ABDOMINAL COMPARTMENT SYNDROME IN ABDOMINAL SURGICAL PATIENTS ADMITTED TO INTENSIVE CARE UNITS OF HARARE TEACHING HOSPITALS

Prevalence and Clinical Significance

By

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ABSTRACT

Background: Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) have been identified as significant risk factors for morbidity and mortality in critically ill surgical patients. Nevertheless, the problem remains underappreciated and routine monitoring has not been established in our intensive care units (ICU). Our units have no protocols or guidelines for IAH/ACS. This study marks the beginning of an objective look at this clinical phenomenon in our own environment for the benefit of patients at risk.

Objectives: To evaluate the prevalence and clinical significance of raised intra-abdominal pressure (IAP) and abdominal compartment syndrome (ACS) in patients who had undergone abdominal surgery and admitted to surgical intensive care units of teaching hospitals in Harare. The clinical significance was evaluated using organ specific parameters assessing dysfunction in relation to intra-abdominal pressures.

Design and setting: Prospective cohort study in intensive care units of Harare's two teaching hospitals.

Patients: Thirty eight critically ill and ventilated patients with ages above 12 years admitted to ICU after any operation involving entry into the peritoneum with no contraindication to transvesical pressure measurements were recruited into the study.

Measurements: IAP was measured every 6 hours from time of admission for 24 hours transvesically with a water manometer in cmH_2O. 20ml saline instillation volume into an initially empty bladder was used before each measurement and the measured values were converted to mmHg using the conversion ratio $mmHg=cmH_2O/1.36$. Various parameters related to cardiovascular, respiratory, gastrointestinal, renal, hepatic, and haematological systems were also recorded in order to relate these to measured intra-abdominal pressures. Patients were then followed up to 30 days post operation.

Results: Thirty eight patients (92%) developed IAH and three (8%) had ACS when maximal IAPs were considered. With mean measured pressures, twenty eight patients (74%) had IAH, with no cases of ACS. Eight patients (21%) died, with the mean pressures of non-survivors being 18±2.77mmHg and for survivors being 14.79±3.67mmHg. These pressures were significantly different (p=0.017). The most commonly affected organ systems were respiratory, cardiovascular and neurological according to SOFA score. There was no relationship between patients' IAP and length of ICU stay and the ASA score failed as a proxy for IAP measurements.

Conclusion: The problem of intra-abdominal hypertension affects our abdominal surgical patients with IAH having demonstrable effect on organ function and outcome. Although numbers are small, this study seems to suggest that measurements of IAP should be part of monitoring of our abdominal surgical patients. Further studies in critically ill patient populations with larger numbers are recommended.

DEDICATION

To

Tsungai, my wife of always;

My dearest son, Taurai Jr;

and my lovely daughter, Ratidzo.

Your sacrifices were beyond measure.

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I have only reached this far through the priceless efforts of many giants on whose shoulders I have been riding. To try and mention them individually may only serve to underestimate my appreciation. Senior members of the Department of Surgery & Radiology, senior registrars and colleagues, members of the Department of Anaesthetics & Critical Care, senior nursing sisters and nursing students of critical care, hospital administrators; this work would have been a more difficult task, and perhaps a pipe dream, without your immense contributions.

I might just mention a few names though. Mr C. Matema, statistician with the Faculty of Science, University of Zimbabwe, for the data analysis and statistical modeling. Mr Sarai Nyawasha from the Department of Medical Records, Parirenyatwa, for the difficult task of following up all the cases during the busy schedules of his usual work. Dr. S. Shumbairerwa, consultant anaesthesiologist, who made me realise critical care has no bounds and encouraged me to think beyond just 'surgery'. Mr. B.A. Mbuwayesango, consultant Paediatric Surgeon, my mentor and supervisor for this work, I have no better words for you. Thank you.

DECLARATION

On my honour, I hereby declare that this submission is my own work and that to the best of my knowledge and belief, it contains no material neither already published or unpublished, nor submitted for the award of a certificate, diploma or degree in any institution of higher learning, except where due acknowledgement is made.

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Taurai Zimunhu

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GLOSSARY OF ABBREVIATIONS

ACS	Abdominal Compartment Syndrome
APP	Abdominal Perfusion Pressure
ASA	American Society of Anaesthesiologists
CVP	Central Venous Pressure
GCS	Glasgow Coma Scale
IAH	Intra-abdominal Hypertension
IAP	Intra-abdominal pressure
ICU	Intensive Care Unit
MAP	Mean Arterial Pressure
PEEP	Positive End-Expiratory Pressure
SOFA	Sequential Organ Failure Assessment
WSACS	World Society of the Abdominal Compartment Syndrome

1. BACKGROUND

Abdominal compartment syndrome (ACS) encompasses a group of physiologic derangements associated with a raised intra-abdominal pressure (IAP) referred to as intra-abdominal hypertension (IAH). Both IAH and ACS are etiologically related to increased morbidity and mortality in critically ill surgical patients. Acute abdominal conditions may result in IAH and risk factors include closed incisions after lengthy abdominal surgery with large volumes of fluid resuscitation, peritonitis, severe abdominal trauma, bowel obstruction, pelvic fractures, burns, acute pancreatitis, intra-abdominal hemorrhage, retroperitoneal hematoma, and surgical closure of the abdomen under undue tension.

Normal IAP values in surgical patients are between 2 –10mmHg. Values above 12mmHg are considered to be intra-abdominal hypertension and if sustained after a minimum of three standardised measurements (taken 4-6 hours apart) beyond 20mmHg with at least one end-organ failure, abdominal compartment syndrome is defined. High intra-abdominal pressure can lead to diminished venous return and consequently reduced cardiac output despite normal arterial pressures. Respiratory failure can supervene with excessive IAH characterized by need for respiratory support in the form of mechanical ventilation. This is exhibited by high inspiratory pressures, hypoxaemia, and reduction in static and dynamic pulmonary compliance. A reduction in renal function as indicated by oliguria or anuria from IAH may be a result of poor renal blood flow, and reduced glomerular filtration rate. This may lead to acute kidney injury. Direct compression of renal vein, cortical arterioles or renal parenchyma may result in oliguria unresponsive to fluid challenge. A decline in mesenteric and bowel mucosal blood flow has

been noted with IAP above 20mmHg. An increase in abdominal pressure may lead to distant effects in other parts of the body leading to failure of renal, respiratory, cardiovascular, and even neurological systems. All these pathophysiological changes can lead to multiorgan dysfunction syndrome and ultimately death.

Knowledge of the prevalence and significance of IAH will help in formulation of relevant preventive and interventional measures appropriate for a resource constrained environment. It was with this in mind that a study in abdominal compartment syndrome and intra-abdominal hypertension in our surgical intensive care units was formulated. Although there is strong evidence supporting the deleterious effects of increased intra-abdominal pressure (IAP), intra-abdominal hypertension (IAH), and abdominal compartment syndrome (ACS) in different patient populations, measurement of IAP is not yet universally applied even among those patients where the evidence is most strong. Our units have no protocols or guidelines for IAH/ACS. This study marks the beginning of an objective look at this clinical phenomenon in our own environment for the benefit of patients at risk.

2. LITERATURE REVIEW

The history of intra-abdominal pressure as an important physiological concept in medicine seems to stretch beyond a century and half ago [1,2]. Many people believe the first documented descriptions of the association of raised abdominal pressure and renal impairment were by Wendt in 1867. Weber in 1851, and Donders in 1854, are said to have reported on the negative effects of raised respiratory pressures on venous return in both animals and humans. However, they did not appreciate the effect of similar pressures in the abdomen on stasis and venous return [3]. The early part of this century passed with little appreciation of the concept of raised abdominal pressures and in fact most believed that intra-abdominal pressure was wholly subatmospheric until the work of Emerson on cats and dogs in 1911 [4]. Another half a century had to elapse before the subject was revisited after Kron et al [5]from the University of Virginia in the early 1980s introduced the idea of abdominal decompression to improve outcome in patients presenting with tense abdomens after surgery. This was when they also introduced the method of measuring intra-abdominal pressure using a urinary catheter and using the results as criterion for indicating abdominal decompression.

The term “abdominal compartment syndrome” (ACS) is said to have been coined by Fietsan in 1989 [1]. Fietsan and colleagues from Michigan described abdominal compartment syndrome in four patients who had been operated for aortic aneurysms [2].

The effect of raised intra-abdominal pressure during laparoscopic surgery in the 1990s aided in the understanding of intra-abdominal hypertension in general surgery[6]. An almost exponential rise in publications on intra-abdominal pressure and abdominal compartment syndrome was

noticeable by the late 1990s. These studies identified not only the relative frequency of abnormal intra-abdominal pressures in general surgical patients but also in patients who had sustained trauma, burns, and those in critical care units [1].

Raised intra-abdominal pressures are associated with a series of pathophysiologic derangements and adverse effects starting even at the cellular level [1,7]. The effects then become regional, with reduction in visceral blood flow, increase in vascular resistance, and ultimately reduction in abdominal perfusion pressures [7]. These regional negative effects eventually become generalized as the afterload increases and the pre-load falls. With the progression of raised intra-abdominal pressure, end-organ failure begins and abdominal compartment syndrome sets in especially in post-laparotomy patients where intra-abdominal sepsis is compounded by pain and poor ventilation [1]. Acid base disturbances ensue and this further worsens cellular and organ dysfunction.

Clinical investigations into intra-abdominal hypertension and abdominal compartment syndrome as causes of significant morbidity and mortality among the critically ill have increased exponentially over the past decades [8,9,10,11]. As understanding of the pathophysiology of these two syndromes has evolved, IAP measurements have been identified as essential to the diagnosis and management of both IAH and ACS and have gained increased prominence in surgical intensive care units worldwide [18]. As with any prevalent disease process, the first step in dealing with IAH/ACS is to arrive at consensus definitions of pathophysiology and diagnostic criteria. The first attempt at consensus definitions occurred in 2004 at the World Congress on

Abdominal Compartment Syndrome in Australia [8,9,12]. These definitions help in standardization of research protocols and management algorithms.

2.1 Intra-abdominal pressure

If the abdomen is considered as a closed box, partially rigid (spine, pelvis, costal arch) and partially flexible (abdominal wall, viscera and diaphragm) acting as a fluid compartment, the pressures within follow Pascal's hydrostatic law [10,14,19], a principle that states that when pressure is applied to a contained fluid, the force is transmitted equally in all directions [20]. The elasticity of the walls and the character of contents determine the pressures within abdominal cavity at any given time. Thus, the IAP measured at any given time may be assumed to represent the IAP throughout the abdomen. The intra-abdominal pressure (IAP) is the steady-state pressure contained within the abdominal cavity. This, however, is affected by a number of factors like respiratory cycle, volume of solid organs or hollow viscera, presence of ascites, blood, and other space occupying lesions such as tumours or gravid uterus. Normal IAP is approximately 2-10mmHg in critically ill adults [11].

2.2 Abdominal perfusion pressure (APP)

Studies have shown that abdominal perfusion pressure (APP) is a more accurate predictor of visceral perfusion and endpoint to resuscitation [18,21,22,23]. Its concept can be viewed as analogous to that of cerebral perfusion pressure. Its measurement considers both arterial inflow (mean arterial pressure) and restrictions to venous outflow (intra-abdominal pressure). It has been demonstrated to be statistically superior in predicting patient survival from intra-

abdominal hypertension and abdominal compartment syndrome. Multiple regression analysis has identified that APP is also superior to other resuscitation endpoints such as urine output, arterial lactate levels and arterial pH. An APP of 60mmHg or better is correlated with improved survival from IAH/ACS [21,23]. The World Society of Abdominal Compartment Syndrome defines abdominal perfusion pressure as follows:

APP=MAP—IAP [8]

where APP= abdominal perfusion pressure, MAP= mean arterial pressure, and IAP= intra-abdominal pressure

2.3 Intra-abdominal pressure measurement

Overwhelming evidence has shown the shortfalls of clinical judgement or physical examination in predicting a patient's intra-abdominal pressure hence the recognition of the importance of many methods for both intermittent and continuous IAP measurement methods [24]. Intra-abdominal pressure can be monitored using a number of differing methods. The most reliable method is via a pressure transducer with an intraperitoneal catheter [24,26,29,30,31]. Less invasive options involve pressure transduction through stomach tube, bladder or rectal catheter [29]. Bladder pressure measurements as originally described by Kron [31] and later modified by Cheatham and Safcek [26] have become the most reliable and yet clinically applicable methods with minimal cost. Bladder pressure measurement through a Foley's catheter is the current standard in monitoring for intra-abdominal hypertension and abdominal compartment syndrome [27]. Accurate pressure transduction requires a continuous fluid column with a small volume of transducing medium [28]. High fluid volume infusion in the bladder has been shown to overdistend the bladder which leads to intrinsic muscular contraction and thus resulting in

falsely high readings of IAP [29]. This effect can be seen with volumes as low as 50ml [28]. Recent opinion has recommended a maximal infusion volume of 25ml though the most commonly quoted volume in literature before the World Society of Abdominal Compartment Syndrome (WSACS) consensus definitions was 50ml. At the 2004 International ACS Consensus Definitions Conference of the second World Congress on Abdominal Compartment Syndrome, the reference standard for intermittent IAP measurement was defined as via the bladder with a maximal instillation volume of 25ml sterile saline [8,9]. Intra-abdominal pressure was also recommended to be expressed in mmHg and measured at end-expiration in the complete supine position after ensuring complete relaxation of abdominal muscles with the transducer zeroed at the level of the midaxillary line [8]. These guidelines became necessary after the realization that early IAH studies utilized water manometers to determine IAP reporting results in cmH_2O [5,8,9,30,31,], while subsequent studies using electronic pressure transducers reported IAP in mmHg leading to difficulties and confusion in comparing results. The pressure relationship between water manometers in cmH_2O and electronic transducer measurements in mmHg is 1mmHg = 1.36cmH_2O [8,16]. Confusion had also been on zero reference point for the abdomen with different investigators using the symphysis pubis, the phlebostatic axis and the midaxillary line, each of which may result in different IAP measurements within the same subject [30]. The current recommendation is to use the mid-axillary line at the level of the anterior superior iliac spine in a completely supine patient.

2.4 Normal and pathological IAP measurements

Normal values may range from 2mmHg to 10mmHg in the critically ill adults [8]. However, some physiological conditions such as obesity or gravid uterus may be associated with chronic IAP elevations of 10-15mmHg to which the patient adapts with no pathological effect [33]. Thus, the clinical importance of IAP should be viewed in line with the baseline steady-state IAP for the individual patient. In the critically ill, IAP is frequently elevated above the patient's baseline value and there are a number of risk factors for IAH/ACS. A sustained increase in IAP reflecting new pathological phenomenon within the abdominal cavity should be the hallmark of a diagnosis of IAH [34].

Table 1: Risk factors for IAH/ACS[8,9]

Acidosis (pH<7.2)
Hypothermia (core temperature <33^{o}C)
Poly-transfusion (>10Units packed red blood cells/24h)
Coagulopathy (platelets <55,000/mm^{3} *or* APTT 2xNormal *or* higher *or* PT >50% *or* INR > 1.5)
Sepsis (American-European Consensus definitions)
Bacteremia
Intra-abdominal infection/abscess
Peritonitis
Liver dysfunction/cirrhosis with ascites
Mechanical ventilation
Use of positive end expiratory pressure (PEEP) or the presence of auto-PEEP e.g. Emphysema, chronic obstructive pulmonary disease
Pneumonia
Abdominal surgery, especially with tight fascial closures
Massive fluid resuscitation (>5L colloid or crystalloid/24h)
Gastroparesis/gastric distension/ileus
Volvulus
Hemoperitoneum/pneumoperitoneum
Major burns
Major trauma
High body mass index (>30)
Intra-abdominal or retroperitoneal tumours
Prone position
Massive incisional hernia repair
Acute pancreatitis
Distended abdomen
Damage control laparotomy
Laparoscopy with excessive inflation pressures
Peritoneal dialysis

2.5 Intra-abdominal hypertension

The 2004 International Abdominal Compartment Syndrome Consensus Definitions Conference which was endorsed by the World Society of the Abdominal Compartment Syndrome defined intra-abdominal hypertension as a sustained or repeated pathological elevation in intra-abdominal pressure $\geq$12mmHg [8,9]. Pathological IAP is a continuum ranging from mild IAP elevations without clinically significant adverse effects to markedly high pressures [35]. High IAP may result in deleterious effects on renal, cardiac, and gastrointestinal function. It was through this realization that a number of grading systems for IAH were suggested over the past decades. Burch and colleagues defined an early grading system for IAH, in cmH_2O, by which to guide therapy [36].

Table 2: Burch and colleagues' original grading of IAH[36]

Grade I	**10-15cmH_2O**	**(7.5-11mmHg)**
Grade II	**15-25cmH_2O**	**(11-18mmHg)**
Grade III	**25-35 cmH_2O**	**(18-25mmHg)**
Grade IV	**>35 cmH_2O**	**(>25mmHg)**

Literature currently defines IAH variously between 12-25mmHg [8,9]. Sugrue et al. reported that even IAP of 10mmHg might induce organ system failure in some patients[27]. However, based upon current understanding of IAH/ACS, a modification of the original Burch et al. grading

system was instituted to stratify patients with elevated IAP and guide treatment. Four severity groups have been established on the basis of IAP values in mmHg with the threshold for IAH established as a value of 12mmHg or greater in a minimum of three standardized measurements taken four to six hours apart [8,9].

Table 3: Current IAH grading system [8]

Grade 0:	**IAP<12mmHg**
Grade I:	**IAP 12-15mmHg**
Grade II:	**IAP 16-20mmHg**
Grade III:	**IAP 21-25mmHg**
Grade IV:	**IAP >25mmHg**

Malbrain et al further sub classified IAH into four groups according to the duration of symptoms, i.e. hyperacute, acute, subacute, and chronic intra-abdominal hypertension [37]. Hyperacute IAH represents elevations of IAP for a few seconds or minutes as in sneezing, laughing or physical activity. Acute IAH develops over a period of hours and is the one associated with surgical patients. This form of IAH is commonly linked to rapid development of ACS. Subacute IAH is mostly seen in medical patients and is related to conditions such as liver dysfunction, ascites or pneumonia. It occurs over several days. In chronic IAH, pressure increase is over several months or years e.g. pregnancy, tumour of abdomen, chronic ambulatory peritoneal dialysis or chronic ascites.

Raised intra-abdominal pressure has been recognized as a harbinger of intra-abdominal mischief by many researchers. Its measurement however, is cheap, simple to perform, and reproducible. Measurement of intra-abdominal pressure may provide an objective bedside stimulus for surgeons to expedite diagnostic and therapeutic intervention of critically ill patients [1]. Patients admitted to ICU after emergency general surgery or trauma should have IAP measurements because about 40% of them will have grade II or greater IAH and this, if unchecked, may progress to ACS with its attendant complications [1]. An increase in pressures resulting in grade III or IV IAH would indicate a need for more urgent and definitive treatment to avert these complications.

2.6 Abdominal compartment syndrome

Abdominal compartment syndrome is defined in current literature by a triad of: (a) sustained IAP >20mmHg with or without an abdominal perfusion pressure of <60mmHg, which (b) adversely affects at least one end organ function or can cause serious wound complications, and in which (c) abdominal decompression has a beneficial effect [8,9]. It is subclassified into primary, secondary or recurrent ACS [35]. Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical decompression or interventional radiology. Secondary ACS is characterized by subacute or chronic IAH that develops from extra-abdominal causes related to conditions like sepsis, major burns, or massive fluid resuscitation. Recurrent ACS refers to the condition in which ACS redevelops following

previous surgical or medical treatment of primary or secondary ACS. Recurrent ACS is associated with significant morbidity and mortality [25,30,37].

Current literature on IAH/ACS suggests a syndrome that impacts on 30-50% of all surgical ICU patients [37]. However, the true prevalence of ACS has been difficult to ascertain from past studies because of varied definitions of both IAH and ACS. A multicenter point prevalence prospective study of IAH based on the current definition of IAH (i.e. ≥12mmHg) conducted in 24hrs in thirteen intensive care units from various centers found a prevalence of IAH to be 50.5% and that of ACS to be 8.2% [10].

ACS has been shown to be an independent predictor of multiple organ failure and has a mortality rate of 70-80% if untreated [27,38]

Despite the current evidence base on IAH/ACS, a significant proportion of intensivists and surgeons are unaware of methods of measuring IAP. Further, Kimball et al and Ravishankar et al in 2003 separately conducted surveys of intensivists in the United Kingdom and concluded that despite widespread awareness of IAH/ACS, many intensive care units never measure IAP [14,]

2.7 Organ dysfunction assessment

Raised intra-abdominal pressures may be associated with dysfunction of various organ systems which result in increase in morbidity or mortality in critically ill patients. A number of organ dysfunction prediction models have been described, including the APACHE (Acute Physiology and Chronic Health Evaluation), SAPS (simplified acute physiology score), and the SOFA (Sequential Organ Failure Assessment score) [39]. The SOFA score was developed by the European Society of Critical Care Medicine (ESCCM), in 1994, as a system for measuring the

status of various systems of a patient in ICU [40]. The assessment is of six different organ systems separately. A number of different variables and parameters are included in each system and a score is assigned to each ranging from 0-4. The total SOFA score is then calculated by adding up the individual system scores to a maximum of 24 points. Organ failure is defined as a SOFA organ subscore equal to or above 3 in this study [10].Table 4: The SOFA Score [39]

The Sequential Organ Failure Assessment (SOFA) Score*

	SOFA Score				
Variables	0	1	2	3	4
Respiratory Pao_2/Fio_2, mm Hg	>400	≤400	≤300	≤200†	≤100†
Coagulation Platelets $\times 10^3/\mu L$‡	>150	≤150	≤100	≤50	≤20
Liver Bilirubin, mg/dL‡	<1.2	1.2-1.9	2.0-5.9	6.0-11.9	>12.0
Cardiovascular Hypotension	No hypotension	Mean arterial pressure <70 mm Hg	Dop ≤5 or dob (any dose)§	Dop >5, epi ≤0.1, or norepi ≤0.1§	Dop >15, epi >0.1, or norepi >0.1§
Central nervous system Glasgow Coma Score Scale	15	13-14	10-12	6-9	<6
Renal Creatinine, mg/dL or urine output, mL/d‖	<1.2	1.2-1.9	2.0-3.4	3.5-4.9 or <500	>5.0 or <200

*Norepi indicates norepinephrine; Dob, dobutamine; Dop, dopamine; Epi, epinephrine; and Fio_2, fraction of inspired oxygen.
†Values are with respiratory support.
‡To convert bilirubin from mg/dL to µmol/L, multiply by 17.1.
§Adrenergic agents administered for at least 1 hour (doses given are in µg/kg per minute).
‖To convert creatinine from mg/dL to µmol/L, multiply by 88.4.

(Ferreira et al, JAMA 2001;286(14):1754-1758)

The score increases as organ dysfunction worsens, thus the assessment can be used as a measure of organ failure or dysfunction and be correlated to intra-abdominal hypertension or compartment syndrome to show the adverse effects of raised abdominal pressures.

3. METHODOLOGY

3.1 Study design:

This was a prospective cohort study of consecutive cases after abdominal surgery or trauma admitted to intensive care units at both Harare Central and Parirenyatwa Group of Hospitals in the period December 2010 to June 2011.

3.1.1 ***Setting:*** Harare Central and Parirenyatwa Group of Hospitals' intensive care units. The two hospitals, located in the capital city of Zimbabwe, Harare, serve as both central hospitals as well as teaching hospitals for the University of Zimbabwe's College of Health Sciences. Each has a five-bed adult intensive care unit dedicated to all medical disciplines.

3.1.2 ***Background:*** On average, about fifteen to twenty surgical patients are admitted to each unit per month with just over a quarter being abdominal surgical patients. The measurement of intra-abdominal pressure is not standard procedure with most surgeons and intensivists relying on palpation and abdominal girth measurements. This study was the first of its kind employing the use of an intra-vesical Foley's catheter to measure intra-abdominal pressure and monitor for intra-abdominal hypertension.

3.1.3 ***Ethical Approval:*** The permission and approval to perform this study was obtained from the Joint Research Ethics Committee of Parirenyatwa Group of Hospitals and the College of Health Sciences, University of Zimbabwe and the Medical Research Council of Zimbabwe.

3.1.4 ***Procedure:*** Recording of relevant preoperative, intra-operative and postoperative data was made using a pre-designed data collecting manual.

3.1.5 ***Measurement of IAP:*** Pressure measurements were made via an indwelling urinary catheter using a water manometer and a set of two stopcocks connected in series. The abdominal pressure was measured with the patient completely supine and the manometer zeroed in the mid-axillary line at the level of the iliac crest. A volume of 20ml saline was instilled into an initially empty bladder 30-60 seconds before measuring. An oscillation test was performed before each measurement to confirm patency of the fluid column from the bladder to the manometer. This was done by applying gentle pressure over the suprapubic region and observing if there was a corresponding rise in the fluid level of the manometer as pressure was applied. After a period of equilibration of about 60 seconds the pressure was read off the manometer and then the patient returned to initial nursing posture. The pressure measurements obtained in cmH_2O were then converted to mmHg using the following equation:

$$\mathbf{IAP_{(mmHg)} = IAP_{(cmH2O)}/1.36}$$

Patients noted to require intervention according to study parameters were highlighted to the responsible surgical team. Recommendations for non-surgical or surgical decompression as intervention were also made where necessary. However, whether recommendations were considered or not was left to the attending surgeon's preferences in managing the patient. Non-surgical treatment recommendations included use of sedation, neuromuscular blockade, nasogastric decompression, rectal enemas, use of

gastro-/colo-prokinetic agents, fluid restriction or use of diuretics. Surgical decompression was the recommended procedure for patients not responding to non-surgical treatments. Intra-abdominal pressure measurements were made at six hourly intervals from time of admission to intensive care unit until 24-hours after. Patients were then followed-up to day 30 to determine outcome of survival or death by any cause.

3.2 General Objectives:

The general objective of the study was to evaluate the prevalence and clinical significance of raised intra-abdominal pressure and abdominal compartment syndrome in patients who had undergone abdominal surgery and admitted to surgical intensive care units of teaching hospitals in Harare.

3.3 Specific Objectives:

These were outlined as follows:-

i. To prospectively assess the prevalence of raised intra-abdominal pressure in abdominal surgical patients admitted to our intensive care units

ii. To compare the mortality in those with increased IAP and those without increased IAP as defined by the grading system for intra-abdominal hypertension by the World Society of Abdominal Compartment Syndrome (WSACS) [8,9]

iii. To determine the prognostic value of IAP measurements in abdominal surgical patients admitted to intensive care units

iv. To determine the relationship of IAP to American Society of Anaesthesiologists (ASA) score of the cases

v. To determine relationship of IAP to measured specific organ function parameters according to the Sequential Organ Failure Assessment (SOFA) score

vi. To determine if raised intra-abdominal pressure has an effect on length of stay in ICU

3.4 Duration of the study:

Cases were accumulated consecutively over a period of seven months starting from December 2010 and followed up to 30 days post-operation.

3.5 Endpoints:

Primary endpoint

- Development of intra-abdominal hypertension

Secondary endpoints

- Morbidity as measured by:
  - Development of multiple organ failure using SOFA score
  - Requirement for medical or surgical treatment of IAH/ACS
- Mortality (all cause)

3.6 Sample size:

The study sample size was 38 patients. The required sample size for abdominal surgical ICU patients who develop IAH and those without IAH was determined as 36 based on the following assumptions:

- 5% significance level
- 80% power
- A 50% prevalence of IAH in abdominal surgical ICU patients [10]
- A 5% prevalence of ACS in abdominal surgical ICU patients with IAH [10]

3.7 Sampling method:

All consecutive patients above 12 years admitted to the intensive care units after elective or emergency abdominal surgery, who stayed intubated and on ventilation for more than 24hrs and met the inclusion criteria were recruited for the study.

3.8 Inclusion criteria:

- Any abdominal surgical patients admitted to intensive care unit
- Age equal to or above 12 years
- Ventilated patients
- ICU stay greater than 24 hours

3.9 Exclusion criteria:

- Patients under 12 years,
- non-abdominal surgical patients,
- those not on ventilation, and
- patients with contraindications to intravesical pressure measurements (pelvic fractures, haematuria, neurogenic bladder or patients who had surgery on the urinary bladder itself)

3.10 Data collection:

A data collection sheet, a copy of which is in appendix I, was used to record patient demographics, nature of abdominal surgery performed, intensive care monitoring parameters relevant to the study, calculated SOFA scores, and outcome at 30 days post admission for each patient.

3.11 Data analysis:

All data was analyzed using Statistical Package for Social Sciences (SPSS) 16.0.0 for Windows® (SPSS Inc., Chicago, IL, USA, 1989-2007) and displayed in tables, or graphs. Demographic measurements such as mean, standard deviation, median, mode and regression analysis were used to describe the data. A $p<0.05$ was considered significant.

4. RESULTS

4.1 Characteristics of study sample

Thirty eight consecutive patients were enrolled into the study. Twenty (53%) were females and eighteen (47%) were males. The mean age for the whole group was 37.29±16.18 years. Twenty nine patients (76%) were admitted to ICU after emergency abdominal surgical operation, with nine (24%) having had elective abdominal surgical operations (Table 5).

Table 5: Distribution of patients according to elective or emergency operation

	Frequency	Valid Percent
Elective Surgery	9	24
Emergency Surgery	29	76
Total	38	100

Table 6 summarizes the distribution of patients by reason for abdominal surgery. Majority of patients (32%, n=38) had abdominal surgery for bowel obstruction, with the least number having had surgery for abdominal trauma (4 patients)

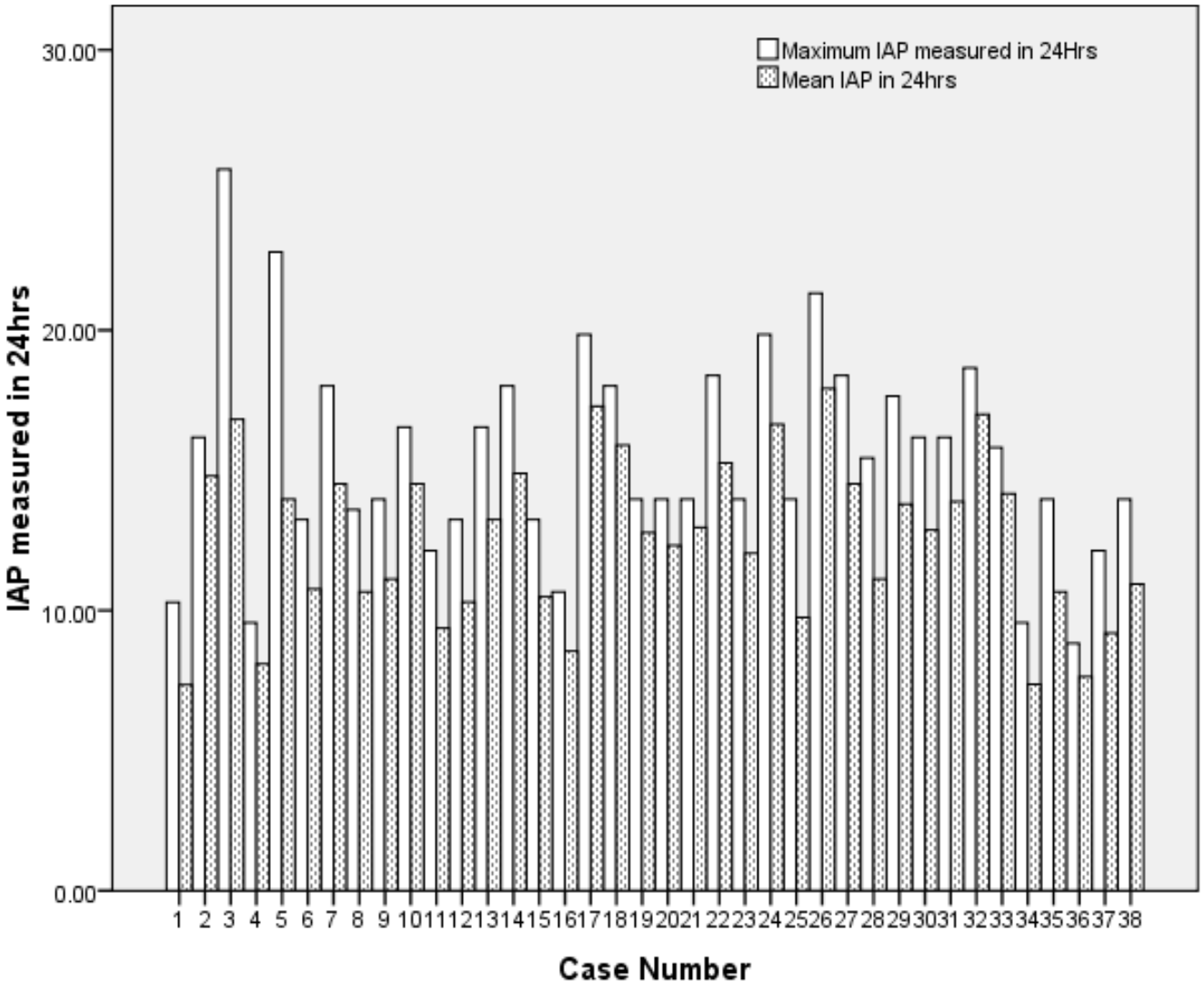
Table 6: Distribution of patients by reason for abdominal surgery

Operation	Frequency	Percent
Bowel Obstruction	12	32
Gynaecologic Operation	9	24
Peritonitis	8	21
Abdominal Trauma	4	10
Other	5	13
Total	38	100

4.2 Prevalence of intra-abdominal hypertension and abdominal compartment syndrome

The general distribution of intra-abdominal pressures for the thirty eight patients is as shown in Figure 1.

Figure 1: Distribution of intra-abdominal pressures by case



Patients were characterized according to mean of recorded intra-abdominal pressures (IAP_{mean}) as well as the maximum recorded intra-abdominal pressure (IAP_{max}) during the 24hrs of

admission (four measurements done 6 hours apart from time of admission for 24 hours) to describe the prevalence of intra-abdominal hypertension and its grade, or abdominal compartment syndrome. Figures 2 and 3 show percentage distribution of intra-abdominal pressure grades for the study population.

Figure 2: Distribution of Intra-abdominal pressure grades according to IAP_{mean}

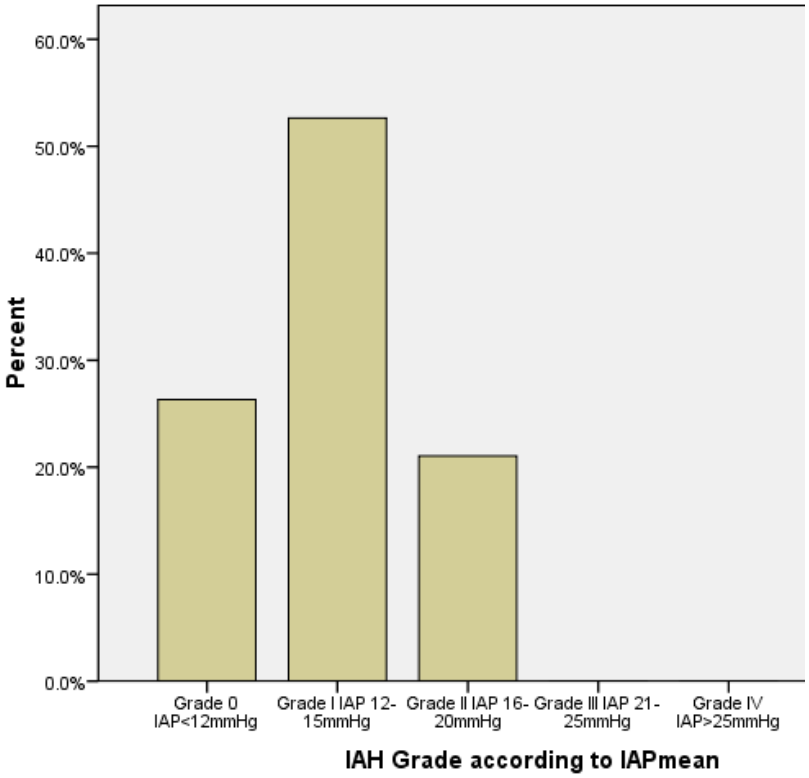
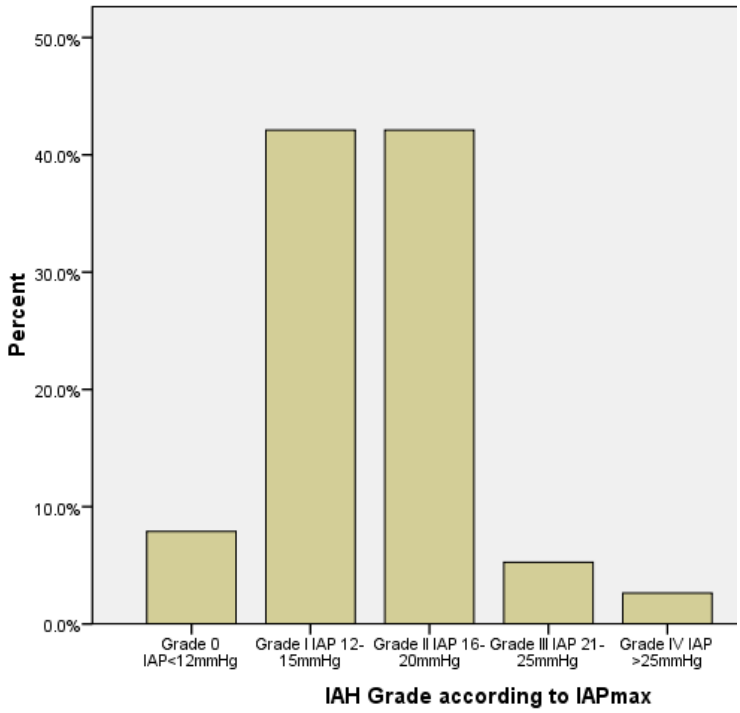


Figure 3: Distribution of intra-abdominal pressure grades according to IAP_{max}



The mean of maximum IAP measured in 24 hours (IAP_{max}) was 15.47mmHg$\pm$3.71mmHg and the mean of mean IAP (IAP_{mean}) was 12.69mmHg$\pm$3.08mmHg. When the IAP_{max} was considered, thirty five patients had intra-abdominal pressures of grade II or worse (IAP $\leq$20mmHg) with only one patient having pressures above 25mmHg (Grade IV intra-abdominal hypertension). When the mean intra-abdominal pressures (IAP_{mean}) for each patient were reviewed, results showed all patients had mean intra-abdominal pressures $\leq$20mmHg (grade II intra-abdominal hypertension or lower). The prevalence of IAH (defined as IAP>12mmHg) was 74% (28 patients) and none of the patients had abdominal compartment syndrome (defined as sustained IAP>20mmHg) when IAP_{mean} was used. However, when IAP_{max} was used, the prevalence of IAH

was 92% (35 patients) with three patients developing abdominal compartment syndrome. The prevalence of IAH differed in relation to whether mean or maximal IAP values were used and IAP_{max} seemed to define IAH of higher grades for the patients than IAP_{mean}.

Tables 7 and 8 below summarise results of IAH grading for the cases according to mean pressures in 24 hours or maximum recorded pressure during the 24 hours of the study.

Table 7: IAH according to IAP_{max}

	Frequency	Valid Percent	Cumulative Percent
Grade 0 IAP<12mmHg	3	8	8
Grade I IAP 12-15mmHg	16	42	50
Grade II IAP 16-20mmHg	16	42	92
Grade III IAP 21-25mmHg	2	5	97
Grade IV IAP >25mmHg	1	3	100
Total	38	100	

Table 8: IAH according to IAP_{mean}

	Frequency	Valid Percent	Cumulative Percent
Grade 0 IAP<12mmHg	10	26	26
Grade I IAP 12-15mmHg	20	53	79
Grade II IAP 16-20mmHg	8	21	100
Total	38	100	

With a test value of 12mmHg, a one sample t-test on the mean of intra-abdominal pressure measurements for the cases (IAP_{mean}) showed that there was no significant difference of the mean values (p=0.177). A similar test was performed for the maximum recorded pressures for each patient and showed a significant difference with values skewed above 12mmHg (p=0.00).

4.3 The effect of IAH on outcome

Outcome of survival or death was assessed at 30 days post admission to ICU and related to IAP. Eight patients (21%) had died by the end of 30 days. The mean of IAP_{max} for those who died was 18$\pm$2.77mmHg with that of survivors at 14.79$\pm$3.67mmHg and an independent samples T-test showed that the intra-abdominal pressures of the survivors and non-survivors significantly differed (p=0.017). When the IAP_{mean} was considered, non-survivors had a mean of 14.57$\pm$2.02mmHg while survivors had a mean of 12.19$\pm$3.14mmHg with an independent samples T-test also showing significantly different pressures for both survivors and non-survivors(p=0.019). The graphs below show the mean and variation of IAP_{max} and IAP_{mean} for both survivors and non-survivors.

Figure 4: Outcome at 30 days in relation to mean IAP_{max}

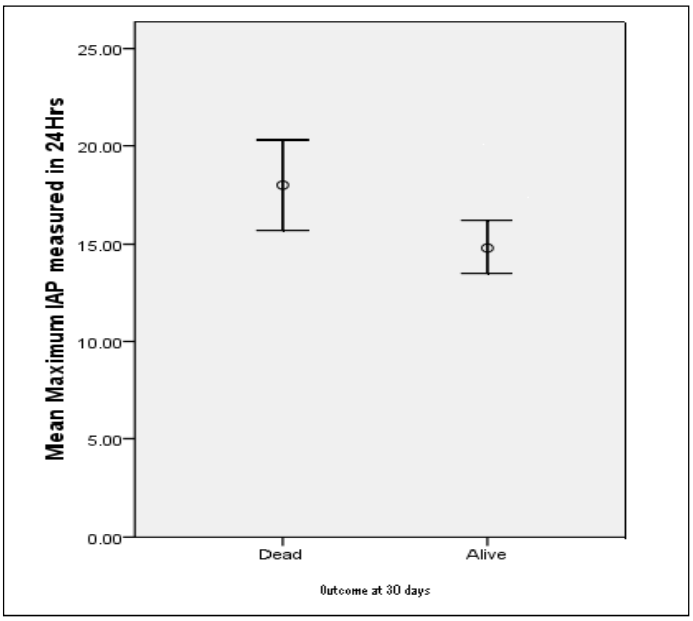
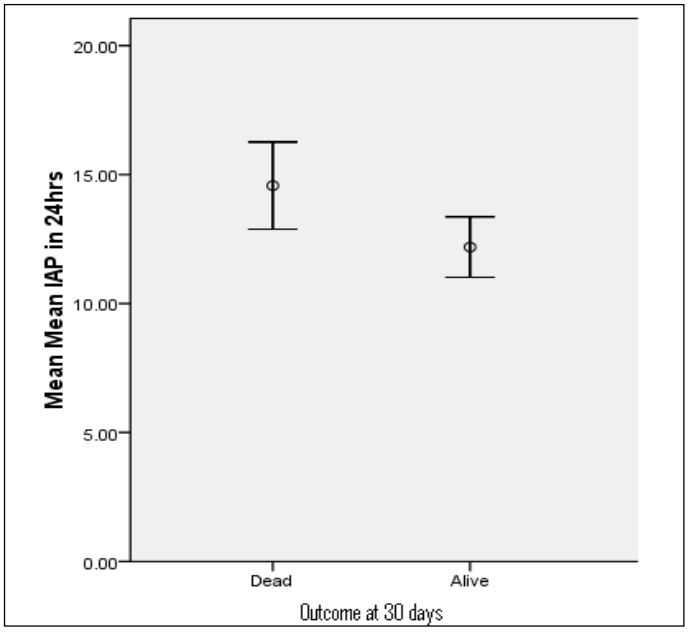


Figure 5: Outcome at 30-days in relation to mean IAP_{mean}



The distribution of IAH grades for the survivors and non-survivors is shown in Table 9. Only one patient among non-survivors had pressures ≥21mmHg if maximum recorded IAP for each case are used with majority (6 patients) having intra-abdominal pressures in the grade II category.

Mean intra-abdominal pressure measurements show majority of non-survivors to be in IAH grade I (12-15mmHg), with none having mean pressure recordings beyond 21mmHg.

Table 9: IAH Grade in relation to outcome at 30 days

		Outcome at 30 days post-op			
		Dead		**Alive**	
		Count	Column N %	Count	Column N %
IAH Grade according to IAP_{max}	Grade 0 IAP<12mmHg	0	0.00%	3	10.00%
	Grade I IAP 12-15mmHg	1	12.50%	15	50.00%
	Grade II IAP 16-20mmHg	6	75.00%	10	33.30%
	Grade III IAP 21-25mmHg	1	12.50%	1	3.30%
	Grade IV IAP >25mmHg	0	0.00%	1	3.30%
IAH Grade according to IAP_{mean}	Grade 0 IAP<12mmHg	0	0.00%	10	33.30%
	Grade I IAP 12-15mmHg	5	62.50%	15	50.00%
	Grade II IAP 16-20mmHg	3	37.50%	5	16.70%
	Grade III IAP 21-25mmHg	0	0.00%	0	0.00%
	Grade IV IAP>25mmHg	0	0.00%	0	0.00%

These results show that hundred percent of non-survivors had intra-abdominal hypertension (pressures >12mmHg) whether IAP_{mean} or IAP_{max} is used to define intra-abdominal pressures. Using IAP_{max} about 13% of non-survivors develop abdominal compartment syndrome. Only about 3% of survivors had abdominal compartment syndrome.

4.4 The effect of IAH on organ function

Organ function was assessed using the Sequential organ failure assessment (SOFA) score which looks at respiratory, cardiovascular, hepatic, haematological, renal, and neurological systems. The minimum score is zero and the maximum is 4 for each organ system. Organ failure was defined as a SOFA organ sub-score of equal to or above 3. The statistics for the SOFA score for the different organ systems are as shown in table 10.

Table 10: Statistics of organ SOFA scores

		Respiratory SOFA Score	CNS SOFA Score	CVS SOFA Score	Coagulation SOFA Score	Renal SOFA Score	Hepatic SOFA Score
N	Valid	38	38	38	38	38	11
	Missing	0	0	0	0	0	27
Mean		1.7	2.6	1.3	0.4	0.6	1.3
Std. Error of Mean		0.18	0.13	0.27	0.13	0.13	0.41
Mode		2	2	0	0	0	0

The respiratory, cardiovascular, and neurological systems seem to have had the worst scores. Thirty six percent of patients had cardiovascular system failure in this study as defined by the SOFA score. The distribution of the SOFA scores for each organ is as shown in table 11.

Table 11: Distribution of SOFA Scores for each Organ system

Organ system	SOFA Score					
	0	1	2	3	4	% with organ failure
Cardiovascular (% of n=38)	60.5	2.6	0	23.7	13.2	37
Neurological (% of n=38)	0	0	65.8	13.2	21.1	34
Respiratory (% of n=38)	23.7	7.9	39.5	28.9	0	29
Hepatic (% of n=11)	36.4	27.3	18.2	9.1	9.1	18
Coagulation (% of n=38)	78.9	7.9	10.5	2.6	0	3
Renal (% of n=38)	57.9	21.1	21.1	0	0	0

Only eleven out of the 38 cases had a full SOFA score computed because of unavailable liver function results for the other cases. To try and have a representative SOFA score for all cases, the liver score was excluded in all patients and a modified score out of 20 points was calculated. The relationship between SOFA score excluding liver and IAP_{max} or IAP_{mean} was examined. A

Pearson correlation procedure was performed and results indicated that the relationship between IAP_{max} and SOFA excluding liver score was positive (coefficient =0.290) but not significant (p=0.077, n=38). When the same procedure was done for IAP_{mean} and SOFA excluding liver score, the relationship was again positive but stronger and significant (coefficient =0.43, p=0.007, n=38). We then did a linear regression analysis to establish a cause effect relationship between IAP_{mean} and SOFA excluding liver score and the results indicated that IAP_{mean} variation significantly led to organ status change. High levels of IAP_{mean} led to organ failure with a unit change in the mean value of IAP leading to a 0.416 increase in the SOFA (excluding liver) score.

4.5 The relationship of ICU stay and intra-abdominal pressure

Length of stay was defined as the number of days from admission to ICU up to day of discharge from ICU either alive or dead. The statistics of length of ICU stay was as shown in Table 7. About 80% of patients stayed less than or equal to 5 days in ICU with the modal stay being 2 days.

Table 6: Statistics of length of stay in ICU (in days)

	Length of stay in ICU(days)
N	38
	0
Mean	3.7
Std. Error of Mean	0.4
Median	3
Mode	2
Std. Deviation	2.6
Minimum	1
Maximum	12
Sum	140

A Pearson correlation procedure failed to show a relationship between IAPmean and ICU stay (coefficient 0.012, p=0.945, n=38). Similar results were noted when IAP_{max} and ICU stay were analysed (coefficient 0.119, p=0.477, n=38). A patient's intra-abdominal pressure does not seem to determine his/her length of stay in ICU.

4.6 The American Society of Anaesthesiologists (ASA) score and IAP values

The most commonly used physiologic scoring system in our unit is the ASA score done pre-operatively by anaesthesiologists. In this study, we sought to find out if there was any relationship between this score and intra-abdominal pressures. All 38 patients had ASA scores recorded. Majority of patients were clustered around scores ASA II and III, with only 23% having an ASA IV.

Table 12: Distribution of ASA scores

	ASA Score	Frequency	Valid Percent	Cumulative Percent
I	Normally Health	1	3	3
II	Mild systemic disease	15	39	42
III	Severe systemic disease that limits activity, not incapacitating	13	34	76
IV	Incapacitating systemic disease which poses a threat to life	9	24	100

Both the mean IAP and maximum IAP failed to show a significant relationship with ASA score when a Pearson correlation procedure was performed (for IAP_{mean}, coefficient 0.011, p=0.948; for IAP_{max}, coefficient 0.072, p=0.666). It seems, according to this study, we cannot use ASA score as a proxy for IAP measurements.

5. DISCUSSION:

The major findings of this study of intra-abdominal hypertension and abdominal compartment syndrome are the demonstration of the occurrence of IAH and ACS in our abdominal surgical patients who are admitted to intensive care units, how IAH affects organ function, length of ICU stay, and the outcome after 30 days from day of admission.

5.1 Prevalence of Intra-abdominal Hypertension

In this study, as expected from literature, the prevalence of IAH differed in relation to whether mean or maximal IAP values were used to define intra-abdominal hypertension. Maximal IAP measurements showed a prevalence of IAH of 92% with ACS of 8% while mean IAP values resulted in a lower prevalence of 74% with none of the cases developing ACS. This reflects very well a fact noted by Malbrain et al [41] that mean IAP values seem to down grade intra-abdominal pressure measurements and may result in some cases of IAH or ACS being missed. The accuracy of mean IAP values could be improved by increasing the frequency of IAP measurement per day, which approximates to continuous monitoring. In the absence of fully automated abdominal pressure monitoring devices, the increase in workload makes it unsustainable. While maximal IAP values may over-diagnose some patients, the overall result of it is positive in terms of IAH diagnosis and prognosis. Most published papers have resorted to using maximal IAP values in prognosticating and diagnosing IAH as it seems to be the most physiologically sensible option over mean or modal values. In a multicentre epidemiological study on the prevalence of IAH and ACS in 2003, the prevalence of IAH was 50.5% and that of ACS 8.2% using maximal measured IAP values [10]. Although our study shows a much higher prevalence for IAH, the prevalence for ACS, which by definition is physiologically the worst form of IAH, are comparable. Previously published reports quoted prevalence rates of IAH ranging from as low as 18% to as high as 81%, and 2% to 36% for ACS. However, these figures were before the standardization and definition of terms by the WSACS during the 2004

International ACS Consensus Definitions Conference. Even then, our figures seem to be much more than those reported in literature, an issue which needs to be pursued. One explanation may be related to the population of patients we are likely to be admitting to our units. Because of shortage of ICU beds, perhaps we admit a group of patients who are more ill and therefore are more likely to have raised intra-abdominal pressures anyway than those admitted to units with bigger ICUs. Another explanation might be related to non-use of paralysing agents in our ventilated ICU patients resulting in spuriously high IAP values. Larger studies incorporating the current understanding and definitions in IAH/ACS are needed to come up with reference prevalence rates particularly in our setting. Table 13 shows previously reported IAH/ACS prevalence figures from literature, majority of studies done before standardization or in varied patient populations which included trauma patients, medical cases and abdominal surgical patients.

Table 13: Previously reported IAH/ACS prevalences

	Reference	Number of patients in the study	Type of study	Incidence %
IAH	Sugrue et al, 1995[42]	88	Prospective	33
	Sugrue et al,1996[43]	73	Prospective	38
	Sugrue et al, 1998[44]	49	Prospective	81
	Ivatury et al, 1998[45]	70	Retrospective	32
	Sugrue et al, 1999[46]	263	Prospective	40
	Biancofiore et al, 2003[47]	108	Prospective	32
ACS	Fietsam et al, 1989[2]	104	Prospective	4
	Morris et al, 1993[48]	107	Retrospective	15
	Meldrun et al, 1997[49]	145	Prospective	14
	Ertel et al, 2000[50]	311	Prospective	5
	Raeburn et al, 2001[51]	77	Prospective	36

5.2 Intra-abdominal hypertension, organ function and outcome at 30 days

Multiple experimental and clinical reports have demonstrated that the presence of IAH affects several abdominal and extra-abdominal organs leading to organ failure and eventually death, sometimes in as

many as 80% of patients[1,2,7,10,17,38]. Current literature on IAH/ACS describes a syndrome that affects 30-50% of all ICU patients, independently predicting multiple organ failure and having a mortality rate of 70-80% if allowed to progress untreated[10]. Elevated IAP induces intra-abdominal organ hypoperfusion leading to multiple organ dysfunction syndrome mediated by inflammatory responses [13]. Our study had a mortality of 21%. The mean IAP_{max} of non-survivors was 18$\pm$2.77mmHg and that of survivors was 14.79$\pm$3.67mmHg. These pressures were significant with a p=0.017 using an independent samples T-test. The fact that non-survivors had higher pressures than survivors points to the presence of an association between raised intra-abdominal pressure and survival outcome. Although IAH is graded into categories with pressures above 20mmHg being more associated with organ dysfunction and mortality, various organ systems are compromised at different pressure thresholds. Polat et al[52] reported intestinal bacterial translocation at IAP $\geq$14mmHg while other authors described pressures >20mmHg leading to translocation. As low as IAP 15mmHg has been found to lead to acute kidney injury in patients developing intra-abdominal hypertension. A prospective multicentre study of 265 mixed ICU patients in 14 ICUs of six countries demonstrated IAH as an independent risk factor for mortality[53]. Almost all organ systems can be affected by IAH including the neurological system. Neurological effects of raised IAP are characterized by raised intra-cranial pressures and decreased cerebral perfusion. These neurological changes are more difficult to appreciate in a ventilated and sedated patient population making IAP monitoring a useful and easier option. The decrease in venous return as IAP rises compromises ventricular filling and eventually cardiac output in these already critically sick patients. IAH also leads to respiratory failure through the direct effect of diplacing the diaphragm into the thoracic cavity. Peak airway pressures have been shown to increase significantly with associated development of compressive atelectasis, decreased lung compliance, barotrauma and reduction in lung volumes. The overall effect is disruption of gas exchange, development of hypoxaemia, hypercapnia and acidosis which in themselves are associated with high morbidity and mortality[13].

The sequential organ failure assessment (SOFA) score was used to assess organ dysfunction in this study because of its simplicity and ease of use compared to other physiologic scoring systems like the APACHE II or III which require better resourced laboratory back-up. Thirty six percent of patients had cardiovascular system failure with neurological and respiratory dysfunction affecting 34% and 29%, respectively. These results are comparable to findings in a reported study of 97 critically ill patients with IAH/ACS who demonstrated mostly respiratory, cardiovascular and neurological organ failure[41]. In the same study, at least one patient had either liver, haematological or renal dysfunction as defined by the SOFA sub-scores. Only eleven patients had complete hepatic function scores in our study, reflecting the limitations of incomplete or unavailable investigations.

No study has directly investigated the relationship between IAH and length of ICU stay with the majority of comments being extrapolations from the known increase in morbidity and mortality in patients with abdominal hypertension. In this study we sought to find out if IAH affected length of ICU stay. The majority of patients stayed for $\leq$5days with a modal stay of 2 days. The mean stay of non-survivors and survivors was 3.12 days and 3.83 days, respectively. These results were not significant (p=0.477). Perhaps further studies with larger numbers are needed to characterize the relationship but in this study, a patient's IAP does not seem to determine their length of stay in ICU.

Many opinion leaders in the management of IAH/ACS have strongly recommended that all critically ill patients should have IAP closely monitored and clinical examination of the abdomen has been shown to be grossly inaccurate in the assessment of IAH/ACS[8,9]. The use of bladder pressure measurement as a proxy of IAP for early detection of IAH has been widely demonstrated to be effective but the medical community's response seems to be slow and sceptical. In an attempt to find a parameter that may act as an early warning sign to prompt the need for measurement of IAP in abdominal surgical patients admitted to ICU in places where it's not standard practice, this study tried to find whether there was any

relationship between IAH and American Society of Anaesthesiologists' score (ASA), a commonly used pre-operative scoring system done on every patient. Results failed to show a significant relationship between IAP and ASA score. The ASA score is both less sensitive and not predictive of the development of IAH. Its main advantage is in its simplicity in grading but lacks objective measurable parameters which are specific and sensitive. The best way to find out if a patient has IAH/ACS is by measuring the IAP.

6. CONCLUSION:

1. This study has shown that the problem of intra-abdominal hypertension in abdominal surgical patients admitted to our intensive care units has a prevalence of up to 92% with about 8% developing abdominal compartment syndrome when maximal intra-abdominal pressures are considered.

2. Intra-abdominal hypertension is significantly linked to patient outcome in this study. Twenty one percent of patients died and 100% of the non-survivors had intra-abdominal hypertension, with 13% having developed abdominal compartment syndrome.

3. Of the patients who developed intra-abdominal hypertension, about 6% developed abdominal compartment syndrome according to maximal intra-abdominal pressures.

4. The study failed to show a significant relationship between intra-abdominal hypertension and the ASA score.

5. The study has also highlighted the adverse effects of raised intra-abdominal pressure on organ function, although some patients had incomplete data on liver function.. The worst affected organ systems were respiratory (29% of patients), neurological (34% of patients) and cardiovascular (37% of patients) as defined by the sequential organ failure

assessment score. This is an addition to already available evidence from global literature but most importantly documents our own experience.

6. According to the findings in this study, a patient's intra-abdominal pressure does not seem to determine his/her length of stay in ICU.

7. The method used for measuring IAP has a number of characteristics which makes it applicable even in a resource limited environment like ours. Although not a direct objective of this study, with the numbers recruited, it appears the method is simple, open to repeated measurements with no risk of injury, no manipulation, or risk of urinary tract infection (because of its closed-system nature).

IAH is a reality confronting patients and need to be addressed with attention if they are to be saved from its effects. IAP should be incorporated into the routine management of ICU patients.

7. RECOMMENDATIONS

Armed with findings in this study, the following recommendations are made:

1. The occurrence of intra-abdominal hypertension and the risk factors for it should be appreciated by both surgeons and intensivists caring for patients after abdominal surgical operations

2. Intra-abdominal pressure measurements should be considered in abdominal surgical patients

3. The bladder method is feasible and cheap for a resource constrained environment and could be adopted as a method of objectively measuring intra-abdominal pressure in addition to clinical judgement.

4. Intervention and treatment guidelines should be formulated for the management of patients who are found to have intra-abdominal hypertension or abdominal compartment syndrome.

5. A scoring system for organ function assessment such as the SOFA score used in this study should be adopted to help in categorizing the extend of organ injury in relation to IAH

6. A larger study not only of abdominal surgical patients but those also at risk of developing intra-abdominal hypertension and/or abdominal compartment syndrome should be conducted.

7. A future study incorporating pre-operative pressure measurements may help characterize the differences before and after operation

8. A future study involving planned intervention strategies for either prevention or treatment of intra-abdominal hypertension

8. LIMITATIONS OF THE STUDY

The limitations of this study were as follows:

1. Use of a manual method of measuring intra-abdominal pressures at intervals which might introduce errors of measurement and is better addressed with an automated electronic transducer with continuous pressure recordings.
2. One of the requirements of correct pressure measurements is for patients to be well sedated or even paralysed to prevent spurious intra-abdominal hypertension. The policy of the ICU was to only sedate with none of the patients paralysed and this might have affected measurements. However, it's not recommended to routinely paralyse patients in ICU (Level 1 evidence) because of the side effects of doing so.
3. Pressure measurements were done through a distensible intra-vesical latex Foley's catheter which could have over dampened readings. Although literature seems to have no studies on this, a rigid silastic catheter might have been more appropriate.
4. Although it's known that some patients may pre-operatively present with significantly high pressures, intra-abdominal pressure measurements were only done post-operatively in this study which fails to capture the contribution of these pre-operative derrangements to outcome or organ function.
5. Because of limited personnel for this study, the period of pressure measurements was limited to initial 24 hours only. Intra-abdominal pressure changes and their effects may go beyond this and could have been missed.
6. The findings of this study are limited to patients after abdominal surgical operation. Studies have shown that the problem of IAH/ACS affects other critically ill patient populations significantly as well and these were not included.

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10. APPENDIX 1

Data Collection Sheet: Abdominal compartment syndrome in abdominal surgical patients admitted to intensive care units of Harare teaching hospitals – incidence and clinical significance

Date: _____ Hospital No:_____ Contact Tel No:_____

(1)Age:_____

(2)Sex:

M		F

Admission date:_____

Discharge date:_____

(3)Diagnosis:

A	Abdominal trauma
B	Bowel obstruction
C	Peritonitis
D	Gynaecologic operation
E	Other

Reason for ICU admission:_____

(4)ASA score:_____ (5)

Elective[EL]	Emergency[EM]

(6)Total Intraoperative IV fluid volume:_____

Time	***6hrs post admission***	***12hrs post admission***	***18hrs post admission***	***24hrs post admission***	***Average ……***
Heart rate	(7)	(8)	(9)	(10)	(11)
MAP	(12)	(13)	(14)	(15)	(16)
CVP (cmH_2O)	(17)	(18)	(19)	(20)	(21)
Ionotropes	(22)Y/N	(23)Y/N	(24)Y/N	(25)Y/N	
VENTILATION MODE [A] VCV [B]PCV [C]SIMV-PC	[A] (26) [B] [C] [D] [E]	[A] (27) [B] [C] [D] [E]	[A] (28) [B] [C] [D] [E]	[A] (29) [B] [C] [D] [E]	

[D]SIMV-VC [E]CPAP/PSV					
FiO_2	(30)	(31)	(32)	(33)	
SpO_2	(34)	(35)	(36)	(37)	(38)
PEEP	(39)	(40)	(41)	(42)	(43)
Glasgow coma scale	(44)	(45)	(46)	(47)	(48)
IAP (intra-abdominal pressure)/ cmH_2O	(49)	(50)	(51)	(52)	(53)
APP	(54)	(55)	(56)	(57)	(58)
Urine output	(59)	(60)	(61)	(62)	(63)

Sequential Organ Failure Assessment Score

Resp System[PaO2/FiO2 (mmHg)] ……. Score: ___

Nervous System(GCS) …… Score: ___

Cardiovascular system (MAP or inotropes) …… Score: ___

Liver (bilirubin, mg/dl) …… Score: ___

Coagulation (platelet count $x10^3$/ml) …… Score: ___

Renal system(creatinine or urine output) …… Score: ___

(64)Total SOFA score: ___

(65)Length of stay in ICU(days): _____

(66)Outcome at 30days: Alive Dead

11 APPENDIX II

Relevant Definitions:

Intra-abdominal hypertension and abdominal compartment syndrome were defined according to the World Society of Abdominal Compartment Syndrome recommendations. Abdominal perfusion pressure was calculated as the difference between the measured mean arterial pressure and the intra-abdominal pressure. Abdominal compartment syndrome was defined as a sustained IAP of more than 20mmHg associated with at least one organ failure with or without an abdominal perfusion pressure less than 60mmHg. The levels of intra-abdominal hypertension were graded as follows: grade 0, IAP <12mmHg; grade I, IAP 12-15mmHg; grade II, IAP 16-20mmHg; grade III, 21-25mmHg; and grade IV, IAP >25mmHg. The Sequential Organ Failure Assessment score (SOFA) was used to assess organ dysfunction as in Table 4 (adopted from Ferreira et al, JAMA 2001).