INVESTIGATION OF COMMON BACTERIA ISOLATES IN MALNOURISHED CHILDREN FIVE (5) YEARS AND BELOW ADMIITED IN TAMALE TEACHING HOSPITAL IN THE NORTHERN REGION OF GHANA

A THESIS

SUBMITTED TO THE DEPARTMENT OF CLINICAL MICROBIOLOGY, SCHOOL OF MEDICAL SCIENCES, KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY, KUMASI, GHANA, FOR THE AWARD OF MASTER OF PHILOSOPHY (MPHIL) DEGREE IN CLINICAL MICROBIOLOGY.

BY

RAABI DARKOM

(PG7956112)

(MPHIL CLINICAL MICROBIOLOGY)

DEPARTMENT OF CLINICAL MICROBIOLOGY

NOVEMBER, 2014

DECLARATION

I hereby declare that this submission is my own work towards the mphil and that, to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

Raabi Darkom Signature ..... Date.....

(Student)

Prof. E.H Frimpong Signature ..... Date.....

(Supervisor/ Head of Department)

DEDICATION

I dedicate this work to my lovely parents, Mr and Mrs Darkom,my supervisors,field supervisor**:** Dr Juventus Ziem and academic supervisor: Professor E. H. Frimpong, my brother, Magistrate Joseph Amidu and Mr Lawrence Adetunde for their love, academic and financial support and care.

ACKNOWLEDGEMENT

My first thanks goes to God for empowering me with strength, wisdom, knowledge, skills,understanding, health and also guidingand protecting me through to the completion of this work. Special thanks go to the peadiatric doctors and nurses of Tamale Teaching Hospital for their excellent support during recruitment and samples taken of participants in this study. I am grateful to the Tamale Teaching Hospital and the Department of Clinical Microbiology for giving me the platform to do my studies.

I am most grateful to Prof. E.H Frimpong my principal supervisor and Head of Department for that matter, for his advice and supervision. I thank God to have him as my surpervisor.

I also appreciate the support of Prof Juventus B. Ziem, my external supervisorfor his contribution in supervising this work through to the completion.

I am grateful to the Tamale Teaching Hospital Laboratory Staff for their contributory knowledge towards the sucessful completion of this work.

I wish to express my deepest appreciationto Gyesi Razak Issahakufor his contributions to the success of this study, and more especially for his support in analysing this data.

I am indebted to Mr Lawrence Adetunde for his contributions, support and advices throughout this study.

My sincere appreciation goes to my whole family, importantly my lovely parents who gave me all the needed support.

TABLE OF CONTENTS

CONTENT **PAGE**

LIST OF TABLES

PAGE

ABBREVIATION/ACRONYMS

AIDS ----------Acquired Immunodeficiency Syndrome

AN----------Adequate Nutrition

BSI----------Bloodstream Infection

CRP----------C-Reactive Protein

CPG----------Clinical Practice Guidline

DMSA----------Dimereaptosuccinc acid

FAO----------Food and Agriculture Organisation

GAIL----------Gastrointesteninal Associated-Lymphoid Tissue

GNB----------Gram Negative Bacilli

PCM----------Protein Calories Malnutrition

MAM----------Mild Acute Malnutrition

MOAM-------Moderate Acute Malnutrition

MUAC--------Mid Upper Arm Circumference

NNIS----------Nosocomial Infections Surveillance

NHS----------National Health Service

PAN----------Pan American Health Organisation

SAM----------Severe Acute Malnutrition

UK ----------United Kingdom

UNICEF----------United Nations Children Education Fund

USA----------United State of America

UTI----------Urinary Tract Infection

WHO----------World Health Organisation

ABSTRACT

OBJECTIVE:The research work was carried out to investigate or determine common bacteria isolates in malnourished children five (5) years and below admitted in Tamale Teaching Hospital in the northern region of Ghana, Tamale.

STUDY DESIGN: This research was a hospital based cross -sectional study.

DURATIONOF STUDY: from April 2013 to January 2014 at the Hospital.

PLACE OF STUDY**:** Malnutrition unit of the Pediatric ward of Tamale teaching hospital in the northern region of Ghana, Tamale.

MATERIAL AND METHODS**:** All children 0 to 60 months old and admitted to the malnutrition unit of the department of child health of the Tamale Teaching Hospital, Tamale. Mid Upper Arm Circumference (MUAC) ≤11.5cm and ≤ 12.5cm, All Children who Show symptom of sepsis, UTI, diarrhoea, Children who are able to produce stool and urine and Parent/guardian signs consent form were included in this study.To determine the types of bacterial infection commonly associated with malnourished children five years and below in blood, urine and stool culture reports were analyzed for bacterial isolates along with their antibiotic sensitivity pattern.

RESULTS**:** Out of this number, 29/198 representing 14.6% were positive blood cultures, children whose parents did not sign consent form for their blood sample to be taken recorded 2/200 representing 1%.Out of total 29 cases of culture proven bacteraemia, the isolates were *Staphylococcus* (34.5%), *Streptococcus* (6.9%) *Klebsiella* (27.6%), *E.coli* ((10.3%), *Pseudonomas* (10.3%) and *Proteus (*10.3%).. Regarding antibiotic resistance pattern; E.coli was 100% resistant to ampicillin and gentamicin, 66.7% to ciprofloxacin ,87.5% of klebsiella was resistant to cotrimoxazole, ciprofloxacin and amikacin, and 50% to levofloxacin, staphylococcus aureus was 80% resistant to ampicillin and cotrimoxazole, 90% to gentamicin and 10% cephalexin.

49/180 representing 27.2% of positive urine cultures and children who were not able to produce urine sample recorded 20/200 representing 10%. Out of total 49 cases of culture proven UTI, the isolates were as follow; klebsiella(38.8%), proteus(16.3%),pseudomonas(16.3%),E.coli(10.2%), citrobacter(8.2%), S.aureus(6.1%), enterobacter(2.0%) and enterococcus (2.0%).88% klebsiella was resistant to gentamicin, 70% to levofloxacin 30% to norvofloxacin, 21.1% to cotrimoxazole and 10.5% to ampicillin. Proteus was 100% resistant to gentamicin, 75% to cephalexin, 62.5% to levofloxacin, and 12.5% to ampicillin, cotrimoxazole and tetracycline.

3/199 representing 1.5% of positive stool cultures, children who could not produce stool sample were 1/200(0.5%), whislt169/198 (85.4%), 131/180 (72.8%) and 196 /199 (98.5%) were tested negative for blood, urine and stool respectively. 100% *Shigella* while was resistant to gentamicin, Amikacin and Levofoxacin, and 33% to cotrimoxazole and ampicillin.

From the controlled analysis (nourished children), 22/198 representing 11.1% positive blood cultures, 13/112 representing 11.6% positive urine cultures and 1/71 representing 1.4% positive stool cultures. Whilst 176/198 (88.9%), 99/112 (88.4%), 70/71 (98.6) were tested negative for blood, urine and stool respectively.The age group 12-23 months showed a significant statistical difference($p < 0.05$) among Severe Acute Malnutrition (SAM), Moderate Acute Malnutrition (MOAM), Mild Acute Malnutrition (MAM) and Adequate Nutrition. Severe acute malnutrition (SAM) was seen to be higher among age group zero (0)-five years old children admitted to the Tamale Teaching Hospital. Males were more susceptible to bacterial infection than females.In conclusion, there is a significantly higher prevalence of bacteial infection in the malnourished children.

CHAPTER ONE

1.1 INTRODUCTION AND BACKGROUND

According to Medilexicon's medical dictionary, Malnutritionis "faulty nutrition resulting from malabsorption, poor diet, or overeating."Under nutrition is "a form of malnutrition resulting from a reduced supply of food or from inability to digest, assimilate, and use the necessary nutrients." According to the World Health Organization (WHO), malnutrition is the gravest single threat to global public health.Malnutrition remains one of the most common causes of morbidity and mortality among children throughout the world and more commonly in sub-Saharan Africa and South Asia. Acute childhood malnutrition affects about a tenth of the world's children under 5 years of age, and contributes to 50–60% of all child deaths (Black *et al*.,2003) particularly those living in circumstances of extreme poverty in the developing world (Black *et al*.,2008). In Ethiopia, more than one in two children (52%) under the age of five are stunted (growth retardation), 11% are wasted (thin for their height) and 47% are underweight (low weight-for-age). Stunting and wasting rates are even higher in rural children (CSA, 2005), where the vast majority of the population is dwelling. Other community based studies in Ethiopia also showed prevalence of wasting from 9–12% (Getaneh *et al*., 1998). Poor infant and young child feeding practice, poor socio-economic background and nutritionally inadequate diet contribute more for severe acute malnutrition (Amsalu and Tigabu, 2008).

Severe acute malnutrition (SAM) affects both acquired and innate host defense mechanisms (Schaible and Kaufmann, 2007). This leads to increased susceptibility to infection, more frequent and prolonged episodes, increased severity of disease (CD-WGE, 2005), reactivation of viral infections, and development of opportunistic infections (Cunninghan-Rundles *et al*.,2005). In

addition, severe acute malnutrition often masks symptoms and signs of infectious diseases making prompt clinical diagnosis and early treatment very difficult. This in turn, increases the morbidity and mortality from communicable diseases (CD-WGE, 2005). However, the contribution of bacteraemia to the morbidity and mortality among severely malnourished children is poorly investigated.

The World Health Organization (WHO) says that malnutrition is by far the largest contributor to child mortality globally, currently present in half of all cases. Underweight births and inter-uterine growth restrictions are responsible for about 2.2 million child deaths annually in the world. Deficiencies in vitamin A or zinc cause 1 million deaths each year. WHO adds that malnutrition during childhood usually results in worse health and lower educational achievements during adulthood. Malnourished children tend to become adults who have smaller babies.While malnutrition used to be seen as something which complicated such diseases as measles, pneumonia and diarrhoea, it often works the other way round - malnutrition can cause diseases to occur (WHO,2013).

In poor, developing nation's malnutrition is commonly caused by:

- Food shortages - in poor developing nation's food shortages are mainly caused by a lack of technology needed for higher yields found in modern agriculture, such as nitrogen fertilizers, pesticides and irrigation. Food shortages are a significant cause of malnutrition in many parts of the world (NHS, 2013).
- Food prices and food distribution; it is ironic that approximately 80% of malnourished children live in developing nations that actually produce food surpluses (Food and

Agriculture Organization). Some leading economists say that famine is closely linked to high food prices and problems with food distribution. (NHS, 2013).

- Lack of breastfeeding– expertssays that lack of breastfeeding, especially in the developing world, leads to malnutrition in infants and children. In some parts of the world, mothers still believe that bottle feeding is better for the child. Another reason for lack of breastfeeding, mainly in the developing world, is that mothers abandon it because they do not know how to get their baby to latch on properly, or suffer pain and discomfort. (NHS. 2013).

1.2 JUSTIFICATION

Numerous studies in developing countries have shown that weaning foods prepared under unhygienic conditions are heavily contaminated with pathogenic agents and are a major risk factor in the transmission of diseases, especially diarrhoea. It is generally recognized that contamination of complementary foods may occur as a result of poor hygiene of food handlers, household equipments and the environment where the preparation of food takes place (Sheth *et al*., 2000).

Not much is known about bacterial infection in malnourished children in Northern Ghana. This study will serve as baseline for future projects, and inform stake holders on policy formation. It will also improve on the treatment guidelines for the malnourished.

1.3 STATEMENT OF OBJECTIVES

Infection adversely affects nutritional states through reductions in dietary intake and intestinal absorption, increased catabolism sequestration of nutrients that are required for tissue synthesis and growth (Mondel *et al*., 2009).

Acute diarrhoea and pneumonia occur most frequently during the first 2–3 years of life when immunocompetence is impaired and when children are first being exposed to pathogens. Infection can suppress appetite and directly affect nutrient metabolism, leading to poor nutrient utilization (Bloss *et al*., 2004).

Infection itself can cause a loss of critical body stores of protein, energy, minerals and vitamins. During an immune response, energy expenditure increases at the same time that the infected host experiences a decrease in nutrient intake (Cunning- Rundles *et al*., 2005). During an infection, a negative nitrogen balance occurs after fever induction and then it increases and persists for days to weeks after the febrile phase. Additionally, negative nitrogen balance appears to correlate with net loss in body weight; both conditions are the result of reduced food intake and infection induced-increased nitrogen excretion (Philips *et al*., 2004).

Malnourished children suffer in greater proportion from bacterial gastrointestinal and respiratory infections (De Onis *et al*., 1993).

1.4 RESEARCH QUESTIONS

1. What are the etiological agents and antibiotic sensitivity patterns of urinary tract infections among malnourished children admitted at the Tamale Teaching Hospital?
2. What are the etiological agents and antibiotic sensitivity patterns of bacteraemia among malnourished children admitted at the Tamale Teaching Hospital?
3. What are the etiological agents and antibiotic sensitivity patterns of gastroenteritis among malnourished children admitted at the Tamale Teaching Hospital?

1.5 GENERAL OBJECTIVE

The overall aim of this study is to determine the types of bacterial infection commonly associated with malnourished children five years and below in the Tamale Teaching Hospital of Ghana.

1.6 SPECIFIC OBJECTIVES

1. To determine the etiological agents and antibiotic sensitivity patterns of urinary tract infections among malnourished children admitted at the Tamale Teaching Hospital.
2. To determine the etiological agents and antibiotic sensitivity patterns of bacteraemia among malnourished children admitted at the Tamale Teaching Hospital.
3. To determine the etiological agents and antibiotic sensitivity patterns of gastric enteritis among malnourished children admitted at the Tamale Teaching Hospital.

CHAPTER TWO

2.0LITERATURE REVIEW

Each year about 13 million infants and children die in the developing countries (Scrimshaw *et al*., 1968). The majority of these deaths are due to infections and parasitic diseases, and many if not most of the children die malnourished. The precise contribution of malnutrition as an immediate cause of death is not known, nor would it be the only relevant figure, for in poor countries children from birth or soon after are caught in a cycle of malnutrition and infection, from which many do not survive (Scrimshaw *et al*., 1957). In Africa, for example, more than 20% on averagedo not reach their fifth birthday (Beck *et al*., 1957). The "malnutrition-infection" complex remains the most prevalent public health problem in the world today. Nutrition and health are closely linked, but advances in nutritional knowledge remain to be applied to the same extent as those in the field of health.

It is more than twenty years since the landmark publication by (Scrimshaw *et* al., 1968;) on "Interactions of Nutrition and Infection", knowledge of this subject has become well-established.The mechanisms of many of these interactions have been elucidated, and the relative importance of such interactions in different circumstances has been clarified.

Infection itself can cause a loss of critical body stores of protein, energy, minerals and vitamins. During an immune response, energy expenditure increases at the same time that the infected host experiences a decrease in nutrient intake (Cunning- Rundles *et al*., 2005). During an infection, a negative nitrogen balance occurs after fever induction and then it increases and persists for days to weeks after the febrile phase. Additionally, negative nitrogen balance appears to correlate with

net loss in body weight; both conditions are the result of reduced food intake and infection induced-increased nitrogen excretion (Powander and Beisel, 2003).

In developing countries, about 3.5% of children under the age of 5 years suffer from severe malnutrition. Although mild and moderate types of childhood malnutrition are even more prevalent, their significance in childhood morbidity and mortality is less well recognized (Ahmed *et al*., 2009).

In children under 5 years of age, malnutrition is responsible, directly or indirectly, for 54% of the 10.8 million deaths per year and contributes to every second death (53%) associated with infectious disease among this age group in developing countries (Benguigui and Stein, 2006). Additionally, mild and moderate forms of malnutrition primarily account for the burden of malnutrition worldwide. For the surviving children, malnutrition has lifelong implications because it severely reduces a child's ability to learn and grow to their full potential. Thus, malnutrition leads to less productive adults and weaker national economic performance (Benguigui and Stein, 2006).

Malnourished children suffer in greater proportion from bacterial gastrointestinal and respiratory infections (De Onis *et al*., 1993).

Chronic malnutrition has been and still remains a persistent problem for young children in sub-Saharan Africa (FAO, 2008). A high percentage of these children fail to reach the normal international standard height for their age often associated with stunted growth. Moreover, the number of undernourished children in sub-Saharan Africa continues to increase and the region has shown little improvement over the past decades (FAO, 2008).

Majority of the infants are introduced to cereal-based complementary foods well before the recommended 6 months age for the introduction of 'safe and nutritionally adequate' complementary foods or in rare instances do not receive these until the second year (Onyango, 2003).

Poor breastfeeding and complementary feeding practices, coupled with high rates of infectious diseases, have been reported as the principal proximate cause of malnutrition during the first-two years postnatal period. The world health organization (WHO) recommends that infants should be exclusively breastfeed for the first 6 months of life to achieve optimal growth, development and health. Thereafter, to meet their evolving nutritional requirements, infants should receive nutritionally adequate and safe complementary foods with breastfeeding continuing for up to two years of age and beyond (WHO/UNICEF, 2003). Exclusive breastfeeding is associated with multiple advantages including child's acquisition of passive immunity against infection, nutrients for physical and mental development, emotional security and closeness to the mother. Breastfeeding drastically reduces deaths from acute respiratory infections and diarrhoea which are the two major causes of infant mortality worldwide and in addition to protection from other infectious diseases.

Despite these advantages, only 39% of all infants aged 0 to 5 months in the developing world are exclusively breastfed while < 60% of those aged 6 to 9 months continues to be breastfed while receiving complementary foods (UNICEF, 2009).

2.1 BACTERAEMIA AMONG MALNOURISHED CHILDREN

Malnutrition remains one of the most common causes of morbidity and mortality among children throughout the world (WHO, 1999) and more commonly in sub-Saharan Africa and south Asia (Manary and Sandige, 2008). Acute childhood malnutrition affects about a tenth of the world's children under 5 years of age (Manary and Sandige, 2008), and contributes to 50–60% of all child deaths (Black *et al*., 2003) particularly those living in circumstances of extreme poverty in the developing world (Black *et al*., 2008). In Ethiopia, more than one in two children (52%) under the age of five are stunted (growth retardation), 11% are wasted (thin for their height) and 47% are underweight (low weight-for-age). Stunting and wasting rates are even higher in rural children (CSA, 2005), where the vast majority of the population is dwelling. Other community based studies in Ethiopia also showed prevalence of wasting from 9-12% (Yimer, 2000). Poor infant and young child feeding practice, poor socio-economic background and nutritionally inadequate diet contribute more for severe acute malnutrition (Amsalu and Tigabu, 2008).

Severe acute malnutrition (SAM) affects both acquired and innate host defense mechanisms (Schaibe and Kaufmann, 2007). This leads to increased susceptibility to infection, more frequent and prolonged episodes, increased severity of disease (CD-WGE, 2005), reactivation of viral infections, and development of opportunistic infections (Cunningham-Rundles *et al*., 2005). In addition, severe acute malnutrition often masks symptoms and signs of infectious diseases making prompt clinical diagnosis and early treatment very difficult. This in turn, increases the morbidity and mortality from communicable diseases (CD-WGE, 2005). However, the contribution of bacteraemia to the morbidity and mortality among severely malnourished children is poorly investigated. Bacteraemia can be caused by Coagulase negative *Staphylococci*species, *S. aureus* and *Enterobacteriaceae* (Bearmann and Wenzel, 2005).

Different studies in different countries demonstrated prevalence of bacteraemia in severely malnourished children from 11.8%-36% (Bachou *et al*., 2006). The National Treatment Protocol for SAM recommends routine use of antibiotics for all children with SAM (FMoH, Ethiopia, 2007). Therefore, it is vital to regularly audit antibiotic sensitivity and the magnitude of bacteremia as the pattern could change due to various factors.

The objective of this study was to establish the magnitude of bacteraemia in severely malnourished children, and describe the types of bacteria and antimicrobial sensitivity.

A significant mortality in neonatal period occurs due tobacteremia. Neonatal infections are estimated to cause 1.6 million of all neonatal deaths occurring in developing countries. Gram negative organisms remain major cause of neonatal septicemia (Akhtar *et al*., 2005). In Nigeria, bacteraemia is a major cause of death in neonates and children (Asindi *et al*., 1991). The outcome of treatment of neonates with bacteraemia has remained poor in Nigeria as shown by reports of mortality rate of 33% to 41% from two tertiary hospitals in the country (Adejuyigbe *et al*., 2001).

Bacteremia are potentially life-threatening andrequire rapid identification and also antibiotic susceptibility testing of the causative agent in order to facilitate specific antimicrobial therapy (Berit *et al*., 2006). Despite advances in anti- microbial therapy and supportive care, bacteraemia continues to be a major cause of morbidity and mortality among children. In developing countries, more than 14 million deaths of children below five years of age (UNICEF, 1994) occur during the childhood, with infections accounting for up to 70% of total mortality for this age group (Mylotte and Tayara, 2001). Around the world is estimated 10 million children under theage of 5 years die each year, the vast majority (90%) in a mere42 countries. Of the major

causes of death among children, are infections such as newborn sepsis (Jones *et al*., 2003). The organisms responsible for bacteraemia vary across geographical boundaries. Organisms like *E. coli, Klebsiella spp*., *Staphylococcus aureus,* Coagulase negative *Staphylococci* (CoNS), *Pseudomonasspp*., *Salmonella spp* and *Acinetobacter spp* are potential pathogens in bacteraemia because of their frequent isolation and multi-drug resistance which has reached worrying levels (Castagnola *et al*., 2005).

In the USA alone, 10–20% of nosocomial infections are estimated to involve the bacteraemia, resulting in 90 000 fatal cases each year. Appropriate antimicrobial treatment of bacteraemiainfections is critical in decreasing morbidity and mortality due tobacteria infection (American Society for Microbiology Reports, 1995).

2.1.1 *STAPHYLOCOCCUS AUREUS*

Staphylococcus species are gram-positive spherical cells, usually arranged in grapelike irregular clusters. The grow readily on many types of mediaand are active metabolically, fermenting carbohydrates and producing pigments that vary from white to deep yellow.Some are members of the normal flora of the skin and muccus membranes of humans ;others cause suppuration ,abscess formation, pyogenic infection and septicamia.The genes *Staphylococcus* has at least 40 species (Jawetz et al ., 2010).

*Staphylococcus aureus*is a major pathogen for humans, ranging from in severity from food poisoning or minor skin infections to severe life-threatening infection.The pathogenic *Staphylococcus* often hemolyze blood, coagulase plasma and produce variety of extracellular enzymes toxin. Staphylococci are nonmotile and donot form spores.Colonies on solid media are

round, smooth, raise and glisteming. Staphylococcus aureus forms gray to deep golden yellow colonies (Jawetz et al., 2010).

Staphylococcus aureus is a major human pathogen that produces a wide array of toxins, thus causing various types of disease symptoms. Staphylococcal enterotoxins (SEs), a family of nine major serological types of heat stable enterotoxins, are a leading cause of gastroenteritis resulting from consumption of contaminated food. In addition, SEs are powerful super antigens that stimulate non-specific T-cell proliferation. SEs share close phylogenetic relationships, with similar structures and activities. Here we review the structure and function of each known enterotoxin (Balaban and Rasooly. 2000).

2.1.2 *SHIGELLA*

Shigellae are nonmotile and usually do not ferment lactose but do ferment other carbohydrates, producing acid but not gas. They do not produce H_2S. The four *Shigella* species are closely related to *E.coli*. Many share common antigens with one another and with other enteric bacteria. Examples are *Hafnia alvei* and *Plesiomonas Shigelloides.*They are cause by faecalcontamination of food and water (Hansen and Yourassowsky; 1984).

2.1.3 *SALMONELLA*

Salmonellae are motile rods that ferment glucose and manose without producing gas but do not ferment lactose or sucrose. Most Salmonellae produces H_2S. Theyare often pathogenic for humans and animals when ingested. Their mode or means transmission is through faecal contamination of water and food (Shelobolina et al., 2004).

2.1.4 *VIBRO CHOLERAE*

Cholera is caused by *Vibrio cholerae,* is a disease which is passed on through contaminated water. The etiological agent is a short, curved, Gram-negative rod having a single polar flagellum. Its exotoxin binds to host cells, and the host epithelial cells secrete large quantities of chloride into the intestinal lumen followed by large amounts of water and sodium and other electrolytes. This disease makes an individual suffer from diarrhoea and vomiting and some times lead to death. Cholera can be managed by fluid intake in order to regain the water loss in the body (Peterson. 2002).

2.1.5 *ESCHERICHIA COLI*

*Escherichiacoli*is the Gram-negative rod which is usually harmless. However, certain strains produce toxins or have the capability of invading tissue, and these strains can cause infections in humans.The newborndiarrhoeaandurinary tractinfections are caused by *E. coli* (Miller el al., 1989).Enteropathogenic E.coli (EPEC)for example attaches to the mucosal epithelial cells and produces cytoskeletal changes; may invade cells. Different from othor E.coli that are enteroaggregative (also adhered or stick to human cells and produce ST-like toxin and hemolysin) and cause chronic diarrhoea.*Escherichiacoli*is afood borne disease and can be manage using antimicrobial therapy (Donnenberg el al., 1989).

2.1.6 CLOSTRIDIUM PERFRINGENS

Clostridial foodpoisoning is caused by *Clostridium perfringens*, a spore forming, and anaerobic rod. This organism when found in meat produces its toxin and eating of infected or impure meat may leads to gastroenteritis.Antibiotics are used to treat this infection.*Clostridium botulinum has*

a toxin which affects the nervous system. *Clostridium botulinumdisease* also spread or passed on throughcontaminated food (Tewari and Juneja. 2008).

2.1.7 *LEPTOSPIRA INTERROGANS*

Leptospiros is transmitted by*Leptospira interrogans*, a spirochete. Is is a disease which affects or caused harm to the liver and kidney of both dogs and human beings. Human beings become infected by contact with urine of the infected dogs as the spirochete enters abrasions in the skin. The symptoms are muscle aches, fever, and liverabnormalities (Adler and Peña, 2010).

2.1.8 *HELICOBACTER PYLORI*

Helicobacter pylori is a spiral -shaped gram-negativerod.It is associated with antral gastritis,duodenal (peptic) ulcer disease,gastric ulcers and carcinoma.It has a multiple flagella at one pole and is actively motile.The colonies are transluscent and 1.2 mm in diameter.Helicobacter pylori is oxidase -positive and catalase -positive and is a strong producer of urease (Xiang et al., 1995).

Helicobacter pylori is a causative agent of disease states of varying severity including chronic gastritis, or gastric adenocarcinoma (Faruta *et al*., 2002). *H. pylori* infection is strongly associated with other gastrointestinal infections and chronic malnutrition. *H. pylori* infection occurs primarily in early childhood, and in developing countries it has a severe impact on general health (Windle *et al*., 2007). In children, *H. pylori* infection can be the initiator of a vicious cycle of events that leads to malnutrition and growth retardation in children that impacts both morbidity and mortality (Windle *et al*., 2007).

2.1.9 CAMPYLOBACTER

In the developed world *Campylobacter jejuni* is the primary cause of bacterial gastroenteritis, with half of these cases associated with exposure to poultry (Gelanis, 2007). In children, bacteria are the cause in about 15% of cases, with the most common types being *Escherichia coli*, *Salmonella*, *Shigella*, and*Campylobacter* species (Webb and Starr, 2005). If food becomes contaminated with bacteria and remains at room temperature for a period of several hours, the bacteria multiply and increase the risk of infection in those who consume the food (Mandell, 2010). Some foods commonly associated with illness include raw or undercooked meat, poultry, sea food, and eggs; raw sprouts; unpasteurized milk and soft cheeses; and fruit and vegetable juices (Nyachuba, 2010). In the developing world, especially sub-Saharan Africa and Asia, cholera is a common cause of gastroenteritis. This infection is usually transmitted by contaminated water or food (Charles and Ryan, 2011).

2.2 GASTROENTERITIS

Gastroenteritis or infectious diarrhoea is a medical condition characterized by inflammation ("-*itis*") of the gastrointestinal tract that involves both the stomach (*"gastro"*-) and the small intestine (*"entero"*-), resulting in some combination of diarrhoea, vomiting, and abdominal pain and cramping (Amandeep, 2010). Dehydration may occur as a result. Gastroenteritis has been referred to as gastro, stomach bug, and stomach virus, although unrelated to influenza, it has also been called stomach flu and gastric flu.

Diarrhoea is the passage of unusually loose or watery stools, usually at least three times within 24 hour period. However, it is the consistency of the stools rather than the number that is most important. Frequent passing of formed stools is not diarrhoea. Babies fed with only breast milk

often pass loose pasty stools; this also is not diarrhoea. Prolonged diarrhoea may lead to excessive loss of fluid, salt and nutrient in the faeces. The main cause of death from acute diarrhoea is dehydration, which result from loss of fluid and electrolyte in stool. Another important cause of death is dysentery and under nutrition. Diarrhoea is an important cause of under nutrition because patients eat less during diarrhoea and their ability to absorb nutrients is reduced. Moreover, nutrient requirement is increased as a result of infection (Sinclair *et al*., 2003).

Risk factors that predispose children to diarrhoea include poor sanitation, poor social and economic status and malnutrition (Andu *et al*., 2002). Vomiting may occur and fever may be present. The most important cause of acute watery diarrhoea in young children in Nigeria include Rotavirus, enterotoxigenic Escherichia coli, Shigella, Campylobacter jejuni, and Cryptosporidia, Vibrio cholerae, Salmonella and enteropathogenic Escherichia coli (Bahal *et al*., 2001)

2.2.1 Bacteria causing Gastrointestinal Infections associated with Malnutrition

Protein calories malnutrition (PCM) and gastrointestinal bacterial infections frequently coexist in humans living in developing countries. It is estimated that more than 10 million children under 5 years of age die each year worldwide (Black *et al*., 2003). More than two million children die each year in developing countries from diarrhoeal diseases. Infection adversely affects nutritional status through reductions in dietary intake and intestinal absorption, increased catabolism and sequestration of nutrients that are required for tissue synthesis and growth (Mondal *et al*., 2009). Of 3 million premature deaths due to diarrhoeal diseases, approximately 58% are associated with malnutrition (Wapir, 2000).

In a recent descriptive and prospective study, 335 children under 6 years of age that were admitted to a hospital in Colombia for severe acute malnutrition (83%) or moderate acute malnutrition associated with illness (17%). The most common complication upon admission was diarrhoea (68.4%) and the most common complication during hospitalization was sepsis (9%). Children with moderate acute malnutrition had similar complications and mortality when compared to children with severe acute malnutrition (Bernal *et al*., 2008).

A broad group of microorganisms cause diarrhoea in children making identification of the etiologic agent difficult .Bacterial enteric pathogens that cause most cases of severe acute diarrhoea include *Vibrio cholerae*, *Shigella* spp., *Salmonella* spp., enteropathogenic *Escherichiacoli* (EPEC), enteroaggregative *E.coli* (EAEC), enterotoxigenic *E.coli* (ETEC) and *Cryptosporidium* spp. (Qadri *et al*., 2007). Furthermore, intestinal helminth infections may also impair intestinal function, absorption and growth (Muniz *et al*., 2002).

2.2.2 Virusesin Diarrhoea

Rotavirus, norovirus, adenovirus, and astrovirus are known to cause viral gastvroenteritis. (Eckardt and Baumgart, 2011) Rotavirus is the most common cause of gastroenteritis in children, (Szajewska and Dziechciarz, 2010) and produces similar incidence rates in both the developed and developing world (Meloni *et al*., 2011).Viruses cause about 70% of episodes of infectious diarrhoea in the pediatric age group (Webb and Starr, 2005). Rotavirus is a less common cause in adults due to acquired immunity (Desselberger and Huppertz, 2011).

Norovirus is the leading cause of gastroenteritis among adults in America, causing greater than 90% of outbreaks (Eckardt and Baumgart, 2011). These localized epidemics typically occur

when groups of people spend time in close physical proximity to each other, such as on cruise ships, (Eckardt and Baumgart, 2011) in hospitals, or in restaurants (Amandeep, 2010). People may remain infectious even after their diarrhoea has ended. (Eckardt and Baumgart, 2011) Norovirus is the cause of about 10% of cases in children. (Amandeep, 2010)

2.2.3 Parasitic

A number of protozoans can cause gastroenteritis – most commonly *Giardia lamblia* but *Entamoeba histolytica* and *Cryptosporidium* species have also been implicated.(Webband Starr, 2005). As a group, these agents comprise about 10% of cases in children (Elliot, 2007).*Giardia* occurs more commonly in the developing world, but this etiologic agent causes this type of illness to some degree nearly everywhere (Escobedo *et al*., 2010). It occurs more commonly in persons who have traveled to areas with high prevalence, children who attend day care, men who have sex with men, and following disasters (Escobedo et al., 2010).

2.2.4 Non-infective

There are a number of non-infectious causes of inflammation of the gastrointestinal tract (Amandeep, 2010). Some of the more common include medications (like NSAIDs), certain foods such as lactose (in those who are intolerant), and gluten (in those with celiac disease). Crohn's disease is also a non-infection source of (often severe) gastroenteritis (Amandeep, 2010). Disease secondary to toxins may also occur. Some food related conditions associated with nausea, vomiting, and diarrhoea include: ciguatera poisoning due to consumption of contaminated predatory fish, scombroid associated with the consumption of certain types of spoiled fish, tetrodotoxin poisoning from the consumption of puffer fish among others, and botulism typically due to improperly preserved food (Lawrence *et al*., 2007).

2.3 TREATMENT OF DIARRHOEA

The main solution todiarrhoea is enough fluids andoral rehydration solution (a combination of water, salts, and sugar). The intravenous fluids from a healthcare centre are also helpful.

2.3.1 Dietary

It is recommended that breast-fed infants continue to be nursed in the usual fashion, and that formula-fed infants continue their formula immediately after rehydration with ORT (Hempel *et al*., 2012). Lactose-free or lactose-reduced formulas usually are not necessary (Hempel *et al*., 2012). Children should continue their usual diet during episodes of diarrhoea with the exception that foods high in simple sugars should be avoided (Hempel *et al*., 2012). Some probiotics have been shown to be beneficial in reducing both the duration of illness and the frequency of stools (Mackway-Jones and Kevin, 2007). They may also be useful in preventing and treating antibiotic associated diarrhoea (Telmesani, 2010). Fermented milk products (such as yogurt) are similarly beneficial (Decamp *et al*., 2008). Zinc supplementation appears to be effective in both treating and preventing diarrhoea among children in the developing world (Mehta and Goldman, 2006).

2.4 BACTERIA CAUSING RESPIRATORY INFECTIONS ASSOCIATED WITH MALNUTRITION

A strong and consistent association has been demonstrated between malnutrition and mortality from respiratory infections; further, malnutrition is considered to be a more important risk factor for pneumonia than for diarrhoea (Berkowitz, 1992). Acute respiratory infections (ARIs) are the leading cause for high mortality and morbidity among children under 5 years of age (Graham, 1990); they are also the most frequent cause of health services used around the world. ARIs represent between 30–50% of pediatric medical consultations and between 20–40% of

hospitalizations in children. The risk factors for acquiring respiratory infections are poverty, restricted family income, low parental education level, lack of breastfeeding and, most importantly, malnutrition (Cashat- Cruz *et al*., 2005).

The establishment of malnutrition depends on the cause and duration of any nutritional deficiency. It can be caused, secondarily, by increase in demand of nutrients (Gomez *et al*., 1956).

The infection may be either aggravating a previously existing deficient nutritional status or triggering malnutrition through disease pathogenesis (Borolli *et al*., 2004). It has been demonstrated that certain infectious diseases cause malnutrition. These diseases cause a reduction in food intake. One example of how respiratory infections can contribute to malnutrition is that chronic infections may be cause cachexia (Melendez *et al*., 2011). The respiratory infections, as pneumonia, occur most frequently during the first 24–36 months of life when immunocompetence is impaired and when children are first being exposed to pathogens. The stimulation of an immune response by respiratory infection increases the demand for metabolically derived anabolic energy, this lead to adverse nutritional status. Moreover, a respiratory infection itself can cause a loss of critical body stores of protein and energy. Additionally, negative nitrogen balance appears to correlate with net loss in body weight; this result in reduced food intake and infection induced-increased nitrogen excretion (Powanda and Beseil, 2003). During an infection, a negative nitrogen balance occurs after fever induction and then it increases and persists for days to weeks after the febrile phase. Therefore, the malnutrition may be a consequence of repeated respiratory infections, common in young children (Cunha, 2000).

2.4.1 *Streptococcus Pneumoniae*

The*Streptococcus pneumoniae*are gram-positive diplococci, often lancet -shaped or arranged in chains, possessing a capsule of polysaccharide that permits typing with specific antisera. *Pneumococc*i are readily lysed by surface-active agent, which probably remove or inactivate the inhibitors of the cell wall autolysins.*Pneumococc*i are normal inhibitants of the upper respiratory tract of 5-40% of humans and can cause pneumonia, sinusitis, otitis, bronchitis, bacteraemia,meningitis, and other infectious processes.*Pneumococc*i form small round colonies, at first dome-shaped and later developing a central plateus with elevated rim (Jawetz et al., 2010).

Streptococcus pneumoniae is a leading cause of bacterial pneumonia, meningitis, and sepsis in children worldwide. Pneumococcal disease is preceded by asymptomatic nasopharyngeal colonization, which is especially high in children. The natural route of infection with *S. pneumoniae* starts with colonization, which may progress to invasive disease if immunological barriers are crossed (Bogaert *et al*., 2004).

Childhood clinical pneumonia is caused by a combination of risk factors related to the host, the environment and infectious agent (Rudan *et al*., 2008). In developing countries, identifying the etiology is difficult, and WHO recommends diagnosing pneumonia based on clinical parameters. However, based on available evidence, several studies have identified *Streptococcus pneumoniae* and *Haemophilus influenzae* as the most important pathogens associated with childhood pneumonia (Berman, 1991). Further, *Staphylococcus aureus* and *Klebsiella pneumoniae* have also been linked to cases of severe pneumonia (Shann, 1986). In microbiologic studies, *Streptococcus pneumoniae* has been identified in 30–50% of pneumonia cases and *H. influenzae*

type b in 10–30% of cases. *S. aureus* and *K. pneumoniae* were the next most prevalent etiologic agents of pneumonia (Rudan *et al*., 2008). However, with the increased use of pneumococcal and *H. influenzae* type b vaccines in developing countries, it is likely that these pathogens will become relatively less important as causative agents of pneumonia (Chisti *et al*., 2009)

2.4.2 *Haemophilus Influenzae*

Haemophilus influenzae type b (Hib) is mostly an opportunistic pathogen that causes invasive infections, such as pneumonia in children under 5 years of age. The incidence of Hib pneumonia and Hib invasive disease in children younger than the age of 5 years in developing countries is 7 and 21–60 per 100,000 per year, respectively. (Rudan *et al*., 2008) reported that in developing countries with a high burden of pneumonia, 15–30% of radiological pneumonia cases, and most likely the same proportion of pneumonia deaths, is due to Hib.

*Streptococcus pneumoniae*and *Haemophilus influenzae*were the two microorganisms isolated most frequently from the blood, lung or pleural fluid from well-nourished (33%) and malnourished children (11%) with pneumonia. However, according to (Chisti *et al*., 2009) *Klebsiella* spp and *S.aureus* were the most common causative organisms in severely malnourished children. These findings suggest that *Klebsiella* species and *S. aureus* are probably the main bacterial causes of pneumonia in malnourished children. Additionally, pathogenic viruses have been isolated from malnourished children with pneumonia. Although *Mycobacterium tuberculosis* was detected in 18% of malnourished children with pneumonia (Adegbola *et al*., 1994), the role of *Mycobacterium tuberculosis* presenting as an acute lower respiratory infection in severely malnourished children has not been well studied.

Furthermore, studies have demonstrated that pneumonia is more common among children with marasmic-kwashiorkor than among other types of malnourishment (Adegbola *et al*., 1994). Additionally, in children under the age of 2 years, malnutrition is associated with a significant increase in ARI morbidity; also, severe pneumonia is associated to increase the mortality rate (Nantanda *et al*., 2008). In a study performed with severely malnourished children, the mortality in children with Kwashiorkor was 13.4%. Mortality was 28% in children with marasmus and 48.3% in children with unclassified malnutrition. The main causes of death in children younger than 18 months of age were dehydration and pneumonia; in children from 19 to 60 months of age, it was pneumonia (WHO, 1981).

2.5 BACTERIA CAUSING UTI ASSOCIATED WITH MALNUTRITION

It is difficult to estimate accurately the incidence of UTI in the pediatric population. Contributing questions include whether the determination of infection is based on symptoms, positive culture, or both; how accurate the method of specimen collection is; how accurate the history is, especially in young children; whether evaluation is focused on a specific age group or gender; whether the data are prospective or retrospective; whether or not the infections are associated with fever; and what the baseline rate of circumcision is in the population. Frequently quoted estimates place the incidence of UTI in infants at approximately 1% during the first year of life (boys and girls), cumulative incidence at approximately 2% at two years of life (boys and girls), and cumulative childhood risk at 2% for boys and 8% for girls (Jakobsson *et al*., 1999). Beyond the age of 2, UTIs in boys are not common enough to alter the childhood incidence through age 17. Boys are at the greatest risk for UTI in the first months of life, but the risk decreases significantly after age 2. Boys who are uncircumcised have a tenfold higher risk of UTI in the first year of life than do circumcised boys (Schoen *et al*., 2000). Girls have an increased risk of

febrile infection in the first year of life, and then the risk steadily declines throughout childhood. Their risk of non-febrile infections is higher during childhood than during infancy.

Urinary tract infection (UTI) by definition is the presence of microorganisms in urinary tract associated with pyuria. UTI remains the most common bacterial infection in childhood (Beetz, 2006). The cumulative incidence of UTI in children by 6 years of age is 3%–7% in girls and 1%-2% in boys (Beetz, 2006). Bacteria particularly *Escherichia coli* (*E. coli*)-gram negative rods are the most common cause of UTI, other organisms include viruses, fungi and parasites (Beetz, 2006). Recurrent urinary tract infections can result in chronic kidney disease and hypertension (Quigley, 2009). Over the recent years importance of UTI is well recognized and yet the management controversies are unsettled. This article will provide the latest evidence in the management of UTI in children.

In young children, the only symptom of a urinary tract infection (UTI) may be a fever. Because of the lack of more obvious symptoms, when females under the age of two or uncircumcised males less than a year exhibit a fever, a culture of the urine is recommended by many medical associations. Infants may feed poorly, vomit, sleep more, or show signs of jaundice. In older children, new onset urinaryincontinence (loss of bladder control) may occur. (Bhat *et al*., 2011)

E. coli is the cause of 80–85% of urinary tract infections, with *Staphylococcussaprophyticus* being the cause in 5–10%.(Nicolle, 2008) Rarely they may be due to viral or fungal infections.(Amdekar *et al*., 2011) Other bacterial causes include: *Klebsiella*, *Proteus*, *Pseudomonas*, and *Enterobacter*. These are uncommon and typically related to abnormalities of the urinary system.The evidence that preventative antibiotics decrease urinary tract infections in children is poor.(Dai *et al*., 2010) However recurrent UTIs are a rare cause of further kidney problems if there are no underlying abnormalities of the kidneys, resulting in less than a third of

a percent (0.33%) of chronickidneydisease in adults.(Salo *et al*., 2011) Whether routine circumcisions prevents UTIs has not been well studied as of 2011(Jagannath *et al*., 2012) Urinary tract infections due to *Staphylococcus aureus* typically occur secondary to blood-borne infections (Lane and Takhar, 2011).

2.5.1 Epidemiology and Pathophysiology of UTI

The distribution and pattern of UTI varies with age, gender, ethnicity, circumcision in boys and presence of congenital malformations. During neonatal period and early infancy males are more affected, probably because of anatomical abnormalities and prepuce colonization (Lopez *et al*., 2007). About 8% of girls, and 2% of boys experience at least one episode of UTI up to the age of 7 (Bauer and Kogan, 2008). In female infants UTI occurs in 0.1–0.4% and increase up to 1.4% during preschool age and 0.7–2.3% in school age (Clark *et al*., 2010).

It is important to divide UTIinto lower urinary tract infection, localized to the bladder and urethra (cystitis and urethritis) versus upper tract infection of the ureter, collecting system, and renal parenchyma (pyelonephritis). Ascending bacterial infection of the urinary tract is acomplex process that has been associated with bacterial adhesion, virulence, and motility properties of infecting microbes as well as host anatomic, humoral, and genetic factors (Svanborg and Godaly, 1997). The presence of fever, chills, and flank pain has usually been considered clinical evidence of upper tract infection. New technologies like technetium 99m-labeled dimercaptosuccinic acid (*DMSA*) scans to diagnose upper tract infections have demonstrated a wide range of estimates (34 to 70%) for the prevalence of pyelonephritis in children with febrile UTI(Lin *et al*., 2003).

2.5.2 Diagnosis of UTI

Clinically any febrile child, presenting without any fever localizing sign, is likely to have UTI. Neonates often present with very nonspecific symptoms such as an undifferentiated febrile illness, irritability, vomiting, or poor feeding, and, less commonly, with late-onset jaundice or failure to thrive (Lin, 2000). In infants and todlers the presentation is likely to be also nonspecific, including fever, diarrhoea, or vomiting with dehydration, failure to thrive, abdominal/flank pain, foul-smelling urine, and new-onset urinary incontinence, but rarely with more specific urinary symptoms (Garcia and Nager, 2002). In cases of serious bacterial infection, signs and symptoms may be subtle. In the older children symptoms and signs may be more specific to the urinary system, and include dysuria, foul-smelling urine, urgency, frequency, new-onset urinary incontinence, or gross hematuria. Systemic symptoms such as fever, abdominal or flank pain, and vomiting are highly suggestive of pyelonephritis. The physical exam is useful to exclude other possible causes for the patient's symptoms. It should be completed in infants and febrile patients. In older patients, the abdomen and genitalia should be examined, and the costovertebral angles should be palpated. Palpable bladder or abdominal mass, poor urinary flow, poor growth, and elevated BP may be seen with obstructive uropathy or chronic kidney disease and should prompt the clinician to consider abnormalities of the urinary tract (Smellie *et al*., 1964).

2.6 ANTIBIOTICS

Antibiotics are not usually used for gastroenteritis, although they are sometimes recommended if symptoms are particularly severe (Mandell *et al*., 2004) If antibiotics are to be employed, a macrolide (such as azithromycin) is preferred over a fluoroquinolone due to higher rates of resistance to the latter (Galanis, 2007).Pseudomembranous colitis, usually caused by antibiotic

use, is managed by discontinuing the causative agent and treating it with either metronidazole or vancomycin (Effa *et al*., 2011). Bacteria and protozoans that are amenable to treatment include *Shigella*(Gonzales *et al*., 2009) *Salmonella typhi*, and *Giardia* species (Escobedo *et al*., 2010).In those with *Giardia* species or *Entamoeba histolytica*, tinidazole treatment is recommended and superior to metronidazole(Escobedo *et al*., 2010). The World Health Organization (WHO) recommends the use of antibiotics in young children who have both bloody diarrhoea and fever (Amandeep, 2010).

In neonates the most likely pathogens are *E. coli* and *E. faecalis*, which require therapy witha β-lactam antibiotic and an aminoglycoside. For pyelonephritis, orally administered antibiotics are second and third-generation cephalosporins. Alternatively, amoxicillin–clavulanate, trimethoprim–sulfamethoxazole (*TMP-SMX*), and first-generation cephalosporins can be used with caution due to increasing resistance of *E. coli* (Fabre *et al*., 2010). Fluoroquinolones (ciprofloxacin) are effective for *E. coli* but should not be used as first-line agents due to their safety concerns in children (Fabre *et al*., 2010). Parenteral therapy with third- or fourth generation cephalosporins and aminoglycosides are appropriate for empiric treatment. When enterococcal UTI is suspected (associated with urinary catheter, instrumentation of the urinary bladder, or genitourinary abnormalities), ampicillin should be included in treatment options. Gentamicin can be used parenterally as an adjunctive treatment in resistant organisms after knowing the renal functions (Sermin *et al*., 2012).

2.6.1 Antimotility Agents

Antimotility medication has a theoretical risk of causing complications, and although clinical experience has shown this to be unlikely,(Warrell *et al*., 2003) these drugs are discouraged in

people with bloody diarrhoea or diarrhoea that is complicated by fever (Lozno, 2012). Loperamide, an opioid analogue, is commonly used for the symptomatic treatment of diarrhoea (Walker *et al*., 2013). Loperamide is not recommended in children, however, as it may cross the immature blood–brain barrier and cause toxicity. Bismuth subsalicylate, an insoluble complex of trivalent bismuth and salicylate, can be used in mild to moderate cases (Warrell *et al*.,2003) but salicylate toxicity is theoretically possible (Amandeep, 2010).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 STUDY DESIGN

The study was a Hospital based cross-sectional study.

3.2 STUDY AREA OR SITE

The study was conducted in the Northern Region of Ghana. Tamale is the capital of the Northern Region of Ghana and the fourth largest city of Ghana. The region shares boundaries with Upper East and West regions on the north, on the west with Ivory Coast, on the east with Togo and on the south with Brong Ahafo and Volta regions. It is also the home of Ghana's first University for Development Studies. Northern Region's total population was 2,479,461. The total male population was 1,229,887 while the females numbered 1,249,574. (Population and Housing Census, 2010)

The Tamale Metropolis is located in the centre of the Northern Region and shares boundaries with 5 other districts. The Metropolis has a total estimated land size of 750 km sq which is about 13% of the total land area of the Northern Region. There are a total of 197 communities in the Metropolis of which 164 are rural. According to 2010 population census, the total population for Tamale Metro was 371,351, comprising 185,995 males and 185,356 females.

The Tamale Teaching hospital (TTH) is a 340 bed capacity hospital situated in the Tamale Metropolitan area. In addition to offering clinical care to inhabitants of the metro and its surrounding districts, it also serves as a referral hospital to the two upper regions of Ghana. TTH

runs six clinical departments including the Paediatric ward which attends to children up to 14 years old. The ward has a nutrition unit that admits treats and provides nutritional rehabilitation to children with acute malnutrition. These children mostly come from the rural parts of Northern Ghana. The Laboratory Department of the hospital has a Bacteriology Unit that runs culture and antimicrobial susceptibility testingon clinical specimen from the hospital and other hospitals within the region.

3.3 STUDYPOPULATION

The study involved all children of five years and below admitted to the malnutrition unit of the Department of Child Health for management and nutritional rehabilitation from February 2013 to January 2014. The study covered 200 children who were sampled daily. Annually, an estimated 260 malnourished children are admitted to the unit for management and rehabilitation. For the years 2010, 2011 and 2012, an estimated 210, 269 and 305 children were admitted in the paediatric ward for nutritional rehabilitation respectively.

3.3.1 Inclusion Criteria

All children were 0 to 60 months old and admitted to the malnutrition unit of the department of child health of the Tamale Teaching Hospital, Tamale.

Anthropometric measurement; Weight for age <70% (Z-Score), Weight for height <80% (Z-Score) and Mid Upper Arm Circumference (MUAC) <11.5cm

All Children who Show symptom of sepsis, UTI, diarrhoea

Children who are able to produce stool and urine

Parent/guardian signs consent form

3.3.2 Exclusion Criteria

Children whose parents refused to sign consent form.

Children who could not produce stool and urine specimen.

3.4 ETHICAL CONSIDERATION

Ethical clearance for the study was obtained from the Ethical Review Committee of the research unit in the Tamale Teaching Hospital, Tamale. Permission to undertake the study at theTamale Teaching Hospital was sought and granted by the hospital management and the head of the laboratory. Consent forms were given to relatives of the study subjects to consent or reject before they were recruited into the study.

3.5 SAMPLING

All children who fulfilled the inclusion criteria were recruited and were provided with clean labeled sterile containers for stool and urine specimens. Additionally 3ml of blood was aseptically collected from all febrile children into Brain heart Infusion broth upon admission into the unit. The specimens were sent to the bacteriology laboratory for culture and susceptibility testing immediately. Staff of the pediatric ward assisted in the collection of samples for laboratory analysis.

3.6 URINE CULTURE PROCEDURE

Guardian/parents were educated on how to obtain urine sample from their wards so as to avoid contamination. Mid stream urine(MSU) was collected from all study children who fulfill the malnutrition criteria in to 10 to 20mls sterile, dry, wide-necked, leak proof containers. Where

there was delay in delivery for more than 2 hours anticipated, boric acid preservative was added to the urine.In the laboratory, the urine (freshly collected clean-catch specimen) was mixed by gently rotating the container. A sterile calibrated wire loop that holds 0.002mls of urine was used to inoculate a loopful of urine on a quarter plate of Cystine Lactose Electrolyte-Deficient (CLED) agar. The plates were incubated aerobically at 35–37 °C for 18-24 hours and examined for bacterial growth.

Plates that had no sign of growth were reported as no bacterial growth. Plates that had growth their count were determined and classified as being significant or non-significant. Significant count depicts a colony count of greater than 200 or 250 colony forming units (cfu) base on the loop used; (i.e. 1/500mls respectively).Growth that was less than the standard counts was reported as non significant growth. Significant growths were further classified as being pure or mixed. A pure growth means identifying the same colonies in one quadrant. A mixed growth means having more than one different colony in the same quadrant. For quadrants with mixed growth colonies, a number of different organisms were determined. Growths in a quadrant with at most two different colonies or organisms were reported as mixed growth and the predominant one was followed. Mix growth in a quadrant with at least two different colonies or organisms were reported as heavy mixed growth and a request for a new sample from the same patient was made to repeat the test. Growth that was pure was again classified as lactose or non lactose fermenter. Lactose fermenter appears yellow on CLED and pinkish on MacConkey agar. Non lactose fermenter appears greenish on CLED and transparent on MacConkey agar. Organisms that were identified as lactose fermenters were stabbed onto biochemical test media such as TSI,

urea, citrate, MIO, for 18-24 hours. The organisms that were identified as non- lactose fermenters were further tested for oxidase to rule out Pseudomonas groups. Oxidase negative organisms were put on biochemical test (TSI, urea, citrate, LIA, MIO) to identify the organism. Staphylococcus species suspected organisms were put on catalase test or gram stain was performed to confirm. Confirm Staphylococcus species were again tested for coagulase to confirm Staphylococcus aureus. Enterococcus (group D Streptococcus) suspected organisms were gram stained to confirm. Gram stain confirmed gram positive cocci in pairs and in chains were stabbed on bile esculin agar to determine the type of enterococcus.

3.7 STOOL CULTURE PROCEDURE

The stool was cultured on *Salmonella Shigella* agar (SSA). Some amount of stool was inoculated to the selenite F broth. *Salmonella Shigella* media was incubated for 18 to 24 hours and examined for growth. Lactose fermenters (pinkish colonies) grown on plates was reported as such and re -incubated for another 18-24 hours. Selenite F broth was sub-cultured onto another *Salmonella Shigella* agar (Secondary plate) and incubated for 18-24hours with the primary plate. Non lactose fermenters (transparent colonies with back spots) grown on plates was confirmed for*Salmonella* using biochemical test. The non-lactose fermenter without spot, the oxidase test was performed to rule out *Pseudomonas* group. For positive oxidase test organisms, *Pseudomonas* was queried and did not follow up. For organisms which was tested oxidase negative, a follow up was done with some biochemical tests such as TSI, Citrate, Urea, Indole, LIA, and MIO). The inoculated peptone water was used for sensitivity on Mueller -Hinton agar.

3.8 BLOOD CULTURE PROCEDURE

Two milliliters of blood was drawn from a peripheral vein under aseptic condition after cleaning the skin with 70% alcohol and 2% tincture of iodine and inoculated into brain heart infusion broth.The inoculated brain heart infusion broth was incubated at 35-37°C for up to 7 days. When there is an indication of growth within this period, 1ml of the inoculated broth was sub cultured onto ¼ plates of Blood agar, Chocolate (heated blood) agar and MacConkey agar. Chocolate agar plates were incubated at 35- 37°C aerobically in a carbon dioxide atmosphere for up to 48 hours, the blood and MacConkey agar plate at 35-37°C aerobically overnightfor bacteria growth. Following the manufacturer's instructions for quality control and blood volume requirements, these media supports the growth of antibiotic-exposed organisms thereby enhancing the recovery of susceptible isolates. Plates were examined for pathogens using standard procedures. Blood cultures were considered positive if pathogenic bacteria such as *Klebsiella spp, Citrobacter spp, Streptococcus spp, Escherichia coli, Salmonella species, Staphylococcus aureus, Proteus species, etc were isolated. Coagulase-negative Staphylococci, Micrococcus species, Bacillus species, Candida spp, Acinetobacter spp* and isolates with scanty growth not on the line of inoculums that failed to grow on subcultures were regarded as contaminants.

All positive plates were gram stained and bacteria species were identify before biochemical and antimicrobial sensitivity tests were performed. (Monica Cheesbrough*et al*., 2000, 2006**)**

3.9 DATA ANALYSIS

Results are presented as numbers (percentages). For all statistical comparisons, a p-value of <0.05 was considered significant. All statistical analysis were done using Microsoft Excel and Statistical Package for Social Scientist (SPSS) version 21.

CHAPTER FOUR

4.0 RESULTS

4.1 AGE AND SEX DISTRIBUTION

Table 4.1 Many (30.0%) of the study population were within the age group 12-23 months with 61.7% of the age group being males compared to 38.3% being female. The age group 48-60 months had the least number of participants (5.5%) with 72.7% of this group being males and 27.7% being females. The total number of children examined was 200. Data was presented as no (%), p- values were found using unpaired t-test, there was statistically no gender variation in the study population ($p > 0.05$).

Table 4.1: Male and Female sex Distributions of Malnourished Children between the Ages of Zero to 60 Months (5years)

Age in month	TOTAL	MALEN (%)	FEMALE N (%)	P-value
≤ 11	48(24.0)	26(54.2)	22(45.8)	0.8124
12-23	60(30.0)	37(61.7)	23(38.3)	0.7586
24-35	50(25.0)	28(56.0)	22(44.0)	0.8045
36-47	31(15.5)	11(35.5)	20(64.5)	0.8616
≥48-60	11(5.5)	8(72.7)	3(27.3)	0.8885

4.2 THE AGE DISTRIBUTION AND PATIENTS COMPLAINTS

From table 4.2, many of these subjects presented with cases of diarrhoea (73.3%), only 18.2% presenting with fever. Data was presented as no (%), p- values were found using one- way anova statistical method. There was however statistically no difference among the age groups with regards to patients complaints (P>0.05).

Table 4.2: Age Distribution and Patients Complaints of MalnourishedChildrenbetween the Ages of Zeroto 60 Months (5years)

AGE GROUP	TOTAL	DIARRHOEA (%)	FEVER (%)	VOMITING (%)	P-value
≤ 11	48(24.0%)	32(66.7)	28(58.3)	26(54.2)	0.9600
12-23	60(30.0%)	44(73.3)	29(48.3)	40(66.7)	0.9217
24-35	50(25.0%)	32(64)	32(64)	33(66)	0.9498
36-47	31(15.5%)	16(51.6)	15(48.4)	15(48.4)	0.9802
≥48-60	11(5.5%)	7(63.6)	2(18,2)	4(36.4)	0.9872
TOTAL	200	131(65.5)	106(53)	118(59)	

4.3 MID UPPER ARM CIRCUMFERENCE (MUAC) AND AGE DISTRIBUTION

Table 4.3 many of the study subjects with SAM (69.2%), with only 3.8% of them having adequate nutrition. There was statistically no difference in the risk of developing any type of malnutrition in all the age groups except in the age group 12-23months, which showed a significant statistical difference ($p < 0.05$). Data was presented as No (%), p-values were found using one-way anovas statistical method. Mid upper arms circumference (MUAC) was not considered among <6 months children i.e. 18(9.0%) number and Oedema children i.e. 23(11.5%) number because of undeveloped arms of the <6 months children and fluid retention of the oedema children, in considering these in MAUC may caused a false increase or decrease in the MAUC values. A special method was used to assess or include these groups of children.

Table 4.3: Mid Upper Arm Circumference (MUAC) and Age Distribution of Children between the Ages of Zero to 60 Months (5years)

MONTHS	TOTAL	SAM (N %)	*MOAM(N*	MAM (N %)	AN (N %)	p-value
>_6≤ 11	28	22(78.6)	*5(17.9)*	0(0.0)	1(3.6)	0.2780
12-23	52	39(75.0)	*9(17.3)*	4(7.7)	0(0.0)	**0.0404**
24-35	41	27(65.9)	*9(22)*	3(7.3)	2(4.9)	0.1768
36-47	28	16(57.1)	*5(17.9*	4(14.3)	3(10.7	0.3629
≥48-60	10	6(60.0)	*1(10)*	3(30)	0(0.0)	0.5244
TOTAL	159	110(69.2)	*29(18.2)*	14(8.8)	6(3.8)	

The MUAC length for Severe Acute Malnutrition (SAM) is<11.5 cm, Moderate Acute Malnutrition (MOAM) is ≥11.5<12.5cm, Mild Acute Malnutrition (MAM) is ≥12.5<13.5cm andAdequate Nutrition (AN) is ≥13.5cm.

4.4 AGE DISTRIBUTION AND TERMS OF SEVERE ACUTE MALNUTRITION

Table 4.4 shows many (71.5%) of the study subjects presented with marasmus, 20% with presenting with kwashiorkor and the remaining 8.5% presenting with marasmus kwashiorkor. There was no significant variation in the risk of developing all the three forms malnutrition among all the age groups. Data was presented as no (%), p- values were found using one- way anovas statistical method.

Table4.4: Age Distribution and Terms of Severe Malnutrition among Malnourished Children between Ages of 0 Month to 60 Months (5 years)

Age	Total	Marasmus	Kwashiorkor	MarasmicKwashiokor	**P-value**
≤ 11	48	43(89.6)	5(10.4)	0(0.0)	0.1897
12-23	60	44(73.3)	7(11.7)	9(15.0)	0.1573
24-35	50	32(64.0)	13(26)	5(10.0)	0.2685
36-47	31	20(64.5)	10(32.3)	1(3.2)	0.4136
≥48-60	11	4(36.4)	5(45.5)	2(18.2)	0.5630
Total	200	143(71.5)	40(20.0)	17(8.5)	

4.5 SEX AND MALNUTRITION, <6 MONTHS AGE GROUP, CHILDREN WITH OEDEMA AND MUAC OF VARIOUS CATEGORIES.

Table 4.5: study subjects presenting with severe acute malnutrition (SAM) represented 55.0% of the study population with 58.2%of this cohort being males and the remaining 41.8% being females.

Adequate nutrition (AN) using MUAC criteria represented 3.0% with the male in this group being 16.7% of the study population and the remaining 83.3% being females. There was however significantly no gender difference among the different types of malnutrition using the MUAC criteria. Data was presented as no (%), p- values were found using unpaired t-test.

Table 4.5: Sex against Malnutrition, <6 Months Age Groupand Children with Oedema among Malnourished Children from Zero to Five Months of Age

	Total	Male	Female	P-values
<6 months children	18(9.0)	9(50.0)	9(50.0)	0.8798
Oedema children	23(11.5)	18(78.3)	5(21.7)	0.8713
SAM (<11.5cm)	110(55.0)	64(58.2)	46(41.8)	0.3845
MOAM(≥11.5<12.5)	29(14.5)	10(34.5)	19(65.5)	0.8652
MAM(≥12.5<13.5)	14(7.0)	8(57.1)	6(42.6)	0.8826
AN(≥13.5)	6(3.0)	1(16.7)	5(83.3)	0.8954

4.6 SEX DISTRIBUTION AND TERMS OF SEVERE ACUTE MALNUTRITION

Table 4.6: Many of the study subjects presented with marasmus with 53.1% of them being males and 46.9% being females. Subjects with marasmic kwashiorkor represented 8.5% of the study population with 70.6% of this study cohort being males and the remaining 29.4% being females. Data was presented as no (%), p- values were found using unpaired t-test, there was statistically no difference on the basis of gender when the different type of malnutrition was compared.

Table 4.6: Sex Distributionand Termsof Severe Acute Malnutrition Cross Tabulation

Type of malnutrition	Total	Male	Female	P-value
Marasmus	143(71.5)	76(53.1)	67(46.9)	0.6367
Kwashiokor	40(20.0)	22(55.0)	18(45.0)	0.8364
Marasmic kwashiokor	17(8.5)	12((70.6)	5(29.4)	0.8805

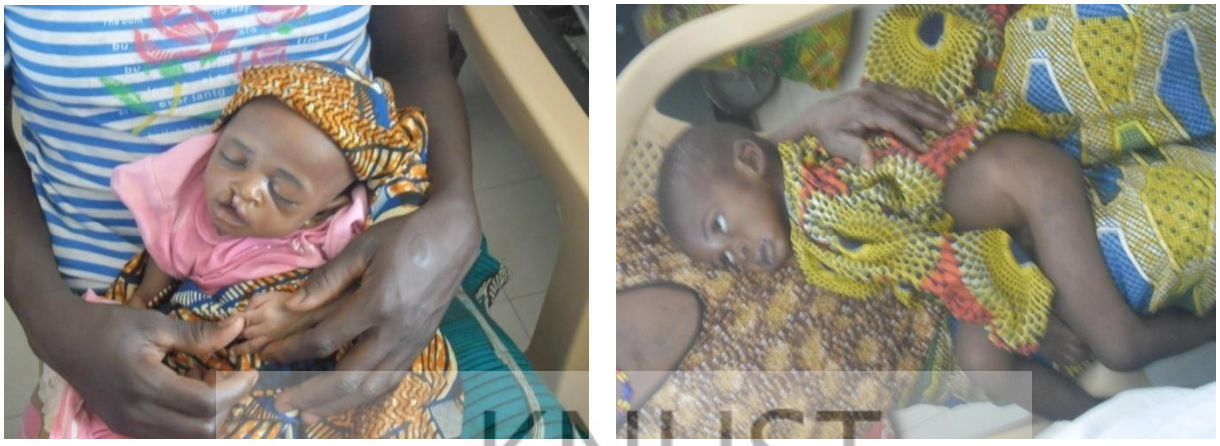

Fig 1.0 PICTURES SHOWING SAM CASES IN THE HOSPITAL

Fig 2.0 SOME CRITICAL SAM CASES SEEN BY THE TAMALE TEACHING HOSPITAL

Fig 3.0 SOME CRITICAL SAM CASES OF 2011

4.7 AGE AND SEX DISTRIBUTION OF BACTERIAL ISOLATES FROM BLOOD.

Table 4.7 the rate of isolation from Table 4.7 below was highest among age group ≤ 11 months (37.9%) for blood isolates. The overall nunber of isolates in blood reduced with increasing age but the types of organisms cultured did not vary with age. The most frequent isolates were *S. aureus* (34.5%), *Klebsiella* sp (27.6%), *E. coli* (10.3%),*Pseudomonas spp* (/10.3%), *Proteus* spp (10.3%) and *Strep spp*(6.9%). However, the prevalence of bacteria isolates in blood was 29/198(14.6%).

Table 4.7: Age and Sex Distribution of Bacterial Isolates from Blood of Malnourished Children between Ages of 0 Month to 60 Months (5 years)

Age	*E. coli*	Kleb.*spp*	*Prot.spp*	*Pseudo.spp*	*S.aureus*	*Strep.spp*	Total N%
≤ 11	2(18.2)	3(27.3)	0(0.0)	0(0.0)	5(45.5)	1(9.1)	11(37.9)
12-23	1(14.3)	0(0.0)	2(28.6)	1(14.3)	2(28.6)	1(14.3)	7(24.1)
24-35	0(0.0)	1(25.0)	1(25.0)	1(25.0)	1(25.0)	0(0.0)	4(13.8)
36-47	0(0.0)	3(50.0)	0(0.0)	1(16.7)	2(33.3)	0(0.0)	6(20.7)
≥48-60	0(0.0)	1(100.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	1(3.4)
Total	3(10.3)	8(27.6)	3(10.3)	3(10.3)	10(34.5)	2(6.9)	29(100.0)
Sex							
Male	3(15.8)	4(21.1)	2(10.5)	1(5.3)	7(36.8)	2(10.5)	19(65.5)
Female	0(0)	4(40)	1(10)	2(20)	3(30)	0(0.0)	10(34.5)
Total	3(10.3)	8(27.6)	3(10.3)	3(10.3)	10(34.5)	2(6.9)	29(100)

4.8 BACTERIA ISOLATES FROM BLOOD FOR THE VARIOUS TYPES OF SEVERE ACUTE OF MALNUTRITION

In table 4.8, most of the bacterial infections resulted in marasmus malnutrition, but the major bacteria isolates that cause marasmus were *Staphylococcus aureus* (70.0%), and *Klebsiella spp* (75.0%).

Table 4.8: Bacteria Isolate from Blood for the Various Types Severe Acute Malnutritionin Children between Ages of 0 Month to 60 Months (5 years)

Bacterial isolates	Marasmus N (%)	Kwashiokor N (%)	Marasmic/kwashiokor N (%)	Total N (%)
Escherichia coli	2(66.7)	1(33.3)	0(0.0)	3(10.3)
Klebsiella spp	6(75.0)	2(25.0)	0(0.0)	8(27.6)
Proteus spp	2(66.7)	0(0.0)	1(33.3)	3(10.3)
Pseudomonas spp	1(33.3)	2(66.7)	0(0.0)	3(10.3)
Stapylococcus aureus	7(70.0)	3(30.0)	0(0.0)	10(34.5)
Streptococcus spp	1(50.0)	1(50.0)	0(0.0)	2(6.9)
Total	19(65.5)	9(31.0)	1(3.4)	29(100)

4.9 BACTERIA ISOLATES FOR, <6 MONTHS AGE GROUP, CHILDREN WITH OEDEMA AND MUAC OF VARIOUS CATEGORIES.

Table 4.9 The severe acute malnutrition (SAM) was as a result of *Staphylococcus aureus, Klebsiella spp, E.coli, Pseudomonas spp* and *Streptococcus spp.* which were found in the blood of the malnourished children of which *Staphylococcus* aureus and *Klebsiella spp* were the leading cause likewise children<6months of age.

Table 4.9: Bacteria Isolates from Blood, MUAC and Non MAUC of Malnourished Children

	NO MUAC		MUAC				
Isolates	<6months	Oedema	SAM	MOAM	MAM	AN	Total
E coli	0(0.0)	0(0.0)	2(66.7)	0(0.0)	1(33.3)	0(0.0)	3
Kleb spp	1(12.5)	1(12.5)	5(62.5%)	1(12.5)	0(0.0)	0(0.0)	8
Proteus spp	0(0.0)	3(100.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	3
Pseud- spp	0(0.0)	1(33.3)	1(33.3)	0(0.0)	0(0.0)	1(33.3)	3
Staph-aureus	1(10.0%)	2(20.0)	6(60.0%)	0(0.0)	0(0.0)	1(10%)	10
*Strep-*spp	1(50.0)	0(0.0)	1(50.0)	0(0.0)	0(0.0)	0(0.0)	2
Total	3(10.3)	7(24.1)	15(51.7)	1(3.4)	1(3.4)	2(6.9)	29

4.10 ANTIBIOTICS RESISTANCE IN BACTERIA FROM BLOOD ISOLATES OF THE MALNOURISHED CHILDREN BETWEEN AGES OF 0 TO 5 YEARS.

In table 4.10, 90% Ciprofloxacin and Cephalexin were resistant to *Staphylococcus aureus*, 100% Cotrimoxazole and 50% Tertracycline, Ciprofloxacin, and gentamicin was resistant to *Streptococcus spp*. in the blood of malnourished children.

Table 4.10 Antibiotic Resistance From Blood Isolates of the Malnourished Children Between ages of 0 to 5 Years

BACTERIAL ISOLATES	Total(N)	RESISTANCE	AS	BA	PR	TR	CP	GM	AK	LE
Escherichia coli	3	R	3(100.0)	3(100.0)	0(0.0)	3(100.0)	2(66.7)	3(100.0)	3(100.0)	0(100)
*Klebsiella*spp	8	R	8(100.0)	7(87.5)	0(0.0)	8(100.0)	7(87.5)	7(87.5)	7(87.5)	4(50.0)
*Proteus*spp	3	R	3(100.0)	3(100.0)	0(0.0)	3(100.0)	3(100.0)	3(100.0)	3(100.0)	0(0.0)
*Pseudomonas*spp	3	R	0(0.0)	0(0.0)	0(0.0)	0(0.0)	3(100.0)	3(100.0)	3(100.0)	3(100.0)
Stapylococcusaureus	10	R	8(80.0)	8(80.0)	1(10.0)	7(70.0)	0(0.0)	9(90.0)	X	X
*Streptococcus*spp	2	R	1(10.0)	0(0.0)	0(0.0)	1(50.0)	1(50.0)	1(50.0)	X	X

AS- AMPICILLIN, BA-COTRIMOXAZOLE, PR-CEPHALEXIN, TE-TETRACYCLINE CP-CIPROFLOXACIN, GN-GENTAMICIN, AK-AMIKACIN, LE-LEVOFLOXACIN AND R=RESISTANCE

.

4.11 AGE AND SEX DISTRIBUTION OF BACTERIAL ISOLATES FROM STOOL OF MALNOURISHED CHILDREN BETWEEN AGES OF 0 MONTH TO 60 MONTHS (5 YEARS).

Table 4.11, the 199 stool cultures yielded 3(1.5%) *Shigella spp*, the number of isolation remain the same for all the age groups except that the males recorded a higher number than females.

Table 4.11: Age and Sex Distribution of Bacterial Isolates from Stool of Malnourished Children between Ages of 0 Month to 60 Months (5 Years)

ISOLATES	Age in months				SEX/Gender	
	0-11	12-23	36-47	TOTAL	MALE	FEMALE
SHIGELLA	1(33.3%)	1(33.3%)	1(33.3%)	3	2(66.7%)	1 (33.3%)

4.12 AGE, SEX, MUAC AND TYPE OF MALNUTRITION DISTRIBUTION OF ISOLATES IN STOOL

In Table 4.12, one (1) male child each between 0-11 months and 12-23 months had marasmus /SAM and kwashiorkor/ MAM respectively, while one (1) female between 36-47 months had marasmus/ SAM.

Table 4.12: The Distribution of Children in which *Shigella spp* was Isolated with Respect to their Nutritional Status, Gender and Age Group

Gender	Age group	Marasmus/SAM	Kwashiorkor/MAM	Total
Male	0-11	1	0	2
	12-23	0	1	
Female	36-47	1	0	1
Total		2	1	3

4.13 ANTIBIOTICS RESISTANCE PATTERN OF BACTERIA ISOLATES FROM STOOL

Table 4.13 Antibiotics activities on *Shigella spp* showed that 100% Ciprofloxacin and 66.7% Ampicillin Sulbactam and cotrimoxazole were sensitive to *Shigella* while 100% gentamicin, Amikacin Levofoxacin and 33% cotrimoxazole were resistant to *Shigella.*

Table 4.13: Antibiotic Resistance Pattern against *Shigella spp* in Stool

ANTIBIOTICS	SUSCEPTIBLE	RESISTANT	TOTAL
AMPICILLIN	2(66.7)	1(33.3)	3
COTRIMOXAZOLE	2(66.7)	1(33.3)	3
TETRACYCLIN	0(0.0)	3(100)	3
CIPROFLOXACIN	3(100)	0(0.0)	3
GENTAMICIN	0(0.0)	3(100)	3
AMIKACIN	0(0.0)	3(100)	3
LEVOFLOXACINE	0(0.0)	3(100)	3

4.14 AGE AND SEX DISTRIBUTION OF BACTERIA ISOLATES FROM URINE OF MALNOURISHED CHILDREN BETWEEN AGES OF 0 MONTH TO 60 MONTHS (5 YEARS)

In table 4.14 the rate of isolation from table 14 was highest among age group 12-23 months (34.7%) for urine isolates. However the isolation reduced afterwards. Males (65.3%) recorded the highest number of isolates than females (34.7%). *Klebsiella* spp took the lead with 38.8% number of isolates and *Enterobacter spp* with 2.0% number of isolates was the least. Prevalence of bacteria isolates in urine was 49/180(27.2%).

Table 4.14: Age and Sex Distribution of Bacteria Isolates from Urine of Malnourished Children

ISOLATES	AGE IN MONTHS						SEX		
	<6-11	12-23	24-35	36-47	48-60	TOTAL	Male	Female	Total
Proteus	1(12.5)	4(50.0)	2(25.0)	0(0.0)	1(12.5)	8	5(62.5)	3(37.5)	8
Citrobacter	2(50.0)	1(25.0)	0(0.0)	1(25.0)	0(0.0)	4	1(25.0)	3(75.0)	4
Enterobacter	1(100)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	1	1(100)	0(0.0)	1
Entrococcus	0(0.0)	0(0.0)	1(100)	0(0.0)	0(0.0)	1	1(100)	0(0.0)	1
E.coli	0(0.0)	3(60.0)	0(0.0)	2(40.0)	0(0.0)	5	2(40.0)	3(60.0)	5
Kleb spp	8(42.1)	7(36.8)	0(0.0)	4(21.1)	0(0.0)	19	15(78.9)	4(21.1)	19
Pseudo spp	2(25.0)	2(25.0)	3(37.5)	1(12.5)	0(0.0)	8	5(62.5)	3(37.5%)	8
S.aureus	1(33.3)	0(0.0)	2(66.6)	0(0.0)	0(0.0)	3	2(66.7)	1(33.3)	3
Total	15(30.6)	17(34.7)	8(16.3)	8(16.3)	1(2.0)	49	32(65.3)	17(34.7)	49

4.15 URINE ISOLATES FROM <6MONTHS CHILDREN, CHILDREN WITH OEDEMA AND MUAC OF VARIOUS CATEGORIES.

Table4.15 shows bacteria isolates from blood and muac of malnourished children between ages of 0 month to 60 months (5 years).

Table 4.15: Bacteria Isolates from Urine against no MUAC and MUAC of Malnourished Children

	NO MAUC		MUAC			
	<6 months	Oedema	<_11.5	>=11.5<12.5	>=12.5<13.5	>=13.5
PROTEUS	0(0.0)	3(37.5)	5(62.5)	0(0.0)	0(0.0)	0(0.0)
CITROBACTE	0(0.0)	0(0.0)	2(50.0)	0(0.0)	1(25.0)	1(25.0)
ENTEROBACT	0(0.0)	0(0.0)	1(100)	0(0.0)	0(0.0)	0(0.0)
ENTEROCOCCU	0(0.0)	0(0.0)	1(100)	0(0.0)	0(0.0)	0(0.0)
E. COLI	0(0.0)	0(0.0)	3(60.0)	1(20.0)	0(0.0)	1(20.0)
KLEB SPP	5(26.3)	4(21.1)	8(42.1)	1(5.3)	1(5.2)	0(0.0)
PSEUDO SPP	0(0.0)	3(37.5)	2(25.0)	3(37.5)	0(0.0)	0(0.0)
S. AUREUS	1(33.3)	0(0.0)	1(33.3)	1(33.3)	0(0.0)	0(0.0)
TOTAL	6(12.2)	10(20.4)	23(46.9)	6(12.2)	2(4.1)	2(4.1)

MUAC= Mid Upper Arm Circumference, SAM (Severe Acute Malnutrition):<11.5cm, MOAM(Moderate Acute Malnutrition: ≥11.5<12.5cm, MAM (Mild Acute Malnutrition): ≥12.5<13.5cm, AN (Adequate Nutrition): ≥13.5cm

4.16 BACTERIA ISOLATE FROM URINE AND TERMS OF MALNUTRITION FROM MALNOURISHED CHILDREN.

Out of the total number of isolates, 63.3% were marasmus, 22.5% were kwashiorkor, and 14.3% marasmic kwashiorkor. These infections may be due to close contacts to pathogenic agents.

Table 4.16: Bacteria Isolate from Urine and Terms or Type of Malnutrition in Malnourished Children

ISOLATES	Marasmus	Kwashiokor	MarasmicKwashiokor	Total
PROTEUS	7(87.5)	1(12.5)	0(0.0)	8
CITROBACTER	3(75.0)	1(25.0)	0(0.0)	4
ENTEROBACTER	1(100)	0(0.0)	0(0.0)	1
ENTEROCOCCUS	0(0.0)	0(0.0)	1(100)	1
E. COLI	2(40.0)	2(40.0)	1(20.0)	5
KLEB SPP	13(68.4)	4(21.1)	2(10.5)	19
PSEUDO SPP	3(37.5)	3(37.5)	2(25.0)	8
STAPY AUREUS	2(66.7)	0(0.0)	1(33.3)	3
TOTAL	31(63.3)	11(22.5)	7(14.3)	49

4.17 ANTIBIOTICS RESISTANCE IN BACTERIA ISOLATES FROM URINE OF MALNOURISHED CHILDREN BETWEEN AGES OF 0 MONTH TO 60 MONTHS (5 YEARS)

In table 4.17 100% and 75.5% of Levofloxacin was resistant to *Escherichia coli, Enterococcus spp* and *Citrobacter,* 100% of gentamicin was resistant to *Proteus spp, citrobacter spp,Enterococcus spp, E.coli Pseudomonas spp, Staphylococcus aureus* and 89% *Klebsiella*spp.100% of cephalexin was resistant to *Staphylococcus aureus* and 85.7% Ampicillin, cotrimoxazole and Tetracycline were resistant to *Pseudomonas*

Table 4.17: Antibiotic Resistance in Urine Isolates Bacterial Isolates from Urine of Malnourished Children between Ages of 0 Months to 60 Months (5 years)

ISOLATES	RESISTANCE		AK	LE	NOR	CP	GN	AS	BA	PR	TE
PROTEUS SPP	8	R	0(0.0)	5(62.5)	0(0.0)	0(0.0)	4(100)	1(12.5)	1(12.5)	3(75.0)	1(12.5)
CITROB SPP	4	R	0(0.0)	3(75.5)	0(0.0)	0(0.0)	2(100)	1(25.0)	1(25.0)	2(100)	0(0.0)
ENTEROBAC SPP	1	R	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
ENTEROCOC SPP	1	R	0(0.0)	1(100)	0(0.0)	0(0.0)	1(100)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
E COLI	5	R	1(20.0)	4(100)	1(100)	0(0.0)	3(100)	0(0.0)	0(0.0)	1(100)	0(0.0)
KLEB SPP	19	R	0(0.0)	1(52.6)	3(30.0)	0(0.0)	8(88.9)	2(10.5)	4(21.1)	7(70.0)	1(5.3)
PEUD SPP	8	R	0(0.0)	1(12.5)	1(25.0)	0(0.0)	4(100)	7(87.5)	7(87.5)	4(100)	7(87.5)
S.AUREUS	3	R	0(0.0)	0(0.0)	0(0.0)	2(6.7)	3(100)	2(66.7)	0(0.0)	3(100)	1(33.3)

AK-AMIKACIN LE-LEVOFLOXACIN PR-CEPHALEXIN NOR- NORVOFLOXACIN CP-CIPROFLOXACIN

GN-GENTAMICIN AS- AMPICILLIN BA-COTRIMOXAZOLE TE-TETRACYCLINE

4.18 AGE AND SEX DISTRIBUTION OF NON MALNOURISHED CHILDREN FROM BLOOD BETWEEN THE AGES OF ZERO TO 60 MONTHS (5YEARS).

Table 4.18 many (62.6%) of the study population were within the age group ≤ 11 months with 70(56.5%) of the age group being males compared to 54(43.6 %) being female. The age group 24-35 and 48-60 months had the least number of participants (8.0%) with 12(75.0%) and 9(56.3%) of these groups being males whiles 4(25.0%) and 7(43.8%) being females. The total number of children examined was 198. Data was presented as No (%), p-values were found using unpaired t-test, there was statistically no gender variation in the study population (p >0.05).

Table 4.18: Male and Female Sex Distributions of Non-Malnourished Children from Blood between the Ages of Zero to 60 Months (5years)

AGE IN MONTHS	TOTAL	FEMALE N (%)	MALE N (%)	P-VALUE
≤ 11	124(62.6%)	54(43.6)	70(56.5)	0.438
12-23	22(11.1%)	9(40.9)	13(59.1)	0.7873
24-35	16(8.0%)	4(25.0)	12(75.0)	0.8005
36-47	20(10.1)	8(40.0)	12(60.0)	0.7923
≥48-60	16(8.0%)	7(43.8)	9(56.3)	0.8024
Total	198	82(41.4)	116(58.6)	

4.19 AGE AND SEX DISTRIBUTION OF BACTERIA ISOLATES FROM BLOOD OF NON MALNOURISHED CHILDREN

In table 4.19 the rate of isolation was highest among age group ≤ 11 months (95.5%) for blood isolates. The overall rate of isolation in blood reduced with increasing age but the types of organisms cultured did not vary with age. The most frequent isolates were *Staphylococcus aureus* 22.7%, *E.coli* 4.5%, *klebsiella spp* 68.2% and *Shigella* spp 4.5%. However, few organisms where isolated as compare to the malnourished children.

Table 4.19: Age and Sex Distribution of Bacterial Isolates from Blood of Non Malnourished Children between Ages of 0 Month to 60 Months (5 years)

	BLOOD ISOLATES				
AGE	*E.COLI*	*KLEB SPP*	*SHIGELLA*	*S AUREUS*	Total
≤ 11	1(4.8)	14(66.7)	1(4.8)	5(23.8)	21(95.5)
12-23	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
24-35	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
36-47	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
≥48-60	0(0.0)	1(100)	0(0.0)	0(0.0)	1(4.5)
Total	1(4.5)	15(68.2)	1(4.5)	5(22.7)	22(100)
SEX					
F	0(0.0)	4(57.1)	1(14.3)	2(28.6)	7(31.8)
M	1(6.7)	11(73.3)	0(0.0)	3(20.0)	15(68.2)
Total	1(4.5)	15(68.2)	1(4.5)	5(22.7)	22(100)

4.20 AGE AND SEX DISTRIBUTION OF NON MALNOURISHED CHILDREN FROM URINE

In table 4.20 many (33.9%) of the study subjects were within the age group ≤ 11months with 55.3 % of the age group being males compared to 44.7 % being female. The age group 48-60 months had the least number of participants (12.5%) with 71.4 % of this group being males and 28.8% being females. The total number of children examined was 112. Data was presented as no (%), p- values were found using unpaired t-test, there was statistically no gender variation in the study population (p >0.05).

Table 4.20: Age and Sex Distribution of Non Malnourished from Urine Children between the Ages of Zero to 60 Months (5years)

AGE IN MONTHS	TOTAL	MALE	FEMALE	P-VALUE
≤ 11	38(33.9)	21(55.3)	17(44.7)	0.5660
12-23	20(17.9)	12(60.0)	8(40.0)	0.7596
24-35	19(17.0)	9(47.4)	10(52.6)	0.7714
36-47	21(18.8)	13(61.9)	8(38.1)	0.7525
≥48-60	14(12.5)	10(71.4)	4(28.8)	0.791
Total	112(100)	65(58.0)	47(42.1)	

4.21 AGE AND SEX DISTRIBUTIONS OF BACTERIA ISOLATES FROM URINE OF NON MALNOURISHED CHILDREN.

In this table, age group ≤ 11 were many as compared to the malnourished children in urine with 12-23 months age group being the majority. Also, few related organisms where isolated.

Table 4.21: Age and Sex Distributions of Bacteria Isolates from Urine of Non Malnourished Children between Ages of 0 Month to 60 Months (5 years)

AGE OF CHILD	*E COLI*	*KLEB SPP*	*SHIGELLA*	*S AUREUS*	Total
≤ 11	0(0.0)	1(14.3)	3(42.9)	3(42.9)	7(53.8)
12-23	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
24-35	0(0.0)	0(0.0)	1(100)	0(0.0)	1(7.7)
36-47	1(20.0)	2(40.0)	2(40.0)	0(0.0)	5(38.5)
≥48-60	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Total	1(20.0)	3(23.1)	6(46.2)	3(23.1)	13(100)
SEX					
M	1(20.0)	1(20.0)	2(40.0)	1(20.0)	5(38.5)
F	0(0.0)	2(25)	4(50.0)	2(25)	8(61.5)
Total	1(7.7)	3(23.1)	6(46.2)	3(23.1)	13(100)

4.22 AGE AND SEX DISTRIBUTION OF NON MALNOURISHED CHILDREN FROM STOOL BETWEEN AGES OF 0 MONTH TO 60

Table 4.22 Many (39.4%) of the study subjects were within the age group ≤11months. with 67.9% of this cohort being males and the remaining (32.1 %) being females.

The age group 48-60 months had the least number of subjects (5.6%) with 50.0% of this group being males and 50.0% being females. The total numberof children examined was 71. Data was presented as No (%), p- values were found using unpaired t-test, there was statistically no gender variation in the study population (p >0.05).

Table 4.22: Age and Sex Distributions of NonMalnourished Children from Stool between the Ages of Zero to 60 Months (5years)

AGE OF CHILD	Total	MALE (N %)	FEMALE (N %)	p-value
≤ 11	28(39.4)	19(67.9)	9(32.1)	0.3118
12-23	26(36.6)	17(65.4)	9(34.6)	0.3809
24-35	11(15.5)	3(27.3)	8(72.7)	0.7369
36-47	2(2.8)	2(100)	0(0.0)	0.7843
≥48-60	4(5.6)	2(50.0)	2(50.0)	0.7744
Total	71(100)	43(60.7)	28(39.4)	

4.23 AGE AND SEX DISTRIBUTION OF BACTERIAL ISOLATES IN STOOL OF NON MALNOURISHED CHILDREN

There was only one *Shigella spp* isolated among the age group 12-23 months.

CHAPTER FIVE

5.0 DISCUSSION OF RESULTS

5.1 AGE AND SEX

The number of children examined was 200, in which 72.7% were males and 27.3% were females.The number of boys was higher than girls in nearly all age groups as shown in table 4.1, this may be due to the fact that the boys during infancy have to build a larger muscle mass than girls. Consequently, boys might have increased demands for micronutrients, and are therefore more at risk of a negative balance, including lack of vitamin A and zinc. This vulnerability mightincrease the risk of diarrhoea and malnutrition, and place the boys as the weaker sex regarding infections, this is in accordance with Molbak. 2000 findings.During neonatal period and early infancy malesare more affected, probably because of anatomical abnormalities and prepuce colonization (Lopez *et al*., 2007).

More so among older children, because boys are more active than girls, boys tend to move around and touch objects in the surrounding ground, whereas girls might tend to stay close to their mothers and/or to play with more hygienic toys. This is in agreement with Thiem *et al*., 2004.

Among the age groups,30% were within the age group of 12-23 months with 67.7% of this age group being males compared to 38.3% being females. The increase in the number of the age groups less than 48-60 months may be as a result of their immune system not well developed to resist infections. The age group 48- 60 months had the least number of participants (5.5%) with 72.7% of this group being males and 27.7% being female. A decrease in number of cases among

older children, 48-60 months might have resulted from the fact that the immune system in older children got stronger in resisting against agents.

5.2 AGE AND PATIENTS COMPLAINTS

The patient's complaints of the malnourished in table 4.2 were multi- complaints of diarrhoea, fever and vomiting. Their p-value was not significant. Diarrhoea with 73.3% complaint was the highest of the complaints and this may lead to other complaints.

Diarrhoea complaint may be as a result of touching and swallowing of contaminated objects by these children. Also Irregular hand washing by mothers aftergoing to toilet and no hand-washing by mothers before feeding children may cause diarrhoea. Likewise, washing hands have been clearly shown to reduce the occurrence of diarrhoea (Fewtrell *et al.*, 2005).

It is generally recognized that contamination of complementary foods may occur as a result of poor hygiene of food handlers, household equipments and the environment where the preparation of food takes place (Sheth *et al.*, 2000).

Improper storage and handling of cooked food is equally responsible for food borne illnesses, as during storage especially at ambient temperature (28 to 35°C) there is the risks of multiplication of pathogenic organisms. Under favourable conditions, a single bacterium can multiply to 500 million bacteria in 10 h.(Sheth *et al.*, 2000).

5.3MID UPPER ARM CIRCUMFERENCE (MUAC) AGAINST AGE.

The mid-upper-arm circumference (MUAC) in table 4.3 of the malnourished patients studied revealed that more than half of the children admitted (69.2%) suffered from severe acute

malnutrition (SAM). This may be as a result of poor feeding and improper feeding, poor personal hygiene by mother, poor sanitation, changes in frequency of breast feeding, dietary changes (in baby or breastfeeding mother), improper food preparation, digestive disorders, stomach condition and microbial infections of contaminated water and food.This could also lead to morbidity and mortality from communicable diseases (CD-WGE, 2005). Again, the condition is also responsible for over two million under-five preventable deaths each year (Black *et al*., 2008). Acute child hood malnutrition affects about a tenth of the world's children under 5 years of age, and contributes to 50–60% of all child deaths (Black *et al*.,2003) particularly those living in circumstances of extreme poverty in the developing world (Black *et al*.,2008)

The number of moderate acute malnutrition (MOAM) and mild acute malnutrition (MAM) recorded in this present study were 18.2% and 8.8% respectively. Children suffering from MOAM and MAM usually present with relatively mild complication compared with those suffering from SAM.

The mid upper arms circumference (MUAC) below 13.5cm which is adequate nutrition (AN) may be due to lack of inadequate and imbalanced consumption of nutrients or they cannot fully utilize the food they eat due to illness. Such patients rarely require admission, and recovery from infection takes a relatively short time similar work is shown in (Manary and Sandige, 2008).Malnutrition can increase the probability of secondary infection occurring thus modifying both disease pathogenesis and prognosis (Borelle *et al*., 2004).

5.4 SEVERE ACUTE MALNUTRITION

Marasmus was pronounced among the children of all ages (table 4.4). Majority of the children had marasmus totaling 71.5%. Each age group showed the highest number of marasmus compared to kwashiorkor and marasmic kwashiorkor. This may result from poor infant and young child feeding practice, poor socio-economic background and nutritionally inadequate diets (Amsalu and Tigabu, 2008). This study is commensurate with a similar work in Nigeria were higher cases of marasmus were present among malnourished children (Hamidu *et al*., 2003).

5.5 SEX OF MALNUTRITION, <6 MONTHS AGE GROUP, CHILDREN WITH OEDEMA AND MUAC OF VARIOUS CATEGORIES

In table 4.5, it was observed in my studies, that male children were insignificantly more malnourished than female children. This agrees with a similar work conducted among refugee children in Sudan (Mamoun *et al*., 2005). The relationship betweensex and acute malnutrition may also be due to young style of living and poor or less attention by parents.

5.6 SEX AND TERMS OF SEVERE ACUTE MALNUTRITION

In table 4.6 male children were again more malnourished than female children because female children develop faster immunity or immune system than males, therefore males are at risk of a negative balance, including lack of vitamin A and zinc, this might increase the risk of marasmus, kwashiorkor and marasmic kwashiorkor in males than females and place the males as the weaker sex regarding infections.

Severe acute malnutrition (SAM) affects both acquired and innate host defense mechanisms (Schaibe and Kaufmann, 2007). This leads to increased susceptibility to infection, more frequent

and prolonged episodes, increased severity of disease (CD-WGE, 2005), reactivation of viral infections, and development of opportunistic infections (Cunningham-Rundles *et al*., 2005). In addition, severe acute malnutrition often masks symptoms and signs of infectious diseases making prompt clinical diagnosis and early treatment very difficult. This in turn, increases the morbidity and mortality from communicable diseases (CD-WGE, 2005).

Beyond the age of 6 months, breast milk alone is no longer sufficient to meet the nutritional demands of the growing infant calling for introduction of complementary foods. However, introduction of complementary foods that is often nutritionally inadequate and microbiologically unsafe increasing the risks of multiple nutrient deficiencies (Kimmons *et al*., 2005).

5.7 AGE AND SEX DISTRIBUTION OF BACTERIAL ISOLATES FROM BLOOD OF MALNOURISHED CHILDREN

The predominantbacterialisolate from the blood of the malnourished children was*Staphylococcus aureus* (34.5%) (Table 4.7).This is in line with earlier studies by (Duncan et al., 2000) *who* opinesthat vitamin A deficiency is possibly a contributory factor. Another study by (Wiedermannet al., 1996) suggested that vitamin A deficiency predisposes to *Staphylococcus aureus* through phagocyte dysfunction and decreased complement activity.

The age group ≤11in table 4.7 had morebacterial isolates (37.9%) compared to the other age groups, this may attributed to the immaturity of the immune system.This is in agreement with (Mohan and Brocklehurst, 2003) who stated that the immaturity of the immune system, especially phagocytic and humoral immunity predisposes children to an increased incidence of sepsis caused by bacteria. Bacteremia are potentially life-threatening andrequire rapid

identification and also antibiotic susceptibility testingof the causative agent in order to facilitate specific antimicrobialtherapy (Berit *et al*., 2006)

Escherichia coli, Klebsiella spp, Proteus spp,Pseudomonas sppStaphylococcus aureus and *Streptococcus spp* were isolated from the blood of malnourished children in table 4.7. Despite advances in anti- microbial therapy and supportive care, bacteremia continues to be a major cause of morbidity and mortality among children.

In developing countries, more than 14 million deaths of children less than five years of age (UNICEF, 1994) occur during childhood, with infections accounting for up to 70% of total mortality for this age group (Mylotte and Tayara, 2001). Around the world is estimated 10 million children under theage of 5 years die each year, the vast majority (90%) in a mere42 countries. Of the major causes of death among children, are infections such as newborn sepsis (Jones *et al*., 2003). Organism are like *E. coli*, *Klebsiellaspp*., *Staphylococcus aureus*, Coagulase negative *Staphylococci* (CoNS), *Pseudomonasspp*., *Salmonella spp*.and *Acinetobacterspp*are potential pathogens in bacteraemia because of their frequent isolation and multi-drug resistance which has reached worrying levels (Castagnola *et al*., 2005).

5.8 BACTERIA ISOLATE FROM BLOOD AND TERMS OF SEVERE ACUTE MALNUTRITION

There was 65.5% bacterial infections in marasmus malnutrition, but the bacterial isolates that were found in marasmus were *Staphylococcus aureus* (36.8%), followed by *Klebsiella spp* (31.6%)(Table 4.8). The relatively high incidence of bacterial infections in the paediatric unit could be due to cross infection which may be controlled to some extent by hygienic measures. The immune system of infants is not completely developed and as a result, they are at an increased risk of suffering from neonatal sepsis... The infecting agents can enter the blood stream

through open cuts on skin and then move on to the organs such as lungs, liver, kidneys and intestines. Once they enter the body, they spread from one part to another using blood as a medium. Blood infection could sometimes be internal in nature.Acute diarrhoea and pneumonia occur most frequently during the first 2–3 years of life when immunocompetence is impaired and when children are first being exposed to pathogens. Infection can suppress appetite and directly affect nutrient metabolism, leading to poor nutrient utilization (Bloss *et al*., 2004).

Infection reported to cause loss of critical body stores of protein, energy, minerals and vitamins. During an immune response, energy expenditure increases at the same time that the infected host experiences a decrease in nutrient intake (Cunning- Rundles *et al*., 2005) .during an infection, a negative nitrogen balance occurs after fever induction and then it increases and persists for days to weeks after the febrile phase. Additionally, negative nitrogen balance appears to correlate with net loss in body weight; both conditions are the result of reduced food intake and infection induced-increased nitrogen excretion (Philips *et al*., 2004)

5.9 ANTIBIOTIC RESISTANCE FROM BLOOD ISOLATES INMALNOURISHED CHILDREN

In table 4.10, *Staphylococcus aureus (*80%) *proteus (*87%) *Escherichia coli* (100%) *and pseudomonas* (100%) were resistance to Cotrimoxazole Tetracycline, gentamicinand Ampicillin in the blood of malnourished children.

The pattern of organisms causing bacteraemia varies from place to place and can change in the same place over a period of time. This is due to the changing pattern of antibiotic use and

changes in life style. These organisms have developed multi-drug resistance over the last two decades (Maryam *et al*., 2001) due to indiscriminate use of antibiotics, over the counter sale of antibiotics, lack of legislation to control their use and ineffective infection control in maternity services (Rehman *et al*., 2001).

Early treatment and appropriate use of antibiotics would minimize the risk of severe morbidity and mortality in bacteraemia, and reduce the emergence of multi-drug resistant organisms in intensive care units by rational antibiotic use. For the success of early empiric treatment, periodic review of cases to assess any changing trends in the infecting organisms and their antimicrobial susceptibility is important.

Few epidemiological studies of bacteraemia undertaken in the Middle East and in other developing countries (Dawodu *et al*., 1997) have shown important differences in the pattern of antibiotic susceptibility of genes compared with studies in patho-European and American countries (Elward and Frase, 2006). The emergence of antimicrobial resistance is recognized as amajor contributor to excess morbidity and healthcare costs indeveloped countries.

5.11 AGE AND SEX OF BACTERIA ISOLATES FROM URINEOF MALNOURISHED CHILDEN

Klebsiella spp was the predominant bacterial isolated from the urine of malnourished children with an estimated prevalence of 38.8%, proteus and pseudomonas recording 16.3% prevalence. This is in direct contrast with earlier reports by Nicolle 2008, who reported E. coli as the main cause of urinary tract infection in children with about 80 - 85% prevalence rates. The differences in the findings could be attribute to differnces in methologies used and geographical locations.

There was 32(65.3%) of bacterial infection in males than 17(34.7%) in females.This may be due to the fact that males always play with contaminated objects, water and foods and swallowed them while females do not the go to play as such.The distribution and pattern of UTI varies with age, gender, ethnicity, circumcision in boys, and presence of congenital malformations. During neonatal period and early infancy malesare more affected, probably because of anatomical abnormalities and prepuce colonization (Lopez *et al*., 2007).

5.12 ANTIBIOTIC RESISTANCE FROM URINE BACTERIAL ISOLATES

From Table 4.17, virtually all the bacteria isolated in the urine of the malnourished children were resisstant to one antibiotic or the other. Despite the availability of newer antibiotics, emerging antimicrobial resistance has become an increasingproblem in many pathogens throughout the world (Stover *et al.,* 2001). Forpracticing physicians, clinical microbiologists and public healthofficials, knowledge of local antimicrobial resistance patternsis essential to guide empirical and pathogen-specific therapy. This information is also critical for optimal decisions regardinghospital formulation of infection control policies, for the rationalformulation of public healthcare policies, and national andinternational research agendas in that area. Unfortunately, data regarding endemic antimicrobial resistance are unavailablein many parts of the world, especially from areas where over-the-counterantibiotic use is common (Stover *et al.*, 2001).

CHAPTER SIX

6.0 CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSION

From the study, many of the patients that were affected with both malnutrition and the bacteria were males, with the age group ≤11months children dominated for bacterial infection and the age group 12-23 months dominated for malnutrition.

29/198 representing 14.6% were positive blood cultures, children whose parents did not sign consent form for their blood sample to be taken recorded 2/200 representing 1%.Out of total 29 cases of culture proven bacteraemia, the isolates were *Staphylococcus* (34.5%), *Streptococcus* (6.9%), *Klebsiella* (27.6%), *E.coli* ((10.3%), *Pseudonomas* (10.3%) and *Proteus (*10.3%). Regarding antibiotic resistance pattern; E.coli was 100% resistant to ampicillin and gentamicin, 66.7% to ciprofloxacin,87.5% of klebsiella was resistant to cotrimoxazole, ciprofloxacin and amikacin, and 50% to levofloxacin, staphylococcus aureus was 80% resistant to ampicillin and cotrimoxazole, 90% to gentamicin and 10% cephalexin.

49/180 representing 27.2% of positive urine cultures and children who were not able to produce urine sample recorded 20/200 representing 10%. Out of total 49 cases of culture proven UTI, the isolates were as follow; klebsiella (38.8%), proteus (16.3%), pseudomonas (16.3%), E.coli (10.2%), citrobacter (8.2%), S.aureus (6.1%), enterobacter (2.0%) and enterococcus (2.0%).

88% klebsiella was resistant to gentamicin, 70% to levofloxacin 30% to norvofloxacin, 21.1% to cotrimoxazole and 10.5% to ampicillin. Proteus was 100% resistant to gentamicin, 75% to cephalexin, 62.5% to levofloxacin, and 12.5% to ampicillin, cotrimoxazole and tetracycline.

3/199 representing 1.5% of positive stool cultures, children who could not produce stool sample were 1/200(0.5%), whislt169/198 (85.4%), 131/180 (72.8%) and 196 /199 (98.5%) were tested negative for blood, urine and stool respectively. 100% *Shigella* while was resistant to gentamicin, Amikacin and Levofoxacin, and 33% to cotrimoxazole and ampicillin.

The age group 12-23 months showed a significant statistical difference ($p < 0.05$) among Severe Acute Malnutrition (SAM), Moderate Acute Malnutrition (MOAM), Mild Acute Malnutrition (MAM) and Adequate Nutrition. Severe acute malnutrition (SAM) was seen to be higher among age group zero (0)-five years old children admitted to the Tamale Teaching Hospital. Almost all the children suffered from multi complaints of diarrhoea, fever and vomiting. More than half of these malnourished children admitted were marasmus. When compared, the isolates in under-nourished children to that of the isolates in the nourished children, shows that bacterial infection has contributed to the high prevalence of malnutrition among children 0-5 years admitted to the Tamale Teaching Hospital of Ghana.

6.2 RECOMMENDATIONS

- Adequate nutrition (including breastfeeding and zinc intake) must be promoted.
- Raising immunization rates among children must be raised.
- Mothers must ensure that children are well cleaned when they defaecate.
- The importants of hand washing in reducing the incidence of diarrhoea must be emphasised.Therefore mothers must be encourage to wash their hands with soap before feeding children or after going to toilet.
- Hands of children must be cleaned after playing.
- Efforts must be made to keep the food ofchildren from getting contaminated.

- Personal hygiene should be practiced among both mothers and children.
- Good environmental hygienic practice help in reducing diseases, must be encouraged.

REFERENCES

1 Adegbola, R.A.; Falade, A.G.; Sam, B.E.; Aidoo, M.; Baldeh, I.; Hazlett, D.; Whittle, H.; Greenwood, B.M.; Mulholland, E.K. (1994): The etiology of pneumonia in malnourished and well-nourished Gambian children. *Pediatric. Infect. Dis. J, 13*, 975-982.

2 Adejuyigbe EA, Adeodu OO, Ako-Nai KA, Taiwo O and Owa JA.(2001).Septicaemia in high risk neonates at a teaching hospital in Ile-Ife, Nigeria. *East African Med J; 78(10):540-543*

3Adler, B., & de la Peña Moctezuma, A. (2010). Leptospira and leptospirosis. *Veterinary microbiol*. *140*(3): 287-296.

4 Ahmed, T.; Haque, R.; Mansur, A.; Ahmed, S.; Petri, W.A., Jr.; Cravioto, A.(2009): Use of metagenomics to understand the genetic basis of malnutrition. *Nutr.Rev. 67*, S201-S206

5 Akuyam, S.A. (2007**).**Review of some metabolic changes in protein-energy malnutrition. *Niger. Postgrad. Med. J: 14*, 155-162.

6 Akhtar R, Haider A and Raza MR (2005) Neonatal sepsis in NICU: Bacterial isolates and theirsensitivity pattern.*J Surg Pak (Intern); 10(4):18-21*.

7 Amdekar, S; Singh, V, Singh, DD (2011). "Probiotic therapy: immunomodulating approach toward urinary tract infection. *Current micro.* 63 (5): 484–90.

8 AmandeepS. (2010)."Pediatric Emergency Medicine Practice Acute Gastroenteritis — an Update". *Emerg. Med. Practice*.**7** (7).

9 American Society for Microbiology (1995) Report of the ASM Task Force on Antibiotic Resistance.*Antimicrob.Agents.Ch. 1: 1–23.*

10 Amsalu S, Tigabu Z. (2008): Risk factors for severe acute malnutrition in children under the age of five: A case-control study. Ethiop J Health Dev.22:21–25.

11 Asindi AA, Ibia EO, Udo JJ (1991) Mortality pattern among Nigerian children in the 1980s**.** *J Trop Med Hyg, 94:152-155.*

12 Bachou H, Tylleskar T, Kaddu-Mulindwa DH, Tumwine JK. (2006): Bacteraemia among severely malnourished children infected and uninfected with the human immunosdeficiency virus-1 in Kampala, Uganda. BMC Infect Dis. 6:160.

13Balaban, N., & Rasooly, A. (2000). Staphylococcal enterotoxins. *Int. journal of food microbiol61* (1): 1-10.

14 Bauer R, Kogan BA (2008). New developments in the diagnosis and management of pediatricUTIs.*Urol. Clinics of North Am.*35:47–58.

15 Beck MD, Munoz JA, Scrimshaw NS. (1957): Studies on diarrheal diseases in Central America. I. Preliminary findings on cultural surveys of normal population groups in Guatemala. Am J Trop Med Hyg 6:62−71?

16 Beetz R. (2006) May we go on with antibacterial prophylaxis for urinary tract infections? Pediatric Nephrology.21:5–13.

17 Bearman GM, Wenzel RP. (2005): Bacteremias: a leading cause of death. Arch Med Res. 36:646–659.

18 Benguigui, Y.; Stein, F. (2006): Integrated management of childhood illness: An emphasis on the management of infectious diseases. *Sem. Pediatr. Infect. Dis.17,* 80-98.

19 Berit EC, Maria PS, Jörg G, Salima M, Martin K, Oleg K.(2006) Identification and characterization of bacterial pathogens causing bloodstream infec- tions by DNA microarray. *J. Clin. Microbiol. 44(7): 2389-397.*

20 Berkowitz, F.E. (1992) Infections in children with severe protein-energy malnutrition.*Pediatr. Infect. Dis. J.11*, 750-759.

21 Berman, S. (1991): Epidemiology of acute respiratory infections in children of developing countries. *Rev. Infect. Dis. 13*, S454-S462.

22 6Bhat, RG; Katy, TA, Place, FC (2011). "Pediatric urinary tract infections."*Emerg.Med. clinic of north America*.29 (3): 637–53.

23 Black RE, Morris SS, Bryce J. (2003): Where and why are 10 million children dying every year? Lancet.361:2226–2234.

24Bloss, E.; Wainaina, F.; Bailey, R.C. (2004): Prevalence and predictors of underweight, stunting, and wasting among children aged 5 and under in Western Kenya, Bailey. *J. Trop. Pediatr. 50,* 260-270.

25 Bogaert, D.; de Groot, R.; Hermans, P.W.M. (2004) *Streptococcus pneumoniae* colonisation: The key to pneumococcal disease. *Lancet Infect. Dis. 4*, 144-154.

26 Borelli, P.; Blatt, S.L.; Rogero, M.M.; Fock, R.A (2004):.Haematological alterations in protein malnutrition.*Rev. Bras.ematol. emoter.26*, 49-56.

27 Cashat-Cruz, M.; Morales-Aguirre, J.J.; Mendoza-Azpiri, M. (2005**)** Respiratory tract infections in children in developing countries.*Semin.Pediatr. Infect. Dis. 16*, 84-92.

28 Castagnola E, Caviglia I, Pistorio A, Fioredda F, Micalizzi C, Viscoli C.(2005): Blood stream infections and invasive mycoses in children undergoing acute leukaemia treatment. *Eur. J. Cancer. 41(10): 1439 – 445.*

29 CD-WGE. (2005): Communicable diseases and severe food shortage situations. World Health Organization Communicable Diseases Working Group on Emergencies.

30 Chisti, M.J.; Tebruegge, M.; La Vincente, S.; Graham, S.M.; Duke, T. (2009**):** Pneumonia in severely malnourished children in developing countries—mortality risk, a etiology and validity of WHO clinical signs: A systematic review. *Trop. Med. Int. Health 14*, 1173-1189.

31 Clark CJ, Kennedy WA, Shortliffe LD (2010) Urinary tract infection in children: when to worry.*Urol. Clinics of North Am*. 37:229–241.

32 Charles, RC; Ryan, ET (2011). "Cholera in the 21st century."*Current Opinion in Infect. Dis*.24 (5): 472–7.

33 Clinical and Laboratory Standard Institute (2005): Performance standards for antimicrobial disk susceptibility tests. NCCLS documents M 100 – SIS, 940 West Valley Road. Wayne, PA, USA, 19087

34 CSA. (2005): Ethiopia Demographic and Health Survey. Addis Ababa, Ethiopia.

35 Cunha, A.L. (2000): Relationship between acute respiratory infection and malnutrition in children under 5 years of age. *Acta Pediatr.89*, 608-609.

36 Cunningham-Rundles S, McNeeley DF, Moon A. (2005): Mechanisms of nutrient modulation of the immune response. J Allergy Clin Immunol.115:1119–1128.

37 Dawodu A, Alumran K, Twum-Danso K. *(1997):* A case control study of neonatal sepsis: Experience from Saudi Arabia. *J. Trop. Pediatri. 43: 84–88.*

38 DeCamp LR, Byerley JS, Doshi N, Steiner MJ (2008). "Use of antiemetic agents in acute gastroenteritis: a systematic review and meta-analysis". Arch Pediatr Adolesc Med162 (9): 858–65.

39 Desselberger U, Huppertz HI (2011). "Immune responses to rotavirus infection and vaccination and associated correlates of protection". J. Infect .Dis.203 (2): 188–95.

40 De Onis, M.; Monteiro, C.; Akré, J.; Clugston, G. (1993): The worldwide magnitude of protein—energy malnutrition: An overview from the WHO global database on child growth. *Bull. World Health Organ.71*, 703-712.

41Donnenberg, M. S., Donohue-Rolfe, A., & Keusch, G. T. (1989). Epithelial cell invasion: an overlooked property of enteropathogenic Escherichia coli (EPEC) associated with the EPEC adherence factor. *J. Infect. Dis. 160* (3): 452-459.

42 Eckardt AJ, Baumgart DC (2011). "Viral gastroenteritis in adults".Recent Patents on Anti-infective Drug Discovery6 (1): 54–63.

43 Effa, EE; Lassi, ZS, Critchley, JA, Garner, P, Sinclair, D, Olliaro, PL, Bhutta, ZA (2011). "Fluoroquinolones for treating typhoid and paratyphoid fever (enteric fever).” In Bhutta, Zulfiqar A. Cochrane Database of Systematic Reviews (10): CD004530.

44 Escobedo, AA; Almirall, P, Robertson, LJ, Franco, RM, Hanevik, K, Mørch, K, Cimerman, S (2010). "Giardiasis: the ever-present threat of a neglected disease.”Infectious disorders drug targets10 (5): 329–48.

45 Elward AM, Fraser VJ. (2006). Risk factors for nosocomial primary bloodstream infection in pediatric intensive care unit patients: a 2-year prospective cohort study. Infect. Cont. Hosp. Ep. 27(6): 553-60.

46 Elliott, EJ (2007). "Acute gastroenteritis in children."BMJ (Clinical research ed.)334 (7583): 35–40.

47 FAO (2008). Soaring Food Prices: Facts, Perspectives, Impacts and Actions required," background paper prepared for the High-Level Conference on World Food Security: The Challenges of Climate Change and Bio energy, Rome, 3-5.

48 Fabre R, Merens A, Lefebvre F, Epifanoff G, Cerutti F, Pupin H (2010).Susceptibility to antibiotics of Escherichia coli isolated from community-acquiredurinary tract infections. Méd Mal Infect. 40:555–559.

49 Federal Ministry of Health of Ethiopia (FMoH) (2007).Author Protocol for the management of severe acute malnutrition. Addis Ababa: Ethiop:. pp. 1–88.

50 Fewtrell L, Kaufmann RB, Kay D, Enanoria W, Haller L, Colford JM Jr. (2005): Water, sanitation, and hygiene interventions to reduce diarrhoea in less developed countries: a systematic review and meta-analysis. Lancet Infect Dis.5 (1): 42-52.

51 Furuta, T.; El-Omar, E.M.; Xiao, F.; Shirai, N.; Takashima, M.; Sugimurra, H. **(**2002**):** Interleukin 1beta polymorphisms increase risk of hypochlorhydria and atrophic gastritis and reduce risk of duodenal ulcer recurrence in Japan. *Gastroenterology. 123*, 92-105.

52 Galanis, E (2007). "Campylobacter and bacterial gastroenteritis.".CMAJ: *Canad. Med. Asso...***177** (6): 570–1.

53 Garcia FJ, Nager AL.(2002) Jaundice as an early diagnostic sign of urinary tract infection in infancy. Pediatrics 109(5):846–51.

54 Getaneh T, Assefa A, Tadesse Z (1998). Protein-energy malnutrition in urban children: prevalence and determinants. Ethiop. Med., J., 36: 153–166.

55 Gómez, F.; Ramos-Galván, R.; Frenk, S.; Cravioto, J.; Chávez, R.; Vázquez, J (1956):. Mortality in second and third degree malnutrition.*J. Trop. Pediatr.2*, 77-83

56 Graham, N.M.H. (1990): The epidemiology of acute respiratory infections in children and adults: A global perspective. *Epidemiol. Rev. 12*, 149-178.

57 Gonzales, ML; Dans, LF, Martinez, EG (2009). "Antiamoebic drugs for treating amoebic colitis." In Gonzales, Maria Liza M. Cochrane Database of Systematic Reviews (2): CD006085.

58Hansen, W., & Yourassowsky, E. (1984). Detection of beta-glucuronidase in lactose-fermenting members of the family Enterobacteriaceae and its presence in bacterial urine cultures. *J. clinical microbiol20* (6): 1177-1179.

59 HamiduJ.L., Salami H.A, EkanemA.U, Hamman, L. (2003): Prevalence of protein—energy malnutrition In Maiduguri, Nigeria, *Afric. J. of Biomed. Research*, Vol. 6:123 – 127

60 Hempel, S; Newberry, SJ; Maher, AR; Wang, Z; Miles, JN; Shanman, R; Johnsen, B; Shekelle, PG (2012). "Probiotics for the prevention and treatment of antibiotic-associated diarrhoea: a systematic review and meta-analysis."JAMA: the J. of the Am.Med. Asso. 307 (18): 1959–69.

61 Jagannath, VA; Fedorowicz, Z; Sud, V; Verma, AK; Hajebrahimi, S (2012). "Routine neonatal circumcision for the prevention of urinary tract infections in infancy."*Cochrane database of systematic reviews (Online)* 11: CD009129PMID 23152269.

62 Jakobsson B, Esbjorner E, Hansson S. (1999).Minimum incidence and diagnostic rate of first urinary tract infection.Pediatrics 104:222-6.

63 Jawetz; E., L. Melnick; J.L and Edward .A. Adelberg; A.A. (2010).Medical Microbiology 25 Edition, Newgen Publishing and Imaging Services.United State of America.PP 1-814.

64 Jones G, Steketee RW, Black RE, Bhutta ZA, Morris SS, Bellagio Child Survival Study Group (2003). "How Many Child Deaths Can We Prevent This Year?" Lancet. 362: 65–71.

65 Kimmons JE, Dewey KG, Haque E, Chakraborty J, Osendarp SJ, Brown KH (2005). Low nutrient intakes among infants in rural Bangladesh are attributable to low intake and micronutrient density of complementary foods. J. Nutr., 135: 444-451.

66 Lane, DR and Takhar, SS (2011): "Diagnosis and management of urinary tract infection and pyelonephritis."*Emerg. Med. Clinics. North. Am.* 29 (3): 539–52.

67 Lawrence, DT; Dobmeier, SG; Bechtel, LK; Holstege, CP (2007). "Food poisoning."Emerg. Med.clinics. North Am. 25 (2): 357–73.

68 Lin KY, Chiu NT, Chen MJ, Lai CH, Huang JJ, Wang YT, Chiou YY.(2003):Acutepyelonephritis and sequelae of renal scar in pediatric first febrile urinary tract infection.Pediatr.Nephrol.18:362-365.

69 Lin DS, Huang SH, Lin CC, Tung YC, Huang TT, Chiu NC.(2000): Urinary tract infectionin febrile infants younger than eight weeks of Age. Pediatrics.105 (2):E20.

70 López Sastre J.B., Aparicio A.R., Coto Cotallo G.D., Fernández Colomer B, CrespoHernández M.(2007): Urinary tract infection in the newborn: clinical and radio imagingstudies. Pediatric Nephrology.22:1735–1741.

71 Lozano, R (2012). "Global and regional mortality from 235 causes of death for 20 age groups: A systematic analysis for the Global Burden of Disease Study.".Lancet380 (9859): 2095–128.

Mamoun, N., S. Homedia, M.Mabyou and H.M. Ahmed Muntasir (2005):.Prevalence, Types and Risk Factors for Malnutrition in Displaced Sudanese Children. Am. J. Infect. Dis.1 (2): 84-86.

72 Manary MJ and Sandige HL (2008): Management of acute moderate and severe childhood malnutrition.*BMJ*, 337:2180.

73 Mandell, Gerald L.; Bennett, John E.; Dolin, Raphael (2004).Mandell's Principles and Practices of Infection Diseases (6th ed.). Churchill Livingstone. ISBN 0-443-06643-4.

74 MaryamW, Laeeq A, Maqbool S. (2001): Neonatal sepsis spectrum of antibiotic resistance. Proceedings of 10th Annual National Pediatric Conference. 57.

75 Mackway-Jones, Kevin (2007). "Does yogurt decrease acute diarrhoeal symptoms in children with acute gastroenteritis?".BestBets.

76 Mehta S, Goldman RD (2006). "Ondansetron for acute gastroenteritis in children".Can Fam Physician52 (11): 1397–8.

77 Meléndez, G. (2011): Fundación Mexicana para la Salud-FUNSALUD. .mx/mexico/2010/198618/6/afecta-desnutricion-a-18-millones-de-mexicanos-menores-de-cinco-anos.htm.

78 Meloni, A; Locci, D, Frau, G, Masia, G, Nurchi, AM, Coppola, RC (2011). "Epidemiology and prevention of rotavirus infection: an underestimated issue?".The journal of maternal-fetal & neonatal medicine: the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians24 (Suppl 2): 48–51.

79Miller, V. L., Farmer, J. J., Hill, W. E., & Falkow, S. (1989). The ail locus is found uniquely in Yersinia enterocolitica serotypes commonly associated with disease. *Infection and immunity*, *57*(1), 121-131.

80 Monica Cheesbrough (2006) District Laboratory Practice in Tropical Countries, Part 2 Second Edition. Published in the United States of America by Cambridge University Press, New York.

81 Mohan P and Brocklehurst P. (2003): Granulocyte transfusions for neonates with confirmed orsuspected sepsis and neutropaenia; 1-26

82 Mondal, D.; Haque, R.; Sack, B.; Kirkpatrick, B.; Petri, W. (2009): Attribution of malnutrition to cause-specific diarrhoeal illness: Evidence from a prospective study of preschool children in Mirpur, Dhaka, Bangladesh. *Am. J. Trop. Med. Hyg.80*, 824-826

83 Muniz, P.T.; Ferreira, M.U.; Ferreira, C.S.; Conde, W.L.; Monteiro, C.A. (2002) Intestinal parasitic infections in young children in Sao Paulo, Brazil: Prevalences, temporal trends and associations with physical growth. *Ann. Trop. Med. Parasitol. 96*, 503-512

84 Mylotte JM, Tayara A. Blood cultur- es: clinical aspects and controversies. Eur. J. (2001): *Microbiol. Infect. Dis. 9: 157–63.*

85 Nantanda, R.; Hildenwall, H.; Peterson, S.; Kaddu-Mulindwa, D.; Kalyesubula, I.; Tumwine, J.K.(2008):Bacterial aetiology and outcome in children with severe pneumonia in Uganda. *Ann. Trop. Paediatr.28*, 253-260.

86 National Health Service (2013). Causes of malnutrition in developing nations.UK.

87 Nyachuba, DG (2010). "Foodborne illness: is it on the rise?"Nutrition Reviews68 (5): 257–69.

88 Nicolle LE (2008). "Uncomplicated urinary tract infection in adults including uncomplicated pyelonephritis".*Urol Clin North Am*35 (1): 1–12.

89 Onyango AW (2003). Dietary diversity, child nutrition and health in contemporary African communities. Rev. Comp. Biochem. Physiol., 136: 61–69.

90 Peterson, K. M. (2002). Expression of Vibrio cholerae virulence genes in response to environmental signals. *Current issues in intestinal microbial. 3*(2): 29-38.

91 Phillips, R.S.; Enwonwu, C.O.; Okolo, S.; Hassan, A. (2004): Metabolic effects of acute measles in chronically malnourished Nigerian children. *J. Nutr. Biochem.15*, 281-288.

92 Population and Housing Census (2010).National Population and Housing Census of Ghana.

93 Powanda, M.C.; Beisel, W.R (2003): Metabolic effects of infection on protein and energy status. J. Nutr. 133, 322S-327S.

94 Qadri, F.; Saha, A.; Ahmed, T.; Al Tarique, A.; Begum, Y.A.; Svennerholm, A.M. (2007): Disease burden due to *enterotoxigenic Escherichia coli* in the first 2 years of life in an urban community in Bangladesh. *Infect. Immun. 75*, 3961-3968.

95 Quigley R. (2009): Diagnosis of urinary tract infections in children.Current Opinion in Pediatrics. 21:194–198.

96 -Rehman S. (2001) Roghani MT, Ullah R.A survey of perinatal care facilities in Pakkistan of 10th Annual Pediatric Conference 34.

97 Rudan, I.; Boschi-Pinto, C.; Biloglav, Z.; Mulholland, K.; Campbell, H.(2008) Epidemiology and etiology of childhood pneumonia. *Bull. World Health Organ.5,* 408-416.

98 Salo, J; Ikäheimo, R, Tapiainen, T, Uhari, M (2011 Nov). "Childhood urinary tract infections as a cause of chronic kidney disease."*Pediatrics*128 (5): 840–7.

99 Schaible UE, Kaufmann SH. (2007): Malnutrition and infection: complex mechanisms and global impacts. PLoS Med.4:115.

100 Schoen EJ, Colby CJ, Ray GT. (2000) Newborn circumcision decreases incidence and costs of urinary tract infections during the first year of life. Pediatrics 105:789-93.

101 Schaible, U. E. and H. Stefan (2007). "Malnutrition and infection: complex mechanisms and global impacts." PLoS medicine4(5): e115.

102 Scrimshaw NS, Behar M, Viteri F, Arroyave G, Tejada C. (1957): Epidemiology and prevention of severe protein malnutrition (kwashiorkor) in Central America. *Am J Pub Health* 47:53−62.

103 Scrimshaw NS, Taylor CE, and Gordon JE. (1968): Interactions of nutrition and infection. World Health Organization Geneva.

Shelobolina, E. S., Sullivan, S. A., O'Neill, K. R., Nevin, K. P., & Lovley, D. R. (2004). Isolation, characterization, and U (VI)-reducing potential of a facultatively anaerobic, acid-resistant Bacterium from Low-pH, nitrate-and U (VI)-contaminated subsurface sediment and description of Salmonella subterranea sp. *Applied and environ.Microbiol. 70*(5): 2959-2965.

104 Sheth M, Dwivedi R (2006).Complementary foods associa-ted diarrhoea.Indian J. Pediatr., 73: 61-64.

105 Sheth M, Patel J, Sharma S, Seshadri S (2000). Hazard analysis and critical control points of weaning foods. Indian J. Pediatr., 67: 405-410.

106 Shann, F. (1986): Etiology of severe pneumonia in children in developing countries. *Pediatr. Infect. Dis. J.5*, 247-252.

107 Smellie JM, Hodson CJ, Edwards D, Normand ICS.(1964) Clinical and radiological featuresof urinary infection in childhood.BMJ 5419:1222–6.

108 Sermin A. Saadeh1 and Tej K. Mattoo (2012). Managing urinary tract infections. Int J Pediatr. 943653.

109 Stover BH, Shulman ST, Bratcher DF, Brady MT, Levine GL, Jarvis.(2001) Nosocomial infection rates in US children's hospitals' neonatal and pediatric intensive care units. *Am. J. Infect. Control*.29 (3): 152-57

110Singh, Amandeep (2010). "Pediatric Emergency Medicine Practice Acute Gastroenteritis — an Update". *Emerg. Med. Practice*.7 (7).

111 Svanborg, C., and Godaly G. (1997): Bacterial virulence in urinary tract infection. *Infect. Dis.Clin. North Am.*11:513-529.

112 Szajewska, H; Dziechciarz, P (2010). "Gastrointestinal infections in the pediatric population."Current Opinion in Gastroenterology26 (1): 36–44.

113 Telmesani, AM (2010). "Oral rehydration salts, zinc supplement and rota virus vaccine in the management of childhood acute diarrhoea."Journal of family and community medicine17 (2): 79–82.

114Tewari, G., & Juneja, V. (Eds.). (2008). *Advances in thermal and non-thermal food preservation*. John Wiley & Sons.

115 Thiem VD, Sethabutr O et al., (2004):Detection of Shigellae by a PCR Assay Targeting the ipaH Gene Suggests Increased Prevalence of Shigellosis in Nha Trang, Vietnam. J Clin Microbiol. 42 (5): 2031-35.

116 UNICEF (2009b): Overview of breastfeeding patterns.

117 UNICEF (1994) the state of the world's children. Oxford, Oxford University Press.

118 United Nations Children's Fund (2009).The State of the World's Children .Maternal and Neonatal Health. New York, USA: United Nations Children's Fund.

119 Walker, CL; Rudan, I; Liu, L; Nair, H; Theodoratou, E; Bhutta, ZA; O'Brien, KL; Campbell, H; Black, RE (2013). "Global burden of childhood pneumonia and diarrhoea."Lancet381 (9875): 1405–16.

120 Wapnir, R. (2000): Zinc deficiency, malnutrition and the gastrointestinal Tract. *J. Nutr.*, *130*, 1388S-1392S.

121 Warrell D.A., Cox T.M., Firth J.D., Benz E.J., ed. (2003). The Oxford Textbook of Medicine (4th ed.). Oxford University Press.

122 Weber MW, Carlin JB, Gatchalian S, Lehmann D, Muhe L, Mulholland EK, WHO Young Infants Study Group(2003): Predictors of neonatal sepsis in developing countries**.** *Pediatr Infect Dis J22 (8):711-7.*

123 Webb, A; Starr, M (2005). "Acute gastroenteritis in children."Australian family physician34 (4): 227–31.

124 WHO. (1981): Clinical management of acute respiratory infections in children a WHO memorandum. *Bull. World Health Organ.59,* 707-716

125 World Health Organization *Children* (2013).*Reducingchild mortality* .

126 WHO (1999): Management of severe malnutrition: a manual for physicians and other senior health workers. Geneva, World health organization.

127 WHO/UNICEF (2003). Global Strategy for Infant and Young Child Feeding.Geneva, World health organization.

128 Windle, H.J.; Kelleher, D.; Crabtree, J.E. (2007): Childhood *Helicobacter pylori* infection and growth impairment in developing countries: A vicious cycle? *Pediatrics, 119*, e754-e759.

129 Yimer G. (2000): Malnutrition among children in Southern Ethiopia: Levels and risk factors. Ethiop J Health Dev.14:283–292.

130Xiang, Z., Censini, S., Bayeli, P. F., Telford, J. L., Figura, N., Rappuoli, R., & Covacci, A. (1995). Analysis of expression of CagA and VacA virulence factors in 43 strains of Helicobacter pylori reveals that clinical isolates can be divided into two major types and that CagA is not necessary for expression of the vacuolating cytotoxin. *Infect.Immunity.63* (1): 94-98.

APPENDICES

APPENDIX 1

Bacterial infection in malnourished children form 1

Patient ID	

Mother/Caregiver's Socio-Demographic Characteristics

Name		Age/*yrs*		
Community				

Mother/caregivers Educational status

No formal education	*Primary*	*JHS*	*SHS/Voc/ Tech*	*Tertiary*

Mother/caregivers Occupation

House wife	Trader	Farmer	Civil servant	Seamstress
Other (specify)				

Household Size *(people eating from the same pot)*	
No of Siblings	

Child's Socio-Demographic and clinical data

Name		Age *(mths)*		Sex	*M*	*F*

Clinical presentations

Diarrhoea	*Yes*	*No*	Vomiting	*Yes*	*No*	

Others						
Medical Diagnosis						
Weight/*Kg*		Height/*cm*		MUAC/*cm*		
Malnutrition Types	*Marasmus*	*Kwashiorkor*	*Marasmus kwashiorkor*			

Laboratory samples

Blood sample ID	
Stool sample ID	
Urine sample ID	

ANTIBIOTIC GIVEN:

APPENDIX 2

2.1 MATERIALS USED FOR THE STUDY

2.2 Reagents

- Cystine lactose electrolyte-deficient (CLED)powder
- Distilled water
- selenite fusion powder
  sodium biselenite (Lp121).
- salmonella shigella powder
- brain heart infusion powder
- nutrient agar powder
- sterile sheep blood
- MacConkey powder
- crystal violet
- ammonium oxalate
- ethanol/methanol
- potassium iodide
- iodine
- Acetone alcohol
- neutral red or safranin
- Mueller Hinton powder
- peptone powder
- urea powder
- urea salt
- citrate powder
- triple sugar iron powder
- motility, indole,ornithine agar
- plasma
- spirit
- Kovac's reagent
- hydrogen peroxide

- immersion oil

- **2.3 Equipment**
- autoclave
- long and short tubes
- sterile wooden stick or a glass rod,
- a slide
- sterile calibrated wire loop
- containers
- bottles fitted with screw caps and small holes at the top
- plastic liners
- Petri dish
- microscope with 10×, 40×, and 100× objectives
- Gloves
- sharpener
- Bio-hazardous Waste Containers
- Cover slips (22mm × 22mm)
- carbon paper
- cottonwood
- staining rack

APPENDIX 3

3.1 CULTURE MEDIA PREPARATIONS

3.2 CYSTINE LACTOSE ELECTROLYTE-DEFICIENT AGAR (CLED)

36.2g of CLED was weighed and suspended in1000ml of distilled water. The solution was mixed and boiled to dissolve completely. The medium was sterilized by autoclaving at 121 degrees Celsius for 15minutes and mixed well before pouring 15mls each into sterile disposable Petri dishes and allowed to solidify and packed. The surface of the agar was always allowed to dry in an incubator each time before used. Cystine Lactose Electrolyte-Deficient agar is widely used by laboratories to isolate urinary pathogens because it gives consistent results and allows the growth of both gram positive and gram negative bacteria. The indicator in Cystine Lactose Electrolyte-Deficient is bromothymol blue and therefore lactose fermenting colonies appear yellow. The medium is electrolyte-deficient and this prevents the swarming of Proteus species.

3.2.1 SELENITE CYSTINE BROTH BASE PREPARATION FOR STOOL CULTURE

19g of selenite fusion powder was dissolved in 1000 ml of distilled water with 4g of sodium biselenite (Lp121). The solution was mixed and warmed. Finally, it was dispensed into containers with a depth of about 60mm and then sterilized in a boiling water bath or free flowing steam for 15minutes. This medium does not need autoclaving.

3.3 SALMONELLA SHIGELLA AGAR (SSA)

15g of salmonella shigella powder was dissolved in 250mls of distilled water and heated until all the particles completely disappeared to form a homogenous solution. 15mls of the medium was

poured into each sterile Petri dish and allowed to solidify. The agar was then packed and stored. The surface of the agar was always allowed to dry in an incubator each timebefore used. This is a differential medium which is based on the fermentation of lactose and subsequent absorption of neutral red by the bacteria colony.The gram positive bacteria and coliforms are usually inhibited by the bile salt, brilliant green and citrate.

Sodium thiosulfate and ferric citrate enable the detection of hydrogen sulfide production as evidenced by colonies with black centers, including some proteus strains. Occasionally few coliforms and other lactose fermenting organisms may grow through the inhibitors and develop into pink or red colonies.

3.4 PREPARATION OF BRAIN HEART INFUSION AGAR FOR BLOOD CULTURE

52.0g of brain heart infusion powder was suspended in 1 liter (1000mls) of distilled water. The medium was mixed and heated until it was completely dissolved. 15mls each of the broth was poured into bottles fitted with screw caps and small holes at the top. The holes of the screw caps were sealed with plastic liners. The broth was autoclaved at 121 degree Celsius for 15 minutes. The broth was allowed to cool at room temperature and then stored in a refrigerator for use.

3.5 PREPARATION OF BLOOD AGAR

2.8g of nutrient agar was weighed and suspended in 100ml of distilled water. The medium was mixed and then sterilize by autoclaving at 121 degree Celsius for 15 minutes. For the blood agar, the medium was allowed to cool to about 45 degree Celsius before sterile sheep blood was added to get blood agar. 15mls ofthe medium was poured into each Petri dish and allowed to settle or solidified and pack for storage. The surface of the gel was always dry before use.

3.6 PREPARATION OF CHOCOLATE AGAR

2.8g of nutrient agar was weighed and suspended in 100ml of distilled water. The medium was mixed and sterilized by autoclaving at 121 degree Celsius for 15minutes .For chocolate agar, as soon as the medium comes out of the autoclave, an amount of sterile sheep blood were added and mixed to get the chocolate agar .i.e. the burnt agar. 15mls of the medium was poured into each Petri dish and allowed to settle or solidify and pack for storage. The surface of the gel was always dried before used.

3.7 PREPARATION OF MAcCONKEY AGAR

52g of MacConkey powder was suspended in 1litre (1000ml) of distilled water, the solution was allowed to boil to dissolved completely.

The medium was then sterilized by autoclaving at 121 degree Celsius for 15minutes and mixed well before pouring 15mls each into steriledisposable Petri dish.The agar was allowed to solidify and packed for storage. The surface of the gel was always dried before used.

APPENDIX 4

4.1 PREPARATION OF GRAM STAIN

4.2 BASIC CRYSTAL VIOLET SOLUTION

20g of crystal violet was added to 9g of ammonium oxalate and these were then added to 95ml of absolute ethanol/methanol plus distilled water to make 1000ml.Staining an organism with Crystal violet (primary stain) depends on the ability of certain bacteria (gram positive bacteria) to retain a complex of crystal violet (purple dye) after a brief wash with alcohol or acetone alcohol.

4.3 LUGOL'S IODINE SOLUTION

20g of potassium iodide was added to10g of iodine and these were dissolved in 1000 ml distilled water to form Lugol's iodine solution. This iodine solution serves as a mordant increasing the interaction between the bacterial cell and the dye so that the dye is more tightly bound or the cell is more strongly stained.

4.4 ACETONE ALCOHOL SOLUTION

500mls of acetone was dissolved in (475mls of absolute ethanol or methanol and 25mls distilled water) to get acetone solution. Acetone alcohol (decolorizer) was used to wash away excess crystal violet and iodine so that the gram positive bacteria retain crystal violet-iodine complex whiles gram negative bacteria loosed their crystal violet –iodine complex and became colorless.

4.5 NEUTRAL RED OR SAFRANIN SOLUTION

0.1% of neutral red or safranin was used. Neutral red (the counter stain) is a basic dye different from the crystal violet .The counter stain stained the colourless gram negative bacteria pink but did not alter the dark purple colour of the gram positive bacteria .The end results is that gram positive bacteria stain deep purple and gram negative bacteria stain pinkish to red.

4.6 TRADITIONAL GRAM-STAIN TECHNIQUE

Smears of colony from the plate were heat fixed on a clean and dry slides.

The slides were placed on the staining rack and flooded with crystal violet and allowed to stand for 1 minute. The slides were then washed with clean water.

The slides were again flooded with Lugol's iodine solution and allowed stand for 1 minute after which they were rinsed with water.

Decolourization was done with acetone-alcohol for 15 to 30 seconds.

Precaution was taken to avoid exceeding the time for decolourization.

The decolourizer was added drop by drop until the crystal violet fails to wash from the slide and the slides was rinsed with water.

Counterstaining was done with neutral red or safranin for 1 minute.

The slides were subsequently given a final rinse with water and allow to air dry and examined microscopically.

First with the 40x objective lens to check the staining properties of the organism and to see the distribution of other materials.The final examination was done with the oil immersion objective lens to report the bacteria and cells.

APPENDIX 5

5.0 SENSITIVITY TEST MEDIA PREPARATION

Antimicrobial Susceptibility Testing was performed on all isolated pathogens using the Kirby-Bauer disk diffusion method and results interpreted according to the CLSI criteria.

5.1 MUELLER HINTON AGAR

38gms of Mueller Hinton powder was dissolved in sterile distilled water. The solution was then heated to dissolve completely. The solution was also sterilized by autoclaving at 121^0C for 15 minutes. It was then allowed to cool at room temperature and dispensed 15mls into each Petri dish. The agar was then allowed to solidify and cool before storage in the refrigerator. The surface of the gel was always dried before use. This agar is used for the culturing of Neisseria sp. and for determination of susceptibility of microorganisms to antibiotics.

5.2 PEPTONE WATER PREPARATION

15g of peptone powder was dissolved into 1 liter (1000mls) of distilled water. The solution was then mixed and dispensed into receptacles for use.

APPENDIX 6

BIOCHEMICAL TEST MEDIA PREPARATION

6.1 UREA MEDIA PREPARATION

5.5g of urea powder was dissolved in 250mls of distilled water and swirled to mix to get urea broth. The broth was then autoclaved at 121c for 15minutes to ensure proper sterilization. The broth was allowed to cool to 48 degree Celsius.6g of urea was added to 13.5mls of sterile distilled water to make urea base. The urea base was aseptically added to urea broth to get urea agar.

6.1.1 Urea Test

Inoculation was done heavily over the entire slope surface and incubated at 37ºc and examined after 4h and after overnight incubation. No tube was reported negative until after 48hours incubation. Urease-positive cultures changed the colour of the indicator from light yellow to pink. Bacteria, particularly those growing naturally in an environment exposed to urine, may decompose urea by means of the enzyme ureases. The occurrence of this enzyme can be tested for by growing the organism in the presence of urea and testing for alkali (NH_3) production by means of a suitable PH indicator.

6.2 CITRATE MEDIA PREPARATION USING SIMMON"S CITRATE AGAR

24.2g of the citrate powder was suspended in 1 litre of purified water and mixed well thoroughly.The solution was heated and boiled with frequent agitation until it was completely dissolved the powder. 5mls each was poured into tubes and cap well. The medium was autoclaved at 121 degree Celsius for 15 minutes. Tubes with medium were allowed to rest and

cool on a slanted position for slants.Media was then pack for use. This test is one of several techniques used to assist in the identification of gram negative bacteria specially enterobacteria. The test is based on the ability of an organism to use citrate as its only source of carbon .The bacteria make use of carbon to grow giving the media a blue colour.

6.2.1 CITRATE TEST

A sterile straight wire loop in the form of a needle was used to touch the test organism and stab at the butt and streak along the stabbed slop. It was then incubated at 35 °C for 48 hours.

Results

Green to bright blue.....Positive citrate test

Green to green colour.....Negative citrate test of medium

6.3 TRIPLE SUGAR IRON (TSI) AGAR PREPARATION

64 .52gms of triple sugar iron powder was suspended in1000mls of distilled water. The solution was mixed thoroughly and allowed to boil with frequent agitation to dissolve the powder. 8mls each was poured into tubes and cap well. The medium was then autoclaved at 121c for 15minutes to ensure proper sterilization. The medium was then allowed to set on slop with a butt of about 1 inch long to solidify and pack for used.

6.3.1 TRIPLE SUGAR IRON (TSI) TEST

A sterile straight wire loop in the form of a needle was used to touch the test organism and stab at the butt and also streak along the stabbed slop. It was then incubated at 35 °C for 48 hours. If the reaction is acidic, the result is written as A; if the reaction is alkaline, result is written as K.

(a)-slant yellow/butt yellow that means organism fermented glucose and lactose. The result is written as A/A;

Slant red/butt yellow that means organism fermented glucose but not lactose. The result is writtenasK/A;

Slant red /butt red that means that organism could not ferment glucose and lactose. The result is written as K/K.

(b)the presence of bubbles or splitting in the agar indicates gas production by the organism and reaction is written as G. (c)A black precipitate in the agar, seen as a black streak ,or blackening of the entire tube indicates the production of hydrogen sulphide and the reaction is written as H2S.

6.4 MOTILITY, INDOLE, ORNITHINE AGAR

31gms of medium was suspended in 1000mls of distilled water and mixed.The solution was heated and boiled with frequent agitation until the powderwas completely dissolved. 5mls each was poured into tubes and cap well. The medium was autoclaved at 121 degree Celsius for 15 minutes. Tubes with medium were allowed to rest and cool on a slanted position for slants.Media was then pack for use.

6.4.1 INDOLE TEST

This test demonstrated the ability of certain bacteria to decompose the amino acid tryptophan to indole which accumulates in the medium. Indole is then tested for by a colorimetric reaction with *p*-dimethyl-aminobenzaldehyde.

Medium was inoculated and incubated for 48h at 37^{o}C. 5ml of Kovac's reagent was added and shaken gently. A red colour in the alcohol layer indicated a positive reaction.

6.5 COAGULASE TEST

This testwas used to identify *S. aureus* which produces the enzyme coagulase. Coagulase causes plasma to clot by converting fibrinogen to fibrin. Two types of coagulases are produced by most strains of *S. aureus*: Free coagulase which converts fibrinogen to fibrin by activating a coagulase-reacting factor present in plasma. Free coagulase is detected by clotting in the tube test. The second type called Bound coagulase (clumping factor) converts fibrinogen directly to fibrin without requiring a coagulase reacting factor. It can be detected by the clumping of bacterial cells in the rapid slide test.

6.6 SLIDE TEST METHOD (DETECTS BOUND COAGULASE)

A drop of distilled water was placed on each end of a slide or on two separate slides. A colony of the test organism was emulsified in each of the drops to make two thick suspensions. A loop full (not more) of plasma was added to one of the suspensions, and mixed gently.

Results

Clumping within 10 sec.....*S. aureus*

Noclumping within 10 secs.....No bound coagulase

6.7 CATALASE TEST

This test is used to differentiate those bacteria that produce the enzyme catalase, such as *staphylococci*, from non-catalase producing bacteria such as *streptococci*. 2–3 ml of the

hydrogen peroxide solution was poured into a test tube. Using a sterile wooden stick or a glass rod, several colonies of the test organism were removed and immersed in the hydrogen peroxide solution. Immediate bubbling was observed.

Results

Active bubbling.....Positive catalase test

No bubbles.....….Negative catalase test

6.8 OXIDASE TEST

This test depends on the presence of certain oxidases in bacteria that will catalyze the transport of electrons in the bacteria and its detection by a redox dye-tetramethyl-p-phenylene-diamine. The dye is reduced to a deep purple colour.

Cultures were made on the growth medium.A freshly prepared 1%solution of tetramethyl-*p*-phenylene-diamine dihydrochloride was poured on to the plate so as to cover the surface, and was then decanted.The colonies of oxidase-positive organisms rapidly developed a purple colour.