OPTIMIZATION OF INTRA ABDOMINAL PRESSURE DURING PERIOPERATIVE PERIOD AND ITS EFFECT ON OUTCOME IN PATIENTS UNDERGOING LAPAROTOMIES IN A TERTIARY CARE SETTING – AN EXPERIMENTAL STUDY

M.S. DEGREE EXAMINATION

BRANCH I - GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY
MADURAI MEDICAL COLLEGE AND GOVT RAJAJI HOSPITAL
Madurai – 625 020

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA.

By

Dr. ASHWIN R.

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**OPTIMIZATION OF INTRA ABDOMINAL PRESSURE DURING PERIOPERATIVE PERIOD AND ITS EFFECT ON OUTCOME IN PATIENTS UNDERGOING LAPAROTOMIES IN A TERTIARY CARE SETTING – AN EXPERIMENTAL STUDY**" submitted by **Dr. R. ASHWIN** to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch I (General Surgery), is a bonafide research work carried out by him under my direct supervision & guidance.

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CERTIFICATE BY THE DEAN

This is to certify that the dissertation entitled "**OPTIMIZATION OF INTRA ABDOMINAL PRESSURE DURING PERIOPERATIVE PERIOD AND ITS EFFECT ON OUTCOME IN PATIENTS UNDERGOING LAPAROTOMIES IN A TERTIARY CARE SETTING – AN EXPERIMENTAL STUDY**" is a bonafide research work done by **DR. R. ASHWIN,** Post Graduate Student, Department of General Surgery, Madurai Medical College and Government Rajaji Hospital, Madurai, under the guidance and supervision of **Prof. DR. MARUTHUPANDIAN, M.S, FICS., FIAS**, Professor and Head of Department of General Surgery, Madurai Medical College and Government Rajaji Hospital, Madurai.

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Dean

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DECLARATION

I, **Dr. R. ASHWIN** declare that, I carried out this work on "**OPTIMIZATION OF INTRA ABDOMINAL PRESSURE DURING PERIOPERATIVE PERIOD AND ITS EFFECT ON OUTCOME IN PATIENTS UNDERGOING LAPAROTOMIES IN A TERTIARY CARE SETTING – AN EXPERIMENTAL STUDY**" at the Department of General Surgery, Govt. Rajaji Hospital during the period of September 2015 to August 2016. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree and diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S. degree examination in General Surgery.

Place: Madurai

Date: **DR. R. ASHWIN**

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My patients, who form the most integral part of the work, were always kind and cooperative. I pray to God give them courage and strength to endure their illness, hope all of them go into complete remission.

And Last but not the least, I would like to thank my parents and The Almighty for being with me throughout the study providing moral support.

LIST OF ABBREVIATIONS

ADH	Anti Diuretic Hormone
APACHE - II	Acute Physiology And Chronic Health Evaluation II
APP	Abdominal Perfusion Pressure
ARDS	Acute Respiratory Distress Syndrome
CVP	Central Venous Pressure
EVLWI	Extra Vascular Lung Volume Index
FG	Filtration Gradient
GEDV	Global End Diastolic Volume
ICH	Intra Cranial Hypertension
ICP	Intra Cranial Pressure
ITP	Intra Thoracic Pressure
MAP	Mean Arterial Pressure
MODS	Multi Organ Dysfunction Syndrome
PAOP	Pulmonary Artery Occlusion Pressure
PEEP	Positive End Expiratory Pressure
PPV	Pulse Pressure Variation
RVEDVI	Right Ventricular End Diastolic Volume Index
SOFA	Sequential Organ Failure Assessment
SVV	Stroke Volume Variation
UTI	Urinary Tract Infection

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INTRODUCTION

In the field of surgery, severe sepsis, sepsis coexisting with hypotension, surgery for trauma and emergency surgery still carries significant mortality and morbidity despite best clinical efforts and perioperative care. As of now multitude of research has been conducted to understand the interplay of various factors and their contribution with the changes in inflammation and the pathogenesis of organ dysfunction and damage in the above-mentioned situations. Though in the current era of molecular and genetic science many etiological mechanisms and pathways for organ dysfunction has been postulated, it is still a grey area that has to be better understood. One such highly speculated factor involved in the pathway of organ dysfunction and damage in critically ill patients is “Intra Abdominal Pressure”.

“INTRA- ABDOMINAL PRESSURE (IAP)”

“IAP is the steady state pressure concealed within the abdominal cavity”. Increased attention is being given to “Intra Abdominal Pressure”

measurement in the critical ill patients as it can have significant changes in the management.

“Normally, IAP is around 5 – 7 mmHg in critically ill patients”.

Grades of IAP

“IAP is graded as follows:

Grade I	12–15 mmHg
Grade II	16–20 mmHg
Grade III	21–25 mmHg
Grade IV	> 25 mmHg”

Intra Abdominal Hypertension (IAH): “IAH is defined by a sustained or repeated pathological elevation in IAP ≥ 12 mmHg”

Abdominal Compartment Syndrome (ACS): "ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure"

"IAP measurement is usually recommended when any known risk factor for IAH/ ACS are present in a critically ill or injured patient".

AIMS AND OBJECTIVES :

- To determine the incidence of perioperative increased intra abdominal pressure among elective and emergency laparotomies at GRH, Madurai.

- Optimization of IAP, if found to be elevated, using WCACS protocols.

- To correlate whether optimization of perioperative IAP has a positive effect in outcome in the abovementioned cases.

REVIEW OF LITERATURE

SURGICAL ANATOMY OF PERITONEUM AND PERITONEAL CAVITY

EMBRYOLOGY OF PERITONEAL CAVITY

Peritoneal cavity develops from the two limbs of the horseshoe shaped intra-embryonic coelom, which is located caudal to septum transversus. The two parts are at first separate, but fuse to form one cavity as result of lateral folding of embryonic disc. The attachment of mesentery of the primitive gut on the abdominal wall is initially in the midline. As a result of changes involving the rotation of the gut and as a result of some parts of the gut becoming retroperitoneal, the line of attachment of mesentery becomes complicated .The peritoneal cavity therefore comes to be subdivided into number of pockets that are separated partially by folds of peritoneum.

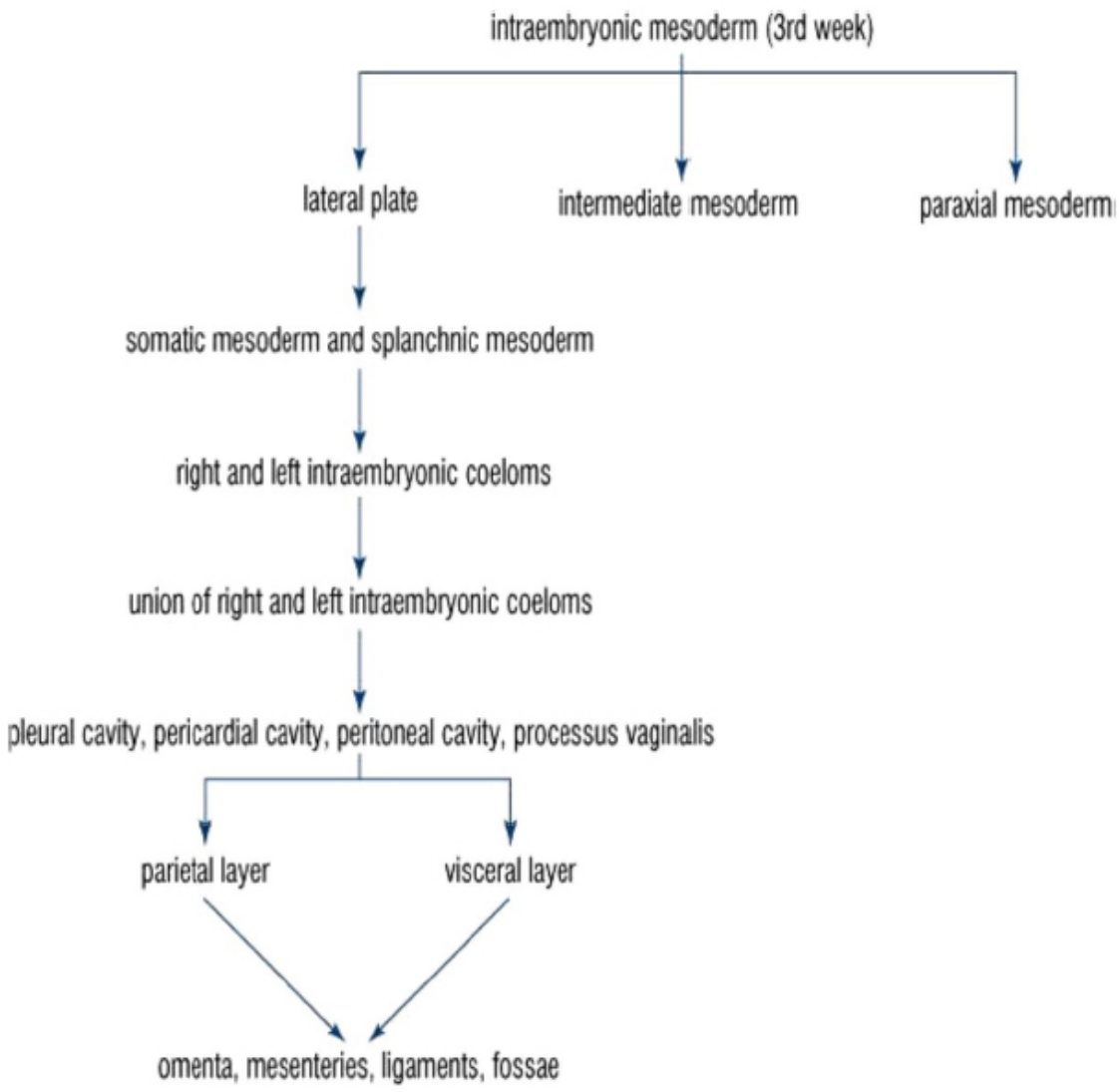


Fig.1. Development of peritoneum

PARIETAL PERITONEUM:

The inner lining of the abdominal and pelvic walls is formed by the parietal peritoneum. In addition, it also lines the lower surface of diaphragm. It is

freely attached to the walls by extra peritoneal connective tissue and thus can be easily ripped off, because of somatic innervations it is pain sensitive.

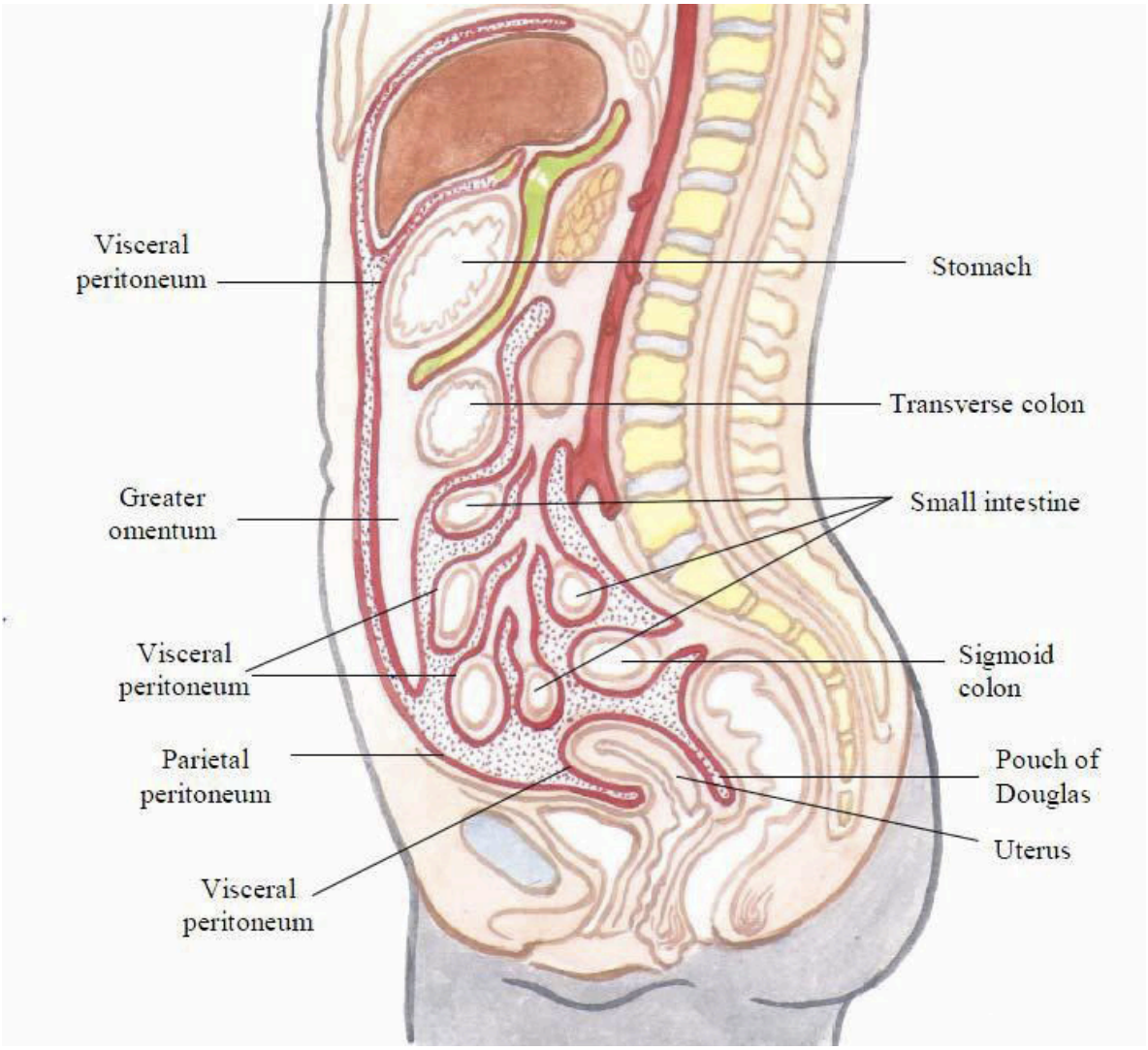


Fig.2. Layers of the peritoneum

VISCERAL PERITONEUM :

The outer surface of the intra-abdominal organs are lined by the visceral peritoneum. In contrast to the parietal peritoneum, it is adherent to the

viscera and cannot be stripped off. It derives its vascular supply and innervation similar to the corresponding viscera. The visceral peritoneum is innervated by the autonomic nervous system rendering it pain insensitive. Microscopically, the visceral layer of the peritoneum contains two layers

1. Fibrous layer that renders support to the membrane
2. Mesenchymal layer that serves for the secretory function

THE PERITONEAL CAVITY

It stretches upto a surface area of $2m^2$ which is almost half the skin's surface area. In males, this cavity becomes a closed sac. Whereas in females, the two ends of the fallopian tubes are open, rendering a communication to the external mileu.

The peritoneal cavity is further demarcated broadly into

1. The greater sac and the lesser sac
2. The pelvic and abdominal portions

The transverse colon along with its mesentery further delineates the abdominal portion into the supra-colic and infra colic regions. The small bowel mesentery divides the infra-colic compartment into the right infra-colic and left infra-colic regions. The ascending and descending colon again divides these right and left

infra-colic regions into the right and left external and internal paracolic gutters respectively. Supracolic compartment is the one that is below the diaphragm and above transverse colon and its mesentery. This space comprises of the liver, gallbladder, spleen, stomach and the duodenum (first part). The subphrenic spaces are formed by the liver and its ligaments

SUBPHRENIC SPACES :

There are seven subphrenic spaces, four intraperitoneal spaces and three extra peritoneal spaces. It is divided into right subphrenic and left subphrenic space by falciform ligament. The intraperitoneal spaces are:

1. Right anterior (superior) (subphrenic)
2. Right posterior (inferior) (subhepatic)
3. Left anterior (superior) (subphrenic)
4. Left posterior (inferior) (subphrenic)

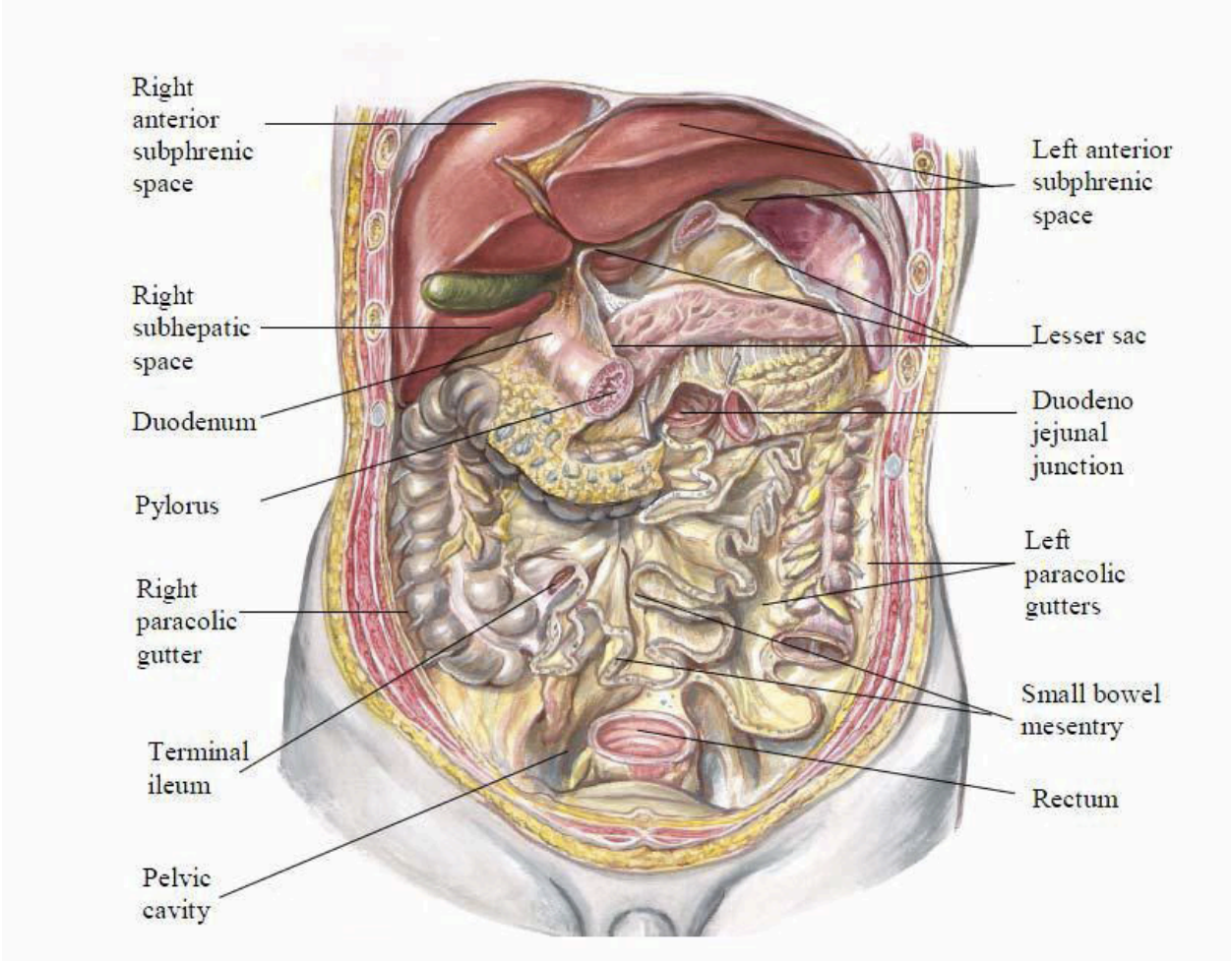


Fig.3. Spaces of the peritoneum

There are three extra peritoneal spaces, which are

- Right and left extra peritoneal space also known as perinephric spaces.
- Midline extra peritoneal area or the bare area of liver.

1. Right anterior (superior) intraperitoneal space (Right subphrenic space)

It is the space between the diaphragm and right lobe of liver. The posterior limits are formed by the anterior layer of the coronary ligament and the right triangular ligament. Leftward, the space is limited by the falciform ligament. This area can be commonly filled by collections arising from a perforated gallbladder, perforated ulcer of the duodenum or a stump blow out arising as a complication post gastrectomy.

2. Right inferior (posterior) intraperitoneal space (Right sub hepatic space)

This space is also termed as the Hepatorenal pouch of Morrison. The right limits are formed by the right lobe of liver and diaphragm. The foramen of Winslow is located to its left and the duodenum lies below. The liver and gall bladder are placed anterior and the upper part of right kidney and diaphragm, posterior. The space is superiorly limited by the liver and inferiorly by the transverse colon and the hepatic flexure. The space is situated in a deep plane and the common causes of collection in this space i.e, a subphrenic abscess are cholecystits, perforated duodenal ulcer, appendicitis and post abdominal surgeries.

3. Left anterior (superior) intraperitoneal space (subphrenic space)

The diaphragm forms the upper boundary and the posterior limit is formed by the left triangular ligament and left lobe of the liver, the lesser omentum and the anterior surface of stomach. The right limits are the falciform ligament and the lesser omentum, diaphragm and spleen forms the left limits. Any surgery on the stomach and pancreatic tail, colon (splenic flexure) or the spleen can cause collections here.

4. Left inferior (posterior) intraperitoneal (left sub hepatic space)

It is also called the lesser sac. Acute pancreatitis with complications like pseudocyst/ necrosis with collection is the common reason for collection in this region. Rarer causes may be a perforated ulcer of the stomach which might lead to collection/ abscess formation in the lesser sac when there are dense adhesions in the peritoneal space.

Extraperitoneal spaces

The right and left extraperitoneal space is the site for perinephric abscess. Midline extra peritoneal space is the same as the bare area of the liver. In amoebic hepatitis and pyogenic liver abscess, the abscess might get collected here. It can cause generalized peritonitis following rupture.

PHYSIOLOGY OF THE PERITONEUM

Mesothelial cells are of two types- cuboidal and flattened. Cuboidal cells have stoma in between them, which is increased in peritonitis. Beneath the epithelium is a collagen layer that forms the basement membrane and still beneath it is a complex connective tissue composed of mast cells, fibroblasts, eosinophils, lymphocytes etc. Mesothelium secretes around 50- 150 ml of peritoneal fluid per day and its composition is similar to plasma. The peritoneal fluid has around 3 gm/dl which is less than that of plasma. Mesothelium and subdiaphragmatic lymphatics absorb fluid. Mesothelial cells also absorb solute by process of endocytosis. This bidirectional movement of fluids across peritoneal membranes has been used in peritoneal dialysis. Two primary forces govern the fluid flux in

the cavity of peritoneum. (a) Gravity (b) Negative pressure that is generated below the diaphragm with respiratory movement.

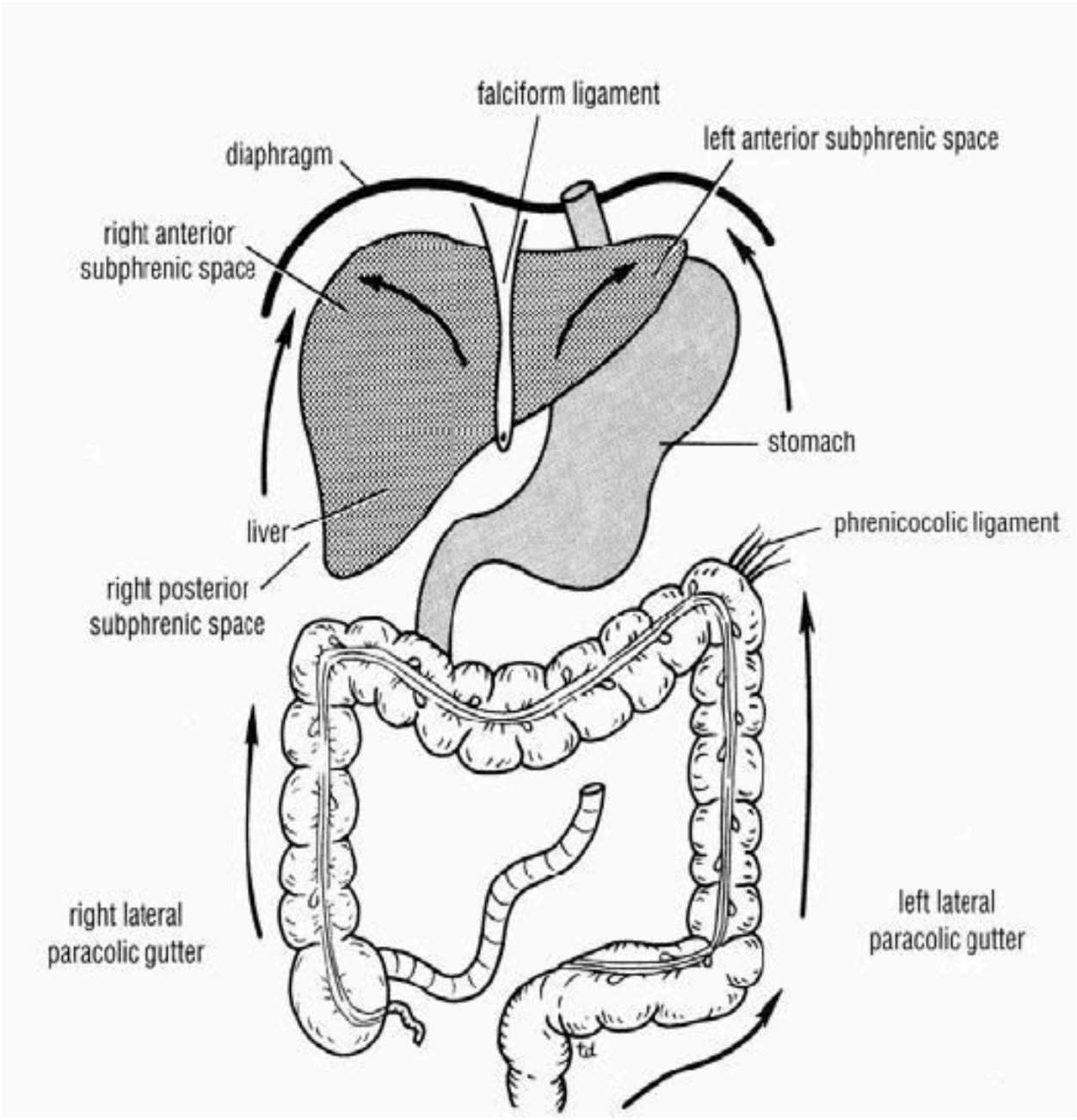


Fig.4 Flow of peritoneal fluid

PERITONEAL RESPONSE TO INJURY

The peritoneum initially reponds to any type of insult by producing a peritoneal reaction. This, microscopically can be visualised as the loss of mesothelial cells in that part of peritoneum. The time taken for large and small defects in the peritoneum are necessarily the same. It has been shown that after three days of peritoneal injury connective tissue cells resembling new mesothelium cover wound surface. At day five, new surface layer closely resembles adjacent normal epithelium. On day eight mesothelium regeneration is complete. The exact origin of cells responsible for mesothelial regeneration remains unknown.

It is postulated, the regeneration mechanisms include Submesothelial cells producing new mesothelial cells. Surviving or floating mesothelial cells or those attached to wound edges migrating into the wound. Peritoneal fluid monocytes and macrophages may also be differentiating into mesothelial cells. Normal peritoneal wound heals without adhesion formation. Adhesion develops in response to factors others than simple peritoneal wounding.

Local tissue hypoxia or ischemia appears to be the most important factor in adhesion formation apart from mechanical sub peritoneal surface injury, intraabdominal infections, and contamination of peritoneal cavity by foreign material. Deposition of fibrin following peritonitis is essential for adhesion formation. It has been shown that fibrinolytic activity is absent in healing wound until mesothelial cells are found. Fibrinolytic activity is minimal at three days in view of few mesothelial cells but complete at the end of eighth day, when mesothelial regeneration is complete. Therefore with intact mesothelial surface and adequate fibrinolysins, early fibrinous adhesions disappear. Formation of adhesion is both a protective response, helping to localize infection and an adoptive response to wound healing by carrying additional blood supply.

PATHOPHYSIOLOGY OF PERITONITIS

Generalized or local inflammation of peritoneum is designated as peritonitis. Each and every case of peritonitis of whatever cause, initiates a sequence of responses involving the peritoneal membrane, the bowel, and the body fluid compartments, which then produce secondary endocrine cardiac, respiratory, renal, and metabolic responses.

PRIMARY RESPONSES IN PERITONITIS

Membrane inflammation Peritoneum reacts to injury by hyperemia and transudation. Edema and vascular congestion occurs in the sub peritoneal layer immediately external to peritoneal membrane. Absorption across inflamed peritoneum in early cases is increased and decreases with chronicity. Absorption of macromolecules appears to be more affected than small molecule absorption. Transudation of fluid with low protein content from the extracellularly interstitial compartment into abdomen is accompanied by diapedesis of polymorphonuclear leucocytes.

During the early vascular and transudative phase of engorgement, the peritoneum acts as a two way street such that toxins and other materials that may be present in the peritoneal cavity are readily absorbed, get into the blood and lymphatic flow and cause generalized symptoms. Transudation of interstitial fluid into the peritoneal cavity across the inflamed peritoneum is shortly followed by exudation of protein rich fluid. The fluid exudates contains large amounts of fibrin and other plasma proteins in concentration sufficient to bring about clotting

later, that results in agglutination of loops of bowel, other viscera and the parities in the area of peritoneal inflammation. There is increased synthesis of lipoproteins and proteolysis. Concentration of uronic acid increases reflecting the exudation of plasma proteins in the early stages of peritonitis and in later stages increased synthesis of glycosaminoglycans due to activation of fibroblasts and mesothelial cells.

Changes in non-collagen and collagen protein synthesis are two events that occur in inflamed peritoneum during peritonitis. In early peritonitis non- collagen protein synthesis are increased and vice versa in later stages owing to increased protein synthesis in total. The RNA: DNA ratio, an index of protein synthesizing capability of tissues, increases during the first week of peritonitis. Bowel response Initially, response of bowel to peritoneal irritation is transient hypermobility. After a short interval, motility becomes depressed and nearly complete adynamic ileus soon follows. Bowel distension with air and fluid accumulation occurs finally.

Hypovolaemia Peritoneum reacts to injury by hyperemia and transudation of fluid that almost resembles plasma from all the three compartments of the body viz., the extracellular, intracellular and interstitial. This extracelluar fluid is trapped as edema by the loose connective tissue that lies below the mesothelium of the organs, mesentery and parieties. This is also accumulated in the bowel. This shift of water along with the essential proteins and electrolytes causes a temporary sequestration also called as third spacing. The fluid in the third space is functionally not available for the body. The amount of extra cellular fluid that is lost in this manner is directly proportional to the area of the peritoneum that is inflamed. Even massive shifts like 4-6 L in 24 hours can occur when there is extensive peritoneal inflammation.

SECONDARY RESPONSES IN PERTIONITIS

Endocrine response

There is almost an immediate adrenal medullar response, with out - pouring of epinephrine and nor-epinephrine producing systemic vasoconstriction, tachycardia and sweating. There is a surge of hormones from the adrenal cortex during the initial few days following the peritoneal insult. The hypovolemia also

triggers secretion of anti-diuretic hormone and aldosterone which increases sodium and water retention. Water retention may be greater than sodium retention resulting in dilutional hyponatremia.

Cardiac response

The effects of peritonitis and cardiac function are a reflection, of both decrease in ECF volume and progress in acidosis. Volume deficit results in decreased venous return and diminished cardiac output. Heart rate increases in an attempt to increase cardiac output, but compensation is usually incomplete. With ongoing acidosis, the contractile mechanism of the heart is affected and this further affects the cardiac output.

Respiratory response

The respiratory system is involved through the diaphragm. Any distension of the abdomen, eg. Ileus pushes the diaphragm and this reflects as reduced ventilator volume and basal atelectacis. This is further aggravated by the restricted movement of the diaphragm and intercostal muscles due to pain.

Renal response

Urine volume is diminished and renal capacity to handle an excess of solute is impaired. Hypovolemia reduces cardiac output and increased secretion of ADH aldosterone in peritonitis, all acting synergistically on the kidney. Renal blood flow is reduced and in turn the GFR and tubular urine flow. Reabsorption of water 20 and sodium is increased often in imbalance and potassium is wasted.

Metabolic response

The metabolic rate is generally increased with increased oxygen demand at the peripheral sites. Also, the oxygen delivering capacity of the heart and lungs are reduced. This poor systemic blood flow leads to a change to anaerobic metabolism in the peripheries like the muscle. This anaerobic metabolism generates lactate, which starts to accumulate. This eventually leads to lactic acidosis. Both 'D' and 'L' isomers of lactate are produced by bacterial metabolism and may be absorbed during peritonitis. Human beings can rapidly metabolize 'L' lactate, but have a relatively limited capacity to handle 'D' lactate. Protein catabolism begins early in peritonitis and progressively becomes severe.

Plasma proteins are preferentially synthesized while muscle proteins are catabolized during peritonitis.

RELEVANCE OF INTRA ABDOMINAL PRESSURE

In critically ill patients intra abdominal pressure monitoring has been observed to play a vital role especially in cases of severe sepsis, sepsis with hypotension, surgery in emergency and trauma situation. Intra abdominal hypertension can provoke or induce organ dysfunction and this can be attributed to the direct pressure effect over the organs inside or close to the peritoneal cavity. Another indirect effect is due to secondarily raised intra thoracic pressure which can affect the distant organ systems like the respiratory and central nervous system.

HISTORICAL BACKGROUND:

Ever since the nineteenth century, the knowledge about intra abdominal pressure and its importance is believed to exist. However, this has not been identified until the recent era of advanced critical care where detailed monitoring of organ function has become a possibility. A plethora of research has

been published over the recent decade, which highlights the negative effects of "Intra Abdominal Hypertension". Today it plays a very major role in critical care and the physicians and surgeons can seldom ignore this phenomenon in their day-to-day practice. "In 2004, the World Society of Abdominal Compartment Syndrome was founded to bring together physicians, nurses and other allied health professions with an interest in ACS to promote education and facilitate research". "The WSACS has published two consensus papers on definition and recommendation and continues to support IAH research around the world." The WSACS has laid down multiple risk factors for the development of "Abdominal Compartment Syndrome".

"RISK FACTORS FOR INTRA ABDOMINAL HYPERTENSION AND ABDOMINAL COMPARTMENT SYNDROME" (AS PER WCACS GUIDELINES)

Decreased compliance of abdominal wall

Surgeries of abdomen

Traumatic injuries

Burn injuries

Prone position

Increased contents in intra-luminal compartment

Gastroparesis/distension of stomach

Ileus

Pseudo-obstruction of the colon

Volvulus

Increased contents in the intra-abdominal compartment

Acute pancreatitis

Distension of the abdomen

Hemo or Pneumoperitoneum

Intra abdominal collection

Infection/abscess

Malignancies

Excessive insufflation during laparoscopy

Cirrhotic disease of liver

Peritoneal dialysis

Capillary leak/fluid resuscitation

Acidosis

Damage control surgery

Hypothermia

Increased APACHE-II or SOFA score

Massive fluid resuscitation or positive fluid balance

Polytransfusion

Others/miscellaneous

Age

Generalised Sepsis

Bleeding disorders

Elevation of head end of bed

Repair of huge incisional hernia

Mechanical ventilation

Obesity or increased body mass index

PEEP

Peritonitis

Pneumonia

Sepsis

Shock

EFFECTS OF INCREASED IAP IN VARIOUS ORGAN SYSTEMS

Abdominal Compartment Syndrome i.e, IAP > 20mmHg is associated with organ dysfunction. However, increased IAP can have deleterious effects on various organ systems in our body. With the brunt of insult borne by the cardiovascular and gastrointestinal system, all other systems are uniformly affected too. Knowledge of this pathophysiology is essential as the surgeon usually ignores the deleterious effects of IAP.

Broadly the effects of Intra Abdominal Hypertension can be grouped by the grade as follows

Fig. 5. EFFECTS OF GRADE-I IAH

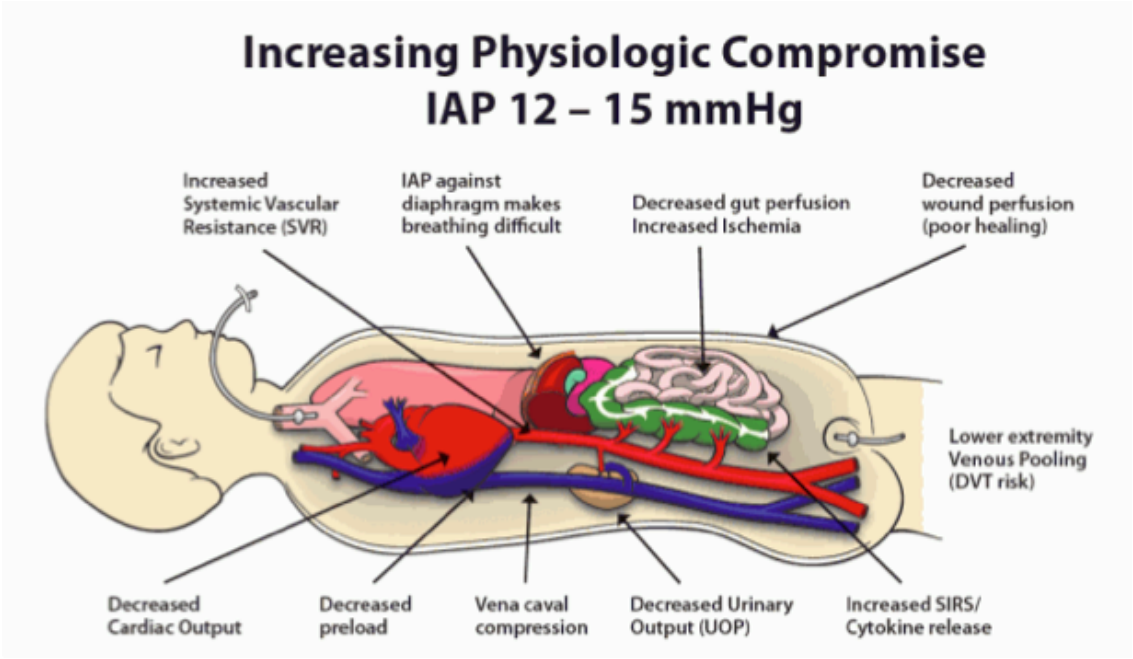


Fig. 6. EFFECTS OF GRADE-II IAH

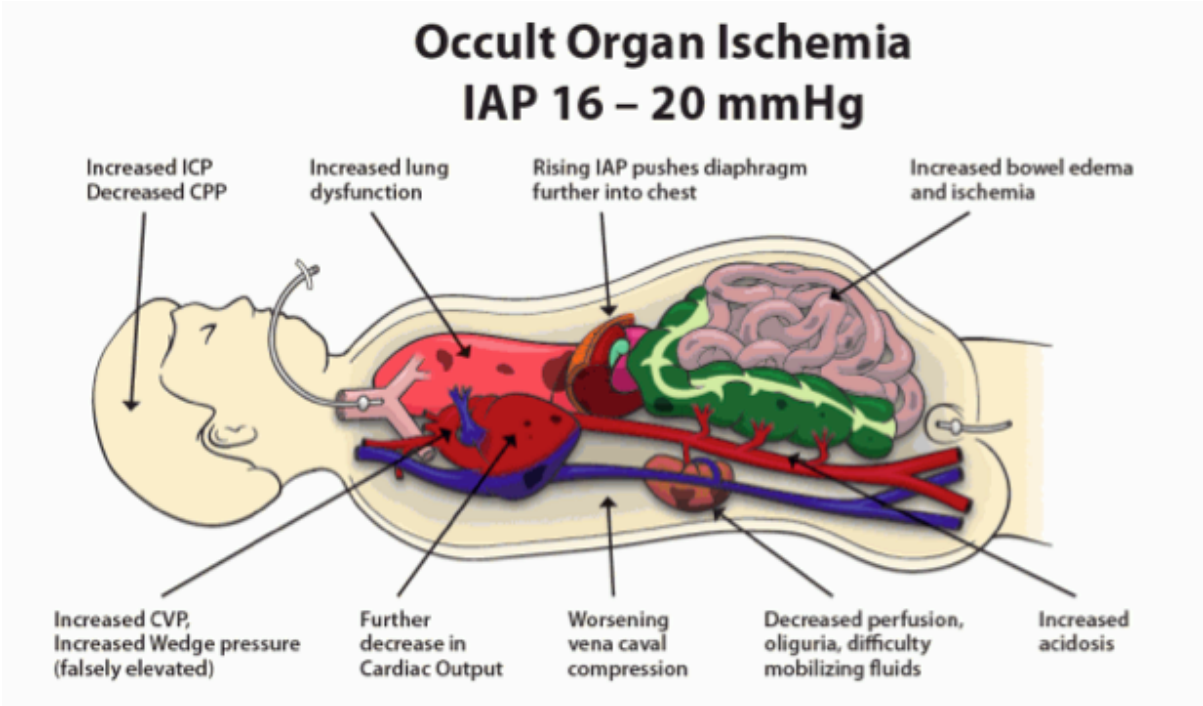
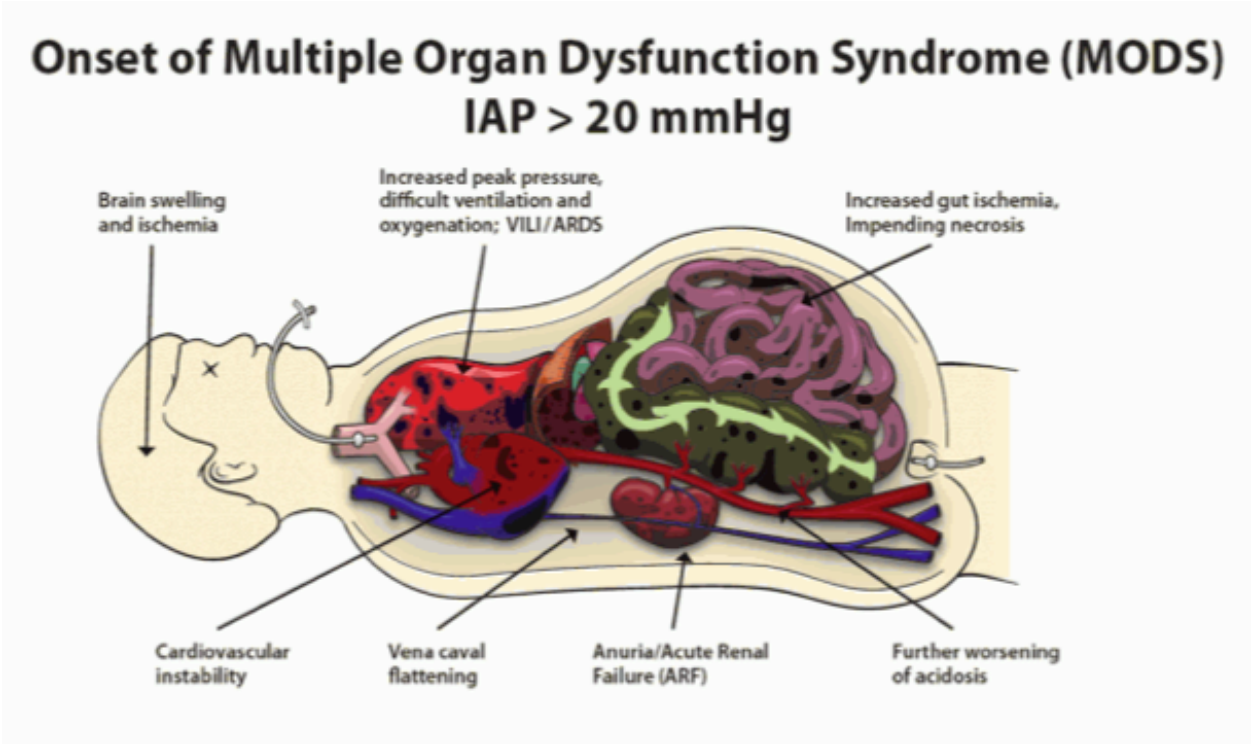


Fig. 7. EFFECTS OF GRADE -III IAH



EFFECTS ON IAH ON THE CARDIOVASCULAR SYSTEM

Multiple factors causing intra abdominal hypertension affect the cardiovascular system in varied ways, importantly, IAH induced organ dysfunction due to Abdomino-thoracic pressure transmission. With increased IAP, there is cephalad displacement of diaphragm leading to increased intra thoracic pressure. i.e., Increased IAP transmitted to thorax. This abdomino-thoracic pressure transmission varies with abdominal wall and chest wall

compliance corresponding to the respiratory cycle. This further causes cardiac compression and reduction of end-diastolic volume.

The main cardiovascular effects of increased intra abdominal pressure are three fold

1. Decreased venous return by the abovementioned mechanisms decreases the cardiac preload and thereby decreases the end diastolic volume
2. The cardiac after load increases due to increased vascular resistance caused by the increase in intra peritoneal pressure and activation of the renin angiotensin system
3. This decreased after load again decreases cardiac output. Mean arterial pressure raises initially due to shunting mechanisms but eventually declines

These mechanisms are further aggravated by hypovolemia and PEEP. Initially hypervolemia (transfusion) is thereby protective.

Management of IAH critically depends on interpretation of hemodynamic parameters. “Abdomino thoracic transmission of pressure falsely elevates traditional filling pressures ie., Central Venous Pressure (CVP) and

Pulmonary Artery Occlusion Pressure (PAOP)". "These do not reflect true cardiac filling". This can be overcome by several methods.

1. Corrected CVP and PAOP values corresponding to rise in IAP can be calculated

   Transmural CVP = CVP – ITP (Intra Thoracic Pressure)

   Transmural PAOP = PAOP – ITP (Intra Thoracic Pressure)

The estimation thus obtained might be crude and to rectify the same, alterative methods have been reported. The change CVP i.e, ΔCVP and change in IAP, ΔIAP, can be calculated bedside by measuring CVP and IAP before and during abdominal compression. This is important as the Surviving Sepsis Guidelines "targets initial and ongoing resuscitation towards a CVP of 8 – 12 mmHg and a MAP of 65 mm Hg"

Other parameter used for volumetric monitoring is “Right Ventricular End Diastolic Volume Index (RVEDVI) or Global End Diastolic Volume Index (GEDV)” . RVEDVI has been shown to be an accurate indicator of “preload recruitable increase in cardiac index [CI]”.

RVDEVI is also independent of ITP or IAP fluctuations and variations in ventricular compliance. The usefulness of RVDEVI has been demonstrated in many situations like shock due to haemorrhagic, neurogenic, cardiogenic causes, acute lung injury and pulmonary hypertension. Thus the correlation between RVDEVI and CI is much more significant than correlating PAOP or CVP with CI especially in cases of preload resuscitation. Also, Diebel et al ”demonstrated that PAOP measurements provide potentially misleading information regarding preload status in 52 % critically ill patients”. In cases of increased ITP and IAP, the PAOP and CVP can give an erroneous picture of preload status and it is in these patients the RVEDVI comes to a timely rescue.

“Pulse pressure variation (PPV) or stroke volume variation (SVV) are the dynamic parameters that can help to gauge fluid responsiveness”. The main drawbacks of the above said parameters ar that they are reliable only in

sedated patients without spontaneous respiratory movements and in patients without cardiac arrhythmias. Duperret et al. “showed in a pig model that SVV and PPV are increased when experimental IAH is induced. However, since the pigs were not subjected to a fluid bolus, it is impossible to determine whether this is due to real hypovolemia induced by the decreased venous return in IAH, or a false increase in SVV and PPV due to erroneous measurement”.

Some key points to be remembered are,

1. The interaction between IAP, ITP, PEEP and cardiac chamber filling pressure must be kept in mind by the surgeon.
2. “Misinterpretation of the minute – minute cardiac status may result in detrimental therapy especially additional volume expansion”.
3. “Transmural filling pressures calculated as the end expiration (ee) value minus ITP reflects preload better” but this has to be monitored with the help of an esophageal balloon device.

   $$CVP(tm) = CVP(ee) - ITP$$

   $$PAOP(tm) = PAOP(ee) – ITP$$

4. “A rough value of transmural filing pressure can also be obtained by using a simple formula by deducting half of IAP from the end expiratory

filling pressure since abdomino thoracic pressure transmission is estimated to be around 50%"

$$CVP(tm) = CVP(ee) - IAP/2$$

$$PAOP(tm) = PAOP(ee) – IAP/2$$

5. "The surviving sepsis campaign guidelines targeting initial and ongoing resuscitation towards a CVP of 8-12 mmHg and MAP of 65mmHg" should be taken with pinch of salt to prevent overzealous resuscitation or under resuscitation

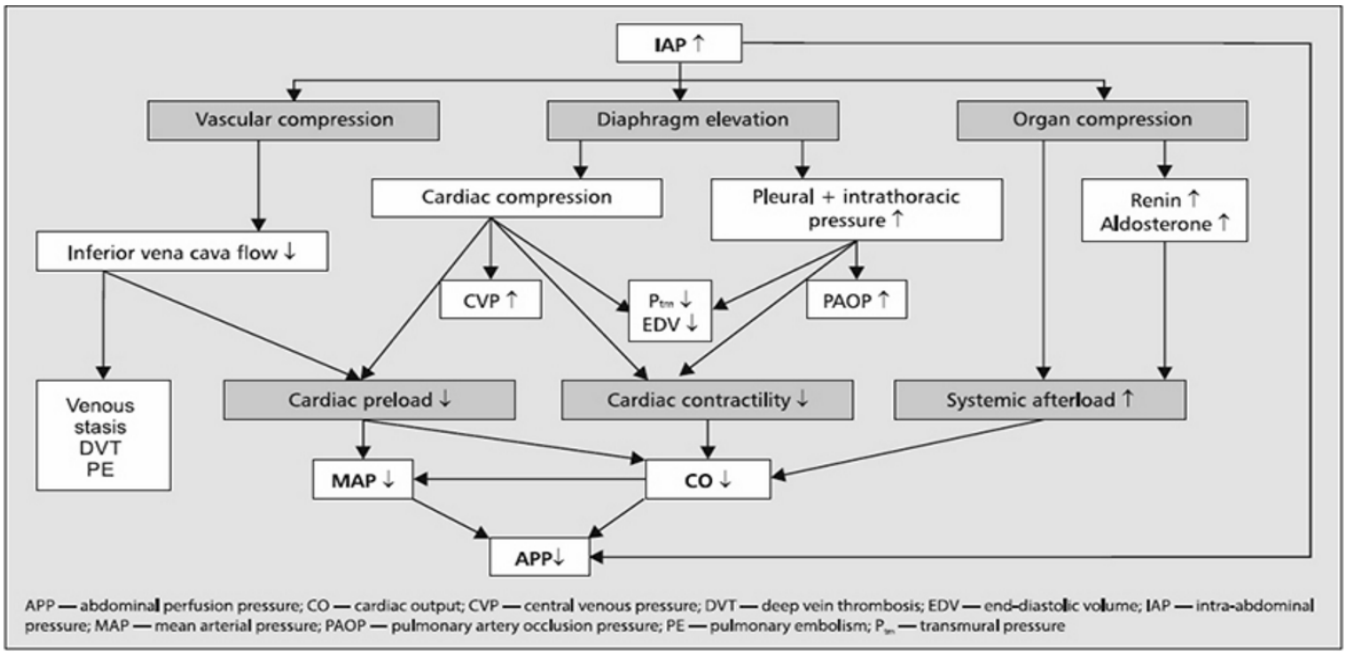


Fig.8. Summary of cardiac effects of Intra Abdominal Hypertension

EFFECTS OF IAH ON THE RESPIRATORY SYSTEM:

The above said "abdominothoracic transmission" has an effect on the respiratory system too. Often in cases with primary ACS, secondary ARDS ensues and the management strategies might be quite different from primary ARDS. The major problem is in the reduction of functional residual capacity.

This, in addition to the changes caused by secondary ARDS leads to a condition described as "baby-lungs". Though the lung compliance remains unaltered, the increased intra abdominal pressure decreases the total respiratory compliance by decreasing the compliance of chest wall. Based on the following pathophysiology the following ventilation strategies are recommended.

1. "PEEP should be set to counteract the intra abdominal pressure and also to avoid over inflation".
2. "During lung protective ventilation, the plateau pressures should be limited to transmural plateau pressures below 35 mmHg

   $$P_{plat(tm)} = P_{plat} - IAP/2$$"

3. "Monitoring of extravascular lung water index (EVLWI) seems warranted in risk patients since IAH is associated with increased risk of lung edema".

EFFECTS OF IAH ON RENAL FUNCTION

One of the organs consistenly affected by IAH changes are the kidneys. The renal effects of IAH are often multifactorial and reflect the interplay of various pathological factors in IAH.

One of the important mechanisms by which the kidneys are affected are

1. IAH causing decreased renal venous flow by direct compression and thereby increasing the pressure in renal veins.
2. "The microcirculatory flow to the cortex is also decreased as there is direct compression of the renal cortex"

These changes end up in triggering the "Renin- Angiotensin-Aldosterone pathway" and also in increased secretion of ADH. But whether these changes are clinically relevant, is still unclear.

Ulyatt suggested that " Filtration Gradient (FG) may be an important factor in explaining renal failure associated with IAH". Filtration Gradient is the "mechanical force across the glomerulus and is equal to the difference in glomerular filtration pressure and the proximal tubular pressure. Glomerular filtration pressure = Renal perfusion pressure and thus to MAP – IAP". "In the

presence of IAH, proximal tubular pressure can be equated with IAP". In these cases, FG can be derived from the following formula

$$FG = MAP - (2 \times IAP)$$

Thus the kidney is more vulnerable to changes in IAP compared to adjuscent viscera and these factors play an important role in the pathogenesis of IAH induced renal failure.

Though there are no specific protocols in the management of IAH induced renal failure, one important approach is improving renal perfusion by using abdominal perfusion as a resuscitation end point. In addition to reflecting the severity of IAH, abdominal perfusion pressure also assesses the adequacy of systemic blood flow. Quite similar to the calculation of Cerebral Perfursion Pressure as Mean Arterial Pressure minus Intra Cranial Pressure, "Abdominal Perfusion Pressure can be calculated as Mean Arterial Pressure minus Intra Abdominal Pressure".

$$APP = MAP - IAP$$

"Abdominal Perfusion Pressure was also found to be statistically superior to arterial pH, base deficit, arterial lactate and hourly urinary output in its ability to predict patient outcome. Malbrain et al, in three subsequent trials in mixed medical –surgical patients (Mean IAP 10 +/- 4 mmHg) suggested that an APP ≥ 60 mmHg represented an appropriate resuscitation goal. Persistence of IAH and failure to maintain an APP ≥ 60 mmHg by day 3 was found to be discriminating between survivors and non-survivors".

"The validity of Abdominal Perfusion Pressure as a resuscitation end point has yet to be established by RCT". Also, the threshold above which raising MAP does not improve APP is yet to be described. However, over zealous fluid resuscitation puts the patient at risk of secondary ACS and should be avoided.

EFFECTS OF IAH ON THE CENTRAL NERVOUS SYSTEM

IAP has found to showing a direct relationship with Intra cranial pressure in many studies. It has been hypothesized that IAH causes a secondary increase in ICP through an increase in ITP. This results in an increase in CVP and diminished cerebral venous return and thus predisposing to venous congestion and

cerebral edema. “Bloomfield et al have further substantiated this by performing a sternotomy and bilateral pleuropericardiotomy in experimental pigs and abolishing this association between IAP and ICP”.

Overall the Cerebral Perfusion Pressure decreases as IAH reduces the preload to heart and increases intra cranial pressure. Also, Abdominal decompression or curarisation as a treatment for refractory intracranial hypertension has been reported.

Considering the effect of IAP on ICP, the following recommendations have been made

1. It is essential to monitor IAP in all cases where an increase in ICH or IAH is suspected.
2. IAH must be avoided in cases with increased ICP
3. In patients who have developed ICH, IAP increase must be avoided.
4. In patients who are at increased risk of ICH, Laparoscopy is better avoided as the pneumoperitoneum created during laparoscopy is analogous to an increase in IAP in experimental settings. This is particularly relevant in cases of trauma where both abdominal and brain injuries may coexist.

EFFECTS OF IAH ON GASTROINTESTINAL SYSTEM:

Intra abdominal hypertension can affect the gastrointestinal tract in multifold ways which includes reduction in the gastrointestinal perfusion, mucosal acidosis and finally culminating in multiple organ failure. “IAH and ACS have been postulated to serve as the second hit in the two-hit phenomenon of the causation of MODS”. Temporal relationship has been proven between ACS and MODS. In experimental animals “ ACS provokes cytokine release and neutrophil migration resulting in remote organ failure”. However in humans, even the absence of systemic hypotension and reduced cardiac output, abdominal compartment syndrome results in reduction in the splanchnic blood flow. The inflammatory cytokines released during this insult is carried by the lymph flow across all organs thereby precipitating multiorgan failure.

EFFECTS OF IAH ON HEPATIC SYSTEM:

Many studies have established the vulnerability of liver to increase in IAP. Even moderate elevation of IAP (around 10mmHg) has been found to affect hepatic cell function and hepatic perfusion. In addition, IAH and ACS can

complicate pre-existing acute liver failure, decompensated chronic liver disease and liver transplantation.

EFFECTS ON IAH ON ABDOMINAL WALL:

"Abdominal wall blood flow is proven to be affected by the direct effect of compression by increased IAP" and this further leads to ischemia and edema at the local site. Poorly perfused abdominal wall and fascia can further add up to the infectious and non-infectious complications like wound dehiscence, rectus dehiscence and necrotizing fasciitis.

ACS IN BURNS PATIENTS

Intra abdominal hypertension and abdominal compartment syndrome are commonly seen in burns patients and the risk of developing IAH and ACS seem to be directly proportional to the area of skin involved. While burns > 50% TBSA carry a risk of IAH, burns > 70% with inhalational injuries are more vulnerable to develop overt ACS. Also, the development of IAH and

subsequently ACS seem to be more related to the massive fluid resuscitation rather than direct abdominal wall involvement by the burns. Nevertheless, these severe burn victims with inhalational injuries usually die of other respiratory complications before ACS coming into play.

ACS IN MORBIDLY OBESE PATIENTS

Recently it has been studied that the baseline values of Intra Abdominal pressure are elevated in obese patients. Thus, an increase in IAP similar to intra abdominal hypertension in a critically ill patient, can have deleterious effects on organ function. Increased IAP in patients with elevated Body Mass Index is now being identified to be involved in multiple diseases like "Obesity Hypoventilation Syndrome, pseudo tumor cerebri, gastro-oesophageal reflux and stress urinary incontinence". Also the increased IAP in these patients might also contribute to poor fascial and wound healing following surgery.

ACS IN TRAUMA PATIENTS

Studies have shown an increased incidence of intra abdominal hypertension and abdominal compartment syndrome in cases of trauma, and this incidence is found to range from “14 to 36%”. Abdominal compartment syndrome is found to be a common complication after damage control surgery. This is due to the fact that damage control surgery is usually done in cases of complex hepatic injuries where multiple packs are placed around the liver to achieve hemostasis. The packs are then taken out usually in a second procedure. “Differences in the proportion of patients who received abdominal packs may account for the difference in IAH incidence between the two groups of comparable patients”. Uniformly many studies have established that there is increased risk of IAH in patients where fascial closure is attempted in the primary sitting. “However even with attempted prophylaxis by the use of a white loosely applied prosthetic mesh for temporary abdominal closure, 25% of patients with penetrating abdominal trauma developed IAH”. ACS developed early in cases of trauma even without abdominal injury due to massive fluid resuscitation.

METHODS TO MEASURE IAP

Varied methods have been described to measure Intra Abdominal Pressure. IAP measurement is based on the Pascal's Law according to which, "the pressure maintained at one point within the abdominal cavity should represent the pressure throughout the cavity provided its contents are primarily fluid in nature". Theoretically, IAP measurement can be done via catheters inserted through any natural orifice. Historically, IAP measurements have been made via the rectum and uterus. However, in present times, the intra peritoneal pressure is derived from

1. Vesical pressure
2. Gastric pressure

"Gastric pressure can be measured by using a balloon tipped catheter placed inside the gastric lumen". Though trans-vesical measurement is the gold standard, trans-gastric measurement is useful where bladder catheterization is technically impossible or contraindicated. Also, trans-gastric measurement allows a continuous IAP monitoring.

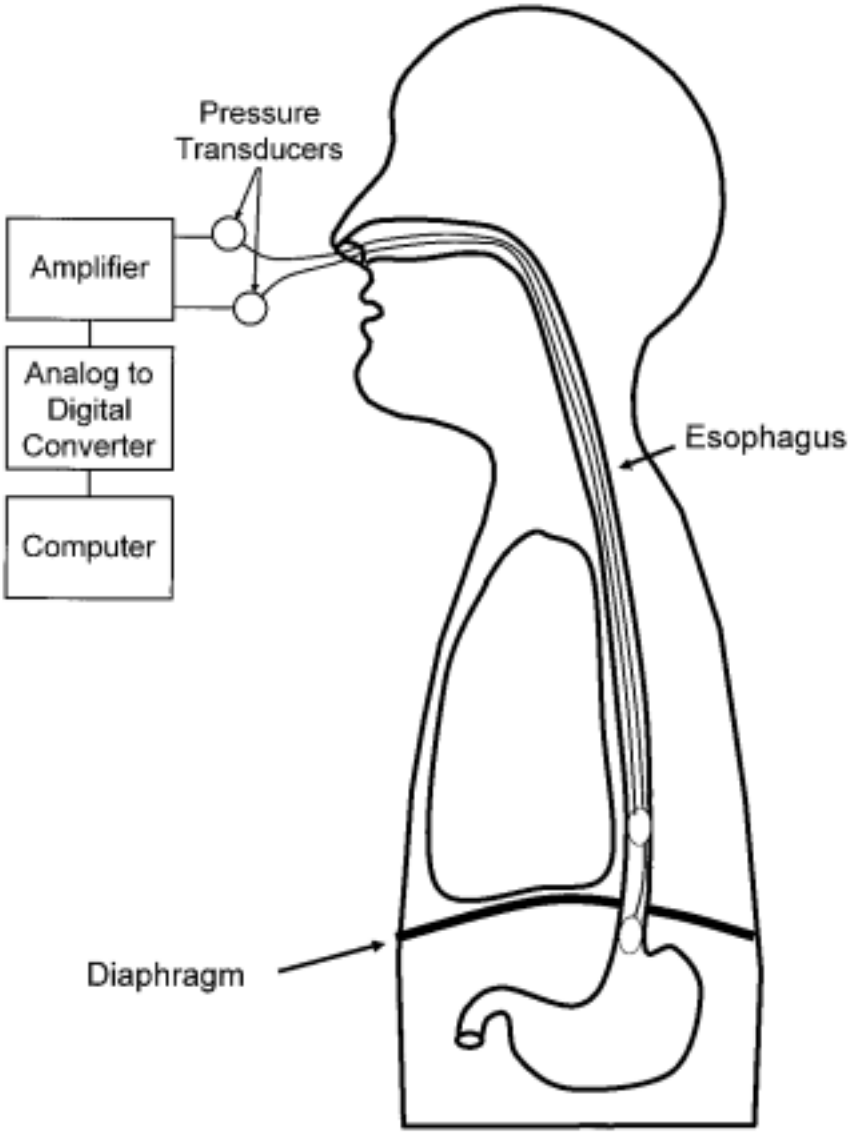


Fig.9. Intra-Gastric pressure measurement using balloon tipped catheter

INTRA-VESICAL PRESSURE MONITORING TECHNIQUES:

Intra vesical pressure can be measured using an indwelling Foley's catheter connected to a pressure transducer system as shown in the figure

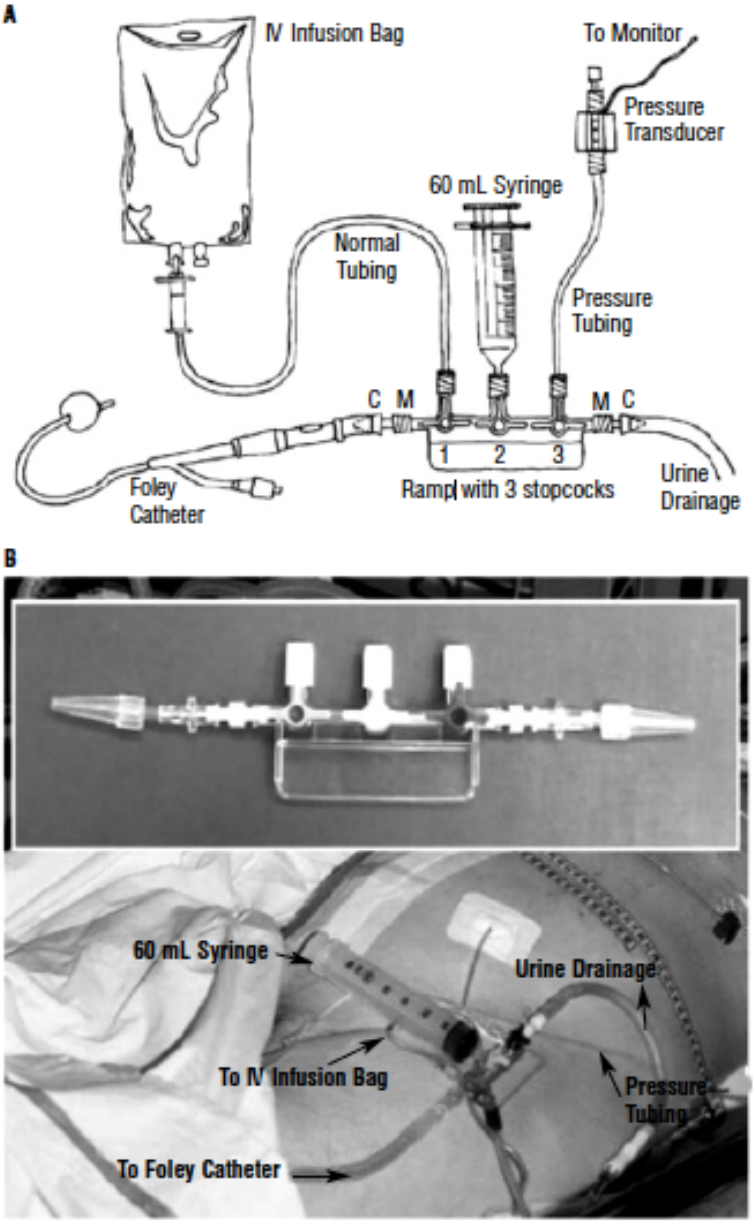


Fig.10. The pressure transducer apparatus for measuring IAP

Procedure of measuring IAP using a pressure transducer

1. The patient should be lying supine with the head end of the bed at 0 degrees

2. The patient should not have active abdominal muscle contractions while measuring IAP. i.e, the patient should not be agitated, coughing or actively talking
3. The pressure transducer must be leveled at the iliac crest at the level of mid axillary line and zeroed.
4. 20 – 25 ml of sterile saline should be drawn and instilled into the bladder
5. After 30 seconds, ensure that the bladder detrusor muscles are relaxed and then obtain the measurement
6. The measurement should be done at end expiration
7. In case of ventilated patients, this end expiratory phase when IAP has to be measured can be identified as the trough of the patient's respiratory waveform

The amount of saline injected for IAP measurement must be detected from the total urine output

Advantages of the pressure transducer technique

1. The values can be measured precisely without inter-observer varitation

2. Once the system is assembled, it becomes a closed circuit and the potential risk of UTI is therefore avoided

Disadvantages of the pressure transducer technique

1. Setting up the system takes considerable time
2. Requires additional training to handle the pressure transducer system
3. Owing to the high cost, it might not be feasible in developing countries

MANOMETER TECHNIQUE

The manometry technique is one of the oldest and original methods of measuring IAP. It is quite similar to the manometry technique of measuring Central Venous Pressure bedside. A manometer tube is placed in between the foley's catheter and the draining bag. This fluid column is open to atmospheric pressure on one side and connected to the bladder on the other end through the indwelling foley's catheter. The manometer is held vertically and level at the pubic symphysis.

The precautions followed in the previous method holds good for the manometer technique also. The column of urine in the tube is measured and this gives the intra-vesical pressure in cm of Hg. The intra abdominal pressure can be derived from the same by using a factor of 1.36. The following figure shows IAP measurement using the manometer technique

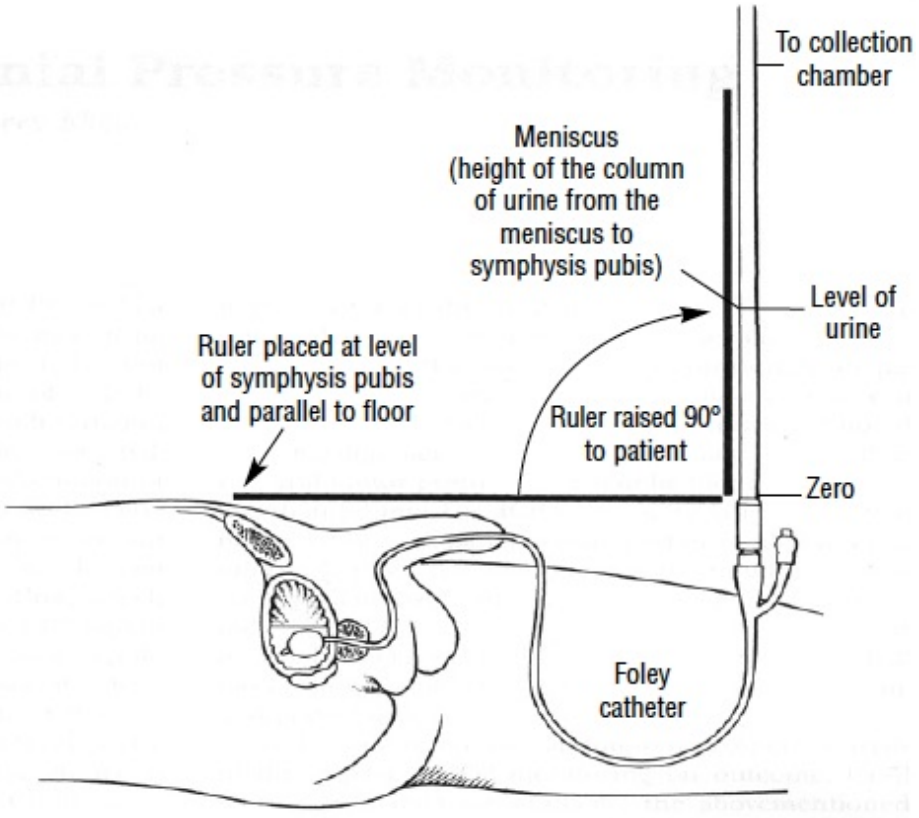


Fig.11. The pressure mannometry technique

Advantages of the manometer technique

1. Easy to practice and can be used both my doctors and paramedical staff

2. Does not need exclusive training
3. Very practical for day to day use in bedside especially in developing countries where cost factor plays an important role

Disadvantages of the manometer technique

1. There are high chances of inter observer variation
2. The sterile circuit is broken every time IAP is measured

In our study, we have used the manometer technique to measure IAP. In order to reduce inter observer variations, all IAP measurements are done by a single observer and sterile precautions akin to catheterization are followed to prevent any additional risk of UTI.

FLUID MANAGEMENT AND INTRA ABDOMINAL HYPERTENSION:

The development of IAH in a patient with sepsis is contemplated to have a two hit mechanism. The first hit being the infection process per se and the second hit involving the large scale inflammation and capillary leak. In these

circumstances, excessive fluid resuscitation may precipitate intra abdominal hypertension.

"Traditional teaching and protocols are aimed at correction of basic physiological parameters such as blood pressure , central venous pressure and urine output. Also the fluid resuscitation is the first and foremost recommended action in Surviving Sepsis Campaign Guidelines. These parameters have become ubiquitous due to their easy bedside availability and also due to the fact that they do not involve expensive equipments. Currently, the concept of resuscitation in septic shock is undergoing an important change and emphasis has been placed on various other parameters too.

"The ultimate goal of treatment in shock is to restore the balance between oxygen demand and delivery, which means the optimized cardiac output and effective circulatory volume". This goal of restoring the circulatory blood volume means that 1) external losses have to be substituted, 2) adequate volume must be supplied to the dilated vessels, 3) internal volume loss due to accumulation in the third space and capillary leak must be supplemented. Saline solution being an isotonic crystalloid has always been the intravenous fluid of choice. Sodium being an extracellular cation, the crystalloid solution will be

distributed in the extracellular compartments uniformly. Though colloids have a theoretical benefit that they favour shift of fluid from interstitial space into the intravascular space, many studies have failed in showing a significant survival benefit in colloid resuscitation. Also colloids incur higher cost and human and animal tissue derived colloids also have carried a meager but significant risk of disease transmission. Nevertheless synthetic colloids have their own adverse effects like anaphylaxis, coagulopathy and renal failure. Thus crystalloid solutions are considered "gold standard of fluid resuscitation"

ROLE OF SMALL VOLUME RESUSCITATION:

As the adverse effect of excessive fluid resuscitation now being widely recognized, therapeutic strategies have been modified to reproduce physiological goals of standard colloid administration albeit smaller volumes. This implies using hypertonic or hyperosmotic fluids. Several indications for the use of hypertonic saline have been brought into attention in American literature. In European literature, however, "Focus is being laid on newer and synthetic colloids like 130kD hydroxyethyl starch(HES)".

Many studies have attempted using a combination of hypertonic saline and colloids example, “Hyper HES, a solution consisting of NaCl 7.2% in HES. The risk of abdominal compartment syndrome has found to be reduced when using hypertonic lactated saline for burns resuscitation.” Also the first 24 hour fluid requirement and “peak inspiratory pressure after 24 hours” have found to be reduced in these patients. Thus the introduction of intra abdominal pressure has a parameter to guide fluid resuscitation is highly warranted and more clinical data on the choice of reliable resuscitation targets are been evaluated.

OPTIMIZING INTRA ABDOMINAL PRESSURE

Increased IAP needs intervention when IAP is ≥ 12 mmHg. According to the WCACS guidelines, “Approaches or techniques of potential utility include sedation and analgesia, neuromuscular blockade, body positioning, nasogastric/colonic decompression, pro-motility agents, diuretics and continuous renal replacement therapies, fluid resuscitation strategies, percutaneous catheter drainage (PCD), and different temporary abdominal closure (TAC) techniques among those requiring an open abdomen”

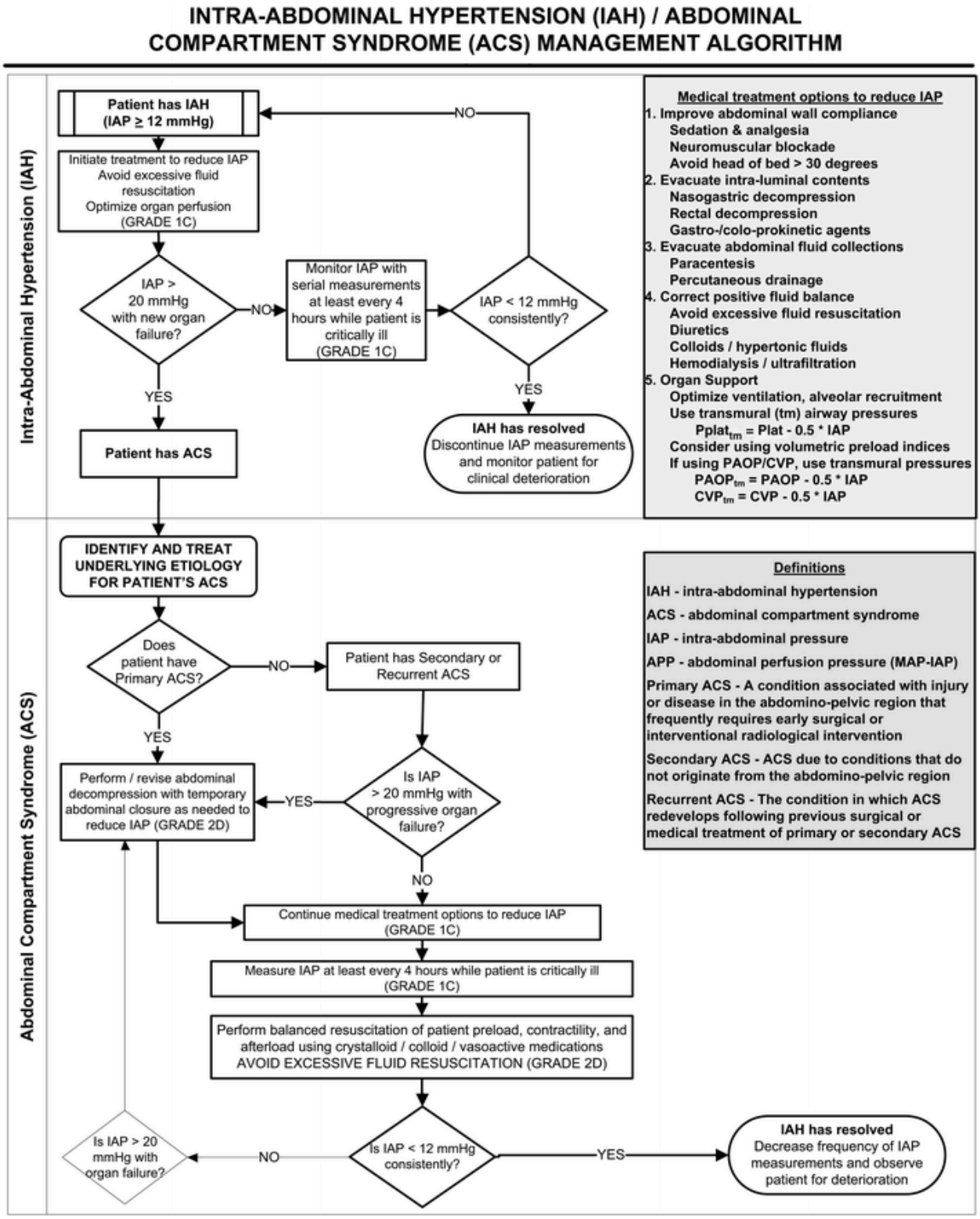


Fig.12. The management algorithm in ACS

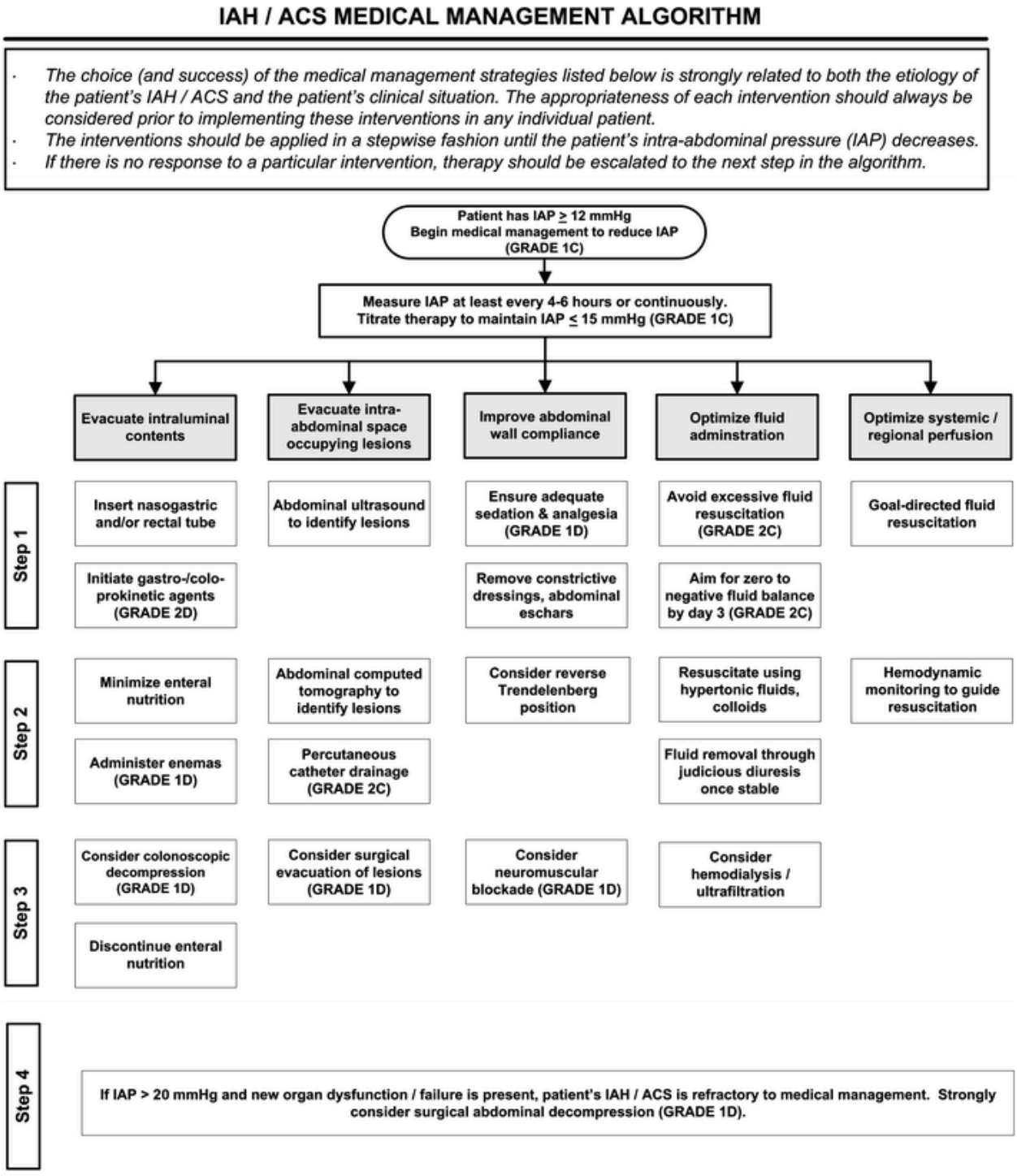


Fig.13 Different components in the management of ACS

TREATMENT STRATEGIES AND THE ROLE OF DECOMPRESSIVE LAPAROTOMY :

"Decompressive laparotomy (DL) is still the only available treatment for established ACS". De Waele et al. have conducted a systemic review and shown that DL has a positive effect on end organ function but carries a very high risk of mortality (50%). This increase in mortality has been possibly explained by the time delay in initiating treatment. Thus, it goes without saying that a patient with overt ACS should undergo "Decompressive laparotomy" on immediate basis, irrespective of the causes. The other management strategies including conservative management would be beneficial in "IAH with or without mild organ dysfunction".

The main pathophysiology in the development of secondary ACS is "increase intra abdominal volume or decreased abdominal compliance" and usually it is a combination of these two. Excessive fluid administration can eventually lead to accumulation of free fluid inside the abdominal cavity and edema formation both in the wall of the intestines and abdominal wall. Bowel edema leads to increased intra abdominal volume and abdominal wall edema reduces compliance. The increase in IAP and ITP also negatively affects "thoraco

abdominal lymphatic flow and this further increases the amount of free fluid in the peritoneum and thus making it a negative spiral".

Thus the main strategies in treatment are "decreasing abdominal volume or abdominal wall compliance". The volume of free fluid in the peritoneal cavity can be decreased by looking for free fluid at possible sites and draining them. Excessive fluid administration should be avoided and "small volume resuscitation" should be used whenever applicable. Another therapeutic target is edema of the abdominal wall which causes reduction in the compliance. The strategy of treatment is to reduce the volume of fluid in the interstitial compartment without jeopardizing "effective circulatory volume". Though this can be achieved in theory by using hypertonic solutions viz., albumin to expand the intravascular volume and to promote fluid flux from the interstitial compartment into the intravascular space and then removing the excess intravascular volume using diuretics. "Some positive results have been reported by the combination of albumin and frusemide. This approach can only be used when renal function is sufficient to respond to diuretic administration". Usually it is the kidney which is usually influenced by IAH even when the "intra abdominal pressure is less than 20mmHg even before overt ACS" and thus combining a diuretic and albumin is of meager benefit.

"Another alternative in patient with poor renal function is renal replacement therapy with aggressive ultrafiltration therapy" and this can give a better control of fluid management. Studies have shown good fluid control along with a positive effect on respiratory parameters and IAP on using "CVVH with ultrafiltration". RRT is also postulated to provide an added benefit as it can filter the cytokines from the blood. This effect of CRRT has been described in a cases of acute pancreatitis by a reduction in both IL-6 levels from the plasma and intra abdominal pressure.

WSACS also provides many nonsurgical options in the treatment of increased IAP not essentially pertaining to fluid management alone. But whenever these interventions failed and there is a trend towards developing ACS, surgical decompression should not be hesitated. Nevertheless there is a uniformly prevailing trend to delay decompressive surgery and this reluctance from the surgeon's side may be partly due to the "practical consequences of DL in terms of fluid loss through the open abdomen, difficult wound dressings, risk of infection or fistula, reinterventions, cost and longer hospital stay. However, Cheathem et al. "demonstrated that physical, social and mental health has been

restored to the level of general population after abdominal wall reconstruction and Dl does not lead to permanent disability or unemployment".

The complications arising from full-fledged DL have prompted surgeons to look for less invasive methods of abdominal decompression. Newer endoscopic techniques like the "component separation" and also the "subcutaneous anterior abdominal closure" have been described and these might also prove to be an effective replacement in the DL in the near future.

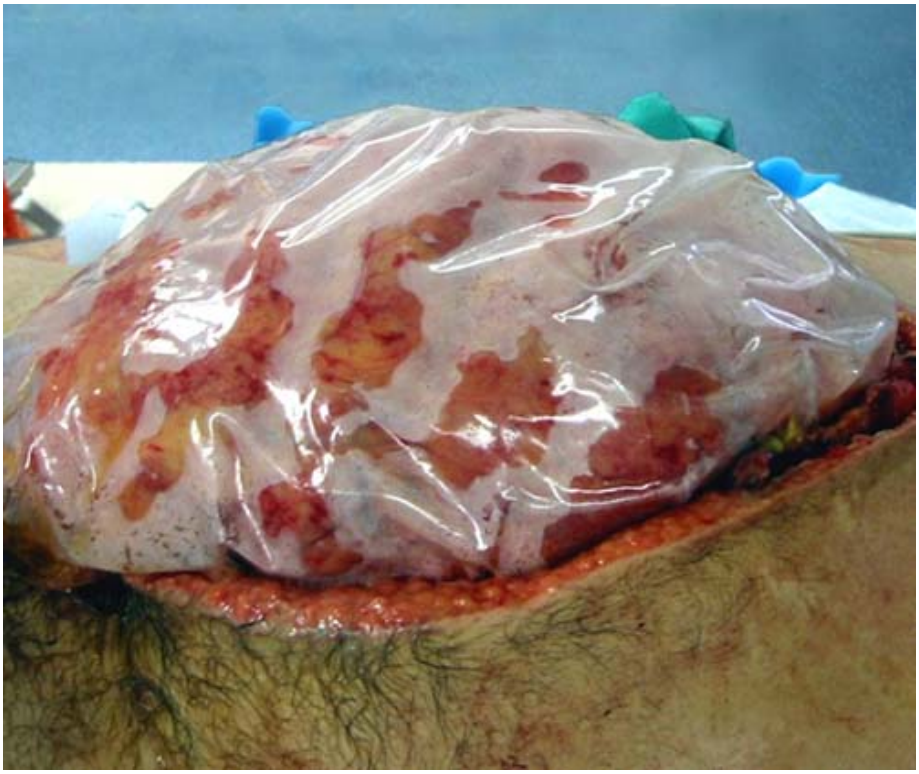


Fig.14 The Whittman patch technique of Decompressive Laparotomy

MATERIALS AND METHODS

DESIGN OF STUDY: Experimental study

PERIOD OF STUDY: 1 year

COLLABORATING DEPARTMENT: NIL

SELECTION OF STUDY SUBJECTS:

All surgical in-patients of Govt. Rajaji Hospital during the study period satisfying the inclusion criteria planned for elective/ emergency laparotomy were recruited for the study after obtaining valid consent. Patients were randomly allotted into study and control groups.

SAMPLE SIZE

84 patients were enrolled for the study after obtaining valid consent. The patients were randomly allotted to case and control groups.

Total sample size (n) = 84

No of cases = 26

No. of controls = 58

No. of dropouts = NIL

ETHICAL CLEARANCE: Applied for approval

CONSENT: Individual informed and written consent

CONFLICT OF INTEREST: None

FINANCIAL SUPPORT: Nil

PARTICIPANTS: All surgical in-patients of Govt. Rajaji Hospital during the study period satisfying the inclusion criteria who are planned for elective/ emergency laparotomy will be recruited for the study after obtaining valid consent.

DATA COLLECTION AND INTERVENTION:

a. Patients under the study group were subjected to non-invasive IAP measurements using indwelling foley's catheter and the fluid manometry technique. Single pre-operative measurement and post operative measurements at 0, 6, 24 and 48 hours were recorded.

b. In cases of increased postoperative IAP in study group (>12 mmHg), steps to reduce IAP were carried out according to World Society of the Abdominal Compartment Syndrome(WSACS) protocols.

c. Incidence of complications like wound/ rectus dehiscence, intra abdominal collection, anastamotic leak and secondary peritonitis among the study and control groups were noted.

INCLUSION CRITERIA

1. All patients more than 18 years admitted in surgical wards and planned for emergency/ elective laparotomy.

EXCLUSION CRITERIA

1. Patients with pre-existing conditions that prevent insertion of a Foley's catheter or pre-existing lesions of urinary bladder.

2. Patients where bladder injury is suspected.
3. Patients where rectus closure is not done in the same sitting.

RESEARCH HYPOTHESIS

Increased IAP in the postoperative period can serve as an early indicator of postoperative morbidity. Optimization of intra-abdominal pressure in the postoperative period thereby can prevent rectus dehiscence, have a positive effect on wound/anastamotic healing, thereby improving the outcome.

STATISTICAL ANALYSIS

The data were analysed using statistical software like SPSS Ver.13.0, Microsoft Excel 2010.

Chi Square test was used to analysed the correlation between the incidence of complications in case and control subsets. Also, individual complications were assessed and p-value for each of them was computed.

RESULTS

AGE AND GENDER DISTRIBUTION

The following table shows the age distribution between case group.

Age group	No. of cases
<20	3
21 - 40	6
41 - 60	11
61 - 80	4

Table 1. Age distribution among cases

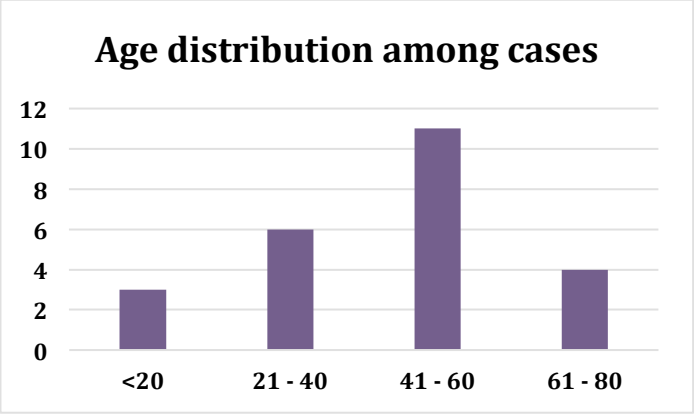


Fig.15 Age distribution among cases

The following table shows the age distribution between control group.

Age group	No. of cases
<20	3
21 - 40	13
41 - 60	33
61 - 80	19

Table 2. Age distribution among controls

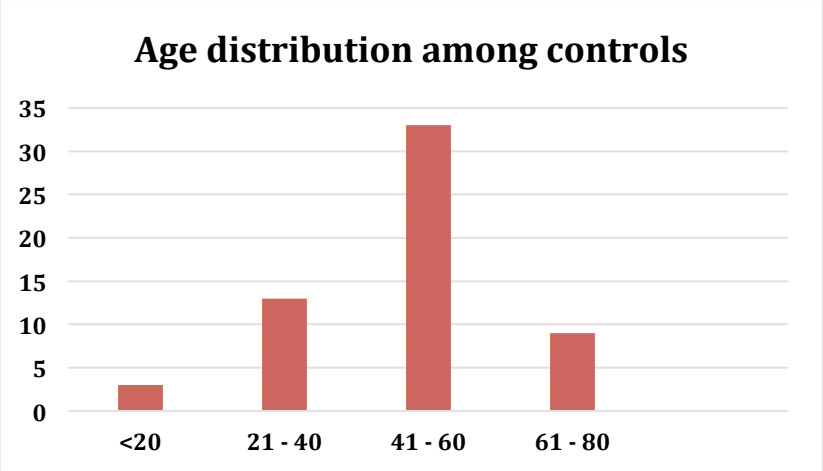


Fig.16 Age distribution among controls

The following table shows the gender distribution among cases and controls

	Male (%)	Female (%)
Cases	73	27
Controls	72	28

Table 3. Gender distribution among cases and controls

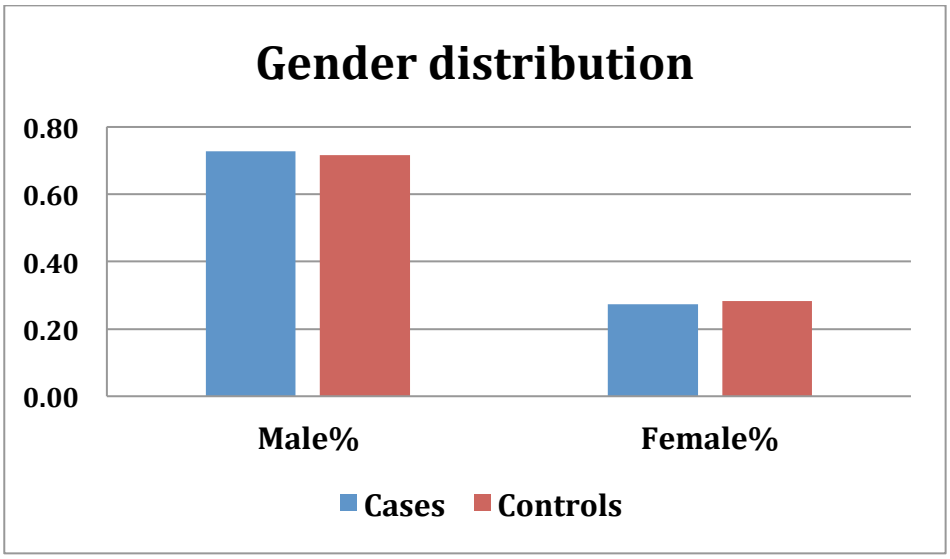


Fig.17 Gender distribution among cases and controls

NATURE OF SURGERY PERFORMED

The following table displays the number of elective and emergency cases distributed among the two study groups

Type of surgeries	Cases	Controls
No of Electives	10	20
No of Emergencies	18	38

Table 4. No. of elective and emergency cases

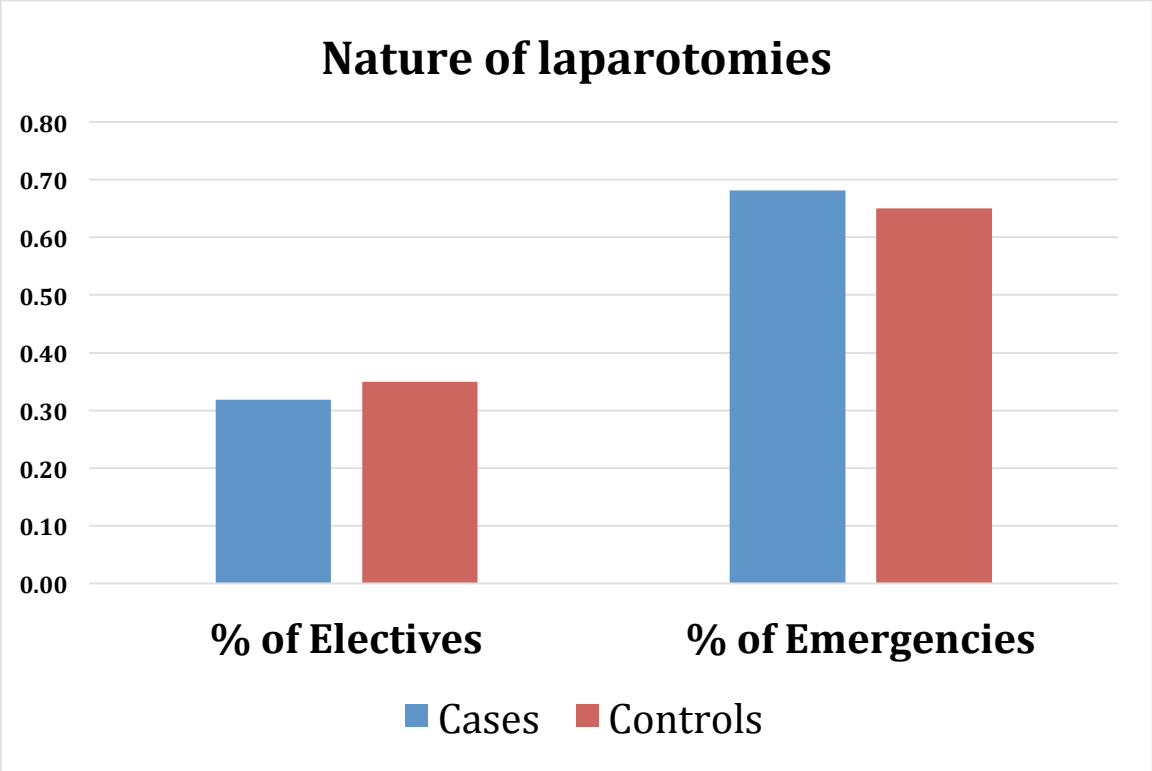


Fig.18 No. of elective and emergency cases

DISTRIBUTION OF INTRA-OPERATIVE FINDINGS

The following table shows the distribution of intra-abdominal findings among cases and controls

	adhesions	contamination	gangrene	mass abdomen	pregangrene	advanced malignancy	obstruction	Total
Cases	2	14	3	6	1	0	0	26
Controls	4	28	2	16	0	1	7	58
Cases(%)	8%	54%	12%	23%	4%	0%	0%	1
Controls(%)	7%	48%	3%	28%	0%	2%	12%	

Table 5. Distribution of intra-abdominal findings

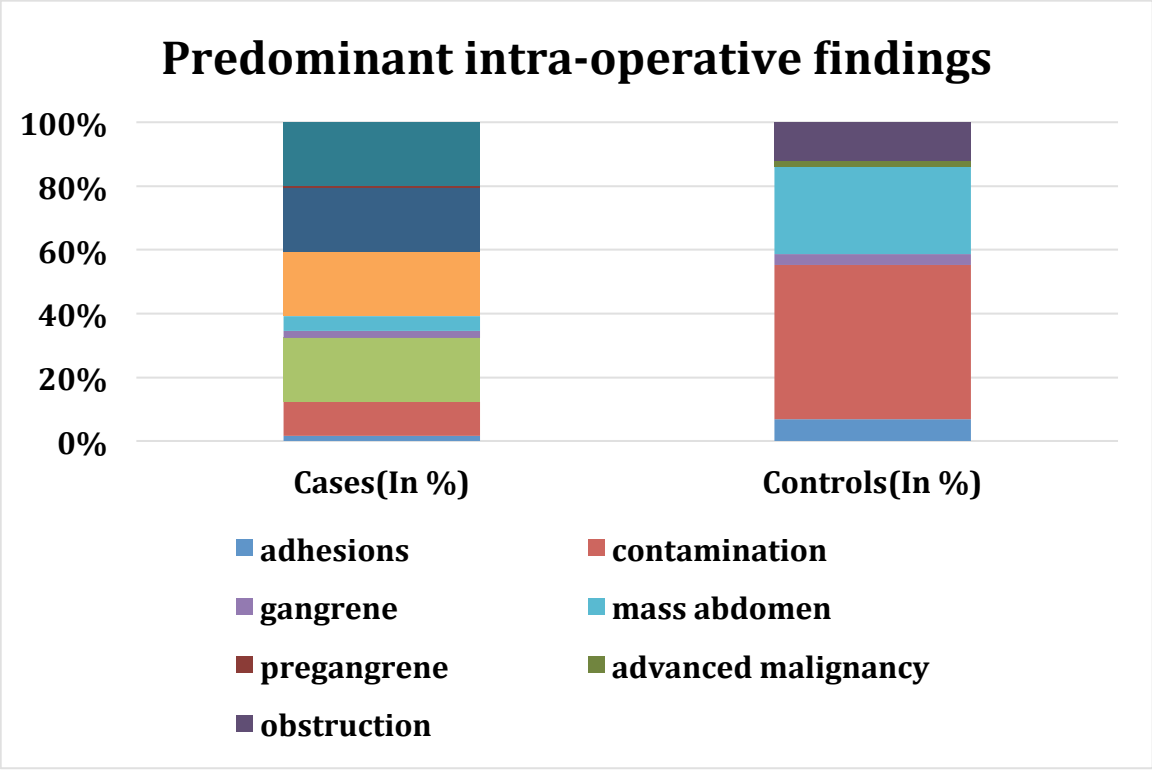


Fig.19 Distribution of intra-abdominal findings

INCIDENCE OF INCREASED IAP IN THE PRE OPERATIVE PERIOD

The following figure shows the incidence of increased IAP in the pre-operative period

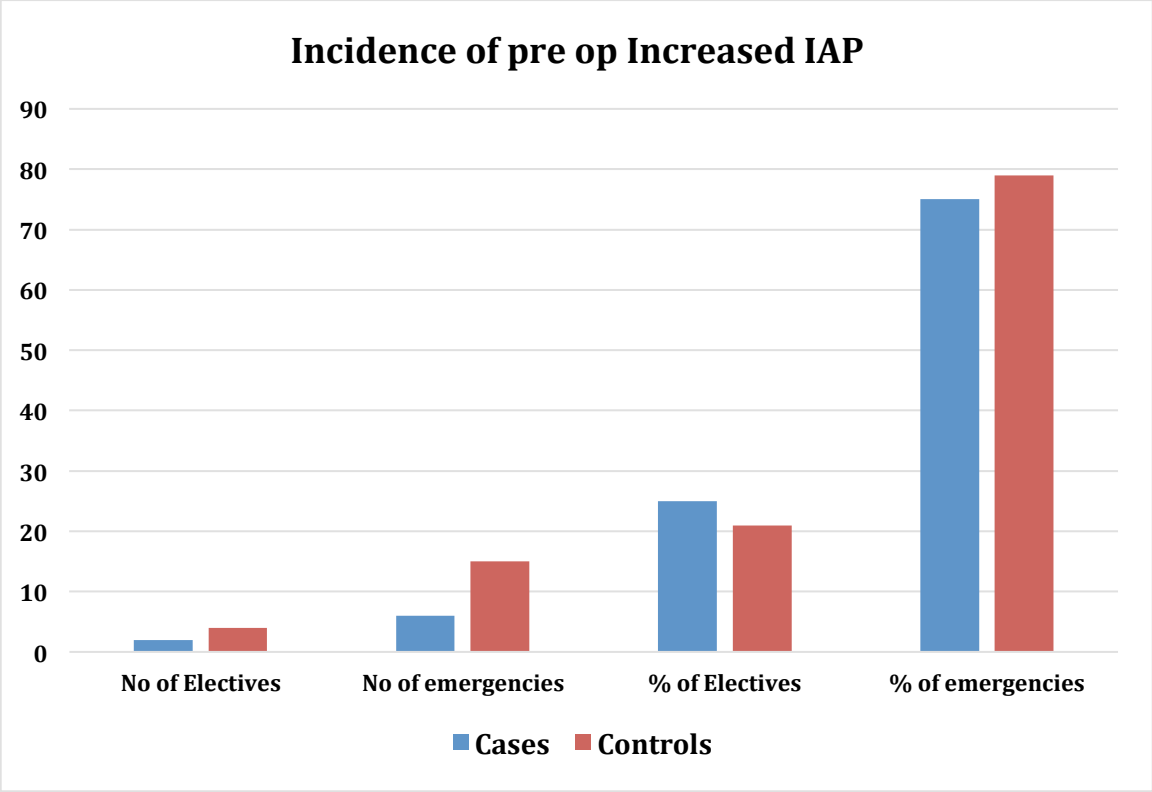


Fig.20 Incidence of increased IAP in the pre-operative period

ANALYSIS OF INTERVENTION TO REDUCE IAP

The following figure shows the number of patients with increased IAP at 0, 6, 24 and 48 hours of post operative period

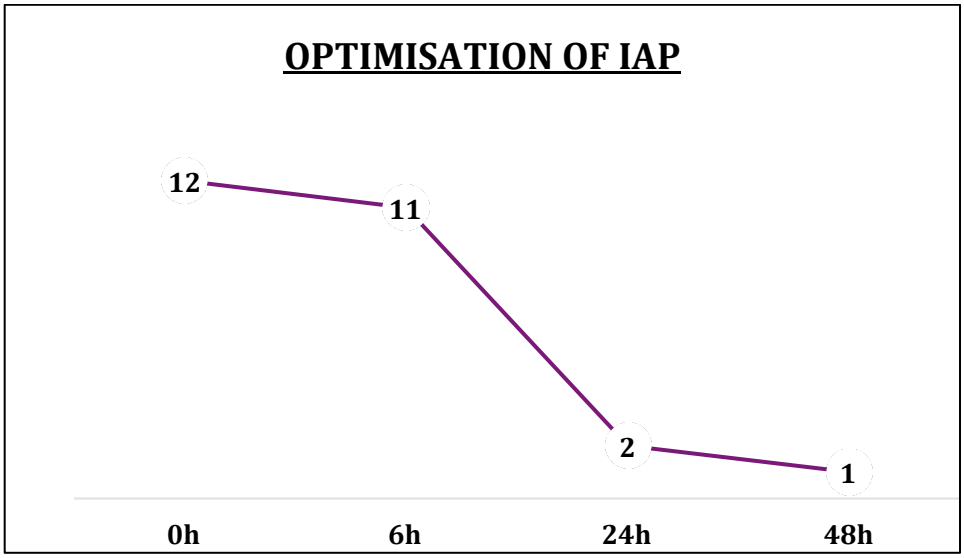


Fig.21 No. of patients with IAH at 0, 6, 24 and 48 h (after intervention)

CORRELATION OF COMPLICATION RATE

The following table shows the correlation between the overall number of complications between cases and controls

Total Complications	Scenarios	No of complications	No complications	Total
	Cases	6	20	26
	Controls	28	30	58
	Totals	34	50	84
	Chi-square score	4.731471368		1
	P-Value	**0.029615**		Significant

Table 6. Correlation of the overall complication rate between the two study groups

CORRELATION OF WOUND AND RECTUS DEHISCENCE

The following table shows the correlation between the no. of wound and rectus dehiscence between cases and controls

Wound and rectus dehiscence	Scenarios	No of complications	No complications	Total
	Cases	4	22	26
	Controls	22	36	58
	Totals	26	58	84
	Chi-square score	4.270064519		1
	P-Value	**0.038789**	Result	Significant

Table 7. Correlation of incidence of wound and rectus dehiscence between the two study groups

CORRELATION OF ANASTAMOTIC LEAK

The following table shows the correlation between the no. of wound and rectus dehiscence between cases and controls

Type 3	Scenarios	No of complications	No of no-complications	Total
	Cases	0	26	26
	Controls	2	56	58
	Totals	2	82	84
	Chi-square score	0.918418839		1
	P-Value	**0.337895**	Result	Insignificant

Table 8. Correlation of incidence of anastomotic leak between the two study groups

DISCUSSION

This study aims at primarily finding the incidence of increased IAP in the pre operative period and post-operative period among patients undergoing elective and emergency laparotomies. Also, it aims correlating whether optimizing IAP in the post operative period improves the outcome of the patient. For analysis of the patient outcome, the following complications were taken into account

1. Wound dehiscence
2. Rectus dehiscence
3. Anastomotic leak
4. Peritonitis

The details of the patients and the nature of surgery were recorded for all the subjects of the study. A pre-operative value of intra-abdominal pressure was recorded. The intra operative findings were noted and the patients were randomly assigned into case and control groups. In both the groups, routine IAP measurement was done in the post-operative period at 0, 6, 24 and 48 hours. In the case group, measures to reduce the IAP were carried out according to WSACS protocols in a stepwise manner. The results of our statistical analysis are summarized as follows

Case and control groups

Our statistical analysis shows that the case and control groups are very much comparable in terms of age and gender.

Elective and emergency

The number of elective and emergency cases were also comparable among both cases and controls.

Intra op findings

The intra operative findings were grouped into 5 broad categories by their significance to the risk of post operative intra-abdominal hypertension. The categories by which the intra-op findings were grouped are

1. Intra-peritoneal contamination with gross amounts of fluid/ feculent material
2. Intra-peritoneal adhesions thereby causing dilatation of bowel loops/ bowel edema
3. Vascular lesions of the bowel (gangrene or pregangrene) or intra-abdominal viscera

4. Intra peritoneal mass lesions which include advanced malignancies
5. Obstructive lesions of the bowel (not included in no.2) eg. Volvulus of the intestine

On analysis, it was found that majority of the patients in both the groups had either findings of peritoneal collection/ contamination or mass lesions in the peritoneal cavity. Vascular lesions of the bowel were the next common intra- operative finding.

Incidence of IAH in the pre-operative period

The incidence of increased intra abdominal pressure in the pre-operative period was comparable in both the groups. Among elective laparotomies, the incidence ranged from 21% - 25% whereas in emergency laparotomies, the incidence was as high as 75 – 79 %

Intervention to reduce the IAP

Interventions to reduce the IAP in the post-operative period were carried out according to the WCACS protocols and in a step-wise manner. The

number of patients who developed IAH in the post-operative period (0 hour value) and eventually the number of patients with IAH at 6, 24 and 48 hours were analysed. It is seen that the interventions were effective in reducing the intra-abdominal pressure as only 2 patients with IAH were recorded at the end of 24 hours. In those two patients, the cause for increased IAP could not be made out even with imaging and one of them, where IAP was persistently elevated even at 48 hours collapsed before decompressive laparotomy could be done.

Correlation of rate of complications among cases and controls

When the overall complication rate was analysed between cases and controls, there was a significant reduction in the complication rate among cases, which was shown by a p value of 0.029.

Also, when the abdominal wall complications of IAH like wound and rectus dehiscence were analysed together, the reduction of complication among the case group was significant, indicated by a p value of 0.038

However, the incidence of anastomotic leak among the two groups was not showing any statistical significance. None of our study patients developed secondary peritonitis in the post-operative period and hence the significance of the same could not be analysed.

Limitations of the study and future scope

(i) The study population could have been augmented to have made it more statistically significant

(ii) Analysis of the effect of post operative ventilation on increased IAP was not done and whether the increase in IAP in these patients need to be intervened as aggressively as patients in room air, has to be further studied.

(iii) The duration of hospital stay was not taken as an indicator of outcome as it is more subjective and varies according to the surgical unit protocols. However, a record of the post operative parameters like reduction in nasogastric aspirate, return of bowel function etc., could have been noted for the study patients to analyse the effect of optimization of post operative IAP even more clearly.

(iv) Effect of IAH on other organ systems were not analysed.

Pilot study

Similar study was conducted by Khan et al have conducted a similar study of the correlation between intra-abdominal hypertension and outcome of emergency laparotomies. However, elective cases were excluded from the study

and no intervention was done to reduce the IAP, if it was found to be elevated. The sample size was 197 and they have seen that 80% of the patients overall had increased IAP at presentation

There was no significance between IAH and occurrence of burst abdomen. Raised intra-abdominal pressure at 6h was a negative predictor of the duration of hospital stay. Pre-op, post-op, 0 hour and 6 hour values were a better predictor of mortality. Incidence of post operative ACS was 3.05 in the general population and 13.16% in trauma patients.

CONCLUSION

This is an experimental study to analyse the effect of optimization of post-operative IAP on the outcome of laparotomies, both elective and emergency. The study population comprised of 84 patients and was carried out over a period of one year. 26 of the patients were taken as cases and the remaining 58 as controls. IAP was monitored both pre-operatively and post operatively and in cases, the increase in IAP post operatively was countered using graded interventions as recommended by the WCACS.

On analysis it was found that the incidence of pre operative IAH was 21 % – 25% in elective cases and 75% – 79% in cases of elective laparotomies. This highlights the importance of vigilant fluid resuscitation in the stabilization phase. Overzealous fluid resuscitation in these patients where the IAP is already elevated would only further worsen end organ failure. Thus routine measurement of IAP at the time admission is highly warranted as it can serve as a very efficient tool to guide fluid administration

Though the overall complication rate is improved by the optimization of IAP in the post-operative period, only the abdominal wall complications were significantly reduced. Thus, preventing IAH in the post-operative period could have a positive effect on wound and rectus healing. In our study, the incidence of anastomotic leak was not significantly altered in both the groups. Larger samples are required to clearly demarcate the effects of optimal post-operative IAP in preventing anastomotic leaks.

Thus from the study we can draw the following conclusions

(i) IAP can be easily monitored using bedside instruments and a routine monitoring is mandatory in patients undergoing abdominal surgeries

(ii) IAP at the time of admission can also serve as a reliable tool to plan the type fluid resuscitation

(iii) Post operative complications like wound and rectus dehiscence can be avoided if IAP is maintained at optimal level. This can be done using graded interventions given by the WSACS

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PROFORMA

Date: **STUDY/ CONTROL**

Name: Age : Sex:

IP no: Height: Weight:

Pre-operative diagnosis:

DETAILS OF SURGERY

Date of surgery:

Procedure done: Elective/ Emergency

Duration of Surgery(hours):

Intra operative findings:

IAP	Pre-op	0	6	24	48

INTERVENTION REQUIRED:

None

Analgesics/ Muscle relaxants Additional imaging

Bowel decompression Re-laparotomy

POST OPERATIVE COURSE

Intensive care (No of days):

Elective Ventilation: If yes, duration(hours):

Complications if any (immediate Post op period):

KEY TO MASTER CHART

Elective/ Emergency	1 = elective 2 = emergency
IAP_inc_Y/N	1 = yes 2 = no
Intervention	0 = no intervention 1 = analgesics/ muscle relaxants 2 = rectus tube/ flatus tube 3 = additional imaging 4 = re-laparotomy
Complications	0 = no complications 1 = wound dehiscence 2 = rectus dehiscence 3 = anastomotic leak 4 = peritonitis 5 = death

MASTER CHART - CASES

sl	name	age	M/F	IP no	diagnosis	Intra-op	Elective/emergency	duration	IAP_inc_Y/N_preop	IAP_inc_Y/N_0h	IAP_inc_Y/N_6h	IAP_inc_Y/N_24h	IAP_inc_Y/N_48	ventilation_(hours)	intervention_(grade)	complications
1	vellaisamy	55	M	1114050	Periampullary growth	periampullary growth	1	8.5	1	2	1	2	2	24	2	0
2	richard arulsamy	48	M	1120432	acute mesenteric ischemia	frank gangrene	2	4.5	2	2	1	1	1	0	4	5
3	karupaiya	80	M	1123213	perforation DU	peritoneal contamination	2		1	2	1	2	2	0	1	0
4	karupaiya	45	M	1123214	obstructed incisional hernia	pregangrene	2		2	2	2	2	2			0
5	jeeva rathinam	42	F	1121747	ca stomach	mass abdomen	1	5	2	2	2	2	2		0	0
6	subha	25	F	1126018	ileocolic intussusception	gangrene	1	3	2	2	2	2	2	0	0	0
7	sikkander	30	M	1127680	post appendicectomy fecal fistula+ ileal	contamination	1	3.5	2	1	1	2	2	0	1	0

					perf											
8	parvathy	63	F	1176812	ca stomach + ca colon	mass abdomen	1	5.5	2	2	2	2	2	0	0	0
9	raju	60	M	1127623	intestinal obstruction	adhesions	2	2.5	2	2	2	2	2	0	0	0
10	veerapandi	40	M	1130784	perforation DU	contamination	2	2	2	1	1	2	2	0	2	0
11	kathiresan	25	M	1114422	DU perforation	contamination	2	2.5	1	2	1	1	2	24	2	1
12	bhavani	32	F	1120927	enterocutaneus fistula	adhesions	1	3	1	1	1	2	2	0	1	0
13	seethalakshmi	38	F	1132273	penetrating injury abdomen	contamination	2	2	2	1	1	2	2	0	1	0
14	suryaprakash	16	M	1135832	perforation DU	contamination	2	3	1	1	1	2	2	24	2	1
15	mookammal	70	F	1135810	perforation DU	contamination	2	3	2	1	1	2	2	24	2	5
16	muthukumar	29	M	1136225	penetrating injury abdomen with omentum herniating	contamination	2	2	2	1	2	2	2	0	1	0
17	murugan	26	M	1120418	DU perforation	contamination	2	2.5	1	1	2	2	2		1	0
18	ponnusamy	70	M	1134592	carcinoma stomach	mass abdomen	1	4	2	1	2	2	2	24	1	0
19	anandha kumar	27	M	1124500	blunt injury abdomen	contamination	2	3.5	1	1	2	2	2	0	1	2
20	mari kumar	31	M	1132501	penetrating injury	contami	2	3	2	1	1	2	2	0	1	0

					abdomen	nation										
21	sabari	19	M	1122702	penetrating injury abdomen	contamination	2	3	2	2	2	2	2	0	2	0
22	rajaguru	17	M	1124503	small bowel gangrene	gangrene	2	3.5	1	1	2	2	2	24	1	2
23	subbammal	65	F	1124504	hepatic flexure growth	mass abdomen	1	2.5	2	2	2	2	2	0	0	0
24	pothumponnu	24	F	1124345	Appendicular Abscess	contamination	2	2	2	2	2	2	2	0	1	0
25	kuruvammal	70	F	1125209	carcinoma stomach with gastric perforation	contamination	1	3	2	2	2	2	2	24	0	0
26	alagarsamy	50	M	1125328	Acute intestinal obstruction	contamination	2	3.5	2	2	2	2	2	0	0	0

MASTER CHART - CONTROLS

sl no	name	age	M /F	IP no	diagnosis	Procedure	intra - oop	Elective/emergency	IAP_inc_Y/N_preop	IAP_inc_Y/N_0h	IAP_inc_Y/N_6h	IAP_inc_Y/N_24h	IAP_inc_Y/N_48	complications
1	Mohammed sheik	30	M	1121826	penetrating injury abdomen with jejunal perforation	emergency laparotomy and primary closure	contamination	1	2	2	1	2	2	1
2	chinnasamy	47	M	1044996	sigmoid volvulus with fecal peritonitis	emergency laparotomy with Hartmann's procedure	gangrene	2	2	2	2	2	2	1
3	rajendran	49	M	1132542	intestinal obstruction	emergency laparotomy with segmental resection Anastomosis	adhesions	2	1	2	2	2	2	0
4	sivakumar	35	M	1479800	penetrating injury abdomen with jejunal perforation	emergency laparotomy with resection anastomosis	contamination	2	1	2	2	2	2	2
5	muruganantham	45	M	4569300	perforation peritonitis - DU perforation	laparotomy and patch closure	contamination	2	2	1	1	2	2	1
6	chellammal	80	F	1078884	intestinal obstruction	sigmoidopexy with peritoneal lavage and	contamination+	2	1	2	2	2	2	0

					with septicaemia	cholecystectomy	obstruction							
7	valli	60	F	1079678	carcinoma descending colon	left Hemicolectomy	mass abdomen	1	1	1	2	2	2	5
8	ramasamy	55	M	1153820	Blunt injury abdomen with perforation	laparotomy with ileal resection and Ileo-ileal anastomosis	contamination	2	2	1	1	2	2	0
9	vellaisamy	45	M		DU perforation	Graham's patch closure	contamination	2	2	2	1	1	2	0
10	andavar	46	M	1078891	Carcinoma head of Pancreas	Whipple's procedure	mass abdomen	1	1	1	1	2	2	5
11	velu	55	M	1081418	acute intestinal obstruction with carcinoma descending colon	laparotomy and Hartman's procedure	mass abdomen + obstruction	2	1	1	1	2	2	0
12	veeramalai	24	M	2200400	stab injury	laparotomy	contamination	2	2	1	1	2	2	2
13	arunkumar	18	M	1085594	DU perforation	Graham's patch closure	contamination	2	2	1	1	2	2	1
14	ramasamy	64	M	1087136	perforation peritonitis - prepyloric perforation	omental patch closure	contamination	2	2	1	2	2	2	0
15	venkatesh	15	M	1089886	acute intestinal obstruction with sigmoid volvulus	laparotomy with resection and colocolic anastomosis	obstruction	2	1	1	2	2	2	3

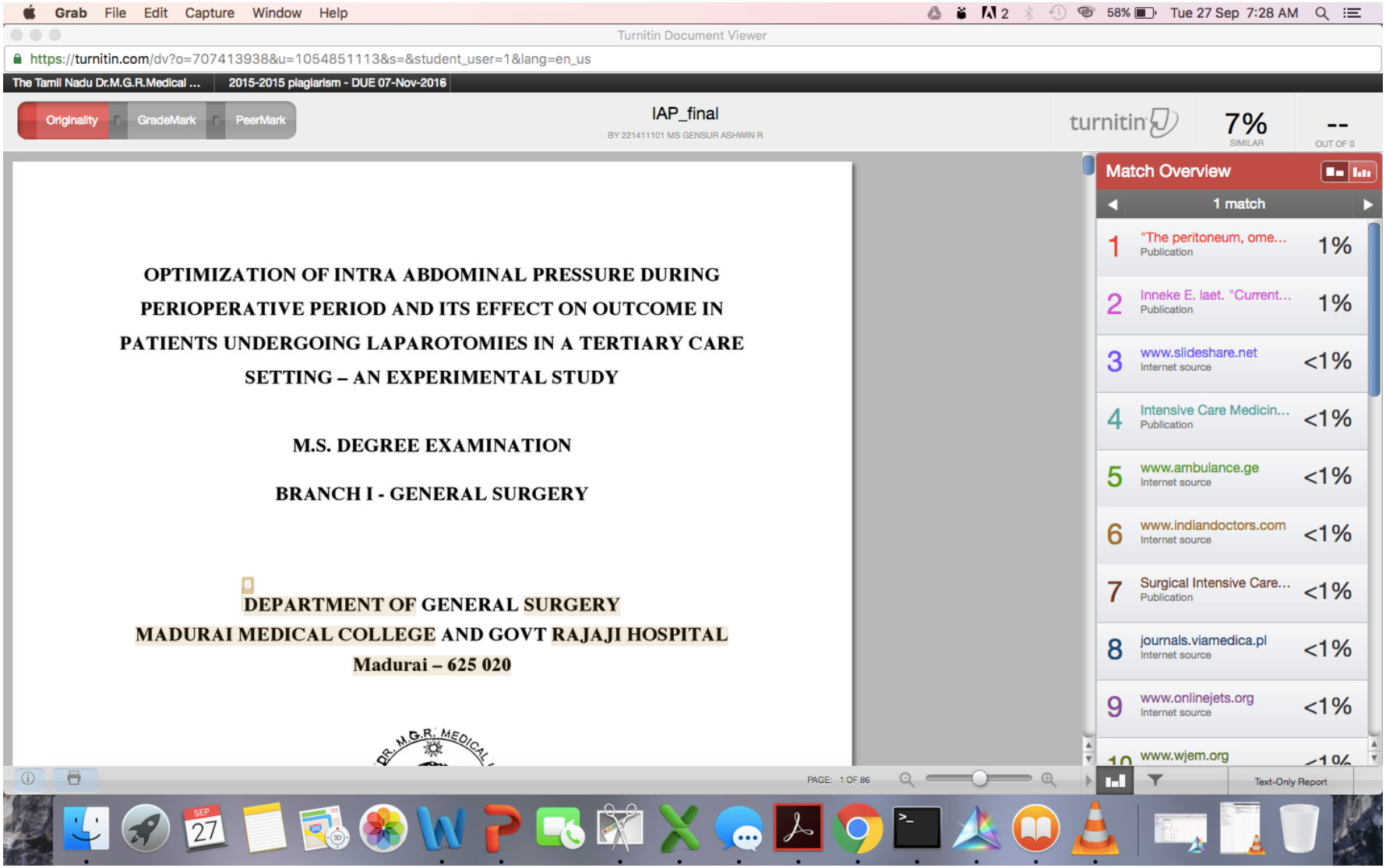
16	Zaheer hussain	29	M	1089238	Acute intestinal obstruction	Ileal resection and Anastomosis	adhesions	2	2	1	2	2	2	0
17	ponnucham y	53	M	4804100	Duodenal perforation with small bowel + cecal gangrene	exploratory laparotomy with omental patch closure and ileocecal resection and ileotransverse anastomosis	gangrene	2	2	1	2	2	2	2
18	ponni	46	F	1090296	carcinoma rectum	Diversion colostomy	advanced malignancy	1	2	1	1	2	2	1
19	ravichandra n	41	M	1092985	DU perforation	Graham's patch closure	contaminati on	2	1	2	2	2	2	2
20	rajathi	40	F		obstructed incisional hernia	emergency laparotomy and Ileal resection anastomosis	contaminati on	2	2	1	2	2	2	0
21	selvaraj	60	M	1095788	perforation peritonitis	laparotomy and primary closure		2	2	2	2	2	2	1
22	sikkandar	44	M	1095727	obstructed incisional hernia	limited resection of caecum with appendix and prox.1/3rd of ascending colon with proximal Ileostomy	contaminati on	2	1	2	2	2	2	2
23	murugan	55	M	1092543	GIST	laparotomy and proceed	mass abdomen	1	2	2	2	2	2	0
24	Irulayee	50	F	1091080	periampullary growth	Whipple's procedure	mass abdomen	1	1	1	2	1	1	5
25	marimuthu	31	M	109743	gastric perforation	omental patch closure	contaminati on	2	2	2	1	2	2	0
26	srinivasan	41	M	1098207	intestinal obstruction	laparotomy and Right Extended	mass abdomen	2	2	2	2	2	2	0

					with hepatic flexure growth	Hemicolectomy								
27	panchavarnam	71	F	1094203	gastric outlet obstruction with cholelithiasis	laparotomy and proceed	mass abdomen	1	2	2	2	2	2	1
28	Raju	40	M	1158800	RTA with Blunt injury Abdomen	resection and anastomosis	contamination	2	2	2	2	2	2	0
29	krishnammal	60	F	1099682	perforation peritonitis	laparotomy and drainage	contamination	2	1	1	1	2	2	1
30	kaliappan	58	M	1098273	gastric outlet obstruction	TV - GJ anastomosis	obstruction	1	2	2	2	2	2	0
31	gurusamy	54	M	1104247	perforation peritonitis	omental patch closure	contamination	2	1	2	2	2	2	
32	muthuirulan	57	M	1105627	Acute intestinal obstruction [complete ileal stricture]	laparotomy and resection Anastomosis	contamination+ obstruction	2	2	1	1	2	2	0
33	balasubramaniam	60	M	1107520	Ruptured liver abscess	emergency laparotomy and Drainage	contamination	2	1	2	1	1	2	1
34	parameswaran	64	M	1104209	Carcinoma Stomach	exploratory laparotomy and Palliative AGJ + JJ &biopsy	mass abdomen	1	2	1	1	2	2	0
35	rajamani	76	M	1105508	periampullary carcinoma	Triple Bypass	mass abdomen+ contamination	1	2	1	1	2	2	0

36	vasantha	50	F	1108511	post gastrectomy Efferent loop obstruction	laparotomy and loop disconnection with Roux en Y anastomosis	mass abdomen	1	2	1	1	2	2	1
37	karuppayee	54	F		carcinoma stomach with gastric perforation	omental patch repair and AGJ	contaminati on	1	2	1	1	2	2	0
38	yagappan	50	M	1152309	Acute intestinal obstruction	Laparotomy and congenital Band Release	adhesions	2	2	1	2	2	2	3
39	babu	44	M		post laparotomy burst abdomen	RE-Laparotomy with FJ and Tension band wiring	relaparoto my	2	1	1	2	2	2	0
40	Chitra	40	F	1112817	DU perforation	laparotomy and Patch closure	contaminati on	2	2	1	2	2	2	0
41	muthukaru ppu	65	M	1108426	Carcinoma Stomach	subtotal gastrectomy with Roux en y GJ	contaminati on	1	2	1	2	2	2	2
42	varnapandi	58	M	1114294	perforation peritonitis	Graham's patch closure	contaminati on	2	1	1	1	2	2	1
43	rajamani	65	M	1112767	Carcinoma Stomach	distal gastrectomy , billroth II anastomosis	mass abdomen	1	2	2	2	2	2	0
44	thangavel	40	M	1117054	carcinoma rectum with liver metastasis	Proximal Diversion colostomy	contaminati on	1	2	1	2	2	2	1
45	Bharathidas an	45	M		biliary peritonitis	laparotomy and primary closure of ileum	contaminati on	1	2	2	2	2	2	1
46	sathya	33	F	1124609	ruptured appendix with	laparotomy and drainage	contaminati on	2	2	2	2	2	2	0

					pelvic abscess									
47	meena	67	F	1127276	irreducible Umblical hernia	laparotomy and mesh repair		2	2	2	2	2	2	0
48	karuppi	55	F		Gallbladder perforation	subtotal cholecystectomy	contaminati on	2	1	2	2	2	2	1
49	nagaraj	45	M	1128910	gastric perforation	laparotomy and omental patch closure	contaminati on	2	2	2	1	2	2	0
50	srinivasan	54	M	1150061	ileal stricture with ileal perforation	exploratory laparotomy with ileal resection and ileotransverse anastomosis	obstruction +contamina tion	2	1	2	2	2	2	2
51	kannan	45	M	1128861	Carcinoma Stomach	palliative Gastrectomy / AGJ Anastomosis	mass abdomen	1	2	2	1	2	2	5
52	kuppu	45	M	1133342	perforation peritonitis	Graham's patch closure	contaminati on	2	2	2	2	2	2	0
53	Jeyaraj	52	M	1131606	Carcinoma head of Pancreas	Whipple's procedure	mass abdomen	1	2	1	1	2	2	0
54	rajammal	60	F	1130509	Right iliac fossa mass	laparotomy and Debulking of tumor with Ileotransverse anastomosis	mass abdomen	1	1	2	2	2	2	0
55	pandiamma l	62	F		Carcinoma stomach	subtotal gastrectomy with GJ	mass abdomen	1	2	2	2	2	2	1
56	valliraja	48	M	1133269	Carcinoma colon with subacute	emergency laparotomy and Hartmann's procedure	mass abdomen	2	2	1	1	2	2	0

					intestinal obstruction									
57	Thavasi	50	M	1136881	strangulated incisional hernia	emergency laparotomy with primary closure of defect	obstruction	2	1	2	1	1	2	0
58	Hariharan	16	M	1137241	perforation peritonitis	laparotomy and omental patch closure	mass abdomen	2	2	1	1	2	2	0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M.S. DEGREE EXAMINATION
BRANCH I - GENERAL SURGERY
DEPARTMENT OF GENERAL SURGERY
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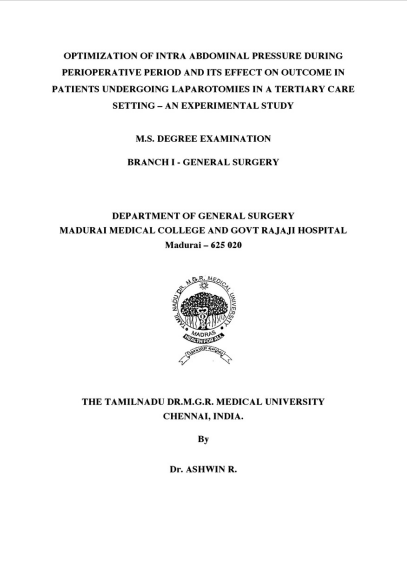

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By

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Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Ashwin. R
Course	:	PG in M.S., General Surgery
Period of Study	:	2014-2017
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Optimization of Intra Abdominal pressure during perioperative period and its effect on outcome in patients undergoing laparotomises in a tertiary care setting – An experimental study
Ethical Committee as on	:	10.06.2016

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

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10 JUN 2016