PREVALENCE OF MALNUTRITION AND ASSOCIATED FEEDING PROBLEMS AMONG CHILDREN WITH CEREBRAL PALSY ATTENDING MUHIMBILI NATIONAL HOSPITAL,

DAR-ES-SALAAM, TANZANIA

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A Dissertation submitted in partial fulfilment of the requirement for degree of Master of Medicine (Paediatrics and Child Health) of Muhimbili University of Health and Allied Sciences

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CERTIFICATION

The undersigned certify that he has read and hereby recommend for acceptance by Muhimbili University of Health and Allied Sciences a dissertation entitled: **PREVALENCE OF MALNUTRITION AND ASSOCIATED FEEDING PROBLEMS AMONG CHILDREN WITH CEREBRAL PALSY ATTENDING MUHIMBILI NATIONAL HOSPITAL, DAR-ES-SALAAM, TANZANIA IN 2010,** in partial fulfilment of the requirements for the degree of Master of Medicine (Paediatrics and Child Health) of the Muhimbili University of Health and Allied Sciences (MUHAS).

Dr Maulidi R. Fataki
(Supervisor)

Date.....17/11/2010.....

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Last but not least, I would like to convey my sincere gratitude to my loving husband Dr. Ruggajo, P.J and our lovely daughter Adrianna-MkaKaya for their constant encouragement and inspiration during the dark hours of this work.

DEDICATION

To my Father, the late Ashendumi Sissya
You will always be my North Star.
To my Mother, Hyasinta G.Mosha
I will never forget you.
To my daughter, Adrianna-MkaKaya
May the Lord enlighten your path.

ABSTRACT

Background: Cerebral palsy (CP) is a chronic disorder of posture and movement associated with other disabilities. Among these is failure of normal development of feeding ability which leads in slowness and inefficiency in feeding. As a result of the feeding problems children with cerebral palsy are at increased risk of inadequate intake of nutrients compared to the general population of children. Ultimately they are at a higher risk of developing malnutrition.

Objectives: To determine the magnitude of malnutrition and associated feeding problems among children with cerebral palsy who were attending at Muhimbili National Hospital, Dar-es-Salaam, Tanzania.

Methods: A descriptive cross-sectional study was conducted for a period of six months. During the study period, 239 children with cerebral palsy were consecutively recruited. Their nutritional status was assessed clinically and using anthropometric measurements (weight and length/height). Malnutrition was categorized using Waterlow classification.

Results: A total of 239 children with CP aged between three months and 14 years were recruited. Among them 146(61.1%) children were males. Spastic type of motor disorder was the most prevalent (56.1%) type of cerebral palsy. About half (50.2%) of the children had severe type of CP. Among the enrolled children 73.1% had variable degrees of wasting. The prevalence of severe wasting was found to be 20.1% while severe stunting accounted for 49.4%.. There was a statistical significant direct relationship between the severity of CP and severity of malnutrition. Severe wasting prevailed more among children with severe CP accounting for 28.3 %(p=0.001). More children with dyskinetic/athetoid type of CP 15(28.6%) had severe wasting. However, this finding was not found to be statistically significant (p=0.616).

Feeding problems commonly noted among the children were inability to self feed 133(73.5%), inability to chew solids 80(34.3%), swallowing problems 71(29.7%), cough/chocking during feeding 65(27.2%), cry/extensor dystonia during feeding 52(21.8%) and inability of tongue lateralization in 47(20.2%) of children.

In general, majority of the children, were taking less than 30minutes time to finish a feed. However, for those children who were taking longer time, more than 30 minutes to more than one hour to finish their feeds, majority were children with feeding problems. This was significant in children who were unable to feed themselves, inappropriate wide mouth opening, inability to

open mouth when fed, inadequate/absent tongue lateralization, no closure of lips around the spoon, chewing problems and cough/chocking during feeding.

The presence of feeding problems was associated with increased prevalence of severe wasting (11.1%-45.5%) and stunting (52.5%-72.3%).

Illiteracy of the mothers/caregivers was associated with having more children who were wasted to variable degrees (p=0.044).

Conclusion: This study has shown that among this selected population of children with CP attending MNH, the prevalence of malnutrition was relatively high. The severity of malnutrition was found to be directly related to the severity of CP and the presence of feeding problems.

Recommendation: Growth monitoring among children with cerebral palsy should be intensified in order to identify malnutrition and feeding problems early on, and instituting appropriate feeding programmes, nutritional counselling and follow-up actions.

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ABBREVIATIONS

CP- Cerebral Palsy

GERD- Gastro Oesophageal Reflux Disease

HFA- Height for Age

MNH –Muhimbili National Hospital

MUAC- Mid-Upper Arm Circumference

OMD- Oral-Motor Dysfunction

OFC- Occipital Frontal Circumference

SD- Standard Deviation

UMNL- Upper Motor Neuron Lesion

WFL- Weight for Length

WFH- Weight for Height

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1.0 INTRODUCTION AND LITERATURE REVIEW

1.1 BACKGROUND

Cerebral palsy is a well recognized neurodevelopmental disability. It can start in utero or early childhood and persists throughout the lifespan. It was first reported by Little in 1861 (originally called cerebral paresis).[1] Little thought of asphyxia during birth as a chief cause of the disorder of mental and physical disability, especially in relation to deformities which were seen in these children. Cerebral palsy has been studied by some of the most eminent medical minds of the past hundred years. At the beginning of the 19th century Sigmund and Sir William Osler both contributed important perspectives on the condition.[2, 3]

DEFINITION

There have been many definitions of cerebral palsy.[4, 5]The most recent definition describes it as, *a group of disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive brain damage that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour, epilepsy and secondary musculoskeletal problems.*[6]

1.2 CLASSIFICATION OF CEREBRAL PALSY BY AMERICAN ACADEMY FOR CEREBRAL PALSY

The classification of CP by the American Academy for Cerebral palsy identifies three key components. These components are based on the **type of motor disorder, anatomical distribution** and **severity** as shown in Table 1 below:

Table 1: Classification of cerebral palsy

Type of motor disorder	Anatomical distribution	Severity
1.Spastic-(UMNL)	1.Monoplegia	1.Mild
2.Athetoid-(Basal ganglia)	2.Diplegia	2.Moderate
3.Rigidity- (Extra pyramidal)	3.Hemiplegia	3.Severe
4.Ataxia-(Cerebellum)	4.Triplegia	
5.Atonic-(Generalized)	5.Double hemiplegia	
6.Mixed	6.Quadriplegia	

Type of Motor disorder

Based on the type of motor disorder the classification includes;

1. Spastic type of motor disorder results from lesions that damage the motor neurones or the pyramidal tract, and presents as an upper motor neuron lesion. There is spasticity of muscles of the 'clasp knife' variety. There is spastic posture of the limbs, hyperreflexia, (exaggerated deep tendon reflexes), sustained ankle clonus and extensor plantar response after the age of two to three years

2. Extrapyramidal lesions - **Dyskinetic CP (Dystonic and Choreoathetoid CP)**

In this category there is muscle stiffness of the 'lead pipe' or 'cogwheel' in nature and athetoid dystonic posturing.

- **Dystonic** type – there are involuntary movements caused by sustained or intermittent muscle contractions leading to repeated writhing motions and/or abnormal postures
- **Choreoathetoid-** There are irregular, slow, writhing or rotating movements which are course, involuntary in nature and usually disappear during sleep. These movements involve the limbs, trunk and face.

3. Cerebellar lesions leading to ataxic type of motor disorder presenting with disorders of balance and coordination

4. Hypotonic type of motor disorder presenting with generalized hypotonia in early infancy and often evolves in some other type of motor disorder as the child grows older

5. Mixed type - In this category there is more than one motor disorder .It can be a mixture of spasticity, rigidity and or athetosis

Anatomical distribution

In this classification there are 6 subgroups. They are;

1. Hemiplegia: This involves two limbs on the same side of the body. The upper extremity being more involved than the lower.

2. Quadriplegia: All four limbs are affected equally

3. Double hemiplegia: All four limbs are involved, however the upper limbs are more involved than lower limbs

4. Diplegia: It involves all four extremities. The lower extremities being more affected than the upper extremities.

5. Monoplegia: Is the involvement of one limb only

6. Triplegia: Is the involvement of three limbs

Severity of cerebral palsy

Severity of cerebral palsy depends on the type of motor disorder and anatomical distribution:

Mild

Hemiplegia- The affected limb is used independently for everyday activity.

Double hemiplegia - Some use of the upper limbs is retained

Diplegia – There is clumsy gait rather than disabled

Ataxia – Patient is clumsy but can walk independently

Moderate

Hemiplegia – The affected hand is used only to assist the unaffected hand

Double hemiplegia- Some use of the lower limbs is retained.

Diplegia – There is unsteady walk and inability to run

Ataxia- Needs some assistance in everyday activities and may need some support for walking.

Severe

Hemiplegia- The affected hand has no useful function except perhaps as a prop support.

Double hemiplegia- No useful function in activities of the daily living hence these children are totally dependent on others.

Diplegia- Walking can occur only by assistance

Ataxia- Needs total assistance even in the simplest daily activities, they cannot walk even with support.

1.3 AETIOLOGY AND RISK FACTORS OF CP

The causes and risk factors for developing cerebral palsy involve damage to the brain either during the prenatal, perinatal or postnatal period. In some cases it may be difficult to identify clear-cut risk factors hence the cause cannot be identified very easily.

In developed countries prenatal causes accounts for the majority of the cases. This is due to the increased survival of very small infants resulting from improved neonatal intensive care units.[7]

In developing countries on the other hand, perinatal and postnatal factors account for the majority of causes of CP. This is due to poor health infrastructure resulting in poor antenatal and obstetrics care. Perinatal factors are due to prematurity, low birth weight and birth related problems in full term infants. In a recent study done in South Africa it was shown that approximately 80% of factors are postnatal, mainly CNS infections (e.g. meningitis, cerebral malaria) and prolonged convulsions.[8-11]

Maternal risk factors for CP include: multiple foetuses, malnutrition, placental problems/infection (e.g. unhealthy placenta, haemorrhages chorioamnionitis).Other maternal risk factors include; maternal infections e.g. cytomegalovirus and rubella, toxin ingestion (e.g., heavy metals like lead and mercury), obstructed labor, hypothyroidism, cigarette smoking and alcohol ingestion.[12-20]

Infant characteristics associated with risk of developing CP include, prematurity, low birth weight, low Apgar score less than 7 at 5 minutes, periventricular haemorrhage, birth asphyxia, neonatal infections, birth trauma, haemolytic disease of newborn, neonatal hyperbilirubinaemia, breech presentation, neonatal seizures and twin/multiple pregnancy.[21-24]

Familial or genetic causes are thought to contribute to approximately 2% of cases in the European populations. The prevalence of cerebral palsy in Asian population in the United Kingdom has been reported to increase by two-folds up to 6.42 cases per 1000 birth. Majority of the affected children were from consanguineous marriage strongly suggesting the existence of a recessive form of CP.[25-27] In one study unknown causes for cerebral palsy accounted for 37%.[28]

1.4 PREVALENCE OF CEREBRAL PALSY

The prevalence of cerebral palsy in western nations ranges between 1.5 to 2.5 per 1000 live births with the exception of Turkey which has a higher prevalence of 4.4 per 1,000 live births, however, data from the Americas are sparse.[29-33]

In developed countries the prevalence of cerebral palsy has been declining. This is due to an increase in health care facilities accompanied with the improvement of neonatal intensive care services. In some developed countries, there is an increase in the prevalence of cerebral palsy, this may be attributed to increased survival of preterm and low birth weight infants owing to better obstetric and neonatal care, and to further improvement in the survival of very small babies receiving intensive care.[34,35] The significant decline in cerebral palsy rate in preterm infants born in Eastern Denmark has been due to a change in treatment at the neonatal intensive care units using less mechanical ventilation.[36]

In contrast in to developing countries, particularly in sub-Saharan Africa child birth is associated with a relatively significant morbidity and mortality both for the mother and the newborn resulting from inadequate quality of care.

Studies done in Cape Town, South Africa the prevalence of cerebral palsy is at 1.25 per 1000live births. While in Manguzi sub district of KwaZulu –Natal cerebral palsy prevalence was as high as 10 per 1000.[37,38] Prevalence of cerebral palsy in Tanzania is unknown but it is estimated to be relatively much higher because of the prevailing high perinatal morbidity and mortality.

1.5 THE DEVELOPMENT AND EATING SKILLS IN INFANTS AND YOUNG CHILDREN

In normal infants and young children, the process of learning to eat is a complex one. At physiologic level, feeding relies on two interrelated factors. The factors are the structure and physiological function of the mouth and pharynx. The basic physiologic complexity of feeding is further compounded by social and cultural aspects. The development of feeding skills is therefore a progression of physiologic tasks and behaviour which are gradually learned in a social context.

1.5.1 Functional anatomy of the mouth and the pharynx

Structural integrity of the mouth and pharynx is necessary for normal feeding. Anatomically the pharynx is made up of three compartments; the nasopharynx, oropharynx and the hypopharynx. The oropharynx and hypopharynx form part of the alimentary and respiratory tracts. These compartments are involved in three categories of motor function; stabilization and maintenance of structural position and form, alimentation and respiration. While these categories of function share the common anatomic structures and motor units, each has its own network of neural control mechanisms and its own calendar of development.[39]

1.5.2 Swallowing physiology

1.5.2.1 Infant sucking, swallowing and breathing

Feeding in the newborn infant differs from the mature feeding of older children.[40] The mature feeding involves multiple methods of feeding and manipulation of food in the oral cavity. Feeding methods involve use of spoon, cups etc, and food manipulation involves chewing. Infant feeding involves a single method which intertwines sucking, swallowing and breathing. The newborn infant feeding is a reflex bound phenomenon and automatically makes certain oral movements e.g. rooting reflex which helps the baby to latch onto the breast or bottle (Table 2). Some reflexes such as rooting and sucking may depend on hunger or satiety, the taste or level of alertness, or the infant's neurologic status.[41]

Table 2: Feeding reflexes present in the newborn infant as described by Richard D, Steven and Janet H, Allaire. [42]

Reflex	Stimulus	Response	Time of disappearance
Suck-swallow	Stroke anterior third of the tongue or center of the lips.	Suckle/suck, swallow.	4 months
Rooting	Stroke around the mouth.	Move head towards the source of the stimulus, latch on to the nipple	3-4 months (later if breastfed)
Biting	Stroke the gums.	Rhythmic up and down biting motions of the jaw.	6 months.
Gagging	Mid-third potion of the tongue.	Gag, eye widening.	Moves to posterior third of the tongue at 7months.
Babkin's	Stroke firmly the palms of both hands	Mouth opens, head flexes and rotate to midline	3-4 months

1.5.2.2 Suckling

The muscles of the tongue provide a peristaltic action which compresses the nipple in a wave like fashion and allows the infant to get the milk out of the breast. At birth the infant's anatomical structure dictates that the tongue moves in an extension –retraction pattern because of the limited room within the oral cavity. This initial in-and –out pattern is called suckling and resembles a lick-suck.[43] A variation on this basic, reflexive suckle pattern is recognized as a true sucking. Most infants utilize a mixture of suck and suckle until 6 months of age. By 6 months the true suck predominates, as it is generally more efficient.[43]

1.5.2.3 Swallowing

Moving the liquid bolus (milk) via the tongue from the front to the back of the oral cavity appears to be a continuous motion which begins with sucking. During this stage the bolus is propelled not only by muscle action but also by the changing pressure gradient through out the mouth, pharynx and oesophagus.

1.5.2.4 Breathing

Respiratory rate is closely co-ordinated with swallowing and must be altered during feeding because of obligatory periods of apnoea which occur with swallowing. During much of infant feeding the suck, swallowing and breathe triad are stable in a 1:1:1 ratio. Co-ordinated breathing in an expiration-inspiration pattern is a necessary requisite to successful feeding.

1.5.2.5 Mature eating and swallowing

This has four phases; **oral-preparatory, oral, pharyngeal** and **oesophageal phase.**

A total of 31 pairs of striated muscles are involved during various phases of swallowing. These muscles are important both as structural and functional elements. The muscles are innervated by mandibular branch of trigeminal nerve, cranial nerves VII, X, XII and ansa cervicalis (C1-C2).This allows for multiple methods of eating and manipulation of food in the oral cavity.

Maturation of feeding skills over the first few years of life is brought about by a change in anatomic relationship of the mouth and pharynx due to physical growth. It is accomplished largely by developmental changes in the CNS coupled with experiential learning.

The collection of food, liquid or saliva into a swallow-ready bolus requires preparation by the tongue, jaw, teeth, palate and the lips.[43-45] Solid foods are lateralized by the tongue to the grinding surface of the teeth, masticated, mixed with saliva and drawn together. When this preparation is complete, the three phases of swallowing begin as outlined in Table 3.

Table 3: Mature swallowing behaviour by Richard D, Steven and Janet H, Allaire [46]

Stage	*Structures*	*Activities*	*Time*
Oral preparation (voluntary)	Lip, tongue, cheeks, palate	Lip close, food mix with saliva and masticated.	Variable depends on substance
Phase I Oral phase (voluntary/involuntary)	Tongue	Moves bolus posteriorly, triggers swallow reflex at the pharyngeal wall.	Less than 1 second
Phase II Pharyngeal phase (involuntary)	Velum, Pharyngeal constrictor, epiglottis, laryngeal strap muscles arytenoids mass, upper oesophageal sphincter	Velum elevates Pharyngeal peristalsis Epiglottis closes, larynx closes, elevate and draw forward Upper pharyngeal sphincter relaxes	1 second or less
Phase III Oesophageal phase (involuntary)	esophagus	Enters esophagus Moves to stomach	8-20 seconds

1.5.2.6 Neurophysiologic control of swallowing

Deglutition or swallowing is defined as the semiautomatic motor action of the muscles of the respiratory and gastrointestinal tract to propel food from the oral cavity to the stomach through the oesophagus.[47] This motor action is stimulated and modified by sensory stimuli from the mouth, pharynx and larynx as well as from CNS. The physiology and neurophysiology of swallowing is quite complex. The neural control of swallowing involved three components;

- Afferent sensory fibers contained in four cranial nerves which are; mandibular branch of trigeminal nerve (V_3), facial, glossopharyngeal and vagus nerve.

- Efferent motor fibers contained in five cranial nerves (V_3, VII, IX, X, XII) and two cervical peripheral nerves (C1, C2).
- Paired brain stem swallowing centres in the Pons, the limbic-hypothalamic system, the cerebellum and the prefrontal cortex.[47,48]

The brainstem swallowing centers integrate and process various input signals from oropharyngeal sensory fibers and higher CNS centers. The brainstem then orchestrates the swallowing process via efferent motor fibers in the cranial nerves.

The neurophysiologic control of the oral-preparatory stage involves the volitional use of several cranio-facial muscles.[49] Passage of the bolus to the posterior portion of the oral cavity initiates the involuntary pharyngeal phase with the concomitant inhibition of respiration. The pharyngeal phase consists of a rapid, stereotypic sequence of events. The neurophysiologic control of this involuntary phase is currently best explained by the central pattern generator hypothesis with modification by peripheral feedback.[49]

1.5.3 Feeding in a normal child

Oral feeding for the newborn infant is entirely reflexive. Rooting, nipple latching, sucking and swallowing do not appear to require suprabulbar activity. Immediately after birth, however, the learning process begins with its dependence on experiential opportunities, sensory input and suprabulbar neurologic maturation. Suprabulbar mechanisms utilize the resources of medullary swallowing centres to accomplish qualitative different feeding tasks, this is called encephalization.[50] These suprabulbar areas interpret the sensory input and exert their higher level of volitional control on the brainstem motor centre, as a result, the older infant and young child acquire the ability to evaluate the physical character of food, manipulate it appropriately and voluntary ingest it.[50]

Growth and neurologic maturation play a role in feeding development, however experiential learning is crucial through sensation and sensory feedback. Other important factors contributing to learning are gross motor and fine motor development, the method of food presentation and cognitive development.

1.6 THE CAUSES OF FEEDING DIFFICULTIES IN CHILDREN WITH CEREBRAL PALSY

Feeding in a normal child is characterised by a progression from reflex-effected suckling followed at around 4 months by gradual introduction of puréed and later solid and lumpy foods (transitional feeding). At about 24 to 30 months, the child is a relatively skilled tool user and can feed completely independently. The development of hand-regard from about 3 months of age is a component of integrating upper limb function into the infants body image. It is a prelude to using visually directed manual assistance in feeding. At around 5 months the infant will start to hold the feeding bottle with both hands, at 6 months she will reach out and grasp a rusk. However not until about 8 to 9 months will be able to finger feed effectively. At this age, the child begins to understand that the spoon and the food go together and may even guide her mother's hand during feeding. At around 15 months of age, the child is able to grasp the spoon and self feed, but rather clumsily.[51]

This natural progression does not happen in children who have CP or other neurological disorders, for several possible reasons.

The underlying neuropathology of OMD is a degree of pseudobulbar palsy or of bulbar palsy and these may coexist. They may be of prenatal, natal or postnatal origin. Failure of normal development of feeding ability and inadequate intake of calories may lead to under malnutrition. About 20% of all disabled children may have an inadequate intake of food.[52] The loss of lean muscle mass in disabled children is probably related more to immobility rather than to inadequate protein intake. Malnutrition in CP children, therefore, is largely a function of an insufficient amount of food being consumed rather than poor quality of food offered. In non-ambulatory disabled children without significant OMD, obesity may become a serious handicap both to themselves and their caretakers.

Although many individuals exhibit more than one disability, the primary cause of inadequate calorie intake in disabled children is slowness and inefficiency in feeding. Feeding efficiencies of less than 10% is a common phenomenon usual in severely disabled children. Patrick and Gisel, observe that CP children take longer up to 10 times longer for their daily feedings than normal children.[53, 54, 55] Time is required to support a normal body and a normal metabolism, while prolonged feeding times may compensate for feeding inefficiency when the disabled child is small, however as the body size increases a point is reached at which no further compensation is possible and growth is limited by energy

intake. Reports have shown that mothers of disabled children spend longer periods to feed their children. Reilly and Skusse assessed infants with moderate to severe OMD and marked failure to thrive. They found that the mean duration of meal-times did not differ from that of controls.[53, 56,57] The dietary intakes of the two groups, however, didn't differ significantly despite the fact that disabled children were offered and consumed less food.[58] This observation of inadequate amounts of food being presented possibly reflects the situation for the majority of disabled children living in institutions with low caretaker levels. This is often associated with insufficient time to properly feed and assist the affected child. Total swallowing time was found to be longer for solid bolus than liquids, therefore feeds for these children should consist mainly of liquid and semi-solid foods.

Some of the factors that can contribute to ineffiency in feeding and growth impairment include; oral-motor dysfunction, disruption of sensitive learning period, dystonia, postural deformity, impaired hand function, immobility. Others are visual impairment, deafness, gastro-oesophageal reflux, recurrent aspiration, dental problems, communication difficulties and behavioural problem.

1.7 CONSEQUENCES OF FEEDING DIFFICULTIES EXPERIENCED BY CP CHILDREN

Breathing and eating are two most fundamental and vital activities of human beings. The anatomical structures which subserve the functions of sucking, chewing, swallowing and respiration are in close proximity to provide a conduit for food and air. They require complex and highly integrated levels of neuromuscular control. Coordination of sucking, chewing, swallowing and breathing is essential for the prevention of aspiration of food, swallowing of air and for efficient intake of nutrients.

1.7.1 Disability and feeding

The development of oral-motor skills mirror general neurological maturation. In order to attain optimum oral-motor skills a child must have the ability to move oral and facial structures independently of the rest of the body.[59]Control of the axial skeleton is also important, as proper body position is essential to promote normal feeding.[51,60] Furthermore, in order to become competent feeders, children must be able to use their arms and hands adroitly, with coordinated hand-to-mouth movements and be sufficiently dextrous to use

forks, spoons or chopsticks as extensions of their own hands. Insults to the developing brain can have dramatically deleterious effects on a child's ability to feed. Studies have found out that 33-80% of children with CP are unable to feed by themselves.[61,62] Lack of independent mobility and inability to ask for food or express preferences further contributes to the difficulties that disabled children have in obtaining an adequate intake of nutrients. In addition, behavioural problems are common in disabled children, and mealtimes may become a focus for the expression of these, thus further compromising feeding.[63, 64, 65]

The major consequences of feeding difficulties experienced by both, children with CP and parents/caregivers are social and nutrition.

1.7.1.1 Social consequences of feeding difficulties

We eat not simply for the purpose of growth and maintenance of our bodies but also as a fundamental social activity shared with others. Failure of an infant to suck properly may be the mother's first indication that there is something wrong with her child.[53,66] Parents need to adapt to child's feeding difficulties. This is an ongoing process throughout the child's life rather than an event accomplished in the first months after diagnosis.[67] Some parents may lack the necessary personal skills to achieve this adaption.

Early in the development children spend a great deal of time exploring the environment with their mouths. This exploration serves a dual purpose. Firstly, to gather information from the outside world, and secondly, to desensitise the mouth to different textures and tastes.[59] Children with abnormal tone and abnormal facial sensitivity respond to the touch of food and feeding utensils with increased muscle tone and abnormal oral patterns. These abnormal reactions make food intake extremely difficult and increase the risk of chocking and vomiting. Mealtimes with disabled children can be prolonged and stressful, with caretakers and children becoming frustrated and anxious.[56,58] Children with severe OMD can take up to 6 to 18 times longer to eat a mouthful of food than normal children.[53,68, 69]

On average, mothers of disabled children spend three and a half hours per day feeding their child as compared to 0.8 hours for mothers with normal children. Some mothers spend over seven and a half hours a day trying to feed their disabled children.[56] Even long meal-times sometimes do not compensate for the severity of these children feeding impairment and they frequently become malnourished.

Feeding disabled children therefore can be unrewarding and unenjoyable, especially with children who have a tendency to vomit on themselves and to their caretakers.[60] Mothers may be unable to leave their disabled children to anyone's else care for the fear that they will be unable to cope at meal times. The difficulties in feeding such a child impose severe restrictions on the activities and opportunities for the mother both within and outside the home.[70]

1.7.1.2 Nutrition consequences of feeding difficulties

Around 13-52% of disabled children are significantly undernourished.[52,71-73]In majority of the studies growth retardation was most closely associated with inadequate intake as a result of self feeding impairment and oral-motor dysfunction. The association between the severity of CP, dysphagia and oral-motor dysfunction has been clearly demonstrated.[74,75]

The suboptimal nutrition resulting from inadequate intake is not without its consequences. In addition, to linear growth failure there is a decrease in muscle strength which in the respiratory system reduces the effectiveness of the cough reflex and predisposes to aspiration pneumonia. In the limbs it causes an increased in circulation time, causing coldness and mottled peripheries frequently seen in wasted CP children.[76-78] Under nutrition decreases cerebral function with possible exacerbation of the existing neurological impairment. Chronic undernutrition may be associated with increased irritability, and decreased motivation and energy available for activities such as play and rehabilitation.[79,80] Disturbances in the immune system secondary to malnutrition predispose these patients to infection, particularly of the lungs and urinary tract. Furthermore, healing of pressure sores is delayed in children with malnutrition. In view of such diverse complications, it is apparent that correction of malnutrition is central to the management of neurologically disabled children.

Children with mild CP who can walk are usually below average height, but aren't malnourished. Very dependent disabled children, however, are short, underweight for stature, with wasted muscles and reduced skinfold thickness.[81] There is abundant evidence that nutritional factors have a role in the growth failure and mortality of children with CP.[72, 78, 82, 83] The magnitude of the impact of nutritional status on linear growth is also proportional to disease severity.[71,80,84-87]

Moreover, growth failure in this group of children is a result of probably multifactorial and not simply a matter of inadequate nutrition. This is indicated by a failure of aggressive nutritional therapy to reverse growth failure especially when instituted in older children even before they have lost growth potential following skeletal maturation.[73]

1.8 PREVALENCE OF MALNUTRITION AND ORAL-MOTOR DYSFUNCTION (OMD)

The prevalence of oral motor dysfunction in children with CP is about 80%. However a population based study done in Norway reported a low prevalence of 26%.[57,61, 88] OMD is prevalent in all forms of CP and its severity is associated with severe forms of CP.[89] Feeding problems in children with OMD ranged from 47.5% - 93%, and are more common in children with spastic quadriplegic CP, spastic diplegia, hypotonia and athetoid type.[57,88,91,93] The relationship between OMD and growth retardation has been clearly outlined.[74]Krick et al, in their study, children with CP and oral-motor difficulties were found to be significantly thinner by age and height than their age-and sex-matched counter parts with CP but no oral-motor impairment. In recent studies, malnutrition in children with OMD has been found to be more common and accounts for 30- 44.8% of children with OMD while over nutrition was rare and present in 4%-9%. [61,90,91,93] Majority of children were wasted according to weight for height. They ranged from 10%-90%, while 22%-46% had low weight for age and 80% were underweight.[93, 94] Growth stunting was present in 30%-40% with height for age below 5th percentile.[61, 88, 92] Poor nutrition status was found to be associated with severity of motor impairment and was more seen in spastic quadriplegic cerebral palsy. Poor nutrition status of children with cerebral palsy was also found to be a summation of several factors.[89]

Oral neuromotor reflexes such as rooting, suckles feeding, gagging and biting are present at birth. They gradually mature so that by 3years of age they are similar to adult patterns. Changing from suckle feeding to transitional feeding occurs mainly as a result of CNS maturation.[50] Oral-motor deficits are associated with both sensory and motor dysfunction. Persistence of primitive oral reflexes will delay the development of mature oral-motor dysfunction.[95, 96]

Failure of development of mature feeding results to frequent spilling of most of the food offered to the children. It is estimated that as much as 11% of all food offered to the profoundly CP children is lost in spillage. CP children also have difficulty with the ignition of chewing. The delay from placement of food in the mouth until the onset of chewing may be a sensitive indicator of feeding impairment.[97] Other feeding problems which were encountered due to OMD were sucking 48%- 57%, swallowing 38%, inabilities in spoon feeding, rejection of solid foods, biting, chewing, cup drinking, straw drinking, chocking, and clearing of food in the mouth.[57,98,99] Dysphagia which interferes with the ability to ingest food was found in 76% in one study, profound dysphagia was present in 15% of the children and it was positively related to severity of motor impairments.[100]

Children with CP are known to have higher incidence of gastro oesophageal reflux disease (GERD), in one study it was found to be 67%, GERD predispose the children to recurrent chest infections.[101, 102]

The predictable deficits that are commonly associated with abnormal oral-motor development includes; poorly coordinated breathing, chocking, difficulties with sucking and swallowing, persistence of the bite reflex, tonic biting, jaw instability, jaw thrusting, tongue thrusting. Others are decreased tongue movements, failure of tongue lateralization, poor lip closure, lip retraction, drooling and hypo or hypersensitive gag response

1.9 OTHER ASSOCIATED DISORDERS

Children with CP may also present with other severe handicaps. These handicaps include epilepsy, mental retardation and behavioural problems.

Epilepsy has been found to occur in approximately 35-40% of patients with CP. It was found in all types of CP and all types of epilepsy can occur in CP children.

Mental retardation was seen in 66.7%. Behavioural problems have been found in about 57% and were thought to be even more in CP children with epilepsy.

Other associated severe handicaps include visual field deficits, strabismus, refractive errors, deafness, speech disorders and blindness. [75,103-106]

2.0: PROBLEM STATEMENT

In countries of sub-Sahara Africa, the prevalence of malnutrition is about 27% in the general paediatrics population. In Tanzania is about 22%. Given this situation therefore the prevalence of malnutrition in CP patients is expected to be even higher. The risk of developing malnutrition is compounded by feeding problems, neglect and stigma, total dependence on others for feeding and secondary emotional stresses experienced by parents when taking care of them. Lack of ability to self feed was found to be significant independent risk factor for developing malnutrition.

Oral motor dysfunction (OMD) in CP patients results in impaired muscular function that affects chewing and swallowing. This causes difficulty in coordinating their muscles in the tongue and mouth to chew and swallow correctly, speaking and facial posture. This then causes extreme difficulty when attempting to feed and leads to an increase in the incidence of malnutrition in individuals with CP.

Despite the causes of malnutrition in individuals with CP being a combination of factors as stated above, feeding problems due to OMD accounts for the majority of cases. The prevalence of OMD in children with CP was found to be 26%-90%. Feeding problems was found to occur in 47.5%-93% leading directly or indirectly to reduction in food and nutrient intake of cerebral palsy patients. Though the most prevalent type of malnutrition is undernutrition, over nutrition was found in 4%-9% of cases.

3.0 RATIONALE

Although it seems malnutrition is a significant public health problem among children with CP exact data on the magnitude of the problem in Tanzania has not been precisely documented and hence it is unknown.

This study would determine the prevalence of malnutrition and associated feeding factors, how feeding problems in children with cerebral palsy and other social factors predispose them to malnutrition. The data obtained in this study will help health care providers to know the prevalence of malnutrition and associated feeding problems in CP children. This data will also provide evidence-based data to help advocate for intervention measures need to be put in place to improve the quality of care provided to children with CP.

4.0 BROAD OBJECTIVE

To determine the prevalence of malnutrition and associated feeding problems among children with cerebral palsy attending cerebral palsy clinic at MNH, Dar es salaam, Tanzania.

4.1 SPECIFIC OBJECTIVES

4.1.1 To determine the prevalence of malnutrition among children with cerebral palsy attending MNH by age.

4.1.2 To determine the severity of malnutrition in relation to the type and the severity of CP.

4.1.3 To describe the types of feeding problems among children with cerebral palsy.

4.1.4 To assess the time it takes to feed an individual child per sitting among children with CP who have feeding problems.

4.1.5 To determine the association between severity of malnutrition and maternal socio-demographic factors.

4.1.6 To determine the association between severity of malnutrition and the types of feeding problems.

5.0 METHODOLOGY

5.1 Study design

A hospital based descriptive cross-sectional study.

5.2 Study duration

The study was carried out over a period of six months from May to October, 2009.

5.3 Study area

The study was conducted at the CP clinic and general paediatric wards at Muhimbili National Hospital (MNH). MNH is a tertiary care referral for Tanzania situated in Dar es Salaam. It also serves as a second level of referral for Ilala, Temeke and Kinondoni Municipal hospitals. It serves about 1000 outpatients per day and has a bed occupancy rate of about 1500 admitted patients per week.

The Department of Paediatrics and Child Health runs a specialized CP clinic that provides a specialized care to children suffering from neurological disabilities. It includes those with cerebral palsy, mental subnormality and seizure disorders once per week on Wednesdays. It is estimated that a total number of 700 children with CP are registered in the CP clinic.

The Annual report of the Department of Paediatrics and Child Health shows that from January to December 2008 a total of 1179 children with CP attended the clinic, among them 141 were new cases

Some of the children with cerebral palsy were also admitted in the wards at MNH. These were those who had various concomitant diseases conditions. In the year 2008 eighty four children were admitted.

5.4 Study subjects

Study subjects were all patients with CP attending CP clinic and those who were admitted in the general paediatric wards at MNH from May to October 2009.

5.5 Inclusion criteria

5.5.1 Children who had a definitive diagnosis of CP as documented in the files and attending CP clinic or admitted in the wards due to other associated health problems during the study period.

5.5.2 Children whose parents/caregivers granted informed consent to participate in the study.

5.6 Exclusion criteria

5.6.1 Children with CP whose parents/caretaker did not grant informed consent to participate in the study.

5.6.2 Children with CP and other concomitant disease conditions that were deemed very sick and needed emergency treatment on admission.

5.7 Sample size

Sample size was calculated using the formula shown below:

$$N = \frac{Z^2 P (1-P)}{E^2}$$

Z is percentage point corresponding to significance. If significance level is 5%, **Z** is 1.96

P is the prevalence of malnutrition in CP children in Portuguese which is 44.8 %(Campos et al [90])

E corresponds to maximum likely error and is 0.06

$$N = \frac{1.96^2 X 0.448 (1-0.448)}{0.06^2}$$

N = 264

Therefore the minimum sample size for the study was **264**

5.8 Sampling technique

Convenient sampling method was used until the anticipated sample size was reached. Eligible patients were consecutively enrolled. Study participants were recruited in the study on working days only from 08:00hours to 16:00 hours.

5.9 DATA COLLECTION PROCEDURES

A structured questionnaire designed purposely for the study was used. It was pre-tested and later used to collect the necessary information. The information collected included, patient's age, sex, parents/caregiver level of education etc (Appendix 2).

In order to help the principle investigator to carry out the study, two research assistants were identified and trained for data collection. They received a two days intensive training on general issues regarding research, research ethics and study specific procedures.

5.9.1 Physical examination

A thorough physical and central nervous system examination especially the motor component was carried out to every participant to confirm the type and severity of cerebral palsy and the associated disorders. All children were assessed for signs of malnutrition i.e. wasting, odema of both feet, and presence of dermatosis. Anthropometric measurements of weight and length or height were taken for each child.

5.9.2 Anthropometric measurements

5.9.2.1 Length/Height

Length was measured in children less than 2 years of age and those who could not stand for any reason. Height was measured in children aged 2 years and above who could stand.

Measurements:

Length

Child's shoes were removed, and laid supine on the length board. To make sure that the child laid straight on the measuring board, the assistant made sure that the child's head was in contact with the head piece. The principal investigator pressed her hands on the child's

knees/shins to make sure that child's legs were straight and the feet were flat against the foot piece at 90°. The principal investigator recorded the length of a child to the nearest 0.1cm.

Height

Child's shoes were removed and stood on a fixed foot piece. Assistant made sure that the child's heels and knees were straight and in contact with the height board. The principal investigator made sure that the child looked straight forward at 90° by touching the chin without bending the head. The head piece was then quickly slide down and height was measured by the principal investigator to the nearest 0.1cm

5.9.2.2 Weight

Instruments:

Salter hanging scale and a weighing sling were used to measure weight in children aged less than 2 years or those who could not stand because of the disability.

Beam balance was used for measuring weight in children aged 2 years and above who could stand.

The weighing machines were checked if they working properly before use.

Measurements:

Parents/guardian removed child's shoes.

The child was then suspended naked or with minimal clothing on the hanging scale.

The weight was recorded to the nearest 10gm

Using Beam balance a child stood straight with shoes removed and not touching the wall or any other thing.

The machine was checked if it is working properly before use. The reading was taken to the nearest 10gm.

5.9.3 Interpretation of Results

Waterlow classification (Table 4) was used to determine the nutritional status of each child [107]. It was based on weight-for-height charts. These Anthropometric reference charts have been adapted from WHO values for African region was used.

Table 4.Waterlow classification chart

	WFH (wasting)	HFA(stunting)	Z-Scores
Normal	>90%	>95%	(1SD-<-1SD)
Mild	80 - 90%	90 -95%	(-1SD-<-2SD)
Moderate	70 - 80%	85-90%	(-2SD-<-3SD)
Severe	<70%	<85%	(-<3SD)

5.9.4 DATA MANAGEMENT

5.9.4.1 Data entry and analysis

All questionnaires were daily checked for completeness and consistencies. Statistical Package for Social Scientists (SPSS) version 15 (Chicago IL) was used for data entry, cleaning and analysis. Frequency distribution and two way tables were used to summarize data. X^2 test and Fisher's exact test were used to determine the association between independent and dependent variables. P-values of less than 5% (0.05) were considered significant.

6.0 ETHICAL CLEARANCE

Ethical clearance for the study was sought from the Research and Publication Committee of MUHAS. Permission to conduct the study at MNH was granted by MNH administration.

6.1 ETHICAL CONSIDERATION

Whenever a child with CP was identified the purpose of the study was explained to the mother/caretaker. Study participants were enrolled after obtaining a written informed consent from their parents/caretakers.

Physical examination was conducted in privacy in the presence of parent/caretaker. Those who declined to grant consent were not in any way deprived of their right to receive medical care and advice.

For those children who were found to have severe acute malnutrition, the principal investigator facilitated their admission to malnutrition ward for appropriate management. Those who had moderate malnutrition received immediate nutrition counselling to provide an extra meal for the age, and arrangements were made with health care team to undertake follow-up for the nutritional problem besides CP.

Confidentiality was adhered to when filling the data collection sheets by using codes instead of names for identifying patients.

7.0 STUDY LIMITATION

This being a cross-sectional study it enabled an exploratory review of the extent of malnutrition among children with CP. Follow-up of the children was not possible.

A larger prospective interventional study would be more appropriate to identify effective interventional measures in our environment.

8.0 RESULTS

8.1 Baseline demographic characteristics

Between May and October 2009 a total of 239 patients were enrolled into the study. Among the 239 children, 93 (38.9%) were females and 146 (61.1%) were males. Their age ranged between three months and 14 years with median age of 35months.

Table 5: Baseline characteristics of patients with CP

Age group	Male	Female	Total
3 months – ≤12 months	23(67.6%)	11(32.4%)	34(14.2%)
>1 years – 2 years	26(55.3%)	21(44.7%)	47(19.7%)
>2 years – 3 years	28(63.6%)	16(36.4%)	44(18.4%)
>3 years – 5 years	31(60.8%)	20(39.2%)	51(21.3%)
>5 years	38(60.3%)	25(39.7%)	63(26.4%)
TOTAL	**146(61.1%)**	**93(38.9%)**	**239(100%)**

p=0.8422

Age range	3 months – 14years
Mean age ± (SD)	44.47 ± 32.92 months
Median	35 months

8.2 Type and severity of CP

Among 239 children with CP, spastic cerebral palsy was the commonest type of motor dysfunction accounting for 134 children (56.1%), followed by dyskinetic 53(22.2%) and mixed/unclassified 31(13.0%). Hypotonic/ataxic type was the least common accounting for 21 (8.7%).Approximately half of all the children studied had severe form of cerebral palsy accounting for 50.2% and majority of children with severe type of CP had dyskinetic/athetoid CP 32 (60.4%).

Table 6: Type and severity of CP

Type of CP	Severity of CP			Total
	Mild	Moderate	Severe	
Spastic	15(11.2%)	42(31.3%)	77(57.5%)	**134(56.1%)**
Dyskinetic/athetoid	1(1.9%)	20(37.7%)	32(60.4%)	**53(22.2%)**
Hypotonic/ataxia	1(4.8%)	13(61.9%)	7(33.3%)	**21(8.7%)**
Mixed/unclassified	16(51.6%)	11(35.5%)	4(12.9%)	**31(13.0%)**
TOTAL	**33(13.8%)**	**86(36.0%)**	**120(50.2%)**	**239(100%)**

Using Fisher's exact test p=<0.001

8.3 Prevalence of malnutrition among children with CP

Among 239 children with CP enrolled, 175 were malnourished to variable degrees giving an overall prevalence of 73.2%. Severe acute malnutrition (SAM) accounted for 20.1% and moderate acute malnutrition 53.1%. The majority of children with SAM were from the age group of 3 years or older up to 5yrs (33.3%).

Table 7: Age groups and prevalence of wasting

	Malnutrition(WFH)				
Age Group	**Normal status**	**Moderate wasting(MAM)**	**Severe wasting(SAM)**	**Total wasted**	**Total**
3 - 12mo	16(47.1%)	14(41.2%)	4(11.8%)	18(52.9%)	**34**
>1yr – 2yr	8(17.0%)	34(72.3%)	5(10.6%)	39(83.0%)	**47**
>2yr - 3yr	5(11.4%)	25(56.8%)	14(31.8%)	39(88.6%)	**44**
>3yr – 5yr	10(19.6%)	24(47.1%)	17(33.3%)	41(80.4%)	**51**
>5yrs	25(39.7%)	30(47.6%)	8(12.7%)	38(60.3%)	**63**
TOTAL	**64(27.1%)**	**127(53.1%)**	**48(20.1%)**	**175(73.2%)**	**239**

p= 0.000

Out of 239 children, 215 were stunted to variable degrees giving an overall prevalence of 90.0%. Almost half of the children with CP were severely stunted accounting for 118(49.4%).Majority of children with severe stunting were from the age group between above 3 years to 5 years 30 (58.8%).

Table 8: Age of the child and severity of malnutrition HFA (Stunting)

	Malnutrition(HFA)				
Age Group	**Normal status**	**Moderate stunting**	**Severe stunting**	**Total stunted**	**Total**
3 - 12mo	7(20.6%)	19(55.9%)	8(23.5%)	27(79.4%)	34
>1yr – 2yr	9(19.1%)	15(31.9%)	23(48.9%)	38(80.9%)	47
>2yr - 3yr	2(4.5%)	10(22.7%)	22(50.0%)	32(72.7%)	44
>3yr – 5yr	4(7.8%)	17(33.3%)	30(58.8%)	47(92.2%)	51
>5yrs	2(3.2%)	26(41.3%)	35(55.6%)	61(96.8%)	63
TOTAL	**24(10.0%)**	**97(40.6%)**	**118(49.4%)**	**215(90.0%)**	**239**

Using Fisher's exact test p= 0.023

8.4 Severity of CP and severity of malnutrition

Severity of wasting was associated with severity of CP. Variable degrees of wasting prevailed more in children with severe cerebral palsy accounting for 100(83.3%). Severe wasting prevailed more in children with severe cerebral palsy accounting for 34(28.3%).

Table 9: Severity of CP and wasting

	Malnutrition (WFH)				
Severity of CP	**Normal status**	**Moderate wasting**	**Severe wasting**	**Total wasted**	**Total**
Mild	11(33.3%)	20(60.6%)	2(6.1%)	22(66.7%)	**33**
Moderate	33(38.4%)	41(47.7%)	12(14.0%)	53(61.6%)	**86**
Severe	20(16.7%)	66(55.0%)	34(28.3%)	100(83.3%)	**120**
TOTAL	**64**	**127**	**48**	**175**	**239**

p= 0.001

Variable degrees of stunting prevailed more in children with severe cerebral palsy accounting for 111(92.5%). The same was for severe stunting whereby it prevailed more in children with severe cerebral palsy accounting for 75(62.5%).

Table 10: Severity of CP and stunting

	Malnutrition (HFA)				
Severity of CP	**Normal status**	**Moderate stunting**	**Severe stunting**	**Total stunted**	**Total**
Mild	4(12.1%)	21(63.6%)	8(24.2%)	29(87.9%)	**33**
Moderate	11(12.8%)	40(46.5%)	35(40.7%)	75(87.2%)	**86**
Severe	9(7.5%)	36(30.0%)	75(62.5%)	111(92.5%)	**120**
TOTAL	**24**	**97**	**118**	**215**	**239**

Using Fisher's exact test p=0.001

8.5 Type of CP and severity of malnutrition

The group of children with dyskinetic/athetoid CP had the majority 77.3% of children with variable degrees of wasting. Severe wasting was seen in 28.3%.

Table 11: Type of CP and severity of wasting

	Malnutrition (WFH)				
Type of CP	**Normal status**	**Moderate wasting**	**Severe wasting**	**Total wasted**	**Total**
Spastic	36(26.9%)	73(54.5%)	25(18.7%)	98(73.1%)	**134**
Dyskinetic/athetoid	12(22.6%)	26(49.1%)	15(28.3%)	41(77.3%)	**53**
Hypotonic/Ataxia	6(28.6%)	13(61.9%)	2(9.5%)	15(71.4%)	**21**
Unclassified/mixed	10(32.3%)	15(48.4%)	6(19.4%)	21(67.7%)	**31**
TOTAL	**64**	**127**	**48**	**175**	**239**

p=0.616

Among children with spastic cerebral palsy stunting of variable degrees accounted for 92.5%. Severe stunting was noted in 56.7% of the children.

Table 12: Type CP and severity of stunting

	Malnutrition (HFA)				
Type of CP	**Normal status**	**Moderate stunting**	**Severe stunting**	**Total malnourished**	**Total**
Spastic	10(7.5%)	48(35.8%)	76(56.7%)	124(92.5%)	**134**
Dyskinetic/athetoid	6(11.3%)	22(41.5%)	25(47.2%)	47(88.7%)	**53**
Hypotonic/Ataxia	4(19.0%)	11(52.4%)	6(28.6%)	17(81.0%)	**21**
Unclassified/mixed	4(12.9%)	16(51.6%)	11(35.5%)	27(87.1%)	**31**
TOTAL	**24**	**97**	**118**	**215**	**239**

Using Fisher's exact test p=0.129

8.6 Types of feeding problems

Feeding problems noted among children with CP were; inability to self feed, 133(73.5%), chewing, 80(34.3%), swallowing, 71 (29.7%). Others were cough/chocking during feeding 65(27.2%), cry/extensor dystonia during feeding 52(21.8%) and inability for tongue lateralization 47(20.2%). The least prevalent feeding problem was inappropriate wide mouth opening accounting for 9(3.8%).

Table 13: Types of feeding problems among children with CP

Feeding Problems	Frequency
Inability to self feed	133(**73.5%**)
Inappropriate wide mouth opening	9 (**3.8%**)
Inability to open the mouth when feeding	33 (**13.8%**)
Inability for tongue lateralization	47 (**20.2%**)
No closure of lips around spoon	30 (**12.9%**)
Inability to suck	1 (**4.3%**)
Chewing problems	80 (**34.3%**)
Swallowing problems	71 (**29.7%**)
Cough/ chocking during feeding	65 (**27.2%**)
Cry/ extensor dystonia during feeding	52 (**21.8%**)

8.7 Duration of feeding per meal

The majority of children regardless of presences or absences of a feeding problem were taking less than 30 minutes to finish their meals. However, children of those who were taking 30 minutes up to 60 minutes or longer to finish their feeds, were children with feeding problems.

Table 14: Time it takes to feed in children with feeding problems

	Time it takes to feed			
Feeding problems	**<30minutes (%)**	**30-60minutes (%)**	**>60minutes (%)**	**Total**
Inability to self feeding				
YES	88(66.2)	40(30.1)	5(3.8)	133
NO	41(97.6)	1(2.4)	0(0.0)	**(p=0.000)***
Inappropriate wide mouth opening				
YES	5(55.6)	2(22.2)	2(22.2)	9
NO	169(75.8)	50(22.4)	5(2.2)	**(p=0.003)***
Inability to open the mouth when fed				
YES	16(48.5)	15(45.5)	2(6.1)	33
NO	160(77.7)	36(17.5)	5(2.4)	**(p=0.001)***
Inadequate/absent tongue lateralization				
YES	28(59.6)	17(36.2)	2(4.3)	47
NO	146(78.5)	35(18.8)	5(2.7)	**(p=0.028)***
No closure of lips around the spoon				
YES	19(63.3)	7(23.3)	4(13.3)	30
NO	155(66.5)	45(22.3)	3(1.5)	**(p=0.002)***
Chewing problems				
YES	50(62.5)	26(32.5)	4(5.0)	80
NO	124(81.6)	25(16.4)	3(2.0)	**(p=0.006)***
Swallowing problems				
YES	48(67.6)	20(28.2)	3(4.2)	71
NO	126(75.0)	32(19.0)	4(2.4)	**(p=0.252)***
Cough/chocking during feeding				
YES	38(58.5)	22(33.8)	5(7.7)	61
NO	136(78.2)	30(17.2)	2(1.1)	**(p=0.034)***
Excessive vomiting				
YES	33(62.3)	16(30.2)	3(5.7)	53
NO	141(75.8)	36(19.4)	4(2.2)	**(p=0.083)***

*Fisher's exact test

8.8 Socio-demographic factors and severity of malnutrition in children with CP

Of the 239 children, majority of their mothers/caregivers were in the age group between 25yrs – 35years (61.1%). Mothers/caregivers with primary school education accounted for (61.5%), whereas two-thirds (67.8%) of the mothers/caretakers were married and about half (49.4%) of them were unemployed.

Maternal socio-demographics factors which were associated with high prevalence of variable degrees of wasting were mothers/caregivers aged 45years and above 9(75%),

illiterate mothers/caregivers 7(100%), mothers/caregivers who were unmarried 17(89.5%) and mothers/caregivers who were self employed 65(76.8%).

Table 15: Maternal socio-demographic factors and severity of wasting

	Malnutrition (WFH)				
Maternal Demographics	**Normal Status**	**Moderate wasting**	**Severe wasting**	**Total wasted**	**Total**
Maternal Age					
15 - <25yrs	13(26.0%)	26(52.0%)	11(22.0%)	37(74.0%)	50(20.9%)
25 - <35yrs	38(26.0%)	79(54.1%)	29(19.9%)	108(74.0%)	146(61.1%)
35 - <45yrs	10(32.3%)	16(51.6%)	5(16.1%)	21(67.7%)	31(13%)
≥ 45yrs	3(25.0%)	6(50.0%)	3(25.0%)	9(75.0%)	12(5.0%)
					p=0.987*
Maternal Education					
Illiterate	0(0.0%)	5(71.5%)	2(28.6%)	7(100%)	7(2.9%)
Primary education	32(21.8%)	80(54.4%)	35(23.8%)	115(78.2%)	147(61.5%)
Secondary education	28(35.9%)	39(50.0%)	11(14.1%)	50(64.1%)	78(32.6%)
College/university	4(57.1%)	3(42.9%)	0(0.0%)	3(42.9%)	7(2.9%)
					p= 0.044*
Marital status					
Not married	2(10.5%)	12(63.2%)	5(26.3%)	17(89.5%)	19(7.9%)
Married	46(28.4%)	85(52.5%)	31(19.1%)	116(71.6%)	162(67.8%)
Cohabiting	10(23.3%)	21(48.9%)	12(27.9%)	33(76.8%)	43(18.0%)
Separated/divorce	6(40.0%)	9(60.0%)	0(0.0%)	9(60.0%)	15(6.3%)
					p= 0.195*
Maternal occupation					
Employed	13(37.1%)	18(51.4%)	4(11.4%)	22(62.8%)	35(14.6%)
Self employed	20(23.3%)	45(53.5%)	20(23.3%)	65(76.8%)	86(36.0%)
Not working/student	31(26.3%)	63(33.4%)	24(20.3%)	87(53.7%)	118(49.4%)
					p= 0.468*

*Fisher's exact test.

Maternal socio-demographics factors which had the majority of children with high prevalence of variable degrees of stunting were mothers/caregivers aged 45years and above and illiterate mothers/caregivers accounting for 12(100%) and 7(100%) respectively. Separated/divorced mothers/caregivers and non working/students mothers/caregivers accounted for 14(93.3%) and 109(92.6%) respectively.

Table 16: Maternal socio-demographics factors and severity of stunting

	Malnutrition (HFA)				
Maternal Demographics	**Normal status**	**Moderate stunting**	**Severe stunting**	**Total stunted**	**Total**
Maternal Age					
15 - <25YRS	6(12.0%)	16(32.0%)	28(56.0%)	44(88.0%)	50(20.9%)
25 - <35YRS	16(11.0%)	63(43.2%)	67(43.8%)	130(87.0%)	149(61.1%)
35 - <45YRS	2(6.5%)	11(35.5%)	18(58.1%)	29(93.6%)	31(13%)
≥ 45YRS	0(0.0%)	7(58.3%)	5(41.7%)	12(100%)	12(5.0%)
					p=0.4679
Maternal Education					
Illiterate	0(0.0%)	3(42.9%)	4(57.1%)	7(100.0%)	7(2.9%)
Primary education	15(10.2%)	49(33.3%)	83(56.5%)	132(89.8%)	147(61.5%)
Secondary education	7(9.0%)	40(51.3%)	31(39.7%)	71(91.0%)	78(32.6%)
College/university	2(28.6%)	5(71.5%)	0(0.0%)	5(71.5%)	7(2.9%)
					p= 0.0171
Marital status					
Not married	2(10.5%)	8(42.1%)	9(47.4%)	17(89.5%)	19(7.9%)
Married	16(9.9%)	67(41.4%)	79(48.8%)	146(90.2%)	162(67.8%)
Cohabiting	5(11.6%)	16(37.2%)	22(51.2%)	38(88.4%)	43(18.0%)
Separated/divorce	1(6.7%)	6(40.0%)	8(53.3%)	14(93.3%)	15(6.3%)
					p= 0.9972
Maternal occupation	6(54.3%)	20(57.2%)	9(25.7%)	29(82.9%)	35(14.6%)
Employed	9(10.5%)	39(45.4%)	38(44.2%)	77(89.6%)	86(36.0%)
Self employed	9(7.6%)	38(32.2%)	71(60.2%)	109(92.6)	118(49.4%)
Not working/student					**p= 0.0057**

8.9 Common feeding problems and malnutrition

The presence of a feeding problem was associated with increased prevalence of wasting. Children with feeding problems with the exception to those children with inappropriate wide mouth opening and those who could not close their mouths around the spoon during feeding showed a lower prevalence of malnutrition compared to those without feeding problems.

Table 17: Feeding problems and severity of wasting

	Malnutrition (WFH)				
Feeding problems	**Normal status (%)**	**Moderate wasting (%)**	**Severe wasting (%)**	**Total wasted (%)**	**Total**
Inability to self feed					
YES	20(15.0)	75(56.4)	38(28.6)	113(85.0)	133
NO	22(52.4)	17(40.5)	3(7.1)	20(47.6)	**p=0.000***
Inadequate/absent tongue lateralization	9(19.1)	21(44.6)	17(36.2)	38(80.8)	47
YES	51(27.4)	104(55.9)	31(16.7)	135(72.3)	**p=0.012***
NO					
Inability to open the mouth when fed					
YES	1(3.0)	17(51.5)	15(45.5)	32(97.0)	33
NO	63(30.6)	110(53.4)	33(16.0)	143(69.4)	**p=0.000***
Chewing problems					
YES	13(16.3)	37(46.3)	30(37.5)	67(83.8)	80
NO	43(29.2)	86(58.5)	18(12.2)	104(70.7)	**p=0.000**
Swallowing problems					
YES	10(14.1)	37(52.1)	24(33.8)	61(85.9)	71
NO	54(32.1)	90(53.6)	24(14.3)	114(67.9)	**p=0.000**
Cough/chocking during feeding					
YES	7(10.8)	38(58.5)	20(30.8)	58(89.3)	65
NO	57(32.8)	89(54.2)	28(16.1)	117(70.3)	**p=0.001**
Cry/extensor dystonia					
YES	9(17.3)	27(52.0)	16(30.8)	43(82.8)	52
NO	55(29.4)	100(53.5)	32(17.1)	132(70.6)	**p=0.049**
Excessive vomiting/regurgitation					
YES	7(13.2)	33(62.2)	13(24.5)	46(86.7)	53
NO	57(30.6)	94(50.5)	35(18.8)	129(69.3)	**p=0.040**

*Fisher exact test

Presence of feeding problems was associated with increased prevalence of malnutrition of variable degrees. Children with inappropriate wide mouth opening and those unable to suck a lower prevalence of malnutrition compared to those without feeding problems

Table 18: Feeding problems and severity of stunting

	Malnutrition (HFA)				
Feeding problems	**Normal Status (%)**	**Moderate stunting (%)**	**Severe stunting (%)**	**Total stunted (%)**	**Total**
Inability to self feeding					
YES	5(3.8)	48(36.1)	80(60.2)	128(96.3)	133
NO	4(9.5)	20(47.6)	18(42.9)	38(90.5)	**p=0.88***
Inability to open the mouth when fed	1(3.0)	9(27.3)	23(69.7)	32(97.0)	30
YES	23(11.2)	88(42.7)	95(46.1)	183(88.8)	**p=0.154***
NO					
Inadequate/absent tongue lateralization	2(4.3)	11(23.4)	34(72.3)	45(95.7)	47
YES	19(10.2)	84(45.2)	83(44.6)	167(89.8)	**p=0.003***
NO					
Chewing problems					
YES	3(3.8)	22(27.5)	55(68.8)	77(96.3)	80
NO	16(10.9)	72(49.0)	59(40.1)	131(89.1)	**p=0.000***
Swallowing problems					
YES	3(4.2)	19(26.8)	49(69.0)	68(95.8)	71
NO	21(12.5)	78(46.4)	69(41.1)	147(87.5)	**p=0.000***
Cough/chocking during feeding					
YES	3(4.6)	26(40.0)	36(55.4)	62(95.4)	65
NO	21(12.2)	71(40.8)	82(47.1)	153(87.9)	**p=0.194***
Cry/extensor dystonia					
YES	4(7.7)	20(38.5)	28(53.8)	48(92.3)	52
NO	20(10.7)	38(41.2)	90(48.1)	128(89.3)	**p=0.843***
Excessive vomiting/regurgitation					
YES	4(7.5)	16(30.5)	33(62.3)	49(92.8)	53
NO	20(10.8)	81(43.5)	85(45.7)	166(89.2)	**p=0.104***

*Fisher's exact test

9.0 DISCUSSION

9.1 Prevalence of malnutrition

Among 239 children with cerebral palsy 175(73.2%) had wasting and 215(90.0%) had stunting of variable degrees. This is a relatively higher prevalence than that documented by Tanzania Demographics and Health Survey (TDHS) in 2004/05 whereby 3% of under five children had moderate/severe wasting and 38% had moderate/severe stunting. This much higher prevalence is because the variable degrees of wasting and stunting were clustered together irrespective whether they are mild, moderate or severe.

This study has shown that severe wasting was prevalent in 48(20.1%) of the children and this finding is similar to the findings of the study by Ifeyinwa et al and Thommessen et al studies, who obtained comparable results of wasting 16.3% and 15% respectively in children with CP.[108,109]Contrary to the findings of this study, higher prevalence were obtained by Dahl et al[91] (43%), Campos et al[90] (44.8%) and Troughton et al[110] (45.6 %.).The differences in prevalence wasting observed between the current study and the Dahl study can be explained by the difference in the selection of the study population. Dahl studied children with moderate to severe cerebral palsy. This subpopulation is known to have a higher prevalence of malnutrition. In this study children with mild CP were as well included. These children tend to have lower prevalence of malnutrition, hence accounting for the attenuation and overall lower rate.

In the Campos and Troughton studies, the study population were much older. The age range was 4-12 years and 2.6-18.7 years respectively. In the current study the age range was 3 months- 14 years. Wasting was less prevalent in children below 2 years as compared to the prevalence seen in those above 2 years, this again has led to the overall lower prevalence.[90, 110]

From this study moderate wasting accounted for 53.1% of children with CP. This prevalence is much higher than that reported by Hou et al, in 2004. He found out that the prevalence for moderate wasting was 22.0%. This low prevalence can probably be explained by the small sample size he used.[88]

In this study, approximately half of the children 118(49.4%) had severe stunting. This finding strikingly comparable to that observed by Thommessen et al, were they found 48% of the children had growth retardation.[109]

In Nigeria, Ifeyinwa et al,[108] observed a lower rate of stunting (9.2%). The possible explanation for the differences observed between his study and this study may stem from the fact that almost half (47.9%) of his study population were infants .Stunting usually assesses the chronicity of malnutrition which tends to occur with advancing age.

Moderate stunting was found in 40.6% of all children with CP. Almost similar findings were reported by Hou et al whereby 32.2% of his study population had moderate stunting.[88]

Severe malnutrition of all types prevailed in children with age between above 36 months and 60 months. Severe wasting and severe stunting accounted for 33.3% (p=0.000) and 58.8% (p=0.023) respectively. Ifeyinwa et al found out that, severe wasting was more prevalent between the age groups 13 months- 24 months accounting for 67.7%. Stunting was more prevalent in the age groups 13 months -18 months accounting for 73.3%.This can be explained by the characteristics of his study population. Nearly two thirds (62.2%) of his study population were children below 18 months.

9.2 Severity of CP and severity of malnutrition

The most common type of CP was spastic cerebral palsy accounting for 56.1% of all children with CP. Many studies have found out the same, that spastic CP is the leading type of motor disorder [91,106,111,]

The magnitude of spastic CP in this study is comparable to a study done by Dahl. [111] He reported almost a similar magnitude of 56.7%. A study done by Karumuna reported a higher magnitude of 76%.These higher differences between the current study and the previous two studies can possibly be explained by differences in assessment criteria used classify CP. [106]

In this study about half of all children with CP had severe CP accounting for 50.2%. Majority of children with severe type of CP were those with dyskinetic/athetoid CP accounting for 60.4%(p=0.000). A comparable distribution was found by M.Thommessen et al whereby 52.4% had severe type of cerebral palsy. Dahl et al out that majority of children with dystonia/dyskinetic CP had severe type of CP accounting for 90%.[109,111]

Severity of stunting was found to be directly related to severity of CP. Seventy five children (62.5%) had severe stunting (p=0.002).This finding is comparable to that found by Dahl who found that among children who were severely disabled, 72.2% of them were undernourished.[91]

The same relationship has been noted in the current study. Children with severe CP were also severely wasted accounting for 34(28.3%) (p=0.000).

Regarding the types of CP and severity of malnutrition, severe wasting was more prevalent in children with dyskinetic/athetoid CP and less prevalent in children with hypotonic CP, (p=0.492). This is comparable to study done by Dahl[111], who he found out that wasting was more prevalent in children with dystonic type of CP. In this study it was not statistically significant. Children with spastic CP were more stunted than children with other forms of CP accounting for 56.7% of all children with spastic cerebral palsy (p=0.028).Ifeyinwa got similar results where by 44% of children who were stunted had spastic cerebral palsy.[108]

9.3 Types of feeding problems

The most prevalent feeding problems were inability to self-feeding in 133(73.5%), inability to chewing solid foods in 80(34.3%), inability to swallow 71(29.7%), cough/chocking during feeding in 65(27.2%), cry/extensor dystonia during feeding in 52(21.8%) and inability for tongue lateralization 47(20.2%). The least common feeding problem was inappropriate wide mouth opening during feeding accounting for 3.9%.

Gangil et al did a study on feeding problems in children with cerebral palsy. He had almost similar results. The most common type of feeding problem was inability to self feed 90%.The rest of the feeding problems had higher magnitudes; that is inadequate/absent tongue lateralization 84%, chewing problems 81%, swallowing problems 63%, and coughing /choking during feeding 62%. These higher magnitudes in his study can be explained by the differences in study design. He used two methods to determine the presence or absence of feeding problems, by interview and observation. The reported feeding problems by parents were far much smaller compared to the observed feeding problems by the aothur.[89]

9.4 Time it takes to finish feeds among children with feeding problems

Regardless of the presence or absence of feeding problems, in general the majority of children with CP were taking less than 30minutes to finish their meals per sitting (55.6 - 82.3%). For those who took 30 minutes to more than 1hour to finish feed 2-4 folds were children with feeding problems. This was significant in children who are unable to feed by themselves (p=0.001), inappropriate wide mouth opening (p=0.003), unable to open mouth when fed(p=0.001), inadequate/absent tongue lateralization(p=0.028), those who could not close the lips around the spoon(p=0.002), chewing problems(p=0.005) and children who cough/chock during meals(p=0.034).

Similar findings were obtained by Reilly et al and Gangil, et al whereby mealtimes were found to be significantly brief and did not differ between the cases and controls. The mean duration of feeding session was 31.5 minutes, and the duration of feeding sessions was longer in children with poor feeding skill score. Different results were obtained by Johnson et al and Gisel et al whereby mealtimes were more prolonged, however this longer meals time were not sufficient to compensate for the severity of these childrens' feeding impairment.[48, 52,53,87,89]

9.5 Severity of malnutrition and maternal socio-demographic factors

The observed distribution of maternal socio-demographics in this study are almost similar to that of Zainah et al in Malaysia, in this study out of 239 mothers/caregivers, majority had the age group between 25 -35years, 61.6% and with regard to education status majority of the mothers/caregiver had primary school education 61.5%, whereas around two-thirds 67.8% of them were married and about half 49.4% weren't employed and formed the majority.[112]

Regarding maternal socio-demographic factor which was significantly (p=0.044) associated with having majority of children with severe wasting was level of education. All the children of illiterate mothers/caregivers had variable degrees of wasting 7(100%) and 2(28.6%) had severe wasting. The rest of the maternal socio-demographics factors were not significantly associated with having more children with severe wasting as seen in mothers/caregivers with the age above 45 years 3(25.0%) (p=0.987), cohabiting mothers/caregiver 27.9 %(p=0.365) and self employed 23.3 %(p=0.491).

However in relation to some of the above socio-demographics factors, maternal education (p=0.017) and maternal occupation (p=0.006) were found to be statistical significant associated with having more children with severe stunting, 4(57.1%) in illiterate mothers/caregivers and 71(60.2%) in not working/students mothers/caregivers. Maternal age and marital status with p= 0.468 and 0.997 respectively were not significantly related to the severity of stunting. These findings agree with those of Ifeyinwa in that lower socio-economic status was found to be associated with high prevalence of malnutrition.[108]

9.6 The association between severity of malnutrition and types of feeding problems

In this study the presence of feeding problems was directly proportional to the severity of wasting and stunting. In addition, majority of children found to have severe wasting had the following feeding problems, inability to open mouth when fed 45.5%, chewing problems 37.5% and children with inadequate/absent tongue lateralization 36.2%

However feeding problems which were significantly associated with severe wasting were inability to self feeding (p=0.000), inadequate/absent tongue lateralization (p=0.004), inability to open mouth when fed (p=0.000), chewing problems (p=0.000), swallowing problems (p=0.000) and cough/choking during feeding (p=0.002).

The same was observed in children with severe stunting. Majority of children with severe stunting had the following feeding problems, inadequate/absent tongue lateralization (72.8%), no closure of mouth around the spoon (66.7%) and inability to self feeding (60.2%). Feeding problems which were significantly associated with increase risk of severe stunting were inadequate/absent tongue lateralization (p=0.006), chewing problems (p=0.001) and swallowing problems (p=0.001).

These findings are similar to that of Dahl et al, were he found out that undernutrition was significantly associated with the presence of feeding problems, 65% of children with feeding problems were undernourished.[91]

Vik et al, in assessing feeding problems and growth disorders among children with cerebral palsy found out that lack of ability to self feeding was a significant independent risk factor for height growth retardation and underweight.[42]

10.0 CONCLUSION AND RECOMMENDATIONS

10.1 Conclusion

This study has shown that approximately 75% of the study participants had wasting, giving a prevalence of severe acute malnutrition of 20% and of moderate acute malnutrition of 53%. There was a statistically significant correlation between the severity of CP and severity of malnutrition.

The presence of feeding problems increases the risk of severe malnutrition. Maternal socio-demographics factors did not have any significantly effect on the severity of malnutrition.

10.2 Recommendations

- It is recommended that growth monitoring should be intensified among children with CP in order to identify those children who have feeding problems and are developing malnutrition very early and to institute remedial interventions and close regular follow-up.
- To institute intensive follow-up action with regard to feeding counselling and support to prevent development of severe forms of malnutrition whenever possible and to improve their quality of life.
- During follow-up of children with CP who show features of malnutrition and are not improving on the recommended intervention measures at home such as intensive supplementary feeding, admission in the ward should be considered on individual basis to facilitate naso-gastric tube feeding and close monitoring.
- A larger prospective interventional study is needed to identify effective interventional measures in our environment.

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