

**“A CLINICAL STUDY OF THE FACTORS AFFECTING
THE OUTCOME OF INTESTINAL RESECTION AND
ANASTOMOSIS”**

A DISSERTATION SUBMITTED TO THE TAMILNADU

Dr. MGR MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of the Regulations

For the award of the Degree of

M.S. (GENERAL SURGERY) BRANCH-I



DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

MAY 2018

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This is to certify that the dissertation entitled “**A clinical study of the factors affecting the outcome of intestinal resection and anastomosis**” is a bonafide research work done by **Dr. AJAY ABRAHAM**, M.S. Postgraduate student in the Department of General Surgery, Tirunelveli Medical College & Hospital, Tirunelveli, in partial fulfilment of the requirement for the degree of M.S. in GENERAL SURGERY.

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This is to certify that the dissertation entitled “**A clinical study of the factors affecting the outcome of intestinal resection and anastomosis**” is a bonafide and genuine research work carried out by **Dr. AJAY ABRAHAM** under the guidance of **Dr. ALEX ARTHUR EDWARDS M.S.**, Associate Professor, Department of General Surgery and HOD, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

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I hereby declare that the dissertation entitled “**A clinical study of the factors affecting the outcome of intestinal resection and anastomosis**” is a bonafide and genuine research work carried out by me under the guidance of **Dr.ALEX ARTHUR EDWARDS, M.S.**, Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

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Lastly, I express my thanks to my patients without whom this study would not have been possible.

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CONTENTS

SL. NO.	TOPIC
1.	INTRODUCTION
2.	REVIEW OF LITERATURE
3.	MATERIALS AND METHODS
4.	RESULTS & DISCUSSION
5.	CONCLUSION
6.	LIMITATIONS
7.	BIBLIOGRAPHY & REFERENCES
8.	ABBREVIATIONS
9.	ANNEXURE I - PROFORMA
10.	ANNEXURE II - CONSENT FORM
11.	ANNEXURE III - MASTER CHART

LIST OF TABLES

SL. NO.	Tables
1.	AGE DISTRIBUTION AND ANASTOMOTIC LEAK
2.	SEX DISTRIBUTION AND ANASTOMOTIC LEAK
3.	COMORBIDITIES DISTRIBUTION
4.	HEMOGLOBIN DISTRIBUTION AND ANASTOMOTIC LEAK
5.	ALBUMIN AND ANASTOMOTIC LEAK
6.	RENAL PARAMETERS AND ANASTOMOTIC LEAK
7.	BLOOD SUGAR AND ANASTOMOTIC LEAK
8.	ETIOLOGY AND ANASTOMOTIC LEAK
9.	DELAY IN PRESENTATION AND ANASTOMOTIC LEAK
10.	ANASTOMOSIS BASED ON TYPE OF BOWEL AND ANASTOMOTIC LEAK
11.	ORIENTATION OF BOWEL AND ANASTOMOTIC LEAK
12.	BLOOD TRANSFUSION AND ANASTOMOTIC LEAK
13.	FFP TRANSFUSION AND ANASTOMOTIC LEAK
14.	TPN TRANSFUSION AND ANASTOMOTIC LEAK

LIST OF FIGURES

SL. NO.	Figures
1.	THE GASTRO INTESTINAL TRACT
2.	WILLIAM STEWART HALSTED
3.	EMIL THEODOR KOCHER
4.	END TO END ANASTOMOSIS
5.	CONNELL'S STITCH
6.	HALSTED SUTURE
7.	LEMBERT'S STITCH
8.	LINEAR STAPLER
9.	CIRCULAR STAPLER
10.	CHEATLE'S CUT

LIST OF CHARTS

SL. NO.	Charts
1.	AGE DISTRIBUTION
2.	SEX DISTRIBUTION
3.	COMORBIDITIES DISTRIBUTION
4.	HEMOGLOBIN DISTRIBUTION IN STUDY POPULATION
5.	ALBUMIN DISTRIBUTION IN STUDY POPULATION
6.	RENAL PARAMETERS DISTRIBUTION
7.	BLOOD SUGAR DISTRIBUTION
8.	ETIOLOGY DISTRIBUTION
9.	DELAY IN PRESENTATION OF THE PATIENT
10.	ANASTOMOSIS BASED ON BOWEL INVOLVED
11.	ANASTOMOSIS BASED ON ORIENTATION OF BOWEL
12.	BLOOD TRANSFUSION
13.	FFP TRANSFUSION
14.	TPN TRANSFUSION

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REF NO:828/GS/2016

PROTOCOL TITLE: A CLINICAL STUDY ASSESSING THE FACTORS AFFECTING THE OUTCOME OF INTESTINAL RESECTION AND ANASTOMOSIS.

PRINCIPAL INVESTIGATOR: Dr. AJAY ABRAHAM, MBBS.,

DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN GENERAL SURGERY

DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear , Dr. Ajay Abraham, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 03.08.2016.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

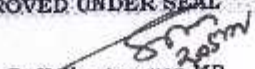
1. TIREC Application Form
2. Study Protocol
3. Department Research Committee Approval
4. Patient Information Document and Consent Form in English and Vernacular Language
5. Investigator's Brochure
6. Proposed Methods for Patient Accrual Proposed
7. Curriculum Vitae of the Principal Investigator
8. Insurance / Compensation Policy
9. Investigator's Agreement with Sponsor
10. Investigator's Undertaking
11. DCGI/DGFT approval
12. Clinical Trial Agreement (CTA)
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)
14. Clinical Trials Registry-India (CTRI) Registration

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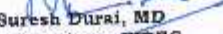
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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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CERTIFICATE - II

This is certify that this dissertation work title **A CLINICAL STUDY OF THE FACTORS AFFECTING THE OUTCOME OF INTESTINAL RESECTION AND ANASTOMOSIS** of the candidate **Dr. AJAY ABRAHAM,MBBS.**, with registration Number **221511351** for the award of **M.S.** in the branch of **GENERAL SURGERY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **2 percentage** of plagiarism in the dissertation.

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INTRODUCTION

Intestines form a major part of human digestive system. Both in terms of length as well as surface area, the small and large intestines constitute about 90% of the digestive system. They play a major role in absorption of nutrients, water and other micro nutrients. Thus they play a major role in growth and proper functioning of the human body. Any pathological condition of the bowel leads to disturbance in the homeostasis of the human body.

Timely intervention and correction of the pathologies affecting the bowel is of utmost importance in providing a healthy functional life to the patient.

One of the most common surgeries done on the intestines is resection and anastomosis. It is the surgical procedure of removing the diseased portion of the bowel and joining the normal viable disease free bowel ends.

History of bowel anastomosis goes back to early 17th and 18th century. Galen was the first person to coin the term “Anastomosis”.

From the 17th century to the modern times intestinal resection and anastomosis remains one of the most common yet very challenging surgeries the surgeon faces. This is the significance of the following study.

The study titled “A clinical study of the factors affecting the outcome of intestinal resection and anastomosis” is a humble attempt to analyse the factors affecting the outcome of bowel anastomosis, so as to implement the factors which produce a favourable anastomotic healing.

THE GASTRO INTESTINAL TRACT

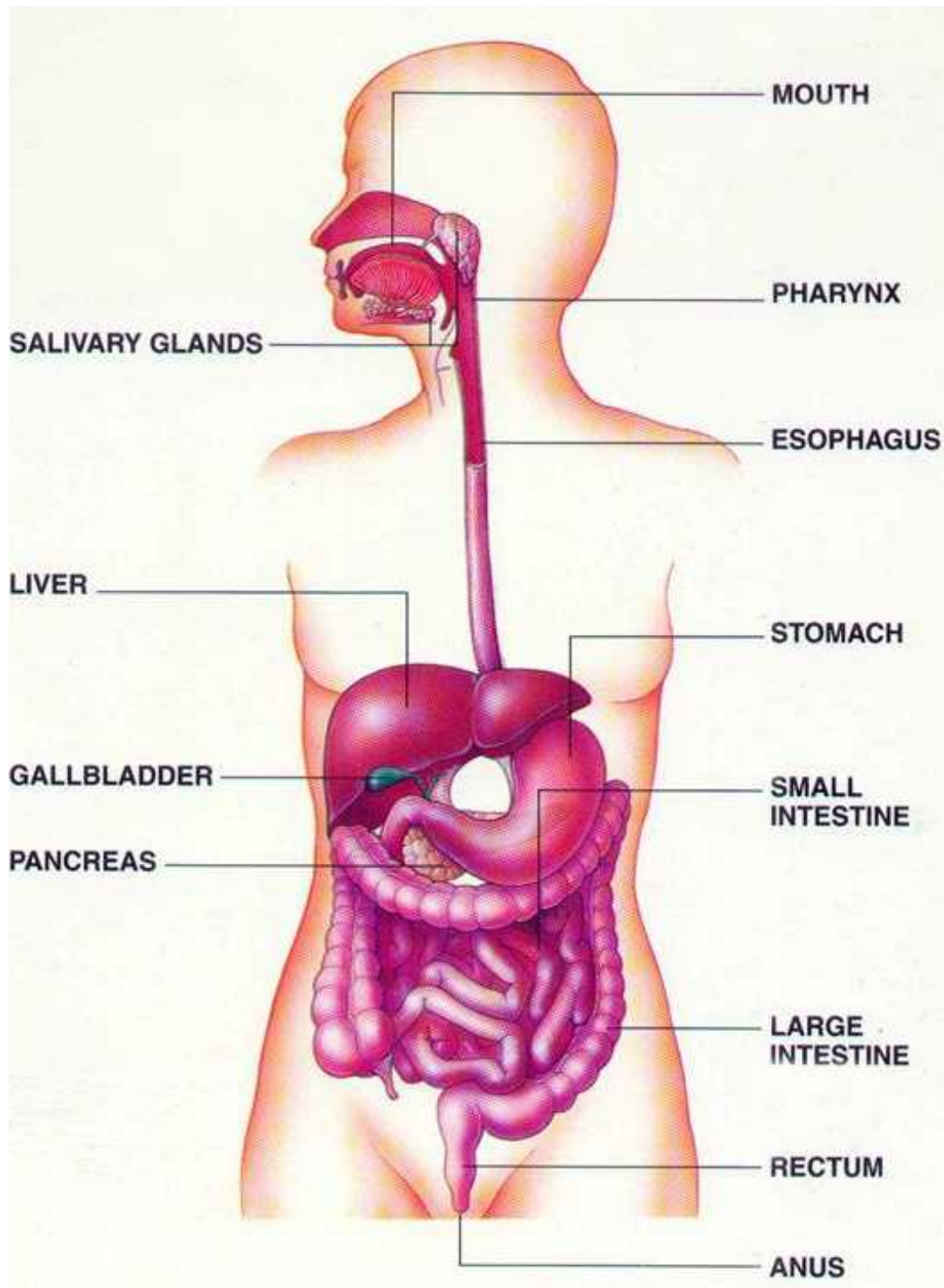


Figure:1

EMBRYOLOGY OF GIT

Germ Layer Contributions

-) Endoderm - epithelium and glands.
-) Mesoderm (splanchnic) - Mesentery, connective tissues, smooth muscle and blood vessels.
-) Ectoderm (neural crest) - enteric nervous system.
-) Both endoderm and mesoderm will contribute to associated organs.

Folding of embryonic disc occurs ventrally around notochord, which forms a rod-like region running rostro-caudally in the midline.

In relation to notochord:

-) **Laterally** lies mesoderm.
-) **Rostrally** lies the buccopharyngeal membrane, above this again is the mesoderm region forming the heart.
-) **Caudally** lies the primitive streak (where gastrulation occurred), below this again is the cloacal membrane.
-) **Dorsally** lies the neural tube then ectoderm.
-) **Ventrally** lies the mesoderm then endoderm.

COELOMIC CAVITY:

- J Mesoderm initially undergoes segmentation to form paraxial, intermediate mesoderm and **lateral plate mesoderm**.
- J Paraxial mesoderm segments into somites and lateral plate mesoderm divides into somatic and **splanchnic mesoderm**.
- J Space forming between them is the **Coelomic cavity**, that will form the 3 major body cavities (pericardial, pleural, **peritoneal**)
- J Most of the gastrointestinal tract will eventually lie within the peritoneal cavity.

PRIMITIVE GUT:

- J The primitive gut forms during the 4th week of gestation when the flat embryonic disc folds in median and horizontal planes to form a tubular structure that incorporates part of the yolk sac into the embryo
- J Ventral folding of lateral sides forms the midgut.
- J Ventral folding of cranial and caudal ends (head and tail folds) form the foregut and the hindgut.

FOREGUT:

- J Gives rise to,
- J Pharynx,

- J Lower respiratory system,
- J Esophagus,
- J Stomach,
- J Proximal duodenum,
- J Liver and the biliary tree,
- J Pancreas.

Partitioning of the foregut by the tracheoesophageal septum:

Esophagus is partitioned from the trachea by the tracheoesophageal septum.

- a. It is initially very short, but elongates rapidly, reaching its final relative length by about week 7.
- b. Elongation is a result of cranial body growth (ascent of the pharynx), development of the heart, and retro flexion of the head.
- c. Endoderm of the oesophagus initially proliferates and almost obliterates the lumen, but recanalizes near the end of the embryonic period
- d. Striated muscle in the upper two-thirds of the esophagus is derived from the mesenchyme of the caudal branchial arches (innervated by cranial nerve X); the smooth muscle of the lower third of the esophagus develops from the surrounding splanchnic mesenchyme (innervated by the visceral nerve splanchnic plexus derived from neural crest cells).

OESOPHAGUS:

- ❖ Elongation occurs during the 2nd month; by the 8th week the proliferating epithelium has partly occluded the lumen.
- ❖ Recanalization occurs during the 3rd month by vacuolation in the multilayered columnar epithelium.
- ❖ Differentiation of stratified squamous epithelium occurs during the 4th month.
- ❖ Induction of muscle formation in the splanchnic mesoderm occurs during the 2nd month in response to signals from the endoderm. Initially only the smooth muscle forms.
- ❖ Transdifferentiation of smooth to skeletal muscle occurs in the upper two-thirds of the oesophagus.

LIVER:

Endoderm and splanchnic mesoderm at the level of the transverse septum (week 4)

- ❖ Stage 11 - hepatic diverticulum development
- ❖ Stage 12 - cell differentiation, septum transversum forming liver stroma, hepatic diverticulum forming hepatic trabeculae
- ❖ Stage 13 - epithelial cord proliferation enmeshing stromal capillaries

The liver initially occupies the entire anterior body. All blood vessels enter the liver (placental, vitelline) and leave to enter the heart.

STOMACH:

This will be considered under 3 aspects:

- Growth,
- Histological differentiation,
- Position adjustment.

a. Growth of the stomach:

-) 4th –8th week the developing stomach grows in all directions to become a sac-like structure.
-) 5th week – the dorsal border grows faster than the ventral border giving rise to the greater and lesser curvatures, respectively 28 days 35 days 56 days Cardiac incisura, Fundus, Lesser curvature and Greater curvature.
-) 8th week the stomach acquires its characteristic shape.

b. Differentiation of the stomach:

-) Foregut endoderm into epithelium, gastric pits, gastric glands.
-) Splanchnic mesoderm into connective tissue, smooth muscle, blood vessels.
-) Neural crest into autonomic nerve plexuses-sub mucosal and myenteric.

c. Histological Differentiation of the Stomach:

Differentiation occurs during the 2nd– 3rd months.

) 8 weeks- rugae, pits, smooth muscle.

) 10 - 20 weeks chief (zymogen), parietal (oxyntic) cells.

) 8 – 9 months: glands become functional secrete HCl and enzymes.

POSITIONADJUSTMENT OF THE STOMACH:

- a. Descent - Due to rapid elongation of the oesophagus, the cardiac end of the stomach descends from C2 at 4 weeks to T11 at 12 weeks.
- b. Tilting - from a vertical position at 4 weeks to an oblique position by 8 weeks. This is due to more rapid growth along the greater curvature 28 days 56 days.
- c. Rotation - 90o around a vertical axis, so that the original dorsal border (greater curvature) becomes left and the original left surface becomes ventral (anterior).
- d. Shift to the left - of both the dorsal mesentery and the stomach.

MESENTERY OF STOMACH:

The stomach develops in the septum transversum and has dorsal and ventral mesenteries.

The liver and spleen develop within the mesenteries of the stomach.

The spleen develops in the dorsal mesentery and liver develops in the ventral mesentery.

ROTATION OF STOMACH:

-) The stomach undergoes 90 degree rotation around cranio-caudal axis during the 5th week.
-) It hinges on the dorsal mesentery and folds 90o to the right.
-) The dorsal mesentery shifts to 8 weeks the left.
-) The vagus nerves serve as markers: Left-ventral, Right-dorsal.
-) The omental bursa forms as a result of rotation of the stomach.
-) As rotation occurs, the dorsal mesentery of the stomach shifts to the left.
-) This occurs by differential proliferation and vacuolation at the broad base of the dorsal mesentery.
-) The stomach is also shifted bodily to the left due to growth of the liver on the right side.

CANALIZATION:

-) Beginning at week 5 endoderm in the GIT wall proliferates
-) By week 6 totally blocking (occluding)
-) over the next two weeks this tissue degenerates reforming a hollow gut tube.

- J By the end of week 8 the GIT endoderm tube is a tube once more.
- J The process is called recanalization (hollow, then solid, then hollow again)
- J Abnormalities in this process can lead to abnormalities such as atresia, stenosis or duplications.

DUODENUM:

- ❖ It is derived from the terminal end of the foregut and the proximal end of the midgut.
- ❖ It receives a dual blood supply from foregut and midgut arteries (coeliac and superior mesenteric).
- ❖ Origins of the liver and pancreatic buds are just proximal to the junction of the two parts.
- ❖ Becomes C-shaped through differential growth.
- ❖ Rotates 90° to the right, the same rotation as occurs in the stomach.
- ❖ Becomes secondarily retroperitoneal, and loses its mesentery. Consequently the pancreas, developing in its mesentery also becomes retroperitoneal.
- ❖ Its lumen is obliterated by rapid cell proliferation during the 2nd month (5-8 weeks) and is re-canalized by apoptosis soon after.

MESENTERY DEVELOPMENT:

-) Ventral mesentery lost except at level of stomach and liver.
-) Contributing the lesser Omentum and falciform ligament.
-) Dorsal mesentery forms the adult structure along the length of the tract and allows blood vessel, lymph and neural connection.
-) At the level of the stomach the dorsal mesogastrium extends as a fold forming the greater Omentum
-) Continues to grow and extend down into the peritoneal cavity and eventually lies anterior to the small intestines.
-) This fold of mesentery will also fuse to form a single sheet.

Errors of the Foregut Development:

- ❖ Errors in partitioning of the laryngo-tracheal tube from the esophagus by the tracheo-esophageal septum result in various forms of esophageal atresia and tracheo-esophageal fistulas (1 in 3000-4500 live births, M>F).
- ❖ Congenital Hypertrophic Pyloric Stenosis is the most common congenital anomaly of the stomach and occurs in 1-8:1000 live births with a 4-6:1 M: F ratio.

- ❖ Pyloric stenosis is a multifactorial and progressive disease that classically presents with non-bilious projectile vomiting in the first few weeks of life.

SPLEEN:

- Mesoderm within the dorsal mesogastrium (week 5) form a long strip of cells adjacent to the forming stomach above the developing pancreas.
- Vascular and immune organ, no direct GIT function.

PANCREAS:

- ❖ It is formed by two buds, dorsal and ventral, originating from the endodermal lining of the duodenum.
- ❖ Whereas the dorsal pancreatic bud is in the dorsal mesentery, the ventral pancreatic bud is close to the bile duct.
- ❖ When the duodenum rotates to the right and becomes C-shaped, the ventral pancreatic bud moves dorsally in a manner similar to the shifting of the entrance of the bile duct.
- ❖ Finally, the ventral bud comes to lie immediately below and behind the dorsal bud.
- ❖ Later, the parenchyma and the duct systems of the dorsal and ventral pancreatic buds fuse.

- ❖ Ventral bud forms the uncinata process and inferior part of the head of the pancreas. The remaining part of the gland is derived from the dorsal bud.
- ❖ The main pancreatic duct (of Wirsung) is formed by the distal part of the dorsal pancreatic duct and the entire ventral pancreatic duct.
- **MIDGUT:**
- gives rise to:
 - Distal duodenum,
 - –Jejunum and ileum,
 - Appendix,
 - Ascending colon,
 - Proximal transverse colon.

MIDGUT HERNIATION:

- At 6 weeks the midgut loop elongates rapidly and the liver enlarges.
- The abdominal cavity becomes relatively small and part of the intestine herniates into the extra-embryonic coelom, through the Coelomic opening next to the umbilical cord.

MIDGUT ROTATION:

- ❖ It forms a U-shaped loop that herniates into the umbilical cord during the 6th weeks of gestation.

- ❖ While in the umbilical cord, the midgut loop rotates 90Degrees.
- ❖ Midgut loop connected to yolk sac via a yolk stalk.
- ❖ During the 10th week of gestation, the midgut loop returns to the abdomen, rotating an additional 180degrees.
- ❖ Sequential return of the gut to the trunk.

Retraction of Herniated Loops:

- ❖ During the 10th week, herniated intestinal loops begin to return to the abdominal cavity.
- ❖ Although the factors responsible for this return are not precisely known, it is thought that regression of the mesonephric kidney, reduced growth of the liver, and expansion of the abdominal cavity play important roles.

Errors in Midgut Development:

- ❖ Omphaloceles result from failure of the intestines to return to the abdominal cavity.
- ❖ Umbilical hernias occur when intestines do return to the abdomen, but later herniate through the umbilicus.
- ❖ Gastroschisis is a linear defect of the abdominal wall that permits extrusion of the viscera without involving the umbilicus.

- ❖ Remnants of the omphalo-mesenteric duct (yolk stalk) are found in 1-4% of infants, making them the most common congenital anomaly of the GI tract.
- ❖ Meckel's or ileal diverticulum accounts for up to 80% of omphalo-mesenteric remnants.

Atresia of the Jejunum, Ileum and Colon:

- ❖ Atresia of the small intestine and colon are rare
- ❖ Most cases are segmental rather than localized. (ie). They involve a long segment of the jejunum or ileum.
- ❖ some cases involve a large segment of the midgut loop derivatives – termed “apple peel” atresia because a short segment of intestine distal to the atresia is coiled around the superior mesenteric artery remnant.
- ❖ Atresia of the jejunum, ileum or colon, unlike duodenal atresia, result from arterial occlusion rather than failure of recanalization.
- ❖ Most cases present as intestinal obstruction a few days after birth.

Malrotation of the gut:

- ❖ Clockwise rotation results in intestinal situs inversus
- ❖ Failure of rotation may result in left-sided caecum, appendix and ascending colon

- ❖ Incomplete rotation – sub hepatic caecum and appendix. Some cases of malrotation are asymptomatic, but may cause diagnostic problems in cases of appendicitis in later life.
- ❖ Volvulus is a rotation of an intestinal loop around a branch of the superior mesenteric artery. It may cause intestinal obstruction or even gangrene. Some cases correct spontaneously, but surgical intervention is usually performed.
- ❖ Intussusception is the invagination of a segment of intestine in itself, causing obstruction.

HINDGUT:

Gives rise to:

- ❖ Distal transverse colon
- ❖ Descending colon, sigmoid, and rectum
- ❖ Proximal anal canal (superior to the pectinate line).

DEVELOPMENT OF HINDGUT:

- ❖ 26 days: After formation of the tail fold, the allantois and hind gut open into a common chamber the cloaca.
- ❖ The cloacal membrane separates cloaca from the proctodaeum.

- ❖ The allantois appears at about 16 days as a small diverticulum projecting from the caudal end of the yolk sac into the connecting stalk.
- ❖ The urorectal septum separates the hindgut from the allantois. It grows towards the cloacal membrane. It is derived from mesoderm at the junction between the connecting stalk and yolk sac.
- ❖ The urorectal septum grows towards the cloacal membrane but does not fuse with it. It is derived from mesoderm at the junction between the connecting stalk and yolk sac.
- ❖ During the 7th week the cloacal membrane disappears, exposing a ventral urogenital sinus opening and a dorsal anal opening.
- ❖ The tip of the urorectal septum, separating the two openings forms the perineal body.

Partitioning of the Cloaca by the Urorectal Septum:

- ❖ During development, a coronal wedge or ridge of mesenchyme, urorectal septum, forms angle between the allantois and hindgut.
- ❖ As the septum grows caudal toward cloacal membrane, it divides the cloaca into an anterior portion, primitive urogenital sinus, and a posterior part, the anorectal canal.
- ❖ By 7 weeks of age, urorectal septum reaches cloacal membrane and fuses with it. Thus, membrane is divided into a posterior anal

membrane and a larger anterior urogenital membrane. The area of fusion of the urorectal septum and the cloacal membrane becomes the primitive perineum or perineal body.

- ❖ In week 9, proliferation of mesenchyme around the anal membrane raises the surrounding ectoderm to form a shallow pit, the anal pit or proctodaeum. The surrounding swellings are called the anal folds.
- ❖ Soon after, the anal membrane, at the bottom of the anal pit, ruptures to establish the anal canal, an open pathway from the rectum to the outside.

Anal Canal:

- ❖ At the end of the 8th week, after rupture of the cloacal membrane, proliferation of ectoderm occludes the anal opening.
- ❖ During the 9th week the opening is recanalized.
- ❖ Thus the terminal part of the anal canal is ectodermal in origin and supplied by the inferior rectal artery.
- ❖ The junction between ectoderm and endoderm is the pectinate line.

ERRORS IN DEVELOPMENT OF HINDGUT:

Hirschsprung's Disease:

- ❖ HD is the partial or total absence of autonomic ganglia resulting from failure of migration of the neural crest cells into the colonic wall during 5th-7th week of gestation.

ANATOMY OF GIT

- ❖ Begins with the oral cavity (mouth and pharynx) where chewing and the secretion of saliva start digestion.
- ❖ Food moves through the GI tract (esophagus to anus)-rings of smooth muscle act as sphincters to separate the tube into segments (esophagus stomach small intestine large intestine) with different functions.
- ❖ Digestive secretions are added to the food by GI epithelium, liver and pancreas, turning it into a soupy mixture called chime.
- ❖ The products of digestion pass out of the lumen into the ECF where they pass into blood or lymph for distribution throughout the body.
- ❖ Any material remaining in the lumen at the end of the GI tract is defecated through the anus.

PERITONEUM:

- ❖ 2 connective tissue membranes in the abdominal cavity which protect the organs in the abdominal cavity from damage by friction/abrasion.
- ❖ Visceral peritoneum
 - covers the external surface of digestive organs.
- ❖ Parietal peritoneum

-line the internal wall of the abdominal cavity.

- ❖ Between the 2 layers of the peritoneum is a peritoneal cavity which is filled with the peritoneal fluid secreted by the cells of each layer.

-fluid functions to lubricate digestive organs, allowing them to slide across one another without creating friction which would lead to inflammation.

Wall of the Alimentary Canal:

4 principle layers of the GI tract

Mucosa (superficial)

- ❖ Inner layer of epithelial, connective and muscular tissues that faces lumen.
- ❖ Sub mucosa
- ❖ loose connective tissue with blood and lymph vessels and submucosal plexus of the Enteric Nervous System.
- ❖ Muscularis
- ❖ 2 layers of smooth muscle (superficial circular and deeper longitudinal) responsible for motility which is innervated by the myenteric plexus of the Enteric Nervous System.
- ❖ Serosa (deep)
- ❖ strong connective tissue membrane that maintains the structural integrity of the alimentary canal (visceral layer of the peritoneum).

MUCOSAL EPITHELIUM OF GIT:

Simple epithelium:

absorptive cells use integral transporting proteins in the apical and basal membranes to absorb ions, water and nutrients out of the lumen into the body by facilitated diffusion, primary and secondary active transport processes.

Secretory cells (endocrine and exocrine)

- ❖ exocytose digestive enzymes and mucus into lumen for digestion and protection against the auto digestion of the mucosa respectively.
- ❖ exocytose hormones and/or paracrine molecules into the ECF for digestive regulation.
- ❖ sensory cells act as mechano- and chemoreceptors which detect the presence of food by the distension of the GI wall and by the presence of specific chemicals (proteins, salts, acids, fats...)

Enteric nervous system:

- A specialized division of the nervous system associated only with the alimentary canal.
- Connected to the CNS via the Parasympathetic NS (stimulates digestion) and Sympathetic NS (inhibits digestion).

- Composed of two major nerve plexuses which send both sensory and motor information throughout the alimentary canal to control digestion.
- Submucosal nerve plexus (submucosa layer)
- associated with mechano- and chemoreceptors in the mucosa.
- controls the endo- and exocrine secretion of the mucosa.
- Myenteric nerve plexus (muscularis layer)
- controls the contraction of smooth muscle.

STOMACH:

Parts of stomach:

-) Proximal= Cardia (attaches to esophagus) attaches at the LES.
-) Fundus= most superior portion, receives food.
-) Body= largest portion, contains parietal, chief and ECL cells.
-) Distal= antrum, contains the G cells.
-) The stomach is almost entirely covered with peritoneum.
-) The peritoneum forms the outer gastric serosa.
-) Beneath the serosa is the Muscularis propria.
-) The MP is made up of 3 layers of smooth muscle.
-) The middle layer is the circular muscle and is the only “complete” layer of muscle.

During the 4th week the 3 distinct portions (fore-, mid- and hind-gut) extend the length of the embryo and will contribute different components of the GIT. These 3 divisions are also later defined by the vascular (artery) supply to each of these divisions.

Foregut - celiac artery (Adult: pharynx, esophagus, stomach, upper duodenum, respiratory tract, liver, gallbladder pancreas).

Midgut - superior mesenteric artery (Adult: lower duodenum, jejunum, ileum, caecum, appendix, ascending colon, half transverse colon).

Hindgut - inferior mesenteric artery (Adult: half transverse colon, descending colon, rectum, superior part anal canal).

SMALL BOWEL:

) Length of the small intestine varies from 10 to 33 feet (3–10 metres).

The average length is considered to be approximately 22 feet (6.5 metres). A considerable length of small bowel can be excised and yet this may be compatible with a normal life. In some cases up to only 18 inches (45 cm) of small bowel has been preserved and the patient has survived satisfactorily.

) Mesentery of the small intestine has a 6 inch (15 cm) origin from the posterior abdominal wall and commences at the duodenal-jejunal junction, just to the left of the second lumbar vertebra. The

mesentery passes downwards towards the right sacral-iliac joint.

-) The mesentery contains the superior mesenteric vessels along with lymphatics and lymph nodes. These drain the small intestine. There are a number of autonomic nerve fibres within the mesentery.
-) Small bowel is divided into three sections. The first section is the duodenum, which is approximately 1 foot in length (25 cm) and extends from the pylorus to the duodenal-jejunal flexure; this point is marked by the ligament of Treitz.
-) Duodenum is anatomically divided into four parts and curves in the shape of the letter C around the head of the pancreas. At its origin the duodenum is covered with peritoneum for about an inch (2.5 cm) after which it becomes a retroperitoneal organ.
-) The upper half of the small intestine is termed the jejunum and the remainder is the ileum. There is no obvious distinction between the two parts and the division is one of convention only. However, the character of the small intestine does change as it is followed distally towards the caecum.
-) The jejunum has a thicker wall as the circular folds of mucosa (valvulae conniventes) are larger and thicker. The proximal small bowel is of greater diameter than the distal small bowel. In addition, the jejunum tends to lie towards the umbilical region of the abdomen

and the ileum to the hypogastrium and pelvis.

) Mesenteric vessels tend to form fewer arcades in the jejunum with long and relatively infrequent terminal branches passing to the intestinal wall. However, the ileum tends to be supplied by shorter and more numerous vessels which arise from a number of complete arcades.

BLOOD SUPPLY OF GASTRO-INTESTINAL TRACT:

SMALL BOWEL:

The small intestine develops from the midgut and this extends from the mid-duodenum to the distal transverse colon and is supplied by the superior mesenteric artery, which arises from the aorta at the level of L1.

The branches of superior mesenteric artery include:

1. The inferior pancreaticoduodenal artery, which supplies the pancreas and duodenum.
2. Jejunal and ileal branches of the superior mesenteric artery; these give the blood supply to the bulk of the small intestine.
3. The ileal-colic artery, which supplies the terminal ileum, the caecum and the proximal part of the ascending colon. This also goes off an appendicular branch to the appendix.

4. The right colic artery, which supplies the ascending colon.
5. The middle colic artery, which supplies the transverse colon to approximately two-thirds along its length. This vessel creates a watershed between the superior mesenteric artery and the inferior mesenteric artery.

The small intestine drains via the superior mesenteric vein and forms a confluence with the splenic vein to form the portal vein. This runs through the free edge of the lesser Omentum and forms part of the superior border to the gastroepiploic foramen, before the portal vein continues to the liver.

PHYSIOLOGY OF GIT

-) The alimentary canal or gastrointestinal (GI) tract is a long muscular tube lined with epithelial tissue passing through the body which is closed off at each end by a sphincter of skeletal muscle.
-) Opens to the outside world therefore the lumen and its contents are part of the external environment.
-) Its primary function is to move water, nutrients and electrolytes from the external environment into the body's internal environment.

MOTILITY:

Contraction of the muscularis causes motility in 2 ways

-) Peristalsis is characterized by progressive waves of contraction that move from one section to the next
-) moves food between 2 and 25 ^{cm}/_{sec}
-) occurs over long distances in esophagus to move food from the pharynx to the stomach and within the stomach where it contributes to the mixing of food
-) occurs over short distances in the small intestine
-) Segmentation is characterized by short segments of the small and large intestines alternately contracting and relaxing which mixes contents and keeps them in contact with absorptive epithelium.

SECRETION AND ABSORPTION:

-) Exocrine (epithelial) cells of the salivary glands, pancreas and liver as well as the GI mucosal cells secrete as much as 7 liters of enzymes, mucus, electrolytes and water into the lumen daily.
-) Occurs in all segments from the mouth to the rectum.
-) The 7 liters of fluid secreted daily into the lumen of the GI tract must be absorbed to prevent dehydration.
-) excessive vomiting or diarrhea can be dangerous.
-) In addition, an average human ingests 2 liters of food and fluid daily that also needs to be absorbed.

) By the time the food and secretions reach the rectum ~98.9% will be absorbed and moved into the body leaving 100 ml of fluid to be defecated.

) Occurs in the small and large intestines.

PANCREAS:

A triangular gland located behind the stomach which has both exocrine and endocrine functions .

Acinar (epithelial) cells secrete pancreatic juice into a duct that empties through the sphincter of Oddi at the duodenum.

Pancreatic islets (islets of Langerhans) secrete the hormones insulin and glucagon to control blood glucose levels.

LIVER AND GALLBLADDER:

) Hepatocytes of the liver secrete bile into the hepatic ducts leading to the gallbladder

) composed of bile acids and phospholipids

) a detergent which causes fat emulsification

-increases the surface area of fat globules.

-increases of lipid hydrolysis by lipase .

) Gallbladder:

) a muscular sac that stores bile secreted from the liver when the sphincter of Oddi is closed.

3 STAGES OF DIGESTION:

) The processes of digestion, secretion, motility and absorption takes place throughout the entire length of the GI tract in 3 overlapping stages named by the location of food.

) Cephalic (head) phase

) thinking about, smelling, or seeing food which has not entered the alimentary canal.

) food is in the mouth.

) Gastric phase

) food is in the stomach.

) Intestinal phase

) food is in the small intestine.

4 Basic Processes of GIT:

) Digestion

) mechanical and chemical breakdown of food.

) Motility

) movement of material along the GI tract.

) Secretion

-) release of substances (hydrolytic enzymes, mucus, acid, bicarbonate, water, ions) from salivary glands, GI epithelial cells, hepatocytes or pancreatic acinar cells into the GI tract lumen or ECF.
-) Absorption
-) active or passive transfer of substances from the lumen of the GI tract to ECF.
-) Processes are regulated by the nervous and endocrine systems as well as paracrine signals.

INTESTINAL ANASTOMOSIS

Intestinal anastomosis is a surgical procedure to establish communication between two formerly distant portions of the intestine. This procedure restores intestinal continuity after removal of a pathologic condition affecting the bowel.

Galen was the first person to coin the term. “Ana” through, “stoma” – mouth.

Anastomosis

Pioneers in anastomosis

1. Lembert – 1826 – sero-muscular technique of suturing
2. Nicholas – 1893 – two layer closure with silk

3. Connell – 1963 – single interrupted full thickness sutures
4. Halsted – Single layer extra mucosal closure
5. Kocher – two layer closure with silk and catgut



Figure: 2



Figure: 3

Ideal anastomosis

1. Zero percentage leakage
2. Early attainment of functionality
3. Early patient recovery and short hospital stay
4. No vascular compromise
5. No luminal narrowing
6. Easy to learn
7. Easy to perform

Need for an anastomosis arises when

1. Part of the bowel is surgically removed
2. Bowel is destroyed by trauma
3. Distal obstruction

Once an anastomosis is made it should heal, giving rise to a healthy continuous channel.

Healing of an anastomosis

Occurs in 3 phases:

1. Acute inflammatory phase
2. Proliferative phase
3. Remodelling or maturation phase

Acute inflammatory phase – it is the early phase of healing of an anastomosis. Occurs 0-4 days after the procedure. There is no intrinsic cohesion between the two ends. There is accumulation of mediators of inflammation at the anastomotic site producing an acute inflammatory response.

Proliferative phase – Occurs 3 to 14 days after the surgery. It is the phase of accumulation and proliferation of fibroblasts. This leads to collagen formation.

Maturation phase – occurs 10 days after the anastomosis. This phase leads to stability and strength to the site. Almost 90% of the tensile strength is gained in 6 months

Types:

Based on the technique of how anastomosis is done:

1. Hand sewn
2. Instrument based – using stapler

Based on bowel included in anastomosis:

1. Small bowel – small bowel eg. Ileo-ileal
2. Small bowel – large bowel eg. Ileo-colic
3. Large bowel – large bowel eg. Colo-colic

Based on orientation of the bowel

1. End to end
2. End to side

3. Side to side

Based on the number of layers

1. Single layer

2. Double layer

In anastomosis like gastro-jejunostomy, the anastomosis is in 4 layers – posterior most sero muscular layer, full thickness posterior 3rd layer, full thickness anterior 2nd layer and finally anterior most sero muscular layer.

In case of anastomosis of small and large bowel, the number of layers are counted as a full thickness suture layer all around, both anteriorly and posteriorly, and then sero muscular sutures all around, making a double layered anastomosis. When the full thickness sutures alone are made, it is said to be a single layer anastomosis. This is frequently done in case of large bowel. Whereas two layered anastomosis is done in case of small bowel.

Indications

Indications for intestinal anastomosis can be broadly divided into two categories: restoration of bowel continuity following resection of diseased bowel and bypass of unresectable diseased bowel. Certain paediatric conditions may also require intestinal anastomosis.

Resection of diseased bowel is performed in the following conditions:

- J Bowel gangrene due to vascular compromise
- J Malignancy
- J Benign conditions like intestinal polyps, intussusception, roundworm infestation with intestinal obstruction
- J Infections like TB causing stricture
- J Multiple perforations
- J Large perforations not amenable to primary closure
- J Radiation enteritis complicated with bleeding, stricture, or perforation
- J Inflammatory bowel disease, ulcerative colitis refractory to medical therapy or with complications
- J Chronic constipation, idiopathic slow transit constipation, or Hirschsprung disease: Subtotal colectomy may be performed if the disease is refractory to medical therapy

Bypass of unresectable diseased bowel is performed in following settings:

- J Locally advanced tumor of bowel causing obstruction
- J Metastatic disease producing intestinal obstruction
- J Poor general condition of the patient

Pediatric conditions for which intestinal anastomosis may be required include the following:

- J Congenital anomalies (e.g., Meckel diverticulum, intestinal atresia, malrotation with volvulus leading to gangrene, Hirschsprung disease)

- J Inflammatory conditions (e.g., necrotizing enteritis, enteric perforation)
- J Other conditions (e.g., intussusception, angiodysplasia, polypoid disease, ascariasis)
- J As a part of other surgical procedures (e.g., Kasai portoenterostomy, choledochal cyst, urinary diversions, pancreatic neoplasms)

Contraindications

Contraindications to intestinal anastomosis include conditions in which there is high risk of anastomotic leak, such as the following:

- J Severe sepsis
- J Poor nutritional status
- J Disseminated malignancy
- J Viability of bowel in doubt
- J Faecal contamination or frank peritonitis
- J Unhealthy bowel condition

Complications

Important complications following intestinal anastomosis include the following:

- J Anastomotic leak
- J Bleeding
- J Wound infection
- J Anastomotic stricture

J Prolonged functional ileus, especially in children

PRINCIPLES OF ANASTOMOSIS

1. Adequate arterial supply and venous drainage should be ensured
2. Anastomosis should be between two disease free ends
3. Do not join ducts without ruling out distal obstruction
4. Direction of peristaltic waves should be considered
5. No tension, no twisting or excessive constriction while anastomosing
6. Avoid back pressure and stagnation

BOWEL ANASTOMOSIS –SPECIAL CONSIDERATIONS

1. Bowel ends should match. If they differ in calibre, an angular cut back can be made on the antimesenteric side of the smaller limb to match the calibre of the bigger limb.
2. Non crushing clamps or stay sutures can be used to hold the two ends.
3. Type of stitch is surgeon dependent. No controlled trials are available comparing these.
4. Use synthetic absorbable suture, mounted on a curved, round bodied, eyeless needle. Smooth monofilament materials have no interstices where microbes can reside. Hence they are safer in contaminated cases. But compared to these, braided multifilament forms reliable knots.

5. Stitches should be taken 3-4mm from the edges and 3-4mm apart from each other.
6. Anastomotic line may lie in the sagittal or coronal plane. If the sutures lie in the sagittal plane, it is easier to work from far to near. If they lie in the coronal plane, start at the end from the non-dominant to the dominant side⁴.
7. Edges should be apposed perfectly, bringing into contact corresponding layers on either side.
8. Stitches produce inflammation and oedema. If they are too tight, blood supply will be cut off leading to delay in healing, mucosal ulceration or worse, anastomotic leak.
9. After completing, check for luminal patency by invaginating the walls on either side through the anastomotic ring.
10. Defect in mesentery should be closed to avoid internal herniation.

METHODS OF ANASTOMOSIS

MOBILE BOWEL, END TO END, SINGLE LAYER, INTERRUPTED

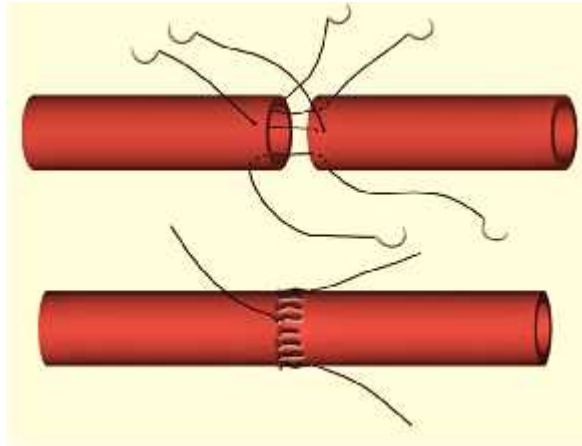


Figure:4

1. Usually used for bowel anastomosis involving mobile bowel ends.
2. Insert sutures in the anterior wall carefully avoiding picking up of posterior wall. Tie the knots outside the bowel.
3. After completing anterior wall, turn the bowel and suture the remaining.
4. Carefully check the mesenteric and antimesenteric edges. Junctions between the anterior and posterior suture lines are most likely to have defects. Insert additional sutures if necessary.

MOBILE BOWEL, END TO END, SINGLE LAYER, CONTINUOUS

1. Suturing is started in the back wall. Started from outside in on one side, inside out on other side and tied. Needle is the inserted back through

- into the lumen and a continuous locked or unlocked spiral stitch is made joining the back walls as far as the other end.
2. If the anastomosis line is in the sagittal plane, start at the near end, complete suturing of the back wall, continue round the far corner and close the anterior walls from far to near to finally reach the starting point. Continuing the suturing in the same fashion requires unnatural action starting with the surgeon's hand held supine and pronating it to drive the sutures through.
 3. CONNELL' STITCH – for right handed surgeons, in order to avoid the above mentioned difficulty, at the far end, having passed the needle to the left side, reverse the needle and pass it from within out creating a loop on the mucosa. The suturing can now be continued naturally from right to left along the anterior wall to reach the starting point. Reverse this for a left hander surgeon.

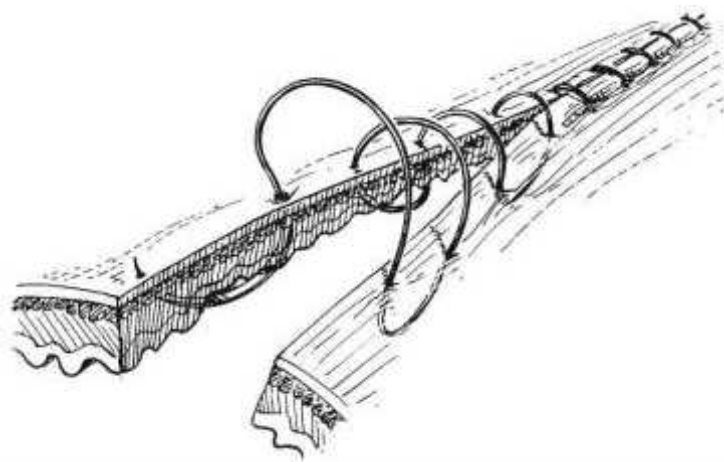


Figure:5

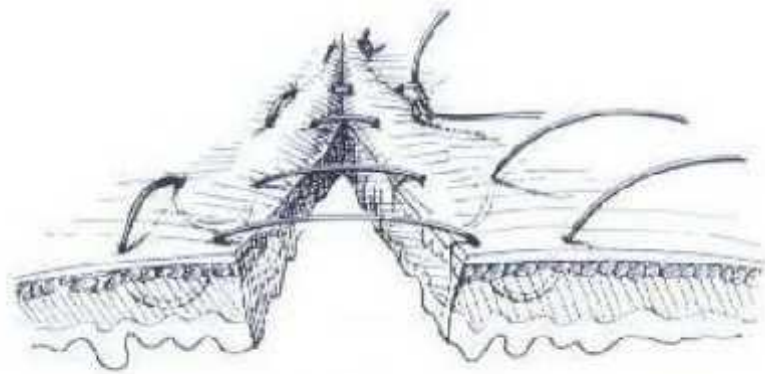


Figure:6 Halsted Suture

FIXED BOWEL, SINGLE LAYER

1. Applicable for large bowel to anastomose it with a fixed structure like rectum. Since rectum lies against the sacrum and cannot be rotated and also since the access is limited we must fashion the anastomosis not at the surface but in the depths¹⁰.
2. Posterior layers are carefully united with all coats sutures with knot is tied inside the lumen⁵. Alternatively, the sutures to the posterior layers can be taken first, held taut and knots fastened at the end, in an order.
3. Posterior layer can also be approximated with vertical mattress sutures or Gambee sutures¹³.
4. Interrupted sutures are then made over the anterior layer to complete the anastomosis.

TWO LAYER ANASTOMOSIS

There is an inner all coats layer which is reinforced by an outer sero-muscular LEMBERT stitch. Often the inner layer of anastomosis is by absorbable suture material and outer layer is by non absorbable material.

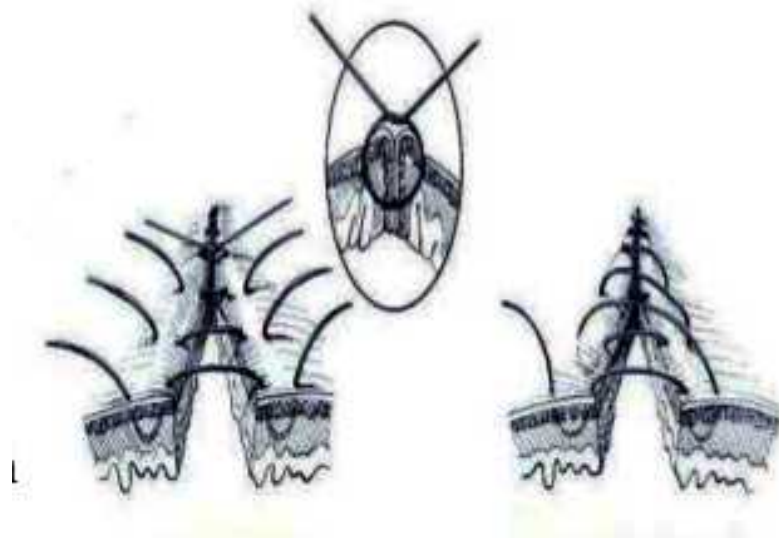


Figure:7

SUTURE MATERIALS

Choice of suture material is surgeon dependent. Knots feel secure with braided material but slippery with monofilament. But monofilament slides better if parachuting is needed. Also the choice between an absorbable and non absorbable suture is also a personal choice. However non absorbable sutures should be avoided in biliary and urinary anastomosis. Materials like silk often act as a nidus for calculus formation later.

RECENT ADVANCES

STAPLING DEVICE BASED ANASTOMOSIS

In recent years stapling devices have been introduced which have improved speed and versatility of anastomosis. They are now being used as an alternative to conventional hand sewn technique.

Advantage – speed and versatility. They can easily access areas which are difficult to approach by hand sewn technique.

Disadvantage – cost.

Stapling devices apply one or more layers of staples to hold the tissue in apposition. When these devices are applied from outside the lumen, the mucosa is held in apposition and an eversion closure is produced. Whereas, when the stapler is employed from within the lumen, serosa is held in apposition, producing an inversion anastomosis.

CLASSIFICATION OF STAPLING DEVICES

1. Linear staplers
2. Circular staplers

Linear Staplers



Figure:8

These devices deliver multiple rows of parallel staples and in certain devices may have additional cutting mechanism in between the rows of the staples.

The staple line may be parallel to or in some devices perpendicular to the handle of the stapling device. These devices may be used according to the accessibility and angulations of the bowel loops to be anastomosed.

Linear staplers are of two types.

- a) Linear staplers without a cutting blade
- b) Linear staplers with a cutting blade

Linear staplers without cutting blade

These staplers fire from outside the bowel and two parallel rows of staplers will appose the mucosa and occlude the lumen. But the bowel tissue is not divided. These devices are often used in bariatric procedures

like gastropexy where they reduce the size of the available stomach, and also to close the rectum below the tumor during anterior resection.

Linear staplers with a cutting blade

These devices deliver four rows of parallel staples and cut between the middle two rows. Hence they are extremely useful in cutting and sealing the cut ends at the same time. Hence these devices are very much helpful in reducing the operating time, especially in elderly patients with co morbidities. They also reduce the contamination of the abdomen while operating on unprepared bowel. Duodenal division during gastrectomy is another surgery where they play a useful role.

When the two blades of the stapler are kept in two different segments of the bowel, firing the device creates an anastomosis. The defects through which the blades are introduced should then be sutured and closed.

These devices are routinely used for gastric anastomosis and creating colonic and ileal pouches.

Smaller versions of these devices can be used for vascular anastomosis as well as in laparoscopy.

Circular staplers



Figure:8

These devices have revolutionised the field of anastomosis especially those involving rectum and oesophagus. This is mainly due to their access to difficult areas.

This instrument can be separated into two separate portions which are later locked together and firing produces a circle shaped anastomosis.

In a classic end to end colorectal anastomosis, the smaller circular head is introduced through the cut end of the mobilised descending colon and is fixed with a purse string suture around it. The locking mechanism alone protrudes out. Then the main body of the instrument is introduced via the anus. The distal bowel wall is securely drawn over the portion of the stapler. Firing of the stapler then produces a secure end to end anastomosis. The complete circular ‘DONUT’ ensures a secure anastomosis.

There are other ways or possibilities of using a circular stapler. When access to an orifice is not available, the main instrument may be introduced through a separate incision made on the bowel wall. Alternatively, the main instrument or the head may be introduced through an open cut end. The opening for introduction may be suture closed after completion of the anastomosis. The accessibility for suture closing this should always be kept in mind before using this procedure.

A circular stapler may also be used to transect and re-anastomose an intact segment of bowel. The instrument is introduced, locked, but separated so that tissue can be drawn into the gun before the gun is closed. Firing produces a single excised donut tissue. This technique is often used in oesophageal transaction for oesophageal varices. Similar principle is often for stapler haemorrhoidectomy.

RISK: Staples that are fired through a great bulk of tissue often produce an insecure anastomosis. This should always be kept in mind while using a circular stapler.

AIM AND OBJECTIVES OF THE STUDY

To identify the patient's clinical and surgical factors that affects the outcome of intestinal resection and anastomosis and predispose to anastomotic leak.

METHODOLOGY:

This study was conducted with the approval of institutional ethical committee. 50 cases requiring intestinal resection and anastomosis admitted in Tirunelveli Medical College were selected after applying inclusion and exclusion criteria.

These cases were followed up intra operatively and post operatively until discharge from hospital or another outcome like anastomotic leak or death of the patient.

INCLUSION CRITERIA:

1. Patients age >18yrs
2. Patients requiring intestinal resection and anastomosis

EXCLUSION CRITERIA:

1. Patient's age <18yrs
2. Pregnant women, prisoners, cognitively impaired subjects
3. Immuno compromised.

Factors studied

In this study the various factors compared and studied can be broadly classified into:

- ❖ Pre operative
- ❖ Intra operative
- ❖ Post operative

Pre operative factors

These factors are patient related. These are non-modifiable factors. In this study the pre operative factors taken into consideration and compared are

1. Age
2. Sex
3. Co morbidities – diabetes, cardiac disease, renal disease
4. Biochemical parameters – Haemoglobin, Albumin, Renal function(Blood urea, serum Creatinine)

Intra operative factors

These are the factors that are influenced by the patient as well as the operating surgeon. These are partly non-modifiable and partly modifiable. This study evaluates how the modifiable factors influence the outcome of intestinal anastomosis

Patient related intra operative factors include

1. Aetiology – Gangrene, malignancy, trauma
2. Delay in surgery – admission to incision time

Surgeon related intra operative factors include

1. Type of anastomosis based on bowel orientation
 - a. End to end
 - b. End to side
 - c. Side to side
2. Type of anastomosis based on bowel involved
 - i. Small bowel- small bowel
 - ii. Small bowel- large bowel
 - iii. Large bowel- large bowel
3. Type of anastomosis based on layers
 - i. Single layer
 - ii. Double layer

Post operative factors studied include

1. Transfusion of blood and blood products
2. TPN transfusion

RESULTS AND DISCUSSION

PRE OPERATIVE FACTORS

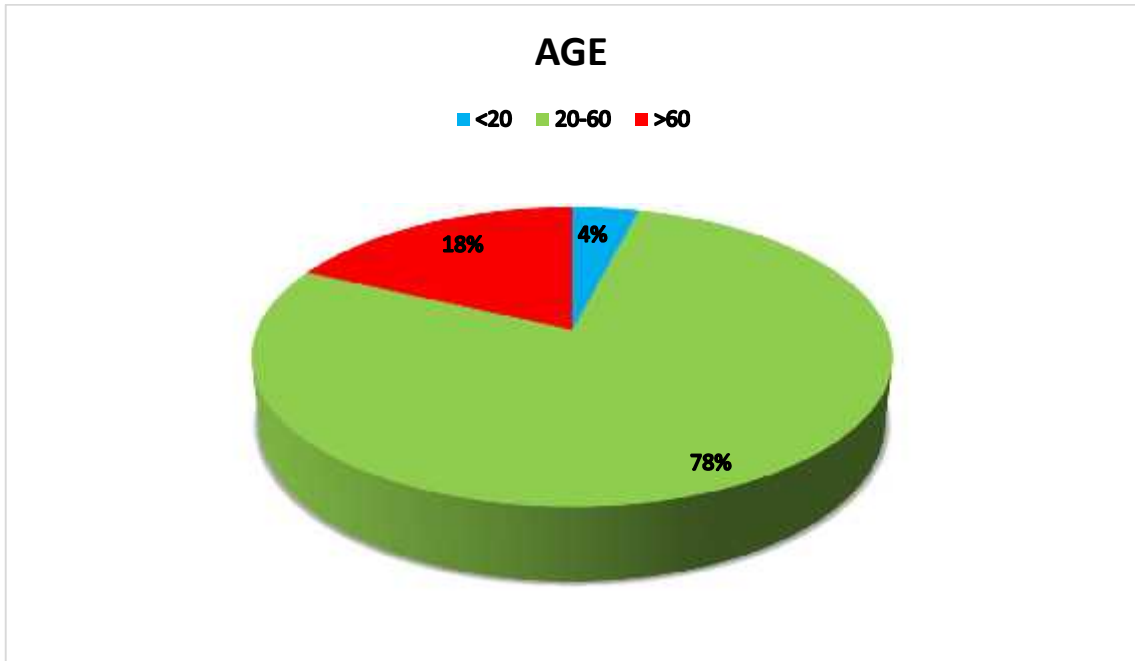
AGE

Age plays an important role in wound healing. As a person ages, the time required for healing increases. This is attributed to factors like collagen production, inflammatory mediators and vascularity. Compared to younger counterparts, the elderly exhibit a delayed or poorer wound healing. This same can be said about anastomotic healing as well.

Table:1

No. of Cases (T=50)			Anastomotic leak	
<20	2	4%	0	0%
20-60	39	78%	3	7.7%
>60	9	18%	3	33.3%

Chart:1



Out of the 50 cases 2 cases were less than 20 years, 39 cases were between 20-60 years and 9 cases were more than 60 years. Upon comparing anastomotic leak in each age group, 33.3% of the patients undergoing anastomosis in the elderly, more than 60 years age group developed anastomotic leak as compared to the 7.7% in the 20-60 years group. None of the patients less than 20 years developed anastomotic leak.

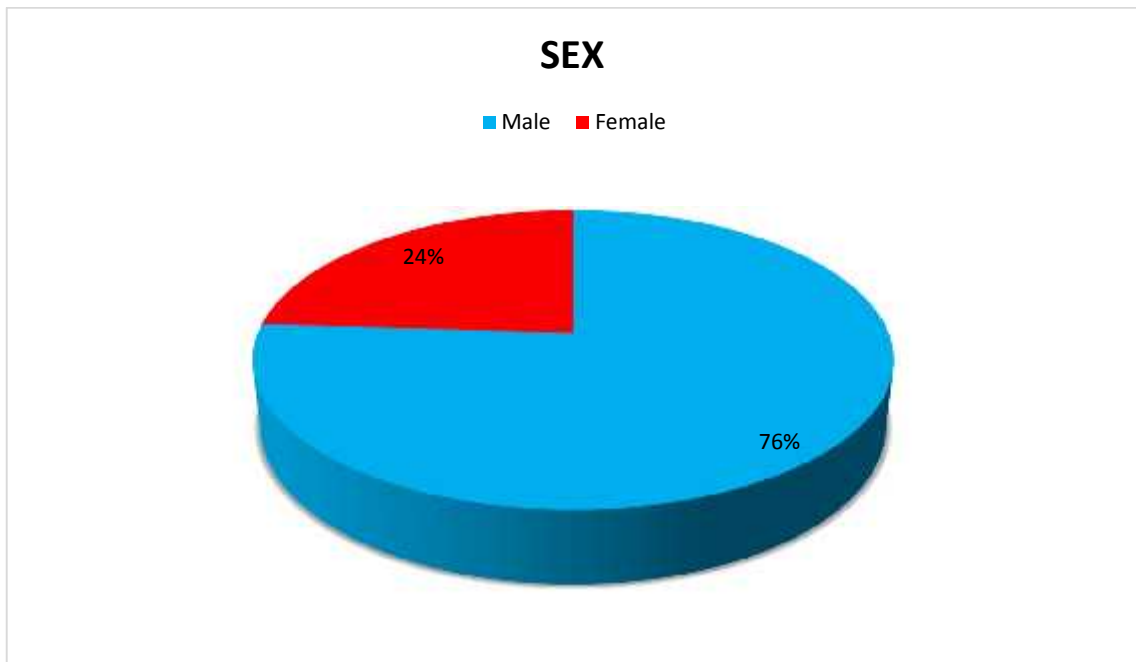
SEX

Hormonal factors come into play when comparing wound healing in male and female sex⁷. Oestrogen plays a proactive role in wound healing. An ongoing Japanese study compares wound healing in normal female mice as compared to mice after removal of ovaries. Early results indicate that wound healing is poorer in mice that have undergone oophorectomy as compared to normal mice. The same could be applicable in case of anastomotic healing.

Table:2

	No of cases (T=50)		Anastomotic leak	
Male	38	76%	6	100%
Female	12	24%	0	0

Chart:2



Out of the total 50 patients including in the study, male patients numbered 38, accounting for 76% of the total study population. Women on the other hand contributed to 24% of the total number.

But upon comparing the percentage of anastomotic leak in both sexes, 100% of the patients having leak were males and none of the female patients developed anastomotic leak.

COMORBIDITIES

Presence of co morbidities play an important role in wound healing. 'Milieu interior' of human beings are altered by presence of co morbidities like diabetes mellitus, cardiac disease and renal disease.

These life style diseases tend to affect the healing property of human body. Hence the time taken for any wound to heal is profoundly increased in patients with diabetes, cardiac and renal disease.

DIABETES MELLITUS

Diabetes mellitus is the most common life style disease among Indian population. National Institute of Health places diabetes in the category of potential epidemic with more than 62 million people currently diagnosed with the same.

Diabetes is an important risk factor affecting healing in patients undergoing bowel surgeries. Diabetes produces a state of decreased perfusion and inadequate angiogenesis, which in turn results in tissue hypoxia which ultimately hinders healing. The levels of Vascular Endothelium derived Growth Factors are decreased in diabetes. This also ultimately leads to inadequate angiogenesis and later poor healing. Hyperglycaemic state directly inhibits wound healing by the formation of advanced glycation end products which result in production of

inflammatory cytokines like TNF-alpha and IL-1 which impair collagen synthesis. Thus the negative effect of diabetes on anastomotic healing is multi factorial.

CARDIAC DISEASE

Cardiac disease points out to two things – atherosclerotic vessels and weakness of heart to pump blood throughout the body.

Atherosclerosis of vessels all around the body results in poor blood supply to all major organs which include the bowel as well. As a result healing is impaired. When an anastomosis is made in a poorly vascularised bowel, the anastomosis is bound to fail and result in anastomotic leak.

The situation is the same when the heart can't pump enough oxygenated blood throughout the body. Oxygen is an essential component of wound healing. So a poor supply of oxygen to the anastomotic site is bound to result in anastomotic failure and leak.

RENAL DISEASE

Kidneys play an important role in metabolism as well as waste management in human body. They play a crucial role in maintaining homeostasis. Hence an adequately functioning kidney is important in all bodily functions including healing. Kidneys, apart from removal of toxic

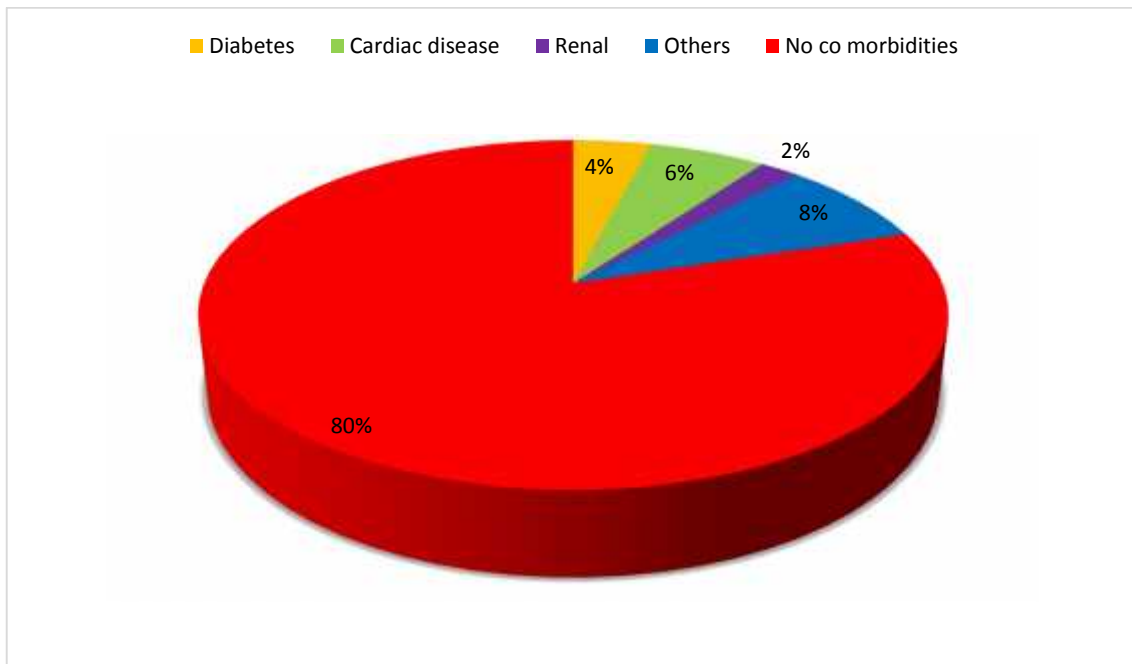
metabolic products, play a critical role in re absorption of essential elements like proteins. This function of kidneys influence wound healing.

Kidney injury ranges from acute kidney injury to chronic kidney disease to end stage renal disease. The common risk factors in patients with chronic and end stage renal disease which influence wound healing are poorly controlled diabetes mellitus, neuropathy, peripheral vascular disease, chronic venous insufficiency and aging. Overall, there is a wide range of uremic toxins which affect mechanisms of wound healing and functioning of multiple systems.

Table:3

	No of cases (T=50)	
Diabetes	2	4%
Cardiac disease	3	6%
Renal	1	2%
Others	4	8%
No comorbidities	40	80%

Chart:3



Overall the number of patients suffering from co morbidities was comparatively less, accounting for only 20% of the total. 40 patients or 80% were free of co morbid conditions.

Only 2 patients or 4% of the total were diabetic. Number of patients suffering from cardiac disease was 3, accounting for 6% of the total. 1 patient was suffering from kidney disease. 4 patients were known to have other co morbidities like hemi paresis.

BIOCHEMICAL PARAMETERS

The biochemical parameters included in the study are:

1. Haemoglobin
2. Serum albumin
3. Blood urea and serum Creatinine

Haemoglobin

It is oxygen-carrying pigment in the red blood cells. Its molecular weight is 64,450. Haemoglobin is a globular molecule which is made up of four subunits. Each subunit contains **heme** (an iron-containing porphyrin derivative). Each heme molecule is conjugated to a polypeptide which is called the **globin**. In each haemoglobin molecule there are 4 chains of polypeptides (2 pairs). In haemoglobin A, which is normal adult human haemoglobin, the two polypeptides are called **chain** and the other two, **chains**.

Synthesis of Haemoglobin

Hb is synthesized inside developing RBC's(In immature RBCs Cytosol) during Intermediate normoblast stages- It begins in the proerythroblasts and continues slightly even into the reticulocyte stage. **Chemical steps in the formation of haemoglobin:**

1. 2 Ketoglutaric acid(it comes from kreb's cycle) + 2 glycine → pyrrole
2. 4 pyrrole → protoporphyrine
3. porphyrine + Fe⁺ → heme
4. 4 heme + 4 polypeptide chain(2 α + 2 β) → 1 haemoglobin molecule

The average haemoglobin-A content in blood is around 15 g/dL. In the whole body of 70-kg man, there are around 900 g of haemoglobin.

Types of haemoglobin

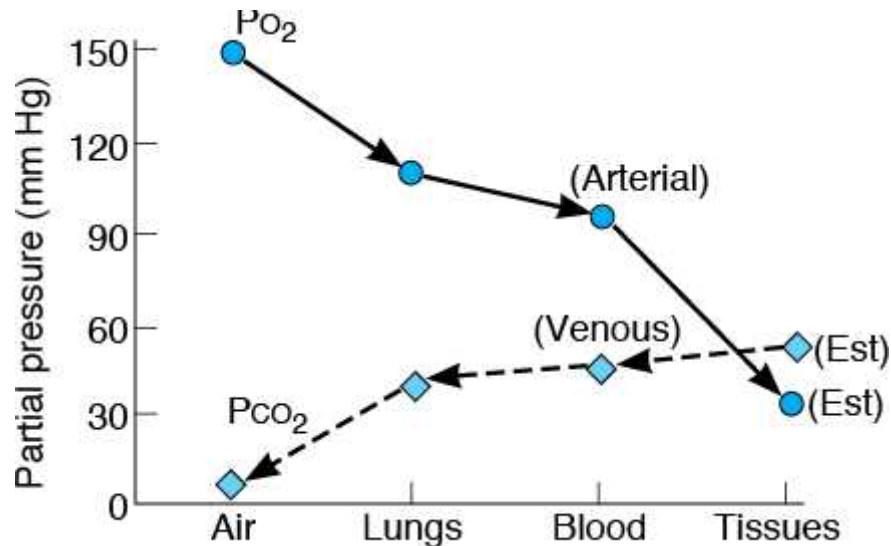
- Haemoglobin A (2, 2 chains)
- Haemoglobin A_{1C} (has a glucose attached to the terminal amino acid valine in each chain, it increases in the blood of people who suffer from diabetes mellitus)
- Haemoglobin A₂ (instead of 2 α chains, there are 2 β chains)
- Haemoglobin F (in the foetus, 2 α chains and 2 β chains)
- Gower 1,2 (in young embryos)

Catabolism of Haemoglobin

When old red blood cell after 120 days are destroyed, the globin portion is split off, the haeme portion is converted to biliverdin and after that it is converted to bilirubin. Bilirubin is a bile pigment which is released

into the blood and later secreted by the liver. The iron from the haeme is either reused for haemoglobin synthesis or excreted out by the body.

Oxygen transportation



The partial pressure gradients for O_2 and CO_2 , plotted in graphical form, emphasize that they are the key to gas movement and that O_2 “flows downhill” from the air through the alveoli and blood into the tissues, whereas CO_2 “flows downhill” from the tissues to the alveoli. However, the amount of both of these gases transported to and from the tissues would be grossly inadequate if it were not for the fact that about 99% of the O_2 that dissolves in the blood combines with the O_2 -carrying protein haemoglobin and that about 94.5% of the CO_2 that dissolves enters into a series of reversible chemical reactions that convert it into other compounds. Thus, the presence of haemoglobin increases the O_2 carrying

capacity of the blood 70-fold, and the reactions of CO₂ increase the blood CO₂ content 17-fold.

ANAEMIA

Anaemia is the decrease in total amount of RBCs over the amount of haemoglobin in the blood or a decreased ability to carry oxygen.

Symptoms of anaemia are usually vague, which includes easy fatigability, weakness, breathlessness etc.

Anaemia is of three types.

1. Due to blood loss
2. Due to decreased red cell production
3. Due to increased red cell break down

Causes of blood loss include trauma and gastro intestinal bleeding. Causes of reduced production include iron deficiency, vitamin B12 deficiency, and thalassemia and bone marrow malignancies. Causes of increased break down include various genetic conditions like sickle cell anaemia, infections like malaria and some autoimmune conditions.

DIAGNOSIS OF ANAEMIA

Anaemia is diagnosed by clinical signs like pallor and biochemical evaluation of level of haemoglobin.

Pallor is elicited by clinical examination of lower palpebral conjunctiva, tongue and tip of fingers.

Haemoglobin estimation helps in quantitative assessment of anaemia. Normal haemoglobin concentration in men is 13g/dl, women is 12g/dl and children it is 11g/dl. A haemoglobin concentration of less than the normal denotes anaemia in each of the categories.

From a surgical point of view, a haemoglobin concentration of less than 10g/dl is inferred as surgically significant anaemia.

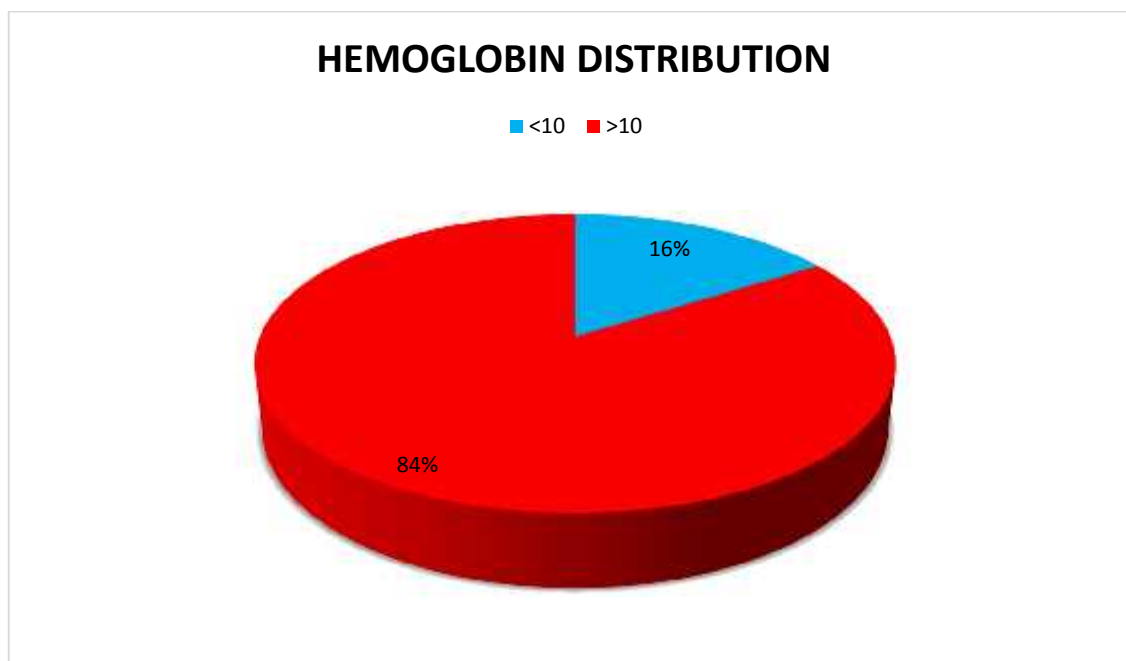
ANAEMIA AND BOWEL ANASTOMOSIS HEALING

When there is anaemia, tissues throughout the body don't receive enough oxygen. The process of wound healing relies heavily on oxygenation. When there is low level of oxygen due to anaemia it halts or slows the wound healing stages, which makes the patient more susceptible to delay in healing. A similar phenomenon happens after a bowel anastomosis. The process of healing at the anastomotic site usually occurs from day 0 to day 4. So when there is reduced tissue perfusion it compromises the healing process in the site of anastomosis and thereby increasing the chances of anastomotic leak.

Table:4

Hb	No of cases (T=50)		Anastomotic leak	
<10	8	16%	2	25%
>10	42	84%	4	9.5%

Chart:4



In this study 8 out of 50 cases studied had a haemoglobin concentration less than 10g/dl. Upon following the patients postoperatively, 2 out of the 8 anaemic patients developed anastomotic, i.e. 25% of the anaemic patients developed anastomotic leak.

On comparison, out of the 42 patients with haemoglobin more than 10g/dl, 4 patients developed anastomotic leak. This amounts to only 9.5% of the 42 patients.

So presence of anaemia in patients undergoing anastomosis is a factor which adversely affects healing and predisposes to anastomotic leak.

ALBUMIN

Serum albumin is a globular protein molecule found in blood. It is the most abundant protein in plasma, constituting half of the total serum proteins. It is produced by the human liver. Albumin is synthesized in the liver as prealbumin, which has an N-terminal peptide that is removed before the nascent protein is released from the rough endoplasmic reticulum. The released prealbumin is cleaved in the Golgi apparatus to produce the secreted serum albumin.

Gene for albumin is located on chromosome 4. Albumin is a heavy molecule weighing about 66.5kDa.

It has a half life of about 20 days.

Normal reference range of albumin in the serum is **3.5-5 g/dl**.

Function of albumin

- Transports hormones, fatty acids etc
- Transports unconjugated bilirubin
- Binds calcium ions
- Buffers pH
- Maintains oncotic pressure
- It is a negative acute phase protein and is a marker of an inflammatory state.

Biochemical evaluation of Serum Albumin

Serum albumin is commonly measured by recording the change in absorbance on binding to bromocresol green or bromocresol purple. Modern laboratory equipments have made quantitative assessment of serum albumin and other serum proteins.

HYPO ALBUMINEMIA

Hypoalbuminemia means a low level of serum albumin. This can be caused by:

- Liver disease
- Excess excretion by kidneys
- Excess loss in bowel like protein losing enteropathy
- Burns

- Redistribution as in pregnancy
- Malnutrition

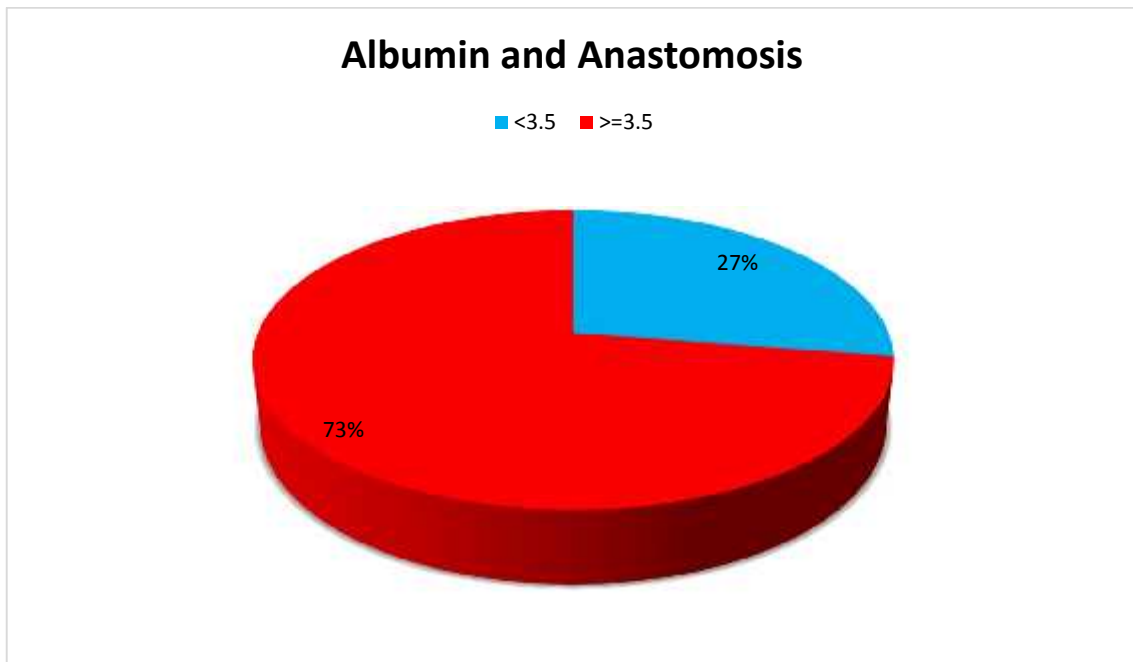
Albumin and Anastomosis

A minimum of 3.5mg/dl of serum albumin is essential for a good healing of anastomosis.

Table:5

	No. of cases (T=50)		Anastomotic leak	
<3.5	11	27.5%	4	36.3%
>=3.5	39	72.5%	2	5.1%

Chart:5



Out of the total 50 patients included in the study, 11 patients suffered hypo proteinemia with a serum albumin level of less than 3.5mg/dl. Post operatively, among the 11 patients with low serum albumin, 4 patients (36.3%) developed anastomotic leaked. Whereas, in patients with normal serum albumin, the percentage of patients who suffered leak was as low as 5%, i.e. only 2 patients out of 39.

This confirms the importance of serum albumin in wound healing, specifically the healing of intestinal anastomosis.

RENAL PARAMETERS

Blood urea and serum Creatinine are two biochemical parameters routinely measured in all pre operative patients. This is to assess the

function of the patient's kidneys. Kidneys play a major role in the elimination of toxic products as well as metabolism of life saving drugs administered to the patient before, during and after the surgery.

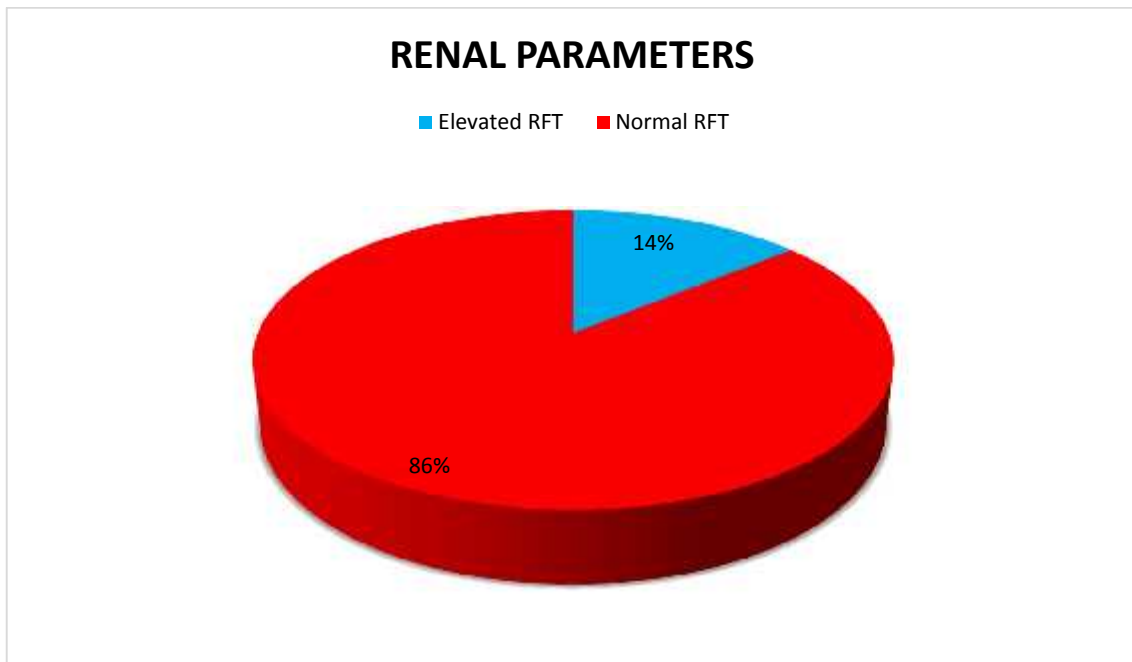
Any elevation in the levels of urea and Creatinine indicates an abnormality in the functioning of the kidneys and there by a disturbance in the milieu interior of human body.

As such any elevation in the renal parameters indicates a sub-optimal functioning of the kidneys. This in turn affects all body processes including wound healing.

Table: 6

	No. of cases (T=50)		Anastomotic leak	
Elevated RFT	7	14%	4	57.14%
Normal RFT	43	86%	2	4.65%

Chart: 6



Among the 50 patients included in the study 7 patients had elevated blood urea and serum Creatinine while 43 patients were in the normal range. Out of the 7 patients with elevated renal parameters, 4 patients developed anastomotic leak, which is about 57.14%. Whereas, among the 43 normal patients, only 2 patients developed anastomotic leak, which accounts of a very minimal 4.6%.

This clearly points towards an elevated renal function test or in other words a poor kidney function being a risk factor for anastomotic leak.

ELEVATED BLOOD SUGAR

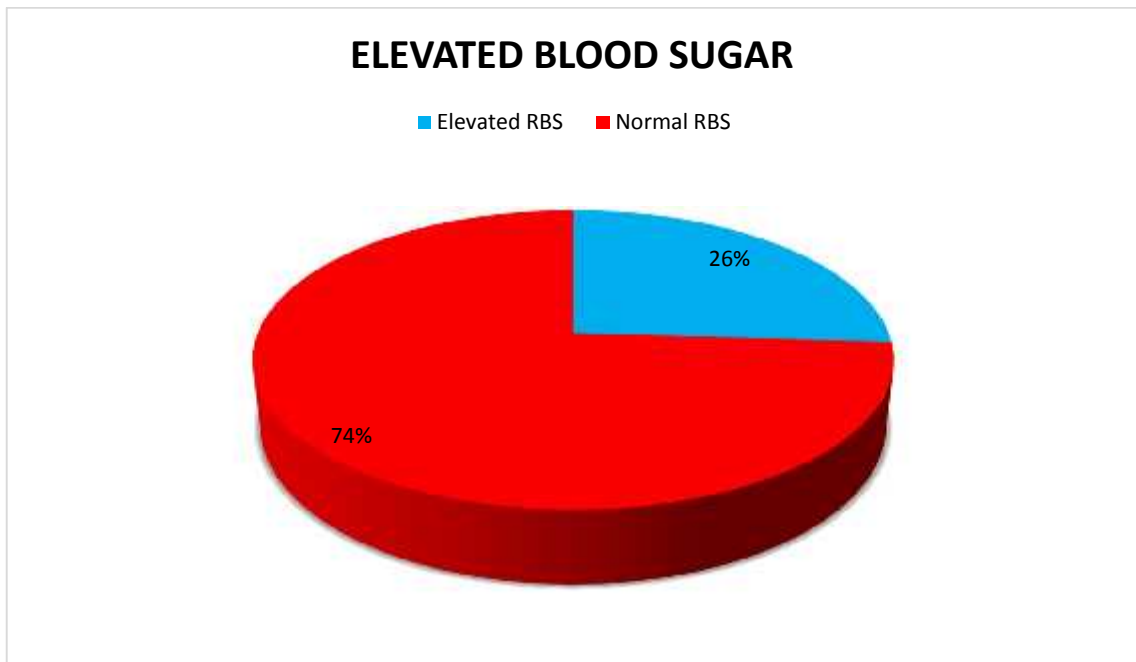
Diabetes as mentioned above leads to a state of poor healing. An elevated blood sugar in biochemical analysis points towards the possibility of hyperglycaemia which later leads on to poor anastomotic healing.

For the purpose of the study, patients were classified into two categories – those with random blood sugar taken prior to surgery less than 140 and those with blood sugar more than 140.

Table:7

	No. of cases (T=50)		Anastomotic leak	
Elevated RBS	13	26%	4	30.7%
Normal RBS	37	74 %	2	5.4%

Chart:7



13 patients, or 26% of the total 50 patients included in the study had an elevated random blood sugar. Among these 4 patients developed anastomotic leak. That is 30.7% of the patients with hyperglycaemia developed anastomotic leak. In contrast, only 2 patients or 5% of the patients with a normal random blood sugar developed anastomotic leak.

This clearly establishes the need for control of blood sugar for a successful anastomotic healing.

INTRA OPERATIVE FACTORS

Intra operative factors can be patient dependent or surgeon dependent or both. Some the factors are modifiable while others like presence of gangrene or malignancy or the aetiology for which the patient is undergoing resection is non-modifiable.

For the ease of analysis, the intra operative factors studied are divided into

- Patient dependent
- Surgeon dependent

Patient Dependent Factors

These include

- Aetiology
- Delay in presentation

Aetiology

The aetiology or the disease process for which the patient is undergoing intestinal resection and anastomosis plays the most crucial role in the outcome.

Aetiology is classified for the purpose of the study into 3 main categories based on the frequency of presentation

They are

- Gangrene
- Malignancy
- Others – trauma, diverticulosis etc

GANGRENE

Gangrene of bowel is one of the major aetiologies resulting in resection and anastomosis. Gangrene occurs as a result of diminished vascular supply to the bowel. It can occur in many ways.

Major pathologies producing bowel gangrene are

- Vascular occlusion
- Injury to supplying vessels

Bowel, both small and large intestine receives its blood supply through its mesentery³. So any occlusion or injury to mesentery and its vessels produce bowel gangrene.

Mesenteric vascular occlusion can occur in two ways. It can be a mechanical occlusion like that of a volvulus or an obstructed hernia or it can be due to an embolic or thrombotic occlusion of the vessels producing mesenteric ischemia.

When there is a mechanical obstruction, first a stage of venous congestion occurs in the bowel. This leads on to accumulation of inflammatory fluids in the bowel wall, which aggravates the congestion and further diminishes blood supply. Then the stage of gangrene sets in. This leads on to peritonitis and its sequelae.

Mesenteric vascular ischemia occurs as a result of occlusion of the mesenteric vessels by an embolus or a thrombus. Superior mesenteric artery is most commonly affected than inferior. This can also occur in a non occlusive fashion, as a result of hypotension or hypo perfusion or due to vasospasm due to shock - Non occlusive mesenteric ischemia (NOMI).

Gangrene of the bowel requires immediate intervention in the form of emergency exploratory laparotomy and resection of the gangrenous bowel. Viability of the cut ends should be ensured before anastomosis. Fresh bleeding from the cut end mucosa indicates viability. If the ends don't bleed or the mucosa is dark red, viability is doubtful. In such cases, the ends should be further trimmed until vascularity is ensured. Once vascularity of the ends is ensured, we can proceed on to anastomosis. Mesenteric window created during resection should be closed to avoid internal herniation.

MALIGNANCY

Tumours of intestine are another pathology requiring resection and anastomosis of bowel. Benign tumours require a limited resection while malignant ones require resection of the entire length of bowel supplied by the particular vessel supplying the segment with tumour along with removal of the corresponding lymph node stations as well².

SMALL BOWEL TUMOURS

Tumours of small bowel are rare. They constitute about 3% of all GI malignancies even though small bowel constitutes 80% of the total length of the Git and 90 % of the total mucosal surface area.

Early diagnosis is difficult as they are very vague in presentation.

Benign tumours of small bowel includes

- Adenoma
- Leiomyoma
- Lipoma
- Haemangioma
- Polyps

These tumours usually present with vague symptoms like colicky abdominal pain, haemorrhage etc. Commonly they are an on table diagnosis

Treatment usually involves resection and anastomosis

Malignant tumours of the small bowel includes

- Adeno carcinoma
- Lymphoma
- Carcinoid
- Liposarcoma
- Secondaries in the small bowel

Treatment includes

- For duodenal tumours pancreatico duodenectomy
- For ileal/jejunal tumours radical resection with 10cm margin along with mesenteric clearance
- Adeno carcinoma of terminal ileum requires right hemicolectomy.

LARGE BOWEL TUMOURS

Large bowel tumours are more common than small bowel tumours. They can be benign or malignant. Benign tumours include different types of polyps, adenoma etc. Malignant tumours are found to arise from different part of the colon like caecum, ascending colon, transverse colon, descending colon, sigmoid colon. Treatment varies according to the location of the tumour.

Caecum and ascending colon – Right hemicolectomy¹

Hepatic flexure – extended right hemicolectomy

Transverse colon – transverse colon with both flexures

Descending colon – left hemicolectomy

OTHERS

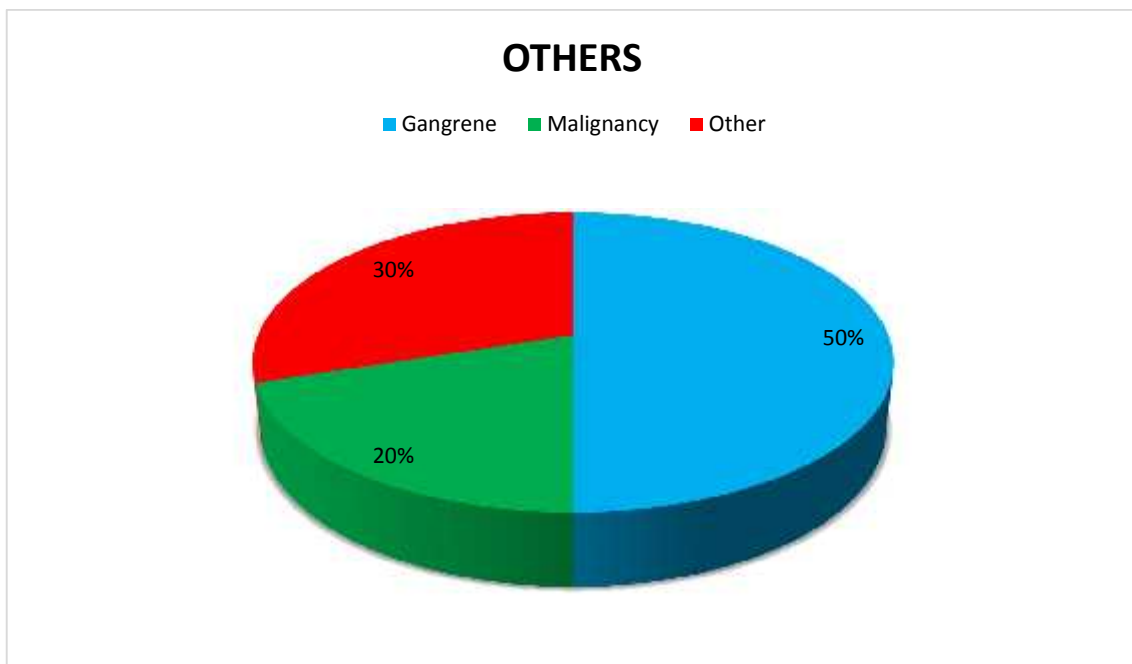
Other pathological conditions requiring intestinal resection and anastomosis include

- Multiple perforations
- Large perforations
- Mesenteric tears compromising vascularity
- Diverticulitis

Table:8

	No of cases (T=50)		Anastomotic leak	
Gangrene	25	50%	5	20%
Malignancy	10	20%	0	0%
Other	15	30%	1	6.7%

Chart:8



50% of the patients included in the study underwent intestinal resection and anastomosis for bowel gangrene commonly as a result of obstructed hernias and other intestinal obstructions.

20% or 10 out of 50 patients underwent resection and anastomosis as a part of treatment for malignancies.

30% or 15 patients underwent resection for miscellaneous conditions like multiple perforations, large perforations, mesenteric tears etc.

On comparing the numbers of anastomotic leak, maximum number of anastomotic leak (5 in number) was encountered in patients who underwent resection for bowel gangrene. This number amounts to a leak rate of a huge 20% among the patients with bowel gangrene.

None of the patients treated for malignancy developed anastomotic leak.

A single patient treated of a miscellaneous aetiology also developed anastomotic leak.

This clearly points out the high risk of developing anastomotic leak in case of patients with bowel gangrene.

All safety precautions like adequate vascularity of the cut ends, adequate level of serum proteins, post operative care should be maintained for a successful outcome.

ADMISSION TO INCISION DELAY

This is more important in case of emergencies like gangrene bowel, mesenteric ischemia, traumatic bowel and mesenteric injuries etc.

The delay occurs in two fronts

- Delayed presentation of the patient
- Delay in operating

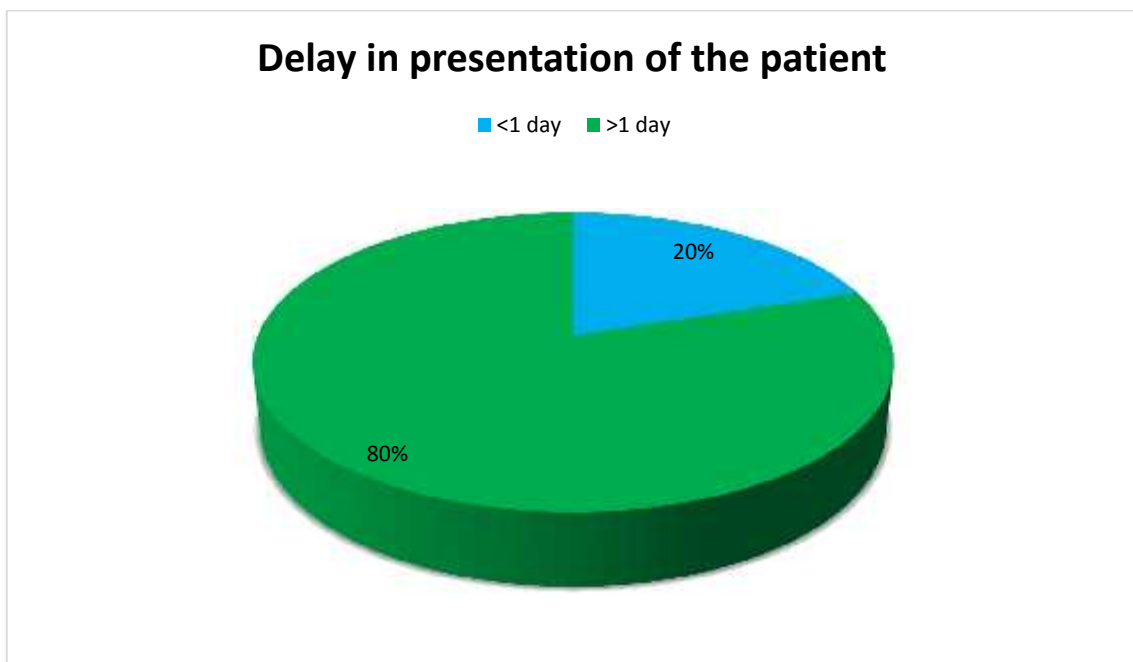
Delay in presentation of the patient

In the study, delay is studied in the form of duration of symptoms before presentation.

Table:9

	No. of cases (T=50)		Anastomotic leak	
<1 day	10	20%	2	33.3%
>1 day	40	80%	4	66.7%

Chart:9



Among the total 50 patients followed, 80% or 40 patients presented with more than 1 day duration of symptoms, whereas 10 patients presented within 1 day of onset of symptoms.

Out of the total 50 patients, 6 patients developed anastomotic leak. Among these 6 patients, 4 patients presented with more than 1 day delay accounting for 66.7% of the total.

Thus it is clear that a delay in presentation influences the outcome of resection and anastomosis.

Delay in operating

Delay in operating is also more important in case of emergency cases.

1 case of small bowel volvulus which was delayed in operating developed anastomotic leak.

ANASTOMOSIS

This is the most important surgeon related factor which influence the outcome

Anastomosis can be studied under various classes.

- Based on bowel involved
- Based on orientation of bowel
- Based on number of layers in which the anastomosis is done

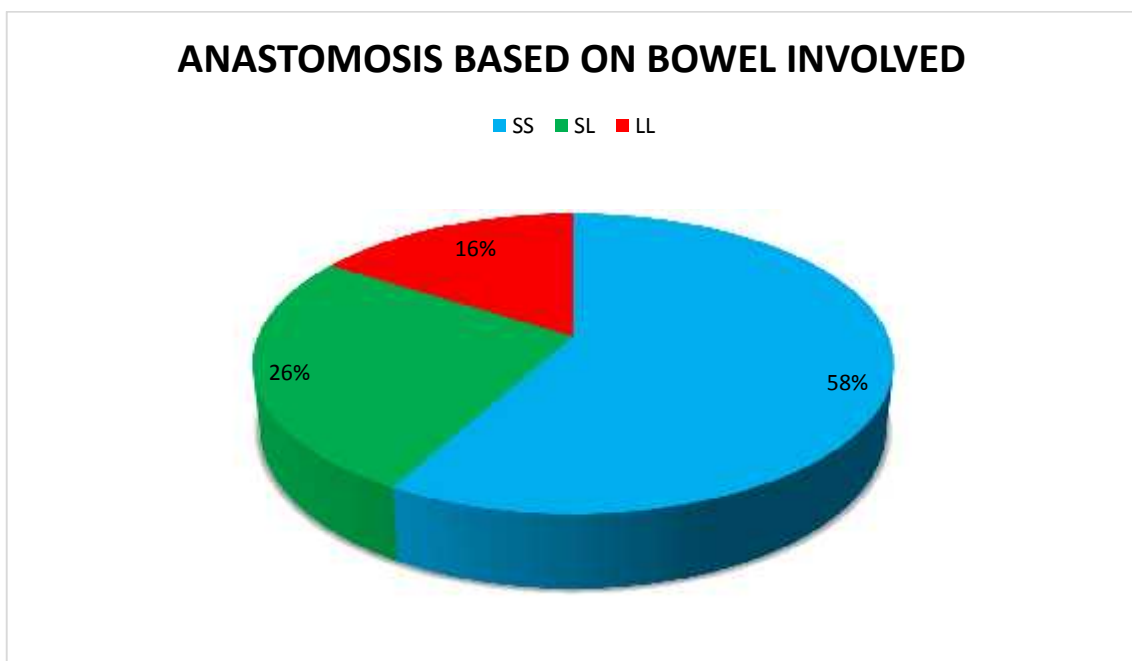
Based on bowel involved

Based on the bowel involved, the anastomosis can be between two small bowel segments, two large bowel segments or between a small bowel and a large bowel as in Ileo – transverse colic anastomosis.

Table:10

	No of cases (T=50)		Anastomotic leak	
SS	29	58%	1	3.44%
SL	13	26%	5	38.4%
LL	8	16%	0	0%

Chart:10



Out of the total 50 patients, 29 patients (58%) underwent a small bowel to small bowel anastomosis. 13 patients or 26% underwent a small to large bowel anastomosis. 9 patients underwent anastomosis between two large bowel segments.

Out of the 6 patients who developed anastomotic leak, 5 patients had undergone anastomosis between a small bowel loop and a large bowel loop, producing a leak rate of 38.4% and a single patient had undergone a small bowel to small bowel anastomosis, with leak rate of only 3.4%.

Difference in type of the bowel loops and disparity in lumen size appears to have influenced the result.

While anastomosing a small bowel to a large bowel, utmost care has to be taken, especially at the anti mesenteric ends.

Based on orientation of the bowel loops

While anastomosing two bowel loops, they may be oriented in different ways.

They may be oriented such that the two ends face each other and an end – to- end anastomosis can be done. Sometimes the antimesenteric ends of the two bowel loops are apposed and a side to side anastomosis is done. When the end of one loop is apposed to side of another, we perform an end – to – side anastomosis.

An end to end anastomosis is done between two ends of small bowel or two ends of large bowel. Whereas, when we anastomose a small bowel to large bowel, we usually anastomose the end of the small bowel to the side of the large bowel. This is because of the size disparity between the ends of small and large bowel.

While anastomosing the ends of small bowel or ends of large bowel, if there is size disparity, a cut can be given on the antimesenteric border of the smaller end and then anastomosis can be done. This is called as a **CHEATLE's cut**.

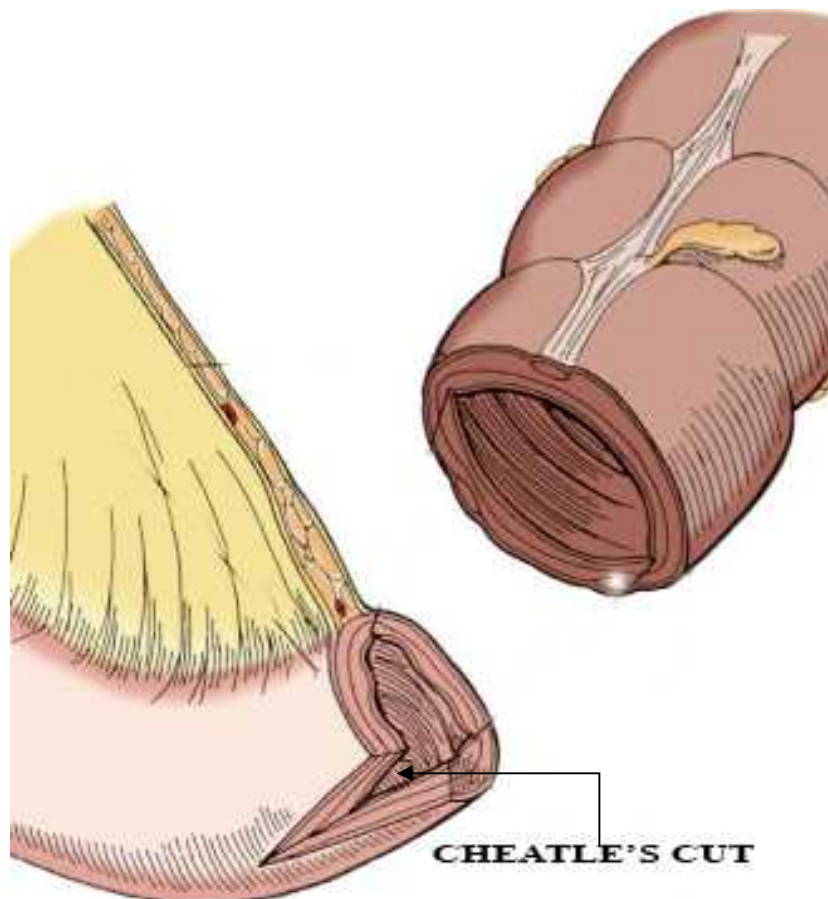
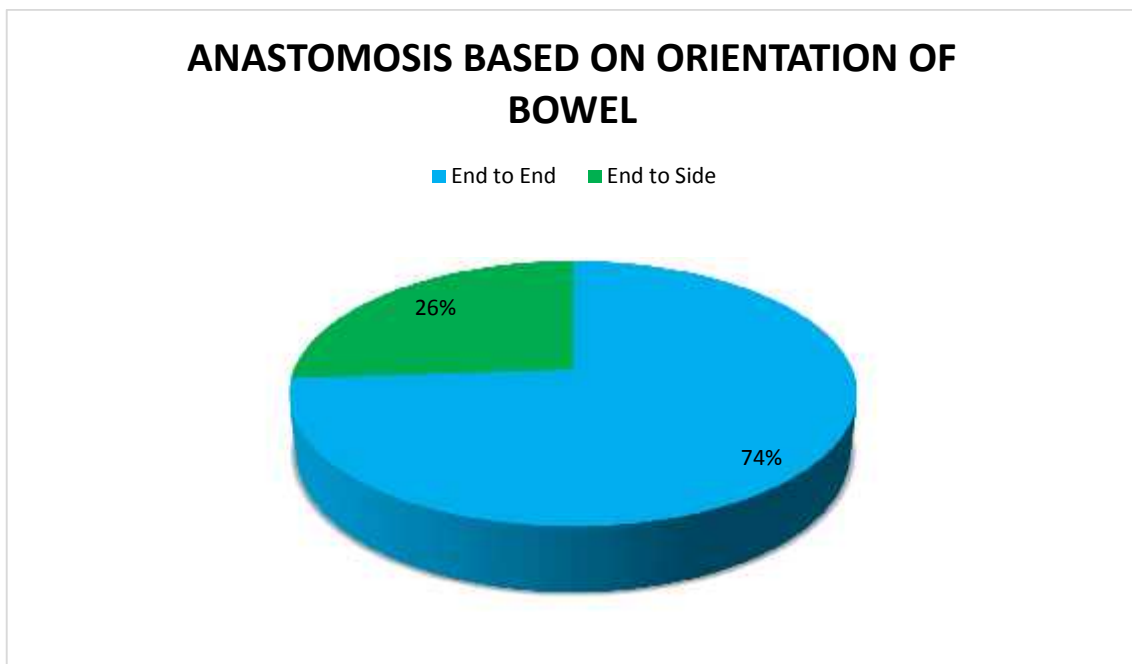


FIGURE: 10

Table:11

		No of cases (T=50)		Anastomotic leak	
End to	End	37	74%	1	2.7%
End to	Side	13	26%	5	38.5%

Chart:11



None of the cases included in the study underwent a side to side anastomosis.

74% of the patients (37 patients) underwent an end to end anastomosis, while 13 underwent end to side anastomosis. Out of the 6 patients who developed leak, 5 patients had undergone end to side anastomosis. This amounts to a leak rate of 38.5% among the patients who underwent end to side anastomosis. On the other hand only 1 patient who underwent an end to end anastomosis developed anastomotic leak, accounting for 2.7% of the total.

This result may be attributed to the risk while suturing the two corner points of the anastomosis. This can be overcome to a great degree

by starting of the middle on one size and ending at the middle on the opposite side rather than starting and ending at the corners.

POST OPERATIVE FACTORS

The important post operative factors studied were transfusion of blood, blood products and TPN (Total Parenteral Nutrition).

BLOOD

Transfusion of blood is a common procedure in all major surgeries that have significant blood loss. This corresponds to the saying “Blood should be replaced by blood”.

Main aims of blood transfusion in relation to bowel surgeries are

- Correction of pre existing anaemia
- Correction of blood loss
- To raise blood pressure

Blood is preserved with anticoagulants and kept at low temperatures. The most common anticoagulant used is Citrate phosphate dextrose adenine solution (CPDA). This preserves blood for 42 days. It is stored at a temperature of 2-35 degree Celsius.

Transfusion of blood in a patient has both merits and de merits.

Blood transfusion produces immediate expansion of vascular compartment, provides immediate nourishment and oxygen.

Demerits include ABO incompatibility, Rh incompatibility, allergic reactions, acute haemolysis, blood borne infections, volume overload etc. With respect to wound healing, the most important complication is the decrease in IL-2 levels. Interleukin-2 is an essential factor for wound healing. It is the factor which determines the tensile strength of collagen that gets deposited during wound healing. Massive blood transfusion decreases the levels of IL-2. As a result the tensile strength of the collagen that gets deposited at the anastomotic site gets reduced. This can predispose to failure of anastomosis and anastomotic leak.

On the other hand blood that is transfused provides oxygen and nutrients which are essential for wound healing. Anaemia adversely affects healing of the anastomosis by producing a state of hypoxia at the local site. Transfusion of fresh blood helps in overcoming this. Also transfusion of fresh blood provides glucose to the site of healing.

So the merits and demerits of transfusing blood for a patient undergoing intestinal resection and anastomosis has to be weighed against each other and decided upon

FRESH FROZEN PLASMA

Fresh frozen plasma or FFP is a blood component prepared by centrifugation of whole blood. It is rich in plasma proteins and coagulation factors. FFP should be stored at a temperature of -4 degree Celsius. It can be stored at this temperature for period of 14-21 days.

The main uses of fresh frozen plasma are

- Correction of hypo proteinemia
- Treatment of bleeding disorders

Hypo proteinemia is one of the major risk factors for anastomotic leak. Correction of hypo proteinemia is there for critical in successful healing of anastomosis.

TOTAL PARENTERAL NUTRITION

Intestinal resection and anastomosis is a major surgery producing a lot of stress to the patient's body. Nutrition is hence of utmost importance to overcome the stress of the surgery. Early nutrition goes a long way in healing and early recovery of the patient. This can be in the form of enteral or parenteral feeding.

Total parenteral nutrition is a method of feeding which bypasses the gastrointestinal tract. It supplies all the daily nutritional requirements. It can be given to the patient at home or at the hospital.

It should not be given to patient with an intact gastrointestinal tract for long duration.

Compared to enteral nutrition it has the following disadvantages:

- It has more complications
- Does not preserve GIT structure and function
- Expensive

Nutritional content

TPN contains water (30 to 40 ml/kg/day), energy (30 to 45 kcal/kg/day, depending on energy expenditure), amino acids (1.0 to 2.0 g/kg/day, depending on the degree of catabolism), essential fatty acids, vitamins, and minerals.

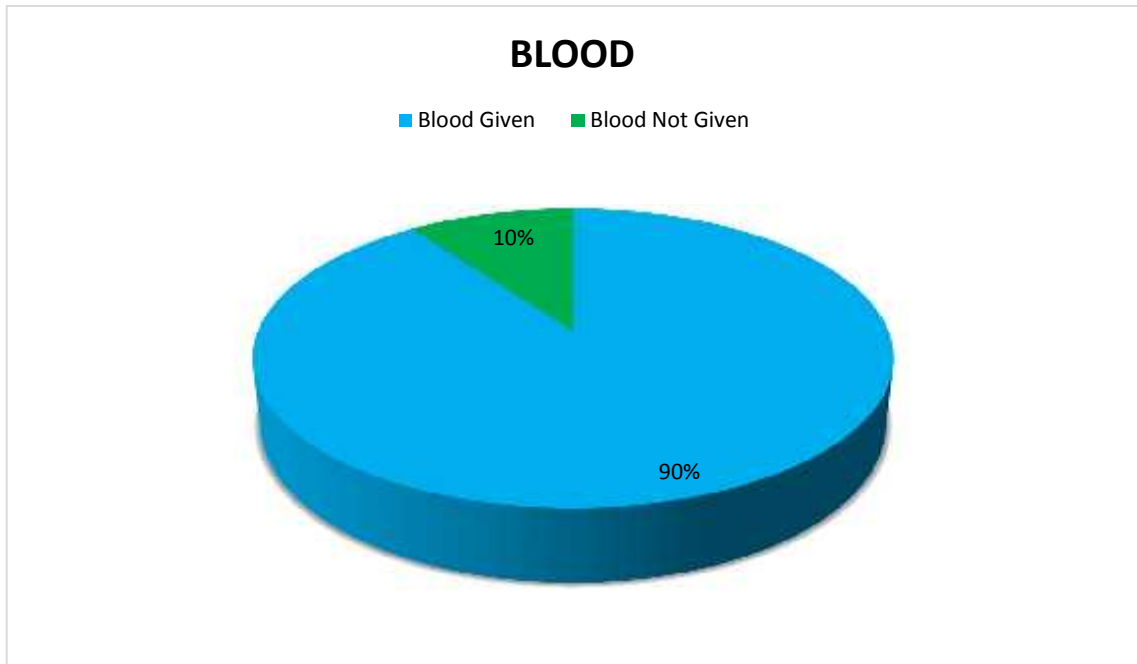
Monitoring is an essential part of administering TPN. Progress should be followed on a flowchart. An interdisciplinary nutrition team, if available, should monitor patients. Weight, CBC, electrolytes, and BUN should be monitored. Plasma glucose should be monitored every 6 hours until patient's glucose levels become stable. Fluid intake and output should be monitored continuously.

BLOOD

Table:12

	No of cases (T=50)		Anastomotic leak	
Blood Given	45	90%	6	13.3%
Blood Not Given	5	10%	0	0%

Chart:12

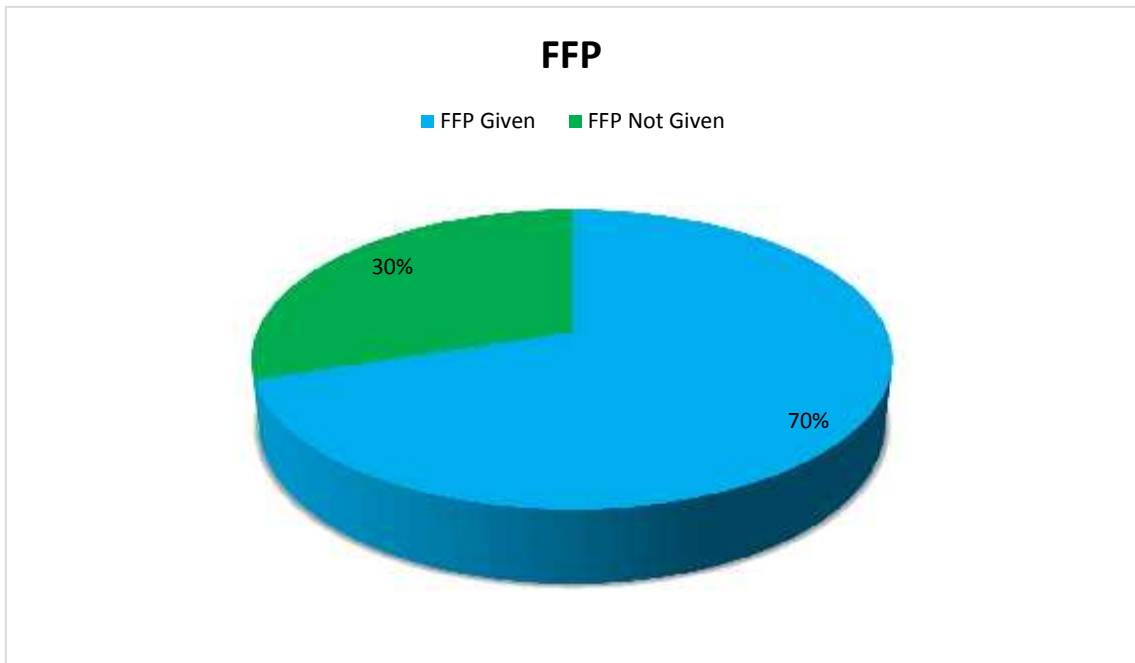


FFP

Table:13

	No of cases (T=50)		Anastomotic leak	
FFP Given	38	70%	5	13.2%
FFP Not Given	12	30%	1	8.3%

Chart: 13



TPN

Table: 14

	No of cases		Anastomotic leak	
	No of cases	Percentage	No of cases	Percentage
TPN Given	25	50%	1	4%
TPN Not Given	25	50%	5	20%

Chart:14



Both blood and FFP were given to almost all patients who underwent resection and anastomosis. Whereas,TPN was transfused to only 50% of the patients.

All the patients who developed anastomotic leak had received blood transfusion and 5 out of the 6 patients had received transfusion of FFP. Commenting on whether transfusion of blood and blood products act as risk factors for anastomotic leak requires a bigger study group and a tightly controlled case and control groups.

On the other hand interestingly, only 1 patient among the total 25 patients receiving TPN transfusion developed anastomotic leak as compared to 5 patients among those who did not receive TPN.

Effective management of post operative nutritional status goes a long way in healing of anastomotic site and an early recovery of the patient.

OTHERS

Presence of a protective stoma – only 2 cases in the study had protective stomas made. None of them suffered anastomotic leak.

Hand sewn vs. Stapler – Since all the cases underwent anastomosis by hand sewn technique, which among the two is superior cannot be pointed out.

2 layers vs. single layer – in our institution all intestinal anastomosis both small and large bowel are done in two layers. So commenting on this is also beyond the scope of my study

CONCLUSIONS

1. Age adversely affects anastomotic healing. Elderly patients are at a higher risk for anastomotic leak.
2. Female sex appears to be better protected against anastomotic leak.
3. Impaired vascularity or gangrene is the predominant risk factor for anastomotic leak.
4. Anaemia, elevated renal parameters, elevated blood sugar and low serum albumin predisposes to anastomotic leak.
5. End to side anastomosis and anastomosis between small bowel and large bowel has a higher risk for anastomotic leak.
6. Transfusion of TPN appears to be protective and helps in better healing of the anastomosis.

LIMITATIONS OF THE STUDY

1. A study group of 50 patients is not at all sufficient to accurately comment on a multifactorial outcome like the successful healing of the anastomosis.
2. Apart from all the factors discussed above, experience of the operating surgeon in handling the bowel and creating an anastomosis influences the outcome of an anastomosis. This is something that cannot be quantitatively assessed.
3. Ability of human body to heal itself and the patient's genetic makeup plays a significant role in the outcome.

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S. NO.	NAME	AGE	SEX	IP No.	DOS	DM	CAD	RENAL DS	Hb	RBS	BU	S.Cr	S.Alb	Etiology	TOA	Bowel	Blood	FFP	TPN	LEAK
1	Palaniammal	48	F	6959	>1	-	-	-	12	120	34	0.6	3.8	GANGRENE	EE	SS	N	Y	N	-
2	shahunthala	33	F	19740	>1	-	-	-	8.5	116	26	0.8	3.5	GANGRENE	EE	SS	Y	Y	N	-
3	bagavathy	58	F	20636	>1	+	-	-	9.2	261	46	2.7	3.5	GANGRENE	EE	SS	Y	Y	Y	-
4	duraipalam	72	M	31496	>1	-	-	-	11	68	222	5.9	2.4	GANGRENE	ES	SL	Y	N	N	+
5	seetharam	50	M	31196	1	-	-	-	10.6	102	39	1.1	4	GANGRENE	EE	SS	N	Y	N	-
6	krishnan	63	M	38042	>1	-	+	-	15	142	18	1.2	3.9	GANGRENE	EE	SS	N	N	N	-
7	senthil	32	M	36622	>1	-	-	-	16	223	55	1.8	3	GANGRENE	ES	SL	Y	Y	N	+
8	velu	48	M	39360	1	-	-	-	8	332	32	1.3	3.5	OTHER	EE	SS	Y	Y	N	-
9	mahesh	35	M	41898	>1	-	-	-	13.4	155	39	0.7	4.1	GANGRENE	EE	SS	Y	N	N	-
10	kanagalakshmi	45	F	41735	>1	-	-	-	11.4	83	23	0.8	4	OTHER	EE	SS	Y	Y	Y	-
11	vella pandi	59	M	43686	1	-	-	-	14	144	20	0.9	3.8	OTHER	EE	SS	Y	Y	N	-
12	jeiyabal	62	M	42898	>1	+	-	-	11.2	299	52	1.4	3.7	MALIGNANCY	EE	LL	Y	Y	N	-
13	vignesh	27	M	53743	1	-	-	-	12.4	126	26	0.8	4.2	GANGRENE	EE	LL	Y	Y	N	-
14	pitchayya	45	M	52621	>1	-	-	-	19	157	62	2.2	3.4	GANGRENE	EE	SS	Y	N	N	-
15	kottursamy	70	M	54845	>1	-	+	-	13.9	72	30	1	4	GANGRENE	EE	SS	N	Y	N	-
16	ganeshan	72	M	56277	>1	-	+	+	6.6	103	81	2.6	2.6	OTHER	ES	SL	N	N	N	+
17	sakthivel	32	M	8181	>1	-	-	-	13.8	100	25	1.4	4	OTHER	EE	SS	N	N	N	-
18	syed meeran	16	M	1632	>1	-	-	-	13.2	112	16	0.9	4	OTHER	EE	SS	N	Y	N	-
19	thangaraj	30	M	2810	1	-	-	-	14	106	18	0.6	4.1	OTHER	EE	SS	N	Y	N	-
20	karthick	23	M	75576	>1	-	-	-	17.6	220	19	1.3	3.6	GANGRENE	EE	SS	Y	Y	N	+
21	masilamani	65	M	80900	>1	-	-	-	13	340	51	2.5	3.6	GANGRENE	ES	SL	N	Y	Y	+
22	balamurugan	18	M	6084	>1	-	-	-	10.8	123	21	0.6	3	OTHER	EE	SS	N	Y	Y	-
23	vigneswaran	36	M	9387	1	-	-	-	10.4	132	23	2	3.8	GANGRENE	ES	SL	Y	N	N	-
24	alagumuthu	59	M	7876	>1	-	-	-	7.5	132	16	0.9	3	GANGRENE	ES	SL	Y	N	N	+
25	subash	48	M	12411	>1	-	-	-	10.8	63	72	1.3	3	OTHER	ES	SL	N	N	Y	-
26	chellappa	45	M	15616	1	-	-	-	12.6	151	54	0.8	3.2	OTHER	EE	SS	Y	N	N	-
27	samy	58	M	11605	>1	-	-	-	10.1	97	16	0.8	3.5	MALIGNANCY	EE	LL	Y	Y	Y	-
28	esthar	65	F	17205	>1	-	-	-	11.5	138	23	0.8	3.6	OTHER	EE	SS	N	Y	Y	-
29	syed mohadeen	48	M	18275	>1	-	-	-	15.6	183	49	1.3	3.6	MALIGNANCY	EE	SS	N	N	N	-
30	natchiammal	50	F	27551	>1	-	-	-	10.1	209	42	1.3	3.1	GANGRENE	EE	SS	N	Y	Y	-

S. NO.	NAME	AGE	SEX	IP No.	DOS	DM	CAD	RENAL DS	Hb	RBS	BU	S.Cr	S.Alb	Etiology	TOA	Bowel	Blood	FFP	TPN	LEAK
31	parvathy	55	F	30509	>1	-	-	-	7.3	89	21	1	3.7	MALIGNANCY	ES	SL	Y	Y	N	-
32	ramasamy	59	M	26536	>1	-	-	-	12.9	112	36	1.4	3.5	MALIGNANCY	ES	SL	N	Y	Y	-
33	thangamani	38	F	29357	>1	-	-	-	12.2	93	18	0.9	4	OTHER	EE	SS	N	Y	Y	-
34	veluappan	59	M	32628	>1	-	-	-	10.9	89	23	1	3.5	GANGRENE	EE	SS	N	Y	Y	-
35	santharuby	49	F	33684	1	-	-	-	9.7	98	20	0.6	2.5	OTHER	ES	SL	N	Y	Y	-
36	arunachalam	64	M	33626	>1	-	-	-	8.5	114	16	1.2	4.1	MALIGNANCY	ES	SL	Y	Y	Y	-
37	thirumalai vadiv	59	F	41989	>1	-	-	-	10.7	120	32	0.9	4	MALIGNANCY	EE	LL	Y	N	Y	-
38	kavitha	25	F	39302	>1	-	-	-	10	99	35	0.7	4	GANGRENE	EE	LL	N	Y	Y	-
39	subbaiah	69	M	41400	>1	-	-	-	11	136	30	1.1	2.9	GANGRENE	EE	LL	N	Y	Y	-
40	anthonyraj	26	M	42586	>1	-	-	-	11.4	126	29	0.6	3.6	OTHER	ES	SL	N	Y	Y	-
41	mariappan	39	M	49022	>1	-	-	-	12.6	130	30	0.8	3.7	GANGRENE	EE	SS	N	Y	Y	-
42	chellaiah	59	M	47555	>1	-	-	-	13.4	136	40	1.1	3.6	GANGRENE	EE	LL	N	Y	N	-
43	santhakumar	49	M	50599	>1	-	-	-	11.6	121	39	1	3.7	MALIGNANCY	EE	LL	N	Y	Y	-
44	selvam	50	M	53567	>1	-	-	-	11	134	38	1.1	3.6	GANGRENE	EE	SS	N	Y	Y	-
45	ramkumar	26	M	49963	>1	-	-	-	14	152	26	0.8	3.8	OTHER	EE	SS	N	Y	N	-
46	sankar	39	M	55473	>1	-	-	-	12.9	112	32	0.7	4	OTHER	EE	SS	N	Y	Y	-
47	kumar	58	M	50909	>1	-	-	-	11	109	40	1.1	3.5	GANGRENE	EE	SS	N	Y	Y	-
48	liyakath ali	53	M	60349	>1	-	-	-	12	138	39	0.9	3.6	MALIGNANCY	ES	SL	N	Y	Y	-
49	marimuthu	39	M	59953	1	-	-	-	14.1	152	26	0.9	3.7	OTHER	EE	SS	N	Y	Y	-
50	manikandan	26	M	59032	1	-	-	-	13.3	129	28	0.7	3.6	OTHER	EE	SS	N	Y	Y	-

PROFORMA

Name -

Age/Sex -

IP no. -

Date of Admission -

Presenting symptoms -

Duration of presenting symptoms -

Co morbidities – 1. Diabetes mellitus

2. CAD

3. Renal disease

4. Others

Diagnosis -

Investigations – 1. Hb

2. RBS

3. RFT

4. Serum albumin

Procedure done –

Anastomosis details – 1. Type of anastomosis – type of bowel anastomosed

2. Bowel orientation

Transfusions – Blood, FFP, TPN

Anastomotic leak – Y/N

**நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)**

ஆய்வு செய்யப்படும் தலைப்பு:

பங்கு பெறுவரின் பெயர்:

பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	<input type="checkbox"/>
	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	<input type="checkbox"/>
	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	<input type="checkbox"/>
	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	<input type="checkbox"/>
	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.	<input type="checkbox"/>

பங்கேற்பவரின் கையொப்பம் / இடம்

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம் / இடம்

ஆய்வாளரின் பெயர்

மையம்

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சாட்சியின் கையொப்பம் / இடம்

பெயர் மற்றும் விலாசம்

ABBREVIATIONS

1. IP no. – In Patient number
2. DM – Diabetes mellitus
3. CAD – Coronary Artery Disease
4. CKD – Chronic kidney disease
5. Hb – haemoglobin
6. RBS – Random blood sugar
7. RFT – Renal function test
8. BU – Blood urea
9. S.Cr – Serum Creatinine
- 10.S.Alb – Serum albumin
- 11.EE – End to End
- 12.ES – End to Side
- 13.SS – Small bowel to small bowel
- 14.LL – Large bowel to large bowel
- 15.SL – Small bowel to large bowel
- 16.FFP – Fresh Frozen Plasma
- 17.TPN – Total Parenteral Nutrition
- 18.GIT – Gastro Intestinal Tract