

ABDOMINAL COMPARTMENT SYNDROME IN THE CRITICAL ILL PATIENT: A  
COMPREHENSIVE REVIEW AND IMPLICATIONS FOR THE ACUTE CARE  
NURSE PRACTITIONER

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## ABSTRACT

Compartment syndrome, defined by increases in pressure within a closed anatomical space, can be a life-threatening condition observed when perfusion pressures drop significantly below tissue pressures. Upon leaving such pressures untreated, tissue necrosis and permanent functional impairment result.

Abdominal compartment syndrome (ACS), occurring due to sustained increases in abdominal pressures from a variety of insults, ultimately causes impaired tissue perfusion to the abdominal organs leading to organ ischemia, multiple organ failure syndrome, and death. It is an occult process that cannot be detected without an established method to measure and monitor abdominal pressures. If ACS is left untreated, it is fatal.

The ACNP needs to recognize ACS and monitor all high-risk patients with intra-abdominal pressure (IAP) measurements to allow early intervention. The goal should be to prevent ACS by early monitoring and non-surgical interventions followed by early surgical intervention if preventative measures fail.

## CHAPTER 1

### Introduction

Compartment syndrome occurs when the pressure within a confined space increases to a point where the vascular inflow is compromised, and the function and viability of the tissues within the compartment are threatened (Edwards, 2004).

Abdominal compartment syndrome (ACS) is defined as the adverse physiologic consequence occurring as a result of an acute increase in intra-abdominal pressure (IAP) due to a variety of insults to the body that cause tissue edema or a collection of free fluid in the abdominal cavity (Fernandez, 2006).

Various terms have been used throughout literature to describe the different pressures within the abdomen. The terms intra-abdominal hypertension (IAH) and ACS have been used interchangeably throughout literature; however, it is important to recognize the distinction between the two terms. IAH is defined as an elevated pressure within the abdomen when the IAP exceeds 12-15 mmHg with the presence of an organ ischemia (Bailey & Shapiro, 2000; Pleva & Mayzlik, 2004; WSACS, 2007). ACS is defined as an IAH  $\geq$ 20 mmHg with at least one organ in failure (Malbrain, Deeren, & DePotter (2005), WSACS, 2007; Meyer, 2006).

Among abdominal and pelvic injuries leading to ACS, blunt abdominal trauma, substantial intra-abdominal and retroperitoneal bleeding, and massive fluid resuscitation are among the top sources in causing sustained increased in abdominal pressures (Gallagher, 2004; Malbrain, Deeren, & DePotter, 2005). Other known cases are seen with extensive edema related to bowel disease states such as ascites, acute hemorrhagic

pancreatitis, and septic peritonitis (Malbrain, Deeren, & DePotter, 2005).

The best way to treat ACS is through prevention. The key to successful ACS prevention is to be able to recognize patients at risk, and to monitor them carefully in order to provide early interventions. Prevention can be accomplished through careful fluid management, knowing when the patient is either under-resuscitated or over-resuscitated state, monitoring abdominal pressures, and knowing when the time is right to provide early medical and surgical interventions (WSACS, 2007; Walker & Criddle, 2003).

The acute care nurse practitioner (ACNP) provides advanced nursing care across the continuum of health care services to meet the needs of patients with complex acute, critical, and chronic health conditions. The care provided is continuous and comprehensive. The ACNP is able to practice in a setting where patients experience episodic illness, exacerbation of chronic illness, or terminal illness. It is the responsibility of the ACNP to be able to practice in a setting requiring complex monitoring and therapies, high-intensity nursing intervention, or continuous nursing vigilance within the range of high-acuity care (Lozen, 1999). Additionally, the ACNP is able to manage patients utilizing invasive interventions and procedures to promote physiologic stability. The ACNP therefore acquires the skills and knowledge needed to care for patients with high acuity such as in patients with ACS.

The ACNP is in a dedicated role that has broadened expertise in providing care to patients in the emergency department (ED), and intensive care units (ICU) as well as under hospitalist services. This specialized role carries with it unique opportunities to

impact the care provided to various patients throughout the hospital (Lozen, 1999).

The clinical importance of understanding ACS is for the ACNP to recognize the patient at risk and knowing when to respond promptly with early interventional strategies that will prevent ACS from developing, leading to a much better prognosis for the patient. The ACNP should be able to distinguish signs and symptoms of ACS in order to promptly recognize patients at risk. In patients at high risk for AC, it is important that the ACNP understands abdominal perfusion pressure measurements and monitoring as well as current medical and surgical management strategies. Intra-abdominal monitoring should be strongly considered in all high-risk patients (Ivatury, Cheatham, & Mabrain, 2006).

ACS ultimately leads to sustained decreases in blood flow to the abdomen wall and organs therefore resulting in overall ischemia and necrosis to the surrounding tissues and vascular system. Ischemia causes the initiation of the acute inflammatory response including multiple cytokines release, free radical formation, and decreased production of cellular adenosine triphosphate. This response results in bacterial translocation from the gut predisposing patients to sepsis and multi-organ dysfunction syndrome (Malbrain, Deeren, & DePotter, 2005). ACS can be described as a constellation of sequelae beginning with regional blood flow disturbances ultimately leading to sustained intra-abdominal hypertension giving rise to a multitude of life-threatening conditions if left untreated (Demetriades, 2000; Ruffolo, 2002; McNelis, Marini, & Simms, 2003).

The increasing pressure in the abdomen leads to dramatic pathophysiological effects on the pulmonary, cardiovascular, renal, splanchnic, musculoskeletal,

integumentary, and central nervous systems (Ivatury, Cheatham, & Mabrain, 2006; Malbrain, Deeren, & DePotter, 2005; WSACS, 2007). Furthermore, such pathophysiological changes can mimic or be confused with other conditions such as the acute respiratory distress syndrome (ARDS) or sepsis (Schulman, 2005). This is an important concept as a practitioner may misdiagnose or miss the diagnosis of ACS.

The onset of the ACS requires prompt recognition and appropriately timely interventions in order to optimize outcome and minimize sustained ischemic effects to the abdominal organs. Although there is no established level at which IAH requires surgical intervention, abdominal decompression is usually performed in a patient who has an IAP  $\geq 20$  mmHg (Gallagher, 2004). The decision to decompress the abdomen also resides with the patient's age, underlying co-morbidities, and functionality of the organs (Schulman, 2005; Malbrain, Deeren, & DePotter, 2005).

The clinical picture of ACS is characterized by hypoxia refractory to increased oxygen, difficulty in ventilation, oliguria or anuria, a distended abdomen, and peak inspiratory pressures (PIP) mounting up to 85 cm H<sub>2</sub>O (Normal PIP: 45-55 cm H<sub>2</sub>O). To note, patients who have IAH but do not have the clinical ACS symptoms described above, may still have adverse physiologic effects from increased abdominal pressures that are not objectively measured by the practitioner (Ivatury, Cheatham, & Mabrain, 2006). As previously stated, recognizing a patient at risk is a crucial step to provide immediate medical or surgical interventions.

## History

Since the 19<sup>th</sup> century, understanding IAP and its effects on respiration and abdominal organs has been an interest of many researchers. In 1890, Emerson confirmed that an IAP of at least 27 mmHg or greater was a significant cause of death in feline and porcine models. However it was not until 1911 when Emerson, following a series of elaborate experiments, concluded that excessive IAPs diminished venous return to the heart, resulting in cardiovascular failure further deteriorating homeostasis in various animals leading to their death (Emerson, 1911; Bailey & Shapiro, 2000). Coombs (1920) further demonstrated the additive effects of hemorrhage and diminished circulating blood volume on cardiovascular compromise from elevated IAP (Bailey & Shapiro, 2000). Bradley and Bradley (1947) further demonstrated that increased pressures in the inferior vena cava and renal veins decreased renal plasma flow and glomerular filtration rate.

Through basic research and clinical observation in the past century, researchers and scientists are able to uncover and confirm the devastating effects of IAH on multiple organ systems. In the early 1980's, Kron, Hartmen, and Nolan (1984) were the first ones to use the term ACS to describe pathophysiological effects of IAH secondary to aortic aneurysm surgery. Presently ACS refers to cardiovascular, pulmonary, renal, splanchnic, abdominal wall/wound, and intracranial disturbances resulting from an increased IAP regardless of the cause (McNelis, Marini, & Simms, 2003).

Initially recognized almost 150 years ago, the pathophysiologic implications of IAH have only recently been re-discovered. The concept behind ACS parallels the

development of compartment syndrome where the abdominal cavity is considered a closed anatomical space with a finite amount of volume. Although descriptions of ACS dated back to the 19<sup>th</sup> century, it has only been in the last 20 years that ACS has been described in the trauma, medical, and surgical setting (Ivatury, Cheatham, Mabrain, & Sugrue, 2006).

Despite various early contributions in defining ACS, the clinical and pathophysiologic significance of elevated abdominal pressure went largely unrecognized; hence, the mortality rate for ACS has far exceeded 50% within the years following an accurate diagnosis of the syndrome (Gallagher, 2004; Malbrain, Deeren, & DePotter, 2005). In the 1980s, there was a renewed interest in the pathophysiologic effects of elevated abdominal pressures. The prevalence of sustained IAP in patients developing organ failure suggests that increases in these pressures play a major role in the development of multiple system organ failure (MSOF) - a known major cause of ICU mortality (Walker, 2003). Several authors published reports of impaired organ function associated with elevated abdominal pressures and showed significant clinical improvement after surgical decompression of the abdomen (Saggi, Sugerman, Ivatury, & Bloomfield, 1999; Biswajit, 2004).

#### Significance of Abdominal Compartment Syndrome

An acute or sustained increase in IAP eventually results in IAH and/or ACS that compromises vascular inflow and threatens the viability of the abdominal tissues and organs (Edwards, 2004). This syndrome originates from various causes associated with intra-abdominal surgery, infections, massive volume resuscitation, blunt abdominal

trauma, or other associated conditions giving rise to increased pressures within the abdomen (Gallagher, 2004; Malbrain, Deeren, & DePotter, 2005).

Critically ill patients often have a multitude of physiologic derangements that stem from various sources that occur simultaneously. Due to the devastating effects and high mortality rate of ACS if not properly recognized and treated, familiarity with its presentation and diagnosis is mandatory (Schulman, 2000).

#### Problem Statement

Although ACS is widely noted in the medical literature, IAH and ACS have received little attention in the nursing literature. It is crucial for ACNPs to be able to recognize the patient at risk for ACS in the critically ill despite the cause. Accurate and prompt nursing assessment skills are essential in order to identify the patient at high risk of ACS.

Furthermore, evidence based interventions are lacking. For example, there is no set abdominal pressure at which to perform surgical decompression of the abdomen in a patient with ACS. Evidence on appropriate wound care management in a patient with post-operative decompressive laparotomy is lacking. There is no protocol available for continuous abdominal pressure monitoring in a patient with an open abdomen. Without appropriate set guidelines, it poses a challenge to the ACNP to use appropriate evidence based practice when caring for a patient who is at risk for ACS.

#### Project Purpose

The purpose of this project is to evaluate peer-reviewed articles and original research on the pathophysiology of ACS, and to provide guidance for assessment and

management of ACS. Increased knowledge about this syndrome can enable the practitioner to appropriately identify patients at risk for ACS, allowing the ACNP to provide early interventions that may increase survival rates. This review will present information on pathophysiology, etiology, prevention strategies, diagnostic criteria, and treatment outcomes related to ACS.

## CHAPTER 2

### Overview of the Pathophysiology of ACS

ACS, otherwise known as IAH due to elevated pressures and associated clinical changes in intra-abdominal and extra-abdominal organs, is a potentially lethal condition that develops from progressive, acute increases in the IAP to 20 mmHg or greater within a confined space (Bailey & Shapiro, 2000; Malbrain, Deeren, & DePotter, 2005; WSACS, 2007).

Normal IAP is between 0-5 mmHg to 6.5 mmHg in a supine position. A variety of insults can raise the IAP significantly to produce deleterious effects to the surrounding tissues and organ systems. Physiologic compromise in the form of ischemia occurs when the IAP rises to 10 mmHg or more. However when pressures reach  $\geq 20$  mmHg, irreversible organ damage occurs producing necrosis and furthermore compounding to ACS (Malbrain, Deeren, & De Potter, 2005).

Abdominal perfusion pressure (APP) is determined by mean arterial pressures (MAP) and IAPs that resists blood delivery to the abdominal organs. The APP is represented by the formula:  $APP = MAP - IAP$ . An APP sustained at measurements greater than 50 mmHg decreases the threat of morbidity and mortality from increases in IAPs (Bailey & Shapiro, 2000). The abdominal cavity is a single compartment and any change in volume within this cavity can elevate IAP further leading to IAH (Harrhill, 1998). ACS is likely to develop after any event affecting the abdomen (Walker, 2003); hence, ACS is considered a syndrome and not a disease in which there can be many different causes (Malbrain, Deeren, & De Potter, 2005).

Although not fully compliant, the abdominal cavity is more amenable than most confined cavities but can become increasingly rigid as it distends. As the abdominal compliance threshold is reached, the IAP rises and the APP decreases. If untreated, this leads to organ ischemia and ultimately, ACS (Saggi, Sugerman, Ivatury, & Bloomfield, 1999). The devastating consequential results of an increased IAP are failure of multiple organ systems (Myers, 1999).

Cellular hypoxia resulting from vasoconstriction and blood shunted away from the skin, muscles, kidney, and the gastrointestinal tract is the primary pathological event leading to IAH and ACS. A sequence of events promotes the development of IAH and its progression to ACS: the release of inflammatory cytokines, the formation of oxygen free radicals, and a decrease in cellular production of adenosine triphosphate (ATP) (Walker, 2003). These events described above often lead to bacterial translocation from the gut and intestinal edema. Bacterial translocation further increases inflammation and swelling in the abdomen predisposing the patients to multi-organ dysfunction syndrome (Walker & Criddle, 2003). These events will be described in detail in the following paragraphs.

The primary pathophysiologic event leading to IAH and ACS is interstitial edema in the bowel and mesentery due to capillary endothelial damage (Walker, 2003; & Fox, Miller, & Nix, 1999). The damage occurs due to ischemia from the original physiological insult: sepsis, hemorrhage, massive fluid resuscitation, etc. Pro-inflammatory cytokines that originate from the initial injury stimulate the acute inflammatory response by attracting nutrients, fluids, clotting factors, and neutrophils/macrophages to the damaged site (Miller, Wayne, Johnson, & Chang, 2004).

As fluid accumulates in response to the acute inflammatory response cycle, the abdominal wall and fascia are slowly stretched until they become less compliant causing the pressure within the abdominal cavity to rise (Walker, 2003). As the pressures mount, intestinal perfusion is impaired and the cycle of cellular hypoxia, cell death, inflammation, and edema continues unabated (Walker, 2003).

Furthermore, the original damaged site initially releases specific mediators such as histamine, prostaglandins, and cytokines that produce a chemotaxic signaling system causing increased capillary permeability leading to edema and vasodilation. The increase in capillary permeability leads to edema that results in increased IAPs diminishing blood flow to the surrounding tissues and organs. Local tissue ischemia results from the increased interstitial pressure overcoming the intravascular pressure causing the vessel walls to collapse. When the bowel expands due to an increasing IAP, the abdominal wall compliance is hindered causing compromised arterial and venous blood flow throughout the abdomen and its housed organs. Anaerobic metabolism results from decreased blood flow and increased ischemic events (Miller, Wayne, Johnson, & Chang, 2004).

ATP is an energy source required for the sodium-potassium pump to actively transport electrolytes in and out of the cell. Normally high intracellular potassium and low intracellular sodium and calcium concentrations are usually maintained. However, with the decline in ATP production, the cellular plasma membrane can no longer maintain active electrolyte gradients. Potassium slowly leaks into the extracellular space while sodium and calcium enter into the cells with water following thus creating a hypotonic cellular state. As the cells swell, the membranes lose their integrity, spilling its

intracellular contents into the extracellular space causing more inflammation in the form of edema throughout the body (Miller, Wayne, Johnson, & Chang, 2004).

The acute inflammatory response triggered initially from a traumatic event or injury is often driven further with reperfusion efforts aimed at restoring large amounts of volume with intravenous fluids such as normal saline, lactated ringer's solution, or blood products (Levine & Braun, 2006). The aim is to restore homeostasis (Jordan, 2000); however, this action only promotes further tissue edema and unknowingly causing ACS (Gallagher, 2004). As the practitioner hastily tries to resuscitate the patient by administering fluids, reperfusion injury occurs causing the release of oxygen free radicals initiating more cytokine release and further initiating another cycle of the acute inflammatory response (Walker, 2003). Detailed information regarding the physiological injury process is illustrated in Figure 1.

Etiology: Incidence and Risk Factors

ACS can be segregated into three different types: 1). Primary, 2). Secondary, and 3). Recurrent. A thorough discussion will be presented on different types of ACS and the causes associated with each type.

Primary ACS occurs when the intra-abdominal pathology is directly and proximally responsible for the compartment syndrome. Various types of primary ACS originate from surgical, trauma, and/or visceral compression/reduction factors such as obesity, ascites, and cirrhosis (Malbrain, Deeren, & DePotter, 2005; Waele, Hoste, & Malbrain, 2006). This type of ACS has been identified as one of the major causes associated with morbidity and mortality in patients presenting with a traumatic injury

(WSACS, 2007). Examples are found in table 1.1.

Table 1.1: Primary Type: Causes of IAH/ACS

Surgical
Intra-operative fluid balance >6 L
Abdominal aortic aneurysm repair
Trauma
Shock requiring resuscitation (ischemia-reperfusion)
Damage Control Laparotomy
Multiple trauma with or without abdominal trauma
Major burns (>25% TBSA)
Visceral compression/Reduction
Large ascites, peritoneal dialysis
Retroperitoneal/abdominal wall bleeding
Large Abdominal Tumor
Laparotomy closed under tension
Gastroschisis/Omphalocele

Abdominal compartment syndrome. (Bailey & Shapiro, 2000)

Secondary ACS is a syndrome occurring in the absence of abdominal injury but cause fluid accumulation resulting in the signs and symptoms commonly associated with primary ACS. The development of secondary ACS has been viewed by many authors as unavoidable sequelae due to aggressive crystalloid resuscitation in an attempt to treat

severe shock. Types of secondary ACS resemble patients with sepsis, systemic inflammatory response (SIRS), ischemia, reperfusion injuries; and critical obstetrical conditions, ischemia, and capillary leakage (Malbrain, Deeren, & DePotter, 2005; Waele, Hoste, & Malbrain, 2006). Example of each type can be located in Table 1.2.

Table 1.2 Secondary Type: Causes of IAH/ACS

Sepsis, SIRS, Ischemia, Reperfusion
Sepsis and resuscitation with >5 L fluid in 24 hrs
Ongoing use of vasopressors to support and maintain hemodynamic stability
Pancreatitis
Peritonitis, colitis
Ileus, bowel obstruction
Mesenteric ischemia, necrosis
Critical Obstetrical conditions
Pregnancy related Disseminated Intravascular Coagulation
Ischemia
Systemic inflammatory response
Capillary leak
Fluid resuscitation
Intra-abdominal hypertension
Tissue Edema (including bowel wall and mesenteric edema)

Abdominal compartment syndrome. (Bailey & Shapiro, 2000)

Lastly, recurrent ACS is a relative new type caused by the persistence of ACS or the development of a new episode of ACS following a temporary abdominal wall closure (Waele, Hoste, & Malbrain, 2006). Recurrent ACS is a real problem as practitioners and hospital staff may not be aware that continuous monitoring and measurements of IAPs are needed despite appropriate medical or surgical interventions done at that time when elevations of IAPs exceeds 20mmHg indicating organ ischemia and/or failure. Some researchers (Gracias, et al. (2002); Waele, et al. (2006) have proposed that a patient with an open abdomen does not rule out the possibility of ACS. Furthermore, patients who experience recurrent ACS have a poorer prognosis. Therefore it is important that bladder pressure measurements should be continued even when managing a patient with an open abdomen post-decompressive laparotomy.

Many authors have published data on the commonality of ACS in their practice setting with a rate from a few percent to over 40 percent. However, as of 2004, only a few researchers have been able to systematically measure IAP in each patient in various ICU's to determine an accurate prevalence of IAH and ACS. Efstathiou et al. (2005) noted IAP levels greater than 12, 15, and 20 mmHg respectively in over 67%, 25%, and 4% of their septic population respectively. Hernandez (2005) found IAP greater than 20mmHg in 51% of their critically ill patients with septic shock.

The occurrence rate of ACS is different based on the population studied. In contrast, there are numerous studies describing the clinical effects of ACS. Malbrain, et al (2004) studied the prevalence of ACS in 97 patients at 13 different medical, surgical, and

trauma units over six different counties. The results showed that over half of the patients had an IAH of 12 mmHg or greater, one-third had severe IAH of 15mmHg or more and one organ in failure, and 1 in 10 patients had ACS with IAH over 20 mmHg and one organ in failure. The significance of this study not only shows the occurrence of IAH and ACS, but it also demonstrates that ACS is not solely a trauma issue, but a medical and surgical issue as well. Therefore, it is important to recognize patients at risk in both the trauma and medical units. The prevalence rate of ACS is summarized in Table 2.

Table 2 Prevalence Rate

Cut-off (mmHg)	Total Prevalence	MICU Prevalence	SICU Prevalence
IAP $\geq$ 12mmHg	58.8%	54.4%	65%
IAP $\geq$ 15mmHg	28.9%	29.8%	27.5%
IAP $\geq$ 20mmHg + organ failure	8.2%	10.5%	5%

Prevalence of intra-abdominal hypertension in critically ill patients: a multi center study. (Malbrain et al., 2004).

(\*Note: MICU=medical intensive care unit; SICU=surgical intensive care unit)

Although an exact incidence of ACS is yet to be determined, it is clearly more common in specific populations (Salomone, 2007). The patient population considered at highest risk of ACS is the patient who requires massive fluid resuscitation of  $\geq$ 5/L in less than 24 hours, and the patient presenting with blunt abdominal trauma. Other forms of trauma placing the patient at risk are caused by the specific examples classified under the primary and secondary types of ACS (Bailey & Shapiro, 2000; Malbrain et al., 2004).

In the trauma population, increased pressure is associated with bleeding that

moves into the intraperitoneal and retroperitoneal space, from massive volume resuscitation, and due to the different forms of shock. In addition, IAH and the ensuing ACS is associated with any process involving an accumulation of fluid and edema in the confined abdominal space resulting in tension on the heart, gut, kidneys, and lungs (Saggi, Sugerman, Ivatury, & Bloomfield, 1999).

Through research and various on-going studies, we now know that virtually every organ, except the adrenal glands, is equally affected by a dramatic rise in IAP (Peralta & Hojman, 2001). As stated previously, many conditions can place the patient at an increased risk for IAH and subsequent ACS (Ruffolo, 2002).

#### Pathophysiologic Effects of Abdominal Compartment Syndrome

According to Malbrain, Deeren, and De Potter (2005), the pathophysiologic effects of ACS affect several functions of the human body and are detrimental to all the organs in a graded fashion.

#### Neurological Dysfunction

IAH, which acts as an independent risk factor for secondary brain injury, impacts intracranial pressure. An increase in IAP pushes the diaphragm up and therefore reduces intra-thoracic volume resulting in an elevated intra-thoracic pressure. An elevated intra-thoracic pressure causes an elevated central venous pressure and subsequent elevated internal jugular venous pressure. The increasing pressures impair regional venous outflow causing intracranial congestion and decreased cerebral perfusion pressure leading to cerebral ischemia. This causes hypoxia and impaired neurological functions (Malbrain, Deeren, & De Potter, 2005).

Table 3 Clinical Effects of Increased Abdominal Pressure on the Neurological System

System	Clinical Effects
*Neurologic	-Increased intracranial pressure -Decreased cerebral perfusion pressure

### Cardiovascular Dysfunction

IAH with elevations in intra-thoracic pressure leads to substantial cardiovascular demise where IAH decreases cardiac output (CO) despite a normal ejection fraction (EF) and normovolemia. In addition, IAH elevates the central venous pressure and pulmonary capillary wedge pressure even in the face of hypovolemia (Figure 2). Furthermore, due to the changes in pressure developing in the abdominal and thoracic cavities, measurements of fluid resuscitation with central venous pressures and pulmonary capillary wedge pressures can be misleading. Failure to recognize these important characteristics of IAH on cardiac functioning can lead to under resuscitation, persistent global ischemia, and worsened patient outcomes. See figure 2 for graph interpretation. See Figure 2 for graph interpretation.

Cardiac functions are therefore arrested with the following dysfunctional residual effects: venous return and CO are decreased, systemic and pulmonary vascular resistance increase, heart rate remains stable or increased, mean arterial pressure is initially increased then slowly decreased, pulmonary arterial pressures is increased and lastly, left ventricular compliance as well as regional wall movements are decreased.

Table 4 Clinical Effects of Increased Pressure on the Cardiovascular System

System	Clinical Effects
*Cardiovascular	<ul style="list-style-type: none"> <li>-Decreased cardiac output</li> <li>-Decreased preload</li> <li>-Increased afterload</li> <li>-Increased CVP and PCWP</li> </ul>

\*Note: CVP: central venous pressure; PCWP: pulmonary capillary wedge pressure

### Pulmonary Dysfunction

An increase in IAP increases the intra-thoracic and pleural pressures often leading to edema and atelectasis causing a decrease in functional residual capacity, total lung compliance, and residual volume thus mimicking restrictive lung disease. Inasmuch, the effects on the respiratory system are largely mechanical. Alveolar collapse due to the smaller intra-thoracic space and the higher intra-thoracic pressure ultimately leads to ventilation-perfusion mismatching, hypoxia, hypercarbia, and respiratory acidosis. The clinical practitioner must monitor for pulmonary hypertension due to sustained elevations in intra-thoracic pressure and hypoxic vasoconstriction.

In patients who are mechanically ventilated, the auto positive-end expiratory pressure (PEEP), peak, plateau, and mean airway pressures increase that often results in alveolar barotrauma. In addition, the dynamic and static total respiratory system compliance drops significantly due to decreased chest wall compliance.

Eventually, the ischemia that develops from hypoxia and reduced cardiac output soon cause inflammatory mediator release that result in ARDS, a condition far too common in the ICU (Malbrain, Deeren, & De Potter, 2005). ARDS that develops is the result of a combination of alveolar collapse, high intra-thoracic pressure, and interstitial

edema.

Table 5 Clinical Effects of Increased Abdominal Pressure on the Pulmonary System

System	Clinical Effects
*Pulmonary	<ul style="list-style-type: none"> <li>-Increased peak inspiratory pressures</li> <li>-Increased airway pressures</li> <li>-Decreased PaO<sub>2</sub></li> <li>-Increased PaCO<sub>2</sub></li> <li>-Decreased dynamic compliance</li> </ul>

\*Note: PaO<sub>2</sub>: partial pressure of oxygen in arterial blood; PaCO<sub>2</sub>: partial pressure of carbon dioxide in arterial blood

### Renal Dysfunction

IAP decreases renal blood flow, renal perfusion pressure, and the filtration gradient. An IAP at or above 15 mmHg produces the clinical sign of oliguria, and an IAP greater than 30 mmHg produces anuria. The cause of renal dysfunction is most likely due to multi-factorial events such as reduced cardiac output, increased renal vascular resistance, and a decreased glomerular filtration gradient.

Reduced cardiac output due to increased IAH on the cardiovascular system leads to an increase in systemic vascular resistance and vasoconstriction of the arterial tree to include the renal artery system. Further exacerbated by humeral factors, arterial blood flow is reduced by the antidiuretic hormone and increased plasma renin-aldosterone activity. Furthermore, IAH compresses the renal veins therefore reducing venous drainage causing venous congestion.

Renal function is dependent upon the filtration gradient which is equal to glomerular filtration pressure minus proximal tubular pressure. Upon an elevated IAP,

the proximal tubular pressure acts in the same way as an elevated intra-abdominal pressure exacerbating an already hindered renal function. Reduction in renal filtration gradient, which is due to a combination of renal congestion, direct renal compression, and reduced arterial blood flow to the kidneys has the most profound effect on urine formation leading to oliguria and anuria (Saggi et al, 1999).

Table 6 Clinical Effects of Increased Abdominal Pressure on the Renal System

System	Clinical Effects
*Renal	<ul style="list-style-type: none"> <li>-Decreased renal plasma flow</li> <li>-Decreased glomerular filtration rate</li> <li>-Decreased glucose reabsorption</li> <li>-Oliguria or anuria</li> </ul>

#### Splanchnic Dysfunction

Decrease in splanchnic blood flow from an increased IAP occurs from mechanical compression of the splanchnic bed and mesenteric vasoconstriction produced by vasopressin (Malbrain, Deeren, & De Potter, 2005). Despite limited research on this area, researchers suggest that decreased blood flow to the splanchnic system results in mucosal acidosis and edema which leads to immediate inflammatory reactions allowing the release of oxygen free radicals resulting in overall organ damage (Malbrain et al., 2005; Lozen, 1999).

#### Hepatic Dysfunction

Portal venous blood flow decreases due to an increase in IAP, at or above 10 mmHg, to which ultimately decreases the total hepatic blood flow to the liver. The effect of an increased IAP on the liver lies within the hepatic artery buffer system that initially

tries to protect the liver against hypoperfusion but soon gets exhausted leading to inadequate blood flow through the portal vein (Malbrain, Deeren, & De Potter, 2005). The consequence of inadequate blood flow to the hepatic system results in ischemia and coagulopathy producing hypothermia, acidosis, and intra-abdominal bleeding. These events then further increase IAH resulting in ACS (Malbrain et al., 2005; Lozen, 1999).

#### Intestinal/Mucosal Dysfunction

An elevated IAP produces tissue ischemia throughout the entire abdominal cavity affecting all organs, excluding the adrenal gland. The vicious cycle is countless as venous congestion increases, and arterial blood flow drops causing tissue ischemia that produces further capillary leakage and cellular damage. Edema is the result causing a self-perpetuating increase in IAPs and subsequent ACS. Sepsis and multiple organ failure is the end result as the gut become more hypoxic from the ischemic episodes and the inflammatory response cycle (Malbrain et al., 2005; Shapiro, 2000).

The available literature on this subject is limited; however, research studies on various animal models have demonstrated decreased blood flow to the mucosal and intestinal organs when the IAP reaches 20 mmHg (Malbrain et al., 2005). Research has also discovered that impaired bowel perfusion has been linked to abnormalities in the normal physiologic gut mucosal barrier function. This produces a permissive effect on bacterial translocation that may contribute to septic complications associated with organ dysfunction and failure (Bailey and Shapiro, 2000; Lozen, 1999).

Table 7 Clinical Effects of Increased Abdominal Pressure on the Gut

System	Clinical Effects
*Intestinal/Mucosal	-Decreased blood flow to all abdominal organs except adrenals -Decreased mesenteric and mucosal blood flow -Decreased pH

### Summary

The primary pathophysiologic event leading to IAH and ACS is interstitial bowel and mesentery edema from capillary endothelial damage occurring from various insults such as sepsis, hemorrhage, etc. In addition, other related mechanisms commencing as secondary damage from pro-inflammatory cytokines released in a graded response to further add insult on the body. As a result, many liters of interstitial fluid accumulate within the intra-abdominal compartment causing the abdominal wall and fascia to slowly stretch until it becomes less compliant causing pressure to rise within the abdomen. Such elevations of IAP have a negative impact on organ perfusion throughout the body (Fox et al., 1999; Walker, 2003; Miller et al., 2004).

A consequence of increasing IAP is the occlusion of capillary blood flow and a compromise in venous return and arterial flow. This ultimately results in global ischemia triggering a viscous cycle of inflammation, capillary leakage, bowel edema, and a further increase IAP (Miller et al., 2004). Upon pressures exceeding 8-10 mmHg, physiologic compromise starts to occur in the gastrointestinal organs. Upon pressures reaching 20 mmHg, irreversible organ damage occurs resulting in ACS and multiple organ failure

where the mortality rate ranges from 16-100% (Miller et al., 2004).

Early recognition of rising IAPs is critically important. It allows for early employment of interventional strategies to reduce the risk of ACS and may lead to a better prognosis for the patient. A brief overview of IAPs and their associated effects to the organ system is shown in Table 4.

Table 8: IAP and Coinciding Organ Effects

	IAP 10-15 mmHg	IAP 16-20 mmHg	IAP >25 mmHg
	Stability	Mild Instability	Circulatory Collapse
*Cardiovascular	-preload increased -contractility unchanged -afterload unchanged -cardiac output increased	-preload decreased -contractility unchanged -afterload increased -cardiac output decreased	-preload markedly decreased -contractility reduced -afterload markedly increased -cardiac output marginal
*Renal	-No affect or slight reduction in urine output that is reversible	-oliguria -azotemia	-anuria -worsening azotemia with renal failure
*Splanchnic	-low grade intestinal ischemia -hepatic ischemia -bacterial translocation	-increased intestinal ischemia -increased hepatic ischemia -bacterial translocation	-bowel infarction -hepatic insufficiency -bacterial translocation
*Treatment	-maintenance of adequate intravascular volume	-volume expansion -possible decompression	-mandatory decompression

The Graded Response to Acute Increases in Intra-Abdominal Pressure. (Saggi et al., 1999).

## CHAPTER 3

### Clinical Presentation

ACS is the end result of sustained, uncorrected IAH. In clinical and laboratory settings, ACS has been shown to adversely affect all vital organ systems except the adrenal glands. Once thought of as a syndrome only associated with trauma patients, ACS are now encountered in all intensive care unit populations particularly those associated with major, life-threatening hemorrhage and shock, massive volume resuscitation, prolonged operation, and coagulopathy. Other related populations at risk consist of post-operative surgery for pancreatitis, repair of leaking or ruptured abdominal and thoracoabdominal aneurysms, and liver transplantation (Saggi et al., 1998; McNelis et al, 2003).

ACS is defined as an increase in IAP followed with an ensuing IAH and subsequent ACS as demonstrated with clinical signs and symptoms of a pathological dysfunctional abdomen with at least one organ in failure (Bailey & Shapiro, 2000; McNelis et al., 2003).

ACS has become an increasingly encountered problem in the critically ill and critical care setting. Although the clinical effects of ACS have been reported in many descriptive studies there is still little research regarding the identification of patients most at risk for the development of ACS (Malbrain, Deeren, & De Potter, 2005). Predictive variables associated with those at risk for ACS include manifestations originated from end organ dysfunction. Clinical of ACS are listed in Table 5.

Table 9 Clinical Parameters Suggestive of ACS

1	Distended Abdomen
2	IAP >20 mmHg
3	Elevated Peak Airway Pressure (up to or $\geq$ 85 cm H <sub>2</sub> O)
4	Massive IV Fluid Requirements ( $\geq$ 5/L in less than 24 hrs)
5	Oliguria to Anuria not Responding to Volume Repletion
6	Decreased Cardiac Output
7	Hypoxemia Refractory to Increases in FiO <sub>2</sub> and PEEP
8	Hypercarbia
9	Hypercapnia
10	Wide Pulse Pressure
11	Acidosis <7.20

Intra-abdominal hypertension in the critically ill: it is time to pay attention. (Malbrain et al., 2005)

A disparity between the pressure-volume curve of the abdominal cavity and its contents results in an elevated IAP causing adverse physiologic consequence as described above. As the patient depletes his/her physiologic reserves, the additional increase in pressure can result in multiple organ failure and death (Fox, Miller, & Nix, 1999). The number of organs in failure gives one an idea of the IAP; the more organs in failure, the higher the IAP (Malbrain, Deeren, & De Potter, 2005; Gallgher, 2004).

Although a direct physical examination of the abdomen can indicate a possible diagnosis of ACS, it should not be solely relied upon as a precise tool to adequately

determine the IAP. A study performed by Kirkpatrick (2000) showed that practitioners had a less than 50% chance of accurately diagnosing ACS based solely on a clinical exam and suggests that a clinical exam with IAP measurements is needed to make an accurate diagnosis.

### Diagnosis

The diagnosis of ACS depends on a very high degree of suspicion and the ability to recognize patients at risk, to be able to identify the clinical syndromes and effects of ACS, and to obtain accurate measurements of IAPs. Research studies do not indicate a clear consensus as to which patients benefit from aggressive IAP monitoring; however, it has been advocated that patients with risk factors for developing ACS do benefit from continuous IAP measurements (Morken & Michael, 2001). An IAH assessment algorithm is presented in Figure 3.

A clinical examination as the sole assessment of IAH is inaccurate (Kirkpatrick, 2000; Morken & Michael, 2001). In ACS, the abdomen is not always distended even though it is usually always tense (Peralta & Hojman, 2001). Because of inaccuracy of a physical exam to identify ACS, direct IAP measurements should be done.

IAP's can be monitored by two means: directly or indirectly. Direct measurements use an intraperitoneal catheter attached to a water manometer or pressure transducer. This method is used primarily during laparoscopic surgery; however, it is impractical to use in the ICU setting due to its potential complications of peritoneal contamination or bowel perforation (Lozen, 1999; Morken & Michael, 2001; Malbrain et al., 2005).

IAP may be indirectly calculated by measuring the pressure within a certain abdominal organ. Placement of a transfemoral catheter into the inferior vena cava is one form of indirect measurements for IAP, but its use is limited due to its potential complications of infection and thrombus formation. The second indirect method involves measurements of gastric pressure through gastrostomy or nasogastric tubes. Lastly, the current standard for monitoring IAPs is through the use of an existing Foley catheter to measure bladder pressures (Saggi et al., 1998; Lozen, 1999; Peralta & Michael, 2001; Morken & Michael, 2001; Malbrain et al., 2005).

The wall of the urinary bladder acts as a passive diaphragm when the bladder volume is between 50 and 100 mls. Recent literature suggests introducing 25-50 mls of fluid into the bladder. This has been found to have the lowest bias at clinically relevant IAPs (WSACS, 2007). Therefore in determining a precise bladder pressure reading, the optimal volume for the bladder is 25-50 mls of sterile solution (Fusco et al., 2001; WSACS, 2007). It is also important to recognize variables that might limit the wall mobility of the bladder and affect the reading. A neurogenic or a contracted bladder is among the variable factors of an inaccurate bladder pressure reading; however, these are fairly uncommon. In this case, other forms of indirect measurements can be used. For instance, inserting a central venous catheter into the inferior vena cava or using using a nasogastric tube to measure IAPs should be sought (Peralta & Hojman, 2001).

Bladder pressure can be measured by hooking a pressure monitoring system to the existing Foley catheter. A three-way Foley catheter is the most desirable, but one could also use a two-way Foley catheter. The three-way catheter allows the pressure to be

monitored by an irrigation limb without the need to repeatedly access a closed system with a needle. If a two-way catheter is used, an 18-gauge needle can be used to connect the pressure tubing into the port of the Foley (Bloomfield, Saggi, Blocher, & Sugerman, 1999). Two- and three-way Foley catheter monitoring system is presented in Figures 4.1 and 4.2.

The technique for measuring bladder pressures is as follows: first, fill the transducer using saline; second, calibrate the transducer to zero at the level of the symphysis pubis (ie, approximately the level of mid-thigh); third, prime the pressure circuit with the normal saline; fourth, connect the pressure tubing to the Foley as follows: 1) for the two way Foley catheter: connect the 18 gauge needle to the end of the pressure tubing and insert into the rubber sampling port of the urinary drainage device; 2) for the three way Foley catheter: connect the tube Y connector to the end of the pressure tubing. Empty the patient's bladder using a Foley catheter and empty the Foley tubing. Clamp the drainage bag as shown in figure 4.2 (Bloomfield, Saggi, Blocher, & Sugerman, 1999). While the patient is placed in a horizontal/supine position, use a 60 ml syringe and instill 25-50 ml of sterile normal saline into the bladder and clamp distally to the collection port. Ensure that the transducer is leveled with the bladder during the measurement. Close the stopcock off to the syringe and obtain the reading. The abdominal blood flow should produce fluctuations in the waveform with the heartbeat. The IAP is expressed in mmHg and taken at the end of expiration in a supine position to make sure that the abdominal muscles are not contracting giving a false reading (Fernandez, 2006).

In the clinical practice setting, the most practical way to measure an IAP is to use a tool that is minimally invasive and reproducible; therefore, the bladder is the best source. Furthermore, measuring a bladder pressure using an existing Foley catheter is well described in the medical literature as a standardized measurement tool for abdominal pressures. This technique is minimally invasive, safe, simple, and accurate (Lozen, 1999; Peralta & Michael, 2001). ACS is defined as IAH with a gradual and consistent increase in an IAP value of  $>20$  mmHg recorded during a minimum of three measurements that are performed 1-6 hours apart with at least one organ system in failure (Malbrain et al., 2005).

#### Management

The key to managing IAH and ACS is having the ability to identify the patient at risk and recognizing the signs and symptoms. Upon arrival to the ICU, bladder pressure measurements should be taken in patients who are considered at risk (Morken & West, 2001). Recent literature reports that ACS can develop within six to eight hours of a precipitating event (see Table 1.1-1.2) and thus frequent IAP measurements should be performed in patients who are at risk for IAH. It is reasonable to suggest measuring IAPs at least every one to two hours until a clear trend has been established and subsequently every four to six hours or as needed depending upon the patient's condition (Malbrain et al., 2005). Detailed information regarding the management of IAH and ACS can be found in Figure 5.

The evaluation and management of critically ill patients requires continuous assessment of clinical, laboratory, and physiologic information. It is ultimately important

for the ACNP to identify patients at risk for IAH and to be able to assess their overall clinical condition. The ACNP needs to be aware of potential misleading hemodynamic values associated with ACS such as inaccurate central venous pressures and pulmonary artery wedge pressures. Patients at risk for ACS should have their IAP measured, trended, and recorded (Malbrain, et al. 2005). Appropriate interventions should be determined based upon the clinical status of the patient and the bladder pressure measurements.

Patients who suffer from either medical, surgical, or trauma related injuries can have improved outcomes if a priority-orientated approach is put into effect. This means prioritizing either medical and/or surgical interventions so the patient can have improved outcomes based upon early decision-making skills (Chaudhry, Tiwari, & Singh, 2006).

Normal IAPs range from 0-5 mmHg. As the pressure increases inside the abdomen, physiologic compromise is revealed as early as 8-10 mmHg. A pressure of 20 mmHg or more is associated with organ compromise and irreversible tissue injury results (Cheatham, White, Sagraves, Johnson, & Block, 2000). However, researchers have yet to agreed upon an established IAP measurement requiring immediate interventional strategies to alleviate such pressures (Morken & Michael, 2001).

#### Classification and Grading Abdominal Compartment Syndrome to Guide Therapy

Measurements of an IAP are classified and graded in order to guide the practitioner's decision-making on medical and/or surgical interventions. The Burch Severity Guide for ACS categorizes IAP values into four severity groups (Table 10)

Table 10 The Burch Severity Guide for ACS

Grade I	10-15 mmHg	*These patients probably have no significant changes short of an acidotic pH value. The recommendation is to maintain normovolemia.
Grade II	16-20 mmHg	*Peak inspiratory pressures are increased in this group of patients. A hypervolemic resuscitation is in order in these patients, and some surgeons would argue decompression is indicated at this point.
Grade III	21-25 mmHg	*In these patients, the clinician should perform hypervolemic resuscitation and decompress at the bedside.
Grade IV	> 25 mmHg	*In these patients, hypervolemic resuscitation is necessary and decompression should be performed in the operating room.

Intraabdominal hypertension and abdominal compartment syndrome in the intensive care unit (Vitae, 2006).

ACS is a gradual process that is consistent with an IAP value  $\geq 20$  mmHg during a minimum of three standardized measurements and confirmed failure in at least one organ (Malbrain et al., 2005). Furthermore, recent literature has shown that an IAP as low as 10 mmHg can cause deleterious effects on organ functions (Fusco, Martin, & Chang, 2001). It is therefore necessary and prudent that the ACNP is aware that an IAP exceeding 10 mmHg should be intervened early to avoid ACS (Fox, Miller, & Nix, 1999; Malbrain et al., 2005).

Close monitoring for IAH and ACS requires surveillance of the physiologic parameters that indicates an elevated IAP. The choice to intervene either through medical or surgical management is dependent upon whether an improvement can be accomplished

through the intervention used. It must be said that it is better to prevent ACS than to allow it to occur and manage the sequelae of events to follow (Fusco, Martin, & Chang, 2001).

### Prevention

The key to stop ACS from emerging is through prevention. The first step in prevention is to recognize the patient at risk. As discussed previously in chapter 2, patients at risk are those that have primary, secondary, or recurrent types of ACS with each associated cause. Upon identifying the patient at risk, the next step is to measure the IAP and determine if the patient requires either medical or surgical interventions (Lozen, 1999; Biswajit, 2004; Wolf & Gallagher, 2006).

Early goal directed therapy consisting of medical interventions used to improve tissue perfusion and oxygen delivery is essential in preventing ACS and ensuring optimal survival of the patient (Wolf & Gallagher, 2006). IAP's can have a dramatic impact on the perfusion throughout the abdomen and oxygen delivery to the organs; therefore, the focus should be on reducing the IAP through perfusion and oxygen preservation (Saggi & Sugerman, 1999). This can be done through accurate IAP measurements and optimal resuscitation endeavors.

### Treatment

In the past, the focus has been emphasized on emergent surgical decompression for IAH and ACS. Today, detection of early IAH is amendable to non-surgical interventions. However, failure to detect IAH may lead to prolonged mesenteric ischemia and multiple organ hypoperfusion increasing the patient's morbidity and mortality.

Insomuch, by implementing medical interventions early, ACS may be prevented (Malbrain et al., 2001).

#### Non-Surgical Interventions

The most important aspect in preventing ACS is through several medical management interventions aimed at reducing the IAPs. As stated previously, fluid management is considered the first line tool used to prevent ACS through improving tissue perfusion and oxygen delivery to the surrounding abdominal tissues and organs.

Fluid management must be monitored carefully as under-resuscitation will often lead to organ hypoperfusion/ischemia, capillary leakage, tissue edema, and further increases in IAPs. If the patient is under-resuscitated, more fluid is required to increase the blood flow to the gut in order to reduce an acidotic state and maintain perfusion to the organs throughout the body. In contrast, over-resuscitation can be just as detrimental as increased fluid causes bowel edema and inflammatory mediator release (Walker, 2003).

There is a fine line of fluid balance in the management of high-risk patients for ACS prevention (Peralta & Hojman, 2001). Optimizing crystalloid infusion to avoid over-resuscitation and under-resuscitation is the key. Multiple indicators for effective resuscitation have been evaluated and used to accurately determine the fluid balance based on the patient's physiological need. According to several randomized clinical trials, lactate, base deficit, and gastric mucosal pH appear to be reliable indicators to guide resuscitation efforts (Fusco, Martin, & Michael, 2001). In addition, the central venous pressures and pulmonary artery wedge pressures can be used if a formula is used to correct for the falsely elevated values (Table 11) (Peralta & Hojman, 2001; Fusco,

Martin, & Michael, 2001; Malbrain et al., 2005). (Table 7).

Table 11 Calculations for Corrected Central Venous and Pulmonary Artery Pressures

$$\text{CVP corrected} = \text{CVP measured} - \text{IAP}/2$$

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$$\text{PAOP corrected} = \text{PAOP} - \text{IAP}/2$$

If the IAP continues to rise despite the initial fluid intervention, various other interventional strategies can be employed. Other non-surgical interventions to reduce IAPs include sedation, pain control, gut emptying, paracentesis, neuromuscular blockade, continuous renal replacement therapy, and continuous negative abdominal pressure.

A patient suffering from pain or agitation may benefit from medications that control such issues and in return, also reduces IAP. All patients with an increased IAP should have a nasogastric tube placed so that all excess air and fluid can be removed from the intestinal lumen. Inasmuch, stool and other intra-lumen contents that can cause bowel expansion should be cleared with medications such as cathartics, enemas, and rectal tubes. Upon inadequate sedation, a neuromuscular blockade may provide further relaxation to the patient's abdominal wall. Reduced because the abdominal wall tension can give the patient an opportunity for the accumulated fluid to mobilize. Patients with ascites or large retroperitoneal collections of fluid may benefit from percutaneous drainage. Paracentesis often needs to be repeated to maintain a low pressure; therefore, continuous IAP monitoring is very important (Malbrain et al., 2001).

Patients may not have fluid that can be easily removed because the fluid houses itself in the form of edema. Colloid infusion and hemofiltration are two appropriate

interventions used to reduce IAP's by reducing cytokine levels and maintaining an adequate intravascular volume to sustain homeostasis. Overall greater reductions in mortality and morbidity have been established in those who received hemofiltration as compared with those who did not receive hemofiltration with a 21% reduction compared to 83% not treated (Malbrain et al., 2001). Clearly, it has been established that upon early identification of an elevated IAP, non-surgical interventions for IAH exist and can be very promising (Peralta & Hojman, 2001).

#### Surgical Intervention for Abdominal Compartment Syndrome

When medical interventions fail to relieve the IAP and/or prevent ACS, surgical management is needed (Malbrain, et al., 2001). The only way to relieve ACS is by decompressive laparotomy where the goal is to release the pressure within the abdomen by opening the skin, fascia, and peritoneum. This particular procedure can be done either at the bedside or in the operating room.

ACS causes organ failure due to increased IAP. In order to prevent multiple organ failure, decompressive laparotomy is used (Miller, Meredith, & Johnson, 2004).

Although there is no set pressure at which to perform decompressive laparotomy, several factors contribute to the decision. These include age, underlying co-morbidities, and physiologic organ dysfunction (Malbrain et al., 2001). Decompressive laparotomy provides immediate relief of IAPs. The peritoneal cavity is usually left open post-operatively and managed by several different techniques (Walker, 2003).

Although there are several methods used to manage the open abdomen, the two most commonly used methods are the placement of a temporary mesh (the polyglactin

910 mesh was shown to be best suited for the open abdomen due to lower fistula rates and lower costs) with the vacuum pack dressing and a simple plastic covering such as the Bogata bag (Pleva & Mayzlik, 2004). The goal of these coverings is to prevent evisceration, protect the underlying viscera, and allow for faster abdominal wall closure (Miller, Meredith, & Johnson, 2004).

#### Complications Post-Operative

Surgical decompression is the treatment of choice for sustained IAH compounding to ACS. However, as with any surgical procedure, the benefit does not come without complications and risks.

Hypotension is one of the many complications after the abdomen is initially opened. It occurs due to the sudden release of vascular compression causing a decrease in central filling pressures and systemic vascular resistance. This complication can be avoided if, prior to surgery, the patient is given a decompressive cocktail of one liter of normal saline, two-three amps of bicarbonate, and 50 mg of mannitol that works to augment pre-load, neutralize the acute release of acids from the mesenteric circulation, and stimulate renal diuresis (Malbrain et al., 2001; Pleva & Mayzlik, 2004). In addition, the patient may also require multiple liters of crystalloid after surgery to avoid further episodes of hypotension (Pleva & Mayzlik, 2004).

Additionally, abdominal decompression may precipitate further adverse physiologic and metabolic events including respiratory alkalosis related to increased pulmonary compliance. If ventilator modifications are properly instituted, the patient may not have such adverse events. Furthermore, an open abdomen necessitates continuous

“washouts” to decrease the chances of infection and fistula formation. An accumulation of intra-abdominal products of anaerobic metabolism can result in excess acid and potassium. This can stimulate adverse cardiac events including arrhythmias or possibly asystole during or after a washout (Bailey & Shapiro, 2000). Interventions aimed at reducing the patient’s chance of adverse cardiac events should always be on the practitioner’s mind.

Fistula formation and inability to close the abdomen are additional consequences and complications of decompressive laparotomy with an open abdomen. Fistulas form due to exposure of the bowel to the specific dressings used, inflammation, and/or infection (Miller & Junger, 1997; De Waele, Hoste, & Malbrain, 2006). Extensive bowel edema, adhesions between the bowel and the abdominal wall, and sustained fistula formation also prevent the abdomen from complete closure (Suliburk et al, 2003; De Waele et al, 2006).

### Nutrition

Although the timing for abdominal wall closure is dependent upon several factors as dictated by the patient’s condition (acidosis, hypothermia, coagulopathy, fistula, etc), several studies show that patients with or without an open abdomen have improved outcomes with the initiation of early enteral feedings (Cothorn et al., 2004; Collier et al., 2007). In fact, withholding enteral nutrition in patients with an open abdomen is not supported by current research. Furthermore, delaying nutrition may result in severe underfeeding and caloric deficiencies that cause increased morbidity and mortality (Collier et al., 2007).

Several studies have demonstrated that early enteral feeding decreases both infectious and noninfectious complications in patients who critically ill or who have been severely injured. The mechanisms for the improved outcomes by early enteral feeding are: 1) preventing acute caloric malnutrition, 2) modulating the immune response, and 3) promoting gastrointestinal structure and function (Collier et al., 2007).

On the contrary, generalized gastrointestinal dysfunction caused by gastroparesis, an ileus, or fistula formation may require another form of nutrition. Parenteral nutrition, otherwise known as TPN, is substituted for enteral nutrition (Cothorn et al., 2004; Collier et al., 2007).

### Summary

Evaluation and management of critically ill patients requires comprehensive laboratory, clinical, and physiologic information. The ACNP should understand the various processes associated with rising IAPs and the subsequent ACS in order to provide appropriate and early interventional strategies.

Rising IAPs is a clinically silent process that may not show itself until the characteristics of organ dysfunction have been demonstrated. Prompt suspicion and recognition of early signs and symptoms is imperative to preventing full-blown ACS. Although a clinical exam is noteworthy, it is not always complete in detecting ACS (Kirkpatrick, 2000). Measurements of IAPs are necessary and can be done through direct or several indirect methods. However, bladder pressure measurements have been shown to be exclusively more accurate than any other indirect or direct measurement tool and should be used to detect rising abdominal pressures (Malbrain et al., 2004).

The clinician must be able to evaluate the patient and the IAP reading to determine if either a medical or surgical intervention is necessary. Several non-interventional strategies have been used and hold promises in preventing further complications leading to ACS. An important term to remember is that IAH is an urgent medical disease whereas ACS is an urgent surgical disease. Sustained IAH >20 mmHg with at least one organ in dysfunction necessitates surgical decompression in order to reduce further problems associated with ACS (Waele et al., 2006).

Decompressive laparotomy reduces IAPs, but it does not come without risks. The ACNP must be aware of several post-operative complications associated with decompressive laparotomy. Reperfusion syndrome is the term used for patients who harbor signs and symptoms of post-operative decompression, which includes hypotension, asystole, and further inflammatory cytokine release. Several post-operative complications are associated with this surgical procedure that the ACNP must be aware of. An important term to remember is that IAH is an urgent medical disease whereas ACS is an urgent surgical disease.

Withholding nutrition in the critically ill or the severely injured patient should be recognized as a possible source for increased morbidity and mortality. Several studies show that providing early enteral nutrition within 5-7 days improves patient outcomes. Patients who are unable to tolerate enteral feedings due to several gastrointestinal dysfunctional characteristics, TPN should be used.

## CHAPTER 4

### Significance of ACS in Advance Nursing Practice

A recently recognized syndrome of clinical importance is ACS. Due to its devastating multi-system effects, the mortality rate ranges from 10-100% during the first 24 hours of initial diagnosis. Although ACS is widely cited in the medical literature, it is not as well reported in nursing research articles. IAP and IAH have received little attention to nursing researchers for unknown reasons. However, it is important that the ACNP pay particular attention to patients considered at risk for ACS so that he/she can possibly prevent the devastating effects of this syndrome. As the ACNP's role is evolving to encompass more specific functions in the emergency department and intensive care units, the ACNP is in a unique position to impact patient outcomes through direct care and education (Howie & Erickson, 2002). The ACNP must stay up-to-date on the current literature in order to stay attuned and prepared to support his/her advanced practice.

Critically ill patients frequently have various physiologic derangements that stem from a variety of sources. It is the ACNP's responsibility to recognize the signs and symptoms associated with rising abdominal pressures. The ACNP has the ability to utilize various evidence based diagnostic tools in order to provide the best care and to improve patient outcomes.

Risk factors for ACS have been summarized in Tables 1.1 and 1.2. Patients at the highest risk are those who have had marked abdominal trauma with concomitant shock. It is important for the ACNP to keep a watchful eye on the clinical signs and symptoms of ACS, and to focus on changes that occur in the GI, renal, pulmonary, cardiovascular, and

neurological systems (Gallagher, 2004). Low urine output with hypovolemic shock, hemodynamic pressures unresponsive to fluid resuscitation, high peak airway pressures, and/or increased intracranial pressures are clinical signs that should prompt the practitioner for a high suspicion of ACS (Walker, 2003).

IAP measurement and clinical assessment of the patient are diagnostic tools in identifying a patient with ACS. Anticipating, recognizing, and treating the adverse effects of ACS is of critical importance for those caring for the critically ill. Nursing knowledge of the risk factors, clinical signs and symptoms, and treatments of IAH and ACS can reduce the morbidity and mortality associated with this syndrome. The ACNP should be able to identify the patient at risk, use appropriate diagnostic tools necessary to evaluate the IAP, and to decide proper initiation and timing of medical or surgical intervention.

#### Future Work

An area requiring research involves early goal directed therapy for sepsis and the development of ACS. According to several sepsis protocols, an extensive amount of fluid is required in certain patients. This places the patient at risk for IAH. Incorporating IAP monitoring into the current “sepsis bundle” protocol should be assessed. This may reduce the chance for unnoticed IAH and prevent the full effects of ACS. It will also promote safe resuscitation in patients with sepsis.

Recurrent ACS is a relatively new concept. There is a need for more knowledge in this new area. Future research should be conducted to determine the occurrence rate as well as any predisposition factors contributing to recurrent ACS. Research should also be conducted to determine whether continuous IAP monitoring is accurate and needed in

patients with an open abdomen. Other areas for future research involve the use of a barometric chamber to treat IAH. Hyperbaric oxygen therapy is a non-invasive medical form of treatment that involves breathing 100% oxygen at an increased pressure. Patients who breath 100% oxygen under pressure allows the oxygen to dissolve into the blood plasma and increases the amount of oxygen delivered to injured tissues (LDC, 2008). This may prevent ischemia due to devastating increases in IAPs and may minimize damage to various organ systems.

#### Summary

Increasingly being recognized as a steady problem, IAH and ACS produce complications in a subset of patients who have significant traumatic or surgical processes. It has been argued in various research articles that ACS is a medical and surgical disease. Research has shown that approximately 80% of patients in medical care units had patients with evidence of ACS. This is significant to the ACNP who provides advance nursing care across the continuum of health care in the ED, ICUs, and medical units.

IAH and the subsequent ACS affect all body systems most notably the cardiac, respiratory, renal, and neurological structures. Consequently, rising IAPs plays a significant role in sepsis and multiple organ failure seen in many trauma patients. ACS is gaining more attention as more cases are being identified. It is ultimately important that the ACNP understands the complex signs and symptoms of ACS as they are often mistakenly ascribed to the progression to a primary illness rather than to the development of ACS. The ACNP aides in the early identification of increased IAPs and initiate medical interventions and/or consult surgical interventions as necessary. However, failure

to recognize the presence of IAH and ACS can lead to severe tissue hypo-perfusion, MODS, and death.

In conclusion, it ultimately important to first recognize patients at risk, and to properly manage patients who demonstrate the signs and symptoms of rising IAPs. Increasing knowledge about this syndrome and its devastating effects can facilitate the ACNP to make appropriate and early choices to intervene that can reduce morbidity and mortality.

## CHAPTER 5

### Summary

Advances in health care during the past 20 years have increased survival rates in patients with significant trauma. However with recent advances in medical technology, new complications and syndromes are being recognized. Although IAH and ACS are not new processes, they have gotten recent attention due to the fact more cases are recognized, especially in medical, surgical, and trauma patients (Malbrain et al., 2005).

Compartment syndrome can occur within any enclosed anatomic space and as pressure increases within this space, capillary perfusion is compromised through direct compression of the venous and arterial system causing local tissue ischemia and necrosis (Edwards, 2004). The abdominal cavity is a space at risk for developing compartment syndrome (Saggi et al., 1999; Edwards, 2004; Malbrain et al., 2005). ACS is a clinical disease resulting from sustained increases in IAPs due to a variety of injuries causing tissue edema or free fluid collection in the abdominal space. Elevated pressures  $\geq 12$  mmHg in the abdomen are referred to as IAH, while the end stage of organ dysfunction and failure that occurs due to the pathophysiologic effects of sustained pressure  $\geq 20$  mmHg is called ACS (Bailey & Shapiro, 2000; Malbrain, et al., 2005). The end result of undetected and untreated IAH results in multi-organ dysfunction and death. The practitioner's approach is reflected in patient outcomes (Bailey & Shapiro, 2000).

The ACNP is in a unique position to recognize patients at risk for ACS and to optimize their care and promote better outcomes. Knowledge and understanding of the processes associated with increased IAPs, IAH, and ACS is a key element in providing

appropriate medical care to patients at risk. It is important to recognize patients at risk by trending their IAPs with precise measurement tools, and to consider medical interventions based upon the pressures measured (Lozen, 1999; WSACS, 2007).

It is no doubt that ACS is a potentially lethal condition caused by any event that produces a rise in IAP. Although there are many causes of ACS, the most common cause is over-resuscitation requiring  $\geq 5/L$  in 24 hrs and blunt abdominal trauma (Walker & Criddle, 2003). Other conditions associated with a rise in IAP are massive intra-abdominal or retroperitoneal hemorrhage, severe gut edema, intestinal obstruction, ascites, etc. (Bailey & Shapiro, 2000; Malbrain et al., 2005). The ACNP should be aware of possible causes of IAH and ACS.

The consequences of ACS are profound and affect many vital organ systems in the body. The pathophysiologic consequences of a raised IAP are transmitted initially to the pleural space decreasing compliance of the lung. Hypoventilation and an altered ventilation/perfusion distribution ultimately result in hypoxemia and hypercapnia. Mechanical ventilation is often necessary with high inspiratory pressures in order to deliver the required tidal volume. With the combined increases in abdominal pressure and pleural pressure, the blood return to the heart is compromised and afterload is severely increased. Due to decreased venous return and cardiac output, perfusion to the intra-abdominal organs is critically reduced leading to oliguria and renal failure. Splanchnic and hepatic ischemia also occurs as reflected by a low mucosal pH, decreased liver metabolism, and bacterial translocation (Peralta & Hojman, 2001; McNelis et al., 2003; Malbrain et al., 2005). The release of cytokines, formation of oxygen free radicals, and

decreased cellular production of adenosine triphosphate are pathophysiological effects of IAH. Such processes are therefore a cause of bacterial translocation from the gut and intestinal edema that produces even higher IAP predisposing the patient to MODS (McNelis et al., 2003). Furthermore, perfusion to the abdominal wall is also decreased resulting in impaired wound healing. Lastly, intracranial pressures may also be increased due to the decrease in cerebral perfusion pressure and increased venous pressure (Malbrain et al., 2005).

With normal IAPs ranging between zero and 5 mmHg, a pressure  $\geq 10$  mmHg has been shown to cause detrimental effects to various organ systems throughout the body. At this point, aggressive fluid resuscitation can be used to maintain perfusion and oxygen delivery to the organs. The practitioner can also choose other interventions to reduce IAP such as using more sedation and/or paralytics, empty the bowel using cathartics or an enema, hemofiltration, etc. Decisions to provide interventions should be discussed early to avoid ACS. When the pressure reaches  $\geq 20$  mmHg, surgical decompression is urgent to relieve ACS (Malbrain et al., 2005).

The diagnosis of this syndrome can often be difficult as it usually occurs in a gradual process in critically ill patients. Obtaining and performing a physical examination as well as recording an IAP measurement are recommended in order to identify IAH and ACS (Malbrain et al., 2005). Indirect monitoring of IAPs is relatively noninvasive and straightforward. It has been shown that using the bladder for pressure measurements is an accurate tool to obtain a pressure that is equivalent to that of an actual IAP (Kirkpatrick, 2000).

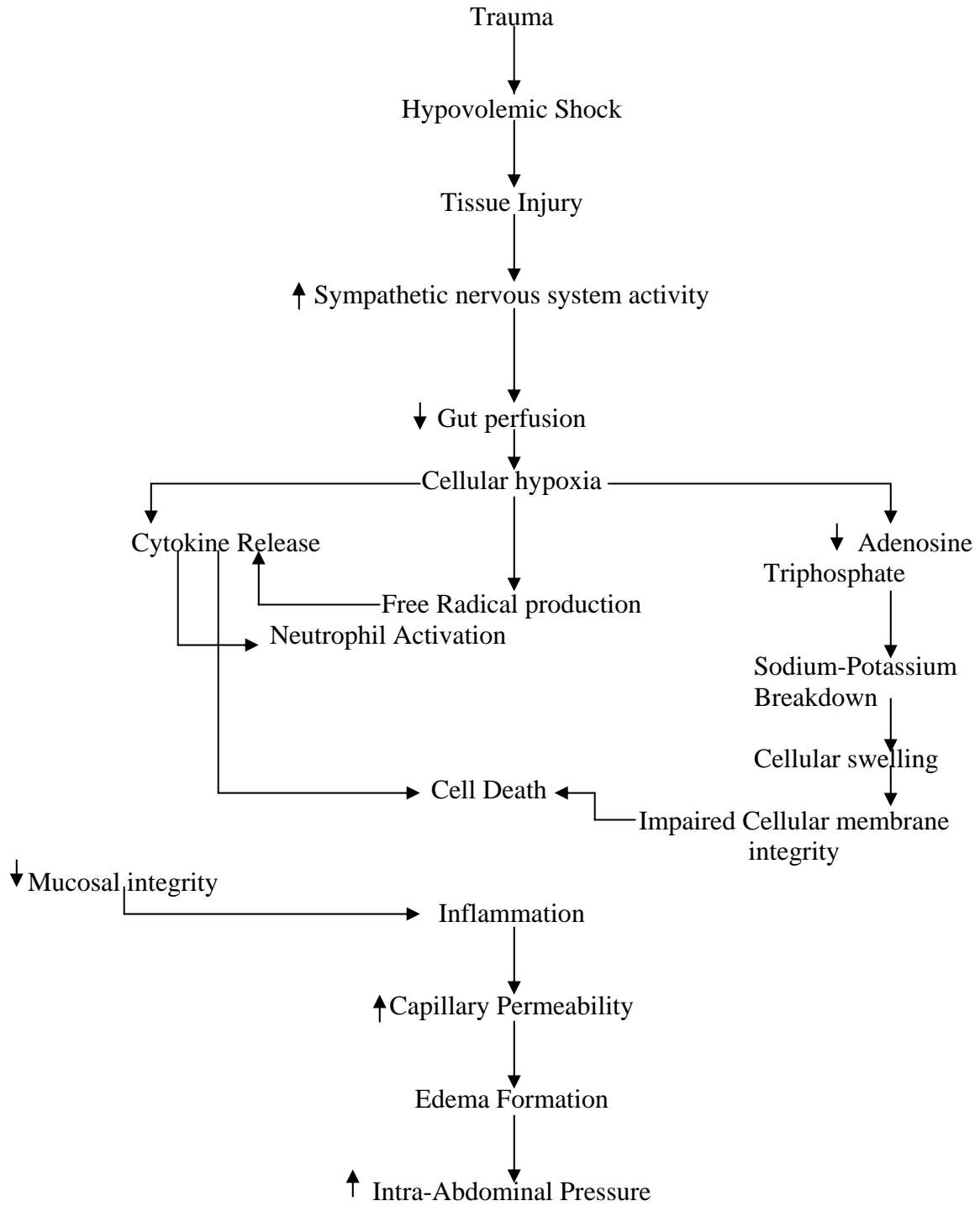
Medical management is often promoted to prevent ACS from occurring. Sustained IAH  $\geq 20$  mmHg with at least one organ in failure suggests that surgical intervention is necessary. Urgent decompressive laparotomy and fluid management are key elements in treating ACS.

The ACNP working with patients in the trauma and medical units must be aware of the potential for IAH and the pathophysiologic effects of ACS. It is important that the ACNP recognizes and anticipates potentially life-threatening complications and promptly initiates medical interventions or consults surgical intervention to decrease the mortality and morbidity of the patient. Therefore, the ACNP must stay abreast to the new and expanding knowledge on IAH and ACS. The mortality rate of ACS is high. Recognizing patients at risk, monitoring the abdominal pressure frequently, and early initiation of treatment could significantly reduce the morbidity and mortality.

The use of an exiting Foley catheter to measure bladder pressure that reflects IAP has been suggested. However, caution should be exercised as the patient may have increased risk for infection.

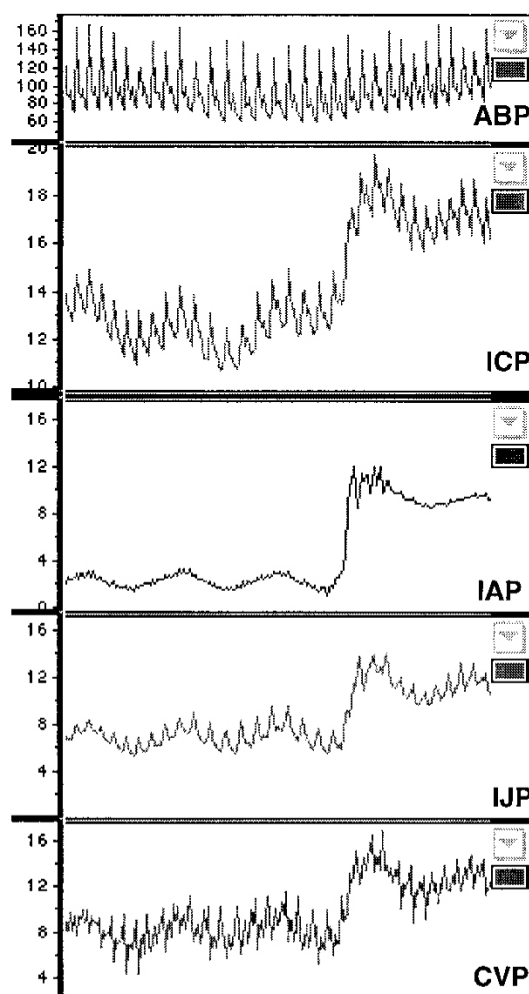
Finally, it is important not to wait for the signs and symptoms of ACS to be present to check the IAP; by then, the chance to provide medical therapy is lost. Monitor all high-risk patients early and often is crucial. Clinicians should trend the IAP similar to vital signs, and intervene early before critical pressures develop. It is also important to point out that the higher the IAP, the more organs in failure and a decrease chance of survival for the patient.

Figure 1 The Pathophysiologic Concept Behind Intra-Abdominal Hypertension



Saleem &amp; Amhed, 2006.

Figure 2: Direct Impact of Intra-Abdominal Pressures on Common Pressure Measurements



(\*Subject: An IAP was simulated by placement of a 15-liter bag placed on the patient's abdomen; IAP elevation causes immediate increases in ICP, IJP and CVP)

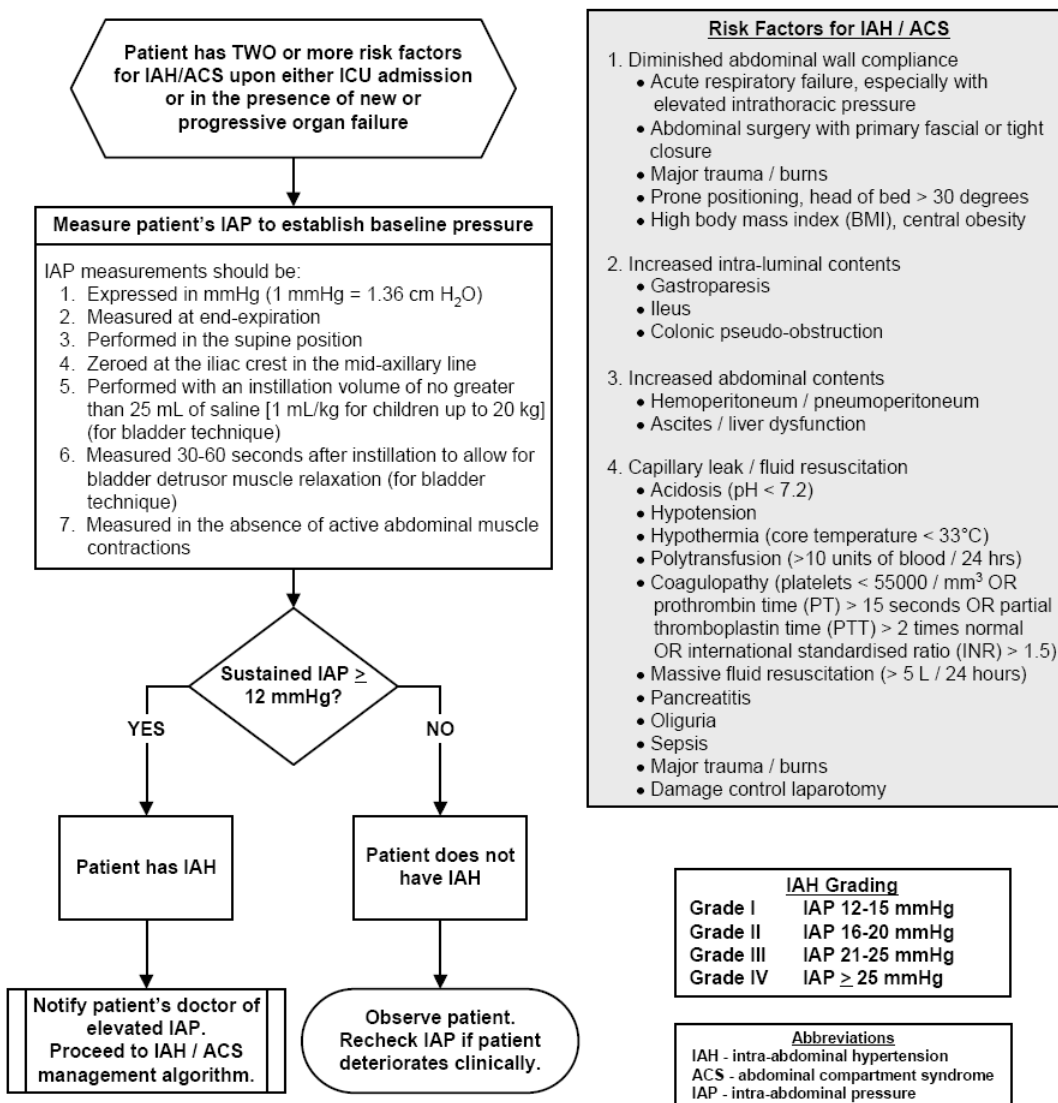
\*Note: ABP: arterial blood pressure; ICP: intracranial pressure; IAP: intra-abdominal pressure; IJP: intra-jugular pressure; CVP: central venous pressure

Induced abdominal compartment syndrome increases intracranial pressure in neurotrauma patients: a prospective study. (Citerio G, et al.,2001).

Figure 3: Intra-Abdominal Hypertension Assessment Algorithm

### INTRA-ABDOMINAL HYPERTENSION (IAH) ASSESSMENT ALGORITHM

- Patients should be screened for IAH/ACS risk factors upon ICU admission and with new or progressive organ failure.
- If two or more risk factors are present, a baseline IAP measurement should be obtained.
- If IAH is present, serial IAP measurements should be performed throughout the patient's critical illness.



\*Note: IAH: intra-abdominal hypertension; ACS: abdominal compartment syndrome; IAP: intra-abdominal pressure; ICU: intensive care unit Assessment Algorithm. (WSACS, 2007).

Figure 4.1 Three-Way Bladder Pressure Device

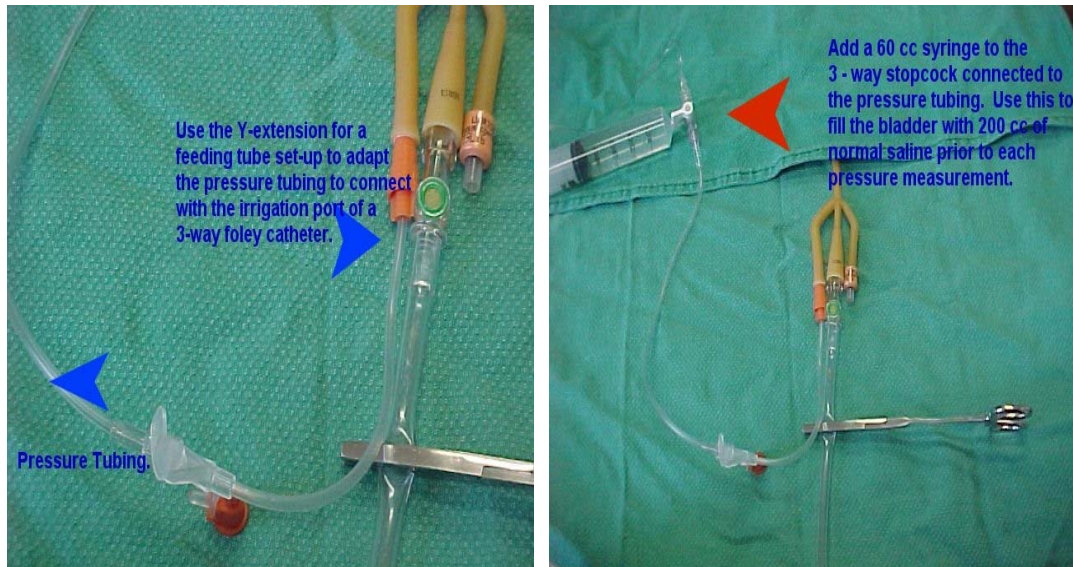


Figure 4.2 Two-Way Bladder Pressure Devices

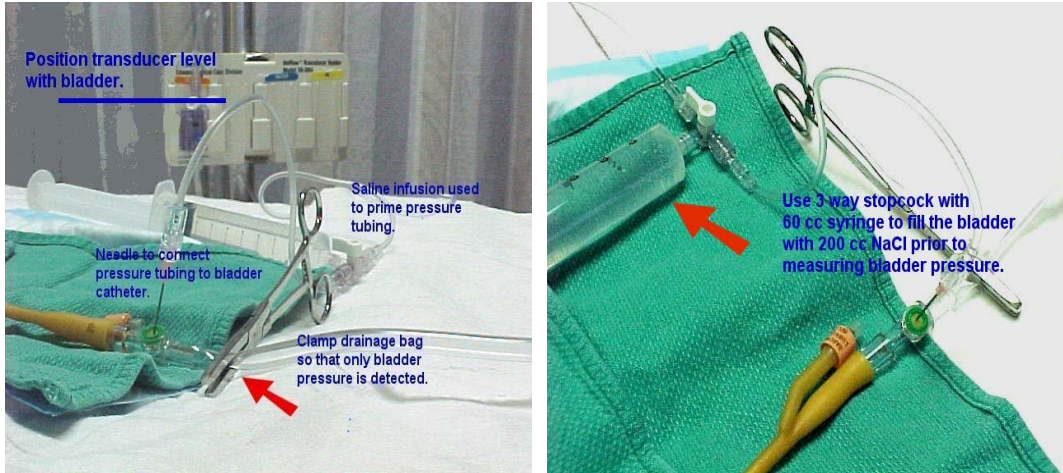
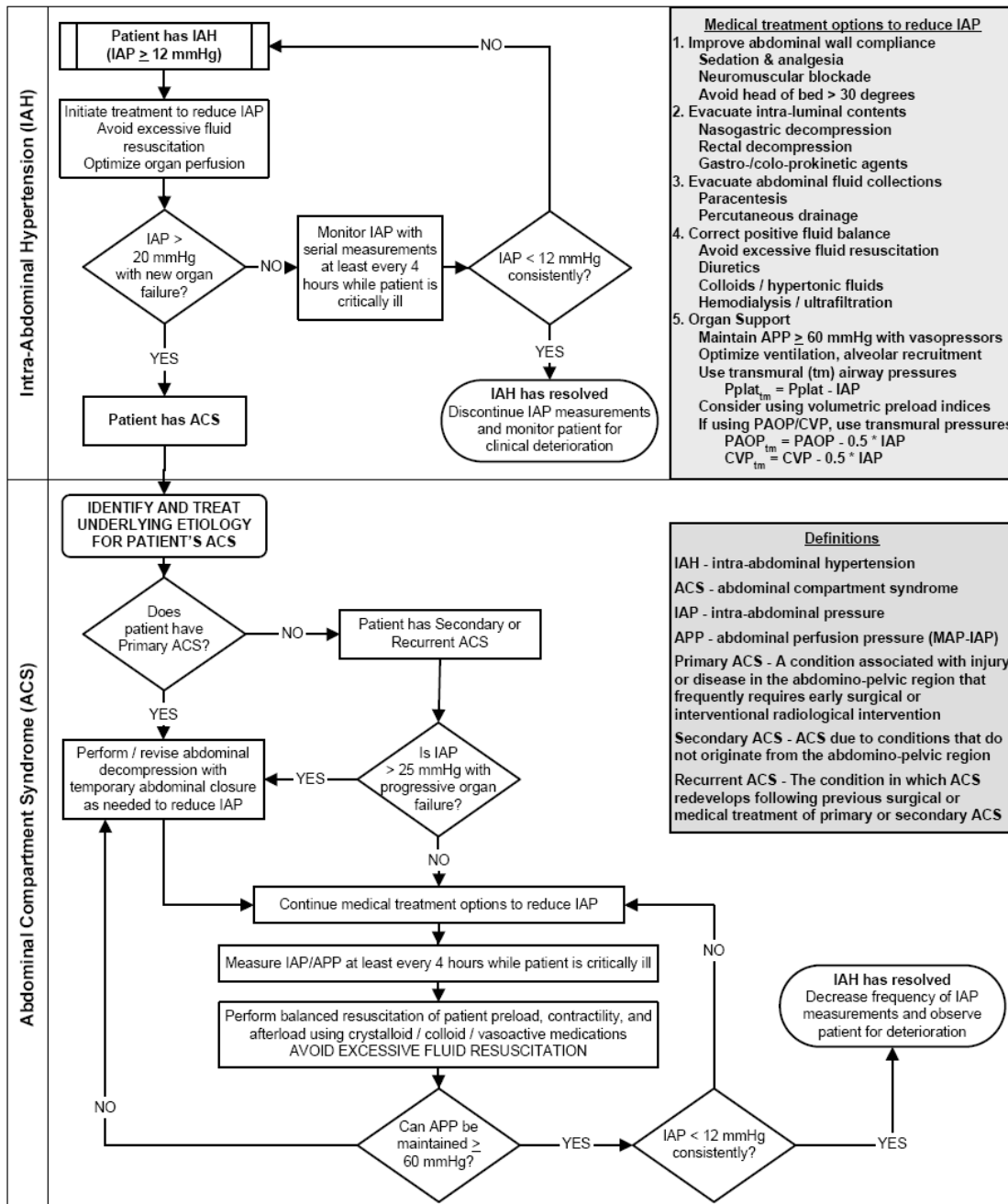


Figure 5: Intra-Abdominal Hypertension/Abdominal Compartment Syndrome Management Algorithm

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



Assessment Algorithm. (WSACS, 2007).

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