FACTORS CONTRIBUTING TO MALNUTRITION IN CHILDREN 0-60 MONTHS ADMITTED TO HOSPITALS IN THE NORTHERN CAPE

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DECLARATION

I declare that the dissertation hereby submitted by me for the Magister degree in Dietetics at the University of the Free State is my own independent work and has not previously been submitted by me to another university / faculty. I further cede copyright of this research report in favour of the University of the Free State.

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Johanna Christina de Lange

May 2010

To my beloved husband, daughter and son

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LIST OF ABBREVIATIONS

abw	actual body weight
AIDS	acquired immune deficiency syndrome
ARI	acute respiratory infections
ART	anti-retroviral treatment
ARVs	anti-retroviral
BCG	Bacille Calmette-Guerin
BMI	body mass index
CD4	cluster of differentiation
CI	confidence interval
cm	centimeter
diff	difference
dL	desilitre
DoH	Department of Health
DRIs	daily recommended intakes
DTP3-HiB	third dose of diphtheria-tetanus-pertussis vaccine and *Haemophilus influenzae* type b vaccine
et al.	et alii
FAO	Food and Agriculture Organization
FBDG	food based dietary guidelines
g	gram
GI	gastrointestinal
HAART	highly active anti-retroviral therapy
HIV	human immune deficiency virus
IMCI	Integrated Management of Childhood Illnesses
INP	Integrated Nutrition Programme
IQ	intelegance quotient
IU	international units
kcal	kilocalorie
kg	kilogram
kJ	kilojoule
L	litre
m	meter
MDGs	Millennium Development Goals
ml	millilitre

mg	milligram
mm	millimeter
mm^3	cubic millimeter
mmol/L	millimol per liter
MUAC	mid upper arm circumference
MTCT	mother to child transmission
N	number
NCHS	National Centre for Health Statistics
NDoH	National Department of Health
NFCS	National Food Consumption Survey
NFCS-FB-1	National Food Concumption Survey Fortification Baseline
NSP	National Supplementation Programme
p	page
PEM	protein-energy malnutrition
PMTCT	prevention of mother to child transmission
R	South African rand
RtHC	Road to Health charts
SAM	severe acute malnutrition
SADHS	South African Demographic and Health Survey
SAVACG	South African vitamin A consultative Group
SD	standard deviation
STD	sexually transmitted diseases
STI	sexually transmitted infections
TB	tuberculosis
UNICEF	United Nations International Children's Emergency Fund
VCT	voluntary counseling and testing
WHO	World Health Organization
µmol	micromol
^0C	degrees Celsius
%	percentage
<	less than
>	greater than
≥	greater than or equal to
-	minus
²	square

CHAPTER 1: FACTORS CONTRIBUTING TO MALNUTRITION

1.1 INTRODUCTION

Malnutrition causes about 5.6 million of 10 million child deaths per year, with severe malnutrition contributing to about 1.5 million of these deaths (Heinkens *et al*., 2008). The nutritional status of children is the best indicator of the well being of children. Issues that cause a decline in the nutritional status of children are multidimensional and difficult to understand (De Onis *et al.,* 2000).

In order to ensure that all South Africans and their children can achieve optimal nutrition and to lower the incidence of infectious disease and malnutrition related deaths in infants and children, it is necessary to understand the factors contributing to malnutrition (National Department of Health (NDoH), 2005a).

The United Nations Children's Emergency Fund (UNICEF) conceptual framework of child malnutrition (Figure 1.1) shows multiple levels for interventions that can reduce morbidity and mortality related to malnutrition. To prevent or treat malnutrition the factors causing the condition need to be evaluated. The different causes of malnutrition are interlinked and include immediate causes, underlying causes and basic causes (UNICEF, 2004). All factors operate together and not independently (Williams, 2005, page (p). 405).

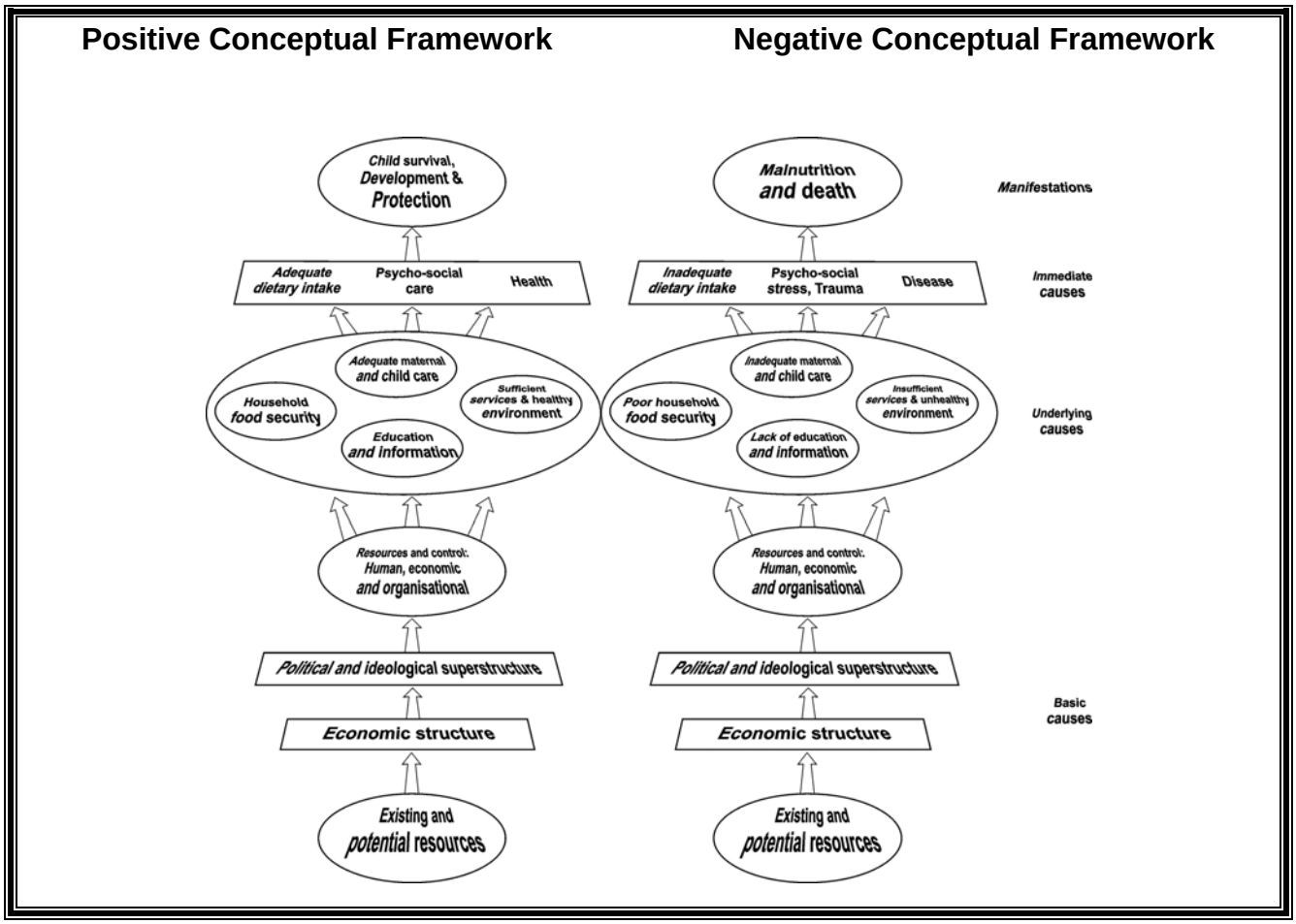


Figure 1.1 UNICEF conceptual framework of the causes of malnutrition (positive/negative) (UNICEF, 2004)

1.2 IMMEDIATE CAUSES

UNICEF (2004) classifies the immediate causes of childhood malnutrition as insufficient diet as well as stress, trauma, disease (severe or frequent infections) and poor psychosocial care. Insufficient dietary intake may refer to poor breastfeeding practices, early weaning, delayed introduction of complementary foods and insufficient protein in the diet. The inadequate intake can also be linked to neglect and abuse (UNICEF, 2004; Williams, 2005, p.405). Other factors that influence food intake include health status, food taboos, growth and personal choice related to diet (Vorster and Hautvast, 2002, p. 6).

1.2.1 INADEQUATE DIET

Inadequate dietary intake and poor nutritional status go hand in hand. It is uncommon for well-nourished children to die from diarrhoea, therefore maintaining a good nutritional status can help with the improvement of child survival (Jackson *et al*., 2006).

Factors contributing to the development of protein-energy-malnutrition (PEM) include cultural and social practices that lead to the exclusion of certain foods due to food taboos, food and dietary fads and migration from rural areas to urban slums (Torún and Chew, 1994, p.951; Torún, 2006, p.882; Piercecchi-Marti *et al*., 2006). Dietary choices are influenced by parents' nutritional ignorance, preference for alternative foods and true or perceived food allergies (Katz *et al*., 2005).

Malnutrition can also develop due to neglect, abnormal mealtimes with a carer or parent or insufficient quantities of food (because of insufficient parental knowledge, poor appetite in the child or neglect, physical or emotional abuse) (Zere and McIntyre, 2003; Duggan and Golden, 2005, p.519). Sometimes the mother restricts the child's food intake. This is either because the mother did not want the child or because a second child is born and there is not sufficient money to buy food for the expanding family (Piercecchi-Marti *et al*., 2006).

When income decreases, the quality and quantity of food also decreases. Evidence shows that when unemployment and low wages are presenting factors, families eat cheaper food, which is less nutritious, leading to weight loss and malnutrition (UNICEF, 2009b). As food products derived from animals are usually more expensive, children's intake of proteins and nutrients from these groups decreases with poverty (Christiaensen and Alderman, 2001). Malnutrition therefore also develops when the food ingested does not meet the high protein and energy needs of the child (Piercecchi-Marti *et al.,* 2006).

Globally, the practice of breastfeeding is declining (Torún and Chew, 1994, p.951; NDoH, 2003, p.8). When exclusive breastfeeding is not practiced it can contribute to a high prevalence of malnutrition (NDoH, 2005a). In South Africa the practice of exclusive breastfeeding is very low. The South African Demographic and Health Survey (SADHS) found that of all three month old babies, only ten percent were exclusively breastfed and 48,3 percent (%) were bottle fed (NDoH, 2005a). In addition, inadequate weaning practices and poor infant feeding practices lead to low protein and energy intake (Torún and Chew, 1994, p.951; NDoH, 2003, p.8).

Factors leading to nutrient deficiencies and low energy and protein intakes seen in children are the increased use of diluted cow's milk and vegetable foods and a delay in giving children family foods (Torún and Chew, 1994, p.952; Kapur *et al*., 2004; Torún,

2006, p.883). Even though breast milk is rich in high quality protein (Monckeberg, 1991, p.122; Torún and Chew, 1994, p.952; Golden and Golden, 2000, p.515; Torún, 2006, p.893), prolonged breastfeeding causes a delay in the introduction of complementary foods and can result in micronutrient deficiencies, as human milk is low in iron and zinc (Kalanda *et al*., 2006).

On the other hand, babies are sometimes weaned too early because of another birth, causing the mother to cease breastfeeding of the first baby. Babies are then often weaned on a thin cereal with low quality protein, causing the older child to become ill when the new baby arrives. Children cannot obtain food for themselves (Monckeberg, 1991, p.122; Torún and Chew, 1994, p.952; Golden and Golden, 2000, p.515; Torún, 2006, p.893); and they have small gastric capacities, meaning they are incapable of ingesting large amounts of, or sufficient, food. This in turn can lead to malnutrition (Torún and Chew, 1994, p.952; Torún, 2006, p.883).

In developing countries malnutrition may develop after breastfeeding is ceased because of low milk production, death of the mother or because the mother decided to bottle-feed her infant. The mother might have decided to bottle-feed because of her Human Immunodeficiency Virus (HIV) status, work commitments or because the baby is not living with her (Berdanier, 1995, p.154). Breast milk substitutes may be unsuitable because of a high renal solute load (cow milk) or low energy density (diluted cow's milk or incorrect formula) (Duggan and Golden, 2005, p.522).

The early introduction of complementary food is associated with an increased risk of respiratory infections, eye infection and a high incidence of malaria morbidity. When complimentary foods are started, there is a reduction in breast milk consumption, which can lead to a loss of protective immunity. This causes a higher morbidity when unhygienic foods are used, due to the development of diarrhoea. According to a study done by Kapur *et al.* (2004) in India, growth curves falter by the fourth month of life due to the early introduction of weaning foods.

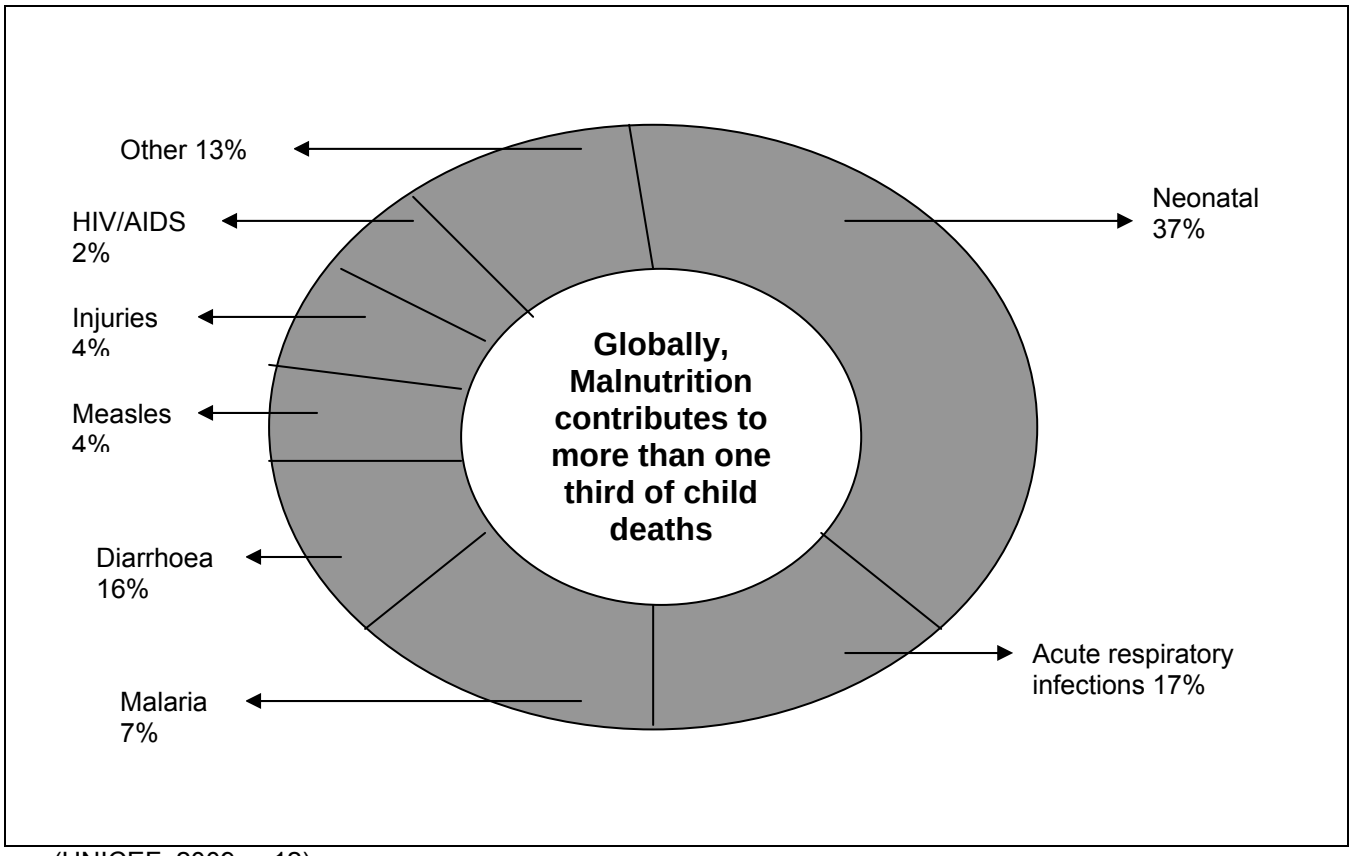
In Prevention of Mother To Child Transmission (PMTCT), mothers that opted for exclusive breastfeeding had a mean duration of exclusive breastfeeding of less than one month (UNICEF, 2007).

1.2.2 DISEASE

Most deaths of children 6-59 months old are related to malnutrition and infection (NDoH, 2005a; Torún, 2006, p.882). Caulfield *et al.* (2004) found that the principal causes of deaths in young children globally in 2004 were: diarrhoea (60,7%), pneumonia (52,3%), measles (44,8%) and malaria (57,3%). All of these can also worsen malnutrition. Some additional causes associated with child mortality were found by Müller and Krawinkel (2005) and UNICEF (2009, p. 12) and include perinatal causes, acute respiratory infections and others (Figure 1.2). Some of the most common infectious diseases in South Africa are HIV and acquired immune deficiency syndrome (AIDS), tuberculosis (TB), measles, diarrhoea and acute respiratory infections (ARI) (NDoH, 2005a).

Infections play a major role in the etiology of PEM because they result in increased needs and a high energy expenditure, lower appetite, nutrient losses due to vomiting, diarrhoea, poor digestion, malabsorption and the utilization of nutrients and disruption of metabolic equilibrium (Golden and Golden, 2000, p.515; NDoH, 2005a; Williams, 2005, p.405; Torún, 2006, p.882). It takes time for a malnourished child to recover from respiratory and diarrhoeal diseases and therefore the risk of morbidity and mortality is higher. Repeated illnesses contribute to ill health and compromised nutritional status (Pereira, 1991, p.143).

Figure 1.2 Causes of mortality in children under five years (2004)



(UNICEF, 2009, p.12)

1.2.2.1 HIV AND OPPORTUNISTIC INFECTIONS

Three million children have HIV and AIDS; with ±800 000 children becoming newly infected yearly and ±500 000 dying from AIDS related illnesses each year. The epidemic is the greatest in Sub-Saharan Africa (Tomkins, 2005, p.486). Complications of paediatric HIV infection are usually seen in growth failure and finally more serious malnutrition (Eley and Hussey, 1999). Half of children presenting with severe malnutrition are HIV infected (Golden and Golden, 2000, p.524).

Globally, all countries are trying to achieve Millennium Development Goals (MDGs) four: to promote child health and six: to combat HIV and AIDS. Anti-retrovirals (ARVs) are becoming more available and therefore severe malnutrition in the context of HIV is becoming increasingly important. The need for malnourished HIV infected children to be treated in facilities is increasing by the day (Heinkens *et al*., 2008). Evidence in sub-Saharan countries shows that HIV infected children can recover their nutritional status when given the correct treatment for severe acute malnutrition (SAM) without ARVs but their recovery is slower than that of uninfected children (Collins *et al.,* 2006; World Health

Organization (WHO), 2007b). In developing countries, the severity of malnutrition in HIV infected children is greater and more severe than in uninfected children (Eley and Hussey, 1999). The role of anti-retroviral therapy (ART) in achieving better nutritional status is vital (Heinkens *et al.,* 2008).

Opportunistic infections or malnutrition are the cause of 75% of the deaths among HIV infected children before the age of five years (Eley and Hussey, 1999). In Sub-Saharan Africa, the mortality rate of malnourished HIV infected children is three times higher than in uninfected children. HIV has changed the epidemiology, clinical presentation, pathophysiology, case management and survival of malnourished children. Even with the WHO guidelines case fatality rates are at 20-50%. More and more HIV infected children are being admitted to hospital (Heinkens *et al*., 2008).

A study done by Bachou *et al*. (2006) showed that within a group of 315 malnourished children, 119 (38%) were female with a median age of 17 months while only 3% were below the age of six months. They also showed a high prevalence of infections (26%) and bacteraemia (18%). The HIV infected children were more likely to have persistent diarrhoea than the HIV uninfected malnourished children (Bachou *et al.,* 2006).

Children of three to six years old are often admitted for persistent diarrhoea with a high case fatality rate and poor prognosis even with management according to guidelines (Heinkens *et al.,* 2008). HIV infected malnourished children are either perinatally infected, underfed or both (Winter, 1996; Heinkens *et al*., 2008) due to HIV infected children usually being present in families that are poor and food insecure (Heinkens *et al.,* 2008). Infants of HIV infected mothers have a low weight gain in the first four months of life and then a decrease in height is also observed (Winters, 1996). Even uninfected children are affected because mothers and caretakers have chronic diseases and high mortality (Winter, 1996; Heinkens *et al.,* 2008).

During breastfeeding babies may be exposed to the HIV virus from HIV infected mothers for prolonged periods (Kalanda *et al*., 2006) and Mother To Child Transmission (MTCT) rates are further influenced by nutritional status and dietary intake (Tomkins, 2005, p.486).

The lower weight gain in HIV infected children can often be ascribed to the presence of infectious diseases in these children (WHO, 2007a). Infections can be viral, bacterial, parasitic and fungal opportunistic (Fenton and Silverman, 2008, p.1009). Some of the infections include TB, pneumonia, skin infections and oral thrush. All of these contribute to the development of malnutrition (Bentley and Lawson, 1988, p.43; Torún and Chew, 1994, p.952; Torún, 2006, p.883; Collins *et al*., 2006; Heinkens *et al.,* 2008). When children have lower respiratory tract infections, TB is 22 times more prevalent in HIV infected children that uninfected children (Heinkens *et al.,* 2008). Children in Africa have trouble thriving when they have an infectious disease. During this time they often do not respond to nutrition therapy even when adequate amounts of food are given (Shetty, 2002, p.320).

Seeing as nutrition and HIV are closely linked, weight loss and wasting are problems associated with inadequate intake due to anorexia, malabsorption, digestion, metabolic irregularities, increased excretion of nutrients through vomiting and decreased absorption. In addition, catabolic processes, abnormal energy utilization, increased requirements, uncontrolled opportunistic infections and/or a lack of physical activity are also involved weight loss and wasting (Bentley and Lawson, 1988, p.43; Torún and Chew, 1994, p.952; Winter, 1996; Eley and Hussey, 1999; Torún, 2006, p.883; Fenton and Silverman, 2008, p.1008).

Decreased oral intake can also occur due to medications, depression, infection, nausea, vomiting, diarrhoea, dyspnoea, fatigue, neurological disease (Winter, 1996; Fenton and Silverman, 2008, p.1008), fever, pain, dementia and despair (Winter, 1996). Low oral intake is also caused by problems in the mouth and oesophagus, such as thrush and oral herpes (Fenton and Silverman, 2008, p.1008) and dysgeusia due to zinc deficiency (Winter, 1996).

The reduced intake causes a deficiency of energy needed for resting energy expenditure (Eley and Hussey, 1999). Other deficiencies due to low food intake in asymptomatic HIV infected children include reduced plasma levels of retinol, beta-carotene, folate and iron, which becomes more severe when clinical AIDS develops (Tomkins, 2005, p.486).

In HIV infected children there is low serum levels of Vitamin A, C, B_6, B_{12} and E, beta-carotene, selenium, zinc, copper and iron. Vitamin A deficiency is associated with a

higher risk of HIV infection and higher risk of MTCT. Deficiencies of copper, zinc, iron, selenium, magnesium, folic acid, vitamin A, C. B_6, B_{12}, beta-carotene and vitamin E leads to a higher risk for opportunistic infections and progression of AIDS which can lead to death (Hendricks *et al.,* 2006).

The gastrointestinal (GI) tract is one of the most important organs in the acquiring of HIV. When children become sick due to HIV infection, it leads to malabsorption resulting from epithelial cell dysfunction and bacterial overgrowth, diarrhoea, and infections (Winter, 1996). Malabsorption causes loose stools, diarrhoea or vomiting, which can be caused by medications, a developed intolerance to lactose, fat or gluten (Winter, 1996; Fenton and Silverman, 2008, p.1008) and small intestine damage (Winter, 1996).

The immune changes seen in AIDS and PEM are similar. Deficiencies of protein, calcium, copper, zinc, selenium, iron, essential fatty acids, pyridoxine, folate and Vitamins A, C, E all interfere with immune function. Direct and indirect mechanisms are responsible for the impact of nutrition on HIV. Nutrition plays a direct role in immune-cell triggering, interaction and expression. Indirectly nutrition plays a role in deoxyribonucleic acid and protein synthesis as well as the physiologic integrity of cell tissues, organ systems and lymphoid tissues (Fenton and Silverman, 2008, p.1009).

HIV can lead to food insecurity through the loss of labour, increased need for health care and funerals, low household agricultural production due to sick household members not able to work, diminished ability to care for young children and vulnerable individuals and the loss of wealth. There is therefore also a relationship between food insecurity and an increase in the HIV epidemic (Hendricks *et al*., 2006).

1.2.2.2 DIARRHOEA

Diarrhoea causes about 30-50% of deaths in developing countries. The risk of death due to persistent diarrhoea is related to a lack of breastfeeding, systemic infections, malnutrition and young age (Ochoa *et al.,* 2004). GI infections are one of the most common infections in children with PEM (Pereira, 1991, p.144-145) and are especially important among children of weaning age that present with severe or frequent episodes of diarrhoea (Torún and Chew, 1994, p.952; Torún, 2006, p.883).

Some of the non-infectious factors that cause diarrhoea include celiac disease, intolerance to cow's milk, allergic colitis and intolerance to carbohydrates. Persistent diarrhoea is mainly an infection-induced illness and is usually the result of continued gram-negative infections, unresolved infections, secondary malabsorption, gastroenteritis syndrome (Ochoa *et al.,* 2004; Heinkens *et al*., 2008), zinc deficiency and changes in intestinal flora (Heinkens *et al*., 2008).

Mucus damage is associated with acute gastro and post-enteritis syndrome. The villi become short, the number and height of microvilli decrease, enterocyte borders are blunted, the glycocalyx is lost, and crypt hyperplasia follows. These structural changes have a negative effect on intestinal digestive, absorptive and barrier functions. Food related antigens could further increase structural and functional damage to the mucosa during intestinal infections (Ochoa *et al.,* 2004; Amadi *et al*., 2005).

Diarrhoea leads to shifts in fluids and electrolytes and is therefore life threatening (Pereira, 1991, p.144-145; Ochoa *et al.,* 2004; Heinkens *et al*., 2008). The malnourished child with diarrhoea presents with potassium depletion and is sensitive to sodium retention. Once the fluid and electrolyte balance has been corrected, the child should receive required minerals and vitamins and adequate amounts of easily digested energy-dense foods (Shetty, 2002, p. 320). Wasting as well as oedema makes the assessment of dehydration in children with diarrhoea difficult (Pereira, 1991, p.144-145; Heinkens *et al*., 2008).

The incidence of diarrhoea among HIV infected patients is estimated to be about 30-70%. Highly active anti-retroviral therapy (HAART) can help with some recovery of the immune system. Sometimes the diarrhoea, associated with infections, may stop once the medication starts to work. Not all cases of chronic diarrhoea amongst AIDS infected patients are however linked to infections. Some of the cases are caused by drug side effects, GI malignancies and HIV enteropathy (Ochoa *et al.,* 2004).

Persistent diarrhoea is part of a vicious cycle between nutrition, poverty, poor hygiene, environmental contamination, inappropriate feeding practices and early weaning. The association between the immune system and the gut is important for the development of malnutrition (Ochoa *et al.,* 2004) and when parents refrain from taking their children, with diarrhoea to a health facility to be treated, the risk for the development of malnutrition

increases (Abate *et al*., 2001). Persistent diarrhoea also affects growth and intellectual function (Ochoa *et al.,* 2004).

Children can be protected against acute and persistent diarrhoea, when probiotics, expressed breast milk and breastfeeding are used in the first six months. Promotion of breastfeeding is an important prevention strategy (Ochoa *et al.,* 2004). Bottles used for milk and other fluids are often unclean and milk is prepared in unhygienic conditions with unclean water. Prevention strategies should include promotion of hygiene and sound milk preparation practices (Monckeberg, 1991, p.123; Berdanier, 1995, p.154).

1.2.2.3 OTHER

Measles is the cause of about one million deaths per year in developing countries. Deaths from measles are seen due to secondary bacterial and viral infections, the immune suppression mechanism that is related to PEM and vitamin A deficiency. Complications such as pneumonia, diarrhoea, malnutrition, otitis media, mouth ulcers, corneal epithelial keratitis, corneal ulceration and blindness occur in about 10-30% of patients with measles (Semba, 2006, p.1403).

When the impact of PEM on the severity of infection was investigated in children with measles, diarrhoea, respiratory infections, and malaria, it was found that the morbidity and mortality in patients with infections is worse if they are malnourished (Semba, 2006, p. 1403). TB is common and leads to increased energy and protein requirements (Tomkins, 2005, p.487). In urban areas, primary TB is a major contributing factor to childhood malnutrition (Pereira, 1991, p.145).

1.2.3 PSYCHOSOCIAL CARE

The mother-baby-bond should be in place early in life for better cognitive, emotional and social development later in life (Play Therapy Africa, 2009). Evidence shows that quality of care is linked to infant nutritional status. The quality of psychosocial care is often determined by the interaction between mother and child. A protective effect on nutritional status is seen by talking to the child, storytelling, hugging the child, having a safe and attractive environment and encouraging independence. Independence gives the child the ability to obtain food and health care later in life (Carvalhaes and Benicio, 2006).

It is important for parents to strengthen their psychosocial care and support skills as part of the intervention programme for malnourished children as the effects of hunger and food insecurity are closely linked to psychosocial stress. Parents should be involved as far as possible with their children's care and they should be taught the importance of play (UNICEF, 2005; Play Therapy Africa, 2009). Hunger and food insecurity put extra stress on parents which can lead to emotional problems and neglect, in turn leading to a decrease in the appetite of the child (Play Therapy Africa, 2009).

All these issues reduce the survival of the child, even when given enough food. Children that do survive these circumstances will have long-term mental and cognitive disabilities and can be stunted with poor growth (Play Therapy Africa, 2009). Psychosocial care is also linked to better care practices in terms of eating and health. A study done in Mexico showed that there is an association between a mother that is not responding to her child, a poor environment and severity of malnutrition in the child. Mothers of malnourished children were more apathetic and dependent and showed more personal and family problems, immaturity and isolation with low self-esteems and feelings of inadequacy (Carvalhaes and Benicio, 2006).

Maternal behaviours are directly linked to the psychosocial care of the child. Children from low-income households have a high risk of malnutrition if the psychosocial environment is insufficient. The risk is also lower in households with a low-income and good psychosocial care, which shows that good psychosocial care, can almost protect the child against their poor socio-economic conditions (Carvalhaes and Benicio, 2006).

Emotional stimulation of the child is vital for preventing severe malnutrition. Children will not improve with only food, but also need attention. The combination of food and emotional support can have a positive effect on physical, mental and emotional outcomes during times of food crisis and can increase survival rates. Children that are not stimulated can have reduced psychomotor activity such as not crawling or playing. The moment children become less active and demanding, parents tend to provide less stimulation (Play Therapy Africa, 2009).

Ogunba (2008) did a study on psychosocial care and complementary feeding of children under two years in Nigeria. About 77% of the mothers in the study cared for their own children while 23.1% used caregivers. Complementary feeding started from one month.

The study found that the percentage of mothers who motivated their children to eat was 58.7%, 76.4% of mothers sat with their children while they ate, 5.3% of mothers talked to their children and 23.6% of the mothers forced their children to eat. About 76.2% of children had their own bowls to eat from. The study showed that the psychology and culture of people strongly influence the care and feeding of children (Ogunba, 2008).

Feeding times are ideal for strengthening the psychosocial bond. This is especially important in times of crises when children need to be resilient and mentally healthy to survive. Parents and caregivers are sometimes unavailable or unable to give psychosocial care because of their own illnesses (Play Therapy Africa, 2009).

Malnourished children that received psychosocial stimulation showed an almost 50% quicker weight gain than those without stimulation. Children showed a 65% improvement in attention, irritability, lethargy and intolerance (Play Therapy Africa, 2009). Studies done by Play Therapy Africa (2009) showed reduced mortality rates from 28.6% to 20.6%, increased speed of recovery, earlier discharge from hospital and prevention of emotional, development and intellectual loss or damage (Play Therapy Africa, 2009).

1.3 UNDERLYING CAUSES OF MALNUTRITION

The underlying causes of malnutrition include inadequate levels of household food security, inadequate care of children and women, low education levels and information, insufficient health services and an unhealthy environment (availability of sanitation and safe water) (Jones, 1998; UNICEF, 2004; Müller and Krawinkel, 2005). For malnutrition to improve there should be specific emphasis on social norms, gender equity and maternal access to education (UNICEF, 2009c, p.37).

1.3.1 HOUSEHOLD FOOD SECURITY

Household food security is seen as all people in the household having access to food at all times. The food must be safe and of high quality and the environment should be hygienic enough to use the food so that all members can lead healthy, productive lives. Food security concentrates on four aspects: availability of food, stability of food supply, access to food and utilization of food (Food and Agriculture Organization (FAO), 1996).

Globally there are about one billion people that go hungry and about 2.6 billion people that are poor. A study done in Bangladesh, Nepal and Pakistan shows that the situation

is worsening. Seeing as the price of staple foods is increasing and economic growth is poor, there is little evidence to show that other countries are doing better (UNICEF, 2009b)

The size and composition of the family, gender equity, rules of food distribution within the household, income, availability and access to food (James *et al*., 1999; Vorster and Hautvast, 2002, p. 6), poverty (NDoH, 2003, p.8; Mason *et al*., 2005; UNICEF, 2009c, p.13) and the death of the breadwinner (Mason *et al*., 2005) can all contribute to food insecurity. Food insecurity can also occur due to poor agriculture production, destruction of infrastructure and markets and therefore loss of income, loss of livestock and insufficient land for food production. Families will also increase their credit to try and survive. These factors influence the quantity and quality of food available (FAO, 1996).

Families will reduce their consumption to match what they have available. Lack of food will have an impact on work performance, productivity and income. When families do not have enough oil for instance to provide enough calories, the child needs to eat more often and that is not possible if the family is food insecure. Not having all foods necessary for growth will lead to weight loss and deficiencies. When there is not enough food in the house, it becomes difficult to decide who will receive what is available (FAO, 1996).

According to a survey done by UNICEF and the Institute for Public Health Nutrition in 2004 in Bangladesh, one in four households is food insecure and two million children are affected by malnutrition (between six months and five years). The survey was designed to assess the impact of the food price increases in Bangladesh. Data showed that 58% of households had insufficient food in the previous year. A link was found between malnutrition and food insecurity, with food insecure households showing a higher percentage of malnourished children (UNICEF, 2009).

Two thirds of the children in South Africa live in households with an income of less than $200 per month and the unemployment rate is about 40% for 8.4 million people (UNICEF, 2007). In a study by Crowther (2008) regarding the association between household food security and mortality in children under five years of age in Agincourt, Limpopo Province, the results showed that 37% of the population's households were food insecure (seen as insufficient food) in the previous month and year.

In South Africa, 52% of children are experiencing hunger and 23% are at risk for experiencing hunger (National Food Consumption Survey (NFCS), 1999). In South Africa, three out of four children live in poor, insecure households (75%)(NFCS, 1999; Crowther, 2008). The moment children experience food insecurity and poverty, it causes low or inadequate food intake and sometimes disease, which leads to the development of PEM and death. These issues are among the most urgent social issues affecting households and their children (Crowther, 2008). Food aid should only be used as a short and mid term intervention while improving the family's long-term situation (FAO, 1996).

1.3.2 INADEQUATE MATERNAL AND CHILD CARE

Ignorance is directly associated with poor infant and child rearing practices, misconceptions about food, inadequate feeding during illness (especially infectious diseases and diarrhoea), improper food distribution among family members (Torún and Chew, 1994, p.951), poor maternal care (James *et al*., 1999) and high birth rates (NDoH, 2003, p.8). Childcare practices also include protecting the children's food and drinks from contamination to reduce the risk of infections. A caregiver's unwashed hands can cause infections such as diarrhoea. (Abate *et al*., 2001).

In a study by Ayaya *et al*. (2004) in Eldoret, Kenya, the social risk factors for PEM included being a single mother and a young mother aged 15- 25 years (Ayaya *et al.*, 2004). Other social problems include child abuse and maternal deprivation (Torún and Chew, 1994, p.951; Torún, 2006, p.882).

In Southern Africa there is a decrease in caring capabilities of caregivers the moment poverty and food insecurity increases (Shoo, 2007). Poverty can indirectly cause poor caring practices when a parent becomes ill and dies; and issues related to feeding and hygiene are exacerbated by emotional instability (Mason *et al*., 2005).

When the household income decreases, it is usually the women who try earning extra wages. This causes the mother to have less time for childcare and ensuring the children eat healthy food. If the female children are also sent out to look for work, this results in poor school attendance, which influences education, leading to poor knowledge and caring practices for her own family (FAO, 1996; UNICEF, 2009b).

Mothers should be protected against malnutrition, seeing as healthy mothers are needed for raising healthy children. Care includes breastfeeding, diagnosing illnesses, and introduction of solids, stimulating language and other cognitive capabilities and emotional support. Care affects the child's nutritional status through better infant feeding practices and breastfeeding, preparation of healthy food, hygiene and through support of the mother so that she has sufficient time to care for the child (FAO, 1996).

In the United States of America, high breastfeeding rates caused a reduction in pneumonia of 32% and gastro-enteritis of 15%. Better maternal knowledge leads to better childcare practices, seeing as maternal education is associated with breastfeeding for longer than six months and the delayed introduction of solids (Kalanda *et al*., 2006). Uneducated mothers with a low socio-economic status have trouble preparing infant formula correctly and the milk is too expensive to give sufficient amounts. Finances force the mothers to use diluted cow's milk (Monckeberg, 1991, p.123; Berdanier, 1995, p.154).

In South Africa, the NDoH (2003) found that other factors contributing to malnutrition include poor maternal health and nutritional status of the mother, anaemia, smoking, the age of the mother, poor access to health services, especially among rural women and the high prevalence of sexually transmitted diseases (STD). When a mother has a syphilis infection the infection can have a direct influence on the vertical perinatal HIV transmission to the child (Lee *et al*., 2009).

Maternal malnutrition before, during and after pregnancy may result in underweight newborn babies. Intrauterine malnutrition increases the occurrence of PEM after birth, seeing as the infant gets insufficient food to meet their requirements for catch-up growth (Torún and Chew, 1994, p.952). Maternal death increases the risk of PEM at all ages. Underfeeding can result because of insufficient breast milk when the mother has died, is ill with HIV or has twins (Duggan and Golden, 2005, p.522).

Maternal smoking had a negative effect on the height-for-age of children in Cambodia, Namibia and Nepal. Maternal smoking and biofuel smoke can lead to growth deficiencies (Kyu *et al.,* 2009). Maternal smoking also leads to low birth weight babies and can predispose the infant to respiratory illnesses. Active smoking during pregnancy had more of a negative effect on the infant than passive smoking after birth. Smoking during pregnancy damages the developing respiratory system, either through the

bronchial tree or the developing lung vasculature. Smoking during pregnancy also interferes with the immune system and can lead to congenital immunodeficiency (Taylor and Wadsworth, 1987).

1.3.3 INADEQUATE HEALTH SERVICES AND ENVIRONMENT

Malnutrition rates in the developing world are still high because of the lack of access to health services (NDoH, 2003, p.8; Oyelami and Ogunlesi, 2007). Even though patients have little or no access to formal health services, there is still the problem that patients do not make use of the services available (Müller and Krawinkel, 2005). According to James *et al*., (1999) there is a need for improved public health services and improved immunization and growth monitoring programmes.

Ayaya *et al.* (2004) found that incomplete immunizations were a risk factor for the development of malnutrition and Iqbal *et al*. (1999) found that incomplete Bacille Calmette-Guerin (BCG) vaccination against TB increased the risk for the development of severe PEM in Bangladesh. The education and promotion of important vaccinations can reduce the occurrence of PEM (Iqbal Hossain *et al*., 1999).

In South Africa, not enough health facilities are available and not all health care workers are knowledgeable about the Road to Health Charts (RtHC). Growth monitoring is a very useful tool to measure infant and child health. Still, the reality remains that caregivers and parents are ignorant regarding growth monitoring and promotion. Of all South African mothers and caregivers with young children of 12-13 months only about 74.6 % had RtHC in 1998 (NDoH, 2005a).

Families that are food insecure and reliant on inadequate health services develop a reduced resistance to infections, which causes malnutrition. The health services are influenced by a loss of health staff, which leads to a higher workload for those that stay behind. This has a serious effect on the quality and quantity of health services rendered. The staff that are available at the facilities lose their skills because of a lack of supplies and equipment, lack of incentives and low morale. Shortages of staff can also lead to remote areas not being covered by health services (FAO, 1996).

One of the biggest public health service challenges is to make sure that the necessary services reach those that are most vulnerable and in need. Even though 40% of under

five deaths are caused by AIDS globally, only 11 000 are receiving ARVs because of inadequate testing procedures and treatment services. These services are mostly available at hospitals and not primary health care facilities (UNICEF, 2007).

Most of the health services in Africa are based on facility-based care. Community-based programmes operate on a smaller scale and with limited support. Poor performance of health services contributes to the high mortality rates of preventable deaths, such as neonatal conditions (27%), pneumonia (21%), malaria (18%), diarrhoea (16%), HIV and AIDS (6%), measles (5%), injuries (2%) and others (5%). In 54% of these deaths, malnutrition was the underlying cause (Shoo, 2007).

In 2000-2001 50% of the deaths in two South African hospitals among severely malnourished children were due to doctor and 28% due to nurse errors. If these could have been prevented the mortality would have been much lower. These are caused by weaknesses in the health system, where doctors and nurses have inappropriate training, inadequate supervision and there is a lack of support systems for staff (Jackson *et al*., 2006).

Unhealthy environments, overcrowding, lack of water and unclean water and poor sanitation, directly lead to malnutrition through infections (FAO, 1996). SAM occurs mainly in families living in unhygienic conditions and with limited access to food. The abovementioned conditions increase the risk of repeated infections (WHO, 2007a). According to Abate *et al*. (2001) poor household hygiene practices are critical in preventing infectious diseases. Child waste inside the house, prolonged storage of cooked food, feeding with unwashed hands and storage of food and water in uncovered containers can cause diarrhoea among malnourished children. These poor hygiene practices lead to contaminated food and fluids (Abate *et al*., 2001).

Overcrowding and poor environmental sanitation is often the cause of illness in children, especially in developing countries (Pereira, 1991, p.143). Overcrowded and unsanitary living conditions are closely linked to poverty (Torún and Chew, 1994, p.951).

Households were there was child waste inside the house had a 7.5 times greater chance of experiencing malnutrition than those that had a clean environment within the house or ten metres from the home (Jeyaseelan and Lakshman, 1997; Abate *et al*., 2001). The

households with human faeces in the house were 73.4%. Households where the cooked food was stored for longer than 24 hours (22.9%) also have a greater risk of malnutrition than the well-nourished households that stored cooked food (59.9%). In 22.3% of households the food was not covered and the uncovered, stored food can lead to a 3.5 times higher risk of being malnourished (Abate *et al*., 2001). Uncovered drinking water can lead to a three times higher risk of being malnourished (Getaneh *et al*., 1998; Abate *et al.,* 2001) and six out of ten households had their own tap for water, whereas 9% of households got their water from a river or dam and 4% got their water from a borehole or well (Labadarios *et al*., 2008). Unwashed hands are 2.5 times more likely to be linked to malnutrition and in 29.7% of households hands are washed before feeding (Abate *et al.,* 2001).

Most of the causes of deaths of infants and toddlers in South Africa are associated with poor socio-economic conditions (Bradshaw *et al.,* 2003) and PEM is also associated with poor socio-economic background in Ethiopia (Getaneh *et al*., 1998). The 2001 census of South Africa showed different living conditions. Over two thirds of households had formal houses, 16% had informal and 14% traditional homes. Clean water is important for health. The census showed that most households had access to piped water (84.5%) in the home, in the yard or somewhere in the area. Nationally, 13.6% have no toilets and little bit more than 50% had regular refuse removal (Bradshaw *et al.,* 2003). Having no toilets available was also associated with PEM in Ethiopia (Getaneh *et al*., 1998).

Getaneh *et al*. (1998) also found an association between PEM and poor housing conditions in Ethiopia, and also temporary housing in Kenya (Ayaya *et al.,* 2004) or mud walled houses in Kampala (Owor *et al*., 2000). The household's economic position has a significant impact on the risk of a child being stunted and underweight (Zere and McIntyre, 2003). The fathers' occupation is the best indication of income and there was an association between PEM and the father being a laborour (Saito *et al*., 1997), having a lower income job (Jeyaseelan and Lakshman, 1997; Rikimaru *et al.,* 1998) and having no land, no livestock such as cattle (Owor *et al*., 2000; Ayaya *et al.,* 2004), no maize, no beans and the grandfather owns only a small piece of land (Ayaya *et al.,* 2004). Iqbal Hossain *et al.* (1999) found a significant association between low household income, parental illiteracy and small family size (less than six members). In this study there was a close to significant association between room density and the prevalence of malnutrition

1.3.4 INFORMATION AND EDUCATION

Malnutrition is worsened by a lack of nutritional information and knowledge, especially maternal nutrition education (NDoH, 2003, p.8), which leads to unhealthy dietary habits, poor nutrition related practices and attitudes, perceptions and socio-cultural influences. All of these issues can negatively influence nutritional status. For families to be healthy with a good nutritional status, they need knowledge regarding growth, purchasing, processing, and preparation and feeding a variety of food, in the right quantities and combinations (NDoH, 2005a). A lack of nutritional knowledge can also lead to misconceptions about food and negative food traditions that are passed on from generation to generation (NDoH, 2005b).

Previous studies done in the Philippines show that maternal education is one of the most important key elements in addressing child malnutrition. The association between maternal schooling and child health still needs to be investigated further. There are three ways how school education and knowledge can influence the child's health and nutritional status: (1) formal education leads directly to a higher knowledge of mothers; (2) literacy acquired in school ensures that mothers are more capable of identifying health problems in children; and (3) when mothers have attended school they are more aware of modern diseases and where to get help and information (Christiaensen and Alderman, 2001).

Even though nutrition knowledge is not gained in the classroom, the school education that mothers receive can help with caring for children and the household. Both female and male education can have a positive effect on the child's nutritional status. Knowledge can lead to a higher household income and better nutritional status when the education is linked with strategies to improve both. Maternal nutrition knowledge matters even more when the child falls within the high-risk group of younger than three years (Christiaensen and Alderman, 2001), as there is an association between low maternal literacy and poor nutritional status of children three to 23 months (UNICEF, 2009c, p.36).

1.4 BASIC CAUSES OF MALNUTRITION

Basic causes, also called national or root causes, of malnutrition include poor availability and control of resources (political, social, ideological and economic), environmental degradation, poor agriculture, war, political instability, urbanization, population growth and size, distribution, conflicts, trade agreements and natural disasters, religious and cultural factors (Torún and Chew, 1994, p.952; Vorster and Hautvast, 2003, p.8; UNICEF, 2004a;

Torún, 2006, p.883). In addition, landlessness and migrant labour are also considered to be basic causes of malnutrition (NDoH, 2003, p.8). Other basic causes include market failures due to economic decline, conflict and political upheavals that can lead to a reduction in food yields and price increases (Mason et al., 2005). Loss of food after a harvest can also occur when storage conditions are poor and food is inadequately distributed (Torún and Chew, 1994, p.952; Torún, 2006, p.883).

If issues related to the economic position of the family are affected negatively, it can influence the chances of a child being stunted and underweight (Grantham-McGregor, 1984; Zere and McIntyre, 2003; UNICEF, 2004a).

1.5 PROBLEM STATEMENT AND MOTIVATION FOR THE STUDY

Worldwide there are about 60 million children with moderate acute and 13 million with severe acute malnutrition. About 50% of the 10-11 million children under five years of age die due to preventable causes. Of all the children that die, 99% are in the developing world (Ashworth *et al.,* 2004). About 9% of sub-Saharan African children have moderate acute malnutrition and 2% of children in developing countries have SAM. Mortality is related to the severity of the malnutrition, where severe wasting has a mortality rate of 73-187 per 1000 children per year (Collins *et al*., 2006).

Poor hospital care of severe acute malnutrition (SAM) contributes to high mortality rates (Ashworth *et al.,* 2004) and the case fatality rates in hospitals in developing countries is still about 20-30% and has changed little since the 1950s. This is despite the fact that protocols can reduce the fatality rates to 1-5% and have been available for the past 30 years (Collins *et al*., 2006). In addition, not all severely malnourished cases are reported as such in hospital statistics. Most of these cases as reported as diarrhoea and pneumonia and therefore statistics are sometimes misleading (Jackson *et al.,* 2006).

Africa still has a high prevalence of PEM. The death rate for under five year old children has decreased after public health interventions such as immunizations, oral rehydration and vitamin A supplementation were implemented (Duggan and Golden, 2005, p.522). The main concern as seen by Collins *et al*. (2006) and Duggan and Golden (2005, p.522) is that the mortality rate is not falling as quickly as hoped and malnutrition can also be an indicator of poor program coverage.

The NFCS of 1999 (NFCS, 1999) found that stunting was more prevalent in South Africa than underweight and wasting, especially in the Eastern Cape and Northern Cape. The Eastern Cape and Northern Cape are the two South African provinces with the highest concentration of poverty (NFCS, 1999).

The Northern Cape is sparsely populated and houses some 840 321 people (2% of the national population) on 361 830 km^2 which is almost 30% of South Africa's area. Seventy percent of the population is situated in urban areas and 30% in rural areas. More than ten percent of the Northern Cape's population is younger than five years and 32.6% are between five to nine years. A unique characteristic of the Northern Cape is its large land mass and low population. This results in a low population density and large distances between centres (Statistics South Africa: Northern Cape Report, 2003).

Education Literacy rate in the Northern Cape was about 83% in 2004, which was the third lowest in South Africa and also lower than the national average rate, which stood at 88,2%. The Northern Cape had about 68 000 female-headed households in 2004. Electricity, wood, coal and other sources were used for cooking, heating and lighting, with wood being the second most popular source for cooking and heating. The Northern Cape contributed to about 2,2% of the economy of South Africa in the period 1996–2004. It recorded the second lowest average annual economic growth rate (2,2%) among all provinces in this period (Statistics South Africa: Provincial Profile 2004: Northern Cape, 2004).

The Northern Cape has a high unemployment rate of 27.4 %. It is the second highest in South Africa with Limpopo and the Eastern Cape having the highest rates (Statistics South Africa: Quarterly Labour Force Survey, 2009). Taking this into account it is clear that resources and money are scarce in the Northern Cape.

At provincial level in 1995, the prevalence of stunting was the highest in the Northern Cape, (31%), Free State (30%), Mpumalanga (26%), then North West (24%), Northern Province (23%) and Eastern Cape (20%)(Table 2.3). The NFCS (1999) reported that the prevalence of malnutrition in the Northern Cape was 27,2 % for stunting, 25.8% for underweight and 13,1% for wasting.

Even though the causes of malnutrition can be broadly categorized into immediate, underlying and basic causes, they differ from area to area. Before interventions can be planned for an area, it is necessary to understand the causes of malnutrition in that area. This study is important to determine the causes responsible for severe malnutrition in children zero to 60 months in the Northern Cape Province.

1.6 AIM AND OBJECTIVES

The aim of this study was to determine the causes of severe malnutrition in children 0-60 months admitted to hospitals in the Northern Cape.

Objectives to achieve the main aim:

- Determine background information on the child and mother/caregiver.
- Determine the anthropometrical status of malnourished children and their caregivers.
- Determine immediate factors contributing to malnutrition (breastfeeding practices, weaning, dietary intake and disease).
- Determine underlying factors contributing to malnutrition (household factors, socio-economic status, maternal and child care, education levels, nutrition information received, healthy environment).
- Determine basic factors contributing to malnutrition (availability and control of resources).
- Determine associations between the above mentioned

1.7 OUTLINE OF THE DISSERTATION

The dissertation is divided into 6 chapters:

The first chapter is an introduction to the study that states the problem and gives an overview of the causes of malnutrition as described in the literature. The aim and objectives are also described.

The second chapter is a literature overview of what PEM is, how it is classified and the treatment for PEM. The literature overview reviews the global and South African perspective on the prevalence of malnutrition, anthropometrical classification with specific emphasis on underweight, stunting and wasting (marasmuss, kwashiorkor and marasmic

kwashiorkor), biochemical and physical signs, as well as physiological changes occurring in the body. Finally, the literature overview will look at the overall treatment of malnutrition.

The third chapter gives an overview of the methodology that was used to implement the study.

The fourth chapter includes the results, while the fifth chapter includes a discussion of the results and how it compares to results of other relevant studies.

The sixth chapter includes conclusions that were drawn from the results and recommendations for further intervention and prevention and possible further research.

CHAPTER 2: LITERATURE REVIEW

2.1 INTRODUCTION

Globally, hunger and malnutrition are two of the most significant challenges (Strobel and Ferguson, 2005, p.487). Globally, malnutrition is a risk factor for illness and death, with millions of pregnant women and young children being affected due to infections, poor and inadequate diet. Malnutrition increases the risk and worsens the severity of infections (Müller and Krawinkel, 2005). Infants and young children are most affected by malnutrition as they have increased nutritional needs to support growth (Torún and Chew, 1994, p.952; Torún, 2006, p.883). Undernourished children, as well as children with severe malnutrition, have a higher risk of dying than children with an optimal nutritional status (Caulfield *et al*., 2004).

The term "malnutrition" is usually used to describe PEM. The comprehensive term of "PEM" is universally accepted and its severe forms are called "marasmus", "kwashiorkor" and "marasmic kwashiorkor" (Torún and Chew, 1994, p.951). The term SAM combines all the different forms of PEM, as SAM refers to a weight-for-height below 70%, referred to as "wasted" or pitting oedema is present in both feet, referred to as "oedematous malnutrition". Severe forms of SAM can also be complicated by infections. The different forms still have different causes and are therefore treated differently (Collins *et al.,* 2006).

2.2 PREVALENCE OF MALNUTRITION

Except for sub-Saharan Africa, the nutritional status of children is improving globally. Progress is however, hindered because of poverty, infection and ineffective governance (Duggan and Golden, 2005, p.524). Even though global data shows a decrease in undernutrition, the malnutrition statistics for Eastern Africa are increasing (Cartmell *et al*., 2005).

There is not enough information available on the prevalence of severe or oedematous malnutrition in communities. The data available from hospitals only shows the severe cases and therefore malnutrition in general is not always recorded because in most cases it is the secondary diagnosis (Duggan and Golden, 2005, p.518-522).

Cartmell *et al*. (2005) found that in the Central Hospital of Maputo the occurrence of malnutrition in the presence of infections, excluding measles, was greater in 2001 than in

1983. More children had marasmus than kwashiorkor in 2001. Possible explanations for this occurrence can be the increase in HIV infection; with marasmic malnutrition occurring more commonly in HIV infected children in South Africa, Maputo and Malawi (Cartmell *et al.,* 2005).

Despite the work done in malnutrition and the reduced prevalence of stunting and underweight in some regions, the number of cases hasn't changed over the last 10 years (Zere and McIntyre, 2003; Müller and Krawinkel, 2005) with about 30 percent of all children in low- and middle-income countries being underweight (Mother and child nutrition, 2007). Malnutrition is and will continue to be a health threat to developing countries, especially in Southern Asia and Sub-Saharan Africa (Müller and Krawinkel, 2005) and might actually be rising in the developing world such as Africa because of the HIV pandemic (Oyelami and Ogunlesi, 2007).

2.2.1 GLOBAL PERSPECTIVE

In 1990 an estimated one out of three children (177 million) younger than five years in the developing world were or had been malnourished at one stage in their lives (Table 2.1). The diagnosis was based on a weight-for-age below two standard deviations (SD) of the National Centre for Health Statistics (NCHS) median. In countries where the prevalence of malnutrition is high, the total number of malnourished children has not decreased with an increase in population (Torún and Chew, 1994, p.951).

Ayaya *et al*. (2004) stated that malnutrition is still one of the leading causes of morbidity and mortality in children younger than five years and according to Kilic *et al.* (2004) severe PEM still affects 2-3% of the paediatric population worldwide.

Table 2.1 Prevalence of PEM among children under 5 years of age in developing countries, 1995 (Müller and Krawinkel, 2005)

Region	Stunting %	Underweight %	Wasting %
Africa	39	28	8
Asia	41	35	10
Latin America and Caribbean	18	10	3
Oceania	31	23	5

The State of the World's Children report published by UNICEF in 1998 stated that malnutrition is a "silent emergency" leading to almost seven million child deaths (approximately 55% of all child deaths) annually. Three quarters of children dying are mildly to moderately malnourished with no obvious outward signs of problems (Jones, 1998).

In 2000-2002 an estimated 852 million children were malnourished, of which 815 million were in developing countries (Zere and McIntyre, 2003; Müller and Krawinkel, 2005) and 34 million in developed countries (Vorster and Hautvast, 2002, p.4) (Table 2.2). During this time malnutrition was directly responsible for about 300 000 deaths per year and indirectly for about half of all deaths in young children (Zere and McIntyre, 2003; Müller and Krawinkel, 2005). More than 199 million children younger than five years suffer from acute or chronic protein and energy deficiencies (Vorster and Hautvast, 2002, p.4). In 2004 an estimated 55% of child deaths worldwide were the result of undernutrition (Caulfield *et al*., 2004).

Table 2.2 Estimated prevalence (and numbers in millions) of undernourished children in developing countries by region in the year 2000 (Shetty, 2002, p.321)

Region	Underweight % (number x 10 6)	Stunted % (number x 10 6)
Africa	28.5 (38.32)	35.2 (47.30)
Asia	29.0 (107.91)	34.4 (127.8)
Latin America and Caribbean	6.3 (3.40)	12.6 (6.82)
Developing countries	26.7 (149.63)	32.5 (181.92)

Micronutrient deficiencies affect about two billion people in the world. Globally 740 million people are deficient in iodine (300 million with goitre and 20 million with brain damage from maternal and iodine deficiency during their foetal development), about two billion people are deficient in zinc and one billion have iron deficiency anaemia (Müller and Krawinkel, 2005).

Globally, vitamin A remains the most important and preventable cause of early blindness (Williams, 2005) and in 1988 an estimated 100 000 infants become blind due to vitamin A deficiency and an equal number died from associated conditions (Bentley and Lawson, 1988). In 2005 about 250 million people, mainly young children and pregnant women, in developing countries, had vitamin A deficiency (Müller and Krawinkel, 2005).

In the last 25 years the prevalence of stunting has decreased globally (Torún, 2006, p.882). The prevalence of stunting has fallen in developing countries from 47% in 1980 to 33% in 2000, although progress has been uneven in different regions. Stunting has increased in Eastern Africa, but decreased in South-eastern Asia, South-central Asia and South America; Northern Africa and the Caribbean show modest improvement; and Western Africa and Central America have shown very little progress. Despite an overall decrease of stunting in developing countries, child malnutrition still remains a major public health problem in these countries. In some countries rates of stunting are rising, while in many others they remain disturbingly high (De Onis *et al*., 2000).

There are still about 800 million undernourished people in the world and in some countries severe malnutrition is the most common reason for paediatric hospitalisation. Around 27% of the children younger than five years of age in the developing world are underweight, 32 % are stunted, and 10 % wasted (seen as a deficit of more than two standard deviations below the WHO reference value) (Torún, 2006, p.882).

2.2.2 SOUTH AFRICAN PERSPECTIVE

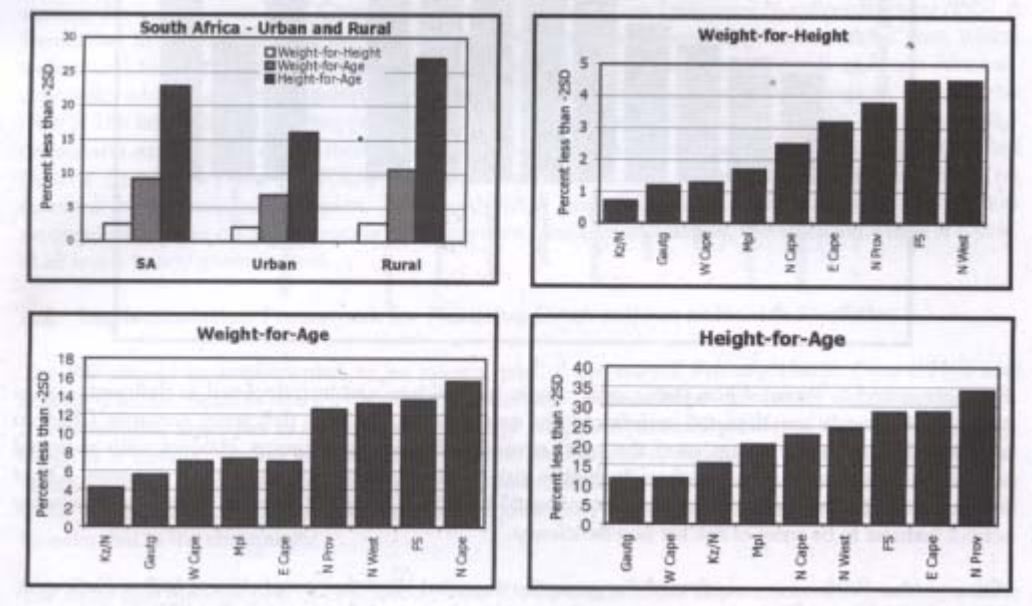
In 1995, the South African Vitamin A Consultative Group (SAVACG) study found that one tenth of all South African children aged one to nine years were underweight, and just more than one fifth were stunted. Younger children (one to three years), living in rural areas and on commercial farms were most severely affected (SAVACG, 1995) (Fig. 2.1). In total about 660 000 preschool children are underweight and 1.5 million are stunted due to chronic malnutrition (SAVACG, 1995).

The Saving Children report that looks at child healthcare in South Africa found that over 60% of children who died between 1 January 2005 and 31 December 2005 were underweight for age and 33% were severely malnourished. Seventeen percent of the cases had no record of nutritional status. The survey was done at 15 hospitals in Gauteng, Mpumalanga, North West, KwaZulu-Natal, Free State and Northern Cape. Children from birth to 18 years of age were included in this survey (Patrick and Stephen, 2005, p.5).

According to the NFCS of 1999, nationally 10.3% of children are underweight, 19.3% were stunted with the age group one to three years having the highest prevalence of 24.4% (NFCS, 1999; Steyn *et al*., 2005) and 3.7% were wasted (NFCS, 1999). In

accordance with the SAVACG results, the NFCS reported that stunting was more prevalent in South Africa than underweight and wasting, especially in the Eastern Cape and Northern Cape (NFCS, 1999).

Figure 2.1 Anthropometric status of children < 6 years of age in South Africa, 1994 (SAVACG, 1995)



At provincial level, the NFCS study found that the prevalence of stunting was the highest in the Northern Cape, (31%), Free State (30%), Mpumalanga (26%), then North West (24%), Northern Province (23%) and Eastern Cape (20%)(Table 2.3). The prevalence of malnutrition in the Northern Cape was 27,2% for stunting, 25.8% for underweight and wasting was 13,1% (NFCS, 1999).

The NFCS (1999) found a South African mortality rate of 45.2 deaths per 1000 live births, 61/1000 for children younger than five years of age and an estimated prevalence of 8.3% for low birth weight. In 2003, South Africa was estimated to be 69th in the under five mortality rate rankings (66/1000) with an infant mortality rate of 50/1000. The national prevalence of low birth weight babies (<2500 gram (g)) was estimated at 16% (NDoH, 2003, p.6).

The Saving Children report also showed that 59% of under five deaths were HIV and AIDS related. In 2005, 46% of all deaths were known to be HIV related and in a further

46% the HIV laboratory status was not known. Only 8% of those that were tested tested negative (Patrick and Stephen, 2005, p.15).

Table 2.3 Anthropometric status of children 1-3 and 4-6 years of age in South Africa, 1999 (NFCS, 1999)

Parameter	PROVINCE									SOUTH AFRICA		
1 – 3 YEARS OF AGE												
Province	***EC***	***FS***	***Gau***	***KZN***	***Mpu***	***NC***	***NP***	***NW***	***WC***	***RSA***	***Urban***	***Rural***
Number (a)	142	93	233	215	55	70	136	113	141	1 198	617	581
%H/A<-2SD	23,2	39,8	26,2	25,1	29,1	30,0	19,9	31,9	14,2	25,5	20,9	30,3
%W/A<-2SD	10,6	20,4	9,9	6,5	7,3	27,1	14,0	18,6	9,9	12,4	9,7	15,1
%W/H<-2SD	2,8	3,2	1,3	2,3	1,8	12,9	11,0	5,3	1,4	4,0	2,6	5,5
4-6 YEARS OF AGE												
Province	***EC***	***FS***	***Gau***	***KZN***	***Mpu***	***NC***	***NP***	***NW***	***WC***	***RSA***	***Urban***	***Rural***
Number (a)	156	81	128	167	60	48	131	82	122	975	468	507
%H/A<-2SD	19,9	27,2	15,6	16,8	25,0	31,3	29,0	18,3	14,8	20,7	15,6	25,4
%W/A<-2SD	3,8	9,9	9,4	6,6	3,3	20,8	16,0	12,2	4,9	8,8	6,2	11,2
%W/H<-2SD	1,9	1,2	1,6	5,4	3,3	4,2	5,3	7,3	0,8	3,4	2,3	4,3

2.3 CLASSIFICATION OF MALNUTRITION

The term PEM describes a spectrum of pathological conditions ranging from kwashiorkor to marasmus. Some of the risk factors and clinical features of the two severe forms of PEM may be similar, but the main feature of kwashiorkor is oedema (Oyelami and Ogunlesi, 2007). Children with PEM can have different symptoms depending on what caused the malnutrition (Gallagher, 2008, p.66).

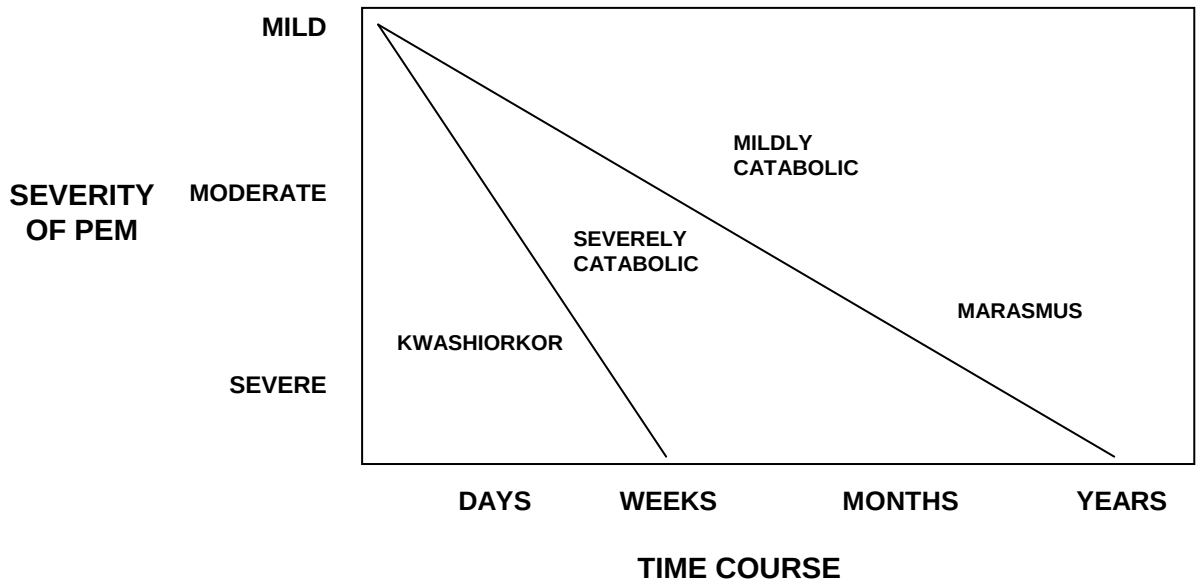
Severe PEM includes deficiencies of protein, energy or both, resulting in kwashiorkor, marasmus and marasmic kwashiorkor (Torún, 2006, p.881), with marasmic kwashiorkor developing because of a combination of chronic energy deficiency and chronic or acute protein deficiency (Torún and Chew, 1994, p.950; Torún, 2006, p.881).

Primary PEM is caused by an inadequate food intake that may be the result of a variety of factors (discussed in chapter 1). Diseases cause secondary PEM through low food intake, decreased absorption and usage of nutrients, increased requirements and increased losses (Torún, 2006, p.881). All factors co-exist in the same individual and therefore there is often a mixed clinical picture. Failure to achieve normal growth is a

sensitive indicator of malnutrition (Golden and Golden, 2000, p.517 & 519) and the first and most important sign of PEM (NDoH, 2003, p.8).

Micronutrient deficiency and clinical characteristics depend on the severity of energy and protein deficiency, the duration and cause of the deficiency, the age of the host and the association with other diseases. The onset can be fast, as with starvation or gradual when food is chronically withheld (Figure 2.2) (Torún, 2006, p.881).

Figure 2.2 Time course of PEM (Morgan and Weinsier, 1998, p.168)



Anthropometry is used to assess nutritional status and growth retardation and to differentiate between acute or chronic malnutrition. The clinical findings and biochemical criteria are not effective to use for classification if the disease is not advanced, but can help to confirm a diagnosis (Torún and Chew, 1994, p.959; Torún, 2006, p.889).

The three combinations of anthropometric measurements that are usually used to categorize malnutrition are: low weight-for-age, an indicator of underweight; low height-for-age, an indicator of stunting; and low weight-for-height, an indicator of wasting. Wasting is an indicator of recent and severe malnutrition (acute malnutrition) and can be effectively used to determine the immediate impact of intervention programmes (Müller and Krawinkel, 2005). Marasmus is characterized by extreme thinness and a weight of below 60% of the reference weight for age (Duggan and Golden, 2006, p.519).

Many classifications have been suggested to classify the syndromes of PEM. The Wellcome Committee's (Table 2.4) categorization is both simple and practical and is based on the presence of oedema and weight-for-age (Bentley and Lawson, 1988, p.42; Wittenberg, 2004, p.203). This classification can help to prevent misclassification of children with oedema due to reasons other than malnutrition (Golden and Golden, 2000, p.517; Wittenberg, 2004, p.203).

Table 2.4 Wellcome Committee categorization of PEM (Bentley and Lawson, 1988, p.42; Wittenberg, 2004, p. 203)

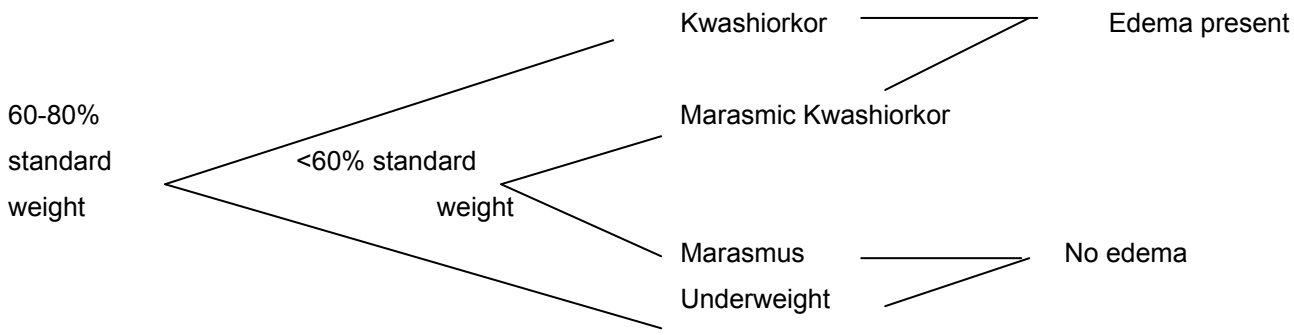
SEVERITY	PERCENTAGE OF STANDARD*
1st degree (mild)	89 – 75 % expected weight for age
2nd degree (moderate)	74 – 60 % expected weight for age
3rd degree (severe)	60 % expected weight for age

* Standard – 50th percentile NCHS standard

Integrated Management of Childhood Illnesses (IMCI) guidelines as developed by the WHO and used by developing countries, recommend that health workers identify severe PEM by the presence of visible severe wasting and oedema on both feet (Hamer *et al*., 2004).

The severity of PEM can be determined by expressing the actual weight as a percentage of the expected weight of a healthy child of the same age using a standard (Bentley and Lawson, 1988, p.42). Children are grouped together according to two criteria: the presence or absence of oedema and the weight-for-age. The only problem is the definition of kwashiorkor is used when any patient presents with nutritional oedema. In this instance kwashiorkor is used with two different meanings: one as a synonym for "oedematous malnutrition" and the other is the clinical syndrome presenting with changes in skin, hair and fatty liver. A child is diagnosed with marasmus when the child has a weight for age of less than 60% of the standard (Table 2.4 and Fig. 2.2) (Golden and Golden, 2000, p.517; Wittenberg, 2004, p.203).

Figure 2.3 Wellcome Committee categorization of PEM



(Bentley and Lawson, 1988, p.43; Wittenberg, 2004, p.203)

Table 2.5 WHO classification of malnutrition (WHO, 1999; Golden and Golden, 2000, p.518; Duggan and Golden, 2006, p.520)

	Moderate Malnutrition	**Severe Malnutrition**
Symmetrical oedema	**No**	**Yes**
Weight for height (wasting)	SD score > -3.0 and < - 2.0 ≥ 70 % and < 80 % reference	SD score < - 3.0 < 70 % reference
Height for age (stunting)	SD score > -3.0 and < - 2.0 ≥ 85 % and < 90 % reference	SD score < - 3.0 < 85 % reference

Severe acute malnutrition, is defined as a weight for height measurement of 70% or more below the median (WHO, Table 2.5), or three SD or more below the mean NHCS reference value, which is called “wasted”; the presence of bilateral pitting oedema of nutritional origin, which is called “oedematous malnutrition” (Collins *et al*., 2006).

According to the Gomez classification, a child is classified as malnourished according to weight in relation to the weight of a normal child of the same age, expressed as a percentage (Table 2.6) (Golden and Golden, 2000, p.517).

Table 2.6 Gomez classification (Cogill, 2003)

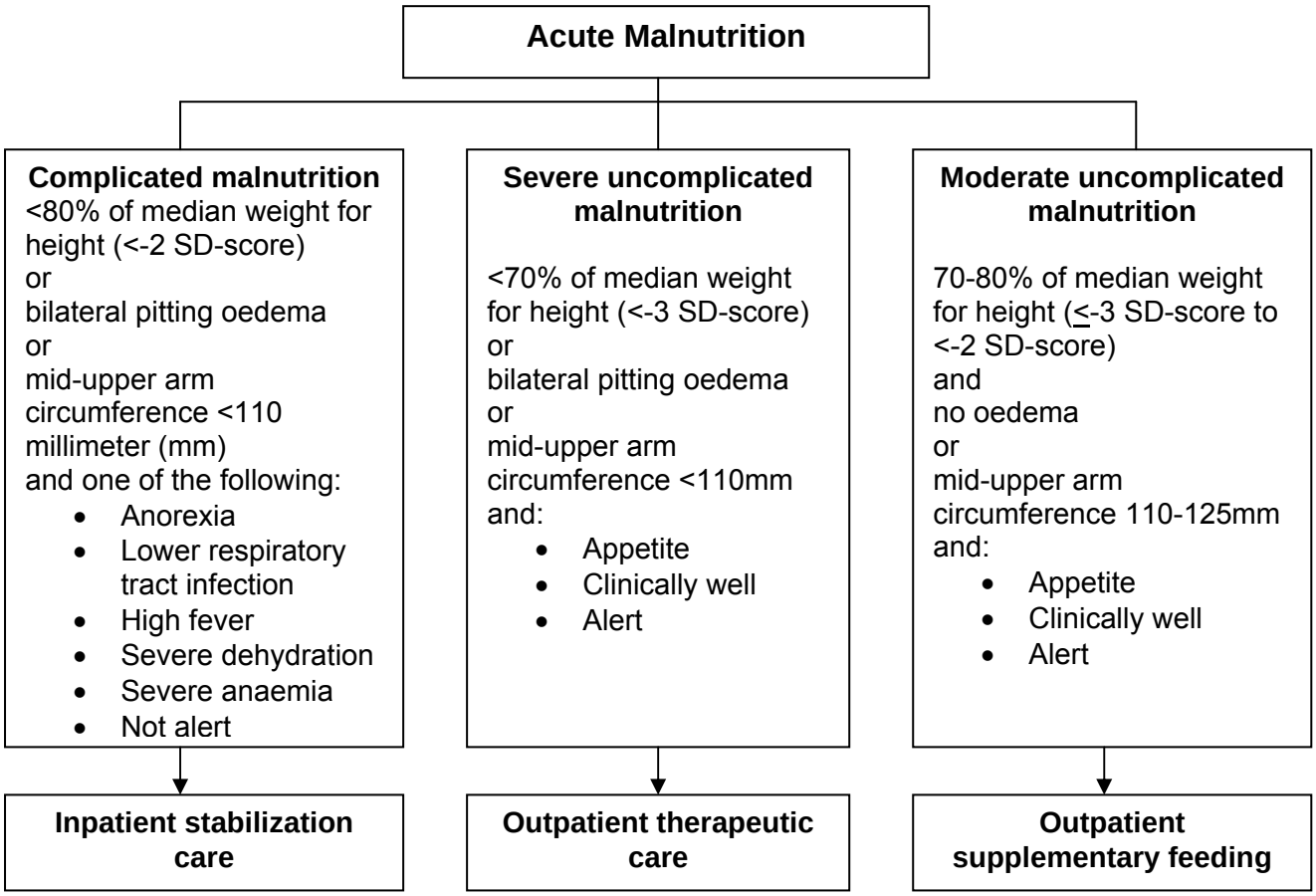
Cut-off	**Malnutrition classification**
> 90 % of median	Normal
75 % - <90 % of median	Mild
60 % - < 75 % of median	Moderate
< 60 % of median	Severe

According to Waterlow in Torún and Chew (1994, p.959), Torún (2006, p.890) and Wittenberg (2004, p.203) patients may fall into four categories: 1) normal, 2) wasted but not stunted (acute PEM), 3) wasted and stunted (acute and chronic PEM) and 4) stunted not wasted (past PEM, present adequate nutrition / “nutritional dwarfism”).

Anthropometry is limited when measurement error influences the interpretation of nutritional status. The basic measurements, height (length) and weight, are used in all nutritional studies, because it gives the simplest measure of attained skeletal size (height / length) and of soft tissue mass (weight) (Bates *et al.,* 2005). Length measurements (until two years of age and height thereafter) were introduced more recently (Duggan and Golden, 2006, p. 581-519).

As community-based therapeutic care is becoming more effective, there is a need to change the classification for acute malnutrition. The WHO classification is divided into categories of moderate and severe malnutrition, according to anthropometry and presence of pitting oedema on both feet. Previously children were treated on in-patient basis for severe acute malnutrition cases and on an outpatient basis for moderate acute malnutrition cases. With the inclusion of community-based care, a third category is needed for complicated malnutrition. Severe and moderate cases can both be complicated and the complicated cases will be treated in a health facility to stabilize the patient (Collins and Yates, 2003) (Figure 2.4).

Figure 2.4 Classification system for acute malnutrition in community-based therapeutic care (Collins and Yates, 2003; Collins *et al*., 2006)



2.3.1 UNDERWEIGHT

The underweight child is common and an important presentation of PEM, which is missed a lot of times (Wittenberg, 2004, p.203). When a diet is insufficient in protein and/or energy there will be a slowing down of linear height, failure to gain weight or weight loss (Wittenberg, 2004, p.203), and this is seen when the child is exposed to an acute food shortage (Golden and Golden, 2000, p.517-518). These children are underweight and undersize, while at the same time they have relatively normal body proportions, e.g. weight-to-height ratios (Golden and Golden, 2000, p.517-518; Wittenberg, 2004, p. 203). Underweight children can also be stunted, wasted or both (UNICEF, 2009c, p.13).

Underweight children must be identified early through regular growth monitoring of weight and height (Wittenberg, 2004, p.204; UNICEF, 2009c, p.13). Underweight children can easily be missed when both weight and height are not showed on the RtHC. When growth monitoring is done and a child presents with a weight for age below the third

percentile (less than 80% expected weight or less than 90% expected height), the child must be suspected of being malnourished (Wittenberg, 2004, p.204).

Underweight children have a dietary deficiency that is not severe and therefore do not produce a clinical disease or symptoms. There are no real physical signs and the serum albumin is only slightly reduced. Underweight children are however, still very susceptible to infections, such as gastro-enteritis, respiratory disease, measles and TB (Wittenberg, 2004, p.204).

In the developing world, 129 million of children younger than five years are underweight and 10% are severely underweight. Underweight is more prevalent in Asia than in Africa, with Asia showing rates of 27% and Africa rates of 21%. Progress is slow and South Africa is not meeting the MDGs with the prevalence being 25% in 2008, whereas it was 28% in 1990 (UNICEF, 2009c, p.17-18).

2.3.2 STUNTING

Stunting is a greater problem than underweight and wasting (UNICEF, 2009c, p. 11) and is an indicator of nutritional deficiencies or status (Shetty, 2002, p. 321; UNICEF, 2009c, p.11) and illness that occurred during times of growth and development (UNICEF, 2009c, p.11) usually in infants and children younger than five years (UNICEF, 2009c, p.11). Stunting is the first clinical sign of malnutrition (Piercecchi-Marti *et al*., 2006) and affects about 195 million children younger than five years in the developing world, where stunting affects about one in three children in Africa (UNICEF, 2009c, p.15).

Stunting can also be called failure to thrive or growth faltering, which refers to slow weight gain or inadequate growth in the infant and young child. Stunting is an indication of chronic malnutrition and long-term insufficient diet because of a chronic energy deficiency (Müller and Krawinkel, 2005; Williams, 2005, p.404; Duggan and Golden, 2006, p.519). As stunting is due to long-term undernutrition, it takes time to develop and to recover (Baker-Henningham and Grantham-McGregor, 2004, p.253).

Growth failure is marked by both “thinness and shortness”. “Nutritional” growth faltering is not only due to underfeeding but also due to infection (Golden and Golden, 2000, p.517-518), psychological disturbance, socio-economic deprivation and underlying illnesses (Williams, 2005, p. 401). Stunting is a cumulative process that starts in utero, and there is

substantial evidence that intrauterine growth is a strong predictor of postnatal growth (De Onis *et al*., 2000).

Stunting is an indication of the height of the child compared to the height of a normal child of the same age (Golden and Golden, 2000, p.518). A "stunted" child is small for his or her appropriate height for age. A height-for-age smaller than 85% of the median (50%) represents an SD score of minus (-) 3SD and is classified as severe stunting (Williams, 2005, p.406).

A stunted child, living in a population with similarly sized children can appear to be thriving. Biological and cultural adaptation causes the body to look the same as the other children in the same environment. An underweight for age child, who is severely stunted, may even appear plump (Duggan and Golden, 2006, p.519). Children appear normal, but when the age becomes apparent, it is obvious that the child is short. Height is more retarded than dental development, so the child's face looks inappropriate for their size (Golden and Golden, 2006, p.519). Stunting is also associated with a poor school outcome, where stunted children usually start school later, do not complete all grades and do not perform as well as children of the same age (UNICEF, 2009c, p.14).

Children in rural communities are at a greater risk of becoming stunted than children living in urban areas. Children living in informal housing have the highest prevalence and the lowest is seen in children whose mothers are well educated. In South Africa the prevalence of stunting was the highest in children living in traditional or informal housing, with poorly educated mothers (NDoH, 2003, p.9) and is currently the developing country that has the 24th highest prevalence of stunting (UNICEF, 2009c, p.10).

The rate of stunting (low height for age) in some places, such as parts of India, is between 50 and 60 percent (Mother and child nutrition, 2007). A study by Mamabolo *et al*. (2005) found a high prevalence of stunting (48%) amongst three year olds in Limpopo, South Africa. The study also found that the length and weight attained at one year of age could predict the nutritional status of the child at three years of age. If children had a higher length at one year, they were more protected against stunting (Mamabola *et al*., 2005).

Prevalence in the developing world has been declining from 40% in 1990 to 29% in 2008. The decline was small in Africa and went from 38% in 1990 to 34% in 2008. This was due to the population growth of children younger than five years with stunting, which increased from 43 million in 1990 to 52 million in 2008 (UNICEF, 2009c, p.17).

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2.3.3 WASTING

"Clinical wasting" is the term used to describe recent severe fat loss due to illness or severe food restriction (Duggan and Golden, 2006, p.519). Inadequate food intake leads to weight loss and growth retardation and when it is prolonged it leads to body wasting and emaciation (Torún and Chew, 1994, p.950; Torún, 2006, p.881). When growth is acutely affected a child falls behind one who is actively growing (Golden and Golden, 2000, p.517-518), with a body weight and height less than ideal for the child's age (Shetty, 2002, p. 321).

Wasting is indicated as a low weight for height, occurring at any age (Shetty, 2002, p. 321) and is used as an indicator for identifying severe acute malnutrition (UNICEF, 2009c, p.13). A child is wasted when the weight for height is less than 70% of the median and is equal to a standard deviation score of –3SD (Williams, 2005, p.406). Wasting is the weight of the sick child compared to that of a normal child of the same height (Golden and Golden, 2000, p.518).

Of the children younger than five years old in the developing countries, 13% are wasted and 5% are severely wasted (about 26 million). Africa and Asia are the two countries with high rates of wasting and exceed 15%. Out of 134 countries, 32 of these countries have wasting prevalence of 10% or more. And ten countries are contributing to about 60% of all wasted children. In South Africa the prevalence of wasting is 5-9.9% (UNICEF, 2009c, p.21).

2.3.3.1 KWASHIORKOR

Kwashiorkor was first described more than 70 years ago in 1933 (Golden and Golden, 2000, p.519; Katz *et al*., 2005). The first description came from the Gold Coast of Africa (now Ghana) (Katz *et al*., 2005), where kwashiorkor means, an "evil spirit that infects the first child when the second child is born". Kwashiorkor sets in at the ages of one to three years (Torún and Chew, 1994, p.961-963; Berdanier, 1995, p.153; Sizer and Whitney, 2000, p.196; Whitney *et al.,* 2001, p.83; Whitney and Rady, 2005, p.198; Torún, 2006,

p.891-893; Gallagher, 2008, p.66) and usually after 18 months (Torún and Chew, 1994, p.952; Torún, 2006, p.883).

The nature and importance of the disease were only recognized in the 1950's, when there were almost 40 names for the disease. One of the names used was "sindrome policarencial de la infancia" (infantile pluricarential syndrome). This showed that young children were affected and that they were deficient in various nutrients. Others names such as "mehlnahrschade" ("damage by cereal flours"), "starch oedema" and "sugar babies", showed that the disease was caused by low protein diets (Torún and Chew, 1994, p.957 & 951; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Torún, 2006, p.891-893) and high carbohydrate or almost exclusively carbohydrate diets (Torún and Chew, 1994, p.951; Gallagher, 2008, p.66).

Considering how kwashiorkor develops, it is easy to see how the Ghanaians came to use the word kwashiorkor. When the second child is born, the first is weaned so that the second can be breastfed. The first child receives food low in protein and starch, even though they were used to protein rich breast milk (Berdanier, 1995, p.153; Whitney *et al.,* 2001, p.83; Katz *et al*., 2005; Whitney and Rady, 2005, p.198-199; Gallagher, 2008, p.66). Inappropriate foods such as non-dairy creamer, flour water, molasses and atole (a corn porridge in Mexico) are then used for infant diets (Katz *et al*., 2005).

Kwashiorkor can present rapidly and usually refers to acute PEM (Torún and Chew, 1994, p.961-963; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Torún, 2006, p.891-893) and can develop within a few weeks (Heimburger, 2006, p.833). Kwashiorkor is generally more typical of rural areas (Monckeberg, p.121, 1991).

All systems and functions are affected in kwashiorkor. No single etiological agent is responsible. It is difficult to determine which factors are major contributors and which are responses. In combination with weight loss, oedema has been accepted as the main criteria to identify kwashiorkor. Kwashiorkor is more prevalent in children who are stunted or wasted but it can occur in children of normal size (Jackson and Golden, 1991, p.134-135). Children with kwashiorkor have a weight-for-age of 80-60 % of expected weight (Wittenberg, 2004, p. 203).

Kwashiorkor presents with growth retardation, skin changes (lesions), abnormal hair that is dry, brittle and easy to pull out, swollen belly, hepatomegaly (enlarged, fatty liver) and apathy (Torún and Chew, 1994, p.961-963; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Strobel and Ferguson, 2005, p.488; Torún, 2006, p.891-893; Heimburger, 2006, p.833).

Children with kwashiorkor have a well-nourished appearance (Heimburger, 2006, p.833) with some retention of body fat (Torún and Chew, 1994, p.961-963; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Torún, 2006, p.891-893) and even though some tissue wastage and weight loss is present, it may be over shadowed by the oedema. The oedema begins in the feet and legs and then spread to the hands, face and body. With oedema, the child may appear "plump". The children are apathetic, have little interest in surroundings, and are listless and dull (Torún and Chew, 1994, p.961-963; Berdanier, 1995, p.153; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Torún, 2006, p.891-893; Heimburger, 2006, p. 833).

In patients with kwashiorkor there is retention of sodium, low blood pressure and signs of hypovolemia and infections. Patients with nutritional oedema are metabolically different from those with marasmus (Golden and Golden, 2000, p.522; Heimburger, 2006, p.833). Mortality in these children is much higher than in marasmic children (Heimburger, 2006, p. 833).

2.3.3.2 MARASMUS

An inadequate intake of macronutrients together with the increased macronutrient requirements needed for maintenance and growth, lead to loss of body tissue. Marasmus is characterized by failure of linear growth (stunting) and loss of weight (wasting) (Jackson and Golden, 1991, p.134). Marasmus is linked to severe deprivation or impaired absorption of protein, energy, vitamins and minerals (Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198).

Anthropometrically, marasmus is seen as a weight-for-age below 60% of the expected weight for age (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198). Marasmus is generally characteristic of urban living,

where factors such as cessation of breastfeeding and the incorrect use of formula milk, result in the development of marasmus (Monckeberg, p.121, 1991; NDoH, 2005b).

Mortality in these children is relatively low if there is no underlying illnesses or infections (Heimburger, 2006, p.833), with a global contribution to child deaths of about 1.7 million per year (Jackson *et al*., 2006).

Table 2.7 shows the comparison of marasmus and kwashiorkor according to clinical setting, time course to develop, clinical features, laboratory findings, clinical course and mortality.

Table 2.7 Comparison of marasmus and kwashiorkor (Heimburger, 2006, p.833)

	MARASMUS	**KWASHIORKOR**
Clinical setting	Low energy intake	Low protein intake during stress state
Time course to develop	Months or years	Weeks
Clinical features	Starved appearance Weight < 80 % standard for height Triceps skin fold < 3 mm Midarm muscle circumference < 15 centimeter (cm)	Well-nourished appearance Easy hair pluckability Edema
Laboratory findings	Creatinine-height index < 60 % standard	Serum albumin < 2.8 g/dL Total iron-binding capacity < 200μg/dL Lymphocytes < 1,500/cubic millimetre (mm^3) Anergy
Clinical course	Reasonably preserved responsiveness to short-term stress	Infections Poor wound healing, decubitus ulcers, skin breakdown
Mortality	Low, unless related to underlying disease	High

2.3.3.3 MARASMIC KWASHIORKOR

Pure conditions of marasmus and kwashiorkor are uncommon as there are many cases which are not purely one or the other, but present rather with signs of both. This can be due to changes in diets and seasons. The term marasmic kwashiorkor therefore is used to describe the wasted form of PEM (as with marasmus, there is no subcutaneous fat), which has the characteristics of dermatoses and/or oedema that is seen with kwashiorkor (Torún and Chew, 1994, p.963; Wittenberg, 2004, p.207; Torún, 2006, p.893).

Infections such as diarrhoea can also change the symptoms and signs that a child presents with (Wittenberg, 2004, p.207) and therefore marasmic kwashiorkor can develop

when a marasmic child experiences stress such as surgery, trauma or sepsis (Torún and Chew, 1994, p.963; Torún, 2006, p.893; Heimburger, 2006, p.834). Marasmic kwashiorkor presents as a weight-for-age of less than 60% expected weight, with oedema (Wittenberg, 2004, p.203), where the oedema disappears after nutritional treatment and the child then resembles a marasmic child (Torún and Chew, 1994, p.963; Torún, 2006, p.893).

2.4 ASSESSMENT OF NUTRITIONAL STATUS

The clinical, biochemical and physiologic characteristics of PEM vary according to the severity of the disease, the age, the presence of nutritional deficits and infections, and the predominance of energy or protein deficiency. To diagnose the malnourished child, a dietary history and the clinical features present should be evaluated (Torún and Chew, 1994, p.959; Torún, 2006, p.889). Assessments are used to provide information on the nutritional and health status of children and are an indirect measure of quality of life of a community or population (Shetty, 2002, p. 321).

In malnutrition, the main clinical feature is weight loss. Subcutaneous fat tissue is decreased and children with chronic PEM show growth retardation in terms of both weight and height. Children's physical activity and energy levels are decreased with a reduced attention span, lack of liveliness, frequent episodes of diarrhoea and varying degrees of apathy. Immunocompetence, GI functions and altered behaviour are also present (Torún and Chew, 1994, p.960; Torún, 2006, p.891).

2.4.1 ANTHROPOMETRY

Nutritional status can be measured using anthropometric measurements (NDoH, 2005a), even in less advanced cases of malnutrition. The benefit of these measurements is that they are less invasive and costly than biochemical evaluation (Zere and McIntyre, 2003). The nutritional status of children under five years is one of the best predictors of child survival (NDoH, 2005a).

The choice of which anthropometric measurements to use depends on their simplicity, accuracy and sensitivity. The availability of measuring instruments and the existence of reference standards for comparison are also important. International or universal standards, such as the NCHS and newer WHO standards for children under five years can be used because of the following: most children have the potential to grow the same

regardless of ethnic background; the relationship of weight and height stays relatively constant in healthy children and the reference standards are not an ideal or target but just used for comparison (Torún and Chew, 1994, p.959; Torún, 2006, p.890).

Both the NCHS and WHO standards use SD from the median and the results are referred to as Z-scores. A child who has Z-scores within -+1SD is within the normal range. Children with the lower portion of these ranges are classified as "moderately malnourished". Children who are more than 3SD below the normal have severe malnutrition. In children older than six months, a deficit of 5% in height-for-age or 10% in weight-for-height is more or less equal to one Z-score (Torún and Chew, 1994, p.959; Golden and Golden, 2000, p.518; Torún, 2006, p.890) (Table 2.8). The accepted anthropometric cut-off for the diagnosis of undernutrition is –2SD (z score) and indicates an increased risk of morbidity and mortality (Shetty, 2002, p. 321).

The new WHO reference standards can be used globally and came into effect in April 2006. Six countries growth standards were used to develop these standards, whereas the NCHS standards were only based on the standards of one country. The main idea of the new WHO standards is to see how children should be growing for the best health outcome, rather than just showing how the average child is growing. The new standards also take into consideration the use of length and height and body mass index (BMI), which was never used in the NCHS standards. The growth charts therefore include length or height for age, weight for age and weight for length or height. The growth charts are also available for boys and girls, infants to one year and children to five years and the BMI of infants to five years of age. The standards used were for healthy, breastfeeding children and their growth patterns. The new WHO standards also look at the milestones that children should reach at specific ages, whereas milestones were not part of the NCHS standards (WHO, 2006).

Table 2.8 Classification of severity of current ("wasting") and past or chronic ("stunting") PEM in infants and children, based on the weight for height and height for age (Torún, 2006)

	NORMAL	MILD	MODERATE	SEVERE
Weight for height (deficit = wasting)	90-110 (± 1 Z-score)	80-89 (-1.1 to –2 Z-score)	75-79 (-2.1 to –3 Z-score)	< 75, or with oedema (< -3 Z-score)
Height for age (deficit = stunting)	95-105 (± 1 Z-score)	90-94 (-1.1 to –2 Z-score)	85-89 (-2.1 to –3 Z-score)	<85 (< -3 Z-score)

Simple measurements of weight, height and waist circumference can identify individuals who are obese, thin, stunted in growth or wasted. Simple anthropometry cannot however, determine if a malnourished infant is overhydrated or underhydrated. Despite this, body weight is the best and most reliable of all anthropometric measurements because of its sensitivity, precision and objectivity (Garrow, 2005, p.74).

Table 2.9 indicates anthropometric measurements that can be used for nutritional assessment.

Table 2.9 Recommended measurements for nutritional assessment (Bates *et al*., 2005)

AGE GROUP (YEARS)	PRACTICAL FIELD OBSERVATIONS	MORE DETAILED OBSERVATIONS
0 – 1	Weight Length	Head and arm circumference Triceps and subscapular skinfolds
1 – 5	Weight Length / Height Arm circumference	Triceps and subscapular skinfolds
5 – 20	Weight Height Arm circumference	Triceps, subscapular and medial calf skinfolds Calf circumference
> 20	Weight Height	Arm and calf circumference Triceps, subscapular and medial calf skinfolds Waist and hip circumference (overnutrition only) Demispan (elderly subjects)

2.4.1.1 WEIGHT

Anthropometry provides a way of estimating the magnitude of a deficiency (Duggan and Golden, 2006, p.519). An immediate effect of malnutrition is weight loss due to muscle wasting and loss of subcutaneous tissue (Marcondes, 1991, p.74). Weight-for-age is most often used as an indicator of children's nutritional status and it is the most widely used in developing countries (Caulfield *et al*., 2004).

The NCHS standards show the child's anthropometry as a percentage of the median for the standard populaton. According to the WHO, appropriate weight and weight-for-height reflects proper body proportion because weight-for-height is sensitive to acute growth changes (Shetty, 2002, p. 321). Severe acute malnutrition is defined by a very low weight-for-height seen as <-3 Z-score of the median of the NCHS or WHO standards. It is also classified by the presence of visible severe wasting, or the presence of nutritional oedema (WHO, 2007b).

Weight measurements must always be interpreted carefully for two important reasons: the presence of oedema can cause the child's true body weight to be overestimated; and the absence of oedema with a low weight is due to chronic energy deficiency ("stunting") rather than recent weight loss ("wasting"). Weight must be interpreted together with a measurement of length (or height if over two years) (Williams, 2005, p.406). Total weight is used as an indicator even though skeletal and essential organ weight has a slow tissue turnover (Duggan and Golden, 2006, p.519).

2.4.1.2 HEIGHT / LENGTH

Height is measured in infants and young children less than 24 months of age by taking recumbent or supine length when the child is lying down, whether they can stand or not. Height measure is done in children two years to five years, in a standing position (NDoH, 2005b). When a measuring board is used, the child must be held firmly to make sure that the head and feet are touching the head and foot panels respectively and the knees are kept down (Beatty, 2004, p.9).

All children should be measured for height at least every three months (NDoH, 2007). Height measurement can be used with weight to measure overall growth for comparison to growth standards (Beatty, 2004, p. 9). According to the WHO, the appropriate height-for-age of a child reflects linear growth, and can therefore measure long-term growth faltering or stunting (WHO, 2007b).

2.4.1.3 MID UPPER ARM CIRCUMFERENCE (MUAC)

When age is not available, weight alone is insufficient to differentiate between an underweight child and a child who is short with an adequate weight. The MUAC works well in field conditions where no scale is available. MUAC is not sensitive, but it can differentiate between moderately and severely malnourished children (Torún and Chew, 1994, p.960; Torún, 2006, p.891). Mother and Child nutrition publication of 2009 found that MUAC was a sentitive makrer for screening and is a better indictor of mortality, and ideal for assessing children that will need more care (Mother and Chid nutrition, 2009b).

There is very little change in a child's arm circumference between the ages of one to five years. This measurement therefore gives a simple measure of wasting. MUAC is a better prognostic indicator for mortality than weight-for-height (Golden and Golden, 2000, p.518; Mother and Child Nutrition, 2009b). Children between twelve to 59 months old can

be screened using the MUAC and when a child is older than six months but longer than 65cm, the MUAC can also be used (Mother and Child Nutrition, 2009a). According to the WHO (2007a) children aged six to 59 months, with an arm circumference less than 110mm are severely and acutely malnourished (Table 2.10) (Collins *et al*., 2006; WHO, 2007a).

In underprivileged communities MUAC is the best indicator for identifying children at high risk of death from malnutrition (Bentley and Lawson, 1988; Collins *et al*., 2006). The use of MUAC as an anthropometric indicator for screening and admitting children into community-based therapeutic care, gives communities a chance to help and take responsibility for their own children. No complicated or expensive measuring equipment is needed and MUAC is easy to teach to community-based workers, making it practical, especially in poor communities (Collins *et al*., 2006).

Table 2.10 Classification of malnutrition in children aged 1-5 years by mid upper-arm circumference (Golden and Golden, 2000, p.518)

Circumference (cm)	**Level of nutrition**
> 14	Normal
12.5 – 14.0	Mild / moderate malnutrition
< 12.5	Severe malnutrition

2.4.2 BIOCHEMICAL FEATURES OF MALNUTRITION

Biochemical alterations are not consistent in mild and moderate PEM. Plasma levels of nutrients vary, with moderately low levels, but do not really reflect the body stores. Laboratory data include low urinary urea nitrogen and hydroxyproline excretions, 3 metylhistidine, altered plasma patterns of free amino acids, and a reduced number of circulating lymphocytes. In kwashiorkor the ratio of nonessential to essential amino acids is elevated while in maramus it remains normal (Torún and Chew, 1994, p.963; Torún, 2006, p.893). Also see Table 2.11 for other laboratory features associated with severe malnutrition.

Children with kwashiorkor have reduced urinary creatinine excretions causing a low creatinine-height index, whereas marasmic children have a normal to low index. In kwashiorkor the serum levels of free fatty acids are often elevated (Torún and Chew, 1994, p.963; Torún, 2006, p.893).

Hemoglobin and hematocrit are lower in kwashiorkor than in marasmus (Torún and Chew, 1994, p.963; Torún, 2006, p.893). Hemoglobin levels are often less than 8g/dL in children with kwashiorkor (Chitambar and Antony, 2006, p.1458).

When infection is present and fever is observed, plasma concentrations of iron and zinc fall, causing hypoferraemie, with the greatest fall in plasma concentrations occurring during fever. Hypoferraemie causes changes in plasma concentrations of iron-binding proteins (Turnham, 2005, p.259). Laboratory findings show a reduced iron-binding capacity of less than 35.8 micromol (μmol) per litre (L) (Morgan and Weisnier, 1998, p.171; Heimburger, 2006, p.833). The iron-binding proteins help with the uptake of iron through the reticuloendothelial system or through the removal and reutilization of hemoglobin from erythrocytes (Turnham, 2005, p.259). Iron deficiency is less frequent in marasmus than in kwashiorkor (Monckeberg, 1991, p.124).

Laboratory findings that can be used for diagnostic purposes include transferrin levels of less than 1.5 g/L (Morgan and Weisnier, 1998, p.171; Heimburger, 2006, p.833). High circulating levels of ferritin, especially in patients with kwashiorkor suggest that this causes oedema by acting as an antidiuretic. Ferritin levels are related to mortality and children who died had levels higher than 2.5 g/L. High ferritin levels are seen with increased iron storage. Children who die of malnutrition often have increased levels of hepatic iron (Jackson and Golden, 1991, p.137).

Laboratory findings that can be used for diagnostic purposes include severely depressed levels of serum protein such as albumin (< 28 g/L). Even though the serum albumin levels are reduced, they usually don't drop below 28 g/L (Morgan and Weisnier, 1998, p.171; Heimburger, 2006, p.833).

Low serum albumin (hypoalbuminemie) and oedema is a feature of kwashiorkor and is caused by a reduced hepatic albumin synthesis (Torún, 2006, p.884; Piercecchi-Marti *et al*., 2006). According to Müller and Krawinkel (2005) the protein concentrations in plasma are not different between marasmic children and those with kwashiorkor, whereas Torún and Chew (1994, p.963) and Torún (2006, p.893) found that the serum concentrations of total protein and albumin are normal or moderately low in marasmus.

Except for a low protein or low quality protein intake and chronic blood loss, patients can also develop severe anaemia if there is a dietary deficiency of iron, folic acid (Jackson and Golden, 1991, p.139; Torún and Chew, p.955, 1994; Torún, p.886, 2006), vitamin B12, copper, vitamin C and riboflavin (Jackson and Golden, 1991, p.139). Monckeberg (1991, p.124) found that anaemia is absent or mild in patients with uncomplicated marasmus, whereas Jackson and Golden (1991, p.139) report varying degrees of clinical anaemia, with it being mild in the absence of unusual blood loss (Torún and Chew, 1994, p.955; Torún, 2006, p.886).

Anaemia can also result because of the negative acute phase response associated with infections and injury (Bates *et al*., 2005; Hoffer, 2006, p.737). The response lowers serum albumin concentrations by sending the albumin into the extravascular space and increasing the catabolism. Hypoalbuminemia will persist if enough protein is not provided (Hoffer, 2006, p.737). A reduced albumin should be used as a marker of disease rather than a measure of nutritional status, where pre-albumin is more suitable, because a low albumin can be the result of an inflammatory response or increased intestinal losses (Chudleigh and Hunter, 2005, p.436).

Anaemia at a critical time, can permanently handicap children in their scholastic development (Shetty, 2002, p. 322). Children with iron deficiency anaemia have poor cognitive and motor development and behavioural problems. There is a delay in the development of cognitive skills, which can be reversed with treatment (Williams, 2005, p.408).

Anaemia may be normocytic and normochromic. Bone marrows are often megaloblastic. Megaloblastosis, resulting from folate deficiency, is usually only seen after protein treatment. Ascites seems to be related to a reduced osmolarity in the blood, caused by severe anaemia (Müller and Krawinkel, 2005).

Cellular immune function is depressed, reflected by lymphopenia (<1500 lymphocytes/mm^3) in older children (Heimburger, 2006, p.833). Leukocyte counts tend to be low in malnourished children. The percentage of sideroblasts in the bone marrow is high (Monckeberg, 1991, p.124). With malnutrition there is a decrease in circulating erythrocyte mass. The erythrocytes show a variety of abnormal morphologies. The destruction of the erythrocytes is associated with a vitamin E deficiency. This causes

alterations to the membrane function and permeability (Jackson and Golden, 1991, p.139). Erythropoietin and reticulocytes are produced in response to acute hypoxia (Torún and Chew, 1994, p.955; Torún, 2006, p.886).

The differences between kwashiorkor and marasmus and the confusing clinical features of kwashiorkor are difficult to understand and can possibly be explained by the imbalance between free radical production and their safe disposal (Oyelami and Ogunlesi, 2007; Gallagher, 2008, p.66). Free radicals are disposed of by a protective mechanism that includes glutathione peroxidase. Oxidant stress in kwashiorkor can reduce the activities of glutathione. In marasmus glutathione levels are normal and in kwashiorkor they are often depleted. The depleted glutathione level causes an increase in the activity of the intracellular sodium pump and high intracellular sodium content (Oyelami and Ogunlesi, 2007).

Children with marasmus or kwashiorkor have decreased blood glucose, serum insulin and growth hormone levels. In marasmus there is a decrease in thyroid hormone levels (Berdanier, 1995, p.154). In marasmus, amino acids are used to maintain the metabolism in the liver and ketone bodies increase in the blood (Piercecchi-Marti *et al*., 2006). In marasmic kwashiorkor biochemical features of both marasmus and kwashiorkor are seen (Torún and Chew, 1994, p.963; Torún, 2006, p.893).

The fluid and electrolyte metabolism of malnourished children is important as they may appear dehydrated due to a decrease in total body water, when they are actually overhydrated (Torún and Chew, 1994, p.957; Torún, 2006, p.887; Garrow, 2005, p.74). Marasmic children often display vomiting and diarrhoea and therefore it seems important to treat them with intravenous fluid, but this is not always indicated and can be dangerous (Garrow, 2005, p.74).

Chronic hypovolemia can lead to secondary hyperaldosteronism, which complicates fluid and electrolyte balance. Muscular dystrophy mobilizes much of the body's potassium and it is then lost through urinary excretion. Affected children do not show signs of hyperkalemia. Their immune system is depressed and often the body cannot even produce the fever that is typical of inflammation (Müller and Krawinkel, 2005). The presence of hyponatremia is an indicator of poor prognosis (Jackson and Golden, 1991, p.136).

Oxidative stress causes changes to structural lipids of cell membranes, which causes them to leak sodium and potassium, which can contribute to the development of oedema (Duggan and Golden, 2006, p.520). The intracellular compartment of the body is more depleted than the extracellular and this causes a deficiency of intracellular potassium and magnesium (Williams, 2005, p.406). Up to 50% of intracellular potassium may be replaced by sodium. The brain is spared even though muscle potassium is depleted (Monckeberg, 1991, p.125). Body potassium is also decreased because of the reduced muscle protein (Torún and Chew, 1994, p.957; Torún, 2006, p.887). Even though plasma potassium is low in kwashiorkor, it does not reflect on the status of the whole body (Jackson and Golden, 1991, p.135).

Cellular exchange of sodium and potassium occurs when potassium is lost and there is an increase in intracellular sodium (Torún and Chew, 1994, p.957; Torún, 2006, p.887). Plasma sodium tends to be low even though there is an excess of total body sodium. The sodium content in the intracellular space, muscles, liver, erythrocytes and leukocytes are often increased (Jackson and Golden, 1991, p.136).

Table 2.11 Laboratory features of severe malnutrition (Müller and Krawinkel, 2005)

Blood or plasma variables	The information derived
Hemoglobin, hematocrit, erythrocyte count, mean corpuscular volume	Degree of dehydration and anemia; type of anemia (iron/folate and vitamin B12 deficienc, hemolysis, malaria)
Glucose	Hypoglycemia
Electrolytes and alkalinity	
Sodium	Hyponatremia, type of dehydration
Potassium	Hypokalemia
Chloride, pH, bicarbonate	Metabolic alkalosis or acidosis
Total protein, transferring, (pre-) albumin	Degree of protein deficiency
Creatinine	Renal function
C-reactive protein, lymphocyte count, serology, thick and thin blood films	Presence of bacterial or viral infection or malaria
Stool examination	Presence of parasites

2.5 IMPACT OF MALNUTRTION ON VARIOUS ORGANS AND SYSTEMS

Loss of appetite (anorexia) is a common feature. Causes can be infection, nutrient deficiency and liver dysfunction (Golden and Golden, 2000, p.519). Malnutrition causes a variety of internal and bone lesions, which can lead to death. Cachexia, severe lesions of the liver, pancreas, brain and bone are related to inadequate protein, vitamin and energy intake (Piercecchi-Marti *et al*., 2006).

During malnutrition the physiology of the body changes to conserve nutrients and therefore the body reduces the amount of work performed. Unlike an undernourished child, a healthy individual can maintain digestive, absorptive, hepatic and renal capacity to deal with environmental changes. Reserves of tissue and functional capacity are "expensive" for the body to synthesize, replace and maintain. This capacity is not working in malnutrition and there is a reduction in the functional capacity of organs and energy requirements (Golden and Golden, 2000, p.520). The lymph nodes in malnourished children are easily palpable (Torún and Chew, 1994, p.961; Torún, 2006, p.892).

Growth results in high metabolic demands during infancy (the first year of life). Early in childhood various organs undergo their most rapid growth. Brain growth is almost completed in the early years of childhood. Prolonged and severe nutrient restriction at this age may be associated with lifelong functional deficits (Williams, 2005, p.379 and 387).

It is difficult to define if PEM, AIDS and / or other body wasting diseases cause the visible signs (Torún and Chew, 1994, p.961; Torún, 2006, p.892). Wide spectrums of clinical features are seen and are the consequence of environmental factors. Virtually all body systems or functions are affected (Jackson and Golden, 1991, p.134-135). Some of the features are acute gastroenteritis, dehydration, respiratory infections and eye lesions caused by hypovitaminesis A. Diarrhoea may be present. Systemic infections may lead to septic shock, intravascular clots and high mortality rates (Torún and Chew, 1994, p.961; Torún, 2006, p.892) and in severe cases it may lead to stupor/coma. Death is usually caused by infection (Katz *et al*., 2005).

Amino acids, such as leucine, and various micronutrients such as, zinc, copper, molybdenum and possibly vitamin A can influence linear growth. The cytokines that are produced in response to infection can slow down bone growth. Frequent or chronic infections, such as HIV and worm infestation can cause growth to falter (Duggan and Golden, 2006, p.521).

The skin and intestine are more affected than the visceral organs and central nervous system. The tonsils are atrophic. Angular stomatitis, lingual atrophy, follicular hyperkeratosis, oral candidiasis and specific signs of nutrient deficiencies, such as in the eyes (vitamin A deficiency) occur (Golden and Golden, 2000, p.520).

Clinical features of both types of PEM are summarized in Table 2.12 and Appendix A.

Table 2.12 Features of marasmus and kwashiorkor (Torún and Chew, 1994, p.961-963; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.198; Torún, 2006, p.891-893)

Marasmus	Kwashiorkor
• Infancy (less than two years) • Severe, deprivation or impaired absorption of protein, energy, vitamins and minerals • Develops slowly, chronic PEM • Severe weight loss • Severe muscle wasting with fat loss • Growth: < 60 % weight-for-age • No detectable edema • No fatty liver • Anxiety, apathy • Appetite may be normal or impaired • Hair is sparse, thin and dry, easily pulled out • Skin is dry, thin and wrinkled	• Older infants and young children (1 to 3 years) • Inadequate protein intake or more commonly infectious • Rapid onset: acute PEM • Some weight loss • Some muscle wasting, with retention of some body fat • Growth: 60 to 80 % weight-for-age • Edema • Enlarged, fatty liver • Apathy, misery, irritability, sadness • Loss of appetite • Hair is dry and brittle, easily pulled out, changes in color, becomes straight • Skin develops lesions

2.5.1 BODY COMPOSITION AND OEDEMA

During the clinical examination, used to assess the severity of the malnutrition, changes in body composition are seen (Jackson and Golden, 1991, p.134-135). Measurement of skin fold thickness is misleading in oedematous patients. With a massive fatty liver containing as much as 50% of the total body fat, the skin fold thickness gives a false impression of the amount of fat in the body (Jackson and Golden, 1991, p.138). The body's chemical composition is also altered because of the changes in the size of the organs (Golden and Golden, 2000, p.521).

As the composition of the body changes in PEM, the subcutaneous fat may disappear and muscle mass may be reduced by more than half (Golden and Golden, 2000, p.521). Muscle tone and strength are reduced (Torún and Chew, 1994, p.962; Torún, 2006, p.892). Losses in fat, muscle, skin, brain, liver, kidneys and intestine contribute to the total loss of body weight, but the losses do not occur in proportion. Muscle wasting is seen because of the loss of soluble and contractile proteins, whereas collagen is conserved (Jackson and Golden, 1991, p.135).

An increase in total body water is seen in kwashiorkor. Plasma volume is expanded when expressed in relation to body weight but normal in relation to the child's height. The total water content of muscle, skin, liver and leukocytosis is increased. The expansion of

the extracellular space with oedema is the main sign of kwashiorkor, but its pathogenesis is unclear (Jackson and Golden, 1991, p.137).

The dominant feature is soft, pitting, painless oedema, in the feet and legs, the perineum, upper extremities and face (Torún and Chew, 1994, p.961; Torún, 2006, p.892). The swelling is first seen in the feet of the child. As it increases, it spreads upwards to the legs, thighs and abdomen, until the child is completely swollen. The oedema is gravitational, with only the hands and forearms being swollen, whereas the shoulders and upper arms are bony and extremely thin. These features distinguish the children from those with nephrotic syndrome. Ascites occurs in very oedematous children (Pereira, 1991, p.143).

Oedema has also been strongly linked to hypoalbumineamia. Other factors that can lead to oedema are potassium deficiency, leading to water and sodium retention, excessive intake of water and sodium and the loss of fluid due to high capillary permeability in infection. Infections and the inflammatory response caused by toxins contribute to the oedema (Torún and Chew, 1994, p.958; Torún, 2006, p.889). The sodium and water retention is about 10-30% of the body weight but can even reach 50% in severe cases (Golden and Golden, 2000, p.519).

Patients with kwashiorkor have trunkal and limb fat, which can obscure muscle wasting (Shetty, 2002, p. 320; Whitney and Rady, 2005, p.199). There is a weight deficiency even though the oedema makes it difficult to determine, but it is not as severe as in marasmus. Height may be normal or retarded, depending on the current episode and the past nutritional history (Torún and Chew, 1994, p.962; Torún, 2006, p.892).

Full cheeks or jowls, are associated with oedematous malnutrition (Golden and Golden, 2000, p.519). The full cheeks are also referred to as a “moon face”. Purpuric spots may be seen on the cheeks of severely malnourished children with kwashiorkor (Pereira, 1991, p.143-144).

In marasmus the children are wasted and grossly underweight without oedema, fatty liver and skin changes (Strobel and Ferguson, 2005, p.487). Patients with non-oedematous PEM have a “skin and bones” appearance because of centralized muscular wasting and absence of subcutaneous fat (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994,

p.952 & 961; Berdanier, 1995, p.154; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Shetty, 2002, p. 320; Whitney and Rady, 2005, p.198). Marasmus is easy to identify in a patient because of the emaciated appearance (Torún, 2006, p.881). The loss of muscle is usually in the shoulders and buttocks (Golden and Golden, 2000, p.519). Children with marasmus have prominent ribs, with very thin limbs that have little muscle or adipose tissue (Monckeberg, 1991, p.124; Berdanier, 1995, p.154; Shetty, 2002, p. 320). The Bichat fat pads are the last subcutaneous adipose depots to disappear and this causes sunken cheeks. It gives the marasmic child's face the appearance of a monkey's or an old person's face (Monckeberg, 1991, p.124; Torún and Chew, 1994, p.961; Torún, 2006, p.892).

The most obvious clinical sign is height and weight retardation and in some cases growth can come to a complete stop (Berdanier, 1995, p.154; Piercecchi-Marti *et al*., 2006). Marasmic patients have less than 60% of the expected weight for their height and retardation in longitudinal growth (Torún and Chew, 1994, p.961 and 964; Torún, 2006, p.892 and 95).

The skin is dry, thin and wrinkled and tends to crack because it is less elastic. The children's hair is sparse, thin and dry and is easily plucked out (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198).

2.5.2 CARDIOVASCULAR SYSTEM

The low intakes lead to wasting and weakening of the muscles, such as the heart (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198). Malnourished children are weak and cannot stand on their own. Heart rate, blood pressure and body temperature is low and tachycardia may be present (Torún and Chew, 1994, p.961; Torún, 2006, p.892).

During malnutrition the heart shows macroscopic and histological evidence of pathological changes and wasting. In severe cases the cardiac function is altered. The reduced cardiac output is due to a decrease in heart rate and stroke volume and longer circulation time (Jackson and Golden, 1991, p.139; Golden and Golden, 2000, p.520). Decreased output leads to a decrease in renal plasma flow and glomerular filtration

(Torún and Chew, 1994, p.955-956; Torún, 2006, p.886). Children can develop a shift in fluid, which can lead to heart failure (Jackson and Golden, 1991, p.139; Golden and Golden, 2000, p.520). Changed cardiovascular reflexes can cause postural hypotension (Torún and Chew, 1994, p.955-956; Torún, 2006, p.886).

Tissue oxygen needs are linked to a reduction in haemoglobin concentration and red cell mass. Malnourished patients have a decreased oxygen requirements because of a reduction in lean body mass and low levels of physical activity (Torún and Chew, 1994, p.955; Torún, 2006, p.885-886).

2.5.3 IMMUNE SYSTEM

Inadequate nutrition or undernutrition can lead to changes in immune function and cause secondary immunodeficiencies (Strobel and Ferguson, 2006, p.488). During PEM the important tissues and cells of the immune systems are reduced in size and number making the body susceptible to infection (Bentley and Lawson, 1988, p.43; Jackson and Golden, 1991, p.138). TB and bronchopneumonia infections are important causes of secondary problems. Measles is a viral complication, which causes high mortality rates in malnourished infants (Bentley and Lawson, 1988, p.43). Infections are characterized by fever, leucocytosis, tachycardia, pus formation, tachypnoea and local inflammation, but when these responses are not seen, life-threatening infections go undiagnosized (Golden and Golden, 2000, p.521).

During malnutrition the bactericidal and fungicidal activity of leukocytes is lower. Serum immunoglobulin levels are increased, because of the repeated infections. Wel-nourished infants may have normal serum immunoglobulin levels. Secretory IgA is decreased in tears, nasopharyngeal secretions, and the jejunal mucosa. The lymphocytes from the thymus and other components of the lymphatic system are atrophic. Production of interferon is decreased in marasmic children (Monckeberg, 1991, p.124).

Skin and mucous membranes can become structurally damaged and can produce a small inflammatory response (Jackson and Golden, 1991, p.138). Except for the alterations seen in the structure and integrity of the skin and mucosa, a decrease in lysozyme concentration in the saliva and tears and polymorpho nuclear leukocytes is also present (Monckeberg, 1991, p.124).

Some of the changes associated with PEM are decreased total and helper T-cell counts, reversal of the helper or suppressor cell ratio, cutaneous energy and decreased lymphokine production (Torún and Chew, 1994, p.956; Eley and Hussey, 1999; Torún, 2006, p.887). The T-lymphocytes from the spleen and lymph nodes are also depleted. Monokine metabolism is altered and there is a decrease in activity of interleukin-1. This leads to a low proliferation of T-cells. Because of these changes, malnourished patients are more susceptible to gram-negative bacterial sepsis. Phagocytosis, chemotaxis and intracellular killing are impaired because of the defects in the complement functional activities (Torún and Chew, 1994, p.956; Torún, 2006, p.887).

The complications associated with malnutrition cause less important infectious diseases to become more severe. With nutritional rehabilitation, abnormal immune function improves (Torún and Chew, 1994, p.956; Torún, 2006, p.887). The response to antigenic stimuli is normal in some and decreased in others, but when nutritional conditions start to improve more than 90% of infants are able to react to antigens again (Monckeberg, 1991, p.124).

2.5.4 GASTRO-INTESTINAL SYSTEM

Anorexia, postprandial vomiting and diarrhoea are common in kwashiorkor (Torún and Chew, 1994, p.962; Torún, 2006, p.892). Changes in GI structure and function lead to malabsorption and worsen nutritional status. GI function in kwashiorkor is seriously disturbed and the gastric mucosa shows structural abnormalities (Jackson and Golden, 1991, p.139). In marasmus the mucosa of the small intestine is normal but its thickness is decreased (Monckeberg, 1991, p.125). Stomach mucosa is often atrophied, gastric acid secretion is reduced and responses to stimulation by pentagastrin are poor. All these abnormalities influence the protective mechanisms against bacterial overgrowth (Jackson and Golden, 1991, p.139).

Malnourished children are susceptible to repeated and chronic gut infections and infestations and these worsen nutritional status. The bacterial contamination is often responsible for the abnormalities in bile salts, impaired intestinal digestion and absorption. Some enteric bacteria can damage the integrity of the brush border of enterocytes in the upper intestine (Jackson and Golden, 1991, p.139: Torún and Chew, 1994, p.957; Torún, 2006, p. 887). The intestine has reduced peristalsis, motility and increased intestinal

transit time (Torún and Chew, 1994, p.962; Golden and Golden, 2000, p.521; Torún, 2006, p.892).

Diarrhoea is almost always present. The diarrhoea may be because of the body not being able to synthesize the enzymes that are needed to use ingested food or because of infections and parasites (Berdanier, 1995, p.153). Diarrhoea aggravates malabsorption. Malabsorption can disappear after nutritional recovery, as long as there is no food intolerance (Torún and Chew, 1994, p.957; Torún, 2006, p. 887).

Microvilli damage is more pronounced in kwashiorkor than in marasmus (Bentley and Lawson, 1988, p.43). The cellular enzymes and transport systems are compromised and the mucosa becomes flattened and the mitotic figures in the crypts are reduced (Golden and Golden, 2000, p.521; Wittenberg, 2004, p.201) to about one third of the values found in well-nourished children (Monckeberg, 1991, p.126). Villi are shortened and the epithelial cells of the villi with the brush-border disaccharidasis become injured and this leads to carbohydrate malabsorption (Bentley and Lawson, 1988, p.43; Jackson and Golden, 1991, p.139; Monckeberg, 1991, p.126; Wittenberg, 2004, p.201) and extensive inflammatory infiltration in the lamina propria (Jackson and Golden, 1991, p.139). Cell renewal is slowed in PEM. Little is known about the digestive capacity of the jejunal mucosa in PEM (Monckeberg, 1991, p.126).

The enzymes responsible for protein digestion, absorption and transport are less active (Berdanier, 1995, p.154) and with severe protein deficiency there is also impaired intestinal absorption of lipids and decreased glucose absorption (Monckeberg, 1991, p.126; Jackson and Golden, 1991, p.139; Torún and Chew, 1994, p.957; Torún, 2006, p.887). Fecal fat excretion is high in comparison to children without malnutrition (Monckeberg, 1991, p.126).

The greater the protein deficit, the greater is the functional impairment. A decrease in gastric and exocrine pancreatic concentrations occur, but with normal to low enzyme and bile acid concentrations. All these changes will also impair absorption (Jackson and Golden, 1991, p.139; Torún and Chew, 1994, p.957; Torún, 2006, p.887).

The abdomen is usually swollen in children with kwashiorkor. This is caused by gas in the intestine and not only the enlarged liver. Bowel sounds are high-pitched and

infrequent (Golden and Golden, 2000, p.520). Abdominal swelling can also be due to ascites, which can be caused by kidney failure, liver disease or congenital heart disease. The basic principle is the same as with the oedema related to malnutrition. Both are due to an imbalance of the pressure between the inside and outside, in this case, of the abdomen. This pressure difference is due to a high portal blood pressure and decreased albumin (Nabili and Davis, 2005).

2.5.5 LIVER

Liver damage indicates a poor prognosis in kwashiorkor. Proteins synthesized by the liver act as carriers for other compounds such as transferrin. Increased levels of bilirubin, hepatocellular enzymes, vitamin B12 and ferritin occur (Jackson and Golden, 1991, p.139). Muscle breakdown is reduced and free amino acids decrease. The supply of amino acids in the muscle is lower and this leads to lower protein synthesis in the liver, particularly albumin (Torún and Chew, 1994, p.958). With acute infection, acute-phase proteins are reduced and this also causes a decrease in albumin (Jackson and Golden, 1991, p.139).

In energy insufficient diets, the enlarged liver is fatty because of the inability of the child to synthesize the apo-B-lipoproteins that is needed to make the transport protein to transport the lipids out of the liver (Pereira, 1991, p.143-144; Berdanier, 1995, p.153-154; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.199). Increased hepatic fatty acid synthesis, impaired lipolysis and the decreased transport proteins result in fatty infiltration of the liver, which makes the liver larger resulting in hepatomegaly (Pereira, 1991, p.143-144; Torún and Chew, 1994, p.958; Berdanier, 1995, p.153-154; Sizer and Whitney, 2000, p.196; Wittenberg, 2004, p.201; Whitney and Rady, 2005, p.199). The fatty liver has a decreased ability to clear poisons from the body, prolonging their toxic effects (Pereira, 1991, p.143-144; Berdanier, 1995, p.153-154; Sizer and Whitney, 2000, p.196; Whitney and Rady, 2005, p.199).

Fat accumulation varies and in severe cases it can lead to hepatocellular failure. The fat accumulation starts in the peri-portal areas and then moves to the area around the central vein. The fat clears after appropriate refeeding (Jackson and Golden, 1991, p.140; Wittenberg, 2004, p.200-201). Impaired lipoprotein synthesis limits the liver's use of fat. During fasting, malnourished children can use fat as a source of energy (Jackson and Golden, 1991, p.140).

The liver is smooth, firm and not usually tender. Half of the weight of the liver can be fat (Golden and Golden, 2000, p.519). The size of the liver can differ (Pereira, 1991, p.143-144; Berdanier, 1995, p.153). Liver steatosis leads to death if untreated and is more prevalent in kwashiorkor than marasmus (Piercecchi-Marti *et al*., 2006).

2.5.6 RENAL SYSTEM

Renal function is reduced in both kwashiorkor and marasmus. Patients cannot maintain their internal environment of water and electrolytes due to the changes in the body and the disruptions in renal function (Monckeberg, 1991, p.125; Jackson and Golden, 1991, p.139; Torún and Chew, 1994, p.956; Golden and Golden, 2000, p.520-521; Torún, 2006, p.886). There is no change in water clearance (Torún and Chew, 1994, p.956; Torún, 2006, p.886).

Torún and Chew (1994, p.958) stipulate that it is possible that a reduced renal blood flow and glomerular filtration rate are due to a decrease in plasma volume and low cardiac output, resulting from hypoalbuminemia (Torún, 2006, p.889). The ability to concentrate and dilute urine is also decreased (Monckeberg, 1991, p.125; Jackson and Golden, 1991, p.139; Torún and Chew, 1994, p.956; Golden and Golden, 2000, p.520-521; Torún, 2006, p.886), and the excretion of free hydrogen ions, titratable acid and ammonia is limited (Jackson and Golden, 1991, p.139; Golden and Golden, 2000, p.520-521).

The low renal blood flow and glomerular filtration rate lead to sodium retention and the production of renin and aldosterone and this would increase the reabsorption of sodium and water causing edema (Jackson and Golden, 1991, p.139; Torún and Chew, 1994, p.958). In kwashiorkor retained sodium and water are not evenly distributed in the extracellular compartments. Low intravascular volume causes the interstitial space to expand, resulting in oedematous patients presenting with dehydration. The patient is actually “hypovolaemic” and should not be treated with oral rehydration, as hypovolemia is a form of shock (Golden and Golden, 2000, p.519).

Other theories include the increase in ferritin that stimulates the production of antidiuretic hormone, energy deficiency that influences the function of the sodium pump, correction of intracellular potassium, and cell membranes that leak because of the damage caused by free radicals (Torún and Chew, 1994, p.958).

There is also a decrease in the capacity of the kidneys to acidify the urine. The capacity of the kidneys to absorp the sodium again is decreased because of endocrine failure (Monckeberg, 1991, p.125).

2.5.7 NEUROLOGICAL DEVELOPMENT AND BEHAVIOUR

The brain is the organ that grows most rapidly during the first months of life. Brain growth is slower, with some atrophy during malnutrition. Marasmic children have a smaller brain and malnourished children in general have a smaller head circumference than normal children of the same age. The smaller brain size leaves a space that is filled by cerebrospinal fluid (Monckeberg, 1991, p.126).

Besides the smaller brain there is also a decrease in nerve myelination, neurotransmitter production, and velocity of nervous conduction (Torún and Chew, 1994, p.957; Torún, 2006, p.887). Malnutrition during the first months of life is associated with histological, biochemical and bioelectrical disturbances with a lack of affective stimulation. There are also some neuronal structure changes. About 20% of infants with severe marasmus however, do not seem to have suffered any disturbances related to brain development (Monckeberg, 1991, p.126).

The first 2-3 years of life are crucial for both nutrition and child development. Rapid growth, including brain development, places high demands on nutrition (Black *et al*., 2008). Poor growth is associated with delayed mental development, and there is a relationship between impaired growth status, poor school performance and reduced intellectual achievement (De Onis *et al*., 2000). However, early brain development also requires environmental stimulation (Black *et al*., 2008). Inadequate cognitive or social stimulation during these early years has lifelong negative consequences on educational performance and psychological functioning (Monckeberg, 1991, p.126; Black *et al*., 2008).

The environment changes the long-term outcome of undernourished children. Severe malnutrition has a greater influence on the development of children living in poverty than those living in middle class homes, as those in middle class homes are usually stimulated more (Baker-Henningham and Grantham-McGregor, 2004, p.254).

Environmental and social support can improve the behaviour and cognitive state of malnourished children (Torún, 2006, p.887). Children in deprived living conditions develop

poorly and have deficiencies in intellectual, cognitive and social behaviour. When both food and environmental stimulation are given to children for one year during six months to three years of age, it can lead to improvements in the child's scholastic performance. The combined effect can cause children to catch up on mental development (Shetty, 2002, p.322).

Severely malnourished children in the hospital show behavioural changes in the acute stage of the disease (Baker-Henningham and Grantham-McGregor, 2004, p.253). They display a series of self-stimulating movements that they repeat constantly (Golden and Golden, 2000, p.519). They are apathetic, inactive, but irritable and cry when picked up (Torún and Chew, 1994, p.962; Golden and Golden, 2000, p.519; Shetty, 2002, p. 320; Baker-Henningham and Grantham-McGregor, 2004, p.253; Katz *et al*., 2005; Torún, 2006, p.892) and they have an expression of misery and sadness (Torún and Chew, 1994, p.962; Torún, 2006, p.892). The crying of the child during physical examination is monotonous, plaintive and without tears (Monckeberg, 1991, p.124).

The marasmic child sleeps for long periods (Katz *et al*., 2005), are catatonic and can develop bedsores (Golden and Golden, 2000, p.519), whereas the child with kwashiorkor shows subtle signs such as decreased voluntary movement, loss of interest in play, irritability, lethargy and apathy (Pereira, 1991, p.143; Katz *et al*., 2005). The children have a normal or impaired appetite and do not cry for food (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198).

Other signs include decreased stamina, steatosis, anemia and increased susceptibility to infection (Katz *et al*., 2005) and children are pale, with cold and cyanotic extremities (Torún and Chew, 1994, p.962; Torún, 2006, p.892). Rumination is sometimes seen in these children; the child regurgitates the last meal, then re-swallows it to give oral stimulation (Golden and Golden, 2000, p.519).

A developing child's behaviour and cognitive functions are influenced by the severity, timing and duration of nutritional deprivation, the quality of nutritional rehabilitation, emotional and psychosocial support, and the degree of care and stimulation provided by family members and caretakers (Torún, 2006, p.887). Even children with mild to moderate PEM have altered behaviour. They do not really explore their environments,

move less than better-nourished children and stay closer to the mother. When in hospital, they show less active distress with abnormal cries (Baker-Henningham and Grantham-McGregor, 2004, p.253).

Infants and toddlers have difficulty with development because they can't communicate their needs, or problems with handling, chewing and swallowing of food (Williams, 2005, p.402). It is impossible to separate nutrition from other factors that can affect gross and fine motor skills, intelligence and behaviour (Torún and Chew, 1994, p.957). Retarded psychomotor development is also characteristic of marasmus (Berdanier, 1995, p.154).

Children with severe malnutrition at age three had a 15-point deficit in intelligence quotient (IQ) at age 11 (Baker-Henningham and Grantham-McGregor, 2004, p.254; Torún, 2006, p.887). For the first three years of life there is a direct association between linear growth and change in development. When there is a change in height between six to 24 months there is a change in development. When there is a change in height within the first twelve months of life, there is a change in mental development in the second twelve months of life (Baker-Henningham and Grantham-McGregor, 2004, p.254). At two years of age, a height less than 2SD below the mean WHO reference (z-score of –2.0) is associated with an IQ deficit of 10 points (Williams, 2005, p.386).

Stunted children have poorer performance in cognitive functions and school achievement than non-stunted children up to twelve years. They have behavioural problems, low attentiveness and have more conduct disorders (Baker-Henningham and Grantham-McGregor, 2004, p.254; Torún, 2006, p.887).

Children of four to five years also play less, stay close to their mothers and are less responsive when given a task (Baker-Henningham and Grantham-McGregor, 2004, p.254). The loss of developmental potential in the first five years of life leads to late starting of school, poor educational attainment, early drop-out, and low earning potential (Black *et al*., 2008). School-aged children who return to environments with poor stimulation have deficits in cognitive development, school achievement and have poorer behaviour in school, such as attention and social skills problems. They are also more aggressive and distractible at home (Baker-Henningham and Grantham-McGregor, 2004, p.254).

Severely malnourished children have problems with creativity and social interaction (Torún and Chew, 1994, p.964; Torún, 2006, p.895). Loss of developmental potential can be far reaching. Globally, 219 million children do not reach their developmental potential, which places an enormous burden on the children, their families and their societies (Black *et al*., 2008) as growth retardation causes functional impairment in adult life, which can cause reduced work capacity and therefore affect economic productivity (De Onis *et al.,* 2000; Black *et al*., 2008).

Children's mental development can be improved by supplementing children's diet with extra nutrients. Extra food and mental stimulation can boost mental development. Food that stimulates longitudinal bone growth also stimulates brain development. Mental processing and nonverbal skills can be improved by parent stimulation (Shetty, 2002, p.322). All the behaviours return to normal with recovery, except that they still have poor levels of development (Baker-Henningham and Grantham-McGregor, 2004, p.254).

In Jamaica, nine to 24 month old stunted children received weekly supplements of milk and cognitive or social stimulation at home for two years and at the end of the study data showed that they had higher developmental scores than those who received neither or only one intervention. They need both nutrition and stimulation to catch up (Black *et al*., 2008).

2.5.8 ENDOCRINE SYSTEM

Kwashiorkor is characterized by enodocrine changes that develop due to a high carbohydrate and low protein intake (Bentley and Lawson, 1988, p.43; (Torún and Chew, 1994, p.953). When carbohydrates are ingested, insulin is released, which decreases epinephrine and cortisol (Hoffer, 2006, p.736; Torún and Chew, 1994, p.958). When insulin levels are low and cortisol increases (Torún and Chew, 1994, p.953-954; Golden and Golden, 2000, p.521; Torún, 2006, p.884-885) somatomedin secretion is reduced. Somatomedien activity is also reduced due to low plasma amino acid levels that stimulate human growth hormone secretion through feedback inhibition (Torún and Chew, 1994, p.953-954; Torún, 2006, p.884-885).

Golden and Golden (2000, p.521) report that growth hormone levels are elevated when insulin concentrations are low and show a reduced insulin response to a test meal; whereas Monckeberg (1991, p.125) report that plasma growth hormone levels in

marasmic children are low, but elevated in kwashiorkor. The levels in marasmic children return to normal with an improvement of their diet (Torún and Chew, 1994, p.953; Torún, 2006, p.884).

Stresses such as low food intake, fever, dehydration and infections stimulate epinephrine release and corticosteroid secretion is higher in marasmus because of the greater energy deficit. The increased levels of growth hormone and epinephrine lead to a reduction in urea synthesis (Torún and Chew, 1994, p.953-954; Torún, 2006, p.883-884).

Thyroxine levels are decreased because of a low iodine uptake by the thyroid. Energy is conserved by the reduction in thyroid hormone levels, decrease of thermogenesis and oxygen consumption (Torún and Chew, 1994, p.953-954; Wittenberg, 2004, p.202; Torún, 2006, p.885).

Gynecomastia or breast development in males is not uncommon (Golden and Golden, 2000, p. 520). The pathophysiology is possibly due to an imbalance of estrogens and androgens, with a decreased testosterone-to-estradiol ratio (Singer-Granick and Granick, 2009). Other common physiological causes (after the neo-natal period) are idiopathic (25%), medications (androgens, estrogens, digitoxin, cimetidine, spironolactone, ketoconazole and antiandrogens) (10 – 20%), cirrhosis or malnutrition (starvation) (8%) or primary hypogonadism (8%), hyperthyroidism, renal failure and liver disease (Kodner, 2000).

Glucose levels are often lower than normal, due to liver dysfunction. Fructose intolerance is similar to glucose intolerance and there is a marked reduction in gluconeogensis. Insulin-like growth factor 1&2, catecholamine and glucagon levels are often decreased (Golden and Golden, 2000, p.521).

Endocrine changes help to maintain energy homeostasis by increasing glycolysis, increasing amino acid mobilization and preserving visceral protein (Torún and Chew, 1994, p.953). Viceral protein is preserved due to increased breakdown of protein, decreased storage of glycogen, fats and protein and decreased energy metabolism (Torún and Chew, 1994, p.958). The amino acids are taken to the muscles at the expense of the liver (Hoffer, 2006, p.736).

Aldosterone levels are high in both marasmus and kwashiorkor and increase even more with the loss of oedema (Jackson and Golden, 1991, p.140). According to Torún and Chew (1994, p.954), the evolution of PEM into kwashiorkor or marasmus is related to differences in adrenocorticol responses. A better response will preserve visceral protein efficiently (Torún and Chew, 1994, p.954). The adaptive mechanisms causing growth retardation are often of endocrine origin. Adrenal response to adrenocortiocotropin stimulation is normal. The hypohysis-thyroid axis is depressed in marasmus (Monckeberg, 1991, p.125).

Monokines and cytokines are peptide or glycoprotein mediators in response to injury and are synthesized by monocytic and phagocytic cells in the liver and spleen. The most important monokines are interleukin-1 and cachectin or tumor necrosis factor. Macrophages of malnourished children with kwashiorkor have a decreased activity of interleukin-1. This lowers leukocyte counts in infections (Torún and Chew, 1994, p.956; Torún, 2006, p.887).

Serum levels of tumor necrosis factor are high in severe malnutrition, due to anorexia, muscle wasting and lipid abnormalities (Torún and Chew, 1994, p.956; Torún, 2006, p.887). Lipolysis decreases and insulin action is better because the free fatty acids that inhibit insulin action are suppressed (Torún and Chew, 1994, p.958).

Leptin is a sensitive marker of nutritional status. Serum leptin levels change with nutritional status and energy intake. It is an indicator of energy storage or chronic fasting. In PEM the suppressed production of leptin leads to an increased energy intake. The decrease of serum leptin levels might lead to a higher food intake through an increased appetite and stimulation of the secretion of cortisol and growth hormone. This causes an increase in energy expenditure. The loss of adipose tissue because of low food intake leads to a decrease in the secretion of leptin. Marasmic children have a higher loss of adipose tissue than those with kwashiorkor; therefore the serum leptin levels in these children are lower (Kilic *et al*., 2004). Kilic *et al.* (2004) found that the serum leptin levels in malnourished children where lower than in healthy children. Serum leptin levels in marasmic children were not significantly lower than in children with kwashiorkor (Kilic *et al*., 2004).

2.5.9 SKELETAL SYSTEM

An enlargement of the costochondrol junctions results in a “rickety rosary”. This is because of vitamin D metabolism abnormalities and phosphorus or calcium deficiency. Dislocation of junctions can be due to vitamin C or copper deficiency (Golden and Golden, 2000, p.520).

When radiological findings are used for diagnosis, it reveals Harris lines and delayed bone maturation. Harris lines appear on x-ray because biological processes are temporarily stopped. Cartilage is not mineralized and hypertrophic cartilage is no longer changed into calcified cartilage (Piercecchi-Marti *et al*., 2006).

2.5.10 HAIR

In kwashiorkor it is common to find atrophied hair roots. The hair is plucked easily and painlessly, and the patient can go bald. The hair becomes thin (sparse), straight and lifeless, without its normal sheen. Straight hair lifts up the curls and gives the appearance of trees with straight trunks. This is called the “canopy” or “forest sign” (Torún and Chew, 1994, p.961; Golden and Golden, 2000, p.519; Torún, 2006, p.892).

Texture, colour and strength of hair are also affected. Black, curly hair becomes silkier, lustreless and brown/reddish-brown and other hair colours can change to red, brown, grey or blond (Berdanier, 1995, p.153; Golden and Golden, 2000, p.519; Whitney *et al.,* 2001, p.83). The shortage of tyrosine needed to make melanin, causes the change in hair colour (Pereira, 1991, p.144; Torún and Chew, 1994, p.962; Torún, 2006, p.892), whereas in marasmus the hair appears to have a normal colour (Berdanier, 1995, p.154).

Hair is brittle, dry and without its normal sheen. Periods of poor and good nutrition produce alternating bands of depigmented and normal hair, which is called the “flag” or “band” sign” (Pereira, 1991, p.144; Torún and Chew, 1994, p.962; Torún, 2006, p.892).

2.5.11 SKIN

The skin is thin and smooth, with little elasticity and wrinkles easily when pinched (Monckeberg, 1991, p.121; Torún and Chew, 1994, p.961; Morgan and Weinsier, 1998, p.171; Torún, 2006, p.892) and lies in folds (Golden and Golden, 2000, p.519). Skin changes include dermal atrophy, ecchymosis, ulcerations and hyperkeratotic desquamation. The loss in skin fold thickness is associated with a loss of energy reserves

(Monckeberg, 1991, p.121; Torún and Chew, 1994, p.961; Morgan and Weinsier, 1998, p.171; Torún, 2006, p.892).

Crazy-pavement dermatosis is characterized by dark or reddish-purple patches in the folds of the body that peel off leaving oozing and raw surfaces that look like burn wounds (Pereira, 1991, p.144; Golden and Golden, 2000, p.519). The skin lesions are usually seen in the areas of the oedema such as the buttocks and back and perineum and thighs (Jackson and Golden, 1991, p.134; Torún and Chew, 1994, p.961; Torún, 2006, p.892) and depigmentation usually appears on the backs of legs, groins and elbows where there is friction (Berdanier, 1995, p.153; Golden and Golden, 2000, p.519; Whitney *et al.,* 2001; Katz *et al*., 2005). Exsudative lesions may appear in the openings of skin folds (Piercecchi-Marti *et al*., 2006).

None of the skin changes are only linked to PEM (Pereira, 1991, p.144; Monckeberg, 1991, p.121; Torún and Chew, 1994, p.961; Morgan and Weinsier, 1998, p.171; Torún, 2006, p.892), except for the flaky-paint dermatoses. Flaky-paint dermatosis develops rapidly and occurs a few days before death (Pereira, 1991, p.144). Skin lesions are different from pellagra and vitamin B_3 deficiency, because of the presence of oedema, fatty liver, discoloured hair and irritability (Jackson and Golden, 1991, p.134; Torún and Chew, 1994, p.961; (WHO, 2000; Torún, 2006, p.892). Pellagra presents as dermatitis very similar to that of PEM. Pellagra can be seen as areas of sunburn especially those parts of the body exposed to sunlight, dermatitis over pressure points, burning and itching of these areas, scaling and exfoliation and thickening of skin (WHO, 2000).

Other clinical signs include skin breakdown and delayed wound healing (Heimburger, 2006, p.833). Inadequate protein synthesis leaves the skin patchy, scaly and with sores that fail to heal (Shetty, 2002, p.320; Whitney and Rady, 2005, p.199). The dry, cracked layer peels off leaving hypopigmented, thin skin. The skin ulcerates easily, particularly in the flexures, perineum and behind the ears. The skin becomes darker, especially over pressure and bony areas, which is called pressure necrosis (Pereira, 1991, p.144; Golden and Golden, 2000, p.519).

2.6 PHYSIOLOGICAL AND METABOLIC CHANGES

In the weeks it takes PEM to develop, the body goes through metabolic and behavioral changes that lead to a decrease in nutrient demands and a nutritional equilibrium. After a

constantly low intake no adaptation occurs and the patient dies. Metabolic disruptions are caused by nutrient deficiencies, complications or inadequate treatment. Marasmic patients have a better adaptation because marasmus develops slowly (Torún, 2006, p.883), whereas kwashiorkor develops more rapidly (Torún and Chew, 1994, p.952).

2.6.1 ENERGY MOBILIZATION AND USAGE

Low energy availability may be due to factors such as malabsorption, high-energy losses in urine and high-energy expenditure because of infection, malignancy and fever. Uncomplicated PEM is characterized by weight loss and wasting, as a result of a negative energy balance. Energy intake isn't sufficient to cover the energy expenditure as a result of low intakes of energy dense foods that are worsened by infection-related anorexia. The extra energy needed is obtained from the energy in adipose and lean mass (Duggan and Golden, 2006, p.520). The resting metabolic rate of severely malnourished children is reduced to 85% of normal (Jackson and Golden, 1991, p.140).

An important adaptation is the slower activity of the sodium pump. This adaptation helps to understand the disruption in the sodium, potassium and water metabolism in kwashiorkor. This ion pump uses one third of the basal energy requirements. Allowing the intracellular sodium concentration to increase and potassium to decrease, the adaptation of the pump to 6 % of its activity can result in a great saving in energy (Golden and Golden, 2000, p.521).

A decrease in metabolic activity causes the child to show little or no reaction to temperature changes (Katz *et al*., 2005). When the growth rate, metabolism and physical activity decreases, hypothermia and a state similar to hibernation follows (Marcondes, 1991, p.74). Malnourished children cannot control body temperature and are sensitive to cold and heat. The normal sweating response is absent and this causes pyrexia to develop (Jackson and Golden, 1991, p.140). Hypoglycemia and hypothermia of 35.5 degrees Celsius (^{o}C) are present and are expecially evident after fasting (Torún and Chew, 1994, p.961 and 962; Torún, 2006, p.892).

Dietary protein is used more efficiently and this leads to lower energy requirements. Malnourished children get 4% of their total energy from protein and after recovery 7%. Amino acids released during tissue breakdown are used for protein synthesis and not oxidized. Proteins are continuously synthesized and broken down. The turnover of

proteins produce about one fourth of the basal energy expenditure (Jackson and Golden, 1991, p.140).

2.6.1.1 FAT

Fat (stored in adipocytes) is a good source of reliable energy during fasting and can be used by muscles such as the heart (Gallagher, 2008, p.67). When the body cannot compensate for insufficient energy intake the body mobilizes fat, which leads to a decrease in adipose cells and weight loss. When the energy deficit becomes severe, subcutaneous fat is reduced (Torún and Chew, 1994, p.952; Torún, 2006, p.883). Preservation of adipose tissue occurs in kwashiorkor, but not necessarily in marasmic kwashiorkor, where it can be depleted (Jackson and Golden, 1991, p.138). When there is little or no insulation (fat) under the skin, the child is not protected against cold (Monckeberg, 1991, p. 122-123; Torún and Chew, 1994, p.952 & 961; Torún, 2006, p.883 & 892; Sizer and Whitney, 2000, p.195; Whitney *et al*., 2001, p. 83; Whitney and Rady, 2005, p.198).

For the body to release fatty acids, it is important to have low insulin levels and high anti-insulin hormones such as glucagons, cortisone, epinephrine and growth hormone. These hormones activate the hormone-sensitive lipase enzyme on the adipocyte membrane. These enzymes open up the stored triglyserides and release fatty acids and glycerol from fat cells. Fatty acids then travel to the liver and easily enter the liver cells. Inside the cell the fatty acids enter the mitochondria via the carnitine acyltransferase transport system. This system carries fatty acid carnitine esters across the mitochondrial membrane (Gallagher, 2008, p.67).

The process of B-oxidation is used to form acytel co-enzyme A from fatty acid co-enzyme A. An excess of acytel co-enzyme A molecules are produced during starvation, therefore the liver can get energy from B-oxidation. This process causes ketones to form, which enter the bloodstream and act as a source of energy for the muscles. The brain and nervous system use ketones for energy and ketone production increases during fasting. Muscle catabolism and gluconeogenesis are decreased because the brain is not using glucose. Low muscle catabolism leads to lower amounts of ammonia received by the liver (Gallagher, 2008, p.67).

2.6.1.2 GLUCOSE

Glucose is the most important source of food for the brain and nervous system, red blood cells and white blood cells. Epinephrine, thyroxine and glucagon ensure the substrates needed for gluconeogenesis, are available. The most used substrate is alanine and when its nitrogen is removed, it becomes pyruvate (Gallagher, 2008, p.67).

Muscle and the brain release pyruvate and lactate for gluconeogenesis through the Cori cycle and muscles release glutamine and alanine. Amino acids are deaminated into ∝-ketoglutarate and transaminated into pyruvate, then into oxaloacetate and then to glucose. Muscle-derived glutamine is used to supply the kidneys with ammonia and ∝-ketoglutarate is used to produce glucose. During starvation glucose production by the kidney increases while production by the liver decreases (Gallagher, p.67, 2008).

Glucose is derived from glycogon (during fasting) by the action of glycogen and epinephrine, but these are depleted within 24 hours. After 24 hours glucose is synthesized using mostly protein. Glucogen is also depleted because some is needed for glycogen resynthesis. After polonged fasting the production of glucose is decreased from 90% to less than 50% through liver gluconeogenesis (Gallagher, 2008, p.67). Liver glycogen reserves are low in marasmus and there is a high risk of fatal hypoglycemia (James *et al.,* 1999).

2.6.1.3 PROTEIN

The loss of body protein due to protein deficiency is primarily skeletal muscle. Visceral protein is only lost early in the development of PEM but stabilizes when nonessential tissue protein is depleted. Lean body mass diminishes slowly because of muscle protein catabolism. With marasmus, the alterations in body composition lead to increased basal oxygen consumption. With kwashiorkor, the dietary protein deficiency leads to depletion of amino acids, which affects cell function and reduces oxygen consumption and then the basal energy expenditure decreases (Torún and Chew, 1994, p.953; Torún, 2006, p.884).

Protein synthesis is disrupted in malnourished patients because of the poor availability of protein. Hepatic export protein is not made in sufficient quantities to maintain circulating concentrations (Golden and Golden, 2000, p.522). Of the free amino acids entering the body via dietary and tissue proteins, 75 % are recycled or reused for protein synthesis, and only 25 % are broken down for other metabolic purposes (Torún and Chew, 1994, p.

953; Torún, 2006, p.884). With 50% of the body's protein stores exhausted, it is difficult to recover from infections. When the respiratory muscles cannot support breathing it can lead to death (Gallagher, 2008, p.67).

Amino acid catabolism reduces urea synthesis and urinary nitrogen synthesis (Torún and Chew, 1994, p. 953; Torún, 2006, p.884). Urea synthesis rate and excretion is also reduced during long periods of fasting. Urea is excreted at the same rate as the kidney produces uric acid. Protein losses are at a minimum and lean body mass is spared (Gallagher, 2008, p.67).

The half-lives of several proteins increase during malnutrition. Albumin synthesis decreases in the beginning, but after a few days the rate of breakdown falls and the half-life increases. Albumin is shifted from the extravascular to the intravascular pool. Thus, even with reduced synthesis, this will help maintain available levels (Torún and Chew, 1994, p. 953; Torún, 2006, p.884). Children with marasmus have a poor arginine stimulation response (Monckeberg, 1991, p.125).

Carbohydrates and increased insulin prevent fat stores from being used for fuel (glucose) to support muscles and the brain, and inhibit fat from being formed into ketones. Insulin secretion limits muscle breakdown and protein cannot be used to make albumin and other visceral protein, whereas protein can be used once fat stores are exhausted, but this may lead to death (Gallagher, 2008, p.67).

2.6.2 MICRONUTRIENTS

More than 2 billion people worldwide suffer from deficiencies in micronutrients (Mother and Child Nutrition, 2007). Illnesses and reduced nutrient intake causing marasmus and kwashiorkor are responsible for some of the major deficiencies of vitamins and minerals, such as iron, iodine, vitamin A and zinc. A reduced intake and abnormal losses of micronutrients through external secretions (zinc in diarrhea fluid / burn exudates) are also common in PEM (Morgan and Weisnier, 1998, p.171; Müller and Krawinkel, 2005).

In India, 50% of all healthy looking children have biochemical deficiencies of vitamin A, B_2, B_6, folic acid and vitamin C and two thirds of children have clinical evidences of iron deficiency and deficiency of iodine and zinc is also common (Singh, 2004).

In South Africa the intake of energy, calcium, iron, zinc, selenium, vitamin A, D, E, riboflavin, niacin, B_6 and folic acid were below two thirds of the recommended daily allowance (NFCS, 1999; Labadarios *et al.,* 2005b). The lowest intake of iron was in the Free State and Northern Cape, where 25-37% of children took in less than 50% of the recommended daily alowance. Zinc intake was very low with 32-53% of children taking in less than 50% of the recommended daily allowance (Labadarios *et al.,* 2005b).

Micronutrient deficiencies are still seen as a major public health problem, even though vitamin C, D and B deficiencies have declined. A lack of one micronutrient is associated with deficiencies of another (Müller and Krawinkel, 2005). Some of the features associated with trace mineral deficiencies are discussed in Table 2.13.

Table 2.13 Features associated with trace mineral deficiencies (Jackson and Golden, 1991, p.137)

Trace mineral	**Associated feature**
Zinc	Ulcerated skin Delayed cutaneous hypersensitivity response Atrophy of the thymus
Copper	Reduced copper-zinc superoxide dismutase activity (12% of the patients on admission)
Selenium	Low glutathione peroxidase activity in 45% of the patients on admission and very low in 5 of 6 children who died
Iron	Relatively high plasma ferritin in all severely malnourished children with no particular relationship to edema. Death associated with ferritin levels > 250μg/dL.

Clinical features of PEM and micronutrient deficiencies can overlap (Müller and Krawinkel, 2005) (Table 2.14). Some of the signs of micronutrient deficiencies are pallor (iron), dermatoses, cheilosis (B vitamins), xerophthalmia (vitamin A), acrodermitis (zinc) and goiter (iodine) (Williams, 2005, p.402).

Table 2.14 Causes, manifestations, management and prevention of the major micronutrient deficiencies (Müller and Krawinkel, 2005)

Nutrient	Essential for the production or function	Causes of deficiency	Manifestations of isolated deficiency	Management and prevention
Iron	Hemoglobin Various enzymes Myoglobin	Poor diet Elevated needs (e.g. while pregnant, in early childhood) Chronic loss from parasite infections (e.g. hookworms, schistosomiasis, whipworm)	Anemia and fatigue Impaired cognitive development Reduced growth and physical strength	Foods richer in iron and with fewer absorption inhibitors Iron-fortified weaning foods Low-dose supplements in childhood and pregnancy Cooking in iron pots
Iodine	Thyroid hormone	Except where seafood or salt fortified with iodine is readily available, most diets, worldwide, are deficient	Goitre, hypothyroidism, constipation Growth retardation Endemic cretinism	Iodine supplement Fortified salt Seafood
Vitamin A	Eyes Immune system	Diets poor in vegetables and animal products	Night blindness, xerophthalmia Immune deficiency Increased childhood illness, early death Contributes to development of anemia	More dark green leafy vegetables, animal products Fortification of oils and fats Regular supplementation
Zinc	Many enzymes Immune system	Diets poor in animal products Diets based on refined cereals (e.g. white bread, pasta, polished rice)	Immune deficiency Acrodermatitis Increased childhood illness, early death Complications in pregnancy, childbirth	Zinc treatment for diarrhea and severe malnutrition Improved diet

2.6.2.1 MINERALS

2.6.2.1.1 IRON

The WHO definition for iron deficiency is a serum value of less than 110 g/L. Causes of iron deficiency are low birth weight, early introduction of whole cow's milk, vegetarian weaning, high tea intake, South Asian ethnic background, and low socioeconomic status (Williams, 2005, p.408). It is common in the first year of life, where the main food is milk, which is low in iron, but iron deficiency anemia can develop at all ages (Wittenberg, 2004, p.210).

Iron is an essential part of haemoglobin, myoglobin and various enzymes. Iron deficiency is the main cause of microcytic, hypochromic anaemia, but can also lead to other adverse effects (Müller and Krawinkel, 2005). Thirty-seven percent of the world's populations suffer from anaemia (Mother and Child Nutrition, 2007). Anaemia and iodine deficiencies threaten the world's children and about 20-50% of children are physically stunted because of these deficiencies (Shetty, 2002, p.322). In the world about 25% of pre-school children are deficient in iron and the proportion of children in Africa with anaemia is 68% (UNICEF, 2009c, p.23). In South Africa, about one in ten children have an iron deficiency (Wittenberg, 2004, p.210).

Anaemic children, as with underweight children, are often unresponsive to stimuli, solemn, do not get involved in activities, are unhappy, stay close to their mothers, do not

show pleasure, are wary and tire easily in a free-play situation. Even after treatment, during infancy, the deficits may remain. Anaemic children show increased social and attention problems. The duration of the anaemia episode is linked to the level of development (Baker-Henningham and Grantham-McGregor, 2004, p.256-257).

Marasmic infants do not seem to suffer from iron deficiency but it does become apparent when growth resumes or during recovery. Once recovery has started it is necessary to provide an additional 1-2mg/kg/day of iron since both the iron content and bioavailability of iron in cow's milk are low (Monckeberg, 1991, p.128).

Some bacteria use iron for growth and therefore iron supplementation can sometimes worsen infections. A deficiency negatively affects T-lymphocyte numbers and function and bactericidal activity of neutrophils, but can be restored by iron supplementation (Strobel and Ferguson, 2006, p.489). Vitamin C can be given with iron as it helps with the absorption of iron (Williams, 2005, p.408).

2.6.2.1.2 ZINC

The use of micronutrients such as iron, copper and zinc in the recovery of malnourished infants is emphasized. Zinc is important and though levels are normal, supplemention can increase the rate of recovery and growth and decrease infections during refeeding (Monckeberg, 1991, p.128).

Globally, severe, prolonged episodes of diarrhoea are often the cause of zinc deficiency. Signs of deficiency include failure to thrive and a classical skin rash associated with zinc deficiency (Williams, 2005, p.410). The differences between kwashiorkor, marasmus and zinc deficiency are shown in Table 2.15. A low weight-for-age is associated with micronutrient deficiencies and zinc deficiency contributes to growth retardation in young children (Caulfield *et al*., 2004).

Human milk contains high levels of bioavailable zinc, but the content decreases over the first six months of lactation. Late (after three to four months of age) zinc deficiency can occur if extremely low birth weight (<1000g) preterm infants are fed human milk. Rapid somatic growth and the long duration of breastfeeding play a role in the development of this deficiency (Williams, 2005, p.410). A zinc deficiency can interfere with a variety of biological functions, such as gene expression, protein synthesis, skeletal growth, gonad

development, appetite and immunity. Zinc is important for cells such as neutrophils and natural killer cells and for balancing T helper cell functions. It can be linked to diarrhoea and pneumonia, but little evidence is available on its role in malaria and growth retardation (Müller and Krawinkel, 2005).

Zinc is an important part of a number of enzymes. The immune system depends on zinc-dependant proteins, which are involved in cellular functions such as replication, transcription and signal transduction in ribonucleic acid and deoxyribonucleic synthesis (Müller and Krawinkel, 2005).

Zinc deficiency (Acrodermatitis enteropathica) is a rare disorder of GI zinc absorption. There are similarities between kwashiorkor and zinc deficiency syndrome such as anorexia, diarrhoea, and flaking of skin and defective immunocompetence. Zinc supplementation is not effective in decreasing oedema but is beneficial for the healing of skin lesions (Jackson and Golden, 1991, p.136; Wittenberg, 2004, p.211).

Hipozincemia is often present in infants with kwashiorkor (Katz *et al*., 2005). Oral zinc supplementation can cause the atrophied thymus gland to grow again (Jackson and Golden, 1991, p.136). Iron, copper and zinc are all absorbed in the GI and therefore a multinutrient supplement is better than a single nutrient supplement (Williams, 2005, p.410). In populations with a high prevalence of zinc deficiency, zinc supplementation has reduced infections and morbidity in children who were undernourished as well as well nourished (Caulfield *et al*., 2004).

Table 2.15 Comparison of the clinical and biological signs of pure protein malnutrition, energy malnutrition and zinc deficiency (Vis, 1991, p.150)

	Pure protein malnutrition (kwashiorkor)	Energy malnutrition (marasmus)	Zinc deficiency
Growth retardation	±	++	++
Loss of weight-for-height	- or +	+++	-
Subcutaneous fat	++	-	-
Edema	+++	- or +	-
Hypogonadism	±	±	++
Hepatomegaly	++	±	+ or ++
Skin and hair lesions	+++	-	++
Anemia	++	+	++
Plasma proteins and albumin	Lowered	Normal	Normal
Alkaline phosphatase	Lowered	Normal	Severely depressed

2.6.2.1.3 IODINE

In the world about 35% of the population are at risk of developing iodine deficiency (Mother and Child Nutrition, 2007). Iodine deficiency is prevalent in both developed and developing countries, where 42% of Africa's population is iodine deficient (UNICEF, 2009c, p.23). An iodine deficiency reduces the production of thyroid hormone and increases the production of thyroid-stimulating hormone. The thyroid gland becomes hyperplastic and hypothyroidism develops (Wittenberg, 2004, p.211; Müller and Krawinkel, 2005).

Iodine deficiency after birth can lead to permanent impairment of mental development. Poor brain development due to iodine deficiency can lead to cretinism (Shetty, 2002, p.322). Signs of cretinism include delayed bone age, growth impairment, neurological problems, deafness, cerebral palsy and learning difficulty. The variety of problems associated with cognitive impairment and cretinism can be mild or serious (Williams, 2005, p.410).

Iodine deficiency can be treated with iodised salt (Wittenberg, 2004, p.211). It is important to fortify food and supplement pregnant women as brain development starts before birth (Williams, 2005, p.411).

2.6.2.1.4 OTHER MINERALS

The concentrations of zinc and copper in muscle and liver are reduced in proportion to soluble protein (Jackson and Golden, 1991, p.136). Copper deficiency is mainly found in extremely low birth weight babies. A deficiency can lead to anaemia and osteopenia, which can cause fractures (Williams, 2005, p.411). Plasma copper concentrations do not give an indication of copper status. Most circulating copper is found in ceruloplasmin (copper-carrying protein) (Jackson and Golden, 1991, p.136). If there is an absence of ceruloplasmin in the blood, it leads to severe copper deficiency and children can suffer from impaired T-cell immunity, high bacterial infections and diarrhoea (Strobel and Ferguson, 2006, p.489). Copper supplementation is required to satisfy the need during recovery (Jackson and Golden, 1991, p.136). Too much copper can also cause impaired immune functions (Strobel and Ferguson, 2006, p.489).

Leukopenia with neutropenia and alterations of bone structure may also be present. These signs disappear when copper is added to the diet. Low levels of plasma copper and ceruloplasmin are found. Decreased activity of superoxide dismutase (enzyme dependant on copper) is present. These disturbances become visible during the period of rapid growth in the early phases of rehabilitation. A copper deficiency is found in infants who have never been breastfed and suffered from acute diarrhoea. A copper supplement of 80 microgram (µg) per kilogram (kg) per day is usually adequate (Monckeberg, 1991, p.128).

Biopsies and post-mortem material show depletion of muscle magnesium in malnourished children. Magnesium retention during recovery is higher than expected (Jackson and Golden, 1991, p.136). Calcium and phosphorus are important in maintaining mineral homeostasis. Phosphorus depletion is the cause of the reduced renal acidifying capacity (Jackson and Golden, 1991, p.136).

Selenium is an essential trace element. It is mainly concentrated in tissues involved in the immune response, such as lymph nodes, liver and spleen. Numerous immune functions can be affected by a selenium deficiency (Strobel and Ferguson, 2006, p.489). In kwashiorkor the plasma and erythrocyte selenium concentrations are reduced and this shows a true selenium deficiency (Jackson and Golden, 1991, p.137).

2.6.2.2 VITAMINS

Vitamin deficiencies exist in the presence of kwashiorkor, but are not a primary cause of disease. Deficiencies of the B vitamins, vitamins C, D, E and K and folic acid have been reported. The frequency of the deficiencies varies with diet, geographical location and season (Jackson and Golden, 1991, p.138).

2.6.2.2.1 FAT SOLUBLE VITAMINS

2.6.2.2.1.1 VITAMIN A

Vitamin A deficiency is still the second most serious micronutrient deficiency worldwide (Strobel and Ferguson, 2006, p.488), while iron deficiency is still the most common and widespread nutritional disorder in the world with about 2 billion people (30% of the world population) being anaemic (WHO, 2010). The SAVACG Survey (1995) found that one in three (33%) children had a marginal vitamin A deficiency and these children were at a higher risk of being anaemic and having iron deficiency anaemia. In 2007 the prevalence of vitamin A deficiency was only about 25% of pre-school age children in South Africa (Mother and Child Nutrition, 2007). In Africa, South East Asia and South Asia vitamin A deficiency is considered a public health problem if the prevalence of night blindness exceeds 1% or >0.05% (Williams, 2005, p.410).

The highest prevalence of vitamin A deficiency is in Africa and Asia (40%), with 33% (190 million) of pre-school children being vitamin A deficit. Deficiencies of other micronutrients also occur, with the highest proportion of pre-school children with anaemia living in Africa (68%)(UNICEF, 2009c, p.23).

Vitamin A deficiency is commonly seen in PEM (Bentley and Lawson, 1988, p.44). If one child in a family is identified as being deficient, the other siblings and the mother should also be treated. Factors causing vitamin A deficiency are low intake of fat and fat-soluble vitamins, ceasing breastfeeding, poverty and increased losses through acute infections, such as measles and diarrhoea (Williams, 2005, p.410). Vitamin A is known as the anti-infection vitamin (Turnham, 2005, p.258). Vitamin A supplementation can reduce mortality in populations regardless of anthropometric status (Caulfield *et al*., 2004).

Iron deficiency anaemia is characterized by reduced serum iron levels, increased serum iron binding capacity and a reduced serum ferritin level, whereas anaemia caused by vitamin A deficiency resembles hypochromic anaemia and the ferrin levels are normal.

Once the vitamin A intake of children improves, there is an increase in serum iron and total iron binding capacity and also an increase in haemoglobin. Studies show that vitamin A deficiency directly impairs synthesis of transferrin (Bloem, 1995).

There is an association between diarrhoea, mortality and Vitamin A deficiency (Müller and Krawinkel, 2005). Vitamin A supplementation reduces the severity of severe diarrhea and is used for the treatment of measles (Turnham, 2005, p.258).

Vitamin A is essential for the functioning of the eyes and the immune system (Müller and Krawinkel, 2005). Low plasma retinol levels are seen in infections and this is caused by a reduced hepatic synthesis of retinol binding protein. This fall in plasma retinol during infection is caused by increased capillary permeability, which can help with quicker distribution of retinol to tissues that must fight off the infection (Turnham, 2005, p.258).

The association between evidence for vitamin A deficiency and lower respiratory tract infections and malaria is weak (Müller and Krawinkel, 2005).

2.6.2.2.1.2 VITAMIN D

Factors causing vitamin D deficiency include the season, prolonged exclusive breastfeeding (for longer than six months) and cultural factors (clothing that covers the body). Clinical presentations include hypocalcaemia tetany in young infants and nutritional rickets. Rickets is also present and coexists with iron deficiency anaemia and growth faltering. Clinical signs are swelling of epiphyses, beading of the ribs (the “rickety rosary”), bossing of the frontal bones and softening of the cranium (“crani-otabes”), as well as a delay in the closure of the fontanels and the appearance of teeth (Williams, 2005, p.409).

2.6.2.2.1.3 VITAMIN E

Vitamin E deficiency leads to a reduced production of antibodies and T-cell proliferation. Immune cell function is affected by deficiencies and low dietary levels. Vitamin E protects cell membrane integrity from lipid peroxidation caused by free oxygen radicals (Strobel and Ferguson, 2006, p.489).

2.6.2.2.2 WATER SOLUBLE VITAMINS

2.6.2.2.2.1 B VITAMINS

A thiamin deficiency can cause beri-beri, cardiac failure, periphera neuropathy, and encephalopathy. Nicotin acid deficiency causes pellagra, diarrhoea and photo-dermatosis. A riboflavin deficiency is associated with cheilosis and anemia. The existence of these deficiencies in malnourished children shows the importance of micronutrient supplementation (Williams, 2005, p.411).

Vitamin B_{12} deficiency is less common and mostly present in infants who are breastfed by vegan mothers and vegan children (Williams, 2005, p.411). Folic acid deficiency causes megaloblastic anemia. Dietary deficiency is less common in children and it is usually seen in the presence of malabsorption in cases of celiac disease (Williams, 2005, p.411).

2.6.2.2.2.2 VITAMIN C

Vitamin C is a water-soluble antioxidant and has an influence on most aspects of the immune system. High concentrations are found in white blood cells. A reduced immune function is linked to low plasma levels (Strobel and Ferguson, 2006, p.488).

Vitamin C deficiency can result in scurvy and is rare in well-nourished children. Vitamin C deficiency presents with bruising, bleeding and bone tenderness, which can cause pseudoparesis. X-rays show subperiosteal hemorrhage with calcification and loss of trabecular bone mineral (Williams, 2005, p.411).

2.6.3 OTHER PHYSIOLOGICAL AND METABOLIC CHANGES

In the body there are major changes in the physiological and metabolic responses because of a decrease in cell activity. The malnourished patient becomes poikilothermic. A modest reduction to 21^{o}C or an elevation to 33^{o}C in environmental temperature can lead to hypothermia or pyrexia. Malnourished patients reduce their oxygen consumption in a cool environment and seldom shiver (Golden and Golden, 2000, p.521).

Not all pathophysiologic changes lead to advantageous adjustments. Certain functions are affected, and some nutrient reserves decreased (Torún and Chew, 1994, p.955; Torún, 206, p.886). Dietary toxins (aflotoxin) can lead to the abnormalities seen in PEM. It is possible that chronic aflatoxin poisoning could cause kwashiorkor because of

infection and diarrhoea (Bentley and Lawson, 1988, p.43; Wittenberg, 2004, p.199). Environmental toxins accumulate in the liver of patients with kwashiorkor, but as the liver in patients with kwashiorkor is fatty, it cannot clear the environmental toxins effectively (Wittenberg, 2004, p.199).

2.7 PROGNOSIS AND RISK OF MORTALITY

The severity of malnutrition depends on the timing and duration of the nutritional stress. Malnutrition increases a child's susceptibility to illnesses, such as infections, which doesn't necessarily lead to death, but it can contribute to mortality due to the other illnesses (Duggan and Golden, 2006, p.523). The life-threatening complications that accompany severe malnutrition include jaundice, severe anaemia, respiratory distress, neurological and consciousness alterations and hypothermia (Torún and Chew, 1994, p.964; Torún, 2006, p.896).

Child mortality rather than infant mortality can give a better idea of the association between malnutrition and death. The nutritional status of the child affects the risk of death due to diarrhoea, respiratory infection and malaria (Duggan and Golden, 2006, p.523). Marasmus is associated with a lower mortality than kwashiorkor (Shetty, 2002, p.320). A high case fatality (from 20% for all types of severe PEM to >50% in kwashiorkor) is seen with severe malnutrition and oedema, which includes infection and metabolic complications. The difference between the long-term effects of severe malnutrition and persistent socio-economic deprivation are difficult to separate (Duggan and Golden, 2006, p.523). There is no clear evidence to show that the damage done by malnutrition and poor living environment cannot be corrected in a good, stimulating environment (Torún and Chew, 1994, p.964; Torún, 2006, p.896).

Mortality rates are also associated with the quality of treatment. With adequate treatment a mortality rate of 5% or less can be achieved. Severe anthropometric deficiencies are associated with a higher mortality rate. Mortality rates can be as high as 40% but with adequate treatment it can be reduced to less than 10% (Torún and Chew, 1994, p.964; Torún, 2006, p.896). In South Africa, malnutrition is one of the five causes of 75,000 child deaths per year, of which 40,200 babies and children can be saved through interventions (promotion of healthy diet, support for exclusive breastfeeding or other feeding options, vitamin A supplementation and prevention and treatment of children with HIV and AIDS) (Every Death Counts, 2008).

A survivor of early malnutrition may recover completely, be stunted or have a delayed adolescent growth spurt. Linear growth can only be caught up once the weight has recovered with nutritional rehabilitation (Duggan and Golden, 2006, p.521-3). Wasting and kwashiorkor are part of the six prognostic indicators of deaths that occur within 48 hours after admission. The other five indicators are part of the remaining list in Table 2.15 (Torún and Chew, 1994, p.964; Torún, 2006, p.896).

The goal with the treatment of mild and moderate PEM is to correct the acute signs (Torún and Chew, 1994, p.964; Torún, 2006, p.895). During rehabilitation the catch-up growth in height may take long (Marcondes, 1991, p.74; Torún and Chew, 1994, p.964; Torún, 2006, p.895). The child will remain stunted, and if the child is small it may influence his/her maximal working capacity as an adult (Torún and Chew, 1994, p.964; Torún, 2006, p.895).

Catch-up is influenced by the time of occurrence, severity and duration of malnutrition. In both children with kwashiorkor and marasmus the neuropsychomotor performance is still poor after four to six months of rehabilitation even though nutritional recovery is good (Marcondes, 1991, p.74).

Table 2.16 Characteristics that indicate poor prognosis in patients with protein-energy malnutrition (Torún, 2006, p.896)

- Age < 6 months
- Deficit in weight for height > 30%, or in weight for age > 40%
- Signs of circulatory collapse: cold hands and feet, weak radial pulse, diminished consciousness
- Stupor, coma, or other alterations in awareness
- Infections, particularly bronchopneumonia or measles
- Petechiae or haemorrhage tendencies (purpura usually associated with septicaemia or a viral infection)
- Dehydration and electrolyte disturbances, particularly hypokalemia and severe acidosis
- Persistent tachycardia, signs of heart failure or respiratory difficulty
- Total serum proteins < 30 g/L
- Severe anaemia (< 50 g haemoglobin/L) with clinical signs of hypoxia
- Clinical jaundice or elevated serum bilirubin
- Extensive exudative or exfoliative cutaneous lesions or deep decubitus ulcerations
- Hypoglycaemia
- Hypothermia

2.8 TREATMENT AND MANAGEMENT OF SEVERE MALNUTRITION

In the 1950's nutrition rehabilitation centres were regarded as the best option for the management of malnutrition. Centres were established in developing countries such as Asia and Latin America as these were cheaper and more effective to run than hospitals.

In Indonesia and Peru studies found that the treatment of children on an outpatient basis led to a reduction in the case fatality rate to 16.6% and 2% respectively. A study in Bangladesh showed that home-based care was five times cheaper than caring for children in hospital (Orach and Kolsteren, 2002).

Malnutrition can be managed on five levels, namely in hospitals, in nutrition rehabilitation centres, in health centres, in the community and at home with regular follow up (Orach and Kolsteren, 2002). Rehabilitation programmes should promote shorter hospital stay and the home or community based treatment, especially in areas were resources such as supplies and personnel are limited (Fuchs *et al.,* 2004).

The goals for the treatment of severe malnutrition can only be reached if the WHO treatment guidelines for PEM are followed. The guidelines can be simplified in such a way that the efficacy and effectiveness of the guidelines is enhanced allowing the guidelines to be used in resource-poor settings or communities. The basic principles of the WHO guidelines should, however not be compromised. Complementary guidelines for nutritional management of severely malnourished children need to be developed for use at community level and in emergency or crisis situations (Fuchs *et al*., 2004). If resources (e.g. staff) are available the WHO guidelines can result in lower mortality rates (Collins *et al*., 2006).

The guidelines are mainly for severe cases of malnutrition even though some children do not fall within the ranges as stipulated (<-3Z weight-for-length or/and oedema). When a low height-for-age alone is used, it can result in only stunted children being admitted. If less severe cases of malnutrition are admitted to malnutrition wards, it causes an unnecessary high risk of cross-infection and increases workload. Underweight children are sometimes referred to a hospital due to other diseases or causes and can benefit from a few days on catch-up formula or enriched family foods and the carers can benefit from the nutrition education. The benefits should be weighed against the risk of hospital-acquired infections (Ashworth *et al*., 2004).

Previously when the WHO guidelines were not followed in South African hospitals, children often waited too long to be admitted. Children with oedema were wrongly given diuretics; children with diarrhoea were given intravenous fluids randomly, increasing their risk of heart failure; antibiotics were not given routinely; and electrolyte and micronutrient

deficiencies were not corrected. Special feeds were not prepared and malnourished children received the same general diet as the adults, but only smaller portions. Children sometimes went more than eleven hours without food at night, which increased the risk of death due to hypoglycaemia (Ashworth *et al*., 2004).

When the WHO guidelines were not implemented, play and stimulation were not provided as part of the treatment regime and there was no link between treatment in hospital and at home. Hospitals in the Eastern Cape, South Africa, lacked basic resources such as nasogastric tubes, vitamin A capsules, multivitamins and scales. Wards were cold because of irregular electricity supply. The hospital staff was demotivated and unrewarding. Mothers also did not have the option of staying with their children throughout the night. Hygiene practices were poor and major changes were needed before the implementation of the WHO guidelines could be successful (Ashworth *et al*., 2004).

When the WHO guidelines were first implemented, it was found to be feasible to implement the guidelines in first-referral under-resourced hospitals and even though the implementation was not perfect, case-fatality rates improved (Ashworth *et al.,* 2004). In Sub-Saharan Africa and India, an additional burden is HIV infection (Müller and Krawinkel, 2005). Even with the availability of the WHO guidelines mortality rates are still high (Fuchs *et al.,* 2004).

Table 2.17 shows a summary of all the elements included in the WHO 10 steps. The implementation steps and duration of each of the steps over the period of initial treatment, rehabilitation and discharge and follow-up are given in Table 2.18 and Appendix E.

Table 2.17 Steps in the management of severe protein-energy-malnutrition (Müller and Krawinkel, 2005)

Problem	Management
Hypothermia	Warm patient up; maintain and monitor body temperature
Hypoglycaemia	Monitor blood glucose; provide oral (or intravenous) glucose
Dehydration	Rehydrate carefully with oral solution containing less sodium and more potassium than standard mix
Micronutrients	Provide copper, zinc, iron, folate, multivitamins
Infections	Administer antibiotic and antimalarial therapy, even in the absence of typical symptoms
Electrolytes	Supply plenty of potassium and magnesium
Starter nutrition	Keep protein and volume load low
Tissue-building nutrition	Furnish a rich diet dense in energy, protein and all essential nutrients that is easy to swallow and digest
Stimulation	Prevent permanent psychosocial effects of starvation with psychomotor stimulation
Prevention of relapse	Start early to identify causes of PEM in each case; involve the family and the community in prevention

For treatment to be successful, both medical problems and problems related to societies should be evaluated and corrected. The management of severe malnutrition is divided into three phases, which are the initial treatment phase where life-threatening conditions are identified and treated, deficiencies are identified and feeding is begun; the rehabilitation phase where feeding is given to improve weight, stimulation is given, parents are educated and preparations for discharge are begun; and the follow-up phase where the child and family are follow-up after discharge to prevent relapse (WHO, 1999).

Table 2.18 Implementation steps (phases) for treatment of severely malnourished child (WHO, 1999; NDoH, 2003; Jackson *et al*., 2006)

ACTIVITY	INITIAL TREATMENT: (week 1)		REHABILITATION:	FOLLOW-UP:
	days 1 – 2	days 3 – 7	weeks 2 – 6	weeks 7 – 26
Treat or prevent:				
Hypoglycaemia	→→→→→→→→			
Hypothermia	→→→→→→→→			
Dehydration	→→→→→→→→			
Correct electrolyte imbalance	→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→			
Treat infection	→→→→→→→→→→→→→→→			
Correct micronutrient deficiencies	→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→			
	← without iron →←		with iron →	
Begin feeding	→→→→→→→→→→→→→→→			
Increase feeding to recover lost weight catch-up growth”	→→→→→→→→→→→→→→→→→→			
Stimulate emotional and sensorial development	→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→→			
Prepare for discharge			→→→→→→→→→→→→→→	

2.8.1 ASSESSMENT FOR TREATMENT

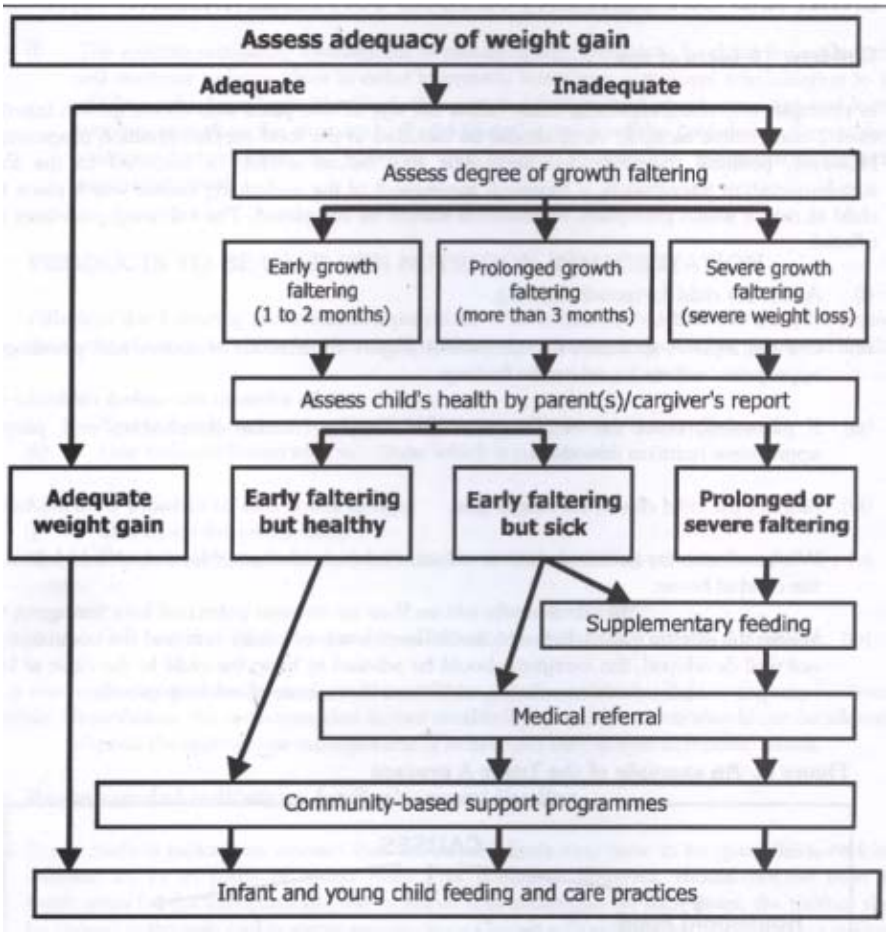
When a child presents with growth faltering over two consecutive months, all possible causes should be investigated before a plan of action can be decided on (NDoH, 2003).

Before treatment can start a child should be evaluated and assessed to decide on the best treatment strategy for the particular child (WHO, 1999). Principles for the treatment of severely malnourished children include the assessment of body proportions to establish whether a child’s low weight is attributable to wasting or stunting. Except for weight and height, hydration status should also be established (WHO, 1999; NDoH, 2003; Williams, 2005, p.406-407).

The clinical assessment of the child should include a history of feeding problems, GI symptoms, infections, recording of growth measurements, examination of the signs of malnutrition, as well as frequent monitoring of haemoglobin and other red blood cell values to detect anaemia. Children with growth faltering should be screened for GI infections; malabsorption and micronutrient deficiencies and acute severe malnutrition should then be managed according to clinical practice (Eley and Hussey, 1999). After

taking all circumstances into consideration a feeding plan should be developed. Figure 2.4 shows what actions to take in deciding how to feed the child (NDoH, 2003).

Figure 2.5 Action for handling failure to grow (NDoH, 2003)



The overall assessment of a sick child, together with the prescription of feeds and medication are directly related to the child's weight. Data collected in South Africa using the Child Healthcare Problem Identification Programme (Child PIP), shows that in 17% of child deaths during 2005 the weight was not known, to 10% in 2004. This shows serious problems in the basic assessment of sick children. It is a cause for great concern that increasing numbers of sick children in hospital are not weighed (Patrick and Stephen, 2005, p.14).

Patients with uncomplicated PEM should be treated outside the hospital whenever possible. While they are hospitalised they have a higher risk of cross-infections and the unfamiliar setting may cause apathy and anorexia, making feeding more difficult. Malnourished children with signs of a poor prognosis or with life threatening complications

and those living in conditions where there is inadequate medical and nutritional treatment should be hospitalised (Torún and Chew, 1994, p.965; Torún, 2006, p.896).

2.8.2 INITIAL PHASE / STABILIZATION PHASE

The primary aims of the treatment of severe malnutrition are to correct nutritional deficiencies, to treat accompanying diseases and to avoid relapse. The initial phase is managed in hospital where the patient is assessed and therapeutic and nutritional interventions are planned to treat life-threatening complications such as septicaemia (WHO, 1999; Orach and Kolsteren, 2002), hypoglycaemia, hypothermia, dehydration, electrolyte imbalances, underlying infections and micronutrient deficiencies (WHO, 1999; Orach and Kolsteren, 2002; NDoH, 2003; Williams, 2005, p.406-407). Malnourished children often have a poor inflammatory response and some of the physical signs may be difficult to diagnose or absent. Infection often presents as apathy, drowsiness, hypothermia, hypoglycaemia and death (Golden and Golden, 2000, p.523).

In this phase the child is a medical emergency with a high risk of mortality. Useful signs and symptoms are a history of watery diarrhoea or vomiting, thirst, dry mouth, low urinary output, weak and rapid pulse, low blood pressure, cool and moist extremities and problems with consciousness. Irritability and apathy complicate the assessment of mental awareness (Torún and Chew, 1994, p.965; Torún, 2006, p.896).

Children should ideally be kept in a special area where they can be monitored throughout the day and where the conditions in the room can be kept at specific temperatures, and while the children are very susceptible to infections it is better to keep them isolated from the rest of the patients (WHO, 1999).

The initial phase usually lasts about two to seven days, but if this phase continues for longer than ten days, the child is not responding to the treatment and more severe measures are needed (WHO, 1999). The initial response will show no change in weight due to the loss of oedema and large diuresis. After the first five to 15 days, there is a period of rapid weight gain or “catch-up”. The catch-up rate is slower in marasmus than in kwashiorkor. The catch-up weight gain is ten to 15 times higher than that of a normal child of the same age, and can be 20-25 times higher (Torún and Chew, 1994, p.969-970; Golden and Golden, 2000, p.524; Torún, 2006, p.902).

2.8.2.1 HYPOGLYCEMIA

The first step in the management of severe malnutrition is the prevention and treatment of hypoglycaemia. Severely malnourished children are prone to developing hypoglycaemia. Hypoglycaemia occurs while the child is waiting for medical attention or admission to hospital, or during the first few days in hospital if they haven't been fed for longer than 4-6 hours due to the fact that solid food is only given once the child has stabilized (WHO, 1999; Golden and Golden, 2000, p.523). Hypoglycaemia is usually the main cause of death within the first two days of treatment (WHO, 1999).

Once the child has started on the therapeutic feeds, giving the hospitalized child small feeds should prevent fasting. The feeds should be given frequently during the day and night (Pereira, 1991, p.145; WHO, 1999; Golden and Golden, 2000, p.523) at a frequency of every three hours without missing a feed. Small feeds of milk-based starter formula should be fed using a cup or spoon, not a bottle (NDoH, 2003).

Features of hypoglycaemia include a low body temperature (less than 36.5^{o}C), lethargy, limpness and clouding of consciousness. Other features are rigidity, twitching or convulsions and sweating and pallor are not usually present. Often the only sign of hypoglycaemia before death is drowsiness (WHO, 1999; Golden and Golden, 2000, p.523). Hypoglycaemic coma is a leading cause of mortality and morbidity (Pereira, 1991, p.145; NDoH, 2003).

Once hypoglycaemia is suspected, without confirmation, the child should be treated immediately. No harm can be done if the diagnosis is incorrect. If the child is conscious, the child must be given 50 milliliters (ml) of 10% glucose or sucrose, or F-75 (or a relevant infant formula) orally. If the 50% glucose is available, using one part glucose to four parts sterile water can dilute the 50% glucose. The child should be monitored until they are alert again. If the child is unconscious or has convulsions they can receive 5ml/kg of body weight of sterile 10% glucose intravenously and then 50 ml of 10% glucose or sucrose through a nasogastric tube. The moment the child is conscious, they must be fed with an infant formula, F-75 or glucose water (60g/L) (WHO, 1999).

2.8.2.2 HYPOTHERMIA

The second step in the treatment of severe malnutrition during the initial phase is the prevention and treatment of hypothermia. Children younger than twelve months, with

marasmus and with large areas of damaged skin or infections are usually prone to developing hypothermia (WHO, 1999; Golden and Golden, 2000, p.523). Hypothermia is present when the under-arm temperature is below 35^{o}C (WHO, 1999; NDoH, 2003) and the rectal temperature is below 35.5^{o}C, and indicates the need to immediately warm and feed the child (WHO, 1999). Children should be kept warm and should not be exposed to cold (WHO, 1999; NDoH, 2003), but the child should not be over heated either. The semi-nude patient should be kept in an ambient temperature of 25-30^{o}C. Even though this seems warm for active, clothed children, it is necessary for small, inactive children that can become hypothermic (WHO, 1999).

Children can be kept warm by practicing kangaroo mother care by putting the child against the parents' skin and covering them both, as well as covering the child's head. Fluorescent lamps and hot water bottles can be dangerous and should not be used. Children should not be lying close to an open window. Children should not be washed except if really necessary and the washing should be done during the day. After washing the child should be dried properly and dressed and covered immediately. The rectal temperature should be monitored every half an hour, but the underarm temperature is not reliable during the warming up of the child (WHO, 1999).

Loss of subcutaneous fat reduces the body's capacity to regulate temperature and water storage. This causes children to become dehydrated, hypothermic and hypoglycaemic more quickly and severely (Müller and Krawinkel, 2005).

Plasma glucose concentrations below 3.3 millimol per litre (mmol/L) (60 milligram (mg) per desilitre (dL) are caused by impaired thermoregulatory mechanisms. Body temperature rises in the hypothermic patient after frequent feeds of glucose foods or solutions (Torún and Chew, 1994, p.968; Torún, 2006, p.899) and therefore hypothermic children should also be treated for hypoglycaemia (WHO, 1999).

2.8.2.3 DEHYDRATION AND SEPTIC SHOCK

The third step in the management of severe malnutrition is to prevent and treat dehydration. Dehydration and septic shock are difficult to differentiate in malnourished children. Hypovolaemia is seen in both conditions and worsens without treatment. Dehydration can lead to 5-10% weight loss. With septic shock there can be diarrhoea and some dehydration and therefore the clinical picture is often confusing (WHO, 1999).

Most signs are unreliable in severely malnourished children, but there are some reliable signs that can help with diagnosis of dehydration. The unreliable signs are the mental state of the child, any abnormalities of the mouth, tongue and tears (malnutrition causes a dry mouth and absent tears) and skin elasticity (absence of subcutaneous fat make the skin loose and thin) (WHO, 1999).

Many children with severe malnutrition suffer from diarrhoea, and may therefore become dehydrated (WHO, 1999; NDoH, 2003). Signs of mild to moderate dehydration are an alert, thirsty child with palpable radial pulses, normal or slightly sunken fontanele and eyes, without loss of skin turgor over the abdomen. With severe dehydration, the child is drowsy, with cold extremities, a weak or feeble pulse, loss of skin turgor, and a decreased output of urine (Pereira, 1991, p.145; WHO, 1999).

Dehydration must be treated even though it is difficult to diagnose in severe malnutrition (Müller and Krawinkel, 2005). Dehydration should be treated orally as the use of intravenous fluids can lead to heart failure and overhydration and should only be used if the child is in shock (WHO, 1999). Rehydration should be slower than with well-nourished children and signs of overhydration must be monitored constantly (NDoH, 2003). Cardiac failure may develop in the presence of severe anaemia or shortly after the introduction of a high protein and high-energy feed or with a high sodium diet. This type of diet can lead to pulmonary oedema and secondary pulmonary infection. The latter can be the result of impaired cardiac function, expansion of the intravascular fluid volume, severe hypoxia or impaired membrane functions. Diuretics can be given but are contraindicated in kwashiorkor (Torún and Chew, 1994, p.968; Torún, 2006, p.898-899).

To restore the circulating volume, oral rehydration solution can be given in limited amounts, before feeds, for a short time (Golden and Golden, 2000, p.523). Children should receive the rehydration fluid orally or via nasogastric tube for the first four hours (except in shock), depending on the degree of dehydration (Pereira, 1991; NDoH, 2003).

The standard oral rehydration solution should be modified and diluted to reduce the sodium concentration (45mmol/L) and enrich the potassium content (40mmol/L), magnesium, zinc, copper and selenium (Table 2.19) (WHO, 1999; Golden and Golden, 2000, p.523; NDoH, 2003; Müller and Krawinkel, 2005; Torún and Chew, 1994, p.965;

Torún, 2006, p.897). The child should also receive magnesium, zinc and copper to restore deficiencies (Torún and Chew, 1994, p.969; WHO, 1999; Torún, 2006, p.900).

Table 2.19 Composition of oral rehydration salts solution for severely malnourished children (ReSoMal) (WHO, 1999)

Component	Concentration (mmol/L)
Glucose	125
Sodium	45
Potassium	40
Chloride	70
Citrate	7
Magnesium	3
Zinc	0.3
Copper	0.045
Osmolarity	300

The child should receive about 70-100 ml ReSoMal (Sorrel) per kg body weight for mild to moderate dehydration (Torún and Chew, 1994, p.966-967). Rehydration should start over 12 hours, beginning with 5 ml per kilogram every 30 minutes for the first two hours orally or via nasogastric tube and then 5-10 ml per kilogram per hour. Children should be reassessed every hour especially when the child is not drinking everything and if there are losses through stools and vomiting. Signs of increased pulse rates, engorged jugular veins and increased oedema should be monitored (WHO, 1999).

If the condition of the child has improved after 12 hours, but the child is still dehydrated the oral rehydration solution should be repeated for 12 hours. If the eyelids become puffy or oedema increases the child can receive breast milk or plain water, but not oral rehydration solution (Torún and Chew, 1994, p.966-967). Mothers should be encouraged to continue with breastfeeding every half hour during the period of rehydration (Torún and Chew, 1994, p.966-967; WHO, 1999).

Intravenous fluids are not indicated but can be used when the child is still vomiting after four hours. When the vomiting improves, oral rehydration solution can be given by mouth and if it is tolerated after two hours the tube can be removed (Pereira, 1991, p.146; Torún and Chew, 1994, p.966-967; Torún, 2006, p.896-897).

Rehydration is finished when the child is not thirsty anymore and is passing urine. A relevant infant formula such as F-75 can be given within the first two to three hours after rehydration was started (WHO, 1999). Alternative feeds of oral rehydration fluid and milk formula can be given. The strength of the milk feed can be increased and enriched feeds can then be given over the next few days (Pereira, 1991, p.146; Torún and Chew, 1994, p.966-967; WHO, 1999; Torún, 2006, p.896-897).

2.8.2.4 CORRECT MICRONUTRIENT DEFICIENCIES

The fourth and sixth step of the management of severe malnutrition is the correction of electrolyte imbalances and micronutrient deficiencies. Magnesium, zinc and phosphorus deficiencies can be common (NDoH, 2003; Ochoa *et al.,* 2004). Supplements containing copper, folic acid, multivitamins with extra potassium (NDoH, 2003), as well as other major electrolytes (Golden and Golden, 2000, p.523) and vitamin A can be supplemented in quantities higher than the daily recommended intakes (DRIs) for well-nourished persons of the same age (Torún and Chew, 1994, p.969; Torún, 2006, p.900). Supplementary calcium should provide 600mg/day, especially when no dairy is used (Torún and Chew, 1994, p.969; Torún, 2006, p.900). Deficiencies and nutrient imbalances can be aggravated with very high intakes (Golden and Golden, 2000, p.523). Folic acid (5 mg) can be given orally on admission (WHO, 1999; Golden and Golden, 2000, p.523) then 1 mg every day thereafter (WHO, 1999). Some children can also benefit from riboflavin, ascorbic acid, pyridoxine, thiamin and the fat-soluble vitamins D, E and K (WHO, 1999; Golden and Golden, 2000, p.523).

Iron should not be started in the initial phase (NDoH, 2003). Supplemental iron should only be given one week after dietary therapy has been initiated. Earlier supplementation of iron will cause a hematologic response and increase bacterial growth (Torún and Chew, 1994, p.969; Torún, 2006, p.900).

Zinc and vitamin A deficiency impair the function of the immune system and the structure and function of the mucosa. Therefore these nutrients can be used for the recovery of the intestinal mucosa. Vitamin A and zinc supplements can reduce persistent diarrhoea as well as reduce the rate of treatment failure and death due to persistent diarrhoea (Ochoa *et al.,* 2004).

When vitamin A deficiency is suspected, it must be treated because diagnosis is difficult, especially in areas with known measles and vitamin A deficiencies. A dosage according to age should be given on admission (Torún and Chew, 1994, p.968; WHO, 1999; Torún, 2006, p.899). Dosages are as follows: 50 000 International Units (IU) for infants less than six months of age, 100 000 IU for infants 6-12 months and 200 000 IU for children older than 12 months. These should only be given if there is evidence that the child hasn't received vitamin A during the last month (WHO, 1999).

Ocular lesions can develop as a result of increased needs for retinol when protein and energy feeds are started. Children with xerosis of the conjunctiva and Bitot's spots can develop keratomalacia with corneal haziness and/or ulceration. When the cornea is involved, the child becomes photophobic (Pereira, 1991, p.146; Torún and Chew, 1994, p, 968; Torún, 2006, p.899). If any of these clinical signs of vitamin A deficiency are present, another dose should be given on day two and a third dose two weeks later (Pereira, 1991, p.146; Torún and Chew, 1994, p, 968; WHO, 1999; Torún, 2006, p.899).

2.8.2.5 INFECTIONS

The fifth step in the treatment of severe malnutrition is the treatment of infections (NDoH, 2003). Almost all malnourished children have bacterial infections when they are admitted to hospital (WHO, 1999). The child should be monitored for weight, temperature, urine output, cardiac output, and fluid intake. Children with SAM are at risk of infections and should be treated with anti-helminthic medication from from six months of age (Marino *et al*., 2007).

If there is no improvement in the child (disappearance of oedema), health professionals should check if the food has been prepared correctly and if the child has consumed the correct amount of food. If the correct amount of food is eaten but the child is not gaining weight, the child should be checked for infections (Pereira, 1991, p.148). Clinical manifestations may be mild, without fever, tachycardia and leukocytosis (Torún and Chew, 1994, p.967; NDoH, 2003; Torún, 2006, p.898) and are difficult to detect in malnourished children that are apathetic and drowsy (WHO, 1999).

TB, otitis media, an abscess or urinary tract infection may cause poor weight gain (Pereira, 1991, p.148). Respiratory tract infections are also common in children with PEM. Increased respiratory rate, cough and subnormal temperature are sometimes the

only signs of bronchopneumonia (Pereira, 1991, p.146). Supportive treatment for respiratory distress, hypothermia and hypoglycaemia is needed (Torún and Chew, 1994, p.967; Torún, 2006, p.898). Other infections may include GI, malaria, measles, or HIV-related illness (Golden and Golden, 2000, p.523; Williams, 2005, p.407). The infection must first be treated before the nutritional status will improve (Pereira, 1991, p.148). Every child should be vaccinated against measles, because of the high mortality rates linked to PEM and measles (Torún, 2006, p.898).

Patients with severe malnutrition should automatically be treated with antibiotics (WHO, 1999; NDoH, 2003) due to the high mortality rate from infections (Torún and Chew, 1994, p.967; Torún, 2006, p.898). The antibiotics should be broad-spectrum (Golden and Golden, 2000, p.523; NDoH, 2003; Williams, 2005, p.407) for gram-negative and gram-positive infections, even though the results of the microbiologic cultures are not always yet available. Gram-negative infections are more common (Torún and Chew, 1994, p.967; Torún, 2006, p.898).

Treatment with antibiotics is divided into first-line and second-line treatment. Children with no obvious signs are treated with first-line antibiotics, such as cotrimoxazole twice daily for five days. Children with complications should receive ampicillin every six hours for two days, then amoxicillin every eight hours for five days and then gentamicin once daily for seven days (WHO, 1999; Fuchs *et al.,* 2004). If the child does not improve within 48 hours, cholramphenicol should be added every eight hours for five days. All antimicrobals should be used for five days. If anorexia persists medication should be given for another five days and if there is no improvement after ten days of treatment, the child should be reassessed (WHO, 1999).

The WHO guidelines do not take into account the different bacteria present in different areas. These differences influence the antimicrobial regimen to be used. This aspect of the WHO guidelines needs to be improved and refined and a complementary guideline should be developed for non-hospital settings (Fuchs *et al.,* 2004).

2.8.2.6 DIARRHOEA

Nutritional management of diarrhoea is important. Mothers are encouraged to give energy-dense, protein-rich food during episodes of diarrhoea, since this gives the child a chance to produce glutathione, which metabolizes aflatoxins and other toxins responsible

for the pathogenesis of kwashiorkor (Oyelami and Ogunlesi, 2007). In children with gastroenteritis, dehydration is present even though there is oedema. These children have sunken eyes, sunken fontanels and decreased skin turgor with oedematous legs (Pereira, 1991, p.145).

If the mother is still breastfeeding, she should not stop feeding the child and should give more frequent, longer breastfeeds during the day and night. Oral rehydration solution must be given after each loose stool. Fermented milk products such as yoghurt or amasi are better tolerated and can form part of nutrient dense semi-solid foods needed by the child (NDoH, 2003).

2.8.2.7 DIETARY TREATMENT

Nutritional treatment should start once the life-threatening conditions, present in the child, have been treated. The slow onset and progression of the disease gives the body time to make metabolic adjustments. Reversal of these adjustments should be gradual during the early stages of nutritional treatment, to avoid metabolic disruptions (Torún, 2006, p.899). Malnourished children cannot tolerate the usual amounts of dietary protein, fat and sodium. It is important to start these children on feeds that are low in the abovementioned nutrients and high in carbohydrates (WHO, 1999).

In order to develop the correct diet or nutritional plan, it is important to see what characteristics of PEM are present. If enough information is not available, the starved, unstressed, hypometabolic patient can develop complications of overfeeding while the stressed, hypermetabolic patient can suffer consequences of underfeeding (Heimburger, 2006, p.834).

Two types of feeds are used for malnourished children, F-75 and F-100, which provide 75 kcal per 100 ml and 100 kcal per 100ml respectively. The F-75 is used during feeding in the initial phase and F-100 is used during the rehabilitation phase (Appendix B and D). Commercial feeds F-75 and F-100 that can be mixed with water are available, but are not currently used in South Africa. The mineral mix that must be added contains potassium, magnesium and other essential minerals (WHO, 1999). In South Africa F-75 and F-100 are prepared by diluting cows milk and adding sugar and oil in specific amounts. If at all possible, other infant formula, such as Nan Pelargon should be used.

During dietary treatment, the main objective is to rebuild wasted tissues so as to achieve catch-up growth. Once a child's appetite has returned, the diet can gradually change from the starter to a catch-up formula. The aim is to reach an intake of 630-840 kJ/kg/day (150-200 kcal/kg/day) and 4-6 g protein/kg/day. The child's appetite should determine the food intake (NDoH, 2003) (Appendix E).

Liquid feeds given orally or nasogastrically as 6-12 feeds per day are dependant on age and condition. Small volume feeds should be given frequently over 24 hours, ensuring the patient does not fast for more than four hours and to prevent vomiting, hypoglycaemia and hypothermia (Torún and Chew, 1994, p.968; WHO, 1999; Torún, 2006, p.899). Feeding should be given day and night and if vomiting occurs, the amount and the intervals should be decreased. The feeds should be given according to a specific schedule as shown in Appendix C. The feeds should increase in volume and decrease in frequency until the child is receiving four hourly feeds (WHO, 1999).

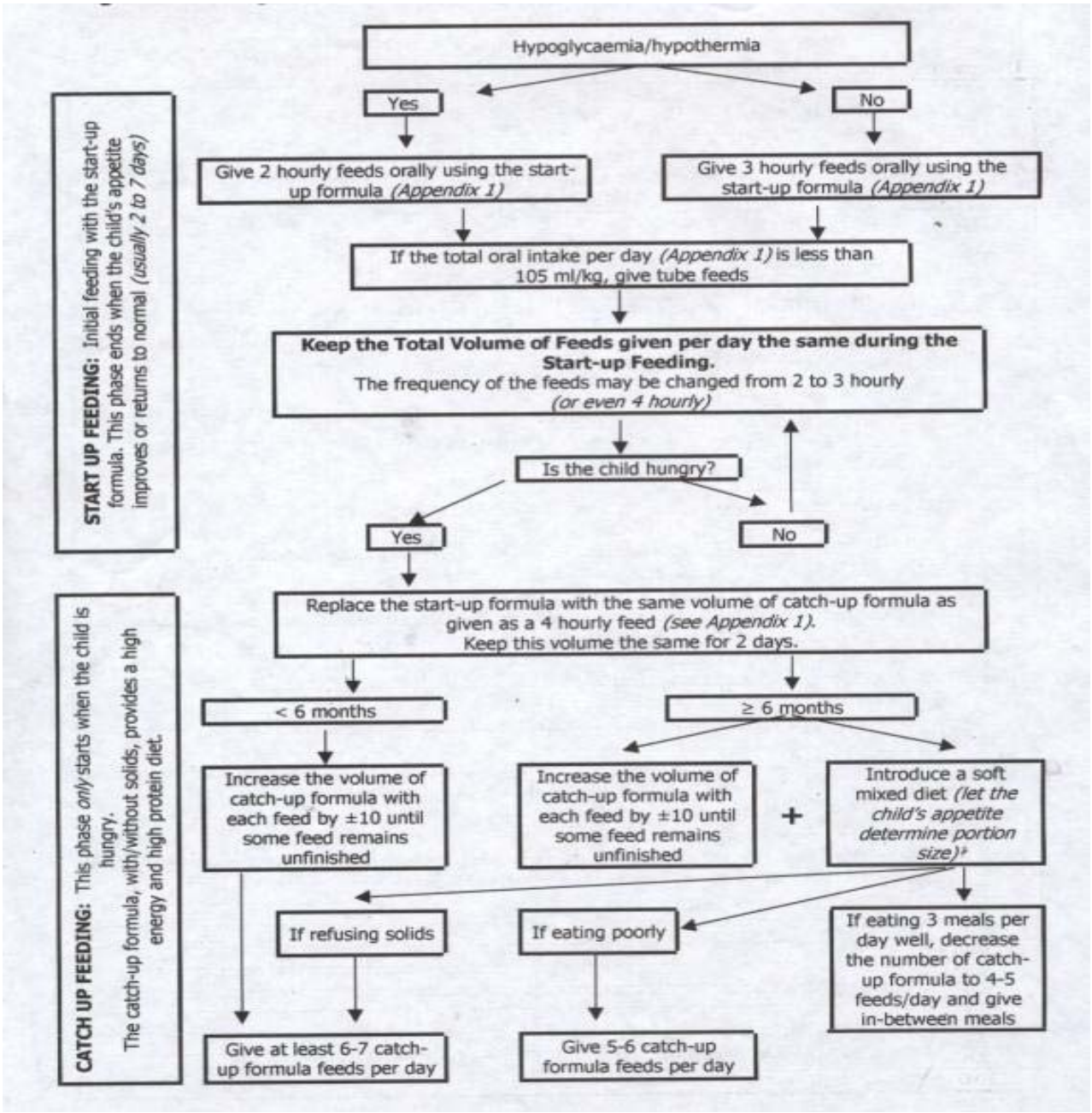
Children should be fed with a spoon or cup, should be coaxed to eat and should sit in an upright position on the mother's lap. Children should never be left alone to eat and if they are very weak can be fed with a syringe or dropper. Children that do not tolerate food orally can be fed using a nasogastric tube, but they need to be evaluated regularly for intake to make sure that there are no problems associated with the tube. Intake and output or losses should be recorded (WHO, 1999).

HIV uninfected children can be treated with nutrition therapy alone, but HIV infected children need specially developed regimens. It is important to take the metabolic and nutrient needs of HIV infected children into consideration when therapeutic diets are designed. The use of F75, F100 and ready-to-use food are generally part of the care of HIV infected malnourished children. In HIV uninfected malnourished children, appetite can be used to determine nutritional recovery but with HIV infected malnourished children, anorexia is common. Severe diarrhoea is associated with high case-fatality rates and feeding regimens should be designed accordingly. Mortality within four to six weeks remains unacceptably high (3-8%) in Sub-Saharan Africa (Heinkens *et al*., 2008). Early on in HIV infection, vitamin A supplementation reduces the morbidity, mortality and infectious complications of measles, immune suppression, pneumonia and diarrhoeal disease (Semba, 2006, p.1403).

The appetite is used as a barometer of progress. When the child has anorexia it is a warning that something is metabolically wrong. When the patient has an appetite and is hungry, it should mark the end of the initial phase (WHO, 1999; Golden and Golden, 2000, p.524). Hunger means that the infections are under control, the liver can metabolise the diet and other metabolic abnormalities are improving. This indicates the beginning of the rehabilitation phase (WHO, 1999).

The graph in figure 2.6 can be used to determine the best route for feeding a child.

Figure 2.6 Feeding a child with severe PEM after stabilization (NDoH, 2003)



(Appendix 1 refers to Appendix B (NDoH, 2003))

2.8.3 REHABILITATION PHASE

Nutritional rehabilitation must start slowly and progress gradually (Torún, 2006, p.899). The child should be encouraged to eat as much as possible and breastfeeding should not be ceased (WHO, 1999). This stage starts when the patient has no serious complications, is eating satisfactorily and gaining weight (Torún and Chew, 1994, p.970; Torún, 2006, p.903). When the appetite returns, it signifies that infections are under control and that there are no major electrolyte imbalances or deficiencies (Golden and Golden, 2000, p.524; Orach and Kolsteren, 2002). This is usually about two to three weeks after admission. This phase starts in the hospital and then progresses to outpatient treatment (Golden and Golden, 2000, p.524; Torún and Chew, 1994, p.970; Torún, 2006, p.903) at home (Orach and Kolsteren, 2002).

During this phase the aim is to replete stores and promote catch-up growth (Williams, 2005, p.407). The patient's physiological responses are still abnormal and his/her capacity may still be limited. Deficiencies of potassium, magnesium, zinc and the other components of new tissue are still present. The production of new tissues will require increased amounts of all these components except for protein and energy (WHO, 1999; Golden and Golden, 2000, p.524). Children must be closely monitored throughout the treatment (WHO, 1999; NDoH, 2003; Williams, 2005, p.406-408), and should be weighed every day with the plotting of their weights. A weight gain of 10-15g/kg/day is satisfactorily and if the weight gain is less than 5g/kg for three consecutive days, it shows that the child is not responding to the treatment. The discharge weight is usually seen as 90% of expected weight for age and most children reach this target weight within two to four weeks (WHO, 1999).

Nutrients can be ingested in high therapeutic amounts, so that enough of the nutrient can be absorbed for nutritional recovery (Golden and Golden, 2000, p.524; Torún and Chew, 1994, p.970; Torún, 2006, p.903). The introduction of therapeutic diets and appropriate rehydration fluids, as well as F-100, ReSoMal (Sorrel) and ready-to-use food has improved the rehabilitation process and shortened hospital stay, especially of HIV uninfected severely malnourished children. Therapeutic diets also address micronutrient and macronutrient deficiencies. Appropriate diets are needed for the severely malnourished infants younger than six months of age, because unmodified F-75 and F-100 is unsuitable for them (Heinkens *et al*., 2008).

F-100 can be given during the rehabilitation phase and the volume can be increased by 10 ml increments. The moment a feed is finished at one feeding time, the next feed should be increased. Intake should be recorded and any food that is not eaten should be thrown away. It may seem as if the child is not gaining weight due to the loss of oedema. The difference in feeding a child younger than 24 months compared to a child older than 24 months is related to the volume given. Solids are still important and a good practice is to start introducing the same foods as available in the communities so that parents can relate to what the child is eating. Once the child is growing well, the feeds can be reduced to five times in 24 hours (WHO, 1999).

The energy and protein intake should start at low maintenance requirements and progress by small increments. The child can receive six to twelve liquid feeds depending on age and condition. Children should not be without food for more than four hours. The solid food that older children receive must consist of food of a high quality, concentration and should be easily digestible (Torún, 2006, p.899), safe and palatable with high-energy content and adequate amounts of vitamins and minerals (Torún and Chew, 1994, p.970; Torún, 2006, p.903). Sometimes it is possible to design an appropriate therapeutic diet with locally available, nutrient-dense foods with added micronutrient supplements (WHO, 2007a) and the child can still eat adequate protein, energy and other nutrients; even when traditional foods are introduced (Torún and Chew, 1994, p.970; Torún, 2006, p.903).

Food given for home treatment should be foods that do not need refrigeration and can be used in areas where hygiene conditions are limited. A malnourished child with an appetite, aged six months or older can be given standardized ready-to-use food if there are no medical complications and if careful monitoring is done (WHO, 2007a). Ready-to-use food (not yet available in South Africa) is soft or crushable food that can be used from the age of six months without adding water (Torún and Chew, 1994, p.970; Torún, 2006, p.903). Together with the ready-to-use food, children need a short course of basic oral medication to treat infections. Children at home can eat food at home with minimal supervision, if they have an appetite (WHO, 2007a).

The attitude of the person giving the feeds together with the appearance, colour and flavour of the foods is important to treat a lack of appetite and low food acceptance. Patience and loving care are needed to encourage children to eat all of the diet (Torún

and Chew, 1994, p.969; WHO, 1999; Torún, 2006, p.902). Mothers must be encouraged to hold their children, smile and talk to their children (WHO, 1999).

Parents and caregivers must be educated on feeding practices and food preparation as well as other aspects of their child's health care (WHO, 1999; NDoH, 2003; Williams, 2005, p.406-408). Parents should also be educated on reasons why the child became malnourished and preventing a relapse. Basic practices such as hygiene, what food to buy, food preparation and play activities and stimulation should also be taught to parents and caregivers (WHO, 1999).

During the rehabilitation phase the child should be assessed for abnormal behaviour such as "frozen watchfulness", rumination or head banging (Williams, 2005, p.408). This phase can be used to update the child's immunizations (WHO, 1999; Williams, 2005, p.408) and for diagnosing and treating any chronic underlying problems such as anaemia or TB (Williams, 2005, p.408).

Rehabilitation involves more than feeding, such as play and environmental stimulation and emotional support that can lead to accelerated catch-up growth and improved long-term cognitive outcomes (WHO, 1999; NDoH, 2003; Williams, 2005, p.406-408). Play programmes should start in the hospital and continue after discharge to prevent permanent mental retardation and emotional impairment. To support these programmes, hospital wards should be improved with bright colours (walls and staff uniforms) and toys must be available in the ward and in the cots. Even inexpensive, homemade toys can be effective. Aside from free play during the day, there must also be some planned activities by staff for at least 15-30 minutes. Play activities should also include physical activities to promote the development of motor skills (WHO, 1999).

There are still challenges regarding patients with extreme anaemia and those who are close to cardiac failure (Müller and Krawinkel, 2005). Vitamin and mineral mixes should still be used throughout the rehabilitation phase and iron can now be added to the diet (WHO, 1999). The WHO are in the process of revising their 10-steps for the Management of Severe Acute Malnutrition to include three problem areas: nutritional problems of children with HIV and AIDS, dietary regimes for infants younger than six months and limited availability of potassium-magnesium-zinc-copper preparations (Müller and Krawinkel, 2005).

2.8.3.1 NUTRIENT REQUIREMENTS

2.8.3.1.1 ENERGY

Recovery is usually delayed by infections, which increase energy requirements. There are times when the patient does not gain weight, even though infections are absent and the diet has sufficient energy and protein. An increased energy intake during the first weeks of treatment shows an improvement in condition (Monckeberg, 1991, p.126). Patients need the essential nutrients to recover. If one nutrient is missing the body cannot use the others and the result will be metabolic stress (Golden and Golden, 2000, p.523).

Oedematous children (kwashiorkor) must receive small, frequent, two hourly meals orally or via nasogastric tube at about 100 ml/kg/day after 4-5 days in hospital. With a high-energy regime the oedema recovers quickly and there is an average weight gain of 70 g/kg/week (Pereira, 1991, p.147; Williams, 2005, p.407). Children with kwashiorkor will recover weight-for-height in about 4-6 weeks (Pereira, 1991, p.147). A specialized feed of low osmolality and lactose should be given. The volume of the feed should be 130 ml/kg/day and provide 1-1.5g/kg/day protein. The feed should be supplemented with breastfeeding and an iron and micronutrient supplement should be added (Williams, 2005, p.407). (Start-up formula recipes – Appendix B)(Feed volumes – Appendix C)(Catch-up formula recipes – Appendix D).

Marasmic children have a lower proportion of fat and higher proportion of lean body mass. Lean body mass is metabolically more active than fat tissue. Marasmic infants should be maintained on energy intakes of 120kcal/kg/day. The efficiency of the diet improves and weight gain starts at a slow rate. Sometimes marasmic patients have lactose malabsorption. Undigested lactose, short chain fatty acids and other substances are found in the faeces (Monckeberg, 1991, p.128).

Two weeks after the high-energy diet has been started, the child may develop signs of nutrition recovery syndrome. The child's appetite decreases once weight-for-height is reached and the clinical features of the nutritional recovery syndrome then begin to fade or disappear. When the children start gaining weight, traditional food should be added (Pereira, 1991, p.147). A general guideline is to provide a total intake of supplements and a home diet of at least twice the protein needs and 1.5 times more than that of energy (Torún and Chew, 1994, p.973).

2.8.3.1.2 PROTEIN

If the protein is of a high quality an intake of 2g-protein/kg-body weight is adequate. Positive results are seen with an intake of 3-4 g milk protein /kg/day (Monckeberg, 1991, p.128; Pereira, 1991, p.147). A very high protein intake is unnecessary. The use of plant proteins is often not sufficient and may cause the rehabilitation to take longer (Pereira, 1991, p.146).

Marasmic children have a reduced renal function and they cannot excrete the substances resulting from the ingestion of large amounts of protein. Proper hygiene in the preparation of formulas is important (Monckeberg, 1991, p.128).

2.8.3.2 REFEEDING SYNDROME

The refeeding syndrome is defined by the metabolic and physiologic consequences of the depletion, repletion and shifts of fluid, electrolytes, vitamins and minerals as the malnourished patient is fed (Morgan and Weinsier, 1998, p.189; Marino *et al.,* 2007). Refeeding syndrome can also be described as a decrease in phosphate levels of below 0.65 mmol/L. Signs associated with refeeding syndrome are hypophosphatemie, hypokalemie, hypomagnesaemia, altered glucose metabolism, fluid balance abnormalities and vitamin deficiency especially thiamine (Marino *et al*., 2007). Refeeding commonly occurs in marasmus and kwashiorkor. Refeeding of chronically starved, hypometabolic patients, can be complicated by heart failure and hypophosphatemie (Morgan and Weinsier, 1998, p.189).

Figure 2.7 Pathogenesis of refeeding (Marino *et al*., 2007)

Refeeding
↓
Conversion to glucose as major energy source instead of protein and ketones
↓
↑requirements for phosphorus for Kreb's cycle
↓
↑Insulin release*
↓
↑ cellular glucose, calcium, phosphorus, magnesium & total body weight uptake,
↓
↑ protein synthesis and utilization of thiamine
↓
Intracellular shifts of calcium, phosphorous, magnesium resulting in extracellular depletion of calcium, phosphorus, and magnesium

The major body fuels of marasmic patients are ketones and fatty acids. A portion of the diet fuel should be fat as marasmic patients rely on fatty acid metabolism. The aim is to replenish body stores slowly (Morgan and Weinsier, 1998, p.190-191) and caution must be taken not to refeed malnourished children too quickly. Giving excessive amounts of fluid and high-energy food will cause a shift in fluid between the intra- and extracellular fluid compartments. This can change plasma electrolyte concentrations, cause cardiac and respiratory failure and may kill the child (Williams, 2005, p.406; Marino *et al*., 2007). Refeeding syndrome can also cause respiratory, neuromuscular, renal, haematological, hepatic and gastrointestinal problems. Replacement treatment must be given each time electrolyte levels are below normal (Marino *et al*., 2007).

Energy should be increased gradually in the first week of treating a patient with refeeding syndrome. In the first week the patient should only receive 75% of total daily requirements for actual body weight. In very severe cases 60% of the requirements should be given according to the hydration status of the patient (Table 2.20). Once the food is tolerated it can be increased gradually (Marino *et al*., 2007).

Table 2.20 Energy requirements for patients with refeeding syndrome (Marino *et al*., 2007)

Age	Total daily requirements according to actual body weight (abw)	75% of the total daily requirements
Birth – 1 years	90 -110kcal/kg/abw/day	75% = 80kcal kcal/kg/day19
1 - 7 years old	80 –100kcal/kg/abw /day	75% = 60kcal – 75kcal/kg/day
7-10 years	60 - 75kcal/kg/abw/day	75% = 55kcal/kg/day
11-14 years	60kcal/kg/abw/day	75% = 45kcal/kg/day
15-18 years	50kcal/abw/day	75% = 35kcal/kg/day
> 18 years	25-35kcal/abw/day	75% = 20 – 25kcal//kg/day

Protein should not be given in excess in the initial refeeding phase as it can result in acidosis, azotaemia, hypertonic dehydration and hypernatraemia. Children can tolerate milk that contains lactose therefore lactose and sucrose free milk should only be used with episodes of severe diarrhoea (Marino *et al*., 2007).

2.8.4 DISCHARGE

The last step in the management of severe malnutrition is to prepare the child or infant for discharge and follow-up (NDoH, 2003). There is no definite line between the end of the rehabilitation phase and the discharge phase. The preparation phase starts in the rehabilitation phase and ends after discharge (Golden and Golden, 2000, p.524). Treatment up to the end of recovery should not take place in the hospital. When all life-threatening conditions have been treated, appetite is back, oedema and skin lesions have disappeared and the patient smiles, interacts with staff and other patients and is gaining weight they should be referred to a clinic or rehabilitation centre for the final stages of treatment (Torún and Chew, 1994, p.972). Before the child is discharged the F-100 or formula being given, must be decreased gradually and the mixed diet should be increased accordingly (WHO, 1999).

When breastfed infants are supplemented it should be with cereals and solid foods that will not influence the infant's thirst and therefore change the infant's demand for breast milk (Torún and Chew, 1994, p.973; Torún, 2006, p.905). Supplementing the home diet with foods that are easily digested, containing protein of high biologic value, a high energy density and adequate micronutrients, should be used to treat less severe forms of PEM at clinic level or at home. It is necessary to provide nutritious food supplements and instructions for use. The quantity of the supplement will depend on the degree of malnutrition (Torún and Chew, 1994, p.972-973; Torún, 2006, p.905). By using outpatient therapeutic care the child will receive about 837 kJ/kg/day, which can be given by using ready-to-use food (Collins *et al*., 2006).

Premature termination of treatment increases the risk of recurrence of malnutrition. All fully recovered patients should reach the weight expected for their height (Torún and Chew, 1994, p.972; WHO, 1999; Torún, 2006, p.904). The weight-for-height of –1 SD (90%) of the median NCHS/WHO reference values must be reached. If a child is discharged before this goal is reached, intensive follow-up is needed (WHO, 1999). A general guideline for dietary therapy is to continue treatment for one month after reaching a normal weight without signs of kwashiorkor or marasmus. Some remain underweight because of a lower growth curve. After one month of adequate dietary intake and weight gain, continuation of a normal growth rate and no complications, treatment can be stopped (Torún and Chew, 1994, p.972; Torún, 2006, p.904).

The mother should be available in the ward as much as possible. Malnourished children need affection and care from the start of treatment and need to interact with other children when they become active. Patience and understanding by staff and the child's relatives is required. Malnourished children have delayed mental and behavioural development, which needs treatment as much as their delayed physical development. Mental retardation can be reduced through psychological stimulation with play programmes in the hospital and at home (Golden and Golden, 2000, p.524). Stimulation, play and loving care will markedly improve the child's response to treatment and decrease the period of hospitalisation (NDoH, 2003).

If care centres are not available, the child should not be discharged until ready. The mother or caregiver must understand the importance of a high energy, high protein diet until the child has recovered. Follow-ups are necessary for out patients at homes or clinics. Persistent diarrhoea, intestinal parasites and other minor complications should be treated and children should be vaccinated during this period (Torún and Chew, 1994, p.972).

Before discharge, parents should be educated on the causes of PEM and how to prevent malnutrition through correct child feeding practices, the use of household foods, food preparation, personal and environmental hygiene, immunizations, and early management of diarrhoea and other diseases (Torún and Chew, 1994, p.972; Torún, 2006, p.904). Parents should also be educated on how to enrich available foods (WHO, 1999). Other children in the household are also at risk of developing malnutrition. Nutrition and health education should include prevention or correction of nutritional problems for all family members in the household as the malnutrition started there. After discharge, the child should be integrated into the family and community (Golden and Golden, 2000, p.524).

Health promotion must include education and promotion for community leaders, local action groups and communities as a whole. These programmes should focus on all the factors involved in the development of malnutrition and include issues such as promotion of breastfeeding, appropriate use of weaning foods, nutritional alternatives and traditional foods, personal and environmental hygiene, water and sanitation, feeding practices in times of illness and convalescence, immunizations, early treatment of diarrhoea, pneumonia, other diseases, family planning, sexually transmitted infections (STIs), improving food security strategies and adult literacy (Golden and Golden, 2000, p.524).

2.8.5 FOLLOW-UP

Recovery is related to the age of admittance to hospital. Treatment before six months of age can result in a complete recovery of psychomotor development. Affective and psychomotor stimulation is useful for treatment and as important as an adequate diet. When the environment does not provide adequate stimulation, intellectual development is impaired. Growth will resume once an adequate nutritional status is achieved (Monckeberg, 1991, p.130).

Affection and tender care are needed from the start of treatment. The involvement of parents or relatives should be encouraged (Torún and Chew, 1994, p.972; Torún, 2006, p.904) and mothers should be part of the treatment program and must be educated in basic childcare (Monckeberg, 1991, p.130). The ability of the family to provide adequate nutrition and care at home must be ensured. Parents and caretakers should be involved in the feeding and care of children (NDoH, 2003). This ensures a continuation of recovery after discharge and prevents relapses (Monckeberg, 1991, p.130). Each child's progress must be regularly followed-up and checked (NDoH, 2003). Follow-up appointments should be scheduled for one week after discharge. Extra care should be taken to prevent relapse and at each visit the mother must be asked about the child's general health (WHO, 1999).

2.9 CONCLUSION

Malnutrition is an individual and societal problem, at medical, social, ethical, moral and political levels. Malnutrition amongst children is the most common serious illness in the world today. The legacy of childhood malnutrition is seen in adults who are physically and mentally stunted (Golden and Golden, 2000, p.525).

With poor nutritional status, the development of kwashiorkor is caused by the occurrence and intensity of infections, toxins, or specific nutrient deficiencies (Jackson and Golden, 1991, p.141). To maintain and restore lost weight and lean body mass the negative effects of the infections must be eliminated and this is done by providing enough calories and nutrients, doing sufficient exercise and receiving nutrition counselling (Fenton and Silverman, 2008, p.1009).

Correct nutrition will ensure healthier children, who grow into more productive adults. Nutrition can also help the body to fight infections, improved pregnancy outcomes and lead to economic growth through enhanced productivity (NDoH, 2003). Improvement in the nutritional status of children depends on the improvement in the socio-economic status of families and improvements in public health (Cartmell *et al*., 2005).

CHAPTER 3: METHODOLOGY

3.1 INTRODUCTION

This main focus of this study was to determine the factors contributing to malnutrition in the Northern Cape. Data was collected using a questionnaire and taking anthropometric measurements. The questionnaire gathered information regarding background information on mother and child, medical history of mother and child, lifestyle choices of the mother, infant feeding and current intake according to the Food Based Dietary Guidelines (FBDGs). The socio-demographic information and educational background of the mother was also determined and associations between variables were investigated.

3.2 METHODS

3.2.1 SAMPLING

3.2.1.1 POPULATION

In the Northern Cape the two biggest hospitals fall within two of the five districts comprising the Northern Cape. The Kimberley Hospital Complex falls within the Frances Baard district and the Gordonia Hospital Complex falls within the Siyanda district.

Very few of the children admitted to pediatric wards / infant care units in these two facilities are admitted with the diagnosis of severe malnutrition. In 2006-2007 about 60 children were admitted to Gordonia Hospital Complex for severe malnutrition, diagnosed as marasmus and kwashiorkor. Data for the same period for Kimberley Hospital Complex was limited seeing as most of the cases seen in this facility had severe malnutrition as the secondary diagnosis.

3.2.1.2 SAMPLE

The sample consisted of a convenience sample of all malnourished children 0-60 months, admitted to paediatric wards / infant care units in Kimberley Hospital Complex in Kimberley and Gordonia Hospital Complex in Upington, in the Northern Cape, between August 2007 and July 2008. The expected sample size was about 70 or more in Gordonia Hospital Complex and 80 or more in Kimberley Hospital Complex and the sampling ended when no less than 150 children were sampled.

Inclusion criteria:

- All malnourished children 0-60 months with a weight-for-age below 80 % of expected weight, admitted to paediatric or infant care units, whose mother/ caregiver was present.
- All malnourished children 0-60 months with a mother / caregiver present that signed the informed consent form (Appendix F, G and H).
- Malnourished children 0-60 months with a RtHC.

3.2.2 STUDY DESIGN

The study comprised of a cross-sectional hospital survey.

3.2.3 OPERATIONAL DEFINITIONS

The following operational definitions were defined:

3.2.3.1 BACKGROUND INFORMATION

For the purpose of this study the background information of the child related to the type of malnutrition present. Children were classified according to the criteria listed in table 3.1. Other background information included information gathered from the RtHC, for instance birth weight, current weight, clinic attended, last clinic visit, reason for last clinic attendance, regularity of clinic visits after birth, the town the clinic is situated in, medical treatment or history, immunizations and vitamin A supplementation. Other information included where the child was born, whether the child was premature, gestational age of the child and if the child was currently part of the Nutrition Supplementation Programme (NSP) at the clinic and for how long.

Table 3.1: Classification of malnutrition (Based on the WHO classification, 1971, Passmore & Eastwood, 1986, p. 281)

FORM OF PEM	BODY WEIGHT AS % OF STANDARD* (50TH PERCENTILE)	OEDEMA	INADEQUATE WEIGHT FOR HEIGHT
Kwashiorkor	80 – 60	+	+
Marasmic Kwashiorkor	< 60	+	++
Marasmus	< 60	0	++
Nutritional dwarfing	< 60	0	Minimal
Underweight child	80 – 60	0	+

\+ Means that oedema and inadequate weight is present

++ Means that oedema and inadequate weight is excessively present.

3.2.3.2 ANTHROPOMETRIC STATUS

For the purpose of this study anthropometric measurements included weight, height and MUAC in children. Measurements taken of the mothers / caregivers included weight and height to determine BMI. The cut-off points for these measurements are shown in tables 3.2, 3.3 and 3.4.

(a) Weight and height

In children, weight and height were classified according to the deviation from the NCHS median (table 3.2) (Gibson, 2005, p.242).

The -2 SD refers to the Z-score that is 2SD below the median and is similar to the 3rd percentile as shown in the RtHC. Weight-for-age, height-for-age and weight-for-height were classified as severe malnutrition (< -3 SD), moderate malnutrition (< -2 SD and > -3 SD) and mild malnutrition (< -1 SD and > -2 SD), normal (+1 SD) and overweight (>2 SD). The applied criteria for Z-scores < -2 SD includes:

Table 3.2: Cut-off points for underweight, stunting and wasting in children

Measurements	**Standard deviation**		**Type of malnutrition**
Weight-for-height	<-2 SD	Wasting	Acute, severe malnutrition
Weight-for-age	<-2 SD	Underweight	Acute malnutrition
Height-for-age	<-2 SD	Stunting	Chronic malnutrition

$$\text{Z-score} = \frac{\text{(observed values)-(median reference values)}}{\text{Standard deviation of the reference population}}$$

(Cogill, 2003, p.40-42)

(b) Body mass index (BMI)

In adults, BMI refers to the current weight (kg) divided by the height $(m)^2$. For the purpose of this study the classification for the BMI of mothers/caregivers is shown in table 3.3.

Table 3.3: Classification of BMI of the mother / caregiver (Gee *et al*., 2008, p.540)

Less than 18,5 kg/ m^2(square meters)	Underweight
Between 18,5 and 24.9 kg/m^2	Normal or healthy weight
Between 25- 29.9 kg/m^2	Overweight
Between 30-34.9 kg/m^2	Obesity, class I
Between 35-39.9 kg/m^2	Obesity, class II
Above 40 kg/m^2	Extreme obesity, class III

(c) Mid-upper arm circumference (MUAC)

For the purpose of this study the cut-off points for MUAC in children to classify malnutrition are shown in table 3.4.

Table 3.4: Cut-off points for classification of malnutrition using MUAC in children

< 11.0 cm	Severe malnutrition
11.1 – 12.5 cm	Moderate malnutrition (with or without oedema)

(Cogill, 2003, p.41)

3.2.3.3 IMMEDIATE FACTORS

For the purpose of this study immediate factors contributing to malnutrition include the following:

- Breastfeeding and other feeding practices where exclusive breastfeeding and partial breastfeeding are classified as follows:

    - **Exclusive breastfeeding**: where an infant receives only breast milk and no other liquids or solids, not even water, with the exception of drops or syrups consisting of vitamins, mineral supplements or medicines (Bland, 2007).
    - **Partial breastfeeding**: where an infant receives some breastfeeds and some artificial feeds, either milk or cereal, or any other food (Bland, 2007).

- Adherence to the FBDGs

- Medical background of the child, with reference to diseases and treatments

- Prevalence of secondary diseases. This refers to the presence of secondary infections such as TB and HIV and AIDS.

3.2.3.4 UNDERLYING FACTORS

For the purpose of this study the following have been classified as underlying factors contributing to malnutrition:

- Maternal and child care are determined by clinic attendance, with reference to the frequency with which the mother went for antenatal visits while she was

pregnant (once per month) and also the monthly visits to clinic where she took the child for growth monitoring and promotion (once per month for children zero to 24 months and once every three months for children 25 to 60 months). Maternal and childcare also includes possible diseases as well as treatments received.

- Nutritional information received by the mother/caregiver refers to the counselling received by mother/caregiver at health facilities regarding diarrhoea, healthy eating, breastfeeding, complementary feeding, food fortification, the growth chart and hygiene.

- Education level of the mother/caregiver refers to the highest grade finished in school or any tertiary education.

- Household background refers to household factors and socio-economic status of the household determined by the composition and amount of all persons living in the house or having a specific address as their place of residence

- Safe environment with reference to smoking habits of the mother/caregiver (seen as the number of cigarettes that the mother or caregiver smoke per day) and alcohol use of the mother/caregiver (seen as the number and size of alcoholic drinks that the mother or caregiver drinks per day).

3.2.3.5 BASIC FACTORS

For the purpose of this study basic factors contributing to malnutrition include the information about:

- Resources (income) and how they are controlled

3.2.4 STUDY PROCEDURES

- Permission to perform the study was obtained from the Ethics Committee of Kimberley Hospital Complex (Appendix I) and Northern Cape Department of Health (DoH) (Appendix J), as well as the Ethics Committee of the Faculty of Health Sciences, University of the Free State (ETOVS nr. 113/07).

- Information letters were sent to the Kimberley and Gordonia Hospitals (Appendix K and L) to inform them of the study.

- A pilot study was carried out in the Frances Baard District of the Northern Cape by the researcher (detail provided later in this chapter).

- The field workers (dieticians) working in the abovementioned hospitals were trained by the researcher on standardized methods to complete the questionnaire and obtain anthropometric measurements (Appendix M).

The field workers obtained consent for the main study from participants that complied with the inclusion criteria, in the participant's language of choice (Afrikaans, Tswana or English) (Appendixes F, G and H).

- The field workers completed a questionnaire (Appendix M) in an interview with each participant and were responsible for gathering the anthropometric measurements.

- The researcher was responsible for the coding of all the questionnaires.

- Data were analysed by the Department of Biostatistics of the University of the Free State.

3.3 TECHNIQUES

3.3.1 QUESTIONNAIRE (Appendix M)

A questionnaire (Appendix M) was designed by the DoH in the Northern Cape, and the researcher adapted the questionnaire for the study to determine the factors contributing to malnutrition.

All background, household, socio-economic and medical information was obtained from the mother or caregiver through a personal structured interview with the researcher or field workers (dieticians) at Kimberley Hospital Complex and Gordonia Hospital Complex. The nutritional information of the child was also gathered through the questionnaire. The RtHC was used to gather any other information needed on the questionnaire. The

majority of people spoke Afrikaans, but where a need for an interpreter was identified, health care workers assisted.

3.3.2 ANTHROPOMETRY (Appendix M)

All children as well as the mother or caregiver were measured. The following standardized measurements were included as recommended by Gibson (2005, p.245):

- Weight
- Height
- MUAC

3.3.2.1 Weight

Mother/ caregiver and older children: Weight was measured using a digital electronic scale, accurate to the nearest 0.1kg. All participants were weighed without shoes and with light clothing on and where possible before a meal and with an empty bladder. The scale was placed on a hard level surface; the mother/caregiver or child stood in the middle and kept still until the measurement was taken (Gibson, 2005, p.252).

Children below 24 months were weighed with an infant scale. The child was naked or wore minimal clothing. The child was placed on the scale so that the weight was distributed evenly. The measurement was taken to the nearest 10g while the child was lying still. If an infant scale was not available, the mother/caregiver and child were weighed together. The weight of the child was then subtracted from the mother/caregiver's weight to get the weight of the child. When weighing both mother/caregiver and child they wore a minimum amount of clothing. The minimum amount of clothing for children was seen as a nappy with underclothes or only one layer of lightweight clothes (Gibson, 2005, p.252). In most cases, the baby wore only a clean nappy. Three weight measures were taken and the average determined.

3.3.2.2 Height / Length

In adults (mother/ caregiver), height measurements were taken by means of a stadiometer, to the nearest 0.1cm. Participants were required to remove their shoes. They stood with heels touching the back of the height measure, legs straight, arms alongside the body, shoulders relaxed and looked straight ahead with their chin level with the ground (Gibson, 2005, p.235).

Length of children below 24 months was measured from the crown to the heel using a paediatric measuring board to the nearest 0.1cm. The measurement was only taken if the head was level with the headboard and the end of the measuring mat or board was against a flexed heal. The measurement was taken at eyelevel (Gibson, 2005, p.235). Three height / length measures were taken and the average determined.

3.3.2.3 MUAC

The researcher used a non- stretch measuring tape to the nearest 1 mm to measure MUAC. The child stood straight with the arms alongside the body. One arm was bent at the elbow. The distance of the upper arm between the point of the bent elbow and the knob at the top of the shoulder was measured. The middle point of this distance was calculated and a mark made on the skin of the upper arm. At this mark the circumference of the upper arm was measured. The measuring tape fitted tightly, but did not make a dent in the upper arm (Gibson, 2005, p.290). The arm was kept in a relaxed position along the side of the body.

3.4 VALIDITY AND RELIABILITY

Validity is defined as the degree to which an instrument measures what it is supposed to measure (Leedy and Ormrod, 2005, p.28). Reliability is ensured when findings generated are the same when the study is repeated under the same conditions (Bailey, 1997, p.71; Leedy and Ormrod, 2005, p.29).

Validity and reliability thus measure the extent to which there may be an error in measurements (Leedy and Ormrod, 2005, p.29).

3.4.1 QUESTIONNAIRE

Reliability was ensured as follows:

Ten percent of all questionnaires were repeated to make sure that information gathered was reliable. Using the same trained interviewers and interpreters at the same hospital also ensured reliability. According to Babby (2001) using interviewers yields an 80-85% higher response rate than when the questionnaires are filled in by the respondents.

For the reliability survey, 10% of the same respondents were contacted for a repeat of the questionnaires and the questionnaire was re-administered. Where the answer to

questions differed with more than 20%, the question was considered unreliable, and the results were not reported.

The researcher was the only person that coded the questionnaires and this also ensured reliability.

Validity was ensured as follows:
The questionnaire was based on published information related to the factors contributing to malnutrition, namely immediate, underlying and basic factors, as discussed in the literature overview (UNICEF, 2004a). All the questions in the questionnaire were designed according to the aims and objectives of the study.

3.4.2 ANTHROPOMETRY

Reliability was ensured as follows:
The researcher and dieticians used standardized techniques as recommended by Gibson (2005, p. 12). All fieldworkers that took anthropometric measurements were trained to ensure that these standardized techniques were used. Using high quality measuring equipment and calibrating the measuring equipment regularly ensured validity.

3.5 PILOT STUDY

The questionnaire was implemented in June 2007 in Hartswater, Jan Kempdorp and Warrenton Hospitals for the pilot study. Five severely malnourished children were included in the pilot study. The pilot study helped with the standardization process, seeing as an opportunity was created for the questionnaire to be tested and adapted for use in the main study and gave an indication of how long it took to complete.

The same questionnaire and measurements that will be used in the main study were piloted. The same inclusion criteria were used for the pilot study. A trained registered dietician filled in the questionnaires and the same procedure that will be used in the main study was followed for the pilot study.

On completion of the pilot study some of the questions in the questionnaire were revisited and some questions rephrased to guarantee that the interviewee gives the correct information. No real problems were experienced when the interviews were conducted. The interviews took about 30-45 minutes to conduct with the mother / caretaker.

The changed questions were as follows:

- Question 20.3 was changed from asking if the child is the oldest or the youngest to giving options in the question, such as first, second, third, fourth child or any other.
- Question 22 was divided into two questions, where in the first question the mother/caregiver was asked about the different types of income and the next question was about the total number of people receiving the different types of income.

3.6 STATISTICAL ANALYSIS

The Department of Biostatistics performed all analyses. Reliability analysis compared results obtained with the initial questionnaire and the reliability questionnaire (10% of sample) and where answers to questions differed in more than 20% of questionnaires, the question was considered unreliable and the results omitted.

Descriptive statistics, namely frequencies and percentages for categorical data and means and standard deviations or medians and percentiles for continuous data, were calculated. Associations will compare differences in parameters of the total percentage of children rather than comparing the differences in parameters of children with the different types of malnutrition. The reason for this is that the main objective of this study was to investigate factors associated with malnutrition in general (including all types of malnutrition) and not to determine differences in parameters between types of malnutrition.

The comparisons were done by means of 95% confidence intervals. A confidence interval is a range of plausible values that account for uncertainty in a statistical estimate or put in another way confidence intervals give a measure of the precision (or uncertainty) of study results for making inferences about the population of similar individuals. Confidence intervals combine information about the strength of an association with information about the effects of chance on the likelihood of obtaining the results. Confidence intervals place a clear emphasis on quantification of the effect, in direct contrast to the p-value approach (which arises from significance testing). Confidence intervals indicate the strength of the evidence about quantities that are directly relevant, such as treatment benefits (Gardner and Altman, 1989, pp. 6 - 19; Altman, 1991, pp. 174 – 175; Cohen, 1994; Ramey, 1999).

As mentioned by Altman et al. (2000) “We prefer the use of confidence intervals, which present the results directly on the scale of data measurement. The confidence interval provides a range of possibilities for the population value, rather than an arbitrary dichotomy based solely on statistical significance. It conveys more useful information at the expense of precision of the P value.”

3.7 ETHICAL ASPECTS

Approval of the research was obtained from the Ethics Committee of the Faculty of Health Sciences at the University of the Free State (ETOVS number 113/07). Approval of the research project by the Ethics Committee of Kimberley Hospital Complex (Appendix I) as well as the Department of Health (Appendix J) was also obtained prior to the study. The Hospital Managers of the Kimberley Hospital Complex and Gordonia Hospital Complex where informed about the written approval for the study to be performed in the two hospitals (Appendix K and L). This was done through a letter explaining the purpose and scope of the study.

Informed consent (Appendix F, G and H) was obtained from participants in the language of the mother / caregivers choice (Afrikaans, English and Tswana) during which procedures were explained to the mothers / caregivers in detail. At least one interpreter was trained per hospital.

According to the Children’s Act of 2005 (Act number 38, 2005) a “caregiver” is seen as any person other than a parent or guardian, who actually cares for a child and this includes:

- foster parents;
- a person who cares for the child with the implied or express consent of a parent or guardian;
- a person who cares for the child whilst in temporary safe care;
- a person at the head of a child care centre where a child has been placed;
- a person at the head of a shelter;
- child care worker who cares for a child who is without appropriate family care in the community; and
- the child at the head of a child-headed household.

Not all mothers / caregivers that were approached to participate in the study signed the consent form and some did refuse to participate.

Confidentiality of the information was maintained by ensuring that no names were made known or written in questionnaires. Coding was used in data analysis and results. . Interviews were done in a private setting to ensure confidentiality.

Participation was voluntary and respondents were given freedom to withdraw from the study at any time. The rights of the child and interviewee were respected if they refused to participate in the study – three caregivers/ mothers refused to take part in the study.

All actions that were undertaken in this study, regarding the child, are part of the normal service delivery actions that would be done under normal circumstances in the hospital setting. Children admitted to paediatric or childcare units were referred to a dietician to make sure that children were evaluated according to the inclusion criteria. All children partaking in the study were already being treated and thus did not have to be referred for management.

A final report will be made available to the Department of Health of the Northern Cape. The results of this study will give the Northern Cape Department of Health an indication whether they need to intensify their strategies for combating severe malnutrition.

CHAPTER 4: RESULTS

4.1 INTRODUCTION

Results regarding socio-demographic information, anthropometric information, household information, maternal information and medical history, maternal education, the child's medical history and biochemical information, infant feeding information and the FBDGs will be reported in this chapter. The associations between the abovementioned information are also included.

4.1.1 SOCIO-DEMOGRAPHIC INFORMATION

Table 4.1 Socio-demographic information

Variable	Category	Number	Percent
Town where interview was held (n = 54)	Kimberley	43	79.63
	Upington	11	20.37
Clinic attended by child (n = 54)	Masakhane, Kimberley	4	7.41
	Betty Gaetsewe, Kimberley	5	9.26
	Platfontein, Barkley Wes	2	3.70
	Ritchie	2	3.70
	Valspan, Jan Kempdorp	2	3.70
	Jan Kempdorp mobile clinic	1	1.85
	Greenpoint, Kimberley	1	1.85
	De Beershoogte, Barkly Wes	2	3.70
	Phutanang, Kimberley	3	5.56
	Galeshewe Day Hospital, Kimberley	1	1.85
	Winsorton	2	3.70
	Mataleng, Barkly Wes	1	1.85
	Griekwastad	2	3.70
	Bongani clinic, Douglas	1	1.85
	Taung clinic, North West	1	1.85
	Recreation, Kimberley	2	3.70
	Hartswater clinic	1	1.85
	Pampierstadt	1	1.85
	Keimoes clinic	2	3.70
	Brakpan, Douglas	1	1.85
	Ikhutseng, Warrenton	1	1.85
	Carnarvon	1	1.85
	Progress, Upington	1	1.85
	Topline, Groblershoop	2	3.70
	Sarah Strauss, Upington	2	3.70
	Lambrechtsdrift, Upington	1	1.85
	Karos, Upington	1	1.85
	Gordonia Hospital, Upington	2	3.70
	Colesberg	1	1.85
	Boshof	1	1.85
	Victoria-Wes	1	1.85
	Boichoko, Postmasburg	1	1.85
	Magagong, Taung, North West	1	1.85
	Wegdraai, Upington	1	1.85
Gender of child / baby (n = 54)	Male	32	59.26
	Female	22	40.74

Age of child / baby at interview (months) (n = 54)	0 - 6	7	12.96
	7 - 12	7	12.96
	13 - 24	30	55.56
	25 – 36	7	12.96
	> 37	3	5.56
Mother / caregiver's age (years) (n = 54)	19-25	16	29.63
	26-35	19	35.19
	36-45	12	22.22
	> 46	7	12.96
Mother / caregiver's education level (grade) (n = 54)	No school/education	6	11.11
	Grade 0-7	19	35.19
	Grade 8-12	28	51.85
	Tertiary education	1	1.85
Mother / caregiver's marital status (n = 54)	Single	44	81.48
	Married / traditional marriage	6	11.11
	Divorced	1	1.85
	Widowed	1	1.85
	Stays with the father of the child	1	1.85
	Stays with boyfriend	1	1.85

Of the 54 children included in this study 79.6% were admitted to Kimberley Hospital Complex and 20.4% were admitted to Gordonia Hospital Complex (Table 4.1). The majority of the children attended Betty Gaetsewe clinic (9.3%), Masakhane clinic (7.4%) and Phutanang clinic (5.6%) in Kimberley and in Upington the majority of the children (3.7%) attended Keimoes clinic, Topline clinic, Sarah Strauss clinic and Gordonia Hospital. Of the total number of children that participated in this study 59.3% were male and 40.7% were female. The children were of different ages, with the majority of the children between 13-24 months (55.6%). Mothers / caregivers were between 19 and 46 years of age. The highest percentage of mothers/caregivers (35.2%) was between 26-35 years of age. The education level of the mothers/caregivers ranged from no education at all to tertiary level education, with 51.9% of the mothers/caregivers having an educational level between grades 8 to 12. As far as marital status was concerned, 81.5% of mothers/ caregivers were single.

4.1.2 ANTHROPOMETRIC INFORMATION

Anthropometric information included birth weight, current weight, height and MUAC of the child as well as BMI of the mother/caregiver.

Table 4.2 Anthropometric information – weight and height/ length

Variable	Range	Median
Birth weight of child / baby (kg) (n = 40)	0.9 – 3.7	2.80
Current weight of child / baby (kg) (n = 54)	2.1 – 11.0	6.65
Height/ length of child / baby (cm) (n = 54)	46.0 – 95.0	72.75

The weight and height/ length of the children included in the study are shown in Table 4.2. The median birth weight was 2.80kg, median current weight was 6.65kg and median height was 72.75 cm.

Table 4.3 Anthropometric information – MUAC and BMI

Variable	Category	Number	Percent	Median
MUAC of child / baby (cm) (n = 54)				
< 11.0 cm	Severe malnutrition	21	38.89	11.55
11.1 – 12.5 cm	Moderate malnutrition (with or without			
> 12.5 cm	oedema)	15	27.78	
	Normal	18	33.33	
BMI of mother / caregiver (n = 54)				
<18,5 kg/m^2	Underweight	11	20.37	20.87
18,5 - 24.9 kg/m^2	Normal or healthy weight	30	55.56	
25- 29.9 kg/m^2	Overweight	4	7.41	
30-34.9 kg/m^2	Obese	5	9.26	
35-39.9 kg/m^2	Morbidly obese	0	0	
> 40 kg/m^2	Severely obese	4	7.41	

The median MUAC measurement of children in this study was 11.55 cm (Table 4.3). Of the 54 mothers/caregivers included in the study, 55.6% had a normal or healthy weight with a BMI of 18.5 to 24.9 kg/m^2. More than 7% (7.4%) were severely obese with a BMI of more than 40 kg/m^2.

4.1.3 HOUSEHOLD INFORMATION

The household information (table 4.4) included the size of the family and the numbers of rooms in the house as well as the income for the family and who the head of the household was.

Table 4.4 Household information

Variable	Category	Number	Percent
How many people depend on the income in the family (n = 54)	2	4	7.41
	3	6	11.11
	4	16	29.63
	5	13	24.07
	6	7	12.96
	7	2	3.70
	8	2	3.70
	9	1	1.85
	10	1	1.85
	11	2	3.70
Head of the household (n = 54)	Mother/caregiver's Boyfriend	1	1.85
	Mother/caregiver's brother	2	3.70
	Mother/caregiver's husband	3	5.56
	Child's father	9	16.67
	Child's grandfather	11	20.37
	Child's grandmother	11	20.37

	Child's mother	14	25.93
	Mother/caregiver's grandmother	1	1.85
	Child's Aunt	1	1.85
	Mother/caregiver's cousin	1	1.85
Room density (n = 54)			
≥ 2 – 5 persons / room	High	34	62.96
< 2 persons / room	Low	20	37.04

Out of the 54 households included in the study, 29.6% consisted of 4 family members dependant on the income in the household. Out of all the households 4 had 9 (1.8%), 10 (1.8%) and 11 (3.7%) members that depended on the income of the household. In a large percentage of the households, the child's mother was the head of the household (25.9%). However, in 22 of the households the child's grandfather (20.4%) and grandmother (20.4%) were the heads of the household. The room density of the majority of households was high (63%) (> 2-5 persons per room).

4.1.4 MATERNAL INFORMATION

The maternal information included in table 4.5 consisted mainly of information related to whom the child was staying with, if the mother was still alive, how many children were born to the mother, how many children had died, reasons for deaths, children admitted to hospital and reasons for admittance.

Table 4.5 Maternal information

Variable	Category	Number	Percent
Is the mother alive? (n = 54)	Yes	52	96.30
	No	2	3.70
Caregiver of child (mother is dead) (n = 2)	Child's grandmother	1	50.00
	Child's aunt	1	50.00
Whom is the child staying with most of the time (n = 54)	Parent/parents	40	74.07
	Grandparents / grandparent	2	14.81
	Aunt/uncle	1	1.85
	Other family	1	1.85
	Mother's sister in law	1	1.85
	Child's great grandmother	1	1.85
	Day Care Centre	1	1.85
	Mother/caregivers Aunt	1	1.85
Person looking after the child during the day (n = 54)	Child's mother	36	66.67
	Child's grandmother	15	27.78
	Neighbour	3	5.56
	Day Care Centre	1	1.85
	Other	9	16.67

Number of live births to the mother (n = 54)	1	20	37.04
	2	10	18.52
	3	14	25.93
	4	5	9.26
	5	5	9.26
Number of children from mother that died (n = 54)	None	48	88.89
	1	5	9.26
	2	1	1.85
Reasons for deaths of infants / children (n = 54)	No children dead	48	88.89
	Do not know	3	5.56
	Pneumonia	1	1.85
	Gastroenteritis	1	1.85
	Liver disease	1	1.85
Birth order of this child (n = 54)	1	21	38.89
	2	8	14.81
	3	13	24.07
	4	7	12.96
	Other: (5^{th} child)	5	9.26
Other children admitted to hospital (n = 54)	Only child	19	35.19
	Yes	18	33.33
	No	17	31.48
Reasons why other children were admitted to hospital (n = 18)	Flu	1	1.85
	Lung problems	2	3.70
	Child swollen	1	1.85
	Obstruction in throat	1	1.85
	Asthma	2	3.70
	Fever	1	1.85
	Gastroenteritis	3	5.56
	TB	2	3.70
	Accident	1	1.85
	Pneumonia	1	1.85
	Sores in mouth	1	1.85
	Malnutrition	1	1.85
	Ear infection	1	1.85
	Blood transfusion	1	1.85
	Liver disease	1	1.85

The majority of the mothers participating in the study were still alive (96.3%). The children of the two mothers that had died were cared for by the child's grandmother (50%) and child's aunt (50%). The children in the study were mostly living with their parents (74%). Of the 54 children included in the study, 66.7% were being cared for by their mother during the day. The person most likely to look after the child after the mother was the other children (brothers and sisters) and the child's aunt (16.7%).

A large percentage of mothers had had one (37%) or three (25.9%) live births. Most mothers (88.9%) had never lost a child. One (1.9%) mother had lost two children. The majority of the mothers with children who had died (5.6%) did not know what the reason for death was. In those that did know, reasons for death were pneumonia (1.9%), gastroenteritis (1.9%) and liver disease (1.9%). The birth order of the child participating in

this study was the first born in 38.9% of cases. A large percentage (35.2%) of the children in the study were an only child. In the remainder of the children, 33% of their brothers and sisters had been admitted to hospital at one time (5.7% were admitted for gastroenteritis)(table 4.5).

4.1.5 MATERNAL MEDICAL HISTORY

The maternal medical history included information related to voluntary counselling and testing (VCT), HIV status, TB status, other diseases, treatment received and pregnancy history (table 4.6).

Table 4.6 Maternal medical history

Variable	Category	Number	Percent	Median
Did the mother / caregiver had Voluntary Counselling and Treatment (n = 54)	Yes	38	70.37	
	No	16	29.63	
Mother/ caregiver's HIV status (n = 54)	Positive	18	33.33	
	Negative	23	42.59	
	Do not know	11	20.37	
	Does not want to reveal	2	3.70	
Mother / caregiver's TB status (n = 54)	Yes	12	22.22	
	No	42	77.78	
Other persons in household with TB (n = 12)	Child's father	1	8.33	
	Mother's brother	2	16.67	
	Child's grandmother	1	8.33	
	Grandmother's sister	1	8.33	
	Mother's mother	1	8.33	
	Mother's sister	1	8.33	
	Child's grandfather	1	8.33	
	Child's mother	3	25.00	
	Child's uncle	1	8.33	
Treatment received by mother / caregiver (n = 54)	HAART	6	11.11	
	PMTCT	5	9.26	
	TB	4	7.41	
	None	43	79.63	
	Other	0	0.00	
Any other diseases of mother / caregiver (n = 54)	Yes	2	3.70	
	No	52	96.30	
Type of diseases of mother / caregiver (n = 2)	Heart defect	1	50.00	
	HIV positive	1	50.00	
Pregnancy history:				
Ante-natal visits during pregnancy (n = 54)	Yes	47	87.04	
	No	4	7.41	
	Do not know	3	5.56	
Alcohol consumption during pregnancy (n = 54)	Yes	18	33.33	
	No	34	62.96	
	Do not know	2	3.70	

Amount of alcohol consumed per day (n = 18)	2 -10 drinks	9	50.00	10.50
	Not every day	1	5.56	
	Do not know	8	44.44	
Frequency of alcohol consumption per week (n = 18)	1 – 2 times	14	77.78	2.00
	Do not know	2	11.11	
	Twice per month	2	11.11	
Smoking during pregnancy (n = 54)	Yes	28	51.85	
	No	24	44.44	
	Do not know	2	3.70	

The majority of mothers/caregivers (70.4 %) had been for VCT. As shown in the table, 33.3% of the mothers/caregivers were HIV positive, 77.8% did not have TB and 3.7% had others diseases such as a heart defect (50%). Out of the 54 households that participated in the study, 2 (16.7%) of the other members in the household, which were the children's uncles, had TB. The majority of the mothers/caregivers (79.6%) taking part in the study were receiving no medical treatment at the time when the questionnaire was completed.

Table 4.6 shows that 87% of the mothers attended clinics for antenatal visits while pregnant with the child. The majority of the mothers (63%) reported that they did not consume any alcohol while pregnant with the child, but 33.3% did consume alcohol. Of the 33.3% of mothers consuming alcohol, 50% consumed 2-10 drinks per day with a median of 10.5 and only 1 (5.6%) did not consume alcohol every day. The majority of the mothers (77.8%) consumed alcohol 1-2 times per week with a median of 2.0. On the other hand, more mothers (51.9%) smoked or used snuff during their pregnancies, whereas 2 (3.7%) did not know because the caregiver answered the questions.

4.1.6 MEDICAL HISTORY OF THE CHILD

The medical history of children is described in table 4.7 and consists of the nutritional diagnosis, the prematurity of the child, gestational age, place of birth, availability of RtHC, if the chart was completed correctly, clinic attendance, reasons for clinic visits, NSP, immunizations and vitamin A supplementation status. Hospital admittance, how often and for what reason, HIV and TB status, treatment received and other diseases of the child were also determined.

Table 4.7 Child's medical history

Variable	Category	Number	Percent	Median
Nutritional diagnosis of child (n = 54)	Kwashiorkor	15	27.78	
	Marasmus	36	66.67	
	Marasmic kwashiorkor	3	5.56	
Prematurity of child (n = 54)	Yes	11	20.37	
	No	42	77.78	
	Do not know	1	1.85	
Weeks premature (n = 54)	≤ 30 weeks	5	50.00	31.00
	31-36 weeks	5	50.00	
Birth place of child (n = 54)	Hospital	48	88.89	
	Clinic	1	1.85	
	Community Health Centre	0	0.00	
	Home	3	5.56	
	Do not know	1	1.85	
	Street	1	1.85	
Road to Health Card of this child available (n = 54)	Yes	54	100.00	
	No	0	0.00	
Road to Health Card of this child completed correctly (n = 54)	Yes	24	44.44	
	No	14	25.93	
	Do not know	16	29.63	
Last clinic attendance with this child (weeks) (n = 54)	1-8	41	76.93	4.00
	9-20	6	11.11	
	24-32	5	9.26	
	>48	2	3.70	
For what did the mother / caregiver take the child to the clinic (n = 54)	Growth monitoring	24	44.44	
	Immunizations	31	57.41	
	Other	29	53.70	
Nutrition Supplementation Program (n = 54)	Yes	22	40.74	
	No	31	57.41	
	Do not know	1	1.85	
How long has the child been on the PEM scheme (months)	1-8	17	77.27	6.00
	≥ 9	5	22.73	
Child's immunizations up to date (n = 54)	Yes	30	55.56	
	No	20	37.04	
	Do not know	4	7.41	
Child's vitamin A supplementation up to date (n = 54)	Yes	19	35.19	
	No	27	50.00	
	Do not know	8	14.81	
Hospital admittance of this child (n = 54)	Yes	31	57.41	
	No	23	42.59	
How often were the child admitted (n = 54)	1	14	45.16	
	2	8	25.81	
	3	7	22.58	
	4	1	3.23	
	5	1	3.23	

Reason for child to be admitted to hospital (n = 31)	Gastroenteritis	19	61.29
	Flu	1	3.23
	Cerebral palsy	2	6.45
	Fits	2	6.45
	Underweight	2	6.45
	Vomiting	3	9.68
	Lung problems	3	9.68
	Cough	2	6.45
	Bleeding nose	1	3.23
	Fever	1	3.23
	Malnutrition	3	9.68
	Pneumonia	2	6.45
	Oral thrush	1	3.23
	Not eating	1	3.23
	Problems with colon	1	3.23
	Sores in mouth	1	3.23
	Heart defect	1	3.23
	Skin rash	1	3.23
	Child was swollen	1	3.23
Who referred the child to hospital (n = 54)	Nurse	24	44.44
	Doctor	22	40.74
	Dietician	1	1.85
	Other	14	25.93
Is child HIV positive (n = 54)	Yes	19	35.19
	No	22	40.74
	Do not know	13	24.07
Does child have TB (n = 54)	Yes	10	18.52
	No	38	70.37
	Do not know	6	11.11
Treatment received by child (n = 54)	HAART	7	12.96
	PMTCT	1	1.85
	TB	11	20.37
	None	38	70.37
	Other	2	3.70
Other diseases of the child (n = 54)	Yes	11	20.37
	No	43	79.63
What other diseases does the child have (n = 11)	Gastroenteritis	2	18.18
	Cerebral palsy	2	18.18
	Respiratory failure	1	9.09
	Gastrointestinal problems	1	9.09
	Oral thrush	1	9.09
	Pneumonia	1	9.09
	Malnutrition	2	18.18
	Enlarged liver	1	9.09
	Skin rash	1	9.09
	Heart defect	2	18.18
	Liver disease	1	9.09

Out of the 54 children that participated in the study, 66.7%, 27.8% and 5.6% were diagnosed with marasmus, kwashiorkor and marasmic kwashiorkor respectively. The majority of the children (77.8%) were not premature babies. Of the 20.4% that were premature, 50% were born before 30 weeks and 50% were born between 31 and 36 weeks. Forty-eight of the children (88.9%) were born in a hospital.

All of the children (100%) had Road to Health Charts, even though some of them were not available in the hospital (an inclusion criteria). Of the 54 children, 44.4% of the RtHCs were filled in correctly and 29.6% couldn't be evaluated because the charts were not available. The majority (76.9%) of the children attended the clinic within the last 1 to 8 weeks, prior to admittance to the hospital, with a median of 4.0 weeks. The children were mainly taken to a clinic for immunizations (57.4%) and growth monitoring (44.4%). As a result, 55.6% of children had up to date immunizations. Some of the other reasons for taking the child to the clinic included: vitamin A supplementation, gastroenteritis, vomiting, losing weight and not eating. Even though children were taken to the clinic for vitamin A supplementation, half of the children (50%) were behind on their vitamin A supplementations.

As shown in table 4.7, 57.4% of the children in the study were not on the NSP. Of the 40.7% that were currently on the NSP, 77.3% had been on the programme for the last one to eight months, with a median of 6.0 months.

The majority of the children (57.4%) had been previously admitted to hospital. Of the 31 that had been previously admitted, only 45.2% were admitted only once before. The main reasons for admittance to hospital were gastroenteritis (61.3%) followed by vomiting (9.7%), lung problems (9.7%) and malnutrition (9.7%). The nurse (44.4%) and the doctor (40.7%) were usually the people that referred the children to hospital. Except for the nurse and the doctor, the other people referring the child to hospital included the mother, the grandmother and the neighbour.

As shown in table 4.7, 40.7% of the children in the study were HIV negative, whereas 35.2% were HIV positive and 70.4% of the children did not have TB. The majority of the children (70.4%) were not receiving any kind of treatment at the time that the questionnaire was completed. Only one child was starting ARV treatment in the near future.

Except for HIV and TB, 20.4% of the children had other diseases such as gastroenteritis (18.1%), cerebral palsy (18.1%), malnutrition (18.1%) and a heart defect (18.1%).

4.1.7 BIOCHEMICAL INFORMATION

As shown in table 4.8, biochemical information of children included serum albumin, haemoglobin, C-reactive protein, absolute cluster of differentiation (CD4) count and CD4 percentage.

Table 4.8 Biochemical information of children (National Health Laboratory Services, 2009)

Variable	Category	Number	Percent	Min	Median	Max
Serum albumin (g/L)	< 32 Low	22	84.62	10.00	25.00	41.00
(n = 26)	32 – 47 Normal	4	15.38			
	> 47 High	0	0.00			
Haemoglobin (g/dL)	< 10 Low	23	52.27	5.10	9.55	14.30
(n = 44)	10 - 15 Normal	21	47.73			
	> 15 High	0	0.00			
C-reactive protein (mg/L)	0 – 10 Normal	10	29.41	5.00	47.00	310.00
(n = 34)	> 10 Infection (15 – 310)	24	70.59			
Absolute CD4 count	455	1	50.00	455.00	566.50	678.00
(mm^3) (n = 2)	678	1	50.00			
CD4 percentage (%)						
(n = 2)		1	50.00	15.00	23.00	31.00
< 12 months	15 – 24 Moderate	1	50.00			
1 – 5 years	> 25 No evidence					

The majority of the children (84.6%) had a low serum albumin with a median of 25.0 g/L. Of the 44 children who had available biochemical data for haemoglobin, 52.3% had low haemoglobin with a median of 9.55 g/dL. The majority of the children (70.6%) had a C-reactive protein count of more than 10mg/L, which is indicative of infection (median 47.0mg/L). Only two children had data available for the absolute CD4 count, where one had a count of 455mm^3 (50%) and the other a count of 678mm^3 (50%), with a median of 566.5mm^3. The CD4 percentage was only available for two of the children participating in the study; one child (50%) had a moderate (15-24%) CD4 percentage and one child had a CD4 percentage >25%.

4.1.8 MATERNAL EDUCATION

Maternal education consisted of education received by the mother at the clinic and to determine if the mother knew how to define diarrhea.

Table 4.9 Maternal education

Variable	Category	Number	Percent
Education received by mother at clinic (n = 53)	Diarrhoea	13	24.53
	Healthy eating	26	49.06
	Breastfeeding	33	62.26
	Complementary feeding	26	49.06
	Food fortification	8	15.09
	Growth Chart	18	33.96
	Hygiene	27	50.94
	None	2	3.77
Know what is diarrhoea (n = 54)	Yes	19	35.19
	No	35	64.81

The majority of the mothers/caregivers (62.3%) had received information/education on breastfeeding at the clinic. Some of the other topics discussed at the clinics, with mothers, were hygiene (50.9%), healthy eating (49.1%) and complementary feeding (49.1%). Of the 54 mothers/caregivers included, 64.8% had no idea how to define diarrhoea and did not know what it was.

4.1.9 INFANT FEEDING INFORMATION

The infant feeding information included if the child was ever breastfed, for how long the child had been breastfed, until when the child was exclusively breastfed, what other milk the child consumed, if the milk was sufficient for their age, if the milk was prepared hygienically, how the milk was fed to the child and when solids were introduced.

Table 4.10 Infant feeding information

Variable	Category	Number	Percent	Median
Child ever breastfed (n = 54)	Yes	48	88.89	
	No	5	9.26	
	Do not know	1	1.85	
To what age was child breastfed (months) (n = 49)	0-6	17	34.69	11.00
	7-12	13	26.53	
	13-18	10	20.41	
	19-24	3	6.12	
	> 25	3	6.12	
	Do not know	3	6.12	
How long was child exclusively breastfed (months) (n = 49)	Do not know	4	8.16	4.00
	0-2	13	26.53	
	3-4	16	32.65	
	5-6	13	26.53	
	7-9	2	4.08	
	>12	5	10.20	
Other milk drank by child (n = 45)	Formula milk	21	46.67	
	Cow's milk	5	11.11	
	Other	19	42.22	

Milk sufficient for age of child (n = 21)	Yes	1	4.76	
	No	19	90.48	
	Do not know	1	4.76	
Hygienic preparation of formula milk (n = 21)	Yes	16	76.19	
	No	4	19.05	
	Do not know	1	4.76	
How was milk fed to child (n = 29)	Bottle	25	86.21	
	Cup	2	6.90	
	Spoon	3	10.34	
Age when solids were introduction (months) (n = 49)	Do not know	4	8.16	6.00
	0-4	19	38.78	
	5-6	17	34.69	
	7-12	8	16.33	
	>13	1	2.04	

As shown in table 4.10, the majority of the children (88.9%) were breastfed at one stage in their lives. Out of the 54 children participating in the study, 34.7% that were breastfed were between the ages of 0-6 months, 26.5% were between the ages of 7-12 months and 20.4% were between the ages of 13-18 months. The median age of children that were breastfed was 11.0 months. Most of the children (32.65%) were exclusively breastfed for 3-4 months, with a median of 4.0 months. Only 13 (26.53%) of the children were breastfed for the recommended 5-6 months. If the child was not breastfed, they received mainly formula milk (46.7%) with 42.2% of the children receiving Nido, Nespray or no milk at all. The majority of the children (90.5%) did not receive enough milk according to their age at the time and only one child (4.8%) received enough milk. The adequacy of the milk given to the child was determined by the volume of water in relation to the number of scoops of formula milk en therefore the volume of prepared milk to the age of the child. In 76.2% of cases, the milk was prepared hygienically. Of all the children in the study, 86.2% received their milk in a bottle and only two (6.9%) were cup fed. The majority of the children (73.5%) were started on solid foods at the age of 0-6 months, with a median of 6.0 months. One child (2%) received solids for the first time only after 13 months.

4.1.10 FOOD BASED DIETARY GUIDELINES

Information related to the food based dietary guidelines is included in Table 4.11.

Table 4.11 Food Based Dietary Guidelines

Variable	Category	Number	Percent	Median
Other food added to porridge (n = 46)	Meat	37	80.43	
	Margarine or oil	40	86.96	
	Milk	32	69.59	
	Sugar	38	82.61	
	Other	8	17.39	
Child eat meat, fish, chicken, eggs or milk each day (n = 46)	Yes	25	54.35	
	No	21	45.65	
Frequency per week (n = 24)	Once per week	16	66.67	7.00
	Twice per week	4	16.67	
	Three times per week	1	4.17	
	8 Times per week	1	4.17	
	13 Times per week	2	8.33	
Child eat soy mince and baked beans in tomato sauce (n = 46)	Yes	36	78.26	
	No	10	21.74	
Glasses or bottles of water (number) (n = 48)	0	2	4.17	2.00
	1	11	22.92	
	2	15	31.25	
	3	7	14.58	
	4	8	16.67	
	5	1	2.08	
	> 5	3	6.25	
	Do not know	1	2.08	
Glasses or bottles of tea (number) (n = 47)	0	10	21.28	2.00
	1	12	25.53	
	2	15	31.91	
	3	6	12.77	
	4	2	4.26	
	5	1	2.13	
	6	1	2.13	
Type of bread bought (n = 46)	White	17	36.96	
	Brown	17	36.96	
	Combination	10	21.74	
	Other: No bread	2	4.35	
Child eat fruit each day (n = 46)	Yes	9	19.57	
	No	37	80.43	
Child eat skins of fruit (n = 46)	Yes	11	23.91	
	No	35	76.09	
Child eat vegetables each day (n = 46)	Yes	17	36.96	
	No	29	63.04	
Items added to food with preparation (n = 45)	Salt	44	97.78	
	Aromat	8	17.78	
	Beef stock blocks	36	80.00	
	Steak ‘n chop spice	29	64.44	
	Chicken spice	37	82.22	
	Soup powder	29	64.44	
	Other	7	15.56	
Items used with preparation of food (n = 45)	Margarine	31	68.89	
	Oil	42	93.33	
	Animal fat	30	66.67	

	None	45	100.00
	Other: Peanut butter	1	2.22
Child eats sugar each day (n = 46)	Yes	39	84.78
	No	7	15.22
Kind of sweets and cool drinks eaten / drank each day (n = 45)	Sweets	40	88.89
	Chocolates	27	60.00
	Coke, fanta, etc.	36	80.00
	Cordials (oros)	32	71.11
	Biscuits	40	88.89
	Cakes, doughnuts, etc	34	75.56
Child plays outside (n = 48)	Yes	33	68.75
	No	15	31.25

The majority of the mothers/caregivers added margarine/oil (87%), sugar (82.6%), meat (80.4%) and milk (70%) to their children's porridge. Some of the other items (17.4%) that were added to children's porridge were formula milk, Purity, peanut butter, juice, yoghurt and chips. Out of all the children already receiving solid food, 54.4% ate meat, fish, chicken, eggs or milk each day. Even though more than half of the children ate animal proteins each day, 66.7% ate these items only once per week, with a median of 7.0 times per week (once per day). The majority of the children (78.3%) sometimes ate soy mince and baked beans in tomato sauce.

Out of all the children participating in the study, 31.3 percent drank two bottles/cups of water per day, with a median of 2.0 cups per day and 31.9% of the children drank two bottles/cups of tea per day, with a median of 2.0 cups per day. A large percentage of the mothers/caregivers (37%) buy both white and brown bread for their children.

As seen in table 4.11, 80.4% of the children did not eat fruit each day and 76.1% did not eat the skins of the fruit. The majority of the children (63 %) did not eat vegetables each day. Out of all the children in the study, 97.8% of the mothers/caregivers added salt to the child's food during preparation. They also added chicken spice (82.2%), beef stock cubes (80%), steak and chop spice and soup powder (64.4%). Some of the other items (15.6%) added during food preparation included mixed masala/ curry, oil, peri-peri spice, and pepper.

A total of 84.8% of the children participating in the study consumed sugar every day. The intake of sweets and cool drinks per day were very high with 88.9% consuming sweets and biscuits, 80% consuming Coke, Fanta and other carbonated cool drinks, 75.6%

consuming cakes, doughnuts, etc., 71.1% consuming cordials such as Oros and 60% of the children consuming chocolates.

The majority of the children (68.8%) played outside.

4.2 ASSOCIATIONS BETWEEN VARIABLES

Associations between variables are reported in the following section.

4.2.1 Nutritional diagnosis and gender

Table 4.12 Nutritional diagnosis and gender

Nutritional diagnosis	Gender (n=54)				95 % CI for the difference (diff) between male and female
	Male		Female		
	Number (N)	%	N	%	
Kwashiorkor (n=15)	11	73.33	4	26.67	
Marasmus (n=36)	19	52.78	17	47.22	
Marasmic kwashiorkor (n=3)	2	66.67	1	33.33	
TOTAL	32	59.26	22	40.74	[0.46:0.71]

A significantly higher occurrence of malnutrition in males than in females occurred with a 95% confidence interval (CI) [0.46:0.71] for the percentage difference (Table 4.12).

4.2.2 Nutritional diagnosis and Nutrition Supplementation Programme

Table 4.13 Nutritional diagnosis and NSP

Nutritional diagnosis	NSP (n=54)						95 % CI for the diff between NSP or not
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	6	40.00	9	60.00	0	0.00	
Marasmus (n=36)	13	36.11	22	61.11	1	2.78	
Marasmic kwashiorkor (n=3)	3	100.00	0	0.00	0	0.00	
TOTAL	22	40.74	31	57.41	1	1.85	[0.29:0.55]

Significantly more children (total) were not on the NSP (57.41%) compared to those that had received food aid (40.74%) with a 95% CI [0.29:0.55] for the percentage difference (Table 4.13).

4.2.3 Nutritional diagnosis and completion of Road to Health Card

Table 4.14 Nutritional diagnosis and completion of RtHC

Nutritional diagnosis	Road to Health Card completion (n=54)						95 % CI for the diff between complete RtHC and incomplete card
	Yes		No		Not available		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	7	46.67	6	40.00	2	13.33	
Marasmus (n=36)	16	44.44	8	22.22	12	33.33	
Marasmic kwashiorkor (n=3)	1	33.33	0	0.00	2	66.67	
TOTAL	24	44.44	14	29.93	16	29.63	[0.47:0.77]

Of the malnourished children included in this study, significantly more (44.44%) had a completed RtHC compared to those whose RtHCs were incomplete (29.93%) with a 95% CI [0.47:0.77] for the percentage difference (Table 4.14).

4.2.4 Nutritional diagnosis and last clinic visit

Table 4.15 Nutritional diagnosis and last clinic visit

Variable	Minimum	Median	Maximum	95 % CI for the median diff between kwashiorkor and marasmus and the last clinic visit
Nutritional diagnosis n=54	1.000	4.000	52.000	[-4:2]
Kwashiorkor (n=15)	1.000	4.000	48.000	
Marasmus (n=36)	1.000	4.000	52.000	
Marasmic kwashiorkor (n=3)	1.000	2.000	4.000	

No significant difference between the median number of weeks since the last clinic visit of children with kwashiorkor and marasmus was found (95% CI for median difference [-4:2]), with both having visited the clinic a median of 4 weeks ago (Table 4.15).

4.2.5 Nutritional diagnosis and immunizations up to date

Table 4.16 Nutritional diagnosis and immunizations up to date

Nutritional diagnosis	Immunizations up to date (n=54)						95 % CI for the diff between immunizations up to date or not
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	7	46.67	7	46.67	1	6.67	
Marasmus (n=36)	21	58.33	13	36.11	2	5.56	
Marasmic kwashiorkor (n=3)	2	66.67	0	0.00	1	33.33	
TOTAL	30	55.56	20	37.04	4	7.40	[0.46:0.72]

Significantly more children (55.56%) had immunizations that were up to date compared to those whose immunizations were behind (37.04%) with a 95% CI of [0.46:0.72] for the percentage difference (Table 4.16).

4.2.6 Nutritional diagnosis and Vitamin A supplementation up to date

Table 4.17 Nutritional diagnosis and vitamin A supplementation up to date

Nutritional diagnosis	Vitamin A supplementation up to date (n=54)						95 % CI for the diff between vitamin A supplementation up to date or not
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	5	33.33	8	53.33	2	13.33	
Marasmus (n=36)	13	36.11	18	50.00	5	13.89	
Marasmic kwashiorkor (n=3)	1	33.33	1	33.33	1	33.33	
TOTAL	19	35.19	27	50.00	8	14.81	[0.28:0.57]

Significantly more malnourished children (50.00%) had vitamin A supplementations that were behind than those with up to date vitamin A supplementations (35.19%) with a 95% CI of [0.28:0.57] for the percentage difference (Table 4.17).

4.2.7 Nutritional diagnosis and breastfeeding

Table 4.18 Nutritional diagnosis and breastfeeding

Nutritional diagnosis	Breastfeeding (n=54)						95 % CI for the diff between being breastfed or not
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	14	93.33	1	6.67	0	0.00	
Marasmus (n=36)	31	86.11	4	11.11	1	2.78	
Marasmic kwashiorkor (n=3)	3	100.00	0	0.00	0	0.00	
TOTAL	48	88.89	5	9.26	1	1.85	[0.80:0.96]

Of the malnourished children in this study significantly more children (88.89%) were breastfed at one stage compared with those that were never breastfed at all (9.26%) with a 95% CI of [0.80:0.96] for the percentage difference (Table 4.18).

4.2.8 Nutritional diagnosis and age when breastfeeding was stopped

Table 4.19 Nutritional diagnosis and age when breastfeeding was stopped

Variable	Minimum	Median	Maximum	95 % CI for the median diff between kwashiorkor and marasmus and the time when breastfeeding was stopped
Nutritional diagnosis n=46	1.000	11.000	37.000	[-8:2]
Kwashiorkor n=14	2.000	9.000	37.000	
Marasmus n=32	1.000	11.500	37.000	
Marasmic kwashiorkor n=3	1.000	2.000	13.000	

No significant median difference was found between kwashiorkor and marasmus and the median age when breastfeeding was reported to be stopped (95% CI [-8:2] for the median difference in age), with children diagnosed with kwashiorkor being breastfed a median of 9 months and marasmic children a median of 11.50 months (Table 4.19).

4.2.9 Nutritional diagnosis and exclusive breastfeeding stopped

Table 4.20 Nutritional diagnosis and exclusive breastfeeding stopped

Variable	Minimum	Median	Maximum	95 % CI for the median diff between kwashiorkor and marasmus and length of exclusive breastfeeding
Nutritional diagnosis n=49	1.000	4.000	13.000	[-2:1]
Kwashiorkor n=14	2.000	3.000	13.000	
Marasmus n=32	1.000	4.000	13.000	
Marasmic kwashiorkor n=3	1.000	2.000	5.000	

No significant median difference was found between kwashiorkor and marasmus and the median age when exclusive breastfeeding was reportedly stopped (95% CI [-2:1] for the median difference in age). Children diagnosed with kwashiorkor were exclusively breastfed for a median of 3 months and marasmic children for a median of 4 months (Table 4.20).

4.2.10 Nutritional diagnosis and other milk consumed

Table 4.21 Nutritional diagnosis and other milk consumed

Nutritional diagnosis	Milk (n=45)					
	Formula milk		Cow's milk		Other	
	N	%	N	%	N	%
Kwashiorkor (n=14)	5	33.33	3	20.00	7	46.67
Marasmus (n=28)	14	50.00	2	7.14	12	42.86
Marasmic kwashiorkor (n=2)	2	100.00	0	0.00	0	0.00
TOTAL	21	46.67	5	11.11	19	42.22

Of the malnourished children in this study, 46.67% consumed formula milk, 11.11% cow's milk and 42.22% other types of milk when they were not breastfed. Significantly more malnourished children consumed formula milk (46.67%) than cow's milk (11.11%) and other milk (42.22%) with a 95% CI of [0.62:0.92] and [0.38:0.67] for the percentage difference respectively. Significantly less malnourished children consumed cow's milk (11.11%) than other milk (42.22%) with a 95% CI [0.09:0.41] for the percentage difference (Table 4.21).

No significant difference was found between children diagnosed with kwashiorkor and marasmus and the consumption of cow's milk with a 95% CI [-7.34:38.57] for the percentage difference. Even though the difference is insignificant, it does seem to indicate a trend.

4.2.11 Nutritional diagnosis and adequacy of milk for age

Table 4.22 Nutritional diagnosis and adequacy of milk for age

Nutritional diagnosis	Milk (n=21)						95 % CI for the diff between sufficient milk and insufficient milk
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=5)	0	0.00	4	80.00	1	20.00	
Marasmus (n=14)	1	7.14	13	92.86	0	0.00	
Marasmic kwashiorkor (n=2)	0	0.00	2	100.00	0	0.00	
TOTAL	1	4.76	19	90.48	1	4.76	[0.01:0.24]

As expected, significantly more malnourished children received insufficient quantities of formula milk for their age (90.48%) than those that received sufficient quantities of formula milk for age (4.76%) with a 95% CI [0.1:0.24] for the percentage difference (Table 4.22).

4.2.12 Nutritional diagnosis and initiation of solid foods

Table 4.23 Nutritional diagnosis and initiation of solid foods

Variable	Minimum	Median	Maximum	95 % CI for the median diff between diagnosis and initiation of solids
Nutritional diagnosis n=49	2.000	6.000	20.000	[-1:3]
Kwashiorkor n=15	2.000	6.000	20.000	
Marasmus n=32	2.000	5.500	20.000	
Marasmic kwashiorkor n=2	5.000	5.500	6.000	

No significant difference was found between children with kwashiorkor and marasmus and the median age at which solids were introduced (95% CI [-1:3] for the median age difference), with children diagnosed with kwashiorkor receiving solids at a median age of 6 months and marasmic children receiving solids at a median age of 5.5 months (Table 4.23).

4.2.13 Nutritional diagnosis and food based dietary guidelines

Table 4.24 Nutritional diagnosis and food based dietary guidelines

Categories	Yes		No		95 % CI for the diff between diagnosis and the food based dietary guidelines
	N	%	N	%	
Meat, chicken, fish, eggs and milk (n=46)	25	54.35	21	45.65	[0.40:0.68]
Baked beans and soy mince (n=46)	36	78.26	10	21.74	[0.64:0.88]
Vegetables (n=46)	17	36.96	29	63.04	[0.25:0.51]
Fruit (n=46)	9	19.57	37	80.43	[0.11:0.33]
Sugar (n=46)	39	84.78	7	15.22	[0.72:0.92]
Sweets (n=45)	40	88.89	5	11.11	[0.77:0.95]
Chocolates (n=45)	27	60.00	18	40.00	[0.46:0.73]
Coke, fanta, carbonated drinks (n=45)	36	80.00	9	20.00	[0.66:0.89]
Cordials (n=45)	32	71.11	13	28.89	[0.57:0.82]
Biscuits (n=45)	40	88.89	5	11.11	[0.77:0.95]
Cake and doughnuts (n=45)	34	75.56	11	24.44	[0.61:0.86]

Comparisons were made between the malnourished children in this study and the reported intake of the various foods according to the FBDGs. A large percentage of children did not consume sufficient amounts of meat, chicken, fish, eggs and milk (45.65%), baked beans and soy mince (21.74%), vegetables (63,04%) and fruit (80.43%). However, intake of less healthy foods was reported to be high, with 84.78% of malnourished children eating sugar, 88.89% eating sweets, 60% eating chocolates. 80% drinking carbonated drinks and 88.89 eating biscuits on a daily basis. Significantly more malnourished children consumed unhealthy foods such as, sugar (84.78%) (95% CI [0.72:0.92] for the percentage difference), sweets (88.89%) (95% CI [0.77:0.95] for the percentage difference), chocolates (60.00%) (95% CI [0.46:0.73] for the percentage difference), carbonated drinks (80.00%) (95% CI [0.66:0.89] for the percentage difference), cordials (71.11%) (95% CI [0.57:0.82] for the percentage difference), biscuits (88.89%) (95% CI [0.77:0.95] for the percentage difference), cakes and doughnuts (75.56%) (95% CI [0.61:0.86] for the percentage difference). Significantly fewer malnourished children consumed vegetables (63.04%) and fruit (19.57%) with a 95% CI of [0.25:0.51] and [0.11:0.33] for the percentage difference respectively (Table 4.24).

4.2.13.1 Unhealthy food intake in association with food based dietary guidelines

Table 4.24.1 Unhealthy foods and meat, chicken, fish, eggs and milk intake

Variable	Categories	Meat, chicken, fish, eggs and milk (n=45)				95 % CI for the diff between the intake of unhealthy foods and meat, chicken, fish, eggs and milk intake
		Yes		No		
		N	%	N	%	
Sweets	Yes	22	55.00	18	45.00	[0.40:0.69]
	No	3	60.00	2	40.00	
Chocolates	Yes	19	70.37	8	29.63	[0.52:0.84]
	No	6	33.33	12	66.67	
Coke	Yes	17	47.22	19	52.78	[0.32:0.63]
	No	8	88.89	1	11.11	
Cordials	Yes	16	50.00	16	50.00	[0.34:0.66]
	No	9	69.23	4	30.77	
Biscuits	Yes	23	57.50	17	42.50	[0.42:0.72]
	No	2	40.00	3	60.00	
Cake and doughnuts	Yes	20	58.82	14	41.18	[0.42:0.74]
	No	5	45.45	6	54.55	

Reported intakes showed that children who ate unhealthy foods such as sweets, chocolates, biscuits and cake and doughnuts were also more likely to eat meat, chicken, fish, eggs and milk than children who did not eat unhealthy foods. Significantly more malnourished children who consumed meat, chicken, fish, eggs and milk also ate unhealthy foods such as, chocolates (70.37%) (95% CI [0.52:0.84] for the percentage difference), biscuits (57.50%) (95% CI [0.42:0.72] for the percentage difference), cake and doughnuts (58.82%) (95% CI [0.42:0.74] for the percentage difference). The same was not, however, true for the intake of carbonated drinks such as Coke (Table 4.24.1).

Table 4.24.2 Unhealthy foods and baked beans and soy mince intake

Variable	Categories	Baked beans and soy mince (n=45)				95 % CI for the diff between the intake of unhealthy foods and baked beans and soy mince intake
		Yes		No		
		N	%	N	%	
Sweets	Yes	32	80.00	8	20.00	[0.65:0.90]
	No	4	80.00	1	20.00	
Chocolates	Yes	23	85.19	4	14.81	[0.68:0.94]
	No	13	72.22	5	27.78	
Coke	Yes	30	83.33	6	16.67	[0.68:0.92]
	No	6	66.67	3	33.33	
Cordials	Yes	26	81.25	6	18.75	[0.65:0.91]
	No	10	76.92	3	23.08	
Biscuits	Yes	32	80.00	8	20.00	[0.65:0.90]
	No	4	80.00	1	20.00	
Cake and doughnuts	Yes	29	85.29	5	14.71	[0.70:0.94]
	No	7	63.64	4	36.36	

Significantly more malnourished children who ate baked beans and soy mince were also more likely to eat unhealthy foods such as sweets (80.00%) (95% CI [0.65:0.90] for the percentage difference), chocolates (85.19%) (95% CI [0.68:0.94] for the percentage difference), Coke (83.33%) (95% CI [0.68:0.92] for the percentage difference), cordials (81.25%) (95% CI [0.65:0.91] for the percentage difference), biscuits (80.00%) (95% CI [0.65:0.90] for the percentage difference) and cake and doughnuts (85.29%) (95% CI [0.70:0.94] for the percentage difference) (Table 4.24.2).

Table 4.24.3 Unhealthy foods and vegetable intake

Variable	Categories	Vegetables (n=45)				95 % CI for the diff between the intake of unhealthy foods and vegetable intake
		Yes		No		
		N	%	N	%	
Sweets	Yes	14	35.00	26	65.00	[0.22:0.51]
	No	3	60.00	2	40.00	
Chocolates	Yes	11	40.74	16	59.26	[0.25:0.59]
	No	6	33.33	12	66.67	
Coke	Yes	10	27.78	26	72.22	[0.16:0.44]
	No	7	77.78	2	22.22	
Cordials	Yes	14	43.75	18	56.25	[0.28:0.61]
	No	3	23.08	10	76.92	
Biscuits	Yes	15	37.50	25	62.50	[0.24:0.53]
	No	2	40.00	3	60.00	
Cake and doughnuts	Yes	12	35.29	22	64.71	[0.22:0.52]
	No	5	45.45	6	54.55	

Of all the malnourished children in this study significantly fewer children ate vegetables when they consumed sweets (35.00%) (95% CI [0.22:0.51] for the percentage difference), chocolates (40.74%) (95% CI [0.25:0.59] for the percentage difference), Coke (27.78%)

(95% CI [0.16:0.44] for the percentage difference), cordials (43.75%) (95% CI [0.28:0.61] for the percentage difference), biscuits (37.50%) (95% CI [0.24:0.53] for the percentage difference) and cake and doughnuts (35.29%) (95% CI [0.22:0.52] for the percentage difference) (Table 4.24.3).

Table 4.24.4 Unhealthy foods and fruit intake

Vaiable	Categories	Fruit (n=45)				95 % CI for the diff between the intake of unhealthy foods and fruit intake
		Yes		No		
		N	%	N	%	
Sweets	Yes	6	15.00	34	85.00	[0.07:0.29]
	No	3	60.00	2	40.00	
Chocolates	Yes	6	22.22	21	77.78	[0.11:0.41]
	No	3	16.67	15	83.33	
Coke	Yes	7	19.44	29	80.56	[0.10:0.35]
	No	2	22.22	7	77.78	
Cordials	Yes	8	25.00	24	75.00	[0.13:0.42]
	No	1	7.69	12	92.31	
Biscuits	Yes	9	22.50	31	77.50	[0.12:0.38]
	No	0	0.00	5	100.00	
Cake and doughnuts	Yes	7	20.59	27	79.41	[0.10:0.37]
	No	2	18.18	9	81.82	

As found with vegetable intake, significantly fewer malnourished children ate fruit if they consumed sweets (15.00%) (95% CI [0.07:0.29] for the percentage difference), chocolates (22.22%) (95% CI [0.11:0.41] for the percentage difference), coke (19.44%) (95% CI [0.10:0.35] for the percentage difference), cordials (25.00%) (95% CI [0.13:0.42] for the percentage difference), biscuits (22.50%) (95% CI [0.12:0.38] for the percentage difference), and cake and doughnuts (20.59%) (95% CI [0.10:0.37] for the percentage difference) (Table 4.24.4).

4.2.14 Nutritional diagnosis in association with hospital admittance

Table 4.25 Nutritional diagnosis in association with hospital admittance

Nutritional diagnosis	Hospital admittance (n=54)				95 % CI for the diff between diagnosis and hospital admittance
	Yes		No		
	N	%	N	%	
Kwashiorkor (n=15)	10	66.67	5	33.33	
Marasmus (n=36)	20	55.56	16	44.44	
Marasmic kwashiorkor (n=3)	1	33.33	2	66.67	
TOTAL	31	57.41	23	42.59	[0.44:0.70]

Significantly more malnourished children had been admitted to hospital on previous occasions (57.41%) compared to those that had not previously been admitted to hospital (42.59%) with a 95% CI [0.44:0.70] for the percentage difference (Table 4.25).

4.2.15 Admittance and reason for admittance

Table 4.26 Admittance and reason for admittance

Variable	Gastroenteritis		
	Minimum	Median	Maximum
Kwashiorkor (n=10)	1.000	1.000	5.000
Marasmus (n=20)	1.000	2.000	4.000
Marasmic kwashiorkor (n=1)	2.000	2.000	2.000

A close to significant median difference was found between kwashiorkor and marasmus and the number of previous hospitalizations for gastroenteritis (95% CI [-1:0] for the median difference). Children with kwashiorkor were previously admitted to hospital for gastro with a median of 1 time compared to children with marasmus who had been admitted to hospital for gastro a median of two times (Table 4.26).

4.2.16 Education level of mother/caregiver in association with food intake

Table 4.27 Education of mother/caregiver in association with food intake

Variable	Categories	Yes (n=46)		No (n=46)		95 % CI for the diff between educational level and FBDG's
		N	%	N	%	
Grade ≤ 7	Meat, chicken, fish, eggs and milk	11	52.38	10	47.62	[0.32:0.72]
	Baked beans and soy mince	18	85.71	3	14.29	[0.65:0.95]
	Vegetables	7	33.33	14	66.67	[0.17:0.55]
	Fruit	4	19.05	17	80.95	[0.08:0.40]
	Sugar	18	85.71	3	14.29	[0.65:0.95]
Grade ≥ 8	Meat, chicken, fish, eggs and milk	14	56.00	11	44.00	[0.37:0.73]
	Baked beans and soy mince	18	72.00	7	28.00	[0.52:0.86]
	Vegetables	10	40.00	15	60.00	[0.23:0.59]
	Fruit	5	20.00	20	80.00	[0.09:0.39]
	Sugar	21	84.00	4	16.00	[0.65:0.94]

When the caregiver had an education level of grade 7 or below or grade 8 and above, malnourished children received significantly more meat, chicken, fish, eggs and milk (52.38% to 56.00%) (95% CI [0.32:0.72] to [0.37:0.73] for the percentage difference), baked beans and soy mince (85.71% to 72.00%) (95% CI [0.65:0.95] to [0.52:0.86] for the percentage difference) and sugary foods (85.71% to 84.00%) (95% CI [0.65:0.95] to

[0.65:0.94] for the percentage difference), respectively. They also received significantly less vegetables (33.33% to 40.00%) (95% CI [0.17:0.55] to [0.23:0.59] for the percentage difference) and fruit (19.05% to 20.00%) (95% CI [0.08:0.40] to [0.09:0.39] for the percentage difference), respectively (Table 4.27).

4.2.17 Nutritional diagnosis in association with number of children (births)

Table 4.28 Nutritional diagnosis in association with number of children (births)

Variable	Number of births		
	Minimum	Median	Maximum
Kwashiorkor (n=15)	1.000	3.000	5.000
Marasmus (n=36)	1.000	2.000	5.000
Marasmic kwashiorkor (n=3)	2.000	2.000	3.000

No significant median difference was found between kwashiorkor and marasmus and the median number of births of the mother (95% CI [-1:1] for the median number of births). In families with children diagnosed with kwashiorkor there was a median of 3 births and with marasmic children a median of 2 births (Table 4.28).

4.2.18 Caretaker during the day in association with food intake

Table 4.29 Caretaker during the day in association with food intake

Variable	Categories		Yes (n=46)		No (n=46)	
			N	%	N	%
Mother	Meat, chicken, fish, eggs and milk	Yes	17	56.67	13	43.33
		No	8	50.00	8	50.00
	Baked beans and soy mince	Yes	22	73.33	8	26.67
		No	14	87.50	2	12.50
	Vegetables	Yes	9	30.00	21	70.00
		No	8	50.00	8	50.00
	Fruit	Yes	5	16.67	25	83.33
		No	4	25.00	12	75.00
	Sugar	Yes	23	76.67	7	23.33
		No	16	100.00	0	0.00
Grandmother	Meat, chicken, fish, eggs and milk	Yes	7	53.85	6	46.15
		No	18	54.55	15	45.45
	Baked beans and soy mince	Yes	8	61.54	5	38.46
		No	28	84.85	5	15.15
	Vegetables	Yes	6	46.15	7	53.85
		No	11	33.33	22	66.67
	Fruit	Yes	4	30.77	9	69.23
		No	5	15.15	28	84.85
	Sugar	Yes	11	84.62	2	15.38
		No	28	84.85	5	15.15

Neighbour	Meat, chicken, fish, eggs and milk	Yes	3	100.00	0	0.00
		No	22	51.16	21	48.84
	Baked beans and soy mince	Yes	3	100.00	0	0.00
		No	33	76.74	10	23.26
	Vegetables	Yes	3	100.00	0	0.00
		No	14	32.56	29	67.44
	Fruit	Yes	1	33.33	2	66.67
		No	8	18.60	35	81.40
	Sugar	Yes	3	100.00	0	0.0
		No	36	83.72	7	16.28
Day Care	Meat, chicken, fish, eggs and milk	Yes	1	100.00	0	0.00
		No	24	53.33	21	46.67
	Baked beans and soy mince	Yes	1	100.00	0	0.00
		No	35	77.78	10	22.22
	Vegetables	Yes	0	0.00	1	100.00
		No	17	37.78	28	62.22
	Fruit	Yes	0	0.00	1	100.00
		No	9	20.00	36	80.00
	Sugar	Yes	1	100.00	0	0.00
		No	38	84.44	7	15.56
Other	Meat, chicken, fish, eggs and milk	Yes	4	50.00	4	50.00
		No	21	55.26	17	44.74
	Baked beans and soy mince	Yes	7	87.50	1	12.50
		No	29	76.32	9	23.68
	Vegetables	Yes	5	62.50	3	37.50
		No	12	31.58	26	68.42
	Fruit	Yes	1	12.50	7	87.50
		No	8	21.05	30	78.95
	Sugar	Yes	8	100.00	0	0.00
		No	31	81.58	7	18.42

The children that were looked after by their mothers during the day received significantly more sugar (76.67%) with a 95% CI [-40.93: -0.79]. When the grandmother looked after the child during the day there seemed to be a greater chance of the children eating less vegetables (46.15%) (95% CI [-51.71:9.95] for the percentage difference) and fruit (95% CI [-49.52:8.87] for the percentage difference) (Table 4.29), but differences were only close to significant.

4.2.19 Nutritional diagnosis in association with household / room density

Table 4.30 Nutritional diagnosis in association with household/room density

Variable	Number of births		
	Minimum	Median	Maximum
Kwashiorkor (n=15)	1.333	2.500	5.000
Marasmus (n=36)	1.000	2.838	5.000
Marasmic kwashiorkor (n=3)	1.333	1.667	4.000

A close to significant median difference was found between the household room density of children with marasmus and kwashiorkor (95% CI [0:1] for the median difference) (Table 4.30), with children diagnosed with marasmus living in households with a slightly higher room density than children with kwashiorkor.

4.2.20 Nutritional diagnosis and diseases of child and mother

Table 4.31 Nutritional diagnosis and HIV status of child

Variable	Child HIV Positive						95 % CI for the diff between the diagnosis and the child's HIV status
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	5	33.33	8	53.33	2	13.33	
Marasmus (n=36)	13	36.11	12	33.33	11	30.56	
Marasmic Kwashiorkor (n=3)	1	33.33	2	66.67	0	0.00	
TOTAL	19	35.19	22	40.74	13	24.07	[0.32:0.61]

Table 4.32 Nutritional diagnosis and TB status of child

Variable	Child Tuberculosis						95 % CI for the diff between the diagnosis and the child's TB status
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	2	13.33	13	86.86	0	0.00	
Marasmus (n=36)	7	19.44	23	63.89	6	16.67	
Marasmic Kwashiorkor (n=3)	1	33.33	2	66.67	0	0.00	
TOTAL	10	18.52	38	70.37	6	11.11	[0.12:0.34]

Table 4.33 Nutritional diagnosis and other diseases of the child

Variable	Child other diseases				95 % CI for the diff between the diagnosis and the presence of other diseases
	Yes		No		
	N	%	N	%	
Kwashiorkor (n=15)	4	26.67	11	73.33	
Marasmus (n=36)	6	16.67	30	83.33	
Marasmic Kwashiorkor (n=3)	1	33.33	2	66.67	
TOTAL	11	20.37	43	79.63	[0.12:0.33]

Although a high percentage of children did present with diseases (35% with HIV, 19% with TB and 30% with other disease) at the time that the survey was undertaken, there were significantly more children who did not present with these diseases (HIV: 95% CI [0.32:0.61] for the percentage difference, TB: 95% CI [0.12:0.34] for the percentage difference, other disease: 95% CI [0.12:0.33] for the percentage difference) (Table 4.31, 4.32 and 4.33).

Table 4.34 Nutritional diagnosis and HIV status of mother

Variable	Mother HIV Positive								95 % CI for the diff between the diagnosis and the mother's HIV status
	Yes		No		Unknown		Not reveal		
	N	%	N	%	N	%	N	%	
Kwashiorkor (n=15)	5	33.33	8	53.33	2	13.33	0	0.00	
Marasmus (n=36)	13	36.11	13	36.11	8	22.22	2	5.56	
Marasmic Kwashiorkor (n=3)	0	0.00	2	66.67	1	33.33	0	0.00	
TOTAL	18	33.33	23	42.59	11	20.37	2	3.70	[0.30:0.59]

Table 4.35 Nutritional diagnosis and TB status of mother

Variable	Mother Tuberculosis				95 % CI for the diff between the diagnosis and the mother's TB status
	Yes		No		
	N	%	N	%	
Kwashiorkor (n=15)	0	0.00	15	100.00	
Marasmus (n=36)	11	30.56	25	69.44	
Marasmic Kwashiorkor (n=3)	1	33.33	2	66.67	
TOTAL	12	22.22	42	77.78	[0.13:0.35]

Of all the mothers of the malnourished children in this study significantly more mothers were not HIV positive (42.59%) compared to those that tested HIV positive (33.33%) with a 95% CI [0.30:0.59] for the percentage difference. In addition, significantly more mothers did not present with TB (77.78%) compared to the mothers that were positive for TB (22.22%) with a 95% CI [0.13:0.35] for the percentage difference (Table 4.34 and 4.35)

4.2.21 Nutritional diagnosis associated with mother's lifestyle choices

Table 4.36 Nutritional diagnosis associated with mother's alcohol use

Nutritional diagnosis	Alcohol use						95 % CI for the diff between the diagnosis and the mother's lifestyle choices
	Yes		No		Do not know		
	N	%	N	%	N	%	
Kwashiorkor (n=15)	5	33.33	10	66.67	0	0.00	
Marasmus (n=36)	11	30.56	23	63.89	2	5.56	
Marasmic kwashiorkor (n=3)	2	66.67	1	33.33	0	0.00	
TOTAL	18	33.33	34	62.96	2	3.70	[0.23:0.48]

Although a large percentage of mothers did consume alcohol during pregnancy (33.33%), significantly more mothers with malnourished children did not consume alcohol during pregnancy (62.96%) with a 95% CI [0.23:0.48] for the percentage difference (Table 4.36).

Table 4.37 Nutritional diagnosis associated with quantity and frequency of mother's alcohol use

Variable	Alcohol used per day		
	Minimum	**Median**	**Maximum**
Kwashiorkor (n=5)	3.000	12.000	12.000
Marasmus (n=11)	2.000	6.000	12.000
Marasmic kwashiorkor (n=2)	2.000	7.000	12.000
Variable	**Alcohol used per week**		
	Minimum	**Median**	**Maximum**
Kwashiorkor (n=5)	1.000	2.000	4.000
Marasmus (n=11)	1.000	2.000	4.000
Marasmic kwashiorkor (n=2)	2.000	2.500	3.000

A close to significant median difference was found between kwashiorkor and marasmus and the amount of alcohol consumed per day by the mother during her pregnancy (95% CI [0: -9] for the median difference). Mothers with children diagnosed with kwashiorkor that reported that they did use alcohol during pregnancy consumed a median of 12 drinks per day, compared to mothers with marasmic children that consumed a median of 6 drinks per day (Table 4.37)

CHAPTER 5: DISCUSSION OF RESULTS

5.1 INTRODUCTION

In this chapter the results of the study will be discussed and where possible compared to the results of relevant studies of the same nature. The limitations encountered during the study will be discussed to evaluate to what extent they influenced the results.

5.2 LIMITATIONS OF THE STUDY

In both Upington and Kimberley there where a few mothers and caretakers who were reluctant to sign the consent form and therefore their children could not be included in the study. In a number of cases the mothers or caregivers were not available at the hospital for the duration of the stay of the child in the ward, and the child could not be included.

The !Xwe and Khwe (the Bushman) from Platfontein are patients at the Kimberley Hospital Complex. When these patients are admitted to hospital there is often no interpreter available. These patients do not understand English, Afrikaans or Tswana and therefore they could not take part in the study.

Problems with obtaining informed consent and qualified interpreters resulted in fewer children being included in the study than originally anticipated. At the beginning of the study a sample of 150 participants were planned. Due to the limitations mentioned above, it was not possible to recruit 150 participants and only 54 children were included in the sample. In certain instances the small sample size made statistical analysis of data difficult.

Kimberley and the clinics within Kimberley's borders are considered as urban areas, whereas Upington and all other areas and clinics are considered as rural areas. The children taking part in the study came from 18 different towns. Some of these towns (Taung, Magagong and Boshof) do not fall within the Northern Cape. Taung and Magagong are in North West and Boshof in the Free State, but since both towns are bordering the Northern Cape Kimberley Hospital Complex is the closest hospital to these patients. Patients from these areas were not excluded.

Due to financial constraints the study did not make provision for taking blood samples. The only blood values that were used in the study were routine values available in the

files of the children. Not all the children had the same blood tests done and therefore all blood values for were not available for all the children.

Even though the mothers were questioned with regard to exclusive breastfeeding, the reported length of exclusive breastfeeding could have been incorrect as mothers are still ignorant regarding the meaning of exclusive breastfeeding.

5.3 RESULTS

5.3.1 SOCIO-DEMOGRAPHIC INFORMATION

The 54 children in this study visited about 31 different clinics or health facilities in the Northern Cape (including the two from North West and one from Free State). Of these 34 facilities, 82% were in rural areas and 18% in urban areas. In this study 70% of the children came from rural areas and 30% came from urban areas. In the Northern Cape in 2001, about 83% of the population lived in urban areas (Statistics South Africa, 2004). The NFCS (1999) found that urban children were less affected by malnutrition (only about 17%) and that informal urban settlement areas were more affected. The NFCS also found that on farms one in three children were malnourished, whereas one in four children were malnourished in tribal or rural areas (NFCS, 1999). Other researchers from South Africa have also reported that rural areas had more stunted children (Kleynhans *et al*., 2006). The NFCS found that rural areas were more threatened, as 70% of the poorest households lived in rural areas (NFCS, 1999).

This study specifically looked at children 0 to 59 months old and found that 55.6% of the malnourished children had an average age of 13-24 months. Cartmell *et al*. (2005) looked at children (six months to five years old) admitted to the malnutrition ward in the Central Hospital of Maputo in 1983 and again in 2001 and found an average age of 23.8 months in 1983 and 21.7 months in 2001 (Cartmell *et al.,* 2005). Kleynhans *et al*. (2006) investigated the nutritional status of children 12 to 24 months old in Limpopo in rural villages and urban informal settlement areas and found a mean age of 18.63 months in malnourished children.

Rikimaru *et al.* (1998) determined the risk factors for developing severe malnutrition, underweight and low birth weight amongst children eight to 36 months old in the Princess Marie Louise Hospital in Accra, Ghana and found that severely malnourished children were more likely to have young mothers. Studies done in the Mulago Hospital in

Kampala, Uganda and the Moi Teaching and Referral Hospital in Eldoret, Kenya looked at children zero to 60 months and three to 35 months respectively and found an association between PEM and young (15-25 years), single mothers (Owor *et al*., 2000; Ayaya *et al.,* 2004). The age of the mother is important when she is pregnant, as younger and older women usually have a higher risk of having babies that are already malnourished or have other complications (Teller and Yimar, 2000). In this study the majority of mothers (35.19%) were between 26-35 years of age and 30% of mothers were younger (19-25 years old), which showed that they were still in their reproductive cycles.

In this study 11% of mothers had no formal education, 35 % had an educational level of up to grade seven, 52% had an educational level of grade eight to grade twelve and only 2% of the mothers had a tertiary education. Christiaenson and Alderson (2001) determined maternal knowledge in Ethiopia and found that the males in a household were often better educated than females. Sometimes parents in urban areas are slightly better educated, but even the general education level of the urban parent is still often very low. The male and female adults that had the highest education level still only had an average of a fourth and fifth grade respectively. Household members in Ethiopia with post secondary education were only found in cities and of all parents with a post-secondary education, only 3% were women and 6% men (Christiaenson and Alderson, 2001). Falbo and Alves (2002) found that 15.2% of mothers of children hospitalised in the Instituto Materno Infantil de Pernambuco in Brazil were illiterate.

The NFCS (1999) found that only a quarter of mothers in South Africa had an education. Of those, 25% had primary school, 27% high school, 25% standard 8-10 and 8% a tertiary level. Caregivers were usually less educated than mothers (NFCS, 1999). Steyn *et al*. (2005) used the anthropometric measurements of the NCFS of children 12 to 108 months old and found that stunting was directly linked to caregiver and maternal educational level.

In this study there was no significant association between the education level of the mother / caregiver and the food given to the child. In South Africa, the NFCS showed higher levels of maternal education were associated with lower levels of stunting, underweight and wasting in all age groups (NFCS, 1999). A significant correlation between level of education and anthropometry was thus confirmed (Labadarios *et al.,* 2005b).

Educational levels of parents in Ghana and India with severely malnourished children were lower than that of parents with healthy children (Jeyaseelan and Lakshman, 1997; Rikimaru *et al.,* 1998). Christiaenson and Alderson (2001) found that female education had a positive and statistically significant effect on a child's nutritional status. Maternal education is still an important issue to address, as the effect of female education on the nutritional status of children was two times larger than that of males. Mechanisms behind the association between mother's schooling and child health are still poorly understood. Mothers with post-secondary schooling had fewer malnourished children than mothers with primary and secondary schooling. Mother's that were better educated fed their children better (Christiaenson and Alderson, 2001). A study by Owor *et al*. (2000), done in Kampala, however did not find an association between PEM and level of education.

Saito *et al*. (1997) found an association between nutrition related knowledge and mild mixed malnutrition in children younger than four years old in India. There was, however no significant difference in the mother's attitudes regarding seeking health care for their children. When the mothers were questioned about their traditional beliefs, they did not believe that medical care was needed to manage childhood illnesses such as malnutrition and measles (Saito *et al*., 1997).

This study's findings correlate well with the findings of a study by Mahgoub *et al*. (2006) undertaken in Botswana amongst children zero to three years old, where 76.4% of the mothers with malnourished children were single and 22.1% of the mothers were married. In this study 81.5% of the mothers/caregivers with malnourished children were single. Maternal marital status also has an effect on child malnutrition, with the married mother being economically sounder than a single, divorced or separated mother. If the mother is married and still living with the child's father, the family can be considered economically stronger (Teller and Yimar, 2000).

In a study by Saito *et al*. (1997) in Tamil Nadu, India amongst children younger than four years old, poor nutritional status was directly associated with the gender of the child (Saito *et al*., 1997). In most studies more males are malnourished. In a study in Bangladesh on malnutrition in children six to 60 months old, there were an equal number of males and females (240 males and 239 females) (Iqbal *et al.,* 1999) and a study in Nairobi, Ethiopia, found that in the malnourished group of children three to 36 months old, 51.2% were males and 48.8% were female (Abate *et al*., 2001).

Christiaensen and Alderman (2001) found that more boys than girls younger than five years old had malnutrition in Ethiopia (Christiaensen and Alderman, 2001) and this was the same for a study in Turkey by Kilic *et al.* (2004) that found 14 male and seven female infants with marasmus and nine male and six female infants with kwashiorkor (Kilic *et al.,* 2004). Mahgoub *et al*. (2006) also found that in the age group of children zero to three years old in Botswana, malnutrition was more prevalent in males than in females. Studies in Tamil Nadu, India also showed that PEM was more prevalent in males five to seven years old. The same study found that older age was more likely to be associated with malnutrition (Jeyaseelan and Lakshman, 1997). This study therefore correlates well with abovementioned data, as a significantly higher percentage of males had malnutrition (95% CI [0.46:0.71]). In the Northern Cape there is a higher percentage of boys in the age group 0-4 years than females (Statistics South Africa, 2004).

5.3.2 ANTHROPOMETRIC INFORMATION

Birth weight is a predictor of malnutrition (Kleynhans *et al*., 2006) and there is a direct link between maternal and child nutrition (Teller and Yimar, 2000). In a study done by Falbo and Alves (2002), the median birth weight of children was 2.80kg. The study was done in Brazil between 1999-2000 and 88.9% of the children with severe malnutrition were younger than six months and 42.4% had low birth weights (Falbo and Alves, 2002). A study done by Ramakrishnan (2004) found that the prevalence of low birth weight babies was 10% for Sub-Saharan Africa, but this is not very reliable, as two thirds of births in Africa are never reported. In India, low birth weight is related to maternal nutritional factors such as energy and protein intake during pregnancy and the weight of the mother before she got pregnant (Ramakrishnan, 2004). Gupta (2008) found that low birth weight babies had a higher risk of developing feeding problems and malnutrition. In this study seventeen (31%) of the children had a birth weight of less or equal to 2.5kg.

A study in Kenya on children twelve to 59 months showed that the clinical features of malnutrition were significantly more common in children that had a weight for height of < -3SD (Berkley *et al.,* 2005). In a study done in Limpopo, South Africa, children were followed from birth up to three years of age and results showed that when a child has a greater height at one year it protects the child against stunting. Normal length and weight at one year are very important as this can predict the nutritional status of the child at three years of age (Mamabola *et al*., 2005).

With the interpretation of the MUAC in this study, a high percentage of children (38.89%) had a MUAC of less than 11.0cm (110mm), showing severe malnutrition and 28% had a MUAC of between 11.1 and 12.5 cm; indicating moderate malnutrition. The median MUAC for the malnourished children in this study was 11.55 cm. In a study done in Kenya on children twelve to 59 months, the clinical features associated with malnutrition were significantly more common in children that had a MUAC of less or equal to 11.5cm (115mm) (Berkley *et al.,* 2005). Kikafunda *et al*. (1998) found that 21.6% of Ugandan children zero to 30 months old had a MUAC lower than 13.5 cm. The risk factors for low MUAC were poor health, lack of meat and cow's milk consumption, low energy through fat, mothers with low educational levels and older mothers (Kikafunda *et al*., 1998).

In this study most of the mothers or caregivers (55.56%) had a BMI in the normal range of 18.5 to 24.9 kg/m^2. The median BMI for the mothers was 20.87 kg/m^2 and thirteen of the 54 mothers or caregivers (24%) were classified as overweight to severely obese. James *et al.* (1999) analysed data from Ethiopia, India and Zimbabwe and found that 56.3% of households had women with an average BMI of less than 18.5 kg/m^2. In only 29.9% of the Indian households, children had a normal weight-for-height and the adults had an average BMI of more than 18.5 kg/m^2 (James *et al*., 1999).

In contrast, Deleuze *et al*. (2005) conducted a study in Benin, West Africa on children six to 59 months and found that 39.1% of mothers were overweight and 15.5% were obese. Both an overweight mother and a malnourished child were found in 16.2% of the households, whereas only 12.8% of the households had an underweight mother. Households with overweight mothers were socio-economically more stable. Wasting was significantly higher in households with underweight mothers (Deleuze *et al*., 2005).

The NFCS (1999) investigated the anthropometric information of children twelve to 108 months and found that 17% of the children were overweight and obese, which was almost as high as for stunting.

James *et al*. (1999) and Deleuze *et al*. (2005) reported a correlation between children's weight-for-height and the BMI of women in the household in India and Benin, West Africa respectively. There was also a correlation between the BMI of the mother and the BMI's of the other adult women in the household. In households where the mother had a normal body weight, but a wasted child, the health issues that needed to be addressed

included parental care and not only improvement in food security. This indicates that other factors than shortage of food may determine the children's size (James *et al*., 1999).

5.3.3 HOUSEHOLD INFORMATION

In South Africa stunted children often live in households that are bigger or have more people (Kleynhans *et al*., 2006) and therefore the risk for stunting has been found to be highest in households with nine or more people in the household (Mamabola *et al.,* 2005). In South Africa about 56% of households have a size of five to nine people (Kleynhans *et al.,* 2006). The risk of children from a household in Zimbabwe and Ethiopia being stunted increased from 7% when it was only one child to 38% when the household had seven children younger than ten. In Ethiopian communities, 24% of households with more than four children were malnourished (James *et al*., 1999). In South Africa the size of a household can therefore be a predictor of malnutrition (Kleynhans *et al.,* 2006).

Of the households that were included in the NFCS in 1999, less than 60% had a monthly income of R100-R1000 (NFCS, 1999). When the NFCS Fortification Baseline (NFCS-FB-1) was repeated in 2005, 55% of households had an income of R1-R1000 per month. The informal urban sector had a higher percentage of households that had no income (6%) and 35% of households had an income of R1-R500 per month (Labadarios *et al*., 2008). Socio-economic status is linked to income and malnutrition (Pierecchi-Marti *et al*., 2006). Only one in four households (25%) in South Africa appeared to be food secure (NFCS, 1999), with 35% of at risk households being food insecure (Hendricks *et al*., 2006). In 2005 the conditions appeared better, but there was still one in two households that were experiencing hunger, one in five households that were food secure and one in three households were at risk of experiencing hunger. The highest percentage of hunger was in the Northern Cape (63%) (NFCS, 1999) with the Eastern Cape and Limpopo having six out of ten households experiencing hunger. Hunger in general did not improve in 2005, due to lower incomes, lower education level of the mother and more participants living in informal dwellings (Labadarios *et al*., 2008).

In this study the majority of the households had four to five people in the households, with only 7% consisting of two people. Two percent of households had more than nine people in the household. Most of the households in this study had a high room density with two to five household members per room. The mother of the child was the head of the

household in 26% of cases followed by the child's grandmother and grandfather in one out of five households. The NFCS (1999) found that in 42% of households the father was the head of the household and in 11% of households the mother was the head of the household. In other households the grandparents, especially the grandmother, were the head of the household (NFCS, 1999).

In this study the father was the breadwinner in 50% of households and in 17% of households the father was unemployed. In 1999 one fifth of households included in the NFCS had a mother as the breadwinner and in half of the households the mother was unemployed (NFCS, 1999). The NutriGro study undertaken by Kleynhans *et al*. (2006) in rural Limpopo and urban Gauteng showed the mother was the primary caregiver in 70.9% of cases, the head of the household in 36% of cases and the father was the head of the household in 29.7% of the cases (Kleynhans *et al.,* 2006). With the NFCS-FB-I in 2005, the survey found that 50% of households had males (father, husband) as the head of the family and the father was the respondent in one in every three households. In the same study the mother's husband and grandfather were the respondents in 17% and 2% of the household, respectively (Labadarios *et al*., 2008).

Some other socio-economic issues that are linked to stunting are the type of house (especially in urban areas), type of toilet in the home, fuel used in cooking, presence of refrigerator or stove and television (NFCS, 1999; Steyn *et al*., 2005) and the educational level of the parents. When paraffin is used as fuel instead of electricity, it can lead to a higher risk for stunting (NFCS, 1999) and Jeyaseelan and Lakshman (1997) found that using dung or firewood as fuel were risks for developing malnutrition. The possession of a flush toilet in the house has a positive effect on height (Christiaenson and Aldeman, 2001).

5.3.4 MATERNAL INFORMATION

In this study almost all the mothers were alive (96%) and in the two cases were the mothers were dead, the grandmother and aunt looked after the child. Kleynhans *et al.* (2006) found that children that lived in households where grandparents were caregivers had the highest rate of stunting. In rural areas it is usually the grandmothers that are the caregivers, but evidence from a study in Limpopo, South Africa amongst children twelve to 24 months of age showed that children had a lower risk of stunting if the mother was the caregiver (Kleynhans *et al.,* 2006). In Nigeria 450 mothers were interviewed and 77%

of mothers cared for their own children, while 23% of mothers had somebody that cared for their children (Ogunba, 2008). In this study 74% of the children were cared for by their parents and other people that cared for the children included grandparents, other family members and day care centres. In a study done in Kenya amongst children three to 36 months old, the caretaker of the malnourished children was most often not married to the child's parent and children with malnutrition had not been staying with both parents during the previous six months (Ayaya *et al.,* 2004).

In the NFCS, 13% of mothers that were stay at home mothers, did so by choice (NFCS, 1999). This study showed that 67% of mothers were stay at home mothers looking after their own children during the day, whereas 28% of children were cared for by grandmothers, 6% by a neighbour, 2% by a day care centre and 17% by other people.

In this study 37% of mothers had only one live birth (the child in the study), 19% had two live births, 26% had three live births, and 19% had more than four live births. Saloojee *et al*. (2007) found in a study done in Limpopo that in most malnourished children (51%) with siblings, there was a high birth order of three or more and 15% of the malnourished children had siblings that had died. This correlates well with the results of this study where 11% of mothers had lost one to two children to death. In most cases, the mothers did not know what the child had died of. Three mothers reported that they had lost their children due to pneumonia, gastroenteritis and liver disease.

About a third of the siblings of the child included in the study had also been previously admitted to hospital (33%). The reasons why they had been admitted to hospital were as a result of respiratory problems or asthma (8%), gastroenteritis (6%) and TB (4%). Other reasons for admittance included flu, fever, accidents, pneumonia, and sores in mouth, malnutrition, ear infections, blood transfusions and liver disease.

This study showed no significant association between the nutritional diagnosis (kwashiorkor, marasmus and marasmic kwashiorkor) and number of births. A study undertaken by Jeyaseelan and Lakshman (1997) in India amongst children five to seven years old, found that the high birth order of a child was associated with the child being malnourished. Similarly, a study undertaken by Teller and Yimar (2000) in Ethiopia amongst mothers 15 to 49 years old and children younger than five years old, showed the

highest rate of stunting in children with a birth order of four or five (54%) and then a birth order of six or more (53%).

5.3.5 MATERNAL MEDICAL INFORMATION

USAID (2001) reported on the progress of MTCT and VCT in Sub-Saharan Africa and stated that mothers, who accessed VCT before or during pregnancy had a lower MTCT rate due to the fact that they could be better counselled on preventative measures (USAID, 2001). In this study, the majority (70%) of mothers had received VCT. Despite this, 30% did not know their status and are at risk of becoming sick if they do not access treatment early.

In Zaire, the severity of maternal disease influences the degree of growth retardation. The intra-uterine growth of infants that are born to HIV infected mothers is not optimal and low birth weight (<2500g) babies were more prevalent in HIV infected than HIV uninfected mothers. As expected, the intra-uterine growth of children of mothers with AIDS was compromised (Eley and Hussey, 1999).

In this study, most mothers that had received VCT were HIV uninfected, 33% were HIV infected, and two mothers did not want to reveal their status, even though they knew what it was. One in five mothers however, did not know their status. Some of the mothers that were HIV and TB infected had received treatment. Some of the mothers were on HAART (11%), some of the mothers were participating in the PMTCT programme (9%) and 7% of the mothers were on TB treatment. Only one mother reported having another disease, such as a heart defect.

Studies reported by the United States Agency for International Development (2009) and Chatterjee *et al*. (2007) showed the importance of mothers receiving treatment for illnesses. The United States Agency for International Development found that HAART treatment started during pregnancy resulted in lower transmission rates to the baby. The study undertaken by Chatterjee *et al*. (2007) amongst pregnant women and their infants up to twelve months of age showed that a lower CD4 count of the mother during pregnancy resulted in higher mortality rates in their infants.

This study also found that significantly fewer children with malnutrition had other diseases such as cerebral palsy, liver and heart disease and GI problems than those that were only

malnourished without any other disease. The study found that significantly fewer mothers with malnourished children were HIV and TB infected. A study by Saloojee *et al.* (2007) in South Africa amongst children younger than five years old found a positive association between the illness of the mother (41%) and the chances of a child being malnourished, with children of mothers that were ill having a higher chance of being malnourished.

Clinic attendance of mothers during pregnancy was relatively acceptable, with 87% accessing antenatal care during pregnancy. In a study by Teller and Yimar (2000) in Ethiopia aimed at determining the nutritional status of women and children younger than five years of age, antenatal visits were related to stunting in a child, with the prevalence of stunting decreasing as the number of antenatal visits of the mother increased.

Of the 54 mothers participating in the study, 18 consumed alcohol while pregnant with the child in the study. Of the respondents that were not the mothers themselves, two did not know if the mother had used alcohol during her pregnancy. Of the mothers that did consume alcohol, the amount of alcohol consumed was quite high, with nine of the mothers consuming between two to ten drinks per day. About 44% of the mothers could not report how much they drank per day. Most of the mothers consumed the mentioned amount of alcohol once or twice per week. A study undertaken by Setswe (1994) in Bophuthatswana amongst children younger than five years of age, showed an association between child malnutrition and the consumption of alcohol.

In this study the majority of the mothers smoked while pregnant with the child in the study (52%). According to Taylor and Wadsworth (1987) the rates of lower respiratory tract illness in children are higher in children with mother's that smoked during and after pregnancy. In a study undertaken in the United Kingdom amongst children from birth to five years of age, most of the hospital admittance of children for bronchitis and upper respiratory infections were related to maternal smoking as well as the number of cigarettes smoked per day. Mothers were followed up after birth and 90% of mothers that smoked during the pregnancy still smoked five years later. If a mother started smoking after birth the impact on the health of the child was lower than during pregnancy (Taylor and Wadsworth, 1987).

In this study significantly fewer mothers did not consume alcohol and for those that did consume alcohol there was a close to significant association between the malnutrition of

the child and the amount of alcohol the mothers consumed. Kyu *et al*. (2009) undertook a study in seven countries on women and their children and found a significant association between smoking and growth deficiencies of children. Other associations found in Indian households were between tobacco and alcohol use and low rates of immunization, higher prevalence of anti-retroviral medication, malnutrition and death of infants before one year of age (Bonu *et al*., 2009). In Bophuthatswana, Setswe (1994) also found an association between alcohol consumption and low resources and malnutrition.

5.3.6 MEDICAL HISTORY OF THE CHILD

In South Africa, rates of malnutrition are generally high. The NFCS survey was undertaken amongst South African children one to nine years old and showed that stunting was prevalent in one out of five children of one to three years old (NFCS, 1999; Labadarios *et al.,* 2005b). Kleynhans *et al*. (2006) reported the same results amongst children twelve to 24 months of age in Limpopo and Gauteng. Stunting in four to six year old children was 21% in the NFCS (Labadarios *et al.,* 2005b). The rate of stunting amongst one to nine-year-old children was the highest in the Northern Cape at 31% (NCFS, 1999). Stunting was also high in the Eastern Cape and Northern Province amongst children younger than five years due to the high levels of poverty (Zere and McIntyre, 2003). Stunting was also quite high in Limpopo amongst three-year-old children with 48% of children there being stunted (Mamabola *et al.,* 2005).

With the NFCS-FB-I in 2005 in South Africa amongst one to nine year old children, the survey found that rates of stunting and underweight increased in twelve to 71 month old children (Labadarios *et al*., 2008). In South Africa, underweight affects one in ten children, with 1.5% being severely underweight (NFCS, 1999). Underweight decreased in one to three year olds and was the highest in four to six year olds (NFCS, 1999); with 9% of children twelve months to 24 months being underweight (Kleynhans *et al.,* 2006).

In South Africa wasting was not that prevalent in children one to nine years old with less than 5% of children in the NFCS being wasted (NFCS, 1999). Wasting was still about 2% in the Limpopo Province amongst children twelve to 24 months old (Kleynhans *et al*., 2006) and severe wasting less than 1% in 2005 (Labadarios *et al.,* 2008). Rates of wasting remained constant in all age groups (NFCS, 1999).

Children included in this study were all malnourished. The prevalence of marasmus was the highest, with 36 (67%) of the children presenting with wasting, 28% with kwashiorkor and only two children with marasmic kwashiorkor. In Kenya 16% of malnourished children twelve to 59 months admitted to hospital presented with severe wasting (9%), kwashiorkor (9%) or both (Berkley *et al.,* 2005). In a study done in Maputo amongst children six months to five years of age, the data from 1983 was compared to that of 2001 and the prevalence of malnutrition of children in hospital was lower in 2001 than in 1983. In 2001 there were 32.9% children with kwashiorkor, 25.8% with marasmus and 28.4% with marasmic kwashiorkor (Cartmell *et al.,* 2005).

A study done in Bangladesh amongst children six months to 60 months used the Gomez classification and classified 96% of those children as malnourished, with 28.4% having mild, 58.2% moderate and 9.2% severe malnutrition. When the Waterlow classification was used, 84% of children were classified as stunted and 67% wasted (Iqbal Hossain *et al.,* 1999). In Nairobi (Ethiopia), 86.2% of children three to 36 months old were stunted, 34.7% were underweight and 3.4% were wasted (Abate *et al*., 2001). In Delhi 75% of children nine to 36 months old were underweight, 35% were severely malnourished, 74% were stunted and 19% were wasted (Kapur *et al*., 2005).

5.3.6.1 BIRTHWEIGHT, RtHC AND CLINIC ATTENDANCE

In a study done in Limpopo, South Africa most children twelve to 24 months old that had a birth weight of less than 2.5kg, were more likely to develop stunting. About 25% of the stunted children weighed less than 2.5kg at birth (Kleynhans *et al*., 2006). In a study undertaken by Falbo and Alves (2002) amongst infants younger than six months old, 36.4% of the children were born prematurely. In this study only 20% of the children were born prematurely.

Most of the children (89%) included in this study were born in a health facility such as a hospital or a community health centre. The remaining 11% were born at a clinic, at home or on the street.

In the Western Cape, undernourished children were missed due to nurses not plotting weights on the RtHC (Hendricks *et al*., 2006). In this study significantly more malnourished children (44%) had completed RtHC compared to the malnourished children who had incomplete cards (30%) (95% CI [0.47:0.77]). Even though the majority

of the children had completed RtHC, cards were not necessarily interpreted correctly and interventions were not put in place to manage and treat these children.

In 1998, only 74.6% of 12-13 month olds had RtHC cards and the target for 2007 was set at 85% (Hendricks *et al*., 2006). In this study all the children that took part in the study had an RtHC as this was one of the inclusion criteria. Even though all the children had a RtHC, only 44% of the cards were filled in correctly and in 30% of cases, the cards could not be evaluated due to the fact that the child had a card, but the card was at home.

In this study most of the children had visited the clinic about one to eight weeks prior to being admitted to hospital and in two cases the children had visited the clinic more than 48 weeks previously. The children were usually taken to clinic for immunizations (57%), growth monitoring (44%) or for other small ailments (54%) such as flu, accidents, vomiting, losing weight, vitamin A supplementation, gastro-enteritis, liver disease, fits, etc. In a study undertaken by Abate *et al.* (2001) amongst children three to 36 months old, 76% of mothers took their children to hospital or clinics for the treatment of diarrhoea.

Even though there was no significant difference between the nutritional diagnosis of the child and the last time the child visited the clinic, the long periods between visits can lead to sick children not being seen early enough to treat effectively. Clinic attendance is also the time when health and nutrition information can be given to the mother and if the child is not taken to the clinic, the mother misses out on important information regarding her child's health.

5.3.6.2 IMMUNIZATIONS AND VITAMIN A SUPPLEMENTATION

In South Africa immunizations in 2006 showed 84% coverage for BCG, Hepatits B, polio, DPT3-Hib (third dose of diphtheria-tetanus-pertussis vaccine and *Haemophilus influenzae* type b vaccine) and measles (Every death counts, 2008). In this study 56% of the children had immunizations that were up to date, but 37% of the children had outstanding immunizations. In Ethiopia 80.2% of children three to 36 months old were fully immunized and the proportion of malnourished children that were fully immunized for age was not significantly different from that of well-nourished children (77,6%) (Abate *et al.,* 2001). In Bangladesh 77% of children between six and 60 months of age received BCG and 82% received full or partial DPT and polio immunizations. There was a significant

association with malnutrition when no vaccines were available. Of the children in Bangladesh, 75% had received measles immunizations (Iqbal Hossain *et al.,* 1999).

The study showed that 56% of children were up to date with their immunizations, but 37% of the children still had outstanding immunizations. These children are more prone to illnesses and infections, which results in a higher chance of developing or worsening malnutrition. Three other studies also found that incomplete immunizations were directly associated with malnutrition. These studies were undertaken in Ethiopia amongst children younger than five years old (Getaneh *et al*., 1998), in Uganda amongst children zero to 60 months (Owor *et al*., 2000) and in Kenya amongst children three to 36 months of age (Ayaya *et al.,* 2004).

Vitamin A is necessary for a well functioning immune system and a deficiency can cause high risk of mortality. In 2008, 71% of 6-59 month old children were protected against a deficiency because of the two doses they received twice per year through the vitamin A supplementation programme. In 2008, 22 of the 34 least developed countries passed the 80% coverage rate. The coverage doubled from 41% in 2000 to 88% in 2008 (UNICEF, 2009c, p.27).

Fifty percent of the children in this study were not up to date with their vitamin A supplementation and 15% of mothers weren't sure whether they had received vitamin A supplements. Only 35% had complete vitamin A supplementation for their age. In a study undertaken in an informal settlement in Durban, South Africa by Coutsoudis *et al.* (1993) amongst children three months to six years, the preschool children presented with low vitamin A status in 44% of the group. Five percent 5% had a vitamin A deficiency (Coutsoudis *et al.,* 1993).

Vitamin A coverage in South Africa is 72.8% in 6-11 month olds and 13,9% in 12-59 month olds. The big difference in coverage can be attributable to poor clinic attendance of children older than two years after immunizations are completed (Hendricks *et al.,* 2006). There are still two out of three children and one out of four women with a poor vitamin A status. In the NFCS-FB-I, 25% of one to four year olds (12-59 months) had received a high dose vitamin A supplement in the previous six months and 10% of mothers weren't sure if their children had received vitamin A or not (Labadarios *et al*., 2008).

Vitamin A is directly linked to infections as well as mortality and therefore it is important to protect children against illnesses and infections by giving them six monthly doses of vitamin A. The coverage for children older than two years of age is still very poor. A study undertaken by Ferraz *et al.* (2005) in Brazil amongst children older than 24 months and younger than 72 months found that 75% of this age group were still deficient in vitamin A.

5.3.6.3 HIV AND TB

The HIV epidemic has worsened the severity of clinical problems associated with malnutrition. In this study 35% of the children tested positive for the HIV infection and in 24% of the cases the HIV status of the child was unknown. Of the HIV positive children, 36% were marasmic. In Lusaka, 25% to 33% of antenatal mothers were HIV infected and 54% of the HIV infected children of six to 24 months old presented with persistent diarrhoea (Amadi *et al*., 2005). In Maputo, 11.6% of children six months to five years were HIV infected (Cartmell *et al*., 2005).

In a study in Zimbabwe amongst children older than fifteen months, marasmus and marasmic kwashiorkor were the dominant forms of malnutrition in HIV positive children (Ticklay *et al.,* 1997). HIV infected children can also present with other infections, such as pneumonia (68%), lymphadenopathy, chronic ear discharge and oral thrush (11%). The high prevalence of HIV infection amongst malnourished children emphasises the impact of the HIV epidemic on childhood morbidity (Ticklay *et al.,* 1997).

A study undertaken in Uganda showed that 3% of HIV infected children of less than six months had kwashiorkor. In 72% of the cases, HIV infected children presented with other infections, such as pneumonia (68%), bacterial infection (18 %), urinary tract infection (26%), malaria (9%), diarrhoea (38%) and oral thrush (11%) (Bachou *et al.,* 2006). Bachou *et al*. (2006) found that 38% of female children that were HIV infected had a median age of 17.0 months. There was no difference in HIV infection between genders (Bachou *et al.,* 2006).

Undernutrition is a major problem in HIV infected children in South Africa. More than 50% of children with HIV infection become stunted or underweight and at least one in five are wasted. Marasmus is more prevalent in HIV than kwashiorkor, with 6.7% of children being severely wasted at the Red Cross Children Hospital in Cape Town. Severity of

malnutrition in HIV infected children is associated with a higher risk of dying (Hendricks *et al.,* 2006).

In this study only 19% of the children tested positive for TB and in 11% the TB status was unknown. Of all the mothers taking part in this study 78% did not have TB at the time of the interviews, but some of the other members in the family did have TB. Some of these members were the mothers' brother (17%), the child's father, the grandmother, aunt, grandfather and uncle. Of the children that were HIV infected and had TB, 13% received HAART and 20% received TB treatment. Therefore all children that had TB were on treatment compared to less than half of the children that were HIV infected receiving HAART. In Maputo 14% of children six months to five years had TB in 2001 and 6% had TB in 1983 (Cartmell *et al.,* 2005). Important risk factors for TB transmission in children were a young age, severe malnutrition, the absence of the BCG vaccine, contact with an adult that is sputum positive and exposure to tobacco smoke (Singh *et al*., 2005).

5.3.6.4 NATIONAL SUPPLEMENTATION PROGRAMME

Even though all the children in the study were malnourished, only 41% of the children had accessed the NSP. Of the children that were part of the NSP, 77% were already on the programme for one to eight months and five of the children were on the programme for longer than nine months and were still malnourished. Significantly more malnourished children had not been entered onto the NSP, than those that had accessed the program (95% CI [-.29:0.55]). Those that need to receive supplements are still missed and those that are on the scheme are not followed-up effectively, which leads to children not exiting the programme. A study by Hendricks *et al*. (2006) found that 38% of children in the Northern Cape were supplemented and showed catch up growth with supplementation.

5.3.6.5 HOSPITAL ADMITTANCE

The children in this study that were admitted to hospital also had other diseases such as gastro-enteritis (18%), cerebral palsy (18%) and a heart defect (18%). Significantly more malnourished children (13.5%) of three to 36 months of age in Ethiopia had diarrhoea than well-nourished (4.2%) children (Abate *et al.,* 2001). A study by Saloojee *et al*. (2007) undertaken in Limpopo, South Africa amongst children younger than five years old, also showed that malnourished children had been admitted to hospital on previous occasions (38%).

There was a close to significant association between marasmus, kwashiorkor and previous admittance to hospital for episodes of diarrhoea, with 57% of children having been admitted to hospital previously for diarrhoea. Children had been admitted once before (45%), some were admitted for the first time (26%) and some admitted three times before (23%).

Abate *et al.* (2001) found that diarrhoea was the reason for 13.5% of admissions of children in Ethiopia of three to 36 months of age. Falbo and Alves (2002) undertook a study in Brazil and found that diarrhoea was the main reason for admission in 55.6% of cases. In South Africa, 76% of well-nourished households took their children to hospital and clinics for the treatment of diarrhoea and only 58% of malnourished households took their children for treatment (Abate *et al*., 2001). In Gambia, the primary diagnosis of children younger than five years of age admitted to hospital was malaria (58.8%), ARI (11.9%), gastro-enteritis (7.5%) and skin infections (4.9%). None of the children had a primary diagnosis of malnutrition, but in 4.8% of cases malnutrition was the secondary diagnosis (Hamer *et al*., 2004). In Maputo, there were fewer admissions of malnourished children of six months to five years, but those that were admitted had a higher percentage of severe underweight and in 2001 there were more secondary infections such as malaria (40%), bronchopneumonia (53%), anaemia (65%), diarrhoea (35.7%), whereas in 1983 the admissions were due to anaemia (37%), malaria (18%), diarrhoea (8%), bronchopneumonia (28%) and measles (4.4%)(Cartmell *et al*., 2005).

5.3.7 BIOCHEMICAL INFORMATION

The biochemical information was not available for all the participants as only blood values that had already been taken routinely were used and no new bloods were drawn and analysed for this study.

During malnutrition low haemoglobin and serum albumin concentrations are common. Haemoglobin and albumin are used as markers of severity of clinical illness (Amadi *et al.,* 2005). In HIV infected Ugandan children, haemoglobin was below 9g/dL (Bachou *et al.,* 2006). This study found that 85% of malnourished children had an albumin of less than 32 g/L.

In Tanzania the mean haemoglobin in malnourished children three to 23 months old was 10.9g/dL. Sixty eight percent were moderately anaemic with haemoglobin less than

11g/dL and 11 % were severely anaemic with haemoglobin less than 7 g/dL. Of all the children, only 21 % were not anaemic with haemoglobin of more than 11 g/dL. Predictors of anaemia were low birth weight and an iron deficiency (Mamiro *et al.,* 2005). This correlates well with results from this study where 52% of the children were anaemic with haemoglobin of less than 10 g/dL.

In this study the majority of children (71%) had a C-reactive protein of more than 10 (between 15-310), which showed that the children had some kind of infection. The absolute CD4 count and CD4 percentage were only available for two children. Malnutrition and the resultant low CD4 count are directly related to a low survival rate of infants and children. In Uganda, the CD4 percentage was less than 25% in one third and 15-24% in 17% of children. CD4 count was lower in the presence of oedema in 12-24 month olds. Marasmic children had a low CD4 count and percentage (Bachou *et al.,* 2006). CD4 count and C-reactive protein levels can characterize the nutritional status of the child (Emwonwu, 2006).

5.3.8 MATERNAL EDUCATION

Most of the mothers of the malnourished children included in this study did not know how to explain what diarrhoea is (65%). Most of the mothers had received some kind of information on childcare practices at the clinic. Some of the information received from the clinic included information on diarrhoea (25%), healthy eating habits (49%), breastfeeding (62%), complementary feeding (49%), food fortification (15%), explanation of the growth chart (34%) and hygiene (51%). Only two mothers said that they hadn't received any information at the clinic. Information received on feeding during times of illness was inadequate, especially if only 35% of mothers knew what diarrhoea was and only 25% of mothers visiting clinics received information regarding diarrhoea.

A study in Ethiopia amongst children three to 36 months old showed no significant difference between the health practices of mothers with malnourished children (38.5%) that withheld food during episodes of diarrhoea and those of well-nourished children (40.1%). The mothers in the Ethiopian study, which withheld food from their children during episodes of diarrhoea, did not give fruit, vegetables and milk. In malnourished children, the foods that were withheld during diarrhoea included porridge and potatoes (Abate *et al.,* 2001).

A study undertaken in India amongst children younger than four years old also showed no significant difference in health practices between mothers of malnourished and well-nourished children. The health practices were often based on traditional beliefs and mothers did not believe in medical care for childhood illnesses (Saito *et al*., 1997).

5.3.9 INFANT FEEDING INFORMATION

A mother's circumstances during pregnancy and birth will influence her choice of infant feeding (Sowden *et al.,* 2009). In 2005 only 178 (37%) of facilities in South Africa were baby friendly according to the Baby Friendly Hospital Initiative, with a target of 15% set for 2007, which was already reached (Hendricks *et al*., 2006). According to UNICEF (2009c, p.23), less than 40% of infants in the developing world receive immediate breastfeeding after birth. Only 39% of babies are put to the breast one hour after birth despite the fact that early initiation of breastfeeding can contribute to reduced neonatal mortality through skin-to-skin contact that can prevent hypothermia (UNICEF, 2009c, p.26).

In South Africa, the SADHS showed that 20.1% of children were never breastfed and only 11,9% of infants zero to four months old were exclusively breastfed (Hendricks *et al*., 2006). In this study 89% of the children had been breastfed at one stage in their lives. Breastfeeding was done in 35% of zero to six month olds, 27% of seven to 12 month olds and 20% of thirteen to 18 month olds. In some instances the mother reported breastfeeding for more than two years. In Limpopo, South Africa a study undertaken amongst children 12-24 months old found that 73% of stunted children were breastfed for thirteen months or more and 10% were breastfed for less than a month (Kleynhans *et al*., 2006). According to the NFCS, in the Northern Cape only 10-20% of babies were breastfed occasionally (NFCS, 1999). In South Africa 75% of children received continued breastfeeding at one year and only 50% at two years (UNICEF, 2009c, p.23). According to a study undertaken in Brazil on children admitted to hospital, 19.2% of mothers never breastfed and 49.5% of children were breastfed for less than two months (Falbo and Alves, 2002).

In South Africa exclusive breastfeeding is not widely practised (Kleynhans *et al*., 2006). In this study 86% of the children were reportedly exclusively breastfed for zero to six months of age. This does not correlate well with other studies undertaken in South Africa and in the world as the exclusive breastfeeding rate is usually very low. It is possible that the mothers were not sure of what exclusive breastfeeding entails and therefore reported

longer periods of exclusive breastfeeding than was actually happening. Worldwide 37% of infants younger than six months of age are exclusively breastfed. The rate is low in Africa with less than one third of infants younger than six months receiving exclusive breastfeeding. Over the last ten to fifteen years exclusive breastfeeding increased in Africa from 33% in 1995 to 38% in 2008 (UNICEF, 2009c, p. 24). In a study undertaken in Malawi, infants were followed up from birth to twelve months and only 13.3% of mothers exclusively breastfed their children (Kalanda, 2006).

Appropriate breastfeeding practices and duration of exclusive breastfeeding is based on the information the mother received on the importance of breastfeeding to fight infant morbidity and promote growth in infancy. According to the WHO (2007b), exclusive breastfeeding for six months is recommended. Mothers that have no education usually exclusively breastfeed for a median of 1.1 months, whereas mothers with a higher education usually exclusively breastfeed for only 0.4 months. Better education is usually linked to better socio-economic status and that can be the reason for better-educated mother's deciding not to breastfeed (Sowden *et al.,* 2009).

Teller and Yimar (2000) undertook a study in Ethiopia amongst children younger than five years and found that exclusive breastfeeding for longer than six months is often a cause of malnutrition as the breast milk or other fluids being given to the children are not sufficient to meet the energy and nutrient requirements for a child older than six months of age. On the other hand, a lack of exclusive breastfeeding can cause stunting (Teller and Yimar, 2000).

Mothers that were not breastfeeding at the time of the interview gave their children formula milk in 47% of the cases, while 11% gave cow's milk and 42% gave other milk such as Nido and Nespray. In most cases the milk was given to the child in a bottle (86%) and only 7% used a cup and 10% used a spoon to give the milk to their babies. According to a study in Bangladesh amongst babies six to 60 months old, 48.3 % of babies received milk via a bottle and only 7% were breastfed (Iqbal *et al*., 1999). Most of the mothers in this study knew how to prepare the milk hygienically even though they did not know what the sufficient amount of milk powder and water was for their child's age. Only 5% of the children receiving other milk than breast milk received enough milk for their age.

This study found that significantly fewer children consumed cow's milk than other types of milk. As expected, Kikafunda (1998) found in Ugandian children younger than 30 months, that children that never consumed milk showed a higher incidence of underweight than those that received milk regularly.

In a study undertaken in Malawi amongst infants up to twelve months old, 83.5% of breastfed babies received water at three months, 65,2 % received porridge and 33.1% received other food (Kalanda, 2006). In Bangladesh, children six to 60 months old started solids at the mean age of eight months (Iqbal Hossain *et al*., 1999). The study in Tanzania amongst three to 36 months old children found the highest prevalence of malnutrition at the age when the child is weaned from breastfeeding and starts with solids (Mamiro *et al*., 2005). Worldwide 60% of six to nine month olds receive solid, semi-solid or soft food while being breastfed. The quality of food given is not always known, but may not be the right type of food for the child's age, can be inadequate for the child's age, has insufficient protein, fat and micronutrients for growth and development and may not be given frequently enough (UNICEF, 2009c, p.26).

According to a study undertaken in Limpopo, South Africa by Kleynhans *et al*. (2006) amongst children twelve to 24 months of age, mothers in rural areas often start earlier with solids. Most of the food given is home prepared (65%), 12% are commercially prepared food and 23% give both home prepared and commercial foods. Mothers started with water at less than one month of age in 79.29% of cases and 20.75% started with water when their children were older than one month of age. Solids are started at less than one month of age (36.2%) and 63.8% started with solids when their children were older than one month of age. Predictors of malnutrition include starting solids too soon and giving complementary foods before three months old (43%)(Kleynhans *et al*., 2006).

Even though significantly more children were breastfed at one time of their lives, there could still have been problems with the care practices of the mother. She may have given insufficient amounts to drink or gave other food and drinks that resulted in the baby not picking up weight even though she was breastfeeding.

Vaahtera *et al.* (2001) undertook a study in Malawi amongst newborn infants and found a positive association between maternal education and prolonged breastfeeding and in a study undertaken in Kenya amongst children zero to two years of age by Kamau-Thuita *et*

al. (2002) found that improved maternal knowledge lead to better care practices. A study in Ethiopia amongst children younger than five years old showed a positive association between malnutrition and prolonged breastfeeding (Getaneh *et al*., 1998) and a study undertaken in Kampala amongst children zero to 60 months showed a positive association between malnutrition and lack of breastfeeding (Owor *et al*., 2000).

A study by Serventi *et al*. (1995) undertaken in Tanzania amongst children younger than two years showed that 62% of children were weaned before two years of age and after weaning there was a drop in their growth curve. However, Martin (2001) found a positive association between prolonged breastfeeding (longer than one year) and malnutrition. Similarly, Coutsoudis *et al*. (1999) found that prolonged breastfeeding could be detrimental to children due to a reduction in the consumption of complementary foods. Children have a high risk of developing micronutrient deficiencies due to human milk having a low concentration of iron and zinc. Prolonged breastfeeding also prolongs the exposure to HIV in an HIV infected mother and early introduction of water can cause a higher morbidity due to diarrhoea, linear growth faltering and increased risk of MTCT (Coutsoudis *et al*., 1999).

A study in Limpopo, South Africa amongst twelve to 24 month old children Kleynhans *et al*. (2006) found that only 7.72% of infants had been breastfed for one month or less, with only 17.89% breastfed for one month to twelve months of age and 74.39% breastfed for thirteen months. In this study there was no significant difference between marasmus and kwashiorkor in terms of when breastfeeding was stopped. In marasmic children, breastfeeding was stopped at a median age of 11.5 months and in kwashiorkor at a median age of nine months.

Kikafunda (1998) undertook a study in Uganda amongst children less than 30 months of age and found an association between stunting and the age of termination of breastfeeding or the duration of breastfeeding. The risk of stunting was lower when a child was breastfed to eighteen months compared to those that were weaned early. If the child was breastfed until two years of age, however, the risk for stunting increased seven times due to breast milk not providing sufficient nutrients (Kikafunda, 1998).

This study found no significant difference between kwashiorkor and marasmus and when exclusive breastfeeding was stopped. Mothers of marasmic babies stopped at a median

age of 4 months and mothers of babies with kwashiorkor stopped at three months. Even though this difference was not statistically significant, it could have clinical value. The malnutrition could possibly have developed due to the children not receiving sufficient milk after breastfeeding was ceased.

In a study undertaken in Delhi amongst children nine to 36 months of age, significantly more children received insufficient quantities of milk for their age. In India the milk consumed was almost sufficient, but in 18% of children, milk intake was inadequate (Kapur *et al*., 2005). Kikafunda (1998) found that both marasmus and kwashiorkor cases in children younger than 30 months in Uganda were linked to a lack of milk consumption. In this study 90% of all the children that consumed milk did not consume enough for their age.

Kalanda (2006) found that children up to twelve months of age have a higher risk of contracting respiratory infections when solids are introduced early. Low maternal literacy was also associated with early introduction of solids. When solids are introduces early, it can lead to a lower weight-for-age at three, six and nine months and a lower height-for-age at nine months (Kalanda, 2006). According to Verhoeff *et al*. (1999) a study undertaken in Malawi amongst pregnant women whose babies were followed up for one year and a study undertaken by Kalanda (2006), showed that when complementary feeding is introduced at a later stage, there is better growth and significantly lower morbidity from respiratory infection, malaria and eye infection.

In this study solid foods were mostly introduced between zero to four months (39%). Thirty five percent introduced solids at five to six months of age and 16% of mothers introduced solids at seven to twelve months of age. One mother introduced solids after thirteen months of age. No significant difference between marasmus and kwashiorkor and the median age when solids were started was found.

5.3.10 FOOD BASED DIETARY GUIDELINES

In this study the food intake of the malnourished children was evaluated using a questionnaire based on the FBDG of South Africa. Other studies often use food frequency or recall questionnaires to determine food or nutrient intake. The NFCS of 1999 determined intake through a 24-hour recall and did not compare intake with the FBDG. In the NFCS the most commonly consumed foods were maize, sugar, tea, whole

milk, brown bread and hard margarine, with a very high consumption of sugar. Intake of animal foods and maize were directly linked to household income (NFCS, 1999).

Children did not consume a variety of food, as there was a low consumption of fruit, vegetables, animal proteins and alternative protein sources. In this study both white and brown bread were bought in equal quantities. The NFCS showed that nine out of ten households bought mostly maize, three out of four buy wheat, and seven out of ten buy cake flour for baking bread. Eighty percent of households bought bread and seven out of ten bought brown bread. Some of the most popular food items in South Africa are vetkoek and steamed bread and salt is always available. As the household income increases less maize is bought and more bread and wheat flour are bought (Labadarios *et al*., 2008).

This study also found that another challenge for healthy eating habits were the way that mothers or caregivers prepared the child's food. This included the items that were added to the staple food of the household, such as the maize porridge. The items that were usually added to the porridge were margarine, sugar, meat and milk, in order of preference. Children had a very high salt intake, as the mothers are prone to adding salt, aromat, stock blocks, spices and soup powders to the child's food during preparation. Fat, oils and animal fats were also often used during preparation. This showed that children were not keeping to the guidelines of the FBDG were it is stated that fats, oils, salt, sugar and sugar containing food must be used sparingly.

Almost 50% of the children in this study did not eat meat and when it was eaten, they only ate it about once a week. Alternative protein sources such as soy mince were used and few children consumed baked beans in tomato sauce. At least about 78% of the children ate some of these items during the week. Meat, chicken, fish, eggs and milk intake can probably be linked to household income. Even though parents complained about income and the food they had to buy, a large percentage often consumed unhealthy foods.

This study found that when the mother looked after the child during the day, the child consumed significantly more sugar than when another caregiver looked at the child. When another caregiver cared for the child, the child ate less fruit and vegetables than when the mother cared for the child. Of all the children in this study, 80% did not consume any fruit and 63% did not consume any vegetables. Malnourished children

consumed significantly more unhealthy foods such as sweets, cakes, cool drinks, chocolates, etc. Sugar was consumed by 85% of the children every day of the week and between 60-89% of the children ate sweet, cookies, cool drinks, etc every day.

In study undertaken by Mamiro *et al*. (2005) in Tanzania amongst children six months to two years, complementary foods usually consisted of a thin maize porridge. In Ethiopia, Getaneh *et al*. (1998) found that diets that are nutritionally inadequate, especially regarding animal food were associated with PEM. Children in India also often had a low intake of green leafy vegetables and fruits (Singh, 2004). These children had a deficiency of green leafy vegetables in 87%. When Iqbal Hossain *et al.* (1999) looked at the intake of Indian children six to 36 months old they found that children had a high intake of starches, sugar, fats and oils and lower intakes of vegetables and fruits (Iqbal Hossain *et al.,* 1999). Kapur *et al*. (2005) undertook a study in Delhi and looked at food intake. Cereals, milk, fruits and sugar were some of the foods that were most preferred (Kapur *et al*., 2005).

This study also found that even though children were not consuming sufficient amounts of milk for their age, 31% of the children also had insufficient water intake. All, except two children did not drink any water. In contrast, some of the children drank more than five bottles of tea per day. Except for the sugar and unhealthy foods consumed by children, tea can cause children to feel satisfied and cause them to not consume adequate amounts of nutritious food.

CHAPTER 6: CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

The results of this study indicated that the main factors that were associated with a child becoming malnourished were:

Socio-demographic information:

- The households that are prone to having malnourished children are either on farms, in rural areas or in informal urban settlements.
- Malnutrition is more prevalent in children between twelve to 36 months of age
- Other factors contributing to malnutrition are low birth weight as a result of maternal illness or poor maternal nutrition during pregnancy, lifestyle choices of the mother such as alcohol use and age of the mothers, especially nineteen to 25 years of age.
- Mothers still have a very low or no level of education and this leads to poor care practices and low socio-economic status with resultant stunting. Educated mothers are more likely to give more nutritious food than their uneducated counterparts.
- Marital status of the mother is associated with PEM, with single mothers experiencing more economic hardships.
- Significantly more boys than girls in this study presented with malnutrition.

Anthropometric information:

- Low birth weight babies were more prone to developing malnutrition or were born with a compromised nutritional status. One third of babies included in this study were born with a low birth weight.
- MUAC was found to be a very good screening tool.
- A mother/caregiver's size is directly associated with a child becoming malnourished. Underweight (BMI less than 18.5), as well as obese or overweight mothers both have a higher chance of having malnourished children.

Household information:

- Household size was directly linked to a child becoming malnourished, with a household of five to nine people being at higher risk.

- The majority of the households had a high room density of more than two people per room; this results in a high risk for the development of infectious diseases. There was a close to significant association between the prevalence of marasmus and a high room density.
- In most cases the mother was the head of the household and in some of the other households either the grandmother or grandfather was the head of the household.
- Even though half of the households had the father as the breadwinner, in other households the father was unemployed.
- In a large percentage of households the mother worked and was unable to take care of her own child.
- Other environmental or household factors that were linked to malnutrition were the type of house (most children lived in informal housing), the fuel used for cooking purposes (those with electricity were less likely to become malnourished).

Maternal information:

- In the majority of cases the mother was still alive and the children stayed with their parents and were cared for by their mother's during the day. If the parents were not caring for the children, they were usually cared for by the grandparents.
- Birth order seemed to be linked to malnutrition with most children included in this study coming from homes with a high birth order (four or more).
- One in ten mothers had lost a child to death. Mothers that had previous children, who had died, had a higher risk of having a child that is malnourished, especially if the sibling died from malnutrition or illness related issues, such as diarrhoea.
- Even if the malnourished child had not lost a sibling to death, the majority of their siblings had been admitted to hospital due to reasons ranging from respiratory disease, gastroenteritis, TB, sores in the mouth, malnutrition and other chronic diseases.

Maternal medical information:

- VCT had been accessed by 70% of the mothers in the study.
- Significantly more mothers had HIV and TB compared to the mothers that were uninfected. Even though a third of the mothers included in this study were HIV infected and one in five had TB, those that were infected were either still healthy or receiving HAART, PMTCT or TB treatment.
- Antenatal clinic attendance was generally acceptable.

- Maternal alcohol use and smoking were two lifestyle choices that were associated with child malnutrition.
- The majority of mothers smoked while they were pregnant.
- Significantly more mothers did not consume alcohol during pregnancy than the mothers that did consume alcohol. Of those that did consume alcohol during pregnancy, there was a close to significant association between the amount of alcohol consumed and the development of malnutrition in the child. .

Medical history of the child:

- Marasmus and wasting were the forms of PEM that were most prevalent in this sample.
- One fifth of children in this study had a low birth weight and were born in a health facility.
- Although all children had RtHC (one of the inclusion criteria), significantly fewer cards were completed correctly compared to the cards that were filled in correctly. Weight was not always plotted and the dots connected, therefore children could not always be evaluated correctly according to the growth curve.
- Even though all the children were reported to have cards, one third of cards were left at home.
- Clinic attendance was a major challenge with some children visiting clinics irregularly and some even visiting the clinic more than a year ago. Time between visits was generally too long. There was no significant difference between clinic attendance of children with kwashiorkor and marasmus.
- Most mothers only took their children to the clinic when it was time for immunizations or if the child was sick. Routine growth monitoring was not done as regularly as it should have been done.
- Significantly more children were up to date with their immunizations compared to the children that had outstanding immunizations.
- Despite the National vitamin A supplementation programme, significantly more children were behind on their vitamin A supplementation compared to the children that had received all their vitamin A dosages.
- Some children had other diseases, such as cerebral palsy and liver and heart disease, which made them more susceptible to becoming malnourished.
- Even though only one third of the children had been diagnosed with HIV infection, many had not been tested and their status was unknown.

- Most children had not been screened for TB, despite significant contact with TB infected persons.
- The main focus of the NSP is to support and treat children with malnutrition. Despite this, a large percentage of children with malnutrition admitted to hospital had been on the NSP for up to eight months or longer. This seems to indicate that the successful follow-up and implementation of the NSP is a challenge at health facilities.
- Significantly more malnourished children had been admitted to hospital on previous occasions, compared to the children that were admitted for the first time during the study. Of the children previously admitted, more than half had been admitted due to diarrhoea. Some children had been admitted on two to three previous occasions.

Biochemical information:

- Even though all blood values were not available for all the children, more than half of those for which they were available were anaemic.
- Of the children with an available C-reactive protein value, 70% showed the presence of an infection.

Maternal education:

- Nutrition information received by the mother regarding basic information necessary to care for children was very low in most mothers.
- Most of the mothers received insufficient health information at the clinic. Only 15% had received information on food fortification, 25% on diarrhoea, 34% on the RtHC, 51% on basic hygiene, 49% on healthy eating habits, 62% on breastfeeding and 49% on complementary feeding.
- Most of the information given at clinics was related to breastfeeding, eating habits, complementary feeding, and hygiene. Despite this, all the children were in hospital due to malnutrition, and therefore the effectiveness of the information given by health care professionals at clinics is questioned.

Infant feeding information:

- Even though most children were breastfed, the rate of exclusive breastfeeding in this study was very low. After six months the rate of breastfeeding decreased

further and only a small percentage of mothers were still breastfeeding at two years of age.

- Except for the mothers that did not breastfeed exclusively for six months, some mothers practiced exclusive breastfeeding for too long. This may also contribute to malnutrition because the breast milk is not sufficient to meet the requirements of an infant older than six months old.
- Marasmic children were breastfed up to a median age of four months and children with kwashiorkor were breastfed for a median age of three months, but the difference was not statistically significant.
- Mothers were still giving their children expensive alternative milk sources such as formula milk, Nido and Nespray rather than cow's milk if they weren't breastfeeding. Significantly more children consumed alternative milk products than cow's milk.
- Only one in twenty children received sufficient milk for their age if they weren't breastfed.
- Cup feeding was not practiced widely and most mothers were using bottles to feed milk to their babies, which may cause diarrhoea if not handled hygienically.
- Even though mothers reported that they prepared milk hygienically, it was difficult to confirm this because the study was not designed for the mothers to demonstrate how they prepared the milk.
- Some mothers introduced water as early as one month of age and most started solids before the baby was three months old. On the other hand, there were mothers that only started solids at the age of seven to twelve months or even later. No significant difference was found between the prevalence of marasmus and kwashiorkor and when solids were introduced.

Food Based Dietary Guidelines:

- Very few children consumed a variety of foods as evidenced by the fact that fruits and vegetables were not consumed every day.
- All the children used starch as the basis of their meals as all children ate porridge. Some items were added to the porridge such as milk, sugar, margarine, purity, peanut butter and yoghurt.
- Bread was the staple food bought by all households. The intake of white and brown bread was more or less equal.
- Animal proteins were only consumed every day of the week by half of the children.

- The FBDG states that children can consume alternative protein sources such as beans, lentils, split peas, and soy often. This was not done as evidenced by the fact that only three out of four children ever ate products such as soy mince and baked beans.
- Even though the FBDG guide recommends that children eat fruit and vegetables every day, the evidence showed that only one in five ate fruit every day and only one in three ate vegetables every day.
- The guideline "use salt sparingly" was not applied as evidenced by the fact that only seven children did not use added salt in their food, and the other children ate added salt, soup powder, beef stock blocks, steak and chop and chicken spice and aromat in their food.
- The FBDG guidelines state that fats should be used sparingly. This guideline was not applied as evidenced by the fact that fats, oil and margarine were used by all the mothers/caregivers in the preparation of the children's food.
- High intakes of sugar and sugary products were common even though the FBDG state that sugar and sugar containing food should be used sparingly.
- The consumption of tea compromised the intake of milk. High tea intakes resulted in lower intake of food because the children were less likely to become hungry.
- Most children applied the guideline "drink, clean, safe water everyday" as the evidence showed that only two children did not drink water.

6.2 RECOMMENDATIONS

According to the United Nations Millennium Summit held in 2000 there are seven goals for preventing malnutrition. These include universal primary education, empowerment of women, improved maternal health, decreased child mortality, prevention and management of HIV and AIDS, malaria and other infectious diseases, improvement in the environment and worldwide partnerships for development of countries (Müller and Krawinkel, 2005). The seven goals led to the development of the Millennium Development Goals (Müller and Krawinkel, 2005). Of the world's undernourished children 80% live in just 20 countries and intense nutrition action is needed to achieve MDG1 (eradicate extreme poverty and hunger) and can also increase the chances of achieving MDG4 (reduce child mortality) and MDG5 (child and maternal mortality) (Bryce *et al.,* 2008; UNICEF, 2009c, p.10-11),

Nutrition should be a priority at national and regional levels because it is important for human and social development and in the long-term undernutrition can also affect a country's socio-economic development (Bryce *et al.,* 2008; UNICEF, 2009c, p.10-11). Many of the MDG's, particular MDG1, MDG4 and MDG5 can and will not be reached if women and child health and nutrition is not nationally seen as national priority when strategies and programmes are developed (UNICEF, 2009c, p.10-11).

Malnutrition has many contributing factors and interventions should consist of multi-sectoral and holistic programmes (Müller and Krawinkel, 2005). In addition to addressing the immediate and basic factors, interventions should also consider the underlying factors of malnutrition, such as social norms, gender and equity, maternal access to education and health care and household food and nutrition security (UNICEF, 2009).

Interventions should be implemented at all levels; international, national and regional, as well as at household level (FAO, 1996). Government policies should reflect the right to nutrition at all levels (Jones, 1998). Nutritional status can be improved if interventions are implemented and sustained at high levels (Bryce *et al*., 2008).

6.2.1 IMMEDIATE FACTORS

Recommendations on breastfeeding practices, infant and young child feeding practices, food aid and supplementation, food fortification and the management of infectious diseases will be discussed under immediate factors contributing to malnutrition.

If programmes do not exist yet, developing countries can learn from successful countries on the implementation of intervention programmes to combat malnutrition (UNICEF, 2008; UNICEF, 2009c, p.7).

6.2.1.1 PROMOTION OF BREASTFEEDING

Even with the poor conditions in developing countries, breastfed children have a six times higher chance of survival in the early months than non-breastfed children. Breastfed babies are also six times less likely to die from diarrhoea and 2.4 times less likely to die from acute respiratory infection (UNICEF, 2009c, p. 13).

Breastfeeding can significantly reduce under five deaths by 13% if the coverage of breastfeeding is increased to a universal coverage of 99% (Jones *et al*., 2003). In 1998,

12 700 hospitals in 114 countries were classified as Baby Friendly through the Baby Friendly Hospital Initiative, which is the basis of a good start to breastfeeding for millions of babies (State of the world's children, 1998).

Optimal infant and young child feeding is based on early initiation of breastfeeding within one hour after birth. Exclusive breastfeeding should then continue for the first six months of life (Labadarios *et al.,* 2005a; Labadarios *et al*., 2005b; UNICEF, 2009c, p. 13) and continue for up to two years or longer, while nutritionally adequate and safe complementary foods are introduced (UNICEF, 2009c, p. 13 and 23).

South Africa is voluntarily participating in the WHO International Code of the Marketing of breast milk substitute. In South Africa the code is called the South African Code of Ethics for the Marketing of breast milk substitute (South African Code) (Hendricks *et al*., 2006). Promotion, protection and support of breastfeeding should also concentrate on the promotion of the Baby Friendly Hospital Initiative, the Code for marketing and distribution of breast milk substitutes and Infant feeding options for HIV infected mothers (Labadarios *et al*., 2005a; Labadarios *et al.,* 2005b).

6.2.1.2 INFANT AND YOUNG CHILD FEEDING PRACTICES

Optimum feeding of infants and young children is important for health, growth and development. Good feeding practices prevent malnutrition, early growth retardation and reduce the severity of infections (Fuchs *et al.,* 2004). Correct complementary feeding can reduce stunting prevalence in first two years (UNICEF, 2009c, p.26), because this is the period when growth faltering and malnutrition usually occur (Fuchs *et al.,* 2004). In this time the appropriate use of complementary and traditional foods are important issues (Torún, 2006, p.906).

In South Africa education and advocacy regarding the feeding of children and infants is done through the Infant and Young Child Feeding Policy (Labadarios *et al*., 2005b). The period from pregnancy to 24 months of age is an important time and the ideal time for interventions in reducing malnutrition and the adverse effects thereof (Baker-Henningham and Grantham-McGregor, 2004, p.256; Bryce *et al*., 2008). Interventions should therefore include adequate feeding during pregnancy, early initiation of breastfeeding and correct complementary feeding (UNICEF, 2009c, p.31).

Infant feeding should not only be the responsibility of the mother. Programmes should address support and approval of the male partner and the maternal grandmother (Fuchs *et al.,* 2004). Interventions directed to parents or caregivers, should include nutrition counselling on feeding and care practices, the use of locally available food, improved access to quality foods through grants, the distribution of micronutrients and macronutrient supplementation (UNICEF, 2009c, p.26). Animal foods are the best protein sources, but often expensive and unavailable, or prohibited due to religious practices. Therefore staple vegetable foods must be complemented with protein vegetable foods in such a way that it is culturally acceptable (Torún and Chew, 1994, p.973; Torún, 2006, p.905).

Beliefs influencing infant feeding include socio-cultural beliefs about infant feeding (such as withholding food during diarrhoea), traditional healthcare practices, influence of family and friends and other commercial pressures (Fuchs *et al.,* 2004). Programs should not only look at improving a child's nutritional status, but also at solving developmental problems that are present. These programs must be combined with health care, psychosocial stimulation and parental education activities (Baker-Henningham and Grantham-McGregor, 2004, p.256).

6.2.1.3 SUPPLEMENTATION PROGRAMMES

In 1997 the lives of about 300 000 children were saved by vitamin A supplementation in developing countries (UNICEF, 1998), but in 2005 the vitamin A status of South African children was still poor (Labadarios *et al*., 2008). Supplementation with vitamin A can reduce the risk of child mortality from all causes by about 23%. High doses for 6-59 months twice per year are one of the most cost-effective nutrition interventions available (Labadarios *et al*., 2005b; UNICEF, 2009c, p.13). Facilities should be aware of the correct storage of vitamin A capsules to ensure that their efficacy is not compromised (Labadarios *et al*., 2008). Children that are older and not attending clinics are missing out on the vitamin A supplementation programme and therefore schools and crèches should be prioritised within promotion programmes.

Supplementation with vitamin A and a multivitamin is directly linked to attaining MDG4 (UNICEF, 2008). Noting of Vitamin A supplementation should be added to the RtHC in the same way as immunizations, to ensure that mothers understand the importance of the supplementation and to ensure that the supplement is administered correctly (Labadarios

et al., 2008). Except for supplementation, there should be constant promotion on the advantages of vitamin A (Hendricks *et al.,* 2006) and how to enrich complementary foods with vitamin A rich foods (Labadarios *et al*., 2008).

In addition to vitamin A, the iron status of children should be assessed regularly (Baker-Henningham and Grantham-McGregor, 2004, p.258-259). The iron status of children and women is still poor with one in three being anaemic (Labadarios *et al*., 2008). Interventions should also look into preventing iron deficiency anaemia by adding iron as one of the micronutrients used for the fortification of food staples and complementary foods in both developed and developing countries (Baker-Henningham and Grantham-McGregor, 2004, p.258-259). Supplementation with micronutrients can reduce anaemia by 45% (UNICEF, 2009, p.27). Once anaemia is confirmed, children should be supplemented with iron sulphate syrup from 6-23 months for at least three years (Labadarios *et al*., 2008).

6.2.1.4 FOOD AID PROGRAMMES

Diet based strategies are probably the most promising approach for the sustainable control of micronutrient deficiencies (Müller and Krawinkel, 2005). Interventions aimed at preventing malnutrition can be implemented through food supplementation schemes, food-based strategies such as home gardens and small livestock and income generation, nutrition education and maternal support (Müller and Krawinkel, 2005).

Children can present with deficiencies of more than one micronutrient at one time. Therefore children that present with one micronutrient deficiency can also have a deficiency of another micronutrient (Ferraz *et al*., 2005), therefore it is important to give food with a high content of absorbable micronutrients as part of food aid programmes. A broad variety of food from home gardens and small livestock production is effective for diet diversification. Households must be educated and supported to increase production of dark green leafy vegetables, yellow and orange fruit, poultry, eggs, fish and milk (Müller and Krawinkel, 2005).

In South Africa, food aid for HIV and AIDS, TB and malnourished children includes a macronutrient meal (such as Philani and Philani Yabantwana) and micronutrients (such as a multivitamin mineral tablet) (Labadarios *et al.,* 2005a; Labadarios *et al.,* 2005b). Entry and exit criteria for receiving macro- and micronutrients must be used for effective

control (Labadarios *et al.,* 2005a). Patients or clients can take part in these supplementation schemes with the help of social sector partners through referrals, monitoring and evaluation (Labadarios *et al*., 2008).

Challenges regarding the nutrition supplementation programme include a lack of staff, inadequate coverage and targeting of malnourished children, a high defaulting rate, incorrect distribution of supplements, ineffective counselling of mothers and caregivers on the use of the products and a lack of integration with all other nutrition programmes (Hendricks *et al.,* 2006). These challenges need to be addressed to ensure the successful implementation of the programme.

6.2.1.5 FOOD FORTIFICATION

In 1999 the NFCS survey was undertaken to determine the nutritional situation of children 0-9 years and to use these findings to help decide what interventions are needed in South Africa. Based on the results of this survey it was determined that children are deficient in a number of micronutrients (NFCS, 1999; Labadarios *et al*., 2005b) and in October 2003 it became legislation that staple food such as maize and wheat be fortified with vitamin A, riboflavin, niacin, pyridoxine, folic acid, iron and zinc to provide the DRI for children ten years and older (Every death Counts, 2008). After the fortification programme was introduced, neural tube defects in South Africa decreased by one third (Every death Counts, 2008).

Micronutrient malnutrition control should be implemented through a combination of strategies such as vitamin A supplementation, food fortification and iodisation of salt (Labadarios *et al*., 2005a). The management of iodine deficiency through food fortification (Labadarios *et al*., 2008) has had a significant impact on iodine deficiency in South Africa. In 36 countries, 90% of households use iodised salt, but globally 41 million of people can still develop brain damage due to an iodine deficiency (UNICEF, 2009c, p.27). Salt iodisation should therefore be part of health programmes (WHO, 2007b), except in the Northern Cape where the iodine content of the water is very high and the local municipalities should address this matter (Labadarios *et al*., 2008).

6.2.1.6 MANAGEMENT OF INFECTIOUS DISEASES

Undernutrition is the biggest cause of deaths related to severe infections; therefore the early and correct management of infectious diseases and prevention of undernutrition

should be a priority (Caulfield *et al.,* 2004; WHO, 2001; Müller and Krawinkel, 2005). In poor communities, the treatment of helminth infections with deworming tablets three times per year has been shown to improve child growth and development (Müller and Krawinkel, 2005). An increase in measles admissions in Nigeria has been directly linked to an increase in the prevalence of kwashiorkor (Oyelami and Ogunlesi, 2007).

6.2.1.6.1 DIARRHOEA

Persistent diarrhoea seriously affects nutritional status, growth and intellectual functions, especially in developing countries. Diarrhoea and other childhood diseases can also lead to deficiencies of vitamin A, zinc, folic acid, copper and selenium, as well as a low immune status (Ochoa *et al*., 2004). Management of persistent diarrhoea includes rehydration with oral rehydration solution (UNICEF, 2009, p.31), adequate diet, micronutrient supplementation and anti-microbial medication. Exclusive breastfeeding for the first six months of a baby's life can protect against acute and persistent diarrhoea and therefore promotion of exclusive breastfeeding is an important preventative intervention (Ochoa *et al*., 2004).

Improved home management of childhood diarrhoea can lead to a decrease in children admitted to hospital (Oyelami and Ogunlesi, 2006). Priority must be given to health education on home management of diarrhoea with oral rehydration solution to combat severe dehydration (Torún and Chew, 1994, p.974; Oyelami and Ogunlesi, 2006; Torún, 2006, p.906). Together with oral rehydration solution, the correct feeding of a child with diarrhoea should also be emphasized (Torún and Chew, 1994, p.974; Ochoa *et al.,* 2004; Torún, 2006, p.906). Children who do not receive health facility based treatment of diarrhoea are about two times more likely to be exposed to malnutrition than those that did get treatment (Abate *et al*., 2001).

Using elemental diets for the management of diarrhoea in both HIV infected and uninfected children can lead to a higher weight and haemoglobin, but not albumin (Amadi *et al*., 2005). Diarrhoea should also be treated with zinc supplementation, as supplementation can reduce the prevalence of diarrhoea by 27% by reducing the duration and severity of the diarrhoea (UNICEF, 2009c, p.31). Dietary treatment with probiotics can help to clear up diarrhoea within five days in 85% of children (Ochoa *et al*., 2004).

6.2.1.6.2 HIV, AIDS AND TB

An important cause of death in children is HIV infection (Every death counts, 2008). VCT should be available for all malnourished children and their mothers (WHO, 2007a). Newly diagnosed HIV positive children should receive cotrimoxazole prophylaxis to decrease the risk of contracting opportunistic infections and should be referred for ART after being assessed (WHO, 2007a).

Children who are HIV infected and develop PEM should receive food aid to improve their nutritional status (WHO, 2007a). Nutritional support has a positive effect on immune function, quality of life and bioactivity of ART (Fenton and Silverman, 2008, p.1008). In South Africa, the National Nutritional guidelines for people living with HIV and AIDS were compiled to improve the nutritional status and support of children and adults with HIV infection (Labadarios *et al*., 2005b).

6.2.1.7 MANAGEMENT OF SEVERE ACUTE MALNUTRITION

MDG4 (reduction in child mortality) can only be reached if the management of SAM is implemented correctly (UNICEF, 2008). Implementation of the WHO 10 steps for the management of severe malnutrition can make a significant impact on improving child survival (Jackson *et al*., 2006; Ashworth, 2004).

Pediatricians should be kept up to date on how to manage malnutrition. In 2005 the International Union of Nutritional Services launched a Task Force on malnutrition. This was done with the support of the International Paediatric Association. The objective of the Task Force was to look at technical expertise and capacity building on treatment with the help of partners, to promote malnutrition as an important strategy for child survival among policy makers, to advocate for adding malnutrition to the medical and nursing curriculum, to encourage health workers to keep track of their performance and to raise resources (Jackson *et al*., 2006). To manage SAM effectively all levels of care and decision-making should be expanded as shown in Figure 6.1.

Weaknesses in the training, supervision and support of doctors and nurses contribute to eight out of ten deaths in children with severe malnutrition. Deaths in the Eastern Cape, South Africa often occur due to sepsis that is not treated with antibiotics and dehydration that is managed incorrectly and leads to overhydration. This can be prevented by giving

training to clinic staff, out patient staff and community health workers who can also assist with follow ups (Ashworth, 2004).

In the acute phase of the management of malnutrition the immediate threats must be addressed. Treatment is aimed at reversing the physiological changes without overloading the reduced capacities of the heart, kidney, intestine or liver. In the intermediate phase, energy and nutrients are given in amounts just a little above the maintenance requirements to correct metabolic abnormalities (Golden and Golden, 2000 p.525).

In the rehabilitation phase, the appetite of the child will determine the progress of the process and attention should be given to emotional and psychological needs before the child is discharged. At the end of this phase up to discharge, the parents and caretakers should be educated on rehabilitation and preventing recurrence (Golden and Golden, 2000 p.525). The recommended criteria used to discharge a child from nutritional rehabilitation are a weight- for-length of –1SD (90%) of the median NCHS/WHO standard (Fuchs *et al*., 2004).

Efforts should be made to look at treatment that can lead to shorter hospital stays and where children can be discharged into a home and community based management programme (Fuchs *et al*., 2004). Outpatient programmes can decrease barriers to access, encourage early identification of malnutrition, reduce inpatient caseloads and so decrease the risks of cross infection, reduce costs associated with treatment, encourage compliance by patients and increases the time available to staff to help the sickest children (Fuchs *et al*., 2004; Collins *et al*., 2006).

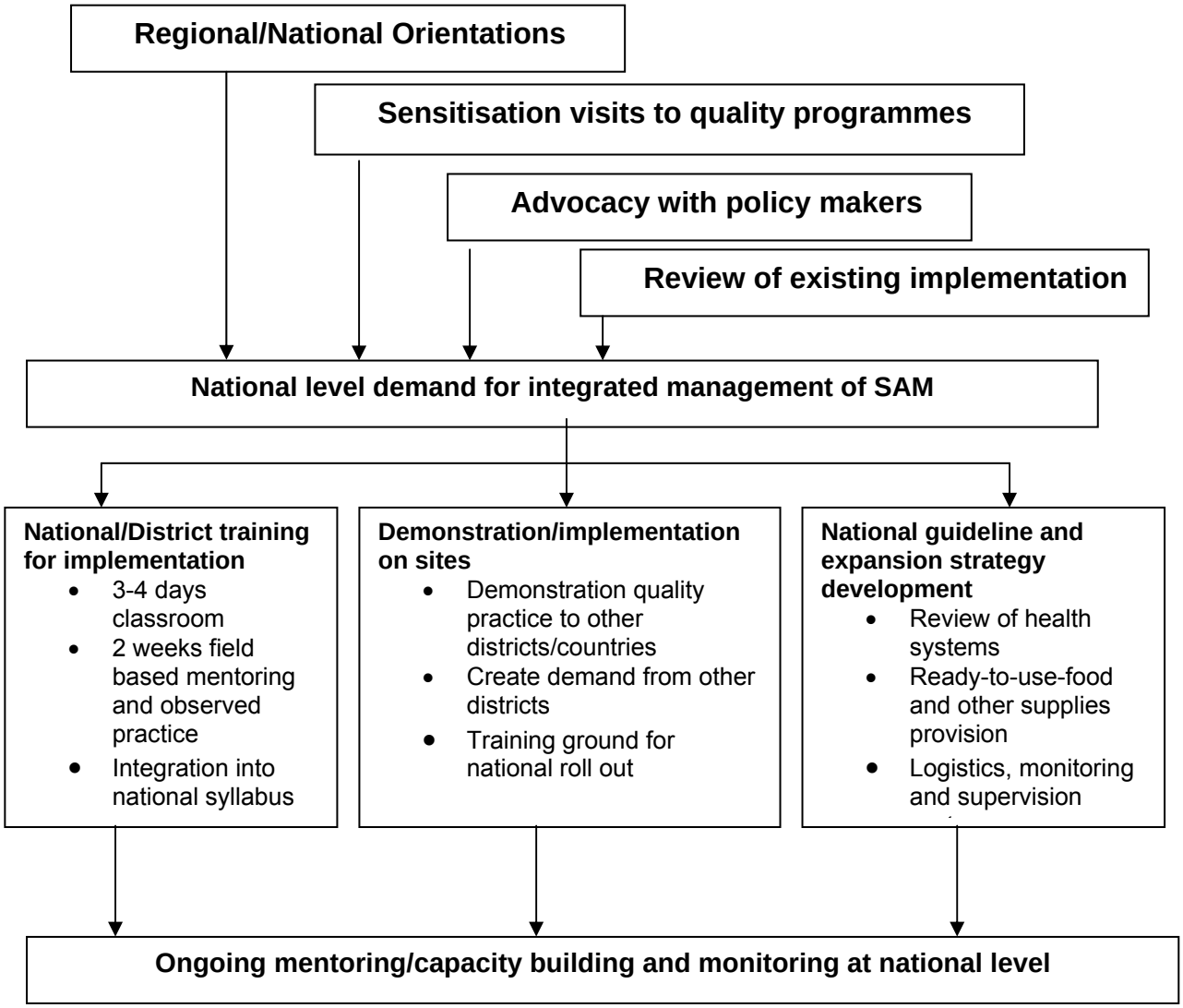
These new approaches have the potential to reduce case fatality rates and increase coverage rates (Collins *et al*., 2006). Nutritional interventions for health facilities for the prevention and management of childhood malnutrition must be a priority intervention (Labadarios *et al*., 2005a; Labadarios *et al.,* 2005b) and governments should look into the possibility of establishing nutrition rehabilitation centres were children could be followed-up after discharge (Labadarios *et al*., 2008).

WHO treatment guidelines for the management of severe malnutrition should therefore be improved and simplified so that they can be effectively used in poor communities or

settings. The basic principles of the WHO guidelines should, however, never be compromised (Fuchs *et al.,* 2004).

Interventions for the management of SAM at community level should look at the use of ready-to-use-foods, treatment of complications and the management of moderate malnutrition (UNICEF, 2009c, p.31).

Figure 6.1 Steps to expand the capacity for the management of SAM (UNICEF, 2008)



6.2.2 UNDERLYING FACTORS

Recommendations related to underlying factors contributing to malnutrition, including health care services, personnel and skills development, growth monitoring and promotion, immunizations, hygiene and sanitation, community and maternal education and household factors will be discussed in the following section.

6.2.2.1 HEALTH CARE SERVICES

The provision of effective, basic, preventative and curative primary health care services for children is an essential component of the government response to malnutrition (Labadarios *et al*., 2005a). It is policy makers' responsibility to provide adequate facilities in appropriate sites, maintain equipment and drugs, provide adequate staff, ensure positions are filled and guarantee adequate transport between institutions (Every death counts, 2008).

Disease specific nutritional support, treatment and counselling include nutritional and dietary practices for the prevention and rehabilitation of nutrition-related diseases and illnesses through counselling, support and management. Assessment and screening, education and counselling, nutritional therapy and prevention are important activities that should form part of basic health care services at primary, secondary and hospital levels (Labadarios *et al.,* 2005a).

6.2.2.1.1 PERSONNEL AND SKILLS DEVELOPMENT

Health care providers should have the correct and appropriate skills to perform the functions that are expected of them and should maintain the skills necessary for their profession. They must also use these skills to care for patients in a respectful manner (Every death counts, 2008). Amongst others, skills must be developed in addressing vitamin A deficiency, growth monitoring and promotion, management of SAM and foetal alcohol syndrome (Hendricks *et al.,* 2006).

Health care workers also need to successfully identify and manage malnourished children. Priorities must include the improvement of training materials to ensure that policies are implemented correctly and that IMCI protocols are used correctly. Policies and guidelines must be implemented in such a way that problems are identified and interventions started early enough to make a difference (Hamer *et al.,* 2004).

Behaviour change communication is important for nutrition interventions to be effective (WHO, 2007b). An adequate number of health care workers are necessary for the promotion and education on breastfeeding, nutrition counselling and growth monitoring (Hendricks *et al.,* 2006).

6.2.2.1.2 GROWTH MONITORING AND PROMOTION

Growth monitoring and promotion are integral parts of public health care services and provide valuable information regarding nutritional status of children and infants (Labadarios *et al.,* 2005a). Despite growth monitoring being performed, a lack of follow-up occurs frequently (Müller and Krawinkel, 2005). Growth monitoring and promotion should be incorporated into all health care programmes (WHO, 2007b) and practices should be improved among public health centre nurses (Hendricks *et al*., 2006). Growth monitoring and promotion should also be promoted as a community-based intervention (Labadarios *et al*., 2008).

Methods of assessing malnutrition should identify those at risk, must be simple to apply and be internationally accepted. In children, height-for-age, weight-for-height and MUAC are most useful (Golden and Golden, 2000, p.525). MUAC is effective in community-based interventions and programmes because it is an indicator of acute malnutrition and shows risk of mortality. It is also easy to use and can be implemented in poor communities by community workers for screening purposes (Collins *et al*., 2006; UNICEF, 2008).

Length should also be measured at facilities to screen for stunting and provinces should ensure that all needed equipment is available. The new WHO standards for height-for-age and BMI should be available and form part of the RtHC (these are currently being added to the new RtHC that will be implemented in 2010) (Labadarios *et al*., 2008).

6.2.2.1.3 IMMUNIZATIONS

An effective prevention intervention for nutrition and health is high vaccination coverage against diseases (Abate *et al.,* 2001; Müller and Krawinkel, 2005; Torún, 2006, p.905) and infections leading to deaths can be fought through immunizations (UNICEF, 2009c, p.31).

Improvement in immunization coverage is one of the South African Primary Health Care successes, with 84% of infants currently being fully immunized with BCG, hepatitis B, polio, DPT3-hib, measles. More recently, immunizations against Hepatitis B and immunizations against Hemophilus influenzae type B (HiB) infections have been introduced (Every Death Counts, 2008).

There are however, still a lot of children not being brought to facilities for immunizations and communities need to be targeted to reach 100% coverage.

6.2.2.2 HYGIENE AND SANITATION

Infectious diseases can be prevented through the implementation of environmental programmes such as improving access to sufficient quantities of safe and clean drinking water, sanitation as well as improved personal and domestic hygiene (WHO, 2001; Bradshaw *et al*., 2003; UNICEF, 2009c, p.31). In addition, comprehensive primary health care, hand washing (UNICEF, 2009c, p.31), reductions in exposure to indoor smoke (Bradshaw *et al*., 2003) and improvements in roads are other interventions that can improve hygiene and sanitation (Torún, 2006, p.905). Hygiene practices are directly linked to the prevention and management of MDG4 (UNICEF, 2008).

Other risk factors that can lead to nutritional problems or infections are the presence of children's faeces inside the house, failure to treat diarrhoea at a health facility, storage of cooked foods for longer than 24 hours, feeding children with unwashed hands, poor handling of drinking water and foods (Abate *et al*., 2001). Hygienic environments and personal hygiene should be a top priority to reduce infections and improve health.

6.2.2.3 EDUCATION

Findings from the NFCS of 1999 contributed to the implementation of the vitamin A supplementation programme, HIV and AIDS and TB nutritional intervention policies, as well as the FBDGs. These dietary guidelines provide communities with the necessary knowledge to reach their nutritional goals. The eleven adopted FBDGs focus on existing nutrient deficiencies and overnutrition, nutrition related public health initiatives and also cultural differences and eating patterns. The guidelines look at affordable, inexpensive and available foods consumed by communities and therefore encourage sustainable agricultural practices, such as the planting of food gardens (Labadarios *et al.,* 2005b). Wider adoption of these guidelines into the primary health care setting should be encouraged.

In the NFCS-FB-1 of 2005, Labadarios *et al*. (2008) reported that mothers received health information via health professionals, television and school children. Cell phones were also widely available and technology can be used to remind parents to bring their children for vitamin A supplementation (Labadarios *et al*., 2008).

6.2.2.3.1 COMMUNITY EDUCATION

Community nutrition education in poor urban communities is essential (Abate *et al*., 2001; Labadarios *et al*., 2008), especially if there are children with PEM present in a community (Golden and Golden, 2000, p.525). All educational programs should include the communities' own assessment of their nutritional problems and how to solve these problems (Torún and Chew, 1994, p.974; Abate *et al*., 2001; Torún, 2006, p.906), as well as the availability of food in the community, preferences and culture (Fuchs *et al.,* 2004). When education regarding infant and young child feeding is given to communities, the types of food and the way it is delivered to make sure that the food reaches and benefits the children that need it should be given priority (Fuchs *et al.,* 2004).

Other issues to discuss with communities are the correct use of weaning foods, using traditional foods, environmental and household hygiene through covered water and food and keeping the house free from faecal material. Personal hygiene through hand washing before eating to prevent communicable diseases, feeding practices during illness and recovery, and treatment of diarrhoea and other preventable diseases are also important issues to address (Torún and Chew, 1994, p.974; Abate *et al*., 2001). Agricultural interventions, health knowledge and practices, safe drinking water and sanitation are particularly important issues as are improved access to high-quality foods (Müller and Krawinkel, 2005; WHO, 2007b).

Community and family involvement can avoid many deaths by making sure families are equipped with appropriate healthcare messages (Every death counts, 2008). Communities must also be aware of how to improve school meals and how to interpret food labelling (WHO, 2007b). Staff at schools and crèches should be educated regarding healthy foods in tuck shops, physical education healthy eating and meals (Labadarios *et al*., 2008).

In South Africa, opportunities already exist for improving health through current programmes such as the Expanded Program of Immunization, antenatal care, the Integrated Child Health Campaigns and the Community IMCI (Shoo, 2007).

6.2.2.3.2 MATERNAL EDUCATION

The improvement of child and maternal nutrition is a feasible, affordable and cost-effective intervention against malnutrition (UNICEF, 2009c, p.7). A community based

nutrition programme for young children, adolescent girls and pregnant women should be a medium term intervention for countries (WHO, 2007b), seeing as pregnant women and young children are often the most vulnerable groups in a community (Müller and Krawinkel, 2005).

Special efforts should be made to uplift woman as primary child carers, with particular reference to health and nutrition throughout the life cycle. Attention should be given to complementary feeding and to the protection and promotion of breastfeeding (De Onis *et al*., 2000).

A critical time period to prevent malnutrition is during the time that a mother is pregnant and during a child's first two years of life (UNICEF, 2009c, p.7). Evidence shows that nutrition interventions implemented at this time can contribute to reaching optimal growth and development (Hendricks *et al*., 2006). Mothers should also receive information on where to get support if they need it (Müller and Krawinkel, 2005), especially regarding the empowerment of women, social security for female-headed households, old age grants and child grants (Labadarios *et al*., 2008).

In an effort to prevent malnutrition, postnatal care needs to be strengthened. HIV coverage interventions are lowest during and after birth, especially in rural areas (Every death counts, 2008). Education should also include maternal and childcare, PMTCT and treatment of childhood diseases, especially during antenatal and postnatal care and follow-ups (Bradshaw *et al.,* 2003; UNICEF, 2007).

During the stay at the hospital, the importance of food for child health (Pereira, 1991, p.148; WHO, 2001), the beneficial effects of breastfeeding and healthy weaning practices must be taught to mothers. Education should also be given on ways to use locally available cereals and proteins and how to adjust the energy density of these cereals through the addition of sugar and oil (Pereira, 1991, p.148).

Mothers should understand the importance of giving enough food and how to utilize available resources such as available edible green leaves, vegetables and fruit to prevent mineral and vitamin deficiencies. Parents should also receive education regarding the importance of stimulation and play (Play Therapy Africa, 2009). Mother should also receive instructions on family planning and child spacing. The importance of

immunizations and monthly growth monitoring and promotion (Pereira, 1991, p.148), as well as food supplementation should be taught to all mothers (Müller and Krawinkel, 2005).

6.2.2.4 HOUSEHOLD FACTORS

Sustainable food production and other agricultural interventions are important interventions to apply in an effort to improve household and school food security (Jones, 1998; Müller and Krawinkel, 2005). Increased household food production can improve food safety (Hendricks *et al*., 2006) and contribute to income generation (Müller and Krawinkel, 2005; Hendricks *et al*., 2006). Household food production through home gardens and small livestock production will ensure dietary diversification and the consumption of a broader variety of foods. Households should also be educated and supported to increase their production of dark green leafy vegetables, yellow and orange fruits, poultry, eggs, fish and milk (Müller and Krawinkel, 2005; Hendricks *et al*., 2006).

The Integrated Food Security Strategy aims to eradicate hunger, malnutrition and food insecurity by 2015. The vision is for all South Africans to have improved access to sufficient, safe and nutritious food for them to have active and healthy lives (WHO, 2001; Hendricks *et al*., 2006). This strategy has been implemented in partnership with Department of Agriculture in the fight against food insecurity (Labadarios *et al*., 2008).

6.2.3 BASIC FACTORS

6.2.3.1 POLICIES

Interventions to reduce malnutrition (Caulfield *et al*., 2004) and address the factors contributing to malnutrition (Torún, 2006, p.905) should be a policy priority (Caulfield *et al*., 2004). Resources can only be used effectively if programmes are implemented according to approved policies. If policy makers cannot recognize the urgency of malnutrition, they may not understand how improved nutritional status can affect national, economic and social goals (UNICEF, 2009c, p.11). National and regional levels can only implement effective, sustainable and long-term preventative measures if there is political commitment to addressing these problems at the highest level (Torún, 2006, p.905; UNICEF, 2009c, p.37))

Policies should incorporate capacity building at all levels, action plans for ways to reach goals and the monitoring and evaluation of interventions (UNICEF, 2008). Strategic and

operational capacity building is essential to ensure that there is adequate capacity of leadership and strategic management (Bryce *et al*., 2008).

Programmes and policies should be managed by making sure there are service delivery systems and resources available (UNICEF, 2009c, p.37). Policy makers and managers are the people that need to make sure that interventions take place through the provisioning of adequate facilities in communities, maintaining of equipment and drugs, human resources (positions needed to be filled) and adequate transport (Every death counts, 2008).

Policies should concentrate on priority programmes with specific emphasis on MDG4 (UNICEF, 2008) and the programmes such as the Direct Observed Treatment Short Course for TB (DOTS), NSP, PMTCT, IMCI and Integrated Nutrition Programme (INP) (Hendricks *et al.,* 2006; Every death counts, 2008). Policies should also take into consideration training for the improved management of PEM at all levels and should provide the resources needed for management of SAM by providing ready-to-use-food to those that need it, as well as enough resources for the free treatment of SAM because most of the affected families are often the poorest (WHO, 2007a).

Decisions regarding nutrition related issues should be based on effective collection of data, monitoring and evaluation. International data is, however, also important for guiding national policies and programmes. If no data is available on important interventions, countries will never know if the coverage excludes those that are really in need of programmes (Bryce *et al*., 2008). Currently availability of reliable data is still a challenge (Jackson *et al*., 2006).

It is critical that Governments have policies that will reach poor communities and this can be achieved with both the help from public and private sectors (Shoo, 2007). Except for health and nutrition interventions, economic and social policies that will be addressing poverty, trade, agriculture (Bryce *et al.,* 2008) and job creation (Labadarios *et al*., 2008) are needed to improve nutritional status (Bryce *et al.,* 2008). Regional / provincial levels should look into financial employment and training schemes for the youth and ensure that there is social protection for all (UNICEF, 2009).

6.2.3.2 POVERTY ALLEVIATION

Improvement in nutritional status of children depends heavily on improvements in the socio-economic status of families (Cartmell *et al*., 2005). The nutritional status of an individual depends on the food that is eaten, health of the individual and the physical environment. Malnutrition is both a medical and social disorder that is usually the end result of poverty (WHO, 2001). Poverty is therefore directly associated with the inadequate supply of food and development of malnutrition (Müller and Krawinkel, 2005).

In South Africa about 7 million children are accessing the social grant system. There are approximately 2.5 million orphans in South Africa and the number is rising annually as a result of HIV and AIDS (UNICEF, 2007). To reach the hunger- and malnutrition-related MDGs, countries need to address poverty (Müller and Krawinkel, 2005). Poverty alleviation is an important intervention required in the fight against malnutrition (Bradshaw *et al*., 2003). Intervention programmes should be integrated into all programmes of various government departments for the interventions to be effective (Müller and Krawinkel, 2005).

Poverty is directly linked to economic growth and poverty alleviation will undoubtedly have a significant impact on food insecurity and malnutrition. Even though economic growth is linked to poverty, economic growth is, however, not always the only cause of poor nutritional status and other immediate and underlying factors should be considered (UNICEF, 2009c, p.35).

6.3 FUTURE RESEARCH

Research needed in the field of the management of SAM include the following:

- the safety of infant formulas, such as F75 and F100 in the management of SAM, specifically for babies less than six months old
- the adjustment of ReSoMal to provide 75 mmol/L sodium or to reduce the WHO oral rehydration solution to 45 mmol/L (UNICEF, 2004);
- development of effective ready to use therapeutic foods in South Africa
- the effect of vitamin A supplementation in HIV infected women and breastfeeding (Labadarios *et al*., 2008).

Research priorities in the field of HIV, AIDS and ARVs include:

- the safe use of ARVs in children with severe malnutrition (UNICEF, 2004)
- safety of supplementation in HIV infected children, specifically with zinc, vitamin A and other micronutrients;
- DRIs for HIV infected children;
- the effect of diet on lipodystrophy in children on ART;
- addressing the problem of growth failure and malnutrition among HIV infected children in South Africa (Hendricks *et al*., 2006);
- and which assessments should be used in the evaluation of HIV infected children, younger than six months old (UNICEF, 2004).

According to Labadarios et al. (2008) an important area of research that should be supported is the repetition of the NFCS every three to five years to establish whether intervention programmes are making a difference. Furthermore, a panel of experts should be established to evaluate the complementary foods used in the supplementation schemes to determine if the products are cost-effective in relation to the outcomes achieved. The panel should evaluate the vitamin A supplementation scheme in relation to compliance, missed children, correct implementation and the recording of the vitamin A on the RtHC, as well as the iodine in the supplements, as evidence shows that iodine deficiency disorder has almost been eliminated. The panel should look at the vitamin B_{12} status of South Africans and maybe only give 400µg of folic acid during pregnancy (Labadarios *et al*., 2008).

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APPENDIX A – PHYSICAL SIGNS

Physical signs indicative or suggestive of malnutrition (Grills and Bosscher, p.6 – 7, 1981; Torún and Chew, 1994, p.961; Golden and Golden, 2000, p.519; Shetty, 2002, p.320; Torún, 2006, p.892)

Body area	Normal appearance	Signs associated with malnutrition
Hair	Shiny, firm, not easily plucked	Lack of natural shine, hair dull and dry, thin and sparse, hair fine, silky and straight, color changes (flag sign), can be easily plucked
Face	Skin color uniform; smooth, pink, healthy appearance, not swollen	Skin color loss (depigmentation), skin dark over cheeks and under eyes (malar and supra-orbital pigmentation), lumpiness or flakiness of skin and nose and mouth, swollen face, enlarged parotid glands, scaling of skin around nostrils (nasolabial seborrhea)
Eyes	Bright, clear, shiny, no sores at corners of eyelids, membranes a healthy pink and are moist, no prominent blood vessels or mound of tissue or sclera	Eye membranes are pale (pale conjunctivae), redness of membranes (conjunctival injection), Bitot's spots, redness and fissuring of eyelid corners (angular palpebritis), dryness of eye membranes (conjunctival xerosis), cornea has dull appearance (corneal xerosis), cornea is soft (keratomalacia), scar on cornea, ring of line blood vessels around corner (circumcorneal injection)
Lips	Smooth, not chapped or swollen	Redness and swelling of mouth or lips (cheilosis), especially at corners ofmouth (angular fissures and scars)
Tongue	Deep red in appearance, not swollen or smooth	Swelling, scarlet and raw tongue, magenta (purplish color) of tongue, smooth tongue, swollen sores, hyperemic and hypertrophic papillae, and atrophic papillae
Teeth	No cavities, no pain, bright	May be missing or erupting abnormally, gray or black spots (fluorosis), cavities (caries)
Gums	Healthy, red, do not bleed, not swollen	"Spongy" and bleed easily, recession of gums
Glands	Face not swollen	Thyroid enlargement (front of neck), parotid enlargement (cheeks become swollen)
Skin	No signs of rashes, swellings, dark or light spots	Dryness of skin (xerosis), sandpaper feel of skin (follicular hyperkeratosis), flakiness of skin, skin swollen and dark, red swollen pigmentation of exposed areas (pellagrous dermatosis), excessive lightness or darkness of skin (dyspigmentation), black and blue marks due to skin bleeding (petechiae), lack of fat under skin
Nails	Firm, pink	Nails are spoon-shape (koilonychias), brittle, ridged nails

Muscular and skeletal systems	Good muscle tone, some fat under skin, can walk or run without pain	Muscles have “wasted” appearance, baby’s skull bones are thin and soft (craniotabes), round swelling of front side of head (frontal and parietal bossing), swelling of ends of bones (epiphyseal enlargement), small bumps on both sides of chest wall (on ribs) – beading of ribs, baby’s soft spot on head does not harden at proper time (persistently open anterior fontanelle), knock-knees or bow-legs, bleeding into muscle (musculoskeletal hemorrhages), person cannot get up or walk properly
Internal systems: Cardiovascular	Normal heart rate and rhythm; no murmurs or abnormal rhythms; normal blood pressure for age	Rapid heart rate (above 100 tachycardia); enlarged heart; abnormal rhythm; elevated blood pressure
Gastrointestinal	No palpable organs or masses (in children, however, liver edge may be palpable)	Liver enlargement; enlargement of spleen (usually indicates other associated diseases)
Nervous	Psychological stability; normal reflexes	Mental irritability and confusion; burning and tingling of hands and feet (paresthesia); loss of position and vibratory sense; weakness and tenderness of muscles (may result in inability to walk); decrease and loss of ankle and knee reflexes

APPENDIX B – START-UP FORMULA RECIPES

Start-up formula recipe (F-75)

The start up formula is a relatively high energy [315 kJ/100ml (75 kcal/100ml)], low protein (0,9g protein/100 ml), high carbohydrate, low sodium and low fat formula aiming to provide ± 100 kcal/kg and 1g protein/kg body weight during the start-up feeding at 130ml/kg body weight.

USE EITHER RECIPE A OR B

Ingredients	RECIPE A		RECIPE B
Whole dried milk	35g		-
Fresh cow's milk / Long life full cream	-	**OR**	300ml
Sugar	100g		100g
Vegetable oil	20ml		20ml
Warm boiled water to make up to:	1 000ml		1 000ml

- Using an electric blender, mix the milk, sugar and oil with warm boiled water and make it up to the 1 000ml mark. Blend at high speed.
- If no blender is available, mix the milk powder, sugar and oil to a paste, then slowly add the warm boiled water, stirring vigorously. Make it up to 1 000ml.

OR

- **Energy enriched starter infant formula** *(this is a less ideal formula for start-up feeding and included only for hospitals unable to produce the made up "start-up feed" above):*
    - To each 1 000ml of normally prepared starter milk formula (e.g. S26-1, Infacare 1, Nan-1) add 10ml of vegetable oil.

- **An alternative energy enriched infant formula with a lower fat and more appropriate protein content, similar to recipes A and B can be made as follows:**
    - To 600ml of normally prepared starter formula (e.g. S26-1, Infacare 1, Nan-1) add 70g of sugar and 10ml of vegetable oil. Dilute to 1 000ml by adding warm boiled water (NDoH, 2003).

APPENDIX C – FEED VOLUMES FOR START-UP FORMULA

Initial start-up formula feed volumes

CHILD'S DRY WEIGHT (*kg*)	**Amount of feed (*mL*)** These volumes aim to provide a maximum of 130ml/kg per 24 hours, thus providing 80-100 kcal/kg and 1g protein/kg dry body weight during the initial feeding			**Naso-gastric feeding (*ml/day*)** is needed if the total amount taken daily is less than the volumes shown in this column. All feeds must be given daily.
	Every 2 hours* (12 feeds/day)	**Every 3 hours (8 feeds/day)**	**Every 4 hours (6 feeds/day)**	
2.0	20	30	45	210
2.2	25	35	50	230
2.4	25	40	55	250
2.6	30	45	55	270
2.8	30	45	60	290
3.0	35	50	65	320
3.2	35	55	70	340
3.4	35	55	75	360
3.6	40	60	80	380
3.8	40	60	85	400
4.0	45	65	90	420
4.2	45	70	90	440
4.4	50	70	95	460
4.6	50	75	100	490
4.8	55	80	105	510
5.0	55	80	110	530
5.2	55	85	115	550
5.4	60	90	120	570
5.6	60	90	125	590
5.8	65	95	130	610
6.0	65	100	130	640
6.2	70	100	135	660
6.4	70	105	140	680
6.6	75	110	145	700
6.8	75	110	150	720
7.0	75	115	155	740
7.2	80	120	160	760
7.4	80	120	160	780
7.6	85	125	165	810
7.8	85	130	170	830
8.0	90	130	175	850
8.2	90	135	180	870
8.4	90	140	185	890
8.6	95	140	190	910
8.8	95	145	195	930
9.0	100	145	200	950
9.2	100	150	200	980
9.4	105	155	205	1 000
9.6	105	155	210	1 030
9.8	110	160	215	1 040
10.0	110	160	220	1 060

** Use 2 hourly x 12 daily feeds when hypoglycemia and/or hypothermia is present.*

(NDoH, 2003)

APPENDIX D – CATCH-UP FORMULA RECIPES

Catch-up formula recipe (F-100)

The start up formula is a high energy [420 kJ/100ml (100 kcal/100ml)], high protein (2,9g protein/100 ml) and high fat formula aiming to provide, with or without solids, [630-840 kJ/kg (150-200 kcal/kg)] and 4-6g protein/kg body weight during the catch up phase.

USE EITHER RECIPE C OR D

Ingredients	RECIPE C		RECIPE D
Whole dried milk	110g		-
Fresh cow's milk / Long life full cream	-	**OR**	880ml
Sugar	50g		50g
Vegetable oil	30ml		30ml
Warm boiled water to make up to:	1 000ml		1 000ml

- Using an electric blender, mix the milk, sugar and oil with warm boiled water and make it up to the 1 000ml mark. Blend at high speed.
- If no blender is available, mix the milk powder, sugar and oil to a paste, then slowly add the warm boiled water, stirring vigorously. Make it up to 1 000ml.

OR

- **Energy enriched starter infant formula** *(e.g. Nan-Pelargon or full cream infant milk) (this is a less ideal formula for catch-up feeding and included only for smaller hospitals unable to produce the made up "catch-up feed" above):*
    - To each 1 000ml of normally prepared Acidified Infant Milk (*or Full Cream Infant Milk*), add 20ml of vegetable oil and 25g of sugar.

- **An alternative energy enriched infant formula with a more appropriate protein content, similar to recipes C and D can be made as follows:**
    - To each 1 000ml of normally prepared Infant Milk (e.g. Lactogen-2, Promil-2, Infacare 2) add 35ml of vegetable oil and 15g sugar (NDoH, 2003).

APPENDIX E – 10 STEPS IN THE TREATMENT OF SEVERE MALNUTRITION

INPATIENT MANAGEMENT OF CHILDREN WITH SEVERE MALNUTRITION

1. INPATIENT MANAGEMENT OF CHILDREN WITH SEVERE MALNUTRITION

(Admit all cases of severe malnutrition for hospital treatment)

With appropriate protocols for the inpatient management of children with severe malnutrition (*kwashiorkor/marasmus*) the mortality rate can be reduced to < 10%, even without advanced medical support.

The principles of the **WHO Ten Steps to Treating Severe Malnutrition** cover these issues.

1.1 WORKING CASE DEFINITIONS

Severe Malnutrition

(*All cases of kwashiorkor, marasmus or marasmic kwashiorkor*)

1.1.1 Kwashiorkor:

A clinically recognizable syndrome of protein energy malnutrition characterized by peripheral oedema, skin changes and fine pale sparse hair.

1.1.2 Marasmus:

A clinically recognizable syndrome protein energy malnutrition characterized bye severe wasting due to loss of muscle and subcutaneous fat and under 60% expected weight for age.

1.1.3 Marasmic Kwashiorkor:

A mixed form of severe malnutrition that has features of both marasmus and kwashiorkor, including oedema.

All forms of severe malnutrition have potential high mortality, especially those with oedema. All these children need special care.

1.2 DANGER SIGNS IN CHILDREN WITH SEVERE MALNUTRITION

1.2.1 Signs indicating the need for increased vigilance and intensive management:

- Shock
- Dehydration
- Respiratory distress
- Fits
- Decreased level of consciousness
- Lethargy
- Hypothermia
- Hypoglycemia

- Jaundice
- Refusing feeds
- Weeping skin lesions

Consider referral of children with danger signs to regional hospital care with due care before and during transfer to begin treatment as outlined in the guide.

1.3 WARD TREATMENT

1.3.1 Investigations

The following should be done on admission:

- Blood glucose test strip
- Ward Hb/Full blood count if available
- Blood cultures if available (*commence anti-biotics irrespective of results*)
- Chest X-ray
- Urine dipstix
- Tine/Mantoux test
- HIV testing with appropriate pre-counseling should be offered

In certain academic institutions other investigations may be carried out for research and clinical reasons but should be interpreted with care, as attempts to correct serum deviations may be dangerous unless the pathophysiology of severe malnutrition is fully understood.

(NDoH, 2003)

2. PREVENT AND TREAT HYPOGLYCEMIA AND INITIATE "START-UP" FEEDING (WHO STEPS 1 AND 7)

A common cause of mortality and morbidity is hypoglycemia, which can be prevented with frequent (3 hourly) regular feeding both night and day (never missing a feed) and prevention of hypothermia plus aggressive treatment of infection.

2.1 Initiate feeding immediately with start-up formula (Diagram 1). Feed using a cup or spoon, not by bottle

2.1.1 Start-up Feeding

Volume: 130ml/kg/day divided into:

- 3 hourly feeds 8 times a day
- 2 hourly feeds 12 times a day
- See Appendix C for feed volumes by child's weight

2.1.2 Type of Feed

- Start-up formula, preferably formula A or B, provides 315 kJ (5kcal) and 0,9g protein/100 ml (See Appendix B for "start-up" recipes)
- If unavailable, use infant formula, modified to give a comparable energy and protein content (See Appendix B for "start-up" recipes)

2.2 Nasogastric tube

2.2.1 If a child refuses to feed, give the feed by nasogastric tube. If a child is not finishing feeds and the 24-hour intake is less than the amount shown in Appendix C, insert a nasogastric tube and give the unfinished amount by this route.

2.2.2 In the rare event that enteral feeding is impossible, ensure careful *IV fluid infusion*. *Use neonatal maintenance fluid (if not available, ½ Darrows, 5% Dextrose) at 80ml/kg/day (rate well controlled).*

2.3 Test blood glucose on arrival

2.4 Treat hypoglycemia

2.4.1 Treat asymptomatic hypoglycemia (*blood glucose of under 3mmol/L or, where a blood glucose machine is not available, under 4mmol/L by visual reading*) with a feed of start-up formula or 10% glucose (*50ml*) or sucrose solution (*1 rounded teaspoon of sugar in 3 and ½ tablespoons of water*), whichever is available. Re-check the blood sugar in 30 minutes to assure it is above 3mmol/L (*4mmol/L if no blood glucose machine*). If not, repeat feed as above.

2.4.2 Treat symptomatic hypoglycemia (*fits/decreased level of consciousness*), severe hypoglycemia *(<1.5mmol/*L) by 5ml/kg IVI of 10% dextrose solution. If only 50% dextrose is available, dilute 1 part of 50% dextrose solution with 4 parts sterile water.

2.5 Test blood glucose 3 hourly in severely ill children
(NDoH, 2003)

3. PREVENT AND TREAT HYPOTHERMIA (WHO STEP 2)

Hypothermia is present when the under-arm temperature is below 36^{o}C, and indicates the need to immediately warm up and feed the child.

3.1 Prevent hypothermia

- Measure under-arm temperature 3 hourly.
- Keep the child covered at all times, including the head, especially at night
- Avoid draughts in the ward
- Keep the child dry
- Avoid exposure (*such as bathing*)
- Use mother-child skin to skin contact (*Kangaroo care*) to keep the child warm

3.2 Treat hypothermia

- Immediately place the child in skin to skin contact (Kangaroo care) with the mother's chest and/or abdomen and wrap both with blankets.
- If the mother is absent clothe and wrap the child (including the head) with a warmed blanket.
- Place a heater nearby.
- Monitor temperature during re-warming to avoid hyperthermia or uncorrected hypothermia. Check the temperature every 2 hours until it rises over 36,5^{o}C.

(NDoH, 2003)

4. PREVENT AND TREAT DEHYDRATION (WHO STEP 3)

Many children with severe malnutrition also suffer from diarrhea, and may therefore become dehydrated.

4.1 To prevent dehydration in a child with diarrhea:

- Replace approximate volumes of stool losses with South African Rehydration solution (*ORS*) after each stool is passed.
    - After each stool give:
        - < 2 years old: 50-100ml of ORS
        - > 2 years old or equal: 100-200ml of ORS
    - Give in small frequent sips using a cup or spoon

- Encourage continued breast feeding if breastfed
- If not breast feeding, start feeding with start-up formula immediately

4.2 Treat diarrhea with dehydration (South African Rehydration solution [*ORS*]):
(Na 64mmol/L, K 20mmol/L, Citrate 10mmol/L, Dextrose 2%)

- Give 5ml/kg over every 30 minutes for 2 hours (*orally or if refused, by nasogastric tube*)
- Thereafter give 10ml/kg every hour for the next 4-10 hours until dehydration is corrected
- Restart feeds after 4 hours, sooner if the child is rehydrated before this
- Monitor for signs of overload at least every hour and stop if necessary
- Monitor for signs of ongoing dehydration and consider the need for more aggressive treatment if dehydration fails to resolve
- Monitor for shock:
    - Shock is present if the child has cold hands and feet, delayed capillary re-filling time, and peripheral pulses that are difficult to feel
    - Fluid overload is present if there is a gallop rhythm and enlarging liver

4.3 Emergency treatment for shock:

- Use *IV Ringers Lactate* to treat shock when present
- Do so with care to avoid circulatory overload and heart failure
- Use a pediatric giving-set (*60 drops per milliliter*)
- Monitor for signs of fluid overload every 10-15 minutes
- If unable to start a reliable IV line in a few minutes, use intra-osseous route
- Shock from both dehydration and sepsis may coexist in severely malnourished children. They are difficult to differentiate on clinical grounds alone.
- Children with dehydration shock will respond to *IV fluids*. Those with septic shock often will not respond.

4.4 To start treatment of shock:

- Give oxygen by mask or head box during the treatment of shock
- Mark the lower edge of the liver, to enable detection of enlargement if fluid overload should occur
- Give *IV Ringers Lactate* 15ml/kg over 10 minutes using a syringe while watching carefully for signs of shock or fluid overload

- Measure and record pulse, respirations, capillary filling time, gallop and liver edge every 5-10 minutes

Then:

- If the child deteriorates with administering of *IV Ringers Lactate* and develops gallop rhythm and enlarging liver, the child probably has septic/cardiogenic shock and needs very special care. Do not give further fluid for shock. Seek consultation or referral **immediately**.

- If there are no signs of improvement the child may have septic shock or have inadequate treatment of hypovolaemic shock. Repeat an aliquote of 10ml/kg of *Ringers Lactate* and watch response:
    - If there is no response or the child deteriorates, treat as in bullet one above
    - If response occurs, manage as referred to bullet three below

- If there are signs of improvement this indicates that the child was in hypovolaemic shock:
    - If the child still has signs of shock, administer a further 10ml/kg over 10 minutes using a syringe. This may be repeated one or more times if the signs of shock still remain, provided no signs of overload have developed (*total infusion 35ml/kg over 30-40 minutes*)
    - When the child no longer has signs of shock, give a further 10ml/kg slowly over 1 hour and then switch to oral or nasogastric rehydration with South African Rehydration solution (*ORS*) 10ml/kg/hour for up to 10 hours (*Leave IV line in place running very slowly in case it is required again*)
    - Begin feeding with start-up feed as soon as child is rehydrated

(NDoH, 2003)

5. TREAT INFECTION (WHO STEP 5)

Infection is common, but signs of infection such as fever, are often absent. Therefore, all patients with severe malnutrition should be automatically treated with antibiotics.

5.1 Treat all admissions routinely as infected:

- **Bacterial infection**
    - ***Uncomplicated child*** *(no danger signs present),* must receive antibiotics as below:

        - Cotrimoxazole 5ml suspension twice a day for 5 days,

        OR

        - Amoxycillin 15mg/kg 8 hourly orally for 5 days.

    - ***Complicated child*** *(hypoglycemia, hypothermia, lethargy, skin lesions, respiratory infection*), must receive antibiotics as below**:**

- Gentamicin 7,5mg/kg IM/IVI 24 hourly for 7 days

AND

- Ampicillin 50mg/kg IM/IVI 6 hourly for 2 days

THEN

- Amoxycillin 15mg/kg 8 hourly for 5 days

- **If child fails to improve in 48 hours:**
  - Confirm all the above steps have being carried out
  - Confirm correct feeding
  - Investigate aggressively for occult infection (*chest/urine/blood/csf*)
  - Where investigation or referral is not possible, add:
    - Chloramphenicol 25mg/kg IMI/IV/PO every 6 hours for 5 days

5.2 Treat all admissions for gastrointestinal infection:

- Oral metronidazole 7,5mg/kg three times a day for 5 days

(NDoH, 2003)

6. CORRECT ELECTROLYTE IMBALANCE AND MICRONUTRIENT DEFICIENCIES (WHO STEP 4 AND 6)

6.1 All severely malnourished children have electrolyte imbalances

- Prepare food without added salt

6.2 Treat and prevent electrolyte imbalances

- Give mineral/trace element mix daily, orally:
  - Zink Sulphate 36mg/ml
  - Copper Sulphate 0,1mg/ml
  - Magnesium Sulphate 280mg/ml
    (2.5ml if <10kg / 5ml if > 10kg)

AND

- Potassium Chloride Oral Solution:
  - 250mg 3 times a day orally up to 10kg
  - 500mg 3 times a day orally if > 10kg

6.3 Treat and prevent vitamin deficiency

- Give Vitamin A:
  - 50 000 units stat orally if < 6 months
  - if 6-12 months, 100 000 units stat, and
  - if > 12 months up to 5 years 200 000 units stat
- Give Folic Acid (*2,5mg/day*)
- Give Multivitamin Syrup (*5ml/day*)

6.4 Treat and prevent iron deficiency only after the child has started to gain weight

- Iron supplementation is ***not given until*** the child starts to gain weight, even if anemic
- Once gaining weight and oedema is lost, give:
  - 0,5ml/day of Ferrous Gluconate Syrup divided into 2 doses daily (*3mg/kg/day element iron) [Ferrous Gluconate Syrup – EDL: 30mg elemental iron per 5ml]*
- At this stage, give:

- Mebendazole 100mg bd orally for 3 days

(NDoH, 2003)

7. REBUILD WASTED TISSUES (WHO STEP 8)

The broad aim of this catch-up phase is to gradually build up to a total energy intake of 630-840kJ/kg (150-200 kcal/kg) body weight and 4-6g of protein/kg body weight over a few days using the catch-up formula, with or without solids (*Figure 2.6)*. The catch-up formula provides 420 kJ/100ml (100 kcal/100ml) of energy and 2,9g/100ml of protein (***Appendix D:*** *Catch-up Formula recipes*). The catch-up phase ***only*** starts when a child's appetite returns to normal (*usually within a week*).

7.1 For the first two days:

- Replace the start-up formula with an equal amount of catch-up formula given every four hours.

7.2 After the two days:

- Increase each feed by 10ml until some feed remains unfinished (*the total intake should not exceed 180-200ml/kg/day*)
- If the child is younger than 6 months, give a total of 6-7 feeds/days, using the catch-up formula
- If the child is older than 6 months and used to eating family meals, give 4-5 feeds of catch-up formula and 3 family meals of high energy and protein.

(NDoH, 2003)

8. PROVIDE STIMULATION, PLAY AND LOVING CARE (WHO STEP 9)

Stimulation, play and loving care will markedly improve the child's response to treatment and decrease the period of hospitalization.

8.1 From admission provide tender loving care

8.2 Structure play and activity in a cheerful stimulating environment encouraging mother's involvement as far as possible.

Some suggestions:

- *Hang colorful objects form cot rails*
- *Pick child up at least hourly for love, play and contact*
- *Sing or have music playing*
- *Use a kind, soothing voice*

(NDoH, 2003)

9. PREPARE FOR DISCHARGE AND FOLLOW-UP (WHO STEP 10)

The ability of the family to provide adequate nutrition and care at home must be assured.

9.1 While still in the ward:

- Involve the parents/caretakers in feeding and caring for the child as soon as possible, as they will care for the child over the long term.

9.2 Discharge the child when the child and the home environment are ready *(usually about 4 weeks after admission)***.**

Signs of readiness for discharge include:

- Persistant and good weight gain
- Good appetite
- A smiling and playful child

9.3 Check that the child has received all appropriate immunizations before discharge

9.4 Repeat the Tine/Mantoux test

9.5 *On discharge*:

- The child should leave with a supply of appropriate milk supplement / enriched porridge.
- The mother/caregivers should have a discharge summary of the child's stay in hospital
- The family should be counseled, and taught to:
    - Prevent and manage diarrhea
    - Provide energy and nutrient dense foods at least **five** times a day
    - Increase the energy content in the normal diet by adding vegetable oil or sugar
    - Add protein and micornutrients to the diet by using beans, vegetables, peanut butter and meat/fish/egg
    - Have a separate plate for the child in the home and carry out "active feeding" *(i.e. the feeder must actively promote and actually feed the child*)
    - Play with the child to improve his/her mental development

9.6 Arrange for follow-up post-discharge

- Make a written referral and appointment with the nearest primary health care facility (*clinic*) and community health worker (*if available*) for home support and encouragement
    - *The heatlh care system must:*
        - Provide appropriate accessible supervision during the child's recovery
        - Provide food supplementation as needed
        - Give Vitamin A supplementation six monthly

9.7 The social care system should provide social grants whenever applicable and the application process should begin before discharge.

- **Child Support Grant** *(for children under 7 years old whose primary caretaker receives no remuneration and where the family income is below the means test)*
- **Foster Care Grant** *(for children formally in foster care and below the means test)*
- **Care Dependancy Grant (***for children between 1 and 18 years with severe of profound mental of physical disability and whose caretaker are below the means test)*

(NDoH, 2003)

APPENDIX F – INFORMED CONSENT FORM AND INFORMATION DOCUMENT – AFRIKAANS

TOESTEMMING VIR DEELNAME AAN ‘N NAVORSINGSTUDIE

Hiermee word u kind versoek om aan ‘n navorsingstudie deel te neem.

U is oor die studie ingelig deur ……...

U kan vir …….…… by …….. skakel indien u enige vrae in verband met die studie het.

Indien u vrae het oor u kind se regte as hy/sy aan die studie deelneem, kan u die Sekretariaat van die Etiek Kommittee, van die Fakulteit van Gesondheidswetenskappe van die Univeristeit van die Vrystaat, skakel by (051) 4052812.

U kind se deelname aan die studie is vertroulik, vrywillig en u kind sal nie benadeel word indien u besluit dat u kind nie aan die studie mag deelneem nie of indien u later besluit om u kind van die studie te onttrek nie.

Wanneer u toestemming gee vir u kind om aan die studie deel te neem, sal u ‘n getekende kopie van die toestemmingsdokument ontvang, sowel as ‘n inligtingsdokument (‘n opsomming van die studie en wat dit behels).

Die navorsingstudie en die bogenoemde inligting is aan my verduidelik. Ek verstaan wat my deelname aan die studie beteken en ek stem in om vrywilliglik deel te neem.

_____ _____

Handtekening van ouer/oppasser Datum

INLIGTINGSDOKUMENT

Faktore wat bydra tot wanvoeding onder kinders 0-60 maande wat in die Noord-Kaap in die hospitaal opgeneem word

Geagte mnr. / me

Ons, by die Departement van Gesondheid van die Noord-Kaap, doen ‘n navorsingstudie oor die faktore wat bydra tot wanvoeding onder kinders 0-60 maande wat in die Noord-Kaap in die hospitaal opgeneem word. Navorsing is die proses waarby ‘n antwoord op ‘n vraag gekry word.

Die doel van die navorsingsopname is om voedingstatus (antropometries en dieetinname) en huishoudelike inligting te verkry om sodoende vas te stel watter spesifieke faktore ‘n rol speel in die ontwikkeling van erge wanvoeding in kinders. Die inligting wat verkry word, sal gebruik word om probleme te identifiseer en oplossings vir hierdie probleme te vind.

Hiermee vra ons toestemming dat die kind aan die navorsingstudie kan deelneem.

Om die nodige inligting te bekom sal dit nodig wees dat u vrae oor die volgende onderwerpe beantwoord:

- Agtergrond inligting bestaande uit sosio-demografiese inligting soos opleidingsvlak, huishoudelike inkomste
- Die tipe en hoeveelheid kos wat u die kind gee en hoe gereeld hy/sy die kos eet.
- Borsvoeding of ander voedingskeuses of gebruike
- Inligting ontvang by die kliniek
- U en die kind se gewig, lengte en bo-arm omtrek gaan gemeet word
- Hospitaal agtergrond
- Voorgeboorte risiko faktore en gebruike
- Mediese behandeling van u en die kind
- Voorkoms van siektes bv. TB & MIV/ VIGS; en
- Kliniek bywoning en betrokkenheid by die PEM skema

Die onderhoud en vraelys gaan voltooi word terwyl die kind in die hospitaal is en daarom sal slegs een kontaksessie nodig wees om die nodige inligting te bekom. Die onderhoud en invul van die vraelys sal omtrent 1 uur duur. Die mates wat geneem gaan word, gaan net eenmalig geneem word en is glad nie skadelik vir die kind of ouer/oppasser nie. Die mates wat geneem gaan word, is lengte, massa en bo-arm omtrek. Mates wat geneem word, word geneem terwyl die kind en ouer/oppasser die minimum hoeveelheid klere aanhet.

Geen spesiale toetse gaan op u kind uitgevoer word nie en slegs beskikbare bloeduitslae gaan gebruik word. Die bloeduitslae verwys na enige toetse en uitslae of verwante toetse en uitslae wat verband hou met MIV/VIGS of ander siektes, van die kind.

Die bogenoemde inligting gaan verkry word vanaf wangevoede kinders 0-60 maande wat opgeneem is in die Kimberley Hospitaal Kompleks in Kimberley en die Gordonia Hospitaal Kompleks in Upington, Suid-Afrika. Die hoeveelheid kinders wat aan die studie

gaan deelneem, sal afhang van die hoeveelheid kinders wat met wanvoeding in bogenoemde hospitale opgeneem word oor ‘n periode van 6-12 maande.

Die deelnemer loop geen risiko met deelname aan hierdie studie nie.

Deurdat u instem dat u kind aan die studie deelneem sal u bydra tot verbetering van gesondheidsdienste in die Noord-Kaap.

U sal voorsien word van inligting rondom die studie soos die studie vorder en ook nadat die resultate beskikbaar is.

Deelname is vrywillig en u sal nie benadeel word indien u besluit om nie deel te neem aan die studie nie. Indien u besluit om nie verder met die studie aan te gaan nie, sal dit nie teen u gehou word nie.

Alles moontlik sal gedoen word om te verseker dat persoonlike inligting vertroulik gehou word. Totale vertroulikheid is nie moontlik nie, maar die persoon sal nie geidentifiseer word met die analisering van data en voordra van resultate aan kollegas en ander betrokkenes nie en ook nie wanneer die resultate in wetenskaplike joernale gepubliseer word nie.

Die studie is deur die Etiek Kommittee van die Fakulteit van Gesondheidswetenspappe van die Universiteit van die Vrystaat (ETOVS nr. 113/07) sowel as die Etiek Kommittee van die Kimberley Hospitaal Kompleks goedgekeur.

Vir meer inligting kan u die navorser kontak by:
Christel de Lange, Geregistreerde Dieetkundige
Tel: 053 – 497 3146
Faks: 053 – 497 3440
Epos: kanivest@joba.co.za

Om enige probleme of klagtes te rapporteer, kontak:
REK Sekretaris en Voorsitter
Tel: 051 – 405 2812
Epos: gndkhs.md@mail.uovs.ac.za

APPENDIX G: INFORMED CONSENT FORM AND INFORMATION DOCUMENT – ENGLISH

CONSENT TO PARTICIPATE IN RESEARCH

You have been asked for your child to participate in a research study.

You have been informed about the study by ……

You may contact …….. at …….. at any time if you have questions about the research.

You may contact the Secretariat of the Ethics Committee of the Faculty of Health Sciences, UFS at telephone number (051) 4052812 if you have questions about your child's rights as a research subject.

Your child's participation in this research is confidential, voluntary, and you will not be penalized if you refuse for your child to participate or decide to terminate participation.

If you agree for your child to participate, you will be given a signed copy of this document as well as the participant information sheet, which is a written summary of the research.

The research study, including the above information has been verbally described to me. I understand what my child's involvement in the study means and I voluntarily agree for my child to participate.

_____ _____

Signature of Parent / Caregiver Date

INFORMATION DOCUMENT

Factors contributing to malnutrition in children 0-60 months admitted to hospital in the Northern Cape

Dear Sir / me

We, the Department of Health, Northern Cape, are doing research on the factors contributing to malnutrition in children 0-60 months that are admitted to hospitals in the Northern Cape. Research is just the process to learn the answer to a question.

The purpose of the research survey is to assess nutritional status (anthropometric and dietary intake) and household information, in an attempt to identify specific factors that play a role in the development of children suffering from malnutrition. The information collected will be used to resolve problems and instigate solutions for these problems.

We are asking you and your child to participate in a research study.

In order to collect this information you will be asked a number of questions regarding:

- Background information that consists of socio- demographic information like education level, household income
- Types & amounts of food given to your child and how often he/ she eat these foods
- Breastfeeding or other feeding practices
- Counseling received at the clinic
- Weight, height and mid-upper-arm circumference measurements of you and the child in your care
- Hospital background
- Ante-natal risks and practices
- Medical treatment of you & your child
- Prevalence of disease (TB & HIV/ AIDS); and
- Clinic attendance and participation in the PEM Scheme

The questionnaire will be completed while the child is in hospital so therefore only one visit is required to collect all the necessary information. The entire interview will take about one hour to complete. The measurements that are going to be taken are not harmful in any way to you or your child and will only be done once. The measurements that are going to be taken are height, weight and mid-upper arm circumference. Measurements will be taken with the child and mother/caregiver wearing a minimum amount of clothes.

No special tests will be done and only available blood results will be used. The blood results refer to any and all tests and results or related tests and results regarding HIV/AIDS and other diseases.

This information is collected from malnourished children 0-60 months in Kimberley Hospital Complex in Kimberley and the Gordonia Hospital Complex in Upington, South Africa. The number of children that are going to take part in the study depends on the number of children admitted to these hospitals over a period of 6-12 months.
There are no risks involved in taking part in this research study.

The benefits for partaking in this research survey will be that you can make a contribution to improving health care services in the country and your child will be treated for any other diseases he/she has.

You will be given pertinent information on the study while involved in the project and after the results are available.

Participation is voluntary, and refusal to participate will involve no penalty and you may discontinue participation at any time and it will not be held against you in any way.

Efforts will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed but the person will not be identified when analyzing and presenting findings to various stakeholders or when publishing the results in scientific journals.

The Ethics Committee of the Faculty of Health Sciences of the University of the Free State and the Ethics Committee of the Kimberley Hospital Complex have approved the study (ETOVS 113/07).

For further information you can contact the researcher at:
Christel de Lange, Registered Dietician
Tel: 053 – 497 3146
Fax: 053 – 497 3440
Email: kanivest@joba.co.za

To report any complaints or problems you can contact:
REC Secretariat and Chair
Tel: 051 – 405 2812
Email: gndkhs.md@mail.uovs.ac.za

APPENDIX H: INFORMED CONSENT FORM AND INFORMATION DOCUMENT – TSWANA

TUMALANO YA GO TSA KAROLO MO PATLISISONG

O kopilwe go nna modiragatsi mo patliso thuto.

O sedimoseditswe ka thuto ke …….

O ka amana le …… mo ……
nako engwe le engwe fa o nale dipotso ka patlisiso e.

O ka amana le Mokwaledi wa Semorafe wa Khuduthamaga ya Lefapha la Boitekanelo jwa Boitsanape wa UFS mo palo mogala (051) 405 2812 fa o nale dipotso ka ditshwanelo tsa semolao ka sediri sa patliso.

Bodiragatsi ba gago mo patlisong ke lekunutu le boithaopo, ga ona o otlhaiwa fa o tsaya karolo kgotsa fo fedisa boithaopi.

Fa o dumela go tsa karolo o tla neelwa lokwalo le o le saenileng le le tlhalosang patliso le tshedimoso e e neetsweng go wena.

Tshedimoso yotlhe ka e tlhaloseditswe ka molomo. Ke tlhaloganya gore go tsaya karolo game mo tlhalosang le boithaopi go nna karolo ya patliso thuto e.

_____ _____

Saeno Motsadi / Motlhokomedi Letlha

LEKWALO LA THEDIMOSO

Pako ya phepelotlase e e tseneletseng go bana 0-60 dikgwedi ba ba amogelwang mo dikokelong mo (Northern Cape) Kapa Bokone

Mme / Re oo rategang

Rona lefapa la Pholo mo Kapa Bokone re tshwaragane le go dira patliso go lemoga gore ke eng se se bakang phepelotlase e e tseneletseng jaana go mase 0-60 dikgwedi mo Kapa Bokone Patliso ke feela tirego ya go ithuta go ka araba dipotso.

Lebaka la patliso ke go tlhatlhoba maemo a phepelo ya mo magaeng, ka boiteko ba go lemogo mabaka a kgethegileng a tsayang karolo mo tlhabologong ya bana ba ba tshwereng ka botlhoko jwa phepelotlase. Tshedimoso ee kgobokantsweng o tla kgontsha go diriswa go ranola bothata le go tlhotlheletsa go tharabolola bothata.

Re kopa motsadile ngwana go nna ba tsaa karolo.

Go phuta tshedimoso o tla kopiwa go araba dipotso mabape le.

- Lemorago la tshedimoso
- Mofuta le tekano ya dijo tse odi neelang ngwana le gore o ja selekano se se kae le gore o ja ga kae
- Go anyiswa kgotsa phepo e ngwe
- Thotloetso e e neelwang ka kliniking
- Boima, Bogodimo le sediko-modiko wa le tsogo palo ya ga mme le ngwana yo o mo tlhokomelang
- Lemorago la Bookelo
- Tirelo ya Baimana ee diphatsa e e dirwa mo magaeng
- Kitso ka morafe (Tekanyetso thuto, lotseno mo magaeng)
- Go tlala tlala ga matlhoko (TB le HIV/AIDS) le
- Tsamayo ya kliniki le go tsa karolo mo PEM scheme

Dipotso di tla dirwa fa ngwana a sale mo kokelong ga ngwe fela go kokanya tshedimoso yotlhe. Puisano yotlhe e tla tsaya ura go fela. Ditekanyo di tla dirwa ka mokgwa o sekang wa utlwisa ngwana botlhoko. Le diteko tse di tlhaologileng di tla Bonwa mo faeleng ya kokelo. Seelo se se ileng go dirwa ke bogodimo, boima le modiko wa bogare ba letsogo.

Ga gona diteko tse di kgethegileng tse di tla dirwang. Go tla dirisiwa dipholo tsa madi tse dileng teng. Dipholo tsa madi di ka ya diteko tsotlhe tse di amanang le mogare wa HIV/AIDS le matlhoko a mangwe.

Tshedimoso e tla kgobokanwa go tswa mo bana ba ba phepelotlase 0-60 dikgwedi mo kokelong tse pedi fela ebong Kimberley Hospital le Gordonia Hospital kwa Upington, mo South Africa. Palo ya bana ga e itsiwe gone patliso e ka nna gareng ga kgwedi 6 to 12.

Ga go na kotsi epe mo dipatlisong.

Mosola wa patliso ke go nna motsa karolo mo patlisong go tlhabolola ditirelo le tlhokomelo ya ditirelo tsa pholo mo nageng ya rona.

O tla neelwa tshedimoso ka thuto fa o santse o tsa karolo le morago ga dipatliso.

Go tsa karolo go Boithaopi ga ona o otlhaiwa – fa o tlogela nako ngwe le ngwe.

Go tla dirwa ka bokgoni jotlhe go dirwa tshedimoso go nna khupa marama jaaka go ka kgonega fela eseng ka nako yotlhe gone fa go tshwanetswe go fetelwa ko pelo ka se se tla beng se pateletsa dipatliso tse di tseneletseng – tse di a karetsang Badira mmogo mo patlisong.

Tlhatlhobo e e rebotswe ke ba lefapha la khuduthamaga ya tlotlo ya lekala boitsanape ba boitekanelo la University ya Free State (EROS nr. 113/07).

For further information you can contact the researcher at:
Christel de Lange, Registered Dietician
Tel: 053 – 497 3146
Fax: 053 – 497 3440
Email: kanivest@joba.co.za

To report any complaints or problems you can contact:
REC Secretariat and Chair
Tel: 051 – 405 2812
Email: gndkhs.md@mail.uovs.ac.za

APPENDIX I: LETTER FOR PERMISSION FROM THE ETHICS COMMITTEE OF KIMBERLEY HOSPITAL COMPLEX

PO Box 110457
Hadison Park
8306

14 Mei 2007

TO: The Ehics Committee of Kimberley Hospital Complex
Kimberley Hospital Complex
Du Toitspanroad
Kimberley
8301

1. SUBJECT

This is to ask permission from the Ethics Committee of Kimberley Hospital Complex to carry out a research project titled “Factors contributing to severe malnutrition in children 0-60 months admitted to hospital in the Northern Cape”, that will be undertaken by a dietician of the Integrated Nutrition Programme (Northern Cape Department of Health) during 2007/ 2008.

2. AIM

The project is aimed at assessing nutritional status (anthropometric and dietary intake) and household information of children admitted to two hospitals in the Northern Cape, in an attempt to identify factors that play a role in the development of severe malnutrition.

3. METHODOLOGY OF STUDY

All severely malnourished children 0-60 months, admitted to Kimberley Hospital Complex and Gordonia Hospital during the study period (July to December 2007), will be included in the study. The researcher and hospital dietitians will either visit the wards or will require the necessary people to refer the patient to them to complete the consent forms (appendix B) and questionnaires (appendix A). I hope to include between 100-150 participants for the study in the Northern Cape.

Information will be obtained from the mother or caregiver during a personal interview. The caregivers will be given an information document (appendix C) explaining the study. Interpreters will be used where respondents cannot understand Afrikaans or English. They will be asked a number of questions about the household as well as what foods are eaten by the malnourished child. Questions are not difficult to answer and anyone will be able to answer them. The mother or caregiver and child will be weighed and measured. Biochemical data will be gathered from the files of the patients taking part in the study.

4. MOTIVATION

The results of the study will serve to help the Integrated Nutrition Programme to evaluate current programmes to establish the effectiveness of these programmes and to see if other interventions are necessary for the prevention and treatment of malnutrition in children younger than 5 years.

It may happen that the results will be published in a Medical Journal or presented at a meeting / congress for professional health workers.

5. FINANCIAL IMPLICATIONS

None

6. RECOMMENDATION

It will be appreciated if approval can be given to perform this research study in two hospitals in the Northern Cape.

7. GENERAL

Find attached relevant appendixes that are relevant to the study. The protocol is in its final stages of completion. If you need any more information you can contact Christel de Lange, the researcher, at:

Telephone number: 053-497 3146
Cellphone: 082 930 7212
Address: PO Box 110457
Hadison Park
8306

Or
Deliver to: Mrs. M. Le Roux
Department of Health
Integrated Nutrition Programme
James Exum Building, Room 62

8. COMPILED BY: Ms C. de Lange

_____ _____

SIGNATURE **DATE**

APPROVED / NOT APPROVED

_____ _____

THE ETHICS COMMITTEE: **DATE**
KIMBERLEY HOSPITAL COMPLEX

APPENDIX J: LETTER FOR PERMISSION FROM THE DEPARTMENT OF HEALTH OF THE NORTHERN CAPE

1. TO:

The Head of Department (acting)
Ms. M. Thuntsi
Department of Health
Private Bag X5049
KIMBERLEY
8300

2. SUBJECT

This is to ask permission from the Head of the Department of Health, in the Northern Cape, to carry out a research project titled “Factors contributing to malnutrition in children 0-60 months admitted to hospital in the Northern Cape”, that will be undertaken by a dietician of the Integrated Nutrition Programme (Northern Cape Department of Health) during 2007/ 2008.

3. AIM

The project is aimed at assessing the nutritional status (anthropometric and dietary intake) and household information, in an attempt to identify factors that play a role in the development of severe malnutrition.

4. METHODOLOGY OF STUDY

All severely malnourished children 0-60 months, admitted to Kimberley Hospital Complex and Gordonia Hospital during the study period (July to December 2007), will be included in the study. The researcher and hospital dietitians will either visit the wards or will require the necessary people to refer the patient to them to complete the consent forms and questionnaires. I hope to get between 100-150 participants for the study in the Northern Cape.

Information will be obtained from the mother or caregiver during a personal interview. Interpreters will be used where respondents cannot understand Afrikaans or English. They will be asked a number of questions about the household as well as what foods are eaten by the malnourished child. Questions are not difficult to answer and anyone will be able to answer them. The mother or caregiver and child will be weighed and measured. Biochemical data will be gathered from the files of the patients taking part in the study.

5. MOTIVATION

The results of the study will serve to help the Integrated Nutrition Programme to evaluate current programmes to establish the effectiveness of these programmes and to see if other interventions are necessary for the prevention and treatment of malnutrition in children younger than 5 years.

It may happen that the results will be published in a Medical Journal or presented at a meeting / congress for professional health workers.

6. FINANCIAL IMPLICATIONS

None

7. RECOMMENDATION

It will be appreciated if approval can be given to perform this research study in two hospitals in the Northern Cape.

8. GENERAL

If you need any more information you can contact Christel de Lange, the researcher, at:

Telephone number:	053-497 3146
Cellphone:	082 930 7212
Address:	PO Box 110457 Hadison Park 8306
Or	
Deliver to:	Mrs. M. Le Roux Department of Health Integrated Nutrition Programme James Exum Building, Room 62

9. COMPILED BY: Ms C. de Lange

_____ _____

SIGNATURE **DATE**

RECOMMENDED/NOT RECOMMENDED

_____ _____

MS. L. NYATI-MOKOTSO **DATE**
DIRECTOR:PRIORITY PROGRAMMES

APPROVED/NOT APPROVED

_____ _____

MS. K.M. THUNTSI **DATE**
ACTING HOD

APPENDIX K: INFORMATION LETTER TO THE HOSPITAL MANAGER, KIMBERLEY HOSPITAL COMPLEX

1. TO:

The Hospital Manager
Kimberley Hospital Complex
Dr. Shabbir
Du Toitspan Road
KIMBERLEY
8300

2. SUBJECT

This letter is to inform you that the acting Head of Department of Health of the Northern Cape, Mrs. K.M. Thuntsi has given her permission for a research study "Factors contributing to severe malnutrition in children 0-60 months admitted to hospital in the Northern Cape" to be carried out at the Kimberley Hospital Complex. Please see the attached letter to Mrs. K.M. Thuntsi.

3. GENERAL

If you need any more information you can contact Christel de Lange, the researcher, at:

Telephone number:	053-497 3146
Cellphone:	082 930 7212
Address:	PO Box 110457 Hadison Park 8306
Or	
Deliver to:	Mrs. M. Le Roux Department of Health Integrated Nutrition Programme James Exum Building, Room 62

4. COMPILED BY: Ms C. de Lange

_____ _____

SIGNATURE **DATE**

APPENDIX L: INFORMATION LETTER TO THE HOSPITAL MANAGER, GORDONIA HOSPITAL, UPINGTON

1. **TO:**
   The Hospital Manager
   Gordonia Hospital Complex
   Mr. Moncho
   UPINGTON

2. **SUBJECT**

   This letter is to inform you that the acting Head of Department of Health of the Northern Cape, Mrs. K.M. Thuntsi has given her permission for a research study "Factors contributing to severe malnutrition in children 0-60 months admitted to hospital in the Northern Cape" to be carried out at the Gordonia Hospital Complex. Please see the attached letter to Mrs. K.M. Thuntsi.

3. **GENERAL**

   If you need any more information you can contact Christel de Lange, the researcher, at:

Telephone number:	053-497 3146
Cellphone:	082 930 7212
Address:	PO Box 110457 Hadison Park 8306
Or	
Deliver to:	Mrs. M. Le Roux Department of Health Integrated Nutrition Programme James Exum Building, Room 62

4. **COMPILED BY: Ms C. de Lange**

_____ _____

SIGNATURE **DATE**

APPENDIX M: QUESTIONNAIRE - MALNUTRITION HOSPITAL SURVEY

QUESTIONNAIRE

FACTORS CONTRIBUTING TO MALNUTRITION IN CHILDREN 0-60 MONTHS ADMITTED TO HOSPITAL IN THE NORTHERN CAPE

INSTRUCTIONS:

- Please complete the questionnaire in black pen
- Please complete the questionnaire in full
- Leave the column marked "For office use only" open
- If an answer must be "specified" please be as accurate as possible
- Use legible writing
- No names or addresses may be written on the questionnaire

ABSTRACT

INTRODUCTION

A wide range of factors, including underlying, immediate and basic factors, play a role in the development of malnutrition. Globally, the prevalence of malnutrition is highest in Sub-Saharan African, with the HIV pandemic further compromising the situation. Both underweight and stunting are threatening the health of children younger than five years old, with the Northern Cape having the highest percentage of stunted children in South Africa. Malnutrition is still the leading cause of mortality and morbidity in children younger than five years old.

The main aim of this study was to determine which of the underlying, immediate and basic factors contributing to malnutrition are prevalent in the Northern Cape.

METHODS

Fifty-four malnourished children 0 to 60 months admitted to Kimberley Hospital Complex and Upington Hospital were included in the study. Inclusion criteria included all malnourished children 0 to 60 months admitted to paediatric or infant care units between August 2007 and July 2008with a weight-for-age below 80% of expected weight, with an RtHC and whose mother/ caregiver was present to sign the informed consent form. The anthropometric measurements of both the child and mother/caregiver were taken. Blood values of the child that were available in the files were consulted. Socio-demographic, household, maternal information, medical history of the child, infant feeding information and adherence to the FBDG were noted on a questionnaire during a structured interview conducted with the mother/caregiver.

RESULTS

Factors contributing to malnutrition were categorized into the immediate, underlying and basic factors as set out in the UNICEF conceptual framework of the causes of malnutrition. Some of the socio-demographic findings associated with malnutrition included rural households, male children, education level and marital status of the mother. Educated and married mothers were less likely to have a malnourished child. Anthropometric findings showed that low birth weight and the size of the child's mother were associated with malnutrition, with undernourished and obese mothers having a higher chance of having a malnourished child. Household food insecurity and inadequate

nutrition information received on care practices were often contributing factors. Most of the malnourished children included in the study were marasmic. The medical history of the child indicated that even though all the children had an RtHC, the cards were often completed incorrectly. Clinic attendance was poor and the screening for HIV and TB was insufficient as the children's statuses were mostly unknown. Significantly more children were up to date with their immunizations, but significantly fewer children were up to date on their vitamin A supplementation. The NSP was not accessed effectively and even children that did access the NSP were found to be malnourished after eight months on the programme.

Some of the other household and maternal findings related to malnutrition included a big household with more than five family members, a high birth order of more than four children and if the child had any siblings that had died of malnutrition related illnesses.

The education levels of the mothers were generally low and health and feeding information given at clinics did not have a significant impact. Information on infant feeding showed that exclusive breastfeeding is still a challenge and mothers are not effectively using milk alternatives when breastfeeding is ceased. Cup feeding was not practiced, and the use of bottles can increase the risk of diarrhoea. Children are either introduced to solid foods too early (before six months) or too late (after six months). When the application of the FBDG was evaluated, the study found that children had high intakes of fats, salt, sugar and sugary foods and tea and low intakes of animal proteins, fruit and vegetables and milk (after breastfeeding was ceased).

CONCLUSIONS

Inadequate access of available interventions programmes such as the NSP, immunizations, vitamin A supplementation, screening and treatment of diseases such as HIV and TB was noted. Parents were generally uneducated, especially regarding infant and young child feeding and the importance of correct food for the prevention of malnutrition. Household factors were a major challenge, especially in rural areas. Low levels of schooling and poverty are basic factors contributing to malnutrition that are prevalent in the Northern Cape.

RECOMMENDATIONS

Maternal and community education are some of the most important interventions to combat malnutrition in the Northern Cape. Intervention programmes at facilities should be strengthened to empower health care professionals and the community they serve to prevent and manage severe malnutrition. Detecting malnourished children earlier in the communities by using the MUAC to screen children is recommended. The management of severe malnutrition according to the 10 Steps of the WHO should be implemented at all levels of care.

KEYWORDS: severe malnutrition, kwashiorkor, marasmus, marasmic kwashiorkor, immediate factors, underlying factors, basic factors, Northern Cape, stunting, breastfeeding

OPSOMMING

INLEIDING

Die oorsake van wanvoeding word deur ‘n wye reeks faktore soos onderliggende, onmiddellike en basies oorsake bepaal. In die wêreld, is die voorkoms van wanvoeding die hoogste in Sub-Sahara Afrika, waar die MIV pandemie die probleem net verder vererger. Ondergewig en groeiinkorting is van die algemeenste probleme wat voorkom onder kinders jonger as five jaar oud, met die Noord Kaap wat die hoogste getal kinders met groeiinkorting het. Wanvoeding bly die hoofoorsaak van mortaliteit en morbiditeit in kinders jonger as vyf jaar oud.

Die hoofdoel van die studie was om te bepaal watter onderliggende, onmiddellike en basies oorsake wanvoeding in die Noord Kaap veroorsaak.

METHODES

Die studie het bestaan uit 54 wangevoede kinders tussen nul en 60 maande wat in die Kimberley Hospitaal Kompleks en Upington Hospitaal opgeneem is. Die insluitingskriteria het ingesluit, al die wangevoede kinders tussen nul en 60 maande wat tussen Augustus 2007 en Julie 2008 opgeneem is in die pediatriese of baba sale met ‘n gewig-vir-ouderdom laer as 80% van die verwagte gewig, met ‘n RtHC en wie se moeder/oppasser beskikbaar was om die toestemmingsbrief te teken. Die antropometriese mates van beide die kind en die moeder/oppasser is bepaal. Die bloedwaardes wat gebruik is, was die wat beskikbaar was in die kind se lêer. Sosio-demografiese en huishoudelike inligting, inligting vanaf die moeder, die mediese geskiedenis van die kind, babavoeding inligting en die vergelyking van voedselinname met die voedselgebaseerde dieetriglyne is deur ‘n onderhoud en vraelys, wat met die moeder/oppasser gevoer is, bepaal.

RESULTATE

Die oorsake van wanvoeding kan soos bepaal deur die UNICEF konseptuele raamwerk vir die oorsake van wanvoeding, uiteengesit word in onderliggende, onmiddellike en basiese oorsake. Plattelandse huishoudings, seuns en die opleidingsvlak en huwelikstatus van die moeder was van die sosio-demografiese oorsake wat in die studie met wanvoeding verband gehou het. Moeders wat opgevoed en getroud was, se kanse om ‘n wangevoede kind te hê was laer as vir moeders wat onopgelei en ongetroud is.

Die antropometriese mates het getoon dat ‘n lae geboortemassa en die grootte van die kind se moeder, met wanvoeding geassosieer word. Beide ondermassa en oormassa moeders het ‘n groter kans gestaan om ‘n wangevoed kind te hê.

Van die ander faktore wat bygedra het tot wanvoeding, was huishoudelike voedselonsekerheid en swak kennis in verband met die sorg van kinders. Die meeste kinders in die studie het marasmus gehad. Met die ontleding van die mediese geskiedenis van die kind, is gevind dat alhoewel die kinders RtHC gehad het, was die kaarte meestal onvolledig of verkeerd ingevul. Die kinders is nie gereeld kliniek toe geneem nie en sifting vir MIV en TB was onvoldoende aangesien van die kinders se MIV en TB status onbekend was. Beduidend meer kinders was op datum met hulle immunisasies en beduidend minder kinders was op datum met hulle vitamien A supplementasie. Die nasionale voedselsupplementasie program (NSP) was nie effektief benut nie, aangesien van die wanvoede kinders al vir agt maande op die programme was, sonder enige verbetering.

Van die huishoudelike inligting en inligting vanaf die moeder wat verband gehou het met wanvoeding, was groot huishoudings met meer as vyf familielede, ‘n hoë geboortesyfer van vier of meer kinders en die dood van ‘n ander kind as gevolg van voedingverwante siektes.

Die moeders was oor die algemeen swak opgelei en die gesondheids- en voedingsinligting wat by klinieke gegee is, was onvoldoende. Die inligting wat vanaf die moeders verkry is, in verband met babavoeding, het gewys dat borsvoeding nogsteeds ‘n probleem is en dat moeders verkeerde melkvervangers gebruik wanneer hulle ophou met borsvoeding. Die moeders het nie koppies gebruik om hulle kinders mee te voed nie en die gebruik van bottels kan die voorkoms van diaree verhoog. Vaste voedsel was te vroeg (voor ses maande) of te laat (na ses maande) aan die kinders bekendgestel. Die voedselinname van die kinders is vergelyk met die voedselgebaseerde dieetriglyne en daar is gevind dat kinders baie vet, sout, suiker en suikerbevattende voedsels en tee inneem en ook dat vrugte, groente, dierlike proteïene en melk (nadat borsvoeding gestop is) onvoldoende ingeneem word.

GEVOLGTREKKINGS

Die studie het gevind dat intervensie programme soos die nasionale supplementasie program, immunisasies, vitamien A supplementasie en die sifting en behandeling van siektes soos MIV en TB nie toeganklik implimenteer is nie. Ouers was onkundig as dit kom by die voeding van babas en jong kinders en besef nie die belang van goeie en korrekte voedsel vir die voorkoming van wanvoeding nie. Huishoudelike faktore bly ‘n uitdaging, veral in plattelandse areas. Die basiese oorsake van wanvoeding wat in die Noord Kaap voorkom, sluit lae vlakke van opleiding en armoede in.

AANBEVELINGS

Van die belangrikste intervensies om wanvoeding in the Noord Kaap te voorkom is die opleiding van gemeenskappe en moeders. Die intervensie programme wat by fasiliteite beskikbaar is, moet versterk word sodat die gesondheidswerkers en die gemeenskap kan help met die voorkoming en behandeling van wanvoeding. Kinders met wanvoeding moet vroegtydig, met behulp van bo-arm omtrek mates, deur gemeenskappe geïdentifiseer word. Die behandeling van wanvoeding moet volgens die 10 Stappe vir die behandeling van wanvoeding van die Wêreld Gesondheidsorganisasie by alle vlakke van gesondheidsorg plaasvind.

SLEUTELWOORDE: wanvoeding, kwasjiorkor, marasmus, marasmiese kwasjiorkor, onmiddellike oorsake, onderliggende oorsake, basiese oorsake, Noord Kaap, groeiinkorting, borsvoeding

APPENDIX M - MALNUTRITION HOSPITAL SURVEY

Office use only

	Questionnaire number (leave open)	1-3
	Date of interview _____	D D M M Y Y 4-9
	Name of interviewer _____	10-11
	Town _____	12-13
	Nearest Clinic _____	14-15
	Date of Birth _____	D D M M Y Y 16-21
	Birthweight _____ kg	. 22-25
	Gendar _____ **(1= Male : 2= Female)**	26
	Current Weight _____ kg	. 27-30
	Height _____ cm	. 31-35
	MUAC _____ mm	36-38
1	**What is the nutritional diagnosis of the child (as indicated in patient file)?** 1. Kwashiorkor 2. Marasmus 3. Marasmic Kwashiorkor	39
2	**Was the child born prematurely?** 1. Yes 2. No	40
	If so, at what gestational age? _____ weeks	41-42
3	**Where was the child born?** 1. Hospital 2. Clinic 3. Community Health Centre 4. Home 5. Other, please specify_____	43
4	**Does the child have a Road to Health Card?** 1. Yes 2. No	44
5	**Is the Road to Health Card correctly completed?** 1. Yes 2. No	45
6	**When last did the child attend a clinic?** _____ weeks ago	46-47

6.1 **For what reason did the child attend the clinic? Tick all that apply.**
(1= Yes, 2= No)

	YES	NO	
1. Growth Monitoring			48
2. Immunisation			49
3. Other, please specify, _____			50

7 **How regularly did the child attend the clinic after birth?** 51
1. Weekly
2. Monthly
3. Other, please specify _____

8 **Is the child currently on the PEM Scheme?** 52
1. Yes
2. No

If yes, for how long? (months) _____ 53-54

9 **Has the mother/ caregiver received counselling on the following topics?**
(more than one option can be marked)
(1= Yes; 2= No)

	YES	NO	
Diarrhea			55
Healthy eating			56
Breastfeeding			57
Complementary feeding			58
Food fortification			59
Growth Chart			60
Hygiene			61
Other _____			62

10 **Is the child's immunisations up to date?** 63
1. Yes
2. No

11 **Is the child's Vitamin A supplementation up to date?** 64
1. Yes
2. No

12 **Was/ Is the child breastfed?** 65
1. Yes
2. No

If NO, skip to Part (B) or if YES, only do Part (A) and continue at Q 13.

12.1 **(A) To what age?** 66-67

_____ months

12.2 **How long was the child exclusively breastfed?** 68-69

_____ months

12.3 **How long was the child partially breastfed (breastmilk and formula or other food and drink)** 70-71

_____ months

12.4 **(B) What milk did the child drink, if not breastfed?** 72
1. Formula Milk
2. Cow's Milk
3. Other, please specify _____

12.5 **If formula is given, please request the mother/ caregiver to explain**

the preparation of feeds.

1. Volume (or amount of water) per feed _____ ml
2. Amount of milk powder per feed _____ scoops
3. Number of feeds per day _____

73-76
77-78
79-80

12.6 Evaluation of formula milk preparation (to be interpreted by interviewer)

Is it sufficient for the child's age?

1. Yes
2. No

1

12.7 Is it prepared hygienically?

1. Yes
2. No

2

12.8 How was the milk fed to the baby?

(1= Yes, 2= No)

	YES	NO	
1. Bottle			3
2. Cup			4
3. Spoon			5

13 At what age did the mother introduce solid foods?

_____ months

6-7

14 Food Based Dietary Guidelines

14.1 What other kinds of food does your child eat together with porridge?

(1= Yes, 2= No)

	YES	NO	
1. Vegetables			8
2. Meat			9
3. Margarine or oil			10
4. Milk			11
5. Sugar			12
6. Other, _____			13

14.2 Does your child eat meat, fish, chicken, eggs or milk every day?

1. Yes
2. No

14

14.3 If yes, when does your child eat these foods?

_____ per week

15-16

14.4 Does your child eat soya mince or baked beans in tomato sauce?

1. Yes
2. No

17

14.5 If yes, when does your child eat these foods?

_____ per week

18-19

14.6 How many glasses or bottles of water does your child drink per day?

_____

20-21

14.7 How many glasses or bottles of tea does your child drink per day?

_____

22-23

14.8 What kind of bread do you buy for your child?

1. White bread

24

2. Brown bread
3. Combination of the two
4. Other, specify _____

14.9 **Does your child eat the skins of fruit?** [ ] 25
1. Yes
2. No

14.10 **Does your child eat vegetables each day?** [ ] 26
1. Yes
2. No

14.11 **How many different kinds of vegetables does your child eat per day?** [ ] 27

_____

14.12 **Does your child eat fruit each day?** [ ] 28
1. Yes
2. No

14.13 **How many different kinds of fruits does your child eat?** [ ][ ] 29-30

_____

14.14 **Which of the following do you add to your child's food?**

(1= Yes, 2= No)	**YES**	**NO**	
Salt			31
Aromat			32
Beef stock blocks			33
Steak 'n chops			34
Chicken spice			35
Soup powder			36
Other, _____			37

14.15 **What do you use to prepare your child's food?**

(1= Yes, 2= No)	**YES**	**NO**	
1. Margarine			38
2. Oil			39
3. Animal fat			40
4. None			41
5. Other, _____			42

14.16 **Does your child eat sugar every day?** [ ] 43
1. Yes
2. No

14.17 **How many teaspoons of sugar does your child consume per day (added to all food and drink)?** [ ][ ] 44-45

_____

14.18 **What kind of sweets or cooldrinks do your child drink and eat?**

(1= Yes, 2= No)	**YES**	**NO**	
1. Sweets			46
2.Chocolates			47
3.Coke, fanta or other carbonated cool drinks			48
4.Cordials (oros, etc)			49
5.Biscuits			50
6.Cakes, doughnuts, etc.			51

14.19 **Does your child play outside each day?** [ ] 52
1. Yes
2. No

15	**Was this child previously admitted to hospital?** 1. Yes 2. No	53
15.1	**If yes, how often?** _____ **For what reason(s) was this child previously admitted?** _____ _____ _____ _____	54-55 56-57 58-59 60-61 62-63
16	**Who referred the child to the hospital?** **(1=Yes, 2= No)** 1. Nurse 2. Doctor 3. Dietitian 4. Other, please specify_____	**YES** / **NO** 64 65 66 67
17	**Who looks after the child during the day?** **(1=Yes, 2= No)** 1. Mother 2. Grandmother 3. Neighbour 4. Day Care Centre 5. Other, please specify_____	**YES** / **NO** 68 69 70 71 72
18	**What is the mother/ caregiver's highest level of education (grade)?** _____	73-74
19	**What is the mother's marital status?** 1. Single 2. Married / Traditional marriage 3. Divorced 4. Widowed 5. Other, _____	75
20	**Number of live births to the child's mother including this child?** _____	76-77
20.1	**Number of children deceased?** _____	78-79

20.2	**Give a reason for deaths as indicated above** _____	1-2

_____ 3-4

_____ 5-6

_____ 7-8

20.3 **Is this child the** 9
1. 1st child
2. 2nd child
3. 3rd child
4. 4th child
5. Other, specify _____

20.4 **If not the only child, have any of the other children ever been admitted to hospital?** 10
1. Yes
2. No

20.5 **If yes, provide a reason (s) for admittance to the hospital**

_____ 11-12

_____ 13-14

_____ 15-16

_____ 17-18

21 **With whom is the child staying most of the time?** 19
1. Parent / parents
2. Grandparents / grandparent
3. Aunt / uncle
4. Other family
5. Other _____

22 **What are the sources of income in the household?**

Source of income	Yes	No	
Salary/ Wage			20
Old Age Pension			21
Disability Grant			22
Child Support Grant			23
Other (please state) _____			24

22.1 **If yes to any of the above sources of income, how many people are receiving each of the following?**

Salary / wage		25
Old age pension		26
Disability grant		27
Child support grant		28
Other (please state)_____		29

22.2 **How many people depend on this income?**

_____ 30-31

22.3 **Who is the head of the household?** 32-33

_____

22.4 **How many rooms (except the bathroom) in the house are used for sleeping?** 34-35

_____

22.5	**How many people sleep in the house at night (> 5 days per week)?** _____	36-37
23	**Is the mother still alive?** 1. Yes 2. No	38
23.1	**If NO, who cares for the child?** _____	39-40

24	Mother's / caregivers Weight _____ kg Mother's / caregivers Height _____ m Mother's / caregivers Age _____ years	41-45 46-50 51-52
25	**Can the mother / caregiver correctly explain what diarrhea is?** **(use the nestle flipchart as visual aid for classification of diarrhea)** 1. Yes 2. No	53
26	**Has the mother / caregiver received VCT at any institution?** 1. Yes 2. No	54
27	**What is the mother / caregivers HIV status?** 1. Positive 2. Negative 3. Unknown 4. Does not want to reveal	55
28	**Is the child HIV+?** 1. Yes 2. No 3. Do not know 4. Does not want to reveal	56
29	**Does the mother/ caregiver or any other person in the household have TB?** 1. Yes 2. No	57
29.1	**If yes, specify** _____	58-59
29.2	**Does the child have TB?** 1. Yes 2. No 3. Do not know	60

30	**Is / was the mother / caregiver on any of the following treatment?** **(1=Yes, 2= No)**	**YES**	**NO**	
	1. HAART			61
	2. PMTCT			62

	3. TB 4. None 5. Other, _____			63 64 65
31	**What treatment does the child receive?** **(1=Yes, 2= No)** 1. HAART 2. PMTCT 3. TB 4. None 5. Other, _____	**YES**	**NO**	66 67 68 69 70
32	**Does the child have any other diseases?** 1. Yes 2. No			71
32.1	**If yes, specify** _____ _____ _____			72-73 74-75 76-77
33	**Does the mother / caregiver have any other diseases?** 1. Yes 2. No			78
33.1	**If yes, specify** _____ _____ _____			79-80 1-2 3-4
34	**Did the mother attend the Ante- Natal Clinic when she was pregnant with this child?** 1. Yes 2. No 3. Do not know			5
34.1	**If yes, how many visits?** _____			6-7
35	**Did the mother consume alcohol during pregnancy?** 1. Yes 2. No 3. Do not know			8
35.1	**If yes, how much?** **How many drinks per day** _____			9-10
35.2	**If yes, how often?** **How many times per week** _____			11-12

36	**Did the mother smoke / "snuff" during pregnancy?** 1. Yes 2. No	13

	3. Do not know	
37	**BIOCHEMICAL INFORMATION (IF AVAILABLE IN FILE)**	
37.1	**Serum Albumin (mg)** _____	[ ][ ][ ] 14-16
37.2	**Heamoglobin (mg)** _____	[ ][ ].[ ] 17-20
37.3	**Transferrin (mg/dL)** _____	[ ][ ][ ][ ] 21-24
37.4	**C-reactive protein (mg/L)** _____	[ ][ ][ ] 25-27
37.5	**Absolute CD4 Count (mm^3)** _____	[ ][ ][ ][ ] 28-31
37.6	**CD4 percentage** _____	[ ][ ][ ] 32-34
	COMMENTS _____ _____ _____	