# “COMPARITIVE STUDY OF DELAYED PRIMARY CLOSURE VERSUS PRIMARY CLOSURE OF SKIN IN CONTAMINATED AND DIRTY ABDOMINAL WOUNDS/ INCISION”


**By**

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## MASTER OF SURGERY

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**Under the guidance of**

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**DR.JADESH BHADRAGOUDRA**

## ABBRIVATIONS

| | |
|---|---|
| PC | PRIMARY CLOSURE |
| DPC | DELAYED PRIMARY CLOSURE |
| SSI | SURGICAL SITE INFECTION |
| P | PRESENT |
| AB | ABSENT |
| POS | POST OPERATIVE STAY |
| DOC | DATE OF CLOSURE |
| IP | ILEAL PERFORATION |
| AP P | APPENDICULAR PERFORATION |
| APC | APPENDICECTOMY |
| GHP | GRAHAM PATCH |
| GB | GANGRENOUS BOWEL |
| RA | RESECTI0N ANASTOMOSIS |
| DP | DUODENAL PERFORATION |
| MI | MESENTERIC INJURY |
| MVL | MESENTERIC VESSEL LIGATION |
| PP P | PREPYLORIC PERFORATION |
| JP | JEJUNAL PERFORATION |
| GB P | GALLBLADDER PERFORATON |
| Ch | CHOLECYSTECTOMY |
| RCT | RANDOMIZED CLINICAL TRIALS |

## ABSTRACT

**Background**:

It is still a matter of debate whether delayed primary closure (DPC) of contaminated abdominal incisional reduces surgical site infection compared with primary closure (PC ).The rate of wound infection for dirty abdominal wound is approximately 40%,but the optimal method of wound closure remains controversial.

**Aims and objectives**:

To determine whether delayed primary skin closure of contaminated and dirty abdominal wounds reduces the rate of surgical site infection (SSI) compared with primary skin closure.

**Method**:

Patient diagnosed as acute peritonitis and posted for exploratory laparotomy during the period of October 1 2013 to September 1 2015 were included. The study was conducted at Shri B M Patil Medical college and Hospital, Vijayapur. In this series a total of 100 patients were included and were divided in two group .Each group had 50 patients.For primary closure group, wounds were closed with monofilament interrupted suture .For Delayed primary closure, skin and subcutaneous tissue are left open and packed with 10 %( betadine) povidone iodine soaked gauge, which was changed daily to prevent excessive collection of exudates. The outcome of wound was assessed on post – operative days.

**Result:**

In this entire series, wound infection developed after incision closure was 33% .The primary group had a higher rate of wound infection 54% and delayed primary

closure was 12%,(P< 0.OOO) and longer length of hospital stay 19.4days in primary closure group and 16.5days in delayed primary closure group, (P value 0.002).

**Conclusion**:

Laparotomy wound complications are multifactorial, it depends on many factors.A strategy of DPC of dirty abdominal wound ,clinically appears to decrease the rate of wound infection, when compared with PC without increasing the length hospital of stay.

**Key word:** Primary closure, surgical site infection, delayed primary closure.

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# INTRODUCTION

Surgical site infections are common following the abdominal surgeries. Centre for disease control has found 45% SSI incidence in abdominal surgeries with contaminated wounds. SSI causes morbidity with additional risk of mortality and also impact on health resources and cost through increased hospital stay, repeated surgeries, nursing care cost and drug treatment. Despite of major improvement in antibiotics, better anesthesia, superior instruments, early diagnosis of surgical problems and better post-operative care but still surgical site infection (SSI) do occur.

The occurrence of SSI , wound dehiscence , incisional hernia are common following primary closure of skin in dirty / contaminated wounds. Disadvantage of primary closure is increases the length of hospital stay and thereby increase in the cost. By delaying the closure of skin in contaminated wounds, and we can reduce SSI. It has better prognosis compared to primary closure. Advantage: there is no specialized equipment required, easy procedure, it allows the soft tissue to drain, it reduces the no. of colonic bacteria, and particularly anaerobes in contaminated wounds. Thus it would be helpful to reduce SSI.

## AIMS AND OBJECTIVES OF THE STUDY

To determine whether delayed primary skin closure of contaminated and dirty abdominal incisions reduces the rate of surgical site infection (SSI) compared with primary skin closure.

# REVIEW OF LITERATURE

In June 26 2013, Aneel Bhangu, Prashant Singh, Jonathan Lundy, Douglas M Bowle et al " Systemic Review and Meta-Analysis of Randomized clinical trials comparing Primary vs Delayed Primary skin closure in contaminated and dirty abdominal incisions." They were included 8 studies randomizing 623 patients with contaminated or dirty abdominal wounds to either DPC or PC .The most common diagnosis was appendicitis i.e. 77.4%, followed by perforated abdominal viscous (11.5%), ileostomy closure 6.5%, trauma 2.5% , intra-abdominal abscess/ other peritonitis 1.9%. The time to first review for DPC was provided between 2 and 5 days post operatively. SSI was assessed across all studies, DPC significantly reduces the chances of SSI (odd ratio 0.65:95%CI, 0.40-0.93: p=0.02) . However heterogeneity was high (72%) and using random effect model, the effect was no longer significant. (Odd ratio, 0.65:95%CI, 0.25-1.64, p=0.36). They concluded that DPC reduce the rate SSI.

In December 15 2011, Ruey-a chiang, Shan Long Chen, Yoa-Chung Tsai "Delayed primary closure versus primary closure for wound management in perforated appendicitis. A prospective RCT" From department of surgery , MacKay Memorial Hospital Taitung Branch Taitung Taiwan , total of 70 patients perforated appendicitis were included .The main outcome measure were incidence of wound infection and length of hospital stay. Wound infection developed after incision closure in 21.4%.of the patients. Primary closure group had a incidence of wound infection 38.9 % . Versus delayed primary closure 2.9%. p <0.001. And longer length of hospital stay 8.4days v/s 6.3days p=0.038. They concluded that Delayed Primary closure is the optimal

management strategy for perforated appendicitis wounds and reduces the wound infection rate length of stay

In August 4, 2001. Stephen M.Cohn. Giovanni Giannottia et all From the Division Of Trauma and Surgical Critical Care and Colorectal Surgery, Department Surgery, University Miami School of Medicine Miami, Florida. They conducted “R C T of Two Wound Management Strategies for Dirty Abdominal Wounds”. They included 51 patients with dirty abdominal wounds related to- perforated appendix other perforated viscus. Traumatic injury more than 4 hours old, Intra-abdominal abscess concluded that the wound infection rate was greater in primary closure group than in the delayed primary closure wounds. The length of hospital stay was similar in two groups.

In 2009 April , Duttaroy DD , Jitendra J ,Duttaroy B, Bansal U, Dameja P, Patel G, et al from Department o of Surgery , Government Medical College and Sir Sayajirao Hospital Baroda , Gujarat, study of “ Management Strategy For Dirty Abdominal Incisions: Primary or Delayed Primary Closure? A Randomized trial” They concluded that SSI developed after incision closure in 23% patients. Infections were significantly common in primary group (42.5%) vs 2.5% for DPC. p=0.0000375.Abdominal dehiscence in primary group 10 (25%) in DPC 1 (2.7%).

# HISTORICAL REVIEW

"Those who cannot remember the past are condemned to repeat it." George Totayana. 1992.[3]

Early in the history of man there was recognition of the interplay between wound, infections and surgical manipulation. In fact, virtually all wounds became infected and infection was associated with high mortality. Treatment was based on trial and error and individual physician experience, yet many forms of effective therapy that varied for different cultures were discovered.

The early Egyptian recognized some form of circulation of the blood based on the doctoring of wounds. In addition, some primitive remedies such as use of the pulverized malachite or honey in wounds may have been extremely effective, as noted by the modern day experiments of Manjo. Sushrutha, 'Father of Indian' surgery had also emphasized on the prevention of wound infections in his ancient scripts. The Greek and Romans employed a variety of remedies that included use of red wine, poultices of the herbs and other compounds that may have had anti-bacterial properties. They also were proponents of laudable pus, since infection was so common that it was considered the norm after wounding.[3]

There have been two phases if intense revolutionary development in the means employed by the surgeons against infections. The first of these two phases was centered on discovery of causes of infections and methods of its prevention. The great names associated with this phase are those of the Fathers of the Bacteriology, such as Pasteur,

Robert Koch and Joseph Lister. Second phase, was that of effective systemic treatment of the same. This phase is associated with great names of Domagk and Florey.

The development of bacteriology as a discipline dates from the time of Louis Pasteur(1822-95). He introduced techniques of sterilization that resulted in the development of steam sterilizer, hot air oven and autoclave. He also established the differing growth needs of different bacteria.[4]

Robert Koch (1843-1910) in Germany, perfected bacteriological techniques during his studies on the culture and characters of the anthrax bacillus(1876). He introduced staining techniques and methods of obtaining bacteria in pure culture using solid media. [4]

Lord Lister (1827-1912). The Father of Antiseptic surgery revolutionized the science of surgery by introducing the antiseptic and aseptic surgical techniques in operative and post operative cases. He chanced upon the antiseptic properties of carbolic acid, which had already been strongly recommended by Francois Jules, Lamaire(1860) for treatment of surgical sepsis. Lister first employed carbolic acid dressings, with tremendous success in dealing with compound fractures. He then crystallized his work and presented them in his renowned paper on "The antiseptic principles in practice of surgery" before the British medical association, in Dublin. Lister virtually brought down the mortality of surgery due to infections from 45% to 15%, a tremendous achievement by any standards, present or past.

Adolfneubar introduced metal instruments and established the first aseptic hospital in 1883. Halsted, was the first to use rubber gloves (1890) and he advocated

gentleness and fineness in the techniques of surgical operations. Berger, from Paris, in the first to adopt the use of cap, gown, and facemask as suggested by bacteriologist Flugge. Willis McDonald was one of the first persons to fix accountability for the development of infection in clean operative wounds on the doctors and nurses. He pointed out that fine spray of infective saliva expelled from the mouth during conversation. He further observed that visitors to operations were a constant menace to surgical operations. In their anxiety to see the surgical procedures, ask questions, cough near the table and bring large quantities of microscopic dirt on their shoes to the operating suite. He took cultures of the air in the operating room and demonstrated that the number of visitors present in the operating room influenced the number of colonies on the plate. In 1926, Meleney demonstrated the necessity of masking adequately the nose as well as the mouth of the surgeon and his team including the anesthetists. Meleny thus proposed that adequate sterilization of suture materials is necessary for effective wound healing and prevention of SSI.

**AETIOPATHOLOGY**

It is almost axiomatic that injury is followed by inflammation. An understanding of the nature mechanisms and consequences of inflammation is important to the surgeon i.e. surgical procedure results in an inflammatory reaction. The surgeon who understands, the nature and mechanism of this reaction to injury lies within his power the ability to minimize the adverse consequences, and to utilize its reaction to the benefit of the patient. Inflammation resulting from trauma may initially appear to differ from that resulting from bacterial infection or from physical agents such as heat, cold and radiant

energy. This is only apparent, the basic response is the same regardless of the initiating factor. Injury triggers an organized and complex cascade of cellular and biochemical events that result in a healed wound.

**Physiology of wound healing** [6.7.8.9.10]

The body's ability to replace injured or dead cells and to repair tissues after inflammation is critical to survival. The repair of tissue damage caused by surgical resection wounds and diverse types of chronic injury can be broadly separated into two processes. Regeneration and healing.

Regeneration results in restitution of lost tissues: healing may restore original structures but involves collagen deposition and scar formation. Tissues with high proliferation capacity such as hematopoietic system , epithelia of the skin and gastrointestinal tract, renew themselves continuously and can regenerate after injury as long as the stem cells of these tissues are not destroyed.

Superficial wounds, such as a cutaneous wound that only damages the epithelium can heal by epithelial regeneration. Incisional and excisional skin wounds that damage the dermis heal through formation of a collagen scar.

Extracellular matrix scaffolds are essential for und healing because they provide the framework for cell migration and maintain the correct cell polarity for the reassembly of multilayer structures. Furthermore cells in the extracellular matrix such as fibroblasts, macrophages and other cell types are the source of agents that are critical for tissue repair.

Healing is a fibro-proliferative response that "patches" rather than restores a tissue. It is a complex but orderly phenomenon involving a number of processes.[19]

1. Induction of an inflammatory process in response to the initial injury with removal of damaged and dead tissue.
2. Proliferation and migration of parenchymal and connective tissue cells.
3. Formation of new blood cells (angiogenesis) and granulation tissue.
4. Synthesis of extracellular matrix proteins and collagen deposition.
5. Tissue remodeling.
6. Wound contraction.
7. Acquisition of wound strength.

Not all of the above mentioned events occur in every repair reaction.

**Forms of healing [9]**

Surgeons customarily divide types of wound healing into first and second "intention". First intention (primary) healing occurs when tissue is cleanly incised and reapproximated and repair occurs without complication.

Second intention (secondary) healing occurs in open wounds through the formation of granulation tissue. Granulation tissue is the red, granular, moist tissue that appears during healing of the open wounds. Microscopically it contains new collagen, blood vessel, fibroblasts and inflammatory cells, especially macrophages. Covering of this tissue is then followed by spontaneous regression of the epithelial cells. Most infected wounds and burned tissue heal by the way of second intention.

**The nature of repair** [9]

In a broader sense, the nature of repair has been depicted schematically.

As this topic is centered on laparotomy wounds and infections, only healing of a surgical incision is described here.

The surgical incision causes death of a limited number of epithelial cells and connective tissue cells as well as disruption of epithelial basement membrane continuity. The narrow incisional space immediately fills with clotted blood containing fibrin and blood cells; dehydration of the surface clot form the well-known scab that covers the wound.

Within 24 hours neutrophils appear at the margins of the incision, moving towards the fibrin clot The epidermis at its cut edges, thickens as a result of mitotic activity of the basal cells, and within 24 hours to 48 hours, spurs of epithelial cells from the edges both migrate and grow along the cut margins of the dermis, depositing basement membrane components as they move. They fuse in the midline beneath the scab, thus producing a continuous albeit, thin epithelial layer.

By day 3, the neutrophils have largely been replaced by macrophages. Granulation tissue progressively invades the incision space. Collagen fibers are now present at the margins of the incision, but at first they are vertically oriented and do not bridge the incision. Epithelial proliferation continues and hence the epidermal covering layer is thickened.

By day 5, the incisional space is filled with granulation tissue. Neovascularization is maximal. Collagen fibrils become more abundant and start

bridging the incision. The epidermis recovers its thickness, and differentiation of surface cells yields a mature epidermal architecture with surface keratinization.

During the second week, there is continued accumulation of collagen and proliferation of fibroblasts. The leukocytic infiltrate, edema, and increased vascularity have largely disappeared. At this time, the long process of blanching begins, accomplished by the increased accumulation of collagen within the incisional scar and by regression of vascular channels.

By the end of first month, scar comprises a cellular connective tissue devoid of inflammatory infiltrate. covered now by intact epidermis, the dermal appendages that have been destroyed by the line of incision are permanently lost. The tensile strength of the wound increases thereafter, but it may take months for the wounded area to attain its maximal strength.

When there is more extensive loss of cells and tissue, as occurs in infarction. inflammatory ulceration, abscess formation and surface wounds creating large defects, the reparative process is more complicated. The common denominator in all these situations is a large tissue defect that must be filled. Regeneration of parenchymal cells cannot completely reconstitute the original architecture. Abundant granulation tissue grows in from the margin to complete the repair. This form of healing is referred to as secondary union or healing by second intention. of the many differences between primary and secondary forms of healing. the most salient is the phenomenon of wound contraction, that is significant feature of healing by secondary intention.

**Mechanisms of wound healing[10]**

Wound healing as we have seen is a complex phenomenon involving a number of processes, including induction of an acute inflammatory process by wounding, regeneration of parenchymal cells, migration and proliferation of both parenchymal and connective tissue cells, synthesis of extra-cellular matrix proteins, remodeling of connective tissue and parenchymal components, and collagenization and acquisition of wound strength.

Cutaneous wound healing is generally divided into three phases:

1. Inflammation (early and late)
2. Granulation tissue formation and re-epithelialization
3. Wound contraction, extracellular matrix deposition and remodeling

Table 1: Growth factors and cytokines affecting various steps in wound healing

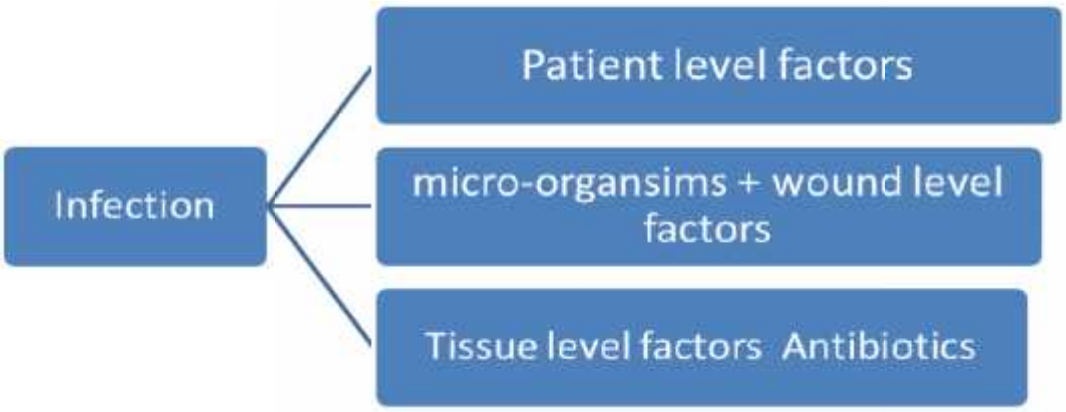
| | |
|---|---|
| Monocyte chemotaxis | PDGF, FGF, TGF β |
| Fibriblast migration | PDGF, FGF, TGF β, EGF, TNF, IL-1 |
| Fibroblast proliferation | PDGF,EGF,FGF, TNF |
| Angiogenesis | VEGF, Angiogenesis, FGF |
| Collagen synthesis | TGF β, PDGF |
| Collagen secretion | PDGF, FGF, EGF, TNF, (TGF β inhibits) |

PDGF- Platlet derived growth factor, FGF- Fibroblast growth factor, EGF- Epidermal growth factor, TNF – Tumour necrosis factor.

Impaired healing occurs due to many reasons and a wise surgeon recognizes them and attempts remedy before he wields his scalpel so as to reduce the rate of surgical site infection and help proper wound healing. Of the many causes incriminated in defective wound healing, tissue hypoxia resulting from cardio pulmonary diseases, peripheral diseases and malnutrition and in chronic inflammatory disorders is a major cause. A prior search into these problems is a must before surgery is undertaken.

The repair process is influenced by many factors iincluding,[10]

1. Tissue environment and extent of tissue damage
2. The intensity and duration of stimulus
3. Condition that inhibits repair, such as the presence of foreign bodies and inadequate blood supply
4. Various diseases that inhibit repair (diabetes in particular) and treatment with steroids.




**Microorganisms Encountered In Wounds[11] :**

Although the microbial flora of infected wounds frequently is so varied, a group designated as organisms most frequently isolated from laparotomy wounds would include the following:

- Staphylococcus aureus.
- Streptococcus pyogenes
- Coliform bacilli(from the lower half of body)
- Bacteroides species and other anaerobic non sporing gram- negative and gram positive rods.
- Proteus species.
- Psuedomonas species.
- Clsotridium species
- Enterococci.

Since anaerobic microorganisms are the predominant micro flora of humans are constantly present in the intestinal tract, upper respiratory tract, and genitourinary tract. It is not unexpected to find them invading both usual and unusual anatomical sites, giving rise to severe and often fatal infections. This is particularly true when the host defenses, either naturally or artificially, have been so altered as to permit an overgrowth of these organisms.

A wide variety of aerobic and anaerobic species of bacteria may be present, either singly or in combination, in infection of wounds and other soft tissues. The commonest pyogenic bacteria are S. pyogenes, pneumococcus and colifrom bacilli such as Escherichia coli. Proteus species and Pseudomonas aeruginosa. Anaerobic organisms, particularly clostridium perfingens and other clostridia, Bacteroides species and anaerobic cocci, may be important in infections of wounds, especially abdominal wounds, soiled deep or lacerated wounds and wherever devitalized tissues provide suitably anaerobic conditions.[11]

In many cases there is a mixed infection with more than one bacterial species, and in some of these cases a pathogenic synergy may be evident with two or more species acting in concert to cause by either alone. Mixed infection with Gram-positive cocci and coliform bacilli are not uncommon and polymicrobial infections with anaerobes such as bacteroides and fusiforms or fuso-spirochaetal associations are well recognized. [11]

Special associations of certain pathogens with particulate conditions should be borne in mind eg. Many postoperative abdominal or pelvic wounds have coliform bacilli associated with a moderate exudate during the early healing stage, the infection being often superficial and resolving without specific therapy. But a combination of coliforms

with bacteroides may cause a more severe synergic infection calling for prompt antibacterial therapy.

Pathogenic micro-organisms are, logically the major determinates of postoperative sepsis. The micro-organisms involved may be endogenous or exogenous in origin. The former are regarded as normal flora at another site in the body. The latter are the target of cross- infection control measures.

Table No 2: Factor affecting wound healing

| PATIENT CHARECTERISTICS | OPERATION CHARECTERISTICS |
|---|---|
| Age | Duration of surgical scrub |
| Nutritional status | Skin antisepsis |
| Diabetes | Pre-operative shaving |
| Smoking | Pre-operative skin preparation |
| Obesity | Duration of operation |
| Co-existant infection at a remote body site | Antimicrobial prophylaxis |
| Colonization with micro organisms | Operating room ventilation |
| Altered immune response | Inadequate sterilization of instruments |
| Length of pre-operative stay | Foreign material in surgical site |
| | Surgical drains |
| | Poor surgical technique |

**Patient level factors affecting the incidence of wound infections :**

Following is a consideration of factors thought to affect the susceptibility of any wound to infection at the whole patient level: further, these have been divided into two categories: endogenous and exogenous. Endogenous refers to unique attributes of the

patient which either may(e.g. obesity. malnutrition) or may not(eg. age) be alterable prior to surgery. Exogenous refers to characteristics of the operative experience not unique to any patient which can frequenty be influenced by the surgeon eg, length of operation).

1. **Endogenous Fators**

**Age:**

Extremes of age have long been thought to influence the likelihood of wound infection, perhaps owing to age owing to decreased immunocompitance.[13] Even in clean contaminated procedures, age has been asscociated with an increased infection rate. Age, obviously, is an immutable patient characteristics. And, even if it is a risk factor for wound infection, it appears to be at most a modest one. With patients more than 66 years old, being 6 times more likely to develop infection than are patients 1 to 14 years old.

**Pre-existing illness:**

It has been logically assumed that wound infection is more common among patients with multiple pre-existing diseases, although how to quantify this factor of generalized illness is unclear. Whether or not newer, more comprehensive measures of patient physiologic status, such as Acute Physiologic Assessment and Chronic Health evaluation (APACHE) ll or III, will give more precise prediction of risk remains to be seen.

**Diabetes mellitus**[14]

Hyperglycemia has several deleterious effects upon host immune function, most notably impaired function of neutrophils and mononuclear phagocytes. Hyperglycemia may also be a marker of the catabolism and insulin resistance associated with surgical stress response, and that exogenous insulin administration may ameliorate the catabolic state.

Poor control of blood glucose during surgery and in perioperative period increases the risk of infection and worsens outcome hence glycemic control decreases the risk. Moderate Hyperglycemia (>200mg/dl) at any time on the first postoperative day increased the risk of SSI fourfold after noncardiac surgery. In a large randomized trial of critically ill postoperative patient, exogenous insulin administration to keep blood glucose concentrations <110mg/dl was associated with a 40% decrease of mortality, fewer nosocomial infections, and less organ dysfunction. Meta-analysis of the approximately 35 existing trials indicate that the risk of postoperative infection decreases significantly by tight glucose control, regardless of whether or not the patient had diabetes mellitus.

**Obesity**

Although intuitively a risk factor, obesity has not consistently been found to be related to wound infections. It is not clear whether this effect was independent of other SSI ; diseases also associated with wound infections, such as diabetes mellitus. But it has been found that obesity to be associated with sternal or mediastinal wound infection, independent of other risk factors. Obesity, therefore, may be only weakly associated with wound infections.

**Length of preoperative hospitalization** [1,13,17]

Prolonged preoperative stay is now proven to increase the SSI risk in all patients. Independent of age, illness and nature of operation. The theory proposed for this is the colonization of nosocomial bacteria that are more drug resistant than their wild counterparts. Cruse and Ford, in their 10 years study have shown that patients hospitalized for 0 to 1 day had a clean SSI rate of 1.2%. With a week stay, 2.1% and in those hospitalized for more than two weeks, a 3.4%, SSI rate. However, length of preoperative stay is likely a surrogate for severity of the illness and co-morbid conditions that require in patient workup and/or therapy before the operation. The current recommendation that can be derived at, from the above data is to minimize the duration of preoparative stay, whenever possible, as especially in clean, elective surgeries. P. K. Agarwal in their study shows the infection rate was lowest in patient who was operated upon within seven days of admission while highest in patient who stayed for more than 21 days before operation in the ward.[17]

**Malignancy**

The presence of malignancy and its addendant, although poorly understood, alteration in immune status has sometimes been considered a risk factor for wound infection. The presence of malignant disease. Especially when widespread and metastatic, is a risk factors in the development of post-operative sepsis. Past malignant disease which had been removed or controlled is probably not a significant factor. The exceptions to these are malignant lymphomas and leukemia's, where the degree of tumor

control may be difficult to assess, and patients with these malignancies often remain immunosuppressed.

**Remote site Infection**

It has been found an epidemiologic correlation between remote site infection and subsequent surgical wound infection. The greatest risk appeared to be with remote infections involving a medical device, such an indwelling urinary catheter.

It is unclear whether preoperative treatment of the remote infection successfully reduced the subsequent risk to the wound. Given the current aggressiveness with which distant infections are sought and treated preoperatively, such as by routine urine analysis, it is doubtful whether this question will ever be fully answered. It seems prudent to continue to consider remote site infection a risk factor and to treat it appropriately prior to operation, if possible.

**Malnutrition:**

For some types of operations, severe Protein energy malnutrition(PEM), is crudely associated with postoperative nosocomial infections, impaired wound healing, dynamics or death. The National academy of sciences, National research council, study on the efficacy of infection control(SENIC) and NNIS schemes for SSI risk stratification do not explicitly incorporate nutritional status as a predictive variable although it may be indirectly represented in the latter too. It is generally assumed by Clinicians that infections are more abundant, more severe and of longer duration in malnourished, as compared to well nourished, patient. This assumption is supported by the observation

that, Immune functions when tested in its component parts, is depressed by Malnutrition (Bistran 1977; Murray and Murray, 1979).

Theoretical arguments can be made for a belief that severe preoperative malnutrition should increase the risk of both incisional and organ/space infection. However, an epidemiological association between incisional SSI and malnutrition has been difficult to demonstrate for all surgical sub-specialties.[1]

**Cigarette smoking'**

The effect of cigarette smoking on wound infection rates has, surprisingly, not been well studied. It is found to be associated with a slightly increased sternal wound infection rate among patients undergoing cardiac surgery. The concept of cigarette smoking as a risk for wound infection should be heeded because it is a potentially alterable behavior in the preoperative period.

**ASA Score**[14]

As incorporated in the National Nosocomial Infections Surveillance System (NNIS), the most recognized factors are the wound classification, American Society of Anesthesiology Class 3 or higher and prolong operative time.

**Box 2. American Society of Anesthesiology (ASA) physical status score.**

ASA 1 : A normal healthy patient

ASA 2 :A patient with mild to moderate systemic disturbance that results in no functional limitations.

Example :Hypertension, diabetes mellitus, chronic morbid obesity, extremes of age.

ASA 3 : A patient with severe systemic disturbance that results in functional limitations: Examples Poorly controlled hypertension, diabetes mellitus with vascular complications, angina pectoris, prior myocardial infarction, pulmonary disease that limits activity.

ASA 4 :A patient with a severe systemic disturbance that is life threatening with or without the planned procedure. Example: congestive heart failure unstable angina pectoris, advanced pulmonary, renal or hepatic dysfunction.

ASA 5 :A morbid patient not expected to survive with or without the operative procedure. Example : Ruptured abdominal aortic aneurysm, pulmonary embolism, head injury with increased intracranial pressure.

ASA 6 : Any patient in whom the procedure is an emergency. Example: ASA 4E

## 2. EXOGENOUS FACTORS :

### Length of operation

Risk of wound infection has repeatedly been shown to be propoertional to length of the operative procedure.[13,24] Wound infection with longer procedures, roughly doubling every hour of the procedures. SEN1C report, also found duration of operation of greater than 2 hours to be the second greatest independent, predictor of risk after multivariate analysis, with a regression coefficient of 1.04. It is unclear from these studies, however, how frequently a prolonged duration of operation was secondary to case's inherent complexity versus a simpler case taking an unusually long time complete. This question is partly addressed by culver et al 1991 modification of the SENIC index. Rather than taking an arbitrary time(e.g. 2 hours) over which an operation was designated prolonged, he considered a procedure lengthy of its length fell above the 75th percentile for other similar procedures. Therefore, an appendectomy was considered prolonged if it lasted more than hour, whereas coronary artery bypass grafting was not prolonged unless it required more than 5 hours. Using this index, operative time was still one of three variables, along with wound class and ASA the independently predicted infection. Prolongation of an operation, whether from an unusually complicated procedure, increased likelihood of normal wound contamination, or lapses in antibiotic coverage, must be considered a significant risk factor for wound infection.

### Glove Punctures [13,16]

Much attention is given to glove perforations and risks they pose. However, the contribution of glove perforation to infection is over-emphasized. The use of electrical permeability to detect perforation is erroneous (Miller at al 1972) and may have

contributed to the high rates quoted. Recent figures put the perforation rate at around 5 percent (Cruse and Foorde, 1973) and the rate at Flinders Medical center is similar. In an investigation of glove perforations using inflation and water immersion our recent perforation rate was 6 percent. The infection risk of glove perforation must be considered in conjunction with hand-carriage of Staphylococcus aureus. Peter J E. Cruse et al. in their study found 11.6 percent of gloves were punctured at the end of surgical procedure, not a single wound infection occurred in these patients. Organisms probably escaped from the glove punctures in insufficient numbers to be a serious hazard in a clean wound with adequate local resistance.[13]

**Emergency Procedures**

Several studies have shown emergency operations to be particularly prone to wound infections of the 4465 wounds studied by GikEgen et al 623 were made under emergent situations, and the wound infection rate for these was 5.1%. versus the 3842 elective wounds with an infection rate of 2.9%. Garibaldi et al reported a wound infection odds ratio of 7.6 (95% confidence interval, 3.2 to 18.2) for emergency versus elective operations, but after multivariate analysis, this factor was no longer significant. At this time, emergency operations do not by themselves clearly predispose to wound infection.

**Time of day**

In their initial study of 23,649 wounds. Ruse and Foord found that the clean wound infection rate more than tripled, to 6.8%, for cases done between midnight and 8 AM, and that the clean contaminated wound infection rate doubled to 18.3% during the

same period. These rates do not account for other factors, such as underlying patient illness, and are therefore difficult to interpret.

**Month of year**

It remains unclear why a consistent rise in wound infection rate appears in the early summer, but Mead et al clearly demonstrated this phenomenon in their study of 844 wounds over an 18-month period, These findings are similar to those found by Cruse an Foord for both clean and nonclean wounds, with a peak clean infection rated in July of 1977, the last year of the 10-year study, versus less than 1% for most of the rest of the year. Condon et al, in a 5-year study of wounds at the Wood Veterans Administration Hospital, also noted peaks in wound infection rates in July whether this weak risk factor is the result of new su staff changes in weather and personal hygiene, or other factors is not known. P. K. Agarwal in their study shows patients operated in winter season (November to March) developed less infection than those operated during summer(April to July) and rainy season(August to October)[17]

**Airborne Contamination**

With the exception of rare epidemics traced to either air handling or surgical staff airborne contamination of wounds in general surgery appears to play a small role in the pathogenesis of wound infections. Whyte et al. studied 188 cholecystectomies and found that although the bacterial concentration on drapes distant from the wound and from the drapes close to the wound depended much more on bacteria or skin flora. In fact, more bacteria were thought to have transferred from the wound and from the drapes close to the wound depended much more on bacteria or skin flora. In fact, more bacteria were thought to have transferred from the wound to the drapes than vice versa. These

results are in contrast to studies of upper-joint procedures, in which 98% of the bacteria found in wounds by Whyte et al were thought to be from the air, and the use of ultra clean operating rooms, age described by Lidwell et al, decreased the rate of joint infection by approximately 25%.[1]

In the prospective study of 190 Patients undergoing elective colorectal surgery by Claesson and Holmlundu in which all wounds were theoretically classified as clean contaminated, multivariate analysis revealed that 5 or more CFU per ml of bacteria in peritoneal fluid were predictive of wound infection. How and if the routine culture of wounds should be incorporated into normal clinical practice is unclear although any further studies of wound infections ought to include this important.

**Preoperative Hair Removal[1,18]**

Shaving is a ritual which may cause increased infection rates (Seropain and Raynoleds). The presence of hair has not been documented as a source of wound infection. If removal of hair is required to achieve adequate visualization or to enhance adhesiveness of dressings then the following should be considered.

1. A depilatory cream (seropian and Reymolds 1971)
2. The use of sterilizable electric clippers
3. If you do decide to shave, use disposable razors
4. Never use a brush because of cross infection hazards(aerosol shaving creams are recommended).
5. Hair should be removed as close to the time of surgery as possible so as to reduce infection of traumatized skin.

**Adhesive Drapes**

The benefits of plastic adhesive wound drapes remain controversial. Cruse and Foord found no benefit to the use of plastic adhesive drapes, with an overall infection of 1.5% in wounds draped in the standard manner and 2.4% in wounds protected plastic drapes. In 1985, however, Alexander et al reported on an effective preparation consisting of a 1-minute alcohol application followed by the application iodine impregnated plastic adhesive drapes currently, any benefit to the use of plastic drapes appears to be small.

**Wound irrigation**

The irrigation of wounds with antibiotic-containing solutions has a long history, starting with the use of topical sulfonamides in wounds in the 1930s. several later studies appeared to show a benefit to wound irrigation, particularly in clean contaminated or contaminated procedures. For example, in a prospective study of 240 patients undergoing colon operations published in 1972 by Anderson et al the infection rate for patients receiving topical ampicillin was 2.5% versus 18.3% in wounds not receiving intestinal antiseptics, which appeared to be more effective than the topical antibiotics With the introduction of more effective antibiotics for prophylactic use in clean contaminated and contaminated procedures, the added benefit of topical antibiotics is probably minimal in all but the most severely contaminated wounds, in clean wounds, in which the wound infection rate is already low, topical antibiotic irrigation is probably of no benefit, although its low cost and minimal morbidity assure its continued use.

**Tissue Level Factors Affecting The incidence of wound infections**

**Tissue Perfusion**

Perfusion of a wound is critical to healing for several reasons, the two most important probably being delivery of oxygen and neutrophils, two essential and interrelated elements of normal wound healing. owing to the inevitable so to 100 µm of poorly perfused adjacent tissue, the normal wound environment has a $PO_2$ of 5 to 10mm Hg, a $PCO_2$ of 50 to 60 mm Hg, and a pH of 65 to 6.9. In vitro studies have demonstrated a decrease in neutrophil killing and response to chemo attractant under these conditions. Further, Knighton et al demonstrated in viva studies a 5 log reduction in wound fluid bacteria counts at 14 days simply by increasing the inspired FI $O_2$ of room air from 20% to 45%. This effect was later noted to be further enhanced by the administration of systemic antibiotics. The deleterious effect of the presence of a wound foreign body are also explained by decreased oxygen tension, as Silver demonstrated in 1978 that the microenvironment immediately adjacent to a foreign body has a P $O_2$ close to 0 mm Hg. It must be noted, however, that clinical experiments to support the use of hyperoxia to aid wound healing have yet to be completed. Meanwhile, it is axiomatic that wounds do not heal in the presence of severe vascular occlusive-disease.

**Local immune response**

Only in the past 10 years have the tools become available to study the systemic and local immune response at a cell and cell mediator level. The keratinocyte has been shown to be an immunologically active cell able to produce and express a wide spectrum of immune response mediators, including intracellular adhesion molecule interleukin (IL-

1), tumor necrosis factor. a IL-6, IL-8, and transforming growth factor-a. The effect of the presence of bacteria on all of these responses is unknown, but it is hoped, manipulation of these events will decrease the likelihood of wound sepsis. It is further interesting to note that, although the uninfected fetal wound has been demonstrated to heal by a process closer to regeneration than scar deposition, Frantz et al have shown that the presence of bacteria in fetal wounds induces a more adult-like collagen deposit fibroplasia, and neovascularization These findings raise the question of the role of bacteria or their products even in normal, uninfected adult wound healing.

## COMPLICATION OF LAPAROTOMY WOUND

### Seroma [22]

A Seroma is a collection of liquefied fat, serum and lymphatic fluid under the incision. The fluid is usually clear, yellow, and somewhat viscous and is found in the subcutaneous(sc) layer of the skin. Seromas represent the most benign complication after an operative procedure and are particularly likely to occur when large skin flaps are developed in the course of the operation.

### Presentation and Management

A seroma is usually manifested as a localized and well-circumscribed swelling, pressure or discomfort, and occasional drainage of clear liquid from the immature surgical wound.

Prevention of seroma formation may be achieved by placing suction drains under the skin flaps or in potential dead space created by lymphadetenectomy. Premature removal of drains frequently results in large seromas that require aspiration under sterile

conditions, followed by placement of a pressure dressing. A seroma that reaccumulates after at least two aspirations is evacuated by opening the incision and packing the wound with saline-moistened gauze to allow healing by secondary intention.

**Hematoma**[2,20]

A hematoma is an abnormal collection of blood, usually in the sc layer of recent incision or in a potential space in the abdominal cavity after extirpation of organ, for example, splenic fossa hematoma after splenectomy or pelvic hematoma Prostrectomy. Hematomas are more worrisome than seromas use of the potential for secondary infection. Hematoma formation is related to inadequate hemostasis, depletion of clotting factors, and the presence of coagulopathy. A host of disease processes contribute to coagulopathy, including myeloproliferative disorders, liver disease, failure, sepsis, clotting factor deficiencies, and medications. Medications most commonly associated with y are antiplatelet drugs, such as aspirin, clopidogrel bisulphate (Plavix), Ticlopidine hydrochloride( Ticlid), eptifibatide (integrilin). And abciximab (ReoPro). And anticoagulants, such as ultrafractionated heparin, low-molecular weight heparin( LMWH: enoxaparin (Lovenox )dalteparin sodium (Fragmin), tinzaparin (Innohep), and warfarin sodium.

**Presentation and Management**

The clinical manifestations of a hematoma vary with its size and location. A hematoma may appear as an expanding. unsightly swelling or pain in the area of surgical incision , or both. on physical examination, a hematoma appears as a localized soft swelling with purplish/blue discoloration of the overlying skin. The swelling from small

to large and may be tender to palpation or associated with drainage of red fluid out of the fresh wound

Hematoma formation is prevented preoperatively by correcting any clotting the abnormalities and discontinuing medications that alter coagulation. One must balance risk of significant bleeding due to uncorrected medication induced coagulopathy and the risk of thrombosis after discontinuation of therapy. In patients at high risk for thrombosis must be who are scheduled to undergo an elective major surgical procedure, warfarin (NR) to discontinued 3 days before surgery to allow the international normalized ratio be less than 1.5.Then given heparin intravenously (IV) or an equivalent dose SC. Those receiving standard heparin can have the medication discontinued 2 to 3 hours before surgery and those receiving LMWH (variable half. Life), 12 to 15 hours before surgery. Anticoagulants a then resumed 24 to 48 hours after surgery. Patients taking clopidogrel must have the medication withheld 5 to 6 days before surgery; otherwise, the surgery must be delayed.

**Acute Wound Failure (Dehiscence)[2,20]**

Acute wound failure (wound dehiscence or a burst abdomen) refers to postoperative separation of the abdominal muscloaponeurotic layers. It is among the most dreaded complications faced by surgeons and of greatest concern because of the risk of evisceration, the need for immediate intervention, and the possibility of repeat dehiscence, surgical wound infection, and incisional hernia formation

Acute wound failure occurs in approximately 1% to 3% of patients who undergo an abdominal operation. Dehiscence most often develops 7 to 10 days postoperatively

but may occur any time after surgery from 1 to more than 20 days. A multitude of factors may contribute to wound dehiscence.

**Factors Associated With Wound Dehiscence**

- Technical error in fascial closure
- Emergency surgery
- Intra-abdominal infection
- Advanced age
- Wound infection, hematoma, and seroma
- Elevated intra-abdominal pressure
- Obesity
- Chronic corticosteroid use
- Previous wound dehiscence
- Malnutrition
- Radiation therapy and chemotherapy
- Systemic disease(uremia, diabetes mellitus)

**Presentation and Management**

Acute wound failure may occur without warming and evisceration makes the diagnosis obvious. Sudden, dramatic drainage of a relatively large volume of a clear, salmon-colored fluid precedes dehiscence in a fourth of patients. Probing the wound with a sterile cotton-tipped applicator or gloved finger may detect the dehiscence.

Prevention of acute wound failure is largely a function of careful attention to technical detail during fascial closure. For very high-risk patients, interrupted closure is often the wisest choice. Alternative methods of closure must be selected when primary

closure is not possible without undue tension. Although retention sutures were used extensively in the past, their use is less common today, with some surgeons opting to use a synthetic prosthesis or tissue graft.

Once dehiscence is diagnosed, treatment depends on the extent of fascial separation and the presence of evisceration or significant intra-abdominal contamination (intestinal leak, peritonitis). A small dehiscence in the proximal aspect of an upper midline incision 10 to 12 days postoperatively can be managed conservatively by packing the wound with saline-moistened gauze and using an abdominal binder. In the event of evisceration, the eviscerated intestines must be covered with a sterile, saline-moistened towel and preparations made to return to the operating room after a very short period of fluid resuscitation. Once in the operating room, thorough exploration of the abdominal cavity is performed to rule out the presence of a septic focus or an anastomotic leak that may have predisposed to the dehiscence. Treatment of the infection is of critical importance before attempting closure. Management of the incision is a function of the condition of the fascia. When technical mistakes are made and the fascia is strong and intact, primary closure is warranted. If the fascia is infected or necrotic, debridement is performed. If after debridement the edges of the fascia cannot be approximated without undue tension, consideration needs to be given to closing the wound with absorbable mesh or the recently developed biologic prostheses (decellularized porcine submucosa and dermis and human cadaveric dermis). Attempts to close the fascia under tension guarantee a repeat dehiscence and possible intra abdominal hypotension. Definitive surgical repair to restore the integrity of the abdominal wall will eventually be required if absorbable mesh is used but not if a biologic prosthesis is used.

Absorbable mesh and biologic prostheses protect from evisceration, maintain the abdominal domain, and provide a barrier to prevent bowel desiccation, bacterial invasion, and nonadherent, potentially permanent closure. Autologous skin grafts are used to reconstitute the epithelial barrier, and flaps(local regional or free) are used to reconstruct the abdominal wall. For short-term management of a dehisced wound, a wound vacuum system can be used that consists of open-cell foam placed on the tissue, semi occlusive drape to cover the foam and skin of the patient, and suction apparatus. The wound vacuum system provides immediate coverage of the abdominal wound and acts as a dressing that minimizes heat loss and does not require suturing to the fascia. By using negative pressure, the device removes interstitial fluid and thus lessens bowel edema, decreases wound size, reduces bacterial colonization, increases local blood perfusion, and induces the healing response. Successful closure of the fascia can be achieved in 85% of cases of abdominal wound dehiscence.

**Surgical site Infection (wound Infection)**[2]

**Presentation and Management**

Superficial and deep surgical site infections are accompanied by erythema, tenderness, edema, and occasionally drainage. The wound is often soft or fluctuant at the site of infection, which is a departure from the firmness of the healing ridge present elsewhere in the wound. The patient may have leukocytosis and a low-grade fever. According to the Joint Commission on Accreditation of Healthcare organizations, a surgical wound is considered infected if it meets the following criteria

1. Grossly purulent material drains from the wound
2. The wound spontaneously opens and drains purulent fluid

3. The wound drains fluid that is culture positive or Gram stain positive for bacteria
4. The surgeon notes erythema or drainage and opens the wound after deeming it to be infected.

At the time of surgery the operating surgeon plays a major role in reducing or minimizing the presence of postoperative wound infections. The surgeon must be attentive to personal hygiene (and scrubbing) and that of the entire team.[7] In addition, the surgeon must make certain that the patient undergoes a thorough skin preparation with appropriate antiseptic solutions and is draped in a sterile careful fashion. During the operation, steps that have a positive impact on outcome are followed:

1. Careful handling of tissues
2. Meticulous dissection, hemostasis, and debridement of devitalized tissue.
3. Compulsive control of all intraluminal contents
4. Preservation of blood supply of the operated organs
5. Elimination of any foreign body from the wound
6. Maintenance of strict asepsis by the operating team(no holes in gloves, avoidance of the use of contaminated instruments, avoidance of environmental contamination such as debris falling from overhead)
7. Thorough drainage and irrigation of any pockets of purulence in the wound with warm saline
8. Ensuring that the patient is kept in a euthermic state, well monitored, and fluid resuscitated.
9. At the end of the case, a judgment with regard to closing the skin or packing the wound.

The use of drains remains somewhat controversial in preventing postoperative wound infections. In general, there is virtually no indication for drains in this setting. However, placing closed suction drains in very deep, large wounds and wounds with large wound flaps to prevent the development of a seroma or hematoma is a worthwhile practice.

Once a surgical site infection is suspected or diagnosed, management depends on the depth of the infection. For both superficial and deep surgical site infections. Skin staples are removed over the area of the infection, and a cotton-tipped applicator maybe easily passed into the wound with efflux of purulent material and pus. The wound is gently explored with the cotton-tipped applicator or a finger to determine whether the fascia or muscle tissue is involved. If the fascia is intact, debridement of any nonviable tissue is performed the wound is irrigated with normal saline solution and packed to its base with saline moistened gauze to allow healing of the wound from the base anteriorly and prevent premature skin closure. If widespread cellulitis is noted, administration of iv antibiotics must be considered. However, if the fascia has separated or purulent material appears to be coming from deep to the fascia, there is obvious condemn about dehiscence or an intra-abdominal abscess that may require drainage or possibly a reoperation. Wound cultures are controversial. If the wound is small, superficial, and not associated with cellulitis tissue necrosis culture may not be necessary. However, if fascial dehiscence and a more complex infection are present, material is sent for culture. A deep surgical site infection associated with grayish, dish water coloured fluid, as well as frank necrosis of the fascial layer, raises suspicion for the presence of a necrotizing type infection.

Most Postoperative infections are treated with healing by secondary intention (allowing the wound to heal from the base anteriorly, with epithelialization being the final event. In some cases when there is a question about the amount of contamination, delayed primary closure may be considered. In this setting, close observation of the skin wound for 5 days may be followed by closure of the skin if the wound look clean and patient is otherwise doing well.

Recently, wound vacuum systems have been used in large, deep, or moist wounds with generally successful outcomes. Their advantage is a decrease in the nursing time previously required for dressing changes, as well as less pain for the patient.[2]

# MATERIALS AND METHODS

## SOURCE OF DATA

This study was undertaken in surgical units of Shri. B.M.Patil Medical College, Hospital and Research Centre, Vijayapur. During the period of October 1, 2013 to September 1 ,2015. A total of 100 patients were studied. Out of 100 patients 50 were in Primary Closure group and 50 were in Delayed Primary closure group cases.

## METHOD OF COLLECTION OF DATA

The patients admitted in B.L.D.E.U.'s Shri. B. M. Patil Medical College Hospital Vijayapur attending surgical OPD who underwent exploratory laparotomy were studied. Details of patient were recorded including Clinical History, Clinical Examination, and Investigation.

## INCLUSION CRITERIA

All diagnosed cases of peritonitis, who underwent exploratory laparotomy and found to be contaminated intra operatively were included in this study from the period of October 1, 2013 to September 1, 2015. Perforated appendicitis, perforated hollow viscous, ileostomy closure, trauma and intra-abdominal abscess / other peritonitis,

Patients> 18 years of age are included.

## EXCLUSION CRITERIA

Immuno compromised patients

Abdominal Malignancy.

**RESEARCH HYPOTHESIS:**

Delayed primary closure reduces length of hospital stay and incidence of surgical site infections in dirty abdominal wounds.

**SAMPLE SIZE**

Prospective Randomized Trial of Two Wound Management Strategies for Dirty Abdominal Wounds.Conducted byStephen M. Cohn MD. Giovanni Giannottia MD et all in Division of Trauma and Surgical Critical Care and Colorectal Surgery, Department of surgery, University of Miami School of Medicine , Miami Florida showed that the length of hospital stay in DPC group 7.1+/-3.5 day and in PC group it is 5.3+/-1.4 days .Considering the average standard deviation of hospital stay 2.4.

Study populations of 100 patients were required for both groups.

Following statistical tests will be used to compare the results.

- Diagrammatic presentation.
- Mean ± S D

**PREOPERATIVE PARAMETERS ASSESSED**

Age

Sex

Duration of symptoms

WBC on Admission

Risk factors – Diabetes mellitus,

Obesity (body mass index > 30kg/m2)

Malnutrition (clinical observation of muscle wasting or albumin

(< 2.5 g/dl)

Cardiovascular diseases

## Procedure

Patient diagnosed as acute peritonitis and posted for exploratory laparotomy during the period of October 1 2013 to September 1 2015 were included. In this series a total of 100 patients were included and were divided in two groups. Each group had 50 patients. Patients underwent laparotomy procedure for acute peritonitis during surgery.Turbid ascites was cultured and peritoneal lavage was performed with warm saline until clear effluent restored. Drain was placed in the pelvis and anastomotic site through a separate incision in the abdominal wall. Peritoneum, muscle and fascia were closed in layers.

For primary closure, wounds were closed with monofilament interrupted suture for delayed primary closure, skin and subcutaneous tissue are left open and packed with 10 %( betadine) povidone iodine soaked gauge, which was changed daily to prevent excessive collection of exudates. If the wound appears clean on post-operative day $5^{th}$ it was closed under local anesthesia. Otherwise wet packing is continued and DPC is done on later date. The presence of purulent discharge at the incision site in both cases was sent for bacterial culture.

**INTRAOPERATIVE FINDING**

- Contamination of wound
- Gangrenous changes
- Grossly inflamed
- Perforation of hollow viscera

In the entire series, the patients who developed wound infection in primary closure group and delayed primary group were observed. The wounds of these patient were opened by removing the skin stitches only and managed by open technique with a daily Betadine socked packing.

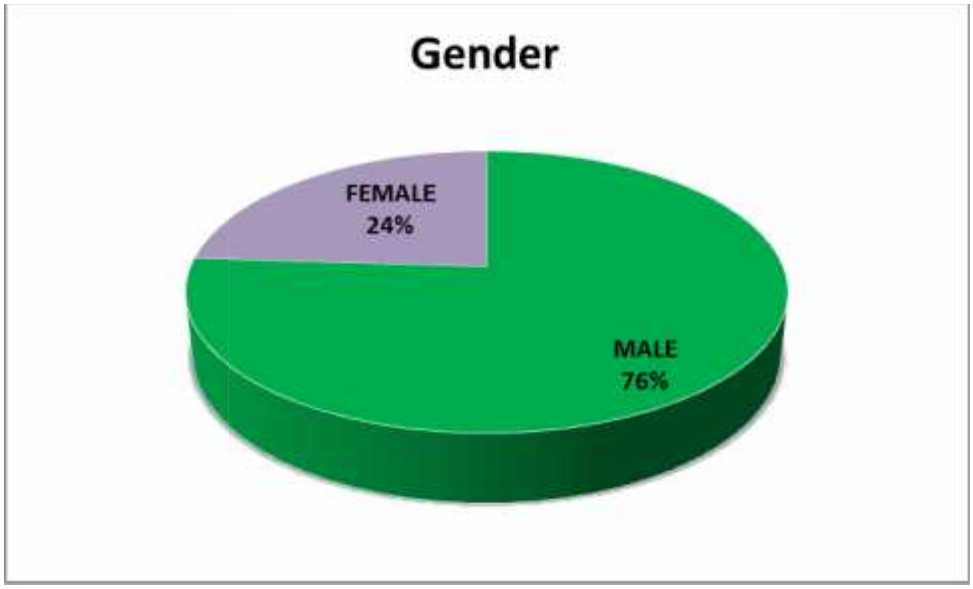
# RESULT

A total of 100 patients, 76 male and 24 female included in this study. (table no 1 ).

**Table 3: Percentage Distribution of Gender**

| Gender | N | Percent |
|---|---|---|
| **MALE** | 76 | 76 |
| **FEMALE** | 24 | 24 |
| **Total** | 100 | 100 |

**Graph 1: Percentage Distribution of Gender**

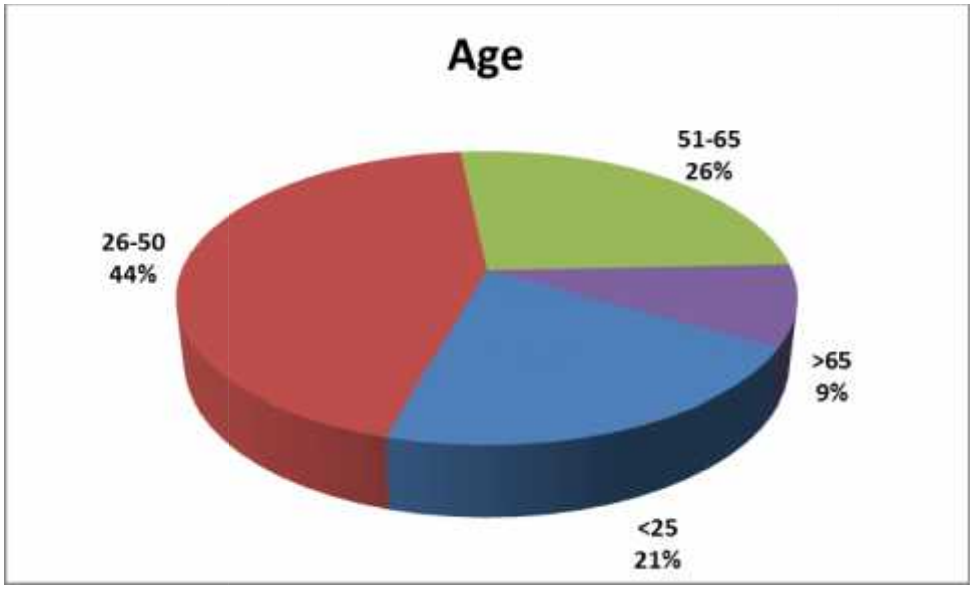


The mean age of the patients was 50±5 years with the range of 18 to 65 years. There were 25 (25%) patients in range of 15 to 25 years, 44(44%) patients were in the range of 26 to 50 years and 26(26%) patient were in the range of 51 to 65 years, more than age of 65 years were 9.

**Table No 4 Percentage Distribution of Age**

| Age | N | Percent |
|---|---|---|
| **<25** | 21 | 21 |
| **26-50** | 44 | 44 |
| **51-65** | 26 | 26 |
| **>65** | 9 | 9 |
| **Total** | 100 | 100 |

**Graph 2 Percentage Distribution of Age**

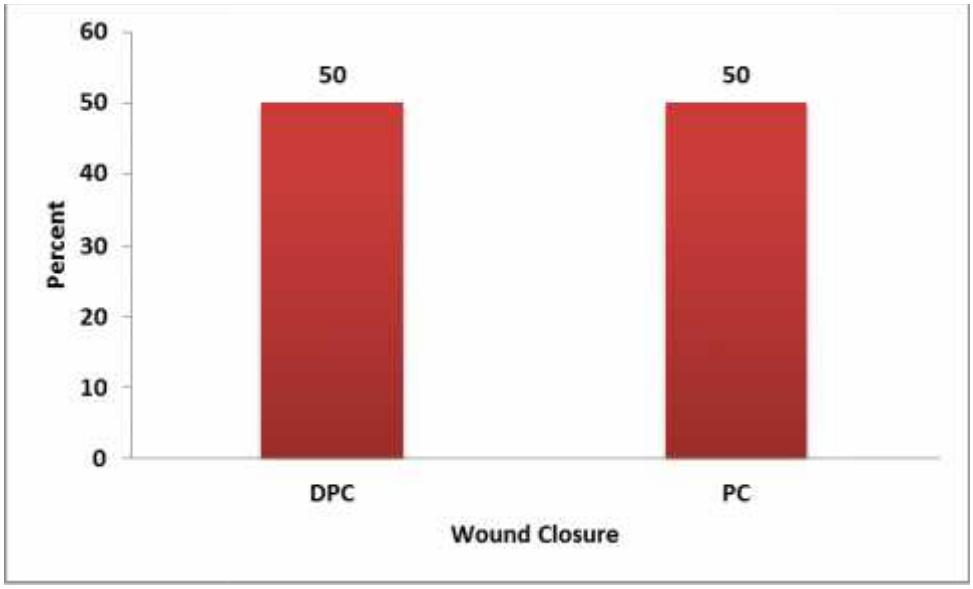


The patients were divided into two equal groups primary closure and delayed primary closure group.

**Table no. 5 Percentage Distribution of Type of Wound Closure**

| TYPE OF WOUND CLOSURE | N | Percent |
|---|---|---|
| **DPC** | 50 | 50 |
| **PC** | 50 | 50 |
| **Total** | 100 | 100 |

**Graph No. 3 Percentage Distribution of Type of Wound Closure**



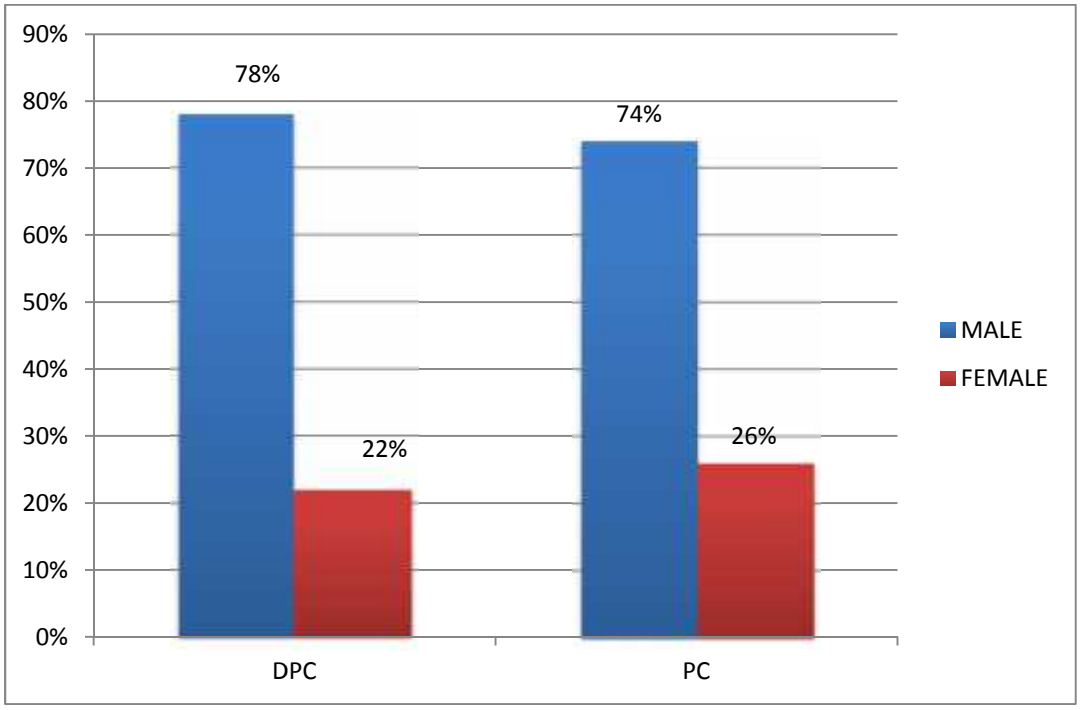
In primary closure group, of 50, 37 were male and 13 were female.

In delayed primary closure group, of 50, 39 were male and 11 were female.

**Table no 6 Distribution of Type of Wound Closure by Gender**

| Procedure | Male | | Female | | TOTAL |
|---|---|---|---|---|---|
| | N | % | N | % | |
| DPC | 39 | 78% | 11 | 22% | 50 |
| PC | 37 | 74% | 13 | 26% | 50 |

**Graph 4 Distribution of Type of Wound Closure by Gender from mail**



In the entire series, 33 patients developed wound infection.

In primary closure group, wound infection was observed in 27 patients (54 %.). The wounds of these patient were opened by removing the skin stitches only and managed by open technique with a daily Betadine socked packing, out of 27 patients, 19 underwent secondary closure and 8 of 27 patients were left open for healing by secondary intention .

In delayed primary closure group, wound infection was observed in 6 patients (12.00%). Forty four (44) patients wound healed without any infection. Infected wound in this group were opened by removing skin stitches and subjected to healing by secondary intention.

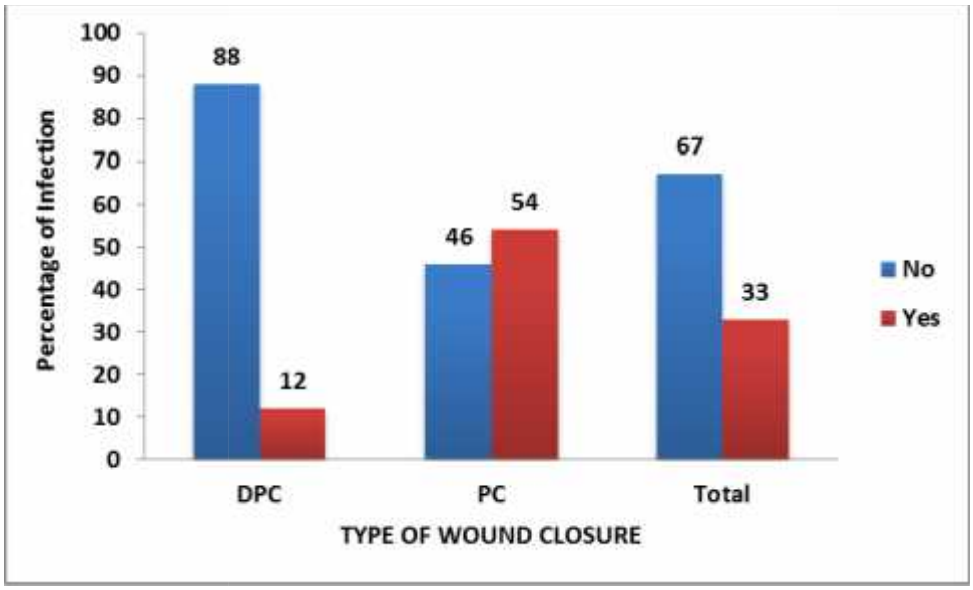
There was a significant association between wound infection and type of skin closure (delayed primary closure 12.00%vs primary closure $p<0.000$)

**Table No 7: Distribution of Type of Wound Closure by SSI**

| Infection | No | | Yes | | Total | p value |
|---|---|---|---|---|---|---|
| | N | Percent | N | Percent | | |
| **DPC** | 44 | 88 | 6 | 12 | 50 | 0.000* |
| **PC** | 23 | 46 | 27 | 54 | 50 | |

*significant

**Graph 5 : Distribution of Type of Wound Closure by SSI**



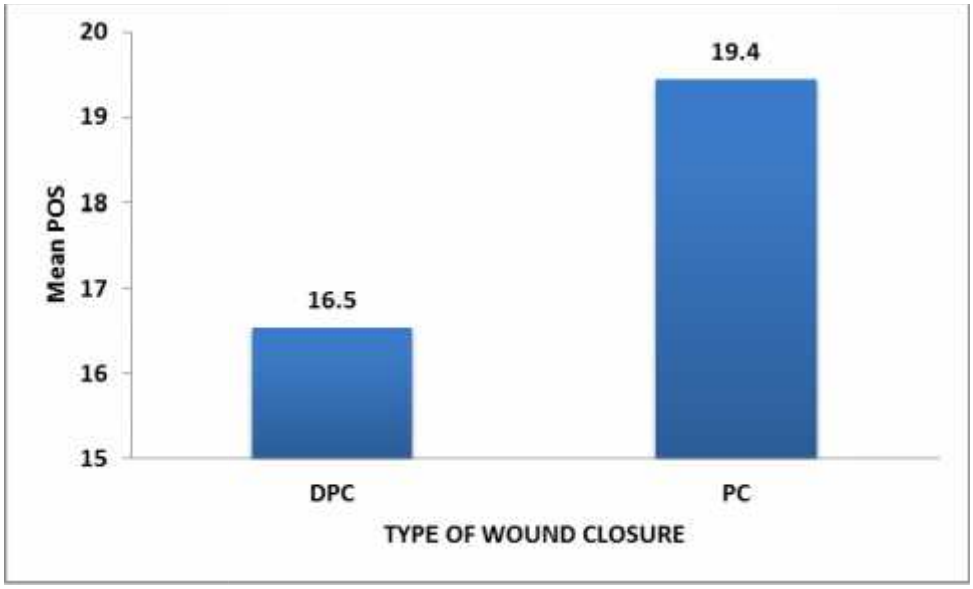
The mean post-operative stay, 16.5± 5 days were seen in delay primary closure group and 19.4 ± 5 days were in primary group,

There was significant association between post-operative stay and SSI (p 0.002)

**Table no 8 : Duration of POS**

| TYPE OF WOUND CLOSURE | DPC | | PC | | p value |
|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | |
| POS | 16.5 | 3.6 | 19.4 | 5.3 | 0.002* |

**Graph 6 : Duration of POS**



**Organism Isolated from SSI**

Out of hundred patients the most common organism cultured from the wounds were E.coli (13) klebsiella (17), pseudomonas (21), staph. aureus(9) coagulase negative staphallococi (4)and sterile (36) enterococci (4).

**Table no 9 Percentage of Distribution of Organisms**

| Organisms | Percentage |
|---|---|
| E.coli | 13 |
| Klebsiela | 17 |
| Staph.aureus | 9 |
| Coagulase negative staphallococci | 4 |
| Enterococci | 4 |
| Sterile | 36 |

# DISCUSSION

This study was undertaken in surgical units of Shri. B.M.Patil Medical College, Hospital and Research Centre, Vijayapur. During the period of October 1, 2013 to September 1 ,2015. A total of 100 patients were studied. Out of 100 patients, 50 were in Primary Closure group and 50 were in Delayed Primary closure group.

Open wound management of contaminated wound is a practical measure that has been used for centuries [34].The use of delayed primary closure was popularized by military surgeons. The method of DPC has the advantage of reducing the numbers of colonic bacteria, particularly anaerobes contaminating to the wound [34]

However, the disadvantages of allowing exogenous bacteria such as staphylococci to contaminate the wound in ward before closure has been recognized [34]

In the entire series, 33 patients developed wound infection. In primary closure group wound infection rate was 54.4% while it was 12 % in delayed primary group. There was significant difference between 2 groups regarding wound infection ($p<0.00$).Our study showed that delayed primary closure was more suitable for wound management for contaminated or dirty wound.

In our study the most common diagnosis was perforated appendix (27%) followed by Ileal perforation (24%), prepyloric (16%), duodenal (18%). And also showed that the mean post –operative stay was 16.5 ±5 in delayed primary group and 19.4 ±5 in primary group $p < 0.002$.There is a significant association between type of wound closure and length of hospital stay .

Study conducted by Duttaroy D D, Jitendra J .et al “Management Strategy For Dirty Abdominal Incisions: Primary Or Delayed Primary Closure? A Randomized trial

.At Department of Surgery , Government Medical College and Sir Sayajirao General Hospital ,Baroda, Gujarat ,India 2009. in their study demonstrated SSI developed after incision closure in 23% of patients infection were significantly more common in the primary group (42.25%vs 2.57%for DPC; p=0.00375)and also mean length of hospital stay were longer after PC ( 18.52 days than DPC (13.86) days ) (P value 0.02) [36]

Stephen M .Cohn, Giovanni Giannottiaet al "Prospective Randomized Trial Of Two Wound Management Strategies For Dirty Abdominal Wounds" Division of Trauma and Surgical Critical Care and Colorectal Surgery, Department of surgery, University of Miami School of Medicine , Miami Florida. Demonstrated that in DPC group wound infection rate was 12%, in PC group was 48%. Wound infection rate was greater in the PC group than DPC. Length of the hospital stay and hospital charges were similar between two groups [35] .

Mukhtar Ahmad ,Kishwar Ali, Humera Latif, et al "Comparison Of Primary Wound Closure With Delayed Primary Closure In Perforated Appendicitis" at Department of Surgery ,*Department of Gastroenterology, Ayub Teaching Hospital, Abbottabad, Pakistan .[37]conducted study on 158 patients , 56 (35.4%) male and 102 (64.6%) female were included in their study .In entire series, 36 (22.8%) patients developed wound infection .There was a significant association between wound infection and type of closure (Delayed primary closure 6.3% vs. Primary Closure 39.2%,p< 0.000 ).Concluded that DPC is the optimal management strategy in case of perforated appendicitis as it decreases the incidence of wound infection [37].

Chiang RA, Chen SL, Tsai YC. "Delayed Primary Closure Verses Primary Closure For Wound Management In Perforated Appendicitis :A Prospective Randomized

Controlled Trial" at Department of surgery, Mackay Memorial Hospital, Taitung Taiwan, ROC .conducted study on Delayed primary closure verses primary closure for wound management in perforated appendicitis: a prospective randomized controlled trial. Showed that, in entire series, wound infection developed after wound closure in 21% of the patients .The PC group had a higher incidence of wound infection (38.9% vs. 2.9%, $p< 0.001$) and longer length of hospital stay (8.4 days vs. 6.3 days ,$p= 0.038$).concluded that DPC is the optimal management strategy for perforated appendicitis wounds. Significantly reduces the wound infection rate and length of stay.

Factors affecting SSI, according to CDC are extremes of age, poor nutritional status, presence of diabetes, obesity, steroid use, a coincident infection or Colonization and a dysfunctional immune system[39].The patient with more than 50 years of age had more complication (P value < 0.05)

**Table no 10: Comparison of result with other studies**

| Serial no | Studies done by | Delayed primary group | Primary group |
|---|---|---|---|
| 1 | Duttaroy D D et al | 2.57% | 42.25% |
| 2 | Stephen M Cohn et al | 12% | 48% |
| 3 | Mukhtar Ahmad et al | 6.3% | 39.2% |
| 4 | Chaing RA et al | 2.9% | 38.9% |
| 5 | Our study | 12% | 54.4% |

## PHOTO GALLERY


**AIR UNDER DIAPHRAM**

**POST OPERATIVE DAY 3 IN PC**

**POST OPERATIVE DAY 3 IN DPC**

**POST OPERATIVE DAY 5 IN PC**

**POST OPERATIVE DAY 5 IN DPC**

**POST OPERATIVE DAY 6 IN DPC**

**POST OPERATIVE DAY 7 IN DPC**

# CONCLUSION

Laparotomy wound complications are multifactorial, it depends on many factors. A strategy of DPC of dirty abdominal wound clinically appears to decrease the rate of wound infection, when compared with PC without increasing the hospital length of stay.

# SUMMARY

The study was conduct on 100 patients out of which 50 patients underwent Delayed Primary Closure and 50 patients underwent Primary Closure of skin in contaminated and dirty abdominal wounds at Shree B M Patil Medical College, Hospital and Research Centre, between October 1,2013 to September 1,2015

Statistical analysis was done accordingly, P-value less than 0.05 was considered significant .The study demonstrated that in primary group wound infection rate was 54% while it was 12% in delayed primary closure. There was significant difference between two groups regarding wound infection P-value <0.00

Our study showed that the main post-operative stay ,16.5 days seen in delayed primary closure group and 19.4 days in primary closure group.

There was significant association between post-operative stay and type of closure P-value 0.002.

Our study concluded that delayed primary closure was more suitable for wound management for contaminated or dirty abdominal wounds.

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# ANNEXURES

## ETHICAL CLERANCE CERTIFICATE

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. SD/13
Date 14-11-13
BLDE U's Shri B.M. Patil Medical College.

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

**INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE**

The Ethical Committee of this college met on 13-11-2013 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Comparative study of delayed primary closure versus primary closure of skin in contaminated and dirty abdominal wounds"

Name of P.G. student Dr Jadesh. G. Bhadragoudra.
Department of Surgery.

Name of Guide/Co-investigator Dr Basavaraj Narasanagi.
Assoc Prof of Surgery.

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

## SAMPLE INFORMED CONSENT FORM

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

**TITLE OF THE PROJECT:**

COMPARITIVE STUDY OF DELAYED PRIMARY CLOSURE VERSUS PRIMARY CLOSURE OF SKIN IN CONTAMINATED AND DIRTY ABDOMINAL WOUNDS/ INCISION.


**PRINCIPAL INVESTEGATOR:** Dr JADESH BHADRAGOUDRA
Department of General Surgery
Email:jadesh121@gmail.com

**PG GUIDE :** Dr BASAVARAJ NARASANAGI M.S.
Associate Professor
Department of Surgery
B.L.D.E. University's
Shri B.M. Patil Medical College Hospital
& Research Centre, Sholapur Road,
VIJAYAPUR - 586103

**PURPOSE OF RESEARCH:**

I have been informed that this study will analyses COMPARITIVE STUDY OF DELAYED PRIMARY CLOSURE VERSUS PRIMARY CLOSURE OF SKIN IN CONTAMINATED AND DIRTY ABDOMINAL WOUNDS/INCISION

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 understand that relevant history will be taken. I will undergo detailed clinical examination after which necessary investigations will be done whenever required, which would help the investigator for appropriate management.

**RISKS AND DISCOMFORTS:**

I understand that I/my ward may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings which are associated with the usual course of treatment.

**BENEFITS:**

I understand that I/my wards participation in this study will help to analyse the effectiveness of delayed primary closure in contaminated wounds to reduce surgical site infection.

**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 JADESH BHADRAGOUDRA 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 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 JADESH BHADRAGOUDRA 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 BASAVARAJ N DR JADESH BHADRAGOUDRA

(Guide) (Investigator)

**STUDY SUBJECT CONSENT STATEMENT:**

I confirm that DR JADESH BHADRAGOUDRA 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.

**PROFORMA**

SL. NO CASE / CONTROL NO

Name

Age IP NO

Sex Unit

Religion DOA

Occupation DOS

Address DOD

SOCIO-ECONOMIC STATUS

Complaints :

A. HISTORY OF PAIN

1. MODE OF ONSET
2. SITE OF PAIN
3. HOW LONG IS THE HISTORY OF PRESENTING COMPLAINT OF PAIN
4. DOSE PAIN RADIATE
5. CHARACTER OF PAIN
6. RELIEF OF PAIN
7. NUMBER OF HOURS SINCE ACUTE PAIN STARTED

B. VOMITING

1. PROJECTILE / NON-PROJECTILE
2. NATURE OF VOMITUS
3. NUMBER OF TIMES

4 HAEMAILMESIS

C. FEVER

D. DISTENSION OF ABDOMEN

E. CONSTIPATION

PAST HISTORY :

PERSONAL HISTORY : SMOKER! ALCOHOLIC

GENERAL PHYSICAL EXAMINATION

BUILT : NOURISHMENT :

PALLOR ICTERUS FEBRILE PEDAL EDEMA

GENERAL LYMPHADENOPATHY NUTRITIONAL STATUS :

. A. GENERAL APPEARANCE : NORMAL/THIN B. ANTHROPOMETRY :

HT

WT

VITAL DATA

TEMPERATURE :

PULSE

RESPIRATORY RATE

BLOOD PRESSURE :

SYSTEMIC EXAMINATION :

PER ABDOMEN :

INSPECTION :

CONTOUR OF ABDOMEN

MOVEMENTS WITH RESPIRATION

UMBILICUS

VISIBLE PERISTALSIS

VISIBLE PULSATION

SKIN OVER ABDOMEN

HERNIAL ORIFICES

GENITALIA

PALPATION

LOCAL RISE OF TEMPERATURE

HYPERAESTHESIA

TENDERNESS

RIGIDITY /GUARDING

LUMP

PALPATION OF HERNIAL SITES

ABDOMEN GIRTH

PERCUSSION

SHIFTING DULLNESS

FLUID THRILL

OBLITERATION OF LIVER DULLNESS

AUSCULTATION

BOWEL SOUNDS

DRE : DIGITAL RECTAL EXAMINATION RESPIRATORY SYSTEM

CARDIOVASCULAR SYSTEM CENTRAL NERVOUS SYSTEM CLINICAL

DIAGNOSIS LABORATORY TESTS

FIB%

TOTAL COUNT

DIFFERENTIAL COUNT N/L/E/D/M

PT

APT

URINE ROUTINE :

RBS

FBS

PPBS

B.UREA

S.CREATININ

TOTAL PROTIN

S.ALEUMIN

SERUM ELECTROLYTES

PERITONEAL ASPIRATION

PERITONEAL FLUID ANALYSIS AND CULTURE SENSITIVITY BLOOD CULTURE

BLOOD GROUPING

CHEST X RAY

ERECT ABDOMEN X-RAY

ULTRA SONOGRAPHY OF ABDOMEN AND PELVIS : CT SCAN OF

ABDOMEN

ARTERIAL BLOOD GAS ANALYSIS

OTHERS

OPERATIVE PROCEDURE (DATE AND TIME)'

INTRA-OPERATIVE DIAGNOSIS :

INTRA –OPERATIVE FINDINGS :

DURATION OF PROCEDURE:

POST OPERATIVE INVESTIGATIONS

LENGTH OF STAY IN HOSPITAL AFTER PROCEDURE

OBSERVATION OF WOUND FOLLOWING ABDOMINAL SURGERY IN PRIMARY CLOSURE OF WOUND.

| Variables | Day 4 | Day 6 | Day 8 | Day 10 | Day 12 | Day 14 |
|---|---|---|---|---|---|---|
| Soakage of | | | | | | |
| Foul smellin | | | | | | |
| Type • of discharge | | | | | | |
| Wound | | | | | | |

OBSERVATION OF WOUND FOLLOWING ABDOMINAL SURGERY IN DELAYED CLOSURE OF WOUND

| Variables | Day 4 | Day 6 | Day 8 | Day 10 | Day 12 | Day 14 |
|---|---|---|---|---|---|---|
| Soakage of | | | | | | |
| Foul smellin | | | | | | |
| Type of discharge | | | | | | |
| Wound | | | | | | |

# MASTER CHART

| | NAME | IP NO. | AGE | SEX | DIAGNOSIS | PROCEDURE | TYPE OF WOUND CLOSURE | SSI | | POS | DOC |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | DISCHARGE | WOUND GAPING | | |
| 1 | SANJAY | 1291 | 30 | MALE | I P | GH P | DPC | P | - | 15 | 4 |
| 2 | PARVATHI | 27417 | 22 | FEMALE | AP P | APC | DPC | P | - | 14 | 5 |
| 3 | NIRMALA | 28764 | 38 | FEMALE | I P | GH P | DPC | P | - | 18 | 4 |
| 4 | REKHA | 23562 | 22 | FEMALE | AP P | APC | PC | P | AB | 10 | 5 |
| 5 | SHANKAR | 38314 | 28 | MALE | I P | GH P | DPC | P | AB | 16 | 4 |
| 6 | PEERAPPA | 25414 | 65 | MALE | GB | RA | DPC | P | AB | 20 | 5 |
| 7 | BHIMANNA | 22136 | 64 | MALE | D P | GH P | DPC | P | AB | 14 | 3 |
| 8 | BANDUCHUR | 25938 | 72 | MALE | D P | GH P | DPC | P | AB | 12 | 5 |
| 9 | SHIVASHANKAR | 20473 | 50 | MALE | I P | GH P | DPC | P | AB | 13 | 4 |
| 10 | DASHRATH | 37237 | 27 | MALE | M T | MVL | DPC | P | AB | 20 | 8 |
| 11 | AMOGI | 24353 | 26 | MALE | I P | GH P | DPC | P | AB | 14 | 5 |
| 12 | PARAPPA | 25417 | 65 | MALE | PP P | GH P | DPC | P | AB | 18 | 4 |
| 13 | VIJAYA | 44174 | 12 | MALE | AP P | APC | PC | AB | AB | 10 | 0 |
| 14 | MUTAWWA | 4464 | 83 | FEMALE | PP P | GH P | DPC | P | AB | 18 | 5 |
| 15 | NINGAPPA | 5059 | 55 | MALE | J P | GH P | DPC | AB | AB | 14 | 0 |
| 16 | LACHAPPA | 5225 | 65 | MALE | PP P | GH P | DPC | P | P | 22 | 6 |
| 17 | NIZAMUDDIN | 5803 | 21 | MALE | AP P | APC | PC | AB | AB | 12 | 0 |
| 18 | MANOJ | 7477 | 18 | MALE | AP P | APC | PC | AB | AB | 10 | 0 |
| 19 | CHIDANNAND | 14505 | 35 | MALE | I P | GH P | DPC | P | AB | 16 | 4 |
| 20 | SANGANNA | 8691 | 75 | MALE | D P | GH P | DPC | P | P | 20 | 5 |
| 21 | ROOPA SINGH | 3890 | 46 | MALE | I P | GH P | DPC | P | AB | 15 | 5 |
| 22 | EKAPPA | 7281 | 38 | MALE | D P | GH P | DPC | P | AB | 14 | 4 |
| 23 | SANDEEP | 29571 | 28 | MALE | J P | GH P | PC | AB | AB | 25 | 0 |
| 24 | SABU | 29681 | 15 | MALE | AP P | APC | PC | AB | AB | 12 | 0 |
| 25 | SANDEEP | 2973 | 28 | MALE | J P | GH P | PC | P | P | 22 | 0 |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | SABU | 29606 | 45 | MALE | D P | GH P | DPC | P | AB | 20 | 5 |
| 27 | SANGAPPA | 28848 | 60 | MALE | GB | RA | PC | P | P | 24 | 0 |
| 28 | BASAPPA | 36235 | 65 | MALE | PP P | GH P | DPC | P | AB | 18 | 5 |
| 29 | BHIMASHANKAR | 35730 | 60 | MALE | PP P | GH P | PC | P | P | 26 | 0 |
| 30 | BALABHIMA | 33320 | 65 | MALE | D P | GH P | PC | P | P | 6 | 0 |
| 31 | UMAR | 15018 | 19 | MALE | AP P | APC | PC | AB | AB | 14 | 0 |
| 32 | SHIVALINGAPPA | 16812 | 82 | MALE | D P | GH P | PC | P | P | 24 | 0 |
| 33 | TARA SHINGH | 5974 | 65 | MALE | PP P | GH P | PC | P | P | 20 | 0 |
| 34 | RAMANGOUDA | 21750 | 42 | MALE | I P | GH P | PC | AB | AB | 16 | 0 |
| 35 | BHAGYASHREE | 27619 | 13 | FEMALE | AP P | APC | PC | P | P | 24 | 0 |
| 36 | JAKNAYYA | 28205 | 14 | MALE | AP P | APC | PC | AB | AB | 12 | 0 |
| 37 | JAYSHREE | 28326 | 32 | MALE | I P | GH P | PC | P | P | 18 | 0 |
| 38 | CHANDRAKALA | 26556 | 15 | FEMALE | I P | GH P | PC | P | P | 16 | 0 |
| 39 | BAGAWWA | 26458 | 42 | FEMALE | D P | GH P | PC | P | P | 22 | 0 |
| 40 | BASAPPA | 36235 | 65 | MALE | GB | RA | PC | P | P | 23 | 0 |
| 41 | JATTEPPA | 896 | 42 | MALE | D P | GH P | PC | P | P | 26 | 0 |
| 42 | MALLAPPA | 36122 | 40 | MALE | I P | GH P | PC | P | P | 24 | 0 |
| 43 | KALAPPA | 3624 | 22 | MALE | J P | GH P | DPC | P | P | 26 | 6 |
| 44 | TARA SHINGH | 7974 | 65 | MALE | PP P | GH P | DPC | P | AB | 16 | 4 |
| 45 | NAGAMMA | 5835 | 65 | FEMALE | J P | GH P | DPC | P | AB | 15 | 4 |
| 46 | BHIMAPPA | 5636 | 60 | MALE | AP P | APC | DPC | P | AB | 13 | 3 |
| 47 | MAHANTESH | 4511 | 25 | MALE | J P | GH P | DPC | P | AB | 15 | 4 |
| 48 | NANDA BASAPPA | 3633 | 65 | MALE | D P | GH P | DPC | P | AB | 14 | 4 |
| 49 | REKHA | 1393 | 11 | FEMALE | I P | GH P | DPC | P | AB | 12 | 3 |
| 50 | JETTAPPA | 896 | 42 | MALE | AP P | APC | DPC | P | AB | 12 | 4 |
| 51 | KALLAPPA | 85 | 80 | MALE | AP P | APC | DPC | P | AB | 15 | 5 |
| 52 | MAHABOOB | 10486 | 36 | MALE | I P | GH P | DPC | P | AB | 14 | 5 |
| 53 | BINDU | 10898 | 27 | MALE | I P | GH P | DPC | P | AB | 16 | 3 |
| 54 | RENUKA | 9297 | 40 | FEMALE | I P | GH P | DPC | P | AB | 15 | 5 |
| 55 | SHANTABAI | 9041 | 41 | FEMALE | AP P | APC | DPC | P | AB | 17 | 3 |
| 56 | MAYAKKA | 7624 | 19 | FEMALE | APP | APC | PC | AB | AB | 13 | 0 |

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| 57 | EKKAPPA | 7218 | 38 | MALE | I P | GH P | DPC | P | AB | 16 | 5 |
| 58 | MALLAPPA | 6236 | 23 | MALE | APP | APC | DPC | P | AB | 12 | 3 |
| 59 | SIDDU | 25377 | 22 | MALE | J P | GH P | PC | P | p | 22 | 0 |
| 60 | DEVAPPA | 24882 | 60 | MALE | PP P | GH P | PC | P | P | 21 | 0 |
| 61 | MOHAN | 22540 | 60 | MALE | D P | GH P | PC | P | P | 23 | 0 |
| 62 | SITABAI | 22261 | 35 | FEMALE | APP | APC | PC | AB | AB | 12 | 0 |
| 63 | RAVI | 19050 | 26 | MALE | AP P | APC | PC | AB | AB | 16 | 0 |
| 64 | ANNAPURANA | 19024 | 45 | FEMALE | J P | GH P | PC | P | P | 22 | 0 |
| 65 | HANAMANTH | 18318 | 30 | MALE | PP P | GH P | PC | AB | AB | 20 | 0 |
| 66 | DEVIKA | 16682 | 45 | FEMALE | D P | GH P | PC | P | P | 25 | 0 |
| 67 | MUTTU | 15177 | 17 | MALE | APP | APC | PC | AB | AB | 20 | 0 |
| 68 | SIDDALINGAPPA | 14405 | 40 | MALE | I P | GH P | PC | AB | AB | 23 | 0 |
| 69 | MALLAPPA | 36122 | 40 | MALE | I P | GH P | PC | AB | AB | 20 | 0 |
| 70 | JAGADEESH | 1896 | 42 | MALE | D P | GH P | PC | P | P | 25 | 0 |
| 71 | MANJU | 5025 | 18 | MALE | AP P | APC | DPC | P | AB | 13 | 5 |
| 72 | APPASAB | 2594 | 55 | MALE | PP P | GH P | DPC | P | P | 24 | 6 |
| 73 | SANGAWWA | 271 | 70 | FEMALE | I P | GH P | DPC | P | P | 25 | 5 |
| 74 | CHANNAPPA | 30399 | 70 | MALE | PP P | GH P | DPC | P | AB | 16 | 6 |
| 75 | HONNAWWA | 27976 | 70 | FEMALE | D P | GH P | DPC | P | AB | 15 | 5 |
| 76 | CHIDANNAND | 2413 | 12 | MALE | APP | APC | PC | AB | AB | 14 | 0 |
| 77 | SIDDAPPA | 29119 | 42 | MALE | PP P | GH P | DPC | P | AB | 16 | 4 |
| 78 | RAMESH | 27724 | 38 | MALE | I P | GH P | DPC | P | AB | 13 | 5 |
| 79 | KRISHNABAI | 25075 | 65 | FEMALE | D P | GH P | DPC | P | P | 24 | 5 |
| 80 | KALLAPPA | 24331 | 42 | MALE | APP | AP C | DPC | P | AB | 15 | 3 |
| 81 | LALASAB | 25068 | 55 | MALE | I P | GH P | DPC | P | AB | 17 | 5 |
| 82 | YELAPPA | 23356 | 24 | MALE | AP P | APC | DPC | P | AB | 20 | 4 |
| 83 | MALIKSAB | 23214 | 65 | MALE | PP P | GH P | DPC | P | AB | 14 | 3 |
| 84 | NIMBAGI | 21435 | 62 | MALE | D P | GH P | DPC | P | AB | 17 | 4 |
| 85 | PRASHANTH | 21051 | 26 | MALE | AP P | APC | DPC | P | AB | 15 | 3 |
| 86 | ROSANBI | 20866 | 45 | FEMALE | PP P | GH P | DPC | P | AB | 24 | 5 |
| 87 | HANAMANTH | 20271 | 24 | MALE | AP P | APC | PC | AB | AB | 22 | 0 |

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| 88 | SHARNAPPA | 18609 | 57 | MALE | AP P | APC | PC | AB | AB | 20 | 0 |
| 89 | BHIMAPPA | 17404 | 30 | MALE | D P | GH P | PC | AB | AB | 25 | 0 |
| 90 | CHANAPPA | 16903 | 48 | MALE | I P | GH P | PC | P | P | 23 | 0 |
| 91 | GULABSAB | 11472 | 30 | MALE | APP | APC | PC | AB | AB | 16 | 0 |
| 92 | BHARATH | 11260 | 60 | MALE | I P | GH P | PC | P | P | 18 | 0 |
| 93 | MUTAPPA | 10529 | 55 | MALE | GB | RA | PC | P | P | 26 | 0 |
| 94 | BHIMRAI | 6603 | 45 | MALE | PP P | GH P | PC | P | P | 18 | 0 |
| 95 | SHREDEVI | 10532 | 55 | FEMALE | PP P | GH P | PC | P | P | 20 | 0 |
| 96 | ANNARAY | 11278 | 72 | MALE | GB | RA | PC | P | P | 30 | 0 |
| 97 | RANIBAI | 16905 | 45 | FEMALE | GLP P | C | PC | P | P | 20 | 0 |
| 98 | RUKMABAI | 17408 | 46 | FEMALE | I P | GH P | PC | P | P | 20 | 0 |
| 99 | PRIYA | 18606 | 26 | FEMALE | APP | APC | PC | AB | AB | 19 | 0 |
| 100 | UMABHARTHI | 18899 | 45 | FEMALE | D P | GH P | PC | AB | AB | 23 | 0 |