

Intra-abdominal Hypertension—Implications for the Intensive Care Physician

G M Joynt,*^{MBBCh, FFA(SA)(Crit Care)}, S J Ramsay,**^{MBBS, FRCA}, T A Buckley,***^{MBChB, FFICANZCA, FICANZCA}

Abstract

Introduction: Intra-abdominal hypertension is becoming increasingly recognised in intensive care patients. The objective of this paper was to review the modern published literature to establish a representative consensus view of the incidence, causes, pathophysiology, management and outcome of intra-abdominal hypertension. **Methods:** A computerised MEDLINE search from 1966 to November 2000 was conducted using the Medical Subject Heading and textwords “abdominal”, “compartment syndromes”, “intra-abdominal” and “hypertension” and “pressure”. The references of recent articles were checked for additional relevant citations. Papers were independently assessed for relevance by authors SJR and GMJ using a data collection format. Data were assessed qualitatively and papers detailing physiologic variables, effects of decompression and outcome were abstracted in table form. **Conclusions:** Intra-abdominal hypertension exists when intra-abdominal pressure exceeds 10 to 12 mmHg as physiological aberrations are manifest above this pressure. Incidence is 5% to 40% in high-risk surgical patients. Pathophysiological changes caused by intra-abdominal hypertension include effects on the gastrointestinal, cardiovascular, renal, respiratory and central nervous systems. An association between intra-abdominal hypertension and subsequent multiple organ failure has been repeatedly shown, although causation remains unproven. Monitoring intra-abdominal pressure (IAP) by measuring urinary bladder pressure is easy and accurate. However, it is unclear what level of intra-abdominal pressure requires intervention (decompression), or what length of time intra-abdominal hypertension can be tolerated before significant end organ damage occurs. In the absence of good outcome data, expert consensus is that an acute increase of IAP to above 20 to 25 mmHg and/or evidence of abdominal compartment syndrome warrants urgent decompression. While some progress has been made in describing and managing this clinical entity, further studies are needed to fully understand the clinical implications and confirm appropriate management of this condition.

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Introduction

Although the detrimental effects of raised intra-abdominal pressure have been known for over 100 years, it is only in the last two decades that the clinical importance of these changes are being recognised.¹⁻⁷ This coincides with the increasing availability of intensive care, which allows many more patients to survive long enough with intra-abdominal hypertension (IAH) to suffer its consequences. While a growing recognition of IAH has resulted in a rapid increase in the number of published observations in animals and humans, there is still much about the aetiology, pathophysiology, consequences and management of IAH that is either controversial or simply not known.⁸

Normal intra-abdominal pressure (IAP) is atmospheric or slightly negative and fluctuates inversely with intra-

thoracic pressure during spontaneous ventilation.⁹ In intensive care many patients are receiving mechanical ventilation during which IAP approaches pleural pressure and becomes positive. Intra-abdominal hypertension can be said to occur when the IAP exceeds 10 mmHg because significant adverse pathophysiological effects can be demonstrated at or above this pressure.^{7,10} The incidence of IAH may be higher than previously suspected and varies according to the exact cut-off pressure used for the definition as well as the population of patients under investigation. The incidence of IAH has been reported to be about 20% in medical intensive care patients,¹¹ 30% in the surgical intensive care population, and may even be higher in patients undergoing major emergency surgery.¹² Abdominal compartment syndrome (ACS) is a severe consequence of

* Associate Professor and Director

** Assistant Professor

Department of Anaesthesia and Intensive Care
The Chinese University of Hong Kong

*** Consultant

Intensive Care Unit
Prince of Wales Hospital

Address for Reprints: Dr Gavin M Joynt, Department of Anaesthesia and Intensive Care, Prince of Wales Hospital, Sha Tin, NT, Hong Kong SAR.
E-mail: gavinmjoynt@cuhk.edu.hk

progressive IAH. It is unique among compartment syndromes because of the profound systemic effects with which it is associated. Abdominal compartment syndrome is considered present when a high intra-abdominal pressure (IAP >25 mmHg) is associated with evidence of renal, cardiovascular, respiratory or other associated organ failure.^{6,13,14} In patients undergoing major surgery, the incidence of ACS ranges from 5.5% to 15%.^{7,14-16}

The reported incidence of IAH and abdominal compartment syndrome implies that IAH and ACS will be encountered quite frequently in general intensive care units. The objective of this article is to address IAH from the perspective of an intensive care practitioner. Issues that potentially affect intensive care based organ support are addressed in more detail, and management recommendations are focused mainly on those that can be influenced by the intensive care practitioner.

Measurement of Intra-abdominal Pressure

Bedside measurement of IAP in the intensive care unit can be accomplished by a variety of means. Transduction of pressures from indwelling rectal, gastric, intra-peritoneal and urinary bladder catheters have all been used. In 1984, Kron et al⁵ reported a method by which to measure IAP at the bedside with the use of an indwelling Foley catheter. Sterile saline (50 to 100 ml) is injected into the empty bladder through the Foley catheter. The tubing of the urinary drainage bag is cross-clamped just distal to the culture aspiration port. The end of the drainage bag tubing is connected to the Foley catheter. The clamp is released just enough to allow the tubing proximal to the clamp to drain fluid from the bladder, then reapplied. A 16-gauge needle is then used to Y-connect a manometer or pressure transducer through the aspiration port of the tubing of the drainage bag. The top of the symphysis pubis is used as the zero point with the patient supine. Modifications of this intravesical pressure recording method are now an accepted standard for measuring IAP in the clinical setting (Fig. 1).^{5,6,14,17} An alternative bedside technique has been described in which intragastric pressure measurements are taken from an indwelling nasogastric tube.¹⁸ This method has been validated and found to vary within 2.5 cmH₂O of urinary bladder pressures. Of these techniques, however, measurement of urinary bladder pressure has gained widest clinical acceptance and application.

Aetiology

Intra-abdominal hypertension occurs when blood, oedematous viscera, or third space losses exceed the capacity of the abdominal cavity. Foreign bodies, such as packing, following “damage control” laparotomy may occupy a significant part of the intra-abdominal compartment.^{7,19} Common causes of IAH are given in

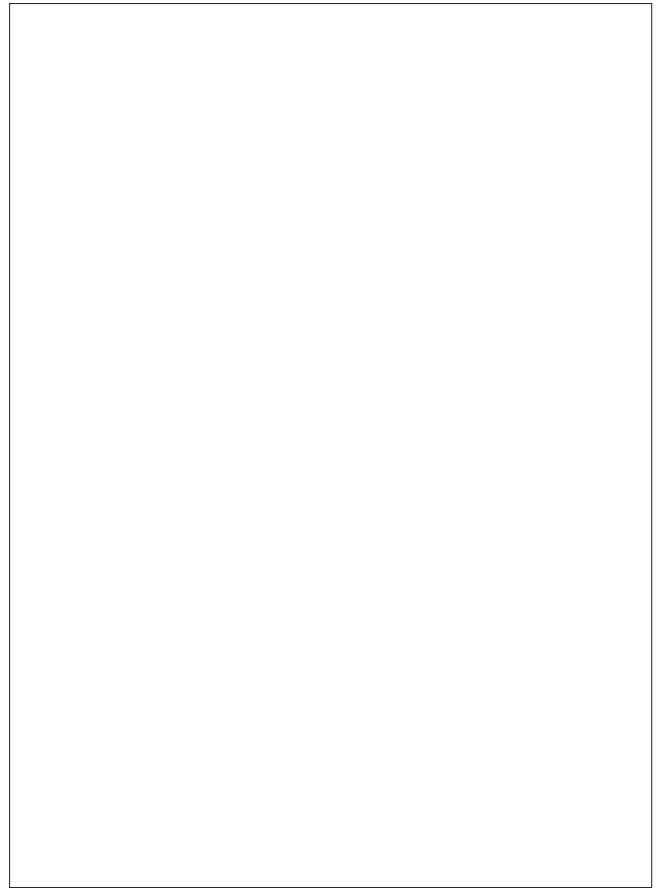


Fig. 1. Picture demonstrating a simple system for measuring intra-abdominal pressure via a Foley catheter. A short piece of rubber tubing connects a Y-piece to a three-way tap which allows connection to a pressure transducer or graduated fluid chamber. An artery clamp is used to occlude the outflow of urine during bladder filling with 50 ml of normal saline and subsequent measurement. The “system” is sterile and does not have to be disconnected between measurements.

Table I. Although frequently overlooked, there are well-established medical causes of IAH, of which massive fluid resuscitation for haemorrhagic or distributive shock is the most frequently encountered in the intensive care unit.^{11,20}

Pathophysiology

An increase in intra-abdominal pressure is accompanied by changes within and outside the abdominal compartment. Available experimental studies and clinical observations suggest that most of the adverse effects of IAH are the result of mechanical factors. There must, of course, be associated neurohumoral changes that influence the changes seen with IAH, however, little data exist to quantify these effects. Intra-abdominal hypertension results in decreased perfusion to abdominal and retroperitoneal organs, compromises cardiovascular and respiratory function and has deleterious effects on intracranial pressure. Although all are interdependent to a greater or lesser extent, for the purposes of clarity, each is discussed separately.

TABLE I: RISK FACTORS FOR ACUTE INTRA-ABDOMINAL HYPERTENSION IN ICU PATIENTS

Medical	High volume fluid resuscitation, particularly for septic shock; decompensated cirrhosis with ascites
Surgical	Peritonitis; ileus; intestinal obstruction; ruptured abdominal aortic aneurysm; intra-abdominal abscess; tension pneumoperitoneum; acute pancreatitis; mesenteric venous thrombosis; acute gastric dilatation; intraperitoneal haemorrhage; pneumatic antishock garments
Postoperative	Intraperitoneal or retroperitoneal bleeding; post-resuscitation visceral oedema; massive transfusion; abdominal packing; reduction of a massive hernia; abdominal closure under tension

The Abdominal Compartment

As pressure within the abdomen increases, it is transferred to all the tissues and vascular structures and even the abdominal wall is affected.²¹ The abdomen then acts as a fluid compartment and follows the laws of Pascal. It is the effect of the high intra-abdominal pressures on regional blood flow that has the most important implications.²² In animal experiments, decreased regional splanchnic blood flow has been demonstrated at intra-abdominal pressures as low as 10 to 15 mmHg^{23,24} (Table II). Hepatic perfusion becomes compromised at similar pressures.^{23,24} While the magnitude of these changes is initially small (<20% flow reduction from baseline), by the time the IAP rises above 20 to 25 mmHg, the reductions are substantial.²⁵ Mesenteric artery blood flow is as low as 30% of baseline at an IAP of 40 mmHg. These changes were mirrored by a decrease in intestinal mucosal blood flow and a low intramucosal gastric pH (pHi), which also supported the presence of mucosal ischaemia.²³ It is important to note that these changes occurred under conditions of stable mean arterial pressure and cardiac output. As expected the effects are exacerbated by hypovolaemia and hypotension.²⁶ A decreased pHi in patients with IAH and a return towards normal with decompression provides indirect evidence supporting the occurrence of splanchnic hypoperfusion in humans.^{27,28} Maintenance of gut perfusion is thought to be especially important in critically ill patients because of the

association between ischaemia, increased gut permeability, intestinal bacterial translocation and the subsequent development of multiple organ failure and death.²⁹⁻³¹

The relationship between inferior vena cava pressure (IVCP) and right atrial pressure (or thoracic central venous pressure) is interesting. In animals, inferior vena cava pressure approximates IAP and differs markedly from right atrial pressure, especially as IAP rises.^{9,32} For this reason it has been suggested that IAP can be measured by transducing intra-abdominal venous pressures. While this may be true in animal studies, in most intensive care patients, intra-abdominal venous pressures (inferior vena cava and internal iliac venous pressures) reflect right atrial pressure and not IAP.^{33,34} It is only when IAP becomes very high (probably >20 to 30 mmHg), that the normally close correlation between venous intra-abdominal pressures and right atrial pressure is lost. The agreement between intra-abdominal and RAP pressure relies on the maintenance of a continuous fluid column from the right atrium to the inferior vena cava. This agreement is lost when the inferior vena cava collapses and there is CT scan evidence that this occurs at a high IAP.³⁵ The pressure measured in the inferior vena cava then becomes the same as the external pressure that has caused the vessel to collapse, namely the pathologically raised intra-abdominal pressure. Although some pressure is transmitted to the right atrium, the very high resistance to flow in the collapsed inferior vena cava dissipates much of this pressure. The situation in animals and humans is then similar, with inferior vena cava pressure reflecting IAP and not right atrial pressure. Under these conditions of very low venous outflow, a vicious cycle of rising abdominal pressure, abdominal tissue oedema and decreased venous return could be expected to ensue with the subsequent development of classical abdominal compartment syndrome (ACS). This will inevitably lead to catastrophic tissue hypoperfusion and ischaemia. Clinical reports of abdominal compartment syndrome suggest that if not decompressed, patients in this category inevitably die.⁶

The main conduit for blood flow from the abdomen is the inferior vena cava. Abdominal perfusion pressure (APP)

TABLE II: PHYSIOLOGICAL CHANGES WITH DIFFERENT DEGREES OF INTRA-ABDOMINAL HYPERTENSION, ANIMAL DATA

System	Intra-abdominal pressure	Physiological effect	Animal model	Source
Gastrointestinal	>10 mmHg	Mesenteric & mucosal blood flow reduced by 30% to 40%	Pig study, MAP & CO maintained by IV fluid, arterial flow measured by ultrasonic flow probe, mucosal flow by laser Doppler	Diebel 1992 ²³
	>15 mmHg	Fall in bowel tissue oxygen partial pressure from 43 to 31 mmHg	Pig study, PO ₂ measured by fluorescence quenching optodes in ileal submucosa, MAP unchanged but CO decreased	Bongard 1995 ²⁵

TABLE II. contd.

System	Intra-abdominal pressure	Physiological effect	Animal model	Source
Gastrointestinal	>20 mmHg	Bacterial translocation and loss of intestinal barrier function	Rodent study, IAP elevated for 60 mins, MAP maintained with IV fluids	Diebel 1997 ²⁹
	>20 mmHg	Progressive decline in mesenteric & mucosal blood flow from 40% to 70%; severe gut mucosal acidosis	Pig study, MAP & CO maintained by IV fluid, arterial flow measured by ultrasonic flow probe, mucosal flow by laser Doppler	Diebel 1992 ²³
	>25 mmHg	Further fall in bowel tissue oxygen partial pressure from 24 to 12 mmHg	Pig study, PO ₂ measured by fluorescence quenching optodes in ileal submucosa, MAP unchanged but CO decreased	Bongard 1995 ²⁵
	All pressures	Adrenal blood flow maintained	Dog study, organ blood flow measured with radio-active biospheres	Caldwell 1987 ²²
Abdominal Wall	10 to 40 mmHg	Progressive decrease in rectus muscle blood flow and poor abdominal wound healing	Pig study, laser Doppler probe, MAP maintained with IV fluids	Diebel 1992 ²¹
Hepatic	>10 mmHg	39% decrease in hepatic arterial blood flow	Pig study, blood flow measured by Doppler flow probe, microcirculatory flow by laser flow probe. MAP and CO constant	Diebel 1992 ²⁴
	>20 mmHg	Microcirculatory blood flow (29%), and portal vein flow (34%) decreased. Splanchnic vascular resistance high	Pig study, blood flow measured by Doppler flow probe, microcirculatory flow by laser flow probe. MAP and CO constant	Diebel 1992 ²⁴
Renal	15 to 20 mmHg	Renal blood flow and glomerular filtration rate reduced to <25% of normal; oliguria	Dog study, expansion of blood volume could restore CO but not renal blood flow and glomerular filtration rate	Harman 1982 ⁹
	>30 mmHg	Anuria	Dog study, expansion of blood volume could restore CO but not renal blood flow and glomerular filtration rate	Harman 1982 ⁹
Cardiovascular	10 to 15 mmHg	Transient increase in venous return and CO	Dog study	Richardson 1976 ⁴³
	>20 mmHg	Significantly reduced venous return and CO; significantly increased systemic vascular resistance	Pig study, cardiopulmonary function measured, with raised intra-abdominal pressure, then after IV fluids to restore cardiac output	Ridings 1995 ⁴⁴
	>30 mmHg	Reduced ventricular compliance, increased systemic vascular resistance, reduced contractility and CO	Dog study	Kashtan 1981 ⁴⁷
	Monitoring	Increased pleural pressure, spurious elevation of central venous & pulmonary occlusion pressures	Pig study, cardiopulmonary function measured, with raised intra-abdominal pressure, then after IV fluids to restore cardiac output	Ridings 1995 ⁴⁴
Pulmonary	>15 to 20 mmHg	Increased peak and alveolar pressures and reduction in all lung volumes	Dog study	Richardson 1976 ⁴³
	>10 mmHg	Peak inspiratory pressure and pleural pressure rise	Pig study, cardiopulmonary function measured, with raised intra-abdominal pressure, then after IV fluids to restore cardiac output	Ridings 1995 ⁴⁴
	>15 mmHg	Significant hypoxia cf baseline (280 to 160 mmHg), peak inspiratory pressure increase from 9 to 32 mmHg	Pig study, cardiopulmonary function measured, with raised intra-abdominal pressure, then after IV fluids to restore cardiac output	Ridings 1995 ⁴⁴
	>25 mmHg	Significant hypercapnia cf baseline (40 to 70 mmHg)	Pig study, cardiopulmonary function measured, with raised intra-abdominal pressure, then after IV fluids to restore cardiac output	Ridings 1995 ⁴⁴
CNS	>25 mmHg	Significant elevation of ICP and decrease in CPP	Pig study. Effects independent of changes in pulmonary and cardiovascular function	Bloomfield 1997 ⁵²

IAP: intra-abdominal pressure; MAP: mean arterial pressure; CNS: central nervous system; ICP: intra-cranial pressure; CPP: cerebral perfusion pressure

could then theoretically be determined by subtracting inferior vena cava pressure, just proximal to the diaphragm, from mean arterial pressure (MAP), i.e. $APP = MAP - IVCP$. Inferior vena cava pressure is generally equal to right atrial pressure and therefore, $APP = MAP - \text{right atrial pressure}$. If $IAP > 20$ to 25 mmHg, inferior vena cava pressure is equal to IAP, and therefore, $APP = MAP - IAP$. Even within the portal circulation venous pressures should not exceed IAP. Bearing in mind the possible adverse consequences of increasing MAP in patients with severe ACS and a collapsed inferior vena cava, it is interesting to speculate whether abdominal perfusion pressure could be increased to improve abdominal and retroperitoneal organ perfusion in patients with IAH.

Renal Effects

The mechanism by which renal function is compromised is multi-factorial. As IAP increases, renal blood flow and glomerular filtration rate decrease (Table II). In animals, improving cardiac output and blood pressure does not fully correct renal dysfunction while stenting of ureters has no beneficial effect.⁹ Other possible mechanisms include direct parenchymal compression, caval or renal vein obstruction, reduction in aortic or regional arterial supply and neurohumoral dysfunction. Renal vascular resistance has been shown to increase with rising IAH and is thought to be the result of renal vein or parenchymal arteriolar compression.⁹ Activation of the renin-angiotensin-aldosterone system has also been demonstrated in this setting.³⁶⁻³⁸ More recent animal studies suggest that it is renal vein compression, rather than parenchymal

compression that is responsible for the decreased glomerular filtration.³⁷ Whatever the cause, raised IAP has been shown to be associated with post-operative renal impairment in humans³⁹ (Table III). Decompression in critically ill patients may reverse renal impairment if achieved early (within 10 to 24 h),^{16,40} but reversal by decompression is not a universal finding, and the renal effects of raised IAP may have more permanent consequences.^{11,41}

Cardiovascular Effects

In animals, the effects of IAH on the cardiovascular system are important and occur at relatively low IAP. Cardiac output decreases, systemic vascular resistance increases and mean arterial pressure decreases. Increased intrathoracic pressure, 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.^{33,34,42} Despite a small, transient increase in venous return and cardiac output as intra-abdominal pressure begins to rise,⁴³ elevated intra-abdominal pressure is associated with a decrease in vascular and cardiac volumes in all animal studies.^{42,44,45} This is because transmural pressures decrease and venous return from the abdomen and lower limbs is decreased.⁴⁶ Mechanically this results in decreased preload and cardiac output.^{44,47,48} Compression of the aorta and major arterial conduits in the thorax and abdomen will increase systemic vascular resistance and afterload. The increased systemic vascular resistance may maintain or even increase blood

TABLE III: PHYSIOLOGICAL EFFECTS OF RAISED INTRA-ABDOMINAL PRESSURE REPORTED IN CRITICALLY ILL PATIENTS

System	Level of IAP	Physiological effect	Notes	Source
Splanchnic	>25 cmH ₂ O	Gastric intramucosal acidosis without classic signs of abdominal compartment syndrome	pHi improves on decompression	Ivatury 1998 ²⁸
Renal	>18 mmHg	Independent cause of post-operative renal impairment	All patients admitted to ICU after abdominal surgery	Sugrue 1999 ³⁹
	>25 mmHg	Urine flow <0.5 mL/kg/hr in 65% patients	Acutely injured patients	Meldrum 1997 ⁷
	>35 mmHg	Urine flow <0.5 mL/kg/hr in 100% patients	Acutely injured patients	Meldrum 1997 ⁷
Cardiovascular	>15 mmHg	Increased central venous and pulmonary occlusion pressures	Right ventricular end diastolic index better indicator of preload	Malbrain 1999 ⁵⁰
	>15 mmHg	SVR >1000 and DO ₂ I <600 in 20% of patients	Acutely injured patients.	Meldrum 1997 ⁷
	>25 mmHg	SVR >1000 and DO ₂ I <600 in 65% of patients	Acutely injured patients	Meldrum 1997 ⁷
	>35 mmHg	SVR >1000 and DO ₂ I <600 in 100% of patients	Acutely injured patients	Meldrum 1997 ⁷
Pulmonary	>15 mmHg	Fall in oxygenation and static compliance	Abstract	Malbrain 1999 ⁵⁰
	>15 mmHg	Peak airway pressure >45 in 40% of patients	Acutely injured patients	Meldrum 1997 ⁷
	>25 mmHg	Peak airway pressure >45 in 78% of patients	Acutely injured patients	Meldrum 1997 ⁷
	>35 mmHg	Peak airway pressure >45 in 100% of patients	Acutely injured patients	Meldrum 1997 ⁷
CNS	Clinical abdominal compartment syndrome	Rising intra-cranial pressure, CO and cerebral perfusion pressure reduced	Case report, combined abdominal and head injury, improved with decompression	Bloomfield 1995 ⁵⁶

pressure at lower levels of IAH, but at the cost of a decreasing cardiac output. As IAP increases, the effect on decreasing cardiac output predominates and the blood pressure cannot be maintained. The important effects of IAH on central venous pressure and pulmonary occlusion pressures make our traditional “volumetric filling pressures” even more unreliable indices of preload. In critically ill patients, right ventricular end diastolic volume index (RVEDVI) measured by volumetric thermodilution, intrathoracic blood volume index measured by dye indicator techniques, and echocardiographic assessment are better preload markers, particularly when IAP is increased.^{27,48-50} They also correlate better with cardiac output. The importance of maintaining cardiac output cannot be stressed enough as the presence of hypovolaemia, haemorrhage, and PEEP exacerbates the deleterious effects of IAH in animals.^{26,51,52} Decompression results in an improvement in most, but not all, cardiovascular parameters measured at the time of decompression in critically ill patients^{7,16,20,27,53} (Table IV).

Respiratory Effects

The effects on the respiratory system are direct and primarily mechanical in nature. Intra-abdominal pressure is transmitted to the diaphragm. Diaphragmatic elevation and changes in diaphragmatic shape result.⁴⁴ Decreased static and dynamic pulmonary compliance cause ventilatory difficulty in humans.^{11,50} Mechanical ventilation results in the production of high airway pressures or low tidal volumes.⁵ Decreases in functional residual capacity, residual volume and total lung capacity, and the consequence of pressure atelectasis lead to ventilation perfusion abnormalities. These effects ultimately result in hypoventilation and hypoxia and the magnitude of the effect is proportional to the rise in IAH^{13,54} (Table III). As these changes are largely mechanical in aetiology, they are reversed by decompression in critically ill patients^{13,44,54} (Table IV). It has been suggested that pulmonary vascular resistance may be increased by a direct pressure effect on the vessels, alveolar hypoxia and possibly the release of vasoactive substances.¹⁴

Neurological Effects

In animal models, the effects of IAH on intracranial pressure appear to be largely the consequence of mechanical changes. For reasons discussed earlier, IAH causes increase in vascular pressures in both the abdominal and thoracic compartments. In the abdomen, the high passively transmitted venous pressures inhibit venous drainage of the central nervous system via the lumbar plexus. In the chest, the raised intrathoracic pressure, transmitted to the internal jugular veins, causes a functional obstruction to cerebral venous outflow, which results in the observed

increase in intracranial pressure. Cerebral perfusion pressure changes depend on the intracranial pressure as well as the response of arterial pressure to IAH, which is variable. In an animal model, sternotomy resulted in a return to baseline of both intrathoracic and intracranial pressures, confirming the largely mechanical nature of the changes.⁵² While these changes may not be life threatening to humans with normal baseline intra-cranial pressure, such increases may turn out to be significant in critically ill patients with decreased intra-cranial compliance. Decompression is necessary to reverse the changes^{55,56} (Table IV).

Clinical Implications in the Intensive Care Unit

Intra-abdominal hypertension is common in high-risk patient groups. It should not be forgotten as a significant complication of massive fluid resuscitation, even in non-surgical patients. The incidence in high-risk critically ill patients justifies the routine use of the bedside intra-vesical method of measurement in this group. Intra-abdominal hypertension and pHi are strongly associated with, and seem to predict, the same adverse outcomes - shock, renal failure, sepsis, need for re-laparotomy and death.^{28,57} Animal studies suggest that decreased gut perfusion places the patient with even moderate IAH at risk for bacterial invasion and delayed multiple organ dysfunction and decreased gut perfusion persists even in the presence of normal cardiovascular indices. 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 decreased by IAH.

Hypovolaemia, hypotension and a decreased cardiac output worsen end organ perfusion, particularly that of the gut and kidneys. Patients with IAH must therefore be aggressively resuscitated to increase cardiac output and end organ perfusion, rather than to achieve arbitrary central venous pressures. Volumetric indices of preload such as RVEDVI may be a useful guide to preload estimation. 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 intra-cranial 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.

TABLE IV: EFFECTS OF DECOMPRESSION IN PATIENTS WITH INTRA-ABDOMINAL HYPERTENSION (IAP) OR ABDOMINAL COMPARTMENT SYNDROME (ACS)

Source	Study population	Study type	Patients	ACS definition	Patients with ACS (%)	Time (hrs) to decompression	CVS	RS	Spl	Renal	CNS	Decompressed	Mortality	Not decompressed
Ertel ¹⁶	Damage control laparotomy for abdominal or pelvic trauma	Retrospective & prospective	311	Clinical and IAP >25 mmHg	5.50	12.9	Y	Y	Y	Y	Y	6/17 (35%)		0
Maxwell ²⁰	Fluid resuscitation No abdominal injury	Retrospective	46	Clinical	13	18	Y	Y	Y	Y		4/6 (67%)		0
Cheatham ⁴⁹	Trauma, vascular and major surgery	Prospective	20	Average preoperative intra-abdominal pressure 38 mmHg	100							15/20 (75%)		9/9 (100%)*
Chang ²⁷	Severe abdominal trauma	Prospective	11	Clinical and IAP >25 mmHg	100	38	Y	Y	Y	Y	Y	7/11 (63%)		0
Meldrum ⁷	Severe trauma, post-laparotomy	Prospective	145	Clinical and IAP >20 mmHg	14	27	Y	Y	Y	Y	Y	6/21 (29%)		0
Meldrum ⁵³	Hepatic injury, with peri-hepatic packing	Retrospective	11	N/A	100	<24	Y	Y	Y	Y		5/11 (45%)		0
Morris ¹⁵	Staged celiotomy after major abdominal surgery	Retrospective	107	Clinical, with improvement after decompression	15			Y				9/14 (64%)		1/1 (100%)
Platell ⁴⁰	Abdominal aortic surgery	Prospective	22	Clinical and IAP >18 mmHg	45	<24				Y		6/10 (60%)		0
Saggi ¹⁴	Combined reports of ACS before 1990	Retrospective	27	N/A	100		Y	Y	Y	Y	Y	11/21 (52%)		6/6 (100%)

* Nine patients developed recurrent compartment syndrome after decompression—all died. These patients are part of the 15 deaths in the previous column. CVS: cardiovascular system; RS: respiratory system; Spl: splanchnic; CNS: central nervous system

TABLE V: THERAPEUTIC RECOMMENDATIONS BASED ON THE SEVERITY OF INTRA-ABDOMINAL HYPERTENSION⁷

Bladder pressure (mmHg)	Recommendation
10-15	Maintain normovolaemia
16-25	Hypervolaemic/Hypertensive resuscitation
26-35	Decompression
>35	Decompression and re-exploration

Definitive Management

The only way to definitively eliminate IAH is by abdominal decompression. No randomised, controlled assessment of decompression of IAH has been reported and management recommendations are therefore based on retrospective observations, uncontrolled prospective studies and expert opinion. The issues of interest to the intensive care clinician are the following. Does decompression reverse the effects of IAH and if so to what degree? What level of IAH requires decompression and how long can IAH be tolerated before irreversible changes occur? Table IV shows the outcome of patients who are decompressed and those not decompressed, or in whom decompression was unsuccessful.^{6,16,27} It is apparent that left untreated, IAH is likely to increase mortality, and certainly untreated ACS is usually fatal. The exact level of IAH that defines "critical IAP" remains subject to debate, but there is expert consensus that decompression should be performed at levels of IAP above 20 to 25 mmHg. 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 mmHg and the presence of gastric intramucosal acidosis in humans, this number may have to be revised to a lower figure. At way present, the grading and general therapeutic system proposed by Meldrum et al⁷ would appear reasonable, and at intra-abdominal pressures of less than 25 mmHg, attempts to increase the abdominal perfusion pressure could be

additionally considered (Table V). The optimal timing of decompression is difficult to determine. Abdominal compartment syndrome has become a well-recognised clinical entity and should be considered an emergency, requiring immediate decompression.^{6-8,14} Close monitoring of IAP is important to provide early warning of progressive IAH and facilitate decompression before classical ACS develops.

In high-risk surgical patients, prevention of IAH 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 far from providing a definitive answer⁵⁸⁻⁶³ (Table VI).

Conclusion

Intra-abdominal hypertension is a real entity that occurs in critically ill medical and surgical patients. The profound effect of high intra-abdominal pressure on multiple organ systems mandates that it should be recognised and routinely monitored in patients at high risk. Understanding the pathophysiology of IAH 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 fully understand the clinical implications and confirm the appropriate management of this condition.

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TABLE VI: MORTALITY OF PATIENTS AT HIGH RISK FOR ABDOMINAL COMPARTMENT SYNDROME IN WHOM PREVENTIVE STRATEGIES HAVE BEEN UTILISED

Source	Study type	Study population	Prevention group	Comparison group	Prevention mortality	Comparison mortality
Ghimenton 2000 ⁶²	Prospective	100 trauma, 57 non-trauma	157	x	69 (44%)	x
Barker 2000 ⁶³	Retrospective	Celiotomy for trauma	112	x	29 (25.9%)	x
Ciresi 1999 ⁶¹	Retrospective	Ruptured abdominal aortic aneurysm	18	x	4 (22%)	x
Ivatury 1998 ²⁸	Prospective	Celiotomy for trauma	45	25	5 (11%)	9 (36%)*
Sugrue 1998 ⁴¹	Prospective	Miscellaneous	49	x	21 (43%)	x
Oelschlager 1997 ⁵⁹	Retrospective	Ruptured abdominal aortic aneurysm	8	15	4 (50%)	11 (73%)
Mayberry 1997 ¹⁹	Retrospective	Celiotomy for trauma	73	73	14 (19%)	14 (19%)

* More multiple organ dysfunction in the comparison group ($P = 0.023$).

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