"A PROSPECTIVE COMPARATIVE STUDY OF EARLY VERSUS LATE ENTERAL FEEDING AFTER MAJOR GASTROINTESTINAL SURGERIES "

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the regulations for the award of the degree of

M.S. GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

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REGISTER NUMBER : 221711318

CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.S.YUVARAJ,** and submitted in partial fulfilment of the requirement for the Degree of M.S. General Surgery, Branch I of the Tamilnadu Dr. M.G.R. Medical University, Chennai.

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DATE: **PROFESSOR & HOD**

DEPARTMENT OF GENERAL SURGERY

DATE: **DEAN**

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COIMBATORE – 641014

INSTITUTIONAL HUMAN ETHICS COMMITTEE
COIMBATORE MEDICAL COLLEGE, COIMBATOR – 14

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CERTIFICATE OF APPROVAL

To
Dr.Yuvaraj S
Post Graduate,
Department of General Surgery,
Coimbatore Medical College & Hospital
Coimbatore -18.

Dear **Dr.Yuvaraj S**

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled **"A Prospective Comparative Study of Early Versus Late Enteral feeding after gastrointestinal surgeries."**No.0117/2017.

The following members of Ethics Committee were present in the meeting held on 30.11.2017.conducted at MM - II Seminar Hall, Coimbatore Medical College Hospital Coimbatore-18

1	Dr.S.Ramalingam MD, Dean, PSG IMS&R, Cbe	Chairman
2	Dr.Usha MD., Professor of General Medicine, CMCH, Cbe	Member Secretary
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4	Dr.N.Renganathan MS., Professor of General Surgery, CMCH,Cbe	Clinicians
5	Dr.Sudha Ramalingam MD., Professor of SPM, PSG IMS&R, Cbe	Clinicians
6	Dr.R. Shanmugavadivu MD., Professor of Physiology, CMC, Cbe	Basic Medical Scientist
7	Dr.N. Shanthi MD., Professor of Pharmacology, CMC, Cbe	Basic Medical Scientist
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9	Dr.L.Madhan MD., Professor of Pharmacology, CMC, Cbe	Basic Medical Scientist
10	Dr.N.Paramasivan MD., Professor of Pharmacology, Sri Ramakrishna Dental College, Coimbatore	Basic Medical Scientist
11	Mrs.A.Sharmila BA., BL., Advocate	Legal Expert
12	Dr.K.P.Sampath Kumar M.Pharm, Ph.D., Asst. Prof. of Pharmacy, CMC, Cbe	Scientific Member
13	Dr.G.Vani Ganesh M.Sc.,Ph.D., Tutor in Medical Surgical Nursing, CMCH, Cbe	Scientific Member
14	Mr.V. Balasubramani MA,MA,MBA,LLB,M.Phil,PG.D.M, DLLAL, Chief Executive, Avinashilingam JSS Self Finance Courses, Cbe	Social Worker
15	Mr.V.A.Shahul Hameed, +2	Lay-Person

We approve the Proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

MEMBER SECRETARY
Member Secretary, Ethics Committee
INSTITUTIONAL HUMAN ETHICS COMMITTE
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14

DECLARATION

I solemnly declare that the dissertation titled **"A PROSPECTIVE COMPARATIVE STUDY OF EARLY VERSUS LATE ENTERAL FEEDING AFTER MAJOR GASTROINTESTINAL SURGERIES"** was done by me from 2018 onwards under the guidance and supervision of **Prof. DR. A.NIRMALA M.S., D.G.O.,**

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfilment of the requirement for the award of M.S Degree in General Surgery (Branch I).

PLACE: **DR. YUVARAJ.S**

DATE: **REGISTER NO: 221711318**

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I am thankful to **The ETHICAL COMMITTEE** of Coimbatore Medical College for permitting me to proceed with this dissertation.

Lastly I am grateful to all the patients whose cooperation made this work possible.

DATE: **SIGNATURE OF THE CANDIDATE**

PLACE: **DR. YUVARAJ.S**

ABSTRACT

BACKGROUND

As per routine protocol, patients undergoing major gastrointestinal surgeries are kept nil by mouth till intestine starts functioning. But evidence from clinical studies suggests that initiating enteral feeding early is beneficial and advantageous to patient and reverses mucosal atrophy induced by starvation and also increases collagen deposition at the surgical sites, promoting wound healing. Finally, early enteral feeding may also reduce septic morbidity and improves postoperative outcome.

OBJECTIVES:

This study was conducted to compare the feasibility, safety, efficacy, tolerability and outcome of early enteral feeding versus late oral feeding after elective and emergency gastrointestinal surgeries.

MATERIALS AND METHODS:

The prospective comparative study included 50 cases of major gastrointestinal surgeries operated at Coimbatore Medical College Hospital from January 2018 to June 2019, from which randomly 25 patients were selected for conventional nil by mouth approach and rest of 25 patients were given early enteral feeding within 1st 24 hours post operatively.

RESULTS:

In the study most common age group, who undergone surgery in both case and control groups was between 30-60 years with male predominance in both groups. Most common surgery performed was perforation closure in both groups. The study group had statistically significant low rate of wound infection, wound dehiscence, paralytic ileus, leak, GI Complications and post operative pain with less hospital stay.

CONCLUSION:

Compare to conventional nil by mouth approach, early enteral feeding significantly reduces the incidence of wound infection, paralytic ileus and pain in post operative patients undergoing major gastrointestinal surgeries, thereby reducing length of hospital stay, which suggest that early enteral feeding is safe, effective and feasible in post operative patients of major gastrointestinal surgeries.

Key words: Enteral feeding, Intestines, Paralytic Ileus, Postoperative Pain, Surgical Anastomosis, Wound Infection, Wound dehiscence.

TABLE OF CONTENTS

INTRODUCTION (1,2,3,4)

A period of starvation (nil orally) is a common practice after most gastrointestinal surgeries. Postoperative dysmotility predominantly affects the stomach and colon, with the small bowel recovering normal function 4-8 hours after laparotomy. Malnutrition predisposes to postoperative complications: increased incidence of infection and prolonged hospital stay. The rationale of ―nil orally‖ is to prevent postoperative nausea and vomiting and to protect the anastomosis repair, allowing time to heal before being stressed by food. But this worsens nutritional state of the patients. Adynamic ileus after abdominal surgery is characterized by absent motility caused byneuromuscular inhibition with sympathetic over activity. It occurs after all abdominal procedures, with motility returning typically in the small bowel within 24 hours, in the stomach within 48 hours, and in the colon within 3 to 5 days. Because of this, patients have been traditionally treated after surgery with nasogastric decompression until evidence that bowel function has returned, after which a liquid diet is initiated and advanced to a regular diet over 4-5 days. Gastrointestinal surgery is associated with postoperative morbidity such as wound infection, leak, intra-abdominal sepsis and other extra intestinal complications. Malnutrition increases post-operative morbidity and rates, and duration and cost of hospital stay4. Delayed feeding has been practiced for fear of physical stress disrupting the anastomosis but GIT secretions present at the anastomotic site with a volume load of approximately 6-8 Liters per day irrespective of delayed or early feeding.

Nausea and vomiting, however, occur more commonly after upper gastrointestinal surgery than resection of thesmall intestine and colon. There is no evidence that bowel rest and a period of starvation are beneficial for healing of wounds and anatomic integrity.

Indeed, the evidence is that luminal nutrition may enhance wound healing and increase anastomotic and perforation repair strength and with better rate of healing, particularly in malnourished patients. Pre-existing nutritional depletion, however, may not only be the nutritional factor associated with postoperative complications after gastrointestinal surgery. There is thus no evidence that early post-operative enteral feeding should be restricted to malnourished patients undergoing gastrointestinal surgery.

JUSTIFICATION FOR THE STUDY:

Routinely patients undergoing major Gastrointestinal surgeries (Elective and Emergency) are kept nil by mouth for various reasons. Idea behind nil by mouth is to prevent post-operative nausea and vomiting. Post-operative dysmotility mainly affects stomach and colon but small intestine recovers within 4-8 hours after surgery. Hence feeding within 24 hours after surgery is very well tolerated. Evidence from clinical studies suggests that initiating feeding early is advantageous and reverses mucosal atrophy induced starvation and also increases collagen deposition at the surgical sites, promoting wound healing. Finally, early enteral feeding may also reduce septic morbidity.

AIMS AND OBJECTIVES:

To compare the feasibility, safety, efficacy, tolerability and outcome of early enteral feeding versus late oral feeding after elective and emergency gastrointestinal surgeries.

STUDY DESIGN: Prospective Comparative Study.

STUDY POPULATION:

Age more than 18 years and below 60 years undergoing Elective and Emergency Gastrointestinal Surgeries.

SAMPLE SIZE: 25in Each Group.

STUDY PERIOD: JANUARY 2018 TO JUNE 2019

INCLUSION CRITERIA:

All patients with age more than 18 years and less than 60 years which are undergoing gastro intestinal surgery (Elective and Emergency) and were hemodynamically stable patients with ready to sign an informed written consent were included.

EXCLUSION CRITERIA:

Age less than 18 years and more than 60 years, hemodynamically unstable patients &incompetent to provide informed written consent were excluded.

METHODOLOGY:

All patients undergoing major gastrointestinal surgeries (Elective and Emergency) in Coimbatore Medical College Hospital based on inclusion and exclusion criteria are enrolled in this study.

1. Informed written consent was obtained from all patients.
2. A detailed history and clinical examination was done on all patients.
3. Baseline Investigation – Complete Haemogram, Blood sugar, Renal function test, Serum Electrolytes, Liver function tests, Electro cardiogram, Chest X-Ray, X-ray Abdomen Erect, USG Abdomen and Pelvis, CT Abdomen and Pelvis(as per the need), HIV I and II, Blood Grouping and Typing.
4. Surgery was performed as per the plan (Elective and/or Emergency).
5. Patients in the study group were started on early enteral feeding through nasogastric tube, feeding Jejunostomy or by mouth – within 1st 24 hours post-operatively.
6. Patients in the control group were kept nil by mouth with nasogastric decompression until evidence that bowel function has returned, after which a liquid diet is initiated and advanced to a regular diet over 4-5 days.

7. The outcomes were compared in the terms paralytic ileus, wound dehiscence, wound infection, GI complications and intraabdominal abscess, length of hospital stay after operation.
8. All data were recorded and statistically analysed. Specific instruction was given to each patient on discharge, to come for periodical review regularly.

FLOW CHART:

PATIENTS PLANNED FOR ELECTIVE AND EMERGENCY GASTROINTESTINAL SURGERIES

INFORMED WRITTEN CONSENT

DETAILED HISTORY AND CLINICAL EXAMINATION

BASELINE INVESTIGATIONS (CBC, BLOOD SUGAR,RFT,LFT,SERUM ELECTROLYTES, CHEST X-RAY, X-RAY ABDOMEN- ERECT, USG-ABDOMEN & PELVIS, CT-ABDOMEN & PELVIS, HIV I & 2, BLOOD GROUPING & TYPING)

SURGERIES ARE PERFORMED AS PER THE PLAN

PATIENTS IN STUDY GROUP STARTED ON EARLY ENTERAL FEEDING WITHIN 1ST 24 HOURS POSTOPERATIVELY AND IN CONTROL GROUP KEPT NIL BY MOUTH

CAREFUL MONITORING OF OUTCOMES IN THE TERMS PARALYTIC ILEUS, WOUND DEHISCENCE, WOUND INFECTION, GI COMPLICATIONS AND INTRA-ABDOMINAL ABSCESS, LENGTH OF HOSPITAL STAY AFTER OPERATION.

EVALUATION OF STUDY RESULTS USING STANDARD STATISTICAL ANALYSIS METHODS

REVIEW OF LITERATURE

- ANATOMIC CONSIDERATIONS
- PHYSIOLOGY OF DIGESTIVE SYSTEM
- PHYSIOLOGICAL AND METABOLIC CHANGES DURING SURGERY
- NUTRIONAL ASSESSMENT OF SURGICAL PATIENTS
- NUTRITIONAL REQUIREMENTS IN SURGICAL PATIENTS
- METHODS OF NUTRITIONAL SUPPLEMENTATION
- ENTERAL FEEDING METHODS
- PARENTERAL FEEDING METHODS
- POSTOPERATIVE OUTCOME
- THE ERAS PROTOCOL

ANATOMIC CONSIDERATIONS

♦ ANATOMY OF DIGESTIVE SYSTEM:

The human digestive tract is a long, coiled, muscular tube that stretches from the mouth to the anus.

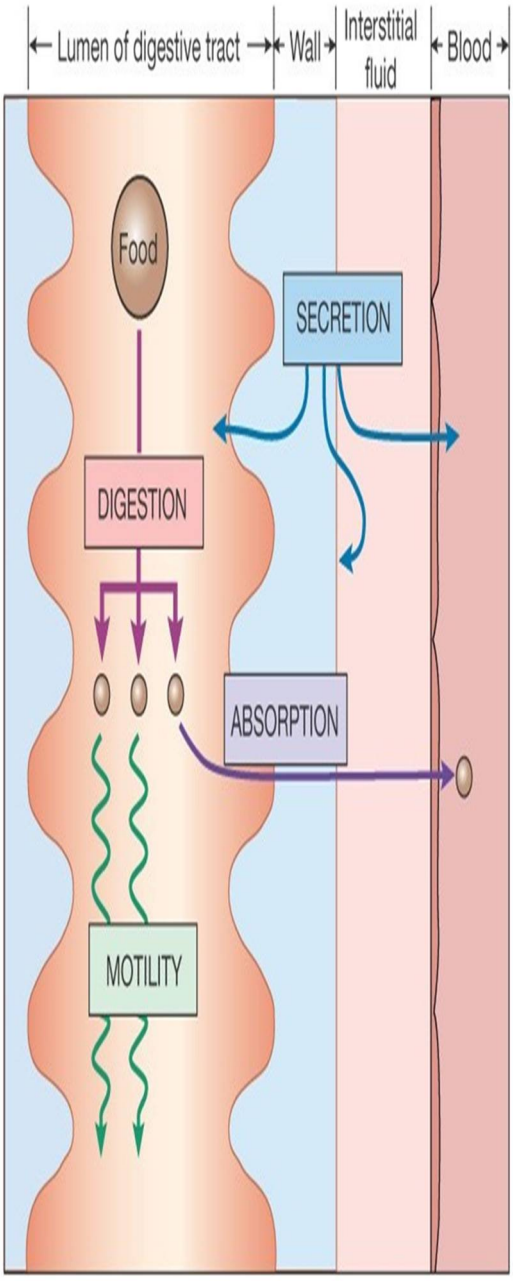
Two main groups

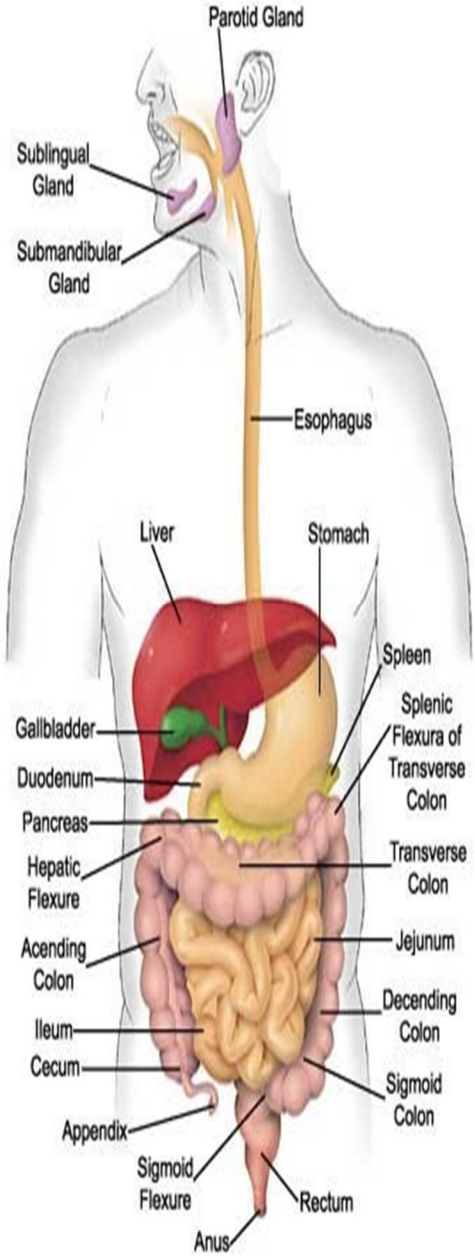
ALIMENTARY CANAL OR GASTROINTESTINAL TRACT: Continuous coiled hollow tube from mouth to anus (9 meters).

ACCESSORY DIGESTIVE ORGANS: Teeth, Tongue, Salivary Gland, Liver, Gallbladder, and Pancreas.

FUNCTION OF THE DIGESTIVE SYSTEM:

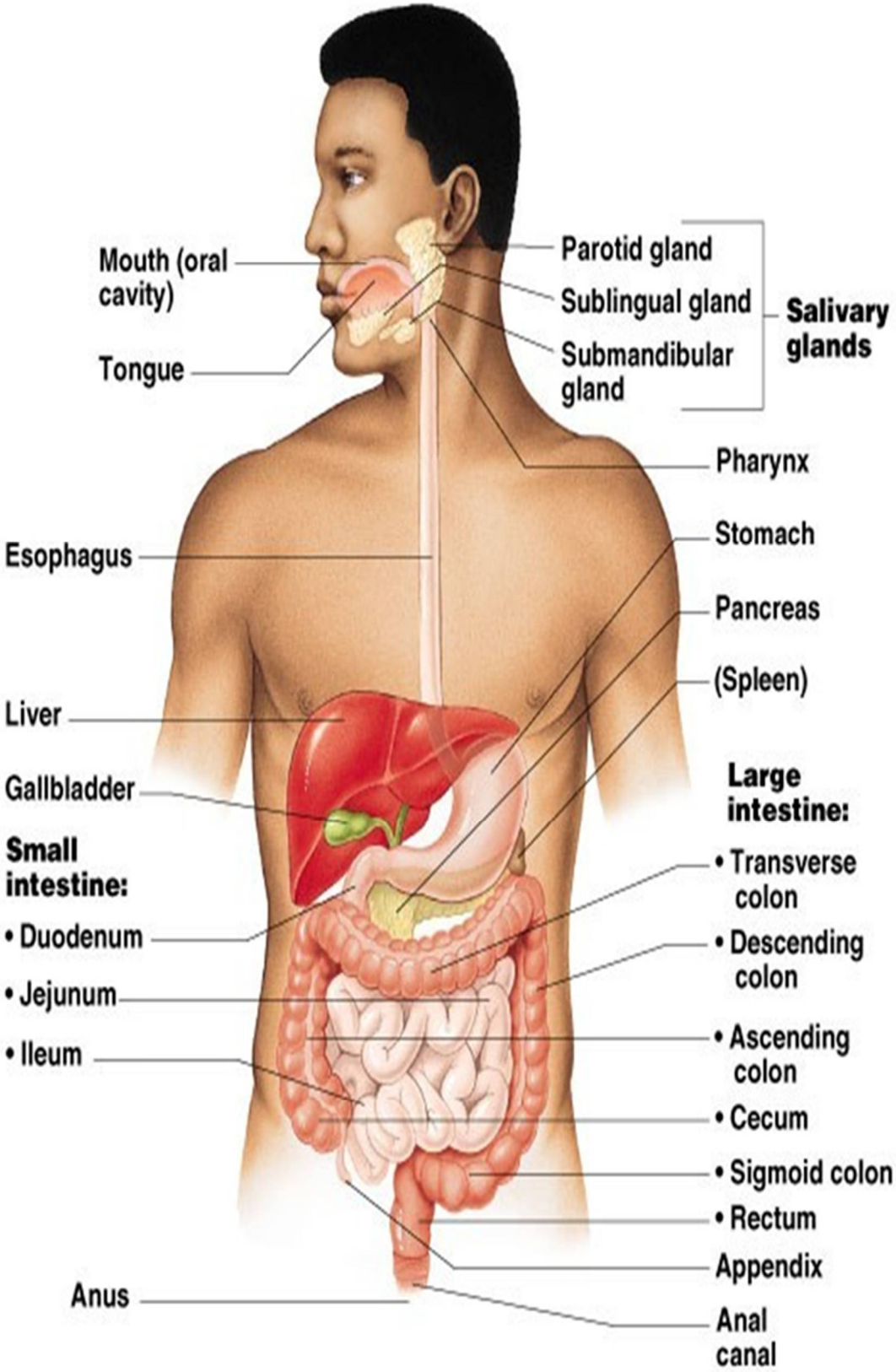
- Ingestion: taking food and liquid into mouth.
- Secretion: total about 7 litres into lumen.
- Mixing and propulsion: through GI muscle and peristalsis and motility.
- Digestion: Breakdown of ingested food (mechanical and chemical).
- Absorption: Passage of nutrients into the blood.
- Metabolism: Production of cellular energy (ATP).
- Defecation: waste substance leaves the GI tract through anus.


Lumen of digestive tract
Wall
Interstitial fluid
Blood
Food
SECRETION
DIGESTION
ABSORPTION
MOTILITY


Parotid Gland
Sublingual Gland
Submandibular Gland
Esophagus
Liver
Stomach
Spleen
Splenic Flexura of Transverse Colon
Gallbladder
Duodenum
Pancreas
Hepatic Flexure
Transverse Colon
Acending Colon
Jejunum
Decending Colon
Ileum
Cecum
Sigmoid Colon
Appendix
Sigmoid Flexure
Rectum
Anus

ORGANS OF THE ALIMENTARY CANAL

- Mouth: Helps in chewing of food, mixing masticated food with saliva to produce easy digested food called bolus, Saliva contain 2 enzymes, salivary amylase and lingual lipase, Initiation of swallowing by the tongue, Allowing for the sense of taste.
- Pharynx: Receives food from mouth passes it into oesophagus.
- Oesophagus: Allows passage of food from pharynx into the stomach, formation of food bolus, swallowing, prevention of gastric reflux.
- Stomach: Storage of food for 2-4 hours, partial digestion of food by the gastric juice, absorption of certain small molecules. Stomach empties its contents into duodenum for further digestion.
- Small intestine: comprises of Duodenum, Jejunum and Ileum. Involved in most of the chemical digestion by the digestive enzymes secreted from Liver and Pancreas. Digestion of proteins, carbohydrates and lipids. Absorption of most of the nutrients from the ingested food takes place in small intestine. Small intestine also supports body's immune system by the presence of intestinal flora.
- Large intestine: it absorbs water and any remaining absorbable nutrients from the food before sending the indigestible matter to the rectum. Colon also absorbs vitamins that are created by the colonic bacteria, such as vitamin K.
- Rectum and Anus: formed stool is stored in the rectum for sometime before being expelled by the act of defecation through anus.


Mouth (oral cavity)
Tongue
Parotid gland
Sublingual gland
Submandibular gland
Salivary glands
Pharynx
Esophagus
Stomach
Pancreas
(Spleen)
Liver
Gallbladder
Small intestine:
• Duodenum
• Jejunum
• Ileum
Large intestine:
• Transverse colon
• Descending colon
• Ascending colon
• Cecum
• Sigmoid colon
• Rectum
Appendix
Anus
Anal canal

PHYSIOLOGY OF DIGESTIVE SYSTEM:

- **TWO PHASES OF DIGESTION:**

1) **MECHANICAL PHASE** – involves the breaking up of food into small pieces, pushing the food down the food tube, and mixing with it digestive juices. This is done in the mouth with the help of four types of teeth (incisors, canine, molars and premolars).
2) **CHEMICAL PHASE** – involves the further breaking up of the larger molecules of food into smaller molecules by the action of digestive enzymes.

DIGESTION IN THE MOUTH:

Digestion of the food begins as soon as ingested food is chewed and mixed with saliva secreted from the salivary glands.

PROPERTIES AND COMPOSITION OF SALIVA:

Volume – 1000 ml to 1500 ml per day is secreted.

Reaction – Slightly acidic with pH of 6.35 to 6.85.

Specific gravity ranges from 1.002 to 1. 012.

Saliva is hypotonic to plasma.

Contribution of Secretion of saliva by each major salivary gland is

Parotid Gland – 25 %

Sub maxillary Gland – 70 %

Sublingual Gland – 5 %

Mixed saliva contains 99.5 %

Water and 0.5 % solids.

SALIVARY ENZYMES: Amylase, Lingual Lipase, Maltase, Lyzozyme, Phosphatase, Carbonic anhydrase and Kallikrein.

ORGANIC SUBSTANCES: Mucin, Albumin, Proline rich proteins, Lactoferrin, IgA, Blood group antigens, Free amino acids and other non protein nitrogenous substances like urea, uric acid creatinine, xanthine and hypoxanthine.

OTHER INORGANIC SUBSTANCES:

Sodium, Potassium, Calcium, Bicarbonate, Phosphate, Bromide, Chloride and Fluoride.

GASES: Oxygen, Nitrogen and Carbon dioxide.

DIGESTIVE FUNCTIONS OF SALIVA:

1. Salivary amylase is a carbohydrate – digestive enzyme. It acts on cooked or boiled starch and converts into dextrin and maltose. Optimum pH, necessary for the activation of salivary amylase is 6. Salivary amylase cannot act on cellulose.
2. Maltase – it is present only in traces in human saliva and it converts maltose into glucose.
3. Lingual Lipase – it is a lipid digesting (lipolytic) enzyme. It is secreted from serous glands situated on the posterior aspect of the tongue. It digests milk fats. It hydrolyses triglycerides into fatty acids and diacylglycerol.

Enzyme lysozyme of saliva kills some bacteria such as staphylococcus, streptococcus and brucella.

Proline rich saliva possesses antimicrobial property and neutralizes the toxic substances such as tannins. Tannins are present in fruits.

Lactoferrin of saliva also has antimicrobial property.

Immunoglobulin IgA in saliva also has antibacterial and antiviral actions. Mucin protects the mouth by lubricating the mucus membrane of mouth.

DIGESTION IN STOMACH:

Formation of Chyme – peristaltic movements of stomach mix the bolus with gastric juice and convert it into the semisolid material known as Chyme.

GASTRIC JUICE:

Volume: 1200 ml / day to 1500 ml / day.

Reaction: Gastric juice is highly acidic with a pH, of 0.9 to 1.2 (Acidity of juice is due to presence of HCL)

Specific gravity: 1.002 to 1.004

COMPOSITION OF GASTRIC JUICE:

Gastric juice present in stomach mainly acts on the proteins. Proteolytic enzymes of the gastric juice are pepsin and renin. Gastric juice also contains some other enzymes like gastric lipase, gelatinase, urase and gastric amylase.

Pepsin – it is secreted as inactive pepsinogen. Pepsinogen is converted into pepsin by HCL. Optimum pH, for activation of pepsinogen is below 6. Pepsin converts proteins into proteoses, peptones, and polypeptides, pepsin also causes curdling and digestion of milk (Casein).

Gastric Lipase – it is a weak lipolytic enzyme when compared to pancreatic lipase. It is active only when the pH, is between 4 and 5 and becomes inactive at pH, below 2.5.

Mucus is a mucoprotein, secreted by mucus neck cells of the gastric glands and surface mucus cells in fundus, body and other parts of stomach. It protects the gastric wall by the following ways. Mucus – Protects the stomach wall from irritation of mechanical injury. Prevents the digestive action of pepsin on the wall of the stomach, particularly gastric mucosa. Protects the gastric mucosa from hydrochloric acid of gastric juice because of its alkaline nature.

Intrinsic factor of Castle, secreted by parietal cells of gastric glands plays an important role in erythropoiesis. It is necessary for the absorption of Vitamin B12 (Extrinsic factor) from GI tract from GI tract into the blood. Vitamin B12 is an important maturation factor during erythropoiesis. Absence of intrinsic factor in gastric juice causes deficiency of vitamin B12 leading to pernicious anemia.

DIGESTION IN THE SMALL INTESTINE:

This is carried out mainly by Bile from the liver and pancreatic juice.

BILE:

Secreted by hepatocytes. It contains bile acids, bile pigments, cholesterol, lecithin and fatty acids. Most of the bile are stored in the gall bladder.

PROPERTIES OF BILE

Volume – 800 – 1200 ml / day.

Reaction – Alkaline pH – 8 to 8.6.

Specific gravity – 1.010 – 1.011.

Colour – Golden Yellow or Green.

COMPOSITION OF BILE

Water – 97.6 %

Salts – 2.4 % (Organic and Inorganic)

As bile is highly alkaline, it neutralizes the acid chyme which enters the intestine from stomach. Thus an optimum pH is maintained for the action of digestive enzymes.

Bile Salts are the sodium and potassium salts of bile acids.

Functions of Bile Salts are -

1. Emulsification of fats – bile salts emulsify the fats by reducing the surface tension due to their detergent action.
2. Absorption of fats – bile salts combine with fats and makes complexes of fat called micelles. The fat in the form of micelles can be absorbed easily.
3. Choleretic Action – Bile salts stimulates the secretion of bile from the liver.
4. Cholagogue Action– Cholagogue is an agent which causes contraction of gall bladder and release of bile into the intestine.

Bile salts acts indirectly by stimulating the secretion of hormone Cholecystokinin. This hormone causes contraction of gallbladder, resulting in the release of Bile.

5. Laxative Action – Laxative is an agent which inducesϖ defecation. Bile acts as laxatives by stimulating peristaltic movements of the intestine.
6. Prevention of Gallstone Formation – Bile salts preventϖ the formation of gallstone by keeping the cholesterol and lecithin in solution. In the absence of bile salts, cholesterol precipitates along with lecithin and forms gallstone.
7. Antiseptic action – Bile salts inhibits the growth of certain bacteria in the lumen of intestine by its natural detergent action.
8. Lubrication Function – The mucin in bile acts as a lubricant for the chyme in the intestine.
9. Excretory function – bile pigments are the major excretory products of the bile. Other substances excreted in the bile - Heavy metals like copper and iron, some bacteria like typhoid bacteria, some toxins, and Cholesterol, Lecithin, and Alkaline phosphatase.

BILE PIGMENTS

Bile pigments are the excretory products of bile. Bilirubin and Biliverdin are the two bile pigments and bilirubin is the major bile pigment in human beings. Bile pigments are formed during the breakdown of

haemoglobin, which is released from the destroyed RBCs in the reticuloendothelial system.

Normal plasma levels of Bilirubin – 0.5 – 1.5 Mg/dl.

FUNCTIONS OF GALLBLADDER:

Bile secreted from liver is stored in gallbladder. The capacity of gallbladder is approximately 50 ml.

Major functions of Gallbladder are as follows-

1. Storage of bile – Bile is continuously secreted by liver is stored in the gallbladder; it is released in the intestine intermittently when it is required. Concentration of bile – Substances like bile pigments, cholesterol, and lecithin helps to concentrate bile 5 to 10 times.
2. Alteration of pH of bile – The pH of bile is decreased from 8 – 8.6 to 7 – 7.6 and it becomes less alkaline when it is stored in gallbladder.
3. Secretion of Mucin – Gallbladder secretes mucin. Mucin is added to bile, when bile is released into intestine, mucin acts as lubricant for movement of chyme in the intestine.
4. Maintenance of pressure in biliary system – Due to the concentrating capacity gallbladder maintains pressure of about 7cm H2O in billiary system. This pressure in the biliary system is essential for the release of bile into the intestine.

PANCREATIC JUICE

Properties - Volume: 500 to 800 ml / day

Reaction: Highly Alkaline with a pH of 8 to 8.3

Specific gravity: 1.010 to 1.018

PANCREATIC JUICE FUNCTIONS

Two functions of pancreatic juice are Digestive & Neutralizing function. **Digestive function** –

Digestion of Proteins – Major proteolytic enzymes are Trypsin and Chymotrypsin. Others are Carboxypeptidases, Nuclease, Elastase and Collegenase. Trypsin – it is a polypeptide, it contains 229 amino acids. It is secreted as inactive trypsinogen, which is converted into active trypsin by enterokinase and it is secreted by the brush – bordered cells of duodenal mucous membrane. – Actions of Trypsin - Most powerful proteolytic enzymes curdling of Milk (Converts caseinogen in the milk to casein Accelerates blood clotting.

Chymotrypsin - It is secreted in an inactive form chymotrpsinogen which is converted into an active form Chymotrypsin by trypsin. Action of Chymotrypsin – Converts proteins into polypeptides It digests caseinogen faster than trypsin No action on Blood Clooting

Digestion of Lipids -

Pancreatic Lipase is a powerful lipolytic enzyme. It digests triglycerides into monoglycerides and fattyacids. Activity of pancreatic lipase is accelerated in the presence of bile.

Digestion of fat by pancreatic lipase requires two more factors/ –

Bile Salts: It is responsible for the emulsification of fat, prior to the digestion. – Colipase: It is a coenzyme necessary for the pancreatic lipase to digest the dietary lipids. About 80 % of the fat is digested by pancreatic lipase. Deficiency or absence of this enzyme leads to excretion of undigested fats in faeces (Steatorrhea)

Digestion of Carbohydrates –

Pancreatic amylase is the amylotic enzyme present in pancreatic juice. Like salivary amylase it also converts starch into dextrin and maltose.

SUCCUS ENTERICUS:

It refers to the clear to pale yellow watery secretions from the glands lining the small intestinal walls. The Brunner's glands secrete large amounts of alkaline mucus in response to (1) tactile or irritating stimuli on the duodenal mucosa; (2) vagal stimulation, which causes increased Brunner's glands secretion concurrently with increase in stomach secretion; and (3) gastrointestinal hormones, especially secretin.

Characteristics of succus entericus: 1.Total quantity: Roughly 2-3 litres in 24 hours

2.Specific gravity: 1.010

3. Reaction: faintly acid to faintly alkaline

4. PH: 6.3 – 9.0

Composition:

Water: 98.5% Solids: 1.5% 1.

Inorganic: 0.8% salts of Na, K, Ca and Mg with that of chloride, bicarbonate and phosphate.

Organic: 0.7% - Enzymes:

Enteropeptidases: activates chymotrypsin to trypsin.

Erepsin: A mixture containing dipeptidases and amino peptidases.

Nucleases: acts on DNA, RNA.

Amylase: acts on starch and dextrin.

Sucrase, Maltase, Lactase, Lipase.

Other enzymes: alkaline phosphatase, cholesterol esterase, lecithinase

Mucin

Functions: Digestion of carbohydrate, fat and protein

It contains oligo- α-(1-6) glucosidase which splits α (1-6) glycosidic linkage of dextrin. Thus provide scope of further activity of amylase to produce glucose and maltose.

Maltase acts on maltose and produce glucose. Sucrase and lactase present in this juice act on sucrose and lactose respectively and produce glucose, fructose and galactose. Erepsin is a mixture of enzymes including

different peptidases. The various peptidases act serially, one after other, upon the smaller and smaller fragments of peptide molecule, till they are completely broken down into amino acids. It contains lipase. Since under normal condition fat digestion is almost completed by pancreatic juice, this lipase is of little importance. It is needed only to deal with little quantity of fat which might have accidently escaped pancreatic digestion.

ABSORPTION OF NUTRIENTS IN GIT

In the mouth no absorption occurs, except for some drugs.

In the oesophagus no absorption occurs.

In the stomach absorption of water, some simple sugars, alcohol & some drugs like aspirin occurs.

The majority of absorption occurs in the Small intestine. Absorption takes place in the villi & microvilli which increase the surface area of SI by 600 times to make the absorption process very efficient.

In Large intestine only water & electrolytes are absorbed.

WATER & ELECTROLYTE ABSORPTION:

Absorption of H2O – is by simple diffusion following the law of osmosis to cause isosmotic equilibrium when ions/nutrients absorbed.

Absorption of Na+ is by active transport from inside the epithelial cells into the intercellular spaces requiring energy supplied by ATPase carrier enzyme in the cell membrane. Part of Na+ is absorbed with Cl- while the other is either with K+ or H+ ions transported in the opposite direction in exchange for Na+ helped by aldosterone when there is need for Na+ in the body as in dehydration. When Na+ is absorbed H+ is secreted into the SI lumen in exchange of Na+, excess H+ combines with excess HCO3- to form carbonic acid which dissociates into H2O & CO2 excreted by the lungs. In the ileum & large intestine there is secretion of HCO3- in exchange of CL- by exchange proteins in luminal membrane of the epithelial cells to provide alkaline medium to neutralize acid formed by the bacteria.

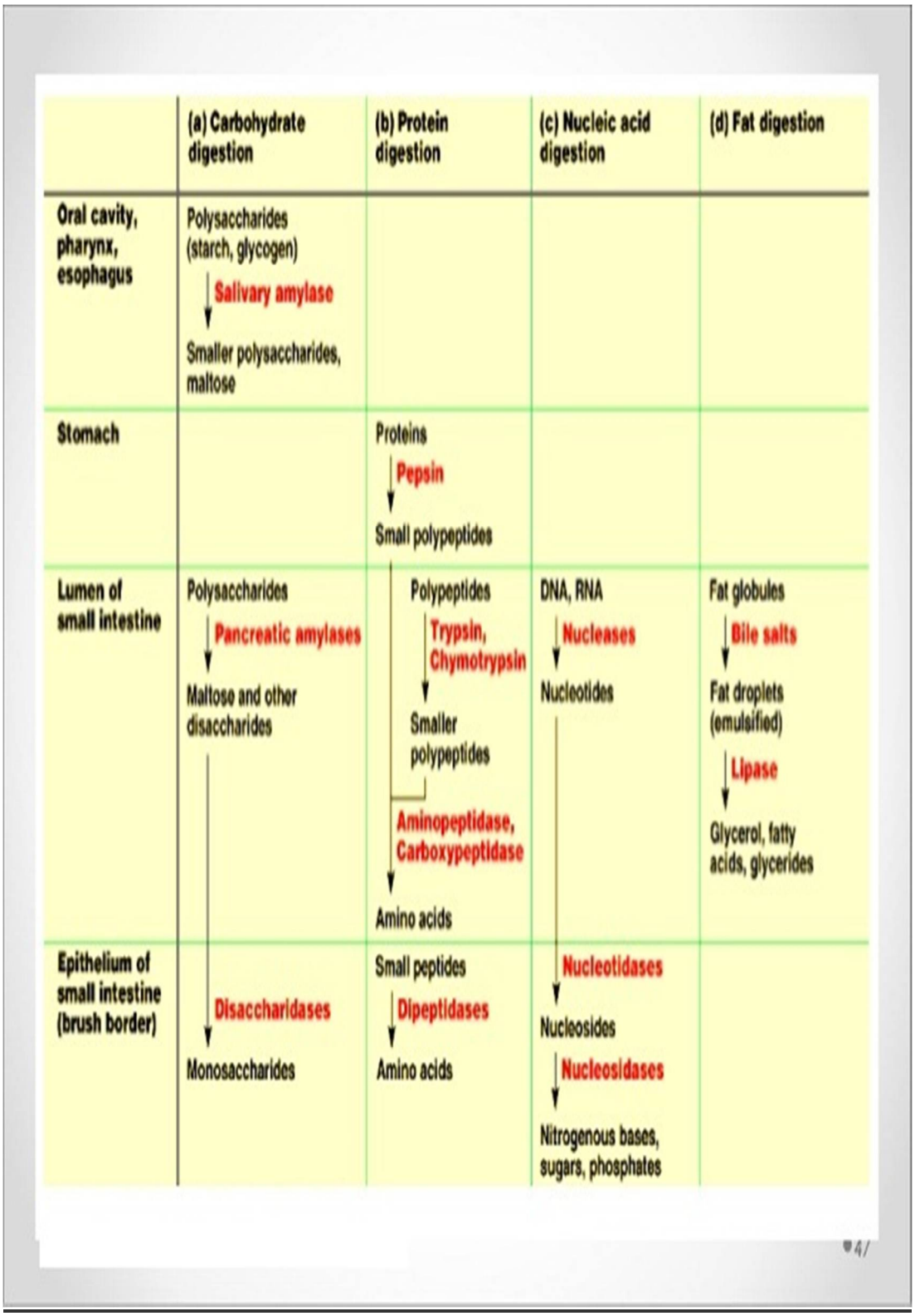
ABSORPTION OF CARBOHYDRATES& PROTEINS:

Glucose is the most rapidly absorbed sugar than other carbohydrates digestion end products; fructose, manose,xylose& arabinose with the exception of galactose which is slightly more absorbed than glucose but glucose is present in very much higher concentrations. Mechanism of glucose & galactose absorption is Na+-cotransport (secondary active transport of glucose & galactose) while that of fructose is different. It does not require energy, but by fascilated diffusion & fructose is partly converted to glucose inside the epithelial cells before entering the portal blood.

Absorption of amino acids is not by Na cotransport but by secondary active transport of amino acids & peptides requiring special membrane transport protein of which there are at least five.

ABSORPTION OF FATS:

Bile salts & pancreatic lipase are the most important factors in fat absorption. The bile salts facilitate fat absorption by reducing the surface tension of fat particles & by forming water soluble complexes called micelles in which fatty acids & glycerol also participate. The fats digestion end products; fatty acids & monoglycerides are recombined after entering the epithelial cells by the smooth endoplasmic reticulum to form triglycerides & chylmicrones again which are easily absorbed by the lymphatics of the villi called the lacteals to the lymphatic system & not by the portal blood, while some short chained fatty acids are absorbed directly into the portal blood rather than lymphatics and this can be used in fat malabsorbtion



	(a) Carbohydrate digestion	(b) Protein digestion	(c) Nucleic acid digestion	(d) Fat digestion
Oral cavity, pharynx, esophagus	Polysaccharides (starch, glycogen) → Salivary amylase → Smaller polysaccharides, maltose			
Stomach		Proteins → Pepsin → Small polypeptides		
Lumen of small intestine	Polysaccharides → Pancreatic amylases → Maltose and other disaccharides	Polypeptides → Trypsin, Chymotrypsin → Smaller polypeptides → Aminopeptidase, Carboxypeptidase → Amino acids	DNA, RNA → Nucleases → Nucleotides	Fat globules → Bile salts → Fat droplets (emulsified) → Lipase → Glycerol, fatty acids, glycerides
Epithelium of small intestine (brush border)	Disaccharidases → Monosaccharides	Small peptides → Dipeptidases → Amino acids	Nucleotidases → Nucleosides → Nucleosidases → Nitrogenous bases, sugars, phosphates	

PHYSIOLOGICAL AND METABOLIC CHANGESS DURING SURGERY:

Systemic response to surgery encompasses a wide range of interlinked endocrinological , metabolic and immunological pathways.

THE ENDOCRINE RESPONSE TO SURGERY:

Hormonal response to surgery is characterized by increased secretion of stress hormones.: adrenaline and cortisol (prominent markers), glucagon, growth hormone, aldosterone, ADH

In response to surgery, not only hormonal pathways but also sympathetic nervous system lead to catabolism with mobilization of catabolism, with mobilization of substrates – to provide energy, salt and water retention cardiovascular retention, cardiovascular haemostasis.

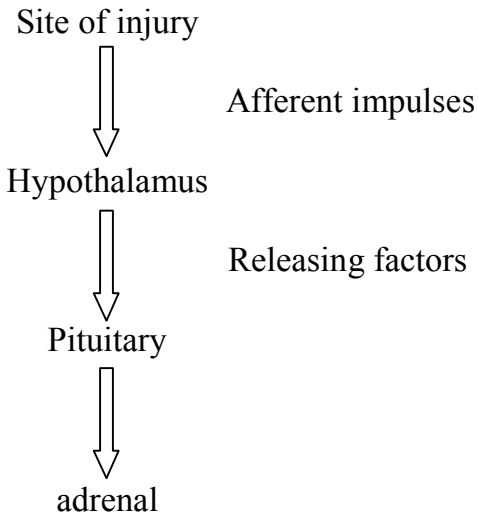
Hormonal response by:

- The sympatho-adrenal response
- The hypothalamo – pituitary – adrenal axis

The sympathoadrenal axis:

- During surgery, sympathetic autonomic nervous system is activated
- This results in increased secretion of catecholamines from adrenal medulla from adrenal medulla.
- Increased activity leads to tachycardia and hypertension and hypertension.

The hypothalamo-pituitary-adrenal axis:



Metabolic sequelae of the endocrine response

The net effect of the endocrine response to surgery - ↑catabolic hormones → breakdown of skeletal muscles and fat and ↑ gluconeogenesis and insulin resistance manifested as hyperglycaemia.

It was correlated to the intensity of the surgical insult surgical insult.

• The ability to recover following surgery may be dependent on the ability to deliver the increased oxygen demand to tissues during the hypermetabolic phase.

• Catabolism may result in marked weight Catabolism may result in marked weight loss and muscle wasting in patients after surgery after surgery.

THE HAEMODYNAMIC RESPONSE TO SURGERY:

• Fluid lost in surgery (blood or body fluids)+ hormonal changes influences salt and water retention and production of concentrated retention and production of concentrated urine by direct action of the kidney.

• ↑ sympathetic efferent stimulation in the kidney → renin from JG cells from JG cells →Angiotensin Angiotensin II →Aldosterone →salt and water retention

THE IMMUNE SYSTEM AND THE SYSTEMIC RESPONSE TO SURGERY:

• A functioning immune system is essential in order to prevent postoperative complications , particularl sepsis.

• However, surgery insigates a number of response from both the specific and non response from both the specific and non specific immune systems, both pro and anti inflammatory anti inflammatory.

EFFECTS OF ANESTHESIA ON THE SYSTEMIC RESPONSE TO SURGERY:

- Drugs used in anesthesia can alter the systemic response to surgery.
- Opioid analgesia : Hypothalamus and pituitary hormone secretion
- Induction agent , etomidate : can interfere with the production of cortisol and aldosterone. (↑mortality when used in critically ill case)
- Benzodiazepines: cortisol production but clinical significance is unknown. Antihypertensive clonidine (alpha2 agonist) sympathetic pathway.
- Regional anaesthesia (epidural block) can block afferent input from the site of surgery to H-P axis and efferent ANS pathways.

NUTRIONAL ASSESSMENT OF SURGICAL PATIENTS (11,12,14)

Nutritional status is very important for health, as it is a major determinant of health. It depends on many factors like nutrient intake, type of food taken, frequency of food, co-morbid conditions like diabetes, any chronic infections like tuberculosis, repeated infections like acute diarrhea, respiratory infections, hormonal factors like thyroid, etc. Socioeconomic status and cultural practices also play an important role in determining nutritional status.

Nutritional intake depends on many factors like appetite, taste, availability, and mood. Adequate amount of food intake is essential for optimal nutritional status. Excessive intake can lead to obesity and decreased intake leads to under nutrition. An adult with moderate activity needs around 2400 Kcal/day. The intake should also be in adequate proportions (balanced diet). These requirements depend on the age, gender, and other physiological status of the person.

Nutritional support is critical at a time of severe stress as the synthesis of acute phase proteins, white cells, fibroblasts, collagen, and other tissue components are required for proper wound healing and recovery. In some circumstances energy requirements can reach as high as 30 kcal/kg ideal body weight, with a daily nitrogen requirement equivalent to a protein intake of 1.5 g/kg ideal body weight. Preferably the protein:fat:glucose caloric ratio should approximate 20%:30%:50% of one's daily intake. It is therefore important for physicians to be able to determine which patients are at greater risk for

postoperative complications and how malnourished these particular patients are prior to surgery.

Nutritional support of surgical and critically ill patients has undergone significant advances since 1936 when Studley demonstrated a direct relationship between preoperative weight loss and operative mortality. Today, malnutrition is considered a risk factor for impaired systemic and intestinal immune function, as well as decreased digestive and absorptive capacity due to the altered architecture of the gut barrier.

The advent of total parenteral nutrition (TPN) followed by the extraordinary progress in parenteral and enteral feedings, in addition to the increased knowledge of cellular biology and biochemistry, have allowed clinicians to treat malnutrition and improve surgical patient's outcomes.

Major stress, such as surgery, can subject a patient to a whole host of metabolic and physiologic changes. The body responds to such stress by increasing its basal metabolic rate (BMR), using up its nitrogen stores and creating a negative nitrogen balance. An increase in gluconeogenesis as well as the synthesis of acute phase proteins is also observed. The body scavenges for the required nutrients during such times of stress, which if continue unchecked for prolonged periods of time could lead to adverse consequences.

Perioperative nutritional supplementation, therefore, should blunt the catabolic effects of such a high energy state. Of interest is the increase in intestinal permeability during periods of surgical stress, which can be as such as fourfold greater in some patients usually normalizing around postoperative

day five. Associated with this increase in permeability, is a decrease in villous height, leading to malabsorption and an impaired ability of the gut to act as a barrier against endogenous bacteria and toxins.

The most common surgical practice of making patients NPO after midnight of the day of any planned surgical procedure has been recently questioned. Brady *et al*. reviewed 38 randomized controlled trials on perioperative fasting and concluded that there was no evidence to suggest overnight fasting for fluids results in a decrease in perioperative aspiration risk or related morbidities.

Evidence is emerging that overnight fasting is not just unnecessary, but may also be harmful. Surgical stress cause postoperative insulin resistance, immunosuppression, and increased patient discomfort. Preoperative ―carbohydrate loading with carbohydrate rich drink three hours prior to scheduled procedure has been shown to attenuate the above adverse effects of fasting, particularly in diabetic patients.

Another previously prevalent practice involves the use of clear fluids only prior to surgery and in the early post operative period. This can induce a starvation state as glycogen stores will be depleted within a few hours and the body promotes gluceoneogenesis through breakdown of muscle and other visceral proteins.

Perioperative nutritional support was devised with the following goals:

(1) To prevent or revert the catabolic effects of surgery,

(2) To meet the energy requirements of the metabolic process,

(3) To maintain core body temperature and provide substrates for adequate tissue repair,

(4) To minimize negative protein balance by avoiding starvation,

(5) To maintain muscle, immune, and cognitive function and, ultimately,

(6) To enhance postoperative recovery and return of function.

NUTRITIONAL ASSESSMENT:

Dietary History and History of weight loss.

Physical Examination:

General appearance (emaciated, apathetic look).

Assessment of body fat stores (Skin fold examination over biceps and triceps, subscapular region).

Assessment of protein stores (Muscle bellies of biceps, triceps, supra and infraspinatus).

Assessment of metabolic stress (indirect calorimetry, temperature, WBC count, pulse, positive blood culture, abscess).

Physiological function – poor wound healing, early fatiguability, grip strength, respiratory muscle function test.

Body weight and Anthropomentry.

Laboratory tests: Serum albumin levels, Creatinine excretion, serum Transferrin, Lymphocyte count, Skin hypersensitivity tests.

The must tool BMI, weightt loss in 3-6 months, acute disease.

MINI NUTRITIONAL ASSESSMENT SCORE

Ask the patient to answer questions A – E and calculate the final score.

A - Has food intake declined over the past three months due to loss of appetite, digestive problems, chewing or swallowing difficulties?

Score 0 = Severe decrease in food intake

1 = Moderate decrease in food intake

2 = No decrease in food intake

B - Involuntary weight loss during the last 3 months?

Score 0 = Weight loss greater than 3 kg (6.6 pounds)

1 = Does not know

2 = Weight loss between 1 and 3 kg (2.2 and 6.6 pounds)

3 = No weight loss

C - Mobility?

Score 0 = Bed or chair bound

1 = Able to get out of bed/chair, but does not go out

2 = Goes out

D - Has the patient suffered psychological stress or acute disease in the past 3 months?

Score 0 = Yes

2 = No

E - Neuropsychological problems?

Score 0 = Severe dementia or depression

1 = Mild dementia

2 = No psychological problems

F - Body mass index (BMI)? (Weight in kg / height in m2)

Score 0 = BMI less than 19

1 = BMI 19 to less than 21

2 = BMI 21 to less than 23

3 = BMI 23 or greater

Nutritional Screening Score (Max. 14 points)

12-14 points: Normal nutritional status

8-11 points: At risk of malnutrition

< 7 points : Malnourished.

Interestingly, malnutrition can occur in obese patients who have low muscle mass. This form of obesity termed sarcopenic obesity may be less recognizable in many cases. In many patients fat-free mass index may be a better predictor for mortality than body mass index. Van Venrooij *et al.* found that low fat-free mass index was associated with increased occurrence of adverse outcomes after surgery. They advocate fat-free mass index as the leading parameter in classifying and treating malnourished surgical patients

Options for addressing malnutrition in preoperative patients include dietary counseling, oral supplementation, and artificial feeding. It is estimated that the average survival time for patients at home that do not address their nutritional needs is 19 days. Perioperative nutritional intervention, therefore, becomes of great interest and value to both the patient and the surgeon for improving postoperative outcomes.

When designing a nutritional plan for a patient at risk one should ensure that the program is of sufficient duration and intensity to improve known markers of malnutrition. Patient age, sex, height, weight and clinical status, in addition to a detailed medical history and medication list, should be taken into account.

NUTRITIONAL REQUIREMENTS IN SURGICAL PATIENTS

- Nutritional goal aims at maintaining Nitrogen balance, preserving or restoring visceral protein, reducing morbidity and mortality and reducing the length of hospital stay
- Enteral requirements are delivery into the GI tract by tube with minimum risk of aspiration or patient effort, delivery of nutrients with minimal need for digestion and control of rate to prevent osmotic diarrhea.
- Energy sources:

    1) Carbohydrates: Limited storage capacity, essential for CNS function.

    Yields 3.4 Kcal/gram.

    Pitfall: too much intake induces lipogenesis and increased CO2 production

    2) Fats: Major endogenous fuel source in healthy adults.

    Yields 9 kcal/gm.

    Pitfall: too little intake may lead to essential fatty acids deficiency-dermatitis and increased risk of infections

    3) Protein: Needed to maintain anabolic state (match catabolism)

    Yields: 4 kcal/gm. Pitfall: must adjust in patient with renal and hepatic failure, elevated creatinine, BUN, and/or ammonia.

- Nutritional requirements for Healthy Adults:

  Calories: 25-35 kcals/kg

  Protein: 0.8-1 gm/kg

  Fluids: 30 mls/kg

- Postoperative nutritional requirements:

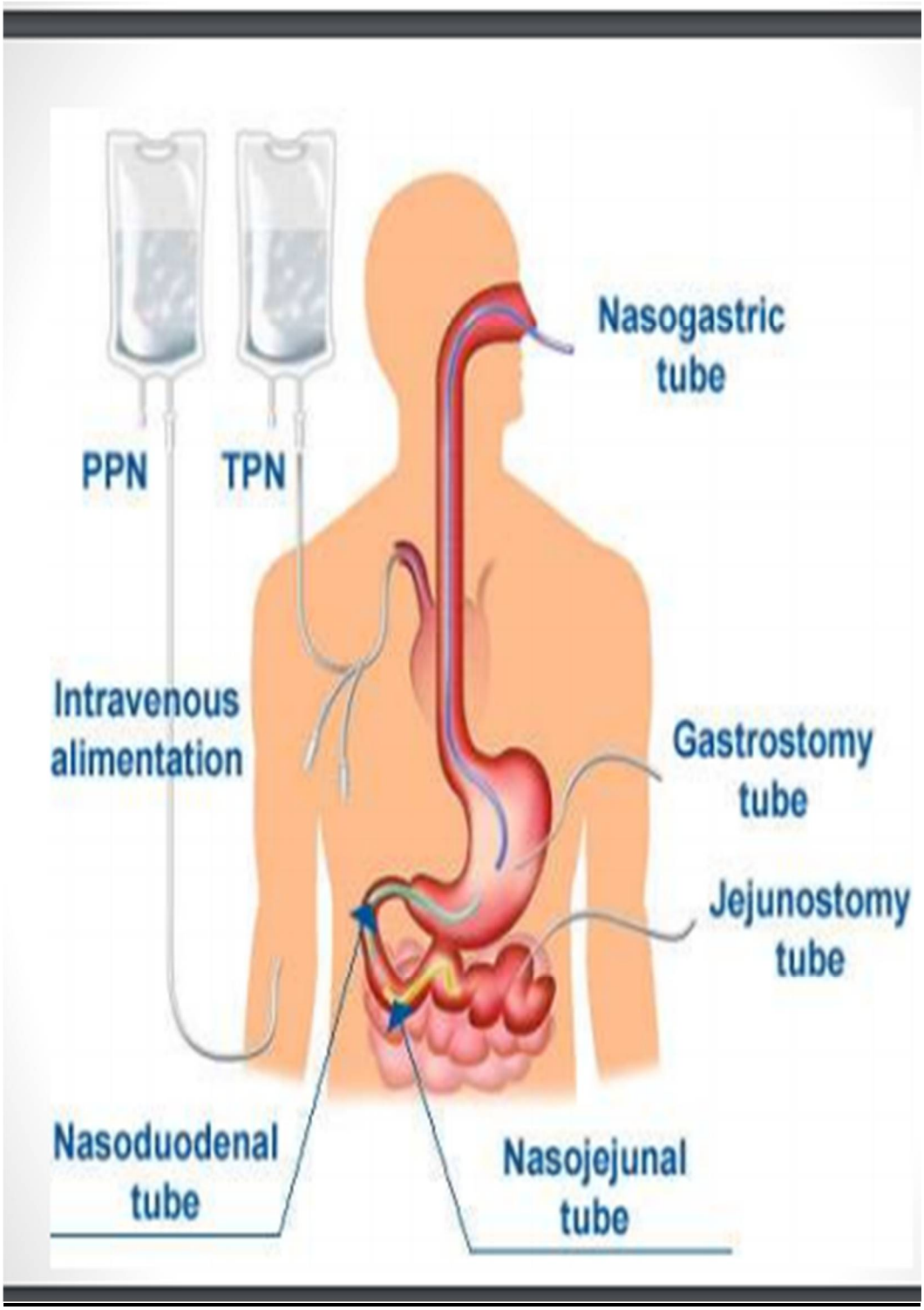
  Calories: Increase to 30-40 kcals/kg, Patient on ventilator usually require less calories ~20-25 kcal/kg

  Protein: Increase to 1-1.8 grams/kg

  Fluids: Individualized

METHODS OF NUTRITIONAL SUPPLEMENTATION:

- Use gastrointestinal tract if available
- Prolonged post-operative starvation has been found to have adverse outcomes in terms of post operative morbidity
- Early enteral nutrition is found to have reduced post-operative morbidity and better outcome.
- Enteral feeding reverses mucosal atrophy induced by starvation and it increases anastomotic collagen deposition and strength. Enteral feeding helps in wound healing. Early Enteral feeding also reduces septic morbidity after abdominal trauma and pancreatitis. Early Enteral feeding helps to increase the strength of the tissues.


Nasogastric tube
PPN
TPN
Intravenous alimentation
Gastrostomy tube
Jejunostomy tube
Nasoduodenal tube
Nasojejunal tube

ENTERAL FEEDING METHODS (20, 22, 23, 29)

• Enteral feeding prevents intestinal mucosal atrophy, supports gut associated immunological shield, attenuates hypermetabolic response to injury and surgery, cheaper than TPN and has fewer complications.

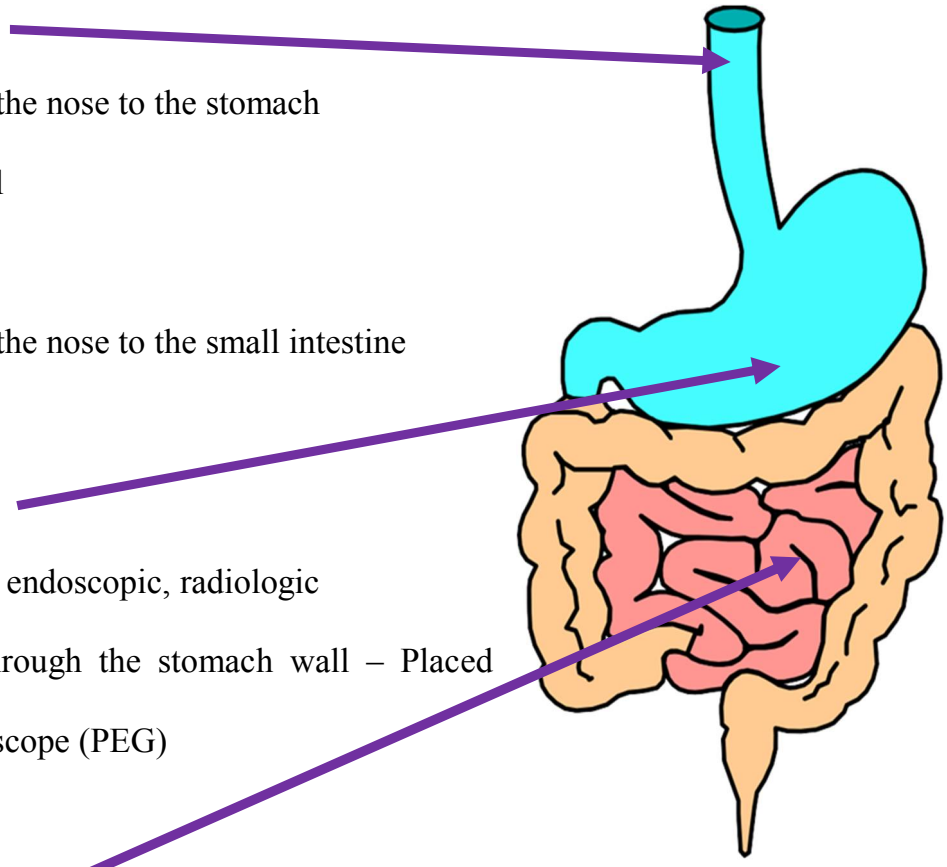
• ROUTES OF ADMINISTRATION :

- Nasogastric:

  Through the nose to the stomach

- Nasoduodenal
- Nasojejunal

  Through the nose to the small intestine



- Gastrostomy-

  percutaneous, endoscopic, radiologic

  Surgically: through the stomach wall – Placed with an Endoscope (PEG)

- Jejunostomy-

  percutaneous, endoscopic, radiologic

  Surgically: through the abdominal wall to the small intestine – Placed with an Endoscope (PEJ)

• Patient must be hemodynamically stable before starting eneteral nutrition.

The choice of route must be made; the least invasive ones are preferred.

Feeding Route Selection:

Short Term :

- Orogastric
- Nasoenteric: nasogastric – nasoduodenal – nasojejunal

Characteristics of nasoenteric tubes:

- Short-term feeding: < 6 - 8 weeks
- Usually small diameter
- Contraindications to nasoenteric tubes – significant GER – aspiration risk – delayed gastric emptying
- Potential complications include: – sinusitis – nasal erosion – tube displacement – aspiration (with GER)

NASOGASTRIC/NASOENTERIC FEEDING:

Can be given in Patients with intact mentation and protective laryngeal reflexes. Head end of the bed is raised to 35 degrees. Residual volumes should be checked 1 hour after meal and it should not exceed 50ml/hr. Signs of intolerance should be monitored and rate and osmolarity are adjusted accordingly.

Feeding Route Selection: Long-term

- Endoscopic or Radiologic: PEG – PEG-J – PEJ
- Surgical: Gastrostomy – Jejunostomy

Gastrostomy:

Characteristics: Appropriate for long-term feeding , Requires normal gastric emptying

Contraindications: – significant reflux or aspiration

May be placed by surgical, endoscopic or radiologic techniques

Potential complications include: infection, leakage, fistula ,buried bumper, perforation

Jejunostomy:

Characteristics : Appropriate for long-term feeding

Indicated for patients with aspiration or poor gastric emptying

May be placed by surgical, endoscopic or radiologic technique. Typically requires feeding pump

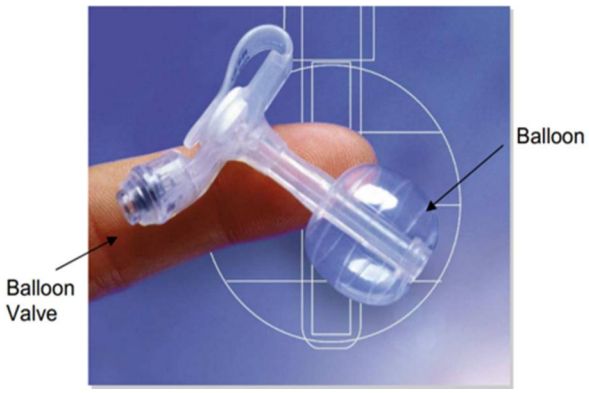
Potential complications include: infection, tube occlusion , intestinal ischemia, bowel obstruction

Low Profile Device:

Characteristics : Appropriate for long-term feeding

Beneficial for: active lifestyle , cosmetic purposes, agitated patient with risk for pulling out tube , possible replacement in the home

Potential complications include: balloon malfunction ,improper insertion , leakage



DEHP • Di(2-ethylhexyl) phthalate:

Plasticizer which softens PVC. It is found in feeding bags and tubes . It is associated with liver toxicity in animals. Lipids leach DEHP from PVC . Kids > risk than adults

	Gastric feeding	ejunal feeding
Solution used	Hypertonic or isotonic	sotonic
Infusion rate	Bolus or continuous	Continuous
Initiation of infusion	25-30mL/hr	
Increments	25-30mL/hr daily	
Intolerance	Vomiting	Distention, diarrhea, colic, eflux to NGT

ADVANTAGES OF ENTERAL FEEDING:

- It Provides the advantage of trophic feeding
- Maintains structural and functional support of intestinal mucosa by providing glutamine
- Preserving blood supply and promoting peristalsis
- Maintains integrity of intestinal mucosa
- Prevents bacterial translocation
- Cheap, easy to administer, safe.

Contraindications to Enteral Nutrition

- Intractable vomiting, diarrhea refractory to medical management
- Paralytic ileus
- Distal high-output intestinal fistulas (too distal to bypass with feeding tube)
- GI obstruction, ischemia
- Diffuse peritonitis
- Severe shock or hemodynamically instability
- Severe GI hemorrhage
- Severe short bowel syndrome (less than 100 cm of small bowel remaining)
- Severe GI malabsorption (e.g., enteral nutrition failed, as evidenced by progressive deterioration in nutritional status)
- Inability to gain access to GI tract
- Need is expected for <7 days

Formula selection is BASED ON:

Functional status of GI tract, Physical characteristics of formula (osmolality, fiber, content, caloric density, viscosity) , Macronutrient ratios, Digestion and absorption capability of patient, Specific metabolic needs, Contribution of the feeding to fluid and electrolyte needs, or restriction , Cost effectiveness

Enteral formulas:

1. Low residue isotonic formulas:

   Calorie density of 1 kcal/ml

   Non protein-calorie:nitrogen ratio =150:1

   No fibre, no bulk, no residue

   Cheap, first line for stable gi tract

2. Isotonic formula with fibre :

   Soluble and insoluble fibre

   Stimulate pancreatic lipase activity

   Degradation into short chain fatty acids

3. Immune enhancing formulas:

Glutamine, arginine, omega-3 fatty acids, nucleotides, beta carotene

4. Calorie dense formula: 2kcal/ml

5. High protein formula

6. Elemental formula: predigested nutrients,

Adv: ease of absorption in gut impairment, pancreatitis,

Disadv: poor in fat, vitamin, trace elements

High osmolarity, high cost

7. Special formulas: renal/pulmonary/hepatic failure patients

MONITORING SCHEDULE

PARAMETER	ACUTE PATIENT	STABLE PATIENT
Electrolytes	Daily	1-2×/week
CBC	Daily	1-2×/week
Glucose level	3×/day; more often if poor control	3×/day; less often if good control
RFT	Daily	Weekly or twice weekly
Nitrogen balance	Daily	2-3×/week
Input and output	Daily	2-3×/week
Body weight	Daily	2-3×/week
Urine output	Hourly	every 4 hours
Stool	Per motion	Daily

Complications:

- Local problems: epistaxis, sinustis, nasal necrosis
- Tube related: Malposition, Displacement, Blockage / Leakage / Breakage, Erosion of skin / mucosa
- Gastrointestinal: Diarrhoea , Bloating, Nausea, Vomiting , Abdominal cramps, Aspiration , Constipation
- Metabolic: Electrolyte disorder ,Vitamin, Mineral, Trace element deficiency ,Drug interactions
- Infection
- Aspiration: Overloading ,Supine position/ unconscious
- Gastroparesis: vomiting, aspiration
- REFEEDING SYNDROME:

  After prolonged fasting period leads to sudden rise in insulin and electrolyte abnormailities resp, hepatic and renal dysfunction. Rate of feeding should be slow at starting of feeds.

- **Solute overload:**

  Diarrhoea, dehydration, electrolyte disturbance, hyperglycemia,

  Loss of trace elements

  In severe cases, pneumatosis intestinalis with bowel necrosis and perforation

PROBLEM	COMMON CAUSES	MANAGEMENT
Diarrhea	**Medications (e.g., antibiotics, H_2blockers, laxatives, hyperosmotic, hypertonic solutions), feeding intolerance (osmolarity, fat), acquired lactase deficiency**	1. **Measure stool output.** 2. **Rule out infection (bacterial, viral, parasitic).** 3. **Supply fibre.** 4. **Change medication or formula.** 5. **Check osmolarity and infusion rate.** 6. **Administer antimotility medications (e.g., loperamide, codeine).**
Nausea and vomiting	**Delayed stomach emptying, constipation, abdominal distention, odor and appearance of formulations**	• **Administer feedings at room temperature.** • **Use isotonic formulations.** • **Use a closed system when possible.** • **Reduce doses of narcotics.** • **Use gastroprokinetic agents**
Constipation, fecal impaction	**Dehydration, lack or excess of fibre**	• **Monitor fluid balance daily.** • **Carry out rectal disimpaction.** • **Consider the use of cathartics, stool softeners, laxatives, or**
Aspiration pneumonitis	**Long-term supine position, delayed stomach emptying, altered mental status, malpositioned feeding tube, vomiting**	• **Place head of bed at 45 degrees during feedings.** • **Stop EN if gastric residual volume exceeds 200 mL.** • **Use nasoduodenal or nasojejunal tubes in patients at risk.**

Hyponatremia, overhydration	**Excess fluid intake, refeeding syndrome, organ failure (e.g., liver, heart, kidney)**	• **Monitor fluid balance and body weight daily.** • **Consider fluid restriction.** • **Change formula (avoid low-sodium intake).** • **Initiate diuretic therapy**
Hypernatremia	**Dehydration, inadequate fluid intake**	**1. Increase free water.**
Dehydration	**Diarrhea, inadequate fluid intake**	**1. Determine cause.** **2. Increase fluid intake.**
Hyperglycemia	**High content of carbohydrate in feedings, insulin resistance**	**1. Evaluate and adjust feeding formula.** **2. Consider insulin regimen.**
Hypokalemia, hypomagnesemia, hypophosphatemia	**Diarrhea, refeeding syndrome**	**1. Correct electrolyte abnormalities.** **Determine cause.** **2. Reduce rate if refeeding syndrome is present and monitor patient.**
Hyperkalemia	**Excess potassium intake, renal impairment**	**1. Change feeding formula.** **2. Reduce potassium intake.** **3. Consider insulin regimen.**

PARENTERAL FEEDING METHODS (25,26,27,28)

It is also called "total parenteral nutrition" or "hyperalimentation." It is a special liquid mixture given into the blood via a catheter in a vein. The mixture contains all the protein, carbohydrates, fat, vitamins, minerals, and other nutrients needed.

INDICATIONS FOR PARENTERAL NUTRITION:

- Malnourished patient expected to be unable to eat > 5-7 days *AND* enteral nutrition is contraindicated
- Patient failed enteral nutrition trial with appropriate tube placement (post-pyloric)
- Enteral nutrition is contraindicated or severe GI dysfunction is present:
    - Paralytic ileus, mesenteric ischemia, small bowel obstruction, enteric fistula distal to enteral access sites

TPN (TOTAL PARENTERAL NUTRITION)

- High glucose concentration (15%-25% final dextrose concentration)
- Provides a hyperosmolar formulation (1300-1800 mOsm/L)
- Must be delivered into a large-diameter vein through central line.

PPN (PERIPHERAL PARENTERAL NUTRITION)

- Similar nutrient components as TPN, but lower concentration (5%-10% final dextrose concentration)
- Osmolarity < 900 mOsm/L (maximum tolerated by a peripheral vein)
- May be delivered into a peripheral vein

- Because of lower concentration, large fluid volumes are needed to provide a comparable calorie and protein dose as TPN

IMMUNONUTRITION:

- Nutrients affecting the immune system.
- Recognised: arginine, glutamine, omega-3 fatty acids, nucleotides

Potential : vit c and e, selenium copper zinc, taurine, branched chain amino acids, n acetyl-cysteine

Parenteral Access Devices

Peripheral venous access

- Catheter placed percutaneously into a peripheral vessel

Central venous access (catheter tip in SVC)

- Percutaneous jugular, femoral, or subclavian catheter
- Implanted ports (surgically placed)
- PICC (peripherally inserted central catheter)

Venous Access Options for TPN

Short Term Access: Peripheral lines, Central venous catheters

Long Term Access: PICCs, Tunneled catheters, Implanted ports

Long Term Access

Non-Surgical: PICC- Peripherally Inserted Central Catheter

Surgical: Tunnelled Catheters: Hickman, Groshong, Broviac Port-A-Caths

Designing parenteral nutrition formula

- Total kilocalories (25-35 kcal/kg/day) 30 kcal/kg/day x 70 kg = 2100 kcal
- Protein (1.5gm/kg/day) 1.5kcal/kg/day x 70kg • = 105gm protein

2 in 1 solution

- 60 -70% dextrose, 10 to 20% amino acids
- Total kilocalories – 2100 kcal
- Calories for amino acids – 105gmx 4 kcal/gm = 420 kcal
- The difference 2100 – 420 = 1680kcal
- Dextrose 3.4kcal/gm so, 1680 x 3.4 = 494g dextrose

3 in 1 solution

- Includes 10 to 30% lipid emulsion
- Total kilocalories =2100kcal
- 20% of lipid , i.e. 2100 x 0.2 = 420kcal
- 9kcal/gm = 47 gm lipid
- Calories from aminoacid 105gmx 4 kcal/gm = 420 kcal
- Remaining calories = 2100- 420- 420 = 1260kcal
- 1260 kcal (3.4kcal/gm) = 370gm dextrose
- Fluid volume = amount of substance/ conc. of substance x 100

Final volume is

- Amino acid (10%) = 105gm = 1050 ml
- Dextrose (70%) = 370 gm = 528ml

- Lipids(20%) = 47gm = 235ml
- So total 1813ml/day

PARENTERAL NUTRITION REGIMEN

Solution	Volume (mls)	Energy (kcals)	Nitrogen (g)	Na (mmol)	K (mmol)	Ca (mmol)	PO4 (mmol)	Mg (mmol)
V amin 9 EF	1000		9.4					
Glucose 40%	500	800						
20% Intralipid	500	1000					7.65	
Addiphos	10			7.5	7.5		10	
15% KCl	20				40			
50% Mg SO4	2							4
Ca Cl	4					3.6		
30% NaCl	50			100				
Vitlipid + Solovito	10 each vial							
Additrace	10							
Requirements	2330	1900	9.5	108	48	3.6	19	3.8
Total	2116	1800	9.4	107.5	47.5	4.3	17.6	4

REQUIREMENTS:

- Energy = 8.1x45+656 =1020 + (153kcals)15% activity + (153kcals) 15 % stress + 500kcals = 1826kcals
- Nitrogen = 0.2g/kg = 9gN
- Fluid = 4 L (35mls/kg (1575mls) + losses 2.5L)
- Na 295mmol (1mmol / kg, GI losses 250mmol/L)
- K 45mmol (1mmol / kg)
- PO 22.5mmol (0.5-0.7mmol/kg)
- Mg 4.5mmol (0.1-0.2mmol/kg)
- Ca 4.5mmol (0.1-0.2mmol/kg)

MONITORING:

Parameter	**Frequency**	**Rationale**
Weight	Daily - weekly	Nutritional Status – fluid balance
Anthropometry	Fortnightly	Nutritional Status
Temperature	Daily	Infection
Line Site	Daily	Infection
Fluid Balance	Daily	Fluid / electrolyte requirement

- Check daily electrolytes and adjust TPN/PPN electrolyte additives accordingly
- Check glucose q 6 hours (regular insulin may be added to TPN/PPN bag for glucose control as needed)
    - Non-diabetics or NIDDM: start with half of the previous day's sliding scale insulin requirement in TPN/PPN bag and increase daily in the same manner until target glucose is reached
    - IDDM: start with 0.1 units regular insulin per gram of dextrose in TPN/PPN, then increase daily by half of the previous day's sliding scale insulin requirement

- Check triglyceride level within 24 hours of starting TPN/PPN
    - If TG >250-400 mg/dL, lipid infusion should be significantly reduced or discontinued
    - Consider adding carnitine 1 gram daily to TPN/PPN to improve lipid metabolism
    - ~100 grams fat per week is needed to prevent essential fatty acid deficiency
    - Check LFT's weekly
    - If LFT's significantly elevated as a result of TPN, then minimize lipids to < 1 g/kd/day and cycle TPN/PPN over 12 hours to rest the liver

- If Bilirubin > 5-10 mg/dL due to hepatic dysfunction, then discontinue trace elements due to potential for toxicity of manganese and copper
- Check pre-albumin weekly
- Adjust amino acid content of TPN/PPN to reach normal pre-albumin 18-35 mg/dL
- Adequate amino acids provided when there is an increase in pre-albumin of ~1 mg/dL per day

♦ Acid/base balance

- Adjust TPN/PPN anion concentration to maintain proper acid/base balance
- Increase/decrease chloride content as needed
- Since bicarbonate is unstable in TPN/PPN preparations, the precursor—acetate—is used; adjust acetate content as needed

COMPLICATIONS OF TPN: (36,37,38)

- First 48 hours: Mechanical – Malposition, Hemothorax,Pneumothorax, Air Embolism, Blood Loss, Puncture Of Subclavian Artery.

  Metabolic- Fluid Overload, Hyperglycemia,Hypophosphatemia, Hypokalemia, Hypomagnesemia, Refeeding Syndrome
- First Two Weeks: Mechanical: Catheter Displacement, Catheter Thrombosis, And Catheter Occlusion.

Metabolic: Hyperglycemia Coma, Acid Base Imbalance, Electrolyte Imbalance • Infection: Catheter Site Infection.

- 1 – 2 Months: Mechanical: Tear Of Catheter, Catheter Thrombosis, Blood Loss, and Air Embolism.

  Metabolic: Essential fatty Acid Deficiency, Vitamin Or Trace Element Deficiency, Metabolic Bone Disease, Liver Diseases • Infection: Tunnel Infection, Sepsis

SPECIFIC COMPLICATIONS:

- Hepatic steatosis
  - May occur within 1-2 weeks after starting PN
  - May be associated with fatty liver infiltration
  - Usually is benign, transient, and reversible in patients on short-term PN and typically resolves in 10-15 days
  - Limiting fat content of PN and cycling PN over 12 hours is needed to control steatosis in long-term PN patients
- Cholestasis
  - May occur 2-6 weeks after starting PN
  - Indicated by progressive increase in TBili and an elevated serum alkaline phosphatase
  - Occurs because there are no intestinal nutrients to stimulate hepatic bile flow
  - Trophic enteral feeding to stimulate the gallbladder can be helpful in reducing/preventing cholestasis

- Gastrointestinal atrophy
    - Lack of enteral stimulation is associated with villus hypoplasia, colonic mucosal atrophy, decreased gastric function, impaired GI immunity, bacterial overgrowth, and bacterial translocation
    - Trophic enteral feeding to minimize/prevent GI atrophy

TPN via right-SC line

2200 ml total volume x 24 hours

Amino acid: 45 g/liter = 45g x 2.2 L= 99 grams x 4 kcals/gram =369 kcals

Dextrose 175 g/liter = 175g x 2.2 L= 385 grams x 3.4 kcals/gram= 1309 kcals

Lipid 20% 285 ml over 24 hours = 285 mls x 2= 570 kcals

Above will provide 2275 kcal, 99g protein.

- Dextrose infusion rate should be < 4 mg/kg/minute (maximum tolerated by the liver) to prevent hepatic steatosis - DIR= (385 g dex/ 70 kg /1440 minute in a day)*1000= 3.8mg/kg/min
- Lipid infusion rate should be less than 1 g/kg/day to minimize/prevent TPN-induced liver dysfunction - LIR= (285 mls lipid * 20%)/ 70 kg=0.8 g/kg/day
- You may need to adjust/eliminate lipids if patient is on propofol. (1 ml propofol =1.1 kcal). Ex. Propofol @ 10 ml/hr would provide 264 kcals

- Initiate TPN at half of goal rate/concentration and gradually increase to goal over 2-3 days to optimize serum glucose control.

REFEEDING SYNDROME

- ♦ This is associated with metabolic and physiologic consequences of depletion, repletion, compartmental shifts, and interrelationships of phosphorus, potassium, and magnesium.
- ♦ There will be severe drop in serum electrolyte levels resulting from intracellular electrolyte movement when energy is provided after a period of starvation (usually > 7-10 days)

Physiologic and metabolic sequelae may include:

ECG changes, hypotension, arrhythmia, cardiac arrest, weakness, paralysis, Respiratory depression, Ketoacidosis / metabolic acidosis

Prevention and Therapy

Correct electrolyte abnormalities before starting nutrition support

Continue to monitor serum electrolytes after nutrition support begins and replete aggressively

Initiate nutrition support at low rate/concentration (~ 50% of estimated needs) and advance to goal slowly in patients who are at high risk.

CONSEQUENCES OF OVERFEEDING:

- Risks associated with over-feeding:
  - Hyperglycemia
  - Hepatic dysfunction from fatty infiltration
  - Respiratory acidosis from increased CO_2 production
  - Difficulty weaning from the ventilator
- Risks associated with under-feeding:
  - Depressed ventilatory drive
  - Decreased respiratory muscle function
  - Impaired immune function
  - Increased infection

♦ Parenteral nutriotion becomes immunosuppressive if
  - Poorly administered
  - Hyperglycemia
  - No nucleotides
  - No arginine
  - No taurine
  - Excessive fats

Conditions requiring careful monitoring of TPN

CONDITION	SUGGESTED CRITERIA
Hyperglycemia	Glucose >300 mg/dL
Azotemia	BUN >100 mg/dL
Hyperosmolality	Serum osmolality >350 mOsm/kg
Hypernatremia	Na >150 mEq/L
Hypokalemia	K <3 mEq/L
Hyperchloremic metabolic acidosis	Cl >115 mEq/L
Hypophosphatemia	Phosphorus <2 mg/dL
Hypochloremic metabolic alkalosis	Cl <85 mEq/L

ERAS PROTOCOL

Enhanced Recovery After Surgery concept proposes: "Patient-centered,evidence based,outcome driven,multidisciplinary team developed pathways for a surgical specialty and facility culture to maintain pre-operative organ function and reduce the profound stress response following surgery,optimize their physiologic function,and facilitate recovery"

Objectives of ERAS:

• Reducing complications and LOS

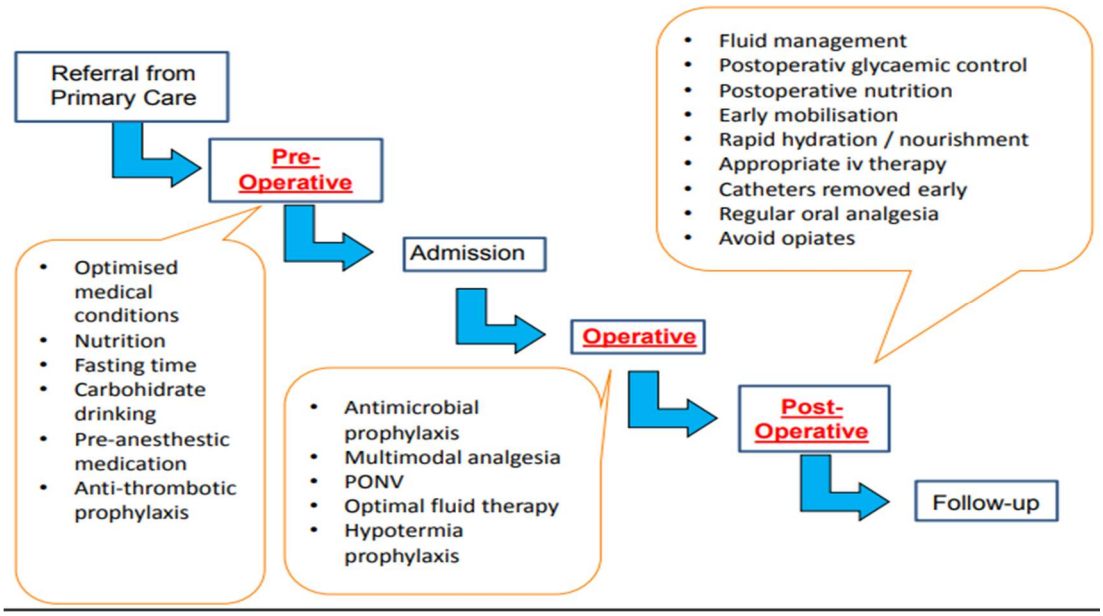
• Reducing variability

• Reducing cost

• Improving quality of care

• Increasing value = quality/cost

ENHANCED RECOVERY IN PRACTICE



PREOPERATIVE ERAS COMPONENTS:

• Health/medical optimisation

• Nutrition

• Fasting time

• Carbohydrate drinking

• Pre-anesthestic medication

• Anti-thrombotic prophylaxis

INTRAOPERATIVE ERAS COMPONENTS:

• Antimicrobial prophylaxis

• Anesthesia protocol

• PONV

• Fluid management

• Hypotermia prophylaxis

POSTOPERATIVE ERAS COMPONENTS:

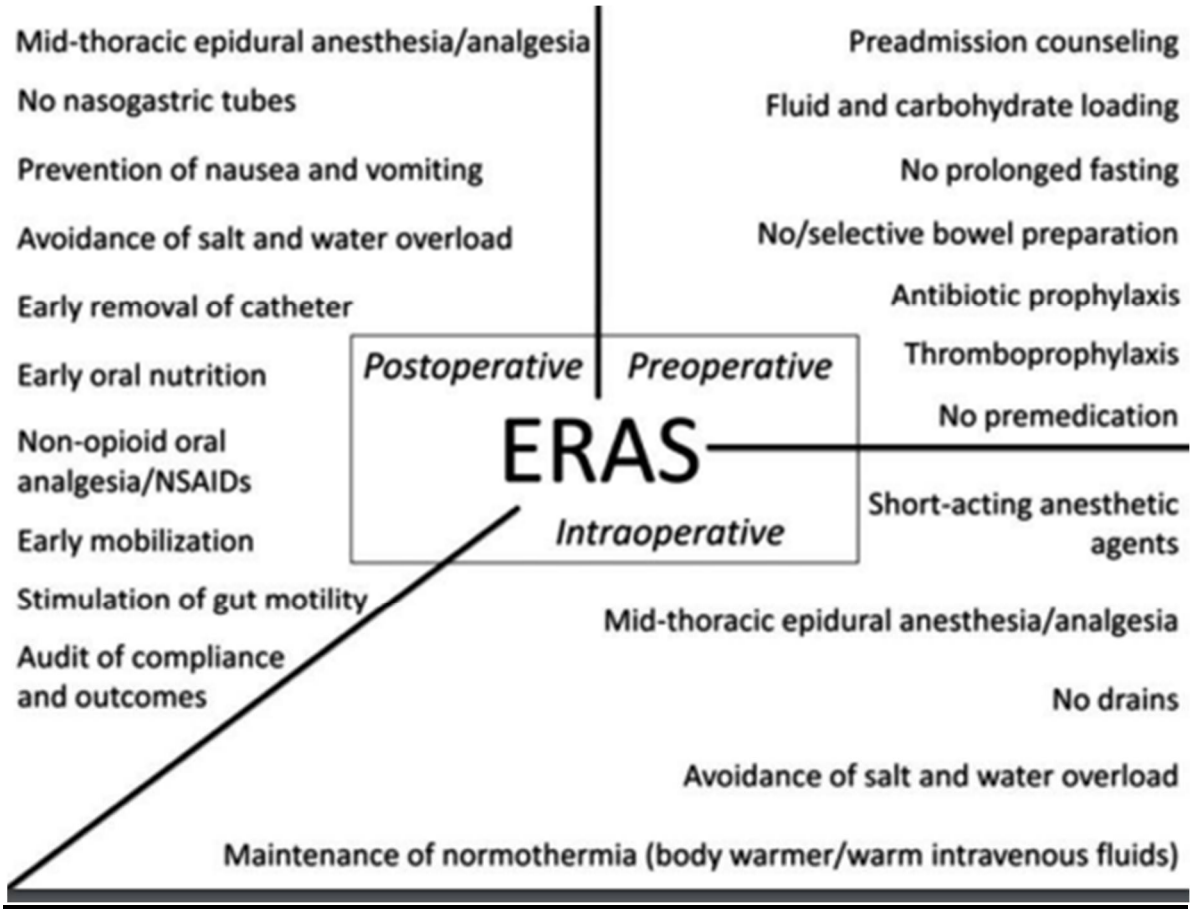
• Postoperative analgesia

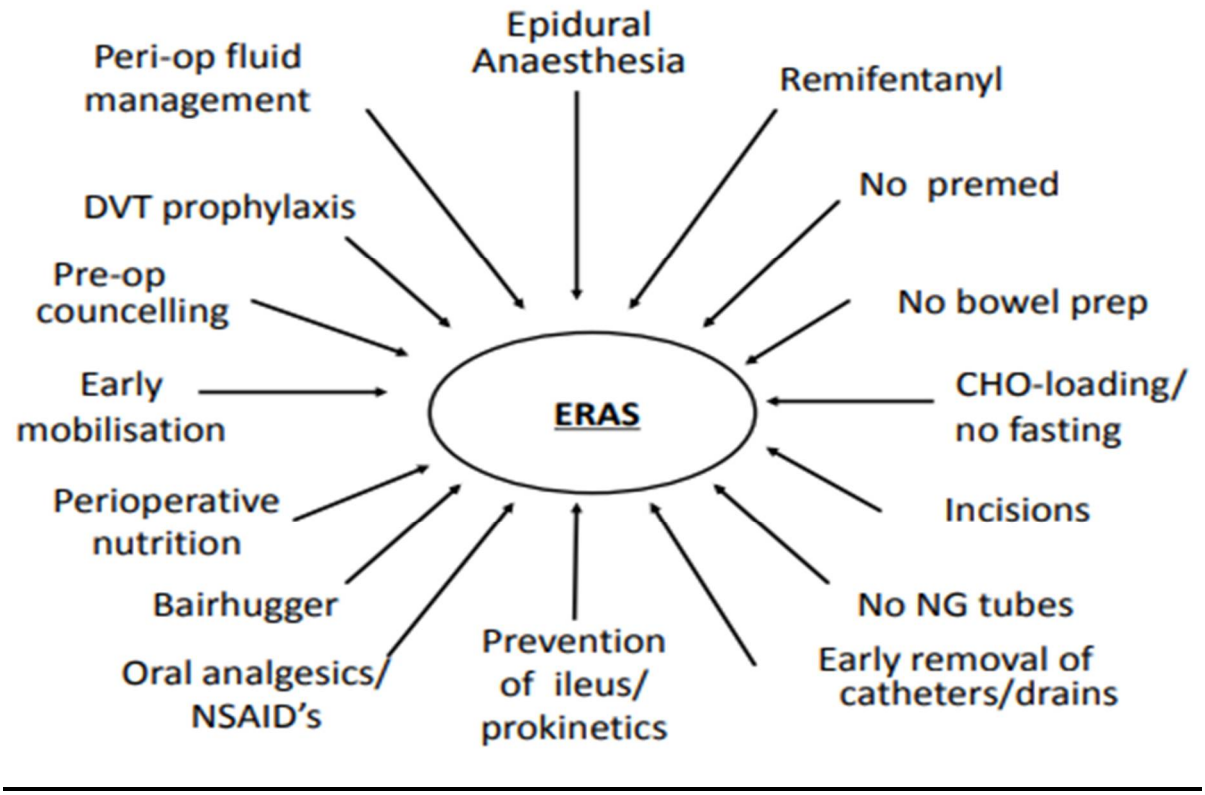
• Fluid management

• Postoperative glycaemic control

• Postoperative nutrition

• Early mobilisation


Mid-thoracic epidural anesthesia/analgesia
No nasogastric tubes
Prevention of nausea and vomiting
Avoidance of salt and water overload
Early removal of catheter
Early oral nutrition
Non-opioid oral analgesia/NSAIDs
Early mobilization
Stimulation of gut motility
Audit of compliance and outcomes
Postoperative
Preoperative
ERAS
Intraoperative
Preadmission counseling
Fluid and carbohydrate loading
No prolonged fasting
No/selective bowel preparation
Antibiotic prophylaxis
Thromboprophylaxis
No premedication
Short-acting anesthetic agents
Mid-thoracic epidural anesthesia/analgesia
No drains
Avoidance of salt and water overload
Maintenance of normothermia (body warmer/warm intravenous fluids)


Peri-op fluid management
Epidural Anaesthesia
Remifentanyl
DVT prophylaxis
No premed
Pre-op councelling
No bowel prep
Early mobilisation
ERAS
CHO-loading/ no fasting
Perioperative nutrition
Incisions
Bairhugger
No NG tubes
Oral analgesics/ NSAID's
Prevention of ileus/ prokinetics
Early removal of catheters/drains

OBSERVATION AND ANALYSIS

Table- 1: Distribution of Study Population according to Gender

Gender	Early enteral feeding		Late enteral feeding		Fisher exact Value	Significance
	No	%	No	%		
Male	21	84	22	88	0.166	0.6835
Female	4	16	3	12		

There is no difference in sex between the two groups

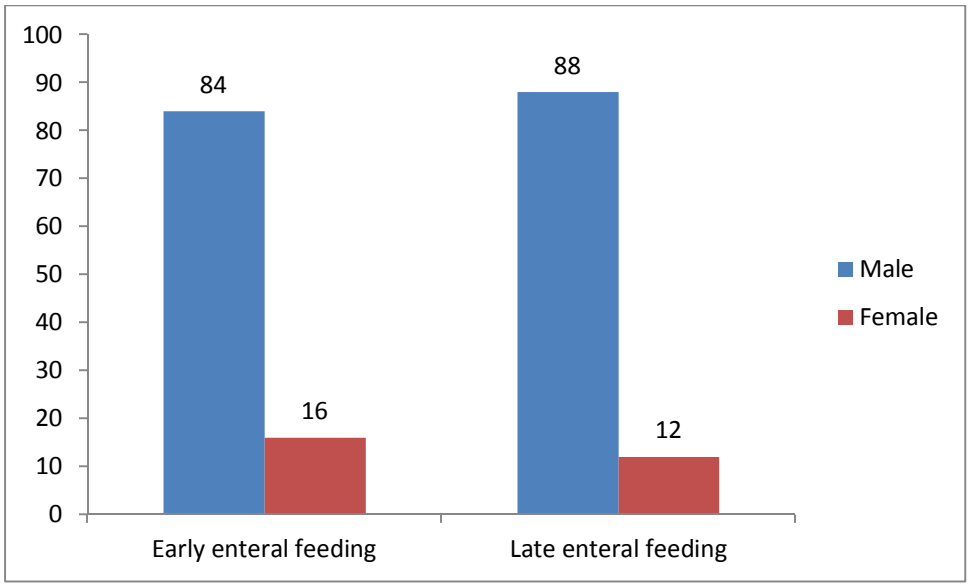


Table 2: Distribution of study population according to age group

Age group (yrs)	Early enteral feeding		Late enteral feeding		Fisher exact Value	Significance
	No	%	No	%		
<30	6	24	4	16		
31-40	8	32	3	12		
41-50	4	16	6	24	5.245	0.345
51-60	4	16	6	24		
61-70	3	12	4	16		
>70	0	0	2	8		

There is a statistically significant equal distribution of study population in both groups

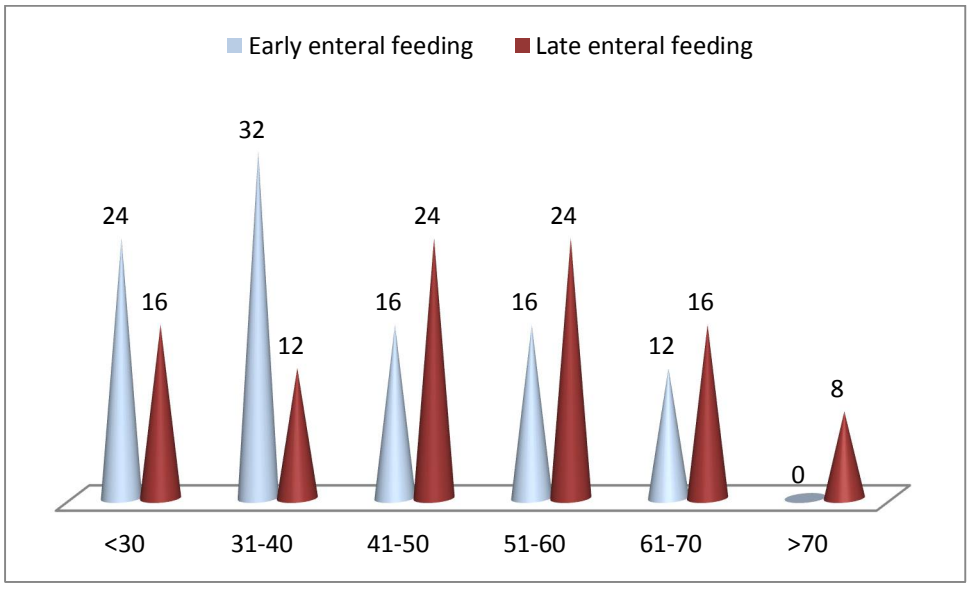
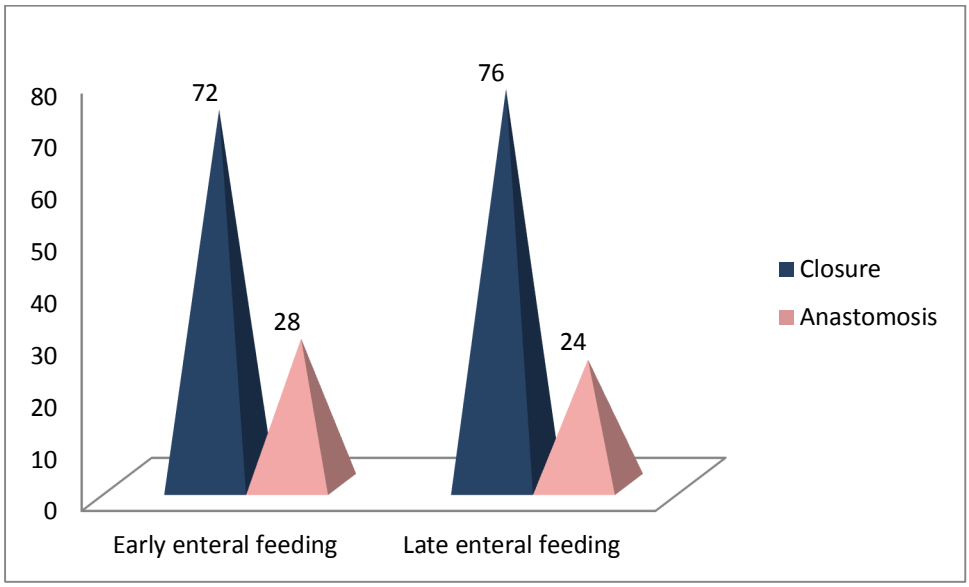


Table 3: Distribution of study population according to procedure done

Procedure done	Early enteral feeding		Late enteral feeding		Chisquare Value	Significance
	No	%	No	%		
Closure	18	72	19	76	0.104	0.74
Anastomosis	7	28	6	24		

Both the groups are more or less equal with respect to procedure done



From the last the tables we infer that there is no statistical significance between the study subjects with respect to age group. Gender and surgical procedure performed. Hence it's very clear that both the groups are comparable. In other words, the sampling technique has been followed perfectly so that both the groups are similar in composition before the start of the study enabling us to compare the difference in both groups effectively.

Table 3: Association between early enteral feeding and late enteral feeding groups with respect to Paralytic ileus

Paralytic ileus	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	22	88	9	36	14.346	0.00015
Present	3	12	16	64		

Only 12 % in the early enteral feeding group had paralytic ileus group compared to 64% in the late enteral feeding group, and the results are statistically significant.

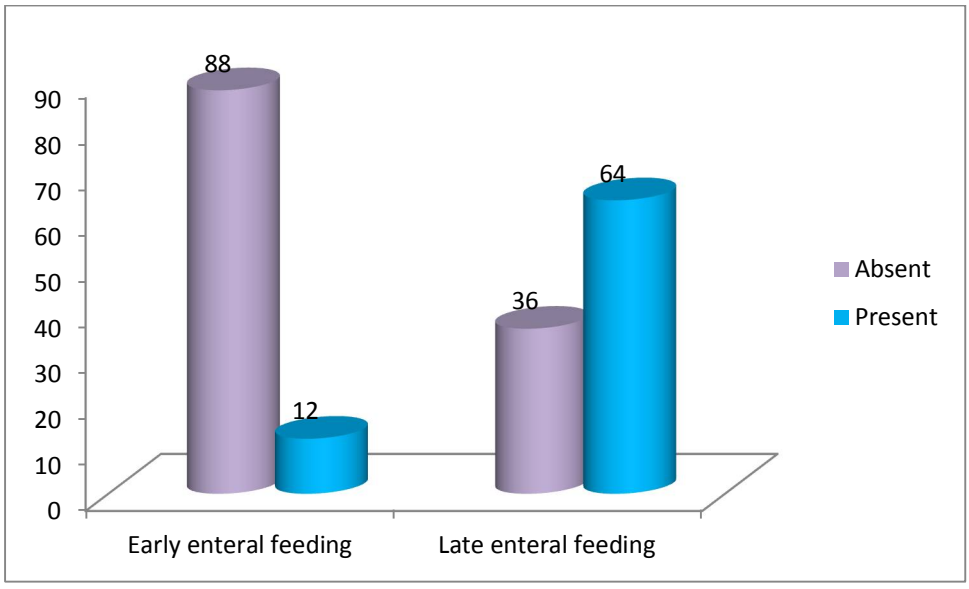


Table 4: Association between early enteral feeding and late enteral feeding groups with respect to wound infection

Wound infection	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	21	84	14	56	4.667	0.03075
Present	4	16	11	44		

Around 15 % in study group had wound infection while around 44 % of the control group had wound infection and the results are statistically significant.

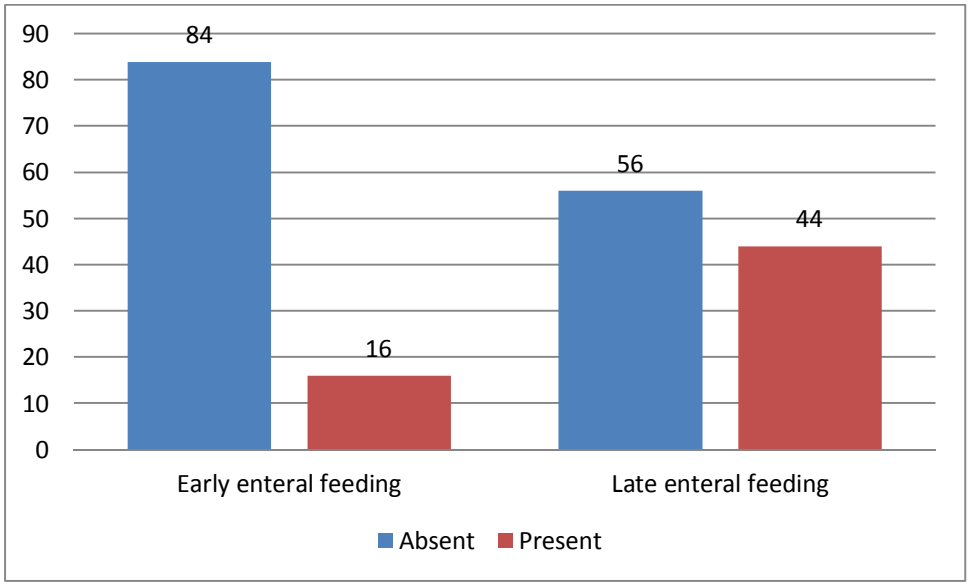


Table 5: Association between early enteral feeding and late enteral feeding groups with respect to wound dehiscence

Wound dehiscence	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	22	88	15	60	5.094	0.024
Present	3	12	10	40		

Around 12 % in study group had wound dehiscence while around 40 % of the control group had wound dehiscence and the results are statistically significant

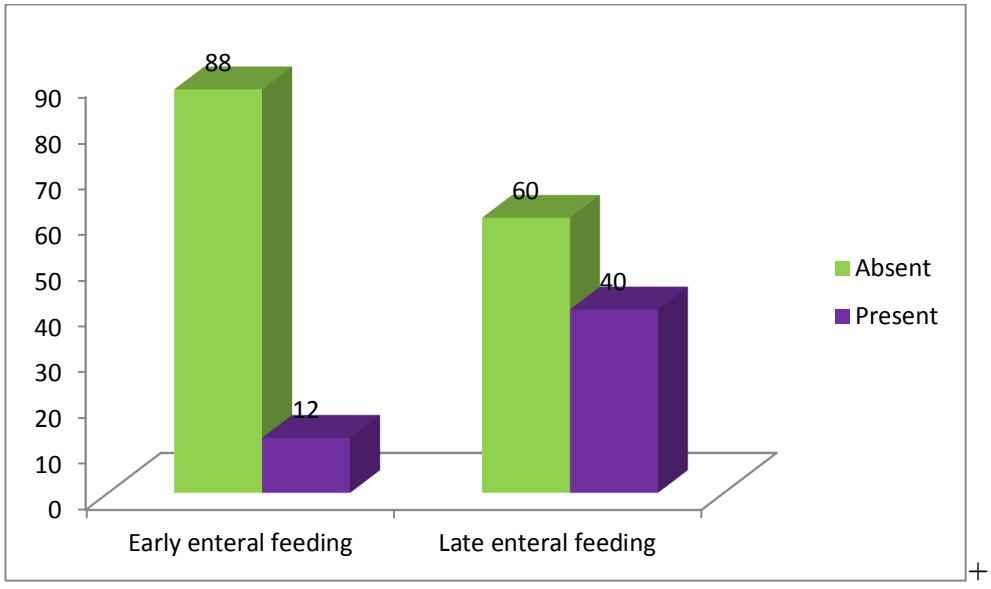


Table 6: Association between early enteral feeding and late enteral feeding groups with respect to Wound Leak

Wound Leak	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	24	96	19	76	4.153	0.042
Present	1	4	6	24		

Only 4 % in study group had wound infection compared to 24 % of the control group and the results are statistically significant

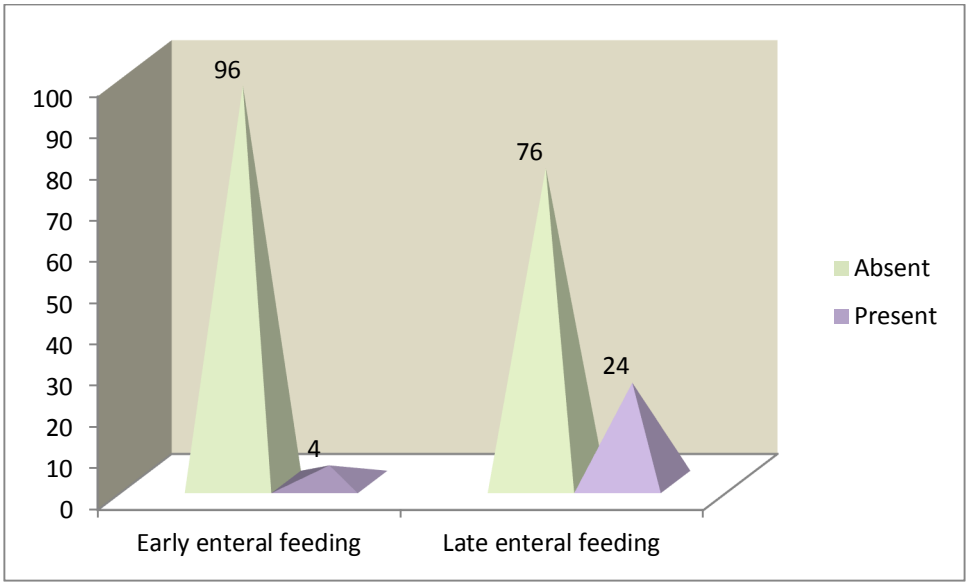


Table 7: Association between early enteral feeding and late enteral feeding groups with respect to Presence of Intra abdominal abscess

Intra abdominal abscess	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	24	96	17	68	6.64	0.0099
Present	1	4	8	31		

Intra abdominal abscess is very minimal in study group while 31% in control group had intra abdominal abscess. The results are statistically significant

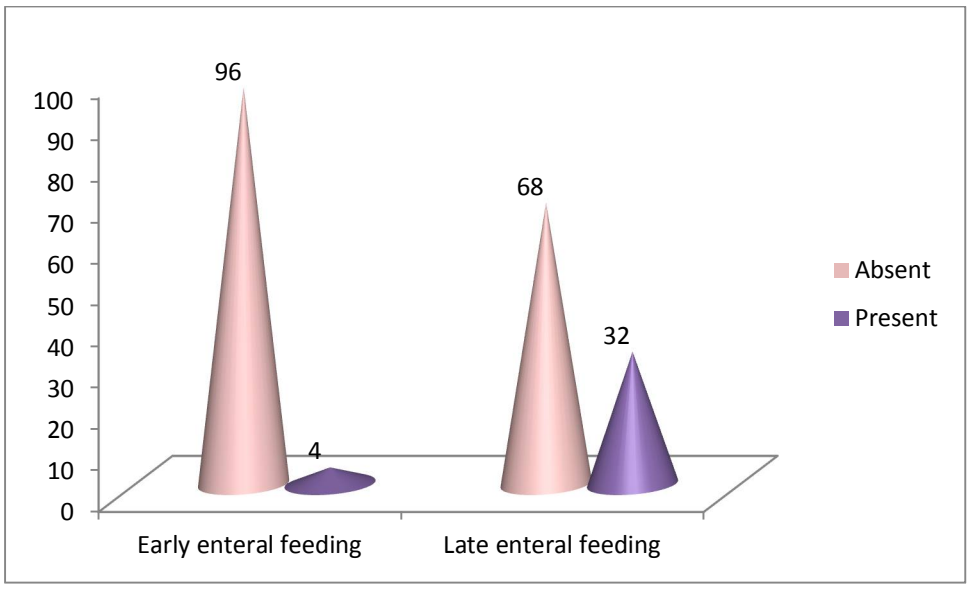
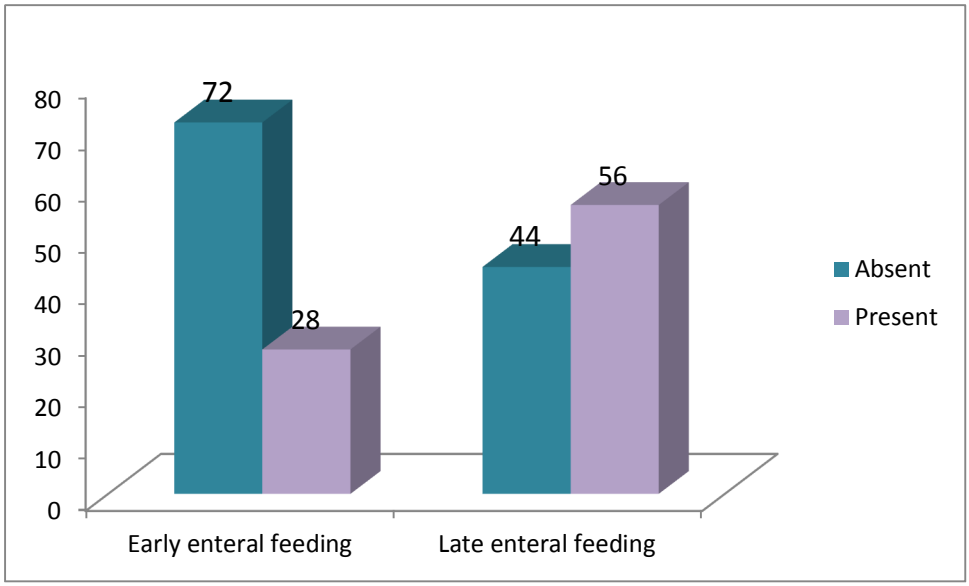


Table 8: Association between early enteral feeding and late enteral feeding groups with respect to occurrence of GI complications

GI complications	Early enteral feeding		Late enteral feeding		Chisquare exact Value	Significance
	No	%	No	%		
Absent	18	72	11	44	4.023	0.044
Present	7	28	14	56		

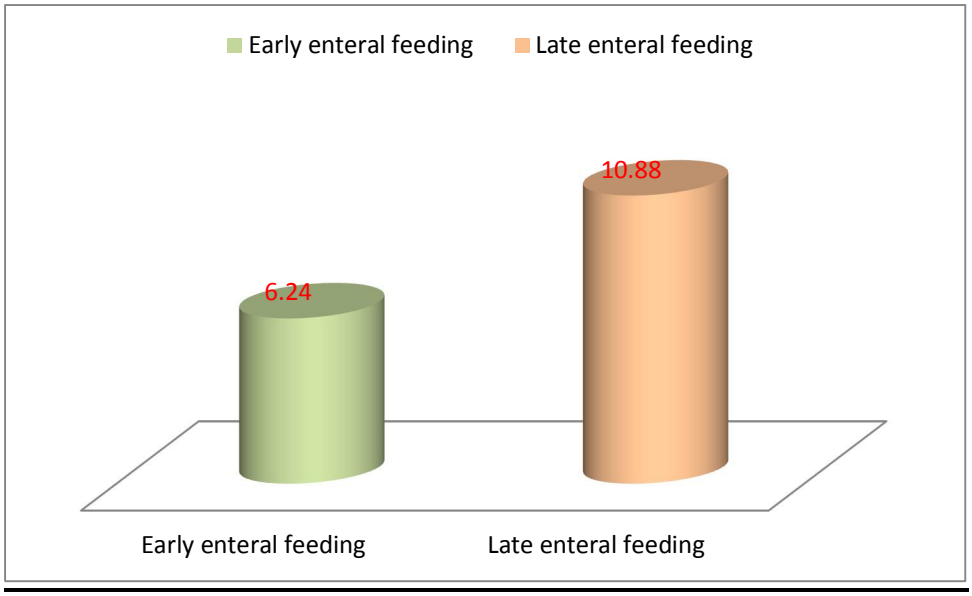
There is a statistically significant difference between study and control group (28% vs. 56% respectively) with respect to occurrence of GI complications



Comparison of mean length of hospital stays between both the groups

Length of stay(days)	Early enteral feeding		Difference in Mean	Significance
	Mean	SD		
Early enteral feeding	6.24	1.2	7.553	0.008
Late enteral feeding	10.88	2.24		

Early enteral feeding group have a lesser duration of hospital stay compared to late enteral feeding group(6 days vs 10 days) and the results are statistically significant



DISCUSSION

Damage to the body tissues by surgery induces a stress responses characterized by hyper metabolism, impaired protein synthesis, and catabolism. Patients who have undergone surgery might have an underlying disease, such as typhoid fever, tuberculosis, peptic ulcer, malignancy which affects his nutritional status adversely. The patient is thus exposed to a very high risk of morbidity and mortality. The decreased whole body protein synthesis and increased catabolism results in a net protein loss. This can quickly cause protein calorie malnutrition, which is associated with the organ dysfunction. A subclinical multiple organ dysfunction syndromes evolve, which increases the patient's risk of septic complications. Acute protein malnutrition is also known to adversely affect both humoral and cell mediated immunity. Traditional practice has been to delay nutritional support until the gut has resumed the peristaltic activity. If, at this stage a complication develops and oral intake is not possible, total parenteral nutrition (TPN) is initiated. This practice does not, in theory, affect the critical period of protein loss and the development of organ dysfunction. The gut is the major interface between the host and environment and is metabolically and immunologically active in the stressed patient. Luminal nutrients are important for intestinal mucosal metabolism and integrity, regardless of the general nutritional status. The enteral nutrition stimulates gall bladder contraction, maintains gut the associated lymphoid tissue, and stimulates immune function and the pancreatic secretions. It also maintains gut integrity, as evidenced by a decreased risk of perforation and

better healing of gut anastomosis. Except for the depression of cellular immunity, the bacteria translocation is the most probable reason of infection after the operation.

Keeping the integrity of gut barrier is important to decrease the morbidity and mortality after operation. Enteral nutrition could protect the gut barrier remarkably, the rational mechanisms were:

1) The stimulation on the bowel wall may increase the blood perfusion.

2) The stimulation on the bowel wall may accelerate the secretion of pancreas and biliary duct to prevent shrinking of gut mucosa.

3) Enteral nutrition supplies the substrate of intestinal mucosal cell metabolism directly.

The stomach, even when not stimulated by oral intake, secretes 0.5 to 1 litre of gastric juice each day. This in turn stimulates secretion of another 0.5 to 1 litre of pancreatic juice. Thus, one to two litres of fluid are presented to the small bowel each day, even though the patient is taking no fluids orally. Most of this is readily absorbed by the small intestine, and very little reaches the colon. This shows that even oral nutrition can be handled by the gut. If one examines the natural history of postoperative ileus, neither the presence of bowel sound nor even passage of flatus determines if the patients will tolerate the oral intake. Therefore, it is not rational that these be the criteria used to decide when to feed a postoperative patient. Recent research in the area of gastrointestinal physiology and motility further indicates that current criteria

for determining when to feed postoperative patients are not rationally based. The myoelectric and motor activity of the stomach, as evaluated by electrogastrography and impedance gastrography is not grossly disturbed by intra-abdominal surgery.

CONCLUSION

Out of a total of 50 patients, that were included in the study, 25 patients were in the early enterally fed group (study group) and the remaining 25 patients were in the late enterally fed group (control group).

In our study, it was found that the incidence of paralytic ileus, wound infection, wound dehiscence, and pain in post operative patients undergoing major gastrointestinal surgeries, thereby reducing length of hospital stay was significantly less in the study group as compared to the control group.

The average length of hospital stay in the study group was 6 days while that in the control 10 days and the results are statistically significant.Finally, there was no death in both groups.

From the above results it is concluded that early feeding is feasible, safe and well tolerated by patients undergoing gastro intestinal surgery. It is also associated with very good postoperative outcome and early recovery of the patients.

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PROFORMA FOR STUDY ON EARLY VERSUS LATE ENTERAL IN PATIENTS OF MAJOR GASTROINTESTINAL SURGERIES

DR. YUVARAJ.S

Surgical Unit: I

Coimbatore Medical College

Coimbatore.

NAME:	AGE:	UNIT:	DOA:
ADDRESS:	SEX:	WARD:	DOS:
			DOD:
OCCUPATION:	SOCIOECONOMIC STATUS:		

SOCIO ECONOMICAL STATUS

COMPLAINTS/HISTORY

PAIN

SITE	**RADIATION TO**	**AGRAVATED BY**	**RELIEVED BY**
SHOULDER		COUGH	VOMITING
GROIN		MOVEMENT	LYING STILL
LUMBER		FOOD	FOOD
R.LOIN			ANTACIDS
L.LOIN			MILK
NIL			
OTHER			

ONSET

PROGRESS	**DURATION**	**TYPE**	**SEVERITY**
NO CHANGE	CONTINUOUS	MILD	
BETTER	PRESENTATION	COLICKY	MODERATE
WORSE		SHARP	SEVERE

SYMPTOMS

NAUSEA/VOMITING: WEIGHT LOSS:

ANOREXIA/DYSPEPSIA

MICTURATION:

FEVER: BOWELS:

JAUNDICE: OTHERS:

PAST HISTORY

SIMILAR COMPLAINTS: DIABETES:

SURGERY: T.B:

MAJJOR ILLNESS:

HYPERTENSION:

OTHERS:

PERSONAL HISTORY

VEG/NON-VEG/MIXED FOOD:

MENSTURATION:

SMOKER:

MARRIED/UNMARRIED:

ALCOHOL: DRUG

INTAKE:

OTHERS:

GENERAL EXAMINATION

BUILT:

TEMPERATURE:

NUTRITION: PULSE:

HYDRATION:

RESPIRATION:

ANAEMIA: B.P:

JAUNDICE: OTHERS:

PEDAL OEDEMA:

EXAMINATION OF OTHER SYSTEMS

C.V.S:

R.S:

C.N.S:

OTHERS:

EXAMINATION OF ABDOMEN

INSPECTION

DISTENSION: SCAR:

MOVEMENT WITH RESP: INJURY:

UMBILICUS: OTHERS:

PALPATION

TEMP: MASS:

TENDERNESS: LIVER:

GUARDING:

SPLEEEN:

RIGIDITY: HERNIA:

OTHERS:

SCRUTUM:

PERCUSSION

LIVER DULLINESS: OTHERS:

SHIFTING DULLINESS:

FLUID THRILL:

AUSCULTAION: BOWEL SOUNDS:

PERRECTAL EXAMINATION

INVESTIGATION

URINE:	**BLOOD:**
ALBUMIN:	H.B %:
SUGAR:	
SUGAR:	T.C.:
UREA:	
DEPOSISTS:	D.C.:
WIDAL:	
SERUM:	PERITONEAL FLUID
CULTURE:	
CREATININE:	ANALYSIS:
AMYLASE:	C/S :

RADIOLOGY

PLAIN XRAY – ABDOMEN ERECT:

CHEST P.A.VIEW

ULTRA SOUND ABDOMEN

CT ABDOMEN AND PELVIS

DIAGNOSIS:

TREATMENT

RESUSCITATION

ANALGESIC:

BLADDER CATHETERISATION

IV F

ANTIBIOTIC

NASOGASTRIC TUBE

OTHERS

CONSERVATIVE:

PROCEDURES:

LAPAROTOMY:

SIMPLE CLOSURE:

DEFINTIVE PROCEDURE:

OMENTAL PATCH:

OTHERS:

RESECTION/ANASTAMOSIS:

BY PASS:

COLOSTOMY:

FINDINGS:

SUTURE

MATERIALS USED

SITE OF PERFORATION:

SIZE OF PERFORATION:

SITE AND EXTENT OF GANGRENE:

DRAIN:

ASSOCIATED FINDINGS:

OTHERS:

POSTOP

NASOGASTRIC SUCTION:

IV F:

INPUT/OUTPUT CHART:

ANTIBIOTIC:

ANALGESIS/SEDATION:

TIMING OF FIRST ENTERAL FEEDING: **COMPLICATIONS**

TYPE OF FEED: **DRAIN REMOVED:**

TOLERANCE TO ENTERAL FEEDING: **PARALYTIC ILEUS:**

ADVANCEMENT TO SEMISOLID DIET:

_____

_____

ON DISCHARGE:

_____

FOLLOW UP:

CONSENT FORM

I_____, do hereby volunteer and consent to the participate in this study being conducted by Dr. YUVARAJ.S on "**A PROSPECTIVE COMPARATIVE STUDY OF EARLY VERSUS LATE ENTERAL FEEDING AFTER MAJOR GASTROINTESTINAL SURGERIES**". I have read and understood the consent form or it has been read and explained to me in my own native langue in my mother tongue. The study has been fully explained to me and I may ask questions at any time during the study period.

Signature/Left Thumb impression of the Volunteer Date:

Place:

Signature and Name of Witness: Date:

Place:

Signature of the investigator:

Name of the investigator:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி:

கோவை அரசு மருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர் **ச.யுவராஜ்** தலைமையில் நடைபெறும் இந்த ஆய்வில் முழு சம்மதத்துடன் கலந்துகொள்ள சம்மதிக்கிறேன் .இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிட ஆட்சேபணை இல்லை என்று தெரிவித்துக் கொள்கிறேன் .எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக்கொள்ளும் உரிமை உண்டு என்று அறிவேன் .

இடம் :

தேதி:

STUDY GROUP

SL.NO	NAME	AGE/SEX	DIAGNOSIS	PROCEDURE	PARALYTIC ILEUS	WOUND INFECTION	WOUND DEHISCENSE	LEAK	INTRA ABDOMINAL ABSCESS	GI COMPLICATIONS	LENGTH OF HOSPITAL STAY
1	AYYAPPAN	54/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
2	SAKUNTHALA	45/F	ACUTE INTESTINAL OBSTRUCTION WITH ILEAL GANGRENE	RESECTION AND ILEO-COLIC ANASTOMOSIS	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	NO	6
3	VIKASH	35/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
4	VIJAYAKUMAR	38/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	5
5	KALIMUTHU	48/M	ACUTE INTESTINAL OBSTRUCTION WITH COLO-COLIC INTUSSUSCEPTION	RIGHT HEMICOLECTOMY WITH ILEO-COLIC ANASTOMOSIS	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	7
6	MUNIYANDI	30/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	6
7	MERCY	36/F	GASTRIC OUTLET OBSTRUCTION	GASTROJEJUNOSTOMY	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	7
8	MURUGAN	42/M	ACUTE INTESTINAL OBSTRUCTION WITH JEJUNAL GANGRENE	RESECTION AND ANASTOMOSIS	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	7
9	SHANKAR	40/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
10	SUNIP	25/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
11	PALANISAMY	55/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	5
12	MANICKAM	70/M	BLUNT INJURY ABDOMEN WITH JEJUNAL PERFORATION	PRIMARY PERFORATION CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	8
13	MAARAN	70/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	6
14	JAYA	40/F	OBSTRUCTED UMBILICAL HERNIA WITH ILEAL GANGRENE	ILEAL RESECTION AND ANASTOMOSIS	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	NO	7
15	BISHAL KUMAR	18/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
16	DINESH KUMAR	35/M	PERFORATIVE PERITONITIS WITH ILEAL PERFORATION	PRIMARY PERFORATION CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	5
17	GOPAL	62/M	ACUTE INTESTINAL OBSTRUCTION WITH ILEAL STRICTURE	ILEAL RESECTION AND ANASTOMOSIS	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	NO	8
18	MOHAN	23/M	PENETRATING INJURY ABDOMEN WITH JEJUNAL PERFORATION	PRIMARY PERFORATION CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	6
19	MURUGAN	40/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	7
20	ANIL	39/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	5
21	MURUGESAN	48/M	PERFORATIVE PERITONITIS WITH JEJUNAL PERFORATION	PRIMARY PERFORATION CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	7
22	SIBIRAJ	18/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	6
23	BACKIYARAJ	30/M	PERFORATIVE PERITONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	6
24	GOVINDASAMY	54/M	PERFORATIVE PERITONITIS DUE TO GASTRIC PERFORATION	OMENTAL PATCH CLOSURE AND FEEDING JEJUNOSTOMY	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	NO	8
25	PARVATHY	60/F	CARCINOMA STOMACH	DISTAL PARTIAL GASTRECTOMY WITH GASTRO-JEJUNOSTOMY	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	YES	9

			CONTROL GROUP								
S.NO.	NAME	AGE/SEX	**DIAGNOSIS**	**PROCEDURE**	**PARALYTIC ILEUS**	**WOUND INFECTION**	**WOUND DEHISCENSE**	**LEAK**	**INTRA ABDOMINAL ABSCESS**	**GI COMPLICATIONS**	**LENGTH OF HOSPITAL STAY**
1	KANNAN	46/M	PERFORATIVE PERTONITIS (GASTRIC)	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	YES	8
2	RAMAN	65/M	PERFORATIVE PERTONITIS (JEJUNAL PERFORATION)	PRIMARY PERFORATION CLOSURE	PRESENT	PRESENT	ABSENT	ABSENT	NO	NO	10
3	CHINNAKAALAI	65/M	SMALL BOWEL ISCHEMIA WITH JEJUNAL GANGRENE	JEJUNAL RESECTION AND ANASTOMOSIS	PRESENT	PRESENT	PRESENT	PRESENT	YES	YES	12
4	JEYASUMAN	23/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	8
5	PREMKUMAR	60/M	PERFORATIVE PERTONITIS (GASTRIC)	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	ABSENT	NO	NO	9
6	RANGAMMAL	78/F	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	PRESENT	PRESENT	ABSENT	NO	YES	14
7	MANIKANDAN	26/M	CA. ASCENDING COLON	RIGHT HEMICOLECTOMY WITH ILEO COLIC ANASTOMOSIS	PRESENT	ABSENT	ABSENT	ABSENT	YES	YES	10
8	KRISHNAN	72/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	ABSENT	NO	NO	11
9	BATHRAPPAN	62/M	PERFORATIVE PERTONITIS (GASTRIC)	OMENTAL PATCH CLOSURE	PRESENT	PRESENT	PRESENT	PRESENT	NO	YES	10
10	CHINNASAMY	38/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	YES	8
11	PALANISAMY	55/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	ABSENT	NO	NO	10
12	MANI	47/M	PERFORATIVE PERTONITIS (JEJUNAL PERFORATION)	JEJUNAL RESECTION AND ANASTOMOSIS	PRESENT	PRESENT	PRESENT	PRESENT	NO	YES	12
13	SELVARAJ	52/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	ABSENT	NO	NO	8
14	MURUGAN	46/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	PRESENT	NO	NO	9
15	KANAGARAJ	61/M	CARCINOMA STOMACH	SUBTOTAL GASTRECTOMY + GJ	PRESENT	PRESENT	PRESENT	ABSENT	YES	YES	16
16	SOLAIAMMAL	29/F	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	YES	12
17	CHINNARAJ	55/M	PERFORATIVE PERTONITIS (GASTRIC)	OMENTAL PATCH CLOSURE	PRESENT	PRESENT	ABSENT	ABSENT	NO	NO	10
18	THANGAVEL	37/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	ABSENT	NO	YES	11
19	RANGAN	55/M	GOO WITH CICATERIZED DU	POSTERIOR GJ WITH TRUNCAL VAGOTOMY	ABSENT	PRESENT	PRESENT	ABSENT	NO	NO	13
20	SUBBULAKSHMI	46/F	PERFORATIVE PERTONITIS (JEJUNAL PERFORATION)	PRIMARY PERFORATION CLOSURE	PRESENT	PRESENT	PRESENT	PRESENT	YES	YES	15
21	HARIRAJ	24/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	NO	12
22	DURAISAMY	32/M	BLUNT INJURY ABDOMEN/ILEAL PERFORATION	ILEAL RESECTION AND ANASTOMOSIS	PRESENT	PRESENT	PRESENT	ABSENT	YES	YES	14
23	NAGARJUNA	42/M	PERFORATIVE PERTONITIS(GASTRIC)	OMENTAL PATCH CLOSURE	PRESENT	ABSENT	ABSENT	PRESENT	NO	YES	10
24	SUBRAMANI	58/M	PERFORATIVE PERTONITIS(JEJUNAL PERFORATION)	PRIMARY PERFORATION CLOSURE	PRESENT	PRESENT	ABSENT	ABSENT	NO	NO	11
25	DHANDAPANI	43/M	PERFORATIVE PERTONITIS	OMENTAL PATCH CLOSURE	ABSENT	ABSENT	ABSENT	ABSENT	NO	YES	9