THE UNIVERSITY OF ZAMBIA
SCHOOL OF MEDICINE
DEPARTMENT OF SURGERY

A STUDY ON THE COMMON HYDROTHERAPY PRACTICES AND THE PREVALENCE OF BURN WOUND BACTERIAL COLONISATION AT THE UNIVERSITY TEACHING HOSPITAL IN LUSAKA, ZAMBIA.

By

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A dissertation submitted to the **University of Zambia** as partial fulfillment for the award of the Master of Medicine Degree in General Surgery.

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ABSTRACT

BACKGROUND Hydrotherapy plays an important role in the management of surgical patients, and especially so in those that have sustained burns to their skin. Different centers practice hydrotherapy differently. At the University Teaching Hospital in Lusaka, Zambia, patients with burns in surgical wards use a common bathtub for cleaning their wounds. This breaches patient isolation and increases risk of cross-infection. Audit records from the department of surgery show that burn wound colonization and infection is an important source of morbidity and mortality. However, there is no evidence yet that the hydrotherapy as practiced at our institution does lead to cross infection among patients with burns.

OBJECTIVE The general objective was to determine if the hydrotherapy practice plays a role in cross-infection. We also wanted to characterize these organisms being spread by cross-infection.

METHODS This was a prospective analytical study. Patients meeting the admission criteria were recruited. Swabs from the burn wounds were collected on admission (day 0), day 4 and day 7. Weekly swabs of the bathtub were also collected, after the tub had been cleaned and declared ready for the next patient. Weekly water samples were also collected. Selected results, for *Staphylococcus aureus* and *Klebsiella pneumoniae,* were subjected to further analysis and PCR. Results were analyzed using statistics software, SPSS version 23.

RESULTS In this study, there were 96 participants of which 51 (53.1%) were males and 45 (46.9%) were females. Age distribution ranged from 5months old to 91 years old. The modal age range was 1 to 2 years old. The modal burn percentage was 6% to 10%, followed by 11 to 15%. Hot water was the cause of burns in 65.6%. *Staphylococcus aureus* and *Klebsiella pneumoniae* were the commonest organisms isolated. These came from wounds that looked clinically clean. Others were enteric organisms. In terms of readily available antibiotics, there was more sensitivity to Amikacin and Chloramphenicol than Ciprofloxacin (our commonly used antibiotic). The bathtub also had *Staphylococcus aureus* and *Klebsiella pneumoniae,* besides enteric organisms. Sixty five point four percent (65.4%) of the Klebsiella were ESBL

producers. The tub had samples that were both ESBL producers as well as widely resistant Klebsiella by other means. Of the ESBLs, 29.4% had the SHV gene, 23.5% had the TEM gene and 47.1% had both SHV and TEM. There was no CTX gene identified. MRSA accounted for 30.6% of all the Staphylococcus in this study. The PVL gene was detected in 11.8%, SPA gene in 35.3%, while 5.9% of the Staphylococcus had both PVL and SPA genes. No growth was obtained from the water samples. Seventy-two point nine percent (72.9%) of the patients were discharged, 19.8% died, while 7.3% left against medical advice.

CONCLUSION Hydrotherapy as currently practiced at the University Teaching Hospital does contribute significantly to cross-infection among burns patients. The organisms transmitted are widely resistant to available antibiotics and this is posing a serious threat to treatment of infections.

DEDICATION

To my lovely wife, Thokozile, for her constant love and support. To my loving parents, mum and dad, for teaching and encouraging me to always do my best. To all my siblings, for always believing in me. I thank you all. And finally, to all the burns patients in our hospitals – it is for the improvement of your care with our limited resources that this research was conducted.

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ABBREVIATIONS

- BLA - Beta-Lactamase
- CTX-M - Cefotaximase
- EMRSA - Epidemic Methicillin-Resistant *Staphylococcus aureus*
- ESBL - Extended Spectrum Beta Lactamase
- LAMA - Left Against Medical Advice
- MRSA - Methicillin-Resistant *Staphylococcus aureus*
- *P. aeruginosa* - *Pseudomonas aeruginosa*
- PCR - Polymerase Chain Reaction
- PVL - Panton-Valentine-Leukocidin
- *S. aureus* - *Staphylococcus aureus*
- SHV - Sulfhydryl Variable
- SPA - *Staphylococcus aureus* Protein A
- SPSS - Statistical Package for the Social Sciences
- TBSA - Total Body Surface Area
- TEM - Temoneira
- UNZABREC - University of Zambia Biomedical Research Ethics Committee
- US - United States
- UTH - University Teaching Hospital
- UTI - Urinary Tract Infection

CHAPTER ONE

1.1 INTRODUCTION

Formerly called hydropathy, hydrotherapy is a part of medicine, in particular of occupational therapy and physiotherapy, that involves the use of water for pain relief and treatment.[2] This treatment utilizes the physical properties of water, especially temperature and pressure. Water is used as a transport to transmit heat or cold to the body which effectively stimulates or reduces blood circulation whilst treating the symptoms of diseases. The water can be used as water jets, underwater massage and/or mineral water.[2]

Historically, various forms of hydrotherapy have been recorded in ancient Egyptian, Persian, Greek and Roman civilisations.[2] Egyptian royalty bathed with essential oils and flowers.[2] Romans had communal public baths for their citizen.[2] Hippocrates prescribed bathing in spring water for sickness.[2] Two English works on the medical uses of water were published in the 18th century that inaugurated the new fashion for hydrotherapy. One was by Sir John Floyer, a physician who, struck by the remedial use of certain springs by the neighbouring peasantry, investigated the history of cold bathing and published a book on the subject in 1702. It was translated into German and became a basis for the book "On the healing virtues of cold water, inwardly and outwardly applied, as proved by experience", by Dr. Hahn published in 1738. The second work was by Dr. James Curne's publication in 1797 which placed the subject on scientific basis.[2]

In today's practice of medicine, hydrotherapy plays an important role in the management of surgical patients. This is very true concerning patients who have sustained burns to their skin. Hydrotherapy in burns typically involves the washing of patients in a tank, shower or agitating bath; however the techniques have evolved over the centuries.[4] Hydrotherapy is said to promote healing by softening and removing the dead tissue and enabling new healthy tissues to form.[3] In addition, hydrotherapy reduces bacterial load, cleans the surface of the wound and removes debris and prevents loss of fluid through the skin thus preventing dehydration.[3,4] Hydrotherapy provides a moist environment for wound healing. It also removes pus and helps

minimize scar tissue formation.[3] A study by Langschmidt et al [8] demonstrated that 96% of respondents in the UK routinely use hydrotherapy in burns, compared to 83.1% from Canadian and American burn centers. In this same study, hydrotherapy is said to assist in the gradual debridement of the burn wound until a healthy bed of granulation tissue is evident, at which point skin grafting can be performed.

Studies have also reported negative outcomes with the use of hydrotherapy, thus raising concerns about hospital acquired cross-infection.[4,8] At the University Teaching Hospital in Lusaka, Zambia, burns patients use the same bathing tub for cleaning their wounds. This breaches patient isolation and increases risk of cross-infection. On the ward, use of wet soaks with normal saline is a common practice. It has been observed that many burns patients develop infection during their hospital stay leading to increased morbidity and mortality.

Audit records for three surgical units in the department of surgery at the UTH show that burn wound colonisation and infection is an important source of morbidity and mortality in burns patients (Appendix E). The audit reports for patients who suffered sepsis but did not end up as mortalities and those who left against medical advice (LAMA) are not highlighted. The audit reports do not have culture results- this is because in most cases, culture results were only ready after the patient had already died. Of particular note is the prevalence of *enterococci* species of bacteria- this may have been acquired through contaminated hydrotherapy equipment (see Appendix F). The microbiology data is arranged per ward so that those wards with burns patients can be easily identified.

Thus despite the several advantages that hydrotherapy in burns has, there is need to continuously work on the negative aspects to minimise morbidity and mortality. This study proposed to undertake a research at the UTH that would look at the possible role that contaminated hydrotherapy equipment at this institution has in spreading infection between burns patients. Also, the study determined the prevalence of culture positive swabs in burns patients following the use of hydrotherapy as practiced at the UTH. This is because despite departmental audit records showing high prevalence of infection, there was no scientific evidence yet that our practice of hydrotherapy

contributed greatly to the spread of this infection. So far it had only been presumptuous, based on studies from other burn centers.

1.2 LITERATURE REVIEW

There is a vast amount of literature that talks about wound colonisation and infections in burns. According to literature on burns, the experience accumulated over the past three decades is that early interventional treatment of burn patients does dramatically change the cause of death.[1] It is now estimated that about 75% of the mortality following burns is related to infections, rather than burn shock and hypovolaemia.[9] This is very significant and efforts are required to find the best preventive measures.

Cross infection is considered to be of particular concern in burn units. Several studies have attributed outbreaks in burn centers to contaminated hydrotherapy equipment.[5,6,14] Another study cited tap water as a significant route of transmission in hospital.[8] It suggested that infections and colonisation could be significantly reduced by placement of filters onto the water taps. Although studies have concluded that precise route by which patients become colonised remains unclear, modern molecular biology technologies have identified the role of contaminated hydrotherapy equipment in strain transmission.[8] Similar results were obtained in a study by Reuter et al. that 36% to 42% of healthcare associated cases of *Pseudomonas aeruginosa* were due to contaminated water from the tap.[7]

Karim and Tredget outlined important patient characteristics that influence morbidity and mortality from burn wound infection and sepsis. They identified large wounds of greater than 30% total body surface area (TBSA) as being a significant risk.[14] In Africa, a comparable study is the Ethiopian study that found TBSA of greater than or equal to 15% as a risk for bacteraemia.[20] The apparent difference in the TBSA percentage between the two studies could be attributed to several factors including differences in availability of resources and differences in infection prevalences. Other

factors listed that influence morbidity and mortality are significant amounts of full-thickness burns and prolonged open wounds or delayed initial burn wound care.[14] A local study by Maimbo et al concluded that early-delayed split skin grafting (within 15 days post burn injury) showed statistically significant reduced length of stay and reduced occurrence of infection as opposed to late or non-grafting.[30] Factors that have favourably impacted the incidence of burn wound infection include early wound closure, topical and prophylactic antimicrobial therapy and advances in infection control measures in modern burn units.[14]

Organisms that infect patients could be endogenous, that is from patient's own normal flora, or exogenous, from the environment or health care personnel.[14] In burns patients, organisms associated with infection include gram positive bacteria, gram negative bacteria and yeast or fungal organisms whose distribution in an individual is said to change overtime.[14] In a typical burn wound, colonisation is initially with gram positive organisms.[14] Within the first week, these are rapidly replaced by antibiotic-susceptible gram negative organisms.[14] If wound closure is delayed and the patient becomes colonised and requires treatment with broad-spectrum antibiotics, these organisms may be replaced by yeast, fungi and antibiotic-resistant bacteria.[14] Gram negative organisms have long been known to cause serious infection in burn patients and have been associated with a 50% increase in predicted mortality for patients with bacteraemia compared with those without bacteremia.[10,14]

Several transmission modes have been given for the infectious organisms, such as contact, droplet and airborne. In burn patients, the primary mode is direct or indirect contact, either by the hands of the personnel caring for the patient or from contact with inappropriately decontaminated equipment. Burn patients are unique in their susceptibility to colonisation from the environment and in their propensity to disperse organisms into the surrounding environment. In general, the larger the burn injury, the greater the volume of organisms dispersed from the patient into the environment. In almost all cases the colonised patient is thought to be a major reservoir for the epidemic strain.[11]

Other important sources include contaminated hydrotherapy equipment, common treatment rooms/areas, and contaminated equipment such as mattresses which seem to

pose unique risks for cross-contamination in the burn care environment. In a survey of directors of burn centers in the US, *P. aeruginosa* was identified as the commonest pathogen nosocomially acquired with hydrotherapy, followed by MRSA.[8] The aquatic environment is difficult to decontaminate because of continuous re-inoculation of the organisms from the patients wound flora and because of the organisms' ability to form a protective glycocalyx in water pipes, drains and other areas, making them resistant to the actions of disinfectants. *Pseudomonas aeruginosa* has been shown to have a propensity for water systems and documented persistent colonisation of hydrotherapy equipment.[5] *Pseudomonas* has evolved to thrive in aquatic environments using its polar pili that allow it to strongly adhere to surfaces and its protective mucopolyssacharide coat that limits the penetration of antimicrobial agents. It can further undergo chromosomal rearrangements resulting in the development of multi-resistant strains. Sharing hydrotherapy equipment among patients breaches patient isolation.[8] Thus adequate decontamination of this equipment (e.g. tubs, tanks, stretchers, showers, straps, etc) is difficult to achieve between patients using this equipment on a daily basis and monitoring techniques are often insufficient to provide timely detection of contamination. In addition, the patient's own flora may be spread through the water and by caregivers to colonise other sites on the patient that are at increased risk of infection. For example, organisms from the wound may migrate to a central venous catheter site or bowel flora may be transferred to the burn wound.[14]

Another principal transmission mode in burn units are by the hands of the personnel and contact with inadequately decontaminated equipment or surfaces.[14] The two areas most likely to become contaminated when caring for the burn patient are the hands and gowns of the personnel, because the surfaces (i.e. beds, side rails, tables, equipment) are often heavily contaminated with organisms from the patient.[12,14] Likewise, all equipment used on the patient (i.e. blood pressure cuffs, thermometers, wheel chairs etc) is also heavily contaminated and the same may be transmitted to other patients if strict barriers are not maintained, and appropriate decontamination performed.[14] A single cause is uncommon in a burn unit outbreak; in almost all instances, multiple factors contribute to occurrences and perpetuation of infecting organisms.[14] To counter transmission by caregivers, some burn centers require that caregivers wear a prescribed dress code.[25]

Specific sites of infection that are particularly important for burn patients include bloodstream infection, pneumonia, burn wound infection and urinary tract infection (UTI).[13] Fever, a highly specific indicator of infection for many populations, often does not correlate well with the presence of infection in burn patients, because of core temperature increases and an increase in heat production , associated with the onset of a hypermetabolic response.[13] As a result, fever alone, in the absence of other signs and symptoms, is not indicative of infection. Furthermore, gauging burn wound sepsis by clinical signs and symptoms is difficult and diagnosis is best made by careful serial evaluations of the wound. Patients with extensive burn wounds generally manifest physiologic changes associated with hypermetabolism, including tachycardia, hypothermia or hyperthermia, tachypnoea, ileus, glucose intolerance and mental status changes. Clinical signs suggestive of burn wound infection that need attention in particular include the progression of partial thickness to full thickness injury and change in wound colour, green discolouration of subcutaneous fat, violaceous discolouration and oedema of wound margins; subeschar haemorrhage or rapid eschar separation.

Burn wound flora and antibiotic-susceptibility patterns have been reported to change during the course of a patient's hospitalisation and further transmission can be prevented by early identification of organisms colonising the wound, monitoring the effectiveness of current wound treatment and controlled perioperative or empiric antibiotic therapy and detecting cross-colonisation, quickly when it occurs. Stringent infection control, including patient isolation, is central to decreasing transmission. This is particularly important with *P. aeruginosa* given its propensity for water systems and documented colonisation of hydrotherapy equipment.[5] The other recommendation is the use of non-occlusive povidone iodine dressing changed every 2 to 4 hours in cases where intravenous access is near the burn wound.

Abid Rashid et al investigated an outbreak of epidemic methicillin-resistant *Staphylococcus aureus*-15 (EMRSA-15) in a regional burns unit.[15] The team showed the presence of EMRSA-15 in some of the patients that were on the ward, and colonisation of some of the staff who were providing care to these patients. The EMRSA-15 was only controlled after the ward was closed, refurbished and

decontaminated. The affected staff were also sent on special leave and treated appropriately.

MRSA outbreak in burn units is not an uncommon occurrence.[16,18] Its main reservoir is the colonised patient, and in the case of burns, older patients and those with large burns are at a higher risk of colonisation. The spread in hospitals occur mainly through hands of healthcare workers and medical equipment, such as hydrotherapy showers.[16] With prolonged hospital stay following complications such as septicaemia and pneumonia, cross contamination is a possibility. There is a relative paucity in the medical literature with regards the management of MRSA in burn units. Some authors have raised concerns about the world-wide increase in the incidence of MRSA and its potential to cause corresponding morbidity and mortality in burns[17], whereas others do not seem to be fully convinced that MRSA poses a real threat.[18,19] Consequently, some have advocated that burns patients colonised with MRSA should be managed aggressively whilst others maintain otherwise. Another infection to worry about is the spread of ESBL producing bacteria. Kenneth M. Wemer et al[27] showed that treatment with fluoroquinolones increases the risk of isolating ESBL producing Klebsiella in hospitalized patients. At the University Teaching Hospital, Ciprofloxacin (a fluoroquinolone) is the most commonly used drug in burns patients.

So far from the literature, it can be seen that infection, following wound colonisation, plays a critical role in causing morbidity and mortality in burns patients. An important route by which this infection is transmitted is by contaminated hydrotherapy equipment.[4,6,7,14] Prevention in this area would greatly improve the care of these patients and reduce morbidity and mortality.

1.3 STATEMENT OF THE PROBLEM

Burn injuries are a common clinical presentation at the UTH in Lusaka, especially in children under five (5) years of age. Following admission to the burns units, most patients develop bacterial wound colonisation followed by infection/sepsis within four (4) days of admission hence becoming a great source of morbidity and mortality.

Once sepsis sets in, the results are usually fatal and this has been a cause of the high mortality among burns patients. There is a lack of local evidence to show that hydrotherapy as practiced at UTH is a major source of cross-infection among burns patients at the local burn unit. As such, there is no agreed departmental protocol on this subject. Other burn centers from around the world have formulated protocols based on their evidence from research.

1.4 STUDY JUSTIFICATION

Based on the audit reports from the department of surgery, and the culture results from the microbiology department, it is clear that wound colonisation followed by infections in burns cause significant morbidities and mortalities in the burns wards at UTH. Research on burns will help us know infection rates, know the routes of spread of this infection, control infections, keep epidemics under control and formulate hospital protocols on the management of burns. This is because currently there is divided opinion on the subject.

1.5 RESEARCH QUESTION

Does hydrotherapy practices (especially the common bath tub) at the UTH contribute to cross infection in burns patients, and, what is the prevalence of culture positive swabs in these patients at the UTH?

1.6 HYPOTHESIS

ALTERNATE HYPOTHESIS

Hydrotherapy as practiced at UTH contributes to cross infection among burns patients, and the prevalence of culture positive swabs in patients practicing the common hydrotherapy techniques at UTH (using the common bath tub, wet soaks) is more than 50%.

CHAPTER TWO

2.1 OBJECTIVES

GENERAL OBJECTIVE

To determine if hydrotherapy practices at UTH (using the common bath tub, wet soaks) plays a role in cross-infections between burns patients.

SPECIFIC OBJECTIVES

i. To determine the prevalence of culture positive swabs in burns patients (all of which use the common bath tub for hydrotherapy) admitted to the burns unit, thus determining the infection rate in our burns wards.
ii. To determine the prevalent organisms responsible for burn wound colonisation and sepsis in burns patients (all of them use the tub for hydrotherapy).
iii. To determine the organisms present in the bath tubs, the major hydrotherapy equipment, that are used by the burns patients.
iv. To determine if there are organisms in the tap water.
v. To determine the organisms being spread by cross-infection and contamination of hydrotherapy equipment.

CHAPTER THREE

3.1 RESEARCH METHODS

Study design: This was a prospective analytical study (explained in detail under procedure and technique).

Study site: The study was conducted in the Department of Surgery at the University Teaching Hospital, Lusaka. Patients were recruited upon their first presentation to hospital in the surgical admission wards. Follow up was done on an in-patient basis in the surgical wards.

Study duration: Six months (April to September 2015).

Target population: All burns patients.

Study population: Patients with burns satisfying the inclusion criteria.

3.2 INCLUSION CRITERIA

a. Patients who presented to the hospital with burns and eventually ended up being admitted to the burns units.
b. Any age.
c. Patients with burns greater than or equal to 10% (children) or 15% (adults) TBSA.
d. Patients with burns to special areas (e.g. face, perineum, hands, feet, joints), regardless of the percentage.
e. Patients that gave informed consent i.e. either the patient consenting for themselves or consent obtained from patient's legal guardian.

3.3 EXCLUSION CRITERIA

a. Non-consenting patients.
b. Burns patients that were likely to spend less than four (4) days in the burns unit (i.e. those admitted just to be taught how to clean the wounds).

3.4 SAMPLING

Sampling strategy: Convenience (or opportunity) sampling was used. This means patients were recruited that satisfied the inclusion criteria based on ready availability, that is, on admission.

3.5 SAMPLE SIZE

Using the prevalence formula and using our hypothesis, this becomes:

$$N = \frac{Z^2 \text{ X } P\ (1 - P)}{D^2}$$

Where N = sample required

Z = Z statistic (usually 1.96)

P = the expected prevalence (in this case we shall use the conservative, 50%)

D = accepted accuracy range (+/- 10%)

$$N = \frac{1.96^2 \text{ X } 0.5\ (1 - 0.5)}{0.1^2}$$

$$N = 96$$

3.6 STUDY PROCEDURES

Procedure

Patients that presented with burns were recruited. They underwent a careful history followed by clinical examination to determine percentage TBSA and the location of the burn wounds. Data was collected using a data collection sheet. Enrollment was then determined by the inclusion/ exclusion criteria.

After proper patient (or parent) counseling, all patients (or parents) were required to fill in a written informed consent. Pus swabs of the burns wounds were collected in the surgical admission wards, to mark day 0 swabs. Follow up swabs were collected on day 4 and day 7 from the patients' respective burns units (G-wards), to mark day 4 and day 7 swabs. Weekly swabs of the hydrotherapy equipment (the bath tubs in this case) and tap water were obtained. Selected samples of culture results were subjected to PCR, based on possible similarities to cultures from the hydrotherapy equipment and tap water.

Technique

Refer to appendix D (Republic Of Zambia, Ministry Of Health, Standard Operating Procedures for Hospital Laboratories Level III).

The procedure of pus swab collection was explained to the patient (or parent). The patient was positioned in the most comfortable position for the procedure, depending on the site of the burns. Procedure was done under clean conditions to avoid external contamination. A representative swab was collected from the burns wounds, and the specimen secured immediately in the appropriate container. This was taken immediately to the lab for culture and sensitivity studies. In a similar manner, swabs were collected from the bath tubs and sent to the lab for microbiology studies.

3.7 VARIABLES

Dependent (outcome) variable: Culture result from microbiology studies.

Independent (exposure) variables: These included age, sex, causative agent of the burns, percentage TBSA of the burns, treatment outcomes (discharged/ still on the ward/ mortality), wound contamination by caregivers and/or hospital staff, wound contamination from contaminated beds, mattresses, etc.

Categorical variables included sex (male/female), treatment outcomes (discharged/ still on the ward/ mortality)

Continuous variables included age, causative agent of the burns, percentage TBSA of the burns, wound contamination by caregivers and/or hospital staff, wound contamination from contaminated beds, mattresses, etc.

Potential confounders: These included wound contamination by caregivers and/or hospital staff, wound contamination from contaminated beds, mattresses, etc.

3.8 DATA MANAGEMENT

Data collection: This was done with the aid of the burns chart of the University Teaching Hospital, and the attached result sheet (appendix A)

Data entry: The data collected was entered into an excel spread-sheet for analysis.

Statistical analysis: A statistical software, SPSS version 23, was used to analyse the collected results. A statistician was consulted for guidance.

3.9 ETHICAL CONSIDERATIONS

Permission was obtained from UTH Management, Microbiology department and Surgery department. Ethical approval was obtained from the University of Zambia Biomedical Research Ethics Committee (UNZABREC).

Participation in this study was voluntary. This study did not affect the patient's management during period of study. Patients were not remunerated. All information obtained was kept confidential. Patients were free to withdraw from the study at any time and with no penalty to them.

All the investigations done were by qualified personnel. Pus swab is a non-invasive procedure. The only anticipated risk to the patient was minimal discomfort. A written consent was obtained from every patient.

CHAPTER FOUR

4.0 RESULTS

4.1 CHARACTERISTICS OF PATIENTS

This study enrolled the required number of 96 participants. All of them fulfilled the inclusion criteria.

4.1.1 Sex distribution

Of the 96 participants, 53.1% (n = 51) were males while 46.9% (n=45) were females.

Table 1 – Sex distribution of patients in the study

	Frequency	Percent
Male	51	53.1
Female	45	46.9
Total	96	100.0

4.1.2 Age distribution

The age distribution ranged from 5 months old to 91 years old. The modal age range was 1 to 2 years old.

Table 2 – Age distribution of patients

Age (yrs)	Frequency	Percent	Cumulative Percent
1-2	37	38.5	38.5
3-4	12	12.5	51.0
5-6	12	12.5	63.5
7-10	11	11.5	75.0
21+	24	25.0	100.0
Total	96	100.0	

4.1.3 Distribution by wards

The majority of the patients came from ward G12 (n = 69, 71.8%)

Table 3 – Distribution of patients by ward

Ward	Frequency	Percent
G12	69	71.8
G02	24	25.0
G21	3	3.1
Total	96	100.0

4.1.4 Burn percentage of TBSA

The majority of the patients had burn percentage in the range 6% to 10% (n = 30). Only 4 patients had a percentage above 30% of TBSA.

Table 4 – Burn percentage of TBSA

Burn % of TBSA	Frequency	Percent	Cumulative Percent
1-5	18	18.8	18.8
6-10	30	31.3	50.0
11-15	25	26.0	76.0
16-20	12	12.5	88.5
21-25	6	6.3	94.8
26-30	1	1.0	95.8
31+	4	4.2	100.0
Total	96	100.0	

4.1.5 Burn agent

Hot water was the cause of burns in 63 patients (65.6%), followed by open flame fire in 15 (15.6%). Frictional burns accounted for only 1%.

Table 5 – Burn agent

	Frequency	Percent	Cumulative Percent
Hot water	63	65.6	65.6
Fire	15	15.6	81.3
Cooking oil	10	10.4	91.7
Hot porridge	7	7.3	99.0
Frictional burns	1	1.0	100.0
Total	96	100.0	

4.1.6 Patient outcome

The majority of the patients (n = 70) were discharged. The mortality rate in this study was 19.8% (n = 19). Seven patients left against medical advice.

Table 6 – Patient outcome

	Frequency	Percent
Discharged	70	72.9
Died	19	19.8
LAMA	7	7.3
Total	96	100.0

4.1.7 Percentage TBSA burns for those who died

Most of those who died (52.6%) had burns equal to or less than 15% of the TBSA.

Table 7 – Percentage TBSA of burns for those who died

Burn % of TBSA	Frequency	Percent	Cumulative Percent
1-5	1	5.3	5.3
6-10	4	21.0	26.3
11-15	5	26.3	52.6
16-20	3	15.8	68.4
21-25	3	15.8	84.2
26-30	0	0.0	84.2
31+	3	15.8	100.0
Total	19	100.0	

4.1.8 Age distribution of patients that died

More than 50% of those that died were children less than 5 years old.

Table 8 – Age distribution of patients who died

Age (yrs)	Frequency	Percent	Cumulative Percent
1-2	7	36.8	36.8
3-4	4	21.1	57.9
5-6	2	10.5	68.4
7-10	3	15.8	84.2
21+	3	15.8	100.0
Total	19	100.0	

4.2 ORGANISMS ISOLATED AND THEIR SENSITIVITIES

The following section contains results of the organisms that were isolated from the burn wounds, and their sensitivities to antibiotics. The organisms analysed in this section are *Staphylococcus aureus, Klebsiella pneumoniae* and *Pseudomonas aeruginosa.* Staphylococcus and Klebsiella happened to be the commonest organisms that were isolated from the burn wounds and the bathtubs. The enteric organisms are not included in this section as they are considered to have been due to faecal contamination of the wounds and the bathtubs. The following are the enteric organisms that have been left out: *Citrobacter diversus, Citrobacter freundii, Enterobacter aerogenes, Enterobacter agglomerans, Enterobacter cloacae, Escherichia coli* and *Serratia marcescens*. Surprisingly, there wasn't much *Escherichia coli* that was isolated.

4.2.1 Day 0 (admission day) results

The prevalence of culture positive swabs was 88.5%. On admission day, no *Pseudomonas aeruginosa* was isolated. No growth was obtained from the water samples.

Staphylococcus aureus

This figure shows the sensitivity pattern of the *Staphylococcus aureus* that was isolated on admission day.

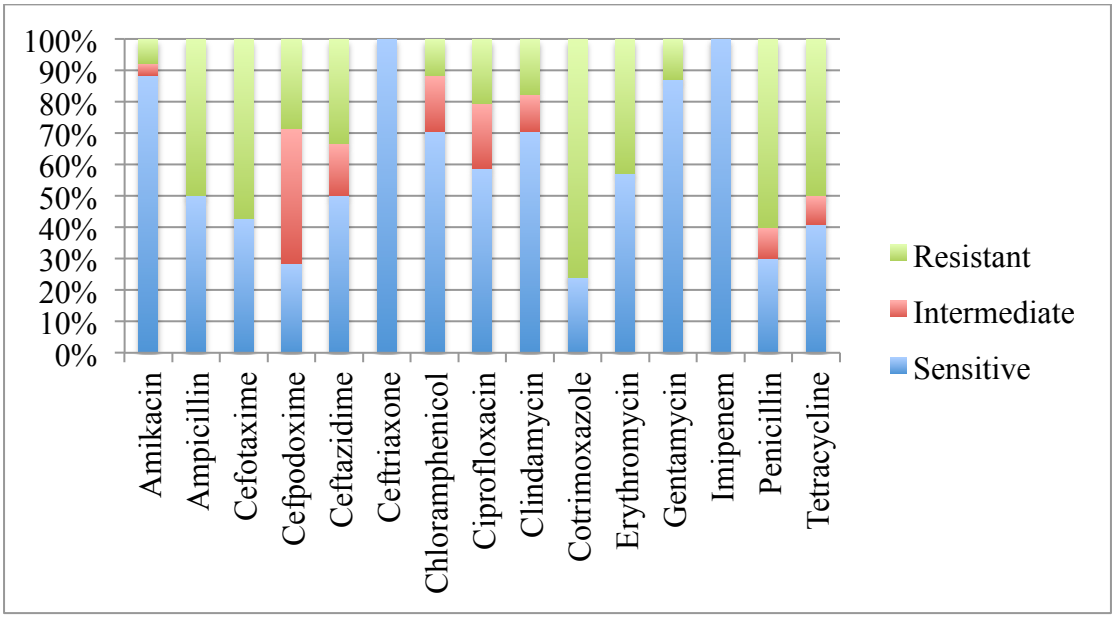

Figure 1 – Day 0 sensitivity pattern for *Staphylococcus aureus*

Klebsiella pneumoniae

This figure shows the sensitivity pattern of the *Klebsiella pneumoniae* that was isolated on admission day.

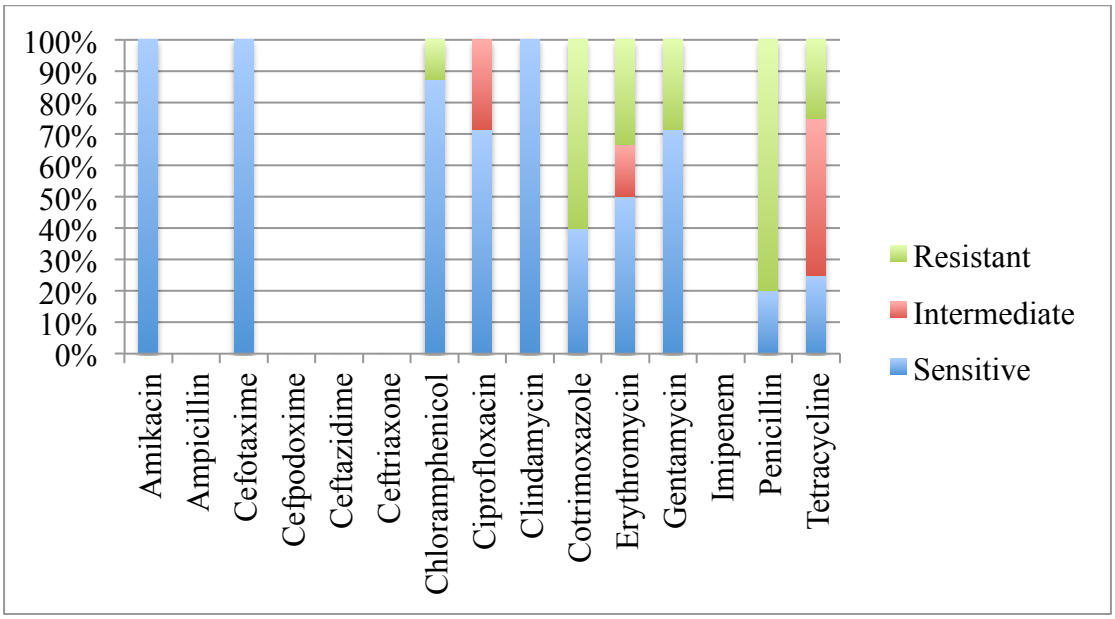

Figure 2 – Day 0 sensitivity pattern for *Klebsiella pneumoniae*

Statistical comparison of antibiotics

Three antibiotics were compared to see if the differences in their sensitivities were of statistical significance. These antibiotics were Ciprofloxacin, Amikacin and Chloramphenicol. Ciprofloxacin is the commonly used antibiotic at this institution and so its activity was compared with the other two using the paired t-test. The three antibiotics were chosen on the basis of common or readily available drugs.

Table 9: Summary results of paired-samples t test on admission day (day 0)

Bacteria	Pairs of drugs	95% CI		t	df	P value
		Lower limit	Upper limit			
Staphylococcus	Amikacin (n=18; $\bar{x}$ =.89) – Ciprofloxacin (n=18; $\bar{x}$ =.44)	-.870	-.019	-2.204	17	.042
	Amikacin (n=19; $\bar{x}$ =1.00) - Chloramphenicol (n=19; $\bar{x}$ =.79)	-.093	.514	1.455	18	.163
	ciprofloxacin (n=25; $\bar{x}$ =.68) - Chloramphenicol (n=25; $\bar{x}$ =.68)	-.337	.337	.001	24	1.00
Klebsiella[a]	Amikacin (n=4; $\bar{x}$ =1.00) - Chloramphenicol (n=4; $\bar{x}$ =.50)	-2.091	1.091	-1.000	3	.391

[a]Due to insufficient data we could not run tests on ciprofloxacin and amikacin, ciprofloxacin and chloramphenicol, and ciprofloxacin and amikacin.

4.2.2 Day 4 results

The prevalence of culture positive swabs on day 4 was 98.9%. Again, no growth was obtained from the water samples.

Staphylococcus aureus

The figure below shows the sensitivity pattern of the *Staphylococcus aureus* isolated from the patients on day 4.

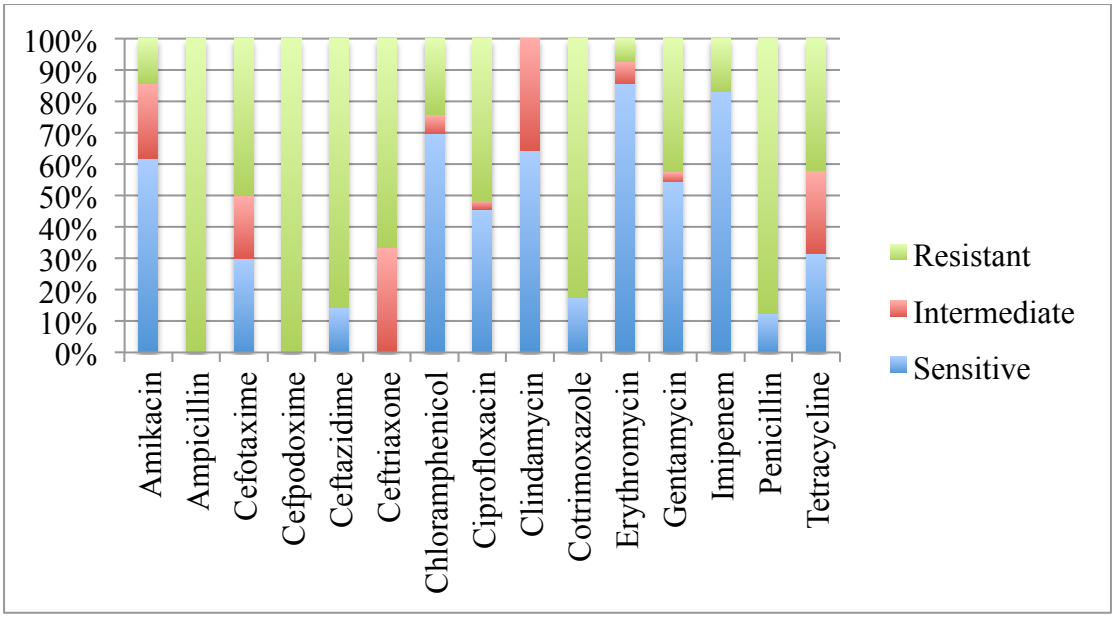

Figure 3 – Day 4 sensitivity pattern for *Staphylococcus aureus*

Klebsiella pneumoniae

The figure below shows the sensitivity pattern of the *Klebsiella pneumoniae* that was isolated from patients on day 4.

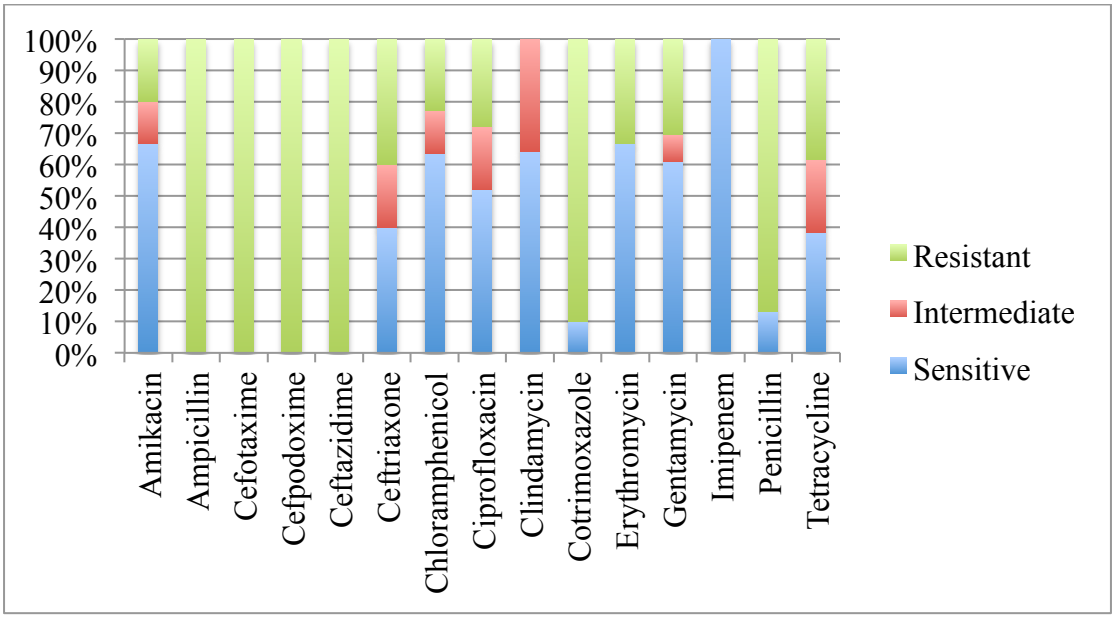

Figure 4 – Day 4 sensitivity pattern for *Klebsiella pneumoniae*

Pseudomonas aeruginosa

On day 4, *Pseudomonas aeruginosa* was isolated from some patients. The figure below shows the sensitivity pattern of this organism.

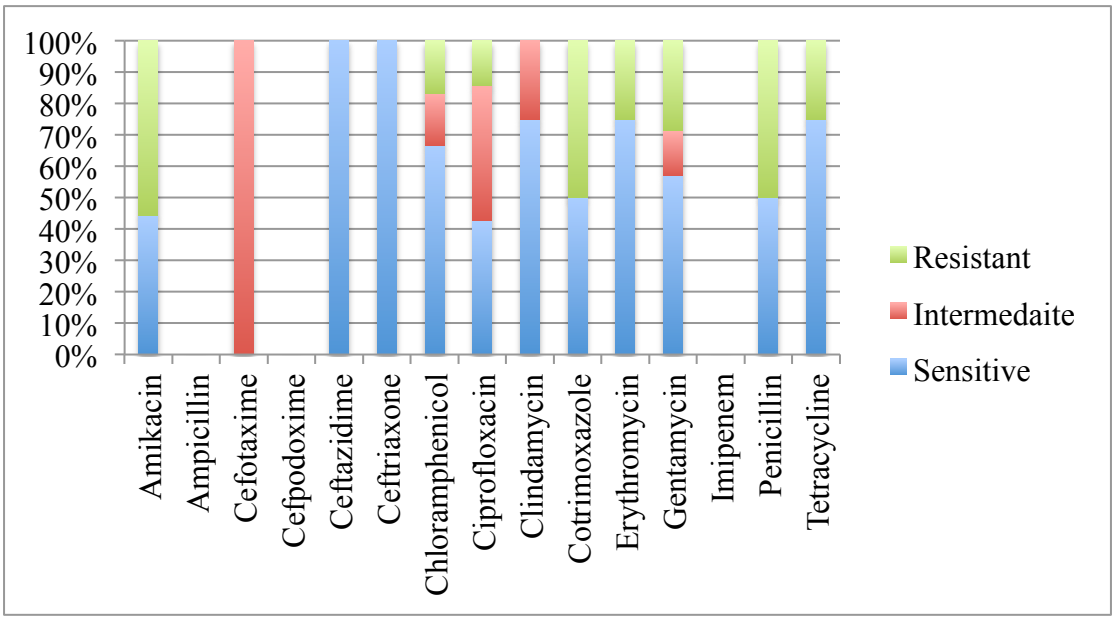

Figure 5 – Day 4 sensitivity pattern for *Pseudomonas aeruginosa*

Table 10: Summary results of paired-samples t test on day 4

Bacteria	Pairs of drugs	95% CI		t	df	P value
		Lower limit	Upper limit			
Staphylococcus	Amikacin (n=14; $\bar{x}$ =1.00) – Ciprofloxacin (n=14; $\bar{x}$ =.43)	.030	1.113	2.280	13	.040
	Amikacin (n=16; $\bar{x}$ =1.00) - Chloramphenicol (n=16; $\bar{x}$ =.88)	-.391	.141	-1.000	15	.333
	Ciprofloxacin (n=22; $\bar{x}$ =.55)-Chloramphenicol (n=22; $\bar{x}$ =.73)	-.560	.196	-1.000	21	.329
Klebsiella	Amikacin (n=8; $\bar{x}$ =.75) – Ciprofloxacin (n=8; $\bar{x}$ =.25)	-.274	1.274	1.528	7	.170
	Amikacin (n=8; $\bar{x}$ =1.00) - Chloramphenicol (n=8; $\bar{x}$ =.75)	-.841	.341	-1.000	7	.351
	Ciprofloxacin (n=11; $\bar{x}$ =.45)-Chloramphenicol (n=11; $\bar{x}$ =1.00)	-1.173	.082	-1.936	10	.082
Pseudomonas[a]	Amikacin (n=3; $\bar{x}$ =1.00) – Ciprofloxacin (n=3; $\bar{x}$ = .33)	-2.202	3.535	1.000	2	.423
	Ciprofloxacin (n=5; $\bar{x}$ =.60)-Chloramphenicol (n=5; $\bar{x}$ =1.00)	-1.511	.711	-1.000	4	.374

NB: [a]Could not run tests on chloramphenicol and amikacin due to insufficient data.

4.2.3 Day 7 results

The prevalence of culture positive swabs on day 7 was 97.50%. Like before, no growth was obtained from the water samples.

Staphylococcus aureus

The figure below shows the sensitivity pattern of the *Staphylococcus aureus* that was isolated from patients on day 7 of their admission to hospital.

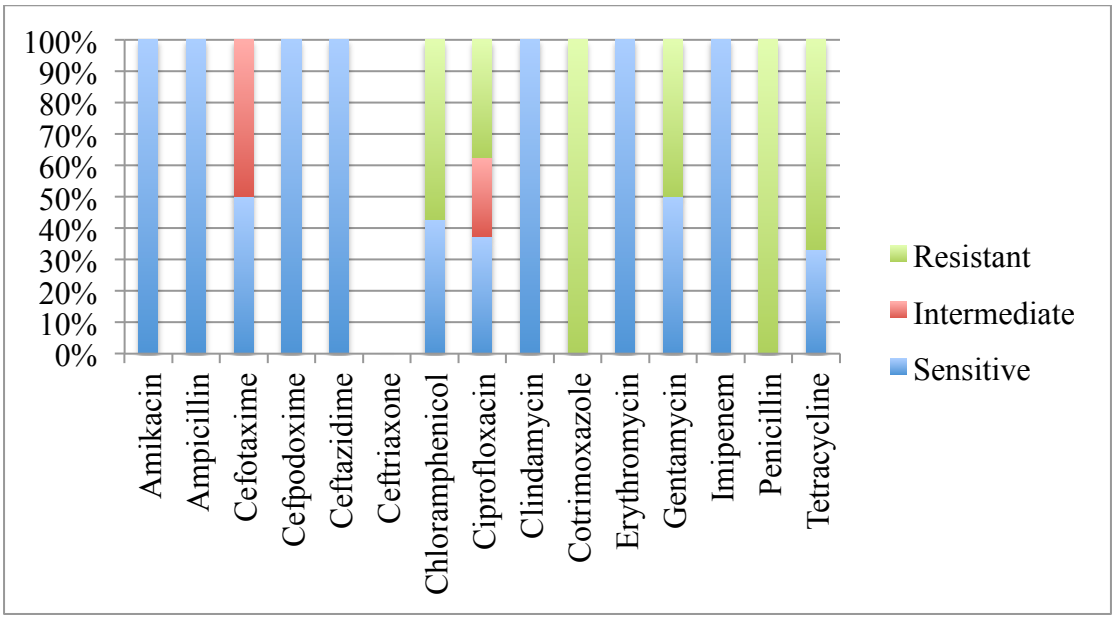

Figure 6 – Day 7 sensitivity pattern for *Staphylococcus aureus*

Klebsiella pneumoniae

The figure below shows the sensitivity pattern of the *Klebsiella pneumoniae* that was isolated from patients on day 7 of their admission to hospital.

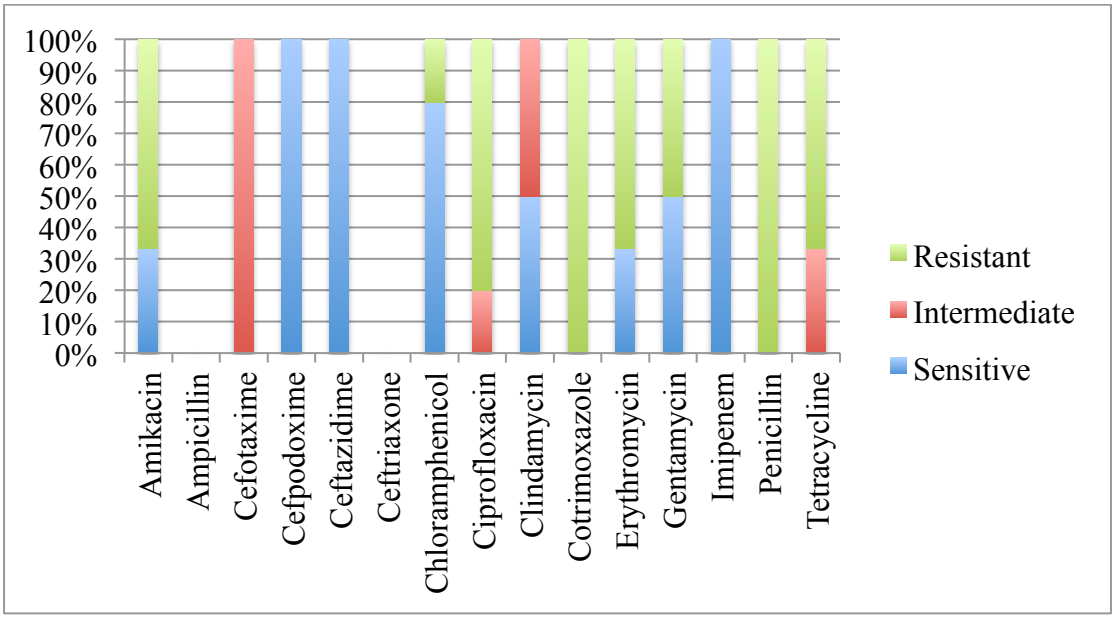

Figure 7 – Day 7 sensitivity pattern for *Klebsiella pneumonia*

Pseudomonas aeruginosa

The figure below shows the sensitivity pattern of the *Pseudomonas aeruginosa* that was isolated from patients on day 7 of their admission.

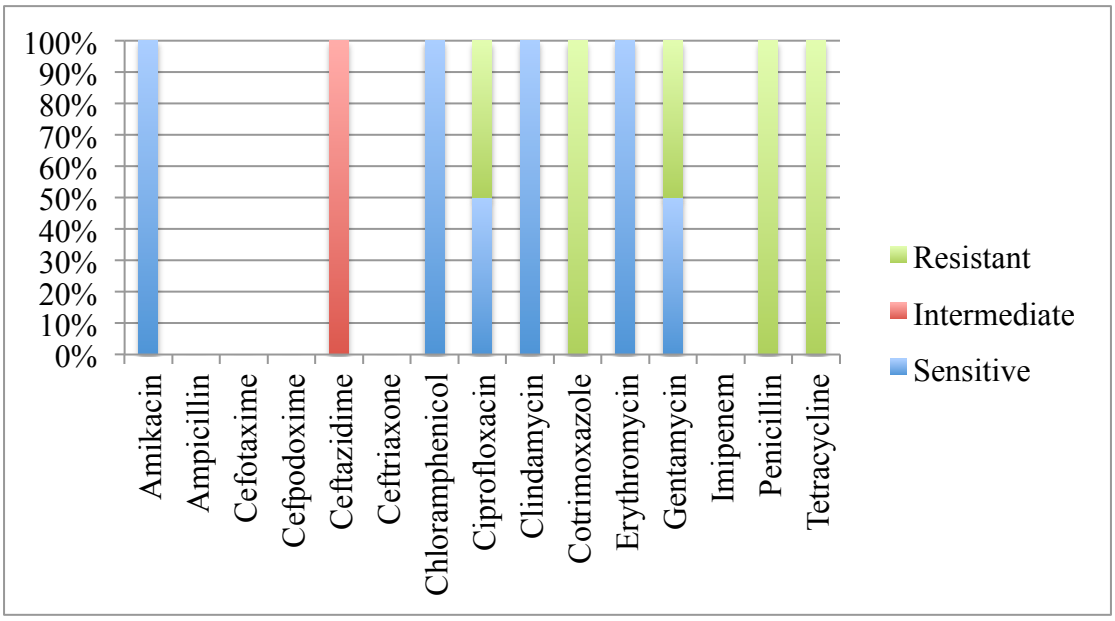

Figure 8 – Day 7 sensitivity pattern for *Pseudomonas aeruginosa*

Table 11: Summary results of paired-samples t test on day 7

Bacteria	Pairs of drugs	95% CI		t	df	P value
		Lower limit	Upper limit			
Staphylococcus	Amikacin (n=8; $\bar{x}$ = 1.00) – Ciprofloxacin (n=8; $\bar{x}$ =.75)	-.841	.341	-1.000	7	.351
	Amikacin (n=9; $\bar{x}$ =1.00) - Chloramphenicol (n=9; $\bar{x}$ =.78)	-.290	.735	1.000	8	.347
	Ciprofloxacin (n=10; $\bar{x}$ =.60)- Chloramphenicol (n=10; $\bar{x}$ =.80)	-.652	.252	-1.000	9	.343
Klebsiella[a]	Amikacin (n=2; $\bar{x}$ =1.00) – Ciprofloxacin (n=2; $\bar{x}$ =.01)	-13.706	11.706	-1.000	1	.500
	Ciprofloxacin (n=3; $\bar{x}$ =.33)- Chloramphenicol (n=3; $\bar{x}$ =1.00)	-3.535	2.202	-1.000	2	.423
[b]Pseudomonas	Ciprofloxacin (n=3; $\bar{x}$ =.33)- Chloramphenicol (n=3; $\bar{x}$ =1.00)	-3.535	2.202	-1.000	2	.423

NB: [a]Could not run tests on chloramphenicol and amikacin due to insufficient data. [b]Could not also run tests on amikacin and ciprofloxacin, and amikacin and chloramphenicol due to insufficient data.

4.2.4 Bathtub results

This section shows results of bacteria that were cultured from the bathtubs. As was the case with cultures from the patients, the commonest organisms were *Staphylococcus aureus, Klebsiella pneumoniae* and the enteric organisms. Surprisingly, there was no *Pseudomonas aeruginosa* that was isolated from the bathtubs despite its preponderance for aquatic environments.

Staphylococcus aureus

The figure below shows the sensitivity pattern of the *Staphylococcus aureus* that was isolated from the bathtubs.

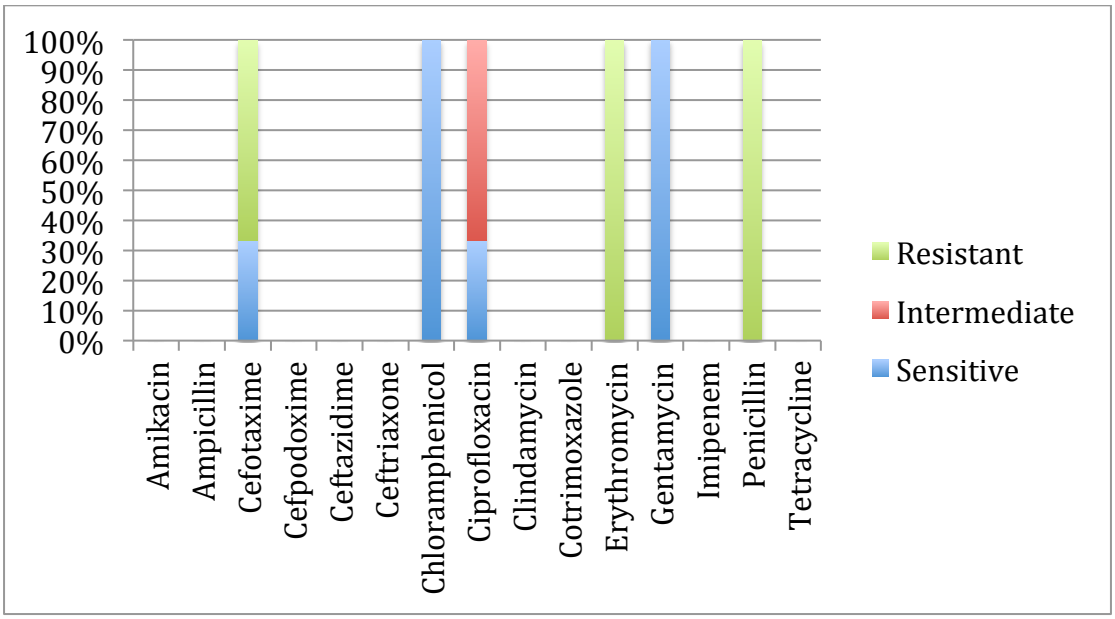

Figure 9 – Bathtub sensitivity pattern for *Staphylococcus aureus*

Klebsiella pneumoniae

The figure below shows the sensitivity pattern of the *Klebsiella pneumoniae* isolated from the bathtubs.

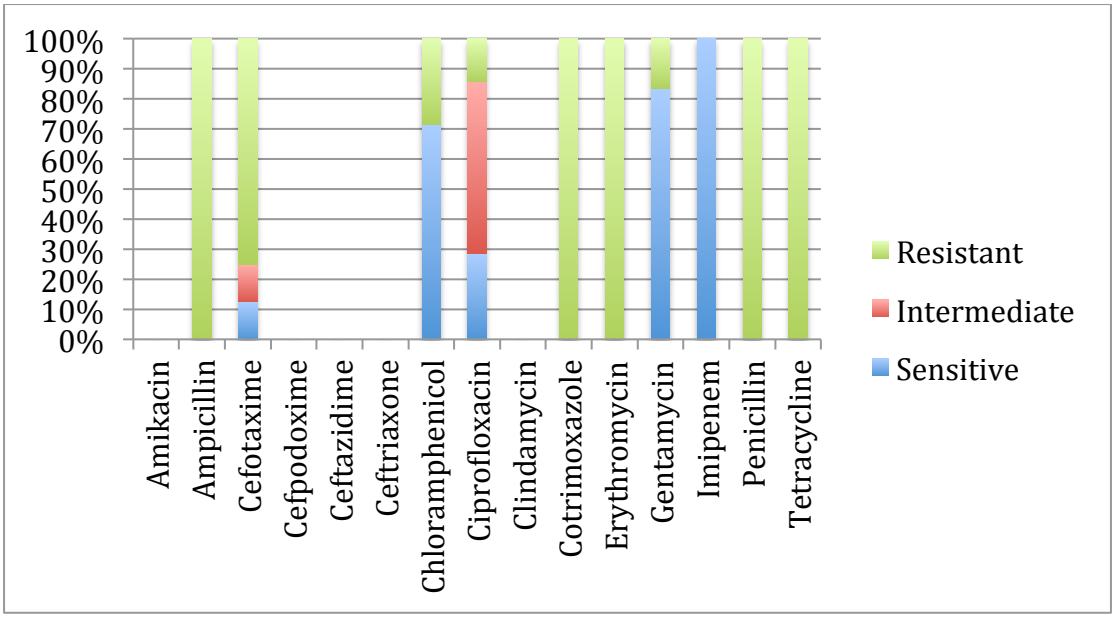

Figure 10 – Bathtub sensitivity pattern of the *Klebsiella pneumoniae*

Table 12: Summary results of paired-samples t test on bathtub samples

Bacteria	Pairs of drugs	95% CI		t	df	P value
		Lower limit	Upper limit			
[a]Staphylococcus	Amikacin (n=7; $\bar{x}$ =1.00) – Ciprofloxacin (n=7; $\bar{x}$ = .43	-1.474	.331	-1.549	6	.172
	Amikacin (n=7; $\bar{x}$ = 1.00) - Chloramphenicol (n=7; $\bar{x}$ = .43)	-.331	1.474	1.549	6	.172
Klebsiella[b]	Amikacin (n=7; $\bar{x}$ = 1.00) – Chloramphenicol (n=7; $\bar{x}$ =.71)	-.413	.985	1.000	6	.356

NB: [a]Could not run tests on ciprofloxacin and chloramphenicol due to insufficient data. [b]Could not run tests on ciprofloxacin and amikacin due to insufficient data. Could not also run tests on pseudomonas due to insufficient data.

4.3 MOLECULAR WORK ON KLEBSIELLA AND STAPHYLOCOCCUS

Klebsiella pneumoniae

Bla Gene	No. of isolates	Frequency (%)
Bla_{TEM}	4	23.5
Bla_{SHV}	5	29.4
Bla_{TEM} and; Bla_{SHV}	8	47.1
Total	17	100

Table 13 – Frequency of *Klebsiella pneumoniae* isolates with Genes encoding ESBL-production

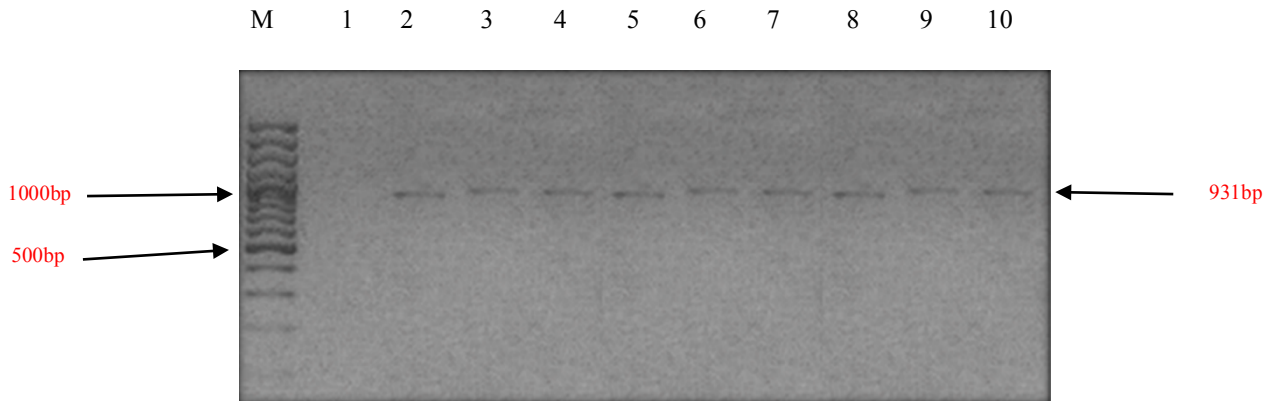

Figure 11 – PCR Detection of Bla_{TEM} ESBL genes. Lane M: 100bp DNA Marker, Lane1: Negative control, Lane 2: Positive control, Lane 3 – 10: Positive isolates for Bla_{TEM}

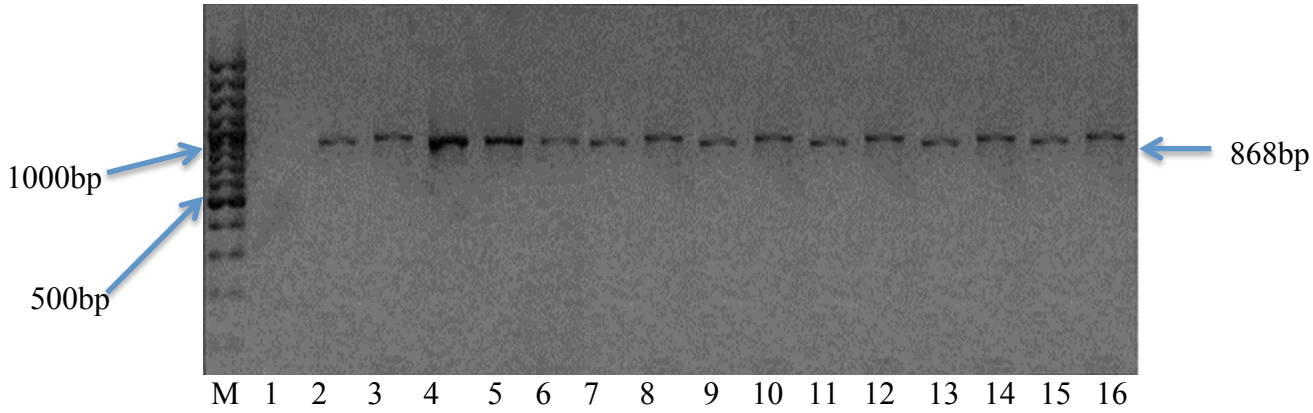

Figure 12 – PCR detection of Bla_{SHV} ESBL genes. Lane M: 100bp DNA Marker; Lane 1: Negative control; Lane 2: ATCC Positive control, Lanes 3-16: Isolates Positive for Bla_{SHV} gene

Staphylococcus aureus

Staphylococcus	No. of isolates	Frequency (%)
MRSA	26	30.6
*PVL*gene	10	11.8
*SPA*gene	30	35.3
Others	19	22.3
Total	85	100

Table 14 – Frequency of *Staphylococcus aureus* isolated.

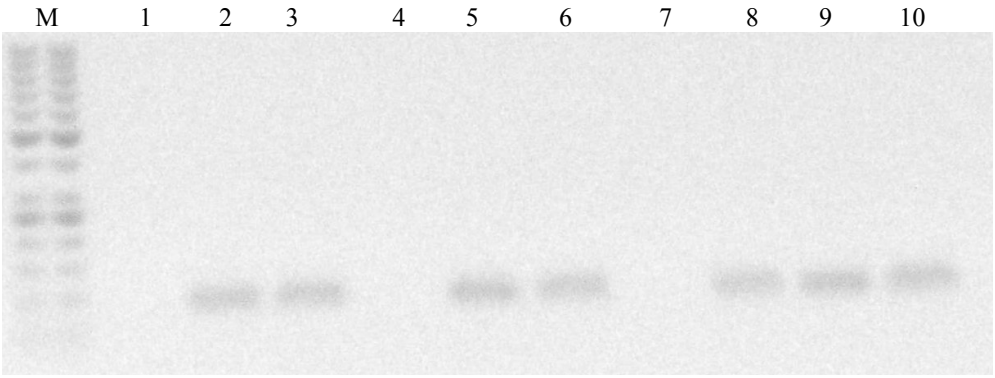

Figure 13 - PVL gene gel picture of controls and selected samples. M: 50bp marker; Lane 1: Negative control; Lane 2: Positive control; Lane 3: 640; Lane 4: 577; Lane 5:599; Lane 6: 1091; Lane 7: 576; Lane 8: 1401; Lane 9: 1408; Lane 10: 1419

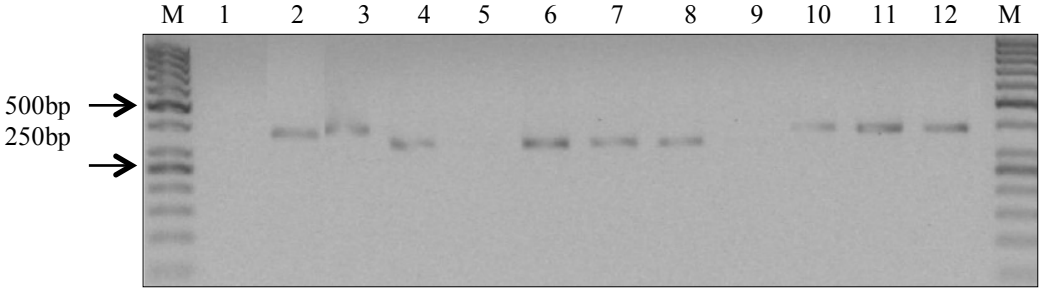

Figure 14 - *spa* typing gel picture of controls and selected samples. M: 50bp marker; Lane 1: Negative control; Lane 2: Positive control; Lane 3: 712; Lane 4: 725; Lane 5:577; Lane 6: 747; Lane 7: 1096; Lane 8: 1091; Lane 9: 1092; Lane 10: 1089; Lane 11: 978; Lane 12: 964

CHAPTER FIVE

5.1 DISCUSSION

In this study, 96 patients were enrolled. This was also the calculated sample size. All of them fulfilled the inclusion criteria. Of the 96 participants, 53.1% (n = 51) were males while 46.9% (n = 45) were females. Thus the ratio of male to female participants was nearly 1:1.

The age distribution of the participants ranged from 5 months to 91 years old. Thirty-seven of the participants (38.5%) were 2 years old or below. More than 50% of the participants were under 5 years old. This was in agreement with what has been observed in the department, as stated in the statement of the problem. It is also in line with what was reported in a systematic review done by Kun Zou et al.[26]

In terms of ward distribution, 71.8% (n = 69) came from ward G12 while 25.0% (n = 24) were from ward G02. Only 3.1% (n = 3) were from ward G21. Ward G12 had more patients because three of the five general surgical units admitted to that ward. The remaining two units admitted to ward G02. The three patients that were admitted to ward G21 happened to have been victims of an industrial accident. They were adult males who had slept after getting drunk.

The modal range of the burn percentage of the TBSA was 6 – 10%. About 76% of the participants had burn percentage of 15% and less. Most of those that died, 52.6% (n = 10), had burns equal to or less than 15% of the TBSA. This is in contrast to the documentation by Karim Rafla and Edward E. Tredget who documented percentage of TBSA greater than 30% as one of the patient characteristics that influenced morbidity and mortality from burn wound infection and sepsis.[14]

Hot water (scalds) was the cause of burns in 65.6% (n = 63) of the participants. The very young usually spend most of their time with their mothers, including when their mothers are cooking. Some of these accidents happened as the mother lost her concentration on the child. The child pulled a pot containing boiling water or porridge onto herself/ himself. Unlike what was found in the review by Kun Zou et al[26] where they found more evidence that home safety interventions were effective in promoting

safe hot tap water temperature, most of these patients came from homes without hot tap water/ geysers. Thus for effective preventive measures, parental education especially on safety in the kitchen and/or the cooking environment would be paramount.

Fire (open flame) was the cause in 15.6% (n = 15) of the participants. These were mostly epileptics that had suffered an attack and fallen onto a fire, in the absence of an attendant. A few sustained burns from fire following a house they were in catching fire; such usually came with a very large percentage of burns. One patient (a victim of gender-based violence) was set ablaze by her husband after he had poured petrol on her. This was after a domestic dispute. The other causes of burns were cooking oil, hot porridge and friction. Frictional burns were due to a patient falling from a moving vehicle. She presented with wounds to her joints, thus classified as wounds to special areas.

The mortality rate in this study was 19.8% (n =19). Seven patients (7.3%) left against medical advice while 72.9% (n = 70) of the participants were discharged. More than 50% of those that died were children less than 5 years old. As already pointed out above, most of those that died had burn percentages of less than or equal to 15% of the TBSA. A number of reasons can be attributed to patients leaving against medical advice. Most of the patients that got burnt had mothers that rarely spent a lot of time with their children at home. This tended to give challenges when it came to treatment compliance from the mothers. The other reason was a cultural myth – seeing a number of other burns children dying created a fear in some mothers who felt running away with their babies was a better option.

Several organisms were isolated from the patients and the bathtubs. A similarity was noted between those from the patients and those from the bathtubs. Also, there were quite a number of organisms isolated from both patients and bathtubs that were thought to be due to wound or tub contamination. These were the enteric organisms. The enteric organisms that were isolated from both the patients and bathtubs are: *Citrobacter diversus, Citrobacter freundii, Enterobacter aerogenes, Enterobacter agglomerans, Enterobacter cloacae, Escherichia coli* and *Serratia marcescens.* The other organisms isolated that were not enteric in origin were *Staphylococcus aureus,*

Klebsiella pneumoniae and *Pseudomonas aeruginosa.* The idea that enteric organisms were contaminants is the main reason why only the other three were analysed in detail. The other reason was due to resource limitation. As mentioned in the results section, there was no *Pseudomonas* isolated from the bathtub. This is despite its preponderance for aquatic environments. This was a rather surprising finding.
This study was conducted in the winter season, April to September 2015. During the winter season, the turnover rate of patients in the burns ward is quite high. This is because the incidences of burns tend to go up. In trying to keep warm, children as well as adults, want to stay near a source of heat. Sometimes a brazier or heater is left on while people have fallen asleep. Bathing water is also warm to hot. As such, accidents from fires and scalds are very likely to happen during the cold season. Due to the high turnover, patients were usually discharged early from the burns wards to create space for others. As such, not all of them had their day 7 swabs taken. The main criteria for discharge was a clean wound (clinically), absence of fever and caretakers that had learned how to take care of the wound. The observation made in this study was that the wounds that appeared clean clinically actually harbored *Klebsiella pneumoniae* (some of which were ESBL producers) and *Staphylococcus aureus* (some of which was MRSA, PVL or SPA genes positive).

Most of the swabs collected from patients yielded positive results. On day 0 (admission day), 88.5% of the swabs were positive. The prevalence of culture positive swabs on days 4 and 7 were 98.9% and 97.5%, respectively. Another observation that was made on analysing the three organisms isolated from the patients was that their resistance to antibiotics tended to increase as the patient stayed longer in hospital i.e. *Staphylococcus* isolated on day 4 was less sensitive to most antibiotics compared to the staphylococcus that was isolated on day of admission. This was equally true for *Klebsiella* and *Pseudomonas.* This observation agrees with the statements by Karim Rafla and Edward E. Tredget in their paper "infection control in the burn unit."[14] In their review paper, they state that the patient is initially infected with gram-positive organisms, which are rapidly replaced by antibiotic-susceptible gram-negative organisms. The antibiotic-susceptible gram-negative organisms are then later replaced by those that are antibiotic-resistant. The source of the organisms could be from poorly decontaminated equipment. Relating the above statements to our study, we could notice that the bacteria isolated on admission was more sensitive than the one

isolated on day 4 or day 7. It indicates that the more resistant bacteria must have replaced the antibiotic-susceptible ones. The bathtubs had organisms that were more resistant to antibiotics. It is highly likely that this could have been the main source of the antibiotic-resistant organisms. However, in this study, there was no yeast that was isolated.

The differences in sensitivity to antibiotics was subjected to analysis for statistical significance. Comparisons with Ciprofloxacin were made. As already stated, Ciprofloxacin is the commonly used antibiotic in our burn ward. It was noted that Amikacin performed better than Ciprofloxacin on admission day in treating the isolated Staphylococcus (P = 0.042). There was no statistical significance when Ciprofloxacin was compared with Chloramphenicol, or Amikacin with Chloramphenicol. Similarly, there was statistical significance when Amikacin was compared with Ciprofloxacin in treating Staphylococcus on day 4 samples (P = 0.040). The other comparisons were not statistically significant. Amikacin and Chloramphenicol were chosen for comparisons because they are readily available at the institution than the other antibiotics.

The *Klebsiella* that was isolated, both from the patients and bathtubs, was tested further for the presence of ESBL genes. Sixty-five point four percent (65.4%) tested positive for the ESBL genes. Among those with ESBL genes, 29.4% had the SHV gene only while 23.5% had the TEM gene only. The majority of them (47.1%) had both SHV and TEM genes. There was no CTX gene that was detected. There was none of the three genes detected in 34.6% of the *Klebsiella*. Yet a good number of them showed wide resistance against most of the antibiotics tested. This implied that they could have had other genes besides the SHV, TEM and CTX that were responsible for their resistance.

The presence of ESBL producers in our burns unit with such a high prevalence of culture-positive swabs is of very serious concern. Mark E. Rupp and Paul D. Fey bring out several points in their review paper on ESBL enterobacteriaceae.[26] Our study yielded quite a number of enteric bacteria, both from the patients as well as the tubs. Enteric organisms are known to exchange resistance genes. This is a very high possibility in our burn unit. It has been said that often times, a single strain or a

genetically related group of strains expand clinically in an institution. Endemic strains have been shown to persist in certain units for years. In that same paper, it is said that the current recommendation is that any organism found to produce an ESBL be regarded as resistant to all extended spectrum beta-lactam antibacterials regardless of the in vitro minimum inhibitory concentration (MIC) results. This now puts us in a serious dilemma due to the limited choice of antibiotics we have as readily available. Most of our patients are prescribed Ciprofloxacin (a fluoroquinolone), but as can be seen in the above figures, it is not as effective as other antibiotics. Kenneth M. Wener et al[27] showed that treatment with fluoroquinolones is a risk factor for isolation of ESBL-producing *Klebsiella* species in hospitalized patients. An important aspect that has not been considered is the impact that these organisms are having on the community out there. This is because (as pointed out earlier) some of the patients were discharged early or left against medical advice, yet their wounds had positive swabs for ESBLs and had not been grafted.

Staphylococcus aureus was the other organism isolated from both the patients and bathtubs. MRSA accounted for 30.6% of the Staphylococcus. PCR was also done to detect the presence of PVL (Panton-Valentine Leukocidin) and SPA (*Staphylococcus aureus* Protein A) genes. It was noted that 11.8% had the PVL gene, 35.3% had the SPA gene and 5.9% had both PVL and SPA genes. The PVL gene is a potent cytotoxin with important virulence in *Staphylococcus aureus*.[29] It is said to cause tissue necrosis, selectively disrupts leukocyte membranes thus leading to enhanced virulence.[29] PVL-carrying *S. aureus* strains have been known to cause serious skin and soft tissue infections and life threatening invasive diseases such as necrotising fasciitis, pulpura fulminans and necrotising haemorrhagic pneumonia (of which upto 75% of cases is lethal)[29]. Our departmental records indicate that pneumonia was a cause of death in 18.5% of the deaths in burns patients in 2012. The above explanation gives an insight of the pathogenesis.

CHAPTER SIX

6.1 CONCLUSION

Hydrotherapy as practiced at the University Teaching Hospital in Lusaka, Zambia does contribute significantly to burn wound bacterial colonization and later infection leading to sepsis in burns patients. The main hydrotherapy equipment (in this case the bathtubs) does act as a reservoir of organisms and a place at which cross infection between patients may take place. A number of resistant organisms are present in these bathtubs. The organisms exchange resistant genes in the bathtubs hence posing a challenge to treatment of infection in burns patients. The decontamination that is done to the bathtubs cannot stop this from happening, as has been shown in this research.

6.2 STUDY LIMITATIONS

- Some patients were discharged prematurely so as to create space in the congested burns ward. This meant that some swabs, especially day 7 swabs, could not be done. Availability of such information would have helped further in the analysis of results. Because of this, the paired t-test could not be done on some results due to insufficiency of data.
- Resource limitation. Our lab couldn't do all tests, and some tests had to be done abroad, thus not all that could have been done was done due to resource limitation. It would have been helpful to do molecular work on other organisms as well.
- There were a number of potential confounders in this study that could have caused cross-infection between patients. These include contaminated hands of caregivers and medical staff.

6.3 RECOMMENDATIONS

1. Formulate protocols for the burns unit. As part of the protocols, make pus swabs collection a routine other than waiting for signs of sepsis before collecting swabs.

2. Establish a burns unit dedicated to the care of burns patients. This should be accompanied by training of staff who will give a standardised care to the burns patients.
3. Maximise on methods of patient isolation so as to minimise infections. Such measures to include:
    a. Introduce showers and do away with bathtubs. In the meantime as we await this, each patient to have his/her own bucket for use when bathing, instead of bathing directly from the bathtub. This will help minimise contact with the highly infectious bathtubs.
    b. Limitation of visitors to the burns wards. The burns ward is supposed to be a no-go area for visitors.
    c. Limit the number of staff entering the burns wards. Hospital workers are known to transmit resistant organisms and this puts these immunocompromised patients at serious risk.
    d. Introduce gowns and shoes for staff entering the burns wards. These should be frequently sterilised. Staff should not enter the burns unit in their clinical coats so as to minimise nosocomial spread of infection.

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APPENDICES

APPENDIX A

BURN CHART

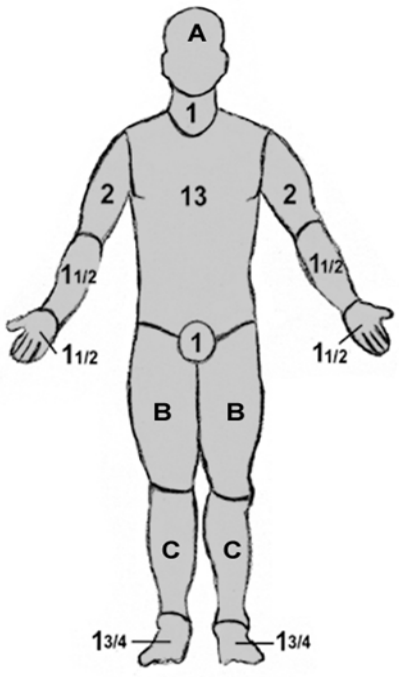
NAME…… SEX/ AGE……. D.O.B……. FILE.....

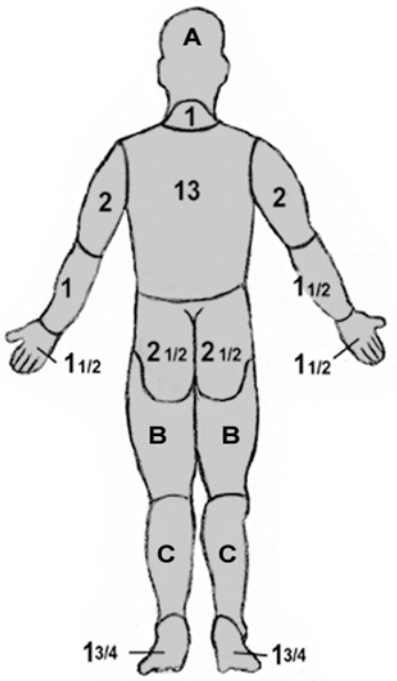
DATE OF ADMISSION …… TIME …… FIRM ……..

Firm Protocol ……

% Total body surface area burn

Be clear and accurate, and do not include erythema.





	%	
Region	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	Age 0	1	5	10	15	Adult
A = 1/2 of head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2	3 1/2
B = 1/2 of one thigh	2 3/4	3 1/4	4	4 1/2	4 1/2	4 3/4
C = 1/2 of one Lower leg	2 1/2	2 1/2	2 3/4	3	3 1/4	3 1/2

☐ Partial

☐ Deep

BURN AGENT…….. TIME OF BEING BURNT ……

1. ADMISSION DAY

Lab Number ……

Clinical appearance of the wound(s)

……

……

……

Microbiology results

……

……

……

……

2. DAY 4 POST ADMISSION

Ward admitted to ……

Lab Number ……

Clinical appearance of wound(s)

……

……

……

……

Microbiology results PLUS quantification

……

……

……

……

……

……

……

3. DAY 7 POST ADMISSION

Lab Number ……

Clinical appearance of wound(s)

……

……

……

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……

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……..

……..

Microbiology results PLUS quantification

……

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4. Microbiology results from BATH TUB cultures

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5. **Microbiology results from TAP WATER cultures**

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Patient outcome……

APPENDIX B

PATIENT INFORMATION SHEET

ASSOCIATION BETWEEN BURN WOUND BACTERIAL COLONISATION AND COMMON HYDROTHERAPY PRACTICES AT THE UNIVERSITY TEACHING HOSPITAL IN LUSAKA, ZAMBIA.

Introduction

I, Ziwa Mudaniso, a Master of Medicine (M.Med) in General Surgery student in the School of Medicine at The University of Zambia, hereby request your participation in the above mentioned research study. This study is in partial fulfillment for the award of a Master of Medicine in General Surgery. I kindly request you to carefully read this document and ask me anything you do not understand. I would like you to understand the purpose of the study and what is expected of you. Kindly remember that participation in this study is absolutely voluntary. If you agree to take part in this study, you will be asked to sign this consent form in the presence of a witness.

Aim of the study

The purpose of the study is to assess the role that the hydrotherapy practices at the University Teaching Hospital (these are using a common bath tub for cleaning wounds, using wet soaks on the burns wounds) has in predisposing to burn wound colonization by bacteria, that eventually leads to infection. It has been noted most of our patients with burns develop infection on the ward and hydrotherapy practices are being suspected as contributing factors.

Procedure of the study

If you agree to participate in this study, information will be obtained from you and entered into the burns chart. The patient (yourself or your child) will be examined to ascertain the site of the burns, depth and percentage of surface area. A swab (a cotton

wool mounted on a stick for collecting specimens from wounds) specimen will be collected from the burns on your first presentation to hospital. Follow-up swab specimens will be collected again from the burns on day 4 and day 7 of your admission. By this time you would have already been admitted to the surgical wards of the hospital.

Possible risks and discomfort

Participation in this study will not expose you to any risks. However, during collection of the swab, you will experience slight pain as the swab is being taken from the burn wound itself. Kindly note that as routine management of all burns patients, adequate pain-killer medication will be given to you.

Confidentiality

All the information collected is strictly confidential. Data that will be collected, analysed and reported on will not include your name and therefore cannot be traced to you.

Consent

Your participation is absolutely voluntary. Thus you are free to withdraw from the study at any time for any reason without any consequence to you.

I am grateful to you for considering participation in this study. For any concerns and clarifications, please contact Dr. Ziwa Mudaniso, or The University of Zambia Biomedical Research Ethics Committee on the following respective addresses:

Dr. Ziwa Mudaniso,
University Teaching Hospital,
Private Bag 1X RW,
Lusaka.
Phone +260977331625

OR

The Chairperson,

The University of Zambia Biomedical Research Ethics Committee (UNZABREC),

School of Medicine,

Ridgeway Campus – Basic Sciences building First floor,

Nationalist Road,

P.O. Box 50110,

Lusaka.

Telephone: +260-1-256067, E-mail: unzarec@unza.zm

PEPALA LA MAU KWA ODWALA

MUGWIRIZANO PAKATI PA MATENDA A PA ZILONDA ZOCHITA KUPSYA NDI KATSUKIDWE KA ZILONDAZO NDI MADZI PA UNIVERSITY TEACHING HOSPITAL MU LUSAKA, ZAMBIA.

Mau apoyamba

Ine, Ziwa Mudaniso, ophunzila maphunziro apamwamba a mankhwala mu surgery ku sukulu yama dotolo pa University of Zambia, niphempha kuti mutengeko mbali mumaphunziro ya kafukufuku yachulidwa pamwamba. Aya maphunzilo yakafukufuku nimbali yina yokwaniritsa kutsiriza kwa degree ya master mu surgery. Chonde ndipempha kuti muwerenge bwinobwino pepela iyi ndipo mundifunse mafunso pambali iriyonse pamene simunamvetse bwino. Ndizafuna kuti mumvesetse chilingo cha phunzilo yakafukufuku ndizamene ziyembekezeka kuchokela kwa inu. Mukumbukire kuti mutengako mbali muphunziro iyi mozipeleka. Ngati muvomela kutengako mbali mu phunzilo iyi, muzapemphedwa kuti musaine pepala yachilolezo pamaso pa ochitila umboni.

Chilingo cha Phuzilo

Chilingo cha phunzilo iyi ndikuti tione pa zochitika ku chipatala chachikulu mu Zambia, cha University Teaching HospitaL. Kodi kugwiritsa nchito madzi kutsuka zilonda zaochita kupsya kukhoza kulengetsa zilonda zizilowewa ndi tudoyo kuti matenda azipitilira patsogolo? Kuoneka monga odwala zilonda zochita kupsya, ambiri matenda awo amakulirapo pomwe achitidwa admit ndikutsukidwa ndi madzi. Ichi chabweretsa ganizo lakuti kapena njira iyi yosamalira zilonda izi mwina itengako mbali mukupitiriza matenda patsogolo.

Njira ya phunzilo

Ngati mubvomela kutengako mbali mu phunzilo iyi, muzalembedwa pa chipepala cha azilonda zochita kupsya. Odwala (imwe kapena mwana wanu) azapimidwa kuti mbali yathupi yakupsa, kunoka komanso kukula kwachilonda kudziwike. Kuzakhala

kugwiritsa nchito kakotoni kopombedwa kukamtengo ku pititsako pa chilonda kutengako zapachilondapo kupereka ku lab. Izi zizachitika pa tsiku loyamba (1), chinayi (4) komanso chisanu ndi chiwiri (7) kuchokela pa tsiku lomwe mwachitiwa admit. Pomwe zonse izi zichitika ninshi muli mu ward.

Zosaenela ndi zosamvetsa bwino

Kutengako mbali mu phunzilo ili sikukupatsani zosaenela zilizonse. Koma nthawi zina potengako zopereka ku lab ndikakotoni pachilonda, muzakhoza kumvelako kuwawa pang'ono. Chonde muzindikire kuti monga momwe tiyang'anira onse amatenda azilonda, muzapatsidwa mankwala oletsa kuwawa.

Kusunga chisinsi

Zonse zolembedwa za inu, zizasungidwa mwachisinsi ndithudi. Zonse zotengedwa, kulowapo kapena kufalitsidwa sizizaonetsa dzina lanu kotero kuti kulibe azakhoza kudziwa dzinu lanu.

Chilolezo

Kutengako mbali kwanu ndi mozipereka, kotelo kuti muli omasuka kulekela panjira nthawi iliyonse mukafuna kopanda kupereka chifukwa chilichonse, komanso mosalipira chilichonse. Ndili oyamika kwa inu poganizila kutengako mbali mu phunzilo ili. Ngati muli ndizodetsa nkhawa kapena mafunso ali onse, mulembele Dr Ziwa Mudaniso, kapena University of Zambia, Biomedical Research Ethics Committee pama keyala opatsidwa pansi;

Dr. Ziwa Mudaniso,
University Teaching Hospital
Private Bag 1X RW
Lusaka
Phone +260 977 331 625

KAPENA:

The Chairperson,

The University of Zambia Biomedical Research Ethics Committee (UNZABREC),

School of Medicine,

Ridgeway Campus – Basic Sciences building First floor

Nationalist Road,

P.O. Box 50110,

Lusaka.

Telephone: +260 – 1 – 256 067, E – mail: unzarec@unza.zm

ASSENT FORM (Children between 7 and 17 years old)

What is a research study?

Research studies help us learn new things. We can test new ideas. First, we ask a question. Then we try to find the answer.

This paper talks about our research and the choice that you have to take part in it. We want you to ask us any questions that you have. You can ask questions any time.

Important things to know…

- You get to decide if you want to take part.
- You can say 'No' or you can say 'Yes'.
- No one will be upset if you say 'No'.
- If you say 'Yes', you can always say 'No' later.
- You can say 'No' at any time.
- We would still take good care of you no matter what you decide.

Why are we doing this research?

We are doing this research to find out more about causes of infections in patients that have burn wounds. We have noticed that most of our patients with burn wounds develop infection in the wards. We are currently suspecting that the procedure of bathing plays a big role in the spread of infection from one patient to the other. When we have found the causes of the spread of the infection, we hope to improve our care of such patients so that the infection is minimised and the patients are better treated.

What would happen if I join this research?

If you decide to be in the research, details concerning your accident will be taken down and your wounds will be examined. I will then rub a cotton stick over your burn wound three times. The first time will be on the day you come to hospital, the second time will be after four (4) days, and the last time will be after seven (7) days. For the second and third time you will be admitted to the ward. Rubbing with a cotton stick over your wound allows me to get samples to take to the lab so that we can determine whether you have infection or not.

Could bad things happen if I join this research?

We will try to make sure that no bad things happen. Rubbing the cotton stick over your wound may sometimes feel uncomfortable and cause you to experience a little pain. However note that you will always be given pain-killers.

What else should I know about this research?

If you don't want to be in the study, you don't have to be. It is also OK to say yes and change your mind later. You can stop being in the research at any time. If you want to stop, please tell the research doctors.

Also note that there is no payment for participating in this research. You can ask questions any time.

Is there anything else?

If you want to be in the research after we talk, please write your name below. We will write our name too. This shows we talked about the research and that you want to take part.

Name of Participant_____

(To be written by child/adolescent)

Printed Name of Researcher_____

Signature of Researcher_____

PEPALA YA CHIBVOMELEZO (Ana a zaka 7 years kufika pa 17 years).

Kodi maphunziro akafukufuku ndiye chani?

Maphunziro akafukufuku atithandiza kuphunzira zatsopano. Tikhoza kuyesa nzeru zatsopano kapena zachilendo. Choyamba tifunsa funso, pambuyo pache tiyesa kupeza yankho.
Iyi pepala ikamba pa kafukufuku wathu ndi mpata muli nao kutengako mbali, kufunsa mafunso onse muli nao. Mungafunse mafunso nthawi ina ili yonse.

Zofunika kuti mudziwe ndi izi;

- Muzisankhila ngati mufuna kutengako mbali
- Mukhoza kukana olo kubvomela
- Kulibe azakalipa chifukwa chakuti mwa kana
- Ngati mwabvomera, mukhoza kukana mutsogolo
- Mukhoza kukana nthawi ina iliyonse
- Tizakusamalirani monga odwala olo mutabvomela kapena kukana kutengako mbali.

Nichani chomwe tichitira kafukufuku uyu?

Tichita kafukufuku uyu kuti tidziwe chomwe chilengetsa matenda kwa odwala ndi zilonda zochita kupsya. Taona kuti odwala zilonda zochita kupsya ambiri akhala otenga matenda ena akachitiwa admit kuma ward. Pakali pano tiona ngati njira zosambikilamo odwala zilonda izi zilengetsa kufalikira kwa matenda kuchokela odwala mmodzi kupita kwa ena. Kuti tipeze chifukwa kapena cholengetsa kufalikila kwa matenda, tikhulupilira tizakhoza kupita patsogolo ndi njila za bwino zoletsa kufalikila kwa matenda komanso ndi njila zabwino zosamalilamo odwala.

Nichani chingachitike ngati nabvomela kutengako mbali mukafukufuku aka?

Ngati mubvomela kutengako mbali mu phunzilo iyi, muzalembedwa pa chipepala cha azilonda zochita kupsya. Odwala (inu kapena mwana wanu) muzapimidwa. Kuzakhala kugwiritsa nchito ka kotoni kopombedwa ku kamtengo ku pititsako pa

chilonda kutengako zapachilondapo kupereka ku lab. Izi zizachitika pa tsiku loyamba (1) mukabwela kuchipala, tsiku lotsatapo ndi ya chinayi (4) komanso lothela la chisanu ndi chimodzi (7) kuchokela pa tsiku lomwe mwachitiwa admit. Kutengako zapachilonda ndikakotoni zizatithandiza kutengako zopeleka ku lab kuti tidziwe ngati mwatengelako matenda kapena iyayi.

Zosaenela ndi zosamvetsa bwino

Kutengako mbali mu phunzilo ili sikukupatsani zosaenela zilizonse. Koma nthawi zina potengako zopereka ku lab ndikakotoni pachilonda, muzakhoza kumvelako kuwawa pang'ono. Chonde muzindikire kuti monga momwe tiyang'anira onse amatenda azilonda, muzapatsidwa mankwala oletsa kuwawa.

Nichani china nifunika kudziwa pali phunzilo ili yakafukufuku?

Ngati simufuna kutengako mbali mu phunzilo iyi, mungatelo. Nizololeka kuti mungabvomele koma nakusintha maganizo patsogolo pake. Mungaleke kutengako mbali mu phunzilo ili pa nthawi ili yonse. Ngati mufuna kuleka, chonde muwadziwitse madotolo ali mu phunzilo ili.

Mufunika kudziwanso kuti simuzalipilidwa kanthu kalikonse kuti mutengeko mbali mu phunzilo ili, kananso mukhoza kufunsa mafunso pa nthawi ili yonse.

Kodi pali zina zapadela kapena zokuonjezela?

Ngati mufuna kutengako mbali mu phunzilo ili titakambitsana, mulembe dzina lanu pansi apa. Naife tizalemba maina athu. Ichi chisonyeza kuti takambitsana pali phunzilo ili ndipo mufuna kapena mwabvomela kutengako mbali.

Dzina la otengako mbali: _____

(Dzina lilembedwe la mwana)

Dzina la ochita kafukufuku/phunzilo: _____

Asaine ochita kafukufuku/phunzilo: _____

APPENDIX C

CONSENT FORM

I, ……. , do hereby confirm that the nature of this clinical study has been sufficiently explained to me. I am aware that my / my child's personal details will be kept confidential and I understand that I may voluntarily, at any point, withdraw my / my child's participation without suffering any consequences. I have been given sufficient time to ask questions and seek clarifications, and of my own free will declare my / my child's participation in this research. I have also received a signed copy of this agreement.

…….....	…….	……..
Name of Participant / Parent	Signature/ Thumb print	Date

……	…….	…….
Witness (Print name)	Signature/ Thumb print	Date

PEPALA LA CHILOLEZO

Ine, ……, nitsimikiza kuti ili phunzilo yamasulidwa kapena kufotokozedwa kwa ine mokwanira. Ndidziwanso kuti zodziwika za ine/ mwana wanga zapadela zizasungidwa mwa chisinsi. Ndidziwanso kuti mozifunila, pa nthawi ili yonse nikhoza kuleka/ kuleketsa mwana wanga kutengako mbali palibe chobwezela. Ndinapatsidwa nthawi yokwana kufunsa mafunso ndi kufuna kumvetsetsa, ndi mwaufulu wanga, ndibvomeleza kutengako mbali kwa ine/ mwana wanga mu phunzilo ili. Komanso nalandilako mbali ya pepala yosainidwa ya kubvomelezana uku.

…….	……...…….	……
Dzina la otengako mbali / makolo	Posaina / Chidindo cha chala	Tsiku

……	…….	……
Ochitila umboni (Dzina)	Posaina/Chidindo cha chala	Tsiku

APPENDIX D

WOUNDS, SKIN SCRAPINGS, AND OTHER MATERIALS MICROSCOPY, CULTURE AND SUSCEPTIBILITY TESTING – MICRO 09

Purpose

To isolate organisms responsible for causing skin and soft tissue infections.

Principle

The skin is colonized by many microorganism most of which exist as part of the skin normal flora. However, the tissue below the skin is normally sterile and virtually any organism that gains access to these sites can cause an infection. Such infections may occur when there is a breach in the skin barrier or may get seeded haematogenously. Infection may be due to endogenous flora or from exogenous flora. Interpretation of cultures should take into consideration Gram stain results of the cultured specimen.

*Organisms isolated from wound specimens are usually considered significant even in low numbers or mixed growth.

Specimen collection

When collecting specimens from a patient, special care must be taken to avoid contamination from the skin flora. Specimens include tissue, aspirates, pus or exudates and pus swabs. Do not send specimens collected in formalin for microorganism culture.

1. **Tissue specimens:** Are the most ideal specimens. The skin and surgical area should be disinfected with 70% alcohol before specimen collection. Aseptically aspirate the specimen and put in an appropriate sterile container.
2. **Aspirates** (e.g. from abscesses or deep tissue wounds): Are the next best specimens. Thoroughly clean the surface or wound with 70% alcohol before specimen collection. Aseptically aspirate the specimen and put in an appropriate sterile container.
3. **Pus and exudates:** Can be aspirated directly into a syringe or specimen trap (without a needle). If collected in a syringe the specimen should be transferred into a sterile container.
4. **Swab specimens:** Are the least desired because of low specimen volume and high frequency of contamination. Thoroughly clean the surface or the wound with 70% alcohol before specimen collection. Swab specimens should be inserted into transport media (e.g. Amies transport media with activated

charcoal or Stuart's transport media) after collection. Should be Pus or a swab should be collected directly into a sterile container and sent to the laboratory immediately. Transport media should be used where possible e.g. Armies transport media, Stuart, etc.

TRANSPORT

- **Tissue, aspirate, pus** and **exudate** specimens should be transported to the laboratory in plain sterile containers and should reach the laboratory within 1 hour of collection.
- **Pus swabs** should be transported in transport media, e.g. Amies transport media with activated charcoal or Stuart's transport media. Specimens should reach the laboratory within 24 h of collection.

Handling

- Use Personal Protective Equipment (PPE) such as lab coats, gloves and goggles at all times.
- Use a biosafety cabinet class II (or class III if indicated by nature of infection) when inoculating specimens and making smears.
- Avoid creating aerosols for fluid specimens.

SPECIMEN REJECTION CRITERIA

Specimen should be rejected if

- Tissue, aspirate, pus, exudates, and swab specimens older than 24h
- Specimens collected in unsterile containers
- Unlabeled specimens
- Mislabeled specimens
- Mismatched specimens
- Wrong specimen type
- Dry specimens
- Insufficient specimen

MEDIA, REAGENTS, AND SUPPLIES

Culture Media:

- Blood agar
- Chocolate
- MacConkey

- Mueller Hinton Agar
- Sabouraud's agar

Biochemical media:

- Triple sugar iron
- Lysine iron agar
- Sulphide indole motility medium
- Citrate agar
- Urea agar
- Bile aesculine
- 6.5% NaCl Tryptone soy broth

Reagents

- Physiological saline
- 3% Hydrogen peroxide
- Oxidase reagent
- Kovacs reagent

Supplies

- Antimicrobial susceptibility discs
- Glass slides
- Cover slips
- Identification discs e.g. bacitracin, optochin, etc
- Sterile containers
- Sterile swabs
- Transport media (e.g. Amies or Stuart's)

Quality control

- Use media and all reagents that have passed internal quality control (IQC) test.
- Do not use media or reagents that have expired.
- If media is prepared in-house, use within 1 week of preparation.

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APPENDIX E

TABLE 15: WHITE FIRM – 2012 AUDIT

	Jan	Feb	Mar	Apr	May	Jun		Jul	Aug		Sep	Oct		Nov	De
No. of burns admissions	7	8	4	4	5	12		5	17		11	5			9
No. of burns mortalities	1	1	1	1*	0	2		0	2		1	2			0
Sex/ Age (mortality)	F/2y	M/4y	F/2y 10m	M/73	-	F/3y	M/1 y	-	F/1 y 5m	M/ 9m	M/ 5m	M/ 1y 8m	F/1 y 4m		-
% TBSA (mortality)	7%	25%	20%	18%	-	25%	15%	-	30 %	12 %	15 %	30 %	20 %		-
No. of days post admission when infection clinically suspected (mortality)	2	2	3	0	-	-	5	-	-	5	-	3	4		-
Clinical infection/ diagnosis (mortality)	S	S	P	S	-	H	S	-	H	S	H	P	S		-
Culture result (mortality)	-	-	-	-	-	-	-	-	-	-	-	-	-		-
No. of days spent in hospital (mortality)	2	21	7	1	-	2	8	-	2	7	2	6	7		-

*****Patient came with already septic burns

KEY:

m = months
y = years
S = Sepsis
P = Pneumonia
H = Hypovolaemia

TABLE 16: GREEN FIRM – 2012 AUDIT

	Jan	Feb	Mar	Apr	May	Jun		Jul	Aug	Sep	Oct	Nov	De
No. of burns admissions		14	2	9	4	8		9		9	9	7	8
No. of burns mortalities		1	0	1	0	2		0		1	1	0	0
Sex/ Age (mortality pt)		M/2 y 6m	-	F/1y 7m	-	M/ 5y	M/2y 9m	-		M/ 33y	M/3 y	-	-
% TBSA (mortality pt)		4%	-	12%	-	30 %	28%	-		96%	34%	-	-
No. of days post admission when infection suspected clinically (mortality pt)		3	-	3	-	2	2	-		-	2	-	-
Clinical infection/ diagnosis (mortality pt)		S	-	P	-	S	P	-		SB	S	-	-
Culture result (mortality pt)		-	-	-	-	-	-	-		-	-	-	-
No. of days spent in hospital (mortality pt)		12	-	3	-	6	6	-		1	2	-	-

KEY:

m = moths
y = years
S = Sepsis
P = Pneumonia
SB = Severe Burns

TABLE 17: BLUE FIRM – 2012 AUDIT

	Jan	Feb	Mar		Apr	May	Jun	Jul	Aug				Sep	Oct	Nov	Dec
No. of burns admissions	10	9	8		7			15	9							17
No. of burns mortalities	0	0	2		1			1	4							2
Sex/ Age (mortality pt)	-	-	F/1 y4m	M/2 8y	F/ 4y 5m			M/3 y	M/8 m	M/ 2y	F/2 1y	F/1 y				M/2 y7m
% TBSA (mortality pt)	-	-	16 %	40 %	30%			30%	40 %	15 %	80 %	15 %				20%
No. of days post admission when infection suspected clinically (mortality pt)	-	-	4	4	3			2	4	4	2	2				2
Clinical infection/ diagnosis (mortality pt)	-	-	S	S	S			S	S	S	S	S				P
Culture result (mortality pt)	-	-	-	-	S*			-	-	-	-	-				-
No. of days spent in hospital (mortality pt)	-	-	6	5	10			2	17	8	14	8				13

KEY:

m = months
y = years
S = Sepsis
P = Pneumonia
SB = Severe Burns
S* = *Staphylococcus aureus* coagulase negative

TABLE 18: CONSOLIDATED TOTALS FOR THE DEPARTMENT BASED ON THE THREE FIRMS

	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEPT	OCT	NOV	DEC
Number of burns admissions	17	30	14	20	9	20	29	26	20	14	7	34
Number of burns mortalities	1	2	3	3	0	4	1	6	2	3	0	2
Cause of mortality and the number of mortalities	S=1	S=2	P=1 S=2	P=1 S=2	-	H=1 P=1 S=2	S=1	H=1 S=5	H=1 SB=1	P=1 S=2	-	P=1 SB=1

Total number of burns patients admitted = 240
Total number of mortalities = 27
Mortality rate = 11.25%

TOTALS

H – Hypovolaemia = 3 (11.1%)
P – Pneumonia = 5 (18.5%)
S – Sepsis = 17 (63.0%)
SB – Severe Burns = 2 (7.4%)

APPENDIX F

TABLE 19: MICROBIOLOGY CULTURE RESULTS FROM THE LAB – 2012

University Teaching Hospital Microbiology

All organisms

Data files=w13zmb.uth

Number of isolates = 111

Use expert interpretation rules

Specimen date=1/1/2012 : 12/31/2012

Specimen type=bs

Isolate listing summary

Number of patients by month

Code	Organism	Number of isolates	Number of patients	Unknown	c13/hc	fsw	g01	g02	g12	g21	g22	msw	vip	Other
		1	1						1					
ac-	Acinetobacter sp.	1	1					1						
cdi	Citrobacter koseri (diversus)	6	6					2	4					
cdp	Corynebacterium sp. (diphtheroids)	3	3						2			1		
eae	Enterobacter aerogenes	1	1						1					
eag	Pantoea agglomerans	4	4						4					
eco	Escherichia coli	3	3					2	1					
en-	Enterobacter sp.	5	5				1	1	2				1	
ent	Enterococcus sp.	2	2					1	1					
kl-	Klebsiella sp.	21	21		1		1	4	15					
kpn	Klebsiella pneumoniae ss. pneumoniae	8	8					4	4					
pae	Pseudomonas aeruginosa	10	10			1		3	6					
pmi	Proteus mirabilis	2	2				1		1					
pr-	Proteus sp.	1	1							1				
prv	Providencia sp.	1	1					1						
ps-	Pseudomonas sp.	9	9		1		1	3	2	1			1	
pvu	Proteus vulgaris	1	1				1							
sau	Staphylococcus aureus ss. aureus	25	24			1	1	10	11		1			
scn	Staphylococcus, coagulase negative	5	5					2	2			1		
str	Streptococcus sp.	2	2						2					