VIA MEDICA
REVIEW
Anaesthesiology Intensive Therapy
ISSN 0209–1712
10.5603/AIT.a2015.0027
www.ait.viamedica.pl

Intra-abdominal hypertension and abdominal compartment syndrome in pancreatitis, paediatrics, and trauma

Jan J. De Waele[1], Janeth C. Ejike[2], Ari Leppaniemi[3], Bart L. De Keulenaer[4], Inneke De laet[5], Andrew W. Kirkpatrick[6], Derek J. Roberts[6], Edward Kimball[7], Rao Ivatury[8], Manu L.N.G. Malbrain[5]

[1]Intensive Care Unit, University Hospital UZ Gent, Ghent, Belgium
[2]Department of Pediatrics, Loma Linda University, Loma Linda, California, USA
[3]Department of Abdominal Surgery, University of Helsinki, Finland
[4]Intensive Care Unit, Fiona Stanley Hospital, Murdoch, Western Australia, Australia and School of Surgery, University of Western Australia, Crawley, Western Australia, Australia
[5] Intensive Care Unit and High Care Burn Unit, Ziekenhuis Netwerk Antwerpen, ZNA Stuivenberg, Antwerp, Belgium
[6] Departments of Surgery, Critical Care Medicine and Community Health Sciences, University of Calgary, Calgary, AB, Canada
[7] Department of Surgery, University of Utah Health Sciences Center, Salt Lake City, Utah, USA
[8] Professor Emeritus, Virginia Commonwealth University, Department Surgery, Richmond, VA, USA

Abstract

Intra-abdominal hypertension (IAH) is an important contributor to early organ dysfunction among patients with trauma and sepsis. However, the impact of increased intra-abdominal pressure (IAP) among pediatric, pregnant, non-septic medical patients, and those with severe acute pancreatitis (SAP), obesity, and burns has been studied less extensively. The aim of this review is to outline the pathophysiologic implications and treatment options for IAH and abdominal compartment syndrome (ACS) for the above patient populations. We searched MEDLINE and PubMed to identify relevant studies. There is an increasing awareness of IAH in general medicine. The incidence of IAH and, to a lesser extent, ACS is high among patients with SAP. IAH should always be suspected and IAP measured routinely. In children, normal IAP in mechanically ventilated patients is approximately 7 ± 3 mm Hg. As an IAP of 10–15 mm Hg has been associated with organ damage in children, an IAP greater than 10 mm Hg should be considered IAH in these patients. Moreover, as ACS may occur in children at an IAP lower than 20 mm Hg, any elevation in IAP higher than 10 mm Hg associated with new organ dysfunction should be considered ACS in children until proven otherwise. Monitor IAP trends and be aware that specific interventions may need to be instituted at lower IAP than the current ACS definitions accommodate. Finally, IAH and ACS can occur both in abdominal trauma and extra-abdominal trauma patients. Early mechanical hemorrhage control and the avoidance of excessive fluid resuscitation are key elements in preventing IAH in trauma patients. IAH and ACS have been associated with many conditions beyond the general ICU patient. In adults and in children, the focus should be on the early recognition of IAH and the prevention of ACS. Patients at risk for IAH should be identified early during their treatment (with a low threshold to initiate IAP monitoring). Appropriate actions should be taken when IAP increases above 20 mm Hg, especially in patients developing difficulty with ventilation. Although on-operative measures should be instituted first, one should not hesitate to resort to surgical decompression if they fail.

Key words: intra-abdominal hypertension, abdominal compartment syndrome, specific conditions, pancreatitis, children, trauma

Intra-abdominal hypertension (IAH) is an important contributor to early organ dysfunction after emergency surgery or trauma and among patients with severe sepsis [1, 2]. However, the impact of increased intra-abdominal pressure (IAP) in non-septic medical, pediatric, and pregnant patients and those with severe acute pancreatitis (SAP), obesity, and burns has been studied less extensively [3]. The aim of this review is to review the definitions, pathophysiologic implications, and treatment options for IAH among pediatric and trauma patients and those with SAP.

METHODS

A MEDLINE and PubMed search was performed using the search terms "abdominal compartment syndrome" or "abdominal hypertension" or "abdominal pressure" and "pancreatitis" or "children" or "pediatrics" or "trauma". This search yielded many references, most of which were not relevant to the subject of this paper. The selected abstracts were screened and selected on the basis of relevance, methodology, and scientific merit. Full text articles of the selected abstracts were used to supplement the authors' expert opinion and experience. The references of the selected papers were also checked for other relevant material. The resulting references were included in the current review, and focuses on pancreatitis, children, and trauma. Each topic will be discussed separately hereafter related to practical clinical questions on definitions, epidemiology, pathophysiology, diagnosis, prevention, and treatment, concluding with some key messages for the reader. This review is an "extended" version of a book chapter on the same topic in the Core Critical Care Series [4].

SEVERE ACUTE PANCREATITIS AND INTRA-ABDOMINAL HYPERTENSION

PATHOPHYSIOLOGY AND EPIDEMIOLOGY

In patients with SAP, IAH can be present on admission or can occur shortly thereafter. When ACS develops, it frequently occurs within the first few days after admission for SAP. IAH and ACS in patients with SAP is partly related to the effects of the inflammatory process itself causing retroperitoneal edema, fluid collections, ascites, and ileus, and partly iatrogenic resulting from aggressive fluid resuscitation; and subsequently causing a decrease in abdominal wall compliance. The incidence of IAH and ACS in patients with SAP is 60–80% and 12–30%, respectively [5–7].

It is difficult to differentiate between the systemic inflammatory response syndrome (SIRS) of the pancreatitis-process itself and the effects of ACS. It is likely that the processes exacerbate each other in a multi-factorial manner such that the ACS has been previously referred to as multiple system organ failure (MSOF) secondary to IAH [8, 9]. However, when ACS develops, it is associated with significant organ dysfunction, which commonly affects the cardiovascular, respiratory, and renal systems. Raised IAP causes splinting of the diaphragms, leading to compression atelectasis and hypercapnia. This, in combination with bilateral pleural effusions, which are common in severe pancreatitis, results in severe hypoxia. The effects of high IAP on the cardiovascular and renal systems are well described in the literature and need urgent intervention to improve organ function and subsequently outcome [10, 11]. The mortality in patients developing ACS is 50–75% (at least when untreated) [12]. Early mortality in SAP may be from unrecognized and untreated ACS [13].

IAH needs to be suspected in all patients with SAP. As clinical examination is an inaccurate predictor of IAP, an IAP measurement is mandatory [14]. Recognizing IAH before ACS develops is essential, as this may allow for timely medical management options to be initiated and ACS to be prevented [15]. A practical approach is summarized in Figure 1.

While progression from IAH to ACS may be hyperacute (although usually it takes 1–2 days), the key to the prevention of ACS is the timely recognition and treatment of IAH. Detailed discussion of the medical management algorithm is beyond the scope of this review and can be found elsewhere [16]. In the context of SAP, percutaneous drainage of abdominal fluid collections and/or fluid removal via dialysis/diuretics has been shown to reduce IAP [17]. However, the effect of these treatments on clinical outcomes is unknown. Within this setting crystalloids should be used judiciously, especially in patients with impending ACS [18].

An IAP above 12 mm Hg should prompt closer vigilance. When the IAP is greater than 20 mm Hg and associated with new onset organ dysfunction, ACS may be diagnosed. One must also realize that at lower levels, IAH has proved to be a good predictor of SAP-related complications and subclinical but adverse consequences, such as bacterial translocation, have long been suspected but are hard to isolate and prove in complex patients [19, 20]. Serial IAP measurements are key for preventing progression to ACS and aggressive medical treatment should be instituted as soon as possible.

MEDICAL TREATMENT AND GENERAL ICU CARE

Decompressive surgery should not be implemented as soon as ACS is diagnosed in most cases. One should attempt the following non-operative and percutaneous interventions first: nasogastric decompression, gastroprokinetics, sedation, short term use of neuromuscular blockers, removal of excess fluid by diuretics or ultrafiltration, or ultrasound guided percutaneous drainage of ascites [16]. If the situation is adequately assessed and determined that time does not allow for medical interventions that will make a significant difference to stop the vicious cycle of ACS then timely decompression is indeed encouraged.

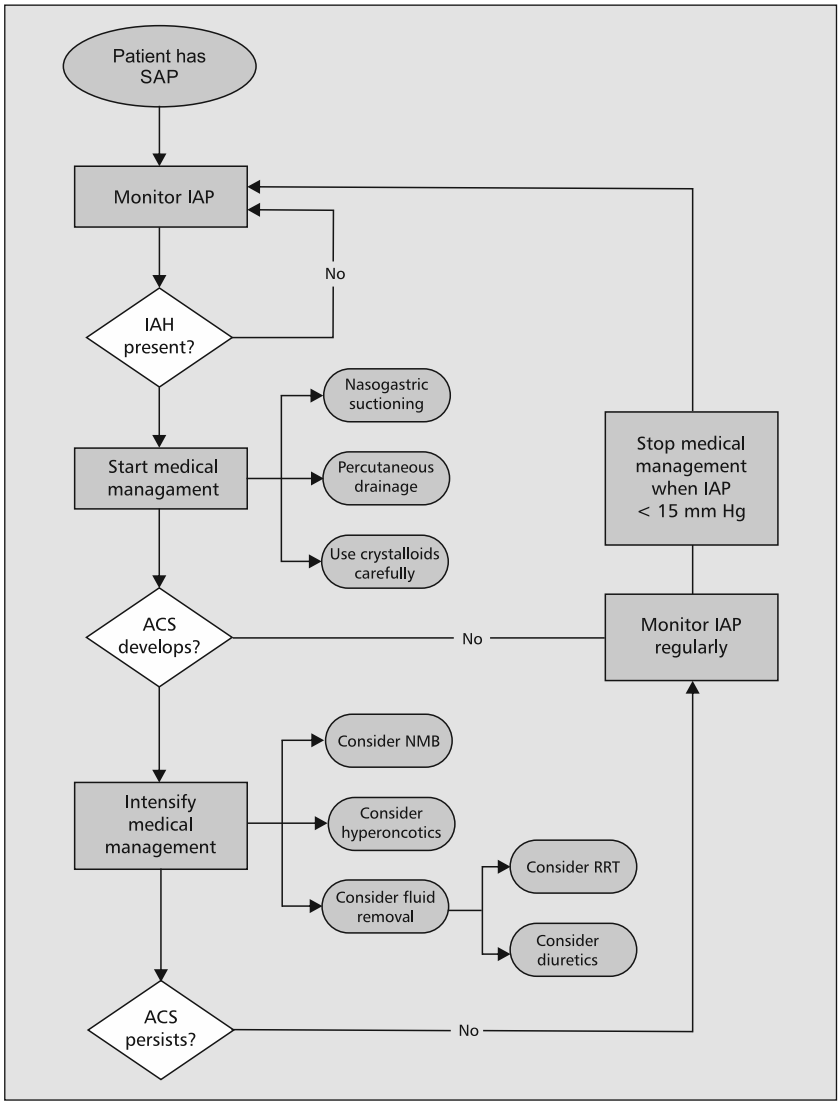

Figure 1. Management of severe acute pancreatitis (SAP) patients with intra-abdominal hypertension (IAH) or abdominal compartment syndrome (ACS). ACS — abdominal compartment syndrome; IAH — intra-abdominal hypertension; IAP — intra-abdominal pressure; NMB — neuromuscular blockers; RRT — renal replacement therapy

Despite the risk of developing ACS, the use of early enteral nutrition has proven to be beneficial in patients with acute pancreatitis [21]. It should be said that most patients in the studies on this topic did not have organ dysfunction (thus they did not have SAP), and the role of IAH in this field has not been studied. From a practical point of view, reduction of enteral feeding to 20 mL per hour should be considered when IAP increases between 15 and 20 mm Hg. Among patients with IAP levels above 20 mm Hg or ACS, enteral nutrition should be (temporarily) stopped. The use of simultaneous parenteral nutrition is important to maintain adequate caloric intake, especially in patients with open abdomen, although recent studies showed some controversy [22, 23]. Indeed, Cheatham showed significant protein loss via VAC dressings in critically ill patients [24].

SURGICAL THERAPY AND OPEN ABDOMINAL MANAGEMENT

When non-operative measures fail to decrease IAP and to improve respiratory, renal and cardiovascular function, one should consider surgical decompression (decompressive laparotomy) [25–27]. Renal dysfunction however may not be corrected immediately, especially after prolonged IAH [12, 28].

The most commonly used method for surgical decompression is the midline laparotomy where all layers (skin, fascia, peritoneum) are divided through a vertical midline incision extending from the xiphoid to the pubis. Alternatively, a bilateral subcostal incision, a few centimeters below the costal margins, can be used to perform a full-thickness laparostomy. A third method utilizes three short horizontal skin

incisions to perform a subcutaneous linea alba fasciotomy (SLAF) with the peritoneum left intact [29, 30]. The choice of the procedure should be determined on an individual basis. Although SLAF can be an elegant first step to reduce IAP in combination with non-operative measures, it may be less effective in terms of reduction of IAP; when SLAF fails, one should consider full decompression through a midline laparotomy [31]. SLAF does result in a giant midline hernia with associated need for surgery at a later stage. Since SLAF keeps the peritoneum closed it can be considered as a way to avoid a true open abdomen. Therefore, the advent of new methods for open abdomen management (e.g. a mesh-mediated VAC system) may not fully obliterate the need for SLAF [32]. Pancreatic necrosectomy is not advised during surgery for decompression and the standard indications for necrosectomy apply. Endoscopic-guided retroperitoneal techniques have also been suggested.

Regardless of the surgical decompression method, it is important to continue to measure IAP postoperatively in order to recognize recurrent ACS: the open abdomen after a full-thickness laparotomy requires temporary abdominal closure (TAC), preferably some type of dressing that creates negative pressure in the abdomen to maintain the abdominal domain and to prevent the retraction of the wound edges [26]. For the TAC one can use a home-made (e.g., Bogota bag or Barker's vacuum pack) or commercially available system [AbThera (Kinetic Concepts Inc.) or Renasys (Smith-Nephew)]. This requires dressing changes either in the operating room or intensive care unit every 2–3 days. The IAP should guide attempts at early closure within the first week.

Late complications associated with IAH can manifest themselves even weeks afterwards, especially infection or bleeding. If urine output deteriorates or other organ dysfunction relapses, one should consider IAH and measure IAP.

INTRA-ABDOMINAL HYPERTENSION IN CHILDREN

IAP MEASUREMENT AND IAH AND ACS IN CHILDREN

IAH and ACS may also develop in children, and organ dysfunction occurs at lower levels of IAP compared to adults. Although measurement of IAP using conventional methods is possible, lower instillation volumes are necessary [33]. Diagnosing IAH and ACS in in children can be challenging. The clinical awareness of the problem is also lower than in adults [34]. A growing interest in IAH and ACS has led to increasing publications, though scientific advances with regards to children lag behind adult data. Therefore, although many current practices in pediatrics are necessarily derived from adult studies, the definitions and standard techniques do not all directly translate to children [35, 36].

Normal IAP in spontaneously breathing subjects is reported to be close to zero mm Hg, but in mechanically ventilated children it is 7 ± 3 mm Hg [33, 37].

Several ways to monitor IAP in children have been described, but the most common method used is intermittent measurement via the bladder [33, 38]. Continuous bladder pressure monitoring, as described in adults, is difficult to apply in children due to the lack of small three-way urethral catheters [39]. To obtain accurate readings, the minimal volume of normal saline recommended for instillation into the bladder should be between 3 to 5 mL in all children up to 50 kg in weight [40]. In children with a weight above 5 kg, as a rule of thumb, an instillation volume of 1 mL kg^{-1} (up to a maximum of 25 mL) could also be used.

Factors that affect accurate IAP readings are similar to those in adults, however unlike in adults, no correlation between body mass index (BMI) percentiles or actual BMI have been reported in children [41]. Of particular note in children are conditions associated with abdominal muscle contraction such as crying and respiratory distress. Infants are abdominal breathers and breathe more rapidly than adults, making the acquisition of measurements at end-expiration (as recommended) challenging. Abdominal breathing in a child with respiratory distress may result in erroneously high IAP readings. Elimination of muscle contraction as a confounding factor can be achieved by adequate sedation and/or neuromuscular blockade in the mechanically ventilated child.

Evidence of organ dysfunction has been reported to occur at IAP as low as 10 to 15 mm Hg [42]. This implies that in children, ACS occurs at a lower IAP than the current definition accommodates. IAP greater than 10 mm Hg should be considered elevated in children. A Pediatric Risk of Mortality (PRISM) III score of ≥ 17 has been associated with increased risk for IAH [37].

DIAGNOSIS AND MONITORING IN THE PEDIATRIC POPULATION

As in adults, recognition of IAH requires a high index of suspicion. The critical IAP at which organ damage occurs is not clear and varies according to pre-morbid conditions, underlying causal factors, and mean arterial blood pressure (MAP). At this critical pressure, reduction in microcirculatory blood flow occurs, and the development of organ dysfunction begins. The current definitions for IAH and ACS are important as guides but are more directly applicable to adults. One has to be careful, when managing children, not to dismiss a diagnosis of ACS because the IAP has not reached 20 mm Hg. The exact IAP at which IAH becomes ACS is not yet known. This value may be even more fluid in children who have lower MAP and demonstrate decreased abdominal perfusion and ACS at lower IAP. Any elevation in IAP associated with new organ dysfunction should be considered ACS in children until proven otherwise. Although the abdominal perfusion pressure (APP) concept may also

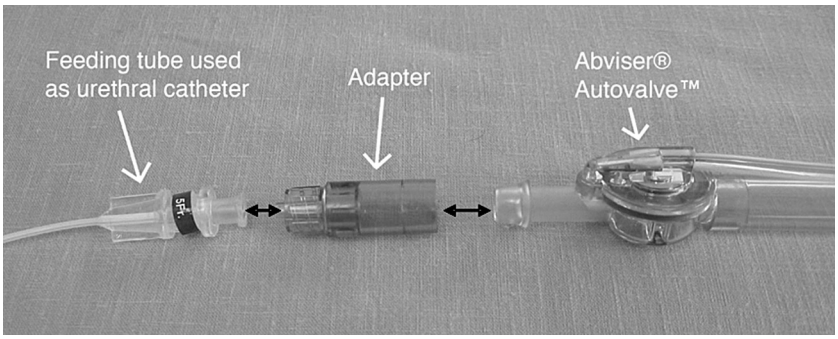

Figure 2. Neonatal Abviser adapter for IAP measurement via the bladder

apply in children, different goals probably apply when compared to adults.

Monitoring IAP trends along with parameters indicating organ dysfunction is especially important in children. In the absence of pediatric outcome studies using APP, practical suggested goals include lowering the IAP to below 10 mm Hg, and keeping the APP ≥ 35 mm Hg in infants and up to 50 mm Hg or greater in larger children. There are no commercial IAP monitoring kits available specifically designed for use in children but the Abviser® Neonate adaptor (ConvaTec Medical) accommodates the use of feeding tubes as improvised urethral catheters in smaller infants (Fig. 2).

MANAGING CHILDREN WITH IAH AND ACS

IAH has been reported in a wide variety of disease states among medical and surgical pediatric patients (Table 1). It is a co-morbid condition in critically ill children and is an independent predictor of mortality [37].

The management of ACS in children is dependent upon, and directed toward, the etiology of the underlying problem. The ideal treatment for ACS is the early recognition of IAH and the prevention of ACS altogether. Medical management is instituted primarily and if it fails, then surgical interventions such as decompressive laparotomies are utilized. Spring loaded silastic silos are used in the management of gastroschisis, and biological membranes are popular for coverage of exposed viscera of ruptured omphalocoeles. In this regard, pediatric surgeons have been managing IAH and purposefully preventing ACS for decades.

ACS mandates expeditious decompression. If accumulation of intra-peritoneal fluid is a factor, decompression may be accomplished by paracentesis or placement of a peritoneal drain. ACS due to visceral swelling will require a decompressive laparotomy and TAC, most commonly done with a vacuum pack, prosthetic mesh, Wittmann patch or VAC. It should be noted that as ACS can recur following TAC, continued vigilance and IAP monitoring are warranted. Medical management must continue in parallel to the surgical release of pressure.

INTRA-ABDOMINAL HYPERTENSION IN TRAUMA

EPIDEMIOLOGY OF IAH AND ACS IN TRAUMA PATIENTS

Although IAH and ACS are often only considered in abdominal trauma patients, all patients sustaining severe trauma are at risk of the development of IAH. Shock and the "lethal triad" often are associated with massive fluid

Table 1. Conditions associated with increased risk for ACS in children

Primary ACS	Secondary ACS
Gastroschisis	Aggressive fluid resuscitation
Omphalocele	Sepsis/Capillary leak syndrome
Necrotizing enterocolitis (NEC)	Multiple transfusions of blood products
Abdominal tumors	Multiple trauma
Intra-abdominal infections e.g. appendicitis, peritonitis, toxic megacolon	Hypothermia
Bowel obstruction, ischemia or infarction	Failed Fontan procedure/Heart failure with increased venous pressure
Abdominal trauma/hemorrhage	Renal failure
Complications of abdominal surgery	Extracorporeal membrane oxygenation
Ascites	Burns
Disproportionate solid organ transplant	Bone marrow transplant
Ileus, aganglionosis, constipation	

Table 2. Predictors of ACS in trauma patients

Primary ACS	Secondary ACS
Temperature below 34° C	Administration of > 7.5 L of crystalloids before ICU admission
Hemoglobin below 8 g dL-1	No indication for lifesaving surgical intervention
Base deficit below 8 mmol L-1	Relatively low urine output (< 150 mL h-1) on ICU admission considering the massive resuscitation
Administration of > 3 L of crystalloids	Poor intestinal perfusion measured by gastric tonometry
Transfusion of ≥ 3 U PRBC	
Need for emergency surgery	

resuscitation and capillary leakage and serve as common final pathways to ACS [43]. IAH and ACS have been described in all injured patients irrespective of the location of the trauma. Clinicians also need to consider the fact that all body compartments are ultimately linked by the concept of the polycompartment syndrome [44–48].

The epidemiology of IAH and ACS in trauma patients is changing rapidly in centers that have embraced these concepts and apply strategies that avoid the occurrence of both primary and secondary ACS. Whereas the incidence of ACS before the damage control era was high, with figures up to 30–40%, it has decreased dramatically in recent years [49]. Damage control resuscitation leads to lower levels of IAH [50, 51]. Some have proposed that the occurrence of ACS in trauma patients is an indicator of suboptimal trauma care, and should not be encountered anymore except for non-operatively managed blunt trauma patients [52, 53].

As in other conditions leading to ACS, early organ dysfunction serves as an early clue to IAH and ACS. As already discussed, other causes of organ dysfunction may be present.

MECHANISMS OF IAH IN TRAUMA PATIENTS

In trauma patients both primary and secondary ACS and combinations of both are commonly encountered. The typical presentation of patients with primary ACS is severe abdominal trauma requiring a damage-control laparotomy. In primary ACS, while an abdominal trauma is the direct cause, also mechanisms typically leading to secondary ACS, such as fluid resuscitation, may contribute to further deterioration of the problem [54]. Moreover, patients with extra-abdominal trauma may develop ACS — in this setting the syndrome is termed secondary ACS, and may develop in any kind of trauma patient who requires massive resuscitation [36, 55, 56]. Primary ACS typically develops early in patients requiring damage control surgery. Due to the liberal use of open abdomen techniques in abdominal trauma patients at risk, the incidence of primary ACS has decreased due to changes in resuscitation with damage control resuscitation and increased plasma to crystalloid ratios. Often, secondary ACS develops later than primary ACS. However, in patients requiring massive transfusion, early fulminant secondary ACS should be considered when organ dysfunction develops [57]. As this may happen within the first 12 hours after injury, IAP monitoring is indicated in severely injured patients early on.

The "bloody vicious cycle" or "lethal triad" in trauma patients refers to the combination of coagulopathy, hypothermia and acidosis [36, 43, 58]. These three factors represent metabolic failure in the injured patient and contribute to ongoing surgical and non-surgical blood loss. Its occurrence represents the need to abbreviate surgical procedures, if possible, and is associated with ischemia/reperfusion injury with resuscitation. Early hemorrhage control and hemostatic resuscitation aimed at restoring the coagulation components of the blood are important elements to avoid this lethal triad. Edema formation both inside and outside the abdominal cavity are the drivers of IAH in this setting, and strategies aimed at the prevention of this lethal triad will also avoid ACS altogether. Damage control surgery aimed at early and rapid bleeding control with prophylactic open abdominal management is the first step; rapid transfer of the patient to the ICU for correction of acidosis, hypothermia and coagulopathy follows, and definitive repair of injuries is delayed to a later stage [59].

In the acute phase ,it is often difficult to distinguish between ACS from shock and related systemic inflammation or ongoing bleeding [55]. Key elements here are IAP measurement and the exclusion of ongoing bleeding. An IAP measurement should be instituted early in trauma patients at risk for IAH.

With increasing proportions of blunt abdominal trauma patients being treated non-operatively, the risk for IAH and ACS may be increasing. Such patients may have lost considerable amounts of blood intraperitoneally, which, in addition to other factors such as edema formation due to shock from extra-abdominal injuries or ileus, may contribute to IAH. These same mechanisms may also lead to IAH in patients treated with angiographic embolization. Although laparotomy is a definitive treatment option, if residual bleeding from the initial injury is a concern, we have been able to avoid surgery by percutaneous drainage using large bore catheters.

IAP MONITORING IN TRAUMA PATIENTS: FROM MONITORING TO PREVENTION

The occurrence of IAH and ACS can be predicted from as early as the evaluation in the emergency department. A list of predictors of ACS is shown in Table 2. When one of these factors is present, measures to prevent ACS should be instituted.

The IAP needs also to be monitored in patients with an open abdomen. Although the risk for ACS in patients with an open abdomen is lower, new problems such as rebleeding may occur where increasing IAP may offer a first or additional clue to diagnose the problem. A midline laparotomy may be inadequate (too small) to fully decompress the abdomen. Moreover, in most patients IAP will decrease but will remain above the threshold for continuous damage to various organ systems. Medical strategies to decrease IAP further can be important tools to further reverse organ dysfunction in these patients. Leaving the abdomen open is often just one element in the treatment of IAH and ACS, and IAP remains a target for treatment.

Early and adequate bleeding control is an obvious way to prevent IAH and ACS in trauma patients [51]. Judicious fluid resuscitation avoiding large amounts of crystalloids, as well as the use of transfusion protocols with the early administration of plasma and platelets (so-called hemostatic resuscitation or damage control hematology) have been associated with lower rates of ACS in trauma patients. The application of a medical management algorithm has certainly improved survival over the last decade [60]. When this is not enough to prevent IAH from developing, the liberal use of open abdomen techniques in surgically treated patients combined with frequent IAP measurement and medical treatment in patients who develop IAH are useful strategies to prevent the catastrophic full blown ACS.

MANAGING IAH IN THE TRAUMA PATIENTS

Similar to patients with SAP, trauma patients who develop secondary ACS may be treated with subcutaneous linea alba fasciotomy (SLAF) [29]. Together with endoscopic abdominal wall components separation (EACS) [61] this may be an attractive alternative in patients who do not require an abdominal surgical intervention. In patients with massive burns and secondary ACS, one useful method is ultrasound-guided paracentesis than can decrease IAP and prevent progression to ACS. The abdomen should be left open as long as it benefits the patient. Continued IAP measurement is indicated, and all attempts should be made to close the abdomen early — that is, within 2 weeks. However, if it is possible to close it earlier, this should be pursued. If IAH persists after leaving the abdomen open, the available medical management options can help to lower IAP and facilitate early abdominal closure [62]. Leaving the abdomen open without due cause increases the risk of complications related to the open abdomen treatment, as well as the need for delayed, planned hernia repair.

SUMMARY KEY POINTS

IAH AND SEVERE ACUTE PANCREATITIS

- IAH and ACS are common in severe acute pancreatitis.
- Always suspect IAH and measure IAP regularly.
- When IAP increases above 20 mm Hg, suspect ACS.
- Start early enteral nutrition.
- Use judicious fluid management.
- Try non-operative measures first (e.g. percutaneous ultrasound or CT guided evacuation of fluid), but do not hesitate to resort to surgical decompression if this fails.

IAH IN CHILDREN

- Normal IAP in mechanically ventilated children is about 7 ± 3 mm Hg.
- Cutoff levels for IAH and ACS in children are lower compared to adults.
- IAP greater than 10 mm Hg in children should be considered IAH.
- ACS may occur in children at an IAP lower than 20 mm Hg. Any elevation in IAP higher than 10 mm Hg associated with new organ dysfunction should be considered ACS in children until proven otherwise.
- Monitor IAP and APP trends and be aware that specific interventions may need to be instituted at a lower IAP than the current ACS definitions accommodate.
- Abdominal breathing in children with respiratory distress may result in erroneously high IAP readings.
- Practical goals in management should include lowering the IAP to below 10 mm Hg and keeping the APP ≥ 35 mm Hg in the infant and up to 50 mm Hg or greater in the larger child.
- Moreover, in children, the focus should be on the early recognition of IAH and the prevention of ACS altogether.

IAH IN TRAUMA PATIENTS

- IAH and ACS can occur both in abdominal and extra-abdominal trauma patients.
- Patients at risk of IAH should be identified early during the treatment.
- Always suspect IAH and measure IAP regularly in all severely injured patients irrespective of the site of injury.
- Burn patients are at special risk for secondary IAH and ACS.
- Hypothermia, acidosis and coagulopathy may trigger the 'bloody vicious' cycle and need to be avoided.
- Early bleeding control and avoiding massive transfusion are key elements in preventing IAH in trauma patients.
- Open abdominal treatment should be applied liberally in patients at risk.

- The use of medical management strategies to reduce IAP remain important even after leaving the abdomen open as they facilitate early closure of the abdomen, and avoid complications related to open abdomen treatment.
- Try non-operative measures first in secondary ACS e.g. multiple trauma, extensive burns (percutaneous ultrasound or CT guided evacuation of fluid, or fluid removal with diuretics or dialysis with net ultrafiltration), but do not hesitate to resort to surgical decompression if they fail.

CONCLUSIONS

The occurrence of IAH and ACS has been associated in many conditions beyond the classical ICU, surgical or trauma patient. The true incidence of IAH in severe acute pancreatitis, children and trauma cases is high and probably underestimated. In adults but also in children, patients at risk of IAH should be identified early during the treatment and the focus should be on the early recognition of IAH and the prevention of ACS. Therefore, clinicians should always suspect IAH and measure the IAP regularly during the resuscitation period. Appropriate actions should be taken when IAP increases above 20 mm Hg, especially in patients difficult to ventilate with new onset organ dysfunction. Although non-operative measures come first, one should not hesitate to resort to surgical decompression if they fail.

ACKNOWLEDGEMENTS

1. All authors are members of the World Society of Abdominal Compartment Syndrome (https://www.wsacs.org/). Dr Derek Roberts is supported by an Alberta Innovates – Health Solutions Clinician Fellowship Award, a Knowledge Translation Canada Strategic Training in Health Research Fellowship, and funding from the Canadian Institutes of Health Research. Dr Manu Malbrain is founding President of WSACS and current Treasurer. He is a member of the medical advisory Board of Pulsion Medical System (part of Maquet Getinge) and consults for ConvaTec, Kinetic Concepts International (KCI) Inc. and Holtech Medical. Dr Rao Ivatury is a consultant for KCI Inc. Dr Andrew Kirkpatrick was the principle Investigator of an investigator-initiated randomized controlled trial on open abdomen managed funded by the ACELITY Corp. Dr Jan De Waele is a Senior Clinical Researcher with the Research Foundation Flanders (Belgium) and has served as a consultant to Smith&Nephew, and KCI Inc. The other authors have no possible conflicts of interest in relation to the contents of this manuscript.
2. Sections of this review formed contributions to the Proceedings of the 6th World Congress on Abdominal Compartment Syndrome (WCACS, www.wcacs.org) in Cartagena, Colombia (May 22–25 in 2013) and were presented at the meeting.

References:

1. *Malbrain ML, Chiumello D, Pelosi P et al.:* Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. Intensive Care Med 2004; 30: 822–829.
2. *Malbrain ML, Chiumello D, Pelosi P et al.:* Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. Crit Care Med 2005; 33: 315–322.
3. *Larsson A:* Clinical significance of elevated intraabdominal pressure during common conditions and procedures. Acta Clin Belg Suppl 2007; 62: 74–77.
4. *Malbrain MLNG, De Waele J:* Section 3. Specific conditions: when to worry more? In: *Vuylsteke A (ed.):* Core critical care series: intra-abdominal hypertension. Cambridge University Press 2013.
5. *De Waele JJ, Hoste E, Blot SI, Decruyenaere J, Colardyn F:* Intra-abdominal hypertension in patients with severe acute pancreatitis. Crit Care (London, England) 2005; 9: R452–457.
6. *Ke L, Tong ZH, Ni HB, Ding WW, Sun JK, Li WQ, Li N, Li JS:* The effect of intra-abdominal hypertension incorporating severe acute pancreatitis in a porcine model. PLoS One 2012; 7: e33125. doi: 10.1371/journal.pone.0033125.
7. *Ke L, Ni HB, Sun JK, Tong ZH, Li WQ, Li N, Li JS:* Risk factors and outcome of intra-abdominal hypertension in patients with severe acute pancreatitis. World J Surg 2012; 36: 171–178. doi: 10.1007/s00268-013-2087-5.
8. *Moore FA:* The role of the gastrointestinal tract in postinjury multiple organ failure. Am J Surg 1999; 178: 449–453.
9. *Raeburn CD, Moore EE:* Abdominal compartment syndrome provokes multiple organ failure: animal and human supporting evidence. In: *Ivatury R, Cheatham M, Malbrain M, Sugrue M (ed.):* Abdominal compartment syndrome. Landes Bioscience, Georgetown 2006: 157–169.
10. *De laet I, Malbrain ML, Jadoul JL, Rogiers P, Sugrue M:* Renal implications of increased intra-abdominal pressure: are the kidneys the canary for abdominal hypertension? Acta Clin Belg Suppl 2007; 62: 119–130.
11. *Cheatham ML, Malbrain ML:* Cardiovascular implications of abdominal compartment syndrome. Acta Clin Belg Suppl 2007; 62: 98–112.
12. *De Waele JJ, Hoste EA, Malbrain ML:* Decompressive laparotomy for abdominal compartment syndrome — a critical analysis. Crit Care 2006; 10: R51.
13. *Leppaniemi A, Johansson K, De Waele JJ:* Abdominal compartment syndrome and acute pancreatitis. Acta Clin Belg Suppl 2007; 62: 131–135.
14. *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: 207–211.
15. De Waele JJ, Leppaniemi AK: Intra-abdominal hypertension in acute pancreatitis. World J Surg 2009; 33: 1128–1133. doi: 10.1007/s00268-009-9994-5.
16. *De Keulenaer BL, De Waele JJ, Malbrain ML:* Nonoperative management of intra-abdominal hypertension and abdominal compartment syndrome: evolving concepts. Am Surg 2011; 77 (Suppl 1): S34–41.
17. *Kula R, Szturz P, Sklienka P, Neiser J, Jahoda J:* A role for negative fluid balance in septic patients with abdominal compartment syndrome? Intensive Care Med 2004; 30: 2138–2139.
18. *De laet IE, De Waele JJ, Malbrain MLNG:* Fluid resuscitation and intra-abdominal hypertension. In: *Vincent J-L (ed.):* Yearbook of intensive care and emergency medicine. Springer-Verlag, Berlin 2008: 536–548.
19. *Diebel LN, Dulchavsky SA, Wilson RF:* Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 1992; 33: 45–48; discussion 48–49.
20. *Diebel LN, Dulchavsky SA, Brown WJ:* Splanchnic ischemia and bacterial translocation in the abdominal compartment syndrome. J Trauma 1997; 43: 852–855.
21. *Petrov MS, Kukosh MV, Emelyanov NV:* A randomized controlled trial of enteral versus parenteral feeding in patients with predicted severe acute pancreatitis shows a significant reduction in mortality and in infected pancreatic complications with total enteral nutrition. Dig Surg 2006; 23: 336–344; discussion 344–335.
22. *Casaer MP, Wilmer A, Hermans G, Wouters PJ, Mesotten D, Van den Berghe G:* Role of disease and macronutrient dose in the randomized controlled EPaNIC Trial: a post hoc analysis. Am J Respir Crit Care Med 2013; 187: 247–255. doi: 10.1164/rccm.201206-0999OC.

23. *Vanderheyden S, Casaer MP, Kesteloot K et al.:* Early versus late parenteral nutrition in ICU patients: cost analysis of the EPaNIC trial. Crit Care 2012; 16: R96. doi: 10.1186/cc11361.
24. *Cheatham ML, Safcsak K, Brzezinski SJ, Lube MW:* Nitrogen balance, protein loss, and the open abdomen. Crit Care Med 2007; 35: 127–131.
25. *De Waele JJ, Hoste EA, Malbrain ML:* Decompressive laparotomy for abdominal compartment syndrome — a critical analysis. Crit Care 2006; 10(2): R51.
26. *Leppaniemi A, Mentula P, Hienonen P, Kemppainen E:* Transverse laparostomy is feasible and effective in the treatment of abdominal compartment syndrome in severe acute pancreatitis. World J Emerg Surg 2008; 3: 6. doi: 10.1186/1749-7922-3-6.
27. *Mentula P, Hienonen P, Kemppainen E, Puolakkainen P, Leppaniemi A:* Surgical decompression for abdominal compartment syndrome in severe acute pancreatitis. Arch Surg 2010; 145: 764–769. doi: 10.1001/archsurg.2010.132.
28. *Sugrue M, Jones F, Janjua KJ, Deane SA, Bristow P, Hillman K:* Temporary abdominal closure: a prospective evaluation of its effects on renal and respiratory physiology. J Trauma 1998; 45: 914–921.
29. *Cheatham ML, Fowler J, Pappas P:* Subcutaneous linea alba fasciotomy: a less morbid treatment for abdominal compartment syndrome. Am Surg 2008; 74: 746–749.
30. *Leppaniemi A:* Surgical management of abdominal compartment syndrome; indications and techniques. Scand J Trauma Resusc Emerg Med 2009; 17: 17. doi: 10.1186/1757-7241-17-17.
31. *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: 1922–1924.
32. *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: 1725–1732. doi: 10.1002/bjs.8914.
33. *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: 2157–2162. doi: 10.1097/CCM.0b013e31817b8c88.
34. *Ejike JC, Newcombe J, Baerg J, Bahjri K, Mathur M:* Understanding of abdominal compartment syndrome among pediatric healthcare providers. Crit Care Res Pract 2010, 2010: 876013. doi: 10.1155/2010/876013.
35. *Cheatham ML, Malbrain ML, Kirkpatrick A et al.:* Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. Intensive Care Med 2007; 33: 951–962.
36. *Malbrain ML, Cheatham ML, Kirkpatrick A et al.:* Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 2006; 32: 1722–1732.
37. *Ejike JC, Humbert S, Bahjri K, Mathur M:* Outcomes of children with abdominal compartment syndrome. Acta Clin Belg Suppl 2007; 62: 141–148.
38. *Davis PJ, Koottayi S, Taylor A, Butt WW:* Comparison of indirect methods of measuring intra-abdominal pressure in children. Intensive Care Med 2005; 31: 471–475.
39. *Balogh Z, De Waele JJ, Malbrain ML:* Continuous intra-abdominal pressure monitoring. Acta Clin Belg Suppl 2007: 26–32.
40. *Suominen PK, Pakarinen MP, Rautiainen P, Mattila I, Sairanen H:* Comparison of direct and intravesical measurement of intraabdominal pressure in children. J Pediatr Surg 2006; 41: 1381–1385.
41. *Ejike JC, Kadry J, Bahjri K, Mathur M:* Semi-recumbent position and body mass percentiles: effects on intra-abdominal pressure measurements in critically ill children. Intensive Care Med 2010; 36: 329–335. doi: 10.1007/s00134-009-1708-9.
42. *Beck R, Halberthal M, Zonis Z, Shoshani G, Hayari L, Bar-Joseph G:* Abdominal compartment syndrome in children. Pediatr Crit Care Med 2001; 2: 51–56.
43. *Cosgriff N, Moore EE, Sauaia A, Kenny–Moynihan M, Burch JM, Galloway B:* Predicting life-threatening coagulopathy in the massively transfused trauma patient: hypothermia and acidoses revisited. J Trauma 1997; 42: 857–861.
44. *Malbrain MLNG, De laet I:* A new concept: the polycompartment syndrome — Part 2. Int J Intensive Care 2009, Spring 2009: 19–25.
45. *Malbrain MLNG, De laet I:* A new concept: the polycompartment syndrome — Part 1. Int J Intensive Care 2008, Autumn 2008: 19–24.
46. *Malbrain ML, Wilmer A:* The polycompartment syndrome: towards an understanding of the interactions between different compartments! Intensive Care Med 2007; 33: 1869–1872.
47. *Scalea TM, Bochicchio GV, Habashi N, McGunn M, Shih D, McQuillan K, Aarabi B:* Increased intra-abdominal, intrathoracic, and intracranial pressure after severe brain injury: multiple compartment syndrome. J Trauma 2007; 62: 647–656.
48. *Balogh ZJ, Butcher NE:* Compartment syndromes from head to toe. Crit Care Med 2010; 38 (9 Suppl): S445–451. doi: 10.1097/CCM.0b013e3181ec5d09.
49. *Deeren D, Malbrain M:* Prevalence and incidence of Intra-abdominal hypertension. In: *Ivatury R, Cheatham M, Malbrain M, Sugrue M (ed.):* Abdominal compartment syndrome. Landes Bioscience, Georgetown 2006: 82–88.
50. *Cotton BA, Reddy N, Hatch QM et al.:* Damage control resuscitation is associated with a reduction in resuscitation volumes and improvement in survival in 390 damage control laparotomy patients. Ann Surg 2011; 254: 598–605. doi: 10.1097/SLA.0b013e318230089e.
51. *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: 41–48; discussion 48–49. doi: 10.1097/TA.0b013e31819313bb.
52. *Balogh ZJ, Martin A, van Wessem KP, King KL, Mackay P, Havill K:* Mission to eliminate postinjury abdominal compartment syndrome. Arch Surg 2011; 146: 938–943. doi: 10.1001/archsurg.2011.73.
53. *Balogh ZJ, Malbrain M:* Resuscitation in intra-abdominal hypertension and abdominal compartment syndrome. Am Surg 2011; 77 (Suppl 1): S31–33.
54. *Balogh Z, McKinley BA, Holcomb JB et al.:* Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma 2003; 54: 848–859; discussion 859–861.
55. *Ball CG, Kirkpatrick AW, McBeth P:* The secondary abdominal compartment syndrome: not just another post-traumatic complication. Can J Surg 2008; 51: 399–405.
56. *Kirkpatrick AW, Balogh Z, Ball CG et al.:* The secondary abdominal compartment syndrome: iatrogenic or unavoidable? J Am Coll Surg 2006; 202: 668–679.
57. *Rizoli S, Mamtani A, Scarpelini S, Kirkpatrick AW:* Abdominal compartment syndrome in trauma resuscitation. Curr Opin Anaesthesiol 2010; 23: 251–257. doi: 10.1097/ACO.0b013e3283358a0f.
58. *Moore FA, McKinley BA, Moore EE:* The next generation in shock resuscitation. Lancet 2004; 363: 1988–1996.
59. *Balogh ZJ, van Wessem K, Yoshino O, Moore FA:* Postinjury abdominal compartment syndrome: are we winning the battle? World J Surg 2009; 33: 1134–1141. doi: 10.1007/s00268-009-0002-x.
60. *Cheatham ML, Safcsak K:* Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? Crit Care Med 2010; 38: 402–407.
61. *Voss M, Pinheiro J, Reynolds J et al.:* Endoscopic components separation for abdominal compartment syndrome. Am J Surg 2003; 186: 158–163.
62. *Ivatury RR, Kolkman KA, Johansson K:* Management of open abdomen. Acta Clin Belg Suppl 2007; 62: 206–209.

Corresponding author:
Manu LNG Malbrain, MD, PhD
ICU and High Care Burn Unit Director
Ziekenhuis Netwerk Antwerpen,
ZNA Stuivenberg
Lange Beeldekensstraat 267
B-2060 Antwerp, Belgium
e-mail: manu.malbrain@skynet.be

Received: 30.03.2015
Accepted: 7.05.2015