Intra-Abdominal Hypertension – An Intensive Care Perspective

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Abstract

**Introduction**: Intra-abdominal hypertension is now well recognized in intensive care patients. Increasing knowledge of the incidence, causes, pathophysiology and outcome of intra-abdominal hypertension has resulted in earlier and more definitive management of the condition in the last decade. Although these advances appear to have improved outcome, many issues remain controversial.

**Methods**: A literature review of relevant papers was conducted.

**Conclusions**: The incidence of intra-abdominal hypertension varies widely depending on the intra-abdominal pressure used to define the condition and the sub-group investigated. Monitoring intra-abdominal pressure by measuring urinary bladder pressure is simple and sufficiently accurate. It has been suggested that intra-abdominal hypertension occurs when intra-abdominal pressure exceeds 10 to 12 mmHg. Abdominal compartment syndrome is a severe form of intra-abdominal hypertension and occurs when intra-abdominal pressure exceeds 20-25 mmHg and associated organ failure is evident. Intra-abdominal hypertension has potentially damaging effects on the gastrointestinal, cardiovascular, renal, respiratory and central nervous systems. There is an association between intra-abdominal hypertension and multiple organ failure, but causation has not yet been convincingly shown. Expert consensus suggests that an acute increase of IAP to above 20-25 mmHg and/or evidence of abdominal compartment syndrome is an indication for surgical decompression.

Introduction

Prior to the advent of modern intensive care management most patients who developed intra-abdominal hypertension (IAH) rapidly died from the associated organ failure. Mechanical ventilation and other organ support now allows patients to survive long enough for the condition to warrant specific management. The rapidly growing recognition of IAH in intensive care units has led to numerous recent observational reports in humans and experimental studies in animals.

Intra-abdominal pressure (IAP) varies inversely with intrathoracic pressure and during spontaneous ventilation is normally atmospheric or slightly negative [1]. Mechanical ventilation usually increases the IAP measurement as it becomes similar to pleural pressure. Adverse pathophysiological effects begin to appear when IAP exceeds 10 mmHg. When using a conservative definition of an IAP of 12 mmHg or greater to define IAH, one investigator reported the incidence of IAH to be about 20% in medical intensive care patients [2]. The rate reported in surgical populations is generally higher, and may even be as high as 30% in patients undergoing major emergency surgery [3]. Depending on the definition used, however, rates vary dramatically, and a recent prospective trauma intensive care series, utilizing a cut-off of 20 mmHg, reported a rate of only 2% [4].

Progressive increases in IAH eventually results in abdominal compartment syndrome (ACS), which is considered present when IAP > 25 mmHg is associated with evidence of renal, cardiovascular, respiratory or other associated organ failure [5-7]. The incidence of ACS is unknown in medical patients but ranges from about 1-15% in surgical intensive care series [4,8,9,10].

Measurement

Kron et al. [11] first reported a method by which
to measure IAP at the bedside with the use of an indwelling urinary bladder catheter. Sterile saline (50-100 ml) was injected into the empty bladder through the Foley catheter. The tubing of the urinary drainage bag was cross-clamped just distal to the culture aspiration port. The clamp was released just enough to allow the tubing proximal to the clamp to drain fluid from the bladder, then reapplied. A 16-gauge needle was inserted into the culture aspiration port to Y-connect to a manometer. The level of the symphysis pubis was used as the zero point with the patient supine. Modifications of this intravesical pressure recording method, are usually used for measuring IAP in the clinical setting [5,7,11,12]. Alternative sites of measurement and techniques have been described, however measurement of urinary bladder pressure has gained wide clinical acceptance and application.

Aetiology and pathophysiology

Intra-abdominal hypertension occurs when foreign bodies such as surgical packing, blood, edematous viscera, and fluid loss into the abdominal space exceed the capacity of the abdominal cavity [8,13]. Common surgical causes of IAH are peritonitis or abscesses, ileus, intestinal obstruction, massive intra-abdominal hemorrhage, pancreatitis, tension pneumoperitoneum, post-surgical edema and IAH associated with massive fluid resuscitation following major burn injury. There are also well established medical causes of IAH, of which massive fluid resuscitation for hemorrhagic or distributive shock is the most frequently encountered in the intensive care unit [14].

An increase in IAP is accompanied by changes within and outside the abdominal compartment. Adverse effects of IAH are usually the result of mechanical factors. Associated neurohumoral changes have not been adequately explored or quantified, but almost certainly contribute to the observed effects.

Effects of IAH within the abdominal compartment

Maintenance of splanchnic perfusion is thought to be important in critically ill patients because of the association between ischemia, increased gut permeability, intestinal bacterial translocation, hepatic function and the subsequent development of multiple organ failure and death [15-17]. High intra-abdominal pressure causes decreased regional splanchnic blood flow in animals at intra-abdominal pressures as low as 10-15 mmHg [18,19]. Hepatic perfusion becomes compromised at similar pressures [18,19]. These changes are initially small, approximately 20% flow reduction from baseline, however once IAP rises above 20-25 mmHg, the decrease in blood flow is significant and mesenteric artery blood flow is as low as 30% of baseline at an IAP of 40 mmHg [20]. Intestinal mucosal blood flow is also decreased and this decrease is associated with the presence of a low intramucosal gastric pH (pHi), which supports the presence of mucosal ischemia [18]. These effects are worsened by hypovolemia and hypotension [21]. The link between splanchnic hypoperfusion and multiple organ failure is not so clear. A recent study in pigs investigated following a hemorrhagic insult, while confirming the decrease in superior mesenteric blood flow and decrease in pHi in response to IAH, failed to demonstrate evidence of bacterial translocation [22]. A rat study, however, demonstrated the release of inflammatory cytokines in response to induced IAH followed by decompression [23].

The only indirect evidence that supports the occurrence of splanchnic hypoperfusion in humans is provided by observations that a low pHi in patients with IAH returns towards normal with decompression [24,25]. In summary, the causative relationship between IAH, splanchnic hypoperfusion, bacterial translocation, cytokine release and subsequent multiple organ dysfunction remains unproved.

The main conduit for blood flow from the abdomen is the inferior vena cava. Abdominal perfusion pressure (APP), can then theoretically be determined by subtracting inferior vena cava pressure, from mean arterial pressure (MAP), thus APP = MAP - IVCP. At IAP below 20-25 mmHg, inferior vena cava pressure is generally equal to right atrial pressure [26], and therefore, APP = MAP - right atrial pressure. When IAP exceeds 20-25 mmHg, inferior vena cava pressure approaches IAP, rather than right atrial pressure (unpublished observations). Therefore, when IAP exceeds 20-25 mmHg, APP = MAP - IAP. It is interesting to speculate whether abdominal per-
fusion pressure could be increased to improve splanchnic and retroperitoneal organ perfusion in patients with IAH.

**Effects of IAH outside the abdominal compartment**

As IAP increases and is transmitted the retroperitoneum, renal blood flow and glomerular filtration rate decreases. In animals, ureteric stenting has no beneficial effect, and improving cardiac output and blood pressure does not completely reverse renal dysfunction, suggesting other mechanisms are responsible for the changes seen. Direct parenchymal compression, caval or renal vein obstruction leading to increased vascular resistance, reduction in aortic or regional arterial blood supply and neurohumoral dysfunction have all been suggested as possible mechanisms. More recent animal studies suggest that renal vein compression is responsible for the decreased glomerular filtration [27].

Abnormal renal function in humans is usually manifested by oliguria and once IAP exceeds 25 mmHg the majority of patients are unable to produce urine flow of > 0.5ml/kg/h [8]. In a human study raised IAP (>18mmHg) has been shown to be independently associated with post-operative renal impairment [28], and decompression in critically ill patients has been shown to reverse renal impairment if achieved early (within 10-24 h) [10,29]. It should be noted that decompression does not always lead to an improvement in renal function, suggesting that permanent damage may occur if decompression is delayed [30].

The effects of IAH on the respiratory system are primarily the result of transmission of high abdominal pressures to the diaphragm and then chest cavity, causing diaphragmatic elevation and changes in shape [31]. Decreased static and dynamic pulmonary compliance result in ventilatory difficulty [32]. Decreases in functional residual capacity, residual volume and total lung capacity, probably the result of compression atelectasis, lead to ventilation perfusion abnormalities. The institution of mechanical ventilation is usually necessary and high airway pressures and/or low tidal volumes become unavoidable [11]. The magnitude of the effect is generally proportional to the rise in IAH, and are reversed by decompression in critically ill patients [31,33]. It has been suggested that pulmonary vascular resistance may be increased by a direct pressure effect on the pulmonary vessels, regional alveolar hypoxia and possibly the release of vasoactive substances [7].

The cardiovascular system is sensitive to increases in IAP. Increased intrathoracic pressure (>15mmHg), transmitted from the abdomen via the diaphragm, results in increases in all conventionally measured vascular and intra-cardiac pressures. In animals and critically ill patients, central venous pressure, pulmonary occlusion pressure and right atrial pressure all increase in a graded way with rising IAH [26,34]. There may be a transient increase in venous return and cardiac output in the initial phases of a rising IAP [35]. Once established, however, elevated intra-abdominal pressure is associated with a decrease in vascular and cardiac volumes in all animal studies as a consequence of decreasing transmural pressures and reduced venous return from the abdomen and lower limbs [31,34]. The low preload causes a reduction in cardiac output [31,34]. Blood pressure may be initially maintained because there is an increase systemic vascular resistance and afterload, probably a result of compression of the aorta and major arterial conduits in the thorax and abdomen. At consistently high IAP, the effect on cardiac output becomes more profound and blood pressure cannot be maintained. Hypovolemia, hemorrhage, and the application of PEEP exacerbate the deleterious effects of IAH in animals [21,36].

Decompression reverses most, but not all, cardiovascular abnormalities recorded in critically ill patients [8,10,24,37].

The consequences of IAH may be seen in organs as apparently distant as the central nervous system. In the abdomen, the IAH increases all venous pressures, and this inhibits venous drainage from the central nervous system via the lumbar plexus. Additionally, in the chest, the raised intrathoracic pressure transmitted from the abdomen causes a functional obstruction to cerebral venous outflow via the internal jugular veins. In susceptible patients this has the potential to increase cerebral blood volume and raise intracranial pressure. In an animal model, sternotomy resulted in a return to baseline of both intrathoracic and intracranial pressures [38]. Increases in intracranial pressure secondary to IAH may be clinically significant in critically ill patients with decreased intra-
cranial compliance and decompression is necessary to reverse the changes [39-41].

**Predictive factors and treatment in the intensive care unit**

It is important to identify patients at high risk for the development of IAH. Certain surgical patients such as those who have undergone procedures like damage control laparotomy, liver transplantation, or laparotomy for life threatening abdominal trauma appear to have a higher incidence of IAH [25,42]. The condition is not, however confined to surgical patients and IAH has also been described in burn populations and medical patients [2]. It is therefore important to identify factors that are associated with, and if possible, can predict the development of IAH. Recently a sufficiently positive 24 hour fluid balance was identified as predictive of severe IAH [43]. Supranormal resuscitation for trauma has also recently been shown to be associated with a higher incidence of IAH and ACS [44]. Closer monitoring of critically ill patients at high risk may allow early intervention and prevent the consequences of IAH and ACS.

Intra-abdominal hypertension and pH are strongly associated with, and seem to predict, the same adverse outcomes - shock, renal failure, sepsis, need for re-laparotomy and death [25,45]. Animal studies suggest that decreased gut perfusion occurs but the relationship with bacterial invasion and delayed multiple organ dysfunction is unclear. Although central cardiovascular pressures are increased with IAH, the ability of these measurements to estimate important clinical end points such as preload and cardiac output is diminished. Preload, cardiac output and systemic oxygen delivery are therefore not only difficult to determine, but are themselves likely to be compromised by IAH. Patients with IAH must therefore be appropriately fluid volume resuscitated to increase cardiac output to optimize end organ perfusion, rather than to achieve arbitrary central venous pressures. Volumetric indices of preload such RVEDVI may be a useful guide to appropriate preload estimation, as over-hydration may be a risk factor for worsening IAH. Even in patients with normal systemic cardiovascular indices, IAH causes a decrease in glomerular filtration rate at moderate levels of IAH and anuria with renal damage at higher abdominal pressures, unless early decompression is achieved. IAH increases intracranial pressure, which may have important effects in critically ill patients with cerebral injury. Acute increases in IAP may be the cause of neurological co-morbidity in multiple trauma patients without obvious signs of head trauma, and abdominal decompression may normalize ICP and cerebral perfusion pressure. IAH affects the ability to mechanically ventilate critically ill patients, a complication that can only be satisfactorily reversed by decompression.

**Definitive management and decompression**

Abdominal decompression is the only definitive treatment for IAH. The outcome of clinical series of patients who underwent decompression compared with those who did not is shown in table 1 [7-10,14,24,29,37,46]. It appears that left untreated IAH is likely to increase mortality, and certainly untreated ACS is usually fatal. Recommendations for therapy are based on these retrospective observations, uncontrolled prospective studies and expert opinion. The exact level of IAH that defines "critical IAP" and warrants decompressive intervention remains subject to debate, but there is expert consensus that decompression should be performed at levels of IAP above 20-25 mm Hg. This would appear to be a sound principle, based on the pathophysiology presented above. However, based on the observation that splanchnic perfusion is compromised at an IAP as low as 10-12 mmHg and the observation of associated gastric intramucosal acidosis in humans, this number may have to be revised to a lower figure. Currently, a modified general therapeutic approach based on a grading by Meldrum et al. [8] appears reasonable (Table 2). At intra-abdominal pressures of less than 25 mmHg, attempts to increase the abdominal perfusion pressure could be additionally considered in an attempt to improve splanchnic perfusion [47]. In the absence of published data an APP of approximately 70mmHg is arbitrarily suggested.

Based on current knowledge, the optimal timing for decompression of IAH is controversial, however, established ACS has become a well-recognized clinical entity and should be considered an emergency, requiring immediate decompression [8]. Close monitoring of IAP may be important to provide early warning of progressive IAH and facilitate decompression before classical ACS develops.

In operative high-risk surgical patients, prevention
Table 1. Effects of decompression in patients with abdominal compartment syndrome

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study population</th>
<th>Study type</th>
<th>No. of patients in series</th>
<th>ACS criteria</th>
<th>Patients with ACS (%)</th>
<th>Mortality Decompressed</th>
<th>Not decompressed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hong⁴ 2002</td>
<td>Trauma intensive care patients</td>
<td>prospective</td>
<td>706</td>
<td>IAP&gt;20mmHg and MODS</td>
<td>1.00</td>
<td>3/6 (50%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Ertel¹⁰ 2000</td>
<td>Damage control laparotomy for abdominal or pelvic trauma</td>
<td>retrospective &amp; prospective</td>
<td>311</td>
<td>Clinical and IAP&gt;25mmHg</td>
<td>5.50</td>
<td>6/17 (35%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Maxwell¹⁴ 1999</td>
<td>Fluid resuscitation No abdominal injury</td>
<td>prospective</td>
<td>46</td>
<td>Clinical</td>
<td>13</td>
<td>4/6 (67%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Cheatam⁶ 1999</td>
<td>Trauma, vascular and major surgery</td>
<td>prospective</td>
<td>20</td>
<td>Pre-operative intra-abdominal pressure average 38mmHg</td>
<td>100</td>
<td>15/20 (75%)</td>
<td>9/9 (100%)*</td>
</tr>
<tr>
<td>Chang²⁴ 1998</td>
<td>Severe abdominal trauma</td>
<td>prospective</td>
<td>11</td>
<td>Clinical and IAP&gt;25mmHg</td>
<td>100</td>
<td>7/11 (63%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Meldrum⁸ 1997</td>
<td>Severe trauma, post -laparotomy</td>
<td>prospective</td>
<td>145</td>
<td>Clinical and IAP&gt;20mmHg</td>
<td>14</td>
<td>6/21 (29%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Meldrum³⁷ 1995</td>
<td>Hepatic injury, with peri-hepatic packing</td>
<td>retrospective</td>
<td>11</td>
<td>Not described</td>
<td>100</td>
<td>5/11 (45%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Morris⁹ 1993</td>
<td>Staged celiotomy after major abdominal surgery</td>
<td>retrospective</td>
<td>107</td>
<td>Clinical, plus improvement after decompression</td>
<td>15</td>
<td>9/14 (64%)</td>
<td>1/1 (100%)</td>
</tr>
<tr>
<td>Platell²⁹ 1990</td>
<td>Abdominal aortic surgery</td>
<td>prospective</td>
<td>22</td>
<td>Clinical and IAP &gt;18mmHg</td>
<td>45</td>
<td>6/10 (60%)</td>
<td>0/0</td>
</tr>
<tr>
<td>Saggi⁵ 1997</td>
<td>Review of reports prior to 1990</td>
<td>retrospective</td>
<td>27</td>
<td>Not described</td>
<td>100</td>
<td>11/21 (52%)</td>
<td>6/6 (100%)</td>
</tr>
</tbody>
</table>

* Nine patients developed recurrent compartment syndrome after decompression - all died. These patients are included in the 15 deaths noted in the previous column.

Table 2. Possible therapeutic approach based on the severity of intra-abdominal hypertension [8,46].

<table>
<thead>
<tr>
<th>Bladder Pressure (mm Hg)</th>
<th>Recommendation</th>
</tr>
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<tbody>
<tr>
<td>10-15</td>
<td>Maintain normovolaemia</td>
</tr>
<tr>
<td>16-25</td>
<td>Hypervolaemic/Hypertensive resuscitation*</td>
</tr>
<tr>
<td>26-35</td>
<td>Decompression</td>
</tr>
<tr>
<td>&gt;35</td>
<td>Decompression and reexploration</td>
</tr>
</tbody>
</table>

* Aim for APP of 70mmHg, see text
of IAH by pre-emptive techniques such as the utilization of prosthetic anterior abdominal wall extensions would appear to be a more efficient way of preventing the adverse effects of IAH and ACS. Recent series evaluating surgical techniques of preventing IAH in very high risk patients appear promising, but are do not provide a definitive answer [48-53].

Conclusion

Intra-abdominal hypertension has been demonstrated in critically ill medical and surgical patients and causes detrimental pathophysiological effects on multiple organ systems. Outcome may be worsened by IAH and patients at high risk should be routinely monitored to allow earlier intervention and prevent the development of severe, established ACS. Understanding the pathophysiology of raised IAP allows intensive care management and organ support to be most effectively applied. While some progress has been made in describing and managing this clinical entity, further study is needed to identify specific factors that place patients at high risk, improve our understanding the clinical implications and confirm appropriate management strategies.

References

3. Malbrain MLNG: Relationship of body mass index (BMI), lactate and intra-abdominal pressure (IAP) to subsequent mortality in ICU patients. Crit Care 1999; 3(suppl 1):20


