

13 Intra-Abdominal Hypertension, Abdominal Compartment Syndrome and the Open Abdomen: Looking Beyond the Obvious to New Understandings in Pathophysiology, Harm-Reduction and Systemic Therapies

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13.1 Introduction

The abdominal organs are subject to marked functional changes due to alterations in both physical pressure and perfusion due to the nature of the viscera themselves, the tendency for inflammatory fluid to accumulate within this container, and even physical changes in the container and contiguous body cavities [\[1](#page-18-0)–[4\]](#page-18-1). The normal relaxed supine pressure within the peritoneal cavity, intraabdominal pressure (IAP), is under 10 mmHg [\[5](#page-18-2)], with 12 mmHg being defined as the beginning range of IAH [[6\]](#page-18-3). Many processes will increase the physical contents of the abdominal cavity such as ileus or obstruction of the hollow viscera or the accumulation of intraperitoneal fluids such as inflammatory ascites, enteric leakage, and/or hematoma. Finally, the container itself can be rendered non-compliant due to inflammation and resuscitation of the abdominal wall itself [\[7](#page-18-4)]. Ultimately when the abdominal contents are increased and especially if the abdominal compliance is decreased, the IAP will rise sometimes markedly [\[4,](#page-18-1) [8\]](#page-18-5).

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Thus, in any critical situation, IAH is common and associated with significant morbidity and mortality in critically ill and injured patients [\[1](#page-18-0), [8\]](#page-18-5). IAH can be somewhat simply equated with malperfusion. The abdominal compartment syndrome (ACS) represents the end of a pathophysiologic spectrum beginning with normal intra-abdominal pressure (IAP) and proceeding through worsening grades of IAH [[6\]](#page-18-3). By consensus, grades of progressive IAH are given as I–IV (Table [13.1](#page-1-0)) [\[6](#page-18-3), [9\]](#page-18-6). We prefer the term "overt ACS" to describe a catastrophically ill/injured patient with severe IAH and new-onset cardiorespiratory and/or renal failure. The effects of IAH/ACS are not limited to intra-abdominal organs; they are enacted systemically through biomediator generation resulting in multiorgan dysfunction syndrome/multisystem organ failure and/or through polycompartmental pressure interactions [\[2](#page-18-7), [6](#page-18-3), [10](#page-18-8)].

13.1.1 Consensus Definitions of the World Society of the Abdominal Compartment Syndrome

Early barriers to studying and thus understanding the IAH/ACS phenomenon related to variable definitions and concepts in the world literature frequently preclude comparison of data and experience. A notable milestone in defining and subsequently studying IAH/ACS was the establishment of the World Society of the Abdominal Compartment Syndrome ([wsacs.org\)](http://wsacs.org) in 2004 to "promote research, foster education, and improve the survival of patients with IAH and ACS" [[9,](#page-18-6) [11\]](#page-19-0). This group published expert consensus definitions relating to IAH/ACS in 2006 [[9](#page-18-6)], clinical practice guidelines in 2007 [\[11\]](#page-19-0), and recommendations for research methodology in 2009 [[12](#page-19-1)]. In 2013, they updated their consensus definitions (Table [13.2\)](#page-2-0) and clinical practice guidelines (Table [13.3](#page-3-0)) [\[6\]](#page-18-3). In these guidelines, a dedicated pediatric subcommittee evaluated the adult definitions for use among children [[6](#page-18-3)]. The subcommittee set the value used to define IAH and ACS in children lower as physiologic IAP values in these patients are lower than in adults [[6](#page-18-3), [13,](#page-19-2) [14\]](#page-19-3). This highlights that alternate definitions and management strategies may be needed for other patient populations, including pregnant women $[15]$, those with obesity $[16]$ or undergoing complex abdominal wall reconstruction [[17](#page-19-6)], and the elderly, which are areas requiring future research.

In an effort to maintain vigilance in preventing ACS, while emphasizing the need to better understand IAH and its relationship to abdominal wall physiology [[2,](#page-18-7) [18\]](#page-19-7), the group was rebranded as the "WSACS—The Abdominal Compartment Society"

Table 13.2 Final 2013 consensus definitions of the World Society of the Abdominal Compartment Syndrome

ACS abdominal compartment syndrome, *APP* abdominal perfusion pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure Reproduced from Kirkpatrick et al. [\[6\]](#page-18-3)

in 2015 [[19\]](#page-19-8). The mission of the Society was broadened to maintain the need to understand the optimal treatment of overt ACS but even more importantly to study IAH in all manner of its acute and chronic forms as an independent and a multifactorial condition in human disease and injury. Further in 2016, the WSACS collaborated closely with the World Society of Emergency Surgery to review contemporary data and to produce consensus guidance statements for OA management that are congruent and follow upon the WSACS/ACS guidelines for IAH/ACS management [\[20](#page-19-9), [21](#page-19-10)] (Table [13.4](#page-4-0)).

Table 13.3 Summarized consensus statement of the World Society of the Abdominal Compartment Syndrome

Recommendations

- 1. We recommend measuring IAP when any known risk factor for IAH/ACS is present in a critically ill or injured patient [GRADE 1C]
- 2. Studies should adopt the trans-bladder technique as the standard IAP measurement technique [not GRADED]
- 3. We recommend the use of protocolized monitoring and management of IAP versus not [GRADE 1C]
- 4. We recommend efforts and/or protocols to avoid sustained IAH as compared to inattention to IAP among critically ill or injured patients [GRADE 1C]
- 5. We recommend decompressive laparotomy in cases of overt ACS compared to strategies that do not use decompressive laparotomy in critically ill adults with ACS [GRADE 1D]
- 6. We recommend that among ICU patients with open abdominal wounds, conscious and/or protocolized efforts be made to obtain an early or at least same-hospital-stay abdominal fascial closure [GRADE 1D]

7. We recommend that among critically ill/injured patients with open abdominal wounds, strategies utilizing negative pressure wound therapy should be used versus not [GRADE 1C] *Suggestions*

- 1. We suggest that clinicians ensure that critically ill or injured patients receive optimal pain and anxiety relief [GRADE 2D]
- 2. We suggest brief trials of neuromuscular blockade as a temporizing measure in the treatment of IAH/ACS [GRADE 2D]
- 3. We suggest that the potential contribution of body position to elevated IAP be considered among patients with, or at risk of, IAH or ACS [GRADE 2D]
- 4. We suggest the liberal use of enteral decompression with nasogastric or rectal tubes when the stomach and colon are dilated in the presence of IAH/ACS [GRADE 1D]
- 5. We suggest that neostigmine be used for the treatment of established colonic ileus not responding to other simple measures and associated with IAH [GRADE 2D]
- 6. We suggest using a protocol to try and avoid a positive cumulative fluid balance in the critically ill or injured patient with, or at risk of, IAH/ACS after the acute resuscitation has been completed and the inciting issues have been addressed [GRADE 2C]
- 7. We suggest the use of an enhanced ratio of plasma/packed red blood cells for resuscitation of massive hemorrhage versus low or no attention to plasma/packed red blood cell ratios [GRADE 2D]
- 8. We suggest the use of PCD to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS when this is technically possible compared to doing nothing [GRADE 2C]. We also suggest using PCD to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS when this is technically possible compared to immediate decompressive laparotomy as this may alleviate the need for decompressive laparotomy [GRADE 2D]
- 9. We suggest that patients undergoing laparotomy for trauma suffering from physiologic exhaustion be treated with the prophylactic use of the open abdomen versus intraoperative abdominal fascial closure and expectant IAP management [GRADE 2D]
- 10. We suggest not to routinely utilize the open abdomen for patients with severe intraperitoneal contamination undergoing emergency laparotomy for intra-abdominal sepsis unless IAH is a specific concern [GRADE 2B]
- 11. We suggest that bioprosthetic meshes should not be routinely used in the early closure of the open abdomen compared to alternative strategies [GRADE 2D]
- *No recommendations*
	- 1. We could make no recommendation regarding the use of abdominal perfusion pressure in the resuscitation or management of the critically ill or injured

Table 13.3 (continued)

- 2. We could make no recommendation regarding the use of diuretics to mobilize fluids in hemodynamically stable patients with IAH after the acute resuscitation has been completed and the inciting issues have been addressed
- 3. We could make no recommendation regarding the use of renal replacement therapies to mobilize fluid in hemodynamically stable patients with IAH after the acute resuscitation has been completed and the inciting issues have been addressed
- 4. We could make no recommendation regarding the administration of albumin versus not to mobilize fluid in hemodynamically stable patients with IAH after acute resuscitation has been completed and the inciting issues have been addressed
- 5. We could make no recommendation regarding the prophylactic use of the open abdomen in non-trauma acute care surgery patients with physiologic exhaustion versus intraoperative abdominal fascial closure and expectant IAP management
- 6. We could make no recommendation regarding the use of an acute component separation technique versus not to facilitate earlier abdominal fascial closure, *ACS* abdominal compartment syndrome, *IAP* intra-abdominal pressure, *IAH* intra-abdominal hypertension, *PCD* percutaneous catheter drainage

Reproduced from Kirkpatrick et al. [\[6\]](#page-18-3)

(continued)

Table 13.4 (continued)

Table 13.4 (continued)

(continued)

Table 13.4 (continued)

Reproduced from Coccolini et al. [\[20\]](#page-19-9)

13.1.2 The Abdominal Compartment and Abdominal Compliance

The concept of abdominal compliance (AC) is critical to appreciate for emergency surgeons. Intra-abdominal pressure is the direct result of both the abdominal volume and the abdominal compliance [[2,](#page-18-7) [4](#page-18-1), [22\]](#page-19-11). The volume of the abdominal contents varies greatly with both physiological and pathophysiological conditions. The second paradigm-changing concept is the related appreciation that abdominal compliance is not fixed. Abdominal compliance is a dynamic property reflecting the underlying tissue properties and health of the abdominal wall, which also reflects the therapies administered to any patient in the inter- and perioperative periods [\[2](#page-18-7), [4](#page-18-1), [23\]](#page-19-12).

13.1.3 Pathophysiology

Although centered upon the abdominal cavity, the pathophysiology of IAH/ ACS affects the entire body physically and biochemically (Fig. [13.1\)](#page-8-0). Cardiac output is reduced owing to decreased preload and right heart volumes. Although increased systemic vascular resistance initially maintains apparent blood pressure, decreases in preload from the pooling of blood in splanchnic and lower extremity vascular beds eventually lead to reduced central venous return [\[24](#page-19-13)[–28\]](#page-19-14). Cardiac

CENTRAL NERVOUS SYSTEM

CARDIOVASCULAR SYSTEM¹ morbid obesity **RESPIRATORY SYSTEM** Intracranial pressure Cerebral perfusion pressure \downarrow Idiopathic intracranial hypertension in morbid obesity

Cardiac output \vee Left ventricular compliance $\mathsf{\downarrow}$ Left ventricle regional wall motion $\mathsf{\Psi}$ Venous return Pulmonary artery occlusion pressure Systemic vascular resistance Pulmonary artery pressure Venous thrombosis Pulmonary embolism Central venous pressure Transmural filling pressure = Δ Intra thoracic blood volume index = Δ Mean arterial pressure $7 = 4$ Extra vacular lung water $=$ 7 Heart rate π = Stroke volume variation π Pulse pressure variation π Difficult preload assessment Global enddiastolic blood volume index $=$ Δ Right ventricular end-diastolic volume = Δ

HEPATIC SYSTEM

Hepatic arterial flow \downarrow Portal venous blood flow \downarrow Lactate clearance \vee Glucose metabolism $\mathsf{\Psi}$ Mitochondrial function \downarrow Cytochrome p450 function \downarrow Indocyanine green Portocollateral flow 个 Plasma disappearance rate

GASTRO-INTESTINAL SYSTEM

Abdominal perfussion pressure \downarrow Celiac blood flow ψ Superior mesenteric artery blood flow \downarrow Blood flow to intra-abdominal organs $\mathsf{\Psi}$ Mucosal blood flow $\overline{\mathsf{v}}$ Intramucosal pH Success enteral feeding \downarrow Regional CO2 \uparrow Intestinal permeability Bacterial translocation Multiple organ failure Variceal wall stress Gastro-intestinal ulcer (re)bleeding Variceal (re)bleeding Peritoneal adhesions CO2-gap 个 Mesenteric vein compression

All lung volumes $\mathsf{\Psi}$ $PaO2 \nightharpoonup$ and PaO2/FiO2 \vee Lower inflection point \downarrow Static chest wall compliance \downarrow Static respiratory system compliance \downarrow Dynamic compliance \downarrow Auto-PEEP Peak airway pressure Plateau airway pressure Activated lung neutrophils Upper inflection point ↑ Dead-space ventilation Intrapulmonary shunt Hypercarbia 个 Extra vascular lung water $=$ π Intrathoracic pressure Pleural pressure \uparrow (~restrictive disease) Static lung compliance = Prolonged ventilation Difficult weaning Functional residual capacity

RENAL SYSTEM Compression atelectasis

Alveolar edema

Pulmonary inflammatory infiltration

Glomerular filtration rate \downarrow Diuresis \downarrow Renal blood flow \vee Filtration gradient \downarrow Renal perfusion pressure \downarrow \uparrow \uparrow Anti-diuretic hormone Compression ureters Renal vein compression \uparrow Renal vascular resistance Tubular dysfunction \uparrow Adrenal blood flow = Abdominal wall complications in CAPD

ABDOMINAL WALL

 Compliance Rectus sheath blood flow \downarrow Incisional hernia Wound complications \uparrow

ENDOCRINE SYSTEM Release pro-inflammatory cytokines (IL-Ib, TNF-a, IL-6)

1 Cardiovascular effects are exacerbated in case of hypovolemia, hemorrhage, ischemia and high PEEP ventilation

Fig. 13.1 Whole body effects of increased intra-abdominal pressure. Reproduced from Malbrain ML [[41](#page-20-0)]

underfilling also occurs despite apparently increased central hemodynamic measurements (central venous pressure and pulmonary artery occlusion pressure).

A distended tight abdomen with IAH physically compresses the lungs especially at the bases created a restrictive lung disease model. As respiratory compliance decreases, mechanical ventilation with increased ventilatory pressures and decreased volumes becomes difficult [\[25,](#page-19-15) [29](#page-19-16), [30](#page-19-17)]. The partial pressures of oxygen will decrease, and carbon dioxide will increase [\[30](#page-19-17), [31\]](#page-20-1). Even modest IAH appears to exacerbate acute lung injury and the acute respiratory distress syndrome (ARDS). When IAP levels greater than 20 mmHg are applied to critically ill animals, a dramatic exacerbation of ARDS-associated pulmonary edema is evident [\[30,](#page-19-17) [32](#page-20-2)]. Furthermore, elevated IAP results in a stiffer chest wall with much lower transpulmonary pressures and therefore less susceptibility to ventilator-induced lung injury [\[33,](#page-20-3) [34\]](#page-20-4).

Oliguria is a common manifestation of the ACS, noting that the degree of renal failure has a dose-dependant relationship with IAH [\[35](#page-20-5)[–37](#page-20-6)]. Further, these effects are exaggerated by hypovolemia and positive end-expiratory pressure [[31,](#page-20-1) [38\]](#page-20-7), and renal failure is often multifactorial in critical care settings. Blood flow to the kidney operates in series, with a high-pressure capillary bed in the glomerulus having a mean pressure of about 60 mmHg, although mean capillary pressure of the peritubular capillary system operates at a mean pressure of approximately 13 mmHg [[39\]](#page-20-8). Such pressure and flow relationships make the kidney's very susceptible to IAH, and the renal recovery after decompression may be dramatic [[40\]](#page-20-9).

Beyond the heart, lungs, and kidneys, almost every other organ system is altered by IAH, even if the effects are not clinically overt. IAH appears to contribute to increased intracerebral pressure (ICP) via transmitted intrathoracic pressure [[41](#page-20-0), [42](#page-20-10)] to the extent that laparotomies have been reported to reduce ICP in patients with secondary ACS [\[43](#page-20-11), [44](#page-20-12)]. Patients in shock are at a particularly high risk for splanchnic malperfusion because even modest elevations in IAP greatly reduce hepatic and splanchnic perfusion [\[45\]](#page-20-13). This effect is exacerbated by prior hemorrhage [\[46\]](#page-20-14) and is observed at much lower IAPs than required to induce other clinical features of ACS.

13.1.4 Pathobiology of IAH/ACS

Owing to intra-compartment physiology, there is a marked reduction to all the viscera inducing relative or actual organ ischemia. This ischemia initiates the inflammatory cascade of vasoactive biomediators common to sepsis. The effects of IAH on the gut are similar to those of prolonged hypoperfusion, and therefore these two issues are compounding. In the face of IAH, the damaged gut seems to act as a continued source of inflammation propagating SIRS and potentiating MODS [[47–](#page-20-15)[49\]](#page-20-16). Even after resuscitation and normalization of hemodynamics, gut vasoconstriction persists and is further exacerbated by IAH. Even relatively mild IAH (e.g., an IAP of 15 mmHg) has been reported to decrease intestinal microcirculatory blood flow, increase bowel wall permeability, and induce irreversible gut histopathological changes, bacterial translocation, and multiorgan dysfunction syndrome [[50–](#page-20-17)[52\]](#page-20-18). 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), proinflammatory cytokines, and other mediators [[53\]](#page-21-0). Thus, IAH may help transition severe injury/infection to subsequent MODS.

This process itself may be exacerbated by series of physiologic stresses associated with prior priming of the immune system elements, such that IAH/ACS will be potentiated due to sequential physiological "hits," which produce a selfperpetuating process termed the "acute intestinal distress syndrome" [\[54](#page-21-1), [55\]](#page-21-2). In the first hit, resuscitation of patients in shock induces injury especially of the splanchnic circulation [[50,](#page-20-17) [55](#page-21-2), [56](#page-21-3)]. This "acute bowel injury" results in release of pro-inflammatory mediators into the peritoneum and systemic circulation, leading to neutrophil priming, increased intestinal wall permeability, extravasation of fluid into the bowel wall and mesentery, translocation of intestinal bacteria, and absorption of bacterial endotoxin [[51,](#page-20-19) [57–](#page-21-4)[60\]](#page-21-5). In any subsequent hit such as a severe infection or delayed bleeding requiring further resuscitation, the resultant abdominal visceral edema leads to further IAH, compressing intra-abdominal lymphatics and resulting in a progressive visceral malperfusion, mucosa-to-serosa intestinal necrosis, a further increase in bowel wall permeability, and heightened bacterial translocation/ endotoxin absorption and release of pro-inflammatory mediators [[51,](#page-20-19) [57\]](#page-21-4). Such a two-hit theory may explain why patients without a primary inciting cause of shock (e.g., during elective abdominal wall reconstruction) may sometimes tolerate IAH/ ACS better than predicted [\[17](#page-19-6), [61\]](#page-21-6), if they do not suffer a secondary insult in the postoperative period.

13.1.5 Epidemiology of IAH/ACS in a Changing Playing Field

Although the incidence of IAH may have not changed substantially, that of overt postinjury ACS has markedly decreased presumably because of increased awareness and the use of prevention strategies [[62–](#page-21-7)[64\]](#page-21-8). These include damage control resuscitation and increasingly well-tolerated and effective methods of open abdominal management [\[62](#page-21-7), [64\]](#page-21-8). Damage control resuscitation is a strategy characterized by rapid hemorrhage control, permissive hypotension, administration of blood products in a ratio approximating whole blood (i.e., 1:1:1 packed red blood cells/ plasma/platelets), and minimization of crystalloid fluids [[65\]](#page-21-9). Such balanced resuscitation practices appear to be one the most profound evolutions in critical care/ trauma in the last several decades [\[66](#page-21-10)].

13.1.6 Diagnosis

A critical pitfall is assuming that IAH/ACS can be excluded clinically without measuring IAP. Clinical examination, however, is unfortunately insufficient for detecting raised IAP [[67,](#page-21-11) [68\]](#page-21-12). The current gold standard technique for diagnosis uses the urinary bladder for pressure transduction [\[6](#page-18-3)]. It is recommended that patients be supine with relaxed abdominal musculature in the end-expiratory phase of respiration and the transducer zeroed at the iliac crest in the midaxillary line [[6\]](#page-18-3). The requirement for supine positioning is often a logistical barrier to frequent measurements in the critically ill/injured and a potential liability regarding supine positioning. Thus, corrections that allow inference of the effective IAP without recumbency or understanding the implications of IAP measurement at the phlebostatic axis are attractive, but not yet widely implemented [[69–](#page-21-13)[71\]](#page-21-14).

13.2 Management of IAH/ACS

The updated 2013 consensus management statements and management algorithm of the WSACS are outlined in Table [13.3](#page-3-0) and Fig. [13.2](#page-11-0), respectively [[9\]](#page-18-6). These recommendations represent the best efforts of an International Collaboration led by the WSACS to update the previous definitions [[9\]](#page-18-6) and recommendations [\[11](#page-19-0)] based on scientific progress over a decade in studying IAH/ACS [\[72](#page-21-15)]. It is hoped that these guidelines will require frequent updating as new scientific evidence emerges from well-performed studies.

IAH / ACS MEDICAL MANAGEMENT ALGORITHM

Fig. 13.2 Abdominal Compartment Society Intra-Abdominal Hypertension/Abdominal Compartment Syndrome Management Algorithm. Reproduced from Kirkpatrick et al. [\[6](#page-18-3)]

13.2.1 Medical and Percutaneous Management

Several medical and minimally invasive management options for IAH and ACS exist. Although many have not been well studied, these should be instituted prior to surgical intervention where safe and feasible. The WSACS medical management algorithm is outlined in Fig. [13.3](#page-12-0) [[3\]](#page-18-9).

INTRA-ABDOMINAL HYPERTENSION (IAH)/ABDOMINAL COMPARTMENT SYNDROME (ACS) MANAGEMENT ALGORITHM

Fig. 13.3 Abdominal Compartment Society Intra-Abdominal Hypertension/Abdominal Compartment Syndrome Medical Management Algorithm. Reproduced from Kirkpatrick et al. [\[6\]](#page-18-3)

Medical management strategies may be broadly divided into those that may increase abdominal wall compliance (sedation/analgesia and neuromuscularblocking agents), evacuate gastrointestinal contents (nasogastric/rectal tubes and prokinetic agents), and decrease fluid balance [[6\]](#page-18-3). As ileus and a positive fluid balance are significant and potentially modifiable risk factors for IAH in critically ill adults [\[73](#page-21-16)], decompressing enteral tubes should be used in patients with gastrointestinal tract distention, and a positive patient fluid balance should be avoided after the acute resuscitation phase has been completed [\[6](#page-18-3)]. Damage control resuscitation should be adopted in managing trauma patients with significant hemorrhage as it has been reported to be associated with a lower incidence of ACS and higher primary fascial closure (i.e., same-hospital-stay abdominal fascia-to-fascia closure) rates after damage control laparotomy when compared to traditional, crystalloidfocused resuscitation [\[74](#page-22-0), [75](#page-22-1)]. Finally, although no studies have examined whether sedative or analgesic agents decrease IAP, neuromuscular-blocking agents are associated with a decrease in IAP and may be used in patients with ACS as a rescue treatment until another more definitive therapy can be performed [[6,](#page-18-3) [76\]](#page-22-2).

Percutaneous catheter drainage is a minimally invasive option suggested to decrease IAP in those with IAH/ACS [\[6](#page-18-3), [77](#page-22-3)]. This intervention has been reported to effectively reduce IAP among patients with burns/acute pancreatitis and drainable closure rates after damage control laparotomy when compared to traditional, crystalloid-focused resuscitation [[56,](#page-21-3) [57\]](#page-21-4). Percutaneous catheter drainage is a minimally invasive option suggested to decrease IAP in those with IAH/ACS [\[77](#page-22-3), [78\]](#page-22-4). This intervention has been reported to effectively reduce IAP among patients with burns/acute pancreatitis and drainable intraperitoneal fluid collections. A casecontrol study of 62 patients with IAH/ACS and free intraperitoneal fluid or blood also reported that percutaneous catheter drainage was as effective as decompressive laparotomy at decreasing IAP and may avoid need for abdominal decompression in up to 81% of patients [[79\]](#page-22-5). In this study, risk factors for percutaneous catheter drainage treatment failure included drainage of less than 1000 mL of fluid or a decrease in IAP of less than 9 mmHg in the first 4 h after catheter insertion [\[79](#page-22-5), [80](#page-22-6)].

13.2.2 Surgical Management of IAH/ACS

Case reports/series have recently examined whether one of a number of different minimally invasive fasciotomy methods may be used instead of decompressive laparotomy in patients with largely secondary causes of ACS [[77,](#page-22-3) [81,](#page-22-7) [82\]](#page-22-8). These studies have reported improvements in IAP and urine output [\[77](#page-22-3)]. Methods evaluated have included subcutaneous anterior rectus sheath fasciotomies, midline subcutaneous fasciotomy, bilateral subcutaneous anterior rectus abdominis muscle fasciotomy, subcutaneous or open linea alba fasciotomy, and midline subcutaneous fasciotomy [\[77](#page-22-3)].

If medical and less invasive strategies for treating IAH/ACS have failed, however, then decompressive laparotomy is lifesaving and should be expediently performed. From a realistic viewpoint, the surgical management of IAH/ACS can be functionally equated to managing the resultant OA with an overall goal of formally closing it as soon as it is safe. The entire potential spectrum of this management can be conceptualized as potentially occurring in up to six stages. These include surgical prevention of IAH/ACS; abdominal decompression (via laparotomy or a minimally invasive fasciotomy); temporary abdominal closure with a temporary abdominal closure (TAC) device; initial management of the open abdominal wound in the ICU; avoidance of wound complications, including deep soft tissue infections, abdominal abscesses, entero-atmospheric fistulae, and complex ventral herniae; and staged abdominal reconstruction (reducing and closing the abdominal defect over time) or, as a last resort, the use of a planned ventral hernia (an open abdominal wound that is allowed to granulate and covered with skin flaps or a split-thickness skin graft) with plans for delayed abdominal wall reconstruction.

It should be emphasized that the contemporary surgeons' obvious goal should be to avoid all these stages if possible, or to minimize them, yet ensuring that the patient survives. In this current era of aggressive non-operative management, it is not a success if the patients succumb due to lack of an intervention. If prophylaxis or medical therapy can avoid or mitigate IAH, then no other interventions may be required. After decompressive laparotomy therefore, primary fascial closure is the goal. IAH/ACS may potentially be completely prevented after laparotomy by leaving the abdomen open where appropriate. However, for surgeons to recognize "when it's appropriate" however is a challenging target within a rapidly moving playing field. While a decade ago surgeons would accept that almost any seriously injured patient requiring a laparotomy would subsequently require postoperative OA to prevent postinjury ACS [[83\]](#page-22-9), this dictum is no longer assured related to rationalized resuscitation strategies. As there is a vacuum of scientific data, opinion is the highest level of guidance. A recent expert appropriateness rating study concluded that appropriate indications for OA use remain the development of a coagulopathy (especially when combined with hypothermia and acidosis), administration of large volumes of crystalloids or packed red blood cells, inability to close the abdominal fascia without tension, development of signs of ACS during attempted abdominal wall closure, and need for a planned relaparotomy to remove intra-abdominal packs or reassess extent of bowel viability [[84–](#page-22-10)[86\]](#page-22-11).

To assist clinicians the World Society of Emergency Surgery in conjunction with the Abdominal Compartment Society recently published consensus guidelines on managing the open abdomen [\[20](#page-19-9), [21](#page-19-10), [87](#page-22-12)]. The summarized recommendations regarding management are presented in Table [13.4.](#page-4-0)

13.2.3 Temporary Abdominal Closure (TAC) Devices

It is implicit in now accepting an OA that an acceptable and safe temporary abdominal closure (TAC) device be utilized to protect the viscera and manage the peritoneal cavity. Current WSACS/ACS and WSES guidelines recommend the use of negative pressure peritoneal therapy (NPPT) with either noncommercial (i.e., the Barker's vacuum pack) or commercial active negative pressure wound therapy devices be used for temporary abdominal closure [\[6](#page-18-3), [20](#page-19-9)]. Other contemporary reviews have also suggested the use of fascial traction methods in addition to NPPT, in a technique known as vacuum-assisted wound closure and mesh-mediated fascial traction (VAWCM) [\[88](#page-22-13)[–90](#page-22-14)], is the preferred therapy [\[91](#page-22-15)], although there is minimal controlled evidence to base decision-making upon.

While open abdomen therapy was described as far back as 1940 using light canvas covered in Vaseline [\[92](#page-22-16)], there has been a rapid evolution in TAC devices. Using plastic bags that did not adhere to the viscera was an advantage of the so-called Bogota bag. Subsequently a number of important principles regarding TAC devices have been recognized. The TAC should be placed well within the peritoneal cavity to maintain as much lateral separation of the viscera from the abdominal wall. It should prevent lateralization of the abdominal musculature which is an important function of VAWCM. It should preferably control peritoneal fluids, which may be one of its most unappreciated benefits.

Preclinical studies have reported that application of negative pressure to the open abdominal wound (i.e., "negative pressure peritoneal therapy") [[93](#page-22-17)] may remove ascites and peritoneal pro-inflammatory mediators, reduce the systemic inflammatory response, and improve the structure and potentially function of the pulmonary, cardiac, and renal systems [[94](#page-23-0), [95\]](#page-23-1). A prospective cohort study of 280 patients published in 2013 reported that the use of the ABThera negative pressure wound therapy device (Kinetic Concepts Inc., San Antonio, Texas, USA) was associated with improved primary fascial closure rates and survival when compared with a device that provides potentially less efficient peritoneal negative pressure, the Barker's vacuum pack [[96](#page-23-2)]. To date, only one RCT published in 2015 has been designed to determine if the ABThera is more efficacious than the Barker's vacuum pack at reducing the extent of the systemic inflammatory response after damage control laparotomy for intra-abdominal injury or sepsis [[97](#page-23-3)]. This trial reported an improved survival with the ABThera that did not appear to be mediated by an improvement in peritoneal fluid drainage, markers of the systemic inflammatory response, or primary fascial closure rates [[97](#page-23-3)].

After decompressive laparotomy, if the abdominal fascia is unable to be closed when the patient is first returned to the operating room, a staged abdominal reconstruction method may be used. These methods have been proposed to include negative pressure peritoneal therapy, the Wittmann Patch (Starsurgical, Burlington, Wisconsin, USA), progressive closure of a synthetic patch sutured between the fascial edges, dynamic retention using sutures or the Abdominal Reapproximation Anchor device (Canica Design Inc., Almonte, Ontario, Canada), and vacuumassisted wound closure (VAC) and mesh-mediated fascial traction. Although no comparative trials have been completed, VAC and mesh-mediated fascial traction has likely received the greatest enthusiasm as it is associated with fascial closure rates of at least 77% [[88,](#page-22-13) [91,](#page-22-15) [98\]](#page-23-4). In this method, patients are fitted with a VAC device at the initial laparotomy [\[88](#page-22-13), [99\]](#page-23-5). At relaparotomy, a perforated polyurethane sheet is placed over the visceral block and a divided polypropylene mesh sheet sutured between the fascial edges. This sheet is subsequently sutured together before a VAC dressing is placed atop and negative suction applied. The VAC dressing is then changed and mesh progressively tightened every 24–72 h until the fascial edges can be reapproximated.

13.2.4 Direct Peritoneal Resuscitation

DPR involves infusion of hypertonic fluid into the abdomen in addition to IV resuscitation. This causes rapid and sustained dilation of the arterioles, especially those in the intestine, which reduces organ ischemia and cellular hypoxia [\[100](#page-23-6), [101\]](#page-23-7). Data from single-center RCTs shows that NPWT and fluid instillation seem to improve outcomes in trauma patients in terms of early and primary closure [[102,](#page-23-8) [103](#page-23-9)]. There is a good deal of animal work supporting the conclusion that DPR prevents intestinal ischemia and helps preserve intestinal blood flow and structural integrity and reduces inflammatory cytokines even in inflammatory states such as brain death [\[101](#page-23-7), [104](#page-23-10)]. With replication of these experiences in other centers, this therapy may become part of the standard OA management.

13.2.5 Current Utilization of the Open Abdomen (OA)

13.2.5.1 The OA for Trauma Surgery

The use of the OA in trauma surgery is decreasing every year. With a dramatic evolution in resuscitation practices involving balanced resuscitation practices, more and more trauma patients who previously become so edematous required OA therapy, are no longer being crystalloid over-resuscitated, and can thus be primarily closed [[64,](#page-21-8) [66,](#page-21-10) [105](#page-23-11)]. This dramatic change in the trauma care paradigm has justified questions regarding the whole premise of damage control surgery for trauma [\[63](#page-21-17)] and justifies the randomized control trial of the practice in trauma patients [\[106](#page-23-12)].

13.2.5.2 The OA for Intra-abdominal Sepsis

The use of the OA for non-trauma general surgery is however increasingly being reported in uncontrolled series as a potentially beneficial option for patients with SCIAS [[107–](#page-23-13)[112\]](#page-23-14). The use of the OA in severe sepsis may allow early identification and increased drainage of any residual infection, control any persistent source of infection, more effectively remove biomediator-rich peritoneal fluid, provide prophylaxis against development of the abdominal compartment syndrome, and allow for the safe deferral of gastrointestinal anastomoses in settings where the risk of anastomotic leak is initially high [\[111](#page-23-15)]. Although the WSACS/ACS guidelines recommended NOT to use the open abdomen for intra-abdominal sepsis [[6\]](#page-18-3), largely based on economic reasons [[113\]](#page-23-16), more contemporary WSES guidelines differ. The 2018 WSES guidelines on OA management state that the open abdomen is an option for emergency surgery patients with 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 ischemia, persistent source of peritonitis (failure of source control), or extensive visceral edema with the concern for development of abdominal compartment syndrome, albeit with the lowest confidence due to the level of evidence (Grade 2C) [\[20](#page-19-9)].

Compared to trauma patients, however, patients undergoing OA management for intra-abdominal sepsis have a greater risk of OA complications, including enteroatmospheric fistula (EAF) and intra-abdominal abscess formation, and a lower rate of primary fascial closure (i.e., fascia-to-fascia closure within the index hospitalization) [[87,](#page-22-12) [91](#page-22-15), [111](#page-23-15), [114,](#page-24-0) [115](#page-24-1)]. Risk factors for frozen abdomen and EAF in OA are delayed abdominal closure, non-protection of bowel loops during OA, the presence of bowel injury and repairs or anastomosis, colon resection during DCS, the large fluid resuscitation volume (>5 L/24 h), the presence of intra-abdominal sepsis/ abscess, and the use of polypropylene mesh directly over the bowel [[116–](#page-24-2)[120\]](#page-24-3). Although RCT data comparing techniques is needed, meta-analyses conducted by our group [\[121](#page-24-4)] and the Amsterdam group [[91\]](#page-22-15) have concluded that negative pressure wound therapy (NPWT) treatment appears to potentially be the safest and most effective OA management technique currently available. Newer commercial active negative pressure peritoneal therapy (ANPPT) systems now available for OA may reduce the risk of enterocutaneous fistula and facilitate enhanced delivery of negative peritoneal pressure to the peritoneal cavity [[1,](#page-18-0) [6,](#page-18-3) [121\]](#page-24-4).

Animal studies [\[94](#page-23-0)] and in silico modeling of these animal studies [[95\]](#page-23-1) have shown that ANNPT provides a greater degree of negative pressure throughout the peritoneum, which may reduce plasma biomediator levels when compared to a more passive peritoneal drainage. Systemic inflammation (TNF-α, IL-1β, IL-6) in one study was significantly reduced in the ANPPT group and was associated with significant improvement in intestine, lung, kidney, and liver histopathology [[94\]](#page-23-0). Although the mortality rate in the NPPT was 17% versus 50% in the control group, this difference was not statistically significant ($p = 0.19$), likely due to the smaller numbers.

As over-resuscitation becomes rare and de-resuscitation becomes a focus [[122\]](#page-24-5), it is intuitive that there will be more abdomens in non-trauma intra-abdominal sepsis patients who may be technically closed without inducing intra-abdominal hypertension (IAH). However, although these abdomens *may* be closed, *should* they be closed? As has been recently emphasized, there are profound differences in the basic science of sepsis and traumatic injury [\[123](#page-24-6)], with the previously unifying concepts of noninfectious systemic inflammatory response syndrome (SIRS) being effectively discarded as a clinically helpful construct [\[124](#page-24-7)[–126](#page-24-8)]. The one nebulous, poorly defined "holy grail" of the optimal management of SCIAS is adequate "source control." It is suggested that even if an abdomen can be physically closed that there may be an advantage to leaving it open to allow better drainage of intraperitoneal contamination, a concept that is supported by remarkable animal lab data suggests the ability of ANPPT to mitigate the elaboration of the inflammatory biomediator cascade [\[94](#page-23-0), [95](#page-23-1), [127](#page-24-9)]. Coupled with technical advances in ANPPT dressings that are safer to utilize and that increasingly protect the viscera, this appears an attractive option for the sickest IAS patients.

13.3 Conclusion

The use of the OA after source control laparotomies for intraperitoneal sepsis is increasingly being adopted without strong controlled evidence to its effectiveness. This has been partially supported by developments in TAC devices that offer greater safety and potentially even a therapeutic modality to mitigate the biomediator propagation leading to systemic inflammation in IAS. Thus, controlled studies to determine optimal therapies are urgently required. The surgical and critical care communities must therefore design RCTs to re-examine whether negative pressure wound therapy improves outcomes over alternate temporary abdominal closure methods in critically ill adults (and determine how this occurs), to determine the optimal method of staged abdominal reconstruction in patients with open abdominal wounds, and to study the role of IAH in critical care both as an independent and how it interacts in a multifactorial way with other physiological stressors in critical illness and injury. Thus, the next decade of study related to IAH/ACS will therefore be one aimed at understanding which treatments may effectively lower IAP and whether these treatments ultimately influence patient important outcomes.

References

- 1. Roberts DJ, Ball CG, Kirkpatrick AW. Increased pressure within the abdominal compartment: intra-abdominal hypertension and the abdominal compartment syndrome. Curr Opin Crit Care. 2016;22(2):174–85.
- 2. Malbrain ML, De Laet I, De Waele JJ, Sugrue M, Schachtrupp A, Duchesne J, et al. The role of abdominal compliance, the neglected parameter in critically ill patients - a consensus review of 16. Part 2: measurement techniques and management recommendations. Anaesthesiol Intensive Ther. 2014;46(5):406–32.
- 3. Malbrain ML, Peeters Y, Wise R. The neglected role of abdominal compliance in organ-organ interactions. Crit Care. 2016;20:67.
- 4. Blaser AR, Bjorck M, De Keulenaer B, Regli A. Abdominal compliance: a bench-to-bedside review. J Trauma Acute Care Surg. 2015;78(5):1044–53.
- 5. Sanchez NC, Tenofsky PL, Dort JM, Shen LY, Helmer SD, Smith RS. What is normal intraabdominal pressure? Am Surg. 2001;67:243–8.
- 6. Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med. 2013;39(7):1190–206.
- 7. Kirkpatrick AW, Pelosi P, De Waele JJ, Malbrain ML, Ball CG, Meade MO, et al. Clinical review: intra-abdominal hypertension: does it influence the physiology of prone ventilation? Crit Care. 2010;14(4):232.
- 8. Maddison L, Starkopf J, Reintam Blaser A. Mild to moderate intra-abdominal hypertension: does it matter? World J Crit Care Med. 2016;5(1):96–102.
- 9. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med. 2006;32:1722–32.
- 10. Kirkpatrick AW, Sugrue M, McKee JL, Pereira BM, Roberts DJ, De Waele JJ, et al. Update from the Abdominal Compartment Society (WSACS) on intra-abdominal hypertension and abdominal compartment syndrome: past, present, and future beyond Banff 2017. Anaesthesiol Intensive Ther. 2017;49(2):83–7.
- 11. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. Intensive Care Med. 2007;33(6):951–62.
- 12. De Waele JJ, Cheatham ML, Malbrain ML, Kirkpatrick AW, Sugrue M, Balogh Z, et al. Recommendations for research from the International Conference of Experts on Intraabdominal Hypertension and Abdominal Compartment Syndrome. Acta Clin Belg. 2009;64(3):203–9.
- 13. Ejike JC, Bahjri K, Mathur M. What is the normal intra-abdominal pressure in critically ill children and how should we measure it? Crit Care Med. 2008;36(7):2157–62.
- 14. De Waele JJ, Ejike JC, Leppaniemi A, De Keulenaer BL, De Laet I, Kirkpatrick AW, et al. Intra-abdominal hypertension and abdominal compartment syndrome in pancreatitis, paediatrics, and trauma. Anaesthesiol Intensive Ther. 2015;47(3):219–27.
- 15. Sawchuck DJ, Wittmann BK. Pre-eclampsia renamed and reframed: intra-abdominal hypertension in pregnancy. Med Hypotheses. 2014;83(5):619–32.
- 16. Malbrain ML, De Keulenaer BL, Oda J, De Laet I, De Waele JJ, Roberts DJ, et al. Intraabdominal hypertension and abdominal compartment syndrome in burns, obesity, pregnancy, and general medicine. Anaesthesiol Intensive Ther. 2015;47(3):228–40.
- 17. Petro CC, Raigani S, Fayezizadeh M, Rowbottom JR, Klick JC, Prabhu AS, et al. Permissive intra-abdominal hypertension following complex abdominal wall reconstruction. Plast Reconstr Surg. 2015;137(4):762e–4e.
- 18. Malbrain ML, Roberts DJ, De Laet I, De Waele JJ, Sugrue M, Schachtrupp A, et al. The role of abdominal compliance, the neglected parameter in critically ill patients - a consensus review of 16. Part 1: definitions and pathophysiology. Anaesthesiol Intensive Ther. 2014;46(5):392–405.
- 19. Kirkpatrick AW, De Waele JJ, De Laet I, De Keulenaer BL, D'Amours S, Bjorck M, et al. WSACS - The Abdominal Compartment Society. A society dedicated to the study of the physiology and pathophysiology of the abdominal compartment and its interactions with all organ systems. Anaesthesiol Intensive Ther. 2015;47(3):191–4.
- 20. Coccolini F, Roberts D, Ansaloni L, Ivatury R, Gamberini E, Kluger Y, et al. The open abdomen in trauma and non-trauma patients: WSES guidelines. World J Emerg Surg. 2018;13:7.
- 21. Coccolini F, Montori G, Ceresoli M, Catena F, Moore EE, Ivatury R, et al. The role of open abdomen in non-trauma patient: WSES Consensus Paper. World J Emerg Surg. 2017;12:39.
- 22. Kirkpatrick AW, Keaney M, Hemmelgarn B, Zhang J, Ball CG, Groleau M, et al. Intraabdominal pressure effects on porcine thoracic compliance in weightlessness: implications for physiologic tolerance of laparoscopic surgery in space. Crit Care Med. 2009;37(2):591–7.
- 23. Malbrain ML, Roberts DJ, Sugrue M, De Keulenaer BL, Ivatury R, Pelosi P, et al. The polycompartment syndrome: a concise state-of-the-art review. Anaesthesiol Intensive Ther. 2014;46(5):433–50.
- 24. Cothren CC, Moore EE, Johnson JL, Moore JB. Outcomes in surgical versus medical patients with secondary abdominal compartment syndrome. Am J Surg. 2007;194:804–8.
- 25. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med. 1989;17:118–21.
- 26. Barnes GE, Laine GA, GIam PY, Smith EE, Granger HJ. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol. 1985;248:208–13.
- 27. Malbrain ML. Intra-abdominal pressure in the intensive care unit: clinical tool or toy? In: JL V, editor. Yearbook of intensive care and emergency medicine. Berlin: Springer; 2002. p. 792–814.
- 28. Malbrain ML, de Laet I. Functional hemodynamics and increased intra-abdominal pressure: same thresholds for different conditions ...? Crit Care Med. 2009;37(2):781–3.
- 29. Meldrum DR, Moore FA, Moore EE, Haenel JB, Cosgriff N, Burch JM. Cardiopulmonary hazards of perihepatic packing for major liver injuries. Am J Surg. 1995;170:537–42.
- 30. Pelosi P, Quintel M, Malbrain ML. Effect of intra-abdominal pressure on respiratory mechanics. Acta Clin Belg Suppl. 2007;62(1):78–88.
- 31. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intraabdominal pressure. J Surg Res. 1976;20:401–4.
- 32. Quintel M, Pelosi P, Caironi P, Meinhardt JP, Luecke T, Herrmann P, et al. An increase of abdominal pressure increases pulmonary edema in oleic acid-induced lung injury. Am J Respir Crit Care Med. 2004;169(4):534–41.
- 33. Kirkpatrick AW, Meade MO, Mustard RA, Stewart TE. Strategies of invasive ventilatory support in ARDS. Shock. 1996;6:S17–22.
- 34. Gattinoni L, Chiumello D, Carlesso E, Valenza F. Bench-to-bedside review: chest wall elastance in acute lung injury/acute respiratory distress syndrome patients. Crit Care. 2004;8:350–5.
- 35. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg. 1999;134:1082–5.
- 36. De Waele JJ, De Laet I, Kirkpatrick AW, Hoste E. Intra-abdominal hypertension and abdominal compartment syndrome. Am J Kidney Dis. 2011;57(1):159–69.
- 37. Mohmand H, Goldfarb S. Renal dysfunction associated with intra-abdominal hypertension and the abdominal compartment syndrome. J Am Soc Nephrol. 2011;22(4):615–21.
- 38. Kashtan J, Green JF, Parsons EQ, Holcroft JW. Hemodynamic effects of increased abdominal pressure. J Surg Res. 1981;30:249–55.
- 39. Guyton AC. Formation of urine by the kidney: I. Renal blood flow, glomerular filtration, and their control. In: Guyton AC, editor. Textbook of medical physiology. 8th ed. Philadelphia, PA: WB Saunders; 1991. p. 286–307.
- 40. McBeth PB, Dunham M, Ball CG, Kirkpatrick AW. Correct the coagulopathy and scoop it out: complete reversal of anuric renal failure through the operative decompression of extraperitoneal hematoma-induced abdominal compartment syndrome. Case Rep Med. 2012;2012:946103.
- 41. Malbrain ML. Is it wise not to think about intraabdominal hypertension in the ICU? Curr Opin Crit Care. 2004;10:132–45.
- 42. Citerio G, Vascotto E, Villa F, Celotti S, Pesenti A. Induced abdominal compartment syndrome increases intracranial pressure in neurotrauma patients: a prospective study. Crit Care Med. 2001;29:1466–71.
- 43. Miglietta MA, Salzano LJ, Chiu WC, Scalea TM. Decompressive laparotomy: a novel approach in the management of severe intracranial hypertension. J Trauma. 2003;55:551–5.
- 44. Joseph DK, Dutton RP, Aarabi B, Scalea TM. Decompressive laparotomy to treat intractable intracranial hypertension after traumatic brain injury. J Trauma. 2004;57:687–95.
- 45. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intra-abdominal pressure. J Surg Res. 1987;43:14–20.
- 46. Friedlander MH, Simon RJ, Ivatury R, DiRaimo R, Machiedo GW. Effect of hemorrhage on superior mesenteric artery flow during increased intra-abdominal pressures. J Trauma. 1998;45(3):433–89.
- 47. Marshall JC. Inflammation, coagulopathy, and the pathogenesis of multiple organ dysfunction, syndrome. Crit Care Med. 2001;29:S99–S106.
- 48. Johnson D, Mayers I. Multiple organ dysfunction syndrome: a narrative review. Can J Surg. 2001;48:502–9.
- 49. Fink MP, Delude RL. Epithelial barrier dysfunction: a unifying theme to explain the pathogenesis of multiple organ dysfunction at the cellular level. Crit Care Clin. 2005;21:177–96.
- 50. Cheng J, Wei Z, Liu X, Li X, Yuan Z, Zheng J, et al. The role of intestinal mucosa injury induced by intra-abdominal hypertension in the development of abdominal compartment syndrome and multiple organ dysfunction syndrome. Crit Care. 2013;17(6):R283.
- 51. Leng Y, Zhang K, Fan J, Yi M, Ge Q, Chen L, et al. Effect of acute, slightly increased intraabdominal pressure on intestinal permeability and oxidative stress in a rat model. PLoS One. 2014;9(10):e109350.
- 52. Kirkpatrick AW, Roberts DJ, De Waele J, Laupland K. Is intra-abdominal hypertension a missing factor that drives multiple organ dysfunction syndrome? Crit Care. 2014;18(2):124.
- 53. Timmermans K, Kox M, Scheffer GJ, Pickkers P. Danger in the intensive care unit: damps in critically ill patients. Shock (Augusta, Ga). 2016;45(2):108–16.
- 54. Malbrain ML, De Laet I. AIDS is coming to your ICU: be prepared for acute bowel injury and acute intestinal distress syndrome. Intensive Care Med. 2008;34(9):1565–9.
- 55. Malbrain ML, Vidts W, Ravyts M, De Laet I, De Waele J. Acute intestinal distress syndrome: the importance of intra-abdominal pressure. Minerva Anestesiol. 2008;74(11):657–73.
- 56. Shah SK, Jimenez F, Letourneau PA, Walker PA, Moore-Olufemi SD, Stewart RH, et al. Strategies for modulating the inflammatory response after decompression from abdominal compartment syndrome. Scand J Trauma Resusc Emerg Med. 2012;20:25.
- 57. Carr JA. Abdominal compartment syndrome: a decade of progress. J Am Coll Surg. 2013;216(1):135–46.
- 58. Diebel LN, Dulchavsky SA, Brown WJ. Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma. 1997;43:852–5.
- 59. Shah SK, Jimenez F, Walker PA, Aroom KR, Xue H, Feeley TD, et al. A novel mechanism for neutrophil priming in trauma: potential role of peritoneal fluid. Surgery. 2010;148(2):263–70.
- 60. Shah SK, Jimenez F, Walker PA, Xue H, Feeley TD, Uray KS, et al. Peritoneal fluid: a potential mechanism of systemic neutrophil priming in experimental intra-abdominal sepsis. Am J Surg. 2012;203(2):211–6.
- 61. Kirkpatrick AW, Nickerson D, Roberts DJ, Rosen MJ, McBeth PB, Petro CC, et al. Intraabdominal hypertension and abdominal compartment syndrome after abdominal wall reconstruction: quaternary syndromes? Scand J Surg. 2016;106(2):97–106.
- 62. Balogh ZJ, Lumsdaine W, Moore EE, Moore FA. Postinjury abdominal compartment syndrome: from recognition to prevention. Lancet. 2014;384(9952):1466–75.
- 63. Schreiber MA. The beginning of the end for damage control surgery. Br J Surg. 2012;99(Suppl 1):10–1.
- 64. Joseph B, Zangbar B, Pandit V, Vercruysse G, Aziz H, Kulvatunyou N, et al. The conjoint effect of reduced crystalloid administration and decreased damage-control laparotomy use in the development of abdominal compartment syndrome. J Trauma Acute Care Surg. 2014;76(2):457–61.
- 65. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015;313(5):471–82.
- 66. Cantle PM, Cotton BA. Balanced resuscitation in trauma management. Surg Clin North Am. 2017;97(5):999–1014.
- 67. Kirkpatrick AW, Brenneman FD, McLean RF, Rapanos T, Boulanger BR. Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients. Can J Surg. 2000;43:207–11.
- 68. Sugrue M, Bauman A, Jones F, Bishop G, Flabouris A, Parr M, et al. Clinical examination is an inaccurate predictor of intraabdominal pressure. World J Surg. 2002;26:1428–31.
- 69. Cheatham ML, De Waele JJ, De Laet I, De Keulenaer B, Widder S, Kirkpatrick AW, et al. The impact of body position on intra-abdominal pressure measurement: a multicenter analysis. Crit Care Med. 2009;37(7):2187–90.
- 70. De Waele JJ, De Laet I, De Keulenaer B, Widder S, Kirkpatrick AW, Cresswell AB, et al. The effect of different reference transducer positions on intra-abdominal pressure measurement: a multicenter analysis. Intensive Care Med. 2008;34(7):1299–303.
- 71. McBeth PB, Zygun DA, Widder S, Cheatham M, Zengerink I, Glowa J, et al. Effect of patient positioning on intra-abdominal pressure monitoring. Am J Surg. 2007;193:644–7.
- 72. Kirkpatrick AW, Roberts DJ, Jaeschke R, De Waele JJ, De Keulenaer BL, Duchesne J, et al. Methodological background and strategy for the 2012-2013 updated consensus definitions and clinical practice guidelines from the abdominal compartment society. Anaesthesiol Intensive Ther. 2015;47:s63–77.
- 73. Holodinsky JK, Roberts DJ, Ball CG, Reintam Blaser A, Starkopf J, Zygun DA, et al. Risk factors for intra-abdominal hypertension and abdominal compartment syndrome among adult intensive care unit patients: a systematic review and meta-analysis. Crit Care. 2013;17(5):R249.
- 74. Cotton BA, Au BK, Nunez TC, Gunter OL, Robertson AM, Young PP. Predefined massive transfusion protocols are associated with a reduction in organ failure and postinjury complications. J Trauma. 2009;66(1):41–8.. discussion 8-9.
- 75. Ball CG, Dente CJ, Shaz B, Wyrzykowski AD, Nicholas JM, Kirkpatrick AW, et al. The impact of a massive transfusion protocol $(1,1,1)$ on major hepatic injuries: does it increase abdominal wall closure rates? Can J Surg. 2013;56(5):E128–34.
- 76. De Laet I, Hoste E, Verholen E, De Waele JJ. The effect of neuromuscular blockers in patients with intra-abdominal hypertension. Intensive Care Med. 2007;33(10):1811–4.
- 77. Ouellet JF, Leppaniemi A, Ball CG, Cheatham ML, D'Amours S, Kirkpatrick AW. Alternatives to formal abdominal decompression. Am Surg. 2011;77(Suppl 1):S51–7.
- 78. Wang T, Liu LY, Luo H, Dai RW, Liang HY, Chen T, et al. Intra-abdominal pressure reduction after percutaneous catheter drainage is a protective factor for severe pancreatitis patients with sterile fluid collections. Pancreas. 2016;45(1):127–33.
- 79. Cheatham ML, Safcsak K. Percutaneous catheter decompression in the treatment of elevated intraabdominal pressure. Chest. 2011;140(6):1428–35.
- 80. Peng T, Dong LM, Zhao X, Xiong JX, Zhou F, Tao J, et al. Minimally invasive percutaneous catheter drainage versus open laparotomy with temporary closure for treatment of abdominal compartment syndrome in patients with early-stage severe acute pancreatitis. J Huazhong Univ Sci Technolog Med Sci. 2016;36(1):99–105.
- 81. Leppaniemi A, Hienonen P, Mentula P, Kemppainen E. Subcutaneous linea alba fasciotomy, does it really work? Am Surg. 2011;77(1):99–102.
- 82. Leppaniemi AK, Hienonen PA, Siren JE, Kuitunen AH, Lindstrom OK, Kemppainen EA. Treatment of abdominal compartment syndrome with subcutaneous anterior abdominal fasciotomy in severe acute pancreatitis. World J Surg. 2006;30:1922–4.
- 83. Johnson JW, Gracias VH, Schwab CW, Reilly PM, Kauder DR, Shapiro MB, et al. Evolution in damage control for exsanguinating penetrating abdominal injury. J Trauma. 2001;51:261–71.
- 84. Roberts DJ, Bobrovitz N, Zygun DA, Ball CG, Kirkpatrick AW, Faris PD, et al. Indications for use of damage control surgery in civilian trauma patients: a content analysis and expert appropriateness rating study. Ann Surg. 2015;263(5):1018–27.
- 85. Roberts DJ, Bobrovitz N, Zygun DA, Ball CG, Kirkpatrick AW, Faris PD, et al. Indications for use of thoracic, abdominal, pelvic, and vascular damage control interventions in trauma patients: a content analysis and expert appropriateness rating study. J Trauma Acute Care Surg. 2015;79(4):568–79.
- 86. Roberts DJ, Bobrovitz N, Zygun DA, Ball CG, Kirkpatrick AW, Faris PD, et al. Indications for use of damage control surgery and damage control interventions in civilian trauma patients: a scoping review. J Trauma Acute Care Surg. 2015;78(6):1187–96.
- 87. Coccolini F, Biffl W, Catena F, Ceresoli M, Chiara O, Cimbanassi S, et al. The open abdomen, indications, management and definitive closure. World J Emerg Surg. 2015;10:32.
- 88. Acosta S, Bjarnason T, Petersson U, Palsson B, Wanhainen A, Svensson M, et al. Multicentre prospective study of fascial closure rate after open abdomen with vacuum and mesh-mediated fascial traction. Br J Surg. 2011;98(5):735–43.
- 89. Rasilainen SK, Mentula PJ, Leppaniemi AK. Vacuum and mesh-mediated fascial traction for primary closure of the open abdomen in critically ill surgical patients. Br J Surg. 2012;99(12):1725–32.
- 90. Bjarnason T, Montgomery A, Ekberg O, Acosta S, Svensson M, Wanhainen A, et al. Oneyear follow-up after open abdomen therapy with vacuum-assisted wound closure and meshmediated fascial traction. World J Surg. 2013;37(9):2031–8.
- 91. Atema JJ, Gans SL, Boermeester MA. Systematic review and meta-analysis of the open abdomen and temporary abdominal closure techniques in non-trauma patients. World J Surg. 2015;39(4):912–25.
- 92. Ogilve WH. The late complications of abdominal war wounds. Lancet. 1940;2:253–6.
- 93. Roberts DJ, Jenne CN, Ball CG, Tiruta C, Leger C, Xiao Z, et al. Efficacy and safety of active negative pressure peritoneal therapy for reducing the systemic inflammatory response after damage control laparotomy (the Intra-peritoneal Vacuum Trial): study protocol for a randomized controlled trial. Trials. 2013;14:141.
- 94. Kubiak BD, Albert SP, Gatto LA, Snyder KP, Maier KG, Vieau CJ, et al. Peritoneal negative pressure therapy prevents multiple organ injury in a chronic porcine sepsis and ischemia/ reperfusion model. Shock. 2010;34(5):525–34.
- 95. Emr B, Sadowsky D, Azhar N, Gatto LA, An G, Nieman G, et al. Removal of inflammatory ascites is associated with dynamic modification of local and systemic inflammation along with prevention of acute lung injury: in vivo and in silico studies. Shock. 2014;41(4):317–23.
- 96. Cheatham ML, Demetriades D, Fabian TC, Kaplan MJ, Miles WS, Schreiber MA, et al. Prospective study examining clinical outcomes associated with a negative pressure wound therapy system and Barker's vacuum packing technique. World J Surg. 2013;37(9):2018–30.
- 97. Kirkpatrick AW, Roberts DJ, Faris PD, Ball CG, Kubes P, Tiruta C, et al. Active negative pressure peritoneal therapy after abbreviated laparotomy: the intraperitoneal vacuum randomized controlled trial. Ann Surg. 2015;262(1):38–46.
- 98. Willms A, Gusgen C, Schaaf S, Bieler D, von Websky M, Schwab R. Management of the open abdomen using vacuum-assisted wound closure and mesh-mediated fascial traction. Langenbecks Arch Surg. 2015;400(1):91–9.
- 99. Petersson U, Acosta S, Bjorck M. Vacuum-assisted wound closure and mesh-mediated fascial traction--a novel technique for late closure of the open abdomen. World J Surg. 2007;31(11):2133–7.
- 100. Weaver JL, Smith JW. Direct peritoneal resuscitation: a review. Int J Surg. 2016;33(Pt B):237–41.
- 101. Weaver JL, Matheson PJ, Matheson A, Graham V, Harbrecht BG, Downard CD, et al. Direct peritoneal resuscitation reduces intestinal permeability after brain death. J Trauma Acute Care Surg. 2018;84(2):265–72.
- 102. Smith JW, Matheson PJ, Franklin GA, Harbrecht BG, Richardson JD, Garrison RN. Randomized controlled trial evaluating the efficacy of peritoneal resuscitation in the management of trauma patients undergoing damage control surgery. J Am Coll Surg. 2017;224(4):396–404.
- 103. Smith JW, Garrison RN, Matheson PJ, Franklin GA, Harbrecht BG, Richardson JD. Direct peritoneal resuscitation accelerates primary abdominal wall closure after damage control surgery. J Am Coll Surg. 2010;210(5):658–64.. 64–7
- 104. Smith JW, Ghazi CA, Cain BC, Hurt RT, Garrison RN, Matheson PJ. Direct peritoneal resuscitation improves inflammation, liver blood flow, and pulmonary edema in a rat model of acute brain death. J Am Coll Surg. 2014;219(1):79–87.
- 105. Chang R, Holcomb JB. Optimal fluid therapy for traumatic hemorrhagic shock. Crit Care Clin. 2017;33(1):15–36.
- 106. Harvin JA, Podbielski J, Vincent LE, Fox EE, Moore LJ, Cotton BA, et al. Damage control laparotomy trial: design, rationale and implementation of a randomized controlled trial. Trauma Surg Acute Care Open. 2017;2:1–5.
- 107. Khan A, Hsee L, Mathur S, Civil I. Damage-control laparotomy in nontrauma patients: review of indications and outcomes. J Trauma Acute Care Surg. 2013;75(3):365–8.
- 108. De Waele JJ. Abdominal Sepsis. Curr Infect Dis Rep. 2016;18(8):23.
- 109. Leppaniemi A, Kimball EJ, De Laet I, Malbrain ML, Balogh ZJ, De Waele JJ. Management of abdominal sepsis--a paradigm shift? Anaesthesiol Intensive Ther. 2015;47(4):400–8.
- 110. Bruns BR, Ahmad SA, O'Meara L, Tesoriero R, Lauerman M, Klyushnenkova E, et al. Nontrauma open abdomens: a prospective observational study. J Trauma Acute Care Surg. 2016;80(4):631–6.
- 111. Sartelli M, Abu-Zidan FM, Ansaloni L, Bala M, Beltran MA, Biffl WL, et al. The role of the open abdomen procedure in managing severe abdominal sepsis: WSES position paper. World J Emerg Surg. 2015;10:35.
- 112. Goussous N, Jenkins DH, Zielinski MD. Primary fascial closure after damage control laparotomy: sepsis vs haemorrhage. Injury. 2014;45(1):151–5.
- 113. van Ruler O, Mahler CW, Boer KR, Reuland EA, Gooszen HG, Opmeer BC, et al. Comparison of on-demand vs planned relaparotomy strategy in patients with severe peritonitis: a randomized trial. J Am Med Assoc. 2007;298(8):865–72.
- 114. Sartelli M, Catena F, Ansaloni L, Coccolini F, Corbella D, Moore EE, et al. Complicated intra-abdominal infections worldwide: the definitive data of the CIAOW Study. World J Emerg Surg. 2014;9:37.
- 115. Quyn AJ, Johnston C, Hall D, Chambers A, Arapova N, Ogston S, et al. The open abdomen and temporary abdominal closure systems--historical evolution and systematic review. Color Dis. 2012;14(8):e429–38.
- 116. Marinis A, Gkiokas G, Argyra E, Fragulidis G, Polymeneas G, Voros D. "Enteroatmospheric fistulae"--gastrointestinal openings in the open abdomen: a review and recent proposal of a surgical technique. Scand J Surg. 2013;102(2):61–8.
- 117. D'Hondt M, Devriendt D, Van Rooy F, Vansteenkiste F, D'Hoore A, Penninckx F, et al. Treatment of small-bowel fistulae in the open abdomen with topical negative-pressure therapy. Am J Surg. 2011;202(2):e20–4.
- 118. Martinez JL, Luque-de-Leon E, Mier J, Blanco-Benavides R, Robledo F. Systematic management of postoperative enterocutaneous fistulas: factors related to outcomes. World J Surg. 2008;32(3):436–43.. discussion 44
- 119. Bradley MJ, Dubose JJ, Scalea TM, Holcomb JB, Shrestha B, Okoye O, et al. Independent predictors of enteric fistula and abdominal sepsis after damage control laparotomy: results from the prospective AAST open abdomen registry. JAMA Surg. 2013;148(10):947–54.
- 120. Richter S, Dold S, Doberauer JP, Mai P, Schuld J. Negative pressure wound therapy for the treatment of the open abdomen and incidence of enteral fistulas: a retrospective bicentre analysis. Gastroenterol Res Pract. 2013;2013:730829.
- 121. Roberts DJ, Zygun DA, Grendar J, Ball CG, Robertson HL, Ouellet JF, et al. Negativepressure wound therapy for critically ill adults with open abdominal wounds: a systematic review. J Trauma Acute Care Surg. 2012;73(3):629–39.
- 122. Malbrain ML, Marik PE, Witters I, Cordemans C, Kirkpatrick AW, Roberts DJ, et al. Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice. Anaesthesiol Intensive Ther. 2014;46(5):361–80.
- 123. Loftus TJ, Jordan JR, Croft CA, Smith RS, Efron PA, Mohr AM, et al. Temporary abdominal closure for trauma and intra-abdominal sepsis: different patients, different outcomes. J Trauma Acute Care Surg. 2016;82(2):345–50.
- 124. Vincent JL. Dear SIRS, I'm sorry to say that I don't like you. Crit Care Med. 1997;25(2):372–4.
- 125. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801–10.
- 126. Shankar-Hari M, Deutschman CS, Singer M. Do we need a new definition of sepsis? Intensive Care Med. 2015;41(5):909–11.
- 127. Kubiak BD, Albert SP, Gatto LA, Vieau CJ, Roy SK, Snyder KP, et al. A clinically applicable porcine model of septic and ischemia/reperfusion-induced shock and multiple organ injury. J Surg Res. 2011;166(1):e59–69.