons who treated injured patients in level 1–3 trauma centres conducted by Roberts *et al.*, most respondents indicated that patients with physiologic derangements that significantly improve or reverse during operative resuscitation were candidates for definitive closure of their laparotomy at the end of the index operation[18]. Further, in a retrospective cohort study of 172 severely injured combat casualties admitted to a US military hospital, use of ratio-driven resuscitation (defined as a ratio of 0.8 to 1.2 U of PRBCs to 1 U of fresh frozen plasma) instead of non-ratio-driven resuscitation was associated with an increased adjusted odds of early PFC after trauma laparotomy[94].

In the postoperative interval, resuscitation of patients with an OA should aim to achieve a balance between management of their deranged physiology and minimization of volume overload and IAH so that early PFC can be achieved[95]. Cohort studies of trauma patients with an OA suggest that PFC rates may be improved by returning to the operating room as early as possible after the index laparotomy (and ideally within 24 hours); preventing and/or treating IAH, enteric fistulae, and intra-abdominal collections after surgery; and limiting use of crystalloid fluids and fluid-related weight gain during the surgical interval (*Table 5*)[96–99]. In support of this, in a retrospective cohort study of patients undergoing OA management with the VAWCM technique, patients with a fluid-related weight gain ≥10 per cent had a lower PFC rate than those with a fluid-related weight gain <10 per cent (PFC rate = 33 *versus* 77 per cent respectively). Further, PFC rates seemed to possibly decrease even further when fluid-related weight gains were >20 per cent[100].

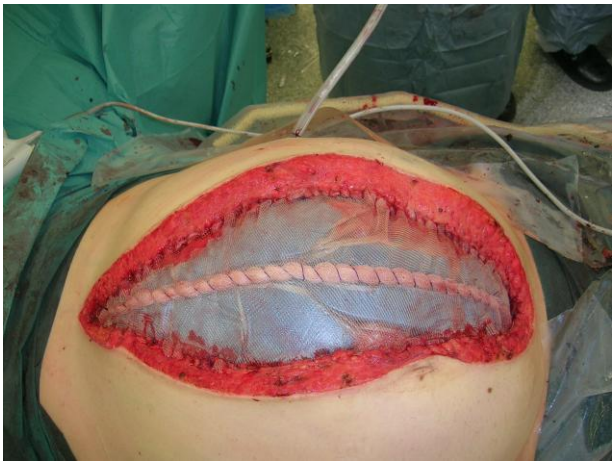

Fig. 4 Vacuum-assisted wound closure and mesh-mediated fascial traction (VAWCM)

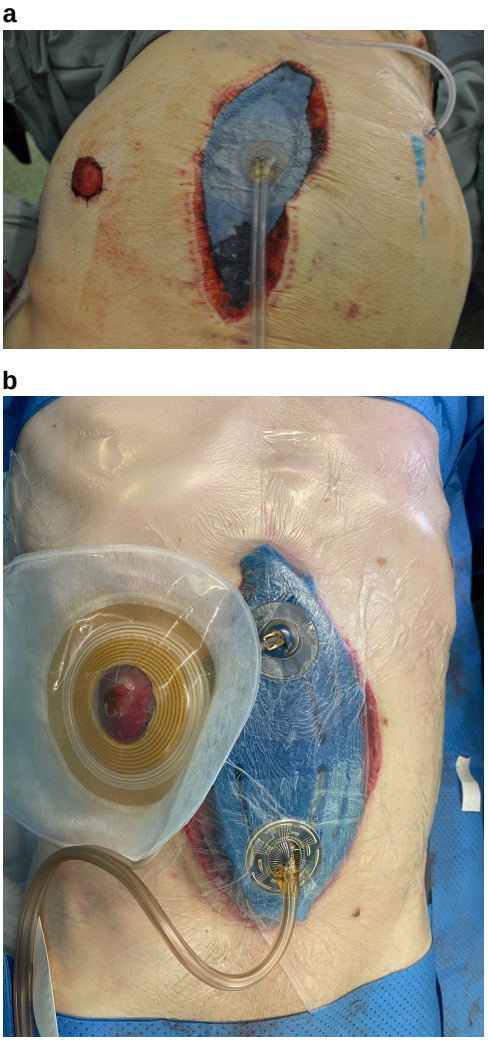

Fig. 5 Drain (a) *versus* Veraflo (b) method of direct peritoneal resuscitation

Enteroatmospheric fistulas and the hostile abdomen

EAFs are a devastating complication of OA management associated with severe morbidity rate, reduced patient health-related quality of life, and increased rates of mortality, duration of hospital stay, and hospital costs[101]. An EAF is defined as a communication between the GI tract and atmosphere (that is instead of the skin as in an enterocutaneous fistula (ECF)) (*Fig. 6*) and may be classified as low (<200 ml/day), moderate (200–500 ml/day), and high (>500 ml/day) output fistulas[102]. Generally, the more proximal the fistula is located within the GI tract, the higher the output.

Delayed abdominal closure, non-protection of bowel loops, failing to keep the viscera separated from the underside of the abdominal wall, careless handling of the viscera, large fluid resuscitation volume (>5 l/24 hours), the number of abdominal re-explorations, and the application of polypropylene mesh directly to the viscera increase risk of this complication[73,103–108]. Although use of NPWT as a TAC method and presence of bowel injury, bowel repairs or anastomoses, and intra-abdominal sepsis/abscesses were previously considered risk factors for EAF, subsequent studies did not support these associations[109,110]. Likely the most important predictors of EAF formation in patients with an OA is the duration of OA management and poor patient nutritional status[109]. There seems to be a cut-off after 2 weeks of having an OA after which the risk for EAF formation increases[12,55].

Every effort should be undertaken to prevent EAF formation in patients with an OA[101]. We believe that the vast majority of failures of delayed primary fascial closures, EAFs, and hostile abdomens can be prevented using effective TAC techniques, optimizing use of resuscitative liquids, closing the abdomen as early as possible, and providing nutritional support. Multiple studies have reported that early enteral nutrition administration improves closure rates and patient outcomes[111–115]. Even if it may increase EAF output (especially for more proximal EAFs), this is not associated with poorer patient outcomes[116,117]. Other effective preventive interventions include covering the bowel with plastic sheets, omentum, or skin and avoiding direct application of synthetic prostheses or NPWT foam to the bowel[82,118,119]. A propensity-matched case-control study by Willms *et al.* also suggested that covering the bowel with a visceral protective layer is important because those with an OA and secondary peritonitis had a 2.9 per cent risk of EAF formation when a visceral protective layer was used compared with 26.5 per cent when it was not[120].

EAFs very rarely heal spontaneously. Their initial surgical treatment should aim to isolate the effluent branch and promote granulation tissue formation around this branch to close it or at least transform it into an abdominal stoma. Several valid techniques have been described to achieve this[82,103–105]. NPWT may also be used to allow effluent isolation and aspiration while simultaneously creating the environment to let surrounding tissues granulate and transform the EAF into an enterostomy. EAF migration is a recently described technique: the use of the doughnut inside the abdomen 'excluding' the fistula site from the rest of the abdomen under NPWT may allow closure of the abdomen by controlling the EAF and migrating it from the midline to laterally under the fascia[121]. Focusing on migrating enteric effluents outside the OA and transforming an EAF into an ECF must be a priority. Definitive treatment of an EAF is generally considered to include closure of the fistula and abdominal wall reconstruction.

Table 3 Temporary abdominal closure and staged abdominal reconstruction techniques[62,67,72–82]

Type	Description	Approximate prognostic estimates from SR/MAs, %		
		PFC	Fistula	Mortality rate
Silo				
Bogotá bag	A sterile X-ray cassette or 3 l urological irrigation bag is sutured between the skin	28–47	0–15	30
Mesh/sheet				
Synthetic absorbable, synthetic non-absorbable or biologic	A non-adherent plastic sheet is placed over the viscera and an absorbable sheet (for example polyglycolic acid or polyglactin 910), non-absorbable sheet (e.g. polyprolene or polypropylene) or biologic (for example human acellular dermal matrix) is sutured between the fascial edges and progressively plicated at relaparotomies	34–39	8–17	14–34
Dynamic fascial traction				
Wittmann patch	The viscera are covered with a perforated, non-adherent plastic sheet. A large, flexible backed-loop sheet is sutured to one side of the abdominal wall fascia. A smaller, more rigid hook sheet is sutured to the opposite abdominal wall fascia. The hook sheet is then placed on the loop sheet and the overlapping edges are trimmed. A hypobaric wound shield consisting of sterile gauze, a closed suction surgical drain, and a self-adhesive plastic drape, is then applied over the wound and connected to wall suction. The two opposing velcro sheets may be progressively advanced towards the midline with serial dressing changes	75–90	2–3	13–24
Non-commercial and commercial negative pressure wound therapy				
Barker's vacuum pack	The viscera are covered with a perforated, non-adherent plastic sheet. This sheet is covered with surgical towels and two closed suction, surgical drains (for example Jackson-Pratt drains) that are tunnelled out the skin in a cranial direction. An adhesive transparent drape is placed over the wound to create an airtight seal and the drains are connected to wall canister suction	52	6	27
RENASYS-F/AB Abdominal Dressing (Smith and Nephew, Inc., Canada and USA)	The abdominal viscera is covered with a non-adherent, perforated plastic drape, and two pieces of polyurethane foam are placed over the plastic drape between the laparotomy edges. An adhesive drape is placed over the wound and surrounding skin and a track pad is connected to a suction sump and fluid drainage system via the track pad	51–60	3–15	18–30
ABThera™ Open Abdomen Negative Pressure Therapy System (Acelity, San Antonio, TX, USA)	The viscera are covered by a protective layer comprised of six radiating foam extensions enveloped in a sheet with small fenestrations. Two layers of perforated foam are then placed over the protective layer. An adhesive open abdomen drape is then placed atop the wound and adjacent skin and an interface pad is used to connect the dressing to a negative pressure source			
Hybrid negative pressure wound therapy and dynamic fascial traction				
Vacuum-assisted wound closure and mesh-mediated fascial traction	A heavy-weight polypropylene mesh is sutured to the bilateral fascial edges. The viscera are covered by a protective sheet. The mesh is then closed under tension, pulling the bilateral fascial edges towards the midline. Two pieces of foam are placed between the laparotomy edges and an adhesive open abdomen drape is placed atop the wound and adjacent skin. An interface pad is used to connect the dressing to a negative pressure source. The mesh is then progressively closed and the dressing steps repeated during serial relaparotomies	84	6	28
Abdominal Reapproximation Anchor (ABRA) device (Canica Design Inc. Almonte, Ontario, Canada)	A fenestrated silicone sheet visceral protector is placed over the viscera. Elastomers are passed 90° through the skin and full thickness through the abdominal wall. Button anchors are placed along the laparotomy skin edge. The tension is set on the silicone elastomers and a VAC sponge (for example ABThera™) is placed ventral to the silicone sheet elastomers and suction is applied. The elastomers are then tightened in intervals to achieve medialization of the fascial edge at the bedside or in the operating room	NA	NA	NA
Negative pressure wound therapy and dynamic fascial traction	Dynamic fascial traction may be achieved with use of sutures, the ABRA, the Wittmann patch, or other measures	73–76	6	12–22

ABRA, abdominal reapproximation anchor device; NA, not available; PFC, primary fascial closure; SR/MA, systematic review and meta-analyses; VAC, vacuum-assisted closure.

Table 4 Approaches to performing direct peritoneal resuscitation

Step	Drain technique	Veraflo technique
Instillation	Left upper quadrant 19F Robinson catheter placed around root of mesentery along left pericolic gutter and into the pelvis	Sensatrac pad of ABThera VAC and infusion using Veraflo
Abdominal coverage	Fenestrated visceral protective layer placed under the abdominal wall fascia	Fenestrated visceral protective layer placed under the abdominal wall fascia
Suction drain	Suction through abdominal NPWT device	Suction through inferior Sensatrac pad of ABThera VAC

NPWT, negative pressure wound therapy; VAC, vacuum-assisted closure.

Abdominal wall reconstruction after open abdomen

Latifi previously reported a nine-step strategy to abdominal wall reconstruction after OA that includes management of a concomitant ECF/EAF[106]. This strategy is known as ISOWATS PL: I = identification of postoperative fistulas, S = sepsis control and eradication, O = optimization of nutrition, W = wound care, A = redefining the anatomy, T = timing of operation or take down of ECF/EAF, S = surgical approach, P = postoperative care, and L = long-term follow-up[106].

Step 1: I = identification of postoperative fistulas

Fistulas must be first identified clinically and using computed tomography (CT).

Step 2: S = sepsis control and eradication

Early antibiotic use and source control of potentially drainable collections should be performed. In patients with severe sepsis and septic shock, open and timely laparotomy for source control is mandatory.

Step 3: O = optimization of nutrition

A combination of total parenteral (TPN) and enteral nutrition should be used in patients with an OA and ECF/EAF whenever possible. For patients undergoing multiple surgical procedures (for example DC laparotomy), nutritional support should include TPN.

Step 4: W = wound care

Wound care is perhaps one of the most important adjuncts in the management of complex wounds and ECF/EAF that likely improves both the physical and mental health of the patient.

Step 5: A = redefining the anatomy

Redefining the anatomy is the extension of the first step, which includes identifying fistulas where present and understanding the anatomy of the hernia and other associated pathologies. During this stage, meticulous analysis of previous operative reports and discussion with the operating surgeon (when possible, in case the same team is not involved) is of paramount importance. Abdominopelvic CT is often utilized.

Step 6: T = timing of surgical repair

If an ECF/EAF does not close within 4 to 6 weeks, it is unlikely to do so spontaneously. The decision when to operate is both surgeon and patient dependent. Operating too early may result in enterotomies, recurrent fistulas, and excision of larger amounts of bowel. Once the sepsis is controlled and nutrition optimized, a definitive operation can be attempted. However, in many cases, waiting several months for tissues to become 'ripe' is recommended even though this may increase the risk of losing further abdominal domain. During the waiting interval, optimal protection of the skin is important. Some patients may benefit from the application of a skin graft. If the raw area around the fistula was covered with a skin graft, it often takes 9–12 months before the graft can easily be separated from the underlying viscera.

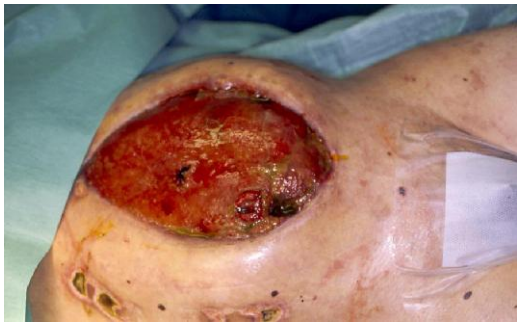

Fig. 6 Enteroatmospheric fistula (stage 4 open abdomen)

Step 7: S = surgical approach

Even though a staged approach to giant hernias has long been advocated[107], many surgeons complete the entire abdominal wall reconstruction in one stage where possible. The surgical approach may be divided into entry into the abdomen, adhesiolysis, and exploration of the entire abdominal cavity and interloop spaces, restoration of GI continuity, and abdominal wall reconstruction.

Entering the abdomen

When an OA was covered with a prior skin graft or granulation tissue, the presence of a 'positive pinch test' is associated with safer abdominal entry (*Fig. 7*). The safest way to enter the abdomen is often via the non-violated area of the incision away from prior scar.

Adhesiolysis

Once inside the abdomen, meticulous dissection of the intestine from the abdominal wall, adjacent bowel loops, and omentum should be performed. While performing adhesiolysis, any foreign body, unhealthy soft tissue, and/or ECF should be freed, marked, and/or resected. All enterotomies and serosal tears should be marked and closed.

Restoring gastrointestinal continuity

Bowel anastomoses should be performed only if bowel ends appear healthy, distal patency can be confirmed, anastomoses are located away from abscess cavities, and the patient is haemodynamically stable. The length of remaining bowel should be measured and this and the location of intestinal anastomoses and bowel repairs documented. Feeding tube placement should be considered intraoperatively if patients may not be able to resume oral nutrition soon after operation.

Abdominal wall reconstruction

Often the midline abdominal wall cannot be approximated because of loss of domain due to lateralization of the fascia, the disease itself, or because a portion of the abdominal wall is missing. One form of components separation technique should be employed to achieve tension-less closure and create a 'neo-linea alba' when the abdominal midline cannot be reapproximated without tension[108]. To reinforce the abdominal wall closure, a mesh is

Table 5 Significant adjusted predictors of primary fascial closure in patients with an open abdomen

Predictor	First author	No. of patients	Patient population	OR (95% c.i.)	Analyses adjusted for
Injury severity					
ISS	Beale[96]	62	Trauma	1.06 (1.00–1.12)	BE upon arrival to the ED, PATI score, penetrating injury
ISS	Hatch[98]	242	Trauma	1.00 (0.96–1.0)	VAC dressing used at initial laparotomy, ICU INR, ICU BD, ICU peak IAP (mmHg), 24 h crystalloid volume, 24 h crystalloid, PRBC, and plasma volume
ISS >15	Dubose[97]	517	Trauma	0.40 (0.17–0.94)	No. of re-explorations, intra-abdominal abscess/sepsis, bloodstream infections, enteric fistula, acute renal failure, age, gender, chest AIS, pH, lactate, estimated intraoperative blood loss, acidosis, intraoperative blood products, operating room fluid balance, number of packs used, small bowel resection, bowel left in discontinuity
PATI score	Beale[96]	62	Trauma	0.94 (0.90–0.99)	BE upon arrival to the ED, ISS, penetrating injury
Degree of physiologic derangement upon presentation					
BE upon arrival to the ED	Beale[96]	62	Trauma	1.27 (1.08–1.52)	ISS, PATI score, penetrating injury
INR upon arrival to ICU	Hatch[98]	242	Trauma	0.18 (0.034–0.98)	ISS, VAC dressing used at initial laparotomy, ICU BD, ICU peak IAP (mmHg), 24 h crystalloid volume, 24 h crystalloid, PRBC, and plasma volume
Intra-abdominal hypertension in the ICU					
Highest IAP in ICU before the first take-back, mmHg	Hatch[98]	242	Trauma	0.85 (0.76–0.95)	ISS, VAC dressing used at initial laparotomy, ICU INR, ICU BD, 24 h crystalloid volume, 24 h crystalloid, PRBC, and plasma volume
Delayed time to first take-back or requirement for multiple take-backs					
Time to first take-back, h	Pommerening[99]	499	Trauma	0.99 (0.98–1.00)	Abdomen AIS ≥3, bowel resection, postoperative antibiotics, intraoperative blood product volume, 24 h crystalloid volume >10 l, number of take-backs
Time to first take-back >48 h	Pommerening[99]	499	Trauma	0.53 (0.29–0.98)	NR
Time to first take-back >48 h in patients with an ISS >15	Pommerening[99]	499	Trauma	0.38 (0.20–0.74)	NR
No. of take-backs	Pommerening[99]	499	Trauma	0.18 (0.11–0.29)	Time to first take-back, abdomen AIS ≥3, bowel resection, postoperative antibiotics, intraoperative blood product volume, 24 h crystalloid volume >10 l
Administration of a large volume of crystalloid fluids in the first 24 h					
24 h crystalloid volume	Hatch[98]	242	Trauma	1.00 (1.00–1.00)	ISS, VAC dressing used at initial laparotomy, ICU INR, ICU BD, ICU peak IAP (mmHg), 24 h crystalloid volume, 24 h crystalloid, PRBC, and plasma volume
24 h crystalloid volume >10 l	Pommerening[99]	499	Trauma	0.51 (0.28–0.94)	Time to first take-back, abdomen AIS ≥3, bowel resection, postoperative antibiotics, intraoperative blood product volume, number of take-backs
Enteric fistula or intra-abdominal sepsis/infectious complications					
Enteric fistula	Dubose[97]	517	Trauma	0.16 (0.030–0.81)	No. of re-explorations, intra-abdominal abscess/sepsis, bloodstream infections, acute renal failure, ISS >15, age, gender, chest AIS, pH, lactate, estimated intraoperative blood loss, acidosis, intraoperative blood products, operating room fluid balance, number of packs used, small bowel resection, bowel left in discontinuity
Intra-abdominal abscess/sepsis	Dubose[97]	517	Trauma	0.42 (0.21–0.82)	No. of re-explorations, bloodstream infections, acute renal failure, enteric fistula, ISS >15, age, gender, chest AIS, pH, lactate, estimated intraoperative blood loss, acidosis, intraoperative blood products, operating room fluid balance, number of packs used, small bowel resection, bowel left in discontinuity

(continued)

Table 5 (continued)

Predictor	First author	No. of patients	Patient population	OR (95% c.i.)	Analyses adjusted for
Bloodstream infections	Dubose[97]	517	Trauma	0.38 (0.18–0.84)	No. of re-explorations, intra-abdominal abscess/sepsis, acute renal failure, enteric fistula, ISS >15, age, gender, chest AIS, pH, lactate, estimated intraoperative blood loss, acidosis, intraoperative blood products, operating room fluid balance, number of packs used, small bowel resection, bowel left in discontinuity
Miscellaneous					
Bowel resection in patients with an ISS >15	Pommerening[99]	499	Trauma	0.59 (0.35–0.98)	NR
Acute renal failure	Dubose[97]	517	Trauma	0.43 (0.22–0.84)	No. of re-explorations, intra-abdominal abscess/sepsis, bloodstream infections, enteric fistula, ISS >15, age, gender, chest AIS, pH, lactate, estimated intraoperative blood loss, acidosis, intraoperative blood products, operating room fluid balance, number of packs used, small bowel resection, bowel left in discontinuity

AIS, Abbreviated Injury Scale; BD, base deficit; BE, base excess; ED, emergency department; IAP, intra-abdominal pressure; ICU, intensive care unit; INR, International Normalized Ratio; ISS, Injury Severity Scale; NR, not recorded; OR, odds ratio; PATI, Penetrating Abdominal Trauma Index; PRBC, packed red blood cells; VAC, vacuum-assisted closure.

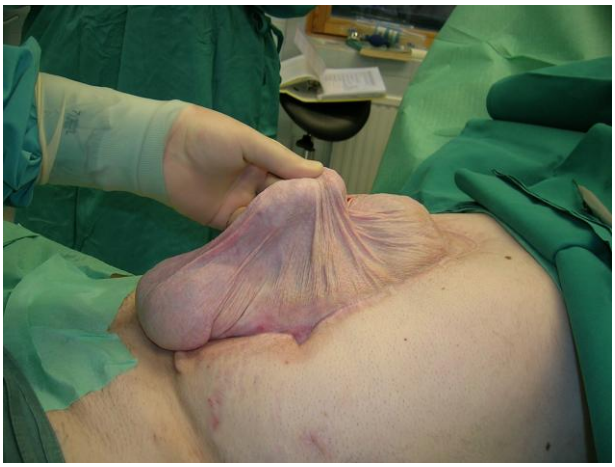

Fig. 7 Positive pinch test

often used. Anterior components separation involves the creation of flaps that extend superiorly to the costal margin, inferiorly to the inguinal ligament, and laterally to just beyond the linea semilunaris (lateral border of the rectus muscles) where the external oblique fascial release should be done[122,123]. Unfortunately, this is associated with several wound-related complications and fails to address hernias that are non-midline, subxiphoid, suprapubic, and parastomal as well as those associated with loss of domain[110]. For these reasons, some surgeons have transitioned to using posterior components separation with transverse abdominal release and mesh reinforcement, particularly for the hernias mentioned above[124–126]. In situations where abdominal wall reconstruction is not possible with a mesh and/or component separation, more advanced reconstructive techniques, such as a tensor fascia lata flap can be used[127,128]. The detailed description of this technique as well as its indications and associated outcomes are beyond the scope of this review but are available in the literature[127,128].

Mesh choice

The abdominal wall reconstruction may be reinforced using a synthetic, biosynthetic/bioabsorbable, or biological mesh (which may be non-cross-linked or cross-linked). In view of conflicting reports and possible industry bias, no definitive opinions as to the best mesh type to use in different situations can be confidently afforded. Some argue that a biological mesh is best used in the setting of a contaminated (infected abdomen or a concomitant ECF in acute settings when it presents as an intra-abdominal catastrophe) or potentially contaminated (grade III hernias, which includes the presence of stoma, violation of the GI tract, previous wound infection, and a concomitant ECF in a patient undergoing elective hernia repair) surgical field. Biological meshes may have a greater ability to be salvaged than prosthetic mesh. However, life-threatening biological mesh infections still occur, and there are dramatic cost differences despite no obvious outcome differences between different biological mesh products.

Further, recent evidence has suggested that prosthetic mesh can be used to repair ventral hernias under a spectrum of clinical circumstances with results that are either comparable or superior to those achieved using biologic mesh[129]. In a single-blind RCT of 165 adults undergoing ventral hernia repair, those allocated to biologic (porcine) instead of (polypropylene) mesh were reported to have a significantly higher rate of hernia recurrence at 2 years[129]. In a subgroup analysis, as compared with those in the synthetic mesh group, there was also a markedly higher rate of hernia recurrence in the biologic mesh group when the wound was contaminated[129]. In a subsequent multicentre, single-blinded RCT of 253 patients with clean-contaminated or contaminated ventral hernias, those allocated to retromuscular synthetic instead of biologic mesh were reported to have a significantly reduced hernia recurrence and 30-day hospital costs[130]. Despite this, there was no significant difference between surgical site infection requiring procedural intervention between the groups[130].

Finally, in some cases where the linea alba cannot be reconstructed, the fascial gap may need to be bridged. A systematic review and meta-analysis of non-randomized comparative studies reported that bridged repair had significantly higher odds of surgical site occurrences (defined as surgical site infection, dehiscence, haematoma, seroma, and site necrosis) and hernia recurrence compared with PFC[131]. Where the fascial gap needs to be bridged, absorbable materials

Table 6 International classification of abdominal wall anatomical planes[133]

Name	Anatomical description
A: Onlay	Anterior: subcutaneous tissue Posterior: anterior rectus sheath and external oblique
B: Anterectus anterior	Anterior: anterior rectus sheath Posterior: rectus abdominis muscle
C: Inlay	Mesh attached to edges of hernia defect with no overlap
D: Interoblique	Anterior: external oblique muscle Posterior: internal oblique muscle
E: Retro-oblique	Anterior: internal oblique muscle Posterior: transversus abdominis muscle
F: Retrorectus	Anterior: rectus abdominis muscle Posterior: posterior rectus sheath
H: Retromuscular (TAR performed)*	Anterior: rectus abdominis muscle (medial); transversus abdominis muscle (lateral) Posterior: posterior rectus sheath (medial; not present caudally, therefore caudal posterior border is transversalis fascia); transversalis fascia (lateral)
I: Transversalis fascial*	Anterior: posterior rectus sheath (medial; not present caudally, therefore caudal anterior border is rectus abdominis muscle); transversus abdominis muscle (lateral) Posterior: transversalis fascia (medial); transversalis fascia (lateral)
J: Preperitoneal	Anterior: transversalis fascia Posterior: peritoneum
K: Intraperitoneal	Anterior: peritoneum Posterior: abdominal cavity

*Below the arcuate line, planes H and I have the same anatomical location. TAR, transversus abdominis muscle release.

(synthetic or biological) will not suffice but instead result in unacceptable high hernia recurrence rates[131].

Mesh placement

There is debate regarding which abdominal plane the mesh should be placed within (see *Table 6* for a description of the International Classification of Abdominal Wall Anatomical Planes)[111,112]. Some surgeons place the mesh in the retrorectus plane for small- to medium-sized abdominal wall defects (≤10 cm); for larger defects, they use the retromuscular space with TAR[112]. Studies suggest that underlay and retrorectus mesh placements reduce hernia recurrence and seroma rates when compared with onlay and interposition mesh repairs[132].

Step 8: P = postoperative care

Patients should be monitored after surgery for increased IAP, venous thromboembolism, atelectasis, and pneumonia[113]. Increases in IAP after abdominal wall reconstruction requiring myofascial release are common but may be transient[134]. One cohort study of 50 patients by Petro *et al.* suggested that 92 per cent developed IAH; however, changes in IAP resolved by postoperative day one[134]. The authors therefore suggested that IAH should be considered 'permissive' in this context[134]. Venous thromboembolism prophylaxis and incentive spirometry should also be initiated early in the postoperative interval. Epidural catheters may be used for pain. Surgical drains should be kept in place until output is minimal.

Step 9: L = long-term follow-up

Patients undergoing abdominal wall reconstruction have a high risk of postoperative wound complications, including surgical site infection, dehiscence, seroma, and wound necrosis, and these complications are associated with poorer outcomes[114,115]. Surgical site infections secondary to resistant bacteria manifest more frequently postdischarge and require more frequent readmissions and reoperations[114]. Those with a history of prior abdominal infections, higher body mass index, enteric fistula take down, and increased duration of hospital stay have been reported to have a higher risk of surgical site infection or hospital readmission following complex abdominal wall reconstruction[114,135]. Most studies follow patients undergoing abdominal wall reconstruction for 2 years. Those who develop suspected wound complications or recurrent hernias during follow-up should be evaluated with abdominopelvic CT.

Conclusion

The OA is an innovation that spans across surgical specialties and has greatly improved surgical understanding of DC, TAC, STAR, viscera and enteric fistula care, and abdominal wall reconstruction. Subsequent stages of the innovation will likely involve the design and conduct of further valid prospective studies to evaluate appropriate indications for its use and effectiveness and safety of the above components of OA management.

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Author contributions

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Disclosure

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