The Use of Routine Intra-abdominal Pressure Monitoring of Adult Surgical ICU Patients for the Early Detection of Abdominal Compartment Syndrome

by

Patricia Carta

A Thesis

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In Partial Fulfillment of the Requirements for the Degree of

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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements of the degree

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MASTER OF NURSING

Dedication

To Arthur,

for your support, encouragement, and patience, you inspire me everyday.

Abstract

Abdominal compartment syndrome (ACS), a condition associated with high mortality rates, results in multiple organ dysfunction secondary to intra-abdominal hypertension (IAH). The three objectives of this study were to determine; i) clinical outcomes associated with systematic tracking of intra-abdominal pressure (IAP) by measuring abdominal perfusion pressures (APP) every 6-8 hours; ii) the relationship between patients with a positive fluid balance and the development of IAH; iii) the relationship between IAP readings and pressure within the inferior vena cava (IVC).

Using a quasi-experimental design, a pilot study of 18 patients at high risk for developing ACS was conducted over a period of one year in a Surgical ICU. Bladder pressure, a non-invasive method to estimate IAP, and mean arterial pressure (MAP) were used to calculate APP (MAP – IAP). IVC pressure was estimated from pressures at a femoral vein catheter.

The findings from this study showed no significant difference in using an APP of 50 mmHg or greater as a predictor for patient survival and development of ACS. However, survivors of IAH had significantly higher APPs than non-survivors (p=0.0337). There was a positive correlation between IVC pressure and IAP (r = 0.92913, P = <.0001), and a positive correlation between maximal IAP and a positive fluid balance (r = 0.35666, p = 0.008).

Evidence from this study suggests that routine monitoring of intra-abdominal pressures may contribute to improved patient outcomes. Further studies of APP monitoring are necessary to determine its value as an aid in early detection of IAH and ACS. The correlation of IAP and IVC pressure provides a foundation for further research.

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Chapter I

Statement of the Problem and Conceptual Framework

Introduction

In this chapter, the significance of elevated intra-abdominal pressure and its measurement in the early detection of abdominal compartment syndrome (ACS) is described.

Abdominal compartment syndrome (ACS) has great relevance in the practice of surgery and the care of critically ill patients because of the effects of elevated pressure on multiple organ systems within the confined space of the abdomen (Cheatham, 1999). ACS has been defined as multiple organ dysfunction secondary to elevated intra-abdominal pressure (IAP) (Cheatham, 1999). ACS results when an acute increase in intra-abdominal pressure occurs that is sufficient to compromise vascular inflow, threatening the viability of the tissues within the abdomen. Early recognition is important, and the need for surgical decompression may be urgent. Medical and nursing expertise is required for the early detection and treatment of ACS. Nursing knowledge of the risk factors and clinical signs of ACS would likely assist in reducing high morbidity and mortality rates associated with this syndrome. Early recognition of the signs of ACS is essential to enhance prevention of the syndrome and to ensure optimal chances of patient survival. The exact numbers of patients diagnosed with ACS is not known, nor have the monetary costs to the Manitoba health care system been identified. However, the Health Sciences Center Surgical ICU in Manitoba has experienced an increase in the number of

patients diagnosed with ACS over the past two years, especially in the burn patient population.

Significance of the Problem

Elevated intra-abdominal pressure (IAP) has been shown to impair cardiovascular function, restrict respiratory function, and impair renal function (Cheatham, 1999). The importance of measuring IAP in critically ill surgical patients is well documented (Cheatham, 1999). This practice is based on three factors: (i) marked intra-abdominal hypertension (IAH) or elevated intra-abdominal pressure (IAP) causes abdominal compartment syndrome (ACS); (ii) untreated ACS is routinely fatal with reported mortality rates ranging between 42% and 100% (Kron, Harmon, & Nolan, 1984; Morris, Eddy, Blinman et al., 1993); (iii) routine intra-abdominal pressure measurements and abdominal decompression surgery are associated with a substantial improvement in survival rates. ACS, therefore, should be viewed as the end result of a progressive, unchecked increase in IAP from a myriad of disorders that eventually leads to multiple organ dysfunction (Saggi, Sugerman, Ivatury, & Bloomfield, 1998).

Most literature on ACS refers to trauma, vascular and general surgery patients (pre-operative and post-operative). Causes of abdominal compartment syndrome include peritonitis, necrotizing pancreatitis, small bowel obstruction, blunt trauma, severe bowel edema following a prolonged operation on the abdominal aorta, and burns (Cheatham, 1999).

ACS in burn patients has not been widely reported (Ivy, Atweh, Palmer, Possenti, Pineau, & D'Aiuto, 2000). Greenhalgh and Warden (1994) first reported the occurrence of ACS in their pediatric burn population. They noted that the incidence of ACS increased with burn size and depth, and their study provided evidence that burn patients are at risk for the development of ACS. Ivy et al. (2000) conducted a prospective study

to determine the occurrence of ACS in adult burn patients. They concluded that ACS was seen in patients with more than 55 % burns, and intra-abdominal hypertension occurred commonly in major burn patients. These authors observed that patients who developed ACS received more than 25 litres of fluid and had a mean IAP of 40 mmHg.

Consequences of ACS are the direct vascular, organ compression and diaphragmatic elevation which are the key mechanisms leading to circulatory collapse. ACS causes cardiovascular, pulmonary, renal, splanchnic, and intracranial disturbances (Saggi, Sugerman, Ivatury, & Bloomfield, 1998). Increased intra-abdominal pressure (IAP) is associated with oliguria and anuria. It has been shown that the kidney is one of the most sensitive organs to the ischemic insults produced by IAP (Altintas, 2001). Acute renal failure in critically ill patients has a multifactorial aetiology. If ACS develops, only prompt relief of the elevated intra-abdominal pressure is associated with the immediate return of renal function.

Cheatham, White, Sagraves, Johnson, & Block (2000) have suggested that calculating abdominal perfusion pressure (APP), defined as mean arterial pressure (MAP) minus IAP, should be of greater diagnostic and therapeutic value than either MAP or IAP alone as a predictor of patient survival. This is a new concept that has not been widely researched. Calculating the APP addresses not only the severity of IAH present, but the adequacy of tissue perfusion and need for additional resuscitation.

ACS has recently been "rediscovered" as a cause of significant morbidity and mortality in critically ill surgical and medical patients. The simple technique of IAP measurements, combined with early surgical intervention, has been demonstrated to improve patient survival (Sugrue et al., 1995; Meldrum et al., 1997; Ivy et al., 2000).

Researchers emphasize that the key to early detection of ACS is routine intra-abdominal pressure measurements. The indirect method of measuring IAP is the intravesicular or "bladder pressure." This has become the "gold standard" for measurement of IAP. This is a non-invasive technique of measurement via a urinary foley catheter. This simple technique can be initiated by the nurse and measured every six - eight hours. The use of routine IAP monitoring is currently not the usual practice for patients at risk of developing ACS; therefore, patients are at a greater risk for developing multiple organ dysfunction which may result in death (Ivatury, 2000; Cheatham, 1999).

Purpose of Study

The primary objective of this study is to investigate clinical outcomes associated with routinely monitoring abdominal perfusion pressures (APP) every 6-8 hours for surgical patients at risk of developing abdominal compartment syndrome (ACS). Abdominal perfusion pressures defined as (mean arterial pressure (MAP) minus intra-abdominal pressure (IAP) will be calculated when intra-abdominal pressures are measured (urinary bladder pressures are used to estimate IAP). Specifically, the objective was to determine if an APP of 50mm Hg or greater is a predictor of either patient survival or the prevention of ACS. A second objective was to determine the relationship between patients with a positive fluid balance and the development of intra-abdominal hypertension (IAH). It was thought that patients with a positive fluid balance had increased IAP. A third objective was to determine the relationship between IAP readings and pressure within the inferior vena cava measured at a femoral vein catheter. It was hypothesized that a positive correlation of IAP and inferior vena cava pressure would

provide evidence supporting the value of monitoring inferior vena cava pressures on a continuous basis. In patients with femoral venous catheters in place, this method of estimating IAP is efficient, less time-consuming and technically less demanding for the nurse compared to measuring urinary bladder pressures.

The Research Questions

1. Is the routine monitoring of APP (abdominal perfusion pressure) every 6-8 hours a clinically useful predictor of patient survival or the development of ACS during the first 96 hours following ICU admission?
2. Is there is a relationship between a positive fluid balance and an increase in intra-abdominal pressures during the first 96 hours following ICU admission?
3. Is there is a correlation between IAP and inferior vena cava pressure measured from the femoral venous catheter?

Hypotheses

1. The routine monitoring of APP (abdominal perfusion pressure) every 6-8 hours is a clinically useful predictor of patient survival or the development of ACS during the first 96 hours following ICU admission.

2. There is a positive correlation between a positive fluid balance and an increase in intra-abdominal pressures during the first 96 hours following ICU admission.

3. In patients with femoral venous catheters, there is a positive correlation between IAP and inferior vena cava pressure.

Assumption

The principle assumption of the study is that earlier detection of intra-abdominal hypertension will lead to earlier treatment and prevention of ACS or multi-organ system dysfunction (Saggi et al., 1998; Ivatury et al., 2000).

Limitations

There are two main limitations of the study. The first limitation is that the nurses may have varying levels of expertise and may not recognize the signs and symptoms of ACS as promptly as a more experienced nurse. The second limitation may be the sample size of this study; eighteen patients may be more reflective of a pilot study.

Conceptual Framework

The nursing model of Human Response to Health and Illness (Mitchell, Gallucci, & Fought, 1991) is the conceptual framework that guided the research. This model was selected because it emphasizes that the human response to illness is central to nursing

science, and viewing the human response from multiple perspectives: behavioral, experiential, pathophysiologic, and physiologic is necessary for a holistic approach to care. Mitchell, Gallucci, & Fought (1991) believe that this approach enables the organization of knowledge emerging from nursing science, and allows the practitioners of nursing to appreciate the link between the perspectives from which they will derive clinical strategies. By considering multiple perspectives, gaps in our knowledge are exposed and directions for future research are provided.

Nursing as a practice discipline focuses on the response of individuals to states of illness and to situational or developmental transitions or crises (Mitchell et al., 1991). Individual human responses in any of these situations can be viewed from one of the four perspectives mentioned earlier. The patient diagnosed with abdominal compartment syndrome can be analyzed from each of these perspectives. This model (Figure 1) depicts the relationship of the four perspectives on human response to the internal and external environment.

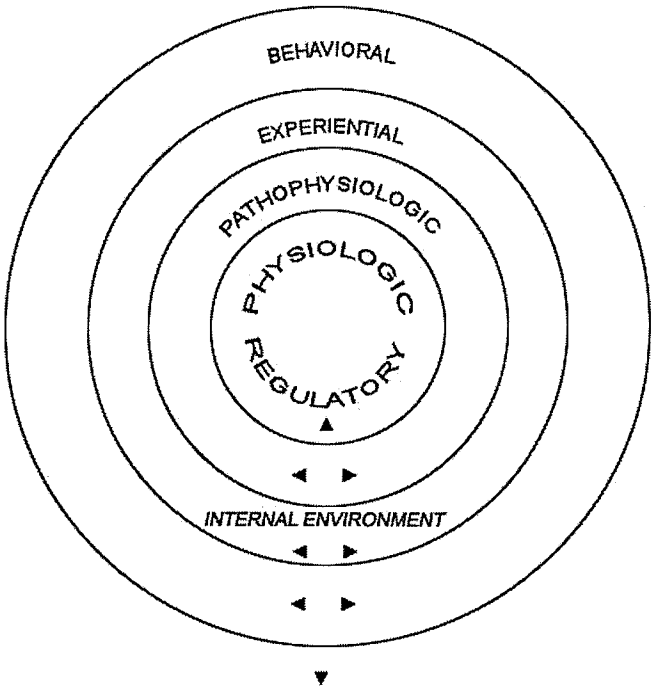


Figure 1. Human Response to Health and Illness Model. Relationships of the four perspectives on human responses to the internal and external environment. The arrows indicate that responses within each perspective are capable of transmitting and receiving information from all other perspectives. The behavioral responses are the ones that allow humans to communicate other responses to the external environment, including other human beings.

(Mitchell, Gallucci, & Fought, 1991, p.155)

Physiologic regulatory responses are based on the concept of normative or usual biologic functioning and include phenomena measured by the instruments of the biological sciences (Mitchell et al., 1991). The body, including the abdomen, is regulated by the microcirculation of arterioles, capillaries, and venules responsible for regulation of material exchange between the plasma and intestinal fluid surrounding the tissues in the body through processes such as diffusion, filtration, and vesicular transport (Sherwood, 1993).

Pathophysiologic responses result from a disordered biologic functioning, with phenomena observable by instruments of the biological sciences. A patient with abdominal compartment syndrome no longer has the normal regulatory response, and decompensation begins very rapidly. This is a result of the excess amount of edema in the abdominal cavity. Increased intraperitoneal volume is the most common source of elevated intra-abdominal pressure (Cheatham, 1999). An increase in intra-abdominal pressure leads to compression of the arteries and veins causes renal impairment resulting in a decreased urine output. This increased abdominal pressure leads to impaired pulmonary ventilation and may lead to respiratory acidosis. Experiential Responses include concepts of introspection; personal experience, and the derivation of shared meaning, which are measured by self report. It is through the patient's verbalization that we can understand how meaning is made of the experience that is engendered by physiologic and pathophysiologic responses (Mitchell et al., 1991). Patients experiencing abdominal compartment syndrome are fearful of the changes they are feeling with a tense abdomen. They report extreme pressure, difficulty breathing, they become restless, and express a feeling of panic. Burn patients with abdominal compartment syndrome experience the most difficulty as they have also sustained a massive burn injury along with the abdominal compartment syndrome.

Behavioral Responses are directly observable and measurable motor and verbal behaviours. A patient with intra-abdominal hypertension will exhibit increased IAP's, increased peak airway pressures making breathing difficult, and is accompanied by abdominal distention, decreased urine output, and hemodynamic instability. Patients may experience severe discomfort, pain, and restlessness due to the difficulty in breathing.

This framework provides the clinician with an appreciation for the complexity of human responses. The categorization of physiologic, pathophysiologic, behavioral, and experiential responses are useful teaching tools in promoting the nurses' ability to appreciate, predict, and assess the full range of responses to illness and injury for the patient diagnosed with abdominal compartment syndrome (Mitchell et al., 1991). In addition, this conceptual framework provides the basis for researching the clinical outcomes of critically ill hospitalized individuals at risk for developing ACS who participated in this study.

Conclusion

Abdominal compartment syndrome has recently been "rediscovered" as a cause of significant morbidity and mortality in critically-ill surgical and medical patients (Cheatham, 1999). Increased IAP can cause marked deficits in global perfusion and end-organ function which, if unrecognized, may cause death. A high index of suspicion is essential to the rapid diagnosis and treatment of ACS. The simple technique of IAP measurements and the calculation of APPs, as well as early surgical intervention, have been demonstrated to improve patient survival (Sugrue et al., 1995; Meldrum et al., 1997; Ivy et al., 2000). This study has the potential to increase the awareness and appreciation of the presence and pathophysiologic consequences of intra-abdominal hypertension and subsequent ACS, on the part of critical care nurses, surgeons, and intensivists working with burn patients, and for those working in intensive care units with other patient populations at risk of developing ACS. Routine IAP monitoring may lead to significant reductions in mortality rates of these critically ill patients.

Definition of Terms

Intra-abdominal Pressure (IAP)

- The measurement of the pressure inside the abdominal compartment (normal is 0 mmHg to subatmospheric). It can be measured indirectly by monitoring the pressure in the urinary bladder (Saggi et al., 1998).

Intra-abdominal Hypertension (IAH)

- Is present when the IAP reading is >15 mmHg. IAH occurs when the abdominal contents expand in excess of the capacity of the abdominal cavity (Saggi et al., 1998).

Abdominal Compartment Syndrome (ACS)

- Is a late manifestation of intra-abdominal hypertension, characterized by increased airway pressures, hypoxia, difficulty in ventilation, and anuria that improves with abdominal decompression surgery (Saggi et al., 1998).

Hypoxia

- An inadequate, reduced tension of cellular oxygen, characterized by cyanosis, tachycardia, hypertension, peripheral vasoconstriction, and mental confusion (Mosby, 1990, p. 598).

Hypoxemia

- An abnormal deficiency of oxygen in the arterial blood (Mosby, 1990, p.598).

Anuria

- Absence of urine formation (Bell, 1998).

Azotemia

- The retention in the blood of excessive amounts of nitrogenous compounds (Mosby, 1990, p.118).

Oliguria

- A diminished capacity to form and pass urine, less than 500 mls in every 24 hours, so that the end products of metabolism cannot be excreted efficiently (Mosby, 1990, p.836).

Acute Renal Failure

- The inability of the kidney to excrete metabolic end-products and to maintain electrolyte balance (Bell, 1998).

Ischemia

- Decreased blood supply to a body organ or part, often marked by pain or organ dysfunction (Mosby, 1990, p.649).

Summary of Chapter

In this chapter, intra-abdominal hypertension as a cause of, and early indication of ACS and the associated multiple and profound physiologic abnormalities both within and outside the abdominal cavity are described.

The main focus of the study, the potential contribution to improved patient outcomes of careful IAP monitoring, prompt recognition and treatment of elevated IAP are discussed. The rationale for using bladder pressure measurements as a proxy for IAP in deciding upon management of high risk patients before the actual onset of the signs

and symptoms of ACS is presented. The significance of a positive correlation between positive fluid balance and intra-abdominal hypertension is outlined (Fusco, Martin, & Chang, 2000), and the rationale for comparing urinary bladder pressures with femoral venous pressures to estimate inferior vena cava pressures is also provided. Inclusion in the study cohort of individuals with major burns was explained.

The assumptions of the study are described, and the use of the Human Response to Illness model as the conceptual framework for the project is discussed (Mitchell, Gallucci, & Fought, 1991).

Chapter II

Literature Review

Introduction

In this chapter, a review of the literature concerning abdominal compartment syndrome (ACS) is presented using the Human Response to Illness Model to direct the review process. The Human Response to Illness Model's framework, including the physiology and pathophysiology of ACS is reviewed and the experiential and behavioral manifestations described. The importance of routine intra-abdominal pressure monitoring is addressed.

Background

Elevated intra-abdominal pressure (IAP) has been shown to impair cardiovascular, respiratory, and renal function (Cheatham, 1999). The importance of measuring intra-abdominal pressure in critically ill surgical patients is well documented (Cheatham, 1999). This practice is based on three concepts. The first is that marked intra-abdominal hypertension (IAH) or elevated intra-abdominal pressure causes abdominal compartment syndrome (ACS) (Ivy, Atweh, Palmer, Possenti, Oineau, & D'Aiuto, 2000). The second is that untreated ACS is routinely fatal with reported mortality rates ranging between 42% and 100% (Kron, Harmon, & Nolan, 1984; Morris, Eddy, Blinman et al., 1993; Ivy et al., 2000). The third is that routine intra-abdominal pressure measurements and abdominal decompression surgery are associated with a substantial improvement in survival rates (Ivy, Atweh, Palmer, Possenti, Oineau, & D'Aiuto, 2000).

Currently, IAP measurements are not routinely implemented for patients in the majority of Canadian and U.S. hospitals (Ivy et al., 2000).

The effects of elevated intra-abdominal pressure (IAP) have been known since 1890 when Heinricus demonstrated that elevation of IAP to 27 and 46 cm H2O led to death in porcine models due to impaired diaphragmatic excursion leading to increased intra-thoracic pressure and respiratory failure (Cheatham, 1999). Abdominal compartment syndrome has great relevance in the practice of surgery and the care of critically ill patients because of the effects of elevated intra-abdominal pressure (IAP) within the confined space of the abdomen on multiple organ systems (Ivatury, Diebel, Porter et al., 1997). Sustained elevation of intra-abdominal pressure also referred to as intra-abdominal hypertension (IAH), causes increased intrathoracic pressure and abnormalities in pulmonary dynamics, increased afterload, decreased venous return, decreased cardiac output, and decreased perfusion to the kidneys and intestinal mucosa. If IAH is not diagnosed early or if it is left untreated, the syndrome of end-organ damage and reduced oxygen delivery (ACS) will lead to multiple-organ failure, and ultimately death (Morris, Eddy, Blinnman et al., 1993; Diebel, Dulchavsky, Brown et al., 1997; Saggi, Sugerman, Ivatury & Bloomfield, 1998).

Kron, Harmon, and Nolan (1984) first recognized the importance of ACS in patients who had undergone major abdominal surgery. The syndrome consists of a combination of increased intra-abdominal pressure (IAP), oliguria, and decreased pulmonary compliance. Eventually patients with untreated ACS become hemodynamically unstable and subsequently die. There is sound evidence that IAH impairs renal blood flow, hepatic blood flow, cardiac function, pulmonary function, and

may lead to bowel ischemia (Ivy et al., 2000; Sugerman, Bloomfield, & Saggi, 1999). The diagnosis is made clinically in a patient with an IAP of > 20 mmHg, elevated peak inspiratory pressures (>45 cm H2O), oliguria (urine output < 0.5 ml/kg/hr), and a noted tight abdomen. Numerous reports have since documented the development of ACS during the resuscitation of critically ill surgical patients after abdominal surgery (Fietsam, Villalba, Glover et al., 1989; Cullen, Coyle, Teplick, et al., 1989; Meldrum, Moore, Francoise et al., 1997). Further research identified that ACS developed in both surgical and nonsurgical patients. Increases in retroperitoneal volume from pancreatitis, hemorrhage, or edema can lead to this syndrome. Increased intraperitoneal volume is the most common source of elevated intra-abdominal pressure. This includes hemorrhage, edema, bowel distention, mesenteric venous obstruction, abdominal packs, tense ascites, and peritonitis (Saggi et al., 1998; Cheatham, 1999). Burch, Moore, and Moore (1996) suggested that the triad of hypothermia, coagulopathy, and refractory metabolic acidosis leads to the development of ACS. This case is seen in traumatically injured patients who commonly develop a shock-related capillary leak which promotes ACS. Numerous studies have attempted to quantitate an occurrence rate for ACS. Researchers identified ACS in 15% of patients with severe abdominal trauma and an Injury Severity Score (ISS) of >15 (Morris, Eddy, & Blinman, 1993; Meldrum et al., 1997).

Kron et al. (1984) first described the technique of using bladder pressure as a means of assessing intra-abdominal pressure. The urinary bladder is an extraperitoneal, intra-abdominal structure with a compliant wall. Changes in intraperitoneal pressure are therefore reflected by a parallel change in intraluminal bladder pressures. The technique involves placing a Foley catheter in the urinary bladder, which is then filled with 50 ml of

sterile saline. The draining tubing is clamped just beyond the aspiration port, and a 19-guage needle is connected to polyethylene tubing and inserted into the port. The tubing is then attached to a pressure transducer using the symphysis pubis as the "zero" reference point. This technique has been validated in animal and human studies showing a high degree of correlation with directly measured intra-abdominal pressure (IAP), ($r = .85$-$.98$; $p < .0001$) over a wide range of IAPs up to 70 mmHg (Kron et al., 1984; Lacey, Bruce & Brooks, 1987; Iberti et al., 1987; Ridings et al., 1995). Given this high degree of correlation at wide ranges of IAP, the ease of use, and the minimal invasiveness of this technique, it is considered the "gold standard" for indirect clinical measurement of IAP.

Mutoh, Lamm, and Slotman (1992) studied the effects of volume infusion on intra-abdominal pressure. They concluded that massive volume resuscitation defined as receiving greater than ten litres of fluid for any reason (burns, severe pancreatitis, hemorrhagic shock) can lead to increased intra-abdominal pressure, particularly in the post-operative period or in a patient with signs of sepsis. A theoretical explanation of this process is that abdominal compartment syndrome (ACS) may result from the effects of "capillary leak", shock with ischemia-reperfusion injury, and the release of vasoactive substances and oxygen-derived free radicals, all combined with massive increases in total extra cellular volume. These increase retroperitoneal and intraperitoneal visceral and vascular volume, leading to elevated IAP (Mutoh et al., 1992).

The circulatory effects of increased IAP, combined with extra cellular hypervolemia from volume resuscitation, may cause abdominal wall edema and ischemia, reducing abdominal wall compliance and further accentuating the IAP increases leading

to ACS (Mutoh et al., 1992). The key to prevention of the multiple organ failure that occurs in ACS is early detection, including IAP monitoring.

Human Response to Illness Framework

Physiologic Regulatory Responses

Physiologic regulatory responses are based on the theory of normative biologic functioning and include phenomena measured by the instruments of the biological sciences (Mitchell, Gallucci, & Fought, 1991). The microcirculation is the system of arterioles, capillaries, and venules responsible for regulation of material exchange between the plasma and interstitial fluid surrounding tissues in the body (Sherwood, 1993). Transcapillary exchange between the interstitial fluid and capillaries occurs via three processes: Diffusion, filtration, and vesicular transport. Simple passive diffusion is the major process by which nutrients and metabolites cross the capillary barrier. Small lipid soluble molecules diffuse easily across the entire capillary membrane, whereas water, simple sugars and ions diffuse easily through the capillary pores (Sherwood, 1993). Larger lipophobic molecules such as sucrose, polysaccharides, and proteins have difficulty diffusing across the capillary membrane or pores and therefore, diffusion is rate-limiting in their transport into or out of capillaries called diffusion-limited transport (Sherwood, 1993). Diffusion of a substance across the capillary is proportional to its concentration difference within and outside the vessel and inversely proportional to the distance over which diffusion must occur (Sherwood, 1993).

Filtration and Reabsorption

The capillary wall acts like a sieve, with the fluid moving through its water-filled pores. When the pressure inside the capillary exceeds pressure on the outside, fluid is pushed out through the pores in a process known as ultrafiltration. The majority of plasma proteins are retained inside the vessels during this process because of the pores filtering effect, although a few do escape. The filtrate is essentially protein free plasma (Sherwood, 1993). When inward driving pressure exceed outward pressures across the capillary wall, net inward movement of fluid from the interstitial fluid compartment into capillaries takes place through the pores, a process known as reabsorption. Fluid movement occurs because of differences in the hydrostatic and colloid osmotic pressures between the plasma and interstitial fluid. Four forces influence fluid movement across the capillary wall (Sherwood, 1993). The first is capillary blood pressure (Pc) which is the fluid or hydrostatic pressure exerted on the inside of the capillary walls by the blood. This pressure tends to force fluid out of the capillaries into the interstitial. Normally, the hydrostatic pressure is around 37mmHg at the arteriolar end of a tissue capillary and declines to 17mmHg at the venous end (Sherwood, 1993). The second force is the plasma-colloid osmotic pressure (pp), also known as oncotic pressure, is a force caused by the colloidal dispersion of plasma proteins; it encourages fluid movement into the capillaries. Since the plasma proteins remain in the plasma rather than entering interstitial fluid, a protein concentration difference exists between the plasma and interstitial fluid (Sherwood, 1993). The plasma has a higher protein concentration and a lower water concentration than does the interstitial fluid. This difference exerts an osmotic effect that tends to move water from the area of higher water concentration in the interstitial fluid to

the area of lower water concentration (or higher protein concentration) in the plasma (Bell, 1998). Therefore, the plasma proteins may be thought of as attracting water. In normal physiological conditions, the only force limiting the movement of fluid out of the capillary is the osmotic effect of plasma proteins, which exert a water retaining force equivalent to 25 mmHg. Without plasma proteins (primarily albumin), water loss from the capillaries would be enormous. The plasma colloid osmotic pressure averages 25mmHg (Bell, 1998).

The third force is the interstitial-fluid hydrostatic pressure (Pif). This is the fluid pressure exerted on the outside of the capillary wall by the interstitial fluid. This pressure forces fluid into the capillaries. This pressure is at atmospheric pressure (around 1mmHg) (Sherwood, 1993). The final force involves the interstitial –fluid colloid osmotic pressure (pif). This pressure does not normally contribute significantly to flow. The small fraction of plasma proteins that leak across the capillary walls into the interstitial spaces are normally returned to the blood by means of the lymphatic system (Sherwood, 1993). Therefore the protein concentration in the interstitial fluid is extremely low, and in the interstitial fluid-colloid osmotic pressure is close to zero. The two pressures that tend to force fluid out of the capillary are capillary blood pressure and interstitial-fluid-colloid osmotic pressure. The opposing pressures that tend to force fluid into the capillary are plasma-colloid osmotic pressure and interstitial-fluid hydrostatic pressure. Ultrafiltration and reabsorption play an important role in regulating the distribution of ECF between the plasma and interstitial fluid (Sherwood, 1993).

Lymphatic System

Because fluid is lost via filtration at the capillaries, a mechanism must exist to return this fluid to the vascular compartment to prevent circulatory collapse. Lymphatic vessels serve this function. The lymphatic vessels are closed-end capillaries that have the primary function of returning protein, water, and electrolytes from the interstitium to the blood (Bell, 1998). Lymph vessels coalesce into larger vessels that contain one-way valves directed toward the heart. The lymphatic system terminates in the thoracic duct, which empties into the vena cava. Interspersed among lymphatics are a series of lymph nodes containing immune system cells (Bell, 1998). Ten percent of the fluid which is filtered is not reabsorbed and enters the lymphatic capillaries. Increase molecular weight proteins cannot enter the venous capillaries but can enter lymph capillaries because of their structure. Endothelial cells are attached by anchoring filaments to connective tissue. The edges of the endothelial cells overlap to form minute valves, fluid can flow in but not out (Bell, 1998). Total lymphatic flow is about 120 ml/hr. Anything that increases filtration of water out of the capillaries will increase lymph flow. Factors which influence lymph flow include: increased capillary pressure, decreased plasma oncotic pressure, increased interstitial fluid proteins, and increased capillary permeability (Bell, 1998).

Pathophysiologic Responses

An increasing body of literature has identified the significant disordered physiologic function that can occur with increased IAP. The effects of IAH are not limited to the intra-abdominal organs, but rather they have an impact either directly or indirectly on every organ system in the body.

Pathogenesis of Edema

Edema is a condition in which there is excessive accumulation of fluid in tissue spaces. Edema interferes with capillary transport and can cause circulatory collapse if edematous fluid forms from a loss of plasma volume. Anything that causes excess fluid filtration at the capillaries or impairs fluid transport through the lymph channels will create edema (Bell, 1998). For example, Burn patients have destroyed their capillary integrity, causing edema through increased capillary permeability, loss of albumin from damaged vessels, and inflammatory vasodilation. Thermal injury disrupts normal homeostasis and creates major physiologic alterations including massive intravascular fluid outflow that results in rapid occurrence of severe edema. Multiple inflammatory mediators are released, causing electrolyte imbalances (Ahrns & Harkins, 1999). Severe hemodynamic changes and tissue hypoperfusion occur. The occurrence of edema in uninjured tissue is a normal process, a beneficial mechanism that removes bacteria and debris through the lymph system, however, fluid shifts after a major burn injury exceeds those in any other form of trauma (Ahrns & Harkins, 1999). Edema occurs immediately after a burn injury and continues for 24 hours. The rapid onset of edema may be explained by increased capillary pressure and decreased interstitial hydrostatic pressure (Ahrns & Harkins, 1999).

Early Cellular Response

In burn injury, multiple events occur at the cellular level. Increased capillary permeability occurs due to the release of chemical mediators including histamine, bradykinin, and oxygen free radicals, which all cause an increase in the permeability of

blood vessels (Ahrns, & Harkins, 1999). Capillary membrane interstices expand, making it feasible for large protein molecules to leave the vascular space (albumin is the largest contributor to oncotic pressure). Interstitial colloid oncotic pressure contributes to sustained capillary permeability and progressive edema (Ahrns & Harkins, 1999).

Mediators, Cellular Response, and Response to Excess Volume Resuscitation

In burns compromising more than 40% total body surface area (TBSA), release of inflammatory mediators from heat-damaged tissue causes systemic permeability changes. Mediators influence the cardiovascular response to thermal injury, and play an important role in inducing edema. Histamine is released from mast cells when burn injury occurs. Thromboxane A2 and B2 are produced by platelets at the site of the injury (Ahrns & Harkins, 1999). This acts as a vasoconstrictor that impairs oxygen delivery to the tissue, an effect that is linked to the conversion of wounds from partial thickness to full thickness. Burn induced shock causes depolarization of myocardial cell membranes and decreases cardiac contractility (Ahrns & Harkins, 1999). Other mediators of edema include angiotensin II and ADH (Vasopressin), both of which are present at high levels after burn injury and cause vasoconstriction. After thermal injury, occurrence of edema is immediate and rapid. Maximum edema occurs within 8 to 24 hours after injury and persists at high levels for 48 to 72 hours (Ahrns & Harkins, 1999). The extent of edema is dependent on the severity of the burn injury and the effectiveness of resuscitation.

Burn, trauma, and abdominal aortic aneurysm repair patients are at risk for developing ACS during fluid resuscitation. Ivy et al. (2000) found that the volume of

fluid infused is related to the development of ACS, as well as the magnitude of the capillary leak, and the colloid oncotic pressure. Burn patients may receive up to 30 litres of fluid in the first 24 hours, and trauma patients may receive 10-12 litres during initial resuscitation. Massive volume resuscitation (greater than ten litres of fluid) leads to increased intra-abdominal pressure. These results from the effects of capillary leak, and release of vasoactive substances and oxygen derived free radicals, all combined with massive increases in total extracellular volume (Mutoh, Lamm, & Slotman, 1992). These increase retroperitoneal and intraperitoneal visceral and vascular volume, leading to elevated IAP (Mutoh et al., 1992). The circulatory effects of increased IAP combined with extracellular hypervolemia from massive volume resuscitation, may lead to abdominal wall edema and ischemia, reducing abdominal wall compliance. This may lead to multiple system organ failure (Mutoh et al., 1992).

In trauma and gastrointestinal surgical patients, increased intra-abdominal pressure occurs in clinical situations such as mechanical obstruction or ileus, retroperitoneal edema, and hemoperitoneum from bleeding. Serious injury, coagulopathy, and postoperative hemorrhage also cause ACS (Mutoh et al., 1992). Factors contributing to this phenomenon include bowel edema and congestion from excessive fluid resuscitation or injury of mesenteric vessels, accumulation of blood and blood clots, abdominal packing after damage-control surgery, and closure of the swollen and noncompliant abdominal wall under tension (Sanchez, Tenofsky, Dort, Shen, Helmer, & Smith, 2001). In trauma patients, bowel edema is most likely caused by metabolic failure, capillary leak related to release of vasoactive substances, extensive handling of bowel, and massive fluid resuscitation (Lozen, 1999).

In patients with ruptured abdominal aortic aneurysms (rAAA), massive fluid resuscitation produces substantial visceral and abdominal wall edema, rendering abdominal closure very difficult. In severe cases, massive edema prevented primary fascial closure (Oelschlager, Boyle, Johansen, & Meissner, 1997). The authors concluded that as in massively fluid resuscitated trauma victims, delayed laparotomy closure in rAAA patients may confer a physiologic and survival benefit.

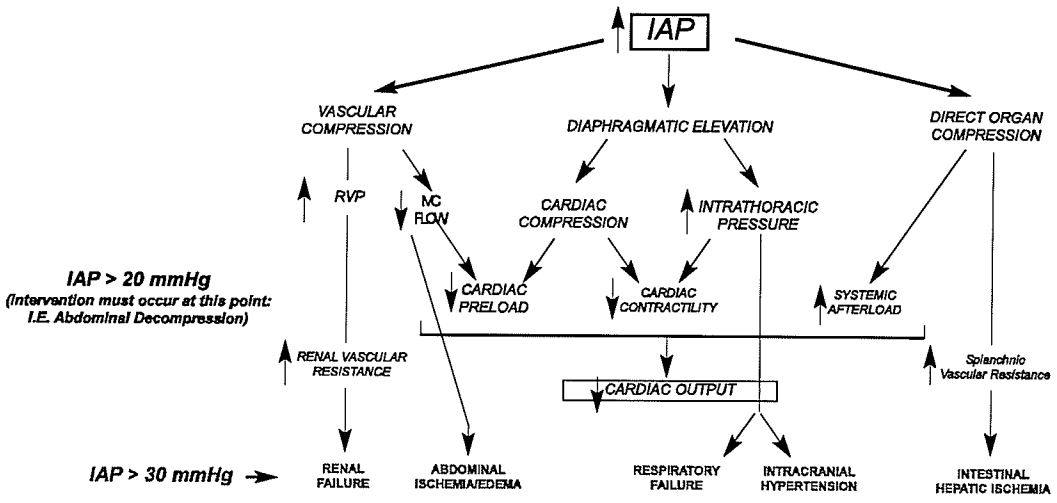
Pathophysiology of ACS

Abdominal compartment syndrome affects multiple organ systems in a graded fashion. Table 1 and Figure 2 summarizes the pathophysiology of IAH leading to ACS in each of the major organ systems. When IAP measurements exceed 30 mm Hg, circulatory collapse occurs, ending in multiple organ failure if surgical abdominal decompression is not performed (Cheatham, 1999).

Table 1 - The Graded Response to Acute Increases in Intra-abdominal Pressure

	IAP		
	10-15 mmHg (Stability)	**16-30mmHg (Instability)**	**>30 mmHg (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 increased cardiac output decreased
Renal	No effect or slight reduction in urine output that is reversible	Oliguria, azotemia	Anuria, worsening azotemia renal failure
Splanchnic	Low-grade intestinal ischemia	Increased intestinal ischemia	Bowel infarction, hepatic insufficiency, bacterial translocation?
Treatment	Maintenance of adequate tissue perfusion	Possible abdominal intravascular volume	Mandatory decompression surgery

(Adapted from Saggi et al., 1998).



RVP, renal vein pressure; IVC, inferior vena cava (Adapted from Saggi et al., 1998)

Figure 2. Pathophysiology of Abdominal Compartment Syndrome

Figure 3 depicts the interactions between organ systems that lead to or exacerbate ACS leading to multiple organ failure. Direct vascular and organ compression and diaphragmatic elevation are the key mechanisms leading to the circulatory collapse that is at the center of the pathophysiology of abdominal compartment syndrome.

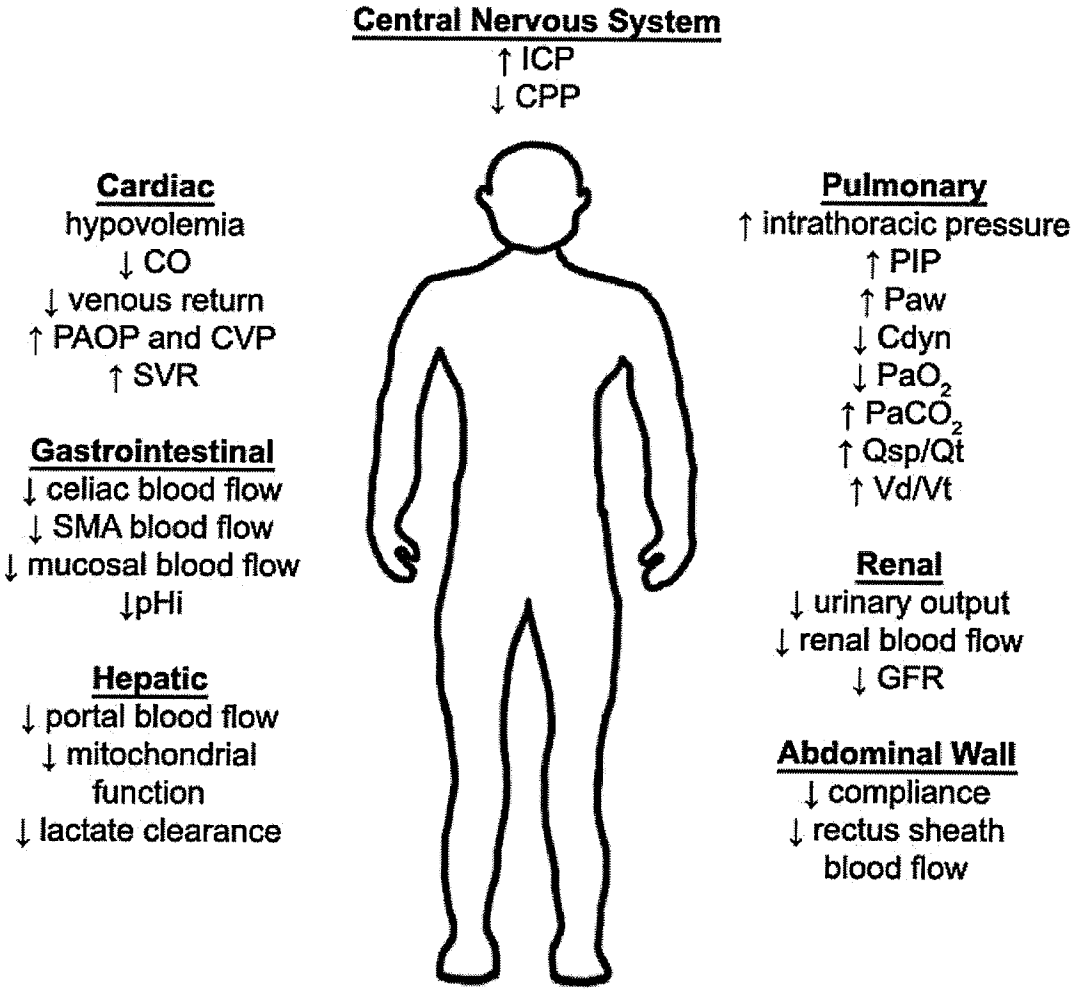


Figure 3. Physiologic Effects of Increased Intra-abdominal Pressure. ICP, intracranial pressure; CPP, cerebral perfusion pressure; CO, cardiac output; PAOP, pulmonary artery occlusion pressure; CVP, central venous pressure; SVR, systemic vascular resistance; PIP, peak inspiratory pressure; Paw, mean airway pressure; Cdyn, dynamic pulmonary compliance; Qsp/Qt, intrapulmonary shunt; Vd/Vt, deadspace ventilation; SMA, superior mesenteric artery; pHi, intramucosal pH; GFR, glomerular filtration ratio.

(Cheatham, 1999, p.99)

Cardiovascular

Elevated IAP leads to a decreased cardiac output. This effect is most commonly seen at an IAP greater than 20 mm Hg. The diminished cardiac output results from decreased inferior vena cava and portal vein pressure as well as from an increased thoracic pressure, which result in decreased inferior and superior vena caval flow. The increased thoracic pressure also leads to cardiac compression with decreased ventricular end-diastolic volumes. Increased systemic afterload is also seen with IAH. All of these lead to a reduced stroke volume with an increased heart rate (Saggi et al., 1998).

Increased thoracic pressure and diaphragmatic elevation are responsible for reductions in ventricular compliance. This, combined with increased systemic afterload, reduces cardiac contractility at IAP greater than 30 mm Hg, will shift the Starling curve to the right and downward (Saggi et al., 1998). IAH also decreases venous return from the lower extremities functionally obstructing inferior vena cava blood flow by two mechanisms. First inferior vena cava pressure increases dramatically in the presence of IAH and has been demonstrated to parallel changes in IAP (Kashton, Green, & Parson, 1981). Second, cephalid deviation of the diaphragm causes a mechanical narrowing of the vena cava at the diaphragmatic crura, further reducing venous return to the heart. Femoral vein pressures are increased and venous blood flow and pulsatility are dramatically reduced. The resulting increases in extremity venous hydrostatic pressure promote the formation of peripheral edema (Kashton et al., 1981).

Respiratory System

With an acute elevation in IAP, respiratory failure characterized by high ventilatory pressures, hypoxia, and hypercarbia eventually develop (Cheatham, 1999). Diaphragmatic elevation leads to a decrease in pulmonary compliance. The increase in IAP also reduces total lung capacity, functional residual capacity, and residual volume. This results in ventilation – perfusion mis-match and hypoventilation, producing hypoxia and hypercarbia, respectively. Pulmonary vascular resistance increases from the collective effects of reduced alveolar oxygen tension and increased thoracic pressure (Saggi et al., 1998). Abdominal decompression improves the acute respiratory failure almost immediately (Saggi et al., 1998).

Abdominal Visceral Abnormalities

Mesenteric arterial, hepatic arterial, intestinal mucosal, hepatic microcirculatory, and portal venous blood flow all have been shown to be diminished with IAH. An IAP of greater than 20 mm Hg impairs intestinal perfusion at the mucosal and submucosal levels, resulting in a reduction of tissue oxygen tension, anaerobic cell metabolism, acidosis, and free radical generation (Cheatham, 1999).

Intestinal ischemia and infarction has been described during prolonged laparoscopy despite apparently normal hemodynamics and renal function. Perhaps more common is the low-grade ischemia seen at IAP of 15 mm Hg. Prolonged low-grade elevation of IAP is linked with bacterial translocation in rat and porcine models (Cheatham, 1999). Despite normal systemic hemodynamics, profound splanchnic ischemia can be ongoing with IAH. Few of the overt manifestations of ACS are evident

at this point to alert one to developing IAH (Cheatham, 1999). It has been suggested that such ischemia is associated with an increased incidence of multiple system organ failure, sepsis, and increased mortality. These are some of the strongest arguments for the use of routine measurement of IAP in critically ill patients.

Hepatic

The hepatic artery, hepatic vein, and portal vein blood flow are decreased by the presence of intra-abdominal hypertension. Hepatic artery flow is directly affected by decreases in cardiac output. Hepatic and portal venous flows are reduced as a result of both extrinsic compression of the liver as well as anatomic narrowing of the hepatic veins as they pass through the diaphragm (Cheatham, 1999). On a microscopic level, hepatic microcirculatory blood flow is diminished resulting in a reduction in hepatic mitochondrial function and production of energy substrates (Cheatham, 1999). The clearance of lactic acid by the liver appears to be compromised. Of noted importance is that these changes have been recognized and documented with IAP increases of only 10 mm Hg and in the presence of both normal cardiac output and mean arterial blood pressure (Cheatham, 1999; Diebel, Wilson, & Dulchavsky, 1992).

Abdominal wall Abnormalities

Increased IAP has been shown to decrease abdominal wall blood flow by the direct, compressive effects of IAH under conditions of stable systemic perfusion, leading to ischemia and edema (Cheatham, 1999). This can cause the abdominal wall compliance to decrease and exacerbate IAH. Abdominal wall muscle and fascial ischemia may

contribute to infections and noninfectious wound complications (i.e., dehiscence, herniation, necrotizing fascitis) (Cheatham, 1999).

Renal System

IAH-induced reductions in renal blood flow and function have been demonstrated in both animal and human models (Sugrue, Buist, Bauman, & Hillman, 1995; Sugrue et al., 1999; Doty, Saggi, Blocher, Fakhry, Gehr, Sica & Sugerman, 2000). Renal artery blood flow has been demonstrated to be diminished with signs of IAH. Blood is shunted away from the renal cortex and glomeruli, leading to the impairment of glomerular and tubular function (Cheatham, 1999).

Oliguria progressive to anuria, and pre-renal azotemia unresponsive to volume expansion, characterize the renal dysfunction of ACS. Oliguria can be seen at IAP of 15 to 20 mm Hg, whereas increases to 30 mm Hg or greater may cause anuria. Decompression and reduction of IAP, however, immediately reverses oliguria, usually inducing a vigorous diuresis of fluids administered during resuscitation (Saggi et al., 1998).

The mechanisms of renal derangements with IAH involve reduced absolute and proportional renal arterial flow, increased renal vascular resistance with decreased glomerular filtration, and increased tubular sodium and water retention (Cheatham, 1999). IAH causes an elevation in both blood urea nitrogen and serum creatinine and a reduction in urine creatinine clearance. Osmolar clearance is also decreased and fractional excretion of sodium is increased. The changes in renal and systemic hemodynamics cause an increase in circulating levels of antidiuretic hormone, rennin,

and aldosterone which further increase renal vascular resistance and produce sodium and water retention. Renin and aldosterone levels are partially diminished with volume expansion and further by abdominal decompression (Sugrue, Deane, Bishop, Bauman, & Hillman, 1999).

Intracranial Abnormalities

Elevated intracranial pressure (ICP) and decreased cerebral perfusion pressure have been described with acute changes in IAP in animal models. Elevated IAP directly elevates intrathoracic and central venous pressures with compromise to cerebral venous outflow. Reduction in IAP by surgical decompression reverses the derangement (Saggi et al., 1998).

Predictors of ACS, Diagnosis, and Management Strategies

Despite our knowledge of the adverse effects of elevated IAP since the early part of the century, clinical application of these concepts in the treatment and management of critically ill patients is a phenomenon of the last two decades. Richards, Scovill, & Shin (1982) first discussed the syndrome of renal failure associated with a "tense abdomen" in modern clinical surgery. Kron et al. (1984) however, were the first to correlate increases in IAP greater than 25 mm Hg with postoperative renal failure and a reduction in IAP with treatment by surgical decompression resulting in the return of normal renal function. Since these early reports, numerous researchers have documented the existence of a distinct clinical syndrome involving multiple organ systems and associated with

increased IAP, in which decompressive celiotomy improves outcome. The research findings suggest that ACS without expedient decompression is uniformly fatal. Surgical decompression, however, is 93% effective at reversing organ dysfunction and is associated with an overall survival of 59% (Saggi et al., 1998).

ACS developed in 21 of 145 patients (14%) who sustained severe abdominal trauma (Meldrum et al., 1997). In this prospective study, 60% of ACS patients suffered blunt trauma. The ACS was defined as the presence of an IAP greater than 20 mm Hg with cardiovascular, pulmonary, or renal impairment (Meldrum et al., 1997). Ivatury & Simon (1997) found the incidence of IAH after penetrating abdominal trauma to be greater in patients undergoing primary fascial closure than in patients receiving prophylactic mesh closure. Fiestam, Billalba, & Glover (1987) reported a 4% incidence of ACS with primary closure after repair of ruptured aortic aneurysms.

Maxwell, Fabian, Croce et al. (1999) studied the effects of fluid resuscitation on intra-abdominal pressure in patients with no abdominal injury and suggested that ACS can occur with no abdominal injury. On the basis of their observations, they recommended that IAP measurements should be routinely checked and acted on appropriately when resuscitation volumes approach ten litres of fluid (crystalloid or colloid fluid).

Despite the vast amount of literature on abdominal compartment syndrome relating to the areas of trauma, vascular and general surgery patients, its occurrence in burn patients has not been widely reported. Greenhalgh and Warden (1994) first reported the occurrence of ACS in their pediatric burn population. They found that the incidence of ACS increased with burn size and depth. Ivy et al., (2000) conducted a prospective

study on ten burn patients to determine the occurrence of ACS in adult burn patients. They concluded that ACS was seen in patients with more than 70 % burns and intra-abdominal hypertension occurred commonly in major burn patients. The patients that developed ACS received more than 25 liters of fluid and had a mean IAP of 40 mmHg. Their findings supported Greenhalgh et al.'s (1994) study; however, both studies consisted of small sample sizes. Greenhalgh and Warden (1994) studied three children who developed ACS with intra-abdominal pressures in excess of 30 mmHg during initial burn resuscitation and two children who developed transient intra-abdominal pressure elevation. They found that the incidence of ACS increased with burn depth and size. Their study provided evidence that burn patients are at risk for the development of ACS. Ivy, Nabil, Atweh et al. (1999) recently reported four case studies of adult burn patients with ACS manifested by oliguria, decreased pulmonary compliance, intra-abdominal hypertension, and hemodynamic instability during initial burn resuscitation. All died due to complications resulting from ACS. IAP measurements were not performed on these patients, and therefore, ACS was diagnosed during the end-stage of multi-organ system failure. Based on their findings from the case studies, Ivy, Nabil, Atweh et al. (2000) concluded that ACS is a common problem in major burn patients. The researchers reported that routine monitoring of intra-abdominal pressure led to the early diagnosis and treatment of ACS patients.

In 1876, Wendt suggested an association between renal function and abdominal pressure when he reported a correlation between IAP and urine output. Increased intra-abdominal pressure (IAP) is associated with oliguria and anuria. It has been shown that the kidney is one of the most sensitive organs to the ischemic insults produced by IAP

(Altintas, 2001). Acute renal failure in critically ill patients has a multifactorial aetiology. There were no investigations that reported the level of raised IAP beyond which ARF develops. Creatinine Clearance is an effective and reliable method for monitoring renal function. BUN and serum creatinine levels may be inaccurate as a result of medications such as loop diuretics, HTN, sepsis, hypotension and age > 60 yrs. If ACS develops, only prompt relief of the elevated intra-abdominal pressure is associated with the immediate return of renal function.

Two recent prospective studies analyzed the use of routine IAP measurement in preceding ACS-related renal dysfunction. In a study involving 42 patients undergoing abdominal aortic surgery, Platell, Hall, & Clark (1990) found that an IAP greater than 18 mm Hg had positive and negative predictive values 85 and 62%, respectively for the development of oliguria. Sugrue et al., (1995) evaluated 100 patients admitted to the ICU after a laparotomy and found a 33% incidence of renal impairment. Furthermore, 69% of the patients with renal impairment had IAH. These studies, combined with the improved results in the management of ACS with the prospective use of IAP strongly argue for the routine measurement of IAP in select ICU patients at risk for ACS.

Serial, routine measurements of bladder pressure in intensive care unit patients should function to identify IAPs that are rising and prepare the surgeon for the impeding possibility of required decompression. Therefore with the diligent use of routine measurements, the severely elevated IAPs should not be encountered (Fusco, Martin, & Chang, 2000; Meldrum, Moore, Moore, Francoise, & Burch, 1997). It provides valuable confirmatory information.

Cheatham et al. (2000) found that abdominal perfusion pressure (APP) was a clinically useful predictor of patient survival during treatment of IAH and ACS. They suggested that an APP of 50mmHg or greater seems to be a positive indicator of patient survival.

The primary objective of this study was to determine if APP was a clinically useful predictor of patient survival or the development of ACS for those patients diagnosed with intra-abdominal hypertension using the cut-off point of an APP $\geq$ 50 mmHg. Cheatham et al., (2000) found that APP was statistically superior to IAP monitoring alone in predicting patient survival from intra-abdominal hypertension and ACS. The simple calculation of APP (MAP-IAP) addresses not only the severity of IAH, but also the adequacy of tissue perfusion and the need for additional resuscitation. APP was found to be the best predictor of patient outcome when compared to MAP, IAP, and hourly urine output alone. The inability to maintain an APP of at least 50mm Hg or greater seems to be an indication for the need of a decompressive laparotomy and maintenance of an open abdomen until the patient improves (Cheatham et al., 2000).

A femoral vein catheter can be used to measure pressure within the inferior vena cava. This correlates well with IAP measured directly and urinary bladder pressure in various animal models. However, no human studies have validated its use (Saggi, Sugerman et al., 1998).

Behavioral Perspective of ACS

Behavioral responses are directly observable and measureable motor and verbal behaviours (Mitchell, Gallucci, & Fought, 1991). Behavioral signs of abdominal

compartment syndrome include abdominal distention, increased intra-abdominal pressure (IAP>15mmHg), oliguria refractory to volume administration, hypoxia with increasing airway pressures, hypercarbia, tense or massively distended abdomen, metabolic acidosis, and severe abdominal pain (Schulman, 2000). The patient may experience anxiety and fear due to the severe pain. The patient experiences difficulty in breathing due to the increase in pressure on the diaphragm. Patients diagnosed with ACS may develop ICU psychosis related to sensory overload. Sleep patterns may become disrupted, and patients may become disoriented to time, place, or persons (Wilson, 1993).

Experiential Responses

Experiential responses include concepts on introspection, personal experience, and the derivation of shared meaning. They are measured by self report (Mitchell et al., 1991). The patient's past experiences with previous illnesses will impact on their coping mechanisms. The realization of their life-threatening injury is frightening. The family also experiences fear and stress as a result of the devastating injuries to their loved one. Family members may become overwhelmed by the ICU environment including ventilators, alarms, and numerous drug infusion pumps. Family members respond differently depending on past experiences (Wilson, 1993).

Person Factors

Quality of life is a mixture of objective circumstances (employment, income, and social support) and behaviors (self-care, ambulation, social interaction), which affect and determine the satisfaction of needs. Other factors include sex, age, and pre-existing morbidity. Konigova (1992) stressed the importance of socioemotional support of family

members (wives, husbands, brothers, sisters). They concluded that the next of kin either stimulate the endogenous disposition in patients with negative cognitions, or make possible to lend purpose of the defence mechanisms of the denial type (Konigova, 1992). Person factors such as culture and prior experience with illness can influence the experiential response in terms of the attribution and the meaning of their injury (Mitchell et al., 1991).

Modifiable factors include enhancing coping mechanisms, providing education to patients and families, providing emotional support, providing continuity of care and allowing patients to have some control with treatments. Non-modifiable factors include age, and pre-existing morbidity.

Environmental Factors

Environmental factors contribute to the patient's ability to cope with their injury. Patients in the intensive care environment experience sleep deprivation due to extensive and continuous monitoring. Harsh lighting, immobilization, painful procedures, and most importantly, interrupted sleep is common experiences in the ICU setting (Wilson, 1993). Preoperative and postoperative immobility have been identified as factors in patients who developed sensory perceptual alteration. Patients developed distorted perceptions in ICUs such as feelings of "being held prisoner and their hands were weighted down." Researchers theorized that these perceptions were derived from the busy, monotonous nature of the ICU in which patients were exposed to unfamiliar hospital personnel, being confined to bed, and possibly restrained (Wilson, 1993). Patients also experience limited

contact with family members. All of these experiences combined represent barriers to accurate sensory perception (Wilson, 1993).

Summary of Chapter

This chapter focuses on a review of the literature on ACS. The Human Response to Illness Model directs the review process including the physiologic regulatory responses, pathophysiologic responses of ACS, as well as the behavioural and experiential perspectives related to ACS. The underlying normative biologic functioning of the microcirculation is described. In the pathophysiological, behavioural, and experiential sections, the literature review emphasizes the devastating effects that ACS has on the different organ systems and the patient's ability to cope in the ICU environment.

Specifically in the pathophysiology section, the literature review emphasizes that elevated intra-abdominal pressures resulted in numerous organ system alterations requiring rapid diagnosis and aggressive therapy. Evidence that bladder pressure estimation of IAP is a non-invasive, easy, and highly reproducible method of assessing the presence of IAH and ensuing ACS is discussed. The indirect method of measuring IAP or "bladder pressure" which has become the "gold standard" for measurement of IAP is described. The benefits of monitoring abdominal perfusion pressure are discussed. Predictors of ACS, diagnosis, and management strategies are identified. Of significance is that the routine monitoring of IAP is not the usual practice in intensive care units.

Chapter III

Methodology

Introduction

This chapter discusses the methods and procedures central to undertaking the study of the incidence, main physiologic effects, and therapeutic management of patients with intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). The correlation between intra-abdominal pressure readings and inferior vena cava pressures via a femoral venous catheter was also analyzed. Consideration is given to the research design, recruitment strategies, instrumentation and analysis.

Design of the Study

Using a quasi-experimental design, a pilot study of patients at risk of developing ACS was conducted at the Health Sciences Centre. A pilot study is defined as a small scale version, or trial run, for a major study (Polit & Hungler, 1999). A prospective study begins an examination of presumed causes and goes forward in time to observe presumed effects (Polit & Hungler, 1999). A quasi-experimental design defined as a study in which subjects are not randomly assigned to treatment conditions, but the researcher manipulates the independent variable. Although the subjects were not randomly assigned to the intervention, the researcher did manipulate the intra-abdominal pressure monitoring by pre-determining the time of each measurement (every six-eight hours). This manipulation enhances control of internal validity. Internal validity is the degree to

which it can be inferred that the experimental treatment, rather than extraneous factors, is responsible for observed effects (Polit & Hungler, 1999).

The correlation between IAP monitoring and inferior vena cava pressures via the femoral venous catheter, and the relationship between a positive fluid balance and increased IAP were studied. This research explored the interrelationships among variables of interest without any active intervention on the part of the researcher.

This prospective study was undertaken over a period of one year in the Surgical ICU at the Health Sciences Center commencing in July 2002. The study evaluated the effectiveness of routine IAP monitoring via bladder pressures over time for the early detection of IAH. All patients who met the inclusion criteria and agreed to participate had routine IAP measurements done upon admission to the unit, and every six-eight hours thereafter in order to assess the patient's level of intra-abdominal hypertension throughout their stay in the Surgical ICU. IAP's were measured until the patient was stabilized and no longer showed symptoms of IAH. In order to ensure accuracy, the standard hospital policy and nursing protocol for IAP measurement was followed.

The Setting

The study was conducted on the surgical intensive care unit at a tertiary care teaching hospital in Winnipeg, Manitoba. Patients at risk for abdominal compartment syndrome are critically ill, therefore the surgical ICU was chosen for this study.

The Sample

The populations of interest were individuals with the following diagnoses: abdominal trauma, laparotomies, necrotizing pancreatitis, patients requiring massive fluid resuscitation including burns, and ruptured abdominal aortic aneurysms. A convenience, purposive sample of 20 subjects were asked to participate in the study. This number was based on the expected availability of patients per year, and the expected length of time that the researcher had to complete the study.

Inclusion Criteria for Study Subjects:

Adult ICU surgical patients 18 years of age or older, who were at high risk for developing ACS. Patients with the following diagnosis were considered for this study:

- burns
- necrotizing pancreatitis
- blunt abdominal trauma
- fluid resuscitation > 10L during the first 96 hours of ICU admission
- hepatic dysfunction
- sepsis
- AAA repair
- G.I. surgery or G.I. problems

Exclusion Criteria

- patients with bladder abnormalities or a neurogenic bladder
- patients with pelvic fractures or pelvic hematomas

Specific inclusion and exclusion criteria for each research question:

Research Question 1: Is the routine monitoring of APP (abdominal perfusion pressure) every 6-8 hours a clinically useful predictor of patient survival or the development of ACS during the first 96 hours following ICU admission ?

Inclusion Criteria:

Patients demonstrating one or more signs of Intra-abdominal Hypertension (IAH) within the first 96 hours of ICU admission:

- abdominal distention
- oliguria refractory to volume administration
- hypercarbia
- hypoxemia refractory to increasing inspired oxygen fractions
- elevated peak inspiratory pressures

  and

- IAP of 15 or greater

Exclusion Criteria:

- IAP of less than 15
- patients with bladder abnormalities or a neurogenic bladder
- patients with pelvic fractures or pelvic hematomas

Research Question 2: Is there is a relationship between a positive fluid balance and an increase in intra-abdominal pressures during the first 96 hours following ICU admission?

Inclusion Criteria:

Patients demonstrating one or more signs of Intra-abdominal Hypertension (IAH) within the first 96 hours of ICU admission:

- abdominal distention
- oliguria refractory to volume administration
- hypercarbia
- hypoxemia refractory to increasing inspired oxygen fractions
- elevated peak inspiratory pressures

  and

- volume administration during the first 96 hours of ICU admission

Exclusion Criteria:

- patients with bladder abnormalities or a neurogenic bladder
- patients with pelvic fractures or pelvic hematomas

Research Question 3: Is there is a correlation between IAP and inferior vena cava pressure measured from the femoral venous catheter?

Inclusion Criteria:

- patients who currently have a femoral venous catheter (prior to the commencement of the study)

Exclusion Criteria:

- patients with bladder abnormalities or a neurogenic bladder
- patients with pelvic fractures or pelvic hematomas

Recruitment of Participants

Permission to conduct the research project at the hospital was obtained and ethical approval from the Ethical Review Committee of the Faculty of Nursing was granted (see Appendix A).

The researcher arranged a meeting with the unit manager to describe the study and subsequently a presentation to nurses at a staff meeting to discuss the project was undertaken. Nurses working in the surgical ICU either full-time or par-time varied in clinical experience. Although the nurses who cared for the patients were required to monitor the intra-abdominal pressures as ordered by the physician, this activity was only performed infrequently. Therefore, to ensure measurement accuracy, the nurses were provided with an inservice on the technique of measuring intra-abdominal pressures as well as the pathophysiology of abdominal compartment syndrome. The policy and procedure for HSC on this measurement were followed (see Appendix B). Because the researcher set up all required equipment for the bedside nurse, the measurement took approximately one minute to perform.

On admission, the Clinical Resource Nurse (CRN), the daily charge nurse for the surgical ICU and not a primary care giver to any potential subject, ascertained if patients who met the inclusion criteria were interested in learning more about the study from the researcher. The CRN decided if the patient was too critically ill to make decisions regarding participation, and approached the authorized third party if appropriate. The researcher explained the study to interested patients or authorized third parties and obtained written consent (Appendix C).

Data Collection

A data base was developed using Microsoft Excel software program. Data collected included demographic information (age, gender, past medical history), the admission date, OR date and procedure, surgical history, and Apache II scores (Acute Physiology and Chronic Health Evaluation II tool used for mortality prediction in ICU). Results from any abdominal CT scans, absence or presence of diagnosis of intra-abdominal hypertension were also included. The intra-abdominal pressures (IAP) were recorded every six – eight hours upon admission until the patient was stabilized or until the first 96 hours of ICU admission. Abdominal perfusion pressures (APP) were also calculated with IAP monitoring (mean arterial pressure – intra-abdominal pressure). Maximal APPs or the highest APP value for each patient was also recorded. Urine output was recorded as well as arterial PH, serum lactate levels, BUN and serum creatinine. Routine hemodynamic (heart rate, mean arterial pressure) and respiratory parameters were also simultaneously recorded (peak airway pressures, oxygen requirements, PaO2, PaCO2, tidal volumes, and ventilator settings). This data was part of the normal routine

care in the intensive care unit. For patients with a femoral venous catheter already in situ, readings from the femoral catheter as well as IAP measurements were also performed simultaneously and recorded every six-eight hours.

The bedside nurse implemented and measured the intra-abdominal pressures using the policy and procedure guidelines explained in Appendix B, and then recorded the numbers on the patient's flow sheet. The attending physician was notified immediately with an IAP reading of > 15 mm Hg.

Research Instrument and Procedures

Intra-abdominal Pressure Measurement

Kron et al. (1984) first described the technique of using bladder pressure as a means of assessing intra-abdominal pressure. The urinary bladder is an extraperitoneal, intra-abdominal structure with a very compliant wall. Changes in intraperitoneal pressure are therefore reflected by a parallel change in intraluminal bladder pressures. The technique involves placing a Foley catheter in the urinary bladder, which is then filled with 50 ml of sterile saline. The draining tubing is clamped just beyond the aspiration port, and a 19-guage needle is connected to polyethylene tubing and inserted into the port. The tubing is then attached to a pressure transducer using the symphysis pubis as the "zero" reference point. A copy of the policy and procedure for Health Sciences Center is provided in Appendix B.

Reliability and Validity

This technique has been validated in animal and human studies showing a high degree of correlation with directly measured intra-abdominal pressure (IAP), ($r = .85$-$.98$; $p < .0001$) over a wide range of IAPs up to 70 mmHg (Kron et al., 1984; Lacey, Bruce & Brooks, 1987; Iberti et al., 1987; Ridings et al., 1995). Given this high degree of correlation at wide ranges of IAP, the ease of use, and the minimal invasiveness of this technique, it is considered the "gold standard" for indirect clinical measurement of IAP.

Apache II (Acute Physiological and Chronic Health Evaluation)

Apache II was developed by Dr. William Knaus at George Washington University Medical Centre in 1978. This tool was designed to be an objective and quantitative measure of the severity of illness of acutely ill patients in intensive care units. The severity of disease classification system also assists in the ability to prognosticate outcomes or evaluate the impact of subsequent care in ICU (Health Sciences Center Guidelines, 1999).This tool has been validated in many centers and shown to be a strong and stable predictor of hospital survival (Beck, Smith, Pappachan, & Millar, 2003).

APACHE II consists of two components: The first is APS (Acute Physiology Score), there are twelve physiologic variables from one or more of the body's seven major physiologic systems: neurological, cardiovascular, respiratory, gastrointestinal, renal, metabolic & hematologic. The second component is Age Group and a history of underlying Chronic Health Condition. This reflects diminished physiologic reserve due to underlying disease conditions (Health Sciences Center Guidelines, 1999) (see Appendix D).

Reliability and Validity

Apache II has been validated in numerous studies (Arabi, Abbasi, Al-Abdulkareem, Kalayoglu, & Wood, 2002; Beck, Smith, Pappachan, & Millar, 2003). These authors concluded that Apache II predictions were calibrated adequately in the patients studied.

Inferior Vena Cava Pressure Measurement

Inferior vena cava pressures are recorded by connecting a transducer monitor to the patient's femoral venous catheter. This is read in the same way as performing bladder pressures. This method is the same principal as measuring central venous pressure from a central line (internal jugular or subclavian vein) (Cheatham, 1999).

Data Analysis

Data was entered and coded into Microsoft Excel and statistical analysis system (SAS). A t-test was used to determine the clinical relevance of APP of 50 or greater as a cut-off point for predicting survival or non-survival or ACS. Due to the fact that patients had a variable number of APPs taken (ranging from 2-23), an array of summary values were used to see if they were predictive of ACS or survival. The average of APPs for each patient, the median of APP values, the maximal or highest APP value, the total number of APPs>50, and lastly, the percentage of APPs>50 were analyzed. A Kruskal-Wallis Test was used to analyze the difference in abdominal perfusion pressures >50

mmHg between survivors and non survivors. Maximal APPs were also compared between survivors and non-survivors. A non-parametric test was used due to the assumption that results would not follow a normal distribution curve. For research question 2, the Spearman Correlation Coefficients was used to analyze the relationship between positive fluid balance and high IAP. Spearman Correlation Coefficient indicates the magnitude of a relationship between variables measured on an ordinal scale (Polit & Hungler, 1999). For the final question, the Pearson Correlation Coefficients was used to analyze the relationship between IAP and FVP. Pearson Correlation Coefficients designate the magnitude of a relationship between two variables measured on at least an interval scale (Polit & Hungler, 1999). Statistical significance was considered to be $p < .05$.

Ethical Considerations

In some cases, patients were critically ill and not able to give valid consent. According to the Tricouncil Policy Statement on Ethical Conduct for research involving humans, should the patient be unable to give consent, an authorized third party can give consent. This was done, so the participant maintained the right to participate, and the patient's best interest was protected. The authorized third party was a family member, or a significant other. Should the participant become competent at a later stage in the study, consent was sought from the participant, as a condition of continuing participation.

Anonymity and Confidentiality

The patients who were approached regarding participation were assured that they would receive the standard nursing care regardless of their choice to participate or not participate in this project. Patients and nurses who participated in this study were assured that the voluntary nature of their participation in the study meant they could withdraw from the study at any time.

Anonymity of patients was assured by identifying the patient in the study reports by number only. Only the researcher and thesis chair had access to the log that matched the participant number and patient name. The vital signs sheet was photocopied and names, hospital numbers, or any other identifying information removed. Demographic information was recorded using the coding system. A master list with patient names corresponding to assigned codes along with the signed consent forms were kept in a separate locked file. This material will be kept under lock and key in the researcher's home filing cabinet for seven to ten years and then destroyed and discarded as confidential waste.

Risks and Benefits

The element of risk for the participant was minimal. The only risk documented in the literature was the rare possibility of a urinary bladder infection if aseptic technique was not used when taking measurements. Every safeguard was undertaken by the researcher to ensure that aseptic technique was maintained by all nursing staff.

The participant may benefit from having routine IAP measurements by the early diagnosis and treatment of intra-abdominal hypertension. The nursing staff participants

will benefit by being a part of the research study and learning about abdominal compartment syndrome, a serious complication that many critically ill patients are at risk for developing. This study will increase the awareness of this syndrome to nurses, surgeons, and intensivists.

Limitations

A limitation to this study was the nurses' inability to maintain the 6-8 hour IAP and APP monitoring schedule during times when the acuity of the patient was too high, or the patient became hemodynamically unstable. Efforts were made to ensure that the 6-8 hour monitoring was maintained for the first 96 hours of ICU admission; however, patients did not have equal time frames for IAP and APP monitoring. This may have affected the findings due to the unequal number of measurements done for each patient.

A second limitation was the study design of a non-randomized convenience sample. This sample is less likely to produce accurate and representative samples. Despite this fact, most research samples in most disciplines, including nursing, are nonprobability samples (Polit & Hungler, 1999).

A small sample size was used resulting from the infrequency of high risk patients in the ICU setting. The larger the sample, the more representative of the population it is likely to be (Polit & Hungler, 1999). The larger the sample, the smaller the sampling error. Smaller samples tend to produce less accurate estimates than larger samples (Polit & Hungler, 1999).

Summary of Chapter

In this pilot study, a prospective quasi-experimental and correlation design is used to investigate the efficacy of routinely monitoring abdominal pressures for all surgical patients at risk for developing ACS. Included is an explanation of the methodology used to calculate abdominal perfusion pressure and determine correlations between femoral venous pressure and bladder pressure. The benefits of routine IAP monitoring are described. Limitations of the methodology section are discussed, and anticipated risks and benefits of the study are outlined.

The extensive training program developed by the researcher to educate and inform nurses volunteering to participate in the study is presented. Ethical issues and guidelines followed to protect patients and nurses participating in the study are outlined. Data collection methodology and data analysis techniques are described.

Chapter IV

Results

This chapter discusses the analysis and findings from three research questions posed in this study concerning the issue of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). A summary of findings including the statistical analyses performed and the relationship to demographic data are discussed. On the advice of the consultant statistician, multivariate analysis was not undertaken due to the small sample size.

Recruitment

In total, 18 patients participated in the study conducted from July 2002 until September 2003 at the Health Sciences Center Surgical ICU. Twenty-one potential subjects were approached, however, three patients refused to participate due to the fear of obtaining a potential bladder infection (one of the risks involved with IAP monitoring).

Demographics

Demographic information gathered included age, gender, past medical history, type of surgical procedure, and APACHE II scores.

The patients ranged in age between 30 and 81 years of age. Of the 18 patients, 7 were 50years of age or younger (39%), and 6 were 71-80 years of age (33%). The mean

age was 58 with a standard deviation of 15. Demographic characteristics are shown in Tables 2a, b.

Demographic Characteristics of the Sample N=18

Table 2a - Age

Age (years)	**Mean**	**Standard deviation**
	58	**15**
Age ranges	**Frequency**	**Percent**
30-40	3	17
41-50	4	22
51-60	3	17
61-70	2	11
71 or greater	6	33

Table 2b - Sex

	Frequency	**Percent**
Female	4	22
Male	14	78

The number of females and males in the study is depicted in Table 2b. There were 14 males and 4 females in the study. Table 3 shows the current medical co-morbidities in descending order of frequency of the illnesses. Of the 18 patients, the majority (61%) had a history of hypertension, followed by 17% with a G.I. history.

Table 3 - Current Medical Morbidities in Descending Order of Frequency

Medical History	Frequency	Percent
HTN	11	61
Peripheral Vascular Disease	2	11
G.I. History	3	17
Renal Insufficiency	1	6
Cardiac History	1	6

* HTN, hypertension; G.I, gastro-intestinal

Table 4 depicts the diagnosis on admission to the Surgical ICU. Of the 18 patients, 4 patients were diagnosed and treated surgically for ruptured abdominal aortic aneurysms (rAAA) (22%). All patients in this category developed intra-abdominal hypertension (IAH), and 2 of these AAA patients went on to develop abdominal compartment syndrome (ACS). Three other patients were admitted with burns (17%) and 3 were trauma patients (17%). In total 13 were diagnosed with intra-abdominal hypertension (72%) and 5 patients went on to develop ACS within the first 96 hours of ICU admission (28%). Four out of 5 patients with ACS were treated surgically. Three ACS patients survived, and two did not survive. The two ACS patients that did not survive died as a result of multiple medical complications.

Table 4 - Diagnosis upon ICU Admission

Diagnosis	**Frequency**	**Patients Diagnosed with IAH**	**Patients Diagnosed with ACS**	**Patients Surgically treated for ACS**
Septic shock	1	1	0	0
Incarcerated Hernia Repair	2	1	1	1
Trauma	3	2	0	0
Burn	3	1	1	1
AAA Repair	4	4	2	1
Perforated Duodenal Ulcer	1	1	1	1
Intra-peritoneal Bleed	1	1	0	0
Perforated Small Bowel-Dehiscence	1	0	0	0
Pancreatitis	1	1	0	0
Retroperitoneal Bleed	1	1	0	0
Total	**18**	**13**	**5**	**4**

*AAA indicates abdominal aortic aneurysm; IAH, Intra-abdominal Hypertension; ACS, Abdominal Compartment Syndrome.

APACHE II scores are depicted in Table 5. Non-survivors had higher APACHE scores than survivors. These scores were calculated on the first day of admission to the ICU. This is a quantitative measure of the severity of illness of the acutely ill patient in ICU. Physiological variables with the greatest deviation from normal are assigned higher scores.

Table 5 - APACHE II Scores

	ACS Patients	(APACHE II Scores)		Non-ACS Patients	(APACHE II Scores)	
		Mean	SD		Mean	SD
Survivors	3	12	3.4	9	16	3.1
Non-Survivors	2	36	4.1	4	22.7	3.7

Research Question 1 - Is the routine monitoring of abdominal perfusion pressures (APP) every 6-8 hours a clinically useful predictor of patient survival for the development of ACS during the first 96 hours following ICU admission?.

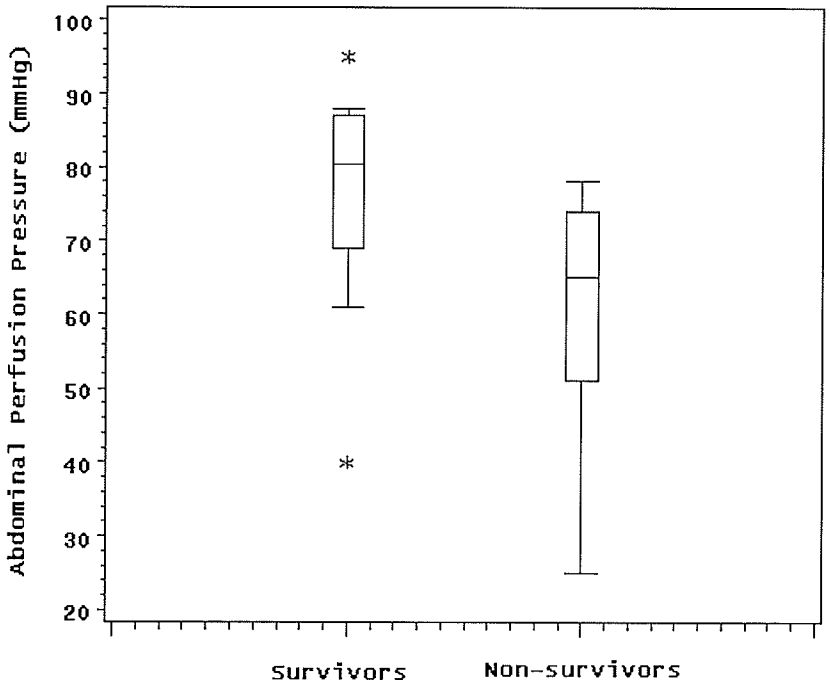
Table 6 presents abdominal perfusion pressures (APPs) in patients with and without abdominal compartment syndrome (ACS), and in relation to survivors and non-survivors of ACS. Due to the fact that patients had a variable number of APPs taken (ranging from 2 to 23), an array of summary values was used to determine if APPs were predictive of ACS or survival. Because of the skewed distribution of the APP values, the non-parametric Kruskal Wallis Test was used to describe the findings. It was determined that APPs ≥50mmHg was not significantly higher in survivors than non-survivors (p =0.1778). The maximal APP or the highest APP values were significantly higher in the survival group (p=.0337) (Figure 4). The number of APPs done for each group were different (survivors (9.83 +/-6.5) vs. non-survivors (4.5 +/- 2.8), p= .0279). When the percentages of APP greater than 50mmHg were compared (p =.49) in survivors and non-survivors, there were no statistically significant differences. Therefore an APP ≥ 50mmHg was not a useful predictor of patient survival.

Table 6 - Characteristics of Abdominal Perfusion Pressures (APPs) in Patients With and Without Abdominal Compartment Syndrome (ACS), and in Relation to Survivors and Non- Survivors of ACS.

	ACS (N=5)	**Non-ACS (N=13)**
Number of APPs done	4 (2-23)	6 (3-18)
Number of APP>50	2 (0-23)	5 (0-16)
Percentage of APP>50	92.3 (0-100)	90.0 (0-100)
Mean APP mmHg	56.0 (35.5-73.09)	62.4 (22-74.5)
Median APP mmHg	56.0 (35.5-73.0)	62.0 (21.0-74.0)
Max APP mmHg	58.0 (40-95)	74 (25-88)
	Survivors (N=12)	**Non-Survivors (N=6)**
Number of APPs done	8 (2-23)	4 (2-10)
Number of APP>50	5.5 (0-23)	3 (0-9)
Percentage of APP>50	90.6 (0-100)	95.0 (0-100)
Mean APP mmHg	62.6 (35.5-74.5)	59.2 (22-65.75)
Median APP mmHg	61.0 (35.5-74.0)	59.0 (21.0-64.0)
Max APP mmHg	80.5 (40-95)	62 (25-78)

*APP, abdominal perfusion pressure; ACS, abdominal compartment syndrome

Figure 4. Maximal Abdominal Perfusion Pressures in Survivors and Non-Survivors



*p= 0.0337

There was no significant difference between individuals who survived and those who did not survive with all demographic variables except APACHE II scores (p=0.0005). APACHE II scores or the severity of illness was significantly higher in non-survivors (Table 7).

Table 7 - Relationship of Demographic Variables to Survivors and Non-Survivors

Demographics	**Survivors**	**Non-Survivors**	**p-value**
Age	48.9 ± 15.7	43.8 ± 15.6	0.53
Apache II	16.75 ± 5.01	30.17 ± 8.26	0.0005
Male	9/12 (75%)	5/6 (83.3%)	0.69
HTN	7/12 (58.3%)	4/6 (66.7%)	0.73
PVD	3/12 (25.0%)	4/6 (66.7%)	0.09
Renal Insufficiency	1/12 (8.3%)	2/6 (33.3%)	0.18
Cardiac	1/12 (8.3%)	2/6 (33.3%)	0.18
GI	3/12 (25.0%)	2/6 (33.3%)	0.71
# of co-morbidity	1 (0-2)	2.5 (1-4)	0.16

* HTN, hypertension; PVD, Peripheral vascular disease; GI, gastro-intestinal

There was no significant difference between individuals who developed ACS and those who did not develop ACS with all demographic variables (Table 8).

Table 8 - Relationship of Demographics Variables to ACS and Non-ACS Patients

Demographics	**ACS**	**Non ACS**	**p-value**
Age	36.5 ± 16.8	50.4 ± 14.0	0.12
Apache II	22.8 ± 12.9	20.6 ± 7.38	0.65
Male	4/5 (80.0%)	10/13 (76.9%)	0.89
HTN	3/5 (60.0%)	8/13 (61.5%)	0.95
PVD	2/5 (40.0%)	5/13 (38.5%)	0.95
Renal Insufficiency	1/5 (20.0%)	2/13 (15.4%)	0.81
Cardiac	2/5 (40.0%)	1/13 (7.7%)	0.10
GI	2/5 (40.0%)	3/13 (23.1%)	0.47
# of co-morbidity	1 (1-4)	1 (0-2)	0.58

* HTN, hypertension; PVD, Peripheral vascular disease; GI, gastro-intestinal

Research Question 2 - Is there a relationship between a positive fluid balance and an increase in intra-abdominal pressures during the first 96 hours following ICU admission?

Table 9 depicts the mean and standard deviation of fluid balance and intra-abdominal pressure values.

Table 9 - Mean and Standard Deviation of Fluid Balance, Intra-abdominal Pressure and Abdominal Perfusion Pressure

Variable	N	Mean	SD
Fluid Balance (mls)	53	5741	6650
MIAP mmHg	55	18.76	5.99
MMAP mmHg	55	82.32	10.73
MAPP mmHg	55	63.40	11.90
IAP50 mmHg	55	18.76	6.06
MAP50 mmHg	55	82.500	11.34
APP50 mmHg	55	63.57	12.71
MxIAP mmHg	55	21.78	7.899
MxMAP mmHg	55	89.61	13.63
MxAPP mmHg	55	71.21	14.21

*MIAP, mean intra-abdominal pressure; MMAP, mean of mean arterial pressure; MAPP, mean abdominal perfusion pressure; IAP50, median Intra-abdominal pressure; MAP50, median mean arterial pressure; APP50, median abdominal perfusion pressure; MxIAP, largest value-Intra-abdominal pressure; MxMAP, largest value- mean arterial pressure; MxAPP, largest value – abdominal perfusion pressure; N, number

Table 10 and Figure 5 shows a significant positive correlation between maximal IAP and a positive fluid balance ($r = 0.35666$, $p = 0.008$).

Table 10 - Spearman Correlation Coefficient Relating Maximal Intra-Abdominal Pressure, Maximum Abdominal Perfusion Pressure and Fluid Balance

Fluid Balance	MxIAP mmHg Largest Value IAP*	MxAPP mmHg Largest Value App*
Spearman Correlation Coefficients	.35666	-.32036
Prob>/r/ under HO: Rho=0	.0088	0.0194
Number of Observations	53	53

*MxIAP, largest value - intra-abdominal pressure; Mx APP, largest value – abdominal perfusion pressure

Figure 5. Relationship of Fluid Balance with Intra-abdominal Pressures

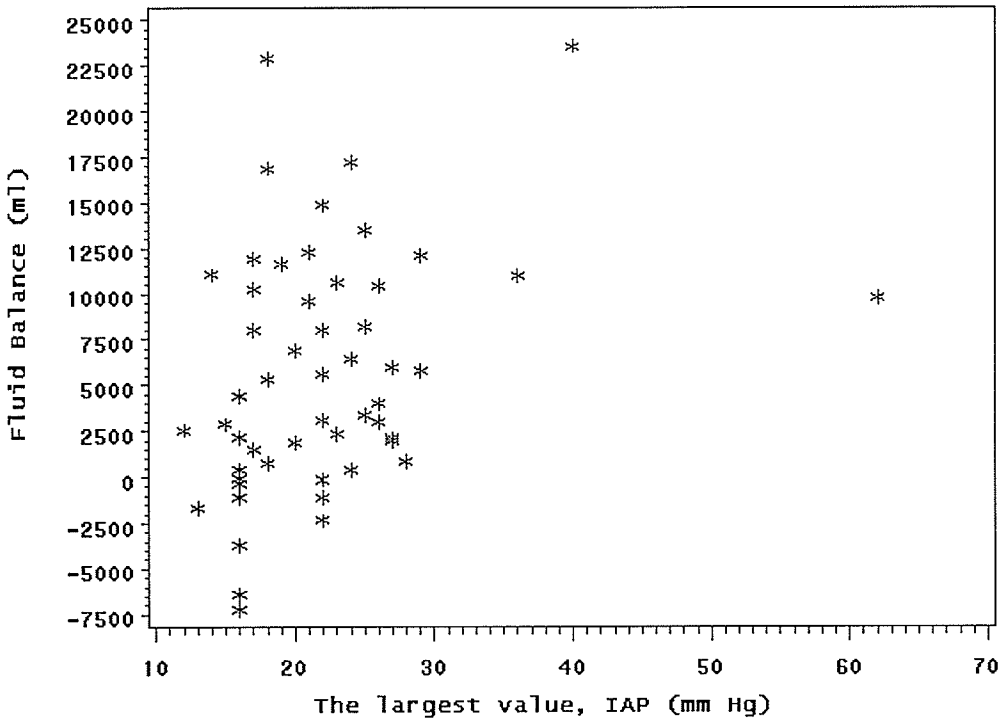
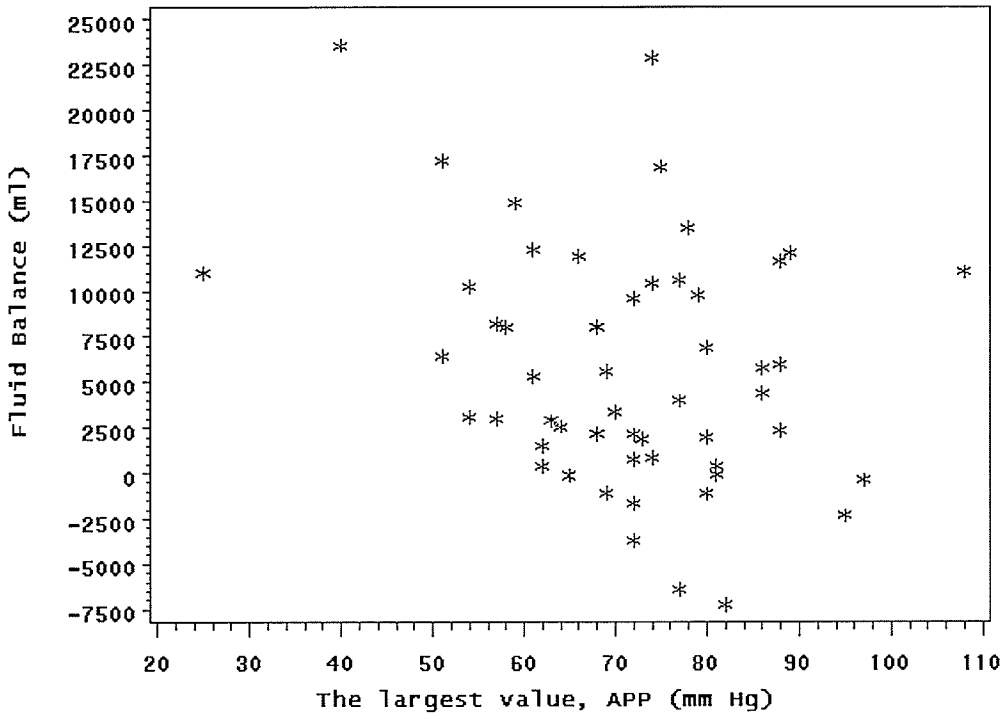


Table 10 and Figure 6 show an inverse relationship between maximal APP and positive fluid balances. Abdominal perfusion pressure increased as the positive fluid balance decreased ($r = -0.32036, p = 0.01$).

Figure 6. Relationship of Fluid Balance with Abdominal Perfusion Pressures



Research Question 3 - Is there is a correlation between IAP and inferior vena cava pressure measured from the femoral venous catheter?

Forty-seven IAP and FVP readings were simultaneously measured on five patients during the first 96 hours of ICU admission. Using Pearson Correlation Coefficients (Table 11 and Figure 7), the results were found to be statistically significant with a positive correlation between inferior vena cava pressure (IVC) and IAP ($r = 0.92913$, $P = <.0001$).

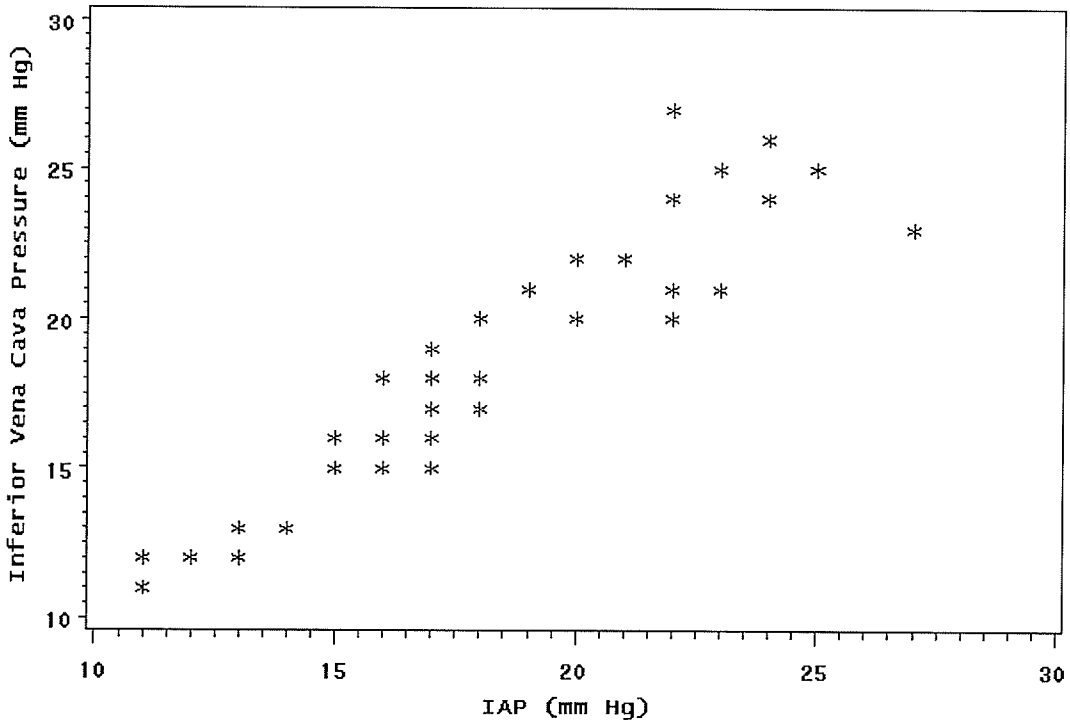
Table 11 - Pearson Correlation Coefficients Relating IAP and IVC

Variable	N	Mean	SD
IAP mmHg	47	18.17	3.90
IVC mmHg	47	18.55	4.30

	IAP	IVC
Pearson Correlation Coefficients, N=47	1.000	0.92913
Prob>/r/ under HO: Rho=0		<0.0001

IAP indicates intra-abdominal pressure; IVC, inferior vena cava pressure. (r 0.92913, p =<0.0001)

Figure 7. Relationship of Inferior Vena Cava Pressure with Intra-abdominal Pressure



Summary of Chapter

This chapter describes the findings obtained in the analyses of the data including the statistical tests used, the values of calculated statistics, and the significance of the findings. A summary of the findings, accompanied by tables highlight the most noteworthy results. A description of study participants, and key variables are provided. The findings from this study confirm the positive correlation between IVC pressure and IAP, and the positive correlation between maximal IAP and a positive fluid balance.

Chapter V

Discussion

Introduction

The purpose of this study was to examine the usefulness of routine intra-abdominal pressure monitoring in the early detection of abdominal compartment syndrome (ACS) in a convenience sample of eighteen patients admitted to a surgical intensive care unit at a tertiary care hospital. In this chapter, interpretation of the research findings of the study will be discussed within the context of relevant literature, overview of the application of the conceptual model, and specific research questions that directed the inquiry. Nursing implications for improving nursing practice and education will be proposed, study limitations will be identified, and recommendations for future nursing research will be made.

Application of the Human Response to Illness Model

The nursing model of Human Response to Health and Illness (Mitchell, Gallucci, & Fought, 1991) was selected as an appropriate conceptual framework to guide this research because it contains components that are applicable to the development of potential strategies that could lead to early diagnosis and treatment of ACS, thereby preventing or ameliorating complications associated with this syndrome. This model, described in detail in Chapter 2 (p.5 to p.22) permits the organization of new knowledge

emerging from data collected. In this study the analysis was principally guided by the model's pathophysiological and behavioural perspectives.

Pathophysiologic Perspective

The pathophysiological effects of increased IAP are widespread and are a source of significant morbidity in critically ill patients (Cheatham, 1999). ACS is a clinical entity that develops from progressive, acute increases in IAP and adversely affects multiple organ systems including the renal, pulmonary and cardiovascular systems (Cheatham, 1999). Seventy-two percent of patients in this study developed intra-abdominal hypertension and 28% went on to develop ACS within the first four days of ICU admission. All of the patients were post –operative, and had received large volumes of fluid during the resuscitation phase. This may have contributed to the development of IAH. Fluid resuscitation can lead to increased intra-abdominal pressure (IAP). The circulatory effects of increased intra-abdominal pressure (IAP) combined with extracellular hypervolemia from fluid resuscitation, may lead to abdominal wall edema and ischemia, reducing abdominal wall compliance. This may lead to multiple system organ failure (Mutoh et al., 1992).

Treatment may involve the need for immediate decompression of the abdomen, without which the syndrome of end-organ damage and reduced oxygen delivery may lead to development of multi organ failure, and ultimately death (Ivy et al., 2000). In this study, five patients were diagnosed with ACS and four of them underwent surgery for abdominal decompression. Increased IAP is an important signal of underlying abdominal complications and may be a prognostic indicator of the patient's physiological status (Cheatham, 1999). IAP is not a new concept but only recently its importance and

therapeutic implications for all ICU patients at risk for developing ACS have become apparent. To prevent and treat such adverse effects from intra-abdominal hypertension, the IAP must be monitored routinely. Monitoring intra-abdominal pressure using bladder pressures is a relatively non-invasive and straightforward procedure, and currently is not performed on a systematic basis in the intensive care unit.

Behavioral Perspective

Behavioral signs of abdominal compartment syndrome include abdominal distention, increased intra-abdominal pressure (IAP>15mmHg), oliguria refractory to volume administration, hypoxia with increasing airway pressures, hypercarbia, tense or massively distended abdomen, metabolic acidosis, and severe abdominal pain (Schulman, 2000).

Research Question 1 - Is the routine monitoring of APP (abdominal perfusion pressure) every 6-8 hours a clinically useful predictor of patient survival or the development of ACS during the first 96 hours following ICU admission?

Cheatham, White, Sagraves, Johnson, & Block (2000) studied the use of abdominal perfusion pressure as a resuscitative endpoint and predictor of survival in patients with intra-abdominal hypertension .APP is physiologically important because it may determine the severity of intra-abdominal hypertension present, and the adequacy of tissue perfusion (Cheatham et al., 2000). It has been suggested by Cheatham et al. (2000) that an APP of 50mmHg or greater seems to be a positive indicator of patient survival. In

this study, all patients admitted to their surgical ICU between 1997 and 1999 were prospectively evaluated for the presence of IAH. IAH was identified as an IAP of 15mmHg or greater. This pressure has been demonstrated in multiple studies to cause physiological problems such as cardiac, pulmonary, renal, gastrointestinal, and hepatic dysfunction (Cheatham et al., 2000). One hundred and forty nine patients who had IAH underwent IAP monitoring every four hours. Sixty-eight percent of patients were from the trauma service, and 14% were from vascular surgery. The mean age was 51 years +/- 19. The mean IAP was 22 +/-8mm Hg. Overall, 47% of patients survived the development of IAH, and 27% survived subsequent progression to ACS. Age was identified as a significant predictor of survival after the development of IAH. Patients surviving IAH were significantly younger (44years vs. 59 years; $p<.0001$). Maintenance of an APP of at least 50 mmHg maximized both the sensitivity (76%) and specificity (57%) of APP as a predictor of patient survival (Cheatham et al., 2000). They found that APP was the best predictor of patient outcome, and statistically superior to IAP monitoring alone in predicting patient survival from intra-abdominal hypertension and ACS. This is currently the only article that has studied the use of abdominal perfusion pressure monitoring.

Cheatham's findings were inconsistent with this study. The mean APP of $\geq$ 50 mmHg was not significantly greater in survivors compared to non survivors of ACS. However, survivors in this study had significantly higher maximal abdominal perfusion pressures than non-survivors. This was the only similarity between the two studies.

There were several dissimilarities that may have contributed to the difference in findings between the two studies. There was a difference in the mean age of study

subjects. The mean age was younger in the Cheatham et al. (2000) study compared to a mean age of 58 years in this study. This current study had no correlation between age and survival as compared to the Cheatham study where a positive correlation was found. There was a difference in illness patterns of patients. Cheatham et al. (2000) had a greater percentage of trauma and vascular patients, where in this study, the study subjects were from several different areas (see Table 4 p.58). Another potential contributing factor may be the difference in sample sizes. This study had eighteen patients compared to one hundred and forty-nine patients in the Cheatham et al. (2000) study.

Although, in this study, there was no significant difference in using an APP of 50 mmHg or greater as a predictor for patient survival, important clinical findings emerged. As mentioned earlier, survivors of IAH had higher maximal abdominal perfusion pressures than non-survivors. This is important to note, since calculation of APP addresses the adequacy of tissue perfusion and the need for additional resuscitation or abdominal decompressive surgery. As noted by Cheatham et al. (2000), the inability to maintain an APP of at least 50 mmHg was seen to be an appropriate indication for performing a decompressive laparotomy allowing for the maintenance of an open abdomen until the patient's clinical status improved (Cheatham et al., 2000).

This study consisted of a much smaller sample size than the previous study; therefore, it was not possible to do multivariate testing. Although frequent monitoring of APP (MAP-IAP) may provide useful information for the early detection of ACS and restoring tissue perfusion, further studies will be necessary to verify the Cheatham findings.

It will also be necessary to provide health care staff with educational programs concerning the value of using APP, an easily calculated physiologic parameter. Knowledge of APP values may aid nurses and surgeons evaluating a patient with intra-abdominal hypertension when the difficult decision of when to perform a decompressive laparotomy must be made (Cheatham, 1999).

Research Question 2 - Is there was a relationship between a positive fluid balance and an increase in intra-abdominal pressures during the first 96 hours following ICU admission.

This research question was developed to determine if large volumes used in resuscitative treatments leads to an increase in intra-abdominal pressures over time. It was demonstrated there was a statistically significant correlation between maximal IAP and a positive fluid balance. This finding was consistent with that of Ivy, Atweh, Palmer, Possenti, Pineau, & D'Aiuto (2000) who studied a group of burn patients. The researchers hypothesized that the massive volume of fluid infused in the patients during the first day of admission would be related to the development of intra-abdominal hypertension (IAH). This study showed a statistically significant correlation between maximal IAP and cumulative fluid load. That is, the volume of fluid in the abdomen and the compliance curve of the abdominal wall determine the IAP (Ivy et al., 2000). The authors suggested that the routine monitoring of IAP led them to diagnose and treat intra-abdominal hypertension and ACS in the early stages. One of the three burn patients in this research study developed ACS and required emergent abdominal decompression. His pre-decompression intra-abdominal pressure was recorded as high as 60mmHg, and he

had received over 20 liters of fluid within the first 24 hour period following admission to the ICU.

Cheatham (1999) also suggested that third-spacing of fluid secondary to fluid resuscitation can cause IAH. Massive volume resuscitation for any reason (burns, severe pancreatitis, hemorrhagic shock) can lead to increased IAP, particularly in the post operative period (Saggi, Sugerman, Ivatury, & Bloomfield, 1998; Cheatham, 2000).

Oelschlager, Boyle, Johansen, & Meissner (1997) investigated fluid resuscitative measures associated with ruptured abdominal aortic aneurysm (rAAA) repair. It was shown large fluid replacement resulted in massive edema of the bowel, but that there was an increased survival in the group of patients with delayed abdominal wall closure. As in massively resuscitated trauma victims (patients receiving >10litres of fluid), delayed laparotomy closure in rAAA patients may confer a physiologic and survival benefit (Oelschlager et al., 1997).

In this research study, four patients had rAAA repairs and all developed intra-abdominal hypertension with positive fluid balances. Two of them went on to develop ACS and all four patients had initial laparotomy closure of their abdominal wall

Research Question 3 - Is there a correlation between IAP and inferior vena cava (IVC) pressure measured from a femoral venous catheter?

The research question was developed to determine if a more efficient method to measure IAP could be demonstrated. Measuring IVC pressures requires placement of femoral line. Ethically, therefore, in this study, only those patients with femoral lines

already in place, and at high risk for intra-abdominal hypertension were enrolled in the study. Using Pearson Correlation Coefficients, the results were found to be statistically significant with a positive correlation between inferior vena cava pressure and IAP.

No other human studies to date have examined this relationship. One study using an animal model studied the correlation between inferior vena cava pressure and IAP. Harman, Kron, McLachlan, Freelender, & Nolan (1982) found that IVC pressures were directly related to IAP.

The findings from this study showed a high correlation between IVC pressure and bladder pressure readings ($p<.0001$). The statistical findings are comparable to Kron et al.'s 1984 study of the correlation between directly measured IAP and bladder pressures which is considered the "Gold Standard" ($p <.0001$). One could question if the IVC method and bladder pressure method are equally reliable techniques for IAP monitoring. The IVC method would allow nurses to easily trend IAP readings and continuously monitor the patient for any sudden increase or change in the IAP measurement. In using the IVC method, nurses would not have to perform bladder pressure measurements. Although this technique is a time saving measure for the bedside nurse, one would not insert a femoral venous catheter for the sole purpose of IAP monitoring because of the associated risks with femoral lines. These risks include infection, deep vein thrombus, and risk of arterial injury (Cheatham, 1999). This method is suggested only for those patients with a femoral line already in situ.

Limitations of the Study

In analyzing the results of this study, the limitations which may have inadvertently weakened the validity of the study must be kept in mind.

A limitation to this study was the nurses' inability to maintain the 6-8 hour IAP and APP monitoring schedule during times when the acuity of the patient was too high, or the patient's condition was very unstable. Every effort was made to ensure that the 6-8 hour monitoring was maintained for the first 96 hours of ICU admission, however, patients did not have equal time frames for IAP and APP monitoring. This may have affected the findings due to the unequal number of measurements done for each patient.

A second limitation was the study design of a non-randomized convenience sample. This sample is less likely to produce accurate and representative samples. Despite this fact, most research samples in most disciplines, including nursing, are nonprobability samples because of their convenience and economy (Polit & Hungler, 1999).

Smaller sample sizes tend to produce less accurate estimates than larger sample sizes. In addition, small sample sizes are less representative of the population of interest, and have larger sampling errors (Polit & Hungler, 1999). Due to the infrequency of admittance of patients at high risk for ACS to the ICU, only a small sample size could be recruited.

Implications for Nursing Practice, Education, and Research

The model used for this study provides a framework for nurses working with critically ill patients. The Human Response to Illness Model encourages the practicing

nurse to utilize the four perspectives: normal physiologic regulatory responses, pathophysiologic responses, experiential responses, and behavioral responses when caring for patients and their families.

Of particular importance is the pathophysiological perspective since intra-abdominal hypertension (IAH) and ACS are detrimental to organ function. Although the exact level of IAH that defines a "critical IAP" remains subject to debate, there is consensus that decompression should be performed at levels of IAP above 25 mmHg (Malbrain, 1999). The best practice to ensure early diagnosis of IAH and ACS was considered using the behavioral perspective. It was shown that it may be appropriate to adopt a more routine approach to IAP measurement. In this study nurses were asked to take IAP measurements every six to eight hours in patients in the ICU, who were deemed to be at high risk for IAH and ACS. It could be recommended that a base-line IAP measurement be undertaken in the operating room, at the end of abdominal closure, to assess "abdominal tightness" before the patient is transferred to the surgical ICU.

Through careful monitoring and physical assessment, the ICU nurse has the opportunity to affect patient outcomes through early recognition of signs and symptoms, and prompt treatment. Nurses may be able to easily trend IAP measurements by using the IVC method in those patients with femoral venous catheters.

The findings from this study also have implications for nursing education. For example, Clinical Nurse Specialists working with patients and staff in intensive care units must be aware of the potential complications of IAH or ACS in high risk patients, and disseminate the rapidly expanding knowledge regarding these potentially life-threatening complications to ICU staff nurses. Education programs must include signs and symptoms

of IAH and ACS as well as knowledge of correct measuring techniques for IAP monitoring.

Larger studies, perhaps multi-center, incorporating the routine measurement of APP and IAP are needed to provide additional support for the findings of this study.

Randomized trials should include the burn population and look at the role of fluid resuscitation and IAP monitoring. Only one study to date has studied the effects of fluid resuscitation of burn patients.

Conclusion

In conclusion, the findings of this research study indicate that IAH and ACS can complicate the hospital course of critically ill surgical patients. It is clear from the results that patients undergoing massive fluid resuscitation are at high risk for developing ACS. Abdominal perfusion pressure monitoring may be an important determining factor in aiding the surgeon's decision when patients require abdominal decompressive surgery. The significant correlation of IAP and inferior vena cava pressure from this study may encourage other researchers to further verify these findings in a larger trial. Bladder pressure estimation of IAP is a non-invasive, easy, and highly reproducible method of assessing the presence of IAH and ensuing ACS (Ivy et al., 2000). The routine monitoring of intra-abdominal pressures may contribute to improved patient outcomes.

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APPENDIX A

APPROVAL CERTIFICATE

14 May 2002

TO: **Tricia Carta** (Advisor B. Naimark)
Principal Investigator

FROM: **Lorna Guse, Chair**
Education/Nursing Research Ethics Board (ENREB)

Re: **Protocol #E2002:040**
"The Use of Routine Intra-abdominal Pressure Monitoring of Adult ICU Patients for the Early Detection of Abdominal Compartment Syndrome"

Please be advised that your above-referenced protocol has received human ethics approval by the **Education/Nursing Research Ethics Board**, which is organized and operates according to the Tri-Council Policy Statement. This approval is valid for one year only.

Any significant changes of the protocol and/or informed consent form should be reported to the Human Ethics Secretariat in advance of implementation of such changes.

APPENDIX B

Policy and Procedure Guidelines

Intra-abdominal Pressure Measurement

(Health Sciences Centre)

Health Sciences Centre

NURSING PROCEDURE

SUBJECT	SECTION	
INTRA ABDOMINAL PRESSURE MONITORING VIA INDWELLING URINARY CATHETER	Gastrointestinal	
	EFFECTIVE DATE November, 2000	CODE 30.50.03
AUTHORIZATION DIRECTOR OF PATIENT SERVICES - CRITICAL CARE MEDICAL DIRECTOR	REVISION DATE	PAGE 1 of 3

PURPOSE:
To measure intra abdominal pressures (IAP's) indirectly. Acceptable range less than 15 mmHg and at the discretion of the physician. Higher levels require physician interpretation.

POLICY:
Registered Nurses in SICU, MICU, and PACU are allowed to transduce IAPs via an indwelling Foley catheter.

EQUIPMENT:

1) Indwelling Foley catheter with Foley drainage bag
2) 60 cc's luer lok syringe
3) Standard IV infusion set
4) 1000 cc sterile NS bag
5) 1 extra stopcock
6) ICP-style transducer with 4-inch pressure tubing
7) 16 or 18 ga needle
8) Non-toothed clamp (or rubber-tipped forceps)
9) *Alcohol swabs*

PROCEDURE:

1) Connect NS IV bag and IV tubing to ICP transducer set and pressure tubing. Prime set to remove all air.

2) Wipe the culture aspirator port with an alcohol swab. Insert the 16 or 18 ga needle into the culture aspirator port of the urinary bag tubing.

3) Attach the needle to the flushed pressure tubing.

4) Turn stopcock off to patient and "zero" transducer to the bedside monitor at the level of the symphysis pubis.

KEY POINTS:

See diagram (appendix A) for complete assembly.

SUBJECT	REVISION DATE	CODE	PAGE
INTRA ABDOMINAL PRESSURE MONITORING VIA INDWELLING URINARY CATHETER		30.50.03	2 of 3

Clamp the urinary drainage bag immediately distal to the culture aspirator port. Turn the stopcock off to the patient and pressure transducer.

6) Aspirate 50-60cc's of NS from IV bag into the luer lok syringe. Volumes of close to 100cc's can falsely elevate IAP readings.

7) Turn stopcock off to NS bag and open to patient

8) Install 50 cc's NS into the bladder.

9) Release clamp on urinary drainage bag momentarily to release air from Foley catheter. Do not allow instilled fluid to drain from Foley catheter. This establishes a fluid column from which to obtain an accurate IAP.

10) Re-clamp.

11) Ensure stopcocks are off to IV bag and luer lock syringe.

12) Measure transduced pressure levelled to the symphysis pubis on end expiration. Pressure is measured at end expiration to negate the effects of intrathoracic pressure.

13) Release clamp from urinary catheter.

14) Frequency of transduced pressure per physician's order.

DOCUMENTATION:

Document pressure in mmHg on ICU flowsheet or addendum.
Document size of needle selected for the procedure.

REFERNCE LIST:

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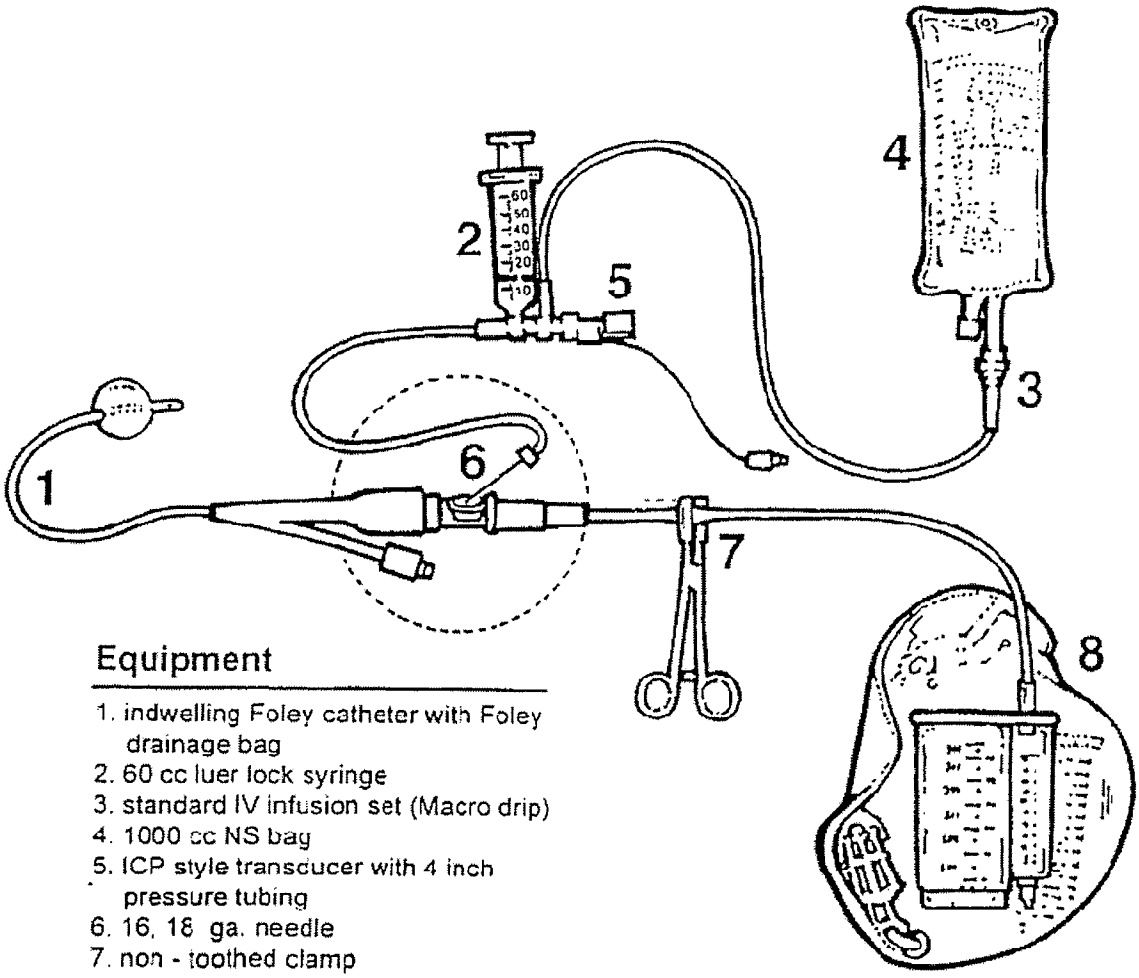
Infection Control Unit, 2000.

SUBJECT	REVISION DATE	CODE	PAGE
INTRA ABDOMINAL PRESSURE MONITORING VIA INDWELLING URINARY CATHETER		30.50.03	3 of 3

APPENDIX A:



Equipment

1. indwelling Foley catheter with Foley drainage bag
2. 60 cc luer lock syringe
3. standard IV infusion set (Macro drip)
4. 1000 cc NS bag
5. ICP style transducer with 4 inch pressure tubing
6. 16, 18 ga. needle
7. non - toothed clamp
8. urine collection bag

APPENDIX C

Patient (or Authorized Third Party) Consent Form

Thesis Study Name: The use of routine intra-abdominal pressure measurements in the early diagnosis of intra-abdominal hypertension for the prevention of abdominal compartment syndrome.

Investigator: Tricia Carta, RN, BN – Master's of Nursing student at the University of Manitoba. Thesis Project

Thesis Committee Members: Dr. Barbara Naimark
Dr. Bill Diehl-Jones
Dr. Perry Gray

This consent form , a copy of which will be left with you for your record and reference is only part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more detail about something mentioned here, or not included here, you should feel free to ask. Please take the time to read this carefully and to understand any accompanying information.

If I (or an authorized third party) decide to sign this consent form, my signature means that:

I have agreed to participate in this study because I may develop an increase in my abdominal pressure signaling the presence of a condition called "Abdominal Compartment Syndrome." The purpose of this thesis project is to study the use of urinary bladder pressure as a measure of abdominal pressure. A gauge will be used to monitor bladder pressure through the urinary catheter that has been inserted on my admission into the intensive care unit as part my usual care. I have been provided with an explanation and diagram that gives me an idea of how this measurement is performed.

The nurses will take bladder pressure measurements every four hours during my stay in the intensive care unit, or until I am no longer at risk for developing increased abdominal pressure.

The research nurse will collect the information from my chart that will include information about my medical condition such as: age, previous medical conditions, previous surgeries, diagnostic test results. Vital signs such as my heart rate, blood pressure, urine output, and other signs will be recorded. All health information collected from my chart will be done according to the Personal Health Information Act Guidelines.

All information will immediately be assigned a code number and my identity will be protected.

The information collected may be shared in confidence between Tricia Carta, Dr. Naimark, Dr. Gray, and Dr. Diehl-Jones (Thesis committee members). The committee members will be the only people to have access to information and / or to my identity.

Information presented at conferences and published will maintain confidentiality through use of summarized data.

I am aware of the possible risk of a bladder infection with intra-abdominal pressure monitoring.

There is no direct benefit to me for participating in the study, but my participation may lead to improved care of patients like me in the future.

There is no remuneration being offered for my participation in this study.

I can withdraw from this study at any time without penalty or effect on my care. In order to withdraw all I will need to do is notify a nurse of my wishes. The nurse will in term notify the appropriate persons.

If you would like a copy of the summary of the research report please notify the investigator by filling in the section below and sending it to Tricia Carta, Health Sciences Centre, Surgical ICU, 820 Sherbrook Avenue, R3A 1R9.

Your signature on this form indicates that you have understood to your satisfaction the information regarding participation in this research project and agree to participate as a subject. In no way does this waiver your legal rights nor release the researcher, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time. Your continued participation should be as informed as your initial consent, so you should feel free to ask for clarification or new information throughout your participation.

Tricia Carta, BN
Dr. Barbara Naimark PhD

The Education / Nursing Research Ethics Board have approved this research. If you have any concerns or complaints about this project you may contact any of the above-named persons or the Human Ethics Secretariat at A copy of this consent form has to be given to you to keep for your records and reference.

_____ We recognize the importance of your participation. Thank-you. _____

Participant Signature_____ Date_____

Significant Other Signature_____ Date_____

Relationship _____

Researcher
or Delegate Signature_____ Date_____

Thesis Study Name: The use of routine intra-abdominal pressure measurements in the early diagnosis of intra-abdominal hypertension for the prevention of abdominal compartment syndrome.

Please send me a copy of the summary of the research report

Send to:_____ (Name)

_____ (Address)

_____

_____

APPENDIX D

APACHE II ICU SCORE INSTRUMENT

3170433887

Winnipeg ICU APACHE Score

Serial: Site: H S C Study #:

Admit Date: Admit Time:

Physiology Information:

Elective Surgery (Y/N)

Chronic History (Y/N)

Active Treatment (Y/N)

Glasgow Coma Scale:

Eye Opening (1 - 4)

Motor Response (1 - 6)

Verbal Response (1 - 5)

Laboratory Data:

Ser Sodium (mMol/L) (130 - 149)

Ser Potass (mMol/L) (3.5 - 5.4)

Hematocrit (%) (30 - 46.9)

WBC'S (1000's) (3 - 14.9)

Ser Creat (uMol/L) (53 - 133)

Acute Renal Failure (Y/N)?

Y = Urine output <136 cc in an 8hr block in the first 24hrs in ICU & creatinine > 133 umols

Vital Signs:

Temp °C (36 - 38.4)

Syst BP mmHg

Dias BP mmHg (mean 70 - 109)

Heart Rate /min (70 - 109)

Resp Rate /min (12 - 24)

ABG's:

FIO_2 % (21 - 100)

PO_2

CO_2

pH (7.33 - 7.48)

Ser CO_2 (22 - 31.9)

Age: AS:

BRIEF Significant PATIENT Past History PRIOR to ICU Admission

- Primary Diagnosis -Primary reason patient admitted to ICU
- Acquired Diagnosis / Complications/Procedures/Surgery

DATES

Comorbidities

Please Circle

Pharmacy Data: YES / NO

Laboratory Data: YES / NO

rev01/04/99

Data : Initial: