A DISSERTATION ON

" A STUDY ON SURGICAL SITE INFECTIONS IN EMERGENCY NON-TRAUMATIC ABDOMINAL OPERATIONS "

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY

CHENNAI

with partial fulfilment of the regulations

for the Award of the degree

M.S. (General Surgery)

Branch – I

INSTITUTE OF GENERAL SURGERY,

MADRAS MEDICAL COLLEGE ,

CHENNAI.

APRIL-2015

CERTIFICATE

This is to certify that the dissertation entitled "A STUDY ON SURGICAL SITE INFECTIONS IN EMERGENCY NON-TRAUMATIC ABDOMINAL OPERATIONS" is a bonafide original work of

Dr. B.PADAM KUMAR., in partial fulfilment of the requirements for M.S.Branch– I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in APRIL 2015 under my guidance and supervision in 2013-14.

Prof.Dr.A.RAJENDRAN, M.S
Professor of General Surgery,
Guide and supervisor
Institute of General Surgery,
Madras Medical College,
Rajiv Gandhi Government
General Hospital,
Chennai – 600003.

Prof.Dr.P.RAGUMANI. M.S
Director and Professor
Institute of General Surgery,
Madras Medical College,
Rajiv Gandhi Government General
Hospital,
Chennai – 600003.

Dr. VIMALA, M.D,
Dean
Madras Medical College &Rajiv Gandhi Government General Hospital,
Chennai-600003

DECLARATION

I hereby solemnly declare that the dissertation titled "A STUDY ON SURGICAL SITE INFECTIONS IN EMERGENCY NON-TRAUMATIC ABDOMINAL OPERATIONS" is done by Me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai during 2013-14 under the guidance and supervision of Prof.Dr.A.RAJENDRAN, M.S, The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfillment of requirements for the award of M.S. Degree (Branch-I) in General Surgery.

Place:
Date:

DR.B.PADAM KUMAR,
Post Graduate student ,
M.S., General Surgery,
Institute of General Surgery,
Madras Medical College,
Chennai-3.

ACKNOWLEDGEMENT

I express my heartful gratitude to the Dean, **Dr.VIMALA** M.D., Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 for permitting me to do this study.

I am deeply indebted to **Prof.Dr.P.RAGUMANI**,M.S., Director& Professor, Institute of General Surgery, Madras MedicalCollege & Rajiv Gandhi Government General Hospital for his support and guidance.

I am very grateful to **Prof.Dr.A.RAJENDRAN** M.S, Professor of Surgery, Institute of General Surgery, Dr.A.SAGAYA INBA SEKAR M.S, Dr.P.RAMADOSS M.S, Madras Medical College & Rajiv Gandhi Government General Hospital who guided and trimmed my work throughout the period of my study.

I am extremely thankful to all the Members of the INSTITUTIONAL ETHICAL COMMITTEE for giving approval for my study. I also thank all the patients who were part of the study and my Professional colleagues for their support and criticisms.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

GRE	:	Glycopeptide Resistant Enterococci
MMC	:	Madras Medical College.
MRSA	:	Methicillin-Resistant *Staphylococcus Aureus*
POD	:	Post Operative Day
SSI	:	Surgical Site Infection
VISA	:	Vancomycin Intermediate S. *Aureus*

INTRODUCTION

1.1 BACKGROUND

The infection of a wound can be defined as the invasion of organisms through tissues following a breakdown of local and systemic host defences, leading to cellulitis, lymphangitis, abscess and bacteraemia. Surgical site infection (SSI) has always been a major complication of surgery and trauma and has been documented for 4000-5000 years. Galen recognized that localization of infection in wounds, inflicted in the gladiatorial arena, often heralded recovery, particularly after drainage. The understanding of the causes of infection came in the 19th century. Microbes had been seen under microscope, but Koch laid down the first definition of infective disease known as Koch's postulates. Koch's postulates providing the agency of an infective organism: it must be found in considerable numbers in the septic focus, it should be possible to culture it in a pure form from that septic focus and it should be able to produce similar lesions when injected into another host. Louis Pasteur recognized that micro-organisms were responsible for spoiling wine, turning it into vinegar (Williams *et al.* 2008).

Surgical Site Infections (SSIs), previously called post operative wound infections, result from bacterial contamination during or after a surgical procedure. Surgical site infections are the third most common hospital

associated infection, accounting for 14-16 per cent of all infections in hospitalized patients. Among surgical patients, surgical site infections are the most frequent cause of such infections, accounting for 38 per cent of the total. Despite every effort to maintain asepsis, most surgical wounds are contaminated to some extent. However infection rarely develops if contamination is minimal, if the wound has been made without undue injury, if the subcutaneous tissue is well perfused and well oxygenated and if there is no dead space. The criteria used to define surgical site infections have been standardized and described three different anatomic levels of infection: superficial incisional surgical site infection, deep incisional surgical site infection and organ/space surgical site infection (Doherty and Way 2006). According to the degree of contamination wounds may be classified as clean, potentially contaminated, contaminated, and dirty. The incidence of infection, morbidity and mortality increases from clean to dirty. The risk of infection is greater in all categories if surgery is performed as an emergency (Kirk and Ribbans 2004). The risk of wound infection is influenced but not entirely determined by the degree of contamination. Multiple risk factors and perioperative characteristics can increase the likelihood of superficial surgical site infections.

Important host factors include – diabetes mellitus, hypoxemia, hypothermia, leucopenia, nicotine, long term use of steroids or immunosuppressive agents, malnutrition, nares contaminated with *Staphylococcus Aureus* and poor skin hygiene. Perioperative / environmental factors are operative site shaving, breaks in operative sterile technique, early or delayed initiation of antimicrobial prophylaxis, inadequate intraoperative dosing of antimicrobial prophylaxis, infected or colonized surgical personnel, prolonged hypotension, poor operative room air quality, contaminated operating room instruments or environment and poor wound care postoperatively (Doherty and Way 2006).

Wound infections usually appear between fifth and tenth post operative day, but they may appear as early as first post operative day or even years later. The first sign is usually fever, and post operative fever requires inspection of the wound. The patient may complain of pain at the surgical site. The wound rarely appear severely inflamed, but edema may be obvious because the skin sutures appear tight (Doherty and Way 2006).

Advances in the control of infection in surgery have occurred in many ways, such as, aseptic operating theatre techniques have replaced toxic antiseptic techniques, antibiotics have reduced post operative infection rates, delayed primary or secondary closure remains useful in contaminated wounds. When enteral feeding is suspended during the peri-operative period, and particularly with underlying disease such as immunosuppression, cancer, shock or sepsis bacteria tend to colonize the normally sterile upper gastrointestinal tract. They may then translocate to the mesenteric lymph nodes and cause the release of endotoxin, which further increases the susceptibility to infection and sepsis, through activation of macrophages and pro-inflammatory cytokine release. The use of selective decontamination of the digestive tract (SDD) is based on the prevention of this colonization (Williams *et al.* 2008).

According to the sources, infection may be classified into two types, primary and secondary or exogenous. Primary infections are those acquired from community or endogenous source. Secondary or exogenous infections are acquired from operating theatre or the ward or from contamination at or after surgery. According to severity, surgical site infections can be divided into two types, major and minor. Criteria of major SSI are — significant quantity of pus, delayed return home and Patients are systemically ill.

Minor SSI may discharge pus or infected serous fluid but should not be associated with excessive discomfort, systemic signs or delay in return home (Williams *et al.* 2008).

There are various types of localized infections, such as abscess, cellulites, lymphangitis etc. Abscess may follow puncture wound as well as surgery, but can be metastatic in all tissues following bacteraemia. Abscess needs drainage with curettage.

Modern imaging techniques may allow guided aspiration. Antibiotics are indicated if the abscess is not localized. Healing by secondary intention is encouraged. Cellulites are nonsuppurative invasive infection of tissues. It is poorly localized in addition to cardinal signs of inflammation. It is usually caused by organisms such as *ß-hemolytic streptococci, staphylococci and C. perfringens.* Tissue destruction, gangrene and ulceration may follow, which are caused by release of proteases. Systemic signs are common, such as SIRS, chills, fever and rigors. These follow the release of organisms, exotoxins and cytokines into the circulation. However, blood cultures are often negative. Lymphangitis presents as painful red streaks in affected lymphatic, often accompanied by painful lymph node groups in the related drainage area (Williams *et al.* 2008).

Systemic inflammatory response syndrome (SIRS) can be defined as, presence of any two of:

hyperthermia (>38°C) or hypothermia (<36° C),

tachycardia (>90 min^{-1}, no ß-blockers) or tachypnoea (>20 min^{-1})

and white cell count >12× 10^{9} l^{-1} or <4 ×10^{9} l^{-1}

(Williams *et al.* 2008). Sepsis is defined as the systemic manifestation of SIRS, with a documented infection. Multiple organ dysfunction syndrome (MODS) is the effect that the infection produces systemically. Multiple system organ failure (MSOF) is the end-stage of uncontrolled MODS (Williams *et al.* 2008).

Specific wound infections such as gas gangrene, tetanus and synergistic spreading gangrene are serious infections. Gas and smell are characteristics of gas gangrene that is caused by *clostridium perfringens.* Immunocompromised patients are most at risk. Antibiotic prophylaxis is essential when performing amputation to remove dead tissue. Tetanus caused by *clostridium tetani,* can develop following implantation of the organisms into tissues or a wound. The spores are wide spread in the soil and manure. Signs and symptoms are mediated by release of exotoxin tetanospasmin. Prophylaxis with tetanus toxoid is the best preventive treatment. The use of anti-toxin using human immunoglobulin ought to be considered in both at risk and established

infection. Synergistic spreading gangrene / Sub dermal gangrene / Necrotizing fasciitis is caused by a mixed pattern of organisms such as, Coliforms, Staphylococci**,** Bacteroides spp, anaerobic Streptococci and Peptostreptococci have all been implicated, acting in synergy. When occurs in the abdominal wall, known as Meleney's synergistic hospital gangrene and when occurs in the scrotum it is known as Fournier's gangrene (Williams *et al.*2008).

The use of antibiotic prophylaxis before surgery has evolved greatly in the last twenty years. It is generally recommended in elective clean surgical procedures using a foreign body and in clean-contaminated procedures that a single dose of cephalosporin, such as cefazolin, be administered intravenously by anesthesia personnel in the operative suit just before incision. Additional doses are generally recommended only when the operation lasts for longer than two to three hours (Nichols 2009).

Surgical site infection is the most important cause of morbidity and mortality in the post operative patients, but it is preventable in most of the cases if proper assessment and appropriate measures are taken by the surgeons, nursing staffs, patients and others in the perioperative period.

1.2 JUSTIFICATION

Surgical site infection still causes considerable morbidity and high cost to the health care system and is becoming increasingly important in medico-legal aspects. Infections increase the discomfort and disability experienced by patients following surgical procedures. Moreover, the most severe form may endanger life. A few studies were conducted in our country on such an important topic. Further research is necessary to identify the important factors responsible for high infection rate following emergency nontraumatic abdominal operations in our country. In this study it has been tried to find out the common organisms responsible for surgical site infections following emergency nontraumatic abdominal operations. In addition, the sensitivity patterns of the micro-organisms were ascertained. Further, factors responsible for infections were determined, that will be helpful to prevent infection in future following similar types of operations. So, these study findings will play an important role to reduce the infection rate and thereby reduce the morbidity and mortality. Furthermore, application of the recommendations of this study in the practical field will reduce the rate of surgical site infections in our country and thereby will improve cosmesis and make the results of operations better as a whole.

1.3 HYPOTHESIS

"*Escherichia Coli* is the commonest micro-organism responsible for surgical site infections following emergency non-traumatic abdominal operations.

1.4 OBJECTIVES

A. General:

To determine the factors responsible for surgical site infections following emergency non-traumatic abdominal operations, which will be helpful in reducing the rate of surgical site infection.

B. Specific:

(1) To determine the host factors responsible for surgical site infections.

(2) To detect the environmental factors contributing to surgical site infections following emergency nontraumatic abdominal operations.

(3) To identify the microorganisms involved in surgical site infections.

Chapter 2

REVIEW OF LITERATURE

REVIEW OF LITERATURE

2.1 Body's response to injury

The body responds to trauma with local and systemic reactions that attempt to contain and heal the tissue damage, and to protect the body while it is injured. The response is remarkably similar whether the trauma is a fracture, burn, sepsis or a planned surgical operation, and the extent of the response is usually proportional to the severity of the trauma. The response, with neuroendocrine and inflammatory cytokine components, increases the metabolic rate, mobilizes carbohydrate, protein and fat stores, conserves salt and water and diverts blood preferentially to vital organs. It also stimulates important protective mechanisms such as the immunological and blood clotting systems. However, the overall result is one of immunosuppression leading to increased vulnerability to infection. The interplay between the many inflammatory mediators and cellular responses is very complex. Major surgery has other inevitable consequences which predispose to postoperative morbidity. With optimal perioperative management, however, their impact can be minimized (Kirk and Ribbans 2004).

Initiation of the response

Various noxious stimuli produce the response but they rarely occur alone, and multiple stimuli often produce greater effects than the sum of single responses. The response is modified by the severity of the stimulus, the patient's age, nutritional status, coexisting medical conditions, medication and if the trauma or operation has affected the function of any particular organ. Recent trauma or sepsis will also modify the response to a subsequent surgical operation**.** Pain, tissue injury, infection, hypovolaemia and starvation play as major stimuli to initiate the response. Hypoxia, hypercarbia or ph changes, hypoglycaemia and hypothermia are important stimuli. Fear, anxiety and emotion also stimulate the sympathetic nervous system. Studies have shown improved recovery times with fewer infections when normothermia is maintained intraoperatively (Kirk and Ribbans 2004).

Systems controlling the response

The response to surgery is modulated both by the neuroendocrine system and the inflammatory mediators and the cells controlling their release. The effects are closely intertwined, with locally produced cytokines having systemic effects proportional to the extent and severity of tissue injury. There are multiple feedback loops which prevent excessive activation of the inflammatory cascades.

1. Sympathetic nervous system: The immediate fight and flight reaction may help the injured person to avoid further injury. It is stimulated particularly by pain and hypovolaemia and this has direct actions and indirect effects by releasing adrenaline and noradrenalin from the adrenal glands. These catecholamines have both á and â effects on sympathetic receptors that prepare the body rapidly for fight or flight by cardiovascular, visceral and metabolic actions. These effects continue for several days into the postoperative period.

Cardiovascular effects: Blood is redistributed from the viscera and skin to the heart, brain and skeletal muscles. There is an increase in heart rate and contractility.

Visceral effects: Non-essential visceral functions such as intestinal motility are inhibited, resulting in paralytic ileus, bladder sphincter tone is increased; other actions are bronchodilatation, mydriasis, uterine contraction and relaxation and visual field increases.

Metabolic and hormonal effects: Blood glucose rises due to increased breakdown of liver and muscle glycogen, gluconeogenesis , suppression of insulin secretion and stimulation of glucagon secretion.

2. Endocrine response: This includes not only the hypothalamic-pituitary-adrenal (HPA) axis but also growth hormone, AVP, thyroxine, insulin and

glucagon, causing some metabolic effects. This response protect against the body's acute phase response from overreacting. The hypothalamic pituitary adrenal (HPA) axis is stimulated mainly by the injury itself, but probably its most important function is to control the effects of systemically released cytokines.

ACTH is released from the anterior pituitary. It stimulates the adrenal cortex to release glucocorticoids and also potentiates the action of catecholamines on cardiac contractility.

Glucocorticoids usually have only a 'permissive' action (allowing other hormones to function) but the increased levels after trauma have important metabolic, cardiovascular and immunological actions proportional to the severity of the trauma. Cortisol, the main glucocorticoid, stimulates the conversion of protein to glucose and the storage of glucose as glycogen. It increase plasma glucose (diabetogenic action); it helps to maintain blood volume by decreasing the permeability of the vascular endothelium and enhancing vasoconstriction by catecholamines and suppressing synthesis of prostaglandins and leucotrienes (anti-inflammatory action); it also inhibits secretion of interleukin-1 and interleukin-2, antibody production and mobilization of lymphocytes (immunosuppressive action) (Kirk and Ribbans 2004).

Aldosterone: Trauma induced ACTH stimulates a short-term release of aldosterone, but the rise may be prolonged if other stimuli such as hypovolaemia or vasomotor changes occur. Aldosterone causes increased reabsorption of sodium and potassium secretion in the distal convoluted tubules and collecting ducts and hence a reduced urine volume.

Arginine vasopressin (AVP): Also referred to as antidiuretic hormone (ADH), this is released from the posterior pituitary by pain, a rise in plasma osmolality, hypovolaemia, anesthetic agents or a rise in plasma glucose. Its secretion increases for about 24 h after operation, so, the urine osmolality remains higher than plasma. After head injury, burns or prolonged hypoxia there may be continued secretion of AVP, resulting in oliguria and hyponatraemia.

Insulin: In the ebb phase after injury, plasma insulin concentration falls. Glucagon also inhibits insulin release and Cortisol reduces the peripheral action of insulin and blood sugar rises. In the flow phase, plasma insulin rises but blood sugar remains elevated because various intracellular changes make the tissues resistant to insulin.

Glucagon: Secretion of glucagon increases after injury and this plays a part in increasing blood sugar by stimulating hepatic glycogenolysis and gluconeogenesis.

Thyroxine: Total T4 and total and free T3 decrease after injury, because

cortisol impairs conversion of T4 to T3.

Growth hormone: Its plasma levels increase after trauma, hypovolaemia, hypoglycaemia or a decrease in plasma fatty acids or increase in serum arginine. Its main effects are to promote protein synthesis and enhance breakdown of lipid and carbohydrate stores.

3. Acute phase response: The wound becomes a 'cytokine organ' whose metabolism and local healing responses are controlled by cytokines and other mediators that are produced locally and also released from activated inflammatory cells, including neutrophils and monocytes. In severe trauma, proinflammatory cytokines produce a systemic 'acute phase' response, with profound changes in protein metabolism and immunological activation; these effects are mostly beneficial but in severe trauma can be lethal (Kirk and Ribbans 2004).

Local effects: Noxious stimuli such as infection, trauma, toxins, haemorrhage or malignancy attract granulocytes and mononuclear cells to the site of injury and these cells, together with local fibroblasts and endothelial cells, release Cytokines. Interleukins 1, 2 and 6, TNF and the interferons are the main cytokines released early. Their actions help to contain tissue damage by contributing to the inflammatory reaction through vasodilatation, increased

permeability of vessels, migration of neutrophils and monocytes to the wound, activation of the coagulation and complement cascades and proliferation of endothelial cells and fibroblasts.

Systemic effects: If cytokine production is large enough, systemic effects occur, such as fever, malaise, headache, myalgia as well as vasodilatation. They also affect the serum levels of acute phase reactants (APRs) which are host-defense proteins synthesized in the liver.

• **Tumour necrosis factor** (TNF or cachectin) released primarily from the macrophages by bacterial endotoxin, causes anorexia, tachypnoea, fever and tachycardia, with proliferation of fibroblasts and widespread effects on neutrophils; it stimulates production of other cytokines, ACTH, APRs and amino acids from skeletal muscle, hepatic amino acid uptake and elevation of plasma triglycerides and free fatty acids. High concentrations cause multiple organ dysfunction syndromes (MODS).

• **IL-1** in low dosage causes fever, neutrophilia, low serum zinc levels, increased APR synthesis, anorexia, malaise, release of ACTH, glucocorticoid and insulin, and in high dose, the features of MODS.

• **IL-2** enhances immune function by T-lymphocyte proliferation and by enhancing the activity of natural killer cells.

• **IL-6** is the main mediator of this altered hepatic protein synthesis.

• **Interferons** are glycoproteins produced by T-lymphocytes which activate macrophages, enhancing both antigen presenting and processing as well as cytocidal activity.

ã-interferon inhibits viral replication and inhibits prostaglandin release.

• **Prostaglandins** can be produced by all nucleated cells except lymphocytes. They increase vascular permeability and cause vasodilatation and leucocytes migration.

• **Leucotrienes** increasing post capillary leakage and they cause increased leucocyte adhesion, vasoconstriction and bronchoconstriction.

• **Kallikreins and kinins**: Bradykinin release is stimulated by hypoxia and it is a potent vasodilator that increases capillary permeability, producing oedema, pain and bronchoconstriction.

• **Heat shock proteins** are produced by virtually all cells in response to many stresses. The ability to produce them declines with age. They protect cells from the deleterious effects of stress and inhibit synthesis of APRs.

• **Histamine:** Histamine is released from mast cells, platelets, neurons and the epidermis by trauma, sepsis and hypotension. Its main action is to cause local vasodilatation and increased vascular permeability, so, large concentrations

may lead to hypotension.

• **Endogenous opioids** such as â-endorphin increase after trauma and produce analgesia, a rise in blood sugar, a lowering of blood pressure and effects on immune function (Kirk and Ribbans 2004).

4. **Vascular endothelial cell response**: This affects vasomotor tone and vessel permeability, so it affects perfusion, circulating volume and blood pressure and can lead to the clinical picture of shock and lung injury. Endothelial damage also activates the coagulation cascades and can result in microvascular clotting despite a generalized abnormal bleeding tendency. Nitric oxide is a powerful vasodilator produced mainly by endothelial cells but also by macrophages, neutrophils, Kupffer cells and renal cells. Endothelins are a family of potent vasoconstricting peptides with mainly paracrine actions. They are released by thrombin, catecholamines, hypoxia, cytokines and endotoxins. Platelet-activating factor (PAF) is released from endothelial cells by the action of TNF, IL-l, arginine vasopressin and angiotensin II. When platelets come into contact with PAF they release thromboxane which causes platelet aggregation and vasoconstriction.

5.) Prostaglandins cause vasodilatation and reduce platelet aggregation. Other arachidonic acid derivatives include thromboxanes, which are also produced by

cyclooxygenase. Atrial natriuretic peptides (ANPs) are potent inhibitors of aldosterone secretion and are released by atrial tissue in response to changes in chamber distension (Kirk and Ribbans 2004

Intracellular signalling processes and regulation of the acute stress response:

- **Gene transcription:** stimulation of cells by cytokines and other products of inflammatory damage, appear to be coupled to signaling systems that lead to upregulation of the genes coding for enzymes and cytokines by increasing RNA transcription. These inducible enzymes then greatly increase the production of mediators, sustaining the inflammatory response.
- **Apoptosis** is the programmed death of cells which ensures turnover of short-lived immune cells. It increases after trauma and also in sepsis, contributing to immunosuppression by loss of lymphocytes. Apoptosis also appears to be under the control of complex intracellular signaling processes.

Clinically apparent systemic effects of the response:

Body temperature: Following correction of any intraoperative hypothermia in the immediate postoperative period, there is often a 1-2°C increases in body temperature because the increased metabolic rate is accompanied by an upward shift in the thermoregulatory set point of the hypothalamus. Some of the effects of fever are detrimental, but more are beneficial.

Cardiovascular system: A mild tachycardia, peripheral vasodilatation and rise of Cardiac output provided intravascular volume is maintained. Hypovolaemia due to blood and other fluid losses can exaggerate the tachycardia and lead to hypotension and peripheral shutdown, indicating inadequate fluid replacement (Kirk and Ribbans 2004)

Pulmonary effects: Reduction in forced vital capacity and functional residual capacity, lead to shunting of blood and a decreasing PaO2 after major surgery. Hypoxaemia is more pronounced and prolonged after upper abdominal surgery. If secretions obstruct bronchioles, basal collapse can progress to pneumonia after any operation, particularly in immobile patients recumbent in bed. Acute lung injury is the inflammatory reaction due to pulmonary capillary endothelial damage and fluid leak into the alveoli and interstitium. There is a spectrum of severity, with the most extensive, acute respiratory distress syndrome (ARDS), leading to severe respiratory failure and widespread infiltrates on X-ray (Kirk and Ribbans 2004).

Effects on the gastrointestinal tract:

• **Adynamic ileus**: There is inhibition of gastric emptying and reduced colonic motility from increased sympathetic tone and the effects of opioid analgesics.

• **Gut mucosal barrier**: Increased permeability is thought to allow translocation of bacterial toxins into the circulation, leading to escalation of the

inflammatory response.

Biochemical and fluid balance disturbance:

1**. Salt and water retention**: This results from the mineralocorticoid effects of both aldosterone and cortisol. This is compounded by raised levels of AVP, further hindering excretion of free water and resulting in lower volumes of high osmolality urine. Any reduction in renal perfusion from hypotension secondary to hypovolaemia or from the administration of non-steroidal anti-inflammatory drugs also worsens oliguria and can lead to acute renal failure.

2. Hyponatraemia and hypokalaemia: This often accompanies the above changes, partly a dilutional effect from retained water and partly because sodium drifts into cells. Serum potassium may rise due to cell death, liberation of potassium by protein catabolism and from impaired potassium excretion. However, it is more usual to see increased urine potassium excretion, which can lead to an overall potassium deficit.

3. Acid-base abnormalities: The commonest change is a metabolic alka-losis**.** In more severe injuries a metabolic acidosis supervenes due to poor tissue perfusion and anaerobic metabolism with accumulation of lactic acid.

Metabolism after injury

1. There is an initial 'ebb' phase of reduced energy expenditure after injury for up to 24 h. These changes to a catabolic 'flow' phase with increased

metabolism, negative nitrogen balance, hyperglycemia, increased heat production, increased oxygen consumption and lean bodyweight loss. The increase in metabolic rate ranges from about 10% in elective surgical operations to 50% in multiple trauma and 200% in major burns.

2. Lipids are the principal source of energy following trauma. Lipolysis is produced mainly by catecholamines and increased sympathetic nervous system activity and also by lower plasma insulin, a rise in ACTH, cortisol, glucagon, growth hormone and, probably, cytokines.

3. Hyperglycaemia occurs immediately after injury because glucose is mobilized from stored glycogen in the liver by catecholamines and glucocorticoids, and because insulin resistance of peripheral tissues impairs their uptake of glucose (the 'diabetes of injury').

4. Body glycogen stores can only maintain blood glucose for about 24 h. Subsequently it is maintained by gluconeogenesis, stimulated by corticosteroids and glucagon, and this is helped by the initially suppressed insulin levels encouraging the release of amino acids from muscle.

5**.** Amino acids, protein and skeletal muscle: Shortly after injury, skeletal muscle protein breakdown supplies the three to fourfold increased demand for amino acids. The nitrogen loss is proportional to the severity of the trauma, the extent of sepsis and the muscle bulk. 6. Other reasons for skeletal muscle loss

include rhabdomyolysis in trauma and limb ischaemia, disuse atrophy from prolonged immobility and denervation from the polyneuropathy of critical illness (Kirk and Ribbans 2004).

Haematological changes:

Serum albumin falls after trauma because production by the liver decreases and loss into damaged tissue increases. The coagulation cascade and platelet activation leads to a state of hypercoagulability. Disseminated intravascular coagulation (DIC) can result. Leucocytosis occurs; it appears to be due mainly to cytokine-stimulated release of neutrophils from bone.

Immunological responses:

Trauma leads to impairment of the immune system, with defects in cell-mediated immunity, antigen presentation, neutrophil and macrophage function, complement activation and bacterial opsonization. This occurs at a time when the initial injury has usually breached mechanical defences, when catabolism impairs the mucosal barrier in the bowel and when many factors contribute to produce pneumonia and other infections.

Ways of reducing the response:

Although the local response to trauma is beneficial, the systemic response becomes less helpful as the degree of trauma increases, and in a hospital setting it is an advantage to suppress and control the response. In trauma and

emergency surgery, pain, bleeding with hypovolaemia, hypoxia and anxiety have often been present for some hours before operation starts, whereas in elective surgery it is usually possible to control these stimuli and thereby reduce the systemic response. Recent studies demonstrate that preoperative optimization of the circulation by the use of fluid loading and inotropes to increase cardiac output and oxygen delivery can improve the outcome of major surgery. Beta blockers given through the perioperative period confer cardiac protection in vulnerable patients. There appears to be a prolonged survival advantage well beyond the duration of administration.

Reduce stimuli causing the response: By reduction of trauma, nutritional support, correction of hypovolaemia, hypoxaemia and metabolic alkalosis or acidosis, control of infection and pain and removal of fear and stress.

Metabolic manipulation: By Protein administration to malnourished patients improves their immune function. Enteral feeding has particular benefits over the parenteral route. Increased intake of arginine and glutamine can be helpful.

Drug administration: Ways of manipulating the body's response to trauma are being sought but are still experimental. Steroids, antiendotoxin antibodies, anti-TNF anti-bodies, IL-1 receptor antagonists and specific PAF receptor antagonists have increased survival in septic animals but have been disappointingly ineffective in humans. A recent study involving activated

protein C in septic shock appears more promising; however, as bleeding tendency is increased it may not be suitable for septic patients undergoing surgery. Other agents that have been used are adrenergic blockers, aspirin, growth hormone, anabolic steroids, mannitol, propranolol, allopurinol and atrial natriuretic factor.

2.2 Wound healing

Wound healing is a mechanism whereby the body attempts to restore the integrity of the injured part (Williams *et al.* 2008**).** In normal skin, the epidermis and dermis exists in steady-state equilibrium, forming a protective barrier against the external environment. Once the protective barrier is broken, the normal process of wound healing is immediately set in motion.

Types of wounds – tidy vs. untidy

The site injured, the structures involved in the injury and the mechanism of injury influence healing and recovery of function. This has led to the management of wounds based upon their classification into tidy and untidy. The surgeon's aim is to convert untidy to tidy by removing all contaminated and devitalized tissues. Primary repair of all structures may be possible in a tidy wound, but a contaminated wound with dead tissue requires debridement on one or several occasions before definitive repair can be carried out that is known as the concept of 'second look' surgery (Williams *et al.* 2008**).**

Factors influencing healing of a wound:

A. Local factors:

- Site of the wound

- Structures involved
- Mechanism of wounding: Incision, Crush, Crush avulsion etc.
- Contamination (foreign bodies/bacteria)
- Loss of tissue
- Other local factors: Vascular insufficiency (arterial or venous), previous radiation, pressure etc.

B. Systemic factors:

- Malnutrition or vitamin and mineral deficiencies,
- Disease (e.g. diabetes mellitus),
- Medications (e.g. steroids)
- Immune deficiencies (e.g. chemotherapy, AIDS),
- Smoking etc. (Williams *et al.* 2008**)**

Process of wound healing

The physiological process of wound healing is usually divided into three phases: the inflammatory phase, the proliferative or fibroblastic phase and the maturation or remodeling phase (Cuschieri *et al.* 2002).

The inflammatory phase: Upon injury to the skin, a set of complex biochemical events takes place in a closely orchestrated cascade to repair the damage (Stadelmann *et al.*1998). Within minutes post-injury, platelets (thrombo-cytes) aggregate at the injury site to form a fibrin clot. This clot acts to control active bleeding (hemostasis). The inflammatory phase begins immediately after wounding and lasts 2–3 days. Platelets stick to the damaged endothelial lining of vessels, releasing adeno-sine diphosphate (ADP), which causes thrombocytic aggregates to fill the wound. When bleeding stops, the platelets then release several cytokines from their alpha granules. These are plateletderived growth factor (PDGF), platelet factor IV and transforming growth factor beta (TGFâ). These attract inflammatory cells such as polymorphonuclear lymphocytes (PMN) and macrophages. Platelets and the local injured tissue release vasoactive amines such as histamine, serotonin and prostaglandins , which increase vascular permeability, thereby aiding infiltration of these inflammatory cells.

Macrophages remove devitalised tissue and microorganisms while regulating

fibroblast activity in the proliferative phase of healing. The initial framework for structural support of cells is provided by fibrin produced by fibrinogen. A more historical (Latin) description of this phase is described in four words: rubor (redness), tumour (swelling), calor (heat) and dolour (pain) (Williams *et al.* 2008**).**

The proliferative phase: The proliferative phase is characterized by angiogenesis, collagen deposition, granulation tissue formation, epithelialization and wound contraction (Midwood *et al.* 2004). In angiogenesis, new blood vessels are formed by vascular endothelial cells (Chang *et al.* 2004). In fibroplasia and granulation tissue formation, fibroblasts grow and form a new, provisional extracellular matrix (ECM) by excreting collagen and fibronectin (Midwood *et al.* 2004). Concurrently, re-epithelialization of the epidermis occurs, in which epithelial cells proliferate and 'crawl' atop the wound bed, providing cover for the new tissue (Garg 2000). In contraction, the wound is made smaller by the action of myofibroblasts, which establish a grip on the wound edges and contract themselves using a mechanism similar to that in smooth muscle cells. When the cells roles are close to complete, unneeded cells undergo apoptosis (Midwood *et al.* 2004). The proliferative phase lasts from the third day to the third week. Fibroblasts require vitamin C to produce collagen.

The wound tissue formed in the early part of this phase is called granulation tissue. In the latter part of this phase, there is an increase in the tensile strength of the wound due to increased collagen, which is at first deposited in a random fashion and consists of type III collagen (Williams *et al.* 2008**).**

The remodelling phase: The remodelling phase is characterised by maturation of collagen (type I replacing type III until a ratio of 4:1 is achieved). There is a realignment of collagen fibres along the lines of tension, decreased wound vascularity and wound contraction due to fibroblast and myofibroblast activity (Williams *et al.* 2008**).** Cells that are no longer needed are removed by apoptosis. However, this process is not only complex but fragile, and susceptible to interruption or failure leading to the formation of chronic non-healing wounds. Factors which may contribute to this include diabetes, venous or arterial disease, old age, and infection (Enoch and Price 2004). The phases of wound healing normally progress in a predictable, timely manner; if they do not, healing may progress inappropriately to either a chronic wound such as a venous ulcer or pathological scarring such as a keloid scar (Midwood *et al.* 2004).

Wound Repair versus Regeneration

There is a subtle distinction between ‘repair’ and ‘regeneration'. An injury is an interruption of morphology and/or functionality of a given tissue. Repair refers to the physiologic adaptation of an organ after injury in an effort to re-establish continuity without regards to exact replacement of lost/damaged tissue.

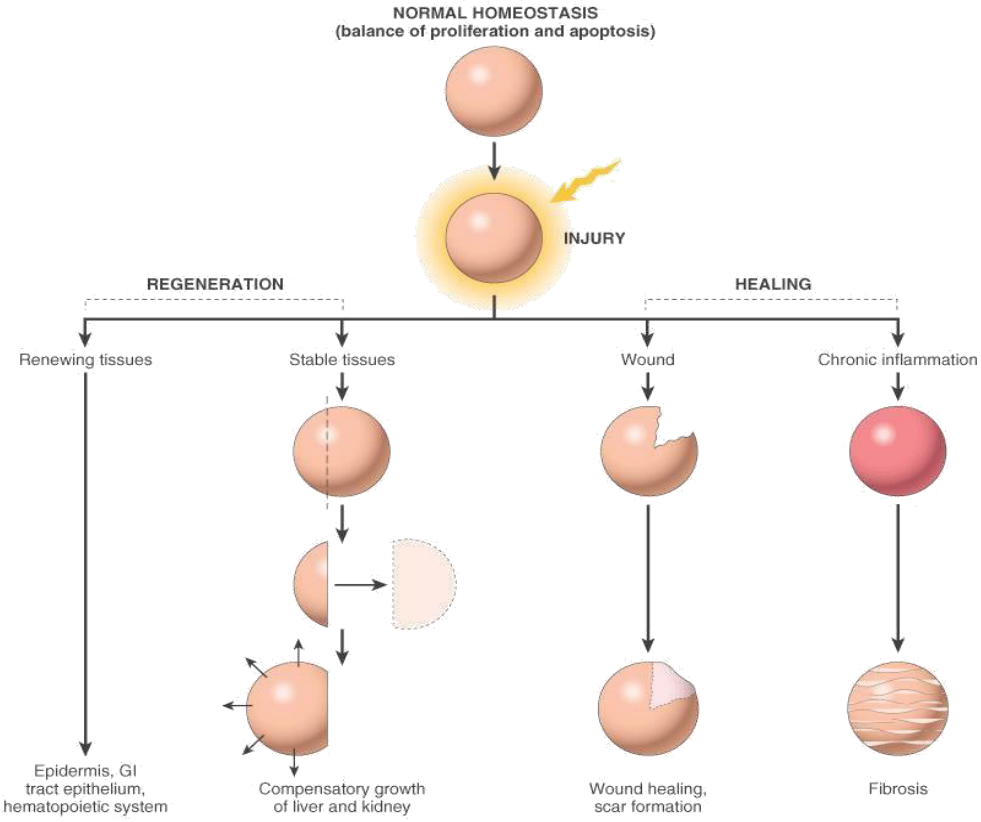


Fig. 1: Repair after injury either by regeneration or healing.

True tissue regeneration refers to the replacement of lost/damaged tissue with an 'exact' copy, such that both morphology and functionality are completely restored. Mammals do not regenerate spontaneously. In some instances, such as skin, 'partial regeneration' may be induced by the use of scaffolds (Nguyen *et al.* 2009).

Types of healing

***1*. Healing by Primary intention:** It is a healing by the process of epithelialization. It occurs when wound edges are brought together so that they

are adjacent to each other. It minimizes scarring. Most surgical wounds heal by primary intention. Wound closure is performed with sutures, staples, or adhesive tape. Examples are well-repaired lacerations, well reduced bone fractures, healing after flap surgery etc.

2. Healing by Secondary intention: Here the wound is allowed to granulate. Granulation results in a broader scar. Wound care must be performed daily to encourage wound debris removal to allow for granulation tissue formation. Examples are gingivectomy, gingivoplasty, tooth extraction sockets, poorly reduced fractures etc.

3. Healing by Tertiary intention: This type of healing occurs following delayed primary closure or secondary suture. The wound is initially cleaned, debrided and observed, typically 4 or 5 days before closure. The wound is purposely left open. An example of this type is healing of wounds by use of tissue grafts (Williams *et al.* 2008).

2.3 Healing of abdominal incisions

Cutaneous wound healing:

The healing wound, as a prototype of tissue repair, is a dynamic and changing process. The early phase is one of inflammation, followed by formation of granulation tissue and subsequent tissue remodeling and scarring. Simple cutaneous incisional wounds heal by first intention. Large cutaneous wounds heal by second intention, generating a significant amount of scar tissue. Different mechanisms occurring at different times trigger the release of chemical signals that modulate the orderly migration, proliferation, and differentiation of cells and the synthesis and degradation of ECM proteins. These proteins, in turn, directly affect cellular events and modulate cell responsiveness to soluble growth factors. The magic behind the precise orchestration of these events under normal conditions remains beyond our grasp. It almost certainly lies in the regulation of specific soluble and membrane-anchored mediators and their receptors on particular cells, cell-matrix interactions, and the effect of physical factors, including ECM remodeling forces generated by changes in cell shape (Kumar *et al.* 2005).

Complications in cutaneous wound healing:

Complications in wound healing can arise from abnormalities in any of the basic components of the repair process. These aberrations can be grouped into three general categories: deficient scar formation, excessive formation of the repair components, and formation of contractures (Kumar *et al.* 2005).

Deficient scar formation: An atrophic scar is pale, flat and stretched in appearance, often appearing on the back and areas of tension. It is easily traumatized as the epidermis and dermis are thinned. Excision and resuturing may only rarely improve such a scar (Williams et al. 2008). Inadequate formation of granulation tissue or assembly of a scar can lead to two types of complications: wound dehiscence and ulceration. Dehiscence or rupture of a wound is most common after abdominal surgery and is due to increased abdominal pressure. This mechanical stress on the abdominal wound can be generated by vomiting, coughing, or ileus. Wounds can ulcerate because of inadequate vascularization during healing. For example, lower extremity wounds in individuals with atherosclerotic peripheral vascular disease typically ulcerate. Nonhealing wounds also form in areas devoid of sensation. These neuropathic ulcers are occasionally seen in patients with diabetic peripheral neuropathy (Kumar *et al*. 2005).

Hypertrophic scar: Excessive formation of the components of the repair process can also complicate wound healing. The accumulation of excessive amounts of collagen may give rise to a raised scar known as a hypertrophic scar (Kumar *et al.* 2005). A hypertrophic scar is defined as excessive scar tissue that does not extend beyond the boundary of the original incision or wound. It results from a prolonged inflammatory phase of wound healing and from unfavourable scar siting (i.e. across the lines of skin tension). In the face, these are known as the lines of facial expression (Williams et al. 2008).

Keloid: If the scar tissue grows beyond the boundaries of the original wound and does not regress, it is called a keloid. Keloid formation appears to be an individual predisposition, and for unknown reasons this aberration is somewhat more common in African- Americans. The mechanisms of keloid formation are still unknown (Kumar *et al*. 2005). It is associated with elevated levels of growth factor, deeply pigmented skin, an inherited tendency and certain areas of the body (e.g. a triangle whose points are the xiphisternum and each shoulder tip). The histology of both hypertrophic and keloid scars shows excess collagen with hypervascularity, but this is more marked in keloids where there is more type B collagen. Hypertrophic scars improve spontaneously with time, whereas keloids do not (Williams *et al.* 2008).

Exuberant granulation: Another deviation in wound healing is the formation of excessive amounts of granulation tissue, which protrudes above the level of the surrounding skin and blocks re-epithelialization. This has been called exuberant granulation (proud flesh). Excessive granulation must be removed by cautery or surgical excision to permit restoration of the continuity of the epithelium (Kumar *et al*. 2005).

Desmoids: Rarely incisional scars or traumatic injuries may be followed by exuberant proliferation of fibroblasts and other connective tissue elements that may, in fact, recur after excision, called desmoids, or aggressive fibromatoses, these lie in the interface between benign proliferations and malignant (though low-grade) tumors (Kumar *et al*. 2005).

Contractures: Contraction in the size of a wound is an important part of the normal healing process. An exaggeration of this process is called a contracture and results in deformities of the wound and the surrounding tissues. Contractures are particularly prone to develop on the palms, the soles, and the anterior aspect of the thorax. Contractures are commonly seen after serious burns and can compromise the movement of joints (Kumar *et al*. 2005). Where scars cross joints or flexion creases, a tight web may form restricting the range of movement at the joint. This may be referred to as a contracture and can cause hyperextension or hyperflexion deformity. In the neck, it may interfere with

head extension. Treatment may be simple involving multiple Z-plasties or more complex requiring the inset of grafts or flaps. Splintage and intensive physiotherapy are often required postoperatively (Williams *et al.* 2008).

Maturation of Scars

The immature scar becomes mature over a period lasting a year or more, but it is at first pink, hard, raised and often itchy. The disorganised collagen fibres become aligned along stress lines with their strength being in their weave rather than in their amount. As the collagen matures and becomes denser, the scar becomes almost acellular as the fibroblasts and blood vessels reduce. The external appearance of the scar becomes paler,

while the scar becomes softer, flattens and its itchiness diminishes. Most of these changes occur over the first 3 months but a scar will continue to mature for 1–2 years. Tensile strength will continue to increase but will never reach that of normal skin (Williams et al. 2008).

Avoidable scarring

If the acute wound has been managed correctly, most of the problems described here should not occur. However, the surgeon should always stress that there will be a scar of some description after wounding, be it planned or accidental. Dirt ingrained /tattooed scar is usually preventable by proper initial scrubbing and cleansing of the wound. Mismatched or misaligned scars result from a failure to recognize normal landmarks such as the lip vermilion/white roll interface, eyelid and nostril free margins and hair lines such as those relating to eyebrows and moustache. Poorly contoured scars can be stepped, grooved or pin cushioned. Most are caused by poor alignment of deep structures such as muscle or fat, but trapdoor or pin cushioned scars are often unavoidable unless the almost circumferential wound can be excised initially. Suture marks may be minimised by using monofilament sutures that are removed early within 3–5 days. Sutures inserted under tension will leave marks. The wound can be strengthened post suture removal by the use of sticky strips. Fine sutures (6/0 or smaller) placed close to the wound margins tend to leave less scarring. Subcuticular suturing avoids suture marks either side of the wound or incision (Williams et al. 2008)**.**

2.4 Immunity to infection

We are constantly being exposed to infectious agents and yet, in most cases, we are able to resist these infections. It is our immune system that enables us to resist infections. The immune system is composed of two major subdivisions: the innate or non-specific immune system and the adaptive or specific immune system. The innate immune system is our first line of defense against invading organisms while the adaptive immune system acts as a second line of defense and also affords protection against re-exposure to the same pathogen. Each of the major subdivisions of the immune system has both cellular and humoral components by which they carry out their protective function. In addition, the innate immune system also has anatomical features that function as barriers to infection. Pasteur showed that protection against a particular disease could be conferred either by past exposure to the disease or by immunization with cultures of the causative agents which first rendered harmless. In nonspecific immunity the immune response evoked is not directed against one particular organism. This immunity is innate and dose not has to be learnt. Specific immunity is the property of vertebrate. It appears phylogenetically with the evolution of lymphoid tissue, thymus and spleen. This type of immunity is directed against specific organism (Janeway *et al.* 2001).

Innate (Non-Specific) Immunity

The elements of the innate (non-specific) immune system include anatomical barriers, secretory molecules and cellular components.

A. Anatomical barriers to infections

1. Mechanical factors: The epithelial surfaces form a physical barrier that is very impermeable to most infectious agents. Thus, the skin acts as our first line of defense against invading organisms. Movement due to cilia or peristalsis helps to keep air passages and the gastrointestinal tract free from microorganisms. The flushing action of tears and saliva helps prevent infection of the eyes and mouth. The trapping effect of mucus that lines the respiratory and gastrointestinal tract helps protect the lungs and digestive systems from infection (Janeway *et al*. 2001).

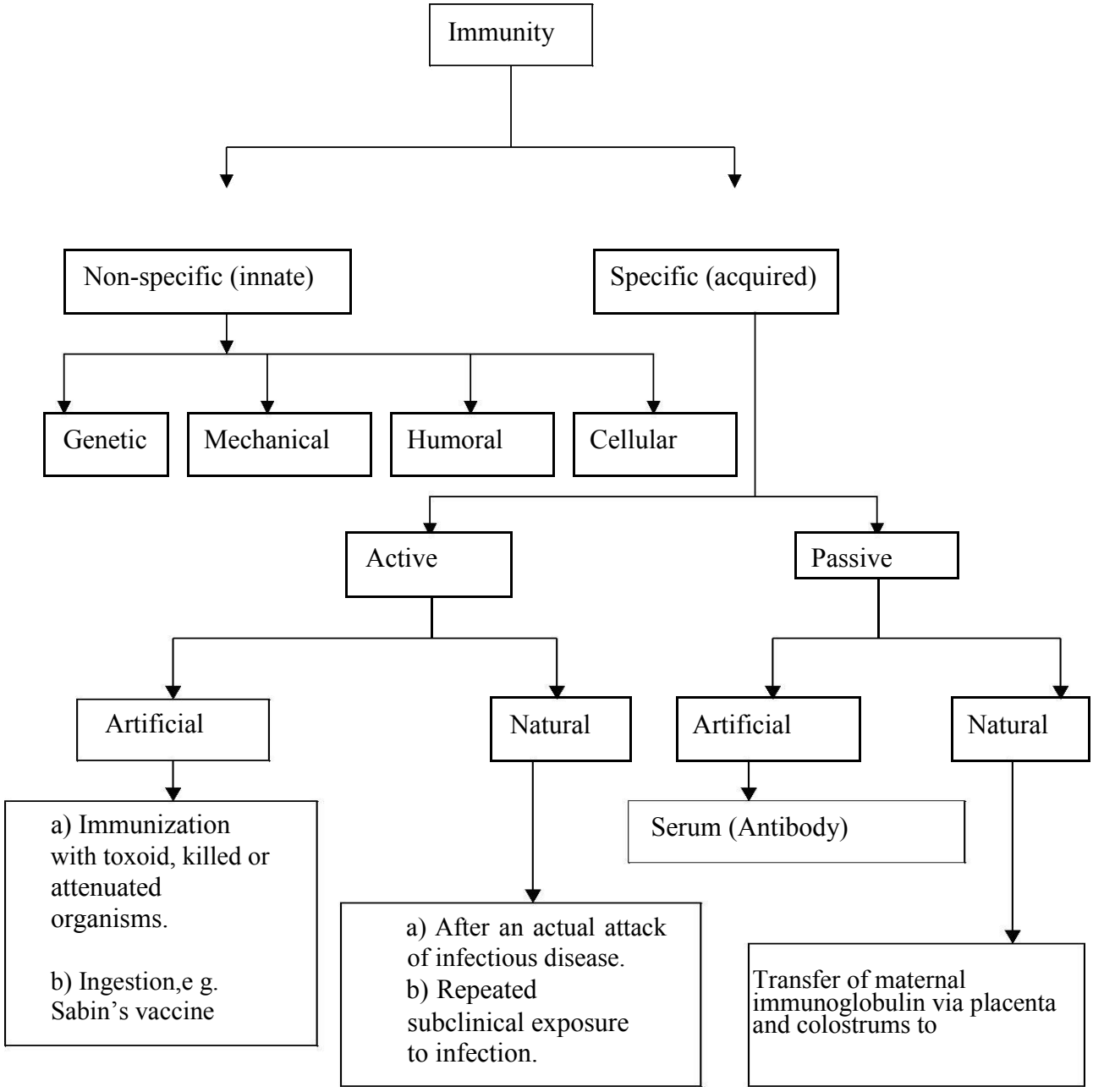


Fig. 2: Types of immunity (Jawetz *et al.* 1982)

2. Chemical factors: Fatty acids in sweat inhibit the growth of bacteria. Lysozyme and phospholipase found in tears, saliva and nasal secretions can breakdown the cell wall of bacteria and destabilize bacterial membranes. The low pH of sweat and gastric secretions prevents growth of bacteria.

3. Biological factors: The normal flora of the skin and in the gastrointestinal tract can prevent the colonization of pathogenic bacteria by secreting toxic substances or by competing with pathogenic bacteria for nutrients or attachment to cell surfaces.

B. Humoral barriers to infection:

When there is damage to tissues the anatomical barriers are breached and infection may occur. Once infectious agents have penetrated tissues, another innate defense mechanism comes into play, namely acute inflammation. Humoral factors play an important role in inflammation, which is characterized by edema and the recruitment of phagocytic cells. These humoral factors are found in serum or they are formed at the site of infection. These include

1. Complement system: Once activated complement can lead to increased vascular permeability, recruitment of phagocytic cells, and lysis and opsonization of bacteria.

2.Coagulation system: Some products of the coa-gulation system can contribute to the non-specific defenses because of their ability to increase vascular permeability and act as chemotactic agents for phagocytic cells. Some of the products are directly antimicrobial; for example, beta-lysin.

3.Lactoferrin and transferrin: By binding iron these proteins limit bacterial growth.

4.Interferons: These are proteins that can limit virus replication in cells.

5.Lysozyme: Lysozyme breaks down the cell wall of bacteria.

6.Interleukin-1: Il-1 induces fever and the production of acute phase proteins, some of which are antimicrobial because they can opsonize bacteria (Janeway *et al*. 2001).

C. Cellular barriers to infection

Part of the inflammatory response is the recruitment of polymorphonuclear, eosinophils and macrophages to sites of infection. These cells are the main line of defense in the non-specific immune system (Janeway *et al*. 2001).

1. Neutrophils – These are recruited to the site of infection where they phagocytose invading organisms and kill them intracellularly.

2. Macrophages – These also function in phagocytosis and intracellular killing of microorganisms. These are capable of extracellular killing of infected or altered self target cells. Furthermore, macrophages contribute to tissue repair and act as antigen-presenting cells.

3. Natural killer (NK) and lymphokine activated killer (LAK) cells –These can nonspecifically kill virus infected and tumor cells. These are not part of the inflammatory response but are important in nonspecific immunity to viral infections and tumor surveillance.

4. **Eosinophils** –these have proteins in granules that are effective in killing certain parasites (Alberts *et al.* 2002).

Response of phagocytes to infection:

Circulating PMNs and monocytes respond to danger (SOS) signals generated at the site of an infection. Some of the SOS signals stimulate endothelial cells near the site of the infection to express cell adhesion molecules such as ICAM-1 and selectins which bind to components on the surface of phagocytic cells and cause

the phagocytes to adhere to the endothelium. The phagocytes cross the endothelial barrier by "squeezing" between the endothelial cells in a process called diapedesis. Once in the tissue spaces some of the SOS signals attract phagocytes to the infection site by chemotaxis. The signals also activate the phagocytes, which results in increased phagocytosis and intracellular killing of the invading organisms.

Initiation of Phagocytosis

Phagocytic cells have a variety of receptors on their cell membranes through which infectious agents bind to the cells. These include: 1. Fc receptors, 2.complement receptors, 3. scavenger receptors and 4. toll-like receptors (Janeway *et al*. 2001).

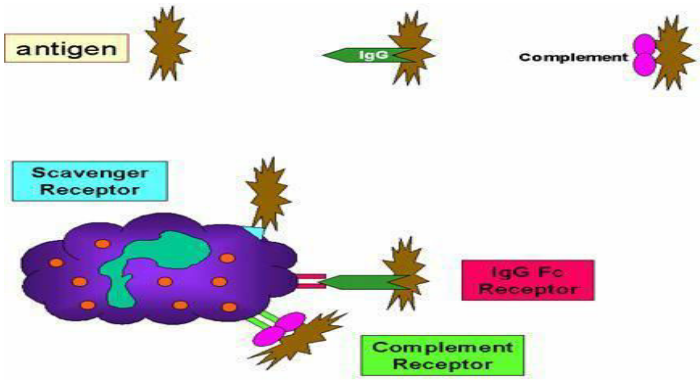


Fig. 3: Adherence of bacteria via receptors.

Phagocytosis

After attachment of a bacterium, the phagocyte begins to extend pseudopods around them. The pseudopods eventually surround the bacterium and engulf it, and the bacterium is enclosed in a phagosome. During phagocytosis the granules or lysosomes of the phagocyte fuse with the phagosome and empty their contents. The result is a bacterium engulfed in a phagolysosome which contains the contents of the granules or lysosomes.

Respiratory burst and intracellular killing:

During phagocytosis there is an increase in glucose and oxygen consumption which is referred to as the respiratory burst. The consequence of the respi-ratory burst is that a number of oxygen-containing compounds are produced which kill the bacteria being phagocytosed (Janeway *et al*. 2001).

Nitric oxide-dependent killing:

Binding of bacteria to macrophages, particularly binding via Toll-like receptors, results in the production of TNF-alpha, which acts in an autocrine manner to induce the expression of the inducible nitric oxide synthetase gene resulting in the production of nitric oxide. Nitric oxide released by the cell is toxic and can kill microorganism in the vicinity of the macrophage (Janeway *et al*. 2001).

Acquired immunity:

Definition: Immunity acquired by infection or vaccination (active immunity) or by the transfer of antibody or lymphocytes from an immune donor (passive immunity) is known as acquired immunity (Webster medical dictionary) The adaptive immune system is composed of highly specialized, systemic cells and processes that eliminate or prevent pathogenic challenges.

Functions: Adaptive immunity is triggered in vertebrates when a pathogen evades the innate immune system and generates a threshold level of antigen. Its functions are the recognition of specific "non-self" antigens, the generation of responses to eliminate specific pathogens or pathogen infected cells and the development of immunological memory (Janeway *et al*. 2001).

Effector cells: The cells of the adaptive immune system are lymphocytes. B cells and T cells are the major types of lymphocytes. The human body has about 2 trillion lymphocytes, constituting 20- 40% of white blood cells; their total mass is about the same as the brain or liver (Alberts *et al.* 2002). B cells play a large role in the humoral immune response, whereas T-cells are intimately involved in cell-mediated immune responses. In an adult animal, the peripheral lymphoid organs contain a mixture of B and T cells in at least three stages of differentiation: naive cells, effector cells and memory cells (Janeway *et al*. 2001).

2.5 Bacteria causing surgical site infections

Streptococci

Streptococci form chains and are gram positive on staining. The most important is the â-heamolytic *streptococcus*. *Streptococcus pyogenes* is the most pathogenic. It has the ability to spread, causing cellulitis, and to cause tissue destruction through the release of enzymes such as streptolysin, streptokinase and streptodornase. *Streptococcus faecalis* is often found in synergy with other organ-isms, as is the ã- haemolytic *streptococcus* and

Pepto streptococcus, which is an anaerobe. Both *Streptococcus pyogenes* and *Streptococcus faecalis* may be involved in wound infection after large bowel surgery. All the streptococci remain sensitive to penicillin and erythromycin. The cephalosporins are a suitable alternative in patients who are allergic to penicillin (Williams *et al*. 2008).

Staphylococci

Staphylococci form clumps and are gram positive. *Staphylococcus aureus* is the most important pathogen in this group and is found in the nasopharynx of up to 15 % of the population. It can cause exogenous suppuration in wounds. Strains

resistant to antibiotics (e.g. MRSA) can cause epidemics and more severe infection.

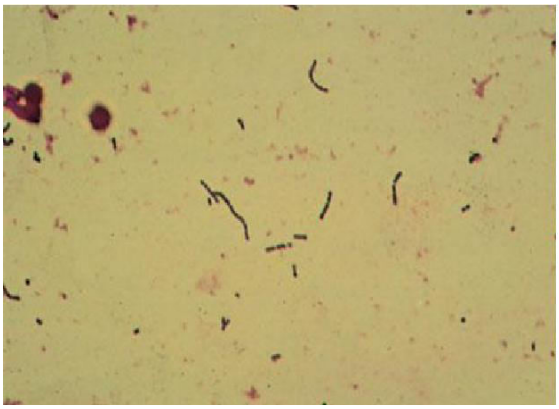


Figure 4**:** Streptococci.

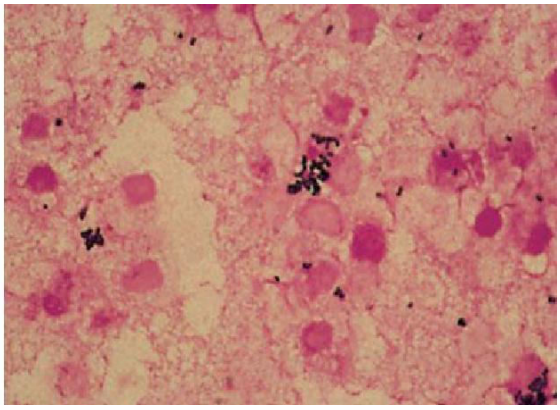


Figure 5: Staphylococcal pus.

It is controversial but, if MRSA infection is found in a hospital, all doctors, nurses and patients may need to be swabbed so that carrier can be identified and treated. In parts of northern Europe, the prevalence of MRSA infections has been kept at a very low level using 'search and destroy' methods which use these screening techniques and the isolation or treatment of carriers. Infections are usually suppurative and localized. Most hospital *Staphylococcus aureus* strains are now beta -lactamase producers and are resistant to penicillin, but most strains remain sensitive to flucloxacillin, vancomycin, aminoglycoside, some cephalosporin, and fusidic acid. There are several novel and innovative antibiotics becoming available that have high activity against resistant strains. Some have the advantage of good oral activity (linezolid), some have wide spectrum (tigecycline), have good activity in bacteraemia (daptomycin) but are

relatively expensive, and some have side effects involving marrow, hepatic and renal toxicity. Their use is justified but needs to be controlled by tight local policies and guide lines that involve clinical microbiologists.

Staphylococcus epidermidis, previously *Staphylococcus albus,* also known as coagulase negative staphylococci was regarded as a commensal but is now recognized as a major threat in prosthetic surgery and in indwelling vascular catheters. They can be multiply resistant to many anti-biotics and represent an important cause of Hospital Acquired Infection (HAI) (Williams *et al.* 2008).

Clostridia

Clostridial organisms are gram positive, obligate anaerobes, which produce resistant spores. Clostridium perfringes is the cause of gas gangrene and *C. tetani* causes tetanus after implantation into tissues or a wound. *Clostridium difficile* is the cause of pseudo membranous colitis. This is another HAI, now more common than the incidence of MRSA bacteraemia, which is caused by the overuse of antibiotics. The cephalosporins and other anti-staphylococcal antibiotics seems to be the most implicated, but the inappropriate sequential use of several antibiotics puts patient most at risk. The key symptom of bloody diarrhea can occur in small epidemic through poor hygiene. The elderly are

particularly at risk and in its most severe form, a severe colitis may lead to perforation and the need for emergency colectomy. There is a high mortality associated with this. Treatment involves resuscitation and antibiotic therapy with an imidazole or vancomycin. The fibrinous exudate is typical and differentiates it from other inflammatory conditions. The recognition of the toxin is an early accurate diagnostic test (Williams et al. 2008).

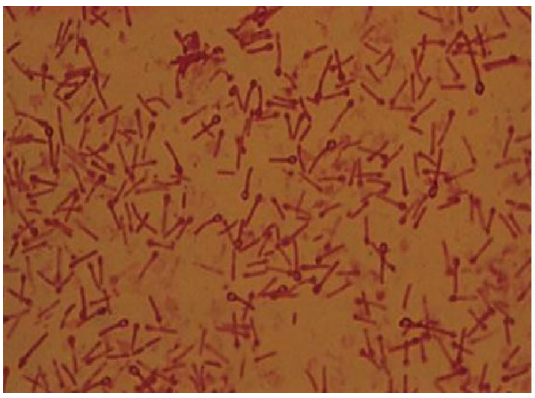


Fig. 6: *Clostridium tetani* (Drumstick spores).

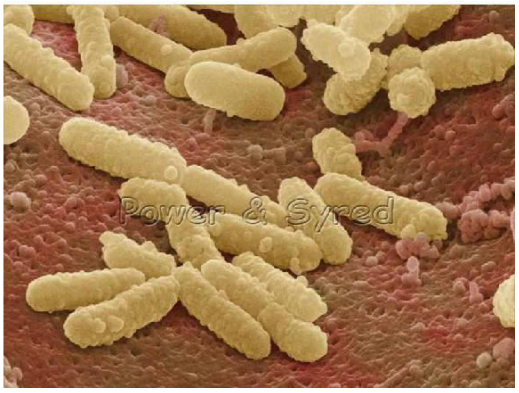


Fig.7: Scanning electron micrograph of E. *coli*

Aerobic gram- negative bacilli

These bacilli are normal inhabitants of the large bowel. *Escherichia coli* and *Klebsiella spp.* are lactose fermenting; proteus is non lactose fermenting. Most organisms of this group act in synergy with *Bacteroides* to cause SSI following bowel surgery.

Escherichia coli:

These are a major cause of the hospital acquired infection (HAI) of urinary tract, although most aerobic Gram-negative bacilli may be involved, particularly in relation to urinary catheterization (Williams *et al.* 2008).

In some study *E. coli* was found as the commonest cause of surgical site infection such as study conducted by Schnuriger *et al.* found that the most commonly isolated species in the presence of colonic injury was E. coli (64.7%) (Schnuriger et al. 2010).

It is one of the two important causes of neonatal meningitis and the agent most frequently associated with traveler's diarrhea. Some strains cause bloody diarrhea. *E. coli* is the most common facultative anaerobe in the colon and feces. It ferments lactose. It has three antigens that are used to identify the organism in epidemiological investigations: the O, or cell wall, antigen; the H or flagellar, antigen; and the K or capsular, antigen. The reservoir of *E. coli* includes both humans and animals. The source of *E. coli* that causes urinary tract infection is the patients own colonic flora ; in case of neonatal meningitis the source is mothers birth canal; in case of traveler's diarrhea it is acquired by ingestion of food or water contaminated with human feces. The main reservoir of enterohaemorrhagic *E. coli* O157 is cattle and the organism is acquired in under cooked meat. *E. coli* has several clearly identified components that

contribute to its ability to cause disease: pili, a capsule, endotoxin, and three exotoxins (Levinson 2005).

Pseudomonas

These *tend* to colonise burns and tracheostomy wounds, as well as the urinary tract. Once *Pseudomonas* has colonized wards and intensive care units, it may be difficult to eradicate. Surveillance of cross infection is important in outbreaks. Hospital strains become resistant to beta lactum antibiotics as resistance can be transferred by plasmids. Wound infections need antibiotic therapy only when there is progressive or spreading infection with systemic signs. The aminoglycosides are effective, but some cephalosporin and penicillin may not be. Many of the carbapenems, such as meropenem are useful in severe infections, whereas quinolones have been made ineffective through their over use (Williams *et al.* 2008).

Bacteroides

Bacteroides are non spore bearing, strict anaerobes that colonise the large bowel, vagina and oropharynx. *Bacteroides fragilis* is the principal organism that acts in synergy with aerobic gram negative bacilli (AGNB) to cause SSIs, including intra-abdominal abscesses, after colorectal or gynaecological surgery. They are sensitive to imidazoles (e.g. metronidazole) and some cephalosporins (e.g. cefotaxim) (Williams *et al.* 2008).

2.6 Hospital infections

Definition: Hospital acquired infection/Nosocomial infections are infections which are a result of treatment in a hospital or a healthcare service unit. Infections are considered nosocomial if they first appear 48 hours or more after hospital admission or within 30 days after discharge.

According to a new study, sepsis and pneumonia, two common conditions caused by hospital-acquired infections like MRSA, killed 48,000 Americans in 2006, and cost the nation over 8 billion dollars to treat. The researchers said that hospital-acquired infections are caused by "superbugs", germs that can't be killed with common antibiotics (Eber *et al.* 2010).

Physiology:

Micro-organisms are normally prevented from causing infection in tissues by intact epithelial surfaces. These are broken down in trauma and by surgery. In addition to these mechanical barriers, there are other protective mecha-nisms, such as, chemical, humoral and cellular barriers (Williams *et al.* 2008). All these natural mechanisms may be compromised by surgical intervention and treatment. Reduced resistance to infection has several causes. Host response is weakened by malnutrition, which can be recognised clinically, and most easily,

as recent rapid weight loss. Metabolic diseases such as diabetes mellitus, uraemia and jaundice, disseminated malignancy and AIDS are other contributors to infection. Iatrogenic causes including the immuno-suppression caused by radiotherapy, chemotherapy or steroids and when enteral feeding is suspended during the perioperative period, bacteria tend to colonise the normally sterile upper gastrointestinal tract. They may then translocate to the mesenteric nodes and cause the release of endotoxins, which further increases susceptibility to infection and sepsis, through activation of macrophages and pro-inflammatory cytokine release. The use of selective decontamination of the digestive tract (SDD) is based on the prevention of this colonisation (Williams *et al.* 2008).

Opportunistic infection:

In the circumstances of reduced resistance, bacteria that

are not normally pathogenic may start to behave as pathogens. This is known as opportunistic infection. Opportunistic infection with fungi is an example, particularly when prolonged and changing antibiotic regimens have been used (Williams *et al.* 2008).

Risks of developing surgical site infection:

The chance of developing an SSI after surgery is also determined by the pathogenicity of the organisms present and by the size of the bacterial inoculum. Devitalised tissue, excessive dead space or haematoma, all the results of poor surgical technique, increase the chances of infection. The same applies to foreign materials of any kind, including sutures and drains. If there is a silk suture in tissue, the critical number of organisms needed to start an infection is reduced logarithmically. Silk should not be used to close skin as it causes suture abscesses for this reason. These principles are important in prosthetic orthopaedic and vascular surgery, when large quantities of foreign material are deliberately left in the wound (Williams *et al.* 2008).

Decisive period: There is a delay before host defences can become mobilised after a breach in an epithelial surface. The acute inflammatory, humoral and cellular defences take up to 4 hours to be mobilised. This is called the 'decisive period', and it is the time when the invading bacteria may become established in the tissues. It is therefore logical that prophy-lactic antibiotics should be given to cover this period and that they could be decisive in preventing an infection from developing. The tissue levels of antibiotics should be above the minimum inhibitory concentration for the pathogens likely to be encountered (Williams *et al.* 2008).

Major and minor surgical site infections:

A **major SSI** is defined as a wound that either discharges significant quantities of pus spontaneously or needs a secondary procedure to drain it. The patient may have systemic signs such as tachycardia, pyrexia and a raised white count.

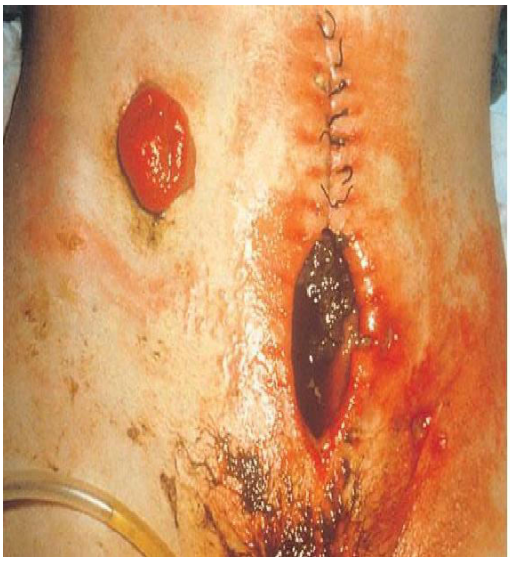


Fig. 8: Major wound infection.

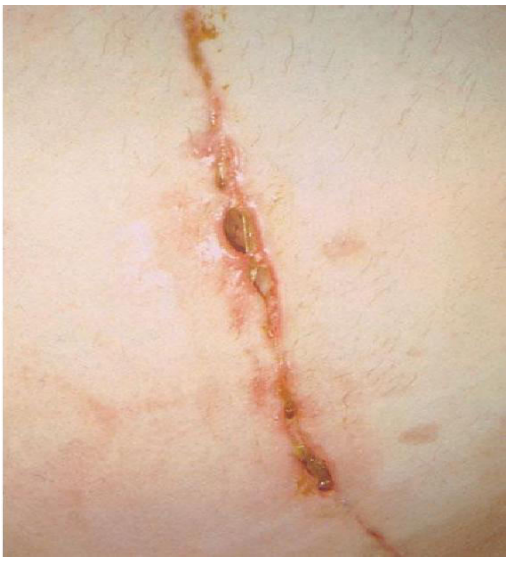


Fig. 9: Minor wound infection.

Minor wound infections may discharge pus or infected serous fluid but should not be associated with excessive discomfort, systemic signs or delay in return home. The differentiation between major and minor and the definition of SSI is important in audit or trials of antibiotic prophylaxis.

Surveillance for surgical site infection:

Accurate surveillance can only be achieved using trained, unbiased and blinded assessors. The US Centers for Disease Control (CDC) definition insists on a 30-day follow-up period for nonprosthetic surgery and 1 year after implanted hip and knee surgery (Williams *et al.* 2008).

Types of localised infection:

Abscess: An abscess presents all the clinical features of acute inflammation originally described by Celsus: calor (heat), rubor (redness), dolour (pain) and tumour (swelling). To these can be added functio laesa (loss of function). They usually follow a puncture wound of some kind, as well as surgery, but can be metastatic in all tissues following bacteraemia (Williams *et al.* 2008).

Cellulitis and Lymphangitis: Cellulitis is the non-suppurative invasive infection of tissues. There is poor localisation in addition to the cardinal signs of inflammation. Spreading infection presenting in surgical practice is typically caused by organisms such as â-haemolytic streptococci, staphylococci and *C. perfringens.* Tissue destruction, gangrene and ulceration may follow, which are caused by release of proteases. Systemic signs are common: SIRS, chills, fever and rigors. These follow the release of organisms, exotoxins and cytokines into the circulation. However, blood cultures are often negative. Lymphangitis is

part of a similar process and presents as painful red streaks in affected lymphatics. Cellulitis is usually located at the point of injury and subsequent tissue infection. Lymphangitis is often accompanied by painful lymph node groups in the related drainage area (Williams *et al.* 2008).

2.7 Prevention of surgical site infections

Protection of surgical patients from infection is a primary consideration throughout the preoperative, peroperative and postoperative phases of care. Bacterial infection of surgical incisions may have results that range from inconvenience to disaster, from small stitch abscess to massive tissue necrosis, septicaemia and even death. Some of the factors that determine surgical site infection and its consequences are beyond the control of surgeons. But others can be controlled. The preventive measures against surgical site infection are described below.

Aseptic measures in operation theatre:

Operation theatre complex: It should be scientifically planned, including barrier system, located away from the inpatient area and located on the top floor. Operation theatre complex should be consists of four zones: outer zone, restricted/ clean zone, aseptic zone and disposal zone. Outer zone is the area for receiving patients, messengers, toilets and administrative function. Restricted

zone/ clean zone consists of changing room, patient transfer area, stores room, nursing staff room, anaesthetist room, and recovery room. Aseptic zone consist of scrub area, preparation room, operation theatre and area for instrument packing and sterilization. Disposal zone is the area where used equipment are cleaned and biohazardous waste is disposed.

Criteria of an ideal operation room:

It should be big enough for free circulation, having two openings—towards scrub area and towards sterile area, openings fitted with swing door, well ventilated, air conditioned by- "High efficacy positive pressure air filter" system. As per US Public Health services minimum requirement for opera-ting room air are 25 changes per hour , positive pressure compared with corridors, temperature between 18 and 24º C and humidity of 50 to 55% .

Operation table to be kept away from the entrance and head end should be close to the sterile area (Modi 2010).

Cleaning and disinfection of operating room:

Cleaning, disinfection and sterilization are the cornerstones in ensuring operation room asepsis. Cleaning is a form of decontamination which removes organic matter and visible soils that interfere with the action of disinfectant, reduces the bacterial count and can be done by scrubbing with detergents and rinsing with water. For disinfection Phenol (Carbolic acid 2%) is used, to wash

floor every day after surgery, mop operating room walls, tables, mats, instrument trolleys, stools followed by a wipe done with 70% alcohol (Modi 2010).

Formaldehyde fumigation:

This procedure is commonly used to sterilize the operating room (OR). Fumigation is advised at weekly intervals. For an area of 1000 cubic feet 500 ml of 40% formaldehyde in one litre of water, stove or hot plate for heating formalin and 300 ml of 10% Ammonia are required.

Procedure: Close all doors & windows air tight and switch off fans and A.C. Heat formalin solution till boiling dry. Leave the OT unentered over night. Enter the OT next day morning with 300ml of ammonia. Keep the ammonia solution for 2-3 hrs to neutralize formalin vapours then open the OT to start surgery (Modi 2010).

Ultra violet radiation:

Daily U.V. irradiation for 12 -16 hours is a useful procedure. It is to be switched off 2 hours before surgery (Modi 2010).

Hand Washing Procedure:

Remove watch and other jewellery. Turn on the tap using the elbow and wet hands and forearm from finger tips to elbows, so that water runs down from fingers to elbow. Apply soap and scrub each hand with the other. Use rotatory

movements from fingertips to elbows with special attention to the nails and the webs of fingers. Rinse thoroughly under running water in the same manner as above. Scrub with soap and water for 7-8 minutes. With povidone iodine or chlorhexidine solution, scrubbing twice for 1 – 2 minutes each is adequate. Close tap with elbow taking care not to touch any spot that has been scrubbed. Dry with a sterile towel, begin with hands and proceed to wrist and forearm. Iodophor or an alcohol is applied following the surgical scrub. Approximately 3-5 ml of alcohol for 5 minutes is rubbed until the hands are dry. The proper method of wearing sterile gown and gloves to be followed. After wearing sterile gloves wash hands with balanced Salt solution or Ringer's lactate to remove talc from the gloves (Modi 2010).

Asepsis and Antisepsis

The term asepsis describes methods for preventing contamination of wounds and other sites by ensuring that only sterile objects and fluids come into contact with them; the risks of airborne contamination are minimized. Antisepsis is the use of solutions, such as chlorhexidine, iodine or alcohol, for disinfection (Kirk and Ribbans 2004).

Theatre clothing

1. Gowns: Woven cotton clothing is relatively ineffective at preventing the passage of bacteria. Disposable non-woven fabric, Goretex or tightly woven polycottons should be choosen. It is an important part of theatre discipline to change into fresh theatre clothing when entering the theatre suite because clothing worn in ward areas has been shown to be more heavily contaminated with micro-organisms than freshly laundered 'scrub suits'. If clothes become wet, they should be changed. 'Soak through' of blood has been shown to occur in over one third of orthopaedic and general surgical operations and may present a risk to the wearer. The risk should be reduced by using impermeable gowns or by wearing plastic aprons under linen gowns in situations where 'soak through' is likely. Clothing made from disposable non-woven fabric is suitable but expensive, as the whole team must wear it to obtain a benefit. Breathable membrane fabrics such as Goretex, or other materials such as tightly-woven washable polycottons are also suitable. Special attention should be paid to the design of the clothing so that bacteria are not 'pumped out' at the neck or the ankles. Most effective of all is the Charnley exhaust gown (Kirk and Ribbans 2004).

2. Mask: Its use is controversial. Few bacteria are discharged from the mouth and nose during normal breathing and quiet conversation, and it is argued that

for general abdominal operations masks are not required for the protection of the patient, particularly by staff members in theatre who are not directly assisting. Mask should be changed for each operation; reuse and manipulation simply contaminates the outside of the mask with skin commensals. Masks should be worn in prosthetic implant surgery. An efficient mask must be capable of arresting low velocity droplets. Paper masks should not be used.

Disposable masks made of synthetic fibres are better. Surgical antifog masks with flexible nosebands are available; they follow facial contours and retain a high efficiency of filtration. Masks continue to be worn to provide protection for the wearer against blood borne viruses as part of a policy of universal precautions. Full face visors also afford similar protection (Kirk and Ribbans 2004).

3. **Eye protection/visors:** These protect mucous membranes and should be worn during any procedure that is likely to generate droplets of blood or other body fluids, in order to protect mucous membranes from blood-borne viruses. A variety of lightweight anti-fog goggles, glasses and visors are available that do not obstruct vision (Kirk and Ribbans 2004).
4. **Tie up long hair:** Hair should be covered completely with a close-fitting cap made of synthetic material. Beards also should be covered fully (Kirk and Ribbans 2004).

5. **Foot wear:** It has a minor role in spreading infection. Clean, comfortable, antislip and antistatic shoes should be worn. If, there is a risk of fluid spillage, ankle length boots that can be cleaned with warm soapy water should be worn. They should be sufficiently robust to protect feet from sharps injury.

6. **Gloves**: It protects both surgeon and patient from blood-borne viruses and prevents the wound from becoming contaminated with the surgeon's skin flora. Single-use surgical gloves from a reputable source, sterilized by irradiation should be used. Nonlatex gloves without powder are better. Gloves should be inspected at the end of each operation for the presence of any hole (Kirk and Ribbans 2004).

7. **Theatre air**

Air-borne bacteria are generally believed to be a source of postoperative sepsis. The number of circulating bacteria is directly related to the number of people in theatre, and their movements, which should both therefore be minimized. Carefully balanced ventilation systems will not operate optimally if theatre doors are left partly open. General operating theatres are equipped with positive pressure or plenum ventilation systems, with the pressure decreasing from theatre to anaesthetic room to entrance lobby. Thus airborne microorganisms tend to be carried out rather than in. In a conventional plenum system there should be a minimum of 20 air changes per hour. Ultraclean air systems are

advocated for prosthetic implant surgery. In these systems, instead of the turbulent airflow associated with plenum pressure systems, there is unidirectional or laminar airflow at about 300 air changes per hour. The air is recirculated through high efficiency particulate air (HEPA) filters. This produces a reduction in circulating microorganisms compared with a conventional system. In these theatres regular bacterio-logical assessment should be undertaken (Kirk and Ribbans 2004).

Surgeon preparation

1. In order to minimize the risk of transmitting infection to patients, a surgeon must all satisfy local occupational health requirements before entering the operating theatre. For example, a surgeon must not operate with bacterial pharyngitis, during the prodromal period of a viral illness or with chronic or infected skin conditions. Surgeon should try to avoid operating if he/she has cuts, cracks, sores or rashes on his/her hands or forearms being brought to the surface. At the start of a list a surgeon should have an initial scrub of 3-5 minutes; thereafter, effective hand-washing with an antiseptic between cases is sufficient. Sterile, single-use brushes of polypropylene should be used

Shower prior to operating should not be practiced, as it increases the number of bacteria shed from the skin (Kirk and Ribbans 2004).

3. Antiseptics commonly used for hand washing are 4% Chlorhexidine gluconate (Hibiscrub), Hexachlorophane (pHisoHex) or Povidone-iodine (Betadine).

4. Surgeon's hands should be dried thoroughly using single-use sterile towels. Hot-air drying machines are not recommended (Kirk and Ribbans 2004).

Preparation of the patient

1. The longer a patient stays in hospital before operation, the greater the likelihood of a subsequent wound infection. Hospital stay should be as short as possible. The patient should be socially clean prior to operation. Infections at other sites increase the risk of surgical wound infection; therefore, diagnose and treat pre-existing infections before elective operation. Similarly, consider eradicating MRSA carriage in colonized patients prior to elective surgery.

2. The patient can be transported to theatre in bed directly, after being changed into a clean operating gown. Remove ward blankets before entering theatre. Trolleys must be cleaned daily.

3. Shaving of the operation site increases wound infection rates because of injury to the skin. If hair removal is necessary, clippers or depilatory cream can be used. If it is essential to shave the area, it should be performed as near as possible to the time of operation, preferably by the surgeon, prior to scrubbing up.

4. The skin area around and including the operation site should be prepared, first, with detergent for cleaning and degreasing, then with antiseptic solutions. For intact skin consider alcoholic solutions of chlorhexidine or povidone-iodine rather than aqueous solutions. Care should be taken regarding fire hazard when applying alcohol solutions and using diathermy. For vaginal or perineal disinfection consider a solution of chlorhexidine and cetrimide (Savlon).

5. Traditionally the periphery of the proposed incision site was protected with sterile cotton drapes; however, these soon become wet, diminishing their protective properties. Incisional plastic drapes have been advocated but Cruse & Frood in 1980 showed that applying adhesive plastic drapes to the operation area does not decrease the wound infection rate; this has since been confirmed in a study of caesarean section (Kirk and Ribbans 2004).

Cleaning and disinfection of instruments

Decontamination, or the process of removing microbial contaminants, can be carried out by cleaning, disinfection or sterilization.

Cleaning: It is a process that removes visible contamination but does not necessarily destroy microorganisms. It is a necessary prerequisite to effective disinfection or sterilization (Kirk and Ribbans 2004).

Disinfection: It is a process that reduces the number of viable micro-organisms to an acceptable level but may not inactivate some viruses, hardy organisms such as mycobacteria and bacterial spores (Kirk and Ribbans 2004).

Antiseptic: A topical disinfectant that may safely be applied to epithelial tissues is known as an antiseptic. Antiseptics include chlorhexidine, iodophores such as povidone-iodine, triclosan and 70% alcohol (Kirk and Ribbans 2004).

1. **Disinfection by moist heat**: Disinfection of heat-tolerant items can be achieved reliably by exposure to moist heat; for items such as surgical equipment and bedpans it can be carried out using a washer-disinfector. Recommended time-temperature combinations are 71 °C for 3 min, 80°C for 1 min or 90°C for 12 s. Boiling water kills bacteria, some viruses including human immunodeficiency virus (HIV) and hepatitis B virus (HBV) and some spores. It does not sterilize. Soft water at 100°C at normal pressure for 10 min is satisfactory. Suitable instruments include specula, proctoscopes and sigmoidoscopes (Kirk and Ribbans 2004).

3. **Chemical disinfection:** It can be used where heat cannot. A good example is the use of glutaraldehyde 2% (Cidex). It is suitable for instruments that cannot be autoclaved, sharp cutting instruments, plastic and rubber items and endoscopes. It is effective against vegetative pathogens in 15 minutes and resistant pathogenic spores in 3 hrs. It is toxic, irritant and allergenic.

Caution: Instruments sterilized by this method should be thoroughly rinsed serially 2 to 3 times in trays filled with sterile water.

Other chemical disinfectants include hypochlorite solutions, chlorine dioxide, super-oxidized water and peracetic acid.

Sterilization of instruments

Definition: Sterilization is defined as the complete destruction of all viable microorganisms, including spores, viruses and mycobacteria (Kirk and Ribbans 2004).

Methods of sterilization: These are mainly of two types- physical and chemical.

PHYSICAL METHODS

1. **Autoclaving:**

Steam under pressure attains a higher temperature than boiling water and the final temperature is directly related to the pressure. Instruments can be reliably sterilized by steam under pressure using autoclaves. The process can kill bacteria, including *Mycobacterium tuberculosis,* viruses and heat-resistant spores. Autoclaving at 121˚C for 20 minutes at 15 lbs per square inch pressure effectively kills most micro-organisms and spores (Modi 2010). The preferred cycle is 134°C at 2 atmospheres for a holding time of

3 min, which entails a total cycle time of at least 30 min to reach the required temperature. Autoclaves should be centralized in specialized units, e.g. the sterile service department (SSD). Autoclaving is suitable for sterilizing metallic instruments, except sharp knives and fine scissors.

Types of autoclaves: Gravity displacement type, pre vacuum type and vertical or horizontal type.

Figure 11: Autoclaves (vertical).

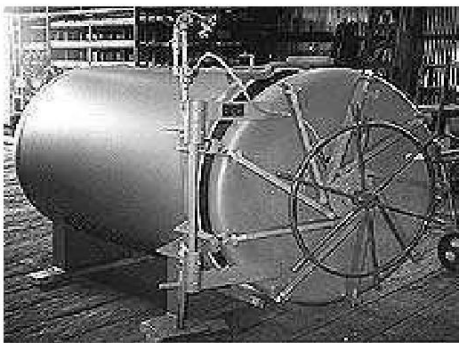


Figure 12: Autoclave (Horizontal)

Working of an autoclave:

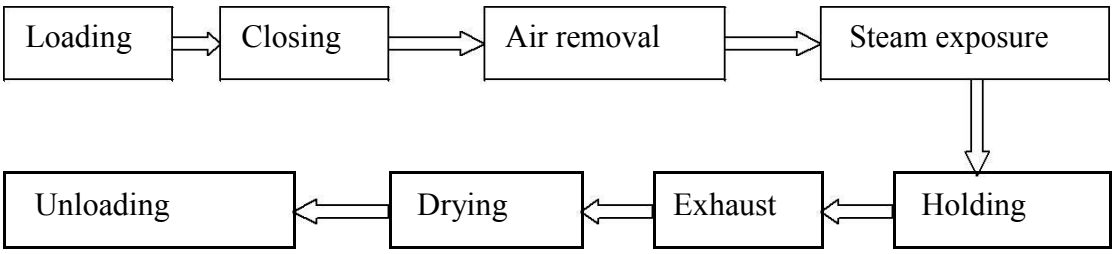


Figure 13: Various stages in the process of autoclaving.

Testing efficacy of autoclaves

Biological and chemical indicators are used to monitor the effectiveness of sterilization.

Biological indicators (BI): Commercially available spore strips (Hi-Media, Mumbai) impregnated with spores of *Bacillus steriothermophillus* are inserted in the cold compartment of the autoclave which is the lowest part of the chamber. After autoclaving of the load the strips are aseptically transferred in trypticase soy broth, and are incubated at 56° C for 5 days. The broth is examined intermittently for signs of turbidity.

Chemical indicator: Bowie–Dick tapes (signolac) show a change of color after exposure to sterilizing temperature when applied to the packs and articles in the load. The tape develops diagonal lines when exposed for the correct time to the sterilizing temperature.

Before sterilization

After sterilization

Figure 14: Bowie–Dick tapes (signolac), (Modi 2010).

2. Dry heat: Sterilization can be achieved by dry heat at 160°C for a holding time of 1 h. The process is inefficient compared with steam sterilization, but has the advantage of being able to treat non-aqueous liquids, ointments and airtight containers. It is also useful for avoiding corrosion of non-stainless metals and instruments with fine cutting edges, such as ophthalmic instruments. Do not use it for aqueous fluids or for materials that are likely to be damaged by the process, such as rubber and plastics. This equipment is subject to rigorous checks and maintenance.

3. Sterilants: These are chemical compounds that, under defined conditions, are able to kill bacterial spores (Kirk and Ribbans 2004).

a. Ethylene oxide (EO): It is a highly penetrative, noncorrosive agent with a broad cidal action against bacteria, spores and viruses. It is also flammable, toxic, irritant, mutagenic and potentially carcinogenic, and should not be used

when heat sterilization is possible. Its main uses are for wrapped and unwrapped heat-sensitive equipment. It is ideal for electrical equipment, flexible fibre endoscopes and photographic equipment. It should not be used for ventilatory equipment. EO sterilization is a mainly industrial process for single-use medical devices (Kirk and Ribbans 2004).

Figure 15: Ethylene Oxide (ETO) sterilization plant.

b. Glutaraldehyde: shorter immersion times provide disinfection, but 3-10 h of exposure to 2% alkaline glutaraldehyde is required for sporicidal activity (Kirk and Ribbans 2004).

c. Other sterilants: include peracetic acid, superoxidized water, gas plasma and chlorine dioxide; however, validation processes have not yet been established by the Department of Health for some of these newer technologies (Kirk and Ribbans 2004).

4. Irradiation (gamma rays):

This is a cold sterilization method with high penetrating power and lethal to DNA (Modi 2010). It is an industrial process suitable for sterilizing large batches of similar products, such as catheters and syringes (Kirk and Ribbans 2004). It is most useful for disposable and rubber items as well as ringer lactate (Modi 2010).

Spillages

Body fluid spillage should be removed as soon as possible. Gloves and a plastic apron should be worn. First, cover spills with an appropriate disinfectant, then absorbent paper towels. Discard as clinical waste (Kirk and Ribbans 2004).

Waste disposal

Hospital waste should be sorted to ensure it is correctly disposed of. 'SHARPS' must be Placed in approved containers, and clinical waste in yellow plastic bags. These are disposed of, usually by incineration, separately from domestic waste, which may be sent for landfill. Other categories of waste requiring segregation include pharmaceuticals and radioactive or cytotoxic waste (Kirk and Ribbans 2004).

Surgical Technique

Postoperative infection rate is influenced by the surgical techniques. The longer the operation, the more likely is the wound to become infected. Operations

should be performed as expediently as safety allows. Operative trauma should be kept to a minimum and handling of tissues must be gentle. Incisions should be made with sharp instruments as they are less likely to become infected than those produced, for example, by cautery; however, cautery may reduce the need for sutures, which can act as a nidus for infection. Finest suitable ligature should be used. Haematomas are at risk of becoming infected. Necrotic or ischaemic areas are also at risk. Leaving a dead space must be avoided. Unwarranted prophylactic drains should be avoided, which increase the risk of infection. A necessary drain should be inserted through a separate stab, not through the main wound. An entirely closed system should be used, and it should be removed as soon as possible to decrease the chance of ascending infection (Kirk and Ribbans 2004).

Surveillance

This is the systematic collection, collation, analysis and distribution of data. It has been shown to be valuable in the prevention of infection. The Study on the Efficacy of Nosocomial Infection Control (SENIC) was carried out in the USA over 10 years in the 1970s. A random sample of 1000 patients from each of 338 hospitals was studied and details about each patient were recorded and analysed. It was found that infections of the urinary tract were the most common nosocomial infections but, surgical site infections were the most

costly, both financially and in terms of delayed discharge from hospital. SENIC data measured intensity of surveillance, control efforts, policy development and teaching and whether or not infection rates were fed back confidentially to individual surgeons. In hospitals with optimal performance in all these categories the wound infection rate was 38% lower. The key factor in this reduction appears to be confidential feedback to individual surgeons. This is known as the Hawthorne effect. There are a number of different types of surveillance of nosocomial infection. Continuous hospital-wide surveillance may be expensive and time consuming, while targeted surveillance may be more practical and cost effective. Whichever method is employed, it is essential that the definitions of infection are clearly understood and reliably applied. Despite the drawbacks, surveillance is useful not only for feedback but also for identifying changes in epidemiology or a rise in infection rates, or for assessing the effect of implementing new preventive strategies (Kirk and Ribbans 2004).

Infection audit

Although a record of overall infection rates by surveillance is the ideal, this may not always be practical.Clinical audit is a way of reviewing clinical practice and outcomes and it has also been shown to be useful in surgical practice. It is important to audit infection rates. An acceptable standard exists and steps can be taken to improve rates in the process of closing the audit loop

Conclusion

Using these strategies, postoperative infection rate should be kept to a minimum. Awareness of infection rates and determination is a must to keep them comparable with rates in other similar units. It can be achieved by surveillance and infection audit (Kirk and Ribbans 2004).

2.8 Antimicrobial chemotherapy

Prophylactic Antibiotics

It has been shown that, for many contaminated and clean-contaminated procedures, postoperative infection can be avoided by using appropriate prophylactic antibiotics given prior to surgery (Kirk and Ribbans 2004).

The general principles of antibiotic prophylaxis:

1. Antibiotic prophylaxis should be used only when wound contamination is expected or when operations on a contaminated site may lead to bacteraemia. It is not required for clean-wound procedures except when an implant or vascular graft has been inserted, in valvular heart disease to prevent infective endocarditis, during emergency surgery in a patient with preexisting or recently active infection, if an infection would be very severe or have life threatening consequences (Kirk and Ribbans 2004).
2. There is no evidence that prolonged prophylaxis has any advantage over short courses - 24 hours. Prolonged administration may lead to superinfection. Normally in a clean operation one dose is sufficient. In contaminated operations three doses are often given (Kirk and Ribbans 2004).
3. Antibiotic should be administered parenterally, immediately prior to operation to achieve effective tissue levels. If they are given soon afterwards

they do not prevent infection. If the procedure continues for more than 3-4 hours, or if there is excessive blood loss, a further dose should be given in theatre (Kirk and Ribbans 2004).

Antibiotics should be selected to cover relevant organisms after discussion with the microbiologist regarding likely contaminants and local resistance patterns. Working together with the microbiologist to develop standard policies for the unit is very important, and they should be followed strictly when they are in place (Kirk and Ribbans 2004). The use of the newer, broad-spectrum antibiotics for prophylaxis should be avoided (Williams *et al.* 2008).

The Decisive Period

If antibiotics are given empirically, they should be used when local wound defences are not established (the decisive period). Ideally, maximal blood and tissue levels should be present at the time of making the first incision (Williams *et al.* 2008).

Examples of prophylaxis in different types of surgery:

Orthopaedic surgery: Here the main pathogens are staphylococci, so, Flucloxacillin is the best choice (Kirk and Ribbans 2004). Lower limb amputation should be covered against *C. perfringens* using 1.2 g of benzylpenicillin intravenously at induction of anaesthesia and 6-hourly thereafter for 48 hours (Williams *et al.* 2008).

Dental Surgery: Single doses of broad-spectrum penicillin, for example amoxicillin, orally or intravenously administered, are sufficient for dental surgery (Williams *et al.*2008).

Urology: A second-generation cephalosporin, such as cefuroxime, is sufficient (Williams *et al.* 2008).

Bowel Surgery: In bowel surgery cover is required for anaerobic and Gram-negative aerobic bowel flora (Kirk and Ribbans 2004). In open viscus surgery, the addition of an imidazole such as Metronidazole should be considered (Williams *et al.* 2008).

Principles of reduction of infection rate

Reduction of infection rate is not possible by concentrating attention in a single area. Control of resistant organisms in all areas within the hospital is mandatory. Aseptic and antiseptic principles should be obeyed strictly. The highest standard of surgical technique should be practiced. Use of prophylactic antibiotics should be logical. Audit of the results will play a major role to maintain and improve standards (Williams *et al.* 2008).

Control of resistant organisms

1. Antibiotics have been in used for more than 50 years and many organisms are now resistant to the older agents. For example, in many hospitals more than 50% of isolates of *Escherichia coli* are resistant to ampicillin.

2. The most obvious example is methicillin-resistant *Staphylococcus aureus* (MRSA). This is resistant to flucloxacillin and has to be treated with drugs such as the glycopeptides, Vancomycin and Teicoplanin. Even more worrying is the reported emergence of 'vancomycin intermediate *S. aureus* (VISA)' with reduced susceptibility to Vancomycin. It will be dangerous in future, if it become impossible to treat S. *aureus* infection.

3. Enterococci are also posing major problems with resistance; glycopeptide-resistant enterococci (GRE) are now found in many hospitals and they may cause life-threatening infections in immunocompromised patients.

Gram-negative organisms such as *Pseudomonas aeruginosa* may also be multiresistant. The increasing use of third-generation cephalosporins appears to be encouraging the emergence of Gram-negative bacilli such as *Klebsiella. pneumoniae* and *Enterobacter cloacae* resistant to these and other beta-lactams.

4.Hand washing and basic infection control practices cannot be overemphasized. Most hospital acquired infections are transmitted on the hands of staff and many studies have shown that hand washing is the single most important and successful method of controlling the spread of infection in hospital. Hands should be washed before and after physical contact with any patient and after any activity where they are likely to become contaminated.

They should be washed with soap, detergent, or with alcohol rubs or gels if they are not visibly soiled. Before carrying out any aseptic procedure, hands should be washed with an antiseptic solution such as povidone-iodine or chlorhexidine.

5. Screening of at-risk patients to identify those who are colonized is important. Reserve this, as a rule, for detecting MRSA so we can implement precautions to prevent spread of the organism to other patients, and also to reduce the risk of infection in those planned for high risk surgery such as vascular graft procedures and prosthetic orthopaedic surgery. Take swabs of nose, throat and perineum. If there is evidence of an outbreak in a unit, the infection control team may advise to screen the unit staff, in case there are carriers.

6. Patients found to be colonized with a significant multiresistant organism, should be isolated, usually in a side room - 'wound and enteric' or 'source' isolation. All staffs must wear disposable gloves and aprons when in contact with the patient. Doctors should remove their white coats before entering the side room. Ideally the same nurses should care for the patient throughout the shift. All other staff must be aware of, and take relevant precautions for, 'wound and enteric/ source isolation.'

The movement of colonized patients between departments should be controlled. Whenever possible, arrange for those carrying multiresistant organisms to be operated upon at the end of the surgical list, so that the theatre can be cleaned

thoroughly afterwards with minimum disruption. Warn the theatre staff of the patient's status in advance. The same applies to visits to other departments (Kirk and Ribbans 2004).

Principles of antimicrobial treatment

Antimicrobials may be used to prevent or treat established surgical infection. Antibiotics do not replace surgical drainage of infection. The use of antibiotics for the treatment of established surgical infection ideally requires recognition and determination of sensitivities of the causative organisms. Antibiotic therapy should not be held back if they are indicated, the choice being empirical and may later be modified depending on microbiological findings. However, once antibiotics have been administered, the clinical picture may become confused and, if a patient's condition does not rapidly improve, the opportunity to make a precise diagnosis may have been lost. It is unusual to have to treat SSIs with antibiotics, unless there is evidence of spreading infection, bacteraemia or systemic complications (SIRS and MODS). The appropriate treatment of localized SSI is interven-tional radiological drainage of pus or open drainage and debridement (Williams *et al.* 2008).

Two Approaches to Antimicrobial Therapy:

1. **A narrow-spectrum antibiotic:** These may be used to treat a known sensitive infection; for example, vancomycin for the treatment of MRSA.

2**. Combination of broad spectrum antibiotics:** These can be used when the organism is not known or when it is suspected that several bacteria, acting in synergy, may be responsible for the infection. For example during and after emergency surgery requiring opening of perforated or ischaemic bowel, any of the gut organisms may be responsible for subsequent peritoneal or bacteraemic infection. In this case, a triple therapy

combination of broad spectrum penicillin, an aminoglycoside and Metronidazole, may be used per and postoperatively to support the patients own body defense (Williams *et al.*2008).

Judicious use of antibiotics

Use of antibiotics should be judicious. These should be used only when there is evidence of clinical infection or as part of a policy regarding perioperative prophylaxis. Choice of antibiotic should be rational. If in doubt, consultation with the microbiologist earlier is better than later. Overuse of antibiotics encourages development of resistance in exposed organisms. It also destroys patients normal flora so they are more susceptible to colonization with hospital organisms. Furthermore, it predisposes to infection with

Clostridium difficile, which can lead to pseudomembranous colitis; third-generation cephalosporins are notorious for this (Kirk and Ribbans 2004).

Chapter 3
MATERIALS AND METHODS

MATERIALS AND METHODS

3.1 Type of study : Descriptive type of cross sectional study.

3.2 Study approval : Prior to commencement of this study - Thesis & Ethical Committee of Madras Medical College and Rajiv Gandhi Government General Hospital, chennai had approved the thesis protocol.

3.3 Place of study : Rajiv Gandhi Government General Hospital

3.4 Period of study : Duration starting from 01 May 2014 to 30 September 2014

3.5 Sample size : 140 cases

3.6 Selection of patients:

a) **Sampling method-** Purposive**.**

b) Inclusion criteria-

a) The patients having emergency nontraumatic abdominal operations.

b) Operations carried out in surgery dept. of Madras Medical College and Rajiv Gandhi Government General Hospital

c) Exclusion criteria: Patients with trauma were excluded from the study.

3.7 Study procedure:

Method of sampling was non-random, purposive. After admission short history was taken and physical examination was conducted on each patient admitted in surgery department with acute abdomen. Only very essential investigations were done urgently for taking correct decision about the management. Patients requiring emergency abdominal surgery and fulfilling the inclusion criteria were offered to participate in the study. All the traumatic cases were excluded from the study. All the necessary information regarding the study was explained to the patients or their valid guardian. Informed written consent was taken from the patients or their guardian willing to participate in the study. Detailed history was taken from the study group to establish proper diagnosis and to know about the presence of the risk factors regarding surgical site infection. Thorough physical examination was done in each case. Only essential investigations were done for proper diagnosis and reduction of risk. Data collection sheets were filled in by the investigator himself. All of the preoperative factors related to SSI present in the patient were noted down in the data sheet. After proper resuscitation (where applicable) and preparation, patients were sent to operation theatre for operation. Strict aseptic precautions were followed during the operation. Meticulous techniques were practiced as far as possible. The

operation procedure and related peroperative factors were observed directly and recorded in the data collection sheet instantly. During the postoperative period all the patients were closely monitored everyday up to the discharge of the patient from the hospital. If any symptom or sign of infection appear during this period then proper investigation was instituted for the diagnosis of infection and to assess the type and severity of the infection. If any collection of pus identified it was drained out and sent for culture and sensitivity test. Proper antibiotic was given to every patient both pre-operative and post-operative periods. Appropriate management was given to each of the patients of surgical site infection. Antibiotic was changed where necessary after getting the report of culture and sensitivity test. Postoperative events were recorded in the data sheet during every day follow up. After completing the collection of data it was compiled in a systematic way.

3.8 Operational definitions:

Surgical site infection (SSI): Infections in the area of operational wound within 30 days of operation, confirmed by microbiological examination were regarded as SSI.

Obesity: BMI was calculated in each patient. Those with BMI > 30 were regarded as obese.

Malnutrition: Those with BMI < 18.5 were regarded as suffering from malnutrition.

Jaundice: Those with **S**. bilirubin **>** 1.2 mg/ dl were recorded as jaundiced.

COPD: Suspected patients were diagnosed with the help of CXR.

Diabetes: Those known as diabetic from history and those with RBS more than 11 m mol/ l were included as diabetic.

Experience of surgeons: Surgeons were classified according to their designation and experience.

Duration of operations: Duration of operation was recorded during each operation.

Types of operations: were recorded during each operation.

Types of incisions: were recorded during each operation.

Delay to initiate operations: It was calculated from the time of onset of first symptom and time of starting operation.

3.9 Variables studied:

Dependent variable: Abdominal surgical site infection (SSI).

Independent variables:

i) Age

ii) Sex

iii) Educational status

iv) Co-morbidities: COPD, jaundice, diabetes, obesity and malnutrition.

v) Types of operations

vi) Types of incisions

vii) Duration of operation

viii) Delay to initiate operation

ix) Experience of surgeon

x) Types of wounds according to level of contamination

xi) Micro-organisms involved in SSI

xii) Sensitivity of the micro-organisms to antibiotics.

3.10 Ethical consideration

All the patients/ legal guardians were given an explanation of the study and about the investigative and operative procedures with their merits and demerits, expected results, and possible complications. If he/she agreed then the case had been selected for this study. The study did not involve any additional

investigation or any significant risk. It did not cause economic burden to the patients. The study was approved by the institutional review board prior to commencement of data collection. Informed consent was taken from each patient/guardian. Data were collected by approved data collection form.

3.11 Data collection

Data were collected by pre-tested structured questionnaire. Data were collected from all the respondents by direct interview after getting informed written consent from them or from their legal guardian.

3.12 Data analysis

Data analysis was done both manually and by using computer. Calculated data were arranged in systemic manner, presented in various table and figures and statistical analysis was made to evaluate the objectives of this study with the help of Statistical Package for Social Science (SPSS).

Chapter 4

RESULTS

RESULTS

This descriptive, cross-sectional study was carried out to determine factors responsible for surgical site infections following emergency non-traumatic abdominal operations that will be helpful in reducing rate of surgical site infections. One hundred and forty patients with emergency nontraumatic abdominal operations were selected purposively from Surgery department of Madras Medical College and Rajiv Gandhi Government General Hospital during the period of 1 may, 2014 to 30 september, 2014. All cases were evaluated clinically. Only essential investigations necessary for diagnosis and preoperative assessment were carried out before operations. Postoperatively swab was sent for culture and sensitivity test in every cases with discharge from the wound or collection of pus anywhere in the abdominal area. The patients of both sexes and different ages were included in the study.

Table I: Age distribution of the patients.

Age in years	Number of patients	Percentage (%)
10-19	31	22.14
20-29	30	21.42
30-39	30	21.42
40-49	34	24.28
50-59	9	6.43
60-69	6	4.29
Total	**140**	**100.00**

Mean ± SD = (32.93± 3.79) years.

It was observed that age of 140 patients ranged from 13-65 years. Most of the patients (89.29 %) were in between 10-49 years.

Table II: Surgical Site Infection (SSI) distribution by different age groups

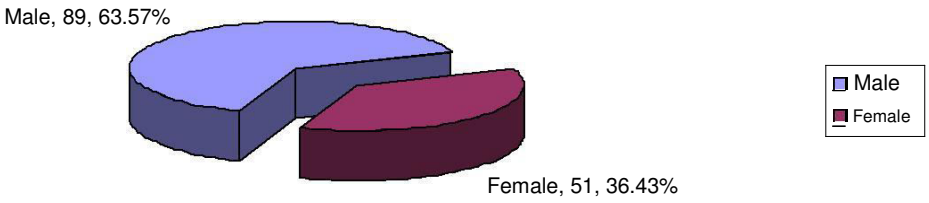
Age in years	SSI status		Total
	Yes	No	
10-19	5 (16.13)	26 (83.87)	31 (100.00)
20-29	2 (6.67)	28 (93.33)	30 (100.00)
30-39	5 (16.67)	25 (83.33)	30 (100.00)
40-49	9 (26.47)	25 (73.53)	34 (100.00)
50-59	2 (22.23)	7 (77.77)	9 (100.00)
60-69	1 (16.67)	5 (83.33)	6 (100.00)
Total	**24 (17.14)**	**116 (82.86)**	**140 (100.00)**

* Figures within parentheses indicate percentage.

$\div^2 = 4.596$; $P > 0.05$ df = 5

It was observed that rate of SSI in different age groups were as follows: 5 (16.13 %) in the 10-19 years, 2 (6.67 %) in the 20 - 29 years, 5 (16.67 %) in the 30 - 39 years, 9 (26.47 %) in the 40 - 49 years, 2 (22.23 %) in the 50 - 59 years and 1 (16.67 %) in the 60 - 69 years. It was highest 26.47 % (9 among 34) in the 40 - 49 years age group. However, these differences were not statistically significant.

Fig. 16: Pie diagram showing distribution of the patients by sex.



Regarding sex distribution, out of 140 patients, 89 (63.57 %) were male and 51 (36.43 %) were female. Male-female ratio was 1.74: 1.

Table III: Surgical Site Infection (SSI) distribution by Sex

Sex	SSI status		Total
	Yes	No	
Male	16 (17.98)	73 (82.02)	89 (100 .00)
Female	8 (15.69)	43 (84.31)	51 (100.00)
Total	24 (17.14)	116 (82.86)	**140 (100 .00)**

* Figures within parentheses indicate percentage.

$\div^{2} = 0.145$; $P > 0.05$ df = 1

Regarding sex distribution of SSI it was observed that among 89 male patients 16 (17.98 %) developed SSI, whereas among 51 female patients 8 (15.69 %) developed SSI. Rate of SSI was slightly higher in males. Sex difference in SSI was not statistically significant ($P > 0.05$).

Table IV: SSI distribution based on different educational status.

Educational status	SSI status		Total
	Yes	No	
Illiterates	6 (24.00)	19 (76. 00)	25(100.00)
Primary	2 (22.22)	7 (77.78)	9(100.00)
Secondary	6 (17.14)	29 (82.86)	35(100.00)
SSC	5 (18.52)	22 (81.48)	27(100.00)
HSC	2 (11.11)	16 (88.89)	18(100.00)
Graduation or above	3 (11.53)	23 (88.47)	26(100.00)
Total	**24 (17.14)**	**116 (82.86)**	**140**(100.00)

* Figures within parentheses indicate percentage.

$\div^2 = 2.33$; $P > 0.05$

df = 5

It was revealed that among 140 patients, 24 (17.14%) developed surgical site infection (SSI). Overall rate of SSI was 17.14 %. Regarding relationship between educational status and SSI it was observed that rate of SSI was highest 6 among 25 (24.00 %) in illiterates. It was 2 among 9 (22.22 %) in primary educated group, 6 among 35 (17.14 %) in secondary education group, 5 among 27 (18.52 %) in SSC passed, 2 among 18 (11.11%) in HSC passed and only 3 among 26 (11.53 %) in graduation or above group. It was observed that rate of SSI decreased with rise in level of education. However, association

between level of education and rate of SSI was statistically insignificant (P > 0.05).

Table V: Number of operations, SSIs and SSI rate (%) by category.

Types of operations	Status of SSI		Total
	Yes	No	
Appendicectomy	5 (8.33)	55 (91.67)	60 (100.00)
Adhesiolysis or resection and Anastomosis	3 (10.00)	27 (90.00)	30(100.00)
Repair of ileal perforation / Ileostomy and thorough peritoneal toileting	8 (42.10)	11 (57.89)	19(100.00)
Repair of duodenal ulcer perforation and thorough peritoneal toileting	3 (20.00)	12 (80.00)	15 (100.00)
Appendicectomy with peritoneal Toileting	4 (33.33)	8 (66.66)	12 (100.00)
Resection of Volvulus of sigmoid colon and primary anastomosis/ Hartmans Procedure	1 (50.00)	1 (50.00)	2 (100.00)
Herniotomy and herniorrhaphy	_	2 (100.00)	2(100.00)
Total	**24** (17.14)	**116** (82.86)	**140** (100.00)

* Figures within parentheses indicate percentage.

Out of 140 patients with emergency nontraumatic abdominal operations, rate of SSI in different operations were observed. It was found that out of 60 acute appendicitis cases 5 (8.33 %) developed SSI, out of 30 small intestinal obstruction cases 3 (10.00%) developed SSI, out of 19 ileal perforation cases 8 (42.10 %) developed SSI, out of 15 duodenal ulcer perforation 3(20.00 %) developed SSI, out of 12 burst appendix cases 4 (33.33 %) developed SSI, out of 2 sigmoid volvulus cases 1 (50.00 %) developed SSI and it was nil between 2 obstructed inguinal hernia cases. The highest rate of SSI (50.00 %) was in volvulus cases and lowest in obstructed hernia operation.

Table VI: SSI distribution based on different types of incision

Type of Incisions	Status of SSI		Total
	Yes	No	
Extended lower midline	1 (50.00)	1 (50.00)	2 (100.00)
Mid midline	8 (42.11)	11 (57.89)	19 (100.00)
Lower right para- median	4 (33.33 %)	8 (66.66)	12(100.00)
Rutherford Morison	3 (20.00)	12 (80.00)	15 (100.00)
Upper midline	2 (13.33)	13 (86.66)	15(100.00)
Extended upper midline	4 (13.33)	26 (86.66)	30(100.00)
Grid iron	2 (05.00)	38 (95.00)	40 (100.00)
Lanz	0 (00.00)	5 (100.00)	5(100.00)
Inguinal	0 (00.00)	2 (100.00)	2 (100.00)
Total	**24 (17.14)**	**116 (82.86)**	**140**(100.00)

* Figures within parentheses indicate percentage.

Rate of SSI was highest, 1 in 2 (50.00 %) operations done through extended lower midline incision, whereas rate of SSI was 8 among 19 (42.11 %) in mid midline, 4 among 12 (33.33 %) in lower right para-median, 3 among 15 (20.00 %) in Rutherford Morison, 2 among 15 (13.33 %) in upper midline, 4 among 30 (13.33 %) in extended upper midline and 2 among 40 (5.00 %) in grid iron incisions. No infection occurred in 5 operations done through Lanz incision and

2 operations through inguinal incisions.

TableVII: SSI distribution based on delay to initiate operation.

Delay to initiate operations (in hours)	SSI status		Total
	Yes	No	
< 6	1 (9.09)	10 (90.91)	11(100.00)
6- 12	2 (10.53)	17 (89.47)	19(100.00)
12 - 24	5 (15.63)	27 (84.37)	32(100.00)
24- 48	7 (18.42)	31 (81.58)	38(100.00)
48– 72	6 (19.35)	25 (80.65)	31(100.00)
> 72	3 (33.33)	6 (66.66)	9 (100.00)
Total	**24 (17.14)**	**116 (82.86)**	**140 (100.00)**

* Figures within parentheses indicate percentage.

With regard to association between delay to initiate operation and rate of SSI it was observed that the surgical site infection rates were 9.09%, 10.53%, 15.63%, 18.42%, 19.35% and 33.33% when operations were initiated <6, 6-12, 12-24, 24-48, 48-72 and >72 hours later respectively. The rate of infection increased as the time lapse between appearance of first symptom and initiation of operation were increased.

Table VIII: SSI distribution based on duration of operations

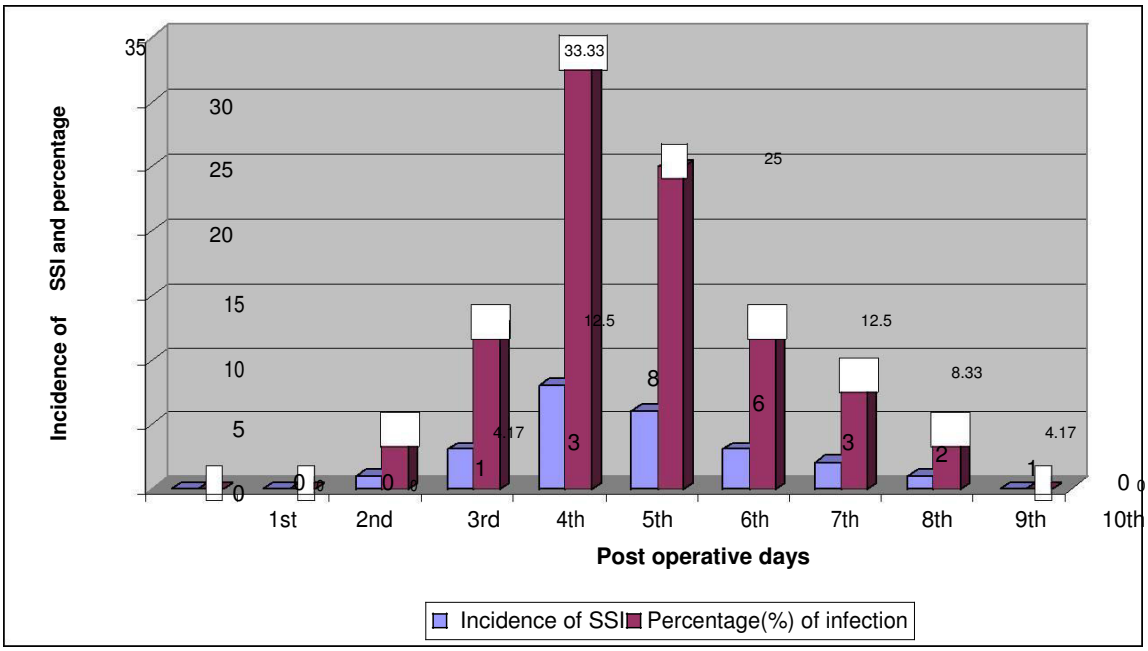
Duration of Operation	SSI status		Total
	Yes	No	
Less than 1 hour	4(4.60)	83 (95.40)	87(100.00)
1 to 2 hours	14 (32.55)	29 (67.45)	43(100.00)
More than 2 hours	6 (60.00)	04 (40.00)	10(100.00)
Total	**24(17.14)**	**116 (82.86)**	**140(100.00)**

* Figures within parentheses indicate percentage.

$\div^2 = 29.79$; $P < 0.001$ df = 2

With respect to duration of operation and percentage of SSI it was observed that 87 operations were done requiring less then one (<1) hour in each case; SSI developed in only 4 (4.60 %) of these cases. Whereas, 43 operations were completed between 1-2 hours each; among them 14 (32.55 %) developed SSI. It was observed that, 10 operations required more than 2 hours each; among these SSI occurred in 6 (60.00 %) cases. The rate of SSI increased with prolongation of duration of operation. The difference in percentage of SSI with duration of operation was statistically significant ($P < 0.001$).

Figure 17: Bar diagram showing incidence of SSI after emergency nontraumatic abdominal surgery in different post operative days.



In relation to appearance of infection on postoperative days it was observed that most of the infections (91.66 %) were started between 4th and 8th post operative days (POD) and it was highest 8 (33.33 %) on 5th POD. Among a total of twenty four patients with surgical site infections, in only one patient (4.17 %) features of infection first appeared on 3rd POD and it was three (12.50 %) on 4th, eight (33.33 %) on 5th, six (25 %) on 6th, three (12.50 %) on 7th, two (8.33 %) on 8th and one (4.17 %) on 9th POD.

Table IX: SSI distribution based on types of wounds by the degree of contamination.

Types of wounds	SSI status		Total
	Yes	No	
Clean	1(4.35)	22 (95.65)	23 (100.00)
Clean contaminated	5 (8.33)	55 (91.67)	60 (100.00)
Contaminated	3 (27.27)	8 (72.73)	11 (100.00)
Dirty	15(32.61)	31 (67.39)	46 (100.00)
Total	**24**(17.14)	**116** (82.86)	**140** (100.00)

* Figures within parentheses indicate percentage.

$\div^2 = 14.49$; $P < 0.01$ df = 3

In relation to different types of wounds, by the degree of contamination, it was observed that among 140 cases 23 were clean wounds; SSI developed only in 1 (4.35 %) of these clean cases. There were 60 clean contaminated cases, among them SSI occurred in 5 (8.33 %); whereas SSI developed in 3 among 11 (27.27 %) contaminated wounds. The rate of SSI was as high as 15 among 46 (32.61 %) dirty cases. The difference had high statistical significance ($P < 0.01$). It can be assumed that the infection rate increased with that of degree of wound contamination.

Table X: SSI distribution based on Co-morbidity status.

Co-morbidity status	SSI status		Total
	Yes	No	
With co-morbidity	17 (40.48)	25 (59.52)	42 (100.00)
Without Co-morbidity	7 (7.14)	91 (92.86)	98 (100.00)
Total	**24** (17.14)	**116** (82.86)	**140** (100.00)

* Figures within parentheses indicate percentage.

$\div^2 = 22.98$; $P < 0.001$

df = 1

In relation to co-morbidity, it was observed that 42 patients had co-morbid disorders associated with the main surgical disease and 98 patients had no co-morbid disorder. Among the patients with co-morbid disorders 17 (40.48 %) developed surgical site infection (SSI), whereas, in the patients without any co-morbidity only 7 (7.14 %) developed SSI. The difference of rate of infection between these two groups was very obvious. It was clear that associated co-morbid disorders played a vital role as a host related risk factor for SSI. Moreover, the difference was statistically highly significant ($P < 0.001$).

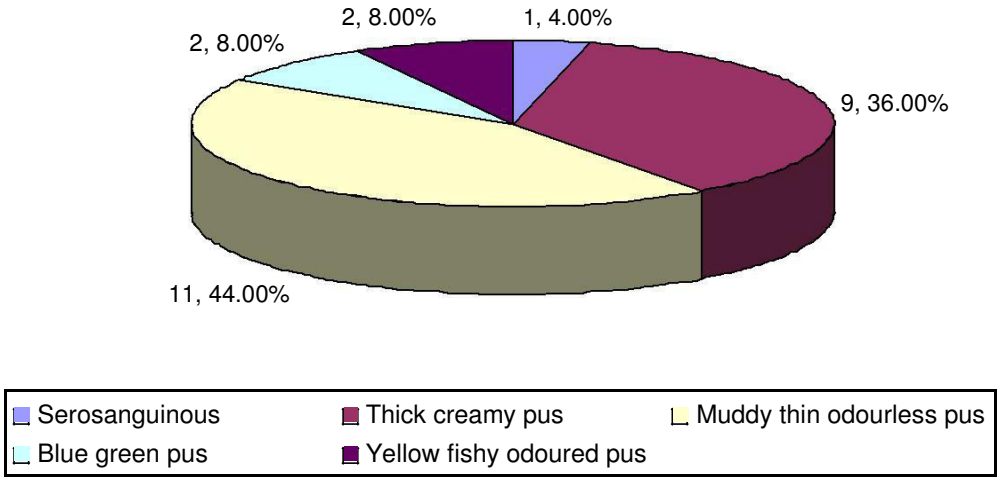
Table XI: Surgical site infection distribution based on presence of different co-morbidities.

Types of co-morbidity	SSI status		Total
	Yes	No	
Malnutrition	11 (45.12)	13 (54.17)	24 (100.00)
COPD	2 (28.57)	5 (71.43)	7 (100.00)
Diabetes Mellitus	2 (33.33)	4 (66.67)	6 (100.00)
Obesity	1 (33.33)	2 (66.67)	3 (100.00)
Medical Jaundice	1 (50.00)	1 (50.00)	2 (100.00)
Total	**17 (40.48)**	**25 (59.52)**	**42 (100.00)**

* Figures within parentheses indicate percentage.

In 24 patients with malnutrition 11(45.12 %) developed SSI, whereas among 7 patients with COPD 2 (28.57 %) developed SSI. 6 persons were diabetic, among them 2 (33.33 %) suffered from SSI. 3 persons were obese, 1 of them (33.33 %) developed SSI, whereas, 1of 2 (50.00 %) persons suffering from medical jaundice developed SSI.

Fig. 18: Frequency of various types of discharge/ pus from 25 wounds.



n = 25

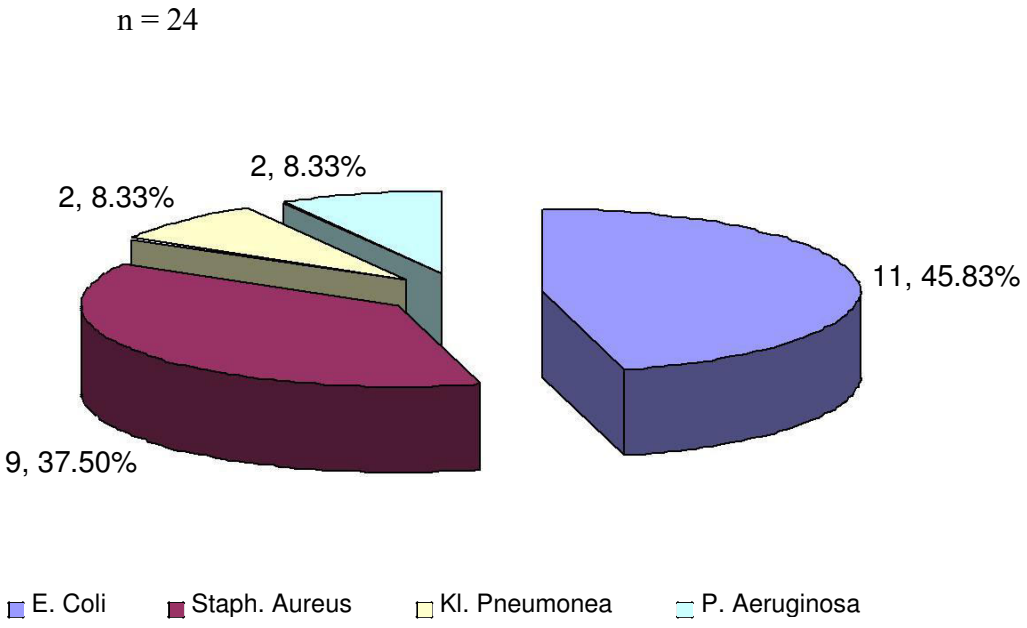
Among 140 patients, 25 developed some type of discharge from the wounds / collection of pus anywhere in the abdominal area. In eleven (44.00%) cases there were mudy thin odourless pus, in nine (36.00%) cases there were thick creamy pus, in two (8.00%) cases there were bluish green pus, in another two (8.00%) cases there were yellow fishy odoured pus and in one (4.00%) case there were serosanguinous discharge.

Table XII: Organisms isolated and cultured from different types of discharge from wound /collection of pus.

Character of discharge/ pus	Frequency	Organisms isolated
Thin muddy odourless pus	11	*Escherischia Coli*
Thick creamy pus	9	*Staphylococcus Aureus*
Yellow fishy odoured pus	2	*Klebsiella Pneumonea.*
Blue green pus	2	*Pseudomonus Aeruginosa*
Serosanguinous discharge	1	No growth
Total	**25**	

Twenty five samples of discharge/ pus from the wounds or peritoneal cavity were sent for culture and sensitivity test. Among them causative pathogens were detected in twenty four cases. *Escherischia coli* was found in 11cases with thin mudy odourless pus, *Staphylococcus aureus* in 9 cases with thick creamy pus, *Klebsiella* in 2 cases with yellow fishy odoured pus, *Pseudomonas aeruginosa* in 2 cases with bluish green pus and no growth was detected in 1 case with serosanguinous discharge.

Fig. 19: Pie diagram showing bacteria isolated from 24 surgical site infections.



E.Coli were found as the commonest organism (11 among 24 cases) causing 45.83 % of the surgical site infections. *Staph. Aureus* were the second most common organism (9 among 24 cases) causing 37.50 % of the infections. Each of *klebsiella* and *pseudomonas* were causing 8.33 % of the surgical site infections (found in 2 cases among 24 SSI).

Table XIII: Sensitivity pattern of the cultured micro-organisms to various antibiotics.

Name of micro-organisms (number of cases)	Antibiotics and their sensitivity in percentage (number of cases)						
	Ciprofloxacin	Cephradin	Cotrimoxazole	Flucloxacin	Nitrofurantoin	Ceftriaxone	Imipenem
Escherischia coli (11)	45.45 **(5)**	54.54 (6)	45.45 (5)	-	9.09 (1)	72.72 (8)	100 (11)
Staphylococcus Aureus (9)	44.45 (4)	44.45 (4)	-	55.55 (5)	-	88.9 (8)	100 (9)
Klebsiella pneumoniae (2)	-	50 (1)	50 (1)	-	-	100 (2)	100 (2)
Pseudomonus aeruginosa (2)	50 (1)	–	–	–	50 (1)	100 (2)	100 (2)

Escherischia coli were sensitive to Ciprofloxacin (45.45% cases), Cephradin (54.54% cases), Cotrimoxazole (45.45 % cases), Nitrofurantoin (9.09 % cases), Ceftriaxone (72.72% cases) and Imipenem (100% cases).

All the cases of E. *coli* were resistant to flucloxacillin. *Staphylococcus aureus* were sensitive to Ciprofloxacin (44.45% cases), Cephradin (44.45% cases), Flucloxacin (55.55% cases), Ceftriaxone(88.9% cases) and Imipenem (100% cases). But, all the cases of *Staph. aureus* were resistant to Cotrimoxazole and Nitrofurantoin.

Klebsiella pneumoniae were sensitive to Cephradin and Cotrimoxazole in 50 per cent cases each and to Ceftriaxone and Imipenem in all (100 per cent) cases. But, all the cases of *Kl. Pneumoniae* were resistant to Ciprofloxacin, Flucloxacillin and Nitrofurantoin.

Fifty (50) per cent cases of *Pseudomonus aeruginosa* were sensitive to Ciprofloxacin and Nitrofurantoin, and all the cases of *P. aeruginosa* (100%) sensitive to Ceftriaxone and Imipenem. All of them (100%) were resistant to Cephradin, Cotrimoxazole and Flucloxacillin.

All (100%) the organisms isolated were sensitive to Imipenem.

Chapter 5

DISCUSSION

DISCUSSION

This descriptive, cross-sectional study was conducted among 140 purposively selected patients with emergency non-traumatic abdominal operations conducted in surgery department Madras Medical College and Rajiv Gandhi Government General Hospital. The study was carried out with a view to determine the factors responsible for surgical site infections (SSI) following emergency non-traumatic abdominal operation which will be helpful in reducing the rate of surgical site infection in the near future.

Age of 140 patients ranged from 13-65 years. Most of the patients (125, 89.29 %) were in between 10-49 years; with mean age 32.93 years and standard deviation 3.79 years (Table I).

It was revealed that among 140 patients 24 (17.14%) developed surgical site infection (SSI). Overall rate of SSI was 17.14 % (Table II).

It was observed that rate of SSI in different age groups was 16.13 % in the 10-19 years, 6.67 % in the 20-29 years, 16.67 % in the 30-39 years, 26.47 % in the 40-49 years, 22.23 % in the 50-59 years and 20.00 % in the 60-69 years. It was highest 26.47 % (9 among 34) in the 40-49 years age group (Table II).

Regarding sex distribution of the patients, among the total 140 cases 89 (63.57 %) were male and 51 (36.43 %) were female. Male-female ratio was 1.74: 1(Fig.16). So, it can be assumed that males are more commonly affected by acute abdominal conditions requiring surgery. Rate of SSI in males were 17.98 %, whereas among females it was 15.69 % (Table III). Rate of SSI was slightly higher in males, which was not statistically significant.

It was observed that host factors like type of disease, presence/absence of co-morbidity and types of co-morbidity and other factors like seniority of surgeon, delay to initiate operation and duration of surgery were associated with the rate of surgical site infection.

Regarding educational status, it was observed that rate of SSI was highest, 6 among 25 (24.00 %), in illiterates. It was 2 among 9 (22.22 %) in primary educated group, 6 among 35 (17.14 %) in secondary education group, 5 among 27 (18.52 %) in SSC passed, 2 among 18 (11.11%) in HSC passed and only 3 among 26 (11.53 %) in graduation or above group (Table IV). It was observed that rate of SSI decreased with rise in level of education. However, the difference of rate of SSI among different groups of patients according to level of education was not statistically significant ($P > 0.05$).

Out of 140 patients with emergency nontraumatic abdominal operations, rate of SSI in different operations were as follows: 5 among 60 (8.33%) acute appendicitis cases, 3 among 30 (10.00%) small intestinal obstruction, 8 among 19 (42.10%) ileal perforation, 3 among 15 (20.00%) duodenal ulcer perforation, 4 among 12 (33.33%) ruptured appendix, 1 between 2 (50.00%) sigmoid volvulus and no SSI occurred in 2 obstructed inguinal hernia cases. The highest rate of infection (50.00%) was in volvulus cases and lowest in obstructed hernia operations (Table V). These findings were consistant with the result of Surgical Site Infection Survillance (SSIS) for general surgery which was published as Wexford General Hospital Surgical Site Infection (SSI) data report in 2009 showing number of SSI and rate of SSI (%) by category of operations. They done 132 appendicectomy, among them SSI occurred in 7 (5.3%) cases. SSI occurred in 10 (19.2 %) cases among 52 Colonic surgeries, 4 (23.5%) cases among 17 Small bowel surgery and 5(26.3%) cases among 19 Laparotomies. No SSI was reported among 82 herniorrhaphy cases (Surgical Site Infection Survillance for general surgery 2009).

Regarding incision-wise infection rate, rate of SSI was highest, 1 in 2 (50.00%) operations done through extended lower midline incision, whereas rate of SSI was 8 among 19 (42.11%) in mid midline, 4 among 12 (33.33%) in lower right para-median, 3 among 15 (20.00%) in Rutherford Morison, 2

among 15 (13.33%) in upper midline, 4 among 30 (13.33%) in extended upper midline and 2 among 40 (5.00%) in grid iron incisions. No infection occurred in 5 operations done through Lanz incision and 2 operations through inguinal incisions (Table VII). In present study infection rate was higher in midline incisions that may be attributed to less vascularity of the linea alba and most contaminated and dirty cases were operated through these incisions. The findings were consistent with the findings of study carried out by Paul in 2004, where the infection rate was 50.00 per cent for Rutherford Morison, 25 per cent for each of right para median and extended midline, 18.18 per cent for upper midline, 9.38 per cent for grid iron incision and nil for inguinal incision (Paul 2004).

With regard to delay to initiate operation and rate of SSI, it was observed that the surgical site infection rate was 9.09%, 10.53%, 15.63%, 18.42%, 19.35% and 33.33% when operation was initiated <6, 6-12, 12-24, 24-48, 48-72 and >72 hours later respectively. The rate of SSI increased as the time lapse between first manifestation of symptoms and initiation of operation prolonged (TableVIII).

With respect to duration of operation and percentage of SSI it was observed that the infection rate varies with duration of operation. It was only 4.6 % when the duration of operation was less then one hour. The rate rises with

the prolongation of operation. Infection rate was 32.55% when the duration of operation was between one and two hours. The infection rate was as high as 60.00 per cent when duration of operation was more then two hours (Table IX). The rate of SSI increased statistically very significantly with that of duration of operation ($P < 0.001$). It may be due to the prolonged exposure of the wound to the environment leading to more chance to inoculation of micro-organisms.

In relation to appearance of infection by features like fever, excessive pain, tenderness or discharge from the wound on postoperative days it was observed that most of the infections were started between 4th and 8th post operative days (PODs) and it was highest (33.33%) on 5th POD. Among a total of twenty four patients with surgical site infection, in only one patient (4.17%) features of infection first appeared on 3rd POD and it was three (12.50%), eight (33.33%), six (25%), three (12.50%), two (8.33%) and one (4.17%) persons who presents with features of infection on 4th, 5th, 6th, 7th, 8th and 9th POD respectively. No infection started on 1st, 2nd and 10th POD (Fig. 24).

In relation to different types of wounds, by the degree of contamination, it was observed that among 140 cases 23 were clean wounds, SSI developed only in 1 (4.35 %) of these clean cases. There were 60 clean contaminated cases, among them SSI occurred in 5 (8.33 %); whereas SSI developed in 3 among 11 (27.27%) contaminated wounds. The rate of SSI was as high as 15

among 46 (32.61%) dirty cases. The difference was statistical significant ($P < 0.01$). It was revealed that the infection rate increased with that of degree of wound contamination (Table X). These findings were consistent with the findings of 10 years prospective study of 62,963 wounds by Cruse and Frood in 1980, where infection rate was 1.5%, 7.7%, 15.2% and 40% in clean, clean contaminated, contaminated and dirty wounds respectively (Cruse and Frood 1980).

In relation to co-morbidity, it was observed that 42 patients had co-morbid disorders associated with the main surgical disease and 98 patients had no co-morbid disorder. Among the patients with co-morbid disorders, 17 (40.48 %) developed surgical site infection (SSI), whereas, in the patients without any co-morbidity only 7 (7.14 %) developed SSI (Table XI).

The difference of rate of infection between these two groups was very obvious. It was clear that associated co-morbid disorders played a vital role as a host related risk factor for SSI. Moreover, the difference was statistically significant ($P < 0.001$). It was observed that infection rate was 45.12 per cent in clinically malnourished patients, whereas it was 28.57 per cent in COPD cases and 33.33 per cent in obese patients. Moreover, two patients underwent laparotomy with medical jaundice. Of them one (50 %) developed SSI. In addition six patients with diabetes mellitus underwent emergency abdominal

surgery. Of them two patients (33.33%) developed SSI (Table XII). Israelsson and Jonsson identified increased rate of SSI among overweight patients (Israelsson and Jonsson 1997). Another study by Cruse and Frood showed that clean wound infection rate rises to 10.7% in patients with diabetes, 13.5% in obesity and 16.6% in malnourished patients (Cruse and Frood 1980).

Among 140 patients, 25 developed some type of discharge from the wounds/ collection of pus anywhere in the abdominal area. In 11cases there were mudy thin odourless pus, in 9 cases there were thick creamy pus, in 2 cases there were bluish green pus, in another 2 cases there were yellow fishy odoured pus and in 1 case there was serosanguinous discharge (Fig. 18). Sample of pus or discharge from wound were sent for culture and sensitivity test in these 25 cases. One of them with serosanguinous discharge showed no growth, but the remaining 24 showed growth of various micro-organisms. *E.Coli* were found in 11 (45.83%) cases, the commonest organism causing surgical site infections (SSI). *Staph. Aureus* were the second most common organism found in 9 (37.50%) cases. Each of *klebsiella* and *pseudomonas* were causing 2 (8.33%) cases of SSI (Fig. 19).

For the prevention of surgical site infection antibiotics such as Ceftriaxone, Cefuroxim axetil, Ciprofloxacin, Metronidazole were used in pre-operative and post - operative period in all of the cases.

Regarding sensitivity of the micro-organisms it was observed that, *Escherischia coli* were sensitive to Ciprofloxacin (45.45% cases), Cephradin (54.54% cases), Cotrimoxazole (45.45 % cases), Nitrofurantoin (9.09 % cases), Ceftriaxone (72.72% cases) and Imipenem (100% cases). All the cases of E. *coli* were resistant to flucloxacillin (Table XIV).

Staphylococcus aureus were sensitive to Ciprofloxacin (44.45% cases), Cephradin (44.45% cases), Flucloxacin (55.55% cases), Ceftriaxone (88.90 % cases) and Imipenem (100.00% cases). But, in all the cases *Staph. aureus* were resistant to Cotrimoxazole and Nitrofurantoin.

These findings can be compared with the findings of a national survey in Ireland done in 1993. The overall percentage of S. aureus sensitivity to the tested antibiotics were as follows: Methicillin 85%, penicillin 8%, gentamycin 89%, ciprofloxacin 85%, erythromycin 80%, fusidic acid 96 % and mupirocin 98% (Moorhouse et al. 1996). Here, sensitivity of the organisms to ciprofloxacin is much higher than the present study. Results are inconstent with that of present study; it may be due to limited number of isolates in the present study and variation in the methodology.

Klebsiella pneumoniae were sensitive to Cephradin and Cotrimoxazole in 50 per cent cases each. All of the cases (100.00%) were sensitive to Ceftriaxone

and Imipenem. But, all the cases of *Kl. Pneumoniae* were resistant to Ciprofloxacin, Flucloxacillin and Nitrofurantoin.

Fifty per cent cases of *Pseudomonus aeruginosa* were sensitive to Ciprofloxacin and Nitrofurantoin. All the cases of *P. aeruginosa* (100.00%) sensitive to Ceftriaxone and Imipenem. All of them (100.00%) were resistant to Cephradin, Cotrimoxazole and Flucloxacillin. This findings is comparable with that of Ozumba, Nigeria, who studied antibiotic sensitivity pattern on 229 clinical isolates of *Pseudomonus aeruginosa.* Majority of isolates tested were susceptible to Ceftazidim (88.5%), Colistin (83.75%), Ciprofloxacin (62.1%) and Ofloxacin (62.5%). These were less susceptible to Ceftriaxone (45.1%), Gentamycin (44.1%), Cotrimoxazole (0.7%) and Nitrofurantoin (6.7%) (Ozumba 2003).

All the organisms isolated (100.00%) were sensitive to Imipenem because this is an excellent newer drug with broad spectrum of activity and another fact is that it is not a commonly used drug. so, development of resistance is uncommon. Use of newer drugs should be reserved for specific cases and must not be used empirically or prophylactically.

LIMITATIONS OF THE STUDY

As this study has been carried out over a limited period of time with a limited number of patients and there was lack of financial and infrastructural support, it could not have been large enough to be of reasonable precision. All the facts and figures mentioned here may considerably vary from those of large series covering wide range of time, but still then, as the cases of this study were collected from a tertiary level hospital in our country, this study has some credentials in reflecting the facts regarding factors responsible for surgical site infection following emergency non traumatic abdominal operations.

SUMMARY

Surgical Site Infections (SSIs), previously called post operative wound infections, result from bacterial contamination during or after a surgical procedure. The risk of infection is greater in all categories if surgery is performed as an emergency. Surgical site infection causes considerable morbidity, mortality and high cost to the health care system and is becoming increasingly important in medicolegal aspects.

A few studies were conducted in our country on such an important topic. Further research is necessary to identify the important factors responsible for high infection rate following emergency nontraumatic abdominal operations in our country. In this study it had been tried to find out the common organisms responsible for surgical site infections following emergency nontraumatic abdominal operations. In addition, the sensitivity patterns of the microorganisms were ascertained. Further, factors responsible for infection were determined, that will be helpful to prevent infection in future during the similar types of surgery. General objective of the study was to determine the factors responsible for surgical site infections following emergency nontraumatic abdominal operations, which will be helpful in reducing the rate

of surgical site infection. This descriptive type of study was conducted in Surgery dept, Madras Medical College and Rajiv Gandhi Government General Hospital, chennai, from 1 may, 2014 to 30 september, 2014. However, 140 cases having emergency nontraumatic abdominal operations were selected purposively. Patients with trauma were excluded from the study. Out of 140 abdominal operations 60 (42.86 %) were for acute appendicitis, while 30 (21.43 %) for small intestinal obstruction, 19 (13.57%) for ileal perforation, 15 (10.71 %) for duodenal ulcer perforation, 12 (8.57%) for gangrenous and / ruptured appendix and 2 (1.43%) each for volvulus of sigmoid colon and obstructed inguinal hernia.

It was revealed that, overall surgical site infection rate was 17.14 per cent. It was observed that among the various host factors studied role played by age, sex, and educational status of the patients were not statistically significant, but presence of co-morbidity played a significant role in causing SSI. Among the perioperative / environmental factors category of operations, types of incisions, experience of surgeons and delay to initiate operation did not played significant role, but duration of operation and degree of wound contamination played statistically significant role. In respect of post operative wound discharge and incriminated organisms it was found that most of the SSIs (11,

45.83 %) were due to *Escherischia Coli*, while 9 (37.50 %) infections were due to *Staph. Aureus* and 2 (8.33 %) each were due to *Klebsiella Pneumonae* and *Pseudomonus Aeruginosa.*

It was revealed that multiple host factors (e.g. presence of co-morbidity including jaundice, diabetes, COPD, malnutrition, obesity etc.), environmental factors (e.g. duration of exposure of the wounds to the environment and degree of wound contamination) and various micro-organisms (including *E. Coli, S. Aureus, Klebsiella* and *pseudomonus)* were responsible for surgical site infections. So, to prevent SSI emphasis should be given to all the important factors responsible for infection.

CONCLUSION

This descriptive type of study was conducted in Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, chennai, from 1 may, 2014 to 30 september, 2014. It can be concluded from the findings of the study that micro-organisms that are normal inhabitants of our body are mainly responsible for surgical site infection (SSI). Various host factors like malnutrition, obesity, patients knowledge about hygiene, presence of co-morbidity etc. coupled with environmental factors such as condition of the wounds, delay to initiate operation, duration of operation, prolonged exposure of peritoneal cavity to environment, prophylactic use of antibiotics and factors associated with surgery like type of incision, type of operation and experience of operating surgeon greatly contribute to occurrences of SSI. So, quality of surgical care including immediate assessment of patients, resuscitative measures, adequate preparation of patients and aseptic environment are important for control of SSI. Moreover in absence of highly advanced surgical amenities, preoperative resuscitative units, modern operation theatre facilities and sophisticated sterilization procedure it is necessary to use prophylactic antibiotics to encounter the various types of micro-organisms responsible for surgical site infection, particularly *E. Coli* and *Staph. Aureus.*

RECOMMENDATIONS

On the basis of the findings of the study, the following recommendations can be made:

1. Prompt diagnosis, proper assessment, quick resuscitation and appropriate preoperative preparation are keys to better outcome in emergency operations, but undue delay should be avoided in treating any emergency condition.
2. Duration of operation should be optimum to minimize the level of wound contamination and prevention of SSI.
3. Emergency abdominal conditions should be managed by the experienced surgeons.
4. Proper care of the patients as a whole throughout the peri-operative period is very vital to reduce the rate of surgical site infection.
5. Appropriate antibiotic prophylaxis should be practiced.
6. Further research is necessary in large scale for guidance regarding prevention of surgical site infections in our country.

BIBLIOGRAPHY

1.Alberts, B., Johnson, A., Lewis, J., Raff, M., Roberts, K., and Walters, P., 2002.

2.Molecular Biology of the Cell (4th ed.). New York and London: Garland Science. Available http://www.ncbi.nlm.nih.gov/books/bv.fcgi?call=bv.View..ShowTOC&rid=mboc4.TOC&depth=2. p 207-8

3.Ali, S.L., Khan, A.N.G.A., 1983. Pattern of surgical infection at Chittagong Medical College Hospital. *Journal of BCPS*, 1(1), 17-20**.**

4.Chang H.Y., Sneddon J.B., Alizadeh A.A., Sood R., West R.B., Montgomery K., et al. 2004. Gene Expression Signature of Fibroblast Serum Response Predicts Human Cancer Progression: Similarities between Tumors and Wounds.c *Public Library of Science* 2(2). PMID 14737219. [Accessed January 20, 2008].

5.Cuschieri, S. A., Steele ,R.J.C., Mossa A.R., 2002. Essential Surgical Practice, 4th ed, 338 Euston Road, London NW1 3BH: Arnold, Vol. 2, 1357-8

6.Cruse, P.J.E., Frood, R., 1980. The epidemiology of wound infection . *Surgical clinics of North America*, 60 (1), 27-40.

7.Doharty, G. M., Way L.W., 2006. Current Surgical Diagnosis, 12th ed.

McGraw Hill, USA; p. 106-107.

8.Eber, M.R., Laxminarayan, R., Perencevich, E.N., Malani, A., 2010, Clinical and Economic Outcomes Attributable to Health Care-Associated Sepsis and Pneumonia, *Arch Intern Med*, 170(4), 347-353.

9.Enoch, S., Price, P., 2004. *Cellular, molecular and biochemical differences in the pathophysiology of healing between acute wounds, chronic wounds and wounds in the elderly*. Worldwidewounds.com.

Garg, H.,G., 2000. *Scarless Wound Healing.* New York, Marcel Dekker, Inc. Electronic book.

10.Hernandez, K., Ramos, E., Seas, C., Henostroza, G., Gotuzzo, E., 2005. Incidence of and risk factor for Surgical site infections in a Peruvian Hospital, *Chicago Journals*, 26 (5) 473-477

11.Huda M. N., 2005. Wound infection profile in different non-traumatic emergency abdominal operations. Dissertation (FCPS). Bangladesh College of Physicians and Surgeons. P 60-61.

12.Israelsson, L. A., Jonsson, T., 1997. Overweight and healing of the midline incisions: the importance of suture technique . *Eur J Surg.*, 163 (3), 175-80.

13.Iqbal, M., Patel, K., Ain, Q., Barney, N., Kiani, Q., Rabbani, K., Z.*et al*.2002. Susceptibility Patterns of Escherichia coli: Prevalence of Multidrug – resistant Isolates and Extended Spectrum Beta-Lactumase Phenotype, *JPMA*

52(407), 1- 4

14.Janeway, C.A., Travers, P., Walport, M., Shlomchik, M.J.,2001. *Immunobiology* (5thed.). New York and London: Garland Science. P 106-7. ISBN0-8153-4101-6.Available at:http://www.ncbi.nlm.nih.gov/books/bv.fcgi?call=bv.View..ShowTOC&rid=imm.TOC &depth=10

15.Jawetz E., 1982. Review of Medical Microbiology, 15th edition, USA, Lanze Medical Publication, 29-241

16.Kirk R.M., Ribbans, W.J., 2004. Clinical surgery in general (RCS course manual), 4th ed. Churchill livingstone, London, UK; 206-382

17.Kumar V., Abbas A. K., Fausto N. 2005. Robbins and Cotran Pathologic basis of disease, Seventh edition, 88-116.

18.Levinson W., 2005. Medical Microbiology & Immunology: Examination & Board review, Eighth edition, England, The McGraw-Hill Companies, 131-34

19.Midwood, K.S., Williams, L.V., Schwarzbauer, J.E., 2004. Tissue repair and the dynamics of the extracellular matrix. *The International Journal of Biochemistry & Cell Biology,* 36 (6), 1031–1037.

20.Modi, A. K., [pdf] Aseptic measures in operation theatre [online].

21.Moorhouse, E., Fenelon, L., Hone, R., Smyth E., McGahon, J., Dillon, M., 1996. Staphylococcus aureus sensitivity to various antibiotics—a national

survey in Ireland 1993, *Ir J Med Sci,165(1),40-3*

22.Nguyen, D.T., Orgill D.P., Murphy G.F., 2009. Chapter 4: The Pathophysiologic Basis for Wound Healing and Cutaneous Regeneration. Arnold, New York, 107-8

23.Nichols R.L., 2009. Preventing Surgical Site Infections: A Surgeon's Perspective,*Centre for Disease Control*; Vol. 7, 1-10

24.Ozumba, U. C., 2003. Antibiotic sensitivity of isolates of *pseudomonus aeruginosa* in enugu, Nigeria, *African journal of clinical and experimental microbiology*, 4 (1), 48-49

25.Paul D., 2004. A study of wound infection following emergency GIT surgery and their bacteriological study, Dissertation (FCPS). Bangladesh College of Physician and Surgeons. 125-138

26.Rasul, G., Ashraf, S. A. 1979. The role of routine antibiotic in the prevention of wound infection afrter surgery *, BMRC Bulletin,* 5(2), 71-74 .

27.Renvall S., Niinikoskij, Aho, A.J., 1980. Wound infection in abdominal surgery: a prospective study on 696 operations. *Acta Chir Scand* .146 (1), 25-30.

28.Schnuriger, B., Inaba, K., Eberle, B. M., Wu, T., Talving, P., Bukur, M. *et al.,* 2010. Microbiological profile and antimicrobial susceptibility in surgical site infections following hollow viscus injury. *J Gastrointest Surg.* 14 (8),

1304-10.

30.Stadelmann, W.K., Digenis, A.G., Tobin G.R., 1998. Physiology and healing dynamics of chronic cutaneous wounds. *The American Journal of Surgery,* 176 (2), 26S-38S.

31.Sultan, J., Bilal, H. B., Kiran, H., Bilal, B. B., Yusuf, A., 2007. Emergency Abdominal Surgery; incidence of intra abdominal sepsis and its management, *Professional Med J*, 14(1), 10-16.32.Surgical Site Infection Survillance for general surgery, 2009. *Wexford General Hospital*

Surgical Site Infection (SSI) data report, [online], 8

33.Williams, N.S., Bulstrode, C.J.K., O'conel, P.R., 2008. *Bailey and Love's Short Practice of Surgery*, 25 th ed. 338 Euston Road, London NW1 3BH: Hodder Arnold, 32-48.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax: 044 25363970

CERTIFICATE OF APPROVAL

To

Dr.B.PADAM KUMAR,
Post Graduate,
Institute of General Surgery,
Madras Medical College,
Chennai – 600 003.

Dear Dr. B.PADAM KUMAR,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Study on surgical site infections in emergency non traumatic abdominal operations"** No.500614.

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C.Rajendran, M.D,	-- Chairperson
2.	Prof. Kalaiselvi, M.D, Vice Principal, MMC, Ch-3	-- Member Secretary
3.	Prof. Nandhini, M.D, Inst. of Pharmacology, MMC, Ch-3	-- Member
4.	Prof.G.Muralidharan, M.S, Prof & HOD General Surgery, MMC, Ch-3	-- Member
5.	Prof.V.Padmavathi, M.D, I/c. Director of Pathology, MMC, Ch-3	-- Member
6.	Thiru. S. Govindasamy, BA., BL	-- Lawyer
7.	Tmt.Arnold Saulina, MA MSW	-- Social Scientist
8.	Thiru.S.Ramesh Kumar, Administrative Officer, MMC, Ch-3.	-- Lay Person

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

Sd/Chairman & Other Members

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

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Appendix-II

DATA COLLECTION SHEET

Title*: "A study on pattern of Surgical Site Infections following emergency non-traumatic abdominal surgery."*

Case ID. No. **Date:**

PATIENT'S INFORMATION

1. Name:
2. Age:..... years..... months.
3. Sex : Male/ Female
4. Address :……
5. Education: 0-5 years/ 5-10years/ SSC / HSC / Graduate / Post graduate

HOSPITAL INFORMATION

1. Hospital reg. no……. Ward:…….Bed:…….SU-1/2/3.
2. Date of admission :...../…./10 Date of operation : …../...../10 Date of discharge:…../……./10
3. No. of days in hospital after operation: 1 / 2 / 3 / 4 / 5 / 6 / 7 / 8 / 9 / 10 / 11 / 12 / 13 /14 / 15 /…..

PRE-OPERATIVE FINDINGS

1. (a) Height : ……cm (b) Weight: ……Kg (c) BMI: ……
2. Nutritional status : Morbid Obese / severe obese / obese / over weight / normal / below average
3. Diabetic status : Diabetic / non-diabetic, if diabetic – controlled /uncontrolled
4. Other concurrent disease : Present/absent, If present, COPD/IHD/jaundice/….
5. Drugs : Steroids/cytotoxic/immunosuppressive/ others
6. Septic focus : Present/absent, if present- local/ distant
7. Starting symptom. :Time…….am/pm Date……./……../2010.
8. Use of Pre-operative antibiotics:
   Preparation
   Dose- 250mg/500mg/1gm/2gm
   Route- iv/ im /oral

Pre-operative diagnosis……

INVESTIGATIONS

Blood: Hb …….% ,**TC-** …… /cu mm of blood, **DC: P…..% L….. % M….. % E….. % B …… %**

ESR-.....mm in 1st hour **Blood sugar……** m. mol/l

Plain X-ray abdomen……

X-ray chest (P/A view) ……..

USG of whole abdomen……

PER-OPERATIVE FACTORS

Date of operation:…./…./2010 Time of starting: …….am/pm

Gap of time between start of symptom and start of operation (in hours) :< 6 /<12 /<24 /< 48/ <72/>72

Duration of operation (in hours): 0-1 / 1-2 / > 2.

Name of incision- Upper midline / Lower midline / Rt.paramedian / Grid iron / Rutherford Morison /Lanz / Inguinal /……..

Findings-

3)

33)

Name of operation- Appendicectomy/ Appendicectomy & peritoneal toileting/ Repair of perforation & thorough peritoneal toileting /Division of bands & adhesions/ Resection & anastomosis/Herniotomy & herniorrhaphy/ ……

Sutures used: Inner layers- Vicryl / Catgut / Dexon /Prolene / Nylon / Silk /others ……

Skin- Vicryl / Catgut / Dexon / Prolene / Nylon / Silk /others……

Per- operative antibiotic used: Preparation……Dose- 250mg/500mg/1gm/2gm

Route- iv/ im /oral

Surgeon: Professor /Assist prof./ R.S/Registrar /Assistant registrar/IMO/HMO/Intern.

Postoperative findings:

Fever- Day of onset- 1/ 2 / 3 / 4 / 5 / 6/ 7 /…POD Range of temp: From…….to..…. ° F

Chest pain – yes/no, if yes starting from 1/ 2/3/4/5/6/7/….POD

Cough- yes/no, if yes starting from 1/2/3/4/5/6/7/….POD

Pain in/ around wound- yes/no, if yes starting from 1/2/3/4/5/6/7/….POD

Tenderness in/ around the wound -yes/no, if yes starting from 1/2/3/4/5/6/7/….POD

Character of Discharge/pus from the wound- Serosanguinous / Thick creamy pus / Thin odorless pus /Bluish green pus / Yellow fishy odoured pus /……..

Findings of Swab for CS- Growth of organism- yes/no; if yes, name of organism-……..

Sensitive to……..

Resistant to…… ….……..

Surgical site infection -Yes/no, if yes type of SSI- Stitch abscess/Superficial layer infection /Deep layer infection /deep space infection/organ infection.

Post-operative antibiotic

Preparation-…… Dose- 250mg/500mg/1gm/2gm. Route- iv/ im /oral

Remarks-……

Appendix – III

Statistical formula

A. Sample size:

To determine the sample size, this formula was used; $n = \frac{z^2 pq}{d^2}$

Where,

n = the desired sample size,

z = the standard normal deviate, usually set at 1.96 at 5% level,

which corresponds to 95% confidence level,

p = proportion of population, q

= 1- p

d = the degree of accuracy level considered as 5.0 %,

which assumes 0.05

If population size, N < 10,000 than the required sample size is very much smaller which was calculated by the following formula –

$$n_f = \frac{n}{1 + \frac{n}{N}}$$

Where,

n_f = the desired sample size, when population size, N < 10,000

n = the desired sample size, when population size, N > 10,000 N

= the roughly estimated population size.

B. Arrithmatic mean, $X = \frac{\sum fx}{N}$ (for grouped data)

C. Standard deviation , $SD = \sqrt{\overline{\sum (X- X)^2}}$

('O' indicates observed value and 'E' indicates expected value)

D. $$Z = \frac{P_1 - P_2}{\sqrt{\left[\frac{P_1 Q_1}{N_1} + \frac{P_2 Q_2}{N_2}\right]}}$$

P_1 indicates proportion in first group

P_2 indicates proportion in second group

$Q_1 = 100 - P_1$

$Q_2 = 100 - P_2$

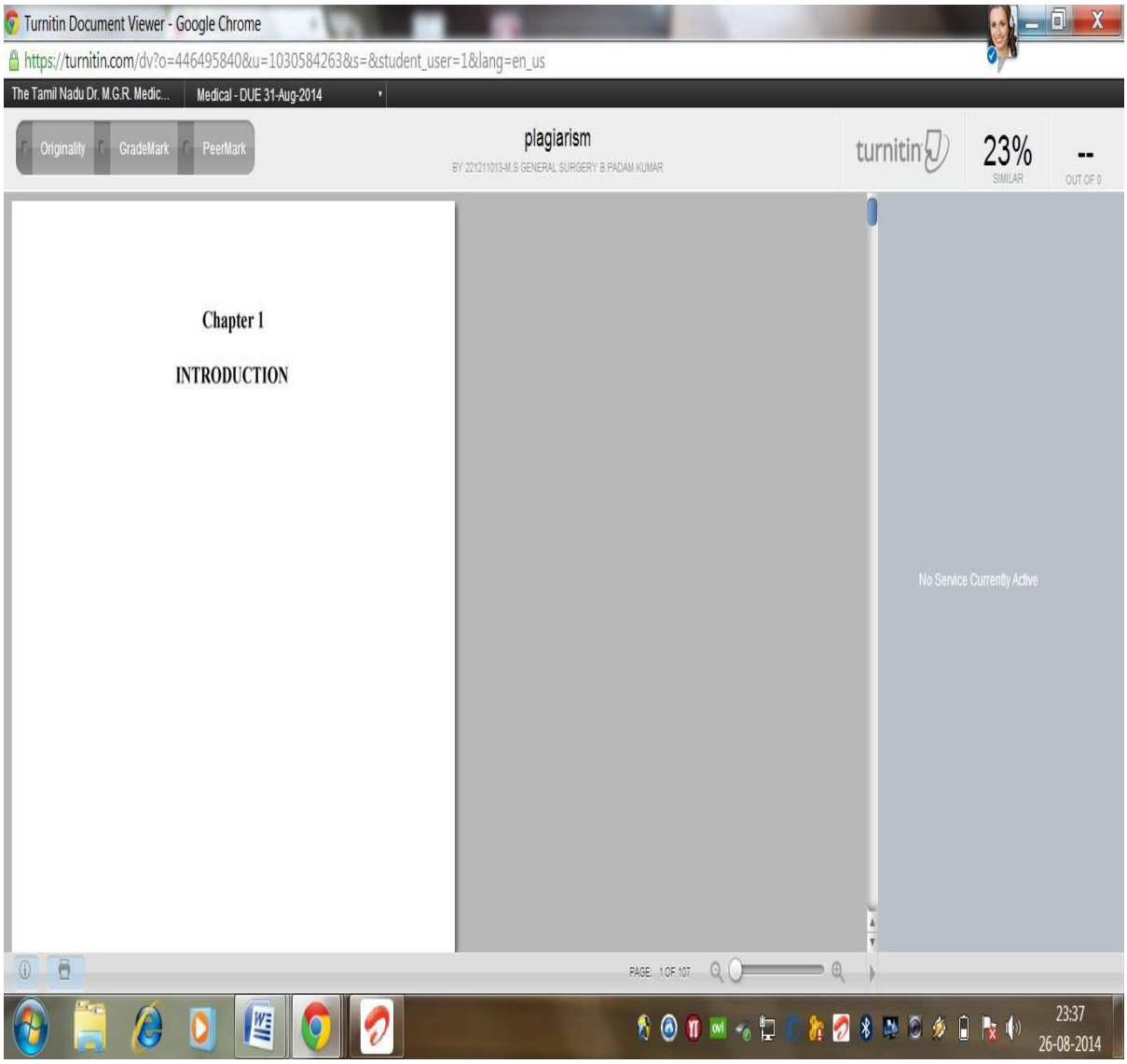
N_1 indicates sample size of first group

N_2 indicates sample size of second group.

E. $$SD = \sqrt{\frac{\sum (X - \bar{X})}{(N-1)}}$$

Here, $\bar{X}$ indicates mean value
X indicates individual value
N indicates sample

Appendix-IV: Plagiarism



S.NO	NAME	AGE	SEX	EDUCATION	SURGERY	INCISION	TIME DELAY(hrs)	DURATION	WOUND	CO MORBID	DISCHARGE	ORGANISM
1	Sudhakar	30	M	Graduate	hernia	Inguinal	10	1 . 10	C			
2	Vignesh	16	M	Primary	Appendix	rutherford	30	45	CC			
3	Premavathy	51	F	Illiterate	Ileal perf	midmidline	76	1 . 15	D	Mal	Blue green	pseudomonas
4	Gokul	16	M	SSLC	Appendix	rutherford	30	45	CC			
5	Prakash	21	M	Graduate	Appendix	grid iron	28	45	CC			
6	Amudha	37	F	Secondary	Appendix	rutherford	60	45	CC			
7	Lakshmi	66	M	Illiterate	hartmann's	ext lower midline	76	2 . 15	Cont	DM	Yellow fishy odour	klebsiella pneumonia
8	Munusamy	39	M	Primary	Du perf	upper midline	4	55	D			
9	Palani	38	M	Illiterate	ileostomy	midmidline	60	55	D			
10	Rajesh	18	M	HSC	Appendix	grid iron	30	45	CC			
11	Sujatha	36	M	Secondary	Ileal perf	midmidline	10	1 . 15	D	Mal		
12	Krithika	25	F	Graduate	Appendix	rutherford	28	45	CC			
13	Daniel	32	M	Secondary	Appendix	grid iron	30	45	CC			
14	Ranjith	17	M	Primary	Appendix	grid iron	60	45	CC			
15	Kalaiselvi	65	F	SSLC	Appendix	grid iron	26	45	CC	DM		
16	Inbaraj	34	M	HSC	Du perf	upper midline	5	55	D			
17	Selvi	39	F	Illiterate	Adhesiolysis	upper midline	34	55	C			
18	Yuvaraj	19	M	Graduate	Appendix	grid iron	30	45	CC			
19	Mumtaz	39	F	SSLC	ileostomy	midmidline	66	55	D	Mal		
20	Ashok	16	M	HSC	Appendix	rutherford	25	45	CC			
21	Rajeshwari	25	F	Primary	Appendix	Lanz	30	45	CC			
22	Loganathan	68	M	Illiterate	Adhesiolysis	ext upper	30	55	C	COPD		
23	Savithri	46	F	SSLC	Adhesiolysis	ext upper	70	2 . 15	C			
24	Afsal	18	M	HSC	Appendix	grid iron	30	45	CC			
25	Siva	17	M	SSLC	Append/lavage	lower RPM	26	55	C			

26	Raja	52	M	Illiterate	Ileal perf	midmidline	74	1 . 30	D	DM	Blue green	pseudomonas
27	Niranjan	38	M	Graduate	Du perf	upper midline	5	55	D			
28	karthik	44	F	Illiterate	Du perf	ext upper	60	1 . 30	D	Mal	creamy	Staph aureus
29	kaniappan	36	M	SSLC	Appendix	grid iron	30	45	CC			
30	Saranya	16	F	HSC	Append/lavage	lower RPM	10	1 . 15	D			
31	Palanisamy	49	M	Illiterate	Du perf	upper midline	60	2 . 15	D	Mal	yellow fishy odour	klebsiella pneumonia
32	Aarmugam	69	M	SSLC	Ileal perf	midmidline	5	55	D	DM		
33	Jyothi	18	F	Primary	Appendix	Lanz	11	45	CC			
34	Selvi	39	F	HSC	Adhesiolysis	ext upper	60	55	C	obesity		
35	Ragunath	44	M	Illiterate	ileostomy	ext upper	60	1 . 30	D	COPD	creamy	Staph aureus
36	Monisha	16	F	SSLC	Appendix	grid iron	18	40	CC			
37	Prabhakar	32	M	HSC	Append/lavage	lower RPM	60	55	D			
38	Monisha	18	F	Secondary	Appendix	grid iron	12	1 . 15	CC	Mal	muddy dourlesss	E.coli
39	Pandian	48	M	Primary	R & A	midmidline	60	2 . 15	Cont		creamy	Staph aureus
40	Mahalakshmi	28	F	Graduate	Appendix	Lanz	30	40	CC			
41	Bhaskar	44	M	SSLC	Du perf	upper midline	4	1 . 15	D	Mal		
42	Lakshmi	26	F	Secondary	Append/lavage	lower RPM	20	2 . 15	D	Mal	muddy dourlesss	E.coli
43	Singaravelan	61	M	Secondary	Ileal perf	midmidline	11	1 . 45	D	COPD		
44	Nayana	26	F	Graduate	Appendix	grid iron	60	45	CC			
45	Logesh	19	M	SSLC	Appendix	rutherford	13	45	CC			
46	Annapurni	29	F	Illiterate	Append/lavage	lower RPM	30	55	D	Mal		
47	Anandan	18	M	SSLC	Appendix	grid iron	15	40	CC			
48	mary	48	F	Primary	Adhesiolysis	ext upper	36	1 . 15	Cont	obesity	creamy	Staph aureus
49	Vasugi	28	F	HSC	Adhesiolysis	ext upper	66	55	C			
50	Sinnarasu	46	M	Graduate	ileostomy	midmidline	36	1 . 15	D	COPD	creamy	Staph aureus

51	Satish	18	M	SSLC	Appendix	Lanz	30	40	CC			
52	Sundari	36	F	SSLC	Append/lavage	lower RPM	11	1 . 30	D			
53	Kayalvizhi	24	F	HSC	Appendix	grid iron	30	40	CC			
54	Munikrishnan	66	M	Illiterate	Du perf	upper midline	4	55	D			
55	Chandrasekar	48	M	HSC	ileal perf	midmidline	36	1 . 15	D	Mal	creamy	Staph aureus
56	Madurai	39	F	SSLC	Adhesiolysis	ext upper	68	1 . 45	C			
57	Rajendran	39	M	Secondary	Append/lavage	lower RPM	20	1 . 45	D		creamy	Staph aureus
58	Sharmila shri	18	F	SSLC	Appendix	grid iron	30	40	CC			
59	Govindasamy	46	M	Primary	Append/lavage	lower RPM	30	55	D			
60	Sathya	38	F	Illiterate	Ileal perf	midmidline	10	55	D	Mal		
61	Mukesh	18	M	SSLC	Appendix	grid iron	18	45	CC			
62	Amsvalli	32	F	Illiterate	Appendix	rutherford	5	40	CC			
63	Ramadass	47	M	SSLC	Du perf	ext upper	10	55	D	Mal		
64	Tamilarasi	28	F	Graduate	Appendix	rutherford	15	45	CC			
65	Sengalvarayan	49	M	Illiterate	sigmoid volvulus	lower midline	60	1 . 45	C			
66	Sasikala	32	F	SSLC	Adhesiolysis	ext upper	30	55	C			
67	Dayanidhi	17	M	Secondary	Appendix	grid iron	14	45	CC			
68	Muniammal	55	F	Illiterate	Append/lavage	lower RPM	11	55	D			
69	Siva	31	M	Graduate	Append/lavage	lower RPM	36	2 . 15	D	jaundice	muddy dourlesss	e.coli
70	Rajagopal	48	M	Secondary	ileostomy	midmidline	60	1 . 20	D	Mal	creamy	Staph aureus
71	Vaishnavi	16	F	SSLC	Appendix	grid iron	11	40	CC			
72	Satish	19	M	Primary	ileostomy	midmidline	66	55	D	DM		
73	Vishal	26	M	Secondary	Append/lavage	lower RPM	20	1 . 20	D	Mal	muddy dourlesss	E.coli
74	Senthil	31	M	Graduate	ileostomy	midmidline	36	1 . 45	D	Mal	muddy dourlesss	E.coli
75	Chelllappan	48	M	Illiterate	Append/lavage	lower RPM	4	55	D	Mal		
76	Eswarammal	36	F	Secondary	Adhesiolysis	ext upper	60	1 . 45	C			

77	Bhavani	32	F	Secondary	Appendix	grid iron	10	40	CC			
78	Rajappan	45	M	HSC	R & A	ext upper	78	1 . 45	Cont			
79	Kadhar basha	55	M	Secondary	hernia	Inguinal	14	55	C			
80	Devika	32	F	Secondary	Appendix	grid iron	60	40	CC			
81	Shanmugam	44	M	Secondary	Du perf	upper midline	4	55	D	DM		
82	Hemalatha	35	F	Graduate	Appendix	grid iron	17	45	CC			
83	Priya	19	F	HSC	Appendix	rutherford	13	45	CC			
84	Arivazhagan	52	M	Graduate	ileostomy	midmidline	66	55	D	jaundice		
85	Bommi	49	F	Secondary	Du perf	upper midline	10	55	D	Mal		
86	Vinod	18	M	Secondary	Appendix	grid iron	13	45	CC			
87	Maheshwari	56	F	SSLC	R & A	ext upper	30	1 . 45	Cont			
88	Somasekar	44	M	Secondary	R & A	ext upper	76	1 . 45	Cont			
89	Rajan	32	M	SSLC	Du perf	ext upper	36	1 . 45	D	Mal	muddy dourlesss	E.coli
90	Sadayappan	46	M	Secondary	R & A	ext upper	68	2 . 15	Cont	COPD		
91	Vanitha	22	F	Secondary	Appendix	grid iron	14	45	CC			
92	Rajammal	38	F	SSLC	Adhesiolysis	upper midline	36	2 . 15	C		creamy	Staph aureus
93	Sheela	13	F	Primary	Appendix	rutherford	11	40	CC			
94	Sundari	17	M	HSC	Appendix	grid iron	13	45	CC			
95	Selvadurai	43	M	Graduate	Du perf	upper midline	60	55	D	Mal		
96	Mythili	19	F	Graduate	Appendix	Lanz	15	35 m	CC			
97	Mannan	56	M	Illiterate	Ileal perf	midmidline	11	1 . 15	D			
98	Radhika	29	F	Graduate	Adhesiolysis	ext upper	70	1 . 45	C			
99	Santosh	28	M	Secondary	R & A	ext upper	78	1 . 45	Cont	Mal		
100	Vigneshwaran	19	M	SSLC	Appendix	rutherford	4	1 .	CC		muddy dourlesss	E.coli
101	Ranjitham	49	F	Graduate	R & A	ext upper	60	1 . 45	Cont			
102	Murugan	27	M	SSLC	Appendix	grid iron	16	45	CC			

103	Banu	44	F	Illiterate	R & A	ext upper	30	1 . 45	contaetd			
104	Sasikumar	24	M	HSC	Appendix	grid iron	11	45	CC			
105	Chinnadurai	51	M	Secondary	Appendix	rutherford	13	45	CC			
106	Venda	46	F	Secondary	Adhesiolysis	ext upper	66	55	C	obesity		
107	Sukumar	29	M	Graduate	Appendix	grid iron	13	40	CC			
108	Rajan	21	M	HSC	Appendix	grid iron	16	50	CC			
109	Muneeswaran	44	M	Illiterate	Ileal perf	midmidline	4	1 . 45	D			
110	Sangeetha	18	F	Secondary	Appendix	grid iron	14	45	CC			
111	Subramani	45	M	Illiterate	R & A	ext upper	60	2 . 15	Cont	COPD		
112	Suresh	26	M	Graduate	Appendix	rutherford	15	45	CC	Mal		
113	Nagaraj	17	M	Secondary	Appendix	rutherford	20	1 . 15	CC		muddy dourlesss	E.coli
114	yukesh	26	M	Graduate	Appendix	grid iron	15	40	CC			
115	Devika	29	F	SSLC	R & A	ext upper	78	1 . 45	Cont			
116	Sampath	44	M	HSC	Du perf	upper midline	4	1 . 15	D			
117	Annamalai	58	M	Illiterate	R & A	ext upper	60	1 . 30	Cont			
118	Praveen	17	M	Secondary	Appendix	grid iron	10	45	CC			
119	Munusami	32	F	SSLC	ileal perf	midmidline	60	1 . 45	D	Mal	muddy dourlesss	E.coli
120	Bhasker	26	M	Secondary	Appendix	grid iron	13	50	CC			
121	Syed Hussain	29	M	Graduate	R & A	upper midline	66	1 . 30	Cont			
122	Shankar	43	M	Secondary	R & A	ext upper	30	1 . 45	Cont			
123	Anandhan	24	M	Graduate	Appendix	grid iron	13	45	CC			
124	Ibrahim	44	M	Illiterate	R & A	ext upper	60	1 . 30	C	COPD		
125	Yogesh	19	M	SSLC	Appendix	rutherford	10	45	CC		muddy dourlesss	E.coli
126	Sanjay	24	M	Secondary	Appendix	grid iron	16	40	CC			
127	Mohan	29	M	Secondary	Appendix	grid iron	30	45	CC			
128	Jerlin shiny	25	F	Graduate	Appendix	grid iron	15	45	CC	Mal		

129	Lalitha	25	F	Secondary	Appendix	grid iron	14	40	CC			
130	Ramesh	23	M	Secondary	Du perf	upper midline	11	55	D			
131	Perumayee	46	F	Illiterate	R & A	ext upper	76	2 . 15	CC			
132	Muthusamy	26	M	Graduate	Appendix	grid iron	14	30	CC			
133	Narasimman	45	M	Secondary	R & A	ext upper	30	1 . 45	C			
134	Manimegalai	47	M	Illiterate	ileostomy	midmidline	60	1 . 30	D			
135	Ajay	17	F	Secondary	Appendix	grid iron	20	45	CC		muddy odourless	e.coli
136	Nataraj	45	M	SSLC	R & A	ext upper	30	1 . 45	Cont			
137	Balaraman	39	M	Secondary	R & A	ext upper	30	1 . 30	Cont			
138	Md. Ghouse	30	M	HSC	Appendix	grid iron	76	45	CC			
139	Inbaraj	41	M	Illiterate	Du perf	upper midline	11	1 . 15	D	Mal		
140	Devadoss	32	M	Graduate	Appendix	grid iron	13	40	CC			

KEY :

C – Clean
CC – Clean Contaminated
Cont – Contaminated
D – Dirty
Mal – Malnoourished
COPD – Chronic Obstructive Pulmonary Disease