"ROLE OF 30% VERSUS 60% PERIOPERATIVE OXYGEN SUPPLEMENTATION IN REDUCING SURGICAL SITE INFECTION"

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ABSTRACT

Background:

The role of supplemental oxygen to prevent surgical site infection (SSI) in clean and clean contaminated cases has been recognized. Higher the concentration of oxygen lesser the is rate of oxygen.

Objective:

To compare the efficacy of perioperative 60% inspired oxygen versus 30% inspired oxygen to reduce surgical site infection.

Methods:

The study group received 60% fraction of inspired oxygen intra operatively and for 2 hours after surgery. The control group received 30% fraction of inspired oxygen intraoperatively and for 2 hours after surgery.

Results:

3 of the 47 study cases of class I surgeries developed SSI, and 5 of the 47 control cases of class I surgeries had significant SSI.

Among the clean-contaminated group, 5 of the 47 study cases and 8 of the 47 control cases developed significant post-operative SSI.

The over all p value when study group was compared to control group was found to be 1.34.

There is significant difference between the two groups.

Conclusion:

The use of supplemental perioperative oxygenation is beneficial in preventing SSI in patients undergoing class I and class II surgeries.

Key words:

Supplemental, perioperative, oxygenation

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INTRODUCTION

The treatment of infection has been an integral part of the surgeon's practice since the dawn of time. The body of knowledge that led to the present field of surgical infectious disease was derived from the evolution of germ theory and antisepsis. Application of the latter to clinical practice, concurrent with the development of anesthesia, was pivotal in allowing surgeons to expand their repertoire to encompass complex procedures that previously were associated with extremely high rates of morbidity and mortality due to postoperative infections. However, occurrence of infection related to the surgical wound was the rule rather than the exception. In fact, the development of modalities to effectively prevent and treat infection has occurred only within the last several decades.

It was in the late 1860s when Joseph Lister introduced the principles of antisepsis that postoperative infections, morbidity and mortality decreased substantially. Lister's work radically changed surgery from an activity associated with infection and death to a discipline that could eliminate suffering and prolong life[1].

Post operative wound infection remains one of the most common, of all post operative complications, and its diagnosis, treatment and prevention are matters of singular importance in pre-operative and post-operative care of all surgical patients.

Based on NNIS system reports(1991), SSIs (surgical site infections) are the third most frequently reported nosocomial infection, accounting for 14% to 16% of all nosocomial infections among hospitalized patients.

Among surgical patients, SSIs (previously known as surgical wound infections) are the most common nosocomial infection, accounting for 38% of all such

infections. Of these SSIs, two thirds are confined to the incision and one third involve organs or spaces accessed during operation.

It is a fundamental clinical observation that wounds do not heal in tissue that does not bleed, and they almost always heal in tissue that bleeds extensively. Continuous supply of oxygen through microcirculation is vital for healing process and for resistance to infection.

Oxidative killing of pathogens by polymorphonuclear leucocytes is the primary mechanism of defense against surgical pathogens. Oxygen partial pressure and wound tissue oxygen tensions have been shown to correlate with oxidative killing and have been reported to predict SSI rates[2].

The use of supplemental perioperative oxygen in surgical patients requires a thorough and accurate assessment of its effects prior to its general inclusion in SSI prevention standards. However evidence exits both in support of and against the use of oxygen therapy. Therefore, using the standards, we performed the study to assess the effect of supplemental perioperative oxygen on SSI incidence, morbidity, mortality and length of stay in elective surgical patients.

We compared the use of high inspired oxygen concentration with standard concentrations to determine the efficacy of this treatment in reducing SSI.

AIMS AND OBJECTIVE

To compare the efficacy of perioperative 60% inspired oxygen versus 30% inspired oxygen to reduce surgical site infection.

REVIEW OF LITERATURE

HISTORICAL REVIEW

The microbes are as old as the mankind itself. Throughout the history of mankind, treating infections has been one of the primary roles of a surgeon. Early in the history of mankind, there was recognition of inter play between wounds, infections and surgical manipulation. In fact, virtually all wounds became infected and infection was associated with high mortality.

There have been two phases of intense revolutionary development in the means employed by surgeons against infections.[3] 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 bacteriology such as Pasteur, Robert Koch, and Joseph Lister. Second phase, was that of effective systemic treatment of the same. This phase is associated great names of Domagk and Florey.

The development of bacteriology as a discipline dates from the time of Louis Pasteur (1822-95).[4] 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.

Robert Koch (1843-1910) in Germany perfected bacteriological techniques during his studies on the culture media. He introduced staining techniques and methods of obtaining bacteria in pure culture solid media. He also proposed the principles of infection.

The work of Phillip Semmelweis on the etiology and pathogenesis of puerperal sepsis and its prevention by asepsis and cleanliness is an important contribution, and even to this date is a broad guideline to those who would like to be practitioners of aseptic surgeries[5].

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[6]. 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. Von Volkman and Nussbaum of Munich hospital, Germany adopted Lister's methods between 1870 and 1880, which dramatically lowered the incidence of hospital gangrene in their institutions.

Ogston discovered staphylococcus in 1884. Frankel described Pneumococcus in 1887.[4] Von Blurgmann introduced steam sterilization in surgery in 1886.

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 finesse in the techniques of surgical operations. Berger, from Paris, in

1897 was probably 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 a fine sprays 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,they coughed near the table and brought 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, Meleny demonstrated the necessity of masking adequately the nose as well as the mouth of the surgeon and his team including the anesthetists. Early in the decade, a series of fatal postoperative infections isolating clostridium organisms causing gas gangrene were demonstrated. A similar organism was found in two tubes from the same lot of catgut used on those fatal cases. Meleny thus proposed that adequate sterilization of suture materials is necessary for effective wound healing and prevention of SSI[5].

Though Fleming discovered and commented on the possible clinical uses of penicillin, it took the combined efforts of Florey and Chain for over ten years of intense research to conclude on the excellent in-vivo activity of penicillin against an array of microorganisms in year 1940. US commercial giants 1940-45 started bulk

synthesis and wide spread use of penicillin. This was the dawn of antibiotic era and was then thought to be the beginning of end of the era of infection.

Despite improvements in operating room practices, instrument sterilization methods, better surgical technique and the best efforts of infection prevention practices, surgical site infections (SSIs) remain a major cause of nosocomial (hospital acquired) infections and rates are increasing globally (Alvarado 2000)[7].

Moreover, in countries where resources are limited, even basic life-saving operations, such as appendectomies and cesarean sections, are associated with high infection

rates and mortality. In these countries, therefore, it makes sense to focus on preventing SSIs in those procedures most frequently performed and/or those having the highest SSI rates.

Risk Factors for Development of Surgical Site Infections
Patient factors
Older age
Immuno-suppression
Obesity
Diabetes mellitus
Chronic inflammatory process
Malnutrition
Peripheral vascular disease
Anemia
Radiation
Chronic skin disease
Carrier state (e.g., chronic *Staphylococcus* carriage)
Recent operation
Local factors
Poor skin preparation
Contamination of instruments
Inadequate antibiotic prophylaxis
Prolonged procedure
Local tissue n
Hypoxia, hypothermia
Microbial factors
Prolonged hospitalization (leading to nosocomial organisms)
Toxin secretion
Resistance to clearance (e.g., capsule formation)

There are certain risk factors which can be modified to reduce the surgical site infections. Recent studies have showed the benefits of the modifications, like Prolonged preoperative hospitalization , Preoperative hair removal should be avoided if it is unnecessary. If hair must be removed, clip it with scissors just before the surgery[8]. The healing of closed surgical wounds depends on many factors, one of the most complex of which is the influence of technique and expertise. Several study showed the incidence of SSIs in relation to the different types of closure techniques used[9].

Along with the above mentioned factors certain other factors which have significant role in preventing surgical site infection. Perioperative hypoxia is one of the important factor which needs to be corrected. There are only few studies done across globally , and those studies have showed signficant improvement not only in reducing surgical site infection but also in reducing hospital stay and cost effectiveness. These studies encouraged us to do the present study on perioperative oxygen supplementation and their effect on surgical wounds.

PATHOLOGY AND PATHOGENESIS OF SURGICAL SITE INFECTIONS

The identification of SSI involves interpretation of clinical and laboratory findings, and it is crucial that a surveillance program use definitions that are consistent and standardized; otherwise, inaccurate SSI rates will be computed and reported. The Center for Disease Control's (CDC), National Nosocomial Infection Survey (NNIS) system has developed standardized surveillance criteria for defining SSIs. By these criteria, SSIs are classified as being either incisional or organ/space. Incisional SSIs are further divided into those involving only skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI).

CDC's CRITERIA FOR DEFINING SURGICAL SITE INFECTION (SSI)[10]

Superficial Incisional SSI

Infection occurs within 30 days after the operation and infection involves only skin or subcutaneous tissue of the incision and at least one of the following:

1) Purulent drainage, with or without laboratory confirmation, from the superficial incision.
2) Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
3) At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat.
4) Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Deep Incisional SSI

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation and infection involves deep soft tissues (e.g., fascial and muscle layers) of the incision and at least one of the following:

1) Purulent drainage from the deep incision but not from the organ/space component of the surgical site.

2) A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (>38ºC), localized pain, or tenderness, unless site is culture-negative.

3) An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination.

4) Diagnosis of a deep incisional SSI by a surgeon or attending physician.

Organ/Space SSI

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation and infection involves any part of the anatomy (e.g., organs or spaces), other than the incision, which was opened or manipulated during an operation and at least one of the following:

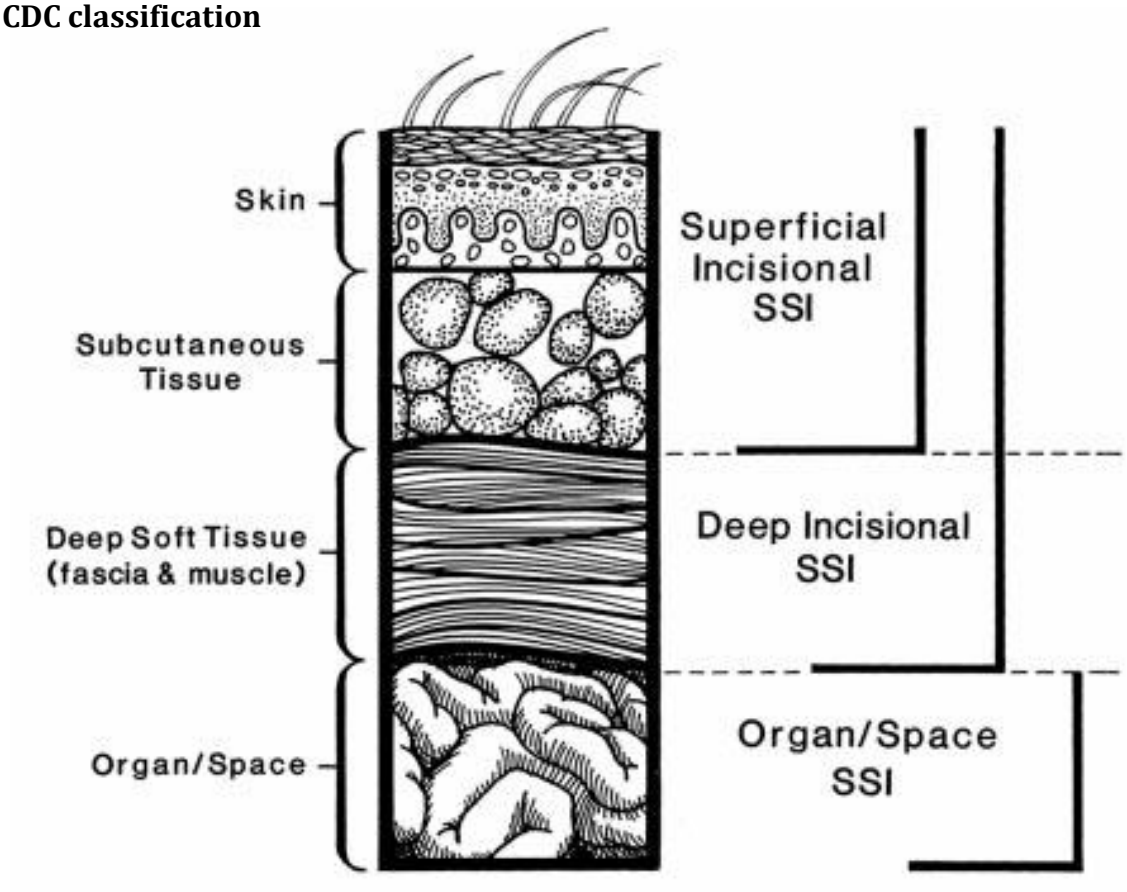
1) Purulent drainage from a drain that is placed through a stab wound into the organ/space.

2) Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.

3) An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination.

4) Diagnosis of an organ/space SSI by a surgeon or attending physician

5) **CDC classification**



6) ASA SURGICAL WOUND CLASSIFICATION

Class I/Clean: An uninfected operative wound in which no inflammation is encountered and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow non-penetrating (blunt) trauma should be included in this category if they meet the criteria.

Class II/Clean-Contaminated: An operative wound in which the respiratory, alimentary, genital or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in technique is encountered.

Class III/Contaminated: Open, fresh, accidental wounds. In addition, operations with major breaks in sterile technique (e.g., open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute, non-purulent inflammation is encountered are included in this category.

Class IV/Dirty-Infected: Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation

Determinants of Infection

Despite the fact that every surgical site is contaminated with bacteria by the end of the procedure, few become clinically infected. The interplay of 4 important determinants lead to either uneventful wound healing or SSI:

(1) inoculum of bacteria.

(2) virulence of bacteria.

(3) adjuvant effects of microenvironment .

(4) innate and acquired host defenses.

Inoculum of Bacteria

The variable that has received the greatest amount of attention is the inoculum of bacteria lodged into the wound during the course of the operation. Bacterial contaminants may enter the wound from the air in the OR (operating room), or from the instruments or surgeon(s) that come into contact with the wound. Skin bacteria are always present despite the thoroughness of the preparation of the skin. The largest inoculum of bacteria at the surgical site occurs when the operation involves a body structure that ordinarily is heavily colonized by bacteria, such as the bowel. The distal small intestine and the colon have very large concentrations of bacteria with 10^3–10^4 bacteria/mL of distal small bowel content, $10^5 – 10^6$ bacteria/mL in the right colon, and $10^{10} – 10^{12}$ bacteria/g of stool in the recto sigmoid colon. Substantial numbers of bacteria are also present in the stomach of older patients who have hypo- or achlorhydria. Significant concentrations of bacteria are encountered in the biliary tract when patients are over 70 years of age or have obstructive jaundice, common bile duct stones or acute cholecystitis. Procedures involving the

female genital tract will encounter $10^6 – 10^7$ bacterial/mL. Procedures that enter into the oropharynx, lung, or urinary tract will have significant contaminants depending upon the duration and types of disease that are responsible for the operation. Notably, SSIs are generally the consequence of intra-operative contamination and seldom result from bacterial contamination from distant blood-borne seeding of the wound site during the postoperative period.

Virulence of the Bacterial Contaminant

A second determinant contributing to SSI is the virulence of the bacterial contaminant. The more virulent the bacterial contaminant , the greater the probability of infection. Coagulase-positive staphylococci require a smaller inoculum than the coagulase-negative species. Uncommon but virulent strains of Clostridium perfringens or Group A streptococci require a small inoculum to cause an especially severe necrotizing infection at the surgical site. Escherichia coli have endotoxin in its outer cell membrane that gives it a particular virulence. Bacteroides fragilis and other Bacteroides species are ordinarily organisms of minimal virulence as solitary pathogens, but when combined with other oxygen consuming organisms, they will result in microbial synergism and cause very significant infection following operations of the colon or female genital tract.

The Microenvironment of the Wound

A third variable that determines infection at the surgical site is the microenvironment of the wound. Adjuvant factors that are products or consequences of the surgical procedure any result in clinical infection by otherwise sub-infectious inoculate of bacteria. Hemoglobin at the surgical site is a well known adjuvant substance. It is generally thought that the release of ferric iron during the degradation of red blood

cells stimulates microbial proliferation. Foreign bodies, particularly braided silk and other permanent braided suture materials, similarly harbor microbes and increase the probability of infection. Dead space within the surgical site also provides a local environment that fosters infection.

The integrity of Host Defenses

The fourth determinant of SSI is the integrity of host defenses. Impaired host defenses can be viewed as innate or acquired. Innate impairment refers to the observation that intrinsic responses in some patients are less effective than in others. Variability is regularly found among all patients in various components of neutrophil function and macrophage mediator production.

By contrast, acquired impairment of host responses is clearly related to increased rates of SSI. Shock and hypoxemia are positively associated with SSI, especially in trauma patients. Transfusion appears to be immunosuppressive. Similarly, chronic illnesses, hypo-albuminemia, and malnutrition are significant factors. Hypothermia and hyperglycaemia are also recognized as variables that impair the host response, while corticosteroids and other medications may also adversely affect the host and increase SSI rates.

The Aggregate Effect

When all 4 determinants are evaluated in the aggregate, it becomes apparent that SSI is a very complex biological process and that determination of the causes of an infection in a specific situation can be problematic. The complexity of these individual variables also underscores the variety of issues that must be considered in the development of preventive strategies.

Microbial factors of importance in the development of infection

Size of the inoculum and nature of the microbe

One of the primary determinants of whether infection develops is the size of the microbial inoculums, which or bacteria is expressed in terms of colony forming units (CFU). Two major reservoirs of microbes exist that can form the initial inoculums leading to infection in surgical patients. They are host endogenous micro flora and microbes within the external milieu, which often represents the nosocomial environment for hospitalized individuals.

The critical factor in the development of SSI is the rate at which microbes proliferate in a specific environment. Microbial division is dependent on ambient temperature and oxygen concentration (varying from one microbial species to another), sources of nutrients and inherent properties that determine the maximal division rate under optimal conditions. Pathogenic microbes are those that are capable of causing disease, and those that cause severe infection consistently are termed "virulent". Certain microbes though not inherently virulent, acquire virulence when there is disruption in or suppression in host defenses.

Physiology of wound healing[10,11]

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 haemopoietic system and the 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 wound 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.

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

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 vessels, 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

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

As this topic is centered on surgical sites 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, the 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. The result is a steady, gradual growth in wound tensile strength that continues for 6 to 12 months. However, scar tissue never reaches the tensile strength of unwounded tissue[12].

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[13]

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.

Growth factors and cytokines affecting various steps in wound healing	
Monocyte chemotaxis	PDGF, FGF, TGF-beta
Fibroblast migration	PDGF, EGF, FGF, TGF-beta, TNF, IL-1
Fibroblast proliferation	PDGF, EGF, FGF, TNF
Angiogenesis	VEGF, Angiogenesis, FGF
Collagen synthesis	TGF-beta, PDGF
Collagen secretion	PDGF, FGF, EGF, TNF, (TGF-beta inhibits)

PDGF- platelet derived growth factor, EGF- epidermal growth factor, FGF- fibroblast growth factor, TNF- tumour necrosis factor

Impaired healing[14,15]

Occurs due to many reasons and a wise surgeon recognizes them and attempts a remedy before he wields his scalpel so as to reduce the rate of surgical site infections and help proper wound healing. Of the many causes incriminated in defective wound healing, tissue hypoxia resulting from cardiopulmonary diseases, peripheral vascular 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 including,

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

Role of Oxygen in Wound Healing and Infection

Wound healing process involves numerous functions, many of which depend on presence of oxygen. Collagen production and development, which influences the strength of the wound is directly correlated with partial pressure of oxygen pressure PO_2 of the tissue. Synthesis of collagen, cross-linking and the resulting wound strength is reliable of the normal function of specific enzymes. The functions of these enzymes is directly related to the amount of oxygen present eg., hydroxylation of proline and lysine by hydroxylase enzymes[16].

The production of epithelial tissue depends primarily on the degree of hydration and oxygen. Although a moist wound environment increases the rate of epithelialization by a factor of 2 to 3, the optimal growth of epidermal cells is found at an oxygen concentration of 10% to 50%[16,17]. Hyperbaric oxygen treatment increases the proliferation of the fibroblasts and the differentiation and epidermopoesis of the keratinocytes, but not proliferation of keratinocytes[18].

Oxygen and Tissue Perfusion

Delayed or arrested healing and development of infection result from decreased perfusion and consequently, oxygenation of the tissues. This is most clearly demonstrated in the well-perfused tissue anal region, where the healing normally is good inspite of massive contamination. P_tO_2 is based on the following factors: (1) delivery of oxygen from lungs to tissue (i.e., oxygenation of arterial blood, circulation); (2) transport of oxygen from blood to tissue (i.e., oxygen partial pressure

in blood, the diffusion distance); and (3) oxygen consumption in tissue[19]. At present, P_tO_2 measurement is the best way to observe the oxygen status of the tissue.

Measurement of P_tO_2 can be accomplished by introducing a small oxygen sensor in the tissue. Subcutaneous tissue is the first tissue to suffer from oxygen deprivation and the last to be normalized , for which reason this tissue level is the optimal place for monitoring general tissue perfusion[20]. Clinically, measurement of the blood saturation (Pulse Oximetry) is used routinely. This method, however, reflects primarily the oxygen conditions in the blood, and it only has value in situations where all factors that influence P_tO_2 are functioning optimally.

Effect of Tissue Hypoxia

Impaired perfusion and inadequate oxygenation are the most frequent causes of healing failure. The critical collagen oxygenases involved have km values for oxygen of about 20 mmHg and maximums of about 200 mmHg, means that reaction rate are regulated by paO_2 and blood perfusion throughtout the physiologic range. The paO_2 of wound fluid in human incisions is about 30-40 mmHg, suggesting that these enzymes normally function just beyond haif capacity. Under ideal conditions, wound paO_2 can be raised to above 100 mmHg by improved perefussion and breathing of oxygen[21].

Oxygen and influencing Factors

Internal as well as external factors influence the P_tO_2. In subcutaneous tissue the perfusion is extremely dependent on hemodynamic conditions, cooling, pain, fear, smoking and medical compounds. Many of these factors are encountered during surgery. The postoperative hours, and the late hypoxia related to a decrease in lung capacity is based primarily on reduced function of the diaphragm 2 to 3days postoperatively[22]. Early hypoxemia and reduced tissue perfusion enhance the risk of wound complications. The influence of late hypoxemia, however, is not well understood.

Oxygen and Post Operative Infection

The most frequent complication found in surgical wounds is still infection. Bacteria in the wounds are normally destroyed by intracellular oxidative mechanisms inside the leukocytes and molecular oxygen is necessary for the production of oxygen radicals, especially bactericidal superoxide. The oxygen concentration in the breathing air is directly correlates with the size of the necrosis generated by the injection of bacteria[23]. The critical level for this seems to be below 30 to 40mmHg.

In one third of all wound infections the bacteria found are sensitive to the antibiotic provided during the treatment course. Decreased perfusion may be the reason for this.

Oxygen and Prevention of Infection

While hypoxia may be important in the coagulation process, the presence of oxygen is critical for infection prevention in the inflammatory phase**.** Reactive oxygen species(ROS) play a central role to the prevention of wound infection. After

coagulation begins, neutrophils and monocytes infilterate the wound site and produce ROS in the process of respiratory burst,which is the main defense against wound infection[24].

Role of oxygen in wound healing at different levels

At Cellular Level

At cellular level oxygen is an essential nutrient for cell metabolism, especially energy production, this energy is supplied by ATP,which is the most important store of chemical energy on molecular level and is synthesized in mitochondria by oxidative phosphorylation.This reaction is obligatory oxygen dependent and cannot take place without oxygen.During the inflammatory phase of the healing process NADPH-linked oxygenase produces high amount of oxygen. Succesfull wound healing can only take place in the presence of the enzyme, because oxidants are required for prevention of wound healing

Recent discoveries have illuminated that not only phagocytes, but almost each and every cell in the wound environment is fitted with a specialized enzyme to convert oxygen to reactive oxygen species(ROS), including oxidizing species such as free radicals and hydrogen peroxide. These ROS contribute as a cellular messengers to promote several important processes that support wound healing. Thus oxygen has a role in healing beyond its function as nutrient and antibiotic.

At Tissue Level

At tissue level oxygen beyond nutritional support has several other effects. Angiogenesis is a critical early aspect in wound healing. Hypoxia can initiate neovascularisation, but cannot sustain it. Supplementary oxygen administered accelerates vessels growth. Collagen production, deposition and development of strength of wound healing is directly correlated to the partial pressure of oxygen in the tissues.

Recently it has been shown that oxygen may also trigger the differentiation of fibroblasts to myofibroblasts, cells responsible for wound contraction.The production of epithelial tissue is primarily dependable on the degree of hydration and oxygen. A moist wound environment increases the rate of epithelisation, the optimal growth of epidermal cells is found at an oxygen concentration of 10-50%.

Clinical Practice

Oxygen has through long time being used for increasing the wound healing in the daily clinical work. Two application forms have been used: Topical/Local application of pure oxygen and systemically application of pure oxygen either at normal pressure or as hyperbaric oxygen[3].

Topical/Local application of oxygen on the wound surface has been used to increase regeneration of epithelium. This oxygen does not diffuse into deeper tissue but may have a advantageous potential to oxygenate superficial area of wound.

Systemic application of oxygen through the lung and cardiovascular system is known to improve wound healing and decrease the risk of infection. Supplementary

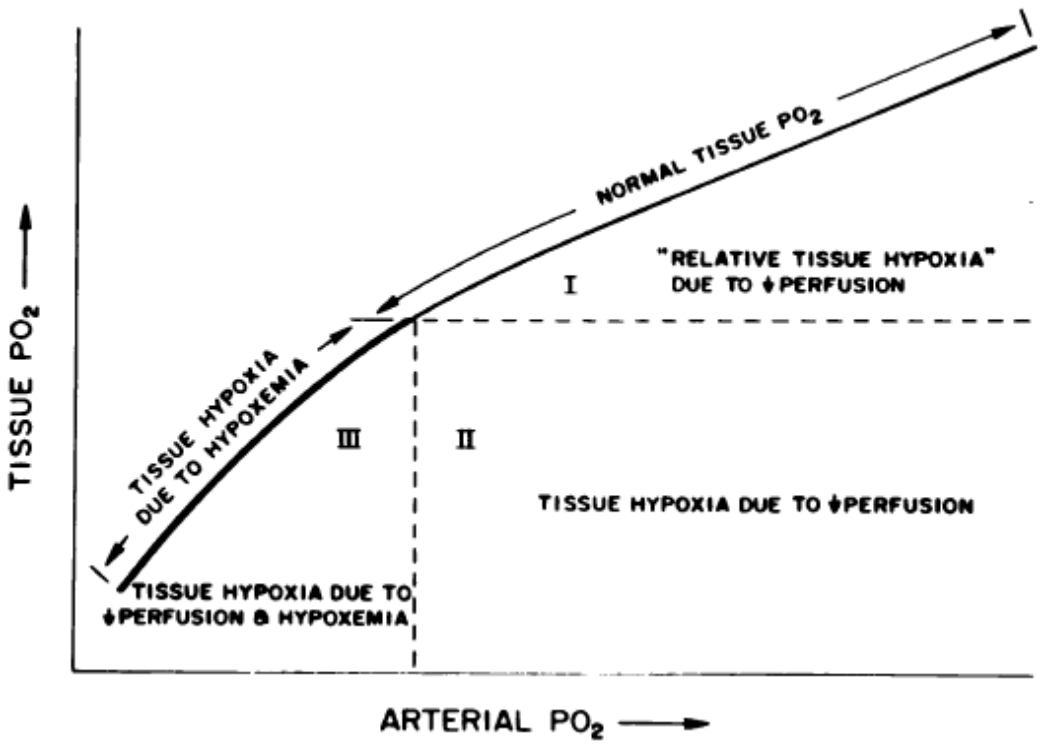
oxygen administered to the breathing air the first two post operative days has decreased the wound infection rate in colorectal patients. It has been shown that the same types of patients benefits of as little as two hours postoperative oxygen supplement administered by a mask. An oxygen concentration of 80% decreased the wound infection rate to half compared to the oxygen concentration of 30%.Oxygen administered during and two hours after surgery is taking place at the operation theatre and the recovery room. This means than it is an easy, cheap and useful way to decrease the infection rate in surgical patients. Furthermore this treatment has shown in no case to develop side effects.

While the local hypoxia and bacterial contamination primarily are dependable of the surgeon, the oxygenation of the patient mainly is based on the anaesthetiological expertise. Oxygen treatment and monitoring of oxygen tension and saturation should be schematized and used in tight collaboration between these groups[2].

Blood transports many substances to and from the tissues. Oxygen is the most pressingly necessary, the most easily measured at tissue level, and the most representative of efficacy of tissue perfusion.

Initially, wound $P0_2$ is high and approaches arterial $P0_2$ because of fresh trauma and bleeding into the wound. Wound $P0_2$ declines as vessels thrombose and leukocytes and fibroblasts, which consume oxygen, increase in number. The lower oxygen tension in a mastectomy wound compared with a minor needle wound in the subcutaneous tissue probably results from a greater degree of disruption of the blood supply and the accumulation of a larger

population of oxygen-consuming cells. A striking finding is the depression of wound-tissue $P0_2$ in the first few days after major operations. The extent and duration of tissue hypoxia depended on the magnitude of the operation. On the day of operation, PtO_2 of all postoperative patients was significantly lower than nonoperated controls, and tissue hypoxia was most severe immediately after operation, recovering to more "normal" values on POD 1. Tissue hypoxia persisted throughout the duration of the study in cardiovascular patients. One of the determinants of tissue $P0_2$ is arterial $P0_2$. Many experiments in animals and man demonstrate that PtO_2 is dependent on PaO_2. Classic teaching of the physiology of oxygen transport states that little improvement in tissue oxygen supply can be obtained by the addition of oxygen to the blood over and above that sufficient to saturate hemoglobin. This is usually taken to mean that elevating $P0_2$ in normal tissue by increasing inspired oxygen concentration has no functional significance. This tenet ignores the well documented effect of increasing environmental $P0_2$ on cells in areas of injury.



Schematic representation of "optimum" $Pt0_2$-PaO_2 curve and classification of tissue hypoxia according to various causes. The dotted horizontal line indicates level below which tissue hypoxia exists. Normal tissue oxygenation is represented by the portion of the curve above the horizontal line. Tissue hypoxia due to hypoxemia alone is indicated by the portion of the curve below the horizontal line. Zone 1 indicates "relative tissue hypoxia" due to hypopersion. Zone II indicates tissuehypoxia due to hypoperfusion. Zone III indicates tissue hypoxia due to combined hypoxemia and hypoperfusion.

The question of clinical importance is whether attempts to maximize tissue oxygen tension and perfusion provide any benefit to patients. Considerable data suggest that there is benefit to be obtained.

Oxygen plays an important role in both wound healing and host resistance to microbial contamination. Collagen synthesis in animal wounds varies in proportionto inspired oxygen concentration, blood oxygen tension, and wound $P0_2$. Hyperoxia leads to increased collagen synthesis, while hypoxia has the opposite effect. A similar relationship has been observed in wound tensile strength. Hyperoxemia promotes epithelization and wound angiogenesis.

Microbicidal function of leukocytes is oxygen-dependent,especially in the range of $P0_2$ found in wounds. Derivatives of molecular oxygen such as peroxide and superoxide participate in bacteria-killing. The availability of these derivatives is $P0_2$-dependent. Clearance of bacteria incubated with leukocytes in vitro and elimination of bacteria from experimental wounds in vivo are significantly impaired under hypoxic conditions and are increased in hyperoxic condition. Intradermal injection of bacteria results in a larger area of skin necrosis in animals kept in hypoxic conditions, while hyperoxia has an opposite and beneficial effect. These data suggest strongly that infection rate and severity can be significantly reduced by maximization of tissue and wound oxygen tension and perfusion[25].

Mode of Delivery of supplemental oxygen

There are number of devices through which supplemental oxygen can be delivered .A nasal cannula which can provide oxygen at low flow rates, 2-6 litres per minute, delivering a concentration of 24-40%. A face mask often used for controlled

air-entrainment known as venturi mask at 6 and 12 litres per minute, can provide concentration between 28-60%[26].

In our study we used venture mask for oxygen supplementation with concentration of 60% and 30% respectively for study and control group.

Greif R et al in their study (From July 1996 to October 1998) total 500 patients, among 500 patients 250 received 80% oxygen and 250 received 30% oxygen. Among 250 patients who received 80% oxygen 13 had surgical wound infection, as compared with 28 of the 250 patients given 30% oxygen[27].

F. Javier et al in their study SSI infection occurred in 35 patients (24.4%) administered 30% FIO_2 in 22 (14.9%) administered 80% FIO_2 (p= .04%). The risk of SSI was 39% lower in the 80% FIO_2 group[28].

Al Niaimi et al in their study supplemental perioperative oxygenation resulted in a reduced incidence of SSI {Relative Risk 0.070 (95% CI 0.52-0.94), p = 0.01}, using a fixed effects model. Thus they concluded that supplemental perioperative oxygenation is beneficial in preventing SSI in patients undergoing colorectal surgery[29].

Motaz Qadan et al in their study, (1998-2007) they observed infection rates of 12% in the control group and 9% in the hyperoxic group, with Relative Risk reduction of 25.3%[30].

John P. Kirby and John E. Mazuski in their article concluded SSI is an important postoperative complication. It is second only to urinary tract infection as the most common nosocomial infection in hospitalized patients. Based on extensive epidemiologic surveys, it has been estimated that SSI develops in at least 2% of hospitalized patients undergoing operative procedures[31].

Pryor *et al.* In their included 160 patients, half received 80% oxygen and half received 30% oxygen. The study population included patients undergoing colorectal surgery and general surgical procedures. They observed infection rates ,14 of 57 patients of (24.4%) in the 80% group and 9 of 51 in the 30%oxygen group (RR 1.39, 95% CI 1.04–2.32). This is the only study that found a higher rate of SSI in patients receiving 80% FiO_2[32].

Belda *et al* .In their large, multi-centre study in Spain 291 patients were included in the analysis, 148 patients received 80% oxygen, and 143 patients received 30% oxygen, observed that patients receiving supplemental oxygen had a significant reduction in the risk of wound infection[33].

METHODOLOGY

SOURCE OF DATA

Patients admitted as in patients in B.L.D.E.U's Shri B. M. Patil Medical College Hospital for Class I (clean) and Class II (clean contaminated) elective general surgeries between October 2009 and May 2011

Samplesize: With the incidence rate of 2% and at ±2 margin of error and 95% level of confidence, the calculated sample size is 188.

Clean surgeries – 94

Clean contaminated surgeries – 94

INCLUSION CRITERIA

Patients who underwent Class I (clean) and Class II (clean contaminated) elective general surgeries admitted under the department of surgery in BLDEU's Shri B. M. Patil Medical College Hospital and Research Center, Bijapur

EXCLUSION CRITERIA:

- Patients with implants or prosthetic material
- Patients with Diabetes mellitus
- Patients on steroids, chemotherapy or immuno-suppression
- Patients with renal and respiratory disease

METHOD OF COLLECTION OF DATA:

Details of cases will be recorded including history and clinical examination. Routine pre-operative investigations performed in both the groups.

The study group received 60% fraction of inspired oxygen intra operatively and for 2 hours after surgery. The control group receive d 30% fraction of inspired oxygen intraoperatively and for 2 hours after surgery.

Operative wound examined on the second, fifth and eighth post-operative day for signs of surgical site infection.

Patients from both the study and control groups were compared for final analyses.

All the patients received single dose of prophylactic antibiotic, that is 3rd generation cephalosporin (CEFTRIAXONE). Hypovolemia correction and Normothermia will be maintained.

Statistical analysis

- Diagrammatic presentation
- Mean +/- SD
- Z test

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RESULTS

The study was conducted on a total of 188 patients aged between 1-90, of which 94 underwent clean general surgical procedures and 94 underwent clean contaminated general surgical procedures in BLDEU's Sri B. M. Patil Medical College and Research Hospital, Bijapur from October 2009 to May 2011.

Among the 94 clean surgical cases, 47 received 60% fraction of inspired oxygen intraoperatively and for 2 hours after surgery and 47 received 30% fraction of inspired oxygen intraoperatively and for 2 hours after surgery.

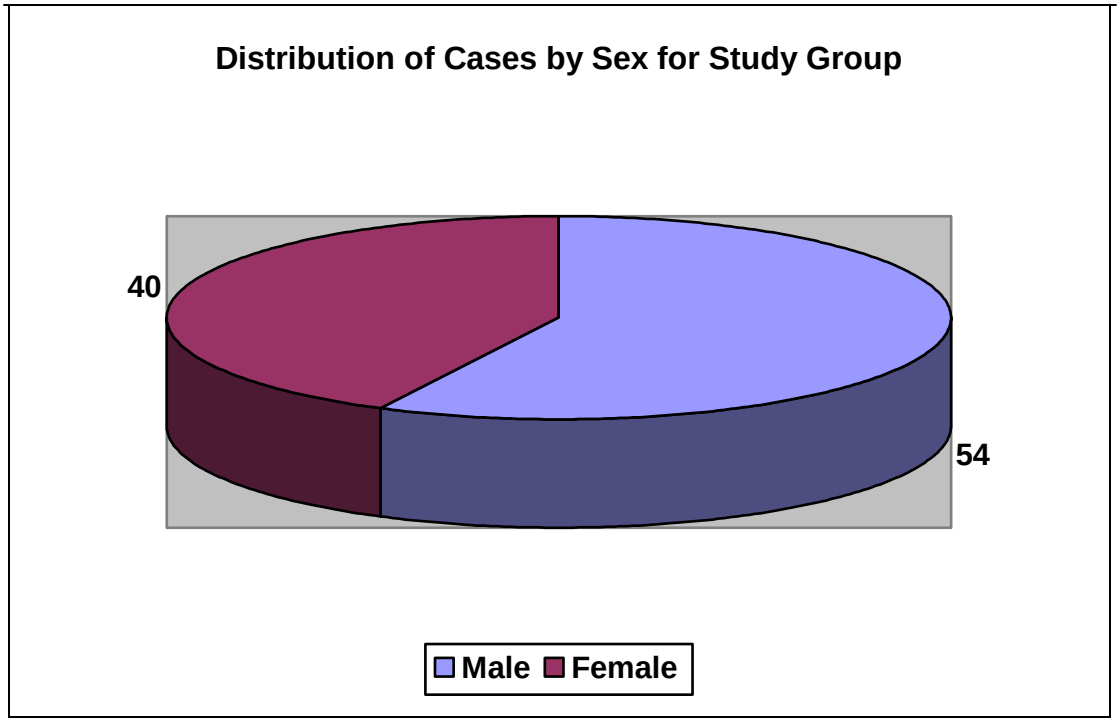
Among the 94 clean-contaminated surgical cases, 47 received 60% fraction of inspired oxygen intraoperatively and for 2 hours after surgery and 47 received 30% fraction of inspired oxygen intraoperatively and for 2 hours after surgery.

SEX DISTRIBUTION

Table 1

Sex distribution in Study group

Sex	Number	Percentage (%)
Male	54	56.98%
Female	40	43.02%

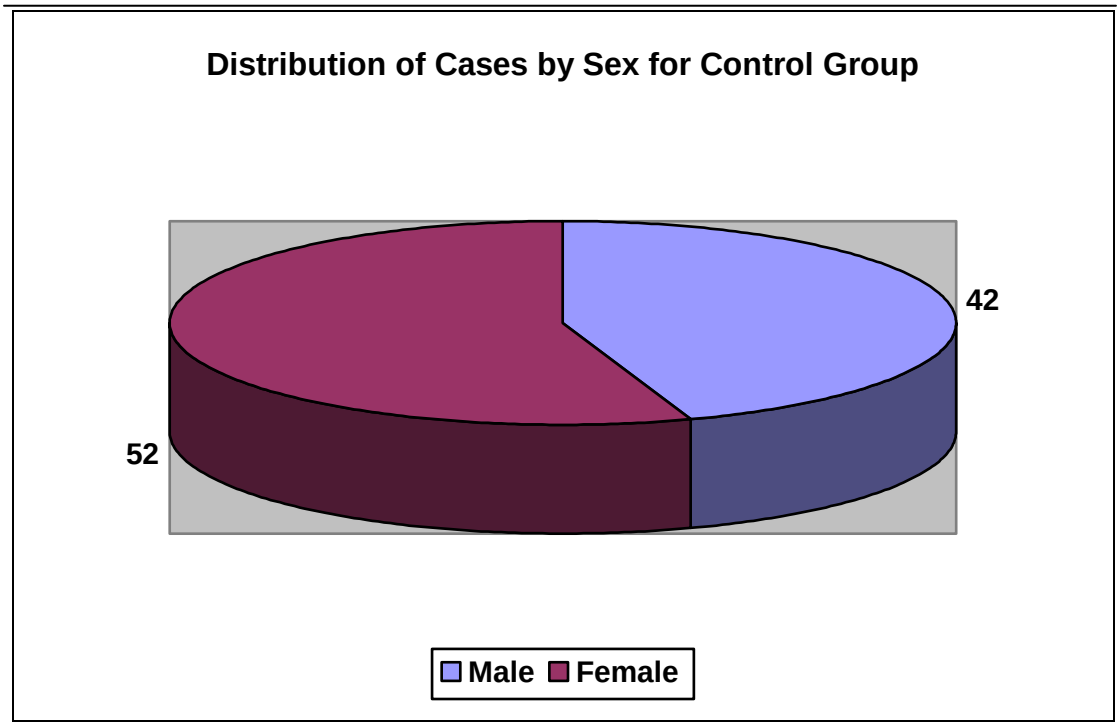


Of the 94 cases study group 56.98% were males and 43.02% were females.

Table 2

Sex distribution in Control group

Sex	Number	Percentage (%)
Male	42	42.10%
Female	52	57.90%



Of the 94 cases of control group 42.10% were males and 57.90% were females.

AGE DISTRIBUTION

Table 3

Distribution of cases by Age for Study Group

Age(yrs)	<10	11-20	21-30	31-40	41-50	51-60	61-70	>71
Total no.	4	12	25	21	12	8	8	4

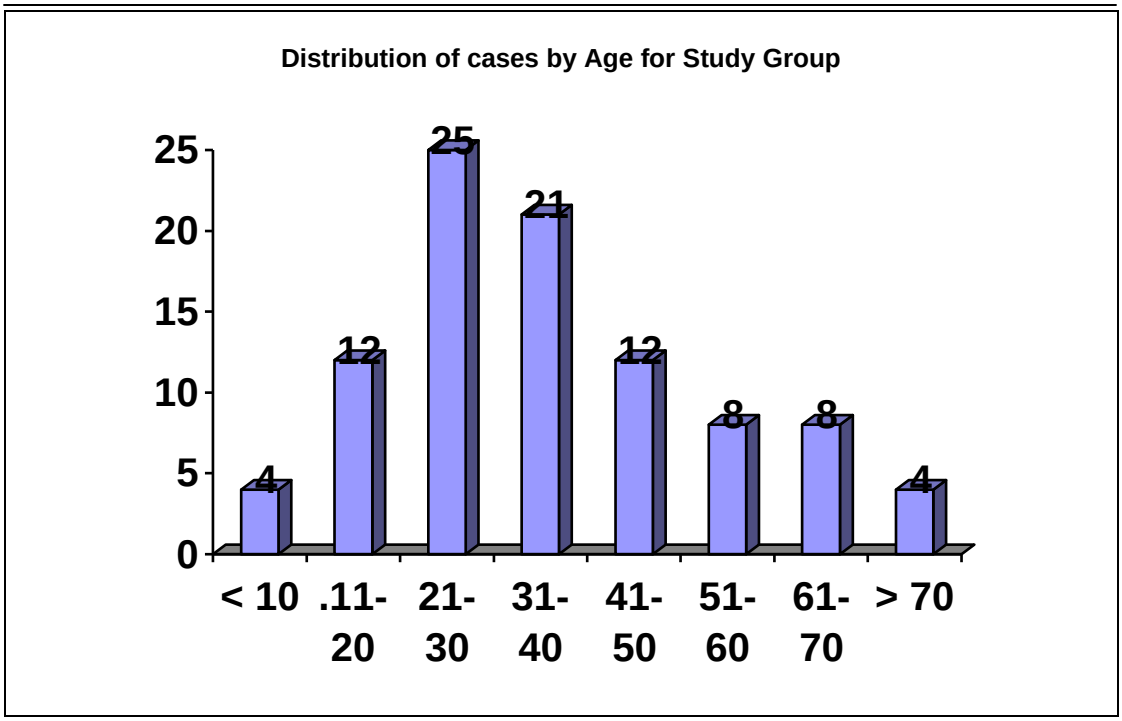
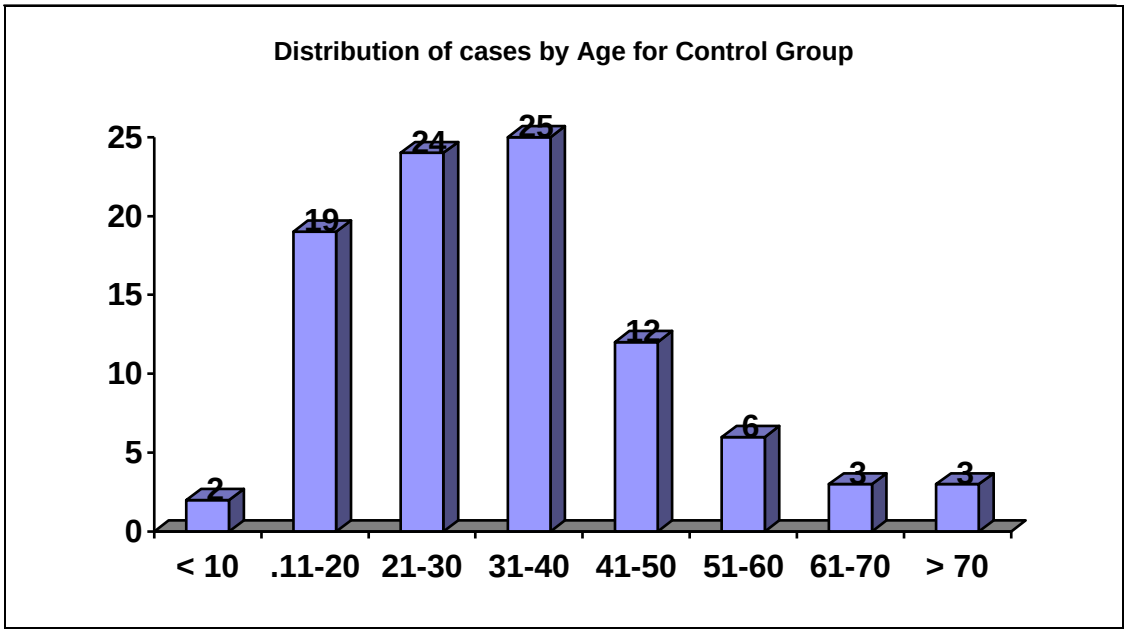


Table 4

Distribution of cases by Age for Control Group

Age(yrs)	<10	11-20	21-30	31-40	41-50	51-60	61-70	>71
Control Group	2	19	24	25	12	6	3	3



Among the patients who received 60% fraction of inspired oxygen perioperatively, the age varied from 1-88 years. The number of patients in the 21-30 years group was highest.

Among the patients who received30% fraction of inspired oxygen perioperatively, the age varied from 1-80 years. The number of patients in the 31-40 years group was the highest.

Table 5

Results in class I group

O_2 supplementation	**SSI**	**NO SSI**	**PERCENTAGE (%)**
60% Inspired oxygen	3	44	6.4%
30%Inspired Oxygen	5	42	10.6%

94 patients who underwent class I surgeries, 47 patients of study group , 3 patients developed features of SSI .

3 (seroma collection with tenderness)

Of the 47 class I surgery in control group, 5 developed features of SSI

3 (seroma collection with tenderness)

2 (frank purulent discharge)

Table 6

Results in class II group

O_2 supplementation	**SSI**	**NO SSI**	**PERCENTAGE (%)**
60% Inspired oxygen	5	42	10.6%
30% Inspired oxygen	8	39	17%

Of the 94 who underwent class II general surgical procedures, 47 patients with study group. 5 patients developed features of SSI .

2 (seroma collection at the incisional site)

1 (erythema and tenderness)

2 (frank purulent discharge)

In 47 patients of control group, 8 developed features of SSI .

6 (seroma collection at the incisional site)

2 (frank purulent discharge)

Table 7

Overall Result of Cases by SSI for Study and Control Group

SSI	Study Group	Control Group	Total
	Oxygen 60 %	Oxygen 30 %	
Yes	8	13	21
No	86	81	167
Total	94	94	188

Thus it was seen that the 8 out of the 94 patients who received 60% fraction of inspired oxygen perioperatively developed surgical site infections and 13 of the 94 patients who received 30% fraction of inspired oxygen perioperatively developed surgical site infections.

Chi-Square Test :

Chi-Square = N(ad-bc)2 / [(a+b)(c+d)(a+c)(c+d)] = 1.34

P value$\geq$ 0.01

DISCUSSION

Surgical site infection is a major complication of surgery, associated with prolonged hospitalization, increased costs and excess mortality. In recent years, randomized trials have identified a number of preventive measures that can substantially reduce the risk of SSI. These include appropriate perioperative antibiotic prophylaxis, maintenance of perioperative normothermia and control of hyperglycemia. The effect of perioperative oxygen supplementation continues to be under debate with proponents and opponents firmly divided over the issue. It has been argued by some researchers that there is benefit in reducing surgical site infection.

Achieving high oxygen tension at the site of surgery has been proposed as a means of reducing the risk of SSI, based on data that oxygen can enhance the oxidative processes in white cells, thus facilitating bacterial killing by oxidative phosphorylation. Thus oxygen plays an important role in both wound healing and host resistance to microbial contamination

Several studies conducted by, Belda *et* al[27], Motaz Qadan et al[30] , Al Niaimi et al[29], showed that supplemental perioperative oxygenation is beneficial in preventing SSI in surgical patients.

Greif R et al in their study (From July 1996 to October 1998) total 500 patients, among 500 patients 250 received 80% oxygen and 250 received 30% oxygen. Among 250 patients who received 80% oxygen 13 had surgical wound infection, as compared with 28 of the 250 patients given 30% oxygen[27].

F. Javier et al in their study SSI infection occurred in 35 patients (24.4%) administered 30% FIO_2 in 22 (14.9%) administered 80% FIO_2 (p= .04%). The risk of SSI was 39% lower in the 80% FIO_2 group[28].

Al Niaimi et al in their study supplemental perioperative oxygenation resulted in a reduced incidence of SSI {Relative Risk 0.070 (95% CI 0.52-0.94), p = 0.01}, using a fixed effects model. Thus they concluded that supplemental perioperative oxygenation is beneficial in preventing SSI in patients undergoing colorectal surgery[29].

Motaz Qadan et al in their study, (1998-2007) they observed infection rates of 12% in the control group and 9% in the hyperoxic group, with Relative Risk reduction of 25.3%[30].

Pryor *et al* ,In their study found a higher rate of SSI in patients receiving higher concentration of supplemental oxygen[32].

The present study had infection rates 8 and 13 respectively in patient who recived 60% and 30% perioperative supplemental oxygen. The p value is 1.34 greater than tabled value of chi-square at 1% (p value$\geq$ 0.01)level of significance, hence there is an association between level of oxygen and the occurrence of SSI. In other words greater the amount of supplemental oxygen lesser the chance of SSI.

STUDY	PERCENTAGE OF SSI	P VALUE
Greif *et al.*	Control group-11.2% Study group –5.2%	0.46
Mayzler *et al*	Control group – 15.7% Study group – 10.5%	0.67
Belda *et al.*	Control group – 24.4% Study group–14.9%	0.61
PRESENT STUDY	Control group - 8.5% Study Group - 13.8%	1.34

CONCLUSION

Our study shows that supplementation of higher concentration of oxygen in clean and clean contaminated surgeries is effective in preventing post-operative surgical site infection.

Since chi-square calculated is greater than tabled value of chi-square at 1% level of significance, hence there is an association between level of oxygen and the occurrence of SSI. In other words greater the amount of supplemental oxygen lesser the chance of SSI.

Thus it can be concluded from this study that supplementation of higher concentration of oxygen plays an important role in reducing surgical site infection in clean and clean contaminated surgeries.

SUMMARY

The study was conducted on 188 patients who underwent either clean or clean contaminated elective general surgical procedures at Sri. B. M. Patil Medical College Hospital, between October 2009 and May 2011.

94 of whom received 60% perioperative supplemental oxygen and two hours after surgery, 94 received 30% perioperative supplemental oxygen and two hours after surgery.

Occurrence of post-operative wound infection was noted in both the groups, but with a higher level of incidence in control group.

Statistical analysis was done accordingly, P value$\geq$ 0.01 was considered significant.

On analysis since chi-square calculated is greater than tabled value of chi-square at 1% level of significance, hence there is an association between level of oxygen and the occurrence of SSI. In other words greater the amount of supplemental oxygen lesser the chance of SSI.

In summary, the administration of supplemental oxygen during surgery and for two hours afterward almost halved the incidence of surgical site infection. Because the cost of and risk associated with supplemental perioperative oxygen are trivial , the provision of supplemental oxygen appears to be a practical method of reducing the incidence of this dangerous and expensive complication.

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ANNEXURES

SAMPLE INFORMED CONSENT FROM BLDEA'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPIT ANNEXURES

SAMPLE INFORMED CONSENT FROM BLDEA'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, BIJAPUR-586103

TITLE OF THE PROJECT -ROLE OF 30% VERSUS 60% PERIOPERATIVE OXYGEN SUPPLEMENTATION IN REDUCING SURGICAL SITE INFECTION.

PRINCIPAL INVESTIGATOR - DR. UDAY G. KARJOL

GUIDE - DR. (MRS) TEJASWINI UDACHAN
PROFESSOR OF SURGERY

CO-GUIDE -Dr. VIJAYKUMAR T.KALYANAPPAGOL
PROFESSOR OF ANAESTHISIOLOGY

PURPOSE OF RESEARCH:

I have been informed that this study is a comparison of role of 30% versus 60% perioperative oxygen supplementation in reducing surgical site infection.in clean and clean contaminated general surgery cases. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I 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 that are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help to compare the use of 30% versus 60% perioperative oxygen supplementation in clean and clean contaminated surgeries.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. 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 location.

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

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime. Dr. Uday G. Karjol 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 the study, which might influence my continued participation.

If during the 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. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that 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. Uday G. Karjol may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would 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 the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Dr. Uday G. Karjol Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr.Uday G. Karjol has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____ Witness to

signature Date

SCHEME OF CASE TAKING:

1) Name:	CASE NO:
2) Age:	IP NO:
3) Sex:	DOA:
4) Religion:	DOS:
5) Occupation:	DOD:

6) Residence:

7) Chief complaints

8) Provisional diagnosis

9) Past History:

1. Diabetes mellitus
2. Hypertension
3. History of any drug intake

10) General Physical Examination

Pallor	present/absent
Icterus	present/absent
Clubbing	present/absent
Generalized Lymphadenopathy	present/absent
Build	Poor/Middle /Well
Nourishment	Poor / Middle / Well

11) Vitals

PR:

BP:

RR:

Temp:

Weight:

12) Other Systemic Examination:

i. Respiratory System

ii. Cardiovascular System

iii. Central Nervous System

13) Investigation:

Blood: Hb

TC

DC

ESR

BT

CT

BLOOD UREA

SERUM CREATININE

RBS

Chest X-ray whenever necessary

Absolute blood gas analysis whenever necessary

Urine: Albumin, Sugar, Micro

14) Final Diagnosis:

15) Details of Surgery:

Diagnosis	Operative Procedure	Duration of Procedure

16) Follow up:

- Local examination of wound

	POD2	POD5	POD8
Clean			
Seroma			
Edema			
Erythema			
Tenderness			
Abscess			
Pus discharge			
Gaping of wound			

Pus culture and sensitivity: positive negative

Resistance to the antibiotic used: yes no

17) Inference:

18) Comments:

MASTER CHART

SL NO	IP NO	NAME	AGE	SEX	DIAGNOSIS	PROCEDURE	DURATION	CLASS	ANTIBIOTIC	OXYGEN	SSI
1	19482	Jayashree	18	F	FB granuloma	Excision	20 mins	I	Prophylactic	30%	N
2	2682	Bellen siddapa	11	M	Cervical LN	Biopsy	15 mins	I	Prophylactic	60%	Y
3	3393	Mahananda	32	F	Fibroadenoma	Excision	30 mins	I	Prophylactic	30%	N
4	3447	Sunanda	28	F	Cervical LN	Biopsy	15 mins	I	Prophylactic	60%	N
5	2681	Mallawa	40	F	Cholelitiasis	Lap cholecystectomy	1 hr 10 mir	I	Prophylactic	30%	N
6	14291	Ramzan	18	M	Congenital hernia	Herniotomy	1 hr	I	Prophylactic	60%	N
7	14308	Mahadev	42	M	Lipoma	Excision	15 mins	I	Prophylactic	30%	Y
8	17396	Manjula	32	F	Cholelitiasis	Lap cholecystectomy	1 hr 10 mir	I	Prophylactic	60%	N
9	18735	Yallappa	38	M	Lipoma	Excision	30 mins	I	Prophylactic	30%	N
10	18737	Sulthan	28	M	Sebaceous cyst	Excision	15 mins	I	Prophylactic	60%	N
11	26748	Reshma	20	F	Fibroadenoma	Excision	30 mins	I	Prophylactic	30%	Y
12	19499	Raju	32	M	Umbilical hernia	Anatomical repair	1 hr	I	Prophylactic	60%	N
13	253	Udupirao	57	M	Cholelitiasis	Lap cholecystectomy	1 hr 35 mir	I	Prophylactic	30%	N
14	856	Shivakantamma	60	F	Cholelitiasis	Lap cholecystectomy	1 hr 45 mir	I	Prophylactic	60%	N
15	976	Halemma	45	F	Sebaceous cyst	Excision	30 mins	I	Prophylactic	30%	Y
16	1490	Devabai	35	F	Multinodular goitre	Hemithyroidectomy	1 hr	I	Prophylactic	60%	N
17	1748	Gangamma	60	F	Cholelitiasis	Lap cholecystectomy	1 hr 30 mir	I	Prophylactic	30%	N
18	1787	Dangegal	42	M	Lipoma	Excision	25 mins	I	Prophylactic	60%	N
19	1786	Magati	29	F	Lipoma	Excision	30 mins	I	Prophylactic	30%	N
20	2165	Shantamma	35	F	Multinodular goitre	Hemithyroidectomy	1 hr 30 mir	I	Prophylactic	60%	N
21	2171	Parvati	24	F	Fibroadenoma	Excision	25 mins	I	Prophylactic	30%	N
22	2205	Mallapa	25	M	Lipoma	Excision	10 mins	I	Prophylactic	60%	Y
23	2670	Chennamma	35	F	Fibroadenoma	Excision	25 mins	I	Prophylactic	30%	N
24	2632	Girimallappa	73	M	Lipoma	Excision	15 mins	I	Prophylactic	60%	N
25	3780	Suvarna	50	F	Cholelitiasis	Lap cholecystectomy	1 hr 45 mir	I	Prophylactic	30%	N
26	4056	Ramzan	45	M	Sebaceous cyst	Excision	10 mins	I	Prophylactic	60%	N
27	4706	Geeta	25	F	Fibroadenoma	Excision	10 mins	I	Prophylactic	30%	N
28	5588	Dr.Yarnal	54	M	Cholelitiasis	Lap cholecystectomy	1 hr 45 mir	I	Prophylactic	60%	N
29	6140	Yamunabai	30	F	Cholelitiasis	Lap cholecystectomy	2 hrs 15 mi	I	Prophylactic	30%	N
30	6283	Peersab	67	M	Cholelitiasis	Lap cholecystectomy	1 hr 45 mir	I	Prophylactic	60%	N
31	6793	Shobha	37	F	Cholelitiasis	Lap cholecystectomy	1 hr 30 mir	I	Prophylactic	30%	N
32	6833	Sanket	11	M	Congenital hernia	Herniotomy	30 mins	I	Prophylactic	60%	N
33	7025	Manisha	40	F	Cholelitiasis	Lap cholecystectomy	1 hr 15 mir	I	Prophylactic	30%	N
34	7569	Ganga	52	F	Cholelitiasis	Lap cholecystectomy	2 hrs 15 mi	I	Prophylactic	60%	N
35	8103	Mallappa	19	M	Umbilical hernia	Herniotomy	50 mins	I	Prophylactic	30%	N
36	8489	Rubina	16	F	Cervical LN	Biopsy	20 mins	I	Prophylactic	60%	N
37	9197	Riyaz	12	M	Dermoid cyst	Excision	15 mins	I	Prophylactic	30%	N
38	8975	Hanammawwa	25	F	Fibroadenoma	Excision	35 mins	I	Prophylactic	60%	N
39	9188	Siddama	28	F	Fibroadenoma	Excision	35 mins	I	Prophylactic	30%	N
40	9799	Meenaxi	35	F	Pre auricular LN	Excision	15 mins	I	Prophylactic	60%	N
41	8892	Bhemmawwa	30	F	Inguinal LN	Biopsy	30 mins	I	Prophylactic	30%	N
42	9741	Shekubai	37	F	Lipoma	Excision	15 mins	I	Prophylactic	60%	N
43	10335	Jyothi	20	F	Multinodular goitre	Hemithyroidectomy	1 hr 45 mir	I	Prophylactic	30%	Y
44	10499	Nagamma	10	F	Lipoma	Excision	20 mins	I	Prophylactic	60%	N
45	11026	Aiyamma	28	F	Ca breast	MRM	3 hrs	I	Prophylactic	30%	N
46	11829	Sasubai	40	F	Sebaceous cyst	Excision	25 mins	I	Prophylactic	60%	N
47	11647	Bassamma	36	F	Fibroadenoma	Excision	10 mins	I	Prophylactic	30%	N
48	12352	Kallappa	14	M	Dermoid cyst	Excision	25 mins	I	Prophylactic	60%	N
49	4877	Shivlingappa	80	M	Cholelitiasis	Lap cholecystectomy	2 hrs 15 mi	I	Prophylactic	30%	N
50	4685	Manikyamma	55	F	Cholelitiasis	Lap cholecystectomy	1 hr 15 mir	I	Prophylactic	60%	N
51	22587	Shankrappa	40	M	Dermoid cyst	Excision	20 mins	I	Prophylactic	30%	N
52	23574	Karishma	72	F	Cholelitiasis	Lap cholecystectomy	1 hr 10 mir	I	Prophylactic	60%	N
53	24283	Ayesha	16	F	Fibroadenoma	Excision	15 mins	I	Prophylactic	30%	N
54	26787	Shankargouda	27	M	Lipoma	Excision	20 mins	I	Prophylactic	60%	N
55	28297	Shantawwa	70	F	Cholelitiasis	Lap cholecystectomy	1hr 15 min	I	Prophylactic	30%	N
56	28672	Sneha Almel	20	F	Fibroadenoma	Excision	15 mins	I	Prophylactic	60%	N
57	1115	Mallawa	35	F	Fibroadenoma	Excision	15 mins	I	Prophylactic	30%	N
58	1127	Mallawa	70	F	Lipoma	Excision	10 mins	I	Prophylactic	60%	N
59	2043	Yallawwa	24	F	Fibroadenoma	Excision	30 mins	I	Prophylactic	30%	N
60	1884	Kallawwa	35	F	Dermoid cyst	Excision	10 mins	I	Prophylactic	60%	N
61	3808	Neela	40	F	Multinodular goitre	Hemithyroidectomy	2 hrs 15 mi	I	Prophylactic	30%	Y
62	3766	Sachin	1	M	Cervical LN	Biopsy	30 mins	I	Prophylactic	60%	N
63	5747	Chandrakanta	62	F	Lipoma	Excision	15 mins	I	Prophylactic	30%	N
64	5788	Sangayya	25	M	Multinodular goitre	Thyroidectomy	2 hrs 15 mi	I	Prophylactic	60%	N
65	72191	Sarojini	32	F	Sebaceous cyst	Excision	15 mins	I	Prophylactic	30%	N
66	7299	Shantabai	31	F	Fibroadenoma	Excision	15 mins	I	Prophylactic	60%	N
67	13084	Madhumati	34	F	Multinodular goitre	Hemithyroidectomy	1 hr 45 mir	I	Prophylactic	30%	N
68	13097	Iramma	36	F	Ca breast	MRM	3 hrs	I	Prophylactic	60%	N
69	13474	Deepa	15	F	Ganglion	Excision	30 mins	I	Prophylactic	30%	N
70	13467	Muttanna	15	M	Cervical LN	Biopsy	20 mins	I	Prophylactic	60%	N
71	13464	Sudharani	16	F	Cholelitiasis	Lap cholecystectomy	1hr 15 min	I	Prophylactic	30%	N
72	14161	Ramabai	42	F	Multinodular goitre	Hemithyroidectomy	1 hr 45 mir	I	Prophylactic	60%	Y
73	14568	Savithri	48	F	Cholelitiasis	Lap cholecystectomy	1 hr 35 mir	I	Prophylactic	30%	N
74	14981	Shantawwa	65	F	Cholelitiasis	Lap cholecystectomy	2 hr 10 mir	I	Prophylactic	60%	N
75	15112	Hanumanth	55	M	Cholelitiasis	Lap cholecystectomy	1 hr 45 mir	I	Prophylactic	30%	N
76	15105	Gurunath	67	M	Cholelitiasis	Lap cholecystectomy	1 hr 35 mir	I	Prophylactic	60%	N

77	15887	Kasturi	45	F	Multinodular goitre	Hemithyroidectomy	1 hr 45 mir	I	Prophylactic	30%	N
78	15217	Maulanbi	50	F	Multinodular goitre	Hemithyroidectomy	2 hr 10 mir	I	Prophylactic	60%	N
79	15794	Mallappa	10	M	Cervical LN	Biopsy	25 mins	I	Prophylactic	30%	N
80	15809	Ameensab	45	M	Sebaceous cyst	Excision	20 mins	I	Prophylactic	60%	N
81	172742	Sheevaleela	27	F	Fibroadenoma	Excision	25 mins	I	Prophylactic	30%	N
82	15833	Gourawwa	28	F	Cholelitiasis	Lap cholecystectomy	1 hr 35 mir	I	Prophylactic	60%	N
83	16367	Shruthi	18	F	Fibroadenoma	Excision	20 mins	I	Prophylactic	30%	N
84	16734	Manoj	24	M	Dermoid cyst	Excision	30 mins	I	Prophylactic	60%	N
85	18444	Krishna prasad	14	M	Tongue Tie	Excision	30 mins	I	Prophylactic	30%	N
86	18450	Kazisab	64	M	Dermoid cyst	Excision	20 mins	I	Prophylactic	60%	N
87	216813	Maruthi	42	M	Dermoid cyst	Excision	10 mins	I	Prophylactic	30%	N
88	21343	Premabai	58	F	Lipoma	Excision	20 mins	I	Prophylactic	60%	N
89	21238	Sunanda	28	F	Fibroadenoma	Excision	25 mins	I	Prophylactic	30%	N
90	21758	Manjunath	22	M	Cholelitiasis	Lap cholecystectomy	1 hr 30 mir	I	Prophylactic	60%	N
91	7764	Govindrao	34	M	Multinodular goitre	Thyroidectomy	1 hr 45 mir	I	Prophylactic	30%	N
92	9237	Vanita	14	F	Cholelitiasis	Lap cholecystectomy	1 hr 35 mir	I	Prophylactic	60%	N
93	9841	Mahadevi	34	F	Fibroadenoma	Excision	25 mins	I	Prophylactic	30%	N
94	10180	Kauvery	28	F	Multinodular goitre	Thyroidectomy	2 hr 10 mir	I	Prophylactic	60%	N
95	14322	Amogsidda	24	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
96	14278	Bhimashi	25	M	Hemorrhoids	Hemorrhoidectomy	45 mins	II	Prophylactic	60%	N
97	14755	Parashuram	24	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	30%	N
98	15212	Shekappa	22	M	Appendicitis	Appendectomy	40 mins	II	Prophylactic	60%	N
99	17642	Kandoba	12	M	Appendicitis	Appendectomy	1 hr 10 mir	II	Prophylactic	30%	Y
100	15770	Kallappa	60	M	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	30%	N
101	16453	Iranna	35	M	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	60%	N
102	16840	Madiwalamma	48	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
103	17996	Shantawwa	30	F	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	60%	N
104	18739	Swafabai	63	F	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	30%	N
105	19128	Shankar	28	M	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	60%	N
106	19095	Indrabai	40	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
107	2204	Parvatamma	35	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
108	2679	Ramesh	18	M	Hemorrhoids	Hemorrhoidectomy	45 mins	II	Prophylactic	30%	N
109	3399	Shanakrawwa	42	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
110	3377	Ayappa	78	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	30%	N
111	19512	Suresh	46	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	60%	N
112	415	Megha	24	F	Appendicitis	Appendectomy	1 hr 10 mir	II	Prophylactic	30%	Y
113	622	Rahul	14	M	Phimosis	Circumcision	30 mins	II	Prophylactic	60%	N
114	1049	Aiyaz	18	M	Appendicitis	Appendectomy	1hr	II	Prophylactic	30%	N
115	2683	Vijaylaxmi	23	F	Appendicitis	Appendectomy	45 mins	II	Prophylactic	60%	N
116	2625	Nagappa	35	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	30%	N
117	2862	Kashibai	55	F	Appendicitis	Appendectomy	40 mins	II	Prophylactic	60%	N
118	4003	Shreedevi	30	F	Appendicitis	Appendectomy	1 hr 10 mir	II	Prophylactic	30%	N
119	3959	Satish	14	M	Phimosis	Circumcision	40 mins	II	Prophylactic	60%	N
120	4320	Savitri	32	F	Appendicitis	Appendectomy	45 mins	II	Prophylactic	30%	Y
121	4656	Bauramma	25	F	Appendicitis	Appendectomy	1hr	II	Prophylactic	60%	N
122	4678	Rudrappa	26	M	Appendicitis	Appendectomy	1 hr 20 mir	II	Prophylactic	30%	N
123	4741	Kamala	32	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
124	4856	Shankrappa	54	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	30%	Y
125	4747	Iranna	40	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	60%	N
126	4846	Kamalabai	50	F	Hemorrhoids	Hemorrhoidectomy	45 mins	II	Prophylactic	30%	N
127	5408	Shantappa	75	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
128	6234	Sangaraj	28	M	Appendicitis	Appendectomy	20 mins	II	Prophylactic	60%	N
129	6286	Suresh	30	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
130	6795	Hanmantraya	18	M	Appendicitis	Appendectomy	1 hr 10 mir	II	Prophylactic	30%	N
131	6804	Ramesh	40	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
132	6782	Sharada	37	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
133	7025	Sharadha	40	F	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	60%	N
134	7190	Anusuyya	28	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
135	7193	Kavita	23	F	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	60%	N
136	7586	Vipul oswal	21	M	Appendicitis	Appendectomy	50 mins	II	Prophylactic	30%	N
137	8053	Nandappa	65	M	Hemorrhoids	Hemorrhoidectomy	45 mins	II	Prophylactic	60%	Y
138	8091	Mallanna	18	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
139	8595	Mutappa	45	M	Hemorrhoids	Hemorrhoidectomy	35 mins	II	Prophylactic	60%	Y
140	9267	Akash	9	M	Appendicitis	Appendectomy	30 mins	II	Prophylactic	30%	N
141	9199	Savithri	28	F	Appendicitis	Appendectomy	1hr 15 min	II	Prophylactic	60%	N
142	9245	Komesh	30	M	Phimosis	Circumcision	30 mins	II	Prophylactic	30%	N
143	8979	Hampanna	32	M	Hemorrhoids	Hemorrhoidectomy	30 mins	II	Prophylactic	60%	N
144	9143	Mallawwa	34	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
145	9711	Gangadhar	55	M	Phimosis	Circumcision	25 mins	II	Prophylactic	60%	N
146	9737	Sidmallappa	30	M	Hydrocele	Eversion of sac	35 mins	II	Prophylactic	30%	Y
147	8981	Ramesh	24	M	Hydrocele	Eversion of sac	30 mins	II	Prophylactic	60%	N
148	9726	Gangabai	38	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
149	9721	Bhimanna	48	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
150	10023	Kallappa	50	M	Hemorrhoids	Hemorrhoidectomy	50 mins	II	Prophylactic	30%	N
151	10853	Somalingappa	88	F	Hemorrhoids	Hemorrhoidectomy	35 mins	II	Prophylactic	60%	N
152	10822	Siddarth	18	M	Appendicitis	Appendectomy	40 mins	II	Prophylactic	30%	N
153	10818	Ramesh	53	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	60%	N

154	11331	Savithri	16	F	Appendicitis	Appendectomy	1 hr 30 mir	II	Prophylactic	30%	N
155	11342	Mallappa	30	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
156	11255	Bowrawwa	45	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
157	11259	Siddappa	40	M	Appendicitis	Appendectomy	30 mins	II	Prophylactic	60%	N
158	11257	Ramesh	38	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
159	11651	Mallappa	46	M	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	60%	N
160	11653	Hananmanth	47	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	30%	Y
161	1186	Mallappa	65	M	Appendicitis	Appendectomy	1 hr 30 mir	II	Prophylactic	60%	Y
162	11835	Iranna	38	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	30%	N
163	12885	Sharanbasu	25	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	60%	N
164	12768	Siddappa	26	M	Appendicitis	Appendectomy	55 mins	II	Prophylactic	30%	N
165	12770	Suresh	35	M	Hydrocele	Eversion of sac	35 mins	II	Prophylactic	60%	Y
166	12928	Bapugouda	50	M	Appendicitis	Appendectomy	40 mins	II	Prophylactic	30%	Y
167	14071	Shantabai	30	F	Appendicitis	Appendectomy	55 mins	II	Prophylactic	60%	N
168	17284	B.M.Terdal	47	M	Appendicitis	Appendectomy	1hr 15 min	II	Prophylactic	30%	N
169	1540	Mahananda	45	M	Fissure-in-ano	Lat sphincterotomy	35 mins	II	Prophylactic	60%	N
170	16370	Renuka	20	F	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	30%	N
171	16904	Sahebgouda	5	M	Phimosis	Circumcision	20 mins	II	Prophylactic	60%	N
172	16728	Kavita	38	F	Fissure-in-ano	Lat sphincterotomy	30 mins	II	Prophylactic	30%	N
173	17576	Parasuram	33	M	Appendicitis	Appendectomy	55 mins	II	Prophylactic	60%	N
174	18502	Pramod	18	M	Phimosis	Circumcision	25 mins	II	Prophylactic	30%	N
175	19101	Dhanaraj	2	M	Phimosis	Circumcision	20 mins	II	Prophylactic	60%	N
176	19791	Meenakshi	30	F	Appendicitis	Appendectomy	1hr 15 min	II	Prophylactic	30%	N
177	25033	Mahesh	35	M	Hydrocele	Eversion of sac	35 mins	II	Prophylactic	60%	N
178	26304	Raghavendra	14	F	Phimosis	Circumcision	20 mins	II	Prophylactic	30%	N
179	27310	Sharadamma	50	F	Fissure-in-ano	Lat sphincterotomy	15 mins	II	Prophylactic	60%	N
180	27805	Kumar	26	M	Appendicitis	Appendectomy	1hr 15 min	II	Prophylactic	30%	Y
181	27806	Shivkumar	19	M	Appendicitis	Appendectomy	55 mins	II	Prophylactic	60%	Y
182	1613	Prashant	22	M	Fissure-in-ano	Lat sphincterotomy	25 mins	II	Prophylactic	30%	N
183	8809	Dundappa	65	M	Hemorrhoids	Hemorrhoidectomy	40 mins	II	Prophylactic	60%	N
184	9843	Malathi	24	F	Appendicitis	Appendectomy	55 mins	II	Prophylactic	30%	N
185	8791	Bhagya	36	F	Fissure-in-ano	Lat sphincterotomy	20 mins	II	Prophylactic	30%	N
186	10250	Saipansab	18	M	Appendicitis	Appendectomy	55 mins	II	Prophylactic	60%	N
187	9840	Mallappa	35	M	Hydrocele	Eversion of sac	35 mins	II	Prophylactic	30%	N
188	6268	Shobha	21	F	Appendicitis	Appendectomy	1hr 15 min	II	Prophylactic	30%	N