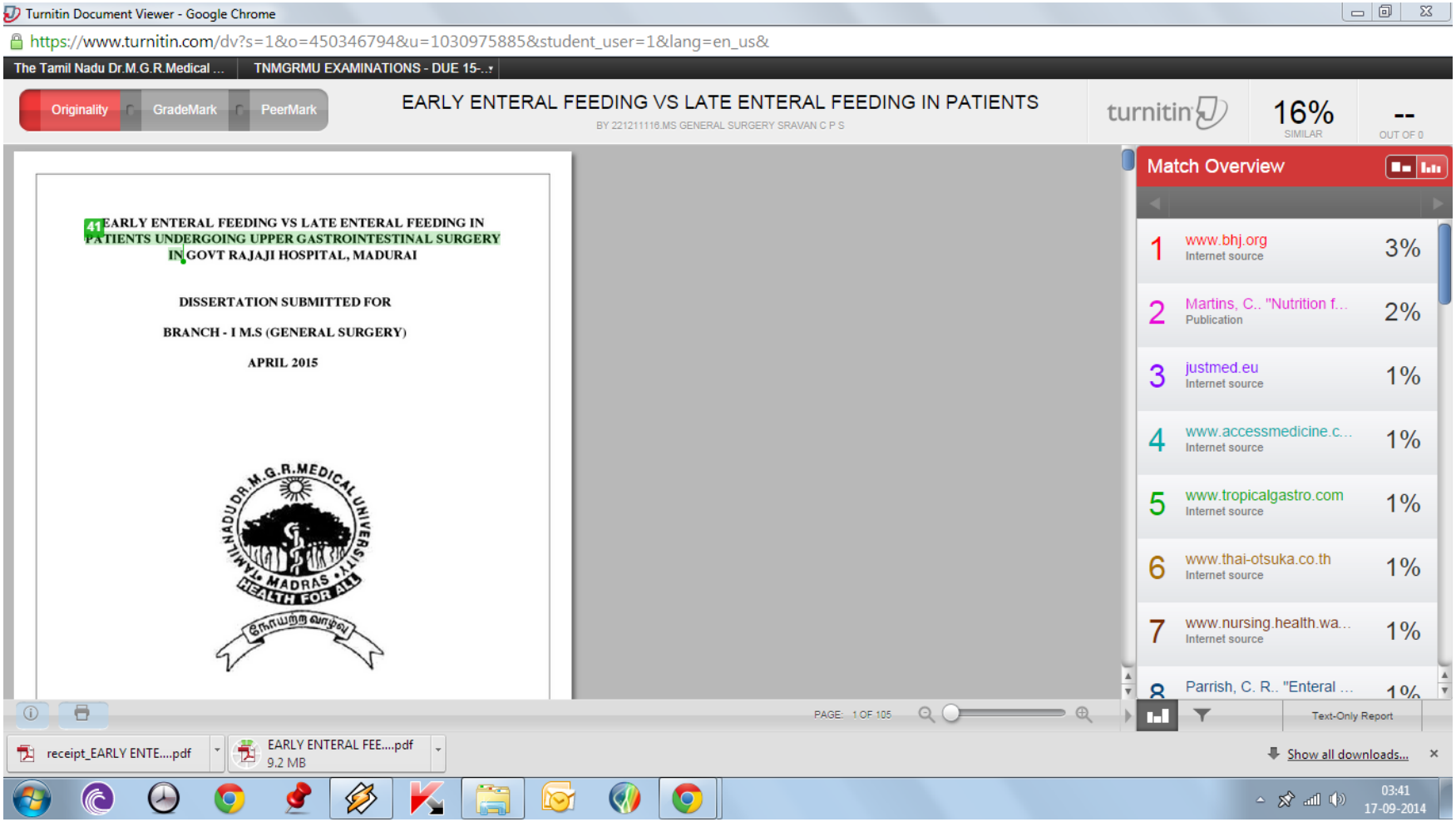
EARLY ENTERAL FEEDING VS LATE ENTERAL FEEDING IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL SURGERY IN GOVT RAJAJI HOSPITAL, MADURAI

DISSERTATION SUBMITTED FOR

BRANCH - I M.S (GENERAL SURGERY)

APRIL 2015

THE TAMILNADU DR.M.G.R.MEDICALUNIVERSITY
CHENNAI

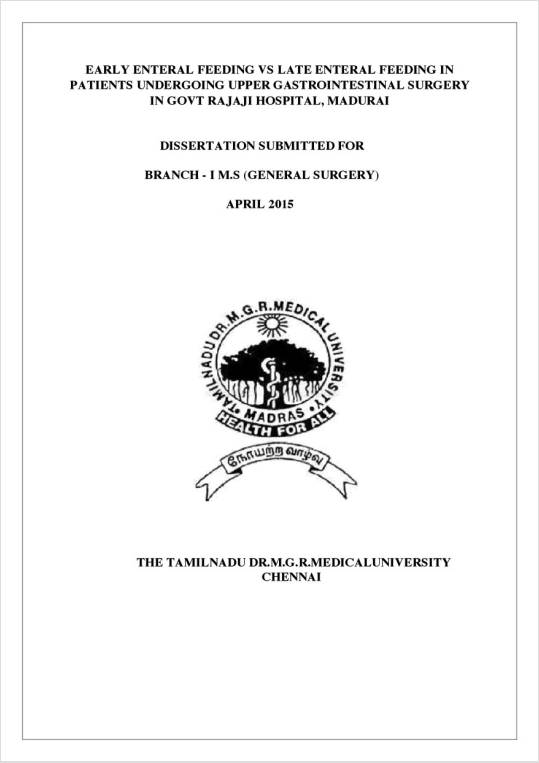

Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?s=1&o=450346794&u=1030975885&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical ...
TNMGRMU EXAMINATIONS - DUE 15-..
Originality
GradeMark
PeerMark
EARLY ENTERAL FEEDING VS LATE ENTERAL FEEDING IN PATIENTS
BY 221211116.MS GENERAL SURGERY SRAVAN C P S
turnitin
16%
SIMILAR
--
OUT OF 0
41EARLY ENTERAL FEEDING VS LATE ENTERAL FEEDING IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL SURGERY IN GOVT RAJAJI HOSPITAL, MADURAI
DISSERTATION SUBMITTED FOR
BRANCH - I M.S (GENERAL SURGERY)
APRIL 2015
THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
MADRAS
HEALTH FOR ALL
நோயற்ற வாழ்வு
Match Overview
1 www.bhj.org Internet source 3%
2 Martins, C.. "Nutrition f... Publication 2%
3 justmed.eu Internet source 1%
4 www.accessmedicine.c... Internet source 1%
5 www.tropicalgastro.com Internet source 1%
6 www.thai-otsuka.co.th Internet source 1%
7 www.nursing.health.wa... Internet source 1%
8 Parrish, C. R.. "Enteral ... 1%
PAGE: 1 OF 105
Text-Only Report
receipt_EARLY ENTE....pdf
EARLY ENTERAL FEE....pdf
9.2 MB
Show all downloads...
03:41
17-09-2014

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221211116.ms General Surgery SR...
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	EARLY ENTERAL FEEDING VS LAT...
File name:	Binder1_with_page_nos.pdf
File size:	778.74K
Page count:	105
Word count:	15,036
Character count:	81,996
Submission date:	17-Sep-2014 03:18AM
Submission ID:	450346794



EARLY ENTERAL FEEDING VS LATE ENTERAL FEEDING IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL SURGERY IN GOVT RAJAJI HOSPITAL, MADURAI

DISSERTATION SUBMITTED FOR

BRANCH - I M.S (GENERAL SURGERY)

APRIL 2015

THE TAMILNADU DR.M.G.R.MEDICALUNIVERSITY
CHENNAI

Declaration

I, **Dr.Sravan.C.P.S,** hereby declare that, I carried out this work on "**EARLY ENTERAL FEEDING VS LATE ENTERAL FEEDING IN PATIENTS UNDERGOING UPPER GASTROINTESTINAL SURGERY IN GOVT RAJAJI HOSPITAL, MADURAI"** at the Department of Surgery, Govt. Rajaji Hospital, Madurai, under the guidance of **Prof. Dr.A.M.Syed Ibrahim MS.**

I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other University or Board either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery (Branch I) to be held in April 2015.

Place: Madurai

Date: **(Dr.SRAVAN.C.P.S)**

Acknowledgement

Any work in this world would be possible only due to the grace of the Almighty and so I thank God for giving me the strength to successfully complete this study. I thank my family for their unconditional love which is my daily pillar of support.

I sincerely thank the Dean Cap.Dr.B.Santhakumar Msc(Forensic),MD(FM), for allowing me to use the facilities in our hospital for conducting this study.

I thank my Head of the Deparment, Prof.Dr.A.Sankaramahalingam MS,for the support and words of wisdom.

I thank my guide and unit chief Prof.Dr.A.M.Syed Ibrahim MS, for his continuous guidance not only for this study but for his daily insight into the practical difficulties in our surgical field and the ways to overcome them in the right way.

I thank Retd.Prof.Dr.S.Meenakshi Sundaram MS, for his teachings on the ethical way of life and work and for being the guiding light for all budding surgeons.

I heartfully thank my Assistant Professors Dr.J.Amuthan MS,Dlo, Dr.M.Lakshinarayanan MS and Dr.D.Geetha MS,Dgo for their daily support and guidance in each and every way possible making my daily routine pleasurable.

I sincerely thank Prof.Dr.S.R.Dhamotharan MS and Prof.Dr.P.Amutha MS for their constant encouragement and critical suggestions.

Place: Madurai.

Date :

Dr.SRAVAN.C.P.S

Post Graduate Student,

Department of General Surgery,

Madurai Medical College,Madurai

Abstract

Post operative ‘nil per mouth’ is the most commonly practiced methodology after a patient undergoes upper gastrointestinal surgeries like gastrectomy, gatrojejunal anastamosis and closure of perforated stomach or duodenum. The rationale behind that was to give time for the anastamosis to heal before being challenged by liquid or solid diets and to prevent post operative nausea and vomiting. The concept of early enteral feeding though having proper advantages has not had widespread following. Contrary to the commonly known opinion the oral feeds following upper gastrointestinal surgeries would increase the risk of anastamotic dehiscence and also worsen the ileus of the bowel, early feeds are absorbed well and also have a faster recovery of paralytic ileus, cause lesser septic complications, improve nutrition and lesser hospital stay.

Objective: To show the advantages of starting the patients undergoing upper gastrointestinal surgeries on early feeding using a nasojejunal tube with milk based diet over the conventional late enteral feeding.

Methodology: Two groups of patients with 25 in each are put up as study and control groups. Patients in the study group are inserted a nasojejunal tube during surgery and started on early enteral feeding with milk based diet following the feeding protocol. Control groups are managed by conventional nil per mouth and late enteral feeding. The parameters monitored are patient weight,

haemoglobin, S.albumin, duration of paralytic ileus, time taken to start oral feeds, duration of hospital stay, septic complications and surgical site infections.

Results: The mean age of the patients in the study group was 46.88yrs whereas in the control group was 47.96yrs. The mean weight of the study cases pre operatively was 57.56 kg but weight increased to about 58.6kg by post operative day 7.The same was not seen in control cases. . The mean pre operative haemoglobin among the cases in study group was 9.7g% and levels increased to 9.98g% by post operative day7. The same was not seen in control group. The pre operative S.albumin levels among the patients started on early feeding was 2.74g/dl and by post operative day7 it was 3.13g%. The same increase was not observed in control patients. The mean duration of paralytic ileus among the cases in the study group was 2.4 days whereas in the control group was 4.04 days. The mean duration taken to start oral feeds is 4.4 days in study group when compared to those cases in the control group where the mean duration is 6 days. The rate of anastamotic leak when comparing both groups was not significant. Among the control group patients in the study about 9 patients developed surgical site infection when compared to to nil patients in study group. Septic complications like pneumonia and urinary tract infections developed in 2 cases of the study group whereas 13 cases in the control group developed the same. Side effects due to feeds were seen among 13 patients of the study group. The mean duration of hospital stay among the patients of the

study group was 7.6 days whereas among those in the control group was 10.2 days.

Conclusion: This study clearly proves that early enteral feeding has great advantages over the conventional method of late enteral feeding in patients undergoing upper gastrointestinal surgeries and that it can be followed as a routine for better post operative outcomes.

TABLE OF CONTENTS

INTRODUCTION

Post operative starvation is the most common wide spread practice after gastro Intestinal surgery, which may not be beneficial [1].The rationale of nil by mouth and gastric decompression is to prevent post operative Nausea and vomiting and protect the anastomosis allowing it time to heal before being Stressed by food.

Early feeding may enhance wound healing and increase anastomotic strength particularly in malnourished patients[2 3]·Pre existing malnutrition is a major clinical problem in surgical patients[4] .Nutritional depletion is an independent determinant of serious complications after major gastro Intestinal surgery[5] ·Early nutritional support was associated with significant reduction in post operative Complications, A reduction that was independent of pre operative nutritional status[6]• [7].

The benefits of post operative Enteral feeding in normally nourished surgical patients indicate that it is reduced nutritional intake that predisposes to develop complications, Including deficits in muscle function and fatigue[6]·Early post operative Enteral nutrition either afforded no advantage over standard care or seemed to have a deleterious effect[8 9].

Early post operative Enteral nutrition may have a beneficial effect on function of intestinal barrier in respect of permeability, bacterial translocation and subsequent development of septic complications [10]·Early post operative nutrition influences intestinal permeability [11].

REVIEW OF LITERATURE

A study conducted in 24 patients who underwent elective surgery for esophageal carcinoma were randomized into immediate Enteral nutrition and parental nutrition group.This study showed beneficial effects on nutritional status, immunological competence, suppression of excessive inflammatory response, plasma nitrate and nitrite levels between the immediate Enteral nutrition group and parental nutrition group[12]

A prospective study trail in 212 patients who underwent pancreaticoduodenectomy were randomized to receive a standard Enteral formula or parenteral nutrition. Patients receiving immunonutrition had a significant better recovery, decrease in rate of post operative complication($p=0.005$), mean length of hospital stay was shorter($p=<0.05$) This study concluded that post operative Enteral feeding may safely and effectively replace parental nutrition in patients undergoing pancreaticoduodenectomy. [13]

In a study a total of 128 patients were participated,67 were randomized to a conventional return to diet group and 61 to free diet group. Results showed the complications are similar in both groups, free diet group tolerated normal diet well when compared to conventional

group($p<0.001$).this study concluded that early resumption of oral intake does not diminish the duration of post operative ileus or lead to a significantly increased rate of nasogastric reinsertion, tolerance of oral diet is not influenced by gastrointestinal recovery, post operative management should include early resumption of diet[14].

A metaanalysis of randomized controlled trail which included 11 studies with 837 patients showed early feeding reduces the risk of infection($p=0.036$),mean length of hospital stay ($p=0.001$),anastomotic dehiscence, wound infection, pneumonia, intraabdominal abscess and mortality. He finally concluded there is no clear advantage of keeping patients nil by mouth after elective gastrointestinal surgery, early feeding may be of benefit [15]

A study conducted on 104 successive patients who underwent colorectal surgery 89 patients started on oral diet out of which 65 patients tolerated early oral feeding.uni variate analysis showed that the use of volume expanders . contributed to intolerance of oral feeding. On multivariate analysis blood loss during the operation was the only factor contributing to failure of early post operative feeding. This study concluded early feeding is safe and feasible [16]

A study conducted in 1716 patients after gastrectomy and surgeries for chronic duodenal obstruction which showed Enteral tube feeding stimulates motor, synthetic, and barrier function of small intestine; it also permits to improve immediate results of Stomach and duodenal surgeries and also reduces the cost of the treatment[17].

A consultant physician D B A Silk had showed that early feeding may enhance wound healing and anastomotic strength particularly in malnourished patients. It also associated with reduction in post operative complications and it also has beneficial effect. On function of intestinal barrier in respect of permeability, bacterial translocation and subsequent development of septic complications [18]

A study showed feeding gut early after surgery is safe and well tolerated and it should represented the first choice for nutritional support in these type of patients[19]

"ENTERAL NUTRITION"

Definition: Nutritional support via placement through the nose, esophagus, stomach, intestine (duodenum, jejunum).

It is often called as tube feeding.

GENERAL PRINCIPLES OF ENTERAL FEEDING

1)When intake of a food with hyper osmotic levels, occurs in order to neutralize the same by the endogenous gastric secretions with time the gastric motility slows down gradually and stops for a limited required period. Once the contents become iso osmotic, gastric motility starts immediately and the now iso osmotic contents are propelled into the duodenum. Hence the first and main defense barrier against hyper osmotic enteral feeds is the stomach. Also the ability of the small bowel to act in the same way is limited especially when such feeds are introduced directly.

2) One of the functions of the gastric secretions, especially the gastric acid secretions is to neutralize the bacterial content in the intake food. By constant enteral infusion this function may be nullified. Also if the feeds are not prepared or maintained properly, bacterial overgrowth may occur in it and this it would lead adverse effects. Proper maintenance of the feeds like adequate refrigeration is a must.

3) Protein absorption: Proteins are broken down into oligopeptides and dipeptides which are easily absorbed than single amino acids. However recent

studies have also proven that single amino acids are directly being absorbed in the small intestine. Proteins are usually absorbed in the initial 120cm of the small intestine. Dipeptides are better absorbed in diseased bowel.

4) Carbohydrate absorption: Simple forms are absorbed easily than complex forms. This also happens in the proximal jejunum. A main factor is the problem of acquired lactase deficiency. This is commonly seen in critically ill patients. This maybe the cause of diarrhea in patients given early enteral nutrition containing lactose as the deficiency is more pronounced in the early phase and it gradually gets corrected with time.

5) Fat absorption: Fat absorption is a complicated process as it involves mixing of the food with the bile and pancreatic enzymes in order for proper breakdown into smaller compounds which is then absorbed easily. Once surgeries of the upper gastrointestinal tract like gastrectomy, pancreatic surgeries or other complex procedures are done, this mixing is prevented from happening in the natural fashion and hence adverse effects may occur. Absorption of fat is reduced after the patients undergo gastrectomy and Billroth II anastamosis. This is comparatively lesser in Billroth I anastamosis. Hence the composition of enteral feeds should be devised in such a way that the fat composition is just adequate to prevent complications.

6) Others: Elemental metals, iron, calcium and other similar compounds and predominantly absorbed in the duodenum. Once procedures which bypass the passage of feeds through the duodenum are performed, deficiencies may occur overtime.

PRACTICALITIES OF ENTERAL FEEDING

“Where Gut is available use it”

A major recent change in nutritional support is the realization that the gut may be more efficacious, at least in burns and trauma, as compared with parenteral nutrition. Enteral nutrition has not been emphasized as much as parenteral nutrition, because it has been assumed that in many disease states, the gut will not work. With effort, it turns out that the gut works and can be used but that perhaps it cannot provide total nutritional support. Still, there is probably significant benefit from utilizing the gut for partial nutritional support.

Therefore, one should approach nutritional support with two goals in Mind:

- ☐ Use the gut if possible
- ☐ If total nutritional supplementation cannot be provided by gut, to administer at least 20% of the caloric and protein requirements by gut.

ENTERAL FEEDING VS PARENTERAL FEEDING

1. The enteral route is **more physiologic** — the liver is not bypassed and hepatic ability to take up, process, and store the various nutrients for later release on nervous or hormonal command is maintained.
2. Increased cardiac output is required when the gut is bypassed. With parenteral nutrition, gut blood flow increases about 15% to 20%, presumably to allow the gut to perform its usual metabolic functions, such as transamination.

3. It is often said that enteral nutrition is safer and more efficacious than the parenteral route.
4. Recent studies in the trauma and burn settings have suggested, improved outcome. The initial study indicating that there might be an advantage to survival with enteral feeding came from a study of burned children.

The study found that increasing the percentage of calories with whey protein from the standard 15% to 25% in severely burned children statistically improved survival. The children who survived with the increased amount of protein received a greater percentage of their feeding by gut as opposed to vein. Alexander and coworkers, in a classic series of investigations, subsequently provided evidence that gut feedings early in burns in guinea pigs and subsequently in man prevented, in part, the burn hypercatabolism. The working hypothesis was that early gut feeding in man, prevented bacteria and/or their products from translocating the gut mucosa, releasing catecholamines and other counter regulatory stimuli, and thus prevented the hypercatabolism.

In posttraumatic situations, two prospective and randomized studies showed that early gut feedings result in lower mortality and septic complication rates. Enteral nutrition is superior to parenteral nutrition with respect to outcome.

TRANSLOCATION MECHANISM

It is a normal process that teleologically may be important in releasing small amounts of endotoxin to prime the immune systems. Translocation is increased in burns and in hemorrhagic shock but not by pure starvation. Whereas the bacteria are normally cleared by the lymph nodes, it is the bacterial products in the portal circulation, presumably interacting with the Kupffer cells and hepatocytes that may contribute to the hepatic dysfunction and multiple organ system failure.

There are two studies of early gut feeding in traumatized patients that seem to indicate that early posttraumatic jejunal feeding results in a lower rate of mortality and sepsis in patients receiving the jejunal feeding than those receiving parenteral nutrition.

One problem with the translocation hypothesis as a whole is that a number of results have been ascribed to it that cloud, rather than clarify, the issue. Any beneficial result of enteral feeding is automatically attributed to improvement in gut mucosal barrier integrity. As stated earlier, there are other possible explanations for beneficial results obtained from gut feeding.

Although it is true that in pre-agonal patients, total breakdown of gut mucosal integrity results in random bacteremias without a focal area of infection, clinically significant loss of gut mucosal integrity has been demonstrated only in burns, trauma, and perhaps hemorrhagic shock.

The final common pathway is lack of perfusion of the gut. Finally, one should focus on clearance, that is, the number of viable bacteria rather than translocation per se.

BENEFITS OF ENTERAL FEEDING

Physiologic and Metabolic Benefits

The gastrointestinal tract can be used for administration of complex nutrients, such as intact protein, peptides, and fiber that cannot be given intravenously. Gut processing of intact nutrients provides a stimulus for hepatic synthetic function of proteins, whereas administration of nutrients directly into the systemic circulation bypasses the portal circulation. In addition to its systemic benefits, enteral feeding has beneficial local effects on gastrointestinal mucosa. These include trophic stimulation and maintenance of absorptive structures by nourishing the enterocytes directly, thus supporting epithelial cell repair and replication. Luminal nutrients such as glutamine and short-chain fatty acids are used as fuel by the cells of the small bowel and colon respectively.

Immunologic Benefits

The presence of food in the gut, particularly complex proteins and fats, supports the mucosa's critical function as an immunologic barrier by triggering feeding-dependent neuroendocrine activity. This activity stimulates the production of immunoglobulins in the gut, particularly secretory immunoglobulin A, which is important for preventing bacterial adherence to gut mucosa and bacterial translocation. The presence of nutrients in the gut also helps maintain normal gut pH and flora, thus diminishing opportunistic bacterial overgrowth in the small bowel.

Safety Benefits

Enteral feeding is generally considered safer than parenteral feeding. Meta-analysis of prospective trials has demonstrated fewer infectious complications with enteral nutrition compared with parenteral nutrition. Subset analysis suggests that enteral nutrition does not result in a lower risk of infection but rather that parenteral nutrition results in a higher risk. Hyperglycemia, and its resulting inhibition of neutrophil-mediated immunity, also occurs more frequently with parenteral feeding. Enteral nutrition has its own potential complications.

Cost Benefits

The direct costs of enteral feeding are generally less than those with parenteral nutrition. Direct costs include formula, feeding pumps, and tube placement. The cost advantage for enteral feeding is even greater when indirect costs such as central line placement, infection or thrombosis, and home health care are considered.

INDICATIONS FOR ENTERAL FEEDING

Enteral nutrition is the preferred method of nutrition support for malnourished patients or those at risk for developing malnutrition and who have an intact gastrointestinal tract with adequate length. Patients who are either unable or unwilling to eat to meet their daily needs are candidates for enteral support. Factors influencing the timing of initiation of enteral nutrition include evidence of pre-existing malnutrition, expected degree of catabolic activity, duration of the current illness, and anticipated return to intake by mouth. Patients with partially functioning gastrointestinal tracts (eg, short bowel syndrome, proximal enterocutaneous fistula) often can tolerate some enteral feeding but may require a combined regimen of both parenteral and enteral nutrition to meet total caloric needs.

POSSIBLE CONTRAINDICATIONS TO ENTERAL FEEDING

There have been no absolute contraindications postulated for early enteral feeding. Most are relative contraindications only. Patients with short inadequate absorbent bowel, gastrointestinal obstruction, gastrointestinal bleeding, protracted vomiting and diarrhea, high fistulas, or active gastrointestinal ischemia may require a period of bowel rest. In times of physiologic stress, the body shunts blood away from the splanchnic circulation. Feeding a patient who is hemodynamically unstable or requires vasopressors may produce bowel ischemia in the setting of pre-existing tenuous perfusion. Most of the contraindications put forward can be avoided by altering factors like changing the feeding site, altering the constituents of the feed, new equipments and newer techniques.

INITIATING FEEDINGS

Though many protocols have been postulated in the past the recent protocol is to start feeds with full strength. This is done slowly and the rate of increase must also be slow. This approach reduces the risk of microbial contamination and achieves full nutrient intake earlier. Formulas are often introduced at full strength at 10–40 mL per hour initially and advanced to the goal rate in increments of 10–20 mL per hour every 4 to 8 hours as tolerated.

Diluted feeds are to be given in patients in the Intensive care and patients those who have not taken oral feeds for some time. For such patients the initial rate of feeds must also be slow and the rate of increment must also be gradual. Similar rules apply to patients initiated on high calorie feeds of hyper osmotic feeds. In such patients, starting feeding at 10 mL per hour yields the trophic benefit of enteral feeds without unduly stressing the gut. In patients with active lifestyles, gastric feeds can be provided as boluses of up to 400 mL each, delivered at intervals of 4 to 6 hours.

MONITORING FEEDINGS

Assessing gastrointestinal tolerance to enteral feeding includes monitoring for abdominal discomfort, nausea and vomiting, abdominal distention, abnormal bowel sounds or stool patterns and abdominal pain. Gastric residual volumes are used to evaluate gastric emptying of enteral feedings. High residuals raise concerns about intolerance to gastric feedings and the potential risk for regurgitation and aspiration. When the gastric residual is greater than 200 mL or is associated with signs or symptoms of intolerance, feedings should be held. If the abdominal examination is unremarkable, feedings should be postponed for at least an hour and the residual volume rechecked. If high residuals persist without associated clinical signs and symptoms, a promotility agent (eg, erythromycin, metoclopramide) may be added to the feeding regimen.

DIETS

Optimal Diet

The optimal diet should have the following distribution of energy sources: carbohydrate 55–60%, fat 30%, and protein 10–15%. Refined sugar should constitute less than 15% of dietary energy and saturated fats no more than 10%, the latter balanced by 10% monounsaturated and 10% polyunsaturated fats. Cholesterol intake should be limited to about 300 mg per day (one egg yolk contains 250 mg of cholesterol). The amount of salt in the average American diet, 10–18 g daily, far exceeds the recommended 3 g/day. For Western societies to meet the criteria for an optimal diet consumption of fat must decrease (from 40%) and consumption of complex carbohydrate should increase. Meat is presently overemphasized as a protein source, at the expense of grain, legumes, and nuts. Diets that include substantial fish intake have been associated with a decrease in mortality from cardiovascular disease and are attributed to high concentrations of -3 fatty acids, principally eicosapentaenoic and docosahexaenoic acids.

Many adults, particularly those who do not drink milk, consume inadequate amounts of calcium. In women this may result in calcium deficiency and skeletal calcium depletion, predisposing women to osteoporosis and axial bony fractures. "Fiber" is the generic term for a chemically complex group of indigestible carbohydrate polymers, including cellulose, hemicellulose, lignins, pectins, gums, and mucilages.

The amount of fiber in Western diets averages 25 g per day, but some people ingest as little as 10 g daily. Those who consume low-fiber diets are more likely to develop chronic constipation, appendicitis, diverticular disease, and possibly diabetes mellitus and colonic neoplasms. Bran cereals and bread, fruit, potatoes, rice, and leafy vegetables are rich sources of fiber.

Regular Diets

Many concepts regarding diets are archaic and based on currently unaccepted views of illness. For example, the utility of a low-residue diet in diverticular disease is questionable. The "progressive diet," designed for postoperative feeding and consisting of a clear liquid (high in sodium), then a full liquid (high in sucrose), then a regular diet, is based on outmoded concepts. When peristalsis returns after operation, as evidenced by bowel sounds and ability to tolerate water, most patients are able to ingest a regular diet. Regular diets have an unrestricted spectrum of foods and are most attractive to the patient. An average regular hospital diet for 1 day contains 95–110 g of protein, with a total caloric content of 1800–2100 kcal. This composition reflects the nutritional needs of healthy persons of average height and weight and will not meet the increased demands imposed by malnutrition or disease.

Lactose Intolerance & Lactose-Free Diets

A lactose-free diet is indicated for patients who have symptoms such as diarrhea, bloating, or flatulence after the ingestion of milk or milk products. Lactose intolerance is genetically determined and occurs in 5–10% of European Caucasians, 60% of Ashkenazi Jews, and 70% of African Americans. Subclinical lactose intolerance may become unmasked following surgery on gastrointestinal tract (eg, gastrectomy). Similarly, avoidance of lactose-containing products is often beneficial advice for patients with Crohn disease, ulcerative colitis, and AIDS. The efficiency of lactose digestion and absorption can be measured by giving 100 g of oral lactose, then measuring the blood glucose concentration at 30-minute intervals over 2 hours. Patients with lactose intolerance exhibit a rise in blood glucose of 20 mg/dL or less. A lactose-free diet may be deficient in calcium, vitamin D, and riboflavin.

Post gastric Bypass Diet

The popularity of gastric bypass surgery for weight loss continues to increase. The diet changes that must occur to ensure safe and appropriate weight loss are quite specific after surgery. Immediately after surgery, only small amounts of liquids (eg, 30mL q3h) should be consumed. After tolerance of liquids is established, pureed foods should be consumed for the 4 weeks after surgery.

Food should be consumed as very small meals and snacks throughout the day. Choosing a variety of foods, avoiding concentrated sweets, and consuming adequate protein are essential to the success of these patients. Protein supplements are often required to ensure adequate protein consumption postoperatively.

Disease-Specific Nutrition Support

Burns

Thermal injury has a tremendous impact on metabolism because of prolonged, intense neuroendocrine stimulation. Extensive burns can double or triple the REE and urinary nitrogen losses, producing a loss of 1500 g per day of lean tissue and a median survival of 7–10 days without nutritional support. The increase in metabolic demands following thermal injury is proportional to the extent of ungrafted body surface. The principal mediators of burn hypermetabolism are catecholamines, which return to baseline only when skin coverage is complete. Decreasing the intensity of neuroendocrine stimulation by providing adequate analgesia and a thermoneutral environment lowers the accelerated metabolic rate and helps to decrease catabolic protein loss until the burned surface can be grafted. Burned patients are prone to infection, and the cytokines activated by sepsis further augment catabolism.

Because infection often complicates the clinical course of patients with burn injury, and infectious complications are more likely with parenteral nutrition, the enteral route of feeding is preferred whenever tolerated. Enteral feeding may be started within the first 6–12 hours postburn to reduce the hypermetabolic response and improve postburn survival. Gastric ileus can be avoided through the use of a nasojejunal tube. Patients with burns have increased caloric requirements. In addition to estimated maintenance needs (females, 22 kcal/kg/day; males, 25 kcal/kg/day), these patients require an additional 40 kcal per percentage point of burned total body surface area (TBSA). A 70-kg man with 40% TBSA burns would require 48 kcal/kg/day. Protein requirements are also markedly increased from the normal 0.8 g/kg/day to approximately 2.5 g/kg/day in severely burned patients. Of course, these are initial estimates, and periodic reassessment of nutritional status (eg, prealbumin levels, nitrogen balance) is required in these patients. During the hypermetabolic phase of burn injury (0–14 days), the ability to metabolize fat is restricted, so a diet that derives calories primarily from carbohydrate is preferable. Following the hypermetabolic phase, the metabolism of fat becomes normal. The burn patient should also be given supplemental arginine, nucleotides, and -3 polyunsaturated fat to stimulate and maintain immunocompetence.

Diabetes

Glucose intolerance often complicates nutritional supplementation, particularly with parenteral administration. Complications associated with TPN administration occur more frequently during prolonged hyperglycemia. Unopposed glycosuria may lead to osmotic diuresis, loss of electrolytes in the urine, and possibly nonketotic coma. Additionally, it is now evident that strict maintenance of serum glucose levels below 110 mg/dL improves mortality and decreases infectious morbidity in critically ill surgical patients. Factors that may aggravate hyperglycemia include the use of corticosteroids, certain vasopressors (eg, epinephrine), preexisting diabetes mellitus, and occult infection.

Maintaining normoglycemia in injured or postoperative patients may be challenging. Serial serum glucose levels should be monitored regularly. If hyperglycemia does not occur, these measurements can be obtained less frequently once the nutritional goal is reached. Patients may require subcutaneous insulin administered on a sliding scale or continuous intravenous insulin infusions to control their hyperglycemia. For patients who do not require an insulin infusion, the previous day's insulin total from a sliding scale may be determined and half to two-thirds of that amount added to the next TPN order to provide a more uniform administration.

Cancer

Cancer is the second leading cause of death in the United States, and over two-thirds of patients with cancer will develop nutritional depletion and weight loss at some time during the course of the illness. Malnutrition and its sequelae are the direct cause of death in 20–40% of these patients. Weight loss is an ominous presenting sign in many malignancies. Furthermore, antineoplastic reatments such as chemotherapy, radiation therapy, or operative extirpation, can worsen preexisting malnutrition. Cancer cachexia manifests as progressive involuntary weight loss, fatigue, anemia, wasting, and tissue depletion. It may occur at any stage of the disease. Nutrition support has become an essential adjunct in caring for the cancer patient.

Many studies have evaluated the effectiveness of nutrition support in patients with cancer, with varying results. Klein reported a meta-analysis of 28 prospective, randomized controlled trials evaluating TPN in patients with cancer. Only 1 of 10 surgical trials showed a significant decrease in mortality in the patients receiving TPN, and no other significant benefit was seen in survival, tolerance to treatment, toxicity, or tumor response in patients receiving chemotherapy or radiation therapy. Increasing efforts have been directed toward the use of enteral nutrition because it is simpler, presumably safer, and less costly.

Seven prospective, randomized controlled trials of enteral nutrition in patients with cancer who were undergoing surgery showed little if any difference in mortality or morbidity in patients who received enteral feedings. In summary, nutritional supplementation in cancer patients may reduce infectious complications or perioperative morbidity, but convincing evidence of improvement in overall survival is lacking.

Patients with cancer may have altered energy expenditure and abnormalities ofprotein and carbohydrate metabolism. REE increases by 20–30% in certain malignant tumors. The increases in REE can occur even in patients with extreme cachexia in whom a similar degree of uncomplicated starvation would produce profound decreases in REE. Whether the increase in REE correlates with the extent of disease or tumor burden is unknown. Changes in carbohydrate metabolism consist of impaired glucose tolerance, elevated glucose turnover rates, and enhanced Cori cycle activity. Owing to the high rate of anaerobic glucose metabolism in neoplastic tissue, patients with extensive tumors are susceptible to lactic acidosis when given large glucose loads during TPN. These patients also exhibit increased lipolysis, elevated FFA and glycerol turnover, and hyperlipidemia.

Patients with cancer avidly retain nitrogen despite losses in most lean tissue. Animal carcass analysis has shown that the retained nitrogen resides in the tumor, which behaves as a nitrogen trap. Synthesis, catabolism, and turnover of body protein are all increased, but the change in catabolism is greatest.

The utility of enteral supplementation with immune-enhancing agents is unclear. These substances include arginine, glutamine, essential fatty acids, RNA, and BCAAs. Several studies have attempted to examine outcomes in patients with cancer who are fed with enteral formulas supplemented with immune-enhancing agents, compared to routine enteral feeding alone. The findings were summarized by Heys and coworkers. Meta-analysis of six studies with a total of 487 cancer patients demonstrated a decrease in overall infectious morbidity and hospital stay, but no change in survival, for patients receiving such "targeted therapy." Exactly which elements confer these benefits remains unknown.

Renal Failure

Whether nutritional support improves the outcome from acute renal failure is difficult to determine because of the metabolic complexities of the disease. Patients with acute renal failure may have normal or increased metabolic rates.

Renal failure precipitated by x-ray contrast agents, antibiotics, aortic or cardiac surgery, or periods of hypotension is associated with a normal or slightly elevated REE and a moderately negative nitrogen balance (4–8 g per day). When renal failure follows severe trauma, rhabdomyolysis, or sepsis, the REE may be markedly increased and the nitrogen balance sharply negative (15–25 g per day). When dialysis is frequent, losses into the dialysate of amino acids, vitamins, glucose, trace metals, and lipotrophic factors can be substantial.

Patients in renal failure (serum creatinine over 2 mg/dL) with a normal metabolic rate who cannot undergo dialysis should receive a concentrated (minimal volume) enteral or parenteral diet containing protein, fat, dextrose, and limited amounts of sodium, potassium, magnesium, and phosphate.

Hepatic Failure

Most patients with hepatic failure present with acute decompensation superimposed on chronic hepatic insufficiency. Typically, a history of poor dietary intake contributes to the chronic depletion of protein, vitamins, and trace elements. Water-soluble vitamins, including folate, ascorbic acid, niacin, thiamin, and riboflavin, are especially likely to be deficient. Fat-soluble vitamin deficiency may be a result of malabsorption due to bile acid insufficiency (vitamins A, D, K, and E), deficient storage (vitamin A), inefficient utilization (vitamin K), or failure of conversion to active metabolites (vitamin D).

Hepatic iron stores may be depleted either from poor intake or as a result of gastrointestinal blood loss. Total body zinc is decreased owing to the above factors plus increased urinary excretion.

The use of BCAA-enriched amino acid formulations for TPN in patients with liver disease is controversial because the results of controlled trials are inconclusive. Therefore, patients with hepatic failure should receive a concentrated enteral or parenteral diet with reduced carbohydrate content, a combination of EFAs and other lipids, a standard mixture of amino acids, and limited amounts of sodium and potassium.

Cardiopulmonary Disease

Malnutrition is associated with myocardial dysfunction, particularly in the late stages, and fatal cardiac failure can develop in extreme cachexia. Cardiac muscle uses FAAs and BCAAs as preferred metabolic fuels instead of glucose. During starvation, the heart rate slows, cardiac size decreases, and the stroke volume and cardiac output decrease. As starvation progresses, cardiac failure ensues, along with chamber enlargement and anasarca.

The profound nutritional depletion that may accompany chronic heart failure, particularly in valvular disease, results from anorexia of chronic disease, passive congestion of the liver, malabsorption due to venous engorgement of the small bowel mucosa, and enhanced peripheral proteolysis due to chronic neuroendocrine secretion.

Attempts at aggressive nutritional repletion in patients with cardiac cachexia have produced inconclusive results. Concentrated dextrose and amino acid preparations should be used to avoid fluid overload. Nitrogen balance should be measured to ensure adequate nitrogen intake. Lipid emulsions must be administered cautiously because they can produce myocardial ischemia and negative inotropy. Feeding these patients with either enteral or parenteral nutrition should be undertaken cautiously to avoid refeeding syndrome and hypophosphatemia.

Patients with severe chronic obstructive pulmonary disease may have difficulty in weaning from the ventilator if they are overfed. This relates to the RQ, a measure of oxygen consumption and carbon dioxide production by the body in metabolism. An RQ of 1 reflects pure carbohydrate utilization, while an RQ greater than 1 occurs during lipogenesis (energy storage). Although normal lungs can tolerate increased CO_2 production (RQ greater than 1) without adversely affecting respiration, patients with chronic obstructive pulmonary disease may experience CO_2 retention and inability to wean. The treatment is to increase the percentage of calories delivered as lipid and to avoid overfeeding at all costs.

Disease of the Gastrointestinal Tract

Benign gastrointestinal disease (eg, inflammatory bowel disease, fistula, pancreatitis) often leads to nutritional problems due to intestinal obstruction, malabsorption, or anorexia. Chronic involvement of the ileum in inflammatory bowel disease produces malabsorption of fat- and water-soluble vitamins, calcium and magnesium, anions (phosphate), and the trace elements iron, zinc, chromium, and selenium. Protein-losing enteropathy, accentuated by transmural destruction of lymphatics, can add to protein depletion. Treatment with sulfasalazine can produce folate deficiency, and glucocorticoid administration may accelerate breakdown of lean tissue and enhance glucose intolerance owing to stimulation of gluconeogenesis. Patients with inflammatory bowel disease who require elective surgery should be evaluated for malnutrition preoperatively.

Patients with gastrointestinal fistulas can develop electrolyte, protein, fat, vitamin, and trace metal deficiencies; dehydration; and acid-base imbalance. Aggressive fluid replacement is often needed. Patients with fistulas often require nutritional support. The choice of feeding route or formula will depend on the level and length of dysfunctional bowel. Patients with proximal enterocutaneous fistulas (from the stomach to the midileum) should receive TPN with no oral intake. Patients with low fistulas should receive TPN initially, but after infection is brought under control, they can often be switched to an enteral formula or even a low-residue diet.

Pancreatitis

The diagnosis of pancreatitis often mandates strict bowel rest for extended periods of time. Ranson criteria can serve as a rough estimate of the need for nutritional support. Patients with acute pancreatitis who present with three or fewer Ranson criteria should be treated with fluid replacement, nasogastric suction, and bowel rest for at least a week before considering parenteral nutrition. Most of these patients can resume an oral diet and do not benefit from TPN. Those with more than three Ranson criteria should receive TPN. Previously, enteral diets, including elemental and polypeptide formulas, were not recommended due to concerns these diets may stimulate the pancreas and aggravate the disease. However, recent data document the successful use of enteral diets, particularly elemental products via jejunal access, in many patients with pancreatitis.

Short Bowel Syndrome

Inadequate intestinal absorptive surface leads to malabsorption, excessive water loss, electrolyte derangements, and malnutrition. The absorptive capacity of the small intestine is highly redundant, and resection of up to half its functional length is reasonably well tolerated. Short bowel syndrome typically occurs when less than 200 cm of anatomic small bowel remain, although the presence of the ileocecal valve may reduce this length to 150 cm.

However, short bowel syndrome also may occur from functional abnormalities of the small bowel resulting from severe inflammation or motility disorder. The optimal nutritional therapy for a patient with short bowel syndrome must be tailored individually and depends upon the underlying disease process and the remaining anatomy. Following resection, the remaining bowel undergoes long-term adaptation, with observed increases in villous height, luminal diameter, and mucosal thickness. The estimated minimum length of small bowel required for adult patients to become independent of TPN is 120 cm.

Adaptation to short gut occurs over time, and initial management should be directed at avoiding electrolyte imbalance and dehydration while providing daily caloric requirements through TPN. Some patients may eventually supplement TPN with oral intake. In these patients, dietary management includes consuming frequent small meals, avoiding hyperosmolar foods, restricting fat intake, and limiting consumption of foods high in oxalate (precipitates nephrolithiasis). Uniquely formulated diets containing glutamine and human growth hormone have shown promise for accelerating intestinal adaptation.

AIDS

Patients with AIDS frequently develop protein-calorie malnutrition and weight loss. Many factors contribute to deficiencies of electrolytes (sodium and potassium), trace metals (copper, zinc, and selenium), and vitamins (A, C, E, pyridoxine, and folate). Enteropathy may impair fluid and nutrient

absorption and produce a voluminous, life-threatening diarrhea. Standard antidiarrheal agents do not control the diarrhea in AIDS patients, but the synthetic somatostatin analogue octreotide may help. Dehydration occurs as a consequence of refractory diarrhea.

Malnourished AIDS patients require a daily intake of 35–40 kcal and 2.0–2.5 g protein. Those with normal gut function should be given a high-protein, high-calorie, low-fat, lactose-free oral diet. Patients with compromised gut function require an enteral (amino acid or polypeptide) or parenteral nutrition.

Solid Organ Transplant Recipients

Patients who have undergone organ transplantation present unique issues in relation to nutritional management due to both the preexisting disease state and the medications taken to prevent graft rejection. During the acute posttransplant phase, adequate nutrition is required to help prevent infection, promote wound healing, support metabolic demands, replenish lost stores, and mediate the immune response. Organ transplantation complications, including rejection, infection, wound healing, renal insufficiency, hyperglycemia, and surgical complications, require specific nutritional requirements and therapies. Obesity is associated with both decreased patient survival and decreased graft survival, in part due to a greater incidence of surgical, metabolic, and cardiovascular complications.

Patients with BMI greater than 30 kg/m^2 show a higher incidence of steroid-induced posttransplant diabetes mellitus. The first 6 weeks following transplantation is characterized by increased nutritional demands due to a combination of surgical metabolic stress and high doses of immunosuppressive medications. Daily protein intake recommendation in the immediate posttransplant phase, as well as during acute rejection episodes, is 1.5 gm/kg actual body weight.

Long-term immunosuppression is associated with protein hypercatabolism,obesity, dyslipidemia, glucose intolerance, hypertension, hyperkalemia, and alteration of vitamin D metabolism. Approximately 60% of renal recipients develop dyslipidemia posttransplant. Alterations in lipid metabolism may be associated with corticosteroids, cyclosporine, thiazide diuretics, or beta-blockers, as well as with renal insufficiency, nephrotic syndrome, insulin resistance, or obesity. There is evidence that abnormal lipoprotein levels lead to glomerulosclerosis, renal disease progression, and even potential graft failure.

Dietary salt restriction is recommended in transplant patients, as salt intake may play a role in cyclosporine-induced hypertension caused by sodium retention. Sodium intake is recommended not to exceed 3 gm/day.

Cyclosporine is associated with hypomagnesemia and hyperkalemia, especially during the immediate posttransplant phase when the dosage is high. Additionally, antihypertensive treatment with beta-blocker agents or with angiotensin-converting enzyme (ACE) inhibitors may exacerbate hyperkalemia. Calcium, phosphorus, and vitamin D metabolism are influenced by prolonged therapy with steroids leading to osteopenia and osteonecrosis. The daily recommendation for dietary calcium is 800–1500 mg, and the recommended intake of phosphorus is 1200–1500 mg/d. Some patients may also require supplementation of active vitamin D. Patients on a low-protein diet often need multivitamin supplements. During the first year, the major nutritional goal is to treat preexisting malnutrition and prevent excessive weight gain.

Major Trauma

Patients involved in major trauma have rapid metabolic changes which need to be addressed immediately and managed in the post traumatic phase. Changes in metabolism may vary from days to weeks depending on the nature and severity. Severe trauma induces alteration of metabolic pathways and activation of the immune system.

Possible changes in metabolism are:

1) Hyperglycemia due to

    a) Increased insulin levels(‘entity called as traumatic diabetes’)

    b) Failure to tolerate glucose load

    c) Insulin resistance

2) Increased catabolism of stored proteins

Metabolic demand by the body spikes upto 2-3 times in the immediate post traumatic period.

Phases of metabolic change in post traumatic period.

Phase 1: Starts immediately after trauma. Lasts up to many hours.The main characteristics of this phase are targeted at containing the energy loss due to trauma and hey include a drop in the oxygen consumption and lowering of body temperature.

Phase 2: The second phase, which occurs after compensation of the state of traumatic-hemorrhagic shock, is associated with an increased metabolic turnover, activation of the immune system, and induction of the hepatic acute-phase response. This results in increased consumption of energy and oxygen. Release of proinflammatory cytokines and activation of the complement system occurs due to initiation of the systemic inflammatory cascade. These metabolic sequelae and inflammatory response are worsened b8y pre exisisting infections or by the translocation of bacteria from the bowel.

The release of anabolic hormones is reduced by the infusion of inotropic support and vasoactive drugs. Also the stored energy in the body is mobilized by the release of massive amounts of hormones like insulin, glucagon and catecholamines. Hepatic gluconeogenesis is accelerated. Acute phase reactants are released. The substrate for these both is glycolysis and protein breakdown from the skeletal muscles. Recent concepts take into account the role of the immune system in acute metabolic response to trauma. Studies show that methods to promote the immune system would enhance healing of wounds and counteract the loss of protein due to muscle breakdown. BMI is rapidly increased as a result of these acute changes with alters the neuro-endocrine axis. The main stimulants for these changes and pain and shock.

Appropriate immunonutrition should be started in the ICU, preferably by enteral route, in order to counteract the effects of the hypermetabolic state after major trauma. Without absolute contraindications, studies recently published support the initiation of early enteral feeding in patients admitted in Intensive care units preferably within 1-2 days. Such patients have to be carefully monitored as over caloric feeding would bring out more unwanted adverse reactions like hyperglycemia, lipogenesis, increased consumption of O2 and large amounts of generation of CO2.

Adverse reactions are more common in the obese and morbidly obese individuals.

Recommendations for feeding in obese and morbidly obese:

20kcal/kg body weight & 2g protein/kg body weight.

Alternate: hypocaloric feeds with increased protein intake.

ENTERAL FEEDING: DISPELLING MYTHS

BOWEL SOUNDS AND PERISTALSIS

The occurrence of bowel sounds has been taught as the indicator for the start of oral feeds for a long time in all teaching hospitals. The basis behind this is that the occurrence of bowel sounds indicates the start of peristalsis and that the bowel is ready to receive an enteral load. The pros/cons on accepting this are

1) The various descriptions of bowel sounds in the standard textbooks are ’high-pitched’, ‘hyperactive’, ‘on-existent’, and ‘hypoactive’.

2)"if you feed them, bowel sounds will follow" is the concept of ”the Field of Dreams Approach to bowel sounds”. Bowel motility is stimulated by hormonal secretion from the gut which in turn is secreted by the enteral feeds taken.

3) Absence of publications in reputed journals which support the fact that peristalsis is indicated by the occurrence of bowel sounds

ASSESSMENT OF PATIENT WITH ABSENT BOWEL SOUNDS

Does patient require gastric decompression? If so, is it meaningful? (i.e., is the volume similar to normal secretions above the pylorus or is it a small volume every shift? Distinguish severity by differentiating those patients requiring:

1. Low constant suction vs
2. Gravity drainage
3. An occasional residual check every 4-6 hours
    - Abdominal examination—distended?
    - Is the patient nauseated, bloated, feeling full?
    - Is the patient passing gas or stool?
    - What is the differential diagnosis?

Are abdominal issues high on the list? If the above clinical parameters are benign, consider a trial of TEN at low rate of 10-20 mL/hour and observe.

RESIDUAL VOLUME

Gastric residual volumes (RV) are recent concepts taken into account to monitor the tolerance of patients to enteral feeds.

Common assumptions are that it is not normal to have any amount of residual volume in the stomach and that the presence of residual volume would cause abdominal distension, feeling of fullness, nausea and vomiting.

There have been a handful of studies only that quote that the presence of residual volumes in the stomach is normal. One of the main functions of the stomach is to act as the reservoir. This entails the presence of atleast a minimal amount of contents in the stomach anytime. Studies have also proven the correlation between physical examination and radiological investigations but not residual volumes and the above two.

A commonly practiced act in hospitals following protocols of enteral feeding is the monitoring of residual volume because to indicates the tolerance of the gut to enteral feeds. However proper protocols explaining the constituents of residual volume, possibility of returning it to the stomach and the frequency of checking for the volume have neither been defined properly nor been published in reputed journals following prospective randomized trials.

A thorough knowledge on gastric physiology is needed when looking into factors affecting levels of residual volume. The average amount of secretions produced in the stomach when accounted with saliva produced is about 3000-4000ml/day.

This shows that the approximate amount of fluid passing through the pylorus per hour is about 145ml.This is in addition to any other diet taken.

If enteral nutrition is given at the rate of 100ml/hr and if the residual volume is about 200ml after a period of 4hrs, this means that about 780ml has passed through the pylorus in this period: [(145 mLsecretions/hr + 100 mL TEN/hr) x 4 hrs] - 200 mL residual = 780 mL

If the state of ileus ie non peristaltic gut was present then the expected volume present as residual volume will be more.

Cascade effect: When the patient lies in a supine position the stomach is divided into two portions by the spine. So occasionally when a nagogastric tube is passed in it may turn around and end up in the fundus. Hence when feeds are given through this tube it gets initially collected in the fundus whose contractility is also poor when compared to the rest of the stomach. Later when it fills up it will pass into or ‘cascade’ over into the antrum and then pass into the pylorus. This effect is highly under recognized.

NORTH AMERICAN SUMMIT ON ASPIRATION

Enteral feeding recommendations in regard to residual volume:

1. Assess tolerance to feeds if the residual volume more than 500ml.
2. Stop feeds if patient develops severe regurgitation and aspiration.
3. If residual volume is less than 400ml it does not mean that the gut is tolerant to the feeds given and that aspiration possibilities are less.
4. If the residual volume level is 200-500ml then while continuing feeds careful clinical evaluation of the patient is needed and measures to prevent aspiration must be followed.
5. If the residual volume is less than 500ml it can be returned to the patient.

SUGGESTED GUIDELINES TO TREAT ELEVATED RESIDUAL VOLUME

1. Check: Is it a residual?
2. Clinical assessment of the patient for signs of intolerance to feeds.
3. Rule out cascade effect: Turn the patient to right side. Measure residual volume again
4. Reduce the amount of feeds without reducing the caloric value of the feed by increasing the concentration of the feed.
5. Drugs like anti emetics and prokinetics can be tried.

6. Guided placement of feeding tubes beyond the pylorus.
7. Avoid hyperglycemia induced gastoparesis by maintaining the sugar value of the feed less then 200mg/dl
8. Avoid opiate analgesia
9. Drugs to reduce endogenous gastric secretions like proton pump inhibitors can be used
10. Raise threshold levels for residual volumes
11. Stop checking residual volume levels once the patient tolerates feeds for more than 48 hrs.

VOMITTING AS A CONTRAINDICATION FOR FEEDING:

When patients are on enteral feeds some may develop vomiting as a reaction of intolerance to feeds. When vomiting is protracted switching the patient to total parenteral nutrition would be helpful. Management is similar to patients with significant residual volumes.

Anti emetics for supportive management can be given.

Concept of ileal brake:

When patients on long term jejunal feeds are initiated on oral feeds some may develop an increase in emesis.This is due to action of negative feedback mechanism initiated from the ileum in reaction to intact nutrients like fatty acids.

This causes gastroparesis and hence passage through the pylorus slows down in the long run. When such patients are started on oral feeds nausea, vomiting and fullness ensues.

Gradually patients improve over days and rarely over weeks. Those patients with bacterial overgrowth are high risk to get such symptoms.

Treatment involves conservative management and antiemetics. Prokinetics can be used, though their usefulness in such situations is questionable. Patients with the risk of bacterial overgrowth can be treated with antibiotics.

DOES ENTERAL NUTRITION CAUSES DIARRHEA

Another common complaint among patients receiving enteral nutrition is the occurrence of diarrhea. Most commonly the cause of diarrhea is other factors rather than enteral nutrition itself.

Prospective studies linking enteral nutrition and diarrhea are also absent. Further studies have also failed to prove statistically that other causes of diarrhea in enterally fed patients are hospital acquired.

Often diarrhea is related to the underlying disease of the patient or any infection which may be hospital acquired or as a side effect to medications or as a result of the surgery done or altered anatomy causes by the disease or surgery.

Also when patients are in nil per oral or on direct enteral feeds most drugs are given in the intravenous route. When oral feeds are initiated most drugs are switched from the parenteral route to oral. This sudden exposure of the bowel mucosa to the effect of drugs may also cause diarrhea.

Many oral preparations of drugs especially liquid forms contain the compound sorbitol. This is a known agent having a laxative effect on most individuals.

Also it is common to have antibiotic induced alteration in the composition of the gut mucosal flora causing easy susceptibility to infections like Clostridium dificile and hence diarrhea.

The process of digestion and absorption are intricately coordinated by speed of transit of foodstuffs, coordination of pancreatic and bile salt secretion, and the tremendous surface area dedicated to absorption (equivalent to approximately two tennis courts).

The site of absorption of digested feeds is predominantly the first three to four feet of the intestine. There has been a study which states that threshold for the occurrence of diarrhea in patients in total enteral nutrition is 275ml per hour. Also some studies have proven that about 60% of the ingested proteins can be digested even after the surgical removal of the pancreas.

A need to change the formula of the enteral nutrient being given is only when malabsoption develops and is documented, significant loss of function of the gut is suspected and medications as the cause has been ruled out.

Enteral nutrition can be continued in a patient with diarrhea provided severe intolerance does not develop and that the patient is monitored closely.

APPROACH TO A PATIENT WITH DIARRHEA WHEN ON ENTERAL NUTRITION:

1. Confirm diarrhea- calculate frequency of stools
2. Review the drugs taken by the patient

Commonly used drugs causing such effects include:

- Acetaminophen
- Theophylline
- Lactulose
- Stool softeners, laxatives, etc.

3. Rule out infectious causes
4. Add fiber to diet
    - Clinical studies establish its advantage in such situations
    - Supports the health of colonic flora
5. Add anti-diarrheal agent

TYPES OF TUBES:

NASO GASTRIC/ NASOENTERAL :

INTRODUCTION: Earliest description date from 17th century[20].

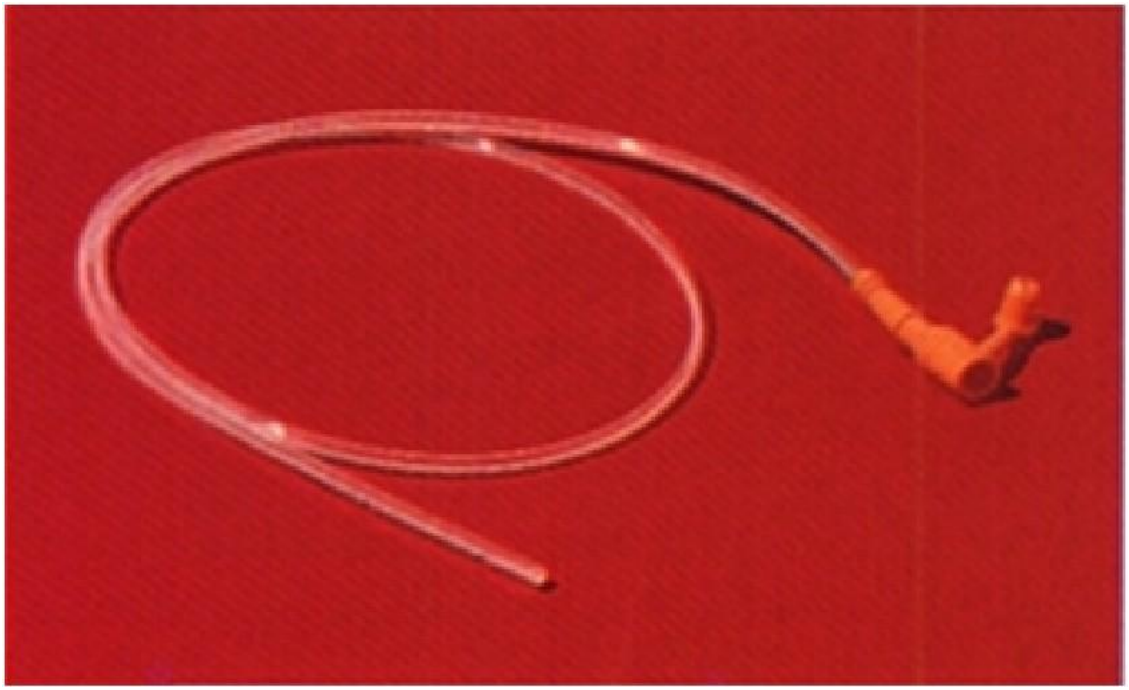
Feeding required for a period of 4-6 weeks can be achieved.

Types : 1) SALEM SUMP TUBE : first described in 1960's

Modification of Levin tube described by him in 1921.

Levin tube:- single lumen tube fenestrated at the distal end for decompression (or) feeding[21] .

Salem tube has second lumen that allows air to be drawn into stomach (or) sump during suctioning thereby avoiding adherence to gastric mucosa.



2) CANTOR INTESTINAL TUBE & BAKER JEJUNOSTOMY TUBE[22] :(Weighted, silicon models)

7 long nasoenteric tubes available as single/multi lumen tubes.

7 Those tubes have air filled (or) balance tipped ends and pass distally & provide intestinal decompression.

7 Very successful in treatment of partial obstruction[23].

Disadvantages:-

7 Lacks superiority over nasogastric decompression.

7 Time consuming.

7 Do not have decompression ports, may allow emesis & aspiration. 3)

DOBBHOFF TUBES:- Nasojejunal feeding tubes

- Smaller caliber, (7-9 french) & soft tubes[24] .
- Used for therapeutic purposes
- Feeding (short term nutritional support)
- Drug administration

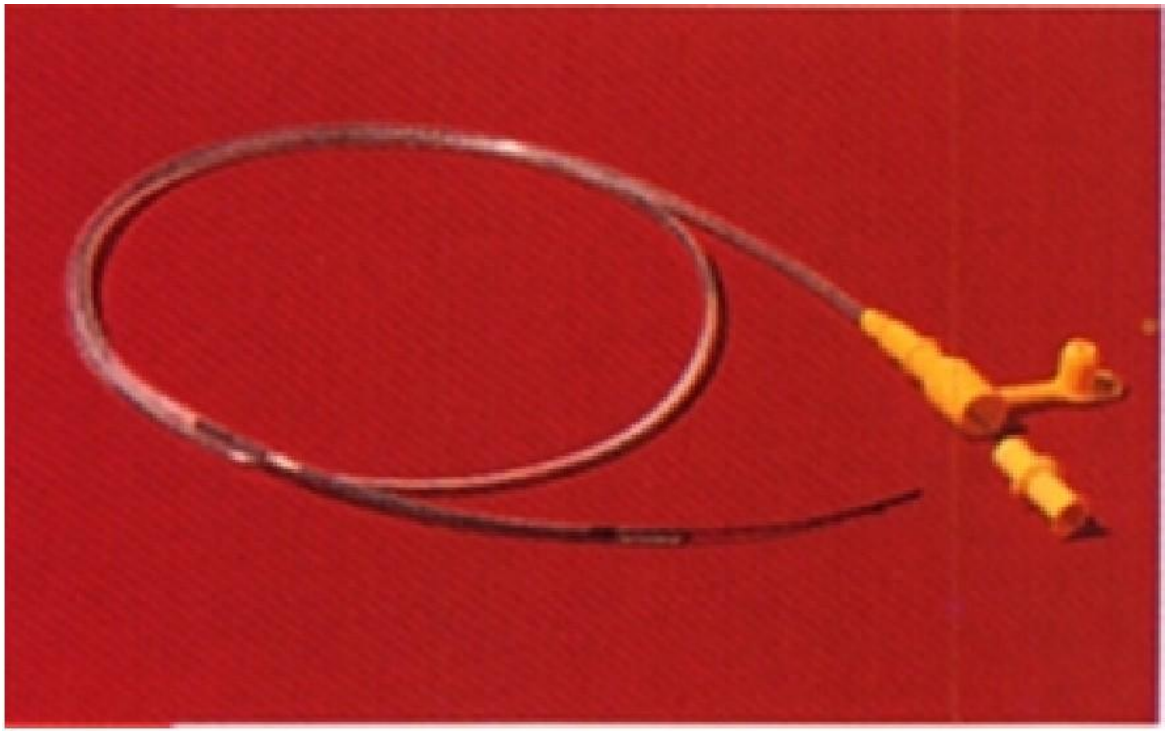
Transpyloric passage is seen in limited number of cases[25] .

COMPLICATIONS: Difficult Placement

Frequent extubation

Higher incidence of tube related complications

Delays nutritional support.



Access Techniques

NASO GASTRIC TUBES[26] :

Most common route of feeding.

Allows use of high concentrate feeds, high volume of feeds and

balloon feeding

INSERTION[27]:-

- Nasogastric tubes can be placed on the ward by trained personnel.
- Patient to be made aware of the procedure to be done
- Measure distance from xiphisternum to ear lobule to nostril and mark same in the tube.
- Lignocaine jelly to be applied on the outer aspect and flush the tube with water also.
- Check the tube for movement of the guide wire if it is present.
- Lignocaine sprayed in the nostril selected by sniff test
- Introduce the tube slowly in and if swallowing difficulty is experienced by the patient provide 5-10 ml water
- Flex the neck if difficulty in passage of tube occurs
- Continue till mark is reached
- Remove guide wire if present
- Fix tube
- Check position
- Note down in daily patient record.

NASOJEJUNAL TUBE INSERTION:-

INDICATIONS:

Problems with gastric reflux

Delayed gastric emptying

Poor GCS patients who require feeds

INSERTION :-

- Similar to nasogastric tube.
- Ensure position in stomach
- Turn patient to right
- Advance 10cm→fails→push 0.5-1L air into stomach→repeat
- Prokinetics can be used
- Proceed pushing in till markings
- Remove guidewire
- Secure tube
- Check position by x-ray
- Endoscopy guided placement →hold tube and guide in→risk of spontaneous removal when scope withdrawn
- Alternate: Endoscopy guided placement of guidewire into jejunum which inturn guides the feeding tube into place

FEATURES OF RECOMMENDED TUBES:

- Be radio Opaque
- Have multiple ports
- Display clear centimeter line markings.
- Have caps attached
- Be available in variety of materials which cater for different clinical situations.
- Be available in a number of lengths & sizes.

NASOGASTRIC TUBE INSERTION DOCUMENT ATION TO INCLUDE:

Date & time

Reason for insertion

Type of tube

Size of tube

Length of tube

Nostril tube inserted

Number of attempts required

Additional comments

Any complications

Method of placement confirmation

Signature: Name & designate sector investing tube.

• Never place anything into a nasogastric tube unless the tip is confirmed as being in the stomach.

• Not permitted to insert a nasogastric tube into patients with possible as confirmed facial/ skull fractures.

• No more than 3 attempts of nasogastric tube insertion are to be made by one doctor.

VERIFICATION OF TUBE PLACENENT :

RECOMMENDED :- Primary confirmation -7 Radiography

Secondary confirmation -7 Mark tube at exit rite

NOT RECOMMENDED METHODS: ,

Auscultatory Methods[28] blue food dye.

REMOVAL OF NASOGASTRIC TUBE :

-7 Disconnect drainage bag (or) feeding device

-7 Insuffiate 10-20ml (adult), l-5ml (child) of air into nasogastric

-7 Ask the pt to take deep breath.

-7 Coil the tube around gloved hand while pulling slowly & 3-

-7 seconds.

AVOIDANCE OF COMPLICATIONS:-

- Proper placement & maintenance
- Placing the patients bed in 30° head up position.
- Proper insertion technique.
- Assessment of placement

PREVENTING TUBE OCCLUSIONS [29] :

- Coagulation of protein based formula.
- Contact with acidic environment (or) medications.
- Routine water flushes (30ml) necessary
    - Every 4th hourly.
    - Before & after intermittent feeding/aspiration
    - Before & after individual medications.

GASTROSTOMY :

Intubation of the stomach (exclusive of the nasogastric route) results in planned gastrocutaneous fixtula.

ADVANTAGES: Low leak rate, less cost, ease of placement, placed adjunct with Gastro intestinal surgery. Spontaneous closure when removed.

DISADVANTAGES: Inadvertent tube removal results in rapid & pre mature loss of enteral access, risk of aspiration, stoma care needed, potential skin excoriation.

INDICATIONS: Head & Neck cancer. Cerebrovascular accident, trauma, respiratory failure. Prolonged intubation.

CONTRAINDICATIONS: Gastro esophageal reflux disease, gastroparesis, gastric out let obstruction, pancreatitis, recent foregut surgery.

OPEN GASTROSTOMY : STAMM METHOD :

-7 Gold standard for transabdominal gastric access.

-7 Requires small laparotomy. Stomach is accessed via a small upper mid line incision.Omentum and transverse colon identified and retracted inferiorly. A relatively avascular site is chosen along the anterior wall of stomach, away from antrum & pylorus. The exit site should be in left upper quadrant.

A large bore (22-24f) tube often with a balloon (or) mushroom tip is placed through the abdominal wall through separate stab incision. One (or) two purse string sutures are placed in seromuscular layer of anterior wall of stomach.

Create a gastrostomy in the middle of purse string suture. Insertion of the tube done. The balloon is inflated and the purse string sutures tied securely, anterior wall of the stomach affixed to abdominal wall entry site & tube secured to skin.

PERCUTANEOUS ENDOSCOPIC ¯GASTROSTOMY:' [31,32]

Indications:Patients requiring feeding for longer time

Altered level of consciousness.

Dysphagia secondary to orpharyngeal cancer

Gastric decompression

Neurologic event precludin g swallowing

Tracheo esophageal fistula.

Contraindications:

- Coagulation disorder
- Marked esophageal obstruction
- Massive ascites
- Obstruction & pseudo obstruction
- Peritoneal dialysis
- Peritoneal metastases
- Poor survival potential
- Respiratory distress
- Severe obesity.

Was introduced in 1980's by Gauderer and Ponsky

Functional upper GI tract & prolonged enteral feeding are essential requirements for PEG placements.

Permits feeding distally in the jejunum with gastric decompression. Well established & safe with minimum anaesthesia & complications. Currently method of choice for gastric intubation for nutritional support.

TECHNIQUE OF PEG[33] :

- PEG insertion requires an over night fasting.
- Single I.V. dose of a broad spectrum antibiotic to be effective in reducing the incidence of peristomal infections.
- Done by either of the following 3 techniques.
    1) Pull through technique(Pansy-Grauderer)
    2) Push technique (Vine)
    3) Introducer technique (Russel)

I) PULL THROUGH TECHNIQUE :

An endoscopist & assistant are required for the procedure.

1) Abdominal wall is prepared & draped.
2) Endoscope is passed & upper G.I. tract is surveyed to rule out gastric outlet obstruction.

3) Stomach is inflated with air, so that anterior wall of stomach juxtaposes anterior abdominal wall.

4) Tip of the endoscope 1s directed towards anterior abdominal wall for transillumination.

5) The indentation on the anterior wall of the stomach is seen by the endoscopist & thus appropriate site of gastrostomy is chosen.

6) Local anaesthetic is infiltrated at the site 0.5cm incision is made & deepened through the subcutaneous fat.

7) Abdominal wall operator pushes the 18 guage needle catheter. Through the incision into the stomach under endoscopic vision. Needle is removed heavil y cannula in place.

8) A 150 cm silk ligature passed throu gh the cannula into the stomach.

9) Endoscopist passes a snare through the scope. The silk ligature is grasped with the snare.

10) Endoscope along with the snare holding the ligature is with drawn out of patients mouth.

11) Ligature is tied to the tapered tip of PEG tube.

12) Abdominal operator removes the plastic cannula & applies steady traction on the ligature to pull the PEG tube, so that tapered end comes out of anterior abdominal wall & the mushroom tip snuguly apposes the anterior gastric wall.

13) The tube is anchored to the skin by plastic bloster.

14) External tube is cut to an appropriate length.

II)THE PUSH TECHNIQUE :

A soft guide wire is passed through the needle catheter into stomach lumen. The guide wire is pulled out of patients mouth using a snare. Tension is applied to both ends of the guide wire while the tapered end of the gastrostomy tube is passed over it & pushed down into the stomach till it comes out of anterior abdominal wall. The tube is then appropriately positioned so that inner bumper of the tube rests on inner gastric wall.

III)THE INTRODUCER TECHNIQUE:

A split sheath introducer is passed over. A J. tipped guide wire inserted into stomach lumen through a needle catheter. The guide wire and the introducer are removed and a 14 french foley catheter is fed through the split steath, which is ultimately feeled away.

REMOVAL OF PEG :

The internal bumper is snared and retrived by using endoscope after the external portion of the tube is cut. Alternatively it can be done with out using endoscope. The external portion of the tube is cut close to the abdominal wall. A foley catheter is placed into the fixtulas tract pushing the internal bumper into the stomach, which is then expelled into faces.

COMPLICATIONS [34]:

- early (within 14 days) - late (after 14 days).

-Minor complication: Tube related (dislodged tubes, leaks, wound infections, mucosal obstruction (buried bumper syndrome) & fever[35] . In 8% of the patients.

-Major complications: peritoneal leakage with peritonitis, necrotizing fascitis of anterior abdominal wall, gastric Haemorhage, Perforation of stomach & colon[36]

Worsening GERD after gastrostomy [37,38]in 1% of the patients.

LAPROSCOPIC GASTROSTOMY [39] **:**

General aneaesthemia & pneumoperitancum is required

Approximation of the stomach to the abdominal wall is accomplished with T- fasteners placed percutaneously Four T-fasteners placed around the prespective gastrostomy site. A gastrostomy tube is then placed percutaneously through the center of T-fasteners into gastric lumen. Stomach can be affixed to abdominal wall via T fasteners (or) sutures & further held in place with an intraluminal balloon.

FLUOROSCOPIC GASTROSTOMY [40] :

Retrograde fluoroscopic : percutaneous technique used.

Fluoroscopic visualization of a needle puncture of stomach. Creation of a tract over the guide wire done & tube fixed & anchored.

JEJUNOSTOMY :

INDICATIONS :

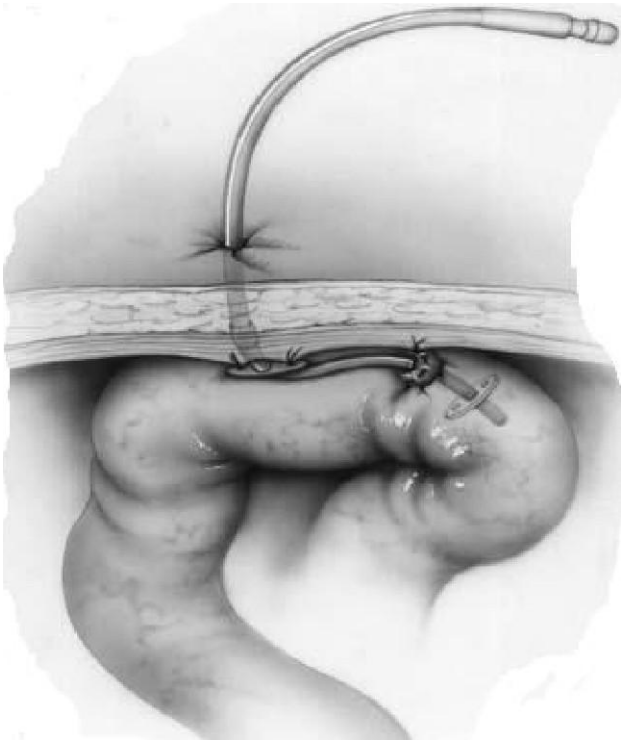
Recent surgery, Gastric outlet obstruction, gastroparesis, pancreatritis, fistula, esophageal reflux, high risk of aspiration.

CONTRA INDICATIONS:

Short bowel syndrome, distal obstruction, inability to provide continuous infusion.

LAPROSCOPIC JEJUNOSTOMY [41] :

'T' fastness placed into antimesenteric border of small bowel under direct laproscopic visualization. An introducer with a peel away sheath is placed into the Jejunum through abdominal wall. The 'T' festers are cut at skin level, 10 -7 14 days later.

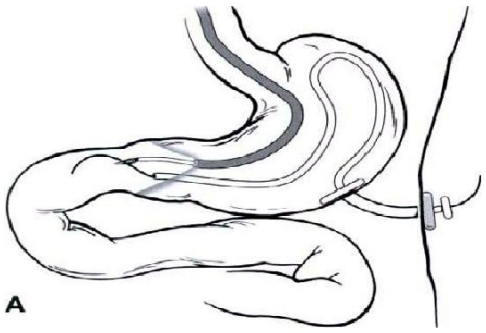


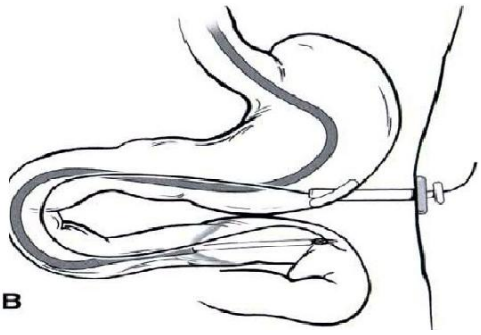
OPEN (WITZEL) JEJUNOSTOMY:

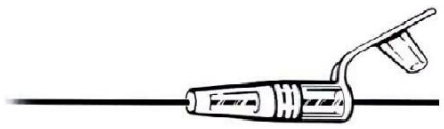
Laparotomy via a small upper midline incision.Site 15-20cm distal to ligament of treitz. Purse string suture placed on anti mesenteric border of jejunum. 14 F silastic. Tube is passed through the adjacent stab incision in soft upper quadrant. Enteretomy is created through purse string. Purse string suture is tightened, and a serosal tunnel is created p roximally for approximately y 3-5cm. Several sutures are used to affix the jejunum to the parietal peritoneum of the anterior abdominal wall at its exit site.

PEGJ PEJ CONVERSION [42] :-

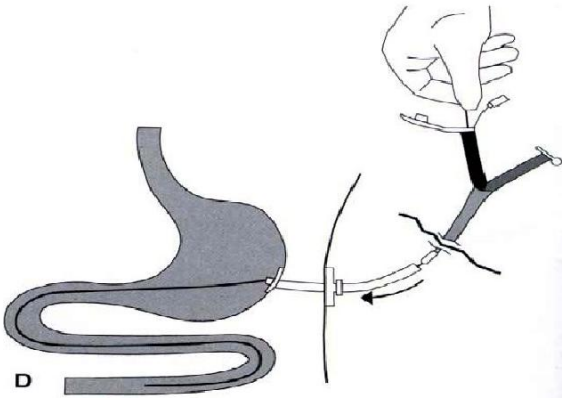
Jejunostomy tube can be placed under endoscope guidance through an established gastrostomy tract after removing the gastrostomy tube. The jejunal tube is fed through gastrostomy tract (or) tube into the stomach.The tube is caught in a snare passed through the endoscope and it is then advanced across the pylorus into the second (or) third part of the duodenum.Endoscope is with drawn taking care not to dislodge the tube.







C



COMPLICATIONS OF ENTERAL NUTRITION [43] :

I) GASTROINTESTINAL COMPLICATIONS [44,45] :

1) NAUSEA & VOMITING:-

- 20% experience this complication
- It increases risk of aspiration
- Commonly caused by delayed gastric emptying[46] .

To avoid vomiting: review patient medication (narcotics) and reduce feeds if required.

2) DIARRHOEA:

-Most common in tube fed patients, occurring in 2% to 63% of patients.

-If patient develops protracted diarrhea

- Add fiber eg : psylium.
- Consider an enteral formula with fiber.
- Change to formula.
- Use an antidiarrheal agent.

3) CONSTIPATION:-

-Common causes are

a) Lack of fiber in diet

b) inadequate fluids

c) factors causing reduction in bowel motility

d)inactivity

-To avoid constipation:Add stool softeners and purgatives,increase fluid intake, add fiber in diet, use prokinetics

4) MALABSORPTION /MALDIGESTION:

-Involves reduced or altered absorption of feeds

-Symptoms:a)weight loss

b)steatorrhoea

c)increased stool frequency

d)pallor

e)glossitis

f)oedema.

II) MECHANICALCOMPLICATIONS :-

1) ASPIRATION:

- Aspiration of enteral content into the lungs may lead to pneumonia and may be deteriorating to life
- symptoms include dypsnoea, Tachypnoea, wheezing rales, Tachycardia, agitation and cyanosis.

Risk factors for aspiration include:

- Diminished gag reflex
- Neurologic injury
- Incompetent lower esophageal sphincter
- Use oflarge bore feeding tubes.
- Large gastric residuals.
- upper & lower air way complications

III) TUBE CLOGGING :

-More common with protein feeds and thick feeds.

-Prevention of clogging can be done by

a)minimal forceful insertion of warm water into the feeding tube

b)add sodabicarbonate into the tube

c)add pancreatic lipase into the tube

These would disintegrate the clog.

IV)METABOLIC COMPLICATIONS :

Complications	Reason	Management
Hyponatremia	Overhydration	Change formula and restrict feeds
Hypernatremia	Inadequate fluid intake	Increase free water
Dehydration	Diarrhea and inadequate fluid intake	Evaluate causes of diarrhea, Increase free water
Hyperglycemia	Too many calories and Lack of adequate nutrition	Evaluate calorie intake and adjust insulin
Hypokalemia	Refeeding syndrome	Replace K^+ and evaluate causes of diarrhea
Hyperkalemia	Excess K^+ intake and renal insufficiency	Change formula

V)REFEEDING SYNDROME'[47]:

Definition: Sudden drop in levels of potassium, magnesium and phosphate in ill nourished patients introduced to enteral feeds

Complications :

a)Cardiac dysarrthmias

b)Cardiac failure

c)Acute respiratory failure

d)Coma

e)Paralysis

f)Acute renal failure

g)Acute hepatic dysfunction

RECOMMENDATIONS TO REDUCE THE RISK: - Prior identification of patients at risk:

Anorexia nervosa

Classic kwashiorkor (or) Marasmus Chronic malnutrition

Chronic alcoholism Prolonged fasting

Significant stress & depletion.

Correct electrolyte abnormalities before starting nutritional support.

Administer volume & energy slowly

Provide appropriate vitamin supplementation and avoid over feeding.

Aim of the study

To compare early enteral feeding with milk based standard feed and late enteral feeding in patients undergoing upper gastrointestinal surgeries

Objectives:

When compared to late enteral feeding

1. To study the impact of early feeding on duration of **paralytic ileus** and start of oral feeds following upper gastrointestinal surgery.

2. To study the rate of **anastamotic leak** after start of early enteral feeding

3. To study the rate of **wound infection** after starting early enteral feeding

4. To compare the incidence of septic complications like **pneumonia and urinary tract infection** in patients on early enteral feeding

4. To compare **duration of hospital stay**

A.Inclusion criteria:

1. Age between 20 and 60 years
2. Any sex
3. Patients undergoing the following surgeries
    a. Elective Gastrectomy for benign/malignant causes
    b. Elective Gastojejunal anastamosis for benign/malignant causes
    c. Emergency omental patch closure for duodenal/gastric perforations
    d. Emergency gastrectomy for benign/malignant causes
4. Patients consented for inclusion in the study

B.Exclusion criteria:

1. Age <20yrs or >60yrs
2. Patients with following co-morbid medical conditions-cardiac/renal/hepatic dysfunction
3. Patients who are lactose intolerant
4. Patient in severe shock, intestinal ischemia,short bowel syndrome
5. Patients undergoing gastrointestinal surgeries other than those mentioned in the inclusion criteria
6. Previous history of gastrointestinal surgery or peritonitis
7. ECOG (Eastern Cooperative Oncology Group) performance status 4
8. Patient not consented for inclusion in the study.

MATERIALS AND METHODS

Type of study: prospective, randomised, control study

50 patients included in this study are divided into two cohorts:

1)Study Group-25

2)Control Group-25

Protocol for patients in study group:In all patients, intra operatively a 12Fr nasojejunal tube with a length of 120cm and made of polyurethane is inserted by another medical personnel through one of the nostril and is slowly introduced forward. The surgeon feels for the entry of the tube at the oesophageal hiatus. Once the tube is felt it is gradually guided into the efferent loop of the gastrojujenostomy in patients undergoing gastrectomy with gastrojejunostomy or gastrojejunostomy only and in patients undergoing omental patch closure for a perforated duodenal ulcer it is guided into the jejunum. The tip of the tube must be 20cm distal to the anastamotic site into the jejunum. For decompression of the stomach, a nasogastric tube may be through the other nostril.

Feeds started through the nasojejunal tube from the 12^{th} post operative hour.

Patient is kept in the semi recumbent position of 45 degrees while giving feeds so that aspiration is prevented.

Post operative period	Feed strength	Rate of feeds
12-24 hours	Normal saline and 5% dextrose Ratio 1:3	100ml/hr
24-48 hours	Half strength	50ml/hr
48-72 hrs	Half strength	100ml/hr
72 hours onwards	Full strength	100ml/hr

Composition of feed:

Content	Amount
Milk powder	150g
Sugar	50g
Vegetable oil	20g
Water	1L

Monitor the patient for side effects:

Look for the following:

1)Abdominal cramps

2)Abdominal distension

3)Ileus

4)Diarrhoea(3 stools per day)

Management of adverse effects:

Reduce infusion rate by 20ml per hour

If no improvement→Stop feeds temporarily for 6-12 hours.

Once symptoms subside → Restart feeds.

Removal of nasogastric tube: Follow routine protocol.

Protocol for patients in control group:

Routine management by nil per oral, intravenous fluids, antibiotics and frequent clinical monitoring for passage of flatus and bowel sounds.

Oral feeds are started once the patient is deemed fit clinically for feeds.

Nutritional parameters monitored are

1)Weight

2)Haemoglobin

3)Serum albumin

These three parameters are monitored

a) pre operatively

b) post operatively day1

c) post operatively day 7

ClinicalParameters monitored are

1)Duration of paralytic ileus

2)Anastamotic leak

3)Wound infection

4)Septic complications- Pneumonia and Urinary tract infections

5)Duration of hospital stay

6)Time taken to start oral feeds

Relevant clinical parameters are checked for three times per day.

All patients are given post operative antibiotics(combination of a third generation cephalosporin and metronidazole).

No oral/intravenous/ rectal agents to stimulate bowel motility are given.

Caloric value of feed

Content	Amount in feed (g)	Kcal per 100g	Kcal in the feed
Milk powder	150	470	705
Sugar	50	388	194
Vegetable oil	20	884	176.8
Total kcal =			1075.8

Protocol

Post operative period(hrs)	Feed given	Amount per 24 hrs (ml)	Kcal
12-24	NS:5%D 1:3 ratio 100ml/hr	2400ml	306
24-48	Half strength feed 50ml/hr	1200	645.48
48-72	Half strength feed 100ml/hr	2400	1290.96
72 onwards	Full strength feed 100ml/hr	2400	2581.92

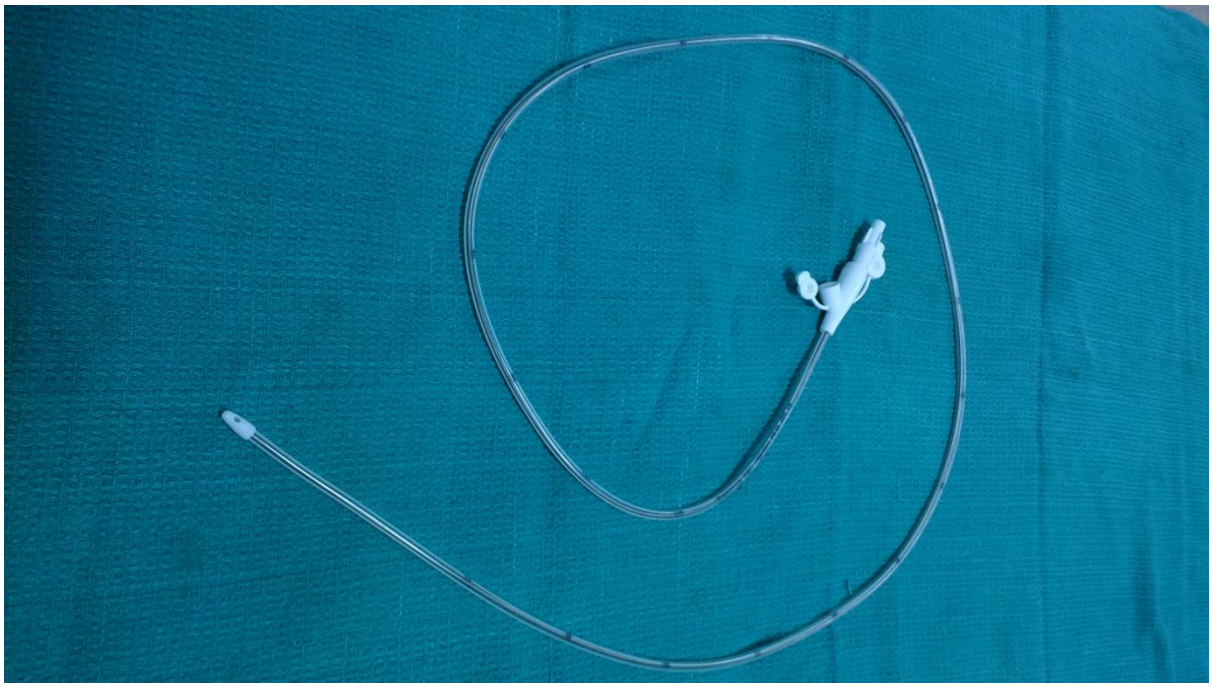
Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using SPSS 16 and Sigma Stat 3.5 version.

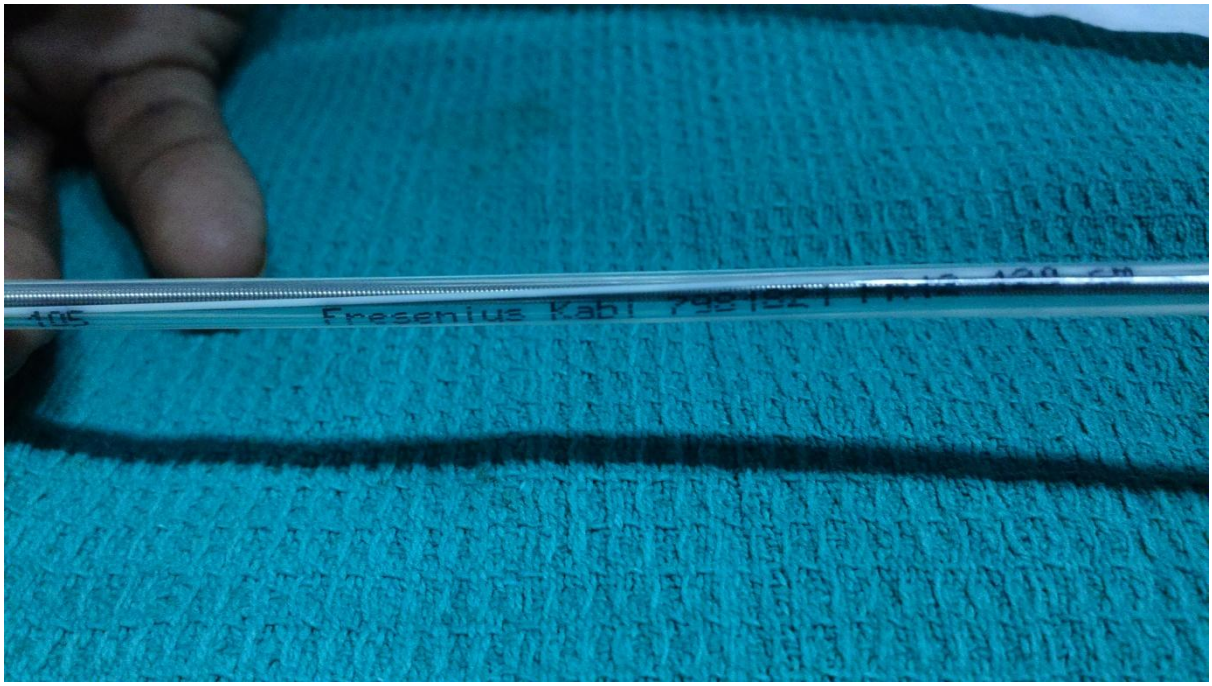
Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated by One way ANOVA and ‘t’ test. Kruskul Wallis Chi-square test was used to test the significance of difference between quantitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

Naso jejunal tube

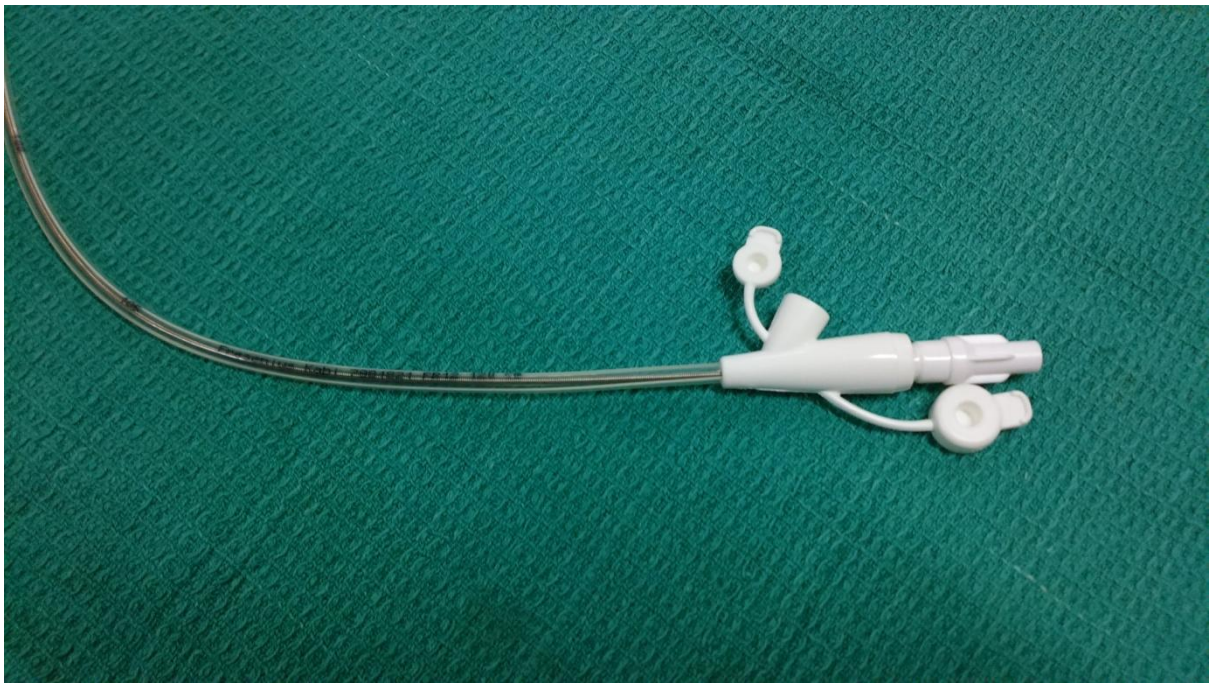


Fresenius Kabi Freka tube-12F-120cm-Polyurethane

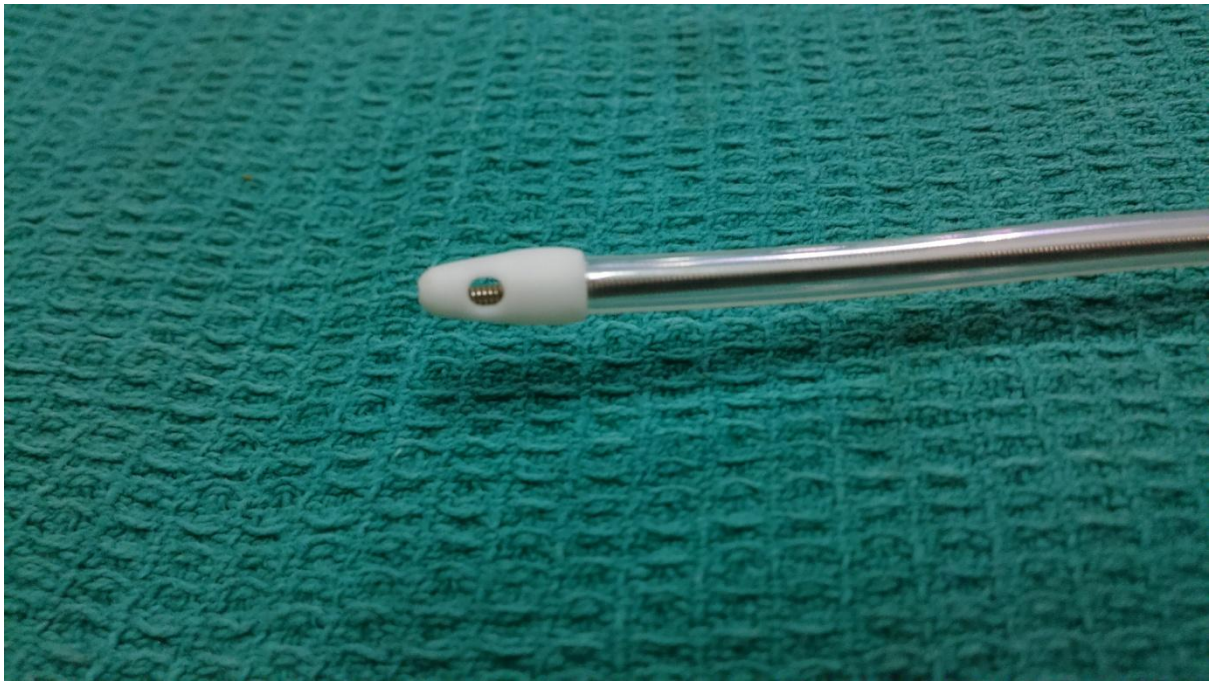


Radio opaque tube with markings every 10cm

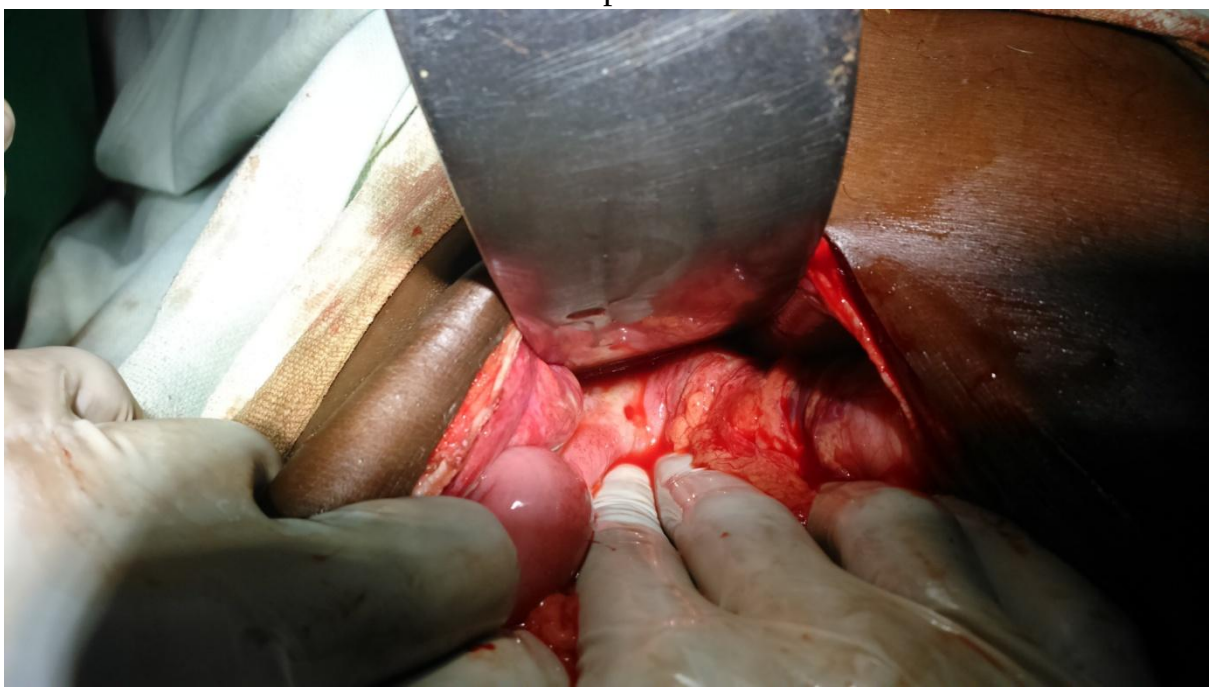
Y inlet with Leur lock adapter with integral cap and guide wire in place



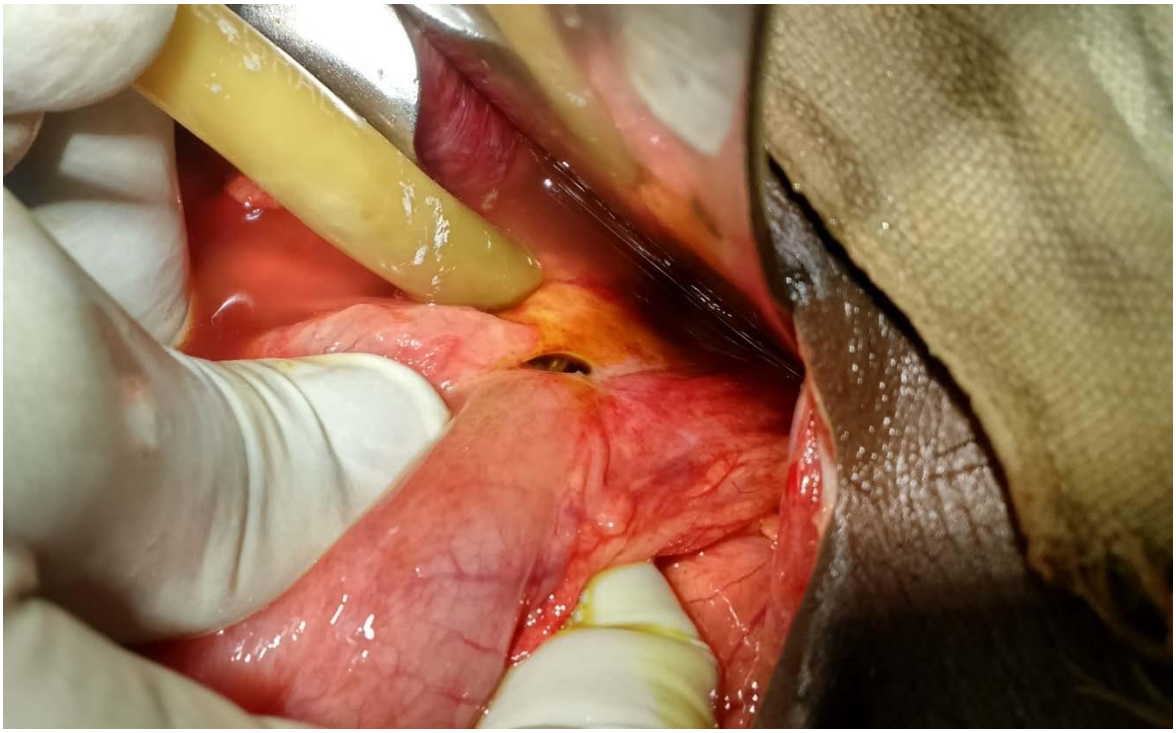
Outlet port with round closed end



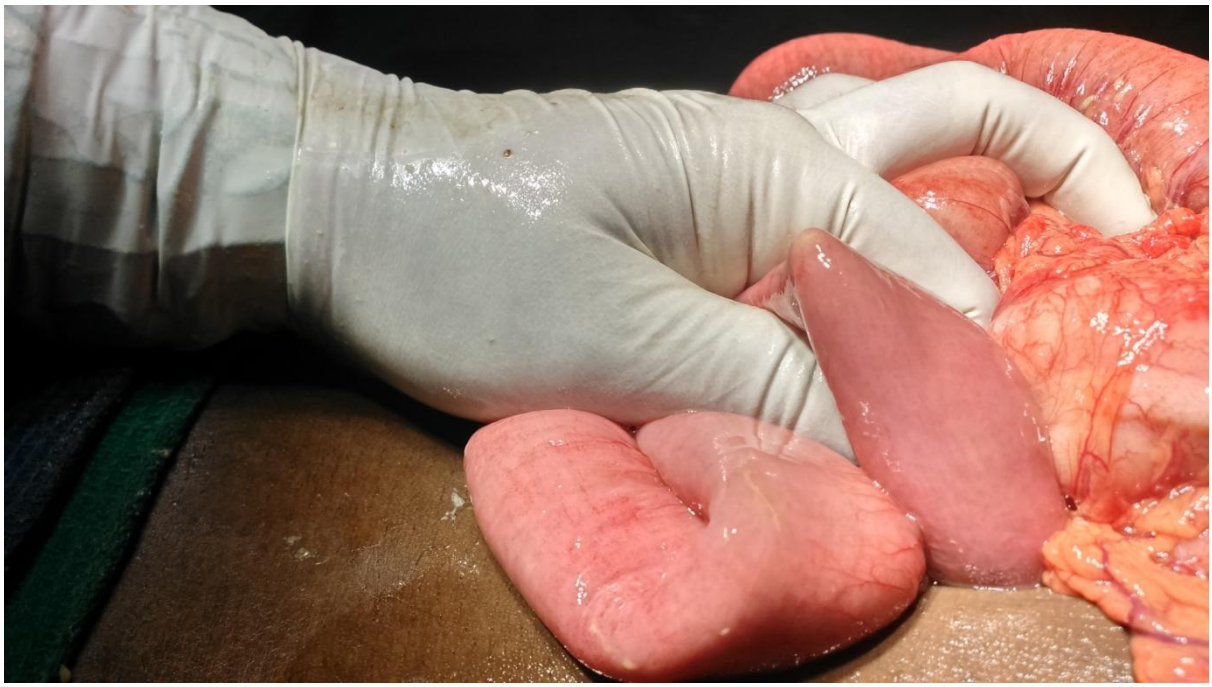
Perforation in the 1st part of the duodenum



Naso jejunal tube seen through the perforation



Tip of the nasojejunal tube seen entering the jejunum



Both Nasogastric ans Nasojejunal tubes in place

Feeding in progress

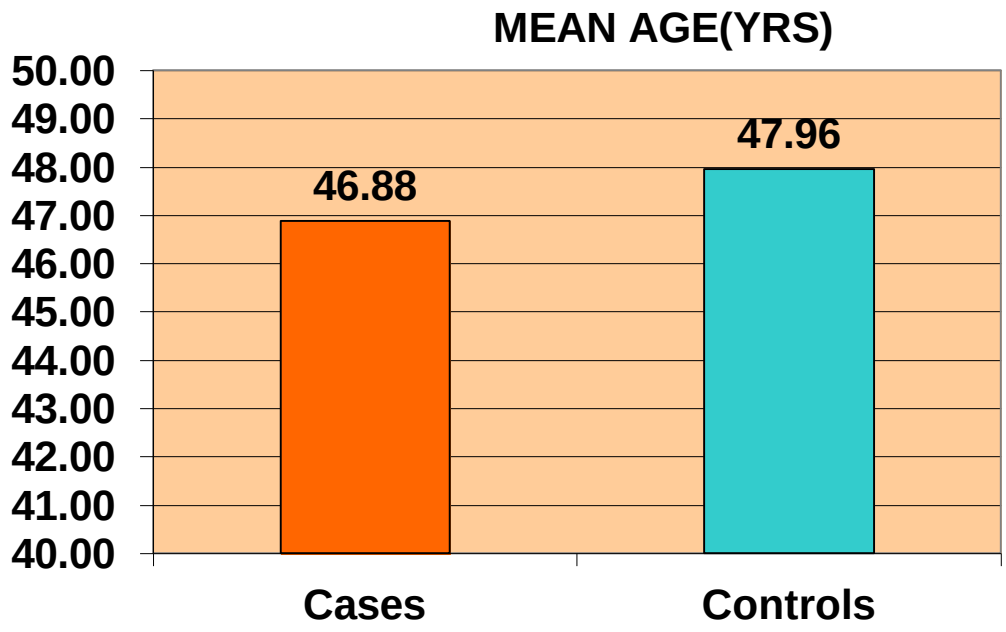
Results

The results of analysis of comparing patients started on early enteral feeding (cases) and patients treated in the conventional manner of late feeding (control) are as follows

Table 1

Age	Mean	SD	p' value
Cases	46.88	13.23	0.753
Controls	47.96	10.75	Not significant

Chart 1

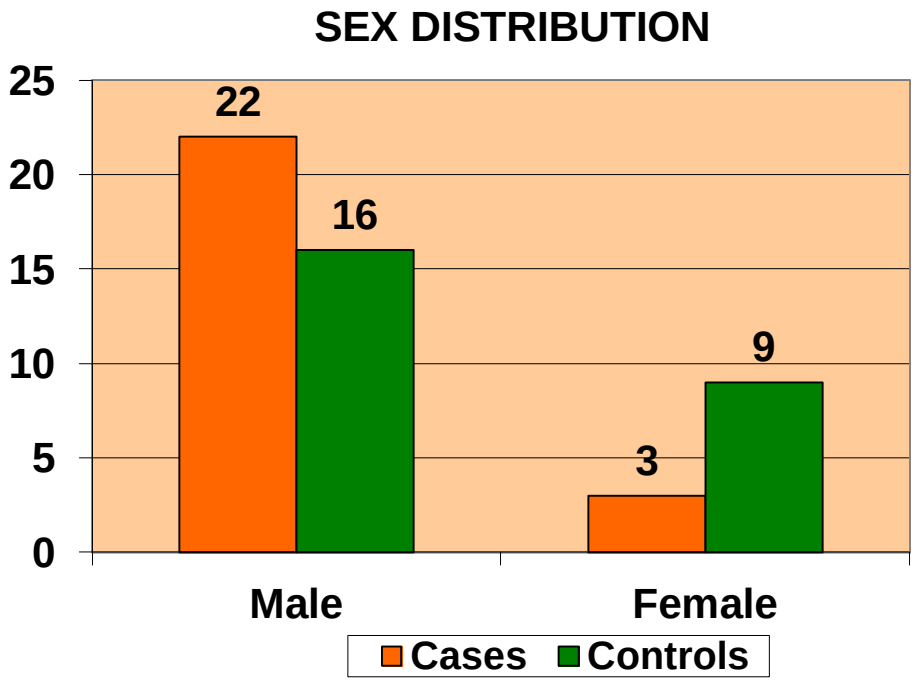


The mean age of the patients started on early feeding was 46.88 yrs and that of the control group was 47.96 yrs. The difference in age group is not statistically significant as the p value is 0.753.

Table 2

Sex	Cases	Controls	p' value
Male	22(88%)	16(64%)	0.603
Female	3(12%)	9(36%)	NotSignificant
Total	25(100%)	25(100%)	

Chart 2



The total number of patients was 50(study cases-25 and control-25).

Among them the male and female distribution was 22(88%) and 3(12%) in the study cases respectively and 16(64%) and 9(36%) in the control group respectively. The difference in age distribution is not statistically significant as the p value is 0.603.

Table 3

Pre operative Weight(kg)	Mean	SD	p' value
Cases	57.56	7.02	
Controls	55.96	5.40	Not significant

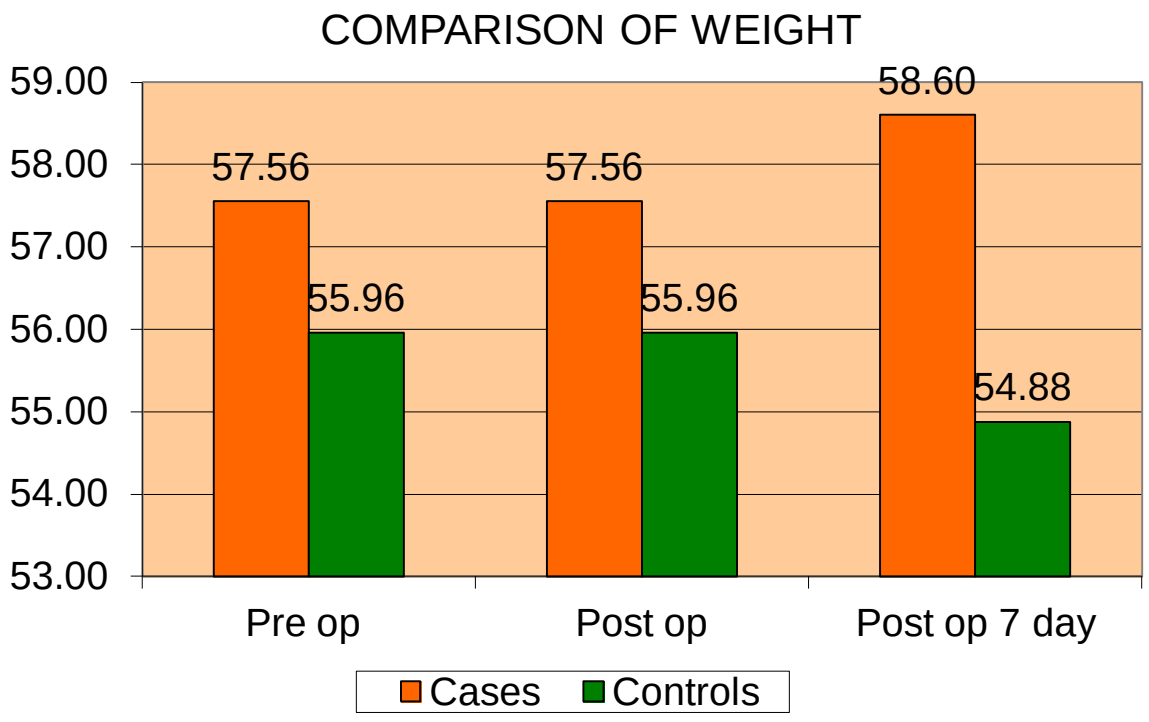
Table 4

Post operative Day 1 Weight(kg)	Mean	SD	p' value
Cases	57.56	7.02	0.371
Controls	55.96	5.40	Not significant

Table 5

Post operative Day 7 Weight(kg)	Mean	SD	p' value
Cases	58.60	6.72	0.032
Controls	54.88	5.10	Significant

Chart 3



The mean weight of the study cases pre operatively was 57.56 kg. There was no change in weight on post operative day1. But the weight increased to a mean of about 58.6kg by post operative day 7. While comparing the same parameter in the patients of the control group, the mean pre operative weight was 55.96kg, the same on post operative day 1 and reduced to 54.88kg by postoperative day7. This difference in post operative weight on day 7 is significant statistically as the p value is0.032.

Table 6

Pre operative Haemoglobin (g%)	Mean	SD	p' value
Cases	9.70	0.81	0.632
Controls	9.60	0.65	Not significant

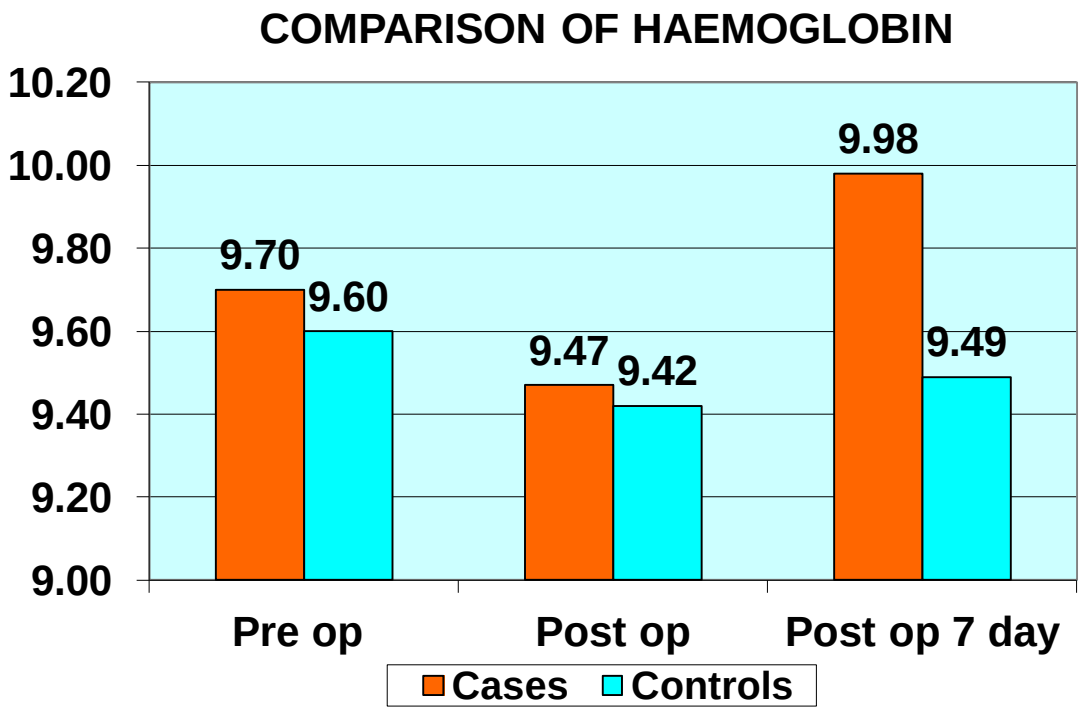
Table 7

Post operative Day1 Haemoglobin (g%)	Mean	SD	p' value
Cases	9.47	0.80	0.813
Controls	9.42	0.61	Not significant

Table 8

Post operative Day 7 Haemoglobin(g%)	Mean	SD	p' value
Cases	9.98	0.74	0.011
Controls	9.49	0.56	Significant

Chart 4



The mean pre operative haemoglobin among the cases in study group was 9.7g%. There is no significant change in post operative day , but the levels increased to 9.98g% by post operative day7. But in the control group the mean preoperative haemoglobin was 9.6g%, on post operative day 1 was 9.42g% and by post operative day7 was 9.49%.This is statistically significant as the p value is 0.011 is post operative day7.

Table 9

Sr. Albumin g/dl	Mean	SD	p' value
Cases	2.74	0.27	0.125
Controls	2.85	0.27	Not significant

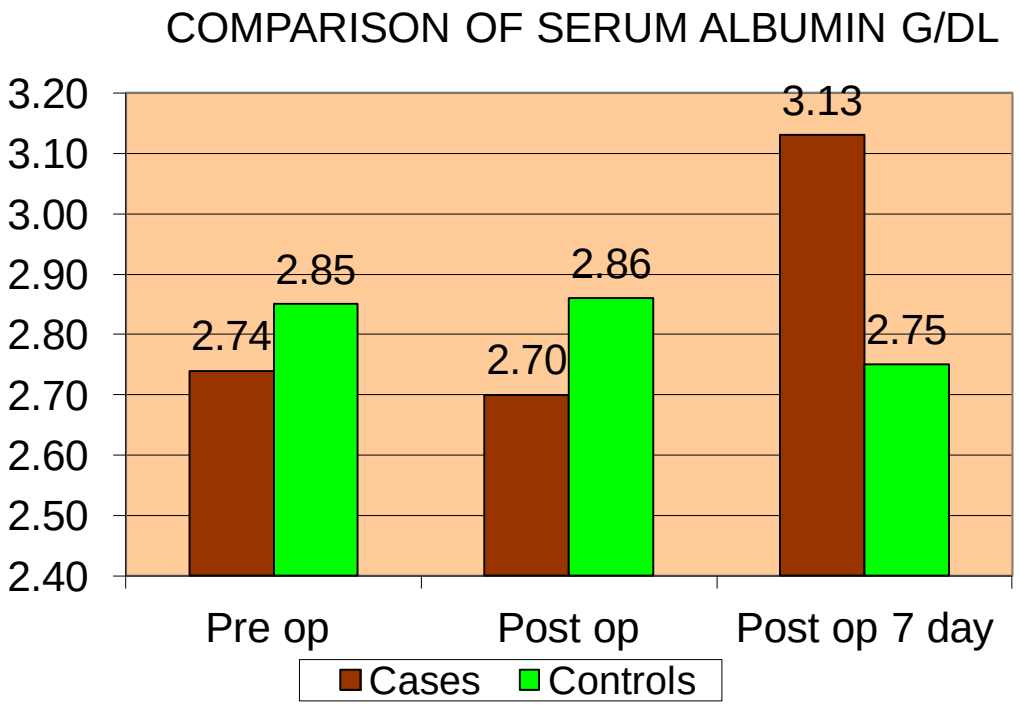
Table 10

Post op Sr. Albumin g/dl	Mean	SD	p' value
Cases	2.70	0.27	0.052
Controls	2.86	0.27	Not significant

Table 11

Post op day 7 Sr. Albu	Mean	SD	p' value
Cases	3.13	0.26	< 0.001
Controls	2.75	0.27	Significant

Chart 5

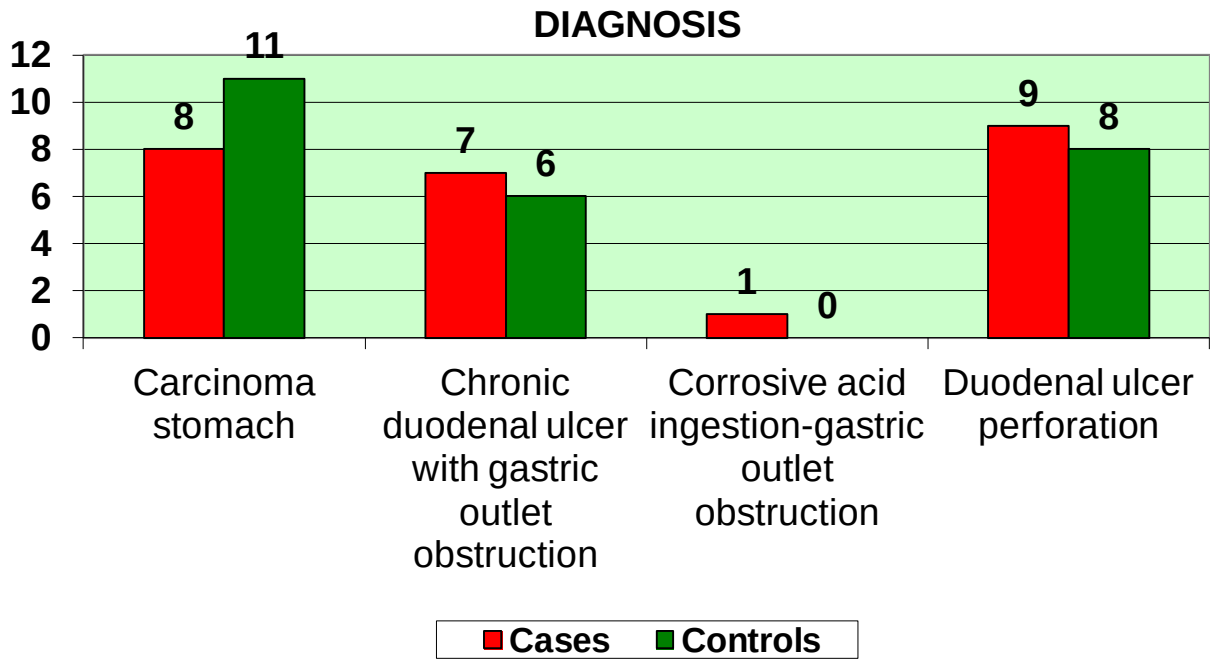


The pre operative S.albumin levels among the patients started on early feeding were 2.74g/dl. On post operative day1 the same was 2.7g% and by post operative day7 it was 3.13g%. Among the control cases the mean preoperative S.albumin levels was 2.85g/dl. On post operative day1 it was 2.86g/dl and by post operative day7 it was 2.7g/dl. This is statistically significant as the p value is <0.001 .

Table 12

Diagnosis	Cases	Controls
Carcinoma stomach	8(32%)	11(44%)
Chronic duodenal ulcer with gastric outlet obstruction	7(28%)	6(24%)
Corrosive acid ingestion-gastric outlet obstruction	19(4%)	0
Duodenal ulcer perforation	9(36%)	8(32%)
Total	25(100%)	25(100%)

Chart 6

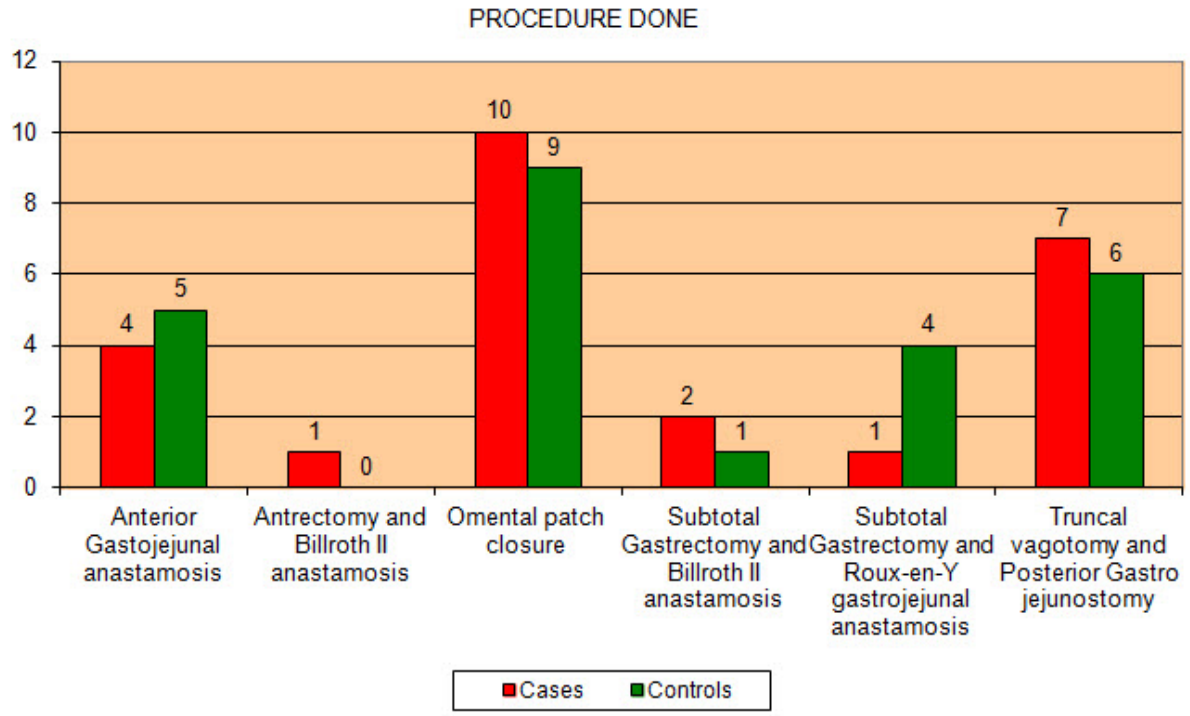


The diagnosis of carcinoma stomach was seen in 8(32%) and 11(44%) patients in the study and control group respectively. Among those in the control group one case presented as gastric perforation with a growth palpable intraoperatively and hence a emergency gastrectomy was done. Chronic duodenal ulcer with gastric outlet obstruction was the diagnosis in 7(28%) and 6(24%) in the study and control groups respectively. Corrosive acid ingestion causing gastric outlet obstruction in the long run was one (4%) of the cases in the study group. Duodenal ulcer perforation was the diagnosis in 9(36%) and 8(32%) patients in the study and control groups respectively.

Table 13

Procedure Done	Cases	Controls
Anterior Gastojejunal anastamosis	4(16%)	5(20%)
Antrectomy and Billroth II anastamosis	1(4%)	0
Omental patch closure	10(40%)	9(36%)
Subtotal Gastrectomy and Billroth II anastamosis	2(8%)	1(4%)
Subtotal Gastrectomy and Roux-en-Y gastrojejunal anastamosis	1(4%)	4(16%)
Truncal vagotomy and Posterior Gastro jejunostomy	7(28%)	6(24%)
Total	25(100%)	25(100%)

Chart 7



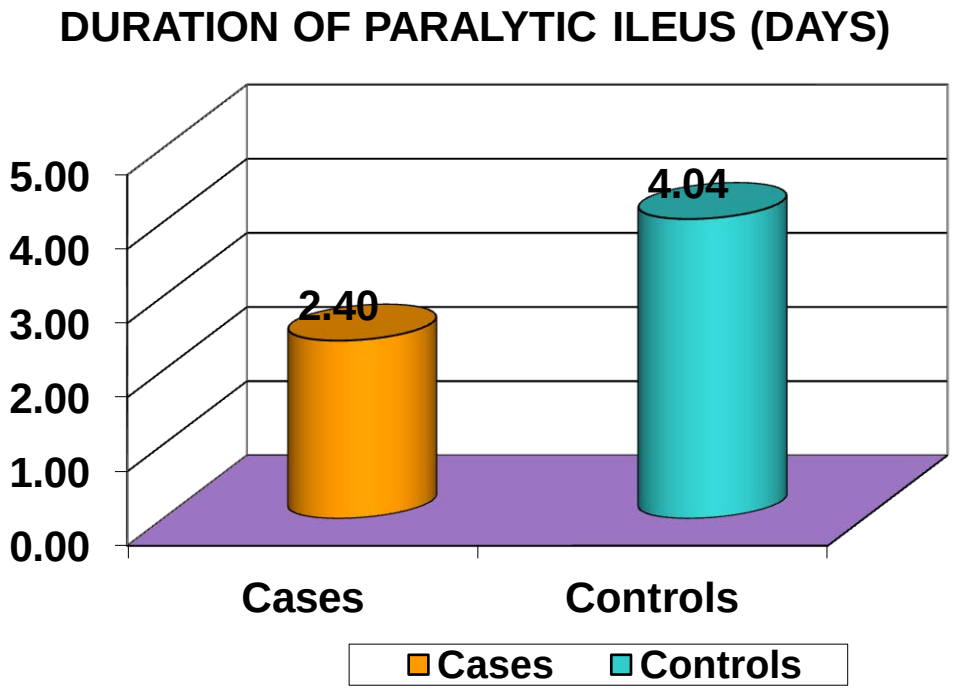
For all inoperable Carcinoma stomach cases requiring a diversion procedure only, anterior gastrojejunal anastamosis was done in the conventional 4 layer method for 4(16%) and 5(20%) cases in the study and control groups respectively. For some operable cases Subtotal Gastrectomy and Billroth II anastamosis was done in 2(8%) and 1(4%) in the study and control groups respectively. For the other operable cases Subtotal Gastrectomy and Roux-en-Y gastrojejunal anastamosis was done in 1(4%) and 4(16%) cases in the study and control groups respectively. Antrectomy and Billroth II anastamosis was done for the case diagnosed with corrosive acid ingestion with gastric outlet

obstruction. For all cases diagnosed with chronic duodenal ulcer with gastric outlet obstruction, Truncal Vagotomy with Posterior Gastrojejunostomy was done in 7(28%) and 6(24%) cases in the study and control groups respectively. When patients were diagnosed with Duodenal ulcer perforation on emergency laparotomy, Omental patch closure was done in the conventional manner in 10(40%) and 9(36%) cases in the study and control groups respectively.

Table 14

Duration of paralytic ileus(days)	Mean	SD	p' value
Cases	2.40	0.57	< 0.001
Controls	4.04	0.89	Significant

Chart 8



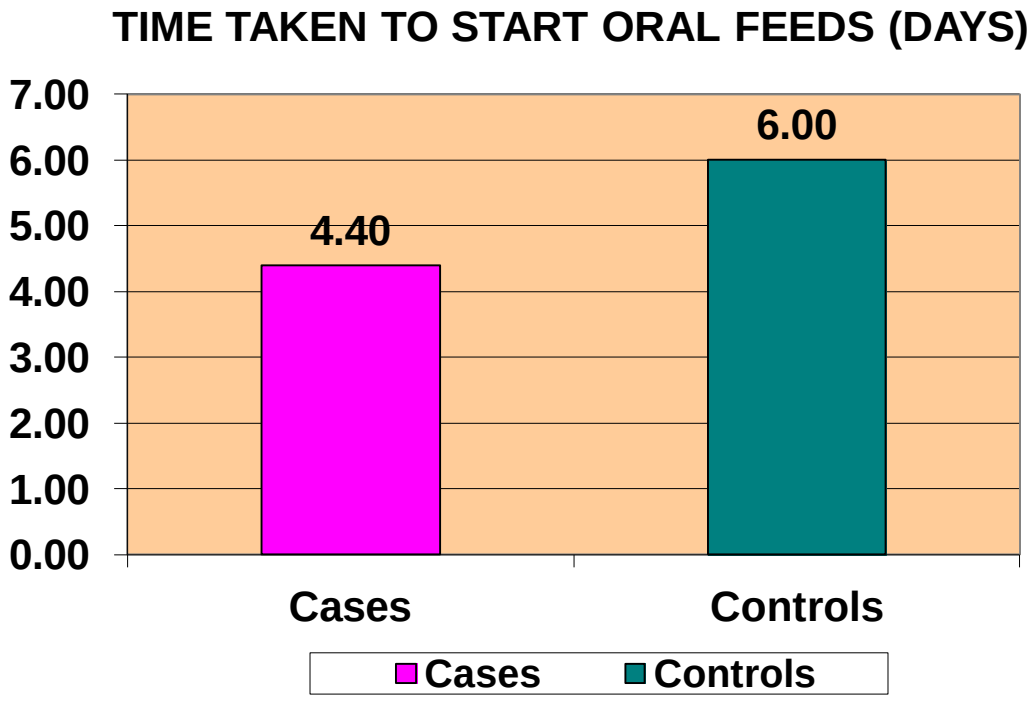
The mean number of days of paralytic ileus among the cases started on early feeding in the study group was 2.4 days while it was 4.04 days among the cases

started on late feeding in the control group. Since the p value is <0.001 the the difference is statistically significant.

Table 15

Time taken to start oral feeds(days)	Mean	SD	p' value
Cases	4.40	0.58	< 0.001
Controls	6.00	1.16	Significant

Chart 9

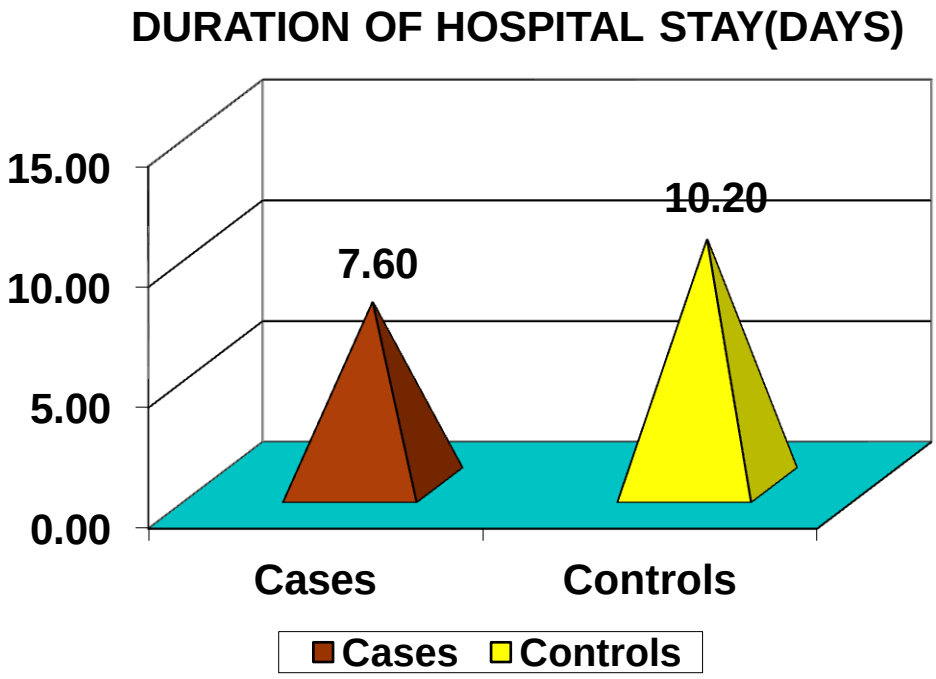


The mean number of days to start oral feeds among the cases started on early feeding was 4.4 days while it was 6 days among the cases in the control group. The p value being <0.001 the difference is statistically significant.

Table 16

Duration of Hospital stay(days)	Mean	SD	p' value
Cases	7.60	0.82	< 0.001
Controls	10.20	2.04	Significant

Chart 10

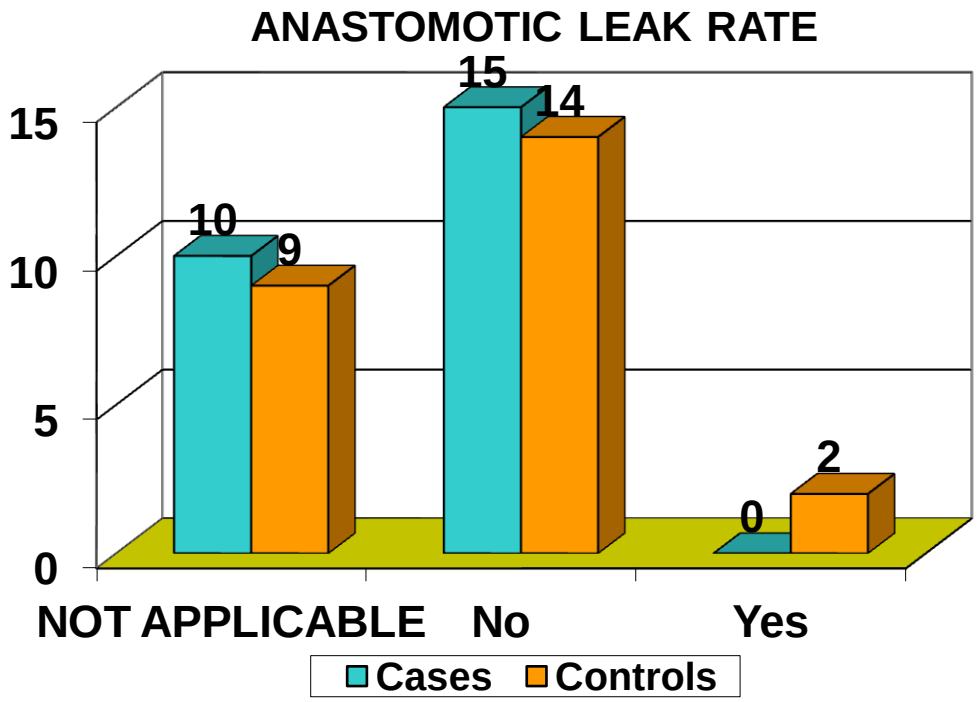


The average number of days of stay in the hospital among the patients initiated on early feeding was 7.6 days. The same among the patients in the control group was 10.2 days. Since the p value was <0.001 the difference is statistically significant.

Table 17

Anastomosis leak rate	Cases	Controls	
NOT APPLICABLE	10	9	p' value
No	15	14	0.505
Yes	0	2	Not Significant

Chart 11

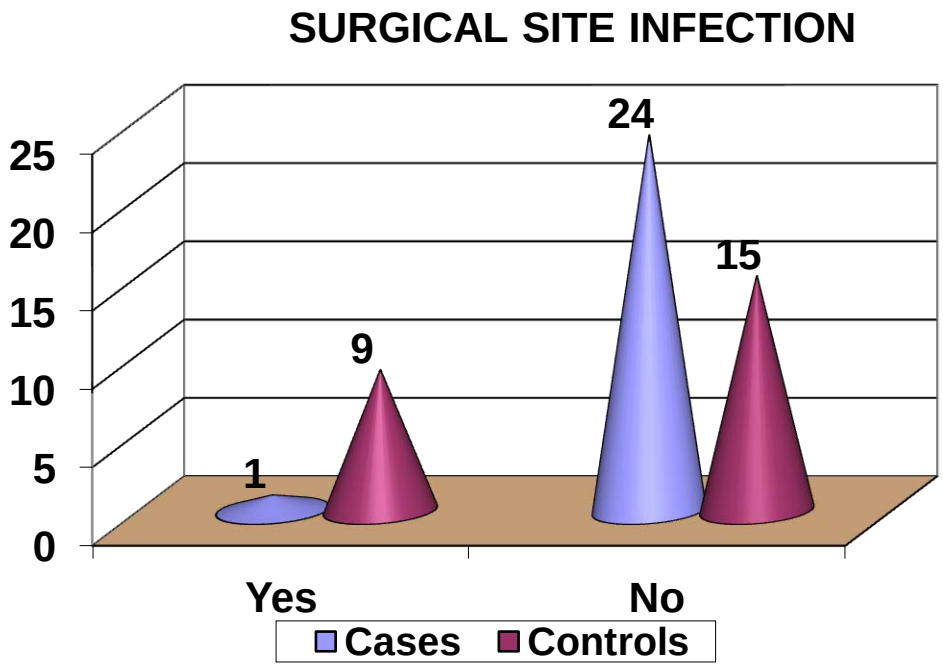


Among all the cases operated, 15 in the study group and 16 in the control group involved bowel anastamosis. None of the cases in the study group who were started on early feeding developed anastamosis leak. Among the patients in the control group only 2 cases developed leak at the anatamosis site. Both were operated for carcinoma stomach. Both underwent Subtotal Gastrectomy with Roux-en-Y anastamosis. Since the p value is 0.505 this difference is not statistically significant.

Table 18

Surgical site infection	Cases	Controls	p' value
Yes	1(4%)	9(36%)	0.033
No	24(96%)	15(64%)	Significant
Total	25(100%)	25(100%)	

Chart 12



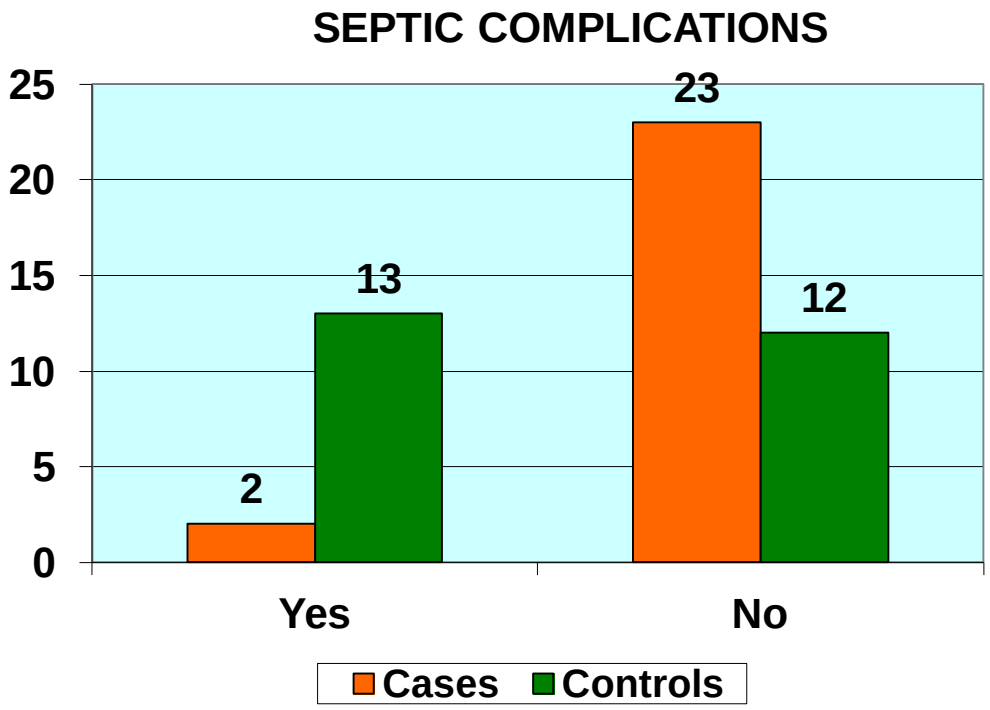
Patients started on early enteral feeding showed a significantly lesser rate of surgical site infection as only one(4%) among the 25 cases developed infection.

Whereas in the control group were late feeding was the rule, 9(36%) of the patients developed infection of the surgical site. This difference is statistically significant as the p value is 0.033.

Table 19

Septic complications	Cases	Controls	p' value
Yes	2(8%)	13(52%)	0.037
No	23(92%)	12(48%)	Significant
Total	25(100%)	25(100%)	

Chart 13



Septic complications like post operative pneumonia and urinary tract infection developed in one 2(8%) of the cases in the study group with the rest 23(92%) of the cases having a normal post operative period. Among the control group patients about 13(52%) of them developed septic complications. This is statistically significant as the p value is 0.037.

Table 20

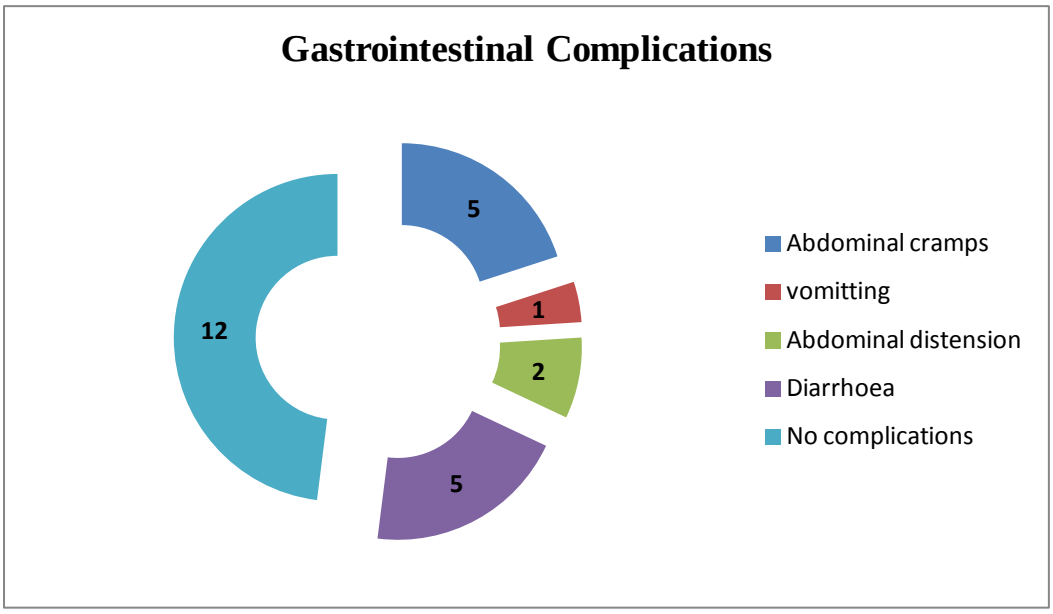
Mechanical complications of feeding tube	Cases
Yes	0
No	25

All the patients in the study group in whom nasojejunal tube was inserted for starting early enteral feeding did not develop mechanical complications in regard to the feeding tube like blockage, difficulty in removal and intolerance to the presence of the tube .

Table 21

Gastrointestinal complications to feeds	Cases
Abdominal Cramps	5(20%)
Vomitting	1(4%)
Abdominal distension	2(8%)
Diarrhoea	5(20%)
No complications	12(48%)
Total	25

Chart 15



None of the patients started on early feeding required withdrawal of feeds due to intolerance to feeds. Patients who developed gastrointestinal complications were managed by reducing the amount of feeds as per protocol. Among the patients started on early feeding 5(20%), 1(4%), 2(8%), 5(20%) patients developed diarrhoea, vomiting, abdominal distension and diarrhoea respectively. 12(48%) of the patients did not develop and gastrointestinal complications to the start of early feeding.

Discussion

Among the various methods of access to the gut for the initiation of early enteral feeding, the choice in this study was the use of a nasojejunal feeding tube. The tube used was manufactured by the company Fresenius Kabi. Due to the small diameter(12F) used none of the patients complained of difficulty of having the tube. Also the tube being made of polyurethane which is more pliable than conventional PVC or silicon tubes helped in the comfortness of having the tube in place. Complications of jejunostomy can be avoided by the use of a nasojejunal tube. On proper maintenance of the tube as per standard feeding protocols none of the tubes used in this study experienced mechanical complications like blockage and difficulty in removal. Carr et al[48]used a similar tube for feeding for 14 cases and recorded no blockage or cessation of feeds. Hence where possible a nasojejunal tube is the ideal route for early enteral feeding with the least complications.

The choice of feeds was milk based. This was due to the feasibility of preparation, maintenance, administration and cost effectiveness. The maximal calorie supplied was 2581kcal/day by the full strength feed. The protocol used was the same used by Singh et al[49] except for a minor change. Whey water was used in the study be Singh et al[49] to make the feed but in this study plain water was used. This was due to the difficulty in obtaining whey water, its storage in large amounts and taking into consideration the cost effectiveness. The only

other practical difficulty of using this feed was the feed getting spoiled making it unusable if prepared in large amounts. There were no other difficulties in regard to the feed used.

The mean age of the patients in the study group was 46.88yrs whereas in the control group was 47.96yrs. This difference is not significant and an age based bias is hence ruled out. Similarly the sex distribution is also not different statistically and hence bias based of sex of the patient can be ruled out.

The laboratory parameters compared in this study were weight of the patient, haemoglobin levels(g%) and the S.albumin levels. The pre operative values were compared to the post operative values in Day1 and Day7. The mean weight of the study cases pre operatively was 57.56 kg. There was no change in weight on post operative day1. But the weight increased to a mean of about 58.6kg by post operative day 7. While comparing the same parameter in the patients of the control group, the mean pre operative weight was 55.96kg, the same on post operative day 1 and reduced to 54.88kg by postoperative day7. This difference in post operative weight on day 7 is significant. This is attributed to the significant maintenance of nutrition right from the early post operative period. The mean pre operative haemoglobin among the cases in study group was 9.7g%. There is no significant change in post operative day , but the levels increased to 9.98g% by post operative day7. But in the control group the mean preoperative haemoglobin was 9.6g%, on post operative day 1 was

9.42g% and by post operative day7 was 9.49%. This also can be attributed to the significant maintenance of nutrition right from the early post operative period. The pre operative S.albumin levels among the patients started on early feeding was 2.74g/dl. On post operative day1 the same was 2.7g% and by post operative day7 it was 3.13g%. Among the control cases the mean preoperative S.albumin levels was 2.85g/dl. On post operative day1 it was 2.86g/dl and by post operative day7 it was 2.7g/dl. The significant rise in levels of S.albumin among the patients in the study group in comparison to those in the control group by post operative day7 can be seen. This signifies the advantage to starting early enteral feeding in order to maintain the nutritional status of the post operative patient. According to previous studies S. Albumin levels are probably not the best to follow up the nutritional status in acute time periods[50]. This is due to the long half life and low turnover rate of S.albumin[51]. However in this study significant changes in the S.albumin levels were also obtained.Probably a more specific indicator for assessing acute changes in the nutritional status of the postoperative patient would be S.transferrin and nitrogen balance[52]. Serum transferrin as a better indicator of nutritional status, especially of acute nutritional changes, when compared to the serum albumin level was proven by Shetty et al[53].

The mean duration of paralytic ileus among the cases in the study group was 2.4 days whereas in the control group was 4.04 days. This difference is significant and shows the advantage of early feeding.

With the paralytic ileus controlled early, the time taken to start oral feeds is also reduced among the cases in the study group with the mean duration taken to start oral feeds being 4.4 days when compared to those cases in the control group where the mean duration is 6 days. This difference is also significant and highlights the advantage of starting the post operative patient on early enteral feeding.

The rate of anastamotic leak when comparing both groups was not significant. Even though there were no cases in the study group who presented with anastamotic leak, only two cases in the control group presented with leak thereby the absence of significant statistical difference. Previous studies however prove otherwise. Probably the study being repeated in a larger setup with more number of cases involved would reveal a different result.

Surgical site infection is a common problem faced in post operative wards in the setup of government hospitals when compared to the more standardised private hospital setup. Hence the need for preventive measures to reduce the rate of surgical site infection is the need of the hour. Among the control group patients in the study about 9 patients developed surgical site infection when compared to to nil patients in study group. The maintenance of

nutrition among the post operative patients of the study group is probably the main factor for this nil rate in the study group.

Septic complications like pneumonia and urinary tract infections developed in 2 cases of the study group whereas 13 cases in the control group developed the same. This difference is also significant and again furnishes the advantage of early feeding.In the study by Singh et al[49] similar results were obtained where 22 vs 8 in the study group developed septic complications. This is probably due to the trophic effect of enteral nutrition on the mucosa of the gut which in turn prevents translocation of bacteria[54,55].

Side effects due to feeds were seen among 13 patients of the study group.Raga et al[56]reported that early enteral feeding related gastrointestinal adverse effects (cramps, bloating, diarrhoea, vomiting, aspiration) were observed in 194/650 patients (29.8%). Fifty-eight (8.9%) subjects had to be switched to parenteral feeding because of refractory intolerance to early enteral feeding. All patients in this study were managed as per protocol by reducing the amount of feeds transiently. None required cessation of feeding. Minimal intolerance to milk based feeds may be prime cause for intolerance to feeds. This can be overcome by reducing the amount of feeds temporarily or by diluting the feed.

Due to the above said statistically significant advantages of early feeding the mean duration of hospital stay among the patients of the study group was

7.6 days whereas among those in the control group was 10.2 days. This difference is also significant and adds to the list of advantages of early feeding.

Limitations of this study are

-Individual planning based on the pre operative nutritional status of the patient is not followed. If done so feeds based on the need of the patient can be given.

-Diagnosis of the patients are varied and so to get a clear picture on the effect of early feeding on a specific condition, individual studies based on specific diagnosis have to be done.

-Procedure done for the same diagnosis also varies in some patients and hence more studies involving more patients undergoing the same procedure have to be done for proper procedure done based protocols.

-Confounding factors like transfusions given, immunonutrition, antibiotics are not taken into account.

-Nutritional assessment based on better factors like S.transferrin not done in this study.

-Larger number of patients needed for comparing major factors like anastamotic dehiscence.

-Age and sex based comparison is needed for more accurate conclusions.

Conclusion

-Nutritional status of the patient clinically and biochemically is better in early feeding.

-Duration of paralytic ileus is lesser in early feeding.

-Time taken to start oral feeds is lesser in early feeding.

-Rate of surgical site infections is significantly less in early feeding.

-Septic complications are lesser in early feeding.

-Anastamotic leak rate could not be compared in this study.

-Duration of hospital stay is lesser in early feeding.

This study clearly shows the advantages of starting early enteral feeding in patients undergoing upper gastrointestinal surgeries over late enteral feeding.

Bibliography

1. Lewis SJ, Egger M, Sylvester PA, Thomas S. Early Enteral feeding versus "nil by Mouth" after gastrointestinal surgery: systematic review and metaanalysis of Controlled trails. BMJ 2001;323:773-6.
2. Schroeder D, Gillanders L, Mahr K, Hill GL.Effects of immediate post operative Entteral nutrition on body composition, musle function and wound healing. J Parenter Enteral Nutr 1991;15:376-83.
3. Hay dock DA, Hill GA. Impaired wound healing in patients with varying degrees of Malnutrition.I Parenter Enteral Nutr 1986;10:550-4.
4. Hill GL, Pickford I, Young GA, Schorah CJ, Blackett RC, Burkinshaw L.

   Malnutrition In surgical patients; an unrecognized problem. Lancet 1977;1:689- 92.
5. Hulsewe KW, Von Meyenfeldt MF, Soeters PB. Nutritional support for surgical Patient. In:J Payne-James, G Grimple,D silk, eds. Artificial nutritional support in In clinical practice.London:Greenwich Medical ;605-16.
6. Keele AM, Bray MJ, Emery PW, Duncan HD, Silk DB. Two phase randomized Controlled clinical trail of post operative oral dietary patients in surgical patients. Gut 1997;40:393-9.

7. Beier-Holgersen R, Boesby S. Influence of post operative Enteral nutrition on post Surgical infections. Gut 1996;39:833-5.
8. Heslin MJ, Latkany L, Leung D,Brooks AD,Hochwalk SN, Pisters PWT, et al. a Randomized controlled trail of early Enteral feeding after resection of upper GI Malignancy.Ann Surg 1997;226:567-80.
9. Watters JM, Kirkpatrick SM, Norris SB, Shamji, FM, Wells GA. Immediate post Operative Enteral feeding results in impaired respiratory mechanics and decreased Mobility. Ann Surg 1997;226:367-80.
10. Reynolds JV, Kanwar S, Welsh FKS, Windsor ACJ, Murchan P, Barclay GR, et al. Does the route of feeding modify gut barrier function and clinical outcome in patients After upper gastrointestinal surgery. J Parent Ent Ntur 1997;21;196-201.
11. O' Boyle CJ, Macfie J, Mitchell CJ, Johnson D, Sagar PM,Sedman PC. Microbiology of bacterial translocation in humans. Gut 1998;42:29-35.
12. Aiko S, Yoshizumi Y, Sugiura Y, Matsuyama T, Naito Y, Matsuzaki j et Et al,Beneficial effects of immediate enteral nutrition after esophageal cancer surgery. Surg Today 2001;31(11):971-8.
13. Gianoiti L, Braga M, Gentilini 0, Balzano G, Zerbi A, Dicarlo V. Artificial nutrition after pancreaticoduodenectomy 2000 Nov;21(1):59-65.

14. Han-Geurts J.M, Hop W.C.J, Kok N.F.M, Lim A, Brouwer K.J &Jeekel J. Randomized clinical trails of the impact of early enteral feeding on post operative leus and recovery BJS 2007:94:555-61.
15. Lewis SJ,Egger M, Sylvester PA, Thomas S. Early Enteral versus "nil by Mouth" after gastrointestinal surgery: Systematic review and metaanalysis Of controlled trails BMJ 2001 Oct 6;323(7316):773-6.
16. Petrelli NJ, Cheng C, Driscoll D, Rodriguez-Bigas M A. Early post operative oral feeding after colectony:an analysis of factors that may predict failure. Ann Surg Oncol 2001Dec;8(10):786-800.
17. Repin VN, Tkachenko IM, Gudkov OS, Repin MV Enteral tube feeding early after surgery on stomach and duodenum. Khirurgiia (Mosk) 2001(2):21-5
18. Silk D.B. A, Menzies Gow N. Post operative starvation after gastrointestinal surgery early feeding is beneficial.BMJ 2001 Ooctober 6;323(7316):761-62.
19. Braga M, Gianotti L, Gentilini 0,Liotta S, Di Carlo V. Feeding the gut early after digestive surgery:results of nine year experience. Clin Nutr 2002 Feb;21(1):59-65.
20. Boyes RJ, Kruse JA: Nasogastric and nasoenteric intubation. Crit Care Clin 8:865,1992.

21. Levin AL: A new gastroduodenal catheter. JAMA 76:1007, 1921.

22. Materese LE: Enteral alimentation: Equipment III. Nutr Support serv 2 ;48, 1982.

23. Wolfson PJ, Bauer JJ, Gelernt IM, et al :Use of long tube in management of patients with small Intestinal obstruction due to adhesion. Arch Surg 120;1001, 1985.

24. Dobbie RP, Hoffmeister JA:Continous pump tube enteric hyperalimentation. Surg Gynecol Obstet 143:273, 1976.

25. Rees RJP, Payne-James JJ. King C, Silk DB: Spontaneous transpyloric passage and performance Of "fine bore" polyurethane feeding tubes; A Controlled clinical trail. JPEN J Parenter Enteral Nutr 12:469. 1988.

26. Broughton WA, Green AE Jr: The technique of placing a nasogastric enteric tube. A revised protocol To avoid serious complications. J Crit111ness 5:1101. 1990.

27. Coufield KA, Page OP:Technique for intraduodenal placement of trananasal Enteral feeding catheters. Nutr Clin Pract 6:23,1991.

28. Salasidis R, Fleiszer T, Johnston R: Air insuffiation technique of Enteral tube insertion. A Randomized controlled clinical trail. Crit Care Med 26: 1036, 1998.

29. Marcuard SP, Stegall KL:Clearing obstructed feeding tubes. JPEN J

Parenter Enteral Nutr 13:81,1989.

30. Mellinger, J.D., and Ponsky, J.L.: Percutaneous endoscopic gastrostomy: State of the art, 1998. Endoscopy, 30: 126, 1998.
31. Gauderer MWL,Ponsky JL, Izant RJ Jr: Gastrostomy with out laparotomy: A percutaneous endoscopic Technique. J Pediatr Surg 15:872. 1980.
32. Moran BJ, Taylor MB, Johnson CD: Percutaneous Endoscopic gastrostomy. Br J Surg 77:858,1990.
33. Petersen, T.I., and Kruse, A: Complications of percutaneous endoscopic gastrostomy. Eur. J. Surg., 163:351, 1997.
34. Friedman JN, Ahmed S, Connoly B, et al: Complications associated with image guided gastrostomy and gastrojejunostomy tubes in children. Pediatrics 144:458- 461,2004.
35. Cosenti EP, Sautner T, Grant M, et al: Outcomes of surgical percutaneous endoscopic and percutaneous radiologic gastrostomies Arch Surg 133:1076,1998.
36. Wheatley MJ, Wesley JR, Tkach DM, Coran AG: long term follow up of brain damaged children requiring feeding gastrostomy: Should an antireflux procedure always be performed? J Pediatr Surg 26:301-304, discussion 304-305.1991.

37. Burd RS, Price MR, Whalen TV: The role of protective antireflux procedures in neurologically impaired children:A decision analysis. J Pediatr Surg 37:500-506, 2002.

38. Rothenberg SS, Bealer JF, Chang JH: Primary Laparoscopic placement of gastrostomy buttons for feeding tubes. A safer and simpler technique. Surg Enclose 13:995-997. 1999.

39. Chait PG, Weinberg J, Connolly BL. Et al: Retrograde percutaneous gastrostomy and jejunostomy in 505 children :A 4 year experience. Radiology 201:691-696. 1996.

40. Michael Brunt, Nathaniel J Soper. Laparoscopic surgery. In Maingot's Abdominal Operations, 10 th edition, MJ Zinner (ed); 269

41. Simon T, Fink AS. Recent experience with percutaneous endoscopic gastrostomy/ jejunostomy (PEG/ J) for enteral nutrition. Surg Enclose 2000; 14 : 436

42. Heimburger DC, Weinsier RL. Handbook of Clinical Nutrition. 3rd ed. St. Louis: Mosby, 1997.

43. Bury KD, Jambunathan G. Effects of elemental diets on gastric emptying and gastric secretion in man. Am J Surg 1974;127:59-66.

44. Jones BJM, Lees R, Andrews J, et al. Comparison of an elemental and polymeric enteral diet in patients with normal gastrointestinal function. Gut 1983;24:78-84.

45. Duncan HD, Silk DB. Problems of treatment-enteral nutrition. In: Nightingale J, ed. Intestinal failure. London: Greenwich Medical Media Ltd, 2001:477-96.

46. Havala T, Shronts E. Managing the complications associated with refeeding. Nutr Clin Pract 1990;5:23-9.

47. Flatt JP, Blackburn GL. The metabolic fuel regulatory system: implications for protein-sparing therapies during caloric deprivation and disease. Am J Clin Nutr 1974;27:175-87.

48. Carr CS, Ling KD, Boulos P, Singer M. Randomised trial of safety and efficacy of immediate postoperative enteral feeding in patients undergoing gastrointestinal resection. BMJ. 1996;312:869–71.

49. Singh G, Ram RP, Khanna SK. Early postoperative enteral feeding in patients with nontraumatic intestinal perforation and peritonitis. J Am Coll Surg. 1998;187:142–6

50. Lipkin EW, Bell S. Assesment of nutritional status-the clinician's perspective. Clin Lab Med. 1993;13:329–52

51. Jeejeebhoy KN. Nutritional asessment. Nutrition. 2000;16:585–90.

52. Hochwald SN, Harrison LE, Heslin MJ et al. Early postoperative enteral feeding improves whole body protein kinetics in upper gastrointestinal cancer patients. Am J Surg.1997;174:325–30

53. Shetty PS, Watrsiewicz KE, Jung RT, James WPT. Rapid turnover transport proteins: an index of subclinical protein energy malnutrition. Lancet. 1979;2:230–2.

54. Gianotti L, Alexander JW, Nelson JL, Fukushima R, Pyles T, Chalk CL. Role of early enteral feeding and acute starvation on postburn bacterial translocation and host defense: prospective, randomized trials. Crit Care Med. 1994;22:265–72.

55. Moore FA, Moore EE, Jones TN, McCroskey BL, Peterson VM. TEN versus TPN following major abdominal trauma-reduced septic morbidity. J Trauma. 1989;29:916–22.

56. Braga M, Gianotti L, Gentilini O, Liotta S, Di Carlo V. Feeding the gut early after digestive surgery: results of a nine-year experience. Clin Nutr. 2002;21:59–65.

PROFORMA

1. NAME:
2. AGE:
3. SEX:
4. IP NO:
5. GROUP:STUDY/CONTROL
6. DIAGNOSIS:

7. SURGERY DONE:

8. LABORATORY VALUES:

	PRE OPERATIVE	POST OPERATIVE DAY1	POST OPERATIVE DAY7
WEIGHT(kg)			
HAEMOGLOBIN(g%)			
S.ALBUMIN(g/dl)			

9. DURATION OF PARALYTIC ILEUS(DAYS):

10.TIME TAKEN TO START ORAL FEEDS(DAYS):

11.ANASTAMOTIC LEAK: YES/NO

12.SURIGAL SITE INFECTION: YES/NO

13.SEPTIC COMPLICATIONS: YES/NO

14.DURATION OF HOSPITAL STAY(DAYS):

15.MECHANICAL COMPLICATIONS OF FEEDING TUBE: YES/NO

16.GASTROINTESTINAL COMPLICAIONS TO FEEDS:

ABDOMINAL CRAMPS/VOMITTING/ABDOMINAL

DISTENSION/DIARRHOEA/NO COMPLICATIONS

							Pre operative levels			Post operative levels- Day1			Post operative values- Day 7										
Serial no	Name	Age	Sex	Ip no	Diagnosis	Procedure done	Weight(kg)	Haemoglobin(g%)	Serum albumin(g/dl)	Weight(kg)	Haemoglobin(g%)	Serum albumin(g/dl)	Weight(kg)	Haemoglobin(g%)	Serum albumin(g/dl)	Duration of paralytic ileus(days)	Time taken to start oral feeds(days)	Anastamotic leak	Surgical site infection	Septic complications(pneumonis, urinary tract infection)	Duration of hospital stay	Mechanical complications of naso jejunal feeding tube	Gastrointestinal complications to feeds
1	Alagar	60	M	47221	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	56	9.6	2.6	56	9	2.6	57	9.6	3	2	4	No	No	No	7	No	Abdominal cramps
2	Ponnusamy	55	M	38734	Corrosive acid ingestion-gastric outlet obstruction	Antrectomy and Billroth II anastamosis	64	8.3	2.8	64	8	2.8	65	8.4	3	2	4	No	No	No	8	No	No complications
3	Shanmugavel	60	M	35730	Carcinoma Stomach	Subtotal Gastrectomy and Billroth II	57	10	3	57	10	2.8	59	10.2	3.2	2	4	No	No	No	8	No	Vomitting
4	Babu	25	M	96257	Duodenal ulcer perforation	Omental patch closure	59	11	2.8	59	11	2.8	59	11.4	3.4	3	5	NA	No	No	8	No	Diarrhoea
5	Rajendran	50	M	37288	Carcinoma Stomach	Anterior Gastojejunal anastamosis	60	8.2	2.2	60	8	2.2	62	9	2.4	2	4	No	No	No	7	No	No complications
6	Paramasivam	60	M	51524	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	65	9	2.6	65	9	2.6	66	9.2	2.8	2	4	No	No	No	7	No	Avdominal Distension
7	Muthupandi	42	M	90035	Duodenal ulcer perforation	Omental patch closure	48	10	3	48	10	3	48	10.4	3.54	4	6	NA	No	Yes	10	No	No complications
8	Marutham	60	M	92452	Duodenal ulcer perforation	Omental patch closure	65	9.6	3	65	9	2.8	67	10	3.2	3	5	NA	No	No	8	No	No complications
9	Krishnan	59	M	46552	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	54	9.8	3.2	54	9	3	55	10	3.2	2	4	No	No	No	7	No	Dairrhoea
10	Mohammed Ali Jinnah	27	M	39256	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	53	10.2	2.8	53	9	2.8	55	9.6	3.4	2	4	No	No	No	8	No	No complications
11	Arokiyam	60	M	40466	Carcinoma Stomach	Subtotal Gastrectomy and Billroth II	60	11	2.6	60	10	2.6	60	11.2	3.2	2	5	No	No	No	8	No	Abdominal cramps

12	Parani	20	M	92418	Gastric ulcer perforation	Omental patch closure	65	8.6	3	65	9	3	66	9.2	3.4	3	4	NA	No	No	7	No	No complications
13	Sudalimadan	52	M	38927	Carcinoma Stomach	Anterior Gastojejunal anastamosis	45	9.2	2.8	45	9	2.8	47	10.2	3.2	2	4	No	No	No	8	No	No complications
14	Nagoor Chettiyan	56	M	92548	Duodenal ulcer perforation	Omental patch closure	50	11	2.6	50	11	2.6	52	11	3.4	3	4	NA	No	No	7	No	Diarrhoea
15	Dhochinnamoorthy	58	M	186	Duodenal ulcer perforation	Omental patch closure	57	10.4	2.4	57	10	2.2	57	10.2	3	3	5	NA	No	No	7	No	No complications
16	Muthuraman	55	M	40130	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	64	10	2.4	64	10	2.4	65	10.2	2.8	2	4	No	No	No	7	No	No complications
17	Karuppiah	55	M	95662	Duodenal ulcer perforation	Omental patch closure	50	9.2	3	50	9	3	52	9.2	3.4	2	4	NA	No	Yes	7	No	Abdominal cramps
18	Karthikeyan	35	M	24355	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	53	9.6	2.6	53	10	2.6	55	10	2.8	2	4	No	No	No	8	No	Diarrhoea
19	Ochu	45	M	3621	Duodenal ulcer perforation	Omental patch closure	63	9.4	2.8	63	9	2.8	64	9.2	3.2	3	5	NA	No	No	8	No	No complications
20	Pappa	45	F	45925	Carcinoma Stomach	Anterior Gastojejunal anastamosis	46	9	2.2	46	9	2.2	48	10	3	2	4	No	No	No	6	No	Abdominal distension
21	Periyachi	31	F	48515	Carcinoma Stomach	Subtotal Gastrectomy and Roux-en-Y	58	10.2	3	58	10	3	57	10.4	3.4	2	5	No	No	No	9	No	No complications
22	Chinniah	55	M	5672	Duodenal ulcer perforation	Omental patch closure	56	11	3	56	11	3	56	11.2	3.2	3	5	NA	Yes	No	8	No	Abdominal cramps
23	Chellapandi	22	M	7298	Duodenal ulcer perforation	Omental patch closure	74	9.2	2.4	74	9	2.4	74	9.6	3	2	4	NA	No	No	7	No	Diarrhoea
24	Aadhi	45	F	40744	Carcinoma Stomach	Anterior Gastojejunal anastamosis	53	9	2.6	53	9	2.6	55	10.2	3.2	2	4	No	No	No	7	No	No complications
25	Chinnakannu	40	M	13627	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	64	10	3	64	10	3	64	10	3	3	5	No	No	No	8	No	Abdominal cramps

26	Periyasamy	38	M	8812	Duodenal ulcer perforation	Omental patch closure	60	10.2	3.2	60	10	3.2	61	10	3	4	6	NA	Yes	No	10	NA	NA
27	Ganapathy	56	M	63896	Carcinoma Stomach	Subtotal Gastrectomy and Roux-en-Y	59	9.6	3.2	59	9	3.2	59	9.4	3.2	4	6	No	No	No	12	NA	NA
28	Veerabathran	47	M	56427	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	53	9.2	3	53	9	3	52	9	2.8	3	5	No	Yes	Yes	9	NA	NA
29	Muthukaruppi	60	F	10718	Duodenal ulcer perforation	Omental patch closure	60	8.4	2.8	60	9	2.8	60	8.8	3	4	5	NA	Yes	No	9	NA	NA
30	Periyakaruppan	60	M	49246	Carcinoma Stomach	Anterior Gastojejunal anastamosis	52	9.4	2.6	52	9	2.4	52	9.4	2.2	3	6	No	No	No	10	NA	NA
31	Boopathiammal	56	F	8530	Carcinoma Stomach	Subtotal Gastrectomy and Roux-en-Y	59	10.2	3	59	10	3	59	10	3	5	7	Yes	Yes	No	11	NA	NA
32	Pushpam	42	F	10838	Carcinoma Stomach	Anterior Gastojejunal anastamosis	57	9.6	2.8	57	10	2.8	57	9.8	2.8	4	6	No	No	Yes	10	NA	NA
33	Kumar	60	M	17232	Duodenal ulcer perforation	Omental patch closure	45	9.4	3.2	45	9	3.2	44	9.6	3	4	6	NA	No	No	9	NA	NA
34	Murugan	47	M	70737	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	53	9.6	2.6	53	10	2.8	54	9.6	2.6	3	5	No	No	Yes	9	NA	NA
35	Muthukaruppan	45	M	27510	Duodenal ulcer perforation	Omental patch closure	59	10	3.2	59	10	3.2	59	9.8	3	4	5	NA	Yes	No	9	NA	NA
36	Senthil Kumar	36	M	52346	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	49	9.4	2.8	49	9	2.8	50	9.2	3	4	6	No	No	No	11	NA	NA
37	Sikkandar kani	24	M	32714	Duodenal ulcer perforation	Omental patch closure	64	8.6	3	64	8	3	63	8.4	2.8	4	6	NA	Yes	Yes	10	NA	NA
38	Dhavamani	50	F	15462	Carcinoma Stomach	Anterior Gastojejunal anastamosis	53	9.2	2.8	53	9	2.8	50	9.2	2.6	3	5	No	No	No	9	NA	NA
39	Vijayadhurai	38	M	32742	Duodenal ulcer perforation	Omental patch closure	65	9.4	3	65	9	3	64	9.2	3	5	7	NA	No	No	11	NA	NA

40	Kathiresan	40	M	2150	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	62	9.6	3.2	62	10	3.2	60	9.4	2.8	4	5	No	No	Yes	9	NA	NA
41	Karuppu	55	M	45977	Carcinoma Stomach	Subtotal Gastrectomy and Roux-en-Y	60	11	2.6	60	11	2.6	59	10.6	2.4	5	8	Yes	No	No	13	NA	NA
42	Kalanjiyarani	29	F	36320	Duodenal ulcer perforation	Omental patch closure	59	9.8	2.8	59	10	2.8	59	9.8	2.6	4	6	NA	Yes	Yes	10	NA	NA
43	Devaraj	60	M	62286	Gastric ulcer perforation	Omental patch closure	54	8.8	3	54	9	3	54	9	2.8	4	6	NA	No	Yes	9	NA	NA
44	Kaliyammal	58	F	52036	Carcinoma Stomach	Anterior Gastojejunal anastamosis	58	9.4	2.4	58	9	2.4	58	9	2.6	3	5	No	No	No	8	NA	NA
45	Muniyammal	55	F	101123	Carcinoma Stomach with perforation	Emergency Gastrectomy and Roux-en-Y	48	9.8	2.2	48	9	2.2	47	9.2	2.2	7	10	No	Yes	Yes	18	NA	NA
46	Palani	45	M	64350	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	60	10	3	60	10	3	60	9.6	2.8	3	5	No	No	No	8	NA	NA
47	Papathi	50	F	68256	Chronic duodenal ulcer with gastric outlet obstruction	Truncal vagotomy and Posterior Gastro jejunostomy	49	10.2	2.6	49	10	2.6	49	10.2	2.6	4	6	No	No	No	10	NA	NA
48	Gandhiammal	60	F	18557	Carcinoma Stomach	Anterior Gastojejunal anastamosis	47	9.6	2.6	47	10	2.6	48	9.6	2.4	4	5	No	No	Yes	9	NA	NA
49	Suresh	32	M	67742	Duodenal ulcer perforation	Omental patch closure	58	8.6	2.8	58	9	2.8	59	8.8	2.6	4	6	NA	Yes	No	10	NA	NA
50	Nagu	56	M	30616	Carcinoma Stomach	Subtotal Gastrectomy and Billroth II	56	11	3	56	11	3	55	10.8	3	5	7	No	No	Yes	12	NA	NA