DYSPHAGIA IN CHILDREN (0-12 YEARS) RECOVERING FROM TUBERCULOUS MENINGITIS (TBM)

by

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DECLARATION

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ABSTRACT

Background information: Tuberculous meningitis (TBM) is the most severe extra-pulmonary complication of tuberculosis (TB) and also the most common bacterial meningitis in the Western Cape. The consequences of childhood TBM include poor motor and neurological outcomes which could lead to dysphagia.

Aims: The aim of the study was to describe dysphagia in children (0-12 years) recovering from TBM at Red Cross War Memorial Children's Hospital (RCWMCH) in the Western Cape, South Africa. The purpose of the study was to determine the occurrence and describe the nature of dysphagia as well as to investigate whether any associations existed between dysphagia and the severity of TBM, neurological sequelae, age, and radiological findings.

Methods: A retrospective descriptive survey design was used to review the medical records of all children with TBM admitted to RCWMCH between January 2006 and June 2011. Data was collected on the demographic, medical, feeding and swallowing characteristics of 133 participants. The average age was 3 years 8months with 72% of the participants being younger than five years of age.

Results: 40% (n =53) of the participants had stage I TBM, 34% (n = 45) stage II TBM, and 25% (n = 33) stage III TBM. The prevalence of dysphagia in the study population was 26% (35, N=133). Sixty-six percent of the participants presented with difficulties in multiple phases of swallowing. Dysphagia occurred more frequently in stage III TBM than in the other stages and was found to be significantly associated with the stage of TBM (χ^2=58.26; p<.001). Other factors significantly associated with dysphagia were, CP (χ^2= 42.22; *p*< .001), being less than five years of age at onset of TBM (χ^2= 6.36; *p* = .01), cerebral infarcts (χ^2=11.47; *p*< .001), as well as hydrocephalus (χ^2= 8.03; *p* = .005).

Conclusions: Children recovering from TBM, especially those with stage III TBM, who present with neurological sequelae, are at risk of dysphagia. Speech Language Therapists (SLTs) should ensure routine dysphagia assessment and intervention for this population.

Keywords: Dysphagia, TBM, Speech Language Therapy, neurological damage, swallowing

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Referencing Style:

The present dissertation has utilized the referencing as per the American Psychological Association, 6th edition (2010).

GLOSSARY – SELECTED TERMS

Aspiration:

The entry of material (e.g. saliva, liquid or food), into the airway below the level of the true vocal cords, which may occur before, during or after the swallow (Arvedson, 2008; Arvedson & Brodsky, 2002; Hall, 2001).

Cerebral Palsy:

A group of non-progressive disorders of the developing brain, which affects movement and/or posture. The motor disorders may be accompanied by impaired sensation, communication and perception (Rosenbaum et al., 2006; Rosenbaum & Rosenbloom, 2012). CP is caused by brain injury or abnormality in the brain that occurs during pregnancy or birth or within the first 2 to 3 years of a child's life (Rosenbaum & Rosenbloom, 2012).

Dysphagia:

Term used to describe disorders of swallowing, which may result from difficulties in one or more phases of swallowing (Arvedson, 2008; Arvedson & Brodsky, 2002).

Enteral feeding:

A mode of feeding that uses tube feeding to deliver food straight into the stomach, duodenum or jejunum. A variety of feeding tubes are available to assist children with feeding and swallowing difficulties and make it easier for the child to grow without the risk of malnutrition, or aspiration (Arvedson & Brodsky, 2002; Hall, 2001). Non-oral feeding may be used for a short-term (nasogastric tube feeds) or long-term period (gastrostomy/PEG).

Gastrostomy Tube- A tube that is placed (surgically or endoscopically) directly into the stomach. Nutritional and fluid needs are provided through the tube for children who cannot swallow and prevents aspiration.

Nasogastric Tube (NG Tube) - A tube is inserted through the nasal passage, the esophagus to the stomach. Nutrition and fluids are then passed through the tube to the stomach to provide adequate caloric intake. An NGT is often used for short-term tube feeding.

Feeding:

The consumption of liquid or food which involves a reciprocal process between a parent/care giver and a child. The term also includes the child's emotional and physiological state as well as the mealtime environment (Arvedson & Brodsky, 2002; Rudolph & Link, 2002).

Feeding disorders:

Difficulties in a range of eating activities that may or may not be accompanied by a difficulty with swallowing liquid and/or food. Feeding disorders include difficulties such as food refusal, disruptive mealtime behaviour, strict food preferences, developmentally delayed self-feeding skills and less than optimal growth (Arvedson, 2008; Arvedson & Brodsky, 2002).

Gastro-Oesophageal Reflux (GOR):

Backflow of the gastric contents from the stomach into the oesophagus (Arvedson & Brodsky, 2002; Hall, 2001; Rommel, De Meyer, Feenstra, & Veereman-Wauters, 2003).

Gastro- oesophageal Reflux Disease (GORD):

A clinical condition that occurs when reflux of stomach acid into the oesophagus is severe enough to impact the patient's life and/or damage the oesophagus (Rommel et al., 2003).

Swallowing:

The placement of food or liquid into the mouth, followed by the preparation and formation of the bolus and the propulsion of the bolus through the oesophagus and then into the stomach (Arvedson & Brodsky, 2002; Hall, 2000). The four phases of swallowing are: oral preparatory, oral, pharyngeal, and oesophageal (Arvedson & Brodsky, 2002).

1. ***Oral preparatory***- The voluntary placement of food into the mouth, followed by oral manipulation in order prepare and form a manageable bolus (Arvedson & Brodsky, 2002).

2. ***Oral phase*** –Transit of the bolus posteriorly into the pharynx (Arvedson & Brodsky, 2002). The oral phase is also under voluntary control. The tongue lifts the bolus toward the hard palate and the posterior movement of the tongue whilst maintaining the palatal seal, transports the bolus until the pharyngeal swallow is triggered (Arvedson, 2008; Arvedson & Brodsky, 2002).

3. ***(a) Pharyngeal phase*** initiation – the pharyngeal swallow is initiated once the bolus reaches either the anterior tonsillar pillars, base of tongue or valleculae (Arvedson, 2008).

   (b)Pharyngeal phase–From the initiation of the swallow, the following series of actions occurs: velar-pharyngeal closure, laryngeal closure, opening of the upper oesophageal sphincter (UOS) and subsequent propulsion of the bolus from the pharynx into the oesophagus (Arvedson & Brodsky, 2002; Arvedson & Lefton-Grief, 2008, Hall, 2001).

4. ***Oesophageal phase***- Begins with the relaxation of the upper oesophageal sphincter as the food leaves the pharynx and enters the oesophagus. The food then moves through the oesophagus into the stomach through a series of peristaltic waves (Arvedson, 2008; Arvedson & Lefton-Grief, 2008).

Tuberculous Meningitis (TBM):

TBM occurs when *Mycobacterium tuberculosis* (TB) invades the membranes and the fluid surrounding the brain and the spinal cord. Tuberculous meningitis (TBM) develops in 2 steps. First the *Mycobacterium tuberculosis* bacilli enter a person through droplet inhalation. This infection then increases within the lungs and spreads to the regional lymph nodes foci (Ramachandran, 2008; Thwaites et al., 2000). TBM occurs when the bacilli seed into the meninges or brain parenchyma, resulting in the formation of lesions, known as Rich foci (Ramachandran, 2008; Thwaites et al., 2000). Once the Rich foci are formed the second step of the development of TB begins. The Rich foci increase in size until it burst into the subarachnoid space (Thwaites et al., 2000). The location of the Rich focus determines the type of CNS involvement. The rupturing of the foci causes the onset of meningitis, which if left untreated, will result in severe and irreversible neurological pathology (Ramachandran, 2008; Thwaites et al., 2000).There are three clinical stages of TBM. The clinical staging proposed by the British Medical Research Council (MRC, 1948) is as follows:

Stage I - nonspecific signs and symptoms, clouding of consciousness not evident, no neurological signs and lethargic or altered behaviour;

Stage II - meningeal irritation, emerging neurological signs, a degree of mental confusion, marked alteration in consciousness and possible cranial nerve palsies;

Stage III - abnormal movements, convulsions, marked alteration in consciousness or coma and severe neurological deficits such as hemiplegia, paraplegia and decerebration.

When TBM is not treated or if there is a delay in diagnosis, it will progress through the three stages and will ultimately be fatal (Joosten et al., 2000).

Videofluoroscopic Swallow Study (VFSS):

A radiographic examination of swallowing that provides dynamic imaging of oral, pharyngeal, and upper oesophageal phases of swallowing. It serves both diagnostic and therapeutic purposes (Arvedson, 2008; Arvedson & Brodsky, 2002).

ABBREVIATIONS

ARV - Antiretroviral

CNS – Cranial Nerve System

CP – Cerebral Palsy

FDA – Food and Drug Administration

GMFCS – Gross Motor Function Classification System

GOR – Gastro-Oesophageal Reflux

GORD – Gastro-Oesophageal Reflux Disease

HBC – High Burden TB Countries

HREC – Human Research Ethics Committee

MBS – Modified Barium Swallow

MRC – Medical Research Council

NGT – Nasogastric Tube

OPT – Oral Phase Time

PEG – Percutaneous Endoscopic Gastrostomy

PTT – Pharyngeal Transit Time

RCWMCH – Red Cross War Memorial Children's Hospital

SLT – Speech-Language Therapist

TB – Tuberculosis

TBM – Tuberculous Meningitis

UCT – University of Cape Town

UOS - Upper oesophageal sphincter

URTI – Upper Respiratory Tract Infection

WHO- World Health Organisation

TABLE OF CONTENTS

1. Introduction.....1
2. Literature review.....3
3. Methodology.....17
   - 3.1 Study aim and objectives.....17
   - 3.2 Research design.....17
   - 3.3 Research method.....19
     - 3.3.1 Study population.....19
     - 3.3.2 Selection criteria.....19
     - 3.3.3 Recruitment procedure.....20
     - 3.3.4 Sampling method.....21
     - 3.3.5 Sample size 21
     - 3.3.6 Description of participants.....22
   - 3.4 Materials and instrumentation.....26
     - 3.4.1 Feeding and Swallowing Checklist.....26
   - 3.5 Validity and reliability.....30
   - 3.6 Data collection procedure.....32
     - 3.6.1 Pilot study.....33
   - 3.7 Data analysis.....34
   - 3.8 Ethical considerations.....35
4. Results.....37
5. Discussion.....43
6. Conclusion.....54
7. References.....56
8. Appendices.....67

LIST OF TABLES AND FIGURES

Tables

Table 1: Summary of studies describing dysphagia in children recovering from TBM.....8

Table 2: Feeding and swallowing assessment protocol.....26

Table 3: Neurological sequelae and their association with dysphagia.....38

Table 4: Radiological findings and their association with dysphagia.....39

Table 5: Oral preparatory and oral phase signs and disorders in participants with dysphagia.....41

Table 6: Pharyngeal signs and disorders in participants with dysphagia.....42

Figures

Figure 1: Age distribution of the participants.....22

Figure 2: Stage of TBM at initial presentation.....23

Figure 3: Age distribution in the different stages of TBM.....23

Figure 4: Presence of neurological sequelae in the participants.....24

Figure 5: Regions / Areas from which the participants originated.....25

Figure 6: Distribution of participants from the Cape Flats district.....25

Figure 7: Data collection procedure flowchart.....33

Figure 8: The prevalence of dysphagia in the sample population of children recovering from TBM.....37

Figure 9: Frequency of dysphagia in the different stages of TBM.....38

Figure 10: Occurrence of dysphagia in the different phases of swallowing.....40

Appendices

Appendix A: Ethics Approval, University of Cape Town (UCT), Faculty of Health Sciences Human Research Ethics Committee (HREC Reference number: 517/2010)

Appendix B: Consent letter to medical superintendent RCWMCH

Appendix C: Feeding and Swallowing Checklist

1. INTRODUCTION

Approximately two billion people are infected with *Mycobacterium tuberculosis* (TB) worldwide (Farinha, Razali, Holzel, Morgan & Novelli, 2000; Ramachandran, 2008; Van Well et al., 2009; World Health Organization, [WHO] 2013). According to the Global Tuberculosis Report, in 2012, approximately 8.6 million people developed TB and 1.3 million died from the disease (WHO, 2013). The largest burden of TB occurs in low income countries and the highest estimated number of cases occurred in South-East Asia (29%), Africa (27%) and Western Pacific regions(19%), in addition India and China alone accounted for 26% and 12% of total cases, respectively(WHO, 2013). In Africa the highest number of cases occur in sub-Saharan Africa with approximately 1.5 million new TB cases recorded annually (Van Well et al., 2009; Walls & Shingadia, 2004; WHO, 2013).

Sub-Saharan Africa also has one of the highest incidences of tuberculous meningitis (TBM) as the incidence of Central Nervous System (CNS) tuberculosis is often directly proportional to the prevalence of TB infection that occurs in the area (Garg, 1999; Ramachandran, 2008). In addition, in areas where the incidence of TB is high, TBM commonly occurs in young children, especially those less than five years of age (Donald & Schoeman, 2004; Ramachandran, 2008). The consequences of childhood TBM have been documented in a number of studies and include poor motor and neurological outcomes (Berman, Kibel, Fourie & Strebel, 1992; Donald, Fourie, & Grange, 1999; Farinha et al., 2000; Kalita, Misra, & Ranjan, 2007; Springer et al, 2008). Cerebral palsy (CP) may manifest as a result of neurological damage caused by TBM and in the Western Cape Province of South Africa, CP has been reported in approximately 53% of the children recovering from TBM (Berman et al., 1992; Donald et al., 1999; Farinha et al., 2000).

Neurological impairment has been linked to dysphagia and it is estimated that between 30 – 90% of children with neurological involvement may have dysphagia (Arvedson, 2008; Calis et al., 2008; Cichero & Murdoch, 2006; Hall, 2001; Rudolph & Link, 2002). In children with CP, dysphagia is reported to be as high as 85-95% (Calis, et al., 2008; Rudolph & Link, 2002; Workinger, 2005). Dysphagia can affect the child's quality of life as it may negatively impact on their respiratory health, growth and quality of life (Rogers, 2004; Schwarz, 2003; Sullivan et al., 2005). However, there is no published literature regarding the prevalence of dysphagia in children recovering from TBM despite an association between neurological impairment and dysphagia. Lack of information regarding feeding and swallowing skills and dysphagia outcomes in the paediatric population recovering from

TBM, makes it difficult for health professionals to manage these patients optimally. Therefore the present study aims to address this knowledge gap and contribute to the development of dysphagia assessment and management protocols, and ultimately advance service delivery for this population.

The present study describes the frequency of dysphagia in children recovering from TBM (January 2006- June 2011) and determines the relationship between the severity of TBM and the frequency and nature of dysphagia. The researcher made use of a retrospective descriptive survey of the medical records of all children (0-12 years) admitted to Red Cross War Memorial Children's Hospital (RCWMCH) with TBM over a period of 5 and a half years (January 2006- June 2011). The study provides information that may assist health professionals in early identification and management of dysphagia in the paediatric TBM population. Early identification may lead to more timely referrals to Speech Language Therapists (SLT's) for dysphagia management which may reduce morbidities associated with feeding and swallowing difficulties (Lefton-Greif & Arvedson, 2007; Schwarz, 2003). The specific nature of dysphagia in children recovering from TBM highlighted in this study can be used by SLT's to create target specific protocols for assessment and management.

2. LITERATURE REVIEW

South Africa is considered a high burden TB country and in 2012 it had one of highest incidence of TB worldwide with an estimated 0.69 million cases reported (World Health Organization [WHO], 2013). In the last decade, the TB incidences in most countries declined, with a reported rate of decline of about 2% in the high burden TB countries (WHO, 2013). Despite this however, the Global Tuberculosis Report (2013) indicate that in 2012, South Africa reported an increase of 1000 or more cases per 100 000. The high incidence of TB and drug-resistant TB combined with the HIV/AIDS epidemic has resulted in a disproportionately high burden of disease on the South African health system compared to other countries globally (Chopra, 2009; WHO, 2009).

The Western Cape Province in South Africa has one of the highest recorded TB incidences worldwide (Marais, Graham, Cotton & Beyers, 2007; Przbojwski, Andronikou & Wilmshurst, 2006; Van Well et al., 2009; Western Cape Metropole & City of Cape Town, 2004). In 2006 the incidence of TB in the Western Cape was 1,033/100 000 (TB Facts.org, 2015). In the Western Cape children less than 13 years of age make up 13.7% of the TB population. The elevated percentage of TB cases in children can be attributed to the high frequency of TB within the adult population (Marais et al., 2007; Przbojwski et al., 2006; Van Well et al., 2009; Western Cape Metropole & City of Cape Town, 2004). In addition children have an increased risk of infection compared to that of adults since the estimated risk of infection decreases with age i.e. 24% in children between one and five five years of age compared to 43% in infants younger than 1 year old (Van Rie et al., 1999; Walls & Shingadia, 2004).

According to the most recent Cape Town TB Control Progress Report (2004) the highest TB notification in the Western Cape were reported in over-crowded socio-economically deprived areas with annual TB notification rates that exceed 1000/100 000 (Western Cape Metropole & City of Cape Town, 2004). The high burden sub-districts, were Khayelitsha (19%), Nyanga (15%) and Oostenberg (13%), all areas in the Cape Flats region; an expansive, low-lying, flat area situated to the southeast of the central Cape Town (Western Cape Metropole & City of Cape Town, 2004). The most recent TB Control Progress Report by the City of Cape Town which could be sourced, was over ten years ago (2004) and the incidence rates of TB in this region may in fact be higher due to the recent increase in TB cases reported in South Africa.

Multiple factors have been identified as key contributors to the high incidence of TB in these areas (Murray et al., 2012).Poverty, unemployment and overcrowding characterise life in the Cape Flats. While the national unemployment rate is estimated at 25%, reports suggest that this figure rises in informal settlements to approximately 57% (Mahajan, 2014). Living conditions in these areas are often substandard, with approximately 40-45% of houses in the Cape Flats considered informal shacks, made of corrugated iron, particleboard, and scrap metal and most do not have access to water (Water Service Development Plan, 2009). The low socio-economic status and overcrowding in these areas results in living conditions that are poorly ventilated, allowing TB to spread more easily from one person to another (Bradley & Corwyn, 2002; Donald et al., 1999; Murray et al., 2012; Ramachandran, 2008; Smith 2001).

HIV/AIDS, malnutrition and drug-abuse have also been linked with high TB incidence (Bradley & Corwyn, 2002; Garg, 1999; Ramachandran, 2008; Rekha & Swaminathan, 2007; Smith 2001). HIV/AIDS reinforces poverty through loss of household income, reduced educational prospects and a growing number of orphans. In 2012, it was estimated that 6.4 million South Africans were infected with HIV, with the Western Cape making up six percent of the HIV population (South African National HIV Prevalence, Incidence and Behaviour Survey, 2012). Although the Western Cape has the lowest reported incidence of HIV/AIDS nationwide, HIV positive people in this region are at higher risk of TB due to the high prevalence of TB in the region (Marais, Graham, Cotton & Beyers, 2007; SA Health Info, 2011; Smith, 2005). It is estimated that TB is responsible for approximately a third of all deaths in HIV infected people in South Africa (Setwe, 2009).

TB manifests as *pulmonary*, which occurs in the lungs or *extra-pulmonary* which presents in other parts of the body including the central nervous system (Farinha et al., 2000; Garg, 1999; Smith, 2001). The highest mortality and morbidity is associated with TB of the central nervous system, with tuberculous meningitis (TBM) being the most severe extra-pulmonary complication of TB (Farinha et al., 2000; Garg, 1999; Singhi & Singhi, 2001; Van Well et al., 2009).

Over the years there has been an increase in the occurrence of drug resistant TB and this has subsequently resulted in a rise in drug-resistant tuberculous meningitis (Seddon et al., 2012). Drug-resistant tuberculous meningitis is serious and is associated with poor outcomes (Seddon et al., 2012; Thwaites et al., 2013). In fact multidrug-resistant (MDR) TBM is often associated with death in children (van Toorn et al., 2014). At the time of data collection for the present study there was limited information regarding drug-resistant TBM in children (Seddon et al., 2012).

The incidence of CNS tuberculosis is often influenced by the prevalence of TB infection that occurs in the area (Cho et al., 2013; Garg, 1999; Ramachandran, 2008). In areas that have a low prevalence of TB, TBM predominately affects adults; however in areas where the prevalence of TB is high, TBM commonly occurs in young children, especially those less than five years of age (Cho et al., 2013; Donald & Schoeman, 2004; Ramachandran, 2008; Thwaites et al., 2009; Walls & Shingadia, 2004). Local studies have reported incidences of between 78-82% in children less than five years, highlighting younger children's vulnerability to TBM (Schoeman, 1990; Van Well, et al., 2009). Reasons suggested for the vulnerability of younger children to TBM include; poor resistance to infection with TBM, early dissemination and the non-specific nature of the symptoms which results in delayed diagnosis (Mahadevan et al., 2002; Thwaites et al., 2009; Van Well et al., 2009). Therefore, the high occurrence of TB in the Western Cape has resulted in TBM being the most common type of bacterial meningitis in children below 13 years of age in the region (Andronikou, Smith, Hatherhill, Douis & Wilmshurst, 2004; Springer et al., 2008).

The clinical outcome of TBM is strongly associated with the stage of the disease at the time of presentation (Chiang et al., 2014; Donald et al, 1999; Farinha et al, 2000; Garg, 1999; Kalita et al., 2007; Newton, 1994; Singhi & Singhi, 2001; Springer et al, 2008). The Medical Research Council (MRC, 1948) proposed three clinical stages that are used to indicate the severity of TBM (see glossary for detailed definitions). Children in stage I present with non-specific signs and symptoms; those in stage II show emerging neurological signs while children in stage III present with severe neurological signs such as marked alteration in consciousness, seizures and motor impairment (Farinha et al., 2000; Garg, 1999; Newton, 1994; Singhi & Singhi, 2001). When TBM is not treated or diagnosis is delayed, the disease could advance through the three clinical stages and may ultimately be fatal (Joosten et al., 2000).

Early detection and treatment is one of the most important factors determining outcomes in children with TBM (Chiang et al., 2014; Kalita et al, 2007; Thwaites et al., 2013; van Toorn et al., 2014). According to Kalita et al. (2007), patients treated in the early stage of the disease are five times more likely to recover completely than those with more advanced TBM. Poorer outcomes such as severe neurological impairments as well as high morbidity and mortality are associated with more advanced TBM (Andronikou et al., 2006; Farinha et al, 2000; Kalita et al, 2007; Schoeman, 1990; van Toorn et al., 2014; Van Well et al., 2009). It is estimated that about 80% of children with advanced disease at diagnosis (TBM stage II and III) will develop neurologic sequelae.

Early diagnosis however, remains a challenge due to the non-specific clinical presentation of TBM (Cho et al., 2013; Nabukeera- Barungi et al., 2014; Thwaites, van Toorn & Schoeman, 2013; van Toorn et al., 2014). Van Well et al. (2009) reported that 57% of children infected with TBM in the Western Cape were ill for more than 7 days before being diagnosed, resulting in the majority being diagnosed at stages II or III of TBM. These findings emphasise the need to develop more accurate as well as affordable diagnostic tests for TBM which will aid early diagnosis and prevent neurologic complications or devastating outcomes in high burden areas (Song et al., 2013; van Toorn et al., 2014). In addition better monitoring and TB control programmes at the community levels can also result in early detection of TBM.

Various negative consequences associated with TBM in childhood have been documented and they include; neurological deficits, altered mental status, stroke, hydrocephalus, cranial neuropathies and failure to thrive (Andronikou et al., 2006; Berman et al., 1992; Chiang et al., 2014; Donald et al., 1999; Farinha et al., 2000; Kalita et al., 2007; Nabukeera- Barungi et al., 2014; Springer et al., 2008; Van Well, et al., 2009). The outcomes in children recovering from TBM vary; complete recovery has been documented in 11-61%, neurological impairment in 13-75% and death in approximately 7-57% (Chiang et al., 2014; Donald et al., 1999; Farinha et al., 2000; Kalita et al., 2007; Nabukeera- Barungi et al., 2014; Van Well et al., 2009). In the Western Cape, Van Well et al. (2009) documented neurological sequelae in up to 71% of children recovering from TBM which they attributed to the high number of children diagnosed with stage II and III TBM in this area.

The types of neurological sequelae resulting from TBM include motor disorders (e.g. hemiparesis, hemiplegia, quadriplegia, athetosis and extra pyramidal syndrome), cognitive impairment, visual impairment, behavioural problems, cranial nerve palsies, hearing loss and speech impairment (Farinha et al., 2000; Kalita et al., 2007; Ramachandran, 2008; Springer et al., 2008). It is estimated that between 47-78.5% of children recovering from TBM that present with neurological damage, develop permanent sequelae. In addition, late diagnosis cerebral palsy (CP) may manifest as a result of neurological damage caused by TBM (Berman et al., 1992) and amongst survivors of TBM in the Western Cape, CP occurs in approximately 53% of paediatric patients (Berman et al., 1992).

The nature of the outcomes of TBM are influenced by different prognostic factors such as age, stage of TBM, hydrocephalus, focal neurological deficits, HIV co-infection, motor deficits and

raised intracranial pressure(Andronikou et al., 2006; Donald et al., 1999; Farinha et al., 2000; Garg, 1999; Hosoglu et al., 2001; Kalita et al., 2007; Mahadevan et al., 2002; Newton, 1994; Singhi & Singhi, 2001; Springer et al., 2008; Van Well et al., 2009). These prognostic factors assist health professionals in their management and recommendations, and also provide parents and caregivers of children affected with TBM realistic expectations regarding outcomes (Andronikou et al., 2006).

Young age and severity of the disease (stage of TBM) are two of the most important prognostic factors as mortality and morbidity are higher in younger children (less than five years) with more advanced stage of TBM (Humphries et al., 1990, Kalita et al., 2007; Mahadevan, et al., 2002; Schoeman, 1990; Van Well et al., 2009). Mahadevan et al. (2002) highlighted that younger age and advanced stage of disease at admission were associated with adverse outcome.

In countries with a high burden of TB, TBM is most commonly seen in children with a peak incidence reported to be in children aged two to four years (Thwaites et al., 2013). Young children especially those less than five years of age are more vulnerable to TBM and are likely to have poorer outcomes of TBM and more severe neurological sequelae (Kalita et al., 2007; Mahadevan et al., 2002; Schoeman, 1990; Thwaites et al., 2009; Van Well et al., 2009). The vulnerability of younger children is supported by local studies on TBM were 78% - 82% of the participants were less than five years of age (Berman et al., 1992; Schoeman, 1990; van Well et al., 2009).

Cerebral infarctions and the resulting neurological deficits, are another prognostic indicator for motor and developmental outcomes in children recovering from TBM (Andronikou et al., 2006; Bernaerts et al., 2003; Farinha et al., 2000; Mahadevan et al., 2002; Springer et al., 2008; Thwaites et al., 2000). Poor prognosis is often linked to infarctions that occur before treatment has begun (Andronikou et al., 2006). Ischaemic cerebral infarctions can also affect the basal ganglia causing basal enhancements and basal ganglia infarctions which are associated with poorer prognosis (Andronikou et al., 2006; Farinha, et al., 2000). Higher incidence of cerebral infarcts, basal ganglia enhancement and hydrocephalus amongst younger children and those with stages II and III of TBM thus explains the poor outcomes (Bernaerts et al., 2003; Springer et al., 2008; Thwaites et al., 2000).

Feeding and swallowing difficulties, including dysphagia, are commonly reported in children with neurological impairment (Arvedson & Brodsky, 2002; Calis et al., 2008; Fung et al., 2002; Gangil, Aneja, Ahuja, & Anand 2001; Hinchcliffe, 2007; Rogers, 2004; Rudolph & Link, 2002; Sullivan, 2009), therefore children recovering from TBM may be at increased risk of developing these difficulties.

However, no literature regarding dysphagia or feeding difficulties could be sourced during this study. A literature search (keywords - feeding, swallowing, dysphagia, TBM; databases – University of Cape Town Health Sciences databases, including CINAHL,8 Cochrane Library, Medline, Pubmed) identified no published studies describing dysphagia associated with TBM. The only information that could be sourced regarding the presence of dysphagia in children recovering from TBM was from two undergraduate studies, Bengsch & Lourenco (2005) and Allies etal. (2009). Table 1 provides an overview of the two different studies with regard to methodologies, sample sizes and the nature of dysphagia reported by the two studies.

Table 1

Summary of studies describing dysphagia in children recovering from TBM

Study	Description	Methodology	N	Signs of Feeding and/or Swallowing Difficulties
Bengsch & Lourenco, (2005)	A descriptive study to determine the oral motor and feeding abilities of children recovering from tuberculous meningitis.	Prospective descriptive study	17	Oral phase difficulties (47%, n=8) ▪ Reduced lip closure ▪ Anterior spillage ▪ Reduced tongue mobility Pharyngeal phase ▪ Not investigated
Allies et.al, (2009)	Dysphagia in children (0-12 years) recovering from Tuberculous Meningitis (TBM)	Prospective descriptive study	12	Oral phase difficulties (33%, n=4) ▪ Reduced lip closure ▪ Anterior spillage ▪ Oral residue ▪ Reduced tongue mobility Pharyngeal phase (25%, n=3) ▪ Absent trigger of the swallow ▪ Delayed trigger of the swallow ▪ Choking after the swallow

As there is no published information regarding dysphagia in the paediatric population recovering from TBM, information regarding dysphagia in the general paediatric population, and more specifically those with neurological impairment and complex medical issues will be discussed to provide background for dysphagia.

Accurate information regarding the prevalence of dysphagia is not available but dysphagia reported in children ranges between 20-45% of minor feeding problems in typically developing children and up to 70% of children with chronic medical conditions (Arvedson, 2008; Manikam & Perman, 2000; Rudolph & Link, 2002; Schwarz, 2003). The exact prevalence of paediatric dysphagia

is unknown because of the different methodologies, different populations as well as different terminology and definitions of dysphagia/feeding and swallowing difficulties used by researchers (Arvedson & Lefton-Greif, 2007; Fung et al., 2002).

In addition, while there are a variety of studies evaluating dysphagia in children with neurological impairments, very few studies were conducted in developing countries, thus the prevalence rates may not accurately represent the entire population. The sample representativeness may also be skewed because the majority of the studies investigate dysphagia in children with CP, while there is limited information on other neurological conditions.

The lack of agreement in the definition of feeding and swallowing difficulties has been raised by several authors (Arvedson & Lefton-Greif, 2007; Benfer et al., 2013; Fung et al., 2002; Oosthuizen, 2012) and although a universally accepted description of feeding and swallowing difficulties has been suggested no specific definition has been agreed upon. Arvedson (2008) describes feeding disorders as “problems in a broad range of eating activities that may or may not be accompanied by a difficulty with swallowing food and liquid” and dysphagia as “problems in one or more phases of the swallow”. The present study focused on dysphagia although children recovering from TBM may also present with feeding difficulties. Feeding difficulties typically seen in children include limited intake of food, food selectivity by type, disruptive mealtime behaviours, and excessive meal duration (Arvedson & Brodsky, 2002; Manikam & Perman, 2000).

The common causes of paediatric dysphagia include neurological disorders, developmental disability, complex medical conditions, sensory disorders, medication side effects (e.g. lethargy, decreased appetite), behavioural factors as well as social, emotional and environmental factors (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; Hall, 2001; Manikam & Perman, 2000; Schwarz, 2003). When children fall ill, any medical condition could result in poor feeding. Conditions such as seizures, otitis media, acute or chronic infections of the head and neck, frequent upper respiratory infection, can result in mild to severe feeding difficulties. Illness reduces motivation to feed and children who are sick may have reduced energy, experience discomfort and pain, deal with distortions of taste and smell, and suffer from fatigue and stress (Arvedson & Brodsky, 2002; Hall, 2001; Manikam & Perman, 2000). For example if a child has a sore throat or gum disease, this may interfere with the child's ability to eat because of pain when chewing and swallowing (Manikam & Perman, 2000).

One of the most common causes of paediatric dysphagia is neurological disorders. Links between neurological impairments and dysphagia have been established and a review of literature indicates dysphagia in 30-90% of individuals with neurological impairment (Arvedson & Brodsky, 2002; Calis et al., 2008; Fung et al., 2002; Gangil et al., 2001; Hinchcliffe, 2007; Rogers, 2004; Rudolph & Link, 2002; Schwarz, 2003; Sullivan, 2009). One example that demonstrates the high risk of dysphagia in children with neurological impairments is the CP population, with an estimated prevalence between 85-95% (Arvedson & Brodsky, 2002; Calis, et al., 2008; Cichero & Murdoch, 2006; Fung et al., 2002; Hall 2001; Sullivan, 2009).

Children with neurological impairments are at high risk of developing dysphagia because any abnormalities with the cranial nerves, the brain stem, cerebral cortex and any muscles involved in swallowing may affect the complex sequential coordination required for feeding and swallowing (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif 1998; Hall, 2001; Schwarz, 2003). Neurologic abnormalities may affect the movement, posture, tone and sensation of the body which results in oral sensorimotor dysfunction, pharyngeal dysmotility, aspiration, oesophageal motility disorders and gastro-oesophageal reflux (Arvedson & Brodsky, 2002; Benfer et al., 2012; Calis et al., 2008; Erasmus et al., 2012; Gangil et al., 2001; Kim, Han, Song, & Chung, 2013; Van den Engel-Hoek et al., 2013). In addition many children with severe neurological have unpleasant experiences associated with feeding, which may result in negative learned behaviours which further exacerbate the feeding difficulties e.g. food refusal and aversive responses to stimulation in and around the mouth.

Children with more severe neurological impairments are more likely to present with dysphagia, than those with less neurological involvement (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif 1998; Hall, 2001). The severity of neurological impairment is strongly associated with the presence of feeding dysfunction (Arvedson & Brodsky, 2002; Benfer et al., 2013; Calis, et al., 2008; Cichero & Murdoch, 2006; Erkin, Culha, Ozel & Kirbiyik, 2010; Fung et al., 2002; Sullivan et al., 2005). According to Arvedson (2008), dysphagia occurs less in children with hemiplegia and diplegia (25–30%) compared to children who have spastic quadriplegia CP (50–75%). In a recent study, Benfer et al. (2013) reported dysphagia in 85% of children with CP and a significant increase in the likelihood of having oropharyngeal dysphagia for children with more severe gross motor impairment (GMFCS V) compared to those with mild gross motor impairment (GMFCS I).

In addition to having a higher risk of dysphagia, children with severe motor impairments often have more severe dysphagia which is associated with growth disorders and affects respiratory

health (Arvedson & Brodsky, 2002; Benfer et al., 2013; Calis et al., 2008; Sullivan et al., 2005, Weir et al., 2011). Children recovering from TBM who have severe permanent neurological sequelae therefore have an increased risk of developing more severe dysphagia than children with normal outcomes.

The most common consequences of dysphagia affect respiratory health, growth and quality of life. Dysphagia may also result in pain and discomfort, constipation and dental problems (Fung et al., 2002). Aspiration due to dysphagia may result in respiratory compromise, respiratory illness and lung disease (Andrew & Sullivan, 2012; Cass et al., 2005; Rogers, 2004; Weir et al., 2011). Dysphagia may also result in limited food intake and poor nutritional intake which may cause failure to thrive, associated short stature and low fat stores (Andrew & Sullivan, 2012; Rogers, 2004; Sullivan, 2000). Growth failure subsequently may lead to poor health and makes the child susceptible to chronic illness and in extreme cases even death (Andrew & Sullivan, 2012; Fung et al., 2002; Hinchcliffe, 2007; Manikam & Perman, 2000; Rogers, 2004; Sullivan, 2009; Veness & Reilly, 2008).

Within the social context, dysphagia also negatively affects the child's development, activity, participation, impacts parent-child interaction, family-life and overall quality of life (Rogers, 2004). Although the primary function of feeding is nutritional, mealtimes serve an important social function and children with severe dysphagia are often unable to participate in family interaction during these times (Arvedson, 2008; Prasse & Kikano, 2009). For children with dysphagia and their caregivers, eating is often stressful and time consuming (Fung et al., 2002; Rogers, 2004). Several authors have highlighted that stressful mealtimes can result in significant health and social implications (Arvedson 2008; Prasse & Kikano, 2009; Rogers, 2004; Veness & Reilly, 2008). Early identification and management of dysphagia and nutrition is therefore important in preventing the potentially negative consequences (Andrew & Sullivan, 2012; Arvedson & Brodsky, 2002; Fung et al., 2002; Rogers, 2004; Weir et al., 2011).

Infants and children with dysphagia present with signs and symptoms of difficulties in the different phases of swallowing (Arvedson, 2008; Arvedson & Brodsky, 2002; Field et al., 2003; Hall 2000; Oosthuizen, 2012; Rommel et al., 2003). The signs and symptoms of dysphagia will be discussed, with particular reference to the paediatric population with neurological impairment because of the increased risk of neurological sequelae following TBM.

The nature of oral phase (both the oral preparatory and oral phases) difficulties in children included both sensory and motor based problems. The majority of the oral motor difficulties in children with neurological difficulties are caused by weakness and/or poor coordination of the muscles. Oral motor difficulties may present as poor lip closure, resulting in anterior loss of food from the mouth, a weak suck in infants, reduced tongue strength and abnormal movement patterns such as tongue thrusting and a tonic bite (Arvedson & Brodsky, 2002; Fung et al., 2002; Hall, 2001; Hinchcliffe, 2007; Schwarz, 2003). These poor oral motor skills result in difficulties that make feeding messy and slow, leading to an increase in meal times, reduced intake and fatigue, as well as increased caregiver stress (Andrew & Sullivan, 2012; Arvedson & Brodsky, 2002; Hall, 2001, Prasse & Kikano, 2009).

Oral sensory difficulties also occur frequently in children with neurological impairments (Fung et al., 2002; Manikam & Perman, 2000). Oral sensory disorders vary in degree and include hypoactive and hyperactive responses (Arvedson & Brodsky, 2002; Manikam & Perman, 2000), which could lead to food selectivity and/or oral aversion which may affect nutritional intake and therefore growth (Andrew & Sullivan, 2012; Arvedson& Brodsky, 2002; Hall, 2001).

The exact prevalence of oral-motor involvement among children with neurological impairment is unknown however; literature indicates that it is a relatively common occurrence (Arvedson & Brodsky, 2002; Fung et al., 2002; Hall, 2001; Hinchcliffe, 2007; Reilly et al., 1996). The only information available regarding oral motor difficulties in children recovering from TBM was sourced from unpublished undergraduate studies by Bengsch and Lourenco (2005) and Allies et al. (2009). Bengsch and Lourenco (2005) reported oral motor difficulties in 47% (N=17) of participants recovering from TBM, while Allies et al. (2009) documented oral phase difficulties in all dysphagic participants (n=4) recovering from TBM. Both these studies had very small sample sizes and recommended the need for further research in this area which could provide a more representative study sample. Hence the present study aims to provide more information regarding the nature of oral phase difficulties in children recovering from TBM and assist in understanding the potential consequences of dysphagia in this population.

While the majority of research regarding the nature of feeding and swallowing skills in children with neurological impairments investigates oral motor skills and occurrence of aspiration, only a few studies evaluated the prevalence of pharyngeal phase difficulties. The pharyngeal phase difficulties typically occurring in children with neurological disorders may result from weakness in or

a lack of coordination within the pharynx and include uncoordinated swallow, delay in the triggering of the pharyngeal swallow or an uncoordinated suck-swallow-breathe sequence (in infants), with aspiration occurring before, during or after the swallow (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; Calis et al., 2008; Fung et al., 2002; Hall, 2001; Hinchcliffe, 2007; Kim et al., 2013; Manikam & Perman, 2000; Oosthuizen, 2012; Rogers, 2004).

Allies et al. (2009) documented pharyngeal difficulties in 75% (n=4) of the participants that presented with dysphagia. The nature of the pharyngeal difficulties included absent trigger of the swallow, delayed trigger of the swallow and coughing/gagging/choking after the swallow (Allies et al., 2009).

One of the most frequently reported pharyngeal phase difficulties is aspiration. Aspiration may occur as a result of oral secretions, food, or liquid or as a consequence of gastroesophageal reflux, entering the airway (Kim et al., 2013; Miller, 2011, Rogers, 2004; Sullivan et al., 2005; Weir et al., 2011). Any aspiration of material into the airway, may lead to mechanical obstruction and bacterial pneumonia resulting in reduced weight gain and growth faltering (Miller, 2011, Weir et al., 2011; Tutor & Gosa, 2012). Neurological impairments can cause weakness and poor coordination in the oral phase, resulting in premature spillage before the swallow and aspiration (Arvedson& Brodsky, 2002; Calis et al., 2008; Gangil et al., 2001; Schwarz, 2003; Tuto, & Gosa, 2012). Problems in the pharyngeal phase in terms of timing of the swallow or inadequate airway closure may also result in aspiration (Miller, 2011; Tutor, & Gosa, 2012). The incidence of aspiration is estimated to be between 25-70% in children with neurological impairment (Kim et al., 2013; Rogers, 2004; Sullivan et al., 2005, Weir et al., 2011). Aspiration is significantly associated with neurological impairment (Kim et al., 2013; Weir et al., 2011). Weir et al. (2011) investigated oropharyngeal aspiration and silent aspiration in 300 children and reported a significant association between both oropharyngeal aspiration and silent aspiration and the presence of neurological impairments.

Instrumental assessments such as videofluoroscopic swallow study (VFSS) are often used to determine aspiration and are considered more reliable than bedside or clinical swallowing assessments (Arvedson, 2008; Kim et al., 2013; Prasse & Kikano, 2009) because aspiration can be silent and not present with obvious clinical signs. Literature indicates that as much as 97% of children with CP or neurological impairment who aspirate, do so silently due to a weakened cough reflex (Benfer et al., 2013; Cass et al., 2005; Kim et al., 2013; Rogers, 2004; Sullivan et al., 2005; Weir et al., 2011;). Often silent aspiration remains undetected resulting in respiratory illnesses such as

pneumonia (Arvedson & Brodsky, 2002; Cass et al., 2005; Erkin et al., 2010; Hall 2001; Tutor & Gosa, 2012; Weir et al., 2011).

In addition to the high occurrence of aspiration in children with neurological impairments, aspiration is also related to the severity of motor impairment (Benfer et al., 2013; Miller, 2011; Sullivan et al., 2005; Weir et al., 2011). A study by Sullivan et al. (2005) reported a significant correlation between the number of chest infections that occurred in the participants and the severity of motor impairment (p=.0007). These findings indicate that children recovering from TBM who present with neurological impairments may be at risk of aspiration thus regardless of the aetiology, aspiration can have serious consequences on a child' respiratory health.

The present study focused on the oral-pharyngeal phases of swallowing from a Speech-Language Therapist (SLT) perspective however children with neurological disorders may also present with oesophageal phase difficulties, which include motility disorders and structural abnormalities (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif1998; Hall, 2001). Disorders of the oesophagus may also impact on feeding and may cause recurrent aspiration (Miller, 2011; Rudolph & Link, 2002).

Gastro-oesophageal reflux (GOR) is reported frequently in children with neurological disorders (Erkin et al., 2010; Kahrilas, 2008; Oosthuizen, 2012; Schwarz, 2003; Talley & Wiklund, 2005). Schwarz et al. (2001) conducted a study with 79 children with moderate-severe neuro-developmental disabilities and documented GOR in 56% of the participants (Schwarz, 2003). GOR in children with neurological impairments has been attributed to factors such as poor posture, increased intra-abdominal pressure, spasticity and prolongation of gastric emptying (Erkin et al., 2010; Rommel, De Meyer, Feenstra, & Veereman-Wauters, 2003; Sullivan et al., 2005). GOR may result in feeding and swallowing difficulties such as: pain and nausea which lead to food refusal and lack of appetite, as well as respiratory symptoms, poor weight gain and dental caries (Erkin et al., 2010; Rommel et al., 2003; Sullivan et al., 2006).

In addition GOR is associated with aspiration especially in children with neurological impairments (Duca et al., 2008; Giambra et al., 2010; Oosthuizen, 2012).Children with neurological disorders who present with oesophageal motility disorders or impaired opening of the lower oesophageal sphincter are at risk of aspirating the reflux which may result in GOR (Arvedson & Brodsky, 2002; Arvedson& Lefton-Greif, 1998; Hall, 2001). Despite the reported association between GOR and aspiration, no causal relationships have been established yet (Rommel et al., 2003;

Oosthuizen, 2012). Nevertheless it is clear that GOR can negatively impact a child's overall health, nutritional intake as well as their respiratory health (Hinchcliffe, 2007; Talley & Wiklund, 2005; Oosthuizen, 2012; Sullivan et al., 2005).

Although the literature review has focused on dysphagia related mostly to neurological deficits, children recovering from TBM may present with additional factors that contribute to dysphagia. As mentioned previously, general illness, hospitalisation as well as the effects of medication on appetite can also contribute to dysphagia in children recovering from TBM. Specifically, TB medications are known to affect appetite and children recovering from TBM are usually hospitalized for extended periods (up to 6 months) to receive treatment. Therefore children recovering from TBM who experience reduced appetite for significant periods of time could be at risk for failure to thrive and growth disorders.

Children with feeding and swallowing difficulties may need enteral feeding due to unsafe swallowing or inadeuqate intake, to ensure that their health and nutritional status are not compromised (Arvedson & Brodsky, 2002; Andrew & Sullivan, 2012; Prasse & Kikano, 2009; Rogers, 2004; Southall and Martin, 2011; Sullivan et al., 2005). The most common alternate feeding methods utilised in infants and children with feeding and swallowing difficulties, include nasogastric tubes (NGT) for short term non-oral feeding or gastrostomy tubes for long term non-oral feeding (Arvedson & Brodsky, 2002; Rogers, 2004; Sullivan et al., 2005). The benefits of using enteral feeding in infants and children with severe dysphagia have been documented in a number of studies and include reduction in vomiting, weight gain, improved general health and improved quality of life for care-givers (Morris, 1998; Rogers, 2004; Samson- Fang, Butler, O'Donell, 2003; Sullivan et al., 2005).

However, there is literature suggesting negative impacts of tube feeding for some individuals with neurological impairments (Morris, 1998, Rogers, 2004; Sullivan et al., 2005). Reported negative consequences of enteral feeding include lack of positive oral stimulation leading to hypersensitivity in the oral area and oral sensory defensiveness (Morris, 1998). Children who are placed in long term enteral feeding should be managed appropriately to minimise the negative side effects of tube feeding.

The outlined difficulties that are likely to present in children recovering from TBM emphasise the need for early identification and timely referrals to SLTs for dysphagia assessment and management in order to reduce the associated morbidities (Lefton-Greif & Arvedson, 2007; Schwarz,

2003). In the Western Cape, dysphagia management is one area in which service delivery is currently limited. At the time of data collection for the present study, SLT services in the Western Cape are mostly available at tertiary hospitals (e.g. Groote Schuur Hospital, Tygerberg Hospital and RCWMCH) only. Since then two district hospitals have open in the Cape Flats (Khayelitsha District Hospital and Mitchells Plain District Hospital). The Speech and Hearing Project (a community-based project) is a pilot project that was implemented in the Mitchell's Plain district at the end of 2014 and has employed 4 SLTs at community level. Although there are now SLT services available in these areas, there is still a very high patient to therapist ratio. Very few therapists have to provide services to a large number of patients that are seen at these hospitals. Therefore there is a need to improve access to services, particularly in the poor socioeconomic areas were the incidence of TBM is high as a large number of children recovering from TBM in this area may present with dysphagia.

In conclusion, South Africa has one of the worst TB epidemics and children in the region have a high risk of infection. The high incidence of childhood TB leads to an increased occurrence of TBM. One challenge faced in the Western Cape is late diagnosis of TBM resulting in later presentation of TBM (i.e. stage II and III) which is associated with neurological sequelae. The neurological impairments reported in children recovering from TBM may lead to difficulties with feeding and swallowing. While there is information regarding dysphagia in children with neurological problems and CP there is no specific research linking TBM to dysphagia. Research to determine whether dysphagia is associated with TBM is thus important, as the known consequences dysphagia may impact negatively on the child's health and quality of life.

The purpose of the study was to determine the occurrence and describe the nature of dysphagia in children recovering from TBM. The study investigated whether any associations existed between the occurrence of dysphagia and various other factors which included severity of TBM, neurological sequelae, age, and radiological findings. Identifying the specific feeding and swallowing difficulties of children recovering from TBM could provide baseline information to improve assessment and management of dysphagia as well and encourage early identification of dysphagia, which could be done by developing a screening protocol. In addition the information obtained may assist health care professionals in making appropriate referrals and decisions with regards to feeding management in children recovering from TBM. For example if health care professionals are alerted to an association between the stage of TBM and dysphagia they could be advised to refer to an SLT for assessment early. Lastly the present study may lay a foundation for future research for dysphagia in children recovering from TBM.

3. METHODOLOGY

3.1 AIMS AND OBJECTIVES

Aim

To describe the nature and prevalence of dysphagia in children (0-12 years) recovering from TBM.

Objectives

In children (0-12 years) recovering from TBM:

1. To describe the occurrence of dysphagia
2. To determine whether the presence of dysphagia is related to
    a) Stage of TBM
    b) Neurological sequelae
    c) Age at onset of TBM
    d) Central nervous system lesion
3. To describe the *nature* of dysphagia in terms of the
    a) Phases of swallowing
    b) Signs and disorders that present in each phase of swallowing
4. To describe the relationship, if any, between the *severity* (stages) of TBM and the *nature* of dysphagia (i.e. phases of swallowing).

3.2. RESEARCH DESIGN

The researcher used a retrospective descriptive survey design. *Descriptive research* involves explaining the characteristics of an observed phenomenon through either narrative or statistical methods (Babbie & Mouton, 2001; Irwin, Pannbacker & Lass, 2008; Leedy & Ormrod, 2005). A descriptive research design cannot be used to identify causal relationships (Babbie & Mouton, 2001). Nevertheless, this design allowed the researcher to describe dysphagia in children recovering from TBM and to explore possible associations between dysphagia and TBM (Leedy & Ormrod, 2005).

The use of a survey together with a descriptive design is referred to as *descriptive survey* design (Leedy & Ormrod, 2005; Maxwell & Satake, 2006; Polgar & Thomas, 1991). *Surveys* are investigations that provide accurate information regarding the characteristics of a population, which can also be used to identify associations and to establish possible links and trends between variables

(Gerrish & Lacey, 2006; Leedy & Ormrod, 2004; Polgar & Thomas, 1991). Descriptive surveys can be used for either prospective or retrospective research and the present study made use of a retrospective design. The methods of data collection usually involve interviews and questionnaires (Maxwell & Satake, 2006). The use of a descriptive survey was therefore selected for this study as it allowed the researcher to review medical records and document the feeding and swallowing abilities of the study population (Hicks, 2009).

An advantage of using the survey method is that it is cost effective (Maxwell &Satake, 2006; McCormack & Hill, 1997). The study could be controlled to meet the budget of the researcher without sacrificing the value of the findings. The survey method is also adaptable and allowed for an array of statistical methods to be used for data analysis, thus reducing the likelihood of false conclusions in interpreting the data (McCormack & Hill, 1997). These advantages therefore allowed the present study to analyse how various factors (e.g. age, stage of TBM) are related to dysphagia.

In *retrospective research* (Brink, 2006; Gerrish & Lacey, 2006; Irwin, et. al, 2008; Kumar, 2005; Maxwell & Satake 2006) a phenomenon is studied using existing data (Gerrish & Lacey, 2006; Kumar, 2005), which have not been collected for the purposes of research (Hess, 2004). The use of a retrospective design therefore allows for more data to be collected which reduces the likelihood of a type II error. Medical records of children diagnosed with TBM from the year 2006 to 2011 which were available at Red Cross War Memorial Children's Hospital (RCWMCH) were used to describe dysphagia in children (0-12 years) recovering from TBM. The availability of these records enabled the researcher to collect more data than could be collected using prospective research methods, hence maximising the data set (Hess, 2004).

The use of readily available records served as an advantage making it both inexpensive and time effective for the researcher to complete the study within a limited time period (Babbie & Mouton, 2001; Hess, 2004; Maxwell & Satake, 2006). A disadvantage of retrospective designs however is that the use of existing records provides the researcher with very little influence over the validity and reliability of the data and it is difficult to control confounding variables (Babbie & Mouton, 2001; Brodie et al., 1994; Hess, 2004). The researcher had to rely on the available data and some data was incomplete or unavailable (Brodie et.al, 1994; Hess, 2004). The medical records with inadequate information were excluded from the data set. However did not significantly reduce the sample size, as only 15 folders were excluded.

The researcher initially intended to collect data both prospectively and retrospectively in order to enhance the study findings by combining the data sets. However, data could only be collected prospectively on 6 participants. This sample size reflected the availability of participants during the data collection period (Maxwell & Satake, 2006). This data alone could not be used as it would have been difficult to analyse and would have yielded statistically insignificant results or a type II error. The data collected from the prospective research was combined with the retrospective data. The retrospective timeline was thus from January 2006 to June 2011. The two data sets could be combined as the same data collection tool was used for both components. Ultimately the study made use of a retrospective descriptive survey design as it allowed the researcher to achieve all the study aims.

3.3. RESEARCH METHOD

3.3.1. Study Population

The study population included records of all children with TBM admitted into Red Cross War Memorial Children's Hospital (RCWMCH) over a period of 5 and half years (January 2006- June 2011).

3.3.2. Selection criteria

As this was a retrospective study, selection criteria were in reference to the medical records of patients.

Inclusion criteria

In order for an individual's records to be selected for inclusion in this study, the following criteria needed to be met:

1. A *diagnosis of TBM* by a medical doctor, as the purpose of this study was to describe dysphagia in children with TBM.
2. *Age 0-12 years*: TBM is more prevalent in children under 13 years of age (Donald & Schoeman, 2004) and the purpose of the study was to report on the presence of dysphagia in this population.
3. Admission to RCWMCH with TBM between January 2006 and June 2011. This site was selected because paediatric patients with TBM are admitted to RCWMCH and there are Speech Therapy services available for the management of dysphagia.

Exclusion criteria

The records of individuals with any of the following were excluded from the study:

1. *Pre-morbid feeding difficulties:* Individuals with documented pre-existing feeding and/or swallowing difficulties were excluded from this study. Exclusion of these records ensured that the feeding or swallowing problems presented post onset of TBM and were not pre-existing difficulties (Terre Blanche & Durrheim, 1999).
2. *Risk factors for feeding difficulties:* Records with any documented risk factors for feeding and/or swallowing difficulties such as cleft palate and prematurity were critically reviewed. Any indications that the initial difficulties had not been overcome at the onset of TBM, resulted in exclusion from the study.
3. *Insufficient data:* All records that did not contain adequate medical and feeding information (e.g. date of diagnosis, stage of TBM and CT scan results) could not be used and were therefore excluded from the study.

   Note: The information from the first comprehensive clinical and instrumental feeding and swallowing assessment (when available) recorded in the participant medical records were documented. A participant was not excluded if they only had a clinical assessment; they were only excluded if there was not enough information regarding their feeding and swallowing skills.

*3.**3.3. Recruitment***

Once ethics approval was obtained from the University of Cape Town (UCT), Faculty of Health Sciences Human Research Ethics Committee (HREC Reference number: 517/2010) (Appendix A), permission was sought from the Western Cape Department of Health as well as the medical superintendent at RCWMCH (Appendix B). The medical records of potential participants were then identified using a list obtained from the hospital's electronic data base and the Speech-Language Therapy Department's clinical statistics. The list included all children diagnosed with TBM admitted into the hospital between January 2006 and June 2011. The medical records of the potential participants were requested and the researcher reviewed them to identify those that met the inclusion criteria.

3.3.4. Sampling Method

The researcher made use of *purposive sampling* (Babbie & Mouton, 2001; Maxwell & Satake, 2006, Teddlie & Yu, 2007). Purposive sampling is a form of non-probability sampling in which participants are selected based on specific criteria, which in this study was a diagnosis of TBM (Babbie & Mouton, 2001; Teddlie & Yu, 2007). Purposive sampling was therefore the most appropriate sampling method as it allowed the researcher to describe dysphagia in a specific population.

There are many advantages to using purposive sampling. Once the most suitable candidates for the study have been selected, the process of data collection becomes less time consuming (Babbie & Mouton, 2001). The fewer time constraints result in lower costs of carrying out the sampling. Another advantage is that when conducting research on a very rare or much sought after group of people, using purposive sampling maybe the only way to select the sample (Babbie & Mouton, 2001; Maxwell & Satake, 2006). This method was therefore appropriate, as the study only included children admitted to RCWMCH who had a diagnosis of TBM, as the study set out to explore dysphagia in children recovering from TBM (Babbie & Mouton, 2001; Macnee & McCabe, 2008).

A limitation of purposive sampling is that it may be prone to researcher bias because participants are selected based on the judgment of the researcher (Babbie & Mouton, 2001; Maxwell & Satake, 2006). In order to prevent researcher bias, selection criteria were identified prior to data collection and all records that met the criteria were included in the study. Another disadvantage of purposive sampling is that the sample obtained may not adequately represent the population which makes it difficult to generalise the findings (Maxwell & Satake, 2006). Despite these potential limitations, the research can still provide baseline information that will be useful to SLT's and other health professionals working with children recovering from TBM as well as prompt further studies in this area.

3.3.5. Sample size

The final sample size was 133 of an initial 309 potential participants. Thirty-three medical records could not be located at the Records Department or in the Archives; 128 did not meet the inclusion criteria; and 15 could not be used as there was missing information.

3.3.6. Description of Participants

The study population consisted of 133 participants' medical records, 49% (65/133) females and 51% (68/133) males.

The average age of the participants at diagnosis (of TBM) was 3 years 8 months and ranged from 1 month to 11 years 11 months.

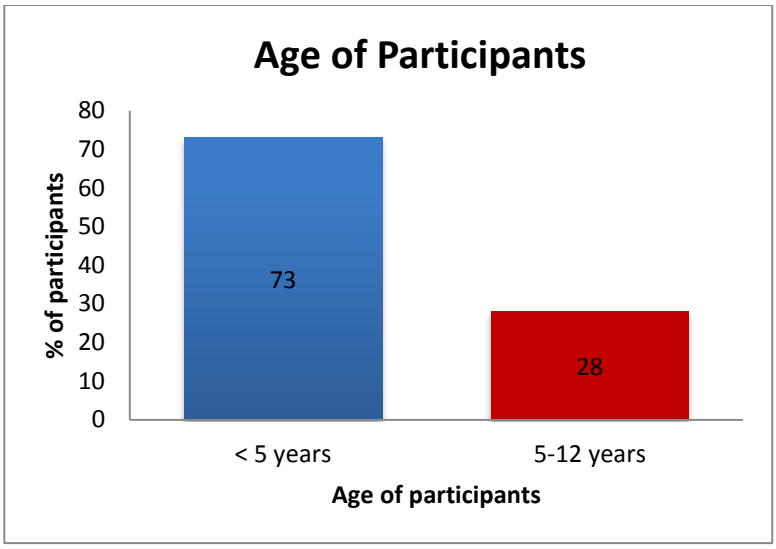


Figure 1: Age distribution of the participants (N=133)

Ninety-six (73%) of the participants were less than five years of age when they were diagnosed with TBM whilst only 37 (28%) were between 5-12 years of age.

The stage of TBM at initial presentation of participants was recorded (See Figure 2). Of the 133 participants, 40% (53/133) were in stage I TBM, 34% (45/133) were in stage II TBM, and 25% (33/133) were in stage III TBM. Two (1%) participants had missing information regarding the stage of TBM. A total of 59% (78/133) of the study population were diagnosed in stages II and III which is similar to other studies reported in the Western Cape.

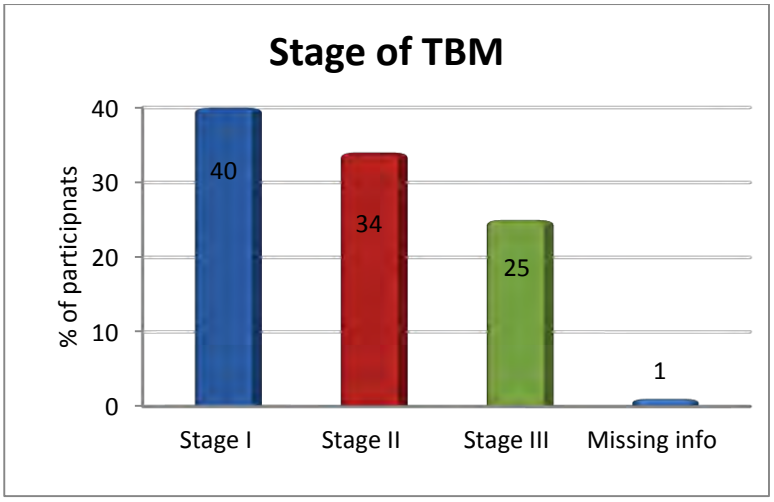


Figure 2: Stage of TBM at initial presentation (N = 133)

The age distribution of the participants showed that more than half of the study population (51%, 68/133) consisted of participants younger than five years of age who were in stage II (27%, 36/133) or stage III (24%, 32/133).

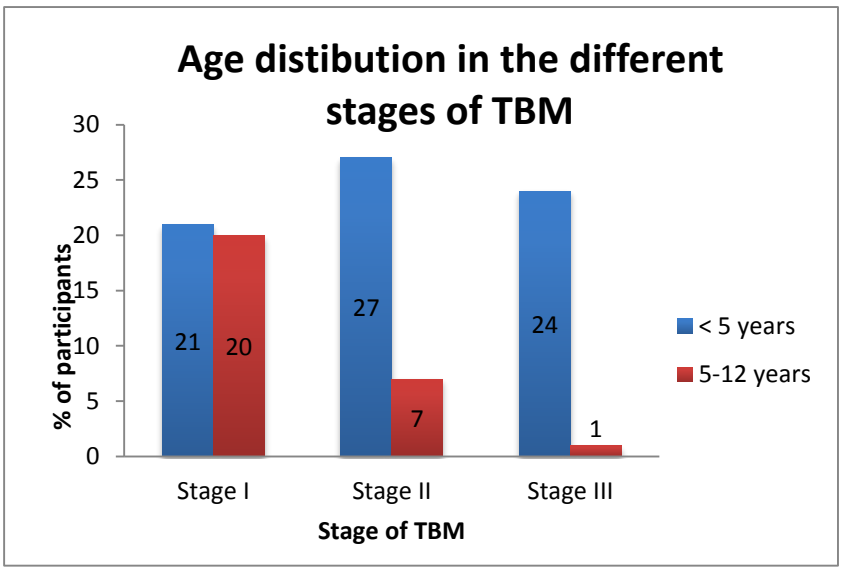


Figure 3: Age distribution in the different stages of TBM (N=133)

Seventy-two percent (96/133) of the participants presented with some form of neurological sequelae as a result of TBM. The nature of the sequelae that presented in the participants is shown in Figure 4. The category "other" neurological sequelae which were not listed included hypertonia, hypotonia, ataxia, cortical blindness and hemi-neglect. These individual sequelae each accounted for less than 4% and were therefore not reported separately.

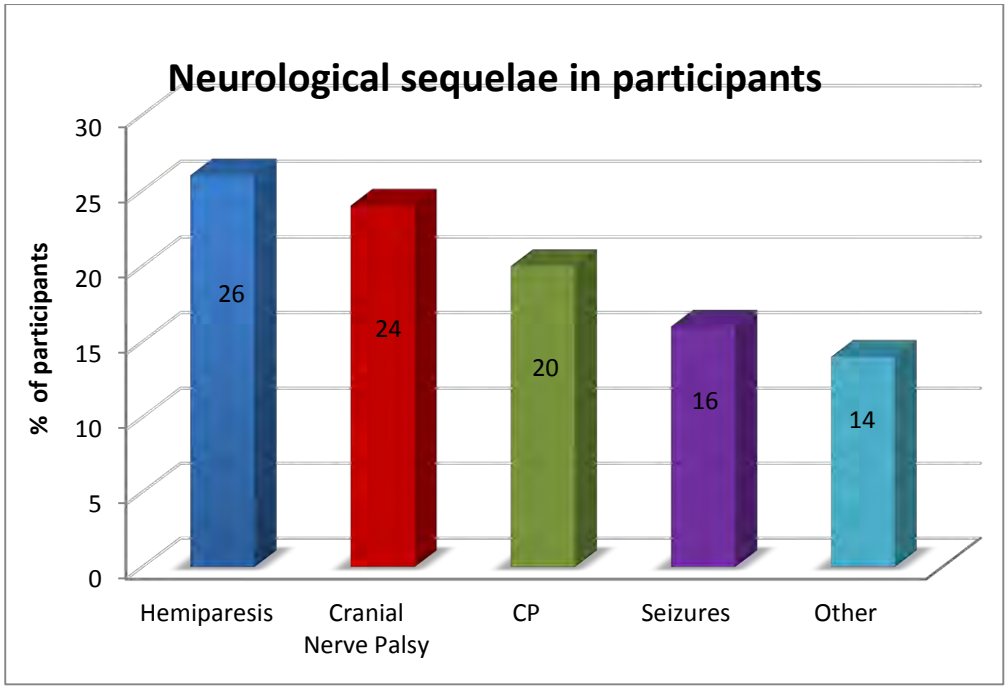
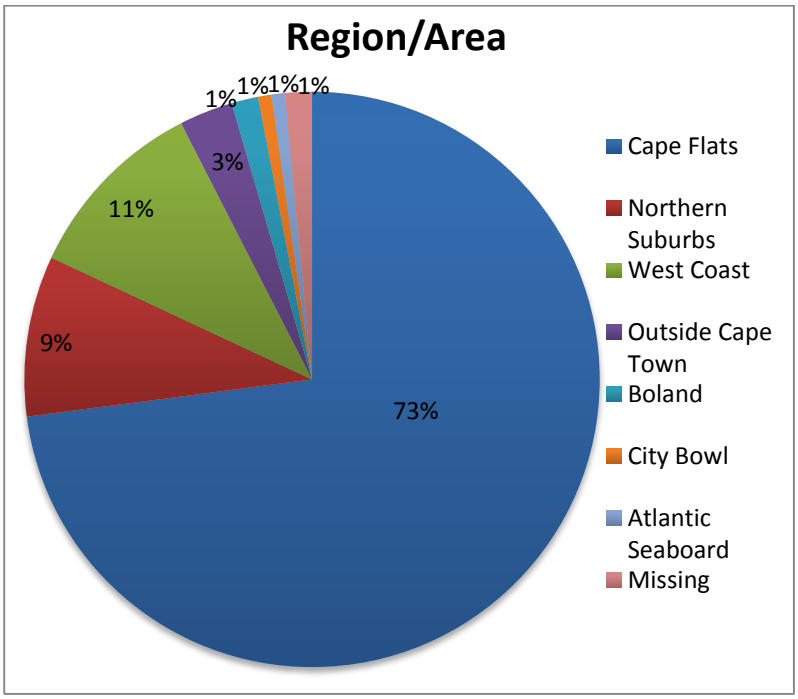


Figure 4: Presence of neurological sequelae in participants (N=133)

(Note: Although hemiparesis, hypertonia and hypotonia may indicate a diagnosis of CP, the researcher had to rely on the diagnoses provided in the participant folders and thus reported the diagnosis that was recorded in the folder. In addition, hemiparesis may be observed during the acute stage of TBM, but may resolve and not evolve into CP)

Nineteen (14%) of the participants were HIV positive of whom 47% (9/19) were already receiving Antiretroviral (ARV) treatment before being diagnosed with TBM; 32% (6/19) started ARV treatment after admission with TBM.

The geographic distribution of the participants is presented in Figure 5.



Key	
Cape Flats	Khayelitsha, Gugulethu, Langa, Mitchell's plain, Hanover Park, Nyanga, Bonteuwel, Athlone, Lansdowne, Mannenburg, Grassy park, Retreat, Mfuleni
Northern suburbs	Kuils river, Maitland Dunoon, Eerster river
West coast	Milnerton, Atlantis, Vredenburg, Saldanha, Clan William
Outside Cape Town	Engcobo, George, Thembalethu, Oudtshoorn
Boland	Ceres, Stellenbosch
City Bowl	Woodstock
Atlantic Seaboard	Mandela park

Figure 5: Areas from which the participants originated (N=133)

The Cape Flats had the largest number of participants with TBM and the distribution within districts is illustrated in Figure 6.

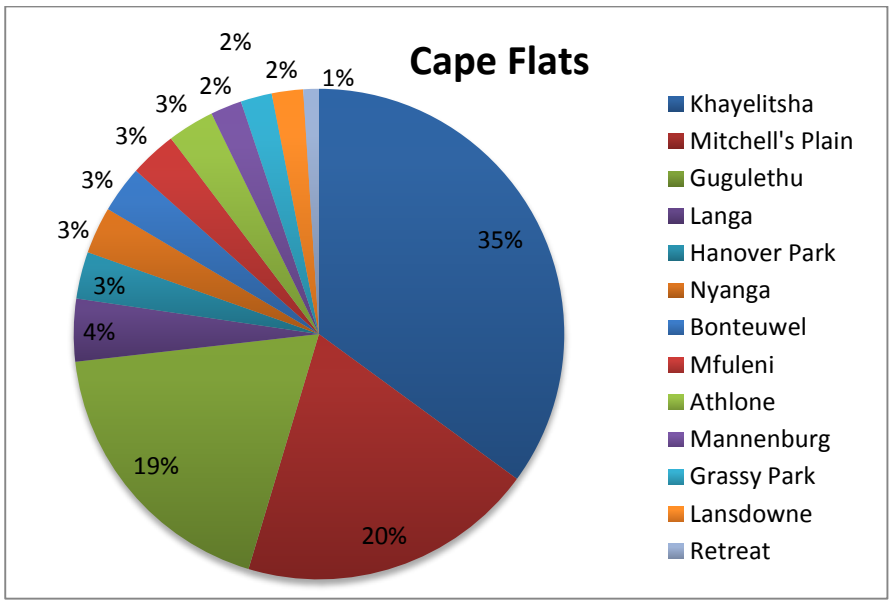


Figure 6: Distribution of participants from the Cape Flats district (n=97)

3.4. INSTRUMENTATION/MATERIALS

Feeding and Swallowing Checklist

The researcher developed a Feeding and Swallowing Checklist (Appendix C) to record data from the medical records. The items selected for inclusion in the checklist were based on available literature on both TBM and paediatric dysphagia. The rationale and references for the items recorded on the checklist are described in Table 2.

Table 2

Feeding and Swallowing Assessment Protocol

DATA	RATIONALE	REFERENCES
1. BIOGRAPHICAL INFORMATION		
1.1 Date of birth	To determine the age of the participant.	Babbie & Mouton, 2001
1.2 Sex	Provided descriptive information of the participant's sex.	Babbie & Mouton, 2001
1.3 Region/Area	Information regarding the place of residence assisted in identifying areas that have a high burden of disease and need with regards to service delivery.	
2. MEDICAL INFORMATION		
2.1 Date of TBM diagnosis	The date of diagnosis enabled the researcher to determine the age at diagnosis for inclusion criteria (i.e. between 0-12 years) and to determine whether there is any association between age and TBM.	Andronikou et al., 2004;Ramachandran, 2008; Springer et al., 2008
2.2 Stage of TBM	Clinical outcomes of TBM are associated with the stage of TBM at the time of presentation e.g. neurological sequelae occur more frequently in Stage 2 and 3. Stage information was used to describe the occurrence of dysphagia in the different stages. The stages were also used to describe any relationships that exist between the frequency and nature of dysphagia and the stages of TBM.	Bernaerts et al., 2003; Donald et al., 1999; Farinha et al., 2000; Kalita et al., 2007; Kumar, Singh & Kohli, 1999; Schoeman et al., 2004; Singhi & Singhi, 2001; Springer et al., 2008

2.3 CT Scan Results	CT scan findings are used to identify the CNS site of lesions that occur in children recovering from TBM. The most common sites of lesions include: hydrocephalus, basilar enhancement, cerebral infarcts, cerebral oedema, tuberculoma, periventricular lucency, exudates. These findings were included in participant description and were also used to determine any association between site of lesion and dysphagia.	Bernaerts et al., 2003; Donald et al., 1999; Farinha et al., 2000; Kalita et al., 2007; Kumar, et al., 1999; Newton, 1994; Schoeman et al., 2004; Singhi & Singhi, 2001; Springer et al., 2008; Thwaites et al., 2000; Tung et al., 2002.
2.4 Neurological Sequelae	Neurological sequelae commonly found in children recovering from TBM include: hemiparesis, athetosis, spastic quadriplegia, extra pyramidal syndrome, cranial nerve palsy, cognitive impairment and cerebral palsy. Feeding and swallowing difficulties often occur in children with neurological impairment. Neurological sequelae were included in the description of participants and also used to determine associations between the sequelae and the presence of dysphagia.	Bernaerts et al., 2003; Donald et al., 1999; Farinha et al., 2000; Kalita et al., 2007; Kumar et al., 1999; Newton, 1994; Schoeman et al., 2004; Singhi & Singhi, 2001; Springer et al., 2008; Thwaites et al., 2000; Tung et al., 2002.
2.5 HIV status (if documented)	HIV and TBM may co-occur; individuals with HIV have a greater risk of developing TBM. HIV/AIDS has also been associated with feeding and/or swallowing difficulties. The HIV status was included in the description of participants and the researcher noted whether participants had dysphagia resulting from TBM and not HIV.	D'Souza et al., 2002; Friedland et al., 2007; Halvorsen, Moelleken & Kearney, 2003; Myers, 2007; Soeters et al., 2005; Tutor & Gossa (2012)
3 FEEDING AND SWALLOWING INFORMATION		
3.1 Feeding difficulties noted	Prior to a clinical feeding and swallowing assessment by a SLT, other health professionals and/or guardians may report certain feeding and swallowing difficulties. These problems may include vomiting, GOR, nasal regurgitation, excessive gagging, recurrent lower respiratory tract infections (LRTI), increased drooling and other signs that indicate signs and symptoms of feeding and swallowing problems.	Arvedson & Brodsky, 2002; Hall, 2001
4 FEEDING AND SWALLOWING ASSESSMENT		
Data on the signs and symptoms of dysphagia were collected to describe the nature of feeding and/or swallowing difficulties. The information from the first comprehensive clinical and instrumental feeding and swallowing assessment (when available) recorded in the participant medical records were documented.		

4.1 ORAL PHASE (as documented clinically/instrumentally)

The participant folders did not specify or differentiate between the presence of oral preparatory and oral phase difficulties therefore the researcher combined these difficulties and reported them together, as is common in paediatric dysphagia literature.

SIGNS AND DISORDERS

4.1.1 Oral sensory difficulties • Hypersensitivity • Hyposensitivity • Abnormal gag • Food selectivity/oral aversion	Oral sensorimotor difficulties occur frequently in children with neurological impairments. Children with TBM present with neurological sequelae and could therefore present with similar difficulties. Oral sensory difficulties may also affect oral motor skills and feeding in general, which could lead to food selectivity and/or oral aversion. In addition oral sensorimotor difficulties may be associated with negative outcomes of using enteral feeding methods due to lack of positive oral stimulation. Therefore if children recovering from TBM require long term enteral feeding they could present with oral sensitivity. Documentation of the oral sensory difficulties was used to describe the nature of difficulties that occur in children with TBM.	Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; ASHA, 2002; Hall, 2001; Morris, 1998.
4.1.2.Oral motor difficulties • Weak/uncoordinated suck • ↓lip closure - anterior spillage • ↓lip tone - residue in anterior sulcus • ↓buccal tone - residue in lateral sulcus • ↓lingual function - reduced bolus formation and/or residue on floor of mouth or tongue • ↓lingual anterior-posterior movement - poor bolus propulsion • ↓lingual elevation - residue on hard palate • ↓lingual-velar seal and/or reduced lingual coordination - premature spillage into pharynx • ↓mandibular movement - ineffective biting or chewing • Increased oral phase time (OPT) • Increased mealtimes	Oral motor difficulties provided the researcher with information that facilitated the description of their nature and frequency they presented in children recovering from TBM. Oral motor skills are essential for the oral preparatory and oral phases of swallowing. Children with neurological impairments frequently exhibit oral motor dysfunction. Oral preparatory and oral phase difficulties include poor lip closure, a weak suck, reduced tongue strength, abnormal movement patterns. These difficulties often make feeding difficult and time consuming. In addition some oral phase difficulties such as premature spillage caused by poor bolus control and/or insufficient lingual-velar seal may also result in aspiration which affects respiratory health. Other negative consequences of oral motor difficulties include oral aversion and malnutrition.	Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; ASHA, 2002; Calis et al., 2008; Fung et al., 2002; Hall, 2001; Hinchcliffe, 2007; Morris, 1998;; Southall & Martin, 2010; Sullivan, 2000; Workinger, 2005.

4.2 PHARYNGEAL PHASE (as documented instrumentally with additional clinical signs)		
SIGNS AND DISORDERS		
4.2.1.Clinical signs • Coughing • Gurgly/Wet voice • Choking/Spluttering	Data on clinical signs was obtained from records of previously administered assessments. These clinical signs provide only suggestions of possible pharyngeal phase difficulties. Therefore coughing, choking, spluttering may be signs of aspiration that can only be confirmed by an instrumental assessment.	Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; ASHA, 2002; Hall, 2001; Southall & Martin, 2010; Sullivan, 2000; Workinger, 2005.
4.2.2. Instrumental • ↓lingual velar seal - delayed trigger of the swallow/bolus in pyriform sinuses or valleculae • ↓ pharyngeal muscle strength - multiple swallows per bolus • ↓velopharyngeal closure/ nasopharyngeal backflow • Laryngeal penetration– bolus in laryngeal area up to the level of the true vocal folds • ↓insufficient laryngeal elevation and closure and/or poor coordination - Aspiration (before, during or after the swallow) • Silent aspiration • ↓anterior laryngeal movement - residue in valleculae • ↓posterior pharyngeal wall contraction - residue on pharyngeal wall • Dysfunctional upper oesophageal sphincter (UOS) • Increased pharyngeal transit time (PTT)	The pharyngeal phase of swallowing is best assessed instrumentally as a radiographic study provides a dynamic view of the swallow process. Instrumental signs and disorders were included to obtain a comprehensive description of the nature of feeding and swallowing difficulties present in this phase. Children with neurological impairment often have difficulties in the pharyngeal phase of swallowing, which include uncoordinated swallow, delay and/or absent triggering of the pharyngeal swallow and uncoordinated suck-swallow-breathe sequence. Aspiration is one of the most common pharyngeal phase difficulties in children with neurological impairments with the incidence estimated to be between 68-70%. Aspiration is considered to be the most life threatening consequence of dysphagia; therefore a comprehensive assessment of this phase is crucial. Aspiration may occur without any outward signs of swallowing difficulty, therefore the pharyngeal phase is best assessed instrumentally i.e. using a VFSS) to ensure that aspiration does not remain undetected resulting in respiratory illnesses. Thus although clinical feeding assessments provide signs of possible pharyngeal phase difficulties, only an instrumental assessment can confirm presence of these difficulties.	Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; ASHA, 2002; Calis et al., 2008; Fung et al., 2002; Gangil et al., 2001; Hall, 2001; Hinchcliffe, 2007; Morris, 1998; Rogers, 2004; Southall & Martin, 2010; Sullivan, 2000; Workinger, 2005.

Note. ↓= reduced or limited

3.5. VALIDITY AND RELIABILITY

Validity relates to whether the research instrument measures what it intended to measure (Babbie & Mouton 2006; Kumar, 2005). Three aspects of validity viz. content, face and construct validity of the checklist were addressed (Babbie & Mouton 2006; Leedy & Ormrod, 2004; Kumar, 2005). The reliability of the data collection tools and process were also addressed.

3.5.1. Face Validity

Face validity is judgment by an expert based on the logical link that exists between the questions the instrument seeks to answer and the objective of the study (Kumar, 2005). This type of validity was used to address the relevance of the checklist (Maxwell & Satake, 2006). When using new materials that have not been standardised, such as the Feeding and Swallowing Checklist, in this study, it is advised to seek expert opinion before finalising the materials (Maxwell & Satake, 2006). Face validity of the checklist was established by expert opinion from two SLTs with five or more years working experience in paediatric dysphagia. The feedback from the SLTs indicated the need to include certain important items that are necessary when assessing paediatric feeding and swallowing.

The checklist was modified to include an additional fifteen items that were suggested by the SLTs (e.g. oral and pharyngeal transit times, laryngeal penetration and dysfunctional upper oesophageal sphincter [UOS]). The checklist was also changed to include signs and symptoms of dysphagia (e.g. delayed trigger of the swallow/bolus in pyriform sinuses or valleculae), because in clinical practice often the sign rather than the disorder may be reported in the medical folder. Some items that were deemed not relevant to children were removed from the checklist.

3.5.2. Content Validity

Content validity addresses whether items included in the checklist are representative of the research area and if they achieve the research aims (Babbie & Mouton, 2006; Howell et al., 2005). The materials have to be evaluated and their inclusion has to be explained logically (Irwin et al., 2008). The present study investigated dysphagia in children; therefore the researcher had to ensure that all the different elements that relate to paediatric dysphagia were included in the checklist. There were no standard materials available to assess the feeding and swallowing abilities of children diagnosed

with TBM therefore, the checklist was based on the available paediatric dysphagia literature to ensure that the relevant content was included (See references in Table 2).

3.5.3. Construct validity

Construct validity is the degree to which inferences can be made from the findings of the study (Babbie & Mouton, 2006; Howell et al., 2005). Construct validity aimed to evaluate the extent to which the checklist created by the researcher was able to capture documented dysphagia in participants. In order to establish construct validity the researcher must seek evidence of an agreement between a theoretical concept and the hypothesis being tested (Howell et al., 2005; Irwin et al., 2008). Construct validity of the Feeding and Swallowing Checklist was determined by convergent validity. Convergent validity refers to the degree to which an operation (i.e. the checklist) is similar to other operations (i.e. other instruments used for assessing feeding and swallowing) (Berg, 2004). The checklist used for data collection contained items that appear on various other feeding and swallowing assessment tools (Arvedson & Brodsky, 2002; Lefton-Greif & Arvedson, 2007).

3.5.4. Reliability

Reliability is the consistency and dependency of the materials used for data collection or the rater conducting it (Babbie & Mouton, 2006; Howell et al., 2005; Polgar & Thomas, 1991). Reliable materials are those that are able to obtain the same information more than once under similar conditions (Kumar, 2005). The consistency of the data collection was determined by intra-rater and inter-rater reliability (Howell et al., 2005; Irwin et al., 2008; Maxwell & Satake, 2006; Waltz & Lenz, 2005).

Intra-rater reliability is the degree of internal consistency displayed by the researcher when the same procedure is completed on two or more occasions (Howell et al., 2005; Leedy & Ormrod, 2005; Waltz & Lenz, 2005). The researcher randomly selected 7% (n=10) of the participant medical records and these were used to calculate intra-rater reliability. Since the researcher had reviewed these records previously, a two-week interval was allowed to lapse prior reassessment to ensure that the researcher was blinded to the previous results (Waltz & Lenz, 2005). A predetermined 95% criterion level for agreement was set and the results of the two data sets were compared. The researcher's intra-rater reliability rating was 98% (Irwin et al., 2008; Maxwell & Satake, 2006).

Inter-rater reliability assesses the degree to which the data collected is consistent when obtained by two different raters (Howell et al., 2005; Waltz & Lenz, 2005). It can be expressed in percentages that reflect the degree of agreement or disagreement and if the findings have a high percentage of agreement, they are likely to be measuring the same construct (Maxwell & Satake, 2006). A second rater (a 4th year student Audiology student at the University of Cape Town) was trained by the researcher and familiarized with the check list before inter-rater reliability was tested. The researcher randomly selected 5% of the participants' medical records and these were reviewed by the second rater blind to the initial findings. Different statistics can be used to calculate inter-rater reliability and the method selected for this study was joint-probability of agreement. Joint-probability of agreement was calculated by analysing the extent to which two raters gave the same rating on items on the checklist. The percentage of agreement between the two raters was 98%.

3.6. DATA COLLECTION PROCEDURE

Ethics approval was granted by the UCT Faculty of Health Sciences Human Research Ethics Committee (HREC Reference no: 517/2010). Thereafter permission was sought and granted by the Western Cape Department of Health. Following this permission was granted by the medical superintendent at RCWMCH. The researcher then began the process of identifying the medical records of potential participants. These records were identified using a list obtained from the hospital's electronic data base and the Speech-Language Therapy Department's clinical statistics. The list included all children admitted into the hospital between 2006 and 2011 that were diagnosed with TBM. The researcher then reviewed the medical records to identify those that met the selection criteria and those that did had codes assigned to them to ensure patient anonymity.

Validity checks (i.e. face, content and construct validity as well as the pilot study) were performed on the checklist before data collection began. Thereafter the validity was verified and the checks were completed, modifications were made to the data collection tool and procedure protocol. Once the checklist was finalised the researcher began data collection and within two weeks of beginning the process of data collection data intra-rater reliability was assessed as described earlier. Once intra-rater reliability was established the researcher then trained the second rater. After training the second rater, inter-rater reliability was determined. Finally when all the medical records had been reviewed, data was extracted as per the checklist for each participant and captured electronically on Excel spreadsheets.

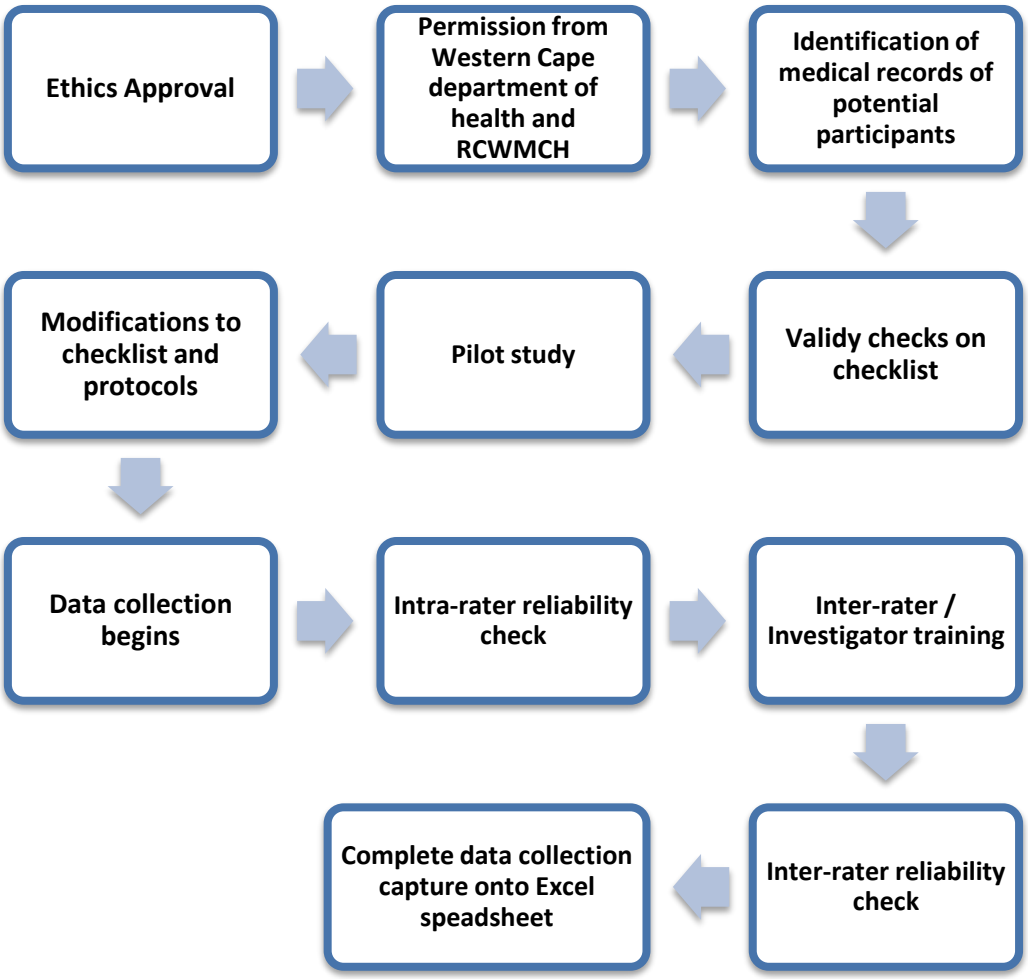


Figure 7: Data collection procedure flowchart

3.6.1. Pilot study

Prior to the initiation of the actual study - a pilot study was conducted to assess the logistical procedures of the study and to gather information to improve its quality and efficiency (Leedy & Ormrod, 2005). The researcher randomly selected medical records of two participants who met the inclusion criteria for the study. The checklist was then used to review the selected medical records. The pilot study revealed deficiencies in the design of the proposed procedures which resulted in the following changes to the checklist:

1. The structure of the checklist was changed by creating boxes for placing a tick or cross so that the researcher did not have to write details when filling out the checklist, making the data collection procedure easier and less time consuming.
2. Provision was made for making comments, which allowed for additional information to be collected were necessary.
3. Additions were made so that the source of the data collected, e.g. clinical assessment or VFSS could be specified.
4. Changes to the data collection tool necessitated a revision of the instructions to the second rater on how to complete the checklist.

The checklist was revised after the pilot study and deemed effective prior to beginning the study. The two medical records used for the pilot study were not included in the results.

3.7. DATA ANALYSIS

The raw data on the checklists were converted to binary data to enable the representation and identification of different categories of entities e.g. the nature of dysphagia and the stage of TBM (Leedy & Ormrod, 2005). The nominal data collected from the checklist was then analysed using descriptive statistics (i.e. frequency counts). Frequency counts as well as measures of central tendency, variance and range were used for participant description; i.e. sample size, age distribution, stage distribution, neurological sequelae and geographical distribution; as well as analysing the prevalence of dysphagia in the study population, the different stages of TBM, age groups, and the frequency of the signs and disorders in the different phases of swallowing

Inferential statistics were used to determine possible associations among variables (Leedy & Ormrod, 2005; Lowry, 2009). The Chi-square test (χ^2test), was used to determine whether the following variables were associated with dysphagia; stage of TBM, age at onset of TBM, site of lesion and neurological sequelae. The χ^2test was also used to evaluate whether any relationship existed between the severity of TBM and the nature of dysphagia. Statistical significance was determined using an alpha level of .05 (Lowry, 2009).

3.8. ETHICAL CONSIDERATIONS

Ethics approval to conduct the study was obtained from the UCT Faculty of Health Sciences Human Research Ethics Committee (HREC Reference number: 517/2010). The principles promoted by the Declaration of Helsinki (2013) as well as the Medical Research Council (MRC) considered in this study were (1) autonomy, (2) beneficence (3) non-maleficence and (4) justice.

3.8.1. Autonomy

Autonomy entails respect for persons and their human dignity (MRC, 2000). The autonomy of the individuals that participate in studies is often ensured by providing the participants with informed consent and by maintaining anonymity and confidentiality (Flick, 2006; McQuoid-Mason, 2008). However, in retrospective chart reviews, the informed consent is often waived as the research involves no risk to the subject and the waiver will not adversely affect the rights and welfare of the subjects and there is no contact with the participant (Flick, 2006).

Researchers are obligated to take all possible steps to keep all the information obtained anonymous and confidential in order to protect the participants (MRC, 2000). Anonymity refers to concealing the identities of participants in all documents resulting from the research and this was obtained by allocating codes to the participants (Flick, 2006). Confidentiality is concerned with who has the right of access to the data provided by the participants. The researcher ensured that the data collected were kept separately from the codes linking the data to the individuals. Once reliability checks were complete, the list of the participants' names was destroyed. All the data collected were stored safely in a password protected file on a laptop computer ensuring that no one else had access to the data (Flick, 2006).

3.8.2. Beneficence

Beneficence requires that the researcher put the welfare of the participant first and does no harm (Orb, Eisenhauer & Wynaden, 2000; Leedy & Ormrod, 2005; Research Ethics Guidelines, 2004). Beneficence involves balancing the benefits of the study against the risks and costs involved (Orb et al. 2000). Due to the retrospective nature of the study, there were no direct benefits for participants. The results of the study will be made available to the institution were the data was

collected (MRC, 2004). In addition the study findings will also be disseminated in the form of a dissertation and journal article allowing other health professionals access to the findings which may assist them in gaining a better understanding of dysphagia in the paediatric TBM population (Leedy & Ormrod, 2005). The information obtained from the current study may therefore have benefits for children recovering from TBM in the future.

3.8.3. Non-maleficence

The principle of non-maleficence involves ensuring that there is no harm to the research participants (Leedy & Ormrod, 2005; MRC, 2000). Since the study entailed a review of records there was no direct risk to any of the participants.

3.8.4. Justice

Justice upholds fairness or equity in research (Orb et al., 2000; Research Ethics Guidelines, 2004). The researcher should ensure fair distribution of benefits and burdens and avoid exploitation and abuse of participants (Leedy & Ormrod, 2005; Orb et al., 2000). The principle of justice holds that no participants should be unfairly excluded from study (Orb et al., 2000). The researcher did not exclude individuals or groups from participation for reasons that are unrelated to the research and therefore all medical records that met the criteria were included in this study.

4. RESULTS

The results of the study are presented according to the aim and objectives described in the methodology.

4.1 THE OCCURRENCE OF DYSPHAGIA IN PARTICIPANTS (0-12 YEARS) RECOVERING FROM TBM

Twenty-six percent (35, N=133) of the participants had dysphagia. The occurrence of dysphagia was calculated as a percentage of the sample population (N=133).

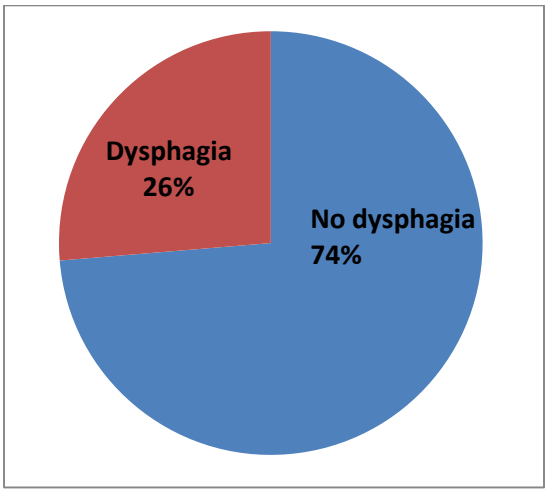


Figure 8: The prevalence of dysphagia in the sample population of children recovering from TBM (N=133)

4.2 RELATIONSHIP BETWEEN DYSPHAGIA AND VARIOUS VARIABLES

4.2.1 The relationship between the severity (stage) of TBM and dysphagia

Dysphagia occurred in all three stages of TBM, but most frequently in participants with stage III TBM (18%; N=133). Figure 8 presents the frequency of dysphagia in the different stages of TBM.

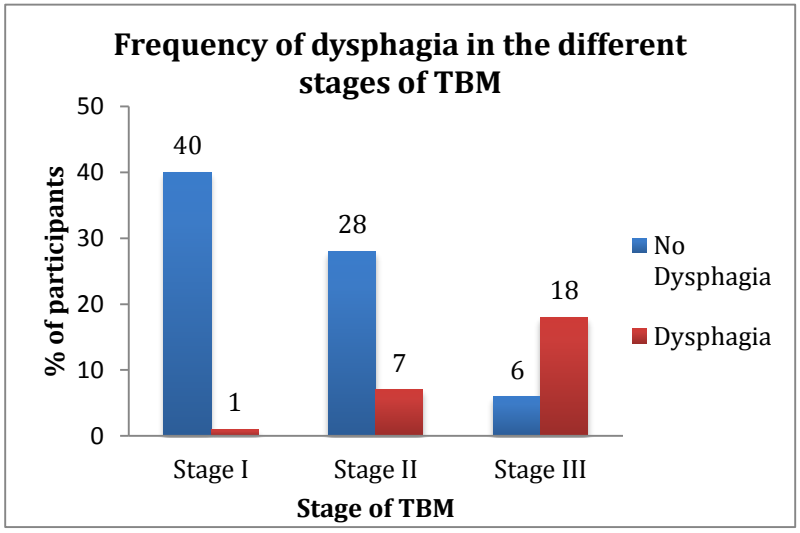


Figure 9: Frequency of dysphagia in the different stages of TBM (N=133)

Significantly more participants in stage III presented with dysphagia than participants in stage I (χ^2=46.74; *p*< .001) and in stage II ($\chi^2$19.74; *p*< .001). Dysphagia also occurred statistically significantly more often in stage II than in stage I (χ^2 8.82; *p*=.002). The occurrence of dysphagia in participants with TBM is significantly associated with the stage of TBM (χ^2=58.26; *p*< .001), suggesting that the frequency of dysphagia increased with the severity of TBM (see figure 8).

4.2.2 The relationship between neurological sequelae and dysphagia

Of the 96 participants who displayed neurological sequelae 33 presented with dysphagia (34%; n=96). The majority of participants diagnosed with CP presented with dysphagia (77%; 20/26). CP was diagnosed more frequently in the participants with more advanced stage of TBM, i.e. stage I (n=0/26), stage II (n=2/26) and stage III (n=24/26).There was a significant association between CP and the presence of dysphagia (χ^2= 42.22; *p*< .001) (See Table 3).

Table 3

Neurological sequelae and their association with dysphagia (n=132)

Neurological sequelae	Dysphagia (%)		Test value	*p*-value
	Present	Absent		
Hemiparesis	8 (6)	26(20)	0.21	.647
Cerebral palsy	20 (15)	6(5)	42.22	<.001*
Cranial nerve palsy	5 (4)	27(20)	2.57	.109

*Note.** *p*< .05.

4.2.3 The relationship between age at onset of TBM and occurrence of dysphagia

Eighty-nine percent (31/35) of participants who presented with dysphagia were younger than five years of age. Being less than five years of age at onset of TBM was significantly associated with the presence of dysphagia (χ^2= 6.36; *p* = .01), suggesting that this group are at high risk of dysphagia.

4.2.4 The relationship between site of lesion and occurrence of dysphagia

The most commonly occurring radiological (CT scan) findings were hydrocephalus (73%, 96/131), basal meningeal enhancement (63%, 82/131) and cerebral infarcts (53%, 69/131). There was a significant association between cerebral infarcts and the presence of dysphagia (χ^2=11.47; *p*< .001), as well as hydrocephalus and the presence of dysphagia (χ^2= 8.03; *p* = .005). There was no significant association with basal meningeal enhancements, cerebral oedema or any other lesions (see Table 4).

Table 4

Radiological findings and their association with dysphagia (n=131)

CT Scan findings	Dysphagia (%)		Test value	*p*-value
	Present	Absent		
Hydrocephalus	32 (24)	64(49)	8.03	.005*
Cerebral infarcts	27 (21)	42 (32)	11.47	<.001*
Basal meningeal enhancement	25 (19)	57 (44)	1.59	.207
Cerebral oedema	18 (14)	33 (25)	3.14	.077

Note. p*< .05.

4.3 THE NATURE OF DYSPHAGIA IN CHILDREN RECOVERING FROM TBM

4.3.1 The nature of dysphagia in terms of the phases of swallowing

The majority of the participants with dysphagia (66%; 23/35) presented with difficulties in both the oral and the pharyngeal phases of swallowing, while 28% (10/35) presented with difficulties in only one phase of swallowing. There was no information regarding the nature of dysphagia for the remaining 2 participants with dysphagia (See figure 9).

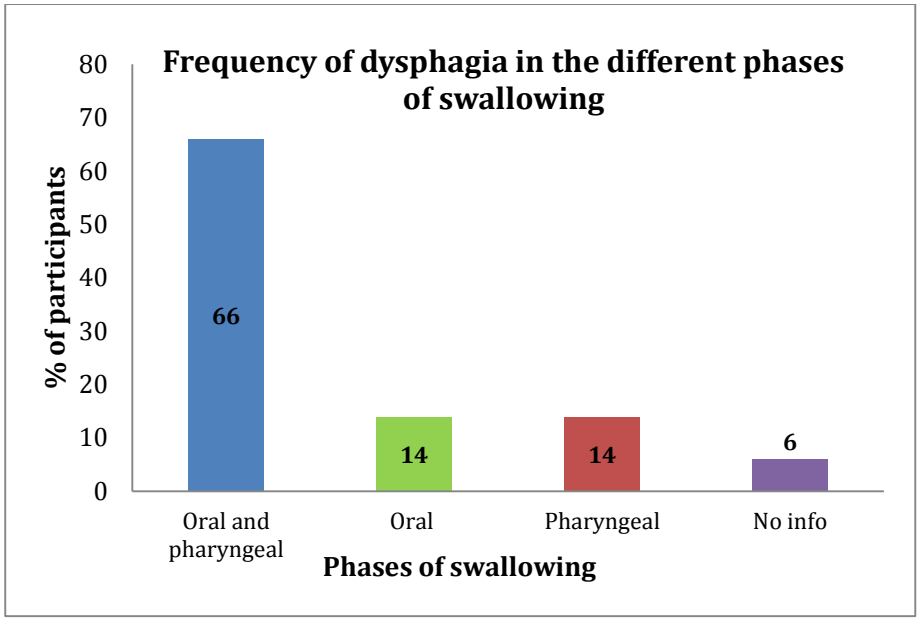


Figure 10: Occurrence of dysphagia in the different phases of swallowing (n=35)

Significantly more participants presented with oral and pharyngeal difficulties than those with oral phase difficulties only (Z= 4.15; two sided *p*-value < .001) and those with pharyngeal difficulties only (Z= 4.15; two sided *p*-value < .001).

4.3.2 The nature of dysphagia as described by the signs and disorders

The signs and disorders the participants displayed were categorised into the different phases of swallowing i.e. oral preparatory and oral phase and pharyngeal phase signs and disorders. Participants had a range of signs and disorders, signalling difficulties in the oral preparatory and oral phases of swallowing. The majority of the participants demonstrated difficulties with *bolus formation*; reduced lingual shaping and coordination (63%, 22/35), *bolus holding*; reduced lip closure (67%, 24/35), reduced lingual strength and/or coordination (66%, 23/35), as well as *bolus propulsion*; reduced lingual anterior-posterior movement (40%, 14/35). Reduced lip tone (40%, 14/35) and reduced buccal tone (43%, 15/35) affected both bolus formation and holding. Aspiration that occurred as a result of oral phase difficulties (11%, 4/35) was attributed to premature spillage resulting from reduced lingual- velar seal (See Table 5).

Table 5

Oral phase signs and disorders in participants with dysphagia (n=35)

ORAL PHASE DISORDERS	**n (%)**
Oral sensory difficulties:	
Hyperactive responses	1 (3)
Hyposensitivity	1 (3)
Food selectivity/oral aversion	2 (6)
Oral motor difficulties:	
Weak/uncoordinated suck	7 (20)
(a)*Bolus formation*	
↓ lip closure - anterior spillage	*24 (67)
↓ lingual shaping and co-ordination - reduced bolus formation	*22 (63)
↓lip tone - residue in anterior sulcus	14 (40)
↓buccal tone - residue in lateral sulcus	15 (43)
↓ mandibular movement - ineffective biting and/or chewing	2 (6)
(b)*Bolus holding*	
↓lingual strength and/or coordination - residue on floor of mouth	*23 (66)
↓ lip tone - residue in anterior sulcus	14 (40)
↓buccal tone - residue in lateral sulcus	15 (43)
↓ lingual-velar seal and/or reduced lingual coordination - premature spillage into pharynx	5 (14)
(c)*Bolus propulsion*	
↓ lingual anterior-posterior movement - poor bolus propulsion	14 (40)
↓ lingual elevation - residue on hard palate	12 (34)
Increased oral transit time (OPT)	10 (29)
Aspiration resulting from oral phase difficulties	4 (11)

Note. ↓ reduced or limited * more than 50%

Dysphagic participants presented with pharyngeal phase difficulties that included; delayed trigger of the swallow and aspiration. Delayed trigger of the swallow was the most frequently occurring difficulty presenting in 71% of the participants with dysphagia and it was also the most common cause of aspiration. Aspiration due to pharyngeal phase difficulties was documented in 45% of the dysphagic participants, with most of the aspiration occurring before and/or during swallowing (See Table 6).

Table 6

Pharyngeal signs and disorders in participants with dysphagia (n=35)

PHARYNGEAL PHASE DISORDERS	**n (%)**
↓ lingual velar seal -delayed trigger of the swallow /bolus in pyriform sinuses or valleculae	*25 (71)
↓ pharyngeal muscle strength - multiple swallows per bolus	3 (9)
Laryngeal penetration – bolus in laryngeal area up to the level of the true vocal folds	1 (3)
↓insufficient laryngeal elevation and closure and/or poor coordination - aspiration	*19 (54)
Before the swallow	9 (25)
During the swallow	9 (25)
After the swallow	2 (6)
Silent aspiration	4 (11)
Not stated	6 (17)
↓anterior laryngeal movement - residue in pyriform sinus	5 (14)
↓ posterior pharyngeal wall contraction - residue on pharyngeal wall	4 (11)

Note. ↓ reduced or limited * more than 50%

The total number of participants with dysphagia that aspirated was 54% (19/35), with GOR occurring in 51% (18/35) of the participants. GOR co-occurred with aspiration as 72% (13/18) of the participants who had GOR also presented with aspiration.

4.4 THE RELATIONSHIP BETWEEN THE SEVERITY OF TBM AND NATURE OF DYSPHAGIA

Oral and pharyngeal phase difficulties were observed in all three stages of TBM. There was no statistically significant difference (χ^2=8.54; p= .074) between the nature of dysphagia that the participants presented with and the stage of TBM. Therefore no relationship could be established between the severity of TBM and the nature of dysphagia.

An association was however noted between aspiration and the severity of TBM. No participants in stage I presented with aspiration, while 89% (n=17) of the participants who aspirated were in stage III. There was a statistically significant difference (χ^2= 6.81, p=.03) in the occurrence of aspiration in the different stages of TBM, indicating that the frequency of aspiration increased with the severity of TBM.

5. DISCUSSION

The major finding of the present study was the occurrence of dysphagia in 26% (n=35) of the study population, indicating that in high burden TBM areas as many as 1 in 4 children recovering from TBM could develop dysphagia. In view of this finding, health service providers should be aware and ensure early identification and speedy referrals to SLT's in order to prevent and/or minimise any negative consequences associated with dysphagia.

Although the literature is limited, two unpublished undergraduate studies evaluated dysphagia in children recovering from TBM. The reported occurrences of dysphagia in these studies were 33% and 47% respectively (Allies et al., 2009; Bengsch & Lourenco, 2005). There are disparities in the occurrence of dysphagia reported by these three studies and these may be attributed to differences in dysphagia definitions, study designs and sample sizes.

A common challenge in dysphagia literature is that, the term 'dysphagia' does not always carry the same definition. In this case, Bengsch & Lourenco (2005) investigated only the oral motor and feeding abilities, and did not consider the pharyngeal phase difficulties and although Allies et al. (2009) investigated dysphagia in the oral and pharyngeal phases of swallowing they used 'dysphagia' and 'feeding and swallowing difficulties' inter-changeably. The studies by Bengsch & Lourenco (2005) and Allies et al (2009), included both feeding difficulties and swallowing difficulties, unlike the present study which only considered swallowing difficulties (i.e. dysphagia), thus these studies may have included more participants resulting in a higher occurrence of dysphagia. Overall, the differences in the definitions make it difficult to compare the occurrence reported in these studies as they investigated different aspects.

Different methodologies and inclusion criteria may also affect the information that is obtained in studies as well as the sample size (Babbie & Mouton, 2001; Maxwell & Satake, 2006). The present study utilised a retrospective design, which enabled the researcher to obtain a large sample size (N=133). Bengsch & Lourenco (2005) and Allies et al. (2009) in contrast used prospective descriptive study designs which yielded small sample sizes of 17 and 12 respectively. The difference reported in the occurrence of dysphagia in these studies may be attributed to effect size, i.e. when the sample size is larger the effect that can be detected is smaller and the reverse is true for smaller sample sizes. The present study is therefore likely to have reported a smaller effect i.e. 26% than Bengsch & Lourenco (2005) and Allies et al. (2009) who may have over-estimated the prevalence of

dysphagia as small samples sizes often report a larger effect. Despite the differences in sample size, methodologies and definitions, the present study, Allies et al. (2009) and Bengsch & Lourenco (2005) clearly indicate that children between 0-12 years recovering from TBM are at risk of dysphagia following TBM.

The results of the study demonstrated a relationship between the occurrence of dysphagia and the severity of TBM, i.e. the frequency of dysphagia increases with the severity of TBM. Although dysphagia occurred in all stages, significantly more participants in stage III presented with dysphagia than participants in stage I (χ^2=46.74; p< .001) and in stage II (χ^2=19.74; p< .001).

The increased occurrence of dysphagia in children with stage II and III TBM was expected as these stages are associated with neurological impairments which are considered a major risk factor for dysphagia in children (Arvedson & Brodsky, 2008). In fact the present study demonstrated that 34% (n=96) of participants who displayed neurological impairments, presented with dysphagia, suggesting that the neurological sequelae resulting from TBM places children at risk for dysphagia. In addition the present study also showed that not only is dysphagia linked to neurological impairments but the severity of neurological impairment is strongly associated with the presence of dysphagia, which is also reported in populations with neurological diagnoses (Arvedson & Brodsky, 2002; Calis, et al., 2008; Cichero& Murdoch, 2006; Fung et al., 2002; Hall 2001; Sullivan, 2000). Stage III, which has higher risks of developing irreversible neurological damage, consequently had the highest percentage of dysphagia.

Allies et al. (2009) obtained similar results, with dysphagia only presenting in participants with stage II and III TBM. The present study and Allies et al. (2009) therefore both highlight that the frequency of dysphagia increases with the severity of TBM, an association which is likely attributed to the links between neurological impairments and dysphagia (Arvedson & Brodsky, 2002; Calis et al., 2008; Fung et al., 2002; Gangil et al., 2001; Hinchcliffe, 2007; Rogers, 2004; Sullivan, 2000).

Although the links between dysphagia and neurological deficits explain the presence of dysphagia in children who presented with neurological sequelae, 6% (3/35) of the dysphagic participants presented with no neurological involvement. Due to the retrospective nature of the present study the researcher was unable to determine the specific causes of dysphagia in these participants, however; clinical features of TBM such as respiratory illness, fever, vomiting and altered levels of consciousness as well as loss of appetite caused by TB medication may also affect a

child's feeding and swallowing abilities (Arvedson & Brodsky, 2002; Hall, 2001; Manikam & Perman, 2000). In addition illness and hospitalization may reduce a child's motivation to feed, as they may be experiencing discomfort, pain, fatigue and stress (Arvedson & Brodsky, 2002; Hall, 2001; Manikam & Perman, 2000). The above mentioned factors thus could also account for the occurrence of dysphagia in children recovering from TBM.

Stage II and III TBM are typically associated with delays in identification and/or diagnosis of TBM (Andronikou et al., 2006; Chiang et al., 2014; Farinha et al, 2000; Kalita et al, 2007; Schoeman, 1990; van Toorn et al., 2014; Van Well et al., 2009. The present study reported a large number of children were diagnosed in stage II and III 56% (78/133) and neurological sequelae in 72% (96/133) of the study sample. Van Well et al. (2009) reported similar findings with 71% of the study population presenting with some kind of neurological sequelae. Although older studies (Donald et al., 1999; Van Well et al., 2009) indicated a trend of late diagnosis in a large number of children in the Western Cape region, a more recent study (Nabukeera- Barungi et al. 2014) reported earlier diagnosis of TBM and mortality lower than in previous studies. Nevertheless it is clear that health care professionals should aim to improve identification methods, and ensure that more children are identified in stage I which has better outcomes and a lower risk of neurological sequelae and dysphagia based on this study. Clinicians should be aware that children presenting with stage II and III have a higher risk of dysphagia than those in stage I, although they would not be expected to identify dysphagia, they can ensure early referrals to SLTs.

The neurological sequelae documented in the present study included, CP, hemiparesis and cranial nerve palsies. CP occurred in fewer participants i.e. 20% of the study population compared to the 53% reported by Berman et al. (1992) amongst survivors of TBM in the Western Cape. Over 20 years have lapsed since the study by Berman and these findings suggest either improved management of TBM which has led to better clinical outcomes or a general improvement in access to health care facilities and primary health care (PHC) post-apartheid.

In the present study, 77% (20/26) of the participants with CP presented with dysphagia; CP was significantly associated with the presence of dysphagia. Children with CP experience sensory-motor impairments which often result in dysphagia (Arvedson & Brodsky, 2002; Benfer et al., 2013; Calis, et al., 2008; Cichero & Murdoch, 2006; Erkin et al., 2010; Fung et al., 2002; Hall 2001; Sullivan, 2000; Workinger, 2005) and the estimated prevalence of dysphagia in children with CP is between 85-95%. The occurrence of dysphagia in children with CP in the present study, not only compares to

estimated prevalence reported in other studies, it also documented a significant association between CP and dysphagia, suggesting that children who present with CP as a result of TBM are at high risk of dysphagia.

In addition to investigating the occurrence of dysphagia, the present study reported on the nature of dysphagia as a function of the phases of swallowing. Oral and pharyngeal phase difficulties occurred unvaryingly, with the majority of the participants displaying difficulties in both phases of swallowing. Results indicated a co-occurrence of oral and pharyngeal phase difficulties in 66% of the dysphagic participants, suggesting that children recovering from TBM who present with dysphagia are more likely to present with challenges in both phases. The co-occurrence of oral and pharyngeal phase difficulties can be explained by established links between neurological impairments and dysphagia discussed earlier. The challenges that children with neurological impairments experience with movement, posture, tone and sensation can result in difficulties in any of the phases of swallowing (Benfer et al., 2012; Calis et al., 2008; Erasmus et al., 2012; Gangil, et al., 2001; Kim et al., 2013; Van den Engel-Hoek et al., 2013).

Allies et al., (2009) documented a co-occurrence of oral and pharyngeal phase difficulties in three out of four dysphagic participants. Whilst there is a discrepancy to what extent the difficulties co-occur, it is clear nonetheless that children recovering from TBM are more likely to present with dysphagia in both phases of swallowing. Literature supports the present findings as the occurrence of dysphagia in multiple phases had been documented in children with complex medical conditions (Field et al., 2003; Oosthuizen, 2012; Rommel et al., 2003). Children recovering from TBM presenting with dysphagia may therefore require both clinical and instrumental assessments.

In the oral phase of swallowing, participants displayed signs and disorders in three main areas namely bolus formation, bolus holding and bolus propulsion. The most commonly occurring difficulties included, reduced lip closure (67%, 24/35), reduced lingual strength and/or coordination (66%, 23/35) and reduced lingual shaping and coordination (63%, 22/35). These difficulties have also been described in children with neurological impairments (Arvedson & Brodsky, 2002; Fung et al., 2002; Hall, 2001; Hinchcliffe, 2007; Kim et al., 2013; Manikam & Perman, 2000; Reilly et al., 1996; Schwarz, 2003) and are usually due to reduced oral muscle strength and poor coordination associated with neurological diagnoses. There are also notable similarities in the nature of difficulties reported by the present study and those documented by Allies et al. (2009) and Bengsch & Lourenco

(2005) in children recovering from TBM. All three studies documented reduced lingual movement as well as reduced lip closure all attributed to the neurological impairments as a result TBM.

Although Allies et al. (2009) and Bengsch & Lourenco (2005) documented reduced lingual movement, which leads to difficulties with bolus management and increases the risk of premature spillage, they did not report on aspiration resulting from oral phase difficulties. In the present study premature spillage was the most commonly reported oral phase difficulty which resulted in aspiration, making up 11% of participants who aspirated. Bengsch and Lourenco (2005) did not conduct instrumental assessments and could not determine whether premature spillage occurred or not. Similarly Allies et al. (2009) conducted only clinical assessments, and therefore could not document the occurrence of premature spillage and possible aspiration as a result of premature spillage. The findings of the present study suggest that children recovering from TBM presenting with oral phase difficulties are at risk of aspiration resulting from premature spillage, therefore assessments need to be comprehensive and include an instrumental assessment when indicated, to ensure that aspiration does not go undetected.

In addition the oral phase difficulties reported may lead to slow feeding and increased meal times which affects nutritional intake (Benfer et al., 2012; Hall, 2001). The present study documented increased oral phase time in 29% (10/35) of the dysphagic participants indicating that children recovering from TBM who present with oral phase difficulties could be at risk of decreased nutritional intake leading to malnutrition, poor growth, and / or stressful mealtimes (Arvedson & Brodsky, 2002; Fung et al., 2002; Hinchcliffe, 2007; Manikam & Perman, 2000; Rogers, 2004; Sullivan, 2000).

As observed in the oral phase, the most common signs and symptoms reported in the pharyngeal phase were similar to those seen in children with neurological impairments (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; Hall, 2001; Calis et al., 2008; Rogers, 2004), suggesting that neurological sequelae resulting from TBM are likely to affect the complex sequential coordination required for safe swallowing. Neurologic sequelae caused by TBM may affect a child's movement, posture, tone and sensation resulting in pharyngeal dysmotility and aspiration (Arvedson & Brodsky, 2002; Benfer et al., 2012; Calis et al., 2008; Erasmus et al., 2012; Gangil et al., 2001; Kim et al., 2013; Van den Engel-Hoek et al., 2013).

The most common pharyngeal phase difficulty observed in the present study was absent and/or delayed trigger of the swallow occurring in 71% (25/35) of the participants with dysphagia. In children with neurological impairments, absent/and or delayed trigger of the swallow may be caused by motor and/or sensory deficits and often leads to aspiration (Cass et al., 2005; Rogers, 2004; Sullivan et al., 2009; Weir et al., 2011). Other factors, such as immobility, weak respiratory muscles, and poor cough observed in children with neurological impairments also increase their chances of aspiration (Rogers, 2004; Sullivan et al., 2009).

Aspiration was documented in 54% (19/35) of the participants with dysphagia and was also significantly associated with the severity of TBM. There was a higher frequency of aspiration in children with more severe TBM, i.e. stage II and III, which may be attributed to the link between aspiration and severity of neurological impairments (Cass, et al., 2005; Rogers, 2004; Sullivan et al., 2009; Weir et al., et al., 2011). Literature suggests that children with severe neurological impairments are at increased risk of aspiration because of the presence of motor and sensory deficits (Cass, et al., 2005; Rogers, 2004; Sullivan et al., 2009; Workinger, 2005) and have a high risk of silent aspiration with as many as 97% aspirating silently (Cass et al., 2005), although the present study only reported silent aspiration in 21% of participants who aspirated.

The disparities between the findings can be attributed to the fact that the present study used a retrospective study design and relied on the available data. It is therefore possible that some children recovering from TBM, aspirating silently may have not been detected or assessed instrumentally. Early identification and management of aspiration as it may prevent the potentially negative consequences which include respiratory compromise, respiratory illness and lung disease (Andrew & Sullivan, 2012; Cass et al., 2005; Rogers, 2004; Weir et al., 2011). The effects aspiration can have may further exacerbate illness in children recovering from TBM and worsen outcomes. Pharyngeal phase difficulties are less easily identified by family members, caregivers and health care professionals than oral phase difficulties, as they require instrumental assessment which is not always available. Therefore it is important to develop protocols that ensure that children with pharyngeal phase involvement do not go undetected and receive the intervention they require.

In addition aspiration co-occurred with GOR. Findings indicated that more than half of the participants (51%, 18/35) with dysphagia also presented with GOR and of these 72% (13/18) also presented with aspiration. The association between GOR and aspiration is seen frequently in children with neurological impairments (Duca, Dantas, Rodrigues, & Sawamura, 2008; Giambra et al.,

2010; Oosthuizen, 2012). These children often present with oesophageal motility disorders, or impaired opening of the lower oesophageal sphincter which may result in GOR (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif 1998; Hall, 2001) and they are at risk of aspirating the GOR. The present study however did not investigate the reason why an association between aspiration and GOR exists and the causal relationship should perhaps be considered in future studies. Nevertheless, it is clear that adequate management of both GOR and aspiration is very important as both are associated with negative consequences. In addition GOR may result in food refusal, increased sensitivity and growth faltering and may therefore possibly account for the presence of dysphagia in some of those participants who did not have neurological impairments (Rommel et al., 2003). Further research into the impact/effect of GOR has on feeding and swallowing is also essential (Rommel et al., 2003; Oosthuizen, 2012).

Emphasis is once again placed on the need for routine assessment of all phases of swallowing in order to avoid overlooking potential cases of dysphagia. Where it is not possible for instrumental assessments to be conducted, monitoring for signs of aspiration (e.g. coughing, choking and spluttering associated with feeding and swallowing) should be implemented as provisional measures of detecting pharyngeal phase difficulties.

In addition to the severity of TBM, the present study also identified other factors associated with dysphagia which could assist health care professionals in identifying children who would be at risk of dysphagia. These factors included late diagnosis CP which was discussed earlier as well as, being less than five years of age at onset of TBM, hydrocephalus and site of lesion.

There was an association between young age (i.e. less than five years at onset of TBM) and the presence of dysphagia, suggesting that this age group is at high risk of dysphagia. Eighty-nine percent of the participants who presented with dysphagia were below five years. Younger children were also at increased risk of severe neurological sequelae (Kalita et al., 2007; Mahadevan, et al., 2002; Schoeman, 1990; Thwaites et al., 2009; Van Well et al., 2009) suggesting that dysphagia in this age group may also be related to neurological sequelae associated with TBM. In addition children less than five years of age (more particularly under 2) are also still developing and consolidating feeding skills which may also place them at greater risk for having dysphagia (Rommel et al., 2003).

In fact younger children are more vulnerable to TBM as can be seen in the present study as they made up over 70% of the study population. The vulnerability of younger children is supported

by other local studies on TBM were 78% - 82% of the participants were less than five years of age (Berman et al., 1992; Schoeman, 1990; van Well, et al., 2009). Young children's vulnerability to TBM emphasizes the need for service providers to enhance prevention programmes and give special consideration to children less than five years, e.g. by conducting educational and awareness programmes for mothers and caregivers on TBM, so they may refer their children early.

The radiological findings in children recovering from TBM are prognostic indicators used to predict outcomes of TBM and in the present study hydrocephalus (73%, 96/131), basal meningeal enhancement (63%, 82/131) and cerebral infarcts (53%, 69/131) were the most commonly occurring. In the present study hydrocephalus and cerebral infarcts were significantly associated with dysphagia. Hydrocephalus and cerebral infarcts place children at risk of poor neurological and developmental outcomes and are linked to late diagnosis of TBM as these lesions occurred in children with stage II and III TBM (Andronikou et al., 2006; Bernaerts et al., 2003; Chiang et al., 2014; Farinha, et al., 2000; Mahadevan et al., 2002; Springer et al., 2008; Thwaites et al., 2000; Van Well et al., 2009). These findings yet again signal the need to promote early diagnosis and reduce poor outcomes which may result in dysphagia.

Bengsch & Lourenco (2005), Allies et al. (2009) and the present study, all studies conducted in the Western Cape where there is a high incidence of TBM, indicate that health professionals in high burden TBM areas are most likely to encounter dysphagia in children recovering from TBM. In the present study the majority of the participants originated from the Cape Flats with the most affected areas being, Khayelitsha, Gugulethu and Mitchells Plain. The Cape Flats area falls in the high burden TB districts in Cape Town Metropole and this suggests why a large number of participants originated from these areas (Western Cape Metropole & City of Cape Town, 2004).

Previously there were only community and primary health care services available in these areas, however since 2012 Khayelitsha District Hospital and Mitchells Plain hospitals were opened. The opening of these hospitals may result better access to general healthcare and possibly earlier diagnosis of TBM. In addition, there are SLT services at these sites which may lead to earlier detection of dysphagia in the paediatric TBM population in these areas. Although the Western Cape has adopted TB promotion and prevention programmes which have improved some clinical outcomes and opened new hospitals, overall findings suggest the continued need for health service providers to improve TB prevention strategies as well as improve service delivery targeting high burden TB districts to reduce the incidence of TB and TBM.

In light of the findings of the present study, service providers and health professionals in high burden TBM areas, should be aware of the occurrence and nature of dysphagia within the paediatric TBM population and develop measures that will assist them in improving outcomes. Management protocols should be put in place which could include screening as well as prompt and appropriate referrals to SLT's for dysphagia management. One example of a screening protocol may include SLTs screening all children presenting with stage II and III TBM. Silent aspiration may however not be detected during screening and health care professionals working with children recovering from TBM should therefore be alerted to this possibility.

Findings indicate that when considering dysphagia in children recovering from TBM, health professionals should not focus on one phase of swallowing, but rather ensure comprehensive assessment of both the oral and the pharyngeal phases. In the Western Cape, instrumental assessment is only available at tertiary hospitals, thus referrals systems should be established between the primary and secondary level facilities and tertiary level hospitals for VFSS in order to view and identify pharyngeal phase difficulties and aspiration. In addition health service providers may also consider the possibility of obtaining the necessary equipment (e.g. radiographic equipment for VFSS) at secondary levels, in order to increase ease of access to SLT services resulting in a general improvement in service delivery.

In order to effect the changes in managing dysphagia in this population, SLTs should be included in the team managing children recovering from TBM. SLT's should consider the information obtained by the present study for developing effective assessment and management tools for children recovering from TBM. Screening protocols, referral systems and obtaining the relevant equipment would improve identification of dysphagia and management, enhance clinical outcomes, reduce the financial burdens associated with prolonged hospitalisations and improve quality of life of children recovering from TBM.

5.1 LIMITATIONS, CLINICAL IMPLICATIONS AND FURTHER RESEACH

The limitations of the present study relate to the methodology and the generalisability of the study results. The present study made use of retrospective descriptive study which surveyed medical records. Although the use of a retrospective design allowed for more data to be collected, the researcher had to rely on the information documented in the medical records and could not verify

the data. The retrospective nature of the study also meant that 48 potential participants had to be excluded from the study because they had missing or inadequate information. A prospective study therefore should be considered in the future to overcome under utilisation of data and to ensure inclusion of as many participants as possible.

Despite these limitations, the results provided a comprehensive description of the nature of dysphagia in children recovering from TBM. The findings highlight the occurrence of dysphagia in children recovering from TBM and impresses on the need to focus on children with more severe TBM (i.e. stage II and III) as well as young children (i.e. less than five years). The present study also draws attention to the nature of feeding and swallowing difficulties that present in children recovering from TBM, which informs health professionals on how to manage children that present with dysphagia. In general, the study shows the need to put in place appropriate referral systems to STLs which will improve the management of dysphagia and encourage early identification of feeding and swallowing disorders for the paediatric population with TBM.

The present study was limited to RCWMACH, thus the study may not be generalised to the entire TBM population (Babbie & Mouton, 2001; Macnee & McCabe, 2008; Teddlie & Yu, 2007). A larger and more expansive research in the Western Cape Province and throughout the country investigating dysphagia in children recovering from TBM is recommended. The study did however manage to highlight the sub-districts which have a high incidence of TB and TBM within Cape Town in the Western Cape of South Africa. Most of the areas identified do not have SLT services, or have limited services, as these are focused at the tertiary hospitals. Efforts should be made to improve access to SLTs in areas that have a greater need for their services. While the introduction of SLT services at the various levels may take time, an interim solution may be to develop an effective referral system between primary, secondary levels and tertiary levels that allows for early identification and timely referrals to SLTs.

Lastly, the present study has highlighted the gaps in literature on dysphagia in children with TBM, indicating the need for further research. Although the undergraduate study by Allies et al. (2009) set the foundation for research in this area and the present study expanded on it, there is still need for further research. Further studies could explore the development of comprehensive assessment and management protocols for dysphagia and/or the effects of long-term tube feeding in the paediatric TBM population. Future studies could also consider including children with co-morbidities to determine whether TBM and other co-morbidities places children at greater risk of

dysphagia. The research knowledge would benefit the health delivery system to improve management of the TBM population.

6. CONCLUSION

The present study has provided a description of the occurrence and nature of dysphagia in children recovering from TBM at a tertiary dysphagia clinic in the Western Cape, South Africa. This information may assist and guide health care professionals who work in high burden TB areas to anticipate the occurrence dysphagia in children recovering from TBM and also inform them on nature of difficulties to expect when managing these children in a clinical setting. The results of the present study demonstrated that as much as 1 in 4 children recovering from TBM may present with dysphagia. A co-occurrence of oral and pharyngeal phase difficulties was noted demonstrating the need for comprehensive feeding and swallowing assessment to manage dysphagia.

Dysphagia was observed in all stages of TBM, but occurred more frequently in stage II and III which are associated with neurological impairment. The nature of the difficulties noted were similar to those seen in children with neurological impairments (Arvedson & Brodsky, 2002; Arvedson & Lefton-Greif, 1998; Hall, 2001; Calis et al., 2008; Rogers, 2004), suggesting that neurological sequelae resulting from TBM are likely to result in dysphagia. A large number of children were diagnosed in stage II and III, which placed them at risk for dysphagia, emphasising the need to improve early identification for TBM.

Aspiration was documented in over half of participants with dysphagia and was associated with the severity of TBM and co-occurred with GOR. These findings suggest need for comprehensive assessment which includes instrumental assessments, of children recovering from TBM who present with dysphagia, particularly when aspiration is suspected. Referral systems between primary and secondary level facilities and tertiary level hospitals for instrumental assessment should also be established.

The present study also established associations between dysphagia and hydrocephalus, site of lesion and young age (i.e. being less than five years of age at onset of TBM) which could assist health care professionals in identifying children who would be at risk of dysphagia. Prompt diagnosis and management of dysphagia may subsequently prevent the negative consequences of dysphagia which include growth failure, malnutrition, and respiratory illness and ultimately affects a child's quality of life.

The importance early identification and referral to SLTs as well as the need for a multidisciplinary team approach to identify dysphagia has been emphasised. The role of the SLT in dysphagia management has been highlighted as has been the need for SLTs to be included in the team managing children recovering from TBM at various levels of care.

Effective management of dysphagia will ensure enhanced clinical outcomes, reduce the financial burdens associated with prolonged hospitalisations as well as improve quality of life of children recovering from TBM. Improving clinical outcomes could lead to reduced child mortality which would assist South Africa achieving the UN millennium development goals (South Africa & UNDP, 2013).

7. References

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8. APPENDICES

APPENDIX A: Ethics Approval

UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6626 • **Facsimile** [021] 406 6411
e-mail: lamees.emjedi@uct.ac.za

12 November 2010

HREC REF: 517/2010

Miss W Makanza
Communication Sciences & Disorders
Health & Rehab
F Floor
OMB

Dear Miss Makanza

PROJECT TITLE: DYSPHAGIA IN CHILDREN (0-12 YEARS) RECOVERING FROM TUBERCULOSIS MENINGITIS (TBM)

Thank you for submitting your new study to the Faculty of Health Sciences Human Research Ethics Committee.

It is a pleasure to inform you that the FHS HREC has **formally approved** the above-mentioned study.

Approval is granted for one year until 15 November 2011.

Please send us an annual progress report (website form FHS 016) if your research continues beyond the approval period. Alternatively, please send us a brief summary of your findings so that we can close the research file.

Please note that there is a standardised application form on the HREC's website to conduct research at the Red Cross Children's Hospital.

Minor typo: Appendix B
....i.e. if your child has (delete is) feeding and swallowing difficulties referrals will be made for (delete to) further assessments.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the REC. REF in all your correspondence.

lemjedi

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637.
Institutional Review Board (IRB) number: IRB00001938

This serves to confirm that the University of Cape Town Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on Harmonisation Good Clinical Practice (ICH GCP) and Declaration of Helsinki guidelines.

The Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.

lemjedi

APPENDIX B: Consent letter to medical superintendent RCWMCH

School of Health and Rehabilitation Sciences
Faculty of Health Sciences
Divisions of Communications Sciences and Disorders, Nursing and Midwifery, Occupational Therapy, Physiotherapy

F45 Old Main Building, Groote Schuur Hospital,
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January 2011

To the Medical Superintendent

I am a Masters Speech and Language Therapy student at the University of Cape Town and I am conducting a study to describe dysphagia in children 0-12 years recovering from tuberculosis meningitis (TBM). I therefore request permission to conduct my research project at Red Cross Children's Hospital /Brooklyn Chest Hospital as a large number of paediatric patients with TBM are admitted to this hospital.

This will involve:

1. Reviewing the medical records of participants.
2. Conducting a clinical swallowing assessment. The assessment will include giving the participants small amounts of liquid (e.g. water, milk), semi-solid (e.g. cereal, yoghurt), and a solid (e.g. biscuit, bread), if the child is eating these foods. The assessment will take between 20-30minutes and the researcher will also observe the child eating during other times.

The study will be conducted during the first 6 months of 2009. The researcher will arrange appropriate times to collect data with the relevant ward staff once permission has been granted. The medical superintendent will be kept informed. The researcher is a qualified Speech and

Language Therapist with clinical experience in paediatric dysphagia and will be supervised by an experienced clinician in the field.

The information gained from this study will assist Speech and Language Therapists in developing appropriate feeding and swallowing management protocols for children recovering from TBM.

Participants' anonymity and confidentiality will be maintained throughout the research process. Participation in this study is entirely voluntary and participants will be informed of their right to withdraw from the study at any stage. There will be no financial cost for Red Cross Children's Hospital / Brooklyn Chest Hospital or the participants. Ethics approval has been obtained from the Faculty of Health Sciences Human Research Ethics Committee (FHS HREC) Reference no: 517/2010

Kind regards

If you have any further questions or concerns, please feel free to contact me or my research supervisor:

Researcher: Wadzanai Makanza

Contact number: 076 435 3343

Email:wadzmakanza@yahoo.com

Research Project Supervisor: Vivienne Norman

Contact number: 083 414 7928

Email: vivienne.norman@uct.ac.za

Head of Faculty of Health Sciences Human Research Ethics Committee: Prof Blockman

Contact number: 021 404 6492

APPENDIX C: Feeding and Swallowing Checklist

Code_____

FEEDING AND SWALLOWING ASSESSMENT PROTOCOL				
BIOGRAPHICAL INFORMATION				
Date of birth				
Sex				
Region/Area				
MEDICAL INFORMATION				
Hospital				
Date of TBM diagnosis				
Stage of TBM				
CT Scan result (✓✗) **Date:_____**	Hydrocephalus		Cerebral infarcts	
	Basal enhancement		Tuberculoma	
	Cerebral edema		Exudate	
	Periventricular lucency		Other:_____	
CT Scan result (✓✗) **Date:_____**	Hydrocephalus		Cerebral infarcts	
	Basal enhancement		Tuberculoma	
	Cerebral edema		Exudate	
	Periventricular lucency		Other: _____	
Neurological sequelae (✓✗)	Spastic quadriplegia		Hemi paresis	
	Cranial nerve palsy		Cognitive impairment	
	Athetosis		Cerebral palsy	
	Extrapyramidal syndrome		Other:_____	
HIV Status (if documented)	Y / N			
ARV's (if documented)	Y / N Date started_____			

FEEDING AND SWALLOWING INFORMATION				
Method of feeding (✓✗)			Start	End
	Oral		Date:_____	Date:_____
	NGT		Date:_____	Date:_____
	Oral + NGT		Date:_____	Date:_____
	PEG		Date:_____	Date:_____
	Other:_____		Date:_____	Date:_____
	Comments:_____			
Reported feeding difficulties (comment)	_____			

ORAL PREPERATORY AND ORAL PHASE					
	LIQUIDS	**SEMI-SOLID**	**SOLID**	**SOURCE**	**DATE**
Presentation (circle)	Bottle Cup Sippy-Cup Spoon Syringe	Cup Spoon Sippy-Cup	Spoon fork finger-feeding	Folder Clinical MBS	
ORAL SENSORY DIFFICULTIES: (✓✗N/A)					
Hyperactive responses				Folder	
				Clinical	
				MBS	
Specify:	_____				
Hyposensitivity				Folder	
				Clinical	
				MBS	
Specify:	_____				
Abnormal gag				Folder	
				Clinical	
				MBS	

Food selectivity/oral aversion				Folder	
				Clinical	
				MBS	
Specify:	_____				
ORAL MOTOR DIFFICULTIES: (✓✖N/A)					

Suck: *Rhythm* *Strength* *Rate* *Bursts*	**(✓✖N/A)**					
	Rhythmic		Disorganised		N /A	
	Absent		Poor		Adequate	
	Slow		Adequate		Rapid	
	Reduced		Normal		Increased	
	Comments:_____					

↓ lip closure/anterior spillage				Folder	
				Clinical	
				MBS	
↓ lip tone/residue in anterior sulcus				Folder	
				Clinical	
				MBS	
↓ buccal tone/residue in lateral sulcus				Folder	
				Clinical	
				MBS	
↓ lingual function/reduced bolus formation and/or residue on floor of mouth or tongue				Folder	
				Clinical	
				MBS	
↓ lingual anterior-posterior movement/poor bolus propulsion				Folder	
				Clinical	
				MBS	
↓ tongue elevation/ residue on hard palate				Folder	
				Clinical	
				MBS	

↓ lingual-velar seal and/or reduced lingual coordination /premature spillage into pharynx				Folder	
				Clinical	
				MBS	
↓ tongue shaping or coordination /residue on floor of mouth or tongue				Folder	
				Clinical	
				MBS	
Tongue thrust				Folder	
				Clinical	
				MBS	
Retraction of tongue				Folder	
				Clinical	
				MBS	
Tonic bite				Folder	
				Clinical	
				MBS	
Increased oral transit time				Folder	
				Clinical	
				MBS	
Increased mealtimes				Folder	
				Clinical	
				MBS	
Normal oral phase				Folder	
				Clinical	
				MBS	

Comment:_____
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PHARYNGEAL PHASE					
	LIQUIDS	**SEMI-SOLID**	**SOLID**	**SOURCE**	**DATE**
Presentation (circle)	Bottle Cup Sippy-Cup Spoon Syringe	Cup Spoon Sippy-Cup	Spoon fork finger-feeding	Folder Clinical MBS	
CLINICAL SIGNS (✓✗N/A)					
Coughing				Folder	
				Clinical	
				MBS	
Gurgly/Wet voice				Folder	
				Clinical	
				MBS	
Choking/Spluttering				Folder	
				Clinical	
				MBS	
Absent swallow				Folder	
				Clinical	
				MBS	
INSTRUMENTAL FINDINGS (✓✗N/A)					
↓ lingual velar seal - Delayed trigger of the swallow/bolus in pyriform sinuses or valleculae				Folder	
				MBS	
↓ pharyngeal muscle strength - Multiple swallows per bolus				Folder	
				MBS	
↓ velopharyngeal closure/ nasopharyngeal backflow				Folder	
				MBS	
Laryngeal penetration – bolus in laryngeal area up to the level of the vocal folds				Folder	
				MBS	
Aspiration					

• *Before the swallow*				Folder	
				MBS	
• *During the swallow*				Folder	
				MBS	
• *After the swallow*				Folder	
				MBS	
Silent aspiration				Folder	
				MBS	
↓ anterior laryngeal movement/residue in valleculae				Folder	
				MBS	
↓ posterior pharyngeal wall contraction/ residue on pharyngeal wall				Folder	
				MBS	
Dysfunctional UES				Folder	
				MBS	
Increased pharyngeal transit time				Folder	
				MBS	
Normal pharyngeal phase				Folder	
				MBS	

Comments:_____

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ADDITIONAL INFORMATION (✓✖N/A)					
	LIQUIDS	**SEMI-SOLID**	**SOLID**	**SOURCE**	**DATE**
Regurgitation				Folder	
				Clinical	
Vomit				Folder	
				Clinical	
Change in respiratory rate/effort with feeding / swallowing				Folder	
				Clinical	
GOR				Folder	
				Clinical	
				MBS	
				Milk Scan	
Nissen Funduplication				Folder	
				Clinical	