EARLY ENTERAL NUTRITION AFTER SURGICAL TREATMENT OF GUT PERFORATIONS: A PROSPECTIVE STUDY

By

DR. ASHISH VERMA

Dissertation submitted to the

BLDE UNIVERSITY BIJAPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

Under the guidance of

DR. VIJAYA PATIL M.S.

ASSOCIATE PROFESSOR

DEPARTMENT OF SURGERY

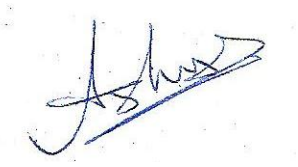
B.L.D.E UNIVERSITY'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR, KARNATAKA.

2013

B.L.D.E UNIVERSITY'S
SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR.

DECLARATION BY THE CANDIDATE

I, **DR. ASHISH VERMA** hereby declare that this dissertation entitled **"EARLY ENTERAL NUTRITION AFTER SURGICAL TREATMENT OF GUT PERFORATIONS: A PROSPECTIVE STUDY"**, is a bonafide and genuine research work carried out by me under the guidance of **DR. VIJAYA PATIL$_{M.S.}$**, Associate professor, Department of Surgery, B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.



Date: **DR. ASHISH VERMA**

Place: Bijapur. Post Graduate Student,

Department of General Surgery,

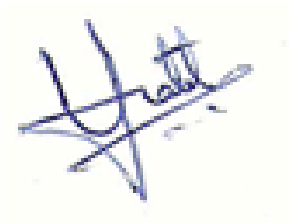
B.L.D.E.U.'s Shri B. M. Patil Medical College,

Hospital &Research Centre, Bijapur.

B.L.D.E UNIVERSITY'S
SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR.

CERTIFICATE BY THE GUIDE

This to certify that the dissertation entitled **"EARLY ENTERAL NUTRITION AFTER SURGICAL TREATMENT OF GUT PERFORATIONS: A PROSPECTIVE STUDY"** is a bonafide research work done by **DR. ASHISH VERMA**, under my overall supervision and guidance, in partial fulfilment of the requirement for the degree of M.S. in Surgery.



Date: **DR. VIJAYA PATIL**M.S.

Place: Bijapur. Associate Professor,

Department of General Surgery,

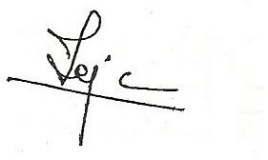
B.L.D.E.U.'s Shri B. M. Patil Medical College,

Hospital &Research Centre, Bijapur.

B.L.D.E UNIVERSITY'S
SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR.

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This to certify that the dissertation entitled **"EARLY ENTERAL NUTRITION AFTER SURGICAL TREATMENT OF GUT PERFORATIONS: A PROSPECTIVE STUDY"** is a bonafide research work done by **DR. ASHISH VERMA** under the guidance of **DR. VIJAYA PATIL$_{M.S.}$**, Associate Professor, Department of surgery at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.



Date: **DR.TEJASWINI UDACHAN$_{M.S.}$**

Place: Bijapur. Professor & Head of Department,

Department of General Surgery,

B.L.D.E.U.'s Shri B. M. Patil Medical College,

Hospital &Research Centre, Bijapur.

B.L.D.E UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR.

ENDORSEMENT BY THE PRINCIPAL

This to certify that the dissertation entitled **"EARLY ENTERAL NUTRITION AFTER SURGICAL TREATMENT OF GUT PERFORATIONS: A PROSPECTIVE STUDY"** is a bonafide research work done by **DR. ASHISH VERMA** under the guidance of **DR. VIJAYA PATIL$_{M.S.}$,** Associate Professor, department of surgery at B.L.D.E.U's Shri B. M. Patil Medical College Hospital and Research Centre, Bijapur.

Date**:** **DR. M. S. BIRADAR$_{M.D.}$**

Place: Bijapur. Principal,

B.L.D.E.U.'s Shri B. M. Patil Medical College,

Hospital &Research Centre, Bijapur.

B.L.D.E UNIVERSITY'S
SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, BIJAPUR.

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE UNIVERSITY BIJAPUR, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

Date:

Place: Bijapur

DR. ASHISH VERMA
Post Graduate Student,
Department of General Surgery,
B.L.D.E.U.'s Shri B. M. Patil Medical College,
Hospital &Research Centre, Bijapur.

ACKNOWLEDGEMENT

Firstly, I pray to the almighty god, thanking him for the bounty of life. I thank my parents who have nurtured me and supported me in all my endeavours, without their love and innumerable sacrifices; I would not be the person I am today.

I would like to express my deep gratitude and indebtedness to my teacher and guide **DR. VIJAYA PATIL$_{M.S.}$,** Associate Professor, Department of Surgery, BLDEU'S Shri B. M. Patil Medical College, Bijapur, who was ever encouraging in her approach while helping me through my postgraduate course. She has always been supportive and allowed me to work and develop at my own pace, guiding wherever necessary. Her meticulous approach and quick attention along with giving equal value to time was inspiring. Without her guidance and support, it would have been impossible to complete this dissertation.

I express my sincere gratitude to **DR. TEJASWINI UDACHAN $_{M.S.}$,** Professor and Head, Department of surgery, BLDEU'S Shri B. M. Patil Medical College, Bijapur, who always stood by my side to encourage me in my study and research and for her motivation, enthusiasm, and immense knowledge. Her guidance helped me in all the times of research and writing of this dissertation.

I am extremely thankful to **DR. SUNIL KRISHNAN,** Assistant professor, **DR. MARUTHI MEDAR**, Assistant professor, and **DR. RASHMI PATIL,** Senior Resident, Department of surgery, BLDEU'S Shri B. M. Patil Medical College, Bijapur, whose inspiration and guidance have helped me in preparing this dissertation.

I am grateful to **DR. M. S. BIRADAR**, Principal of B.L.D.E.U'S Shri B.M.PATIL Medical college hospital and Research Centre, Bijapur, for allowing me do this work, to access medical records, utilize clinical material and facilities in this institution

I am extremely thankful to **DR. MADAGI**, for his guidance in statistical analysis.

I thank my friends and my seniors **DR. KUNAL SHAHI,** PG in Department of Otorhinolaryngology and Head & Neck Surgery, and my colleagues **DR. DAYANAND BIRADAR** and **DR. HARIKRISHNAN,** PGs in Department of General Surgery who rendered immense help and support during my postgraduate course. I thank them from my heart.

I would also like to thank all the non-teaching staff of my department for their constant encouragement and moral support.

I also thank all library staff for their kind corporation in bringing out this dissertation.

Finally, I acknowledge with gratitude my indebtedness to all the patients who contributed in no small way to this dissertation but for whom this entire exercise would have been unimaginable.

Last, but not the least, I thank my beloved fiancée **DR. DEEPIKA PATIL,** who is also my colleague. She stood by my side, all through this work with her love and care, despite all the ups and lows she had to face during this study. This study is dedicated to her.

Date: **DR. ASHISH VERMA**

Place: Bijapur. Post Graduate Student,

Department of General Surgery,

B.L.D.E.U.'s Shri B. M. Patil Medical College,

Hospital &Research Centre, Bijapur.

CONTENTS

Introduction

INTRODUCTION

Nutritional support is an integral part of the management of a critically ill patient. Evidence suggests a strong association between poor nutritional status and poor outcome in surgical patients.[1] Patients with perforative peritonitis are often severely malnourished upon presentation to the hospital. Muscle wasting, pedal edema, and low serum albumin levels are common in such patients seen in our clinics. Traditional customs in India entail the use of only clear liquids in patients with fever and abdominal pain. Inadequate oral intake contributes to the malnutrition. The problem is compounded by the presence of sepsis and its related metabolic alterations, such as an increase in energy demands and changes in substrate utilization. All of these factors combine to increase the patient's risk for morbidity and mortality.

After emergency gastrointestinal surgery, the trend has been to keep the patient 'nil by mouth' and decompress the stomach by a naso-gastric tube. Provision of oral nutrients in the postoperative period historically has been withheld until bowel activity returns. Return of bowel sounds, flatus, and defecation are used to herald the return of bowel activity. This period may be prolonged in patients with peritonitis. There is a general consensus that gastric and colonic atony following laparotomy lasts 24-48 hours and that the small bowel, in fact, recovers functions within four to six hours.[2]

Over the last few years, great emphasis has been laid on early enteral feeding by a naso-jejunal tube or via jejunostomy distal to the site of

anastomosis.[3,4] Very few clinical trials have evaluated the efficacy and safety of early enteral nutrition in patients who have undergone laparotomy for generalized peritonitis following perforation of the gut. [2,5,6,7]

So this work is designed to assess the safety, feasibility and benefits of enteral feeding by a naso-gastric tube 24-48 hours after emergency gastrointestinal surgery.

Aims & Objectives

AIMS AND OBJECTIVES

To compare the results of **"early enteral feeding"** and **"nil by mouth"** after surgical treatment of gut perforations in terms of specific outcomes.

Review of Literature

REVIEW OF LITERATURE

HISTORY

A 'nil by mouth' (NBM) approach after major gastrointestinal (GI) surgery has been well known for many years. The successful administration of parenteral nutrition (PN) in the late 1960s and 1970s provided clinicians a way to feed patients with significant loss of intestinal mass or function who would otherwise starve. In the 1980s and early 1990s, laboratory and clinical data demonstrated that there were benefits gained when nutrition is delivered via the gastrointestinal tract rather than parenterally. Simultaneously, clinicians noted that most of the "ileus" that occurs in patients remains limited to the colon and the stomach, while the intestine remains capable of absorbing and processing those nutrients if delivered into the small intestine.[8]

As a result, the concept of "resting the bowel" or to bypass the "ileus" through the use of PN has been replaced with the concept of providing enteral nutrition (EN) whenever the gastrointestinal tract is functional. "Starving the gut" is no longer a standard of practice in the critically ill or injured patient, or even in disease states.

Early enteral nutrition (EN), as opposed to the conventional NBM and intravenous fluids (IVF) approach, has received increasing attention in recent years. Several advantages have been propounded, though the evidence has not always been clear. A look through the pages of history of EN unravels answers to many of these questions:

What is the earliest evidence supporting EN after major GI surgery?

The earliest study to address the issue of elemental diet (ED) in the early post-operative period was in 1979, when 30 patients who had undergone major GI operations were given either ED or conventional treatment postoperatively.[9] The ED group did significantly better than controls clinically and metabolically and lost less weight. Energy intake was higher in the ED group and negative nitrogen balance was more in the control group throughout the initial seven postoperative days. The authors concluded that ED could be given from the first postoperative day, with patients faring better metabolically and requiring shorter hospitalization.[9]

Is solid diet safe after major GI surgery?

While the 1980s saw more studies addressing this concept, the first randomized study was reported in 1992.[10] This study evaluated 171 patients who had undergone an intervention affecting the integrity of the GI tract (gastroenterostomy, cystogastrostomy, Billroth II anastomosis, suture of perforation, small bowel anastomosis, colocolostomy, enterobiliary anastomosis, Whipple resection, etc.). The study subjects were randomized into two groups. The first group began liquid oral intake 4 hr after nasogastric tube removal, while the second group began regular solid intake soon after tube removal. The criterion to remove the tube was based on the finding of normal bowel sounds (confirmed by a minimum of two senior surgeons). There was no significant difference between the two groups with regard to occurrence of GI

disturbances. Most disturbances (i.e., vomiting, abdominal distension, acute gastric dilatation, etc.) were observed after lower intestinal tract operations. Nine patients in the first group and seven in the second required reinsertion of the nasogastric tube. This study became the first RCT to suggest that early EN, comprising solid food, immediately after nasogastric tube removal could be *safely* advised after major GI operations.[10]

Does early EN influence incidence of postoperative infections?

This question was addressed in 1996 by an RCT where 30 patients received Nutri-drink (a brand of a nutritional orange flavoured supplement for early postoperative enteral nutrition) and 30 received placebo through a naso-duodenal feeding tube[9] starting from the day of surgery itself. Only two of 30 patients in the EN group developed infectious complications compared to 14 of 30 patients in the control (placebo) group and these differences were obviously significant. More pertinently, the two groups were similar with regard to preoperative nutritional status and the type of surgeries performed (esophagectomies, gastrectomies, and major colorectal resections with anastomosis) and a single investigator was involved in the study. Furthermore, the total postoperative complications were significantly more in the placebo group than in the EN group (19 *vs.* 8). It was concluded that early EN in patients after major abdominal surgery resulted in an important reduction in infectious complications.[11]

What is the influence of early EN on whole-body protein kinetics in upper GI diseases?

Patients with upper GI cancers are at increased risk for malnutrition and associated morbidity and even mortality. Hochwald *et al*. evaluated the protein kinetic effects of early EN, comparing them with standard postoperative care of NBM and IVF.[12] Twelve patients were randomized to receive early EN and 17 received conventional treatment. It was observed that early EN decreased fat oxidation and whole-body protein catabolism while improving net nitrogen balance. By significantly improving protein metabolism, it was suggested that early EN could decrease postoperative morbidity and mortality in upper GI cancer patients.

What is the impact of early EN (immunonutrition) on clinical outcomes and cost after major upper GI surgery?

To assess the impact of early enteral immunonutrition (compared to an isocaloric, isonitrogenous diet) on clinical outcomes and cost after major upper GI cancer surgery, a randomized, prospective, multicenter trial was conducted in the mid-90s and results were reported in 1997 and updated again in 1999.[13] The EN diet was supplemented by arginine, dietary nucleotides, and omega-3 fatty acids and administered to 77 patients, while another 77 received an isocaloric, isonitrogenous diet (placebo). Thus a total of 154 patients were evaluated after EN was started 12-24 hr after surgery and the amount gradually increased up to 80 ml/h by the 5th postoperative day. The complications were

divided into early (postoperative days 1-5) and late (after the fifth postoperative day) postoperative complications. Both groups tolerated early EN, and the rate of tube feeding−related complications was low. Postoperative complications occurred in 17 patients in the immunonutrition group compared to 24 patients in the control group and this difference was insignificant. Also, in the early period, complications occurred to a similar extent in both groups. However, in the late period (after postoperative day 5), significantly fewer patients in the immunonutrition group developed complications than in the placebo group (5 *vs.* 13). Furthermore, there was a significant reduction in the frequency of development of late complications and wound infections in the immunonutrition group. Since a retrospective cost-comparison analysis was also performed, the above findings ensured that patients in the immunonutrition group incurred substantially lesser costs compared to those in the placebo group. Evaluating outcomes and cost-effectiveness of perioperative EN (immunonutrition) in patients undergoing elective major upper GI surgery, the same group[12] reported significantly decreased early occurrence of postoperative infections and reduced treatment costs of the complications in those who were administered perioperative EN.

Around the same period, another prospective RCT (involving 260 patients undergoing pancreaticoduodenectomy or gastrectomy for cancer) evaluated the route of delivery and formulation of postoperative nutritional support on host defences, protein metabolism, infectious complications, and outcomes after these major resections.[14] While one group received standard

EN, another received an immunonutrition, and the third received TPN. All the three regimes were isocaloric and isonitrogenous. This trial reported that the immunonutrition group fared better than the other two groups (better recovery of immune parameters on postoperative day 8). Postoperative infection rate was 14.9% in the immunonutrition group, 22.9% in the standard group, and 27.9% in the parenteral group ($P = 0.06$). Mean (± SD) length of hospital stay was 16.1 ± 6.2, 19.2 ± 7.9, and 21.6 ± 8.9 days in the immunonutrition, standard, and parenteral groups, respectively ($P = 0.01$ *vs.* standard group; $P = 0.004$ *vs.* parenteral group). It thus became evident that early EN was a valid alternative to TPN in patients undergoing major surgery. Furthermore, the study concluded that immunonutrition enhanced the host response, induced a switch from acute-phase to constitutive proteins, and perhaps improved outcomes.[14]

The several advantages of immunonutrition reported by the above studies, were not corroborated by data from the Memorial Sloan Kettering Cancer Center.[4] In their RCT of 195 patients (undergoing surgery for esophageal, gastric, peripancreatic, and bile duct cancers) they evaluated early EN (immune-enhancing formula, i.e., IEF) after resection of upper GI malignancy *vs.* controls; they reported no significant differences in the number of minor, major, or infectious wound complications between the groups. There was one bowel necrosis associated with IEF requiring re-operation. The hospital mortality was 2.5% and median length of hospital stay was 11 days, which was not different between the groups. This study concluded that early

EN (immunonutrition) was not beneficial and could not be recommended as a routine after surgery for upper GI malignancies.[4]

Thus, we can conclude that standard enteral nutrition is comparable to immunonutrition but is superior to TPN in terms of support on host defences, protein metabolism, infectious complications, and other minor or major complications after gastrointestinal surgeries.

Is early EN (immunonutrition) useful in malnourished and transfused patients?

The above question about early immunonutrition[4] was answered by another trial in 1998.[16] This RCT, involving 166 patients undergoing curative resections for gastric or pancreatic cancer, evaluated the impact of the route of administration of artificial nutrition and the composition of the diet on outcomes. At operation, the patients were randomized into three groups to receive: a) a standard enteral formula (control group; n = 55); b) the same enteral formula enriched with arginine, RNA, and omega-3 fatty acids (enriched group; n = 55); and c) total parenteral nutrition (TPN group; n = 56). The three regimens were isocaloric and isonitrogenous. EN was started within 12 h following surgery. The infusion rate was progressively increased to reach the nutritional goal of 25 kcal/kg/day on postoperative day 4. Early enteral infusion was well tolerated. Side effects were recorded in 22.7% of the patients, but only 6.3% failed to reach the nutritional goal. The enriched group had a significantly lower severity of infection than the parenteral group (4.0 *vs.*

8.6). In subgroups of malnourished (*n* = 78) and homologous transfused patients (*n* = 42), the administration of the enriched formula significantly reduced both severity of infection and length of stay compared with the parenteral group. Moreover, in transfused patients, the rate of septic complications was 20.0% in the enriched group, 38.4% in the controls, and 42.8% in the TPN group. This trial was different from the previous trials since it not only suggested that early EN is a suitable alternative to TPN after major abdominal surgery, but also demonstrated that an enriched diet was of benefit in malnourished and transfused patients.[15]

Outcomes of systematic review and meta-analyses?

It was only in 2001 that a systematic review and meta-analysis was done to determine whether a period of starvation (nil by mouth) after GI surgery is beneficial in terms of specific outcomes.[7] Three electronic databases (PubMed, EMBASE, and the Cochrane controlled trials register) were used for this study. Eleven studies with 837 patients were evaluated. In six studies, patients in the intervention groups were fed directly into the small bowel and in five studies patients were fed orally. Early feeding reduced the risk of any type of infection and the mean length of hospitalization. Also, risk reductions were seen for anastomotic dehiscence, wound infection, pneumonia, intra-abdominal abscess, and mortality, though these failed to reach statistical significance. However, the risk of vomiting was increased among patients fed early. It was concluded that there seemed to be no clear advantage in keeping patients NBM after elective

GI resection and early feeding could be of benefit. It was also concluded that an adequately powered trial was required to confirm or refute the benefits seen in small trials.[7] In 2009, a systematic review and meta-analysis evaluating whether EN within 24 hr of intestinal surgery is beneficial as compared to late commencement of feeding in patients was published by the same authors.[16] Thirteen RCTs fulfilled their inclusion criteria and a total of 1,173 patients were included. Mortality was reduced with early postoperative feeding but increased vomiting was noted. There was a suggestion of reduced postsurgical complications and hospital stay. The study concluded supporting the notion that early feeding may be of benefit.

Is EN is superior to TPN?

The questions raised by the meta-analysis[7] appear to have been answered by a RCT that was published soon after.[17] This study aimed to test the hypothesis that postoperative EN is better (i.e., there were fewer postoperative complications) than parenteral nutrition containing similar energy and nitrogen amounts [112 kJ/kg/day and 1.4 gm aminoacid/kg/day]. Malnourished patients undergoing major GI surgery (317) were assigned to EN or TPN. Analysis was by intention to treat. Postoperative complications occurred in 54 (34%) patients fed enterally *vs.* 78 (49%) fed parenterally. Length of postoperative stay was 13.4 days and 15.0 days in the EN and TPN groups, respectively. Both the differences were significant. Adverse effects occurred in 56 (35%) patients fed enterally *vs.* 22 (14%) patients fed

parenterally. Fourteen (9%) patients on EN had to switch to TPN, whereas none with TPN crossed over to EN. This trial clearly concluded that early EN significantly reduced the complication rate and duration of postoperative stay compared with TPN, although TPN was perhaps better tolerated than EN.[17]

Effect of early EN on functional GI recovery?

Postoperative convalescence is mainly determined by the extent and duration of postoperative ileus. A recent study conducted in 2007 [18] evaluated the effects of early EN on functional GI recovery and quality of life. One hundred and twenty-eight patients undergoing elective open colorectal or abdominal vascular surgery were enrolled. Of these, 67 were randomized to a conventional return to diet and 61 to a regimen allowing resumption of an oral diet as soon as tolerated ('free diet' group). It was observed, that reinsertion of a nasogastric tube was necessary in 20% of the free diet group and in 10% of the conventional group; the difference was not statistically significant. The complication rate was similar for both groups, as was return of GI function. A normal diet was tolerated after a median of 2 days in the free diet group compared with 5 days in the conventional group and this difference was significant. Quality of life scores were similar in both groups. This trial thus proved that early resumption of oral intake did not diminish the duration of postoperative ileus or lead to a significantly increased rate of nasogastric tube reinsertion. Tolerance of oral diet was thus not influenced by gastrointestinal functional recovery. The inference was that postoperative management should include early resumption of diet.[18]

Role of EN in Major GI resections?

Surgical advances have increased the certainty of a successful esophago-enteric anastomosis, making early oral enteral feeding after surgery feasible. A recent study[18] compared the benefits of EN and TPN in patients undergoing total gastrectomy for gastric cancer. Nutrition and intestinal permeability were assessed. Complications were similar in both groups. Treatment cost was less and length of hospital stay was shorter in the EN group. We thus have evidence that EN is an efficient way to provide nutrition to patients and possibly prevent intestinal atrophy in patients who traditionally have had to endure prolonged postoperative fasting.[19]

Role in non-traumatic intestinal perforation and peritonitis?

A study on the role of early EN in the setting of perforative peritonitis was published in 1998, by Singh *et al*.[5] Immediate postoperative EN was shown to be effective in reducing septic morbidity in patients with abdominal trauma. This study was designed to investigate the feasibility and efficacy of immediate EN in patients with non-traumatic intestinal perforation and peritonitis. Forty-three patients (21 in the study group and 22 in the control group) were included. Patients in the study group achieved a positive nitrogen balance by the third postoperative day; patients in the control group remained in negative nitrogen balance throughout the study. The mortality rate was similar in both groups (18.2% *vs.* 19.1%). The control group had a total of 22 septic complications *vs.* eight in the study group, and this difference was

significant. The authors concluded that immediate postoperative feeding was feasible in patients with perforative peritonitis and that it reduced septic morbidity.

In 2004, Malhotra *et al.*[2] reported that early enteral nutrition through nasogastric tube in patients with perforation peritonitis is safe and is associated with beneficial effects such as lower weight loss, early achievement of positive nitrogen balance as compared to the conventional regimen of feeding in operated cases of gut perforation.

In 2005, Kaur *et al.*[6] reported that early enteral feeding through a nasoenteric tube in patients with perforation peritonitis is well tolerated by these patients and helps to improve energy and protein intake, reduces the amount of nasogastric aspirate, reduces the duration of postoperative ileus, and reduces the risk of serious complications.

CAUSES OF INTESTINAL PERFORATIONS

Perforation peritonitis is a frequently encountered surgical emergency in tropical countries like India, most commonly affecting young men in the prime of life as compared to the studies in the west where the mean age is between 45–60 years. In majority of cases the presentation to the hospital is late with well-established generalized peritonitis with purulent/faecal contamination and varying degree of septicaemia. The signs and symptoms are typical and it is possible to make a clinical diagnosis of peritonitis in all patients.[20]

Gastroduodenal perforations

Perforations of peptic ulcers form the major group among the gastroduodenal perforations. These perforations are usually encountered along the first part of the duodenum anteriorly and in the pylorus of the stomach. The advances in the medical treatment of the peptic ulcer disease have led to a dramatic decrease in the number of elective surgeries performed. However, the number of patients undergoing surgical intervention for complications such as perforation remains relatively unchanged or has increased. Such patients present with the classical signs and symptoms of peritonitis, and need early surgery for a favourable outcome. Although the surgical options are many – from simple closure to definitive acid reducing procedures – it has been observed that simple closure of the perforation using a pedicled omental patch gives good results, even in large perforations upto 3 cms diameter.[21] This

should therefore, be the preferred surgical method of closure, as it is easy to perform, is technically straightforward, and gives comparable results to that of definitive surgery. The mortality rate of these perforations varies from 4 – 11%, and is higher in the elderly, those with concomitant disease, preoperative shock, larger size of the perforation, delay in presentation and delay in operation. Perforation of ulcers at other sites within the stomach and gastric cancers has been uncommonly reported, and emergency gastrectomy is the treatment of choice.[21]

Small bowel perforations

The next common types of perforations encountered are those arising in the small intestine. These usually arise on a background of enteric fever, when the ulcerated Payer's patches in the terminal ileum perforate to give frank peritonitis. These typhoid ileal perforations have a high mortality rate, upto 60%. Aggressive resuscitation, antibiotics and early surgery has reduced the mortality rate and complications in this subset of small bowel perforations to less than 10%. Although early surgery is associated with a better outcome, there is, however, no uniformity of opinion about the operative procedure to be performed in these perforations, and various procedures have been described such as simple closure, wedge excision or segmental resection and anastomosis, ileostomy, and even, side to side ileo-transverse anastomosis after primary repair of the perforation. A primary anastomosis (simple closure) is to be considered only when the patient presents early and the bowel is healthy.[21]

A 'non-specific' etiology is attributed to small bowel perforations when the perforation cannot be classified on the basis of clinical symptoms, gross examination, serology, culture and histopathological examination into any disease state such as enteric fever, tuberculosis or malignancy. These ulcers are usually single and commonly involve terminal ileum. It has been proposed that submucus vascular embolism, chronic ischemia due to atheromatous vascular disease or arteritis, or drugs such as enteric coated potassium tablets are responsible for them.[21]

These 'non-specific' ileal perforations are closely followed by small bowel perforations occurring in intestinal tuberculosis. Most of these (50 – 80 %) occur in the ileum, usually proximal to strictures of the bowel. Free tubercular perforations are rare. The mortality rate reported in these tubercular perforations is very high, upto 70 %. The diagnosis of perforated tubercular enteritis is usually not one that is made pre-operatively, because of the non-specific signs and symptoms and absence of radiological evidence of tuberculosis in the chest. Even in the presence of tubercular lesions in the chest skiagram, the diagnosis is not entertained or established until the histology and culture of the biopsied tissue turns out to be positive. The recommended treatment after source control is multidrug anti tubercular therapy.[21]

In contrast to these common causes of small bowel perforation in the developing countries, small bowel perforations are rare in the oriental countries. Apart from enteric fever and 'non-specific' ulcers, the other reported causes of such perforations from these countries include Crohn's disease,

Behcet's disease, radiation enteritis, adhesions, ischemic enteritis, SLE and very rarely, intestinal tuberculosis. Free perforations are a rare complication of Crohn's disease, and their incidence is reportedly highest from Japan, where it ranges from approximately 3% to 10%. These perforations are usually solitary, and occur mainly in the ileum. However, they can be multiple, and can occur at any site in the small or large bowel. Similarly the incidence of Behcet's disease is much higher in Japan, and perforation of the intestinal ulcers can occur in upto 56% of cases. These are usually multiple and occur commonly in the terminal ileum and caecum, and need removal of a long segment of the ileum to prevent post-operative recurrence.[21]

Appendicular and colorectal perforations

High incidence of appendicular perforations is mainly seen in younger age of patients where appendicitis and consequently the complications are known to be much higher. Colorectal perforations are uncommon. Perforations secondary to colonic neoplasms account for the majority of such cases. The perforation may occur at the site of the malignancy or proximally, as a 'blow out' of the proximal large bowel due to obstruction from the lesion. The incidence of such perforations is low, but carries a high mortality of about 17%.[21]

The other causes that have been reported are perforation of colonic diverticula, inflammatory conditions of the colon, volvulus, mesenteric ischemia, trauma, iatrogenic complications and idiopathic perforations. In the

Asian communities diverticular disease is more common in a younger age group, and the right colon is more commonly involved. One-third of these patients present with perforation of the large bowel and faecal peritonitis that requires surgical intervention. Amoebic colitis is another condition that is common in the tropical countries, with an incidence of perforation around 2%, but with a high mortality rate (up to 50%) regardless of the treatment.[21]

Traumatic Perforations

Perforation of the gastrointestinal tract is relatively infrequent sequel of blunt abdominal trauma. Incidence of hollow visceral injury varies from <1% - 8.5%.[21] It is more commonly seen in penetrating trauma. Almost any of the part of bowel can be involved depending upon the site of trauma. Diagnostic delay is associated with increased morbidity and hospital stay and perhaps increased mortality especially when there is associated severe head injury. [22]

METABOLIC CHANGES DUE TO PERFORATION

Spillage of intestinal contents into the peritoneal cavity as a result of primary intraabdominal disease (e.g., perforated peptic ulcer, appendicitis, diverticulitis, perforated carcinoma), penetrating trauma, or iatrogenic perforation after instrumentation or radiological procedures causes of acute suppurative peritonitis. Usually, suppurative peritonitis has an abrupt onset and a relatively short course with a rapid progression. Mortality results from fluid shifts and systemic endotoxin, which may cause hypovolemia and septic shock. Early diagnosis with prompt surgical intervention and aggressive preoperative and postoperative management is essential to reduce the morbidity and mortality from multiple organ system failure resulting from untreated peritonitis.[23]

The peritoneum mounts rapid response to infection, injury, and leakage into the peritoneal cavity of digestive fluid, bile, pancreatic juice, urine, or blood. The result is vascular permeability, fluid exudation, and both neutrophil and cytokine response. Pain fibers within both the visceral and parietal peritoneum are activated. These fibers are believed to be C-fibers containing substance P and calcitonin gene-related peptide (CGRP). Reflex pathways cause muscular contraction in the abdominal wall to limit movement (guarding and rigidity). Similarly, peristaltic movement of the intestine is arrested (hypoactive or absent bowel sounds).[24]

Patients with severe sepsis demonstrate a characteristic picture in which hypermetabolism occurs, protein and fat are consumed, and body water and salt are conserved.[25]

Vascular permeability, as a result of tissue damage or infection, causes fibrin-rich plasma to flow into the peritoneal cavity. This leads to the formation of fibrin, which later organizes into collagen and causes adhesion formation. Untreated, generalized peritonitis most commonly cause death secondary to gram-negative septicaemia, septic shock, and disseminated intravascular coagulation. On other occasions, generalized peritonitis leads to intraabdominal abscesses, which tend to be multiple.[24]

The clinical syndrome of sepsis results from a systemic host response to infection that, in turn, triggers the cytokine cascade. A number of cytokines have been identified and are generally classified as either proinflammatory (e.g., tumour necrosis factor-α [TNF-α] and interleukin-1 [IL-1]) or anti-inflammatory (e.g., IL-10 and IL-1 receptor antagonist). It is generally accepted that a careful balance exists between proinflammatory and anti-inflammatory cytokines; a disruption in this balance leads to an exaggerated immune response resulting in multiple organ system failure and possibly death.[23]

Tissue metabolism is severely altered during the response to peritonitis. The metabolic rate is increased owing to the increased secretion of catecholamines and cortisol. However, hypovolemia reduces cardiac output. Tissue hypoxia develops as a result of reduced oxygen delivery, owing to both decreased perfusion as well as shunting. Increasing anaerobic glycolysis

produces accumulating amounts of lactic acid and acid by-products. Renal and pulmonary clearance of this increased acid load leads to metabolic acidosis, unless perfusion is restored. A significant conversion in substrate metabolism also occurs in peritonitis. Following the early depletion of hepatic glycogen stores, protein catabolism is augmented in the skeletal muscles to release branched chain amino acids for the use by myocytes as an energy source. Other amino acids are released into the circulation to be utilized in hepatic gluconeogenesis as well as the production of acute-phase proteins to support the systemic inflammatory response. Though the body lipolysis rate is also increased, utilization of free fatty acids as an energy source is not efficient in the early septic period. The severe loss of lean body mass that can occur from the net protein catabolism occurs rapidly and is only partially ameliorated by the use of nutritional support.[26]

There is a reprioritization of hepatic protein synthesis in severe sepsis that is obligatory and independent of changes in total body protein. Concentrations of the constitutive plasma proteins fall, and levels of the acute-phase protein, C-reactive protein, rise early in the course of illness. After a few days, as the acute-phase reaction subsides, levels of the constitutive proteins returns to the normal range. These obligatory changes in hepatic protein levels occurs in the face of continuing massive proteolysis and high energy expenditure. There are no correlations between the changes in total body protein and those of the constitutive plasma proteins.[25]

The patients retains > 12 L of resuscitative fluids by the time they are haemodynamically stable. After this time, body weight begins to fall because of the loss of body water, mainly extracellular water. Intracellular water falls steadily also, but in proportion to the loss of total body potassium. Cellular compositions are abnormal when measured at the time hemodynamic stability had been reached. Critical illness has been shown to be associated with an alteration in muscle cell composition, as measured by a decrease in skeletal muscle transmembrane potential difference, increased cellular sodium and water levels, and depletion of intracellular potassium and magnesium. Intracellular potassium level does not falls further in the face of continuing hypermetabolism and proteolysis.[25]

The total body protein changes in the patients show that the massive losses that occur in association with severe sepsis and show that early on, most (approximately 70%) of this protein comes from the hydrolysis of skeletal muscle protein. Even approximately 12 days after the onset of sepsis, even while receiving nutritional support, the patients continue to loose protein, but mainly from tissues other than skeletal muscle, presumably the viscera. The average loss of skeletal muscle is approximately 3 kgs, and the loss of visceral mass is also similar. These losses occurred despite nutritional support.[25]

This period of hypermetabolism probably lasts 3 weeks or longer in most such patients. Most of the administered resuscitative fluids are retained within the extracellular space. Once hemodynamic stability is reached, body hydration returns to normal, but slowly. It seems unlikely that much can be

done to preserve cellular composition before hemodynamic stability is achieved, but intensive care management appears to prevent further deterioration.[25]

Fat oxidation is a function of energy intake; if it is important clinically to preserve fat stores, this can be done by ensuring that total energy requirements are met. Protein losses, which occurred early on from skeletal muscle and later from the viscera, are greater than had been thought in the past. It is likely that this degree of loss profoundly affects muscle function and hence weaning from the ventilator and convalescence.[25]

THE CLINICAL SCENARIO

Peritonitis after intestinal perforation is one of the most common septic states. In contrast to the trauma victim, who is well nourished and healthy until the accident, the patient with perforative peritonitis often has an underlying disease, such as typhoid fever, tuberculosis, or duodenal ulcer, which affects his nutritional status adversely. As stressed earlier, social customs in India compound this problem. The patient is thus exposed to a very high risk of morbidity and mortality.

There is an unsuspected prevalence of malnutrition in hospitalized patients. Up to 40% of patients are malnourished at the time of admission to the hospital. Those patients who undergo major surgery are at further risk of malnutrition as a result of starvation, the stress of surgery, and a subsequent increase in their metabolic rate.[27]

In the critically ill patient, malnutrition results in impaired immunologic function, impaired ventilatory drive, and weakened respiratory muscles leading to prolonged ventilator dependence and increased infectious morbidity and mortality rates.[28]

Damage to the body induces a stress response characterized by hypermetabolism, impaired protein synthesis, and catabolism. The degree of the stress response differs with the causative agent, but metabolism and oxygen consumption may increase by as much as 50% in patients with peritonitis.[5] The decreased whole-body protein synthesis and increased catabolism result in a net

protein loss. This can quickly cause protein-calorie malnutrition, which is associated with organ dysfunction. A subclinical multiple-organ dysfunction syndrome evolves, which increases the patient's risk of septic complications. Acute protein malnutrition is also known to adversely affect both humoral and cell-mediated immunity. The protein catabolism is largely obligatory, but protein synthesis increases with substrate availability, and provision of adequate proteins during this period can reduce the net nitrogen loss.[5]

Prior to surgery patients are often feel nauseas or are starved for investigations. After surgery there is classically a period of being 'nil by mouth' before fluids then solids are gradually introduced. Within 24 hr of starvation changes in the body metabolism are evident including increased insulin resistance and reduced muscle function.[29]

Provision of oral nutrients in the postoperative period historically has been withheld until bowel activity returns. Return of bowel sounds, flatus, and defecation are used to herald the return of bowel activity. This period may be prolonged in patients with peritonitis. Traditionally, when required, nutritional support under these circumstances has been delivered by total parenteral nutrition (TPN).

Clearly, outcome also is influenced by other factors as well. The presence of advanced age, renal, cardiac, hepatic, or pulmonary insuffiency, malignancy and diabetes all increase the mortality associated with bacterial peritonitis, perhaps as much as three fold.[30]

EFFECT OF NUTRITION

Surgical and accidental trauma is well known to cause a transient suppression of the immune system, which increases the infection risk. There is consensus that nutritional support is an essential component of the multidisciplinary treatment of surgical and critically ill patients, especially when the illness is associated with prolonged catabolism and with the inability to use the GI tract. Such circumstances occur frequently in severely septic surgical patients; artificial nutrition can optimise their recovery by supplying vital energy and nitrogen substrates, along with vitamins and oligoelements.[31]

The general benefits of nutritional support include improved wound healing, a decreased catabolic response to injury, improved gastrointestinal permeability, decreased bacterial translocation, and improved clinical outcomes, including a decrease in complication rates and length of stay with accompanying cost savings.[28]

Experimental data from both humans and animals suggests that providing nutrition in the postoperative period improves wound healing (relevant to the integrity of the intestinal anastomosis), muscle function and reduces sepsis. Furthermore, it has been suggested that early enteral nutrition is useful for recovering gastrointestinal motility and maintaining the nutritional status for patients undergoing gastrointestinal surgery.[29]

The deficiencies of almost all nutrients may influence negatively host defences; conversely, many nutrients have the ability to enhance the immune defence.

There are four main modalities of artificial nutrition: oral supplementation of nutrients; enteral nutrition (EN); total parenteral nutrition (TPN); mixed parenteral and enteral nutrition (Figure 6).

In response to the awareness of the deleterious effects that malnutrition has on patients, significant advances have been made in the field of enteral and parenteral nutritional support during the peri- and postoperative periods. Whenever safe and efficient access to a functional gastrointestinal tract can be achieved, gut feeding is preferred over TPN. With gut feeding, the liver has the first opportunity to clear, process, and distribute the nutritional components. Also, the vital gut functions such as substrate traffic, the gut mucosal barrier, and immunocompetence are maintained. This route is also most economical.

Compared to parenteral nutrition, the EN provides nutrients in a more physiologic manner. EN is associated with reductions in infectious complications and cost when compared with parenteral feeding.[32] Additionally, EN when compared with current TPN solutions prevents gastro-intestinal mucosa atrophy, attenuates the injury stress response and preserves normal gut flora. Several studies have shown that the early administration of EN promotes the restoration of GI mucosa integrity in nutritionally depleted patients; such benefit is not observed with TPN, because with TPN the mucosa continues to present increased permeability, in spite of improved general nutritional status.

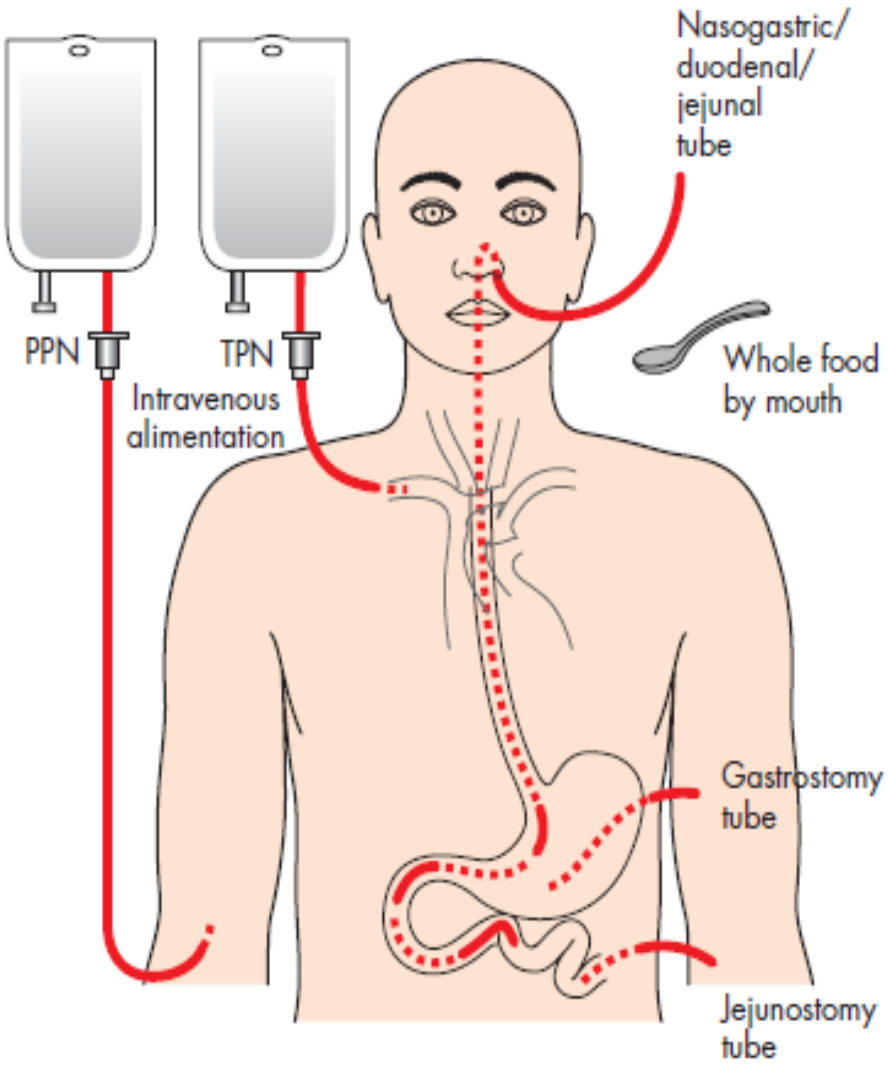


Figure 6: Various modalities of artificial nutrition

The EN has a specific trophic effect on the GI tract; such effect is potentially valuable in preventing microbial translocation from the gut to the blood stream and subsequent gut derived infection.[31]

However, nutritional support is not without adverse effects and risks. Early EN may be associated with high gastric residuals, bacterial colonization of the stomach, and increased risk of aspiration pneumonia. PN has been associated with gut mucosal atrophy, overfeeding, hyperglycaemia, an increased risk of infectious complications and increased mortality rates in critically ill patients. Both forms of nutritional support can affect cost and workload.[28]

The potential complications of enteral and parenteral nutrition are described in Table 1 and 2.[31]

Table 1. Complications of enteral nutrition

Gastro-intestinal complications
- Diarrhoea
- Gastric bloating and abdominal distension
- Abdominal pain
- Nausea and vomiting
- Regurgitation – inhalation pneumonia
Mechanical complications
- Misplacement of feeding tube
- Occlusion of feeding tube
- Inflammation, bleeding, perforation of nose, pharynx, oesophagus, stomach
Metabolic complications
- Hyperglycaemia
- Electrolyte alterations

Table 2. Complications of parenteral nutrition

Complications related to the venous catheter placement technique
- Pneumothorax
- Misplaced catheter
- Subclavian artery injection / trauma
- Haemothorax
- Venous embolism
- Venous thrombosis
Complications related to catheter maintenance
- Occlusion
- Sepsis
- Intravascular knotting
Metabolic complications
- Hyperglycaemia
- Hypoglycaemia
- Electrolyte alterations
- Adverse reactions hypersensitivity (amino acids, fats, etc.)

EARLY VERSUS DELAYED NUTRITION

There have been over 14 randomized controlled trials comparing early enteral nutrition vs. delayed nutrient intake (i.e. delayed enteral nutrition, parenteral nutrition or oral diet). In all the trials, except one (started within 72 hours of injury), enteral nutrition in the intervention group was started within 24-48 hours of admission/resuscitation. Of these, there were 8 studies comparing early vs. delayed EN whereas in 6 studies early EN was compared to no EN/IV fluids. The comparison revealed the following results:[33]

Mortality:

When all the studies that looked at the effect of early EN on mortality were aggregated, and compared for delayed nutrient intake, early enteral nutrition was associated with a trend towards a reduction in mortality (RR 0.68 95% CI 0.46,1.01, p =0.06, no heterogeneity present). In a subgroup analysis, early EN vs. no EN/IV fluids was associated with a trend towards a reduction in mortality (RR 0.62, 95% CI 0.37, 1.05, p =0.08, no heterogeneity present), whereas early vs. delayed EN had no effect on mortality (RR = 0.77, 95% CI 0.43, 1.38, p = 0.39, no heterogeneity present).

Infections:

Nine studies reported on infections and of these only 7 studies reported on the number of patients with infections and when these were aggregated,

early enteral nutrition when compared to delayed nutrient intake was associated with a significant reduction in infectious complications (RR 0.76, 95 % confidence intervals 0.59, 0.98, p = 0.04). In a subgroup analysis, early EN vs. no EN/IV fluids was associated with a trend towards a reduction in infections (RR 0.70, 95% CI 0.48, 1.02, p= 0.06, moderate heterogeneity present), whereas early vs. delayed EN had no effect on infections (RR = 0.79, 95% CI 0.5, 1.25, p = 0.31, no heterogeneity present).

Length of Stay (LOS) and Ventilator days:

Thirteen studies looked at LOS (5 reported on ICU LOS only, 3 reported on hospital LOS only and 5 reported on both ICU and hospital LOS). When the results were meta-analysed, early enteral nutrition had no effect on ICU stay (WMD -0.18, 95% CI -3.32, 2.96, p =0.91) or hospital length of stay (WMD – 0.18, 95%CI -8.15, 7.80 p = 0.97). A total of 7 studies reported on ventilator days and all showed no significant differences between the early vs. delayed fed groups (WMD 0.03, 95% CI -3.01, 3.06 p= 0.99).

Other:

All thirteen studies that reported nutritional endpoints showed a significant improvement in the groups receiving early enteral nutrition (calorie intake, protein intake, % goal achieved, faster nitrogen balance achieved). There were no differences in other complications between the groups.

From the above results, following conclusions can be reached:

1) Early enteral nutrition, when compared to delayed nutrient intake is associated with a trend towards a reduction in mortality in critically ill patients.

2) Early enteral nutrition, when compared to delayed nutrient intake is associated with a significant reduction in infectious complications.

3) Early enteral nutrition, when compared to delayed nutrient intake has no effect on ICU or hospital length of stay.

4) Early enteral nutrition, when compared to delayed nutrient intake improves nutritional intake.

ROUTES OF DELIVERING ENTERAL NUTRITION

Enteral nutrition can be achieved using oral feeds, nasogastric tubes (Ryle's), fine-bore feeding tubes inserted into the stomach, surgical or percutaneous endoscopic gastrostomy (PEG) or, finally, post-pyloric feeding utilising nasojejunal tubes or various types of jejunostomy. The choice of method will be determined by local circumstances and preference in many patients. Whichever method is adopted it is important that tube feeding is supervised by an experienced dietician who will calculate the patient's requirements and aim to achieve these within 2–3 days of the instigation of feeds. Conventionally, 20–30 ml is administered per hour initially, gradually increasing to goal rates within 48–72 hours. In most units feeding is discontinued for 4–5 hours overnight to allow gastric pH to return to normal. There is some evidence that this might reduce the incidence of nosocomial pneumonia and aspiration. There is good evidence to confirm that feeding protocols optimise the tolerance of enteral nutrition. In these, aspirates are performed on a regular basis and if they exceed 200 ml in any 2-hour period then feeding is temporarily discontinued.[34]

Nasoenteric and Postpyloric Feeding

Nasoenteric feeding (gastric, duodenal, or jejunal) is the least expensive and most widely used modality of enteral nutrition. Most commonly, postsurgical patients have nasogastric tubes in place. These tubes are

reasonable for the short term because they are typically large bored, do not clog easily, and allow gastric residuals to be checked in assessing GI tolerance.

A variety of approaches have been tried in an attempt to address poor gastric emptying in critically ill patients, including the use of promotility agents such as metoclopramide or erythromycin. Another strategy proposed as a means of bypassing the region of gastroduodenal ileus is postpyloric feeding. Nasoenteric feeding tubes can be placed with their tip positioned in the duodenum or jejunum, either under fluoroscopic guidance or by endoscopic manipulation and visualization. The hypothesis is that the jejunum may be more tolerant of continuous feeding and that by administering nutrients beyond the ligament of Treitz, the risk for aspiration is lessened. However, when these putative advantages have been studied in prospective randomized trials, there did not appear to be any difference when compared with intragastric feeding practices. In fact, in studies involving the use of radiolabeled feeding, regurgitation of postpylorically delivered nutrients and the incidence of actual aspiration or clinically definable pneumonia were no different than in patients fed gastrically.[35]

As for feeding tolerance, there also does not appear to be any clear benefit attributable to postpyloric feeding. When aggressive advancement protocols are followed, nasogastrically fed patients, despite having higher gastric residual volumes, receive amounts of enteral nutrition equivalent to

those fed nasojejunally. This finding has been confirmed in two separate prospective randomized trials that included 180 patients.[35]

Gastrostomy

If long-term access to the stomach will be needed, a permanent gastrostomy can be placed. This goal can be achieved either by the open approach or by percutaneous techniques, the latter using endoscopic, radiologic, or laparoscopic methods. The Stamm gastrostomy, which requires a small laparotomy incision, is the most widely used open technique for insertion of a gastric tube. Drawback of gastrostomy tubes of all types is that they generally do not lie in a dependent position, so it is difficult to aspirate and check gastric residual volumes.[35]

Jejunostomy

Jejunal or small bowel feeding tube access can be achieved by open jejunostomy (either at the time of laparotomy or as a separate procedure), percutaneously by extension through an existing gastrostomy tube (often termed a *G-J tube*), by a laparoscopic approach, or very rarely as a percutaneous jejunostomy placed under fluoroscopic or CT guidance by the interventional radiologist. This latter procedure has an undefined but presumably high frequency of complications and is of dubious value. True percutaneous jejunostomies (as opposed to G-J tubes), though often lifesaving, are complicated more frequently than desired by dislodgement, occlusion,

bowel obstruction, and small bowel ischemia. Furthermore, because the small bowel does not accommodate bolus feeding, nutrients delivered to the jejunum must be delivered in continuous fashion while carefully watching for signs of intolerance such as abdominal distension, abdominal pain or tenderness, diarrhoea, or constipation. In a critically ill patient, hypo-osmolar or at most iso-osmolar solutions should generally be used. Hyper-osmolar solutions are often not tolerated in critical illness because the bowel is stressed to begin with and such solutions are much more likely to result in pneumatosis, necrosis, perforation, and death.[35]

THE RATIONALE OF EARLY ENTERAL NUTRITION FOLLOWING GASTROINTESTINAL SURGERIES

The rationale of nil by mouth and gastric decompression is to prevent postoperative nausea and vomiting and protect the anastomosis, allowing it time to heal before being stressed by food. Nausea and vomiting, however, occur more commonly after upper gastrointestinal surgery than after resection of the small intestine and colon. There is no evidence that bowel rest and a period of starvation are beneficial for healing of wounds and anastomotic integrity. Instead, there is evidence that luminal nutrition may enhance wound healing and increase anastomotic strength, particularly in malnourished patients.[36]

Enteral feeding has gained popularity for the nutritional support of surgical patients. Biologically, there have been several reasons reported including better substrate utilization, prevention of mucosal atrophy, preservation of normal gut flora, gut integrity, and immune competence. In addition, improvement of peripheral and whole body protein kinetics with enteral nutrition has been shown.[4]

Large volumes of gastric and pancreatic secretions (1–2 L/d) are handled safely by the intestine during the period of ileus. Small-bowel peristaltic activity returns within 6–12 hours of the operation, and thus nutrients delivered directly into the small bowel in the immediate postoperative period can be used effectively.[5]

The gut is a major interface between the host and the environment and is metabolically and immunologically active in the stressed patient. Luminal nutrients are important for intestinal mucosal metabolism and integrity, regardless of the general nutritional status. Animal studies have demonstrated that TPN causes a decrease in villous height, mucosal weight, and the content of DNA, RNA, and protein. Altered disaccharidase activity also has been observed. These alterations may allow bacteria to translocate across the gut into the circulation, which in turn could stimulate the release of cytokines responsible for the hypermetabolic response seen in sepsis. Total enteral nutrition may prevent this scenario.[5]

There are several other reasons why TEN is more beneficial than TPN. Tremendous differences exist between the costs of therapy and the complications associated with each. Total enteral nutrition has physiologic advantages over TPN because it stimulates gall bladder contraction, maintains gut associated lymphoid tissue, and stimulates immune function and pancreatic secretion. It also maintains gut integrity, as evidenced by a decreased risk of perforation and better healing of gut anastomoses.[5]

In the past few years, some studies have examined the role of early feeding after gastrointestinal anastomosis and found that it improved immumocompetence, decreased septic complications, improved wound healing and possibly improved anastomotic strength.[8,37,38,39,40,41]

Two studies on postoperative enteral feeding showed that nutritional support was associated with a significant reduction in postoperative

complications, a reduction that was independent of preoperative nutritional status.[11,42]

The benefits of postoperative enteral feeding in normally nourished surgical patients indicate that it is the reduced nutritional intake that predisposes patients to developing complications, including deficits in muscle function and surgical fatigue.[42] There is thus no evidence that early postoperative enteral feeding should be restricted to malnourished patients undergoing gastrointestinal resection. Indeed, one study has found that supplementing "normal" oral diet in hospital wards with as little as 1250 kJ (300 kcal) and 12 g of protein per day resulted in a reduction of postoperative complications in patients undergoing gastrointestinal surgery.[42]

The timing of feeding, as related to surgery, also influences the clinical outcome. The earlier the patient is fed enterally; the better is the clinical outcome. The EN usually can begin postoperatively as soon as the patient is haemodynamically stable. Preferably it should start within 24 hours after surgery and no later than 48 hours. As long as there is no significant abdominal distension, enteral feeding is not contraindicated, even with markedly diminished bowel sounds. Most patients can be fed enterally without waiting for flatus. Immediate or early postoperative EN stimulates the splanchnic and hepatic circulation; it improves intestinal mucosa blood flow**,** it prevents intramucosal acidosis and permeability disturbances and it eliminates the need for stress ulcer prophylaxis.[31]

Perforative peritonitis

A large number of patients who present to our hospital for emergency surgery for perforation peritonitis are malnourished at the time of admission. In peritonitis, enteral nutrition is not routinely used because of the oedematous and paralytic characteristics of the bowel. However, the role of early enteral nutrition (EEN) in peritonitis has been investigated by many workers.

EEN by jejunostomy was found to reduce septic complications in patients with severe pancreatitis and secondary peritonitis.[43] Glutamine-enriched enteral diet was found to be well tolerated in patients with purulent peritonitis.[45]

Singh *et al.*[5] described a prospective study for early postoperative feeding in patients with non-traumatic intestinal perforation and peritonitis. Study spanning 1 year was conducted on patients with nontraumatic intestinal perforation and peritonitis. After laparotomy, patients were assigned randomly to a control or study group. The study group underwent a feeding jejunostomy and received enteral feeding from 12 hours postoperatively. A low-residue, milk-based diet was used. All patients underwent assessment for severity of sepsis and nutritional status at admission. Studies of nutritional status and nitrogen balance were repeated on days 4 and 7. Forty-three patients (21 in the study group; 22 in the control group) were included. The two groups were comparable except for a higher sepsis score in the study group ($p < 0.05$). Patients in the study group achieved a positive nitrogen balance by the third postoperative day; patients in the control group remained in negative nitrogen

balance throughout the study. Abdominal distension (four patients) required temporary withdrawal of feeding. Diarrhoea occurred in four patients but was controlled easily. The mortality rate was similar in the control and study groups (18.2% versus 19.1%). The control group had a total of 22 septic complications, versus eight in the study group ($p < 0.05$). They concluded that immediate postoperative feeding is feasible in patients with perforative peritonitis and reduces septic morbidity.

However, since patients with perforation peritonitis require supplementation for only a short period, the feasibility of EEN through a nasoenteric and nasogastric tubes in patients presenting with nontraumatic perforation peritonitis with malnutrition have also been evaluated.[46,2]

Kaur *et al.*[6] conducted a prospective study to assess the feasibility and short-term efficacy of early enteral feeding through a nasoenteric tube placed intraoperatively in patients with nontraumatic perforation peritonitis with malnutrition. One hundred patients with nontraumatic perforation peritonitis with malnutrition undergoing exploratory laparotomy were randomly divided into a test group (TG) and a control group (CG) of 50 patients each. TG patients had a nasoenteric tube placed at the time of surgery in duodenum or jejunum and were started on an enteral feeding regime 24 hours postoperatively. Patients in CG were allowed to eat orally once they passed flatus. The differences between the two groups with respect to nutritional intake in terms of energy and protein, changes in nutritional status as assessed by anthropometric, biochemical, and haematological values, amount of

nasogastric aspirate, return of bowel motility, and complication rates were analysed. The nasoenteric feeding was well tolerated. Total calorie and protein intake in TG was significantly higher than in CG: 981 vs. 505 kcal ($p < 0.01$), protein 24 vs. 0 g on day 3 and 1498 vs. 846 kcal ($p < 0.01$), protein 44 vs. 23 g ($p < 0.01$) on day 7, respectively. There was reduction in the amount of nasogastric aspirate in TG compared with that in CG: 431 vs. 545 ml/24 h on day 2 and 301 vs. 440 ml/24 h on day 3, respectively. There was much faster recovery of bowel motility in TG than in CG at 3.34 vs. 4.4 days ($p < 0.01$). Complications developed in 39 of 50 patients in TG and in 47 of 50 in CG. The major complications occurred in 6 patients in TG and 12 patients in CG ($p < 0.05$). Patients with perforation peritonitis with malnutrition are likely to develop large energy deficits postoperatively, resulting in higher incidence of infective complications. They concluded that early enteral feeding through a nasoenteric tube is well tolerated by these patients and helps to improve energy and protein intake, reduces the amount of nasogastric aspirate, reduces the duration of postoperative ileus, and reduces the risk of serious complications.

Malhotra *et al.*[2] described a prospective randomized study for early enteral nutrition after surgical treatment of gut perforation. Patients undergoing surgical intervention for peritonitis following perforation of the gut were randomised to the study group receiving feedings of a balanced diet formula through a naso-gastric tube from the second postoperative day, or the control group in which patients were managed with the conventional regimen of intravenous fluid administration. The groups were compared for incidence and

duration of complications, biochemical measurements and other characteristics like weight loss/gain. One hundred patients were enrolled in each group. 88% patients in the study group achieved positive nitrogen balance on the eighth postoperative day as compared to none in the conventionally managed group. The relative risks (95% confidence interval) of morbidity from wound infection, wound dehiscence, pneumonia, leakage of anastomoses and septicaemia were 0.66 (0.407-1.091), 0.44 (0.141-1.396), 0.70 (0.431-1.135), 0.54 (0.224-1.293) and 0.66 (0.374-1.503) respectively. Average loss of weight between the first and tenth day was 3.10 kg in the study group as compared to 5.10 kg in the conventionally managed group ('P' value < 0.001, 95% Confidence Interval - 2.46 - 1.54). They concluded that early enteral nutrition is safe and is associated with beneficial effects such as lower weight loss, early achievement of positive nitrogen balance as compared to the conventional regimen of feeding in operated cases of gut perforation.

Material & Methods

MATERIALS AND METHODS

SOURCE OF DATA:

All patients coming to B.L.D.E.U.'s Hospital and admitted patients in whom the diagnosis of gut perforation and peritonitis was made, who underwent exploratory laparotomy.

METHOD OF COLLECTION OF DATA:

- Patients who underwent exploratory laparotomy for gut perforation in B.L.D.E.U.'s Hospital, Bijapur, from October 2010 to May 2012.
- Patients underwent necessary investigations and standard treatment, prior to surgery, as per protocol.
- Naso-gastric tube aspiration, as routine, was performed, before surgery.
- Following surgery, those patients who did not undergo ileostomy / colostomy were included in this study, after obtaining informed consent.
- The patients were allotted, alternately, either to receive enteral formula within 24-48 hours, along with intravenous fluids (Group A) or to receive only intravenous fluids for up to 5 days (Group B).
- Postoperatively, continuous aspiration through a naso-gastric tube was provided for 24 hours, to both groups.

- In the patients belonging to Group A, the naso-gastric tube was used for both feeding and aspiration. Feeds were given slowly at a rate of 30-50 ml/hour by an intravenous drip set connected to a naso-gastric tube. This was over and above the dextrose-containing fluids given intravenously.
- The conventionally managed patients received calories only in the form of dextrose-containing fluids intravenously.
- From the third postoperative day, in addition to enteral feeds, patients belonging to Group A were kept on intravenous patency line. Between the third and fifth day the naso-gastric tube was removed and complete oral feeds in the form of semi-solid diet was commenced.
- Patients in Group B were assessed for the feasibility of oral intake on the fifth postoperative day and those found suitable were given sips of an appetizing liquid. Those tolerating the sips graduated to 500-ml liquids and then semi-solids over the next two days. Those who did not tolerated oral feed stayed on intravenous fluids till they could take feeds orally.
- Patients were closely monitored and feeding was slowed or stopped if complications related to tube feeding occurs. The patients were watched closely for signs of a leak from the repaired perforation of the gut.
- Postoperatively, the patients were subjected to certain investigations at regular intervals:

  i) Determination of weight on the first, fourth, seventh and tenth postoperative days and/or at the time of discharge.

ii) Biochemical and haematological investigations that were done, included: estimation of haemoglobin concentration, levels of albumin and creatinine in the serum on the third and eighth postoperative days.

- The groups were compared for complications, biochemical measurements and other characteristics like weight loss/gain and duration of stay.
- Final outcome was evaluated.

INCLUSION CRITERIA:

All patients who underwent emergency laparotomy for gut perforation in B.L.D.E.U.'s Hospital, Bijapur.

EXCLUSION CRITERIA:

1. Following surgery, those patients who did not underwent ileostomy / colostomy.
2. Those patients who did not gave consent.
3. Those patients who left the study in between, due to any reason.

RESEARCH HYPOTHESIS:

Early enteral nutrition is as safe as 'nil by mouth' approach after surgical treatment of gut perforation and is associated with beneficial effects such as lower weight loss and lower rate of complications.

SAMPLING:

Study period from: October 2010 to May 2012.

All the patients admitted during this period, who fulfilled the inclusion criteria, were included in this study.

Allowable error was considered as 15%.

Formula used to calculate the sample size was

$$n = [(1.96)^2 \times p \times (1-p)]/L^2$$

Using this formula, the minimum sample size was n= 43, in each group.

Following statistical tests were used to compare the results:

i) Mean ± S.D.

ii) Chi square test

iii) 'Z' test – difference between two proportions.

iv) Diagrammatic presentations.

INVESTIGATIONS / INTERVENTIONS:

Investigations or interventions required in this study were routine standardized procedures.

There were no animal experiments involved in this study.

These investigations were required as routine before taking any patient for laparotomy and for routine postoperative follow-up:

1. Complete blood count.
2. Urine – sugar, albumin and microscopy.
3. Random blood sugar, Serum creatinine, Serum albumin, Blood urea.
4. Electro-cardio-gram and Chest X-ray (when age of patient is >35yrs, or if necessary).
5. X-ray erect abdomen – to look for free gas under diaphragm.
6. Ultrasonography of abdomen, if required.
7. Tests of detect infection with Human Immunodeficiency Virus and Hepatitis B Virus (in accordance to Universal Safety Precautions).
8. Pus / Peritoneal fluid culture and sensitivity.

Estimation of haemoglobin concentration, levels of albumin and creatinine in the serum were repeated on the third and eighth postoperative days.

Observations & Results

OBSERVATION AND RESULTS

During the study period, i.e. from October 2010 to May 2012, a total of 108 patients underwent emergency exploratory laparotomy for perforative peritonitis in our hospital.

Out of these, five patients did not give consent, and the remaining 103 patients were alternatively allotted on the intervention (Group A - 52) and control group (Group B - 51).

In this study, primarily the effect of early enteral feeding over the traditional ‘nil by mouth’ custom were analysed in terms of rate and duration of complications and the change in nutritional parameters of the patients. However, during the study, observations were also made regarding the age, sex and site of perforation in the patients.

Following are the observations made during this study shown in both tabular and graphical form:

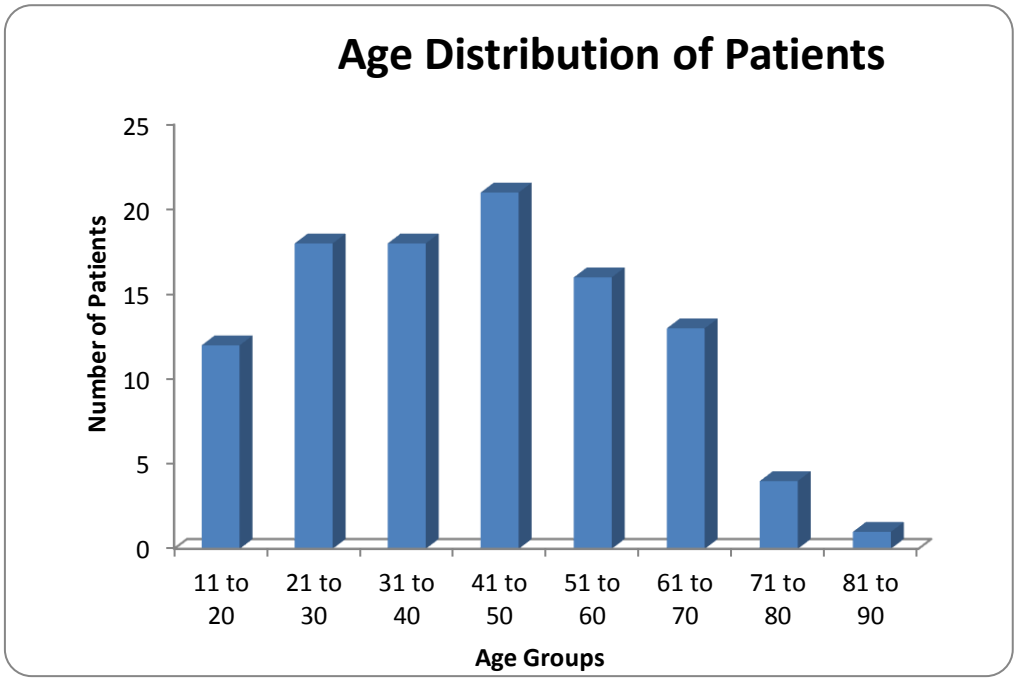
AGE DISTRIBUTION:

In this study, the age of patients ranged from 11 to 85 years. About 71% of the patients were in the age group of 21-60 years and 16.5% were in the age group of 61-80 years, as shown below:

Table 1

Age wise Distribution of Patients for Perforative Peritonitis

Age	Number	Percentage
11-20	12	11.65
21-30	18	17.48
31-40	18	17.48
41-50	21	20.39
51-60	16	15.53
61-70	13	12.62
71-80	04	3.88
81-90	1	0.97



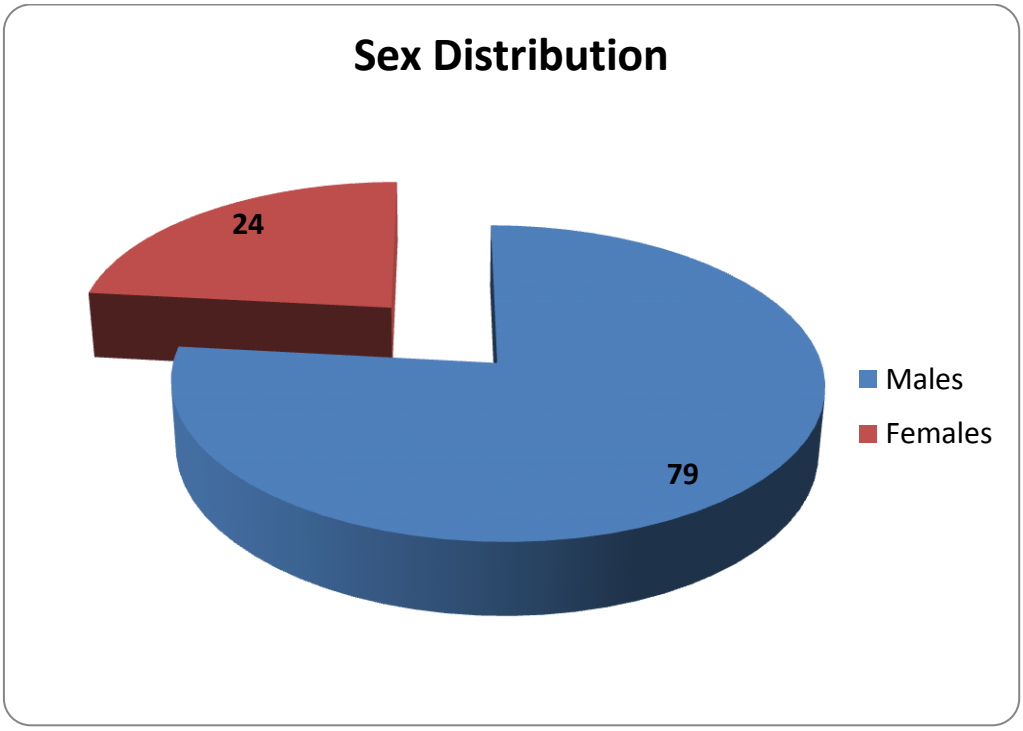
SEX DISTRIBUTION

About two-third of the patients were male in this study. Male:female ratio was 3:1, as shown in the following table:

Table 2

Sex wise Distribution of Patients for Perforative Peritonitis

Sex	Number	Percentage
Male	79	76.7
Female	24	23.3



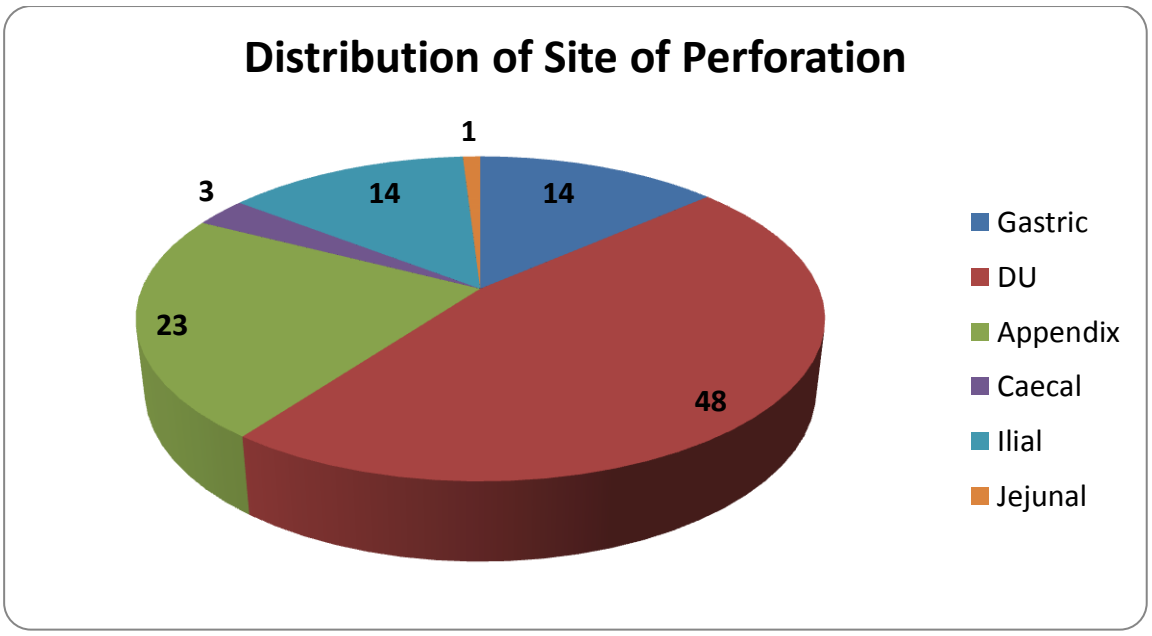
DISTRIBUTION OF SITE OF PERFORATION

The site of perforation involved almost all of the gastrointestinal tract, with over half of the patients presenting with duodenal ulcer perforation, followed by appendicular perforation in one-fourth of patients, followed by gastric, ileal, caecal and jejunal perforation.

Table 3

Distribution of Site of Perforation

Site of Perforation	Number	Percentage
Gastric	14	13.59
DU	48	46.60
Appendix	23	22.33
Caecum	3	2.91
Ileal	14	13.59
Jejunal	1	0.97



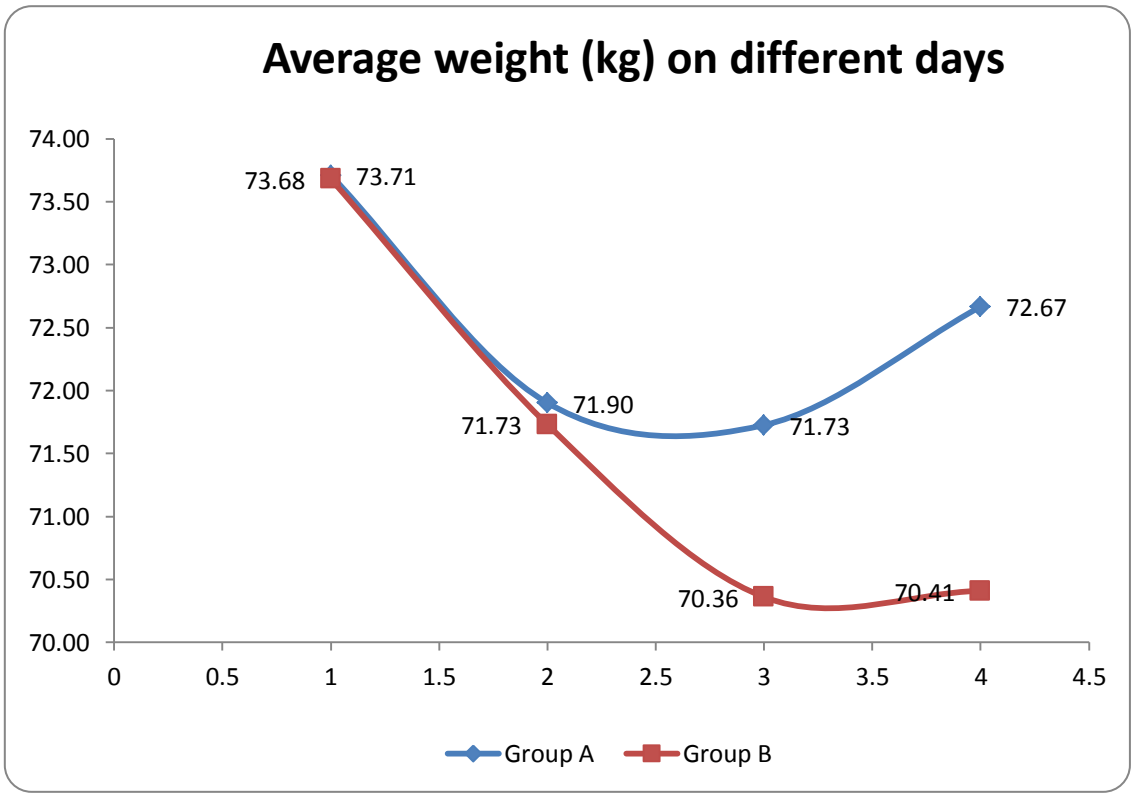
TREND OF CHANGE IN WEIGHT

Patients in both the group had an initial loss in weight on post-op day 3, but those in Group A fared better than those in Group B, both in terms of total weight loss and rate and duration of weight loss in later part of the study due to early commencement of feeding, as shown in the following table:

Table 4

Trend of change in weight (kg) in the two groups

	Day 0	Day 4	Day 7	Day 10
Group A	73.71	71.9	71.73	72.67
Group B	73.68	71.73	70.36	70.41



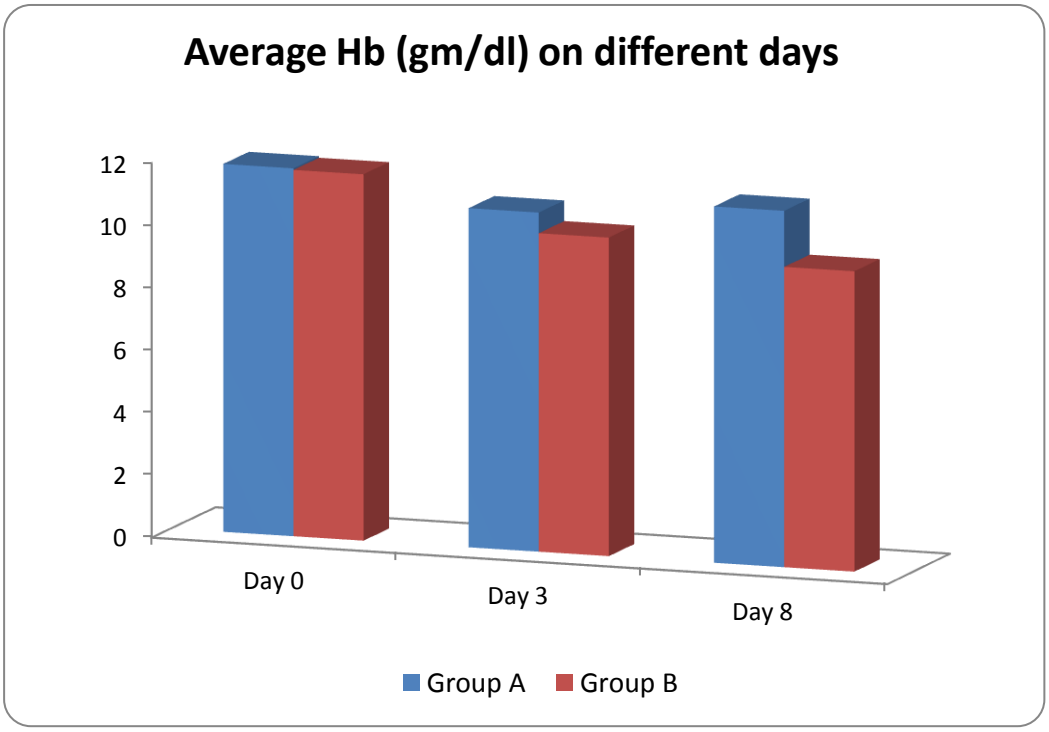
TREND OF CHANGE IN HAEMOGLOBIN LEVELS

Patients in both the group had an initial decrease in haemoglobin levels on post-op day 3, partially due to intra-op blood loss and partially due to haemodilution following resuscitation, but those in Group A fared better than those in Group B in later part of the study, due to early commencement of feeding, as shown in the following table:

Table 5

Trend of change in haemoglobin (gm/dl) levels in the two groups

	Day 0	Day 3	Day 8
Group A	11.79	10.87	11.42
Group B	11.74	10.21	9.63



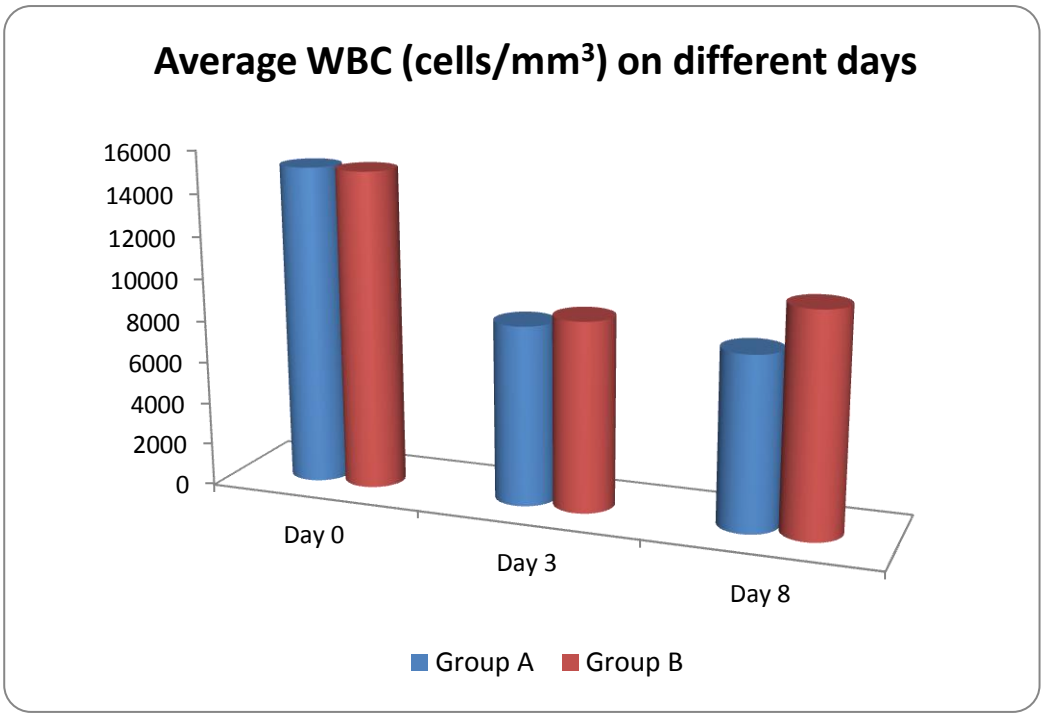
TREND OF CHANGE IN TOTAL LEUCOCYTE COUNTS (TLC)

Patients in both the group had an initial relative decrease in TLC on post-op day 3, following control of peritonitis, but those in Group A fared better than those in Group B in later part of the study, in terms of lesser post-op infective complications, as shown in the following table:

Table 6

Trend of change in total leucocyte counts (cells/mm^3) levels in the two groups

	Day 0	Day 3	Day 8
Group A	15117.65	8547.06	8237.25
Group B	15090.90	9018.18	10461.36



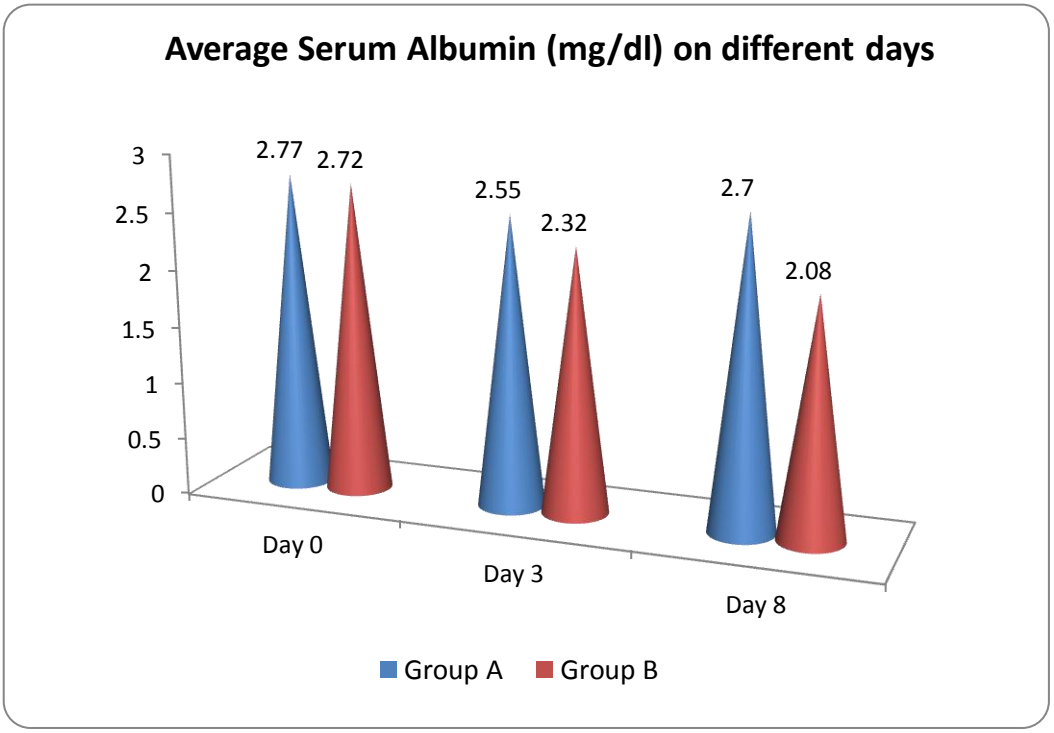
TREND OF CHANGE IN SERUM ALBUMIN LEVELS

Patients in both the group had an initial decrease in serum albumin levels on post-op day 3, but those in Group A fared better than those in Group B in later part of the study, in terms of recovery of levels due to early commencement of feeding, as shown in the following table:

Table 7

Trend of change in Serum Albumin (mg/dl) levels in the two groups

	Day 0	Day 3	Day 8
Group A	2.77	2.55	2.70
Group B	2.72	2.32	2.08



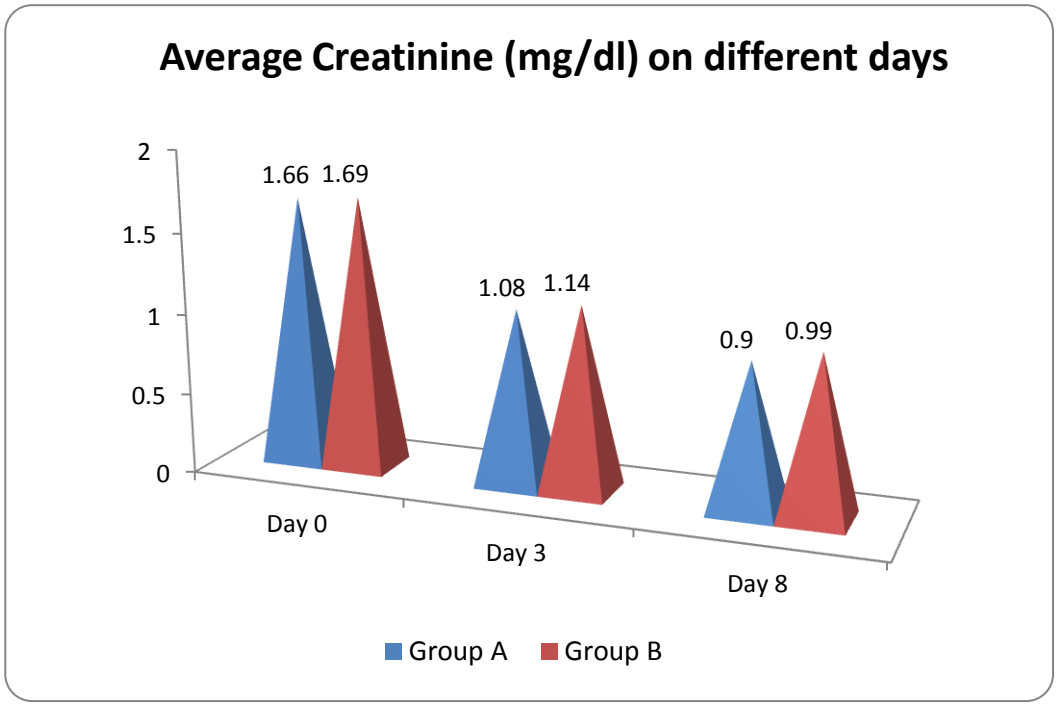
TREND OF CHANGE IN SERUM CREATININE

Patients in both the group had an initial improvement in serum creatinine levels on post-op day 3, following control of peritonitis, but those in Group A fared better than those in Group B in later part of the study, in terms of lesser post-op infective complications and early resolution of septicaemia, as shown in the following table:

Table 8

Trend of change in serum creatinine (mg/dl) levels in the two groups

	Day 0	Day 3	Day 8
Group A	1.66	1.08	0.90
Group B	1.69	1.14	0.99



RISK OF VARIOUS COMPLICATIONS

This study showed an increased incidence of abdominal distension and vomiting in Group A, probably due to early commencement of enteral feeds; while Group B showed increased incidence of infective complications, as shown in the following table:

Table 9

Relative Risk of Major and Minor Complications

Complications	Study Group	Control Group	P Value	Odds Ratio	Relative Risk (95% CI)
Abdominal Distension	12	8	0.2514	1.38	1.29 (-0.06 to 0.168)
Vomiting	10	4	<0.0001	2.44	2.16 (-0.023 to 0.223)
Pneumonia	8	18	0.0001	0.27	0.38 (0.092 to 0.4068)
Wound Infection	11	19	0.0082	0.36	0.50 (-1.916 to 2.356)
Wound Dehisence	2	5	0.088	0.32	0.35 (-0.04 to 0.18)
Leak	4	7	0.114	0.45	0.49 (-0.057 to 0.217)
Septicaemia	9	12	0.1314	0.57	0.65 (-0.076 to 0.276)
Death	1	5	0.034	0.16	0.17 (-0.008 to 0.188)

DURATION OF STAY AND COMPLICATIONS

This study showed increased duration of both total hospital stay and ICU stay and increased duration of infective complications in Group B, as compared to group A, as shown in the following table:

Table 10

Duration of Stay and Complications in the Two Groups

Parameter	Group A (Mean ± S.D.)	Group B (Mean ± S.D.)	Z Value	P Value
Duration of Total Stay	10.78 ± 1.14	17.07 ± 3.28	12.11	<0.0001
Duration of ICU Stay	5.89 ± 0.78	10.45 ± 2.25	12.80	<0.0001
Duration of Wound Infection	7.80 ± 0.79	9.79 ± 2.23	5.62	<0.0001
Duration of Pneumonia	6.25 ± 1.04	9.56 ± 2.87	7.25	<0.0001
Duration of Septicaemia	6.44 ± 0.73	8.58 ± 1.51	8.58	<0.0001
Duration of Postoperative Ileus	2.82 ± 0.56	3.82 ± 1.08	5.53	<0.0001

Most of the perforations (99/103) included in this study were more than 48 hours old, with severe peritonitis and septicaemia. Of the 52 patients in Group A, only 51 patients completed the study, one patient died. No patient left the study in between due to any complication of the intervention, as was in the study done by Malhotra *et al.* where three patients had to be withdrawn from the study group for the occurrence of intolerable side-effects (two cases of intractable diarrhoea and one case of intractable vomiting) [2]. In Group B, there were five deaths and two patients left the hospital against medical advice.

Analysis & Discussion

ANALYSIS AND DISCUSSION

AGE AND SEX DISTRIBUTION

In this study, majority of patients were males (male: female ratio was 3:1), with 67% of the patients below the age of 50 years and 33% above it. This was comparable to what was observed in the study done by Kaur *et al.*[6] The mean age of patients in the study group was 41.4 ± 16.8 years and mean age of patients in the control group was 43.7 ± 17.7 years, which is comparable to the observation made by Singh *et al.*[5] in their study.

SITE OF PERFORATION

In this study duodenal ulcer perforation comprised of almost half of the cases (46.6%) followed by appendicular perforation in one-fourth of cases (22.33%). Gastric and ileal perforation comprised of only about 13.59% each. These findings were in sharp contrast with the observations made by Singh *et al.*[5] and Kaur *et al.*[6] where incidence duodenal ulcer perforation was followed by ileal perforation. This difference can in part be attributed to better diagnostic and treatment modalities available at present times leading to decrease in intestinal complications of tubercular and typhoid.

EFFECT OF EARLY ENTERAL NUTRITION

Similar to some other studies[2-7, 16], our study has demonstrated that there is no evidence to suggest that bowel rest and a period of starvation are beneficial for the healing of wounds and anastomotic integrity. Indeed, the evidence is that luminal nutrition may enhance wound healing and increase anastomotic strength, particularly in malnourished patients.

In our study, weight loss between the first and seventh day was 1.98 kg in the study group as compared to 3.5 kg for the conventionally managed group. The weight gain between the seventh and tenth day it was 0.94 kg for the study group and 0.05 kg for the conventionally managed group. The total weight loss between the first and tenth day was 1.04 kg vs. 3.32 kg, for the study and control groups respectively. Between the seventh and tenth day, in the study group, almost all patients, recorded a gain in weight during the latter part of their stay. These results are comparable to those observed by Malhotra *et al.*[2] and Kaur *et al.*[6] in their studies.

Table 11

Comparison of loss of weight between various studies

Study	Loss of weight between day of admission and discharge (in kg)	
	Study Group	**Control Group**
Malhotra *et al.*[2]	1.97	2.79
Kaur *et al.*[6]	3.10	5.10
Our study	1.04	3.32

Also, there was a statistically significant difference in haemoglobin and serum albumin levels in study group vs. control group. On an average, Haemoglobin levels in study cases showed an increase of 0.55 gm/dl between postop day 3 & 8, while levels of control cases declined by 0.58 gm/dl during the same period. Similarly, while levels of serum albumin, in study cases, increased by 0.15 gm/dl, those of control cases decreased by 0.24 gm/dl, between postop day 3 & 8, as also shown in other studies.[3,6] Also, patients in the study group were in much better general condition than the patients who received conventional management, signifying the importance of alimentation.

Table 12

Comparison of Serum Albumin Levels between various studies

Study done by	Serum Albumin levels (gm/dl) (Mean ± SD)					
	Study Group			Control Group		
	Day 0	Day 3	Day 7/8	Day 0	Day 3	Day 7/8
Kaur *et al.*[6]	2.40 ±0.47	2.41 ±0.54	2.56 ±0.52	2.41 ±0.40	2.9 ±0.37	2.20 ±0.35
Vaithiswaran *et al.*[3]	3.11 ±0.41	3.06 ±0.41	3.11 ±0.36	3.16 ±0.59	3.14 ±0.54	3.11 ±0.57
Our study	2.77 ±0.27	2.55 ±0.32	2.70 ±0.36	2.72 ±0.41	2.32 ±0.33	2.08 ±0.41

Most studies[2,5-7,46] showed that the duration of septicaemia was significantly reduced along with a reduction in the duration of hospital stay. In our study, the average duration of stay in the study group was 10.78 ± 1.14 days, as compared to 17.07 ± 3.28 in conventionally managed group (p <0.0001). Similarly, the mean duration of septicaemia was 6.44 ± 0.73 days in the study group, as compared to 8.58 ± 1.51 days in control group (p<0.0001). Also, the mean duration of ICU stay was significantly lower in the study group as compared to the control group (5.89 ± 0.78 vs. 10.45 ± 2.25 days, respectively).

Table 13

Comparison of hospital & ICU stay between various studies

	Total Hospital Stay (days)		**ICU Stay (days)**	
Study done by	**Study**	**Control**	**Study**	**Control**
Malhotra *et al.*[2]	10.59	10.70	1.59	2.01
Our study	10.78	17.07	5.89	10.45

Though, in our study, as shown in table 9, the incidences and relative risks of morbidity from various major and minor complications like pneumonia, wound infection, anastomotic leak and death was lower in the

study group as compared to the control group. The difference was statistically not much significant. But on the other hand, the duration and severity of these complications, including the duration of postoperative ileus, were significantly lower in the study cases when compared with the control cases. This difference in duration of complications was statistically very much significant (Table 10). This translates to lower hospital stay, lower ICU stay and faster return of the patient to his work with a better health and nutritional level. Similar observations were made in studies done by Singh *et al.*[5], Malhotra *et al.*[2] and Kaur *et al.*[6]

Table 14

Comparison of complication frequency between various studies

Complication	Singh *et al*[5]		Malhotra *et al*[2]		Kaur *et al*[6]		Our study	
	Study	Control	Study	Control	Study	Control	Study	Control
Pneumonia	3	8	21	30	9	13	8	18
Infection	1	4	27	31	7	8	11	19
Dehisence	1	1	4	9	3	4	2	5
Leak	4	3	7	13	NR	NR	4	7
Septicaemia	1	2	20	30	3	8	9	12

NR – Not Reported

In our study, the incidence of nausea and vomiting was higher in study group (19.6%) as compared to control group (0.09%), with a relative risk of 2.16. This correlated well with most of the other studies[2-5] but was different from Carr *et al.*[45] who in fact demonstrated less distension and diarrhoea in their enterally fed group. The difference in the route of feeding, naso-gastric or naso-jejunal versus feeding jejunostomy could be the reason for this difference. Also, jejunostomy feeding may result in certain complications like clogging, dislodgement, abdominal wall infection, enterocutaneous fistula, bowel necrosis,[4,47] which are avoided by our technique.

Table 15

Comparison of frequency of abdominal distension and vomiting between various studies

Study done by	Incidence of Abdominal Distension (%)		Incidence of Vomiting (%)	
	Study	Control	Study	Control
Malhotra *et al.*[2]	20	18	13	7
Vaithiswaran *et al.*[3]	10	0	3	0
Heslin *et al.*[4]	58	54	25	20
Singh *et al.*[5]	19	0	NR	NR
Our study	24	18	20	9

NR – Not Reported

Lastly, as results our study and that done by Malhotra *et al.*[2] (done using naso-gastric feeding) are comparable to that done by Kaur *et al.*[6] who employed naso-jejunal feeding, the tedious procedure of insertion of and complications of naso-jejunal tube can also be avoided by our simple technique, while obtaining similar results.

Summary

SUMMARY

This study was conducted from October 2010 to May 2012, where a total of 103 patients underwent emergency laparotomy for perforative peritonitis due to various causes. The study group comprised of 52 patients, who were given enteral feeds via naso-gastric tube within 24-48 hr of surgery, over and above to intravenous fluids. The control group comprised of 51 patients who were managed traditionally by keeping them 'nil by mouth', providing them only intravenous fluids for a minimum of five days. The two groups were compared in terms of nutritional improvement and incidence and duration of various complications.

In our study:

- Male : Female ratio was 3:1
- Majority of the patients belonged to the age group of 21-60 years (70.88%).
- Most common site of perforation was duodenum (46.6%), followed by appendix (22.33%), gastric and ileal (13.59% each).
- The average total weight loss between the first and tenth day was 1.04 kg vs. 3.32 kg, for the study and control groups respectively ('P' value < 0.0001, 95% Confidence Interval → 2.01 – 2.44).
- Haemoglobin levels in study cases showed an increase of 0.55 gm/dl between postop day 3 & 8, while levels of control cases declined by

0.58 gm/dl during the same period ('P' value < 0.0001, 95% Confidence Interval → 1.103 – 1.157).

- Similarly, while levels of serum albumin, in study cases, increased by 0.15 gm/dl, those of control cases decreased by 0.24 gm/dl, between postop day 3 & 8 ('P' value < 0.0001, 95% Confidence Interval → 0.350 – 0.429).
- There was decreased incidence and decreased duration of complications like pneumonia, wound infection, wound dehiscence, leak, septicaemia and death, in the intervention group as compared to the control group [Relative risks (95% confidence interval) of 0.38 (0.092 – 0.4068), 0.50 (-1.916 – 2.356), 0.35 (-0.04 – 0.18), 0.49 (-0.057 – 0.217), 0.65 (-0.076 – 0.276) and 0.17 (-0.008 – 0.188) respectively].
- However, there was increased incidence of abdominal distension and vomiting in the intervention group as compared to the control group, probably due to early enteral feeding [Relative risks (95% confidence interval) of 1.29 (-0.06 – 0.168) and 2.16 (-0.023 – 0.223), respectively].
- The duration of postoperative ileus was significantly reduced in the study group compared to control group (2.82 ± 0.56 vs. 3.82 ± 1.08 days; p<0.0001).
- The length of total hospital stay and ICU stay were significantly lower in the study group as compared to control group (10.78 ± 1.14 days, vs.

17.07 ± 3.28 ($p < 0.0001$) and 5.89 ± 0.78 vs. 10.45 ± 2.25 ($p<0.0001$), respectively).

- Similarly, the mean duration of infective complications like duration of wound infection, pneumonia and septicaemia were significantly lower in the study group as compared to control group (7.80 ± 0.79 days, vs. 9.79 ± 2.23 ($p < 0.0001$); 6.25 ± 1.04 vs. 9.56 ± 2.87 ($p<0.0001$); and 6.44 ± 0.73 vs. 8.58 ± 1.51 ($p<0.0001$), respectively.
- The outcome of early enteral feeding after surgery for perforative peritonitis via naso-gastric tube was as good as naso-jejunal tube or feeding jejunostomy. So the simplest and least complicating of all the three i.e. naso-gastric is the best and most economical among the three.

Conclusion

CONCLUSION

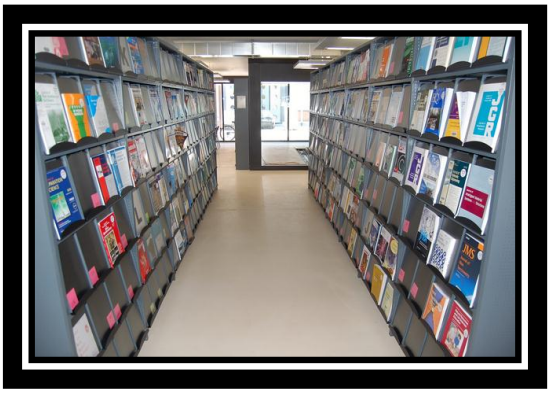
The analyses of the results indicate that even after generalised peritonitis the gastrointestinal tract recovers its tone and function within 48 hours. The gut perforation after repair remains secure, and is not put to any risk of leakage by enteral nutrition started within 24-48 hours after surgery. The already proven advantages of early enteral nutrition[3,4,12,16,18,29,36] after elective gastrointestinal surgery are clearly seen in patients with peritonitis due to gastrointestinal perforation as well. The study cases clearly did better as far as parameters such as weight, haemoglobin, serum albumin and complications like wound infection, wound dehiscence, anastomotic leak, pneumonia and death were concerned.

Unfortunately, in this study we have not been able to calculate the savings in terms of manpower and cost. The long-term results in the form of intestinal obstructions and incisional hernias are also not available. A study comparing enteral and parenteral feeds supplying the same amounts of nutrition would shed more light on the exact benefits of the enteral route, if any, over the parenteral route. There was no way of knowing how much of the weight loss was due to oedema fluid and how much was the lean body mass lost.

Though the incidence of major complications was reduced, the differences were not statistically significant, but there was statistically significant difference between the duration of these complications between the

two groups. This implies that the complications in enterally fed patients were controlled much more quickly than in conventionally managed patients. This suggests that the incidence of complications cannot be the parameter for the usefulness of enteral feeding in cases of emergency surgery for perforations of the gut, because the complications are inherent in the condition, e.g. wound infection or septicaemia in a case of faecal peritonitis.

Thus, early enteral nutrition in operated cases of gut perforations is very strongly recommend in view of decreased incidence and duration of infective complications and better nutritional status, leading to decreased duration of hospital stay and thus deceased cost of treatment and early return to work.



Bibliography

BIBLIOGRAPHY

1. Dempsey DT, Mullen JL, and Buzby GP. The link between nutritional status and clinical outcome: can nutritional intervention modify it? *Am J Clin Nutr* 1988;47:352–356.

2. Malhotra A, Mathur AK, Gupta S. Early enteral nutrition after surgical treatment of gut perforations: A prospective randomised study. *J Postgrad Med* 2004;50:102-6.

3. Vaithiswaran V, Srinivasan K, Kadambari D. Effect of early enteral feeding after upper gastrointestinal surgery. *Tropical Gastroenterology* 2008.29;2:91-94

4. Heslin MJ, Latkany L, Leung D, Brooks AD, Hochwald SN, Pisters PW, et al. A prospective, randomised trial of early enteral feeding after resection of upper GI malignancy. *Ann Surg* 1997;226(4):567-80.

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

6. Kaur N, Gupta MK, Minocha VR. Early enteral feeding by Nasoenteric tubes in patients with perforation peritonitis. *World J Surg* 2005 August;29(8):1023-1028.

7. Lewis SJ, Egger M, Sylvester PA. Early enteral feeding versus "nil by mouth" after gastrointestinal surgery: systemic review and meta-analysis of controlled trials. *BMJ* 2001;323:773-6.

8. Curtis CS, Kudsak KA. Enteral feeding in hospitalized patients: Early versus delayed enteral nutrition. *Pract Gastroenterol* 2009 Oct;XXXIII(10):22-30.

9. Sagar S, Harland P, Shields R. Early postoperative feeding with elemental diet. *Br Med J* 1979;1:293-5.

10. Bickel A, Shtamler B, Mizrahi S. Early oral feeding following removal of nasogastric tube in gastrointestinal operations. A randomized prospective study. *Arch Surg* 1992;127:287-9.

11. Beier-Holgersen R, Boesby S. Influence of postoperative enteral nutrition on postsurgical infections. *Gut* 1996;39:833-5.

12. Hochwald SN, Harrison LE, Heslin MJ, Burt ME, Brennan MF. Early postoperative enteral feeding improves whole body protein kinetics in upper gastrointestinal cancer patients. *Am J Surg* 1997;174:325-30.

13. Senkal M, Zumtobel V, Bauer KH, Marpe B, Wolfram G, Frei A, *et al*. Outcome and cost-effectiveness of perioperative enteral immunonutrition in patients undergoing elective upper gastrointestinal tract surgery: a prospective randomized study. *Arch Surg* 1999;134:1309-16.

14. Gianotti L, Braga M, Vignali A, Balzano G, Zerbi A, Bisagni P, *et al*. Effect of route of delivery and formulation of postoperative nutritional support in patients undergoing major operations for malignant neoplasms. *Arch Surg* 1997;132:1222-9; discussion 1229-30.

15. Braga M, Gianotti L, Vignali A, Cestari A, Bisagni P, Di Carlo V. Artificial nutrition after major abdominal surgery: impact of route of

administration and composition of the diet. *Crit Care Med* 1998;26:24-30.

16. Lewis SJ, Andersen HK, Thomas S. Early enteral nutrition within 24 h of intestinal surgery versus later commencement of feeding: a systematic review and meta-analysis. *J Gastrointest Surg* 2009;13:569-75.

17. Bozzetti F, Braga M, Gianotti L, Gavazzi C, Mariani L. Postoperative enteral versus parenteral nutrition in malnourished patients with gastrointestinal cancer: a randomised multicentre trial. *Lancet* 2001;358:1487-92.

18. Han-Geurts IJ, Hop WC, Kok NF, Lim A, Brouwer KJ, Jeekel J. Randomized clinical trial of the impact of early enteral feeding on postoperative ileus and recovery. *Br J Surg* 2007;94:555-61.

19. Kamei H, Hachisuka T, Nakao M, Takagi K. Quick recovery of serum diamine oxidase activity in patients undergoing total gastrectomy by oral enteral nutrition. *Am J Surg* 2005;189:38-43.

20. Jhobta RS, Attri AK, Kaushik R, Sharma R, Jhobta A. Spectrum of perforation peritonitis in India-review of 504 consecutive cases. *World J Emerg Surg.* 2006;1:26. doi:10.1186/1749-7922-1-26.

21. Gupta S, Kaushik R. Peritonitis- the Eastern experience. *World J Emerg Surg.* 2006;1:13. doi:10.1186/1749-7922-1-13.

22. Abbas SM, Upadhyay V. Hollow viscus injury in children: Starship Hospital experience. *World J Emerg Surg.* 2007;2:14. doi:10.1186/1749-7922-2-14.

23. Larson SD, Evers BM. Diseases of the peritoneum, retroperitoneum, mesentery, and omentum. In: Yamada T (editor), *Textbook of Gastroenterology*, 5th ed. Volume 2. Oxford, UK: Backwell Publishing; 2009: 2506-2507.

24. Debas HT. *Gastrointestinal Surgery Pathophysiology and Management*. New York: Springer; 2004:363-364.

25. Plank LD, Connolly AB, Hill G. Sequential changes in the metabolic response in severely septic patients during the first 23 days after the onset of peritonitis. *Ann Surg* 1998;228(2):146-148.

26. Hiyama DT, Bennion R. Peritonitis and Intraperitoneal Abscess. In: Zinner MJ, Schwartz SI, Ellis H (editors), *Maingot's Abdominal Operations*, 10th ed. Volume 1. London: Prentice Hall International; 1997:633-647.

27. McWhirter JP, Pennington CR. Incidence and recognition of malnutrition in hospital. *BMJ*. 1994 April 9; 308(6934): 945–948.

28. Gramlich L, Kichian K, Pinilla J, Rodych NJ, Dhaliwal R, Heyland DK. Does enteral nutrition compared to parenteral nutrition result in better outcomes in critically ill adult patients? A systemic review of literature. *Nutrition* 2004;20:843-8.

29. Andersen HK, Lewis SJ, Thomas S. Early enteral nutrition within 24h of colorectal surgery versus later commencement of feeding for postoperative complications. *Cochrane Database of Systemic Reviews* 2006, Issue 4. Art. No.: CD004080. doi:10.1002/14651858.CD004080.pub2.

30. Wittman DH, Walker AP, Condon RE. Peritonitis and intraabdominal infection. In: Schwartz S, Shires G, Spencer F, editors. *Principle of Surgery*, 6th ed. New York, NY: McGraw-Hill; 1991:1449-1483.

31. Dominioni L, Rovera F, Pericelli A, Imperatori A. The rationale of early enteral nutrition. *Acta Bio Medica* 2003;74; Suppl.2:41-44.

32. Kattelmann KK, Hise M, Russel M, Charney P, Stokes M, Compher C. Preliminary evidence for a medical Nutrition therapy protocol: Enteral feedings for critically ill patients. *J Am Diet Assoc* 2006;106(8):1226-1241.

33. Heyland *et al*. Early vs. Delayed nutrition intake. 2009 Jan 31st. [Internet] Available from: *www.criticalcarenutrition.com/docs/cpg/ 1.0envspn_07_FINAL.pdf*

34. MacFie J. Nutrition and fluid therapy. In: William NS, Bulstrode CJK, O'Connell PR (editors), *Bailey & Love's Short Practice of Surgery*, 25th ed. London: Hodder Arnold; 2008:229-230.

35. Tawa NE Jr, Fischer JE. Metabolism in surgical patients. In: Townsend CM Jr, Beauchamp RD, Evers BM, Mattox KL (editors), *Sabiston Textbook of Surgery*, 18th ed. Volume 1, Philadelphia: Saunders; 2008:143-190.

36. Silk DBA, Gow NM. Postoperative starvation after gastrointestinal surgery. *BMJ* 2001;323:761-2

37. Moore FA, Feliciano DV, Andrassy RJ, McArdle AH, Booth FV, Morgenstein-Wagner TB, Kellum JM Jr, Welling RE, Moore EE.

Early enteral feeding, compared with parenteral, reduces postoperative septic complications. *Ann Surg.* 1992 Aug;216(2):172-83.

38. Braunschweig CL, Levy P, Sheean PM, Wang X. Enteral compared with parenteral nutrition: a meta-analysis. *Am J Clin Nutr* 2001;74:534-42.

39. Marwah S, Godara R, Goyal R, Marwah N & Karwasra R. Early enteral nutrition following gastrointestinal anastomosis. *The Internet Journal of Gastroenterology* [Internet] 2008; Volume 7 Number 1. Available from: http://www.ispub.com:80/journal/the-internet-journal-of-gastroenterology/volume-7-number-1/early-enteral-nutrition-following-gastrointestinal-anastomosis.html

40. Suresh V. Feasibility and safety of early oral feeding after cervical esophagogastrostomy. *The Internet Journal Of Gastroenterology* [Internet] 2002; Volume1 Number 2. Available from: http://www.ispub.com:80/journal/the-internet-journal-of-gastroenterology/volume-1-number-2/feasibility-and-safety-of-early-oral-feeding-after-cervical-esophagogastrostomy.html

41. Ashrafi AN, Pochin R. 'Nil by mouth' post gastrointestinal surgery – is there any evidence? *N Z Med J* [Internet] 2006 October 13; Volume 119 Number 1243. Available from: http://www.nzma.org.nz/journal/119-1243/2271/

42. Keele AM, Bray MJ, Emery PW, Duncan HD, Silk DB. Two phase randomised controlled clinical trial of postoperative oral dietary supplements in surgical patients. *Gut* 1997;40:393-9.

43. Pupelis G, Selga G, Edmunds A, et al. Jejunal feeding, even when instituted late, improves outcomes in patients with severe pancreatitis and peritonitis. *Nutrition* 2001;17(2):91–94.

44. Furukawa S, Hideaki S, Ming-tsan L, et al. Enteral administration of glutamine in purulent peritonitis. *Nutrition* 1999;15(1):29–31.

45. 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.

46. Windsor AC, Kanwar S, Li AG, Barnes E, Guthrie JA, Spark JI, et al. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. *Gut* 1998;42:431-5.

47. Melis M, Fichera A, Ferguson MK. Bowel necrosis associated with early jejunal tube feeding. *Arch Surg* 2006;141:701-704.

Annexure – I

Informed Consent

ANNEXURE – I

SAMPLE INFORMED CONSENT FORM

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

TITLE OF THE PROJECT: Early enteral nutrition after surgical treatment of gut perforation: A prospective study.

PRINCIPAL INVESTEGATOR: Dr. Ashish Verma
Postgraduate student,
Department of General Surgery

PG GUIDE: Dr. Vijaya Patil M.S.,
Assoc. Professor of Surgery,
B.L.D.E. University's
Shri B.M. Patil Medical College &
Research Centre, Sholapur Road,
BIJAPUR - 586103

PURPOSE OF RESEARCH:

I have been informed that this study will analyse the effectiveness of early enteral feeding versus nil by mouth after surgical treatment of enteric perforation.

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

PROCEDURE:

I have been explained that depending upon the group allocated to me/my ward, I'll/my ward will either be given feeds through my naso-gastric tube after

24 hours of my surgery, or will be kept nil by mouth for 5 days; and that I'll/my ward will be subjected to certain routine blood and urine investigations, chest and erect abdomen x-rays and USG, if needed; and that my/my ward's weight will be measured on the first, fourth, seventh and tenth postoperative day and also on discharge.

RISKS AND DISCOMFORTS:

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

BENEFITS:

I understand that my/my wards participation in this study will help to analyse the effectiveness of early enteral feeding versus nil by mouth after surgical treatment of enteric perforation.

CONFIDENTIALITY:

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

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

REQUEST FOR MORE INFORMATION:

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

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

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

REFUSAL OR WITHDRAWL OF PARTICIPATION:

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

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

INJURY STATEMENT:

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

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

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

_____	_____	_____
Date	Dr. Vijaya Patil (Guide)	Dr. Ashish Verma (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

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

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

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

Annexure – II

Proforma

ANNEXURE II

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

Pts. Name _____ Age _____ Sex _____ I.P.No. _____

Diagnosis _____ DOA _____ Pt. Code _____

Surgery done _____ DOD _____

Intra-operative findings _____

_____

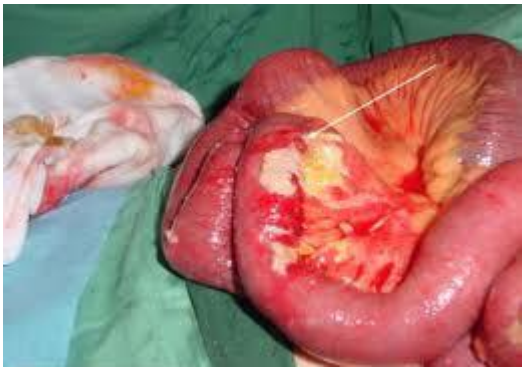
	PRE-OP	POD-1	POD-2	POD-3	POD-4	POD-5	POD-6	POD-7	POD-8	POD-9	POD-10
PULSE RATE (/min)											
BLOOD PRESS. (mm Hg)											
TEMPERATURE											
RESP. SYSTEM											
C.V.S.											
P/A -											
ABD. GIRTH (cm)											

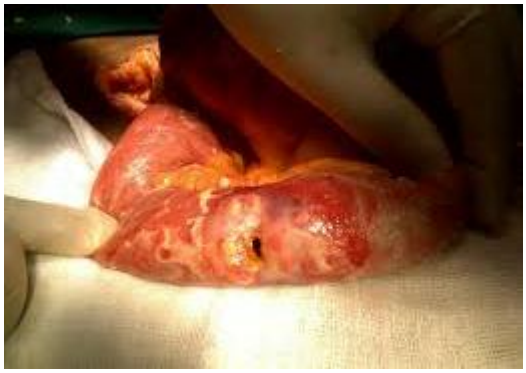
		PRE-OP	POD-1	POD-2	POD-3	POD-4	POD-5	POD-6	POD-7	POD-8	POD-9	POD-10
RT ASPIRATION (ml)												
INPUT	RT / ORAL (ml)											
	IVF (ml)											
OUTPUT (ml)												
DRAIN	PERF. SITE (ml)											
	PELVIC (ml)											
INCIS-ION SITE	LEAK											
	DEHISCENCE											
	INFECTION											
NAUSEA / VOMITING												
FLATUS / STOOLS												
WEIGHT (kg)												
HAEMOGLOBIN (gm/dl)												
W.B.C. COUNT (cells/mm_3)												
SERUM ALBUMIN (gm/dl)												
S. CREATININ (mg/dl)												
ANY OTHER COMPLAINTS												
REMARKS, IF ANY												

Annexure – III

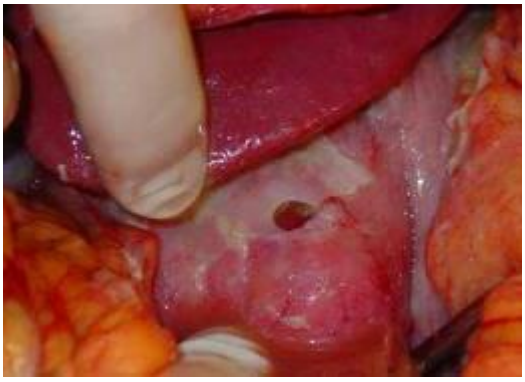
Color Plates

COLOUR PLATES

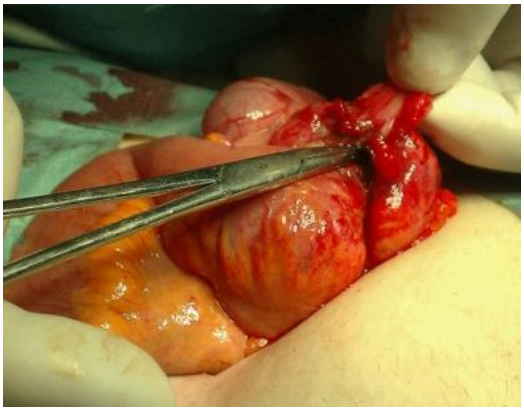




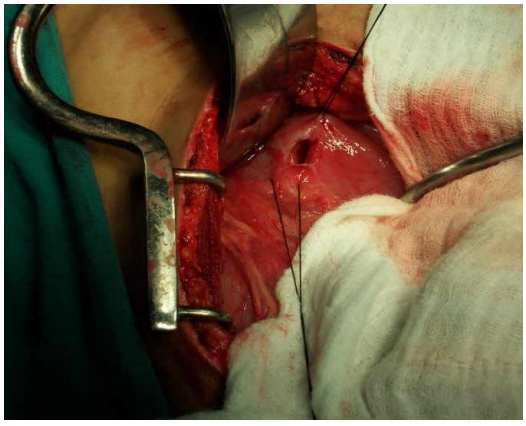
Jejunal Perforations



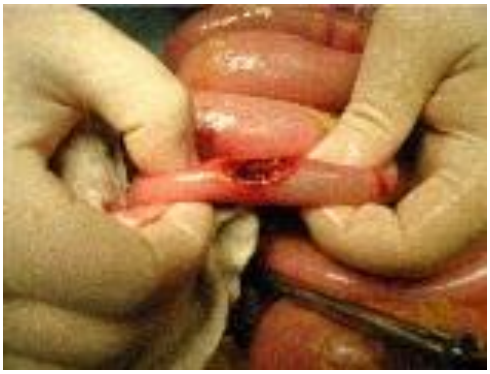
Duodenal Perforations



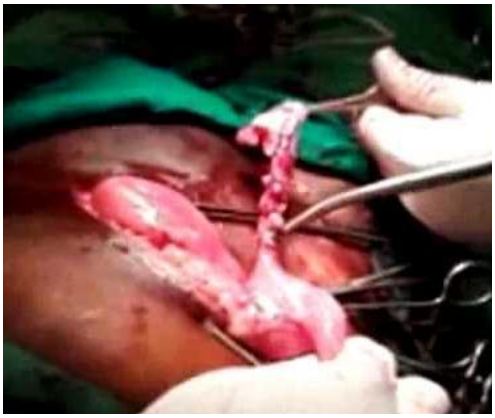
Caecal Perforation

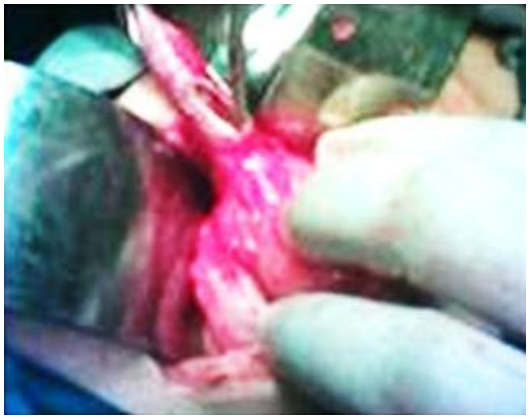


Gastric Perforation



Ileal Perforations





Appendicular perforations

Early Nasogastric feeding after surgery for perforation peritonitis

Annexure – IV

Key To Master Chart & Master chart

ANNEXURE IV

KEY TO MASTER CHART

Wt.0	Weight of patient on admission
Wt.4	Weight of Patient on Postoperative day 4
Wt.7	Weight of Patient on Postoperative day 7
Wt.10	Weight of Patient on Postoperative day 10
Hb.0	Hemoglobin of patient on admission
Hb.3	Hemoglobin of Patient on Postoperative day 3
Hb.8	Hemoglobin of Patient on Postoperative day 8
WBC.0	Total WBC count of patient on admission
WBC.3	Total WBC count of Patient on Postoperative day 3
WBC.8	Total WBC count of Patient on Postoperative day 8
Albu.0	Serum Albumin of patient on admission
Albu.3	Serum Albumin of Patient on Postoperative day 3
Albu.8	Serum Albumin of Patient on Postoperative day 8
Creat.0	Serum Creatinine of patient on admission
Creat.3	Serum Creatinine of Patient on Postoperative day 3
Creat.8	Serum Creatinine of Patient on Postoperative day 8

ANNEXURE IV

MASTER CHART

Sl. No.	Patients Name	Age (yrs)	Sex (M/F)	I.P. No	Pts. Code	Date of Admission	Date of Discharge	Total Stay (days)	Site of Perforation	Wt. 0 (kg)	Wt. 4 (kg)	Wt. 7 (kg)	Wt.10 (kg)	Hb. 0 (gm/dl)	Hb.3 (gm/dl)	Hb.8 (gm/dl)	WBC. 0 (cells/mm^3)	WBC. 3 (cells/mm^3)	WBC. 8 (cells/mm^3)	Albu.0 (gm/dl)	Albu.3 (gm/dl)	Albu.8 (gm/dl)	Creat.0 (mg/dl)	Creat.3 (mg/dl)	Creat.8 (mg/dl)	Ilius (days)	Fever	Abdominal Distension	Vomiting	Wound Complication	Pulmonary Complication	Septicaemia	Wound Infection (days)	Pneumonia (days)	ICU stay (days)	Septicaemia (days)	Death / AMA
1	Nagamma	45	M	###	A01	01-10-10	10-10-10	11	Appendix	65	63	63	64	11	9.4	11	15400	10200	8300	2.8	2.2	2.6	1.6	1	0.9	3	0	Yes	2	No	No	No					
2	Anita	30	F	###	B01	04-10-10	19-10-11	16	Appendix	52	51	48	47	11	9.8	8	16500	9300	13400	2.8	2.4	2	1.4	0.9	1	3	2	No	0	Infection	Pneumonia	No	10	8			
3	Ravi Kumar	35	M	###	A02	07-10-10	16-10-10	10	DU	67	66	65	66	14	12.4	14	15400	8200	6800	2.5	2.3	2.5	2.6	1.2	0.8	3	0	Yes	1	No	No	No					
4	Chandbai	58	F	###	B02	10-10-10	01-11-10	23	Ilial	86	84	82	81	12	9.2	9	18300	9100	15400	2.5	2.2	2	2	1.1	1.2	6	1	No	0	Dehisence	Pneumonia	Yes		15	11	10	
5	Sharanappa	52	M	###	A03	11-10-10	22-10-10	12	DU	80	78	78	79	12	10.4	12	16400	9200	12300	2.8	2.6	2.9	1.2	0.9	0.9	3	0	No	0	Infection	No	No	8				
6	Kallawwa	80	F	###	B03	04-11-10	09-11-10	16	Appendix	64	62	60	59	10	9.1	8.8	16400	8200	8600	2.3	2	2.1	1.4	1	0.9	3	0	No	0	No	No	No					
7	Mallappa	76	M	###	A04	04-11-10	13-11-10	10	DU	87	85	85	86	11	10.2	11	18200	10300	12200	2.6	2.4	2.5	3.2	2.1	0.9	3	1	No	0	Leak	Pneumonia	Yes		6	7	6	
8	Raju	35	M	###	B04	18-11-10	04-12-10	18	DU	53	51	52	53	10	9.4	8.2	####	10600	14800	2.9	2.5	1.5	3.1	1.6	1	6	0	No	0	Infection	No	Yes	10			9	
9	Ramesh	38	M	###	A05	23-11-10	01-12-10	10	Ilial	64	62	62	63	11	10.4	11	15300	9200	6800	2.4	2.2	2.4	2	1.2	0.9	3	0	No	0	No	No	No					
10	Ramappa	60	M	###	B05	25-11-10	11-12-10	18	DU	83	81	79	78	12	11	9.2	16300	8400	12500	2.7	2.4	2.2	1.5	1.1	1	6	0	Yes	2	Infection	No	No	12				
11	Basalingamma	58	M	###	A06	29-11-10	07-12-10	10	Appendix	83	81	81	82	10	10	10	14400	13200	8200	2.9	2.8	3	1.3	0.9	0.8	3	0	Yes	2	No	No	No					
12	Manohar	38	M	###	B06	02-12-10	22-12-10	21	DU	86	84	82	83	10	9.3	9.2	11600	8200	9400	3.4	2.8	2.6	1.3	0.9	0.9	4	0	No	0	Leak	No	No					
13	Gangadhar	14	M	###	A07	03-12-10	13-12-10	11	Traumatic	51	49	50	51	11	10.8	11	8200	7400	10800	3.1	3	3.1	0.9	0.9	0.9	3	2	No	0	Infection	Pneumonia	No	9	8			
14	Arjun	45	M	###	B07	04-12-10	20-12-10	17	Gastric	90	88	88	87	15	11.5	10	10400	8200	12400	1.9	1.7	1.5	1.4	0.9	1	3	0	No	0	Infection	No	No	13				
15	Siddram	30	M	###	A08	05-12-10	15-12-10	11	Gastric	74	72	72	73	16	12.3	13	16500	8400	7200	3.3	3.1	3.2	1.1	0.9	0.9	3	0	No	0	No	No	No					
16	Hanumanth	65	M	###	B08	07-12-10	26-12-10	20	DU	86	84	83	82	16	12.9	12	6700	5200	8400	2.5	2.2	2	1.4	1.1	0.9	3	0	No	0	Leak	No	No					
17	Mallappa	58	M	###	A09	16-12-10	28-12-10	13	DU	85	83	83	84	11	10.8	11	18400	10400	12500	3	2.8	2.9	2.8	1.6	1.1	3	3	No	0	Infection	Pneumonia	Yes	8	7	6	7	
18	Lalitha	46	F	###	B09	30-12-10	12-01-11	14	DU	104	100	97	97	10	9.4	9.2	13400	10000	12000	2.8	2.4	2	1.8	1.1	1	5	0	No	0	Infection	No	No	8				
19	Irappa	35	M	179	A10	03-01-11	13-01-11	11	Appendix	84	83	83	83	11	11	11	18800	8400	6400	2.9	2.7	2.9	2.9	1.2	0.9	2	0	No	0	No	No	Yes			5	7	
20	Alisab	85	M	235	B10	04-01-11	15-01-11	12	Caecum	74	72	70		9.8	9		13400	12300		2.5	2.2		2	1.1		5	3	Yes	3			Yes					Died
21	Saraswati	45	F	228	A11	06-01-11	16-01-11	11	Appendix	65	63	63	64	9.2	8.8	9	14800	8200	12800	2.6	2.4	2.7	1	0.9	0.9	2	1	No	0	Dehisence	Pneumonia	No		5			
22	Ramanna	70	M	884	B11	13-01-11	28-01-11	16	Caecum	65	63	62	62	12	10.2	9	19300	10400	11400	2.6	2	1.6	2.1	1.5	1.1	4	3	No	0	Infection	Pneumonia	Yes	10	8	9	8	
23	Sharanawwa	39	M	1348	A12	20-01-11	31-01-11	12	Appendix	84	83	83	84	11	11	11	15400	10200	8200	2.9	2.7	2.9	1	0.9	0.9	3	0	No	0	No	No	No					
24	Vivek	36	M	1810	B12	27-01-11	09-02-11	14	DU	85	83	82	82	11	10.5	10	16400	10800	8400	3.5	2.8	2.5	1.3	0.9	0.9	3	0	Yes	1	No	No	No					
25	Medar	45	M	2276	A13	06-02-11	18-02-11	13	Gastric	86	84	84	85	11	10	11	10600	7500	8100	2.9	2.6	2.8	1.2	0.9	0.9	2	1	No	0	Leak	No	No					
26	Dayswappa	63	M	2720	B13	08-02-11	23-02-201	22	DU	89	87	86	87	13	10.2	11	10000	6400	12600	3.4	2.8	2.4	1.6	1.1	1	3	2	No	0	Dehisence	Pneumonia	No		9			
27	Nagappa	35	M	3060	A14	12-02-11	23-02-11	12	DU	78	76	76	77	16	14.2	15	18400	6800	6000	2.2	2	2.2	2.3	1.2	0.9	3	0	Yes	1	No	No	No					
28	Mahadev	45	M	2276	B14	08-02-11	28-02-11	21	Gastric	83	81	80	81	12	10.4	9	12300	8400	6800	2.4	2.2	2.3	1.3	1	0.9	3	0	No	0	Leak	No	No					

28	Mahadev	45	M	2276	B14	08-02-11	28-02-11	21	Gastric	83	81	80	81	12	10.4	9	12300	8400	6800	2.4	2.2	2.3	1.3	1	0.9	3	0	No	0	Leak	No	No					
29	Roopa	22	F	4063	A15	26-02-11	06-03-11	10	Appendix	36	34	34	35	6.4	9.1	11	9200	8200	11200	2.9	3	3.1	1	0.9	0.8	3	1	No	0	Infection	No	No	7				
30	Chandrashekhar	22	M	4758	B15	08-03-11	19-03-11	12	Traumatic	68	66			8.6	8		12300	6400		2.2	1.9		1.3	1		4	3	No	3			No					AMA
31	Mohan	35	M	5509	A16	17-03-11	30-03-11	14	DU	66	64	64		12	11.6		18200	8400		2.9	2.5		0.9	0.8		3	0	No	2			Yes					Died
32	Hiramma	40	M	5899	B16	22-03-11	10-04-11	17	Ilial	90	88	86	86	10	9.6	9.1	19400	11500	12800	2.8	2.3	2.1	2.1	1.2	1	5	1	No	0	Infection	Pneumonia	Yes	11	10	8	7	
33	Vijayashree	30	F	6020	A17	24-03-11	05-04-11	12	DU	88	86	86	87	12	11.2	12	12800	10400	7100	2.4	2.1	2.2	1.2	1	0.9	3	0	Yes	0	No	No	No					
34	Keshu	43	M	6034	B17	24-03-11	16-04-11	24	Appendix	89	87	86	87	16	12.3	11	16800	13700	10200	2.6	2	1.8	1.7	1.1	0.9	4	1	Yes	0	Dehiscence	Pneumonia	No		7			
35	Dyamongoud	45	M	6008	A18	24-03-11	06-04-11	13	DU	64	62	62	63	13	12	12	15800	4300	4200	2.6	2.2	2.5	1.7	1.1	0.8	3	0	No	0	No	No	No					
36	Muttawwa	20	F	6179	B18	26-03-11	08-04-11	14	DU	54	52	50	51	11	10.4	9.6	15200	9400	10800	2.7	2	1.8	1.3	1	0.9	5	2	No	0	Infection	Pneumonia	No	6	5			
37	Pandu	24	M	6672	A19	24-04-11	14-04-11	13	Ilial	64	62	61	62	12	11.4	12	16400	8200	6200	2.9	2.5	2.8	1.4	1	0.8	2	0	No	0	No	No	No					
38	Revansiddappa	11	M	6748	B19	04-04-11	25-04-11	22	Appendix	27	26	25	26	11	10	9.1	11200	8400	9000	2.4	1.9	1.6	1.9	1.1	0.9	3	1	No	0	Leak	No	Yes			7	6	
39	Gangabai	45	F	6937	A20	06-04-11	16-04-11	11	Caecum	89	87	87	88	10	9.9	10	14500	10800	12400	2.8	2.6	2.9	1.2	0.9	0.9	2	1	No	0	Infection	No	No	9				
40	Bhirappa	50	M	6964	B20	07-04-11	24-04-11	18	DU	138	136	135	134	13	10.5	8.6	18500	10200	10400	1.9	1.8	1.9	2.3	1.7	1.2	4	3	No	0	Infection	Pneumonia	Yes	10	15	12	8	
41	Mahadevi	62	F	7066	A21	08-04-11	18-04-11	11	DU	94	92	91	92	12	11.6	12	14500	9200	5400	3	2.8	2.1	1.8	1.1	0.8	2	3	No	0	No	No	No					
42	Chetan	13	M	7133	B21	09-04-11	29-04-11	21	Appendix	36	35	34	34	16	12.3	13	16500	8400	9200	3.3	3.1	3.2	1.1	0.9	0.9	3	0	No	0	Leak	No	No					
43	Sharanappa	20	M	7384	A22	12-04-11	22-04-11	10	Ilial	51	49	48	49	13	12	12	15800	4300	4200	2.6	2.2	2.5	2.1	1.2	0.8	3	0	Yes	1	No	No	No					
44	Sachin	12	M	7982	B22	21-04-11	01-05-11	11	Appendix	33	32	31	31	13	10.1	11	15200	8300	7100	2.6	2.4	2.7	1.3	0.9	0.9	3	1	No	0	No	No	No					
45	Suresh	45	M	8467	A23	28-04-11	10-05-11	13	DU	74	72	71	72	16	12.3	13	16500	8400	7200	3.3	3.1	3.2	1.1	0.9	0.9	3	0	No	0	No	No	No					
46	Sonu	25	M	9516	B23	12-05-11	26-05-11	14	DU	68	66	65	66	14	12.9	12	6700	5200	8400	2.5	2.2	2	1.9	1.1	0.9	3	0	Yes	0	No	No	No					
47	Hanumantage	40	M	2805	A24	17-05-11	26-05-11	10	DU	85	83	83	84	11	10.8	11	10400	6400	13400	3	2.8	2.9	1.5	1	0.9	3	2	No	0	Infection	Pneumonia	No	7	6			
48	Mallappa	60	M	###	B24	23-05-11	06-06-11	15	DU	98	96	94	93	10	9.4	9.2	13400	10000	12000	2.8	2.4	2	1.4	1.1	1	5	0	No	0	Infection	No	No	8				
49	Basamma	40	M	###	A25	26-05-11	06-06-11	11	DU	86	84	84	85	11	11	11	18800	8400	12400	2.9	2.7	2.9	2.5	1.3	1	2	3	No	0	Dehiscence	Pneumonia	Yes		5	6	6	
50	Shantawwa	41	F	###	B25	01-06-11	13-06-11	13	DU	74	72	70	71	9.8	9	8.8	13400	8300	6800	2.5	2.2	2	1.6	1.1	0.9	3	0	No	0	No	No	No					
51	Jakkawwa	64	F	###	A26	01-06-11	11-06-11	11	Appendix	65	63	63	64	9.2	8.8	9	14800	8200	6800	2.6	2.4	2.7	1	0.9	0.9	2	0	Yes	0	No	No	No					
52	Laxmibai	60	F	11048	B26	02-06-11	18-06-11	16	DU	65	63	62	62	9.4	9.1	9	19300	10400	11400	2.9	2.3	1.6	2.1	1.5	1.2	4	4	No	0	Infection	Pneumonia	Yes	6	9	8	8	
53	Drstagirisab	65	M	11107	A27	02-06-11	13-06-11	12	Appendix	85	83	83	84	11	11	11	10400	8200	6200	2.9	2.7	2.9	1	0.9	0.9	3	0	No	0	No	No	No					
54	Merasab	60	M	###	B27	15-06-11	29-06-11	15	Gastric	80	78	76	75	11	10.5	10	16400	10800	8400	3.1	2.8	2.5	1.3	0.9	0.9	3	0	Yes	1	No	No	No					
55	Machandra	58	M	12761	A28	23-06-11	04-07-11	12	DU	89	87	87	88	13	10.1	11	15200	8300	7100	2.6	2.4	2.7	1.3	0.9	0.9	3	0	No	0	No	No	No					
56	Lalsab Ali	28	M	###	B28	05-07-11	21-07-11	17	Ilial	78	76	75	74	13	10.2	11	10000	6400	8600	3.4	2.8	2.4	1.6	1.2	1	3	1	No	0	Leak	Pneumonia	No		12			
57	Ogeppa Pujari	28	M	###	A29	17-07-11	28-07-11	12	DU	82	80	79	80	16	14.2	15	18400	6800	11800	2.2	2	2.2	1.4	1	0.9	3	1	No	0	Infection	No	No	7				
58	Ramchandra	65	M	15431	B29	26-07-11	09-08-11	14	DU	78	76	74	75	13	10.9	9	12300	8400	6800	2.4	2.2	2.3	1.3	1	0.9	3	0	No	0	No	No	No					
59	Siddappa	30	M	###	A30	29-07-11	09-08-11	12	DU	68	66	66	67	6.4	9.1	11	19200	8200	6200	2.9	3	3.1	1.8	1.1	0.8	3	0	Yes	2	No	No	No					
60	Ningaraj	16	M	16241	B30	06-08-11	18-08-11	13	Appendix	41	40	39	40	11	9.2	8.1	12300	6400	11600	2.2	1.9	1.8	1.3	1	0.9	4	2	No	0	Infection	Pneumonia	No	7	6			
61	Shreeshail	35	M	###	A31	11-08-11	20-08-11	10	Gastric	90	88	88	89	12	11.6	13	18200	8400	6400	2.9	2.5	2.8	3.1	1.5	1	3	0	No	0	No	No	Yes			5	7	
62	Sharnappa	40	M	16814	B31	13-08-11	27-08-11	15	Ilial	76	73	72	71	10	9.6	9.1	11400	6500	10800	2.8	2.3	1.6	1.2	1	0.9	5	3	No	0	Infection	Pneumonia	No	11	9			

63	Mallappa	23	M	16928	A32	14-08-11	23-08-11	10	DU	64	63	62	62	12	11.2	11.6	12800	10400	7100	2.4	2.1	1.4	1.2	0.9	0.9	3	0	Na	0	Na	Na	Na					
64	Nadeem	16	M	16948	B32	15-08-11	27-08-11	13	Traumatic	46	45	44	44	16	12.3	11	16800	13700	10200	2.3	2	1.8	1.4	1.1	0.9	4	0	Na	0	Na	Na	Na					
65	Mahadev	60	M	18026	A33	27-08-11	06-09-11	11	DU	64	62	62	63	13	12	12.3	15800	4300	10200	2.6	2.2	2.5	1	0.9	0.8	3	0	Na	0	Infectian	Na	Na	8				
66	Yashwant	74	M	18951	B33	12-09-11	30-09-11	19	DU	66	64			11	10.4		15200	12400		2.3	2		1.3	1		5	2	Yes	0		Pneumania	Yes					Died
67	Laxmibai	70	F	19170	A34	13-09-11	22-09-11	10	DU	66	63	63	64	12	11.4	12	16400	8200	6200	2.9	2.5	2.8	1.8	1	0.8	2	0	Na	0	Na	Na	Na					
68	Irappa	50	M	19383	B34	14-09-11	04-10-11	21	Gastric	68	66	66	67	11	9.1	9.1	17200	8400	9000	2.4	1.9	1.6	3.1	2.3	1.5	3	1	Na	0	Leak	Na	Yes			12	10	
69	Anuruyya	22	F	19444	A35	15-09-11	24-09-11	10	Appendix	48	47	47	48	10	9.9	10	14500	10800	8400	2.8	2.6	2.9	1.2	0.9	0.9	2	0	Na	0	Na	Na	Na					
70	ShreeshailHadpa	40	M	***	B35	30-09-11	17-10-11	18	DU	95	93	92	92	10	8.4	8.6	18500	10200	10400	2.8	2.3	1.9	2.3	1.7	1.2	4	3	Na	0	Infectian	Pneumania	Yes	12	11	14	10	
71	AbiKumar	14	M	21456	A36	10-10-11	19-10-11	9	Traumatic	35	34	34	35	13	12	12.3	5800	4300	4200	2.6	2.2	2.5	1	0.9	0.8	3	0	Yes	1	Na	Na	Na					
72	Abdul	45	M	21488	B36	11-10-11	25-10-11	14	DU	70	67	65	65	11	10.5	8	16500	9300	16400	2.2	1.9	2	1.4	0.9	1	3	2	Na	0	Infectian	Pneumania	Na	8	6			
73	Shivayaqappa	50	M	23315	A37	04-11-11	13-11-11	10	Gastric	67	65	65	66	14	12.4	13.8	15400	8200	6800	2.5	2.3	2.5	1.3	0.9	0.8	3	0	Na	0	Na	Na	Na					
74	Jiyat Singh	38	M	***	B37	11-11-11	29-11-11	19	Gastric	86	84	82	81	12	9.2	9	18300	9100	15400	2.5	2.2	2	2	1.1	1.2	6	3	Na	0	Dehirence	Pneumania	Yes		12	11	9	
75	Bhimappa	70	M	24311	A38	17-11-11	26-11-11	10	Gastric	79	77	77	78	12	10.4	11.6	13400	9200	11300	2.8	2.6	2.9	1.2	0.9	0.9	3	0	Na	0	Infectian	Na	Na	7				
76	Nagat Singh	60	M	***	B38	19-11-11	05-11-11	17	DU	74	72	70	71	10	9.1	8.8	16400	8200	12600	2.3	2	2	1.4	0.9	1	3	1	Yes	0	Infectian	na	Na	11				
77	Gangabai	51	F	***	A39	24-11-11	03-12-11	10	DU	87	85	85	86	11	10.2	11	18200	10300	9200	2.6	2.4	2.5	3.4	2.6	1.2	3	1	Yes	2	Leak	Na	Yes			5	7	
78	Rovappa	45	M	***	B39	24-11-11	11-12-11	18	DU	73	71	70	71	10	8.6	8.2	20400	10600	14800	2.9	2.4	1.5	3.1	2	1.2	6	0	Na	0	Infectian	Na	Yes	12		13	11	
79	Sharnappa	45	M	***	A40	28-11-11	08-12-11	10	Illial	64	62	62	63	11	10.4	11	12300	9200	6800	2.4	2.2	2.4	1.5	0.9	0.9	3	0	Na	0	Na	Na	Na					
80	Bhimappa	62	M	***	B40	03-12-11	17-12-11	15	DU	81	80	79	78	9.9	9.4		16300	8400	12500	2.7	2.4	2.2	1.5	1.1	1	6	2	Yes	3		Pneumania	Yes					Died
81	Hanamappa	30	M	***	A41	18-12-11	26-12-11	9	Traumatic	83	81	81	82	10	10	10.1	18400	13200	8200	2.9	2.8	3	2.8	1.4	1	3	0	Na	0	Na	Na	Yes			7	6	
82	Irhuar	52	M	27512	B41	26-12-11	08-01-12	14	DU	86	84	82	83	10	9.3	9.2	11600	8200	6400	3.2	2.8	2.6	1.3	0.9	0.9	4	0	Yes	1	Na	Na	Na					
83	Mallikarjun	23	M	27813	A42	30-12-11	08-01-12	10	DU	50	48	48	49	11	10.8	11	18200	7400	6800	3.1	3	3.1	0.9	0.9	0.9	3	0	Yes	1	Na	Na	Na					
84	Shabbir	48	M	880	B42	09-01-12	24-01-12	16	Traumatic	90	88			15	11.5		10400	8200		2.8	2.6		0.9	0.9	0	5					Na					AMA
85	Bauramma	20	F	2181	A43	26-01-12	04-02-12	9	Appendix	74	72	72	73	16	12.3	12.5	16500	8400	7200	3.3	3.1	3.2	1.1	0.9	0.9	3	0	Na	0	Na	Na	Na					
86	Umabai	18	F	2331	B43	29-01-12	11-02-12	14	Appendix	46	44	43	43	14	12.9	12.1	6700	5200	8400	2.5	2.2	2	1.6	1.1	0.9	3	0	Na	0	Na	Na	Na					
87	Hanumantraya	40	M	2557	A44	01-02-12	10-02-12	10	DU	85	83	83	84	11	10.8	11.1	20400	11400	12600	3	2.8	2.9	3	1.9	1.2	3	2	Na	0	Infectian	Pneumania	Yes	8	6	6	5	
88	Jattoppa	24	M	3121	B44	07-02-12	25-02-12	19	Illial	78	76	74	73	10	9.4	9.2	13400	10000	12000	2.8	2.4	2	1.6	1.1	1	5	0	Na	0	Infectian	Na	Na	13				
89	Sharnauua	30	F	3525	A45	13-02-12	23-02-12	11	Illial	85	83	82	83	11	11	11.2	12800	8400	6400	2.9	2.7	2.9	1.2	0.9	0.9	2	0	Na	0	Na	Na	Na					
90	Gangabai	65	F	4552	B45	24-02-12	19-03-12	25	DU	84	82	81	81	11	10.8	11.1	13400	10400	11300	3.1	2.8	2.9	1.5	1	0.9	3	0	Na	0	Dehirence	Pneumania	Na		8			
91	Neelappa	60	M	4598	A46	25-02-12	07-03-12	12	DU	104	100	97	97	10	9.4	9.2	13400	10000	8200	2.8	2.4	2	1.4	1	0.9	5	0	Na	0	Na	Na	Na					
92	Sabu	50	M	5468	B46	08-03-12	24-03-12	17	Illial	86	84	82	82	11	11	11.2	18800	8400	6400	3.2	2.7	2.9	2.6	1.2	1	2	0	Na	0	Na	Pneumania	Yes		12	10	7	
93	Yallappa	30	M	7483	A47	03-04-12	12-04-12	10	DU	86	84	84	85	10	9.3	9.2	11600	8200	13400	3	2.8	2.6	1.3	0.9	0.9	4	1	Na	0	Infectian	Pneumania	Na		7			
94	Karhibai	50	F	7647	B47	06-04-12	19-04-12	14	Appendix	74	72	71		9.8	9	8.8	19400	8300	6800	2.5	2.2	2	2.6	1.5	1.2	3	2	Yes	1	Na	Pneumania	Yes					Died
95	Mahadevi	12	F	8249	A48	14-04-12	22-04-12	9	Appendix	36	35	35	36	9.2	8.8	9	14800	8200	6800	2.6	2.4	2.7	1.8	0.9	0.9	2	0	Yes	2	Na	Na	Na					
96	Kalavati	38	F	8455	B48	17-04-12	02-05-12	16	DU	65	63	62	62	12	10.6	9	19300	10400	11400	2.8	2.4	1.6	1.3	1	0.9	4	3	Yes	0	Infectian	Pneumania	Na	8	10			
97	Laxman	56	M	8773	A49	21-04-12	30-04-12	10	Appendix	84	83	83	84	11	11	11.2	20400	8200	6200	2.9	2.7	2.9	2.6	1.1	0.9	3	0	Na	0	Na	Na	Yes			6	7	
98	Shankarappa	70	M	9279	B49	28-04-12	13-05-12	16	Gastric	85	83	81	80	11	10.5	10.3	16400	10800	8400	3.5	2.8	2.5	1.3	0.9	0.9	3	0	Na	0	Na	Na	Na					
99	Durlappa	45	M	9594	A50	02-05-12	11-05-12	10	DU	88	86	86	87	13	10.1	11.2	15200	8300	7100	2.6	2.4	2.7	1.3	0.9	0.9	3	0	Na	0	Leak	Na	Na					
100	Gurubarappa	70	M	10702	B50	14-05-12	31-05-12	18	Illial	78	76			13	10.2		10000	6400		3	2.8		1.6	1.1		3	2	Yes	0	Na	Pneumania	Na					Died
101	Surendra	24	M	11632	A51	16-05-12	26-05-12	10	DU	82	82	82	83	16	14.2	15.1	18400	6800	6000	2.2	2	2.2	2.3	1.2	0.9	3	0	Na	0	Na	Na	Na					
102	Shantibai	56	F	12856	B51	19-05-12	03-06-12	15	Appendix	49	47	47	48	9.2	8.8	9	12300	8400	6800	2.4	2.2	2.3	1.3	1	0.9	3	0	Na	0	Na	Na	Na					
103	Mallappa	76	M	13971	A52	21-05-12	31-05-12	10	Appendix	65	66	67	68	6.4	9.1	10.6	9200	8200	6200	2.9	3	3.1	1	0.9	0.8	3	0	Na	0	Na	Na	Na					