

# Intra-abdominal hypertension and the abdominal compartment syndrome: 'ARDS' of the gut

The expansion of substance within a compartment of relatively fixed volume creates an increase in intra-compartmental pressure. Within the anatomical confines of the human body this pressure phenomenon can occur within several different anatomic compartments i.e. the skull, pericardium, limbs and the thoraco-abdominal cavity. Intra-abdominal hypertension (IAH), defined as an intra-abdominal pressure (IAP) of 12 mmHg or higher, occurs in over 50% of intensive care patients. Elevated intra-abdominal pressure leads directly to progressive organ dysfunction in the intestinal, renal, pulmonary, cardiovascular and central nervous systems. This syndrome occurs with equal prevalence in surgical and medical intensive care units. Early detection of IAH allows the clinician to manage this condition with medical therapies. Progressive increase in IAP to levels above 20–25 mmHg, with associated organ failure, is defined as the abdominal compartment syndrome (ACS), a process that usually requires surgical intervention. This article will describe the close relationship of ACS with other inflammatory diseases such as ARDS, the prevalence of intra-abdominal hypertension, the medical therapies available for treatment and the outcome data available to support these interventions.

During the war between the USA and Vietnam, in the 1960s and early '70s, advances in aero-medical transport allowed for the rapid evacuation of severely wounded soldiers, casualties who would have died in the field in previous conflicts. Surgeons in well-equipped hospitals rapidly abated bleeding and aggressively resuscitated these critically ill patients. Never before had patients been consistently retrieved from such severe resuscitative debt. However, this debt in turn triggered an immune modulated inflammatory response that had also rarely been seen. Within days of their initial 'successful' resuscitation, these soldiers began to suffer from significant respiratory distress and approximately 60–70% went on to die. Treating physicians labelled this syndrome 'Da Nang lung' and struggled to understand its aetiology.<sup>1</sup>

In August 1967, DG Ashbaugh published a case series describing a 'new' respiratory syndrome seen in critically ill patients.<sup>2</sup> This disease process, soon labelled the 'acute respiratory distress syndrome' (ARDS), would become the focus of intense research for the next four decades. Results of this research have demonstrated that ARDS is a highly morbid process resulting from hyper-activation of the immune system and its mediators. Unregulated, this immune response becomes systemic resulting in physiological impairment of the lungs as well as multiple other organ systems.<sup>3,4</sup> Soon it was clear that Da Nang lung was just another face of ARDS – the same hyperactive immune response described by Ashbaugh. The advances in medical transport and trauma surgery arising from the Vietnam

experience literally helped create a new pathological syndrome that had not been consistently seen in the past. Since then, ARDS has become a ubiquitous problem for intensive care physicians with more than 150,000 cases per year seen in the USA. Despite 40 years of research and millions of dollars expended to understand this pathophysiology, there has been only a modest decrease in ARDS-related mortality.<sup>3,4</sup>

In 2006 we are faced with another emerging syndrome related to medical advances and systemic inflammation – abdominal compartment syndrome (ACS). Like ARDS, ACS is caused by a diverse set of medical diseases resulting in a systemic inflammatory response that leads to a unified pathophysiological process.

Though many articles published in the last century allude to this syndrome,<sup>5–7</sup> Dr Irving L Kron is credited with the first clinical series describing the recognition and management of ACS.<sup>7</sup> Kron's case series, published in 1984, described 10 patients with abdominal distension, elevated IAP and oliguria following chest or abdominal surgery. Seven of these 10 patients responded to abdominal decompression through surgical re-exploration; five survived to discharge. The three patients who were not surgically decompressed had progressively increasing IAPs and ultimately died of multi-system organ failure.

Prior to 1967, others had described respiratory syndromes similar to those seen in Ashbaugh's patients.<sup>8,9</sup> However, his clinical description and title put ARDS on the medical map. Similarly, intra-abdominal pressure and its pathophysiology had been described in numerous treatises dating back to the 19th century.<sup>5–7</sup> Kron's clinical recognition of ACS and its response to decompression initiated a renewed clinical awakening to the morbidity and mortality associated with this syndrome. However, this awakening was slow: it was not until the mid-1990s that significant clinical research into IAH and ACS was initiated (Figure 1). The clinical impact of this growing body of research is just now being realised. Like ARDS, ACS is now recognised as another deadly cousin in the family of systemic inflammatory diseases that can have impact on a broad spectrum of critically ill patients.

## PATHOPHYSIOLOGY OF ACS

Dr Kron sparked renewed clinical interest in a pathophysiology that results from the expansion of tissue and fluid in a fixed compartment (the abdomen). This causes increased pressure, compromised perfusion and tissue ischaemia, in turn resulting in both direct and – through an inflammatory response – indirect multiple organ failure (MOF). More specifically, diverse insults such as sepsis, trauma, pancreatitis and ischaemia-reperfusion trigger an unregulated immune response resulting in the

EJ Kimball MD, MSc  
Assistant Professor of  
Surgery and Emergency  
Medicine,  
University of Utah,  
Health Sciences Center,  
Salt Lake City, Utah,  
USA



systemic inflammatory response syndrome (SIRS). The capillary leak caused by this indiscriminate inflammatory response, coupled with fluid resuscitation, results in the accumulation of interstitial and intra-abdominal free fluid.<sup>10</sup> In the relatively fixed volume of the abdominal cavity, this fluid accumulation gradually results in increased intra-abdominal pressure. Once that pressure exceeds a certain threshold, substantial physiological derangements occur, both from the mechanical effects of this pressure and from the perpetuation of systemic inflammation that occurs due to tissue ischaemia.<sup>10-26</sup>

International consensus now defines IAP >12 mmHg as IAH<sup>16</sup> and several studies have demonstrated that patients with IAH suffer significantly higher morbidity and mortality.<sup>10-26</sup> As IAP increases, tissue ischaemia worsens and, if sustained, can result in organ failure. The abdominal compartment syndrome, defined as IAP >20 mmHg with at least one organ failure, is the end result of unchecked IAP increases. This final syndrome triggers or perpetuates systemic inflammation resulting in a vicious cycle of distant organ compromise (including ARDS) and mortality approaching 80%.<sup>10-26</sup> For purposes of discussion, we will refer to this pathophysiology as 'IAH/ACS' because physiological compromise can occur with IAH alone well before the ACS is clinically present.<sup>14,15</sup>

This pressure-induced pathophysiology is analogous to the Monroe/Kelly doctrine used to describe the relationship of intracranial pressure (ICP) in another fixed compartment, the skull. Rising ICP results in compromised perfusion to the brain. This pathophysiology is caused by a reduction in cerebral perfusion pressure (CPP) and is described by the formula:

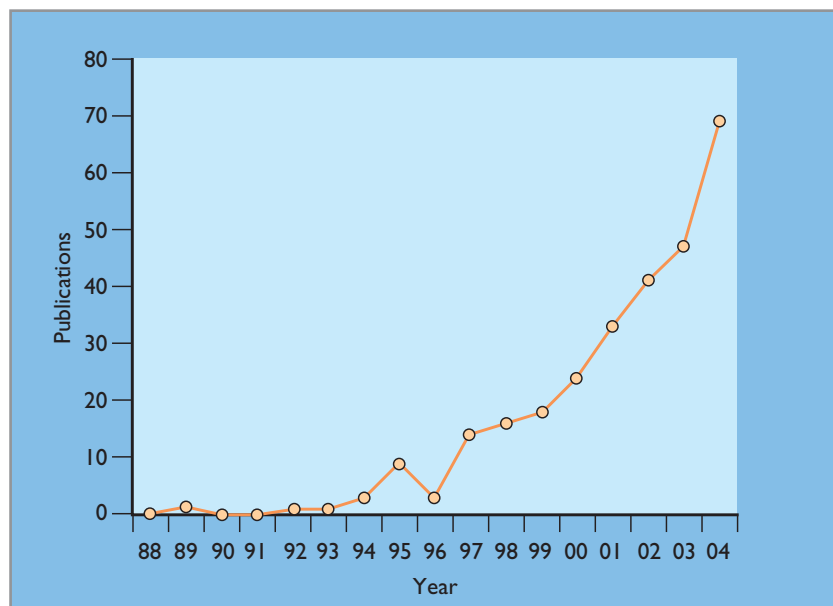
$$\text{CPP} = \text{mean arterial pressure (MAP)} - \text{ICP}$$

Indeed, a similar formula has been applied to the abdomen. Cheatham *et al.*, using the formula:

$$\text{abdominal perfusion pressure (APP)} = \text{MAP} - \text{IAP}$$

demonstrated that APP is a more accurate predictor of outcome than IAP in the setting of IAH/ACS.<sup>27</sup>

Figure 1. IAH/ACS publications by year.



Both Ashbaugh and Kron described a 'similar response to a variety of stimuli', with Ashbaugh concluding that 'a common mechanism of injury may be postulated'.<sup>2</sup> As noted, there is now ample evidence that this 'common mechanism of injury' for ARDS is a hyperactive immune response triggered by a host of primary or secondary aetiologies. There is now growing evidence that this same immune mediated inflammatory cascade, triggered by diverse aetiologies, is a direct cause of IAH/ACS.<sup>28,29</sup> In addition, prevalence studies indicate that IAH/ACS is as common or more common than ARDS among critically ill patients.<sup>11</sup>

#### IMMUNE-MEDIATED SYSTEMIC INFLAMMATION AND IAH/ACS

Pro-inflammatory cytokines have been shown to play an important role in the development of SIRS and ARDS. Varying types of primary insults, e.g., trauma, burns, pancreatitis and infection, can trigger the immune system into a poorly regulated inflammatory response that leads to a second insult, often resulting in multi-system organ failure and death.<sup>29</sup> Da Nang lung, with its devastating mortality, was an early example of this double insult model. Today this pattern of primary insult followed by inflammatory injury is repeated with increasing frequency in ICU patients. Microbial invasion, ischaemia reperfusion or mechanical tissue disruption trigger predictable biochemical and cellular immune cascades. Neutrophils play a fundamental role in this host defence cascade, directly attacking invading organisms and releasing pro-inflammatory cytokines (e.g. tumour necrosis factor, interleukins 1 and 6) that potentiate the neutrophil response.<sup>28,29</sup>

Of proximal importance to this discussion is the growing body of literature that supports the important role of IAH/ACS in this model. Researchers at the University of Texas-Houston have performed elegant work demonstrating the post-injury priming of the innate immune system and the role of IAH/ACS in this process.<sup>28,29</sup> Specifically, they have shown that neutrophils from traumatically injured patients are first up-regulated or primed for inflammatory response.<sup>28,29</sup> Moderate insults trigger a controlled neutrophil response; however, severe insults can lead to an unregulated hyper-immune response and consequently systemic inflammation and end-organ injury. The severity of the inflammatory response that follows this priming is determined by the severity of the primary insult and the timing and adequacy of subsequent therapy.<sup>29</sup> Work by these and other investigators indicates that focal ischaemia-reperfusion injury from IAH/ACS may incite distant organ injury (ARDS) through systemic circulation of inflammatory cytokines. It is also apparent that focal insult away from the gut can initiate systemic inflammation resulting in gut capillary permeability, visceral oedema and IAH/ACS. Thus a self-perpetuating vicious cycle ensues with significant morbidity and mortality.

#### IAH/ACS TRIGGER OR SECONDARY SIDE EFFECT OF SIRS

In a debate reminiscent of the academic dialogue surrounding ARDS, many question whether IAH/ACS is merely a collateral phenomenon of established organ failure or the actual cause of multi-system organ failure. As noted, current evidence suggests that unchecked IAH/ACS can be both the priming event and the fuel for the engine

of SIRS, leading to increased morbidity and mortality.<sup>10–24</sup> For example, primary causes of elevated IAP, such as retroperitoneal bleeding, abdominal trauma, intra-abdominal tumours and distended bowel, can result in direct mechanical obstruction of capillary perfusion to the gut, while simultaneously reducing cardiac output through impairment of venous return to the heart.<sup>17,18</sup> This initial direct insult to the abdominal cavity leads to gut ischaemia, resulting in the triggering of a systemic inflammatory response. In turn, the systemic inflammatory response causes capillary leak leading to bowel oedema, further increasing IAP and resulting in a morbid cycle of ischaemia and oedema that can progress to MOF.

In contrast, secondary causes of elevated IAP, such as sepsis, non-abdominal trauma and burns, may be the primary trigger of the systemic inflammatory response resulting in subsequent capillary leak, visceral oedema, increased IAP and finally ACS. Regardless of the initial trigger, IAH/ACS is an independent predictor of mortality that can and should be specifically addressed.<sup>11,30</sup> As noted, the ‘common mechanism of injury’ is the immune mediated systemic inflammation that is a common end-pathway of a myriad of disease processes seen in critically ill patients. Thus IAH/ACS should no longer be considered a disease limited to trauma patients any more than ARDS should be thought of as an isolated pulmonary disease. While multiple trauma may be the ‘perfect storm’ for the development of ACS, it is just the tip of the pathological iceberg of IAH/ACS triggers.

In a large survey of intensivists on the diagnosis and management of IAH/ACS, respondents described over 40 separate disease processes that resulted in IAH/ACS in their clinical practice.<sup>31</sup> Recent studies of the prevalence of IAH/ACS also bear this point out. As previously noted, Malbrain *et al.* demonstrated that IAH/ACS is as common in medical ICUs as in surgical/trauma ICUs with 30–50% of patients affected by this pathophysiology.<sup>11</sup> A number of recently published abstracts support this high prevalence in the septic population as well. Several studies have also revealed that IAH is an independent predictor of renal failure and overall ICU mortality in a broad range of ICU patients.<sup>11,30</sup>

#### RECOGNITION AND MANAGEMENT OF IAH/ACS

Current literature relating to IAH/ACS presents us with a syndrome that impacts on 30–50% of all ICU patients; is an independent predictor of MOF; and has a mortality rate of 70–80% if allowed to progress untreated.<sup>11,16</sup> Indeed, IAH/ACS is beginning to mirror – if not eclipse – the clinical impact of ARDS, and is often unrecognised.<sup>4,11</sup> Despite this growing evidence base, our survey of intensivists on the diagnosis and management of IAH/ACS revealed that 23% of medical intensivists were unaware of a method of measuring IAP.<sup>31</sup> Further, a survey of intensivists in the United Kingdom concluded that ‘despite widespread awareness of IAH/ACS, many intensive care units never measure IAP’.<sup>32</sup>

In some regards, this may reflect the medical communities’ slow response to many evidence-based medical advances, e.g. low tidal volumes in ARDS or early goal-directed therapy in sepsis, which continue to have concerning levels of non-acceptance among practising physicians. In contrast, others may be awaiting the results of randomised control trials on IAH/ACS and consensus standards for management of this syndrome. Some

IAH/ACS experts have weighed in strongly with comments on this clinical approach, asking if ‘a century later – isn’t it time to pay attention?’<sup>33</sup> It is clear that critically ill patients are at risk of IAH/ACS and ‘paying attention’ begins with monitoring.

#### IAH/ACS monitoring

The massive effort directed at various types of monitoring in the ICU is focused on the early detection of organ compromise, in order to allow for intervention before organ damage occurs. As with any disease process, early recognition also allows for a significantly broader approach to treatment. IAH/ACS is no exception. Unrecognised IAH leads to ACS and, by definition, organ failure. Waiting for ACS and concomitant organ failure before intervening is comparable to allowing myocardial ischaemia to persist until myocardial infarction occurs.

Monitoring, prevention and early intervention are fundamental components of good critical care. With this paradigm in mind, opinion leaders in the management of IAH/ACS have strongly recommended that all critically ill patients should have IAP closely monitored.<sup>34,35</sup> These recommendations make clinical sense for several reasons. First, clinical examination of the abdomen has been shown to be grossly inaccurate in the assessment of intra-abdominal hypertension.<sup>36,37</sup> The use of bladder pressure measurement as a surrogate of IAP for early detection of IAH can lead to adjustments in therapy before permanent organ damage ensues, potentially avoiding the need for surgical decompression.

In contrast to the case with ARDS, where organ damage has occurred before radiological evidence or hypoxia presents, IAP monitoring provides an early warning of impending IAH/ACS. Lastly, as IAP rises, it has direct impact on other monitoring systems and clinical markers.<sup>17–19,24</sup> For example, fluid resuscitation and vasoactive medications are frequently titrated based on information gathered from central venous and pulmonary artery catheters (i.e. early goal-directed therapy in sepsis).<sup>38</sup> These catheters rely on the principle that pressure extrapolates to volume. When extrinsic pressures such as PEEP or intra-abdominal pressure are present, the principle of pressure equating to volume becomes flawed. Patients with significant IAP may present with elevated central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP) and decreased cardiac index. In this scenario, IAP falsely elevates CVP and PAOP and this information may be incorrectly interpreted as fluid overload and left ventricular failure. Without monitoring for IAP, clinicians cannot accurately interpret these haemodynamic parameters.

Urine output, another important marker of volume status, can also be misinterpreted in the setting of IAH. As IAP rises, direct mechanical pressure and increased renal venous pressure compromise glomerular blood flow leading to oliguria and anuria.<sup>22,23</sup> This oliguria, combined with analysis of urine electrolytes, would indicate a pre-renal condition in which fluid challenge might be selected for therapeutic management. In the setting of IAH, additional fluid may exacerbate visceral oedema, thus increasing intra-abdominal pressure and pushing the patient towards worsening abdominal compartment syndrome. Again, knowledge that intra-abdominal hypertension is present would be critical information in the interpretation of these important physiological data.



## IAP measurement

The clinical task of monitoring IAP can be accomplished using several different methods. The most reliable method is via pressure transduction through a catheter within the peritoneal cavity, though this approach has little clinical application. Other less invasive options include pressure transduction through a tube placed in the stomach, bladder or rectum. Of these options, bladder pressure has emerged as the simplest, most reliable method and has now been adopted as a standard by international consensus.<sup>16</sup> This method was originally described by Kron<sup>7</sup> and later modified by Cheatham and Safcek.<sup>39</sup> Detailed descriptions of these techniques can be reviewed in the original papers. Industry has also recognised the emerging need for accurate, easily used IAP monitoring capabilities and several devices are now commercially available, e.g. the Foley Manometer (Holtech Medical, Charlottenlund, Denmark); the AbViser (Wolfe Tory Medical, Salt Lake City, Utah, USA) (Figure 2); the IAP-Monitor (Spiegelberg GmbH & Co KG, Hamburg, Germany); and the CiMON (Pulsion Medical Systems AG, Munich, Germany). (For an exhaustive review of IAP monitoring methods see Malbrain<sup>40</sup>).

## IAH/ACS treatment

Many have suggested that the only true treatment for IAH/ACS is surgical decompression. This approach is probably a carry-over from the time when IAH was usually recognised in its extreme form of high pressure ACS, where emergent decompression was the only option and often still resulted in high mortality.

More recently, careful monitoring of IAP has resulted in early recognition of patients at risk of ACS, and new preventative therapies are beginning to emerge. Prevention and/or careful management of the 'second insult' has become the focus, rather than damage control in the setting of cardiorespiratory collapse. In other words, efforts to attenuate the rise of IAP can allow for preservation of gut perfusion resulting in prevention or minimisation of IAH/ACS-induced ischaemia and subsequent systemic inflammation. This approach should apply whether IAH/ACS is the primary or secondary insult.

While decompression laparotomy remains a critically important treatment option, these less invasive approaches may obviate the need for more aggressive and morbid interventions. Figure 3 represents an evidence-based monitoring protocol currently utilised in numerous intensive care units.

## Fluid management

Fluid resuscitation is a fundamental component of many disease processes found in the ICU. In fact, recent efforts in the management of sepsis have called for 'early goal-directed therapy'<sup>38</sup> in many forms, including fluid therapy. However, in most treatment protocols, the emphasis has been on the avoidance of under-resuscitation with little direction on the morbidity and mortality that can be caused by over-resuscitation.

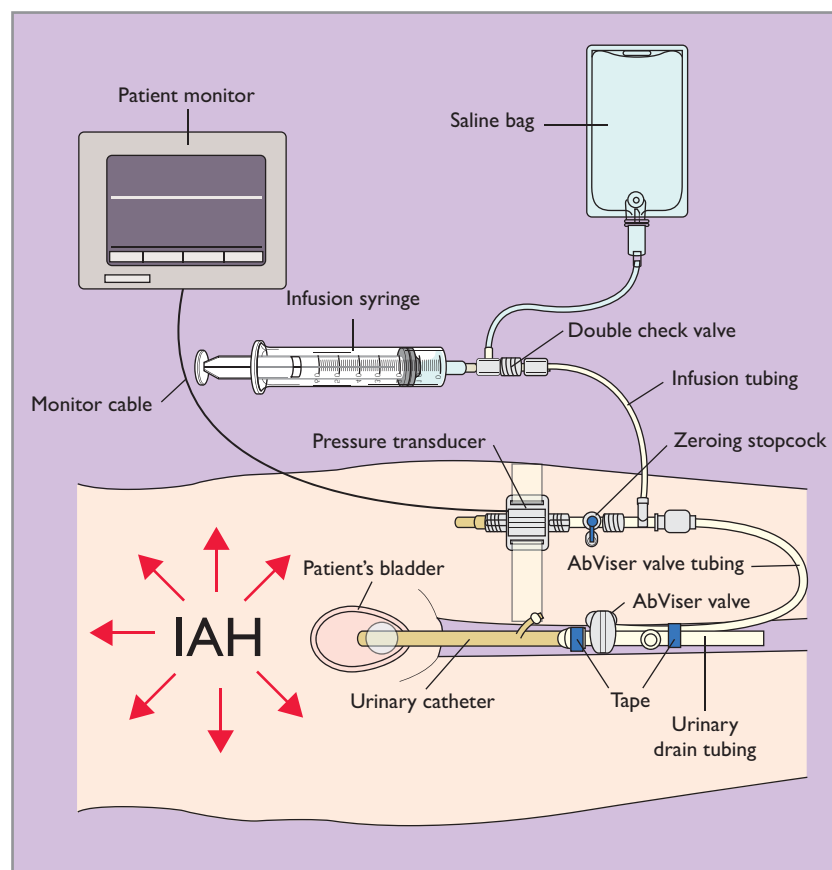
It is not uncommon for septic patients to receive in excess of 20 litres of fluid in less than 24 hours and then to hear clinicians tell the patient's family members that the resulting anasarca is merely a cosmetic problem that will resolve as the patient recovers. Nothing could be further from the truth, as the visible external oedema also reflects

the internal oedema in the mesentery and bowel wall. As this oedema worsens, IAP rises, compromising gut mucosal blood flow and resulting in a clinically silent ischaemia. Unrecognised, this can lead to a 'second insult' to the already critically ill patient. Without monitoring IAP, this early ischaemia will go unrecognised until ACS presents as multiple organ failure. At this point, even decompression will not prevent significant mortality. Resuscitation with crystalloid, even to carefully monitored goal-directed endpoints, can result in 'the salt-water vicious cycle' of crystalloid, capillary leak, visceral oedema and IAH/ACS.<sup>41</sup> As a result of this, new efforts in examining the role of colloids, isotonic saline and blood substitutes have shown promise.<sup>42-48</sup> Preliminary observations in our institution have demonstrated that albumin resuscitation can result in a reduction of IAP. At a minimum, more careful titration of resuscitation with optimisation of cardiac function and abdominal perfusion pressure is warranted in the setting of elevated IAP.

## Continuous ultrafiltration

In appropriately selected patients who will tolerate anticoagulation and continuous veno-venous haemodiafiltration (CVVHDF), there is emerging evidence that this therapy is effective in reducing IAP, eliminating inflammatory cytokines, reducing organ dysfunction and improving physiological parameters. Oda *et al.* investigated CVVHDF prospectively in patients with severe acute pancreatitis who were demonstrating progressive IAP elevation or increases in interleukin-6 levels.<sup>49</sup> They chose IL-6 because of the existing evidence of its link to bowel capillary hyperpermeability and they believed reduction of this cytokine would translate into reduced IAP and

Figure 2. The AbViser IAP monitoring kit.



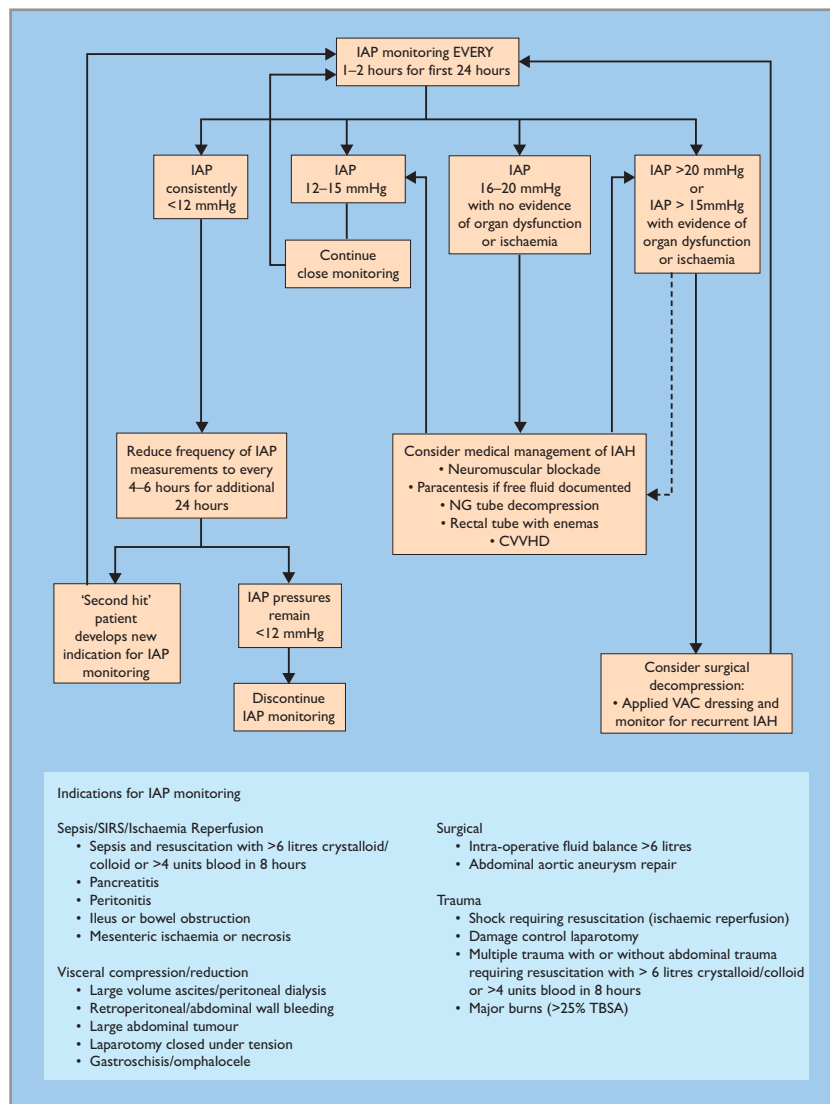


Figure 3. An evidence-based monitoring protocol.

improved outcomes. To maintain adequate circulating volume, they monitored CVP, lactate and colloid osmotic pressure and infused albumin to optimise these parameters. In all 17 cases studied, IAP and IL-6 were successfully reduced, with only one death in a patient who presented with established sepsis-induced MOF. Future studies will help clarify the subgroup of patients that may benefit from this intervention.

#### Sedation and neuromuscular blockade

Patients are most often at risk for IAH/ACS during the resuscitative phase of their illness. As IAP increases, pharmacological interventions can often relax the abdominal musculature, reduce IAP and improve abdominal perfusion pressure. At times, simple pain control and sedation is all that is needed. As the pressure continues to rise, neuromuscular blockade may also be effective.<sup>50</sup> We have demonstrated that the utilisation of neuromuscular blocking agents (NMBA) during this phase can significantly attenuate rising IAP, thus preventing full-blown ACS. Specifically, we have demonstrated that NMBA utilisation reduced IAP by an average of 9 mmHg in patients with elevated IAP. Of note, NMBA use had minimal effect on IAP in patients with abdominal pressures less than 14 mmHg.<sup>51</sup>

#### Paracentesis and bowel evacuation

A significant subgroup of patients suffering from IAH/ACS have intra-abdominal free fluid contributing to the rise in IAP. Several studies have demonstrated that ultrasound location and needle paracentesis drainage of this fluid can reduce intra-abdominal pressure and improve organ function.<sup>52,53</sup> This procedure can be performed repeatedly, or a stopcock drain system can be left in place for ongoing or intermittent drainage. Similarly, evacuation of bowel and/or stomach contents can reduce IAP. Nasogastric tubes should be standard therapy in all patients at risk of IAH. Colon evacuation can be accomplished with enemas and/or rectal tubes with a significant acute reduction in IAP.<sup>16</sup>

#### Initial operative management

For operative patients, early monitoring in the operating theatre of patients at risk of IAH/ACS is paramount. Patients who have a significant resuscitative debt, e.g. elevated lactate, oliguria, hypotension or ischaemia reperfusion, should be identified and, when necessary, left with an open abdomen and a vacuum-assisted 'vac pack' dressing in place (Figure 4). This allows for adequate resuscitation in the face of visceral oedema without the resultant IAH. Careful monitoring of even the 'vac pack' abdomen is important as up to 25% can re-accumulate pressure under these occlusive dressings, resulting in recurrent or tertiary ACS.<sup>54</sup> Recurrent ACS can easily be managed at the bedside with re-application of the 'vac pack' allowing for additional expansion of the oedematous viscera. In a recently abstracted 17-year review of ruptured abdominal aortic aneurysm (rAAA) patients from our institution, we found that, prior to June 2000, no patients with significant resuscitative debt (pre-operative hypotension, OR blood loss >6 L and crystalloid/colloid >12 L) returned to the ICU with 'open abdomens' (vac-pack dressings). After June 2000, 47% of comparable rAAA patients had intra-operative recognition of IAH risk and returned to the ICU with 'open abdomens'. Comparison of these two groups demonstrated a significant difference in post-operative mortality (first 24 hours) with a 26% mortality for closed abdomens compared to 0% mortality for open abdomens ( $p = 0.04$ ).<sup>55</sup>

#### Decompression laparotomy

As noted, surgical decompression remains an important intervention in patients who have not responded to non-invasive management or who are in the subgroup of the critically ill who rapidly progress to, or present with, ACS. For these patients, decompression laparotomy can be immediately restorative of cardiopulmonary stability. However, many clinicians allow ACS to persist for extended periods of time before turning to decompression, resulting in significant tissue ischaemia and/or necrosis and little long-term benefit in morbidity and mortality.<sup>12</sup>

Although there is no strict pressure threshold at which all patients have physiological compromise and should therefore be decompressed, patients with an IAP near 20 mmHg and early signs of organ failure should be seriously considered for urgent decompression.<sup>16,34,35</sup> Our survey of intensivists indicated that 35% would decompress the abdomen based on IAP elevation alone and 85% would proceed with decompression with IAP elevation and signs of one organ failure.<sup>31</sup> Early recognition and decompres-



Figure 4. A 'Vac-Pac' Dressing.

sion minimises the 'second insult' inflammatory response and therefore reduces capillary leak and visceral oedema. This approach allows for a shorter SIRS response and faster resolution of third-space fluids with expedited closure of the abdomen. In our experience early intervention can result in primary closure in 5–7 days, a stark contrast to the protracted open abdomens of the past that required mesh, skin grafting and multiple delayed surgeries before definitive closure. Recent advances in vacuum-assisted dressings and drainage techniques have also contributed to improved outcomes.<sup>56,57</sup> Overall, decompression laparotomy for intra-abdominal hypertension management has become a far less morbid procedure than it was even five years ago.<sup>56,57</sup>

### CONCLUSION

In summary, IAH/ACS is a disease process that has been recognised for over a century. A slow but growing recognition of IAH/ACS clinical prevalence has occurred over the past two decades. It is not clear whether this is due to a realisation of a pathophysiology that was always there or to advances in resuscitation that have delivered an ever-increasing number of patients who have survived initial insults, allowing for a second hit of uncontrolled systemic inflammation. For whichever reason, this increased prevalence has led to a near exponential increase in IAH/ACS literature over the past 10 years (Figure 1).

As with any prevalent disease process, the first step in confronting IAH/ACS is to arrive at consensus definitions of the pathophysiology and diagnostic criteria. Despite research on IAH/ACS spanning more than a century, the first attempt at broad consensus definitions occurred in 2004 at the inaugural World Congress on Abdominal Compartment Syndrome in Noosa, Australia.<sup>16</sup> These definitions will help standardise research protocols and management algorithms. With the growing prevalence of the syndrome, these standards are urgently needed. IAH/ACS is clearly an ICU problem and it will fall to intensivists to be the thought-leaders in research and clinical management of this morbid disease process.

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**CORRESPONDENCE TO:**

**Edward J Kimball MD, MSc**  
**Assistant Professor of Surgery and Emergency**  
**Medicine**  
**University of Utah**  
**Health Sciences Center**  
**50 North Medical Drive**  
**Salt Lake City**  
**Utah 84132, USA**  
**E-mail: Edward.Kimball@hsc.utah.edu**