A STUDY OF ABDOMINAL SURGICAL EMERGENCIES IN GERIATRIC PATIENTS

By

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In partial fulfilment of the requirements for the degree of

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ABSTRACT

Background and objectives

Geriatric population is a special subgroup of population undergoing emergency abdominal surgeries. Both higher age group and emergency surgical procedure are considered as high risk factors.

In this study, we study the most common cause for geriatric population to undergo an emergency abdominal surgery and the therapeutic outcomes.

Results and Observations

64 patients aged 60 years or more who presented with abdominal emergency surgical conditions were studied.

Most common cause for emergency abdominal surgery was perforated peptic ulcer (38%) followed by intestinal obstruction (17%).

The most common post operative complication was surgical site infection (29%). Mortality rate was 17%. Most common cause of death was septic shock with multi organ dysfunction.

Conclusion

Geriatric population is an important subgroup of population undergoing emergency abdominal surgeries. Most common cause is peptic ulcer perforation followed by intestinal obstruction due to adhesions.

More than the age per say, the delay in presentation may be the cause for mortality in this age group. The therapeutic outcome in patients with co morbid factors like hypertension and diabetes mellitus, in good control, were similar to other patients.

CONTENTS

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INTRODUCTION

Characteristics of high-risk population[1];

- Elderly
- Co morbid conditions
- Needing emergency surgery (no time for optimisation).

Life expectancy and the geriatric population have increased steadily in recent decades. By 2030, people more than 65 years of age will account for 20% of overall population[2, 3].

Our society is continuing to age and with luck, fortunately this trend is continuing. Advances in healthcare systems have enabled people to live longer and to remain healthy for a significantly greater amount of time.

Today, major surgical operations are offered to increasing numbers of geriatric patients. As in other surgical specialties, the frequency of digestive operations performed in elderly patients, and even in subgroups of older patients (i.e. ≥80 or ≥85 years) has increased[3].

Senescence or physiological ageing is decreased functional reserve of critical organ systems resulting in decreased ability in coping with operative stress[3]. Patients > 65 years old account for approximately 50 % of all emergent operations and 75 % of operative mortality[3].

Geriatric patients are often viewed as high-risk surgical candidates. Consequently, elective surgery may not be performed, with the result that a potentially treatable disease process may develop into an acute catastrophic event[2, 3]. Surgery in geriatrics poses unique challenge to surgeons and anaesthetists. There are certain types of age related problems in elderly that may affect the outcome of surgery[5].

Gastrointestinal diseases are a frequent cause of morbidity, mortality and hospital admissions in the geriatric[6]. They present with subtle clinical manifestations and life threatening complications[2, 3, 5].

OBJECTIVES OF THE STUDY

Abdominal surgeries in geriatric population are common in general surgery. The study was conducted to evaluate -

1. The common causes for emergency abdominal surgeries in geriatric patients
2. The therapeutic outcome and effect of co-morbid conditions on the same.

REVIEW OF LITERATURE

EMBRYOLOGY OF DIGESTIVE SYSTEM:

The early developing alimentary canal is a tube suspended in the midline of abdominal cavity. The primitive gut forms foregut, midgut and hindgut. The endoderm forms epithelial lining of digestive tract and gives rise to glands. The peritoneal and muscular components of the gut wall are derived from splanchnic mesoderm.

Derivatives – *Foregut*

- Oesophagus, stomach and duodenum
- Liver and gall bladder

Midgut –

- Jejunum
- Ileum
- Proximal 2/3rd of colon

Hindgut –

- Distal 1/3rd of colon
- Rectum and anal canal

Each has got its own source of blood supply –

- *Foregut – celiac artery*
- *Midgut – superior mesenteric artery*
- *Hindgut – inferior mesenteric artery*

Midgut elongates faster than trunk: herniates into umbilical cord in 6th week

Midgut in adult begins immediately distal to the entrance of bile duct into duodenum and terminates at the junction of the proximal two thirds of transverse colon with its distal one third.

At its apex it remains in open connection with the yolk sac by the narrow vitelline duct. The cephalic limb of the loop develops into distal part of duodenum, the jejunum and part of the ileum. The caudal limb becomes the distal ileum, caecum and appendix, the ascending colon and proximal one third of the transverse colon. Junction of cranial and caudal limb in adults can be recognised if there is presence of Meckel's diverticulum.

Rotation of midgut:

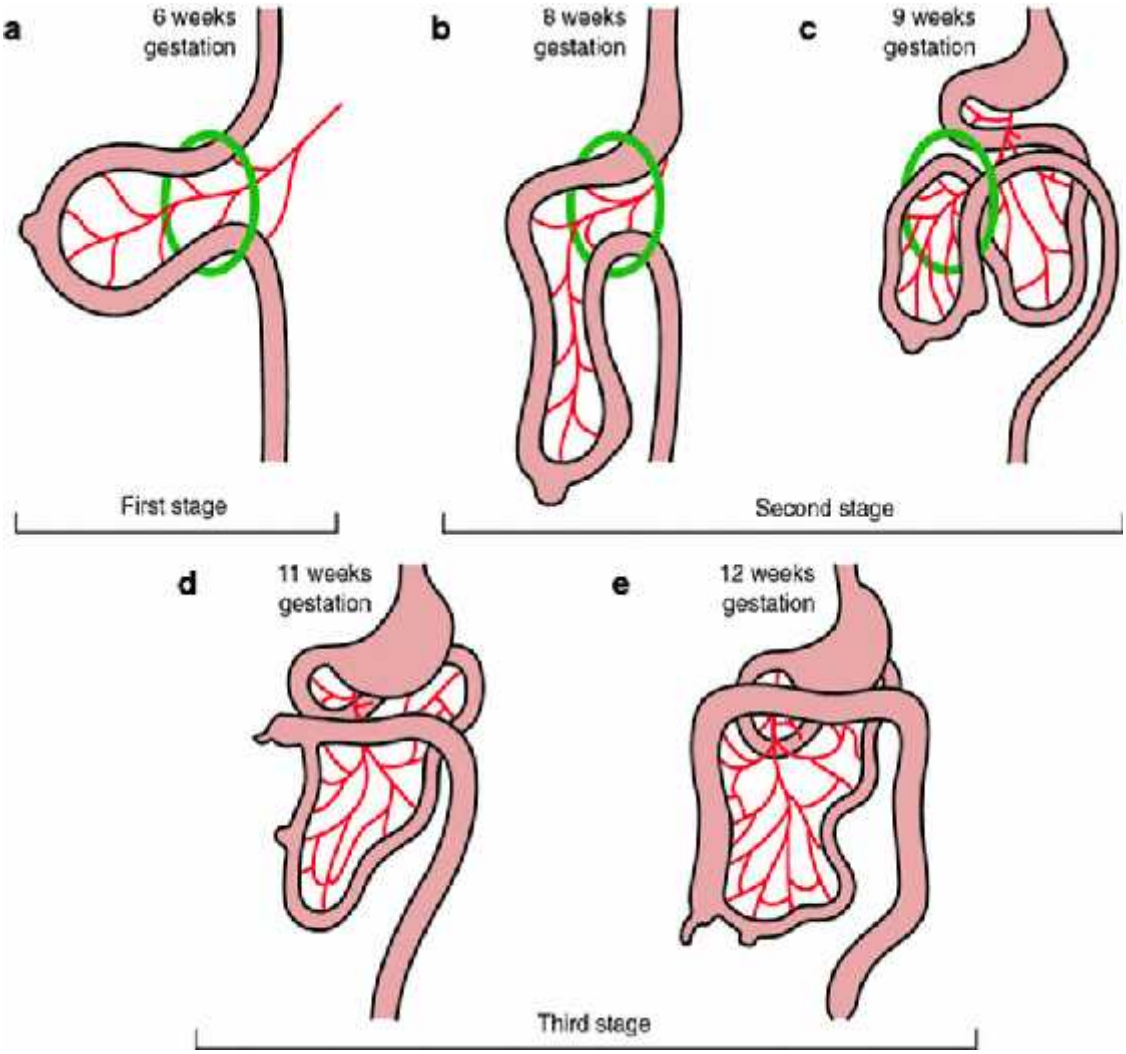
Along with the growth in length, the primitive intestinal loop rotates around an axis formed by superior mesenteric artery. When viewed anteriorly, the rotation occurs in a counter clockwise direction and for approximately 270^{o} when it is completed. Elongation of the small intestinal loop continues during this period, with jejunum and ileum forming a number of coiled loops. The large intestine grows in length considerably but is not involved in the coiling phenomenon. Rotation occurs in stages, first during herniation of loop (about 90^{o}) as well as during the return of intestinal coils into peritoneal cavity (remaining 180^{o}).

At about the end of third month the herniated intestinal loops begin to return to abdominal cavity. Though the precise factors responsible for this are not known, they are thought to be

1. Regression of mesonephros
2. Reduced growth of liver
3. Actual expansion of abdominal cavity

As the intestines return to abdominal cavity, their mesenteries are pressed against posterior abdominal wall and in several cases may fuse with the parietal peritoneum, thus fixing a few intestinal loops against posterior abdominal wall.

Fig.1: Embryology of Gut



ANATOMY

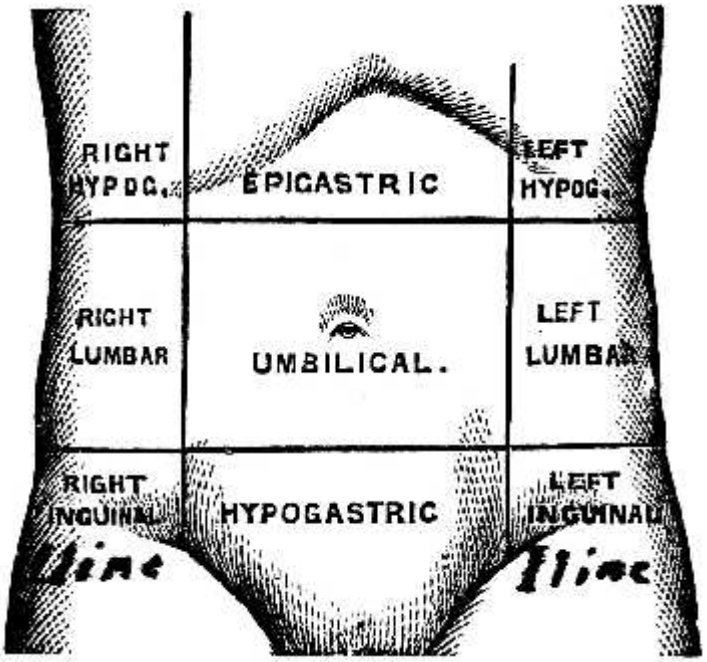
ABDOMEN:

It is an extensive space which extends upwards, deep to the costal margins, into the concavity of the diaphragm and projects downwards and backwards into the bony pelvis as the pelvic cavity. Thus a considerable part of the abdominal cavity is overlapped by the thoracic cage above and by the bony pelvis below.

Regions of abdomen:

Abdomen is divided in nine regions by two vertical and two horizontal lines. Vertical lines are drawn from midclavicular point to midpoint of inguinal ligament on the same side. Horizontal lines are transpyloric line, horizontal line passing through midpoint between xiphisternum and umbilicus. And transtubercular line, a horizontal line passing through midpoint between umbilicus and the pubic symphysis in midline.

Fig.2: Regions of the Abdomen



Abdominal cavity:

Abdominal cavity is an extensive space which extends upwards, deep to the costal margins, into the concavity of the diaphragm and projects downwards and backwards into the bony pelvis as the pelvic cavity. Thus a considerable part of the abdominal cavity is overlapped by the thoracic cage, above and the bony pelvis below.

Boundaries

Superiorly – the diaphragm. It extends to fifth intercostal space superiorly (more inferior around the edges). Hence the superior limit of the liver is also the fifth intercostal space, since it pushes the diaphragm upwards.

Posteriorly – lumbar vertebrae, quadrants lumborum and tranverse abdominis muscles.

Anterolaterally – the muscles of abdominal wall (tranversus abdominis, internal and external oblique).

Inferiorly – pelvic brim.

Fig.3: Boundaries of the Abdominal Cavity

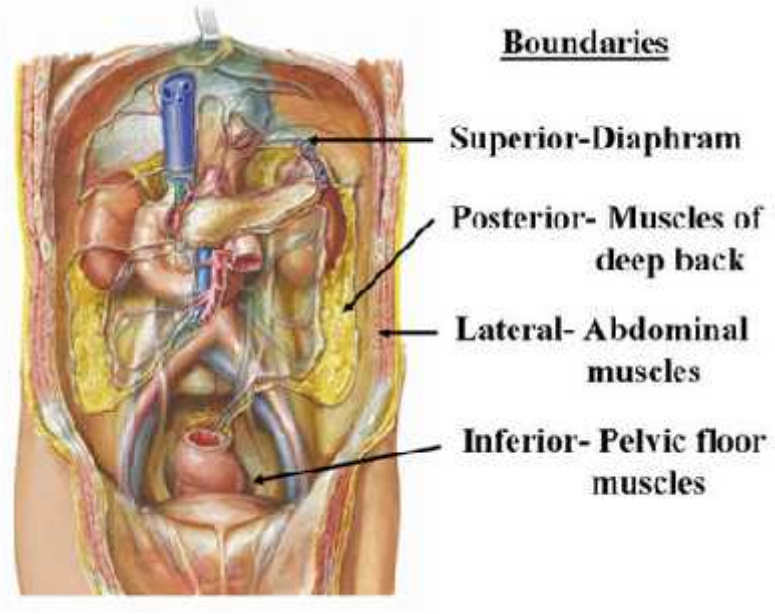
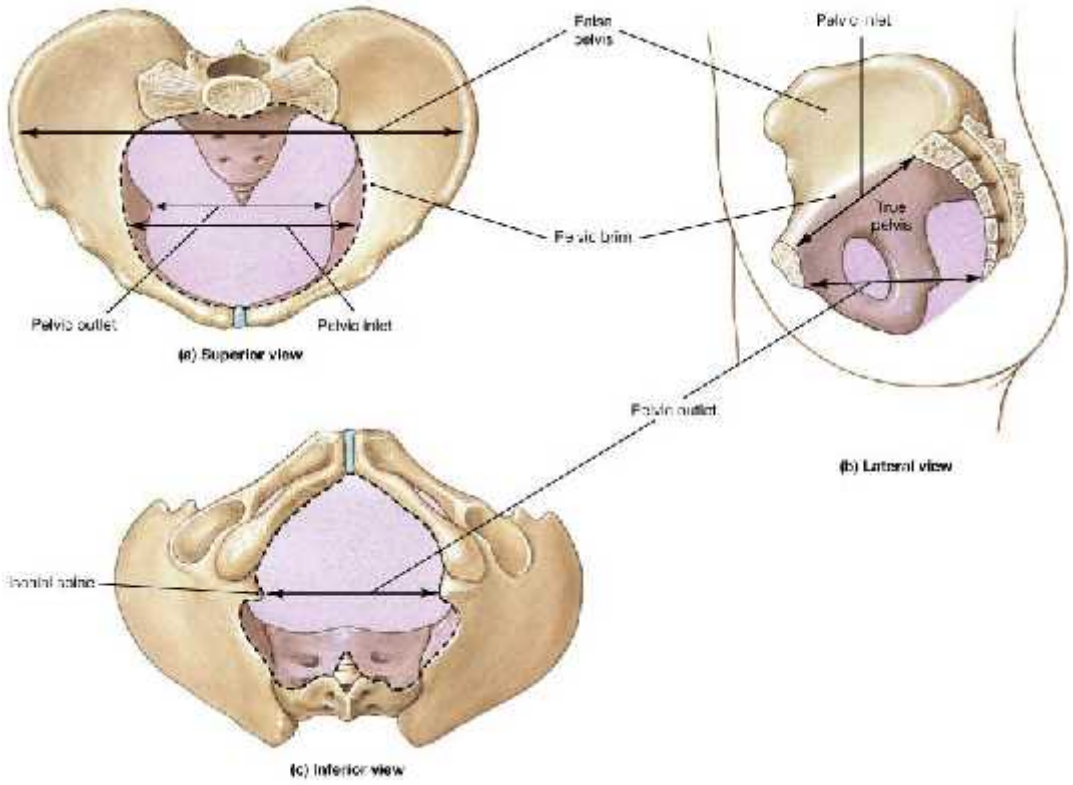


Fig.4: Pelvic Brim – Inferior Boundary of the Abdominal Cavity



Pelvic Brim:

It is the inferior border of the abdomen. It consists of the right and left coxal bones. Each coxal bone is made up of an ilium, ischium and pubic bone.

Iliac crest: the superior portion of the iliac bone. The iliac tubercles are bony prominences on the iliac crest.

Anterior superior iliac spine (ASIS): the anterior most feature on the iliac crest. Pubic tubercle: lateral edge of pubic bone.

Inguinal ligament: found between the ASIS and the pubic tubercle, running in the same direction as the ASIS.

The femoral vessels and the inguinal canal are both related to the inguinal ligament which is formed from the aponeurotic part of the external abdominal oblique muscle.

Layers of the abdominal wall:

Skin

Epidermis- the part we shed

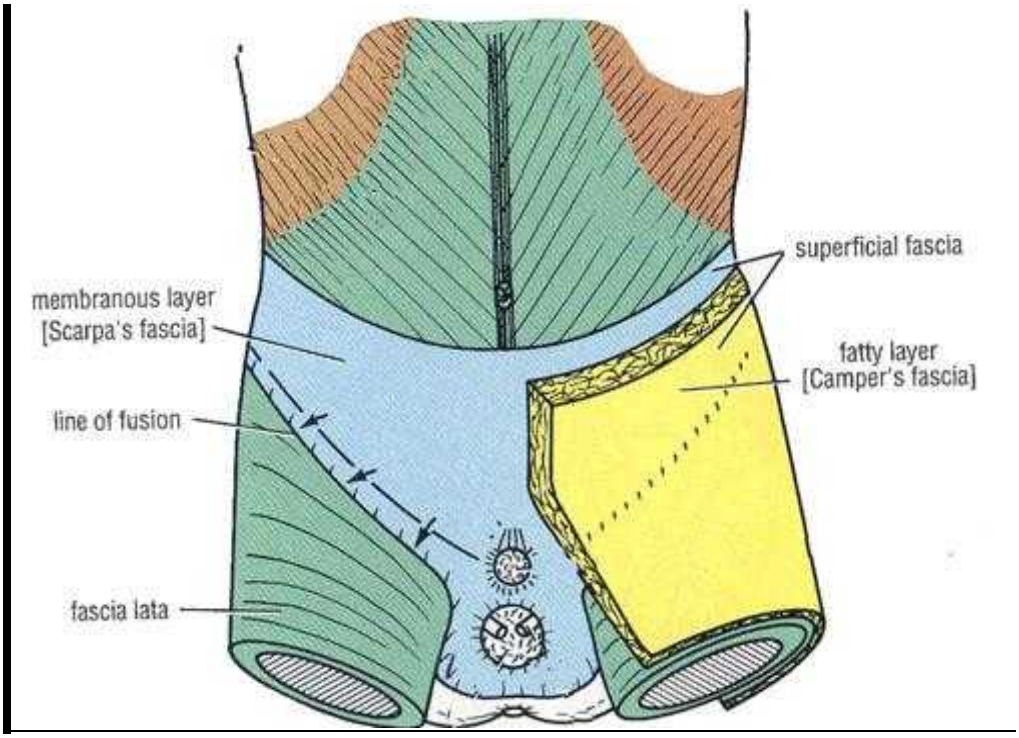
Dermis- contains nerves, capillaries, sweat glands, hair follicles. It has collagen fibres that tend to be horizontal, forming the creasing of the skin. These are called Langer's lines or Kraissl's lines.

Superficial fascia – it is formed by the connective tissue that is not aponeurosis, tendon or ligament. This is the same thing as the hypodermis.

Camper's fascia – fatty layer, first of the two layers. It is found throughout.

Scarpa's fascia – lower layer, found in the lower third of the anterior abdominal wall. It has a restrictive location, defined by the extent of damage occuring with a straddle injury.

Fig.4: Superficial Fascia of Abdominal Wall



Limits:

The area is restricted to the anterior abdominal wall.

Lateral limit: basically the inguinal ligament, where it intersects with fascia lata, so that fluid does not pass into the thigh.

Inferior limit- the base of the scrotum.

Posterior limit- it goes back to the anus and fills the pelvis in between.

The outlined region is called the superficial perineal space.

It is called different fascia at different places: Dartos fascia in scrotum/ labia majora and Colles facsia around perineum.

Fundiform ligament: the false suspensory ligament of the penis or clitoris. It is an extension of superficial fascia.

Deep fascia, a true suspensory ligament occurs in the deep fascia layer, which extends into the penis/ clitoris. So, we have both a true suspensory ligament (deep fascia) and

a false one (fundiform ligament/superficial fascia). Deep fascia encompassess all muscles of the entire body.

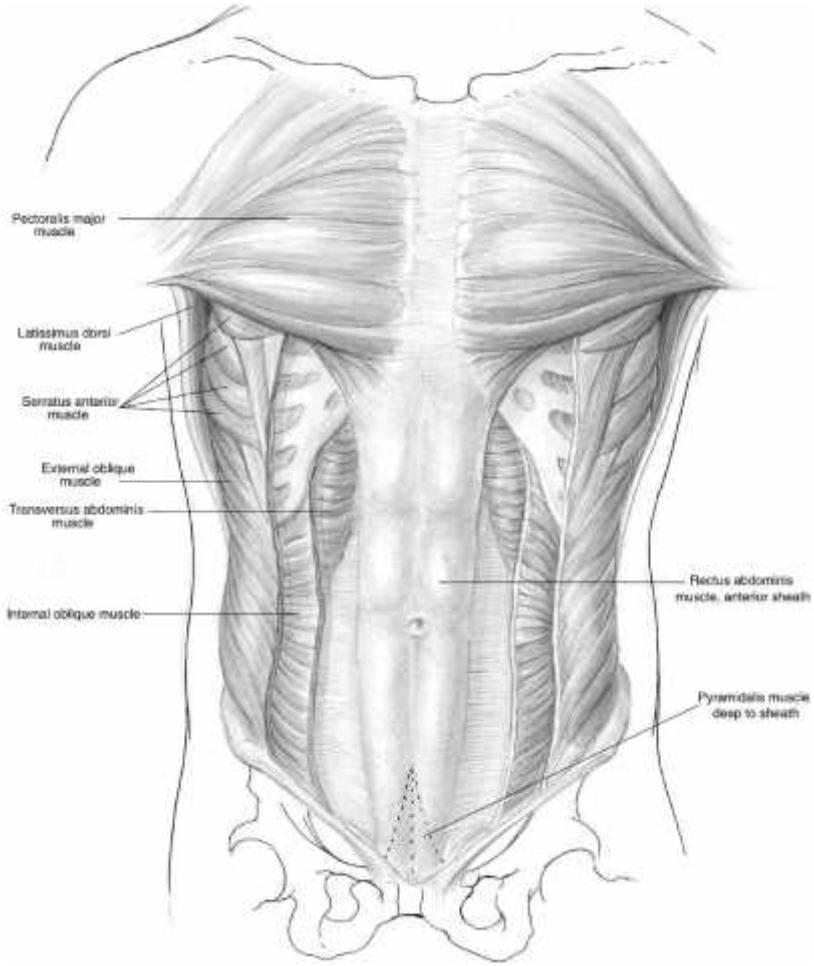
Muscles of the anterior abdominal wall:

Muscles- three flat muscles plus the longitudinal rectus sheath muscle.

Name of muscle	Origin	Insertion	Nerve supply	Action
External oblique	Lower 8 ribs	Xiphoid process, linea alba, pubic crest,pubic tubercle,iliac crest	Lower 6 thoracic nerves and iliohypogastric and ilioinguian nerves(l1)	Supports abdominal contents, compressess abdominal contents.assissts in flexing and rotation of trunk, assissts in forced expiration, micturition, defecation, parturition and vomiting
Internal oblique	Lumbar fascia,iliac crest, lateral two thirds of inguinal ligament	Lower three ribs and costal cartilages, xiphoid process, linea alba, synthesis pubis	Lower 6 thoracic nerves and iliohypogastric and ilioinguinal nerves(l1)	As above
Transversus abdominis	Lower 6 costal cartilages, lumbar fascia, iliac crest, and lateral	Xiphoid process linea alba, symphysis pubis	Lower 6 thoracic nerve and iliohypogastric and ilioinguinal nerves(l1)	Compresses abdominal contents

	third of inguinal ligament.			
Rectus abdominis	Symphysis pubis and pubic crest	5th and 6th costal cartilages and xiphoid process	Lower 6 thoracic nerve	Compresses abdominal contents and flexes vertebral column, accessory muscles of expiration
Pyramidalis (if present)	Anterior surface of pubis	Linea alba	12th thoracic nerve	Tenses the linea alba

Figure 4; Muscles of Anterior Abdominal Wall

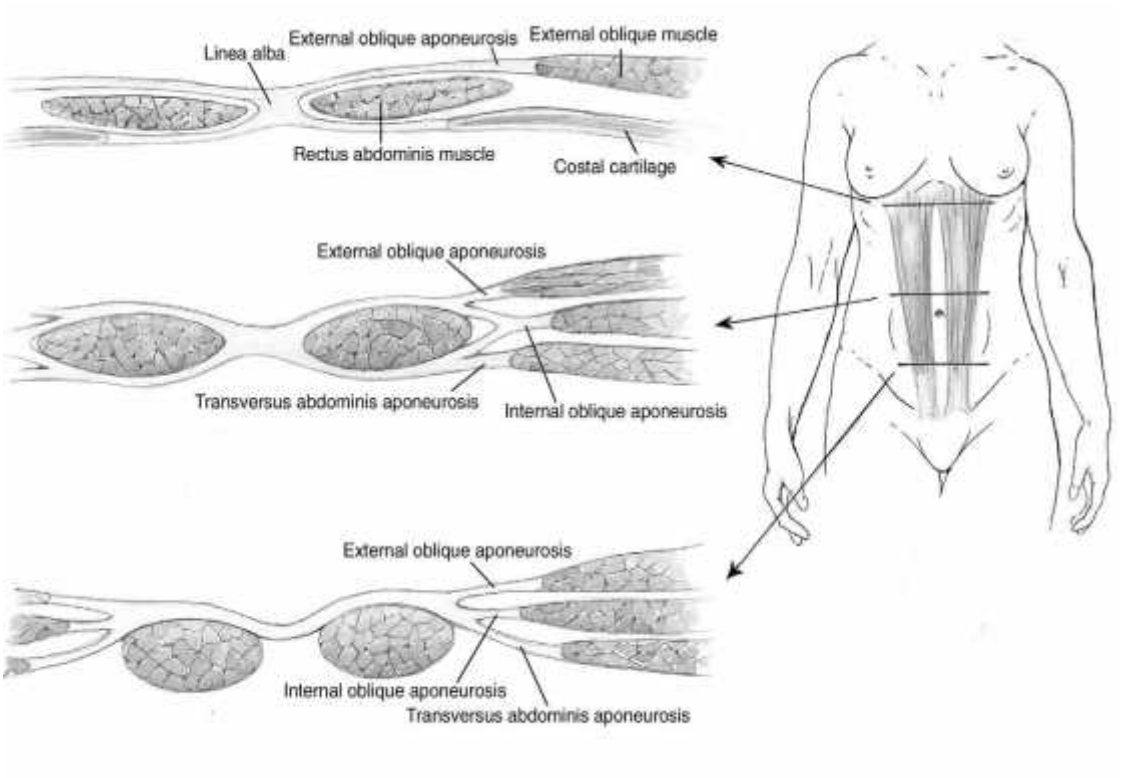


Rectus sheath holds this rectus muscle in place. It is directly shallow to it, formed by the aponeurosis of the three flat muscles. It has a posterior and anterior layer, formed from the aponeurosis of the three flat muscles.

Upper ¾ of abdominal wall: all three muscle layers cover on rectus sheath and pass both anteriorly (external aponeurosis) and posteriorly (transverse aponeurosis). This part of the wall is suturable in surgery.

Lower ¼ of abdominal wall is transversalis fascia. Here all three muscle layers pass anteriorly. Here it is called transversalis fascia. This part of the wall is not suturable in surgery.

Fig.6: Rectus Muscle and Sheath



Nerve supply of Anterior Abdominal Wall

Anterior abdominal wall is supplied by ventral rami of T7-T12 and L1.

Dermatomes: How nerves innervate the anterior abdominal wall- in sections.

Referred pain: For example, in appendicitis pain will go to sympathetic nervous system→ refers to umbilical region (T10). When rupture occurs toxins are irritate the peritoneum, resulting in localized effect.

Iliohypogastric nerve: directly superior to ilioinguinal nerve.

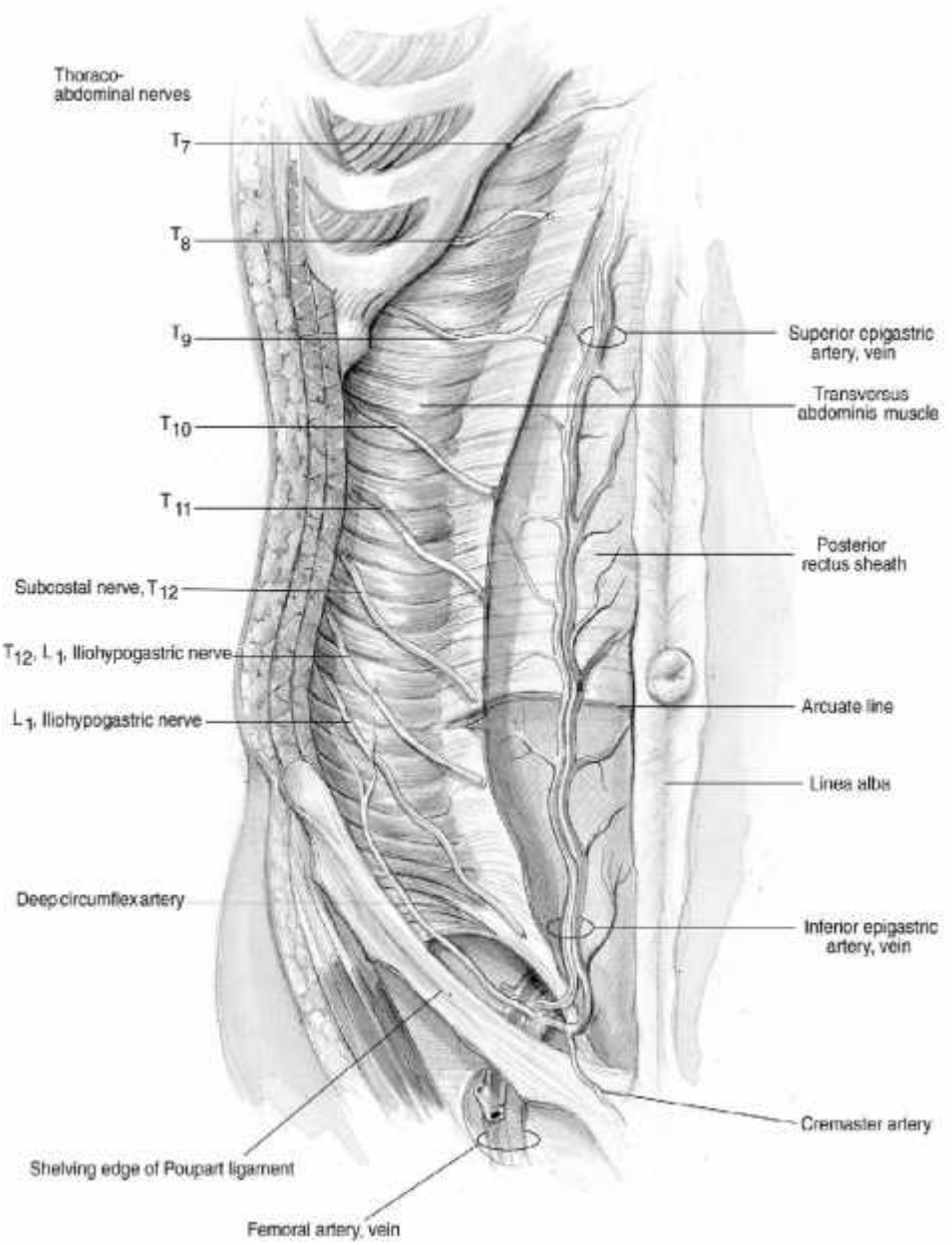
Innervates the suprapubic area

Ilioinguinal nerve: goes through the inguinal canal, with the spermatic cord(male) or round ligament(female).

Suppies scrotum (or labia majora) and medical aspect of thigh.

Both iliohypogastric and ilioinguinal nerve may come off as a single nerve and branch later.

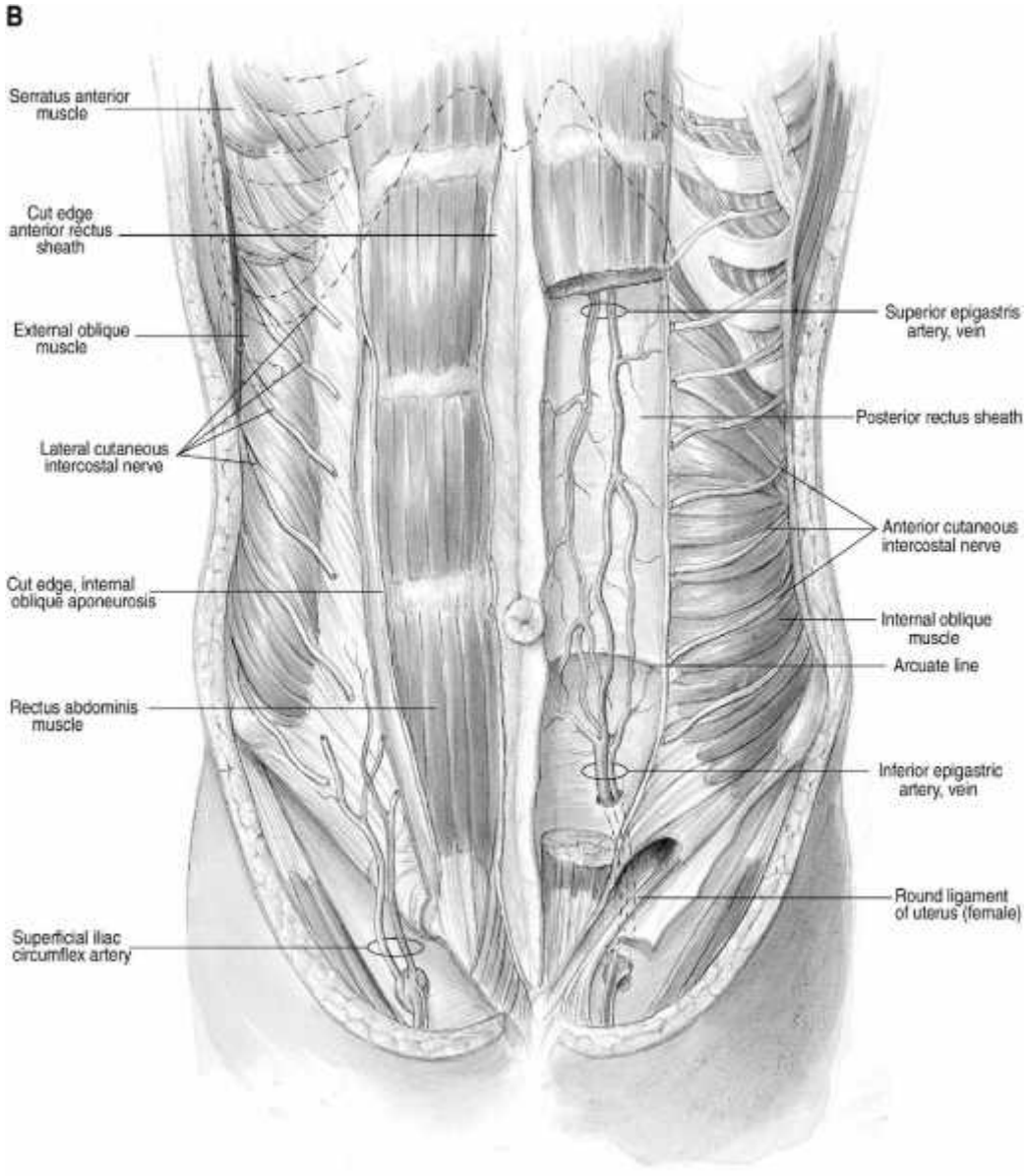
Fig 7; Nerve Supply of Abdominal Wall



Arterial supply of Anterior Abdominal Wall:

The superficial tissues of the lower anterolateral abdominal wall are supplied by three branches of the femoral artery. These branches from lateral to medial are the superficial circumflex iliac artery, the superficial epigastric artery and the superficial external pudendal artery. Branches of these arteries travel toward the umbilicus in the subcutaneous connective tissues. All three arteries have anastomosis with the deep arteries.

Fig 8; Arterial Supply of Abdominal Wall



The deep arteries lie between the tranverse abdominis and the internal oblique muscle. They are the posterior intercoastal arteries 10 and 11, the anterior branch of the subcostal artery, the anterior branches of the four lumbar arteries and the deep circumflex iliac artery.

The rectus sheath is supplied by two arteries. The superior epigastric artery arises from the internal thoracic artery. The inferior epigastric artery arises from the external iliac artery, just above the inguinal ligament. To avoid injury to the major vessels in abdominal operative laparoscopic procedures, laterally situated trocars should be placed at least 8cm from the midlines and at least 5cm above the pubic bone.

Venous supply of anterior wall: the veins follow the arteries.[2, 3 and 9]

Embryology of peritoneum and peritoneal cavity:

The embryogenesis of the peritoneum derives from the mesoderm. The first indication of the intraembryonic coelom occurs during the initial stages of somatic differentiation when a midline cavity forms in the mesoderm. Around the third week, the mesoderm differentiates into lateral plate mesoderm, intermediate mesoderm and paraxial mesoderm. As differtiation continues to the lateral plate divides into somatic (parietal) and splanchnic (visceral) mesoderm. These envelop the intraembryonic coelom on each side of the midline. The splanchnic layer with its underlying endoderm becomes the slpanchnopleure which ultimately contributes to the formation of the viscera by differentiating into the muscle, blood vessels, lymphatics and connective tissues of the alimentary tract. Growth of the somatopleure results in the development of an inverted u-shaped tube that in its early stages communicates freely with the extra embryonic coelom thus allowing the free movement of fluid into the

interior of the embryo at this stage of the development. Later the right and left intraembryoinic coelom will unite to form a single cavity. This single cavity will subdivided again into pleural, pericardial and peritoneal cavities and into the processus(tunica) vaginalis. Differentiation continues by the formation of omenta, mesenteries, ligaments and fossae. As the mesodermal and ectodermal elements of the primordial somatopleure meet in the ventral midline the peritoneal cavity becomes scaled.[2]

Surgical anatomy of peritoneum.

The peritoneum is the largest serous membrane in the body, with a surface area of about 2.4 to 2.7 square metres. It can be divided into parietal and visceral portions. The parietal layer lines the abdominal and pelvic cavities and the abdominal surface of the diaphragm. The visceral layer covers the abdominal and pelvic viscera and includes the mesenteries.[2]

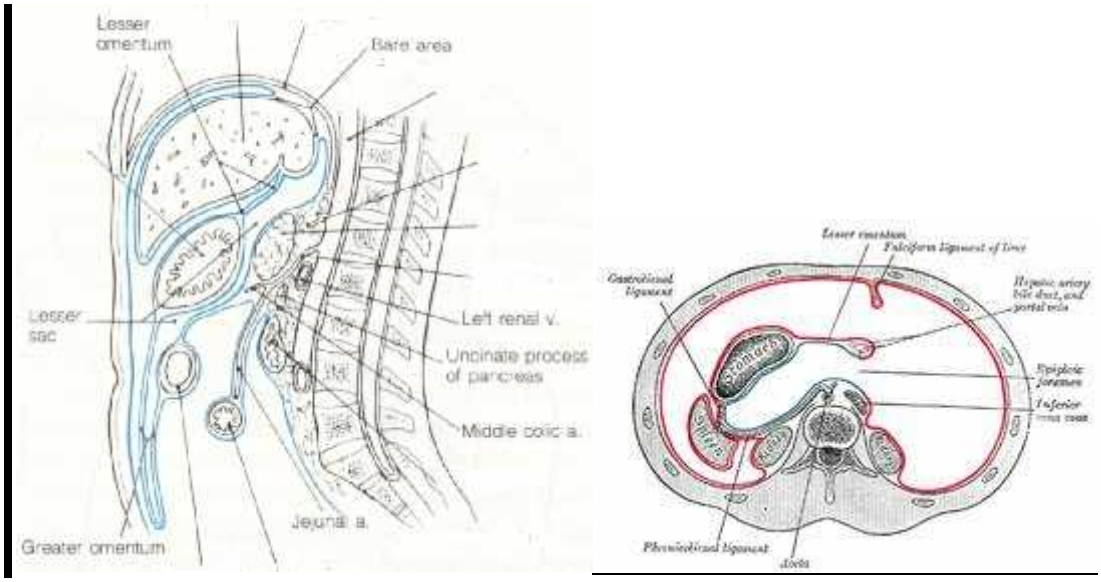
The parietal peritoneum is only loosely connected with the bony wall, separated from it by an adipose layer, the tela subserosa; whereas the visceral peritoneum is usually tightly attached to the organs it covers. The peritoneum consists of a fibrous layer (the tunica subserosa) and a surface layer of mesothelium (the tunica serosa).

The peritoneal cavity is a peritoneal space. It normally contains only thin film of fluid which lubricates the surfaces, allowing frictionless movements of the gastro intestinal tract. Under the effects of certain pathologic conditions, great quantities of fluid can occupy the peritoneal cavity.

Peritoneum does not line the entirety of the abdominopelvic cavity. It is lifted from the body wall, especially posteriorly, by the organs located against the wall during embryologic development. This chain of events causes the formation of a

retroperitoneal space between the peritoneum and the body wall, with organs situated within the space. An organ that is covered only in part by the peritoneum is referred to as retroperitoneal organ. An organ that is covered by peritoneum essentially everywhere except for the site of entrance of vessels is referred to as an intraperitoneal organ.

Fig 9; Peritoneum



Vascular supply of the peritoneum

The blood supply to the abdominal parietal peritoneum is from the branches of arteries of the abdominal wall. The blood supply of the pelvic parietal peritoneum is from the blood vessels of the pelvic wall. Blood to the visceral peritoneum is from branches of the celiac trunk and from branches of the superior and inferior mesenteric arteries or the pelvic visceral blood vessels.

Lymphatics of the peritoneum

The lymphatic of the parietal peritoneum join the lymphatics of the body wall and all drain to parietal lymphnodes. However the lymphatics of the visceral peritoneum join the lymphatics of the related organs and are drained accordingly.

Innervation of the peritoneum

Parietal peritoneum contains somatic afferent nerves. The peritoneum contains many sensory fibres for the sensation of pain; the anterior portion of the parietal peritoneum is especially sensitive.

The visceral peritoneum has no somatic afferent nerves and is relatively insensitive to pain. Sensation which do occur are poorly perceived and not clearly localized by the brain as is characteristic of visceral afferent fibres carried by autonomic nerves to viscera in general. The principal stimulus which can evoke pain from visceral peritoneum is tension upon or stretching of the tissue or ischemia. Perforated viscus produce anterior abdominal wall rigidity and an intraperitoneal fluid collection may produce pain like sensations of traction or tension on the mesentery in the retroperitoneal space but not localized pain.

The innervations of the parietal peritoneum from above downward is as follows, The peritoneum that covers the abdominal surface of diaphragm is innervated at the periphery by the lower six pairs of intercaostal nerves and the subcostal nerves.

Phrenic nerves convey sensory fibres from the peritoneum over the more centrally disposed parts of the diaphragmatic peritoneum. Pathological process of the centrally-located peritoneum over the diaphragm is referred as pain by the phrenic nerves to the distribution of spinal nerve levels C3, C4 and C5 over the shoulder regions.

The lower 6 intercostal and lumbar nerves innervate the parietal peritoneum of the abdominal cavity. The pelvic peritoneum is innervated by the obturator nerve.

The inneravation of the visceral peritoneum is uncertain, but sensory fibres for pain are carried by thoracic and lumbar splanchnic nerves.

Omenta: Peritoneum surrounding the stomach.

Lesser omentum: Peritoneum along the lesser curvature of the stomach covering the pancreas. It is superior and medial to the stomach and posterior to parts of the liver and anterior to pancreas.

Lesser omental bursa: It is the space between the stomach and the liver. It lies anterior to the lesser curvature of the stomach and posterior to the liver.

Epiploic foramen: a pathway that allows entrance from the lesser peritoneum sac to the greater peritoneal sac.

- The inferior vena cava goes directly posterior to it (retroperitoneum).
- The portal triad is directly anterior to it in the peritoneum, along the lesser curvature of the stomach.

Greater omental bursa: the space between the stomach and anterior abdominal wall.

Greater omentum: greater omentum hangs from the greater curvature of the stomach in the form of a thin walled sac, which helps form the omental sac or bursa. In the adult, the greater omentum extends downward to the transverse colon, fusing with it and the transverse mesocolon to a varying degree. Continuing further down as a free fat apron, it covers loops of the small bowel and (occasionally) pelvic organs.

Superior recess: where the lesser omentum stops at the coronary ligament of the liver and reflects back onto the liver. Essentially, it is the space between the stomach and liver.

Inferior recess: along the greater curvature of the stomach. Here the greater omentum reflects onto the transverse mesocolon. Essentially, it is the space between stomach and transverse colon, inferior to the stomach.

Intra-peritoneal organs: organs completely or almost completely enclosed by peritoneum include stomach, liver, gall bladder, transverse colon, jejunum, ileum, caecum.

Retro-peritoneal organs: organs those located mostly or completely behind the posterior parietal peritoneum. These include duodenum, ascending colon (only 25-50% covered), descending colon (only 25-50% covered), sigmoid colon, pancreas, kidneys, great vessels and their primary branches: abdominal aorta and inferior vena cava, celiac trunk and superior inferior mesenteric arteries and veins.

Mesentery:

Two layers of peritoneum opposing each other. Vessels and nerves often lie in the mesentery, where they can easily reach the organ where the peritoneal layers separate and reflect off the organs.

Mesentery: the one that connects the small intestine to the posterior abdominal wall.

The root of the mesentery is where the mesentery connects to the posterior wall and extends from left upper quadrant (L1-L2) to right sacroiliac joint.

Transverse mesocolon: specific mesentery connecting the transverse colon to the posterior abdominal wall.

Sigmoid mesocolon: specific mesentery connecting the sigmoid colon to the posterior abdominal wall.[2, 3]

AGEING AND SURGERY

Over the past 2 decades alone, the percentage of operations in which the patient is older than age 65 increased from 19% of all operations to 37%. When obstetric procedure excluded, this portion rises to 43%. In 2000, the rate of surgery for persons older than age 65 was over two and half times the rate of people aged 45 to 64 years. Discharge data from short say hospitals in 2000 show that 36% of cholecystectomy, 52% of hernia repairs, 55% of coronary artery bypass grafts and 57% of bowel resections were performed on patients older than age 60. It is now estimated that 55% of patients in most general surgical practiced are older than age 60.[2, 3]

This increase in the percentage of operation of operations in which the patient is older than age 60 is not entirely due to the increase in the number of older patients, it is due to reflection of a greater willingness to offer surgical treatment to the geriatric patients. Over the past several decades, advances in surgical and anaesthetic techniques have allowed us to operate with much greater control and safety. Operative mortality in older patients has declined sharply.

The likelihood of receiving surgery for cancers of the breast, ovary, uterus, colon and rectum has increased more rapidly among patients older than age 75 than in those younger than 55.

The pattern of symptoms and the natural history of the surgical disease in older patients may not be identical to that seen in their younger counterparts. The absence of typical signs and symptoms often leads to error in diagnosis and delays in treatment. As a result it is not unusual for an acute complication to be first indication of disease. This is unfortunate because emergency operative mortality is 3 to 10 times higher than in compare to elective cases.

No doubt that increasing age appears to have a negative effect on the outcome of surgery. However most studies indicate that chronological age alone has little effect on outcome.

Physiologic decline:

With ageing there is decline in physiologic function in all organs systems although the magnitude of this decline is variable among organs and among individuals. In the resting state this decline usually has minimal functional consequence, although physiologic reserves may be utilised just to maintain homeostasis. However when physiologic reserves are required to meet the additional challenges of surgery or acute illness, overall performance may deteriorate. This progressive age- related decline in organ system homeostatic reserves, known as "homeostenosis" was first described by the physiologist Walter Cannon in 1940s. With increased age there is an increased utilization of physiologic reserves just to maintain normal homeostasis. Therefore when reserves are stressed there are fewer available to met the challenge and overall function may be pushed over the 'precipice' of organ failure or death.[2, 3]

Over the past several decades an enormous amount of research has been conducted to define the specific changes in organ function that are directly attributable to ageing. It is often difficult to determine whether an observed decline in function is secondary to ageing per se or to disease associated with ageing. Understanding the changes in organ function can minimize these errors.[2, 3]

Cardiovascular:

With increasing age there are morphologic changes are found in myocardium, conducting pathways, valves and vasculature of the heart and great vessels. The number of myocytes declines as the collegen and elastin content increases, resulting in fibrotic areas throughout the myocardium and an overall decline in ventricular compliance. Nearly 90% of the autonomic tissue in the sinus node is replaced by fat and connective tissue and fibrotic interface with conduction in the intermodal tracts and bundle of his. These changes contribute to the high incidence of sick sinus syndrome, atrial arrhythmias and bundle branch block. Sclerosis and calcification of the aortic valve is common but usually of no functional significance.[21, 23]

Progressive dilation of all four valvular annuli is probably responsible for the multivalvular regurgitation demonstrated in healthy older persons. Finally there is a progressive increase in rigidity and decrease in distensibilty of both the coronary arteries and great vessels. Changes in the peripheral vasculature contribute to increased systolic blood pressure, increased resistance to ventricular emptying and compensatory loss of myocytes with ventricular hypertrophy.[3, 29]

The direct functional implication of these changes are difficult to accurately assess because age related changes in body complication, metabolic rate, general state of fitness and underlying disease all influence cardiac performance. It is now generally accepted that systolic function is well preserved with increasing age. Cardiac output and ejection fraction are maintained in spite of the increase in after load imposed by the softening of the outflow tract. The mechanism, by which cardiac output maintained during exercise, however is somewhat different. In younger person output is maintained by increasing heart rate in response to β adrenergic stimulation. With ageing there is relative “hyposympathetic state” in which the heart becomes less

responsive to catecholamines, possible secondary to declining receptor function. The ageing heart therefore maintains cardiac output not by increasing rate but by increasing ventricular filling (preload). Because of the dependence on preload, even minor hypovolemia can result in significant compromise in cardiac function.[21, 27]

Diastolic function however which depends on relaxation rather than contraction is affected by ageing. Diastolic dysfunction is responsible for upto 50% of the cases of heart failure in patients older than age 80 years. Myocardial relaxation is more energy dependent therefore requires more oxygen than does contraction. With ageing there is progressive decrease in the partial pressure of oxygen. As a result even in mild hypoxemia can result in prolonged relaxation, higher diastolic blood pressure and pulmonary congestion. Because early diastolic filling is impaired, maintenance of preload becomes even more reliant on the atrial kick. Loss of the atrial contraction to preload can result in further impairment of cardiac function.[21]

It is important remember that the manifestation of cardiac diseases in the elderly may be nonspecific and atypical. While chest pain is still the most common symptom of myocardial infarction as many as 40% of older patients will present in a non-classic manner with symptoms such as shortness of breath, syncope, acute confusion or stroke.[2, 29]

Respiratory system:

With ageing there is a decline in respiratory function that is attributable to changes in both the chest wall and the lung. Chest wall compliance decreases secondary to changes in structure caused by kyphosis and exaggerated by vertebral collapse. Calcification of the costal cartilage and contractures of the intercostals muscles results in a decline in rib mobility. Maximum inspiratory and expiratory force

decreases by as much as 50% secondary to progressive decrease in the strength of respiratory muscles.[2, 22]

In the lung there is a loss of elasticity which leads to increased alveolar compliance with collapse of small airways and subsequent uneven alveolar ventilation with air trapping. Uneven alveolar ventilation leads to ventilation perfusion mismatches which in turn cause a decline in arterial oxygen tension of 0.3 or o.4mm Hg per year.[28, 2]

The pCO_2 does not change in spite of an increase in dead space. This may be due in part to the decline in the production of CO_2 that accompanies the filling basal metabolic rates. Air trapping is also responsible for an increase in the residual volume or the volume remaining after maximal expiration.

The loss of support of the small airways also leads to collapse forced expiration, which limits dynamic lung volumes and flow rates. Forced vital capacity decreases by 14 to 30ml/yr. The overall effect of loss of elastic inward recoil of lung is balanced somewhat by the decline in chest wall outward force. Total lung capacity therefore remains unchanged and there is only a mild increase in resting lung volume or functional residual capacity. Because total lung capacity remains same, the increase in respiratory volume results in a decrease in vital capacity.[19, 2]

The control of ventilation is also affected by ageing. Ventilatory responses to hypoxia and hypercapnia fall by 50% and 40% respectively. The exact mechanism of this decline has not been well defined but may be the result of declining chemoreceptor function either at the peripheral or central nervous system level.[22]

In addition to these intrinsic changes pulmonary function is affected by alterations in the ability of the respiratory system to protect against environmental

injury and infection. There is progressive decrease in T-cell function, a decline in mucociliary clearance and a decrease in several components of swallowing function. The loss of cough reflex secondary to neurological disorders, combined with swallowing dysfunction may predispose to aspiration. The increased frequency and to an increased and severity of pneumonia in older persons has been attributed to these factors and to an increased incidence of oropharyngeal colonization with gram-negative organisms. This colonization correlates closely with comorbidity and with the ability of older patients to perform activities of daily living. This fact tends to support the idea that functional capacity is crucial factor in assessing the risk of pneumonia.[22]

Renal:

Between the ages of 25 and 85 there is a progressive decrease in the renal cortex in which approximately 40% of the nephrons become sclerotic. The remaining functional units hypertrophy in a compensatory manner. Sclerosis of the glomeruli is accompanied by atrophy of the afferent and efferent arterioles and by a decrease in renal tubular cell number. Renal blood flow also falls by approximately 50%.

Functionally there is a decline in glomerular filtration rate of approximately 45% by age 80years. This decrease is reflected in a decline in creatinine clearance of 0.75ml/min/yr in healthy older men. The serum creatinine value however remains unchanged because there is a concomitant decrease in lean body mass and thus a decrease in creatinine production. Estimates of creatinine clearance in the healthy aged can be made from the serum creatinine by using the formula derived by Cockcroft and Gault.

(140-age in years) x (weight in kg) /[72x(serum creatinine in mg/dl)]

Caution must be exercised when applying this formula to critically ill patients or those on medications that directly affect renal function. [2, 23]

Renal tubular function also declines with advancing age. The ability to conserve sodium and excrete hydrogen ion falls, resulting in a diminished capacity to regulate fluid and acid-base balance. Dehydration becomes a particular problem because losses of sodium and water from nonrenal causes are not compensated for by the usual mechanisms of increased renal sodium retention, increased urinary concentration and increased thirst. The inability to retain sodium is believed to be due to a decline in the activity of the rennin-angiotensin system. The increasing inability to concentrate the urine is related to a decline in end organ responsiveness to antidiuretic hormone. The marked decline in the subjective feeling of thirst is also well documented but not well understood. Alteration of osmoreceptors function in the hypothalamus may be responsible for the failure to recognize thirst inspite of significant elevations in serum osmolality.[23, 26]

Alterations in renal function also have important implications for the type and dosage of drugs used in older patients. Although drugs are handled by the kidney in several different ways, most changes in renal drug processing parallel the decline in glomerular filtration rate. Therefore creatinine clearance can be used to determine the appropriate clearance of most agents processed by the kidney.[3]

The lower urinary tract also changes with increasing age. In the bladder increased collagen leads to limited distensibility and impaired emptying. Over activity of the detrusor secondary to neurological disorders or idiopathic causes has also been identified. In women decreased circulating levels of estrogen and decreased tissue responsiveness to this hormone cause changes in the urethral sphincter that predispose to urinary incontinence. In males prostatic hypertrophy impairs bladder emptying.

Together these factors lead to urinary incontinence in 10% to 15% of elderly persons living in the community and 50% of those in nursing homes.[2, 26]

There is also an increased prevalence of asymptomatic with age which varies from 10-50% depending on gender, level of activity, underlying disorders and place of residence. Urinary tract infections alone are responsible for 30-50% of all cases of bacteremia in older patients. Alterations in the local environment and declining host defences are thought to be responsible. Because of the lack of symptoms in elderly patients with bacteriuria, preoperative urine analysis becomes increasingly important.[2]

Hepatobiliary:

Morphologic changes in the liver with age include a decrease in the number of hepatocytes and overall weight and size. This is however a compensatory increase in cell size and proliferation of bile ducts. Functionally hepatic blood flow falls by 0.3-1.5% per year to 40-45% of earlier values after age 60.[2]

The synthetic capacity of the liver as measured by the standard test of liver function remains unchanged. However the metabolism and sensitivity to certain kinds of drugs is altered. Drugs requiring microsomal oxidation (phase I reactions) before conjugation (phase II reactions) may be metabolised more slowly where as those requiring only conjugation may be cleared at a normal rate. Drugs that act directly on hepatocytes such as warfarin (coumarin) may produce the desired therapeutic effects at lower doses in the elderly owing to an increased sensitivity of cells to these agents.

The lost significant correlate of altered hepatobiliary function in the aged is the increased incidence of gallstones and gall stone- related complications. Gallstone prevalence rises steadily with age although there is variability in the absolute percentage depending on the population. Stones have been demonstrated in as many

as 80% of nursing home residents older than age 90 years. Biliary tract disease is the single most common indication for abdominal surgery in the elderly population.[2, 23]

Immune function:

Immune competence like other physiologic parameters declines with advancing age. This immunosence is characterised by an increased susceptibility to infections, increase in autoantibodies and monoclonal immunoglobulin and an increase in tumorigenesis. Also like other physiologic systems, this decline may not be apparent in the non challenged state. For example there is not decline in neutrophil count with age but the ability of the bone marrow to increase neutrophil production in response to infection may be impaired. Elderly patients with major infections frequently have normal white blood cell counts but the differential count will show a profound shift to the left with a large proportion of immature forms.

With ageing there is an involution of the thymus gland and a decline in the production of thymic polypeptide factors such as thymosin A-1. This and other thymic hormones control the differentiation and proliferation of thymocytes into mature T lymphocytes. The resulting alterations in T-cell populations, products and response to stimuli best describe the changes in immune function that accompany ageing. Although other factors may be involved, the decline in T-cell responsiveness, to a variety of antigens is demonstrated by the high incidence of angry to delayed hypersensitivity skin tests seen in progress older than age 60.[2]

Some B-cell defects have been identified, although it is thought that the functional deficits in antibody production are related to altered T-cell regulation rather than to intrinsic B-cell changes. In-vitro there is an increased helper T-cell activity for nonspecific antibody production and there is a decreased ability of suppressor T-cells

from old mice to recognized and suppress specific antigens from self. This reflected in an increase in the prevalence of autoantibodies to more than 10% by age 80years. The mix of immunoglobulins also changes. IgM levels decrease while IgG and IgA increase slightly.

The clinical implications of these changes are difficult to determine. When superimposed on the known immunosuppression caused by the physical and psychological stress of surgery, insufficient immunologic responses in the elderly should be expected. Increased susceptibility to many infectious agents in the post operative period, however is more likely a result of a stress and comorbid disease rather than physiologic decline.[24, 25]

Nutritional status

Surgeons recognize the value of optimal nutritional status to minimize perioperative mortality and morbidity. However, older patients are at particular risk for malnutrition and therefore at increased risk for adverse perioperative events. It remains imperative for surgeons to continue to assess nutritional status and attempt to correct malnutrition to achieve optimal results. Although this may be difficult in any patient, detection plus correction of malnutrition in older patients is crucial.[1, 2]

The impact of poor nutrition as a risk factor for perioperative mortality and morbidity such as pneumonia and poor wound healing has long been appreciated. Malnutrition is estimated to occur in approximately 0% to 15% of community-dwelling older persons, 35% to 65% of older patients in acute care hospitals, and 25% to 60% of institutionalized older adults. Factors that lead to inadequate intake and uptake of nutrients in this population include the ability to obtain food (e.g., financial constraints, availability of food, limited mobility), desire to eat food (e.g., living

situation, mental status, chronic illness), ability to eat and absorb food (e.g., poor dentition, chronic gastrointestinal disorders such as GERD or diarrhea), and medications that interfere with appetite or nutrient metabolism.

In the frail older adult, a number of factors contribute to neuroendocrine dysregulation of the signals that control appetite and satiety and lead to what is termed *the anorexia of ageing.* Although the anorexia of ageing is a complex interaction of many interrelated events and systems, the result is chronic undernutrition and loss of muscle mass (sarcopenia). Malnutrition has also been associated with increased risk of falls and hospital admission. Measurement of nutritional status in geriatric age group can be difficult as standard anthropometric measurements do not take into account the change in body composition and structure that accompanies ageing.

The Subjective Global Assessment (SGA) is a relatively simple, reproducible tool for assessing nutritional status from history and physical examination. SGA ratings are most strongly influenced by subcutaneous tissue, muscle wasting and weight loss. The Mini Nutrition Assessment which measures 18 factors, including body mass index, weight, cognition, mobility, dietary history, and self assessment among others, is also a reliable method for assessing nutritional status.

Serum albumin can be a strong predictor of nutritional status. There is evidence of prolonged hospital stay associated with low levels of serum albumin.[2, 3]

SPECIFIC CONSIDERATIONS

Peptic Ulcer disease:

The incidence of peptic ulcer disease increases with age. Upto 80% of peptic ulcer related deaths occur in patients older than the age of 65.Other factors that increase the risk of peptic ulcer disease in the elderly population are use of non

steroidal anti-inflammatory agents (NSAIDS) and infection with Helicobacter pylori. NSAIDS are well established inducers of peptic ulcer disease, the mechanism being inhibition of formation of prostaglandins, essential components of the gastric mucosal barrier. Use of NSAIDs increases the risk of developing complicated peptic ulcer disease in the elderly, when compared with younger patients. Actual NSAID use is also a useful prognostic indicator; the mortality rate from peptic ulcer disease in elderly patients who take NSAIDs twice that of those who do not.[40, 3]

Helicobacter pylori infections are believed to occur at a rate of 1% per year, yielding a substantial percentage of the elderly population harbouring infections. Helicobacter pylori screening and treatment has increased significantly in recent years, while counselling about the risks of NSAID therapy has not. Yet in one study, treatment for Helicobacter pylori did not reduce the risk of re-hospitalisation of death within 1 year of initial hospitalization whereas counselling about NSAIDs did both.

Elderly patients typically present for surgical correction of peptic ulcer disease in a delayed fashion and with more advanced disease. This translates to statistically significant increases in operative mortality for elderly patients undergoing surgery for complicated peptic ulcer disease. Using multivariate analysis, Boey and co-workers identified three risk factors for operative mortality in perforated ulcer. They were the presence of concomitant diseases, preoperative shock and more than 48 hours of perforation. Age, amount of peritoneal soiling and length of history of ulcer disease are not significant risks. When zero risk factors were present, the mortality was 0%. Mortality was10%, 46% and 100% with 1, 2 or 3 risk factors respectively.[2, 41]

In a similar study of patients with bleeding duodenal ulcer by the same authors, multivariate logistic regression analysis revealed that age played only a minor role in determining the outcome. The presence of concomitant medical illness, a greater than

5 unit haemorrhage and to a lesser degree, ulcer size negatively affected the outcome. If all three risk factors were present, the mortality rate was 47%.If none was present, the rate was 0.1%.[2]

Biliary Tract Disease:

Biliary tract disease is the single most common cause of acute abdominal complaints and accounts for approximately one-third of all abdominal operations in the elderly. In nearly all populations, the prevalence of gallstones increases with age. There is also an increased incidence of common duct stones with increasing age. Choledocholithiasis is found at the time of Cholecystectomy in as many as 30% of patients in the seventh decade of life and upto 50% of those in the eighth decade.[2, 7]

The increased development of gallstones in elderly is thought to result from both changes in the composition of bile and form impaired biliary motility. Alterations in the composition of bile with advancing age include the increase in the activity of HMG-CoA(The rate limiting enzyme in the synthesis of cholesterol)and a decrease in the activity of hydroxylase(the rate limiting enzyme in the synthesis of bile salts from cholesterol) during supersaturation of the bile with cholesterol and a decrease in the primary bile salt pool. The ratio of secondary to primary bile salts promotes cholesterol gallstone formation by enhancing cholesterol synthesis, increasing protein content of the bile, decreasing secretion time and increasing the production of specific phospholipids that are thought to affect the production of mucin. It has also been suggested that the increase in the secondary bile salts in the aged may promote a recycling of bilirubin,which in turn leads to the unconjugated bilirubin supersaturation necessary for pigment stone formation.[2, 12]

Alterations in gallbladder motility and bile duct motility are thought to be central to the development of cholesterol stones and brown pigment stones respectively. Biliary motility is a complex interaction of hormonal and neural factors. However, the major stimulus of gallbladder emptying is cholecystokinin. The sensitivity of the gallbladder wall to CCK has been shown to decrease with increasing age in animal models. Exogenous administration of CCK to animals fed a lithogenic diet inhibits the age dependent development of cholesterol gall stones. In humans, gall bladder sensitivity to CCK is also decreased.[2, 44, and 45]

Regardless of the pathogenesis, gall stones are associated with complications in 40% to 60% of older patients requiring treatment of the disease, than 20% of younger patients. The increased rate of complicated disease seen in this age group may be directly attributable to the increased severity of the disease or to increased prevalence of comorbid illness. It is more likely, however, to be due to a combination of factors including delays in diagnosis and treatment caused by the frequent absence of typical biliary tract symptoms, biliary colic or episodic right upper quadrant pain radiating to the back. Even in the presence of acute cholecystitis, one-fourth of older patients may have no abdominal tenderness, one-third no elevations in temperature or white blood cells, and as many as one-half, no peritoneal signs in the right upper quadrant.[45]

Small bowel obstruction:

Small bowel obstruction (SBO) is by far most common and surgically relevant disorder of small intestinal function encountered in the aged. Although the exact incidence of small bowel obstruction in the elderly is difficult to ascertain, lysis of adhesions is the third most common gastrointestinal procedure after cholecystectomy

and partial excision of large bowel. 50% of deaths associated with SBO occur in patients older than age 70.[2, 3]

SBO can result from lesions or objects extrinsic to bowel wall, intrinsic to bowel or within bowel lumen. Studies more than 25years ago show that just over a half of the Small Bowel obstruction in older patients was caused by incarcerated abdominal wall hernias. Post operative adhesions are now responsible for over 50% of SBO and hernias for another 15% to 20%.

In addition, certain kinds of hernias, such as those that occur through the obturator foramen, are found almost exclusively in elderly and are particularly more difficult to diagnose.

Luminal obstructions, from other than deliberately swallowed foreign bodies, while uncommon, occur most often in the geriatric. Phytobezors or large concretions of poorly digested fruit and vegetable matter form with increased frequency in stomach of geriatric patients with poor dentations, decreased gastric acid and impaired gastric motility.

Obstruction of small bowel by aberrantly located gall stones is another uncommon cause of obstruction seen primarily in geriatric age. Although it only accounts for 1% to 3% of all SBO it has been implicated in as many as 25% of obstructions in patients older than age 60 years with no hernia of history or prior surgery.

It is important to note however that the three important management issues distinguishing functional (ileus) from mechanical obstruction, distinguishing simple from strangulated obstruction and determining the optimal timing of operation for partial obstruction are even further exaggerated in geriatric patients.

Many of the factors associated with ileus (Systemic infections, intraabdominal functions, metabolic abnormalities and medications) are common in older patients abdominal distension is not always appreciated. Signs and symptoms of underlying infections such as pneumonia, urinary tract infection or appendicitis may be subtle. Bowel distension may be erroneously considered the primary problem rather than a secondary event. A variety of non obstructive causes can rapidly lead to dehydration and subsequent electrolyte abnormality in geriatric population.

The accurate distinction between strangulated and simple mechanical small bowel obstruction is difficult to make in all age group but even more so in this age group. Clinical findings of fever, tachycardia, elevated white blood cell count, and focal tenderness are notoriously particularly in geriatric in whom the risk of strangulation is the highest. In only 50% patients of all ages with strangulations in white blood cells elevated.

In patients suspected of having a partial adhesive SBO, in geriatric, several additional consideration are important. Although the natural reflex is to avoid unnecessary operations in sick older patients, prolonged nasogastric intubation is associated with an increased incidence of aspiration and pneumonia. Even short period of nutritional deprivation may present a significant risk to the geriatric patients with a baseline nutritional deficit.

In a review of patients treated for SBO, recurrence occurred in 34% in 4 years and 42% at 10 years. In the operated group, majority of recurrences occurred with malignant group rather than adhesive group.[2, 7and 46]

Appendicitis

Although appendicitis typically occurs in the second and third decades of life, 5% to 10% of cases present in old age. Appendicitis in the elderly has increased in recent decades while the incidence in younger patients is declining. Inflammation of the appendix now accounts for 2.5% to 5% of acute abdominal disease in patients older than age 60to 70.The overall mortality from appendicitis is only 0.8%, but the vast majority of deaths occur in the very young and the very old. In patients younger than 65, the mortality is less than 0.5%, whereas for those older than 65, the overall mortality rate is nearly 5%.

The classic presentation of appendicitis-periumbilical pain that localizes over a period of several hours to the right lower quadrant-is present only half as often in older patients as in younger patients. In one study, only 55% of patients older than age 80 had right lower quadrant pain and 18% had no abdominal pain at all. In addition, because vague abdominal pain is a common complaint in older persons, its significance is frequently overlooked.

Other signs of acute appendicitis are also unreliable in the elderly. White blood cell counts are less than 10,000 /mm in 20% to 50% of older patients with simple appendicitis. Similarly, temperature is less than 37.6 in as many less frequently in older patients.[3]

The indolent nature of the initial symptoms of appendicitis in the elderly usually leads to delays of 48 to 72 hours before medical attention is sought. In only 30% to 70% of cases is the correct diagnosis made on admission. Delays in operating for greater than 24 hours are three times as likely to occur in older than in younger patients.

Perforated appendicitis is far more common in the elderly. Rates of perforation increase directly with age; by age 70 years, 70% to 90% of patients will present with perforation.[2, 16]

The long duration of symptoms, errors in diagnosis and long delays until operation are most likely responsible. Mortality rates for perforated appendicitis also increase with age from 0 to 70% and 32% to those older than age 70.

If there is a suspicion for and periappendiceal abscess, CT should be obtained before operation. Percutanoeous drainage and intravenous antibiotics are often preferable for exploration in the presence of large abscess. In younger patients, this approach is followed by interval appendicectomy approximately 6 weeks after the abscess is resolved. In elderly, recurrent appendicitis after resolution of the abscess is uncommon and interval appendicectomy, is therfore, not necessary in all cases. However, the possibility of perforated cancer in this age group does mandate a thorough evaluation of the colon when the acute process is controlled.[2, 47]

Hernia:

The estimated incidence of abdominal wall hernia in persons older than the age of 60 is 13 per 1000, with a fourfold to eight fold increase in incidence in men. Fifty percent of all hernias are indirect inguinal, 20% are direct inguinal, 10% are ventral, 6% are femoral, 3% are umbilical and 1% are esophageal hiatal. Whereas 85% of all groin hernias occur in males, 84% of femoral hernias occur in females. The geriatric population is also at risk for the more occult type of hernias that do not become apparent until a complication has occurred.

Repair of groin hernia can be performed as an outpatient procedure using either epidural anaesthesia or local anaesthesia with intravenous sedation. Mortality

rates are low even in patients with concomitant medical disease, with many reports showing rates of 0%.

Morbidity and mortality rates for emergency hernia repair are over 50%, 8% to 14% respectively. This is largely because of the high incidence of bowel incarceration at presentation. Intestinal resections are required in upto 30% of cases.[3, 46]

PREOPERATIVE EVALUATION

Primary care physicians often find themselves in the role of preoperative medical consultant. In this role, as to what is indicated based on the patient's risk factors, history and physical examination, type of surgery and the overall health of the patient.

A recently updated set of guidelines by American College of Cardiology/American Heart Association (ACC/AHA) for stratifying patients undergoing non-cardiac surgery and to help assess the need for stress testing can be found on the internet.[48, 50]

I. COAGULATION TESTS

In a study over 4 months, 49 out of 1872 patients required more blood transfusion than anticipated.

Good bleeding history must be taken, including questions related to spontaneous bruising, excessive bleeding after minor trauma, dental extractions or prior surgery or with menses.

In the setting of a negative bleeding history and exam, PT/PTT testing would be indicated then for conditions that have the potential to cause bleeding such as in patients with severe liver disease, those with malnutrition (who may be vitamin K deficient) and those on anticoagulants.

II. COMPLETE BLOOD CELL COUNT

In multiple studies, baseline haemoglobin and estimated perioperative blood loss have been shown to predict the need for transfusion in patients who are undergoing surgery expected to have at least moderate blood loss(greater than 500 ml)Procedures with potential blood loss of less than 500 ml include arthroscopy ,laparoscopic cholecystectomy and inguinal hernia repair. Surgeries with greater than 500 ml potential blood loss include hysterectomy, joint replacement, major gastrointestinal or genitourinary surgery and major vascular or cardiothoracic procedures as well as intracranial procedures. Preoperative blood loss is indicated in surgeries where blood loss is more than 500 ml, history of bleeding, clinically significant anaemia, and history of renal failure, chemotherapy or radiotherapy in the past 6 months. Other conditions that may warrant complete blood count include those with chronic severe illness, such as those in ASA class 3 or 4, although such patients will generally be eligible for ambulatory surgery only if going for minor surgery.

III. ELECTROLYTES

The chief reason to obtain electrolytes is to determine if patients have abnormalities that may increase the risk of arrhythmias or complications in the setting of volume shifts, or to identify renal problems that may increase the risk of acute renal failure postoperatively. Preoperative creatinine greater than 2 posed greater risk for postoperative complications, thus advocating that preoperative testing of renal function be done for patients with a substantial likelihood of renal insufficiency and in those undergoing major surgery.

Besides renal failure, conditions that increase the probability of electrolyte or glucose abnormalities that may affect management include those on medication s such as diuretics, digoxin or steroids or patients with diabetes.

IV. URINE ANALYSIS

There are two instances in which routine urinalysis is felt to be indicated; in those with symptoms of UTI and in those undergoing an implant device or prosthetic joint or other orthopaedic hardware.

V. ELECTROCARDIOGRAM

The main reason to obtain preoperative ECG in an asymptomatic patient is to help decide if further cardiac testing is needed to help assess risk of perioperative cardiac complications. In terms of stratifying cardiac risk in general for major, two guidelines stand out. The Modified Lee Index, based on a prospective validation, came up with 6 factors with prognostic value for major cardiac factors. One of these factors is ischemic heart disease. Thus, an ECG which shows evidence of IHD, if not already known or suspected from the history, could alter management. The other guidelines(ACC/AHA updated in 2002) includes evidence of a prior MI by ECG or by history as a factor which may place a patient in a risk group that warrants preoperative cardiac stress testing. Thus the finding of ECG changes consistent with a silent MI not otherwise suspected could change management of a recognized MI. Preoperative ECG is indicated in males over the age of 50 and females over 60. In patients under these ages, ECG is indicated in those with a history of a treated arrhythmia, coronary artery disease or congestive heart failure, severe peripheral vascular disease, morbid obesity, long-standing or poorly controlled hypertension or

diabetes, smoking over 20 pack years and family history of premature MI (BEFORE AGE 60) All of these are felt to be conditions that increase the pre-test probability of an abnormal ECG.

If an abnormal ECG is found on preoperative evaluation, it should be compared with prior ECG when possible. If the ECG is not changed, but suggests ischemia or MI, then one should document the results of a formal evaluation for coronary artery disease performed in the past two years. If that evaluation either demonstrated myocardium at risk or was not performed, then the patient would require preoperative cardiology evaluation. If the ECG showed new abnormalities, which were suggestive of ischemia or MI, the patient also would require cardiology consultation.[27, 28]

VI. CHEST X-RAY

The frequency of abnormal chest radiograph increases with age. One study showed that 0.3% of patients younger than 60 years has unsuspected abnormal CXR results or clinical findings suggestive of underlying cardiac or pulmonary disease compared to 22% of patients older than 60 years. [30, 31]

LIVER DISEASE:

If evidence of impaired liver function is noted on routine liver function tests or by history during preoperative evaluation, poor surgical outcome is likely. The only consequences of liver disease that can be corrected preoperatively are coagulation abnormalities which can be managed with Vitamin K or blood products (fresh frozen plasma, coagulation concentrates).[55]

RENAL DISEASE:

Renal function is assessed by measuring blood urea nitrogen and serum creatinine levels. In the elderly, serum creatinine level must be adjusted for age and decreased lean body mass using one of several formulas. Dosages of renal excreted drugs must be adjusted based on renal clearance. Dehydration may lead to prerenal azotemia, which can be corrected by administering fluids. If blood urea nitrogen and serum creatinine levels remain high, peritoneal dialysis or haemodialysis may reverse the uremia and reduce the high surgical risk.[2]

CREATININE:

The prevalence of elevated creatinine levels in asymptomatic patients ranges from 0.2-2.4% and increases with age. Approximately 9.8% of patients aged 46-60 years have elevated creatinine levels. Patients with mild-to-moderate renal insufficiency are usually asymptomatic but have an increased risk of perioperative morbidity and mortality. Accordingly, testing renal function with serum creatinine level is recommended for all patients older than 40 years, especially if hypotension or use of nephrotoxic medications is anticipated.

BLOOD SUGAR:

The frequency of abnormal glucose laboratory results in asymptomatic patients ranges from 1.8-5.5%.The frequency increase with age, so that nearly 25% of patients older than 60 years have a fasting blood sugar level above 120mg/dl.

Only in certain operations, such as vascular surgery and coronary artery bypass grafting was diabetes associated with higher perioperative risks; hence, routine blood sugar determination is not recommended unless the patient has high risk of diabetes

(e.g. Obesity, steroids, strong family history) or will be undergoing vascular or bypass surgery.

POSTOPERATIVE CLINICAL ASSESSMENT AND MONITORING

- Any anaesthetic, surgical or intra-operative complications
- Any specific postoperative instruction concerning possible problems
- Any specific treatment or prophylaxis required (e.g. Fluids, nutrition, antibiotics, analgesia, antiemetics, thromboprophylaxis)
- Intraoperative history and postoperative instructions
- Circulatory volume status
- Respiratory status
- Mental status

Patients with the following risk factors for deterioration should be assessed within two hours of the first postoperative assessment:

- American Society of Anaesthesiologists (ASA) grade more than or equal to 3
- Emergency or high risk surgery
- Operation out of hours
- Drug treatment of pre-existing cardiovascular and respiratory disorders
- Treatment of postoperative nausea and vomiting

Patients requiring the frequent monitoring of multiple variables should be considered for care at level 2 or above.

Any patient with circulatory disturbance should be catheterised and the urine output measured hourly.

Consider catheterisation in patients with no urine production after few hours.

DAILY CLINICAL ASSESSMENT

Postoperative monitoring should be continued on a daily basis.

The monitoring regimen should be reviewed daily so as best to provide data for clinical decision making.

Any change in a monitoring regimen should prompt reassessment of the level of care.

CARDIOVASCULAR MANAGEMENT

Postoperative blood pressure should always be reviewed with reference to the preoperative and intra-operative assessments.

Further more careful assessment is required in patients with

- Heart rate below 50 beats per minute
- Heart rate above 100 beats per minute
- Blood pressure below 100 mm Hg systolic
- If patients are hypertensive, ensure that they are receiving adequate anti hypertensive medications. If hypertension persists, seek specialist medical advice and review the level of care.
- Patients on regular antihypertensive medication should normally be maintained on this medication perioperatively. If the patient becomes hypotensive, then it may be appropriate to discontinue some drugs.
- Beta blockers and intravenous (IV) nitrates may be used safely and effectively in postoperative hypertension.[55]

Medical treatment to reduce Perioperative cardiac risk:

- Beta blockers should be continued perioperatively in patients previously taking these drugs for coronary disease, congestive heart failure, hypertension or arrhythmias.
- The occurrence of supraventricular arrhythmias should provoke a search for underlying causes such as hypoxia, hypovolemia, electrolyte abnormality, and sepsis or drug toxicity.
- Direct current (DC) shock should be considered as a first treatment option where there is haemodynamic deterioration as a result of a tachyarrhythmia.
- ECG at baseline, immediately following surgery, and daily for the two subsequent days has to be monitored.
- Cardiac troponin measurements should be done 24 hours after surgery.
- In patients without documented coronary disease, surveillance for perioperative MI should be restricted to those who develop cardiac symptoms or signs.
- Thrombolysis is not indicated in the management of perioperative MI, but all other aspects are as for MI in any other setting.[27, 28]

HYPOTHERMIA

- Maintain normothermia in the postoperative period.
- Active warming is appropriate for patients who are hypothermic postoperatively.

OXYGENATION

Patients with coronary artery disease or major risk factors for coronary artery disease should receive oxygen continuously until mobile.[2]

RESPIRATORY MANAGEMENT

Reducing Postoperative Pulmonary Complications

Oxygen therapy should be used in those patients at high risk of postoperative complications, or who are hypoxaemic following surgery (oxygen saturation measured by a pulse oximeter[SpO2] less than 92%

Monitoring and Diagnosis

Respiratory rate, pulse rate and conscious level should be monitored routinely to identify postoperative respiratory complications.

The following indicate the possible development of respiratory complications:

- Respiratory rate less than 10 or more than 25 breaths per minute
- Pulse rate more than 100 beats per minute
- Reduced conscious level

Patients in whom there is a suspicion of postoperative pulmonary complications should have an arterial blood gas analysis, a sputum culture and ECG. Chest X-Ray should be performed on suspicion of major collapse, effusions, pneumothorax or haemothorax.

Other investigations should be used only if there are specific indications.[31, 41]

TREATMENT

Oxygen should be given to patients with hypoxaemia using a device that is best tolerated to achieve the necessary SpO2.

In a study conducted in august 2011, at France regarding the risks for mortality in major digestive surgeries in the elderly of 3322 patients aged >65 years from 2002-2011, concluded that characterisation of independent validated risk indicators in elderly patients is essential for improving surgical outcomes[8].

In a study conducted at Guwahati, India, in 2006 about clinical and laboratory evaluation of gastrointestinal (luminal) diseases in the elderly, they concluded that gastrointestinal disorders are an important cause of morbidity and hospital admission in the elderly. Malignancy is the commonest cause of gastrointestinal morbidity in elderly patients[6].

In a study done in 2011, at Côte D'ivoire about Non traumatic abdominal surgical emergencies in elderly patients, they concluded that most non traumatic abdominal surgical emergencies in elderly patients were related to complications of neglected or undiagnosed pre-existing disease. Prognosis was related to the stage of the disorder, initial surgical management, and deterioration of the coexisting medical problems[4].

A study done at U.S.A in 1981 studied mortality and gastrointestinal surgery in the aged. They concluded while elective gi surgery in the elderly has a significant risk, death is almost always the result of an associated disease (pulmonary, renal, or cardiac). Emergency procedures in the elderly indeed carry greater risk, statistically the same as in the 50- to 69-year-old group. Death is frequently related to an acute process complicating a treatable disease[7].

MATERIALS AND METHODS

SOURCE OF DATA:

- All the patients aged more than 60 years coming to surgical department BLDEU'S hospital with acute abdominal conditions.
- Study period; October 2011 to July 2013.

METHOD OF COLLECTION OF DATA:

- Patients aged more than 60 years old admitted with abdominal emergency conditions in department of surgery in B.L.D.E.U.'S Hospital, from October 2011 to July 2013.
- History noted, with emphasis on past history of surgeries, chronic medical illnesses.
- Thorough clinical examination was done.
- Their management, course of treatment, effect of co-morbid conditions on the course, events in operative and post operative period studied.

INCLUSION CRITERIA:

- All patients aged more than 60 years old admitted with abdominal emergency conditions in department of surgery in B.L.D.E.U.'s Shri B M Patil Medical College and Research Centre Hospital, from October 2011 to July 2013.
- Geriatric patients coming with blunt trauma of abdomen also included.

EXCLUSION CRITERIA:

- Immunocompromised patients.

RESEARCH HYPOTHESIS:

- Finding out the common causes for abdominal surgical emergencies in elderly and identification of effect of co-morbid factors can help us to be better prepared and to provide better care to elderly patients.

INVESTIGATIONS / INTERVENTIONS:

Investigations or interventions in this study are according to routine standardized protocol.

There are no animal experiments involved in this study.

These routine investigations were done and repeated as per patient requirements:

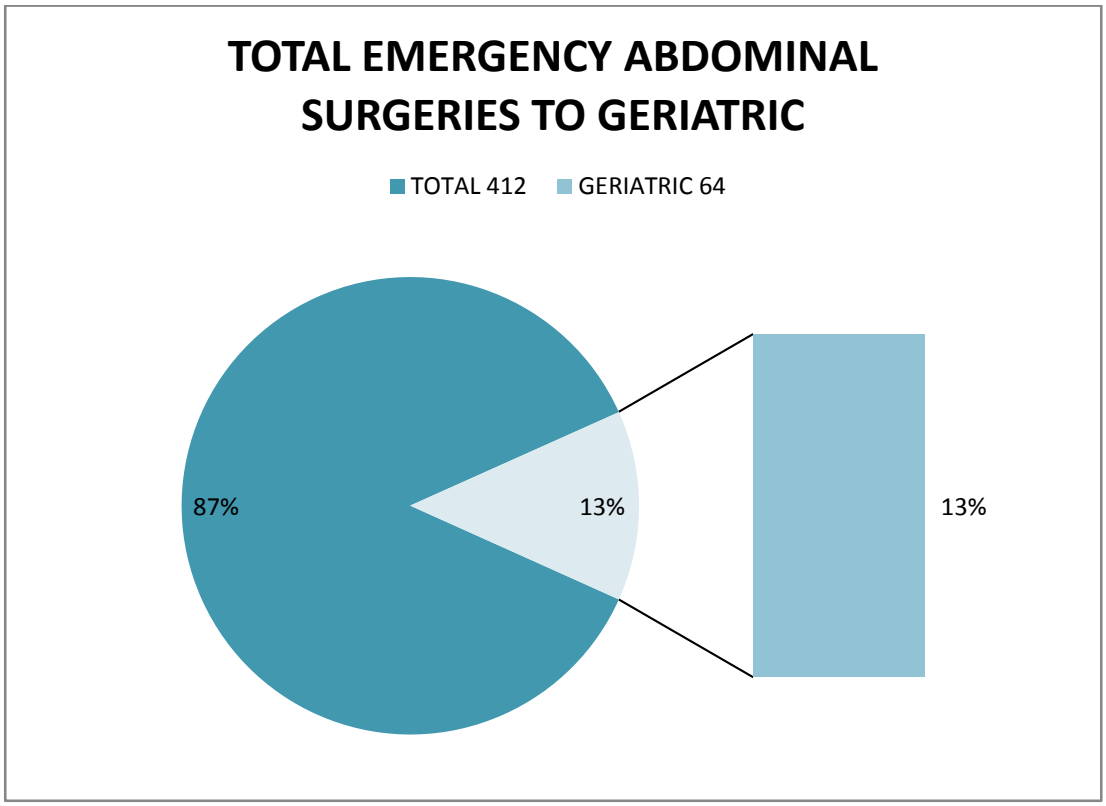
1. Complete blood count, BT, CT.
2. Blood group, Rh typing.
3. Urine – sugar, albumin and microscopy.
4. Random blood sugar, serum creatinine, serum albumin, blood urea, prothrombin time.
5. Electrocardiogram and chest x-ray.
6. 2 d Echocardiogram, as and when required.
7. X-ray erect abdomen.
8. Ultrasonography of abdomen and pelvis.
9. Tests of detect infection with human immunodeficiency virus and hepatitis B virus (in accordance to universal safety precautions).

RESULTS AND ANALYSIS

64 patients aged 6o years or more who underwent emergency abdominal surgery were studied. Their preoperative status and post operative outcomes were studied.

The total abdominal emergency surgeries conducted under all units of surgery department were 412, out of which 64 were in geriatric age group (13%).

Graph 1: Percentage of geriatric patients undergoing emergency abdominal surgeries



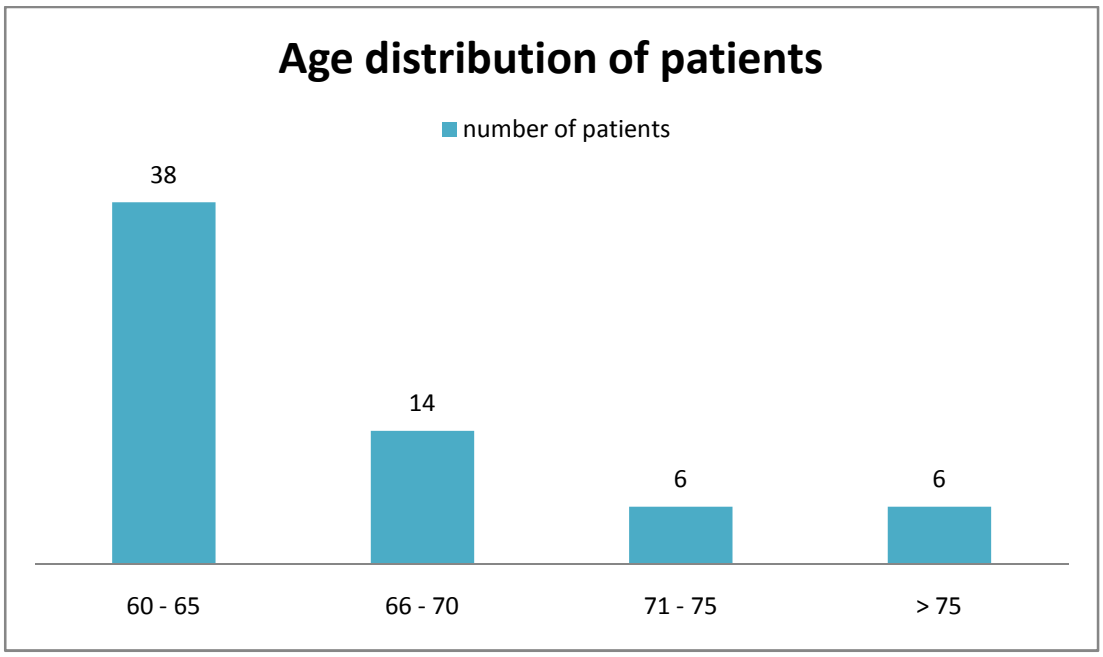
Age and sex incidence

Table 2; Age Distribution

Highest numbers of patients were in the age group of 60 to 65 years. 2 patients were aged 80 years. The distribution was as follows.

AGE	PATEINTS
60 – 65 years	38 (59.38%)
66 – 70 years	14 (21.88%)
71 – 75 years	6 (9.38%)
>75 years	6 (9.38%)

Graph 2: Age distribution of patients



- Mean age of the patients studied is 67 years; Range is 60-80 years.
- 46(71.88%) were male patients and 18(28.12%) were female patients. Male to female ratio is 1:2.5

Table 3: Incidence of various abdominal emergencies in geriatric age group

Etiology	*Cases*	*Percentage*
Perforated peptic ulcer	24	37.5
Obstruction due to adhesions	11	17.19
Ileal perforation	5	7.8
Cholecystitis	5	7.8
Appendicitis	8	12.5
Bowel gangrene	4	6.25
Trauma	2	3.125
Hernia	3	4.68
Malignant obstruction	1	1.56
Gastric volvulus	1	1.56
Ruptured liver abscess	1	1.56

Graph 3: Sex incidence of emergency abdominal surgeries

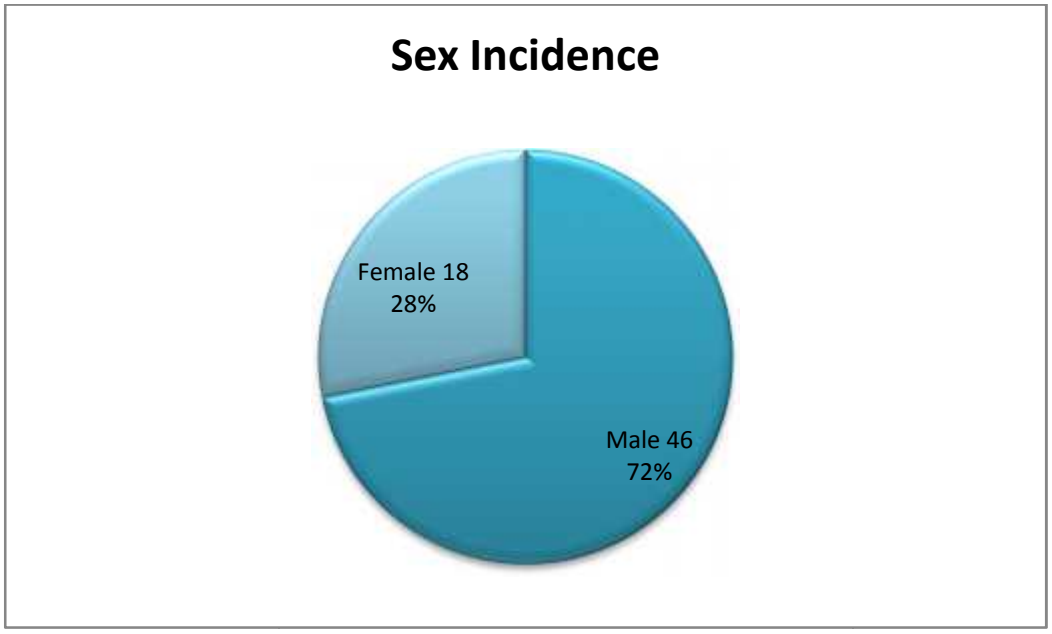


Table 4: Incidence of co-morbidities

Diabetes Mellitus	17	26.56%
Chronic Obstructive Lung Disease	17	26.56%
Hypertension	11	17.19%

Graph 4: **Incidence of various abdominal emergencies in geriatric age group**

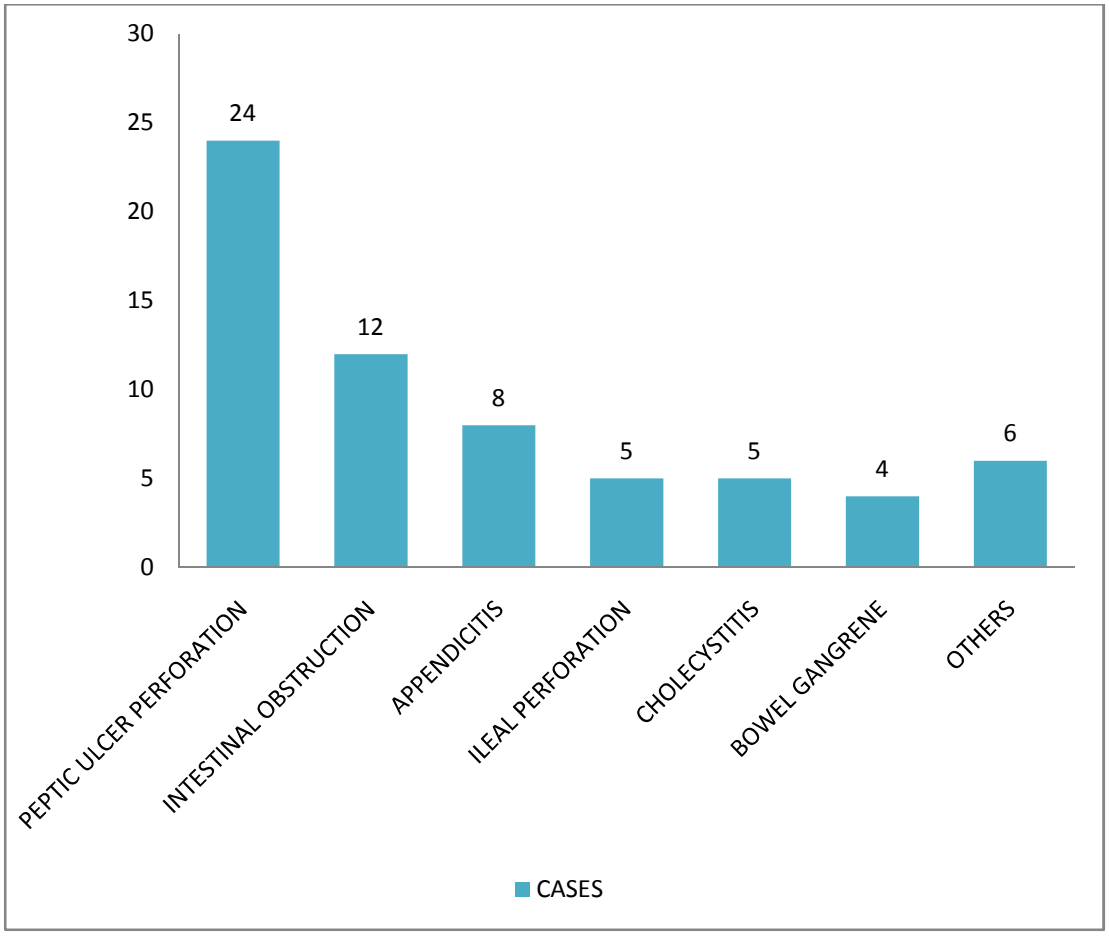


Table 5: General Physical Examination

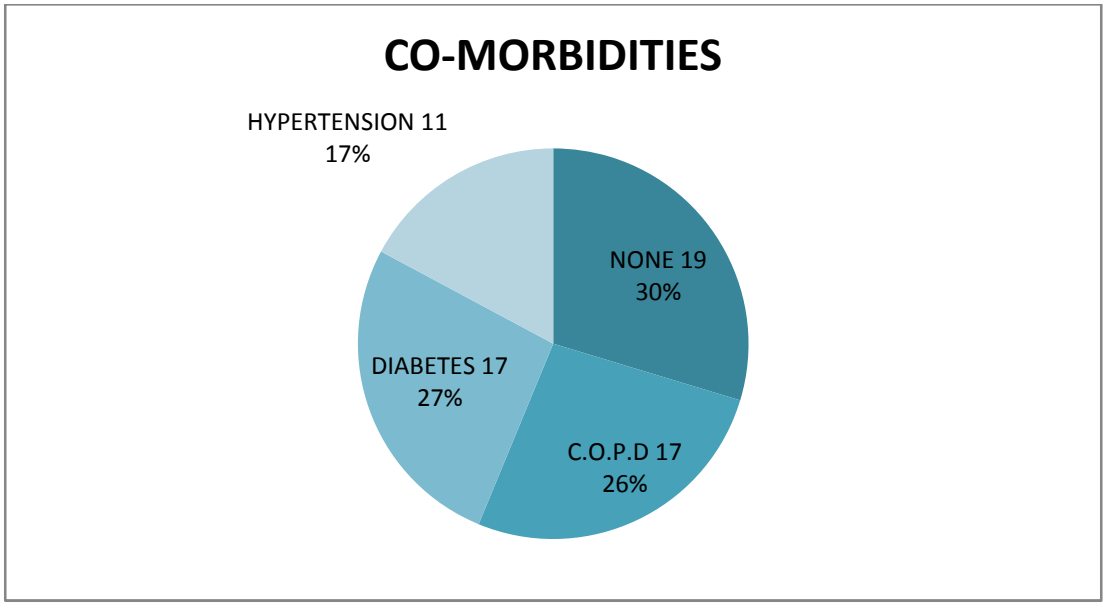
Pallor	29 (45.3%)
Icterus	1 (1.56%)
Pedal oedema	9 (14.1%)
Dehydration	14 (21.87%)

Pallor was the most common finding 45.3% and dehydration 21.8%.

Pulse Rate, Blood Pressure:

19 patients had tachycardia. It was associated with hypotension in 10 patients. Pulse and blood pressure was not recordable in 2 patients.

Graph 5: **Incidence of co-morbidities**



- Past history of abdominal surgeries was present in 11 patients (17.19%).
- History of blunt trauma abdomen was present in 2 patients (3.12%)

Haemoglobin:

Table 6; Haemoglobin values

<6 g%	0
6-8 g%	5 (7.81%)
8-10 g%	9 (14.1%)
>10 g%	50 (78.1%)

WBC Counts

24 patients (37.5%) had leucocytosis (WBC counts > 11000 cells/cumm)

4 patients (6.25%) had leucopenia (WBC counts < 4000 cells/cumm)

Biochemical investigations:

23 patients (35.93%) had high Blood Urea levels (> 40 mg/dl).

17 patients (26.56%) had high Serum Creatinine levels (> 1.4 mg/dl).

26 patients (40.63%) had low Serum Albumin levels (< 2.8g/dl).

Chest X-ray findings

26 patients (40.63%) showed changes attributed to Chronic Obstructive Lung Diseases.

8 patients (12.5%) had Pleural Effusion.

2 patients (3.125%) had Fibrosis in the upper lobes of lungs.

6 patients (10%) showed Left Ventricular Hypertrophy.

Table 7; ECG recordings

ST segment elevation	4 (6.25%)
T Wave inversion	7 (10.9%)
Right Axis Deviation	9 (14.1%)
Left Axis Deviation	5 (7.81%)
Bundle branch block	4 (6.25%)

Patients with T wave inversion and ST segment elevation underwent biochemical tests for cardiac enzymes (CPK-MB and Troponin T) and were diagnosed with Acute Myocardial Infarction in 7(10.93%).

SURGICAL AND POST OPERATIVE OUTCOMES

Duration of Surgical Procedure

Mean duration of surgical procedure – 97 minutes

Range – 30 minutes to 200 minutes

Mean duration of surgery for duodenal ulcer perforation repair was 89 minutes.

Mean duration of surgery for intestinal obstruction due to adhesions was 59 minutes.

In 2 patients, additional surgical procedures were carried out in the same sitting. Suprapubic cystostomy for acute retention of urine in a patient with Duodenal Ulcer Perforation with Benign Prostatic Hyperplasia was done along with Graham's omental patch repair. Another patient underwent repair of ruptured urinary bladder during splenectomy for grade 3 splenic injury due to blunt abdominal trauma.

One patient had cardiac arrest and death on table, during surgery for acute intestinal obstruction.

One patient underwent placement of intra peritoneal drainage tube under local anesthesia as her general condition was poor and could not withstand general anesthesia.

POST OPERATIVE MANAGEMENT

All patients were treated according to standard protocols of intravenous fluids according to individual patient's needs, appropriate prophylactic and specific antibiotics, proton pump inhibitors and symptomatic therapy.

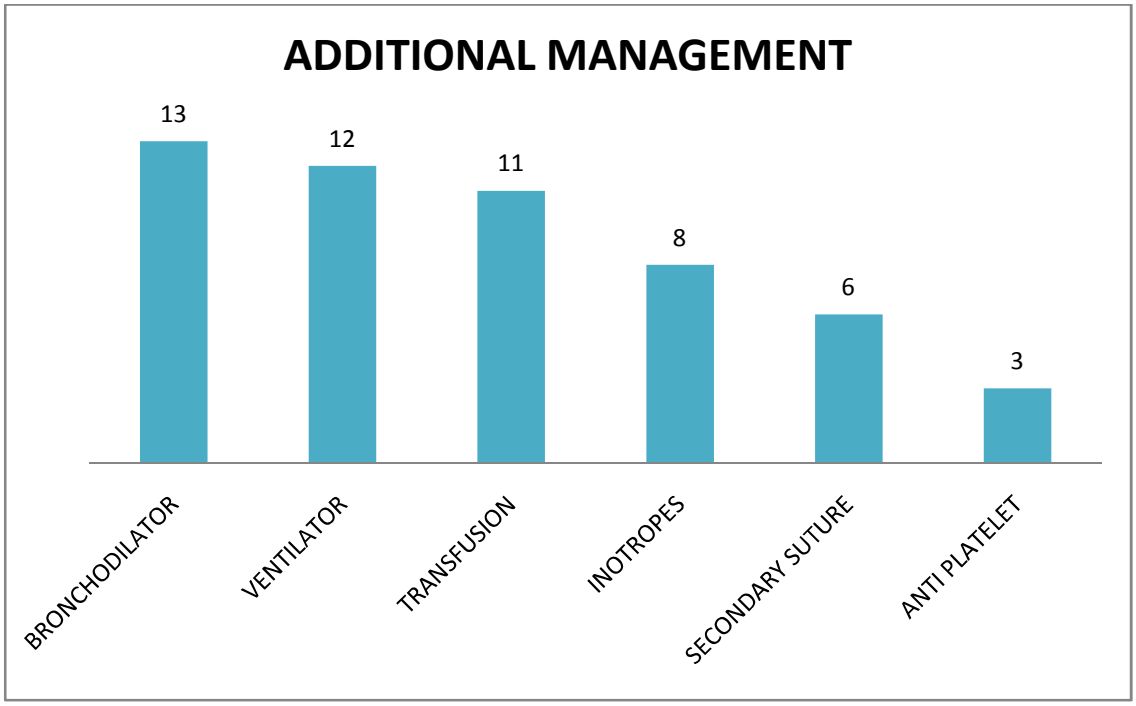
All patients underwent chest physiotherapy. Appropriate management for diabetes mellitus and hypertension was administered.

Additional treatment included;

Table 8; Additional Post Operative Management

Blood Transfusion	11(17.19%)
Inotrophic Support	8 (12.5%)
Bronchodilators	13 (20.31%)
Anti-Platelet medication	3 (4.69%)
Ventilator support	12 (18.75%)
Secondary suturing	6 (9.375%)

Graph 6; Additional Post Operative Management



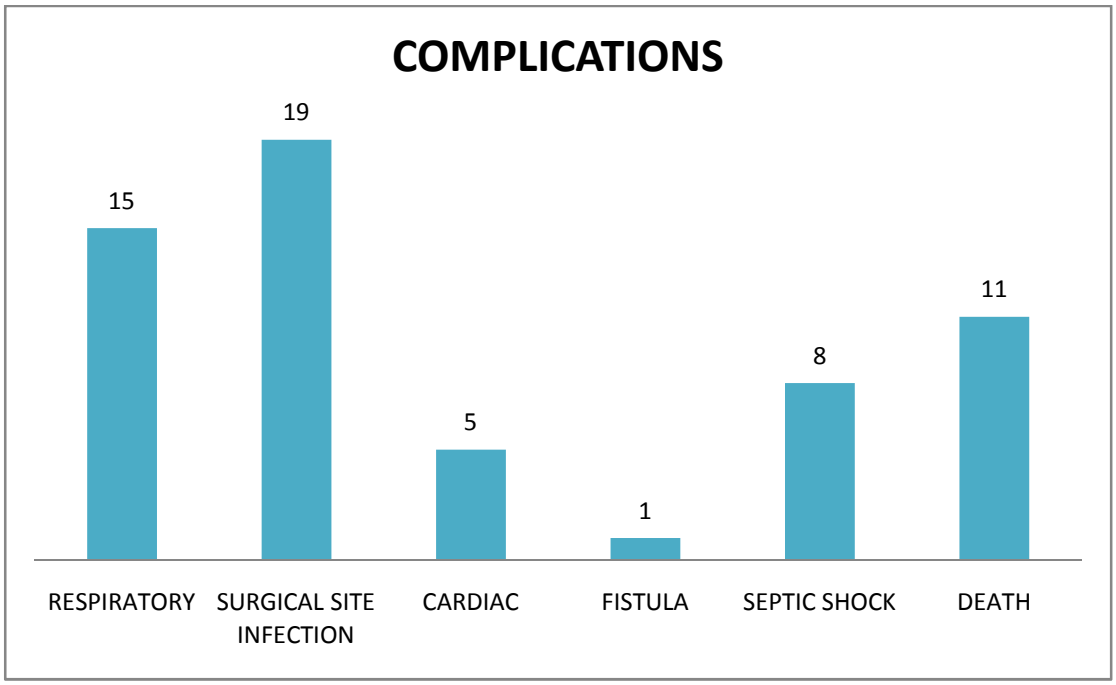
Most common additional post operative management included use of bronchodilators, ventilator support and blood transfusion.

Post operative Complications

Table 9; Post Operative Complications

Respiratory Complications	15 (23.44%)
Cardiac Complications	5 (7.81%)
Surgical Site Infection	19 (29.69%)
Septic Shock	8 (12.5%)
Death	11 (17.19%)
No Complications	25 (39.1%)

Graph 7; Post Operative Complications

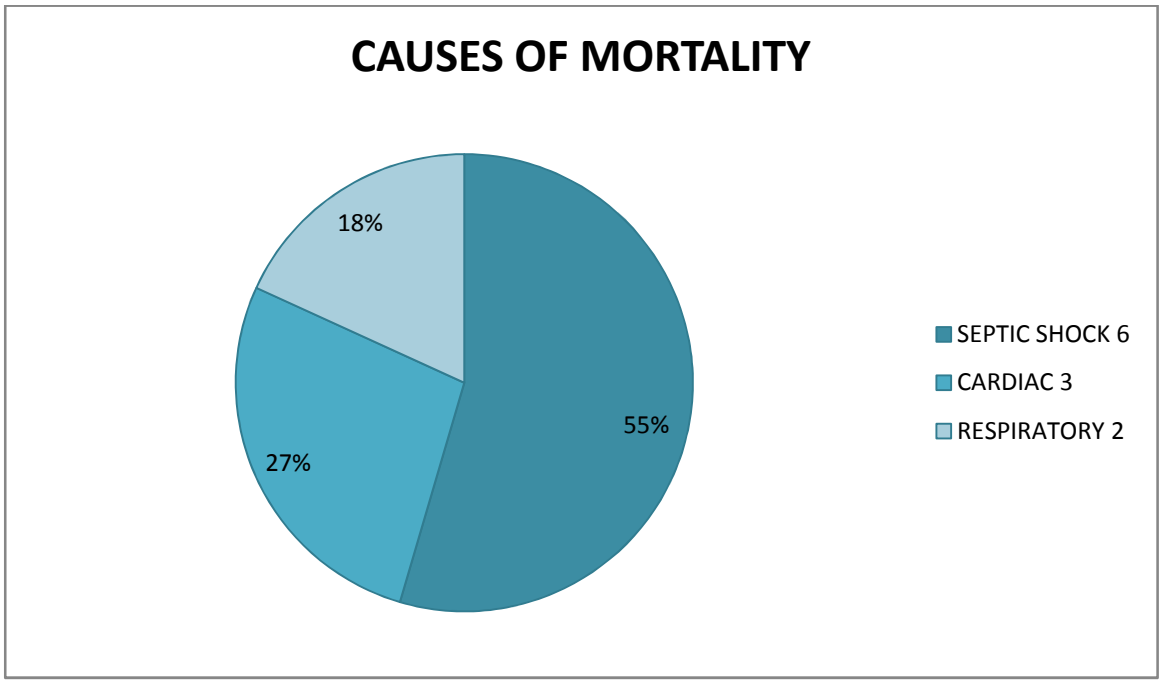


The most common post operative complication was surgical site infection (30%). Mortality was in 11 patients (17%).

3 patients with cardiac complications (myocardial infarction) recovered subsequently with treatment where as 3 patients had death due to cardiac causes.

There were no complications seen to occur in 25 patients (39%).

Different causes of mortality were septic shock in 6 (55%), cardiac causes in 3 (27%) and respiratory failure in 2 (18%).

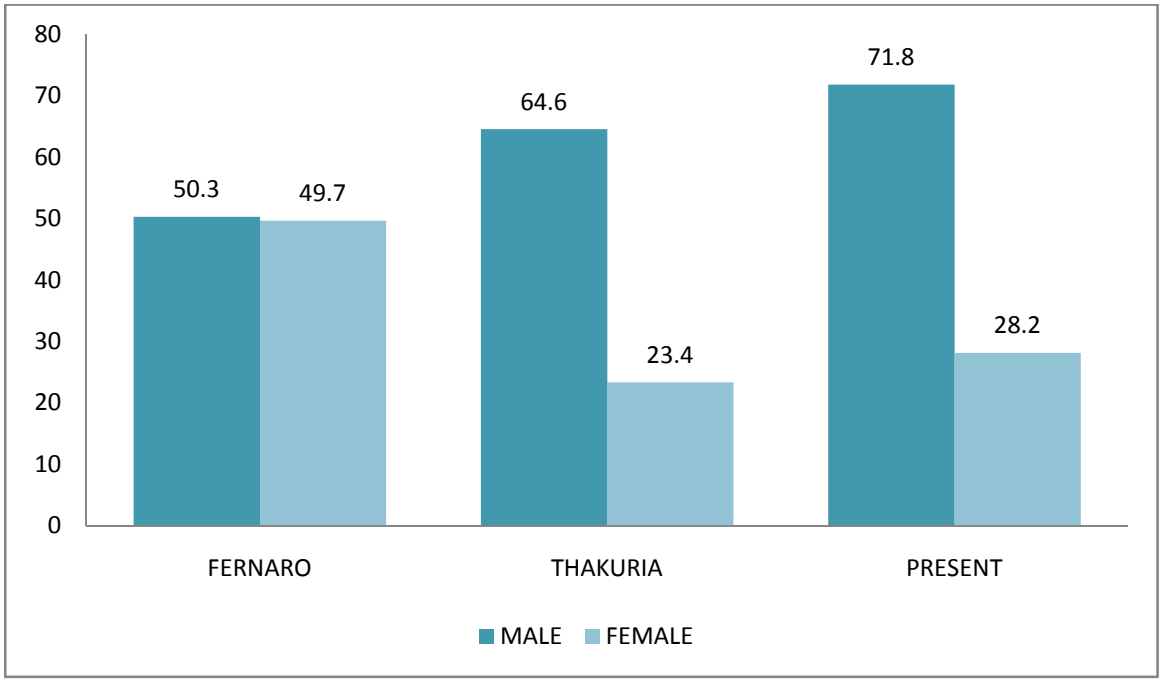


DISCUSSION

Male to female ratio

	Fernaro R[11] (n=718)	Thakuria[6] (n=56)	Present study (n=64)
Male	361 (50.3%)	42 (64.6%)	46 (72%)
Female	357 (49.7%)	18 (23.4%)	18 (28%)

Male to female ratio in present study was more as compared to study on emergency abdominal surgeries by Fernaro R[11] and colleagues from Italy and similar in a study by Thakuria[6] and colleagues in Gauhati, India

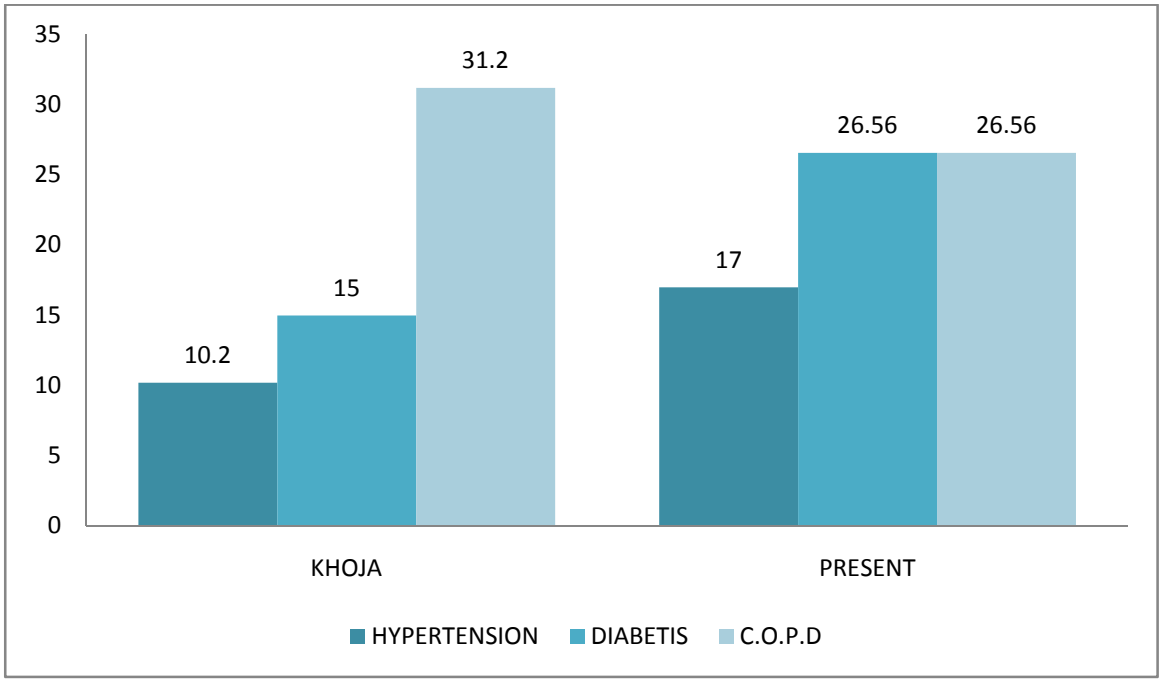


Mean age in Thakuria[6] – 66 years

Mean age in present study – 67 years

Incidence of medical co morbid medical factors

	HYPERTENSION	DIABETES	C.O.P.D
KHOJA H R (n=93)[9]	10 (10.2%)	14 (15%)	29 (31.2%)
PRESENT (n=64)	11 (17%)	17 (26.56%)	17 (26.56%)



Incidence Of chronic obstructive lung disease was found to be more in study by Khoja R and colleagues[9], done in Jodhpur, India, than that in the present study.

28.3% patients in the Khoja R[9] study had anemia.

21.87% Patients in present in the present study had anemia.

Incidence of various surgeries

Etiology	*Present (n=64)*	*Lebeau R*[4] *(n=137)*
Perforated peptic ulcer	24 (37.5%)	29 (21.17)
Obstruction due to adhesions	11 (17.19%)	32 (23.36%)
Cholecystitis	5 (7.8%)	6 (4.4%)
Appendicitis	8 (12.5%)	23 (16.8%)
Trauma	2 (3.125%)	0
Hernia	3 (4.68%)	40 (29.2%)
Gastric volvulus	1 (1.56%)	0
Ruptured liver abscess	1 (1.56%)	4 (2.92%)

Rate of Complications;

COMPLICATIONS	KHOJA HR (n=93)	PRESENT (n=64)
Cardiac	6 (6.45%)	5 (7.8%)
Pulmonary	14 (15%)	15 (23.4%)
Surgical site infections	27 (29%)	19 (29.7%)
Septic shock/ multi organ dysfunction	17 (18.3%)	8 (12.5%)
Death	16 (17.2%)	11 (17.2%)

Rate of complications showed similar results as compared to study on abdominal emergency surgeries by Khoja H R and colleagues

SUMMARY

64 patients aged 6o years or more who underwent emergency abdominal surgery were studied. Their preoperative status and post operative outcomes were studied.

Range of age was 60 to 80 years and mean age was 67 years.

The total abdominal emergency surgeries conducted under all units of surgery department were 412, out of which 64 were in geriatric age group (13%). 72 % of the patients were male and 28 % female.

Most common cause being peptic ulcer perforation (38%) followed by intestinal obstruction due to adhesions (17%).

Co morbid conditions in our study showed 27 % patients having diabetes mellitus and chronic obstructive lung disease, 17 % had hypertension.

Most cases of acute appendicitis (5 out of 8) presented with appendicular perforation.

In our study 61 % patients had post operative complications, of which most common was surgical site infection (30 %) and respiratory complications (23%).

The rate of mortality in our study was 17.2%. Most common cause of death was multi organ dysfunction due to sepsis.

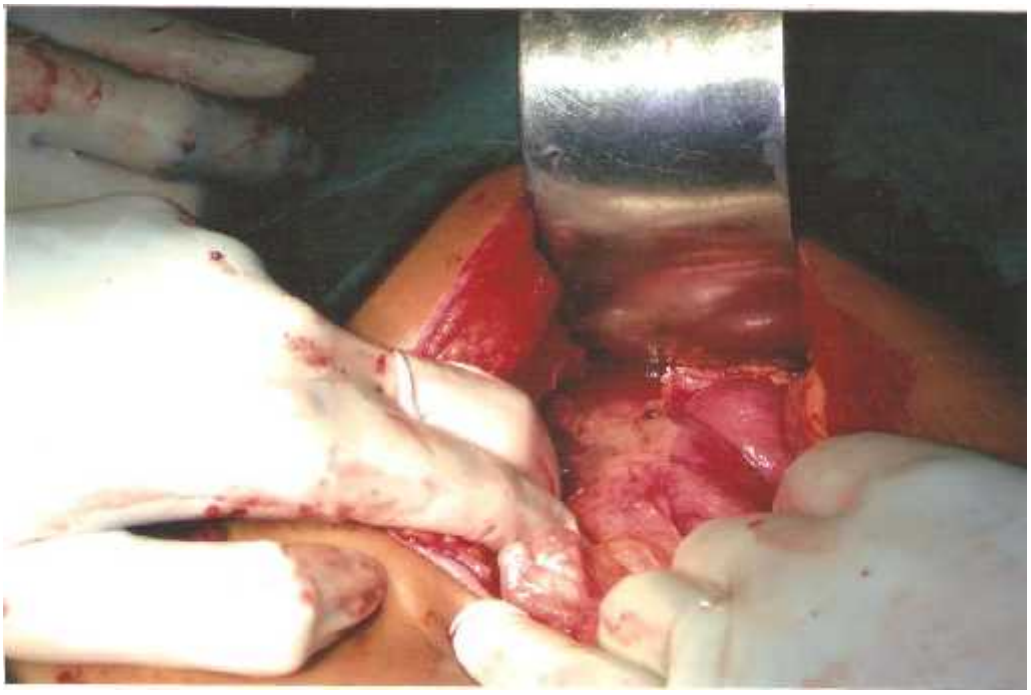
Most common additional post operative management included use of bronchodilators, ventilator support and blood transfusion.

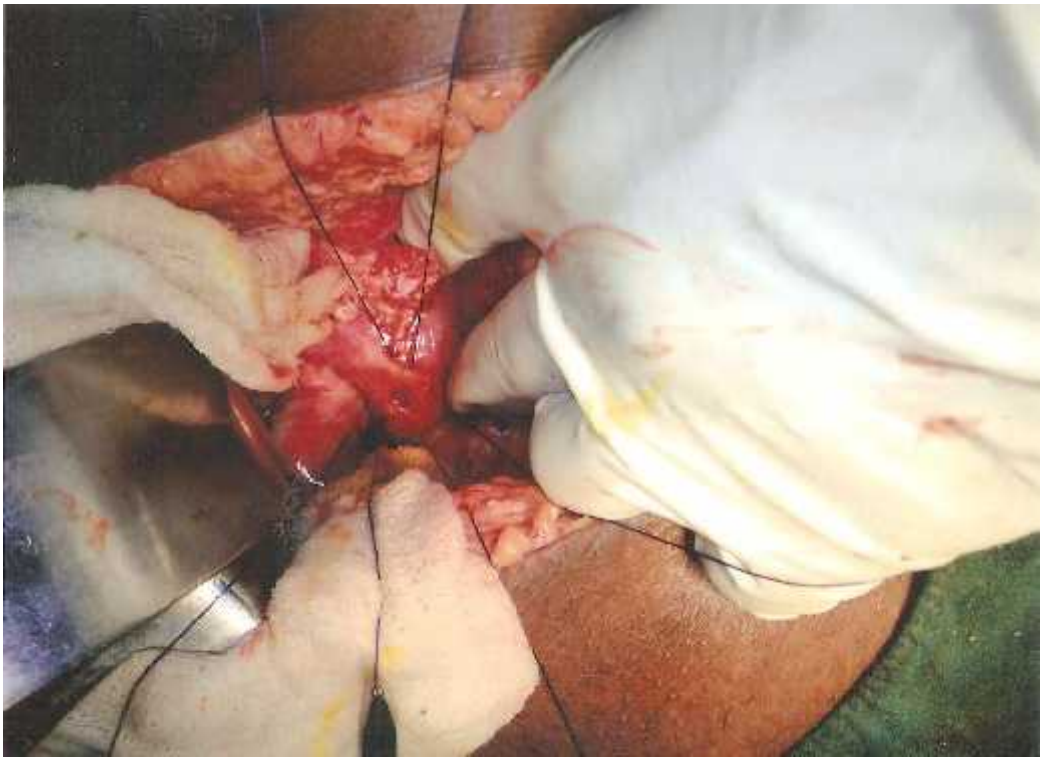
CONCLUSION

Geriatric population is an important subgroup of population undergoing emergency abdominal surgeries (13%) of the total number. Most common cause being peptic ulcer perforation (38%) followed by intestinal obstruction due to adhesions (17%).

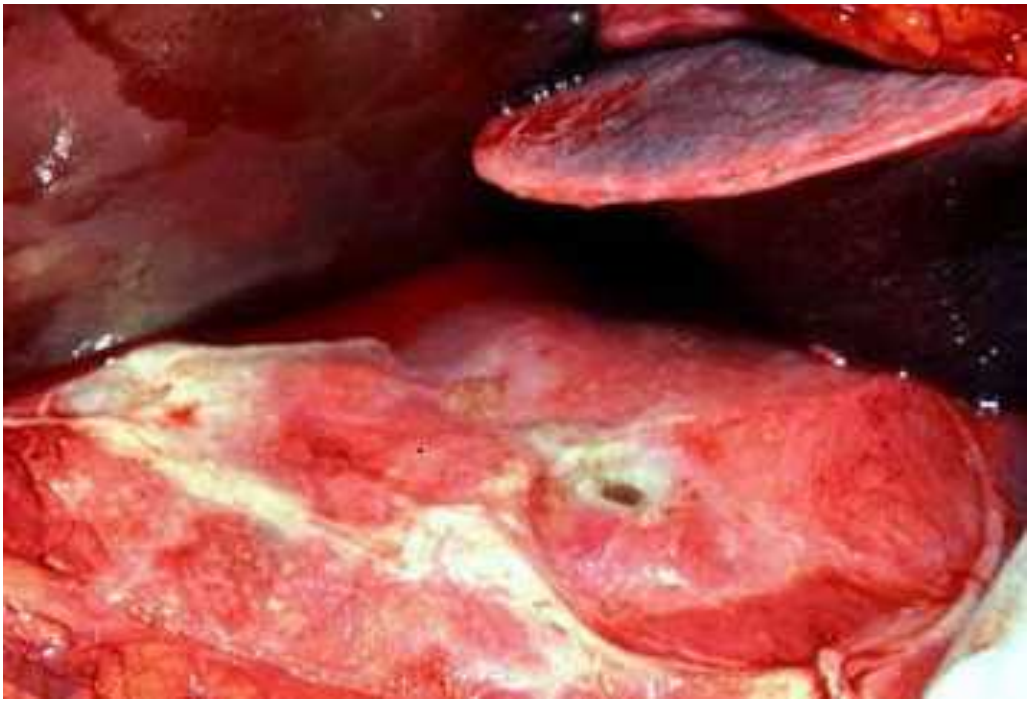
Most common cause of death in abdominal emergency in geriatric patients is septic shock (55%). Thus it indicates that more than the age per say, the delay in presentation may be the cause for mortality in this age group. The therapeutic outcome in patients with co morbid factors like hypertension and diabetes mellitus, in good control, were similar to other patients.

SURGICAL PHOTOGRAPHS

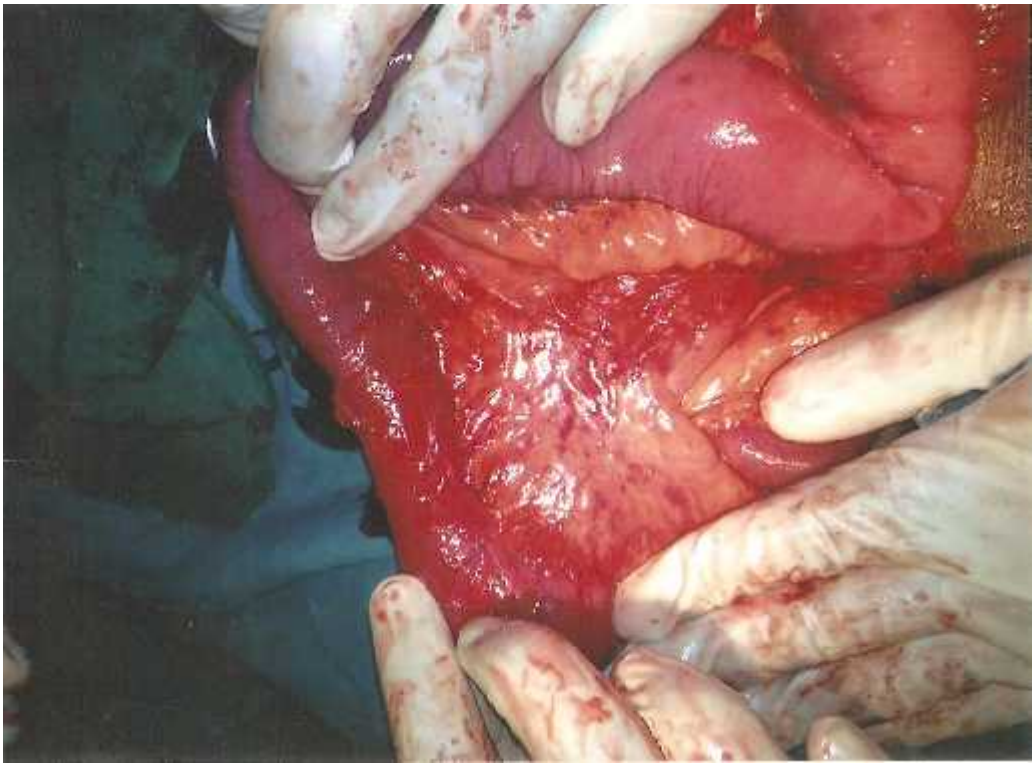




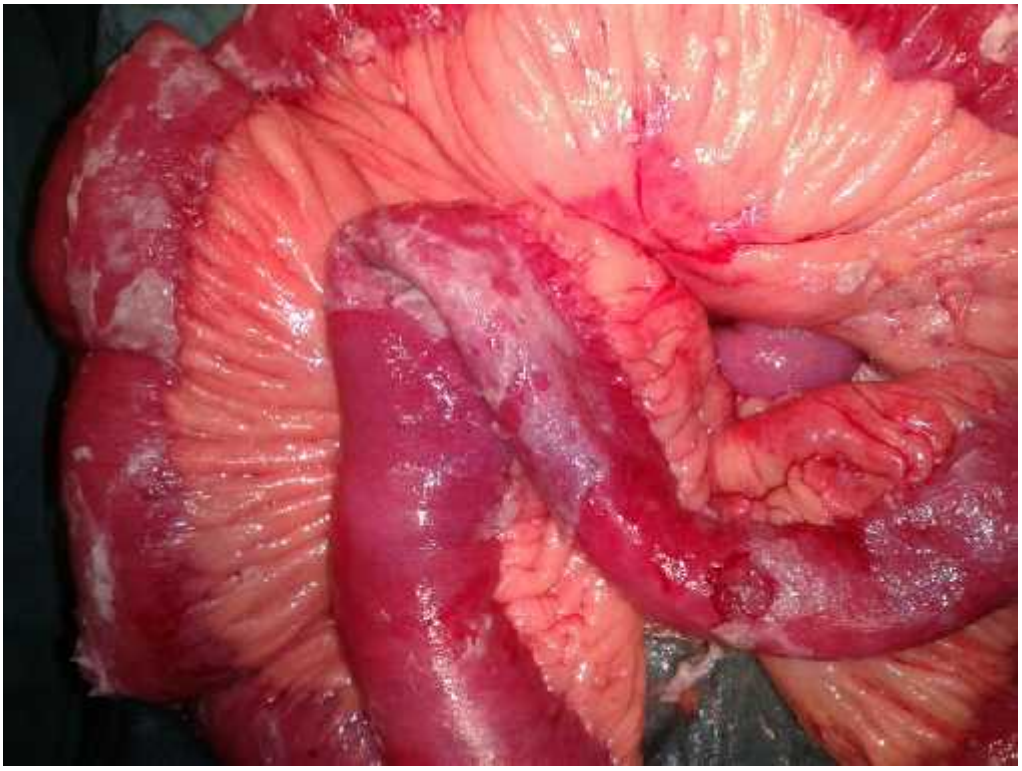
Intra operative photograph of 60 year old patient with pre pyloric perforation.



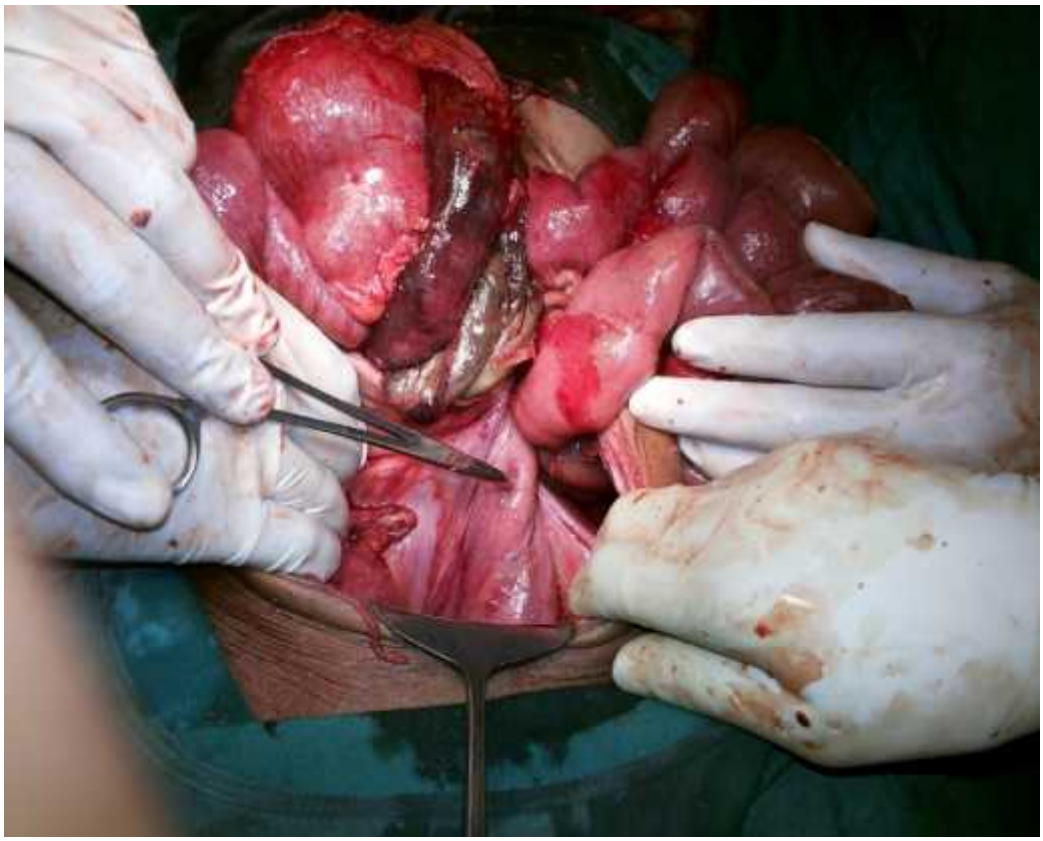
A case of duodenal ulcer perforation in 65 year old male patient.



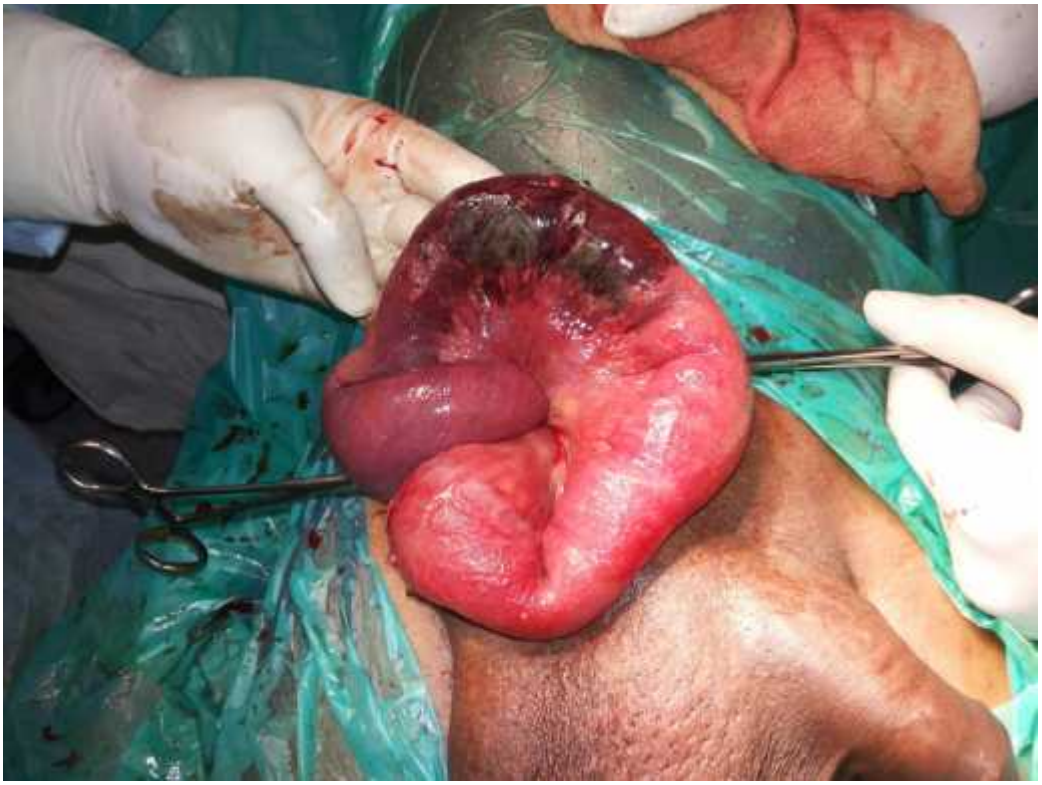
Case of post surgical adhesions leading to acute small bowel obstruction in a 70 year old female patient.



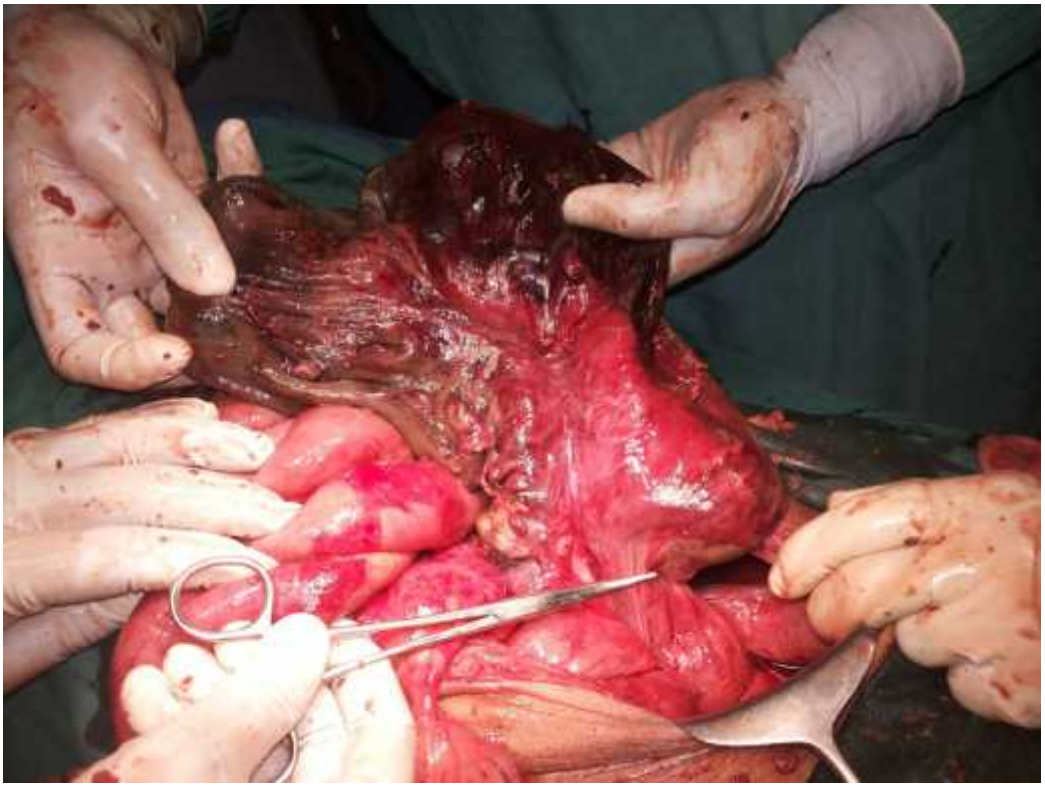
Ileal perforation with flakes in a 70 year old male patient



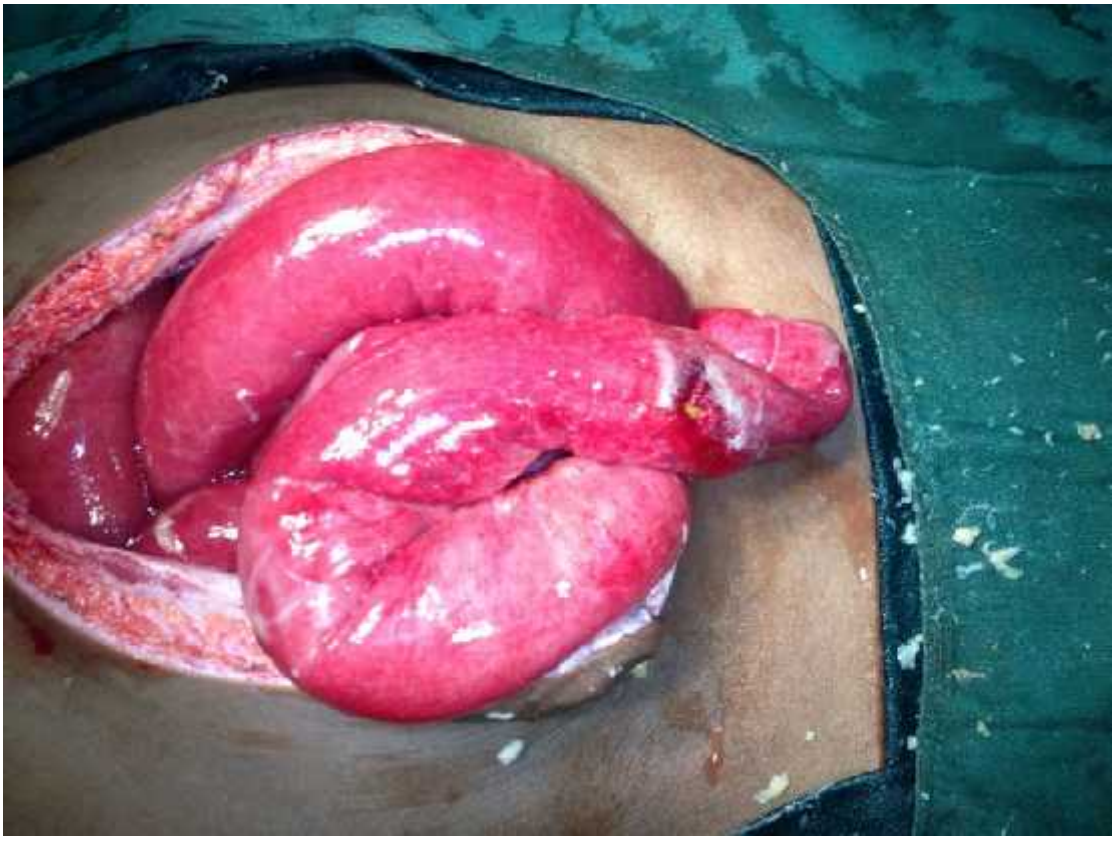
Post hystererectomy adhesions leading to small bowel obstruction and vascular compromise



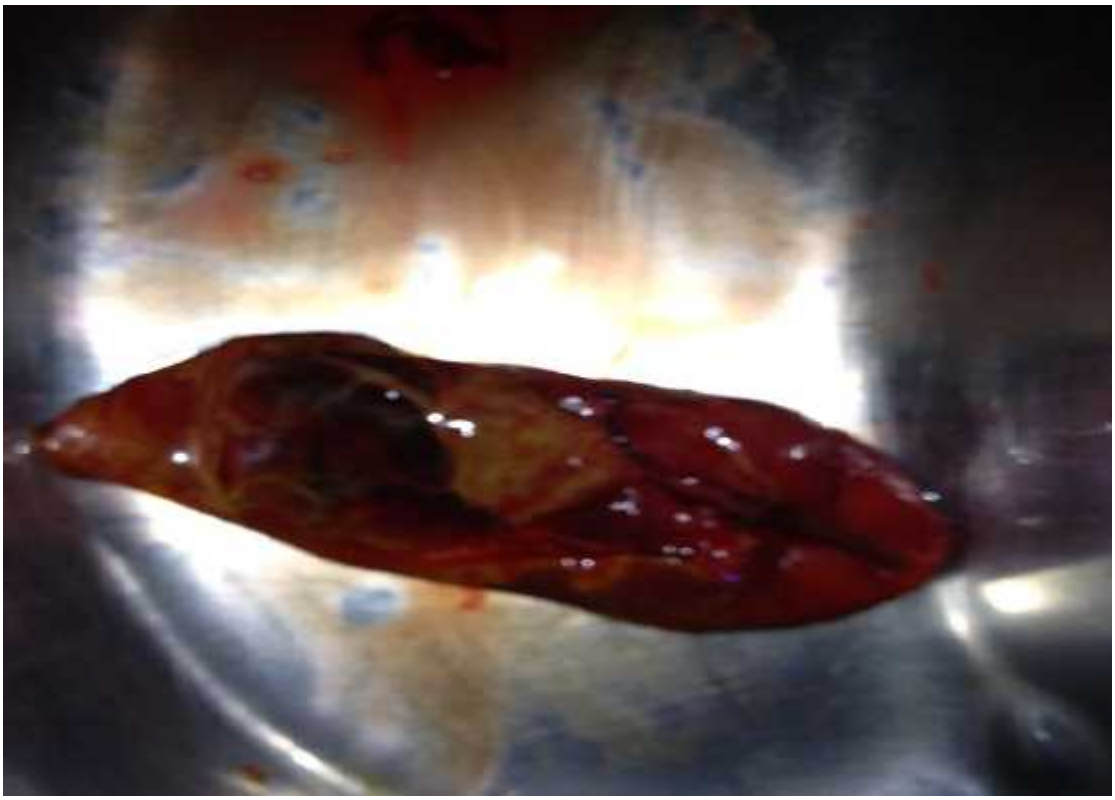
Case of strangulated inguinal hernia with gangrene of a segment of ileum. Underwent resection and primary end to end anastomosis.



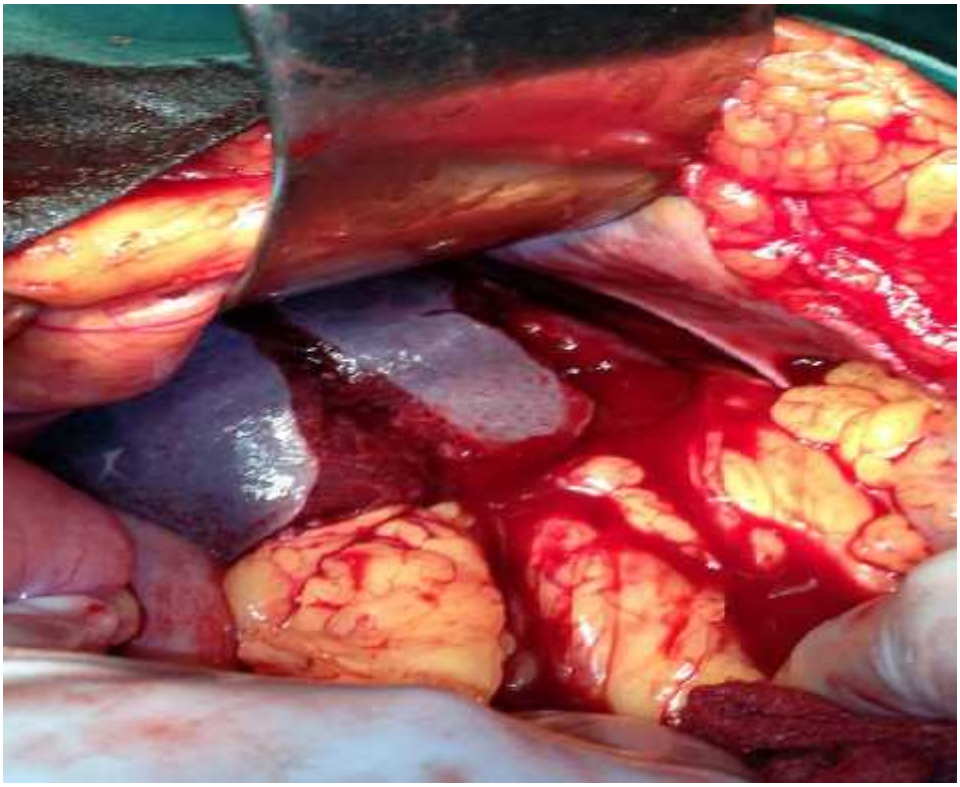
Case of bowel gangrene with peritoneal contamination. Resection of gangrenous bowel with end to end anastomosis was performed.



Case of ileal perforation in a 70 year old male patient.



Gangrenous gall bladder that lead to generalized peritonitis in 60 year old patient.



A case of blunt abdominal trauma that lead to grade 3 splenic injury.

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KEY TO MASTER CHART

M : Male

F : Female

PA : Per Abdomen

CVS : Cardiovascular System

RS : Respiratory System

CNS : Central Nervous System

Prf Per: Perforation with generalized peritonitis

Hb : Haemoglobin

TC : Total Count

DC : Differential Count

ECG : Electrocardiogram

CXR : Chest X Ray

COPD: Chronic Obstructive Pulmonary Disease

LVH : Left Ventricular Hypertrophy

RVH : Right Ventricular Hypertrophy

RBBB: Right Bundle Branch Block

LBBB: Left Bundle Branch Block

TWI : T wave inversion

RAD: Right Axis Deviation

LAD : Left Axis Deviation

ST E : ST segment elevation

GUD : Gas Under Diaphragm

MAF : Multiple Air Fluid Levels

MI : Myocardial Infarction

BPH : Benign Prostatic Hyperplasia

DU Perf: Duodenal Ulcer Perforation

GP : Graham's Patch repair

SBO : Small Bowel Obstruction

App Perf: Appendicular Perforation

GB : Gall Bladder

AC : Acute Cholecystitis

BT : Blood Transfusion

VS : Ventilator Support

SSI : surgical site infection

Bd : Bronchodilators

P : Present

A : Absent

DM : Diabetes Mellitus

HTN : Hypertension

ANNEXURES

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT: **A STUDY OF ABDOMINAL SURGICAL EMERGENCIES IN GERIATRIC PATIENTS**

PRINCIPAL INVESTIGATOR: DR. SHAILESH KANNUR,

POST GRADUATE, GENERAL SURGERY

PG GUIDE **:** DR. B B METAN
M.S. (GENERAL SURGERY)

PROFESSOR OF SURGERY

DEPARTMENT OF SURGERY

PURPOSE OF RESEARCH:

I have been informed that this study will analyse the common etiologies and effect of co-morbid conditions on emergency abdominal surgical conditions in geriatric age group.

I have been explained about the reason for doing this study and selecting me/my relative as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I have been explained that patient that posted for general surgery within 12 hours admission. And sending all investigations which required for study.

RISKS AND DISCOMFORTS:

I understand that I/my relative may experience some nausea or vomiting due to the feeds, may run temperature, there may pain at the operated site, there may be leak from the wound, that I/my relative may lose some weight and that these are expected complications of any exploratory laparotomy, and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

By this study we can ensure availability of better preparedness and better care to a sensitive and more vulnerable subset of population, the geriatric age group and work to improve the outcome in this group of patients coming with acute abdominal surgical conditions.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Shailesh Kannur is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Shailesh Kannur will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my relative, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr B B Metan Dr Shailesh Kannur

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Shailesh Kannur has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

PROFORMA FOR CASE TAKING

SL NO

NAME CASE NO

AGE IP NO

SEX UNIT

RELIGION DOA

OCCUPATION DOO

ADDRESS DOD

SOCIO ECONOMIC STATUS

Complaints:

HISTORY OF PRESENT ILLNESS

HISTORY OF PREVIOUS SURGERY:- PRESEN /ABSENT

HISTORY OF TRAUMA:- PRESENT/ABSENT.

PAST HISTORY

H/O PREVIOUS SURGRY:- ABSENT/PRESENT

H/O TRAUMA PRESENT/ABSENT

PERSONAL HISTORY: SMOKER/ALCOHOLIC

GENERAL PHYSICAL EXAMINATION:

TEMPERATURE PULSE

B.P RESPIRATORY RATE

BUILT: WELL/MODERATE/POOR

NOURISHMENT: WELL/MODERATE/POOR

PALLOR

ICTERUS

PEDAL EDEMA

GENERAL LYMPHADENOPATHY

NUTRITIONAL STATUS:

a. GENERAL APPEARANCE: NORAMAL/THIN

b. ANTHROPOMETRY: HT

WT

SYSTEMIC EXAMINATION:

PER ABDOMEN

RESPIRATORY SYSTEM

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

LABORATORY TESTS

HB%	:	TOTAL COUNT	:
DIFFERENTIAL COUNT:		N/L/E/B/M:	:
PT	:	APTT	:
INR	:	URINE ROUTINE	:
RBS	:	B.UREA	:
S.CREATININE	:	TOTAL PROTEIN	:
S.ALBUMIN	:	HIV	:

HBsAg

CHEST X RAY:

ERRECT ABDOMEN XRAY :

ULTRASONOGRAPHY OF ABDOMEN AND PELVIS:

OTHERS ;

CLINICAL DIAGNOSIS :

OPERATIVE PROCEDURE :

DURATION OF PROCEDURE :

LENGTH OF STAYING IN HOSPITAL AFTER PROCEDURE

POST OPERATIVE INVESTIGATIONS.

POST OPERATIVE COMPLICATIONS

1. RESPIRATORY COMPLICATIONS.
2. URINAY COMPLICATIONS
3. POST OPERATIVE SURGICAL SITE INFECTION
4. CARDIAC
5. SEPTIC SHOCK
6. BLEEDING
7. OTHERS

	I.P No	NAME		SEX	D.O.A	D.O.S	D.O.D	DEATH	OCCUPATION	DURATION OF SYMPTOMS	PAIN ABD	VOMITING	CONSTIPATION	DISTENSION	TRAUMA	H/O ANALGESIC INTAKE	DIABETIS MELLITUS	HYPERTENSION	RESP DIS	PREVIOUS SURGERY	HABITS	*Pallor*	*Icterus*	*Pedal oedema*	*dehydration*	Pulse	*BP*	*PA*	*CVS*	*RS*	*CNS*	*Hb*	*TC*	*N/L/E/B/M*	*UREA*	*CREATININE*	*RBS*	*PROTEIN*	*ALBUMIN*	*ECG*
1	20518	sidram	70	male	10/5/2011	10/5/2011		10/6/2011	farmer	2days	1d	2epi	1d	P		P	P	A	P	no	Sm,alc	P	A	A	P	110	70/40	Prf Per		BC	N	11	3500	84/12/2/2	71	2.2	110	5.8	3.3	RAD
2	22713	shantappa	68	male	10/27/2011	10/28/2011	11/20/2011		carpenter	1d	1d				P		A	A	P	no	Sm	P	A	A	A	74	100/70		N	N	N	9.6	9700	90/7/2/1	32	1.2	109	5.2	2.8	N
3	22568	ramabai	75	female	10/24/2011	10/25/2011	11/1/2011		housewife	4d	4d						A	P	A	no	none	A	A	A	A	76	180/90		htn	N	N	9.7	7700	85/12/3	23	0.9	96	6.6	3.7	LAD
4	24311	bhimappa s	70	male	11/17/2011	11/17/2011	11/30/2011		farmer	2days	2d	3epi	2d	P			A	A	A	no	Sm,alc	P	A	A	P	110	100/70		N	N	n	15.5	8400	86/11/3	41	1	110	5.9	3.7	N
5	25684	bhimappa bajantari	62	male	12/3/2011	12/3/2011	12/20/2011		labourer	3d	3d	3epi	3d	P		P	A	A	P	no	Sm,alc	P	A	A	A	96	154/90	Prf Per	N	BC	n	14.2	2100	89/15/3/2	67	1.7	92			RAD
6	2652	hanumant n	65	male	2/1/2012	12/2/2012		12/2/2012	farmer	1d	2d		1d				A	A	P	no	Sm	P	A	P	P	110	150/90		N	BC	n	11.7	5100	78/20/2	76	2.1	80			N
7	4552	gangabai	65	female	2/24/2012	2/24/2012		3/9/2012	housewife	14d	1d		1d	P		P	A	A	A	no	none	A	A	A	P	118	nr	Prf Per	hypotension	N	n	15.7	10400	86/10/4	53	2.1	102	4.8	1.8	ST E
8	4598	neelappa	60	male	2/25/2012	2/25/2012	3/8/2012		farmer	12d	4d	3epi	2d	P		P	A	A	A	no	Sm	P	A	A	P	nr	nr	Prf Per	hypotension	BC	drowsy	15.6	2500	73/22/3/2	55	2.5	191	4.3	1.9	RAD
9	6056	ratnewwa	70	female	3/15/2012	3/15/2012		3/18/2012	housewife	3d	7d	3epi	2d	P			A	A	A	no	none	P	A	A	A	90	160/80		N	N	n	11.6	5500	75/19/42	62	0.8	162	3.4	1.6	LAD
10	18503	kenchappa	65	male	8/21/2012	8/21/2012	9/1/2012		labourer	10d	1d	2epi	2d	P		P	A	A	A	no	tbc	A	A	A	A	96	150/80	Prf Per	N	N	n	14.6	13100	89/8/3	30	1.1	155	6	3.3	N
11	18583	laxmibai	65	female	8/21/2012	8/23/2012	9/4/2012		housewife	14d	4d	2epi					A	A	A	no	none	A	A	A	A	94	140/90	SBO	N	N	n	11.3	10400	85/13/2	19	1.1	106			TWI
12	18866	kashiray	70	male	8/24/2012	8/25/2012	9/10/2012		farmer	18d	15d	3epi					P	P	A	no	tbc	A	A	A	A	88	180/90	AC	N	N	n	11.2	8600	60/33/5/2	31	1.4	268	7.8	4.8	N
13	20061	ramappa	70	male	9/7/2012	9/7/2012	9/17/2012		farmer	10d	1d	3epi	2d			P	A	P	P	no	Sm	A	A	A	A	70	193/100	Prf Per	N	BC	n	13.4	14100	92/7/1	35	1.6	127	5.8	2.6	LAD
14	20493	neelawwa	60	female	9/11/2012	9/12/2012	9/25/2012		housewife	15d	3d	3epi	3d	P			A	A	P	P		P	A	A	A	90	130/80	Prf Per	N	N	n	12.2	6900	85/13/2	26	1.2	118	5.2	2.5	N
15	22732	laxmibai	80	female	10/8/2012	10/9/2012		10/10/2012	housewife	3d	5d	3epi		P			A	A	A	no		P	A	P	A	104	100/60	SBO	N	BC	drowsy	8.8	8300	86/12/1/1	38	2.1	60	5.5	2.2	N
16	23780	kadappa	60	male	10/19/2012	10/23/2012	5-Nov		farmer	18d	15d	3epi	2d	P			P	A	P	no	Sm,alc	P	A	A	P	110	120/70	SBO	N	BC	n	8	16200	87/10/3	15	0.9	319	5.9	2.8	N
17	24339	pandurang	68	male	10/26/2012	10/26/2012	9-Nov		farmer	15d	3d	3epi	5d	P			A	A	A	no	alc	P	A	A	A	98	90/60	Prf Per	N	N	n	10.6	6300	82/15/3	34	1.3	106	4.8	2.4	N
18	24706	ningamma	63	female	10/30/2012	10/30/2012	11/21/2012		labourer	22d	4d						A	A	A	no	none	P	A	A	A	94	90/60	App Perf	N	BC	n	7.6	8600	59/32/9/2	18	1.2	78	4.1	1.9	LAD
19	25334	maruti	70	male	11/6/2012	11/6/2012	12/10/2012		barber	35d	6d		1d				P	A	A	no	none	A	A	A	A	98	122/80	SBO	N	N	n	11.9	9300	94/5/1	125	2	220	4.4	2.2	N
20	25780	siddappa	60	male	11/11/2012	11/11/2012	11/23/2012		farmer	13d	1d	2epi	2d			P	A	A	A	no	Sm,alc	A	A	A	A	84	120/70	Prf Per	N	N	n	14	12000	84/12/2/2	48	0.9	92	5.4	2.8	N
21	26176	basappa	80	male	11/16/2012	11/16/2012	11/29/2012		farmer	14d	4d		4d	P			A	A	A	P	none	P	A	A	A	74	116/78	SBO	N	N	n	12.3	10600	50/45/3/2	25	1	124			N
22	27550	chandrakant	70	male	11/28/2012	11/30/2012	12/27/2012		labourer	30d	2d	2epi					P	A	A	no	none	P	A	A	A	94	110/80	AC	N	N	n	7.2	20800	91/6/3	106	1.5	224	5	2.6	TWI
23	27748	laxmibai	70	female	11/30/2012	12/4/2012	12/9/2012		housewife	10d	2d	2epi		P			A	A	A	P	none	P	A	A	A	80	122/80	SBO	N	N	n	14.4	5300	79/18/2/1	24	0.8	117	4	2	N
24	28397	shrimanta	80	male	12/5/2012	12/5/2012		12/7/2012	farmer	3d	3d	5epi	2d	P			A	A	P	no	tbc	P	A	A	P	112	90/60	Prf Per	hypotension	BC	n	11	11300	94/3/3	104	2.8	145	5.5	3	TWI
25	28774	mahboobi	65	female	12/7/2012	12/8/2012	12/20/2012		housewife	14d	2d		2d			P	A	P	A	no	none	P	A	A	A	88	170/90	Prf Per	N	N	n	8.6	9900	60/35/5	35	1.1	165	5.1	2.4	LAD
26	29148	kallappa	60	male	12/12/2012	12/15/2012		12/15/2012	labourer	3d	4d	1epi	2d	P	P	P	P	A	A	no	none	A	A	A	A	100	130/80	SBO	N	N	n	13.5	9100	86/11/3	24	0.9	60	6.2	4	TWI
27	30422	shrishail	74	male	12/26/2012	12/26/2012	1/1/2013		farmer	7d	2d	2epi					A	A	A	no	none	A	A	A	A	96	130/80	Ac App	N	N	n	10.9	10300	87/10/2/1	18	0.8	130			N
28	30413	pundalik	60	male	12/26/2012	12/26/2012	1/4/2013		farmer	10d	1d	2epi				P	A	A	A	no	none	P	A	A	A	86	118/70	Prf Per	N	BC	n	11.3	14500	90/8/2	45	1.4	86	6.2	3.2	N
29	54	rudrappa	65	male	1/1/2013	1/1/2013	1/14/2013		labourer	14d	2d	3epi	2d				A	A	A	no	tbc	A	A	A	A	90	112/70	Prf Per	N	N	n	13.2	8900	86/8/3/3	40	1.2	110	5.7	2.8	N
30	1337	honappa	60	male	1/15/2013	1/15/2013	1/31/2013		shopkeeper	16d	3d	2epi				P	P	A	P	no	Sm	P	A	A	A	96	132/80	Prf Per	N	BC	n	14.1	10400	84/14/2	38	1.6	194	5.4	1.8	RBBB
31	1722	somu	65	male	1/19/2013	1/19/2013	2/5/2013		labourer	18d	3d		3d			P	A	A	A	no	Sm,alc	A	A	A	A	100	122/90	Prf Per		BC	n	12.1	9200	82/12/2/4	22	0.8	130	5.9	2.2	N
32	1866	chandramma	70	female	1/21/2013	1/21/2013	2/2/2013		housewife	12d	3d	3epi	3d	P			P	A	A	P	none	A	A	A	A	94	104/60	SBO		N	n	11.4	9600	84/12/2/2	32	1.3	184	5	2.8	N
33	3998	lalsab	70	male	2/12/2013	2/12/2013	2/28/2013		farmer	16d	8d	5epi	2d	P			A	A	A	P	alc	A	A	A	A	96	130/80	SBO	N	BC	n	14.9	21600	89/6/5	34	1	104	4.6	2.8	RAD
34	3958	nagappa	60	male	2/12/2013	2/12/2013	2/24/2013		labourer	12d	1d	2epi	2d			P	A	A	P	no	Sm	P	A	A	A	96	94/60	Prf Per	hypotension	BC	n	12.9	12400	89/6/2/3	52	1.4	104	5.2	2.8	N
35	4548	siddaappa	60	male	2/18/2013	2/18/2013	3/2/2013		shopkeeper	12d	1d	2epi	1d				A	A	P	no	Sm,alc	A	A	A	P	104	128/78	Prf Per	N	N	n	11.4	19100	87/9/4	31	0.8	86	6.2	3.1	N
36	5370	laxman	62	male	2/26/2013	2/26/2013	3/10/2013		labourer	12d	3d	2epi	3d	P			A	P	A	no	tbc	A	A	A	A	88	152/90	Str Hrn	N	BC	n	12.1	8700	82/14/2/2	26	1.1	120	5.4	2.9	RAD
37	5547	jairam	80	male	2/26/2013	2/27/2013	3/15/2013		farmer	18d	3d		3d			P	A	A	P	no	Sm	A	A	A	A	78	140/94	Prf Per	N	BC	n	14.6	9900	78/16/3/2	46	1.5	104	4.4	2.1	TWI
38	5661	gopal	60	male	2/28/2013	2/28/2013	3/12/2013		labourer	13d	2d	3epi	1d			P	A	A	A	no	Sm	A	A	A	A	90	118/70	Prf Per	N	N	n	10.2	8600	86/12/4	28	1.1	122	6	2.9	N
39	7883	rukamawwa	70	female	3/21/2013	3/22/2013		3/25/2013	farmer	4d	3d	2epi	2d	P			A	A	A	P	none	P	A	A	P	110	100/60	SBO	N	BC	n	11.7	11300	74/22/2/2	31	1.2	130	5.4	2.4	ST E
40	7885	natu	65	male	3/21/2013	3/21/2013	3/28/2013		farmer	8d	5d	3epi	3d	P			A	A	P	P	Sm,alc	A	A	A	A	90	122/80	SBO	N	N	n	11.4	9400	78/20/2	30	1.1	124			RAD
41	7749	bandagisab	78	male	3/19/2013	3/21/2013		3/21/2013	farmer	3d	3d	3epi	3d	P			P	A	A	no	alc	P	A	P	P	120	90/60	SBO	hypotension	BC	drowsy	8.6	14300	70/24/4/2	64	2.1	218	4.8	2.1	N
42	8382	golawwa	60	female	3/25/2013	3/26/2013		3/28/2013	housewife	3d	2d	3epi	3d	P			P	A	A	no	none	P	A	P	A	98	94/60	Prf Per	hypotension	BC	n	6.3	2300	85/13/2	107	3.5	91	5.2	2.5	TWI
43	9161	ishwarappa	80	male	4/2/2013	4/2/2013	4/15/2013		farmer	14d	1d	4epi	2d	P			A	A	A	no	Sm	P	A	A	P	110	106/60	SBO	N	BC	n	9.3	21400	87/12/1	50	1.5	192	5.8	3	RAD
44	9438	gurappa	65	male	4/4/2013	4/5/2013	4/15/2013		farmer	11d	1d	2epi	2d				A	A	A	no	Sm,alc	P	A	A	A	90	112/70	Prf Per	N	N	n	10.7	6900	90/8/2	54	1.3	90	5.7	3	N
45	9817	bhagyawanti	72	female	4/8/2013	4/8/2013	4/18/2013		housewife	10d	3d	3epi	1d				P	A	A	no	none	A	A	A	A	98	104/66	Ac App	N	N	n	11.7	12300	89/8/3	38	1.1	196	5.8	2.9	N
46	10358	malakari	64	male	4/14/2013	4/14/2013	4/29/2013		farmer	15d	4d		2d	P			A	A	A	no	alc	A	A	A	A	80	122/70	Str Hrn	N	N	n	12.1	7100	72/23/5	33	1.2	126	6.1	3	N
47	17217	shivanna	61	male	6/4/2013	6/4/2013	6/22/2013		shopkeeper	18d	3d	3epi	2d			P	A	A	P	no	Sm	A	A	A	A	98	118/90	Prf Per	N	BC	n	14.2	8600	80/15/3/2	42	1.4	118	5.4	2.8	N
48	17251	omkar	75	male	6/4/2013	6/4/2013	6/18/2013		labourer	15d	5d	3epi	4d	P			A	P	A	P	none	P	A	P	P	100	140/90	SBO	N	N	n	13.1	7100	72/22/3/3	50	1.6	90	5.6	2.6	N
49	18609	sharmappa	62	male	7/7/2013	7/7/2013	7/28/2013		farmer	22d	2d	2epi	2d				P	A	A	no	none	A	A	A	A	94	112/70	Ac App	N	N	n	11.4	11800	82/12/3/3	24	1.1	126	6	3.1	LBBB
50	18893	mallappagouda	60	male	7/9/2013	7/10/2013	7/29/2013		farmer	20d	2d	3epi					A	P	A	no	Sm,alc	A	P	A	A	80	150/90	AC	N	N	n	13.7	28700	89/5/6	51	1.4	94	5.2	2.3	TWI
51	19346	siddappa	72	male	7/13/2013	7/14/2013	7/30/2013		farmer	18d	2d	2epi					P	P	A	no	alc	P	A	P	A	98	164/90	AC	N	N	n	10.2	14300	88/8/3/1	68	1.8	188	5.7	2.6	RAD
52	21435	mimbaji	62	male	8/4/2012	8/4/2012	8/17/2013		labourer	13d	2d	2epi	1d			P	A	A	P	no	Sm	A	A	A	A	90	112/80	Prf Per	N	N	n	11.6	10900	80/23/4/3	36	1.1	90	6	2.8	N
53	21680	boramma	65	female	8/5/2012	8/6/2012	8/19/2012		housewife	14d	3d	4epi	3d	P			A	P	A	P	none	P	A	P	A	88	150/90	SBO	N	BC	n	13	7800	74/24/2	46	1.4	124	5.4	2.6	N
54	21438	vivekanand	63	male	8/6/2012	8/7/2012	8/18/2012		shopkeeper	12d	1d				P		A	A	A	no	none	A	A	A	A	88	118/80	AC	N	BC	n	6.9	9500	80/16/2/1	24	1.1	109	5.9	2.5	N
55	22343	basavaraj	64	male	8/13/2012	8/13/2012	8/24/2012		farmer	12d	2d	2epi	2d			P	A	A	P	no	Sm	A	A	A	A	98	106/66	Prf Per	N	BC	n	13.8	15400	84/10/3/3	34	1.2	98	5.6	2.7	N
56	22563	bhirappa	65	male	8/13/2012	8/14/2012	8/18/2012		labourer	15d	4d	3epi	3d	P			P	A	A	no	none	P	A	A	A	76	130/90	SBO	N	N	n	8.8	8400	78/19/3	44	1.3	194	5.8	2.7	RBBB
57	23002	sadashiv	63	male	8/18/2012	8/19/2012	9/3/2012		farmer	15d	3d	2epi	1d				P	A	P	no	Sm,alc	P	A	P	P	110	96/60	AC	N	BC	n	9.4	34600	89/9/2	65	1.8	188	5.4	2.5	RAD
58	22860	gurubai	60	female	8/21/2012	8/21/2012	8/30/2012		housewife	10d	3d	4epi	3d	P			P	A	A	P	none	A	A	A	A	80	114/70	SBO	N	N	n	13.2	8100	84/15/1	26	0.9	212	5.4	2.6	N
59	23194	devappa	60	male	8/22/2012	8/22/2012	8/31/2012		shopkeeper	9d	1d	2epi					A	A	A	no	tbc	A	A	A	P	104	108/70	Ac App	N	N	n	11.2	16800	82/16/2	24	0.9	134	6	2.9	N
60	23214	malliksab	65	male	8/21/2012	8/21/2012	8/22/2012		labourer	2days	3d	3epi	3d				A	A	A	no	Sm	P	A	A	A	88	106/74	Prf Per	N	BC	n	14.2	11400	85/12/2/1	54	1.6	148	5.4	2.5	N
61	23434	lalappa	60	male	8/22/2012	8/22/2012	9/2/2012		farmer	11d	2d	2epi	1d				P	A	A	no	tbc	P	A	A	A	90	112/74	Ac App	N	N	n	9.1	13300	78/16/4/2	34	1	118	5.8	2.7	RBBB
62	21468	kashibai	75	female	7/13/2013	7/14/2013	8/7/2013		housewife	24d	3d	2epi	1d				A	P	A	P	none	A	A	A	A	74	160/100	SBO	N	BC	n	13.6	9300	74/23/2/1	24	1.2	98	5.8	3	ST E
63	24345	sangappa	63	male	7/24/2013	7/24/2013	8/4/2013		farmer	12d	3d	1epi					A	P	A	no	none	A	A	P	A	88	112/90	Ac App	N	N	n	11.8	13600	80/8/2	26	0.8	104	6	2.1	N

CHEST X RAY	ERECT ABD	USG ABD	DIAGNOSIS	ASSOCIATED	SURGERY	DURATION	POST OP STAY	ADDITIONAL TREATMENT	COMPLICATIONS	CONCLUSION
A	G U D		DU Perf		GP	120	14	inotropes,VS	SS	death
N	G U D	Free fluid++	posterior gastric perf	fracture femur	primary closure	180	24	BT	RF	delayed stay
LVH	N	N	gastric volvulus		fundoplicn	150	7	BT	none	
COPD	G U D		DU Perf		GP	75	14	none	none	
COPD	G U D	Free fluid++	DU Perf	ACUTE RETENTION	GP	60	16	aspirin	MI	MI, recovered
fibrosis	G U D		ileal perf		resection with stoma	140	1	inotropes,VS	SS, RF	death
N	G U D	Free fluid++	DU Perf	MI	GP	120	14	inotropes,VS	SS, RF	death
COPD	G U D	Free fluid++	DU Perf		GP	130	12	none	SSI	recovered
LVH	MAF		spleenic flexure growth		R and A	200	3	VS support	SSI	death
COPD	G U D		DU Perf		GP	100	9	none	none	
effusion	N	collection with echoes	App Perf		appendicectomy	100	12	none	none	
N	N	acute calculous C	AC	BPH	cholecystectomy	60	15	none	SSI	recovered
LVH	G U D	Free fluid++	DU Perf	COPD	GP	60	9	Bd	RF	recovered
N	G U D	Free fluid++	ileal perf		R and A	110	9	none	none	
COPD	MAF		bowel gangrene	COPD	R and A	140	2	VS support	SS	death
fibrosis	MAF	C	AC	COPD	cholecystectomy	110	16	Bd, BT	RF	recovered
N	G U D		DU Perf		GP	80	15	none	none	
COPD	N	App Perf	App Perf	COPD	appendicectomy	110	22	BT, Bd, 2nd str	RF	recovered
effusion	G U D	Free fluid++	DU Perf		GP	80	34	BT, Bd, 2nd Str	RF	recovered
COPD	G U D	Free fluid++	DU Perf		GP	110	12	none	none	
N	MAF		SBO		adhesiolysis	120	12	none	none	
effusion	ground glass	ruptured liver abscess	ruptured liver abscess	MI	laparotomy lavage	60	28	Bd, BT	RF, SSI	recovered
N	MAF	Free fluid++	ileal perforation		primary repair	135	5	none	none	
COPD	G U D		ileal perforation	TB	ileal R and A	110	2	inotropes,VS	SS	death
LVH	G U D		DU Perf		GP	50	12	BT	none	
N	MAF		SBO	epilepsy	adhesiolysis	30		VS	cardiac arrest	death
N	N	App Perf	Ac App		appendicectomy	75	7	none	none	
COPD	G U D	Free fluid++	DU Perf		GP	80	12	none	none	
N	G U D		DU Perf		GP	70	13	none	none	
COPD	G U D		DU Perf		GP	90	15	Bd	SSI	recovered
COPD	G U D		DU Perf		GP	70	15	none	RF, SSI	recovered
N	MAF		SBO		adhesiolysis	80	14	none	RF, SSI	recovered
COPD	MAF		SBO		R and A	150	17	Bd	RF, SSI	recovered
COPD	G U D		DU Perf		GP	80	13	none	none	
COPD	G U D		DU Perf		GP	80	12	none	none	
COPD	N	bowel gangrene	Str Hrn		R and A	120	11	Bd	RF	recovered
COPD	G U D	Free fluid++	ileal perforation		primary repair	90	15	Bd	RF	recovered
N	G U D		DU Perf		GP	80	12	none	none	
COPD	MAF		SBO	MI	adhesiolysis	80	3	VS	cardiac arrest	death
COPD	MAF	dilated bowel loops	SBO		adhesiolysis	55	7	none	none	
COPD	MAF		bowel gangrene		R and A	90		inotropes,VS,BT	SS,MODS	death
COPD	G U D		DU Perf	MI	GP	90	3	inotropes,VS,BT	SS,MODS	death
COPD	MAF		bowel gangrene		R and A	110	13	Bd	RF, SSI	recovered
N	G U D		DU Perf		GP	90	10	none	none	
N	N	App Perf	App Perf		appendicectomy	70	10	none	SSI	recovered
COPD	N	obstructed rt hernia	Str Hrn		ileal R and A	120	14	Bd	none	
COPD	G U D		DU Perf		GP	90	18	Bd,2nd Str	RF, SSI	recovered
N	MAF	dilated bowel loops	SBO		adhesiolysis	80	14	none	SSI	recovered
N	N	App Perf	App Perf		appendicectomy	80	21	2nd Str	SSI	recovered
effusion	N	gangrenous GB	GB gangrene	MI	cholecystectomy	120	22	2nd Str	SSI	recovered
effusion, LVH	N	calculous C	AC		cholecystectomy	135	17	none	SSI	recovered
N	G U D		DU Perf		GP	70	12	Bd	RF	recovered
effusion	MAF	dilated bowel loops	SBO	MI	adhesiolysis	60	13	aspirin	cardiac arrest	recovered
effusion		grade 3 splenic injury	splenic injury	bladder injury	splenectomy	135	12	BT	none	
COPD	G U D		DU Perf		GP	70	11	none	none	
N	MAF		SBO		adhesiolysis	75	14	BT	SSI	recovered
COPD		acute calculous C	AC, empyema		cholecystectomy	150	14	inotropes, VS	SSI, fistula	recovered
N	MAF	dilated bowel loops	SBO		adhesiolysis	70	9	none	none	
N	N	App Perf	App Perf		appendicectomy	90	8	none	none	
COPD	G U D		Prf Per		drainage tubes	40	2	inotropes,VS	SS	death
N		Ac App	Ac App		appendicectomy	90	10	none	SSI	recovered
LVH	MAF	dilated bowel loops	SBO	MI	adhesiolysis	60	16	aspirin, 2nd Str	cardiac	recovered
N		Ac App	Ac App		appendicectomy	45	8	none	none	